

THE ASSOCIATIONS BETWEEN PERMANENT CHANGE OF STATION (PCS) MOVES,
PARENTING STRESS, AND FAMILY RESILIENCE IN RELATION TO THE PHYSICAL
AND PSYCHOLOGICAL HEALTH IN A SAMPLE OF HIGH-RISK MILITARY
ADOLESCENTS

By

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Dissertation submitted to the Faculty of the
Medical and Clinical Psychology Program
Uniformed Services University of the Health Sciences
In partial fulfillment of the requirements for the degree of
Doctor of Philosophy, 2022

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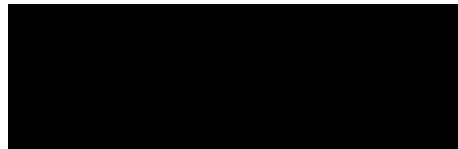
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UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES

SCHOOL OF MEDICINE GRADUATE PROGRAMS

Graduate Education Office (A 1045), 4301 Jones Bridge Road, Bethesda, MD 20814



APPROVAL OF THE DOCTORAL DISSERTATION IN THE DEPARTMENT OF MEDICAL AND CLINICAL PSYCHOLOGY

Title of Dissertation: "The Associations between Permanent Change of Station (PCS) Moves, Parental Stress, and Family Resilience on the Physical and Psychological Health in a Sample of High-Risk Military Adolescents"

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ABSTRACT

Title of Dissertation: The Associations between Permanent Change of Station (PCS) Moves, Parenting Stress, and Family Resilience in Relation to the Physical and Psychological Health in a Sample of High-Risk Military Adolescents.

Phillip C. Kroke, Doctoral Candidate, 2022

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Permanent change of station (PCS) moves are a frequent and significant stressor for military families. At any given time, approximately one-third of military families are experiencing disruptions due to a PCS move. Military families rank PCS moves as highly stressful — second only to deployment. Consequently, it is important to understand how the stress of military relocation may adversely impact servicemembers, their families, and the military's family readiness system. Past work has focused on how PCS moves influence servicemember readiness and operational performance. Comparatively little research has examined the biopsychosocial sequelae of PCS moves for military children and adolescents. The existing research on the impact of PCS moves for military adolescents suggests relocation stress interacts with: the stress, mental health, and coping of the parent; community/familial resources; and the frequency/recency of relocation events. Youth with inherent vulnerabilities (e.g., anxiety, loss of control eating) and psychosocial risk history may be particularly affected by PCS

moves. Assessment methods utilizing both biological and psychological data offer a promising method of understanding military relocation stress' influence on adolescent development – especially for at-risk youth samples. Given the dearth of research on this important topic, this study's goals are: 1) to study the association between PCS moves and physical/psychological health in a vulnerable adolescent sample; and 2) to determine if the proposed association is moderated by: community/familial factors (i.e., family resilience) and the stress, mental health, and coping of the parent (i.e., parenting stress). To accomplish these goals, the study conducted a secondary analysis drawn from the Preventing Obesity in Military Communities – Adolescents study (ClinicalTrials.gov ID: NCT02671292). The parent study was an obesity prevention trial designed to investigate the efficacy of group interpersonal psychotherapy for military adolescents with elevated anxiety and/or the presence of loss-of-control eating. Given this inclusion criteria, the sample was considered at high-risk for developing binge-eating disorder and adult obesity and may be at a heightened risk of the potential negative outcomes associated with PCS moves. We hypothesized that PCS moves in adolescents would be associated with worse physiological health and reduced psychological well-being. It was also expected family processes would moderate the association such that the negative association between PCS mobility and physical/psychological health would be attenuated at greater levels of family resilience and exacerbated at higher levels of parenting stress. Participants were 164 military-associated adolescents (age: 14.5 ± 1.6 yr, 58.5% female, 57.9% white, BMIz: 1.9 ± 0.4) drawn from baseline assessments prior to treatment randomization. No significant difference was found for any of the demographic variables between those with a history of relocation (77.5%) and those who had no history of relocation (22.5%). For the primary analysis, a series of multiple linear regression models were conducted using multiply imputed data to examine the relationship

between PCS moves with 1) PSS total score, 2) BDI-II total score, 3) the allostatic load index, after adjusting for covariates. To examine if *PCS moves* interacted with the parenting stress (PSI-SF) or family resilience (FRAS), the linear regression models were repeated with the addition of two separate interaction terms. The interaction term was computed by the product of mean-centered PCS moves and the mean-centered interaction terms (i.e., PSI, FRAS). Results from the analyses did not support the hypothesized relationship between PCS moves and physical/psychological health. Family resilience processes and parenting stress were also not found influence the relationship between PCS moves and worse psychological/physical health. Contrary to expectations, PCS moves were found to be significantly negatively associated ($b = -0.42$, $SE[b] = 0.21$, $p = .044$, 95% CI $[-0.83, -0.01]$) with scores on a measure of general stress (PSS), such that participants who reported more moves also reported lower perceived stress. Although this unexpected finding should be interpreted with caution, it seems to refute a number of common misconceptions regarding the deleterious effect of PCS moves for military-associated youth. In fact, it suggests PCS moves may enhance resilience under certain circumstances. Future research should expand on the current study's limitations by including longitudinal designs, operationalize relocation to incorporate PCS recency, and replicate findings with larger community samples.

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CHAPTER 1: INTRODUCTION

Readiness, Relocation, and the Military Adolescent

There are currently over 1.6 million children within Active Duty (AD) and Selected Reserve (SR) military families ([99](#)). Military families have historically played an important role in national defense strategy ([435](#)). In our current era, the Department of Defense (DoD) regards the health and well-being of military families as essential to servicemember operational readiness ([408](#)). Considerable resources have been spent developing a comprehensive family readiness system to enhance the health and well-being of military families ([98](#); [349](#)). To ensure family readiness programs successfully navigate the challenges experienced in the context of military service, DoD policymakers and researchers have a vested interest in understanding unique challenges experienced by military families. Past research identified deployment and other military lifestyle stressors as having a significant impact on family functioning and well-being ([172](#); [252](#)).

One important challenge for military families are frequent relocations to national and international duty stations. Relocations associated with military service are classified as permanent change of station (PCS) moves. Over a third of military personnel are in the midst of a PCS move at any given time ([408](#)) and PCS moves are ranked as a prominent stressor for military families ([29](#); [49](#); [385](#)). Exposure to relocation-related stress is problematic for children and adolescents since stress may result in lower physical health and/or the onset of psychological disorders ([36](#); [331](#); [407](#)). The available research examining frequent PCS moves on the health and well-being of military families is limited, especially considering the prevalence of PCS moves for military families. Despite these limitations, there is sufficient evidence to suggest military relocation stress is influenced by: 1) the stress, mental health, and coping of the parent;

2) community/familial resources; 3) inherent vulnerability and risk history; and 4) the frequency and recency of relocation events ([56](#); [288](#)).

Relocation stress may impact vulnerable sub-populations who are already at-risk for developing significant physical and mental health conditions. In particular, military-connected adolescents with existing morbidities may be particularly vulnerable to relocation stress due to stress sensitivity in emerging biological systems ([146](#); [252](#)). Accordingly, it is important to understand which military adolescents are at particular risk for adverse adjustment to PCS moves. By using an ethnically diverse sample of military-connected adolescents at-risk for developing obesity and/or binge eating disorder, this study was designed to examine relations between relocation stress and biopsychosocial functioning.

The aim of this introductory chapter is to describe the known relations between military adolescent relocation, stress, and adverse health outcomes while identifying gaps in existing knowledge. To achieve these aims, this chapter will review the history of the U.S. military family, the family readiness system, and adolescent military relocation. Next, the associations between military relocation and adverse physical/psychological health outcomes will be reviewed. A conceptual model of Military Relocation Stress will be presented incorporating relocation factors thought to contribute to the psychosocial adjustment of a PCS move. Resilience factors and the family resilience model ([425](#)) will be examined as moderators of military relocation stress in order to better elucidate the relationship between relocation, stress, and adverse health outcomes. The two-stage allostatic model will be introduced to conceptualize military relocation stress' impact on physiological systems. Lastly, a proposed biological index of allostatic load (i.e., chronic stress) will be defined and justified for inclusion into the proposed study.

Families and Adolescents in the Military Context

The U.S. military has been aptly designated a “greedy institution,” in that it imposes a pattern of demands on servicemembers beyond many civilian professions ([353](#)). Accordingly, the “military lifestyle” involves a unique combination of stressful commitments, including, but not limited to extended duty hours, familial separation, frequent relocations, and exposure to combat ([194](#)). In addition, DoD-wide cyclical budget cuts, manpower adjustments, and other policies can significantly impact servicemember and family functioning. A high operational tempo over the past 20 years has led to an accumulation of many of these unique stressors ([326](#)). Although this stress load is generally attributed to the servicemember, military families are also affected ([339](#)). Despite the potential hardships of the military lifestyle, families are essential to the well-being and mission readiness of their respective services. Families were not always afforded such concern by military or political leadership. Indeed, the perception of military families has transitioned substantially over the past 50 years.

History of U.S. Military Families

The military family has played an important, multi-faceted role throughout the history of the U.S. Armed Forces. For the purposes of this study, military family is defined as immediate family related by blood, adoption, or marriage to a current service member. In the early U.S. military, the spouses, children, and assorted hangers-on informally fulfilled critical functions in the sustainment of the military formation ([204](#); [421](#)). However, the term “military family” gained a formal connotation in the 1970s along with one of the most significant demographic shifts in U.S. military history: the 1973 transition to an all-volunteer force (AVF). Pre-transition, the U.S. military relied on a small core of professional soldiers supplemented in times of war through universal conscription of young males. Conscripted, military-age males historically delayed

having a family until the completion of their service commitment (362). The transition to AVF eliminated conscription and, as a result, dramatically increased the prevalence of military families. To illustrate this demographic shift, military family members now outnumber servicemembers by over 500,000 (99). Of the 2.1 million Servicemembers, nearly half (48.7%) are married, close to a third (32.9%) are married with children, and a relatively low percentage (5.9%) are single parents (99).

Eliminating conscription also relegated the military recruiting process to standard market forces to compete with the civilian sector. Retirement plans, competitive wages, and mandated healthcare are necessary to attract and retain motivated workers. Introducing the “free market” of voluntary military service also introduced another pressure: the amenability of families to the military lifestyle (362).

The Military Family Readiness System

Throughout the majority of U.S. military history, consideration for family well-being was piecemeal, improvisational, and reactive – save for families of senior military leadership (362). It took the decade following the transition to AVF for the family’s role in retention and well-being to come under significant scrutiny by the higher echelons of military leadership. In his seminal 1983 white paper, *The Army Family*, the Army Chief of Staff outlined the “Army Family” and the “Army Family Research Program” as essential components for military readiness of an all-volunteer force (435). As a result of these concentrated efforts from the Army and its sister branches, a considerable amount of government-sponsored research studied how military lifestyle stressors adversely influence the readiness of the armed forces (362).

Many of the military’s modern systematic family-care programs are a result of this concerted push by military decision-makers. For the U.S. Army, the earliest systemic change was

guided by the Army Family Action Plan (AFAP). The AFAP was formal process wherein grassroots delegates convened, forwarded concerns, and recommended improvements to remedy family-related issues (376). By any measure, the AFAP was highly effective at addressing systemic change for military families. [Shinseki \(362\)](#) reviewed AFAP's progress between 1984 to 2003 to find 82 legislative changes in congress, 130 revised DOD/Army regulations, and 140 improvements to existing military programs/services.

The legacy of AFAP and similar programs continues today with 5% of the FY2020 Department of Defense (DOD) personnel budget allocated to programs designed to strengthen military families (100). Examples of such improvements include: quality child-care and housing (62), access to unique medical needs through the Exceptional Family Member Program (430), transition assistance for students in the Department of Defense Education Activity (DODEA) school system (38), and support for Family Readiness Groups (362). There appears to be no shortage of services afforded to military families; [Conforte et al \(83\)](#) identified hundreds of DOD/VA sponsored and private/non-profit family support services tailored specifically to military families/adolescents.

The plethora of resources has led some to suggest military families might be best served by improving the delivery, efficiency, and surveillance of available services (83). One proposed solution to improve the family readiness system is to target vulnerable military sub-populations for detailed surveillance and evidence-based interventions/policies. One such sub-population of the U.S. military family, the military adolescent, appears to be especially vulnerable to military lifestyle stressors.

Adolescents in the Military context

Military children and adolescents have undergone dramatic scrutiny by military leaders, policy makers, and social scientists during the transition to the AVF. As a result, the 1970s generated significant research interest on the possible adverse impact of the military lifestyle on children/adolescents ([154](#)). Military adolescents have become increasingly important as they emerge to be a substantial sub-population within modern military communities. Of the 1.6 million military children, 389,510 are adolescents between the ages of 12 and 18 ([99](#)).

Leaders and policy-makers have also increasingly recognized the importance of military-connected youth for national security. There is a wide body of evidence to support the health and well-being of youth has a demonstrable impact on family/parental functioning ([104](#); [210](#); [251](#)). Indeed, parenting a child with special medical and/or psychiatric considerations necessitates heavy parental involvement and, without proper support/resources, may result in parental distress ([43](#)). In chronic illness, the severity of a child's symptomatology is one of the most consistent predictors of parenting stress ([231](#)). In the military context, the downstream impact of parenting-related stress adversely influences operational performance and retention ([21](#)). Nearly 10% of military families identify at least one family member with special medical considerations ([430](#)). Although the military has developed programs to support these families ([430](#)), military-specific stressors (e.g., deployment, relocation) can exacerbate existing physical and mental health conditions. Given the identified relationship between the well-being of the military child and servicemember performance, the family readiness system has a vested interest in preventative measures for vulnerable family members ([83](#)).

There is also evidence to suggest America's military is increasingly becoming a "family business" ([403](#)). Nearly 80% of veterans have a family member who served versus only 69% of

the general public, and the gaps are even more pronounced amongst younger veterans ([375](#)). This data suggests U.S. military families are increasingly shouldering the burden of service in the Armed Forces. Currently, only 29% of U.S. youth meet military eligibility requirements which further reduces the pool of qualified volunteers. These trends have raised serious interest in addressing the physical and emotional needs of military-connected family members ([375](#)). The inter-related factors of weight status and psychological health conditions in military-connected youth have emerged to be the most significant medical factors limiting eligibility for military service ([201](#)). As a result, policy makers have begun to view U.S. military-connected youth as a diminishing resource requiring serious intervention for their physical and emotional well-being.

In the 1970s, [Lagrone \(208\)](#) compiled his clinical observations into what he termed as a “Military Family Syndrome;” thought to be caused by military culture/policies inimical to healthy family functioning. Although [Lagrone \(208\)](#) should be credited for bringing attention to military children/adolescents, later researchers characterize his work to be more representative of a post-Vietnam polemic against military culture ([86](#)). Fortunately, later researchers contested his chief assertions: there is limited evidence for a “military family syndrome” nor any consistent differences in behavioral health outcomes between military and civilian samples ([187](#)). For example, [Williamson et al \(439\)](#) conducted a recent systematic review of child well-being across civilian and military-connected samples. Across the nine studies identified, military-connected children/adolescents did not differ substantially from their civilian counterparts on externalizing behavior, substance use, and/or mental health problems. However, specific demographics (i.e., age, sex) and lifestyle (i.e., parental/sibling deployment) factors were associated with greater risk for substance use, depressive symptoms, and externalizing behaviors. [Cozza and Lerner \(86\)](#) likewise characterized military children/adolescents as a healthy and adaptive cohort, but as

vulnerable to adjustment difficulties when exposed to specific military lifestyle stressors (e.g., deployment/family separation).

Historical interest has centered primarily on deployment-related concerns for military families. There is evidence to suggest deployment adversely impacts servicemember ([172](#)) and family well-being ([281](#)). For example, [Higgins Neyland et al \(172\)](#) found that for those parents with high parental distress greater deployment frequency was associated with indices of adolescent psychopathology. Given the extensive research interest deployment has generated, it may be valuable to examine other military lifestyle stressors. Relocation, one such lifestyle stressor for military adolescents, has generated increasing interest from policy makers and social scientists ([408](#)).

Relocation in the Military Context

Relocations associated with military service are classified as permanent change of station (PCS) moves. The PCS move process originated in the 1800s but its policies have undergone significant revision in the past 50 years ([212](#)). The primary impetus for these changes also coincided with AVF-related research which suggested high rates of relocation adversely influenced attrition rates, retention, well-being, and overall mission readiness ([36](#); [44](#); [115](#); [275](#); [329](#); [344](#); [417](#)). Recently, [Tong et al \(408\)](#) reviewed the military relocation literature and posited three pathways which explain the known deleterious effects of relocation on readiness: 1) a reduction in servicemember retention intention; 2) reduced spousal employment and earnings; 3) and increased stress leading to psychiatric illness, child behavioral issues, and financial issues. To note, the third category mentioned will be of special consideration for the purposes of this study.

As a result of this concerted research effort, policymakers and stakeholders have amended policy and instituted programs to mitigate stress associated with the military lifestyle - including relocation ([408](#)). To illustrate this shift, there have been over 108 changes to relocation policy for the families of armed services since the 1980s ([362](#)). Despite such progress, relocation policy has several recognized areas of improvement: 1) address the relocation stress induced by family disruptions; 2) improve surveillance and programmatic evaluation of existing services and programs ([408](#)). The proposed study will address both areas by operationalizing relocation stress and evaluating current trends in a sample of vulnerable youth. The following section will provide an overview of current PCS policy, introduce the concept of military relocation stress, and describe relocation-induced disruptions for the military family and child.

PCS Process and Military Relocation Stress: First-Order and Second-Order Disruptions

The PCS is a lasting assignment (i.e., > 6 months), detail, or transfer of a servicemember and their family to a different duty station ([408](#)). The rationale for multiple relocations varies, but is generally done to meet manning requirements, provide broadening opportunities, and maintain good order and discipline. Duty stations are spread throughout the continental United States (CONUS) and outside of the continental United States (OCONUS; i.e., Hawaii, Alaska). OCONUS also includes various overseas military bases and territories across the world ([18](#)). A PCS may be initiated in response to accessions or separation of a servicemember, rotational requirements, or operational transfers to another duty station within the CONUS ([176](#)). PCS moves are generally routine (i.e., every 3-4 years) and expected. The flexibility for any given servicemember to select their duty station is variable and contingent upon their specialty, rank, and time in service ([18](#)).

Military relocation stress is the cumulative stress load caused by a PCS move. The stress of relocation can be caused by first-order and/or second-order disruptions ([105](#)). *First-order disruptions* are stressors concurrent to the relocation process ([408](#)). Stressors in this category are contingent on the resolution of an expected, resolvable, and normative stressor. Examples of first-order disruptions include: the need to find a new home, changing schools, or acclimating to a new community. *Second-order disruptions* are indirect stressors that occur at any time during or following a relocation. This category of disruptions consists of the lasting psychosocial sequelae of a move. Altered family functioning, mental health, peer support, and academic outcomes are some examples second-order disruptions. These disruptions often co-occur and can be mutually reinforcing ([415](#)), with the specific composition of stressors depending on the dynamic of each individual family ([408](#)). Military and civilian samples share overlapping first and second-order disruptions, but military relocation stress is distinguished by the uniqueness of the PCS process and the military lifestyle.

Military Lifestyle Disruptions for the Military Family and Adolescents

It is important to understand the pathways by which PCS moves may contribute to military relocation stress. First-order disruptions are relatively self-explanatory: they are directly related to the regular stressors, hassles, and frustrations of the relocation process. Therefore, this section will focus primarily on several prominent second-order disruptions unique to the military lifestyle: 1) family separation and role adjustment due to relocation; 2) military spouse underemployment; 3) dependent children's education; 4) and a lack of control over the military career ([121](#)).

Separation is stressful for all members of a military family ([281](#)). Although deployment is the prototypical example of familial separation, certain PCS moves may cause temporary

disruption for families. The ensuing separation from immediate and extended family members may alter the “homeostasis” of the family structure causing a revaluation of familial roles (86). The adolescent or family experience relocation stress as they adjust to their new roles and responsibilities, find new resources to cope, and/or cultivate different family processes. This is especially relevant for the small minority (7-9%) of servicemembers who elect to live geographically separated from their family as “geographic bachelor/bachelorettes” (76).

The effect of relocation on military spouse employment is a topic of increasing interest as traditional gender roles become less restrictive (75). Routine relocations can significantly hinder professional careers and overall earnings (84). Military spouses must make the decision to live separate from their families or lose key opportunities for professional development. In addition, an increasing prevalence of military families rely on a dual income to maintain their lifestyle (281). Consequently, military spouse underemployment may increase financial strain thereby influencing relocation stress (263).

Adolescent educational and social trajectories are also influenced by relocation. PCS moves are generally both residential and academic relocations. The Department of Defense Education Activity (DoDEA) school system provides some consistency and operates over 194 schools across the continental U.S., foreign countries, and U.S. territories (372). However, the vast majority of military children attend public schools across the United States. Quality of schools, educational standards, and peer groups can be dramatically altered by a PCS move (281). It is well known social support has significant implications for healthy adolescent development (378) and may help mitigate stressful life experiences (321).

Military families are often stressed by the minimal influence over their duty stations. Although PCS moves are normative stressors for military families, the end duty location can be

unpleasant. Postings to such disagreeable duty stations are commonplace (385). Manning considerations and/or mandatory training make it expected that at least one duty assignment in the course of a career will be outside of the servicemember's control. Military adolescents are especially affected by relocations to unpleasant duty locales, unfamiliar cultures, and/or unsupportive communities (86).

Readiness, Relocation, and the Military Adolescent: Summary and Conclusions

There is a need for greater research examining vulnerable military populations' responses to military lifestyle stressors – including PCS moves. The need for quality research has emerged as the U.S. military family has become an essential component of most, if not all, military communities. The historical transition to AVF heralded a demographic shift thereby significantly increasing the proportion of military family members (99). Accordingly, extensive resources have been afforded to create a military family readiness system to ensure family well-being and servicemember retention (362). However, the family readiness system has considerable overlap and little programmatic surveillance in many of its programs. This had led some to suggest military families might be best served by improving the delivery, efficiency, and surveillance of available services (83). One proposed solution is to eschew a “one-size-fits-all” approach to family readiness by ensuring adequate research and evidence-based intervention strategies for vulnerable sub-populations.

This section highlighted the importance of examining the military adolescent- an at-risk sub-population of the U.S. military family. While a purportedly resilient sub-population, military adolescents may be particularly vulnerable to various military lifestyle stressors to include PCS moves (86). With over 50 years of amendments to military relocation policy, it is essential to

understand if we are successfully getting the necessary resources/programs to those at greatest need ([83](#)).

Relocation and the Adolescent

In the United States children relocate frequently with 13% of children moving in any given year ([57](#); [182](#)). Relocation is even more common for children in military families who relocate at an annual rate of 30% ([57](#); [408](#)). Relocations are ranked by both military and civilian samples as highly stressful life events ([186](#); [385](#)). The stress of relocation and the well-established relationship between stress and health outcomes ([392](#); [400](#)) make it important to understand the possible implications of frequent relocations – especially in high-risk adolescent samples. The following section will describe the known associations between military adolescent relocation and adverse physical/psychological health outcomes. A conceptual model of Military Relocation Stress will be introduced. Lastly, the model will be applied to the study’s sample of military-connected adolescents at high-risk for developing eating pathology and obesity.

