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14. ABSTRACT
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Emotional intelligence (EI) is defined as the ability to accurately perceive, understand, and use emotional information toward adaptive functioning. The goal of the present investigation was to understand the neurobiological basis of EI and to develop a training program to enhance these capacities. During the first 3 years of this study, 70 participants completed neuroimaging and EI testing. Analyses revealed that the functioning of inhibitory brain regions was related to facets of personality, daytime sleepiness, and gender. Higher EI was associated with greater responsiveness within core emotion processing circuitry of the brain. Furthermore, psychometric analysis suggested that trait and ability EI are in fact unique constructs, with Trait EI closely related to personality and Ability EI more correlated with standard cognitive intelligence (IQ). These findings then formed the basis for the development of an EI modification program during an additional supplemental year of funding. During this final phase of the study, we developed a preliminary version of a 6-module internet-based training program for enhancing EI skills, requiring less than 6 hours of time. This pilot version was evaluated in a randomized placebo-controlled investigation with 62 healthy participants (31 active EI training; 31 non-EI placebo training). The preliminary program significantly enhanced EI capacities, including Total EI, Perceiving Emotions and Facilitating Thought subscales of a primary outcome measure of EI. These findings suggest that it is possible to enhance EI skills with a brief on-line training program. Future work will focus on optimizing the pilot training program.

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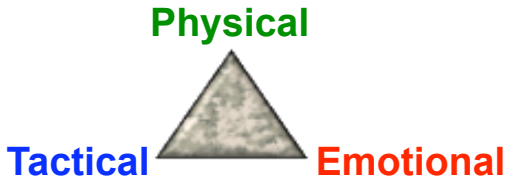
INTRODUCTION:

Military service, especially during deployment to combat or other dangerous environments, can take an extraordinary toll on emotional functioning. Members of the U.S. military often find themselves assigned to difficult missions under harsh conditions. These missions can be emotionally demanding, not only because of the physical challenges and potential exposure to life-threatening circumstances, but also because of the long separation from family and other close support networks. The challenges of combat and peacekeeping missions require warfighters to possess numerous skills and capacities, including physical strength and endurance, as well as well-honed tactical skills and technical knowledge. Additionally, the warfighter must be trained to deal effectively with the emotional stresses associated with military operations, which can range from the ever-present threat of harm, to the frequent exposure to differences in culture and belief systems, and long monotonous work days. Many military experiences, by their very nature, are extremely emotionally demanding and activate widespread neural, cognitive, endocrine, and physiological systems involved in self-preservation (Lieberman et al., 2005; Shalev, Bonne, & Peri, 1996). After returning home from a hazardous duty assignment, many servicemembers notice changes in emotional functioning compared to their pre-deployment status (Hoge et al., 2004). These changes can be wide-ranging, including episodes of hyperarousal, anger, aggression, and persistent low-level negative emotions such as sadness, depression, guilt, and cognitive rumination, but may also include emotional numbness and disinterest in previously pleasurable activities (Jerg-Bretzke, Walter, Limbrecht-Ecklundt, & Traue, 2013; Klemanski, Mennin, Borelli, Morrissey, & Aikins, 2012; Wright, Foran, Wood, Eckford, & McGurk, 2012). It is vital that new methods to protect military personnel against these emotional assaults be developed and made widely available.

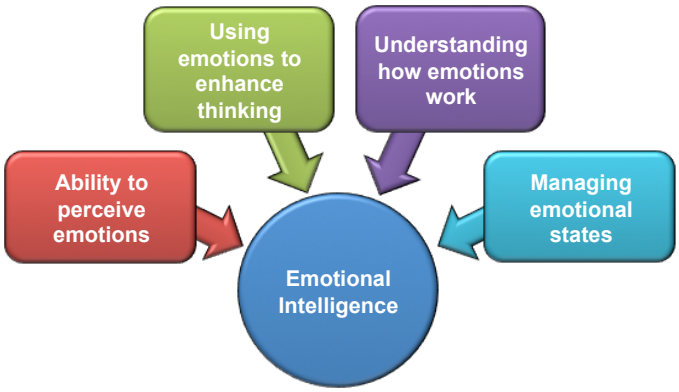
Although significant advances continue to be made in the development of new equipment, physical training techniques, and education in tactical capabilities, comparatively little effort has been aimed at strengthening the emotional skills that Soldiers need to cope effectively with the stresses of combat or to bounce back from the mental and emotional strains that are encountered during deployment. Just as a Soldier with inadequate training, poor physical conditioning, and insufficient body armor is at great risk of battlefield injury, so too a Soldier with poorly developed emotional capacities and fragile coping abilities is at increased risk for psychological wounds including depression, post-traumatic stress disorder, and even suicide. It is therefore imperative that warfighters be trained in emotional skills in addition to their training in physical and tactical capabilities.



Over the past decade, several large-scale attempts have been made to develop programs to help build emotional resilience capacities in military personnel (Adler, Bliese, McGurk, Hoge, & Castro, 2009; Castro, Adler, McGurk, & Bliese, 2012; Cornum, Matthews, & Seligman, 2011; Fravell, Nasser, & Cornum, 2011; Lester, McBride, Bliese, & Adler, 2011; Orsingher, Lopez, & Rinehart, 2008; Reivich, Seligman, & McBride, 2011). These efforts have led to the recent implementation of psychologically based initiatives such as the U.S. Army's Battlemind and Comprehensive Soldier Fitness programs. These attempts have made laudable and important strides toward enhancing resilience, but have also been criticized for being too broad in focus

(Steenkamp, Nash, & Litz, 2013) or providing too much emphasis on building optimism and positive mood states (Smith, 2013). Furthermore, progress in this area has been limited by the dearth of knowledge regarding the underlying neurobiology that contributes to the emotional capacities that allow a Soldier to cope effectively and remain resilient in the face of extreme and difficult challenges. More effective methods for developing these vital emotional skills are necessary. In particular, high yield training approaches that are focused on a well-defined theoretical model of emotional functioning while remaining brief, effective, and easily accessible are vitally needed to equip servicemembers with these crucial skills.

The goal of the present investigation was to understand the neurobiological basis of one aspect of emotional skills, known as emotional intelligence (EI), and to develop a training program to enhance these capacities. EI can be defined as the ability to use emotions and emotional information to function adaptively across a variety of situations (Mayer, Salovey, & Caruso, 2000). There are a number of competing theories of EI, but one of the most widely accepted views suggests that EI comprises 4 major domains, including 1) the ability to perceive emotions in others, 2) the ability to use emotions to enhance thought processes and problem solving, 3) knowledge and understanding about how emotions work, and 4) the ability to manage and control emotional states to achieve long-term goal states. Just as standard cognitive intelligence provides the foundation for successful learning, problem solving, and adaptation to a variety of occupational, educational, and intellectual settings, it is likely that EI capacities provide the foundation for successful coping and resilience across a variety of emotionally challenging situations (Mayer, Caruso, & Salovey, 1999; Mayer, Salovey, Caruso, & Sitarenios, 2001), including those encountered during military operations. In order to effectively identify these capacities and promote their enhancement among Soldiers through targeted training programs, we felt it would be necessary to understand the brain-behavior links that serve as the foundation of EI. At present, there is almost no information regarding the underlying brain systems involved in EI (Bar-On, Tranel, Denburg, & Bechara, 2003). Thus, for the first 4 years of the project, this study aimed to fill this gap by collecting neuroimaging data during emotional tasks and correlating such data with trait and ability models of emotional intelligence. Near the completion of the initial project (Year 4), we received notification that we were to receive an additional one-year of supplemental funding to create and test a preliminary EI training program.



As part of our ongoing effort to develop a rapid EI training system, the initial study involved using functional neuroimaging to map the neurocircuitry associated with normal variations in EI traits and abilities. Over the three-year funding period, 70 normal healthy participants ranging in age from 18 to 45 completed a comprehensive neurocognitive assessment battery that included two widely accepted measures of EI, assessment of standard cognitive intelligence (IQ), measures of coping, personality and resilience, as well as a host of emotional perception, decision-making, and problem solving tasks. These participants also underwent several

structural and functional magnetic resonance imaging scans at 3 Tesla while engaged in a variety of affective probe tasks designed to engage specific aspects of the neurocircuitry hypothesized to contribute to EI. The major goals of the study include: 1) identification of the neurocircuitry that is parametrically related to variability in EI scores, 2) evaluation of how EI brain systems differ from those of standard cognitive intelligence, 3) determination of whether the two commercially available tests of EI are measuring similar or different hypothetical constructs, and 4) determination of which test of EI is most predictive of brain activation within the hypothesized neurocircuitry and actual performance on emotional tasks.

Data completion for the study was completed within the initial three-year portion of the study and all accomplishments originally specified in the SOW were completed. After completion of data collection during the first three-years of the study, we requested a six-month no-cost extension to allow continued analysis of the data for additional validation. The extension was granted and we reported interim findings in the last annual report. As that 6-month no-cost extension came to a close, we received an additional 1-year of supplemental funding to permit us to develop and pilot test a preliminary on-line training program to enhancing EI capacities. The present report will also include the final results of the preliminary EI training program.

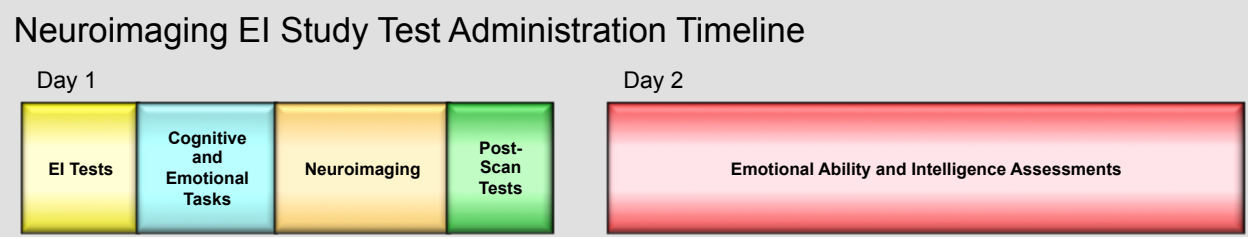
BODY:

Product Deliverables

All components of the initial and modified SOW have been accomplished. As of the date of this report, this project has yielded a total of ***107 published scientific abstracts*** and ***22 peer reviewed journal articles***.

Overview of the Research Design: Neuroimaging Study (Study 1)

The initial neuroimaging study (study 1) involved two separate testing days for each participant. During the first day, participants arrived at the laboratory and completed several standard measures of EI. These included the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), which is a measure of the Ability model of EI, and the Bar-On EQ-i, which is considered to be a measure of the Trait model of EI. Participants then completed a large battery of cognitive and emotional tasks followed by a neuroimaging session. The neuroimaging scans included several functional magnetic resonance imaging (fMRI) scans to examine brain responses to emotional stimuli, as well as a resting state functional connectivity scan, a structural MRI scan, and a diffusion tensor imaging (DTI) scan to examine the integrity of the white matter axonal tracts. After the scans, the participants completed several post-scan tests to determine their attention and memory for tasks in the scanner. Participants then returned for a second day and underwent approximately four hours of cognitive testing. These tests included standard intelligence (IQ) tests, as well as other emotional and cognitive ability tests. The general overview of the testing is shown below:



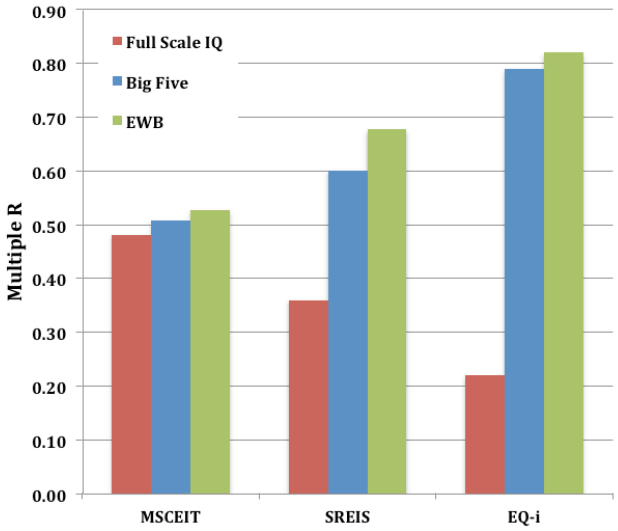
Research Findings: Primary EI Outcomes

The major goals of this project have been accomplished, as described in the subsections below. Our first goal was to validate the core construct of EI through psychometric analysis in a sample of 70 healthy participants. Within this same sample, we then focused on the role that EI contributes to decision-making relative to standard cognitive intelligence. Once it was clear that EI was a distinct hypothetical construct from other types of intelligence, we then examined EI as a potential mediator of the association between anxiety sensitivity (AS) and the expression of actual psychopathological symptoms of anxiety, suggesting it's importance for reducing mental health issues for servicemembers. Our next goal was to explore the structural brain correlates of EI, including gray matter volume and the integrity of the white matter axonal connections. After showing that EI is, in fact, correlated with these brain structure metrics, we then examined its relation to brain function. Using task-based fMRI techniques, we showed that EI is related to activation in key regions of the emotion processing systems, including ventromedial prefrontal cortex, insula, and amygdala. Next, we explored association between EI and functional connectivity within the brain, showing that EI is associated with greater ability to switch between default mode network (DMN) and task positive networks (TPN). After demonstrating the neurobiological correlates of EI, we then also conducted several secondary analyses to examine the associations between brain structure, brain function, and several key factors that are relevant to military health, including sleep loss, exercise, and responses to food stimuli. Finally, we developed a brief web-based EI Training program and validated it in a randomized placebo-controlled clinical trial with a sample of 62 healthy individuals. This final phase of the study demonstrated that the pilot version of the EI training program was significantly more effective than a matched placebo control program for enhancing EI skills. Together, these findings suggest that EI is a stable and valid construct, that it has identifiable and meaningful associations with brain structure, brain function, and neural connectivity, and that it can be significantly enhanced through a brief on-line training program. The sections below outline these findings in greater detail:

Validation of the Core Construct of Emotional Intelligence (EI)

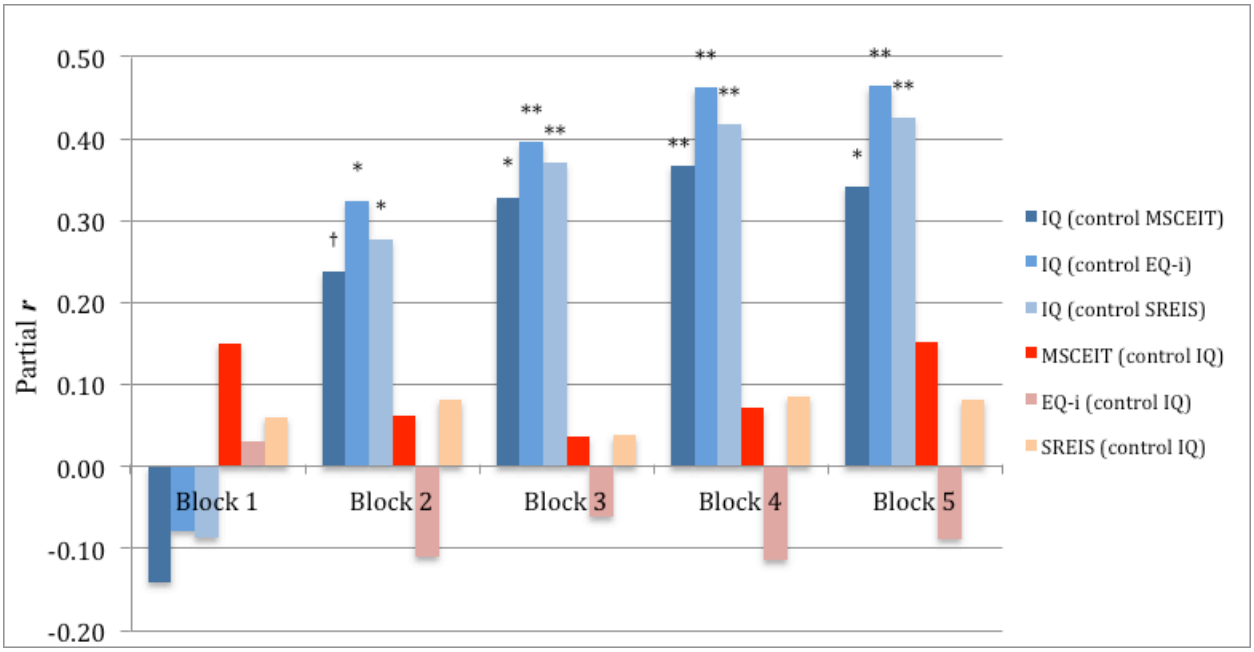
We aimed to validate the basis of emotional intelligence (EI) as a construct and whether it is indeed unique from traditional IQ. We used ability and trait measures of EI, which themselves appear to evaluate different psychological constructs. Results indicated that significant variability in the self-report EI measures was accounted for by personality and emotional well-being measures, whereas the MSCEIT was more strongly associated with IQ. Overall, nearly two-thirds (62%) of the variance in EQ-i scores was accounted for by Big Five personality traits, emotional well-being and full scale IQ; whereas only 14% of the variance in MSCEIT scores

was accounted for by these same variables. The present findings indicated that 1) competing measures of EI exhibit surprisingly small correlations with one another, and 2) significant variability in the self-report (but not performance-based) EI measures was accounted for by personality and emotional well-being measures. In summary, the current findings question the extent to which self-report measures of EI are appropriate given their substantial overlap with existing measures of personality and emotional state. This paper was recently published in the journal *Intelligence* (Webb et al., 2013) (see Appendix).



Cognitive versus Emotional Intelligence in Decision-Making

Debate persists regarding the relative role of cognitive versus emotional processes in driving successful performance on the widely used Iowa Gambling Task (IGT). From the time of its initial development, patterns of IGT performance were commonly interpreted as primarily reflecting implicit, emotion-based processes. Surprisingly, little research has tried to directly compare the extent to which measures tapping relevant cognitive versus emotional competencies predict IGT performance in the same study. The current investigation attempts to address this question by comparing patterns of associations between IGT performance, cognitive intelligence (Wechsler Abbreviated Scale of Intelligence; WASI) and three commonly employed measures of emotional intelligence (EI; Mayer-Salovey-Caruso Emotional Intelligence Test, MSCEIT; Bar-On Emotion Quotient Inventory, EQ-i; Self-Rated Emotional Intelligence Scale, SREIS). Results indicated that IGT performance was more strongly associated with cognitive, than emotional, intelligence. As illustrated in the Figure below, after controlling for Full Scale IQ, there were no



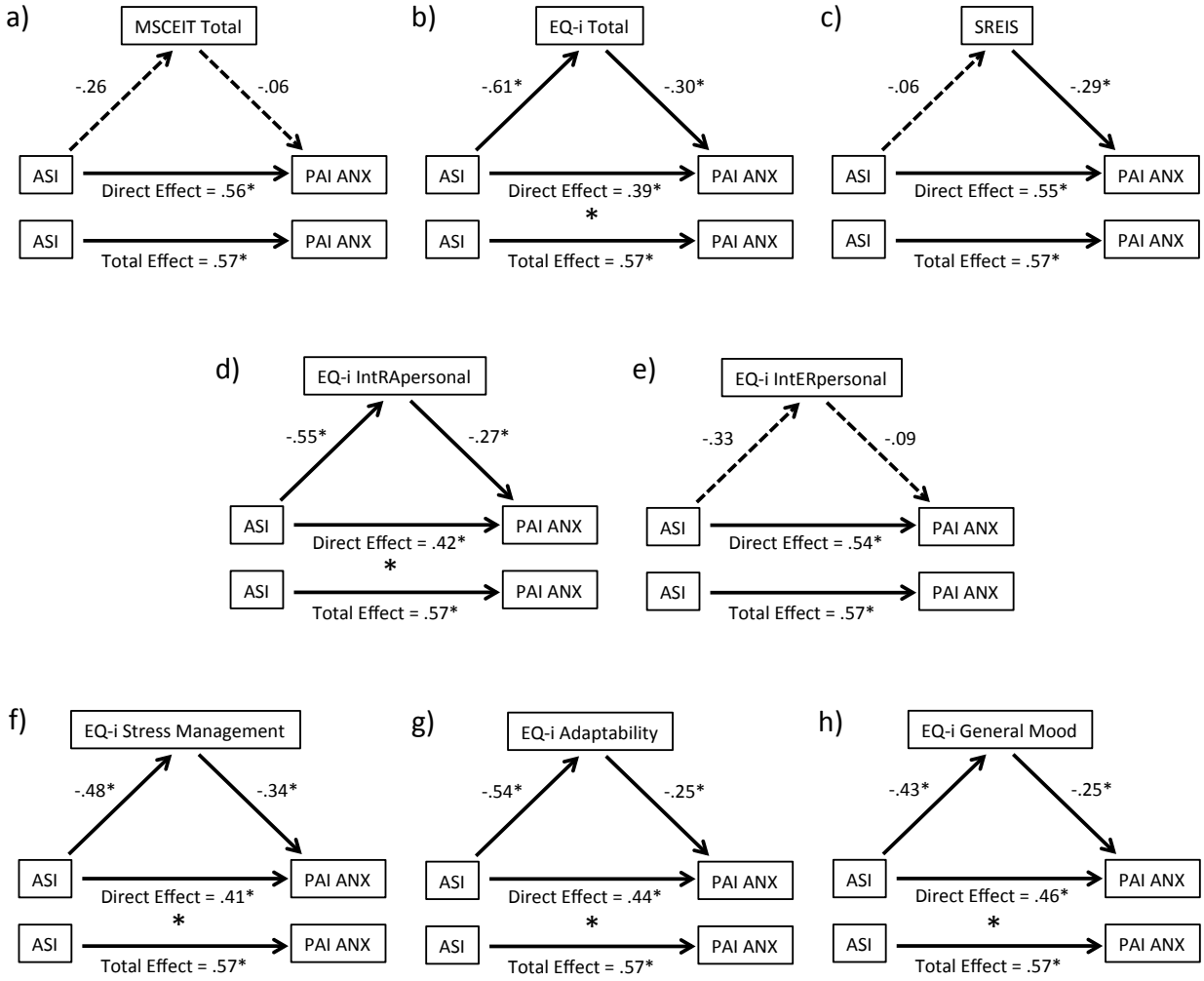
significant associations between any of the three EI measures and IGT performance across any blocks (for MSCEIT, all $r < .16$ & $p > .27$; for EQ-i, all $r < .04$ & $p > .41$; for SREIS, all $r < .09$ & $p > .53$). In contrast, when controlling for the different measures of EI, Full Scale IQ remained significantly associated with several IGT performance variables. Specifically, when controlling for MSCEIT scores, Full Scale IQ was significantly associated with IGT performance in blocks 3-5 (block 3, $r = .33$; $p = .016$; block 4, $r = .37$; $p = .006$; block 5, $r = .34$; $p = .011$). When controlling for EQ-i scores, Full Scale IQ was significantly associated with IGT performance in blocks 2-5 (block 2, $r = .33$; $p = .016$; block 3, $r = .40$; $p = .003$; block 4, $r = .46$; $p < .001$; block 5, $r = .46$; $p < .001$). Similarly, when controlling for SREIS scores, Full Scale IQ was significantly associated with IGT performance in blocks 2-5 (block 2, $r = .28$; $p = .042$; block 3, $r = .37$; $p = .006$; block 4, $r = .42$; $p = .002$; block 5, $r = .43$; $p = .001$). To the extent that the IGT indeed mimics "real-world" decision-making, our findings, coupled with the results of other research discussed below, may highlight the role of deliberate, cognitive capacities over implicit, emotional processes in contributing to at least some domains of decision-making relevant to everyday life. This finding was recently published the journal *Intelligence* (Webb, DelDonno, & Killgore, 2014)(see Appendix).

Emotional Intelligence as a Mediator of the Association between Anxiety Sensitivity and Anxiety Symptoms

The construct of Anxiety Sensitivity (AS), which refers to the fear of anxiety-related symptoms, including the physical sensations, thoughts, and social consequences associated with anxiety, has been theorized to be a cognitive vulnerability that contributes to the development of an anxiety disorder. However, the extent to which anxiety sensitivity predicts anxiety disorder symptoms may depend on emotional factors. Among individuals high in anxiety sensitivity, non-acceptance of emotional distress and less access to emotion regulation strategies is related to greater anxious arousal. We hypothesized that the level of Emotional Intelligence (EI) would mediate the relationship between AS and self-rated anxiety symptoms.
Sixty-one healthy adults (30 men) aged 18 to 45 completed measures of AS (Anxiety Sensitivity Index, ASI), anxiety symptoms (Personality Assessment Inventory, PAI), a "trait" measure of EI (Bar-On Emotional Quotient Inventory, EQ-i), and two "ability" measures of EI (Mayer-Salovey-Caruso Emotional Intelligence Test, MSCEIT; Self-Rated Emotional Intelligence Scale, SREIS). Mediation analyses were used to assess the influence of each of the measures of EI on the relationship between AS and anxiety symptoms.

EQ-i was a significant partial mediator of the relationship between AS and PAI anxiety symptoms ($z = 2.95$, $p = .003$). However, there were no mediation effects for the ability measures of EI, either for MSCEIT scores ($z = .614$, $p = .539$) or SREIS ratings ($z = .549$, $p = .583$), on the relationship between AS and anxiety symptoms. Additional mediation analyses revealed that four of the subscales of the EQ-i (Intrapersonal, Stress Management, and General Mood) partially mediated the association between anxiety sensitivity and anxiety symptomatology, but there was no mediation effect for the Interpersonal subscale, $p = .32$.

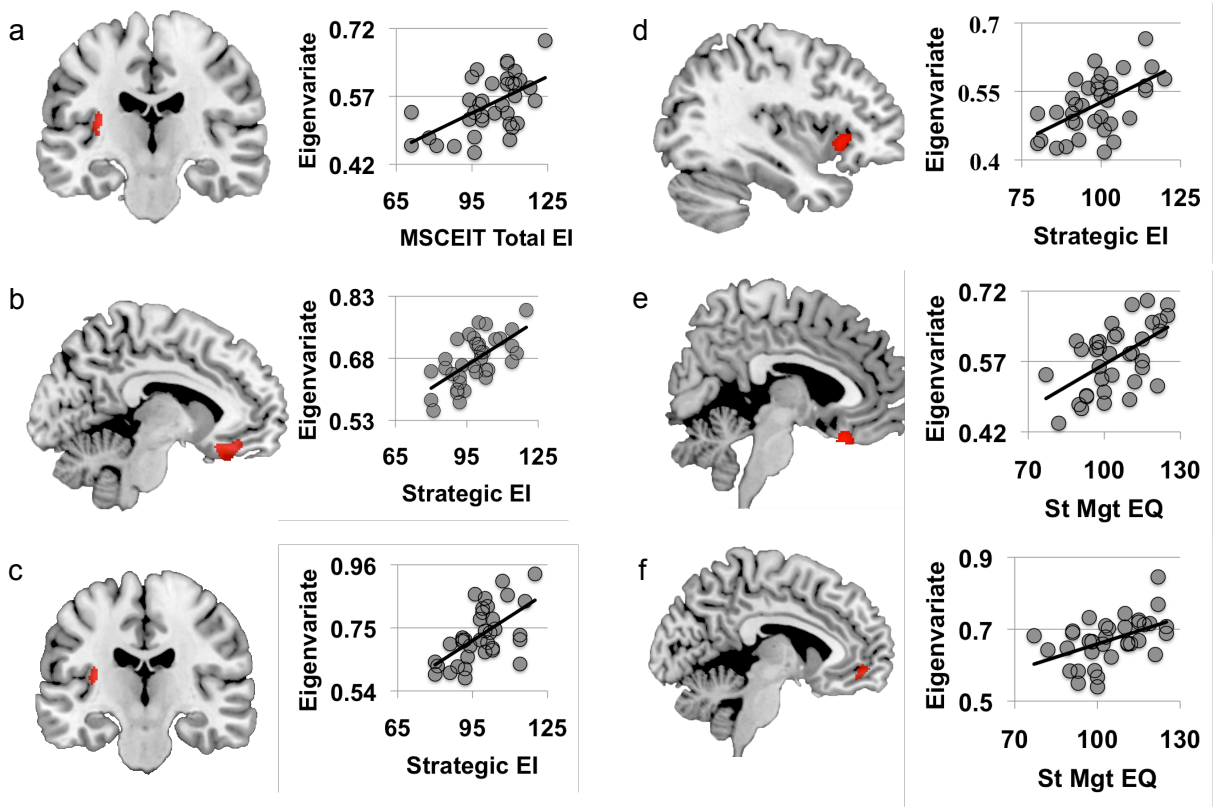
Findings showed that trait EI, but not ability EI, mediated the relationship between anxiety sensitivity and anxiety symptoms. Whereas the EQ-i measures a broad range of EI traits, which overlap with general emotional wellbeing, the MSCEIT and SREIS assess specific emotional skills. These findings suggest that factors related to emotional wellbeing, rather than specific emotional skills and abilities, mediate the relationship between anxiety sensitivity and anxiety symptoms. Findings may have implications for interventions designed to reduce anxiety in military personnel by targeting the mediating factor of EI through programmed training.



Emotional Intelligence and Gray Matter Volume of the Cerebral Cortex

Research suggests that emotional intelligence capacities may be related to the functional integrity of the corticolimbic regions including the ventromedial prefrontal cortex, insula, and amygdala. No study has yet examined regional brain volumes in relation to the two dominant models of emotional intelligence: the Ability model, which posits a set of specific demonstrable capabilities for solving emotional problems, and the Trait model, which proposes a set of stable emotional competencies that can be assessed through subjectively rated self-report scales. Thirty-six right-handed, primary English-speaking adults (mean age 30.0±8.9, range 18–45; 20 men) were recruited from the Boston metropolitan area and received payment for their time. Participants

had no history of neurological, psychiatric, or substance use disorders (including alcohol and illicit drugs). Each participant completed two validated and commercially available tests that measure alternative models of emotional intelligence. As an index of Ability emotional intelligence, participants completed the Mayer–Salovey–Caruso Emotional Intelligence Test (MSCEIT) [2]. The MSCEIT yields a Total emotional intelligence score and two Area scores, Experiential emotional intelligence and Strategic emotional intelligence. Experiential emotional intelligence reflects the ability to perceive emotions in oneself, other persons, and various inanimate stimuli, and to utilize emotional information in facilitating cognition. Strategic emotional intelligence reflects the ability to understand emotions and their evolution in oneself and others, and to manage them in an efficient and effective manner. Raw scores were converted to scaled scores on the basis of the general normative group, without adjustment for sex. As a measure of Trait emotional intelligence, participants completed the Bar-On Emotional Quotient Inventory (EQ-i) [4]. The Interpersonal scale provides a measure of perceived empathy and interpersonal skills, whereas the Intrapersonal scale reflects self-perceived awareness of one's own emotions and self-regard. The Adaptability scale reflects the perceived ability to objectively analyze problematic situations, to solve them, and to adapt to changing environments. Stress Management reflects tolerance of and perceived self-control during stressful or demanding situations. The General Mood scale reflects self-reported positive thinking and overall contentedness with personal life. Regional brain volumes were analyzed using voxel-based morphometry. Total Mayer–Salovey–Caruso Emotional Intelligence Test scores were positively correlated with the left insula grey matter volume. The Strategic emotional intelligence subscale correlated positively with the left ventromedial prefrontal cortex and insular volume.

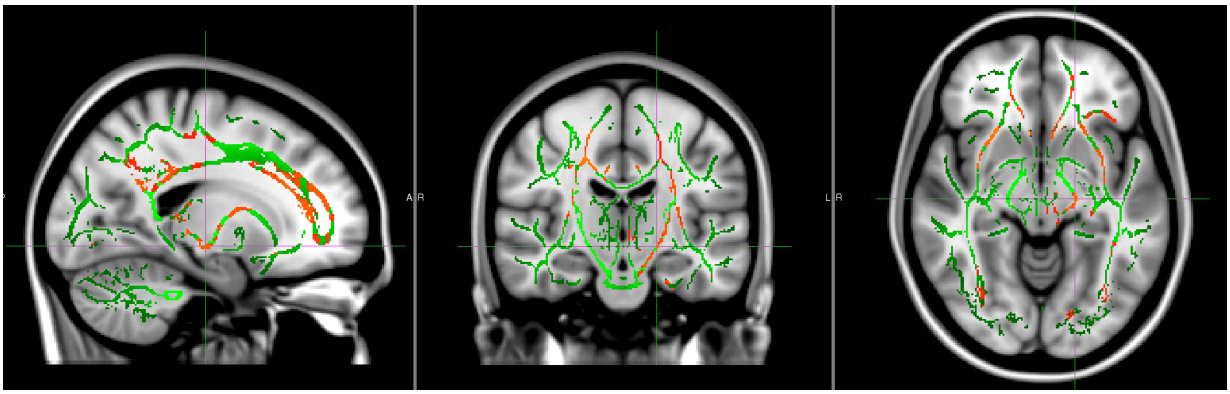


Total MSCEIT emotional intelligence was positively correlated with gray matter volume of the left posterior insula (see figure below, Fig. a). When evaluated by subscale, only the Strategic emotional intelligence Area scale of the MSCEIT correlated positively with Somatic Marker Circuitry gray matter, including the bilateral medial prefrontal cortex (i.e. gyrus rectus and the orbital region of the medial frontal gyrus; Fig. b), left posterior insula (Fig. c), and left anterior insula/ventrolateral prefrontal cortex, including the inferior frontal gyrus (Fig. d). Experiential emotional intelligence was not correlated with gray matter volume. For the EQ-i, Total score was unrelated to gray matter volume. Of the subscales, however, Stress Management was significantly positively correlated with gray matter volume within the right (gyrus rectus; Fig. e) and the left ventromedial prefrontal cortex (orbital region of the medial frontal gyrus between the anterior rostral and paracingulate sulci; Fig. f). None of the other EQ-i subscales were significantly correlated with gray matter volume in the Somatic Marker Circuitry

Trait and Ability measures involving emotional regulation facets of emotional intelligence were both related to gray matter volume in the ventromedial prefrontal cortex, whereas only Ability emotional intelligence was specifically associated with gray matter volume in the insular cortex. These findings support the role of the ventromedial prefrontal cortex and insula as key nodes in the Somatic Marker Circuitry, and further suggest that larger volume of these regions in nominally healthy adult participants is associated with greater capacities for understanding and using emotional information and for demonstrating resilience and effective coping in the face of stress and adversity. This study was published in the journal *NeuroReport* (Killgore et al., 2012).

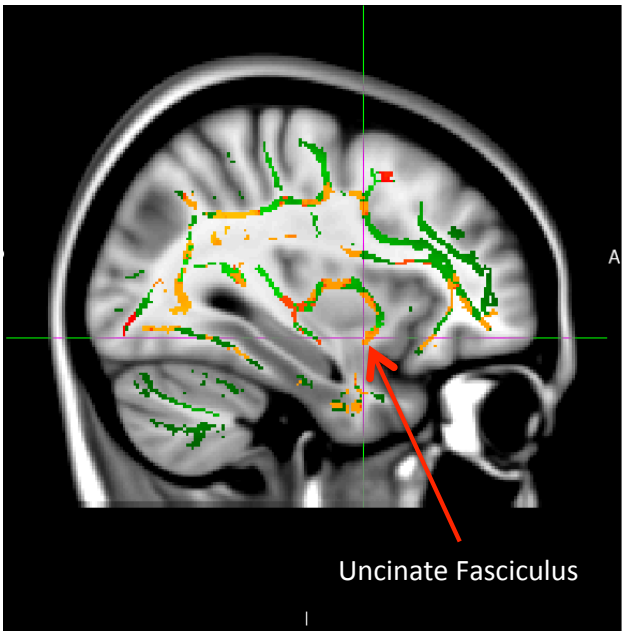
Emotional Intelligence and Subcortical White Matter Integrity

We have also collected diffusion tensor imaging (DTI) data for a subset of participants in the study. DTI is a technique that allows quantification of the movement of water molecules, which permits an inference of the integrity of the axonal tracts. The most commonly used metric of DTI is known as Fractional Anisotropy (FA), which provides a index of the degree of directionality of molecular diffusion. High values of FA indicate that diffusion occurs along a single directional axis, suggesting high integrity of the myelinated axonal pathways and tracts (see significant orange regions overlaid on the green tracts below). On the other hand, low FA suggests that diffusion is multidirectional, suggesting damage or low integrity to the axonal pathways. Our preliminary investigation using Diffusion Tensor Imaging and Tract Based Spatial statistics (TBSS) to analyze white matter FA, has revealed a number of significant



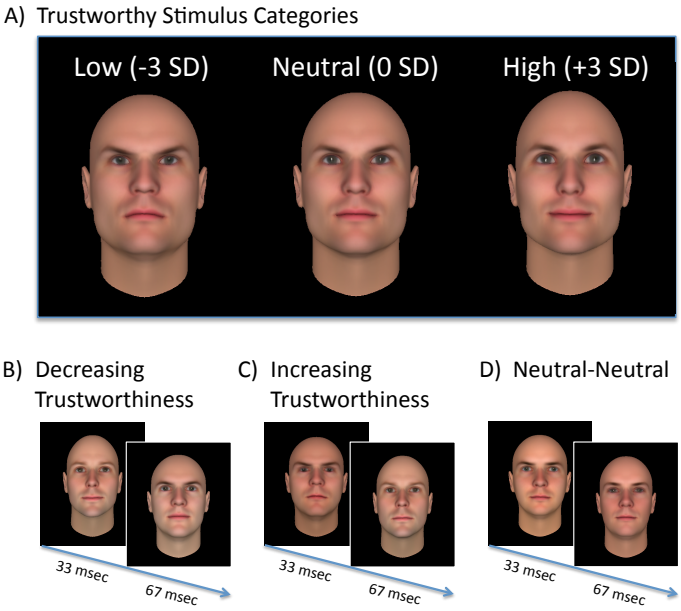
relationship between EI and white matter integrity in the brain. Prior DTI research has explored the relationship between EI and neurobiological correlates, however its methodology has been limited to self-report measures of EI (as opposed to performance based measures).

Of the 45 participants with complete DTI data, images from nine participants were omitted during quality control due to significant EPI distortion in frontal areas and/or rotational warping. A standard preprocessing pipeline was utilizing using the FSL 5.0 diffusion toolkit. Standard eddy correction was performed, along with bvec rotation, and conventional FSL TBSS preprocessing of FA images. MSCEIT total values for each respective participant were mean-centered and entered into FSL's Generalized Linear Model graphical user interface to generate design.con and design.mat files. Finally, FSL's 'randomise' was used with the T2 cluster-based thresholding option and ten thousand permutations to generate p-value significance image corrected for multiple comparisons.

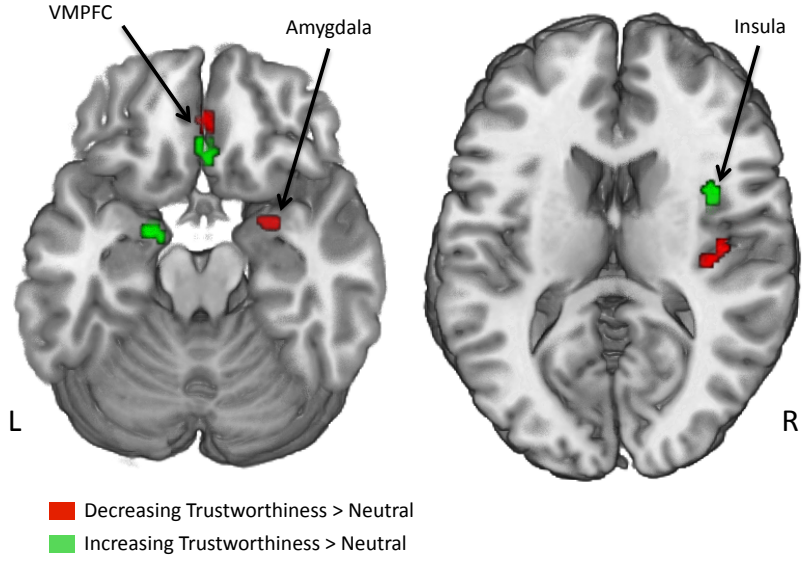


Using a $p < .02$ threshold (corrected for multiple comparisons) in fslview, analysis revealed significant correlations between Ability EI and white matter integrity across multiple regions of the brain (shown in orange-red in the figure at right). Of particular interest, we hypothesized that EI would be associated with greater FA values in the uncinate fasciulus, a fiber pathway connecting the orbifrontal cortex and the amygdala, which is believed to play an important role in emotional regulation. In addition, EI correlated with higher FA values within tracts connecting insular cortex, and to lesser extent, the amygdala, hippocampal and parahippocampal gyri. We also found stronger FA in the frontal cortex, particularly areas in and around the orbito-frontal cortex and frontal pole, regions that are also involved in emotional regulation. Furthermore, we observed very significant correlations between the MSCEIT and the precuneus, precentral gyrus, cingulate gyrus, and corpus collosum. Occipital areas responsible for visuospatial processing and object recognition, such as the temporal occipital fusiform cortex, optic radiation, and occipital fusiform gyrus, also showed heightened FA. Finally, increased FA in the planum polare, heschl's gyrus, planum temporal, and other scattered areas of the inferior temporal gyrus suggests greater integrity of language processing centers of the brain. Finally, given the extent of these correlations, and to rule out any possible interaction with confounds, a parallel analysis was conducted using only mean-centered ages in the correlation. In our participant pool, results showed no significant correlations between age and FA anywhere in the brain. Overall, these findings suggest that higher EI is associated with greater structural integrity of the white matter axonal tracts connecting key emotional regulation and emotional processing regions of the brain.

Emotional Intelligence and Functional Responses to Dynamic Changes in Facial Trustworthiness



While little is known about the neurobiological substrates that underlie EI, some evidence suggests that these capacities may involve a core neurocircuitry involved in emotional decision-making that includes the ventromedial prefrontal cortex (vmPFC), anterior cingulate cortex (ACC), insula, and amygdala. In a sample of thirty-nine healthy volunteers (22 men; 17 women), scores on the Bar-On EQ-i (a Trait/Mixed model of EI) and Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT; an Ability model of EI) were correlated with functional magnetic resonance imaging responses during brief presentations of moving facial expressions that changed in the level of perceived trustworthiness. As shown in *the figure below*, facial features were morphed along a continuum of trustworthiness according to the methods outlined by Oosterhof and Todorov (2008). A) Three categories of faces were used, selected from those rated 3 standard deviations (SD) below the mean in trustworthiness (left), those at the mean of trustworthiness (center), and those rated 3 SD above the mean in trustworthiness features (right). During the DFTT, pairs of faces were presented to give the appearance of subtle facial movement. B) During the *Decreasing Trustworthiness* presentations, a High Trustworthy face (+3 SD) was presented for 33 ms, followed by a Neutral Trustworthy face (0 SD) for 67 ms, which gave the impression of movement toward lesser trustworthiness. C) During the *Increasing Trustworthiness* presentations, a Low Trustworthy face (-3 SD) was presented for 33 ms, followed by a Neutral Trustworthy face (0 SD) for 67 ms, which gave the impression of movement toward greater trustworthiness. D) During the *Neutral* presentations, a Neutral Trustworthy face (0 SD) was presented for 33 ms, followed by a different Neutral Trustworthy face (0 SD) for 67

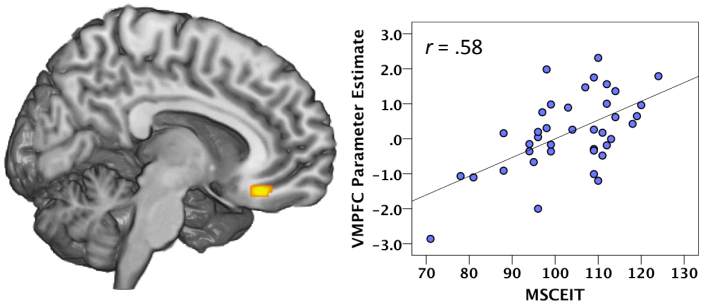


ms, which provided a control for potential movement effects associated with changing face identities independent of changes in trustworthiness.

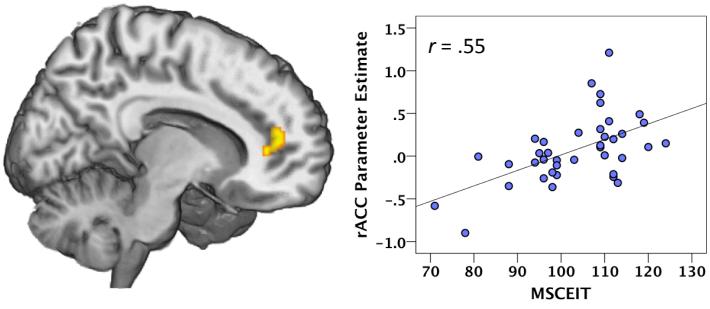
Core emotion neurocircuitry was responsive to dynamic changes in facial features, regardless of whether they reflected increases or decreases in apparent trustworthiness. In response to facial movements indicating decreasing trustworthiness, MSCEIT correlated positively with functional responses of the vmPFC and rostral ACC, whereas the EQ-i was unrelated to regional activation. The *Figure below* shows regions of functional activation associated with the contrasts between *Decreasing Trustworthiness* > *Neutral* (red) and *Increasing Trustworthiness* > *Neutral* (green). Significance was evaluated using a small volume correction for multiple comparisons within each search territory at $p < .001$ (uncorrected), $p < .10$, False Discovery Rate (FDR) corrected, k (extent) ≥ 10. The image shows that the *Decreasing Trustworthiness* > *Neutral* contrast was associated with increased activation of the ventromedial prefrontal cortex (vmPFC) and right amygdala (left image), and posterior insula (right image). The *Increasing Trustworthiness* > *Neutral* contrast was also associated with increased activation of the vmPFC as well as the left amygdala (left image) and anterior insula (right image).

As evident below in the *Figure* below, there were significant clusters of activation that correlated with Emotional Intelligence (EI), $p < .10$ (small volume corrected), $k \geq 10$. A) Total scores on the MSCEIT were positively correlated with responses of the ventromedial prefrontal cortex (vmPFC) for the contrast of *Decreasing Trustworthiness* versus implicit baseline (left) [x = 6, y = 32, z = -16]. For visualization purposes, the scatterplot (right) shows the relationship between MSCEIT scores and the first eigenvariate extracted for the entire correlated cluster. B) Total EI scores on the MSCEIT were positively correlated with responses within the rostral ACC (rACC) for the contrast of *Decreasing* versus *Increasing Trustworthiness* (left) [x = 14, y = 44, z = 12]. For visualization purposes, the scatterplot (right) shows the relationship between MSCEIT scores and the first eigenvariate extracted for the entire correlated cluster.

A) Decreasing Trustworthiness



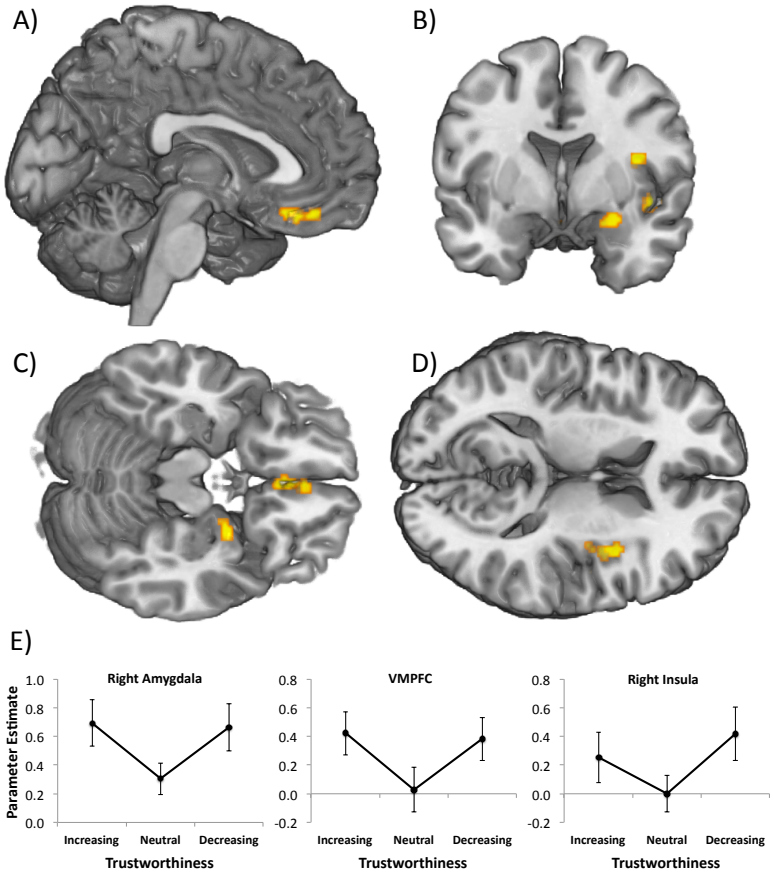
B) Decreasing Trustworthiness > Increasing Trustworthiness



Finally, as shown in the *Figure* below, a trend analysis revealed a quadratic pattern of responsiveness across the 3 trustworthiness conditions of *Increasing Trustworthiness*, *Neutral*, and *Decreasing Trustworthiness* within key regions of interest, including the ventromedial prefrontal cortex (vmPFC), right amygdala, and right insula. Clusters showing this quadratic pattern can be seen on the A) sagittal slice (vmPFC), B) coronal slice (right amygdala and insula), and slices showing C) inferior axial (vmPFC and right amygdala), and D) superior axial

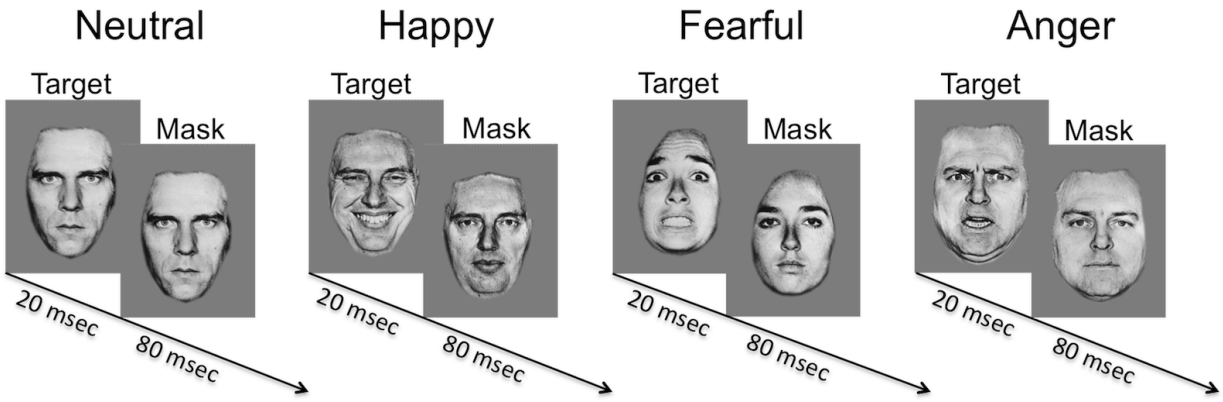
(right insula) perspectives. E) Parameter estimates from the right amygdala, vmPFC, and right insula were extracted from the displayed clusters and plotted for visualization.

Overall, these findings suggest that greater EI was associated with increased responsiveness of the medial prefrontal cortex during a socially relevant dynamic face perception task, providing partial support for the role of the SMC in these capacities. Discrete nodes of the SMC, including the vmPFC and rostral ACC, were specifically correlated with *Ability* EI capacities, while *Trait* EI was not significantly related to the responsiveness of the hypothesized regions during dynamic facial displays communicating trustworthiness information. Overall, systematic differences in EI capacities appear to be significantly related to the responsiveness of higher order emotion assessment and regulation regions of the medial prefrontal cortex and rostral anterior cingulate. These findings were published in the journal *NeuroImage* (Killgore, Schwab, Tkachenko, et al., 2013).



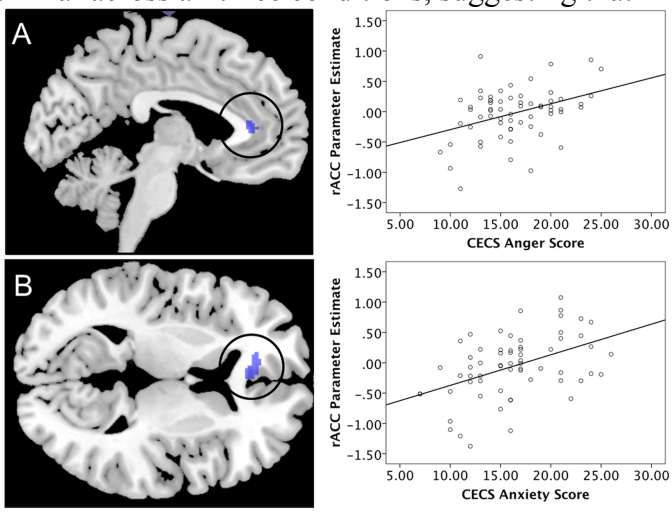
Emotional Suppression and Functional Brain Responses to Angry Faces

Emotional suppression (ES) is a critical component of the ability to self-regulate emotion. However, people who chronically employ ES as a primary strategy often experience heightened anxiety or depression. Although functional neuroimaging studies have extensively mapped the brain regions involving in emotional regulation, the neural substrates of ES as a trait construct remains relatively unexplored. Using a validated backward masked facial affect paradigm (see figure below), we examined the association between ES and functional brain responses to masked angry, fearful, and happy faces. Each affective face was presented for 20 msec, followed by a neutral mask for 80 msec. This procedure effectively prevents conscious awareness of the target affective face.



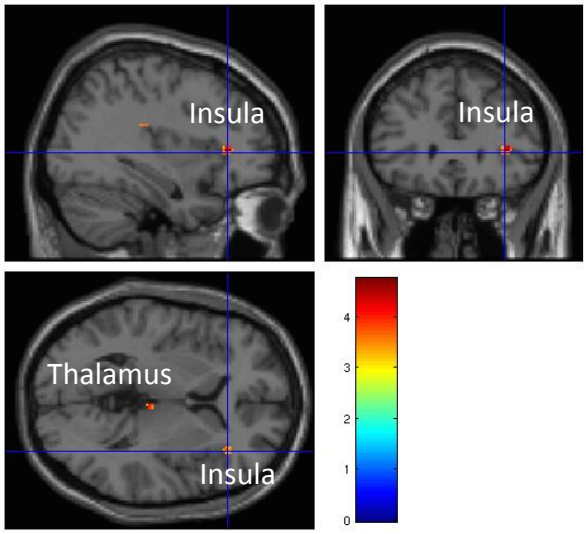
Sixty-three right-handed healthy native English-speaking adults (mean age 30.3±8.1 years, range 18-45; 33 males, 30 females) were recruited from the Boston metropolitan area and underwent functional MRI and completed the Courtauld Emotional Control Scale (CECS) as a measure of ES. Correlations between self-reported ES and brain responses to the facial affect stimuli (affective > neutral) were evaluated within the brain regions involved in emotional processing, including the amygdala, insula, anterior cingulate cortex, medial prefrontal cortex, and orbitofrontal cortex.

In response to masked angry faces, a cluster of activation located within the rostral ACC (rACC) was correlated with higher CECS Anger Score (43 voxels; T=5.5; MNI coordinates: x=-4, y=36, z=10; p=0.002, FDR corrected). A cluster of activation within the rACC was also positively correlated with CECS Anxiety Score (214 voxels; T=5.67; MNI coordinates: x=-4, y=36, z=10; p=0.001, FDR corrected) in response to angry faces (see Figure at right). For masked fearful and happy faces, no significant correlations were observed within any of the ROIs. Additionally, to verify that the specificity of the results was not simply driven by greater range of BOLD activation responses within the masked anger condition relative to the other conditions, we extracted and plotted the mean and standard deviation of the functional responses from each ROI, including the original cluster of activation in the rACC defined by the significant correlation with CECS Anger Scores above. As shown in the figure, the range of BOLD responses, particularly within the ACC was similar across all three conditions, suggesting that the specificity of our findings to masked anger cannot be explained by restricted range for the other two conditions. Thus, when confronted with images of angry faces, higher trait tendency to suppress anger and anxiety was significantly correlated with increased activation within the rostral anterior cingulate cortex, whereas no correlation was observed for masked happy or fearful faces. This finding suggests that rostral anterior cingulate cortex contributes to the unconscious suppression of emotional responses to angry facial affect, and may



play a role in the mediating anatomy of trait ES.

Next, we hypothesized that higher trait ES would also correlate with greater functional deactivation in the insula, a region associated with the visceral experience of emotion, and the thalamus, a region critical to information transfer, in response to subliminal presentations of anger. In this case, we found that masked angry faces, CECS scores correlated with significant ($p<.10$ FDR corrected; $k \geq 16$) deactivation within the right insula and the right and left thalamus. Specifically, in response to masked angry faces, a cluster of deactivation located within the right insula was correlated with higher CECS total scores (19 voxels; T=4.74; MNI coordinates: x=36 y=30 z=6; p=.039 FDR corrected). Moreover, multiple clusters of deactivation were identified within the right, and particularly the left thalamus, albeit both thalamic regions showed less statistical significance (12 voxels; T=4.23; MNI coordinates: x=-16 y=-12 z=14; p=.055 FDR corrected) and (19 voxels; T=3.94; MNI coordinates: x=4 y=-24 z=4; p=.083 FDR corrected), respectively. Consistent with our hypothesis, those with higher ES exhibited greater deactivation in the insula and thalamus to masked angry faces. In the context of prior work suggesting that ES is associated with increased rACC activation, our findings suggest that greater ES might also involve top down-regulation of the interoceptive perception systems, perhaps to reduce emotional experience. These findings point to a neural system involved in emotional control. This study was published in the journal NeuroReport (Cui et al., 2014).

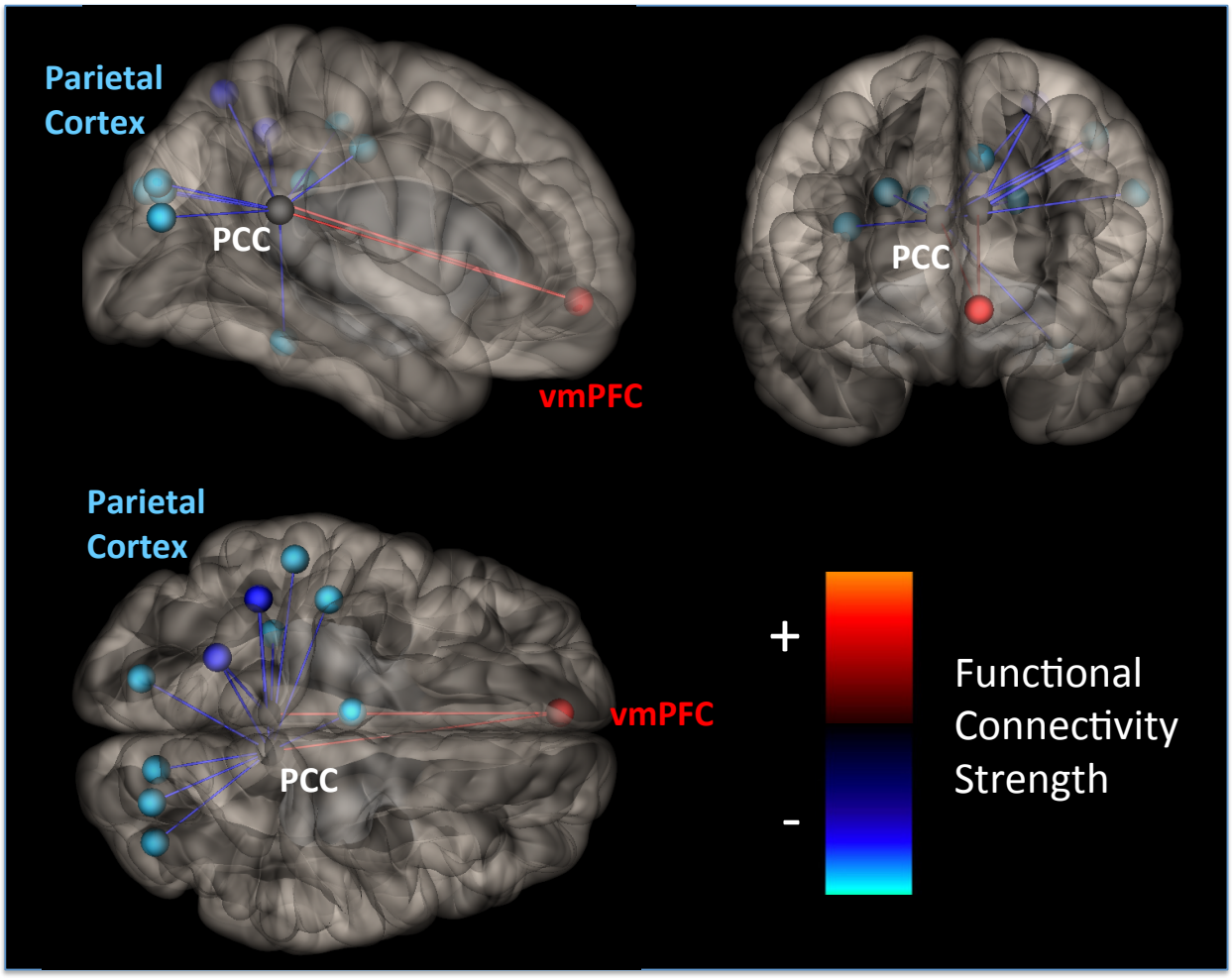


Trait Emotional Intelligence and Resting State Functional Connectivity

Some evidence suggests that healthy emotional capacities may involve the ability to shift flexibly between internal and external focus. Accordingly, we hypothesized that an individual's self reported level of EI would correlate with inverse connectivity relationships between areas related to internal focus and self-reflective processing (Default Mode Network; DMN) and external environmental focus (Parietal Task Positive Network; P-TPN), as mediated by the posterior cingulate cortex (PCC). Sixty healthy adults (50% female; Mean: 30.4 years) completed the Bar-On Emotional Quotient Inventory and a six-minute resting state functional magnetic resonance imaging (fMRI) scan at 3T.

Bilateral regions of interest were placed in the PCC (see figure below, gray spheres), along with individual regions of interest placed in the ventromedial prefrontal cortex (vmPFC; see figure, red sphere) and regions of the parietal cortex as defined by the Automated Anatomical Labeling Atlas (see figure, blue spheres). Functional connectivity was analyzed utilizing the CONN toolbox and SPM12. EI correlated positively with increased functional connectivity between the PCC and left vmPFC (i.e., DMN), but was associated with anticorrelated functional connectivity between the PCC and several parietal regions (i.e., P-TPN). Self-rated scores on one of the most

widely used Trait EI measures were associated with inverse resting state connectivity between DMN and TPN regions. The results suggest that one component of higher EI may involve the flexibility of transition between cognitive states involving internal self-reflective focus and engagement with external stimuli.

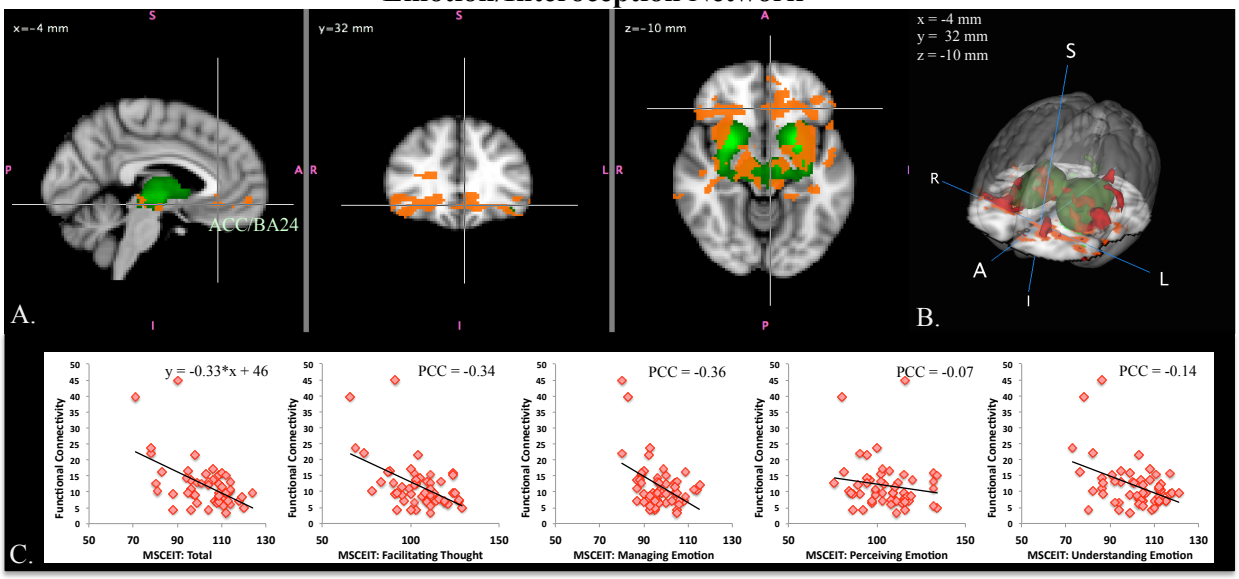


Ability Emotional Intelligence and Synchronized Resting State Functional Activity

The capacity to accurately perceive, understand, and regulate emotions, and to apply that information to facilitate thought and performance is known as Emotional Intelligence (EI). Although research suggests that EI plays an important role in mental health and success in academic, professional, and social realms, the underlying neurocircuitry contributing to this capacity remains poorly understood. Here, we explored the regional functional connectivity underlying two leading alternative models of EI. Healthy, right-handed adults (n = 54, 26 men), with a mean age of 30.1 years (SD=7.5 years) completed standardized validated measures of the Trait (EQ-i) and Ability (MSCEIT) EI models and then underwent resting state functional magnetic resonance imaging (rsfMRI). FSL MELODIC was used to implement an independent components analysis (ICA) with dual regression to investigate resting state networks (RSNs), whose activity was thought to be associated with greater EI capacities. Results are reported at $p<0.05$ (FWE corrected).

As evident in the figure below, the *Emotion/Interoception Network was associated with Ability EI measures:* A) Three orthogonal slices showing regions whose functional connectivity with the EIN (green) is statistically significantly associated with MSCEIT (red-yellow, p<0.05 corrected). The crosshairs show one region in the medial PFC. B) 3D view to better illustrate the spatial extent/pattern. C) Functional connectivity values (regression coefficients averaged over voxels in red-yellow in A) plotted against MSCEIT: Total and MSCEIT sub-scales for Facilitating, Managing, Perceiving, and Understanding. Partial correlation coefficients (PCC) between functional connectivity values and MSCEIT values are shown for each sub-scale.

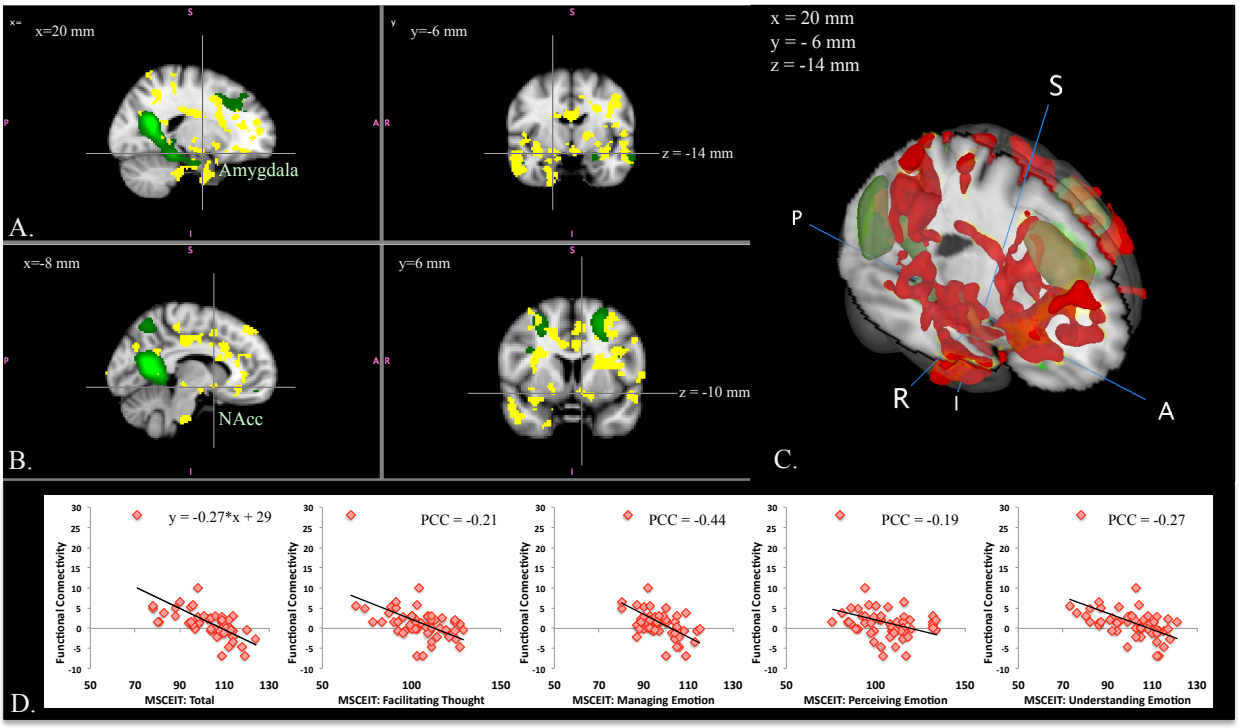
Emotion/Interoception Network



The figure below shows the findings for the *DMN-Related Network.* Two orthogonal slices for two different x locations showing regions whose functional connectivity with the DMN is inversely related to MSCEIT (red-yellow, $p < 0.05$, corrected). Crosshairs in A) bisect the amygdala, B), the Nucleus Accumbens, and C) 3D view to better illustrate the spatial extent/pattern. D) Functional connectivity values plotted against MSCEIT values (PCCs are shown for each sub-scale).

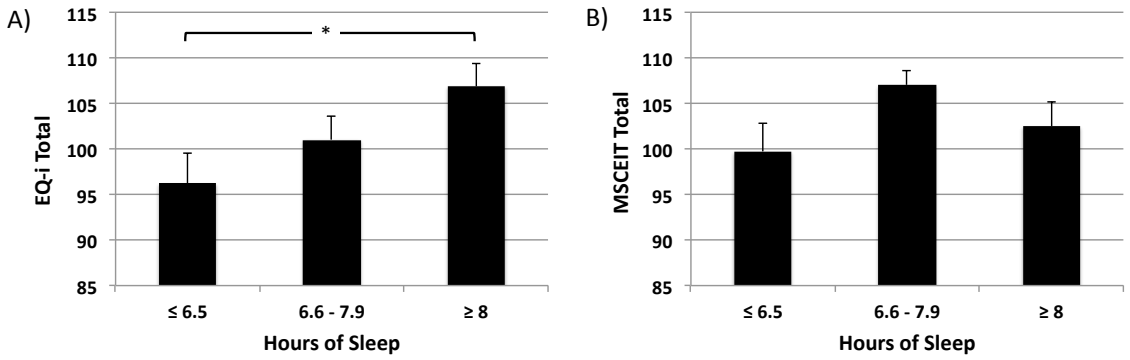
Overall, these findings show that higher scores for Ability EI (as opposed to Trait EI) are associated with stronger inverse correlations of the spontaneous fMRI signals from RSNs involved in affective regulation (e.g., prefrontal) and fMRI signals involved in somatic emotional responses (e.g., insula), and also correlated with the strength of connectivity between FMRI signals from these emotionally responsive networks and those networks involved in self-reflective cognition (e.g., medial frontal/posterior cingulate). These findings suggest that stronger inverse correlations between signals from key intrinsic emotional regulation and interoceptive experience networks serve as a marker of higher emotional intelligence skills and abilities, perhaps reflecting greater capacity to regulate emotional responses through executive control processes.

DMN-Related



Sleep, Emotional Intelligence, and Cortico-Limbic Functional Connectivity

Prior research suggests that sleep deprivation is associated with declines in some aspects of emotional intelligence and increased severity on indices of psychological disturbance. Sleep deprivation is also associated with reduced prefrontal-amygdala functional connectivity, potentially reflecting impaired top-down modulation of emotion. It remains unknown whether this modified connectivity may be observed in relation to more typical levels of sleep curtailment. We examined whether self-reported sleep duration the night before an assessment would be associated with these effects. Participants documented their hours of sleep from the previous night, completed the Bar-On Emotional Quotient Inventory (EQ-i), Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), Personality Assessment Inventory (PAI), and underwent resting-state functional magnetic resonance imaging (fMRI). Sixty-five healthy adults (33 men, 32 women), ranging in age from 18-45 years underwent resting state functional connectivity scanning and completed measures of emotional intelligence and emotional functioning.



Greater self-reported sleep the preceding night was associated with higher scores on all scales of the EQ-i but not the MSCEIT, and with lower symptom severity scores on half of the psychopathology scales of the PAI. Longer sleep was also associated with stronger negative functional connectivity between the right ventromedial prefrontal cortex and amygdala. Moreover, greater negative connectivity between these regions was associated with higher EQ-i and lower symptom severity on the PAI. As shown in the *Figure above*, mean emotional intelligence scores for the entire sample ($n = 65$) divided by terciles for Sleep Last Night, including Low Sleep (≤ 6.5 hours, $n = 22$), Moderate Sleep (6.6 – 7.9 hours, $n = 21$), and High Sleep (≥ 8 hours, $n = 22$). A) Analysis of variance indicated a significant main effect of sleep on scores on the Bar-On EQ-i ($p = .032$), with a significant difference between the High and Low Sleep groups. B) There was no main effect of sleep on the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT). *$p < .05$, corrected.

As evident in Figure 6, at right, effect sizes of the correlations between hours of self-reported sleep obtained the preceding night and scores on the Bar-On Emotional Intelligence Inventory (EQ-i) and the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT). Black bars: All scales of the EQ-i showed significant Pearson correlations with *Sleep Last Night*, whereas none of the MSCEIT scales showed significant correlations. Gray bars: Similar trends were observed after statistically controlling for insomnia complaints, but only Adaptability remained significant. *$p < .05$, **$p < .005$.

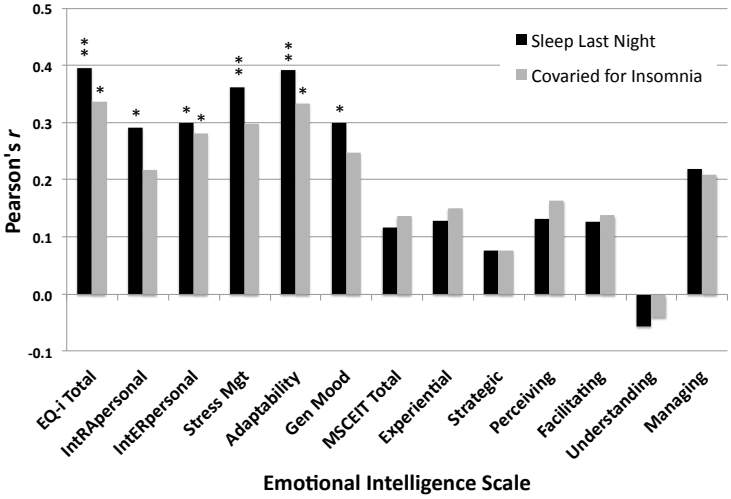
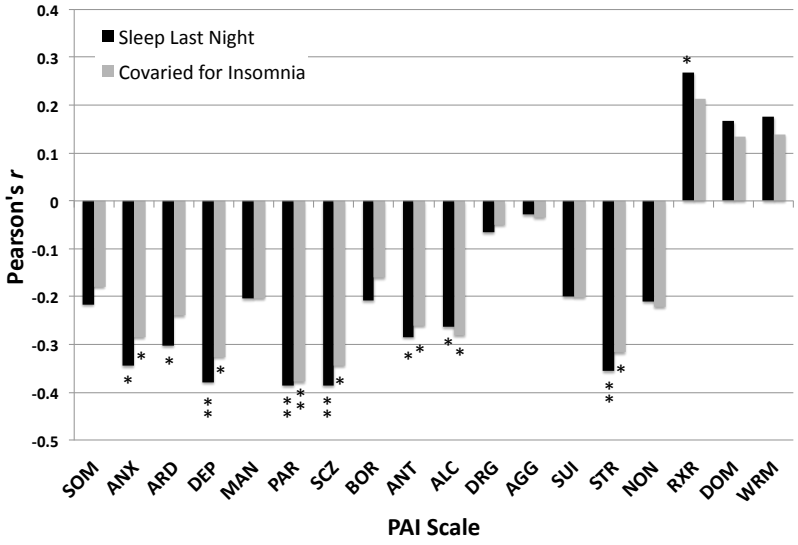
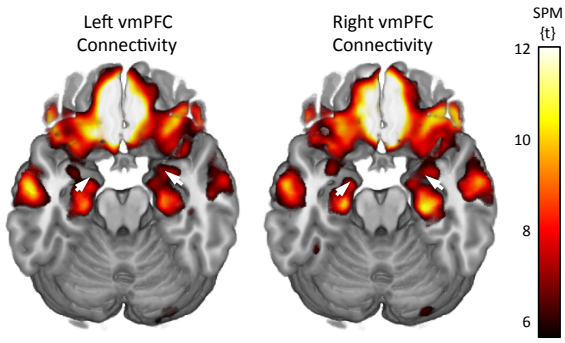


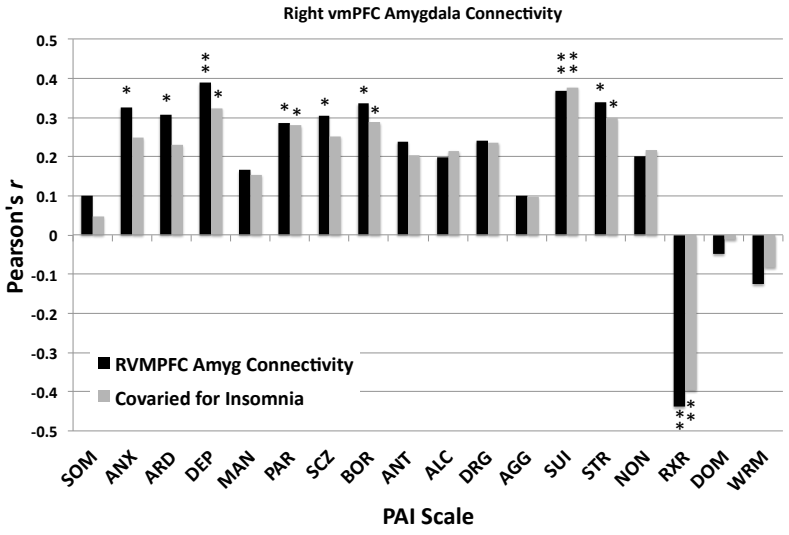
Figure at right shows the effect sizes of the correlations between hours of self-reported sleep obtained the preceding night and scores on the Personality Assessment Inventory (PAI). Black bars: Greater sleep the preceding night was associated with lower scores on several indices of psychopathology based on bivariate correlations. Gray Bars: Most of the correlations between *Sleep Last Night* and psychopathology remained significant after statistically controlling for insomnia complaints. *$p < .05$, **$p < .005$.



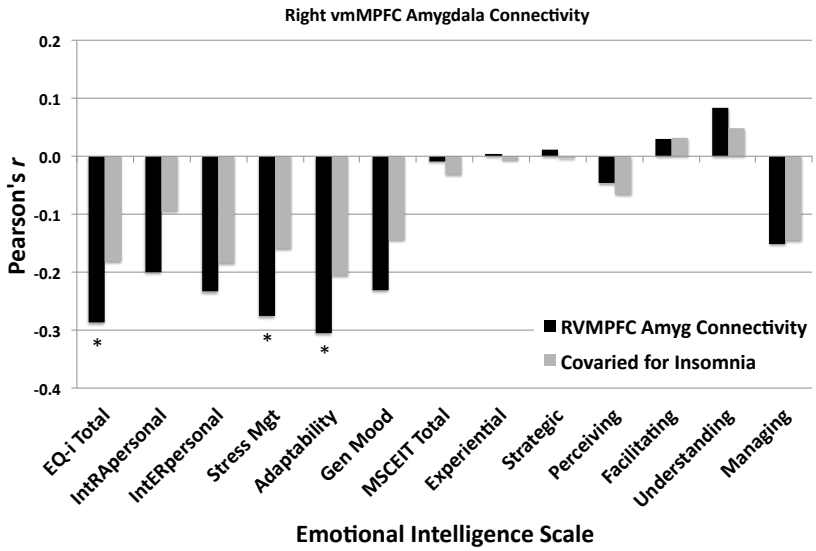
In the *Figure at right*, the functional connectivity maps are displayed for the left and right vmPFC seed regions of interest (ROIs). The brain images show axial slices that include both the vmPFC and amygdala regions. The white arrows show the location of the amygdala target ROIs. The maps were set to a whole brain threshold of $p < .05$, FWE corrected for multiple comparisons.



*The Figure*below shows that self-reported *Sleep Last Night* was significantly correlated with negative functional connectivity between the right ventromedial prefrontal cortex (vmPFC) and the right amygdala. The figure shows the cluster in the right amygdala [MNI coordinates: x = 24, y = 2, z = -22] that showed negative functional connectivity with the right vmPFC seed region as a function of greater reported sleep time. For visualization, the cluster is height thresholded at ($p <$ .001, uncorrected, spatial extent $p <$ .05 FWE-corrected). Figures are displayed in sagittal (top left), axial (bottom left), and coronal (top right) views. The scatterplot (bottom right) shows the linear relationship between hours of sleep and the connectivity values extracted from the displayed cluster.

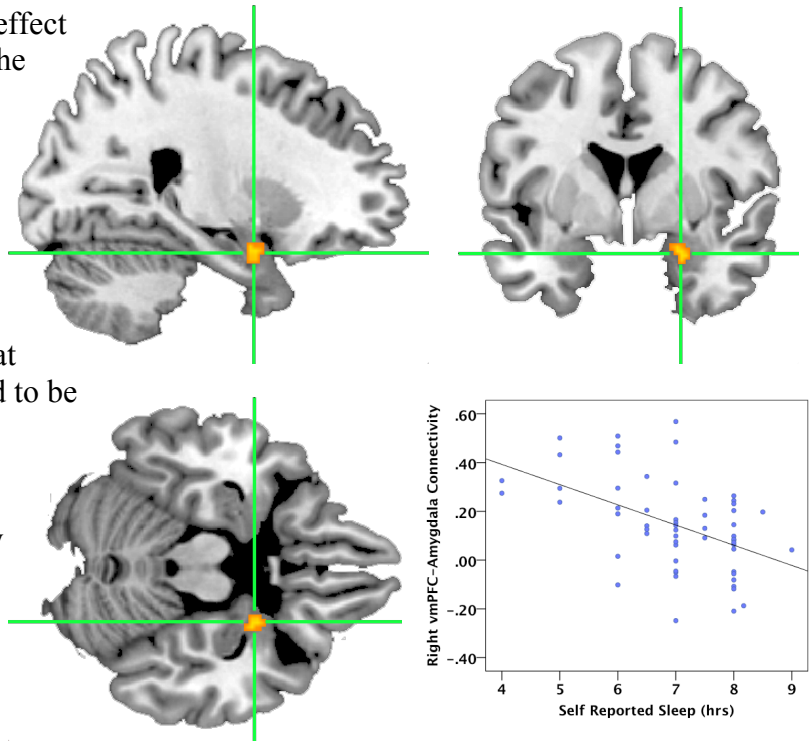


As evident in the *Figure at right*, the effect sizes show the correlations between the magnitude of ventromedial prefrontal cortex (vmPFC) – amygdala functional connectivity and scores on the Bar-On Emotional Intelligence Inventory (EQ-i) and the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT). Black bars: Total EQ-i, as well as composite scale scores for Stress Management and Adaptability showed significant negative



bivariate correlations with functional connectivity, indicating that higher emotional intelligence on the EQ-i was associated with greater *negative connectivity* between these two regions. In contrast, MSCEIT scales were not significantly correlated with functional connectivity between these two regions. Gray bars: After controlling for insomnia complaints, the observed correlations with emotional intelligence were no longer significant. *$p < .05$.

As evident in the Figure at right, the effect sizes show the correlations between the magnitude of ventromedial prefrontal cortex (vmPFC) – amygdala functional connectivity and scores on the Personality Assessment Inventory (PAI). Black Bars: Scores on several PAI scales showed positive bivariate correlations with functional connectivity, indicating that symptoms of psychopathology tended to be higher as these two regions covaried positively together, while psychopathology was reduced as these two regions covaried negatively with one another. Gray bars: Partial correlations controlling for insomnia complaints remained significant for the majority of PAI scales. *$p < .05$, **$p < .005$.

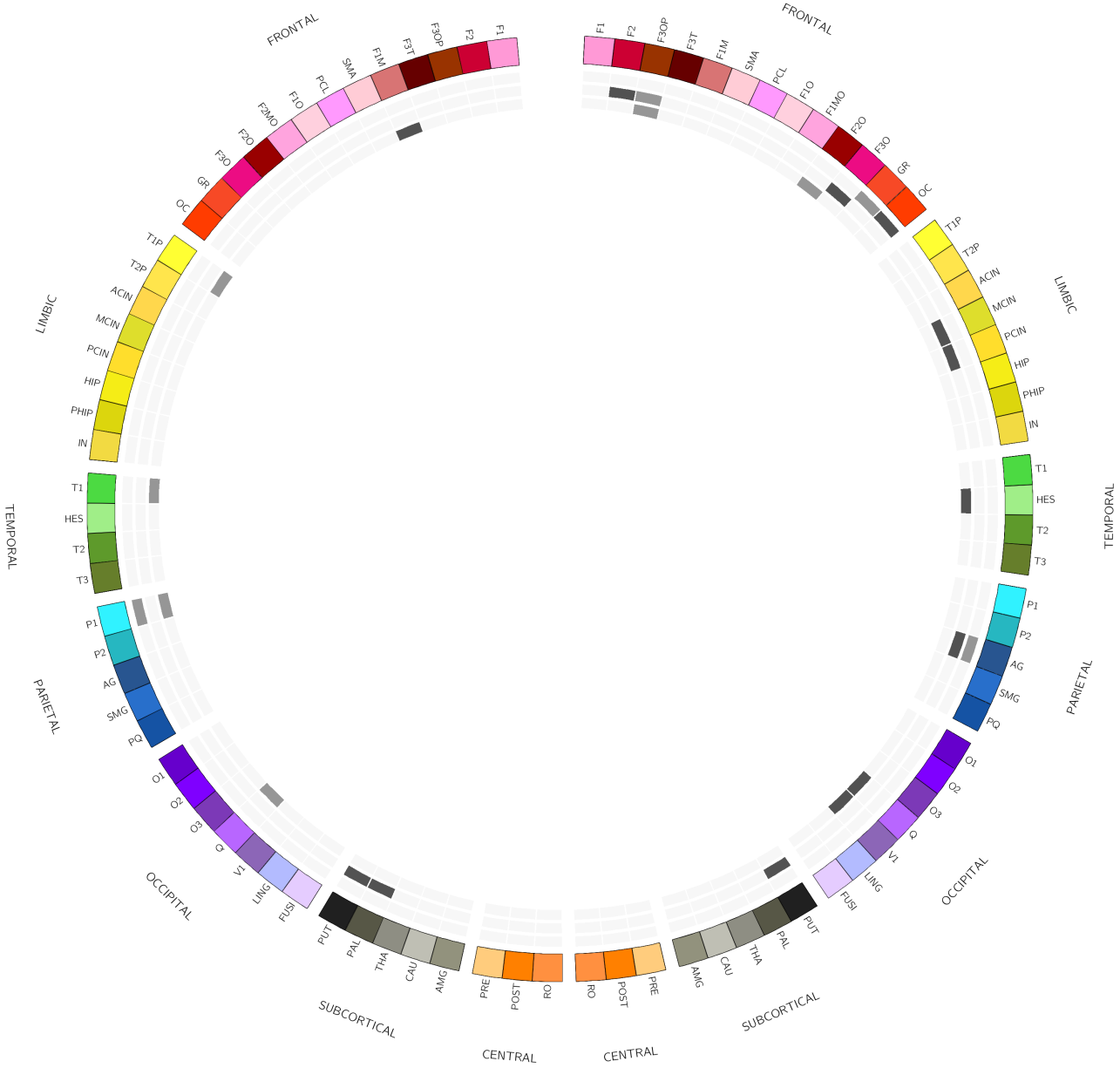


Self-reported sleep duration from the preceding night was significantly correlated with negative prefrontal-amygdala connectivity, perceived emotional intelligence, and the severity of subjective psychological distress. More sleep was associated with higher emotional and psychological strength. This finding was published in the journal *SLEEP* (Killgore, 2013).

Sleep Habits and Functional Brain Network Organization

Cognitive performance and emotional wellbeing vary with sleep, with insufficient sleep exerting powerful, yet heterogeneous effects on brain function. In this part of the project, we applied graph theory to resting-state functional MRI data to investigate patterns of functional brain network organization in relation to positive and negative habitual sleep balance (sleeping more and less than subjectively needed respectively). We analyzed resting-state functional MRI data from 42 healthy adults (positive habitual sleep balance: n=23; negative habitual sleep balance: n=19). Neuroimaging data were processed in SPM8 using the CONN, ART and Graph Theory toolboxes. Age, gender, IQ and hours of sleep obtained the night prior to MRI served as nuisance variables in all analyses.

The figure below shows group differences in FDA of topological network measures between *Positive Sleep Balance* and *Negative Sleep Balance* groups. Light gray depicts *Positive Sleep*

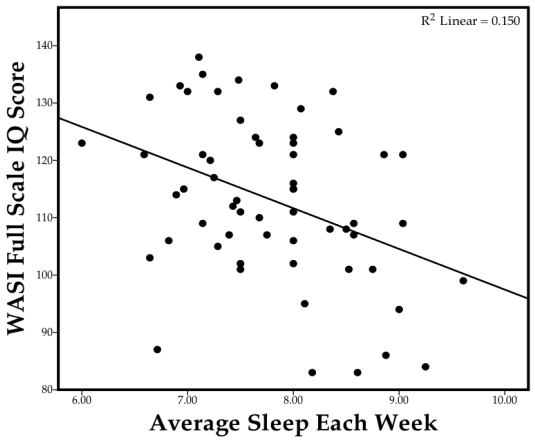


Balance > Negative Sleep Balance, dark grey depicts *Negative Sleep Balance > Positive Sleep Balance*. Across all regions, negative sleep balance was associated with greater characteristic path length compared to positive sleep balance, suggesting a less effective information transfer. Compared to positive habitual sleep balance, negative habitual sleep balance was associated with greater nodal degree and betweenness of several regions, including right anterior and posterior cingulate, bilateral putamen, right pallidum, right angular gyrus, and right dorsomedial prefrontal cortex, suggesting greater functional interaction and integration of these regions. In contrast, compared to negative habitual sleep balance, positive habitual sleep balance was associated with greater cluster coefficients of several brain regions, including the right ventromedial prefrontal cortex and right angular gyrus, indicating greater local functional segregation. Greater network interaction and integration in association with positive habitual sleep balance was indicated by greater nodal betweenness of the ventromedial prefrontal cortex and left superior temporal pole

among other regions. The right thalamus and bilateral amygdala emerged as functional hubs of network interaction and integration in the positive sleep balance group.

These findings suggest that sleep habits reflect in the organization of functional brain networks in such that brain regions involved in emotion regulation, gating of information and decision-making, including the amygdala, the thalamus and the ventromedial prefrontal cortex, are more integrated in the overall brain network if sufficient sleep is obtained on a habitual basis.

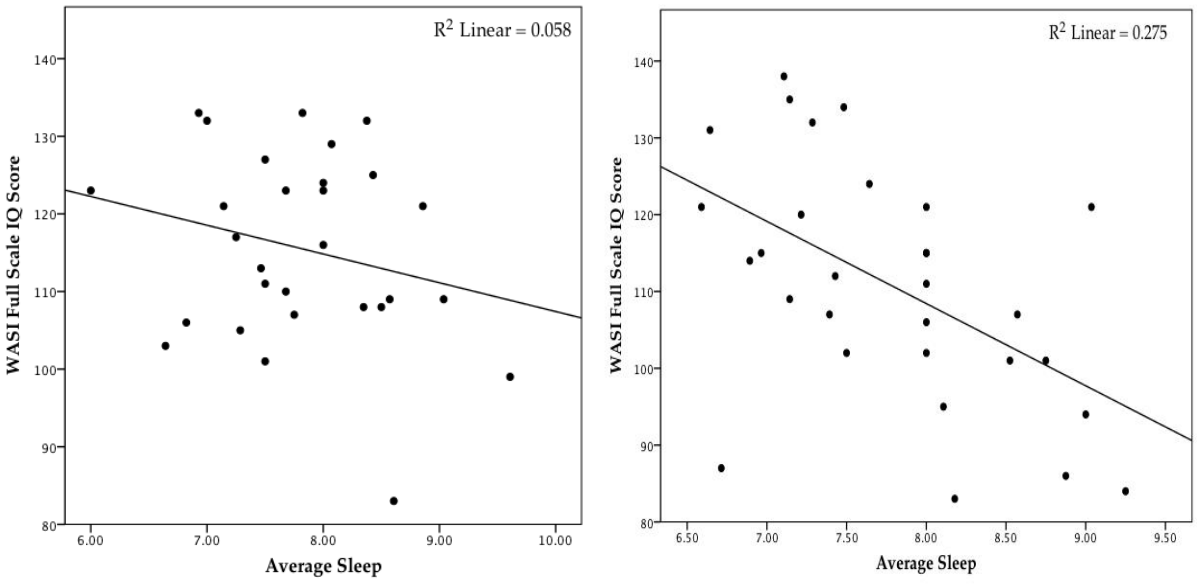
Sex Differences in the Association Between Sleep and Intelligence



Another goal of the current research program has been to understand the role of sleep in various aspects of Cognitive and Emotional Intelligence. Sleep deprivation studies have demonstrated decreased performance on cognitive tasks with decreased sleep. However, the Neural Efficiency Hypothesis suggests that individuals of higher intelligence may possesses more efficient brain organization and thus require less sleep time for recovery of cognitive functioning. This perspective is also consistent with the Cognitive Reserve Hypothesis, which suggests that individuals with greater cognitive reserve are more efficient in using brain networks, and will tolerate more brain damage or strain (e.g., sleep deprivation) before experiencing functional impairment. In the present set of analyses, we examined relationships among IQ, gender, and sleep patterns in our sample of healthy adults.

Subjects completed a measure of standard intelligence (Wechsler Abbreviated Scale of Intelligence; WASI), the Epworth Sleepiness Scale (ESS), and a questionnaire about their sleep habits. Pearson correlations were used to explore the relationship between the Full Scale IQ of the WASI and the average number of hours of sleep on typical week- and weekend nights, controlling for age, education, ESS, and Socioeconomic Status (SES). To calculate SES, data was obtained on mean inflation-adjusted 12-month household income and the percentage below the poverty line of the participant's neighborhood based on home address (U.S. Census Bureau, 2010).

WASI Full Scale IQ scores were negatively correlated with the average amount of sleep (r=-0.437, p<0.01) (*Figure below left*). When analyzed by gender, no significant correlation was found between FSIQ and average nightly sleep in males (r= -.122). In females, a significant negative correlation was observed (r= -0.617, p<0.01) (*Figure below right*). A Fisher's z-transformation revealed that the two correlations differ significantly (z=2.01, p=0.04, two-tailed).



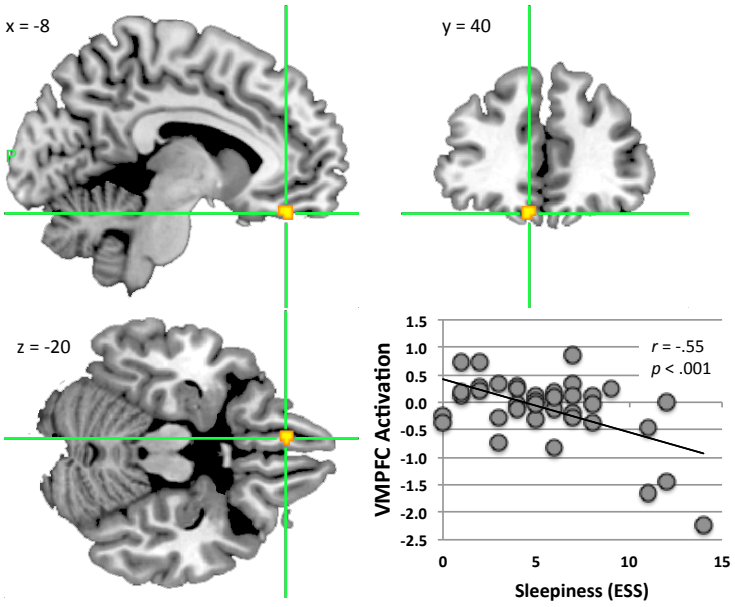
Findings suggest that females with greater intellectual ability obtain less sleep. Several possible explanations exist for this effect. The first supports the Neural Efficiency Hypothesis, indicating that individuals with higher cognitive functioning may also display higher efficiency in neuronal recovery during sleep. Another suggests that individuals with greater cognitive reserve may require less sleep to maintain the same level of functioning. Alternatively, individuals with shorter sleep duration may benefit from a longer period of wakefulness and greater opportunity for cognitive stimulation. Several key aspects may account for the gender disparity: previously identified differences in brain morphology, particularly the role of white and gray matter in intellectual functioning; differences in levels of hormones, such as testosterone; and societal and cultural pressures specific to each gender, which may play into the differences in sleep habits and cognitive functioning.

Daytime Sleepiness Alters the Temporal Frame of Emotionally Based Judgment Capacities

Sleep deprivation is associated with performance decrements on some measures of executive functioning. For instance, our prior work has shown that sleep deprivation results in altered decision making on the Iowa Gambling Task, an emotionally based judgment and decision-making task. However, it is unclear which component processes of the task may be driving the effect. In this analysis, Iowa Gambling task performance was decomposed using the Expectancy-Valence model. Participants (n = 55; 29 female, 26 male) ages 18-45 years were recruited via posted advertisements. Participants completed a neurocognitive and emotional task battery over the course of two testing days. On the first day, they completed an MRI scan and sleep questionnaires. They slept at home that night and were unmonitored. On the second day, they completed the IGT, as well as the Wechsler Abbreviated Scale of Intelligence (WASI: Pearson, 1999) to yield Full-Scale IQ (FSIQ). The total score on the ESS was used as the measure of daytime sleepiness. In addition, recent sleep debt was calculated by subtracting the hours that the participant reported sleeping prior to the first day of testing from the hours that the participant reported typically sleeping on weeknights (calculated as the difference between reported typical bedtime and wake time) (i.e., typical sleep minus recent sleep). Positive values

reflect recent sleep debt. For the IGT, trial-by-trial data were analyzed in Matlab R2012b using scripts to derive EV parameters provided by Eldad Yechiam (www.technion.ac.il/~yeldad/papers.html). For each participant, α, *w*, and *c* were derived using maximum likelihood methods. The variable G^2, reflecting goodness of the model fit over the baseline model, was also calculated (Busemeyer & Stout, 2002; Eldad Yechiam et al., 2005). For the baseline model, we employed a statistical model assuming constant choice probabilities across trials where behavior does not change based on trial-by-trial feedback (Busemeyer & Stout, 2002). Daytime sleepiness was not significantly associated with either the attention weight parameter (*w*), Spearman's *r* (30) = -0.173, *p* = 0.343, or the sensitivity parameter (*c*), Spearman's *r* (30) = -0.255, *p* = 0.159. However, greater daytime sleepiness was significantly correlated with higher values of the updating parameter, (*α*), Spearman's *r* (30) = 0.390, *p* = 0.027. Participants who reported higher levels of daytime sleepiness demonstrated reduced tendency to use information from outcomes that were more distant in time when making decisions. Recent sleep debt was also associated with higher values of the updating parameter (*α*), Spearman's *r* (30) = 0.413, *p* = 0.019, but not the attention weight (*w*) or sensitivity (*c*) parameters (*p*s > .05). In other words, recent sleep debt and greater daytime sleepiness were associated with higher scores on the updating parameter, which reflects the extent to which recent experiences are emphasized over remote ones. Findings suggest that the effects of insufficient sleep on IGT performance are due to shortening of the time horizon over which decisions are integrated. These findings may have important military (and clinical) implications in that individuals with sleep problems may not integrate more temporally distant information when making decisions. This could have a severe detrimental effect on mission success.

Daytime Sleepiness Affects Prefrontal Cortex Regulation of Food Intake



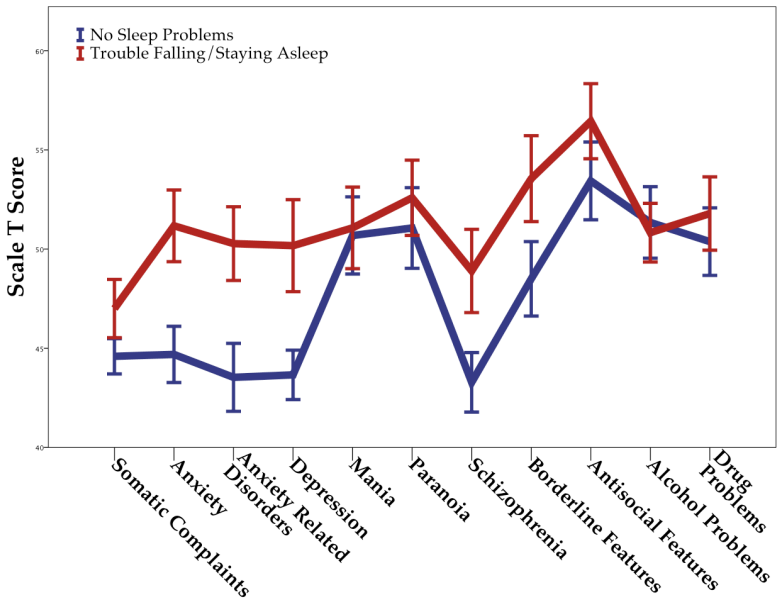
The recent epidemic of obesity corresponds closely with the decline in the average number of hours of sleep obtained nightly. Excess weight gain is a critical issue within the military and can affect mission readiness of servicemembers. While growing research suggests that sleep loss may affect hormonal and other physiological systems related to food intake, no studies have yet explored the role that sleepiness may play in reducing prefrontal inhibitory control over food intake. Because evidence suggests that women may be more prone to obesity and eating disorders, as well as more likely to suffer from sleep problems, we examined the relation between general daytime sleepiness, brain responses to food stimuli, and self-reported overeating separately for men and women. Thirty-eight healthy adults (16 women; 22 men) aged 18 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing pictures of high- and low-calorie

foods. Subjects completed the Epworth Sleepiness Scale (ESS) and provided a rating to the query "how often do you eat more than you intend to." Contrast images comparing brain activation derived from the high- versus low-calorie conditions were correlated voxel-wise with scores from the ESS in a second-level regression model, the output of which was used to predict self-reported overeating. As hypothesized, daytime sleepiness correlated with reduced activation in the ventromedial prefrontal cortex during perception of high- versus low-calorie food images. Moreover, activation within this cluster predicted overeating, but only for women. Findings suggest that normal fluctuations in sleepiness may be sufficient to affect brain regions important for regulating food intake, but that these effects may differ between men and women. These findings were published in the journal *NeuroImage* (Killgore, Schwab, Weber, et al., 2013).

Difficulty in Falling and Staying Asleep Linked to a Sub-Clinical Increase in Symptoms of Psychopathology.

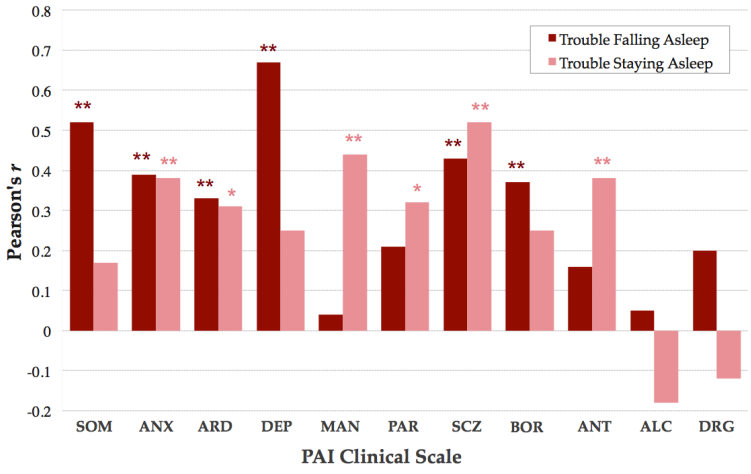
Sleep problems are linked with a broad spectrum of psychopathologies, particularly affective disorders. We have previously shown that laboratory sleep deprivation elicits significant increases in self-reported symptoms of psychopathology. Here we extend prior laboratory research to a non-laboratory naturalistic setting. We hypothesized that participants complaining of difficulties with sleep initiation or sleep maintenance would also score higher on measures of symptoms of psychopathology.

65 healthy adults completed a questionnaire on sleep habits and frequency experiencing trouble falling and/or staying asleep, along with the Personality Assessment Inventory (PAI) to assess clinical symptoms of psychopathology. A one-way MANOVA was used to assess differences in scores on PAI clinical scales between participants who had sleep difficulties and those that did not. Pearson correlations were used to evaluate the association between the frequency of estimated sleep disturbances per year and PAI scores.



Participants who endorsed sleep difficulties scored significantly higher ($p<0.05$) than those who did not on clinical scales measuring anxiety, anxiety-related disorders, depression, and schizophrenic symptoms. For these scales, further analysis of subscales was conducted using Bonferroni corrected one-way MANOVA. Subscale analyses revealed that scores on the Anxiety scale were elevated for Cognitive, Affective, and Physiological dimensions, while Anxiety-Related Disorders scores were driven predominantly by elevations in the Phobias subscale. Similarly, higher Depression scores among those with sleep

complaints were driven predominantly by the Cognitive and Physiological subscales, while the elevated scores on the Schizophrenia scale were driven mostly by greater Psychotic Experiences scores. For individuals endorsing sleep onset problems, the reported frequency of these experiences was significantly correlated ($p < .05$, Bonferroni corrected) with increased Somatic Complaints, Anxiety, Depression, Schizophrenia, and Borderline Features. On the other hand, for individuals who reported having trouble staying asleep, the reported frequency of insomnia-related complaints was significantly correlated ($p < .05$, Bonferroni corrected) with higher measures of Anxiety, Mania, Schizophrenia, and Antisocial Features.

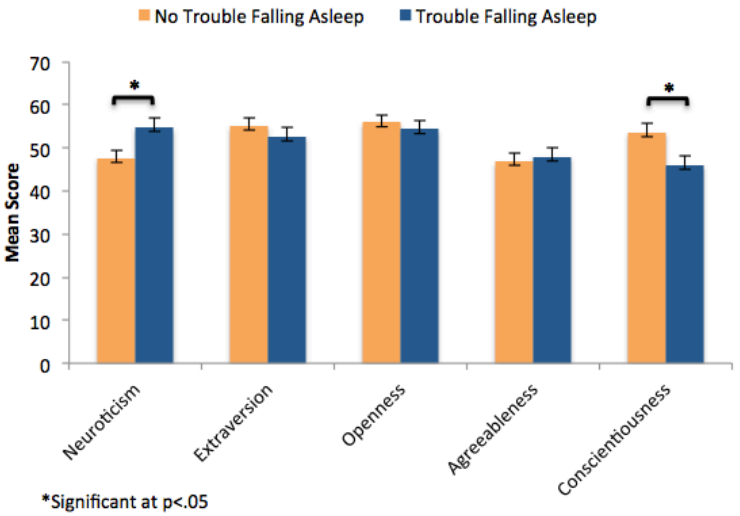


Difficulty with falling or staying asleep was associated with sub-clinical elevations in symptoms of psychopathology. Individuals reporting higher frequencies of sleep disturbances presented increased symptom severity across a number of clinical scales, suggesting a linear relationship between sleep disruption and psychopathological symptom complaints at a subclinical level. Furthermore, differences in associations were found between individuals who have trouble falling asleep and staying asleep. While causal directionality cannot be inferred, these findings support the notion that sleep plays a significant role in emotional functioning and may be an underlying risk factor for affective disorders. These findings were published in the journal *Experimental Brain Research* (Tkachenko et al., 2014).

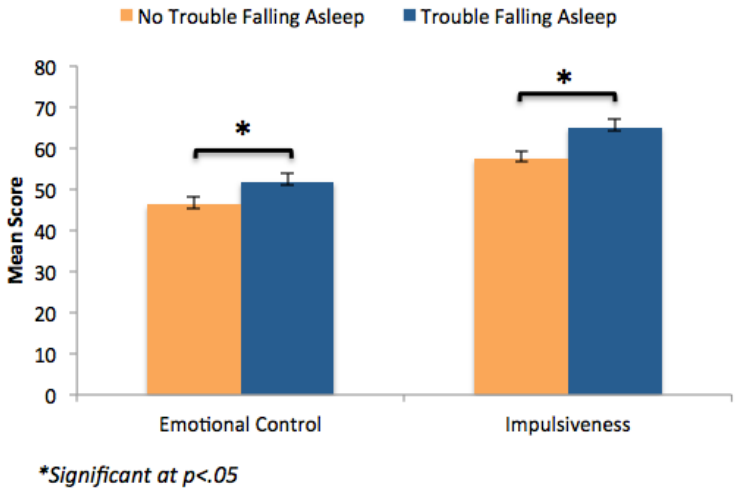
Linking Sleep Initiation Trouble to Neuroticism, Emotional Control, and Impulsiveness

Difficulty initiating sleep is a key component of insomnia and a common symptom of many psychiatric disorders. Certain combinations of personality, cognitive and emotional factors may differentiate individuals with sleep onset problems from normal sleepers. For example, neuroticism and impulsiveness are related to the subjective experience of insomnia and difficulty falling asleep. The cognitive model of insomnia argues that negatively toned cognitive activity triggers arousal and distress, which induces a pattern of selective attention to sleep loss cues, erroneous beliefs about sleep loss, and maladaptive coping strategies that perpetuate sleep disturbance. Both neuroticism and impulsiveness may be accompanied by counterproductive methods to control negative thoughts and emotions that interfere with sleep. Neuroticism and cognitive and emotional arousal are vulnerability factors for insomnia. Furthermore, dysfunctional thought control strategies mediate the association between impulsiveness and insomnia. It was hypothesized that people who reported trouble falling asleep would have a higher degree of neuroticism, emotional control, and impulsiveness than normal sleepers and that minutes to fall asleep would be associated with these personality and cognitive factors.

Sixty-one healthy adults (31 men) aged 18 to 45 completed a questionnaire about typical sleep habits, indicating whether they had trouble falling asleep and how many minutes they took to fall asleep, the Revised NEO Personality Inventory (NEO-PI-R), the Courtauld Emotional Control Scale (CECS), and the Barratt Impulsiveness Scale (BIS). A multivariate analysis of variance was used to determine whether people who reported trouble falling asleep (N=26) differed from those who did not have trouble falling asleep (N=35) in terms of neuroticism, emotional control, and impulsiveness. Additionally, correlation analyses were used to examine relationships between minutes to fall asleep on weekdays and degree of neuroticism, emotional control, and impulsiveness.



People who reported trouble falling asleep differed from those who did not in terms of neuroticism, emotional control, and impulsiveness, (MANOVA, p=.015). Univariate between-groups comparisons revealed that trouble sleeping was associated with greater neuroticism (p=.013), emotional control (p=.042) and impulsiveness (p=.008). Minutes to fall asleep on weekdays was significantly positively associated with neuroticism (r=.475, p<.001) and impulsiveness (r=.394, p=.002), but not emotional control (p=.196).

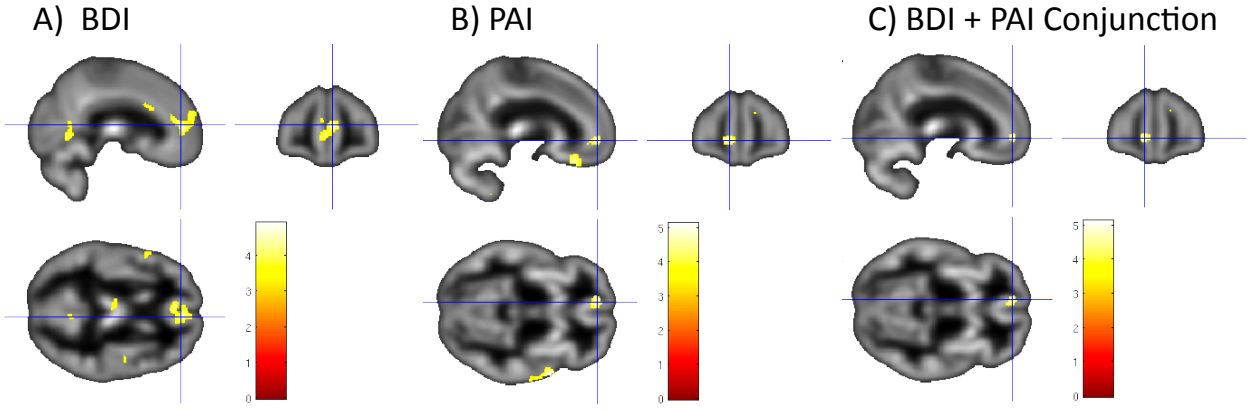


Neuroticism, emotional control, and impulsiveness was higher in people reporting trouble falling asleep when compared to normal sleepers. Likewise, minutes to fall asleep was also associated with neuroticism and impulsiveness. These findings indicate that trouble falling asleep is related to degree of characteristic negative affect, the extent to which individuals are unable to cope with their negative emotions, and impulsiveness. Findings may have implications for treatment of sleep initiation trouble, mood disturbance, and impulsive behavior. These findings were published in the *Journal of Sleep Disorders: Treatment & Care* (Preer, Tkachenko, Gogel, Bark, & Killgore, 2014).

Cerebral Cortex Volume and Mild Depression

Studies investigating structural brain abnormalities in depression have typically employed a categorical rather than dimensional approach to depression (i.e., comparing subjects with DSM-defined Major Depressive Disorder [MDD] vs. healthy controls). More recently, the National Institute of Mental Health (NIMH), through their Research Domain Criteria (RDoC) initiative, has encouraged a dimensional approach to the study of psychopathology as opposed to an overreliance on DSM-based diagnostic categories. Moreover, subthreshold levels of depressive symptoms (i.e., severity levels below DSM criteria) have been found to be associated with a range of negative outcomes, yet have been relatively neglected in neuroimaging research.
To examine the extent to which depressive symptoms - even at subclinical levels - are linearly related to gray matter volume reductions in theoretically important brain regions, we employed whole-brain voxel-based morphometry (VBM) in a sample of 54 participants.

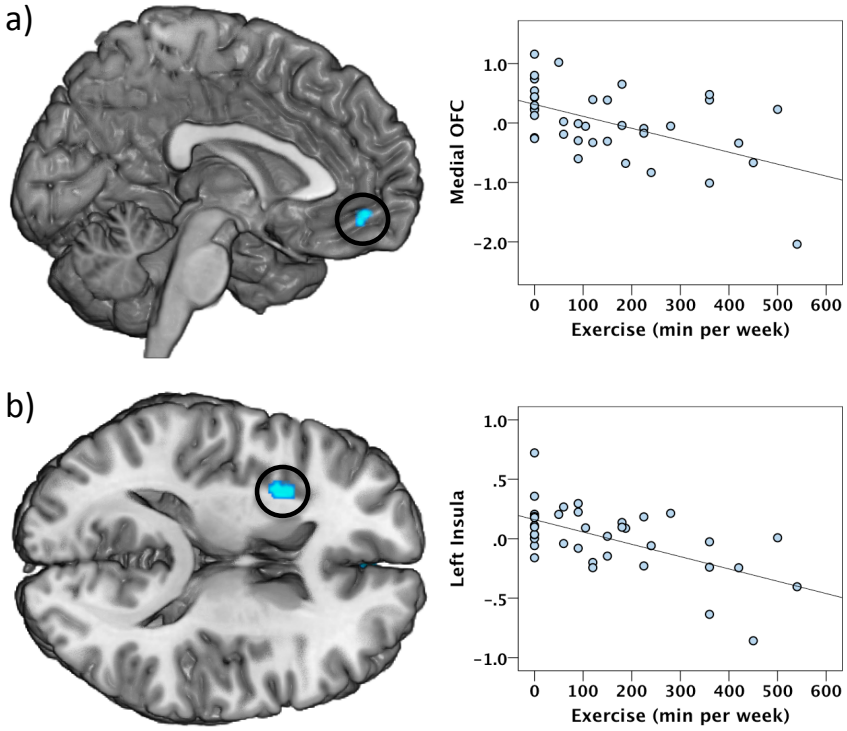
The severity of mild depressive symptoms, even in a subclinical population, was associated with reduced gray matter volume in the orbitofrontal cortex, anterior cingulate, and thalamus. A conjunction analysis revealed concordance across two separate measures of depression. Specifically, relatively higher BDI scores were associated with reduced gray matter volume in 16 clusters, including (i) bilateral anterior cingulate, bilateral medial frontal cortex and left medial orbitofrontal cortex, (ii) bilateral anterior/mid cingulate, (iii) left thalamus and (iv) left insula (see Figure A below). Relatively higher PAI-Depression scores were associated with reduced gray matter volume in 9 clusters, including (i) left anterior cingulate and left medial orbitofrontal cortex (see Figure B below), (ii) bilateral medial orbitofrontal cortex and (iii) bilateral thalamus. The conjunction between the two primary analyses was used to identify the regions showing common overlap between the gray matter volume correlations for the two depression measures used in the current study. This analysis showed that the BDI and PAI-DEP scores were both associated with reduced gray matter volume in 4 common regions, including (i) left medial orbitofrontal cortex and anterior cingulate (see Figure C below), (ii) left thalamus, (iii) right superior medial frontal gyrus/superior frontal gyrus, and (iv) right superior temporal gyrus extending to the superior temporal pole.



Reduced gray matter volume in theoretically important brain regions can be observed even in sample that does not meet DSM criteria for MDD, but who nevertheless report relatively elevated levels of depressive symptoms. Overall, and consistent with NIMH's RDoC initiative, these findings highlight the limitations of restricting the study of abnormal cognitive, emotional and behavioral processes in depression to DSM-based categorical comparisons, and the need for

additional research using dimensional conceptual and analytic approaches. These findings were published in the journal *Psychological Medicine* (Webb, Weber, Mundy, & Killgore, 2014).

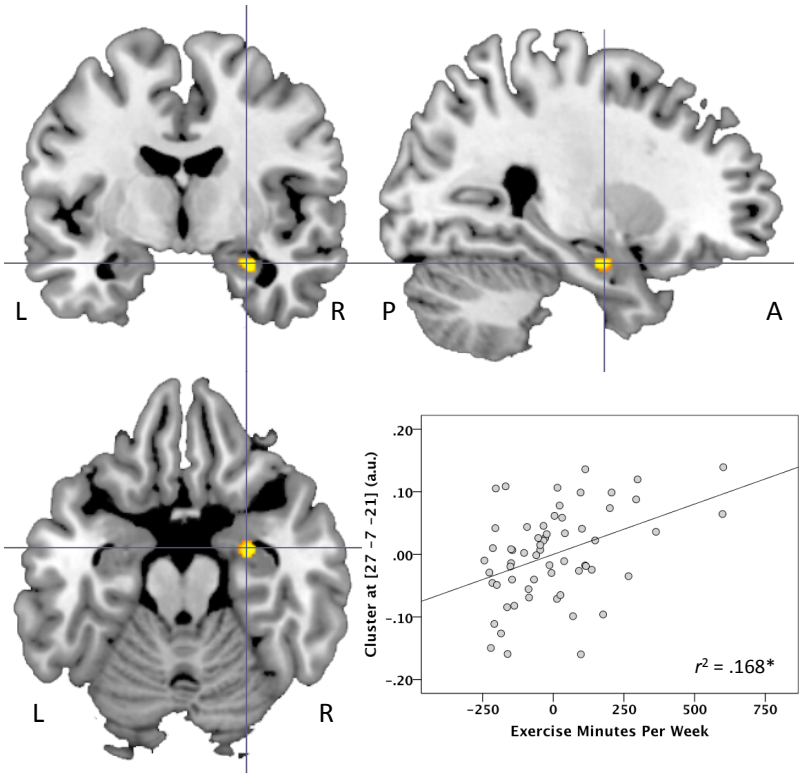
Physical Exercise and Brain Responses to Images of High Calorie Food



Physical exercise has many health benefits, including improved cardiovascular fitness, lean muscle development, increased metabolism, and weight loss, as well as positive effects on brain functioning and cognition. Recent evidence suggests that regular physical exercise may also affect the responsiveness of reward regions of the brain to food stimuli. We examined whether the total number of minutes of self-reported weekly physical exercise was related to the responsiveness of appetite and food reward related brain regions to visual presentations of high- and low-calorie food images during functional magnetic resonance imaging (fMRI). Secondly, we examined whether such responses would correlate with self-reported food preferences. While undergoing scanning, 37 healthy adults (22 men) viewed images of high- and low-calorie foods and provided desirability ratings for each food image. The correlation between exercise minutes per week and brain responses to the primary condition contrast (high-calorie > low-calorie) was evaluated within the amygdala, insula, and medial orbitofrontal cortex (mOFC), brain regions previously implicated in responses to food images. As evident in the figure, higher levels of exercise were significantly correlated with lower responsiveness within the mOFC and left insula to high-calorie foods. Furthermore, activation of these regions was positively correlated with preference ratings for high-calorie foods, particularly those with a savory flavor. These findings suggest that physical exercise may be associated with reduced activation in food-responsive reward regions, which are in turn associated with reduced preferences for unhealthy high-calorie foods. Physical exercise may confer secondary health benefits beyond its primary effects on cardiovascular fitness and energy expenditure. These findings were published in the journal *NeuroReport* (Killgore, Kipman, et al., 2013).

Physical Exercise and Brain Volume in Healthy Adults

Physical activity facilitates neurogenesis of dentate cells in the rodent hippocampus, a brain region critical for memory formation and spatial representation. Recent findings in humans also suggest that aerobic exercise can lead to increased hippocampal volume and enhanced cognitive functioning in children and elderly adults. However, the association between physical activity and hippocampal volume during the period from early adulthood through middle age has not been effectively explored. Here, we correlated the number of minutes of self-reported exercise per week with gray matter volume of the hippocampus using voxel-based morphometry (VBM) in 61 healthy adults ranging from 18 to 45 years of age. After controlling for age, gender, and intracranial volume, total minutes of weekly exercise correlated significantly with volume of the right hippocampus (see Figure). Findings highlight the importance of regular physical exercise to brain structure during early to middle adulthood. These findings were published in the journal *Scientific Reports* (Killgore, Olson, & Weber, 2013).

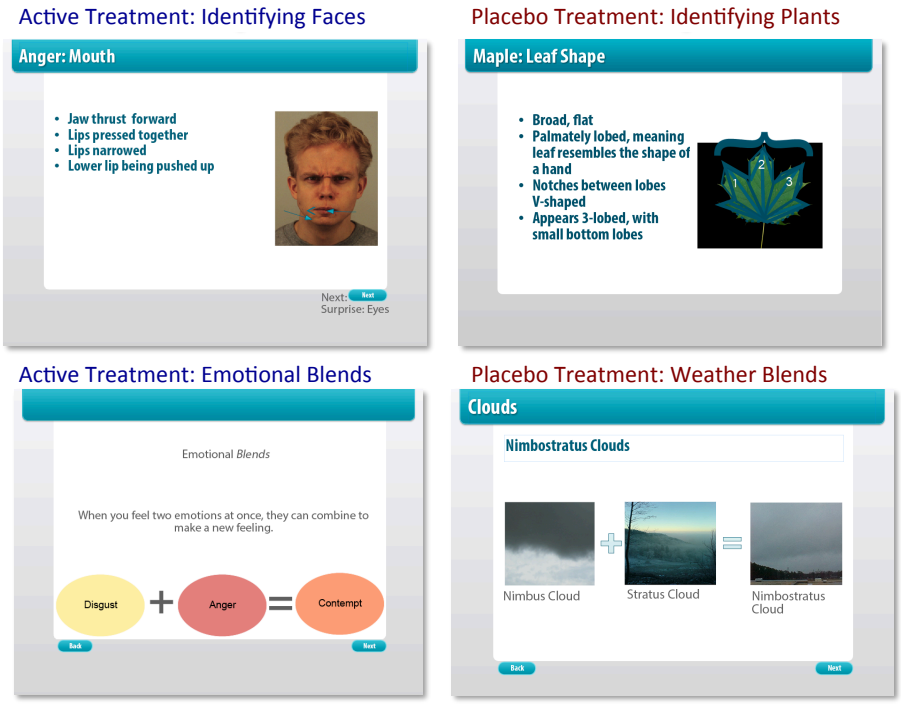


<u>Research Findings Pertaining to the Final Year SOW Modification: Development of an EI Training Program (Study 2)</u>

Although not part of the initially funded study, a modification to the SOW was made during our no-cost extension after the third year of funding. This modification involved 1-year of additional funding to develop a pilot EI Training Program based on the Mayer-Salovey-Caruso Model of Emotional Intelligence.

We have now completed the development and validation of the pilot version of an internet-based EI training program designed to enhance a core set of emotional skills rapidly and with minimal time investment. Over the past year, we have collected preliminary data demonstrating the efficacy of this program in improving four major emotional ability domains, including emotional perception, understanding the complexities of emotion, management and control of emotions, and flexible use of emotion to enhance cognition. Together, these four abilities constitute the construct of Emotional Intelligence (EI) (Mayer, Salovey, & Caruso, 2002). Briefly, EI can be conceptualized as a core feature of emotional resilience along with mental and social fitness, as it

includes capacities to perceive and understand emotions in oneself and others, to manage and regulate emotions effectively, and to use that information to solve problems and to make better decisions. As described in the previous sections, our cognitive and neuroimaging study has shown that EI abilities are unique from other measures of cognitive intelligence and personality (Webb et al., 2013), and are associated with indices of brain health, including greater cortical volume (Killgore et al., 2012) and functional activation (Killgore, Schwab, Tkachenko, et al., 2013) within a cortico-limbic brain network that integrates emotion, cognition, and interoceptive sensory experiences. Moreover, we have shown that EI can serve as a moderating variable that may minimize the association between predisposing factors and the expression of anxiety symptoms (Preer et al., 2014), as well as protecting against degradation of interpersonal problem-solving and decision-making caused by external stresses such as sleep loss (Killgore et al., 2007). Based on these behavioral and neuroimaging findings, we developed and validated a brief EI training program based on the 4-Branch Model proposed by Mayer, Salovey, & Caruso (2002).

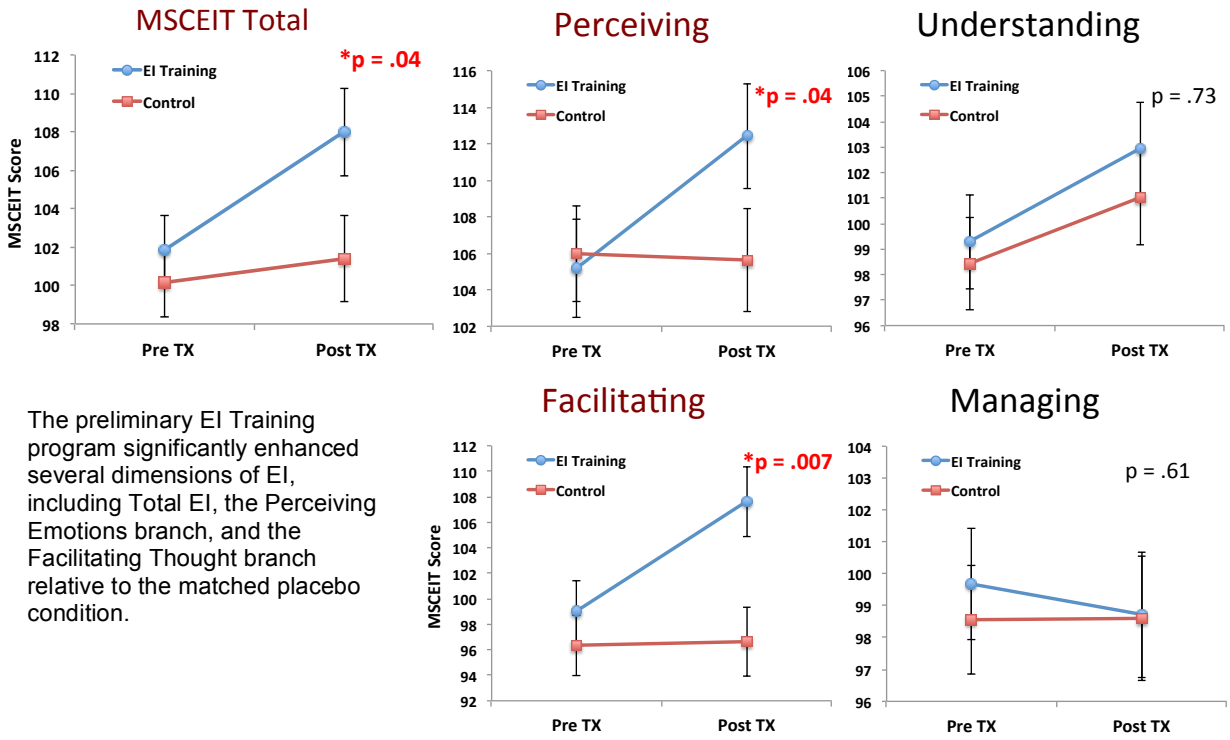


The preliminary version of the training program consists of six online modules, each requiring about 30-minutes to complete (see Figure below). Each module was developed based on published empirical research addressing a specific skill or ability to be trained. These six self-contained training lessons include: 1) an introductory module, 2) a module to train emotional perception (e.g., decoding faces), 3) a module to enhance understanding of the complexities of emotions, their antecedents, and their logical progression over time, 4) a module to train cognitive-behavioral techniques for managing emotions (e.g., reappraising situations; mindfulness breathing), 5) a module to train methods for facilitating thought through emotions (e.g., modulating arousal to enhance performance), 6) a review module to reinforce key elements of the training. Each lesson was also followed by a brief homework assignment to consolidate major points and generalize learning to each participant's own situation.

We have conducted a preliminary validation and efficacy study of the previously described EI training program compared to a tightly matched placebo-control training program (see Figure below). Participants in each group (EI $n = 29$; Placebo $n = 30$) were closely matched in terms of age, gender, education, intelligence (IQ) and EI (MSCEIT). As shown below, participants underwent a four hour baseline assessment session, and were then randomly assigned to receive either the EI Training Program or matched Placebo Control Training Program over a 3-week period. At the end of the training period, all participants returned to the lab for a follow-up assessment.

	EI Training	Control Group
N	29	30
Age	27.9	27.2
% Males	41.9%	48.4%
Education	15.8 (SD = 1.8)	15.5 (SD = 2.2)
IQ	113.2 (SD = 13.7)	113.5 (SD = 11.8)
MSCEIT	102.4 (SD = 10.9)	99.5 (SD = 11.3)

As evident in the figure below, this program significantly enhanced Total EI scores relative to the placebo condition. Further analysis of the four branches of EI revealed that the preliminary training program was effective at improving scores on the Perceiving Emotions and Facilitating Thought branches of EI, but not at improving the Understanding Emotions and Managing Emotions branches of EI (see figure below). These preliminary findings are encouraging and suggest that the program was effective in increasing some EI capacities by over half a standard deviation in only a few short lessons. We propose to develop a training program that will increase performance in all four domains.

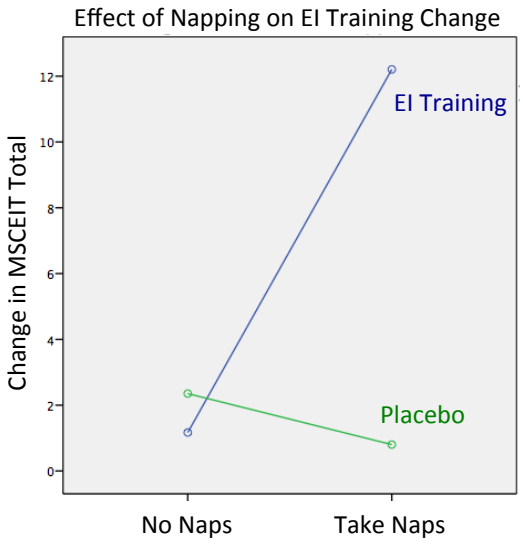


The preliminary EI Training program significantly enhanced several dimensions of EI, including Total EI, the Perceiving Emotions branch, and the Facilitating Thought branch relative to the matched placebo condition.

Moderating Variables

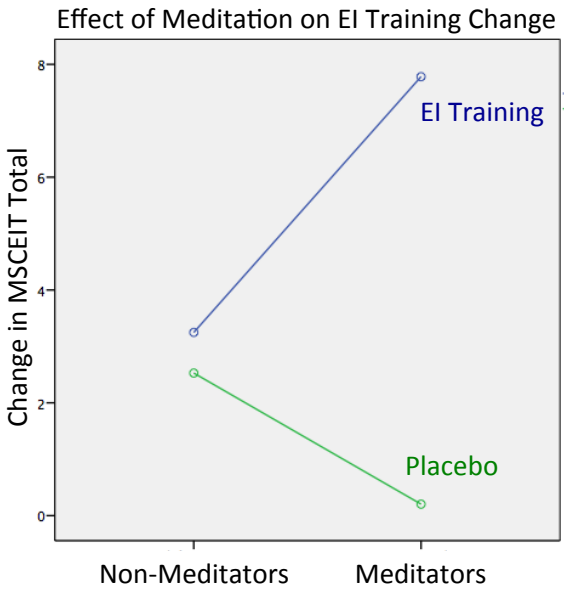
In addition to the primary analysis showing the effectiveness of the program for enhancing several aspects of emotional intelligence, we have also examined several potential moderators that may affect the outcome of the preliminary treatment program.

First, because considerable evidence suggests that sleep plays a crucial role in emotional functioning and memory consolidation, we expected that daytime napping may play an important moderating effect on the effectiveness of the EI Training program. Specifically, we hypothesized that regular daytime nappers would show greater improvement from the training than non-nappers. Sixty-two healthy 18-50 year olds (31 men) were randomized to receive either a 6-lesson on-line EI-training program over a 3-week period, or a matched placebo training program with similar intellectual challenge and activities. Although napping was not controlled, twenty-eight participants (16 men) also reported voluntarily taking naps at least one or more times per week throughout the course of the training. As a measure of EI, participants completed the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) at baseline and after completion of the program. As



shown in the figure, a 2 x 2 ANOVA yielded a significant interaction between EI training and napping conditions with regard to improvement in EI scores ($p = .006$). Whereas EI training was enhanced among nappers relative to non-nappers, the placebo condition was not effective at changing EI scores, regardless of napping. The findings suggest that inclusion of napping for individuals in the online training program may provide an increase in the efficacy of EI training over the duration of the program. Napping may improve memory consolidation, emotional regulation, or cognitive performance during the training.

Second, since past research has shown a relationship between the practice of meditation and increased internal awareness, we hypothesized that meditators would show greater EI increases than non-meditators during EI training. The same sixty-two healthy adults (31 men), ranging in age from 18-50 years, were also queried about their practice of medication. The sample included 28 individuals who endorsed that they regularly practice meditation, and 34 who did not meditate regularly. Interestingly, individuals who practiced meditation reported higher occurrences of sleep problems than controls. After accounting for the variance associated with sleep problems, a 2 x 2 ANOVA showed a significant interaction between meditation group and improvement in EI scores ($p = .04$), with meditators showing greater benefit from the EI training program than non-meditators. This relationship may be the result of individuals attempting to mitigate their sleep problems by engaging in meditation. Promoting introspective behaviors (i.e. engaging in meditation) may augment EI training in a manner that increases its effectiveness, regardless of whether individuals are experiencing the detrimental effects incurred by interruptions in sleep.



Training Program: Future Directions

The preliminary data regarding the effectiveness of the EI training program is highly encouraging and suggests that EI skills can be modified via a very brief (4 to 6 hours time investment) internet-based training program. Our EI training program is brief, web-based, easily accessible, and sufficiently focused such that it could be completed by large numbers of military personnel with minimal time burden, leading to rapid enhancement of these critical emotional capacities. The effect sizes demonstrated by the pilot version of the program are larger than those reported for other programs currently in use by the military. With further refinement and validation, this program will likely have a tremendous positive impact on the emotional health and resilience of servicemembers and their families by enhancing core EI capacities that are vital to resilience. To that end, we have recently submitted a grant proposal to the FY14 MOMRP PH/TBI PHRA announcement to request funding support for an extensive follow on study that would substantially refine the current pilot version of the program and validate it in military populations. We believe that the proposed follow-on study will produce an optimized EI

Training program that will be easily disseminated to servicemembers and which will provide meaningful enhancement of their EI skills. The proposed program may have far reaching consequences for protecting the mental health of warfighters and their families.

KEY RESEARCH ACCOMPLISHMENTS:

INITIAL NEUROIMAGING STUDY-

- 70 participants were enrolled, and the study is closed to new enrolment.
- 65 participants completed scanning/study procedures, providing usable data.
- Primary data analysis is complete, but secondary will continue after the funding period.
- 102 conference abstracts published/presented overall during the course of the study.
- 22 peer-reviewed articles published during the course of the study

SOW MODIFICATION: DEVELOPMENT OF EI TRAINING PROGRAM-

- All training materials have been researched, developed, proofread, and programmed into the web-presentation system.
- A secure web-server was established.
- 62 participants completed the training programs and all pre- and post-testing.
- Preliminary data have been analyzed.
- 5 conference abstracts have been submitted on the recent findings.

REPORTABLE OUTCOMES:

As of the date of this report, this project has yielded a total of ***107 published scientific abstracts*** and ***22 peer reviewed journal articles***. The following is a list of publications produced by the present award, and which are available in the Appendix:

Published Peer-Reviewed Journal Articles (22 total)

1. **Killgore, WD**, Weber, M, Schwab, ZJ, DelDonno, SR, Kipman, M, Weiner, MR, & Rauch, SL. Grey matter correlates of trait and ability models of emotional intelligence. Neuroreport 23, 551-555, 2012.

2. **Killgore, WD**, Schwab, ZJ, Kipman, M, DelDonno, SR, Weber, M. Voxel-based morphometric grey matter correlates of daytime sleepiness. Neurosci Lett, 518(1), 10-13, 2012.

3. **Killgore, WD**, Schwab, ZJ, & Weiner, MR. Self-reported nocturnal sleep duration is associated with next-day resting state functional connectivity. Neuroreport, 23, 741-745, 2012.

4. **Killgore, WD**, & Schwab, ZJ. Sex differences in the association between physical exercise and cognitive ability. Perceptual and Motor Skills, 115, 605-617, 2012.

5. Kipman, M, Weber, M, Schwab, ZJ, DelDonno, SR, & **Killgore, WD**. A funny thing happened on the way to the scanner: Humor detection correlates with gray matter volume. Neuroreport, 23, 1059-1064, 2012.

6. **Killgore, WD**, Schwab, ZJ, Weber, M, Kipman, M, DelDonno, SR, Weiner, MR, & Rauch, SL. Daytime sleepiness affects prefrontal regulation of food intake. NeuroImage, 71, 216-223, 2013.

7. **Killgore, WD**, Schwab, ZJ, Kipman, M, DelDonno, SR, & Weber, M. Insomnia-related complaints correlate with functional connectivity between sensory-motor regions. Neuroreport, 24, 233-240, 2013.

8. Weber, M, Webb, CA, DelDonno, SR, Kipman, M, Schwab, ZJ, Weiner, MR, & **Killgore, WD**. Habitual 'Sleep Credit' is associated with greater gray matter volume of the medial prefrontal cortex, higher emotional intelligence, and better mental health. Journal of Sleep Research, 22, 527-534, 2013.

9. **Killgore, WD**, Schwab, ZJ, Tkachenko, O, Webb, CA, DelDonno, SR, Kipman M, Rauch SL, and Weber M. Emotional intelligence correlates with functional responses to dynamic changes in facial trustworthiness. Social Neuroscience, 8, 334-346, 2013.

10. **Killgore, WD**. Self-reported sleep correlates with prefrontal-amygdala functional connectivity and emotional functioning. Sleep, 36, 1597-1608, 2013.

11. **Killgore, WD**, Kipman, M, Schwab, ZJ, Tkachenko, O, Preer, L, Gogel, H, Bark, JS, Mundy, EA, Olson, EA, & Weber, M. Physical exercise and brain responses to images of high calorie food. Neuroreport, 24, 962-967, 2013.

12. **Killgore, WD**, Weber, M, Schwab, ZJ, Kipman, M, DelDonno, SR, Webb, CA, & Rauch, SL. Cortico-limbic responsiveness to high-calorie food images predicts weight status among women. International Journal of Obesity, 37, 1435-1442, 2013.

13. Webb, CA, Schwab, ZJ, Weber, M, DelDonno, SR, Kipman M, Weiner, MR, & **Killgore WD**. Convergent and divergent validity of integrative versus mixed model measures of emotional intelligence. Intelligence, 41, 149-156, 2013.

14. **Killgore, WD,** Olson, EA, & Weber, M. Physical exercise habits correlate with gray matter volume of the hippocampus in healthy humans. Scientific Reports, 3, 3457, doi: 10.1038/srep0347, 2013.

15. Cohen-Gilbert, JE, **Killgore, WD**, White, CN, Schwab, ZJ, Crowley, DJ, Covell, MJ, Sneider, JT, & Silveri, MM. Differential influence of safe versus threatening facial expressions on decision-making during an inhibitory control task in adolescence and

adulthood. Developmental Science, 17, 212-223, 2014.

16. Preer, L, Tkachenko, O, Gogel, H., Bark, JS, & **Killgore, WD**. Personality traits associated with sleep initiation problems. Journal of Sleep Disorders: Treatment and Care, 3, 1-5, doi:10.4172/2325-9639.1000127, 2014.

17. Tkachenko, O, Olson, EA, Weber, M, Preer, LA, Gogel, H, & **Killgore, WD.** Sleep difficulties are associated with elevated symptoms of psychopathology. Experimental Brain Research, 232, 1567-1574, 2014.

18. Cui, J., Olson, EA, Weber, M, Schwab, ZJ, Rosso, SL, & **Killgore, WD**. Trait emotional suppression is associated with increased activation of the rostral anterior cingulate cortex in response to masked angry faces. NeuroReport, 25, 771-776, 2014.

19. Webb, CA, DelDonno, S, & **Killgore, WD**. The role of cognitive versus emotional intelligence in Iowa Gambling Task performance: What's emotion got to do with it? Intelligence, 44, 112-119, 2014.

20. **Killgore WD**, & Gogel, H. The Design Organization Test (DOT): Further Demonstration of Reliability and Validity as a Brief Measure of Visuospatial Ability. Applied Neuropsychology: Adult, 21, 297-309, 2014.

21. Webb, CA, Weber, M, Mundy, EA, & **Killgore, WD**. Reduced gray matter volume in the anterior cingulate, orbitofrontal cortex and thalamus as a function of mild depressive symptoms: A voxel-based morphometric analysis. Psychological Medicine, 44, 2833-2843, 2014.

1. Olson, EA, Weber, M, Rauch, SL, & **Killgore, WD**. Daytime sleepiness is associated with reduced integration of temporally distant outcomes on the Iowa Gambling Task. Behavioral Sleep Medicine (in press).

Published Conference Abstracts and Presentations (107 total)

2. Weiner, MR, Schwab, ZJ, Rauch, SL, & **Killgore WD**. Personality factors predict brain responses to images of high-calorie foods. Abstract presented at the McLean Hospital Research Day, January 13, 2011.

3. Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD.** Emotional and cognitive intelligence: Support for the neural efficiency hypothesis. Abstract presented at the McLean Hospital Research Day, January 13, 2011.

4. Crowley, DJ, Covell, MJ, **Killgore, WD**, Schwab, ZJ, Weiner, MR, Acharya, D, Rosso, IM, & Silveri, MM. Differential influence of facial expression on inhibitory capacity in adolescents versus adults. Abstract presented at the McLean Hospital Research Day, January 13, 2011.

5. Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD**. Neural correlates of cognitive and emotional intelligence in adults. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

6. Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD**. Cognitive and emotional intelligences: Are they distinct or related constructs? Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

7. Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD**. Discrepancy scores between cognitive and emotional intelligence predict neural responses to affective stimuli. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

8. **Killgore, WD**, Schwab, ZJ, Weiner, MR, & Rauch, SL. Smart people go with their gut: Emotional intelligence correlates with non-conscious insular responses to facial trustworthiness. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

9. **Killgore, WD**, Weiner, MR, Schwab, ZJ, & Rauch, SL. Whom can you trust? Neural correlates of subliminal perception of facial trustworthiness. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

10. Weiner, MR, Schwab, ZJ, & Rauch, SL, **Killgore, WD**. Impulsiveness predicts responses of brain reward circuitry to high-calorie foods. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

11. Weiner, MR, Schwab, ZJ, & Rauch, SL, **Killgore, WD**. Conscientiousness predicts brain responses to images of high-calorie foods. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

12. Crowley, DJ, Covell, MJ, **Killgore, WD**, Schwab, ZJ, Weiner, MR, Acharya, D, Rosso, IM, & Silveri, MM. Differential influence of facial expression on inhibitory capacity in adolescents versus adults. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

13. Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD.** Emotional and cognitive intelligence: Support for the neural efficiency hypothesis. Abstract presented at the 66th Annual Meeting of the Society for Biological Psychiatry, San Francisco, CA, May 12-14, 2011.

14. Weiner, MR, Schwab, ZJ, Rauch, SL, & **Killgore WD**. Personality factors predict brain responses to images of high-calorie foods. Abstract presented at the 66th Annual

Meeting of the Society for Biological Psychiatry, San Francisco, CA, May 12-14, 2011.

15. Weiner, MR, Schwab, ZJ, & **Killgore, WD.** Daytime sleepiness is associated with altered brain activation during visual perception of high-calorie foods: An fMRI study. Abstract presented at the 25th Annual Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 11-15, 2011.

16. Schwab, ZJ, Weiner, MR, & **Killgore, WD.** Functional MRI correlates of morningness-eveningness during visual presentation of high calorie foods. Abstract presented at the 25th Annual Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 11-15, 2011.

17. **Killgore, WD,** Weiner, MR, & Schwab, ZJ. Daytime sleepiness affects prefrontal regulation of food intake. Abstract presented at the McLean Hospital Research Day, January 11, 2012.

18. Kipman, M, Schwab ZJ, Weiner, MR, DelDonno, S, Rauch SL, & **Killgore WD**. The insightful yet bitter comedian: The role of emotional versus cognitive intelligence in humor appreciation. Abstract presented at the McLean Hospital Research Day, January 11, 2012.

19. Weber, M, & **Killgore, WD**. Gray matter correlates of emotional intelligence. Abstract presented at the McLean Hospital Research Day, January 11, 2012.

20. Schwab, ZJ, & **Killgore, WD**. Sex differences in functional brain responses to food. Abstract presented at the McLean Hospital Research Day, January 11, 2012.

21. DelDonno, S, Schwab, ZJ, Kipman M, Rauch, SL, & **Killgore, WD**. The influence of cognitive and emotional intelligence on performance on the Iowa Gambling Task. Abstract presented at the McLean Hospital Research Day, January 11, 2012.

22. Song, CH, Kizielewicz, J, Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD**. Time is of the essence: The Design Organization Test as a valid, reliable, and brief measure of visuospatial ability. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

23. Kipman, M, Schwab, ZJ, DelDonno, S, & **Killgore, WD**. Gender differences in the contribution of cognitive and emotional intelligence to the left visual field bias for facial perception. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

24. Kipman, M., Schwab, ZJ, Weiner, MR, DelDonno, S, Rauch, SL, & **Killgore, WD**. Contributions of emotional versus cognitive intelligence in humor appreciation. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

25. Schwab, ZJ, & **Killgore, WD**. Disentangling emotional and cognitive intelligence. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

26. Schwab, ZJ, & **Killgore, WD**. Sex differences in functional brain responses to food. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

27. DelDonno, S, Schwab, ZJ, Kipman, M, Rauch, SL, & **Killgore, WD**. The influence of cognitive and emotional intelligence on performance on the Iowa Gambling Task. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

28. **Killgore, WD**, & Schwab, ZJ. Emotional intelligence correlates with somatic marker circuitry responses to subliminal cues of facial trustworthiness. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

29. **Killgore, WD**, & Schwab, ZJ. Trust me! Neural correlates of the ability to identify facial trustworthiness. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

30. **Killgore, WD**, Schwab, ZJ, Weiner, MR, Kipman, M, DelDonno, S, & Rauch SL. Overeating is associated with altered cortico-limbic responses to images of high calorie foods. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

31. **Killgore, WD**, Weiner, MR, & Schwab, ZJ. Daytime sleepiness affects prefrontal regulation of food intake. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

32. Weber, M, DelDonno, S, Kipman M, Schwab, ZJ, & **Killgore WD**. Grey matter correlates of self-reported sleep duration. Abstract presented at the Harvard Medical School Research Day, Boston, MA, March 28, 2012.

33. **Killgore, WD**, Schwab, ZJ, & Rauch, SL. Daytime sleepiness affects prefrontal inhibition of food consumption. Abstract presented at the 67th Annual Meeting of the Society of Biological Psychiatry, Philadelphia, PA, May 3-5, 2012.

34. Kipman, M, Weber, M, DelDonno, S., Schwab, ZJ, & **Killgore, WD**. Morningness-Eveningness correlates with orbitofrontal gray matter volume. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

35. Kipman, M, Schwab, ZJ, Weber, M, DelDonno, S, & **Killgore, WD**. Yawning frequency

is correlated with reduced medial thalamic volume. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

36. Weber, M, DelDonno, S, Kipman M, Schwab, ZJ, & **Killgore WD**. Grey matter correlates of daytime sleepiness. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

37. Weber, M, DelDonno, S, Kipman M, Schwab, ZJ, & **Killgore WD**. Grey matter correlates of self-reported sleep duration. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

38. DelDonno, S, Weber, M, Kipman M, Schwab, ZJ, & **Killgore, WD**. Resistance to insufficient sleep correlates with olfactory cortex gray matter. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

39. DelDonno, S, Schwab, ZJ, Kipman, M, Weber, M, & **Killgore, WD**. Weekend sleep is related to greater coping and resilience capacities. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

40. Schwab, ZJ, DelDonno, S, Weber, M, Kipman M, & **Killgore, WD**. Habitual caffeine consumption and cerebral gray matter volume. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

41. Schwab, ZJ, & **Killgore, WD**. Daytime sleepiness affects prefrontal regulation of food intake. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

42. **Killgore, WD**, Schwab, ZJ, DelDonno S, Kipman, M, Weber M, & Rauch, SL. Greater nocturnal sleep time is associated with increased default mode functional connectivity. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

43. Sneider, JT, **Killgore, WD**, Crowley, DJ, Cohen-Gilbert, JE, Schwab, ZJ, & Silveri, MM. Inhibitory capacity in emerging adult binge drinkers: Influence of Facial Cues. Abstract presented at the 35th Annual Scientific Meeting of the Research Society on Alcoholism, San Francisco, CA, June 23-27, 2012.

44. Cohen-Gilbert, JE, **Killgore WD**, Crowley, DJ, Covell, MJ, Schwab, ZJ, Weiner, MR, Acharya, D, Sneider, JT, & Silveri, MM. Differential influence of safe versus threatening facial expressions on inhibitory control across adolescence and adulthood. Abstract presentede at the Society for Neuroscience 2012 Meeting, New Orleans, LA,

October 13-17, 2012.

45. Weber, M, DelDonno, S, Kipman M, Schwab, ZJ, & **Killgore WD**. Grey matter correlates of self-reported sleep duration. Abstract presented at the Harvard Division of Sleep Medicine Annual Poster Session, Boston, MA, September 27, 2012.

46. Sneider, JT, **Killgore, WD**, Crowley, DJ, Cohen-Gilbert, JE, Schwab, ZJ, & Silveri, MM. Inhibitory capacity in emerging adult binge drinkers: Influence of Facial Cues. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

47. Cohen-Gilbert, JE, **Killgore WD**, Crowley, DJ, Covell, MJ, Schwab, ZJ, Weiner, MR, Acharya, D, Sneider, JT, & Silveri, MM. Differential influence of safe versus threatening facial expressions on inhibitory control across adolescence and adulthood. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

48. Tkachenko, O, Schwab, ZJ, Kipman, M, DelDonno, S, Gogel, H., Preer, L, & **Killgore, WDS**. Smarter women need less sleep. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

49. DelDonno, S, Kipman, M, Schwab, ZJ, & **Killgore, WDS**. The contributions of emotional intelligence and facial perception to social intuition. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

50. Kipman, M, Schwab, ZJ, DelDonno, S, Weber, M, Rauch, SL, & **Killgore, WDS**. The neurocircuitry of impulsive behavior. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

51. Preer, LA, Tkachenko, O, Gogel, H, Schwab, ZJ, Kipman, M, DelDonno, SR, Weber, M, Webb, CA, & **Killgore, WDS**. Emotional intelligence as a mediator of the association between anxiety sensitivity and anxiety symptoms. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

52. Gogel, H, DelDonno, S, Kipman M, Preer, LA, Schwab, ZJ, Tkachenko, O, & **Killgore, WDS**. Validation of the Design Organization Test (DOT) in a healthy population. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

53. Cohen-Gilbert, JE, Schwab, ZJ, **Killgore, WDS**, Crowley, DJ, & Silveri MM. Influence of Binge Drinking on the Neural Correlates of Inhibitory Control during Emotional Distraction in Young Adults. Abstract presented at the 3rd International Conference on Applications of Neuroimaging to Alcoholism (ICANA-3), New Haven, CT, February 15-18, 2013.

54. Weber, M, & **Killgore, WDS**. The interrelationship between 'sleep credit', emotional intelligence and mental health – a voxel-based morphometric study. Abstract presented at Harvard Medical School Psychiatry Research Day, April 10, 2013.

55. Cohen-Gilbert, JE, Schwab, ZJ, **Killgore, WDS**, Crowley, DJ, & Silveri MM. Influence of Binge Drinking on the Neural Correlates of Inhibitory Control during Emotional Distraction in Young Adults. Abstract presented at Harvard Medical School Psychiatry Research Day, April 10, 2013.

56. Preer, LA, Tkachenko, O, Gogel, H, Schwab, ZJ, Kipman, M, DelDonno, SR, Weber, M, Webb, CA, Rauch, SL, & **Killgore, WDS**. Linking Sleep Trouble to Neuroticism, Emotional Control, and Impulsiveness. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

57. Preer, LA, Tkachenko, O, Gogel, H, Schwab, ZJ, Kipman, M, DelDonno, SR, Weber, M, Webb, CA, Rauch, SL, & **Killgore, WDS**. Emotional Intelligence as a Mediator of the Association between Anxiety Sensitivity and Anxiety Symptoms. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

58. Kipman, M, Schwab, ZJ, DelDonno, S, Weber, M, Rauch, SL, & **Killgore, WDS**. The neurocircuitry of impulsive behavior. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

59. Tkachenko, O, Schwab, ZJ, Kipman, M, Preer, LA, Gogel, H, DelDonno, SR, Weber, M, Webb, CA, Rauch, SL, & **Killgore, WDS**. Difficulty in falling asleep and staying asleep linked to a sub-clinical increase in symptoms of psychopathology. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

60. **Killgore, WDS**, Schwab, ZJ, Kipman, M, DelDonno, SR, Rauch, SL, & Weber, M. Problems with sleep initiation and sleep maintenance correlate with functional connectivity among primary sensory cortices. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

61. **Killgore, WDS**, Schwab, ZJ, Kipman, M, DelDonno, SR, Rauch, SL, & Weber, M. A Couple of Hours Can Make a Difference: Self-Reported Sleep Correlates with Prefrontal-Amygdala Connectivity and Emotional Functioning. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

62. Weber, M, & **Killgore, WDS**. The interrelationship between 'sleep credit', emotional intelligence and mental health – a voxel-based morphometric study. Abstract presented at the SLEEP 2013 Annual Meeting, Baltimore, MD, June 1-5, 2013.

63. **Killgore, WDS**, Schwab, ZJ, Kipman, M, DelDonno, SR, & Weber, M. Problems with Sleep Initiation and Sleep Maintenance Correlate with Functional Connectivity Among Primary Sensory Cortices. Abstract presented at the SLEEP 2013 Annual

Meeting, Baltimore, MD, June 1-5, 2013.

64. **Killgore, WDS**, Schwab, ZJ, Kipman, M, DelDonno, SR, & Weber, M. A Couple of Hours Can Make a Difference: Self-Reported Sleep Correlates with Prefrontal-Amygdala Connectivity and Emotional Functioning. Abstract presented at the SLEEP 2013 Annual Meeting, Baltimore, MD, June 1-5, 2013.

65. Tkachenko, O, Schwab, ZJ, Kipman, M, DelDonno, SR, Preer, LA, Gogel, H, Weber, M, Webb, CA, & **Killgore, WDS**. Difficulty in falling asleep and staying asleep linked to a sub-clinical increase in symptoms of psychopathology. Abstract presented at the SLEEP 2013 Annual Meeting, Baltimore, MD, June 1-5, 2013.

66. Preer, LA, Tkachenko, O, Gogel, H, Schwab, ZJ, Kipman, M, DelDonno, SR, Weber, M, Webb, CA, & **Killgore, WDS**. Linking Sleep Initiation Trouble to Neuroticism, Emotional Control, and Impulsiveness. Abstract presented at the SLEEP 2013 Annual Meeting, Baltimore, MD, June 1-5, 2013.

67. **Killgore, WDS**. Sleep duration contributes to cortico-limbic functional connectivity, emotional functioning, & psychological health. Abstract accepted for presentation at the 52nd Annual Meeting of the American College of Neuropsychopharmacology, Hollywood, FL, December 8-12, 2013.

68. Preer, L, Tkachenko, O, Gogel, H, Bark, JS, Kipman, M, Olson, EA, & **Killgore, WDS**. The role of personality in sleep initiation problems. Abstract presented at the Annual McLean Hospital Research Day, January 22, 2014.

69. Demers, LA, Olson, EA, Weber, M, Divatia, S, Preer, L, & **Killgore, WDS**. Paranoid traits are related to deficits in complex social decision-making and reduced superior temporal sulcus volume. Abstract presented at the Annual McLean Hospital Research Day, January 22, 2014.

70. Tkachenko, O, Weber, M, Gogel, H, & **Killgore, WDS**. Predisposition towards unhealthy foods linked with increased gray matter in the cerebellum. Abstract presented at the Annual McLean Hospital Research Day, January 22, 2014.

71. Olson, EA, Weber, M, Tkachenko, O, & **Killgore, WDS**. Daytime sleepiness is associated with decreased integration of remote outcomes on the IGT. Abstract presented at the Annual McLean Hospital Research Day, January 22, 2014.

72. Cui, J, Tkachenko, O, & **Killgore, WDS**. Can the activation of anterior cingulate predict the emotional suppression? An fMRI study with masked faces. Abstract presented at the Annual McLean Hospital Research Day, January 22, 2014.

73. Gogel, H, & **Killgore WDS**. A psychometric validation of the Design Organization Test (DOT) in a healthy sample. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

74. **Killgore, WDS**, Weber, M, Bark, JS, Kipman, M, Gogel, H, Preer, L, Tkachenko, O, Demers, LA, Divatia, SC, & Olson, EA. Physical exercise correlates with hippocampal volume in healthy adults. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

75. **Killgore, WDS**, Tkachenko, O, Weber, M, Kipman, M, Preer, L, Gogel, H, & Olson, EA. The association between sleep, functional connectivity, and emotional functioning. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

76. Preer, L, Tkachenko, O, Gogel, H, Bark, JS, Kipman, M, Olson, EA, & **Killgore, WDS**. The role of personality in sleep initiation problems. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

77. Tkachenko, O, Weber, M, Olson, EA, Gogel, H, Preer, LA, Divatia, SC, Demers, LA, & **Killgore, WDS**. Gray matter volume within the medial prefrontal cortex correlates with behavioral risk taking. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

78. Olson, EA, Weber, M, Bark JS, Demers L, Divatia, SC, Gogel, H, Kipman M, Preer, L, Tkachenko, O, & **Killgore, WDS**. Sex differences in threat evaluation of emotionally neutral faces. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

79. Cui, J, Tkachenko, O, & **Killgore, WDS**. Can the activation of anterior cingulate predict the emotional suppression? An fMRI study with masked faces. Abstract presented at the 36nd Annual Conference of the Anxiety Disorders Association of America, Chicago, IL, March 27-30, 2014.

80. Webb, CA, Weber, M, Mundy, EA, & **Killgore, WDS**. Reduced gray matter volume in the anterior cingulate, orbitofrontal cortex and thalamus as a function of depressive symptoms: A voxel-based morphometric analysis. Abstract presented at the 36nd Annual Conference of the Anxiety Disorders Association of America, Chicago, IL, March 27-30, 2014.

81. Cui, J, Tkachenko, O, & **Killgore, WDS**. Can the activation of anterior cingulate predict the emotional suppression? An fMRI study with masked faces. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

82. Divatia, S, Demers, LA, Preer, L, Olson, EA, Weber, M, & **Killgore, WDS**. Advantageous decision making linked with increased gray matter volume in the ventromedial prefrontal cortex. Abstract presented at the 21st Annual Meeting of the

Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

83. Demers, LA, Olson, EA, Weber, M, Divatia, S, Preer, L, & **Killgore, WDS**. Paranoid traits are related to deficits in complex social decision making and reduced superior temporal sulcus volume. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

84. Preer, LA, Weber, M, Tkachenko, O, Divatia, S, Demers, LA, Olson, EA, & **Killgore, WDS**. Gray matter volume in the amygdala is associated with facial assessments of trustworthiness. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

85. Tkachenko, O, Weber, M, Gogel, H, & **Killgore, WDS**. Predisposition towards unhealthy foods linked with increased gray matter volume in the cerebellum. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

86. Olson, EA, Weber, M, Gogel, H, & **Killgore, WDS**. Daytime sleepiness is associated with decreased integration of remote outcomes on the IGT. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

87. Demers, LA, Preer, LA, Gogel, H, Olson, EA, Weber, M, & **Killgore, WDS**. Left-hemifield bias on sad chimeric face task correlates with interpersonal emotional intelligence. Abstract presented at the 69th Annual Meeting of the Society of Biological Psychiatry, New York, NY, May 8-10, 2014.

88. **Killgore, WDS**, Weber, M, Olson, EA, & Rauch, SL. Sleep reduction and functioning of the emotion regulation circuitry. Abstract presented at the 69th Annual Meeting of the Society of Biological Psychiatry, New York, NY, May 8-10, 2014. ***[*Blue Ribbon Finalist for Top Poster Award: Basic Neuroscience]***

89. Webb, CA, Weber, M, Mundy, EA, & **Killgore, WDS**. Reduced gray matter volume in the anterior cingulate, orbitofrontal cortex and thalamus as a function of depressive symptoms: A voxel-based morphometric analysis. Abstract presented at the 69th Annual Meeting of the Society of Biological Psychiatry, New York, NY, May 8-10, 2014.

90. Weber, M, & **Killgore, WDS**. Sleep habits reflect in functional brain network organization. Abstract presented at SLEEP 2014, Minneapolis, MN, May 31-June 4, 2014. ***[*2014 AASM Young Investigator Award, Honorable Mention]***

91. Alkozei, A, Pisner, D, & **Killgore, WDS**. Emotional intelligence is differentially correlated with prefrontal cortical responses to backward masked fearful and angry faces. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

92. Alkozei, A, Schwab, Z, & **Killgore, WDS**. Looking for evil intent: Emotional intelligence and the use of socially relevant facial cues during an emotional decision making task. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

93. Shane, BR, Alkozei, A, & **Killgore, WDS**. The contribution of general intelligence and emotional intelligence to the ability to appreciate humor. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

94. Markowski, SM, Alkozei, A, & **Killgore, WDS**. Sleep onset latency and duration are associated with self-perceived invincibility. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

95. Pisner, D, Alkozei, A, & **Killgore, WDS**. Visuospatial reasoning mediates the relationship between emotion recognition and emotional intelligence. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

96. Vanuk, JR, Fridman, A, Demers, LA, Divatia, S, & **Killgore, WDS**. Engaging in meditation and internet based training as a means of enhancing emotional intelligence. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

97. Vanuk, JR, Divatia, S, Demers, LA, Markowski, SM, & **Killgore, WDS**. Napping in conjunction with brief internet-based training as a means of enhancing emotional intelligence. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

98. **Killgore, WDS**, Olson, EA, Weber, M, Rauch, SL, & Nickerson, LD. Emotional intelligence is associated with coordinated resting state activity between emotion regulation and interoceptive experience networks. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

99. **Killgore, WDS**, Demers, LA, Divatia, S, Kipman, M, Tkachenko, O, Weber, M, Preer, LA, Gogel, H, Olson, EA, Vanuk, JR, & Rauch, SL. Enhancing emotional intelligence via brief internet-based training. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

100. Markowski, SM, Alkozei, A, Rauch, SL, & **Killgore, WDS**. Self-perceived invincibility is associated with sleep onset latency and duration. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

101. Vanuk, JR, Rosso, IM, Rauch, SL, Alkozei, A, Markowski, SM, Pisner, D, Shane, BR, Fridman A, Knight, SA, & **Killgore, WDS**. Daytime sleepiness is associated with altered thalamocortical connectivity. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

102. Alkozei, A, Pisner, D, Rauch, SL, & **Killgore, WDS**. Emotional intelligence and subliminal presentations of social threat. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

103. Vanuk, JR, Shane, BR, Alkozei, A, & **Killgore, WDS**. Trait emotional intelligence is associated with greater resting state functional connectivity within the default mode and task positive networks. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

104. Vanuk, JR, Fridman, A, Demers, LA, & **Killgore, WDS**. Engaging in meditation and internet-based training as a means of enhancing emotional intelligence. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

105. Pisner, D, Alkozei, A, & **Killgore, WDS**. Trait emotional suppression is associated with decreased activation of the insula and thalamus in response to masked angry faces. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

106. **Killgore, WDS**, Rosso, IM, Rauch, SL, & Nickerson, LD. Emotional intelligence correlates with coordinated resting state activity between brain networks involved in emotion regulation and interoceptive experience. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

107. **Killgore, WDS**, Demers, LA, Divatia, S, Rosso, IM, & Rauch, SL. Boosting Emotional intelligence with a brief internet-based program. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

108. **Killgore, WDS**, Vanuk, JR, Alkozei, A, Markowski, SM, Pisner, D, Shane, BR, Fridman, A, & Knight, SA. Greater daytime sleepiness correlates with altered thalamocortical connectivity. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

CONCLUSION:

The principal neuroimaging study and the subsequent modification study have both been fully completed. The project met or exceeded all of the goals and requirements of the SOW and its subsequent modification.

In summary, the initial neuroimaging study sought to verify the conceptual basis of EI and to link it with underlying brain systems. Findings from this project suggest that EI is a useful construct that has predictive ability above and beyond standard measures of IQ and personality, although the Trait and Ability models of EI appear to measure distinct and orthogonal aspects of these capacities and may be important predictors in different contexts. Greater EI capacities, particularly those related to the Ability model, are strongly related to larger cortical volume and functional responsiveness within several key systems involved in emotional regulation and experience, particularly the ventromedial prefrontal cortex and insular cortex. Furthermore, greater EI is also related to enhanced functional connectivity and white matter integrity within brain networks that interconnect these regions. On the other hand, the Trait measures of EI showed limited associations with stable measures of neuroanatomy and functional neurocircuitry, though were affected by stresses such as insufficient sleep. These findings suggest that EI can be considered an important psychological capacity that can be effectively measured, is related to important emotional and cognitive performance metrics, and has its foundation in the principal brain circuitry involved in emotional experience and regulation. These novel findings provide a solid foundation upon which future work can build and test strategies to enhance EI capacities.

In addition to the core focus on EI, this project also produced vast amounts of information related to secondary questions of high importance to the military, including the brain systems linking sleep, sleep loss, insomnia, diet, and exercise to various aspects of health, wellbeing, and emotional functioning. These secondary analyses and associated published findings have contributed extensively to the current knowledge base regarding the effects of insufficient sleep on brain structure and function, and its effects on emotional systems, the role of brain connectivity in insomnia, and the association between physical exercise and brain structure, among others.

Finally, with the additional year of funding provided by the SOW modification, we completed a second project that involved conceptualizing, developing, and pilot testing a brief internet-based training program for enhancing EI skills. We successfully created the program content and developed a secure website to present the training via six on-line lessons in an easy to access format, and completed data collection to validate the program. The pilot data from this preliminary, randomized placebo-controlled clinical trial showed that the EI Training program produced significant enhancement of several key components of the Ability model of EI. This suggests that EI enhancement is possible through the use of a brief, targeted, and easily disseminated training program. Based on these exciting preliminary findings, we have submitted a proposal to Funding Opportunity Number: W81XWH-14-PHTBI-PHRA to request funds to develop this training program to its full potential and validate it for widespread use in various military contexts.

Not only has the current award provided extensive understanding of the neurobiological basis of EI and the potential for developing a training program to enhance such skills, the project has yielded an exceptional number of published products. Although analyses are still ongoing and further publications are anticipated in the coming year, thus far we have **published/presented 107 abstracts**, posters, and oral presentations at various scientific conferences and have successfully **published 22 manuscripts** in peer-reviewed journals, with numerous others in submission and in preparation. Several of these initial findings have already received wide-spread attention in the scientific press and popular media, including write-ups in the Los Angeles Times (http://articles.latimes.com/2011/jun/14/news/la-heb-sleep-carbs-20110614), Chicago Tribune (http://articles.chicagotribune.com/2012-04-25/health/sc-health-0425-bit-of-fit-20120425_1_junk-food-unhealthy-food-high-calorie-foods), Huffington Post (http://www.huffingtonpost.com/2014/03/07/happier-morning_n_4892107.html), and many other newspaper, magazine, web, and television news outlets. One of our papers was also selected to be the cover image and cover story for the 12 September 2012 issue of the journal NeuorReport.

Overall, EI appears to be a modifiable capacity with a clearly defined neurobiological basis. Modification of these skills through brief and targeted training may provide an effective method for equipping warfighters with many of the core capacities necessary to navigate stressful and/or traumatic experiences. This approach may provide an effective alternative or adjunctive approach to existing large-scale efforts that attempt to tackle broad and loosely related emotional/social/spiritual constructs, which have thus far shown limited success. Based on our findings, we propose that targeted EI training may be a practical approach for enhancing these skills in servicemembers and their families. With further optimization and validation, we believe that the preliminary EI Training program we have developed could be expanded to provide and rapid and easily disseminated approach for developing these core skills.

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APPENDICES:

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Examples of the On-Line EI Training Program **…… 58 - 89**

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Note: As the study is closed to recruitment, a list of the assessments is included in this report rather than a copy of each assessment.

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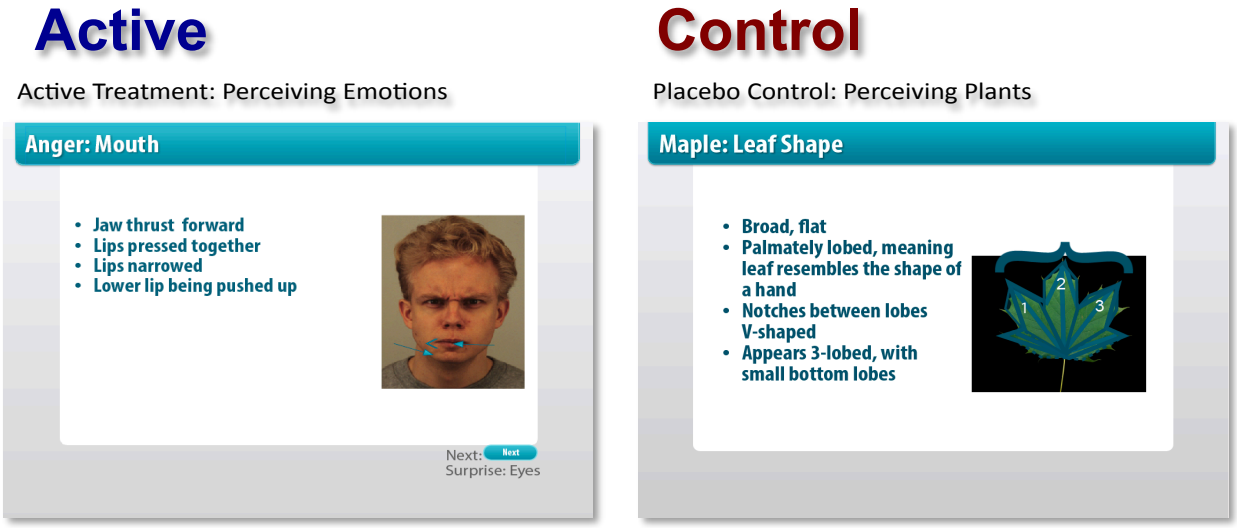
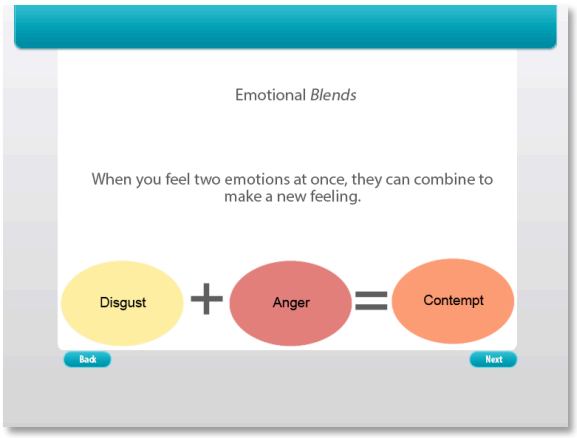
EXAMPLES OF THE ON LINE EI TRAINING PROGRAM

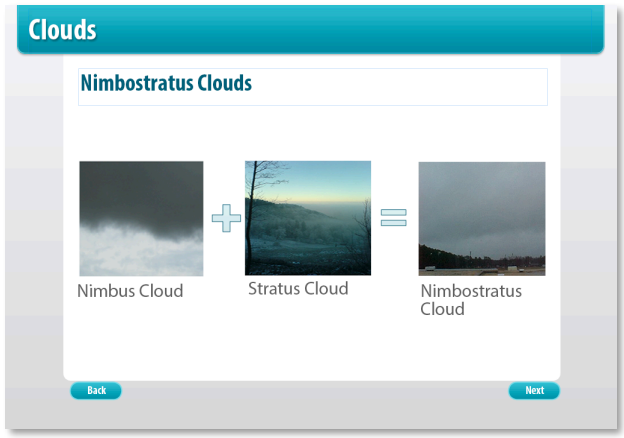
The internet based training programs each last between 15 to 45 minutes to complete, depending on individual variability in participant engagement and reading speed. After each lesson, participants also complete a homework assignment. The next several pages provide a summary of the course content for the active EI (Internal Awareness) Training program, with example screen images of the program interface:

Overview: Participants were assigned to one of two computer administered training programs, described as Emotional Intelligence Training (EIT) or Placebo Control Training (PCT). The two programs are nearly identical in format and layout, differing only in the presence or absence of EI content. Both programs comprised 6 online training modules. Module 1 provided an introduction to the program. Modules 2 – 5 provided the primary lesson content, and Module 6 provided a summary and review of previously learned content. The EIT program focused on the 4-Branch Model of EI, while the PCT follows the same format, but replaces the EI content with training in environmental awareness (i.e., weather patterns; recognizing plants; etc).

As shown in the screenshots below, the Active and Control programs look similar and present information in nearly identical ways, but differ only in content. In the figure, the Active treatment condition teaches the participant how to read facial expressions of emotion or learn about emotional blends, while the Control condition teaches the participant how to identify the plants based on the shape of the leaves and how cloud formations can combine to make new cloud formations. Both tasks involve similar learning strategies, but one is emotional while the other is not. This congruence in presentation format is consistent throughout the programs.



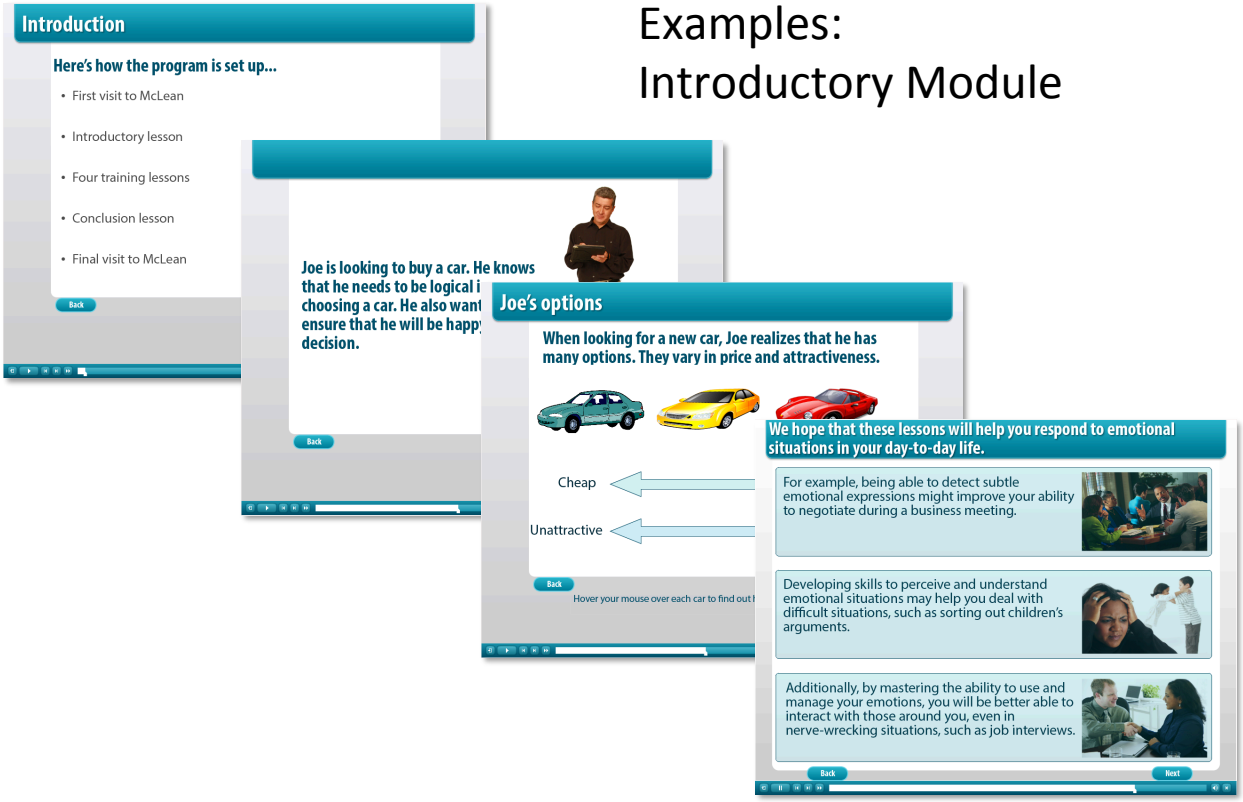




General Training Procedure: Participants was be assigned a private username and password to log into a secure Internet server. Upon secure login, participants completed the assigned lesson. Each lesson typically required between 30 to 45 minutes to complete. The lessons were didactic and interactive, requiring participants to solve problems and make responses as they moved through the content. The website logged the participant's login time and general activity so that it was possible to verify that they in fact completed the lesson in the prescribed manner. A brief homework assignment was also given after each lesson in order to generalize these skills to actual life situations and ensure that participants are actively engaged in the training.

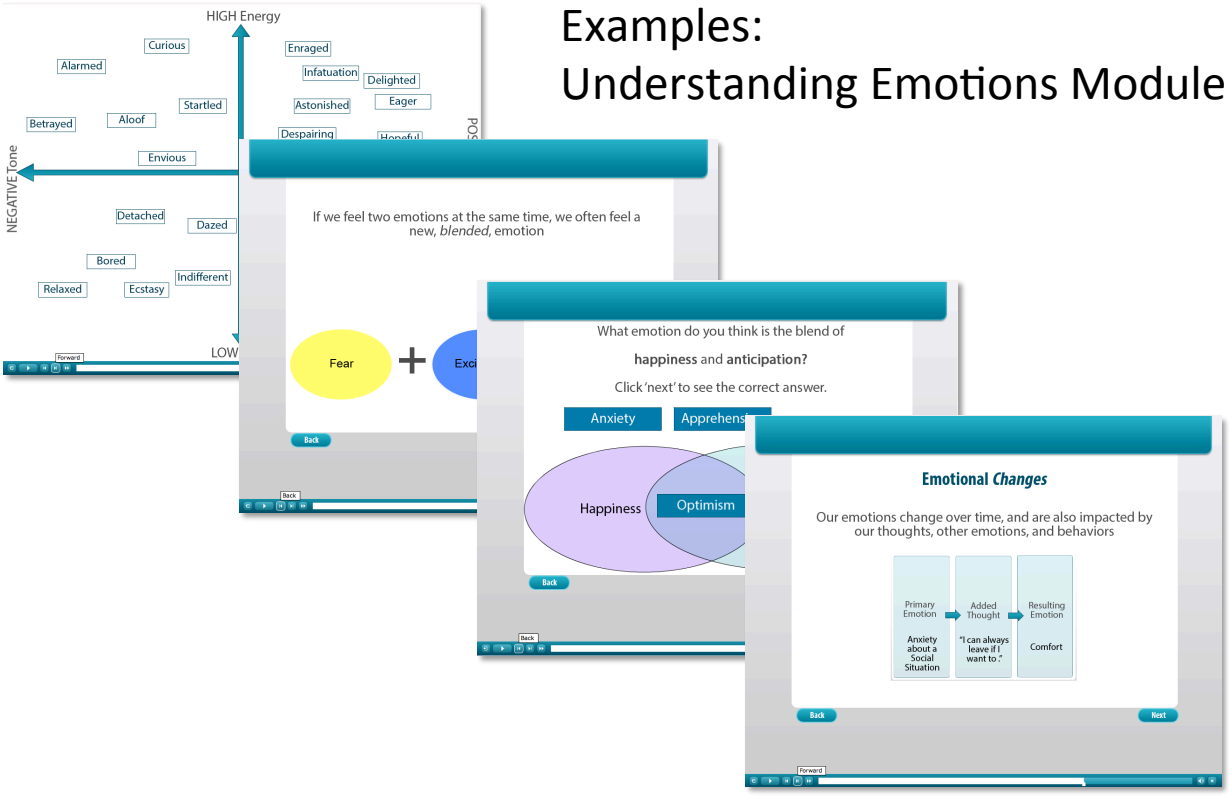
Emotional Intelligence Training (EIT): This program was described as "Internal Awareness Training" for the research participant, so that the focus was not specifically on "emotional intelligence" and to equate it with the "External Awareness" modules that are used for the PCT. Each lesson typically lasted between 30 to 45 minutes. The first lesson provided an overview of the 4-branch model of EI and will guide the participant through some basic examples of each type of skill. Participants were then be given a homework assignment involving identification of relevant situations where EI principles might prove adaptive in their daily lives. Lessons 2 through 5 will each focused on developing and enhancing a different branch ability of EI through programmed instruction and assessment tasks completed on-line through a secure internet website. The final lesson involved a review and integration of the previously acquired skill sets and a final homework assignment applying these skills in complex situations. The following screenshots detail samples of the lesson content:

Examples:
Introductory Module

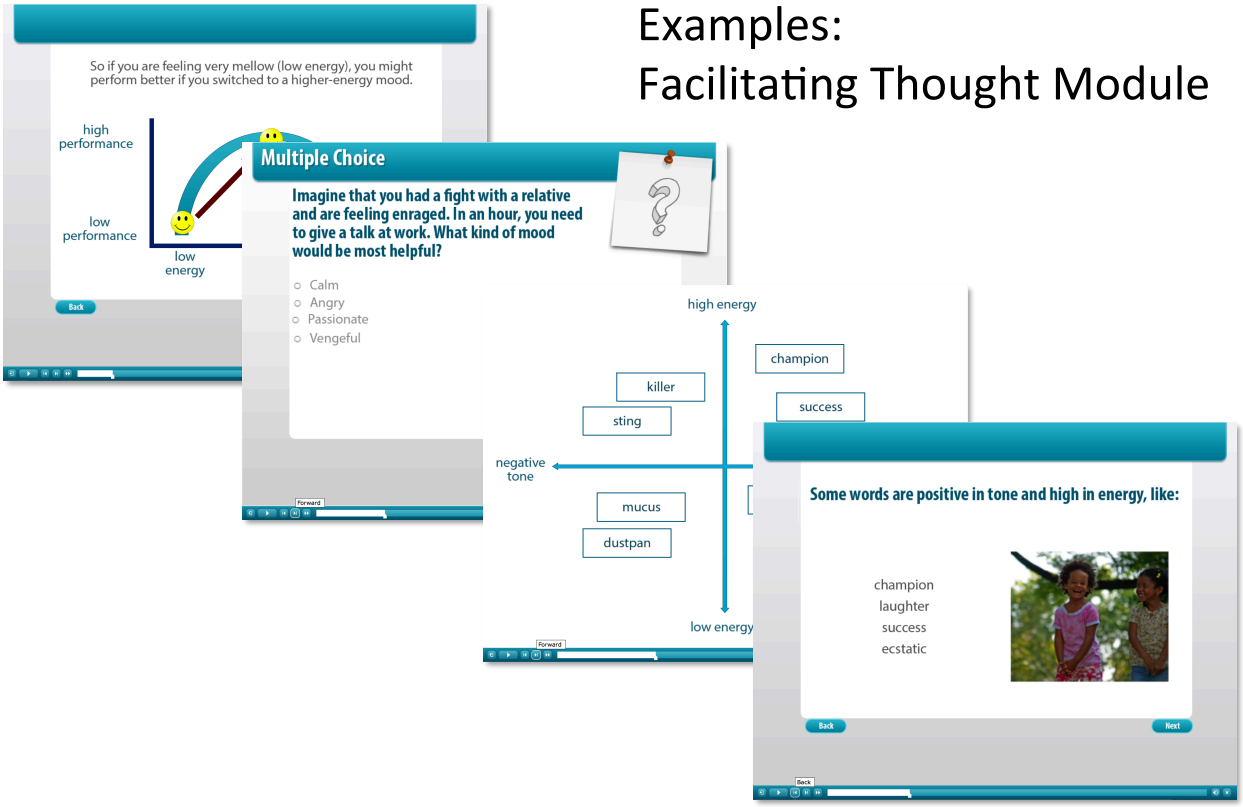


Examples:
Perceiving Emotions Module

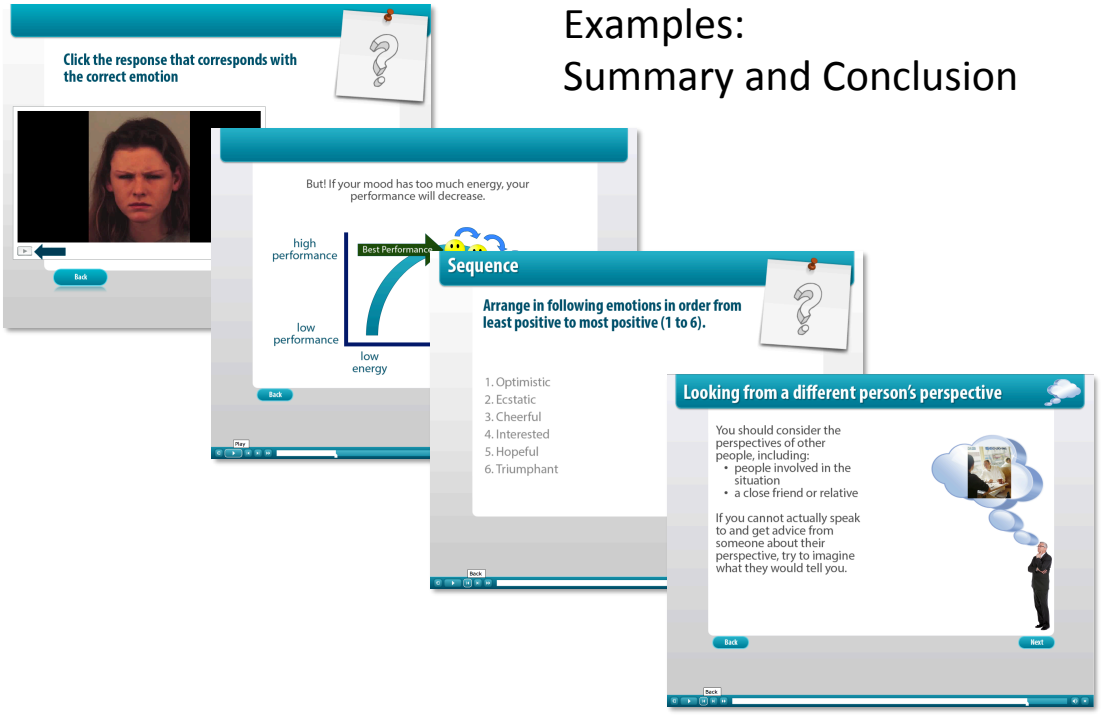
Examples:
Understanding Emotions Module



Examples:
Facilitating Thought Module



Examples:
Managing Emotions Module



Examples:
Summary and Conclusion

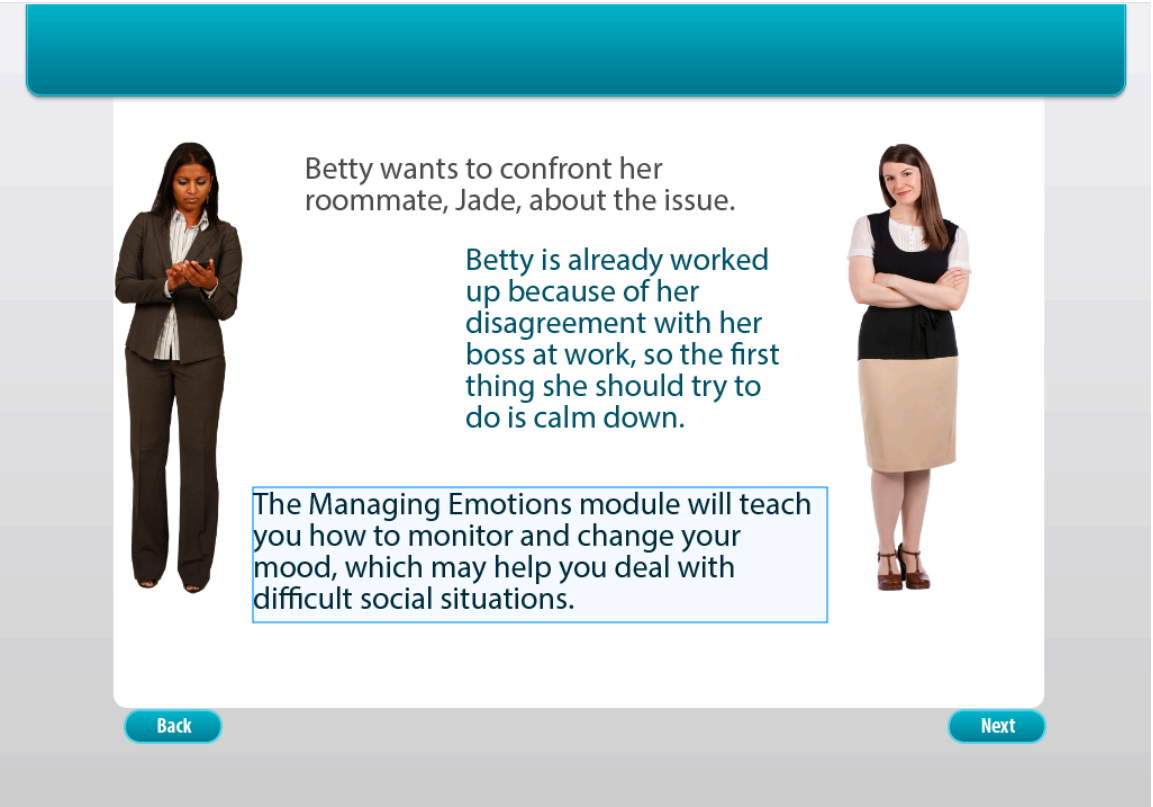
Placebo Control Training (PCT): This program was described as "External Awareness Training" for the research participant, so that the focus is not specifically on "emotional intelligence." Each lesson was closely matched to the tasks and presentation style of the active

EIT program. Each lesson typically lasts between 30 to 45 minutes. An example of some of the matched PCT module screens is shown below:

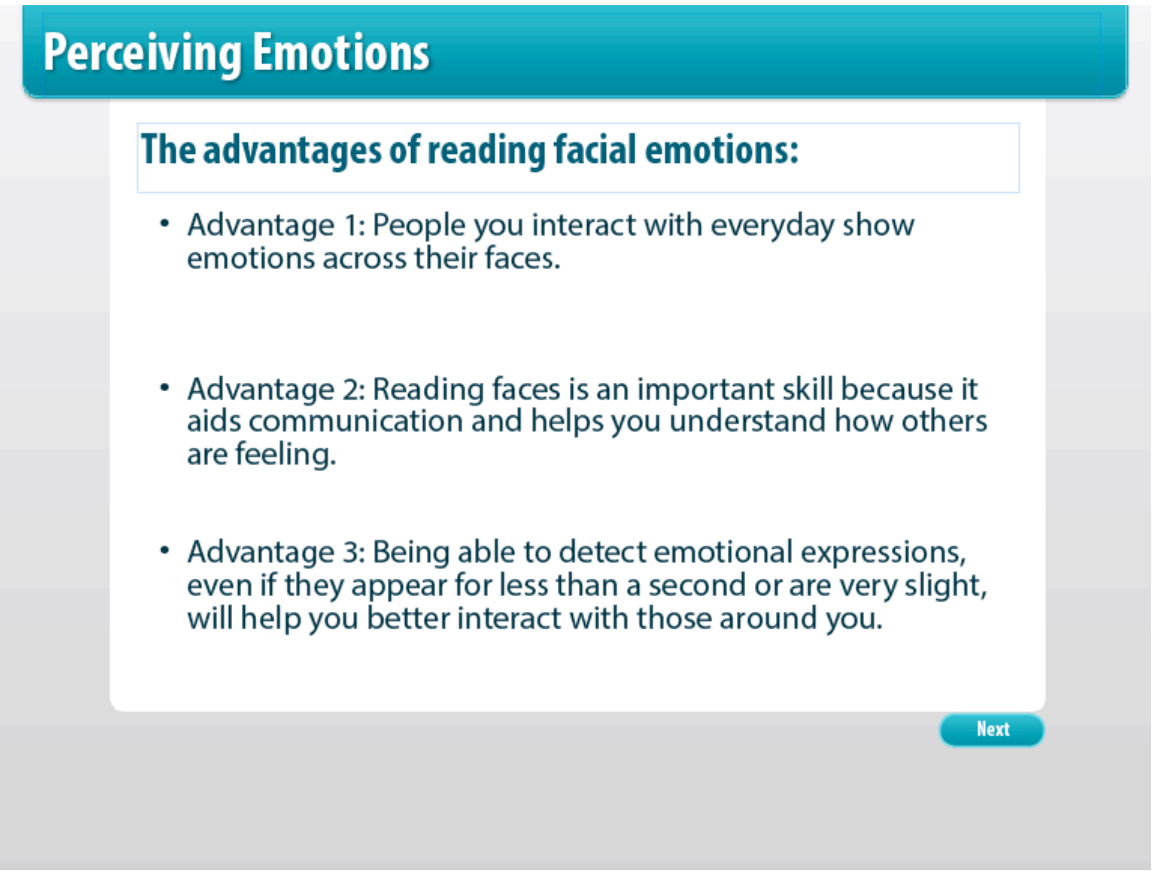
Detailed Outlines of Each Lesson:

Internal Awareness

1) Introduction
   - Introduce program structure
   - Rationale for increasing internal awareness
   - Evolutionary perspective of value of emotions
   - Examples of how internal awareness is useful (with characters and scenarios)
   - True/False questions about common myths about emotional functioning
   - Introduce program content
   - Example of how program will be useful (with characters and scenarios)
   - Multiple choice questions about emotions most likely to be experienced in a situation
   - Conclusion



2) Perceiving Emotions
   - Advantages of reading facial emotions
   - Go through features of faces expressing the six basic emotions – focus on eyes and mouth. Use arrows pointing to each component.
     - Ekman descriptions of features/cues
     - Karolinska Directed Emotional Faces as stimuli
   - Matching game: emotion to face
   - Videos of people going from neutral to emotional expression -- first at normal speed and then slowed down with features previously taught described in real time (words show up at bottom of video as the person is expressing emotion)
     - The Amsterdam Dynamic Facial Expression Set videos
   - Matching game: emotion to face
   - 6 multiple choice: quick flashes of emotion (slow version – 3sec – and correct answer is displayed and explained)
   - 48 multiple choice: quick flashes of emotion (fast version – 0.5sec – just correct answer is displayed, not explained)
   - Conclusion – advantages


Perceiving Emotions
The advantages of reading facial emotions:
• Advantage 1: People you interact with everyday show emotions across their faces.
• Advantage 2: Reading faces is an important skill because it aids communication and helps you understand how others are feeling.
• Advantage 3: Being able to detect emotional expressions, even if they appear for less than a second or are very slight, will help you better interact with those around you.
Next

Anger: Mouth

- Jaw thrust forward
- Lips pressed together
- Lips narrowed
- Lower lip being pushed up

Next: Next
Surprise: Eyes

Click the response that corresponds with the correct emotion

- A) Anger
- B) Fear
- C) Sadness
- D) Disgust
- E) Happiness
- F) Surprise

Back

Submit

Good Job!

Now you can use your new skills to read emotional expressions on faces in your day-to-day life!

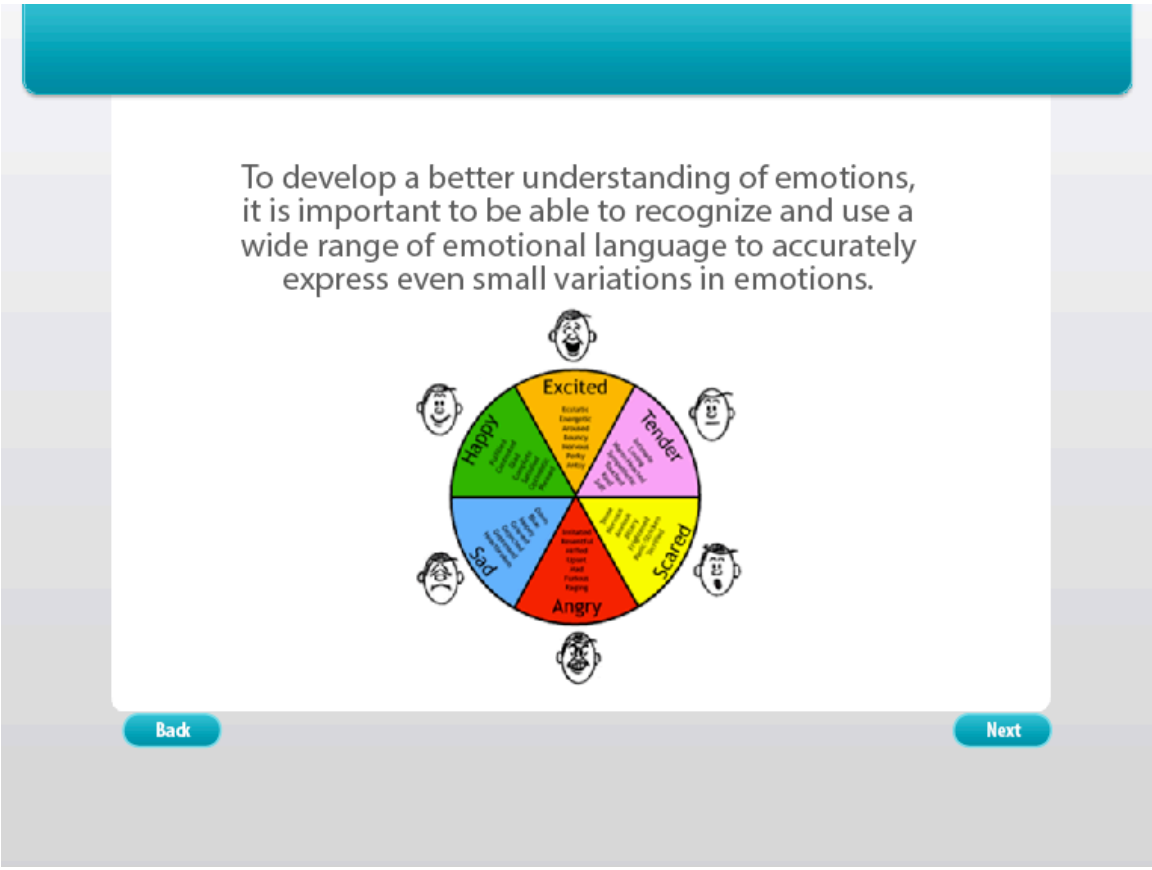
For instance, if someone is acting strangely and you are not sure why, perhaps trying to read his emotions shown on his face may help you understand what is going on.

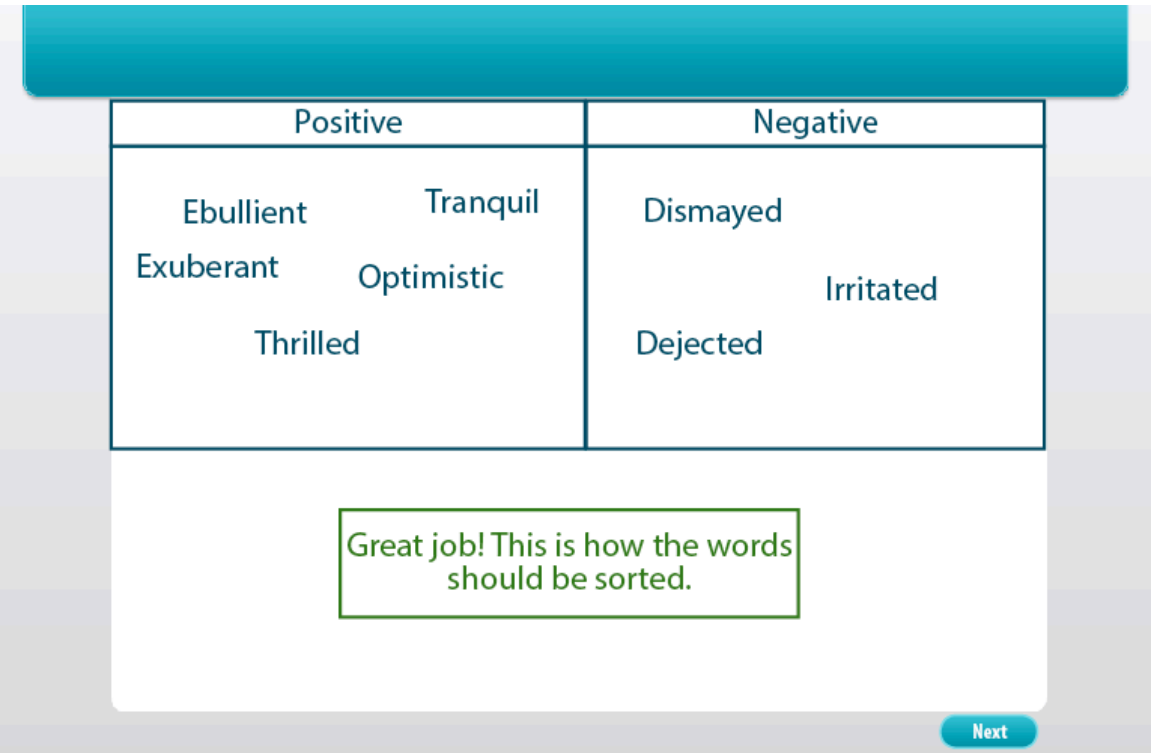
If you are having a discussion with someone and she is saying unexpected things, if you are able to read her emotions by looking at their facial features, you may be able to tell why she is acting this way.

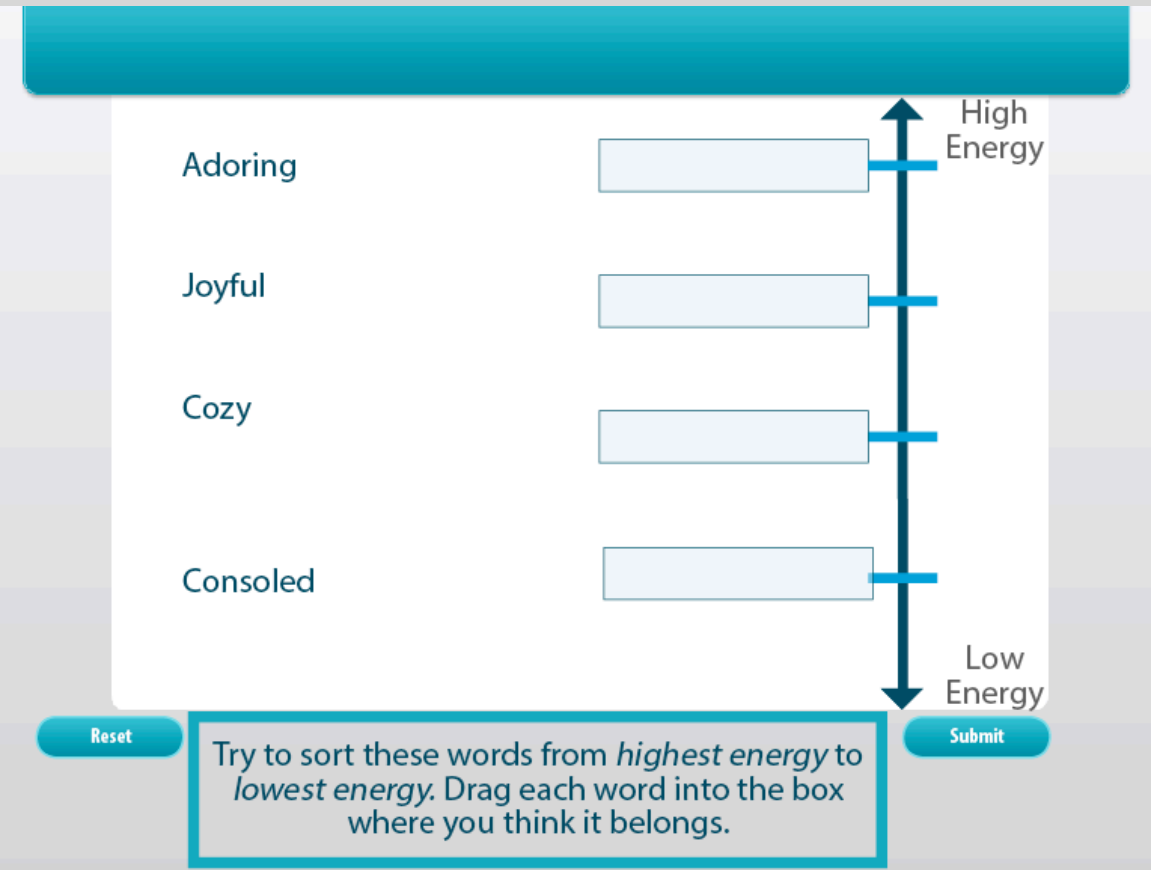
Back

Next

3) Understanding Emotions
    - Introduction: advantages and basic concepts
    - Tone and Energy descriptions
    - Sorting words into positive or negative tone categories
        - ANEW Word List (Bradley & Lange)
    - Sorting words into positive or negative tone dimensions
    - Sorting words into high to low energy dimension
    - Explains how to think about tone and energy
    - Sorting words in 4 quadrants (tone, energy)
    - Blends – descriptive explanation, strategy for how to come up with blended emotion
        - Plutchik's eight primary emotions…
    - Questions – choose the correct blended emotion
    - Changes – descriptive explanation
    - Questions – choose the event that changed the person's emotion from A to B
    - Point of view – descriptive explanation, strategy for how to come up with
    - Point of view multiple choice questions
    - Conclusion



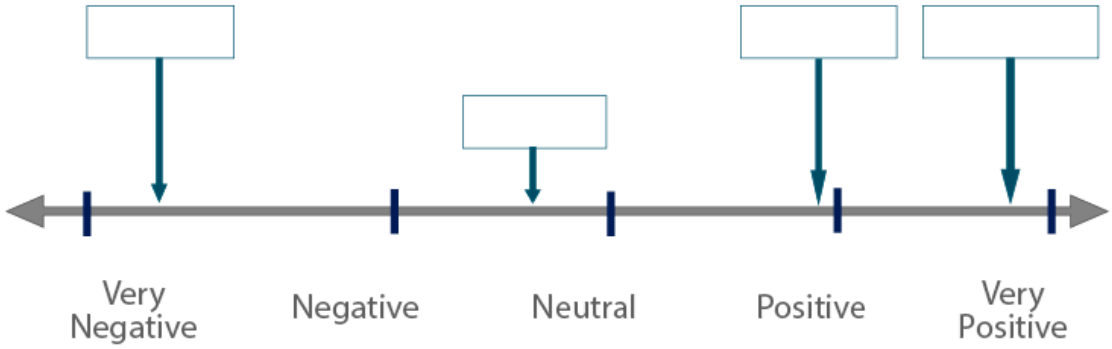

Positive
Negative
Ebullient
Tranquil
Exuberant
Optimistic
Thrilled
Dismayed
Irritated
Dejected
Great job! This is how the words should be sorted.
Next


Adoring
Joyful
Cozy
Consoled
High Energy
Low Energy
Reset
Submit
Try to sort these words from *highest energy* to *lowest energy*. Drag each word into the box where you think it belongs.

Enthusiastic Trusting

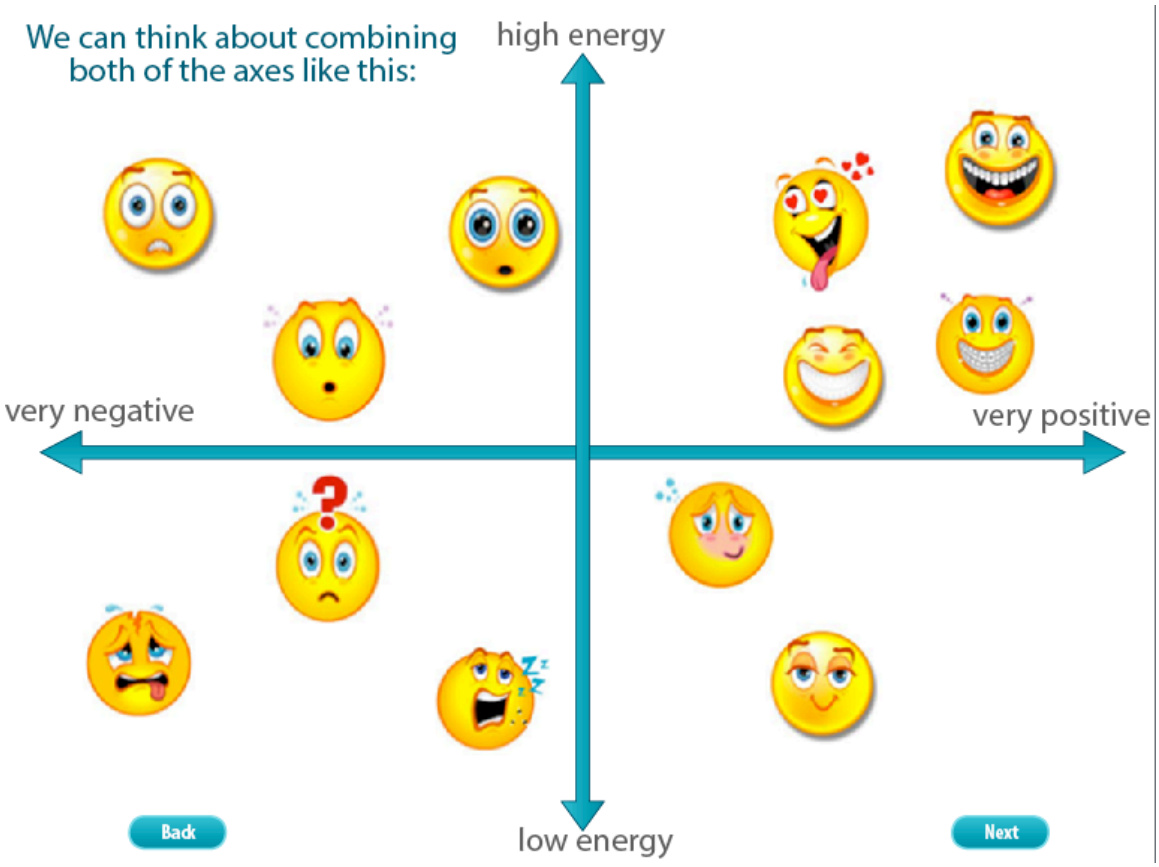
Subdued Panicked

Now let's practice. Try to sort these words based on their tone.



Back Reset Submit

We can think about combining both of the axes like this:



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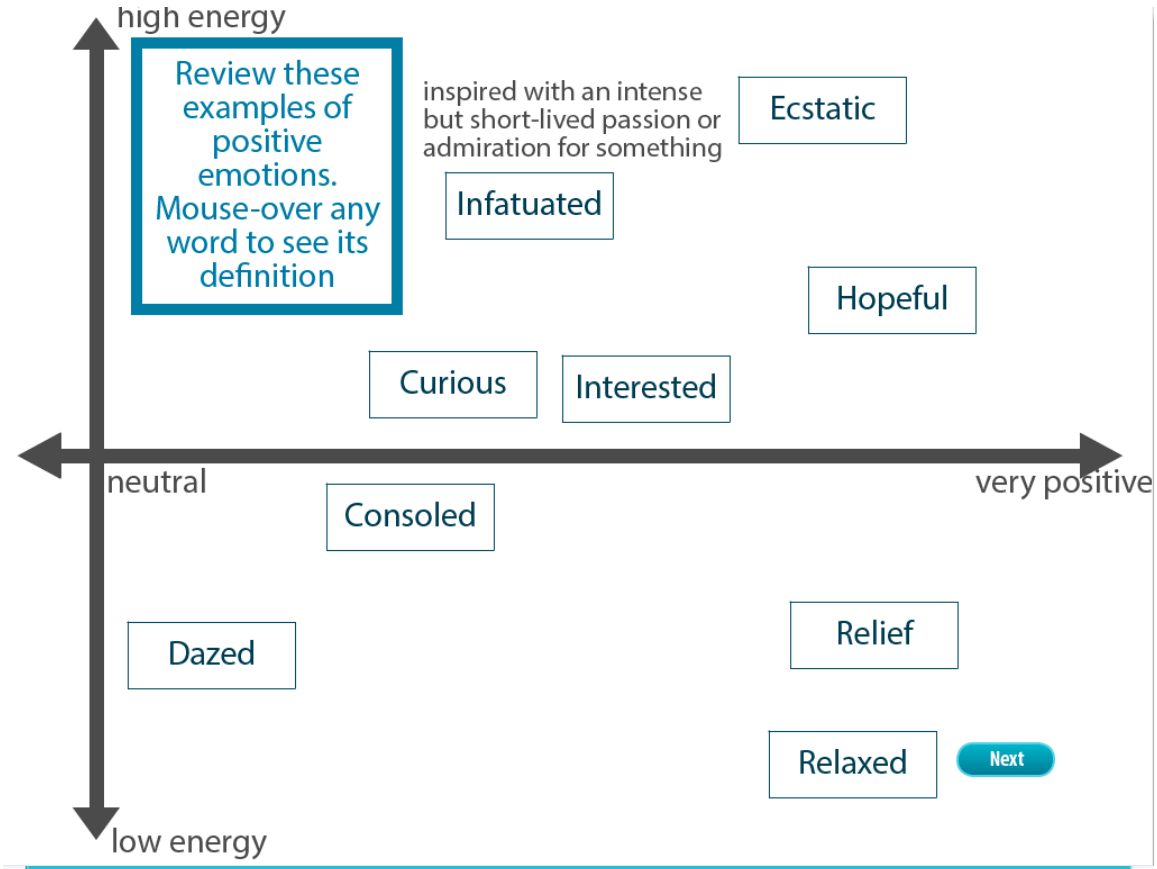
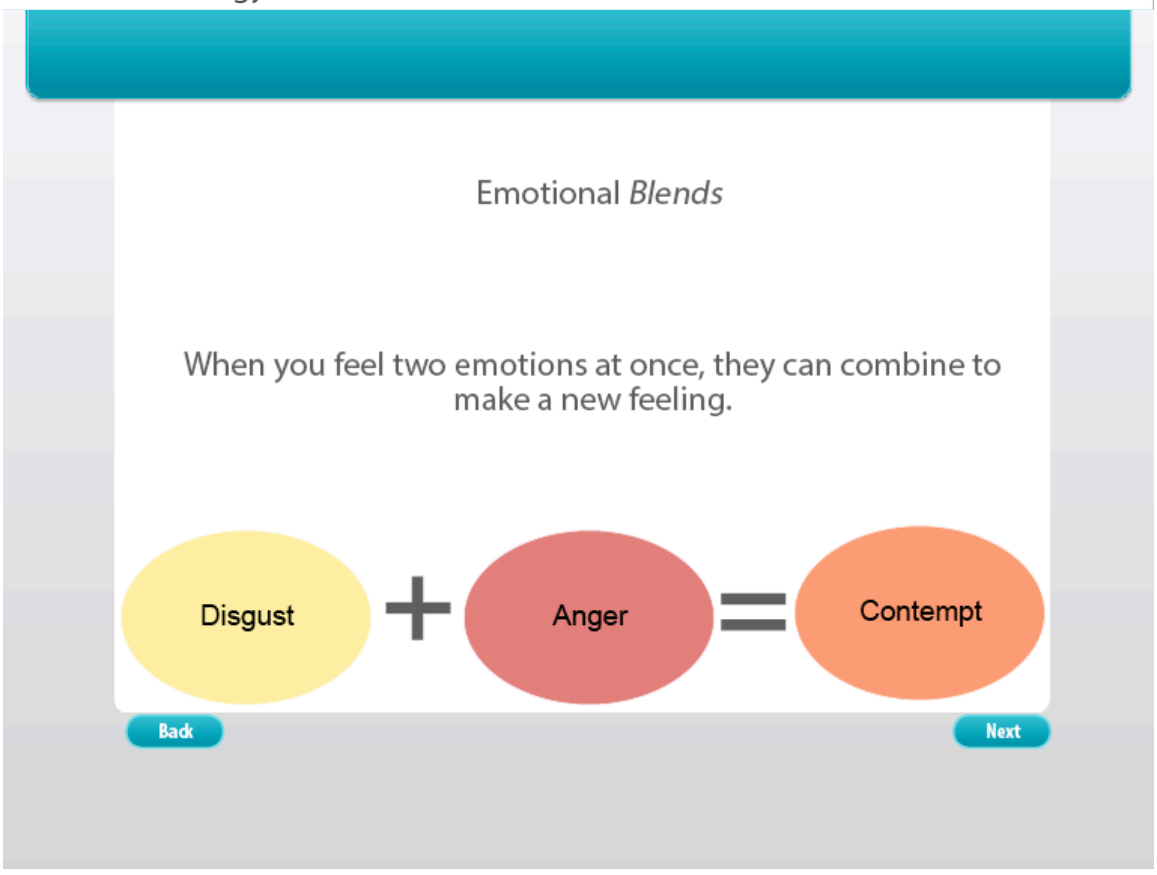
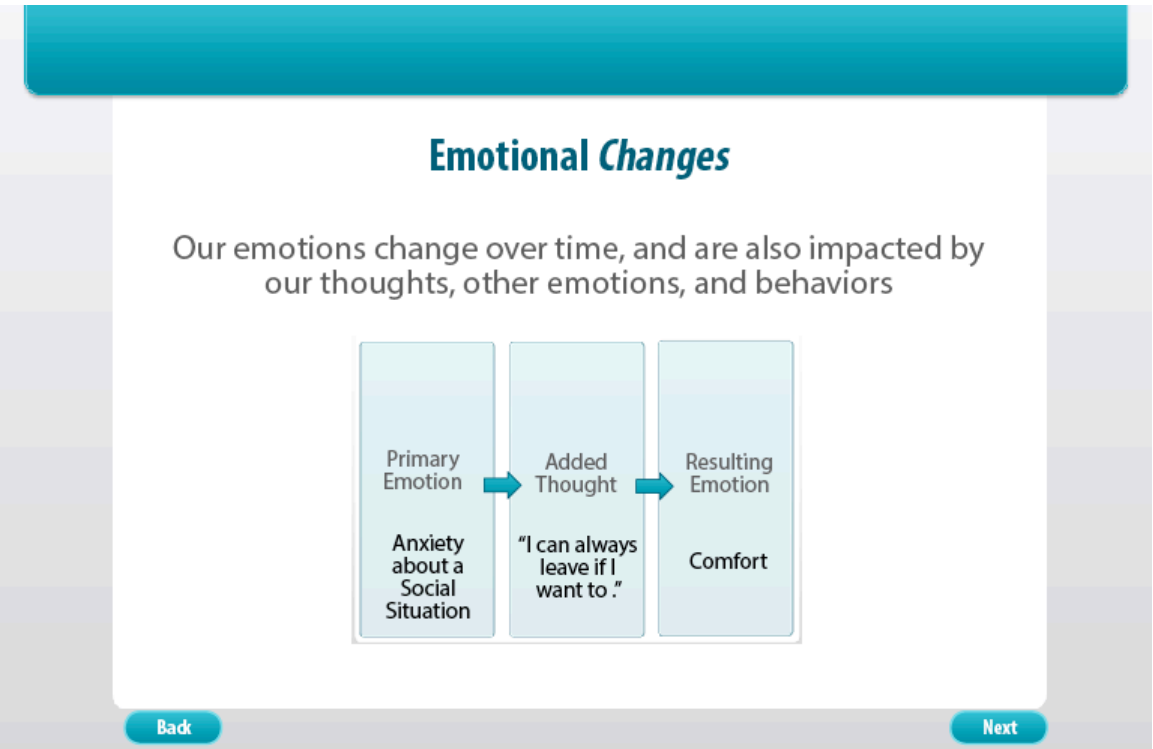

high energy
Review these examples of positive emotions. Mouse-over any word to see its definition
inspired with an intense but short-lived passion or admiration for something
Ecstatic
Infatuated
Hopeful
Curious
Interested
neutral
very positive
Consoled
Dazed
Relief
Relaxed
Next
low energy

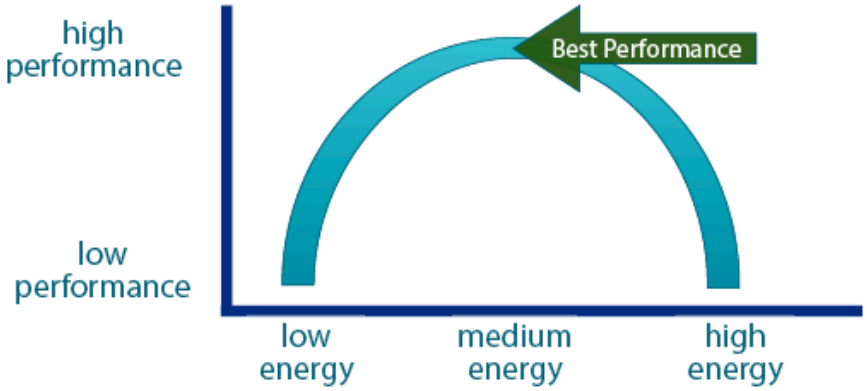

Emotional *Blends*
When you feel two emotions at once, they can combine to make a new feeling.
Disgust
+
Anger
=
Contempt
Back
Next



4) Facilitating Emotions
   - Introduction – conceptually how a helpful mood matches the tone of the task you are trying to work on

- "Energy hill" explanation
    - Yerkes, & Dodson. (1908). The relation of strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology and Psychology, 18*, 459-482.
- Multiple choice questions – where on energy hill – scenarios
- Categorizing non-emotional words into positive/negative tone and high/low energy introduction
    - ANEW Word List (Bradley & Lange)
- Categorizing non-emotional words – 4 quadrant drag-and-drop
- Multiple choice – which emotion is this non-emotional word most like?
- Conclusion

We tend to perform best when we are in a "medium energy" state on that hill.



Back Next

Multiple Choice

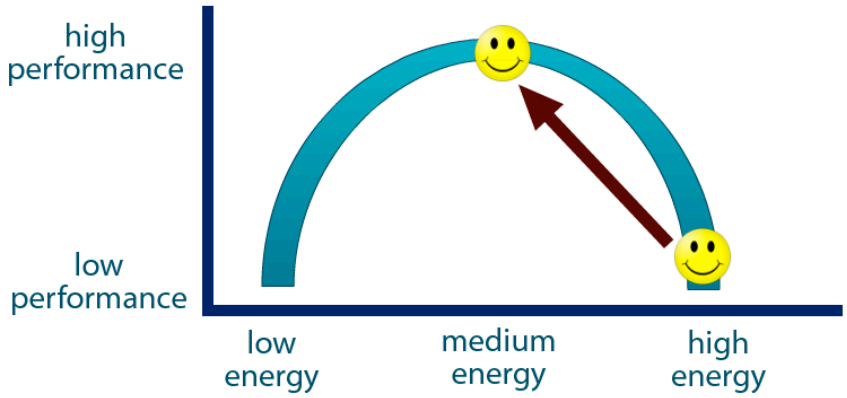
Imagine that you were overcharged for car repairs. You have tried talking to the repairman, who was not helpful. You now ask to speak to the manager.

- (•) Enraged
- ( ) Happy
- ( ) Furious
- ( ) Irritated

Incorrect. Actually, a more neutral mood like 'irritated' will help you remain calm while you speak with the manager . Press the 'Submit' button again to continue.

Back Submit

So if you are feeling very "fired-up" (high energy), you might perform better if you switched to a lower-energy mood.

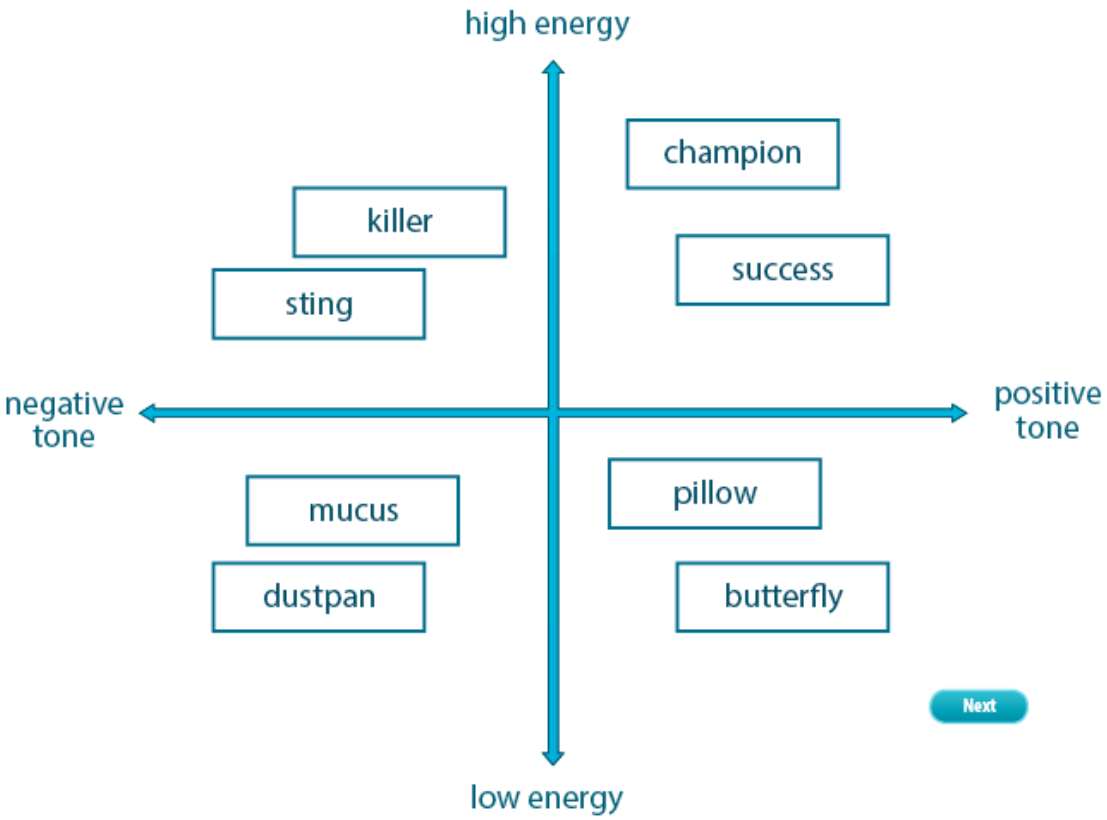


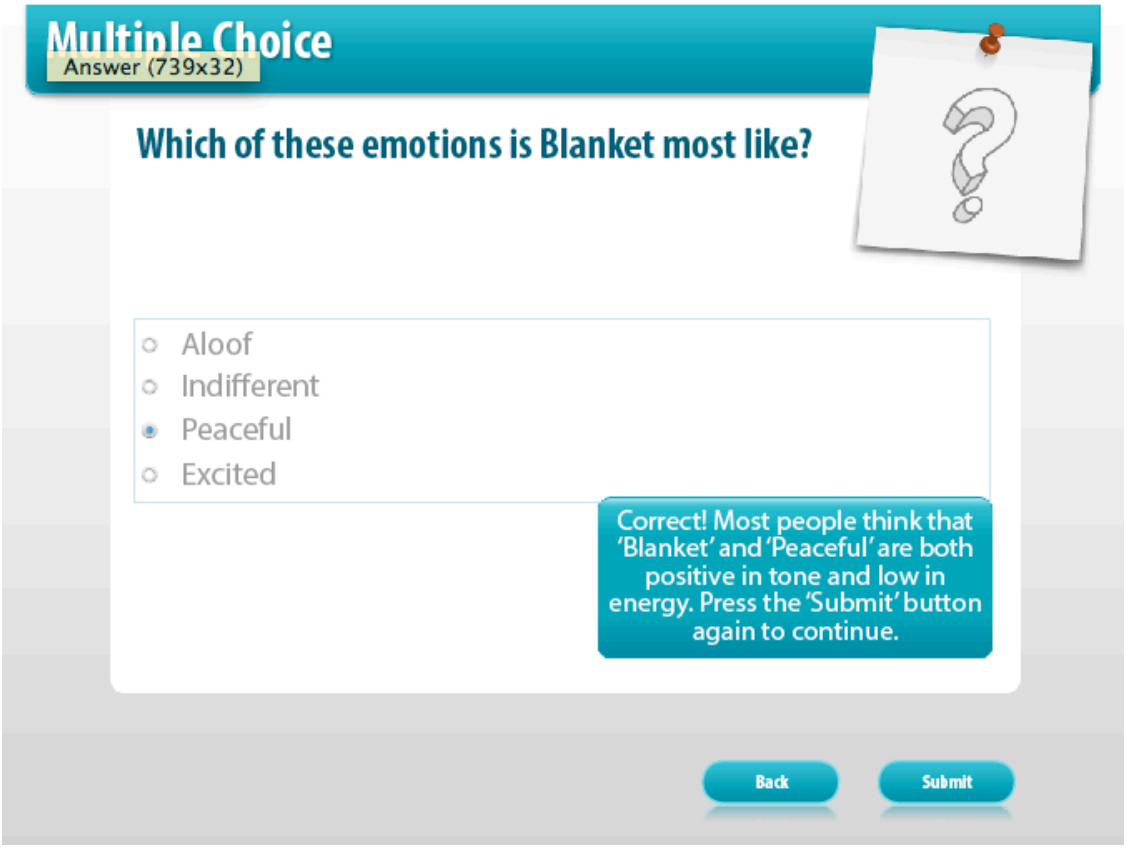
Back Next

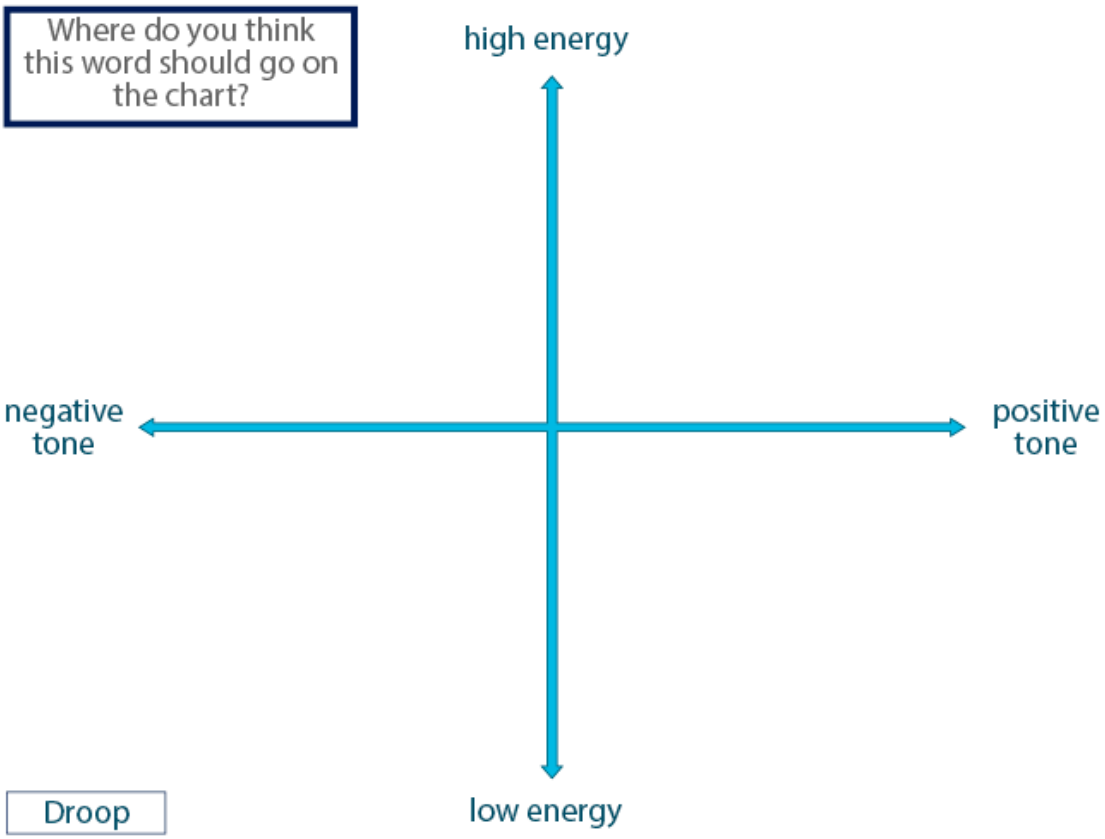
Some words are negative in tone and high in energy, like:

terrified
killer
sting
roach

Back Next


high energy
champion
killer
success
sting
negative tone
positive tone
mucus
pillow
dustpan
butterfly
Next
low energy


Multiple Choice
Answer (739x32)
Which of these emotions is Blanket most like?
Aloof
Indifferent
Peaceful
Excited
Correct! Most people think that 'Blanket' and 'Peaceful' are both positive in tone and low in energy. Press the 'Submit' button again to continue.
Back
Submit



5) Managing Emotions
   - Introduction – advantages
   - Sorting problems into easy to deal with vs difficult to deal with categories
   - Steps to dealing with a problem
   - 2 ways to deal with a problem – find a solution or change the way you think about it
   - Example scenario of character's solvable problem and how he solves it using the steps
   - Change the way you think – positive reappraisal and mindfulness intros
     - Folkman, & Lazarus. (1988). Coping as a mediator of emotion. *Journal of Personality and Social Psychology, 54*(3), 466-475.
   - Positive reappraisal explanation – bigger picture or different person's perspective
     - Schartau, Dalgleish, & Dunn. (2009). Seeing the bigger picture: Training in perspective broadening reduces self-reported affect and psychophysiological response to distressing films and autobiographical memories. *Journal of Abnormal Psychology, 118*(1), 15-27.
     - Finkel, Slotter, Luchies, Walton, & Gross. (2013). A brief intervention to promote conflict reappraisal preserves marital quality over time. *Psychological Science OnlineFirst, doi: 10.1177/0956797612474938.*
   - Example scenario of character's problem – use positive reappraisal strategy – big picture thinking and finding benefits
   - Quiz – benefit finding

- Example scenario of character's problem – use positive reappraisal strategies – perspective taking
- Quiz – multiple choice questions about scenario that requires positive reappraisal
- Mindfulness
    - Garland, Gaylord, & Park. (2009). The role of mindfulness in positive reappraisal. *Explore (NY)*, *5*(1), 37-44.
    - Grossman, Niemann, Schmidt, & Walach. (2004). Mindfulness-based stress reduction and health benefits: A meta-analysis. *Journal of Psychosomatic Research, 57*(1), 35-43.
- Mindfulness 3-part breath
- Mindfulness observing/accepting your body sensations
- Mindfulness observing your thoughts
- Mindfulness exercise – view pictures and notice thoughts/feelings
- Example scenario of character's problem – use positive reappraisal strategy, mindfulness, and problem-solving
- Conclusion

The advantages of being able to manage your emotions

Learning how to manage your emotions is important because it can keep you from getting too worked up or upset about a problem.

Most of the time you can either change the problem or change how you think about it.

Back Next

Drag and drop the problems into the following groups

Easy to deal with	Difficult to deal with
You missed the last bus	Your cat disappeared
There is always a traffic jam on the shortest route to work	Your friend moves far away for a better job
There's no milk in the fridge	Your significant other wants to end your relationship

Notice how the "easy to deal with" problems can be easily solved.

- For instance, if you miss the last bus, you can take a taxi or train.

The "difficult to deal with"problems take more advanced coping.

- For instance, if your cat disappears, you can try to remember your pet fondly to make yourself feel better.

Back Next

In general, there are 3 easy steps to deal with a problem.

- **Step 1:** What is the problem? Size it up!

- **Step 2:** What could I do? Look at different options!

- **Step 3:** Choose what works best!

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Step 2

Frank considers whether there is a solution to his problem or if he just has to change the way he thinks about it.

Find a solution or key to your problem.

OR

Change the way you think about your problem.

Frank thinks he may be able to come up with a solution.

Back Next

The steps of positive reappraisal

First, you have your original view.

Original View

"I am angry because it is raining."

Then, you introduce a new view that is positive.

New View

"Now I get to watch that movie I have been wanting to watch."

Then, you are left with a positive view.

More Positive View

"Today turned out to be a good day because I saw a movie."

Next

Positive Reappraisal Example

How will this affect me today?
I am upset that I didn't get into the program.

How will this affect me next month?
I may be a little upset I am not in the program.

How will this affect me next year?
I can apply again and writing the application will be easier the second time.

How will this affect me in five years?
I probably won't care that I didn't get into the program the first time.

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Quiz: Benefit Finding

Drag the corresponding benefits that would be most helpful in achieving a positive view of the situation into the chart below.

Event	Benefits
Failed a science test	

Don't need to learn this stuff anyways

Teacher is to blame for me failing

Chance to explore new academic areas

Chance to learn new study strategies

Motivation to work harder

No reason to study because I will fail again

Submit

If you are in a situation when you cannot talk to a friend or relative about your problem, try to imagine what they would tell you.

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Next

Positive Reappraisal Example

Event	Benefits
Not accepted into academic program	Applying next year will be easier because I have already gone through the application process.
	I will have more time to work on other projects this semester without having to commit time to the program.

Back Next

Mindful Breathing

- Now try to focus only on your breath going in and out.
- Try to move your breath in and out as the balloon inflates and deflates.
- Inhale through your nose and exhale through your mouth.

Back Next

Observing the body

- Now move your attention up to your torso.
- Can you feel your breath in your lower back, belly, chest, and shoulders?

Back Next

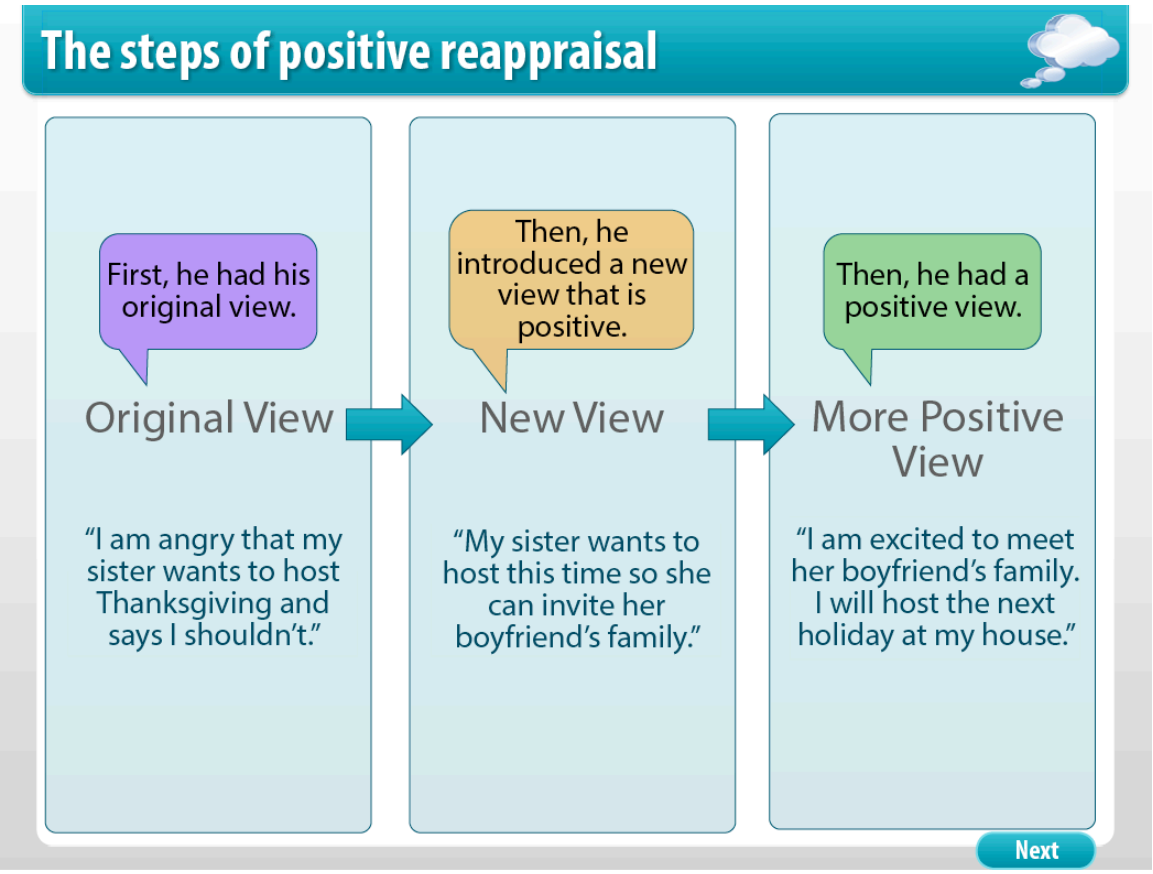
Dealing with a difficult problem

Change the way he thinks about the problem

Carl can also try to change the way he thinks about his high cholesterol levels.

Let's see what Carl thinks are the benefits of his situation...

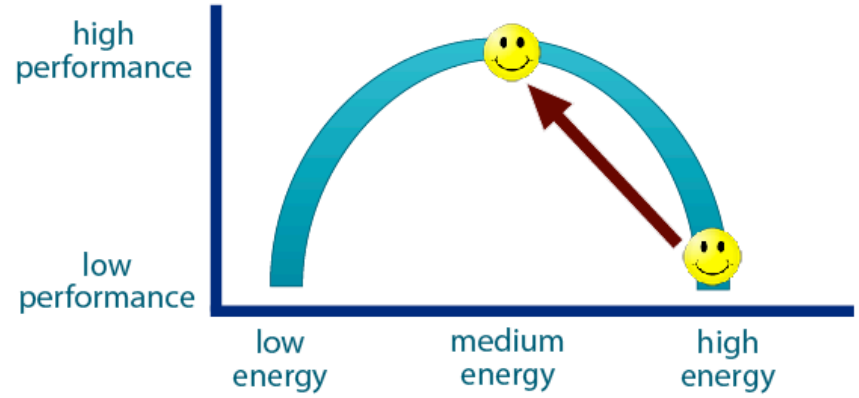
Back Next



6) Conclusion
   - Introduction
   - Review of perceiving
   - Review of facilitating
   - Review of understanding
   - Review of managing
   - Scenarios with characters incorporating all of the lessons
   - Final review

Final Review

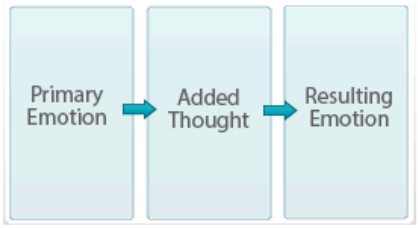
You have learned the importance of controlling your energy level to perform different tasks.

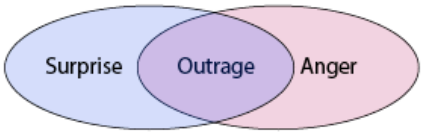


Back Next

Final Review

You have learned how emotions combine and progress over time.





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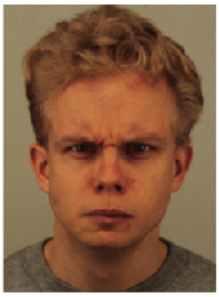
Final Review

You have learned to focus on the eyes and mouth when reading emotional expressions on faces. You have also learned what to look for specifically when identifying specific emotions.



Back Next

Final Review

You have learned how to approach difficult problems.

Find a solution or key to your problem.

Change the way you think about your

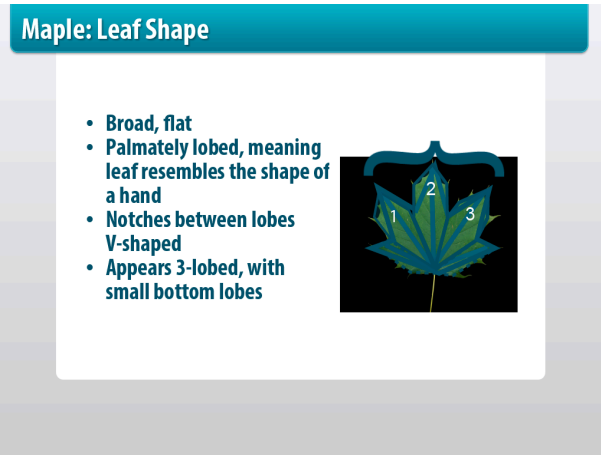
Back Next

External Awareness

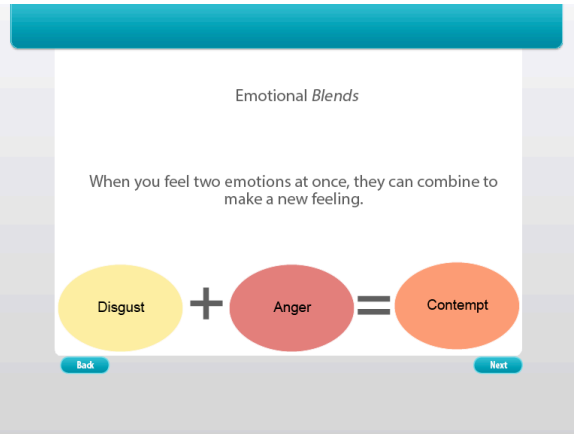
A matched set of training materials has also been developed to serve as a non-emotion-focused control condition. The materials are matched in length, difficulty, and general time requirements, but focus the participant on awareness of the external environment. The modules are matched along the same dimensions as the emotionally based Internal Awareness, including Perceiving, Understanding, Facilitating, and Managing. The pages that follow show a sample of screen images depicting some of the content of these control modules:

Active Treatment: Perceiving Emotions

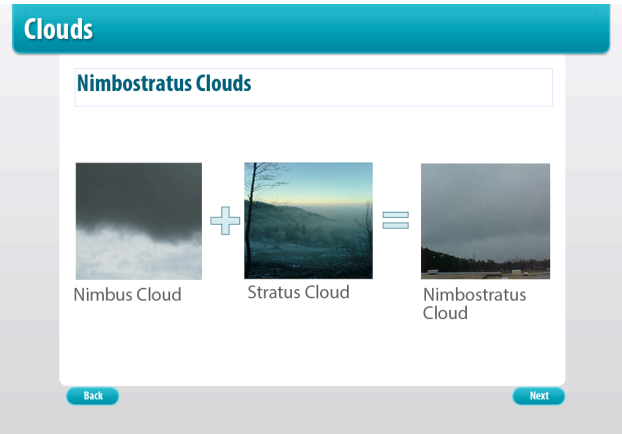
Placebo Control: Perceiving Plants



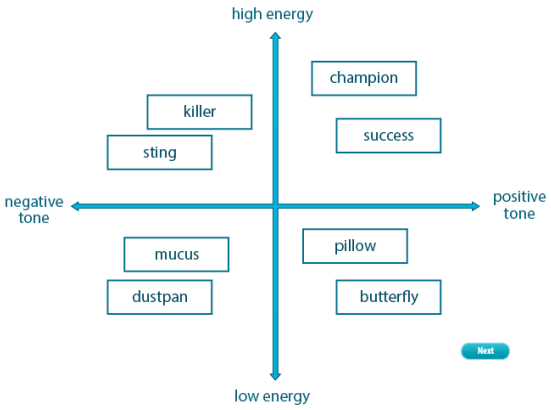
Active Treatment: Understanding Emotions



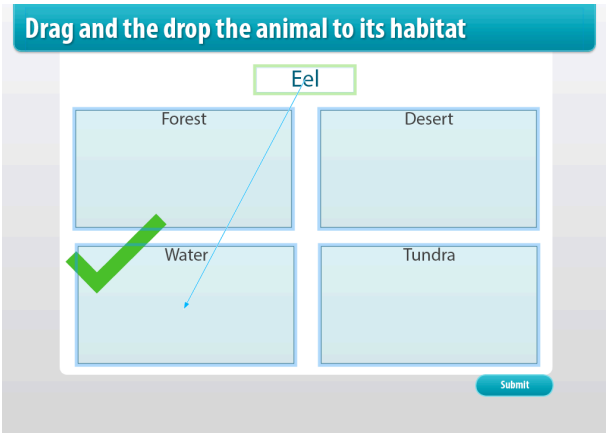
Placebo Control: Understanding Weather



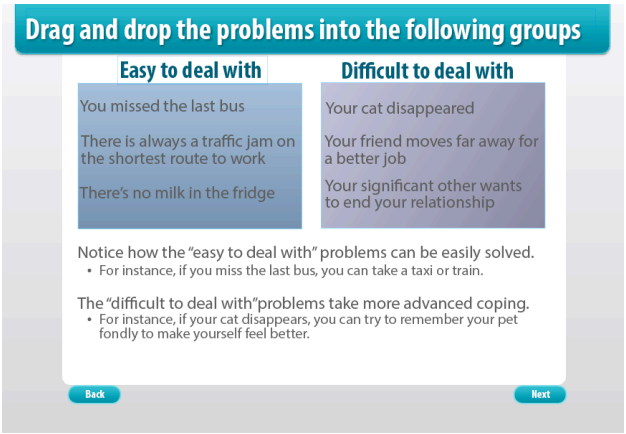
Active Treatment: Facilitating Emotions



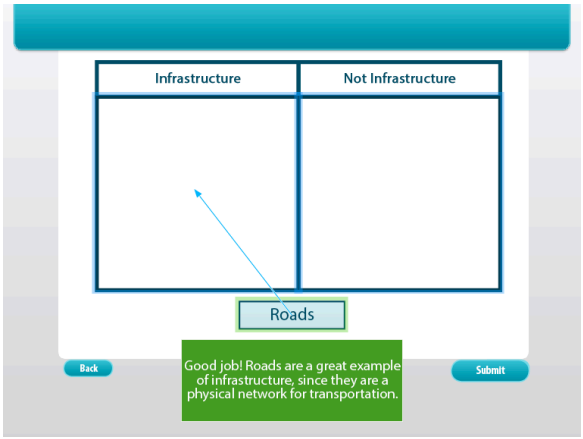
Placebo Control: Animals



Active Treatment: Managing Emotions



Placebo Control: Infrastructure Awareness



Appendix: List of Assessments

1. Pre-Scan Information Questionnaire (PSIQ)
2. Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT V2.0)
3. Bar-On Emotional Quotient Inventory (EQi)
4. Self-Rated Emotional Intelligence Scale (SREIS)
5. Memory Suppression Task Training (2-4 Trials)
6. Memory Suppression Phases: A, B, C
7. Positive and Negative Affect Schedule (PANAS)
8. Memory Suppression Task-Suppression (4^{th} phase) (MST1)
9. Memory Suppression Recall (MST2; NO SCANNING)
10. Memory Suppression Task-Face Interference (MST3)
11. Memory Suppression Task-Scene Interference (MST4)
12. Emotional Distraction Task (EDT)
13. Social Dominance Task (SDT)
14. Food Perception Task (FPT)
15. Food/Activity Decision Task (FDT)
16. BMAT Anger
17. BMAT Fear
18. BMAT Happy
19. BMAT Trustworthy
20. Overt Trustworthiness Task (OTT)
21. Resting fMRI
22. Memory Suppression Task Post Test
23. Emotion Distraction Post Test
24. Masked Affect Post Test
25. Food Recognition Post Test
26. Food Ratings
27. Barratt Impulsivity Scale (BIS11)
28. Connor-Davidson Resilience Scale (CD-RISC)
29. Invincibility Belief Index (IBI)
30. Evaluation of Risks Questionnaire (EVAR)

31. Brief Sensation Seeking Scale (BSSS)
32. Happy Chimeric Test (CFT)
33. Sad Chimeric Test (CFT)
34. Balloon Analogue Risk Task (BART)
35. Ekman 60 Face Test (60 FT)
36. Wechsler Abbreviated Scale of Intelligence (WASI)
37. Karolinska Airport Trustworthiness Test (KATT)
38. Intuition Test
39. Facial Assessment of Trustworthiness Test (FATT)
40. Design Organization Test (DOT) – FORM A and B
41. Iowa Gambling Task (IGT)
42. Revised NEO Personality Inventory (NEO-PI-R)
43. Anxiety Sensitivity Index (ASI)
44. Morningness-Eveningness Questionnaire (MEQ)
45. Courtauld Emotion Control Scale (CECS)
46. Beck Depression Inventory (BDI)
47. Trust Go/NoGo (Form A or X)/Trust Go/NoGo Reversed (Form B or Y)
48. Personality Assessment Inventory (PAI)
49. Humor Appreciation Test (HAT)
50. Global Assessment Tool (GAT)
51. WRAT-3 Reading Assessment
52. Intolerance of Uncertainty Scale
53. Toronto Alexithymia Scale (TAS-20)
54. Social Discounting Task
55. Trait Meta-Mood Scale
56. Emotion Regulation Questionnaire
57. Reading the Mind in the Eyes Task
58. Empathy Quotient Index
59. Beck Anxiety Index (BAI)
60. Mindful Attention Awareness Scale

The Neurobiological Basis and Potential Modification of Emotional Intelligence through Affective / Behavioral Training

W81XWH-09-1-0730

PI: William D. Killgore, Ph.D. **Org:** McLean Hospital **Award Amount:** $551,961

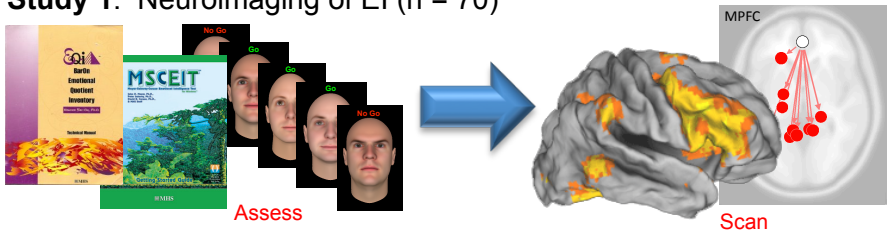
Study/Product Aim(s)

- Validate the two major theories of Emotional Intelligence (EI-Ability vs. Trait) using behavioral testing and neuroimaging.
- Quantify the association between major EI instruments and actual behavioral skills associated with emotional processes.
- Identify the specific brain regions, structures, and systems involved in EI.
- Develop a pilot training program for improving EI capacities.

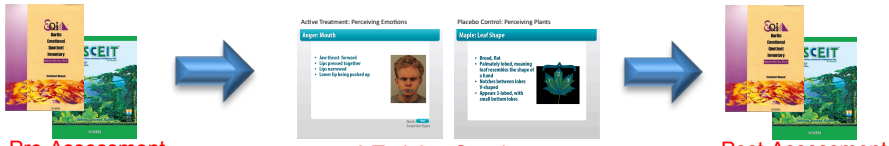
Approach

Two studies: 1) Cross-sectional neuroimaging study of 70 participants who complete EI assessments, emotion tests, and undergo functional and structural neuroimaging; 2) Development of a 6 module EI training program based on scientific data, which was evaluated against a placebo training program in a mixed 2 (group) x 2 (pre-post) longitudinal design in 62 participants.

Study 1: Neuroimaging of EI (n = 70)



Study 2: Training of EI (n = 31 Tx; n = 31 placebo)



Accomplishments: Both Study 1 and Study 2 are ***complete***. Thus far, this study has yielded ***22 peer-reviewed publications*** and ***101 published conference abstracts***.

Activities CY	09	10	11	12	13	14
Study preparations: Study 1						
Data collection: Study 1						
Analysis/dissemination: Study 1						
Study preparations: Study 2						
Data collection: Study 2						
Analysis/dissemination: Study 2						
Estimated Budget ($552K)	**10K**	**132K**	**132K**	**140K**	**69K**	**69K**

Updated: 5 DEC 2014

Goals/Milestones

CY10 Goal – Study 1 preparations, approvals, recruitment, data collection
☑ Completed study preparations early, started data collection early
CY11 Goals – Data collection, quality checks
☑ Test approximately 20 subjects per CY
CY12 Goal – Complete Data collection/analyze and publish findings
☑ Study 1 data collection completed
☑ Data analyzed; 15 papers published; 66 conference presentations
CY13 Goal –Study 2 SOW Modification to develop training program
☑ Modification approved; additional $138K awarded for 1-year extension
☑ Run 60 subjects through EI/placebo training with pre-post assessments
☑ The program successfully improved Total EI and 50% of EI subscales
☑ Data analyzed; *22 peer-reviewed publications*, *101 published abstracts*
☑ Application pending for follow-on funding to validate program in a military sample (DMRDP opportunity W81XWH-14-PHTBI-PHRA)

Budget Expenditure to Date

Awarded Amount:	$551,961
Actual Expenditure:	$550,670

Curriculum Vitae

Date Prepared: **December 2, 2014**

Name: WILLIAM DALE (SCOTT) KILLGORE

Office Address: Suite 7303B
Department of Psychiatry
University of Arizona HSC
1501 North Campbell Ave.
PO Box 245002
Tucson, AZ 85724 United States

Home Address: 3303 North Paseo de los Rios
Apt. 11205
Tucson, AZ 85712 United States

Work Phone: (520) 621-0605

Work Email: killgore@mclean.harvard.edu

Killgore@psychiatry.arizona.edu

Work FAX: (617) 855-2770

Place of Birth: Anchorage, AK

Education

1985	A.A. (Liberal Arts), San Antonio College
1985	A.A.S (Radio-TV-Film), San Antonio College
1990	B.A. (Psychology), Summa cum laude with Distinction, University of New Mexico
1992	M.A. (Clinical Psychology), Texas Tech University
1996	PH.D. (Clinical Psychology), Texas Tech University

Postdoctoral Training

08/95-07/96	Predoctoral Fellow, Clinical Psychology, Yale School of Medicine
08/96-07/97	Postdoctoral Fellow, Clinical Neuropsychology, University of OK Health Sciences Center
08/97-07/99	Postdoctoral Fellow, Clinical Neuropsychology, University of Pennsylvania Medical School
07/99-09/00	Research Fellow, Neuroimaging, McLean Hospital/ Harvard Medical School
09/13-05/14	Certificate in Applied Biostatistics, Harvard Medical School

Faculty Academic Appointments

10/00-08/02	Instructor in Psychology in the Department of Psychiatry Harvard Medical School, Boston, MA
09/02-07/07	Clinical Instructor in Psychology in the Department of Psychiatry Harvard Medical School, Boston, MA

08/07-10/10	Instructor in Psychology in the Department of Psychiatry Harvard Medical School, Boston, MA
04/08-	Faculty Affiliate, Division of Sleep Medicine Harvard Medical School, Boston, MA
10/10-10/12	Assistant Professor of Psychology in the Department of Psychiatry Harvard Medical School, Boston, MA
10/12-	Associate Professor of Psychology in the Department of Psychiatry Harvard Medical School

Appointments at Hospitals/Affiliated Institutions

10/00-08/02	Assistant Research Psychologist, McLean Hospital, Belmont, MA
08/02-07/04	Research Psychologist, Department of Behavioral Biology, Walter Reed Army Institute of Research, Silver Spring, MD
09/02-04/05	Special Volunteer, National Institute on Deafness and Other Communication Disorders (NIDCD), National Institutes of Health (NIH), Bethesda, MD
09/02-07/07	Consultant in Psychology, McLean Hospital, Belmont, MA
08/07-	Research Psychologist, McLean Hospital, Belmont, MA

Other Professional Positions

11/01-08/02	First Lieutenant, Medical Service Corps, United States Army Reserve (USAR)
08/02-07/05	Captain, Medical Service Corps, United States Army
08/05-10/07	Major, Medical Service Corps, United States Army
10/07-07/12	Major, Medical Service Corps, United States Army Reserve (USAR)
10/07-3/10	Chief Psychologist, GovSource, Inc., U.S. Department of Defense Government Contractor
08/08-	Consulting Psychologist, The Brain Institute, University of Utah
07/12-	Lieutenant Colonel, Medical Service Corps, United States Army Reserve (USAR)

Major Administrative Leadership Positions

Local

1988-1989	Undergraduate Teaching Assistant-Introduction to Psychology 102, University of New Mexico
1990-1991	Graduate Teaching Assistant-General Psychology 1300, Texas Tech University
1991-1992	Graduate Teaching Assistant-Psychology of Learning Laboratory 3317, Texas Tech University
2004-2007	Chief, Neurocognitive Performance Branch, Walter Reed Army Institute of Research, Silver Spring, MD
2005-2006	Neuropsychology Postdoctoral Program Training Supervisor, Walter Reed Hospital, Washington, DC
2011-	Co-Director, Social, Cognitive, and Affective Neuroscience Laboratory, McLean Hospital, Belmont, MA

Committee Service

Local

2003 Scientific Review Committee, Walter Reed Army Institute of Research (WRAIR), Silver Spring, MD

2005 Scientific Review Committee, Walter Reed Army Institute of Research (WRAIR), Silver Spring, MD

2012- McLean Hospital Research Committee, McLean Hospital, Belmont, MA

Regional

2005-2006 Undergraduate Honors Thesis Committee, Jessica Richards [Chairperson], University of Maryland, Baltimore County

2011 Scientific Review Committee, U.S. Army Institute of Environmental Medicine (USARIEM), Natick, MA

National

2011- National Network of Depression Centers, Military Task Group

International

2005-2006 Doctoral Thesis Committee, Belinda J. Liddell, University of Sydney, Australia

Professional Societies

1995-1997 American Psychological Association, Member

1998-2000 National Academy of Neuropsychology, Member

2012- American Academy of Sleep Medicine, Member

2014- Organization for Human Brain Mapping, Member

Grant Review Activities

National

2004 University of Alabama, Clinical Nutrition Research Center (UAB CNRC) Pilot/Feasibility Study Program Review Committee

2006 U.S. Small Business Administration, Small Business Technology Transfer (STTR) Program Review Committee

2006 Cognitive Performance Assessment Program Area Steering Committee, U.S. Army Military Operational Medicine Research Program Funding Panel

2007 Cognitive Performance Assessment Program Area Steering Committee, U.S. Army Military Operational Medicine Research Program Funding Panel

2008 United States Army Medical Research and Materiel Command (USAMRMC) Congressionally Directed Medical Research Programs (CDMRP) Extramural Grant Review Panel

2009 NIH-CSR Brain Disorders and Clinical Neuroscience N02 Member Study Conflict Section Review Panel

2009 Sleep Physiology and Fatigue Interventions Program Area Steering Committee, U.S. Army Military Operational Medicine Research Program

2011 National Science Foundation (NSF) Grant Reviewer

2012 National Science Foundation (NSF) Grant Reviewer

International

2009	Scotland, UK, Biomedical and Therapeutic Research Committee, Grant Reviewer
2010	Canada, Social Sciences and Humanities Research Council of Canada, Grant Reviewer
2011	Israel, Israel Science Foundation (ISF), Grant Reviewer
2013	Israel, Israel Science Foundation (ISF), Grant Reviewer

Editorial Activities

2001-2012	Reviewer, Psychological Reports
2001-2012	Reviewer, Perceptual and Motor Skills
2002	Reviewer, American Journal of Psychiatry
2002-2013	Reviewer, Biological Psychiatry
2003	Reviewer, Clinical Neurology and Neurosurgery
2004, 2013	Reviewer, NeuroImage
2004-2006	Reviewer, Neuropsychologia
2004	Reviewer, Journal of Neuroscience
2004	Reviewer, Consciousness and Cognition
2005	Reviewer, Experimental Brain Research
2005	Reviewer, Schizophrenia Research
2005-2012	Reviewer, Archives of General Psychiatry
2005	Reviewer, Behavioral Brain Research
2005-2009	Reviewer, Human Brain Mapping
2005-2013	Reviewer, Psychiatry Research: Neuroimaging
2006	Reviewer, Journal of Abnormal Psychology
2006	Reviewer, Psychopharmacology
2006	Reviewer, Developmental Science
2006	Reviewer, Acta Psychologica
2006	Reviewer, Neuroscience Letters
2006-2014	Reviewer, Journal of Sleep Research
2006-2013	Reviewer, Physiology and Behavior
2006-2014	Reviewer, SLEEP
2007	Reviewer, Journal of Clinical and Experimental Neuropsychology
2008	Reviewer, European Journal of Child and Adolescent Psychiatry
2008	Reviewer, Judgment and Decision Making
2008-2010	Reviewer, Aviation, Space, & Environmental Medicine
2008	Reviewer, Journal of Psychophysiology
2008	Reviewer, Brazilian Journal of Medical and Biological Research
2008	Reviewer, The Harvard Undergraduate Research Journal
2008	Reviewer, Bipolar Disorders
2008-2013	Reviewer, Chronobiology International
2008	Reviewer, International Journal of Obesity
2009	Reviewer, European Journal of Neuroscience
2009-2014	Reviewer, International Journal of Eating Disorders
2009	Reviewer, Psychophysiology
2009	Reviewer, Traumatology
2009	Reviewer, Clinical Medicine: Therapeutics

2009	Reviewer, Acta Pharmacologica Sinica
2009	Reviewer, Collegium Antropologicum
2009	Reviewer, Journal of Psychopharmacology
2009-2014	Reviewer, Obesity
2009	Reviewer, Scientific Research and Essays
2009	Reviewer, Child Development Perspectives
2009-2010	Reviewer, Personality and Individual Differences
2009-2010	Reviewer, Noise and Health
2009-2010	Reviewer, Sleep Medicine
2010	Reviewer, Nature and Science of Sleep
2010	Reviewer, Psychiatry and Clinical Neurosciences
2010	Reviewer, Learning and Individual Differences
2010	Reviewer, Cognitive, Affective, and Behavioral Neuroscience
2010	Reviewer, BMC Medical Research Methodology
2010-2011	Reviewer, Journal of Adolescence
2010-2012	Reviewer, Brain Research
2011	Reviewer, Brain
2011	Reviewer, Social Cognitive and Affective Neuroscience
2011	Reviewer, Journal of Traumatic Stress
2011	Reviewer, Social Neuroscience
2011-2014	Reviewer, Brain and Cognition
2011	Reviewer, Frontiers in Neuroscience
2011-2012	Reviewer, Sleep Medicine Reviews
2012	Reviewer, Journal of Experimental Psychology: General
2012	Reviewer, Ergonomics
2012	Reviewer, Behavioral Sleep Medicine
2012	Reviewer, Neuropsychology
2012	Reviewer, Emotion
2012	Reviewer, JAMA
2012	Reviewer, BMC Neuroscience
2012	Reviewer, Cognition and Emotion
2012	Reviewer, Journal of Behavioral Decision Making
2012	Reviewer, Psychosomatic Medicine
2012-2014	Reviewer, PLoS One
2012	Reviewer, American Journal of Critical Care
2012-2014	Reviewer, Journal of Sleep Disorders: Treatment and Care
2013	Reviewer, Experimental Psychology
2013	Reviewer, Clinical Interventions in Aging
2013	Reviewer, Frontiers in Psychology
2013	Reviewer, Brain Structure and Function
2013	Reviewer, Appetite
2013	Reviewer, JAMA Psychiatry
2014	Reviewer, Acta Psychologica
2014	Reviewer, Neurology
2014	Reviewer, Applied Neuropsychology: Child
2014	Reviewer, Journal of Applied Psychology

Other Editorial Roles

2009-	Editorial Board Member	International Journal of Eating Disorders
2012-	Editor	Datasets in Neuroscience
2012-	Editor	Datasets in Medicine
2012-	Editor	Journal of Sleep Disorders: Treatment and Care

Honors and Prizes

1990	Outstanding Senior Honors Thesis in Psychology, University of New Mexico
1990-1995	Maxey Scholarship in Psychology, Texas Tech University
2001	Rennick Research Award, Co-Author, International Neuropsychological Society
2002	Honor Graduate, AMEDD Officer Basic Course, U.S. Army Medical Department Center and School
2002	Lynch Leadership Award Nominee, AMEDD Officer Basic Course, U.S. Army Medical Department Center and School
2003	Outstanding Research Presentation Award, 2003 Force Health Protection Conference, U.S. Army Center for Health Promotion and Preventive Medicine
2005	Edward L. Buescher Award for Excellence in Research by a Young Scientist, Walter Reed Army Institute of Research (WRAIR) Association
2009	Merit Poster Award, International Neuropsychological Society
2009	Outstanding Research Presentation Award, 2009 Force Health Protection Conference, U.S. Army Center for Health Promotion and Preventive Medicine
2010	Best Paper Award, Neuroscience, 27th U.S. Army Science Conference
2011	Published paper included in *Best of Sleep Medicine 2011*
2011	Blue Ribbon Finalist, 2011 Top Poster Award in Clinical and Translational Research, Society of Biological Psychiatry
2012	Defense Advance Research Projects Agency (DARPA) Young Faculty Award in Neuroscience
2014	Blue Ribbon Finalist, 2014 Top Poster Award in Basic Neuroscience, Society of Biological Psychiatry
2014	Harvard Medical School Excellence in Mentoring Award Nominee
2014	AASM Young Investigator Award (co-author), Honorable Mention, American Academy of Sleep Medicine

Report of Funded and Unfunded Projects

Funding Information

Past

2001-2003	fMRI of Unconscious Affect Processing in Adolescence. N.I.H., 1R03HD41542-01 P.I.: Killgore ($79,000.)

2003-2006 The Effects of Sleep-Loss and Stimulant Countermeasures on Judgment and Decision Making.
U.S. Army Medical Research and Materiel Command (USAMRMC) Competitive Medical Research Proposal Program (CMRP),
P.I.: Killgore (Total Award: $1,345,000.)

2004-2005 Sleep/wake Schedules in 3ID Aviation Brigade Soldiers.
Defense Advanced Research Projects Agency (DARPA)
P.I.: Killgore (Total Award: $60,000.)

2005-2006 Functional Neuroimaging Studies of Neural Processing Changes with Sleep and Sleep Deprivation.
U.S. Army Medical Research and Materiel Command (USAMRMC)
Task Area C (Warfighter Judgment and Decision Making) Program Funding
P.I.: Killgore (Total Award: $219,400.)

2006-2007 Establishing Normative Data Sets for a Series of Tasks to Measure the Cognitive Effects of Operationally Relevant Stressors.
U.S. Army Medical Research and Materiel Command (USAMRMC)
Task Area C (Warfighter Judgment and Decision Making) Program Funding,
P.I.: Killgore (Total Award: $154,000.)

2006-2007 Military Operational Medicine Research Program (MOM-RP), Development of the Sleep History and Readiness Predictor (SHARP).
U.S. Army Medical Research and Materiel Command (USAMRMC)
P.I.: Killgore (Total Award:$291,000.)

Current

2009-2014 The Neurobiological Basis and Potential Modification of Emotional Intelligence through Affective Behavioral Training.
U.S. Army Medical Research and Materiel Command (USAMRMC),
P.I.: Killgore (Total Award: $551,961.)
Major Goal: To identify the neurobiological basis of cognitive and emotional intelligence using functional and structural magnetic resonance imaging.

2011-2014 Effects of Bright Light Therapy on Sleep, Cognition, and Brain Function following Mild Traumatic Brain Injury.
U.S. Army Medical Research and Materiel Command (USAMRMC),
P.I.: Killgore (Total Award: $941,924)
Major Goal: To evaluate the effectiveness of morning exposure to bright light as a treatment for improving in sleep patterns among individuals with post-concussive syndrome. Effects of improved sleep on recovery due to this treatment will be evaluated using neurocognitive testing as well as functional and structural neuroimaging.

2012-2015 Internet Based Cognitive Behavioral Therapy Effects on Depressive Cognitions and Brain function.

U.S. Army Medical Research and Materiel Command (USAMRMC),
Co-PI: Killgore (Total Award: $1,646,045)
Major Goal: To evaluate the effectiveness of an internet-based cognitive behavioral therapy treatment program on improving depressive symptoms, coping and resilience skills, cognitive processing and functional brain activation patterns within the prefrontal cortex.

2012-2014 Multimodal Neuroimaging to Predict Cognitive Resilience Against Sleep Loss
Defense Advance Research Projects Agency (DARPA) Young Faculty Award in Neuroscience
P.I.: Killgore (Total Award: $445,531)
Major Goal: To combine several neuroimaging techniques, including functional and structural magnetic resonance imaging, diffusion tensor imaging, and magnetic resonance spectroscopy to predict individual resilience to 24 hours of sleep deprivation.

2012-2016 A Model for Predicting Cognitive and Emotional Health from Structural and Functional Neurocircuitry following Traumatic Brain Injury
Congressionally Directed Medical Research Program (CDMRP), Psychological Health/Traumatic Brain Injury (PH/TBI) Research Program: Applied Neurotrauma Research Award.
P.I.: Killgore (Total Award: $2,272,098)
Major Goal: To evaluate the relation between axonal damage and neurocognitive performance in patients with traumatic brain injury at multiple points over the recovery trajectory, in order to predict recovery.

2012-2014 Neural Mechanisms of Fear Extinction Across Anxiety Disorders
NIH NIMH
Site Subcontract PI: Killgore (Subcontract Award: $505,065)
Major Goal: To examine the neurocircuitry involved in fear conditioning, extinction, and extinction recall across several major anxiety disorders.

2014-2017 Bright Light Therapy for Treatment of Sleep Problems following Mild TBI.
Psychological Health and Traumatic Brain Injury Research Program (PH/TBI RP) Traumatic Brain Injury Research Award-Clinical Trial.
P.I.: Killgore (Total Award: $1,853,921)
Major Goal: To verify the effectiveness of morning exposure to bright light as a treatment for improving in sleep patterns, neurocognitive performance, brain function, and brain structure among individuals with a recent mild traumatic brain injury.

2014-2018 A Non-pharmacologic Method for Enhancing Sleep in PTSD
P.I.: Killgore (Total Award: $3,821,415)
Major Goal: To evaluate the effectiveness of blue light exposure to modify sleep in PTSD and its effects on fear conditioning/extinction, symptom expression, and brain functioning.

Report of Local Teaching and Training

Laboratory and Other Research Supervisory and Training Responsibilities

2005-2006 1 Fellow for 250 hrs/year, Neuropsychology Postdoctoral Research Training Program

Supervisor, Walter Reed Hospital

2011- 2 Fellows for 2080 hrs/year, Harvard Research Fellow Supervisor, McLean Hospital

Formally Supervised Trainees

1997-1999 David Glahn, Ph.D. Associate Professor, Yale University School of Medicine
Provided mentorship in clinical neuropsychological assessment and research at the University of Pennsylvania Hospital, which resulted in the development of a new psychometric test, 1 co-authored published conference abstract, and 1 co-authored published journal article.

1997-1999 Daniel Casasanto, Ph.D. Assistant Professor, University of Chicago
Supervised this trainee while at the University of Pennsylvania Hospital, which resulted in the development of a new psychometric test, 9 co-authored published conference abstracts, and 5 co-authored published journal articles.

2002-2005 Alexander Vo, Ph.D. Associate Professor, UTMB; Vice President, Electronically Mediated Services, Colorado Access
Served as one of his research mentors at the Walter Reed Army Institute of Research, which resulted in 3 co-authored published conference abstracts, and 3 co-authored published journal articles.

2002-2007 Rebecca Reichardt, M.A. Human Subjects Protection Scientist, USAMRMC
Supervised her research training in my lab at the Walter Reed Army Institute of Research, which resulted in 10 co-authored published conference abstracts, and 2 co-authored published journal articles.

2003-2004 Stan Liu, M.D. Medical Intern, Johns Hopkins Medical School
Supervised his research training in my lab at the Walter Reed Army Institute of Research, which primarily involved training in neuropsychological assessment and sleep research methods.

2003-2004 Neil Arora, B.A. Student, Yale University
Supervised his research project in my lab at the Walter Reed Army Institute of Research and NIH, which primarily involved training in brain imaging analysis and led to 2 co-authored published conference abstracts.

2003-2005 Nancy Grugle, Ph.D. Assistant Professor, Cleveland State University
Supervised her Doctoral Dissertation research project in my lab at the Walter Reed Army Institute of Research, which resulted in 23 co-authored published conference abstracts, and 10 co-authored published journal articles.

2003-2005 Joshua Bailey, B.A. Seminary Student
Supervised his computer programing development and research in my lab at the Walter Reed Army Institute of Research, which resulted in 1 co-authored published conference abstract, and 1 co-authored computer analysis package submitted for U.S. patent.

2003-2006 Athena Kendall, M.A. Lab Manager, Walter Reed Army Medical Center
Supervised part of her masters degree research project and other research work in my lab at the Walter Reed Army Institute of Research, which resulted in 4 co-authored published conference abstracts, and 4 co-authored published journal articles.

2003-2006 Lisa Day, M.S.W. Clinical Social Worker, Washington D.C.
Supervised her research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 3 co-authored published conference abstracts, and 1 co-

authored published journal article.

2004-2005 Merica Shepherd, B.A. Laboratory Coordinator
Supervised her research training in my lab at the Walter Reed Army Institute of Research, which primarily involved training in neuropsychological assessment and sleep research methods.

2004-2005 Cynthia Hawes, B.A. Research Program Coordinator
Supervised her research training in my lab at the Walter Reed Army Institute of Research, which primarily involved training in neuropsychological assessment and sleep research methods.

2004-2006 Christopher Li, B.A. Graduate Student
Supervised his research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 3 co-authored published conference abstracts, and 1 co-authored published journal article.

2004-2007 Jessica Richards, M.S. Ph.D. Student, University of Maryland College Park
Served as Chair of her Senior Honors Thesis Committee and supervised her research work in my lab at the Walter Reed Army Institute of Research, which resulted in 8 co-authored published conference abstracts, a senior honors thesis, and 2 co-authored published journal articles.

2004-2007 Erica Lipizzi, M.A. Graduate Student, Emory University
Supervised her research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 16 co-authored published conference abstracts, and 12 co-authored published journal articles.

2004-2007 Brian Leavitt, B.S. Research Technician, Walter Reed Army Institute of Research
Supervised his research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 4 co-authored published conference abstracts, and 1 co-authored published journal article.

2004-2007 Rachel Newman, M.S. Senior Laboratory Manager, Walter Reed
Supervised her research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 6 co-authored published conference abstracts, and 1 co-authored published journal article.

2004-2007 Alexandra Krugler, B.S. Medical Student, Louisiana State University
Supervised her research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 5 co-authored published conference abstracts, and 1 co-authored published journal article.

2005 Amy Conrad, PH.D. Clinical Psychologist, Washington D.C.
Supervised her research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 4 co-authored published conference abstracts, and 1 co-authored published journal article.

2005-2006 Nathan Huck, PH.D. Clinical Neuropsychologist, Walter Reed Army Institute of Research
Served as his post-doctoral research training supervisor at the Walter Reed Army Institute of Research, which resulted in 1 co-authored published conference abstract and 1 co-authored published journal article.

2005-2006 Ellen Kahn-Greene, Ph.D. Post-Doctoral Fellow, Boston VA
Supervised her research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 7 co-authored published conference abstracts and 5 co-authored published journal articles.

2005-2006 Alison Muckle, B.A. Research Technician
Supervised her research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 1 co-authored published conference abstract and 1 co-authored published journal article.

2005-2006 Christina Murray, B.S. Medical Student, Drexel University
Supervised her research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 2 co-authored published conference abstracts.

2005-2007 Gautham Ganesan, M.D. Medical Student, UC Irvine
Supervised his research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 1 co-authored published conference abstract and 1 co-authored published journal article.

2005-2007 Dante Picchioni, Ph.D. Research Psychologist, Walter Reed Army Institute of Research
Supervised part of his post-doctoral brain imaging research training at the Walter Reed Army Institute of Research, which resulted in 1 co-authored published conference abstract and 1 co-authored published journal article.

2006-2007 Tracy Rupp, Ph.D. Research Psychologist, Walter Reed Army Institute of Research
Supervised part of her post-doctoral sleep research training at the Walter Reed Army Institute of Research, which resulted in 17 co-authored conference abstracts and 2 co-authored published journal articles.

2006-2007 Kacie Smith, B.A. Study Manager, Walter Reed Army Institute of Research
Supervised her research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 7 co-authored published conference abstracts.

2006-2007 Shane Smith, B.S. Medical Student, University of the West Indies
Served as his research mentor at the Walter Reed Army Institute of Research, which primarily involved training in neuropsychological assessment and sleep research methods.

2006-2007 Shanelle McNair Research Technician, Walter Reed Army Institute of Research
Supervised her research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 1 co-authored published article.

2006-2007 George Watlington Research Technician, Walter Reed Army Institute of Research
Supervised his research training and work in my lab at the Walter Reed Army Institute of Research, which resulted in 1 co-authored published article.

2008 Grady O'Brien Undergraduate Student
Served as his summer volunteer research mentor at McLean Hospital, which resulted in 1 oral research presentation

2008-2009 Alex Post Undergraduate Student, Carnegie Mellon University
Served as his summer volunteer research mentor at McLean Hospital, which resulted in 2 oral research presentations and 1 co-authored published abstract.

2008-2009 Lauren Price, B.A. Senior Clinical Research Assistant, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital, which resulted in 11 co-authored published conference abstracts and 4 co-authored published articles.

2009-2013 Zachary Schwab, B.S. Medical Student, University of Kansas
Supervised his research training and work in my lab at the McLean Hospital, which resulted in 79 co-authored published conference abstracts and 15 co-authored published

articles.

2009-2011 Melissa Weiner, B.S. Graduate Student, Yale School of Public Health
Supervised her research training and work in my lab at the McLean Hospital, which resulted in 35 co-authored published conference abstracts and 7 co-authored published articles.

2010-2011 Norah Simpson, Ph.D. Post-Doctoral Fellow, Beth Israel Deaconess/Harvard Medical School
Served as a research mentor on her federal K-Award grant application.

2010-2012 Vincent Capaldi, M.D. Medical Resident, Walter Reed Army Medical Ctr.
Served as his post-doctoral research mentor, which resulted in 1 co-authored published conference abstract and 2 co-authored published articles.

2010-2011 Christina Song Undergraduate Student, Smith College
Served as her summer volunteer research mentor at McLean Hospital, which resulted in 1 co-authored published abstract.

2011 Jill Kizielewicz Undergraduate Student, Hamilton College
Served as her summer volunteer research mentor at McLean Hospital, which resulted in 1 co-authored published abstract.

2011-2013 Sophie DelDonno, B.A. Doctoral Student, University of Illinois, Chicago
Supervised her research training and work in my lab at the McLean Hospital, which resulted in 34 co-authored published conference abstracts and 9 co-authored published articles.

2011- Maia Kipman, B.A. Research Assistant, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital, which resulted in 42 co-authored published conference abstracts and 10 co-authored published articles.

2011 Michael Covell, B.A. Graduate Student, Baruch College
Served as one of his research mentors at McLean Hospital, which resulted in 4 co-authored published conference abstracts, and 1 co-authored published article.

2011- Mareen Weber, Ph.D. Instructor, Harvard Medical School
Supervised her post-doctoral research training and work in my lab at the McLean Hospital, which has resulted in 49 co-authored published conference abstracts, 15 co-authored published articles, 1co-authored book chapter, 1 travel award, five federal grant submissions, and 2 successfully funded grants.

2012- Julia Cohen, Ph.D. Post-Doctoral Fellow, Harvard Medical School
Served as one of her research mentors at McLean Hospital, which resulted in 6 co-authored published conference abstracts and 1 peer-reviewed publication.

2012- Christian Webb, Ph.D. Post-Doctoral Fellow, Harvard Medical School
Currently supervising his post-doctoral research training and work in my lab at the McLean Hospital, which has resulted in 9 co-authored published conference abstracts and 6 peer-reviewed publications.

2012- Hannah Gogel, B.S. Research Assistant, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital, which resulted in 21 co-authored published conference abstracts and 4 co-authored published articles.

2012- Olga Tkachenko, A.B. Research Assistant, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital, which resulted in 23 co-authored published conference abstracts and 4 co-authored published articles.

2012- Lilly Preer, B.A. Research Assistant, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital, which resulted in 22 co-authored published conference abstracts and 3 co-authored published articles.

2012-2013 Elizabeth Mundy, Ph.D Postdoctoral Fellow, Harvard Medical School
Supervised her post-doctoral research training and work in my lab at the McLean Hospital, which resulted in 3 co-authored published conference abstracts and 2 co-authored published articles.

2012- John S. Bark, B.A. Lab Volunteer, McLean Hospital
Supervised his research training and work in my lab at the McLean Hospital, which resulted in 5 co-authored published conference abstracts, and 2 co-authored published articles.

2013- Shreya Divatia, B.S. Research Assistant, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital, which resulted in 9 co-authored published conference abstracts.

2013- Lauren Demers, B.A. Research Assistant, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital, which resulted in 10 co-authored published conference abstracts.

2013- Jiaolong Cui, Ph.D Postdoctoral Fellow, Harvard Medical School
Supervised his post-doctoral research training and work in my lab at the McLean Hospital, which resulted in 9 co-authored published conference abstracts.

2013- Allison Jorgensen Lab Volunteer, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital, which resulted in 2 co-authored published conference abstracts.

2013 Leslie Amrein Lab Volunteer, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital.

2013 Alexa Curhan Lab Volunteer, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital.

2013-2014 Kate Manganello High School Lab Volunteer, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital.

2013-2014 Mia Kaminsky High School Lab Volunteer, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital.

2013-2014 Jennifer Buchholz Research Assistant, McLean Hospital
Supervised her research training and work in my lab at the McLean Hospital.

2014 Joseph Dagher, Ph.D. Assistant Professor, University of Arizona
Mentored his K-Award and CECS grant applications.

2014 Ryan Smith, B.S. PhD Candidate, University of Arizona
Mentored his F32- grant application.

2014 John Vanuk, B.A. Research Assistant, University of Arizona
Supervised his research training in my lab.

2014 Sarah Markowski Research Assistant, University of Arizona
Supervised her research training in my lab.

2014 Derek Pisner, B.S. Research Assistant, University of Arizona
Supervised his research training in my lab.

2014 Bradley Shane, B.S. Research Assistant, University of Arizona
Supervised his research training in my lab.

2014 Andrew Fridman, B.A. Research Assistant, University of Arizona
Supervised his research training in my lab.

2014 Anna Alkozei, Ph.D. Postdoctoral Fellow, University of Arizona
Supervised her post-doctoral research training and work in my lab.

Local Invited Presentations

2000 The Neurobiology of Emotion in Children, McLean Hospital
Lecturer: 30 participants, 2 hours contact time per year, 10 hours prep time per year.
[Invited Lecture]

2001 The Neurobiology of Emotion in Children and Adolescents, McLean Hospital
Lecturer: 60 participants, 2 hours contact time per year, 10 hours prep time per year.
[Invited Lecture]

2001 Using Functional MRI to Study the Developing Brain, Judge Baker Children's Center
Lecturer: 8 participants, 2 hours contact time per year, 10 hours prep time per year *[Invited Seminar]*

2005 Briefing to the Chairman of the Congressional Committee on Strategies to Protect the Health of Deployed U.S. Forces, John H. Moxley, on the Optimization of Judgment and Decision Making Capacities in Soldiers Following Sleep Deprivation, Walter Reed Army Institute of Research, Washington, DC*[Invited Lecture]*

2005 Lecture on Functional Neuroimaging, Cognitive Assessment, and the Enhancement of Soldier Performance, Walter Reed Army Institute of Research, Washington, DC *[Invited Lecture]*

2006 Lecture on Optimization of Judgment and Decision Making Capacities in Soldiers Following Sleep Deprivation, Brain Imaging Center, McLean Hospital, Belmont MA *[Invited Lecture]*

2006 Briefing to the Chairman of the Cognitive Performance Assessment Program Area Steering Committee, U.S. Army Military Operational Medicine Research Program, entitled Optimization of Judgment and Decision Making Capacities in Soldiers Following Sleep Deprivation, Walter Reed Army Institute of Research *[Invited Lecture]*

2010 Lecture on Patterns of Cortico-Limbic Activation Across Anxiety Disorders, Center for Anxiety, Depression, and Stress, McLean Hospital, Belmont, MA *[Invited Lecture]*

2010 Lecture on Cortico-Limbic Activation Among Anxiety Disorders, Neuroimaging Center, McLean Hospital, Belmont, MA *[Invited Lecture]*

2011 Lecture on Shared and Differential Patterns of Cortico-Limbic Activation Across Anxiety Disorders, McLean Research Day Brief Communications, McLean Hospital, Belmont, MA *[Invited Lecture]*

2012 Briefing to GEN (Ret) George Casey Jr., former Chief of Staff of the U.S. Army, entitled Research for the Soldier. McLean Hospital, Belmont, MA. *[Invited Lecture]*

2014 Lecture entitled Sleep Loss, Brain Function, and Cognitive Performance, presented to the Psychiatric Genetics and Translational Research Seminar, Massachusetts General Hospital/Harvard Medical School, Boston, MA *[Invited Lecture]*

2014 Grand Rounds Lecture entitled Sleep Loss, Brain Function, and Performance of the Emotional-Executive System. University of Arizona Psychiatry Grand Rounds, Tucson, AZ *[Invited Lecture]*

2014 Psychology Department Colloquium entitled Sleep Loss, Brain Function, and Performance of the Emotional-Executive System. University of Arizona Department of Psychology, Tucson, AZ *[Invited Lecture]*

2014 Lecture entitled Supporting Cognitive and Emotional Health in Warfighters. Presented to the Senior Vice President for the Senior Vice President for Health Sciences and Dean of the Medical School, University of Arizona, Tucson, AZ *[Invited Lecture]*

Report of Regional, National and International Invited Teaching and Presentations

Invited Presentations and Courses

Regional

2002 Cortico-Limbic Activation in Adolescence and Adulthood, Youth Advocacy Project, Cape Cod, MA
Lecturer: 45 participants, 2 hours contact time per year, 10 hours prep time per year
[Invited Lecture]

2006 Lecture on Norming a Battery of Tasks to Measure the Cognitive Effects of Operationally Relevant Stressors, Cognitive Performance Assessment Program Area Steering Committee, U.S. Army Military Operational Medicine Research Program, Washington, DC*[Invited Lecture]*

2007 Lecture on Cerebral Responses During Visual Processing of Food, U.S. Army Institute of Environmental Medicine, Natick, MA*[Invited Lecture]*

2007 Briefing on the Measurement of Sleep-Wake Cycles and Cognitive Performance in Combat Aviators, U.S. Department of Defense, Defense Advanced Research Projects Agency (DARPA), Washington, DC

2008 Lecture on Sleep Deprivation, Executive Function, and Resilience to Sleep Loss; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2008 Lecture on the Role of Research Psychology in the Army; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA*[Invited Lecture]*

2008 Lecture on Combat Stress Control: Basic Battlemind Training; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA*[Invited Lecture]*

2009 Lecture entitled Evaluate a Casualty, Prevent Shock, and Prevent Cold Weather injuries; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA*[Invited Lecture]*

2009 Lecture on Combat Exposure and Sleep Deprivation Effects on Risky Decision-Making; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2009 Lecture on the Sleep History and Readiness Predictor (SHARP); 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2009 Lecture on The Use of Actigraphy for Measuring Sleep in Combat and Military Training; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2010 Lecture entitled Casualty Evaluation; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2010 Lecture entitled Combat Stress and Risk-Taking Behavior Following Deployment; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2010 Lecture entitled Historical Perspectives on Combat Medicine at the Battle of Gettysburg; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2010 Lecture entitled Sleep Loss, Stimulants, and Decision-Making; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2010 Lecture entitled PTSD: New Insights from Brain Imaging; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2011 Lecture entitled Effects of bright light therapy on sleep, cognition and brain function after mild traumatic brain injury; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2011 Lecture entitled Laboratory Sciences and Research Psychology in the Army; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2011 Lecture entitled Tools for Assessing Sleep in Military Settings; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2011 Lecture entitled The Brain Basis of Emotional Trauma and Practical Issues in Supporting Victims of Trauma, U.S. Department of Justice, United States Attorneys Office, Serving Victims of Crime Training Program, Holyoke, MA *[Invited Lecture]*

2011 Lecture entitled The Brain Altering Effects of Traumatic Experiences; 105th Reinforcement Training Unit (RTU), U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2012 Lecture entitled Sleep Loss, Caffeine, and Military Performance; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2012 Lecture entitled Using Light Therapy to Treat Sleep Disturbance Following Concussion; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2013 Lecture entitled Brain Responses to Food: What you See Could Make you Fat; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2013 Lecture entitled Predicting Resilience Against Sleep Loss; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2014 Lecture entitled Get Some Shut-Eye or Get Fat: Sleep Loss Affects Brain Responses to Food; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

2014 Lecture entitled Emotional Intelligence: Developing a Training Program; 105th IMA Detachment, U.S. Army Reserve Center, Boston, MA *[Invited Lecture]*

National

2000 Lecture on the Neurobiology of Emotional Development in Children, 9th Annual Parents as Teachers Born to Learn Conference, St. Louis, MO *[Invited Lecture]*

2002 Lecture on the Changes in the Lateralized Structure and Function of the Brain during Adolescent Development, Walter Reed Army Institute of Research, Washington, DC*[Invited Lecture]*

2004 Lecture on Sleep Deprivation, Cognition, and Stimulant Countermeasures: Seminar Presented at the Bi-Annual 71F Research Psychology Short Course, Ft. Detrick, MD, U.S. Army Medical Research and Materiel Command *[Invited Lecture]*

2004 Lecture on the Regional Cerebral Blood Flow Correlates of Electroencephalographic Activity During Stage 2 and Slow Wave Sleep: An H215O PET Study: Presented at the Bi-Annual 71F Research Psychology Short Course, Ft. Detrick, MD, U.S. Army Medical Research and Materiel Command*[Invited Lecture]*

2004 Oral Platform Presentation: Regional cerebral metabolic correlates of electroencephalographic activity during stage-2 and slow-wave sleep: An H215O PET Study, 18th Associated Professional Sleep Societies Annual Meeting, Philadelphia, PA.