Child and Adolescent Relocation: Physical and Psychological Health

Relocation outcomes denote a broad range of research related to residential, academic, geographic, and community mobility. As a result, many studies are difficult to generalize given the majority of studies are cross-sectional, examine unique populations, and inconsistently operationalize relocation ([36](#); [79](#); [163](#); [340](#); [365](#)). Despite these challenges, several reviews have concluded individual, relational, and community factors interact with high rates of relocation to adversely influence psychological and physiological health outcomes ([186](#); [262](#)). This section will briefly summarize pertinent findings in military child/adolescent samples as they relate to physical and psychological functioning.

Physical Health

Researchers have not examined child/adolescent indices of physical health and/or biological functioning (i.e., biomarkers) in the context of military relocation. Given the lack of research, it is useful to examine the extant literature in civilian samples. There is a wide consensus that civilian residential moves, especially in early childhood, are associated with the development of health conditions in later life ([28](#); [412](#)). For example, [Hutchings et al \(180\)](#) found residential moves between the ages of 1-5 were associated with preventable health hospitalizations for ear, nose, and throat infections, convulsions/epilepsy, asthma, and influenza/pneumonia. In this study, and others like it, there is no clear evidence if physical health outcomes were due to the stress of the move itself, lack of engagement with primary care providers, or mediated by another combination of risk factors instigated and maintained by relocation events. Indeed, the specific influence of such second-order disruptions as socioeconomic deprivation (i.e., Poverty), familial/community factors (i.e., domestic violence), and/or altered availability/utilization of healthcare services is difficult to determine ([101](#); [106](#); [132](#)). Regardless, the unique contribution of the military lifestyle and PCS process presents concerns over the generalizability of civilian relocation outcome research to military samples ([281](#)). As a result, the lack of physical health outcome data for adolescent military samples is relative gap in the literature which warrants further research.

Psychological Health

The limited research on the impact of PCS moves indicates military children/adolescents are, when provided adequate resources, relatively resilient to the psychological consequences of military relocation stress ([47](#); [153](#); [252](#); [344](#)). For example, [Rippe \(316\)](#) found no difference on measures of school performance between samples of military adolescents with low, medium, and

high relocation rates when compared to a non-military control. In fact, there is evidence higher rates of relocation are associated with improved adolescent behavior and psychological adjustment in military samples (383). In another study, [Weber and Weber \(432\)](#) surveyed 179 parents of military adolescents to assess the effect of relocation on adolescents' school conduct and behavior. The results suggested that, when controlling for age, higher rates of relocation was associated with better adolescent outcomes.

Although a significant number of military adolescents appear to be resilient, it is evident that some of adolescents are adversely affected by military relocation stress (252). [De Pedro et al \(93\)](#) and [Gilreath et al \(148\)](#) found school transitions in samples of military-connected adolescents were associated with increased physical violence, weapon carrying, and greater rates of victimization. In a study of younger military adolescents, [Richardson et al \(314\)](#) found multiple school changes were associated with greater endorsement of anxiety-related cognitions and symptoms. However, these studies were limited in that they did not control for contemporaneous events known to influence adolescent behavior (e.g., deployment), were not exclusive to adolescent dependents of military servicemembers, and did not directly assess PCS moves.

The most compelling evidence that PCS moves contribute to adverse psychological health outcomes can be found in the healthcare utilization literature. [Millegan et al \(253\)](#) examined a large sample (N = 548,336) of children/adolescents with a servicemember parent in the military healthcare system. The authors found military adolescents who relocated in the past year sought mental health outpatient (odds ratio [OR] 1.04; 95% CI 1.01-1.07), psychiatric hospitalization (OR 1.19; 95% CI 1.07-1.32), and emergency psychiatric visits (OR 1.20; 95% CI 1.07-1.32) more frequently than those who were geographically stable. The psychiatric

diagnoses were predominantly adjustment disorder, drug usage, suicide/ self-injurious behavior, and attention-deficit/conduct disorder. Millegan and team concluded the increase in externalizing disorders can be primarily attributed to the challenges in adjustment to a new location.

Military Relocation Factors Influencing Adolescent Health Outcomes

Given the mixed evidence regarding military relocation stress' influence on adolescent outcomes, multiple studies have attempted to determine what individual, relational, and military-specific factors contribute to risk and resiliency for adverse health outcomes. The current literature examining PCS moves supports military relocation stress is: 1) influenced by the stress, mental health, and coping of the parent(s) ([56](#); [234](#); [384](#)); 2) attenuated by community/familial resources ([47](#); [56](#); [384](#)); 3) altered by inherent vulnerability and risk history ([253](#)); 4) contingent on the frequency and recency of relocation events ([14](#); [56](#); [288](#)). The relationship between these relocation factors can be found modeled in Figure 1.

Stress, Mental Health, and Coping of the Parent(s)

The stress, mental health, and coping of the parent(s) may influence PCS outcomes for military adolescents more than the PCS itself. For example, a study examining 86 mother-child dyads in military families found mobility to have a negligible influence on psychological adjustment ([128](#)). In fact, maternal depression was the most significant predictor for their child's externalizing behavior (i.e., aggression/non-compliance). This finding has been corroborated in recent findings in a sample of Canadian military adolescents. [Perreault et al \(288\)](#) asked Canadian servicemembers to complete online questionnaires on their adolescents' relocation history, academic performance, and internalized/externalized difficulties. Again, relocation history had a negligible influence on adolescent outcomes. Instead, parental perceived stress emerged as the most significant predictor for military adolescent outcomes. Overall, these results

suggest adolescent outcomes to relocation are indirectly influenced by the mental health and coping of the parent(s). This is analogous to the posited effect of second-order disruptions thought to engender chronic relocation stress.

Familial and Community Resources

Similarly, familial/community resources have been suggested to attenuate the association between PCS moves and adverse outcomes in military adolescents ([219](#); [234](#)). The variable that has been the most examined is the relationships within the family – especially that of the parent-child dyad ([128](#)). To provide an illustrative example, [Bullock \(56\)](#) conducted a mixed methods study related to the effects of relocation on the well-being of a sample of Canadian military adolescents. The authors found self-reported affective reactivity to relocation was associated with worse adolescent well-being. However, the quality of the parent child-relationship moderated this association, in that a strong parent-child relationship attenuated the relationship between affective reactivity to relocation and adolescent psychological outcomes. In aggregate these results suggest family relationships can encourage resilience by exerting a protective influence on adolescents who may be susceptible to military relocation stress.

Inherent Vulnerability and Risk History

Past adverse childhood events, families with special needs, and past psychiatric history interact with military relocation stress may also exacerbate adjustment difficulties ([101](#)). [Millegan et al \(253\)](#) found the most significant predictor for mental health outpatient services for adolescents with a recent relocation (< 12 months) was a past personal psychiatric history (OR 12.44; 95% confidence interval 12.19-12.70). The impact of PCS moves on outpatient visits was still significant even after adjusting for contemporaneous variables associated with mental healthcare utilization.

Similarly, [Higgins Neyland et al \(173\)](#) examined a military adolescent sample at-risk for developing binge-eating disorder and obesity. The study authors found PCS moves were associated with disordered eating behaviors for those individuals who reported greater weight-based victimization. Disordered eating behaviors, in turn, are strongly associated with worsening physical health ([266](#); [269](#)). In addition, there is strong evidence to support poor physical health in youth is associated with worse health prognosis in later life ([381](#)). Considering the outcomes of Millegan et al. (251) and Higgins Neyland et al. (170), there is preliminary evidence to support that PCS moves may interact with existing morbidities to indirectly impact psychopathology and physical health outcomes.

Frequency and Recency of Relocation Events

Whether the frequency and recency of PCS is associated with adolescent outcomes is unclear. Frequency and recency of relocation events have been found to influence adolescent psychological health and well-being ([144](#)). The mobility rate is often used to assess relocation frequency and represents the total number of moves divided by the age of the child/adolescent. Mobility rate has clear advantages over total relocations in determining the extent of exposure to military relocation stress. Several studies had found the mobility rate to be more predictive than total relocations for adolescent adjustment ([316](#); [432](#)).

However, other studies have found mobility rate to be of negligible utility in predicting military relocation outcomes ([56](#); [128](#); [288](#)). Recency of relocation has also emerged as an important influence on military relocation stress ([144](#)). A relocation within the past year has been associated with depressive symptoms in adolescents ([128](#)). However, these depressive symptoms appear transitory and dissipate following a normative adjustment period of roughly 12 months ([166](#)). Given roughly a third of military adolescents relocate any given year it is probable a third

of any representative sample of military adolescents recently relocated. [Perreault et al \(288\)](#) found an association between mobility rate and military adolescent externalizing behavior was no longer significant when relocation recency (i.e., < 12 months) and parental stress were accounted for. Overall, this research suggests relocation recency and mobility rate are important factors to reconcile in the relocation outcome literature.

Relocation and the Adolescent: Summary and Conclusions

The stress of relocation and the well-established relationship between stress and health outcomes ([392](#); [400](#)) make it important to understand the possible implications of frequent relocations – especially in high-risk adolescent samples with psychological/physical vulnerabilities. The few studies which have incorporated multiple relocation factors have found nuanced associations between relocation and adolescent health outcomes ([56](#); [288](#)). However, these studies are difficult to generalize with U.S. military samples and excluded individuals at greatest risk for adverse sequelae related to PCS moves. As discussed, the U.S. military's family readiness system may be improved by allotting resources for surveillance and intervention to high-risk groups ([83](#)).

This study intends to examine the Military Relocation Stress model by incorporating the relocation factors discussed (see Figure 1) to explore both risk and resiliency to PCS moves. First, this study intends to use an inherently vulnerable adolescent sample with overweight and/or high-trait anxiety at-risk for developing a variety of physical and psychological conditions in later life. Second, the study will examine parenting distress and the quality of the familial relationships as candidate moderator variables for the hypothesized relationship.

Risk and Resilience in Military Youth

In the Military Relocation Stress model, individuals with greater vulnerability to stress and physical illness are the most likely to experience adverse effects following PCS moves. Military-connected youth with inherent physical and psychological vulnerabilities provide a valuable opportunity to test the Military Relocation Stress model. It is important to elaborate on the characteristics of this high-risk sample and discuss their implications on military readiness requirements. Specifically, this section will review the combination of psychological and physical vulnerabilities in adolescents who are at risk for developing disordered eating/obesity.

There is considerable evidence that anxiety, depression, disordered eating attitudes/behaviors, and weight status are highly inter-related and share complementary biological and social determinants ([301](#); [409](#)). Military-specific lifestyle factors, including PCS, may exacerbate these underlying psychosocial vulnerabilities thereby promoting an obesogenic environment for military adolescents ([260](#)). Similarly, those adolescents that are at risk for disordered eating attitudes and behaviors, and obesity, may be at increased risk for negative psychological and physical outcomes compared to those without those risk factors. Therefore, it is essential to understand the mutual reinforcing relationships between these biopsychosocial factors, the military lifestyle, and their ensuing implications for military readiness and adolescent well-being.

Pediatric Obesity, Overweight, and Weight Status

Over the past 50 years, the national prevalence of pediatric obesity has quintupled ([109](#)). Obesity is a chronic medical condition defined by excess adiposity in the body and has been associated with many deleterious health consequences ([394](#)). Pediatric obesity, and its clinical precursor overweight, is standardized by using the body mass index (BMI; kg/m²). Pediatric

overweight and obesity are defined by having a BMI between the 85th and 94.9th percentile and greater than the 95th percentile after adjusting for age and sex, respectively (205).

The causes of obesity are multifactorial with evidence of complex gene (135) and environment (203; 407) interactions. One prominent obesogenic factor for adolescents is a parent with obesity or overweight (203). This increased likelihood of adolescent obesity can be attributed to genetic loading or through an obesogenic environment with limited physical activity (63), unhealthy diets (65), or a stressful home environment (428).

Pediatric Obesity, Overweight, and Weight Status in Relation to Physical Health

Pediatric obesity and overweight are associated with a host of adverse health outcomes. Specifically, youth obesity has been linked to obstructive sleep apnea, diabetes, metabolic syndrome, and depression (401). Excess weight status also exacerbates concurrent chronic health conditions and unhealthy lifestyle behaviors – especially during adolescence. For example, the musculoskeletal development of high-BMI youth is associated with axis deviations in the lower extremities and functional gait impairment into adulthood (379; 434). This limitation to functional movement may foster lifestyle sedentariness, increased weight gain, and chronic orthopedic conditions (434). In addition, rapid youth weight gain has been associated with early pubertal development in both sexes. Early pubertal development, in turn, has a demonstrable physical, psychological, and social toll on adolescent youth (58; 423).

The presence of co-morbidities in obesity and overweight can be partially attributed to dysregulated lipid profiles, glucose metabolism, and indicators of inflammation (401). High-density lipoproteins (HDL) cholesterol – an indicator of cardiovascular health – is significantly lower for individuals with severe obesity (401). Insulin resistance is also markedly higher for youth with obesity as represented by greater indices of HOMA-IR (401). Lastly, severe obesity is

associated with increased inflammatory (i.e., C-reactive protein) markers ([92](#)). All of these biomarkers are recognized adult risk factors for hypertension ([73](#)), heart disease ([92](#)), and metabolic syndrome ([286](#)). Together these results support that high weight during youth is a particularly vulnerable period for the instigation or exacerbation of physical illness and can be partially attributed to underlying physiological derangement. However, in addition to physical ailments, there is also evidence of behavioral/psychological differences for youth with obesity or overweight ([401](#)).

Pediatric Obesity, Overweight, and Weight Status in Relation to Anxiety and Depression

Pediatric obesity and overweight have a robust, bidirectional association to anxiety and depression ([15](#); [304](#)). In a metanalytic review of psychological comorbidities correlated with childhood obesity, [Pulgaron \(301\)](#) found evidence for an association between obesity and internalizing/externalizing disorders, including both depression and anxiety. In one meta-analytic review, obesity treatment interventions were associated with a reduction in both symptoms of depression and anxiety further supporting a causal relationship ([338](#)). Even in youth samples without obesity/overweight, higher levels of BMI have been associated with mood disorders and higher levels of perceived stress ([319](#); [401](#)). Other studies have found null effects or even inverse relationships to the proposed link between weight status and psychological illness ([319](#)).

[Pulgaron \(301\)](#) attributed part of this discrepancy to the under-examination of resilience factors and discussed several intervening psychosocial variables (i.e., family support/weight stigma) to explain the heterogeneity between studies. For example, [Lim et al \(221\)](#) contrasted several family/parental and child characteristics in a youth sample with obesity/overweight. The study authors found that, amongst youth with obesity/overweight, clinically significant anxiety was associated with greater body dissatisfaction and parental distress. In other studies, adolescent

weight status is associated with lower evaluations of physical appearance, interpersonal effectiveness, and self-esteem ([120](#); [155](#)). In turn, depression and anxiety are highly comorbid ([10](#)) and have also been linked to perceptions of physical appearance ([416](#)), the quality of interpersonal relationships ([232](#)), and self-esteem ([103](#)). This evidence suggests excess weight status shares many common biological and social determinants consistent with depression and anxiety-related pathology and, under certain conditions, weight status and psychopathology may be mutually reinforcing.

Obesity, Overweight, and Weight Status in Military Youth

There is limited surveillance on the prevalence of obesity and overweight in military populations. Of the available data, estimates suggest that approximately 24.7% to 30% of military youth meet the criteria for obesity or overweight ([25](#); [109](#)). The prevalence of obesity and overweight in military youth is roughly equivalent to nationally representative samples ([109](#); [276](#)). Although obesity and overweight are a public health issue for all Americans ([276](#)), there is considerable evidence that military youth identifying as female or belonging to ethnic/racial minorities are disproportionately affected ([50](#); [334](#)). These findings are consistent with nationally representative data which finds obesity rates are higher for Hispanic and non-Hispanic Black adolescents compared to non-Hispanic White and Asian adolescents ([279](#)).

These prevalence rates for military youth persist despite universal healthcare (i.e., TRICARE) and resources afforded by extant policy from the military family readiness system ([362](#)). This suggests current policy is insufficient or military settings promote high weight status through several obesogenic factors. Indeed, military communities are thought to contribute to an obesogenic environment thereby increasing the risk for excess weight gain for military adolescents ([394](#)). Certain military communities may be defined as “food deserts” or areas

lacking in access to healthy and affordable food ([108](#)). Despite food assistance programs ([225](#)), some research suggests some members of the military community adopt unhealthy diet/feeding practices due to the proximity and convenience of calorie dense fast foods ([369](#)). For example, servicemembers across three Army installations reported their perceived barriers to weight management as the proximity/density of fast-food outlets, the cost of nutritious foods, poor food quality, and a stressful operational tempo ([72](#)). In another study, a sample of veterans with overweight or obesity endorsed military-specific eating behavior, feeding practices, and food scarcity were considered to influence their post-service weight status ([369](#)).

Given the known association between parental and adolescent weight status, the increasing incidence of servicemember overweight and the obesogenic military environment may place military adolescents at-risk for excess weight gain ([260](#); [394](#)). Further, there is preliminary evidence that community transmission of these unhealthy eating and dietary practices may dispose military adolescents to increased weight gain in later life ([313](#)).

Pediatric Disordered Eating Behavior and Attitudes

Disordered eating attitudes and behavior are common in youth populations: estimates of sub-clinical eating/weight-related attitudes and behaviors range from 33% to 57% in girls and 15% to 31% in boys ([87](#); [167](#); [270](#)). Although diagnoses of more severe eating and feeding (e.g., anorexia/bulimia nervosa, binge eating disorder) pathology are less common ([389](#)), the symptoms of disordered eating attitudes and behavior tend to commence during adolescence with prodromal symptoms arising as early as middle childhood ([211](#); [393](#)). For example, [Swanson et al \(389\)](#) reviewed a nationally representative cohort of adolescents to find anorexia nervosa, bulimia nervosa, and binge-eating disorder (BED) had a median age of onset at approximately 12

years of age. The instigation of pathological eating around adolescence suggests youth are a viable target for intervention.

BED is especially prevalent amongst adolescents with overweight or obesity with some estimates as high as 22.2% ([164](#)). Binge eating symptoms have also been associated with reduced psychological health and weight gain ([150](#)); to include higher levels of anxiety and depression, as well as reduced self-esteem ([183](#)). The hallmark characteristic of BED is binge eating, which must meet the following criteria: 1) the consumption of an objectively large quantity of food in a discrete period of time; and 2) the perception of lacking control during the eating episode ([20](#)). Historically, there has been significant contention over the first criteria as to what exactly constitutes an “objectively large quantity of food” ([271](#)). Available research suggests the objective/subjective binge meal size may be less important for prognosis and severity than other factors ([363](#)). As a result, researchers have increasingly focused on the second criterion for binge eating: Loss of Control (LOC) eating ([59](#)).

LOC eating is the subjective feeling of being unable to control the quantity of food consumed irrespective of the meal size ([393](#)). Community estimates of adolescent LOC eating have ranged 23.3% to 28.4% ([111](#); [343](#)). Youth with higher BMI, especially adolescents with overweight or obesity, are at even greater risk for LOC eating ([164](#)). Indeed, in one prospective study examining children at high risk for adult obesity, [Tanofsky-Kraff et al \(398\)](#) found the presence of LOC eating – regardless of the amount of food consumed – was the most prominent predictor for excess weight gain over an eight-year study period.

The implications of LOC eating for the physical and psychological health of adolescent youth are significant. Numerous cross-sectional and prospective studies support youth LOC eating predicts greater disordered eating attitudes/behaviors, excess weight status,

psychopathology, and physiological derangement ([2](#); [250](#); [373](#); [398](#)). The interpersonal model of binge eating disorder has been proposed as a theoretical basis for many of these observed associations between LOC eating and its deleterious health outcomes ([111](#); [396](#)). The interpersonal model posits socio-evaluative stressors elicit negative affect which, in turn, trigger LOC eating episodes. Studies in both laboratory and naturalistic settings have supported negative affect as a key mediator in the instigation of LOC eating episodes ([306](#); [307](#); [356](#)). Entry into adolescence is a recognized stage of psychosocial development wherein youth are acutely influenced by social stress of their peers and community ([440](#)). Accordingly, there is evidence that variables salient to socio-evaluative stress during childhood/adolescence, such as higher shape concern and weight-based teasing, predict LOC eating ([174](#)).

Pediatric Disordered Eating Behavior and Attitudes in relation to Physical Health

Notwithstanding the clear implications for psychological distress, disordered eating behavior and attitudes are associated with poor physical health, dysregulated metabolism, adverse cardiovascular events, and – in extreme cases – death ([20](#)). Even sub-clinical or prodromal presentations of disordered eating behavior in youth have implications on physical health ([398](#)). With the rise in rates of obesity, metabolic syndrome, and diabetes in vulnerable youth, there has been increasing interest in modifying disordered eating behaviors to alleviate the impact of these chronic conditions ([94](#)). LOC eating, in particular, has been identified as a viable point of intervention for improving obesity and metabolic health ([397](#)).

LOC eating has been both cross-sectionally and prospectively associated with metabolic dysfunction in youth ([174](#); [359](#); [398](#)). For example, [Byrne et al \(60\)](#) examined a sample of non-treatment seeking youth to find anxiety was associated with fasting insulin and insulin resistance, but only for those youth with LOC eating. In a complementary study, [Radin et al \(305\)](#) used a

mixed sample of treatment seeking and non-treatment seeking adolescents to determine the influence of LOC eating on metabolic functioning. The study authors found that LOC eating was associated with higher systolic blood pressure and greater levels of LDL cholesterol, even after adjusting for sociodemographic characteristics, adiposity, and treatment status.

A third study suggests the relationship between LOC eating and metabolic functioning may be causal: treatment-seeking female youth whose LOC eating remitted at a 6-month follow-up exhibited greater levels of HDL cholesterol, reduced triglycerides, and reduced glucose levels ([359](#)) when compared to those whose LOC persisted. Lastly, increased inflammatory markers (i.e., C-reactive protein) are also associated with LOC eating and poor diet selection ([358](#)). The human immune system and metabolic functioning are highly interrelated ([179](#)) and therefore a plausible mechanism by which LOC eating could lead to chronic metabolic dysfunction. In aggregate, there is reasonable evidence to support LOC eating influences certain aspects of youth metabolic functioning even after adjusting for adiposity. Metabolic dysregulation, in turn, is associated with a host of adverse physical illnesses and chronic conditions ([179](#); [286](#)).

Pediatric Disordered Eating Behavior and Attitudes in relation to Anxiety and Depression

In further support of the interpersonal model of LOC eating, there is a plethora of evidence to support LOC eating in youth is associated with depression and anxiety – two related constructs of negative affect. Several prospective studies suggest pre-adolescent LOC eating can be viewed as an early behavioral marker of disordered mood. [Sonneville et al \(373\)](#) conducted a prospective examination of a cohort of adolescents and young adults to find LOC eating was predictive of overweight/obesity and greater depressive symptoms. However, evidence in pre-adolescent samples suggest depression may be more critical to the maintenance of LOC eating in adolescence. For example, in one prospective study of community youth conducted by [Hilbert et](#)

[al \(174\)](#) found pre-adolescent LOC eating predicted the development of partial BED and eating disorder psychopathology. However, Hilbert and team could not demonstrate a prospective relationship of LOC eating on depressive symptoms. Similarly, [Tanofsky-Kraff et al \(395\)](#) found that while baseline LOC eating predicted anxiety at follow-up, the same relationship was not evidenced for depression. Rather, the persistence of LOC eating from baseline through to follow-up was associated with increased depressive symptoms. [Hilbert et al \(174\)](#) suggested that as salient socio-evaluative stressors (e.g., weight concern) decrease, depression – another aspect of negative affect – may increase likelihood for LOC eating. Taken together, these results suggest an adolescent’s social environment interacts significantly with youth’s inherent vulnerability to negative affect thereby instigating and/or exacerbating LOC eating.

Disordered Eating Behavior and Attitudes in Military Youth

Despite the limited data on eating pathology in military samples – especially for military youth – there is compelling evidence that diagnoses of eating-related pathology in the military health system have increased over the past 20 years ([19](#); [260](#)). A recent survey of active duty servicemembers found the crude annual incidence rate for eating disorder diagnosis increased by 44.7% from 2013 to 2016 ([438](#)). Despite the increase in eating-related pathology, diagnoses of eating-related disorders are still comparatively rare in military servicemembers when compared to civilians ([438](#)). It has been proposed that formal diagnoses under-report the prevalence of disordered eating in military servicemembers. Indeed, multiple studies demonstrate military servicemembers endorse sub-threshold disordered eating behaviors at rates greater than their civilian counterparts ([291](#); [429](#)).

Members of the military family may also be at greater risk for disordered eating behaviors/attitudes than their civilian counterparts ([260](#)). Amongst a sample of treatment-seeking

female adolescents, [Schvey et al \(348\)](#) found military-connected females experienced disordered eating attitudes/behaviors and more severe depression than a matched civilian sample. While the majority of research has focused on adolescent females, there is also evidence suggesting military adolescent males are at comparable risk for eating-related pathology and psychosocial stressors ([303](#)). The limited data available from non-clinical military adolescent samples is similarly concerning: [Waasdorp et al \(422\)](#) found 21% of military adolescents met the criteria for disordered eating which surpasses prevalence estimates in comparable civilian samples ([164](#)).

The Impact of Military Service on Adolescent Weight Status and Eating

Behavior/Attitudes

The U.S. military environment and culture may contribute to an obesogenic environment thereby increasing the risk for excess weight gain and eating-related pathology in military adolescents ([260](#)). Military culture has its own norms, communities, values, language, and class structure distinct from the conventional American milieu ([259](#)). In line with the unique nature of military culture, there is a corresponding set of social demands and expectations inherent in military service. As discussed, the unique contribution of these military lifestyle factors is thought to influence the observed rates of adverse health outcomes – include mood and anxiety disorders ([260](#)).

Military-connected youth are exposed to these lifestyle experiences by virtue of their familial ties and experiences within the larger military community. Research in military samples supports community and familial transmission of obesogenic factors and disordered eating behaviors/attitudes ([252](#); [260](#); [422](#)). Embedded expectations and societal stigma have both been proposed as an explanation for the increasing prevalence of disordered eating and obesity in military populations ([52](#); [244](#); [255](#)).

Weight stigma has also been identified as prominent psychosocial contributor to obesity and pathological eating in military samples ([260](#)). Indeed, weight-based stigmatization is highly prevalent in U.S. society ([17](#)) with demonstrated implications for physical and psychological health ([13](#); [110](#)). Weight stigma broadly denotes a range of negative cultural/societal attitudes and prejudices towards individuals of high weight status ([300](#)). Military culture highly values individual fitness, military bearing, and athletic performance. Accordingly, the Armed Forces have strict weight/fitness standards which are assessed in accordance to service-specific guidelines ([246](#)). Although these fitness requirements are necessary for arduous duty, these values may have the unintended consequence of fostering weight-related bias during promotions, evaluations of competency, and foster workplace discrimination ([71](#); [130](#)). A burgeoning body of literature suggests military servicemembers – due to stringent weight and fitness requirements ([246](#)) – may be particularly vulnerable to internalized weight stigmatization and the associated obesogenic consequences ([298](#); [299](#); [347](#); [387](#)).

Sizable minorities of servicemembers endorse organizational pressures for athletic performance and physical appearance ([422](#)). As weight status increases, servicemembers report greater rates of weight stigmatization and the associated consequences. [Schvey et al \(345\)](#) examined a sample of military servicemembers with overweight/obesity to find nearly half had experienced at least one instance of weight-based stigmatization. In the same sample, the study authors also found significant associations between weight stigma and depressive symptoms, eating in response to anxiety/anger/depression, and maladaptive coping. In another study of military servicemembers, [Shank et al \(357\)](#) found military-specific weight stigma was associated with worse physical health in adult servicemembers as indicated by the presence of a self-reported medical condition.

The prevalence of weight stigma, and the associated impact on psychosocial functioning and weight gain, has also been replicated in samples of military adolescents ([347](#)). For example, in one sample of military adolescents at-risk for binge eating disorder and adult obesity, [Pearlman et al \(285\)](#) found approximately 48% reported family (i.e., parents/ siblings) weight-based teasing. Family weight-based teasing, similar to studies of adult servicemembers ([345](#)), was also associated with poorer social functioning, self-esteem, and depressive symptoms ([285](#)). The correlates of weight stigma are well-recognized obesogenic factors, further supporting military settings may predispose military adolescents for excess weight gain and pathological eating.

In addition to weight stigma, there is evidence that marginalized racial/ethnic groups and gender minorities are disproportionately affected by obesity and experience reduced psychological health ([139](#); [248](#); [346](#)). Indeed, [Nolte et al \(273\)](#) examined a national sample of young adults to find evidence racial minorities and women were less likely to meet military accessions standards. The pathways by which these health disparities emerge is varied ([315](#)), but may be partly attributable to discrimination and systemic inequality ([124](#); [249](#)). With over 36% of U.S. military personnel identifying as racial/ethnic minorities, it is important to acknowledge the implications discrimination and stigma may present for current and prospective servicemembers ([50](#); [394](#)).

Considering all of the potential impacts of LOC, overweight and obesity on the biopsychosocial functioning of military adolescents, a population at risk for LOC, overweight and obesity may be at particular risk of negative outcomes when exposed to the stressors of a PCS. Therefore, it is particularly important to study this population and to consider factors that may promote resilience.

Resilience in Children and Adolescents

PCS moves are normative stressors which may not necessarily have negative long-term repercussions. Therefore, it is also critical to understand the pathways by which military relocation stress can lead to positive adjustment. Childhood and adolescence are not only sensitive developmental windows for dysregulation and illness, but are also opportunities for exhibiting positive adjustment and resilience in the face of adversity. The traditional paradigm of health research has been to investigate illness to the detriment of the processes supporting healthy functioning. To address this gap, the construct of resilience has permeated across numerous disciplines as a systematic methodology to investigate positive adaptation while under significant adversity.

According to the Military Relocation Stress Model (Figure 1), familial/community processes and parental psychopathology are key factors related to military adolescent's psychosocial adjustment to PCS moves. It is important to describe how the family network serves to foster risk or resilience to PCS moves. The following section will define resilience as it relates to military relocation, distinguish resilience from trait resiliency, and briefly outline the rationale of family processes as an important resilience factor.

The Resilience Framework

Resilience is the positive adaptation of an organism undergoing significant adversity ([129](#); [230](#)). Several aspects in this succinct definition warrant further elaboration as it relates to the current study. First, resilience requires a stressor of sufficient magnitude to warrant an adaptation. Sub-threshold stressors that are not salient to an individual may fail to elicit compensatory adjustment ([129](#)). Not all military adolescents will find PCS moves as stressful events to warrant either positive or negative adaptation. Therefore, it is valuable to examine a

sample vulnerable to psychosocial stress to demonstrate both risk and resiliency for post-PCS psychosocial adjustment.

Second, resilience is dynamic and fluctuates across time and development. In this regard, trait “resiliency” is conceptually distinct from dynamic “resilience” (230). Modern theorists tend to avoid trait resiliency due to the possible consequences of stigmatizing at-risk adolescents (22). Instead, resilience is contextual: a dynamic interaction between stress, resilience factors, and a specific evaluation of adaptation. For example, a child’s trait “resiliency” may be a consequence of strong mentorship by a valued teacher or through the support of an institution (e.g., school lunch). As a result, the child’s resilience to adversity is not static, but expected to fluctuate as different stressors and extrinsic resilience factors interact. The present study seeks to emulate the resilience framework (as opposed to trait resiliency) by examining the contextual relocation factors thought to contribute to risk or resilience.

Resilience Factors: Compensatory and Protective Models

Resilience factors are measurable aspects of the resilience process (126). These resilience factors can be modeled through either a compensatory or a protective model. Both models have significant empirical support and are used to describe the specific nature of the interaction between resilience and risk factors (125; 126). The compensatory model is when a resilience factor is posited to have a direct, independent effect to counteract a known risk factor. In the protective model, the resilience factor (i.e., assets or resources) moderates the risk factor, and only attenuates risk in the presence of a stressor. See Figure 4 for graphical depictions of the compensatory and protective models of resilience.

Protective processes are generally more appropriate to the resilience framework since compensatory processes exhibit their positive effects regardless of latent risk. Indeed, a few

notable studies have suggested specific familial/community factors can serve as protective processes to buffer the adverse impact of PCS moves ([56](#); [288](#)). Indeed, the relative inconsistency in the literature may be related to the presence of risk and resiliency factors which determine an adolescent's response to military relocations stress.

Family Systems Approach of Stress and Resilience

Resilience factors are not static measurements of risk or positive adaptation but interpreted as a dynamic process within a cohesive theoretical framework. Family functioning and composition is recognized as one such resilience factor during childhood and adolescent development ([245](#)). As a result, family stress and resilience models have been widely incorporated into research and intervention in both military and civilian populations ([333](#)).

The systems perspective of family resilience emphasizes the processes which serve to strengthen families to overcome adversity. This strengths-based system avoids the traditional stigma associated with parenting as it focuses on processes in lieu of characteristics/risk factors ([245](#); [284](#)). The model relies on the premise that families are able to maximize resilience by focusing on idiosyncratic expressions of several key family processes. [Walsh \(425\)](#) placed these resilience processes into three general categories: 1) a shared belief system; 2) organizational patterns; 3) and communication/problem solving skills. A shared belief system incorporates meaning-making of stressors, optimism, and spirituality. Organizational patterns include the subcategories of connectiveness, flexibility, and financial resources. Lastly, communication/problem solving is composed of emotional expression, collaboration, and clarity in communication. Due to the emphasis on processes, the systems perspective is amenable to interventions designed to foster family resilience in both civilian and military populations ([332](#); [333](#)). Interventions designed to enhance family resilience has been demonstrated to be effective

in reducing parent and child distress ([219](#); [220](#)). Further research has supported reduction in distress of a child is mediated by improvement in family resilience processes ([332](#)).

A complementary method of conceptualizing family resilience is through the assessment of family stress. According to a family systems approach to resilience, chronic family stress occurs in the absence or relative weakness of family resilience processes ([425](#)). Therefore, family stress constructs provide ancillary evidence for the presence (or lack thereof) of family resilience. The construct of parenting stress is commonly used to assess family stress by examining child characteristics, parent characteristics, and situational/demographic life stress ([1](#)). Factor analytic studies suggest parenting stress loads onto two constructs: parental pathology and dysfunctional parent-child interactions ([162](#)). Indeed, several studies support higher levels of family resilience are be linked to reduced parenting stress – especially in vulnerable youth populations ([147](#); [294](#); [364](#)).

Risk and Resilience: Summary and Conclusions

The Military Relocation Stress model endeavors to predict how military adolescents will respond to relocation stress. The few studies which have incorporated multiple relocation factors have found nuanced associations between relocation and adolescent health outcomes ([56](#); [288](#)). However, these studies are difficult to generalize with U.S. military samples and excluded individuals at greatest risk for adverse sequelae related to PCS moves. To address this gap, it is necessary to examine those U.S. adolescents most likely exhibit poor adaptation following the first and second-order disruptions of a PCS move. As discussed, the U.S. military's family readiness system may be improved by allotting resources for surveillance and intervention to these high-risk groups ([83](#)).

The Military Relocation Stress model suggests that the interactions between these risk and resilience factors predict biopsychosocial stress responses. The next section summarizes the potential mechanisms and impact of PCS-related stressors, LOC eating, and weight on biopsychosocial stress responses.

Stress Mechanisms and Consequences

Military relocation stress is a necessary and natural consequence of first and second-order disruptions during PCS moves. Although some degree of stress is normative, repeated chronic stress contributes to physical and psychological illness. To understand the sequelae associated with PCS moves, “stress” must be defined in relation to its constituent physiological/psychological components and how these processes contribute to chronic illnesses. In addition

Stress is a biopsychosocial process that has a profound impact on behavior, affect, and cognition. Living organisms are complicated systems requiring a flexible but reliable internal state (e.g., body temperature) to adapt to environmental demands ([337](#); [355](#)). This ideal, dynamic internal state is known as homeostasis and is evidenced by the narrow range of biological set-points which set the conditions for life. Stress itself is a self-regulative process to restore homeostatic balance in response to perceived or real external demands. Stressors elicit a disruption in the homeostasis of a particular organism which results in the organism coordinating a stress response to restore homeostatic balance ([337](#)). Although the core tenets in the original formulation of homeostatic stress are valid today, influences from the social sciences advanced stress beyond the classical model of homeostatic balance.

The Allostatic Stress Response: Physiological Mechanisms

Allostasis was proposed to remedy the limitations of homeostasis by proposing a dynamic, active system which predicts and adjusts the body’s physiology. Social scientists’ work

with appraisal, affective states, and cognitions elevated the role of the central nervous system (CNS) in modulating a dynamic response to the environment. It became evident that without CNS mediation, localized neuroendocrinological models are unable to account for the diversity in individual responses ([142](#)). Allostasis better incorporates the brains' role in mediating the onset, maintenance, and outcome of the stress response ([382](#)).

The allostatic model provides a robust physiological framework to understand how PCS moves can result in adverse health outcomes. Allostasis effectively models: 1) the dynamic process between internal resources (e.g., coping) and external demands; 2) anticipatory adjustment to stressors; 3) adaptation over periods of prolonged stress; 4) and stress' role in ecologically consistent models ([142](#); [382](#)). The allostatic model accomplishes this by proposing two interacting systems of accommodation: central allostatic accommodation and peripheral allostatic accommodation (see Figure 2). It is the dynamic interaction between both systems of accommodation which allows for adaptive predictions and responses to external demands.

Central and Peripheral Allostatic Accommodation

The disruptions associated with PCS moves cause alterations in central allostatic regulatory systems designed to adapt to military relocation stress. Central allostatic accommodation is associated with the core affective, regulatory, and sensory processing regions of the brain ([382](#)). Central allostatic accommodation serves as the primary mediator of environmental stressors by exerting executive control and command over the entire stress response- including peripheral allostatic accommodation ([113](#); [142](#)). In this executive capacity, central allostatic accommodation controls the existing neurocircuitry in the brain to formulate emotions. Emotions are discrete motivational states which serve as expression of central allostatic accommodation to guide human adaptation ([142](#); [214](#)). The neural circuitry

undergirding emotional processing is generated in the brain and integrated across all CNS functions including perception, behavior, and cognition ([290](#)). As emotional stimuli increase in intensity or salience, the brain enlists added neural circuitry to facilitate allostatic accommodation ([158](#)). Although emotional processing is distributed throughout the brain, functioning is grossly localized in the basal ganglia, extended amygdala, and other associated brain regions ([142](#); [214](#)).

According to the two-stage model of allostatic accommodation, mental health outcomes are synonymous with the efficiency of central allostatic accommodation ([142](#)). As such, positive mental health is the product of a well-synchronized network: each constituent element of central accommodation (i.e., the cognitive and emotional neural circuitry) operate in-tandem to adjust human behavior. Conversely, poor mental health outcomes occur when these same systems meet extreme adversity or are otherwise dysregulated through chronic activation ([6](#); [178](#)).

The PCS move process results in peripheral allostatic accommodation to meet the demands caused by first and second-order disruptions. Peripheral accommodation mobilizes several complementary physiological systems to guide adaptation to external demands and provides feedback to the central allostatic system ([142](#)). As central accommodation is synonymous with mental health, physical health is likewise reflected in the efficiency of peripheral accommodation. The most significant physiological systems at work in peripheral accommodation are two complementary neuroendocrine axes: the sympathetic-adrenal-medullary (SAM) axis response and the hypothalamic-pituitary-adrenal axis (HPA) response ([151](#)). These two systems operate in concert to satisfy general adaptation to a stressor. The SAM response is a rapid short-term adaptation which complements the HPA's comparatively slower, sustained response. Although other physiological processes (e.g., beta-endorphins) are operative

in the stress response, the SAM/HPA axes are considered the predominant and most well-researched mechanisms (78).

The SAM response mobilizes energy stores, modulates blood flow/circulation, suppresses restorative functions, and increases alertness to meet the external demand (151). These physiological changes are designed to prepare an organism to engage or withdraw (i.e., “fight-or-flight”). Sympathetic nervous system (SNS) innervation is the key reason for the immediacy of the SAM response. The SNS stimulates the adrenal medulla to secrete catecholamines (i.e., epinephrine, norepinephrine, and dopamine), which largely mediate the physiological alterations (337). Following the withdrawal of the stressor, the parasympathetic nervous system will enact a compensatory relaxation response (i.e., “rest-and-digest”) to restore homeostatic balance.

The HPA complements the adrenomedullary response by coordinating sustained neuroendocrinological activity. A hormone cascade signals each component of the HPA axis to culminate in a release of stress hormones from the adrenal cortex (151). Cortisol, the body’s primary stress hormone, alters how the body regulates macronutrients, inflammation, blood pressure, glucose, and the sleep/wake cycle. The relative slowness of the HPA axis is due to the circulatory system’s sluggish dispersal of hormones throughout the body. However, the addition of the slower HPA response allows a more sustained physiological effect on adaptation. The outputs of the HPA axis initiate an inhibitory feedback loop reducing the antecedent hormones which initiated the cascade (371).

Allostatic Overload and the Mental and Physical Consequences

The impact of PCS moves on adolescent biopsychosocial functioning cannot be understood without a method of distinguishing unhealthy, chronic stress from benign, adaptive stress. PCS moves may create enduring disruptions contributing to chronic dysregulation of

allostatic regulatory systems. The cumulative toll of repeated, excessive, and/or dysregulated accommodation can lead to allostatic overload ([337](#)). McEwen (2008) termed the harmful influence of chronic stress as “allostatic overload” to differentiate it from adaptive stress (i.e., allostasis). Allostatic overload specifically refers to the systemic “wear-and-tear” associated with extreme environmental stressors or the inability to appropriately regulate the stress response (see Figure 3). Allostatic load offers a compelling explanation for how stress eventually becomes embodied as physical and psychological illness.

Central allostatic accommodation is particularly susceptible to dysregulation and “wear-and-tear” when chronically activated ([142](#)). Effects of allostatic overload are most apparent in the functional and morphological alterations of the brain’s core emotional regions ([68](#)). The amygdala serves as a useful case example as to how severe or chronic stress leads to lasting morphological changes. In animal models, induced stress enhances synaptic connectivity in the basolateral amygdala ([256](#)) and alters transmission at the neuronal level ([3](#)). The association between chronic stress and amygdala morphology has been similarly demonstrated with human participants ([8](#); [153](#); [261](#); [427](#)). In turn, these morphological changes in neuronal architecture result in persistent functional changes in measurable brain activity, neuroendocrinological correlates to stress, and behavior ([141](#); [146](#); [168](#); [342](#)). Under sufficient allostatic load (i.e., stress) these changes become permanent and are expressed by impaired mental health ([312](#); [322](#); [388](#)).

The physiological responses from the SAM and HPA axes are adaptive to meet external demands, but can have deleterious effects when chronically activated. For example, catecholamines indirectly damage the cardiovascular system resulting in adverse events such as stroke or heart attack ([4](#)). The damage associated with allostatic overload can lead to enduring

changes in human organ systems (e.g., circulatory/ nervous systems). Aside from systemic “wear-and-tear,” chronic dysregulation of stress hormones can also lead to structural changes in the brain and debilitating medical conditions including: insomnia, diabetes, and obesity ([151](#)). Military adolescents may also be particularly susceptible to chronic alterations in allostatic regulatory systems in a process known as biological embedding. Not only do military youth have unique psychosocial stressors contributing to allostatic load, but these stressors concurrent to normative developmental milestones, such as childhood and adolescence, can result in persisting allostatic dysregulation into adulthood ([91](#)). Past work suggests that early adversity interacts with the maturing brain to produce distinct, time-sensitive risk windows for allostatic dysregulation ([165](#)). Population health researchers have proposed biological embedding to explain the lasting health consequences of early life adversity ([169](#)).

Biological embedding is the process by which transient, environmental stressors during sensitive periods of development enduringly alter an organism’s functioning. It is thought durable changes in allostatic regulation are responsible for how early adversity “embeds” in an organism. Past work has documented the lasting physiological changes in neurological, immune, endocrine, autonomic, and metabolic systems after exposure to early adversity ([37](#)). Further, increased adaptive plasticity during periods of development has been noted in neurological ([133](#)), immune ([23](#)), and metabolic systems ([254](#)); all key components of allostatic regulatory systems. These well-documented psychobiological alterations in response to stress during childhood and adolescence are complicated by limited understanding of the mechanisms. Much of the historical research has focused on correlates of central allostatic accommodation (i.e., emotions/psychological constructs; [90](#)). Increasingly, ecologically-oriented approaches

incorporating gene-environment interaction have been proposed to describe how stress becomes “embedded” into the functioning of a developing organism.

Epigenetic control of gene expression has been suggested as a mediator for adaptive plasticity in biological embedding ([37](#)). Encoded in every gene is the information necessary to produce a specific protein. Controlling gene expression enables the human body to adjust functioning at the cellular (e.g., neuronal) level ([323](#)). The epigenome is sensitive to modification by exposure to environmental stimuli, can be inherited, and – in some contexts- reversed ([323](#)). A study by [Szyf et al \(390\)](#) utilized an animal model to explore the epigenetic effect of parental nurturance on methylation of hippocampal glucocorticoid gene promoters. Szyf and colleagues found that the differential epigenomic state persisted into adulthood unless reversed behaviorally or with an infusion of a methyl inhibiting enzyme in adulthood. The results are similar, albeit less conclusive, for studies with human populations: early exposure to stressors modifies methylation and gene expression with important implications for human health and well-being ([114](#); [188](#); [323](#); [444](#)).

The Pathophysiology of Stress: Implications for Physical and Psychological Health

The downstream impact of military relocation stress in adolescent youth may result in adverse physical and psychological health. Allostatic overload has been proposed as an explanation as to how psychosocial stressors – such as relocation – eventually progress into chronic illness. According to [McEwen \(240\)](#), correlates of allostatic regulation (i.e., catecholamines, cortisol, blood pressure, etc.) are demonstrated measures of acute allostatic load within the body and follow a predictable pattern of dysfunction.