2005 Lecture on The Sleep History and Readiness Predictor: Presented to the Medical Research and Materiel Command, Ft. Detrick, MD*[Invited Lecture]*

2006 Lecture on The Sleep History and Readiness Predictor: Presented at the Bi-Annual 71F Research Psychology Short Course, Ft. Rucker, AL, U.S. Army Medical Research and Materiel Command*[Invited Lecture]*

2007 Lecture on the Effects of Fatigue and Pharmacological Countermeasures on Judgment and Decision-Making, U.S. Army Aeromedical Research Laboratory, Fort Rucker, AL *[Invited Lecture]*

2008 Lecture on the Validation of Actigraphy and the SHARP as Methods of Measuring Sleep and Performance in Soldiers, U.S. Army Aeromedical Research Laboratory, Fort Rucker, AL*[Seminar]*

2009 Lecture on Sleep Deprivation, Executive Function, and Resilience to Sleep Loss: Walter Reed Army Institute of Research AIBS Review, Washington DC*[Invited Lecture]*

2009 Lecture Entitled: Influences of Combat Exposure and Sleep Deprivation on Risky Decision-Making, Evans U.S. Army Hospital, Fort Carson, CO*[Invited Lecture]*

2009 Lecture on Making Bad Choices: The Effects of Combat Exposure and Sleep Deprivation on Risky Decision-Making, 4th Army, Division West, Quarterly Safety Briefing to the Commanding General and Staff, Fort Carson, CO*[Invited Lecture]*

2009 Symposium Entitled: Sleep Deprivation, Judgment, and Decision-Making, 23rd Annual Meeting of the Associated Professional Sleep Societies, Seattle, WA *[Invited Symposium]*

2009 Symposium Session Moderator: Workshop on Components of Cognition and Fatigue: From Laboratory Experiments to Mathematical Modeling and Operational Applications, Washington State University, Spokane, WA *[Invited Speaker]*

2009 Lecture on Comparative Studies of Stimulant Action as Countermeasures for Higher Order Cognition and Executive Function Impairment that Results from Disrupted Sleep Patterns, Presented at the NIDA-ODS Symposium entitled: Caffeine: Is the Next Problem Already Brewing, Rockville, MD *[Invited Lecture]*

2010 Oral Platform Presentation: Sleep deprivation selectively impairs emotional aspects of cognitive functioning, 27th Army Science Conference, Orlando, FL.

2010 Oral Platform Presentation: Exaggerated amygdala responses to masked fearful faces are specific to PTSD versus simple phobia, 27th Army Science Conference, Orlando, FL.

2011 Lecture Entitled: The effects of emotional intelligence on judgment and decision making, Military Operational Medicine Research Program Task Area C, R & A Briefing, Walter Reed Army Institute of Research, Silver Spring, MD *[Invited Lecture]*

2011 Lecture Entitled: Effects of bright light therapy on sleep, cognition, brain function, and neurochemistry following mild traumatic brain injury, Military

Operational Medicine Research Program Task Area C, R & A Briefing, Walter Reed Army Institute of Research, Silver Spring, MD *[Invited Lecture]*

2012 Oral Symposium Presentation: Shared and distinctive patterns of cortico-limbic activation across anxiety disorders, 32nd Annual Conference of the Anxiety Disorders Association of America, Arlington, VA. *[Invited Symposium]*

2012 Lecture Entitled: Effects of bright light therapy on sleep, cognition, brain function, and neurochemistry following mild traumatic brain injury, Military Operational Medicine Research Program In Progress Review (IPR) Briefing, U.S. Army Medical Research and Materiel Command, Fort Detrick, MD *[Invited Lecture]*

2013 Lecture entitled Brain responses to visual images of food: Could your eyes be the gateway to excess? Presented to the NIH Nutrition Coordinating Committee and the Assistant Surgeon General of the United States, Bethesda, MD *[Invited Lecture]*

2013 Lecture Entitled: Update on the Effects of Bright light therapy on sleep, cognition, brain function, and neurochemistry following mild traumatic brain injury, Military Operational Medicine Research Program In Progress Review (IPR) Briefing, U.S. Army Medical Research and Materiel Command, Fort Detrick, MD *[Invited Lecture]*

2013 Lecture Entitled: Internet Based Cognitive Behavioral Therapy: Effects on Depressive Cognitions and Brain Function, Military Operational Medicine Research Program In Progress Review (IPR) Briefing, U.S. Army Medical Research and Materiel Command, Fort Detrick, MD [*Invited Lecture*]

2013 Symposium Entitled: Predicting Resilience Against Sleep Loss, United States Military Academy at West Point, West Point, NY [*Invited Symposium*].

2014 Symposium Entitled: Operating Under the Influence: The Effects of Sleep Loss and Stimulants on Decision-Making and Performance, Invited Faculty Presenter at the 34th Annual Cardiothoracic Surgery Symposium (CREF), San Diego, CA [*Invited Symposium*].

2014 Symposium Entitled: The Effects of Sleep Loss on Food Preference, SLEEP 2014, Minneapolis, MN *[Invited Symposium]*

2014 Lecture Entitled: Internet Based Cognitive Behavioral Therapy: Effects on Depressive Cognitions and Brain Function, Military Operational Medicine Research Program In Progress Review (IPR) Briefing, U.S. Army Medical Research and Materiel Command, Fort Detrick, MD *[Invited Lecture]*

International

1999 Oral Platform Presentation: Functional MRI lateralization during memory encoding predicts seizure outcome following anterior temporal lobectomy, 27th Annual Meeting of the International Neuropsychological Society, Boston, MA.

2001 Oral Platform Presentation: Sex differences in functional activation of the amygdala during the perception of happy faces, 29th Annual Meeting of the International Neuropsychological Society, Chicago, IL.

2002 Oral Platform Presentation: Developmental changes in the lateralized activation of the prefrontal cortex and amygdala during the processing of facial affect, 30th Annual Meeting of the International Neuropsychological Society, Toronto, Ontario, Canada.

2002 Oral Platform Presentation: Gray and white matter volume during adolescence correlates with cognitive performance: A morphometric MRI study, 30th Annual Meeting of the International Neuropsychological Society, Toronto, Ontario, Canada.

2007 Symposium on Cortical and Limbic Activation in Response to Visual Images of Low and High-Caloric Foods, 6th Annual Meeting of the International Society for Behavioral Nutrition and Physical Activity (ISBNPA), Oslo, Norway *[Invited Lecture]*

2008 Lecture on Sleep Deprivation, Executive Function, & Resilience to Sleep Loss, First Franco-American Workshop on War Traumatism, IMNSSA, Toulon, France *[Invited Lecture]*

2012 Oral Platform Presentation: Shared and unique patterns of cortico-limbic activation across anxiety disorders. 40th Meeting of the International Neuropsychological Society, Montreal, Canada.

Report of Clinical Activities and Innovations

Current Licensure and Certification

2001- Clinical Psychologist, New Hampshire

Practice Activities

1991- Psychology, Clinical, Psychology Clinic, Texas Tech University, Lubbock, TX
1995 Clinical Activity Description: Provided psychotherapy and other supervised psychological services for a broad spectrum of client problems. Duties included regular therapy contacts with four to eight clients per week for approximately four years. Clients ranged in age from preschool through middle age. Clinical responsibilities included intake evaluations, formal testing and assessment, case formulation and treatment plan development, and delivery of a wide range of psychotherapy services including crisis intervention, behavior modification, short-term cognitive restructuring, and long-term psychotherapy.
Patient Load: 6/week

1993-1995 Psychology, Neuropsychology, Methodist Hospital Rehabilitation Institute, Lubbock, TX
Clinical Activity Description: A two year placement consisting of two days per week within a large rehabilitation unit of a major regional medical center. Responsibilities included administration, scoring, and writing of neuropsychological assessments/reports, primarily emphasizing the Halstead-Reitan Neuropsychological Battery. Assessment services were provided on both inpatient and outpatient basis.
Patient Load: 2/week

1995-1996 Psychology, Neuropsychology, Yale University School of Medicine, Connecticut Mental Health Center
Clinical Activity Description: Neuropsychological and psychodiagnostic assessment of chronic and severe mentally ill patients. Duties included patient interviewing, test administration, scoring, interpretation, and report writing. Assessment and consultation services were provided for both the inpatient and outpatient units.
Patient Load: 2/week

1995-1996 Psychology, Clinical, Yale University School of Medicine, West Haven Mental Health Clinic
Clinical Activity Description: Provided short-term, long-term, and group psychotherapy services, consultation, and psychological assessments for adults, children, and families. Duties also included co-leading a regular outpatient group devoted to treatment of moderate to severe personality disorders.
Patient Load: 12/week

1996-1997 Psychology, Neuropsychology, University of Oklahoma Health Sciences Center
Clinical Activity Description: Full-time placement in the Neuropsychological Assessment Laboratory, which meets INS/Division 40 guidelines for post-doctoral training in clinical neuropsychology. Responsibilities included comprehensive neuropsychological assessment and consultation services, including test administration, scoring, interpretation, and report writing. Regular outpatient psychotherapy was also provided for approximately two patients per week.
Patient Load: 4/week

1997-1999 Psychology, Neuropsychology, University of Pennsylvania Medical Center
Clinical Activity Description: Full-time two-year placement in the Department of Neurology, which meets INS/Division 40 guidelines for post-doctoral training in clinical neuropsychology.

Responsibilities included neuropsychological assessment, consultation, and psychotherapy services for the Departments of Neurology and Neurosurgery.
Patient Load: 3/week

Report of Education of Patients and Service to the Community

Recognition

2003-2007 Who's Who in America, Marquis Who's Who
2004-2005 Who's Who in Medicine and Healthcare, Marquis Who's Who

Report of Scholarship

Publications

Peer reviewed publications in print or other media

A) Research Investigations:

1. **Killgore WD**. The Affect Grid: a moderately valid, nonspecific measure of pleasure and arousal. Psychol Rep. 83(2):639-42, 1998.

2. **Killgore WD**. Empirically derived factor indices for the Beck Depression Inventory. Psychol Rep. 84(3 Pt 1):1005-13, 1999.

3. **Killgore WD**. Affective valence and arousal in self-rated depression and anxiety. Percept Mot Skills. 89(1):301-4, 1999.

4. **Killgore WD**, Adams RL. Prediction of Boston Naming Test performance from vocabulary scores: preliminary guidelines for interpretation. Percept Mot Skills. 89(1):327-37, 1999.

5. **Killgore WD**, Gangestad SW. Sex differences in asymmetrically perceiving the intensity of facial expressions. Percept Mot Skills. 89(1):311-4, 1999.

6. **Killgore WD**. The visual analogue mood scale: can a single-item scale accurately classify depressive mood state? Psychol Rep. 85(3 Pt 2):1238-43, 1999.

7. **Killgore WD**, DellaPietra L, Casasanto DJ. Hemispheric laterality and self-rated personality traits. Percept Mot Skills. 89(3 Pt 1):994-6, 1999.

8. **Killgore WD**, Glosser G, Casasanto DJ, French JA, Alsop DC, Detre JA. Functional MRI and the Wada test provide complementary information for predicting post-operative seizure control. Seizure. 8(8):450-5, 1999.

9. **Killgore WD**. Evidence for a third factor on the Positive and Negative Affect Schedule in a

college student sample. Percept Mot Skills. 90(1):147-52, 2000.

10. **Killgore WD**, Dellapietra L. Item response biases on the logical memory delayed recognition subtest of the Wechsler Memory Scale-III. Psychol Rep. 86(3 Pt 1):851-7, 2000.

11. **Killgore WD**, Casasanto DJ, Yurgelun-Todd DA, Maldjian JA, Detre JA. Functional activation of the left amygdala and hippocampus during associative encoding. Neuroreport. 11(10):2259-63, 2000.

12. Yurgelun-Todd DA, Gruber SA, Kanayama G, **Killgore WD**, Baird AA, Young AD. fMRI during affect discrimination in bipolar affective disorder. Bipolar Disord. 2(3 Pt 2):237-48, 2000.

13. **Killgore WD**. Sex differences in identifying the facial affect of normal and mirror-reversed faces. Percept Mot Skills. 91(2):525-30, 2000.

14. **Killgore WD**, DellaPietra L. Using the WMS-III to detect malingering: empirical validation of the rarely missed index (RMI). J Clin Exp Neuropsychol. 22(6):761-71, 2000.

15. Maldjian JA, Detre JA, **Killgore WD**, Judy K, Alsop D, Grossman M, Glosser G. Neuropsychologic performance after resection of an activation cluster involved in cognitive memory function. AJR Am J Roentgenol. 176(2):541-4, 2001.

16. **Killgore WD**, Oki M, Yurgelun-Todd DA. Sex-specific developmental changes in amygdala responses to affective faces. Neuroreport. 12(2):427-33, 2001.

17. **Killgore WD**, Yurgelun-Todd DA. Sex differences in amygdala activation during the perception of facial affect. Neuroreport. 12(11):2543-7, 2001.

18. Casasanto DJ, **Killgore WD**, Maldjian JA, Glosser G, Alsop DC, Cooke AM, Grossman M, Detre JA. Neural correlates of successful and unsuccessful verbal memory encoding. Brain Lang. 80(3):287-95, 2002.

19. **Killgore WD**. Laterality of lesions and trait-anxiety on working memory performance. Percept Mot Skills. 94(2):551-8, 2002.

20. **Killgore WD**, Cupp DW. Mood and sex of participant in perception of happy faces. Percept Mot Skills. 95(1):279-88, 2002.

21. Yurgelun-Todd DA, **Killgore WD**, Young AD. Sex differences in cerebral tissue volume and cognitive performance during adolescence. Psychol Rep. 91(3 Pt 1):743-57, 2002.

22. Yurgelun-Todd DA, **Killgore WD**, Cintron CB. Cognitive correlates of medial temporal lobe development across adolescence: a magnetic resonance imaging study. Percept Mot Skills. 96(1):3-17, 2003.

23. **Killgore WD**, Young AD, Femia LA, Bogorodzki P, Rogowska J, Yurgelun-Todd DA. Cortical and limbic activation during viewing of high- versus low-calorie foods. Neuroimage.

19(4):1381-94, 2003.

24. **Killgore WD**, Yurgelun-Todd DA. Activation of the amygdala and anterior cingulate during nonconscious processing of sad versus happy faces. Neuroimage. 21(4):1215-23, 2004.

25. **Killgore WD**, Yurgelun-Todd DA. Sex-related developmental differences in the lateralized activation of the prefrontal cortex and amygdala during perception of facial affect. Percept Mot Skills. 99(2):371-91, 2004.

26. **Killgore WD**, Glahn DC, Casasanto DJ. Development and Validation of the Design Organization Test (DOT): a rapid screening instrument for assessing visuospatial ability. J Clin Exp Neuropsychol. 27(4):449-59, 2005.

27. **Killgore WD**, Yurgelun-Todd DA. Body mass predicts orbitofrontal activity during visual presentations of high-calorie foods. Neuroreport. 16(8):859-63, 2005.

28. Wesensten NJ, **Killgore WD**, Balkin TJ. Performance and alertness effects of caffeine, dextroamphetamine, and modafinil during sleep deprivation. J Sleep Res. 14(3):255-66, 2005.

29. **Killgore WD**, Yurgelun-Todd DA. Social anxiety predicts amygdala activation in adolescents viewing fearful faces. Neuroreport. 16(15):1671-5, 2005.

30. **Killgore WD**, Yurgelun-Todd DA. Developmental changes in the functional brain responses of adolescents to images of high and low-calorie foods. Dev Psychobiol. 47(4):377-97, 2005.

31. Kahn-Greene ET, Lipizzi EL, Conrad AK, Kamimori GH, **Killgore WD**. Sleep deprivation adversely affects interpersonal responses to frustration. Pers Individ Dif. 41(8):1433-1443, 2006.

32. McBride SA, Balkin TJ, Kamimori GH, **Killgore WD**. Olfactory decrements as a function of two nights of sleep deprivation. J Sens Stud. 24(4):456-63, 2006.

33. **Killgore WD**, Yurgelun-Todd DA. Ventromedial prefrontal activity correlates with depressed mood in adolescent children. Neuroreport. 17(2):167-71, 2006.

34. **Killgore WD**, Vo AH, Castro CA, Hoge CW. Assessing risk propensity in American soldiers: preliminary reliability and validity of the Evaluation of Risks (EVAR) scale--English version. Mil Med. 171(3):233-9, 2006.

35. **Killgore WD**, Balkin TJ, Wesensten NJ. Impaired decision making following 49 h of sleep deprivation. J Sleep Res. 15(1):7-13, 2006.

36. **Killgore WD**, Stetz MC, Castro CA, Hoge CW. The effects of prior combat experience on the expression of somatic and affective symptoms in deploying soldiers. J Psychosom Res. 60(4):379-85, 2006.

37. **Killgore WD**, McBride SA, Killgore DB, Balkin TJ. The effects of caffeine, dextroamphetamine, and modafinil on humor appreciation during sleep deprivation. Sleep.

29(6):841-7, 2006.

38. **Killgore WD**, McBride SA. Odor identification accuracy declines following 24 h of sleep deprivation. J Sleep Res. 15(2):111-6, 2006.

39. **Killgore WD**, Yurgelun-Todd DA. Affect modulates appetite-related brain activity to images of food. Int J Eat Disord. 39(5):357-63, 2006.

40. Kendall AP, Kautz MA, Russo MB, **Killgore WD**. Effects of sleep deprivation on lateral visual attention. Int J Neurosci. 116(10):1125-38, 2006.

41. Yurgelun-Todd DA, **Killgore WD**. Fear-related activity in the prefrontal cortex increases with age during adolescence: a preliminary fMRI study. Neurosci Lett. 406(3):194-9, 2006.

42. **Killgore WD**, Killgore DB, Ganesan G, Krugler AL, Kamimori GH. Trait-anger enhances effects of caffeine on psychomotor vigilance performance. Percept Mot Skills. 103(3):883-6, 2006.

43. **Killgore WD**, Yurgelun-Todd DA. Unconscious processing of facial affect in children and adolescents. Soc Neurosci. 2(1):28-47, 2007.

44. **Killgore WD**, Yurgelun-Todd DA. The right-hemisphere and valence hypotheses: could they both be right (and sometimes left)?. Soc Cogn Affect Neurosci. 2(3):240-50, 2007.

45. **Killgore WD**, Killgore DB. Morningness-eveningness correlates with verbal ability in women but not men. Percept Mot Skills. 104(1):335-8, 2007.

46. **Killgore WD**, Killgore DB, Day LM, Li C, Kamimori GH, Balkin TJ. The effects of 53 hours of sleep deprivation on moral judgment. Sleep. 30(3):345-52, 2007.

47. Rosso IM, **Killgore WD**, Cintron CM, Gruber SA, Tohen M, Yurgelun-Todd DA. Reduced amygdala volumes in first-episode bipolar disorder and correlation with cerebral white matter. Biol Psychiatry. 61(6):743-9, 2007.

48. Kahn-Greene ET, Killgore DB, Kamimori GH, Balkin TJ, **Killgore WD**. The effects of sleep deprivation on symptoms of psychopathology in healthy adults. Sleep Med. 8(3):215-21, 2007.

49. **Killgore WD**. Effects of sleep deprivation and morningness-eveningness traits on risk-taking. Psychol Rep. 100(2):613-26, 2007.

50. **Killgore WD**, Gruber SA, Yurgelun-Todd DA. Depressed mood and lateralized prefrontal activity during a Stroop task in adolescent children. Neurosci Lett. 416(1):43-8, 2007.

51. **Killgore WD**, Yurgelun-Todd DA. Positive affect modulates activity in the visual cortex to images of high calorie foods. Int J Neurosci. 117(5):643-53, 2007.

52. Vo AH, Satori R, Jabbari B, Green J, **Killgore WD**, Labutta R, Campbell WW. Botulinum

toxin type-a in the prevention of migraine: a double-blind controlled trial. Aviat Space Environ Med. 78(5 Suppl):B113-8, 2007.

53. **Killgore WD**, Yurgelun-Todd DA. Neural correlates of emotional intelligence in adolescent children. Cogn Affect Behav Neurosci. 7(2):140-51, 2007.

54. **Killgore WD**, Kendall AP, Richards JM, McBride SA. Lack of degradation in visuospatial perception of line orientation after one night of sleep loss. Percept Mot Skills. 105(1):276-86, 2007.

55. **Killgore WD**, Lipizzi EL, Kamimori GH, Balkin TJ. Caffeine effects on risky decision making after 75 hours of sleep deprivation. Aviat Space Environ Med. 78(10):957-62, 2007.

56. **Killgore WD**, Richards JM, Killgore DB, Kamimori GH, Balkin TJ. The trait of Introversion-Extraversion predicts vulnerability to sleep deprivation. J Sleep Res. 16(4):354-63, 2007.

57. **Killgore WD**, Kahn-Green ET, Killgore DB, Kamimori GH, Balkin TJ. Effects of acute caffeine withdrawal on Short Category Test performance in sleep-deprived individuals. Percept Mot Skills. 105(3 pt.2):1265-74, 2007.

58. **Killgore WD**, Killgore DB, McBride SA, Kamimori GH, Balkin TJ. Odor identification ability predicts changes in symptoms of psychopathology following 56 hours of sleep deprivation. J Sensory Stud. 23(1):35-51, 2008.

59. **Killgore WD**, Rupp TL, Grugle NL, Reichardt RM, Lipizzi EL, Balkin TJ. Effects of dextroamphetamine, caffeine and modafinil on psychomotor vigilance test performance after 44 h of continuous wakefulness. J Sleep Res. 17(3):309-21, 2008.

60. Huck NO, McBride SA, Kendall AP, Grugle NL, **Killgore WD**. The effects of modafinil, caffeine, and dextroamphetamine on judgments of simple versus complex emotional expressions following sleep deprivation. Int. J Neuroscience. 118(4):487-502, 2008.

61. **Killgore WD**, Kahn-Greene ET, Lipizzi EL, Newman RA, Kamimori GH, Balkin TJ. Sleep deprivation reduces perceived emotional intelligence and constructive thinking skills. Sleep Med. 9(5):517-26, 2008

62. **Killgore WD**, Grugle NL, Killgore DB, Leavitt BP, Watlington GI, McNair S, Balkin TJ. Restoration of risk-propensity during sleep deprivation: caffeine, dextroamphetamine, and modafinil. Aviat Space Environ Med. 79(9):867-74, 2008.

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39. **Killgore, WDS,** Arora, NS, Braun, AR, Belenky, G, Wesensten, NJ, & Balkin, TJ An H215O PET study of regional cerebral activation during stage 2 sleep [abstract]. Poster presented at the 17th Congress of the European Sleep Research Society, Prague, Czech Republic, October 5-9, 2004.

40. Wesensten, N, **Killgore, WDS,** Belenky, G, Reichardt, R, Thorne, D, & Balkin, T. Caffeine, dextroamphetamine, and modafinil during 85 H of sleep deprivation. II. Effects of tasks of executive function [abstract]. Poster presented at the 17th Congress of the European Sleep Research Society, Prague, Czech Republic, October 5-9, 2004.

41. Balkin, T, Reichardt, R, Thorne, D, **Killgore, WDS,** Belenky, G, & Wesensten, N. Caffeine, dextroamphetamine, and modafinil during 85 hours of sleep deprivation. I. Psychomotor vigilance and objective alertness effects [abstract]. Oral paper presentation at the 17th Congress of the European Sleep Research Society, Prague, Czech Republic, October 5-9, 2004.

42. Belenky, G, Reichardt, R, Thorne, D, **Killgore, WDS,** Balkin, T, & Wesensten, N. Caffeine, dextroamphetamine, and modafinil during 85 hours of sleep deprivation. III. Effect on recovery sleep and post-recovery sleep performance [abstract]. Oral paper presentation at the 17th Congress of the European Sleep Research Society, Prague, Czech Republic, October 5-9, 2004.

43. Vo, A, Green, J, Campbell, W, **Killgore, WDS,** Labutta, R, & Redmond, D. The quantification of disrupted sleep in migraine via actigraphy: A pilot study [abstract]. Abstract presented at the Associated Professional Sleep Societies 19th Annual Meeting, Denver, CO, June 18-23, 2005. SLEEP, 28 (Supplement), A281.

44. Kendall, AP, **Killgore, WDS,** Kautz, M, & Russo, MB. Left-visual field deficits in attentional processing after 40 hours of sleep deprivation [abstract]. Abstract presented at the Associated Professional Sleep Societies 19th Annual Meeting, Denver, CO, June 18-23, 2005. SLEEP, 28 (Supplement), A143.

45. Reichardt, RM, Grugle, NL, Balkin, TJ, & **Killgore, WDS.** Stimulant countermeasures, risk propensity, and IQ across 2 nights of sleep deprivation [abstract]. Abstract presented at the Associated Professional Sleep Societies 19th Annual Meeting, Denver, CO, June 18-23, 2005. SLEEP, 28 (Supplement), A145.

46. Killgore, DB, McBride, SA, Balkin, TJ, & **Killgore, WDS.** Post-stimulant hangover: The effects of caffeine, modafinil, and dextroamphetamine on sustained verbal fluency following sleep deprivation and recovery sleep [abstract]. Abstract presented at the Associated Professional Sleep Societies 19th Annual Meeting, Denver, CO, June 18-23, 2005. SLEEP, 28 (Supplement), A137.

47. **Killgore, WDS,** Balkin, TJ, & Wesensten, NJ. Impaired decision-making following 49 hours of sleep deprivation [abstract]. Abstract presented at the Associated Professional Sleep Societies 19th Annual Meeting, Denver, CO, June 18-23, 2005. SLEEP, 28 (Supplement), A138.

48. **Killgore, WDS,** McBride, SA, Killgore, DB, & Balkin, TJ. Stimulant countermeasures and risk propensity across 2 nights of sleep deprivation [abstract]. Abstract presented at the Associated Professional Sleep Societies 19th Annual Meeting, Denver, CO, June 18-23, 2005. SLEEP, 28 (Supplement), A136.

49. McBride, SA, Balkin, TJ, & **Killgore, WDS.** The effects of 24 hours of sleep deprivation on odor identification accuracy [abstract]. Abstract presented at the Associated Professional Sleep Societies 19th Annual Meeting, Denver, CO, June 18-23, 2005. SLEEP, 28 (Supplement), A137.

50. Picchioni, D, **Killgore, WDS,** Braun, AR, & Balkin, TJ. PET correlates of EEG activity during non-REM sleep. Poster presentation at the annual UCLA/Websciences Sleep Training Workshop, Lake Arrowhead, CA, September, 2005.

51. **Killgore, WDS,** Killgore, DB, McBride, SA, & Balkin, TJ. Sustained verbal fluency following sleep deprivation and recovery sleep: The effects of caffeine, modafinil, and dextroamphetamine. Poster presented at the 34th Meeting of the International Neuropsychological Society, Boston, MA, February 1-4, 2006.

52. **Killgore, WDS,** Balkin, TJ, & Wesensten, NJ. Decision-making is impaired following 2-days of sleep deprivation. Poster presented at the 34th Meeting of the International Neuropsychological Society, Boston, MA, February 1-4, 2006.

53. **Killgore, WDS,** & Yurgelun-Todd, DA. Neural correlates of emotional intelligence in adolescent children. Poster presented at the 34th Meeting of the International Neuropsychological Society, Boston, MA, February 1-4, 2006.

54. **Killgore, WDS,** & Yurgelun-Todd, DA. Social anxiety predicts amygdala activation in adolescents viewing fearful faces. Poster presented at the 34th Meeting of the International Neuropsychological Society, Boston, MA, February 1-4, 2006.

55. McBride, SA & **Killgore, WDS.** Sleepy people smell worse: Olfactory deficits following extended wakefulness. Paper presented at the Workshop on Trace Gas Detection Using Artificial, Biological, and Computational Olfaction. Monell Chemical Senses Center, Philadelphia, PA, March 29-31, 2006.

56. **Killgore, WDS,** Day LM, Li, C, Kamimori, GH, Balkin, TJ, & Killgore DB. Moral reasoning is affected by sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A137.

57. **Killgore, WDS,** Killgore DB, Kahn-Green, E, Conrad, A, Balkin, TJ, & Kamimori, G. H. Introversion-Extroversion predicts resilience to sleep loss [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A137.

58. Newman, R, Kamimori, GH, **Killgore, WDS.** Sleep deprivation diminishes constructive thinking [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A136-137.

59. Huck, NO, Kendall, AP, McBride, SA, **Killgore, WDS.** The perception of facial emotion is enhanced by psychostimulants following two nights of sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City,

UT, June 17-22, 2006. SLEEP, 29 (Supplement), A136.

60. O'Sullivan, M, Reichardt, RM, Krugler, AL, Killgore, DB, & **Killgore, WDS.** Premorbid intelligence correlates with duration and quality of recovery sleep following sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A372.

61. McBride, SA, **Killgore, WDS,** Kahn-Green, E, Conrad, A, & Kamimori, GH. Caffeine administered to maintain overnight alertness does not disrupt performance during the daytime withdrawal period [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A136.

62. McBride, SA, Killgore DB, Balkin, TJ, Kamimori, GH, & **Killgore, WDS.** Sleepy people smell worse: Olfactory decrements as a function of sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A135.

63. Day, LM, Li, C, Killgore, DB, Kamimori, GH, & **Killgore, WDS.** Emotional intelligence moderates the effect of sleep deprivation on moral reasoning [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A135.

64. Murray, CJ, Killgore, DB, Kamimori, GH, & **Killgore, WDS.** Individual differences in stress management capacity predict responsiveness to caffeine during sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A43.

65. Murray, CJ, Newman, R, O'Sullivan, M, Killgore, DB, Balkin, TJ, & **Killgore, WDS.** Caffeine, dextroamphetamine, and modafinil fail to restore Stroop performance during sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A370-371.

66. Richards, J, Killgore, DB, & **Killgore, WDS.** The effect of 44 hours of sleep deprivation on mood using the Visual Analog Mood Scales [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A132.

67. Richards, J, & **Killgore, WDS.** The effect of caffeine, dextroamphetamine, and modafinil on alertness and mood during sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A43.

68. Lipizzi, EL, Leavitt, BP, Killgore, DB, Kamimori, GH, & **Killgore, WDS.** Decision making capabilities decline with increasing duration of wakefulness [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A131.

69. Lipizzi, EL, Killgore, DB, Kahn-Green, E, Kamimori, GH, & **Killgore, WDS.** Emotional intelligence scores decline during sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A131.

70. Kahn-Green, E, Day, L, Conrad, A, Leavitt, BP, Killgore, DB, & **Killgore, WDS.** Short-term vs. long-term planning abilities: Differential effects of stimulants on executive function in sleep deprived individuals [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A370.

71. Kahn-Green, E, Conrad, A, Killgore, DB, Kamimori, GH, & **Killgore, WDS.** Tired and frustrated: Using a projective technique for assessing responses to stress during sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A130.

72. Killgore, DB, Kahn-Green, E, Balkin, TJ, Kamimori, GH, & **Killgore, WDS.** 56 hours of wakefulness is associated with a sub-clinical increase in symptoms of psychopathology [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A130.

73. Killgore, DB, McBride, SA, Balkin, TJ, Leavitt, BP, & **Killgore, WDS.** Modafinil improves humor appreciation during sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A42.

74. Reichardt, RM, Killgore, DB, Lipizzi, EL, Li, CJ, Krugler, AL, & **Killgore, WDS.** The effects of stimulants on recovery sleep and post-recovery verbal performance following 61-hours of sleep deprivation [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A42.

75. Bailey, JD, Richards, J, & **Killgore, WDS.** Prediction of mood fluctuations during sleep deprivation with the SAFTE Model [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A60.

76. Kendall, AP, McBride, S. A, & **Killgore, WDS.** Visuospatial perception of line orientation is resistant to one night of sleep loss [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A369.

77. Kendall, AP, McBride, SA, Kamimori, GH, & **Killgore, WDS.** The interaction of coping skills and stimulants on sustaining vigilance: Poor coping may keep yoiu up at night [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A129.

78. Muckle, A, Killgore, DB, & **Killgore, WDS.** Gender differences in the effects of stimulant

medications on the ability to estimate unknown quantities when sleep deprived [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A369.

79. Krugler, AL, **Killgore, WDS,** & Kamimori, G. H. Trait anger predicts resistance to sleep loss [abstract]. Abstract presented at the 20th Meeting of the Associated Professional Sleep Societies, Salt Lake City, UT, June 17-22, 2006. SLEEP, 29 (Supplement), A129.

80. **Killgore, WDS,** Cotting, DI, Vo, A. H, Castro, CA, & Hoge, CW. The invincibility syndrome: Combat experiences predict risk-taking propensity following redeployment [abstract]. Abstract presented at the 9th Annual Force Health Protection Conference, Albuquerque, NM, August 6-11, 2006.

81. **Killgore, WDS,** Wesensten, NJ, & Balkin, TJ. Stimulants improve tactical but not strategic planning during prolonged wakefulness [abstract]. Abstract presented at the 9th Annual Force Health Protection Conference, Albuquerque, NM, August 6-11, 2006.

82. **Killgore, WDS,** Balkin, TJ, Wesensten, NJ, & Kamimori, G. H. The effects of sleep loss and caffeine on decision-making [abstract]. Abstract presented at the 9th Annual Force Health Protection Conference, Albuquerque, NM, August 6-11, 2006.

83. **Killgore, WDS,** Balkin, TJ, & Kamimori, GH. Sleep loss can impair moral judgment [abstract]. Abstract presented at the 9th Annual Force Health Protection Conference, Albuquerque, NM, August 6-11, 2006.

84. **Killgore, WDS,** Lipizzi, EL, Reichardt, RM, Kamimori, GH, & Balkin, TJ. Can stimulants reverse the effects of sleep deprivation on risky decision-making [abstract]? Abstract presented at the 25th Army Science Conference, Orlando, FL, November 27-30, 2006.

85. **Killgore, WDS,** Killgore, DB, Kamimori, GH, & Balkin, TJ. Sleep deprivation impairs the emotional intelligence and moral judgment capacities of Soldiers [abstract]. Abstract presented at the 25th Army Science Conference, Orlando, FL, November 27-30, 2006.

86. **Killgore, WDS,** Cotting, DI, Vo, AH, Castro, C.A, & Hoge, CW. The post-combat invincibility syndrome: Combat experiences increase risk-taking propensity following deployment [abstract]. Abstract presented at the 25th Army Science Conference, Orlando, FL, November 27-30, 2006.

87. Adam, GE, Szelenyi, ER, **Killgore, WD,** & Lieberman, HR. A double-blind study of two days of caloric deprivation: Effects on judgment and decision-making. Oral paper presentation at the Annual Scientific Meeting of the Aerospace Medical Association, New Orleans, LA, May, 2007.

88. Killgore, DB, Kahn-Greene, ET, Kamimori, GH, & **Killgore, WD.** The effects of acute caffeine withdrawal on short category test performance in sleep deprived individuals [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A43.

89. Richards, JM, Lipizzi, EL, Kamimori, GH, & **Killgore, WD.** Extroversion predicts change in attentional lapses during sleep deprivation [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A137.

90. Lipizzi, EL, Richards, JM, Balkin, TJ, Grugle, NL, & **Killgore, WD.** Morningness-Eveningness and Intelligence [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A345.

91. Lipizzi, EL, Richards, Balkin, TJ, Grugle, NL, & **Killgore WD.** Morningness-Eveningness affects risk-taking propensity during sleep deprivation [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A136.

92. McBride, SA, Ganesan, G, Kamimori, GH, & **Killgore, WD.** Odor identification ability predicts vulnerability to attentional lapses during 77 hours of sleep deprivation [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A135.

93. Smith, KL, McBride, S. A, Kamimori, GH, & **Killgore, WD.** Individual differences in odor discrimination predict mood dysregulation following 56 hours of sleep deprivation [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A136.

94. McBride, SA, Leavitt, BP, Kamimori, GH, & **Killgore, WD.** Odor identification accuracy predicts resistance to sleep loss. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A137.

95. Killgore, DB, McBride, SA, Balkin, TJ, Grugle, NL. & **Killgore, WD.** Changes in odor discrimination predict executive function deficits following 45 hours of wakefulness [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A136.

96. Rupp, TL, Killgore, DB, Balkin, TJ, Grugle, NL, & **Killgore, WD.** The effects of modafinil, dextroamphetamine, and caffeine on verbal and nonverbal fluency in sleep deprived individuals [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A43.

97. Newman, RA, Krugler, AL, Kamimori, GH, & **Killgore, WD.** Changes in state and trait anger following 56 hours of sleep deprivation [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A138.

98. Rupp, TL, Grugle, NL, Krugler, AL, Balkin, TJ, & **Killgore, WD.** Caffeine, dextroamphetamine, and modafinil improve PVT performance after sleep deprivation and recovery sleep [abstract]. Abstract presented at the 21st Meeting of the Associated

Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A44.

99. **Killgore, WD,** Lipizzi, EL, Balkin, TJ, Grugle, NL, & Killgore, DB. The effects of sleep deprivation and stimulants on self-reported sensation seeking propensity [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A42.

100. **Killgore, WD,** Richards, JM, Balkin, TJ, Grugle, NL, & Killgore DB. The effects of sleep deprivation and stimulants on risky behavior [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A41.

101. Newman, RA, Smith, KL, Balkin, TJ, Grugle, NL, & **Killgore, WD.** The effects of caffeine, dextroamphetamine, and modafinil on executive functioning following 45 hours of sleep deprivation [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A45.

102. Richards, JM, Lipizzi, EL, Balkin, TJ, Grugle, NL, & **Killgore, WD.** Objective alertness predicts mood changes during 44 hours of sleep deprivation [abstract]. Abstract presented at the 21st Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 9-14, 2007. SLEEP, 30 (Supplement), A56.

103. **Killgore, WD,** & Yurgelun-Todd, DA. Cortical and Limbic Activation in Response to Visual Images of Low and High-Caloric Food [abstract]. Oral symposium presented at the 6th Annual Conference of the Society of Behavioral Nutrition and Physical Activity (ISBNPA), Oslo, Norway, June 20-23, 2007. Proceedings of the ISBNPA, 2007, 75.

104. Estrada, A, **Killgore, WD,** Rouse, T, Balkin, TJ, & Wildzunas, RM. Total sleep time measured by actigraphy predicts academic performance during military training [abstract]. Abstract presented at the 22nd Meeting of the Associated Professional Sleep Societies, Baltimore, MD, June 7-12, 2008. SLEEP, 31 (Supplement), A134.

105. **Killgore, WD,** Lipizzi, EL, Smith, KL, Killgore, DB, Rupp, TL, Kamimori, GH, & Balkin, T. J. Nonverbal intelligence is inversely related to the ability to resist sleep loss [abstract]. Abstract presented at the 22nd Meeting of the Associated Professional Sleep Societies, Baltimore, MD, June 7-12, 2008. SLEEP, 31 (Supplement), A134.

106. **Killgore, WD,** Lipizzi, EL, Killgore, DB, Rupp, TL, Kamimori, GH, & Balkin, TJ. Emotional intelligence predicts declines in emotion-based decision-making following sleep deprivation [abstract]. Abstract presented at the 22nd Meeting of the Associated Professional Sleep Societies, Baltimore, MD, June 7-12, 2008. SLEEP, 31 (Supplement), A134.

107. Reid, CT, Smith, K, **Killgore, WD,** Rupp, TL, & Balkin, TJ. Higher intelligence is associated with less subjective sleepiness during sleep restriction [abstract]. Abstract presented at the 22nd Meeting of the Associated Professional Sleep Societies, Baltimore, MD, June 7-12, 2008. SLEEP, 31 (Supplement), A375.

108. Newman, R, **Killgore, WD,** Rupp, T. L, & Balkin, TJ. Better baseline olfactory discrimination is associated with worse PVT and MWT performance with sleep restriction and recovery [abstract]. Abstract presented at the 22nd Meeting of the Associated Professional Sleep Societies, Baltimore, MD, June 7-12, 2008. SLEEP, 31 (Supplement), A375.

109. Smith, KL, Reid, CT, **Killgore, WD,** Rupp, TL, & Balkin, TJ. Personality factors associated with performance and sleepiness during sleep restriction and recovery [abstract]. Abstract presented at the 22nd Meeting of the Associated Professional Sleep Societies, Baltimore, MD, June 7-12, 2008. SLEEP, 31 (Supplement), A376.

110. Lipizzi, EL, **Killgore, WD,** Rupp, TL, & Balkin, TJ. Risk-taking behavior is elevated during recovery from sleep restriction [abstract]. Abstract presented at the 22nd Meeting of the Associated Professional Sleep Societies, Baltimore, MD, June 7-12, 2008. SLEEP, 31 (Supplement), A376.

111. Lipizzi, EL, Rupp, TL, **Killgore, WD,** & Balkin, TJ. Sleep restriction increases risk-taking behavior [abstract]. Poster presented at the 11th Annual Force Health Protection Conference, Albuquerque, NM, August, 9-15, 2008.

112. **Killgore, WD,** Estrada, A, Balkin, TJ, & Wildzunas, RM. Sleep duration during army training predicts course performance [abstract]. Poster presented at the 6th Annual Force Health Protection Conference, Albuquerque, NM, August, 11-17, 2008.

113. **Killgore, WD,** Lipizzi, EL, Smith, KL, Killgore, DB, Rupp, TL, Kamimori, GH, & Balkin, TJ. Higher cognitive ability is associated with reduced relative resistance to sleep loss [abstract]. Poster presented at the 6th Annual Force Health Protection Conference, Albuquerque, NM, August, 11-17, 2008.

114. **Killgore, WD,** Rupp, TL, Grugle, NL, Lipizzi, EL, & Balkin, TJ. Maintaining alertness during sustained operations: Which stimulant is most effective after 44 hours without sleep [abstract]? Poster presented at the 6th Annual Force Health Protection Conference, Albuquerque, NM, August, 11-17, 2008.

115. **Killgore, WD,** Newman, RA, Lipizzi, EL, Kamimori, GH, & Balkin, TJ. Sleep deprivation increases feelings of anger but reduces verbal and physical aggression in Soldiers [abstract]. Poster presented at the 6th Annual Force Health Protection Conference, Albuquerque, NM, August, 11-17, 2008.

116. Kelley, AM, Dretsch, M, **Killgore, WD,** & Athy, JR. Risky behaviors and attitudes about risk in Soldiers. Abstract presented at the 29th Annual Meeting of the Society for Judgment and Decision Making, Chicago, IL, November, 2008.

117. **Killgore, WD,** Ross, AJ, Silveri, MM, Gruber, SA, Kamiya, T, Kawada, Y, Renshaw, PF, & Yurgelun-Todd, DA. Citicoline affects appetite and cortico-limbic responses to images of high calorie foods. Abstract presented at the Society for Neuroscience, Washington DC, November 19, 2008.

118. Britton, JC, Stewart, SE, Price, LM, **Killgore, WD,** Gold, AL, Jenike, MA, & Rauch, SL. Reduced amygdalar activation in response to emotional faces in pediatric Obsessive-Compulsive Disorder. Abstract presented at the Annual meeting of the American College of Neuropsychopharmacology, Scottsdale, AZ, December 7-11, 2008.

119. **Killgore, WD,** Balkin, TJ, Estrada, A, & Wildzunas, RM. Sleep and performance measures in soldiers undergoing military relevant training. Abstract presented at the 26th Army Science Conference, Orlando, FL, December 1-4, 2008.

120. **Killgore, WD** & Yurgelun-Todd, DA. Cerebral correlates of amygdala responses during non-conscious perception of affective faces in adolescent children. Abstract presented at the 37th Annual Meeting of the International Neuropsychological Society, Atlanta, GA, February 11-14, 2009.

121. **Killgore, WD,** Killgore, DB, Grugle, NL, & Balkin, TJ. Odor identification ability predicts executive function deficits following sleep deprivation. Abstract presented the 37th Annual Meeting of the International Neuropsychological Society, Atlanta, GA, February 11-14, 2009.

122. **Killgore, WD,** Rupp, TL, Killgore, DB, Grugle, NL, and Balkin, TJ. Differential effects of stimulant medications on verbal and nonverbal fluency during sleep deprivation. Abstract presented the 37th Annual Meeting of the International Neuropsychological Society, Atlanta, GA, February 11-14, 2009.

123. **Killgore, WD,** Killgore, DB, Kamimori, GH, & Balkin, TJ. When being smart is a liability: More intelligent individuals may be less resistant to sleep deprivation. Abstract presented the 37th Annual Meeting of the International Neuropsychological Society, Atlanta, GA, February 11-14, 2009.

124. **Killgore, WD,** Britton, JC, Price, LM, Gold, AL, Deckersbach, T, & Rauch, SL. Introversion is associated with greater amygdala and insula activation during viewing of masked affective stimuli. Abstract presented the 37th Annual Meeting of the International Neuropsychological Society, Atlanta, GA, February 11-14, 2009.

125. **Killgore, WD,** Britton, JC, Price, LM, Gold, AL, Deckersbach, T, & Rauch, SL. Amygdala responses of specific animal phobics do not differ from healthy controls during masked fearful face perception. Abstract presented the 37th Annual Meeting of the International Neuropsychological Society, Atlanta, GA, February 11-14, 2009.

126. **Killgore, WD,** Britton, JC, Price, LM, Gold, AL, Deckersbach, T, & Rauch, SL. Small animal phobics show sustained amygdala activation in response to masked happy facial expressions. Abstract presented the 37th Annual Meeting of the International Neuropsychological Society, Atlanta, GA, February 11-14, 2009. ***[*Merit Poster Award]***

127. Price, LM, **Killgore, WD,** Britton, JC, Kaufman, ML, Gold, AL, Deckersbach, T, & Rauch, SL. Anxiety sensitivity correlates with insula activation in response to masked fearful faces in specific animal phobics and healthy subjects. Abstract presented at the Annual Conference of the Anxiety Disorders Association of America, Santa Ana Pueblo, New Mexico, March 12-15, 2009.

128. **Killgore, WD,** Britton, JC, Price, LM, Gold, AL, Deckersbach, T, & Rauch, SL. Neuroticism is inversely correlated with amygdala and insula activation during masked presentations of affective stimuli. Abstract presented at the Annual Conference of the Anxiety Disorders Association of America, Santa Ana Pueblo, New Mexico, March 12-15, 2009.

129. **Killgore, WD,** Kelley, AM, & Balkin, TJ. Development and validation of a scale to measure the perception of invincibility. Abstract presented at the Annual Conference of the Anxiety Disorders Association of America, Santa Ana Pueblo, New Mexico, March 12-15, 2009.

130. Kelly, AM, **Killgore WD,** Athy, J, & Dretsch, M. Risk propensity, risk perception, risk aversion, and sensation seeking in U.S. Army soldiers. Abstract presented at the 80th Annual Scientific Meeting of the Aerospace Medical Association, Los Angeles, CA, May 3-7, 2009.

131. Britton, JC, Stewart, SE, Price, LM, **Killgore, WD,** Jenike, MA, & Rauch, SL. The neural correlates of negative priming in pediatric obsessive-compulsive disorder (OCD). Abstract presented at the 64th Annual Scientific Meeting of the Society of Biological Psychiatry, Vancouver, Canada, May 14-16, 2009.

132. **Killgore, WD,** Killgore, DB, Kamimori, GH, & Balkin, TJ. Caffeine protects against increased risk-taking behavior during severe sleep deprivation. Abstract presented at the 23rd Annual Meeting of the Associated Professional Sleep Societies, Seattle, Washington, June 7-12, 2009.

133. Killgore, DB, **Killgore, WD,** Grugle, NL, & Balkin, TJ. Executive functions predict the ability to sustain psychomotor vigilance during sleep loss. Abstract presented at the 23rd Annual Meeting of the Associated Professional Sleep Societies, Seattle, Washington, June 7-12, 2009.

134. **Killgore, WD,** & Yurgelun-Todd, DA. Trouble falling asleep is associated with reduced activation of dorsolateral prefrontal cortex during a simple attention task. Abstract presented at the 23rd Annual Meeting of the Associated Professional Sleep Societies, Seattle, Washington, June 7-12, 2009.

135. **Killgore, WD,** Kelley, AM, & Balkin, TJ. A new scale for measuring the perception of invincibility. Abstract presented at the 12th Annual Force Health Protection Conference, Albuquerque, New Mexico, August 14-21, 2009.

136. **Killgore, WD,** Killgore, DB, Grugle, NL, & Balkin, TJ. Executive functions contribute to the ability to resist sleep loss. Abstract presented at the 12th Annual Force Health Protection Conference, Albuquerque, New Mexico, August 14-21, 2009.

137. **Killgore, WD,** Killgore, DB, Kamimori, GH, & Balkin, TJ. Caffeine reduces risk-taking behavior during severe sleep deprivation. Abstract presented at the 12th Annual Force Health Protection Conference, Albuquerque, New Mexico, August 14-21, 2009. ***[*Best Paper: Research]***

138. **Killgore, WD,** Castro, CA, & Hoge, CW. Normative data for the Evaluation of Risks

Scale—Bubble Sheet Version (EVAR-B) for large scale surveys of returning combat veterans. Abstract presented at the 12th Annual Force Health Protection Conference, Albuquerque, New Mexico, August 14-21, 2009.

139. **Killgore, WD,** Castro, CA, & Hoge, CW. Combat exposure and post-deployment risky behavior. Abstract presented at the 12th Annual Force Health Protection Conference, Albuquerque, New Mexico, August 14-21, 2009.

140. **Killgore, WD**, Price, LM, Britton, JC, Simon, N, Pollack, MH, Weiner, MR, Schwab, ZJ, Rosso, IM, & Rauch, SL. Paralimbic responses to masked emotional faces in PTSD: Disorder and valence specificity. Abstract presented at the Annual McLean Hospital Research Day, January 29, 2010.

141. **Killgore, WD**, Killgore, DB, Kamimori, GH, & Balkin, TJ. Caffeine minimizes behavioral risk-taking during 75 hours of sleep deprivation. Abstract presented at the 38th Annual Meeting of the International Neuropsychological Society, Acapulco, Mexico, February 3-6, 2010.

142. **Killgore, WD** & Balkin, TJ. Vulnerability to sleep loss is affected by baseline executive function capacity. Abstract presented at the 38th Annual Meeting of the International Neuropsychological Society, Acapulco, Mexico, February 3-6, 2010.

143. **Killgore, WD**, Smith, KL, Reichardt, RM., Killgore, DB, & Balkin, TJ. Intellectual capacity is related to REM sleep following sleep deprivation. Abstract presented at the 38th Annual Meeting of the International Neuropsychological Society, Acapulco, Mexico, February 3-6, 2010.

144. **Killgore, WD** & Yurgelun-Todd, DA. Cerebral correlates of amygdala responses to masked fear, anger, and happiness in adolescent and pre-adolescent children. Abstract presented at the 38th Annual Meeting of the International Neuropsychological Society, Acapulco, Mexico, February 3-6, 2010.

145. **Killgore, WD**, Post, A, & Yurgelun-Todd, DA. Sex differences in cortico-limbic responses to images of high calorie food. Abstract presented at the 38th Annual Meeting of the International Neuropsychological Society, Acapulco, Mexico, February 3-6, 2010.

146. **Killgore, WD** & Yurgelun-Todd, DA. Self-reported insomnia is associated with increased activation within the default-mode network during a simple attention task. Abstract presented at the 38th Annual Meeting of the International Neuropsychological Society, Acapulco, Mexico, February 3-6, 2010.

147. **Killgore, WD**, Price, LM, Britton, JC, Gold, AL, Deckersbach, T, & Rauch, SL. Neural correlates of anxiety sensitivity factors during presentation of masked fearful faces. Abstract presented at the 38th Annual Meeting of the International Neuropsychological Society, Acapulco, Mexico, February 3-6, 2010.

148. **Killgore, WD**, Grugle, NL, Conrad, TA, & Balkin, TJ. Baseline executive function abilities predict risky behavior following sleep deprivation. Abstract presented at the 24th Annual

Meeting of the Associated Professional Sleep Societies, San Antonio, Texas, June 5-9, 2010.

149. **Killgore, WD**, Grugle, NL, & Balkin, TJ. Judgment of objective vigilance performance is affected by sleep deprivation and stimulants. Abstract presented at the 24th Annual Meeting of the Associated Professional Sleep Societies, San Antonio, Texas, June 5-9, 2010.

150. Killgore, DB, **Killgore, WD**, Grugle, NL, & Balkin, TJ. Resistance to sleep loss and its relationship to decision making during sleep deprivation. Abstract presented at the 24th Annual Meeting of the Associated Professional Sleep Societies, San Antonio, Texas, June 5-9, 2010.

151. Killgore DB, **Killgore, WD**, Grugle, NL, & Balkin, TJ. Subjective sleepiness and objective performance: Differential effects of stimulants during sleep deprivation. Abstract presented at the 24th Annual Meeting of the Associated Professional Sleep Societies, San Antonio, Texas, June 5-9, 2010.

152. Rupp, TL, **Killgore, WD**, & Balkin, TJ. Vulnerability to sleep deprivation is differentially mediated by social exposure in extraverts vs. introverts. Oral presentation at the "Data Blitz" section at the 24th Annual Meeting of the Associated Professional Sleep Societies, San Antonio, Texas, June 5-9, 2010.

153. Rupp, TL, **Killgore, WD**, & Balkin, TJ. Extraverts may be more vulnerable than introverts to sleep deprivation on some measures of risk-taking and executive functioning. Abstract presented at the 24th Annual Meeting of the Associated Professional Sleep Societies, San Antonio, Texas, June 5-9, 2010.

154. Rupp, TL, **Killgore, WD**, & Balkin, TJ. Vulnerability to sleep deprivation is differentially mediated by social exposure in extraverts vs. introverts. Abstract presented at the 24th Annual Meeting of the Associated Professional Sleep Societies, San Antonio, Texas, June 5-9, 2010.

155. Capaldi, VF, Guerrero, ML, & **Killgore, WD**. Sleep disorders among OIF and OEF Soldiers. Abstract presented at the 24th Annual Meeting of the Associated Professional Sleep Societies, San Antonio, Texas, June 5-9, 2010.

156. **Killgore, WD**, Killgore, DB, Kamimori, GH, & Balkin, TJ. Caffeine reduces behavioral risk-taking during sleep deprivation. Abstract presented at the 65th Annual Meeting of the Society for Biological Psychiatry, New Orleans, Louisiana, May 20-22, 2010.

157. **Killgore, WD**, Price, LM, Britton, JC, Simon, N, Pollack, MH, Weiner, MR, Schwab, ZJ, Rosso, IM, & Rauch, SL. Paralimbic responses to masked emotional faces in PTSD: Disorder and valence specificity. Abstract presented at the 65th Annual Meeting of the Society for Biological Psychiatry, New Orleans, Louisiana, May 20-22, 2010.

158. Rosso, IM, Makris, N, Britton, JC, Price, LM, Gold, AL, Deckersbach, T, **Killgore, WD**, & Rauch SL. Anxiety sensitivity correlates with insular cortex volume and thickness in specific animal phobia. Abstract presented at the 65th Annual Meeting of the Society for Biological Psychiatry, New Orleans, Louisiana, May 20-22, 2010.

159. Rupp, TL, **Killgore, WD**, & Balkin, TJ. Vulnerability to sleep deprivation is mediated by social exposure in extraverts versus introverts. Oral platform presentation at the 20th Congress of the European Sleep Research Society, Lisbon, Portugal, September 14-18, 2010.

160. **Killgore, WD**, Estrada, A, & Balkin, TJ. A tool for monitoring soldier fatigue and predicting cognitive readiness: The Sleep History and Readiness Predictor (SHARP). Abstract presented at the 27th Army Science Conference, Orlando, FL, November 29-December 2, 2010.

161. **Killgore, WD**, Kamimori, GH, & Balkin, TJ. Caffeinated gum minimizes risk-taking in soldiers during prolonged sleep deprivation. Abstract presented at the 27th Army Science Conference, Orlando, FL, November 29-December 2, 2010.

162. **Killgore, WD**, Britton, JC, Schwab, ZJ, Weiner, MR, Rosso, IM, & Rauch, SL. Exaggerated amygdala responses to masked fearful faces are specific to PTSD versus simple phobia. Oral platform presentation at the 27th Army Science Conference, Orlando, FL, November 29-December 2, 2010. ***[*Winner Best Paper in Neuroscience]***

163. **Killgore, WD**, Kamimori, GH, & Balkin, TJ. Sleep deprivation selectively impairs emotional aspects of cognitive functioning. Oral platform presentation at the 27th Army Science Conference, Orlando, FL, November 29-December 2, 2010.

164. Rupp, TL, **Killgore, WD**, & Balkin, TJ. Evaluation of personality and social exposure as individual difference factors influencing response to sleep deprivation. Oral platform presentation at the 27th Army Science Conference, Orlando, FL, November 29-December 2, 2010.

165. **Killgore, WD**, Britton, JC, Rosso, IM, Schwab, ZJ, Weiner, MR, & Rauch, SL. Shared and differential patterns of amygdalo-cortical activation across anxiety disorders. Abstract presented at the 49th Annual Meeting of the American College of Neuropsychopharmacology, Miami Beach, FL, December 5-9, 2010.

166. Rosso, IM, **Killgore, WD**, Britton, JC, Weiner, MR, Schwab, ZJ, & Rauch, SL. Neural correlates of PTSD symptom dimensions during emotional processing: A functional magnetic resonance imaging study. Abstract presented at the 49th Annual Meeting of the American College of Neuropsychopharmacology, Miami Beach, FL, December 5-9, 2010.

167. **Killgore, WD,** Rosso, IM, Britton, JC, Schwab, ZJ, Weiner, MR, & Rauch, SL. Cortico-limbic activation differentiates among anxiety disorders with and without a generalized threat response. Abstract presented at the McLean Hospital Research Day, January 13, 2011.

168. Weiner, MR, Schwab, ZJ, Rauch, SL, & **Killgore WD**. Personality factors predict brain responses to images of high-calorie foods. Abstract presented at the McLean Hospital Research Day, January 13, 2011.

169. Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD.** Emotional and cognitive intelligence: Support for the neural efficiency hypothesis. Abstract presented at the McLean Hospital Research Day, January 13, 2011.

170. Crowley, DJ, Covell, MJ, **Killgore, WD**, Schwab, ZJ, Weiner, MR, Acharya, D, Rosso, IM, & Silveri, MM. Differential influence of facial expression on inhibitory capacity in adolescents versus adults. Abstract presented at the McLean Hospital Research Day, January 13, 2011.

171. **Killgore, WD**, Britton, JC, Rosso, IM, Schwab, ZJ, Weiner, MR, & Rauch, SL. Similarities and differences in cortico-limbic responses to masked affect probes across anxiety disorders. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

172. Rosso, IM, **Killgore, WD**, Britton, JC, Weiner, MR, Schwab, ZJ, & Rauch, SL. Hyperarousal and reexperiencing symptoms of post-traumatic stress disorder are differentially associated with limbic-prefrontal brain responses to threatening stimuli. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

173. Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD**. Neural correlates of cognitive and emotional intelligence in adults. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

174. Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD**. Cognitive and emotional intelligences: Are they distinct or related constructs? Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

175. Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD**. Discrepancy scores between cognitive and emotional intelligence predict neural responses to affective stimuli. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

176. **Killgore, WD**, Schwab, ZJ, Weiner, MR, & Rauch, SL. Smart people go with their gut: Emotional intelligence correlates with non-conscious insular responses to facial trustworthiness. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

177. **Killgore, WD**, Weiner, MR, Schwab, ZJ, & Rauch, SL. Whom can you trust? Neural correlates of subliminal perception of facial trustworthiness. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

178. Weiner, MR, Schwab, ZJ, & Rauch, SL, **Killgore, WD**. Impulsiveness predicts responses of brain reward circuitry to high-calorie foods. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

179. Weiner, MR, Schwab, ZJ, & Rauch, SL, **Killgore, WD**. Conscientiousness predicts brain responses to images of high-calorie foods. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

180. Crowley, DJ, Covell, MJ, **Killgore, WD**, Schwab, ZJ, Weiner, MR, Acharya, D, Rosso, IM, & Silveri, MM. Differential influence of facial expression on inhibitory capacity in adolescents versus adults. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

181. Gruber, SA, Dahlgren, MK, **Killgore, WD**, Sagar, KA, & Racine, MT. Marijuana: Age of onset of use impacts executive function and brain activation. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

182. **Killgore, WD,** Conrad, TA, Grugle, NL, & Balkin, TJ. Baseline executive function abilities correlate with risky behavior following sleep deprivation. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

183. **Killgore, WD,** Grugle, NL, Killgore, DB, & Balkin, TJ. Resistance to sleep loss and decision making during sleep deprivation. Abstract presented at the 39th Annual Meeting of the International Neuropsychological Society, Boston, MA, February 2-5, 2011.

184. **Killgore, WD,** Rosso, IM, Britton, JC, Schwab, ZJ, Weiner, MR, & Rauch, SL. Cortico-limbic activation differentiates among anxiety disorders with and without a generalized threat response. Abstract presented at the 66th Annual Meeting of the Society for Biological Psychiatry, San Francisco, CA, May 12-14, 2011. ***[*Blue Ribbon Finalist for Top Poster Award: Clinical/Translational]***

185. Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD.** Emotional and cognitive intelligence: Support for the neural efficiency hypothesis. Abstract presented at the 66th Annual Meeting of the Society for Biological Psychiatry, San Francisco, CA, May 12-14, 2011.

186. Weiner, MR, Schwab, ZJ, Rauch, SL, & **Killgore WD**. Personality factors predict brain responses to images of high-calorie foods. Abstract presented at the 66th Annual Meeting of the Society for Biological Psychiatry, San Francisco, CA, May 12-14, 2011.

187. **Killgore, WD,** Grugle, NL, & Balkin, TJ. Sleep deprivation impairs recognition of specific emotions. Abstract presented at the 25th Annual Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 11-15, 2011.

188. **Killgore, WD,** & Balkin, TJ. Does vulnerability to sleep deprivation influence the effectiveness of stimulants on psychomotor vigilance? Abstract presented at the 25th Annual Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 11-15, 2011.

189. Killgore, DB, **Killgore, WD,** Grugle, NJ, & Balkin, TJ. Sleep deprivation impairs recognition of specific emotions. Abstract presented at the 25th Annual Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 11-15, 2011.

190. Weiner, MR, Schwab, ZJ, & **Killgore, WD.** Daytime sleepiness is associated with altered brain activation during visual perception of high-calorie foods: An fMRI study. Abstract

presented at the 25th Annual Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 11-15, 2011.

191. Schwab, ZJ, Weiner, MR, & **Killgore, WD.** Functional MRI correlates of morningness-eveningness during visual presentation of high calorie foods. Abstract presented at the 25th Annual Meeting of the Associated Professional Sleep Societies, Minneapolis, MN, June 11-15, 2011.

192. **Killgore, WD,** Weiner, MR, & Schwab, ZJ. Daytime sleepiness affects prefrontal regulation of food intake. Abstract presented at the McLean Hospital Research Day, January 11, 2012.

193. Kipman, M, Schwab ZJ, Weiner, MR, DelDonno, S, Rauch SL, & **Killgore WD**. The insightful yet bitter comedian: The role of emotional versus cognitive intelligence in humor appreciation. Abstract presented at the McLean Hospital Research Day, January 11, 2012.

194. Weber, M, & **Killgore, WD**. Gray matter correlates of emotional intelligence. Abstract presented at the McLean Hospital Research Day, January 11, 2012.

195. Schwab, ZJ, & **Killgore, WD**. Sex differences in functional brain responses to food. Abstract presented at the McLean Hospital Research Day, January 11, 2012.

196. DelDonno, S, Schwab, ZJ, Kipman M, Rauch, SL, & **Killgore, WD**. The influence of cognitive and emotional intelligence on performance on the Iowa Gambling Task. Abstract presented at the McLean Hospital Research Day, January 11, 2012.

197. Song, CH, Kizielewicz, J, Schwab, ZJ, Weiner, MR, Rauch, SL, & **Killgore, WD**. Time is of the essence: The Design Organization Test as a valid, reliable, and brief measure of visuospatial ability. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

198. Kipman, M, Schwab, ZJ, DelDonno, S, & **Killgore, WD**. Gender differences in the contribution of cognitive and emotional intelligence to the left visual field bias for facial perception. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

199. Kipman, M., Schwab, ZJ, Weiner, MR, DelDonno, S, Rauch, SL, & **Killgore, WD**. Contributions of emotional versus cognitive intelligence in humor appreciation. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

200. Schwab, ZJ, & **Killgore, WD**. Disentangling emotional and cognitive intelligence. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

201. Schwab, ZJ, & **Killgore, WD**. Sex differences in functional brain responses to food. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

202. DelDonno, S, Schwab, ZJ, Kipman, M, Rauch, SL, & **Killgore, WD**. The influence of cognitive and emotional intelligence on performance on the Iowa Gambling Task. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

203. **Killgore, WD**, Britton, JC, Rosso, IM, Schwab, ZJ, Weiner, MR, & Rauch, SL. Shared and unique patterns of cortico-limbic activation across anxiety disorders. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

204. **Killgore, WD**, & Balkin, TJ. Sleep deprivation degrades recognition of specific emotions. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

205. **Killgore, WD**, & Schwab, ZJ. Emotional intelligence correlates with somatic marker circuitry responses to subliminal cues of facial trustworthiness. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

206. **Killgore, WD**, & Schwab, ZJ. Trust me! Neural correlates of the ability to identify facial trustworthiness. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

207. **Killgore, WD**, Schwab, ZJ, Weiner, MR, Kipman, M, DelDonno, S, & Rauch SL. Overeating is associated with altered cortico-limbic responses to images of high calorie foods. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

208. **Killgore, WD**, Weiner, MR, & Schwab, ZJ. Daytime sleepiness affects prefrontal regulation of food intake. Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

209. Weber, M, DelDonno, S, Kipman M, Schwab, ZJ, & **Killgore WD**. Grey matter correlates of self-reported sleep duration. Abstract presented at the Harvard Medical School Research Day, Boston, MA, March 28, 2012.

210. **Killgore, WD**. Overlapping and distinct patterns of neurocircuitry across PTSD, Panic Disorder, and Simple Phobia. Abstract presented at the 32nd Annual Conference of the Anxiety Disorders Association of America, Arlington, VA, April 12-15, 2012.

211. **Killgore, WD**, Britton, JC, Rosso, IM, Schwab, ZJ, & Rauch, SL. Shared and unique patterns of cortico-limbic activation across anxiety disorders. Abstract presented at the 67th Annual Meeting of the Society of Biological Psychiatry, Philadelphia, PA, May 3-5, 2012.

212. **Killgore, WD**, Schwab, ZJ, & Rauch, SL. Daytime sleepiness affects prefrontal inhibition of food consumption. Abstract presented at the 67th Annual Meeting of the Society of Biological Psychiatry, Philadelphia, PA, May 3-5, 2012.

213. Rosso, IM, Britton, JC, Makris, N, **Killgore, WDS**, Rauch SL, & Stewart ES. Impact of major depression comorbidity on prefrontal and anterior cingulate volumes in pediatric OCD. Abstract presented at the 67th Annual Meeting of the Society of Biological Psychiatry, Philadelphia, PA, May 3-5, 2012.

214. Kipman, M, Weber, M, DelDonno, S., Schwab, ZJ, & **Killgore, WD**. Morningness-Eveningness correlates with orbitofrontal gray matter volume. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

215. Kipman, M, Schwab, ZJ, Weber, M, DelDonno, S, & **Killgore, WD**. Yawning frequency is correlated with reduced medial thalamic volume. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

216. Weber, M, DelDonno, S, Kipman M, Schwab, ZJ, & **Killgore WD**. Grey matter correlates of daytime sleepiness. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

217. Weber, M, DelDonno, S, Kipman M, Schwab, ZJ, & **Killgore WD**. Grey matter correlates of self-reported sleep duration. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

218. DelDonno, S, Weber, M, Kipman M, Schwab, ZJ, & **Killgore, WD**. Resistance to insufficient sleep correlates with olfactory cortex gray matter. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

219. DelDonno, S, Schwab, ZJ, Kipman, M, Weber, M, & **Killgore, WD**. Weekend sleep is related to greater coping and resilience capacities. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

220. Schwab, ZJ, DelDonno, S, Weber, M, Kipman M, & **Killgore, WD**. Habitual caffeine consumption and cerebral gray matter volume. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

221. Schwab, ZJ, & **Killgore, WD**. Daytime sleepiness affects prefrontal regulation of food intake. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

222. **Killgore, WD**, Schwab, ZJ, DelDonno S, Kipman, M, Weber M, & Rauch, SL. Greater nocturnal sleep time is associated with increased default mode functional connectivity. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

223. **Killgore, WD**, Kamimori, GH, & Balkin, TJ. Caffeine improves efficiency of planning and sequencing abilities during sleep deprivation. Abstract presented at the 26th Annual Meeting of the Associated Professional Sleep Societies, Boston, MA, June 9-13, 2012.

224. Sneider, JT, **Killgore, WD**, Crowley, DJ, Cohen-Gilbert, JE, Schwab, ZJ, & Silveri, MM. Inhibitory capacity in emerging adult binge drinkers: Influence of Facial Cues. Abstract presented at the 35th Annual Scientific Meeting of the Research Society on Alcoholism, San Francisco, CA, June 23-27, 2012.

225. **Killgore WD**. Multimodal neuroimaging to predict cognitive resilience against sleep loss. Abstract presented at the DARPA Young Faculty Award 2012 Meeting, Arlington, VA, July 30-31, 2012. ***[*Winner Young Faculty Award in Neuroscience]***

226. Cohen-Gilbert, JE, **Killgore WD**, Crowley, DJ, Covell, MJ, Schwab, ZJ, Weiner, MR, Acharya, D, Sneider, JT, & Silveri, MM. Differential influence of safe versus threatening facial expressions on inhibitory control across adolescence and adulthood. Abstract presentede at the Society for Neuroscience 2012 Meeting, New Orleans, LA, October 13-17, 2012.

227. Weber, M, DelDonno, S, Kipman M, Schwab, ZJ, & **Killgore WD**. Grey matter correlates of self-reported sleep duration. Abstract presented at the Harvard Division of Sleep Medicine Annual Poster Session, Boston, MA, September 27, 2012.

228. Weber, M, DelDonno, SR, Kipman, M, Preer, LA, Schwab ZJ, Weiner, MR, & **Killgore, WD.** The effect of morning bight light therapy on sleep, cognition and emotion following mild traumatic brain injury. Abstract accepted for poster presentation at the 2012 Sleep Research Network Meeting, 22-23 October 2012, Bethesda, MD.

229. Sneider, JT, **Killgore, WD**, Crowley, DJ, Cohen-Gilbert, JE, Schwab, ZJ, & Silveri, MM. Inhibitory capacity in emerging adult binge drinkers: Influence of Facial Cues. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

230. Cohen-Gilbert, JE, **Killgore WD**, Crowley, DJ, Covell, MJ, Schwab, ZJ, Weiner, MR, Acharya, D, Sneider, JT, & Silveri, MM. Differential influence of safe versus threatening facial expressions on inhibitory control across adolescence and adulthood. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

231. Tkachenko, O, Schwab, ZJ, Kipman, M, DelDonno, S, Gogel, H., Preer, L, & **Killgore, WDS**. Smarter women need less sleep. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

232. DelDonno, S, Kipman, M, Schwab, ZJ, & **Killgore, WDS**. The contributions of emotional intelligence and facial perception to social intuition. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

233. Kipman, M, Schwab, ZJ, DelDonno, S, Weber, M, Rauch, SL, & **Killgore, WDS**. The neurocircuitry of impulsive behavior. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

234. Preer, LA, Tkachenko, O, Gogel, H, Schwab, ZJ, Kipman, M, DelDonno, SR, Weber, M, Webb, CA, & **Killgore, WDS**. Emotional intelligence as a mediator of the association between anxiety sensitivity and anxiety symptoms. Abstract presented at the Annual McLean

Hospital Research Day, January 16, 2013.

235. Gogel, H, DelDonno, S, Kipman M, Preer, LA, Schwab, ZJ, Tkachenko, O, & **Killgore, WDS**. Validation of the Design Organization Test (DOT) in a healthy population. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

236. Brennan, BP, Schwab, ZS, Athey, AJ, Ryan, EM, Pope, HG, **Killgore, WDS**, Jenike, MA, & Rauch, SL. A functional magnetic resonance imaging study of rostral anterior cingulate cortex activation in obsessive-compulsive disorder using an emotional counting stroop paradigm. Abstract presented at the Annual McLean Hospital Research Day, January 16, 2013.

237. Cohen-Gilbert, JE, Schwab, ZJ, **Killgore, WDS**, Crowley, DJ, & Silveri MM. Influence of Binge Drinking on the Neural Correlates of Inhibitory Control during Emotional Distraction in Young Adults. Abstract presented at the 3rd International Conference on Applications of Neuroimaging to Alcoholism (ICANA-3), New Haven, CT, February 15-18, 2013.

238. Weber, M, & **Killgore, WDS**. The interrelationship between 'sleep credit', emotional intelligence and mental health – a voxel-based morphometric study. Abstract presented at Harvard Medical School Psychiatry Research Day, April 10, 2013.

239. Cohen-Gilbert, JE, Schwab, ZJ, **Killgore, WDS**, Crowley, DJ, & Silveri MM. Influence of Binge Drinking on the Neural Correlates of Inhibitory Control during Emotional Distraction in Young Adults. Abstract presented at Harvard Medical School Psychiatry Research Day, April 10, 2013.

240. Mundy, EA, Weber, M, Rauch, SL, **Killgore, WDS**, & Rosso, IM. The relationship between subjective stress levels in childhood and anxiety as well as perceived stress as an adult. Abstract presented at Harvard Medical School Psychiatry Research Day, April 10, 2013.

241. Webb, CA, **Killgore, WDS**, Britton, JC, Schwab, ZJ, Price, LM, Weiner, MR, Gold, AL, Rosso, IM, Simon, NM, Pollack, MH, & Rauch, SL. Comparing categorical versus dimensional predictors of functional response across three anxiety disorders. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

242. Preer, LA, Tkachenko, O, Gogel, H, Schwab, ZJ, Kipman, M, DelDonno, SR, Weber, M, Webb, CA, Rauch, SL, & **Killgore, WDS**. Linking Sleep Trouble to Neuroticism, Emotional Control, and Impulsiveness. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

243. Preer, LA, Tkachenko, O, Gogel, H, Schwab, ZJ, Kipman, M, DelDonno, SR, Weber, M, Webb, CA, Rauch, SL, & **Killgore, WDS**. Emotional Intelligence as a Mediator of the Association between Anxiety Sensitivity and Anxiety Symptoms. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

244. Kipman, M, Schwab, ZJ, DelDonno, S, Weber, M, Rauch, SL, & **Killgore, WDS**. The

neurocircuitry of impulsive behavior. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

245. Weber, M, **Killgore, WDS**, Rosso, IM, Britton, JC, Simon, NM, Pollack, MH, & Rauch, SL. Gray matter correlates of posttraumatic stress disorder—A voxel based morphometry study. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

246. Weber, M, Penetar, DM, Trksak, GH, DelDonno, SR, Kipman, M, Schwab, ZJ, & **Killgore, WDS**. Morning blue wavelength light therapy improves sleep, cognition, emotion and brain function following mild traumatic brain injury. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

247. Tkachenko, O, Schwab, ZJ, Kipman, M, Preer, LA, Gogel, H, DelDonno, SR, Weber, M, Webb, CA, Rauch, SL, & **Killgore, WDS**. Difficulty in falling asleep and staying asleep linked to a sub-clinical increase in symptoms of psychopathology. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

248. **Killgore, WDS**, Schwab, ZJ, Kipman, M, DelDonno, SR, Rauch, SL, & Weber, M. Problems with sleep initiation and sleep maintenance correlate with functional connectivity among primary sensory cortices. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

249. **Killgore, WDS**, Schwab, ZJ, Kipman, M, DelDonno, SR, Rauch, SL, & Weber, M. A Couple of Hours Can Make a Difference: Self-Reported Sleep Correlates with Prefrontal-Amygdala Connectivity and Emotional Functioning. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

250. Brennan, BP, Schwab, ZS, Athey, AJ, Ryan, EM, Pope, HG, **Killgore, WDS**, Jenike, MA, & Rauch, SL. A functional magnetic resonance imaging study of rostral anterior cingulate cortex activation in obsessive-compulsive disorder using an emotional counting stroop paradigm. Abstract presented at the 68th Annual Meeting of the Society of Biological Psychiatry, San Francisco, CA, May 16-18, 2013.

251. Weber, M, & **Killgore, WDS**. The interrelationship between 'sleep credit', emotional intelligence and mental health – a voxel-based morphometric study. Abstract presented at the SLEEP 2013 Annual Meeting, Baltimore, MD, June 1-5, 2013.

252. Weber, M, Penetar, DM, Trksak, GH, DelDonno, SR, Kipman, M, Schwab, ZJ, & **Killgore, WDS**. Morning blue wavelength light therapy improves sleep, cognition, emotion and brain function following mild traumatic brain injury. Abstract presented at the SLEEP 2013 Annual Meeting, Baltimore, MD, June 1-5, 2013.

253. **Killgore, WDS**, Schwab, ZJ, Kipman, M, DelDonno, SR, & Weber, M. Problems with Sleep Initiation and Sleep Maintenance Correlate with Functional Connectivity Among Primary Sensory Cortices. Abstract presented at the SLEEP 2013 Annual Meeting, Baltimore, MD, June 1-5, 2013.

254. **Killgore, WDS**, Schwab, ZJ, Kipman, M, DelDonno, SR, & Weber, M. A Couple of Hours Can Make a Difference: Self-Reported Sleep Correlates with Prefrontal-Amygdala Connectivity and Emotional Functioning. Abstract presented at the SLEEP 2013 Annual Meeting, Baltimore, MD, June 1-5, 2013.

255. Tkachenko, O, Schwab, ZJ, Kipman, M, DelDonno, SR, Preer, LA, Gogel, H, Weber, M, Webb, CA, & **Killgore, WDS**. Difficulty in falling asleep and staying asleep linked to a sub-clinical increase in symptoms of psychopathology. Abstract presented at the SLEEP 2013 Annual Meeting, Baltimore, MD, June 1-5, 2013.

256. Preer, LA, Tkachenko, O, Gogel, H, Schwab, ZJ, Kipman, M, DelDonno, SR, Weber, M, Webb, CA, & **Killgore, WDS**. Linking Sleep Initiation Trouble to Neuroticism, Emotional Control, and Impulsiveness. Abstract presented at the SLEEP 2013 Annual Meeting, Baltimore, MD, June 1-5, 2013.

257. **Killgore, WDS**. Sleep duration contributes to cortico-limbic functional connectivity, emotional functioning, & psychological health. Abstract accepted for presentation at the 52nd Annual Meeting of the American College of Neuropsychopharmacology, Hollywood, FL, December 8-12, 2013.

258. Preer, L, Tkachenko, O, Gogel, H, Bark, JS, Kipman, M, Olson, EA, & **Killgore, WDS**. The role of personality in sleep initiation problems. Abstract presented at the Annual McLean Hospital Research Day, January 22, 2014.

259. Demers, LA, Olson, EA, Weber, M, Divatia, S, Preer, L, & **Killgore, WDS**. Paranoid traits are related to deficits in complex social decision-making and reduced superior temporal sulcus volume. Abstract presented at the Annual McLean Hospital Research Day, January 22, 2014.

260. Tkachenko, O, Weber, M, Gogel, H, & **Killgore, WDS**. Predisposition towards unhealthy foods linked with increased gray matter in the cerebellum. Abstract presented at the Annual McLean Hospital Research Day, January 22, 2014.

261. Olson, EA, Weber, M, Tkachenko, O, & **Killgore, WDS**. Daytime sleepiness is associated with decreased integration of remote outcomes on the IGT. Abstract presented at the Annual McLean Hospital Research Day, January 22, 2014.

262. Cui, J, Tkachenko, O, & **Killgore, WDS**. Can the activation of anterior cingulate predict the emotional suppression? An fMRI study with masked faces. Abstract presented at the Annual McLean Hospital Research Day, January 22, 2014.

263. Gogel, H, & **Killgore WDS**. A psychometric validation of the Design Organization Test (DOT) in a healthy sample. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

264. **Killgore, WDS**, Kipman, M, Tkachenko, O, Gogel, H., Preer, L, Demers, LA, Divatia, SC, Olson, EA, & Weber, M. Predicting resilience against sleep loss with multi-modal

neuroimaging. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

265. **Killgore, WDS**, Weber, M, Bark, JS, Kipman, M, Gogel, H, Preer, L, Tkachenko, O, Demers, LA, Divatia, SC, & Olson, EA. Physical exercise correlates with hippocampal volume in healthy adults. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

266. **Killgore, WDS**, Tkachenko, O, Weber, M, Kipman, M, Preer, L, Gogel, H, & Olson, EA. The association between sleep, functional connectivity, and emotional functioning. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

267. Preer, L, Tkachenko, O, Gogel, H, Bark, JS, Kipman, M, Olson, EA, & **Killgore, WDS**. The role of personality in sleep initiation problems. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

268. Tkachenko, O, Weber, M, Olson, EA, Gogel, H, Preer, LA, Divatia, SC, Demers, LA, & **Killgore, WDS**. Gray matter volume within the medial prefrontal cortex correlates with behavioral risk taking. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

269. Olson, EA, Weber, M, Bark JS, Demers L, Divatia, SC, Gogel, H, Kipman M, Preer, L, Tkachenko, O, & **Killgore, WDS**. Sex differences in threat evaluation of emotionally neutral faces. Abstract presented at the 42nd Annual Meeting of the International Neuropsychological Society, Seattle WA, February 12-15, 2014.

270. Cui, J, Tkachenko, O, & **Killgore, WDS**. Can the activation of anterior cingulate predict the emotional suppression? An fMRI study with masked faces. Abstract presented at the 36nd Annual Conference of the Anxiety Disorders Association of America, Chicago, IL, March 27-30, 2014.

271. Webb, CA, Weber, M, Mundy, EA, & **Killgore, WDS**. Reduced gray matter volume in the anterior cingulate, orbitofrontal cortex and thalamus as a function of depressive symptoms: A voxel-based morphometric analysis. Abstract presented at the 36nd Annual Conference of the Anxiety Disorders Association of America, Chicago, IL, March 27-30, 2014.

272. Weber, M, Penetar, DM, Trksak, GH, Kipman, M, Tkachenko, O, Bark, JS, Jorgensen, AL, Rauch, SL, & **Killgore, WDS**. Light therapy may improve sleep and facilitate recovery from mild traumatic brain injury. Abstract presented at the 10th World Congress on Brain Injury, San Francisco, CA, March 19-22, 2014.

273. Cui, J, Tkachenko, O, & **Killgore, WDS**. Can the activation of anterior cingulate predict the emotional suppression? An fMRI study with masked faces. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

274. Divatia, S, Demers, LA, Preer, L, Olson, EA, Weber, M, & **Killgore, WDS**. Advantageous decision making linked with increased gray matter volume in the ventromedial prefrontal

cortex. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

275. Demers, LA, Olson, EA, Weber, M, Divatia, S, Preer, L, & **Killgore, WDS**. Paranoid traits are related to deficits in complex social decision making and reduced superior temporal sulcus volume. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

276. Preer, LA, Weber, M, Tkachenko, O, Divatia, S, Demers, LA, Olson, EA, & **Killgore, WDS**. Gray matter volume in the amygdala is associated with facial assessments of trustworthiness. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

277. Tkachenko, O, Weber, M, Gogel, H, & **Killgore, WDS**. Predisposition towards unhealthy foods linked with increased gray matter volume in the cerebellum. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

278. Olson, EA, Weber, M, Gogel, H, & **Killgore, WDS**. Daytime sleepiness is associated with decreased integration of remote outcomes on the IGT. Abstract presented at the 21st Annual Meeting of the Cognitive Neuroscience Society, Boston, MA, April 5-8, 2014.

279. Demers, LA, Preer, LA, Gogel, H, Olson, EA, Weber, M, & **Killgore, WDS**. Left-hemifield bias on sad chimeric face task correlates with interpersonal emotional intelligence. Abstract presented at the 69th Annual Meeting of the Society of Biological Psychiatry, New York, NY, May 8-10, 2014.

280. Weber, M, **Killgore, WDS**, Olson, EA, Rosso, IM, & Rauch, SL. Morphological brain network organization in relation to trauma and posttraumatic stress disorder. Abstract presented at the 69th Annual Meeting of the Society of Biological Psychiatry, New York, NY, May 8-10, 2014.

281. Divatia, S, Demers, LA, Preer, L, Gogel, H, Kipman, M, & **Killgore, WDS**. Schizotypal and manic traits are associated with poorer perception of emotions in healthy individuals. Abstract presented at the 69th Annual Meeting of the Society of Biological Psychiatry, New York, NY, May 8-10, 2014.

282. **Killgore, WDS**, Weber, M, Olson, EA, & Rauch, SL. Sleep reduction and functioning of the emotion regulation circuitry. Abstract presented at the 69th Annual Meeting of the Society of Biological Psychiatry, New York, NY, May 8-10, 2014. ***[*Blue Ribbon Finalist for Top Poster Award: Basic Neuroscience]***

283. Webb, CA, Weber, M, Mundy, EA, & **Killgore, WDS**. Reduced gray matter volume in the anterior cingulate, orbitofrontal cortex and thalamus as a function of depressive symptoms: A voxel-based morphometric analysis. Abstract presented at the 69th Annual Meeting of the Society of Biological Psychiatry, New York, NY, May 8-10, 2014.

284. Marin MF, Song H, Landau AJ, Lasko NB, Foy Preer LA, Campbell A, Pace-Schott EF, **Killgore WD**, Orr SP, Pitman RK, Simon NM, Milad MR (2014). Psychophysiological and

Neuroimaging Correlates of Fear Extinction Deficits Across Anxiety Disorders. Abstract presented at the 69th Annual Meeting of the Society of Biological Psychiatry, New York, NY, May 8-10, 2014.

285. **Killgore, WDS**. The effects of sleep loss on food preference. Abstract presented at SLEEP 2014, Minneapolis, MN, May 31-June 4, 2014.

286. Weber, M, & **Killgore, WDS**. Sleep habits reflect in functional brain network organization. Abstract presented at SLEEP 2014, Minneapolis, MN, May 31-June 4, 2014. ***[*2014 AASM Young Investigator Award, Honorable Mention]***

287. Freed, MC, Novak, LA, **Killgore, WDS**, Koehlmoos, TP, Ginsberg, JP, Krupnick, J, Rauch S, Rizzo, A, Engle, CC. DoD IRB delays: Do they really matter? And if so, why and for whom? Abstract presented at the Military Health System Research Symposium, Fort Lauderdale, FL, August 18-21, 2014.

288. Freed, MC, Novak, LA, **Killgore, WDS**, Koehlmoos, TP, Ginsberg, JP, Krupnick, J, Rauch S, Rizzo, A, Engle, CC. DoD IRB delays: Do they really matter? And if so, why and for whom? Abstract accepted for presentation at the AMSUS Annual Meeting, Washington DC, December 2-5, 2014.

289. **Killgore, WDS**, Demers, LA, Olson, EA, Rosso, IM, Webb, CA, & Rauch, SL. Anterior cingulate gyrus and sulcus thickness: A potential predictor of remission following internet-based cognitive behavioral therapy for major depressive disorder. Abstract accepted for presentation at the 53rd Annual Meeting of the American College of Neuropsychopharmacology, Phoenix, AZ, December 7-11, 2014.

290. Olson, EA, Buchholz, J, Rosso, IM, **Killgore, WDS**, Webb, CA, Gogel, H, & Rauch, SL. Internet-based cognitive behavioral therapy effects on symptom severity in major depressive disorder: preliminary results from a randomized controlled trial. Abstract accepted for presentation at the 53rd Annual Meeting of the American College of Neuropsychopharmacology, Phoenix, AZ, December 7-11, 2014.

291. Brennan, B, Tkachenko, O, Schwab, Z, Ryan, E, Athey, A, Pope, H, Dougherty, Jenike, M, **Killgore, WDS**, Hudson, J, Jensen, E, & Rauch SL. Abstract accepted for presentation at the 53rd Annual Meeting of the American College of Neuropsychopharmacology, Phoenix, AZ, December 7-11, 2014.

292. Alkozei, A, Pisner, D, & **Killgore, WDS**. Emotional intelligence is differentially correlated with prefrontal cortical responses to backward masked fearful and angry faces. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

293. Alkozei, A, Schwab, Z, & **Killgore, WDS**. Looking for evil intent: Emotional intelligence and the use of socially relevant facial cues during an emotional decision making task. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

294. Shane, BS, Alkozei, A, & **Killgore, WDS**. The contribution of general intelligence and emotional intelligence to the ability to appreciate humor. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

295. Markowski, SM, Alkozei, A, & **Killgore, WDS**. Sleep onset latency and duration are associated with self-perceived invincibility. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

296. Pisner, D, Alkozei, A, & **Killgore, WDS**. Visuospatial reasoning mediates the relationship between emotion recognition and emotional intelligence. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

297. Vanuk, JR, Fridman, A, Demers, LA, Divatia, S, & **Killgore, WDS**. Engaging in meditation and internet based training as a means of enhancing emotional intelligence. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

298. Vanuk, JR, Divatia, S, Demers, LA, Markowski, SM, & **Killgore, WDS**. Napping in conjunction with brief internet-based training as a means of enhancing emotional intelligence. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

299. Cui, J, Tkachenko, O, Gogel, H, Kipman, M, Preer, LA, Weber, M, Divatia, SC, Demers, LA, Olson, EA, Buchholz, JL, Bark, JS, Rosso, IM, Rauch, SL, & **Killgore, WDS**. Fractional Anisotropy of frontoparietal connections presicts individual resistance to sleep deprivation. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

300. **Killgore, WDS**, Olson, EA, Weber, M, Rauch, SL, & Nickerson, LD. Emotional intelligence is associated with coordinated resting state activity between emotion regulation and interoceptive experience networks. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

301. **Killgore, WDS**, Demers, LA, Divatia, S, Kipman, M, Tkachenko, O, Weber, M, Preer, LA, Gogel, H, Olson, EA, Vanuk, JR, & Rauch, SL. Enhancing emotional intelligence via brief internet-based training. Abstract accepted for presentation at the 43rd Annual Meeting of the International Neuropsychological Society, Denver, CO, February 4-7, 2015.

302. Buchholz, JL, Rosso, IM, Olson, EA, **Killgore, WDS**, Fukunaga, R, Webb, CA, & Rauch, SL. Internet-based cognitive behavioral therapy is associated with symptom reduction and cognitive restructuring in adults with major depressive disorder. Abstract submitted for presentation at the Anxiety and Depression Conference, Miami, FL, April 9-12, 2015.

303. Alkozei, A, Markowski, SM, Shane, BR, Rauch, SL, & **Killgore, WDS**. Emotional resilience is not associated with increased emotional resistance to sleep deprivation. Abstract submitted

for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

304. Alkozei, A, Pisner, D, Markowski, SM, Rauch, SL, & **Killgore, WDS**. The effect of emotional resilience on changes in appetitie for high-sugary food during sleep loss. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

305. Markowski, SM, Alkozei, A, Rauch, SL, & **Killgore, WDS**. Self-perceived invincibility is associated with sleep onset latency and duration. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

306. Markowski, SM, Alkozei, A, Rauch, SL, & **Killgore, WDS**. Sex differences in the association between personality and resistance to sleep deprivation. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

307. Shane, BR, Alkozei, A, & **Killgore, WDS**. Physical exercise may contribute to vulnerability to sleep deprivation. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

308. Cui, J, Tkachenko, O, Gogel, H, Kipman, M, Sonis, LA, Weber, M, Divatia, SC, Demers, LA, Olson, EA, Buchholz, JL, Rosso, IM, Rauch, SL, & **Killgore, WDS**. Resistance to sleep deprivation involves greater functional activation and white matter connectivity within a fronto-parietal network. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

309. Vanuk, JR, Rosso, IM, Rauch, SL, Alkozei, A, Markowski, SM, Pisner, D, Shane, BR, Fridman A, Knight, SA, & **Killgore, WDS**. Daytime sleepiness is associated with altered thalamocortical connectivity. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

310. Sneider, JT, Jensen JE, Silveri, MM, & **Killgore, WDS**. Prefrontal GABA predicts resistance to sleep deprivation. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

311. **Killgore, WDS**, Tkachenko, O, Gogel, H, Kipman, M, Sonis, LA, Weber, M, Divatia, SC, Demers, LA, Olson, EA, Buchholz, JL, Rosso, IM, & Rauch, SL. Individual differences in rested activation of the ventral striatum predict overeating during sleep deprivation. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

312. **Killgore, WDS**, Tkachenko, Rosso, IM, Rauch, SL, & Nickerson, LA. Multimodal neuroimaging to predict resistance to sleep deprivation. Abstract submitted for presentation at the SLEEP 2015 Meeting, Seattle, WA, June 6-10, 2015.

313. Alkozei, A, Pisner, D, Rauch, SL, & **Killgore, WDS**. Emotional intelligence and subliminal presentations of social threat. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

314. Shane, BR, Alkozei, A, Vanuk, JR, Weber, M, & **Killgore, WDS**. The effect of bright light therapy for improving sleep among individuals with mild traumatic brain injury. Abstract

submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

315. Vanuk, JR, Shane, BR, Alkozei, A, & **Killgore, WDS**. Trait emotional intelligence is associated with greater resting state functional connectivity within the default mode and task positive networks. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

316. Vanuk, JR, Fridman, A, Demers, LA, & **Killgore, WDS**. Engaging in meditation and internet-based training as a means of enhancing emotional intelligence. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

317. Pisner, D, Alkozei, A, & **Killgore, WDS**. Trait emotional suppression is associated with decreased activation of the insula and thalamus in response to masked angry faces. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

318. Markowski, SM, Alkozei, A, & **Killgore, WDS**. The trait of neuroticism predicts neurocognitive performance in healthy individuals. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

319. Buchholz, JL, Rosso, IM, **Killgore, WDS**, Fukunaga, R, Olson, EA, Demers, LA, & Rauch, SL. Amygdala volume is associated with helplessness in adults with major depressive disorder (MDD). Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

320. Sneider, JT, **Killgore, WDS**, Rauch, SL, Jensen, JE, & Silveri, MM. Sex differences in the associations between prefrontal GABA and resistance to sleep deprivation. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

321. **Killgore, WDS**, Rosso, IM, Rauch, SL, & Nickerson, LD. Emotional intelligence correlates with coordinated resting state activity between brain networks involved in emotion regulation and interoceptive experience. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

322. **Killgore, WDS**, Demers, LA, Divatia, S, Rosso, IM, & Rauch, SL. Boosting Emotional intelligence with a brief internet-based program. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

323. **Killgore, WDS**, Vanuk, JR, Alkozei, A, Markowski, SM, Pisner, D, Shane, BR, Fridman, A, & Knight, SA. Greater daytime sleepiness correlates with altered thalamocortical connectivity. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

324. **Killgore, WDS**, Tkachenko, O, Gogel, H, Kipman, M, Sonis, LA, Divatia, SC, Demers, LA, Olson, EA, Buchholz, JL, Rosso, IM, & Rauch, SL. Activation of the ventral striatum predicts overeating during subsequent sleep loss. Abstract submitted for presentation at the 70th Annual Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

Narrative Report (limit to 500 words)

My research has emphasized the study of higher order cognition and executive functions and how these cognitive abilities are influenced and guided by subtle affective processes. Over the past 12 years, my research has utilized functional and structural magnetic resonance imaging to study the interaction of affective processes and cognition within limbic networks of the medial temporal lobes and prefrontal cortex. This line of research has led to the refinement of a developmental model of prefrontal cortical-limbic maturation that explains how these processes contribute to the way adolescents perceive emotionally and motivationally relevant stimuli such as affective faces and visual images of food. As a result of the Iraq War, I took an extended leave of absence to serve in the Active Duty Army as the Chief of the Neurocognitive Performance Branch at the Walter Reed Army Institute of Research from 2002-2007. During that time, I extended the scope of my affective processing research to also examine the effects of stressors such as prolonged sleep deprivation, chronic sleep restriction, nutritional deprivation, and the use of stimulant countermeasures on the cognitive-affective systems within the brain. This line of investigation suggests that sleep deprivation alters the metabolic activity within the medial prefrontal cortex, resulting in subtle but profound effects on specific aspects of cognition. These sleep-loss related prefrontal decrements impair the ability to use affective processes to guide judgment and decision-making, particularly in high-risk or morally relevant situations. My recent investigations also suggest that while commonly used stimulants such as caffeine, modafinil, and dextroamphetamine are highly effective at reversing sleep-loss induced deficits in alertness and vigilance, they have virtually no restorative effect on the cognitive-affective decision-making systems of the brain. Having left military service to return to McLean Hospital full time in the summer of 2007, I have since been extending my previous work to identify the extent to which these cognitive-affective decision-making systems and their neurobiological substrates are impaired or altered in patients suffering from anxiety disorders and post-traumatic stress. During the past five years I have also successfully secured multiple grants from the DoD and DARPA totaling more than $7.8M, including a study of the neural basis of emotional intelligence, a study of a novel light treatment for improving sleep and cognitive functioning in mTBI, and a neuroimaging study of the effectiveness of an internet based cognitive-behavior therapy program, a neuroimaging study of axonal damage in mTBI, and a study of the neural basis of resilience against the adverse effects of sleep deprivation. In early 2011, I was named Co-Director of the Social, Cognitive, and Affective Neuroscience Lab at McLean Hospital.

My recent teaching activities have primarily involved daily supervision and training of student research assistants and postdoctoral fellows, as well as occasional seminar presentations. Over the past 6 years, I have closely and regularly mentored more than 25 students at the undergraduate, graduate, and post-doctoral level. This involvement has included one-on-one supervision and training in basic research methods, neuropsychological assessment, statistical analysis, and manuscript preparation. Nearly all of my advisees have served as co-authors on abstracts, posters, talks, and published manuscripts based on my research program.

Personality Factors Predict Brain Responses to Images of High-Calorie Foods

Melissa R. Weiner, Zachary J. Schwab, Scott L. Rauch, & William D. S. Killgore

Affective Neuroscience Laboratory, McLean Hospital, Harvard Medical School

Of the many factors that may contribute to the current obesity epidemic, the relationship between personality traits and inhibitory neural circuits has been under-studied. We hypothesized that traits such as high Conscientiousness (C) and low behavioral impulsivity would be related to greater activation of inhibitory brain circuitry (i.e., prefrontal cortex) and reduced activation of reward systems (i.e., nucleus accumbens) and gustatory processing areas (i.e., insula) during visual perception of enticing high calorie food images.

Eleven healthy adults (5 men) aged 19 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing pictures of high- and low-calorie foods. In SPM5, contrast images comparing brain activation derived from the high- versus low-calorie conditions were correlated voxel-wise with C scores from the NEO-PI-R personality inventory and scores from the Barratt Impulsiveness Scale (BIS-11A) in a second-level regression model ($p<.005$, $k>10$).

As hypothesized, C positively correlated with greater activation to high-calorie foods in the left medial OFC and negatively with activation in the dorsal anterior cingulate gyrus and bilateral insula. BIS correlated positively with activation in the left nucleus accumbens ($r=0.89$, $p<.001$), and negatively with activation in the left lateral OFC ($r=-0.85$, $p=0.001$) and left anterior insula ($r=-0.92$, $p<.001$).

During visual perception of high-calorie food imagery, greater conscientiousness and lower impulsivity was associated with increased activation of cerebral regions involved in behavioral control and reduced activation within areas involved in reward and gustatory stimulation. Findings suggest a link between stable personality traits and brain responses to potentially unhealthy food options.

Emotional and Cognitive Intelligence: Support for the Neural Efficiency Hypothesis

Zachary J. Schwab, Melissa R. Weiner, Scott L. Rauch, & William D. S. Killgore

Affective Neuroscience Laboratory, McLean Hospital, Harvard Medical School

Emotional Intelligence (EI), the ability to understand, manage, and accurately perceive emotional information to guide decision making, is proposed to be distinct from cognitive intelligence (IQ). Construct validity for EI would be supported by functional activation distinct from that of IQ during emotion processing tasks. The neural basis of EI is proposed to involve the Damasio somatic marker circuitry (medial prefrontal cortex [MPFC], amygdala, and insula). We hypothesized that activation within these regions during an emotion processing task would negatively correlate with EI, but not IQ, suggesting greater neural efficiency among individuals with greater emotional capacities.

Twelve healthy adults (6 male) ranging from 19 to 45 years of age completed the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), the BarOn Emotional Quotient Inventory (EQ-i), and the Wechsler Abbreviated Scale of Intelligence (WASI). Subjects underwent functional magnetic resonance imaging (fMRI) while viewing a masked angry-face paradigm. Assessment scores were correlated voxel-wise with emotion circuitry activation during the task (masked anger > masked neutral contrast) using SPM5 ($p<.005$, $k^3 5$).

MSCEIT scores were negatively correlated with left insula and MPFC activation. Higher EQ-i was similarly associated with reduced bilateral insula and MPFC activation. Interestingly, WASI scores were also negatively correlated with reduced bilateral insula and MPFC activation.

Findings show that higher EI individuals required fewer neural resources in processing emotional information, consistent with the neural efficiency hypothesis. However, findings also show similar neural activation patterns for both EI and IQ, suggesting that these constructs ma not be as distinct as described by current theoretical conceptualizations.

Neural Correlates of Cognitive and Emotional Intelligence in Adults

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The ability to accurately perceive, understand, and manage emotional information is known as Emotional Intelligence (EI), a construct that is claimed to be distinct from traditional cognitive intelligence (IQ). Construct validity would be bolstered by evidence of neural processing of EI that is distinct from that of IQ during emotion processing tasks. We hypothesized that EI, but not IQ, would correlate negatively with neural responses in emotion processing regions of the amygdala, insula, and ventromedial prefrontal cortex (VMPFC), consistent with greater neural efficiency in higher ability individuals.

Twelve healthy adults ranging in age from 19 to 45 (6 male) underwent functional magnetic resonance imaging (fMRI) while viewing a masked angry-face perception paradigm that minimizes conscious perception of the affective stimulus. Two assessments of EI, the BarOn Emotional Quotient Inventory (EQ-i) and Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), as well as the Wechsler Abbreviated Scale of Intelligence (WASI), a measure of Full Scale intelligence (FSIQ), were administered. EQ-i, MSCEIT and FSIQ scores were correlated voxel-wise with emotion circuitry activation during the masked anger > neutral contrast using SPM5 ($p<.005$, $k\geq5$).

Higher EQ-i was associated with reduced left insula and MPFC activation. Similarly, higher MSCEIT was associated with reduced bilateral insula and MPFC activation. Interestingly, higher WASI scores were similarly correlated with reduced bilateral insula and MPFC regions. Amygdala activation was not correlated with EI or IQ.

Findings support the neural efficiency hypothesis (i.e., higher EI individuals recruit less neural resources to deal with emotional information), but also suggest that the neural activation patterns were highly similar to that seen for IQ. Findings suggest that the constructs of EI and IQ may share considerable variance and may not be as distinct as suggested by current theoretical conceptualizations.

Cognitive and Emotional Intelligences: Are they Distinct or Related Constructs?

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Emotional intelligence (EI), the ability to accurately perceive, understand, and manage emotional information to guide decision-making, is proposed to be a distinct construct, unrelated to personality or traditional cognitive intelligence (IQ). Despite widespread claims that indices of EI, such as the BarOn Emotional Quotient Inventory (EQ-i) and Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) are unrelated to IQ, there are no published studies correlating EI measures with the gold standard Wechsler scales of intelligence. We hypothesized that 1) EQ-i and MSCEIT would be correlated with one another, 2) EQ-i would correlate with personality but not IQ, and 3) the MSCEIT would correlate with IQ but not personality.

Thirteen healthy adults (7 females) ranging from 19 to 45 completed the MSCEIT, EQ-i, Revised NEO Personality Inventory (NEO), and the Verbal (VIQ), Performance (PIQ), and Full (FSIQ) scales of the Wechsler Abbreviated Scale of Intelligence (WASI). Data were analyzed with bivariate correlation and stepwise linear regression (alpha=.05).

MSCEIT and EQ-i were significantly correlated (r=0.62). The EQ-i correlated with FSIQ (r=0.74), VIQ (r=0.69), PIQ (r=0.72), Neuroticism (NEO-N) (r=-0.83), and Openness (NEO-O) (r=0.64). MSCEIT correlated with FSIQ (r=0.74), VIQ (r=0.67), PIQ (r=0.74), and NEO-O (r=0.71). In the regression analysis, MSCEIT was predicted by PIQ only (R=0.74). The EQ-i was significantly predicted by a linear combination of VIQ and NEO-N (R=0.92).

Contrary to the theoretical claims of EI, we find a significant correlation between measures of EI and IQ. As predicted, however, EQ-i shared significant variance with personality variables, and to a lesser extent, verbal IQ, whereas MSCEIT was most related to performance IQ. The findings clarify our understanding of emotional intelligence, showing that the two major models share significant common variance, but are each predicted by unique combinations of cognitive ability and personality.

Discrepancy Scores Between Cognitive and Emotional Intelligence Predict Neural Responses to Affective Stimuli

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Emotional intelligence (EI) is the ability to perceive, understand, and manage emotional information. As a construct, EI is posited to be independent of cognitive intelligence (IQ). We examined discrepancy scores between both constructs and correlated these difference scores with neural responses during a passive affect perception task. We hypothesized that discrepancies favoring EI over IQ ("Feeling" types) would correlate with task-related activation of limbic and paralimbic emotion processing regions than those with greater IQ than EI ("Thinking" types).

Twelve healthy adults ranging in age from 19 to 45 (6 male) underwent functional magnetic resonance imaging (fMRI) while viewing a masked angry-face perception task that minimizes conscious perception of the affective stimulus. Participants completed measures of EI (BarOn Emotional Quotient Inventory (EQ-i); Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT)), and IQ (Wechsler Abbreviated Scale of Intelligence (WASI)). Discrepancy scores (EQ-i–WASI; MSCEIT–WASI) were calculated and correlated voxel-wise with activation within the search territory defined by the medial prefrontal cortex, insula, and amygdala during the masked anger > neutral contrast using SPM5 ($p<.005$, $k\geq5$).

On the EQ-i, feeling scores correlated with activation of the right amygdala and anterior cingulate gyrus. Similarly, on the MSCEIT, feeling correlated with activation in the anterior cingulate gyrus. In contrast, discrepancy scores favoring a "thinking" style were unrelated to activation within the limbic and paralimbic search territories.

During a passive emotion-viewing task, participants with relatively greater EI than IQ scores showed increased activation within a network of regions involved in emotional processing. Findings support the construct validity of EI by showing that it may provide useful information about emotional functioning when juxtaposed with measures of related but distinct constructs.

Smart People Go with Their Gut: Emotional Intelligence Correlates with Non-Conscious Insular Responses to Facial Trustworthiness

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Emotional intelligence (EI) is the ability to accurately perceive, understand, and use emotional information to guide decision-making. The neural basis of EI is not well delineated but it has been proposed to involve the Damasio somatic marker circuitry (medial prefrontal cortex [MPFC], insula, and amygdala). We hypothesized that activation within this circuitry during subliminal presentations of facial cues of trustworthiness would be correlated with EI.

Twelve healthy adults (6 male; 6 female) ranging from 19 to 45 years of age completed the Bar-On Emotional Quotient Inventory (EQi) and Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT). During fMRI, participants viewed masked presentations of faces rated high (H) or low (L) in trustworthiness in a blocked paradigm. Conscious awareness of the trustworthiness of each face was effectively prevented via rapid presentation of the target (H or L) face (20 msec) followed by a neutral expression mask (80 msec). Contrast images comparing H vs L conditions were constructed in SPM5 and entered into second level regression analyses with EQi and MSCEIT. Three bilateral search territories comprising the somatic marker circuitry were interrogated ($p<.005$, $k\geq5$), including MPFC, insula, and amygdala.

Higher EQi scores were associated with reduced MPFC and increased anterior insula responses to lower trustworthiness in faces. EQi was unrelated to amygdala responses. Higher MSCEIT was similarly associated with greater left middle insula and dorsal anterior cingulate gyrus responses to low facial trustworthiness. Amygdala responses were unrelated to MSCEIT.

During subliminal perception of low facial trustworthiness, EI was associated with increased responsiveness of insular cortex, a region of the somatic marker circuitry posited to be critical for social emotions and interoceptive processing (i.e., "gut feelings"). Individuals with higher EI may be more interoceptively responsive to socially relevant stimuli.

Whom Can You Trust? Neural Correlates of Subliminal Perception of Facial Trustworthiness

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Judging the trustworthiness of others is critical to survival. Prior research suggests that overt perception of untrustworthiness activates the amygdala, but no study has yet examined how perceptual brain responses relate to behavioral discrimination of facial trustworthiness. We hypothesized that greater accuracy in discriminating trustworthiness would be related to activation of the amygdala and medial prefrontal cortex during subliminal presentation of trustworthiness cues.

Eleven healthy adults (6 male) ranging from 19 to 45 years of age underwent fMRI while viewing masked presentations of faces classified as either high (H) or low (L) in facial trustworthiness. Conscious awareness of trustworthiness information was prevented via rapid presentation of the target face (20 msec) followed by a neutral expression (N) mask (80 msec). Participants then made overt trustworthiness judgments (OTJ) for 100 pairs of similar faces previously rated on trustworthiness. Contrast images comparing H and L fMRI conditions with N were entered into a regression analyses with OTJ accuracy as the independent variable. Whole brain analyses were evaluated at $p<.001$, $k\geq20$ voxels. An amygdala search territory was interrogated at $p<.05$, k $k\geq5$ voxels.

OTJ accuracy ranged from 57% to 87%. During H>N, greater accuracy on the OTJ task correlated with increased activation within the right superior medial frontal gyrus. During L>N, OTJ accuracy correlated with increased activation within right superior frontal, middle frontal, medial orbitofrontal gyri, and left middle frontal gyrus. Greater accuracy was correlated with increased amygdala responses to facial untrustworthiness.

Accuracy in discriminating overt facial trustworthiness is related to the responsiveness of the medial prefrontal cortex and bilateral amygdala during subliminal presentations of facial features communicating trustworthiness information. Results support the hypothesized role of these regions in social evaluation.

Impulsiveness Predicts Responses of Brain Reward Circuitry to High Calorie Foods

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Impulsive individuals often fail to inhibit behavioral responses to rewarding stimuli. Thus, impulsiveness may be a risk factor for making unhealthy food choices and overeating. We hypothesized that impulsiveness would be positively correlated with activation in areas involved in the anticipation of reward (i.e., nucleus accumbens) and negatively correlated with regions involved in inhibitory control and evaluation of punishing stimuli (i.e., lateral orbitofrontal cortex) during passive perception of high-calorie food images.

Eleven healthy adults (5 men) aged 19 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing pictures of high-calorie foods, low-calorie foods, and control images of plants and rocks. Subjects viewed 5 alternating 30-second blocks of experimental and control stimuli, each consisting of ten images. Participants completed the Barratt Impulsiveness Scale (BIS-11A), a self-report questionnaire of impulsive personality traits. Contrast images comparing brain activation to high-calorie versus low- calorie conditions were created using SPM5 and then correlated voxel-wise with total BIS scores in a second-level regression model ($p<.005$, $k>10$).

As hypothesized, total BIS scores were positively correlated with activation for high-calorie versus low-calorie foods in the left nucleus accumbens ($r=0.89$, $p<.001$). BIS scores were negatively correlated with activation in the left lateral orbitofrontal cortex ($r=-0.85$, $p=0.001$) and left anterior insula ($r=-0.92$, $p<.001$).

Results are consistent with our hypothesis that when confronted with unhealthy high calorie food options, individuals with greater impulsiveness show increased activation in regions involved in the anticipation of reward and reduced activation within regions involved in suppression and control of appetite and behavior. Findings suggest a potential neurobiological link between impulsiveness and responses to food stimuli that may relate to unhealthy food intake.

Conscientiousness Predicts Brain Reponses to Images

of High-Calorie Foods

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With the growing obesity epidemic, it is important to understand the behavioral, characterological, and neural bases of human responses to unhealthy food stimuli. Conscientiousness (C), a personality trait defined by the tendency be self-disciplined, controlled, and motivated, may contribute to an individual's behavioral responses when confronted with unhealthy dietary choices. We hypothesized that C would be positively correlated with activation in areas involved in inhibitory control (i.e. prefrontal cortex) and negatively correlated with regions involved in hunger, craving, and other visceral responses (i.e., insula) during passive viewing of high-calorie food images.
Eleven healthy adults (5 men) aged 19 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing images of high-calorie foods, low-calorie foods, and control images of plants and rocks. Subjects viewed 5 alternating 30-second periods of experimental and control stimuli, each consisting of ten images (2500 msec stimulus presentation; 500 msec inter-stimulus interval). Subjects completed the Revised NEO personality inventory (NEO-PI-R), which includes a factor scale measuring C. Contrast images comparing high-calorie versus low-calorie conditions were correlated voxel-wise with C scores in a random-effects regression model in SPM5 ($p<.005$, $k>10$). C positively correlated with greater activation to high-calorie foods in the left medial orbitofrontal cortex. In contrast, C was negatively correlated with activation in the dorsal anterior cingulate gyrus as well as anterior and posterior insular cortex bilaterally.

Individuals with higher C responded to appetizing high calorie food images with increased activation of regions involved in inhibitory control and reduced activation within areas involved in craving, hunger, and visceral sensations. Understanding the neural basis of C may contribute to efforts help individuals modulate their responses to food and minimize dietary excesses.

Differential Influence of Facial Expression on Inhibitory Capacity in Adolescents versus Adults

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Objective : Adolescence is a time of notable alterations in cognitive functioning, including significant gains in behavioral self-control and an improved ability to ascribe emotional significance to stimuli. In the present study, we examined age differences in response inhibition using a Go No Go behavioral paradigm, which required subjects to respond or inhibit responding based on threat or safety cues present in the expression of facial stimuli.

Participants and Methods: Subjects were first required to respond (Go) to safe faces while inhibiting responding (No Go) to threatening faces, and then to respond (Go) to threatening faces and inhibit responding (No Go) to safe faces. Percent accuracy data for Go and No Go trials were acquired from 32 subjects, 19 adolescents aged 13.5 ± 0.9 years and 13 adults aged 33.8 ± 9.4 years.

Results : Adults exhibited significantly better accuracy, on both Go and No Go trials, when safe faces were presented (93% and 92%, respectively) compared to when threatening faces were presented (76% and 81%, respectively). While adolescents also exhibited significantly better accuracy for safe faces than for threatening faces, this pattern was only observed on Go trials (93% for safe faces and 84% for threatening faces). In contrast, adolescents performed significantly worse than adults on No Go trials, regardless of facial expression (61% for safe faces and 56% for threatening faces).

Conclusions : These findings suggest an age-related influence of facial expression on inhibitory capacity. Consistent with previous reports, adolescents in the present study demonstrated worse inhibitory control than adults. These data also indicate that while facial expression does not influence response inhibition in adolescents, the presence of a safe stimulus serves to enhance inhibitory capacity in adults. Thus, developmental changes in the ability to discriminate and utilize social information may contribute to improvements in inhibitory capacity with age. Supported by K01AA014651 & R01AA018153 (MMS).

Emotional and Cognitive Intelligence: Support for the Neural Efficiency Hypothesis

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Emotional Intelligence (EI), the ability to understand, manage, and accurately perceive emotional information to guide decision making, is proposed to be distinct from cognitive intelligence (IQ). Construct validity for EI would be supported by functional activation distinct from that of IQ during emotion processing tasks. The neural basis of EI is proposed to involve the Damasio somatic marker circuitry (medial prefrontal cortex [MPFC], amygdala, and insula). We hypothesized that activation within these regions during an emotion processing task would negatively correlate with EI, but not IQ, suggesting greater neural efficiency among individuals with greater emotional capacities.

Twelve healthy adults (6 male) ranging from 19 to 45 years of age completed the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), the BarOn Emotional Quotient Inventory (EQ-i), and the Wechsler Abbreviated Scale of Intelligence (WASI). Subjects underwent functional magnetic resonance imaging (fMRI) while viewing a masked angry-face paradigm. Assessment scores were correlated voxel-wise with emotion circuitry activation during the task (masked anger > masked neutral contrast) using SPM5 ($p<.005$, $k^3 5$).

MSCEIT scores were negatively correlated with left insula and MPFC activation. Higher EQ-i was similarly associated with reduced bilateral insula and MPFC activation. Interestingly, WASI scores were also negatively correlated with reduced bilateral insula and MPFC activation.

Findings show that higher EI individuals required fewer neural resources in processing emotional information, consistent with the neural efficiency hypothesis. However, findings also show similar neural activation patterns for both EI and IQ, suggesting that these constructs may not be as distinct as described by current theoretical conceptualizations.

Personality Factors Predict Brain Responses to Images of High-Calorie Foods

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Of the many factors that may contribute to the current obesity epidemic, the relationship between personality traits and inhibitory neural circuits has been under-studied. We hypothesized that traits such as high Conscientiousness (C) and low behavioral impulsivity would be related to greater activation of inhibitory brain circuitry (i.e., prefrontal cortex) and reduced activation of reward systems (i.e., nucleus accumbens) and gustatory processing areas (i.e., insula) during visual perception of enticing high calorie food images.

Eleven healthy adults (5 men) aged 19 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing pictures of high- and low-calorie foods. In SPM5, contrast images comparing brain activation derived from the high- versus low-calorie conditions were correlated voxel-wise with C scores from the NEO-PI-R personality inventory and scores from the Barratt Impulsiveness Scale (BIS-11A) in a second-level regression model ($p<.005$, $k>10$).

As hypothesized, C positively correlated with greater activation to high-calorie foods in the left medial OFC and negatively with activation in the dorsal anterior cingulate gyrus and bilateral insula. BIS correlated positively with activation in the left nucleus accumbens ($r=0.89$, $p<.001$), and negatively with activation in the left lateral OFC ($r=-0.85$, $p=0.001$) and left anterior insula ($r=-0.92$, $p<.001$).

During visual perception of high-calorie food imagery, greater conscientiousness and lower impulsivity was associated with increased activation of cerebral regions involved in behavioral control and reduced activation within areas involved in reward and gustatory stimulation. Findings suggest a link between stable personality traits and brain responses to potentially unhealthy food options.

Daytime Sleepiness is Associated Altered Brain Activation During Visual Perception of High-Calorie Foods: An fMRI Study

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With the average sleep duration in our population declining and the obesity epidemic on the rise, it is of particular importance to understand the relationship between sleep-related factors, brain responses to food, and eating behavior. Prior evidence suggests that healthy adults activate inhibitory regions of the prefrontal cortex in response to high- calorie food images. However, insufficient sleep is often associated with reduced metabolic activity within these same prefrontal regions. We hypothesized that greater daytime sleepiness would correlate with reduced prefrontal responses during passive viewing of appetizing high-calorie food images.

Twelve healthy adults (6 men) aged 19 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing pictures of high-calorie, low-calorie foods, and control images of plants and rocks. Subjects completed the Epworth Sleepiness Scale (ESS) for daytime sleepiness regarding their likeliness to doze during various activities (i.e reading, watching television, resting, etc). In SPM5, contrast images comparing brain activation derived from the high- versus low-calorie conditions were correlated voxel-wise with scores from the ESS sleepiness in a second-level regression model ($p<.005$, $k=10$).

As hypothesized, greater ESS scores correlated with reduced activation in the dorsolateral prefrontal cortex during perception of high- versus low-calorie food images. Greater daytime sleepiness was also associated with increased activation in the right parietal and inferior temporal cortex.

During the visual presentation of enticing high-calorie food images, greater sleepiness was associated with decreased activation in the prefrontal cortex, a region normally implicated in attention and inhibitory processing. Findings raise the speculative possibility that sleepiness may affect inhibitory control when confronted with highly appetizing high calorie foods. The extent to which the correlation between sleepiness and increased posterior cortex activation may reflect compensatory recruitment and whether these patterns may relate to actual food consumption remain unknown, but may be critical topics for further research.

Functional MRI Correlates of Morningness-Eveningness During Visual Presentation of High Calorie Foods

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Obesity rates are increasing while average hours of sleep are decreasing. It is unclear whether the link between short sleep duration and obesity might be related to sleep chronotype (i.e., Morningness-Eveningness). Evening types are more likely to have reduced sleep duration and quality than Morning types, who often show higher levels of conscientiousness and impulse control. The relationship between morningness-eveningness and functional brain responses to food has not been previously studied. Therefore, we examined this relationship using functional magnetic resonance imaging (fMRI). We hypothesized that Morningness would correlate with greater activity in the prefrontal attentional and behavioral control regions during a cognitively engaging task involving appetizing food images.

Twelve healthy adults (6 male) ranging from 19 to 45 years of age completed the Morningness-Eveningness Questionnaire (MEQ) and then underwent functional magnetic resonance imaging (fMRI) while viewing images of high and low calorie foods. Scores on the MEQ were correlated voxel-wise with brain activation during the task (high calorie foods > low calorie foods contrast) using linear regression in SPM5 ($p<.005$, $k^3 10$). All scans were performed in the afternoon between 1200 and 1600.

Morningness (higher MEQ scores) was associated with increased activation within the lateral prefrontal cortex. In contrast, Eveningness (lower MEQ scores) was associated with increased default mode network activation, including posterior cingulate and medial prefrontal cortex.

This study suggests that chronotype is associated with patterns of brain activation that may have implications for appetitive behavior and which may be relevant to the current obesity epidemic. Overall, greater morning preferences were associated with increased activation within prefrontal inhibitory networks when confronted with appetizing food images, while evening preferences were associated with a pattern commonly associated with behavioral disinhibition, self-referential thinking, and environmental disengagement. The extent to which these patterns relate to actual food consumption remain to be studied.

Daytime Sleepiness Affects Prefrontal Regulation of Food Intake

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Despite the alarming rate of obesity there has been minimal scientific progress in identifying and combating the causes of this epidemic. The prefrontal cortex is critical in the ability to modulate emotion and inhibit behavior. However, insufficient sleep is often associated with reduced metabolic activity within prefrontal regions. We tested whether daytime sleepiness would correlate with reduced prefrontal activation to appetizing high-calorie food images and whether this would predict difficulties modulating food intake.

Forty healthy adults (22 men) aged 18 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing pictures of high- and low-calorie foods. Subjects completed the Epworth Sleepiness Scale (ESS) and provided a rating to the query "how often do you eat more than you intend to." In SPM5, contrast images comparing brain activation derived from the high- versus low-calorie conditions were correlated voxel- wise with scores from the ESS in a second-level regression model ($p<.001$, $k=10$).

Daytime sleepiness correlated with reduced activation in the ventromedial prefrontal cortex during perception of high- versus low-calorie food images ($r=-.54$, $p<.001$). Moreover, activation within this cluster was related to the tendency to eat more than intended, but only for women ($r=-.47$, $p=.048$).

For participants viewing enticing high-calorie food images, greater daytime sleepiness was associated with decreased activation in the prefrontal cortex, a region implicated in emotional and behavioral modulation. Activation of this region was directly correlated with overeating in women. Findings suggest that normal fluctuations in sleepiness may be sufficient to affect brain regions important for regulating food intake.

The Insightful Yet Bitter Comedian: The role of Emotional versus Cognitive Intelligence in Humor Appreciation

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The ability to appreciate humor involves both cognitive and emotional processes. Prior research suggests that cognitive intelligence (IQ) is highly correlated with humor appreciation. We evaluated the individual and combined influences of IQ and emotional intelligence (EI) on performance on the Penn Humor Appreciation Test (HAT), a validated measure of the ability to appreciate subtle aspects of humor.

36 healthy adults (18 females) aged 18-45 completed the HAT, the Wechsler Abbreviated Scale of Intelligence (WASI) and two measures of EI; the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) and the Bar-On Emotional Quotient Inventory (EQ-i).

In a hierarchical linear regression, verbal and performance IQ were entered at the first level, followed by stepwise entry of MSCEIT and EQ-i subscales to predict HAT scores. IQ variables accounted for a significant proportion of the variance in HAT (R^2=0.34, p=0.001). Above and beyond IQ, the MSCEIT Understanding Emotions factor (b=0.57) and EQ-i General Mood factor (b=-0.29) each accounted for additional variance (combined model R^2=0.55, p=0.04). In a subsequent analysis, all IQ and EI subscales were entered stepwise to predict HAT performance. In combination, only MSCEIT Understanding (b=0.80) and EQ-i General Mood (b=-0.28) survived tolerance thresholds (R^2=0.53, p<0.001).

Both emotional and cognitive intelligence are correlated with humor appreciation. Findings suggest, however, that the most important factors contributing to humor appreciation ability include strong capacities related to labeling and reasoning with emotions in conjunction with a more negative general mood. EI appears to provide better prediction of humor appreciation ability than traditional measures of IQ.

Abstract presented at the McLean Hospital Research Day, January 11, 2012.

Grey Matter Correlates of Emotional Intelligence

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Recent theories of cognitive ability have emphasized the possibility of multiple intelligences that encompass a broader range of capacities than the traditional view of intellectual functioning. One such capacity, known as Emotional intelligence (EI), includes the ability to recognize, understand emotions in oneself and others, and to control and direct emotional processes adaptively to enhance decision-making. Damasio's influential Somatic Marker Hypothesis suggests an underlying neural network that is crucial to EI, including the amygdala, insula, and ventromedial prefrontal cortex. To evaluate the role of these structures in EI, we used voxel-based morphometry to examine the relationship between regional grey matter and standard measures of EI. We also examined two approaches to EI, including the Trait Model, which views EI as a stable trait similar to personality, and the Ability Model, which views EI as a set of capacities that are expressed through behavioral performance. 36 healthy participants completed the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) and the Baron-On Emotional Quotient Inventory (EQ-i) followed by structural magnetic resonance imaging (MRI) at 3T. Using SPM8, brain tissue images were first normalized to standard stereotaxic space. Then, using an automated algorithm of the VBM8 toolbox, images were segmented into grey matter, white matter, and cerebrospinal fluid, and spatially smoothed. Scores on the MSCEIT and EQ-i were correlated with grey matter volume of the Somatic Marker circuitry, $p < .001$, uncorrected, with cluster extent established empirically as the statistically expected number of voxels per cluster in each analysis. Measures of EI were significantly correlated with grey matter volume within several regions of the Somatic Marker circuitry. As hypothesized, total MSCEIT EI was positively correlated with grey matter volume in the left insula. When evaluated by subscale, the Strategic EI subscale correlated positively with right medial prefrontal cortex volume. In contrast, for the EQ-i, grey matter correlations were localized primarily within the ventromedial and orbitofrontal cortex regions, with findings particularly strong for the Stress Management subscale. The findings link trait and ability measures of EI to grey matter volume, suggesting that these constructs are related to well defined neuroanatomical substrates. Notably, the ability and trait models of EI were differentially associated with grey matter volume in distinct brain regions. Whereas trait EI, which is measured through self-report, was associated primarily with greater volume of medial prefrontal cortex regions which are often associated with self-reflective thought and introspection, while the ability to use emotional information in decision-making was primarily associated with greater volume of the insular cortex, a region involved in interoception and somatic-visceral sensations. These findings support the theoretical basis of the Somatic Marker circuitry in EI.

Abstract presented at the McLean Hospital Research Day, January 11, 2012.

Sex Differences in Functional Brain Responses to Food

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To effectively combat the growing epidemic of obesity it will be important to understand the neurobiological underpinnings of human responses to food stimuli. There are significant sex differences in the rates of eating disorders, and emerging evidence suggests that men and women may show differential responses to food stimuli within brain regions that are critically involved in appetite regulation and eating behavior. Here, we examined sex differences in neural responses to images of foods.

Forty healthy adults (22 men) ranging in age from 18 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing images of appetizing high-calorie and low-calorie foods. In SPM5, contrast images of brain activation (high-calorie foods > low-calorie foods) were created in a first level analysis and then compared between men and women in a two-sample t-test, while controlling for BMI ($p<.005$, $k\geq10$).
Men showed greater activation in response to high calorie foods than women in the anterior insular cortex (bilateral) and prefrontal cortex. Women showed greater activation in the right amygdala.
The brain responses of men and women to appetizing food imagery were significantly different in regions involved in gustatory and visceral responses (anterior insula), emotional salience (amygdala), and behavioral control (prefrontal cortex). Whereas women tended to activate a primary node in the emotional salience network when viewing enticing foods, men showed greater activation of inhibitory and visceral sensation regions, raising the possibility that observed sex differences in the prevalence of eating disorders may be related to differential activation of this neurocircuitry.

The Influence of Cognitive and Emotional Intelligence on Performance on the Iowa Gambling Task

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Emotional Intelligence (EI), the ability to accurately perceive, understand, manage, and use emotional information to solve problems, is purported to be a capacity distinct from traditional cognitive intelligence. We sought to validate the EI construct by examining the contribution of EI to performance on the Iowa Gambling Task (IGT), a behavioral index of the ability to use emotional cues to guide advantageous decision-making. Thirty-one healthy adults (16 females, ages 18-45) completed an "ability" test of EI (Mayer-Salovey-Caruso Emotional Intelligence Test; MSCEIT), a "trait" measure of EI (Bar-On Emotional Quotient Inventory; EQi), a measure of standard intelligence (Wechsler Abbreviated Scale of Intelligence; WASI), and the IGT. High and low EI groups were defined by a median split. Data were analyzed with repeated-measures ANCOVA.

For the MSCEIT, there was a main effect of EI group (p=.03), with high scorers showing better decision-making on the IGT than low scorers. However, this effect was no longer significant with IQ held constant (p=.11). Conversely, there was no main effect of EQi on IGT regardless of whether IQ was controlled (p=.62) or not (p=.82). Ability EI correlated significantly with performance on the last block of the IGT (r=.47), but this effect was lost after controlling for IQ.

Ability EI is a better predictor of performance on an emotional decision-making task than ability EI. However, the considerable shared variance between trait EI and standard intelligence appears to account for this effect. These findings raise doubts about the unique predictive validity of EI beyond standard cognitive intelligence.

Time is of the Essence: The Design Organization Test as a Valid, Reliable, & Brief Measure of Visuospatial Ability

Christina H. Song, Jill Kizielewicz, Zachary J. Schwab, Melissa R. Weiner, Scott L. Rauch, & William D. S. Killgore

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The Wechsler Scales are some of the most frequently used measures of intelligence. However, these scales are time consuming to administer, and there is a need for more time efficient measures that provide the same information. The Design Organization Test (DOT; Killgore et al., 2005) was developed as brief 2-minute alternative to the Wechsler Block Design (BD) subtest. The initial development study showed the DOT to be reliable and valid for assessing college students and clinical populations. The present study further examined the validity and reliability of the DOT in normal healthy adults.

36 healthy right-handed adults (13 male, 23 female) ranging in age from 18 to 45 completed the Wechsler Abbreviated Scale of Intelligence (WASI) and 2 alternative versions of the DOT. Test-retest reliability, alternate forms reliability, and concurrent validity were evaluated.

DOT scores correlated significantly with the WASI ($r=.73$, $p<.001$). Notably, Block Design (BD) scores were strongly correlated with the DOT, $r=.80$, $p<.001$. Alternate versions of the DOT were highly correlated with each other ($r=.82$, $p<.001$). Scores increased approximately 5 points between first ($M=36.03$, $SD=9.96$) and second ($M=41.00$, $SD=10.40$) administrations, $t(33) = -7.13$, $p<.05$, suggesting a small but reliable practice effect.

The DOT was found to be a valid measure of visuospatial ability that correlated highly with BD and total WASI scores. The DOT is recommended as an efficient alternative measure when the lengthy block design procedure is not practical.

Abstract presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal, CA, February 15-18, 2012.

Gender differences in the contribution of cognitive and emotional intelligence to the left visual field bias for facial perception

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Most right-handed individuals show a lateralized left visual field (LVF) bias in face processing, presumably due to right hemisphere dominance of this aspect of cognition. The magnitude of this bias is dependent on gender, as well as several cognitive and emotional characteristics. We examined the contributions of gender, cognitive intelligence (IQ), and emotional intelligence (EI) on the right hemisphere dominance for facial perception.

39 Healthy adults (21 males) aged 18-45 completed two Chimeric face tasks (Happy and Sad), measures of IQ (Wechsler Abbreviated Scale of Intelligence) and EI (Mayer-Salovey-Caruso Emotional Intelligence Test; MSCEIT & Bar-On Emotional Quotient Inventory; EQ-i).

Neither EI nor IQ predicts right hemisphere dominance in females. For males, IQ is correlated with greater LVF bias (Happy; r=0.320 p=0.047, Sad; r=0.402 p=0.011) while MSCEIT and EQ-i are not correlated. When IQ is controlled for, MSCEIT and EQ-i as full tests are not correlated with LVF bias in males. However, when EI is broken into subsets: MSCEIT Experiential strongly predicts less LVF bias when IQ is controlled for (Happy; r=-0.504 p=0.03, Sad; r=-0.491 p=0.03). A stepwise linear regression for sad faces in males accounts for 27% of the variance r=0.53 with IQ alone (b=0.569) and 45% of the variance r=0.67 when MSCEIT Experiential is added (b=-0.421). The effect of adding MSCEIT Experiential is significant at p=0.006.

This demonstrates that male LVF bias is predicted by cognitive intelligence, which increases the bias and experiential emotional intelligence, which decreases it. Strategic EI and EQ-i don't predict hemispheric dominance for face perception.

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Contributions of Emotional versus Cognitive Intelligence in Humor Appreciation

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The ability to appreciate humor involves both cognitive and emotional processes. Prior research suggests that cognitive intelligence (CI) is highly correlated with humor appreciation. We evaluated the individual and combined influences of CI and emotional intelligence (EI) on performance on the Penn Humor Appreciation Test (HAT), a validated measure of the ability to appreciate subtle aspects of humor.

36 healthy adults (18 females) aged 18-45 completed the HAT, the Wechsler Abbreviated Scale of Intelligence (WASI) and two measures of EI; the Meyer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) and the Bar-On Emotional Quotient Inventory (EQ-i).

In a hierarchical linear regression, verbal and performance CI were entered at the first level, followed by stepwise entry of MSCEIT and EQ-i subscales to predict HAT scores. CI variables accounted for a significant proportion of the variance in HAT (R^2=0.34, p=0.001). Above and beyond IQ, the MSCEIT Understanding Emotions factor (b=0.57) and EQ-i General Mood factor (b=-0.29) each accounted for additional variance (combined model R^2=0.55, p=0.04). In a subsequent analysis, all CI and EI subscales were entered stepwise to predict HAT performance. In combination, only MSCEIT Understanding (b=0.80) and EQ-i General Mood (b=-0.28) contributed independent predictive variance (R^2=0.53, p<0.001).

Both emotional and cognitive intelligence are correlated with humor appreciation. Findings suggest, however, that the most important factors contributing to humor appreciation ability include strong capacities related to labeling and reasoning with emotions in conjunction with a more negative general trait mood. EI appears to provide better prediction of humor appreciation ability than traditional measures of CI.

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Disentangling Emotional and Cognitive Intelligence

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Emotional intelligence (EI) has been described as the ability to perceive, understand, and use emotional information to facilitate thinking. While the construct of EI has garnered considerable lay attention over the past decade, there has been only modest scientific validation of the basis of this construct and whether it is indeed unique from traditional cognitive intelligence, as measured by the Wechsler scales (IQ). This issue has been clouded by contrary conceptualizations of EI as an "Ability" versus a "Trait" more akin to personality. To disentangle these constructs, we examined the inter-correlations among measures of EI, IQ, and personality.

Forty-one healthy adults (22 men) ranging from 18 to 45 completed the Bar-On EQ-I ("Trait" EI), Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT-"Ability" EI), Revised NEO Personality Inventory (NEO-PI-R), and the Verbal (VIQ), Performance (PIQ), and Full (FSIQ) scales of the Wechsler Abbreviated Scale of Intelligence (WASI). Data were analyzed with bivariate correlation and stepwise linear regression (alpha=.01).

MSCEIT and EQ-i were not significantly correlated (r=0.15). MSCEIT correlated with FSIQ (r=0.53), VIQ (r=0.53), and PIQ (r=0.43), but not personality. EQ-i was not correlated with IQ, but significantly correlated with Neuroticism (r=-0.65), Extraversion (r=0.49), and Conscientiousness (r=0.44). In regression analyses, EQ-i was predicted by a combination of Neuroticism, Conscientiousness, and Extraversion (R=0.83). MSCEIT was predicted by VIQ (R=0.53).

Ability and Trait measures of EI appear to be measuring different psychological constructs. Ability EI shares considerable variance with cognitive IQ (up to 28%), while Trait EI appears to be primarily a measure of personality.

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Sex Differences in Functional Brain Responses to Food

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There are significant sex differences in the rates of eating disorders, and emerging evidence suggests that men and women may show differential responses to food stimuli within brain regions that are critically involved in appetite regulation and eating behavior. Here, we examined sex differences in neural responses to images of foods.

Forty healthy adults (22 men) ranging in age from 18 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing images of appetizing high-calorie and low-calorie foods. In SPM5, contrast images of brain activation (high-calorie foods > low-calorie foods) were created in a first level analysis and then compared between men and women in a two-sample t-test, while controlling for BMI ($p<.005$, $k\geq10$).

Men showed greater activation in response to high calorie foods than women in the anterior insular cortex (bilateral) and prefrontal cortex. Women showed greater activation in the right amygdala.

The brain responses of men and women to appetizing food imagery were significantly different in regions involved in gustatory and visceral responses (anterior insula), emotional salience (amygdala), and behavioral control (prefrontal cortex). Whereas women tended to activate a primary node in the emotional salience network when viewing enticing foods, men showed greater activation of inhibitory and visceral sensation regions, raising the possibility that observed sex differences in the prevalence of eating disorders may be related to differential activation of this neurocircuitry.

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The Influence of Cognitive and Emotional Intelligence on Performance on the Iowa Gambling Task

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Emotional Intelligence (EI), the ability to accurately perceive, understand, manage, and use emotional information to solve problems, is purported to be a capacity distinct from traditional cognitive intelligence. We sought to validate the EI construct by examining the contribution of EI to performance on the Iowa Gambling Task (IGT), a behavioral index of the ability to use emotional cues to guide advantageous decision-making.

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For the MSCEIT, there was a main effect of EI group (p=.03), with high scorers showing better decision-making on the IGT than low scorers. However, this effect was no longer significant with IQ held constant (p=.11). Conversely, there was no main effect of EQi on IGT regardless of whether IQ was controlled (p=.62) or not (p=.82). Ability EI correlated significantly with performance on the last block of the IGT (r=.47), but this effect was lost after controlling for IQ.

Ability EI is a better predictor of performance on an emotional decision-making task than trait EI. However, the considerable shared variance between ability EI and standard intelligence appears to account for this effect. These findings raise doubts about the unique predictive validity of EI beyond standard cognitive intelligence.

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Emotional Intelligence Correlates with Somatic Marker Circuitry Responses to Subliminal Cues of Facial Trustworthiness

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Emotional intelligence (EI) involves the ability to accurately perceive, understand, and use emotional information to improve cognition. The neural basis of EI has not been well delineated but may involve the Damasio somatic marker circuitry (medial prefrontal cortex [MPFC], insula, and amygdala). We tested the hypothesis that activation within this circuitry would be correlated with measured of EI during subliminal presentations of untrustworthy faces.

Forty-one healthy adults (22 male) ranging from 19 to 45 years of age completed the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) and the Bar-On Emotional Quotient Inventory (EQi). Participants viewed faces varying in trustworthiness. Conscious awareness of trustworthy cues was minimized via rapid presentation of the target face (20 msec) and subsequent masking by a neutral expression (80 msec). Brain activation was correlated with EQi and MSCEIT. Three bilateral search territories comprising the somatic marker circuitry were interrogated ($p<.01$, $k\geq10$), including MPFC, insula, and amygdala.

Higher MSCEIT correlated with greater left insula and MPFC activation to low facial trustworthiness, but reduced activation of the rostral and middle cingulate gyrus and posterior orbitofrontal cortex. Higher EQi scores were associated with increased bilateral anterior insula responses and reduced amygdala responses to facial cues of untrustworthiness.

During subliminal perception of facial untrustworthiness, both measures of EI were associated with increased responsiveness of insular cortex, a region of the somatic marker circuitry posited to be critical for social emotions and interoceptive processing (i.e., "gut feelings"). Higher EI may involve increased interoceptive sensitivity to stimuli with high social relevance.

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Trust Me! Neural Correlates of the Ability to Identify Facial Trustworthiness

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The ability to identify trustworthy individuals is a critical aspect of human survival. Overt perception of untrustworthiness has been shown to activate the amygdala, but it is not clear how these patterns of activation relate to the actual ability to discriminate facial cues of trustworthiness.

Thirty-six healthy adults (20 male) ranging from 19 to 45 years of age underwent fMRI while viewing masked presentations of faces classified as either Trustworthy (T) or Untrustworthy. Conscious perception of trustworthiness cues was prevented via rapid presentation of the target face (20 msec), which was masked immediately by a neutral expression (N) mask (80 msec). Afterword, participants made overt trustworthiness judgments (OTJ) for 100 pairs of faces differing in qualities of trustworthiness. Contrast images comparing T and U fMRI conditions were regressed against OJT accuracy scores in SPM5. Whole brain analyses were evaluated at $p<.005$, $k\geq20$ voxels. A search territory within the amygdala was interrogated at $p<.01$, $k\geq5$ voxels.

OTJ accuracy ranged from 47% to 87%. During T>U contrasts, greater accuracy on the OTJ task correlated with increased activation within face processing regions of the fusiform and lingual gyri, and cerebellar vermis. During U>T contrasts, OTJ accuracy correlated with increased activation within affect processing regions such as the medial prefrontal cortex, insula, and hippocampus, and at a more liberal threshold, bilateral amygdala.

Individuals who were better at discriminating between overtly presented trustworthy and untrustworthy faces showed greater task-related activation of facial feature and affect processing systems during subliminal presentations of facial signals of trustworthiness.

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Overeating is Associated with Altered Cortico-Limbic Responses to Images of High Calorie Foods

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Developed countries are witnessing an alarming epidemic of obesity, yet the neurobiological underpinnings of excessive food intake remain poorly understood. Neuroimaging research has identified an important network of cortical and limbic regions that are activated by images of appetizing high calorie foods. Using whole brain functional magnetic resonance imaging (fMRI), we examined the correlation between self-reported difficulty modulating food intake and cortico-limbic responses to high-calorie food images. We hypothesized that the tendency to overeat would be associated with reduced activation of the prefrontal cortex, which is involved in behavioral inhibition, and increased responsiveness of limbic and paralimbic regions, which are involved in emotional and motivational processing.

During fMRI, 40 healthy adults (22 men) aged 18 to 45 viewed images of high- and low-calorie foods. Participants also completed several questions about dietary behavior. Contrast images comparing brain activation derived from the high- versus low-calorie conditions were correlated voxel-wise with responses to an excessive eating scale in a second-level regression model.

When viewing high- versus low-calorie foods, the tendency to eat more than intended was correlated with reduced activation within several regions of the dorsolateral prefrontal cortex bilaterally ($p<.001$), and increased activation of the right amygdala ($p<.005$).

When confronted with images of appetizing foods, self-reported difficulty regulating food intake was associated with reduced activation within regions of the brain purported to mediate behavioral control and increased activation of limbic regions involved in ascribing salience to motivationally relevant stimuli. Findings highlight a functional neurocircuitry that may be relevant to excessive food consumption.

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Daytime Sleepiness Affects Prefrontal Regulation of Food Intake

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Despite the alarming rate of obesity there has been minimal scientific progress in identifying and combating the causes of this epidemic. The prefrontal cortex is critical in the ability to modulate emotion and inhibit behavior. However, insufficient sleep is often associated with reduced metabolic activity within prefrontal regions. We tested whether daytime sleepiness would correlate with reduced prefrontal activation to appetizing high-calorie food images and whether this would predict difficulties modulating food intake.

Forty healthy adults (22 men) aged 18 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing pictures of high- and low-calorie foods. Subjects completed the Epworth Sleepiness Scale (ESS) and provided a rating to the query "how often do you eat more than you intend to." In SPM5, contrast images comparing brain activation derived from the high- versus low-calorie conditions were correlated voxel-wise with scores from the ESS in a second-level regression model ($p<.001$, $k=10$).

Daytime sleepiness correlated with reduced activation in the ventromedial prefrontal cortex during perception of high- versus low-calorie food images ($r=-.54$, $p<.001$). Moreover, activation within this cluster was related to the tendency to eat more than intended, but only for women ($r=-.47$, $p=.048$).

For participants viewing enticing high-calorie food images, greater daytime sleepiness was associated with decreased activation in the prefrontal cortex, a region implicated in emotional and behavioral modulation. Activation of this region was directly correlated with overeating in women. Findings suggest that normal fluctuations in sleepiness may be sufficient to affect brain regions important for regulating food intake.

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Morningness-Eveningness Correlates with Orbitofrontal Gray Matter Volume

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Individuals show considerable variability in preferences for diurnal activity and sleep. These preferences comprise a continuum of "morningness-eveningness," with morning chronotypes showing greater preference for activity in the morning hours and an earlier bedtime, while evening chronotypes show the opposite pattern. Despite the robustness of this phenomenon, little is known about the underlying neurobiological mechanisms that may contribute to these individual differences. Here we examined whether structural differences in prefrontal gray matter volume correlate with individual differences in circadian preferences.

36 healthy participants (20 males), ranging in age from 18-45, completed the Horne-Ostberg Morningness-Eveningness Questionnaire (MEQ) and underwent structural magnetic resonance imaging (MRI) at 3T. Using SPM8, brain tissue images were first normalized to standard stereotaxic space, segmented into grey matter, white matter, and cerebrospinal fluid, and spatially smoothed. Individual scores on the MEQ were correlated with gray matter volume of the orbitofrontal cortex after controlling for age and sex. This region was defined by the Wake Forest PickAtlas Toolbox for SPM.

MEQ scores ranged from 30 to 73 (M=50.4, SD=10.6). Greater eveningness (i.e., lower MEQ score) was significantly correlated with increased gray matter volume in the right lateral Orbitofrontal Cortex ($p<.001$, uncorrected; $k = 32$).

Individuals with stronger evening preferences tended to show increased gray matter volume in the Orbitofrontal cortex, a highly complex region of the brain that mediates complex executive functions such as set shifting and reward learning. Prior research has found that eveningness traits correlate with greater intelligence and verbal ability, but also with extraversion, impulsivity, and mood disturbance. The present findings suggest that some of these individual differences may be related to variability in prefrontal cortical structure and organization.

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Yawning Frequency is Correlated with Reduced Medial Thalamic Volume

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Although yawning is a universal human experience, its purpose and neurobiological mechanisms remain poorly understood. Some evidence suggests that yawns may occur to reduce cerebral temperature, to increase oxygen intake, or to communicate empathy. Little structural neuroimaging evidence exists to link brain morphology to yawning, but some evidence suggests that patients with lesions to the center-median nucleus of the thalamus show an unusual tendency to yawn when hyperventilating. Here we used voxel-based morphometry (VBM) to explore the link between yawning tendency and gray matter volume.

Thirty-six healthy participants aged 18 to 45 (20 males) rated their normal frequency of yawning on a scale from 1 (never yawn) to 10 (always yawning) followed by structural magnetic resonance imaging (MRI) at 3T. Structural T1-weighted neuroimaging data were preprocessed using the VBM toolbox in SPM8, including DARTEL-normalization to MNI space, tissue segmentation, and spatially smoothing with an 8mm FWHM Gaussian kernel. Yawning frequency was then entered as a covariate of interest, with age and gender as nuisance covariates, and modulated gray matter volumes as the dependent variable. Data were evaluated at a threshold of $p<.001$, uncorrected, with an empirically defined extent threshold of $k>72$ voxels, based on the statistically expected number of voxels per cluster.

Yawning frequency was negatively correlated with a single cluster (99 voxels) gray matter in the right posterior dorsomedial thalamus. No other regions were positively or negatively correlated with yawning frequency.

Self-reported yawning frequency was associated with reduced gray matter volume within the posterior medial thalamus, even after controlling for age and sex. As yawning is a poorly understood phenomenon, these preliminary findings raise the possibility that yawning may be related to arousal systems mediated by the medial or central nuclei of the thalamus.

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Grey Matter Correlates of Daytime Sleepiness

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Sleep deprivation has been associated with reduced glucose metabolism within the prefrontal cortex in healthy individuals. Within many of these same regions of the prefrontal cortex, grey matter volume appears to be reduced among patients with narcolepsy, obstructive sleep apnea, and chronic insomnia. It is, therefore, possible that prefrontal grey matter volume may be affected by chronic sleep loss or, conversely, may contribute to symptoms of sleep disorders. There are currently no data on grey matter correlates of daytime sleepiness in healthy individuals. Using voxel-based morphometry (VBM), we investigated the association between self-reported daytime sleepiness and grey matter volume. Based on the findings from experimental sleep deprivation and clinical findings, we hypothesized that daytime sleepiness would be associated with reduced grey matter volume in the prefrontal cortex.