The pathophysiological parameters for allostatic load include outputs of the neuroendocrine axes (e.g., HPA/SAM), cardiovascular systems, anthropometrics, and metabolic

processes ([54](#)). Stress hormones, the primary mediator of the stress response, are the first to become dysregulated. Secondary mediators, correlates to the metabolic and cardiovascular systems, respond to regulate the imbalance caused by the primary mediators' chronic or excessive activation. The ensuing dysregulation of the primary and secondary mediators contributes to tertiary outcomes: adverse health and clinical diagnoses exacerbated by chronic activation of the stress response ([142](#)). Anxiety, depression, and aspects of metabolic syndrome in adolescent youth are all associated with stress-related biomarkers. The presence of similar pathophysiological stress responses in these conditions suggests that these nosologically distinct medical conditions share a common biological determinant.

Given chronic stress' known implications on health and that military youth - under certain conditions - experience significant distress following PCS moves, it is probable psychosocial stress will manifest as dysregulated biomarkers indicative of central/peripheral allostatic overload. In addition, the presence of existing physical and psychological comorbidities concurrent to a PCS move would place these youth at greater risk for allostatic dysregulation. This section will review the known associations between allostatic biomarkers and the tertiary outcomes of interest: aspects of MetS, anxiety, and depression.

Stress and Physical Health

In line with the allostatic model, PCS moves can cause stress which may induce peripheral allostatic overload. Physiological variables are expressions of peripheral allostatic accommodation which manifest as measurable correlates of the stress response ([142](#)). Allostatic regulatory systems governing the stress response contribute significantly to physiological functioning across immune, metabolic, inflammatory, and other associated biomarkers. Metabolic Syndrome (MetS) is an example of condition associated with systemic allostatic

dysregulation, and represents a constellation of medical biomarkers considered clinical precursors for heart disease, stroke, atherosclerosis, and type-II diabetes ([11](#)). MetS is defined as a cluster of at least three of the following: visceral obesity, insulin resistance, hypertension, low High-Density Lipoproteins (HDL) cholesterol, and high triglycerides ([443](#)). This condition is completely preventable but it still estimated to affect over 25% of the global population ([330](#)). In recent decades, metabolic syndrome has increasingly developed into a major medical concern for pediatric populations ([107](#); [138](#)).

Although the causes of MetS are multifactorial, chronic psychosocial stress and dysregulated allostatic regulation have been consistently linked with metabolic syndrome ([39](#)). Adults exposed to adverse childhood stressors are particularly vulnerable to MetS and other metabolic abnormalities ([91](#); [215](#)). In addition, various experimental paradigms have demonstrated induced stress leads to elevations in blood serum content consistent with the clinical markers of metabolic syndrome ([5](#); [95](#); [134](#)).

Adolescent Weight Status and Dyslipidemia

The influence of stress on weight status/dyslipidemia is multifactorial and bidirectional ([407](#)). For adolescents increased weight, adiposity, and altered body shape is normative as they develop into sexual maturity ([378](#)). However, adolescence is also a risk window for disordered eating, overweight, obesity, dyslipidemia and associated behavioral risk factors ([185](#)). As a result, adolescence is a natural period of profound alterations in energy intake, storage, and expenditure. The most immediate consequences of dysregulated energy intake, expenditure storage, and utilization are weight gain and dyslipidemia ([63](#)).

Dyslipidemia is a disorder of lipoprotein metabolism characterized by an excess or deficiency of lipoproteins. Lipoproteins facilitate the transport of hydrophobic fat molecules (i.e.,

cholesterol, triglycerides) throughout the circulatory system. Unsurprisingly, dyslipidemia is heavily associated with obesity and fat mass ([366](#)). Dyslipidemia in metabolic syndrome is classified as an excess of triglycerides, decreased high density lipoproteins (HDL) cholesterol, and/or increased low-density lipoproteins (LDL) cholesterol. Dyslipidemia is considered a risk factor for heart disease and other medical conditions ([39](#); [366](#)).

Studies by [Cardel et al \(63\)](#) and [Tomiyaama \(407\)](#) [ENREF 52](#) both suggest an adolescent's maturing stress response (i.e., the HPA axis) and related neurocircuitry is an important cause for lasting weight gain and dysregulated lipid metabolism. Indeed, the HPA axis is initiated by physical stressors - to include hunger ([289](#)). Evidence supports the association between childhood stress and obesity ([145](#)). Perceived stress is associated with higher body mass index (BMI), waist circumference, serum triglyceride levels, and cholesterol: all correlates to overweight and obesity ([39](#); [399](#)). Psychosocial stressors have been consistently associated with the development of dyslipidemia ([39](#)). Specifically, a number of stress measures have been linked to low HDL ([149](#); [302](#)), elevated triglycerides ([149](#)), and increased overall cholesterol levels ([198](#)). Indeed, there is limited evidence that certain stress-related psychiatric conditions contribute an effect independent of obesity/weight-related factors on dysregulated HDL, total cholesterol, and LDL ([112](#); [414](#)).

Plausible physiological mechanisms exist to justify cortisol and other stress hormones' influence on weight gain ([40](#); [272](#); [321](#)). Obesity is thought to be both a cause and effect of dysregulated SAM/HPA axes ([227](#)). The SAM response is often overactive in individuals with obesity, and especially those with high levels of abdominal obesity ([12](#)). The mechanism of action is still unclear, but it has been proposed that adipose tissue (i.e., fat) produces signaling adipokines which modulate SNS activity. In turn, excessive SNS activity is thought to impair β -

adrenergic signaling in adipocytes, thereby altering metabolic functioning to result in obesity and insulin resistance ([370](#)).

Similarly, HPA axis functioning is overstimulated and dysregulated in individuals with obesity ([227](#)). In the HPA axis, a hormone cascade prompts the paraventricular nucleus of the hypothalamus to secrete corticotropin-releasing hormone (CRH). In turn, CRH stimulates the anterior pituitary to release adrenocorticotrophic hormone (ACTH) thereby producing cortisol. Cortisol, a primary stress hormone, is responsible for attenuating the HPA axis via a negative feedback loop. In individuals with obesity, adrenocorticotrophic hormone (ACTH) is overstimulated and present at greater levels ([419](#)). This overstimulation can be exacerbated in obesity via 1β -HSD1, an enzyme partly responsible for inactivating cortisol. For individuals with obesity, enduring peripheral allostatic accommodation fosters more 1β -HSD1 in hepatic and adipose tissues. Inactivation of cortisol via 1β -HSD1 dysregulates cortisol's negative feedback loop preventing inhibition of the stress response ([287](#)). Cortisol has also been associated with increased accumulation of visceral fat in the abdomen ([264](#)). Visceral fat, in turn, is associated with dysregulated lipid metabolism in the form of greater cholesterol, lower HDL, and higher LDL ([228](#)). Taken together, this evidence suggests individuals with obesity, especially those with significant stores of abdominal obesity, have over-responsive HPA/SAM axes and are less able to regulate cortisol diminishing attenuation of the stress response ([227](#)).

Adolescent Hyperglycemia and Insulin Resistance

Hyperglycemia is medical condition characterized by an excess of blood sugars and is associated with a host of potentially fatal complications, insulin resistance, and diabetes mellitus ([51](#)). Insulin is a critical peptide hormone which regulates blood glucose by facilitating metabolism and glucose uptake ([436](#)). Insulin resistance is when normal or elevated levels of

insulin elicit a diminished response, and is a clinical precursor to the chronic insulin dysregulation associated with acquired diabetes mellitus (i.e., Type 2 diabetes). Blood sugar concentrations can be measured directly through glucose levels, insulin levels, or inferred through the presence of glycated hemoglobin (A1c) ([175](#)). The formation of A1c is directly related to the duration of hyperglycemia in the blood in the last three to four months, thereby serving as a proxy measure for dysregulated glucose and insulin functioning ([175](#)).

The preponderance of evidence supports bidirectional associations of stress on hyperglycemia, insulin resistance, and diabetes mellitus ([159](#)). Acute stressors enlist peripheral allostatic regulatory systems (e.g., HPA axis) to dramatically alter blood sugar concentrations in response to acute stressors ([235](#); [257](#)). Animal models also demonstrate that severe and chronic stress adversely impacts glucose/insulin metabolism ([335](#); [386](#)). The literature is equivocal in adult human samples ([39](#); [296](#); [386](#)). The influence of stress on glucose/insulin resistance is rarely studied without the presence of metabolic syndrome or comorbid medical complications. Regardless, numerous studies suggest stress influences hyperglycemia ([149](#)) and diabetes mellitus ([207](#); [274](#)). Stress' association with hyperglycemia, insulin resistance, and acquired diabetes mellitus is also found in adolescent populations. Pubertal development is associated with alterations in insulin sensitivity and glucose regulation ([45](#); [378](#)). Life stress, psychiatric illness, and stress hormones have all been associated with correlates of insulin resistance in adolescent populations. ([48](#); [181](#); [278](#)).

The HPA axis and its associated biomarkers have also been implicated in the pathogenesis of insulin resistance and diabetes ([97](#)). A primary mediator in the HPA axis, glucocorticoids (e.g., cortisol) are important regulators of energy metabolism in response to stress. For example, cortisol promotes hepatic gluconeogenesis and facilitates other

hyperglycemic hormones in order to increase the availability of energy substrates (i.e., glucose, lipids). In this capacity, glucocorticoids promote endogenous glucose production and indirectly attenuate insulin's metabolic actions ([16](#)).

There is evidence that this physiological process can be dysregulated leading to enduring changes to allostatic regulatory systems. For example, juvenile diabetics are less responsive to ACTH and have lower concentrations of cortisol ([360](#)). At first glance this blunting of the stress response can be seen as adaptive when one considers the limited availability of endogenous insulin to restore homeostatic balance. However, rapid alterations in blood sugar in-tandem with sluggish glycemic control could result in catastrophic damage at the cellular level. In contrast, adult-onset diabetes the HPA axis appear hyperactive ([297](#)) and is characterized by higher levels of both ACTH and cortisol ([70](#)). The discrepancy between juvenile and adult-onset diabetes reflects the differing etiology of the illness, and the complicated reciprocal relationships involved in the stress response and tertiary health outcomes.

Adolescent Hypertension

The human body relies on the circulatory system to regulate optimal blood pressure in response to external demands. In fact, blood pressure is a quintessential measurement of acute stress load ([337](#)). Dysregulated blood pressure can damage arterial walls and other associated regulatory systems. Hypertension is the medical term for excessive levels of arterial blood pressure resulting in numerous adverse health outcomes including reduced quality of life and cardiovascular complications ([265](#); [295](#); [318](#)).

Various indices of psychological stress (e.g., occupational stress, socioeconomic stress, and anxiety/depression) have reliably been associated with hypertension ([223](#)). However, this association is moderated by gender effects ([39](#)), health behaviors ([318](#)), and other factors ([170](#);

[277](#)). In other studies, the association between stress and hypertension is not present - especially in healthy populations ([123](#); [197](#)). Stress' association with adult hypertension is thought to have its origins in childhood and adolescence ([327](#)). Blood pressure is highly sensitive to the pubertal alterations in weight and height and it is thought that the normative changes in puberty share a common regulating mechanism ([411](#)). Studies have prospectively linked stress-induced blood pressure elevations during adolescence to eventual development of adult hypertension ([441](#)). Adolescent and childhood hypertension are strongly associated with adult hypertension, a genetic loading for elevated blood pressure, and increased susceptibility to mental stress ([118](#); [119](#)). Increased rates of overweight and obesity have corresponded to increases in adolescent hypertension over the past decade amongst children and adolescents ([265](#)).

A consistent bidirectional association between stress and hypertension exists ([192](#)). Several physiological pathways mediate the influence of stress on blood pressure including the neuroendocrine axes ([209](#)), vagal withdraw ([367](#)), and the immune response ([7](#)). Induction of acute stress reliably results in increased blood pressure in both human and animal models ([442](#)). Chronic and severe stress in animal studies has been shown to reliably result in lasting hypertensive states ([218](#)).

The pathophysiology of hypertension has been associated with biomarkers indicative of allostatic dysregulation. In terms of central allostatic accommodation, there is evidence hypertensive individuals are more likely to exhibit both global brain atrophy and atrophy in specific brain regions associated with executive function, memory, and attention ([152](#); [308](#); [424](#)). Similarly, elevations of cortisol and dysregulated negative feedback control have been implicated in structural brain abnormalities and neuropsychological deficits in cognitive function ([229](#)) ([46](#)). Taken together these findings implicate the allostatic stress response in the pathophysiology of

hypertensive patients. Indeed, [Gold et al \(152\)](#) found evidence that dysregulated feedback control of cortisol may partially explain the structural brain deficits in individuals with hypertension after adjusting for age, gender, and BMI.

Peripheral allostatic accommodation is also implicated in the pathogenesis of hypertension. Sympathetic nervous system activity, often measured via correlates of the SAM axis (i.e., epinephrine/norepinephrine), has been associated with pre-, early-, and established hypertension ([233](#)). Excessive innervation of SNS pathways has been demonstrated in hypertension – especially for females ([268](#)). Further, SAM axis activity has been demonstrated to increase human cell growth in vascular muscle tissues contributing to the formation of atherosclerotic plaques ([67](#));([233](#)).

Stress, Anxiety, and Depression

The onset or maintenance of depression may be impacted by military relocation stress. In support of this assertion, there is sufficient evidence to suggest a causal, bidirectional relationship between depressive disorders and stress ([160](#); [196](#); [224](#)). Depressive disorders are a broad category of potentially debilitating psychological conditions which share similar cognitive, somatic, and behavioral characteristics. Major depressive disorder – the prototypical depressive condition – is relatively common with a lifetime prevalence rate of nearly 16% ([20](#)). Stress-related research on depression has been studied extensively ([224](#)) and has generally followed a stress exposure paradigm.

The stress exposure model of depression posits that discrete life events precipitate depressive episodes ([224](#)). This finding has been widely demonstrated with acute and chronic stress ([160](#); [195](#)), adverse life events in youth and adulthood ([161](#)), and across different periods of development ([80](#); [82](#)). It appears the type and quality of the stressor influences the course and

onset of the depressive episode ([400](#)). For example, sustained chronic stressors are generally superior in predicting depression than acute measures of stress load ([242](#)). Stressors in the key developmental periods of childhood and adolescence appear to increase the risk of developing a depressive disorder in later life ([202](#)). Further, evidence for the stress exposure model is corroborated by findings that life stressors which are independent of any depression-related sequelae (e.g., sudden death of a parent/natural disaster) are most often associated with onset of an initial depressive episode ([361](#)). This stress exposure paradigm has been extremely valuable and inspired numerous stress-diatheses models of depression ([224](#)).

Further evidence for a robust relationship between stress and depression is evidenced by the comorbidity of anxiety and depression in adolescent youth ([89](#)). Among depressed youth, diagnoses of comorbid anxiety-related disorders are extremely common with estimates as high as 75% ([89](#); [446](#)). Even non-diagnostic assessments using dimensional measures have shown high correlations between anxiety and depression ([377](#)). Interestingly, only 10% to 15% of adolescent youth with a primary diagnosis of an anxiety-related disorder endorse comorbid depression ([24](#); [85](#)).

Researchers have proposed several explanations for the asymmetry between anxiety and depressive comorbidities ([143](#)). First, there is evidence of considerable diagnostic overlap in the symptoms and measures differentiating depression and anxiety. Incorporating modifications to reduce overlapping items/constructs in traditional diagnostic inventories only yields modest reductions in the shared variance in the trait constructs ([81](#)). Second, sequential morbidity of depression may be the consequence of anxiety activated in early childhood. The symptoms of anxiety are often over-represented in early childhood ([66](#)) and tend to predict the development of adolescent depression - especially for female youth ([292](#)). Accordingly, the sequelae of anxiety

may be indicative of prodromal depression manifesting into a full depressive disorder during adolescence ([143](#)). Lastly, there is considerable evidence of shared biological and social determinants undergirding both anxiety and depression. An underlying propensity for negative affect ([30](#)) and family risk history ([243](#)) have both been implicated in the etiology of both anxiety and depression.

The Pathophysiology of the Stress Response in Depression

All three putative explanations for the relationship between stress, anxiety, and depression posit underlying determinants with a common physiological foundation: the allostatic stress response. Theorists suggest depression – much like stress – confers several evolutionary advantages but may become dysfunctional through chronic or repeated stressors. This dysfunction can appear remarkably similar to those under severe or chronic stress ([217](#)). The HPA axis - a neuroendocrine pathway implicated in the stress response - has long been associated with the development, maintenance, and relapse of depression ([402](#)). Indeed, genetic markers coding for stress-related proteins have been found to be predictive of depression in response to major stressors ([64](#); [195](#)).

Despite robust support for HPA-axis dysregulation in depressive conditions, there is evidence for heterogeneity in effect sizes across studies ([199](#)). A possible explanation for this discrepancy is that standardized assessment of depression obfuscates specific depressive presentations whose symptoms may align more closely to peripheral allostatic accommodation and HPA-axis functioning ([137](#)). For example, one longitudinal study over a 14-year period found cortisol and C-Reactive Protein (CRP) were strongly associated with the somatic symptoms of depression while, after adjusting for confounders, were only weakly associated with cognitive-affective symptoms ([247](#)). There is also significant comorbidity between

depressive conditions and other chronic health conditions characterized by dysregulated peripheral allostatic accommodation (282). Taken together, this suggests an underlying basis for peripheral allostatic load contributing to somatic depressive symptoms.

FK506-binding protein 51 (FKBP51) has been identified as an important modulator of glucocorticoid receptors in response to environmental stress and provides a useful illustration of how pathophysiological HPA-axis functioning is related to numerous chronic medical conditions (447). In depressive conditions, glucocorticoid receptors often appear desensitized to the inhibitory feedback of cortisol resulting in hypersecretion of CRH (177). When bound to glucocorticoid receptors, FKBP51 reduces receptor affinity for glucocorticoids resulting in prolonged stress system activation (41). Upregulation of FKBP51 has been linked to increased recurrence of depressive episodes and treatment response to antidepressant medication (42). Indeed, genetic variations of FKBP51 have been associated with both central and peripheral allostatic accommodation via morphological alterations in several brain regions (410) and cardiac stress reactivity (226), respectively. In terms of other chronic medical conditions, FKBP51 has been implicated in the pathophysiology of metabolic functioning, obesity, and diabetes (27; 280). In summary, FKBP51's influence on numerous distinct medical conditions supports that anxiety, depression, and MetS share an underlying biological determinant.

Allostatic Load: Modeling Multi-Systemic Dysregulation

The available evidence suggests pathophysiological alterations to the stress response are pervasive in anxiety/LOC eating, depression, and MetS' constituent components. This pathophysiology may be mutually reinforcing as evidenced by the high comorbidity between these conditions and the co-occurrence of dysregulated biomarkers. A parsimonious explanation for these findings is that the etiology of these nosologically distinct disorders share a common

factor: a dysregulated allostatic stress response. Allostatic theory supports this asserting by proposing multisystem physiological functioning can be partially explained by the common factor of allostatic load.

The allostatic load index (ALI) has been proposed to effectively operationalize the cumulative physiological dysregulation caused by chronic activation of the stress response. The conceptual rationale for the ALI is in how these biomarkers become dysregulated in a predictable sequence ([26](#)). Stress hormones, the primary mediator of the stress response, are the first to become dysregulated. Secondary mediators, correlates to the metabolic and cardiovascular systems, respond to regulate the imbalance caused by the primary mediators' chronic or excessive activation. The ensuing dysregulation of the primary and secondary mediators contributes to tertiary outcomes: adverse health and clinical diagnoses exacerbated by chronic activation of the stress response ([142](#)).

The first validation of the ALI was conducted using a geriatric sample which incorporated the following ten biological parameters: cortisol, dehydroepiandrosterone (DHEA), epinephrine, norepinephrine, cholesterol, glycosylated hemoglobin, systolic/diastolic blood pressure, BMI, and waist-hip ratio ([351](#)). The study's findings suggested that ALI was prospectively associated with 7-year mortality, declines in mental/physical health, and cardiovascular illness. In addition, the ALI model had greater prognostic value than either individual biomarkers or the contribution of other diagnostic constructs (i.e., MetS).

Allostatic Load in Relation to Stress, Depression, and Physical Health

Since its original inception over 20 years ago, The ALI has been widely incorporated in numerous studies and its formulation has undergone significant scientific scrutiny ([190](#)). Indeed, [Guidi et al \(157\)](#) conducted a recent systematic review identifying a total of 267 original studies

incorporating allostatic load. Although the operationalizations of ALI are somewhat heterogeneous, systematic reviews find widespread support that various indices of ALI are associated with worsening physical and psychological health in both the clinical and general population ([157](#); [190](#); [237](#)). Cross-sectional and prospective studies in adult samples also demonstrate that indices of allostatic load are associated with stress ([74](#); [239](#); [293](#); [405](#)), depression ([189](#); [191](#); [200](#)), and worsening self-reported and actual physical health ([9](#); [34](#); [336](#); [351](#)), and these associations are persistent across cultures ([350](#)). There is also a wide body of evidence suggesting adverse childhood experiences (ACEs) are prospectively associated with higher allostatic load in adulthood ([28](#); [90](#); [188](#); [226](#)).

The allostatic load index has also been validated in pediatric populations with significant implications for a number of tertiary outcomes ([26](#); [116](#); [117](#); [320](#)). [Evans \(116\)](#) compared ALI to a measure of cumulative risk history in a sample of 339 youth from low-income families. The study authors found that an increase in cumulative risk history – denoted by the presence of physical, physical, and environmental stressors – was associated with a higher ALI. The prospective association between allostatic load and tertiary health outcomes in youth samples appears to be moderated by the presence of concurrent psychosocial stressors. After a four year follow-up, [Evans et al \(117\)](#) examined the same cohort to find a prospective relationship between cumulative risk history, allostatic load, and cardiovascular reactivity/recovery. After adjusting for the ALI at baseline, only youth with low maternal responsiveness evidenced an association between cumulative risk history and ALI. In another study, [Rogosch et al \(320\)](#) conducted an examination comparing rates of allostatic load between maltreated children and non-maltreated in a sample of low-income youth participating in a summer program. Childhood maltreatment and allostatic load were independently associated with worse physical health and increased

behavioral problems. This ALI – a combination of seven biomarkers – was also associated with attention problems, somatic complaints, and thought problems but only for those youth who experienced maltreatment. In terms of depressive symptoms, neither childhood maltreatment nor the ALI were independently significant. However, secondary analyses revealed several aspects of ALI (i.e., waist-height ratio, cortisol, and DHEA) predicted depression, but only for maltreated children.

When taken together, the limited research suggests children and adolescents, when exposed to sustained psychosocial stress, may evidence physiological derangement consistent with allostatic overload. Various indices of allostatic load have been associated with stress, depression, and worsening physical health. Furthermore, the work by [Evans et al \(117\)](#) and [Rogosch et al \(320\)](#) suggests that dysfunctional family dynamics may interact with existing psychosocial vulnerabilities resulting in greater allostatic load in youth. When applying this phenomenon to military youth, the research suggests military relocation stress and dysfunctional family dynamics may contribute to increased allostatic load – particularly for those endorsing comorbid health conditions.

Calculating the Allostatic Load Algorithm

The physiological stress response is generally considered more amenable to objective measurement than psychological constructs ([78](#)). Assessing the length or frequency of systemic processes within the body has the advantage of being independent from self-report and, therefore, less prone to respondent bias. However, physiological measures are still problematic for assessing chronic stress since they differ significantly in each individual, can quickly fluctuate, and more accurately assess acute stress load ([39](#)). To address these limitations, [McEwen and Seeman \(241\)](#) proposed an ALI which compiles numerous different physiological

measures representing complementary pathophysiological pathways designed to mitigate measurement error and better predict pathology. The ALI has been widely studied over the past 20 years and its design has been refined since its first formulation in the 1990s ([157](#); [241](#)). Refinements to the ALI's algorithm have generally fallen into three categories: 1) adjustment to the specific biomarkers used; 2) how the cumulative index is calculated; 3) differentiating ALI from other clinical constructs, namely MetS.

Candidate Biomarkers Used in the Allostatic Load Index

Despite calls for standardization, there remains significant heterogeneity in the candidate biomarkers used to operationalize ALI across studies. The contemporary “allostatic load battery” is generally constructed from a broad array of candidate biomarkers assessed primarily by their functional significance in a specific biological pathway implicated in stress. Indeed, one review found over 50 biomarkers have been used to formulate an ALI across the cardiovascular/respiratory, anthropometric, neuroendocrine, metabolic, and immune systems ([190](#)). The most common biomarkers included in the ALI are systolic and diastolic blood pressure, waist-hip ratio, BMI, cholesterol, triglycerides, and blood sugar levels (i.e., HbA1c). These commonly used biomarkers are primarily secondary mediators drawn from cardiovascular, anthropometric and metabolic systems. The total number of biomarkers used to formulate an ALI can also vary substantially based on availability and study aims; from as few as 4 to as many as 20 ([190](#)).

There is evidence, however, that the specific biomarkers used to formulate the ALI are less important than ensuring the range of biomarkers are inclusive of multiple allostatic regulatory systems [Wiley et al \(437\)](#). Indeed, the ALI appears to be relatively resilient to biomarker variations provided a range of biological systems are incorporated ([55](#)). Although evidence in adults supports that certain clusters of biomarkers are superior in predicting specific

tertiary outcomes ([193](#)), statistical modeling procedures suggests tertiary outcomes are best explained by including a common allostatic load factor ([437](#)).

When compared to adults, there is relatively limited research incorporating the ALI in pediatric samples ([26](#); [61](#); [116](#); [117](#); [320](#)). In addition, the ALI is not consistently operationalized across pediatric studies. Indeed, several studies highlight the discrepancy in the total number of biomarkers used: [Evans et al \(117\)](#) and [Rogosch et al \(320\)](#) created an ALI from six biomarkers while [Bahreinian et al \(26\)](#) included eight biomarkers. In these same studies, there is also variation in the specific biomarkers used and the allostatic regulatory systems the biomarkers are derived from. For example, [Evans et al \(117\)](#) and [Bahreinian et al \(26\)](#) both included anthropometrics in their ALI composite but utilized BMI and waist-to-hip ratio, respectively. Despite the heterogeneity in these studies, the positive findings across various formulations of the ALI suggests the construct is relatively robust and is a flexible measure to implement to assess chronic stress in vulnerable youth populations.

Calculating the Allostatic Load Composite

Various statistical techniques and algorithmic formulations have been applied in creating an allostatic load composite score. [Juster et al \(190\)](#) identified over 12 different methodologies involving increasingly sophisticated procedures to formulate an ALI. The most common and least sophisticated technique is the group allostatic load index, which has also been used exclusively in the identified pediatric studies ([117](#); [190](#); [320](#); [351](#)). In this formulation, the sampling distribution of a designated biomarker is used to designate a pre-determined risk percentile (i.e., >25th percentile) as a cut-off. A biomarker is then dichotomized as either a “1” or “0” depending on the cut-off determined by the sample’s distribution of biomarker values. Summing all the dichotomized biomarkers which fall within the designated cut-offs provides a

cumulative risk score. Depending on the study's aims and the availability of population-level normative data, it is also possible to construct the high-risk cut-offs from biomarker parameters. Indeed, the use of normative data improves generalizability in that it mitigates the likelihood of the ALI inadvertently incorporating the sample's inclusion characteristics.

Despite widespread use, the group allostatic load index has several clear drawbacks. The summation of dichotomized biomarkers values necessarily dilutes individual/group (i.e., gender, SES) differences. Individual differences, namely sex, have been implicated in sex-specific allostatic load profiles for illness ([352](#)). For example, [Gruenewald et al \(156\)](#) designed a study to identify sex differences in high-risk allostatic load profiles. The study authors noted biological pathways contributing to mortality varied significantly as a consequence of sex. More specifically, several neuroendocrine and inflammatory markers were predominant in male allostatic load profiles but completely absent for females ([156](#)).

The equal weighting of all biomarkers has also been questioned. [Buckwalter et al \(54\)](#) compared the predictive power for a single, equally weighted composite ALI versus a multisystem construct which weighted each biological system differently. Unsurprisingly, the multisystem construct explained more of the variance in several physical/psychological illness and provided insight into the specific biological pathways. However, the differentially weighted composite was only marginally superior in predicting the majority of health outcomes, thereby suggesting increasingly sophisticated techniques yield only modest gains ([54](#); [190](#)).

Lastly, the selection of pre-determined risk percentiles has also been critiqued. There is a consistent trade-off between specificity and sensitivity in identifying individuals at risk for allostatic overload. No definitive cut-off has been identified for an ideal ALI in studies of either adult or youth samples. In youth, some formulations of the ALI have incorporated less restrictive

(i.e., <50th percentile) cut-offs ([117](#)), used normative or clinical guidelines to calculate the cumulative ALI ([61](#); [320](#)), or kept the traditional quartile-based (i.e., 25th/75th percentile) formulation. The presence of significant findings despite variation in the pre-determined risk percentiles suggests ALI is a reliable construct to assess chronic stress. Indeed, evidence from adult studies suggest adjusting the pre-determined cut-offs has a only modest bearing on the ALIs association with a variety of health outcomes ([190](#)).

Various statistical and algorithmic formulations have enhanced the predictive utility of the ALI and explored casual pathways implicated in adverse health outcomes. Despite these advances, the less sophisticated ALI formulations (i.e., group allostatic load index) are robustly associated with a range of adverse physical and psychological health outcomes. The ease and availability of constructing an ALI using normative data is valuable for exploratory research in under-examined populations. The limited research in vulnerable military youth provides a valuable opportunity to examine a group-based ALI in the context of high rates of relocation.

Distinguishing Allostatic Load from MetS

Allostatic load is conceptually coherent with MetS and a superior construct to understand the deleterious influence of chronic stress on bodily systems. MetS has been consistently associated with chronic stress and adverse health outcomes ([11](#)). This association is troubling in light of the conflicting evidence on the relationship between chronic stress and MetS' constituent components ([39](#)). We still understand very little about the complicated reciprocal associations between different aspects of bodily systems, metabolism, and the stress response. As a result, there are likely several different pathways by which MetS emerges. For example, dyslipidemia can be both a consequence and a cause of dysregulated glucose metabolism ([283](#)). Allostasis

provides a framework to understand these bidirectional, non-linear effects in the context of multi-systemic regulation and offers several practical advantages ([142](#)).

Agreement rates vary dramatically for MetS' diagnostic criteria and the normative values used to identify MetS criteria differ across age, sex, and culture ([380](#); [406](#); [445](#)). There are numerous different criteria for diagnosing MetS in adults, and no definitive standard for diagnosing MetS in childhood/adolescence ([11](#)). Indeed, a review by [Ford and Li \(131\)](#) identified 40 unique definitions of MetS in children and adolescents and that the factor structure thought to underlie MetS varies substantially across studies. This inconsistency in the diagnostic definition of MetS is somewhat allayed by its clinical function as a powerful tool to guide surveillance/intervention ([413](#)). Most research related to allostatic load eschews a distinct diagnostic construct, and instead provides a framework to identify how psychosocial stressors become embodied into physical/psychological illness. In addition, this framework also allows for modeling of human systems under increasing levels of allostatic load. Given that ecological systems under stress/adversity can behave very differently than un-stressed systems ([230](#)), a cumulative ALI composite will give greater insight of dose-dependent relationships for a system under higher levels of allostatic load.

The original purpose of MetS was to identify individuals at risk for diabetes and cardiovascular illness. There is significant evidence that various formulations of the ALI are superior at predicting an array of adverse health outcomes - including cardiovascular illness – than MetS alone ([190](#); [236](#)). Indeed, when MetS and AL are modeled in-tandem, a greater proportion of the variance in tertiary outcomes (i.e., health) is explained by AL ([140](#); [216](#)). The allostatic load construct's superior predictive power is thought to be the consequence of integrating biomarkers from a greater range of biological systems. Several factor analytic studies

support the allostatic model with the AL construct subsuming MetS' constituent factors ([238](#); [437](#)).

In summary, allostatic load provides several benefits over MetS. Formulating an ALI for vulnerable military youth is both preceded and - despite heterogeneity in the ALI construct across studies - suggests a robust paradigm to understand how psychosocial stressors progress into illness. In addition, youth at high risk for binge eating and obesity experience higher levels of allostatic load ([61](#)), and therefore may be acutely sensitive to military lifestyle stressors. Overall, the ALI may prove a valuable tool to assess chronic stress in youth in accordance with the military relocation stress model.

The Current Study

Military lifestyle stressors – including PCS moves – have come under increased scrutiny as U.S. military family readiness has become essential for most, if not all, military communities. Past research has examined deployment as the most characteristic and impactful military lifestyle stressor ([172](#)). However, military relocation stress is consistently endorsed as a top stressor by military servicemembers, spouses, and their families. Although relocation is considered disruptive and stressful, the relationship between relocation and physical/psychological health is less clear. The existing research suggests outcomes related to PCS moves may interact with: the stress, mental health, and coping of the parent; community/familial resources; inherent vulnerability and risk history; and the frequency and recency of relocation events (see Figure 1).

The unique characteristics of the current sample to include youth with overweight/obesity, trait anxiety, and/or LOC eating presents a unique opportunity to understand how inherent vulnerability interacts with PCS moves. Pediatric obesity/overweight and disordered eating/attitudes are robustly inter-related and may be mutually reinforcing. Further, both pediatric weight status and disordered eating are associated with: 1) poor physical health as reflected by dysregulated lipid profiles, glucose metabolism, and indicators of inflammation; 2) poor psychological well-being via indicators of depression, anxiety, and negative affect. Indeed, the fact these co-morbid physical and health conditions exhibit similar biological and social determinants suggest they could be partially attributed to overlapping biological systems consistent with the human stress response.

Allostasis and allostatic load provide a valuable method to understand how psychosocial stressors manifest into illnesses; especially for those organisms already under significant allostatic load. Frequent or severe external stressors, to include relocation, may lead to

dysregulation of the stress response and to physical/psychological illness. Military-connected youth at-risk for developing binge-eating disorder and obesity are disposed towards poor physical/psychological health which may, in turn, further enhance their susceptibility to the deleterious effects of PCS moves. Various formulations of allostatic load have evinced associations with poor health and well-being in both adult and adolescent samples, and may provide a valuable tool to assess military relocation stress.

Given the paucity of research related to military relocation outcomes, this study examined the effects of military relocation stress on military adolescents' physiological and psychological health while considering candidate moderator variables. More specifically, this study aimed to: 1) determine if PCS moves impact adolescent functioning in a high-risk sample; 2) determine if family resilience and/or parental stress moderates the association between military relocation and adolescent functioning. To accomplish these aims, the proposed study incorporated anthropometric, cardiometabolic factors, and psychological variables to examine central and peripheral allostatic regulatory processes thought to contribute to adolescent adjustment. Figure 5 displays a summary of the hypothesized relationships between the variables of interest.

Aims and Hypotheses

Specific Aim 1: To examine the relationships among *PCS moves*, parenting stress, family resilience, and adolescent physiological health (see Figure 5).

Hypothesis 1a: There will be a significant positive association between *PCS moves* and scores on a summary biological index of allostatic load (ALI), after adjusting for age in years, sex, race, loss-of-control eating status, anxiety symptoms, and parental military rank.

Hypothesis 1b: Parental-report on the Family Resilience Assessment Scale (FRAS) will moderate the association between *PCS moves* and ALI, such that as the FRAS increases, the association between *PCS moves* and the ALI will decrease, after adjusting for covariates.

Hypothesis 1c: The Parenting Stress Index (PSI) will moderate the association between *PCS moves* and ALI such that as PSI increases, the association between *PCS moves* and ALI will increase, after adjusting for covariates.

Specific Aim 2: To examine the relationships among *PCS moves*, parental stress, family resilience, and adolescent psychological health (see Figure 5).

Hypothesis 2a: There will be a significant positive association between *PCS moves*, the Beck Depression Inventory (BDI-II), and scores on the Perceived Stress Scale (PSS) after adjusting for age in years, sex, race/ethnicity, anxiety symptoms, parental military rank, and BMI-z.

Hypothesis 2b: Parental-report on the Family Resilience Assessment Scale (FRAS) will moderate the association between *PCS moves* and psychological health (i.e., BDI-II & PSS) such that as the FRAS increases, the association between *PCS moves* and psychological health will decrease, after adjusting for covariates.

Hypothesis 2c: Parental-report on the Parenting Stress Index (PSI) will moderate the association between *PCS moves* and psychological health (i.e., BDI-II & PSS) such that as PSI increases, the association between *PCS moves* and psychological health will increase, after adjusting for covariates.

Exploratory Aim 3: To determine the utility of the ALI in detecting stress-based physiological dysregulation in a sample of high-risk military-connected adolescents.

Hypothesis 3a: The present study's formulation of the ALI will prove superior in detecting stress-based physiological dysregulation in a sample of high-risk military-connected adolescents than either MetS or analyses of the constituent biomarkers.

CHAPTER 2: METHOD

Research Design

The study is a secondary analysis drawn from the Preventing Obesity in Military Communities – Adolescent study (ClinicalTrials.gov ID: NCT02671292), an ongoing prevention trial. The current study used a cross-sectional design using data collected prior to intervention to examine the association between relocation, chronic stress, and physical/psychological health. Only the pertinent procedure and methodology from the parent study will be described.

Participants and Recruitment

Participants were male and female adolescents (12-17 years old) and TRICARE beneficiaries who completed assessments prior to participating in a binge-eating disorder and adult obesity prevention program designed to test the efficacy of interpersonal psychotherapy. Qualified adolescents were identified through the Defense Enrollment Eligibility Reporting System (DEERS) and recruited through direct mailings, approved advertisements, referrals from military health care providers, and in-person recruitment at Ft. Belvoir Community Hospital (FBCH) and the Walter Reed National Military Medical Center (WRNMMC). The study was approved by both the FBCH Research Office and the USUHS Institutional Review Board (IRB).

Youth were eligible if they were 12 to 17 years old at the start of the study, had a BMI at or above the 85th percentile for age and sex, spoke English proficiently, had acceptable ability to complete study procedures, endorsed high anxiety symptoms (State-Trait Anxiety Inventory – Children (STAI-C) ≥ 32) and/or loss-of-control (LOC) eating episode during the three months prior to assessment, and have a parent enrolled in TRICARE at the time of study initiation ([374](#)). In addition, the parent/guardian was required to understand English for the child to meet eligibility criteria.

Adolescents were excluded from the study if there was the presence of: chronic major medical illness (e.g., hypothyroidism, hematological); an obesity-related medical complication requiring aggressive treatment (e.g., type 2 diabetes); current or recent pregnancy; regular use of prescription medication known to affect appetite, mood, or body weight unless their weight has been stable over the last three months; concurrent involvement in a weight loss program or psychotherapy; weight loss exceeding 3% of body weight within the past two months; and the presence of a significant psychiatric condition (excluding binge-eating disorder).

Procedures

Preliminary eligibility of potential youth participants was determined through telephonic screening conducted by trained research assistants. If the youth was deemed potentially eligible (i.e., BMI %ile ≥ 85 , the presence of elevated anxiety symptoms, and/or LOC eating episodes), a baseline assessment was scheduled to determine final eligibility. The proposed study will only examine the data collected at baseline before any intervention was conducted.

Baseline Assessment

The baseline assessment occurred at one of two sites in the DC/VA/MD area: Uniformed Services University, Bethesda, MD or Fort Belvoir Community Hospital, Ft. Belvoir, VA. During the baseline visit, written consent and assent were given by the parents/guardians and adolescents, respectively. Youth participants were required to conduct an overnight fast prior to their arrival and the visit lasted approximately five hours. Immediately upon completion of the baseline assessment, the research team, the adolescent, and parent/guardian determined eligibility for the study. Procedures at baseline assessment included: height and weight, the consent and assent process, body composition, metabolic markers, completion of two semi-structured interviews, and a questionnaire completion (either online or in-person). The online

survey platform (<https://www.surveymonkey.com>) is a secure web-based platform for data input and retrieval. The baseline visits also included a series of questionnaires for the accompanying parent/guardian. These questionnaires were either completed during the baseline assessment or returned to the study team at a later date. Parents/guardians and participants were expected to complete all risk assessment measures in-person during the interview.

If in-person interviews were thought to impact the safety of the research staff or participants (e.g., a global pandemic, crisis, or other extreme scenario) the decision was made to hold components (e.g., clinical interview) of the baseline visits electronically. Blood serum values and anthropometric data were collected when circumstances permitted.

Measures

Relocations and Demographics

Demographic Questionnaire (DEMQ)

The DEMQ is a measure developed by the investigators to assess a number of standard (e.g., race, family composition) and military-related family factors (e.g., number of deployments/relocations). Each parent/guardian completes the DEMQ during or immediately following the baseline. The study examined total *PCS moves* (reported by parent) as the primary measure of relocation. Total *PCS moves* will be used in this study in lieu of *Mobility Rate*, which is defined as the sum of all reported lifetime PCS moves divided by the adolescent's age. Although this methodology has been used across several studies and is thought to provide a precise estimate of mobility ([128](#); [288](#)), the analyses will adjust for age rather than compute a transformed (*PCS moves x Age*) independent variable. The DEMQ also provides several other variables in the study. Parental rank (enlisted vs. officer), race (non-Hispanic white vs. other) and age in years are also assessed as covariates.

Physiological measures

Height, Weight, and Body Mass Index (BMI)

A stadiometer and calibrated scale were used to measure height (without shoes) and fasting weight, respectively. A BMI z-score is calculated from the CDC growth charts, after adjusting for age and sex ([206](#)).

Cardiometabolic Risk Factors

A standard panel for youth with overweight was measured through blood samples drawn by a trained phlebotomist or registered nurse at FBCH or WRNMMC. The panel of cardiometabolic risk factors included: triglycerides, cholesterol levels, glucose, insulin, hemoglobin A1c (HbA1c). Systolic (SBP) and diastolic blood pressures (DBP) were measured once after 5 minutes rest at the right brachial artery via a blood pressure monitor. Waist circumference (WC) measurements were taken in triplicate, with a tension tape measure at the iliac crest. Fasting triglycerides, glucose and cholesterol were measured from blood samples using a Cobas 6000 c 501 or 701/ 702 analyzer using reagents from Roche Diagnostics (Indianapolis, IN). LDL-c was calculated using the following formula: $\text{Total Cholesterol} - \text{HDL} - (\text{TGL}/5)$.

Stress Assessment Measures

Perceived Stress Scale (PSS)

The PSS is a self-report questionnaire measuring the impact of life events on an individual's level of perceived psychological stress. The 14-item questionnaire asks participants to evaluate the degree to which they appraise situations as stressful within the past month (e.g., "In the last month, how often have you felt that things were going your way?"). The PSS has

adequate psychometric properties and has been validated in community and laboratory settings (77). Psychological stress was defined by using the PSS total score.

Allostatic Load Index (ALI)

The ALI was computed from 8 extant biological parameters associated with obesity-related weight gain from the parent study: low density lipoprotein (LDL) cholesterol, high density lipoprotein (HDL) cholesterol, triglycerides, systolic and diastolic blood pressure, HbA1c, BMI, and waist circumference (267). These specific biomarkers have been used extensively with adult and adolescents to assess cumulative allostatic load in an organism by accounting for compound physiological pathways simultaneously (61; 190; 351). Age, sex, ethnicity, and/or height cut-offs were used to denote a value of one (1) for each dysregulated biomarker. There is no standardized protocol for establishing cut-offs, but the top quartile (i.e., 25%) is generally used (437). The parent study examines an at-risk population experiencing pediatric overweight or obesity. The proposed study utilized more rigorous adolescent cutoffs (e.g., 85th to 95th percentile) to ensure an adequate distribution. The limited data comparing optimal scoring for the ALI indicate more sophisticated methodologies have only modest bearings on predictive utility (140).

Waist circumference cutoff was calculated using the 90th percentile while controlling for age, sex, and ethnicity (127). Total cholesterol, LDL cholesterol, and triglycerides' cutoff was computed using the 95th percentile while controlling for age and sex (171). HDL cholesterol used the cutoff of the 5th percentile while controlling for age and sex (171). HbA1c's cutoff was in the 95th percentile for sex (328). Systolic and diastolic blood pressure's cutoff are based on the 95th percentile for age, sex, and height (317). See Table 1 for a list of the biomarkers to be used in the formulation of the ALI.

Adjusted Allostatic Load Index (ALI)

To validate the utility of the present study's ALI formulation, an alternative ALI formulation will be calculated. The adjusted ALI uses the sample generated cut-offs from the 75th percentile (i.e., BMIz, SBP, DBP, LDL, TRI, WC, HbA1c) or the 25th percentile (i.e., HDL) of all participants' biomarkers. Otherwise, the adjusted ALI was calculated as described previously.

Metabolic Syndrome (MetS)

There is no uniform clinical definition of MetS for youth ([310](#)). Accordingly, this study elected to closely follow the definition provided by [Weiss et al \(434\)](#) which diagnoses MetS if at least three of the following criteria are met: BMIz ≥ 2.0 , SBP and/or DBP $\geq 95^{\text{th}}$ percentile for age/sex/height, triglycerides $\geq 95^{\text{th}}$ percentile for age/sex, HDL $\leq 5^{\text{th}}$ percentile for age/sex, and fasting glucose ≥ 100 mg.

State-Trait Anxiety Inventory for Children-A trait scale (STAI-C)

The STAI-C is a self-report questionnaire consisting of 20-items administered to children to assess trait anxiety symptoms ([374](#)). The frequency of feelings (e.g., I worry too much) are rated on a scale of 1 (hardly-ever) to 3 (often). The STAI-C has good internal consistency (mean alpha coefficient = .89) for the trait scale ([31](#)). This measure was used as inclusion criteria for the parent study and will be adjusted as a covariate.

Adolescent Psychological Questionnaires

Negative Affect

The Beck Depression Inventory- 2nd Edition (BDI-II)

The BDI-II is a widely used measure used to assess the intensity of depressive symptoms in the last two weeks ([32](#)). The BDI-II consists of 21 items which load onto a single scale. The BDI-II has demonstrated validity and reliability amongst clinical and community samples ([33](#)).