36 healthy participants aged 18 to 45 (mean age 30.0±8.9; 20 males) completed the Epworth Sleepiness Scale (ESS) followed by structural magnetic resonance imaging (MRI) at 3 T. Using an automated algorithm of the VBM8 toolbox in SPM8, T1-weighted structural images were first DARTEL-normalized to MNI space, segmented into grey matter, white matter and cerebrospinal fluid, and spatially smoothed with an 8mm FWHM Gaussian kernel. Modulated images were used to provide an estimate of voxelwise grey matter volume. Scores of the ESS were correlated with grey matter volume, $p<.001$, uncorrected, with a cluster threshold of 40 voxels. Gender and age served as covariates.

In line with our hypothesis, daytime sleepiness negatively correlated with grey matter volume in a cluster of 48 voxels within the left orbitofrontal cortex (MNI coordinates x=-9, y=27, z=-26).

This is the first VBM study to link self-reported daytime sleepiness with reduced grey matter volume in the orbitofrontal cortex. As the orbitofrontal cortex is involved in decision-making and emotion processing, future studies should also investigate neuropsychological performance in this context.

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Gray Matter Correlates of Self-Reported Sleep Duration

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While the National Sleep Foundation recommends that most healthy individuals should obtain between 7 to 8 hours of sleep per night, evidence suggests that most people do not routinely obtain the sleep they need. Furthermore, some people seem to need more sleep than others to maintain similar levels of daytime performance. It is currently not known how typical sleep duration is related to structural differences in brain morphology. Here we examined the correlation between self-reported average sleep duration and gray matter volume using voxel-based morphometry (VBM) in healthy individuals.

Thirty-six healthy participants aged 18 to 45 (20 males) completed a questionnaire about their sleep habits and then underwent structural magnetic resonance imaging (MRI) at 3T. Data were preprocessed using the SPM8 VBM toolbox. Structural T1-weighted images were DARTEL-normalized to MNI space, tissue segmented, and spatially smoothed with an 8mm FWHM Gaussian kernel. Modulated images were used to provide an estimate of voxelwise grey matter volume. Self-reported sleep during the week and during weekends were combined as a weighted average and entered as the covariate of interest to predict gray matter volume, while gender and age were entered as nuisance covariates. Data were evaluated at a threshold of $p<.001$, uncorrected, $k>100$ voxels.

Average nighttime sleep was positively correlated with gray matter volume in bilateral insular cortices (Left 121 voxels; MNI coordinates x=-45, y=-1, z=-6; Right 418 voxels; MNI coordinates x=33, y=-4, z=4). No regions were negatively correlated with average sleep.

Greater self-reported average nightly sleep was associated with greater gray matter volume in the insular cortex bilaterally. This region is associated with integration of somatosensory and visceral sensations with emotional and motivational processes. Because these data are correlational, further research will be necessary to determine whether sleep duration leads to gray matter changes or whether gray matter volume affects sleep duration.

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Resistance to Insufficient Sleep Correlates with Olfactory Cortex Gray Matter

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Some evidence suggests that resistance to the cognitively degrading effects of sleep deprivation is partially related to prefrontal functioning and executive control. We have previously demonstrated that individuals with greater baseline olfactory identification capacities, a putative index of orbitofrontal cortex integrity, are better able to resist sleep deprivation up to three consecutive days when compared to individuals with poorer ability to discriminate and identify various smells. We hypothesized that individuals with greater self-reported resistance to sleep deprivation would have greater volume of the olfactory region of the orbitofrontal cortex using voxel-based morphometry (VBM).

Thirty-six healthy participants aged 18 to 45 (20 males) were queried about the threshold of sleep restriction that leads to a noticeable impairment in the ability to function at work (impairment threshold). Structural T1-weighted magnetic resonance images (MRI) were collected at 3T and analyzed using the SPM8 VBM toolbox. Images were DARTEL-normalized, segmented, and spatially smoothed (8mm FWHM). Impairment thresholds were correlated with gray matter volume in the olfactory cortex, using a small volume correction, $p<.05$, FWE for height and extent thresholds.

The self-reported impairment threshold ranged from 2 to 10 hours of minimal sleep necessary to avoid work impairments ($M=5.4$, $SD = 1.4$). As hypothesized, gray matter volume in the olfactory cortex was significantly negatively correlated with the impairment threshold, but this was only significant on the right side.

Larger gray matter volume in the right olfactory cortex, an area of the posterior orbitofrontal cortex, was associated with a greater self-reported ability to function effectively despite minimal amounts of sleep. Findings support the notion that prefrontal cortex integrity, including the olfactory cortex, confers some resistance to the degrading effects of sleep loss. Future research could examine the relationship between gray matter volume in this region and resistance to sleep loss under a controlled experimental environment.

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Weekend Sleep is Related to Greater Coping and Resilience Capacities

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Sleep deprivation has significant degrading effects on mood and emotional processes and has been linked to decreased behavioral coping abilities, increased risk-taking behavior, and increased scores on indices of some aspects of psychopathology. Notably, poor sleep is one of the most common symptoms reported among a diverse set of psychopathologies including PTSD, depression, and anxiety. Adequate sleep may play a protective role in preserving coping and resilience capacities. The present study investigated relationships between self-reported sleep quality during the workweek and on weekends and several facets of resilience.

Forty-four healthy individuals (ages 18-45, M = 30.0, SD = 8.7; 21 female) completed the Connor-Davidson Resilience Scale (CD-RISC), Invincibility Belief Index (IBI), NEO Personality Index Revised (NEO-PI-R), and a questionnaire asking about average sleep duration and sleep onset latency. Data were analyzed with Pearson's correlations.

Although average weekday sleep duration was unrelated to measures of resilience, weekend sleep duration was significantly correlated with higher scores on the CD-RISC and lower NEO Neuroticism ($p<.05$). Regarding the latency to fall asleep, individuals with shorter sleep onset latency on weekdays showed higher scores on the CD-RISC, global Invincibility, Audacity/Boldness/Courage, and lower Neuroticism ($p<.05$). Likewise, shorter sleep onset latency on weekends was related to higher CD-RISC, general invincibility, Audacity/Boldness/Courage, Adroitness/Cunning/Skill, and lower Neuroticism ($p<.05$).

Participants who reported obtaining more sleep and falling asleep more quickly, particularly on weekends reported greater resilience, boldness/courage, and lower neuroticism. Results suggest that emotional resilience may be mediated by the amount of sleep obtained on weekends and the latency to fall asleep. These findings suggest that "catching up" on sleep on weekends may actually have more beneficial effects on coping and resilience capacities than previously realized. Further research will be necessary to establish the causal direction of these relationships, however.

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Habitual Caffeine Consumption and Cerebral Gray Matter Volume

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Although caffeine is the most consumed stimulant in the world, little is known about its effects on brain structure. Some evidence suggests that caffeine may be protective against some types of dementia. One recent study reported that high or low levels of coffee consumption among women may be associated with larger hippocampal volume (Periaki et al., 2011). Here we examined the relationship between habitual caffeine intake and gray matter volume as measured by voxel-based morphometry (VBM).

Healthy participants (n=36), ranging in age from 18 to 45 (16 females) completed structural magnetic resonance imaging (MRI) at 3T. The T1-weighted scans were normalized to MNI space, tissue segmented, and spatially smoothed with an 8mm FWHM Gaussian kernel. Questionnaire information regarding habitual caffeine intake was transformed into estimated mg of caffeine based on data available from the website for the Center for Science in the Public Interest. Mean caffeine intake was entered as the covariate of interest, with age, gender, and weight as nuisance covariates and used to predict modulated gray matter volumes ($p<.005$, uncorrected, with an empirically defined extent threshold of $k>139$ voxels).

Caffeine intake was positively correlated with gray matter volume (1269 voxels) within the left medial temporal lobe, including the parahippocampal gyrus, hippocampus, amygdala, and fusiform gyrus. Caffeine intake was also associated with reduced gray matter volume in the superior medial prefrontal cortex (142 voxels).

Self-reported habitual caffeine consumption was associated with greater gray matter volume within medial temporal lobe structures critical for memory and emotional processing and reduced volume in a prefrontal region important for executive control and top down regulation of stress responses. Because of the bidirectional nature of the correlations, further research will be necessary to determine whether these differences in brain morphology cause increased consumption of caffeine, or whether the increased consumption produced the observed differences.

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Daytime Sleepiness Affects Prefrontal Regulation of Food Intake

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Background: Over the past few decades, there has been an unprecedented explosion in the rates of overweight and obesity, yet the neurobiological underpinnings of excessive food intake remain poorly understood. Notably, this epidemic corresponds closely with the decline in the average number of hours of sleep obtained each night. Because insufficient sleep has been linked to reduced metabolic activity within the prefrontal cortex and associated declines in inhibitory control, we hypothesized that daytime sleepiness would be related to reduced activation of the prefrontal cortex during perception of appetizing high-calorie foods and that this decline would be correlated with difficulties regulating food intake.

Methods: Forty healthy adults (22 men) aged 18 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing photographs of high- and low-calorie foods in a blocked design. Subjects also completed the Epworth Sleepiness Scale (ESS) and provided a rating to the query "how often do you eat more than you intend to" on a scale ranging from 1 (never) to 10 (always). In SPM5, contrast images of the difference in brain activation derived from the high- versus low-calorie conditions were correlated voxel-wise with scores from the ESS in a second-level regression model ($p<.001$, $k=10$).

Results: Daytime sleepiness correlated with reduced activation in the ventromedial prefrontal cortex during perception of high- versus low-calorie food images for the sample as a whole ($r=-.54$, $p<.001$). Moreover, activation within this cluster was related to the tendency to eat more than intended, but only for women ($r=-.47$, $p=.048$).

Conclusion: When presented with enticing high-calorie food images, greater daytime sleepiness was associated with decreased activation in the prefrontal cortex, a region implicated in emotional and behavioral control. Activation of this region was directly correlated with overeating in women but not men. Normal fluctuations in sleepiness may be sufficient to affect brain regions important for regulating food intake.

Greater Nocturnal Sleep Time is Associated with Increased Default Mode Functional Connectivity

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Sleep deprivation is associated with reduced cerebral metabolic activity, particularly within medial regions of the brain commonly associated with the default mode network. Recent evidence suggests that sleep deprivation also reduces the functional connectivity between the medial prefrontal cortex and the amygdala during emotional processing, possibly explaining some of the mood and emotional changes often associated with sleep loss. Here we examine the correlation between cerebral functional connectivity and the amount of sleep obtained the night preceding the neuroimaging scan among healthy volunteers who slept at home according to their own schedules.

Thirty-nine healthy individuals (ages 18-45, *M* = 30.4, *SD* = 8.7; 21 female) completed a questionnaire asking about their recent sleep habits. Participants underwent resting state functional magnetic resonance imaging (fMRI) for 6 minutes at 3T. Data were preprocessed in SPM8, including slice-time correction, segmentation, realignment, normalization, and spatial smoothing (6mm FWHM). The Functional Connectivity Toolbox (CONN) was used to regress out tissue- and movement-related nuisance covariates and to calculate seed-to-voxel and region-of-interest (ROI) to ROI random effects connectivity analyses. Analyses were corrected for multiple comparisons, $p<.05$, FDR.

Self-reported at home sleep ranged from 5.5 to 9 hours (M=7.4, SD = 0.84). More sleep was associated with significantly enhanced functional connectivity between the medial prefrontal cortex and dorsal posterior cingulate cortex, retrospenial cingulate, amygdalo-hippocampal region, and dorsal prefrontal cortex. Sleep was associated with greater positive connectivity between the posterior cingulate region and anterior prefrontal cortex, anterior cingulate, and medial prefrontal region, and greater anticorrelation with associative visual cortex.

Participants who obtained more sleep at home the night preceding their scan showed significantly enhanced functional connectivity among a network of structures involved in self-reflection, emotional control, and memory processing. The effect of this enhanced functional connectivity on cognitive performance and mood remains to be explored.

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INHIBITORY CAPACITY IN EMERGING ADULT BINGE DRINKERS: INFLUENCE OF FACIAL CUES

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Binge alcohol consumption has been associated with alterations in cognitive functioning, behavioral self-control and the ability to ascribe emotional significance to stimuli. In the current study, response inhibition was examined in emerging adult binge alcohol drinkers and light alcohol drinkers, using two Go No Go (GNG) behavioral paradigms, one that required response inhibition to shapes and one that required response inhibition to threat or safety cues present in the expression of facial stimuli. Percent accuracy data for GNG trials were acquired from 8 binge drinkers (BD) aged 21.5 ± 1.1 years and 10 light drinkers (LD) aged 22.2 ± 1.5 years. For shapes GNG, although no group differences were observed, LD exhibited similar accuracy on Go and No Go trials, whereas BD performed worse on No Go trials than on Go trials (p=.020). For faces GNG, significantly better accuracy was observed on Go trials for safe faces than for threatening faces in both LD (p=.016) and BD (p=.028). For faces No Go trials, LD demonstrated better accuracy on threat (p=.038) and safe trials (p=.009) than BD, whereas BD performed worse on No Go Trials regardless of facial cue. BD displayed significantly faster reaction times on faces Go trials, regardless of facial cue (p<.05), but not on shapes Go trials. These findings suggest that binge alcohol consumption is associated with impaired inhibitory capacity particularly in the presence of facial cues of threat and safety. These data also indicate that while facial cues do not differentially influence the poor response inhibition observed in BD, the presence of a safe stimulus serves to enhance inhibitory capacity in LD. Thus, drinking pattern-related differences in the ability to discriminate and utilize social information may compromise inhibitory capacity in binge drinkers, which may in turn impair social decision-making.

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Differential influence of safe versus threatening facial expressions on inhibitory control across adolescence and adulthood

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Inhibitory control improves throughout adolescence and into adulthood, due largely to the maturation of prefrontal cortex (PFC). Connectivity between PFC and limbic circuits critical to emotional processing also develop during this time. Thus, the impact of emotional information on response inhibition may change over the course of development. In the present study, developmental differences in response inhibition were examined using a Go-NoGo task that required subjects to respond or inhibit responding based on threat or safety cues present in the expression of facial stimuli. The task included two conditions: one in which subjects were required to respond (Go) to safe faces while inhibiting responding (NoGo) to threatening faces, and a second in which subjects were asked to respond (Go) to threatening faces and inhibit responding (NoGo) to safe faces. Inhibitory control was measured as percent accuracy on NoGo trials in each condition. Eighty-seven subjects (44 female) between 12 and 45 years of age completed this task. Subjects were subdivided into three age groups: adolescent (12-14 years, N = 33), emerging adult (18-25 years, N = 25) and adult (25-45 years, N = 29). Results showed a significant main effect of age on NoGo accuracy across conditions. Significant improvements in response inhibition were seen between the adolescent and the two adult groups, but not between the two adult groups. When NoGo accuracy for threatening versus safe stimuli was compared in each age group, the two adult groups showed significantly fewer impulsive errors for safe versus threatening faces. This effect was not significant in the adolescent group. Given the rapid cognitive changes occurring in early adolescence, this group was further subdivided into 12-13 year-olds (N = 20) versus 14 year-olds (N = 13). Analyses of these two groups revealed a significant interaction between age and face type for NoGo accuracy. While the younger group showed no difference in performance based on facial expression, the older adolescents showed a significant advantage on 'safe' NoGo trials compared to 'threat' trials, as was seen in the adult groups. These findings suggest a developmental change, early in adolescence, in the influence of safe versus threatening facial expressions on inhibitory capacity. Further studies will be needed to differentiate the effects of developmental changes in speeded recognition versus emotional reactivity to safe and threatening facial cues.

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Sex Differences in the Association Between Sleep and Intelligence

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The role of sleep in cognitive functioning remains poorly understood, though some developmental evidence implies that higher intellectual capacity may be associated with a decreased need for sleep. This study explored the relationship between habitual sleep and cognitive functioning in an adult population, focusing on differences between men and women.

55 healthy volunteers, aged 18 to 45 (mean age 29.8±7.9; 28 males) were administered the Wechsler Abbreviated Scale of Intelligence (WASI). Additionally, participants completed the Epworth Sleepiness Scale (ESS) and a questionnaire about their sleep habits. The Full Scale IQ on the WASI was correlated with the average number of hours of sleep on typical week- and weekend nights, with age, education, and Socioeconomic Status (SES) as covariates.

A significant negative correlation was observed between IQ scores and the average amount of sleep on both week- and weekend nights ($p<0.01$). Analysis by gender reveals that this correlation is significant only in females. These relationships remained significant when controlling for ESS scores and the amount of sleep obtained the night prior to administration of the WASI.

Findings suggest that females with greater intellectual ability require less sleep. Two possible explanations exist for this effect. The first supports the Neural Efficiency hypothesis, indicating that individuals with higher cognitive functioning may also display higher efficiency in neuronal recovery during sleep. The other suggests that individuals with shorter sleep duration benefit from a longer period of wakefulness and greater opportunity for cognitive stimulation. Several key aspects may account for the gender disparity: previously identified differences in brain morphology, particularly the role of white and gray matter in intellectual functioning; differences in levels of hormones, such as testosterone; as well as societal and cultural pressures specific to each gender, which may play into the differences in sleep habits and cognitive functioning.

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The Contributions of Emotional Intelligence and Facial Perception to Social Intuition

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Intuition can be defined as a quick, a-logical, subconscious process that enables an individual to accurately extract information from a situation, depending on the amount of previous exposure in that domain. Intuition may be a component of emotional intelligence (EI), which is the ability to perceive, manage, and understand emotions and use that information to enhance cognition and achieve goals. Previous work has suggested that EI may play a larger role than executive function in intuitive decisionmaking tasks, perhaps because EI and intuition capacities both recruit the "somatic marker" neurocircuitry. In the present study, we studied the influence of EI on a social intuition-based decision-making task. We aimed to identify factors that contribute to the ability to intuitively learn a nonexplicit rule for categorizing faces. We hypothesized that EI accounts for a greater proportion of the variance in social intuition than the ability to simply identify emotional facial expressions. Sixty-two healthy volunteers (ages 18– 45, M=30.2, SD=8.1; 31 females) completed the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), the Ekman 60 Faces Test (EFT), and an Intuition Task. The stimuli in the intuition task were taken from 300 computer-generated faces previously rated along 14 traits. These traits were reduced to a single dimension using Principal Components Analysis and the top and bottom 100 images were selected for the task. Without explicit explanation of the trait dimension, participants decided whether each face was high or low on the undefined attribute. Participants were provided with feedback on the accuracy of their responses in order to learn to facilitate learning. In a stepwise regression, total MSCEIT score emerged as a better predictor ($\beta = .401$, $p = .001$) of performance on the Intuition Task than EFT score ($\beta = .188$, $p = .15$). After dividing the MSCEIT into its four subscores and entering all variables into a linear regression, the Understanding subscore accounted for the most variance in the Intuition Task ($\beta = .536$, $p < .001$). EI predicted performance on the Intuition Task better than a test of emotional facial expression discrimination. This suggests that successful intuitive judgment of faces may require the subtle and complex skills encompassed by EI, to a greater extent than the ability to simply identify facial expressions of emotion. Additionally, the Understanding subscore strongly predicted Intuition Task performance, suggesting that this type of intuition may partly lie in the ability to label emotions, recognize groups of emotions, and understand the transitions between emotions.

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Linking Sleep Initiation Trouble to Neuroticism, Emotional Control, and Impulsiveness

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Sleep disturbance is often linked to negative affect and plays a role in psychopathology. In addition, impulsiveness and maladaptive thought control strategies have both been associated with insomnia and lack of sleep. It was hypothesized that trouble falling asleep would be related to a high degree of neuroticism, emotional control, and impulsiveness.

Sixty-one healthy adults (31 men) aged 18 to 45 completed a questionnaire about typical sleep habits, indicating whether they had trouble falling asleep and how many minutes they took to fall asleep, the Revised NEO Personality Inventory (NEO-PI-R), the Courtauld Emotional Control Scale (CECS), and the Barratt Impulsiveness Scale (BIS). A multivariate analysis of variance (MANOVA) was used to compare groups in terms of neuroticism, emotional control, and impulsiveness, and followed up with correlational analyses between these variables.

The MANOVA was significant (p=.015), and showed that trouble sleeping was associated with greater neuroticism (p=.013), emotional control (p=.042) and impulsiveness (p=.008). Minutes to fall asleep on weekdays was significantly positively associated with neuroticism (r=.475, p<.001) and impulsiveness (r=.394, p=.002), but not emotional control (p=.196).

Neuroticism, emotional control, and impulsiveness were higher in people with trouble falling asleep than normal sleepers. Likewise, minutes to fall asleep was associated with neuroticism and impulsiveness. These findings indicate that trouble falling asleep is related to degree of characteristic negative affect, the extent to which individuals are unable to cope with their negative emotions, and impulsiveness. Findings may have implications for treatment of sleep trouble, mood disturbance, and impulsive behavior.

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Emotional Intelligence as a Mediator of the Association between Anxiety Sensitivity and Anxiety Symptoms

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The construct of Anxiety Sensitivity (AS), which refers to the fear of the physical sensations, thoughts, and social consequences associated with anxiety, has been theorized to be a cognitive vulnerability that contributes to the development of an anxiety disorder. However, some evidence suggests that certain emotional factors may affect this relationship. We hypothesized that the level of Emotional Intelligence (EI) would mediate the relationship between AS and self-rated anxiety symptoms.

Sixty-one healthy adults (30 men) aged 18 to 45 (mean age 30.42 ±8.14) completed measures of AS (Anxiety Sensitivity Index, ASI), anxiety symptoms (Personality Assessment Inventory, PAI), a "trait" measure of EI (Bar-On Emotional Quotient Inventory, EQ-i), and two "ability" measures of EI (Mayer-Salovey-Caruso Emotional Intelligence Test, MSCEIT; SelfRated Emotional Intelligence Scale, SREIS). Mediation analyses were used to assess the influence of each of the measures of EI on the relationship between AS and anxiety symptoms.

Results: EQ-i was a significant partial mediator of the relationship between AS and PAI anxiety symptoms ($z=2.95$, $p=.003$). However, there were no mediation effects for the ability measures of EI, either for MSCEIT scores ($z=.61$, $p=.539$) or SREIS ratings ($z=.55$, $p=.583$), on the relationship between AS and anxiety symptoms.

Conclusion: Results showed that trait EI, but not ability EI, mediated the relationship between anxiety sensitivity and anxiety symptoms. Whereas the EQ-i measures a broad range of EI traits, which overlap with general emotional wellbeing, the MSCEIT and SREIS assess specific emotional skills. These findings suggest that factors related to emotional wellbeing, rather than specific emotional skills and abilities, mediate the relationship between anxiety sensitivity and anxiety symptoms. This may have implications for interventions designed to reduce anxiety by targeting the mediating factors.

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Validation of the Design Organization Test (DOT) in a Healthy Population

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A great deal of effort has been put into decreasing the duration of various neuropsychological measures in order to reduce patient fatigue and administration costs. The Design Organization Test (DOT; Killgore et al., 2005) was developed as a time-efficient alternative for the Block Design (BD) task that is a key subtest of the Wechsler Intelligence Scales. The DOT is a 2- minute paper-and-pencil task designed to assess visuospatial abilities. Participants use a coded key of block images to replicate images of various designs within a grid of squares. Although the DOT has been validated among clinical neurological patients, we sought to verify the reliability of the DOT in a healthy, more diverse population.

Two alternate versions of the DOT and the Wechsler Abbreviated Scale of Intelligence (WASI) were administered to sixty-one healthy participants between the ages of 18 and 45. Correlations were calculated to assess the reliability of both forms of the DOT.

Both forms of the DOT were found to correlate with the WASI BD task ($r = .756$, $p < .001$). The alternative forms of the DOT correlated highly with each other ($r = .802$, $p < .001$) regardless of the order of administration (Fisher's r-to-z transformation, $z = -.609$, ns.).

These data, along with previous work (Killgore et al., 2005), suggest that the DOT is a reliable and valid visuospatial measure in clinical and healthy populations. It is also possible for the DOT to be used as an equivalent alternative to the Block Design subtest of the WASI, allowing administrators to save time during a complete assessment of intelligence. The potential for verbal administration of this task could lead to use with a broader variety of patients.

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The Neurocircuitry of Impulsive Behavior

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The proclivity to make impulsive and potentially risky choices often has detrimental effects on an individual's health and wellbeing. Risk taking and impulsivity are particularly problematic for adolescents as well as for certain psychiatric populations such as patients with borderline personality disorder. The interplay between reward and cognitive control regions in the brain is thought to underlie an individual's propensity to engage in impulsive or risky behavior. Based on prior research suggesting that the nucleus accumbens mediates reward-seeking activity while the insula mediates loss aversion, we hypothesized that connectivity between these areas and orbitofrontal regions would predict individual variance in impulsivity. Fifty-eight healthy adults (29 females) ages 18 to 45 underwent functional magnetic resonance imaging (fMRI) at 3T while resting quietly with their eyes open for 6 minutes. Outside of the scanner participants completed the Barratt Impulsiveness Scale (BIS), which measures impulsive personality traits. A resting-state functional connectivity analysis was conducted using the CONN toolbox. Seed regions were placed bilaterally in the medial orbitofrontal cortex, lateral orbitofrontal cortex, insula, and nucleus accumbens. BIS scores ranged from 44 to 95 (M=61.90, SD=10.59). Impulsivity correlated with increased functional connectivity between the medial orbitofrontal cortex and the nucleus accumbens as well as with anticorrelated connectivity between the lateral orbitofrontal cortex and the insula. Participants who scored higher on the BIS had increased positive functional connectivity between the medial orbitofrontal cortex and nucleus accumbens and increased negative connectivity between the lateral orbitofrontal cortex and insula. These findings are congruent with literature on reward circuitry and risk taking propensity, and suggest that impulsiveness is associated with greater positive intercorrelations between reward areas and simultaneous inverse relationships between visceral sensation regions and brain regions involved in behavioral regulation. This pattern of neurocircuitry responses may promote impulsive and risky decisions.

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Difficulty in Falling and Staying Asleep Linked to a Sub-Clinical Increase in Symptoms of Psychopathology

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Sleep problems are linked with several mental disorders, particularly depression and anxiety. We have previously shown that laboratory sleep deprivation elicits significant increases in self-reported symptoms of psychopathology. Presently, we compared the effect of self-reported difficulty falling or staying asleep on these same indices of mental health concerns. We hypothesized that sleep problems would lead to a similar profile as reported previously during sleep deprivation.

Sixty-five healthy adults (33 males), aged 18-45 (30.2±8.0), completed a Sleep Questionnaire, reporting whether and how often they experienced trouble falling and/or staying asleep, and the Personality Assessment Inventory (PAI). Scores on the PAI clinical scales were compared between participants who had sleep difficulties (n=36, 19 males) and those that did not (n=29, 14 males), and correlated with frequency of weekly sleep disturbances.

Participants who endorsed sleep difficulties scored significantly higher ($p<0.05$) than those who did not on clinical scales measuring anxiety, anxiety-related disorders, depression, and schizophrenic symptoms. Subscale findings revealed that anxiety scores were elevated for cognitive, affective, and physiological dimensions, while anxiety-related disorders were driven by the phobias subscale. Depression scores were influenced by the cognitive and physiological subscales, while the elevated scores on the schizophrenia scale were driven by greater psychotic experiences scores. Higher instances of difficulty falling asleep showed significant positive correlations ($p<0.05$, Bonferroni corrected) with somatic complaints, anxiety, anxiety-related disorders, depression, schizophrenic symptoms, and borderline features.

Difficulty falling or staying asleep was associated with elevated sub-clinical psychopathology symptoms, similar to that reported during laboratory sleep deprivation. Furthermore, the increase in self-reported symptoms was strongly linked with the frequency of sleep disturbance. Although causal direction cannot be inferred, the similarity in symptoms to that produced by experimental sleep deprivation raises the possibility that sleep disturbance may serve as an underlying risk factor for disorders such as depression and anxiety.

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Problems with Sleep Initiation and Sleep Maintenance Correlate with Functional Connectivity Among Primary Sensory Cortices

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According to the hyperarousal theory of insomnia, difficulty initiating or maintaining sleep occurs as a result of increased cognitive and physiological arousal brought on by acute stressors and associated cognitive rumination, placing the individual in a perpetual cycle of hyperarousal and increased sensitivity to sensory stimulation. We tested the hypothesis that difficulty initiating or maintaining sleep would be associated with increased functional connectivity among primary sensory processing and motor planning regions.

Fifty-eight healthy adults (29 men, 29 women), between 18-45 years completed a self-report inventory about sleep onset and maintenance problems and underwent a 6-minute resting state functional MRI scan at 3T. Bilateral regions of interest (ROIs) were placed in primary visual cortex, auditory cortex, olfactory cortex, and the supplementary motor cortex and the mean processed signal timecourse was extracted and correlated with the other ROIs.

Difficulty falling asleep was associated with increased functional connectivity between the primary visual cortex and other sensory regions such as the primary auditory cortex, olfactory cortex, and the supplementary motor cortex. Primary auditory cortex also showed greater connectivity with supplementary motor cortex for those with sleep initiation problems. Problems with sleep maintenance were associated with greater connectivity between the primary visual cortex and olfactory cortex.

Consistent with the predictions of the hyperarousal model, difficulty falling asleep was associated with greater functional connectivity among primary sensory and supplementary motor cortices. Such augmented functional connectivity may contribute to sustained sensory processing of environmental stimuli, potentially prolonging the latency to sleep.

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A Couple of Hours Can Make a Difference: Self-Reported Sleep Correlates with Prefrontal-Amygdala Connectivity and Emotional Functioning

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Prior research suggests that sleep deprivation is associated with declines in some aspects of emotional intelligence and increased severity on indices of psychological disturbance. Sleep deprivation is also associated with reduced prefrontal-amygdala functional connectivity, potentially reflecting impaired top-down modulation of emotion. It remains unknown whether this "functional disconnect" may be observed in relation to more typical levels of sleep curtailment. We examined whether self-reported sleep duration the night before the assessment would be associated with these effects.

Sixty-five healthy adults (33 men, 32 women), ranging in age from 18-45 years documented their hours of sleep from the previous night, completed the Bar-On Emotional Quotient Inventory (EQ-i), Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), Personality Assessment Inventory (PAI), and underwent resting-state functional magnetic resonance imaging (fMRI). Connectivity data were analyzed using the Functional Connectivity Toolbox for SPM8.

Greater self-reported sleep the preceding night was associated with higher scores on all scales of the EQ-i but not the MSCEIT, and with lower symptom severity scores on half of the psychopathology scales of the PAI. Longer sleep was also associated with stronger inverse functional connectivity between the right ventromedial prefrontal cortex and right amygdala. Moreover, greater inverse connectivity between these regions was associated with higher EQ-i and lower symptom severity on the PAI.

Self-reported sleep duration from the preceding night is significantly correlated with inverse prefrontal-amygdala connectivity, perceived emotional intelligence, and the severity of subjective psychological distress. More sleep was associated with higher emotional and psychological strength.

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The interrelationship between 'sleep credit', emotional intelligence and mental health – a voxel-based morphometric study

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Sleep curtailment is common in today's society, but most people also acknowledge obtaining 'sleep credit' from time to time. This means that they not only meet their individual sleep need, but also get more sleep than they subjectively need to prevent degraded daytime performance. Given the rich data on adverse effects of insufficient sleep, one could argue that getting more sleep is better, but surprisingly, not much is known about behavioral and neuroanatomical correlates of 'sleep credit'.

Before undergoing structural magnetic resonance imaging, 55 healthy right-handed adults aged 18 to 45 (mean age = 30.74, SD = 8.13) completed a questionnaire about habitual sleep duration, minimum sleep needed before an impairment is noticed (in hours), and the Bar-On Emotional Quotient Inventory (EQ-i) and Personality Assessment Inventory (PAI). 'Sleep credit' was conceptualized as habitual sleep minus minimum sleep needed before impairment is noticed. A voxel-based morphometric whole-brain multiple regression examined structural correlates of habitual 'sleep credit' ($p < .001$, uncorrected, $k \geq 90$; nuisance covariates: age, gender, total intracranial volume), which were then used to predict perceived emotional intelligence and indices of psychopathology (Bonferroni-corrected $p < .017$).

Greater habitual subjective 'sleep credit' correlated with greater gray matter volume of clusters in the left medial prefrontal cortex (892 voxels, $T = 4.81$, MNI coordinates: $x = -6$, $y = 52$, $z = -21$) and right orbitofrontal gyrus (149 voxels, $T = 4.43$, MNI coordinates: $x = 39$, $y = 51$, $z = -18$). Extracted volume data from the medial prefrontal cortex cluster predicted greater interpersonal emotional intelligence capacities, and lower somatic complaints, symptoms of paranoia, and depression on the PAI.

These data support the medial prefrontal cortex's putative role in linking sleep and emotional functioning, but more importantly suggest that behavior and brain structure may vary with 'sleep credit'.

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Abstract accepted for presentation at the 27th Annual Meeting of the Associated Professional Sleep Societies, Baltimore, MD, June 1-5, 2013.

Problems with Sleep Initiation and Sleep Maintenance Correlate with Functional Connectivity Among Primary Sensory Cortices

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The hyperarousal theory of insomnia suggests that difficulty initiating or maintaining sleep occurs as a result of increased cognitive and physiological arousal brought on by acute stressors and associated cognitive rumination, placing the individual in a perpetual cycle of hyperarousal and increased sensitivity to sensory stimulation. We tested the hypothesis that difficulty initiating or maintaining sleep would be associated with increased functional connectivity among primary sensory processing and motor planning regions.

Fifty-eight healthy adults (29 men, 29 women), between 18-45 years completed a self-report inventory about sleep onset and maintenance problems and underwent a 6-minute resting state functional MRI scan at 3T. Bilateral regions of interest (ROIs) were placed in primary visual cortex, auditory cortex, olfactory cortex, and the supplementary motor cortex and the mean processed signal timecourse was extracted and correlated with the other ROIs.

26% of the sample reported difficulty falling asleep at least 1 or more times per week, while 19% had difficulty maintaining sleep at least 1 or more times per week. Difficulty falling asleep was associated with increased functional connectivity between the primary visual cortex and other sensory regions such as the primary auditory cortex, olfactory cortex, and the supplementary motor cortex. Primary auditory cortex also showed greater connectivity with supplementary motor cortex for those with sleep initiation problems. Problems with sleep maintenance were associated with greater connectivity between the primary visual cortex and olfactory cortex.

Consistent with the predictions of the hyperarousal model, difficulty falling asleep was associated with greater functional connectivity among primary sensory and supplementary motor cortices. Such augmented functional connectivity among poor sleepers may contribute to sustained sensory processing of environmental stimuli and motor restlessness, potentially prolonging the latency to sleep.

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Sleep Duration Contributes to Cortico-Limbic Functional Connectivity, Emotional Functioning, & Psychological Health

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Insufficient sleep has numerous adverse effects on cognitive and emotional functioning. Previous research has shown that total sleep deprivation is associated with degradation of some aspects of emotional intelligence, constructive thinking, frustration tolerance, and moral judgment, as well as increased severity on indices of psychological disturbance. While the causes of these changes are poorly understood, neuroimaging evidence suggests that sleep deprivation is associated with decreased metabolic activity in the prefrontal cortex and reduced prefrontal-amygdala functional connectivity. These alterations have been hypothesized to contribute to impaired top-down modulation of emotion. While such findings are apparent during prolonged total sleep deprivation, it remains unknown whether this altered connectivity may be observed during more typical levels of sleep curtailment, such as that experienced by most individuals from time to time. We examined whether self-reported sleep duration the night before the assessment would be associated with these effects.Sixty-five healthy adults (33 men, 32 women), ranging in age from 18-45 years documented their hours of sleep from the night preceding the assessment session, completed the Bar-On Emotional Quotient Inventory (EQ-i), Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), Personality Assessment Inventory (PAI), and underwent a 6-minute eyes-open resting-state functional magnetic resonance imaging (fMRI) on a 3T scanner. Connectivity data were analyzed using the Functional Connectivity Toolbox for SPM8. Correlations between functional connectivity and self-report inventories were Bonferroni corrected at $p < .05$.Greater self-reported sleep the night preceding the assessment was associated with higher scores on all scales of the EQ-i but not the MSCEIT, and with lower symptom severity scores on half of the psychopathology scales of the PAI, including reduced Anxiety, Depression, Paranoia, Schizophrenia, Alcohol Related Problems, and greater Treatment Resistance. Likewise, longer sleep duration was also associated with stronger inverse functional connectivity between the right ventromedial prefrontal cortex and right amygdala. These connectivity values were extracted and correlated with emotion and psychopathology scores. Overall, greater inverse connectivity between these regions was associated with higher EQ-i and lower symptom severity on the PAI, including Anxiety, Anxiety Related Disorders, Depression, Paranoia, Schizophrenia, Borderline Features, Suicide, and Stress, and greater Treatment Resistance. Self-reported sleep duration from the preceding night was significantly correlated with inverse prefrontal-amygdala connectivity, perceived emotional intelligence, and the severity of subjective psychological distress. These data suggest that even small variations in sleep of only 1 or 2 hours—a variation in sleep duration that is frequently encountered in everyday life—may be significantly associated with differences in some aspects of perceived emotional intelligence and the severity of psychological distress. Conversely, getting a full night of sleep appears to be connected with bolstered emotional strength and mental health.

The Role of Personality in Sleep Initiation Problems

Lily Preer, Olga Tkachenko, Hannah Gogel, John S. Bark, Maia Kipman, Elizabeth A. Olson, & William D.S. Killgore

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Difficulties initiating and maintaining sleep may be influenced by cognitive, affective, and behavioral factors. Some evidence suggests that particular personality traits are prone toward enhanced arousal, worry, rumination, and poor cognitive and behavioral control, which may contribute to difficulties falling asleep. Presently, we tested the hypothesis that sleep initiation problems would be related to the personality traits of neuroticism, impulsivity, and excessive emotional control.

Sixty-one healthy adults (31 males; 30 females) ranging in age from 18 to 41 completed a questionnaire about sleep problems and several measures of personality, including the NEO-PI-R, Barratt Impulsivity Scale (BIS 11), and Courtauld Emotional Control Scale (CECS). T-tests were used to determine whether individuals with self-reported trouble falling asleep differed from normal sleepers in terms of personality, emotional control, and impulsiveness. Pearson correlations were used to examine the association between the personality factors and average daily sleep onset latency. Logistic and multiple linear regression analyses were used to assess the combined influences of the personality factors on sleep onset problems.

Participants endorsing problems with sleep initiation scored higher on scales of neuroticism, impulsivity, and emotional control ($p<.05$). When personality traits were entered into a stepwise logistic regression, only impulsivity was retained as a significant predictor of the presence or absence of sleep onset difficulties ($p=.013$). When sleep latency in minutes was analyzed as a continuous variable, linear regression analyses revealed that both neuroticism and impulsivity were significant predictors of the self-reported time to fall asleep ($p=.030$).

Findings suggest that personality factors involved in negative emotional arousal and rumination are related to problems falling asleep, but that most of the variance appears to be attributable to deficits in cognitive and emotional control. Treatment approaches that address these cognitive and emotional control issues should be explored.

Key words: Insomnia; Sleep; Personality; Neuroticism; Impulsiveness; Emotional Control

Paranoid traits are related to deficits in complex social decision-making and reduced superior temporal sulcus volume

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Paranoid traits are often characterized by inaccurate interpretation of other people's motives. Individuals with high trait paranoia may have difficulties in interpersonal situations involving complex social judgments. Social decision-making has previously been related to superior temporal sulcus volume. To explore these relationships, 59 healthy adults (ages 18-45, *M*=30.66, *SD*=8.00; 30 males) underwent neuroimaging and completed the Personality Assessment Inventory (PAI) to measure paranoid tendencies, the Ekman 60 Faces Test (EFT) to control for potential differences in emotional face recognition abilities, and a novel task, the Facial Intuition Task (FIT), probing social complex decision-making. In FIT, participants decided whether computer-generated face stimuli were high or low on an unspecified trait determined by principal components analysis of previously rated traits. Trial-by-trial feedback was provided to help participants learn to make correct discriminations. Higher scores on the PAI paranoia scale correlated with lower average accuracy on FIT, even when controlling for basic emotional recognition scores on the EFT, $r(58)=-.33$, $p=.01$. Voxel-based morphometry, controlling for age and gender, was used to explore neural correlates of this finding, using a mask to restrict the analysis to the superior and middle temporal gyrus. Multiple regression analysis revealed a negative relationship between paranoia scores and a cluster of gray matter volume (k=69 voxels) in a region proximal to the superior temporal sulcus ($p<.001$, uncorrected; MNI:68,-43,6). Results suggest that paranoid traits are related to reduced gray matter volume in a region associated with social processing and poorer ability to integrate learning of subtle social cues into complex decision-making.

Predisposition Towards Unhealthy Foods Linked with Increased Grey Matter in the Cerebellum

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The role of the cerebellum is often confined to sensory-motor function. However, growing literature suggests its implication in higher cognitive and emotional processes, particularly executive control and the salience network. Furthermore, several areas have been linked with addiction mechanisms and nicotine dependence. Yet relatively little has been done to examine cerebellum structure with regard to salience.

Thirty-seven healthy right-handed adults (20 males) between the ages of 18 and 45 (M=30.3, SD=8.8) underwent structural neuroimaging at 3T and completed a Food Recognition Task. Participants viewed a series of images in the scanner of healthy and unhealthy foods. Afterwards, subjects were asked to discern whether or not they had previously viewed each image. Participants also indicated how hungry they were at the moment using a 7-point scale. A voxel-based morphometric (VBM) multiple regression analysis was conducted to explore the grey matter correlates of a predisposition to better remember unhealthy foods rather than healthy foods. Age and gender were used as covariates, along with total food recognition accuracy, subjective hunger, body mass index, and IQ.

Higher accuracy in the recognition of unhealthy foods compared with healthy foods was positively linked with increased grey matter volume in the cerebellum, particularly Crus I. Whole brain exploratory analyses indicated that grey matter volume within the Crus I was positively correlated with greater recall of unhealthy foods (1572 voxels, p=0.04, FWE corrected). In line with the literature, present results posit that the cerebellum may be implicated in salience detection.

Daytime sleepiness is associated with decreased integration of distant outcomes on the Iowa Gambling Task

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Negative effects of sleep loss on cognition have been robustly demonstrated. Though early research focused primarily on decrements in performance on basic aspects of cognition such as processing speed, in recent years the emphasis has shifted towards examining more complex aspects of decision-making and executive functioning. Multiple recent studies have demonstrated that individuals with sleep disorders and healthy individuals experiencing sleep deprivation show worse performance on the Iowa Gambling Task, a complex decision-making task that requires individuals to learn unstated reward and punishment contingencies. At the same time, methods to model IGT performance with respect to multiple underlying contributing factors have been developed. For example, the expectancy valence theory models IGT behavior as a function of three underlying parameters, which model attention to gains versus losses, emphasis on recent versus distant outcomes, and behavioral randomness. In this study, healthy individuals completed questionnaires regarding sleep. The Epworth sleepiness scale was used to assess daytime sleepiness. The standard version of the IGT was administered, and the expectancy valence model was applied. Increased sleepiness was related to increased emphasis on recent outcomes (versus more distant outcomes), Spearman's r (30) = 0.390, p =0.027. Sleep loss may affect behavior on decision-making tasks by shortening the time horizon over which information is integrated. This finding may have important implications for clinical populations, since sleep loss is a prominent feature of numerous psychiatric disorders.

A Psychometric Validation of the Design Organization Test (DOT) in a Healthy Sample

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Abbreviated tests of cognitive functioning are becoming increasingly necessary in order to reduce patient testing burden and to collect relevant clinical information in a time efficient and cost effective manner. The Design Organization Test (DOT; Killgore et al., 2005) was developed as a brief, 2-minute, paper-and-pencil alternative to the Wechsler Block Design (BD) subtest for evaluating visuospatial ability. The DOT was initially validated in a sample of high functioning university students and subsequently in a sample of clinical neurologic patients. The DOT showed good reliability (r=.80) and excellent correlation with the WAIS BD subtest (r=.92). To further develop the DOT, we presently examined its psychometric properties in a well-characterized sample of healthy adults.

Two alternate versions of the DOT and the Wechsler Abbreviated Scale of Intelligence (WASI) were administered to 61 racially and educationally diverse (11 to 20 years of formal education) healthy adult participants (30 males) ranging in age from 18 to 45 years (*M* = 30.3, *SD* = 8.1). Participants were screened to exclude significant medical, neurological, substance abuse, or psychiatric problems.

The DOT showed high alternate forms reliability (*r* = .90 to .92) and the two versions yielded equivalent levels of performance, suggesting that they are interchangeable. The DOT was highly correlated with raw BD (*r* = .76 to .79) and Full Scale IQ (r = .68 to .69), and yielded nearly identical outcomes when used in lieu of BD in the calculation of WASI IQ scores (i.e., total scores differed by less than 1/3 of an IQ point).

Findings provide further support for the reliability and validity of the DOT as a brief measure of visuospatial ability in a healthy population, and add to prior findings in clinical neurological patients and university students. The DOT may serve as an efficient estimate of intellectual functioning when lengthier tests may be excessively fatiguing or impractical.

Key words: Block Design; Validity; Reliability; Psychometrics; Assessment

Physical Exercise Correlates with Hippocampal Volume in Healthy Adults

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Physical activity has been shown to facilitate neurogenesis of dentate cells in the rodent hippocampus, a region of the brain critical for memory formation and spatial representation. As for humans, recent data suggest that physical exercise can lead to increased hippocampal volume and enhanced cognitive functioning in elderly individuals. However, no studies have examined the association between physical activity and hippocampal volume among healthy young to middle-aged adults.

Sixty-one healthy right-handed adult volunteers (33 males), ranging in age from 18 to 45, completed a questionnaire about their exercise habits and underwent structural neuroimaging at 3T. Three voxel-based morphometric (VBM) multiple regression analyses were conducted to examine the gray matter correlates of self-reported frequency of workouts per week, minutes per workout, and total weekly minutes of exercise (weekly frequency x minutes), with age and gender as nuisance covariates. The hippocampus was set as the primary region of interest (ROI, $p < .001$, $k \geq 20$).

Within the a priori hypothesized hippocampal ROIs, neither the frequency of workouts nor minutes per session correlated with hippocampal volume. However, total weekly minutes of exercise correlated significantly with larger gray matter volume in the right hippocampus (23 voxels, $p = .04$, FDR corrected). Whole brain exploratory analyses also showed that gray matter volume in several cortical areas, including the medial prefrontal cortex, insula, and postcentral gyrus was correlated with minutes of exercise per session and total minutes per week, but not with frequency of workouts per week.

Consistent with findings from animal studies and neuroimaging studies of the elderly, we found that the number of minutes of self-reported weekly physical exercise correlates with increased gray matter volume of the hippocampus and other cortical regions involved in memory and cognitive functioning even in healthy young to middle age adults.

The Association Between Sleep, Functional Connectivity, and Emotional Functioning

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Sleep deprivation is associated with altered emotional functioning and increased ratings of psychological distress. These behavioral changes correspond to patterns of reduced prefrontal metabolism and weakened prefrontal-amygdala functional connectivity, which may affect top-down modulation of emotion. Here, we expand this line of research beyond typical laboratory settings by studying whether self-reported sleep duration at home would be associated with altered cortico-limbic functional connectivity and emotional functioning.

Sixty-five healthy adults (33 men), ranging in age from 18-45 years completed a questionnaire about their sleep the previous night, as well as the Personality Assessment Inventory (PAI), Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), and Bar-On Emotional Quotient Inventory (EQ-i), followed by resting-state functional magnetic resonance imaging (fMRI). The Functional Connectivity Toolbox for SPM8 was used for data analysis.

Self-reported sleep the preceding night was not associated with MSCEIT scores, but was correlated with higher scores on the EQ-i and lower scores on the psychopathology scales of the PAI. Sleep duration was also associated with stronger negative functional connectivity between the right ventromedial prefrontal cortex and right amygdala. Moreover, the magnitude of this inverse connectivity was associated with higher self-reported emotional intelligence and fewer symptoms of psychopathology.

Sleep duration the night before the scan was significantly correlated with greater inverse prefrontal-amygdala connectivity, higher perceived emotional intelligence, and lower psychological distress. Thus, even variations in a single night of sleep are significantly related to the strength of functional connectivity and emotional functioning the following day.

Key words: Sleep; Psychopathology; Emotional Intelligence; Amygdala; Prefrontal Cortex; Functional Connectivity

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The Role of Personality in Sleep Initiation Problems

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The arousal theory of insomnia posits that problems with sleep onset are secondary to excessive activation of cognitive, affective, and physiological systems that hinder sleep. Evidence suggests that certain personality traits are prone to arousal, worry, and poor cognitive control, which may contribute to sleep onset problems. We hypothesized that sleep onset insomnia would be related to the personality traits of neuroticism, impulsivity, and excessive emotional control.

Sixty-one healthy adults (31 males; 30 females) ages 18 to 41 completed a questionnaire about sleep problems and measures of personality, including the NEO-PI-R, Barratt Impulsivity Scale, and Courtauld Emotional Control Scale. T-tests were used to determine whether individuals with self-reported trouble falling asleep differed from normal sleepers in terms of personality, emotional control, and impulsiveness. Pearson correlations were used to examine the association between the personality factors and minutes to fall asleep on weekdays and weekends. Logistic and multiple linear regression analyses were used to assess the combined influences of the personality factors on sleep onset problems.

Participants with sleep initiation problems scored higher on neuroticism, impulsivity, and excessive emotional control ($p<.05$). When personality traits were combined using stepwise logistic regression, only impulsivity was a significant predictor of sleep onset problems ($p<.05$). When linear regression analyses were conducted, only neuroticism predicted sleep latency on weekdays, whereas impulsivity was the only predictor of sleep latency on weekends.

Findings suggest that personality factors involved in negative emotional arousal are related to sleep onset problems, but most of the variance is attributable to deficits in cognitive and emotional control. Treatment approaches that address these cognitive and emotional control issues should be explored.

Key words: Insomnia; Sleep; Personality; Neuroticism; Impulsiveness; Emotional Control

Abstract submitted for presentation at the 42nd Annual Meting of the International Neuropsychological Society, Seattle, WA, February 12-15, 2014.

Gray Matter Volume within the Medial Prefrontal Cortex Correlates with Behavioral Risk Taking

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Risk-taking behavior has been associated with altered functioning of the ventromedial prefrontal (vmPFC) and dorsolateral prefrontal cortex (dlPFC). The Balloon Analog Risk Task (BART) is a behavioral task that has been used to examine risk-taking behavior by having participants win money by inflating a virtual balloon. Value accrues with additional pumps to inflate the balloon, but all money is lost if the balloon pops before it is cashed in. Little is known about the relation between prefrontal gray matter volume and BART performance. We explored the structural neural substrates of this task using voxel-based morphometry (VBM).

Fifty healthy right-handed individuals (25 males) between the ages of 18 and 45 underwent structural neuroimaging at 3T, and completed an offline version of the Balloon Analogue Risk Task (BART), a behavioral measure of risk-taking. Two VBM multiple regression analyses were conducted using SPM8 to explore the gray matter correlates of risk taking tendencies exhibited on the BART (whole brain $p < .001$, $k \geq 68$ voxels). Risk taking was assessed with two indices from the BART: 1) the adjusted average number of pumps (AP) and 2) a calculated cost to benefit ratio (CBR) based on the percent of total exploded balloons versus the percentage of money won. Higher scores indicate greater risk-taking. Both age and gender served as covariates.

Greater AP was positively associated with larger gray matter volume in the ventromedial prefrontal and left lateral orbitofrontal cortex, while increased CBR correlated significantly with increased gray matter volume in the medial gyrus rectus.

Findings suggest that gray matter volume within the ventromedial prefrontal and orbitofrontal cortex is positively correlated with risk-taking behavior as measured by the BART. However, further work may be necessary to elucidate the degree to which this reflects actual risk-taking behavior versus response optimization to maximize potential winnings.

Key words: Risk-Taking; BART; Prefrontal Cortex; VBM

Sex differences in threat evaluation of emotionally neutral faces

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The amygdala and posterior face-sensitive brain regions are involved in the evaluation of emotionally neutral faces. Sex differences in neural activation in response to threatening facial stimuli have been previously identified. We examined whether healthy men and women would differ in their risk assessment of neutral faces and whether this would influence decisions related to potential threat. We further examined whether risk-related decisions would be associated with variations in emotional intelligence and differences in gray matter volume of face responsive regions (i.e., amygdala; fusiform gyrus).

62 participants (31 F, 31 M) ages 18-45 completed a facial threat assessment task requiring them to act as a simulated airport security agent, deciding whether to allow each of 66 pictured individuals displaying neutral expressions to board an airplane during a terrorist threat. Participants also completed the Bar-On Emotional Quotient Inventory (EQ-i) and underwent MRI scanning at 3T. Voxel-based morphometric (VBM) analysis was conducted in SPM8.

Women allowed a higher number of passengers to board than men ($p=.02$). Men who scored lower in stress management ability allowed fewer passengers, whereas more stress-tolerant men performed comparably to women. Greater leniency was correlated with reduced gray matter volume in a right fusiform gyrus cluster (12 voxels, $p<.001$). There was a significant interaction with sex; the slope of the association between number allowed and gray matter volume in a left fusiform gyrus cluster was higher in women than in men (14 voxels, $p <.001$).

Sex differences in decisions in response to emotionally neutral faces were associated with gray matter volume in posterior face-sensitive regions, but not in the amygdala. Sex differences also were related to stress tolerance, with more stress tolerant men behaving comparably to women.

Key words: Face processing; Threat Assessment; Emotional Intelligence; VBM; Amygdala; Fusiform Gyrus

Can the Activation of Anterior Cingulate Predict the Emotional Suppression? A fMRI Study with Masked Faces

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Purpose: Emotional suppression (ES) plays a very important role in emotional self-regulation. People who demonstrate chronic ES often experience heightened anxiety or depression [1]. The role of ES in brain responses to emotional stimuli has been relatively unexplored. Currently, we aim to examine the relationship between the level of ES and the functional response to backward-masked facial expressions of various affects presented below the threshold of conscious perception [2].

Method: Sixty-three healthy adults (Age: 30±8; 33 males) were recruited. The Courtauld Emotional Control Scale (CECS) was used to assess the tendency to suppress negative emotions including anxiety, anger, and depression. Blood oxygen level-dependent fMRI was acquired at 3T with TR/TE/flip angle=3.0s/30ms/90degree, 60 images per slice. Three 3-minute sessions were acquired during backward masked presentations of anger/happy/fearful faces separately. Each session included a neutral Control face masked with a Neutral face, the stimulus (Angry/Happy/Fearful) masked with Neutral. Each trial was 1500ms, containing the 20ms-Target, 80ms-Mask, and 1400ms-Blank screen. Data were preprocessed and analyzed in SPM8. CECS-anger was the covariate of interest, controlling for age and gender. The threshold was set at FDR corrected $p<0.05$.

Result: There was a cluster located within the rostral anterior cingulate gyrus (rACC) (MNI: -2, 36, 8; 297 voxels; $p=0.006$, FDR corrected) showing a significant positive correlation between Anger/Control contrast and CECS-anger score. No significant correlation was found with Happy or Fearful faces.

Conclusion: Functional activation within rACC was positively correlated with higher CECS anger suppression during masked-anger perception, suggesting a role of rACC in emotional control.

Advantageous Decision Making Linked with Increased Gray Matter in the Ventromedial Prefrontal Cortex

Shreya Divatia, Lauren A. Demers, Lily Preer, Elizabeth Olson, Mareen Weber, William "Scott" Killgore

The Iowa Gambling Task (IGT) is a decision making task that involves adaptive and implicit learning. Advantageous performance requires individuals to determine which decks will allow them to avoid losses and maximize potential long-term reward. Behavioral and neuroimaging studies have shown that individuals with lesions to the ventromedial prefrontal cortex (VMPFC) often are impaired in these capacities and show lower scores on personality traits of conscientiousness and higher impulsiveness. However, little is known about the association between gray matter volume of the VMPFC and IGT performance in healthy individuals.

Fifty-three healthy right-handed adults (26 males) between the ages of 18 and 45 (M=30.8, SD=8.0) completed structural neuroimaging at 3T, the IGT, and the NEO PI-R (NEO), a multidimensional measure of personality. A voxel-based morphometric multiple regression analysis was conducted in VBM8 to explore the gray matter correlates of IGT hunch performance. Age and gender were used as covariates.

As hypothesized, the ability to implicitly learn which decks were advantageous more quickly was positively correlated with increased gray matter volume in the VMPFC (57 voxels, p=.03, FWE corrected). Furthermore, IGT performance was correlated with personality variables on the NEO PI-R, including lower scores on Conscientiousness (p=.022) and higher Openness to experience (p=.002). These results suggest that decision-making ability, particularly the capacity to forgo short-term gains in the service of long-term goals, was associated with greater gray matter volume in the VMPFC and a personality style of greater openness to experience.

Paranoid traits are related to deficits in complex social decision-making and reduced superior temporal sulcus volume

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Paranoid traits are often characterized by inaccurate interpretation of other people's motives. Individuals with high trait paranoia may have difficulties in interpersonal situations involving complex social judgments. Social decision-making has previously been related to superior temporal sulcus volume. To explore these relationships, 59 healthy adults (ages 18-45, *M*=30.66, *SD*=8.00; 30 males) underwent neuroimaging and completed the Personality Assessment Inventory (PAI) to measure paranoid tendencies, the Ekman 60 Faces Test (EFT) to control for potential differences in emotional face recognition abilities, and a novel task, the Facial Intuition Task (FIT), probing social complex decision-making. In FIT, participants decided whether computer-generated face stimuli were high or low on an unspecified trait determined by principal components analysis of previously rated traits. Trial-by-trial feedback was provided to help participants learn to make correct discriminations. Higher scores on the PAI paranoia scale correlated with lower average accuracy on FIT, even when controlling for basic emotional recognition scores on the EFT, $r(58)=-.33$, $p=.01$. Voxel-based morphometry, controlling for age and gender, was used to explore neural correlates of this finding, using a mask to restrict the analysis to the superior and middle temporal gyrus. Multiple regression analysis revealed a negative relationship between paranoia scores and a cluster of gray matter volume (k=69 voxels) in a region proximal to the superior temporal sulcus ($p<.001$, uncorrected; MNI:68,-43,6). Results suggest that paranoid traits are related to reduced gray matter volume in a region associated with social processing and poorer ability to integrate learning of subtle social cues into complex decision-making.

Gray Matter Volume in the Amygdala is Associated with Facial Assessments of Trustworthiness

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Structural abnormalities in the amygdala may interfere with processing of perceptions of threat. Evidence has shown that amygdala damage is associated with impaired recognition of fearful faces and decreased accuracy in determinations of facial trustworthiness. Some evidence suggests that greater amygdala volume is related to increased anxiety and fearfulness. The present study tested the hypothesis that amygdala volume would be inversely correlated with accuracy in assessing trustworthiness in faces.

Fifty-seven healthy individuals ages 18-45 (M=30.2, SD=30.2) completed a computerized facial assessment of trustworthiness task. In this task, participants were presented with a series of 100 pairs of computer-generated facial expressions varying along a continuum of rated trustworthiness and selected which face in the pair was "more trustworthy." Participants also underwent structural magnetic resonance imaging at 3.0 Tesla. Voxel-based morphometric random effects multiple regression whole-brain analyses were used to assess whether gray matter volume was related to trustworthiness accuracy ($p<.001$, non-stationary cluster extent corrected, cluster threshold $k \geq 87$). Reduced gray matter volume in the left amygdala was related to better performance on the trustworthiness task (89 voxels, T=4.33, MNI coordinates: x=-14, y=2, z=-24).

Findings support prior research on the role of amygdala structure in facial determinations of trustworthiness, but expand on this by showing that smaller gray matter volume is related to greater accuracy in determining trustworthiness. We speculate that larger gray matter volume in the amygdala may increase emotional interference, reducing the accuracy of determinations of trustworthiness.

Predisposition Towards Unhealthy Foods Linked with Increased Grey Matter in the Cerebellum

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The role of the cerebellum is often confined to sensory-motor function. However, growing literature suggests its implication in higher cognitive and emotional processes, particularly executive control and the salience network. Furthermore, several areas have been linked with addiction mechanisms and nicotine dependence. Yet relatively little has been done to examine cerebellum structure with regard to salience.

Thirty-seven healthy right-handed adults (20 males) between the ages of 18 and 45 (M=30.3, SD=8.8) underwent structural neuroimaging at 3T and completed a Food Recognition Task. Participants viewed a series of images in the scanner of healthy and unhealthy foods. Afterwards, subjects were asked to discern whether or not they had previously viewed each image. Participants also indicated how hungry they were at the moment using a 7-point scale. A voxel-based morphometric (VBM) multiple regression analysis was conducted to explore grey matter correlates of a predisposition to better remember unhealthy foods rather than healthy foods (Unhealthy Food Recognition Accuracy - Healthy Food Recognition Accuracy). Age and gender were used as covariates, along with total food recognition accuracy, subjective hunger, body mass index, and IQ.

Higher accuracy in the recognition of unhealthy foods compared with healthy foods was positively linked with increased grey matter volume in the cerebellum, particularly Crus I. Whole brain exploratory analyses indicated that grey matter volume within the Crus I was positively correlated with greater recall of unhealthy foods (1572 voxels, p=0.04, FWE corrected). In line with the literature, present results posit that the cerebellum may be implicated in salience detection.

Daytime sleepiness is associated with decreased integration of remote outcomes on the IGT

Elizabeth Olson[1,2], Mareen Weber[1,2], Olga Tkachenko[1], William D. S. Killgore[1,2];[1]McLean Hospital, [2]Harvard Medical School

Sleep loss is associated with deficits in basic aspects of attentional control such as processing speed and vigilance, in both healthy and clinical populations. Recently, deficits in higher-level aspects of executive functioning and decision-making also have been described. For instance, multiple research groups have reported impairment in Iowa Gambling Task (IGT) performance, both in healthy controls experiencing acute sleep deprivation and in individuals with sleep disorders. Traditional metrics of assessing IGT performance such as subtracting good minus bad deck choices do not discriminate between multiple underlying features that may contribute to changes in task performance. By using the expectancy valence model, it is possible to fit parameters to describe attention to gains versus losses, emphasis on recent versus remote outcomes, and behavioral randomness. In the current study, 32 participants ages 18 to 45 completed the IGT as well as questionnaires concerning sleep patterns and fatigue. Individuals who reported greater daytime sleepiness on the Epworth Sleepiness Scale (ESS) had higher values of the updating parameter, reflecting decreased integration of remote versus recent outcomes in decision-making, Spearman's r (30) = 0.390, p = 0.027. Results suggest that sleep loss affects IGT performance by shortening the time horizon over which decisions are integrated. This finding has important implications for healthy individuals experiencing sleep loss, as well as for clinical populations with sleep disorders.

Primary Keyword = EXECUTIVE PROCESSES: Other Secondary Keyword = EXECUTIVE PROCESSES: Monitoring & inhibitory control

Left-Hemifield Bias on Sad Chimeric Face Task Correlates with Interpersonal Emotional Intelligence

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Background: Chimeric face tasks measure left visual field bias, which has been associated with right hemisphere lateralization of face processing. As emotional intelligence (EI) has been linked to the right hemisphere, we hypothesized that the extent of left side bias on a chimeric faces task would be associated with increased EI.

Methods: 60 healthy, right-handed adults (31 women) completed the Bar-On Emotional Quotient Inventory (EQi), Wechsler Abbreviated Scale of Intelligence (WASI), and a chimeric face task. Chimeric faces, with one neutral side and one emotional side (happy or sad), were presented in mirror-imaged pairs. Subjects selected which face in each pair appeared happier (or sadder). A left side bias was calculated from the proportion of choices where the emotional expression was on the left side of the face.

Results: The EQi interpersonal composite scale was positively correlated with left side bias for sad chimeric faces, $r(58)=.27$, $p=.04$. At the subscale level, the relationship was significant for the empathy subscale, $r(58)=.26$, $p=.04$, and the interpersonal relationship subscale, $r(58)=.31$, $p=.02$. When controlling for WASI score, left side bias remained related to the interpersonal composite scale, $r(57)=.26$, $p=.048$, and the interpersonal relationship subscale, $r(57)=.30$, $p=.02$. There were no significant relationships between EQi scores and left side bias for happy chimeric faces.

Conclusion: Results suggest right hemisphere processing of sad facial expressions is linked to interpersonal components of EI. Greater lateralization of sad face processing may be related to increased ability to understand and relate to others.

Sleep Reduction and Functioning of the Emotion Regulation Circuitry

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BACKGROUND
Sleep plays a crucial role in emotional stability and is often disrupted in psychiatric disorders. Total sleep deprivation affects prefrontal metabolic activity, functional connectivity, emotional intelligence, coping, and symptoms of psychopathology in otherwise healthy people. We hypothesize that even normal variations in recent sleep duration may be associated with alterations in top-down modulation of emotion as indexed by prefrontal-amygdala functional connectivity, which in turn might be associated with increased depressive mood and anxiety sensitivity.

METHODS
Fifty-eight healthy adults (50% male; age 18-45) reported their hours of sleep from the preceding night and then underwent resting-state functional magnetic resonance imaging (fMRI). The following day, they completed the Anxiety Sensitivity Index (ASI) and Beck Depression Inventory (BDI). Functional connectivity (FC) between the ventromedial prefrontal cortex (vmPFC) and amygdala was analyzed and regressed against scores on the BDI and ASI.

RESULTS
Shorter sleep duration was associated with higher ASI ($p=.004$) and BDI scores ($p=.09$). Longer sleep duration was associated with inverse vmPFC-amygdala connectivity ($p<.001$), while short sleep was associated with positive coupling. Furthermore, as connectivity between these regions became more positively coupled, participants also showed higher ASI ($p=.02$) and BDI ($p=.03$) scores.

CONCLUSIONS
With fewer hours of sleep the night prior to neuroimaging, healthy adults showed a pattern of resting-state functional connectivity consistent with poorer top-down modulation of the amygdala, which was in turn associated with higher anxiety sensitivity and self-reported depressive symptoms the following day. Even a single night of insufficient sleep may be a risk factor for reduced emotion regulation capacity.

Emotional intelligence is differentially correlated with prefrontal cortical responses to backward masked fearful and angry faces

Anna Alkozei, Derek Pisner & William D.S. Killgore

Introduction

Emotional intelligence (EI) is the ability to understand and regulate emotions to facilitate problem solving. The neurobiological mechanisms behind this ability are unclear, however brain regions including the prefrontal cortex (PFC), amygdala, insula, and anterior cingulate cortex appear to be crucial. We hypothesized that in response to subtle social threat cues (masked fearful and angry faces), EI would correlate positively with activation within these brain regions.

Method

Fifty-four 18-45 year olds underwent blood oxygen dependent (BOLD) functional magnetic resonance imagining (fMRI) while viewing images of fearful and angry faces, each presented for 20ms and "masked" immediately by a neutral face for 100ms to prevent conscious visual perception. Measures of Trait and Ability EI were individually regressed against BOLD activation in response to the masked face tasks in SPM8.

Results

In response to masked fear, higher Ability EI correlated positively with bilateral activation in the PFC, and the left insula ($p<.005$, uncorrected). In contrast, in response to masked angry faces, increases in Ability EI correlated negatively with bilateral activation in the PFC, and the insula. No associations with Trait EI were found.

Conclusion

The results suggest different adaptive responses to nonconscious fearful versus angry faces in those with higher EI. Increased activation within the PFC in response to fearful faces suggests motivation to localize the source of danger and possible coping strategies. Contrarily, PFC deactivation in response to angry faces might provide a survival advantage by circumventing relatively slow top down regulatory processes to allow more rapid and automatic defense systems to respond unencumbered.

Looking for Evil Intent: Emotional intelligence and the use of socially relevant facial cues during an emotional decision making task

Anna Alkozei, Zachary Schwab, William D.S. Killgore

Introduction

Emotional intelligence (EI) is defined as the ability to understand, perceive and manage emotions. However, there is little research investigating how EI influences decision-making during emotionally difficult situations. We hypothesized that higher EI would correlate with greater utilization of socially relevant facial cues in emotional decision-making.

Method

Sixty-two 18-45 year olds completed a decision making task mimicking an airport security screening involving an established terrorist threat. Participants were presented with a series of facial photographs of white men and women and required to decide which ones to detain for further interrogation. The faces were previously rated for certain character traits (e.g., aggression) by independent judges. Participants also completed measures of Trait and Ability EI and cognitive intelligence (IQ).

Results

With higher Ability EI, participants were more likely to detain individuals based on higher negative traits (e.g., "aggression," $r=.27$, "meanness" $r=.31$) and lower positive traits (e.g., "trustworthy," $r=-.31$, "emotionally stable," $r=-.35$, all p's $<.05$). These associations were driven primarily by the Facilitating Branch of EI (i.e., the ability to generate and use emotion to facilitate decision making). On the other hand, no association between Trait EI or IQ and detained individuals' character traits was found.

Conclusion

Individuals with greater Ability EI, in particular greater capacity to use emotions to facilitate cognitive processes, were more likely to utilize more of the limited social information (i.e., facial features independently rated as indicative of underlying dispositional traits) when completing an emotional decision making task. These findings have implications for real-life situations involving similarly difficult emotional decision-making processes.

The Contribution of General Intelligence and Emotional Intelligence to the Ability to Appreciate Humor

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Objective:
The ability to appreciate humor is an extraordinarily complex capacity that requires numerous cognitive processes, rapid perceptual-cognitive switches, intelligence, and creativity. It remains unclear the extent to which capacities like traditional intellectual capacity (e.g., cognitive intelligence (IQ) contribute to this ability relative to emotional problem solving capacities such as emotional intelligence (EI)). The current study examined the relative contribution of IQ and EI on the ability to accurately identify humor.

Participants and Methods:
Sixty-one 18-45 year olds completed the Humor Appreciation Test (HAT), which presents a series of pairs of nearly identical fictional newspaper headlines or nonverbal cartoons. Participants were asked to select the more humorous item from each pair. Participants also completed measures of IQ, and ability and trait EI. Stepwise multiple linear regression analyses were used to predict total HAT accuracy, picture HAT accuracy, and headline HAT accuracy using IQ and EI as predictors.

Results:
IQ significantly predicted HAT total accuracy (R^2=0.35, $p<0.001$), headline accuracy (R^2=0.45, $p<0.001$) and picture accuracy (R^2=0.11, $p<0.010$). However, neither trait nor ability EI explained additional variance in any of the models.

Conclusions:
The ability to appreciate humor is strongly related to general IQ. However, once cognitive intelligence is accounted for, EI does not make any unique additional contribution to humor appreciation. Thus, the ability to appreciate humor appears to rely predominantly on basic intellectual capacities rather than on the attunement of emotional skills or affective status.

Sleep Onset Latency and Duration are Associated with Self-Perceived Invincibility

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Objective:

Insufficient sleep alters risk-taking propensity, but the direction and magnitude of the effect appear to differ according to a number of factors that remain poorly understood. One potential modifying influence on risk-taking is the extent to which a person believes that they will not be affected by the consequences of risky behavior—i.e., "invincibility." The present study explored the association between self-reported sleep parameter and self reported invincibility.

Participants and Methods:

Sixty-one healthy individuals (Males = 31, M age = 30, range = 18-45) completed a series of self-report measures including a brief questionnaire about typical sleep habits, and the Invincibility Beliefs Index (IBI). A bivariate correlational analysis was used to examine the relationships between sleep onset latency (SOL), sleep debt, and scores on the IBI.

Results:

Shorter SOL on weeknights was associated with higher self-perceived Total Invincibility scores ($r = -.292$, $p = .023$), and the Audacity subscale ($r = -.377$, $p = .003$). Furthermore, participants who typically slept less than their optimum preferred amount (i.e., typical sleep hours – hours of sleep necessary to feel best) tended to show lower scores on Total Invincibility ($r = -0.248$, $p = .045$) and Audacity ($r = -.355$, $p = .005$) of the IBI.

Conclusions:

Individuals who typically fall asleep faster and obtain more sleep tend to report greater self-perceptions of invincibility than those who receive less sleep. These findings are consistent with evidence that sleep loss reduces motivation and self-confidence. Thus, it is unlikely that prior findings of increased risk-taking during sleep loss stem from increased perceptions of invincibility and may be due more to altered decision-making or reduced alertness.

Visuospatial reasoning mediates the relationship between emotion recognition and emotional intelligence

Derek Pisner, Anna Alkozei, & William D.S. Killgore

Introduction

A major component of emotional intelligence (EI) is the ability to understand emotions, a skill that goes beyond mere emotion recognition, and includes knowledge of how emotions form an interrelated symbol set. We propose that greater visuospatial reasoning may allow individuals to better analyze how emotions interrelate, which may in turn explain higher EI. Therefore, we hypothesize that visuospatial reasoning ability mediates the relationship between emotion recognition ability and EI.

Method

Sixty-one 18-45 year olds (males=31, mean age = 30) completed: 1) the Ekman 60 Faces Task, which assesses emotion recognition accuracy in response to a series of emotional faces, and 2) the Design Organization test (DOT), a rapid visuospatial screening instrument similar to the ubiquitous 'Block Design' task, and 3) an ability based EI assessment.

Results

In a standard mediation analysis, we first used linear regression to confirm that: 1) emotion recognition significantly predicts EI (R^2=.22, p<.001); 2) emotion recognition significantly predicts visuospatial reasoning (R^2 = .22, p<.001); and 3) visuospatial reasoning, controlling for emotion recognition, significantly predicts EI (R^2=.28, p<.001). Finally, using a Sobel Test, we confirmed that visuospatial reasoning serves as a partial mediator between emotional recognition and EI, with a standardized indirect effect of .29 at a marginal level of significance p=.052.

Conclusion

The results imply that Emotional Intelligence involves more than just recognizing emotions—it also involves visuospatial reasoning. Future work may explore whether enhancing visuospatial ability might improve EI, as well as investigate fMRI activity of highly visuospatial individuals during EI tasks.

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Engaging in Meditation and Internet-Based Training as a Means of Enhancing Emotional Intelligence

John R. Vanuk, Andrew Fridman, Lauren A. Demers, Shreya Divatia, William D. Killgore

Objective : Emotional Intelligence (EI) refers to the ability to understand, perceive, and manage emotions in oneself and others. A three-week online training regimen, composed of six lessons, was created in an attempt to improve EI faculties. Since past research has shown a relationship between the practice of meditation and increased internal awareness, we hypothesized that meditators would show greater EI increases than non-meditators during EI training.

Participants and Methods: Sixty-two healthy adults (31 men), ranging in age from 18-50 years, were randomly assigned to one of two forms of web-based cognitive training; one employing non-EI or "external awareness" and the other employing EI or "internal awareness" training. Training differed in content, but was balanced on intellectual challenge, activities, and time requirements. As a measure of EI, the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) was administered at baseline and at completion of the program. Due to the importance of sleep in emotional functioning, sleep problems were also assessed.

Results : Individuals who practiced meditation reported higher occurrences of sleep problems than controls. After accounting for the variance associated with sleep problems, a 2 x 2 ANOVA showed a significant interaction between meditation group and improvement in EI scores ($p = .04$), with meditators showing greater benefit than non-meditators.

Conclusions : This relationship may be the result of individuals attempting to mitigate their sleep problems by engaging in meditation. Promoting introspective behaviors (i.e. engaging in meditation) may augment EI training in a manner that increases its effectiveness, regardless of whether individuals are experiencing the detrimental effects incurred by interruptions in sleep.

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Napping in Conjunction with Brief Internet-Based Training as a Means of Enhancing Emotional Intelligence

John R. Vanuk, Shreya Divatia, Lauren A. Demers, Sarah Markowski, Anna Alkozei, William D. Killgore

Objective : The ability to perceive, understand, and manage emotions in oneself and others is known as Emotional Intelligence (EI). Greater EI is thought to be associated with better coping and resilience, successful interpersonal relationships and increased work performance. We developed a three-week online training program to enhance EI skills. Because considerable evidence suggests that sleep plays a crucial role in emotional functioning and memory consolidation, we hypothesized that regular nappers would show greater improvement from the training than non-nappers.

Participants and Methods: Sixty-two healthy 18-50 year olds (31 men) were randomized to receive either a 6-lesson on-line EI-training program over a 3-week period, or a matched placebo training program with similar intellectual challenge and activities. Although napping was not controlled, twenty-eight participants (16 men) also reported voluntarily taking naps at least one or more times per week throughout the course of the training. As a measure of EI, participants completed the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) at baseline and after completion of the program.

Results : A 2 x 2 ANOVA yielded a significant interaction between EI training and napping conditions with regard to improvement in EI scores ($p=.006$). Whereas EI training was enhanced among nappers relative to non-nappers, the placebo condition was not effective at changing EI scores, regardless of napping.

Conclusions : The findings suggest that inclusion of napping for individuals in the online training program may provide an increase in the efficacy of EI training over the duration of the program. Napping may improve memory consolidation, emotional regulation, or cognitive performance during the training.

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Emotional Intelligence is Associated with Coordinated Resting State Activity Between Emotion Regulation and Interoceptive Experience Networks

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Emotional Intelligence (EI) reflects an individual's capacity to accurately perceive, understand, and regulate emotions, and to apply that information to facilitate thought and performance. Although EI has been shown to play an important role in mental health and success in academic, professional, and social realms, the neurocircuitry underlying this capacity remains poorly understood. We explored the relationship between regional functional connectivity and two alternative models of EI (i.e., Trait versus Ability models).

Fifty-four healthy, right-handed adults (28 women, 26 men), with an average age of 30.1 years (SD=7.5 years) completed standardized validated measures of Trait and Ability EI followed by resting state functional magnetic resonance imaging (rsfMRI). FSL and FSL MELODIC were used to implement an independent components analysis (ICA) with dual regression to investigate brain circuits (resting state networks, RSNs), whose activity was thought to be associated with greater EI capacities. All results are reported at $p<0.05$, FWE corrected.

Higher Ability EI (as opposed to Trait EI) was associated with stronger inverse correlations of the spontaneous FMRI signals from RSNs involved in affective regulation (e.g., prefrontal) with those involved in emotional responses and experiences (e.g., insula), and also between FMRI signals from emotionally responsive networks (e.g., insula) and those involved in self-reflective cognition (e.g., medial frontal/posterior cingulate).

Importantly, these findings suggest that stronger inverse correlations between signals from key intrinsic emotional regulation and interoceptive experience networks serve as a marker of higher emotional intelligence skills and abilities, perhaps reflecting greater capacity to regulate emotional responses through executive control processes.

Key words: Emotional Intelligence; Functional Connectivity; Independent Components Analysis; Emotional Regulation

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Enhancing Emotional Intelligence via Brief Internet-Based Training

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The capacity to understand emotions and use them to enhance cognition is known as Emotional Intelligence (EI). Evidence suggests that EI is important to success in many settings, and correlates with cortical volume and functional activation of the insula and ventromedial prefrontal cortex. Whether EI capacities are malleable and can be improved through focused training is currently a topic of debate. We developed and tested a brief, six-lesson (three week), on-line training course to enhance EI abilities based on established literature.

Sixty-two healthy adults (31 men), ranging in age from 18-50 years were randomly assigned to undergo one of two parallel online training programs, matched in terms of activities and intellectual challenge, but differing only in content (e.g., EI or "internal awareness" training versus non-EI or "external awareness" training).

The EI training program significantly enhanced Total EI scores on the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) relative to the placebo condition (p=.04). Further analysis of the four MSCEIT subscales revealed that the preliminary training program was effective at improving scores on the Perceiving Emotions (p=.04) and Facilitating Thought (p=.007) branches of EI, but not at improving the Understanding Emotions and Managing Emotions branches relative to controls.

Findings suggest that at least some EI capacities are malleable and can be improved through a brief internet-based intervention. With further development, such a program could be used to enhance critical emotional skills in a variety of settings. Whether these changes are associated with concomitant changes in brain structure and function remains to be explored.

Key words: Emotional Intelligence; Affect; Training

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Self-Perceived Invincibility is Associated with Sleep Onset Latency and Duration

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Introduction:

Insufficient sleep is associated with altered risk-taking tendencies, but the magnitude and direction of these effects seem to differ according to a number of factors that remain poorly understood. One potential modifying influence on risk-taking is their level of perceived "Invincibility", i.e., the degree to which an individual believes that he or she will not be affected by the consequences of high-risk behavior. Here, we examined the relationship between subjective sleep parameters and perceived invincibility.

Methods:

Sixty-one healthy individuals (Males = 31, M age = 30, range = 18-45) completed a number of self-report instruments including a brief questionnaire about typical sleep habits, and the Invincibility Beliefs Index (IBI), a validated measure that measures beliefs about the probability of various behavioral consequences during risk-taking. A bivariate correlational analysis was used to examine the relationships between scores on the IBI, sleep onset latency (SOL), and sleep debt.

Results:

Shorter SOL on weeknights was related to higher self-perceived total Invincibility scores ($r = -.292$, $p = .023$). Furthermore, participants who typically slept less than their optimum preferred amount (i.e., typical sleep hours – hours of sleep necessary to feel best) tended to show lower scores on total Invincibility ($r = -0.248$, $p = .045$). These relationships were found to be driven by the Audacity (i.e., boldness) subscale of the IBI for both participants who slept less than their optimum preferred amount ($r = -.355$, $p = .005$) and those with shorter SOL ($r = -.377$, $p = .003$).

Conclusion:

Individuals who typically obtain more sleep and fall asleep faster tend to report greater self-perceptions of Invincibility than those who receive less sleep. These findings are consistent with evidence that sleep loss reduces motivation and self-confidence. Thus, it is doubtful that prior findings of increased risk-taking during sleep loss originate from increased self-perceptions of Invincibility and may be due more to altered decision-making, impaired inhibition, or limited information processing.

Support:

Daytime Sleepiness is Associated with Altered Thalamocortical Connectivity

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Alertness, attention, neural information transfer and regulation of sleep states rely critically on thalamocortical circuitry. Some evidence suggests that total sleep deprivation disrupts connectivity of this circuitry, in turn contributing to deficits in alertness, vigilance, and information processing. Whether such altered connectivity is associated with daytime sleepiness under non-sleep deprived conditions is unknown. Accordingly, it was hypothesized that greater daytime sleepiness would be associated with lower thalamocortical resting state functional connectivity.

Sixty healthy adults (30 male, 30 female; M age: 30.4 years), completed the Epworth Sleepiness Scale (ESS) and underwent a six-minute resting state functional connectivity neuroimaging scan at 3T. Regions of interest for the thalamus (bilaterally) and parcellated regions of the cortex were interrogated using the CONN toolbox. Specifically, we examined the functional connectivity between the thalamus and other regions of the cortex ($p < .05$, FDR corrected for height and cluster threshold).

Greater sleepiness was associated with significant inverse connectivity between the right thalamus and widespread cortical regions, most prominently including the ventral prefrontal cortex, motor, and sensory regions. For the left thalamus, daytime sleepiness was only associated with four small clusters of cortical sensory and motor regions that were inversely connected with thalamic responses.

The present findings suggest that daytime sleepiness is associated with altered thalamocortical connectivity during rested wakefulness, potentially reflecting the disengagement of sensory and motor processing from the stream of consciousness. Future work may examine whether these patterns of connectivity may be affected by wake promoting agents or other treatments that minimize sleepiness.

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Emotional Intelligence and Subliminal Presentations of Social Threat

Anna Alkozei, Derek Pisner, Scott L Rauch, & William D.S. Killgore

Introduction

The ability to rapidly process emotional facial cues is crucial for successful social interactions. Altered brain responses to subliminal (i.e., backward masked) facial cues have been associated with several forms of psychopathology. However, even healthy individuals vary widely in their capacity to regulate and understand their own emotions and those of others, a construct known as Emotional Intelligence (EI). The aim of this study was to identify the brain networks associated with EI during masked presentations of fearful (withdrawal threat) and angry (approach threat) faces.

Method

Fifty-four 18-45 year olds (50% females) underwent blood oxygen dependent (BOLD) functional magnetic resonance imagining (fMRI) while viewing images of fearful and angry faces, each presented for 20ms and "masked" immediately by a neutral face for 100ms, minimizing or preventing explicit visual perception. Participants also completed measures of EI.

Results

EI was only associated with prefrontal cortex (PFC) responses to approach cues of social threat. For masked angry faces higher EI correlated negatively with bilateral activation in the dorsolateral superior frontal gyrus and the inferior frontal gyrus, whereas no association between EI and brain activation in response to masked fearful faces was found.

Conclusion

Higher EI was correlated with reduced activation within the dorsolateral PFC in response to subliminal presentations of approach cues of social threat, in particular anger. Higher EI may be associated with automatic adaptive responses that facilitate rapid decision-making in response to social cues indicative of potential threat. EI may be an important dimension for understanding the neurocircuitry underlying some forms of psychopathology.

Trait Emotional Intelligence is Associated with Greater Resting State Functional Connectivity within the Default Mode and Task Positive Networks

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Objective: Emotional intelligence (EI) is defined as the ability to accurately perceive, understand, regulate, and utilize emotion in oneself and others; to facilitate the ability to solve emotionally driven problems. Some evidence suggests that healthy emotional capacities may involve the ability to shift flexibly between internal and external focus. Accordingly, we hypothesized that an individual's self reported level of EI would correlate with inverse connectivity relationships between areas related to internal focus and self-reflective processing (Default Mode Network; DMN) and external environmental focus (Parietal Task Positive Network; P-TPN), as mediated by the posterior cingulate cortex (PCC).

Methods: Sixty healthy adults (50% female; Mean: 30.4 years) completed the Bar-On Emotional Quotient Inventory and a six-minute resting state functional magnetic resonance imaging (fMRI) scan at 3T. Bilateral regions of interest were placed in the PCC, along with individual regions of interest placed in the ventromedial prefrontal cortex (vmPFC) and regions of the parietal cortex as defined by the Automated Anatomical Labeling Atlas. Functional connectivity was analyzed utilizing the CONN toolbox and SPM12.

Results: EI correlated positively with increased functional connectivity between the PCC and left vmPFC (i.e., DMN), but was associated with anticorrelated functional connectivity between the PCC and several parietal regions (i.e., P-TPN).

Conclusions: Self-rated scores on one of the most widely used Trait EI measures were associated with inverse resting state connectivity between DMN and TPN regions. The results suggest that one component of higher EI may involve the flexibility of transition between cognitive states involving internal self-reflective focus and engagement with external stimuli.

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Engaging in Meditation and Internet-Based Training as a Means of Enhancing Emotional Intelligence

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Background: Emotional Intelligence (EI) comprises a set of skills to effectively appraise, express, and regulate emotions in oneself and others. Recent research efforts have focused on methods for enhancing EI skills. One key component of EI is "internal awareness" which can be facilitated through the practice of medication. Here, we tested the effect of meditation among participants undergoing an online training regimen to enhance EI faculties.

Methods: Sixty-two healthy volunteers (31 men, ages 18-50 years) completed a three-week, web-based cognitive training, composed of six lessons. Participants were randomly assigned to either an EI, "internal awareness," training or a non-EI, "external awareness," training. EI was measured by the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) at baseline and after completion of the program. Participants also completed questionnaires about other aspects of health, including meditation practice and sleep problems.

Results: Unexpectedly, compared to controls, we found that individuals that self-engaged in mediation also had increased instances of sleep problems. Thus, self-reported sleep difficulties were included as a covariate. A 2 x 2 ANCOVA showed a significant interaction between mediation and EI training ($p=0.04$), with meditators obtaining greater benefit from the training than non-meditators.

Conclusions: The present findings suggest that the benefits of EI training may be more effectively gleaned when conducted in parallel with practices that enhance introspection, such as meditation. Future work may focus on evaluating combined training programs that experimentally manipulate introspection promoting behaviors as another intervention for improving EI, even among individuals suffering from sleep-based problems.

Abstract submitted for presentation at the 70th Annual Scientific Meeting of the Society of Biological Psychiatry, Toronto, Ontario, CA, May 14-16, 2015.

Trait Emotional Suppression is Associated with Decreased Activation of the Insula and Thalamus in response to Masked Angry Faces

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Introduction:

Prior research has shown that Emotional suppression (ES) is associated with increased activation of the rostral anterior cingulate cortex (rACC) during subliminal presentations of angry faces, suggestive of an increased ability to regulate emotions. Further, individuals exhibiting trait ES show increased anterior insula volume. Presently, we hypothesized that higher trait ES would correlate with greater functional deactivation in the insula, a region associated with the visceral experience of emotion, and the thalamus, a region critical to information transfer, in response to subliminal presentations of anger.

Methods:

53 healthy participants (27 males) aged 20-43 years completed the Courtauld Emotional Control Scale (CECS), a self-report measure of emotional suppression. Participants also underwent functional magnetic resonance imagining (fMRI) while viewing backward masked images of angry faces (minimizing or preventing explicit visual perception). Data were analyzed in SPM12, with bilateral regions of interest set for the insular cortex and thalamus based on the Automated Anatomical Labeling Atlas.

Results:

In response to masked angry faces, CECS scores correlated with significant ($p<.10$ FDR corrected; $k \geq 16$) deactivation within the right insula and the right and left thalamus.

Conclusion:

Consistent with our hypothesis, those with higher ES exhibited greater deactivation in the insula and thalamus to masked angry faces. In the context of prior work suggesting that ES is associated with increased rACC activation, our findings suggest that greater ES might involve top down-regulation of the interoceptive perception systems, perhaps to reduce emotional experience. Future research is necessary to establish the possible impact of this neurocircuitry in emotional disorders.

The trait of neuroticism predicts neurocognitive performance in healthy individuals

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Background:
Neuroticism predicts cognitive performance in healthy individuals, but certain aspects of cognitive performance and neuroticism are poorly understood. Some potential aspects of cognitive performance that neuroticism could have an influence on are memory recall, language, and attention. The present study examined the relationship between Neuroticism and different facets of neurocognition.

Methods:
Forty-six healthy individuals (males=23, M age = 25, range = 20-43) completed the NEO Personality Inventory, Revised (NEO PI-R), followed by the Repeatable Battery for the Assessment of Neuropsychological Status Update (RBANS) at baseline. Stepwise and backward multiple regression analyses were used to examine the relationships between Neuroticism and neurocognitive performance on the RBANS.

Results:
Neuroticism was found to be a significant predictor for increased total RBANS scores, accounting for 23% of the variance in the total RBANS score (R^2 =.231, p=.001). Furthermore, higher scores on neuroticism were also found to be a significant predictor for Immediate memory (R^2 =.274, p < .001), Language (R^2 =.179, p=.004), and Attention (R^2=.093, p=.042).

Conclusions:
Higher Neuroticism was found to predict better cognitive performance, in line with previous research. This might have important clinical applications for individuals experiencing high levels of neuroticism, often seen in depression and anxiety.

Emotional Intelligence Correlates with Coordinated Resting State Activity Between Brain Networks involved in Emotion Regulation and Interoceptive Experience

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The capacity to accurately perceive, understand, and regulate emotions, and to apply that information to facilitate thought and performance is known as Emotional Intelligence (EI). Although research suggests that EI plays an important role in mental health and success in academic, professional, and social realms, the underlying neurocircuitry contributing to this capacity remains poorly understood. Here, we explored the regional functional connectivity underlying two leading alternative models of EI.

Healthy, right-handed adults (n = 54, 26 men), with a mean age of 30.1 years (SD=7.5 years) completed standardized validated measures of the Trait and Ability EI models and then underwent resting state functional magnetic resonance imaging (rsfMRI). FSL MELODIC was used to implement an independent components analysis (ICA) with dual regression to investigate resting state networks (RSNs), whose activity was thought to be associated with greater EI capacities. Results are reported at $p<0.05$ (FWE corrected).

Higher scores for Ability EI (as opposed to Trait EI) were associated with stronger inverse correlations of the spontaneous FMRI signals from RSNs involved in affective regulation (e.g., prefrontal) with those involved in somatic emotional responses (e.g., insula), and also correlated with the strength of connectivity between FMRI signals from these emotionally responsive networks and those involved in self-reflective cognition (e.g., medial frontal/posterior cingulate).

These findings suggest that stronger inverse correlations between signals from key intrinsic emotional regulation and interoceptive experience networks serve as a marker of higher emotional intelligence skills and abilities, perhaps reflecting greater capacity to regulate emotional responses through executive control processes.

Key words: Emotional Intelligence; Functional Connectivity; Independent Components Analysis; Emotional Regulation

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Boosting Emotional Intelligence with a Brief Internet-Based Program

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Emotional Intelligence (EI) involves the ability to perceive and understand emotional information and reason effectively about emotions. Research suggests that EI is important to success in many aspects of life and correlates with mental health. Despite the large popular interest in EI, there is very little data from well-designed placebo-controlled clinical trials regarding the malleability and trainability of EI capacities. For the present study, we developed and tested a brief, six-lesson (three week), on-line training course to enhance EI abilities.

Healthy adults (n=62; 31 men), ranging in age from 18-50 years were randomly assigned to one of two matched online training programs. The programs were closely matched with regard to activities and intellectual difficulty, but differed only in content (e.g., EI or "internal awareness" training versus non-EI or "external awareness" training).

Total EI scores on the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) were significantly enhanced by the active EI Training relative to the placebo condition (p=.04). Follow-on analysis of the four MSCEIT subscales showed that the preliminary training program significantly improved scores on the Perceiving Emotions (p=.04) and Facilitating Thought (p=.007) subscales, but not at improving the Understanding Emotions and Managing Emotions branches compared to controls.

We found that some EI capacities, particularly those involving the experiential aspects of emotional functioning were malleable and were improved through a brief internet-based intervention. With additional development, such a program could be used to enhance critical emotional skills and mental health in a variety of vocational and academic settings.

Key words: Emotional Intelligence; Affect; Training

Funding: W81XWH-09-1-0730

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Daytime Sleepiness is Associated with Altered Thalamocortical Connectivity

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Alertness, attention, neural information transfer and regulation of sleep states rely critically on thalamocortical circuitry. Some evidence suggests that total sleep deprivation disrupts connectivity of this circuitry, in turn contributing to deficits in alertness, vigilance, and information processing. Whether such altered connectivity is associated with daytime sleepiness under non-sleep deprived conditions is unknown. Accordingly, it was hypothesized that greater daytime sleepiness would be associated with lower thalamocortical resting state functional connectivity.

Sixty healthy adults (30 male, 30 female; M age: 30.4 years), completed the Epworth Sleepiness Scale (ESS) and underwent a six-minute resting state functional connectivity neuroimaging scan at 3T. Regions of interest for the thalamus (bilaterally) and parcellated regions of the cortex were interrogated using the CONN toolbox. Specifically, we examined the functional connectivity between the thalamus and other regions of the cortex ($p < .05$, FDR corrected for height and cluster threshold).

Greater sleepiness was associated with significant inverse connectivity between the right thalamus and widespread cortical regions, most prominently including the ventral prefrontal cortex, motor, and sensory regions. For the left thalamus, daytime sleepiness was only associated with four small clusters of cortical sensory and motor regions that were inversely connected with thalamic responses.

The present findings suggest that daytime sleepiness is associated with altered thalamocortical connectivity during rested wakefulness, potentially reflecting the disengagement of sensory and motor processing from the stream of consciousness. Future work may examine whether these patterns of connectivity may be affected by wake promoting agents or other treatments that minimize sleepiness.

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Gray matter correlates of Trait and Ability models of emotional intelligence

William D. S. Killgore, Mareen Weber, Zachary J. Schwab, Sophie R. DelDonno, Maia Kipman, Melissa R. Weiner and Scott L. Rauch

Research suggests that emotional intelligence capacities may be related to the functional integrity of the corticolimbic regions including the ventromedial prefrontal cortex, insula, and amygdala. No study has yet examined regional brain volumes in relation to the two dominant models of emotional intelligence: the Ability model, which posits a set of specific demonstrable capabilities for solving emotional problems, and the Trait model, which proposes a set of stable emotional competencies that can be assessed through subjectively rated self-report scales. In 36 healthy participants, we correlated scores on the Mayer–Salovey–Caruso Emotional Intelligence Test (an Ability measure) and the Bar-On Emotional Quotient Inventory (a Trait measure) with regional brain volumes using voxel-based morphometry. Total Mayer–Salovey–Caruso Emotional Intelligence Test scores were positively correlated with the left insula grey matter volume. The Strategic emotional intelligence subscale correlated positively with the left ventromedial prefrontal cortex and insular volume. In contrast, for the Bar-On Emotional Quotient Inventory, Stress Management scores correlated positively with the bilateral ventromedial prefrontal cortex volume. Amygdala volumes were unrelated to emotional intelligence measures. Findings support the role of the ventromedial prefrontal cortex and insula as key nodes in the emotional intelligence circuitry. *NeuroReport* 23:551–555 © 2012 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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Keywords: amygdala, emotional intelligence, insula, somatic marker hypothesis, ventromedial prefrontal cortex, voxel-based morphometry

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Introduction

Although some people cope skillfully with adversity and seem resilient in the face of challenging situations, others have greater difficulty managing their emotions and are prone to poor decision-making under stress. Together, these social, affective, and coping capacities have been described as emotional intelligence. The construct of emotional intelligence comprises a number of traits and competencies that involve attunement to multiple levels of emotional information and the ability to flexibly regulate and use emotions in an adaptive manner to facilitate effective judgment and decision-making, foster relationships, and achieve goals [1–3]. The exact nature of these capacities continues to be debated, with some researchers describing emotional intelligence in terms of trait-like competencies that can be measured through self-report (i.e. Trait models) [4], whereas others have argued that emotional intelligence is only validly conceptualized and measured in terms of demonstrable emotional reasoning abilities, more akin to traditional cognitive approaches to intelligence (i.e. Ability models) [2].

The integration of emotional information with other aspects of cognition appears to rely on the interaction of several key brain regions, particularly the medial orbitofrontal cortex, ventromedial prefrontal cortex, insular cortex, and amygdala [5]. Together, these constitute the Somatic Marker Circuitry [5], which is hypothesized to integrate emotional states, previous learning, and conscious cognition to guide decision-making toward advantageous outcomes [6]. The ventromedial prefrontal cortex appears to be particularly crucial, given its roles in emotional regulation [7], judgment, and advantageous decision-making [8]. The insular cortex is important for processing the interoceptive cues involved in emotion [9,10], and integrating them with ongoing cognition [11]. Despite the burgeoning literature on various social, cognitive, and interpersonal aspects of emotional intelligence and social cognition, there is only limited research into the neurobiological basis of emotional intelligence. Functional neuroimaging has supported the role of the medial prefrontal cortex in emotional intelligence [12,13], and two published studies have examined the correlation between self-reported perceptions of emotional intelligence traits with voxel-wise gray matter volume in the brain [14,15]. Although both studies implicated gray matter volume in the ventromedial prefrontal cortex, the two studies were discordant in the direction of the reported association, and both used only self-report trait measures of emotional intelligence. To our knowledge, no study has yet examined the relationship between brain structure and ability-based measures of emotional intelligence. In the present study, we used voxel-based morphometry to evaluate the relationship between gray matter volume of the Somatic Marker Circuitry and emotional intelligence as measured by

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both the Trait and Ability models. We hypothesized that higher emotional intelligence as assessed by both models, particularly regarding facets involving emotional control, would be associated with greater gray matter volume of the ventromedial prefrontal cortex, insula, and amygdala, as key regions of the Somatic Marker Circuitry.

Methods

Participants

Thirty-six right-handed, primary English-speaking adults (mean age 30.0 ± 8.9, range 18–45; 20 men) were recruited from the Boston metropolitan area and received payment for their time. Participants had no history of neurological, psychiatric, or substance use disorders (including alcohol and illicit drugs). This research was approved by the McLean Hospital Institutional Review Board. All participants provided written informed consent.

Materials and procedure

Each participant completed two validated and commercially available tests that measure alternative models of emotional intelligence. The Ability model defines emotional intelligence in terms of measurable capacities to reason about and solve emotional problems in a manner similar to traditional intelligence tests [2]. This can be contrasted with Trait (or Mixed) models of emotional intelligence, which view these capacities more loosely as personal competencies that reflect an individual's potential to cope with environmental demands [4], and which can be assessed by self-report inventories rather than through objective problem solving tests. As an index of Ability emotional intelligence, participants completed the Mayer–Salovey–Caruso Emotional Intelligence Test (MSCEIT) [2]. The MSCEIT uses 141 computer-administered items to measure the ability to identify emotions, to understand what causes different emotions, and to utilize emotions to facilitate behavior and achieve goals. Participants rate various stimuli such as abstract pictures, music, and faces on several emotional dimensions, and answer questions about how various moods and emotions affect thinking. Other items ask questions about how various emotions blend together or how one emotion (e.g. anger) can transition into another (e.g. rage). Participants also rate various strategies for regulating emotions in different situations and the effectiveness of such strategies for achieving goals. The MSCEIT yields a Total emotional intelligence score and two Area scores, Experiential emotional intelligence and Strategic emotional intelligence. Experiential emotional intelligence reflects the ability to perceive emotions in oneself, other persons, and various inanimate stimuli, and to utilize emotional information in facilitating cognition. Strategic emotional intelligence reflects the ability to understand emotions and their evolution in oneself and others, and to manage them in an efficient and effective manner. Raw scores were converted to scaled scores on the basis of the general normative group, without adjustment for sex.

As a measure of Trait emotional intelligence, participants completed the Bar-On Emotional Quotient Inventory (EQ-i) [4]. This 125-item self-report inventory yields a Total Emotional Quotient and five composite scores (i.e. Interpersonal, Intrapersonal, Adaptability, Stress Management, General Mood). The inventory includes items such as 'I'm aware of the way I feel' and 'I don't hold up well under stress', which must be answered on a five-point Likert scale ranging from 'Very Seldom or Not True of Me' to 'Very Often True of Me or True of Me.' The Interpersonal scale provides a measure of perceived empathy and interpersonal skills, whereas the Intrapersonal scale reflects self-perceived awareness of one's own emotions and self-regard. The Adaptability scale reflects the perceived ability to objectively analyze problematic situations, to solve them, and to adapt to changing environments. Stress Management reflects tolerance of and perceived self-control during stressful or demanding situations. The General Mood scale reflects self-reported positive thinking and overall contentedness with personal life.

Magnetic resonance imaging parameters

On the same day as emotional intelligence testing, structural MRI was conducted at 3.0 T (Siemens Tim Trio, Erlangen, Germany) using a 12-channel head coil. A T1-weighted 3D MPRAGE sequence (TR/TE/flip angle = 2.1 s/2.25 ms/12°) was used to obtain 128 sagittal slices (256 × 256 matrix) with a slice thickness of 1.33 mm and a voxel size of 1 × 1 × 1.33 mm.

Voxel-based morphometry

Voxel-based morphometric analysis was performed with the VBM8 toolbox (*http://dbm.neuro.uni-jena.de/vbm.html*) in SPM8 (Wellcome Department of Imaging Neuroscience Group, London, UK; *http://www.fil.ion.ucl.ac.uk/spm*). Preprocessing used the VBM8 default settings for modulated voxel-based morphometry (i.e. gray matter volume was corrected for total brain volume). T1-weighted structural images were DARTEL-normalized to Montreal Neurological Institute space and then segmented into gray matter, white matter, and cerebrospinal fluid using a fully automated algorithm. Normalized gray matter images were then smoothed with an 8-mm full-width at half-maximum Gaussian kernel.

Statistical analysis

Normalized smoothed gray matter images were entered into a series of random effects multiple regression analyses in SPM8. We conducted a search territory region-of-interest analysis of the Somatic Marker Circuitry (i.e. bilateral insula, amygdala, ventromedial prefrontal cortex) using an anatomical mask on the basis of the Automated Anatomical Labeling Atlas [16], as implemented in the Wake Forest University PickAtlas Utility for SPM [17]. Within the region of interest, separate regression analyses were used to predict gray matter volume from MSCEIT and EQ-i total and subscale scores. All analyses used an

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uncorrected height threshold ($P < 0.001$) and an extent threshold established empirically as the statistically expected number of voxels per cluster in each analysis (i.e. 79) according to the SPM8 output. Age and sex served as nuisance covariates in all analyses.

Results

Table 1 shows descriptive statistics for the Ability and Trait measures of emotional intelligence. Both were significantly related to gray matter volume within several regions of the Somatic Marker Circuitry (Table 2). Total MSCEIT emotional intelligence was positively correlated with gray matter volume of the left posterior insula (Fig. 1a). When evaluated by subscale, only the Strategic emotional intelligence Area scale of the MSCEIT correlated positively with Somatic Marker Circuitry gray matter, including the bilateral medial prefrontal cortex (i.e. gyrus rectus and the orbital region of the medial frontal gyrus; Fig. 1b), left posterior insula (Fig. 1c), and left anterior insula/ventrolateral prefrontal cortex, including the inferior frontal gyrus (Fig. 1d). Experiential emotional intelligence was not correlated with gray matter volume. For the EQ-i, Total score was unrelated to gray matter volume. Of the subscales, however, Stress Management was significantly positively correlated with gray matter volume within the right (gyrus rectus; Fig. 1e) and the left ventromedial prefrontal cortex (orbital region of the medial frontal gyrus between the anterior rostral and paracingulate sulci; Fig. 1f). None of the other EQ-i subscales were significantly correlated with gray matter volume in the Somatic Marker Circuitry.

Table 1 Mean, SD and range of Ability and Trait measures of Emotional Intelligence

EI measure	Mean±SD	Range
MSCEIT		
Total	102.2±12.74	71–124
Experiential EI	105.1±15.73	70–143
Strategic EI	98.3±10.06	80–120
EQ-i		
Total EQ	104.1±14.12	70–126
Adaptability EQ	103.1±12.61	75–126
General Mood EQ	104.6±11.91	68–121
Interpersonal EQ	102.4±15.31	59–125
Intrapersonal EQ	104.7±15.61	59–126
Stress Management EQ	104.4±12.29	77–125

EI, Emotional Intelligence; EQ, Emotional Quotient; EQ-i, Bar-On Emotional Quotient Inventory; MSCEIT, Mayer-Salovey-Caruso Emotional Intelligence Test.

Discussion

Specific facets of the Ability and Trait models of emotional intelligence correlated positively with gray matter volume in regions of the Somatic Marker Circuitry. Total scores on the MSCEIT, an Ability measure of emotional intelligence, correlated positively with gray matter volume within the left posterior insula, a region implicated in somatic [9] and emotional processing [18], and which may be particularly activated by focused attention toward one's own emotional state [19]. As an Ability measure, the MSCEIT assesses emotional intelligence in terms of measurable performance capacities involved in reasoning about and solving emotional problems, which are divided into two broad areas or domains of functioning. Of the two MSCEIT Area scores, Experiential and Strategic emotional intelligence, only Strategic emotional intelligence was significantly related to gray matter volume in the Somatic Marker Circuitry regions. Strategic emotional intelligence, which involves the ability to understand the meaning of emotional information (e.g. understanding how various emotions are related and what factors affect emotional change) and the ability to regulate emotions in oneself and others (e.g. modulating emotional states to achieve particular goals) [2], was associated with greater gray matter volume in the ventromedial prefrontal cortex, anterior insula, and posterior insula. This is consistent with previous findings suggesting that the ventromedial prefrontal cortex is important for emotional control [7,20], and evidence suggesting that damage to this region may lead to deficits in regulating emotion, poor judgment, and impaired decision-making [8]. Recent functional neuroimaging findings suggest that the ventromedial prefrontal cortex plays a prominent role in resilience against stress and trauma [21], whereas morphometric findings have shown that coping with stress early in life is associated with expansion of ventromedial prefrontal cortex volume in nonhuman primates [22]. Experiential emotional intelligence, which involves perceptual awareness of emotional signals and the ability to use emotions to facilitate thought [2], was unrelated to gray matter volume

Table 2 Gray matter correlates of Emotional Intelligence

			MNI coordinates				
EI measure	Region	Cluster size	*x*	*y*	*z*	*T*	*r*
MSCEIT (Ability Emotional Intelligence)							
Total EI	Posterior insula	159	−32	−19	12	4.66	0.56
Strategic EI	VMPFC	584	−4	39	−15	4.69	0.63
		123	10	58	−14	4.47	0.56
	Posterior insula	111	−32	−19	13	4.38	0.56
	Anterior insula/ventrolateral PFC	225	−38	21	−2	4.31	0.60
EQ-I (Trait Emotional Intelligence)							
Stress Management EQ	VMPFC	185	4	24	−24	4.47	0.56
		110	−8	51	−8	3.73	0.48

EI, Emotional Intelligence; EQ, Emotional Quotient; EQ-i, Bar-On Emotional Quotient Inventory; MNI, Montreal Neurological Institute; MSCEIT, Mayer-Salovey-Caruso Emotional Intelligence Test; PFC, prefrontal cortex; VMPFC, ventromedial prefrontal cortex.

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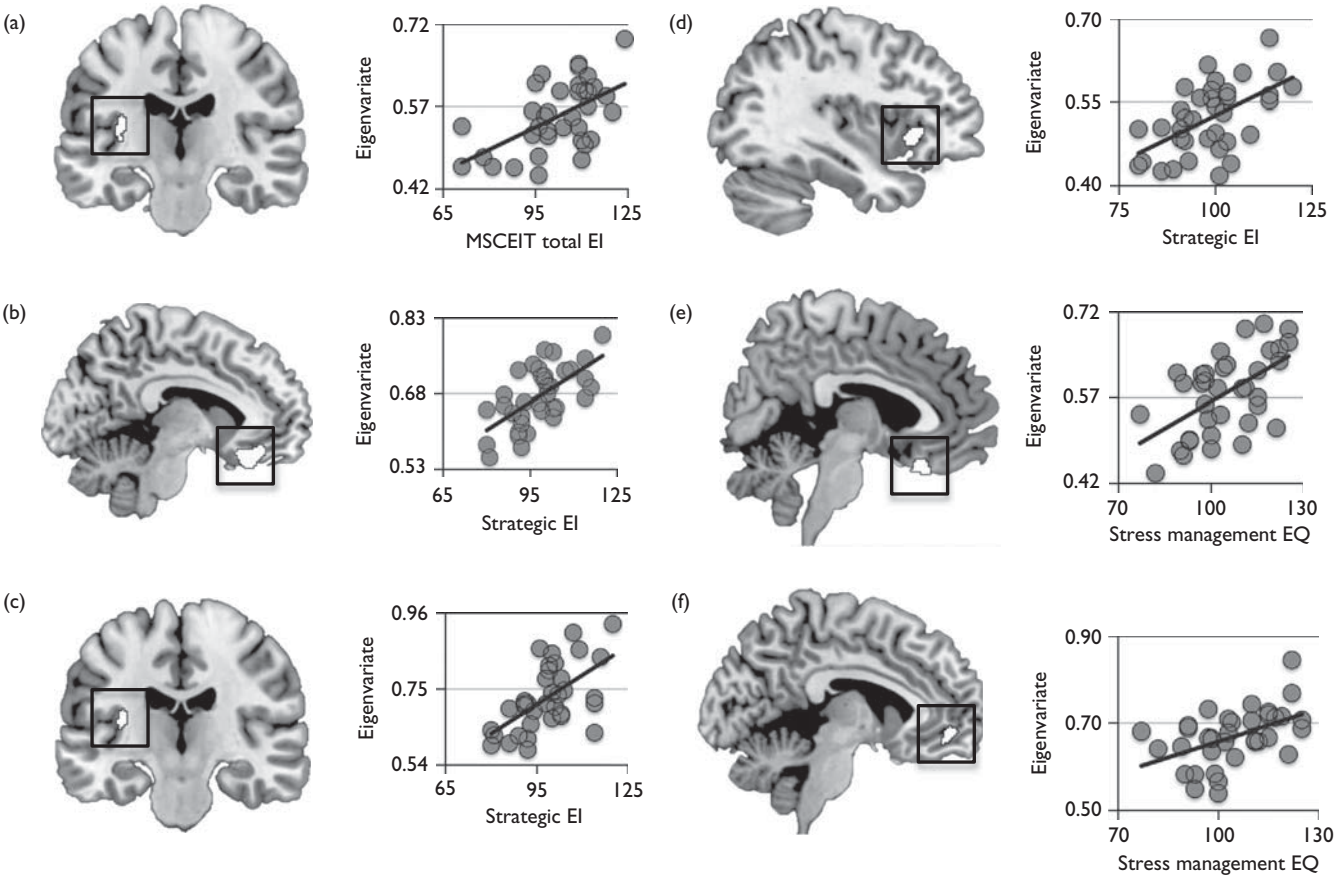
Fig. 1



Brain regions with significant positive correlations between gray matter volume and Emotional Intelligence measures superimposed on a single-subject T1-weighted structural image and corresponding scatterplots. (a) Coronal view of the left posterior insula gray matter volume that correlated with the total MSCEIT emotional intelligence. (b) Sagittal view of the left ventromedial prefrontal cortex (gyrus rectus and medial frontal gyrus, orbital region) gray matter volume that correlated with Strategic emotional intelligence. (c) Coronal view of the left posterior insula gray matter volume that correlated with Strategic emotional intelligence. (d) Sagittal view of the left anterior insula/ventrolateral (inferior frontal gyrus) prefrontal cortex gray matter volume that correlated with Strategic emotional intelligence. (e) Coronal view of the right ventromedial prefrontal cortex (gyrus rectus) gray matter volume that correlated with Stress Management EQ. (f) Sagittal view of the left ventromedial prefrontal cortex (medial frontal gyrus, orbital region) gray matter volume that correlated with Stress Management EQ. EI, Emotional Intelligence; EQ, Emotional Quotient; MSCEIT, Mayer-Salovey-Caruso Emotional Intelligence Test.

in any of the hypothesized regions. This suggests that while the volume of structures within the hypothesized neurocircuitry was associated with reasoning about emotions, it was not related to perception of emotional information or using emotions to facilitate thought. Notably, while the amygdala has also been implicated as an important node of the Somatic Marker Circuitry, volumetric measures of this structure were not significantly related to Ability emotional intelligence measures.

We also examined gray matter volume correlates of Trait emotional intelligence on the EQ-i. Total EQ-i and four of its five subscale scores, including the Interpersonal, Intrapersonal, Adaptability, and General Mood scores, were not significantly correlated with gray matter volume in the hypothesized regions. Higher scores on the Stress Management subscale were, however, significantly correlated with greater gray matter volume within the bilateral ventromedial prefrontal cortex. Persons with high Stress Management scores are reported to cope with stress in an active and resourceful manner while maintaining a positive, composed, and self-confident outlook [4]. This ability to maintain emotional control and demonstrate positive coping and resilience in the face of adversity was reported to a greater degree among those with elevated gray matter volume of ventromedial prefrontal cortex regions, which have previously been implicated in emotional control [7,20,23]. As with the Ability measure of emotional intelligence, amygdala volume was not significantly correlated with Trait emotional intelligence scores.

To our knowledge, this is the first study to examine the brain morphometric correlates of the two dominant models of emotional intelligence by employing widely used, standardized, well-validated, and commercially available assessment instruments. Findings converge on the key

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role of the ventromedial prefrontal cortex in emotional intelligence, as gray matter volume in this region was positively correlated with facets of emotional intelligence related to emotional control, regardless of whether assessed via the Trait or Ability models. Higher gray matter volume in ventromedial prefrontal cortex was associated with demonstrated ability to understand and manage emotional information (i.e. MSCEIT Strategic emotional intelligence) and self-reported traits suggestive of the ability to cope with adversity without losing emotional control (i.e. EQ-i Stress Management). A previous study also showed that gray matter volume in this same region was associated with one facet of perceived emotional intelligence that involves directing attention toward conscious subjective emotional experience [15]. Previous work has also suggested that higher Trait emotional intelligence is associated with reduced ventromedial prefrontal cortex and frontal pole activation [12,13], suggesting greater neural efficiency within these prefrontal regions among individuals with higher emotional functioning. The present findings also support the role of the insular cortex in emotional intelligence. We found that greater gray matter volume in the posterior insula, a region important for interoceptive perception [9], and in the anterior insula, a region that is critical for integration of cognition and emotion [11], was associated with higher Total and Strategic emotional intelligence scores. In contrast to the significant correlations between emotional intelligence and gray matter volume in the ventromedial prefrontal cortex and insula, there was no relationship with amygdala volumes. Although the amygdala is part of the Somatic Marker Circuitry, previous morphometric work has also failed to demonstrate volumetric relations between the amygdala and emotional intelligence [14,15]. This may be a consequence of the fact that the amygdala comprises several functionally distinct nuclei, each of relatively small volume compared with the spatial resolution of modern voxel-based morphometry methods, such as those used here. Future work may examine these emotional intelligence measures in groups with various forms of psychopathology, or even study the potential of emotional intelligence to predict resilience against stress or psychiatric disease.

Conclusion

Trait and Ability measures involving emotional regulation facets of emotional intelligence were both related to gray matter volume in the ventromedial prefrontal cortex, whereas only Ability emotional intelligence was specifically associated with gray matter volume in the insular cortex. These findings support the role of the ventromedial prefrontal cortex and insula as key nodes in the Somatic Marker Circuitry, and further suggest that larger volume of these regions in nominally healthy adult participants is associated with greater capacities for understanding and using emotional information and for demonstrating resilience and effective coping in the face of stress and adversity.

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Conflicts of interest

There are no conflicts of interest.

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Voxel-based morphometric gray matter correlates of daytime sleepiness

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ABSTRACT

Sleep disorders such as narcolepsy, obstructive sleep apnea, and chronic insomnia have been associated with reduced gray matter volume of the ventromedial prefrontal cortex (VMPFC). Functional neuroimaging and behavioral data also implicate this region as important in sleep-related problems and the ability to resist the impairing effects of sleep loss on cognition. However, no study has linked gray matter volume within this region to normal self-reported levels of daytime sleepiness. We therefore hypothesized that reduced gray matter volume within the VMPFC would be related to greater self-reported levels of general daytime sleepiness, as assessed by the Epworth Sleepiness Scale (ESS) in a sample of 36 healthy non-clinical participants. Using voxel-based morphometry, scores of the ESS were correlated with gray matter volume, after controlling for age, gender, and whole brain volume. Daytime sleepiness correlated negatively with gray matter volume in a cluster of voxels within the left gyrus rectus and medial orbitofrontal cortex. Findings converge with prior evidence to suggest that the VMPFC and medial orbitofrontal cortex may play a particularly important role in sleep–wake related phenomena including sleep disorders and trait-like individual differences in vulnerability to the impairing effects of sleep deprivation on neurobehavioral performance, and also in normal variations in self-reported daytime sleepiness.

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1. Introduction

Sleepiness refers to the biological pressure to fall asleep and can be differentiated from other related concepts such as fatigue, tiredness, or low energy, which do not necessarily reflect sleep propensity [1]. While excessive sleepiness can be a symptom of a number of medical disorders, even healthy normal individuals report feeling sleepy immediately following awakening, during early morning hours, or when the accumulated sleep debt is extended sufficiently beyond normal [14]. Without sufficient nocturnal sleep, most individuals will report increased daytime sleepiness on subjective measures and will show a shortened objective latency to fall asleep [23]. Thus, the most common and obvious effect of sleep loss is sleepiness.

Behavioral and neuroimaging studies have shown that insufficient sleep can have a number of adverse effects on brain functioning. Total overnight sleep deprivation is associated with significant reductions in cerebral glucose metabolism, particularly within the prefrontal cortex [25,29]. These declines appear to be especially prominent in the ventromedial prefrontal cortex (VMPFC) and orbitofrontal cortex (OFC) regions [25]. Furthermore, behavioral tasks sensitive to dysfunction within VMPFC and OFC, such as emotional decision-making, moral judgment, and olfactory perception, seem to be particularly sensitive to sleep deprivation [13,16–18,15]. In particular, tasks involving sensitivity to rewards and punishments and their valuation during decision-making processes appear to be adversely affected by sleep deprivation and are often associated with altered functional activation within the VMPFC [20,27], a region which includes subgenual cingulate cortex, gyrus rectus, and medial orbitofrontal regions. Behavioral data further suggest that poorer baseline performance on tasks sensitive to OFC integrity is predictive of vulnerability to the impairing effects of sleep loss on vigilance performance [19]. Together, the data suggest that sleep loss has a particularly notable effect on VMPFC functioning.

While it is clear that sleep loss can affect brain functioning in the VMPFC, it is not clear whether sleepiness or sleep-related problems are related in a more durable way to measureable differences in brain structure of this region. Only a small handful of studies have examined the relationship between sleep-related variables and brain structure. Using voxel-based morphometry (VBM), one study showed that patients with narcolepsy and cataplexy showed significant reductions in gray matter concentration within VMPFC, particularly the left gyrus rectus [11]. Similarly, a separate study of patients with obstructive sleep apnea (OSA) showed reduced gray matter concentrations in the left gyrus rectus as well

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[12]. Finally, reduced gray matter volume was also found in the left OFC among a sample of patients with chronic insomnia [2]. These findings suggest that morphology of the VMPFC may be either affected by, or may itself contribute to some sleep-related difficulties. To our knowledge, no study has examined the relationship between brain structure and self-reported daytime sleepiness among healthy normal individuals. Therefore, we correlated gray matter volume (GMV), as measured by VBM, with self-ratings of daytime sleepiness, hypothesizing that higher ratings of general sleepiness would be associated with reduced GMV within the VMPFC.

2. Methods

2.1. Participants

Thirty-six right-handed volunteers (mean ± standard deviation = 30.0 ± 8.9 years, range 18–45; 20 males; 16 females) were recruited from the local area of Boston, MA, via flyers posted in community centers and advertisements in local newspapers and the Internet. Participants received payment for their participation. All participants underwent a detailed intake interview that included screening questions from the Structured Clinical Interview for DSM-IV Disorders (SCID-I/P) [7] and questions on prior psychological, psychiatric or other mental health counseling and diagnoses. Exclusion criteria were a history of Axis I disorder, neurological illness or head injury, sleep-related disorder, current use of psychotropic medication or other medications known to affect functional neuroimaging, or current chemotherapy or radiation therapy. This study was approved by the McLean Hospital Institutional Review Board. All participants provided written informed consent.

2.2. Materials and procedure

On the day of magnetic resonance scanning, each participant completed the Epworth Sleepiness Scale (ESS), a self-report measure of typical or trait-like daytime sleepiness [8]. This self-administered inventory consists of eight situations (e.g. sitting and reading; as a passenger in a car for an hour without a break) which participants rate for the chance of dozing on a 4-point scale (0 – would never doze, 1 – slight chance of dozing, 2 – moderate chance of dozing, 3 – high chance of dozing). A sum score is calculated (maximum: 24), whereby a high score reflects greater daytime sleepiness. Volunteers also completed an information questionnaire about their sleep the night before and caffeine intake during the hours prior to the scan. The ESS has been shown to have good internal consistency reliability (α ranging from .70 to .88) [9,24], and shows high test–retest reliability following a 5-month interval ($r = .82$) [9], suggesting that the construct of sleepiness measured by the scale is highly stable. The ESS also correlates significantly with other subjective and objective measures of sleep quality [4,24].

2.3. MRI parameters

Participants underwent structural magnetic resonance imaging at 3.0 T (SIEMENS Tim Trio) using a 12-channel head coil. Using a T1-weighted three-dimensional MPRAGE sequence (TR/TE/flip angle = 2.1 s/2.25 ms/12°), 128 slices were obtained in the sagittal plane (256 × 256 matrix) with a slice thickness of 1.33 mm and a voxel size of 1 mm × 1 mm × 1.33 mm.

2.4. Voxel-based morphometry (VBM)

Analysis of gray matter volumes was undertaken using voxel-based morphometry (VBM) as implemented in the VBM8 toolbox (http://dbm.neuro.uni-jena.de/vbm.html) in SPM8 (Wellcome Department of Imaging Neuroscience Group, London, UK; http://www.fil.ion.ucl.ac.uk/spm). Preprocessing was completed using the VBM8 default settings for a non-linearly modulated normalized VBM (i.e., total brain volume served as a covariate). In brief, T1-weighted structural images were DARTEL-normalized to the standard stereotaxic space of the Montreal Neurological Institute (MNI), resliced to 1.5 mm × 1.5 mm × 1.5 mm, and then segmented into gray matter, white matter and cerebrospinal fluid. Following preprocessing, data quality checks that are part of the default VBM8 data analysis (i.e., inspection of all normalized bias-corrected volumes for artifacts; visualization of covariance between normalized gray matter volumes) did not identify artifacts or outliers. Spatial smoothing of the normalized gray matter images was conducted by application of an 8 mm full-width at half-maximum (FWHM) Gaussian kernel.

2.5. Statistical analysis

The Shapiro–Wilk Test in SPSS 16.0 was used to assess normal distribution of ESS scores. To evaluate the relationship between ESS scores and regional GMV, the normalized and smoothed gray matter images were entered into a random effects multiple regression analysis (absolute threshold: 0.2) in SPM8 with age and gender included as nuisance covariates. Regression diagnostics including Cook's distance were run in SPSS 16.0 to identify outliers. For our specific hypothesis regarding the VMPFC, data were evaluated at an initial threshold of $p < .001$ (uncorrected), with an extent $k \geq 40$ voxels, and then a small volume correction (SVC) was applied at a threshold of $p < .05$ within bilateral 10 mm spheres centered on the VMPFC coordinates showing the greatest sensitivity to sleep deprivation as reported in the Thomas et al. [25] study of glucose metabolism (i.e., Talairach coordinates, $x = -12$, $y = 24$, $z = -20$; $x = 12$, $y = 16$, $z = -20$). Whole brain analyses of all other significant regions are also reported ($p < .001$, uncorrected, $k \geq 30$), along with separate post hoc analyses by gender. Because these latter analyses were not hypothesized a priori, they are presented only for completeness of reporting, but are not interpreted.

3. Results

Participants reported obtaining 7.0 ± 1.0 h of sleep the night before testing and consumed an average of 74.5 ± 105.2 mg (range: 0–275) of caffeine on the day of their assessment. The mean score on the ESS was 5.4 ± 3.2 (range: 0–14), and was normally distributed ($W = .96$, $p = .16$). There was no significant correlation between ESS scores and self-reported sleep the previous night ($r = -15$, ns) or caffeine intake ($r = -11$, ns) on the day of testing, suggesting that neither of these variables significantly influenced sleepiness ratings.

Consistent with our hypothesis, the data showed that typical daytime sleepiness was significantly negatively correlated with GMV for a cluster of 48 voxels within the left VMPFC (Fig. 1), which remained significant with a small volume correction (FWE, $p = .016$). Regression diagnostics did not identify outliers that may have biased regression findings ($D = 0.04$; range: .00–.63). There were several other regions that also emerged as significant ($p < .001$, uncorrected, $k \geq 30$) in the whole brain analyses and are listed in Table 1 for completeness, although none of these survived FWE correction for multiple comparisons ($p < .05$).

Although not hypothesized, we also undertook separate exploratory post hoc analyses of the data to determine whether the correlation between ESS and gray matter volume within the VMPFC was similar for men and women. For men, the negative correlation was significant within a cluster of 288 voxels of the left

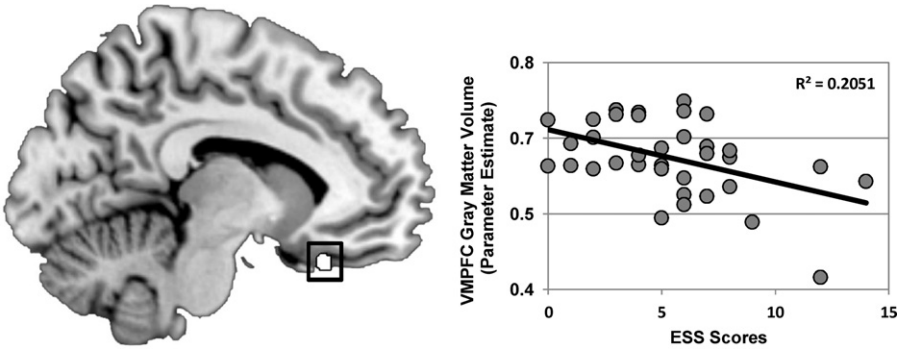

Fig. 1. Sagittal view of the left VMPFC (gyrus rectus) cluster that was inversely correlated with daytime sleepiness and the corresponding scatterplot showing the relationship between ESS and average gray matter volume for the cluster located at MNI coordinates $x = -9$, $y = 27$, $z = -26$.

VMPFC [MNI: $x = -15$, $y = 27$, $z = -21$], and remained significant following small volume correction (FWE, $p < .001$). In contrast, this relationship was not significant among the smaller female sample.

4. Discussion

As hypothesized, reduced GMV in the left VMPFC was significantly related to greater self-reported daytime sleepiness on the ESS. In fact, the region of reduced volume was near the same coordinate location as that which has previously been associated with the greatest medial OFC reductions in absolute glucose metabolism following 24 h of sleep deprivation [25]. The region of reduced GMV was also proximal to an area of the VMPFC that has been implicated in altered decision-making during sleep deprivation [27], and which shows reduced GMV in patients with narcolepsy [11], sleep apnea [12], and chronic insomnia [2]. Notably, a recent neuroimaging study also demonstrated that the wake-promoting agent modafinil leads to increased functional activation within regions of the VMPFC and medial OFC that essentially encompass the same region reported in the present study [10]. The OFC also shows the greatest relative change in A_1 adenosine receptor (A_1AR) upregulation following a night of total sleep deprivation [6], a process that is believed to contribute directly to homeostatic sleep regulation and to modulate the effects of caffeine on alertness. Overall, the converging evidence points to an important role for the VMPFC/medial OFC regions in conditions that may affect sleepiness and wakefulness.

Interestingly, the VMPFC/OFC region has not been directly implicated in basic arousal or alertness, but does show significant covariation of activation across various stages of sleep and conscious wakefulness [3,21]. Recent data from our group suggest that poorer baseline functioning on tasks associated with OFC integrity is associated with greater vulnerability to the adverse effects of sleep deprivation on psychomotor vigilance performance [19]. In the context of those prior findings, the present data raise the possibility that individual variation in GMV of the VMPFC/OFC may contribute to the well-documented trait-like individual differences in the ability to resist the cognitively impairing effects of sleep deprivation [26].

Although reduced GMV in the VMPFC was clearly related to increased daytime sleepiness in the present study, the mechanisms underlying this relationship remain uncertain. The participants in the present study were not sleep deprived, and neither total sleep time the night before the study nor caffeine intake on the day of the study was related to self-reported sleepiness. It is possible that increased scores on the ESS reflect chronic sleep debt, which over extended periods might lead to reduced GMV in the VMPFC through cellular apoptosis or which may affect gray matter development during sensitive periods. This explanation, however, appears to be contradicted by several animal studies which suggest that chronic sleep restriction has no adverse effect on neuronal health [5], and may, in some instances, be mildly protective against neurotoxicity [22] and inflammatory processes [28]. Thus, a more tenable explanation would be that smaller GMV within VMPFC/medial OFC regions might be a pre-existing condition that contributes to the vulnerability to excessive daytime sleepiness or other sleep-related problems. Ultimately, this question would be best addressed by longitudinal research tracking both GMV and sleep-related variables. Future research may address the potential of this region as a marker for susceptibility to sleep-related disorders or as a target for possible neurobiological intervention among such populations. The present study was also limited by the relatively small sample size, which may have reduced statistical power to detect some associations. Finally, although not hypothesized, post hoc analyses revealed that the correlation between daytime sleepiness and VMPFC gray matter volume was significant only among males when the group was divided by gender. However, because this was not hypothesized *a priori* and due to the reduction in power that occurred when the sample was divided, these differences cannot be validly interpreted without replication. However, the possibility of gender differences in these relations should be addressed in future research.

Table 1
Regional correlations between ESS scores and GMV.

Region of activation	Volume	*x*	*y*	*z*	*T*-score
Positive correlations					
R. superior occipital gyrus	157	24	−76	33	5.44
L. calcarine cortex	187	−18	−70	10	4.67
L. cuneus	135	−3	−78	21	4.33
L. superior frontal gyrus	72	−24	0	45	4.19
Negative correlations					
R. cerebellar crus area 2	373	20	−79	−32	3.86
R. cerebellum area 8	58	30	−65	−50	3.79
L. gyrus rectus/sup orbitofrontal gyrus	48	−9	27	−26	3.78*

Notes: L, left hemisphere; R, right hemisphere; Atlas coordinates are listed in the standard space of the Montreal Neurological Institute (MNI). Volumes are in voxels (1.5 mm × 1.5 mm × 1.5 mm). All clusters are significant at $p < .001$ (uncorrected), $k \geq 40$. ESS = Epworth Sleepiness Scale; GMV = Gray Matter Volume.
* $p < .05$ (small volume corrected).

5. Conclusion

In a sample of healthy normal individuals, reduced GMV of the left VMPFC region was significantly correlated with increased self-reported daytime sleepiness. The anatomical concordance of this region with similar loci reported using a variety of neuroimaging and behavioral techniques suggest that the VMPFC/medial OFC may play a particularly important role in the individual expression of

sleep–wake related phenomena. Exploration of the potential role of this region as a focus for treatment of sleep and arousal problems is warranted.

Conflicts of interests

None declared.

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Self-reported nocturnal sleep duration is associated with next-day resting state functional connectivity

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Sleep deprivation affects cerebral metabolism and reduces the functional connectivity among various regions of the brain, potentially explaining some of the associated mood and emotional changes often observed. Prior neuroimaging studies have only examined the effects of sleep deprivation or partial sleep restriction on functional connectivity, but none have studied how such connectivity is associated with normal variations in self-reported sleep duration the night before the scan. We examined the relationship between sleep duration and resting state functional connectivity among healthy volunteers who slept at home according to their own schedules. Thirty-nine healthy individuals aged 18–45 (21 females) completed a questionnaire asking about their recent sleep habits and entries in their sleep diary for the previous night, followed by resting state functional MRI at 3 T. Participants reported sleeping between 5.0 and 8.5 h the night before the scan ($M = 7.0$, SD = 0.9). Seed regions were placed in the medial prefrontal cortex and posterior cingulate cortex nodes of the default mode network, regions previously implicated in sleep deprivation. Longer self-reported sleep duration was associated with significantly enhanced functional connectivity between the medial prefrontal cortex and posterior cingulate, as well as greater anticorrelations with parietal, occipital, and lateral prefrontal regions. Findings suggest that even normal variations in sleep duration measured by self-report are related to the strength of functional connectivity within select nodes of the default mode network and its anticorrelated network. *NeuroReport* **23:741–745** © **2012 Wolters Kluwer Health | Lippincott Williams & Wilkins.**

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Introduction

Sleep is vital to normal cognitive functioning [1]. Without adequate sleep, attention and central processing systems are adversely affected [2], potentially leading to deficits in many cognitive and affective capacities [3]. Sleep loss has been associated with slowed response times and increased lapses in vigilant attention [4], memory deficits [5], increased risk taking [6], altered mood and emotional processing [7], and impairments in executive functions such as behavioral inhibition, planning, mental flexibility, and decision making [3,8].

Although alertness and vigilance are the primary capacities impaired by sleep loss [4], some higher cognitive deficits may result from altered functioning in prefrontal executive systems [3,8]. Regional cerebral metabolism appears to be notably reduced within the ventral and medial prefrontal regions after one night of total sleep deprivation [9,10]. One brain system that involves both attention and complex cognition is the default mode network (DMN) and its anticorrelated network (ACN). The DMN is believed to reflect internal spontaneous cognitive activity that occurs in the absence of a focused cognitively demanding task (e.g. resting state), and includes the medial prefrontal cortex (MPFC), posterior cingulate cortex (PCC), and retrosplenial cortex, as primary nodes. In contrast, the ACN reflects the regions that are typically positively activated by mentally engaging tasks, including a bilateral dorsal attention system and a right lateralized ventral attention system. Recent evidence suggests that even one night of controlled sleep deprivation in the laboratory is associated with reduced functional connectivity (i.e. interregional temporal correlations) within the DMN and ACN [11]. Similarly, restriction of sleep to 3.5 h during a single night in the laboratory was also associated with reduced functional connectivity of DMN and ACN nodes [12].

Although experimental laboratory sleep deprivation appears to be associated with reduced functional connectivity in these critical networks, it was of interest to determine whether similar findings would be observed in relation to the normal variability in sleep obtained by most healthy adults living in unconstrained environments and allowed to sleep according to their own schedules. Here, participants reported their previous night's sleep duration on a sleep diary and underwent a resting state functional MRI (fMRI) scan. We hypothesized that greater duration of sleep on the prescan night would be associated with stronger functional connectivity among brain regions associated with the DMN.

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Methods

Participants

Thirty-nine (21 male; 18 female) right-handed, healthy adults ranging in age from 18 to 45 years ($M = 30.4$, SD = 8.7) were recruited from the Boston metropolitan area. Participants were predominantly White (59%), African American (23%), or Asian American (10%), with an average of 15 years of education (SD = 2.0), and were screened by a trained research technician via a detailed series of medical and psychiatric history questions. Participants were excluded for severe medical conditions (e.g. diabetes, heart conditions), history of head injury, loss of consciousness for more than 30 min, seizures, brain tumors, other neurologic conditions, or any prior history of axis I disorders or reported symptoms consistent with such diagnoses. Participants were not currently using any psychoactive medications or illicit substances, and were free from history of drug or alcohol treatment. Alcohol use was required to be lower than Center for Disease Control criteria for excessive drinking (*http://www.cdc.gov/alcohol*). All were self-reported normal sleepers, averaging 5.5–9 h of sleep on weekdays ($M = 7.4$, SD = 0.9) and 4–10 h on weekends ($M = 7.6$, SD = 1.3). Participants were low-to-moderate caffeine users ($M = 107.7$ mg/day, SD = 118.2), and had consumed close to their typical caffeine intake on the day of the scan ($M = 81.6$ mg, SD = 113.3). This research was approved by the McLean Hospital Institutional Review Board. All participants provided written informed consent and were compensated for their time.

Materials and procedure

Participants arrived for the study between 9 and 11 a.m., completed informed consent procedures, and filled-out a questionnaire asking about sleep habits and the number of hours of sleep obtained the preceding night. Between 1 and 3 p.m., participants underwent a series of structural and functional MRI scans including a 6-min resting state fMRI scan with instructions to rest with eyes open.

Magnetic resonance imaging parameters

Data were acquired on a 3 T Siemens Tim Trio scanner (Erlangen, Germany) using a 12-channel head coil. For use in spatial normalization and to remove tissue confounds, structural images were collected using a T1-weighted 3D MPRAGE sequence (TR/TE/flip angle = 2.1 s/2.25 ms/12°) yielding 128 sagittal slices (256 × 256 matrix) with a slice thickness of 1.33 mm and a voxel size of 1 × 1 × 1.33 mm. Resting scan images were collected over 34 transverse interleaved slices using a T2*-weighted blood oxygen level dependent echoplanar imaging sequence (TR/TE/flip angle = 2.0 s/30 ms/90°), with 180 images per slice (3.5 mm thickness, no skip; 22.4 cm field of view; 64 × 64 acquisition matrix).

Image processing

Data were preprocessed using standard algorithms in SPM8, including motion correction, slice-timing correction, coregistration to each subject's anatomical image, spatial normalization, and spatial smoothing using an isotropic Gaussian kernel (full-width at half-maximum = 6 mm, and resliced to 2 × 2 × 2 mm). Functional connectivity analysis was conducted using the Functional Connectivity Toolbox version 13i (*http://www.nitrc.org/projects/conn*). Data were band-pass filtered (0.008, 0.10 Hz), corrected for physiological noise using a CompCor strategy [13], significant principle components of white matter and cerebrospinal fluid were removed, and motion parameters were statistically controlled. Two seed regions were placed corresponding to two primary nodes of the DMN, including the MPFC and PCC. Seed regions were defined as 10 mm spheres located at the coordinates derived from prior published work for the MPFC ($x = -1$, $y = -47$, $z = -4$) and PCC ($x = -5$, $y = -49$, $z = -40$) [14], and implemented as standard regions in the toolbox.

Statistical analysis

In the first level analysis, the residual blood oxygen level dependent signal timecourse from each seed region was extracted and Pearson correlations were computed with all other voxels in the brain to derive connectivity maps. In the second level random-effects analysis, z-score transformed connectivity maps were entered into a general linear model regression analysis evaluating the relationship between sleep the preceding night and the strength of functional connectivity for the two seed regions. In addition, as a control comparison, the same analyses were conducted again using a nonhypothesized predictor variable expected to be unrelated to resting state functional connectivity (i.e. participant self-reported height in inches). Consistent with the standard procedures suggested by the authors of the Functional Connectivity Toolbox (*http://www.nitrc.org/projects/conn*) for seed-to-voxel analyses, connectivity maps were thresholded using a combination of height and extent thresholds to control for false positives. Specifically the correlation maps were interrogated at a height threshold of P less than 0.001 (uncorrected), whereas spatial extent (i.e. cluster size) was simultaneously corrected for family-wise error at P less than 0.05.

Results

Participants all reported obtaining some sleep the night before the scan, ranging between 5.0 and 8.5 h ($M = 7.0$, SD = 0.9). Figure 1 shows that greater duration of self-reported sleep was associated with increased functional connectivity for both nodes of the DMN. Individuals who slept more on the prescan night showed greater positive connectivity between the MPFC seed region and activation within the posterior cingulate gyrus and vermis of the cerebellum, and significant anticorrelations with bilateral activation within the white matter and cortical regions near the medial frontomarginal sulcus and extending posterior to the anterior horn of the lateral ventricles (see Table 1 for coordinates). Similarly, greater

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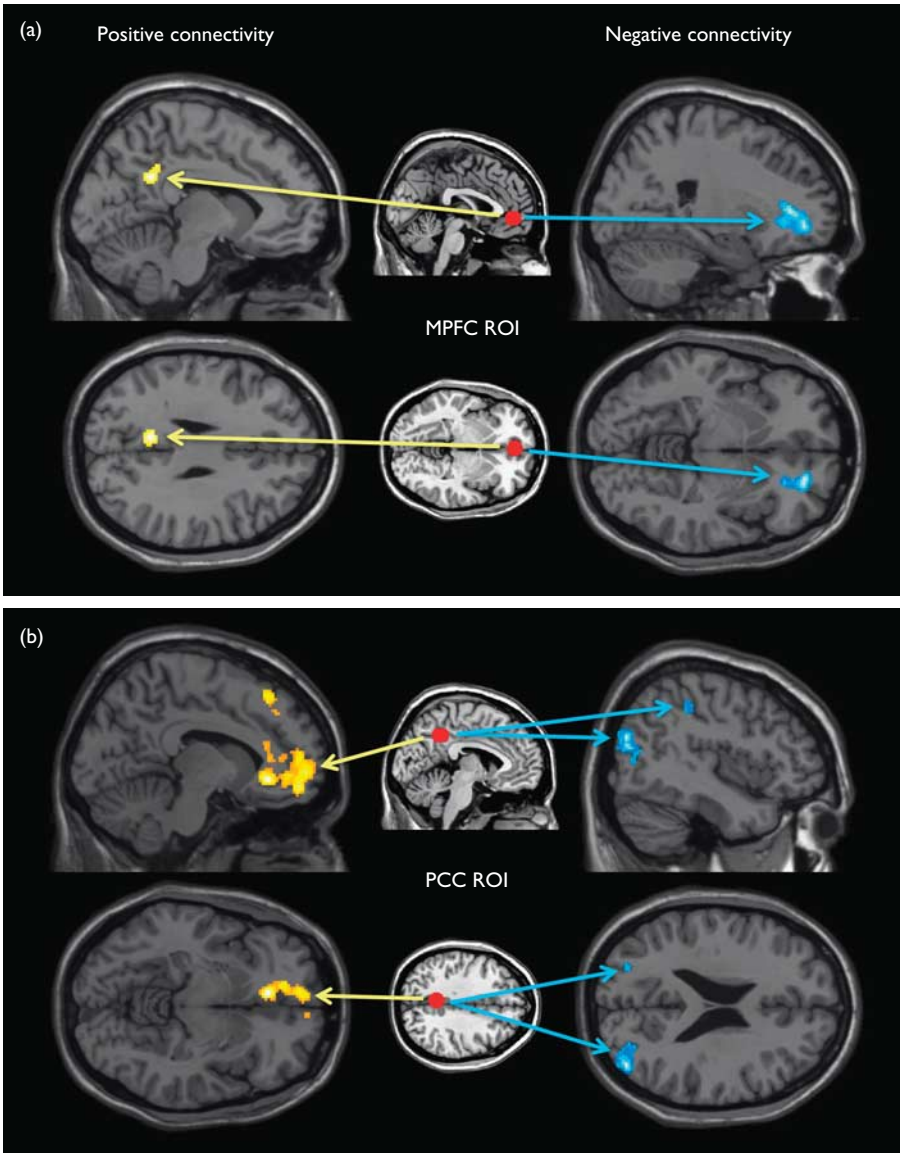
Fig. 1



Self-reported sleep duration correlates with enhanced functional connectivity with two 10 mm spherical seed-regions of interest (ROI) placed at specific nodes of the default mode network, including the medial prefrontal cortex (MPFC) and posterior cingulate cortex (PCC). (a) Self-reported sleep duration correlated with greater positive connectivity between the MPFC seed-ROI and a region in the posterior cingulate gyrus (left) and correlated with greater negative connectivity between the MPFC and a region within the white matter of the lateral prefrontal cortex (right). (b) Self-reported sleep duration was correlated with greater positive connectivity between the PCC seed-ROI and several regions within the rostral and dorsal medial prefrontal cortex (left) and greater negative connectivity with posterior parietal and occipital cortex regions (right).

self-reported sleep duration was positively correlated with greater connectivity between the PCC seed region and several regions of the MPFC (Brodmann Area 10), subgenual anterior cingulate cortex, middle frontal gyrus, and regions of the dorsal and rostral regions of the superior frontal gyrus. Longer sleep duration was associated with significantly greater anticorrelated connectivity between the PCC and bilateral regions of the superior and inferior parietal lobe, middle occipital gyrus, and inferior postcentral sulcus. In contrast, when self-reported sleep was substituted with a control parameter (i.e. participant height) as the covariate of interest, there was no correlation with resting state functional connectivity for either the MPFC or PCC.

Discussion

Longer self-reported sleep duration was associated with significantly greater next-day functional connectivity between specific nodes of the DMN and its ACN. The increase in functional connectivity with additional sleep

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Table 1 Regions showing enhanced functional connectivity with longer sleep duration

			MNI coordinates			
Seed region	Target region	Cluster size	*x*	*y*	*z*	*T*
MPFC positive						
	L. cerebellum (vermis)	919	−2	−50	−40	4.75
	L. posterior cingulate	888	−9	−51	29	4.59
MPFC negative						
	R. prefrontal WM	47 970	22	46	−6	5.15
	L. prefrontal WM	17 130	−8	24	10	5.03
PCC positive						
	L. anterior cingulate	12 216	−10	32	−8	6.50
	L. sup. frontal gyrus	15 520	−13	33	55	4.85
	R. sup. frontal gyrus	12 340	13	39	37	4.75
	L. inf. orbital frontal gyrus	671	−34	24	−20	4.69
PCC negative						
	R. mid. occipital gyrus	41 810	−10	32	−8	6.50
	R. sup./inf. parietal cortex	62 160	19	−51	43	5.41
	L. inf. parietal cortex	38 000	−27	−49	49	5.29
	L. sup./mid. occipital gyrus	912	−25	−77	27	4.83

All voxels significant at $P<0.001$, extent corrected at $P<0.05$ for family-wise error. Voxel size = $1 \times 1 \times 1$ mm.
inf., inferior; L, left; MPFC, medial prefrontal cortex; mid., middle; MNI, Montreal Neurological Institute; PCC, posterior cingulate cortex; R, right; sup., superior; WM, white matter.

is particularly remarkable, as our participants were not intentionally sleep deprived, having obtained on average, about the same amount of sleep that is typically reported on weeknights by most healthy American adults (*http://www.sleepfoundation.org*). These findings replicate and extend prior laboratory findings, which have shown that experimentally induced total sleep deprivation [11] and severe restriction of sleep to subnormal levels [12], significantly reduces functional connectivity among DMN circuits. Our findings suggest that even within the range of variability in sleep durations normally experienced by most healthy adults on weeknights, the accumulation of additional sleep up to about 8.5 h appears to be associated with greater functional connectivity within the DMN/ACN circuitry. In contrast, these relationships were not observed when a nonhypothesized control variable was used as a predictor.