The Life Events and Coping Inventory (LECI)

The LECI is a self-report questionnaire consisting of over 125 stressful life events and 49 common coping strategies ([96](#)). Items are rated on a 9-point Likert scale with the following anchors: 1 = “No stress at all;” 3 = “A little stress;” 5 = “Pretty much stress;” 7 = “A lot of stress;” 9 = “An extreme amount of stress.” Item generation, refinement, and validation utilized a normative sample of 681 majority white (94%) middle-class adolescents. The LECI’s indices of life stress demonstrated adequate test-retest reliability over an 11-week interval with moderate to high correlations with standardized measures of anxiety/depression in the original normative sample.

Adult Psychological Questionnaires

Parenting Stress Index – Short Form (PSI-SF)

The PSI consists of 36 items across three scales: dysfunctional parent-child interaction, difficult child characteristics, and parental distress ([1](#)). The proposed study will use PSI total stress, defined as the stress a parent is experiencing specific to their role. The PSI has adequate psychometric properties for the population of interest ([311](#)).

Family Resilience Assessment Scale (FRAS)

The FRAS is a 54-item measure that assesses family resilience through six subscales: communication and problem-solving, utilizing social and economic resources, maintaining a positive outlook, family connectedness, family spirituality, and ability to make meaning of adversity ([368](#); [425](#)). The FRAS has adequate psychometric properties across a range of samples

(53; 69). The proposed study will use the FRAS total composite score consisting of all six subscales.

Data Analytic Approach

Initial Analyses were conducted on the data collected from the baseline assessment using SPSS v.28 (IBM Corp., 2020). Across all variables, 15 (< 5%) were identified as extreme outliers (i.e., > 3 SD from the mean) and were adjusted to fall within three standard deviations from the mean (391). To examine for normality, Q-Q plots were conducted for all study variables and were observed to have acceptable fit for normally distributed data. Independent samples *t*-tests will be conducted to investigate differences between adolescents with no relocations and those with one or more relocations on demographic variables, covariates, and study variables. Unless reported otherwise, all analyses adjusted for age in years (continuous), anxiety symptoms (continuous), parental military rank (enlisted vs. officer), race (coded as non-Hispanic White vs. other), loss-of-control eating status (presence vs. absence), and BMI-z (continuous). Statistical significance was set at $\alpha \leq .05$ for all analyses. Give the presence of age/sex as covariates, the analyses were also run using the baseline BMI score. There were no differences in findings between using the BMIz score versus the unadjusted baseline BMI as a covariate. When feasible, continuous variables were used in order address the study's modest shortfall in statistical power. No efforts were made to adjust for multiplicity in the present study which may increase the risk of type I error. In cases where both parents are servicemembers, parental rank was determined to be the highest rank in the family household. For example, if a mother is enlisted while the father is an officer the resultant classification would be coded under the officer category.

For the primary analyses, a series of three multiple linear regression models were conducted using the multiply imputed data to examine the relationship between *PCS moves* with 1) PSS total score, 2) BDI-II total score, 3) the allostatic load index, after adjusting for covariates. Visual inspection of scatterplots and tests for linearity will be conducted to ensure no deviance from the expected linearity. To examine if *PCS moves* interacted with the parenting stress or family resilience, the linear regression models were repeated with the addition of two separate interaction terms. These interaction terms were completed even in the absence of a significant main effect. As discussed, high levels of family resilience or high levels of family stress may attenuate or exacerbate, respectively, the impact of PCS moves and adolescent poor health/well-being (56). Therefore, an interaction term was computed for analysis by calculating the product of mean-centered *PCS moves* and the mean-centered interaction terms (i.e., PSI, FRAS).

Specific Aim 1

To examine the relationships among *PCS moves*, parental stress, family resilience, and adolescent physiological health.

Hypothesis 1a: To determine the presence of a significant positive association between *PCS moves* and a summary biological index of allostatic load (ALI), a linear regression model was created with *PCS moves* as a predictor variable and ALI as the dependent variable, after adjusting for covariates.

Hypothesis 1b: A simple moderation analysis was performed to investigate the hypothesis that the Parenting Stress Index (PSI) would moderate the association between *PCS moves* and ALI such that as the PSI increases, the association between *PCS moves* and ALI would increase. The outcome variable was ALI. The predictors variables were *PCS moves* and

PSI. An interaction was computed from the mean-centered predictors between *PCS moves* and PSI. Examination of the interaction plot was used to determine the nature of the proposed moderation. All modeled effects adjusted for covariates (excluding BMI-z).

Hypothesis 1c: A simple moderation analysis was performed to investigate the hypothesis that parental-report on the Family Resilience Assessment Scale (FRAS) would moderate the association between *PCS moves* and ALI, such that as the FRAS increases, the association between *PCS moves* and the ALI would decrease. The outcome variable was ALI. The predictors variables were *PCS moves* and FRAS. The moderation analyses was performed as described previously. All modeled effects adjusted for the covariates (excluding BMI-z).

Specific Aim 2

To examine the relationships among *PCS moves*, parental stress, family resilience, and adolescent psychological adjustment (i.e., Perceived Stress Scale (PSS); Beck Depression Inventory (BDI-II)).

Hypothesis 2a: To determine the presence of a significant positive association between *PCS moves* with the PSS and BDI-II, a series of linear regression models were created with *PCS moves* as a predictor variable and the PSS total score and BDI-II total score as the dependent variables.

Hypothesis 2b: A series of simple moderation analyses were performed to investigate the hypothesis that parental stress (PSI) moderates the association between *PCS moves* with the PSS and BDI-II such that as the PSI increases, the association between *PCS moves* and PSS and BDI-II would increase. The outcome variables were the PSS total score and BDI-II total score. The predictor variables will be *PCS moves* and PSI. The moderation analyses was conducted as described previously. All modeled effects adjusted for the covariates.

Hypothesis 2c: A series of simple moderation analyses were performed to investigate the hypothesis that parental-report on the Family Resilience Assessment Scale (FRAS) moderates the association between *PCS moves* with the PSS and BDI-II such that as the FRAS increases, the association between *PCS moves* and PSS and BDI-II would decrease. The outcome variables were the PSS total score and BDI-II total score. The predictor variables were *PCS moves* and FRAS. The moderation analyses was conducted as described previously. All modeled effects adjusted for the covariates.

Exploratory Aim 3:

To determine the utility of the ALI in detecting stress-based physiological dysregulation in a sample of high-risk military-connected adolescents.

Hypothesis 3a: The results from Specific Aim 1 were compared to three exploratory analyses: the adjusted ALI, MetS, and each of the constituent biomarkers. For the adjusted ALI, a multiple linear regression was calculated to predict the adjusted ALI based on participant's total PCS moves after adjusting for age in years, anxiety symptoms, parental military rank, race, and loss-of-control eating status. For MetS, A logistic regression was calculated to predict the presence of MetS (binary) based on each participant's total PCS moves after adjusting for age in years, anxiety symptoms, parental military rank, race, and loss-of-control eating status. For the constituent biomarkers, a series of multiple linear regressions were calculated to predict each individual biomarker in the ALI based on a participant's total PCS moves after adjusting for age in years, anxiety symptoms, parental military rank, race, and loss-of-control eating status.

Power

Power analyses for all the aims were conducted using G*Power 31 ([122](#)). The relocation outcome literature precludes meta-analysis due to heterogeneity in study methodology and

quality. As a result, the estimated effect size for any effects was expected to be small-to-medium (Cohen's $f^2 = .09$). A series of power analyses were conducted using an alpha level of .05 at a power of 80%. The sample size required given these conditions can be viewed in Table 2 with both the baseline data and multiply imputed data. The total available cases for the independent variable (*Total PCS moves*) for the specific aims was fixed at 164. Using a post-hoc power analysis, the shortfall in sample size only equates to a modest reduction power. Regardless, effect sizes were included for all analyses to supplement this shortfall in statistical power. Table 2 details the achieved power (%) across analyses.

Missing Values Analyses

Missing values analyses suggest the current sample would benefit from imputation procedures. The primary cause for missing data was item non-response across the study's measures. Total missingness was relatively low with less than 5% missing data across all values. However, 74% of variables are incomplete with only 54% of participants having complete data for analysis. An analyses of item-level missing data indicated missing data in excess of 5% in several variables: the FRAS (13%), ALI (13%), PSS (13%), BDI (9%), WC (8%), Parental Rank (5.5%), and HbA1c (5.5%). Consequently, a complete case analysis via listwise deletion could potentially discard nearly half of participant data and result in incongruent samples across the study's aims. See Table 4 for a summary of missing data across study variables in the observed data.

To meet required statistical power and in accordance to published/unpublished missing data procedures, person-mean imputation was performed on the FRAS, PSS, and BDI for participants missing 10% or less of their item-level responses. Person-mean imputation is a widely utilized statistical procedure used to address missing data. This procedure substitutes

missing values in a scale for the participants' statistical mean ([102](#)). Mean imputation is convenient but is also known to introduce statistical bias by ignoring sampling variability. In the current study, person-mean imputation was considered to introduce excessive bias into the analyses despite a modest increase of statistical power ([35](#)). Table 4 summarizes the results of the mean imputation procedure.

In lieu of listwise deletion and mean imputation, the observed dataset underwent Multiple Imputation (MI). MI is an improvement over single imputation procedures by incorporating: 1) non-responders' sampling variability through independent random draws from a predictive model (i.e., the imputation model); and 2) uncertainty regarding non-response by pooling multiple permutations of the imputation model ([325](#)). MI has been demonstrated to produce valid statistical inferences for missing values when total missingness is less than 40% ([184](#)).

In accordance with Rubin's nomenclature ([324](#)) and established imputation procedures ([184](#)), MI is considered reliable under the assumption of Missing Completely at Random (MCAR) or Missing at Random (MAR). Pattern analysis of missing data was not observed to have any significant trends to indicate a consistent pattern of missing data across participants. In addition, Little's MCAR test was not significant ($\chi^2 = 246.412$; $P = .392$) thereby failing to reject the null hypothesis that the data is MCAR. Lastly, independent samples t-tests and chi-square analyses found no significant differences ($\alpha = .05$) between completed cases and cases with missing data for the FRAS, ALI, BDI, PSS, WC, and Parental Rank across all the study's variables.

In the event the missing data is Missing Not at Random (MNAR), one would expect item non-response to be directly related to the reason the data is missing. There is no compelling evidence to suggest item missingness is related to the pattern of observed data. It is possible

measures of parenting stress/resilience may induce defensiveness and/or item non-response; however, there is no evidence of an association between missingness in the PSI and FRAS. Significant negative correlations (i.e., $r > -.50$) between the FRAS and PSI denote divergent validity in the constructs of family resilience and parenting stress, respectively. If item non-response was related to defensiveness/family functioning it would be expected the missingness between these measures would be correlated.

In summary, there is no evidence to suggest the observed missingness in the data violates MCAR or MAR given: 1) the limited ($< 5\%$) missingness across all variables, 2) no evidence of any correlates of missingness amongst the study's variables of interest, and 3) an insignificant Little's MCAR test. However, there is still the possibility the data is Missing Not at Random (MNAR) and some observed and/or unobserved latent variable could be responsible for the missingness. As a precaution the results of the observed and MI analyses are reported. In the event of significant discrepancies across tested models, a sensitivity analysis will be performed to assess the robustness of the imputation model ([136](#)).

Multiple Imputation Model

The Imputation Model was constructed using SPSS V.28 (IBM, 2020). SPSS provides a set of tools to assist in missing data analysis and MI. Initial missing values analysis confirmed the utility and feasibility of multiple imputation for the current dataset. 19 variables (see Table 4) were included into the final imputation model under the assumption of MAR. Two interaction terms (PCS Total * FRAS/PSI) were computed using the imputed data and subsequently included into the final analysis ([404](#)). No auxiliary variables were identified to be associated with missingness in the data and therefore none were considered for inclusion into the final imputation model. All variables were constrained using the observed data's minimum and

maximum after adjusting all values to fall within 3 standard deviations of the mean. To assist in reproducibility of the imputation model a Mersenne Twister was set to the fixed value of “200000” as the method of random number generation ([426](#)).

SPSS was set to automatically select the imputation method based on the pattern of missingness in the observed dataset. The pattern of missing data was determined to be arbitrary by both the software and during the author’s missing value analysis, therefore an iterative Markov chain Monte Carlo (MCMC) was used. A fully conditional specification (FCS) method used all available variables in the model as predictors, then imputes missing values for the variable being fit. The method continues until the assigned number of permutations, and the imputed values are saved to a separate imputed dataset ([184](#)). A total of five imputed datasets were produced and all analyses were pooled in accordance with Rubin’s rules ([325](#)). A total of 157 values were imputed into the model across 14 variables. Visual inspection of the Q-Q plots and of the tabulated data (see Table 4) indicate the imputed values compare reasonably well to the observed values. As a result, the imputation model’s results are considered valid and presented in the ensuing analysis. It should be noted SPSS does not produce regression model statistics for the pooled MI results. However, given the study’s aims and focus on individual predictors, the pooled model statistics were considered extraneous.

CHAPTER 3: RESULTS

Participant Demographics

A total of 164 military-dependent adolescents were included in the final analysis. The sample's demographics were majority female (58.5%) with a participant mean age of 14.5 years ($SD = 1.6$). Participants' racial/ethnic identification were: 57.9% Caucasian, 22.0% Black/African American, 22.0% Hispanic/Latino, 12.2% Multiple Races, 3.0% Asian, and 2.4% unknown. Regarding inclusion characteristics: approximately 5.0% of the sample endorsed LOC eating within the past month only, 45.5% scored high in trait anxiety only (at or above 32 on the STAI-C), and 42.1% endorsed both LOC eating in the last month and scored high in trait anxiety. The sample's mean BMI- z was 1.9 ($SD = 0.4$).

The sample's mean of cumulative PCS moves at baseline was 3.5 ($SD = 3.0$), ranging from no PCS moves to 14 PCS moves. The median number of PCS moves for the current sample was 3.0. Approximately one quarter of the sample (22.5%) reported no relocations on the DEMQ while 77.5% endorsed at least one relocation. There was a distinct positive skew in the distribution of PCS moves, with the single most common number of relocations (i.e., mode) being 0. The percentile distributions also demonstrate the positive skew: individuals with 5 relocations were in the 75th percentile while those with 8 relocations were in the 95th percentile. There were no significant differences between participants with no relocation against those with at least one relocation on any of the pertinent demographic variables. Table 3 shows demographics for the full sample, as well as information by relocation status.

The sample's mean on the ALI at baseline was 2.3 ($SD = 1.3$), with the cumulative index score of dysregulated biomarkers ranging from 0 to 6. The ALI appears normally distributed

after visual inspection of the sample's histogram and P-P Plot. Table 1 lists the sample percentage meeting the assigned normative cut-offs.

Approximately 16.4% met the criteria for MetS by meeting 3 or more of the following criteria: BMI ≥ 2.0 , SBP and/or DBP $\geq 95^{\text{th}}$ percentile for age/sex/height, triglycerides $\geq 95^{\text{th}}$ percentile for age/sex, HDL $\leq 5^{\text{th}}$ percentile for age/sex, and fasting glucose ≥ 100 mg. The sample percentage meeting the cut-off criteria for SBP, DBP, Triglycerides, and HDL can be found in Table 1. 39.6% and 5% met the aforementioned cut-off criteria for BMI and fasting glucose, respectively.

Specific Aim 1

To examine the relationships among PCS moves, parental stress, family resilience, and adolescent physiological health.

Hypothesis 1a: There will be a significant positive association between PCS moves and a summary biological index of allostatic load (ALI).

Results of Hypothesis 1a: The regression model was not statistically significant [$F(7,123) = 1.47, p = .19$] with an adjusted $R^2 = .03$. Individual predictors were examined further and none of the variables were significantly associated with allostatic load ($ps > .05$). For the pooled MI model, only the presence of LOC eating in the past month was a significant predictor ($b = 0.43, SE[b] = .20, p = .03, 95\% \text{ CI } [0.03, 0.82]$) of increases in the ALI. See Table 5 for the complete summary of the regression analyses. Observed power is reported in Table 2.

Hypothesis 1b: The FRAS will moderate the association between PCS moves and ALI such that as the FRAS increases, the association between PCS moves and ALI will decrease.

Results of Hypothesis 1b: The regression model was not significant [$F(9, 105) = 1.31; p = .24$] with an adjusted $R^2 = .02$. Likewise, individual predictors were examined and none were significant ($ps > .05$). For the pooled MI model, being male was significantly associated with

increased ALI ($b = 0.43$, $SE[b] = .21$, $p = .04$, 95% CI [0.02, 0.84]; see Table 5). Observed power is reported in Table 2.

Hypothesis 1c: Parental-report on the PSI will moderate the association between PCS moves and ALI, such that as the PSI increases, the association between PCS moves and the ALI will decrease.

Results of Hypothesis 1c: The model was not statistically significant [$F(9, 117) = 1.46$; $p = .17$] with an adjusted $R^2 = .03$. Likewise, individual predictors in both the observed and pooled model were examined and none were found to be significant ($ps > .05$). See Table 5 and Table 2 for the complete summary of the regression analyses and observed power analyses, respectively.

Specific Aim 2

To examine the relations between PCS moves, parental stress, family resilience, and adolescent psychological adjustment (i.e., PSS/BDI-II).

Hypothesis 2a: There will be a significant positive association between PCS moves with the PSS and BDI-II.

Results from Hypothesis 2a: The regression model for the PSS was significant [$F(8, 118) = 5.01$; $p < .001$] with an adjusted $R^2 = .21$. Individual predictors were examined further and indicated only the baseline STAI-C score ($b = 0.51$; $SE[b] = 0.11$, $p < .001$, 95% CI [0.29, 0.73]) was significantly positively associated with PSS scores. For the Pooled MI model, the baseline STAI-C score retained significance ($b = 0.49$; $SE[b] = 0.10$, $p < .001$, 95% CI [0.30, 0.68]). See Table 6 for the summary of the complete regression analysis. Observed power is reported in Table 2.

The regression model for the BDI-II was significant [$F(8, 123) = 5.11$; $p < .001$] with an adjusted R^2 of .20. Being female ($b = -2.89$, $SE[b] = 1.27$, $p = .03$, 95% CI [-5.41, -0.38]) and the

baseline STAI-C score ($b = 0.56$, $SE[b] = 0.10$, $p < .001$, 95% CI [0.36, 0.76]) were both associated with higher BDI-II scores. Similarly, for the Pooled MI model, both being female ($b = -3.53$, $SE[b] = 1.21$, $p < .004$, 95% CI [-5.93, -1.14]) and the baseline STAI-C score ($b = 0.57$, $SE[b] = .01$, $p < .001$, 95% CI [0.39, 0.76]) retained significance. See Table 7 for the summary of the complete regression analysis. Observed power is reported in Table 2.

Hypothesis 2b: Family Resilience will moderate the association between PCS moves with the PSS and BDI-II such that as the FRAS increases, the association between PCS moves and PSS and BDI-II will increase.

Results from Hypothesis 2b: Multiple linear regressions were calculated to conduct a series of simple moderation analyses to determine if the FRAS moderates the association between PCS moves and psychological outcomes (i.e., PSS/BDI-II). For the PSS outcome variable, the regression model was significant [$F(10, 99) = 3.17$; $p < .001$] with an adjusted $R^2 = .19$. Individual predictors were examined and only the STAI-C ($b = 0.49$, $SE[b] = 0.12$, $p < .001$, 95% CI [0.25, 0.73]) was significantly associated with higher PSS scores. For the pooled MI model, the STAI-C retained significance ($b = 0.49$, $SE[b] = 0.10$, $p < .001$, 95% CI [0.30, 0.69]; see Table 6). However, PCS total moves were significantly negatively associated with lower PSS scores in both the observed ($b = -0.53$, $SE[b] = 0.27$, $p = .048$, 95% CI [-1.07, -0.01]) and pooled data ($b = -0.42$, $SE[b] = 0.21$, $p = .044$, 95% CI [-0.83, -0.01]). For the BDI-II outcome variable, the regression model was significant [$F(10, 103) = 4.34$; $p < .001$] with an adjusted R^2 of .23. Individual predictors were examined to indicate the baseline STAI-C score ($b = 0.60$, $SE[b] = 0.11$, $p < .001$, 95% CI [0.38, 0.81]) was associated with higher BDI-II scores. For the Pooled MI model, the baseline STAI-C score ($b = 0.57$, $SE[b] = 0.10$, $p < .001$, 95% CI [0.38, 0.77]) retained significance and directionality, while being female ($b = -3.49$, $SE[b] = 1.24$, $p = .01$, 95% CI [-

5.96,-1.02]) was associated with higher BDI-II scores. See Table 7 and Table 2 for the summary of the complete regression analysis and power analyses, respectively.

Hypothesis 2c: Parental-report on the Parenting Stress Index (PSI) will moderate the association between PCS moves and psychological health (i.e., BDI-II & PSS) such that as PSI increases, the association between PCS moves and psychological health will increase, after adjusting for covariates.

Results from Hypothesis 2c: A multiple linear regression was calculated to conduct a simple moderation analysis to determine if the PSI moderates the association between PCS moves and PSS. The regression model was significant [$F(10, 102) = 3.73; p < .001$] with an adjusted $R^2 = .18$. Individual predictors were examined further and only the STAI-C ($b = .49$, $SE[b] = 0.11$, $p < .001$, 95% CI [0.26, 0.71]) was significantly associated with higher PSS scores. For the pooled MI model, the STAI-C retained significance and directionality ($b = .49$, $SE[b] = 0.10$, $p < .001$, 95% CI [0.30, 0.69]; see Table 6). Observed power is reported in Table 2.

In addition, a multiple linear regression was calculated to conduct a simple moderation analysis to determine if the PSI moderates the association between PCS moves and BDI-II. The regression model was significant [$F(10, 118) = 3.89; p < .001$] with an adjusted $R^2 = .18$. Individual predictors were examined to indicate the baseline STAI-C score ($b = 0.55$, $SE[b] = 0.10$, $p < .001$, 95% CI [0.35, 0.76]) and being female ($b = -2.83$, $SE[b] = 1.29$, $p = .03$, 95% CI [-5.38, -0.28]) was associated with higher BDI-II scores. For the Pooled MI model, both being female ($b = -3.52$, $SE[b] = 1.22$, $p = .01$, 95% CI [-5.94, -1.10]) and the baseline STAI-C score ($b = 0.57$, $SE[b] = 0.10$, $p < .001$, 95% CI [0.39, 0.76]) retained significance and their respective directionality. See Table 7 and Table 2 for the summary of the complete regression analysis and power analyses, respectively.

Exploratory Aim 3:

To determine the utility of the ALI in detecting stress-based physiological dysregulation in a sample of high-risk military-connected adolescents.

Hypothesis 3a: The present study's formulation of the ALI will prove superior in detecting stress-based physiological dysregulation in a sample of high-risk military-connected adolescents than either MetS or analyses of the constituent biomarkers.