The relevance of these findings to actual cognitive performance remains an open question, but emerging evidence suggests that the DMN plays an important role in various states of consciousness, particularly the balance of directed awareness toward the internal versus external milieu [15,16]. We found that longer sleep on the prescan night was associated with stronger positive functional coupling of the anterior region of the DMN (i.e. the MPFC) with the posterior cingulate, and stronger negative coupling with lateral prefrontal regions. Similarly, more sleep was associated with greater positive coupling between posterior cingulate with dorsal and rostral regions of the MPFC and negative coupling with ACN regions such as the parietal attention and occipital sensory cortices. Although the function of the DMN is not fully understood, it appears to be relatively more activated when individuals are engaged in self-referential thought or other inwardly directed mentation and is decreased as the focus of attention is directed outward toward external stimuli or focused on other cognitively challenging tasks [15].

Interestingly, recent evidence suggests that components of the DMN become uncoupled during sedation [17] and with the onset of sleep [18], pointing to its possible role in conscious awareness [12]. Total sleep loss or induced sleep pressure from partial sleep restriction also reduces functional connectivity of these systems [11,12]. Some nodes of the DMN, such as the ventromedial prefrontal cortex, have been hypothesized to play an important role in the ability to integrate emotion and cognition, a synthesis that is believed to be advantageous to effective judgment and decision making [19]. Recent evidence suggests that sleep loss alters functional connectivity between the ventromedial prefrontal cortex and emotion processing regions such as the amygdala [20], which may underlie many of the cognitive-affective deficits often seen during acute sleep deprivation [3], such as impaired moral judgment [21], poor decision making [6,22], increased risk taking [23], and difficulty coping with stress [24]. It is therefore possible that the functional connectivity of these systems may be critical in maintaining normal functioning of cognitive-affective processes, and their alteration by even modest shifts in sleep duration may underlie some of the often experienced changes in mood and cognition that occur on nights when sleep has been truncated.

A major limitation of the present study is the singular focus on resting state connectivity, so the role of sleep duration on the dynamic communication among these networks during cognitive task processing cannot be elucidated. It will be important in future work to explore the effects of various degrees of sleep pressure on the dynamic functional connectivity of these networks during active task engagement. We also highlight the fact that our data are correlational in nature and that causal inferences are not possible in this type of study design. It is, therefore, plausible that instead of greater sleep producing enhanced functional connectivity, it may be that greater connectivity leads individuals to require longer sleep. Additional research will be necessary to determine the causal chain, although when considered in the context of prior work on sleep deprivation and sleep restriction [11,12], our findings support the notion that sleep duration causally affects functional connectivity. Finally, it is important to acknowledge that our estimate of sleep duration was based on self-report, which is admittedly less reliable than objective measures such as electroencephalography or actigraphy. Replication of these findings with more objective measures of sleep will be an important next step. With these limitations in mind, we believe that the present data are compelling and suggest a significant relationship between normal variations in self-reported sleep duration and greater resting functional connectivity among several nodes of the DMN.

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Conclusion

Self-reported sleep duration within the bounds of what most healthy adults obtain each day was significantly correlated with resting functional connectivity among several nodes of the DMN and ACN. These findings highlight the important role of sleep in the normal functioning of brain neurocircuitry. Even relatively minor differences in sleep duration may be related to significant differences in the strength of the functional connectivity among these systems.

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Conflicts of interest

There are no conflicts of interest.

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SEX DIFFERENCES IN THE ASSOCIATION BETWEEN PHYSICAL EXERCISE AND IQ[1]

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Summary.—Previous research suggests that physical exercise may have beneficial effects on cognitive performance in children and the elderly, but little research has yet examined these associations in healthy adults. It was hypothesized that self-reported frequency and duration of physical exercise would correlate positively with measured intelligence on the Wechsler Abbreviated Scale of Intelligence in healthy young to middle aged adults (25 men, 28 women). Although there was a modest positive association between physical exercise and intelligence (IQ) for the group as a whole, when examined separately by sex, greater physical activity was associated with higher intelligence scores for women, whereas exercise level was essentially unrelated to intelligence among men. These associations remained consistent even after controlling for demographic and socioeconomic factors. The association between exercise and IQ appears to be moderated by sex in healthy adults, possibly through its effects on glucoregulation, insulin sensitivity, or other factors that differ between men and women.

Regular exercise and physical activity have numerous long-term health benefits. Physical activity has been associated with cardiovascular health (Shiroma & Lee, 2010), and reduced risk of type II diabetes (Laaksonen, Lindstrom, Lakka, Eriksson, Niskanen, Wikstrom, *et al.*, 2005), osteoporosis (Gomez-Cabello, Ara, Gonzalez-Aguero, Casajus, & Vicente-Rodriguez, 2012), some cancers (Lee, 2003), and psychiatric problems (Knochel, Oertel-Knochel, O'Dwyer, Prvulovic, Alves, Kollmann, *et al.*, 2012). Moreover, emerging research also points to the beneficial effects of physical activity on cognitive performance (Hillman, Erickson, & Kramer, 2008; Voss, Nagamatsu, Liu-Ambrose, & Kramer, 2011). The majority of human research has focused on the relationship between physical activity and intelligence in school age children or how exercise may stave off cognitive decline in the elderly. For instance, considerable evidence now suggests that physical activity is associated with better performance among children in school or on standardized tests (Castelli, Hillman, Buck, & Erwin, 2007; Chomitz, Slining, McGowan, Mitchell, Dawson, & Hacker, 2009; Van Dusen, Kelder, Kohl, Ranjit, & Perry, 2011), with cardiovascular fitness and aerobic capacity generally showing the strongest relation to academic performance (Fedewa & Ahn, 2011; Van Dusen, *et al.*, 2011).

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Similarly, aerobic fitness in children is associated with better performance on neuropsychological tests and measures of brain structure and function (Buck, Hillman, & Castelli, 2008; Chaddock, Pontifex, Hillman, & Kramer, 2011; Voss, Chaddock, Kim, Vanpatter, Pontifex, Raine, *et al.*, 2011). Recent studies of school age children suggest that physical exercise interventions result in improved spatial working memory and simple attention (Fisher, Boyle, Paton, Tomporowski, Watson, McColl, *et al.*, 2011), as well as improved executive functioning and enhanced functional activation of the prefrontal and parietal cortices (Davis, Tomporowski, McDowell, Austin, Miller, Yanasak, *et al.*, 2011). Preliminary evidence also suggests that increasing physical activity in children can lead to improvements on indices of fluid intelligence (Reed, Einstein, Hahn, Hooker, Gross, & Kravitz, 2010), but little is known about the effects of exercise on well normed and standardized measures of intellectual ability such as the Wechsler scales or Stanford-Binet tests.

Exercise has also been shown to improve neuropsychological and cognitive functioning in the elderly (Molloy, Beerschoten, Borrie, Crilly, & Cape, 1988), and there is some evidence that physical activity may even lead to enhanced brain volume over time in healthy geriatric individuals (Mortimer, Ding, Borenstein, Decarli, Guo, Wu, *et al.*, 2012). Elderly women in their 80s who had greater activity levels, as quantified by actigraphy, showed better performance on mental status and cognitive set-shifting tasks (Barnes, Blackwell, Stone, Goldman, Hillier, & Yaffe, 2008). A recent study showed that a six-month intervention consisting of low to moderate physical activity yielded significant improvement in memory performance and increased gray matter volume within prefrontal and cingulate cortices among elderly participants (Ruscheweyh, Willemer, Kruger, Duning, Warnecke, Sommer, *et al.*, 2011). Similarly, a randomized trial of physical activity among elderly individuals was associated with significant improvements in cognitive functioning at one-year, including psychomotor processing speed, auditory recall, and Stroop performance (Williamson, Espeland, Kritchevsky, Newman, King, Pahor, *et al.*, 2009), and moderate to high physical activity levels have been associated with reduced risk of cognitive decline over a two year period compared to inactive individuals (Etgen, Sander, Huntgeburth, Poppert, Forstl, & Bickel, 2010). Recent evidence in older adults also suggests that there may be sex differences in the beneficial effects of exercise on cognitive abilities, with the greatest positive effects observed among women (Middleton, Kirkland, & Rockwood, 2008).

In contrast to the large amount of data suggesting a relationship between physical activity and cognition in children and geriatric samples, there has been comparatively little research on this topic in healthy adult

populations. Notably, a recent large-scale population study of male Swedish military conscripts showed that cardiovascular fitness at age 18 years was significantly related to several cognitive abilities, including higher scores on an index of intelligence (Åberg, Pedersen, Toren, Svartengren, Backstrand, Johnsson, *et al.*, 2009). Moreover, examination of a subset of the sample comprising monozygotic twin pairs revealed that more than 80% of the relation between cardiovascular fitness and cognition was accounted for by environmental rather than genetic factors. That study included a number of tasks assumed to measure intellectual functioning, but no study has yet evaluated the association between exercise and intelligence in healthy adults using a well established, psychometrically validated scale of intelligence, such as one of the Wechsler scales. Furthermore, no study has examined the role of sex as a moderator of the relationship between physical activity and intelligence in healthy adults. Here, the relation between physical activity and measured intellectual ability was studied in a sample of young to middle-aged adult men and women, using a well-validated, highly reliable, and individually administered scale of intelligence.

Method

Participants

Fifty-three healthy adults (25 men, 28 women), ranging in age from 18-44 years were recruited from the Boston metropolitan area via internet advertisements and flyers. Participants had no history of serious or chronic diseases, neurological, psychiatric, or substance use disorders (including alcohol and illicit drugs) as assessed by telephone screening. The Body Mass Index (BMI) of participants was within the normal range ($M = 24.3$, $SD = 3.2$). Participants were predominantly Euro-American (62.3%), although other ethnic groups were represented, including African American (17.0%), Asian American (11.3%), Hispanic/Latino (5.7%), and Other (3.8%). Men and women did not differ significantly with regard to age, body mass, or ethnicity (all ps $> .05$). To evaluate the contribution of socioeconomic status and neighborhood poverty on the relation between exercise and intelligence, data regarding median inflation-adjusted 12-month household income and the percentage of the participant's neighborhood below the poverty line (U.S. Census Bureau, 2010) were extracted based on census tract of home address. On average, men lived in neighborhoods where the median income was $73,718, while women lived in neighborhoods with a slightly lower income of $59,263, ($p = .05$), although the proportion of the population living below the poverty line was not significantly different between the two groups. All participants provided written informed consent and were compensated for their time.

Materials and Procedure

Participants completed a brief questionnaire about their exercise habits, including their average number of workouts per week and the average number of minutes of exercise per workout (see Table 1). The total hours of exercise per week was calculated as the product of those two variables for each individual. Intelligence quotient (IQ) was then individually assessed with the Wechsler Abbreviated Scale of Intelligence (WASI; Pearson Assessment, Inc., San Antonio, TX), which provides scores for Full Scale IQ, Verbal IQ, and Performance IQ. The WASI is one of the most widely used intelligence scales in the world and has reported reliability of .98 for Full Scale IQ, with extremely high test-retest reliability, and correlates .92 with the more comprehensive Wechsler Adult Intelligence Scale-III (WAIS; Pearson Assessment, Inc., San Antonio, TX), the current gold standard in intelligence testing. A trained and experienced bachelor's level research assistant who was blind to the study hypotheses administered the WASI under the supervision of a licensed doctoral level neuropsychologist.

Analysis

Pearson correlations were used to evaluate the linear association between exercise and IQ variables. Furthermore, based on responses to the exercise questionnaire, participants were divided into three categories of exercise level: None (0 minutes per week; $n = 11$), Low-Moderate (1–180 minutes per week; $n = 21$), High (>180 minutes per week; $n = 21$). These categories were used in a series of three 2 (sex) × 3 (exercise level) analyses of covariance (ANCOVAs) with Full Scale, Verbal, and Performance IQ as dependent variables. To control for possible socioeconomic and demographic factors on intelligence and exercise behavior, the following covariates were entered in the model: age, BMI, neighborhood median household income, percentage of the neighborhood below the poverty line, and minority ethnic status.

Results

Total Sample Correlations

Men and women did not differ significantly with regard to age, education, measures of intelligence, or frequency of workouts, although men showed a longer duration of each workout session and more total minutes of weekly exercise (see Table 1). For the sample as a whole, however, the frequency of workouts per week was significantly correlated with Full Scale IQ ($r = .42$, $p = .002$), Verbal IQ ($r = .35$, $p = .01$), and Performance IQ ($r = .43$, $p = .001$). Of note, these correlations remained significant in partial correlation analysis after controlling for socioeconomic and demographic variables including age, BMI, neighborhood median household income,

TABLE 1
DESCRIPTIVE STATISTICS AND PEARSON CORRELATIONS BETWEEN SELF-REPORTED PHYSICAL ACTIVITY AND INTELLIGENCE QUOTIENT (IQ) BY SEX

Variable	Men ($n = 25$)		Women ($n = 28$)		Group Difference p
	M	*SD*	*M*	*SD*	
Means					
Age	30.8	8.7	28.0	6.3	.18
Education	14.8	2.0	15.5	2.2	.26
Full Scale IQ	113.0	15.3	110.0	17.0	.52
Exercise Sessions per week	3.5	2.2	3.0	2.3	.41
Minutes per session	58.9	42.3	36.9	24.0	.02*
Minutes per week	252.1	213.1	132.1	102.5	.01*
Pearson Correlation					
Correlations with Full Scale IQ					
Exercise Sessions per week	.09		.67‡		.01
Minutes per session	−.22		.36		.04*
Minutes per week	−.18		.63‡		.002*
Correlations with Verbal IQ					
Exercise sessions per week	.17		.49*		.20
Minutes per session	−.14		.18		.28
Minutes per week	−.13		.47*		.03*
Correlations with Performance IQ					
Exercise Sessions per week	−.04		.72‡		.001†
Minutes per session	−.30		.48*		.004†
Minutes per week	−.23		.68‡		.0004‡

Note.—Correlations were compared via Fisher's *r*-to-*z* transform. *$p < .05$. †$p < .005$. ‡$p < .001$.

percentage of the neighborhood below the poverty line, and minority ethnic status (all ps $< .05$). In contrast, the number of minutes of exercise per workout session and the total minutes of exercise per week were not significantly correlated with IQ for the sample as a whole and were unchanged when socioeconomic and demographic variables were statistically controlled.

Correlations by Sex

As evident in Table 1, the magnitude of the aforementioned associations differed significantly between men and women. Overall, correlations between these variables were consistently nonsignificant for men but were significant and moderate to high for women for Full Scale IQ and Performance IQ, but weak for Verbal IQ. These relationships did not change markedly when socioeconomic and demographic variables were statistically controlled, with most partial correlations showing a modest but slight increase in magnitude. These findings provide further support for the link between physical activity and IQ, but also suggest that these relationships may be moderated by sex.

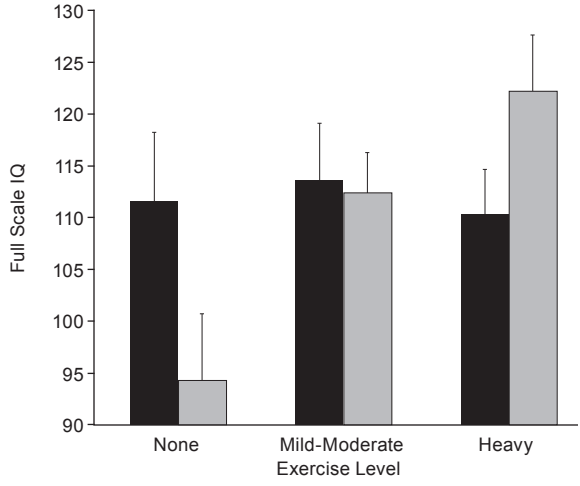

FIG. 1. Interaction between level of exercise and sex on Full Scale Intelligence Quotient (IQ) scores. Men (■); Women (▒).

Exercise Level by Sex

To more effectively evaluate the relation between exercise and intelligence, the sample was divided into three exercise level categories and compared using ANCOVA, with socioeconomic and demographic variables held constant. As evident in Fig. 1, there was a significant interaction between sex and exercise category ($F_{2,42} = 3.21$, $p = .05$, partial $\eta^2 = 0.13$). Whereas men showed no significant effect of exercise level on intelligence, women showed a trend of increasing intelligence with more exercise ($F_{2,42} = 5.28$, $p = .009$, partial $\eta^2 = 0.20$). *Post hoc* comparisons revealed that women who exercised at low-moderate ($p = .02$) and high ($p = .002$) levels had significantly higher Full Scale IQ than those who did not exercise at all. For Verbal IQ, there was no significant main effect of sex or exercise group, and no significant interaction between these variables. However, for Performance IQ, there was a significant interaction between sex and exercise level ($F_{2,42} = 7.12$, $p = .002$, partial $\eta^2 = 0.25$). Fig. 2 shows that there was no effect of exercise level on Performance IQ in men, whereas women showed significantly greater ability with increasing exercise ($F_{2,42} = 10.40$, $p = .0002$, partial $\eta^2 = 0.33$). *Post hoc* comparisons revealed that women who exercised at low-moderate and high levels had significantly higher Performance IQ than those who did not exercise at all ($p < .001$). Furthermore, women who did not exercise showed lower Performance IQ than men who did not exercise ($p < .006$), while women who were heavy exercisers tended to score higher than men who exercised at a similar level ($p < .02$).

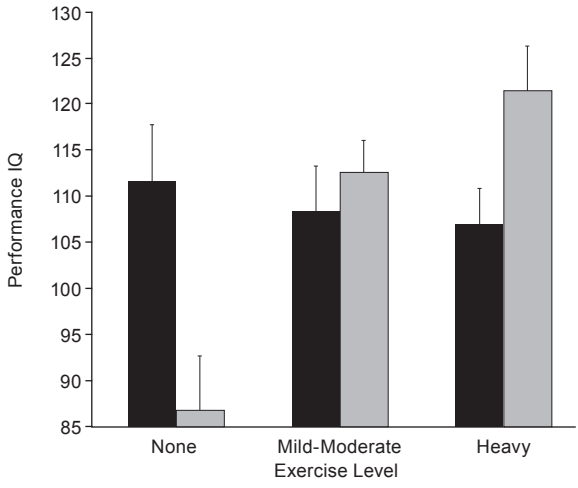

FIG. 2. Interaction between level of exercise and sex on Performance Intelligence Quotient (IQ) scores. Men (■); Women (□).

DISCUSSION

In a cross-sectional sample of healthy adults, those who exercised more frequently tended to have higher measured intelligence, including Full Scale, Verbal, and Performance IQ, while total minutes of exercise per workout or total minutes per week were unrelated to IQ. Notably, however, the strength of these associations was moderated by the sex of the participant, with women showing highly significant correlations between measures of intelligence and both workout frequency and total time spent exercising per week. Furthermore, the correlations remained statistically significant even after controlling for a variety of demographic and socioeconomic factors that could potentially affect physical activity or intelligence as measured by standardized tests. Men, on the other hand, showed no significant correlations between physical exercise and intelligence in this sample. On the whole, similar findings emerged when the data were grouped according to broad exercise categories (i.e., none, low to moderate, high) and subjected to an analysis of covariance, controlling for demographic and socioeconomic variables, with women who exercise showing significantly greater full scale and performance IQ scores than those who did not report that they engaged in physical activity. For men, these variables appeared to be unrelated. Overall, these findings suggest that physical exercise is significantly related to relatively stable intellectual capacities among healthy adults, but that this relationship appears to be moderated by the sex of the individual.

The present findings are consistent with prior work in children and

older adults showing that physical activity leads to improvements in cognitive functioning (Ahn & Fedewa, 2011) and confers some protection against the cognitive declines of aging (Colcombe & Kramer, 2003; Jedrziewski, Ewbank, Wang, & Trojanowski, 2010; Voss, Nagamatsu, *et al.*, 2011). Indeed, considerable evidence now suggests that physical exercise is beneficial for cognition and may be associated with actual neuroplastic changes in brain structure and concomitant changes in functioning (van Praag, 2009). Aerobic training, in particular, appears to be associated with enhanced cognition, and has been associated with improved memory performance (Voss, Nagamatsu, *et al.*, 2011). Physical exercise leads to greater cardiovascular fitness in general (Shiroma & Lee, 2010), and has been shown to increase blood flow within the dentate gyrus of the hippocampus in particular, a cerebral structure that is critical for memory formation and recall (Pereira, Huddleston, Brickman, Sosunov, Hen, McKhann, *et al.*, 2007). In addition to its effects on cerebral blood flow, animal research has also pointed to a number of neurogenerative and neuroprotective effects of physical activity. Aerobic exercise has been shown to increase neurogenesis of hippocampal neurons (van Praag, Christie, Sejnowski, & Gage, 1999; van Praag, Kempermann, & Gage, 1999), and appears to influence long-term potentiation (O'Callaghan, Ohle, & Kelly, 2007), both of which may contribute to some aspects of memory performance. In one recent study, a controlled trial of aerobic exercise led to a 2% increase in hippocampal volume, which was accompanied by improved spatial memory (Erickson, Voss, Prakash, Basak, Szabo, Chaddock, *et al.*, 2011). Physical exercise has also been shown to increase the proliferation of new blood vessels within the brain (Voss, Nagamatsu, *et al.*, 2011), a finding that may play a role in cognitive functioning. The brain may also benefit from neuroprotective effects of exercise, such as its reported ability to reduce oxidative stress (Cotman, Berchtold, & Christie, 2007) and to increase levels of growth factors, including brain-derived neurotrophic factor (BDNF; Knaepen, Goekint, Heyman, & Meeusen, 2010), which is critical for brain metabolism and neurogenesis (Jak, 2012). Physical activity may also be protective against neuronal loss with aging, as structural neuroimaging studies have also shown that older adults who exercise had less volume reduction in the brain compared to less active individuals (Colcombe, Erickson, Raz, Webb, Cohen, McAuley, *et al.*, 2003). Thus, the present findings are well aligned with prior works, which have shown that physical activity, particularly aerobic exercise, is associated with changes in brain structure and function that are associated with better cognitive performance.

The present results also showed that the relationship between physical exercise and measured intelligence was most evident for women compared to men. Prior studies have also suggested that the effects of physical

exercise on cognition appear to be most evident in female samples (Colcombe & Kramer, 2003; Middleton, *et al.*, 2008). A recent controlled trial of aerobic exercise in elderly adults showed that the benefits were primarily observed among women, particularly for cognitive variables involved in executive functions such as selective attention, processing speed, cognitive flexibility, and search efficiency (Baker, Frank, Foster-Schubert, Green, Wilkinson, McTiernan, *et al.*, 2010). While the basis for the sex differences in responses to exercise remain uncertain, some evidence suggests that it may be due in part to sex-related differences in the responses of glucometabolic and hypothalamic-pituitary-adrenal axis systems to aerobic physical activity (Baker, *et al.*, 2010). Specifically, a study by Baker and colleagues showed that a 6-month aerobic exercise program comprising four exercise sessions per week (45 to 60 minutes each) yielded significantly better improvement on executive function tasks for women, and this was associated with improvements in glucose disposal, insulin sensitivity, cortisol regulation, and plasma BDNF, which were all more improved for women compared to men (Baker, *et al.*, 2010). These findings suggest that women may show greater metabolic effects from exercise than men, which may contribute to its more prominent effects on cognition among females. Additionally, it is possible that exercise may have developmental effects on brain structure and function that differ between males and females. For instance, adolescent boys and girls differ in the magnitude of correlations between brain structure volumes and cognitive performance (Yurgelun-Todd, Killgore, & Young, 2002; Yurgelun-Todd, Killgore, & Cintron, 2003). Further research may need to explore how physical activity may play a role in these associations.

Most prior studies on the role of physical exercise in cognition have focused primarily on cognitive capacities such as executive functioning, attention, and processing speed, which are easily affected by state variables such as fatigue, mood, and sleep loss. Although fewer studies have focused on relatively stable cognitive capacities such as intelligence, evidence suggests that even these capacities are affected by physical exercise. One study of Grade 3 children found that a 30-min. physical activity period just three times a week during the school year was associated with improved academic achievement scores and higher scores on a measure of fluid intelligence (Reed, *et al.*, 2010). Similarly, a study of older patients with chronic obstructive pulmonary disease showed that engagement in a 3-month exercise program led to significant improvement on an index of fluid intelligence (Etnier & Berry, 2001). The present cross-sectional data are consistent with these prior findings, as greater levels of exercise were associated with higher measured full scale intelligence and performance IQ in women. The role of exercise on performance IQ suggests that these

motor and visuospatial capacities may be more amenable to the effects of physical activity than verbal abilities, or alternatively, that those with greater motor capacities are more likely to seek out physical activity.

The present findings should be considered in light of several methodological limitations. Because these data are cross-sectional and correlational in nature, it is not possible to infer the causal direction of the relationship between physical activity and intelligence. While considerable longitudinal evidence suggests that greater physical activity leads to positive improvement in cognitive functioning and brain health, alternative explanations for this association cannot be ruled out in the present study. For instance, it may be that individuals with greater intelligence are more likely to seek out physical exercise due to greater knowledge regarding the importance of exercise for general health. It is also well known that higher intellectual capacity is generally associated with greater financial income and upward mobility. Persons with greater financial resources are more likely to live in more affluent neighborhoods where it may be safer to exercise or where access to physical fitness facilities may be easier. The present data argue against these explanations, however, as the correlations between exercise and intelligence scores were just as high or higher after statistically removing the effects of socioeconomic, demographic, and cultural factors that might confound these relationships. Nonetheless, it is also possible that early socialization or income disparities between genders may contribute to the observed relationships in ways that were not controlled or evaluated. Future research would benefit from more specific control of these factors, as well as from the use of prospective longitudinal data collection in healthy adult samples. Additionally, only a limited set of self-report questions were asked regarding physical activity. Consequently, this analysis did not address the type of exercise (e.g., aerobic vs strength training) and the present data did not account for the duration of lifetime exercise or activity habits, each of which could also affect the data. With due consideration to the aforementioned limitations, the present findings suggest that there appear to be sex differences in the relation between physical activity and IQ. As described previously, some initial work suggests that such sex differences may be due to factors associated with the effects of exercise on glucoregulation and/or insulin sensitivity, but further research is needed to explore the potential mechanisms underlying these differences.

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A funny thing happened on the way to the scanner: humor detection correlates with gray matter volume

Maia Kipman, Mareen Weber, Zachary J. Schwab, Sophie R. DelDonno and William D. S. Killgore

The detection and appreciation of humor is a complex cognitive process that remains poorly understood. Although functional neuroimaging studies have begun to map the brain systems involved in humor appreciation, there are virtually no data on the structural correlates between gray matter volume and this capacity. Using voxel-based morphometry, the present study examined the association between gray matter volume and the ability to detect and appreciate humor. Fifty-nine healthy adults aged 18–45 years (30 men) underwent structural MRI and completed the University of Pennsylvania Humor Appreciation Test (HAT). After controlling for age and sex, gray matter volume of the left inferior frontal gyrus, left temporal pole, and left insula correlated positively with the appreciation of visual and verbal humor on the HAT, whereas the gray matter volume of the right inferior frontal gyrus correlated only with verbal humor appreciation scores. There were no negative correlations between gray matter volume and HAT performance. These data support a neurobiological basis for humor appreciation, particularly involving left-hemispheric cortical systems, and further suggest that individual differences in humor appreciation may be related to differences in regional gray matter volume. *NeuroReport* **23:1059–1064** © **2012 Wolters Kluwer Health | Lippincott Williams & Wilkins.**

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Introduction

The ability to detect and appreciate humor is an extraordinarily complex and sophisticated cognitive process [1]. Neuroevolutionary theories posit that humor developed as a false inference detection mechanism [2]. This perspective suggests that as humans evolved to think more creatively and solve increasingly complex problems in novel ways, it became advantageous to utilize mental shortcuts, or heuristics, in lieu of fully assimilating and comprehensively analyzing large amounts of data about the environment [3]. By definition, however, heuristics are crude approximations of reality and sometimes lead to incorrect solutions. To help avoid the pitfalls associated with false inferences, a system of error detection appears to have developed that rewards individuals for error checking. This system is hypothesized to be our sense of humor [3]. In this context, humor, or the experience of finding something is 'funny,' can be regarded as an affectively based cognition that involves deriving pleasure from the reconciliation of an apparent discrepancy or an unexpected insight into the cause of an anomaly. Laughter and smiling, from this perspective, can be thought of as methods for communicating false alarms in inference, indicating to conspecifics that there is no danger in the situation [2]. Thus, the primary components of humor include false inference detection, incongruity resolution, mirth production, reward, and social communication.

Although the neurobiological basis underlying humor appreciation remains largely unexplored, recent functional neuroimaging studies have begun to map the systems that contribute to this capacity. Functional MRI (fMRI) studies evaluating the funniness of jokes have found that activation of the anterior cingulate cortex (ACC) correlates with increased ratings of funniness [4,5], consistent with its role in error detection [6]. Greater ACC activation was also found for funny compared with unfunny stimuli irrespective of whether participants had to actually rate funniness [4,7,8]. In addition, salience-detection processes involving the amygdala alert the organism to potential danger, threat, or emotional consequences associated with an ambiguous situation or potential error [9]. Accordingly, several functional MRI studies have found greater amygdala activation in response to funny compared with unfunny stimuli [7,8,10,11].

Humor appreciation also appears to involve several higher order cognitive and social processing systems. For example, one study using event-related fMRI to separate detection, comprehension, and mirth of humor specifically found that the inferior frontal gyrus (IFG) was activated during the process of integrating new information from the environment to resolve or clarify discrepancies [10]. This finding is congruent with previous reports on both humans and primates that implicate the

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Fig. 1

Examples of the Pennsylvania Humor Appreciation Test (HAT) visual (left) and verbal (right) stimuli. These stimuli are from G. Mordillo and modified with the permission of his agent. Reprinted with permission from Ruben Gur, PhD. Copyright, Trustees of the University of Pennsylvania.

IFG in the resolution of ambiguous information [12–14]. Furthermore, humor appreciation may also involve a social evaluation component, as several fMRI studies [11,15] have found activation in the temporal pole during the process of deciphering jokes that involve social scenarios or that require the ability to infer the thoughts or emotions of others. Finally, several studies have suggested that the insular cortex shows an increase in functional activation once a joke is comprehended, suggesting that the insula may play a role in the somatic–emotional sensations of mirth that contribute to humor appreciation [7,10,16]. These studies collectively illustrate a plausible humor-processing network involving the ACC, amygdala, IFG, temporal pole, and insula that contributes to the ability to detect and comprehend a joke or other humorous situation.

Although a number of studies have investigated the functional regions involved in humor appreciation, to our knowledge, no study has directly mapped this capacity to gray matter volume within the hypothesized neurocircuitry outlined above. Thus, whereas it is currently known that differences in the ability to detect and appreciate humor are reflected in functional brain activation, it remains to be shown whether the same applies for morphological differences. In the present study, we therefore set out to identify the morphological correlates between humor appreciation ability and gray matter volume using voxel-based morphometry (VBM) in healthy individuals. On the basis of the previously reviewed literature, we predicted that performance on a test of humor appreciation would be correlated positively with greater gray matter volume (GMV) in a network comprising the ACC, amygdala, insula, IFG, and temporal pole.

Methods

Participants

Fifty-nine right-handed, healthy, native English-speaking adults (mean age 30.6±8.1 years, range 18–45; 30 men, 29 women) were recruited from the Boston metropolitan area through posted flyers and internet advertisements. By means of a detailed screening interview, all participants were determined to be free from any history of neurological, psychiatric, alcohol, or substance use disorders. Participants were compensated for their time. This research was approved by the McLean Hospital Institutional Review Board and was conducted in accordance with the 1964 Declaration of Helsinki. All participants provided written informed consent before participation.

Materials and procedure

Participants completed the computerized Pennsylvania Humor Appreciation Test (HAT) [17], which was administered outside of the MRI scanner. The HAT presents the participant with 40 pairs of stimuli (20 verbal, 20 visual) in a sequential pseudorandom order. The visual scenarios comprised 20 pairs of nearly identical nonverbal cartoons and the verbal scenarios comprised 20 pairs of nearly identical fictitious newspaper headlines (see Fig. 1 for an example). In each pair, one stimulus differed subtly from its counterpart by virtue of some contextual incongruity or alternative interpretation, making it humorous, whereas the other stimulus was not designed to be funny. By forced-choice button press, participants selected the funnier item of each pair or indicated that the two stimuli were equally funny. Time to respond was not restricted. Test performance was scored by summing the number of correct responses for the verbal and visual sections and for

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the total HAT. Higher HAT scores indicate greater capacity to detect and appreciate humor in the stimuli.

Magnetic resonance imaging parameters

Structural images were collected at 3.0 T using a Siemens Tim Trio scanner (Erlangen, Germany) equipped with a 12-channel head coil. Volumetric data were acquired with a T1-weighted 3D MPRAGE sequence (repetition time/echo time/flip angle = 2.1 s/2.25 ms/12°), 128 sagittal slices (256 × 256 matrix). The in-plane resolution was 1 × 1 mm with a slice thickness of 1.33 mm.

Voxel-based morphometry

Image analysis was carried out on SPM8 (Wellcome Department of Imaging Neuroscience Group, London, UK; *http://www.fil.ion.ucl.ac.uk/spm*) using the VBM8 toolbox (*http://dbm.neuro.uni-jena.de/vbm.html*). For preprocessing, the default settings in the VBM8 toolbox for modulated VBM were used (i.e. GMV was corrected for the total brain volume). For each participant, the T1-weighted structural images were first DARTEL-normalized to match the Montreal Neurological Institute (MNI) template. Using a fully automated algorithm, each image was segmented into gray matter, white matter, and cerebrospinal fluid. For spatial smoothing, an 8 mm full-width at half-maximum isotropic Gaussian kernel was applied to the normalized gray matter images.

Statistical analysis

In SPM8, the normalized smoothed gray matter images were entered into three separate random-effects multiple regression analyses to predict GMV from the overall HAT sum score as well as from the HAT headline and HAT cartoon subscale scores, respectively. On the basis of previous fMRI findings specifying the functional neurocircuitry involved in humor appreciation [8,18], we solely carried out a region-of-interest (ROI) analysis of the bilateral insula, amygdala, IFG, ACC, and temporal pole regions. The ROI anatomical mask was created using the Automated Anatomical Labeling Atlas [19] as part of the Wake Forest University PickAtlas Utility for SPM [20]. Thus, the insula included gray matter internal to its circular sulcus. Similarly, the precentral sulcus defined caudal limits of the IFG, whereas dorsal and rostral region borders were defined by the inferior frontal sulcus. The corpus callosum and the paracingulate sulcus represented the caudal and rostral borders to the ACC. Finally, the temporal pole was limited by the anterior part of the superior temporal gyrus and thus included portions of both the superior and the middle temporal gyri anterior to the anterior commissure. For a more comprehensive description of the ROI borders, the reader is referred to the published atlas [19]. Each ROI analysis was thresholded at P less than 0.001, uncorrected, with an empirically defined cluster extent threshold defined as the number of voxels expected per cluster in each analysis (i.e. 90) derived from the SPM8 output. Age and sex were controlled as nuisance covariates in each regression model.

Results

The mean HAT total score was 29.23 (SD = 7.24), the mean HAT visual score was 14.48 (SD = 3.81), and the mean HAT verbal score was 14.76 (SD = 4.14). Higher HAT scores indicate greater capacity to detect and appreciate humor in the stimuli. The HAT total score correlated positively with GMV in two clusters. The first cluster encompassed the left insula and IFG pars opercularis and pars triangularis (1128 voxels, $T = 4.89$, MNI coordinates: $x = -38$, $y = 11$, $z = -3$; Fig. 2a), whereas the second included the left inferior orbitofrontal gyrus and the left superior temporal pole (414 voxels, $T = 3.95$, MNI coordinates: $x = -46$, $y = 21$, $z = -12$; Fig. 2b). The HAT visual score correlated positively with GMV in a cluster comprising the left superior temporal pole, the left IFG pars opercularis, and the left insula (424 voxels, $T = 4.78$, MNI coordinates: $x = -32$, $y = 15$, $z = -23$; Fig. 2c). The HAT verbal score correlated positively with GMV of three clusters, including (i) the left insula and left IFG pars triangularis and pars opercularis (1039 voxels, $T = 4.71$, MNI coordinates: $x = -36$, $y = 12$, $z = -2$; Fig. 2d); (ii) the left orbital IFG and left superior temporal pole (178 voxels, $T = 3.99$, MNI coordinates: $x = -46$, $y = 30$, $z = -11$; Fig. 2e); and (iii) the orbital portion of the right IFG (118 voxels, $T = 3.61$, MNI coordinates: $x = 50$, $y = 26$, $z = -12$; Fig. 2f). There were no significant negative correlations within any of the ROIs.

Discussion

The ability to detect and appreciate humor, as reflected in HAT performance, was associated with GMV in three brain regions that have also been implicated in humor processing using fMRI [8,11]. These regions included the left IFG, an area known to engage in the resolution of incongruity in general [12–14] and of humorous stimuli in particular [5,8,11,15,21–23], the left temporal pole, a structure important for social and emotional problem solving [24], and the left insula, which has been implicated in the feeling of mirth during humor [7,10,16]. For all three brain regions, greater humor detection ability was associated with greater GMV. These structural volumetric findings comport with previous fMRI research showing greater activation in these areas in response to funny versus unfunny stimuli [8,10]. Furthermore, our data suggest that individual differences in humor appreciation may be at least partially related to trait-like differences in morphology between individuals. Importantly, GMV of these structures was predicted similarly by the HAT visual as well as the HAT verbal score, suggesting that these structure–function relationships exist independent of humor presentation modality. Interestingly, however, the HAT verbal score also uniquely predicted GMV of the right IFG, raising the possibility

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Fig. 2

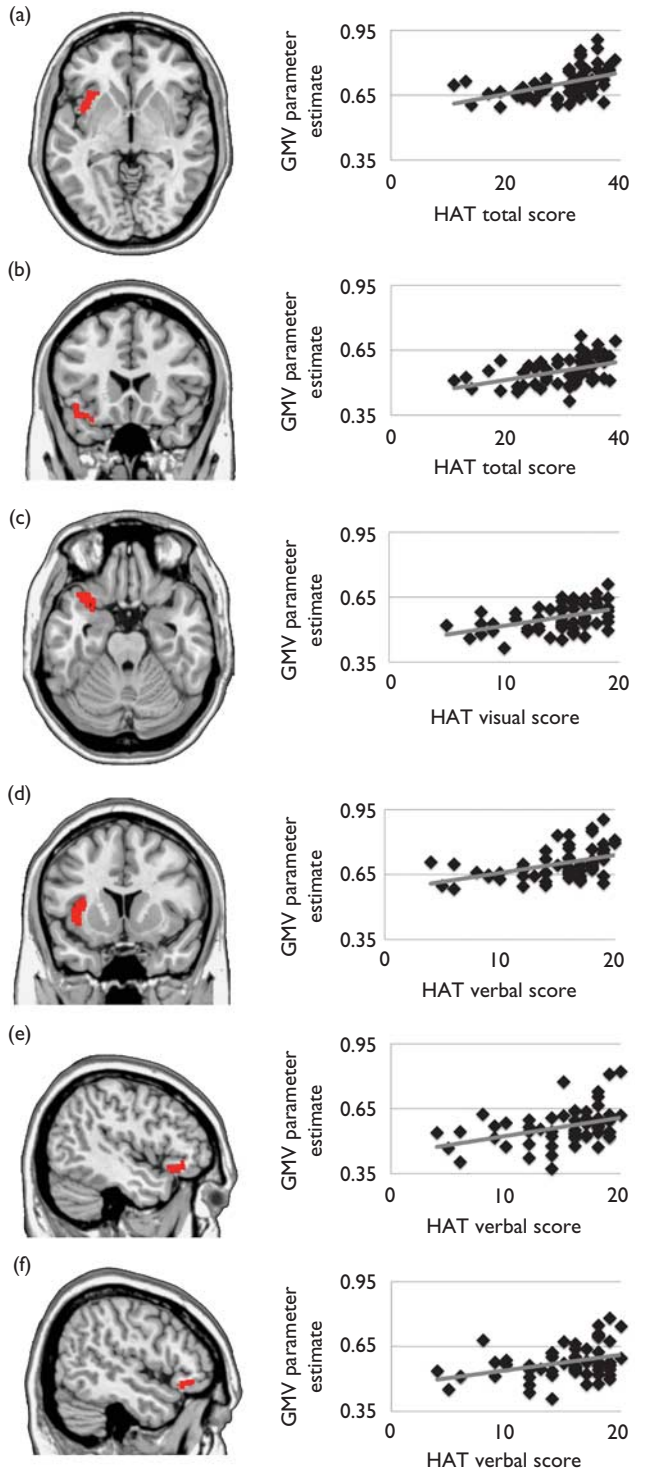

Fig. 2

Brain regions with significant positive correlations between gray matter volume and Humor Appreciation Test (HAT) scores superimposed on single-subject T1-weighted structural images and corresponding scatterplots. (a) Axial view of the left insula and inferior frontal gyrus regions that correlate positively with the HAT total score. (b) Coronal view of the left inferior orbitofrontal gyrus and left temporal pole regions that correlate positively with the HAT total score. (c) Axial view of the left superior temporal pole, left inferior frontal gyrus, and left insular regions that correlate positively with the HAT visual score. (d) Coronal view of the left insula and left inferior frontal gyrus regions that correlate positively with the HAT verbal score. (e) Sagittal view of the left inferior frontal gyrus and left superior temporal pole regions that correlate positively with the HAT verbal score. (f) Sagittal view of the right inferior frontal gyrus regions that correlate positively with the HAT verbal score. GMV, gray matter volume.

that this region may also play an important role in some of the unique processes involved in detecting or appreciating the humor of verbal stimuli with multiple meanings (i.e. double entendre).

Consistent with some previous fMRI research [7,8,10, 11,15,23], our data show a predominant left lateralization of most of the structure–function associations involved in humor appreciation, particularly for the insula and the temporal pole regions. This pattern is intriguing and raises the possibility that the neurocircuitry of humor appreciation may rely largely on language systems [21] and possibly also emotion-processing streams [25], even when the modality of presentation involves nonverbal cartoon images. A previous event-related fMRI study showed left IFG activation during the humor detection phase and bilateral insula activation during the experience of mirth, or the appreciation phase, of watching humorous video clips [10]. Because the present study only examined detection/appreciation performance as a variable of interest, it is impossible to distinguish between the detection and mirth experience phases of the task, and both are likely to be necessary components for successful humor appreciation.

The neuroevolutionary inference error detection theory suggests that a major component of humor appreciation is the ability to notice incongruities, errors, or discrepancies, and orient the organism toward their resolution [2]. Functional neuroimaging has suggested that the ACC and amygdala are predominantly involved in these error detection and alerting responses [8,10]. Contrary to this theory, however, we did not find the volume of either of these structures to be related to HAT performance. Of course, larger structural volume does not necessarily imply better function, and humor appreciation may be too subtle a capacity to be related to volumetric differences in these structures. Alternatively, it is possible that the simple forced-choice nature of the task itself was too gross a metric to draw heavily upon error detection or too artificial a situation to require vigilance to potential environmental threat, thus minimizing the role of these factors. This null finding might have been further influenced by the rather conservatively set cluster extent threshold (i.e. $k \geq 90$), meaning that small structures such as critical affect-related subnuclei of the amygdala were less likely to survive the analysis. Future research

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might benefit from more sensitive and ecologically valid paradigms within a more naturalistic setting, such as watching humorous television episodes or stand-up comedy routines, to map the relationship between humor detection and GMV.

For the present study, we focused specifically on only a limited number of brain regions. The HAT specifically assesses humor detection ability as opposed to the participant's experience of reward. Therefore, for the purposes of the present study, we did not place ROIs in dopaminergic structures such as the nucleus accumbens and ventral striatum, even though functional activation in these areas has been demonstrated in the humor literature [7,8,11,16,22]. Future work might include scaled funniness ratings in addition to forced-choice funniness decisions to explore these relationships more comprehensively. Although potentially relevant, we also did not record behavioral aspects of humor reactions, such as the amount of participant laughter or smiling in response to the material presented in the HAT. Therefore, in the present study, we did not place ROIs in motor response regions such as the supplementary motor area or the cerebellum, which are believed to contribute to the physical communication of humor [8,10,21]. Incorporating psychophysiological measures of humor expression in response to HAT stimuli and exploring other reward-related brain regions might help answer the question of whether GMV within these networks might be related to the extent to which an individual communicates an affective humor response, irrespective of the stimuli's subjective funniness.

Several caveats should be kept in mind when evaluating these findings. First, because this is the first study to use VBM to predict GMV from a humor detection task, our data should be considered as preliminary and in need of independent replication. Second, it is important to consider the limitations of the specific task used. Notably, because of its artificial nature, the HAT is somewhat removed from naturalistic and more spontaneous settings in which humor and its appreciation typically occur. However, the HAT task was used for this initial investigation because it allows for a very controlled assessment to isolate and examine humor detection and appreciation. In addition, no data were collected examining the extent of amusement or pleasure produced by the stimuli. This may be a key element in the process of humor appreciation and should be investigated further. Of additional interest may be sex differences in humor appreciation, which were not investigated in the current study. Finally, future research should focus on structural and functional connectivity among areas of the humor detection network to determine whether the strength of connectivity is related to humor detection ability or to the subjective experience of reward or amusement to humorous material.

Conclusion

The ability to detect and appreciate humor, irrespective of whether assessed in the verbal or the visual modality, was associated with greater GMV in several brain regions involved in higher order cognitive processing, social inference, and somatic–emotional processing. Our data are concordant with a number of functional neuroimaging studies and suggest that the capacity of humor appreciation is related to distinct morphological differences in the brain structures comprising this complex system.

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Conflicts of interest

There are no conflicts of interest.

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Daytime sleepiness affects prefrontal regulation of food intake ☆

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ABSTRACT

The recent epidemic of obesity corresponds closely with the decline in the average number of hours of sleep obtained nightly. While growing research suggests that sleep loss may affect hormonal and other physiological systems related to food intake, no studies have yet explored the role that sleepiness may play in reducing prefrontal inhibitory control over food intake. Because evidence suggests that women may be more prone to obesity and eating disorders, as well as more likely to suffer from sleep problems, we examined the relation between general daytime sleepiness, brain responses to food stimuli, and self-reported overeating separately for men and women. Thirty-eight healthy adults (16 women; 22 men) aged 18 to 45 underwent functional magnetic resonance imaging (fMRI) while viewing pictures of high- and low-calorie foods. Subjects completed the Epworth Sleepiness Scale (ESS) and provided a rating to the query "how often do you eat more than you intend to." Contrast images comparing brain activation derived from the high- versus low-calorie conditions were correlated voxel-wise with scores from the ESS in a second-level regression model, the output of which was used to predict self-reported overeating. As hypothesized, daytime sleepiness correlated with reduced activation in the ventromedial prefrontal cortex during perception of high- versus low-calorie food images. Moreover, activation within this cluster predicted overeating, but only for women. Findings suggest that normal fluctuations in sleepiness may be sufficient to affect brain regions important for regulating food intake, but that these effects may differ between men and women.

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Introduction

Over the past several decades there has been an alarming increase in the rate of excessive weight gain in Western societies (Flegal et al., 2002), with over one in three adults in the United States now meeting criteria for obesity (Ogden et al., 2012). While there are many factors that have arguably contributed to this trend, it is hard to ignore the fact that obesity rates have closely paralleled the decline in average nightly sleep during the latter portion of the 20th century. Evidence suggests that during the 1960s, Americans were sleeping between 8.0 and 8.9 h per night (Kripke et al., 1979). By the mid 1990s, average sleep had declined to about 7.0 h (Gallup Organization, 1995), and recent data from 2005 suggest that most Americans are now sleeping less than 7 h per night (National Sleep Foundation, 2005). In fact, a 2012 report by the Center for Disease Control and Prevention (CDC) found that one in three workers now report that they routinely sleep six or fewer hours nightly (Center for Disease Control and Prevention, 2012). Shorter sleep duration is related to a variety of health problems including obesity (Patel et al., 2008). Moreover, short sleep duration earlier in life is related to increased risk of weight gain later in life (Gangwisch et al., 2005; Hasler et al., 2004; Patel, 2009). The relation between sleep and weight gain is poorly understood, but may prove crucial to stopping or even reversing the current trends.

Notably, the epidemic of obesity has particularly affected women. Epidemiological studies suggest that for the past few decades, women have shown significantly higher rates of obesity compared to men (Ogden et al., 2012), and extreme levels of obesity (i.e., Body Mass Index > 40) are more than twice as prevalent among women than men (Ogden et al., 2006). It has also long been known that women tend to be at much greater risk for developing a number of different eating related problems and clinical eating disorders relative to men (Lewinsohn et al., 2002; Striegel-Moore and Bulik, 2007; Striegel-Moore et al., 2009). While the reasons for the gender differences in eating disorders are not fully understood, some evidence suggests that there may be some cognitive and behavioral differences in responses to food, with women more frequently reporting a greater perception of loss of control over the amount of food consumed during meals (Striegel-Moore et al., 2009). Functional neuroimaging studies have also suggested that there may be sex differences in responses of key appetite regions to images of food (Killgore and Yurgelun-Todd, 2010). Interestingly, the reported sex differences in food consumption

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also appear to be mirrored in the frequency of some sleep-related complaints and disorders. For instance, a recent meta-analysis of studies of insomnia showed that women were over 1.4 times more likely to suffer from insomnia compared to men (Zhang and Wing, 2006). Although sleep apnea is more common among middle to older age men (O'Connor et al., 2000), general non-respiratory sleep complaints such as poor sleep quality, longer sleep onset latency, and difficulty with sleep maintenance tend to be more common among women in the same age range (Middelkoop et al., 1996). A recent poll by the National Sleep Foundation reported that 67% of women experience sleep problems at least a few nights each week and 46% report that they suffer from sleep problems every night (National Sleep Foundation, 2007). Thus, over the past few decades, sleep duration has declined while obesity has increased, and these problems appear to be particularly common among women.

Most studies linking insufficient sleep to excess food consumption and weight gain have emphasized the effects of sleep loss on physiological variables such as reduced energy expenditure and alterations in the hormones leptin and ghrelin, which are key regulators of appetite (Knutson et al., 2007; Patel and Hu, 2008). However, lack of sleep can affect other systems that play a role in food intake as well. For instance, sleep loss is associated with altered functional activity within a number of brain regions. Of particular relevance to food consumption is the prefrontal cortex, particularly the ventromedial prefrontal cortex (vmPFC), a complex brain region that is particularly important for evaluating the reward value of objects (Paulus and Frank, 2003), regulating emotional responses (Hariri et al., 2003), and controlling behavior (Blasi et al., 2006; Ridderinkhof et al., 2004). Sleep loss is associated with a number of changes in the vmPFC, including reduced glucose metabolism (Thomas et al., 2000; Wu et al., 2006), altered functional responses during risky decision-making (Venkatraman et al., 2007) and judgments of economic value (Libedinsky et al., 2011; Venkatraman et al., 2011), as well as reduced functional connectivity with other brain regions important for self-referential and emotional processing (De Havas et al., 2012; Killgore et al., 2012c; Samann et al., 2010; Yoo et al., 2007). When sleep is lacking, these prefrontal changes appear to contribute to deficits in decision-making and inhibitory control (Drummond et al., 2006; Harrison and Horne, 2000; Killgore, 2010). Interestingly, recent data suggest that general daytime sleepiness is associated with reduced gray matter volume within the vmPFC (Killgore et al., 2012b). Some of these same sleep-sensitive prefrontal systems have previously been shown to be critical in responding to the caloric content of visually presented food stimuli (Killgore and Yurgelun-Todd, 2005b; Killgore et al., 2003) and may even relate to greater body mass index (BMI) (Killgore and Yurgelun-Todd, 2005a). Thus, evidence suggests that insufficient sleep alters functioning in key brain regions that are particularly responsive to the caloric content of food and which are important for regulating and inhibiting behavior.

The goal of the present study was to examine the relation between self-reported general daytime sleepiness and prefrontal cortex responses to the calorie content of food images. Based on the neuroimaging and behavioral literature outlined above, we hypothesized that greater general daytime sleepiness would be associated with reduced functional responsiveness of the vmPFC to high- versus low-calorie food images, and that the magnitude of responsiveness within this inhibitory region would predict self-reported problems with overeating. Furthermore, given the sex differences in the current rates of obesity, eating disorders, and sleep complaints, we also hypothesized that the relationships would be stronger in women than in men.

Methods

Participants

Thirty-eight healthy right-handed adults, ranging in age from 18 to 45 years (16 women, 22 men), were recruited via flyers and internet advertisements posted around Boston, MA, and the surrounding areas. Participants were thoroughly screened by a trained research technician during a semi-structured interview. Based on this screening, enrolled participants were deemed to be free of any evidence or history of severe medical conditions, head injury, loss of consciousness >30 min, brain tumors, seizures, neurologic conditions, symptoms consistent with Axis I psychopathology, or drug or alcohol treatment. Additionally, potential participants were excluded for current or recent use of any psychoactive medications or illicit substances, or excessive alcohol intake. Table 1 provides basic demographic information for the women and men separately. Body mass index (BMI) ranged from normal (19.80) to moderately obese (34.78) for the sample as a whole (M = 24.60, SD = 3.75), but this did not differ between women and men (see Table 1). Each participant completed detailed logs of all food consumed on the day of the scan. Two independent raters used the food logs to calculate each participant's calorie consumption during the hours preceding the scan via a primary web-based resource for determining calorie content from foods (http://ndb.nal.usda.gov), and relied on a secondary resource when a definitive answer could not be obtained from the first (http://caloriecount.about.com). Inter-rater reliability in calorie scoring was extremely high (ICC = 0.97, CI = 0.95–0.98), and the independent ratings were averaged for each participant to obtain a final estimate of total calorie consumption. On the whole, participants consumed an average of 327.8 calories (SD = 243.6) during the hours leading up to the scan, with no significant difference between women and men in calorie intake (see Table 1). Overall, typical caffeine use was modest, ranging from 0 to 444 mg per day (M = 104.08, SD = 117.65), and did not differ between women and men. Similarly, caffeine use on the day of the scan was not significantly different for the women and men. Participants reported generally normal amounts of weeknight (M = 7.36, SD = 0.88 h) and weekend sleep (M = 7.71, SD = 1.32 h), as well as normal amounts of sleep the night before the scan (M = 7.04,

Table 1
Demographic and performance variable information for participants.

	Men (n = 22)		Women (n = 16)			
	M	SD	M	SD	t (df = 36)	sig.
Age (years)	31.50	9.30	28.25	7.48	1.19	ns
BMI (weight [kg]/height [m]^2)	24.24	3.60	25.08	4.01	−0.68	ns
Pre-scan calories consumed	358.8	236.7	285.1	254.1	0.92	ns
Typical caffeine use (mg/day)	101.94	127.48	107.02	106.64	−0.13	ns
Study day caffeine use (mg)	73.96	122.17	88.91	106.27	−0.39	ns
Weeknight sleep (h)	7.36	0.94	7.36	0.81	0.02	ns
Weekend sleep (h)	7.82	1.31	7.56	1.38	0.58	ns
Last night sleep (h)	6.91	0.97	7.22	0.93	−0.99	ns
ESS	5.77	3.74	5.31	3.18	0.40	ns
Current hunger (1–7)	4.64	1.33	5.00	1.41	−0.81	ns
Typical appetite (1–10)	6.18	1.40	6.63	1.45	−0.95	ns
Eat more than intend to (1–10)	3.55	2.61	5.06	2.14	−1.90	ns
Flower picture ratings	1.00	0.16	1.01	0.04	−0.80	ns
Low-calorie picture ratings	3.63	1.35	3.83	1.36	−0.44	ns
High-calorie picture ratings	4.33	1.26	4.13	1.19	0.49	ns
Low-calorie picture memory	0.75	0.15	0.80	0.07	−1.19	ns
High-calorie picture memory	0.77	0.14	0.84	0.10	−1.76	ns

The table shows that there are no differences between men and women on demographic and performance variables. BMI = Body Mass Index; ESS = Epworth Sleepiness Scale; Current Hunger was rated on a 7-point scale (1 = not at all hungry; 7 = extremely hungry); Typical Appetite was rated on a 10-point scale (1 = never hungry; 10 = always hungry); Eat More than Intend to was rated on a 10-point scale (1 = never; 10 = always). Ratings refer to post-scan ratings taken for each image shown in the scanner. Participants responded to the question "how much you would like to eat each item right now" (1 = do not want to eat it; 7 = strongly desire to eat it). Memory scores indicate the proportion of correct recognition responses for each category of images (i.e., previously seen versus new foils) shown during the post-scan recognition test.

SD = 0.95 h). No sex differences were observed on any of these variables (see Table 1). All participants provided written informed consent prior to enrollment and were compensated for their time. This research study was approved by the McLean Hospital Institutional Review Board.

Materials and procedure

Each participant arrived for the study and underwent informed consent between 9:00 and 11:00 a.m. For the remainder of the morning, participants completed several self-report inventories, including questionnaires about demographic information, sleep habits, recent sleep, caffeine use, dietary intake, and appetite. In particular, participants were asked to respond to the query "what is your appetite like?" on a 10-point scale (*Appetite*: 1 = never hungry; 10 = always hungry), and to respond to the query "do you feel you eat more than you intend to" on a 10-point scale (*Overeating*: 1 = never; 10 = always). Participants also completed the Epworth Sleepiness Scale (ESS) (Johns, 1991), a self-report measure of general daytime sleepiness. The ESS is the most widely used self-report measure of general subjective sleepiness in the world (Drake, 2011) and shows high internal consistency reliability (Cronbach's alpha = 0.88) as well as high test-retest reliability ($r = 0.82$) over five months in healthy individuals (Johns, 1992). This scale requires the respondent to rate their chance of falling asleep in eight different situations (e.g. sitting and reading; as a passenger in a car for an hour without a break) in recent times along a 4-point scale (0 = would never doze, 1 = slight chance of dozing, 2 = moderate chance of dozing, 3 = high chance of dozing). Responses are summed to provide a total score (maximum: 24). Higher scores indicate greater general daytime sleepiness, and scores greater than 10 reflect excessive daytime sleepiness in the clinically significant range. Thus, the scale measures a general level of chronic propensity to fall asleep across a variety of settings rather than the acute level of sleepiness that may be measured with other state indices. This may allow greater unmasking of chronic sleep debt by focusing on the broader behavioral propensity for sleep than other measures of acute sleepiness (Pilcher et al., 2003). Participants were not restricted in their food consumption throughout the morning, but were required to document all intake for the day on a dietary log. No food was consumed within an hour prior to the functional neuroimaging scans.

Between 12:30 and 3:00 in the afternoon, participants underwent functional magnetic resonance imaging (fMRI) while completing a food perception task (FPT). The present task was a slightly modified version of the same task we have used in our prior work (Killgore and Yurgelun-Todd, 2005a,b, 2006, 2007, 2010; Killgore et al., 2003, 2010). During the FPT, participants viewed a series of 30-second blocks of images depicting high (H) calorie foods (e.g., cheeseburgers, French fries, cake, ice cream, candy), low calorie (L) foods (e.g., fresh salads, fruits, vegetables, fresh fish, whole grain bread), or control (C) images (i.e., non-edible rocks, flowers, shrubs). During each block, ten images were shown for three seconds each. The task included 15 s of resting fixation (+) at the beginning and end of the scan. The total duration of the FPT was 240 s and followed a constant presentation order (+, C, L, H, C, H, L, C, +) (see Supplementary Fig. 1). Participants were given the following instruction: "For this task, you will see a series of photographs. Try your best to remember the photographs, because your memory for the pictures will be tested after the scan." At the conclusion of the scan, participants completed a recognition task that presented all of the previously seen food items along with an equal number of distractor items. Following the recognition task, participants completed a 7-point rating scale to indicate their current level of hunger (i.e., 1 = not at all hungry; 7 = extremely hungry), and were then shown all of the previously seen food and control images. For each image, they were asked to rate "how much you would like to eat each item right now" (1 = do not want to eat it; 7 = strongly desire to eat it).

Magnetic resonance imaging parameters

Participants were scanned using a 3.0 Tesla SIEMENS Tim Trio scanner and a 12-channel head coil. At the outset, structural MRI scans were collected using a T1-weighted 3D MPRAGE sequence (TR/TE/flip angle = 2.1 s/2.25 ms/12°) over 128 sagittal slices (256 × 256 matrix) and a slice thickness of 1.33 mm, yielding a voxel size of 1 × 1 × 1.33 mm. During the FPT, a 4-minute blood oxygenation level dependent (BOLD) fMRI was acquired over 43 transverse interleaved slices using a T2*-weighted echo planar imaging sequence (TR/TE/flip angle = 3.0 s/30 ms/90°), with 80 images per slice (3.5 mm thickness, no skip; 22.4 cm field of view; 64 × 64 acquisition matrix), yielding a voxel size of 3.5 × 3.5 × 3.5 mm.

Image processing

The functional data were pre-processed and analyzed using standard algorithms in SPM5 (Wellcome Department of Cognitive Neurology, London, UK). For each subject, the time series of images was spatially realigned and motion corrected, co-registered to the individual's own anatomical image, spatially normalized to fit the template of the Montreal Neurological Institute (MNI), spatially smoothed using an isotropic Gaussian kernel (full width at half maximum [FWHM] = 6 mm), and resliced to 2 × 2 × 2 mm. Finally, the time series was also convolved with the canonical hemodynamic response function in SPM5, the effects of serial autocorrelation were removed using the first-level autoregressive model, and a high-pass filter of 128 s was used to remove low frequency drift in the signal.

Statistical analysis

The analysis proceeded in several stages. First, sex differences on questionnaire and behavioral indices were examined via independent samples t-tests. Next, zero-order correlations between behavioral variables, including total ESS, appetite, and overeating were examined for the sample as a whole and separately for women and men. Gender comparisons of correlations were undertaken with Fisher's r-to-z-transformation with comparison to a directional z-distribution. Third, the functional neuroimaging data were analyzed in a multi-stage process. At the initial stage, the primary effect of the calorie conditions within the food perception task was determined. This entailed constructing a general linear model for each individual that contrasted the difference in BOLD response between the high calorie and low calorie food conditions (see Supplementary Fig. 1). These contrast images were then taken to a second level random effects analysis via the multiple linear regression module of SPM5 to examine the relationship between total ESS scores and greater responsiveness of the brain to the high calorie condition of the FPT within the vmPFC. Based on our *a priori* hypotheses derived from previous research on the effects of sleep loss on brain function, we restricted the primary analyses to two bilateral search territories within a subregion of the ventromedial prefrontal cortex (i.e., left and right gyrus rectus) defined by the Automated Anatomical Labeling Atlas (Tzourio-Mazoyer et al., 2002) and implemented via the Wake Forest University PickAtlas Utility (Maldjian et al., 2003) as a toolbox in SPM5. Dilation factor for the utility was set to 0. Correlations were initially thresholded at $p < .001$, k (extent) ≥ 10 contiguous voxels, and then subjected to small volume correction for multiple comparisons within each search territory at $p < .05$, corrected for false discovery rate (FDR). Finally, to evaluate the relationship between sleepiness-related brain activation and eating behavior, mean signal intensity was extracted from each significant cluster of activation during the FPT and correlated with behavioral indices, including ratings for the items querying about typical appetite and eating more than intended. These relationships were examined separately for women and men. Differences in the magnitude of correlation coefficients between men

and women were evaluated via Fisher's r-to-z transformation against a directional z-distribution.

Results

Sex differences in questionnaire and eating behavioral indices

As evident in Table 1, women and men showed no significant differences in demographic, sleep, or appetite variables. Critically, there also was no sex difference in calories consumed on the day of the scan. During neuroimaging, both groups attended well to the task, with men ($M = 76\%$ correct, $SD = 14\%$; $t(21) = 8.61$, $p < .001$) and women ($M = 82\%$ correct, $SD = 8\%$; $t(21) = 16.74$, $p < .001$) scoring significantly above chance in their recognition of the previously seen food items after the scan.

Correlations between sleepiness and self-reported appetite/overeating behavior

Simple whole brain contrasts between food and control conditions and between High and Low Calorie conditions are presented in the online Supplementary Results (see Supplemental Fig. 2, and Supplemental Table 1 and 2). For the sample as a whole, ESS scores were positively correlated with typical appetite ratings ($r = .53$, $p = .001$), suggesting that greater general daytime sleepiness was associated with greater appetite. As evident in Fig. 1, this was true for men ($r = .44$, $p = .038$), but was nonsignificantly stronger for women ($r = .72$, $p = .002$)($z = -1.21$, $p = .11$). In contrast, ESS was not significantly correlated with the tendency to overeat ($r = .18$, $p = .29$), which did not differ for men ($r = .13$, $p = .58$) or women ($r = .37$, $p = .16$) ($z = -0.72$, $p = .24$). Similarly, ESS was not significantly correlated with current hunger at the time of the scan ($r = .24$, $p = .14$), and these nonsignificant relations were similar for men ($r = .23$, $p = .30$) and women ($r = .30$, $p = .26$) ($z = -0.21$, $p = .42$).

Correlations between sleepiness and brain responses

The correlation between ESS and brain responses to the High > Low Calorie contrast was evaluated for the entire sample as a whole. Within the search territories investigated, there were no regions that were significantly positively correlated with general daytime sleepiness scores. There was, however, a single cluster within the vmPFC that emerged as significantly negatively correlated with ESS scores. Fig. 2 shows the location of this cluster within the left gyrus rectus (MNI: x = −8, y = 40, z = −20; $k = 18$ voxels; $p = .047$ FDR small volume corrected). No other regions within the search territories were significantly correlated with ESS. To ensure that our findings were not driven by observations with extreme influence, we examined standardized residuals and leverage values. No standardized residuals exceeded 3.0, suggesting that none of the observations were extreme outliers. One observation exceeded threshold for leverage (i.e., $2(k+1)/n > 0.105$, where k = the number of predictors and n = sample size). With this observation removed, the correlation between the ESS and extracted parameter estimates for the left gyrus rectus cluster remained significant at $p < .01$. Furthermore, to determine whether this finding was influenced by current hunger or previous food ingestion, we correlated the mean signal intensity values of this region with these scores. Activation in this cluster was not correlated with hunger for the sample as a whole ($r = -.05$, $p = .79$), and this was true for both men ($r = -.10$, $p = .65$) and women ($r = .06$, $p = .84$) ($z = -0.45$, $p = .33$). Similarly, mean cluster activation was not significantly related to prior calorie consumption for the sample as a whole ($r = -.12$, $p = .46$), a finding that was similar among men ($r = -.13$, $p = .55$) and women ($r = -.04$, $p = .90$) ($z = -0.25$, $p = .40$).

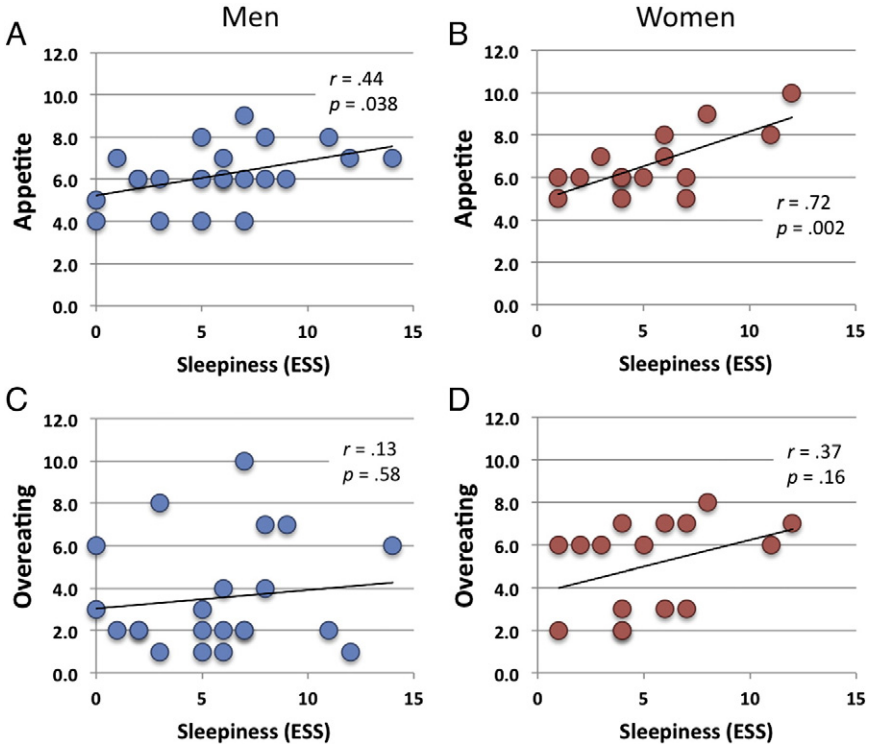

Fig. 1. Scatterplots showing the association between general daytime sleepiness on the Epworth Sleepiness Scale (ESS) and eating-related variables separately for men and women. Daytime sleepiness was significantly correlated with general appetite ratings for both A) men and B) women. In contrast, there was no significant correlation between general daytime sleepiness scores and the tendency to overeat among C) men or D) women.

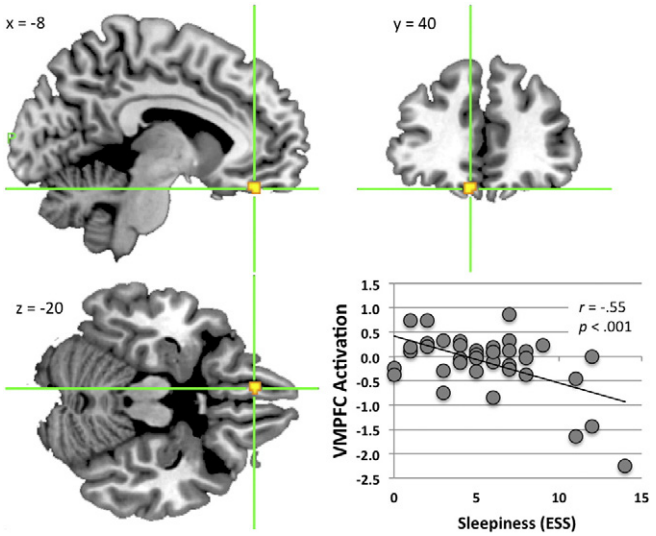

Fig. 2. Daytime sleepiness ratings on the Epworth Sleepiness Scale (ESS) were significantly negatively correlated ($p<.05$, FDR) with a cluster of activation within the ventromedial prefrontal cortex (vmPFC), at the location of the left gyrus rectus (cluster size = 18 voxels). The cluster of activation is shown in the sagittal (top left), coronal (top right), and axial (bottom left) views. The scatter plot (bottom right) shows the association between ESS scores and the mean beta values extracted from the activated cluster [MNI: −8, 40, −20].

Correlations between brain responses and self-reported appetite/overeating behavior

The mean beta values from the cluster identified above were extracted for each individual and correlated with self-reported appetite and overeating behavior. For the sample as a whole, there was no significant relationship between vmPFC activation and appetite ($r=-.22$, $p=.18$) for either men ($r=-.29$, $p=.19$) or women ($r=-.28$, $p=.29$) ($z=-0.03$, $p=.49$). With regard to overeating, there was also no significant relationship with the vmPFC for the sample as a whole ($r=.24$, $p=.14$). However, men and women appeared to differ in this response pattern (see Fig. 3). While men showed no significant association between vmPFC activation and self-reported overeating ($r=.10$, $p=.65$), women showed a significant negative correlation ($r=-.52$, $p=.04$), a difference that was statistically significant ($z=1.88$, $p=.03$), suggesting that reduced responsiveness of this region during food perception was associated with a greater tendency to eat more than intended.

Discussion

We found that self-reported daytime sleepiness was associated with several factors that could lead to excessive food consumption. First, general daytime sleepiness, as assessed by the ESS, was associated with increased ratings of global appetite. Second, when participants were confronted with images of enticing high-calorie foods during neuroimaging, general daytime sleepiness was also associated with reduced activation within a cluster located within the vmPFC, a brain region involved in the ability to inhibit and control emotions and behavior. Third, this reduction in prefrontal activation was directly predictive of self-reported difficulty curtailing food intake, although this association was only significant for women. These findings add to a growing literature suggesting that insufficient sleep and its sequelae are associated with weight gain and the development of obesity (Patel, 2009; Van Cauter and Knutson, 2008), but also sheds light on some additional neurobiological mechanisms in this process that may have heretofore been overlooked.

Recent reviews of the literature have proposed several key physiological mechanisms through which short sleep duration is likely to

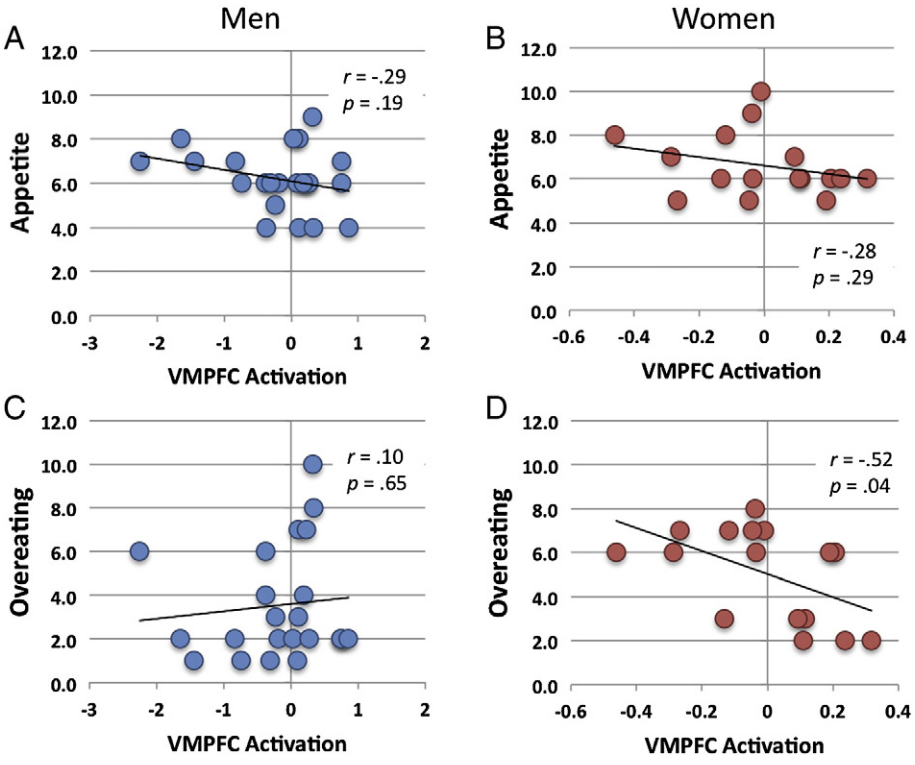

Fig. 3. Scatterplots showing the association between the mean extracted beta values from the ventromedial prefrontal cortex (vmPFC) cluster [MNI: −8, 40, −20] and eating related variables separately for men and women. Activation in the vmPFC was not significantly correlated with general appetite ratings for either A) men or B) women. However, while there was no significant correlation between vmPFC activation and the tendency to overeat among C) men, activation within this cluster was significantly negatively correlated with overeating among D) women.

contribute to increased risk for obesity and diabetes (Knutson et al., 2007; Van Cauter et al., 2008). Most current hypothesized models propose that sleep curtailment leads to 1) increased calorie intake due to alterations in the balance of appetite related hormones and more time available to eat, and 2) reduced energy expenditure due to increased fatigue and altered thermoregulation (Knutson et al., 2007; Patel and Hu, 2008). A prominent feature of these models is evidence that sleep loss exerts a powerful influence on the balance of the hormones that regulate hunger and food consumption, including the appetite stimulating hormone ghrelin and the appetite suppressing hormone leptin (Gale et al., 2004; van der Lely et al., 2004). One large scale study with over a thousand participants showed that polysomnographically measured sleep during an overnight laboratory stay was positively correlated with leptin and negatively correlated with ghrelin levels measured from blood samples the next morning (Taheri et al., 2004). In rodents, laboratory sleep deprivation leads to excessive food consumption (Rechtschaffen and Bergmann, 1995), and studies in humans also suggest that sleep deprivation leads to increased appetite, hunger (Benedict et al., 2012; Pejovic et al., 2010; Spiegel et al., 2004), total food intake (Brondel et al., 2010), and snacking on empty calories (Nedeltcheva et al., 2009). Such findings have led many researchers to suggest that these physiological changes following short sleep duration may contribute to the propensity toward overweight and obesity (Knutson et al., 2007; Patel, 2009; Patel and Hu, 2008; Patel et al., 2008; Van Cauter and Knutson, 2008; Van Cauter et al., 2008).

While physiological factors such as altered leptin and ghrelin levels and reduced energy expenditure appear to play key roles in food intake following sleep loss, the present findings suggest an additional effect of chronically insufficient sleep that may also contribute to overeating. Specifically, we found that higher levels of general daytime sleepiness, as measured by the ESS, were associated with reduced responsiveness of a small cluster within vmPFC when exposed to visual images depicting unhealthy high-calorie foods. This region of the brain has been shown to be critical to a number of emotional and behavioral functions that may directly affect appetitive behavior, including inhibitory capacity (Hodgson et al., 2002; Hornberger et al., 2011; Silbersweig et al., 2007; Szatkowska et al., 2007), representation of the affective value of stimuli (Doallo et al., 2012), and the ability to suppress short-term gains in the service of obtaining longer-term advantageous outcomes (Bechara, 2004; Bechara et al., 2000). Damage to this region has been associated with shortsighted decision-making (Bechara et al., 1994) and problems inhibiting behavior (Hornberger et al., 2011; Szatkowska et al., 2007). Moreover, we found that the lower the activation within this region of the brain, the greater the self-reported tendency to overeat. Thus, not only does lack of sleep affect the metabolic energy balance and hormonal regulators that contribute to increased hunger or appetite, but general daytime sleepiness itself appears to be associated with reduced functional activation within an inhibitory brain region that is critical for regulating behavior, a decline which in turn relates to excessive food consumption.

These findings are consistent with evidence from studies of the effects of laboratory-based sleep deprivation on brain functioning and behavioral control. Studies using PET imaging have demonstrated reduced regional glucose metabolism within the vmPFC following as little as one night of sleep loss (Thomas et al., 2000; Wu et al., 2006). This alteration in brain function within inhibitory and emotional control regions following sleep deprivation corresponds with increased impulsiveness and risk-taking behavior. For example, behavioral studies have shown that sleep deprivation is associated with reduced inhibitory capacity on a go/no-go task (Drummond et al., 2006), short-sighted preference for immediate risky gains over the potential for greater long-term losses (Killgore et al., 2006, 2012a), increased impulsive risk-taking (Killgore, 2007; Killgore et al., 2011), and a bias for risky choices when the outcome is viewed in terms of potential gains rather than losses (McKenna et al., 2007). Overall, sleep deprived individuals tend to show a greater expectation that their risky decisions will lead to gains and a diminished expectation for losses (Venkatraman et al., 2007). Additionally, during sleep deprivation, individuals begin to shift their decision-making strategies away from a focus on avoiding losses toward one of seeking increased gains, a pattern that also involves changes in the activation of the vmPFC (Venkatraman et al., 2011). Evidence also suggests that sleep loss alters the economic value ascribed to stimuli, increasing the perceived reward value of stimuli for some individuals while decreasing it for others (Libedinsky et al., 2011), a pattern that correlates significantly with changes in the activation of the vmPFC following sleep deprivation. When interpreted in light of these prior studies, the inverse relationship between general sleepiness and prefrontal activation we observed suggests that generally sleepy individuals may experience reduced ability to inhibit impulses to eat high calorie foods or they may show increased valuation or expectation of reward from such foods relative to their less sleepy counterparts. Future research will be necessary to determine the relative contribution of prefrontal disinhibition versus altered reward value in the tendency to overeat among individuals with chronically elevated levels of sleepiness.

Interestingly, whereas greater general daytime sleepiness was associated with increased appetite ratings for both sexes, we found that the correlation between food-specific brain responses and self-reported overeating was present only among women, despite similar levels of overeating between sexes overall. These findings are consistent with recent evidence suggesting that women and men may differ in their ability to inhibit activation of the orbitofrontal cortex and suppress corresponding feelings of hunger (Wang et al., 2009). Such findings have previously been proposed as one contributing factor to the higher rates of obesity and eating disorders among women (Hoek, 2006; Striegel-Moore and Bulik, 2007; Striegel-Moore et al., 2009). Prior studies have suggested that women tend to report greater loss of control when eating (Striegel-Moore et al., 2009), a finding that comports well with our results showing that sleepiness-related reductions in prefrontal activation in women were associated with the tendency to eat more than intended. Recent research has also shown that men and women respond differently to high- and low-calorie food stimuli, with women tending to show stronger activation in the insular cortex than men in response to high calorie foods (Killgore and Yurgelun-Todd, 2010). Other studies have also suggested that women show greater activation of dorsolateral prefrontal regions to hedonic foods (Cornier et al., 2010), and greater responsiveness of visual processing regions than men when in a hungry versus sated state (Frank et al., 2010). The precise cause of these sex differences in brain responsiveness still remain elusive, but it seems likely that such distinctions may relate to differences in gonadal sex hormones, which appear to have significant effects on a number of neurological and physiological systems related to food consumption, particularly with regard to the regulation of insulin and leptin (Woods et al., 2003). Further research aimed at addressing the underlying sex differences in responses to food stimuli and the role which sleep may play in this process will be critical in addressing the urgent problem of rising rates of obesity in Western cultures.

While the present study suggests that general daytime sleepiness is related to the functioning of prefrontal regions important to behavioral control and eating behavior, several limitations of this research should be considered. First, we explored the construct of general subjective "sleepiness" rather than short sleep duration or total sleep deprivation. While sleepiness is a common outcome of insufficient sleep, the two constructs are not directly interchangeable. Second, general sleepiness on the ESS reflects a stable assessment of the propensity for sleep to occur across a variety of settings relative to "acute sleepiness" as assessed by other state measures of immediate sleepiness, which were not used here. Of course, there may be measurement error and recall biases inherent in any such subjective measure which queries about recent experience. The present findings can only be

validly generalized to general or chronic levels of sleepiness that tend to emerge with accumulated sleep debt over time, as is generally assessed with the ESS. Third, participants in this study were not selected for weight status, and we specifically screened out individuals with a history of psychopathology, including eating disorders. Consequently, these findings may not generalize to individuals with eating disorders or problems with obesity. Further research with such populations would be an important extension of this work. Fourth, based on prior research suggesting that the vmPFC may be a particularly important region affected by short sleep duration, we focused specifically on that region. Obviously, other regions or networks, such as reward circuitry, interoceptive signaling systems, emotional processing, and visual perception regions, may also be important and should be explored in future research. Fifth, our study lacked any assays for appetite-related hormones such as leptin and ghrelin, so it is not possible to directly rule out the influence of these factors on performance. Lastly, rather than having our participants tested following a fasting period, we opted to have them assessed following their normal daily intake of food during the morning. Nonetheless, energy consumption was monitored closely via food logs, and no food was consumed within an hour before the scanning. Although we controlled for the effects of caloric intake in our analyses, it is possible that the findings may have been different if participants were tested in a strictly fasting or sated state. With due consideration of these potential limitations, we believe the present study provides important new data regarding the role of general daytime sleepiness on brain responses to high-calorie, unhealthy foods. These findings suggest that, in addition to the effects of sleep loss on hormonal levels and energy balance, general daytime sleepiness may also affect brain regions that are critical to the ability to make effective decisions, regulate emotions, and inhibit behavior. Such altered brain functioning due to sleepiness may be an additional and potentially modifiable factor that contributes to the current epidemic of obesity.

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Conflicts of interest

None declared.

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Insomnia-related complaints correlate with functional connectivity between sensory–motor regions

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According to the hyperarousal theory of insomnia, difficulty in initiating or maintaining sleep occurs as a result of increased cognitive and physiological arousal caused by acute stressors and associated cognitive rumination, placing the individual in a perpetual cycle of hyperarousal and increased sensitivity to sensory stimulation. We tested the hypothesis that difficulty in initiating or maintaining sleep would be associated with increased functional connectivity between primary sensory processing and motor planning regions. Fifty-eight healthy adults (29 men, 29 women) completed a self-report inventory about sleep onset and maintenance problems and underwent a 6-min resting-state functional MRI scan. Bilateral regions of interest (ROIs) were placed in primary visual cortex, auditory cortex, olfactory cortex, and the supplementary motor cortex, and the mean processed signal time course was extracted and correlated with each of the other ROIs. Difficulty in falling asleep was associated with increased functional connectivity between the primary visual cortex and other sensory regions such as the primary auditory cortex, olfactory cortex, and the supplementary motor cortex. The primary auditory cortex also showed greater connectivity with the supplementary motor cortex in those with sleep initiation problems. Problems with sleep maintenance were associated with greater connectivity between the primary visual cortex and the olfactory cortex. Consistent with the predictions of the hyperarousal model, difficulty in falling asleep was associated with greater functional connectivity between primary sensory and supplementary motor regions. Such augmented functional connectivity may contribute to the sustained sensory processing of environmental stimuli, potentially prolonging the latency to sleep. *NeuroReport* 24:233–240 © 2013 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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Introduction

Insomnia can be defined as a condition involving difficulty in obtaining sufficient restorative sleep either because of excessive latency to fall asleep, difficulty in remaining asleep, or poor quality sleep that leads to notable impairment in daytime functioning [1]. Formal diagnosis requires symptoms to be present for 4 weeks or longer, and depending on the classification scheme, insomnia can be designated as primary insomnia (PI), insomnia related to a medical or psychiatric condition, or insomnia due to the influence of a substance [2]. Whereas the diagnostic prevalence of PI in the general population is between 3 and 5% [3], close to half of the population experience some symptoms of insomnia that may not necessarily be associated with a formal diagnosis [1]. Over one-third of the population reports symptoms of insomnia, including difficulty in initiating sleep, maintaining sleep, or obtaining nonrestorative sleep at least three times per week [4].

The hyperarousal theory of insomnia posits that difficulty in initiating or maintaining sleep occurs as a result of increased cognitive and physiological arousal caused by acute stressors and associated cognitive rumination [5]. As the latency to sleep is prolonged and rumination continues, the individual becomes even more emotionally aroused because of concerns over his/her inability to sleep and its potential consequences, resulting in a vicious cycle that escalates somatic and cortical hyperarousal, leading to further difficulty in sleeping. Compared with healthy sleepers, individuals with PI show greater high-frequency electroencephalographic activity (EEG) in the β range (14–35 Hz) around the time that they are falling asleep [6]. Increased β power has also been observed during stage 2 nonrapid eye movement (NREM) sleep in PI [7]. Some evidence suggests that this hyperaroused EEG pattern may be persistent even during normal waking [8], indicative of a chronic state of hyperarousal throughout the day. Because this pattern of EEG activity is believed to be fundamental to cognitive and sensory processing, a core feature of the hyperarousal theory of insomnia involves increased sensory processing that interferes with the onset and maintenance of sleep [1]. Thus, individuals with insomnia find themselves in a perpetual cycle of hyperarousal and increased sensitivity to sensory stimulation, which leads to further cortical arousal and difficulty in sleep initiation and maintenance.

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On the basis of the hyperarousal theory of insomnia, we hypothesized that heightened sensory processing would be evident in the form of increased resting-state functional connectivity between sensory regions and motor preparatory regions of the cortex among individuals reporting problems in initiating and/or maintaining sleep. We therefore expected that daytime functional connectivity among sensory–motor regions would be related to difficulty in falling asleep at night among those with insomnia. Healthy normal participants completed a questionnaire about their sleep problems and underwent a 6-min resting-state functional connectivity scan. Individuals reporting difficulty in falling asleep or remaining asleep were hypothesized to show greater functional connectivity between the primary visual cortex, primary auditory cortex, olfactory cortex, and the supplementary motor cortex compared with those without such complaints.

Methods

Participants

Fifty-eight healthy adults (50% women) between 18 and 45 years of age (mean = 30.5; SD = 8.0) participated in the study. A thorough telephone screening was conducted to rule out medical, neurological, or psychiatric problems, including excessive substance use or abuse. All participants provided written informed consent and were compensated for their time. This research protocol was reviewed and approved by the Institutional Review Board of McLean Hospital.

Materials and methods

Sleep questionnaire

Each participant was scheduled for an intake session between 9:00 and 11:00 a.m. After provision of informed consent, each participant completed a number of self-report inventories about sleep and mood. For this analysis, we asked the participant to answer the question 'Do you ever have trouble falling asleep?' This item was identified as sleep initiation difficulty (SID). On the basis of the answer to this question, participants were categorized into SID or non-SID (NSID) groups. If the question was answered affirmatively, participants were also asked to indicate the weekly frequency of trouble falling asleep. Participants were also asked 'Do you ever have trouble staying asleep?' This item was identified as sleep maintenance difficulty (SMD). On the basis of the answer to this question, participants were categorized into SMD or non-SMD (NSMD) groups. If answered affirmatively, participants were also asked to report the weekly frequency of trouble staying asleep. In addition, we also asked participants 'How much sleep did you get last night?' This variable, identified as recent sleep, was scored in hours.

Neuroimaging

A 6-min resting-state functional MRI scan was obtained with eyes open. The scan was taken between 1:00 and 3:00 p.m. Neuroimaging was conducted on a 3T Siemens Tim Trio scanner (Siemens, Erlangen, Germany), using a 12-channel head coil. Initially, a T1-weighted 3D MPRAGE sequence (repetition time/echo time/flip angle = 2.1 s/2.25 ms/12°) over 128 sagittal slices (256 × 256 matrix) was obtained (slice thickness = 1.33 mm; voxel size = 1 × 1.33 × 1 mm). The functional connectivity scan comprised 180 images (3.5 mm thickness, 0 skip; 22.4 cm field of view; 64 × 64 acquisition matrix) over 34 axial interleaved slices obtained using a T2*-weighted blood oxygen level-dependent echoplanar imaging sequence (repetition time/echo time/flip angle = 2.0 s/30 ms/90°).

Image processing and analysis

Standard processing steps were completed in SPM8 (i.e. motion correction, slice-timing correction, coregistration, spatial normalization, and spatial smoothing at 6 mm full width at half maximum), and reslicing dimensions were 2 × 2 × 2 mm. Following preprocessing, functional connectivity analyses were undertaken with the Functional Connectivity Toolbox version 13i (*http://www.nitrc.org/projects/conn*) [9]. Data were band-pass filtered (0.008, 0.10 Hz), and physiological noise was removed using the aCompCor strategy [10]. Principle components analysis was used to eliminate the effects of white matter and cerebrospinal fluid. Motion parameters were also regressed out of the signal. To conduct region-of-interest to region-of-interest (ROI-to-ROI) analysis, eight ROIs from the Automated Anatomical Labeling (AAL) Atlas [11] were imported into the Functional Connectivity Toolbox. These bilateral ROIs are defined in detail by Tzourio-Mazoyer *et al.* [11] and include the following sensory and motor regions: (a) the primary visual cortex [i.e. cuneus – defined as 'the upper part of the medial wall of the occipital lobe, limited rostrally by the parieto-occipital sulcus and ventrally by the calcarine fissure. The cortex surrounding the calcarine fissure and its branches constituted the region of the primary visual area' (p. 285)]; (b) the primary auditory cortex [i.e. Heschl's gyrus – defined within the superior temporal gyrus bound by 'the deep temporal sulcus caudally and the posterior part of the circular sulcus of the insula rostrally' (p. 285)]; (c) the olfactory cortex – defined as 'the olfactory tubercle, lying in the caudal side of the gyrus rectus within the two branches of the fourth frontal sulcus, and the Broca's olfactory cortex located under the corpus callosum genu' (p. 285); and (d) the supplementary motor area (SMA) – defined as including 'the functional definition of the supplementary motor area and presupplementary motor area. Its posterior limit was the paracentral sulcus, its inferior limit was the cingulate sulcus, and we chose to use the Talairach atlas anterior limit: 20 mm ahead of the VAC plane' (p. 282) [11]. For each ROI, the mean time course across all voxels in the labeled mask region was extracted and correlated with the mean time course from each of the other ROIs using standard procedures in the

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Functional Connectivity Toolbox. These correlation values were transformed to a Fishers *z*-score for each subject to permit second-level general linear model analyses. The *z*-scores reflecting the strength of functional connectivity were compared between the SID and NSID groups and between the SMD and NSMD groups using a one-tailed between-group *t*-test, with the number of hours of sleep obtained the previous night included as a nuisance covariate to control for potential effects of sleepiness or sleep debt on the connectivity findings. First, to control for multiple comparisons, all ROI-to-ROI connectivity maps were interrogated simultaneously set-wise at a *P*-value less than 0.10, with the false discovery rate corrected for multiple comparisons across all comparisons and ROIs in the network analysis. Second, within this network, individual ROI-to-ROI analyses were conducted for each seed region at a *P*-value less than 0.05, with false discovery rate corrected for the number of regions within each analysis.

Results

Self-report measures

Sleep initiation difficulty findings

The majority of participants reported that they did not have SID ($n = 33$; 57%), whereas a sizable minority reported that they did ($n = 25$, 43%). Of those who endorsed SID problems, nine (36%) had trouble getting to sleep less than one time per week, 11 (44%) had difficulty one to two times per week, and five (20%) reported having trouble three or more times per week. On average, those reporting trouble falling asleep had such difficulty on 1.71 nights per week (SD = 1.77).

Sleep maintenance difficulty findings

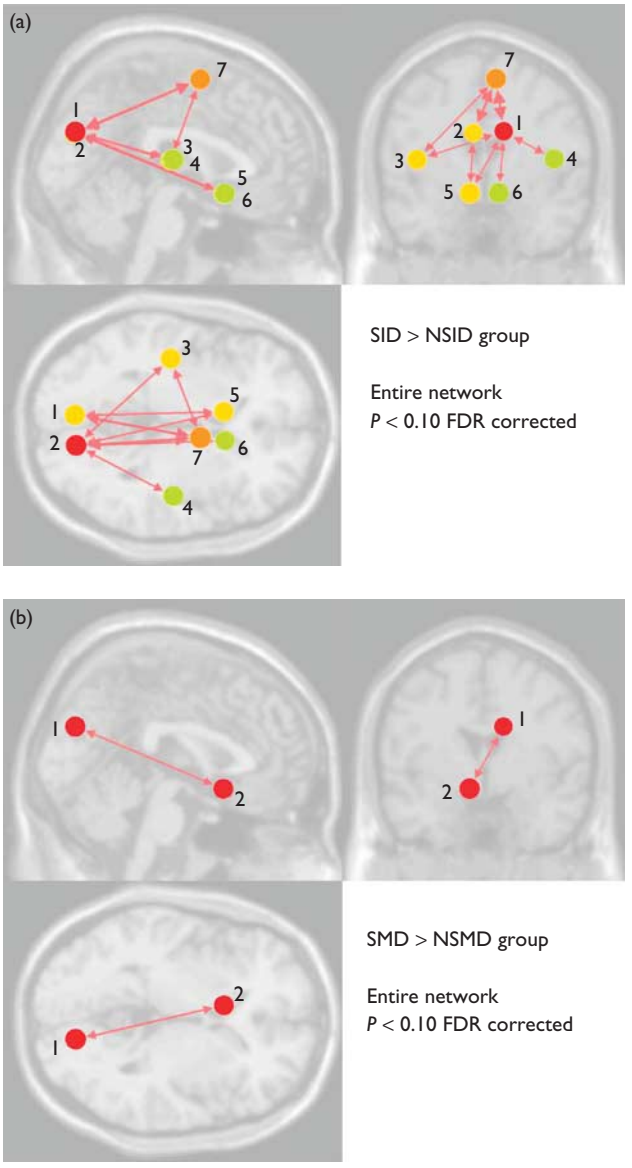
Most participants did not report problems with SMD ($n = 43$, 74%), whereas about a quarter did ($n = 14$, 24%). Among those endorsing sleep maintenance difficulties, three (21%) participants had problems less than once a week, seven (50%) had difficulty one to two times per week, and four (29%) had trouble three or more times per week. The mean number of nights per week an individual had trouble staying asleep was 1.82 (SD = 1.77) among those reporting SMD.

Neuroimaging

Sleep initiation difficulty findings

Compared with those without sleep onset complaints, participants reporting problems with falling asleep showed significantly greater functional connectivity across a network of sensory and motor activation regions (Fig. 1a). Notably, all eight ROIs remained significant when considered simultaneously as a set. Figure 2 shows the individual seed ROI-to-ROI connections for each seed region. Overall, difficulty in falling asleep was associated with increased functional connectivity between the primary visual cortex (i.e. cuneus) and other sensory regions such as the primary auditory cortex (Heschl's gyrus), olfactory cortex, and the supplementary motor cortex. The primary auditory cortex also showed greater connectivity with the supplementary motor cortex in the SID group compared with the NSID group. Statistics for these connections are listed in Table 1. There was no evidence of any connection showing greater

Fig. 1



Wire diagrams showing the network of regions with significantly greater functional connectivity between groups [$P < 0.10$, false discovery rate (FDR) corrected for all network comparisons and regions of interest (ROIs)]. (a) Compared with the no sleep initiation difficulty group (NSID), the sleep initiation difficulty (SID) group showed significantly greater functional connectivity among several regions including (1) the left visual cortex, (2) the right visual cortex, (3) the left auditory cortex, (4) the right auditory cortex, (5) the left olfactory cortex, (6) the right olfactory cortex, and (7) the right supplementary motor area. (b) Compared with the non-sleep maintenance difficulty (NSMD) group, the sleep maintenance difficulty (SMD) group showed greater functional connectivity between (1) the visual cortex and (2) the olfactory cortex.

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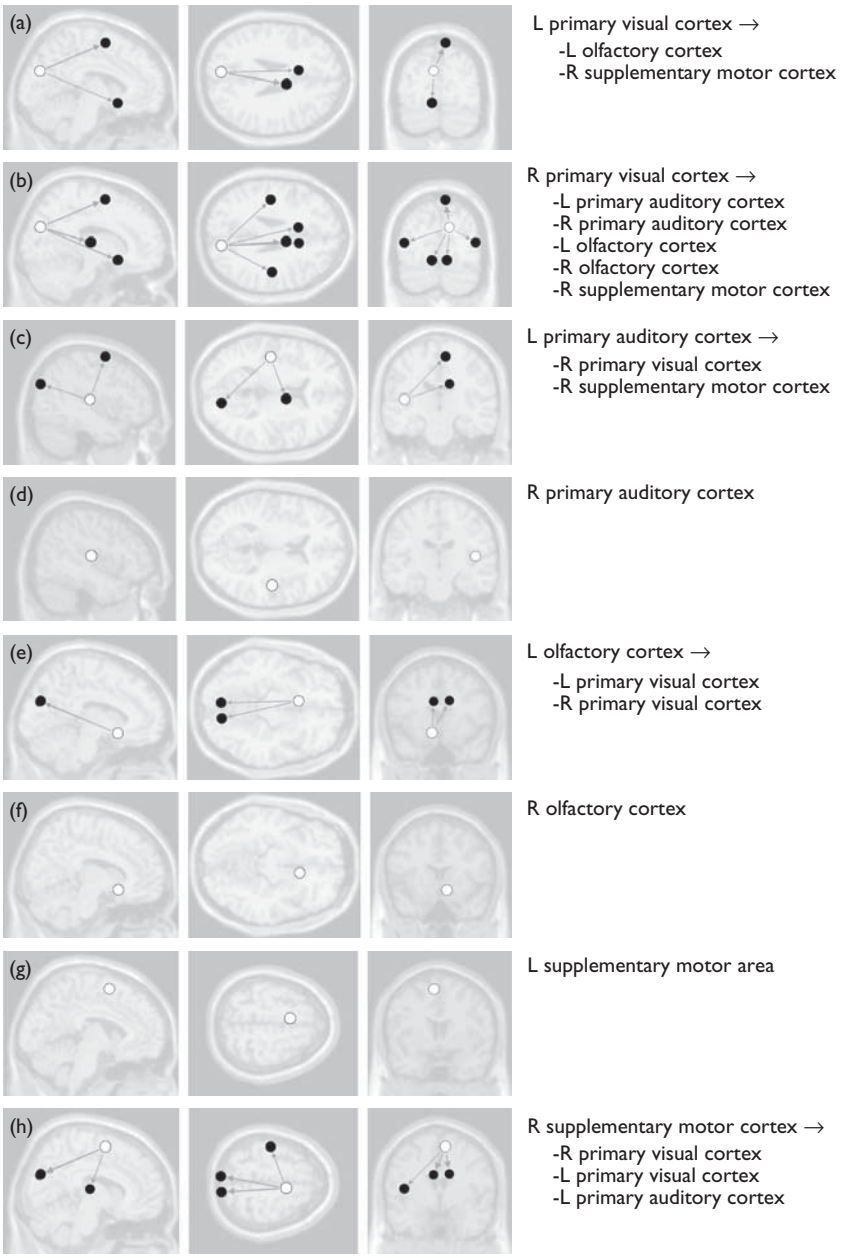
Fig. 2



Individual connectivity maps for each seed ROI-to-ROI comparison that was significantly greater among the SID group compared with the NSID group ($P < 0.05$, false discovery rate corrected). (a) The left primary visual cortex was functionally connected to the left olfactory cortex and right supplementary motor area; (b) the right primary visual cortex was functionally connected to the left and right primary auditory cortex, left and right olfactory cortex, and the right supplementary motor area; (c) the left primary auditory cortex was functionally connected to the right primary visual cortex and right supplementary motor area; (d) the right primary auditory cortex was not functionally connected to other ROIs; (e) the left olfactory cortex was functionally connected to the left and right primary visual cortex; (f) the right olfactory cortex was not functionally connected to other ROIs; (g) the left supplementary motor area was not functionally connected to other ROIs; (h) the right supplementary motor area was functionally connected to the right and left visual cortex and the left primary auditory cortex. NSID, non-sleep initiation difficulty; ROI, region of interest; SID, sleep initiation difficulty.

anticorrelated patterns among the SID group compared with the NSID group.

Sleep maintenance difficulty findings

Individuals reporting problems with maintaining sleep showed greater connectivity between the primary visual cortex and the olfactory cortex compared with the NSMD group, even after setwise correction for all comparisons (Fig. 1b). Figure 3 presents the results from the individual seed ROI-to-ROI analyses, again showing that participants in the SMD group showed greater functional connectivity between the visual cortex and the olfactory

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Table 1 ROI-to-ROI functional connectivity statistics for an individual seed region *t*-test comparison between the SID and non-SID groups

Target region	β	*t*	*d.f.*	P_{unc}	P_{FDR}
Primary visual cortex (L)					
Supplementary motor area (R)	0.20	3.67	55	0.0003	0.0019*
Olfactory cortex (L)	0.11	2.34	55	0.0114	0.0399*
Primary visual cortex (R)					
Supplementary motor area (R)	0.22	3.86	55	0.0002	0.0011*
Primary auditory cortex (L)	0.13	2.48	55	0.0081	0.0210*
Primary auditory cortex (R)	0.13	2.44	55	0.0090	0.0210*
Olfactory cortex (L)	0.10	2.26	55	0.0139	0.0243*
Olfactory cortex (R)	0.11	2.02	55	0.0242	0.0338*
Primary auditory cortex (L)					
Supplementary motor area (R)	0.14	2.51	55	0.0076	0.0282*
Primary visual cortex (R)	0.13	2.48	55	0.0081	0.0282*
Primary auditory cortex (R)	–	–	–	–	–
Olfactory cortex (L)					
Primary visual cortex (L)	0.11	2.34	55	0.0114	0.0486*
Primary visual cortex (R)	0.10	2.26	55	0.0139	0.0486*
Olfactory cortex (R)					
Primary visual cortex (R)	0.11	2.02	55	0.0242	0.1692*
Supplementary motor area (L)	–	–	–	–	–
Supplementary motor area (R)					
Primary visual cortex (R)	0.22	3.86	55	0.0002	0.0001*
Primary visual cortex (L)	0.20	3.67	55	0.0003	0.0001*
Primary auditory cortex (L)	0.14	2.51	55	0.0076	0.0177*

FDR, false discovery rate; NSID, non-sleep initiation difficulty; P_{FDR}, *P*-false discovery corrected for number of regions analyzed; P_{unc}, *P*-uncorrected; ROI, region of interest; SID, sleep initiation difficulty.
*$P<0.10$ FDR setwise corrected for all comparisons across the entire network.

cortex compared with the NSMD group. Table 2 presents the statistics associated with this connectivity. No anticorrelated connectivity differences were observed.

Discussion

We examined resting-state functional connectivity differences between healthy individuals endorsing or denying two types of insomnia-related sleep problems, focusing on the connectivity patterns between several primary sensory processing and motor action preparatory areas. Overall, we found rates of sleep problems similar to those reported previously in the general population [4]. Moreover, participants who reported difficulty in falling asleep showed significantly greater resting-state functional connectivity between several hypothesized regions than those without such problems. Specifically, we found increased connectivity between the primary visual cortex and the supplementary motor areas, as well as between the primary visual and auditory regions and between the auditory cortex and the supplementary motor regions, in those reporting difficulty in falling asleep. The olfactory cortex also showed modestly greater connectivity with the primary visual cortex in the SID group compared with the NSID group. We also examined the functional connectivity within the same network among those reporting difficulty in maintaining sleep. Those reporting SMD showed greater functional connectivity between the primary visual cortex and the olfactory cortex while resting with their eyes open. These patterns of increased functional connectivity were observed even after controlling for the amount of sleep obtained the night preceding the scan, suggesting that the findings are not state related but instead appear to reflect a stable pattern of functioning that may contribute to the reported sleep difficulties.

Our findings are consistent with the hyperarousal model of insomnia [1,5], which suggests that PI is associated with heightened arousal of the central nervous system. This excess arousal is proposed to be manifested as exaggerated cortical, somatic, and cognitive activation, which leads to increased sensory and information processing, ultimately hampering the ability to initiate or maintain sleep [1,6]. We found that individuals reporting difficulty in falling asleep showed significantly greater functional connectivity among several primary sensory regions, as would be expected in the case of increased sensory processing. We also found that those with problems in falling asleep showed greater functional connectivity between these sensory regions and the premotor cortex, a region implicated in the intention to move, the activation of motor plans, and the spontaneous generation of movement [12]. In practical terms, one implication of this finding is that stimulation of one sensory modality, whether internally or externally generated, might be associated with increased activation of other sensory and motor preparatory regions, for example, as in the case in which the sound of the ticking clock leads to activation of the primary visual cortex, increased visual awareness of the environment, and spontaneous body movement. Greater connectivity among these sensory and motor activating regions could conceivably sustain arousal and enhance unwanted sensory awareness, leading to difficulty in falling asleep or awakening easily from sleep.

It is interesting that SID was associated with extensive functional connectivity among the primary sensory and supplementary motor cortex, whereas SMD only showed evidence of greater functional connectivity between the primary visual cortex and the olfactory cortex. This suggests that state functional connectivity observed during waking rest may be more strongly associated with processes interfering with sleep onset than with processes occurring during the sleep state that lead to premature waking. In line with this, some evidence from research examining event-related potentials during sleep suggests that individuals with insomnia show reduced sensory gating of auditory stimuli and a failure to produce stimulus-related K-complexes compared with good sleepers [13]. The K-complex, an EEG waveform commonly seen during stage 2 NREM sleep, is believed to play a role in protecting sleep against spontaneous waking [14]. Thus, problems with SMD may be more related to these aspects of sensory gating and EEG abnormalities than to increased functional connectivity among various sensory and supplementary motor regions. However, the finding of increased connectivity between the medial olfactory cortex and the visual cortex in those who report difficulty

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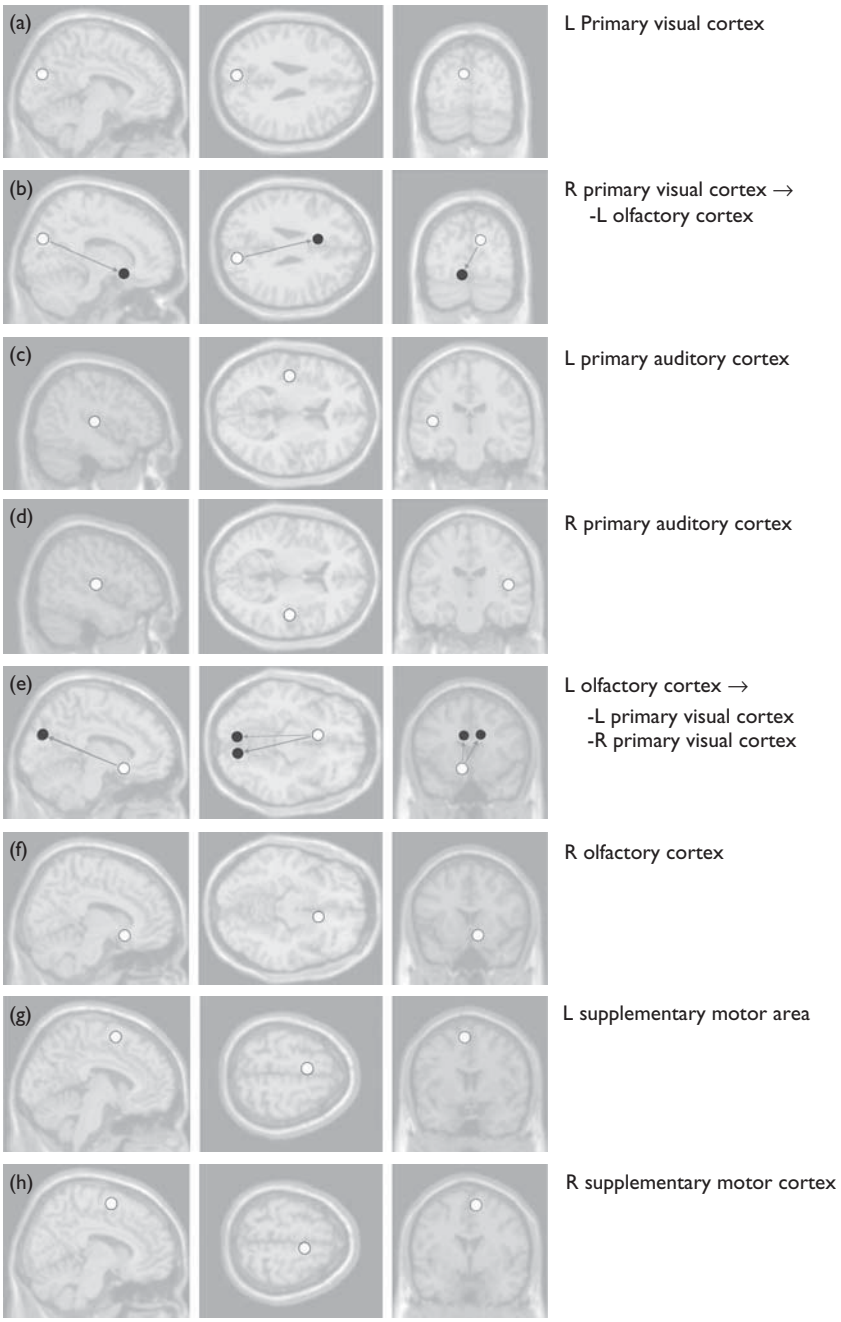
Fig. 3



Individual connectivity maps for each seed ROI-to-ROI comparison that was significantly greater among the SMD group compared with the NSMD group ($P < 0.05$, false discovery rate corrected). (a) The left primary visual cortex was not functionally connected with other ROIs; (b) the right primary visual cortex was functionally connected with the left olfactory cortex; (c) the left primary auditory cortex was not functionally connected with other ROIs; (d) the right primary auditory cortex was not functionally connected with other ROIs; (e) the left olfactory cortex was functionally connected with the left and right primary visual cortex; (f) the right olfactory cortex was not functionally connected with other ROIs; (g) the left supplementary motor area was not functionally connected with other ROIs; (h) the right supplementary motor area was not functionally connected with other ROIs.

in maintaining sleep is intriguing, suggesting a potential sleep disrupting or alerting network that involves these two systems in this group of individuals. Recent data suggest that the olfactory tubercle, one structure located within the olfactory cortex ROI, is involved in cross-modal sensory convergence of smell and sound [15], and it is conceivable that this region may be involved in other aspects of cross-modal convergence. Early work has suggested that individuals with an intolerance to chemical odors show objectively poorer sleep patterns than those

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Table 2 ROI-to-ROI functional connectivity statistics for an individual seed region *t*-test comparison between SMD and non-SMD groups

Target Region	β	*T*	*d.f.*	P_{unc}	P_{FDR}
Primary visual cortex (L)	–	–	–	–	–
Primary visual cortex (R)	–	–	–	–	–
Olfactory cortex (L)	0.15	2.81	55	0.0034	0.0240*
Primary auditory cortex (L)	–	–	–	–	–
Primary auditory cortex (R)	–	–	–	–	–
Olfactory cortex (L)					
Primary visual cortex (R)	0.15	2.81	55	0.0034	0.0240*
Primary visual cortex (L)	0.13	2.29	55	0.0130	0.0454
Olfactory cortex (R)	–	–	–	–	–
Supplementary motor area (L)	–	–	–	–	–
Supplementary motor area (R)	–	–	–	–	–

FDR, false discovery rate; NSMD, non-sleep maintenance difficulty; P_{FDR}, *P*-false discovery corrected for number of regions analyzed; P_{unc}, *P*-uncorrected; ROI, region of interest; SMD, sleep maintenance difficulty.
*$P<0.10$ FDR setwise corrected for all comparisons across the entire network.

without such sensitivity [16], and more recent evidence indicates that individuals with better odor identification abilities are more resistant to sleep deprivation [17]. Although some data suggest that odors can influence the emotional tone of dreams [18] or can enhance the alerting effect of trigeminal nerve stimulation [19], most studies have failed to find an alerting effect of specific odors when presented in isolation during sleep [20]. No study has compared the sensitivity of good and poor sleepers to odors yet.

Thus far, surprisingly few studies have used functional neuroimaging to study insomnia-related problems [21]. An early study found hypoperfusion within cortical regions and basal ganglia in a small sample of five patients with PI [22]. Similarly, Altena *et al.* [23] found that insomnia is associated with reduced functional activation within the medial prefrontal cortex during verbal fluency tasks, which recovered following cognitive behavioral treatment. To our knowledge, only one other study has examined the resting-state functional connectivity of individuals with insomnia-related problems and found that patients with PI show reduced functional connectivity between the amygdala and regions such as the insula, thalamus, and striatum [24]. However, the same study also found increased connectivity of the amygdala with the premotor and sensory–motor cortex. This is consistent with the present findings of increased functional connectivity between the primary sensory and the supplementary motor cortex.

One of the strengths of the present study is the relatively large sample size; however, our findings are limited by the use of a self-report measure to identify sleep difficulties. Consequently, no clinical diagnosis of sleep disorders or PI was made. In fact, our sample was screened to exclude individuals with medical or psychiatric disorders that may affect sleep, but no specific screening for clinical sleep disorders was conducted. This likely restricted the range of sleep disturbances observed. Future research should examine connectivity of these same regions in clinical patients with PI. Finally, because the data are cross-sectional and involve correlational techniques, the causal direction of these results cannot be inferred.

Conclusion

Consistent with the predictions of the hyperarousal model of insomnia, healthy individuals reporting difficulty in falling asleep showed significantly greater functional connectivity among several primary sensory regions and the supplementary motor area compared with those without such complaints. Increased functional connectivity among these regions may contribute to sustained sensory processing of environmental stimuli, which may prolong the latency to sleep.

Acknowledgements

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Conflicts of interest

There are no conflicts of interest.

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Habitual 'sleep credit' is associated with greater grey matter volume of the medial prefrontal cortex, higher emotional intelligence and better mental health

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SUMMARY

In modern society, people often fail to obtain the amount of sleep that experts recommend for good health and performance. Insufficient sleep can lead to degraded cognitive performance and alterations in emotional functioning. However, most people also acknowledge that on a regular basis they obtain more sleep than they subjectively perceive they need at a minimum to stave off performance decrements, a construct we describe as subjective 'sleep credit'. Few people would contest the notion that getting more sleep is better, but data on both behavioural and neuroanatomical correlates of 'sleep credit' are surprisingly limited. We conducted a voxel-based morphometric study to assess cerebral grey matter correlates of habitually sleeping more than one's subjective requirements. We further tested whether these structural correlates are associated with perceived emotional intelligence and indices of psychopathology while controlling for age, gender, and total intracranial volume. In a sample of 55 healthy adults aged 18–45 years (28 males, 27 females), whole-brain multiple regression showed that habitual subjective 'sleep credit' was correlated positively with grey matter volume within regions of the left medial prefrontal cortex and right orbitofrontal gyrus. Volumes were extracted and regressed against self-report emotion and psychopathology indices. Only grey matter volume of the medial prefrontal cortex cluster correlated with greater emotional intelligence and lower scores on several indices of psychopathology. Findings converge with previous evidence of the role of the medial prefrontal cortex in the relationship between sleep and emotional functioning, and suggest that behaviour and brain structure vary with habitual 'sleep credit'.

INTRODUCTION

In today's society, many healthy adults often sleep less than the 7–8 h per night recommended by most experts (Ferrara, 2001; National Sleep Foundation, 2005). Both acutely and habitually insufficient sleep have been demonstrated to affect various aspects of daytime functioning adversely, such as sleep propensity, attention and alertness (Banks and Dinges, 2007; Punjabi *et al.*, 2003; Van Dongen and Maislin, 2003). Relative to being well rested, prolonged sleep debt may also increase somatic complaints and symptoms of depression, anxiety and paranoia (Kahn-Greene *et al.*, 2007). Even measures of self-perceived emotional intelligence may also be impacted negatively by sleep deprivation. More specifically, previous research indicates decreases in both global emotional intelligence, as well as in inter- and intrapersonal functioning (e.g. lower empathy towards others; reduced self-regard) and stress management (e.g. impaired impulse control) following prolonged sleep deprivation compared to rested baseline (Killgore *et al.*, 2008). Thus, there is clear and ample evidence that people do not sleep as much as is recommended physiologically, and that insufficient sleep

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impairs daytime functioning. Interestingly, however, some data also suggest that approximately 40% of adults get more sleep than they think they need subjectively (National Sleep Foundation, 2005). One could argue that getting more sleep than needed subjectively would benefit behaviour and possibly even counteract the effects of physiologically insufficient sleep. Indeed, Rupp and colleagues showed in a series of studies that a brief period of 'banking sleep' by sleeping for longer than normal before a period of insufficient sleep enhances resilience to and recovery from the actual sleep loss (Rupp *et al.*, 2009a,b). One relatively unexplored aspect of the relationship between sleep and behaviour concerns whether sleeping more than needed on a habitual basis (i.e. 'sleep credit') is also associated with behavioural or emotional benefits, and whether this may be reflected in specific differences in regional brain volume.

For chronic sleep restriction, at least in the context of sleep disorders (e.g. chronic insomnia, obstructive sleep apnea, narcolepsy) and daytime sleepiness in otherwise healthy adults, persistently insufficient sleep and excessive daytime sleepiness have been linked to reduced grey matter volume, particularly of the ventromedial and orbitofrontal cortex (Altena *et al.*, 2010; Joo *et al.*, 2010; Killgore *et al.*, 2012b; Morrell, 2003). The mechanisms underlying this relationship remain unknown. Reduced grey matter could emerge as a function of insufficient sleep. However, animal research has not demonstrated conclusively an adverse effect of chronic sleep restriction on neuronal health (Cirelli *et al.*, 1999), although there is some recent evidence that sleep deprivation can reduce axonal sprouting in animal models of stroke (Gao *et al.*, 2010; Zunzunegui *et al.*, 2011) and may inhibit hippocampal volume and neurogenesis in laboratory animals (Mueller *et al.*, 2011; Novati *et al.*, 2011). In humans, no grey matter volume changes were observed in patients with obstructive sleep apnea following successful intervention (O'Donoghue, 2005). Thus, it may be that reduced grey matter in this region may serve as a diathesis that precedes and increases vulnerability to disordered sleep, but it is also conceivable that increased grey matter in this region may emerge as a consequence of sleeping more than the minimum needed to function without impairment. However, given the lack of empirical data on sleep and brain structure, the first step would be to demonstrate this association in humans. Therefore, the present study aimed to investigate voxel-based morphological correlates of sleeping in excess of minimal subjective requirements in a sample of healthy adults. In addition, we also attempted to test whether morphological correlates of 'sleep credit' would be associated with indices of psychopathology and emotional intelligence that have been shown previously to be sensitive to sleep loss. Based on the literature reviewed above, we hypothesized that sleeping habitually in excess of minimal subjective requirements would be associated with increased grey matter volume in ventromedial and orbitofrontal cortices. In addition, we hypothesized that grey matter volume in this brain region would be correlated negatively with lower scores on indices of psychopathology and positively with greater emotional intelligence.

METHODS

Participants

Using posted flyers and internet advertisements, we recruited 55 healthy right-handed adults (mean age 30.74 $\pm$ 8.13, range 18–45; 28 males, 27 females; mean years of education 14.96 $\pm$ 2.17, range 11–20) from the Boston metropolitan area. There was no age difference between females and males. All participants were native English speakers and underwent a detailed screening interview to determine eligibility. Based on this screening, all participants included in the study were deemed healthy (i.e. no history of neurological, psychiatric, alcohol, illicit substance use disorders or sleep disorders). Any other conditions that may influence magnetic resonance imaging (MRI; e.g. chronic pain that would not allow the subject to remain still in the scanner) and psychoactive medications (e.g. antidepressants, analgesics, anticonvulsants) were also exclusionary. Participants were compensated at a rate of $25 per hour. The McLean Hospital Institutional Review Board approved this research, which was conducted in accordance with the 1964 Declaration of Helsinki. Prior to study participation, each participant provided written informed consent.

Materials and procedures

On the day of the MRI scan, participants responded to two open-ended questions on sleep habits: (i) how much do you typically sleep on weeknights (Sunday to Thursday); and (ii) how much do you typically sleep on weekend nights (Friday to Saturday)? In addition, all participants completed the following statement on subjective sleep need: 'If I get less than __ hours of sleep, I notice an impairment in my ability to function at work'. Participant response to the first two items was used to calculate the weighted average habitual sleep (in hours). 'Sleep credit' was conceptualized as the difference between the weighted average habitual sleep and the subjectively reported minimum hours of sleep necessary until functional impairment is noticed.

In addition, participants completed the Bar-On Emotional Quotient Inventory (EQ-i; Bar-On, 2006), a self-report measure of trait emotional intelligence. The inventory contains 125 items yielding a total emotional quotient plus five composite scores (interpersonal, intrapersonal, adaptability, stress management, general mood). Individual items are answered on a five-point Likert scale ranging from 'very seldom or not true of me' to 'very often true of me or true of me'. The interpersonal composite score reflects perceived empathy and interpersonal skills, whereas the intrapersonal composite provides a measure of self-perceived awareness of personal emotions and self-regard. Adaptability reflects the perceived ability to

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scrutinize challenging circumstances objectively, to resolve them and to adapt flexibly to changing situations. Stress management provides a measure of tolerance of, and perceived self-control during, taxing or challenging situations. Finally, the general mood composite reflects self-reported positive thinking and overall satisfaction with personal life. Based on previous research showing that sleep loss affects specific scales of the EQ-i (Killgore *et al.*, 2008), we restricted our analyses to the total emotional quotient and the interpersonal, intrapersonal and stress management composites.

Every participant also completed the computerized Personality Assessment Inventory (PAI; Morey, 1991) as an index of several dimensions of psychopathology. The PAI contains 344 statements that are rated using one of four response options ('false, not at all true', 'slightly true', 'mainly true', 'very true'). It yields 11 clinical subscales (somatic complaints, anxiety, anxiety-related disorders, depression, mania, paranoia, schizophrenia, borderline features, antisocial features, drug-related problems, alcohol-related problems). Based on previous findings from the literature showing that sleep deprivation affects specific scales on the PAI (Kahn-Greene *et al.*, 2007), we restricted our primary analyses to four clinical scales (i.e. somatic complaints, anxiety, depression, paranoia). Raw scores for each scale and subscale used in this study were converted into T scores based on the normative data provided with the scoring programme and in the test manual (Morey, 1991).

MRI parameters

We acquired structural magnetic resonance images at 3.0 Tesla using a 12-channel head coil (Siemens Tim Trio, Erlangen, Germany) and a T1-weighted three-dimensional MPRAGE sequence (TR/TE/flip angle: 2.1s/2.25 ms^{-1}/12°; 128 sagittal slices; 256 × 256 matrix; in-plane resolution: 1 × 1 × 1 mm; slice thickness: 1.33 mm).

Voxel-based morphometry

The VBM8 toolbox in SPM8 was used for preprocessing of structural images (Wellcome Department of Imaging Neuroscience Group, London, UK; http://www.fil.ion.ucl.ac.uk/spm/; http://dbm.neuro.uni-jena.de/vbm.html). The modulated voxel-based morphometry (i.e. grey matter volume was corrected for total brain volume) applied default settings. That is, each structural image was first DARTEL-normalized to match the Montreal Neurological Institute template. Then, a fully automated algorithm within SPM8 segmented each image into grey matter, white matter and cerebrospinal fluid. Finally, normalized grey matter images were smoothed with an 8-mm full-width at half-maximum isotropic Gaussian kernel.

Statistical analysis

The statistical analysis involved three nested steps. The first step tested the association between the amount of 'sleep credit' and voxelwise grey matter volume. Thus, normalized smoothed grey matter images were entered into a whole-brain general linear model in SPM8 using a threshold of $P < 0.001$, uncorrected, with a cluster extent $k \geq 90$, which was determined statistically as the expected number of voxels per cluster that would be expected by chance based on the theory of Gaussian random fields applied to this analysis (as provided in the standard VBM8 output). Age and gender were included as nuisance covariates. The second step tested whether the grey matter volume clusters from the first step were associated with scores on the emotional intelligence and psychopathology indices. Here, eigenvariates, as extracted from each significant cluster, were entered stepwise as regressors into a series of multiple regression models in IBM SPSS Statistics for Macintosh version 20 (IBM Corp., Armonk, NY, USA). To limit type I error, we ran one primary analysis for the total score of the EQ-i and PAI, respectively (i.e. five models: EQ-i total; PAI somatization; PAI anxiety; PAI depression; PAI paranoia) using a Bonferroni-corrected significance threshold $P < 0.01$ for each model. The third analysis step examined associations between grey matter volume and composite scores of the two tests used (i.e. EQ-i: interpersonal, intrapersonal, stress management; PAI: somatization conversion/somatization/health concerns; anxiety cognitive/affective/physiological; depression cognitive/affective/physiological; paranoia hypervigilance/persecution/resentment). Follow-up analyses were conducted only for significant primary analyses and significant regressors. Normal distribution of all dependent variables was tested with the Shapiro–Wilk test in SPSS. For normally distributed data (i.e. all EQ-i composites), we derived Pearson correlations, whereas Spearman correlations were computed for the non-normally distributed composite scores (i.e. all PAI composites). Again, per inventory, we applied a Bonferroni-corrected significance threshold $P < 0.017$ ($\alpha = 0.05$ divided by three subscores).

RESULTS

On average, self-reported habitual sleep was 7.46 h [standard deviation (SD) = 0.80, range 5.5–9.0]. The minimum hours of sleep needed subjectively before functional impairment was noticeable to the individual varied greatly from 2 to 10 h (mean = 5.65, SD = 1.35). Therefore, on average, participants 'banked' 1.8 h of 'sleep credit' on a habitual basis (SD = 1.49, range −3 to 5.5). The mean total EQ-i was 100.60 (SD = 13.89, range 69–126; interpersonal: mean = 99.98, SD = 14.76, range 59–125; intrapersonal: mean = 100.55, SD = 14.88, range 59–125; stress management: mean = 102.89, SD = 12.21, range 76–125). All mean PAI scale and subscale scores were within normal ranges (somatic complaints: mean = 46.07, SD = 6.88, range 39–89; anxiety: mean = 47.47, SD = 9.39, range 35–73; depression: mean = 47.20, SD = 10.82, range 36–101; paranoia: mean = 51.53, SD = 10.46, range 32–74).

As evident in Fig. 1, greater 'sleep credit' correlated with greater grey matter volume in two clusters: (i) left gyrus

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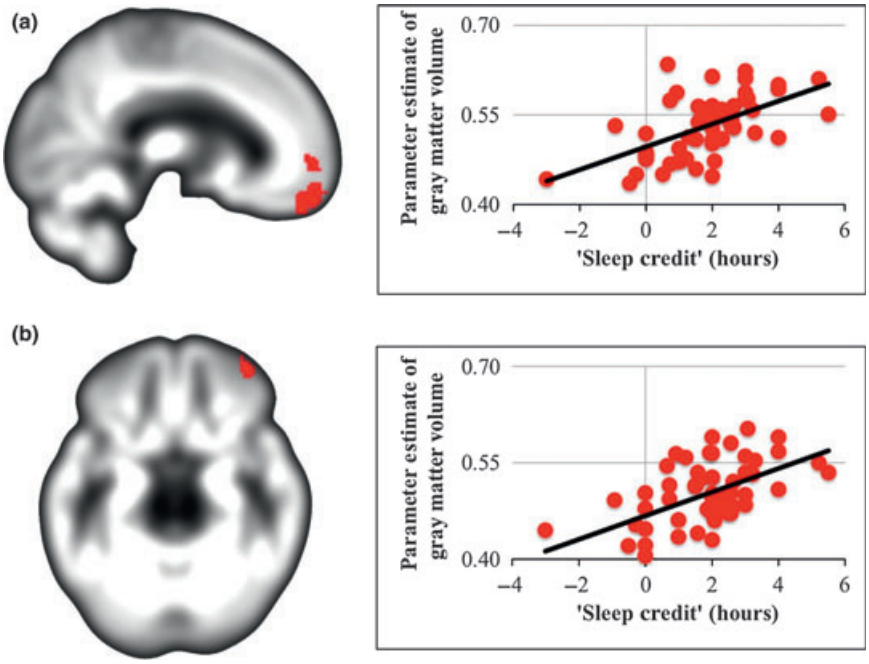

Figure 1. (a) Sagittal view of the left gyrus rectus/orbitofrontal gyrus cluster (overlaid on sample-specific T1 mean image) that was correlated positively with sleeping in excess of subjective need and the corresponding scatterplot showing the relationship between sleeping more than needed subjectively (in hours) and average grey matter volume for the cluster located at Montreal Neurological Institute (MNI) coordinates $x = -6$, $y = 52$, $z = -21$. (b) Axial view of the right middle orbitofrontal gyrus cluster (overlaid on sample-specific T1 mean image) that was correlated positively with sleeping in excess of subjective need and the corresponding scatterplot showing the relationship between 'sleep credit' (i.e. sleeping more than needed subjectively) and average grey matter volume for the cluster located at MNI coordinates $x = 39$, $y = 51$, $z = -18$.

rectus/superior and medial orbital frontal gyrus (OFG; 892 voxels, $T = 4.81$, peak-level Montreal Neurological Institute (MNI) coordinates: $x = -6$, $y = 52$, $z = -21$) and (ii) right middle OFG (149 voxels, $T = 4.43$, peak-level MNI coordinates: $x = 39$, $y = 51$, $z = -18$).

Table 1 presents results of the multiple regression analysis on the total EQ-i. Only grey matter volume of the cluster comprising the left gyrus rectus, superior and medial OFG correlated with total EQ-i. This means that greater grey matter in this medial PFC cluster was associated with greater global perceived emotional intelligence. Follow-up Pearson correlations with Bonferroni correction to $P < 0.017$ showed positive associations between this cluster's grey matter volume and the interpersonal subscale only [interpersonal: $r = 0.38$, $P < 0.017$; intrapersonal: $r = 0.28$, not significant (NS); stress management: $r = 0.23$, NS].

Table 2 presents results of the multiple regression analyses on the PAI clinical scales somatization, depression, anxiety and paranoia. Similar to the findings for the EQ-i, only grey matter volume of left gyrus rectus/medial and superior OFG correlated with PAI psychopathology. Overall, greater grey matter volume in this cluster in the medial PFC correlated with lower scores on indices of psychopathology, but this was true only for somatic complaints, depression and paranoia, but not anxiety. For PAI somatization, none of the follow-up Spearman correlations survived Bonferroni correction (conversion: $r = -0.29$, $P = 0.03$; somatization: $r = -0.15$, $P = 0.29$; health concerns: $r = -0.29$, $P = 0.03$). For PAI depression, follow-up Spearman correlations showed negative associations between grey matter volume of this cluster in the medial PFC and cognitive and physiological, but not affective, symptoms of depression (cognitive: $r = -0.34$, $P < 0.017$; affective: $r = -0.29$, $P = 0.03$; physiological: $r = -0.36$, $P < 0.017$). For PAI paranoia, follow-up Spearman correlations also showed negative associations between grey matter volume of this

Table 1 Stepwise linear regression of grey matter volume on emotional intelligence

Total EQ-i	*B*	*SE B*	ΔR^2	β
Step 1				
Constant	47.84	18.81		
L. gyrus rectus/superior and medial OFG	99.31	35.26	0.13	0.36*
Step 2				
Constant	44.95	20.77		
L. gyrus rectus/superior and medial OFG	92.30	41.00	0.13	0.34
R. middle OFG	13.22	38.55	0.01	0.05

EQ-i, emotional quotient; OFG, orbitofrontal gyrus; SE, standard error.
*$P < 0.01$.

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Table 2 Stepwise linear regression of grey matter volume on hypothesized PAI psychopathology scales

	B	*SE B*	ΔR^2	β
PAI somatization				
Step 1				
Constant	47.84	18.81		
L. gyrus rectus/superior and medial OFG	99.31	35.26	0.13	0.36*
Step 2				
Constant	44.95	20.77		
L. gyrus rectus/superior and medial OFG	92.30	41.00	0.13	0.34
R. middle OFG	13.22	38.55	0.01	0.05
PAI anxiety				
Step 1				
Constant	76.16	13.05		
L. gyrus rectus/superior and medial OFG	−53.99	24.46	0.08	−0.29
Step 2				
Constant	72.64	14.38		
L. gyrus rectus/superior and medial OFG	−62.49	28.38	0.08	−0.34
R. middle OFG	16.03	26.68	0.01	0.09
PAI depression				
Step 1				
Constant	96.64	14.15		
L. gyrus rectus/superior and medial OFG	−93.07	26.53	0.19	−0.43*
Step 2				
Constant	103.90	15.45		
L. gyrus rectus/superior and medial OFG	−75.52	30.50	0.19	−0.35
R. middle OFG	−33.13	28.67	0.02	−0.16
PAI paranoia				
Step 1				
Constant	106.14	13.20		
L. gyrus rectus/superior and medial OFG	−102.80	24.74	0.25	−0.40*
Step 2				
Constant	114.63	14.30		
L. gyrus rectus/superior and medial OFG	−82.29	28.23	0.25	−0.40*
R. middle OFG	−38.72	26.54	0.03	−0.20

PAI, personality assessment inventory; OFG, orbital frontal gyrus; SE, standard error.
*Bonferroni-corrected $P < 0.01$.

medial PFC cluster and all three subscores of the PAI paranoia scale (hypervigilance: $r = -0.44$, $P < 0.017$; persecution: $r = -0.40$, $P < 0.017$; resentment: $r = -0.37$, $P < 0.005$).

Finally, it was conceivable that even subclinical psychopathology or level of education could have affected the subjective estimates of how much sleep was needed before experiencing impairments. Therefore, to address this possibility, we correlated scores on each of the hypothesized PAI scales and education (in years) with the minimum amount of sleep reported before noticeable impairment. Sleep need was not related significantly to depression ($r = 0.22$, $P = 0.11$), somatization ($r = 0.11$, $P = 0.43$) or anxiety ($r = 0.17$, $P = 0.21$), but was related to level of paranoia ($r = 0.34$, $P = 0.01$). Subjective minimum sleep need before the emergence of noticeable work impairment was correlated negatively with years of education ($r = -0.30$, $P = 0.02$). This suggests that higher education level was associated generally with a lower perceived sleep need.

DISCUSSION

Habitual sleep in excess of perceived minimal need to avoid impairment, defined here as 'sleep credit', was associated with greater grey matter volume of the medial frontal and orbitofrontal cortex, regions important to emotional perception and affective regulation. Furthermore, individual variation in grey matter volume of the medial prefrontal cortex cluster was associated with global self-perceived emotional intelligence, in particular capacities involving interpersonal skills that contribute to the ability to understand and relate well with others. Similarly, greater grey matter volume of the same cluster was correlated with reduced severity on several indices of psychopathology, particularly in terms of overall somatic complaints, depression and paranoia. In short, sleeping more than the minimum required to sustain performance was associated with increased grey matter volume in cortical areas critical to emotional regulation, and larger volume of these areas was associated with better emotional and psychological health.

Our data offer additional support to a line of converging empirical studies showing that function, structure and connectivity of medial prefrontal and orbitofrontal cortices are vital to the understanding of sleep and its relationship to behaviour. For example, decreased metabolic activity following one night of sleep deprivation was not restricted to, but predominant within, this brain region, particularly within the bilateral gyrus rectus (Thomas *et al.*, 2000). Furthermore, attentional lapses appear to be linked to diminished activity in the medial prefrontal cortex (Chee *et al.*, 2008). Volumetric data also point to an important connection between the medial prefrontal cortex and sleep-related problems. For instance, grey matter volume in the context of both sleep disorders and increased daytime sleepiness in healthy adults tends to be reduced in this region in association with greater sleep-related pathology (Altena *et al.*, 2010; Joo *et al.*, 2009; Killgore *et al.*, 2012b; Morrell, 2003). Lastly, functional connectivity of the medial prefrontal cortex with other emotional and socially relevant brain regions has been shown to be correlated inversely with sleep duration (Killgore *et al.*, 2012a) or is particularly weakened following a full night of sleep deprivation (Yoo *et al.*, 2007). Our data complement these findings by showing that grey matter structure within the medial prefrontal cortex varies systematically with habitual 'sleep credit'.

The present study also builds upon and extends previous research into the beneficial effects of excess (Anderson *et al.*, 2009) or 'banked' sleep (Rupp *et al.*, 2009a,b). Previously, it was shown that 'banking sleep' for 1 week was associated with greater resilience to, and better recovery

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from, a period of insufficient sleep (Rupp *et al*., 2009a,b). In these studies, 'banking sleep' was conceptualized as getting more than habitual sleep (e.g. 10 h time in bed instead of an individual's typical 8 h). The authors argued that sleep history needs careful consideration in experimental sleep deprivation studies due to its potential moderating effect, and that one night of baseline sleep might not provide a sufficient baseline measure. Our data suggest habitual 'sleep credit' - that is, sleeping more than needed subjectively (e.g. getting 8 h if one perceives 5 h to be the point at which an impairment would be noticed) - as an additional putative moderator of the sleep–behaviour relationship. We showed that sleeping more than needed subjectively was associated with greater grey matter volume within the medial prefrontal cortex, a brain region that has been implicated strongly in sleep, sleep deprivation and their behavioural correlates. While the cross-sectional nature of our data do not allow us to draw conclusions regarding the direction of this effect, it allows us to pose the question of whether individuals who habitually sleep more than their subjective need might also be more resistant to acute sleep deprivation – possibly because of an advantageous neuronal substrate. Future studies employing longitudinal assessment, neuroimaging and acute sleep deprivation may be able to address this possibility.

Probably very few would contest that surplus sleep provides a range of benefits. Our data showed that sleeping in excess of the amount needed subjectively to avert degraded performance was linked to greater grey matter volume in a cortical region critical to both cognition and emotion (Fuster, 2008). Thus, in additional to highlighting the neurostructural benefits associated with 'sleep credit', our data replicated previous findings of behavioural deficits associated with insufficient sleep, particularly in terms of complex cognition such as emotional intelligence and psychological health (Kahn-Greene *et al*., 2007; Killgore *et al*., 2008). In those previous studies, prolonged sleep deprivation led to a degradation of both global and specific aspects of emotional intelligence, whereas psychopathological symptoms, including somatization, anxiety, depression and paranoia, increased without sleep. Our data showed that most of these same symptom dimensions, including somatization, depression and paranoia, were correlated with grey matter volume of the medial prefrontal cortex cluster, the very region that was implicated in habitually sleeping longer than needed subjectively. Longitudinal data are needed to establish whether habitual 'sleep credit' in fact induces grey matter changes in this region or whether these cortical volume differences reflect a biological substrate permitting decreased sleep need.

One previous study investigated whether the difference between habitual sleep duration and perceived sleep need was associated with a variety of sleep and trait measures such as sleep propensity, subjective daytime sleepiness, psychomotor vigilance, anxiety and personality (Anderson *et al*., 2009). Interestingly, no difference emerged in these putative behavioural correlates of 'sleep credit' (i.e. getting more sleep than needed), sleep deficit (i.e. getting less sleep than needed) and getting as much sleep as needed. However, neurostructural and higher-cognitive correlates such as emotional intelligence were not investigated, and the accumulated 'sleep credit' in our sample was, on average, greater than in the Anderson *et al.* (2009) study, suggesting limited comparability between studies. Future research should investigate whether 'sleep credit' contributes to elementary cognitive functions such as psychomotor vigilance, as well as higher-order cognitive processes such as emotional intelligence to resolve this apparent discrepancy.

Finally, although our primary hypotheses were restricted to four PAI subscales (i.e. somatization, depression, anxiety, paranoia), as previous research has shown sleep deprivation to affect these scales specifically (Kahn-Greene *et al*., 2007), we also conducted additional exploratory analyses. For comprehensiveness and to obviate any bias in reporting, we conducted multiple regressions with the remaining non-hypothesized PAI subscales (i.e. anxiety-related disorders, mania, schizophrenia, borderline features, antisocial features) at a Bonferroni-corrected threshold of $P < 0.05$. Interestingly, the specificity of the association between sleep deprivation and PAI psychopathology demonstrated by Kahn-Greene *et al.* (2007) was also observed in the present study of 'sleep credit'. Specifically, after correction for multiple comparisons, grey matter volume did not relate to any of these scales except for schizophrenia, which showed a negative association with grey matter volume within the left gyrus rectus, superior and medial OFG only (Table S1). We also found that formal education level was correlated negatively with the perceived amount of sleep necessary before functional work impairments become noticeable. While not hypothesized, this negative relationship suggests that individuals with higher educational attainment perceive themselves as able to function with less sleep before experiencing impaired performance. There are several possible explanations for this relationship, including greater cognitive ability among those with higher educational attainment, which might confer greater cognitive reserve, or individuals with lower education may have been less reliable in reporting their sleep need and impairment levels. These questions were not addressed directly in the present study and will require additional research.

The study is not without limitations and our results need to be replicated, preferably in a larger independent sample with prospective objective measurement of actual sleep via actigraphy or some method of ambulatory monitoring (Anderson *et al*., 2009). Despite including 55 healthy adults, our sample size was still at the lower end of the ideal number of subjects to be included in a multiple regression analysis with two predictors. Also, whereas most regression diagnostics (i.e. standardized residuals, Durban–Watson test statistic, collinearity statistic, Cook's distance, Mahalanobis distance, covariance ratio) did not reveal any violation of multiple regression assumptions for any of the variables of interest, hat values exceeded common thresholds on PAI scales of

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somatization, anxiety and depression for a few selected subjects, suggesting possible excess leverage effects. We therefore cannot readily generalize results regarding these three variables beyond our sample. Additionally, our definition of 'sleep credit' is not unassailable. Here, we defined 'sleep credit' as the amount of sleep that an individual obtains habitually relative to that person's own subjectively perceived sleep need, defined as the threshold of sleep necessary before daytime impairment is noted. Alternatively, 'sleep credit' could be defined in relation to subjective sleep necessary for optimal performance in daily life. While conceptually similar and potentially related, 'lack of impairment' and 'optimal functioning' are clearly distinct constructs that may be affected differentially by sleep loss. It is likely that the behavioural and brain structural correlates of excess sleep beyond an 'optimal functioning' threshold would be different to those explored here examining excess sleep beyond a 'no impairment' threshold. Teasing apart the role of sleep and brain structure on each of these deserves further study. It remains to be shown whether and how differences in operationalization of 'sleep credit' might influence findings of grey matter correlates and their association with emotional intelligence and mental health. Future investigations should also include a more detailed assessment of the type of impairment (e.g. worse memory, difficulties in concentration, changes in emotional response and expression) that is noticed when sleep is less than subjective need, including daytime sleepiness (Killgore *et al*., 2012b). Also of interest might be an assessment of each participant's specific work situation and job requirements to determine whether the effects of 'sleep credit' might depend upon such work-setting factors. Furthermore, it may be useful to explore the reasons why some participants habitually obtain the sleep they need subjectively while others do not. Lastly, readers should bear in mind that these data are correlational, and therefore causality cannot be inferred.

In conclusion, this is the first voxel-based morphometric study to show that sleeping in excess of subjective minimal need is associated significantly with greater grey matter volume within the medial prefrontal cortex, a region critical to the monitoring and control of affective processes. Notably, the volume of this same region was correlated independently with greater emotional intelligence and lower scores on indices of psychopathology in the same participants. These data support the notion that sleeping beyond the minimal subjective requirements for adequate performance may affect brain structure and relevant emotional capacities.

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CONFLICTS OF INTEREST

No conflicts of interests declared.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Table S1. Stepwise linear regression of grey matter volume correlates of non-hypothesized PAI psychopathology.

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Emotional intelligence correlates with functional responses to dynamic changes in facial trustworthiness

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Emotional intelligence (EI) refers to a constellation of traits, competencies, or abilities that allow individuals to understand emotional information and successfully navigate and solve social/emotional problems. While little is known about the neurobiological substrates that underlie EI, some evidence suggests that these capacities may involve a core neurocircuitry involved in emotional decision-making that includes the ventromedial prefrontal cortex (vmPFC), anterior cingulate cortex (ACC), insula, and amygdala. In a sample of 39 healthy volunteers (22 men; 17 women), scores on the Bar-On EQ-i (a trait/mixed model of EI) and Mayer–Salovey–Caruso Emotional Intelligence Test (MSCEIT; an ability model of EI) were correlated with functional magnetic resonance imaging responses during brief presentations of moving facial expressions that changed in the level of perceived trustworthiness. Core emotion neurocircuitry was responsive to dynamic changes in facial features, regardless of whether they reflected increases or decreases in apparent trustworthiness. In response to facial movements indicating decreasing trustworthiness, MSCEIT correlated positively with functional responses of the vmPFC and rostral ACC, whereas the EQ-i was unrelated to regional activation. Systematic differences in EI ability appear to be significantly related to the responsiveness of the vmPFC and rostral ACC to facial movements suggesting potential trustworthiness.

Keywords: Emotional intelligence; Somatic Marker Hypothesis; Ventromedial prefrontal cortex; Amygdala; Insula.

Human beings vary widely in their ability to acquire new information, understand their environment, think rationally, apply their knowledge to adapt to changing conditions, solve problems, and achieve goals. Broadly speaking, these capacities comprise what is known as "intelligence" (Wechsler, 1958). While the concept of intelligence as a unitary construct has persisted for over a century, some authors have hypothesized the possible existence of multiple forms of intelligence (Gardner, 1983). In particular, the construct of emotional intelligence (EI) has garnered considerable interest both within scientific and popular writings since the mid-1990s (Bar-On, 2006; Goleman, 1995; Mayer, Salovey, Caruso, & Sitarenios, 2001). While scholars differ in the exact criteria used to define EI, most current conceptualizations generally agree that the construct involves some constellation of traits, competencies, or abilities that allow an individual to understand emotional information and successfully navigate and solve social/emotional problems (Bar-On, 2006; Mayer et al., 2001). From one perspective, EI is described as a relatively stable constellation of emotionally related competencies and traits that underlie the potential to cope adaptively with demanding situations and to use emotional knowledge to succeed in achieving goals (Bar-On, 2006). This *Trait* (or Mixed) model views EI as similar in many ways to personality, though more modifiable through life experience and reflection (Webb et al., 2013). In contrast, the *Ability* model of EI defines the construct

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in terms of measurable performance on tasks requiring the ability to solve emotional problems as well as demonstrate reasoning and knowledge about emotional processes (Webb et al., 2013). Both theoretical models continue to be actively researched and both have yielded well-normed, standardized, commercially available tests (Brackett & Mayer, 2003). At present, however, there exists very little scientific understanding of the neurocircuitry involved in EI.

One hypothesized model of the neurocircuitry underlying EI was proposed by Bar-On and colleagues (Bar-On, Tranel, Denburg, & Bechara, 2003), and suggests that the key features of EI can be parsimoniously explained by the emotional decision-making circuitry outlined by Antonio Damasio (Damasio, 1996), known as the Somatic Marker Circuitry (SMC) (Bar-On et al., 2003; Bechara & Damasio, 2005). The Somatic Marker Hypothesis (SMH) essentially provides an explanation of how the brain learns from emotional experiences and uses "somatically remembered" experiential knowledge to influence future decisions (Damasio, 1994). Simply put, the SMH suggests that our cognitive deliberations during decision-making are aided by emotional body signals, "hunches," or "gut feelings" that were initially formed through prior experience with a stimulus or situation, and that are automatically reactivated during future encounters resembling the earlier experience. According to the SMH, three primary brain structures (in addition to many secondary ones) are involved in the development of these somatic biasing signals to influence judgment and decision-making. First, the amygdala is proposed to be responsible for triggering initial signals of emotional salience in response to a relevant stimulus that leads to enactment of the somatic states characteristic of an emotion. Second, the insula contributes to the "feeling" of emotion by neurally mapping these somatosensory and visceral sensations, which can later be "simulated" within the brain when a comparable emotion-evoking stimulus is encountered in the future. Finally, the ventromedial prefrontal cortex (vmPFC) is posited as the core integrative system that joins these somatic signals with past and present cognitive representations of stimuli and situations. Once a stimulus has been associated with a pleasant or unpleasant feeling (i.e., a somatic marker), future reactivation of these cognitive representations of the stimulus (or actual re-encounters) can evoke a similar somatic experience (i.e., a "good" or "bad" feeling), biasing subsequent judgments and decisions toward advantageous outcomes. Bar-On and colleagues suggest that the SMC is the primary neurocircuitry that underlies the capabilities and competencies that comprise EI (Bar-On et al., 2003). The anterior cingulate cortex, particularly in the rostral regions, may also serve as part of the extended medial prefrontal cortex as it plays a key role in regulating emotion and resolving emotional conflict (Etkin, Egner, & Kalisch, 2011; Etkin, Egner, Peraza, Kandel, & Hirsch, 2006).

Some data from neuroimaging studies now exist to support the role of the SMC in EI. Based on the assumption that EI capacities involve reasoning and problem solving about emotion, most studies of the neurobiological basis of this construct have focused on prefrontal cortical regions involved in problem solving and emotional regulation, as well as somatic and emotional processing regions such as the insular cortex and amygdala. Early studies using functional magnetic resonance imaging (fMRI) showed that activation of some of these hypothesized regions of the brain, particularly the medial prefrontal cortex, was negatively correlated with measures of *Ability* EI (Reis et al., 2007) and *Trait* EI (Killgore & Yurgelun-Todd, 2007). The inverse relationships between EI and brain responses to these very simple task designs, using static fearful facial expressions or rule-based card selection tasks, were interpreted as evidence of neural efficiency, suggesting that individuals possessing greater EI were able to engage in emotional processing with less neural and cognitive effort (Killgore & Yurgelun-Todd, 2007). Whereas those initial studies compared emotional/social-processing tasks to resting baseline, a subsequent study using a more complex auditory paradigm with a nonresting comparison condition found positive correlations between EI and some cortical emotional processing regions, while failing to find activation in more primitive regions such as the amygdala (Kreifelts, Ethofer, Huberle, Grodd, & Wildgruber, 2010). Recent structural neuroimaging studies have also suggested that measures of EI are related to gray matter volume within the vmPFC (Killgore et al., 2012; Koven, Roth, Garlinghouse, Flashman, & Saykin, 2011; Takeuchi et al., 2011), further supporting this region as a potentially important contributor to EI capacities.

A key aspect of social and emotional intelligence involves the ability to discriminate between individuals who are safe to approach and those who should be avoided (Winston, Strange, O'Doherty, & Dolan, 2002). The amygdala, one of the regions involved in the SMC, appears to be critical to this process. Prior work has shown that lesions to the amygdala can impair the ability to distinguish between trustworthy and untrustworthy individuals based on facial appearance (Adolphs, Tranel, & Damasio, 1998) and that such lesions often lead to inappropriate levels of social trust (Koscik & Tranel, 2011). Other work suggests that the vmPFC is also important to these types of

social judgments involving the ability to discriminate trustworthy from untrustworthy individuals (Koscik & Tranel, 2011). The vmPFC may be especially important in the process of integrating social, emotional, and cognitive information for determining trustworthiness decisions (Bzdok et al., 2012; Damasio, 1994). While the vmPFC is highly complex and involved in many aspects of social and emotional functioning, consistent evidence suggests that it is particularly activated when a person is considering the mental state or goals of another person (Frith, 2007). At present, no research has examined the relationship between EI capacities and the responsiveness of these brain regions to social trust stimuli.

The goal of the present study was to build upon the prior work evaluating the relationship between EI and SMC responses to simple static facial expressions by using a more ecologically valid task of processing dynamically changing facial attributes affecting trustworthiness judgments. In the "real world," facial features are rarely static, and clues to the intentions of others often come from the subtle changes in facial movement that occur during interpersonal exchanges. Here, during fMRI, we presented healthy participants with brief glimpses of faces that rapidly and dynamically changed in features associated with perceived trustworthiness. In each condition, faces either morphed from appearing highly trustworthy to neutral (i.e., decreasing in trustworthiness) or from low trustworthiness to neutral (i.e., increasing in trustworthiness). We hypothesized that higher scores on tests assessing both *Trait* and *Ability* models of EI would be associated with increased activation of the primary nodes of the SMC, particularly the vmPFC, in response to dynamically changing facial expressions indicative of decreasing trustworthiness.

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METHODS

Participants

Thirty-nine right-handed healthy volunteers (22 men; 17 women) ranging in age from 18 to 45 years ($M = 29.9$, $SD = 8.6$) participated in the study. All participants had completed at least 11 years of formal education ($M = 15.0$, $SD = 2.0$) and were native English speakers. The sample was racially diverse, including 24 individuals self-identified as Caucasian (61.5%), 8 as African American (20.5%), 4 as Asian American (10.3%), and 3 as "other" or "multi-racial" (7.7%). Volunteers were recruited via Internet advertisements and posted flyers within the Boston metropolitan area and were paid for their participation. Based on a detailed screening interview including questions adapted from the Structured Clinical Interview for DSM-IV Disorders (SCID-I/P) (First, Spitzer, Gibbon, & Williams, 2002), potential subjects were excluded for any history of Axis I mental disorder, neurological illness, head injury with loss of consciousness >30 minutes, sleep-related disorder, current use of psychotropic medications or substances known to affect functional neuroimaging, or current chemotherapy or radiation therapy. Written informed consent was obtained from all participants. The McLean Hospital Institutional Review Board approved the procedures for this study.

Materials and procedure

Following completion of the informed consent process, each participant completed computerized administrations of two well-validated commercially available tests of EI. The Bar-On Emotional Quotient Inventory (EQ-i) (Bar-On, 2002) was included as an index of *Trait* (or Mixed) EI. The EQ-i comprises 125 self-report items that yield a *Total EQ* score and five composite scores (i.e., *Interpersonal*, *Intrapersonal*, *Adaptability*, *Stress Management*, and *General Mood*). Individuals scoring high on the *Interpersonal* scale describe themselves as empathic and interpersonally aware, while those scoring high on the *Intrapersonal* scale describe themselves as self-aware, in-tune with their own emotions, and high in self-esteem. High scorers on the *Adaptability* scale perceive themselves as objective problem solvers who can adapt quickly to new situations. Those high in *Stress Management* describe themselves as well-controlled and unflappable in difficult or stressful situations. Individuals with high scores on the *General Mood* scale are self-described positive thinkers who are content with life. All EQ-i scores were scaled based on the general normative group, without adjustment for sex. To measure *Ability* EI, participants also completed the Mayer–Salovey–Caruso Emotional Intelligence Test (MSCEIT) (Mayer, Salovey, & Caruso, 2002), which includes 141 computer-administered items to assess individual skill at identifying, understanding, and using emotions. The test presents the participants with various types of stimuli that have to be rated for emotional characteristics or potential solutions that need to be selected to effectively address a given emotionally salient situation. The MSCEIT yields a *Total EI* score and two area scores, *Experiential* EI and *Strategic* EI. High scorers on *Experiential* EI are skilled at perceiving emotions and are effective at using that information to facilitate thought and

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performance. This area includes two subscales measuring abilities described as *Perceiving* and *Facilitating* emotions. The second area is *Strategic* EI. Those scoring high on this area have excellent capacity for understanding emotional information and are skilled at managing emotions in themselves and in others. *Strategic* EI comprises two subscales measuring abilities described as *Understanding* and *Managing* of emotions. MSCEIT scoring was based on the consensus scoring methods outlined in the manual (Mayer et al., 2002). Following completion of the EI tests, participants underwent structural and functional neuroimaging.

Dynamic facial trustworthiness task (DFTT)

During fMRI, participants completed a 4-minute task involving the visual perception of dynamically changing facial displays of trustworthiness. The face stimuli were selected from a freely available database (http://webscript.princeton.edu/~tlab/databases/database-6-trustworthiness-dataset/) of 100 computer-generated facial identities at three different trustworthiness levels. These faces were generated using the FaceGen 3.1 modeling program (http://facegen.com) and morphed to vary along the dimension of trustworthiness according to the methods described by Oosterhof and Todorov (2008). Briefly, that group used the computer-modeling program to generate 300 neutral faces of European origin, which were then subsequently rated by 29 judges on a 9-point scale of trustworthiness. By mathematically identifying the features in the faces that related to the dimension of trustworthiness, Oosterhof and Todorov (2008) randomly generated a new set of computer-generated faces varying systematically in these characteristics along a scale of standard deviation (SD) units. The stimuli used in the present study were pseudo-randomly drawn from a pool of 300 faces constructed from 100 distinct facial identities that varied along the dimension of trustworthiness at three different levels. All 100 facial identities were used, though only a subset was pseudo-randomly selected from each trustworthiness condition: 40 low trustworthy faces were selected from the 100 faces in the −3 SD data set; 60 neutral faces were selected from the 100 faces in the 0 SD data set; 40 high trustworthy faces were selected from the 100 faces in the 3 SD data set (see Figure 1).

During the DFTT, participants viewed brief presentations of faces that appeared to change expression. The DFTT comprised three different trial types: (1) *Decreasing Trustworthiness* (D; high trustworthy changing to neutral), (2) *Increasing Trustworthiness* (I; low trustworthy changing to neutral), and (3) *Neutral* (N; face identity change but no change in neutral trustworthiness level). Each trial was presented for 100 ms, with the first face (F1) shown for two screen refresh cycles (i.e., 33 ms), followed by the second face (F2) for four screen refresh cycles (i.e., 67 ms), and a 1400 ms intertrial interval (ITI). Thus, a new stimulus appeared every 1500 ms. This is essentially the same presentation speed as traditional digital video recording and gives the appearance of human-like movement on the face. During stimulus presentation, face identity change was not explicitly apparent, but the facial features appeared to subtly change expression.

Each of the F1 identities was always paired with a different identity at F2 in a pseudorandom fashion. All 100 face identities were used at F1 and 40 identities were recycled through to create a total of 140 trials, with the requirement that no F1 identity was ever

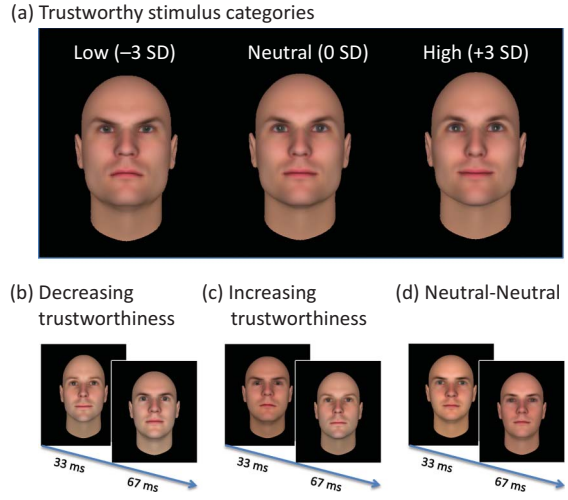

Figure 1. Examples of the stimuli used in the dynamic facial trustworthiness task (DFTT). Facial features were morphed along a continuum of trustworthiness according to the methods outlined by Oosterhof and Todorov (2008). (a) Three categories of faces were used, selected from those rated 3 SD below the mean in trustworthiness (left), those at the mean of trustworthiness (center), and those rated 3 SD above the mean in trustworthiness features (right). During the DFTT, pairs of faces were presented to give the appearance of subtle facial movement. (b) During the *Decreasing Trustworthiness* presentations, a high trustworthy face (+3 SD) was presented for 33 ms, followed by a neutral trustworthy face (0 SD) for 67 ms, which gave the impression of movement toward lesser trustworthiness. (c) During the *Increasing Trustworthiness* presentations, a low trustworthy face (–3 SD) was presented for 33 ms, followed by a neutral trustworthy face (0 SD) for 67 ms, which gave the impression of movement toward greater trustworthiness. (d) During the *Neutral* presentations, a neutral trustworthy face (0 SD) was presented for 33 ms, followed by a different neutral trustworthy face (0 SD) for 67 ms, which provided a control for potential movement effects associated with changing face identities independent of changes in trustworthiness.

shown twice for the same condition (e.g., if an identity was shown as a neutral expression at F1, it would either be shown again later as a high or low trustworthy face), and never appeared in two trials in a row. Notably, because the F2 faces were always neutral, both of the primary conditions reflect change from extremes in trustworthiness (high or low) toward the neutral intermediate appearance.

During the fMRI scan, the DFTT was presented in alternating 30-second blocks of the primary conditions flanked by 15 seconds of a crosshair fixation point (+) at either end of the task. The total duration of the task was 240 seconds with the following block order: +, N, D, I, N, I, D, N, +. Each of the seven 30-second blocks presented 20 trials (out of 140 trials total). To ensure that participants remained engaged with the task, they were required to make a simple button response with their dominant hand as quickly as possible each time the stimulus appeared on the screen.

Neuroimaging parameters

Participants underwent neuroimaging on a Tim Trio 3T scanner (Siemens, Erlangen, Germany) using a 12-channel head coil. Structural images were first acquired using a T1-weighted 3D MPRAGE sequence (TR/TE/flip angle = 2.1 s/2.25 ms/12°) over 128 sagittal slices (256 × 256 matrix) and a slice thickness of 1.33 mm (voxel size = 1 × 1 × 1.33 mm). T2*-weighted functional MRI scans were collected over 43 transverse slices (3.5 mm thickness, 0 skip) using an interleaved sequence (TR/TE/flip angle = 3.0 s/30 ms/90°), with 80 images collected per slice. Data were collected with a 22.4 cm field of view and a 64 × 64 acquisition matrix.

Image processing

The data were preprocessed and analyzed in SPM8 (http://www.fil.ion.ucl.ac.uk/spm/). According to standard algorithms, raw images were realigned to the first image in the series, unwarped, coregistered to each participant's high-resolution anatomical image, spatially normalized to the stereotaxic coordinate system of the Montreal Neurological Institute (MNI), spatially smoothed using an isotropic Gaussian kernel of 6 mm full-width at half-maximum (FWHM), and resliced to 2 × 2 × 2 mm voxels. The time series data were convolved with the SPM8 canonical hemodynamic response function, the AR(1) option was used to correct for serial autocorrelation, and low-frequency confounds were removed with the standard 128-second high-pass filter. Individual scans were visually inspected using the Artifact Detection Tool (http://www.nitrc.org/projects/artifact_detect/). Scan volumes exceeding 3 SD in mean global intensity or scan-to-scan motion that exceeded 1.0 mm were regressed out of the first-level analysis as a nuisance covariate.

Statistical analysis

For each participant, a general linear model was created to identify the regions showing greater task-related activation to the three primary task conditions, including the *Decreasing Trustworthiness, Neutral Trustworthiness,* and *Increasing Trustworthiness* blocks compared to the implicit baseline. Next, contrasts were created by comparing the *Decreasing Trustworthiness > Neutral*, *Increasing Trustworthiness > Neutral*, and *Decreasing Trustworthiness > Increasing Trustworthiness* conditions. The contrast images created from this analysis for each participant were carried forward as the dependent variables within second-level random effects regression analysis models with EI score as the predictor variable. Separate regression models were created for the EQ-i and MSCEIT predictors to examine the association between emotional intelligence and brain responses to changing levels of trustworthiness. Finally, based on the lack of amygdala findings for some of the *a priori* analyses, we undertook a series of additional *post-hoc* quadratic trend analyses to explore the possibility that some key regions might respond to trustworthiness in a curvilinear manner. Based on our *a priori* hypotheses, bilateral search territories including the primary emotional regulation and emotional response nodes of the SMC were created using the Wake Forest University PickAtlas Utility (Maldjian, Laurienti, Kraft, & Burdette, 2003) and the boundaries defined by the Automated Anatomical Labeling Atlas (Tzourio-Mazoyer et al., 2002). We focused on the bilateral gyrus rectus, ACC, and amygdala and insula bilaterally. Analyses were subjected to small volume correction for multiple comparisons within each search territory at $p < .001$ (uncorrected), $p < .10$, false discovery rate (FDR) corrected, k (extent) ≥ 10 contiguous voxels.

RESULTS

Primary comparisons

As evident in Table 1 and Figure 2, the *Decreasing Trustworthiness > Neutral* comparison was associated with significant activation within several regions of

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TABLE 1
Locations of maximally activated voxels during primary trustworthiness comparisons

Comparison region	Cluster size (voxels)	MNI coordinates X	y	z	SPM {t}
Decreasing trustworthiness > Neutral					
R insula	16	46	−4	−6	3.83
Gyrus rectus	13	0	38	−16	3.68
R amygdala	10	24	2	−14	3.62
R insula	17	36	−20	12	3.55
Increasing trustworthiness > Neutral					
L amygdala	14	−20	−2	−20	4.81
R insula	40	34	−10	16	4.10
Gyrus rectus	15	0	30	−16	3.94

Notes: All analyses significant at $p < .001$, uncorrected; $p < .10$ (FDR, small volume corrected). R, right; L, left.

the SMC, including the right posterior insula, right amygdala, and medial gyrus rectus. On the other hand, the contrast between *Increasing Trustworthiness > Neutral* was associated with increased activation of the right anterior insula, left amygdala, and medial gyrus rectus (see Table 1 and Figure 2). These findings suggest that changes in facial trustworthiness are associated with significant activation of regions of the hypothesized neurocircuitry. However, when the *Increasing Trustworthiness* and *Decreasing Trustworthiness* conditions were directly contrasted, there were no regions showing significant differences.

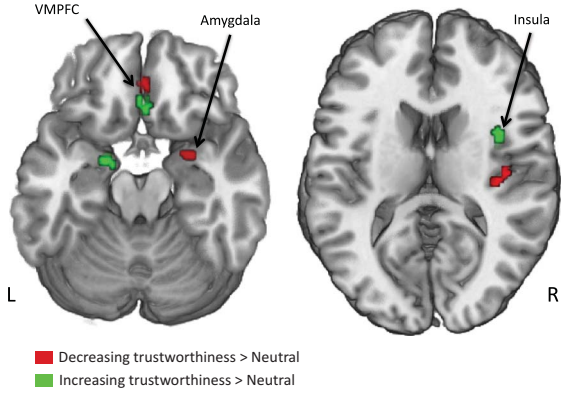

Figure 2. Regions of functional activation associated with the contrasts between *Decreasing Trustworthiness > Neutral* (red) and *Increasing Trustworthiness > Neutral* (green). Significance was evaluated using a small volume correction for multiple comparisons within each search territory at $p < .001$ (uncorrected), $p < .10$, FDR corrected, k (extent) ≥ 10. The image shows that the *Decreasing Trustworthiness > Neutral* contrast was associated with increased activation of the ventromedial prefrontal cortex (vmPFC) and right amygdala (left image), and posterior insula (right image). The *Increasing Trustworthiness > Neutral* contrast was also associated with increased activation of the vmPFC as well as the left amygdala (left image) and anterior insula (right image).

Bar-On EQ-i correlations

Within the hypothesized search regions, we found that Total EQ-i and subscale scores were not associated with functional activation in response to the *Decreasing Trustworthiness* or *Increasing Trustworthiness* conditions or the primary condition contrasts.

MSCEIT correlations

Total scores on the MSCEIT were not significantly correlated with activation during the *Increasing Trustworthiness* condition, but were positively correlated with a cluster of 22 activated voxels within the right vmPFC (i.e., gyrus rectus) [$x = 6$, $y = 32$, $z = -16$], $t(37) = 4.37$, $p = .09$ (FDR small volume corrected), during the *Decreasing Trustworthiness* condition (see Figure 3). Additionally, the beta parameters for each participant were extracted from the activated cluster region and correlated with the two area and four branch scores of the MSCEIT to identify the components of EI that contributed most to the observed effects. Only correlations below $p < .005$ are interpreted to avoid inflation of type I error. As detailed in Table 2, the *Strategic* EI area scores and the *Understanding Emotions* branch scores were found to correlate positively with the activated cluster within the vmPFC.

Additionally, we examined the correlation between MSCEIT scores and the primary condition contrasts. Whereas MSCEIT scores were unrelated to responses to the *Increasing Trustworthiness > Neutral* and *Decreasing Trustworthiness > Neutral* contrasts, MSCEIT scores were found to correlate positively with activation within the emotional regulation region of rostral ACC during the *Decreasing* versus *Increasing Trustworthiness* contrast (see Figure 3b). This cluster was located directly rostral to the genu of the corpus callosum in the right hemisphere [$x = 14$, $y = 44$, $z = 12$], $t(37) = 4.05$, $p = .07$ (FDR small volume corrected), with a cluster extent of 25 voxels. No other regions within the search territories were associated with MSCEIT scores. There were no significant correlations associated with the reverse contrast (i.e., *Increasing Trustworthiness* versus *Decreasing Trustworthiness*). Again, the beta parameters were extracted from the activated cluster region and correlated with MSCEIT subscale scores. This analysis

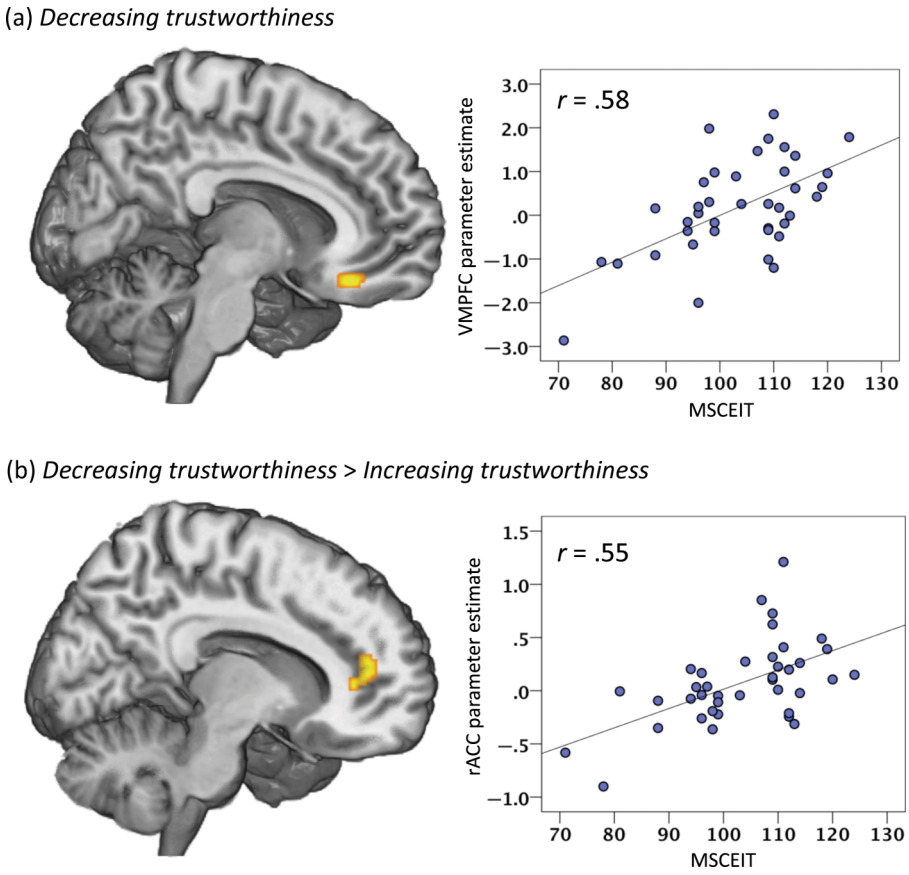

Figure 3. Sagittal brain slices showing significant clusters of activation that correlated with EI, $p < .10$ (small volume corrected), $k \geq 10$. (a) Total scores on the MSCEIT were positively correlated with responses of the ventromedial prefrontal cortex (vmPFC) for the contrast of *Decreasing Trustworthiness* versus implicit baseline (left) [$x = 6$, $y = 32$, $z = -16$]. For visualization purposes, the scatterplot (right) shows the relationship between MSCEIT scores and the first eigenvariate extracted for the entire correlated cluster. (b) Total EI scores on the MSCEIT were positively correlated with responses within the rostral ACC (rACC) for the contrast of *Decreasing* versus *Increasing Trustworthiness* (left) [$x = 14$, $y = 44$, $z = 12$]. For visualization purposes, the scatterplot (right) shows the relationship between MSCEIT scores and the first eigenvariate extracted for the entire correlated cluster.

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showed that the rostral ACC cluster was significantly correlated with the two area scores of *Experiential* and *Strategic* EI (see Table 2).

Nonlinear responses

Because of the important role of the amygdala in social and emotional processing of facial expressions and recent evidence suggesting that the amygdala might show a nonlinear pattern of responses to facial trustworthiness (Said, Baron, & Todorov, 2009), we undertook a *post-hoc* analysis to examine potential nonlinear responses of the hypothesized neurocircuitry in the present sample. A test for quadratic trend in the data was evaluated across the three conditions of *Increasing Trustworthiness*, *Neutral*, and *Decreasing Trustworthiness*. Table 3 presents the results of the test for quadratic trend. As evident in Figure 4, significant quadratic trend was found for responses within the right amygdala, vmPFC, and right insular cortex, suggesting significantly greater responses within these regions to images showing either *Increasing* or *Decreasing Trustworthiness* relative to *Neutral* images.

DISCUSSION

The ability to detect dynamic changes in facial cues signifying the intentions of others is vital to human survival. Consistent with prior work (Winston et al., 2002), we found that changes in facial cues reflecting trustworthiness were associated with increased responsiveness of key regions of the SMC, including the vmPFC, amygdala, and insula. We also found that in comparison to a condition of no change in facial

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TABLE 2
Correlations between emotional intelligence subscales and cluster activation within the vmPFC and rostral ACC

			fMRI cluster correlation	
Emotional intelligence scale		*Mean (SD)*	*vmPFC [6, 32, –16]*	*rostral ACC [14, 44, 12]*
EQ-i total		103.36 (14.06)		
	Interpersonal	101.44 (15.76)	.317	.158
	Intrapersonal	104.26 (15.66)	.166	.217
	Stress Management	103.31 (12.41)	.168	.138
	Adaptability	102.18 (12.63)	.075	.261
	General mood	104.05 (12.11)	.352	.161
MSCEIT total		103.23 (11.82)		
	Experiential	105.92 (14.75)	.364	.448*
	Perceiving	105.56 (13.89)	.183	.341
	Facilitating	104.44 (14.39)	.426	.414
	Strategic	99.18 (9.67)	.595*	.451*
	Understanding	100.69 (11.89)	.600*	.390
	Managing	97.18 (8.28)	.413	.420

Note: $^{*}p < .005$

TABLE 3
Locations of maximally activated voxels during analysis of quadratic trend across increasing, neutral, and decreasing trustworthiness conditions

		MNI coordinates			
Region	*Cluster size (voxels)*	*x*	*y*	*z*	*SPM {t}*
R insula	27	46	−4	−6	4.41
R insula	66	36	−10	18	3.86
Gyrus rectus	30	0	30	−16	3.65
R amygdala	19	24	0	−16	3.44

Notes: All analyses significant at $p < .001$, uncorrected; $p < .10$ (FDR, small volume corrected). R, right; L, left.

trustworthiness, the activation of these SMC regions was increased regardless of whether the changes in facial attributes involved decreasing or increasing levels of trustworthiness. Given the importance of social trustworthiness judgments to human survival, we further hypothesized that this capacity might be directly related to the construct of EI, a complementary form of intelligence that has been posited to depend critically on the underlying neural system of the SMC (Bar-On et al., 2003). We found that higher scores on one of two standardized and widely used measures of EI were associated with increased activation of specific nodes of the SMC in response to facial feature changes indicative of decreasing trustworthiness during fMRI. Whereas scores on the EQ-i, a self-report *Trait* measure of EI, were unrelated to responsiveness of the SMC to changing trustworthiness, better performance on the MSCEIT, an *Ability* measure of EI, was associated with increased responsiveness of the vmPFC and rostral ACC to these same dynamic changes in facial cues signifying untrustworthiness. Other regions comprising the SMC were not significantly correlated with either index of EI during this task. These findings provide support for the hypothesized role of some components of the SMC in EI, while emphasizing in particular the role of discrete regions of the medial prefrontal cortex and ACC in these capacities.

The SMH posits a central role of the vmPFC in integrating somatic emotional signals with ongoing cognition to guide decision-making (Damasio, 1994, 1996). Indeed, Bar-On and colleagues have suggested that the vmPFC is a critical node of the SMC involved in EI (Bar-On et al., 2003). Presently, we found that the responsiveness of the vmPFC to dynamic facial cues indicating decreasing trustworthiness was positively correlated with MSCEIT *Total EI*, *Strategic EI*, and the *Understanding Emotions* scale. Our finding that individuals with higher MSCEIT scores showed greater responsiveness of the vmPFC to these subtle facial displays indicative of potential threat or dubious character is consistent with a large body of neuroscience evidence pointing to the role of that region in emotional appraisal and emotion regulation. For instance, the process of making inferences about the intentions and emotional states of others, a capacity known as Theory of Mind (ToM), is correlated with increased gray matter volume (Lewis, Rezaie, Brown, Roberts, & Dunbar, 2011) and increased functional activation within the vmPFC (Sebastian et al., 2012). Dysfunction of the vmPFC, whether through actual brain lesions (Leopold et al., 2012) or via disruption of ongoing activity by slow repetitive transcranial magnetic stimulation (rTMS) (Lev-Ran, Shamay-Tsoory, Zangen, & Levkovitz, 2012), also impairs affective ToM performance. Similarly, some evidence suggests that processing of the vmPFC can be disrupted by naturally occurring stresses such as sleep deprivation (Thomas et al., 2000), a process that has been shown to impair emotionally guided moral judgments (Killgore, Killgore, et al., 2007), risky decisions (Killgore, Balkin, & Wesensten, 2006), and *Trait* EI (Killgore, Kahn-Greene, et al., 2007).

Interestingly, we found that the vmPFC responded to changes in facial characteristics indicating either decreasing or increasing trustworthiness. Decreasing

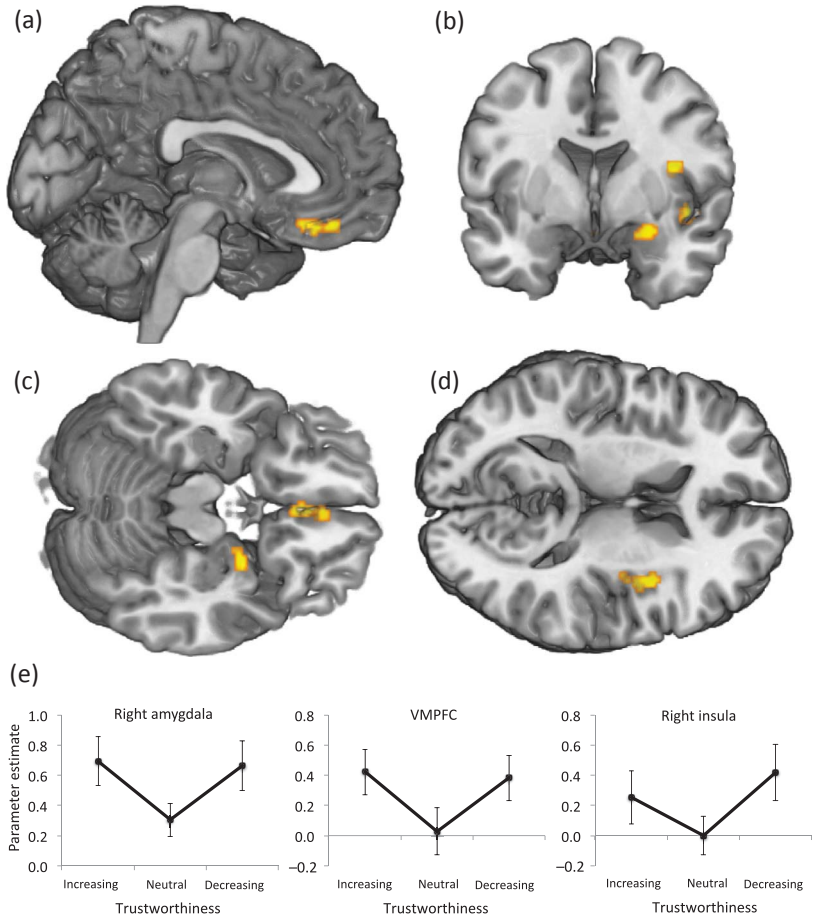

Figure 4. A trend analysis revealed a quadratic pattern of responsiveness across the three trustworthiness conditions of *Increasing Trustworthiness*, *Neutral*, and *Decreasing Trustworthiness* within key regions of interest, including the ventromedial prefrontal cortex (vmPFC), right amygdala, and right insula. Clusters showing this quadratic pattern can be seen on the (a) sagittal slice (vmPFC), (b) coronal slice (right amygdala and insula), slices showing (c) inferior axial (vmPFC and right amygdala), and (d) superior axial (right insula) perspectives. (e) Parameter estimates from the right amygdala, vmPFC, and right insula were extracted from the displayed clusters and plotted for visualization.

trustworthiness was associated with increased activation of a cluster within the gyrus rectus that was centered only about 8 mm anterior to the cluster associated with viewing faces showing increasing trustworthiness. Furthermore, *post-hoc* analyses revealed that a cluster encompassing this same region showed a quadratic pattern of activation, responding to changes in trustworthiness in either direction. Some evidence suggests that the vmPFC represents the reward value of stimuli and plays a role in learning when reinforcement contingencies have changed (Blair, 2008). Kringelbach and Rolls propose that the primary role of the medial orbitofrontal cortex is to represent the reward value of stimuli and to identify when stimuli are no longer reinforcing (Kringelbach & Rolls, 2004), which corresponds well with our findings that this medial prefrontal region was activated when the target faces changed in level of trustworthiness, regardless of whether that change was increasing or decreasing. The vmPFC is also associated with emotional control, including voluntary regulation of negative affect and the corresponding dampening of amygdala responses (Urry et al., 2006). Importantly, the vmPFC has been shown to play a role in the maintenance of extinction memory following fear conditioning in humans (Milad et al., 2007), leading to inhibition of fear responses when encountering a previously feared stimulus (Milad & Quirk, 2002). A recent meta-analysis of neuroimaging studies also showed that patients with posttraumatic stress disorder (PTSD) show abnormal deactivations of the vmPFC (Etkin & Wager, 2007). These findings suggest that the vmPFC may be a key component of resilience and the ability to sustain mental and emotional health following exposure to traumatic events. Our results suggest that this same resilience system is engaged to a greater extent during facial trustworthiness assessments among individuals

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showing higher *Ability* EI, as measured by the MSCEIT.

In addition to the vmPFC, a second key region of the SMC, the amygdala, was also activated by dynamic facial cues related to trustworthiness. Direct contrasts between each dynamic trustworthiness condition versus the neutral condition revealed left amygdala activation to *Increasing Trustworthiness* and right amygdala activation to *Decreasing Trustworthiness*. This is consistent with prior research showing that the process of evaluating facial trustworthiness activates the amygdala (Engell, Haxby, & Todorov, 2007; Rule, Krendl, Ivcevic, & Ambady, 2013; Winston et al., 2002), and with a large literature suggesting that the amygdala is involved in detecting facial cues associated with threat and fear (Killgore & Yurgelun-Todd, 2004, 2005; Phelps et al., 2001; Whalen et al., 1998), as well as other emotional expressions (Fitzgerald, Angstadt, Jelsone, Nathan, & Phan, 2006). Furthermore, such responsiveness of the amygdala to trustworthiness information appears to be more strongly correlated with facial features that are commonly agreed upon by consensus raters as a signal of untrustworthiness than to idiosyncratic judgments unique to the individual perceiver (Engell et al., 2007; Rule et al., 2013). Here, we used a standardized set of facial identities that varied on structural facial features that had been previously shown to covary with consensus ratings of trustworthiness (Oosterhof & Todorov, 2008), so we are reasonably confident that the amygdala responses we observed are associated with the differing levels of trustworthiness of the faces. However, it is important to consider that there may be other factors that may contribute to how the facial stimuli were interpreted. Although all of the computer-generated face stimuli used in the present study were designed to display a "neutral" emotional expression, there is some evidence to suggest that structural facial features that resemble emotional expressions can actually affect trait judgments in a systematic manner (Said, Sebe, & Todorov, 2009). For example, Said and colleagues (2009) compared human trait ratings of neutral faces to a computerized face classification system to identify features in the same faces that resembled particular emotions. They found that neutral faces tended to be rated as more positive if they had structural features that resembled expressions of happiness, while features that resembled expressions of anger tended to be judged as more threatening (Said, Sebe, et al., 2009). This tendency to overgeneralize emotions to neutral expressions based solely on the structural features of the face has been shown to affect complex impression formation (Adams, Nelson, Soto, Hess, & Kleck, 2012) and could have contributed to the current findings by giving the visual impression of changing emotion rather than changing trustworthiness per se. Additional research that disentangles these components will be necessary to provide further clarification.

The present findings also need to be considered in light of recent findings suggesting that amygdala responses to facial trustworthiness may not follow a linear pattern (Said, Baron, et al., 2009). Notably, Said, Baron, and Todorov (2009) demonstrated that amygdala responses to facial trustworthiness cues showed a quadratic rather than linear trend, with greater responsiveness to faces judged to be at the extremes of perceived trustworthiness (i.e., either high or low) (Said, Baron, et al., 2009), a finding that has since been replicated (Mattavelli, Andrews, Asghar, Towler, & Young, 2012). Accordingly, we conducted a *post-hoc* analysis of our data to test for nonlinear responses. Our analysis also showed this quadratic pattern for the amygdala, as well as for other SMC regions such as the vmPFC and insula when perceiving facial movement communicating information about potential trustworthiness. Overall, changes in trustworthiness in either direction led to increased activation within specific regions of the SMC, including the amygdala, vmPFC, and insula. In fact, there was no significant difference in the responsiveness of SMC regions when the *Increasing* and *Decreasing Trustworthiness* conditions were directly contrasted, suggesting similar levels of activation.

Although the hypothesized regions showed similar levels of increased activation to both trustworthiness conditions, it was also of interest to examine whether the differential response to decreasing versus increasing trustworthiness of faces might be associated with EI. We found that MSCEIT scores were positively correlated with differential activation of the rostral ACC to displays of decreasing versus increasing trustworthiness. This is important, as the rostral ACC is a brain region that is strongly implicated in error detection (Bush, Luu, & Posner, 2000; Taylor et al., 2006), emotional control (di Pellegrino, Ciaramelli, & Ladavas, 2007), assessing affective salience (Klumpp et al., 2011), and resolving emotional conflict (Etkin et al., 2006). Activation of the rostral ACC is associated with enhanced processing of threatening faces when attentional resources are limited (De Martino, Kalisch, Rees, & Dolan, 2009). Abnormal responses within the rostral ACC have also been reported in a number of psychopathological conditions involving emotional dysregulation such as depression (Cooney, Joormann, Eugene, Dennis, & Gotlib, 2010), posttraumatic stress disorder (Hopper, Frewen, van der Kolk, & Lanius, 2007; Kim et al., 2008), and high trait anxiety (Klumpp et al., 2011). There is evidence to suggest

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that the rostral ACC is functionally connected with the amygdala, and that increased rostral ACC activation is frequently associated with corresponding reduction of amygdala responses (Etkin et al., 2006). In our study, we used a task that assesses brain responses to dynamic changes in facial trustworthiness, a social signal that could have important survival implications. This change in the target expression from one of high trustworthiness to one of lesser trustworthiness would be expected to require a rapid reassessment of the intention of the target face, leading to engagement of error detection and affective conflict monitoring regions of the rostral ACC once an initial face assessment was determined to be incorrect. Our finding that increasing rostral ACC activation to these cues correlated with higher scores on the MSCEIT is consistent with the putative role of this region in assessing affective salience, resolving affective conflict, and preparing the individual for a potential response. These findings suggest that individuals with higher *Ability* EI, including both the *Experiential* and *Strategic* aspects, are more sensitive and responsive to these subtle facial cues within this key affective regulating region, potentially conferring a survival advantage.

When considered together, the present findings suggest that greater *Ability* EI is associated with increased responsiveness of the vmPFC region to dynamic facial cues that could communicate the need for increased concern, vigilance, and a potential behavioral response. Higher scores on *Ability* EI and, in particular, subscales assessing the capacity to perceive, respond to, and control emotions (*Experiential* EI), and the ability to understand and direct emotions (*Strategic* EI) were associated with increased responsiveness of the rostral ACC. Because of the role of the rostral ACC in error detection and affective conflict monitoring (Etkin et al., 2006), these findings suggest that greater EI is associated with enhanced responsiveness of these error detection and response systems. However, while the present findings provide partial support to the hypothesized network of the SMC in EI as suggested by Bar-On et al. (2003), there were several regions of this system that failed to show correlated responses with either the EQ-i or MSCEIT. Specifically, although changes in facial trustworthiness were reliably associated with amygdala responses, this activation pattern did not correlate significantly with EI. This was unexpected, but could be related to the nonlinear nature of the amygdala responses, limited power to detect such relationships, or some other indeterminate aspect of our stimuli or experimental design. We did find, however, that *Ability* EI was reliably associated with activation within the medial prefrontal cortex and rostral ACC, suggesting that these emotion regulating and integrating regions appear to play an important role in these capacities. Further work will also be necessary to determine the behavioral implications of these findings, such as whether activation of these EI correlated regions actually confers performance advantages on other emotionally relevant tasks or is related to resilience under actual stressful circumstances. On the other hand, *Trait* EI was not significantly correlated with measured responses within the SMC during the trustworthiness conditions.

CONCLUSION

Greater EI was associated with increased responsiveness of the medial prefrontal cortex during a socially relevant dynamic face perception task, providing partial support for the role of the SMC in these capacities. Discrete nodes of the SMC, including the vmPFC and rostral ACC, were specifically correlated with *Ability* EI capacities, while *Trait* EI was not significantly related to the responsiveness of the hypothesized regions during dynamic facial displays communicating trustworthiness information. Overall, systematic differences in EI capacities appear to be significantly related to the responsiveness of higher order emotion assessment and regulation regions of the medial prefrontal cortex and rostral anterior cingulate.

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Self-Reported Sleep Correlates with Prefrontal-Amygdala Functional Connectivity and Emotional Functioning

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Study Objectives: Prior research suggests that sleep deprivation is associated with declines in some aspects of emotional intelligence and increased severity on indices of psychological disturbance. Sleep deprivation is also associated with reduced prefrontal-amygdala functional connectivity, potentially reflecting impaired top-down modulation of emotion. It remains unknown whether this modified connectivity may be observed in relation to more typical levels of sleep curtailment. We examined whether self-reported sleep duration the night before an assessment would be associated with these effects.

Design: Participants documented their hours of sleep from the previous night, completed the Bar-On Emotional Quotient Inventory (EQ-i), Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), and Personality Assessment Inventory (PAI), and underwent resting-state functional magnetic resonance imaging (fMRI).

Setting: Outpatient neuroimaging center at a private psychiatric hospital.

Participants: Sixty-five healthy adults (33 men, 32 women), ranging in age from 18-45 y.

Interventions: N/A.

Measurements and Results: Greater self-reported sleep the preceding night was associated with higher scores on all scales of the EQ-i but not the MSCEIT, and with lower symptom severity scores on half of the psychopathology scales of the PAI. Longer sleep was also associated with stronger negative functional connectivity between the right ventromedial prefrontal cortex and amygdala. Moreover, greater negative connectivity between these regions was associated with higher EQ-i and lower symptom severity on the PAI.

Conclusions: Self-reported sleep duration from the preceding night was negatively correlated with prefrontal-amygdala connectivity and the severity of subjective psychological distress, while positively correlated with higher perceived emotional intelligence. More sleep was associated with higher emotional and psychological strength.

Keywords: Emotional intelligence, psychopathology, functional connectivity, prefrontal cortex, amygdala

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INTRODUCTION

Sleep loss has a profound effect on elementary cognitive processes such as simple alertness, psychomotor vigilance, and response speed.[1,2] However, there is now growing literature suggesting that sleep loss also has significant effects on mood and emotional functioning.[3-6] Sleep deprivation increases physiological reactivity in response to emotional stressors,[7] reduces the psychological threshold for coping with stress,[8] and even leads to poorer frustration tolerance and an altered perception of the motives of others.[9] Sleep deprivation impairs emotionally-based decision making[10] and the ability to use emotions to effectively guide moral judgment.[11-13] Moreover, extended sleep deprivation is associated with significant declines on standardized measures of coping skills and emotional intelligence (EI), a set of capabilities and traits that involve the ability to understand and regulate emotions.[14] In one study, sleep deprivation was associated with significant declines in the ability to understand emotional responses in others and led to reduced self-reported interpersonal skills, including degraded empathy, stress management capacities, and impulse control.[14] Even in healthy individuals, prolonged sleep deprivation leads to significant worsening on several standardized indices of psychopathology, including scales measuring somatic complaints, anxiety, depression, and paranoia.[15] In short, when sleep is lacking, affective functioning becomes poorly regulated and emotionally salient stimuli may have a greater influence over cognitive processes.

Although the causal mechanisms for these mood and emotional changes are poorly understood, some evidence suggests that they may emerge from measurable alterations in brain functioning that occur following insufficient sleep or total sleep deprivation. Early brain imaging studies suggested that sleep deprivation is associated with significant declines in global cerebral energy metabolism.[16] These declines are particularly notable within the prefrontal cortex, including the ventromedial regions,[16] which are important to emotional regulation and behavioral control.[17,18] More recent studies using functional magnetic resonance imaging (fMRI) suggest that the increased emotional responsiveness during sleep deprivation may be due in part to changes in the strength of functional connectivity between the emotional regulating regions of the medial prefrontal cortex and the amygdala, a structure involved in triggering emotional responses to salient stimuli.[19] Other evidence suggests that this modified connectivity between the emotional regulation and emotional responsive regions of the brain may contribute to altered responsiveness to both positive and negatively

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valenced stimuli under conditions of sleep deprivation.[20] Speculatively, such an alteration in the functional balance between these regions may affect higher-order regulation of emotional processes, which could conceivably be observed as declines in EI and even the emergence of symptoms of psychopathology.

Although the effects of total sleep deprivation on EI capacities and symptoms of psychopathology have been well documented within the confines of highly controlled laboratory environments,[14,15] virtually no information exists regarding the relation between typical nightly sleep duration in the natural home environment and changes in emotional capacities. A recent fMRI study by our group demonstrated that self-reported sleep duration was reliably associated with next-day resting-state functional connectivity within the brain's default mode network, which includes regions of the medial prefrontal cortex and posterior cingulate cortex.[21] It is, therefore, possible that subtle reductions in nocturnal sleep, even well within the normal range obtained by most healthy individuals on any given night, may be sufficient enough to be associated with variations in EI and psychopathology. Accordingly, we collected self-report information regarding typical and recent sleep patterns as well as standardized indices of EI and psychopathology in a sample of healthy participants. Based on our prior findings of changes in EI and psychopathology during total sleep deprivation, and other work showing altered prefrontal-amygdala connectivity under similar conditions, we hypothesized that (1) greater amounts of sleep reported for the night before an assessment would correlate with higher scores on measures of EI and lower scores on indices of psychopathology, (2) more sleep would be associated with increased negative functional connectivity between the ventromedial prefrontal cortex (vmPFC) and amygdala (i.e., suggesting a negative relationship between the intrinsic activation patterns of the amygdala and vmPFC in rested individuals), and (3) the strength of this negative functional connectivity would be directly related to higher EI and lower psychopathology scores in healthy adults.

METHOD

Participants

Sixty-five healthy adults (33 men, 32 women), ranging in age from 18-45 y (mean [M] = 30.2; standard deviation [SD] = 8.0) were recruited via internet advertisements and flyers from the vicinity of the Boston metropolitan area. Participants were screened via telephone interview using standard psychiatric diagnostic criteria[22] and deemed to be free from any history of serious medical illnesses, including neurological, Axis I psychiatric, or substance use disorders (including alcohol and illicit drugs), or evidence of clinically significant sleep disorders. All 65 participants provided complete data for the questionnaires. A subsample (n = 58) of this group (29 men, 29 women), ranging in age from 18-45 y (M = 30.5; SD = 8.0) also provided usable resting state fMRI data that were correlated with the questionnaire data. All participants provided written informed consent and were compensated for their time. This research protocol was reviewed and approved by the Institutional Review Board of McLean Hospital.

Materials and Procedure

Sleep Questionnaires

Participants arrived at the laboratory between 09:00 and 11:00 in the morning. Upon arrival, each participant completed a brief questionnaire about their recent sleep schedule and typical habits. The primary question of interest simply asked participants: "How much sleep did you get last night?" This variable, identified as *Sleep Last Night*, was scored in hours. The questionnaire also included queries about typical bedtimes and wakeup times for weekdays and weekends. Based on this information an estimated *Sleep Debt* variable was also computed by subtracting a weighted average of typical sleep on weekdays and weekends from the *Sleep Last Night* variable. Additionally, participants also reported whether they had problems falling or staying asleep as a simple index of insomnia complaints. Participants also completed the Morningness-Eveningness Questionnaire (MEQ).[23] Higher scores on the MEQ indicate a preference for earlier rise times and bedtimes and a tendency to function most effectively earlier in the day.

EI Scales

Participants completed two normed, well-validated, and commercially available measures of EI. As a mixed model, or *Trait* measure of EI, participants completed the Bar-On Emotional Quotient Inventory (EQ-i).[24] A 125-item self-report measure, the EQ-i provides a global score of EI (*Total EQ*), as well as five composite subscales measuring various self-perceived facets of the construct, including the ability to relate well with others (*Interpersonal*), emotional self-awareness and self-confidence (*Intrapersonal*), emotional flexibility and problem solving (*Adaptability*), ability to cope with stress (*Stress Management*), and general optimism and contentedness (*General Mood*). The raw EQ-i scores were transformed into standard scores based on the general population norms provided by the test manual and scoring program.[24] We also tested *Ability* EI using the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT),[25] a performance-based test of the capacity to reason about and solve emotional problems. This test yields a *Total EI* score, as well as two Area scores. The first Area score measures the ability to perceive emotions and use that information to facilitate thought (*Experiential EI*), and is composed of two branch scores known as *Perceiving* and *Facilitating* emotions. The second Area score measures the ability to understand and control emotions (*Strategic EI*), and is composed of two branch scores known as *Understanding* and *Managing* emotions. Raw MSCEIT scores were transformed into standardized scores based on the general consensus scoring method (as opposed to expert consensus method) described in the test manual.[25] Based on our prior published findings,[14] we specifically hypothesized that greater sleep and increased functional connectivity variables would correlate with *Total EQ*, *Intrapersonal*, *Interpersonal*, and *Stress Management* variables from the EQ-i.

Psychopathology Scale

As a measure of psychopathologic symptom severity, participants completed the Personality Assessment Inventory (PAI), an objective measure that includes 344 statements that

are self-rated on a four-point Likert scale.[26] The PAI includes 18 primary scales: *Somatic Complaints* (SOM), which measures concerns about health and physical functioning; *Anxiety* (ANX), which measures general tension and negative affect; *Anxiety Related Disorders* (ARD), which assesses symptoms related to specific anxiety disorders; *Depression* (DEP), which measures common cognitive, affective, and physiological symptoms of depression; *Mania* (MAN), which measures common clinical features of hypomania and mania; *Paranoia* (PAR), which assesses paranoid features such as hypervigilance, resentment, and feelings of persecution; *Schizophrenia* (SCZ), which measures a variety of symptoms including unusual beliefs and perceptions, social deficits, and attentional problems; *Borderline Features* (BOR), which assesses broad problems with interpersonal functioning; *Antisocial Features* (ANT), which taps into constructs of adventuresomeness, egocentricity, lack of empathy, and antisocial attitudes; *Alcohol Problems* (ALC), which assesses behaviors and consequences associated with alcohol abuse and dependence; *Drug Problems* (DRG), which measures attitudes and behaviors related to drug abuse and dependence; *Aggression* (AGG), a scale assessing general attitudes conducive to aggressive behavior; *Suicidal Ideation* (SUI), which assesses thought content related to death and suicide; *Stress* (STR), which provides an index of current life stressors; *Nonsupport* (NON), which measures the perception of unavailability of social support; *Treatment Rejection* (RXR), which assesses a tendency to be satisfied with the current status quo and a disinterest in or unwillingness to participate in therapy; *Dominance* (DOM), an interpersonal scale that measures a bipolar dimension of dominance (versus submissiveness); and *Warmth* (WRM), an interpersonal scale that measures a bipolar dimension of empathy (versus rejecting). Raw scores on the PAI were transformed to standardized T-scores based on a normative sample of 1,000 community-dwelling adults as described in the test manual.[26] According to prior published findings,[15] we specifically hypothesized that greater sleep and increased functional connectivity variables would correlate with *SOM, ANX, DEP,* and *PAR* variables from the PAI.

Neuroimaging

Participants underwent a 6-min, eyes open, resting state fMRI scan between 13:00 and 15:00 in the afternoon. Due to the nature of the resting state scan (i.e., nontask engagement—mind wandering), we did not ask the participants to engage in any sort of vigilance control task during this data collection. Images were collected on a 3T Siemens Tim Trio scanner (Erlangen, Germany) and fitted with a 12-channel head coil. Standard structural images were acquired first for use in spatial normalization and for removal of tissue confounds. These images comprised a T1-weighted three-dimensional MPRAGE sequence (TR/TE/flip angle = 2.1s/2.25ms/12°), which yielded 128 sagittal slices (256 × 256 matrix) with a slice thickness of 1.33 mm and a voxel size of 1.33 × 1 × 1 mm. For the resting state scan, 180 images were collected (3.5-mm thickness, no skip; 22.4 cm field of view; 64 × 64 acquisition matrix) over 34 transverse interleaved slices using a T2*-weighted blood oxygen level dependent (BOLD) echoplanar imaging (EPI) sequence (TR/TE/flip angle = 2.0 sec/30 msec/90°).

Image Processing

Resting state data were preprocessed using standard algorithms in SPM8, including motion correction, slice-timing correction, anatomical co-registration, spatial normalization, and spatial smoothing (full width at maximum [FWHM] = 6 mm). Voxels were resliced to 2 × 2 × 2 mm. The time series of resting state data was analyzed using the Functional Connectivity (CONN) Toolbox[27] version 13i (http://www.nitrc.org/projects/conn). As part of this process, the data were bandpass filtered (0.008, 0.10 Hz), and corrected for physiological noise using the *aCompCor* strategy.[28] Major confounders were removed using principal components analysis to control for the effects of white matter and cerebrospinal fluid, and motion parameters were included as nuisance covariates; the resultant residual BOLD time series was used for subsequent functional connectivity analyses. To examine functional connectivity, four regions of interest (ROIs) were placed (right ventromedial prefrontal cortex [vmPFC] seed regions = left and right gyrus rectus; target regions = left [220 voxels] and right [248 voxels] amygdala) using the Automated Anatomical Labeling (AAL) Atlas.[29] The gyrus rectus was selected based on recent work suggesting that gray matter of this region is associated with EI traits[30] as well as sleep-related problems.[31,32]

Data Analysis

Questionnaires

Sleep variables from the questionnaires were evaluated for bivariate intercorrelations. Further, based on a tercile division of self-reported sleep (*Sleep Last Night*), participants were initially divided into three groups of low (≤ 6.5 h, n = 22), moderate (6.6-7.9 h, n = 21), or high (≥ 8 h, n = 22) sleep per night. Initial analyses were conducted using one-way analysis of variance to determine whether groups differed significantly on the primary EI and psychopathology variables. Secondary analyses were then undertaken using Pearson correlations to more closely examine the association between the sleep questionnaire item, *Sleep Last Night*, and scores on the primary and subscale scores of the EQ-i, MSCEIT, and PAI. Significance was evaluated at $P < 0.05$. Due to the large number of correlations, the P values were adjusted using a Bonferroni correction for all nonhypothesized associations within each analysis set.

Resting State Connectivity

Within the CONN Toolbox, the mean BOLD time series from the resting state scan was calculated for all voxels within the two seed regions for each participant. As a measure of functional connectivity, the zero-lagged bivariate correlation was calculated between the mean time course of each vmPFC seed region and every other voxel in the brain on a participant-by-participant basis. To improve normality before entry into the second level random effects general linear model, all bivariate correlation maps were Fisher transformed (i.e., z-score transformed) via an inverse hyperbolic tangent function.[27] Each participant's self-reported sleep was then regressed against these individual beta maps to determine the correlation between functional connectivity and sleep. We corrected for all voxels in the bilateral amygdala ROI with a height threshold of $P < 0.05$ (family-wise error [FWE]-corrected), whereas spatial extent

Table 1—Means and intercorrelations among sleep variables

Variable	Mean (SD)	1	2	3	4	5	6	7	8
1. Age (y)	30.15 (8.01)	–	0.38*	-0.02	-0.27*	-0.36*	-0.37*	-0.63**	-0.02
2. MEQ	49.90 (10.27)		–	0.23	-0.34*	-0.38*	-0.50**	-0.62**	0.18
3. Sleep last night (h)	6.97 (1.07)			–	0.04	-0.03	0.00	-0.09	0.72**
4. Weeknight bedtime (h)	23:04 (03:13)				–	0.96**	0.74**	0.63**	-0.13
5. Weekend bedtime (h)	23:56 (03:23)					–	0.58**	0.79**	-0.15
6. Weekday wakeup (h)	07:23 (01:37)						–	0.72**	-0.16
7. Weekend wakeup (h)	08:38 (01:57)							–	-0.15
8. Sleep debt (h)	-00:26 (01:03)								–

MEQ, Morningness-Eveningness Questionnaire; SD, standard deviation; sleep debt = sleep last night – (((average weekday sleep × 5) + (average weekend sleep × 2))/7); *P < 0.05, **P < 0.001.

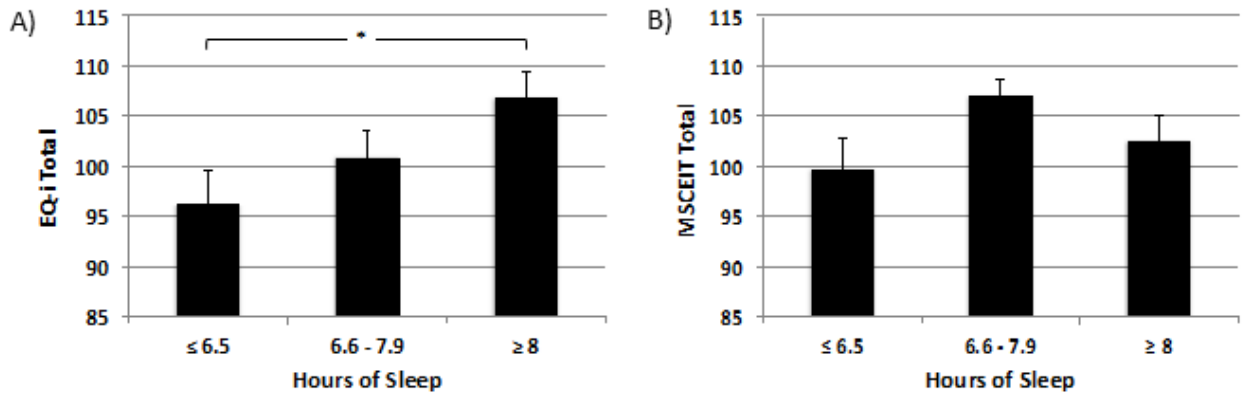

Figure 1—Mean emotional intelligence scores for the entire sample (n = 65) divided by terciles for *Sleep Last Night*, including *Low Sleep* (≤ 6.5 h, n = 22), *Moderate Sleep* (6.6-7.9 h, n = 21), and *High Sleep* (≥ 8 h, n = 22). **(A)** Analysis of variance indicated a significant main effect of sleep on scores on the Bar-On Emotional Intelligence Inventory (EQ-i) (P = 0.032), with a significant difference between the High and Low Sleep groups. **(B)** There was no main effect of sleep on the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT). *P < 0.05, corrected.

(i.e., minimum cluster size) was set at 10 voxels. The mean of the beta values from the resulting cluster was extracted for each individual and correlated with the indices of emotional intelligence and psychopathology.

RESULTS

Questionnaires

Sleep Last Night

As evident in Table 1, participants reported sleeping an average of 6.97 h (SD = 1.07) the night before the assessment session. Hours of reported sleep ranged from 4.0 to 9.0. From questions pertaining to estimated weekday and weekend sleep, participants reported generally sleeping 7.43 h (SD = 0.79) per night on average. These data were used to calculate an index of estimated *Sleep Debt*, which suggested that participants had reduced their sleep by approximately 26 min on the night preceding the scan compared with their typical sleep (Table 1). *Sleep Last Night* was not correlated with age, MEQ, or typical bed/wakeup times. As might be expected, however, MEQ was strongly correlated with typical bedtimes and wakeup times.

For preliminary analysis, *Sleep Last Night* was divided into three categories: low (≤ 6.5 h), moderate (6.6-7.9 h), or high (≥ 8 h). When divided in this manner, 22 participants were in the low sleep (M = 5.75 h, SD = 0.75), 21 were in the moderate sleep (M = 7.09 h, SD = 0.20), and 22 were in the high sleep (M = 8.08 h, SD = 0.23) groups. Because age is sometimes associated with sleep duration, this relationship was examined in the current dataset. However, age was not correlated with *Sleep Last Night* (r = -0.02, P = 0.88), so it was not included as a covariate in subsequent analyses. Finally, 43.8% of the sample (n = 28) reported that they occasionally had insomnia complaints (i.e., difficulty falling asleep or staying asleep). This variable was included as a nuisance covariate in subsequent partial correlation analyses involving emotional intelligence and psychopathology measures.

Emotional Intelligence

As evident in Figure 1A, there was a main effect of sleep category for *Total EQ-i* scores, $F(2,62) = 3.64$, P = 0.032, suggesting that a greater amount of sleep the preceding night was associated with higher *Total EQ*. Tukey *post hoc* tests showed that this effect was driven primarily by significantly

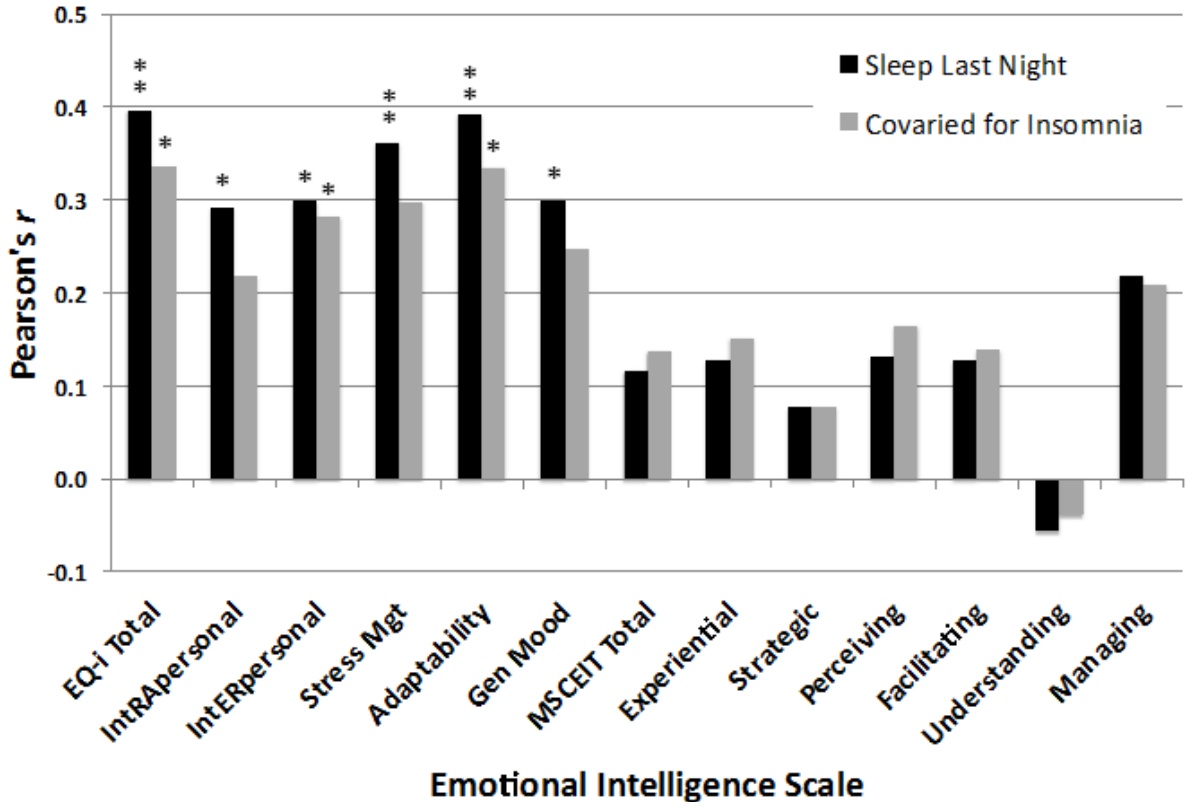

Figure 2—Effect sizes of the correlations between h of self-reported sleep obtained the preceding night and scores on the Bar-On Emotional Intelligence Inventory (EQ-i) and the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT). Black bars: All scales of the EQ-i showed significant Pearson correlations with *Sleep Last Night*, whereas none of the MSCEIT scales showed significant correlations. Gray bars: Similar trends were observed after statistically controlling for insomnia complaints, but only *Adaptability* remained significant. *P < 0.05, **P < 0.005.

higher EQ scores for the high versus low sleep group (P < 0.05). As shown in Figure 2, the number of h of *Sleep Last Night* was significantly correlated with higher scores for *Total EQ* (r = 0.396, P = 0.001), and all five composite EQ-i subscale scores, including *Intrapersonal* (r = 0.291, P = 0.019), *Interpersonal* (r = 0.299, P = 0.015), *Stress Management* (r = 0.362, P = 0.003), *Adaptability* (r = 0.392, P = 0.001; Bonferroni corrected P = 0.009), and *General Mood* (r = 0.300, P = 0.015; Bonferroni corrected P = 0.135), suggesting that more sleep was linearly associated with higher EQ-i scores. In contrast, MSCEIT scores did not differ significantly across sleep duration categories (Figure 1B), and none of the scales of the MSCEIT were significantly correlated with h of *Sleep Last Night* (Figure 2), (all r < 0.14, all P > 0.30). To address possible concern that this difference between the strength of correlations on the two scales might be accounted for by response biases affecting the self-report measures, the same analyses were conducted for the self-report scales using partial correlations to control for scores on the *Negative Impression Index* of the EQ-i (i.e., the tendency to "fake bad"). For the EQ-i, four of the six partial correlations remained significant after controlling for response bias, including *Total EQ* (r = 0.301, P = 0.015), *Intrapersonal* (r = 0.185, P = 0.142), *Interpersonal* (r = 0.272, P = 0.030), *Stress Management* (r = 0.306, P = 0.014), *Adaptability* (r = 0.297, P = 0.017; Bonferroni corrected P = 0.034), and *General Mood* (r = 0.239, P = 0.057; Bonferroni corrected P = 0.114). Finally, we explored the potential effect of insomnia complaints on these associations. Figure 2 shows that statistically controlling for insomnia complaints modestly reduced the strength of the correlations, although *Total EQ* and *Adaptability* remained significantly correlated with *Sleep Last Night* even after removing the influence of insomnia problems.

Psychopathology

Linear associations were also obtained among several variables of the PAI with *Sleep Last Night* (Figure 3). Obtaining more h of *Sleep Last Night* correlated with significantly lower scores for *ANX* (r = -0.344, P = 0.007), *ARD* (r = -0.302, P = 0.018; Bonferroni corrected P = 0.252), *DEP* (r = -0.378, P = 0.003), *PAR* (r = -0.385, P = 0.002), *SCZ* (r = -0.385, P = 0.002; Bonferroni corrected P = 0.028), *ANT* (r = -0.284, P = 0.027; Bonferroni corrected P = 0.378), *ALC* (r = -0.263, P = 0.040; Bonferroni corrected P = 0.56), *STR* (r = -0.355, P = 0.005; Bonferroni corrected P = 0.07), and higher scores for *RXR* (r = 0.268, P = 0.037; Bonferroni corrected P = 0.518). In contrast, *Sleep Last Night* was not significantly correlated with scores for *SOM*, *MAN*, *BOR*, *DRG*, *AGG*, *SUI*, *NON*, *DOM*, and *WRM* (all r < |0.22|, all P > 0.09). Figure 3 also shows that statistically controlling for insomnia complaints had only minimal effects on the strength of most correlations between *Sleep Last Night* and PAI variables.

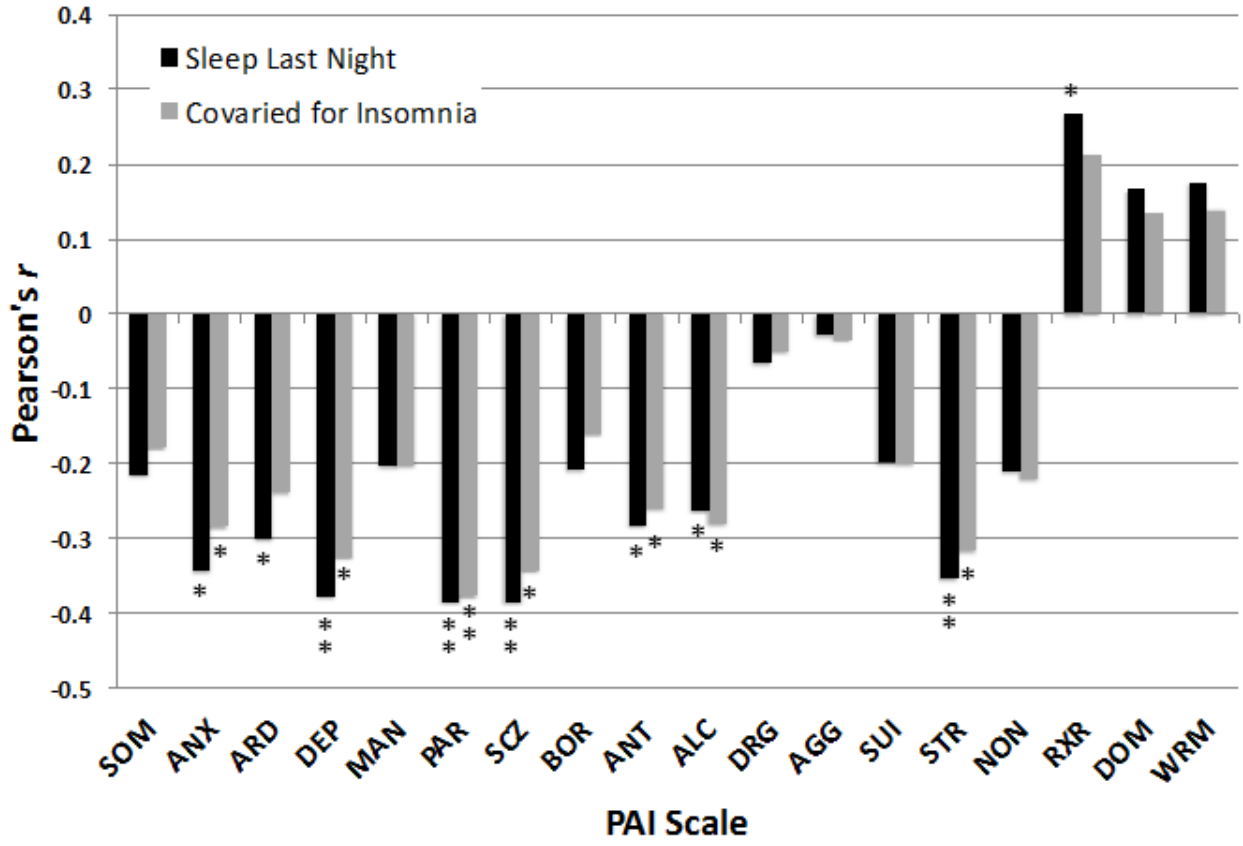

Figure 3—Effect sizes of the correlations between h of self-reported sleep obtained the preceding night and scores on the Personality Assessment Inventory (PAI). Black bars: Greater sleep the preceding night was associated with lower scores on several indices of psychopathology based on bivariate correlations. Gray bars: Most of the correlations between *Sleep Last Night* and psychopathology remained significant after statistically controlling for insomnia complaints. See text for expansion of abbreviations. *P < 0.05, **P < 0.005.

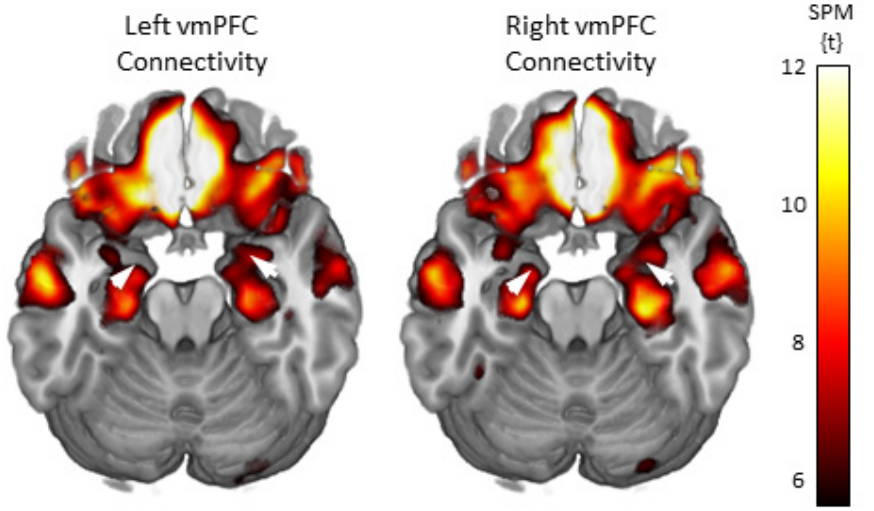

Figure 4—Functional connectivity maps for the left and right ventromedial prefrontal cortex (vmPFC) seed regions of interest (ROIs). The brain images show axial slices that include both the vmPFC and amygdala regions. The white arrows show the location of the amygdala target ROIs. The maps were set to a whole brain threshold of P < 0.05, family-wise error (FWE) corrected for multiple comparisons. SPM = statistical parametric mapping.

Neuroimaging

VMPFC-Amygdala Functional Connectivity

Whole brain connectivity maps through an axial slice showing the vmPFC and amygdala are displayed in Figure 4. These maps are corrected for multiple comparisons using FWE at P < 0.05 for the whole brain. From these maps, initial connectivity values (Fisher transformed correlation coefficients) were extracted for the left and right amygdala ROIs for all voxels exceeding the corrected threshold and are presented in Table 2.

Functional connectivity between the left vmPFC seed region and voxels within either the right or left amygdala was not significantly correlated with *Sleep Last Night*. In contrast, greater *Sleep Last Night* was associated with greater negative functional connectivity between the right vmPFC seed region and voxels within the right amygdala

(Figure 5). This analysis yielded a cluster of 10 voxels in the right amygdala ROI [Montreal Neurologic Institute (MNI) coordinates: x = 24, y = 2, z = -22]. Connectivity was negatively correlated with greater self-reported sleep time (T = 4.20, r = -0.49). Age was not associated with connectivity strength (r = -0.009, P = 0.94), so it was not included as a covariate in the analysis.

Neuroimaging and Questionnaire Correlates

Connectivity Strength and EI

The beta values from the right amygdala cluster representing the strength of connectivity with the vmPFC that covaried with *Sleep Last Night* were extracted and entered into a correlation analysis with the various EI indices. As shown in Figure 6, the strength of the negative functional connectivity between the right vmPFC and right amygdala was linearly correlated with three EI measures, including *Total EQ* (r = -0.287, P = 0.029), *Stress Management* (r = -0.276, P = 0.036), and *Adaptability* (r = -0.305, P = 0.020; Bonferroni corrected P = 0.18). These findings suggest that greater negative connectivity between these regions was associated with higher EI scores on these scales, but not to the *Intrapersonal*, *Interpersonal*, or *General Mood* scales (all r < |0.34|, all P > 0.07). In contrast, the strength of connectivity between the right vmPFC and amygdala was unrelated to any of the scores on the MSCEIT, including *Total EI*, *Experiential EI*, *Strategic EI*, *Perceiving EI*, *Facilitating EI*, *Understanding EI*, *Managing EI* (all r < |0.16|, all P > 0.25). Figure 6 also shows that once insomnia complaints were statistically controlled, the strength of the correlation between EI scales and connectivity was reduced and none of the findings reached significance.

Table 2—Connectivity values between the vmPFC and amygdala

	Left Amygdala		Right Amygdala	
Seed region	**Mean (SD)**	**Range**	**Mean (SD)**	**Range**
Left vmPFC	0.13 (0.14)	-0.13 to 0.51	0.13 (0.13)	-0.19 to 0.42
Right vmPFC	0.13 (0.14)	-0.21 to 0.57	0.14 (0.13)	-0.13 to 0.47

Values reflect Fisher transformed correlation coefficients (i.e., z-scores). vmPFC, ventromedial prefrontal cortex (i.e., gyrus rectus).

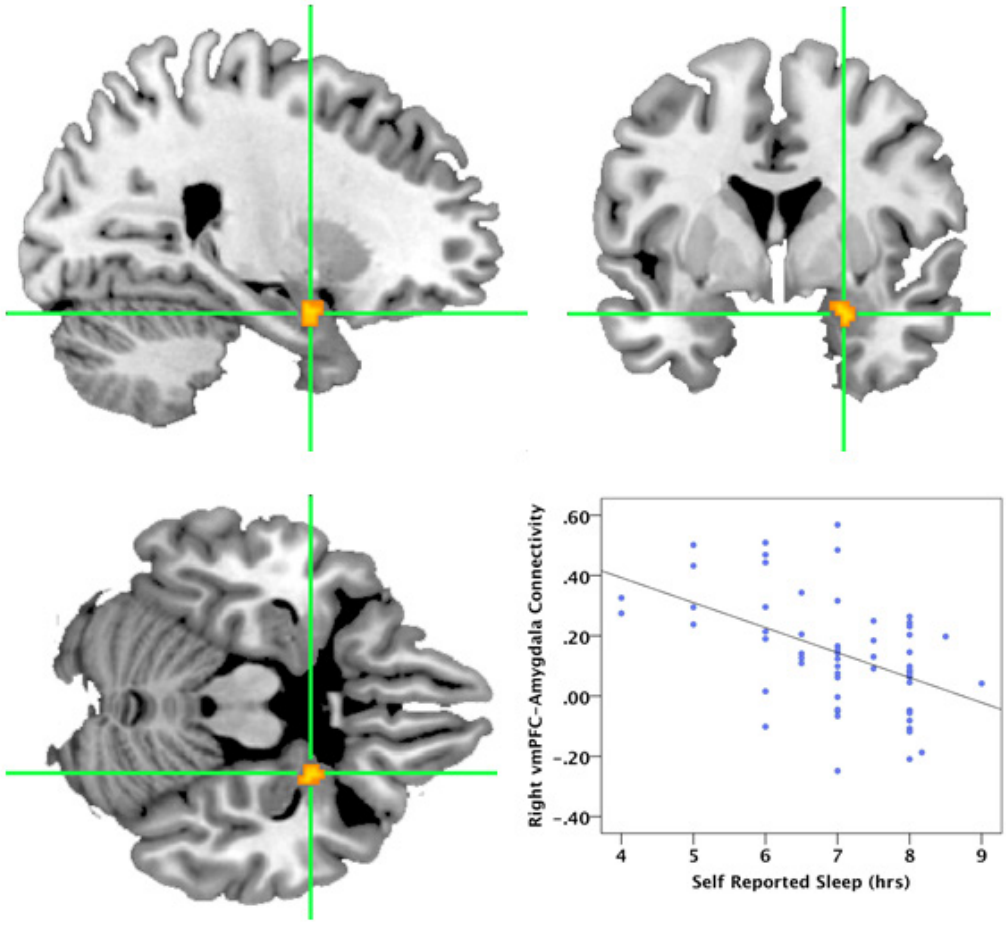

Figure 5—Self-reported *Sleep Last Night* was significantly correlated with negative functional connectivity between the right ventromedial prefrontal cortex (vmPFC) and the right amygdala. The figure shows the cluster in the right amygdala [MNI coordinates: x = 24, y = 2, z = -22] that showed negative functional connectivity with the right vmPFC seed region as a function of greater reported sleep time. For visualization, the cluster is height thresholded at (P < 0.001, uncorrected, spatial extent P < 0.05 family-wise error [FWE] corrected). Figures are displayed in sagittal (top left), axial (bottom left), and coronal (top right) views. The scatterplot (bottom right) shows the linear relationship between hours of sleep and the connectivity values extracted from the displayed cluster.

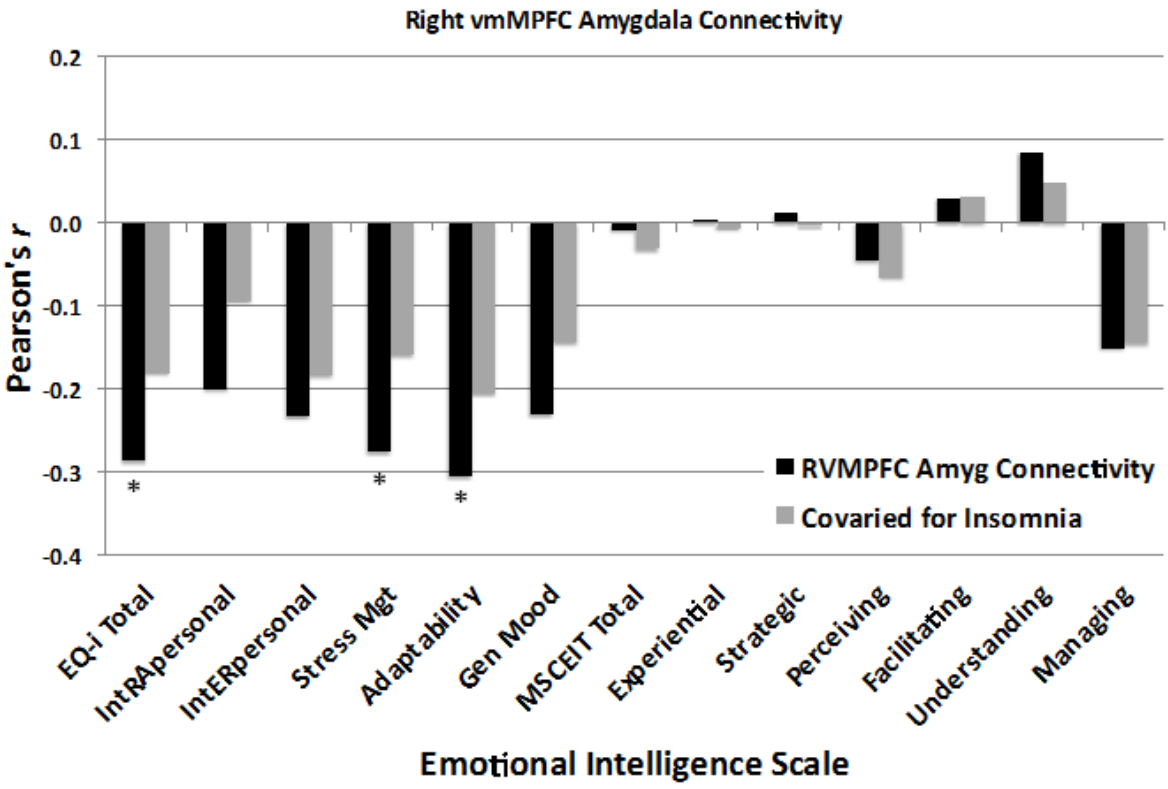

Figure 6—Effect sizes show the correlations between the magnitude of ventromedial prefrontal cortex (vmPFC) – amygdala functional connectivity and scores on the Bar-On Emotional Intelligence Inventory (EQ-i) and the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT). Black bars: Total EQ-i, as well as composite scale scores for *Stress Management* and *Adaptability* showed significant negative bivariate correlations with functional connectivity, indicating that higher emotional intelligence on the EQ-i was associated with greater negative connectivity between these two regions. In contrast, MSCEIT scales were not significantly correlated with functional connectivity between these two regions. Gray bars: After controlling for insomnia complaints, the observed correlations with emotional intelligence were no longer significant. *P < 0.05.

Connectivity Strength and Psychopathology

The extracted vmPFC-amygdala connectivity data were also correlated with the indices of psychopathology from the PAI. As evident in Figure 7, greater positive connectivity was associated with increased scores for *ANX* (r = 0.325, P = 0.013), *ARD* (r = 0.306, P = 0.021; Bonferroni corrected P = 0.294), *DEP* (r = 0.388, P = 0.003), *PAR* (r = 0.286, P = 0.031), *SCZ* (r = 0.305, P = 0.021; Bonferroni corrected P = 0.294), *BOR* (r = 0.335, P = 0.011; Bonferroni corrected P = 0.154), *SUI* (r = 0.368, P = 0.005; Bonferroni corrected P = 0.070), STR (r = 0.339, P = 0.010; Bonferroni corrected P = 0.140). In contrast, greater negative connectivity between vmPFC and amygdala was associated with healthier scores on RXR (r = -0.440, P = 0.001; Bonferroni corrected P = 0.014). Other scales were not significantly related to functional connectivity, including *SOM*, *MAN*, *ANT*, *ALC*, *DRG*, *AGG*, *NON*, *DOM*, and *WRM* (all $r \leq 0.24$, all P > 0.07). Together, these findings suggest that when the vmPFC and amygdala covaried together positively, participants had higher psychopathology scores on several hypothesized scales, but as these regions showed greater negative connectivity, the severity of psychopathology scores was lower. Figure 7 shows that removing the effects of insomnia complaints had only minimal effects on the correlations between vmPFC-amygdala connectivity and PAI scores.

DISCUSSION

Consistent with predictions from prior work,[14,15] greater self-reported sleep the night before the assessment was significantly related to higher *Trait* EI scores and lower scores on several indices of psychopathology. Sleep duration the night before the scan was also negatively correlated with functional connectivity between the right vmPFC and amygdala. Moreover, the strength of this negative prefrontal-amygdala connectivity pattern was directly related to scores on *Trait* EI and psychopathology scales. Generally, the more strongly negative the functional connectivity, the higher the EI scores and less severe the psychopathology scores.

The current finding that greater self-reported nocturnal sleep duration was associated with higher *Trait* EI scores is in line with our previous work showing that total sleep deprivation was correlated with a decline on the same indices[14] and other evidence suggesting a link between fatigue and lower EI.[33] In our prior study, sleep deprivation led to degradation of several aspects of EI, including emotional self-awareness, perceived effectiveness in dealing with interpersonal relationship issues, and the ability to cope with stress. We currently show that EI is associated with the duration of sleep during a single night, as those who obtained fewer h of sleep the night before the assessment achieved lower *Total EQ-i* scores than those who obtained the most sleep. In fact, those participants obtaining

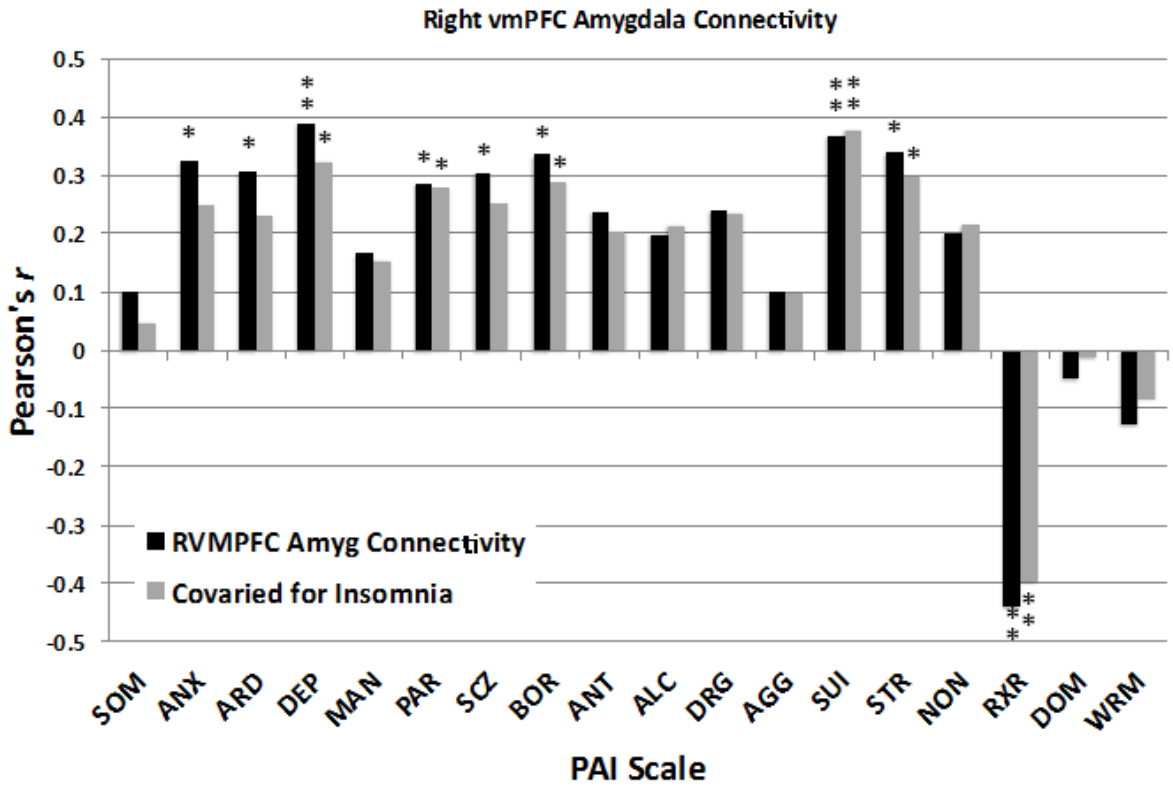

Figure 7—Effect sizes show the correlations between the magnitude of ventromedial prefrontal cortex (vmPFC) – amygdala functional connectivity and scores on the Personality Assessment Inventory (PAI). Black bars: Scores on several PAI scales showed positive bivariate correlations with functional connectivity, indicating that symptoms of psychopathology tended to be higher as these two regions covaried positively together, whereas psychopathology was reduced as these two regions covaried negatively with one another. Gray bars: Partial correlations controlling for insomnia complaints remained significant for most PAI scales. See text for expansion of abbreviations. *P < 0.05, **P < 0.005.

8 or more h of sleep typically had *Total EQ-i* scores about 10 points (i.e., about two thirds of an SD) higher than those obtaining 6.5 h of sleep or less the preceding night. Moreover, all five EQ-i composite subscales also showed significant positive correlations with sleep duration from the previous night. In contrast, we found no association between sleep and *Ability* EI, as assessed by the MSCEIT.

The current finding that *Trait*, but not *Ability*, EI is related to sleep duration is enlightening, as these two models conceptualize the EI construct in very different ways.[34] Whereas *Trait* EI taps subjective emotional experience, global mood and optimism, self-confidence, self-awareness, and self-perceived interpersonal sensitivity,[35] *Ability* EI involves the accuracy of emotional perception, the ability to use emotional information to facilitate performance, the quality of reasoning about emotional information, and the capacity to regulate or manage emotions.[25] Our findings suggest that, within the range of sleep duration obtained by most people on a typical night, getting more sleep appears to be reliably associated with better self-perceived emotional functioning, interpersonal attunement, coping capacity, self-confidence, and self-awareness, but is not reliably related to performance based capacities, such as reasoning about emotional information and solving emotional problems. To the extent that these varied aspects of EI involve distinct neuroanatomical regions,[30] it makes sense that sleep deprivation may have differential effects on such traits and capacities.

Greater sleep duration the night before the assessment was also associated with lower scores on several indices of psychopathology from the PAI. Specifically, increased sleep time was most strongly associated with reduced severity of complaints associated with anxiety, depression, paranoia, and schizophrenia. These findings are congruent with previous findings showing that total sleep deprivation was associated with increased symptoms of psychopathology.[15] Together, these studies suggest that lack of sleep, or even modest curtailment of sleep, may be associated with a subtle nonclinical elevation of a number of emotional distress complaints, even among healthy normal individuals.

Although reduced sleep has long been perceived as a consequence or symptom of psychopathology, emerging evidence suggests that sleep disruption may also play a contributory role in the etiology of some psychiatric conditions. A recent large-scale study showed that behaviorally induced insufficient sleep among adolescents was associated with a significantly elevated risk of suicidal ideation.[36] Currently, the precise neurobiological basis for the link between sleep loss and psychopathology remains uncertain, but some evidence suggests that lack of sleep may lead to altered neurochemistry within the prefrontal cortex,[37] alterations in neurotransmitter receptor sensitivity,[38-40] and increases cortical excitability, particularly in prefrontal regions.[41] Considerable evidence points to dysfunction of the vmPFC during a number of

psychopathological conditions including depression,[42-45] anxiety disorders,[46-49] and psychopathy.[50] Metabolic activity in the vmPFC also appears to be particularly affected by sleep deprivation,[16] and a number of studies have shown that tasks sensitive to vmPFC functioning are particularly impaired by lack of sleep.[10,51-53] Notably, functional connectivity between the emotion regulating regions of the medial prefrontal cortex and the emotionally responsive regions of the limbic system, such as the amygdala and other cortical regions, appears to be altered by sleep deprivation.[19,54]

In the current study, the correlation between self-reported sleep duration and functional connectivity between the prefrontal cortex and amygdala was also investigated. With greater sleep duration the night before the scan, there was stronger negative functional connectivity between the vmPFC and amygdala in the right hemisphere. Emerging evidence suggests that the medial prefrontal regions are critical to normal top-down regulation of the amygdala and other limbic emotional engagement systems.[55] Interestingly, this corticolimbic regulatory capacity can be depleted by overuse or fatigue.[18] One interpretation of our findings, therefore, is that greater nocturnal sleep may facilitate the daily replenishment of this top-down regulatory capacity, leading to more effective modulation of affective responses by the prefrontal cortex. Of course, the correlational nature of the findings precludes the ability to draw directional conclusions. The current findings are consistent with those of Yoo and colleagues,[19] who found that 35 h of sleep deprivation was associated with increased amygdala responsiveness to negative emotional stimuli and reduced functional connectivity between the medial prefrontal cortex and amygdala, but further extend these findings to more common levels of occasional sleep curtailment or the short sleep periods experienced periodically by most people. The right-lateralized nature of the finding was not hypothesized, but is interesting in light of other findings suggesting that sleep deprivation may have a greater impairing effect on cognitive processes mediated by the right compared with the left hemisphere.[56-58]

The final question we addressed was whether the sleep-related strength of functional connectivity between the vmPFC and amygdala might correlate directly with EI and psychopathology scores. The functional connectivity values between these two regions were extracted for each participant and used to predict scores on measures of EI and psychopathology. Higher *Trait* EI, *Stress Management*, and *Adaptability* scores were associated with a pattern of greater negative prefrontal-amygdala functional connectivity. In contrast, no association was observed for *Ability* EI, suggesting that this aspect of the vmPFC-amygdala emotion regulation system appears to be more related to affective response traits rather than behaviorally measured emotional problem solving abilities. Similarly, we found that the strength of vmPFC-amygdala functional connectivity was modestly but significantly positively correlated with half of the psychopathology scales on the PAI, most notably depression, but also anxiety, paranoia, treatment rejection, and marginally to suicidal ideation. Although correlational in nature, these findings are consistent with a number of studies that have shown that affective regulation is associated with a negative relationship between the medial prefrontal cortex and the amygdala[55,59] and that some forms of affective psychopathology may involve alteration or disruption of this neurocircuitry.[46,60,61]

Some limitations should be borne in mind when interpreting these findings. First, we used a self-report index of sleep, which is likely to suffer from some loss in precision and reliability, particularly when compared with objective methods such as ambulatory electroencephalographic or actigraphic monitoring. Future studies would benefit from the use of objective measurements of sleep. Second, although the current sample is relatively large for a neuroimaging study, our participants were all thoroughly screened to exclude clinical levels of psychopathology. Consequently, the current findings cannot be validly generalized to more severe forms of psychopathology. Third, because the findings from this study are correlational, it is not possible to determine the causal direction of the observed relationships or whether the findings may be due to an unmeasured third variable. Although insomnia complaints, response biases, and age did not seem to account for most of the findings, it is possible that other unexplored variables might have contributed. Furthermore, although we collected data regarding the amount of sleep obtained the preceding night and attempted to tightly control the time of scan administration, we did not specifically control for wakeup time on the day of the scan. Thus, it is possible that this could have added some uncontrolled variance to the data, potentially obscuring some important relationships. This is particularly important in light of the fact that we had no objective control for level of vigilance within the scanner, such as simultaneous electroencephalography. Thus, it is not possible to conclusively determine whether the observed differences in functional connectivity might have been due to fluctuations in vigilance occurring during the scan. Future studies would benefit from the use of simultaneous electroencephalography in this regard. Finally, we only examined functional connectivity between two regions, the vmPFC and amygdala. Emotional experience and regulation are extraordinarily complex processes and undoubtedly encompass a much larger neurocircuitry than the limited set of regions examined here. Future work will need to expand upon this neurocircuitry to include other candidate nodes such as the insular cortex, striatum, brainstem nuclei, and other higher-order associative regions.

Nonetheless, with appropriate consideration to the aforementioned limitations, we believe the current study advances our understanding of the association between recent sleep, prefrontal-amygdala connectivity, and emotional functioning. These data suggest that even small variations in sleep of only 1 or 2 h may be significantly associated with differences in some aspects of perceived emotional intelligence and the severity of psychological distress. Conversely, getting a full night of sleep appears to be connected with bolstered emotional strength and mental health.

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DISCLOSURE STATEMENT

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Physical exercise and brain responses to images of high-calorie food

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Physical exercise has many health benefits, including improved cardiovascular fitness, lean muscle development, increased metabolism, and weight loss, as well as positive effects on brain functioning and cognition. Recent evidence suggests that regular physical exercise may also affect the responsiveness of reward regions of the brain to food stimuli. We examined whether the total number of minutes of self-reported weekly physical exercise was related to the responsiveness of appetite and food reward-related brain regions to visual presentations of high-calorie and low-calorie food images during functional MRI. Second, we examined whether such responses would correlate with self-reported food preferences. While undergoing scanning, 37 healthy adults (22 men) viewed images of high-calorie and low-calorie foods and provided desirability ratings for each food image. The correlation between exercise minutes per week and brain responses to the primary condition contrast (high-calorie>low-calorie) was evaluated within the amygdala, insula, and medial orbitofrontal cortex, brain regions previously implicated in responses to food images. Higher levels of exercise were significantly correlated with lower responsiveness within the medial orbitofrontal cortex and left insula to high-calorie foods. Furthermore, activation of these regions was positively correlated with preference ratings for high-calorie foods, particularly those with a savory flavor. These findings suggest that physical exercise may be associated with reduced activation in food-responsive reward regions, which are in turn associated with reduced preferences for unhealthy high-calorie foods. Physical exercise may confer secondary health benefits beyond its primary effects on cardiovascular fitness and energy expenditure. *NeuroReport* **24:962–967** © **2013 Wolters Kluwer Health | Lippincott Williams & Wilkins.**

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Introduction

Physical exercise has numerous beneficial effects on health, including improved cardiovascular fitness, lean muscle development, increased metabolism, and weight loss, among others [1]. Recent evidence suggests that regular physical activity may also affect food consumption, which could further contribute to the health benefits of exercise. The effects of physical exercise on appetite and food intake are complex and not fully understood, but empirical evidence suggests that while greater physical activity may increase fasting hunger sensations, it may also lead to improvements in the satiety response and control over aspects of appetite regulation [2]. One potential mechanism for greater appetite control may be alterations in the subjective reward value of food. Recent functional neuroimaging findings suggest that chronic regular exercise may be associated with reduced responsiveness of brain reward regions, such as the insula and cingulate gyrus, to visual food cues in overweight individuals [3,4]. While some reward regions seem to show reduced responsiveness to food images after a chronic exercise program, it is not clear whether these brain activation changes directly relate to reduced desire to consume the food represented in the images.

Using functional MRI, we examined the association between self-reported weekly physical exercise levels and brain responses to images of food differing in calorie density. We surveyed several key regions associated with appetite regulation and the assessment of reward value for visually presented food stimuli, specifically the amygdala, insula, and medial orbitofrontal cortex (mOFC) [5–7]. We hypothesized that individuals with higher levels of physical exercise per week would show reduced responsiveness of these key regions when viewing images of high-calorie versus low-calorie foods, and that the activation of these regions would correlate positively with self-rated desirability of high-calorie food items.

Methods

Participants

Thirty-seven healthy adults (15 women; 22 men) between 18–45 years (mean = 29.7; SD = 8.4) of age completed questionnaires about exercise habits and

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underwent functional neuroimaging. Exclusionary criteria included any significant history of medical, neurological, or psychiatric problems, illicit substance use, alcohol treatment, recent use of psychoactive medications, and abnormal visual acuity not correctable with contact lenses. Although the present findings are novel and have never been published previously, other data from a subset of this same sample have been reported elsewhere [8,9]. Participants ranged from normal weight to moderately obese, with an average BMI of 24.5 (SD = 3.7; range 19.8–34.8). Written informed consent was provided before enrollment and all participants were compensated for their time. This research protocol was reviewed and approved by the Institutional Review Board of McLean Hospital.

Materials and procedure

Exercise questionnaire

Following informed consent, each participant completed several self-report questionnaires about food intake on the day of the scan, as well as typical dietary, sleep, and exercise habits. In particular, participants answered questions about their typical physical exercise routines, including the number of exercise sessions completed during an average week and the typical duration of their workouts. The product of these two values was calculated to derive each individual's typical exercise minutes per week. Participants consumed an average of 323.5 calories (SD = 245.5; range 0–929.5) throughout the day before undergoing the scan, but had no food intake for an hour before entering the scanner.

Neuroimaging

While undergoing functional MRI, participants viewed a series of food and nonfood images presented in 30-s blocks that alternated between pictures of foods differing in calorie density, including high-calorie (H) foods (e.g. cheeseburgers, ice cream, cake, French fries, candy), low-calorie (L) foods (e.g. fresh salads, vegetables, fruits, fresh fish, whole grain bread), or control (C) images (i.e. nonedible rocks, flowers, shrubs). The paradigm is similar to that reported in our previous publications [5,10,11]. Briefly, food and control stimuli were presented in seven alternating stimulus blocks of 10 images each (3 s/image), bounded at the beginning and end by a 15-s fixation cross (+) presented in the following order (+ , C, L, H, C, H, L, C, +). The entire scan lasted 240 s. Following the scan, the participants completed an offline rating for each of the food images, indicating on a seven-point scale how much they desired to eat the depicted food item at that moment. For analysis, the food images were further subdivided into those with flavors that were either savory (e.g. cheeseburgers, green salads; H = 9; L = 12) or sweet (e.g., ice cream, fruit; H = 11; L = 8).

Magnetic resonance imaging parameters

Scans were collected using a 3.0-T Siemens Tim Trio scanner (Siemens, Erlangen, Germany) and a 12-channel head coil. First, structural T1-weighted 3D MPRAGE images were collected for anatomical coregistration (TR/TE/flip angle = 2.1 s/2.25 ms/12°) over 128 sagittal slices (256 × 256 matrix) providing a slice thickness of 1.33 mm (voxel size = 1 × 1 × 1.33 mm). For functional scanning, we collected a T2*-weighted echo planar imaging sequence (TR/TE/flip angle = 3.0 s/30 ms/90°) with 80 images/slice over 43 transverse interleaved planes (3.5 mm thickness, no skip; 22.4 cm field of view; 64 × 64 acquisition matrix), yielding a voxel size of 3.5 × 3.5 × 3.5 mm. To ensure steady-state equilibrium, the first three functional scans were discarded before data collection.

Image processing

Data were preprocessed and analyzed in SPM8 (Wellcome Department of Cognitive Neurology, London, UK), using standard realignment and motion correction parameters. The echo planar imaging images were coregistered, spatially normalized, and smoothed using an isotropic Gaussian kernel (full width at half maximum = 6 mm), and resliced to 2 × 2 × 2 mm. Time series data were convolved with the canonical hemodynamic response function and the effects of serial autocorrelation were removed with a first-level autoregressive model. The default 128-s high-pass filter was used to remove low-frequency drift in the signal.

Statistical analysis

Within SPM8, a series of general linear models were created for the H, L, and C conditions against an implicit baseline, followed by construction of a direct contrast between the high-calorie versus low-calorie conditions. In a second-level random effects regression model, these contrast images were correlated with the previously calculated variable of interest, exercise minutes per week. Because previous work has suggested that men and women process images of food stimuli differently [11], participant sex was entered as a nuisance covariate. According to our *a priori* hypotheses, the primary analyses were restricted to six search territories (i.e. bilateral insula, amygdala, and mOFC) as defined by the Automated Anatomical Labeling Atlas [12], implemented within the Wake Forest University SPM8 Toolbox PickAtlas Utility (*http://www.fmri.wfubmc.edu/downloads/WFU_PickAtlas_User_Manual.pdf*) [13]. Statistical thresholds were selected with consideration to the guidelines for principled correction for false positives [14]. Accordingly, activation maps for the regression analyses were initially thresholded at P less than 0.001 uncorrected, k (extent) at least 10 contiguous voxels (based on the expected number of voxels per cluster as reported in the SPM output, which was $k = 11.1$; this was rounded down to $k \geq 10$ for consistency with other publications), and then subjected to small volume correction for multiple comparisons within each search territory at P less than 0.05, corrected for false discovery rate. Significant clusters within the search territories were extracted and

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correlated in SPSS 20 (IBM Corporation, Armonk, New York, USA) with postscan food desirability ratings made by the participants for each of the food images.

Results

Physical exercise

Twenty-five participants (67.6%) reported engaging in regular physical exercise (i.e. at least once a week or more), whereas 12 (32.4%) indicated that they did not exercise with any regularity, or at all. The number of reported workout days per week for the entire sample ranged from 0 to 7 (mean = 2.8, SD = 2.3). The average duration of workouts ranged from 0 to 120 min (mean = 38.4, SD = 36.1). For each individual, the product of these variables was calculated as the average number of exercise minutes per week, which ranged from 0 to 540 min (mean = 151.1, SD = 159.9).

Neuroimaging

Voxel-wise correlation analysis was used to examine the association between exercise minutes per week and responses within each of the search territories for the H > L contrast. As shown in Fig. 1a, exercise minutes per week was significantly negatively correlated with a cluster of 10 voxels within the mOFC (MNI coordinates: $x = 0$, $y = 48$, $z = -10$; $T[34] = 3.58$; $R^2 = 0.28$). Similarly, Fig. 1b shows that exercise minutes per week was negatively correlated with a cluster of 22 voxels in the left anterior insula (MNI coordinates: $x = -30$, $y = 128$, $z = 160$; $T[34] = 4.32$; $R^2 = 0.35$). In contrast, there were no clusters in the right or left amygdala where exercise minutes per week was significantly correlated with responses to the H > L contrast. Furthermore, there were no clusters in any regions showing positive correlations with exercise minutes per week.

Correlations with food ratings

The first eigenvariate was extracted from significant clusters in the preceding analysis and correlated with each individual's exercise minutes per week and food ratings. As shown in Table 1, exercise minutes per week was negatively correlated with preference ratings for savory high-calorie foods. In addition, extracted data from the mOFC and left insula (in response to the H > L contrast) were positively correlated with preference ratings for savory high-calorie foods, but not for sweet high-calorie foods. None of these variables were related to ratings for low-calorie foods.

Discussion

We examined the association between self-reported physical exercise minutes and brain responses to visually presented images of high-calorie versus low-calorie-dense foods. Greater physical exercise was significantly correlated with reduced activation within the mOFC and left anterior insula in response to foods with higher calorie density. The insula plays a major role in visceral interoceptive sensations and has been associated with craving for drugs and food [15]. The mOFC also appears to be a key region in processing the reward value of stimuli, particularly pleasure responses to preferred foods [16]. Furthermore, we also found that the magnitude of these brain responses was positively associated with self-rated desire to consume the specific high-calorie foods presented, particularly those foods with a savory flavor (e.g. cheeseburgers, French fries, hot dogs), whereas those with a sweet flavor (e.g. ice cream, cake, chocolate candy) did not show a significant association. In short, regular physical exercise was associated with reduced responsiveness of appetite-related brain regions, and this lower responsiveness was associated with reduced preference and desire for high-calorie foods.

These findings build upon an emerging literature that suggests that physical exercise may contribute to weight loss in ways that extend beyond simple energy expenditure and altered metabolism. Research evidence indicates that exercise may also alter brain responses to food stimuli, particularly within reward processing and appetite-related regions such as the orbitofrontal cortex and insula. For instance, in a recent study, researchers found that a 60-min bout of acute exercise led to an immediate postworkout reduction in the responsiveness of the orbitofrontal cortex, insula, and other reward-related brain regions during presentation of high-calorie foods [17]. Findings from that study are consistent with evidence suggesting that acute exercise not only burns calories, but also leads to reduced food intake. This likely occurs due to alterations in brain systems involved in pleasure responses and salience processing, leading to reduced incentive motivation toward food [3,18]. Moreover, the effects on brain functioning do not appear to be limited to acute postexercise changes, as insular responses to high-calorie food images were attenuated following a 6-month exercise program [3,4] and were highly correlated with actual weight loss and body fat reduction [3]. Such findings suggest that the effects of exercise on brain functioning are sustained and not simply a transient effect of acute physical activity. Our data are congruent with these previous findings, suggesting that even self-reported habitual exercise levels are reliably associated with reduced insula and mOFC responses to images of high-calorie foods. Moreover, our findings extend previous work by showing that reduced activation within these brain regions was also associated with reduced desire to consume high-calorie foods, particularly those with a savory flavor. This could be particularly important for weight reduction, as preference for savory foods has been posited as a risk factor for overweight and obesity [19].

The exact mechanisms by which exercise may exert the observed effects on brain functioning remain to be determined, but recent studies suggest that exercise may lead to an enhancement of leptin sensitivity in animals [20]

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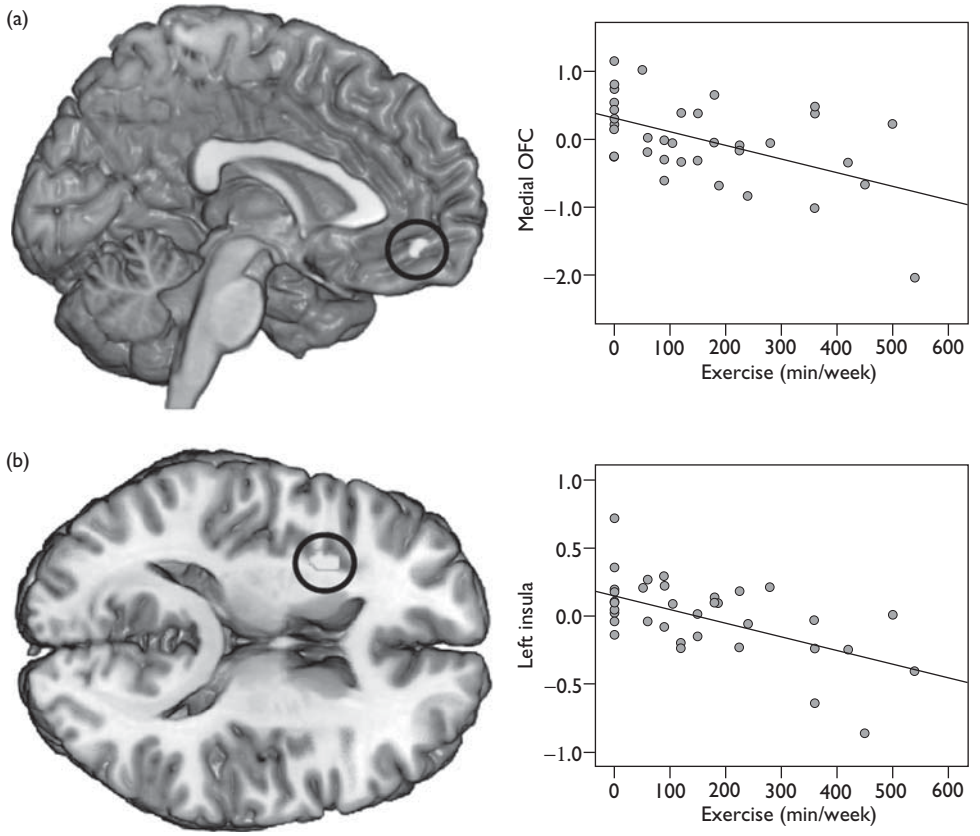
Fig. 1



Self-reported physical exercise (minutes per week) was negatively correlated with responses within the (a) medial orbitofrontal cortex (OFC) (MNI: $x=0$, $y=48$, $z=-10$) and (b) left insula (MNI: $x=-30$, $y=128$, $z=160$) superimposed on the standard T1 template from SPM8. Scatterplots are displayed for descriptive purposes and show the pattern of association between exercise and the first extracted cluster eigenvariate. MNI, Montreal Neurological Institute.

Table 1 Mean food ratings and Pearson correlations with exercise minutes per week and extracted brain activation clusters

Food rating	Mean (SD)	Exercise (min/week)	mOFC (0, 48, −10)	L insula (−30, 12, 16)
High calorie	4.3 (1.2)	−0.14	0.32†	0.36*
Savory	4.3 (1.5)	−0.33*	0.33*	0.33*
Sweet	4.3 (1.5)	0.06	0.20	0.25
Low calorie	3.7 (1.4)	0.06	0.00	0.10
Savory	3.5 (1.4)	0.06	−0.02	0.14
Sweet	4.0 (1.5)	0.05	0.04	0.03

Coordinates are within stereotaxic space of the Montreal Neurological Institute.
L, left; mOFC, medial orbitofrontal cortex.
*$P<0.05$.
†$P<0.10$.

and humans [21]. As leptin can affect insula and prefrontal cortex responses to food stimuli [22], it is possible that the effects of exercise on leptin sensitivity may be one avenue for this effect. It is also conceivable that cardiovascular fitness alters basic brain physiology and information processing, leading to greater neural efficiency. Exercise is associated with many beneficial effects on brain structure and function, including neuroplastic changes [23], increased blood flow [24] and neurogenesis within the hippocampus [25], as well as the proliferation of new blood vessels within the brain [26]. However, the specificity of the effects we observed to high-calorie versus low-calorie foods argues against a simple improvement in global brain functioning, suggesting instead that greater physical activity was associated with specific and circumscribed responses of appetite and food relevant regions such as the insula and orbitofrontal cortex. The data are, of course, correlational

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and could be affected by other factors. One alternate explanation would be that the observed correlations are due to the influence of an unmeasured third variable, such as heightened health awareness or fitness consciousness, which may drive the frequency of exercise as well as the decreased preference for calorie-dense foods. Further research into the potential causes of these specific changes will be an important step toward understanding the association between physical activity and brain function.

In contrast to the findings for the mOFC and insula, we did not find any significant correlation between physical exercise and amygdala responses. Our previous work has shown that the amygdala is responsive to both high-calorie and low-calorie food images [5] and that this pattern of activation appears to be developmentally invariant between childhood and early adulthood [10]. This invariance of activation suggests that the amygdala response to food is established early in development and may reflect a broad and generic salience response to the presence of food irrespective of calorie density or hedonic value. The present findings further suggest that the responsiveness of the amygdala to food images remains relatively stable irrespective of chronic levels of physical exercise.

While the present results suggest that there is a significant association between regular physical exercise and reduced responses to high-calorie food stimuli within specific appetite and reward processing regions of the brain, the findings should be interpreted in light of some limitations. First, our findings are based on subjective estimates of exercise frequency and duration per week, which may be less reliable than objective measures such as wrist actigraphy or heart rate monitoring. Second, we did not specify the type of exercise in which participants engaged (e.g. cardiovascular training, strength training), which could potentially have differential effects on brain functioning or appetite. This would be an important area for further study. Third, to permit some variability in brain responses to food, we did not specifically restrict dietary intake during the day of the scan, although no food was allowed for an hour before neuroimaging. This could have added further variance in the data, and future work should consider controlling food intake on the day of scanning. Fourth, our method used statistical control for false positives as implemented in SPM8, which may influence the probability of type I versus type II errors. Alternate approaches for correction, such as Monte Carlo simulations might also be appropriate for consideration in future analyses. Finally, the present data are correlational, so the causal direction of the association cannot be inferred. Further research will be necessary to determine whether there is a causal link between exercise, brain responsiveness, and food desire, or whether all may be driven by general attitudes surrounding health and fitness consciousness. Nevertheless, the present findings are intriguing and suggest that there are significant associations between physical exercise and the responsiveness of key appetite and reward regions related to food intake.

Conclusion

Self-reported regular physical exercise was associated with reduced functional responses to calorie-rich foods within brain regions involved in reward processing, appetite, and visceral interoceptive sensations, including the mOFC and insula. Moreover, lower activation in clusters in these regions was significantly correlated with reduced preference ratings for specific high-calorie foods, particularly those with a savory flavor. While it is well established that physical exercise increases calorie expenditure and cardiovascular fitness, our findings raise the possibility that regular physical exercise may also have indirect effects on health by diminishing functional brain activation in regions that influence preferences for less-healthy high-calorie foods.

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Conflicts of interest

There are no conflicts of interest.

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ORIGINAL ARTICLE

Cortico-limbic responsiveness to high-calorie food images predicts weight status among women

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OBJECTIVES: Excessive weight gain and obesity are currently among the world's major threats to health. Women show significantly higher rates of obesity and eating disorders relative to men, but the factors contributing to these gender differences remain uncertain. We examined the correlations between regional brain responses to images of high-calorie versus low-calorie foods and self-reported motivational status, including ratings of general appetite, overeating propensity, state hunger and desire for specific foods.
SUBJECTS: Thirty-eight healthy right-handed adults (22 male; 16 female) ages 18–45 participated. There were no differences between males and females with regard to age or body mass index (BMI).
RESULTS: Overall, motivational status correlated significantly with activation within the amygdala, insula and orbitofrontal cortex. Regional activation was then used to predict BMI, an indicator of long-term food consumption and energy expenditure. The combined model was significant, accounting for 76% of the variance in BMI for women, whereas the same regions were not predictive of weight status among men.
CONCLUSIONS: Findings suggest that long-term weight status is related to visual responsiveness to calorie-dense food imagery among women.

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Keywords: food; appetite; sex differences; fMRI; neuroimaging

INTRODUCTION

Problems with overweight and obesity are currently among the major public health concerns facing westernized societies. Within the United States, 2 of every 3 adults are classified as overweight,[1] and over 1 in 3 now meet criteria for obesity.[2] Excess weight gain is associated with numerous long-term health problems, including increased risk of type 2 diabetes, hypertension, stroke, cardiovascular disease, and a range of other negative health outcomes.[3] In addition, those meeting criteria for obesity are twice as likely than their normal weight peers to succumb to premature death from a variety of causes.[3] Women, in particular, appear to show the greatest problems with excess weight gain,[2] extreme obesity,[4] and higher rates of eating disorders relative to men.[5–7] Food consumption and weight gain among humans is an extraordinarily complex phenomenon, regulated by genetic,[8] central,[9] and peripheral neurobiological factors,[10] as well as complex social, cognitive and psychological variables.[11–14] Consequently, the basis for these sex differences in weight gain and food consumption remain unclear, but some evidence suggests that there are distinctions between men and women in their behavioral responses to food stimuli,[7] and even in the responsiveness of critical brain regions involved in regulating appetite and food intake.[15,16]

Individuals vary in their motivational status and behavioral control when confronted with food stimuli.[17] Incentive to consume food in the immediate environment is dictated by an individual's current hunger state[18] and general hedonic preference for specific foods.[19,20] While the long-term ability to regulate food consumption and to maintain a stable weight is associated with individual hedonic preferences for food stimuli, it may also be related to a general capacity to inhibit behavior and affective responses.[21] In particular, the ability to modulate behavior in response to tempting food is strongly linked to several aspects of impulse control, such as executive attention, inhibitory control and affect regulation,[22] capacities that are often associated with the behavioral[23] and emotion[24] regulation functions of the prefrontal cortex. The medial orbitofrontal cortex, in particular, appears to be one among several nodes within a complex neurocircuitry involved in responding to food stimuli[25–29] and regulating food intake,[30,31] a system that also likely includes the amygdala and posterior insula, among others. In the present study, we used functional magnetic resonance imaging (fMRI) to examine the responsiveness of this system to visual images of unhealthy high-calorie versus relatively healthy low-calorie foods, and correlated that activation with several self-report variables important to food motivation, including general appetite level (*Appetite*), the propensity to overeat (*Overeating*), state hunger (*Hunger*), and hedonic attraction to the individual foods (*Food Desire*). Based on prior research described above, we restricted our primary analyses to the amygdala, insula and medial orbitofrontal cortex. Activation within the regions found to be related to each of these food motivational indices was then used to predict body mass index (BMI), a stable measure of long-term food consumption. Based on our prior findings that cerebral responses to images of food stimuli were stronger in women than men,[16] and similar findings reported by others,[15] we hypothesized that such responses would be related to BMI among women, but would be weaker or non-existent among men.

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MATERIALS AND METHODS

Participants

The participants included thirty-eight healthy right-handed adults (22 male; 16 female) recruited via internet advertisements and posted flyers from the Boston metropolitan region, ranging in age from 18 to 45 years ($M = 30.1$, $s.d. = 8.3$). There were no differences between males ($M = 31.5$, $s.d. = 9.3$) and females ($M = 28.3$, $s.d. = 7.5$) with regard to age. A trained research technician screened all potential volunteers during a semi-structured telephone interview. Based on this screening, enrolled participants were deemed to be free from any history of severe medical conditions, head injury, loss of consciousness >30 min, brain tumors, seizures, neurologic conditions, symptoms consistent with Axis I psychopathology, or drug or alcohol treatment. Additionally, potential participants were excluded for current or recent use of any psychoactive medications, illicit substances or excessive alcohol intake. Normal or corrected normal visual acuity with contact lenses was required. BMI was determined through self-reported height and weight recorded on the prescan questionnaire. Men and women ranged from low normal BMI to Stage I obesity (males = 24.24, $s.d. = 3.60$, range = 19.80–33.47; females = 25.08, $s.d. = 4.01$; range = 19.84–34.78), which did not differ significantly between the groups. Written informed consent was obtained before enrollment and all participants were compensated for their time. This research study was reviewed and approved by the McLean Hospital Institutional Review Board.

Materials and procedure

Informed consent and prescan procedures began for each participant between 0900 and 1100 hours, during which participants completed a number of self-report inventories querying about demographic information, dietary intake and appetite/food consumption behavior. For the present analysis, participants answered the following questions: (1) 'what is your appetite like?' on a 10-point scale (*Appetite*: 1 = never hungry; 10 = always hungry), and (2) 'do you feel you eat more than you intend to' on a 10-point scale (*Overeating*: 1 = never; 10 = always). In order to ensure some variability in hunger ratings, participants were permitted to consume food if desired throughout the prescan period, although all intake throughout the day was documented on a food diary. However, no food was permitted for an hour before the fMRI scans. Men and women did not differ on any of these scales (all P-values >0.05).

Functional neuroimaging occurred between 1230 and 1500 hours. During fMRI, participants completed a food perception task, similar to the task we have reported in previous papers.[16,25–27,29,32,33] Briefly, the food perception task consisted of a series of visual images of various food and non-food items. Images were presented in 30-second blocks that alternated between images of high-calorie (H) foods (for example, ice cream, cheeseburgers, cake, French fries, candy), low-calorie (L) foods (for example, fruits, vegetables, fresh salads, whole-grain bread and fresh fish) or control (C) images (that is, non-edible flowers, rocks and shrubs). Each block consisted of 10 images, each displayed for 3 s. A fixation cross (+) was displayed for 15 s at the beginning and end of the task to allow stabilization of the signal. The food perception task followed a constant presentation order (+, C, L, H, C, H, L, C, +) and lasted for a total duration of 240 s. Participants were asked to try to attend to the images in order to identify them in a later recognition test. After the scan, participants indicated their current level of hunger (that is, *Hunger*: 1 = not at all hungry; 7 = extremely hungry). Finally, participants were again shown all of the previously seen images and asked to rate 'how much you would like to eat each item right now' (that is, *Food Desire*: 1 = do not want to eat it; 7 = strongly desire to eat it).

Magnetic resonance imaging parameters

Neuroimaging was completed on a 3.0 Tesla SIEMENS Tim Trio scanner (Erlangen, Germany) using a 12-channel head coil. A T1-weighted 3D MPRAGE sequence (repetition time/echo time/flip angle = 2.1 s/2.25 ms/12°) was collected over 128 sagittal slices (256×256 matrix) with a slice thickness of 1.33 mm (voxel size = $1 \times 1 \times 1.33$ mm). For the 4-minute fMRI during the food perception task, a T2*-weighted echo-planar imaging sequence (repetition time/echo time/flip angle = 3.0 s/30 ms/90°) was collected over 43 transverse interleaved slices with 80 images per slice (3.5 mm thickness, no skip; 22.4 cm field of view; 64×64 acquisition matrix), with a voxel size of $3.5 \times 3.5 \times 3.5$ mm. The first 3 functional scans were discarded in order to achieve a steady-state equilibrium before data collection.

Image processing

Functional neuroimaging data were preprocessed and analyzed using SPM8 software (Wellcome Department of Cognitive Neurology, London, UK). Standard realignment and motion correction algorithms were employed to remove the effects of participant movement. The echo-planar images were coregistered to each individual's own T1 anatomical image, and spatially normalized to the template of the Montreal Neurological Institute. An isotropic Gaussian kernel (full width at half maximum = 6 mm) was used to spatially smooth the images, which were then resliced to $2 \times 2 \times 2$ mm. During preliminary statistical modeling, the time series was convolved with the canonical hemodynamic response function and a first-level autoregressive model was used to remove the effects of serial autocorrelation. Low frequency drift in the signal was removed by applying the default 128-second high pass filter.

Statistical analysis

The fMRI data were analyzed using a two-stage process. First, the various conditions (that is, high-calorie foods, low-calorie foods and control images) were each modeled against an implicit baseline and contrasts comparing the various conditions were constructed (for example, high-calorie versus low-calorie conditions). The high greater than low calorie contrast image for each subject was then used as the dependent variable in a second-level random effects multiple regression analysis. In this analysis, individual responses to the questions about *Appetite*, *Overeating*, *Hunger* and *Desire* were entered as separate predictor variables. The linear relation between each predictor variable and brain activation was examined separately while holding the effects of the other variables constant. Based on our *a priori* hypotheses, we restricted the primary analyses to six bilateral search territories (that is, bilateral amygdala, insula and medial orbitofrontal cortex) as defined by the Automated Anatomical Labeling Atlas,[34] implemented within the Wake Forest University SPM8 Toolbox PickAtlas Utility.[35] Activation maps for the regression analyses were initially thresholded at $P<0.001$, k (extent) $\geqslant 10$ contiguous voxels, and then subjected to small volume correction for multiple comparisons within each search territory at $P<0.05$, corrected for family-wise error. Finally, to determine the role of these activation regions in long-term responses to food, brain activation data were extracted from the entire activated cluster in each SPM analysis and entered simultaneously into a multiple linear regression analysis to predict BMI in SPSS 20. Based on prior evidence of sex differences in brain responses to food, we also evaluated this prediction separately for men and women. The multiple correlation coefficients from the separate regression models for men and women were compared directly using Fisher's r-to-z transform.

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RESULTS

Scale intercorrelations

Scale intercorrelations among the various items are presented in Table 1. General *Appetite* was only significantly correlated with *Food Desire*. *Overeating* was significantly correlated with greater BMI and higher *Food Desire* ratings. State *Hunger* at the time of the scan was only related to *Food Desire* ratings of the images following the scan. Other associations were not significant. Together, these correlations provide preliminary evidence of the convergent and discriminant validity of the scales.

Appetite correlations

The relation between self-reported general *Appetite* ratings and brain responses to the high- versus low-calorie food perception condition was evaluated using multiple linear regression analysis. After statistically controlling for the influence of the other three variables in the regression (that is, *Overeating, Hunger and Food Desire*), *Appetite* was not significantly correlated with greater activation within any of the regions of interest to the high-calorie versus low-calorie food images (see Table 2). However, *Appetite* was associated with significantly reduced task-related activation of a cluster of voxels within the left amygdala (see Figure 1). Table 3 shows the R^2 for the overall model and individual β contributions of each of the predictor variables to the activation of this cluster. Figure 1 and Table 2 also show that *Appetite* was correlated with reduced activation of a cluster within the left posterior insula.

Overeating correlations

The relationship between self-reported *Overeating* and task-related brain responses was also evaluated. Holding other variables constant (*Appetite, Hunger and Food Desire*), self-reported *Overeating* was associated with increased responsiveness to the high-calorie food condition for an activation cluster located within the right medial orbitofrontal gyrus (see Table 2 and Figure 1). In contrast, there were no negative correlations between *Overeating* and task-related brain responses within any of the search regions.

Hunger correlations

Hunger ratings taken immediately after the scan were also examined independently in the regression. After controlling for the other variables (that is, *Appetite, Overeating and Food Desire*), self-rated *Hunger* was positively correlated with activation within a small cluster of the right amygdala (see Table 2 and Figure 1). There were no activation clusters showing a negative correlation between *Hunger* and task-related brain activation within the regions of interest.

Food desire correlations

Actual ratings of the food images obtained immediately after the scan were also examined for their independent contribution to brain responses for the high- versus low-calorie foods. With the other variables (that is, *Appetite, Overeating and Hunger*) statistically controlled, *Food Desire* was positively correlated with activation within a small cluster within the left amygdala (see Table 2 and Figure 1), but no clusters showed any negative correlation with *Food Desire* during the task.

Exploratory whole-brain analyses

To aid in generation of future hypotheses, each of the preceding regression analyses were also examined at the whole-brain level (that is, not constrained to the hypothesized regions of interest). However, no regions of activation survived whole brain (family-wise error $P<0.05$) correction for multiple comparisons within any of the analyses.

Table 1. Intercorrelations among primary food motivation questions

Scale	*1*	*2*	*3*	*4*	*5*
1. BMI	—	−0.214	0.335*	−0.204	−0.086
2. Appetite		—	0.230	0.293	0.517**
3. Overeating			—	0.067	0.524**
4. Hunger				—	0.519**
5. Food desire					—

Abbreviation: BMI, body mass index. *$P<0.05$, **$P\leqslant0.001$

Table 2. Locations of maximally activated voxels during multiple regression analysis

Comparison region	*Cluster size (voxels)*	*x*	*y*	*z*	*SPM {t}*
Appetite					
Positive					
No active voxels	—	—	—	—	—
Negative					
Left amygdala	11	−16	−2	−14	4.09
Left insula	26	−40	−8	8	4.77
Overeat					
Positive					
R medial orbitofrontal gyrus	16	8	34	−14	4.14
Negative					
No active voxels	—	—	—	—	—
Hunger					
Positive					
Right amygdala	3	18	−2	−14	4.11
Negative					
No active voxels	—	—	—	—	—
Food desire ratings					
Positive					
Left amygdala	9	−18	−4	−16	4.62
Negative					
No active voxels	—	—	—	—	—

Abbreviation: SPM, statistical parametric map.

Multiple regression to predict BMI

The final goal was to determine whether the combined brain activation clusters identified in the preceding analyses could be used to predict an independently obtained indicator of an individual's long-term eating behavior; in this case we attempted to predict BMI from these cluster responses. For each of the five activation clusters identified in the previous analyses, the cluster eigenvariate was extracted and entered as a predictor variable in a multiple regression analysis with BMI as the dependent variable. Standard regression diagnostics were undertaken to identify particularly influential observations that may have affected the analyses. No participants scored more than 3 s.d. from the mean BMI score and no cases showed excessive influence on the regression analysis (that is, high leverage values or Cook's Distance scores). For the sample as a whole, a model including all five

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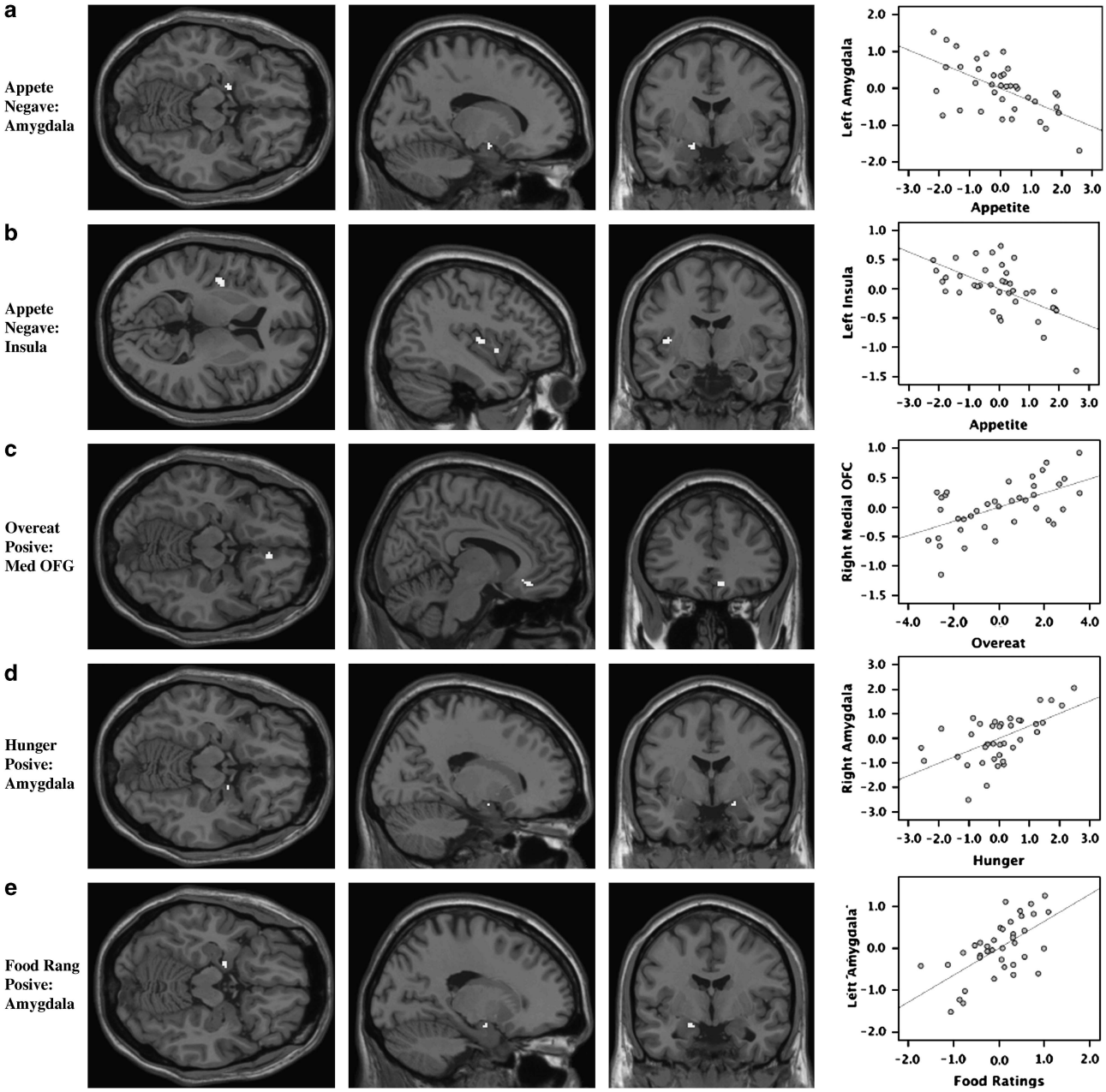

Figure 1. The figure depicts the results of the primary regression analyses for each of the four food motivation scales on brain responses to the high-calorie > low-calorie food contrast, while holding the other three scales constant. The first three columns show the locus of the primary cluster of activation revealed in the regression, and the right hand column depicts the partial correlation scatterplot between the food motivation variable and cluster signal intensity for the data in the highlighted cluster. The figure shows that general Appetite was negatively correlated with clusters in the (**a**) left amygdala and (**b**) left insula. Overeating was associated with greater responsiveness within (**c**) the right medial orbitofrontal cortex (OFC). Hunger ratings were positively correlated with a cluster in the (**d**) right amygdala, while Food Desirability ratings were positively correlated with a cluster in the (**e**) left amygdala.

activation clusters as predictors did not significantly predict BMI, $R^2 = 0.166$, $P = 0.30$. However, when the same model was tested separately by sex, we found striking differences in model prediction. Whereas there was no significant relation between combined activation from the extracted regions and BMI, $R^2 = 0.138$, $P = 0.76$ for males, activation within these same regions was highly predictive of BMI for females, $R^2 = 0.756$, $P = 0.007$. To directly compare the variance explained by these two models, we used a Fisher's r-to-z transformation of the two multiple correlation coefficients and compared the resulting difference using the z-distribution. This comparison was significant ($z = 2.62$, $P = 0.009$), suggesting that the combined activation from the five brain regions was significantly more predictive of BMI for males than for females. The results were virtually unchanged when menstrual phase was statistically controlled as a nuisance covariate. These findings suggest that long-term weight status among females is closely related to the responsiveness of these brain regions to images of calorie-rich foods. Figure 2 presents the partial correlation plots showing the association between BMI and the standardized predicted scores from the combined activation clusters for males and females.

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Table 3. Multiple regression analyses predicting extracted brain responses from food motivation

Predictor region	*β*	*t*	*Significance*
Appetite predicts left amygdala (Model $R^2 = 0.476$)			
Appetite	−0.625	−4.241	0.0002
Overeat	−0.138	−0.892	0.379
Hunger	0.159	1.036	0.308
Desire	0.714	3.637	0.001
Appetite predicts left insula (model $R^2 = 0.371$)			
Appetite	−0.685	−4.240	0.0002
Overeat	0.111	0.655	0.517
Hunger	0.065	0.387	0.701
Desire	0.142	0.661	0.513
Overeating predicts right medial orbitofrontal gyrus ($R^2 = 0.332$)			
Appetite	−0.053	0.321	0.750
Overeat	0.696	3.994	0.0003
Hunger	0.147	0.845	0.404
Desire	−0.456	−2.055	0.048
Hunger predicts right amygdala ($R^2 = 0.352$)			
Appetite	−0.172	−1.049	0.302
Overeat	0.173	1.009	0.320
Hunger	0.703	4.114	0.0002
Desire	−0.382	−1.751	0.089
Food desire predicts left amygdala ($R^2 = 0.444$)			
Appetite	−0.506	−3.528	0.001
Overeat	−0.169	−1.124	0.269
Hunger	0.053	0.354	0.726
Desire	0.869	4.548	0.0001

DISCUSSION

We examined the covariation between regional brain responses to food images and several components of food motivation that might contribute to weight gain and obesity, including general appetite ratings, overeating propensity, current hunger status and ratings of food desirability. These motivational variables were each related to activation within several regions hypothesized a priori to be central in regulating food intake, including the amygdala, posterior insula and medial orbitofrontal cortex. These regions were selected based on prior evidence of their role in processing of visual images of food[25–29] and regulating food intake,[30,31] but likely reflect only a subset of potential brain regions that may be involved. Overall, within this limited set of regions of interest, we found that those who reported greater general appetite tended to show reduced activation within the left amygdala and posterior left insula to images of high- relative to low-calorie foods, while those reporting a tendency to overeat showed greater responsiveness within the medial orbitofrontal cortex to such images. Greater self-rated hunger at the time of the scan was associated with increased responsiveness of voxels within the right amygdala to the high-calorie images, while greater desire to eat the foods depicted was associated with increased activation within the left amygdala. These regions together appear to be reliably responsive to food imagery and correlate significantly with several behaviorally relevant dimensions of eating behavior that may contribute to unhealthy weight gain. Moreover, when the activation within these regions was combined to predict a global measure of long-term food consumption (that is, BMI), there were clear distinctions between men and women in the relation between these brain responses and weight status. Whereas brain responses within these specific food-responsive regions were essentially unrelated to BMI for males, combined activation in these same regions accounted for 76% of the variance in body mass among females. These findings suggest that motivational processing of food images within the brain regions studied here may be reliably related to weight status among women, but may be less so among men.

Several important findings emerge from this study. First, we confirm that specific aspects of food motivation are related to the responsiveness of a core set of brain regions that have been implicated in prior studies of visual food imagery.[36] General appetite, which reflects an individual's self-reported persistent desire for food across settings, was inversely correlated with left amygdala and insular responses to images of high-calorie foods, such that greater responses within these regions were associated with lower appetite ratings. The amygdala has long been implicated in studies of appetitive behavior and food motivation,[37] and lesions to the amygdala often result in severe changes in food seeking and consumption.[38,39] In a prior study, appetite ratings were suppressed following a 6-week regimen of daily citicoline administration, and the magnitude of appetite decline was inversely correlated with amygdala and insular responses to images of high-calorie foods,[29] suggesting that these structures may have a role in appetite for food. The present findings are also consistent with the hypothesized role of the amygdala in detecting and responding to potential threat or harmful stimuli in the environment,[40,41] and the role of the insula in internally generated sensations of disgust.[42,43] The insula is believed to be part of the extended gustatory cortex and a key region involved in visceral sensation and interoceptive awareness.[44] Activation of this region occurs in response to satiety,[45] perception of painful and disgusting stimuli,[46] and with greater sensitivity to the visceral somatic sensations associated with anxiety.[47] This may be important for general appetite, as individuals with greater disgust sensitivity tend to be more restrained in their eating.[48] When considered in light of existing research on these brain regions, we speculate that the present finding suggest that individuals with a lower general appetite might have a broad propensity to perceive calorie-rich foods as less appealing, more aversive or even potentially threatening, leading to increased amygdala and insular responses to such stimuli. Of course, the causal direction of this association cannot be inferred from these cross-sectional data, so it remains to be determined whether reduced appetite leads to increased amygdala and insula responses to food, or whether the activation of these regions contributes causally to a decreased desire for food.

Regardless of general appetite, some individuals are particularly prone to eat more than they intend when snacking or consuming a meal. In response to images of calorie-rich foods, self-reported overeating was uniquely associated with increased activation within the medial orbitofrontal cortex, a region that is consistently implicated in reward processes and food preferences.[49,50] For example, in one compelling study, participants underwent positron emission tomography scanning while eating pieces of chocolate to the point of satiety.[51] Early in the scan, when the chocolate was still perceived as highly pleasurable, elevated brain activity was found within the caudal regions of the medial orbitofrontal cortex, proximal to the region activated here, but as participants continued to consume additional pieces of chocolate to the point of repulsion, this activation diminished and was replaced by activation within the lateral prefrontal cortex.[51] A number of studies have now suggested that the medial orbitofrontal cortex directly tracks the subjective pleasantness of stimuli,[52] and this region may contribute directly to decision-making processes that involve pleasure and reward.[53] Higher scores on a food addiction scale also correlated with greater activation within the medial orbitofrontal cortex when anticipating the receipt of highly palatable food,[54] and this region is also more responsive to food images following a fasting relative to a satiated state.[55] In fact, some studies have pointed to

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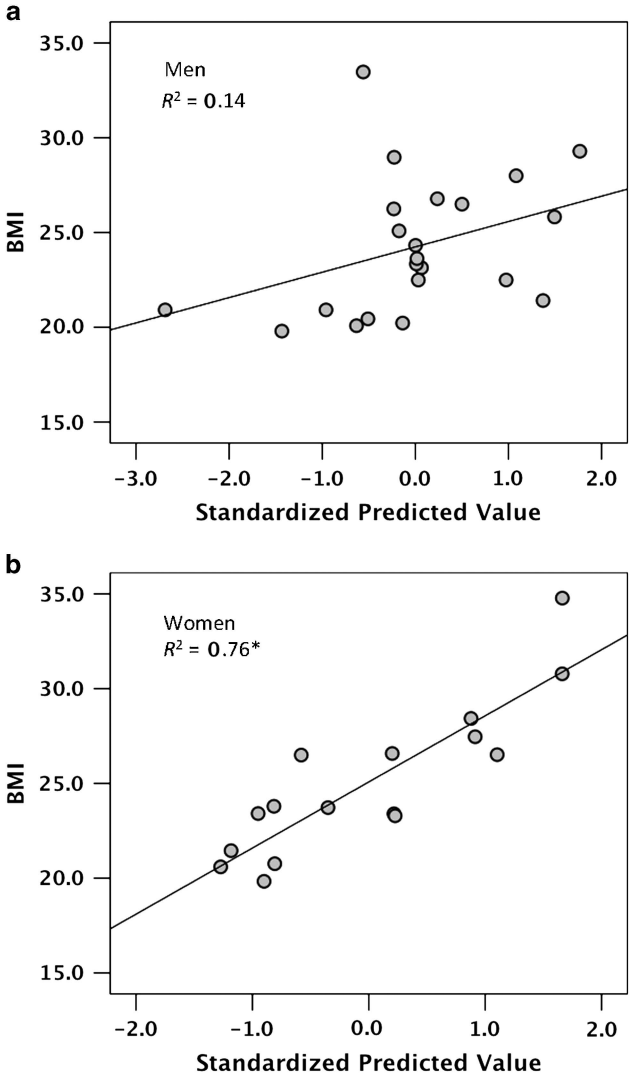

Figure 2. The figure depicts the partial correlation plots from the secondary regression analyses. In this analysis, the combined brain responses extracted from the primary analyses were used to predict BMI scores separately for men and women. Whereas the extracted functional clusters were not related to BMI among men, they were highly significantly related for women ($R^2 = 0.76$, $^*P = 0.007$).

an association between altered functioning[32,56] or structure[57] of the medial orbitofrontal cortex and general weight status. Greater responsiveness of the medial orbitofrontal cortex to high-calorie food cues may reflect a hyper-sensitivity to the reward value of such foods,[54,58] and might even serve as a risk factor for obesity. Future research may explore whether the responsiveness of this region to rewarding food stimuli may be predictive of long-term weight gain.