Results from Hypothesis 3a: For the adjusted ALI, Table 8 lists the summary of the linear regression analysis using the different scoring methodology. In the observed data, the regression model for the adjusted ALI was significant [$F(7, 139) = 2.13; p = .05$] with an adjusted $R^2 = .05$. However, examination of the individual predictors demonstrated only being male ($b = 0.90, SE[b] = 0.05, p = .004, 95\% \text{ CI } [0.30, 1.51]$) was associated with increased ALI. Being male ($b = 1.14, SE[b] = 0.29, p < .001, 95\% \text{ CI } [0.57, 1.72]$) retained significance and directionality in the Pooled MI model. For MetS, the results of the logistic regression indicated none of the predictor variables were significantly associated with MetS ($\alpha = .05$; see Table 9).

The results of the regressions for each constituent biomarker are summarized according to their respective biological system: cardiovascular (Table 10), metabolic (Table 11), and anthropometric (Table 12). The following results summarize only the imputed data. When examining the biomarkers independently, PCS moves were not significantly associated with any of the cardiometabolic/anthropometric biomarkers which constitute the ALI.

In terms of the cardiovascular markers, only age ($b = 0.18, SE[b] = 0.05, p < .001, 95\% \text{ CI } [0.08, 0.29]$) and maleness ($b = 5.29, SE[b] = 2.03, p = .01, 95\% \text{ CI } [1.30, 9.29]$) was significantly associated with higher SBP. For the metabolic variables, having an officer as a parent was associated with lower blood glucose ($b = -0.12, SE[b] = 0.05, p = .02, 95\% \text{ CI } [-0.22,$

-0.02]) and greater levels of HDL ($b = 7.55$, $SE[b] = 2.31$, $p = .001$, 95% CI [2.97,12.12]). Being male was associated with increased triglycerides ($b = 20.37$, $SE[b] = 7.96$, $p = .01$, 95% CI [4.77, 35.97]) and lower HDL ($b = -7.24$, $SE[b] = 2.04$, $p < .001$, 95% CI [-11.26, -3.22]) In terms of anthropometric markers, being male was associated with greater BMIz ($b = 0.19$, $SE[b] = 0.06$, $p = .002$, 95% CI [0.07, 0.31]) and greater waist circumference ($b = 6.30$, $SE[b] = 1.89$, $p < .001$, 95% CI [2.58, 10.01]). Older ages ($b = 0.14$, $SE[b] = 0.05$, $p = .01$, 95% CI [0.04, 0.24]) were associated with greater waist circumference. LOC eating in the past month ($b = 3.96$, $SE[b] = 1.86$, $p = .03$, 95% CI 0.31,7.60]) was associated with greater waist circumference.

CHAPTER 4: DISCUSSION

The primary purpose of this study was to examine if PCS moves – a common and frequency military lifestyle stressor – are disruptive to the physical and psychological health of an ethnically diverse sample of at-risk military adolescents exhibiting overweight, elevated trait anxiety, and/or LOC eating. This sample's comorbidities were expected to place these adolescents at higher risk of post-PCS maladjustment. Further, this study sought to determine if parental stress and family resilience moderates the proposed relationship between PCS moves and physical/psychological health. The identification of modifiable family factors may provide valuable points of intervention in alleviating the proposed sequelae of PCS moves. The following section highlights the results and interpretation by study aim.

Specific Aim 1

Specific aim 1 examined if the relationship between PCS moves, parenting stress, and family resilience influenced adolescent physiological health. Specifically, it was hypothesized: 1) there would be a significant positive association between PCS moves and scores on the ALI after adjusting for covariates; 2) Family resilience would moderate the association between PCS moves and ALI, such that as family resilience increases, the association between PCS moves and the ALI would decrease, after adjusting for covariates; and 3) parenting stress would moderate the association between PCS moves and ALI such that as parenting stress increases, the association between PCS moves and ALI will increase, after adjusting for covariates.

Contrary to expectations, the relationship between PCS moves and the ALI was not significant across any of the analyses. Analyses of individual predictors likewise demonstrated negligible effect sizes for PCS moves' impact on the ALI; a finding replicated across many of

the study's predictor variables. Further, there was no evidence to suggest either family resilience or parenting stress interacted to influence the relationship between PCS moves and the ALI.

This is the first study to specifically examine physical health outcomes in relation to PCS moves. Although research in military-connected youth is sparse, the wider civilian literature suggests relocation has deleterious physical effects in the presence of significant psychosocial stressors (27). Indeed, several studies demonstrate childhood adversity and dysfunctional family dynamics manifest as poor physical health as indicated by elevations in allostatic load scores (26; 116; 117; 320). Given these trends, it was expected the current study's inclusion characteristics of overweight, high trait anxiety, and/or LOC eating would confer sufficient vulnerability to maladaptation following PCS moves to culminate in evident physiological dysregulation. There are several possibilities as to why this study failed to replicate previous findings among at-risk youth.

Evaluation the ALI as a Measure of Cumulative Stress for Military-Associated Youth

One possible reason for the lack of significant findings is that the ALI may be an invalid measure of cumulative stress and “wear-and-tear” for military-associated youth. There are relatively few studies examining allostatic load in youth and it could be inadequate to for the purposes of assessing psychosocial stress. There are several critiques of the allostatic load concept germane to the current study which need to be addressed: 1) the applicability of cumulative physiological stress for youth samples with a myriad of health concerns; and 2) inconsistent operationalization of the allostatic load construct.

It is possible that youth – despite the presence of physical vulnerabilities – are unlikely to exhibit an elevated ALI due to the natural resilience of adolescence. The original ALI was validated on a geriatric population most likely to experience the cumulative stress-induced

physiological dysregulation for a lifetime ([193](#)). The process of senescence is associated with the attenuation of the body's physiological development which exacerbates cumulative stress load. On the other hand, adolescence is the period in which allostatic load would be least impactful and conflated with pubertal development, lifestyle behaviors, and/or cultural factors. It may be that military lifestyle stressors – to include PCS moves – contribute to discernable allostatic load profiles only as the body progresses into adulthood. Delayed onset for relocation-related sequelae has been documented in civilian samples. [Lin et al \(222\)](#) found relocation during adolescence, after adjusting for confounders, was associated with increased physical and psychological health complaints in middle adulthood. Similarly, [Webb et al \(431\)](#) found spikes in relocation frequency during adolescence corresponded to greater likelihood of adverse outcomes in adulthood.

It may also be the case that an at-risk sample may not be the most amenable to determining an association between psychosocial stressors and physical illness. Selecting at-risk youth based off elevated anxiety, LOC eating, and high weight may inadvertently introduce “floor” and “ceiling” effects thereby limiting the ability to detect discernable differences. Several of the biomarkers had an upper limit which, if surpassed, would preclude entry into the study. For example, a youth with diabetes or a serious anxiety condition requiring aggressive intervention would be excluded. In addition, the current sample consisted of youth with high weight/obesity. Because cardiometabolic and anthropometric outcomes are strongly associated with weight/adiposity, it is probable the inclusion criteria limited the present sample's variability. Visual inspection ALI's histogram demonstrates a slight positive skew with which partially supports the presence of a ceiling effect. That no participant received a maximum score of eight on the ALI is also suggestive of the aforementioned sample limitations. Despite these concerns,

visual inspection of the ALI's distribution confirms that the distribution is relatively normal suggesting that an at-risk sample has only a modest bearing on the utility of the ALI.

Aside from the applicability of the ALI with at-risk military youth, there is considerable heterogeneity in the operationalization of allostatic load which need to be addressed. The present study selected eight biomarkers based on availability and informed by past research ([190](#)). Although cardiovascular, anthropometric, and metabolic biomarkers were well-represented in the ALI, this study was unable to include several primary mediators (i.e., stress hormones, inflammatory markers). In other studies examining youth, [Evans et al \(117\)](#) and [Rogosch et al \(320\)](#) formulated an allostatic load composite which directly measured primary mediators such as epinephrine, norepinephrine, DHEA, and cortisol. Moreover, there is evidence that LOC eating – an inclusion criterion in the present study – is associated with chronic inflammation ([358](#)). Accordingly, the inability to include HPA-axis correlates/inflammatory biomarkers suggest the ALI was limited in detecting prodromal stress-related dysregulation.

PCS-Related Disruptions as Low-Threshold Stressors

Establishing the utility of the ALI for the purposes of this study, the most probable remaining explanation for the lack of significant findings are: 1) disruptions induced by PCS moves are not sufficiently stressful or lasting to manifest in long-term physiological dysregulation; and/or 2) the current resources/programs available to military youth attenuate the adverse impact of high mobility. Past surveys have suggested a majority of military families endorse PCS moves as one of the most consistent and distressing military lifestyle stressors ([121](#)). A descriptive analysis of the Life Event and Coping Inventory (LECI) in the current sample suggests military youth do not consider relocations as particularly stressful life events. In the current sample, self-reported stress reactivity when “moving to a new home (Item 4)” on the

LECI was in the lowest quartile of 123 life events with mean stress reactivity rated at 4.5 and a standard deviation of 2.2 (using a 9-point Likert scale). Of note, there was no statistically significant difference in mean stress reactivity between adolescents with no relocation history and adolescents with 1+ relocations.

It must be noted that the original validation of the LECI only presented psychometric data related to the cumulative life event score without providing detailed data as to the reliability or validity of the LECI's individual life events ([96](#)). Moreover, the original normative sample was limited to primarily white adolescents between the ages of 11 to 14 drawn from the larger civilian community. Despite these limitations, there are several justifications to support usage of a single-item to assess stress reactivity in the current sample. First, self-reported stress reactivity in response to relocation is sufficiently specific and common enough to be thought as reasonably face valid. Relatedly, the participants' specific score is less important when considering the relative ranking of PCS-related stress reactivity in the bottom quartile of over 100 stressful life events. The usage of a brief reactivity assessment measure has a precedent in the relocation literature. More specifically, [Bullock \(56\)](#) utilized a similar paradigm ([341](#)) to assess affective reactivity in regard to PCS moves in a sample of Canadian military youth. Some researchers have even proposed single-item measures demonstrate reasonable validity when used to assess generally familiar multi-modal constructs ([418](#)). A significant positive correlation ($r = .17, p = .04$) between Item 4 from the LECI (i.e., "You Moved to a New Home") and the total STAI-C score further suggests a relationship between single LECI item and overall stress sensitivity.

The evidence from the LECI in the present study corroborates other studies which support that relocation's impact on physical and psychological health is best explained by first/second-order disruptions induced or maintained by the move itself ([101](#); [117](#)). Specifically,

moving itself is only stressful insofar as the events preceding or following a move are stressful. For example, [Dong et al \(101\)](#) found the association between residential mobility and adverse health outcomes was primarily attributable to adverse childhood experiences (e.g., familial poverty/death) which prompted the relocation. Of the studies specific to youth, the researchers found the ALI was elevated in the presence of significant environmental stressors such as impoverishment and maltreatment ([116](#); [320](#)). This would suggest that PCS moves – despite their frequency – are unlikely to result in sustained psychosocial stress which would culminate in poor physical health, unless there are other significant environmental stressors present.

It is also probable that the first/second-order disruptions induced by PCS moves are attenuated by the military family readiness system and substantial financial/social support. Past research related to ALI in youth has found effects in the presence of sustained social stressors unrelated to the military. As discussed, a significant portion of the DOD personnel budget is spent on initiatives to strengthen military families to include: quality child-care and housing ([62](#)), access to unique medical needs through the Exceptional Family Member Program ([430](#)), transition assistance for students in the Department of Defense Education Activity (DODEA) school system ([38](#)), and support for Family Readiness Groups ([362](#)). This institutional support results in PCS moves that are expected, routine, and well-supported by DOD initiatives.

Notable adverse experiences which could instigate a relocation (e.g., death of a parent, eviction) are uncommon for PCS moves and are less generalizable to military samples. In fact, the aims of the parent study are a prime example of the institutional efforts to prioritize family well-being. Therefore, it is possible those youth who do endorse high stress reactivity to PCS-related disruptions are able to secure services and support from the wider military community. The prevalence of resources and support for military families may also explain why key family

processes (i.e., PSI-SF, FRAS) failed to moderate the association between PCS moves and the ALI.

Specific Aim 2

Specific aim 2 examined if the relationship between PCS moves, parenting stress, and family resilience influenced adolescent psychological health (i.e., PSS/BDI-II). Specifically, it was hypothesized: 1) there will be significant positive association between PCS moves and scores on the PSS and BDI-II after adjusting for covariates; 2) family resilience will moderate the association between PCS moves and PSS/BDI-II, such that as family resilience increases, the association between PCS moves and PSS/BDI-II will decrease, after adjusting for covariates; and 3) parenting stress will moderate the association between PCS moves and PSS/BDI-II such that as parenting stress increases, the association between PCS moves and PSS/BDI-II will increase, after adjusting for covariates.

PCS moves and Perceived Stress

It was expected that the present sample's inclusion criteria conferred sufficient vulnerability for post-PCS maladjustment. Indeed, surveys of military families have ranked PCS moves – and the associated first/second-order disruptions – as one of the most routine and significant military lifestyle stressors ([385](#)). The interaction between the sample's risk factors with PCS-related disruptions was expected to exacerbate stress reactivity as measured by the PSS. Indeed, this was partially supported in the data as only trait anxiety predicted scores on the PSS. This suggests baseline anxiety is a risk factor for greater appraised stress.

However, the findings of the present study suggest that despite significant comorbid vulnerabilities, military-associated youth with high weight have minimal adverse reactions to relocation stress. The diathesis of the sample for physical/psychological illness would be

expected to respond negatively to military relocation stress. This was not the case, however, as these findings bolster the evidence-base that military-associated youth exhibit resilience in response to PCS moves ([344](#); [383](#)). In fact, there is distinct trend in the data to suggest PCS moves themselves may confer resilience to appraised stress. Contrary to the hypothesized directionality, the present study found total PCS moves were negatively associated with PSS scores, such that participants who reported more moves also reported lower perceived stress. Across models, each additional PCS move was associated with an approximately 0.40 decrease in total PSS scores. The PSS may range from 0 to 40, therefore a 0.40 change in PSS scores is relatively modest. It should be noted PCS moves did not significantly predict perceived stress in all models. The other two analyses barely failed to meet the threshold for significance (i.e., $p = .053$; $p = .052$). These analyses differed by the inclusion or removal of several moderator variables (i.e., PSI-SF/FRAS). The most probable rationale for Models 1 and 3 (see Table 6) not being significant is likely a consequence of the modest shortfall in observed statistical power (see Table 3).

PCS moves and the Challenge Model of Resilience

The rationale for these unexpected findings is likely attributed to the challenge model of resilience ([213](#)). In the challenge model a putative risk factor (i.e., PCS moves), provided it does not induce excessive stress, confers resilience to future stressors. As discussed, the mean score for self-reported stress reactivity (LECI 4) to PCS moves was in the lowest quartile of 123 life events thereby supporting that PCS moves are a low-threshold stressor. In other words, if a PCS move is resolved successfully, it may enhance self-efficacy and responses to future military lifestyle stressors. This is consistent with the allostatic framework whereby these low-threshold stressors are met with successful adaptation thereby averting allostatic overload (see Figure 3).

The evidence for the challenge model is further bolstered by several other studies. Relocation frequency is often operationalized as mobility rate, a variable computed by dividing a participant's total relocations by their age ([144](#)). Mobility rate provides a valuable method of assessing the “dosage” of PCS moves over a participant's lifetime. For example, a 14-year-old adolescent with five relocations and a 14-year-old with 10 relocations would exhibit mobility rates of 0.36 and 0.71, respectively. If PCS moves follow a challenge model of resilience, it would be expected that a greater “dosage” of military relocation stress, as operationalized by mobility rate, would correspond to improved adolescent outcomes. Indeed, several studies support that greater relocation frequency is associated with beneficial youth outcomes ([144](#); [433](#)). For example, [Weber and Weber \(433\)](#) surveyed military parents to find higher mobility rates were superior at predicting positive adjustment than total relocations alone. Although the present study did not compute a mobility rate variable, the inclusion of age adjustments in the regression model served a similar function.

In addition to frequency, it is important to reconcile the extant literature on relocation recency for the present study. Past research has found externalizing disorders are associated with relocation ([344](#); [383](#)). However, in a recent article by [Perreault et al \(288\)](#), externalizing behavior was no longer significant when relocation recency (i.e., < 12 months) and parental stress were accounted for in a sample of Canadian military-associated youth. Accordingly, it is plausible that a normative adjustment period (i.e., “acting out”) is expected and followed by a return to pre-move functioning. Given the high frequency of relocations, it is probable a third of any representative sample of military adolescents recently relocated and are in the normative adjustment period. This may partially explain the presence of some findings regarding adverse outcomes to PCS moves. Unfortunately, the present study was unable to utilize relocation

recency or examine externalizing conditions as a viable predictor variable due to sample size limitations and methodological concerns. Regardless, the present study's findings in tandem with the literature on relocation frequency/recency would suggest military lifestyle stressors (i.e., PCS moves) induce time-sensitive disruptions to adolescent functioning and that, over time, exposure to such disruptions may confer resilience to perceived stress.

PCS moves and Depressive Symptoms

It was expected that PCS-related disruptions would interact with the sample's risk profiles to induce depressive symptoms as measured by the BDI-II. Although there was evidence that the sample's trait anxiety was predictive of depressive symptoms, there was no association between PCS moves and self-reported depressive symptoms in the present sample of vulnerable youth. In fact, only being female and greater trait anxiety were significantly associated with depressive symptoms in the present sample. It was hypothesized that relocation stress would occur as a result of first/second-order disruptions wherein military youth must navigate the stress of relocation, a novel environment, and new peer groups. Indeed, the symptoms of depression (e.g., rumination, altered help-seeking) are thought to aid adaptation to novel environment/stressors ([420](#)).

These findings are in contrast to previous studies wherein a relocation has been associated with youth depressive symptoms ([128](#)). Relocation recency appears to moderate the impact of PCS moves on depressive symptoms, with studies finding these depressive symptoms dissipating after roughly 12 months ([166](#)). Given the lack of an adequate assessment of relocation recency it is difficult to determine with youth are currently experiencing the sequelae of a recent relocation.

Family Resilience and Parenting Stress

The results also indicated family processes such as family resilience and parenting stress did not influence the relationship between PCS moves and the PSS. This is in contrast to two recent studies of the Canadian military by [Perreault et al \(288\)](#) and [Bullock \(56\)](#) which found parental perceived stress and the quality of the parent-child relationship moderated the relationship between military moves and adolescent outcomes. There are several potential explanations for this inconsistency. First, if the challenge model is applicable to PCS moves, the hypothesized protective or compensatory effects of family processes would be unnecessary to mitigate the mild and transitory impact of PCS-related disruptions. Second, past studies involving PCS moves examined community samples without examining adolescents with existing physical and psychological vulnerabilities. The relative impact of high trait anxiety, greater BMI, and/or LOC eating may dilute the importance of PCS moves of physical/psychological adjustment. In addition, the parents who voluntarily commit for an extended research study likely exhibit a modicum of functional family processes absent from a broader community sample. Lastly, as discussed previously, the existing support of the family readiness system may attenuate the disruptions associated with PCS moves.

Exploratory Aim 3

Exploratory aim 3 examined the utility of the ALI in detecting stress-based physiological dysregulation in the present sample. Specifically, three exploratory analyses were conducted to compare the original ALI, the adjusted ALI, MetS, and each of the constituent biomarkers. The intent behind the comparison of the three established clinical markers was to provide a sensitivity analysis for the present study's ALI formulation. It was expected the present study's ALI using - group-based norms and higher cut-offs - would be equal or superior to the other three clinical constructs in assessing stress-based physiological dysregulation.

The Adjusted ALI

This study differed from other studies in that the ALI's cut-offs were calculated using group-based norms in lieu of the sampling distribution ([437](#)). Group-based norms were considered to enhance both the internal and external validity of the study. In terms of internal validity, the inclusion age, sex, and/or race-adjusted norms was thought to enhance the ALI's validity to detect cumulative stress load in lieu of phenotypic differences. In terms of external validity, adolescent youth with overweight, elevated trait anxiety, and/or LOC eating are expected to exhibit high allostatic load. Reliance on the sampling distribution's cut-offs would limit generalizability of the results to other military-associated youth.

Given the relative dearth of research specific to the group-based normative formulation, a supplementary analysis was performed with an adjusted ALI calculated using the sampling distribution (See Table 8). The only notable finding was that males exhibited greater scores in the adjusted ALI. This is unsurprising and consistent with known phenotypic, metabolic, and cardiovascular differences between sexes ([445](#)). Based on the results, it is unlikely the adjusted ALI is superior in modeling cumulative physiological dysregulation. In fact, the results appear to indicate the group-based normative ALI effectively adjusted the model for sex-based physiological differences.

Metabolic Syndrome (MetS) and the Constituent Biomarkers

The ALI is thought to provide incremental validity over established clinical constructs, including MetS and the ALI's constituent biomarkers. Notably, LOC eating was not associated with MetS in the analysis. This contrasts with the past literature that has found relations between LOC eating, deregulated metabolism, inflammation, and stress. Sampling variability was limited due to the relatively homogenous adolescent sample selected for exhibiting high weight and

psychopathology. Such limitations in sampling variability may explain the discrepancy between past research and the present study's findings. Therefore, these results suggest markers other than MetS may be more sensitive at detecting stress-based physiological dysregulation with this relatively homogenous sample of high-risk youth.

The ALI is also thought to provide incremental validity over independent analyses of constituent biomarkers. The current study's analyses of individual biomarkers suggest that the ALI may be sufficiently sensitive method of operationalizing cumulative physiological dysregulation. The results of the regressions are summarized according to their respective biological system: cardiovascular (Table 10), metabolic (Table 11), and anthropometric (Table 12). Significant differences in the cardiovascular markers, metabolic variables, and anthropometric markers for gender were expected and in-line with recognized phenotypic variation ([309](#));([354](#)). For HDL and HbA1c, having an officer as a parent was associated with lower blood glucose and greater levels of HDL. This association may be related to findings suggesting parental educational attainment and socioeconomic circumstances improves their children's health outcomes ([258](#)). Military officers generally require an undergraduate education and generate significantly more earnings than their enlisted counterparts.

Out of the eight biomarkers used to formulate the ALI, only WC was associated with LOC eating in the present sample. These findings lend credibility for the overall construct validity of the ALI in the current sample. LOC eating has been associated with indices of poor physical health ([359](#)). Since LOC eating was associated with the ALI in the original analysis, this is initial evidence that the ALI may successfully captures the cumulative allostatic load conferred by the summation of individual biomarkers. Alternatively, at the very least, it might be said that

the ALI formulation is sensitive to each of its constituent biomarkers. It should be noted that no efforts were made to adjust for multiple analyses which required caution for interpretation.

Strengths and Limitations

This study has several strengths which address common shortfalls in relocation outcome research for children and adolescents. Historically, research has relied on parental perceptions of their children's functioning ([432](#)). Parental perceptions have been found to be highly discrepant from adolescent self-report – especially in stressed families ([88](#)). Accordingly, the current study's use of youth self-report in lieu of parental report is a relative strength. In addition, [Garboden et al \(144\)](#) suggest relocation outcome research's inconsistent results are, in part, a failure to adhere to a consistent operational definition for relocation. The military is uniquely suited to several methodological critiques introduced by [Garboden et al \(144\)](#). Specifically, military relocations are broadly standardized to be consistent in their operationalization and generally independent of common intervening variables (i.e., servicemember/ family choice in moving). Therefore, not only is the military in need for relocation outcome research but it is well suited to be studied in a scientifically rigorous method. Moreover, the presence of a highly diverse sample at-risk for physical/psychological illness would be those most likely to be adversely impacted by military relocation stress. Another prominent strength of this study is the objective measurement of anthropometric and cardiometabolic factors. Lastly, the inclusion of a measure of allostatic load represents a novel method of examining chronic stress in military adolescents.