We also examined acute hunger at the time of the scan and found that it correlated positively with activation within the right amygdala during perception of the high- versus low-calorie food images, after controlling for other motivational variables. The present findings corroborate prior work showing that acute hunger is a powerful modulator of amygdala responses to images of food.[18,20,30,55] For instance, significantly greater right amygdala activation was found in response to food images during a hungry state (that is, 14 h fasting) compared with satiation (that is, an hour after ingesting pizza *ad libitum*).[59] A recent meta-analysis supported the modulating effect of hunger on right amygdala responses to food pictures.[36] These findings are also consistent with other research suggesting that the amygdala has an important role in determining the motivational salience of a stimulus,[60] and suggest that this salience detection system may be influenced by the motivational status of the individual. Similarly, after controlling for global appetite, overeating and hunger, we also found that the strength of desire to eat the foods depicted in the images was associated with greater response magnitude within the left amygdala to the high- versus low-calorie foods. It is particularly interesting to note that a cluster within the left amygdala was positively correlated with actual ratings of food desirability while a nearby cluster of activation was negatively correlated with general appetite, as described earlier. Although the resolution of our data precludes precise localization within the amygdala, we did find that the cluster associated with ratings of greater food desirability was located slightly more posteriorly than that associated with lower general appetite. Both clusters were collocated within an area corresponding to the superficial and centromedial nucleus groups,[61] which project extensively to the orbitofrontal cortex[62] and are broadly implicated in generating autonomic, behavioral and emotional signals based on prior learning.[63]

The primary goal of the present study was to determine the degree to which the identified food-responsive brain activation patterns might relate to long-term weight status among men and women. We found that motivation-related responses to the calorie-rich food images were highly predictive of BMI for women, accounting for up to 76% of variance in weight status, but these same regional brain responses were essentially unrelated to body mass among men. These findings raise the possibility that different factors may contribute to body weight composition for men and women. For women, body mass appears to be significantly related to specific cortico-limbic responses when confronted with visual food cues, particularly images of foods high in calorie density. On the other hand, such an association between brain responses to visual food cues and BMI was essentially absent for men, suggesting that body mass among men is likely to be more affected by any of a number of other factors that were not examined in the current study. The present findings build upon prior work showing that women tend to show greater cortico-limbic responses to visual images of high-calorie food compared with men,[16,64] and further suggest that long-term body weight status in women may be associated with greater responsiveness of the food motivation network to visual images depicting highly palatable food. Although further research will be necessary to determine the extent to which these findings may relate to actual food consumption and weight gain, these preliminary findings may have important implications regarding the higher rates of obesity[2,4] and eating disorders[5–7] among women. In light of these findings, future interventions may benefit by focusing on developing methods to circumvent the neurobehavioral links between visual responses to food and eventual food consumption. Even simple awareness of the possibility that women may be particularly responsive to visual cues of food stimuli may serve as a potential method for curtailing food intake by overtly restricting exposure to such cues.

Several limitations should be borne in mind when interpreting these findings. First, we only explored self-reported food motivation, including self-ratings of general appetite, overeating, hunger and food preferences. As self-reported motivation may differ from actual behavior, it will be important to corroborate these findings using experimental techniques that involve measuring objective eating behavior. Second, participants were screened to exclude psychopathology, and no attempts were made to recruit based on weight status, so the findings may have limited generalizability to patients with eating disorders or those at the extremes of the weight continuum. Third, with the exception of preventing food intake for an hour prior to the scan, we did not directly manipulate hunger status or total calorie

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intake. This permitted us to measure brain responses across a normal spectrum of hunger and satiety, but may have also introduced error variance that potentially reduced our power to detect some statistical effects. Future research may benefit from direct manipulation of hunger status by holding calorie intake constant for a longer interval before the scan. Fourth, although we found significant differences between men and women in the relationship between regional brain responses and BMI, we cannot exclude the possibility that the findings were driven by other factors that were not examined or controlled in the current study. It is conceivable that body mass among men may be better accounted for by some other combination of elementary biological or physiological factors such as serum testosterone, age-related somatic changes, activity level or even to social or gender-role variables that influence the circumstances surrounding food and beverage consumption in western cultures. It is also possible that the present sex difference emerged because of the greater variation in lean muscle mass as a component of BMI among men versus women[65] or even that men were simply less reliable at reporting height and weight than women. Future work should examine other more direct indices of fat to lean muscle mass to verify the currently observed sex differences. Finally, our primary hypotheses only focused on a small number of discrete brain regions. Although whole-brain analyses failed to show additional regions of correlation following stringent corrections for multiple comparisons, it is likely that at less stringent thresholds, other critically important regions may also emerge as significant. Thus, we make no claims that the regions observed here are the only ones that may be important in this process. Future work examining other regions important for food processing may also enhance our understanding of the neural underpinnings leading to weight gain and obesity. Despite these limitations, the present findings provide further support for a key network of regions involved in food motivation and further suggest that the responses within this network during visual perception of high-calorie foods are directly and strongly related to long-term weight status among women. These findings raise the speculative possibility that the vulnerabilities to weight gain, obesity and eating disorders, which predominate among women may be influenced to some extent by a greater neurocognitive responsiveness to the visual cues associated with food images.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Convergent and divergent validity of integrative versus mixed model measures of emotional intelligence

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ABSTRACT

The construct of emotional intelligence (EI) has garnered increased attention in the popular media and scientific literature. Several competing measures of EI have been developed, including self-report and performance-based instruments. The current study replicates and expands on previous research by examining three competing EI measures (Mayer–Salovey–Caruso Emotional Intelligence Test, MSCEIT; Bar-On Emotion Quotient Inventory, EQ-i; and Self-Rated Emotional Intelligence Scale, SREIS) and their relationships with cognitive functioning (Wechsler Abbreviated Scale of Intelligence; WASI), Big Five personality traits (NEO-PI-R) and emotional well-being (Beck Depression Inventory, BDI and Positive and Negative Affect Schedule, PANAS). Results indicated that significant variability in the self-report EI measures was accounted for by personality and emotional well-being measures, whereas the MSCEIT was more strongly associated with IQ. Overall, nearly two-thirds (62%) of the variance in EQ-i scores was accounted for by Big Five personality traits, emotional well-being and full scale IQ; whereas only 14% of the variance in MSCEIT scores was accounted for by these same variables. The present findings raise questions regarding the divergent validity of self-report EI measures from existing personality and emotional well-being measures. The implication of these results and directions for future research are discussed.

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1. Introduction

Since the late 1980s, there has been increased interest in the popular media and psychological literature in the concept of *emotional intelligence* (EI; Goleman, 1995; Salovey & Mayer, 1990). Conceptualizations of EI differ widely, as reflected by the broad array of available instruments that have been developed to assess the construct. Existing measures can be broadly categorized as either reflecting *specific ability*, *integrative model*, or *mixed model* conceptualizations of EI (Mayer, Roberts & Barsade, 2008). Specific ability models, as the term implies, highlight the role that a specific ability or set of abilities contribute to EI (e.g., accuracy of emotion perception and emotion regulation). According to integrative models, EI is best defined as an integration of several abilities. The most commonly used integrative measure is the Mayer–Salovey–Caruso Emotional Intelligence Test (MSCEIT; Mayer, Salovey & Caruso, 2002b). The developers of the MSCEIT define EI as the capacity to (1) accurately perceive emotion, (2) use emotions to facilitate thought, (3) understand emotion, and (4) regulate emotions. Accordingly, the test consists of four "branches" (each involving two tasks), which are designed to assess each of the four abovementioned abilities.

In contrast, mixed models adopt a relatively broader conceptualization of EI. For example, Bar-On (2004) defined EI as consisting of "*an array of noncognitive capabilities, competencies, and skills that influence one's ability to succeed in coping with environmental demands and pressures*" ([emphasis in original];

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p. 14). The breadth of the EI definition in mixed models is reflected in the widely used Bar-On Emotional Quotient Inventory (EQ-i; Bar-On, 2002; see http://ei.mhs.com and Bar-On, 2012 for commentary on the recently released EQ-i 2.0), which includes a total of 15 subscales organized under 5 primary scales: 1) *Intrapersonal* (self-regard, emotional self-awareness, assertiveness, independence, and self-actualization), 2) *Interpersonal* (empathy, social responsibility, and interpersonal relationship), 3) *Adaptability* (reality testing, flexibility, and problem solving), 4) *Stress Management* (stress tolerance and impulse control) and 5) *General Mood* (happiness and optimism). Another example of a mixed model conceptualization of EI is reflected in the work of Goleman (1995), who helped popularize the term with his book *Emotional Intelligence.* Goleman's writings suggest that he views EI as a conglomeration of characteristics, including empathy, motivation, persistence, optimism and social skills (Mayer, 1999; Mayer et al., 2002b).

Proponents of integrative models of EI have argued that popular mixed model conceptualizations are overly broad and have become "unmoored" from the core constructs of emotion and intelligence (Mayer, Salovey & Caruso, 2008). For example, Mayer, Caruso, and Salovey (1999) stated that the EQ-i assesses a range of qualities including problem-solving and reality testing that "seem more closely related to ego strength or social competence than to emotional intelligence" (p. 268). Indeed, several studies have reported significant associations between the EQ-i, Big Five personality traits and measures of emotional well-being (e.g., Brackett & Mayer, 2003; Grubb & McDaniel, 2007; Newsome, Day, & Catano, 2000; O'Connor & Little, 2003). The divergent validity of the MSCEIT has also been questioned, with studies reporting significant associations between the latter measure, key personality dimensions, and general intelligence (e.g., Schulte, Ree, & Carretta, 2004; Fiori & Antonakis, 2011).

In summary, debate persists in the field regarding how best to conceptualize and measure EI. Although EI is often discussed in the popular media and psychological literature as if it were a single monolithic construct, dominant EI measures assess a variety of facets (e.g., as reflected by the 15 subscales of the EQ-i and 4 branches of the MSCEIT). The extent to which different EI measures overlap (i.e., convergent validity) and are discriminable (i.e., divergent validity) from theoretically related cognitive (e.g., intelligence quotient [IQ]), personality (e.g., neuroticism and extraversion), and emotional constructs (e.g., affect and mood) is not well understood and is critical for establishing the validity of the EI construct. To our knowledge, no study has concurrently examined the relationship between the most commonly used measures of EI, the Big Five personality traits, emotional well-being, and full scale IQ using one of the Wechsler Intelligence Scales. This is perhaps not surprising given the subject burden and time involved in administering such a large number of measures in one study.

With regard to the existing literature, the majority of previous EI studies have included only one measure of EI (e.g., either an integrative or mixed model measure), rather than comparing several EI measures representing different theoretical models. However, Brackett and Mayer (2003) did include several EI measures in their study (including the EQ-i and MSCEIT), and examined the correlation between EI, Big Five personality traits, and psychological well-being. Interestingly, results indicated that the EQ-i shared substantial variance with personality traits and psychological well-being; whereas the MSCEIT was discriminable from these variables. In addition, and relevant to the divergent validity of EI from traditional cognitive intelligence, Bracket and Mayer found that the MSCEIT, but not the EQ-i, was significantly positively correlated with verbal SAT scores. Although the authors did not assess full scale IQ, verbal SAT scores could be considered a proxy measure of verbal intelligence.

Similar to Brackett and Mayer (2003), previous studies that have examined the association between EI measures and IQ have not used "gold standard" measures of IQ (i.e., full scale Wechsler or Stanford–Binet intelligence scales) but rather have typically used proxy, and less time-consuming, measures of intellectual capacity or restricted their analyses to particular subtests within IQ measures (e.g., see Brackett & Mayer, 2003; Ciarrochi, Chan, & Caputi, 2000; Fiori & Antonakis, 2011; MacCann, Roberts, Matthews, & Zeidner, 2004; O'Connor & Little, 2003; Roberts, Zeidner, & Matthews, 2001; Schulte et al., 2004; Zeidner, Shani-Zinovich, Matthews, & Roberts, 2005). Given the emphasis placed on establishing EI as a type of intelligence, complimenting conventional "cognitive" IQ (Mayer et al., 1999), it is surprising that no study, to our knowledge, has examined the extent to which competing measures of EI correlate with full scale IQ using gold standard measures. It should be noted, however, that Boyatzis, Good, and Massa (2012) recently reported that cognitive intelligence (assessed via the Ravens Advanced Progressive Matrices and Mill Hill Vocabulary test) was not significantly correlated with an informant, multisource assessment of emotional and social intelligence (i.e., the Emotional and Social Competency Inventory; Boyatzis & Goleman, 2007).

The goals of the current study were to examine the convergent validity (i.e., correlation between different EI measures) and divergent validity (i.e., correlation between EI, full scale IQ, Big Five personality traits and emotional well-being) of several commonly used measures of EI. We selected the MSCEIT and EQ-i as they are the most commonly used performance-based and self-report measures of EI, respectively. Differences emerging between the MSCEIT and EQ-i in patterns of associations may be due to differences in the *method of assessment* (performance-based test vs. self-report; i.e., *method variance*) or *content* (i.e., differences in the underlying EI constructs being assessed; i.e., *trait variance*). To control for differences in method of administration, an additional self-report EI measure was included (Self-Rated Emotional Intelligence Scale [SREIS]; Brackett, Rivers, Shiffman, Lerner, & Salovey, 2006), which assesses the same content domains as the MSCEIT but is administered via self-report.

We tested three hypotheses:

1) The two self-report EI measures would be significantly correlated with one another, whereas the performance-based MSCEIT would not be significantly correlated with either self-report EI measure.
2) The EQ-i, but not the MSCEIT, would be significantly associated with personality and emotional well-being measures.
3) The MSCEIT, but not the EQ-i, would be significantly associated with full scale IQ.

2. Method

2.1. Participants

Sixty-five healthy participants (33 males; 32 females) were recruited from the Boston metropolitan area via flyers and internet advertisements. The age of the participants ranged from 18 to 45 with a mean age of 30 (SD = 8.01). The sample was 69.2% Caucasian, 15.4% African-American, 9.2% Asian, 3.1% Other, and 3.1% "more than 1 race." In addition, 4.6% classified themselves as Hispanic. The native language of all participants was English. Participants were screened for evidence of psychopathology and medical conditions by a trained Bachelor's level technician using a structured series of questions. Based on screening, all participants were determined to be free of any history of Axis I psychopathology, excessive substance use, drug or alcohol treatment, or severe medical or neurological conditions. Screening questions were adapted from the Structured Clinical Interview for DSM-IV-TR (SCID-I; First, Spitzer, Gibbon, & Williams, 2001).

2.2. Measures

2.2.1. Emotional intelligence performance-based test

The Mayer–Salovey–Caruso Emotional Intelligence Test (MSCEIT; Mayer et al., 2002b) consists of 141 computer-administered items assessing the perception, use, understanding, and management of emotions. The MSCEIT yields a *Total EI* score and two Area scores, *Experiential EI* and *Strategic EI*. Experiential EI reflects the ability to perceive emotions in oneself, other persons, and different stimuli such as music and art, and to utilize emotional information to facilitate thought. Strategic EI reflects the ability to understand emotions and their evolution in oneself and others, and to manage them in an efficient and effective manner. Mayer, Salovey and Caruso (2002a) reported good reliability values for total MSCEIT scores, including internal consistency (split-half reliability = .91) and test–retest reliability (.86). For additional information on the psychometric properties of the MSCEIT, see Mayer et al. (2002a) and Mayer, Salovey, Caruso, and Sitarenios (2003). The MSCEIT was scored with the recommended "General" scoring option in which scores are based on a normative sample of 5000, rather than the Expert scoring option (i.e., relative to the responses of "emotion experts"; see Mayer et al., 2002a for detailed descriptions of scoring options).

2.2.2. Emotional intelligence self-report measures

Two self-report measures of EI were included in the present study. The Self-Rated Emotional Intelligence Scale (SREIS; Brackett et al., 2006) is a 19-item self-report measure that maps on to the emotional abilities assessed by the MSCEIT. Specifically, similar to the MSCEIT, the SREIS assesses the perception, use, understanding and management of emotions (in both oneself and others). Responses are made on a 5-point Likert scale ranging from 1 ("very inaccurate") to 5 ("very accurate"). In a series of studies, Brackett et al. (2006) reported the following Cronbach's alphas for the SREIS (.84, .77, and .66 for Studies 1, 2, and 3, respectively).

The Bar-On Emotional Quotient Inventory (EQ-i; Bar-On, 2002) is a commonly used 133-item self-report inventory of EI that yields a *Total Emotional Quotient* (EQ) and five composite scores (i.e., *Interpersonal*, *Intrapersonal*, *Adaptability*, *Stress Management*, and *General Mood*). The Interpersonal scale provides a measure of perceived empathy and interpersonal skills, whereas the Intrapersonal scale reflects self-perceived awareness of one's own emotions and self-regard. The Adaptability scale reflects the perceived ability to objectively analyze problematic situations, to solve them and to adapt to changing environments. Stress Management reflects tolerance of and perceived self-control during stressful or demanding situations. The General Mood scale reflects self-reported positive thinking and overall contentedness with personal life. Bar-On (2004) reported that the EQ-i demonstrated good reliability (internal consistency and test–retest reliability). For detailed information on the psychometric properties of the EQ-I, see Bar-On (2004). The EQ-i was scored using the "General Population" norm option (i.e., relative to a North American normative sample of 3831). Responses are made on a 5-point scale (1 = not true of me, 5 = true of me).

2.2.3. Cognitive functioning

The Wechsler Abbreviated Scale of Intelligence (WASI; Pearson Assessment, Inc., San Antonio, TX) was administered to assess cognitive ability. The measure provides scores for Full Scale IQ, Verbal IQ, and Performance IQ. The WASI is one of the most widely used intelligence scales and has reported reliability of .98 for Full Scale IQ, with high test–retest reliability, and correlates .92 with the more comprehensive Wechsler Adult Intelligence Scale-III (WAIS; Pearson Assessment, Inc., San Antonio, TX), the current gold standard in intelligence testing. A trained and experienced bachelor's level research assistant who was blind to the study hypotheses administered and scored the WASI under the supervision of a licensed doctoral level neuropsychologist.

2.2.4. Big Five personality traits

The Revised NEO Personality Inventory (NEO PI-R; Costa & McCrae, 1992) is a 240-item, self-report measure of the Five-Factor Model of personality (i.e., Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness). Responses are made on a 5-point Likert scale ranging from *strongly disagree* to *strongly agree*. The NEO PI-R has demonstrated generally adequate reliability: internal consistency = .56–.81; test–retest reliability = .66–.92 (see Costa & McCrae, 1992).

2.2.5. Emotional well-being

Two commonly used measures of emotional well-being were administered to participants. The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) is a 20-item self-report measure assessing current affective state. The measure consists of 10 items assessing positive affect (e.g., interested and enthusiastic) and 10 items assessing negative affect (e.g., upset and ashamed). Each item is rated on a 5-point scale ranging from 1 ("very slightly or not at all") to 5 ("extremely"). Ratings were averaged to obtain separate subscale scores for positive and negative affect. PANAS scores have demonstrated high test–retest reliability and acceptable convergent validity (Watson et al., 1988). The Beck Depression Inventory (BDI; Beck & Steer, 1987) is a 21-item self-report measure of depressive symptoms, with good psychometric properties (Beck, Steer, & Garbin, 1988).

2.3. Procedure

To minimize participant fatigue, given the relatively large number of performance-based tests and self-report measures administered, testing occurred over two consecutive days. During the first testing session, participants completed demographic forms, consent forms, the MSCEIT, EQ-i, SREIS and PANAS. On the second day of testing, participants completed the WASI, NEO-PI-R and BDI. All assessments were administered according to the standardized instructions provided in the manuals or other published materials. All measures were administered on paper with the exception of the EQ-i and MSCEIT, which were administered online, and the NEO, which was administered via computer. Only one participant was tested at a time. Average total duration of testing was approximately 5 h for Day 1 and 4 h for Day 2. Participants were compensated $200 following the completions of all assessments.

3. Results

3.1. Data analytic strategy

Pearson correlations were used to test the linear associations between EI (MSCEIT, EQ-i and SREIS), IQ (WASI), Big Five personality traits (NEO) and emotional well-being (BDI and PANAS) variables. Means, standard deviations, and correlations for investigated variables are listed in Table 1. Briefly, mean IQ was approximately two-thirds of a standard deviation above the population mean, which is perhaps not surprising given the education level of our sample recruited from the greater Boston area (mean years of schooling = 14.9). EI, personality traits and emotional well-being means were similar to population norms. Three participants did not complete the NEO, WASI or BDI, and one participant did not complete the PANAS. These participants were excluded from the analyses involving these measures.[1] To control for potential confounds associated with socioeconomic status, U.S. Census Bureau data were obtained on the median inflation-adjusted 12-month household income and the percentage of the participant's neighborhood below the poverty line (U.S. Census Bureau, 2010) based on census tract of home address. These census data and categorical data for racial background were used as covariates in a series of partial correlation analyses.[2] Finally, we also conducted a series of hierarchical multiple regression analyses to evaluate the proportion of variance in EI measures accounted for by linear combinations of the predictor variables. Correlations were corrected using the conservative Bonferroni method for multiple comparisons within each separate set of analyses.

3.2. Convergent validity

Using Bonferroni-adjusted alpha levels (.05/3 = .017), scores on the two self-report EI measures (EQ-i and SREIS) were significantly positively correlated with each other ($r = .50$; $p < .001$). Scores on the performance-based MSCEIT were significantly correlated with the SREIS ($r = .32$; $p = .011$) but not with the EQ-i ($r = .11$; $p = .379$).

3.3. Divergent validity with IQ

Using Bonferroni-adjusted p-values (.05/9 = .006), the MSCEIT was significantly correlated with Full Scale IQ ($r = .52$; $p < .001$), Verbal IQ ($r = .52$; $p < .001$), and Performance IQ ($r = 43$; $p < .001$). The SREIS was significantly correlated with Verbal IQ ($r = .37$; $p = .003$), but not with Performance IQ ($r = .17$; $p = .180$) or Full Scale IQ ($r = .30$; $p = .017$). As evident in Table 1, the EQ-i did not correlate with Full Scale IQ ($r = .22$; $p = .085$), Performance IQ ($r = .24$; $p = .060$), or Verbal IQ ($r = .17$; $p = .194$).

Of note, the MSCEIT remained significantly associated with all three IQ variables in partial correlation analyses controlling for socioeconomic and demographic variables including age, neighborhood median household income, percentage of the neighborhood below the poverty line, and ethnic status (all $rs > .45$ and $ps < .006$). However, the SREIS was no longer significantly associated with Verbal IQ ($r = .28$; $p = .049$).

3.4. Divergent validity with Big Five personality traits

Using Bonferroni-adjusted alpha levels (.05/15 = .003), the MSCEIT was not significantly correlated with any of the Big Five traits (Neuroticism $r = -.17$, $p = .198$; Extraversion $r = -.02$, $p = .890$; Agreeableness $r = .11$, $p = .404$; Conscientiousness $r = -.14$, $p = .265$; and Openness $r = .23$, $p = .068$).

In contrast, significant associations did emerge between the two self-report EI measures and the Big Five. Specifically, the SREIS was significantly correlated with Extraversion ($r = .41$; $p = .001$) and Openness ($r = .45$; $p < .001$). The SREIS, however, was not significantly correlated with either Neuroticism ($r = -.28$; $p = .030$), Agreeableness ($r = .20$; $p = .121$) or Conscientiousness ($r = .20$; $p = .113$). The EQ-i was significantly correlated with three of the Big Five factors (Neuroticism $r = -.60$, $p < .001$; Extraversion $r = .46$, $p < .001$; Conscientiousness $r = .49$, $p < .001$; but not with Openness $r = .32$, $p = .011$ or Agreeableness $r = .20$, $p = .121$).

3.5. Divergent validity with emotional well-being

Next, and while using Bonferroni-adjusted p-values (.05/9 = .006), we examined the association between EI, negative and positive affect (PANAS — NA/PA) and depressive symptoms (BDI). The MSCEIT was not significantly associated with scores on the BDI ($r = -.10$; $p = .433$), PANAS — NA ($r = -.17$; $p = .193$) or PANAS — PA ($r = -.19$; $p = .132$).

[1] Three outliers were identified and deleted. Specifically, one outlier for the BDI ($z = 4.91$) and two outliers for the PANAS — Negative Affect subscale ($z = 4.33$; $z = 3.40$) were identified. These datapoints were deleted.

[2] There were no significant gender differences in mean EI, IQ, Big Five personality traits, BDI or PANAS scores. In addition, given some evidence that IQ and EI increase with age (e.g., Bar-On, 2004; Mayer et al., 2002a), we tested the correlation between age and scores on the WASI, MSCEIT, EQ-i and SREIS. All correlations were non-significant with the exception of a significant *negative* correlation between age and SREIS scores ($r = -.39$; $p = .001$). Given that the SREIS is a self-report measure of EI, such a negative association may reflect the fact that younger participants are more likely to *perceive* themselves as having higher EI than older participants (whether or not their actual EI is higher than older participants). Of course, this one significant correlation must be interpreted in the context of non-significant correlations between age and the most commonly used self-report (EQ-i) and performance-based (MSCEIT) EI measures.

Table 1
Means, standard deviations and correlations for investigated variables.

Variable	M	SD	2	3	4	5	6	7	8	9	10	11	12	13	14
1. MSCEIT	103.06	12.09	.11	.32*	.52**	.52**	.43**	−.17	−.02	.23	−.14	.11	−.10	−.19	−.17
2.EQ-i	101.42	13.63	—	.50**	.22	.17	.24	−.60**	.46**	.32*	.49**	.20	−.46**	.32**	−.26*
3. SREIS	3.85	0.42		—	.30*	.37**	.17	−.28*	.41**	.45**	.20	.20	−.54**	.13	−.08
4.IQ-Full	111.10	16.08			—	.92**	.91**	−.27*	.12	.40**	−.11	.28*	−.35**	−.19	−.05
5.IQ-Verbal	110.40	15.76				—	.69**	−.21	.07	.37**	−.14	.23	−.38**	−.26**	.02
6.IQ-Perf.	108.85	15.43					—	−.30*	.15	.36**	−.06	.28*	−.25*	−.10	−.09
7. NEO-N	50.81	11.21						—	−.25	−.20	−.11	−.11	.48**	−.04	.19
8. NEO-E	54.55	11.70							—	.10	.04	.15	−.24	.25	.06
9. NEO-O	55.16	10.40								—	.13	.35**	−.32*	.00	.00
10. NEO-C	50.10	12.21									—	−.04	−.26*	.35**	−.22
11. NEO-A	47.53	11.02										—	−.21	.06	−.07
12. BDI	3.61	4.44											—	−.21	.24
13. PANAS — PA	29.03	7.46												—	−.17
14. PANAS — NA	11.65	2.33													—

Note: MSCEIT = Mayer–Salovey–Caruso Emotional Intelligence Test; EQ-i = Bar-On Emotional Quotient Inventory; SREIS = Self-Rated Emotional Intelligence Scale; IQ-Full/Verbal/Performance = Wechsler Abbreviated Scale of Intelligence — Full-Scale/Verbal IQ/Performance IQ; NEO-N/E/O/C/A = The Revised NEO Personality Inventory — Neuroticism, Extraversion, Openness, Conscientiousness, and Agreeableness; BDI = Beck Depression Inventory; PANAS = Positive and Negative Affect Schedule.
* $p<.05$.
** $p<.01$.

In contrast, a significant negative correlation emerged between the SREIS and BDI ($r=-.54$; $p<.001$). However, there was no significant association between the SREIS and either the PANAS — NA ($r=-.08$; $p=.533$) or PANAS — PA ($r=.13$; $p=.291$). Similarly, the EQ-i was significantly negatively correlated with the BDI ($r=-.46$; $p<.001$), but not with the PANAS — NA ($r=-.26$; $p=.044$) or PANAS — PA ($r=.32$; $p=.010$).

3.6. Multiple regressions including all predictors

Given the associations between IQ, Big Five traits, and emotional well-being measures (see Table 1), multiple regression analyses were conducted to test which of these variables would remain significantly associated with EI when competing variables are included as covariates (standardized betas [βs] and associated p values are reported below). In addition, such a comprehensive model allowed us to estimate the percentage of variance (adjusted R^2) associated with each EI measure that is accounted for by the combination of independent variables (IVs). See Fig. 1 for a graphical representation of multiple regression results.

First, a comprehensive model was tested in which full scale IQ, each of the Big Five traits, and the three emotional well-being variables were included as IVs, and the MSCEIT was the dependent variable (DV). A nonsignificant trend emerged for the overall model (F (9, 48) = 2.05, $p=.057$), with an adjusted $R^2=.14$. Only full scale IQ remained significantly associated with the MSCEIT when all other independent variables were controlled ($\beta=0.45$, $p=.006$; all other $ps>.37$).

The same comprehensive model, including all study IVs, was examined with the SREIS as the DV. In this case, the overall model was significant (F (9, 48) = 4.51, $p<.001$), with an

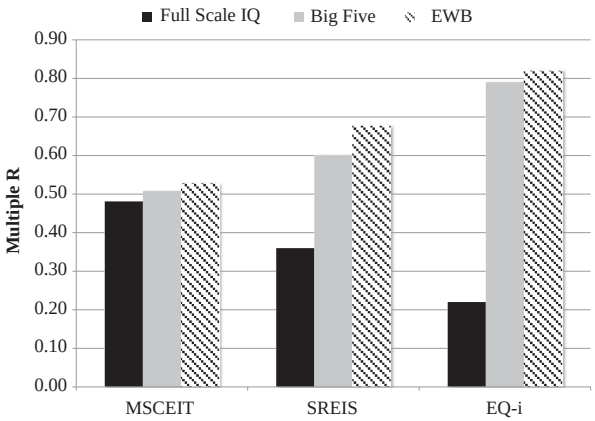

Fig. 1. Multiple *R*s for Full Scale IQ, Big Five traits and emotional well-being (EWB) scales regressed on total EI scores for the MSCEIT, EQ-i, and SREIS. Specifically, three hierarchical multiple regressions were conducted (one for each EI measure). For each regression, independent variables were entered in three blocks (Full Scale IQ in Block 1, Big Five traits in Block 2, and emotional well-being scales in Block 3). The sample size for each of these three regressions was 58. With regards to collinearity diagnostics, Tolerance values ranged between .62 and .83, and thus did not indicate any concerning multicollinearity (Field, 2009; Menard, 1995).

adjusted $R^2 = .36$. The Big Five traits of Extraversion ($\beta = 0.31$, $p = .012$) and Openness ($\beta = 0.30$, $p = .030$) were positively associated with SREIS scores. In addition, scores on the BDI were negatively associated with the SREIS ($\beta = -0.39$, $p = .006$). All other $ps > .38$.

Finally, the above comprehensive model was tested with the EQ-i as the DV. The overall model was significant ($F(9, 48) = 11.25$, $p < .001$), with an adjusted $R^2 = .62$. (The adjusted R^2 remained .62 even when full scale IQ was excluded from the model.) Three of the Big Five traits were significantly associated with EQ-i scores (Neuroticism $\beta = -0.32$, $p = .002$; Extraversion $\beta = 0.34$, $p < .001$; and Conscientiousness $\beta = 0.32$, $p < .001$). Openness was associated with the EQ-i at the level of a nonsignificant trend ($\beta = 0.21$, $p = .053$). In addition, scores on the PANAS — PA were positively associated with the EQ-i ($\beta = 0.22$, $p = .021$). All other $ps > .34$.

4. Discussion

Although there is general agreement that the ultimate relevance of emotional intelligence (EI) lies in its ability to predict important life outcomes (e.g., quality of interpersonal relationships, academic or occupational success), debate persists in how best to operationalize (e.g., integrative versus mixed models) and measure EI (e.g., self-report versus performance-based instruments). Different conceptualizations of EI are reflected in the broad array of available instruments that have been developed to assess the construct. The current study investigated the convergent and divergent validity of several competing measures of EI (MSCEIT, EQ-i, and SREIS). Consistent with our hypotheses, and paralleling the findings of Brackett and Mayer (2003), we found that the performance-based MSCEIT was discriminable from the personality and emotional well-being (EWB) variables we examined, whereas the two self-report measures of EI (EQ-i and SREIS) shared significant variance with these variables. This was particularly true for the EQ-i. Indeed, whereas the MSCEIT was not significantly associated with any of the personality or EWB variables examined, the EQ-i was significantly correlated with all but one of these variables at conventional statistical significance levels ($p < .05$; i.e., 4 of the Big Five personality traits and all 3 of the EWB variables; see Table 1). In addition to the findings of Bracket and Mayer, several previous studies have also reported significant associations between the EQ-i, Big Five personality traits, and measures of emotional/psychological well-being (e.g., Grubb & McDaniel, 2007; Newsome et al., 2000; O'Connor & Little, 2003). Overall, and as discussed in more detail below, the current findings raise concerns regarding the extent to which the EQ-i is discriminable from existing personality and EWB constructs.

As noted above, the EQ-i was designed to assess a broad "*array of noncognitive capabilities, competencies and skills*" ([emphasis in original]; Bar-On, 2004, p. 14), including items measuring constructs that seem to overlap substantially with EWB and Big Five personality traits (e.g., scales assessing optimism, happiness, stress tolerance, self-regard, self-actualization, and impulse control). Thus, given the items comprising the EQ-i, it is perhaps not surprising that the measure was found to be significantly associated with the EWB and personality measures included in the current study.

In addition to the overlap in semantic content between the EQ-i and emotion/personality measures, similarities in method of assessment may help account for the patterns of associations we observed. More specifically, given that the EQ-i, Big Five personality traits, and EWB variables were each assessed via self-report, significant correlations between these variables may have been due in part to *shared method variance* (Kazdin, 2003). It should be noted, however, that a third EI measure (SREIS) was included in the current study, which was also administered via self-report but was designed to assess the same content domains as the MSCEIT. The fact that the SREIS exhibited relatively weaker associations with EWB and personality variables than did the EQ-i suggests that the particularly strong associations between the latter measure and EWB/personality variables may be due at least in part to overlap in actual content (i.e., *trait variance*) rather than due entirely to shared method variance.

Furthermore, the difference in patterns of correlations between the performance-based MSCEIT and self-report measures may have been due in part to the influence of mood/affect on *perception* of EI. For example, those subjects with relatively higher levels of depressed mood may be more likely to *perceive* themselves as possessing low levels of EI, regardless of their "actual" EI (reflected in a significant negative association between BDI scores and both SREIS and EQ-i total scores). In contrast, given that the MSCEIT is a performance-based test rather than a self-report questionnaire, it may be less susceptible to perceptual biases associated with emotional state (as reflected in a lack of association between EWB variables and MSCEIT scores).

It is important to note that significant correlations with personality and EWB variables do not necessarily impugn the validity of the EQ-i. Indeed, as noted above, the scale was explicitly designed to contain subscales assessing a variety of facets, including domains overlapping with EWB and personality dimensions (Bar-On, 2002, 2004). In other words, the domains assessed in the EQ-i ultimately reflect the perspective of the test developer regarding what constitutes EI (Bar-On, 2006). On the other hand, the current findings indicate that nearly *two-thirds* (62%) of the variance in EQ-i scores was accounted for by Big Five personality traits and EWB; whereas only 14% of the variance in MSCEIT scores was accounted for when all variables were included in the model. These findings raise the important question: How much additional information does the EQ-i provide above and beyond existing personality and EWB measures? It will be important for future studies of EI to include Big Five personality traits and EWB measures as covariates in their analyses to reduce the risk of third variable confounds (e.g., a significant association between EI and important life outcomes may in fact be a spurious correlation accounted for by Big Five personality traits and/or EWB).

In contrast to the findings regarding personality and EWB, and consistent with our hypothesis, IQ was more strongly associated with the MSCEIT than either of the two self-report EI measures (although a significant correlation did emerge between the SREIS and verbal IQ). In addition to including several competing measures of EI, one of the strengths of the current study was the assessment of full scale IQ using a reliable and well-validated instrument (i.e., WASI). Previous studies have typically used proxy, and less time-consuming,

measures of IQ or restricted their analyses to particular subtests of IQ measures (e.g., Brackett & Mayer, 2003; Ciarrochi et al., 2000; MacCann et al., 2004; O'Connor & Little, 2003; Roberts et al., 2001; Schulte et al., 2004; Zeidner et al., 2005). In light of the current pattern of findings, it is interesting to note that the developers of the MSCEIT emphasized the importance of establishing EI as a type of intelligence, complementing traditional IQ (Mayer et al., 1999; Mayer & Salovey, 1993). In statistical terms, the authors argued that a valid measure of EI should correlate moderately with, but remain discriminable from, IQ. Correlations between the MSCEIT and IQ scales were in the "moderate" to "large" range in the current study (Cohen, 1992). Interestingly, the EI measure with the second strongest association with IQ was the SREIS (same content as MSCEIT but different assessment method), followed by the EQ-i (both different content and different method of assessment than the MSCEIT). These findings suggest that it will also be important to control for IQ in future EI studies, particularly when performance-based measures of EI are being employed. Moreover, given the distinction intelligence theorists have made between fluid (G*f*) and crystallized intelligence (G*c*), fruitful findings may also emerge from future research examining the relationship between EI and G*f* versus G*c*. Previous findings have suggested a relationship between the MSCEIT and both G*f* (e.g., Di Fabio & Palazzeschi, 2009; Fiori & Antonakis, 2012) and G*c* (Mayer, Roberts, et al., 2008). As well, within our own dataset, we found significant correlations between the MSCEIT and variables arguably tapping G*f* (MSCEIT–WASI [Performance IQ] $r = .43$) and G*c* (MSCEIT–WASI [vocabulary subtest] $r = .542$).

Finally, with regard to convergent validity, it is interesting to note that the two self-report measures of EI (i.e., EQ-i and SREIS) – which cover different content – were more highly correlated than the MSCEIT and SREIS which were designed to cover the same content, but used different methods of assessment (i.e., performance-based versus self-report, respectively). There are a number of different interpretations of these results, but the latter pattern of findings may reflect the effect of shared method variance on influencing the strengths of correlations between EI measures. Furthermore, to the extent that the MSCEIT does accurately assess EI, these findings may reflect the fact that, on average, an individual's report of their own EI abilities and actual EI performance do not correlate highly. Indeed, self-report measures are inherently limited by the fact that they rely on an individual's ability to accurately assess and report on the construct being assessed (in this case, EI). Similar criticisms have also been raised of the validity of self-report measures of IQ (Paulhus, Lysy, & Yik, 1998). Worthwhile findings may emerge from research utilizing other approaches to assessing EI, including multisource ("360°") assessments, videotapes of simulations or behavioral coding of taped interviews (Boyatzis, 2009; Boyatzis et al., 2012).

4.1. Limitations

Several limitations of the current study should be noted. First, our sample size was small, limiting statistical power. Nevertheless, we found a number of significant and intriguing relationships in line with our hypotheses, despite the fact that the especially conservative Bonferroni method was employed to adjust for multiple comparisons (Perneger, 1998; Rothman, 1990). Second, the current study was cross-sectional and not a prospective, longitudinal investigation, which prevented us from drawing inferences regarding the direction of associations (e.g., between emotional well-being and EI), and predicting longer-term life outcomes from EI scores (e.g., changes in interpersonal relationships, academic or occupational success). Third, we used the abbreviated WASI rather than the full WAIS to assess IQ. Finally, we should also reiterate that the SES data we obtained was from U.S. census tract statistics (U.S. Census Bureau, 2010) rather than a measure of each participant's individual SES status.

4.2. Future directions

More studies are needed which directly compare the psychometric characteristics of competing EI measures, including their convergent and divergent validity relative to existing measures of personality traits, emotion constructs and cognitive functioning. Ultimately, as noted above, it will be critical for studies to compare the incremental predictive validity of competing EI measures in predicting relevant life outcomes, after controlling for relevant personality variables and IQ. The results of such research may help inform the development of improved EI measures. In addition, the application of EI research to clinical populations may yield fruitful findings. For example, although effective psychosocial interventions have been developed and tested for clinical depression, the "active ingredients" of these treatments and precise mechanisms of symptom change remain unclear. Perhaps improvements in EI, or particular facets of EI (e.g., emotion management), in part mediate the therapeutic improvement experienced by clinically depressed patients in psychotherapy (e.g., in cognitive behavioral therapy which directly targets emotion regulation skills).

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Physical Exercise Habits Correlate with Gray Matter Volume of the Hippocampus in Healthy Adult Humans

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Physical activity facilitates neurogenesis of dentate cells in the rodent hippocampus, a brain region critical for memory formation and spatial representation. Recent findings in humans also suggest that aerobic exercise can lead to increased hippocampal volume and enhanced cognitive functioning in children and elderly adults. However, the association between physical activity and hippocampal volume during the period from early adulthood through middle age has not been effectively explored. Here, we correlated the number of minutes of self-reported exercise per week with gray matter volume of the hippocampus using voxel-based morphometry (VBM) in 61 healthy adults ranging from 18 to 45 years of age. After controlling for age, gender, and total brain volume, total minutes of weekly exercise correlated significantly with volume of the right hippocampus. Findings highlight the relationship between regular physical exercise and brain structure during early to middle adulthood.

The human brain is in a constant state of morphological change. Throughout the lifespan new neurons may be formed while others will die[1], some dendrites will branch while others retract[2], and new synapses are created while others are eliminated[3,4]. This dynamic remodeling of gray matter occurs against the backdrop of the more protracted process of axonal myelination, which forms the bulk of cerebral white matter[5]. While much of this remodeling is scripted by genetic factors[6] and follows a fairly well-characterized developmental course from infancy through old age[7–9], there is growing evidence that the organization and development of some cerebral systems is not purely hard-wired and can be significantly influenced by many non-genetic factors, including cognitive activity[10], nutrition[11,12], and even physical exercise[13–15].

Within the animal literature, physical exercise appears remarkably effective at facilitating some aspects of neural plasticity, particularly within the hippocampus, the brain region where the most significant expression of neurogenesis during adulthood has been observed[16]. Greater levels of physical exercise have been shown to increase the formation of new neurons[17,18] and to expand dendritic complexity within the dentate gyrus of adult rodents[19]. Moreover, the beneficial effects of exercise on brain structure and function appear to translate to humans as well. For instance, adolescents with higher physical fitness levels also show greater gray matter volume within the hippocampus and achieve better scores on cognitive tests than their peers with poorer aerobic capacity[14,20]. Similarly, physically fit elderly individuals also show larger volumes of the hippocampus and demonstrate correspondingly better memory performance[21]. Perhaps most importantly, older adults who are randomly assigned to engage in a regular aerobic exercise program show significant increases in hippocampal and cortical volumes and improved cognitive performance after a year relative to matched elderly participants in a non-exercise control group[13,15], suggesting a causal role of aerobic activity on remodeling critical brain tissues and connections. The underlying mechanisms associated with these changes still remain to be elucidated, but likely include increased blood flow and oxygenation to the hippocampus[22], as well as increased production of brain neutrotrophic factors and their receptors[23,24]. At present, the evidence for the beneficial effects of physical exercise on increasing brain volume and enhancing cognition within developing children and older adults is quite convincing. However, there is a dearth of information regarding the association between physical exercise and brain structure within the years comprising early to middle adulthood. This lack of data for this age range is concerning, as it is perhaps the period of greatest vocational productivity, family investment, and effortful contribution to society during the human lifespan.

Here, we examined the correlation between physical exercise and gray matter volume within the hippocampus among healthy adults in the second through fifth decades of life. Participants underwent structural magnetic resonance imaging (MRI) and completed questionnaires about their exercise habits, including the frequency and

duration of typical workouts during a given week. Using voxel-based morphometry (VBM), the exercise variables were entered into a series of multiple regression analyses to predict gray matter volume within the hippocampus, after controlling for age, gender, and total brain volume. It was hypothesized that the number of minutes of exercise per week would be positively correlated with left and right hippocampal volumes.

Results

Exercise levels. Overall, 46 (75%) of participants indicated that they engaged in some regular form of exercise, while 15 (25%) did not. The ratio of men (72.7%) to women (78.6%) who exercised routinely was not significantly different, $\chi^2 = 0.28$, $p = .60$. Participants reported working out an average of 3.16 *Sessions Per Week* ($SD = 2.31$), with the frequency ranging from 0 to 7 workouts during a typical week. On average, participants reported that they worked out 45.00 *Minutes Per Session* ($SD = 36.40$), with sessions ranging from 0 to 120 minutes in duration. Finally, the mean calculated total *Minutes Per Week* of exercise per individual was 189.06 ($SD = 189.45$), and ranged from 0 to 840 minutes.

Total hippocampal volume correlations. We correlated the extracted hippocampal volumes with each of the exercise measures, controlling for age and gender. The number of exercise *Sessions Per Week* was not significantly correlated with either left (partial $r = .204$, $p = .120$) or right (partial $r = .143$, $p = .279$) hippocampal volume. The number of *Minutes Per Session* was marginally correlated with the volume of the left (partial $r = .245$, $p = .061$) and right (partial $r = .254$, $p = .052$) hippocampus, but only at a non-significant trend level. However, when these two variables were combined, there was a significant correlation between the number of *Minutes Per Week* of exercise and the volume of both left (partial $r = .310$, $p = .017$) and right (partial $r = .305$, $p = .019$) hippocampus (see Figure 1).

As a control region, we extracted the volume estimates from the right and left thalamus, which have previously been found to be unrelated to physical exercise[15]. As expected, the number of exercise *Sessions Per Week* was not correlated with either left (partial $r = .132$, $p = .320$) or right (partial $r = -.031$, $p = .814$) thalamic volume estimates. Similarly, the number of *Minutes Per Session* was also not correlated with left (partial $r = .173$, $p = .189$) or right (partial $r = .018$, $p = .895$) thalamus. Finally, the number of *Minutes Per Week* of exercise was also unrelated to the volume of the thalamus on either the left (partial $r = .218$, $p = .097$) or right (partial $r = .079$, $p = .552$).

Voxel-wise hippocampal correlations. To further explore the association between exercise variables and hippocampal volume, we conducted a voxel-wise analysis of gray matter volume within two search territories defined by the hippocampal ROIs. These analyses were corrected for multiple comparisons within the volume of the hippocampus. For this analysis, there were no correlated voxels within either the left or right hippocampus for *Sessions Per Week* or *Minutes Per Session*. Additionally, no voxels survived correction for multiple comparisons within left hippocampus for the number of exercise *Minutes Per Week*. There was, however, a cluster of 21 voxels within the right hippocampus (MNI: $x = 27, y = -7, z = -21$; $T_{57} = 3.38$; $p = .072$, FWE corrected) where the gray matter volume showed a significant correlation with the number of *Minutes Per Week* of exercise (see Figure 2). There were no voxels within either hippocampus showing a negative correlation between physical exercise and gray matter volume.

As a control region, the left and right thalamus were also examined using a voxel-wise analysis constrained to the anatomically defined ROIs, with appropriate small volume correction for multiple comparisons. As expected, no voxels within either thalamus were found to correlate with *Sessions Per Week*, *Minutes Per Session, or Minutes Per Week* of exercise.

Exploratory voxel-wise whole brain correlations. To provide additional information beyond the a priori hypothesized ROIs, we also conducted a series of exploratory whole-brain voxel-wise correlational analyses. Applying an FWE cluster correction ($p < .05$) across the entire brain, we found that neither the number of exercise *Sessions Per Week* nor the number of *Minutes Per Session* of exercise were associated with any gray matter volume differences. However, as evident in Figure 3, we did find that the number of *Minutes Per Week* of exercise was positively correlated with larger gray matter volume within a cluster of 1047 voxels located within the left posterior insula (MNI: $x = -51, y = -21, z = 12$; $T_{57} = 4.23$; $p = .015$, cluster-wise FWE). For this analysis, there were no clusters showing negative correlations with any of the exercise variables.

Discussion

Better aerobic fitness has been reliably associated with increased hippocampal volume and improved cognitive functioning in developing children[14] and elderly adults[15], but almost no data are available concerning this relationship in healthy early to middle aged adults. We therefore examined the association between self-reported exer-

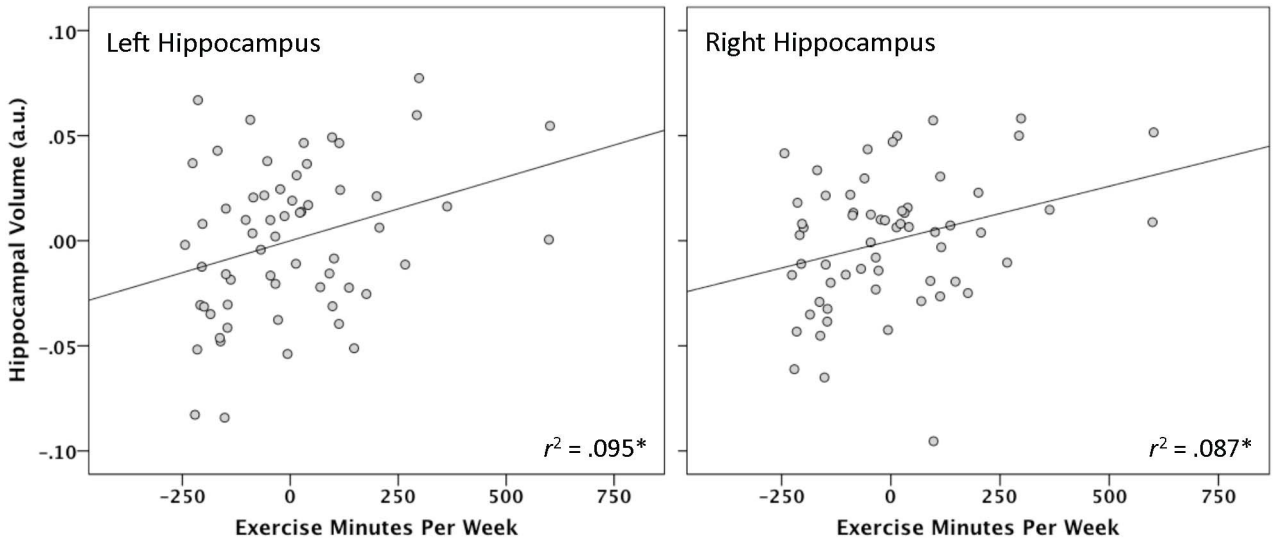

Figure 1 | Partial correlation plots showing the positive association between the residualized values for the mean number of minutes of exercise per week and the residualized modulated volume data extracted from each hippocampus after controlling for age and gender (*p < .05).

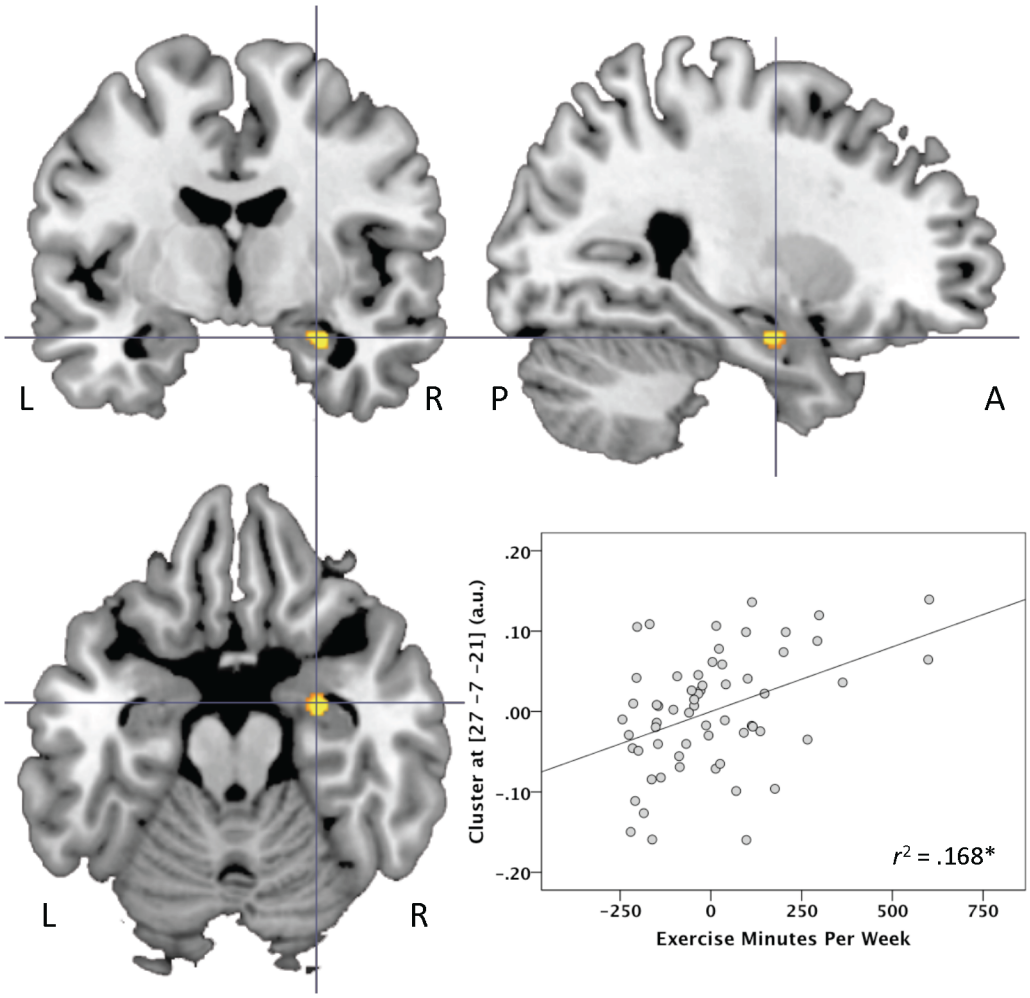

Figure 2 | The figure shows the coronal (upper left), sagittal (upper right), and axial (lower left) slices highlighting the location of a cluster of 21 voxels within the right hippocampus (MNI: x = 27, y = −7, z = −21) where the tissue volume showed a significant correlation with the number of *Minutes Per Week* of exercise, after small volume correction for multiple comparisons (*p < .10, FWE). For visualization purposes, the scatterplot (lower right) shows the significant association between the residualized values for the mean number of minutes of exercise per week and the mean extracted data from the significant cluster in the hippocampus after controlling for age and gender.

cise levels and gray matter volume of the hippocampus among adults ranging in age from 18 to 45. First, using a standardized atlas-based extraction of non-linearly modulated normalized VBM gray matter volume estimates, we found that the total gray volume measures within the left and right hippocampal regions were significantly positively correlated with the total number of minutes of weekly physical exercise, but not with the number of workouts per week or the number of minutes of exercise per workout session when considered individually. On the other hand, volume estimates for the left and right thalamus, which served as control regions, were unrelated to exercise variables. Second, to provide better spatial localization and statistical control, we conducted a voxel-wise analysis of gray matter volume within each hippocampal ROI, correcting for family-wise error within the search territories, and found that a small region of the right anterior hippocampus was significantly positively correlated with the number of minutes of exercise reported per week, but not with the frequency of workouts or typical session duration. Finally, we conducted an exploratory whole-brain VBM analysis correlating each of the exercise variables with gray matter volume. After correcting for multiple comparisons, only a single region of the left posterior insula showed a positive association between weekly total minutes of exercise and gray matter volume. These findings are consistent with the association between physical exercise and hippocampal volume that is well established in developmental and geriatric samples, but the present data further extend that work by showing that these associations are robust even during the developmentally stable period of early to middle adulthood and may include other regions such as the posterior insula.

The causal role of exercise in modifying hippocampal neuroplasticity has been well documented in the animal literature[25]. Numerous studies have demonstrated that increased wheel-running in mice leads to enhanced neurogenesis, cell survival, dendritic complexity, and long-term potentiation within the dentate gyrus[17,18,26,27]. These neural changes are due, in part, to an increase in the production of brain derived neurotrophic factor (BDNF)[24] and its associated receptor tyrosine kinase trkB[23]. Moreover, exercise and the accompanying brain changes are also associated with enhanced cognition and memory performance in rodents[28,29]. For instance, wheel-running was associated with enhanced spatial learning and retention in the Morris water maze[18,29], and leads to improved discrimination of spatial location among adjacent stimuli with identical physical appearance[30]. Together these findings suggest a strong link between increased physical activity, hippocampal growth, and cognitive functioning in animal models.

The benefits of exercise on brain structure and cognition extend to humans as well. Accumulating research suggests that physical exercise increases hippocampal volume in humans and leads to concomitant enhancement of cognitive functioning. However, most studies

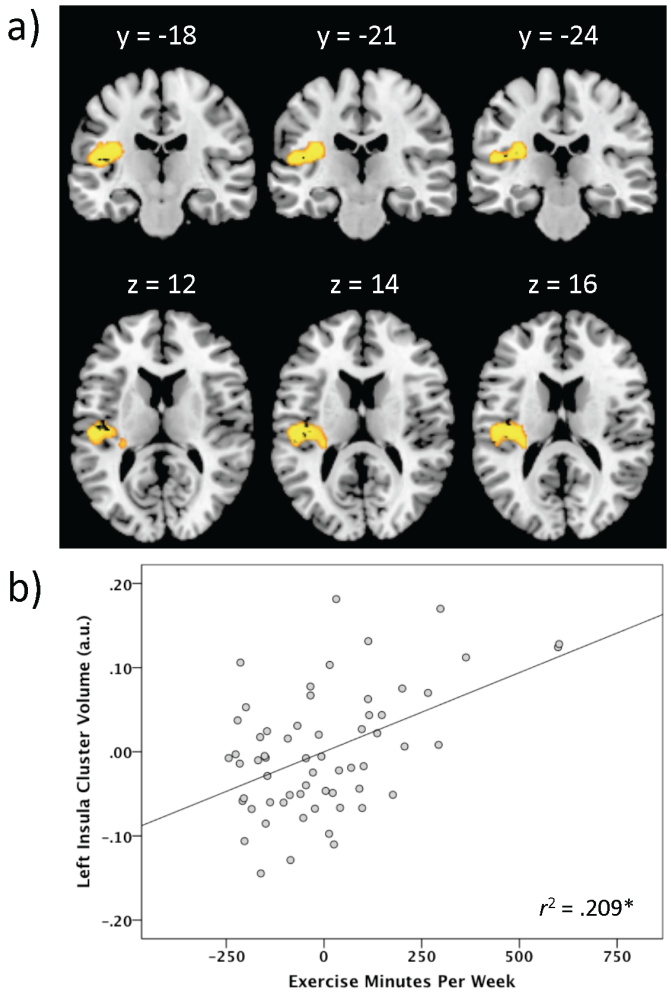

Figure 3 | (a) The figure shows the spatial location of a large cluster of 1047 voxels where there was a significant association between the number of *Minutes Per Week* of exercise and the mean gray matter volume within the left posterior insula (MNI: x = −51, y = −21, z = 12; *p < .05 cluster corrected). The top three slices show the cluster in the coronal view and the bottom three slices show it in the axial view. (b) For illustrative purposes only, the scatterplot shows the linear association between the residualized number of *Minutes Per Week* of exercise and the mean gray matter volume within the left posterior insula cluster.

of the effects of exercise on brain structure and function have examined either children, adolescent, or elderly populations. For instance, correlational studies have shown that physical fitness levels among preadolescent children are associated with larger hippocampal volumes, which in turn correlate with greater relational memory performance[14]. Similarly, adolescents with higher fitness levels also show larger volumes of the hippocampus, which correlates with greater learning of visuospatial information[20]. While existing neuroimaging research among children and adolescents is primarily correlational, a growing number of studies have demonstrated a causal connection between exercise and hippocampal volume in older adults. Whereas the hippocampus typically declines in volume at a rate of about 1 to 2% each year after the fifth decade of life in healthy adults[31], regular aerobic activity may provide a significant protective effect that can eliminate or even reverse this shrinkage of the hippocampus[15,21] and medial temporal lobe regions[32]. For example, one year of aerobic exercise training halted this decline in healthy older adults and was associated with an increase in hippocampal volume by approximately 2%, a finding that has also correlated with improved memory performance[15]. Aerobic training is also effective at increasing

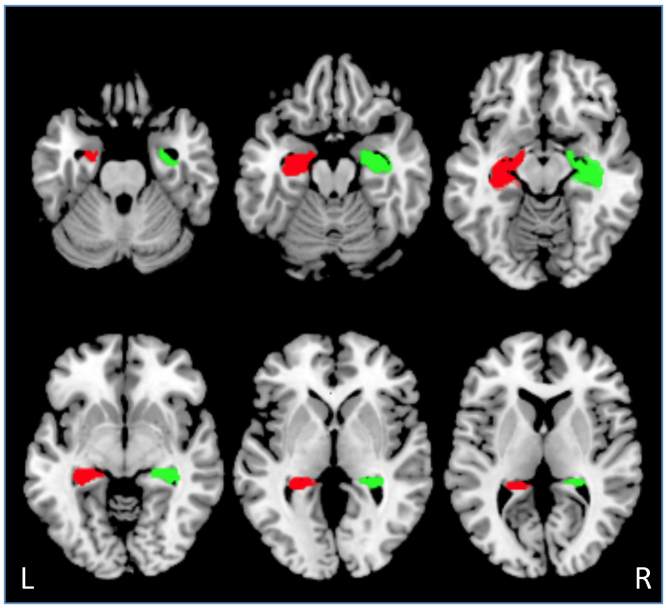

Figure 4 | **Axial views showing the left (red) and right (green) hippocampal regions of interest (ROIs) as defined by the Automated Anatomical Labeling (AAL) Atlas[50] within in the Wake Forest University PickAtlas Utility for SPM8[51].**

overall brain tissue volume[13] and is associated with improved cognitive functioning among older adults[33]. Thus, the accumulating evidence strongly suggests that regular physical exercise is associated with increased hippocampal volume and enhanced cognitive functioning among developing children and the elderly.

In contrast to the burgeoning literature examining the effects of exercise on brain structure and function in the early and later years of life, studies examining these relationships in healthy younger to middle aged adults are presently few in number. To our knowledge, this is the first study of the association between physical exercise and hippocampal volume among healthy individuals during the second through fifth decades of life, a period when total cortical gray matter volumes are typically much more stabilized and show gradual decline relative to the dynamic reductions that occur during the early and late life periods[34,35]. We found that the reported number of minutes of exercise per week was significantly correlated with hippocampal volume, particularly within a small region of the right anterior hippocampus. It is noteworthy that the region where exercise appears to correlate with hippocampal volume corresponds roughly to the spatial location of the dentate gyrus, the primary region where hippocampal neurogenesis has been well established to occur[16]. Our findings are consistent with other preliminary work showing that regular exercise training was associated with increased cerebral blood volume (CBV) within the dentate gyrus of healthy adults, a finding that was interpreted as evidence of increased neurogenesis[22]. When considered in light of existing findings, the present results suggest that physical exercise is not only beneficial for supporting brain and cognitive development in children and adolescents and staving of brain degeneration in older age, but may also be associated with improved structure and function within the years of early and middle adulthood. For many, this phase of life represents a period dedicated to intense formal higher education, interpersonal and family investment, sustained work productivity, and professional achievement. It is not difficult to conceive of the potential benefits of maximizing natural brain architecture and cognitive performance through optimized levels of physical exercise and other health-promoting behaviors during this phase of life.

Finally, to contribute to the general body of knowledge and subsequent hypothesis generation, we also conducted an exploratory

corrected whole-brain analysis without a priori hypotheses. In that analysis, we found that physical exercise was positively correlated with gray matter volume within a region of the posterior left insula. Interestingly, several studies have suggested that gray matter volume within the insular region may be specifically affected by physical exercise. The insular cortex is believed to play a role in "cardiovascular control" or "central cardiovascular command," the regulation of physiological processes such as heart rate and arterial blood pressure[36]. A recent study suggested that greater aerobic capacity in humans was positively correlated with gray matter density within insular cortex[37], although their findings were localized toward the anterior regions and lateralized to the right. Aerobic activity appears to be protective against volume loss in the insula, which is often observed among sedentary non-exercising individuals[38]. Other findings have also shown greater insular volume in long-term practitioners of Tai Chi Chuan[39] and following a year of twice-weekly resistance training[40]. The insular cortex is a complex brain region that plays a number of crucial roles in interoceptive awareness of physiological status[41]. The insula has been shown to contribute to a broad spectrum of human affective responses including visceral sensations that can predispose individuals to mood and anxiety disorders[42,43] and sensations associated with attraction or repulsion to food stimuli[44–46]. It is conceivable that exercise induced changes in insular gray matter volume could emerge as a profound mediating factor in a number of aspects of human emotional, motivational, and appetitive functions. Future research may explore the potential for these exercise induced neuroplastic effects to contribute to reducing the burden of emotional disorders and obesity.

Although the present findings are consistent with a growing number of studies suggesting a link between physical exercise and regional brain tissue volume, several weaknesses should also be considered. Perhaps the major limitation of this work was that exercise frequency and duration were estimated via one-time self-reports, which are likely to introduce some error into the data. Future work should incorporate daily exercise logs or some form of activity monitoring to remove this subjective element. Second, we focused exclusively on the frequency and number of minutes of exercise sessions, but did not further divide these data according to the type of exercise (e.g., aerobic versus resistance training) or general intensity level (e.g., walking versus running). It will be important to explore the effect of exercise type on gray matter volume in subsequent research. Finally, the use of VBM for measuring volumetric data is not without its limitations. While VBM is a valid and widely used methodology that provides exceptional consistency in the treatment of data across participants, it also loses some precision by convolving all datasets to a common template space. Alternative approaches that use expert traced or automated anatomical segmentation and parcellation methods should also be considered in future work. Despite these limitations, we believe that the present findings provide compelling data to suggest that greater levels of physical exercise are associated with larger volume of the hippocampus during the years of early to middle adulthood. The relation between these findings and healthy adult cognition deserve further exploration.

Methods

Participants. Sixty-one healthy right-handed adult volunteers (33 males; 28 females) ranging in age from 18 to 45 ($M = 30.5$ years, $SD = 8.1$) were recruited from the Boston metropolitan area to participate in a larger neuroimaging study. Participants were pre-screened via a detailed telephone questionnaire to exclude any history of Axis I psychiatric disorder, severe medical or neurological illness, moderate to severe head injury, current psychotropic medication use, or other drugs or substances that might affect functional neuroimaging. No attempt was made to select participants based on particular physical exercise habits or physical fitness level. The body mass index (BMI) of the sample ranged from 19.2 to 34.8 ($M = 24.6$, $SD = 3.4$). Participants provided written informed consent and were compensated for their time. This study was approved by the McLean Hospital Institutional Review Board. Separate data from a subset of this sample have been published elsewhere[47–49], but the correlations between physical exercise and brain volume are novel and have never before been reported.

Materials and procedure. Upon arrival at the laboratory, participants completed an information questionnaire about their daily routines, which included questions about exercise, diet, height, weight, and sleep habits. For the present report, two questions were of relevance. Participants indicated whether they routinely engaged in regular exercise ("Do you engage in regular exercise? YES, NO"), and if so, estimated the typical weekly frequency of workouts (i.e., "If 'YES': How many days per week do you exercise (circle one)? 1 2 3 4 5 6 7", which was coded as *Sessions Per Week*) and the duration of typical workout sessions (i.e., "If 'YES': How many minutes per exercise session (on average):___", which was coded as *Minutes Per Session*). From these, we calculated the simple product of *Sessions Per Week* × *Minutes Per Session* to determine the average *Minutes Per Week* of physical exercise. If participants did not routinely engage in regular exercise (i.e., endorsing "NO" to the question above), their physical exercise total was counted as zero. The threshold and specific nature of physical activity to be defined as "regular exercise" was left to the participant's discretion. Exercise was not further subdivided by type (e.g., walking; stretching; aerobic; strength) or intensity level. After completion of the questionnaires, participants underwent an MRI scan.

MRI parameters. Structural MRI was collected on a 3.0 Tesla (SIEMENS Tim Trio, Erlangen, Germany) scanner, fitted with a 12-channel head coil. For each participant, a three-dimensional T1-weighted MPRAGE sequence (TR/TE/flip angle = 2.1 s/2.25 ms/12°) was obtained over 128 slices in the sagittal plane (256 × 256 matrix). Slice thickness was 1.33 mm, yielding a voxel size of 1.33 mm × 1 mm × 1 mm.

Voxel-based morphometry (VBM). The brain images were analyzed using voxel-based morphometry in SPM8 (Wellcome Department of Imaging Neuroscience Group, London, UK; http://www.fil.ion.ucl.ac.uk/spm), using the VBM8 toolbox (http://dbm.neuro.uni-jena.de/vbm.html). The default settings for a non-linearly modulated normalized VBM were used for preprocessing in VBM8. Essentially, the T1-weighted structural brain images were DARTEL-normalized to the standard stereotaxic space of the Montreal Neurological Institute (MNI) and resliced to 1.5 × 1.5 × 1.5 mm. We used the non-linear modulated normalization procedure. Following this procedure, tissue class images are produced in alignment with the MNI template, which are then multiplied by the non-linear component of the Jacobian determinant. Thus, this method applies a proportional scaling directly to the data to control for individual variability in brain size. The images were then segmented into tissue types of gray matter, white matter and cerebrospinal fluid. Data quality checks were also undertaken within VBM8 and no artifacts or outliers were identified. Finally, the normalized gray matter images were spatially smoothed with an 8 mm full-width at half-maximum (FWHM) Gaussian kernel.

Statistical analysis. The analysis proceeded in several stages. First, we tested our a priori hypothesis that the volume of the hippocampus would correlate with physical exercise. This involved using the Region Extraction Tool (REX) to extract the mean values for the left and right hippocampi from the segmented, modulated, normalized gray matter images. The hippocampi were defined using a region of interest (ROI) approach utilizing masks created from the Automated Anatomical Labeling (AAL) Atlas[50] as implemented in the Wake Forest University PickAtlas Utility for SPM8[51] (see Figure 4). Prior work in this area also included the thalamus as a control region because it was not expected to correlate with exercise[15]. Therefore, the same ROI procedure was used to identify and extract values from the left and right thalami, which were used as control regions in the analysis. Extracted mean values for each ROI from each individual were then entered into a partial correlation analysis with each of the three exercise variables (i.e., *Sessions per Week*, *Minutes per Session*, and *Minutes per Week*) in SPSS 20 for Macintosh, controlling for age and gender. Second, a voxel-wise analysis was undertaken in SPM8 to identify the specific regions of the hippocampus that showed the strongest correlation between gray matter volume and the physical exercise variables for each of the three exercise variables, with age and gender controlled. This analysis was constrained to the hippocampal ROIs and was family-wise error (FWE) corrected for voxel-wise multiple comparisons within the ROIs ($p < .10$). Due to the small size of the region of interest and the specific a priori hypotheses based on prior animal and human literature, we tested for significance at a slightly more liberal FWE correction threshold than for an unplanned comparison. Finally, we conducted an exploratory whole-brain voxel-wise analysis, correlating each of the three exercise variables with gray matter volume throughout the brain ($p < .05$, FWE cluster volume corrected).

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Author contributions

W.K. designed the study, conducted the primary neuroimaging and statistical analyses, and wrote major sections of the manuscript. M.W. conducted the initial processing of the neuroimaging data. E.O. and M.W. assisted in the conceptualization and writing of the manuscript. All authors reviewed the manuscript.

Additional information

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PAPER

Differential influence of safe versus threatening facial expressions on decision-making during an inhibitory control task in adolescence and adulthood

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Abstract

*Social cognition matures dramatically during adolescence and into early adulthood, supported by continued improvements in inhibitory control. During this time, developmental changes in interpreting and responding to social signals such as facial expressions also occur. In the present study, subjects performed a Go No-Go task that required them to respond or inhibit responding based on threat or safety cues present in facial expressions. Subjects (*N = *112) were divided into three age groups: adolescent (12–15 years), emerging adult (18–25 years) and adult (26–44 years). Analyses revealed a significant improvement in accuracy on No-Go trials, but not Go trials, during both safe and threat face conditions, with changes evident through early adulthood. In order to better identify the decision-making processes responsible for these changes in inhibitory control, a drift diffusion model (DDM) was fit to the accuracy and reaction time data, generating measures of caution, response bias, nondecision time (encoding + motor response), and drift rate (face processing efficiency). Caution and nondecision time both increased significantly with age while bias towards the Go response decreased. Drift rate analyses revealed significant age-related improvements in the ability to map threat faces to a No-Go response while drift rates on all other trial types were equivalent across age groups. These results suggest that both stimulus-independent and stimulus-dependent processes contribute to improvements in inhibitory control in adolescence with processing of negative social cues being specifically impaired by self-regulatory demands. Findings from this novel investigation of emotional responsiveness integrated with inhibitory control may provide useful insights about healthy development that can be applied to better understand adolescent risk-taking behavior and the elevated incidence of related forms of psychopathology during this period of life.*

Research highlights

- Three age groups (12–14, 18–25 and 26–44 years) performed a Go No-Go task in response to safe or threatening face stimuli.
- Impulsive errors on No-Go trials decreased significantly between each age epoch.
- Caution and nondecision time increased with age, while bias towards the Go response decreased with age.
- Drift rate increased with age on threat No-Go trials, but did not change with age for any other trial type.
- Study findings may provide insights about healthy development to better understand adolescent risk-taking behavior.

Introduction

The successful transition from childhood to adulthood requires the rapid refinement of social cognitive skills (Nelson, Leibenluft, McClure & Pine, 2005). As adolescents become more independent and progress into a

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peer-dominated social sphere, it becomes necessary for them to successfully navigate increasingly complex social interactions (Arnett, 1999). Successful negotiation of such exchanges requires the convergence of multiple cognitive skills. One such ability is the capacity to inhibit impulsive responses in order to avoid making socially inappropriate responses or taking maladaptive actions. A second key social cognitive skill is the ability to accurately interpret cues from others, such as those discernible in facial expressions. Given that these cues are often subtle and fleeting, adolescents must learn to rapidly interpret and respond to facial cues while simultaneously regulating their own behavior in order to achieve their goals within social contexts.

Numerous studies to date examining the development of inhibitory control during adolescence have generated substantial evidence that the ability to withhold impulsive or prepotent responses improves progressively during adolescence (Cohen-Gilbert & Thomas, 2013; Hooper, Luciana, Conklin & Yarger, 2004; Johnstone, Pleffer, Barry, Clarke & Smith, 2005; Jonkman, 2006; Lamm, Zelazo & Lewis, 2006; Rubia, Smith, Woolley, Nosarti, Heyman, Taylor & Brammer, 2006). Parallel neuroimaging research has suggested that this improvement in regulatory ability is supported largely by maturation of the prefrontal cortex (PFC) (Casey, Giedd & Thomas, 2000; Paus, 2005). Such findings include consistent evidence for developmental changes in PFC recruitment during inhibitory control tasks over the course of adolescence (see Luna, Padmanabhan & O'Hearn, 2010, for review) and structural changes in the PFC (Giedd, Blumenthal, Jeffries, Castellanos, Liu, Zijdenbos, Paus, Evans & Rapoport, 1999; Gogtay, Giedd, Lusk, Hayashi, Greenstein, Vaituzis, Nugent, Herman, Clasen, Toga, Rapoport & Thompson, 2004). However, despite evidence of ongoing PFC development into emerging adulthood (Gogtay *et al*., 2004), few studies have extended developmental investigation by comparing inhibitory control between emerging adults and adults. Emerging adulthood is a developmental phase that is demographically and subjectively distinct from both adolescence and early adulthood, characterized by a shift away from direct parental supervision without acquisition of full adult roles. This period typically involves extensive identity exploration and is associated with elevated risk-taking behaviors relative to adulthood (Arnett, 2000). While largely culturally defined, emerging adulthood also features ongoing neural development in brain regions relevant to both self-regulation and social cognition (Sowell, Thompson, Holmes, Jernigan & Toga, 1999). There is, however, a dearth of cognitive studies that directly compare emerging adulthood to both adolescent and adult age groups.

Emotional responding and inhibitory control are mutually influential processes. Cognitive control may be used to alter emotion-driven responses, as well as to overcome powerful emotions that can disrupt self-control (Cyders & Smith, 2008). Brain regions critical to supporting these processes include the PFC and the amygdala, which are extensively interconnected (Ochsner & Gross, 2005). These connections demonstrate marked maturational changes throughout adolescence (Cunningham, Bhattacharyya & Benes, 2002). Emotional Go No-Go tasks have been used to explore the developing interaction between responses to emotion cues and inhibitory control in adolescence (Cohen-Gilbert and Thomas, 2013; Hare, Tottenham, Galvan, Voss, Glover & Casey, 2008; Ladouceur, Dahl, Williamson, Birmaher, Axelson, Ryan & Casey, 2006; Lewis, Lamm, Segalowitz, Stieben & Zelazo, 2006; Somerville, Hare & Casey, 2011; Tottenham, Hare & Casey, 2011). These studies have found evidence of improvements in facial emotion discrimination and inhibitory control across adolescence, with larger age-related improvements in inhibitory control observed when emotional stimuli were presented as compared to neutral stimuli (Tottenham *et al*., 2011). Neuroimaging studies using emotional faces as stimuli for Go No-Go tasks have revealed increased amygdala reactivity to faces depicting fearful affect in adolescents relative to children and adults (Hare *et al*., 2008). These adolescents also showed slowed responding to fear faces compared to adults, interpreted as a weaker capacity to override an instinctive withdrawal from a negative stimulus (Hare et al., 2008). In the same subject sample, increased impulsive errors to happy versus calm faces were also evident in adolescents relative to children and adults, a finding interpreted as an inability to withhold approach behaviors to emotionally positive stimuli. This pattern was paralleled by increased recruitment of ventral striatum, a brain area implicated in reward-processing (Somerville *et al*., 2011). Together, these studies suggest heightened sensitivity to emotional information during Go No-Go task performance in adolescents relative to other age groups. Thus, the presence of social information in the form of overt emotional facial expressions appears to be particularly disruptive to inhibitory control efforts during adolescence. To date, however, all facial affect Go No-Go tasks used to examine adolescent development have employed highly prototypical examples of basic emotions (e.g. happiness, sadness, fear, anger and neutral). Optimal social functioning in daily life requires the discernment of much subtler cues. The current study aims to explore the impact of less obvious threat and safety cues, present in facial expressions, on inhibitory control across development in adolescence, emerging adulthood and adulthood.

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The interpretation of, and responding to, facial expressions of emotion outside the context of an inhibitory control task also continues to develop during adolescence and into emerging adulthood. Evidence suggests that both the ability to discern subtle differences in facial cues and speed of recognition for emotional expressions continue to develop throughout adolescence (McGivern, Andersen, Byrd, Mutter & Reilly, 2002; Thomas, De Bellis, Graham & LaBar, 2007). In particular, the ability to distinguish subtle anger cues may remain immature throughout most of adolescence and then improve rapidly in early adulthood while the ability to identify similarly subtle fear cues improves linearly across adolescence (Thomas *et al.*, 2007). Brain activation in response to viewing facial expressions, as measured by fMRI, has also been found to change during adolescence. In particular, amygdala activity in response to viewing fearful faces was elevated in adolescents relative to adults (Guyer, Monk, McClure-Tone, Nelson, Roberson-Nay, Adler, Fromm, Leibenluft, Pine & Ernst, 2008; Monk, McClure, Nelson, Zarahn, Bilder, Leibenluft, Charney, Ernst & Pine, 2003) and decreased with age across adolescence (Killgore, Oki & Yurgelun-Todd, 2001), while PFC activity increased (Yurgelun-Todd & Killgore, 2006). Thus, processing of negative emotional facial expressions appears to shift somewhat from emotion-processing areas and to regulatory brain regions during adolescence, potentially affecting the ability to recognize and respond to emotional cues. In addition to these developmental changes in processing emotional faces, sex differences in brain activity in response to facial affect have been reported in adolescent populations (Killgore *et al.*, 2001; Killgore & Yurgelun-Todd, 2004), as well as sex differences in performance of an emotional Go No-Go task (Tottenham et al., 2011). Therefore, both age and sex may impact inhibitory control during the processing of facial cues, though the interactions between these two factors are not yet well understood.

The objective of the current study was to evaluate developmental changes in adolescence and emerging adulthood in performance of a Go No-Go task that requires fast, on-line processing of subtle computer-generated facial cues indicating threat or safety (Oosterhof & Todorov, 2008) at the same time as inhibitory demands are being placed on the subject. In this way, this protocol taps into inhibitory control as well as the ability to discriminate between safe and threatening faces. Given that a number of other factors, such as cautiousness of responding (i.e. their speed/accuracy trade-off), may impact accuracy and reaction time measures, a drift-diffusion model (DDM; Ratcliff, 1978) of simple decisions was fitted to the behavioral data. This model uses accuracy and response time data to extract values for response caution, response bias, nondecision time (time needed to encode the stimulus and execute a motor response), and stimulus processing for each individual. This type of model-based approach has been successfully applied to examine group differences in developmental studies (Ratcliff, Love, Thompson & Opfer, 2012), aging studies (Starns & Ratcliff, 2012) and psychopathology studies (White, Ratcliff, Vasey & McKoon, 2010a, 2010b).

Given that both inhibitory control and social information processing are actively developing during adolescence, it was hypothesized that accuracy on No-Go trials would improve with age and that the impact, or disruptiveness, of emotionally salient threat stimuli on accuracy would be greatest in the adolescent group. Response caution was also predicted to increase with age, while the ability to correctly discriminate between the two face types was also predicted to improve between adolescence and adulthood.

Methods

Participants

The sample consisted of 112 participants, subdivided into three age groups: (1) 33 adolescents (ADO, 16 male), mean age = 13.6 ± 0.9 years, age range = 12–15 years; (2) 38 emerging adults (EA, 17 male), mean age = 22.0 ± 1.8 years, age range 18–25 years; and (3) 41 adults (ADU, 21 male), mean age = 35.0 ± 6.3 years, age range = 26–46 years. Ages 18–25 years were chosen to represent emerging adulthood based on demographic data (Arnett, 2000) and neurodevelopmental findings (Sowell *et al.*, 1999). The racial composition of the sample was 70% Caucasian, 16% African American, 9% Asian, and 6% multiracial/other. Four percent of the sample identified as Hispanic. Participants were recruited from the surrounding community via internet postings and flyers. All potential subjects were screened via telephone interview. Exclusion criteria included history of severe medical conditions, head injury, loss of consciousness (> 30 min), brain tumors, seizures, other neurologic conditions, sensory deficits, symptoms consistent with Axis I psychopathology, or drug or alcohol dependence. In addition, potential participants were excluded for current or recent use of any psychoactive medications, illicit substances, or excessive alcohol intake. One young adult female was a statistical outlier (overall accuracy > 3 *SD* below the mean) and was therefore excluded from all analyses. Written informed consent was obtained prior to enrollment from all adult

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participants and from parents of adolescent participants. Adolescent participants provided written informed assent. All participants were compensated for their time. The research protocol was approved by the McLean Hospital Institutional Review Board.

Go No-Go paradigm

Subjects performed a computer-based social threat Go No-Go task, presented using E-Prime (Psychology Software Tools, Inc., Sharpsburg, PA, USA). The task consisted of two blocks of 180 trials (25% No-Go trials). Within each trial an image depicting a 'safe' or 'threat' face was displayed for 100 milliseconds (msec) followed by an intertrial interval of 1400 msec during which the subject could respond. In two separate blocks, subjects were instructed to either press the spacebar (Go) when safe faces were presented and refrain from pressing (No-Go) when threat faces were presented (condition A, see Figure 1a), or press the spacebar (Go) when threat faces were presented and refrain (No-Go) when safe faces were presented (condition B, see Figure 1b). Each block lasted 4.5 minutes. Due to the higher number of Go trials presented within each block, subjects developed a prepotent tendency to press and needed to actively inhibit pressing during No-Go trials. Percent accuracy on Go and No-Go trials and response time on correct Go trials were recorded. While task condition order was not fully counterbalanced, a subsample of individuals completed the task in the inverse order (condition B followed by A) and revealed no significant differences in performance due to order of task condition.

Safe and threatening face stimuli were selected from stimulus sets developed by Oosterhof and Todorov (2008) using the Facegen Modeller program (Singular Inversions Inc., Toronto, Canada, http://facegen.com, Version 3.1.). The selected face stimuli consisted of 45 different male Caucasian faces. Two different versions of these 45 faces were selected: one morphed in each direction along the 'trustworthiness' vector by three standard deviations. Across the full task (conditions A and B), each safe or threat face was presented once as a No-Go stimulus and three times as a Go stimulus.

Drift diffusion model

A DDM (Ratcliff, 1978; Ratcliff & Smith, 2004) was fit to each participant's behavioral data to extract components of psychological processing. The model assumes that noisy evidence is accumulated over time until a

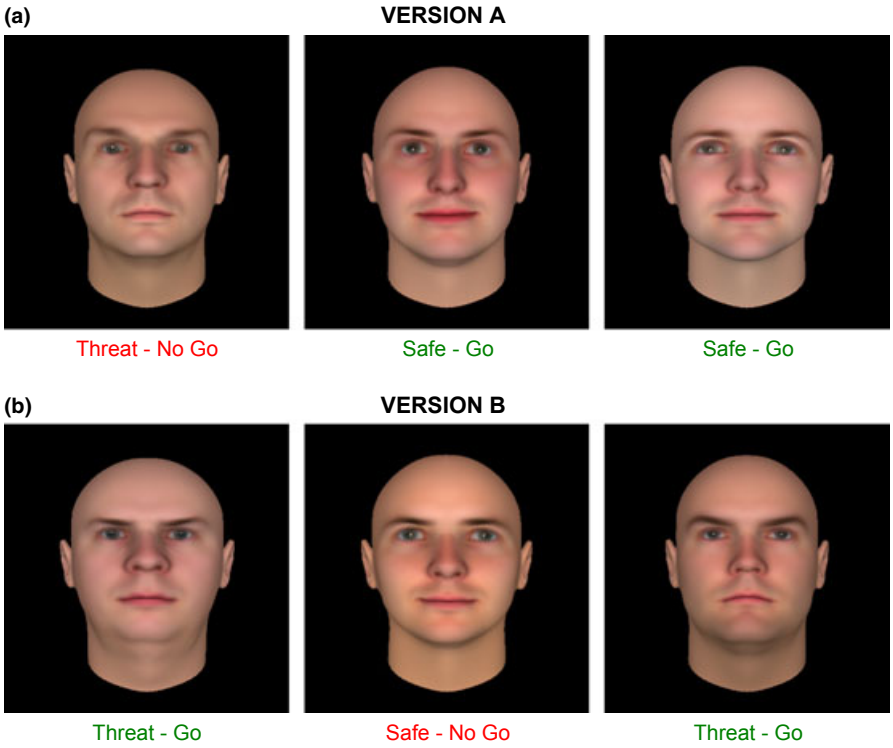

Figure 1 *Sample task stimuli. (a) In condition A, participants were instructed to press (Go) for safe faces and withhold pressing (No-Go) for threatening faces. (b) In condition B, participants were instructed to press (Go) for threatening faces and withhold pressing (No-Go) for safe faces.*

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boundary is reached, signaling a commitment to that response (Figure 2). The decision time is calculated as the time taken to reach a boundary, and the overall response time is equal to the decision time plus a value of nondecision time that accounts for the duration of other processes such as encoding and motor execution. Previous work shows that the DDM can successfully account for No-Go data by assuming that there is an implicit boundary representing the No-Go option, meaning that the individual commits to withhold the response once that boundary is reached (Gomez *et al.*, 2007).

The primary components of the model are the boundary separation, starting point, drift rate, and nondecision time. The boundary separation provides an index of response caution (the speed/accuracy trade-off); wide boundaries indicate a cautious response style that favors accuracy over speed. The starting point, *z*, provides an index of response bias. In a Go No-Go task where Go trials are much more probable than No-Go trials, people are generally biased toward the Go response and movement towards the No-Go boundary requires active suppression of the Go response (Gomez *et al.*, 2007). This is reflected by a shift in the starting point toward the Go boundary, meaning less evidence is required for that response. The nondecision time (*Ter*) provides an index of the duration of nondecision processes, including encoding of the stimulus and execution of the motor response. Finally, the drift rate (*v*) provides an index of how readily a person can extract evidence from the presented stimulus; higher absolute values of drift rate indicate strong evidence and lead to fast and accurate responses. In this present task, drift rate indexes how effectively subjects map threat and safe faces onto the correct responses. This involves not only discriminating threat from safe faces, but also relating those faces to either response execution (Go trials) or inhibition (No-Go trials). Four drift rates were calculated based on the stimulus conditions; safe face Go trials, safe face No-Go trials, threat face Go trials, and threat face No-Go trials.

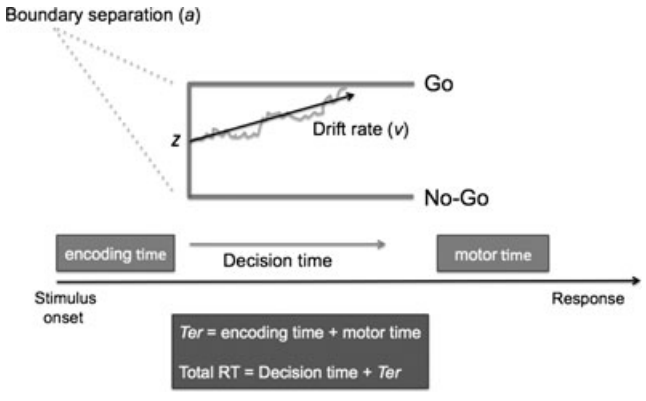

Figure 2 *A schematic of the DDM. After the stimulus is encoded, noisy evidence is sampled over time until one of the boundaries is reached (see text for details). Measures obtained from the model include boundary separation, response bias (z), nondecision time (Ter), and drift rate (v).*

The DDM was designed to account for the full observable data from all trials. This includes accuracy and the distribution of RTs for Go trials, as well as the percentage of commission errors and their RTs on No-Go trials. The model was fit to each participant's data separately for the two sessions, estimating for each session a value of response caution, drift rate for safe faces, drift rate for threat faces, response bias, and nondecision time. A parameter for across-trial variability in starting point was added to capture trial-by-trial fluctuations in response bias, allowing the model to account for extremely fast responses (i.e. guesses). Although this parameter helps the model fit the data, it is not generally used for inference and thus is not discussed further. The fitting process used the χ^2-minimization technique (Ratcliff & Tuerlinckx, 2002) based on the quantiles of the RT distribution. For correct Go responses, the standard quantiles of the RT distribution (.1, .3, .5, .7, .9) were used to compare against the predicted data from the model. The .1 quantile is the time at which 10% of responses have occurred, the .3 quantile is the time at which 30% of response have occurred, and so on (Figure 4). These RT quantiles were used with the proportion of each trial type (correct or omission) to provide the χ^2 fit index, which was minimized by a SIMPLEX routine.

Estimating the RT quantiles and the subsequent DDM parameters is facilitated by the collection of a very large number of trials. The participants in this study had over 100 observations for each Go condition, which is fewer than previous applications of the model, but enough to produce stable estimates of the distribution of RTs for each condition. Nonetheless, the estimation of the DDM parameters may have more variability because of the relatively low number of observations to constrain the model. This is a general concern for the application of DDMs in situations where data collection is limited by a variety of factors. However, there is no reason to expect that this limitation differentially affects the age groups in this study; thus the differences among the groups should not be confounded.

Statistical analyses

Accuracy on No-Go trials was analyzed as the principal index of inhibitory control while accuracy on Go trials

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was used to measure sustained attention to the task. Reaction time on correct Go trials was used to examine total encoding, decision-making, and response speed. Measures of caution (boundary separation), response bias (*z*), nondecision time (*Ter*), and drift rate (*v*) were derived from the DDM. Each outcome variable was analyzed using analyses of variance (ANOVAs) including Age (ADO, EA, ADU) and Sex (male, female) as between-subject factors. In cases where separate measures were computed for Safe and Threat trials and/or Go and No-Go trials, Face Type and Trial Type were included as within-subject factors. All post-hoc *t*-tests were two-tailed and corrected for multiple comparisons using Tukey HSD. All ANOVAs and post-hoc tests were conducted using PASW Statistics 18.0 (SPSS Inc. Released 2010. PASW Statistics for Mac, Version 18.0. Chicago: SPSS Inc.)

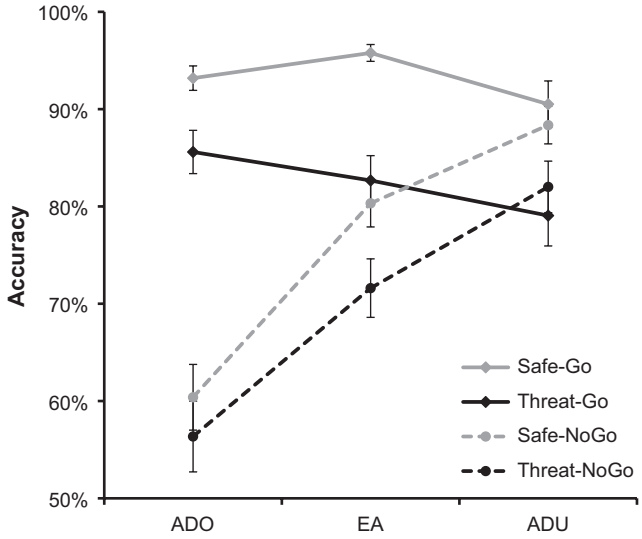

Figure 3 *Accuracy across age groups. On Go trials, accuracy did not change significantly with age and was consistently higher on Safe versus Threat trials. No-Go accuracy increased significantly with age between each age group (ADO = adolescent, EA = emerging adult, ADU = adult) and was also significantly higher on Safe versus Threat trials.*

Results

Accuracy and reaction time data

Go and No-Go accuracy

A 3(Age) × 2(Sex) × 2(Face Type) × 2(Trial Type: Go vs. No-Go) mixed-model ANOVA revealed main effects of Age, $F(2, 108) = 11.72$, $p < .001$, $\eta^2 = .178$, Face Type, $F(1, 108) = 35.78$, $p < .001$, $\eta^2 = .249$, and Trial Type, $F(1, 108) = 83.35$, $p < .001$, $\eta^2 = .436$, as well as significant Age × Trial Type, $F(2, 108) = 31.93$, $p < .001$, $\eta^2 = .372$, and Face Type × Trial Type, $F(1, 108) = 9.08$, $p = .003$, $\eta^2 = .078$, interactions. No other main effects or interactions were statistically significant.

Post-hoc analysis of the Age × Trial Type interaction was conducted using two one-way ANOVAs ($\alpha = .025$ to correct for multiple comparisons) examining the effect of Age separately for accuracy on Go trials and on No-Go trials. While the main effect of Age was not significant, $p = .14$, for Go trial accuracy, a significant main effect of Age was evident for No-Go trial accuracy, $F(2, 108) = 30.67$, $p < .001$, $\eta^2 = .362$. Significantly lower No-Go accuracy was observed in ADO compared to EA, $p = .001$, and ADU, $p < .001$, and significantly lower No-Go accuracy was observed in EA compared to ADU, $p = .019$ (Figure 3).

The Face Type × Trial Type interaction was followed up via two paired-samples *t*-tests comparing accuracy on Safe versus Threat faces separately for Go and No-Go trials. Results showed a significant effect of Face Type on both Go, $t(110) = 7.16$, $p < .001$, and No-Go trials, $t(110) = 3.88$, $p < .001$, with accuracy on Safe trials being higher in both cases. The observed interaction may be explained by a larger effect of face type on Go trials, (Safe: Mean = 93.1%, *SD* = 10.7%; Threat: Mean = 82.2%, *SD* = 16.7%) than on No-Go trials (Safe: Mean = 77.4%, *SD* = 19.3%; Threat: Mean = 70.9%, *SD* = 21.2%).

Reaction time: correct Go trials

Reaction times were analyzed via a 3(Age) × 2(Sex) × 2 (Face Type) mixed-model repeated measures ANOVA. This analysis revealed significant main effects of Age, $F(2, 108) = 3.91$, $p = .028$, and Face Type, $F(1, 108) = 21.33$, $p < .001$. No other significant effects were found. Post-hoc comparisons between each of the three age groups revealed significantly longer reaction times in ADU relative to ADO, $p = .029$, and a trend towards longer reaction times in ADU relative to the EA, $p = .09$. No significant difference in reaction time was found between ADO and EA. The main effect of Face Type was due to longer reaction times on Threat versus Safe trials. Reaction time data are summarized in Table 1.

Application of the drift diffusion model

The fit quality of the DDM was assessed quantitatively by examining best-fitting χ^2 values and qualitatively by plotting the observed data against predicted data from the model parameters. The χ^2(ADO: Mean = 187.17, *SD* = 250.85; EA: Mean = 81.06, *SD* =67.16; ADU:

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Table 1 *Reaction time and DDM measures: Mean(SD)*

Measure	Face Type	ADO	EA	ADU
Response Time (msec)	Safe	467.6(76.3)	462.4(65.3)	504.3(90.0)
	Threat	495.4(91.9)	487.0(79.4)	534.1(84.7)
Response Caution	Safe	0.126(0.02)	0.128(0.02)	0.130(0.02)
	Threat	0.127(0.02)	0.129(0.02)	0.139(0.02)
Response Bias	Safe	0.420(0.07)	0.426(0.06)	0.427(0.07)
	Threat	0.312(0.09)	0.331(0.10)	0.360(0.07)
Nondecision time	All	0.207(0.09)	0.254(0.06)	0.292(0.08)

Mean =66.28, *SD* = 34.34) values were found to be in line with results from similar studies (e.g. White *et al*., 2010a, 2010b), and the predicted data were highly similar to the observed data (Figure 4). Together these findings suggest that the DDM captured the behavioral data well, allowing confidence in the resulting parameter values.

Diffusion model parameters

Response caution

A 3(Age) × 2(Sex) × 2(Face Type) mixed-model ANOVA revealed a main effect of Age, $F(2, 105) = 4.02$, $p = .021$, $\eta^2 = .071$, on response caution. No other significant effects were found for this variable. Post-hoc comparisons between each of the three age groups revealed a significantly lower caution for ADO compared to ADU, $p = .022$, while neither of these age groups differed significantly from EA. Values for response caution, response bias and nondecision time are summarized in Table 1.

Response bias

A mixed-model ANOVA including Age and Sex as between-subject factors and Face Type as a within-subject factor revealed only a main effect of Age on response bias, $F(2, 105) = 3.43$, $p = .036$, $\eta^2 = .061$. Post-hoc comparisons showed a significant difference in response bias between ADO and ADU, $p = .049$, but no significant differences between either of these age groups and EA. Overall, ADO were more biased toward the Go response than ADU, which reduces the likelihood of successful inhibition on No-Go trials.

Nondecision time

A univariate ANOVA including Age and Sex as fixed factors revealed a main effect of Age on nondecision time, $F(1, 105) = 12.37$, $p < .001$, $\eta^2 = .191$. No other significant effects were found. Post-hoc comparisons

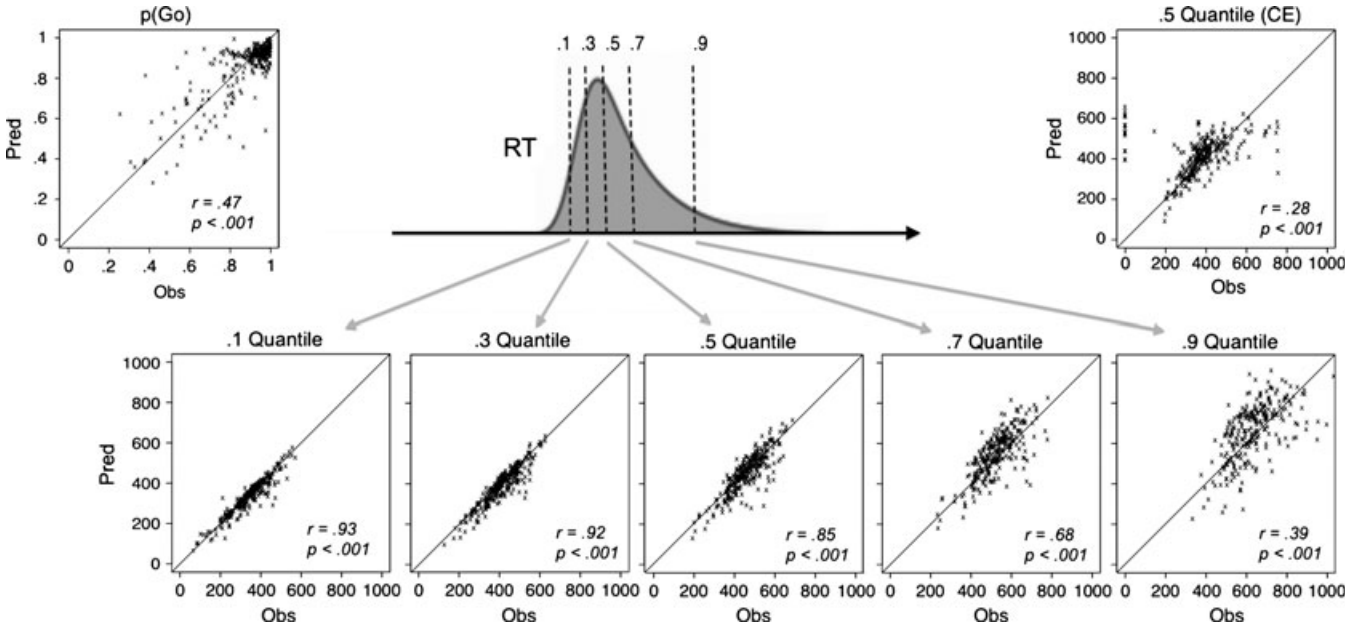

Figure 4 *Fit quality from the DDM. Predicted values from the best fitting DDM parameters are plotted against the observed values for the proportion of correct Go responses (upper left), the RT quantiles for Go responses (lower), and the median RT for commission errors (CE, upper right). Each point represents a participant. The correspondence between observed and predicted values shows that the model captured the data well.*

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between age groups revealed significantly shorter nondecision times in ADO compared to EA, $p = .024$, and ADU, $p < .001$, and a trend towards shorter nondecision times in EA relative to the ADU, $p = .056$.

Drift rate

Higher values of drift rate indicate a better ability to translate the stimulus face into the appropriate response (Go or No-Go). A 3(Age) × 2(Sex) × 2(Face Type) × 2 (Trial Type: Go vs. No-Go) mixed-model ANOVA revealed significant main effects of Face Type, $F(1, 105) = 71.64$, $p < .001$, $\eta^2 = .406$, and Trial Type, $F(1, 105) = 56.30$, $p < .001$, $\eta^2 = .349$, as well as significant Face Type × Trial Type, $F(1, 105) = 12.81$, $p = .001$, $\eta^2 = .109$, and Age × Face Type × Trial Type, $F(2, 105) = 5.80$, $p = .004$, $\eta^2 = .099$, interactions. A trend-level effect was also found for a Sex × Face Type interaction, $F(1, 105) = 3.77$, $p = .055$, $\eta^2 = .035$.

Post-hoc analyses of the Age × Face Type × Trial Type interaction were conducted first by using two mixed-model ANOVAs ($\alpha = .025$) examining the effects of Age and Face Type separately on Go trials and on No-Go trials. Drift rates showed main effects of Face Type (Go trials, $F(1, 105) = 16.80$, $p < .001$, $\eta^2 = .138$; No-Go trials, $F(1, 105) = 68.28$, $p < .001$, $\eta^2 = .394$) with drift rates being greater for safe faces than for threat faces on both Go and No-Go trials. However, only the No-Go trial analysis also revealed a significant main effect of Age, $F(2, 105) = 3.77$, $p = .026$, $\eta^2 = .067$, and a significant Age × Face Type interaction, $F(2, 105) = 4.70$, $p = .011$, $\eta^2 = .082$. Examining No-Go trials only, separate one-way ANOVAs were used to assess the impact of Age on drift rate for safe face trials versus threat face trials. While no effect of Age was observed on safe trials, $p = .755$, a significant effect of Age was found for threat trials, $F(2, 108) = 5.82$, $p = .004$, $\eta^2 = .097$. For No-Go threat trials, post-hoc *t*-tests contrasting each of the three age groups revealed a significant difference between ADO and ADU, $p = .003$, but no significant differences in drift rate between EA and the other two age groups. In summary, the ability to map safe face stimuli onto a No-Go response did not differ as a function of age while the ability to map threat faces to a No-Go response improved across the sampled age range. Drift rate findings are summarized in Figure 5.

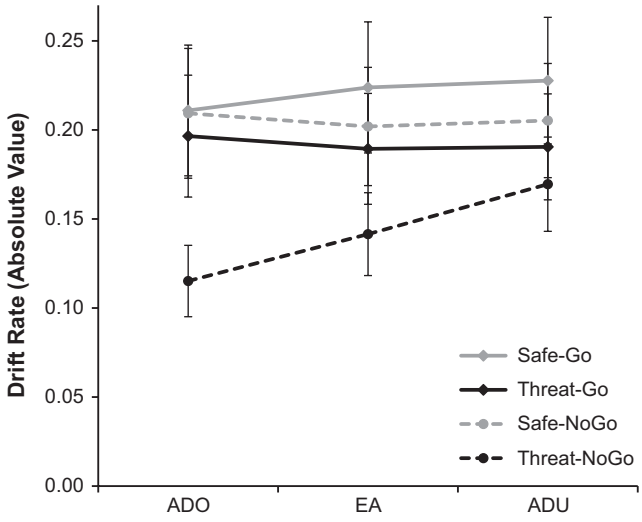

Figure 5 *Drift rate across age groups. On both Safe and Threat Go trials and Safe No-Go Trials, drift rate did not change significantly with age. Drift rate on Threat No-Go trials increased significantly with age between the adolescent (ADO) and adult (ADU) groups, while the emerging adult (EA) group did not differ significantly from either the older or the younger age group.*

Discussion

Consistent with previous reports, adolescents in the present study demonstrated worse inhibitory control than emerging adults and adults, reflected by higher error rates on No-Go trials (Hooper *et al.*, 2004; Johnstone *et al.*, 2005; Jonkman, 2006; Lamm *et al.*, 2006; Rubia *et al.*, 2006; Silveri, Sneider, Crowley, Covell, Acharya, Rosso & Jensen, 2013). While few studies have directly examined the possibility of ongoing changes in inhibitory control that extend into emerging adulthood, the current study revealed significant improvements in response inhibition between the emerging adult group (mean age = 22 years) and the adult group (mean age = 35 years). These findings are in contrast with the only existing investigation of emerging adults, in which no significant performance differences were observed between 20-year-old and 36-year-old subjects on a non-emotional Go No-Go task (Johnstone et al., 2005). However, the current findings suggest that developmental changes in inhibitory capacity do extend into adulthood when tasks are sufficiently complex, such as when simultaneous emotional information processing and response inhibition are required.

The current adult group displayed improved inhibitory control relative to both groups of younger subjects, as well as slower reaction times on Go trials, suggesting that this group sacrificed response speed in order to commit fewer impulsive errors. This interpretation is supported by the age-related increases found on the response caution measure derived from the DDM. Changes in speed/accuracy trade-off, however, do not solely account for the observed developmental changes in impulsivity.

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Age-related differences in response bias and nondecision time were also found and likely also contribute to the reduction of impulsive errors with age. In other words, multiple developmental changes in cognitive processing contribute to reductions in impulsivity between adolescence and adulthood: a shift in speed–accuracy trade-off, a reduction in the bias toward Go responding, and the use of longer intervals to encode the stimulus and execute the button press. Notably, significant changes on the majority of these metrics were not observed between the adolescent and emerging adult groups, or between the emerging adult and adult groups, suggesting that these cognitive skills develop very gradually and continue to mature well beyond the teen years. In contrast with the current findings, nondecision times have been previously reported to decrease with age between adolescence and early adulthood on a number discrimination task (Ratcliff *et al.*, 2012). Given the demands of the current task, however, adults may respond in part by increasing encoding time for the more complex stimuli or slowing motor responses to account for the need to periodically override the prepotent Go response.

The presence of a threat stimulus negatively impacted accuracy on both Go and No-Go trials, while increasing reaction times on Go trials, indicating that a threat stimulus disrupted task performance across all age groups in this study. The absence of any interaction effects between face type and age group on these measures suggests that, contrary to our hypothesis and previous findings (Hare et al., 2008), the presence of a social threat stimulus was not more disruptive than a safe stimulus in adolescents relative to the older participants. The integration of facial emotion recognition and inhibitory control into a single task provides a useful model of social cognition. However, because of this integration, this task confounds the ability to interpret facial expressions with the impact of emotional content on inhibitory control, making accuracy scores on inhibitory trials difficult to interpret. That is, accurate responding on No-Go trials depends both on the ability to correctly identify the face stimulus as either safe or threatening, and on the ability to withhold a prepotent response to an emotional stimulus. Thus, errors could result either from inaccurate identification of the stimulus, or from failed response inhibition. By fitting a DDM to the accuracy and reaction time data, this study succeeded in isolating specific aspects of the decision-making process that mature between adolescence and adulthood.

The DDM measures provide important insight into the development of cognitive skills underlying impulse control in response to threatening and safe social cues. Importantly, drift rate provides a measure of how readily a participant can extract evidence from a stimulus towards a correct response. In other words, drift rate measures how readily the safe and threat cues were mapped onto a correct response: pressing (Go trials), or refraining from pressing (No-Go trials). This measure revealed an interaction between trial type, face type and age group that was not evident when accuracy or reaction time data were examined alone. Specifically, while drift rates on both safe and threat Go trials and on safe No-Go trials appeared to be at adult levels throughout the age range studied, drift rate on threat No-Go trials was lower in adolescents versus adults. This suggests that adolescents were less able to map the negative social cue onto the correct withholding response. Thus, while increased impulsive errors were observed in the younger age groups for safe and threat No-Go trials, these errors resulted from different components of the decision-making process. Reduced caution, greater Go bias, and shorter nondecision times, resulted in more errors on safe trials in the adolescent group, with an additional factor affecting performance on threat No-Go trials: the ability to map a threatening cue onto a successful inhibitory response. This finding is congruent with previous studies that have reported a more protracted development of executive functions on motivationally or emotionally salient tasks relative to tasks with a minimal emotional component (Prencipe, Kesek, Cohen, Lamm, Lewis & Zelazo, 2010; Tottenham et al., 2011). Likewise, Go No-Go performance in the context of negative emotional distractors has been found to lag behind developmental improvements in other emotional contexts (Cohen-Gilbert and Thomas, 2013). An argument could be made that adolescents have more difficulty mapping threat onto inaction as opposed to action due to an evolutionary bias towards sensation seeking and threat tolerance in order to enable migration away from the family unit at puberty, thus reducing chances of inbreeding (Spear, 2000, 2007). A bias to act rather than freeze in response to threat may also help juveniles compete for resources and mates. These possibilities are, however, purely speculative.

It is surprising that the present study did not show any significant sex differences, as sex differences in emotional processing have often been reported among adolescents (Killgore et al., 2001; Killgore and Yurgelun-Todd, 2004) and adults (Killgore & Yurgelun-Todd, 2001). The stimuli in the present study, however, were 'facial identities' differing in perceived trustworthiness, not emotional expressions *per se*. Thus, the influence of the sex may be specific to responding to explicit emotional rather than social-threat aspects of face processing. In addition, the present study employed a homogeneous set of facial identities, all with features consistent with those

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of Caucasian males, which reduced the number of potential complex interactions and focused the analysis on the primary dimensions of interest. It is possible, however, that including female stimuli and identities consistent with other racial backgrounds might have yielded additional interaction effects, which should be explored in future research.

A strength of this study was the inclusion of relatively large sample sizes within each age group; however, it is worth noting that the adolescent group was restricted to a three-year age span, whereas the emerging adult group spanned seven years and the adult group spanned 20 years. This design assumes a slowing rate of change in cognitive skill with age across the studied range. This assumption, however, is not without basis, as the rapid development of cognitive control occurring in childhood and adolescence begins to plateau as individuals reach adulthood (Luna, Garver, Urban, Lazar & Sweeney, 2004; Luna *et al*., 2010).

The shift from a parent-centered to a peer-centered social environment places novel cognitive demands upon the developing adolescent. The absence of supervision by a caregiver increases the demand for self-regulation, as actions and decisions become increasingly independently determined. The complexity of social relationships also increases as friendships and romantic relationships become more influential sources of emotional support, self-conception, and self-esteem. Understanding the thoughts and emotions of others becomes increasingly necessary for the successful maintenance of these relationships. Thus, both inhibitory control and accurate recognition of social cues are critical skills for developing adolescent social functioning. The current study provides further evidence that these skill sets continue to improve across adolescence, and that response inhibition continues to develop during emerging adulthood, albeit potentially at the cost of response speed. Moreover, results of the current study suggest a specific impairment in translating negative social stimuli into a non-response, a finding with clear implications for adolescents who frequently may need to regulate their actions during stressful or negative interactions. It is not yet clear whether the observed developmental improvements in socially relevant cognitive skills are driven by continued maturation in brain areas such as the PFC, or if the increasingly complex social demands placed upon the individual during this period promote the continued development of relevant neural networks. It is likely that these processes are mutually influential. Whether biologically or environmentally driven, acquisition of social skills is critical to the successful navigation of adolescence.

Both reduced cognitive control and heightened responding to emotional cues have been associated with elevated risk for multiple forms of psychopathology in adolescents (Killgore and Yurgelun-Todd, 2005, 2006; Ladouceur *et al*., 2006; Silveri, Rogowska, McCaffrey & Yurgelun-Todd, 2011) and other age groups (Leppänen, 2006; Silbersweig, Clarkin, Goldstein, Kernberg, Tuescher, Levy, Brendel, Pan, Beutel, Pavony, Epstein, Lenzenweger, Thomas, Posner & Stern, 2007; Wessa, Houenou, Paillère-Martinot, Berthoz, Artiges, Leboyer & Martinot, 2007). To the extent that adolescent mental health is closely tied to successful navigation of social interactions with parents, teachers and peers (Parker, Rubin, Erath, Wojslawowicz & Buskirk, 2006; Spear, 2000), development of top-down control networks to regulate both behavior and emotional responding is critical to healthy development during adolescence and into emerging adulthood. Thus, the social-threat Go No-Go task implemented in this study may be readily used to explore questions of risk for psychopathology during development through the exploration of individual differences in task performance.

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Research Article

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Personality Traits Associated with Sleep Initiation Problems

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Abstract

Difficulties initiating and maintaining sleep may be influenced by cognitive, affective, and behavioral factors. Some evidence suggests that particular personality traits are prone toward enhanced arousal, worry, rumination, and poor cognitive and behavioral control, which may contribute to difficulties falling asleep. Presently, we tested the hypothesis that sleep initiation problems would be related to the personality traits of neuroticism, impulsivity, and high emotional control. Sixty-one healthy adults (31 males; 30 females) ranging in age from 18 to 41 completed a questionnaire about sleep problems and several measures of personality, including the NEO-PI-R, Barratt Impulsivity Scale (BIS 11), and Courtauld Emotional Control Scale (CECS). On average, participants who indicated that they had a problem with sleep initiation scored higher on scales of neuroticism, impulsivity, and emotional control. When personality traits were entered into a stepwise logistic regression, only impulsivity was retained as a significant predictor of the presence or absence of sleep onset difficulties. When sleep latency in minutes was analyzed as a continuous variable, linear regression analyses revealed that both neuroticism and impulsivity combined as significant predictors of the self-reported time to fall asleep. Findings suggest that personality factors involved in negative emotional arousal and rumination are related to problems falling asleep, but that most of the variance appears to be attributable to deficits in cognitive and emotional control.

Introduction

Sleep problems are prevalent in the adult population, with approximately 42.6% of individuals in the United States reporting at least one significant sleep problem [1]. According to the National Sleep Foundation, 26% of the general population report difficulties initiating sleep at least several nights per week [2]. Although many factors may contribute to sleep onset difficulties in otherwise healthy individuals, it is likely that factors related to personality, cognitive style, and sleep-related behavior may play a major role in the development and perpetuation of these problems.

Relative to psychiatric and medical influences on sleep problems, the potential influence of personality factors has been less extensively studied. Early work suggested that sleep initiation difficulties were often associated with an "internalizing" personality style, which was proposed to lead to exaggerated emotional arousal and physiological hyperactivation [3]. More recent conceptualizations of personality, such as the "Big Five" personality domains [4], have tended to focus on the relationship between sleep difficulties and traits associated with negative affectivity, worry, tension, and irritability, such as neuroticism. The anxious worry that characterizes individuals high in neuroticism may contribute to sleep problems by increasing cognitive rumination and sympathetic hyperarousal over concerns about uncontrollable life experiences, daily hassles, and other problems. Consistent with this notion, prior work has shown that neuroticism is associated with poor sleep quality and subjective perceptions of insomnia [5-9]. However, neuroticism may not fully explain the variability in sleep problems, and other personality factors may be important as well. Impulsiveness, for instance, is a trait that may interact with neuroticism to affect sleep onset. There is some evidence that engaging in impulsive actions during the day can be associated with feelings of guilt and regret at bedtime, ultimately hindering the ability to fall asleep easily [10]. In fact, higher impulsivity has been associated with trouble falling asleep in a healthy population [11], while some facets of impulsiveness, including urgency and lack of perseverance, have also been found to relate to insomnia severity [12].

Both neuroticism and impulsivity may be accompanied by maladaptive attempts to control negative thoughts and emotions that interfere with sleep. The extent to which an individual attempts to suppress negative emotions such as anger, anxiety, and depressive experiences comprises a construct referred to as emotional control [13]. Excessive emotional control has been linked to negative mood and health complaints, such as increased fatigue and physical symptoms of stress [14]. Likewise, other methods of emotional regulation, including thought control strategies, have been associated with sleep disturbances. For example, the use of an analytical emotion regulation strategy, specifically focusing on thoughts rather than feelings, compared with an experiential emotional regulation strategy, has been associated with sleep disruption, including shorter total sleep time, more frequent awakenings, greater wakefulness after sleep onset, and lower sleep efficiency [15]. Similarly, the use of a strategy known as aggressive suppression, whereby participants employ critical and self-punishing thoughts, is also positively associated with insomnia severity [16].

Although the constructs of neuroticism, impulsiveness, and maladaptive emotional control have each been linked to sleep difficulties in individual studies, it is not clear whether and to what extent these three factors may combine to affect sleep problems. The aim of the present study was to examine whether healthy individuals who report sleep onset trouble would differ from those without sleep onset problems with respect to neuroticism, impulsiveness, and the cognitive strategy of emotional control, and to determine if these traits may combine to affect sleep difficulties and sleep onset latency. It was hypothesized that otherwise healthy participants who endorsed trouble falling asleep would score higher on measures of neuroticism, emotional control, and impulsiveness in contrast to normal sleepers and that self-reported minutes to fall asleep would be associated with a linear combination of these personality and cognitive factors.

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Method

Participants

Sixty-one healthy adults (31 men, 30 women) aged 18-45 (M=30.3, SD=8.1) were recruited from the Boston metropolitan area via flyers and internet advertisements. Participants had obtained an average of 14.8 (SD=2.2) years of formal education. Following a detailed screening interview, all participants were deemed free of any DSM-IV Axis I psychopathology, substance use disorders, sleep disorder or serious neurological or medical conditions. As described below, participants were divided into groups according to their self-reported assessment of the presence (n=26) or absence (n=35) of difficulties falling asleep. The McLean Hospital Institutional Review Board and the U.S. Army Medical Research and Materiel Command Office of Research Protections, Human Research Protection Office approved this research, which was conducted in accordance with the 1964 Declaration of Helsinki. Written informed consent was obtained for all participants, who were compensated for their time.

Materials and procedure

Participants completed a questionnaire designed to collect information about typical sleep habits and problems, such as weekday and weekend bedtimes and wake times, daytime sleepiness, and sleep onset latency. Central to the current investigation were the participants' responses to two questions. First, all participants indicated whether they ever experience trouble falling asleep (yes/no forced choice) and, secondly, indicated how many minutes they generally take to fall asleep on typical weeknights and weekend nights, respectively. Minutes to fall asleep on weekdays and weekends were combined to create a weighted average of minutes to fall asleep per day (i.e., [(weekday minutes * 5) + (weekend minutes * 2)]/7).

To assess personality characteristics, a computerized version of the NEO Personality Inventory – Revised (NEO-PI-R) was administered [4]. The NEO-PI-R consists of 240 items rated on a five-point Likert scale from "strongly disagree" to "strongly agree." Scores are summed into five domain scales: Neuroticism, Extraversion, Openness to Experience, Agreeableness, and Conscientiousness. The Neuroticism domain scale assesses the degree of negative affectivity and comprises facets such as anxiety, angry hostility, depression, and impulsivity. The Extraversion domain scale assesses personality along a continuum from extraversion to introversion, comprising facets of warmth, gregariousness, and assertiveness. The Openness to Experience scale measures the extent to which individuals are receptive to ideas, values, and aesthetics. The Agreeableness domain includes facets of trust, compliance, and tender-mindedness, while the Conscientiousness domain is comprised of competence, dutifulness, and self-discipline. The NEO-PI-R has excellent internal consistency for the domain scales, with Cronbach's alpha ranging from 0.86 to 0.90 and shows convergent validity with Eysenck's personality dimensions [17,18].

The Barratt Impulsiveness Scale (BIS-11a) was used to measure the construct of impulsiveness [19]. The BIS contains 34 items rated on a 4-point scale from 1 "rarely/never" to 4 "almost always/always." Responses are summed to measure total impulsiveness, as well as three dimensions including: Attentional/Cognitive Impulsiveness, Motor Impulsiveness, and Nonplanning Impulsiveness [20]. Total BIS-11 scores and subscale scores were calculated from the BIS-11a scores and prorated to BIS-11 scores according to the procedures outlined by Marijn Lijffijt [21].

Participants also completed the Courtauld Emotional Control Scale (CECS), a 21-item measure that assesses the extent to which individuals attempt to exert excessive control over their negative emotions [13]. The CECS comprises three subscales, which examine degree of control over the three affective states of anger, depressed mood, and anxiety. Responses are made on a 4-point Likert scale from 1 ("almost never") to 4 ("almost always"). Sample items include "When I feel angry, I bottle it up," "When I feel unhappy, I smother my feelings," and "When I feel afraid, I say what I feel." This measure has shown good internal consistency, with Cronbach's alpha ranging from 0.86-0.88 for the subscales, and has acceptable test-retest reliability [13].

Analysis

Based on responses to the question "Do you ever have trouble falling asleep?" on the sleep questionnaire, participants were divided into two groups: those who reported any trouble falling asleep (n=26) and those who denied trouble falling asleep (n=35). All analyses were conducted in SPSS 20 for Macintosh. Shapiro-Wilk tests were used to determine the normality of each of the variables. All variables were normally distributed. Box's test was used to test that the observed covariance matrices of the dependent variables were equal across groups and Levene's test for equality of variances was used to examine variances for homogeneity. Independent samples t-tests were used to determine whether individuals who reported trouble falling asleep differed from those who did not in terms of personality (NEO-PI-R), Emotional Control (CECS), and Impulsiveness (BIS-11a).

Paired t-tests were used to compare self-reported minutes to fall asleep on weekdays versus weekends. As there were no significant differences between minutes to fall asleep on weekdays and weekends, these two variables were combined to create a weighted daily average of minutes to fall asleep. Additionally, Pearson correlations were used to examine the association between average minutes to fall asleep and personality (NEO-PI-R), Emotional Control (CECS), and Impulsiveness (BIS-11a). Finally, stepwise logistic and stepwise multiple linear regression analyses were conducted to examine the combined influences of the various personality factors on sleep onset problem category (yes/no) and sleep onset latency (minutes to fall asleep), respectively.

Results

Significant differences were observed between individuals who reported difficulties falling asleep and those who did not on two scales of the NEO-PI-R. Specifically, those who reported trouble falling asleep showed higher scores on Neuroticism (t(59)=-2.57, p=.013) and lower Conscientiousness (t(59)=2.54, p=.014) than individuals who did not have sleep initiation difficulties (Table 1). In contrast, there were no differences between individuals who endorsed trouble falling asleep and normal sleepers in terms of Extraversion (p=.377), Openness to Experience (p=.558), or Agreeableness (p=.701). Furthermore, individuals with trouble initiating sleep also reported a higher degree of Emotional Control (CECS: t(59)=-2.08, p=.042), and Impulsiveness (BIS-11a: t(59)=-2.74, p=.008) than normal sleepers (Table 1).

Next, to examine the combined contribution of neuroticism, impulsivity, and emotional control to dichotomously defined sleep onset difficulties, we conducted a forward stepwise logistic regression analysis, with the three personality factors entered as independent variables and sleep initiation problems as a binary dependent variable.

With forward entry, BIS 11a was entered at the first step, yielding a significant model, χ^2=7.14, *df*=1, *p*=.008. The odds ratio (OR=1.07) suggested that knowledge of an individual's impulsivity improved prediction of the sleep difficulty category by approximately 7%, (β=.067, *SE*=.03, *p*=.013). However, once impulsivity was statistically accounted for, neuroticism and emotional control failed to add additional predictive power and were not selected for entry into the equation.

In addition to examining sleep onset problems as a dichotomous variable, we also treated typical self-reported sleep onset latency as a continuous variable (regardless of whether it was perceived as problematic or not). As shown in Table 2, sleep onset latency was positively correlated with NEO-PI-R Neuroticism and negatively correlated with Conscientiousness, but was not related to Extraversion, Openness, or Agreeableness. Similarly, the number of minutes to fall asleep was also positively correlated with Impulsiveness and all three subscales of the BIS-11a: Attentional Impulsiveness, Motor Impulsiveness, and Non-Planning Impulsiveness. In contrast, Emotional Control was unrelated to minutes to fall asleep.

Finally, we evaluated the combined contribution of neuroticism, impulsivity, and emotional control to the continuous variable of sleep onset latency. This was accomplished using a multiple linear regression analysis with stepwise entry and deletion, with neuroticism, impulsivity, and emotional control entered as independent variables and sleep onset latency as the dependent variable. In the first step, Neuroticism was entered, which yielded a significant model, $F(1,59)=17.97$, $p<.001$, with Neuroticism accounting for approximately 23% of the variance in sleep onset latency (β=.48, R^2=.23, Adjusted R^2=.22). When Impulsivity was added to the model, the model remained significant F(1, 58)=12.05, p=.030. The combination of Neuroticism (β=.34) and Impulsivity (β=.28) accounted for 29% of the variance in sleep onset latency (R^2=.29, Adjusted R^2=.27) and these two predictors were retained. Emotional Control failed to account for additional unique variance in prediction of sleep onset latency.

Discussion

Consistent with the study hypotheses, we found that individuals reporting sleep onset difficulties demonstrated higher scores on scales measuring neuroticism, impulsivity, and emotional control than those who indicated that they did not experience such difficulties. Moreover, when these traits were assessed in combination, only the trait of impulsivity emerged as uniquely predictive of self-reported problems with sleep initiation. Also consistent with the study hypotheses, our findings showed that longer latency to fall asleep on weekdays and weekends, as assessed via self-report, was associated with higher scores on neuroticism and impulsivity. Overall, these findings suggest that there are specific personality traits that are related to sleep initiation problems, but that the specific association of these traits may be manifest differently depending on how the sleep latency issues are framed.

Our findings contribute to the growing body of literature documenting the association between personality traits and sleep problems [22-25]. Notably, while each of the three personality factors was individually related to self-perceived sleep onset difficulties, our

Table 1: Means and Standard Deviations for Personality Scales.

	Trouble Falling Asleep				
	No		Yes		*p*
	M	*SD*	*M*	*SD*	
NEO-PI-R					
Neuroticism	47.63	9.86	54.81	11.90	.013
Extraversion	55.14	11.25	52.58	10.97	.377
Openness	55.91	9.67	54.31	11.59	.558
Agreeableness	46.86	10.87	47.96	11.34	.701
Conscientiousness	53.60	11.38	45.96	11.97	.014
Emotional Control	46.26	11.51	51.85	8.61	.042
Impulsiveness	57.54	10.0	65.05	11.36	.008

Table 2: Correlations between Self-Reported Sleep Latency and Personality Measures.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Sleep Latency (Minutes)	--										
NEO-PI-R											
2. Neuroticism	.483**	--									
3. Extraversion	.043	-.293*	--								
4. Openness	-.088	-.193	.127	--							
5. Agreeableness	-.026	-.124	.112	.365**	--						
6. Conscientiousness	-.253*	-.093	.098	.119	-.019	--					
Courtauld Emotional Control Scale (CECS)											
7. CECS Total	.189	.319*	-.259*	-.013	.017	-.240	--				
Impulsiveness (BIS-11A)											
8. Total	.450**	.489**	-.079	-.063	-.208	-.593**	.222	--			
9. Attentional	.420**	.517**	-.215	-.021	-.349	-.354**	.242	.705**	--		
10. Motor	.340**	.292	.048	.210	-.071	-.427**	.139	.809**	.375**	--	
11. Nonplanning	.331**	.385**	-.052	-.299*	-.120	-.598**	.163	.851**	.416**	.525**	--

*Significant at *p*<.05
**Significant at *p*<.001

results suggest that they all share considerable variance, which appears to be primarily encompassed within the trait of impulsivity. Not only was impulsivity the only variable to be retained in the equation for predicting subjective difficulties with sleep onset, it was also positively related to longer self-reported sleep latency. Thus, individuals higher in impulsivity were more likely to complain of trouble falling asleep and the magnitude of impulsivity was associated with a longer self-reported latency to fall asleep.

The presently observed relationship between impulsive personality traits and sleep onset delays is generally consistent with a larger literature showing that impulsivity is related to sleep problems [26,27]. Several studies have shown that certain dimensions of impulsiveness, such as urgency and lack of perseverance are related to sleep problems in healthy populations [12,25,28]. Another study found an association between impulsivity and trouble falling asleep; however, this was not significant after controlling for the effects of somatic and psychiatric disorders [11]. The association between impulsivity and prolonged sleep latency may reflect deficits in cognitive control and the ability to inhibit irrelevant cognitive processes [29]. These capacities are among those often associated with the functioning of the prefrontal cortex of the brain [30-32]. In fact, some neuroimaging research suggests that individuals with insomnia show reduced gray matter volume within the orbitofrontal cortex [33], while excessive daytime sleepiness is associated with reduced gray matter in the same general region [34]. Finally, functional neuroimaging evidence suggests that insomnia is associated with reduced medial prefrontal functioning during complex verbal processing [35]. Thus, the prefrontal cortex may be a particularly important target for future study and for developing potential interventions to improve sleep onset problems.

Neuroticism was also individually associated with self-reported difficulties falling asleep. However, when considered in conjunction with the other personality traits, neuroticism failed to contribute to the prediction of sleep problems beyond the variance accounted for by impulsivity. Neuroticism was, however, predictive of sleep onset latency, suggesting that it may relate specifically to rumination and anxious worry, which may be associated with increased arousal and prolonged wakefulness before sleep onset. Interestingly, while we did not predict differences between individuals with and without sleep initiation trouble on other dimensions of the NEO-PI-R, we also found that participants who reported trouble falling asleep also had significantly lower scores on the factor of conscientiousness. While not hypothesized, this finding extends prior work that has shown that individuals scoring lower on the trait of conscientiousness tend to have poorer sleep quality, greater sleep disturbances, and shorter sleep duration [5,8,36]. To our knowledge, the present findings are the first to link lower conscientiousness scores to trouble initiating sleep. However, because this finding was not hypothesized, we did not include it in our regression analyses to discriminate groups or predict sleep latency. This would be a potential target for further research.

Finally, we found that individuals reporting trouble falling asleep scored higher on a measure of excessive emotional control than those denying such problems. This finding is consistent with other studies that have shown a relationship between insomnia severity and self-critical thought suppression prior to sleeping [16,37]. However, when considered in combination with the other personality variables included in the analysis, emotional control did not contribute significantly to the prediction of sleep onset complaints or sleep latency on weekdays or weekends above and beyond the measure of impulsivity. Together, these findings suggest that sleep onset difficulties are associated with considerable worry, rumination, and maladaptive attempts to control emotions, but that this occurs within the context of heightened impulsivity, perhaps reflecting an overarching deficit in the ability to effectively modulate these arousing emotional and cognitive factors.

Our findings may be relevant to conceptualizations of sleep onset problems that focus on the influence of cognitive-emotional processing prior to sleep [22,38]. Recent research suggests that individuals with difficulty falling asleep show increased functional connectivity among sensory and motor regions of the brain, suggesting greater cognitive and behavioral engagement in those without such difficulties [39]. In conceptualizing strategies to address sleep onset problems, it may be useful to consider the maladaptive role of personality factors that may lead to exaggerated cognitive and behavioral engagement, such as neuroticism and excessive emotional control, but particularly impulsiveness. Further research should examine whether interventions that target impulsive cognitions and behavior, negative affect, and maladaptive strategies to control negative emotions might improve sleep latency and perhaps even overall sleep quality.

Our findings should be considered in light of several limitations. First, given our modest sample size, these results should be interpreted cautiously and will require replication in other samples. Further, our study relied on non-standardized self-report measures of sleep onset trouble in a healthy non-clinical sample, rather than in a clinically diagnosed sample with well-characterized insomnia with objective physiological measures of sleep latency such as actigraphy or ambulatory polysomnographic monitoring. Self-reported sleep is often notoriously poorly quantified, so more objective measures should be employed in future work on this topic. It may also be useful to employ standardized measures of sleep problems or clinical insomnia to allow broader comparison of the findings across studies. Finally, the present study was cross-sectional in design, which prevents identification of causal relationships. While our hypotheses were based on the assumption that personality traits may lead to sleep onset difficulties, it is impossible to rule out the alternate explanation that sleep onset difficulties may have led to the impulsive and neurotic patterns of personality traits observed here. By employing longitudinal or developmental studies, future work may be able to disentangle these factors. Nonetheless, with appropriate appreciation of the aforementioned limitations, we believe that the present findings may provide additional insight into the potential contribution of personality traits in sleep onset difficulties.

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RESEARCH ARTICLE

Sleep difficulties are associated with increased symptoms of psychopathology

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Abstract Sleep problems often co-occur with psychopathological conditions and affective dysregulation. Individuals with mood disorders have significantly higher rates of sleep disturbances than healthy individuals, and among those with mood disorders, sleep problems are associated with lower rates of remission and response to treatment. Sleep disruption may itself be a risk factor for various forms of psychopathology, as experimental sleep deprivation has been found to lead to increased affective, cognitive, and somatic symptoms within healthy volunteers. However, little is known about the relationship between recurring sleep complaints in a naturalistic environment and symptoms of psychopathology among healthy individuals. In the present study, 49 healthy adults (21 males and 28 females) reported sleep quality and completed the Personality Assessment Inventory, a standardized self-report assessment of symptoms of psychopathology. Consistent with prior published findings during total sleep deprivation, individuals endorsing self-reported naturally occurring sleep problems showed higher scores on scales measuring somatic complaints, anxiety, and depression. Furthermore, the reported frequency of sleep disturbance was closely linked with the severity of self-reported symptoms. While causal directionality cannot be inferred, these findings support the notion that sleep and emotional functioning are closely linked.

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Introduction

Sleep difficulties are implicated in a wide range of psychopathological conditions, most commonly depression and anxiety, both of which include at least one type of sleep disturbance as a symptom (American Psychiatric Association 2000). Indeed, the prevalence of insomnia is 40 % higher among those diagnosed with mood and anxiety disorders than among healthy individuals (Soehner and Harvey 2012). This is particularly problematic, as comorbid self-reported sleep problems are associated with lower remission rates of depression, poorer response to treatment, and future relapse (Dew et al. 1997; Perlis et al. 1997; Thase et al. 1997).

Sleep problems have long been considered as secondary symptoms of primary mood or anxiety disorders. Under the assumption that the sleep problems were secondary to the primary psychopathology, effective treatment of the mood disturbance was expected to ameliorate the sleep disturbance (Nutt et al. 2008). However, growing evidence suggests that successful treatment of depression does not necessarily alleviate sleep problems, calling into question their status as a secondary symptom versus a primary contributing factor leading to depression (Nierenberg et al. 1999; Thase et al. 2002; Mouchabac et al. 2003; Carney et al. 2007). Conversely, cognitive behavioral treatment for insomnia has been shown to improve clinical outcomes for patients with comorbid depression and to yield higher rates of remission of depression (Edinger et al. 2001; Taylor et al. 2007; Manber et al. 2008). Similar findings have been reported for the relationship between sleep and anxiety disorders (Gillin

1998; Monti and Monti 2000; Papadimitriou and Linkowski 2005; Mellman 2006; Ramsawh et al. 2009). Sleep disturbance appears to exacerbate psychopathological symptoms, with higher frequencies of insomnia correlating with greater severity of anxiety and depression (Taylor et al. 2005). Together, these findings suggest that sleep disruption may not only be a symptom of some forms of psychopathology, but rather that sleep disturbance itself may also be an underlying risk factor for developing an affective disorder (Ford and Kamerow 1989; Taylor et al. 2003; Perlis et al. 2006; Neckelmann et al. 2007). Such a possibility opens new avenues for treatment of these forms of psychopathology.

The potential causal role of sleep loss in the manifestation of psychopathology was demonstrated in a laboratory study of healthy participants (Kahn-Greene et al. 2007). After two nights of total sleep deprivation under controlled laboratory conditions, participants reported significant, though sub-clinical, increases in several facets of psychopathology relative to baseline, including increased symptoms of somatic complaints, anxiety, depression, and paranoia. These increased psychopathological symptoms primarily involved affect dysregulation, whereas there was no effect of sleep loss on symptoms of thought disorder or psychotic thinking. Another report by the same group showed that emotional intelligence and constructive thinking skills tend to decline significantly following total sleep deprivation (Killgore et al. 2007). The authors interpreted their findings in light of neuroimaging data showing that sleep loss leads to a reduction in metabolic activity within the prefrontal cortex (Thomas et al. 2000), a region of the brain that is involved in emotion regulation (Drevets et al. 1998; Videbech 2000). Subsequent work demonstrated that gray matter volume within the medial prefrontal cortex (mPFC) was linearly related to the typical amount of sleep obtained relative to subjective nightly requirements for normal functioning (Weber et al. 2013); additionally, the volume of gray matter within the mPFC was inversely related to symptoms of psychopathology. The role of the prefrontal cortex in regulating affective responses was further demonstrated in a neuroimaging study that showed that sleep deprivation may alter normal functional connectivity between the emotional regulating regions of the prefrontal cortex and the emotionally responsive amygdala (Yoo et al. 2007). These study findings suggest that sleep deprivation leads to altered mood and impaired emotion regulation capacities, potentially due to altered prefrontal regulation of limbic emotion regions (Yoo et al. 2007). Such affective dysregulation could contribute to the manifestation of some affective disorders and other psychopathological conditions.

Outside of the laboratory setting, chronic sleep restriction or insufficient sleep can occur for any number of reasons, including lifestyle choices, work demands, environmental issues, or medical and psychiatric conditions. Additionally, poor sleep may be a primary condition in and of itself. Some individuals have difficulty initiating sleep (i.e., sleep onset insomnia), while others fall asleep normally but have difficulty remaining asleep long enough to feel rested (i.e., sleep maintenance insomnia) (Riemann et al. 2010). Although laboratory sleep deprivation has been shown to lead to increased symptoms of psychopathology (Kahn-Greene et al. 2007), it is not clear whether self-reported sleep onset or maintenance problems at home might also be associated with elevations of affective complaints in an otherwise healthy population. Therefore, the primary goal of the present investigation was to extend prior laboratory research showing an association between sleep deprivation and psychopathology to a non-laboratory naturalistic setting. In the present study, we queried a sample of generally healthy individuals about their normal sleep quality and quantity and asked them to complete a standardized measure of psychopathological symptom severity. Based on the outcome of prior laboratory research (Kahn-Greene et al. 2007), we hypothesized that participants complaining of difficulties with sleep initiation or sleep maintenance would also score higher on measures of symptoms of psychopathology, including depression, anxiety, somatic complaints, and paranoia.

Methods

Subjects

Sixty-five healthy adults (33 males and 32 females) were recruited from the Greater Boston area through internet advertisements and flyers. Some participants were excluded due to incomplete and inconsistent data, or validity indices on the measure of psychopathology exceeding two standard deviations above the reference sample (indicating an invalid response set), yielding a final sample size of 49 complete and valid datasets (21 males and 28 females). Included participants identified themselves as Caucasian (65.3 %), African American (16.3 %), Asian (12.2 %), or Other (6.1 %). The final sample ranged in age from 18 to 45 ($M = 30.0$, $SD = 7.8$) and had an average of 15.3 years of education (range 11–20, $SD = 2.0$).

All participants were native English speakers, with no reported history of medical, neurological, or psychiatric problems. Individuals were excluded if they reported significant substance abuse or any use of cannabis in the past year, or obtained a score of 15 or higher on the Beck Depression Inventory. All participants provided written informed consent and were compensated for their time. The McLean Hospital Institutional Review Board and the US Army Human Research Protection Office approved the study protocol.

Materials

To measure sleep quality and quantity, participants completed a brief open-ended questionnaire regarding their sleep habits, including whether (i.e., yes/no) and how often (i.e., frequency per week, month, or year) they experienced trouble either falling and/or staying asleep. The frequencies of sleep problems were then transformed across participants to reflect the estimated yearly frequency of sleep disturbance. Participants were grouped based on whether or not they endorsed any sleep difficulties (i.e., yes/no), regardless of the frequency.

The Personality Assessment Inventory (PAI) was administered as an objective measure of psychopathological complaints. The PAI is composed of 344 statements, each read by the participant and rated along the following 4-point Likert scale: "False, not at all true", "Slightly True", "Mainly True", or "Very True." This study utilized only the 11 clinical scales of the PAI to assess symptoms of psychopathology. These scales include: Somatic Complaints (SOM), which assesses preoccupation with physical health and somatic concerns; Anxiety (ANX), which measures negative affect and physical signs of stress; Anxiety Related Disorders (ARD), which measures behavioral aspects of specific anxiety disorders; Depression (DEP), which assesses various symptoms of depression such as pessimism and physical features such as lack of energy; Mania (MAN), which measures signs of mania and hypomania; Paranoia (PAR), which measures characteristics of paranoia such as hypervigilance and persecution; Schizophrenia (SCZ), which assesses a range of symptoms including unusual beliefs, poor social skills, and problems with attention or concentration; Borderline Features (BOR), which addresses problems with adjustment or personal organization; Antisocial Features (ANT), which assesses personality and behavioral aspects of psychopathy; Alcohol Problems (ALC), which targets consequences and behaviors related to alcohol use and dependence; and Drug Problems (DRG), which assesses consequences and behaviors related to drug use and dependence. Each clinical scale has diagnostic relevance as well as high test–retest reliability between 0.85 and 0.94 (Morey 1991). Raw scores for each PAI scale were converted to normalized *T* scores (M = 50, SD = 10) based on a standardization sample of 1,000 adults, as published in the manual for the inventory. A *T* score of 70 or above on any of the clinical scales is commonly accepted as reflecting a clinically significant elevation. Based on prior reported findings in sleep-deprived subjects (Kahn-Greene et al. 2007), our hypotheses focused on 4 of 11 clinical scales, including SOM, ANX, DEP, and PAR. In addition to the clinical scales, four additional validity scales were used to screen the sample for consistency, careless responses, malingering, and response bias. Individuals who obtained a *T* score of 70 or greater on any of the validity scales were excluded from the analysis.

Basic demographic data were also collected, including age, race, gender, and education. Additionally, as financial concerns and environmental influences could potentially play a role in depression and anxiety (Green and Benzeval 2013; Lorant et al. 2003; Murphy et al. 1991), information regarding general neighborhood income and poverty level was also examined. To account for a potential effect of socioeconomic status on measures of psychopathology, data were obtained on mean inflation-adjusted 12-month household income and the percentage below the poverty line of the participant's neighborhood based on their home address (US Census Bureau 2010).

Analysis

All analyses were conducted in IBM SPSS Statistics for Macintosh version 20 (IBM Corp., Armonk, NY, USA). Scores on the PAI clinical scales did not conform to a normal distribution, so a ranked order ANCOVA was conducted to examine differences in scores on the clinical scales between individuals who endorsed trouble sleeping ($n = 22$) and those who did not ($n = 27$), with median estimated household income as a covariate. Following the process described by LaVange and Koch (2006), ranks were computed for each of the PAI scales and for median household income, without regard for groups and using midranks in the case of ties. Using linear regression, ranks of each PAI scale were regressed onto median household income and the residuals were saved. For each scale, the residuals were entered into a Spearman correlation with group (trouble sleeping vs. no trouble sleeping) as the other variable. For the four clinical scales with a priori hypotheses derived from the literature, no correction was made for post hoc multiple comparisons. In contrast, for the remaining seven clinical scales that were not hypothesized to show differences, α levels were adjusted by applying a Bonferroni corrected α-criterion of 0.007 (i.e., 0.05/7). As a secondary analysis, for those individuals who reported having trouble sleeping, Bonferroni corrected (α-criterion of $0.05/11 = 0.004$) Spearman rank-order correlations were conducted for all 11 PAI clinical scales to examine the association between the annual frequency of sleep complaints and scores on each PAI clinical scale.

Results

Based on responses to the sleep questionnaire, 22 participants reported at least one sleep problem (i.e., trouble falling asleep or trouble staying asleep), while 27 participants denied experiencing either of those problems. Among those individuals reporting trouble sleeping, the frequency of sleep complaints ranged from 2 to 156 times per year, with

Table 1 Demographic variables

Characteristics	No trouble sleeping ($n = 27$)	Trouble sleeping ($n = 22$)	F value	Pearson χ^2	p value
Age (years)	29.70 (SD = 7.8)	30.45 (SD = 8.0)	0.11		0.74
Sex				0.06	0.80
Male	12	9			
Female	15	13			
Race				2.64	0.62
Caucasian	17	15			
African American	6	2			
Asian	3	3			
Hispanic	0	0			
Other	1	2			
Years of education	14.8 (SD = 1.7)	15.9 (SD = 2.2)	3.71		0.06
% Below poverty line	15.0 (SD = 12.4)	9.8 (SD = 9.6)	2.58		0.12
Median household income	$57.5 K (25th ‰–75th ‰: $44 K–$75 K)	$74.1 K (25th ‰–75th ‰: $60.5 K–$93 K)	Mann–Whitney $U = 179.5$		0.02*

* Comparison is significant at the 0.05 level (two-tailed)

a median of 52 times per year (1st quartile = 24, 3rd quartile = 82). In comparing groups endorsing or denying problems falling and/or staying asleep, no significant difference was observed for age, sex, race, or education. The median neighborhood household income was found to be significantly different between the two groups (Mann–Whitney $U = 179.5$, $p = 0.02$) and was therefore used as a covariate in further analyses. Table 1 presents the demographic data for this sample.

In evaluating our primary hypothesis using a ranked ANCOVA, there were significant differences between the two groups (sleep problems vs. no sleep problems) on three of the four hypothesized clinical scales. Specifically, after adjusting for median neighborhood household income, individuals who reported having trouble sleeping scored higher than those with no sleep problems on the following PAI scales: SOM (Spearman's $r_s = 0.284$, $p = 0.048$), ANX (Spearman's $r_s = 0.377$, $p = 0.008$), and DEP (Spearman's $r_s = 0.325$, $p = 0.023$). In contrast, there was no significant relationship between trouble sleeping and the PAR scale after adjusting for median household income (Spearman's $r_s = 0.136$, $p = 0.350$). While none of the remaining clinical scales were significantly associated with sleep problems after adjusting for median household income when the Bonferroni corrected p value of $p = 0.007$ was used, ARD was significant at a trend level (Spearman's $r_s = 0.337$, $p = 0.018$), when using the Bonferroni corrected α-criterion (see Table 2).

Although the scores of a few individuals fell just outside of the normal range for clinical scores on the PAI, mean T scores for all scales were below 70, indicating that, on the whole, symptoms of psychopathology observed in this sample were in the subclinical range. However, because the present study only focused on a nonclinical population, it was also of interest to determine whether extreme scores in the clinical range might have affected the results. Therefore, the preceding analyses were run a second time while excluding all individuals with any T scores above 70 ($n = 11$ excluded). Overall, the results of the more conservative analysis are similar to the total sample analysis and confirm our main findings. These findings are summarized in Table 3.

In addition to our primary hypothesis, for those individuals who reported having trouble falling asleep, Bonferroni corrected Spearman rank-order correlations were computed

Table 2 Rank order ANCOVA: comparison between sleep difficulty groups

Clinical scale	Spearman's r_s	p value
Scales of a priori interest		
Somatic complaints	0.284	0.048*
Anxiety	0.377	0.008**
Depression	0.325	0.023*
Paranoia	0.136	0.350
Other PAI clinical scales[a]		
Anxiety-related dis.	0.337	0.018
Mania	−0.122	0.404
Schizophrenia	0.290	0.043
Borderline features	0.250	0.084
Antisocial features	0.055	0.707
Alcohol problems	0.015	0.921
Drug problems	0.165	0.256

* Comparison is significant at the 0.05 level (two-tailed)

** Comparison is significant at the 0.01 level (two-tailed)

[a] Bonferroni correction was applied to the α value to determine significance

Table 3 Rank order ANCOVA: comparison between sleep difficulty groups, excluding individuals with *T* scores above 70 T on any PAI scale

Clinical scale	Spearman's r_s	*p* value
Scales of a priori interest		
Somatic complaints	0.308	0.060
Anxiety	0.471	0.003**
Depression	0.457	0.004**
Paranoia	0.384	0.017*
Other PAI clinical scales[a]		
Anxiety-related dis.	0.409	0.011
Mania	0.168	0.313
Schizophrenia	0.360	0.026
Borderline features	0.384	0.017
Antisocial features	0.437	0.006*
Alcohol problems	0.173	0.299
Drug problems	0.178	0.285

* Comparison is significant at the 0.05 level (two-tailed)

** Comparison is significant at the 0.01 level (two-tailed)

[a] Bonferroni correction was applied to the α value to determine significance

Table 4 Spearman rank-order correlations between the frequency of sleep disturbances and PAI symptoms among individuals endorsing sleep problems ($n = 22$)

PAI clinical scale	Spearman's r_s	*p* value
Somatic complaints	−0.109	0.647
Anxiety	0.300	0.199
Anxiety-related dis.	0.291	0.214
Depression	0.521	0.018
Mania	0.192	0.417
Paranoia	0.200	0.397
Schizophrenia	0.412	0.071
Borderline features	0.319	0.170
Antisocial features	0.090	0.705
Alcohol problems	0.107	0.652
Drug problems	0.142	0.550

None are significant after Bonferroni correction is applied to the alpha value

between the reported frequency of sleep disturbance and scores on all PAI clinical scales. While none of the scales achieved statistical significance when the Bonferroni corrected *p* value of $p = 0.004$ was used, the DEP scale trended significance (Spearman's $r_s = 0.521$, $p = 0.018$) and the SCZ clinical scale approached a trend correlation (Spearman's $r_s = 0.412$, $p = 0.071$) between the frequency of reported sleep disturbances and severity of psychopathological symptoms (see Table 4).

Discussion

In the present study, we examined the differences in symptoms of psychopathology between generally healthy individuals endorsing or denying self-reported sleep problems. Based on prior research on the effects of experimentally induced sleep deprivation on psychopathological symptoms (Kahn-Greene et al. 2007; Killgore et al. 2007), we hypothesized that sleep-related complaints would be associated with higher scores on several dimensions of psychopathology, specifically SOM, ANX, DEP, and PAR. Indeed, we found that sleep problems (i.e., trouble falling or staying asleep) were associated with greater symptoms of three of the four hypothesizes scales of psychopathology on a standardized self-report instrument, including more severe complaints of depression, anxiety, and somatic problems. Contrary to the prior laboratory findings, there was no association between sleep problems and symptoms of paranoia. In contrast, no group differences were found on the remaining scales (i.e., ARD, MAN, SCZ, BOR, ANT, DRG or ALC), which is consistent with the prior laboratory findings as well. Overall, these findings suggest that sleep complaints involving difficulty with sleep onset or sleep maintenance, even within a nonclinical sample in their home environment, are associated with a subclinical elevation of specific cognitive and affective features often associated with psychopathology.

Further analyses also suggest the possibility that there may be a positive association between the frequency of sleep disturbances and the severity of some psychopathological symptoms. Repeated sleep disturbance may result in the accumulation of "sleep debt", which has been demonstrated to accrue over sessions of laboratory sleep deprivation. Such cumulative sleep debt is linked with steady declines in cognitive performance and emotion processing (Van Dongen et al. 2003; Killgore 2010; Durmer and Dinges 2005). Increased reports of trouble sleeping approached trend level of association with increased scores on the PAI scale of DEP, consistent with previous research showing that individuals with worse sleep endorsed greater depression (Taylor et al. 2005). In the laboratory, studies of chronic partial sleep restriction, a situation most analogous to naturalistic sleep difficulties, have reported cumulative declines in neurobehavioral functions over time (Durmer and Dinges 2005). In the present sample, the negative consequences of sleep disruption may have accrued over time for individuals who reported higher frequencies of sleep problems and contributed to greater severity of DEP symptoms. Additionally, greater sleep disruption showed a nonsignificant trend association with increased severity of scores on the PAI SCZ scale, indicating greater difficulty concentrating, disordered cognition, worsening of social interactions, and unusual perceptions. These findings

support prior work demonstrating worsening of cognitive functioning (Killgore 2010) as well as disrupted emotional processing (Killgore et al. 2008; Tempesta et al. 2010) as a result of sleep deprivation, which may be captured by the various components of the PAI SCZ scale. It has also been demonstrated that sleep deprivation results in the attenuation of visual perception (Kendall et al. 2006; Roge and Gabaude 2009), hypothesized to be the result of deactivation within the occipital cortex due to an overall decline in top–down cognitive control (Chee et al. 2008; Chee and Tan 2010). Other studies have indicated similar declines in auditory (Babkoff et al. 2005), tactile (Haack et al. 2009), and olfactory perception (Killgore and McBride 2006), all of which combined may contribute to increases in unusual perceptions as captured in the SCZ scale of the PAI.

The present results are consistent with our initial predictions that individuals who report experiencing sleep disturbances will score higher on measures of affective psychopathology. However, in contrast to findings from experimental sleep deprivation (Kahn-Greene et al. 2007), the current study did not demonstrate any significant increase in feelings of persecution associated with disrupted sleep. One possible interpretation of this null finding is that the PAR symptoms previously observed may have been an epiphenomenon of the enforced sleep deprivation conditions within the laboratory setting. In such a controlled laboratory context, feelings of persecution or suspicious attitudes may not be fully unjustified, due to the reality that participants are in fact being observed and periodically subjected to mildly stressful tasks. The present findings extend the prior work on laboratory sleep deprivation suggesting that sleep plays an important role in healthy emotional functioning, and further reinforce the notion that the cognitive and affective problems associated with sleep onset or maintenance insomnia in the home environment are not completely analogous to those seen during laboratory sleep deprivation.

Because of the correlational nature of these findings, it is impossible to infer directional causality of the effects. The commonly accepted position is that primary psychopathology leads to secondary effects on sleep quality (Nutt et al. 2008). However, when the present findings are interpreted in light of the prior laboratory results, it is also possible that insufficient sleep, particularly over prolonged periods, may contribute to the development of various forms of psychopathology or an increase in symptom expression. While the present data cannot unambiguously answer such questions, they do build upon prior experimental findings to suggest some clear relationships between sleep quality and several dimensions of psychopathology. These findings are also consistent with the general prefrontal model of affective dysregulation in depression and sleep loss. Functional neuroimaging studies have shown decreased regional glucose metabolic rates in the prefrontal cortex and parietal regions (Thomas et al. 2000) after continuous periods of sleep deprivation. Because of the prominent role of these regions in cognitive control, affective regulation, and higher order functioning, these reductions in metabolic activity during sleep loss have been proposed as leading to deficits in a number of cognitive and emotional capacities (Horne 1988; Harrison and Horne 2000; Killgore 2010). Furthermore, sleep deprivation appears to be associated with reduced prefrontal activation and heightened amygdala responsiveness to emotionally salient stimuli (Yoo et al. 2007). Under such circumstances, the limbic system may be sensitized by sleep deprivation. Left relatively unchecked by a hypoactive prefrontal cortex, this situation could predispose an individual toward exaggerated emotional responses. This same pattern is consistent with several models of psychopathology. For instance, neuroimaging studies of affective disorders have often shown decreased activation in the prefrontal cortex along with increased limbic-paralimbic activation, especially in depression and anxiety (Mayberg et al. 1999; Drevets 2001). Other studies have shown increased glucose metabolism in the amygdala among depressed individuals (Drevets et al. 2008). The ventromedial prefrontal cortex, in particular, is frequently associated with altered functioning during sleep deprivation (Libedinsky et al. 2011; Venkatraman et al. 2011) and shows altered structural volume in individuals with excessive daytime sleepiness (Killgore et al. 2012). This same region also shows altered functioning with increased dysphoric mood (Killgore and Yurgelun-Todd 2006) and depression (Drevets 2007; Sheline et al. 2009). The congruence between the sleep deprivation and affective disorders in cognitive, affective, and physiological brain responses suggests that sleep loss may serve as a potential model for the neurobiological basis of affective disorders.

When interpreting these data, several methodological limitations should be kept in mind. It is important to note that the observed PAI scores were well within the normal range for the vast majority of the sample, indicating that increased symptoms of psychopathology for those individuals reporting trouble sleeping did not reach clinically meaningful levels. Consequently, the findings reported here should be considered as most relevant to understanding the cognitive and affective systems affected by sleep disruption rather than as evidence of the contribution of sleep problems to clinical presentations. Even with severe sleep disruptions, most individuals are unlikely to show clinically impairing elevations on scales of psychopathology. Another point to keep in mind is the intercorrelated nature of the PAI clinical scales. While it might not be possible to distinguish the relationship between sleep disruption and each of the scales independently, this may best serve as a model of affective psychopathologies, which are often highly comorbid in a clinical population. As a further

limitation, the current study included a relatively modest sample size, and further replication will be necessary to determine the reliability of the observed effects. Additionally, it should be noted that all indicators of sleep problems as well as psychopathological symptom complaints were collected from self-report measures. Such subjective measures can be inherently biased and may not necessarily reflect objective measures of psychological or sleep functioning. Along this line, it is possible that individuals with high levels of psychopathology may misinterpret or misperceive the severity of their sleep difficulties. Therefore, future studies would benefit from objective measures of sleep and sleep disturbances, such as wrist actigraphy monitors or ambulatory electroencephalographic monitoring, as well as assessments for symptoms of psychopathology garnered from clinical interviews. Most importantly, the current findings cannot establish causality between sleep problems and psychopathology. Longitudinal research tracking sleep disturbances over time would be best suited to capture the onset of psychopathology in individuals across different histories of sleep patterns. However, the current findings are consistent with prior work in laboratory-induced acute sleep deprivation, lending support to the notion that disrupted sleep could contribute to psychopathology. With these limitations in mind, the present findings indicate that trouble initiating or maintaining sleep may be associated with a subclinical increase in several dimensions of psychopathology, particularly those involving affect regulation. These findings suggest that difficulties initiating or maintaining sleep are closely related to emotional and cognitive functioning. Treatment of emerging or comorbid sleep problems may be a useful intervention in the management, and possibly prevention, of some forms of clinical psychopathology.

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Trait emotional suppression is associated with increased activation of the rostral anterior cingulate cortex in response to masked angry faces

Jiaolong Cui, Elizabeth A. Olson, Mareen Weber, Zachary J. Schwab, Isabelle M. Rosso, Scott L. Rauch and William D.S. Killgore

Emotional suppression (ES) is a critical component of the ability to self-regulate emotion. However, people who chronically use ES as a primary strategy often experience heightened anxiety or depression. Although functional neuroimaging studies have extensively mapped the brain regions involving in emotional regulation, the neural substrates of ES as a trait construct remain relatively unexplored. Using a validated backward masked facial affect paradigm, we examined the association between ES and functional brain responses to masked angry, fearful, and happy faces. Healthy adults underwent functional MRI and completed the Courtauld Emotional Control Scale as a measure of ES. Correlations between self-reported ES and brain responses to the facial affect stimuli (affective > neutral) were evaluated within the brain regions involved in emotional processing, including the amygdala, insula, anterior cingulate cortex, medial prefrontal cortex, and orbitofrontal cortex. In response to angry faces, higher trait tendency to suppress anger and anxiety was significantly correlated with increased activation within the rostral anterior cingulate cortex, whereas no correlation was observed for masked happy or fearful faces. This finding suggests that the rostral anterior cingulate cortex contributes to the unconscious suppression of emotional responses to angry facial affect and may play a role in the mediating anatomy of trait ES. *NeuroReport* 25:771–776 © 2014 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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Introduction

Emotional suppression (ES) can be defined as an attempt to control the expression of negative affect through voluntary downregulation of emotional experience [1]. ES may provide short-term relief from unpleasant negative emotions and produce a sense of well-being in the short term [2]. However, using ES as a chronic strategy for regulating emotions can have damaging effects on health in the longer term. For example, chronic inhibition of anger has been associated with hypertension or the development of coronary heart disease, and it may even be linked to cancer onset and progression [3,4]. People who engage in chronic ES also tend to experience heightened anxiety or depression [1]. Clearly, this trait-like tendency to regulate emotional experience through suppressive mechanisms is an important factor in mental and physical health.

The neurobiology underlying emotional processing and its regulation has become a focus of much research in recent years. In healthy humans, neuroimaging studies have made significant progress in mapping the systems involved in emotional experience and emotional information processing. The limbic system, including the amygdala, is critical to emotional learning and mediates the rapid formation of associations between encountered stimuli and aversive experiences, whereas the prefrontal cortex (PFC) has a primary role in the regulation of emotion (upward and/or downward) [5,6]. In particular, voluntary regulation of emotion is associated with increased activity in the PFC and anterior cingulate cortex (ACC) [7].

Although ES appears to play a significant role in mental and physical health, the association between ES and brain responses to negative displays of facial affect has remained virtually unexplored. Facial expression is perhaps the most important nonverbal channel for conveying social and emotional information between humans [8]. By attending to facial expressions, an individual is able to monitor the emotional and motivational states of others and glean important cues that regulate social interaction. Moreover, subtle facial affective cues can be perceived, registered, and responded to even when they are presented below the threshold of conscious visual perception [9]. In this study, healthy participants underwent functional MRI (fMRI) while we used a backward masking technique that presents affective facial stimuli so rapidly that they are not typically consciously perceived. We examined the association between self-reported ES

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scores and brain activation in response to backward masked facial expressions of angry, fearful, and happy affects [9]. Further, because individual differences in ES may partially reflect baseline levels of emotional intelligence (EI) and experiences with social interactions [10], participants' EI scores were also measured and included as nuisance covariates for statistical control. On the basis of prior research [11], several key brain regions of emotional processing were investigated, including the amygdala, insula, ACC, medial PFC (mPFC), and orbitofrontal cortex. We hypothesized that individuals with higher ES would show increased activation within prefrontal and ACC regions and reduced limbic and paralimbic activation when viewing the negative affective faces (angry or fearful faces), but no increased brain responses to happy faces.

Methods

Participants

Sixty-three right-handed healthy native English-speaking adults (mean age 30.3 ± 8.1 years, range 18–45 years; 33 men, 30 women) were recruited from the Boston metropolitan area and underwent functional neuroimaging. The exclusionary criteria included any significant history of medical, neurological, or psychiatric disorders. All participants provided written informed consent before participation and were compensated for their time. This research protocol was reviewed and approved by the Institutional Review Board of McLean Hospital and the U.S. Army Human Research Protection Office.

Materials and procedure

Each participant completed the Courtauld Emotional Control Scale (CECS) [12], a self-report measure of emotional regulation in response to particular negative emotional experiences. It comprises 21 items including three subscales measuring the tendency to suppress or express feelings of anger, anxiety, and depression. Participants were asked to respond to phrases such as 'When I feel angry…', 'When I feel anxious (worried)…', or 'When I feel unhappy (miserable)…' with statements such as 'I keep quiet', 'I bottle it up', 'I tell others about it', or 'I let others see how I feel'. They indicated the frequency of such feelings by rating them on a four-point scale, ranging from 1 = 'almost never' to 4 = 'almost always' with regard to the extent to which they attempted to control emotional expression. Higher scores indicated a greater extent of ES. To assess the participants' trait and ability EI, each was also asked to complete two well-validated commercially available tests: the Bar-On Emotional Quotient Inventory [13] and the Mayer–Salovey–Caruso Emotional Intelligence Test [14].

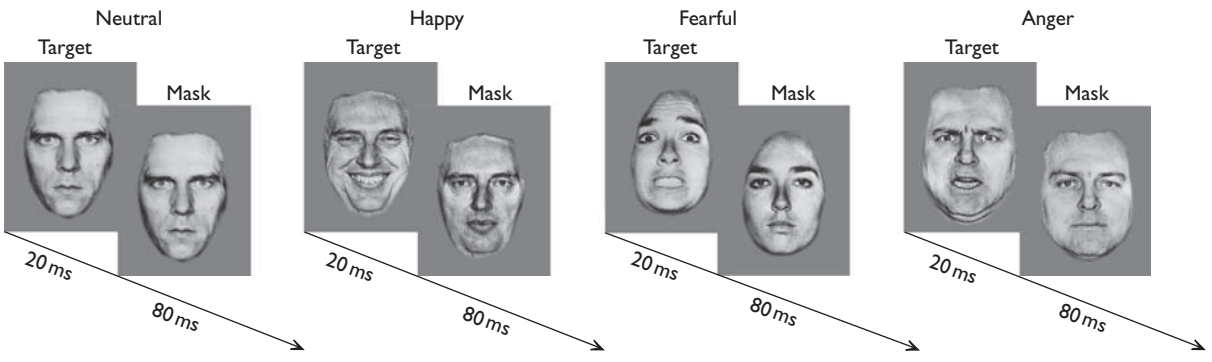
Stimulation paradigms

During fMRI, each participant participated in three task runs that involved masked presentations of angry, fearful, and happy affective faces in a counterbalanced order. The paradigm is similar to that reported in our previous publications [15–17]. Each scanning run lasted for 3 min and was presented in five 30-s blocks that alternated between masked neutral and masked affect conditions. Each block consisted of 20 1.5-s trials. Each trial included a facial expression 'target' image presented for 20 ms, followed immediately by a neutral facial expression 'mask' for 80 ms (Fig. 1) and a 1400-ms period of blank screen. The stimulus in each target–mask pair was posed by the same actor [18]. Masked neutral trials (N) presented a neutral target followed by a neutral mask, whereas affective trials (A) presented an affective target followed by a neutral mask. Each run was bound at the beginning and end by a 15-s fixation cross (+) and was presented in the following order (+ , N, A, N, A, N, +).

MRI parameters

Scans were obtained using a 3.0 T Siemens Tim Trio scanner (Siemens, Erlangen, Germany) and a 12-channel head coil. Structural T1-weighted three-dimensional

Fig. 1



Each masked stimulus trial consisted of two rapidly presented stimuli: a 'target' face depicting angry, fearful, happy, or neutral affects presented for 20 ms, followed by a 'mask' face of the same poser expressing a neutral emotion for 80 ms.

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magnetization-prepared rapid acquisition with gradient echo images were collected (TR/TE/flip angle = 2.1 s/2.25 ms/12°) over 128 sagittal slices (256 × 256 matrix) with a slice thickness of 1.33 mm (voxel size = 1 × 1 × 1.33 mm). T2*-weighed fMRI scans were obtained over 43 transverse slices (3.5 mm thickness, no gap) using an interleaved sequence (TR/TE/flip angle = 3.0 s/30 ms/90°), with 60 images collected per slice. Data were collected with a 22.4 cm field of view, a 64 × 64 acquisition matrix, and a voxel size of 3.5 × 3.5 × 3.5 mm.

Image processing

Data were preprocessed and analyzed using SPM8 (Wellcome Department of Cognitive Neurology, London, UK; *http://www.fil.ion.ucl.ac.uk/spm*). Raw images were realigned to the first image in the series, unwarped, registered to each participant's high-resolution anatomical image, and normalized to the three-dimensional space of the Montreal Neurological Institute (MNI). Data were spatially smoothed with an isotropic Gaussian kernel (full width half maximum = 6 mm) and resliced to a voxel size of 2 × 2 × 2 mm using 4th Degree B-Spline interpolations. The time series data were convolved with the SPM8 canonical hemodynamic response function; the AR(1) option was used to correct for serial autocorrelation, and a 128-s high-pass filter was used to remove low-frequency confounds. The Artifact Detection Tool (*http://www.nitrc.org/projects/artifact_detect/*) implemented in the SPM8 toolbox was used for comprehensive analysis of artifacts in time series data including spiking and motion. Scan volumes exceeding 3 SD in mean global intensity or scan-to-scan motion that exceeded 1.0 mm and the first volume of each run were regressed out of the first-level analysis as nuisance covariates.

Statistical analysis

Within SPM8, a series of general linear models was created for the masked affective and neutral face conditions against the resting fixation baseline. Contrast images evaluating within-subject effects of each masked affect versus masked neutral baseline were then created. In a second-level random effects regression model, these contrast images were correlated with CECS scores, while statistically controlling for age, sex, and EI (subscale scores of the Emotional Quotient Inventory and Mayer–Salovey–Caruso Emotional Intelligence Test). Separate models were created for masked angry, fearful, and happy conditions. On the basis of our a-priori hypotheses derived from previous research [11], regions of interest (ROIs) were restricted to the bilateral amygdala, insula, ACC, mPFC, and orbitofrontal cortex, defined by the Automated Anatomical Labeling Atlas as implemented in the Wake Forest University PickAtlas Utility. Correlations were thresholded within ROIs at P less than 0.05, voxelwise false discovery rate (FDR) corrected, k (extent) 20 or higher contiguous voxels.

Table 1 Mean, SD, and range of emotional control of participants (n = 63)

CECS	Mean	SD	Range
Anger	16.35	3.76	9–25
Anxiety	16.30	4.59	7–26
Depression	16.16	4.08	8–26

CECS, Courtauld Emotional Control Scale.

Results

The detailed CECS scores of the participants (n = 63) are summarized in Table 1. In response to masked angry faces, a cluster of activation located within the rostral ACC (rACC) was correlated with a higher CECS Anger Score (43 voxels; T = 5.5; MNI coordinates: x = −4, y = 36, z = 10; P = 0.002, FDR corrected). A cluster of activation within the rACC was also positively correlated with the CECS Anxiety Score (214 voxels; T = 5.67; MNI coordinates: x = −4, y = 36, z = 10; P = 0.001, FDR corrected) in response to angry faces (Fig. 2). For masked fearful and happy faces, no significant correlations were observed within any of the ROIs.

In addition, to verify that the specificity of the results was not simply driven by the greater range of BOLD activation responses within the masked anger condition relative to the other conditions, we extracted and plotted the mean and SD of the functional responses from each ROI, including the original cluster of activation in the rACC defined by the significant correlation with CECS Anger Scores above. As shown in Fig. 3, the range of BOLD responses, particularly within the ACC, was similar across all three conditions, suggesting that the specificity of our findings to masked anger cannot be explained by the restricted range for the other two conditions.

Discussion

We examined the association between self-reported ES scores and brain responses to images of facial affects presented using a backward masking technique, so as to probe relatively automatic (rather than conscious) information processing. Greater ES of anger and anxiety was significantly correlated with increased activation within the rACC in response to masked angry faces, but not to faces expressing fear or happiness. This finding supports the primary hypothesis and is consistent with the growing literature suggesting that rACC plays a key role in regulation of emotional responses to negative stimuli in healthy individuals [19–22]. Previous research has shown that rACC function is reduced in patients with post-traumatic stress disorder during negative emotional regulation, and that the magnitude of activation of the rACC is negatively correlated with post-traumatic stress disorder symptom severity [6]. Such findings suggest that abnormal functioning of the rACC may impair the ability to downregulate negative emotion. Phan *et al.* [11] showed that greater dorsal ACC responses were also

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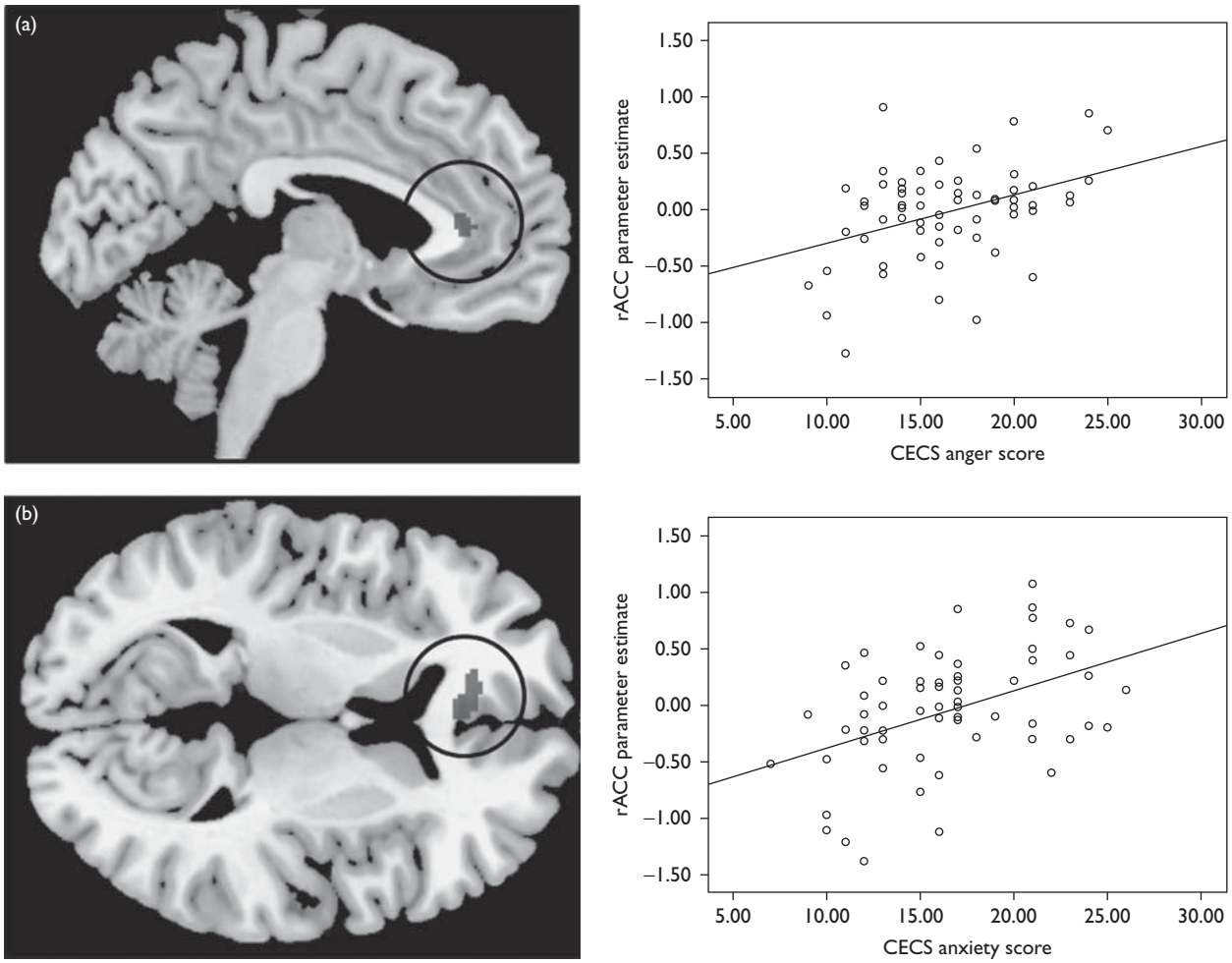
Fig. 2



Brain responses to a masked angry face within the rACC (MNI: $x=-4$, $y=36$, $z=10$) were correlated with the (a) CECS anger score and the (b) CECS anxiety score. Scatter plots show the pattern of association between the CECS score and the first extracted cluster eigen variate. CECS, Courtauld Emotional Control Scale; MNI, Montreal Neurological Institute; rACC, rostral anterior cingulate cortex.

associated with inhibition of negative affect. However, their participants were instructed to perform an emotional regulation protocol voluntarily using deliberate cognitive strategies. Meta-analysis of brain-mapping studies on cognitive and emotional tasks strongly supports the functional delineation of ACC into dorsal–caudal cognitive and ventral–rostral affective subdivisions [21]. Considerable evidence suggests that the dorsal–caudal cognitive subdivision is implicated in modulation of attention, monitoring competition, complex motor control, motivation, novelty, error detection, working memory, and anticipation of cognitively demanding tasks. The ventral–rostral affective subdivision is primarily involved in assessing the salience of emotional and motivational information and in the regulation of emotional responses. Our present data showed the activation just at the cusp of these two subdivisions, suggesting that a greater tendency to routinely suppress anger and anxiety is associated with increased responsiveness of this intermediate zone of the rACC to backward masked angry facial expressions.

In the current investigation, we used backward masked stimuli during fMRI to demonstrate the role of rACC in automatic emotional processing of facial affects. In the present paradigm, a 'target' stimulus was presented very quickly, and immediately replaced by an alternate 'mask' stimulus for a slightly longer duration [9]. This paradigm minimizes or prevents conscious perception of the target affective stimulus. Advantages of using this backward masked paradigm include avoidance of extraneous cognitive processing during the scan and evocation of reliable patterns of activation within affect processing regions during functional neuroimaging [15–17]. Prior work with this paradigm has shown that masked affect perception is commonly associated with greater activation within the

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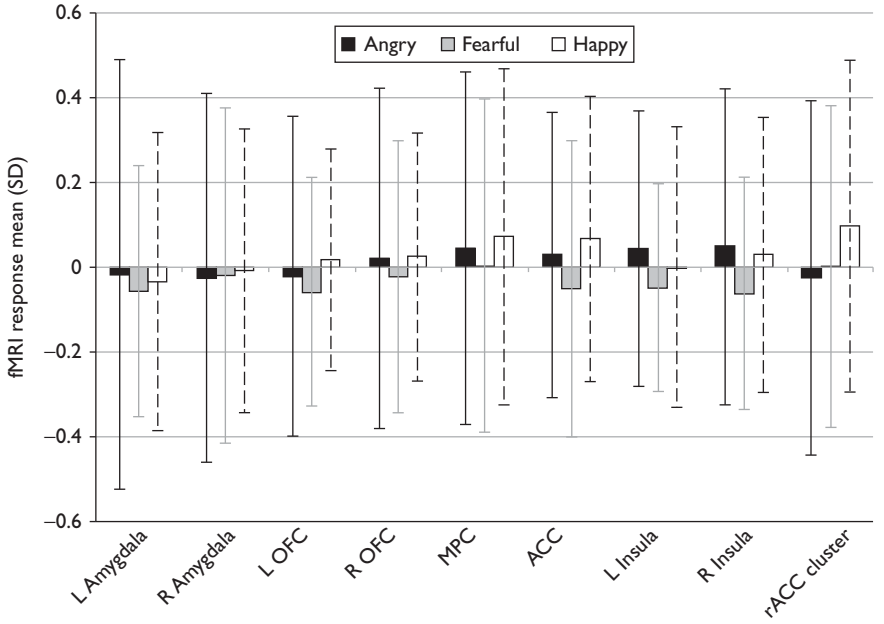
Fig. 3



Mean BOLD activation was extracted and plotted for each of the ROIs for each condition, as well as for the activation cluster observed in the rACC for masked angry faces. Black, masked angry faces; gray, masked fearful faces; white, masked happy faces. ACC, anterior cingulate cortex; fMRI, functional MRI; L, left; MPC, medial prefrontal cortex; OFC, orbitofrontal cortex; R, right; rACC, rostral anterior cingulate cortex. Error bars = 1 SD.

amygdala and anterior cingulate regions in response to the target affective stimuli [15]. The present results extend earlier findings by demonstrating that rACC is also implicated in the process of ES. Interestingly, the correlation between brain activation and emotion-specific ES was only detected in response to angry faces, rather than happy or fearful faces. This suggests a potential evolutionarily relevant role for ES in regulating emotional responses to subtle facial cues of anger. Compared with fear and happiness, anger is an energizing emotion that communicates potential interpersonal threat [23]. Our findings suggest that individuals with a greater tendency to suppress their own angry emotions also show increased responsiveness of the rACC to subtle facial threat cues – potentially reflecting the neurobiology underlying an important component of civilized social discourse.

The present findings may also have relevance to psychiatric disorders. For instance, trait vulnerability to emotional disorders, such as anxiety, has been associated with a bias or sensitivity toward threat-related information (e.g. threatening words, angry faces) [24]. We found that individuals with higher ES of anxiety also showed greater activation of the rACC in response to masked angry faces. These results suggest that individuals who report a greater trait tendency to suppress their anxiety also show greater responsiveness of the rACC region when confronted with subtle facial threat cues presented below normal thresholds of awareness. It is conceivable that heightened sensitivity to threatening facial cues might predispose individuals toward anxious responses and attempts to regulate such experiences. This is a question that could be addressed in future research.

There are several limitations that should be considered. First, the CECS is a self-report questionnaire, which may provide a biased representation of self-perceived ES rather than actual suppression capacities. Second, we limited our primary analyses to only five relatively small ROIs to address specific hypotheses. Clearly, other regions of the brain are also likely to be important in the experience and regulation of emotion. Future research may consider the use of whole-brain analytic strategies. Third, the amygdala is a key brain structure involved in responses to nonconsciously presented emotional stimuli [15,17,25]. The present study did not find an association between amygdala responses and ES. Given the amygdala's important role in emotional processes, its potential involvement should be more closely examined in future studies on nonconscious emotional control using additional complementary imaging techniques, such as connectivity analysis.

Conclusion

Self-reported ES of anger and anxiety is associated with increased functional responses within the rACC to backward masked angry faces, but not fearful or happy faces. The findings suggest that the rACC may play an important role in mediating the association between

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emotion suppression as a trait behavioral tendency and automatic brain responses to threatening stimuli.

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Conflicts of interest

There are no conflicts of interest.

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Intelligence

The role of cognitive versus emotional intelligence in Iowa Gambling Task performance: What's emotion got to do with it?

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ABSTRACT

Debate persists regarding the relative role of cognitive versus emotional processes in driving successful performance on the widely used Iowa Gambling Task (IGT). From the time of its initial development, patterns of IGT performance were commonly interpreted as primarily reflecting implicit, emotion-based processes. Surprisingly, little research has tried to directly compare the extent to which measures tapping relevant cognitive versus emotional competencies predict IGT performance in the same study. The current investigation attempts to address this question by comparing patterns of associations between IGT performance, cognitive intelligence (Wechsler Abbreviated Scale of Intelligence; WASI) and three commonly employed measures of emotional intelligence (EI; Mayer–Salovey–Caruso Emotional Intelligence Test, MSCEIT; Bar-On Emotional Quotient Inventory, EQ-i; Self-Rated Emotional Intelligence Scale, SREIS). Results indicated that IGT performance was more strongly associated with cognitive, than emotional, intelligence. To the extent that the IGT indeed mimics "real-world" decision-making, our findings, coupled with the results of existing research, may highlight the role of deliberate, cognitive capacities over implicit, emotional processes in contributing to at least some domains of decision-making relevant to everyday life.

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1. Introduction

The relative role of emotional versus cognitive processes in driving judgment and decision-making in everyday life remains a topic of substantial interest in the empirical literature (see Kahneman, 2011; Vastfjall & Slovic, 2013). The Iowa Gambling Task (IGT) is among the most extensively used neuropsychological paradigms designed to assess "real-world" decision-making (Bechara, 2004; Toplak, Sorge, Benoit, West, & Stanovich, 2010; for a detailed description of the IGT see Bechara, Damasio, Damasio, & Anderson, 1994; Bechara, Damasio, & Damasio, 2000). In its most common form, the IGT is presented as a simple card game with the explicit goal of winning as much money as possible by selecting cards, one at a time, from any of four decks. With each card selection, the participant wins or loses varying amounts of money. As the game progresses, the participant has the opportunity to learn from experience that some of the decks produce relatively large wins but even larger losses (i.e., "bad decks"), while other decks have modest wins but even smaller losses (i.e., "good decks"). Consistently selecting from the bad decks will ultimately lead to total loss, while selecting consistently from the good decks will lead to long term gain. Early work with the IGT showed that healthy participants begin the game by selecting randomly among the decks, but they soon appear to learn the contingencies as the game proceeds, progressively avoiding the bad decks in favor of the good ones. Critically, during the early phases of the game, healthy individuals also begin to show increased skin conductance responses when considering "bad" deck selections, even before they claim any conscious awareness of the contingencies of the task. This

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increase in skin conductance has been suggested as evidence that participants have begun to learn the deck values at a pre-conscious, emotional level, before they have formed an explicit cognitive understanding of the task (Bechara et al., 2000, 1994). However, the extent to which successful IGT performance is driven more by implicit, emotion-based versus explicit, cognitive processes remains a matter of significant debate (Demaree, Burns, & DeDonno, 2010; Maia & McClelland, 2004).

The IGT is believed to mimic real-life decision-making in that it incorporates the experience of rewards and losses, as well as factoring uncertainty of outcomes and risk (Bechara, Damasio, Tranel, & Damasio, 1997). Whereas overt decision-making is thought to rely on explicit knowledge and reasoning, patterns of IGT performance initially reported in the literature indicated that participants are able to decide advantageously without declarative knowledge of the best strategy (e.g., Bechara et al., 1997). Such findings have been used to bolster the argument that successful IGT performance may be driven more by implicit, emotion-based processes (i.e., "hot" decision-making), rather than primarily through explicit insight of the most favorable strategy derived from data-oriented cost/benefit analyses (i.e., "cold" decision-making; Dunn, Dalgleish, & Lawrence, 2006).

The somatic marker hypothesis (SMH; Damasio, Tranel, & Damasio, 1991; Damasio, 1994, 1996, 2004) provides an explanatory framework for understanding how emotion-based decision-making processes may be driving successful IGT performance. More specifically, the SMH posits that, through prior experience with stimuli or situations, individuals acquire emotion-based biasing signals generated from the physiological systems of the body ("somatic markers"), and that these signals are re-activated when considering analogous response options in the future. These somatic markers may be experienced as visceral "hunches" or "gut feelings" that can bias decision-making (Damasio, 2004). These markers are proposed to help direct attention toward or away from particular response options and thus facilitate more streamlined and efficient decision-making. Interestingly, individuals with damage to a specific region of the brain, the ventromedial prefrontal cortex (VMPFC), appear to be impaired in this process, and tend to exhibit relatively poor performance on the IGT, despite otherwise preserved intellectual capacities (Bechara et al., 1994). It is important to note that although VMPFC lesion patients tend to have generally intact cognitive abilities, they often show profound deficits in social–emotional domains, including deficits in emotion expression, affective experience and regulation, and frequently show a pattern of maladaptive decision-making in their everyday lives (Damasio, 1994). The term "myopia for the future" has been applied to these VMPFC lesion patients as they often display a relatively heightened preference for immediate reward, while neglecting longer-term consequences. It is also critical to point out that, while these VMPFC lesion patients exhibit relatively normal skin conductance responses (SCRs) after a win or loss, they fail to show the anticipatory SCRs exhibited by healthy controls when contemplating a high-risk choice on the IGT (Bechara, Tranel, Damasio, & Damasio, 1996). This pattern of findings has formed the crux of the SMH, suggesting that the VMPFC may be a key brain region involved in integrating physiological responses with cognitive data to form a feeling or hunch that biases decision selection (Bechara et al., 1996). Other regions proposed to underlie the "somatic marker circuitry" (SMC) include the amygdala, insula, anterior cingulate, basal ganglia and somatosensory cortex (Bechara & Damasio, 2005; Dunn et al., 2006). Indeed, one hypothetical model posits that emotional intelligence capacities rely heavily upon the SMC (Bar-On, Tranel, Denburg, & Bechara, 2003). Additionally, a recent review found IGT performance to be generally uncorrelated with traditional "cold cognition" types of executive function tasks (Toplak et al., 2010), suggesting that emotional, rather than cognitive or executive, abilities may primarily drive performance on the IGT.

However, the role of emotion in biasing decision-making on the IGT has not been universally observed. For example, Maia and McClelland (2004) reported results contradicting the notion that IGT participants decide advantageously without declarative knowledge of the best strategy (Bechara et al., 1997). Specifically, the study showed that when participants performed advantageously in the IGT, they tended to be consciously aware of the "goodness" and "badness" of relative decks. Paralleling these findings, Guillaume et al. (2009) showed that better performance on the IGT was associated with explicit knowledge of the underlying contingencies. Moreover, conscious knowledge was not associated with anticipatory SCRs in that study, suggesting that explicit awareness and somatic cues may have two distinct influences on decision-making (Guillaume et al., 2009). Despite the large body of research examining the influence of emotion or cognitive ability separately on IGT performance, there is a surprising paucity of research that aims to disentangle the relative contributions of cognitive versus emotional processes within the same study. To our knowledge, only one study (Demaree et al., 2010) has directly compared the influences of cognitive intelligence (IQ) versus emotional intelligence (EI) on IGT performance in a healthy sample. Interestingly, findings from the latter study showed IQ to be a better predictor of IGT performance than EI, suggesting that the IGT may, in fact, tap cognitive processes to a greater extent than emotional ones (at least EI).

However, the conclusions of the Demaree et al. (2010) study are limited by several factors. First, the authors used a single, self-report measure of emotional intelligence, the Schutte Emotional Intelligence Scale (SEIS; Schutte et al., 1998), which implicitly assumes that patients can reliably access and accurately report on their EI abilities. As noted by the authors, a self-report measure of emotional intelligence may not be sensitive to the fact that participants might rely, at least in part, on implicit, rather than on explicit, knowledge of emotional cues to make their decisions on the IGT, thereby likely limiting the validity of the self-report SEIS. In an attempt to address this limitation, in the current study we employed concurrent EI measures utilizing self-report methodologies (i.e., Bar-On Emotional Quotient Inventory; EQ-i; Bar-On, 2002; Self-Rated Emotional Intelligence Scale; SREIS; Brackett, Rivers, Shiffman, Lerner, & Salovey, 2006), as well as the most commonly used performance-based measure of EI (Mayer–Salovey–Caruso Emotional Intelligence Test; MSCEIT; Mayer, Salovey, Caruso, & Sitarenios, 2003). Second, to assess cognitive ability, the Demaree et al. study relied on the Mill Hill Vocabulary Scale rather than on a "gold standard" measure of IQ (i.e., Wechsler or Stanford–Binet intelligence scales). Thus,

in the current study, we use the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1997). Third, Demaree et al. used a convenience sample of undergraduate students, while our sample consisted of a more diverse group of volunteers with a broader age and education range.

In the present study, we aimed to assess the unique contributions of EI and IQ in predicting IGT performance. We used a Wechsler scale to measure IQ and several commonly-employed EI measures (both self-report and performance-based instruments) to assess the relative predictive value of these constructs. We hypothesized that IQ would be more strongly associated with IGT performance relative to our measures of EI, supporting the notion that the IGT may rely more on cognitive than emotional intelligence.

2. Method

2.1. Participants

Sixty-five healthy volunteers were recruited from the greater Boston area (32 females; ages 18–45, $M = 30.15$, $SD = 8.01$). Some convergent and divergent validity data related to cognitive and emotional intelligence from these participants have been reported elsewhere (Webb et al., 2013), but the associations with the IGT are novel and have not been previously reported. Participants classified themselves as 69.2% White, 15.4% African-American, 9.2% Asian, 3.1% Other, and 3.1% "more than 1 race." Additionally, 4.6% of the sample identified as Hispanic. The primary language of all participants was English. The mean number of years of education was 14.9 (range 11–20). Participants were screened by a trained clinical research assistant for history of DSM-IV Axis I psychopathology, substance abuse, and serious medical or neurological conditions, based on a series of questions adapted from the Structured Clinical Interview for DSM-IV-TR (SCID-I; First, Spitzer, Gibbon, & Williams, 2001). All participants provided written informed consent and received compensation. The study protocol was approved by the McLean Hospital Institutional Review Board and the US Army Human Research Protection Office.

2.2. Measures

2.2.1. Iowa Gambling Task

The Iowa Gambling Task (IGT; Bechara et al., 1997) is a widely used decision-making paradigm that involves the learning of a punishment–reward contingency. Presented as a computerized game, participants start with a play loan of $2000 and choose cards from four identically appearing decks (A′, B′, C′, or D′) in an effort to win as much money as possible. All card selections yield a monetary gain, and some card selections are also associated with a loss that immediately follows the win. Decks A′ and B′ are disadvantageous, high-risk ("bad") decks, meaning that these cards reveal large rewards but even larger losses (expected value < 0). Decks C′ and D′ are advantageous, low-risk ("good") decks, in which cards represent more modest rewards but also much smaller losses (expected value > 0). There are 100 trials and participants are free to switch between decks as often as they like. Participants received standard instructions (Bechara, Tranel, Damasio, & Damasio, 1994) emphasizing that some decks are worse than others and it is possible to win the game by avoiding the bad decks.

2.2.2. Emotional intelligence

Three indices of EI were administered to the participants. The Mayer–Salovey–Caruso Emotional Intelligence Test (MSCEIT; Mayer et al., 2003) is a computerized, performance-based measure of emotional intelligence. The MSCEIT consists of 141 items designed to assess the following four "branches" of EI: (1) perceiving emotions (*Perceiving*), (2) using emotions to facilitate thought (*Facilitating*), (3) understanding emotions (*Understanding*), and (4) managing emotions (*Managing*). The measure yields a *Total EI* score and two Area scores, *Experiential EI* and *Strategic EI*. Experiential EI reflects the ability to perceive emotions in oneself, other persons, and various stimuli (e.g., music and art), and to utilize emotional information to facilitate thought. Strategic EI reflects the ability to understand emotions and their development in oneself and others, and to manage them effectively. Mayer, Salovey and Caruso (2002) reported good reliability values for total MSCEIT scores, including internal consistency (split-half reliability = .91) and test–retest reliability (.86). For additional information on the psychometric properties of the MSCEIT, see Mayer et al. (2002) and Mayer et al. (2003).

The Bar-On Emotional Quotient Inventory (EQ-i; Bar-On, 2002) is a computerized, 133-item self-report assessment of trait EI (i.e., self-perceived EI). The EQ-i yields a *Total Emotional Quotient* (EQ) and five composite scores (i.e., *Interpersonal*, *Intrapersonal*, *Adaptability*, *Stress Management*, *General Mood*). Bar-On (2004) reported that the EQ-i demonstrated good reliability (internal consistency and test–retest reliability). For detailed information on the psychometric properties of the EQ-I, see Bar-On (2004).

The Self-Rated Emotional Intelligence Scale (SREIS; Brackett et al., 2006) is a 19-item self-report questionnaire that taps into similar EI capacities assessed by the MSCEIT. Like the MSCEIT, the SREIS measures the perception, management, use, and understanding of emotions in oneself and others, but uses a self-report format that is similar to that of the EQ-i. Items such as "By looking at people's facial expressions, I recognize the emotions they are experiencing" are rated on a 5-point Likert scale ranging from 1 ("very inaccurate") to 5 ("very accurate"). In a series of studies, Brackett et al. (2006) reported the following Cronbach's alphas for the SREIS: .84, .77, and .66 for Studies 1, 2, and 3, respectively.

2.2.3. Cognitive intelligence

The Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1997) was used to assess IQ. The WASI is one of the most widely used intelligence scales and has reported reliability of .98 for Full Scale IQ, with high test–retest reliability, and correlates .92 with the Wechsler Adult Intelligence Scale-III (WAIS; Pearson Assessment, Inc., San Antonio, TX). The measure yields scores for Full Scale IQ, Verbal IQ, and Performance IQ. The WASI was individually administered by a trained research technician under the supervision of a licensed doctoral level neuropsychologist.

2.3. Procedure

Given the relatively large number of performance-based tests and self-report measures administered, testing occurred over two consecutive days in order to reduce participant fatigue. During the first testing session, participants completed demographic forms, consent forms, the MSCEIT, EQ-i, and SREIS. On the second day of testing, participants completed the WASI and IGT. All testing took place at McLean Hospital in a private testing room. All assessments were administered according to published instructions and procedures. Complete data from ten participants were not available for either the WASI or the IGT due to computer malfunctions or participant discontinuation, so those data were excluded from the analyses reported below.

3. Results

3.1. IGT performance

Performance on the IGT was scored by dividing the task into 5 blocks of twenty trials (e.g., block 1 = cards 1–20, block 2 = cards 21–30, etc.). A net score was calculated for each block by subtracting the number of cards chosen from the high-risk decks (A′ and B′) from the number of cards chosen from the low-risk decks (C′ and D′). Positive net scores reflected overall advantageous decision-making, while a negative value reflected overall disadvantageous decision-making for each block.

Consistent with prior reports in healthy samples, participants showed the typical learning curve indicative of increased advantageous decision-making across the 5 blocks of the IGT (see Bechara, 2004; Bechara et al., 2000), with scores sharply improving over the first 2 blocks and plateauing during blocks 3–5 (see Fig. 1). To statistically test for this learning curve, a repeated-measures analysis of variance (ANOVA) was conducted, with an independent variable of block and a dependent variable of the net scores. There was a main effect of block, $F(3,166) = 17.67$, $p < .001$ (Greenhouse–Geisser corrected). Post-hoc Least Significant Difference (LSD) tests revealed that block 1 performance ($M = -5.27$, $SD = 9.08$) was significantly worse than performance on blocks 2 through 5 (block 2: $M = 3.71$, $SD = 8.53$; block 3: $M = 5.38$, $SD = 10.46$; block 4: $M = 5.64$, $SD = 11.20$; block 5: $M = 5.49$, $SD = 11.30$; $p < .001$ for each comparison). No other significant differences emerged.

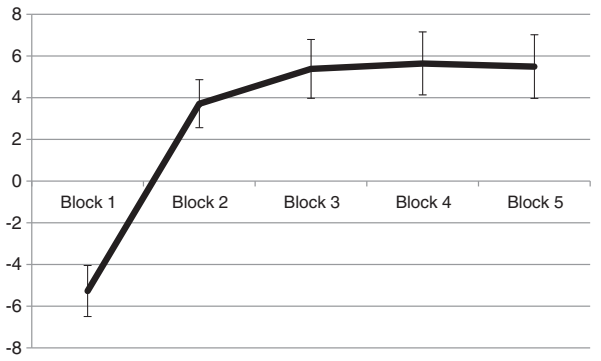

Fig. 1. IGT performance (+/−1 SEM) as a function of block. The y-axis denotes the number of cards chosen from the "good" decks minus cards chosen from the "bad" decks.

3.2. Zero-order correlations

Bivariate Pearson correlations were run to test for linear associations between performance on the IGT and the IQ and EI measures. Means (*M*), standard deviations (*SD*) and intercorrelations are listed in Table 1. IGT performance (IGT Total) was significantly correlated with Full Scale IQ ($r = .47$, $p < .001$), including both Verbal IQ ($r = .40$, $p = .002$) and Performance IQ ($r = .45$, $p = .001$). IGT performance was also significantly correlated with the MSCEIT ($r = .36$, $p = .007$). When examining the constituent branches of the MSCEIT, IGT Total score was significantly correlated with Facilitating ($r = .28$, $p = .038$) and Understanding ($r = .43$, $p = .001$), but not Managing ($r = .11$, $p = .415$); a nonsignificant trend emerged for the Perceiving branch ($r = .24$, $p = .082$). However, there were no significant correlations between IGT performance and the two self-report measures of emotional intelligence (EQ-i: $r = .01$, $p = .937$; SREIS: $r = .23$, $p = .099$; see Table 1 for results by IGT block).

3.3. Partial correlations

Next, given the significant bivariate correlations between EI and IQ scores (see Table 1), a series of partial correlations was conducted to test the association between IGT performance (net score on blocks 1–5) and scores on each EI measure (MSCEIT, EQ-i and SREIS total scores) after controlling for Full Scale IQ. As illustrated in Fig. 2, after controlling for Full Scale IQ, there were no significant associations between any of the three EI measures and IGT performance across any blocks (for MSCEIT, all $r < .16$ & $p > .27$; for EQ-i, all $r < .02$ & $p > .45$; for SREIS, all $r < .10$ & $p > .46$).

In contrast, when controlling for the different measures of EI, Full Scale IQ remained significantly associated with several IGT performance variables. Specifically, when controlling for MSCEIT scores, Full Scale IQ was significantly associated with IGT performance in blocks 3–5 (block 3, $r = .33$; $p = .015$; block 4, $r = .36$; $p = .008$; block 5, $r = .34$; $p = .011$). When controlling for EQ-i scores, Full Scale IQ was significantly associated with IGT performance in blocks 2–5 (block 2, $r = .33$; $p = .016$; block 3, $r = .40$; $p = .003$; block 4, $r = .46$; $p < .001$; block 5, $r = .46$; $p < .001$). Similarly, when controlling for SREIS scores, Full Scale IQ was significantly associated with IGT performance in blocks 2–5 (block 2, $r = .28$; $p = .042$; block 3, $r = .37$; $p = .006$; block 4, $r = .42$; $p = .002$; block 5, $r = .43$; $p = .001$).

3.4. Hierarchical multiple regressions

Separate hierarchical multiple regression analyses were performed to examine the incremental contribution of the *combined* EI measures over IQ in predicting IGT performance. Similarly, we tested whether IQ remained significantly associated with IGT performance after controlling for all three EI measures simultaneously (and providing accompanying R^2 change values). Given that several of our predictor variables were significantly correlated (see Table 1), Tolerance values were examined to test for issues of multicollinearity in our

Table 1
Means, standard deviations, and correlations for investigated variables.

	M	*SD*	2	3	4	5	6	7	8	9	10	11	12
1. MSCEIT	103.06	12.09	.11	.32*	.52**	.52**	.43**	.36**	.06	.25	.23	.30*	.35**
2. EQ-i	101.42	13.63	–	.50**	.22	.17	.24	.01	.00	−.02	.03	.00	.02
3. SREIS	73.06	8.04		–	.30*	.37**	.17	.23	.02	.18	.14	.21	.20
4. IQ — Full	111.10	16.08			–	.92**	.91**	.47**	−.08	.31*	.39**	.45**	.46**
5. IQ — Verbal	110.40	15.76				–	.69**	.40**	−.09	.26	.33*	.39**	.43**
6. IQ — Perf.	108.85	15.43					–	.45**	−.04	.31*	.39**	.43**	.41**
7. IGT Total	14.95	34.96						–	.34**	.59**	.72**	.87**	.85**
8. Block 1	−5.27	9.08							–	.10	−.10	.10	.18
9. Block 2	3.71	8.53								–	.34*	.33*	.36**
10. Block 3	5.38	10.46									–	.63**	.49**
11. Block 4	5.64	11.20										–	.78**
12. Block 5	5.49	11.30											–

Note: MSCEIT = Mayer–Salovey–Caruso Emotional Intelligence Test; EQ-i = Bar-On Emotional Quotient Inventory; SREIS = Self-Rated Emotional Intelligence Scale; IQ-Full/Verbal/Performance = Wechsler Abbreviated Scale of Intelligence — Full-Scale/Verbal IQ/Performance IQ; IGT = Iowa Gambling Task (blocks 1–5 and Total score).
* $p < .05$.
** $p < .01$.

models. It has been suggested that Tolerance values of 0.2 or below are worthy of concern (Field, 2009; Menard, 1995). In the below models, Tolerance values ranged from .68 to .95, thus indicating no significant multicollinearity. All predictor variables were forced into entry in our multiple regression models.

The first set of hierarchical multiple regressions predicted IGT performance (i.e., net score on IGT blocks 1–5 and IGT Total score) from Full Scale IQ (Step 1) and our three EI measures (Step 2). Six separate multiple regressions were conducted, predicting IGT performance in blocks 1–5 and Total IGT score separately. As presented in Table 2, in none of the multiple regressions did the combined EI measures (Step 2) predict a significant amount of variance above and beyond Full Scale IQ scores (Step 1).

Finally, as presented in Table 3, we conducted a similar set of six hierarchical multiple regressions with Steps 1 and 2 reversed. Specifically, we predicted IGT performance (i.e., net score on IGT blocks 1–5 and IGT Total score) from our three EI measures (Step 1) followed by Full Scale IQ (Step 2). Even after controlling for all three EI measures, Full Scale IQ remained a significant predictor of IGT Total score and performance in blocks 3–5 (it should be noted that, when IQ was not statistically controlled, the combined EI measures did significantly predict IGT performance on block 5 and for IGT Total score).

4. Discussion

The present study sought to directly compare the relative influence of cognitive intelligence (IQ) versus emotional intelligence (EI) on IGT performance. Both IQ and the performance-based measure of EI (MSCEIT) were significantly correlated with IGT performance, whereas neither of the self-reported measures (EQ-i and SREIS) was associated with the IGT. It is interesting to note that the MSCEIT evidenced relatively small correlations with the two EI measures that were not associated with IGT performance (MSCEIT-EQ-i $r = .11$; MSCEIT-SREIS $r = .32$), whereas the EQ-i and SREIS exhibited a significant and relatively strong association with one another ($r = .50$).

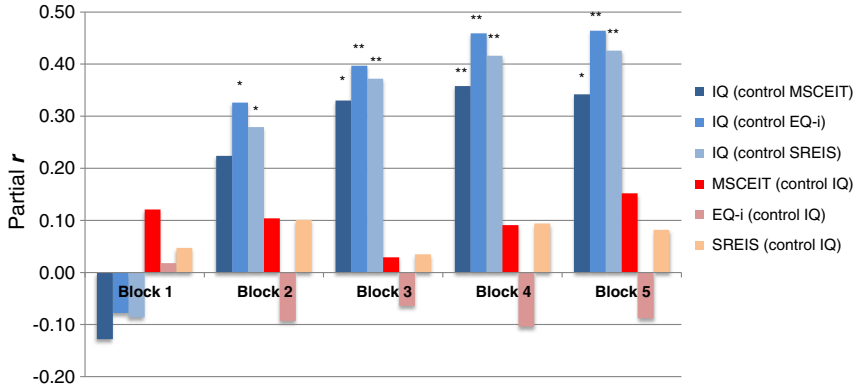

Fig. 2. The partial correlation of IGT performance (blocks 1–5) with our measures of IQ and EI. Each bar represents a partial *r*: i.e., the correlation between EI scores and IGT performance, after controlling for Full Scale IQ; and the correlation between Full Scale IQ and IGT performance, controlling for scores on the MSCEIT, SREIS, or EQ-i. **$p < .01$; *$p < .05$.

Table 2
Hierarchical multiple regression analyses testing incremental contribution of combined EI measures over IQ in predicting IGT performance.

Criterion (IGT block)	Regression step	Predictor	ΔR^2	ΔF	Sig *F*. change (*p* values)
Block 1	1	IQ	.006	.30	ns
	2	EI	.015	.26	ns
Block 2	1	IQ	.099	5.81	.019
	2	EI	.037	.72	ns
Block 3	1	IQ	.155	9.70	.003
	2	EI	.008	.17	ns
Block 4	1	IQ	.202	13.41	.001
	2	EI	.033	.72	ns
Block 5	1	IQ	.209	14.01	<.001
	2	EI	.035	.76	ns
IGT Total	1	IQ	.218	14.73	<.001
	2	EI	.044	.99	ns

Note: IQ = Wechsler Abbreviated Scale of Intelligence — Full Scale IQ; IGT = Iowa Gambling Task. EI = All three EI measures entered in Step/block 2 (Mayer–Salovey–Caruso Emotional Intelligence Test; Bar-On Emotional Quotient Inventory; Self-Rated Emotional Intelligence Scale).
ns = nonsignificant.

A series of hierarchical regression analyses revealed that most of the variance in IGT performance was accounted for by IQ rather than by EI measures. Specifically, the MSCEIT failed to contribute significantly above and beyond IQ in the prediction of IGT performance. On the other hand, as hypothesized, IQ contributed uniquely to the prediction of IGT even after controlling for scores on EI measures. Our results build upon and further advance those of Demaree et al. (2010) who reported that IQ, but not EI, was associated with IGT performance. However, the current investigation augments the previous findings by also addressing several limitations of the prior work. In particular, whereas the study by Demaree and colleagues relied on a single, self-report measure of EI, the current study used several EI measures, including popular self-report measures (EQ-i & SREIS), as well as the most commonly used performance-based measure (MSCEIT). In addition, to assess cognitive ability, the Demaree et al. study relied on the Mill Hill Vocabulary Scale rather than on one of the widely used "gold standard" measures of IQ (i.e., Wechsler or Stanford–Binet intelligence scales). Accordingly, to address this limitation, in the current study we employed an abbreviated version of the Wechsler scales. Third, Demaree et al. used a convenience sample of undergraduate students, while our sample consisted of a relatively diverse group of participants with a broader age and education range.

Our work is also consistent with other recent studies that have found associations between better IGT performance and greater executive function (e.g., Brand, Recknor, Grabenhorst, & Bechara, 2007) and possessing explicit knowledge of the advantageous strategy (e.g., Maia & McClelland, 2004; also see Roca et al., 2010 for finding linking IGT performance to "*g*"). Taken together, these results suggest that IGT performance may recruit more deliberate, cognitive processes than implicit, emotional ones. As noted above, the observation that VMPFC lesion patients perform poorly on the IGT – and do not evidence anticipatory SCRs (Bechara et al., 1996) – was interpreted as evidence that such individuals lacked integrity of the neural circuitry needed to generate somatic marker biasing-signals that could guide selection of cards from the "good" decks. However, emerging evidence suggests that poor performance on the IGT may be alternatively explained by cognitive, rather than by emotion-based, deficits. As noted by Demaree et al. (2010), the standard IGT requires reversal learning – a cognitive skill – to be successful on the task. Specifically, the "bad" decks in fact provide larger rewards and no losses early in the IGT, and losses only accrue after

Table 3
Hierarchical multiple regression analyses testing incremental contribution of IQ over combined EI measures in predicting IGT performance.

Criterion (IGT block)	Regression step	Predictor	ΔR^2	ΔF	Sig *F*. change
Block 1	1	EI	.004	.07	ns
	2	IQ	.017	.86	ns
Block 2	1	EI	.089	1.67	ns
	2	IQ	.047	2.70	ns
Block 3	1	EI	.061	1.10	ns
	2	IQ	.103	6.12	.017
Block 4	1	EI	.120	2.31	.087
	2	IQ	.115	7.53	.008
Block 5	1	EI	.142	2.82	.048
	2	IQ	.101	6.70	.013
IGT Total	1	EI	.155	3.11	.034
	2	IQ	.106	7.21	.010

Note: IQ = Wechsler Abbreviated Scale of Intelligence — Full Scale IQ; IGT = Iowa Gambling Task. EI = All three EI measures entered in Step/block 1 (Mayer–Salovey–Caruso Emotional Intelligence Test; Bar-On Emotional Quotient Inventory; Self-Rated Emotional Intelligence Scale).
ns = nonsignificant.

several trials, thus requiring an eventual shift to the "good" decks (which provide relatively small wins, but even smaller losses). Fellows and Farah (2005) tested VMPFC lesion patients in a variant of the IGT and found that the poor performance of these patients seemed to be attributable to a specific deficit in reversal learning.

Contrary to the present findings, it should be noted that there are prior studies supporting the claim that emotion-based processes drive successful IGT performance. For example, other studies have failed to find significant associations between IGT performance and either executive functions or intelligence (see Toplak et al., 2010 for a review). A key strength of the current study, however, was the fact that competing measures of EI and IQ were directly compared in the same investigation. Undoubtedly, more research is warranted to fully disentangle the relative role of, and interaction between, cognitive and emotional processes in driving IGT performance.

Of course, it is important to note that current EI measures assess a variety of emotion-related constructs (Webb et al., 2013), and it may be that they do not adequately tap the implicit, emotion-based processes proposed by the SMH that may be relevant to decision-making. It should be highlighted that the MSCEIT was significantly correlated with performance on the IGT, indicating that those with higher emotional intelligence as measured by the MSCEIT did perform better on the task (although, as noted above, this relationship was no longer significant after controlling for IQ). Interestingly, the two MSCEIT branches that correlated most strongly with success on the IGT were those designed to assess the ability to use emotional information to facilitate thought (*Facilitating*) and the ability to understand emotions and their development in oneself and others (*Understanding*). In line with the SMH, one could argue that those who are especially adept at noticing emotional fluctuations in themselves (including "emotional hunches" regarding advantageous response options) and using that information to facilitate decision-making would do particularly well on the IGT. In addition, although the emotional intelligence field is relatively young, a growing body of research has begun to investigate the underlying neural substrates of EI. Interestingly, the regions identified seem to overlap with critical regions of the SMC, in particular the VMPFC (Killgore & Yurgelun-Todd, 2007; Killgore et al., 2013; Krueger et al., 2009). Thus, these findings provide at least some evidence that EI and SMC may have partially overlapping neural substrates.

The fact that the MSCEIT was significantly associated with IGT performance, but that this association was no longer significant after controlling for IQ is intriguing. The MSCEIT has been shown to correlate with measures of cognitive intelligence, including IQ (e.g., Mayer, Roberts, & Barsade, 2008; Webb et al., 2013). Thus, one interpretation of our finding is that the aspects of the MSCEIT that predict IGT performance are the more "cognitive" features of the measure.

Ultimately, more research is also needed to establish the ecological validity of the IGT task. Namely, to what extent does IGT performance in fact mirror "real-world" decision-making, as its developers intended. To the extent that the IGT does bear relevance to decision-making in real life, our findings, along with those of others (e.g., Demaree et al., 2010), may highlight the contribution of cognitive capacities over emotional intelligence in contributing to at least some domains of "real-world" decision-making.

4.1. Limitations

Several limitations of the current study should be noted. First, our sample size was relatively small, limiting statistical power. Nevertheless, we found a number of significant and intriguing relationships in line with our hypotheses. Second, we used the WASI rather than the full-length WAIS. Although the WASI has been to shown to correlate very highly with the WAIS, it still would have been ideal to use the full-length measure. Finally, although we used several of the most commonly used measures of EI, the field is relatively new and debate persists regarding the validity of existing measures in adequately capturing EI (Webb et al., 2013). Additional research is clearly needed to further operationalize EI and to refine existing measures, or develop new measures, to more adequately assess this complex construct and investigate its role in relevant domains of decision-making.

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Development and Validation of the Design Organization Test (DOT): A Rapid Screening Instrument for Assessing Visuospatial Ability

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A brief paper-and-pencil instrument was developed to rapidly assess visuospatial ability and serve as an alterative to the WAIS Block Design subtests during screening or when assessment time is limited. The Design Organization Test (DOT) consists of square black-and-white grids with visual patterns similar to those of the Block Design subtests. Administration is straightforward and requires examinees to reproduce as many designs as possible in 2 minutes using a numerical code key. For 411 college students, alternate forms of the DOT yielded reliability estimates comparable to that of the test-retest reliability of WAIS-III Block Design subtest. In a clinical sample, the DOT was significantly correlated (r = .92) with WAIS-III Block Design scores and was successfully substituted in place of Block Design raw scores without significant change in Performance IQ or Full Scale IQ. The results suggest that the DOT provides a useful and rapid screening measure of visuospatial ability.

The Wechsler scales are the most widely used tests of intellectual functioning (Daniel, 1997). The most recent publication of the 3rd Edition of the Wechsler Adult Intelligence Scale (WAIS-III) (Wechsler, 1997) includes 11 core subtests used in calculating Full Scale IQ. The Block Design subtest was retained in the recent revision virtually unchanged in any substantive way from previous editions of the Wechsler scales. This subtest presents patients with a set of plastic blocks, with two sides colored solid red, two sides colored solid white, and two sides bisected diagonally to form a red and a white triangle on each surface. Examinees are required to put the blocks together as quickly as possible to match an exemplar design presented in a stimulus booklet. Of all of the Performance subtests on the WAIS-III, the Block Design subtest has been found to have one of

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the highest loadings on the general intelligence factor "g" (Sattler & Ryan, 1998). Block Design scores also correlate highly with neuropsychological measures of visuospatial ability (Merten, 2002) and everyday visuospatial skills (Groth-Marnat & Teal, 2000).

Despite its strengths as a measure of visuospatial ability, there are some drawbacks to the Block Design subtest that make it difficult to use in certain settings. Because Block Design requires the use of fine motor skills to construct the designs, scores on the test may not provide the best assessment of visuospatial abilities for patients suffering from motor impairments. In such instances, other subtests such as Matrix Reasoning, which requires examinees to visually inspect colored design patterns and make a response choice, may provide a more valid assessment of visuospatial performance-related intellectual ability (Schoop, Herrman, Johnstone, Callahan, & Roudebush, 2001). Furthermore, comprehensive clinical neuropsychological assessments often require many hours of administration time, leading to fatigue in patients who are likely to have limited stamina due to their medical or neurological condition. Administration time for the WAIS-III may range from 60 to 90 minutes for healthy subjects (Wechsler, 1997), and can easily require over two hours for some clinical patients (Ryan, Lopez, & Werth, 1998). The Block Design subtest itself can easily exceed 15 minutes of administration time with some slower examinees. The need to gather more clinically relevant data in less time has led to an increasing emphasis on the development of short-forms of existing assessment instruments (Axelrod, Dingell, Ryan, & Ward, 2000; Donders, 2001; Kulas & Axelrod, 2002; Merten, 2002; Mount, Hogg, & Johnstone, 2002; Purdon & Waldie, 2001; Ringe, Saine, Lacritz, Hynan, & Cullum, 2002). Tests that can provide the same information about cognitive status or ability in significantly less time are likely to be preferred by patients, examiners, and third party payers alike (Donders, 2001). In addition, there are some occasions where examiners need only a brief screening instrument, or for practical reasons are not able to carry bulky testing equipment (e.g., multiple blocks, stimulus booklets).

To accommodate the need for a rapidly administered screening instrument for visuospatial ability, we developed a single-page paper-and-pencil assessment instrument designed to provide similar visuospatial information to that of the Block Design subtest in significantly less time and with less demand for fine-motor coordination or multiple pieces of equipment. This new instrument, the Design Organization Test (DOT), consists of 9 black and white square patterned grids similar in appearance, though not identical, to the WAIS-III Block Design stimuli. Below each design pattern is a grid of empty squares. A code key with 6 black and white squares and their corresponding numbers is placed at the top of the page. Examinees are required to reproduce the designs by writing in the corresponding numbers obtained from the key at the top of the page. In Study 1, we developed two alternate forms of the DOT and administered them to a large sample of college students to obtain fundamental reliability estimates. In Study 2, we administered the DOT to a sample of clinic patients presenting with a variety of neurological and psychiatric complaints during a neuropsychological evaluation. This second study permitted direct comparison of the DOT to the Block Design subtest of the WAIS-III to determine the new instrument's validity and clinical usefulness for assessing visuospatial ability.

Study 1: Item Development and Preliminary Assessment of Reliability

Overview

Two alternate forms of the DOT were prepared and administered to a large sample of undergraduate students in order to obtain estimates of reliability and to develop a preliminary normative database.

Method

Subjects. Participants (n = 418) were recruited from a large undergraduate psychology course at the University of Pennsylvania and included 181 males and 237 females ranging in age from 17 to 27 years (M = 18.8, SD = 1.0). Participation was voluntary and no incentives were provided. Data were examined initially for outliers and it was determined that a few subjects demonstrated statistically atypical response patterns (i.e., obtained scores differing by more than 2.5 SDs in their relative performance on the two alternate versions of the test administered during the same session). These 7 subjects accounted for less than 1.7% of the total sample and were excluded from further analysis. This final sample (n = 411) included 179 males and 232 females ranging in age from 17 to 26 years (M = 18.8, SD = 1.0).

Materials. Two alternative forms of the DOT were developed. Each form included nine square designs (5 small designs and 4 large designs) comprised of smaller black and white squares and triangles (Figure 1 shows the sample practice item). Beneath each design was a response grid of identical size and shape (five 2×2 grids; four 3×3 grids), yielding a total of 56 response blanks on the form. At the top of each form was a response key consisting of 6 numbered squares (1 black square, 1 white square, and 4 squares that are half black and half white, divided along the diagonal in different orientations).

Procedure. Subjects completed the two alternate forms of the DOT as a large group in a lecture hall. Forms were administered in a counterbalanced order (i.e., 259 subjects received Form A first and Form B second; 161 subjects received Form B first and Form A second). A copy of the practice page of the DOT was displayed on an overhead projector as the investigator read the following instructions aloud:

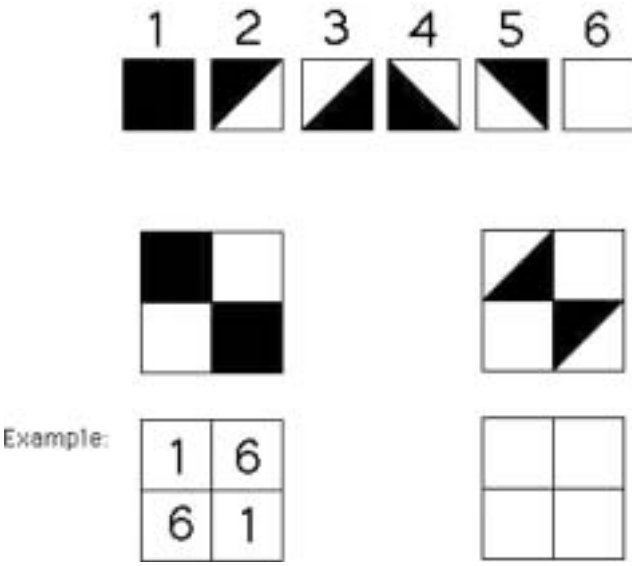

Figure 1. Code key and practice items given to examinees prior to administration of the DOT test page.

> Look at these six boxes. Each box is different. Every box has a different design and has its own number from 1 to 6. Number 1 is solid black. Number 2 is half black and half white. So is number 3, but if you look closely, you'll see that the design for box number 3 is different from number 2. All six boxes are different, and each one has its own number.

The investigator then pointed to the example items below the key and read:

> Now look down here. This square design is made up of four of the boxes I just showed you. Down below are empty squares for you to put the numbers that match each box. This first one is already done for you. See, the first square is solid black. Look at the code key, the number for all solid black squares is number 1. So number "1" goes in this square. The lower right box is also black, so I also put a "1" in that square too. Here are two white boxes. See, the solid white boxes are always number 6, so the number "6" goes here and here.

The investigator then pointed to the next example item and said:

> Over here is a set of empty squares. See if you can match the design by filling in the code numbers. See, the first box is solid white. So what number would you put down here to match? Go ahead and complete the rest of the practice items.

Once the participants had completed the practice items, the investigator said:

> So all you have to do is put the correct number in each of the empty boxes to match the pattern above it. On the next page there are many designs much like the ones we just did. Look at each design and fill in as many of the empty boxes below it as you can with the correct numbers to match the designs. Do as many as you can. You will have only two minutes, so work as quickly as you can. Do you have any questions?

Subjects then worked for 120 seconds. At the end of the 120 seconds the examiner told the subjects to stop and put down their pencils. Subjects were then told that they would complete a second page in the same manner as the one they had just finished. Subjects were told to turn the page and begin. Again, subjects were given 120 seconds to complete as many of the 56 items as possible. There was no requirement for items to be completed in any particular order to be scored as correct. An item was counted as correct if its grid square contained a readable numeral that matched the corresponding correct item on the response key. The score was tallied as the total number of correctly completed response squares.

Results

Correlation Between Forms A and B. As an estimate of alternate forms reliability, the two forms were subjected to correlation analysis using Pearson's *r*. Overall the two forms were highly correlated with each other, $r = .80$, $p < .00001$, suggesting good alternate forms reliability. Furthermore, the strength of the correlation was not dependant on the administration order of the two forms (Fisher's r-to-z transformation, $z = 0.65$, ns), with

the correlation obtained for Form A followed by Form B (r = .81) similar to the correlation obtained when Form B preceded Form A (r = .79).

Comparability of Mean Scores Between Forms A and B. When administered as the first form, there was no significant difference between the scores obtained from subjects taking Form A (M = 44.25, SD = 7.79) and those taking Form B (M = 43.61, SD = 7.83), t_{409} = 0.80, ns. When administered as the second form in the series, there was also no significant difference between groups taking Form A (M = 48.23, SD = 7.04) and Form B (M = 48.20, SD = 7.45), t_{409} = 0.05, ns. This suggests that the two forms provide comparable mean scores and are essentially identical in difficulty level.

Practice Effects. As expected for a psychomotor task, there was a significant practice effect between the administrations of the alternate forms within the same session, t_{410} = 17.93, $p < .001$. On average, the first administration of the DOT, regardless of form, resulted in an average of 44.00 (SD = 7.80) items correct, while the second administration yielded an average of 48.22 (SD = 7.19) items correct, an overall increase of 4.22 (SD = 4.77) units due to practice effects. For subjects administered Form A followed by Form B, the mean practice effect was 3.99 (SD = 4.62) units, t_{252} = 13.74, $p < .001$. Similarly, for subjects administered Form B then Form A, there was also a significant increase of 4.58 (SD = 4.99) units, t_{157} = 11.54, $p < .001$).

Discussion

Despite a large group administration with a homogenous student population from a competitive private university, the DOT had high alternate forms reliability. Furthermore, the two forms of the DOT yielded nearly identical mean scores and standard deviations when compared between groups at the same administration time. These data suggest that the DOT can be administered rapidly and that reliable data can be obtained using either version. The present data provide a preliminary normative database for further research and eventual application of the DOT in clinical settings.

Study 2: Validity and Clinical Utility

Overview

In order to establish its validity within a clinical setting, the DOT was administered in conjunction with a comprehensive neuropsychological test battery to a sample of patients with various types of neurologic illness. We hypothesized that DOT performance would be significantly correlated with performance on the Block Design subtest of the WAIS-III and that replacement of Block Design scores with a score derived from the DOT when calculating Performance and Full Scale IQ, would not lead to any significant change in either IQ score.

Method

Subjects. While undergoing a comprehensive neuropsychological evaluation at a large medical center in the Northeastern United States, 41 neurologic patients (23 male; 18 female) were administered the DOT in conjunction with a large battery of neuropsychological tests that included the WAIS-III Block Design subtest. A subset of these patients

received the entire WAIS-III (n = 20) and/or enough of the Performance subtests to permit calculation of Full Scale and Performance IQ scores. The clinical sample included patients ranging in age from 18 to 76 years (M = 47.7, SD = 15.3), with an average of 14.6 (SD = 3.4) years of formal education. Because our aim was to examine the validity of the DOT in predicting Block Design scores regardless of diagnosis, and not to describe any particular neurologic condition, we made no effort to screen for particular disorders. Patient diagnoses included seizure disorder (n = 6), intracranial tumor (n = 6), dementia (n = 4), toxic/metabolic problems (n = 3), traumatic brain injury (n = 1), cerebrovascular accident (n = 6), demylinating disorder (n = 1), psychiatric or unclassified disorder (n = 14).

Materials and Procedure. Patients were tested individually and were administered the WAIS-III subtests in accordance with the standard instructions published in the test manual (Wechsler, 1997). Depending on the presenting medical condition, most patients received a number of other neuropsychological tests as well. To prevent any bias in actual clinical assessments, the DOT was always administered near the completion of the test battery, at least an hour after the WAIS-III. Patients were presented with Form A of the DOT and given the same general instructions as those described in Study 1. Patients were given a pencil and instructed to follow along as the examiner demonstrated the first practice item, which was printed on a separate sheet from the DOT test form (see Figure 1). The patient then completed the second practice item in front of the examiner. Patients were permitted to ask questions and were given further guidance and prompting if they appeared to be having difficulty completing the practice item. Once the practice item had been completed and all questions were answered to the patient's satisfaction, the patient was given the test form and timed for 120 seconds. There was no requirement for patients to complete the items in any particular order, but they were encouraged to complete as many items as possible within the two-minute time limit.

Results

Performance Assessment. Consistent with their status as neurologic patients, the participants in this study obtained a mean WAIS-III Block Design non-age corrected scaled score of 8.58 (SD = 3.71), which is significantly below the standard scaled score of 10, t_{40} = -2.44, p = .019, established by the normative group (Wechsler, 1997). On the DOT, patients obtained a mean score of 24.32 (SD = 12.75), which is significantly lower than the mean score for Form A obtained by the healthy college students in Study 1, t_{300} = 13.03, p < .001. This difference remained significant even when age was entered as a covariate between groups, $F_{1,297}$ = 4.04, p = .045.

Concurrent Validity. The scores on the DOT and the WAIS-III Block Design scaled scores (non-age corrected) were subjected to a Pearson correlation analysis. Scores on the DOT were highly correlated with those on the Block Design test, r = .92, p < .0001 (see Figure 2), suggesting that both tests measure similar visuospatial abilities and supporting the concurrent validity of the DOT. Scores on the DOT also correlated significantly with age (r = -.58, p < .001), but not with level of education (r = .13, ns). Even after removing the effects of age, however, the DOT remained highly correlated with the Block Design subtest, (partial r = .90, p < .0001). Table 1 summarizes the correlations between the DOT, Block Design, and other commonly used subtests of the WAIS-III.

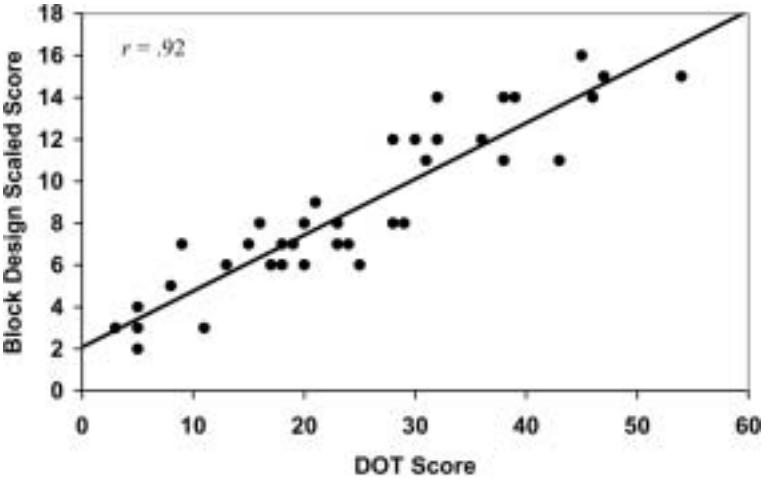

Figure 2. Scatterplot showing the linear relationship between DOT scores and scaled scores on the Block Design subtest of the WAIS-III.

Table 1
Correlations between DOT scores, Block Design scores, and Criterion measures of intelligence and mood

Criterion test	DOT	Block Design	*n* pairs
Age	-.58**	-.46**	41
Education	.13	.27	36
VIQ	.54**	.69**	23
PIQ	.74**	.86**	24
FSIQ	.66**	.82**	23
Block design	.92**	-	41
Information	.36	.51**	29
Digit span	.58**	.62**	36
Vocabulary	.40	.59**	23
Arithmetic	.62**	.74**	25
Comprehension	.44**	.59**	34
Similarities	.57**	.68**	23
Picture completion	.63**	.69**	29
Picture arrangement	.54**	.62**	24
Digit symbol	.68**	.59**	25
Beck Depression Inventory	.03	-.11	32
State anxiety	-.02	-.10	30
Trait anxiety	.15	.01	30

*$p < .05$, 2-tailed; **$p < .01$, 2-tailed.

Discriminant Validity. The DOT was not expected to correlate with clinical variables unrelated to the theoretical construct of visuospatial ability. To test this, Pearson correlations were calculated between the scores on the DOT and three measures of mood given to most patients during the same assessment session. As expected, there was no correlation between the DOT and the Beck Depression Inventory (Beck & Steer, 1993) (r = .03, ns, n = 32), or the state (r = -.02, ns, n = 30) or trait (r = .15, ns, n = 30) components of the Spielberger State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970). These data are also summarized in Table 1.

Clinical Utility. Given the significant positive correlation between the DOT and the Block Design scaled scores, it was therefore of interest to determine whether the DOT could serve in lieu of Block Design raw scores when calculating WAIS-III Performance and Full Scale IQ scores. For this analysis, we replaced each subject's obtained block design raw score with an estimated raw score derived from their performance on the DOT. Specifically, scores from the DOT were entered into a linear regression analysis to estimate raw scores on the Block Design subtest (21 of the 41 patients had raw block design scores and complete WAIS-III data for calculating Performance and Full Scale IQ scores). Twenty-one separate regression analyses were conducted using a "leave-one-out" bootstrap procedure whereby each subject in the sample was temporarily excluded from the dataset and the DOT scores of the remaining 20 subjects were used to estimate Block Design scores. The regression equation obtained during each iteration was then used to predict the Block Design score of the excluded subject from that subject's own DOT score. All regression analyses were highly significant (all ps < .0001), with R^2 values ranging from 0.92 to 0.93. The estimated Block Design raw scores that were yielded by the regression equations were then substituted in place of the Block Design raw scores during a recalculation of Performance and Full Scale IQ scores. Replacement of actual Block Design raw scores with scores estimated from the DOT yielded Performance IQ scores that were nearly indistinguishable from those calculated with true Block Design scores, t_{20} = 1.10, ns. As evident in Figure 3, Performance IQ scores calculated using the DOT (M= 96.5, SD = 18.2) differed by approximately one-half a point from actual Performance IQ calculated in the standard manner using the Block Design subtest (M = 95.9, SD= 18.1). Similarly, Full Scale IQ using DOT scores in place of Block Design

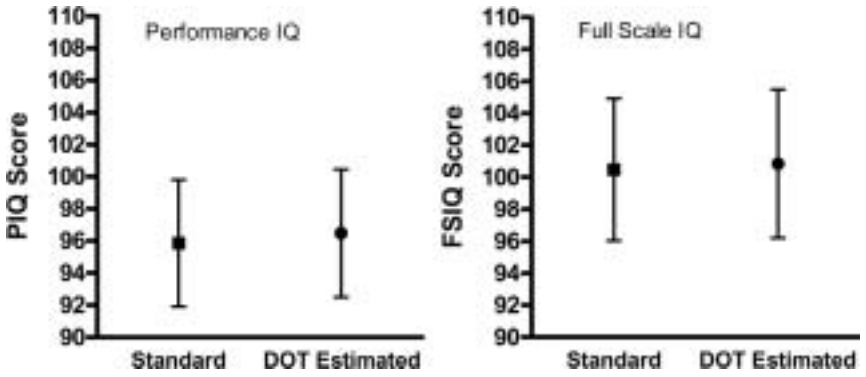

Figure 3. Mean IQ scores obtained by including the Block Design subtest in the standard manner and by substituting with DOT scores. (a) PIQ and (b) FSIQ scores were not significantly changed by substituting DOT estimated scores for Block Design scores.

(M = 100.9, SD = 20.8) was nearly identical to Full Scale IQ calculated in the standard manner (M = 100.5, SD = 20.9; t_{19} = 1.16, ns).

Discussion. The present data support the validity of the DOT by demonstrating significant differences between high functioning college students and a sample of clinical neurologic patients. Concurrent validity was demonstrated by the very high correlation between the DOT and the Block Design subtest in a clinical sample. The DOT also demonstrated clinical utility by yielding nearly identical Performance and Full Scale IQ scores when substituted in place of the Block Design subtest in calculating these indices. Thus, the DOT appears to be a valid and clinically useful instrument for rapidly screening visuospatial ability.

General Discussion and Conclusions

In these two studies we described the development and preliminary validation of the DOT as a screening instrument for rapid assessment of visuospatial abilities. Overall, the DOT demonstrated good alternate forms reliability, effectively discriminated healthy subjects from clinical patients with neurologic complaints, and correlated highly with the Block Design subtest of the WAIS-III in the patient sample. Given that the DOT requires only two minutes of administration time, it can provide useful information about visuospatial ability in screening situations or when a clinician is faced with time constraints that prevent the administration of more extensive tests or batteries. In some situations, the DOT may serve as a preliminary visuospatial screen that can alert the examiner to the need for a more extensive evaluation with additional tests.

Estimation of reliability is critical in the development of a new instrument, but for timed tests such as the DOT, commonly used estimates of internal consistency such as coefficient alpha, are not appropriate (Nunnaly, 1978). In such cases, reliability is often estimated via the administration of alternate forms of the test during the same occasion or via repeated administrations of the same test on two different occasions. In Study 1, two forms of the DOT were developed and administered during the same session and were significantly correlated (r = .80) in a sample of high functioning college students. The alternate forms reliability of the DOT was comparable to the test-retest reliability reported for the Block Design subtest of the WAIS-III (r = .82) (Wechsler, 1997). Furthermore, there was no significant difference between the scores obtained on Form A or Form B, suggesting that they provide essentially the same information. Although the alternate forms reliability of the DOT was good, it may have underestimated the true reliability in the normal population due to the atypical nature of the student sample in Study 1. Participants were drawn from a highly selective private university with extremely competitive admissions standards, suggesting that the sample was biased toward the high end of the cognitive ability distribution. This homogeneous sample may have contributed to a lower correlation between the two forms by restricting the range of scores and by inflating the ceiling effect, as evidenced by the fact that 10.5% of these bright, well-educated participants achieved the maximum score on both administrations of the DOT. By contrast, the scores from the clinical sample in Study 2 were more evenly distributed and none of the neurologic patients achieved the maximum score on the DOT. These findings suggest that the correlation between the alternate forms in Study 1 represents a lower bound on the reliability estimate for the DOT.

A test cannot be valid if the data it provides are not reliable. Thus, another estimate of the lower bounds of reliability can be gained from the correlation between a new test and

another test designed to measure the same theoretical construct (Nunnaly, 1978). In Study 2, we found that the scores of neurologic patients on the DOT were highly correlated (r = .92) with their scores on the Block Design subtest of the WAIS-III. The magnitude of this correlation suggests that the DOT measures abilities similar to those assessed by the Block Design test, supporting its validity as a measure of visuospatial ability and suggesting that the true reliability of the instrument should be at least as high as the obtained validity coefficient. Discriminant validity was also demonstrated in the clinical sample by the nonsignificant correlations with tests measuring mood-related constructs, such as depression and anxiety, which should be unrelated to visuospatial ability. Finally, the present results supported the clinical usefulness of the DOT as a potential alternative for Block Design in screening situations or when other factors limit the time or resources available for a comprehensive assessment. When DOT performance was used to replace Block Design raw scores in the calculation of Performance and Full Scale IQ there was no significant change in either of these IQ scores.

While these preliminary findings support the reliability, validity, and clinical usefulness of the DOT, there are a number of methodological limitations that require consideration. As mentioned above, the initial reliability study was conducted on a college student sample from a highly selective university and is not representative of the general population. Further development will be necessary with more diverse samples that include subjects from a broad range of cognitive ability and education level. Furthermore, the present validity analysis was limited to the sample of patients presenting with neurological complaints. Future research will need to evaluate the correlation between the DOT and visuospatial performance measures such as Block Design in a non-clinical population and with a wider range of cognitive ability instruments. Future studies will be needed to develop an adequate normative base and to evaluate the potential of the DOT to be completed with only verbal responses, removing the manual motor component completely. With the aforementioned limitations in mind, the present study can serve as a basis for further development and validation of the DOT as an alternative visuospatial assessment instrument that can be used in situations where patients have limited motor capacity, when a full assessment is not warranted, or when time constraints limit the amount of testing that can be performed.

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ORIGINAL ARTICLE

Reduced gray matter volume in the anterior cingulate, orbitofrontal cortex and thalamus as a function of mild depressive symptoms: a voxel-based morphometric analysis

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Background. Studies investigating structural brain abnormalities in depression have typically employed a categorical rather than dimensional approach to depression [i.e. comparing subjects with Diagnostic and Statistical Manual of Mental Disorders (DSM)-defined major depressive disorder (MDD) *v.* healthy controls]. The National Institute of Mental Health, through their Research Domain Criteria initiative, has encouraged a dimensional approach to the study of psychopathology as opposed to an over-reliance on categorical (e.g. DSM-based) diagnostic approaches. Moreover, subthreshold levels of depressive symptoms (i.e. severity levels below DSM criteria) have been found to be associated with a range of negative outcomes, yet have been relatively neglected in neuroimaging research.

Method. To examine the extent to which depressive symptoms – even at subclinical levels – are linearly related to gray matter volume reductions in theoretically important brain regions, we employed whole-brain voxel-based morphometry in a sample of 54 participants.

Results. The severity of mild depressive symptoms, even in a subclinical population, was associated with reduced gray matter volume in the orbitofrontal cortex, anterior cingulate, thalamus, superior temporal gyrus/temporal pole and superior frontal gyrus. A conjunction analysis revealed concordance across two separate measures of depression.

Conclusions. Reduced gray matter volume in theoretically important brain regions can be observed even in a sample that does not meet DSM criteria for MDD, but who nevertheless report relatively elevated levels of depressive symptoms. Overall, these findings highlight the need for additional research using dimensional conceptual and analytic approaches, as well as further investigation of subclinical populations.

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Key words: Depressive symptoms, DSM, gray matter volume, research domain criteria, voxel-based morphometry.

Introduction

A large body of research has explored functional and structural brain abnormalities associated with major depressive disorder (MDD). Most previous studies examining structural abnormalities in depression have utilized a region-of-interest (ROI) rather than a whole-brain approach. Findings from such studies may be biased towards investigating only a subset of brain regions, such as those that are more easily defined via anatomical scans or those of primary theoretical interest (e.g. amygdala and hippocampus; Koolschijn *et al.* 2009; Bora *et al.* 2012). An important benefit of voxel-based morphometry (VBM) is that it allows for a more comprehensive whole-brain assessment of structural abnormalities without the *a priori* selection of ROIs (Ashburner & Friston, 2000). Structural magnetic resonance imaging (MRI) studies have the additional benefit of facilitating comparison across studies, as they are paradigm-free, unlike functional MRI studies in which cross-study comparisons may be obscured due to differences in the paradigms employed.

A growing number of VBM studies of MDD have been conducted, allowing for the aggregation of findings using meta-analytic methods. Across three separate and recent meta-analyses of VBM studies, the most consistent finding reported was that subjects with MDD had reduced gray matter volume in the anterior cingulate cortex (ACC) relative to those without MDD (Bora *et al.* 2012; Du *et al.* 2012; Lai, 2013; for examples of individual studies, see Tang *et al.* 2007; Treadway *et al.* 2009). The ACC is a particularly large and

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heterogeneous structure in terms of function, receptor architecture and cytology (Vogt *et al.* 2005; Palomero-Gallagher *et al.* 2008; Pizzagalli, 2011), and has been subdivided into a more dorsal portion (dorsal ACC; dACC) and a more ventral subregion, the latter of which has been further divided into the rostral ACC (rACC; also referred to as the 'pregenual' or 'perigenual' ACC) and the subgenual ACC (sgACC; Etkin *et al.* 2011). Broadly speaking, a distinction may be made between the more 'cognitive' dACC *versus* the more 'affective' ventral subdivision encompassing the rACC and sgACC. Indeed, the ventral portion of the ACC has rich interconnections with limbic and paralimbic structures [e.g. amygdala, nucleus accumbens, orbitofrontal cortex (OFC), periaqueductal gray], whereas the dACC has strong connections with regions in the dorsolateral prefrontal cortex (DLPFC), parietal cortex and supplementary motor areas. Consistent with these differential connectivity patterns, the ventral ACC has been implicated in emotion expression and regulation, as well as assigning emotional valence to both internal and external stimuli. In contrast, the dACC has been associated with the processing of cognitively demanding information and response selection (Etkin *et al.* 2011; Pizzagalli, 2011).

Given these patterns of findings, it is interesting to note that gray matter volume reductions in the ACC in MDD subjects seem to be largely concentrated in a relatively focal region of the rACC (Bora *et al.* 2012), a core hub in Mayberg's limbic–cortical dysregulation model of depression (Mayberg, 1997). The structural deficit in the rACC observed in MDD is intriguing given that this region has been implicated in cognitive and emotional processes that are believed to be abnormal in many depressed individuals. These include: (1) emotion regulation and assigning emotional valence to stimuli (Pizzagalli, 2011; D'Avanzato *et al.* 2013); (2) self-referential processing (Northoff *et al.* 2006; Yoshimura *et al.* 2009); (3) inhibitory processes (e.g. Bush *et al.* 2000; Shafritz *et al.* 2006; Eugene *et al.* 2010); (4) error processing (e.g. Holmes *et al.* 2010; Santesso *et al.* 2012); and (5) optimistic biases (i.e. relatively high probability estimates of positive events occurring to oneself compared with others; Sharot *et al.* 2007; Blair *et al.* 2013). Indeed, hyper-responsiveness of this region has been shown to correlate with subclinical depressive mood in adolescent children (Killgore & Yurgelun-Todd, 2006). It is important to note that in addition to ACC abnormalities, other areas found to have reduced gray matter volume within MDD subjects in the above-mentioned meta-analyses include the middle and inferior frontal gyrus, hippocampus and thalamus (Kim *et al.* 2008; Vasic *et al.* 2008; Zou *et al.* 2010; Wagner *et al.* 2011; Du *et al.* 2012), as well as the DLPFC and dorsomedial prefrontal cortex (Bora *et al.* 2012). Given that the thalamus connects the cortex to negative emotion-generating limbic structures such as the amygdala (Price & Drevets, 2009), relatively reduced thalamic gray matter volume may help account for deficits in top-down regulation of negative affect among individuals more prone to experiencing depressive symptoms. Moreover, reduced hippocampal volume in depression may be attributable to stress-related or neurotoxic processes associated with cumulative exposure to stress and depressive symptomatology (MacQueen *et al.* 2003; Warner-Schmidt & Duman, 2006).

As is the case with the bulk of functional MRI research, studies investigating structural brain abnormalities in depression have typically employed a categorical rather than dimensional approach to conceptualizing depression. Namely, studies have usually compared a group of subjects diagnosed with MDD, as defined by current Diagnostic and Statistical Manual of Mental Disorders (DSM) criteria, with a group of individuals who do not meet MDD diagnostic criteria [also see the International Classification of Diseases (ICD) diagnostic system; World Health Organization, 1993]. The extent to which depressive symptoms – irrespective of DSM-defined MDD status – are linearly related to gray matter volume reductions in theoretically important brain regions (e.g. the ACC) is not well understood. Indeed, the National Institute of Mental Health (NIMH), through their Research Domain Criteria (RDoC) initiative, has encouraged a dimensional approach to the study of psychopathology as opposed to an over-reliance on traditional diagnostic categories (e.g. see NIMH funding opportunity 'Dimensional Approaches to Research Classification in Psychiatric Disorders' no. RFA-MH-13-080). Previous studies comparing dimensional *versus* categorical models of depression, including findings from taxometric analyses, have tended to support dimensional conceptualizations of depressive symptoms (Prisciandaro & Roberts, 2009), and it is likely that such approaches may ultimately lead to a more valid understanding of the underlying neurobiological basis of depression than research focused on categorical diagnostic distinctions.

It is also important to note that subthreshold levels of depressive symptoms (i.e. severity levels below DSM criteria) have been found to be associated with a range of negative outcomes (Cuijpers *et al.* 2004; Fergusson *et al.* 2005), including a significantly increased risk of developing MDD (Klein *et al.* 2013). These findings highlight the importance of investigating the neurobiological underpinnings of subclinical levels of depressive symptoms. A few previous studies have explored structural abnormalities in subclinical depression, but these studies differed widely in study

samples (e.g. youth *versus* the elderly), analytic strategy (ROI *versus* whole-brain approach, group comparisons *versus* dimensional analyses) and depression measures employed. For example, in a ROI-based study focused on rACC volumes in children and adolescents (aged 7–17 years) with no history of MDD or psychiatric disorder, Boes *et al.* (2008) found that males with depressive symptoms had lower rACC volumes bilaterally than males without depressive symptoms. This pattern was not observed in females. In addition, in a recent VBM study comparing adults with elevated, but subthreshold, levels of depressive symptoms *versus* those with no depressive symptoms, Hayakawa *et al.* (2013) reported that subclinically depressed females had smaller gray matter volume in the bilateral ACC and in the right rectal gyrus. The pattern was not observed in males. There has also been a series of studies examining structural abnormalities in elderly populations with subthreshold depression (e.g. Taki *et al.* 2005; Dotson *et al.* 2009; Ries *et al.* 2009), with mixed findings regarding the ACC (Ries *et al.* 2009). None of the above-mentioned studies employed the commonly used Beck Depression Inventory (BDI; Beck & Steer, 1993; Beck *et al.* 1996).

In line with the NIMH's RDoC initiative, the current study examines – in a dimensional rather than categorical manner – the association between gray matter volume and depressive symptoms in a non-clinical sample. We employed a whole-brain VBM regression approach with a sample of adults (aged 18–45 years) deemed free of any history of DSM-IV Axis I diagnoses. Importantly, to examine the consistency of any emerging findings, two separate instruments were used to assess depressive symptoms, including the commonly used BDI and the Depression subscale of the Personality Assessment Inventory (PAI-DEP; Morey, 1991). The PAI-DEP was selected in addition to the BDI for several reasons. First, the BDI, like the Hamilton Rating Scale for Depression (Hamilton, 1960), tends to exhibit very low mean scores and little variability in healthy samples. In contrast, the PAI-DEP includes items intended to assess the full range of depressive symptoms and with greater variability of scores across non-clinical samples (Morey, 2007). Moreover, whereas the BDI has a relatively heavy representation of cognitive aspects of depression, the PAI-DEP was designed to tap cognitive, affective and physiological symptoms of depression with generally equal weighting across these three domains.

Informed by the literature discussed above, we hypothesized that the severity of mild depressive symptoms, even in a healthy subclinical population, would be associated with reduced gray matter volume in the rACC, hippocampus, thalamus, as well as the DLPFC and dorsomedial prefrontal cortex.

Method

Participants

A total of 54 healthy participants (28 females), aged 18 to 45 years (mean=30.9 years, S.D.=8.13 years), were recruited from the greater Boston region through flyers posted in the area and from Internet advertisements. The sample was 69.1% Caucasian, 16.4% African–American, 9.1% Asian, 1.8% other, and 3.6% 'more than one race'. In addition, 3.6% classified themselves as Hispanic. All participants identified English as their primary language. A trained Bachelor's-level technician screened all participants for evidence of mental disorders and medical conditions using a structured series of questions adapted from the Structured Clinical Interview for DSM-IV, text revision (SCID-I; First *et al.* 2001). All participants included in the current study were determined to be free of any history of DSM-IV Axis I mental disorders, excessive substance use, drug or alcohol treatment, or severe medical or neurological conditions. The current study was embedded within a larger study on the neural correlates of emotional intelligence in which subjects completed a battery of self-report measures and behavioral tasks. Unrelated findings emerging from that larger study have been reported elsewhere (Killgore *et al.* 2012*a*, *b*, 2013; Webb *et al.* 2013) and will not be discussed further here. To reduce subject burden, self-report measures (including the BDI and PAI) were administered 1 day after the MRI scan.

BDI

The BDI (Beck & Steer, 1993; Beck *et al.* 1996) is a commonly used 21-item self-report measure of depressive symptoms with strong psychometric properties (Beck *et al.* 1988). The BDI has been shown to have acceptably high internal consistency reliability in psychiatric (α=0.86; Beck *et al.* 1988) and non-clinical (α=0.88; Killgore, 1999) samples.

Personality Assessment Inventory (PAI)

The PAI personality assessment (Morey, 1991) contains 344 statements that are rated using one of four response options ('false, not at all true', 'slightly true', 'mainly true', 'very true'). The measure yields 11 clinical subscales (Somatic Complaints, Anxiety, Anxiety-Related Disorders, Depression, Mania, Paranoia, Schizophrenia, Borderline Features, Antisocial features, Drug-Related Problems, Alcohol-Related Problems). Given the current study's focus on depressive symptoms, we restricted our analyses to the Depression subscale of the PAI (PAI-DEP). Raw scores were converted into T scores. The PAI-DEP scale shows high internal consistency reliability for clinical (α=0.87) and

non-clinical (α=0.93) samples (Morey, 2007). The use of the PAI, in addition to the BDI, allows us to test the consistency of our findings across two measures of depression, which sample somewhat different aspects of depression (i.e. whereas the BDI weights cognitive items more heavily, the PAI is designed to tap cognitive, affective and physiological symptoms of depression with generally equal weighting across these three domains).

MRI parameters

Structural magnetic resonance images were acquired at 3.0 T using a 12-channel head coil (Siemens Tim Trio; Germany) and a T1-weighted three-dimensional magnetization-prepared rapid acquisition with gradient echo (MPRAGE) sequence using the following parameters: repetition time=2.1 s; echo time=2.25 ms; flip angle=12°; 128 sagittal slices; 256×256 matrix; in-plane resolution=1×1×1 mm; slice thickness=1.33 mm.

VBM

Preprocessing of structural images was conducted using the VBM8 toolbox (http://dbm.neuro.uni-jena.de/vbm.html) in SPM8 (Wellcome Department of Imaging Neuroscience Group, UK; http://www.fil.ion.ucl.ac.uk/spm). The VBM8 default settings were used (i.e. modulated VBM – gray matter volume was corrected for total intracranial volume). Each structural image was DARTEL-normalized (diffeomorphic anatomical registration through exponentiated lie algebra) to Montreal Neurological Institute (MNI) space and subsequently segmented into gray matter, white matter and cerebrospinal fluid using a fully automated algorithm within SPM8. Standard quality checks of segmentation and sample homogeneity were conducted, before normalized gray matter images were smoothed with an 8 mm full-width at half-maximum (FWHM) Gaussian kernel. In other words, the data were preprocessed such that the dependent variable of interest was the local concentration of gray matter at each local voxel without concern for differences in morphological shape (Ashburner & Friston, 2000).

Statistical analysis

Scores on both depression scales were initially tested for normality. To assess the severity of skew and kurtosis, *z* scores were computed (Field, 2009*a*). Consistent with prior work examining the distribution of BDI scores in the population (Veerman *et al.* 2009), scores in the present sample were positively skewed (z=4.10, p<0.001; Kline, 2005; Field, 2009*b*). Accordingly, a square-root transformation was applied to reduce the positive skew (transformed BDI, z=0.861, p>0.05). For simplicity, the term BDI is used henceforth, rather than repeating the more accurate but unwieldy term 'square-root transformed BDI'.

Normalized smoothed gray matter images were entered into a series of three random-effects multiple regression analyses in SPM8. A non-stationary cluster extent correction was implement in SPM8 according to the random field theory (RFT) framework (Hayasaka *et al.* 2004). In the first regression, we tested the association between BDI scores and voxelwise gray matter volume. Normalized smoothed gray matter images were entered as the dependent variable into a whole-brain general linear model. For statistical inference, a whole-brain false discovery rate (FDR)-corrected height threshold of p<0.05 was applied. A cluster extent threshold of $k \geqslant 81$ (i.e. determined statistically as the number of voxels per cluster that would be expected by chance based on the theory of Gaussian random fields applied to this analysis as provided in standard VBM8 output) was also applied for the purpose of reducing noise in the images and to minimize reporting of clusters of little interest, but was not considered for statistical inference. For the second regression, we conducted a similar analysis with PAI-DEP as the covariate of interest (height threshold of FDR p<0.05 for statistical inference, with an empirically derived cluster extent of $k \geqslant 65$). Finally, to examine the overlap in gray matter volume correlations across the BDI and PAI-DEP analyses, the activation clusters obtained from the first regression analysis (i.e. BDI) were used as a region of interest (ROI) mask in conjunction with the output from the second regression (i.e. PAI-DEP; height threshold FDR p<0.05, cluster extent $k \geqslant 65$). Thus, the resulting findings from the latter analysis reflect overlapping regions where gray matter volume correlated significantly with self-reported depression symptoms on both the BDI and PAI-DEP scales. Age and gender were included as nuisance covariates in all analyses.

Results

The mean raw BDI score was 3.89 (S.D.=4.56, range=0–17), with seven participants scoring above the suggested clinical criterion for 'minimal depression' (i.e. total BDI score >9; Beck *et al.* 1996). The mean PAI-DEP score was 46.20 (S.D.=7.97, range=36–71)[1]†. The Pearson correlation between the BDI and PAI-DEP in the current sample was r=0.67 (p<0.001).

As detailed in Table 1, relatively higher BDI scores were associated with reduced gray matter volume in

† The notes appear after the main text.

Table 1. *Brain regions with reduced gray matter volume associated with higher Beck Depression Inventory scores*

Region	Cluster size	Peak-level MNI coordinates[a]			T
		x	y	z	
Bilateral anterior cingulate	1669	6	46	10	4.33*
Right superior temporal/insula/Heschl's gyrus	737	51	−9	4	4.35*
Bilateral dorsal anterior cingulate/mid-cingulate	598	0	20	28	4.62†
Left superior temporal gyrus	562	−51	6	−6	4.33†
Right middle frontal gyrus	549	44	21	46	4.34*
Left middle cingulate gyrus	346	−8	−33	49	4.30
Right superior temporal pole/insula	297	48	15	−9	4.92†
Left inferior frontal operculum	260	−52	14	15	4.20
Left postcentral gyrus	190	−50	−18	43	4.16
Left superior temporal pole	168	−32	14	−30	4.38
Right lingual gyrus	135	8	−61	0	4.03
Left thalamus	122	−8	−18	9	3.59
Right precentral gyrus	101	52	−9	40	3.78
Right middle temporal gyrus	101	68	−18	−3	3.96
Left insula	84	−33	18	4	3.71
Left middle occipital gyrus	83	−44	−81	0	4.16

MNI, Montreal Neurological Institute.

[a] All false discovery rate-corrected $p<0.05$; $k \geqslant 81$.

* $p<0.05$, † $p<0.10$ cluster level, family-wise error corrected.

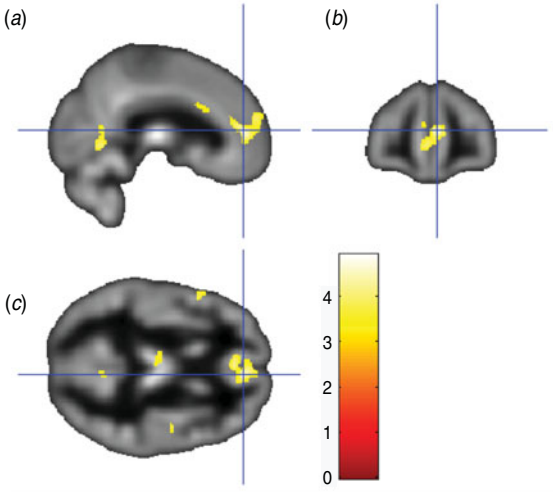

Fig. 1. Sagittal view (*a*), coronal view (*b*) and axial view (*c*) of the anterior cingulate/medial orbitofrontal cortex cluster (overlaid on sample-specific T1 mean image) that was negatively correlated with depressive symptoms on the Beck Depression Inventory. Montreal Neurological Institute (MNI) coordinates x=6, y=46, z=10; cluster size=1669 voxels.

16 clusters, including: (i) bilateral anterior cingulate, bilateral medial frontal cortex and left medial OFC (see Fig. 1); (ii) bilateral anterior/mid-cingulate; (iii) left thalamus; and (iv) left insula. As shown in Table 2, relatively higher PAI-DEP scores were associated with reduced gray matter volume in nine clusters, including: (i) left anterior cingulate and left medial OFC (see Fig. 2); (ii) bilateral medial OFC; and (iii) bilateral thalamus.

The conjunction between the two primary analyses was used to identify the regions showing common overlap between the gray matter volume correlations for the two depression measures used in the current study. As shown in Table 3, this analysis showed that the BDI and PAI-DEP scores were both associated with reduced gray matter volume in four common regions: (i) left medial OFC and anterior cingulate (Fig. 3); (ii) left thalamus (Fig. 4); (iii) right superior medial frontal gyrus/superior frontal gyrus; and (iv) right superior temporal gyrus extending to the superior temporal pole.

Discussion

The present study complements prior research on structural brain abnormalities in depression. Specifically, previous studies examining structural deficits in depression have typically relied on categorical comparisons of subjects diagnosed with MDD *versus* healthy controls. The extent to which depressive symptoms in individuals who do not meet DSM criteria for MDD are linearly related to structural differences in key brain regions is not well understood. Inspired by the NIMH's RDoC initiative, which highlights

Table 2. *Brain regions with reduced gray matter volume associated with higher scores on the Depression subscale of the Personality Assessment Inventory*

Region	Cluster size	Peak-level MNI coordinates[a]			T
		x	y	z	
Right cerebellum	747	24	−78	−27	4.58
Right superior temporal gyrus/temporal pole	660	62	4	−2	5.13*
Bilateral thalamus	395	0	−19	10	4.47
Left superior orbitofrontal gyrus	248	−9	28	−24	4.56
Right superior frontal gyrus	213	14	56	25	4.21
Left medial OFC/ACC	209	−9	51	−5	4.68
Left cerebellum	163	−15	−55	−60	3.79
Bilateral caudate	96	0	10	1	4.61
Bilateral medial OFC	83	0	62	−12	4.32

MNI, Montreal Neurological Institute; OFC, orbitofrontal cortex; ACC, anterior cingulate cortex.
[a] All false discovery rate-corrected $p<0.05$; $k \geqslant 65$.
* $p<0.05$, † $p<0.10$ cluster level, family-wise error corrected.

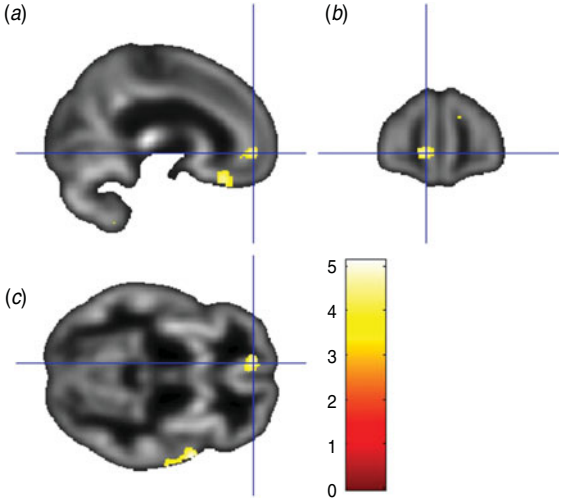

Fig. 2. Sagittal view (*a*), coronal view (*b*) and axial view (*c*) of the left anterior cingulate/medial orbitofrontal cortex cluster (overlaid on sample-specific T1 mean image) that was negatively correlated with depressive symptoms on the Depression subscale of the Personality Assessment Inventory. Montreal Neurological Institute (MNI) coordinates x=−9, y=51, z=−5; cluster size=209 voxels.

dimensional/continuous – rather than DSM-based categorical – conceptualization of psychopathology, we examined the linear associations between gray matter volume and depressive symptoms in a sample of adults deemed free of any history of DSM-IV Axis I diagnoses. Paralleling findings from prior studies employing a categorical (i.e. MDD *versus* healthy controls) analytic approach, we found that relatively higher depressive symptoms were associated with reduced gray matter volume in the left rACC (extending into the medial OFC). Perhaps the most consistently reported finding in the existing literature is that subjects with MDD have reduced gray matter volume in the ACC relative to those without MDD (for recent meta-analytic reviews, see Bora *et al.* 2012; Du *et al.* 2012; Lai, 2013). The current findings extend prior work by suggesting that reduced ACC/OFC gray matter volume can be observed even in a sample that does not meet DSM criteria for MDD, but who nevertheless report relatively elevated levels of depressive symptoms. In addition, we also observed an association between relatively elevated depressive symptoms and reduced gray matter volume in the left thalamus, as well as the right superior medial frontal gyrus/superior frontal gyrus, and right superior temporal gyrus extending to the superior temporal pole. The fact that this finding emerged across both of our depressive symptom measures, including the commonly used BDI, as well as the depression subscale of the PAI (PAI-DEP), suggests that these findings probably reflect true and robust associations between gray matter and depressive symptoms within the subclinical range.

Regarding our ACC finding, it is interesting to note that gray matter volume reductions were concentrated in the rACC. The rACC has been implicated in a range of cognitive/affective functions, including: (1) optimistic biases (e.g. Blair *et al.* 2013); (2) error processing (e.g. Holmes *et al.* 2010; Santesso *et al.* 2012); (3) self-referential processing (Northoff *et al.* 2006; Yoshimura *et al.* 2009); (4) inhibitory processes (e.g. Bush *et al.* 2000; Shafritz *et al.* 2006; Eugene *et al.* 2010); and (5) regulation of emotional conflict and dampening of amygdala hyperactivity (Etkin *et al.* 2006). Given these patterns of findings, one could speculate that

Table 3. *Brain regions with reduced gray matter volume associated with higher scores on both the Beck Depression Inventory and the Depression subscale of the Personality Assessment Inventory*

Region	Cluster size	Peak-level MNI coordinates[a]			T
		x	y	z	
Right superior temporal gyrus/temporal pole	251	62	4	−2	5.13*
Right superior frontal gyrus	153	14	56	25	4.21*
Left medial OFC/ACC	116	−9	51	−5	4.68*
Left thalamus	79	−2	−19	10	4.39†

MNI, Montreal Neurological Institute; OFC, orbitofrontal cortex; ACC, anterior cingulate cortex.
[a] All false discovery rate-corrected $p<0.05$; $k \geqslant 65$.
* $p<0.05$, † $p<0.10$ cluster level, family-wise error corrected.

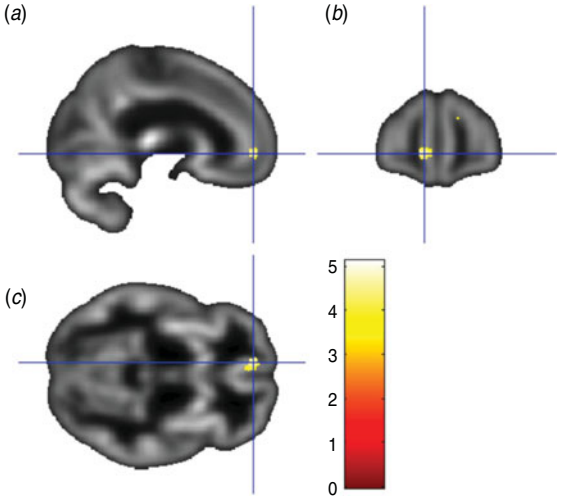

Fig. 3. Sagittal view (*a*), coronal view (*b*) and axial view (*c*) of the results of a conjunction analysis showing a cluster where gray matter volume in the left rostral anterior cingulate/medial orbitofrontal cortex (overlaid on sample-specific T1 mean image) was negatively correlated with depressive symptoms on both the Beck Depression Inventory and Depression subscale of the Personality Assessment Inventory. Montreal Neurological Institute (MNI) coordinates x=−9, y=51, z=−5; cluster size=116 voxels.

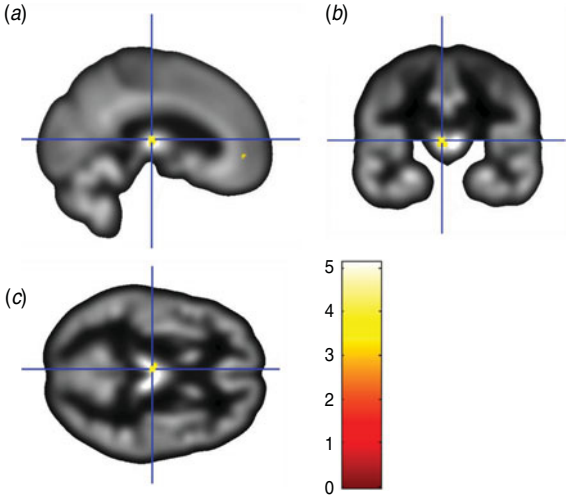

Fig. 4. Sagittal view (*a*), coronal view (*b*) and axial view (*c*) of the results of a conjunction analysis showing a cluster where gray matter volume in the left thalamus (overlaid on sample-specific T1 mean image) was negatively correlated with depressive symptoms on both the Beck Depression Inventory and Depression subscale of the Personality Assessment Inventory. Montreal Neurological Institute (MNI) coordinates x=−2, y=19, z=10; cluster size=79 voxels.

structural abnormalities in the rACC in part underlie the biased information processing and negative maladaptive cognitive features described in the cognitive model of depression (Beck & Alford, 2009; DeRubeis *et al.* 2010). More specifically, to the extent that the rACC does play a role in the aforementioned functions, relatively reduced gray matter volume in this region may help account for the depressogenic, cognitive features of depression, including: (1) pessimistic (rather than optimistic) biases (e.g. Strunk *et al.* 2006); (2) hypersensitivity to perceived errors and negative feedback (e.g. Elliott *et al.* 1998; Pizzagalli *et al.* 2006); (3) maladaptive self-referential cognitive processes (e.g. Nolen-Hoeksema, 1991; Nolen-Hoeksema *et al.* 2008; Pizzagalli, 2011); (4) difficulties inhibiting negative information (e.g. Goeleven *et al.* 2006); and (5) emotion regulation deficits (D'Avanzato *et al.* 2013). These abnormal cognitive and emotion regulatory features of depression do not appear to be categorical in nature (i.e. present in MDD, yet absent in those who do not meet DSM criteria for the disorder). Rather, behavioral and neural findings suggest that at least some of these features are linearly related to depressive symptoms across a broad range of severity, from no or very low levels of depressive symptoms to very high levels

(e.g. Joormann, 2004; Pizzagalli *et al.* 2006; Strunk *et al.* 2006). Overall, and consistent with the NIMH's RDoC initiative, these findings may highlight the limitations of restricting the study of abnormal cognitive, emotional and behavioral processes in depression to DSM-based categorical comparisons, and the need for additional research using dimensional/continuous conceptual and analytic approaches.

Although perhaps not as consistently reported as rACC findings, prior studies have also identified gray matter reductions in the thalamus among MDD subjects (Bielau *et al.* 2005; Kim *et al.* 2008; Vasic *et al.* 2008; Lee *et al.* 2011). The current study suggests that reductions in thalamic gray matter volume can also be observed in individuals with subclinical, yet relatively elevated, levels of depressive symptoms. The latter finding is intriguing given that the thalamus connects the cortex to negative emotion-generating limbic structures such as the amygdala (Price & Drevets, 2009). Thus, relatively reduced thalamic gray matter volume, coupled with volumetric reductions in the rACC, may also help account for the deficits in top-down regulation of negative emotions among individuals more prone to experiencing depressive symptoms.

In addition, previous depression studies have also reported volume reductions in the superior temporal gyrus/temporal pole (e.g. Caetano *et al.* 2004; Abe *et al.* 2010) and superior frontal gyrus (e.g. Serra-Blasco *et al.* 2013). Interestingly, Caetano *et al.* (2004) found that right superior temporal gyrus volume was inversely correlated with length of depression illness. Similarly, the findings of Serra-Blasco *et al.* (2013) revealed that participants who suffered from chronic, treatment-resistant depression exhibited the smallest volumes in frontotemporal regions. It is tempting to attribute such volumetric reductions to neurotoxic effects of stress and depression accumulating over time. However, conclusions regarding the direction of effects are necessarily tempered when cross-sectional designs are employed.

It may be that reduced gray matter volume in the regions highlighted in the current study (e.g. rACC, thalamus) increase the risk of experiencing depressive symptoms. Conversely, these structural differences may also reflect the neurotoxic effects of prior depressive symptoms and stress. It should be noted that we intentionally restricted our sample to those without prior episodes of depression. Nevertheless, reduced gray matter volume could of course still be due to cumulative exposure to subthreshold depressive symptoms and neurotoxic stress processes. Methodologically sound longitudinal studies are needed to disentangle causal direction and test whether or not reduced gray matter volume in certain brain regions (e.g. rACC) prospectively predicts depression onset, and/or whether structural abnormalities are a consequence of depression due to particular neuropathological processes associated with the disorder. Longitudinal studies could help delineate factors that predict the development of these structural brain abnormalities over time. For example, prior research has found that smaller ACC volumes are associated with retrospective reports of early life stressors (Cohen *et al.* 2006). This finding highlights the possible neurotoxic effects of early life stress on the developing brain. Ideally, however, given that current symptomatology may bias recall of early life stressors (e.g. Southwick *et al.* 1997), prospective longitudinal, rather than retrospective studies would be needed to more conclusively establish the link between early life stress and structural brain abnormalities, ideally complementing self-report measures of stressors with more 'objective' measures (e.g. assessments of stress hormones).

A strength of the current study is that we tested the consistency of our findings across two separate measures of depressive symptoms. In addition, in contrast to many prior studies, we used a whole-brain VBM approach, rather than an ROI approach, the latter of which may bias investigators towards examining only a subset of brain regions and possibly ignoring other relevant regions (Ashburner & Friston, 2000). However, despite these strengths, several limitations of the current study should be noted. As discussed above, our findings were cross-sectional in nature and we cannot draw strong inferences regarding causal direction between structural brain differences and depressive symptoms. Moreover, our study focused on a sample without a history of Axis I pathology. It will be important for future studies to examine the full continuum from no depression to very severe levels of depressive symptoms, to fully examine the extent to which depressive symptoms are linearly related with structural brain differences. In addition, given our non-clinical sample, variability in depression scores was, as expected, limited. Thus, restriction in the range of depression scores may have limited our ability to detect significant effects in other brain regions. It should be noted that the current findings indicate that we did have sufficient variability to detect reduced gray matter volume in a number of regions, across two separate depression measures.

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The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

Declaration of Interest

None.

Note

[1] Two outliers were identified. Specifically, one subject was an outlier on both the BDI (z = 4.68) and PAI Depression subscale (z = 4.00). This subject's data were deleted from the current dataset.

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Daytime Sleepiness Is Associated With Reduced Integration of Temporally Distant Outcomes on the Iowa Gambling Task

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Sleep deprivation is associated with performance decrements on some measures of executive functioning. For instance, sleep deprivation results in altered decision making on the Iowa Gambling Task. However, it is unclear which component processes of the task may be driving the effect. In this study, Iowa Gambling task performance was decomposed using the Expectancy-Valence model. Recent sleep debt and greater daytime sleepiness were associated with higher scores on the updating parameter, which reflects the extent to which recent experiences are emphasized over remote ones. Findings suggest that the effects of insufficient sleep on IGT performance are due to shortening of the time horizon over which decisions are integrated. These findings may have clinical implications in that individuals with sleep problems may not integrate more temporally distant information when making decisions.

Sleep loss is a prevalent and costly public health problem, in part because it contributes to an increased probability of cognitive errors (Daley, Morin, LeBlanc, Grégoire, & Savard, 2009; Hillman, Murphy, & Pezzullo, 2006). While research on the cognitive effects of sleep loss has focused extensively on deficits in basic aspects of alertness, vigilance, and attentional

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processing (Doran, Van Dongen, & Dinges, 2001), deficits in more complex aspects of executive functioning and affective processing also have become of particular interest (Killgore, 2010). Sleep deprivation results in performance decrements on tasks measuring various aspects of executive functioning, including response inhibition (Drummond, Paulus, & Tapert, 2006; Harrison, Jones, & Waterhouse, 2007), working memory (Groeger et al., 2008), resistance to distracting information ("filtering efficiency"; Drummond, Anderson, Straus, Vogel, & Perez, 2012), planning (Killgore, Kahn-Greene, Grugle, Killgore, & Balkin, 2009), and judgment/moral reasoning (Olsen, Pallesen, & Espevik, 2013). While deficits on complex executive functioning and decision-making tasks have been identified (Killgore, Balkin, & Wesensten, 2006; Killgore, Grugle, & Balkin, 2012; Killgore, Lipizzi, Kamimori, & Balkin, 2007), it is unclear whether these are related to specific disturbances in executive functioning or to more basic underlying cognitive processes such as sustained attention that also contribute to overall performance on these tasks (Jackson et al., 2013; Tucker, Whitney, Belenky, Hinson, & Van Dongen, 2010).

Impaired performance in the context of sleep deprivation has been demonstrated using the Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994; Bechara, Damasio, Damasio, & Lee, 1999), which captures the learning of reward-related contingencies in the context of initial uncertainty. Strong performance on the IGT occurs when individuals forego larger short-term gains in favor of longer-term outcomes. Our group has demonstrated that acute sleep deprivation impairs performance on the IGT (Killgore et al., 2006; Killgore, Grugle et al., 2012; Killgore et al., 2007). Other studies have shown that IGT performance is better when tested after sleep than after wakefulness, suggesting that the process of sleep may somehow facilitate the learning of the underlying task contingencies (Pace-Schott, Nave, Morgan, & Spencer, 2012). Impaired IGT performance has been demonstrated in individuals with sleep disorders, including narcolepsy-cataplexy (Bayard, Abril, Yu, & Scholz, 2011), REM sleep behavior disorder (Delazer et al., 2012), obstructive sleep apnea (Daurat, Ricarrère, & Tiberge, 2013), and restless legs syndrome (Bayard, Langenier, & Dauvilliers, 2013). Sleep-related deficits on the IGT are not remediated with stimulant medication use (Killgore, Grugle, et al., 2012; Killgore et al., 2007), raising the possibility that they are not attributable to impairment in basic attentional processes. Because the IGT was developed to assess affectively guided decision making, the aforementioned deficits have been interpreted as evidence that sleep loss adversely affects the ability to use affective information to influence choice selection (Killgore et al., 2006; Killgore, Grugle, et al., 2012; Killgore et al., 2007).

In the standard version of the IGT, four decks of cards are presented on a computer screen. Participants select cards one at a time to win as much money as possible. Each card selection results in some monetary gain, and some cards also result in a loss. Two of the decks (i.e., decks C and D) are characterized as "advantageous" because they result in a net gain over time (netting $250 over the course of 10 trials), while decks A and B are "disadvantageous" because they lead to a net loss (−$250). The decks also differ in a variety of other aspects, including loss frequency, gain magnitude, and loss magnitude. "Net score" is typically calculated by subtracting disadvantageous deck choices from advantageous deck choices and examining performance across five 20-trial blocks. Bechara et al. (1994) demonstrated that healthy controls eventually established a preference for advantageous decks C and D during the course of the game, while patients with ventromedial prefrontal cortex lesions continued to prefer decks A and B, where larger short-term gains are available. A large body of subsequent literature has

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demonstrated IGT deficits in individuals with a variety of neurological and psychopathological conditions, including substance dependence (Verdejo-García, Pérez-García, & Bechara, 2006) and pathological gambling (Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2005), among many others.

Though the IGT has shown a robust ability to detect deficits in decision making, critiques have been raised regarding the original analytic strategy used for this task. Notably, recent evidence suggests that healthy participants consider other factors in addition to long-term gains when making decisions on the IGT (Horstmann, Villringer, & Neumann, 2012). For instance, healthy individuals often show a "prominent deck B" phenomenon, where "bad" deck B is selected much more frequently than "bad" deck A, at times reaching levels comparable to deck D (Lin, Song, Lin, & Chiu, 2012). Similarly, there is a "shrunken deck C" phenomenon, where deck D is preferred to deck C even though they have identical long-term outcomes (Chiu & Lin, 2007). These findings support the idea that long-term outcome is not the sole driver of IGT behavior in healthy participants, who are influenced by additional deck features such as reward and punishment frequency.

Acknowledgment of the complex contingencies underlying the IGT has resulted in several attempts to develop cognitive models to decompose IGT performance into underlying components. The most widely adopted has been the Expectancy-Valence (EV) model (Busemeyer & Stout, 2002). The model assumes that after each choice, the participant has an affective reaction in response to the gains and losses. The affective response to each card selection is termed a "valence" and is calculated as a weighted average of the gains and losses, where the weight (i.e., emphasis placed on losses versus gains) is the first parameter (w) estimated for each subject. Values of w range from 0 to 1, with higher values reflecting more attention weight toward losses instead of gains. The second parameter (α) reflects the extent to which recent experiences are emphasized over remote experiences. The expectancies for a given deck are updated each time it is selected, with recently experienced valences receiving more weight than remotely experienced ones. Values of α range from 0 to 1, with higher values reflecting strong recency effects (i.e., limited integration of outcomes that are more distant in time). Finally, the extent to which choices occur randomly versus being driven strictly by deck expectancies is captured by a third parameter (c). The choice made on each trial is a probabilistic function of the four expectancies associated with the four decks, and this parameter reflects the extent of randomness in the decision-making process. Higher (positive) values reflect less randomness. The trial-dependent c parameter was used in this analysis.

The EV model can be used to more clearly delineate the factors underlying decision making on the IGT in relation to clinical conditions and individual differences. The attention weight parameter (w) has been described as a motivational parameter, capturing affective components of the decision-making process. The updating parameter (α) is a cognitive parameter, reflecting memory processes. The sensitivity parameter (c) is a response parameter, capturing the extent of random responding (Busemeyer & Stout, 2002). In an analysis of 10 different clinical populations, distinct patterns of disturbance were evident in these parameters across various groups (Yechiam, Busemeyer, Stout, & Bechara, 2005). For instance, individuals with chronic cannabis abuse had higher attention to recent outcomes versus more distant outcomes (as evidenced by higher values of α), while people with Asperger's Syndrome displayed hyperattention to losses (higher w) and erratic choice consistency (lower c). Thus, poor performance on the IGT relative to healthy controls can occur in relation to a variety of underlying functions; not all individuals

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who perform poorly on the IGT achieve that poor performance in the same way (Yechiam et al., 2008).

The goal of the present study was to better understand the mechanisms by which recent sleep debt and measured daytime sleepiness affect IGT performance in healthy participants by using the EV model to decompose task behavior into cognitive, motivational, and response parameters. Here we examined the association between recent sleep loss, daytime sleepiness, and EV metrics in a cross-sectional sample of healthy individuals. It was hypothesized that recent sleep debt and daytime sleepiness would be associated with decreased attention to losses (lower w), decreased integration of more remote information (higher α), and increased random responding (higher c).

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METHODS

Participants ($n = 55$; 29 female, 26 male) ages 18–45 years were recruited via posted advertisements. Exclusion criteria included self-reported history of serious or chronic medical conditions or of neurological, psychiatric, or substance use disorders on a telephone screen. There were no additional exclusion criteria related to sleep. Participants were right-handed and fluent in English. Demographic variables for the sample are presented in Table 1. Participants reported that their race was White ($n = 38$), African American ($n = 10$), Asian ($n = 3$), other ($n = 2$), and more than one race ($n = 2$). Study procedures were approved by the McLean Hospital IRB and participants provided informed consent in accordance with local IRB requirements.

Participants completed a neurocognitive and emotional task battery over the course of two testing days. On the first day, they completed an MRI scan (reported elsewhere; Killgore, Weber, et al., 2012; Kipman, Weber, Schwab, DelDonno, & Killgore, 2012) and sleep questionnaires. They slept at home that night and were unmonitored. On the second day, they completed the IGT, as well as other tasks and questionnaires. On the second day, the Wechsler Abbreviated Scale of Intelligence (WASI; Pearson, 1999) was administered to yield Full-Scale IQ (FSIQ). Although data from this sample have been reported elsewhere (Webb, DelDonno, & Killgore,

TABLE 1
Demographics

	Whole Sample	*Final Sample*
N	55	32
Sex	29 F: 26 M	19 F: 13 M
Age (years)	30.65 (8.120)	31.03 (8.205)
Age (range)	18–45	20–45
WASI Verbal	110.76 (16.805)	110.25 (17.027)
WASI Perf.	110.05 (16.154)	108.47 (16.347)
WASI FSIQ	108.62 (16.257)	109.34 (16.734)

Note. Values represent means (and standard deviations). WASI = Wechsler Abbreviated Scale of Intelligence.

2014), we have not previously published on relationships between IGT performance and sleep measures in this sample.

Sleep Questionnaires

Participants completed several sleep questionnaires, including the Epworth Sleepiness Scale (ESS; Johns, 1991) as well as an in-house questionnaire regarding sleep habits, including their sleep the night before as well as their typical sleep. The total score on the ESS was used as the measure of daytime sleepiness. In addition, recent sleep debt was calculated by subtracting the hours that the participant reported sleeping prior to the first day of testing from the hours that the participant reported typically sleeping on weeknights (calculated as the difference between reported typical bedtime and wake time; i.e., typical sleep minus recent sleep). Positive values reflect recent sleep debt.

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Iowa Gambling Task (IGT)

On the second day, the computerized IGT was administered (Busemeyer & Stout, 2002; computerized version v. 2.0). Standard IGT instructions were read aloud to participants (Bechara et al., 1999). The standard deck configuration was used. Specifically, decks A and B were disadvantageous and resulted in average gains of $100 per trial, with variable losses on some trials. Deck A had a net loss of $250 over the course of 10 trials, with frequent losses of small magnitude. Deck B had a net loss of $250 over the course of 10 trials, with infrequent losses of large magnitude. Decks C and D were advantageous and resulted in gains of $50 per trial, with variable losses on some trials. Deck C had a net gain of $250 over the course of 10 trials, with frequent losses of small magnitude, while deck D had a net gain of $250 over the course of 10 trials, with infrequent losses of large magnitude. In accordance with standard programming of the task, participants could not select more than 60 out of 100 cards from a single deck. Trial-by-trial data were analyzed in Matlab R2012b using scripts to derive EV parameters provided by Eldad Yechiam (www.technion.ac.il/~yeldad/papers.html). For each participant, α, w, and c were derived using maximum likelihood methods. The variable G^2, reflecting goodness of the model fit over the baseline model, was also calculated (Busemeyer & Stout, 2002; Yechiam et al., 2005). For the baseline model, we employed a statistical model assuming constant choice probabilities across trials where behavior does not change based on trial-by-trial feedback (Busemeyer & Stout, 2002).

Data Analysis

The IGT data were inspected for validity. Cases where a participant maximized a deck by selecting at least 60 cards from one deck were eliminated from further consideration. The EV model was fit to the remaining cases, and individuals with poor model fit also were eliminated. A standard IGT analysis was performed, using a one-way repeated measures ANOVA with block as a within-subjects factor. Pearson correlations were used to examine relationships between standard IGT measures and ESS scores/recent sleep debt. Because the EV parameters were not normally distributed, nonparametric tests (Spearman rank-order correlations) were used to examine relationships between EV parameters and ESS scores.

RESULTS

Sleep Measures

Scores on the ESS ranged from 1 to 16 ($M = 6.19$, $SD = 3.856$); these scores fell roughly in the typical range for healthy adults (Johns, 1991). Recent sleep debt ranged from 3 h of sleep reduction to 1.5 h of sleep gain ($M = 0.84$ hours of sleep debt, $SD = 1.286$). Daytime sleepiness and recent sleep debt were positively but nonsignificantly correlated, Spearman's $r(30) = 0.151$, $p = 0.410$.

Deck Maximization

Thirteen participants (23.6%) "maxed out" a deck by selecting at least 60 cards from the same deck. Deck maximization occurred on decks B ($n = 2$), C ($n = 1$), and D ($n = 10$). Many maximizations occurred within the final 5 trials ($n = 6$), but others occurred in earlier (trials 72, 79). Data collected after deck maximization likely reflects a different set of decision rules than data prior to deck maximization. For this reason, only participants who did not maximize a deck were included for further analysis. Deck maximizers did not significantly differ from nondeck maximizers in ESS scores, $t(39) = -0.626$, $p = 0.535$, or recent sleep loss, $t(39) = -0.312$, $p = 0.757$.

Expectancy Valence Model

Of the 42 remaining subjects, 9 had a poor fit of the EV model to their IGT data (defined as $G^2 < 0$ when the EV model is compared to the baseline model). Poor fit was not associated with age, sex, or FSIQ. Subjects with bad fit did not significantly differ from subjects with good fit in ESS scores, $t(39) = -0.626$, $p = 0.535$, or recent sleep debt, $t(39) = -0.312$, $p = 0.757$. Data from these participants were eliminated. One participant did not fully complete the sleep questionnaire and was also excluded. Thus, $N = 32$ participants were included in the final analyses.

Standard IGT Analysis

As expected, net good minus bad scores increased across blocks (Table 2). A repeated-measures ANOVA was conducted to examine change in net score across blocks (5 levels). Mauchly's W was significant, $\chi^2(9) = 23.390$, $p = 0.005$, indicating a violation of the assumption of sphericity. Therefore, the Greenhouse-Geisser correction was used. There was a significant main effect of block, $F(2.96, 91.88) = 11.72$, $p < 0.001$, reflecting the fact that performance improved over the course of the task. Bonferroni-corrected post hoc tests indicated that scores were significantly lower in the first block than in all subsequent blocks.

Standard IGT Analysis: Associations With Daytime Sleepiness and Recent Sleep Debt

ESS scores were not significantly correlated with overall net score, $r(30) = -0.148$, $p = 0.419$, or performance in any of the five blocks. Similarly, recent sleep debt was not significantly

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TABLE 2
Standard IGT Analysis: Net Score (Good Minus Bad Deck Choices) by Block

	Block 1	*Block 2*	*Block 3*	*Block 4*	*Block 5*	*Total Task*
$N = 32$	−4.88 (9.129)	3.25 (6.520)	5.75 (9.821)	6.63 (9.791)	7.25 (10.470)	18.00 (28.041)
Lower ESS scorers, $N = 17$*	−6.00 (6.205)	4.82 (7.485)	6.71 (8.060)	8.00 (10.100)	6.47 (10.898)	20.00 (30.537)
Higher ESS scorers, $N = 15$*	−3.60 (11.72)	1.47 (4.868)	4.67 (11.70)	5.07 (9.528)	8.13 (10.267)	15.73 (25.789)

Note. Values represent means (and standard deviations). *Lower ESS = scores of 5 and below. Higher ESS = scores of 6 and above. ESS = Epworth Sleepiness Scale.

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correlated with overall net score, $r(30) = -0.006$, $p = 0.974$, or performance in any of the five blocks.

EV Parameters: Demographic Associations

Spearman rank-order correlations were used to examine relationships between EV parameters and age and FSIQ. None of these correlations were significant. Mann-Whitney U tests were used to evaluate sex differences on the EV parameters, and there were no significant differences between men and women.

EV Parameters: Associations With Daytime Sleepiness and Recent Sleep Debt

Daytime sleepiness was not significantly associated with either the attention weight parameter (w), Spearman's $r(30) = -0.173$, $p = 0.343$, or the sensitivity parameter (c), Spearman's $r(30) = -0.255$, $p = 0.159$. However, greater daytime sleepiness was significantly correlated with higher values of the updating parameter, (α), Spearman's $r(30) = 0.390$, $p = 0.027$. Participants who reported higher levels of daytime sleepiness demonstrated reduced tendency to use information from outcomes that were more distant in time when making decisions. Recent sleep debt was also associated with higher values of the updating parameter (α), Spearman's $r(30) = 0.413$, $p = 0.019$, but not the attention weight (w) or sensitivity (c) parameters ($ps > .05$).

Analysis Including Deck Maximizers and Poor Model Fit

As a supplemental analysis, EV parameters were calculated for individuals who maximized a deck ($n = 13$) by only including trials prior to deck maximization. All but one had acceptable model fit ($G^2 > 0$). This resulted in a final combined sample of 44. As before, higher values of the updating parameter were evident in those with higher daytime sleepiness, Spearman's $r(42) = 0.364$, $p = 0.015$, and in those with greater recent sleep debt, Spearman's $r(42) = 0.333$, $p = 0.027$. There were no significant correlations between the sleep measures and the other EV parameters. Similarly, re-including the 10 participants with poor model fit

($N = 54$) resulted in significant correlations between the updating parameter and ESS scores, Spearman's $r(52) = 0.332$, $p = 0.017$, and between the updating parameter and recent sleep debt, Spearman's $r(52) = 0.275$, $p = 0.044$; again, there were no significant correlations between sleep measures and the other EV parameters. Though these results are treated with caution because EV parameters may be less stable since they are estimated on the basis of fewer trials for those who maximized decks, they support the key findings described above.

DISCUSSION

In healthy adult participants, daytime sleepiness was associated with increased values of the updating parameter on the IGT, reflecting reduced incorporation of temporally remote information into ongoing decision making. That is, individuals who reported greater sleepiness had a shorter time horizon over which they were integrating information when making decisions. This change in the updating parameter was also evident in participants who reported greater levels of recent sleep debt.

To our knowledge, this is the first study to apply the EV model to examine associations between sleep-related variables and IGT performance. In this study, daytime sleepiness and recent sleep debt were not associated with total net score. This differs from prior work examining total sleep deprivation, which has consistently been associated with significant declines in net scores for later blocks of the task when compared to the normally rested state (Killgore et al., 2006, 2007; Killgore, Grugle, et al., 2012). Even though the association between sleepiness and IGT performance was not evident using the coarse metric of net score, changes in underlying cognitive processes were found when task performance was examined using the EV model. By breaking down the IGT into underlying component processes, this study begins to address the "task impurity problem" (Jackson et al., 2013) that has posed a challenge to research in cognitive effects of sleep deprivation (Whitney & Hinson, 2010), and joins a growing body of literature demonstrating that mathematical modeling can elucidate multiple factors underlying task performance during sleep loss (Ratcliff & Van Dongen, 2011, 2009).

Our data suggest that in healthy adults, insufficient sleep and daytime sleepiness appear to be associated with a shortening of the time horizon over which decisions are integrated. This finding may have important implications for professional fields such as medicine and aviation that require integration of complex information and updating of information over varying time horizons to guide decision making. These findings are particularly striking because the measured levels of sleepiness and recent sleep debt are not atypical of the normal variations often encountered within the general population. In other words, even mild daytime sleepiness and modest curtailment of sleep were associated with a narrowing of the time horizon over which information may be integrated into decision making.

One possible explanation of the present findings is that sleep loss may primarily affect performance on decision making tasks by simply disrupting basic underlying cognitive processes such as the ability to sustain attention or hold information in working memory over intermediate periods of time. It is well established that sustained attention processes are impaired by insufficient sleep (Doran et al., 2001), which could explain the shift in the updating parameter. However, the sensitivity (c) parameter, which measures randomness of responding,

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was unrelated to either sleep measure, suggesting that attention to the task was not particularly affected. One possible explanation is that proactive interference from early task trials interfered with subsequent learning during later blocks, though others have found no increases in proactive interference in working memory functioning under total sleep deprivation (Tucker et al., 2010). Sleep loss may actually have resulted in an increase in retroactive interference; learning of outcomes from recent trials may have impaired recall of information from more remote trials in individuals experiencing sleep loss. In fact, medial temporal lobe memory systems play a critical role in IGT performance, particularly in terms of maintaining and updating the relationship between decks and their reward and punishment contingencies (Gupta, Duff, Denburg, & Cohen, 2009). Source memory for the outcomes (i.e., recalling which deck produced which result) is critical for IGT performance (Whitney & Hinson, 2012). It is possible that the alteration in the updating parameter reflects difficulty with the memory requirements of the IGT under sleep loss. Alternatively, it is also conceivable that sleep loss may alter the affective salience of more recent versus more distant information. This would suggest that there is an affective discounting of the importance of information acquired further back in time. If the change in the updating parameter is related to decreased utilization of more remote information rather than decreased maintenance of it, then this change might be susceptible to manipulations such as altering temporal attention via framing effects. Finally, some evidence also suggests that sleep deprivation may affect time perception (Miró, Cano, Espinosa-Fernández, & Buela-Casal, 2003), possibly through altered prefrontal cortical functioning (Soshi et al., 2010). It is therefore conceivable that early trials are considered to be less important and further away in time due to altered time perception in those with greater sleepiness.

It will also be important to examine whether this shift in the updating parameter also explains the changes in IGT performance that are evident in clinical populations with sleep disorders. This demonstration of change in EV parameters in healthy participants reporting daytime sleepiness and recent sleep curtailment highlights the importance of considering sleep in future studies investigating executive functioning deficits in clinical populations, including individuals with psychiatric disorders. Applying this type of analysis in examining disruption of IGT task performance under sleep deprivation is an important future direction, as is examining how disrupting specific sleep phases may result in changes to EV parameters.

This study has several limitations, the most significant of which is its reliance on retrospective self-reports of sleep patterns. It would be ideal to prospectively track sleep (e.g., via actigraphy or ambulatory polysomnography) and then examine associations with IGT task performance. It would be preferable for sleep history to be tracked over the course of several days, rather than only on the night prior to the first study visit, as was done in this study. The lack of data regarding sleep between study Day 1 and Day 2 is also a limitation, though the converging results for recent sleep debt and daytime sleepiness support our conclusion that recent sleep loss is associated with alterations in IGT performance. Additionally, this was a naturalistic, population-based study and we did not exclude participants for factors that might affect sleep such as shift work or recent travel across time zones. Also, the IGT was part of an extensive battery of cognitive tasks and questionnaires, and it is possible that fatigue interacted with daytime sleepiness and sleep debt to produce the present findings. Additionally, in this study, over 20% of our original sample maximized one of the decks. Deck maximization has been described as "arising very seldom" (Bechara et al., 1999), but this was not the case in the present sample. IGT investigators are encouraged to describe whether and how deck

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choices were constrained, particularly for analyses that examine decision making on a trial-by-trial basis. The presence of a minority of participants with poor model fit is consistent with others' findings that participants employ heterogeneous strategies in approaching the IGT (Worthy, Hawthorne, & Otto, 2013). Our finding that sleep loss affects the updating parameter on the IGT may therefore only apply when participants are employing the most common strategy, which is captured by the EV model. Finally, in this study we tested only the EV model and did not fit and compare other models, such as the Prospect Valence-Learning (PVL) model (Ahn, Busemeyer, Wagenmakers, & Stout, 2008; Fridberg, Queller, & Ahn, 2010; Steingroever, Wetzels, & Wagenmakers, 2013). It also is possible to fit the EV model by task block. Performing these additional types of analyses could be an important direction for future work.

In summary, the EV model was used to examine IGT performance. Adults who reported greater daytime sleepiness showed increased values of the updating parameter, indicating a lower tendency to integrate outcomes that occurred more distally in time. We conclude that by shortening the time horizon over which decisions are integrated, sleep loss may affect decision making in healthy adults.

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