This study also has several weaknesses that are important to consider. Most importantly, pertinent findings in the current study are caveated by limitations inherent to cross-sectional research using available baseline data thereby limiting casual inference. This limitation is

compounded by evidence suggesting allostatic dysregulation becomes evident in adulthood – years following the introduction of psychosocial stressors (90). For example, past research has noted adverse childhood experiences are associated with adult illness and psychopathology (101). Despite measures taken to increase the available data for analysis, the sample was modestly underpowered despite the use of multiple imputation which may have contributed to the inconsistent findings across analyses.

The results of the study may also be ungeneralizable to military youth to the wider civilian population. The sample also consisted entirely of treatment-seeking military dependents with overweight/obesity who endorsed trait anxiety and/or LOC eating. Also given the evidence of possible “ceiling” and “floor” effects from the parent study, it is probable this sample precludes some high-risk youth. Overall, it must be emphasized this sample is not representative of military youth in general and may not extend to other sub-populations.

Given the literature’s emphasis on relocation timing/recency, this study was further limited by its inability to operationalize relocation more broadly. It is quite plausible the developmental state of the child preceding the PCS move may influence adjustment. The present study did not examine the periods of early, middle, or late childhood. Instead, the study solely focused on the adolescent or teenage years due to the parent study’s inclusion criteria. No adjustment was feasible to account for the developmental state of the child during each of their moves.

Other unavoidable aspects of conducting a secondary data analysis also presented complications to include limitations in subjective reporting of relocation history, simplified demographic data, and time-inconsistent measure selection. The lack of an objective measure of relocation timing, recency, and duration is a relative limitation in the present study. Additionally,

the operationalization of parental rank was a binary classification not taking into account the nuances of dual-military families. There was also some inconsistency in the time period covered by individual measures. For example, the PSS asked to rate stress reactivity in the past month while the BDI-II evaluates depressive symptoms in the past two weeks. Although such measures have proven psychometric validation to a recognized clinical construct, there remains the possibility of the timing of such questions introducing error through contemporaneous adverse events.

Lastly, there were several limitations associated with the use of the ALI. Most importantly, the ALI used the available cardiometabolic biomarkers and could not incorporate primary mediators (i.e., cortisol) indicative of prodromal allostatic overload. In the present study the ALI was only calculated cross-sectionally and would benefit from longitudinal assessment of cumulative allostatic load.

Clinical and Systemic Implications

Following the modern military's transition into the AVF, the health and well-being of military dependents has been of increasing concern to both clinicians and policymakers. Past research has suggested military lifestyle factors (i.e., PCS moves) and family systems may dispose military-connected youth to adverse health outcomes and, therefore, serve as prime candidates for targeted intervention ([56](#)). This research has culminated into a number of intervention programs ([332](#)) specifically designed to foster family processes and mitigate the impact of military lifestyle stressors.

Clinical research has informed military leaders and policymakers' as to how military-connected youth impact national defense and readiness. Physically and psychologically resilient military-connected youth are critical to military readiness – either through their impact on

servicemember retention/performance or as candidates for recruitment. Accordingly, the present study's examination of PCS moves, parenting stress, family resilience, and physical/psychological health in vulnerable military-connected youth has several important clinical and systemic implications for youth within the military family readiness system.

Clinical Implications

The present study did not replicate previous findings that military-connected youth experience adverse adjustment following PCS moves. Even amongst this vulnerable sample, the data supports that military-connected youth are relatively resilient to relocation stress. In contrast to previous studies, modifiable family factors (i.e., resilience/parenting distress) also do not appear to influence the relationship between PCS moves and post-PCS adjustment ([56](#)). PCS moves appear to have minimal bearing on common psychological conditions (i.e., anxiety, depression), poor cardiometabolic health, and modifiable family factors in the present sample. These results challenge the prevailing narrative by some clinicians and researchers that the military lifestyle is damaging to the well-being of military families ([208](#)). In fact, a high-mobility military lifestyle – when properly supported – may actually confer resilience even amongst vulnerable youth samples. Accordingly, the results of this study suggest clinical attention and resources are best directed towards other military lifestyle factors (i.e., deployment, family loss) rather than relocation.

Despite relocation's modest impact on youth psychosocial functioning, the evidence from the present study suggest inclusion of PCS-related topics may be beneficial in fostering resilience. Community-based programs often include voluntary targeted interventions for servicemembers' families. As an illustrative example, Families OverComing Under Stress (FOCUS) delivers resilience training by enhancing family processes to common military

stressors ([332](#)). Several studies have supported such interventions improve family well-being across several indices of functioning ([219](#); [220](#)). Although the evidence for deleterious effects of relocation for adolescent youth is modest, the present study suggests that resilience processes from PCS moves may generalize across other military lifestyle stressors (i.e., deployment). Therefore, a prevention program should consider including discussions of PCS-related material. Overall, clinicians and researchers are encouraged to consider military lifestyle stressors, such as PCS moves, from a strengths-based perspective wherein the military experience is an opportunity for growth and development.

Systemic Implications

Clinical research and programmatic evaluations conducted by the DoD have provided valuable insights as to the disruptions induced by frequent PCS moves. The findings of this research has compelled the DoD to institute over 100 changes to the armed services relocation policies since the 1980s ([362](#)). Despite these changes, the armed forces relocation policy is still undergoing significant scrutiny. The present presidential administration has committed to several initiatives designed to increase recruitment for military youth and attend to the health needs of military families. Two of the proposed initiatives detail the importance of increasing time between PCS moves and access to medical services for military families.

The findings of the present study provide valuable insights for current initiatives for improving military family readiness. Even amongst a sample of vulnerable youth, there is little indication that the frequency of PCS moves has adverse health implications. In fact, the frequent PCS moves may actually foster youth resilience to future military lifestyle stressors through a challenge model of resilience. Although current relocation policy may still influence servicemembers' retention/attrition, the present findings do not support that increasing time

between PCS moves will have a significant clinical impact on youth well-being. This study also suggests contextualizing PCS moves as a growth experience may foster resilience to more severe military stressors such as parental separation, deployment, or death. Preventative strategies (i.e., FOCUS) would mitigate the need for time intensive and expensive outpatient services for military-associated youth.

Future Directions

Future research should expand on the current study's limitations by including longitudinal designs, operationalize relocation to incorporate PCS recency, and replicate current findings with larger community samples. These recommendations would provide valuable avenues for clinical research while informing the DoD relocation policy. First, the incorporation of a longitudinal design may clarify the downstream impact of PCS moves for military youth. As discussed, research supports stress-induced allostatic overload during adolescence may only become perceptible in adulthood ([90](#)). Future research should assess relocation characteristics and physiological functioning across the developmental span to include: adolescence, adulthood, and geriatrics. With such information, DoD policy makers could better determine the exact implications of relocation policy to increase family readiness and secure potential recruits for military service.

Future research should also incorporate relocation recency. Prior research suggests a recent relocation may induce time-sensitive disruptions to adolescent well-being, and that such disruptions can be conflated with lasting psychological sequelae ([56](#)). A more thorough review of relocation history may provide insight as to the common first/second-order disruptions which instigate or maintain adverse adjustment to PCS moves. Understanding the specific disruptions

concurrent with PCS moves would provide clinicians and policymakers insight as to points of intervention.

This study also focused primarily on what was considered a “vulnerable” sample for the eventual development of physical/psychological illness. However, larger community samples provide an opportunity to replicate the current findings in a more generalizable sample. It is important to determine if the present cohort’s risk profile mitigated the impact of frequent PCS moves. It is plausible the relative impact is greater in a community sample due to the stress load experienced in the present study’s sample. Such findings would inform the viability of general prevention interventions versus more intensive treatment for military-associated youth.

Conclusion

In conclusion, the findings from this study added to the existing literature on PCS moves, modifiable family risk/resilience factors, and the physical/psychological adjustment in the military context. Understanding the impact of PCS moves on adolescent military dependents has important implications for clinical care, policy, and military readiness. The study results refute a number of common misconceptions regarding the deleterious effect of PCS moves for military-associated youth. Future studies should elaborate on the present findings by longitudinal research, incorporating relocation recency, and drawing from a larger community sample. Considering the prevalence of PCS moves, clinical researchers developing targeted interventions and DoD policymakers allocating resources must understand the possible benefits and drawbacks to the highly mobile military lifestyle.

Table 1. Allostatic Load Candidate Biomarkers with Cut-Offs and References

Type	Biomarker	Description	Normative Cut-Off	Ref.	Sample (%) Meeting Normative Cut-Off
METABOLIC	High-density lipoprotein cholesterol	Lipoprotein synthesized in the liver. Transports cholesterol from tissues to the liver. Commonly referred to as “good cholesterol”, as its high protein/low cholesterol form is more easily removed by blood in the liver and excreted in bile.	$\leq 5\%$ Age/Sex	(171)	5.8%
	Low-density lipoprotein cholesterol	Lipoprotein synthesized in the liver. Transports cholesterol to tissues that synthesize cell membranes and secretions. Commonly referred to as “bad cholesterol”, as its low protein/high cholesterol form is more likely to be deposited in the walls of blood vessels and contribute to atherosclerosis.	$\geq 95\%$ Age/Sex	(171)	9.7%
	Triglycerides	Glyceride formed from glycerol and three chains of fatty acids. Functions as an important source of energy and as a transporter of dietary fat.	$\geq 95\%$ Age/Sex	(171)	5.6%
	Glycosylated hemoglobin	Hemoglobin used to index the average glucose concentration over many days, weeks and even months. This proportion represents the amount of glucose that the analyzed hemoglobin has been exposed to during its cell cycle.	$\geq 85\%$ Sex	(328)	22.5%
CARDIOVASCULAR	Systolic blood pressure	Measured using a sphygmomanometer. Represents the maximal force exerted by blood against the blood vessel walls when the left ventricle is contracting during systole.	$\geq 95\%$ Age/Sex/Height	(317)	18.5%
	Diastolic blood pressure	Measured using a sphygmomanometer. Represents the minimal force exerted by blood against the blood vessel walls when the left ventricle is relaxed during diastole.	$\geq 95\%$ Age/Sex/Height	(317)	11.1%
ANTHROPOMETRIC	Waist Circumference	Measure of waist circumference using measuring tape values that are then calculated into a ratio by dividing waist by hip. Higher levels represent greater visceral adipose fat distribution obese individuals. Body shapes that are commonly referred to as “apple shapes” (greater waist size) are considered to be at greater risk of health problems.	$\geq 90\%$ Age/Sex/Ethnicity	(127)	81.9%
	Body mass index	Measure of weight and height that is then calculated into an index by dividing weight by height. Represents a proxy measure of an individual’s relative body fat percentage.	$\geq 95\%$ Age/Sex	(206)	75%

*Other common biomarkers for Allostatic Load: Cortisol, Dehydroepiandrosterone, Epinephrine, Norepinephrine, Dopamine, Aldosterone, Interleukin-6, Tumor necrosis factor-alpha, C-reactive protein, Insulin-like growth factor-1, Fibrinogen, Albumin, Creatinine, Homocysteine, Peak expiratory flow, Heart rate/pulse; adapted from Juster, R.P., McEwen, B.S., Lupien, S.J., 2010

Table 2. Power Analyses for Aims

Hypothesis	Type of Test	# of IV/Covariates	Sample Required (N)	Observed Power (%)	MI Achieved Power (%)
1a	MLR	1/6	167	67%	79%
1b	MLR	3/6	183	54%	74%
1c	MLR	3/6	183	60%	74%
2a	MLR	1/7	175	62-65%	77%
2b	MLR	3/7	190	49-51%	72%
2c	MLR	3/7	190	55-58%	72%

Note. MLR = Multiple Linear Regression

Table 3. Baseline Participants Characteristics

	Total (N = 164)	Relocation (n = 127, 77.4%)	No Relocation (n = 37, 22.5%)	p
	<i>n (%)</i>	<i>n (%)</i>	<i>n (%)</i>	
Sex*				.80
Male	68 (41.5%)	52 (40.9%)	16 (43.2%)	
Female	96 (58.5%)	75 (59.1%)	21 (56.8%)	
Parental Rank*				.20
Officer/Warrant	96 (58.5%)	77 (60.6%)	19 (51.3%)	
Enlisted	59 (36.0%)	42 (33.1%)	17 (46.0%)	
Missing/Unknown	9 (5.5%)	8 (6.3%)	1 (2.7%)	
Race*				.59
White/Caucasian	95 (57.9%)	75 (59.1%)	20 (54.1%)	
Black/African American	36 (22.0%)	25 (19.7%)	11 (29.7%)	
Multiple Races	20 (12.2%)	17 (13.4%)	3 (8.1%)	
Asian	5 (3.0%)	3 (2.4%)	2 (5.4%)	
Unknown	4 (2.4%)	4 (3.2%)	0 (0.0%)	
Ethnicity*				.21
Hispanic or Latino	36 (22.0%)	31 (24.4%)	5 (13.5%)	
Not Hispanic or Latino	118 (72.0%)	90 (70.9%)	28 (75.7%)	
Unknown	10 (6.1%)	6 (4.7%)	4 (10.8%)	
LOC Eating*				.82
Presence	78 (47.6%)	61 (48.0%)	17 (45.9%)	
Absence	86 (52.4%)	66 (52.0%)	20 (54.1%)	
STAI-C**				.46
<32	11 (6.7%)	10 (7.3%)	1 (2.7%)	
≥32	151 (92.1%)	115 (84.1%)	36 (97.3%)	
Unknown/Missing	2 (1.2%)	2 (1.6%)	0 (0%)	
Range	27-54	27-54	29-54	
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	
Cumulative PCS Moves	3.5 (3.0)	4.5 (2.6)	0 (0)	
Range	0 - 14	1-14	N/A	
Age (years)**	14.5 (1.6)	14.4 (1.5)	14.8 (1.6)	.09
BMI**	30.5 (4.4)	30.1 (4.8)	29.5 (3.0)	.37
BMI-z**	1.9 (0.4)	1.9 (0.4)	1.9 (0.3)	.46

Note. *p-values computed via chi-squared tests (at least one relocation vs. no history of relocation): Sex (Male/Female); Race (White/Black/Asian/Multiple Races/Unknown); Ethnicity (non-Hispanic/Hispanic); Loss-of-control (LOC) Eating (endorsed LOC eating at least on month prior vs. No LOC eating at least one month prior) **p-values computed via Independent Samples T-Test (at least one relocation vs. no history of relocation): STAI-C = State Trait Anxiety Inventory – Children; BMI = Body Mass Index; BMI-z = Body Mass Index adjusted for age/sex.; PCS moves = Permanent Change of Station (PCS) moves.

Table 4. Summary of Missing Values Analyses

	Observed Data				Mean-Imputation Data				Multiple Imputation Data (Pooled)	
	Complete <i>n</i> (%)			Incomplete <i>n</i> (%)	Complete <i>n</i> (%)			Incomplete <i>n</i> (%)	Complete <i>N</i> (%)	
Missing Data										
Variables	5 (26.3%)			14 (73.7%)	6 (31.6%)			13 (68.4%)	19 (100%)	
Cases	88 (53.7%)			76 (46.3%)	110 (67.1%)			54 (32.9%)	164 (100%)	
Values	2,982 (95.7%)			134 (4.3%)	3,015 (96.8%)			101 (3.2%)	3,116 (100%)	
	<i>M</i>	<i>SD</i>	<i>n</i> (%)	<i>n</i> (%)	<i>M</i>	<i>SD</i>	<i>n</i> (%)	<i>n</i> (%)	<i>M</i>	<i>SE</i>
ALI	2.3	1.3	142 (86.6%)	22 (13.4%)	2.3	1.3	142 (13.4%)	22 (13.4%)	2.3	.10
FRAS*	170.1	19.0	142 (86.6%)	22 (13.4%)	169.9	19.1	151 (92.1%)	13 (7.9%)	169.6	1.6
PSS*	26.3	7.5	143 (87.2%)	21 (12.8%)	26.4	7.5	152 (92.7%)	12 (7.3%)	26.4	.66
BDI-II*	12.8	7.5	15 (90.9%)	15 (9.1%)	12.8	7.5	164 (100.0%)	0 (0.00%)	12.7	.61
PSI-SF*	67.0	20.6	159 (97.0%)	5 (3.0%)	67.0	20.6	159 (97.0%)	5 (3.0%)	67.1	1.6
STAI-C	38.9	5.9	162 (98.8%)	2 (1.2%)	38.9	5.9	162 (98.8%)	2 (1.2%)	38.9	.46
Rank (Parental)	-	-	9 (94.5%)	9 (5.5%)	-	-	9 (94.5%)	9 (5.5%)	-	-
Race/Ethnicity	-	-	157 (95.7%)	7 (4.3%)	-	-	157 (95.7%)	7 (4.3%)	-	-
LOC Eating	-	-	164 (100%)	0 (0%)	-	-	164 (100%)	0 (0%)	-	-
Sex	-	-	164 (100%)	0 (0%)	-	-	164 (100%)	0 (0%)	-	-
Age	14.5	1.6	164 (100%)	0 (0%)	14.5	1.6	164 (100%)	0 (0%)	14.5	.12
PCS Total	3.5	3.0	164 (100%)	0 (0%)	3.5	3.0	164 (100%)	0 (0%)	3.5	2.3
WC	96.0	12.0	13 (92.1%)	13 (7.9%)	96.0	12.0	13 (92.1%)	13 (7.9%)	96.2	.97
HbA1C	5.3	0.3	9 (94.5%)	9 (5.5%)	5.3	0.3	9 (94.5%)	9 (5.5%)	5.3	.02
LDL	103.6	31.4	157 (95.7%)	7 (4.3%)	103.6	31.4	157 (95.7%)	7 (4.3%)	103.8	2.5
DBP	72.5	9.2	157 (95.7%)	7 (4.3%)	72.5	9.2	157 (95.7%)	7 (4.3%)	72.5	.73
SBP	118.8	12.2	157 (95.7%)	7 (4.3%)	118.8	12.2	157 (95.7%)	7 (4.3%)	118.9	.98
HDL	50.6	12.3	159 (97.0%)	5 (3.0%)	50.6	12.3	159 (97.0%)	5 (3.0%)	50.4	.98
TRI	92.1	47.7	159 (97.0%)	5 (3.0%)	92.1	47.7	159 (97.0%)	5 (3.0%)	92.6	3.9
BMI-z	1.9	.39	164 (100%)	0 (0.0%)	1.9	.39	164 (100%)	0 (0.0%)	1.9	.39
Valid <i>n</i> Listwise	Pre-Imputation <i>n</i> = 88				Post-Imputation <i>n</i> = 110				Post-Imputation <i>N</i> = 164	

Note. *Mean-Person Imputed Variables; Abbreviations: ALI = Allostatic Load Index, FRAS = Family resilience Assessment Scale, PSS = Perceived Stress Scale, BDI-II = Beck Depression Inventory II, PSI-SF = Parenting Stress Index - Short Form, STAI-C = State Trait Anxiety Inventory – Children, WC = Waist Circumference, HbA1C = Hemoglobin A1C, LDL = Low-Density Lipoproteins, HDL = High-Density Lipoproteins, DBP = Diastolic Blood Pressure, SBP = Systolic Blood Pressure, TRI = Triglycerides, BMI-z = Body Mass Index adjusted for age/sex, Rank (Parental) = Warrant Officer/Officer vs. enlisted, Race/Ethnicity = Non-Hispanic White vs. Other, Loss-of-Control (LOC) Eating Presence = Endorsed LOC eating at least on month prior; PCS moves = Permanent Change of Station moves; **Bolded** terms were used in the final imputation model.

Table 5. Summary of Linear Regression Analyses between PCS and ALI

	Observed/Baseline Data					Multiple Imputation Data (Pooled)			
MODEL # (Valid Listwise)	R^2	Adjusted R^2	F	p		R^2	Adjusted R^2	F	p
MODEL 1 ($n = 131$)	.08	.03	1.47	.19	-	-	-	-	-
MODEL 2 ($n = 114$)	.10	.02	1.31	.24	-	-	-	-	-
MODEL 3 ($n = 127$)	.10	.03	1.46	.17	-	-	-	-	-
	b	SE(b)	95% CI	β	p	b	SE(b)	95% CI	p
MODEL 1									
PCS Total	-0.04	0.40	[-0.12, 0.04]	-.10	.31	-0.02	0.04	[-0.09, 0.05]	.66
Age (Months)	-0.002	0.01	[-0.01, 0.01]	-.03	.78	-0.001	0.01	[-0.01, 0.01]	.86
Sex	0.37	0.24	[-0.10, 0.85]	.14	.12	0.41	0.21	[-0.01, 0.83]	.06
Rank (Parental)	-0.26	0.25	[-0.76, 0.23]	-.10	.29	-0.24	0.22	[-0.67, 0.19]	.28
White (Non-Hispanic)	-0.31	0.23	[-0.76, 0.15]	-.12	.18	-0.33	0.21	[-0.75, 0.09]	.12
LOC Eating Past Month	0.39	0.23	[-0.07, 0.84]	.15	.09	0.43	0.20	[0.03, 0.82]	.03*
STAI-C	0.004	0.02	[-0.04, 0.04]	.02	.83	0.003	0.02	[-0.03, 0.04]	.88
MODEL 2									
PCS Total	<0.00	0.05	[-0.09, 0.09]	-.001	.99	-0.01	0.04	[-0.08, 0.07]	.85
Age (Months)	-0.004	0.01	[-0.02, 0.01]	-.06	.57	-0.003	0.01	[-0.01, 0.01]	.62
Sex	0.36	0.25	[-0.14, 0.87]	.14	.16	0.43	0.21	[0.02, 0.84]	.04*
Rank (Parental)	-0.40	0.27	[-0.94, 0.14]	-.15	.14	-0.29	0.22	[-0.72, 0.15]	.19
White (Non-Hispanic)	-0.20	0.25	[-0.69, 0.29]	-.08	.42	-0.31	0.22	[-0.74, 0.11]	.15
LOC Eating Past Month	0.41	0.24	[-0.07, 0.90]	.16	.09	0.38	0.21	[-0.03, 0.78]	.07
STAI-C	0.01	0.02	[-0.03, .06]	.06	.54	0.01	0.02	[-0.03, 0.04]	.74
FRAS	-0.01	0.01	[-0.02, .001]	-.17	.08	-0.01	0.01	[-0.02, 0.002]	.12
Interaction (PCS Total * FRAS)	0.01	0.12	[-0.23, 0.25]	.01	.95	0.04	0.12	[-0.21, 0.28]	.78
MODEL 3									
PCS Total	-0.05	0.04	[-0.13, 0.04]	-.10	.28	-0.02	0.04	[-0.09, 0.06]	.68
Age (Months)	-0.003	0.01	[-0.02, 0.01]	-.05	.60	-0.001	0.01	[-0.01, 0.01]	.84
Sex	0.42	0.25	[-0.06, 0.91]	.16	.09	0.41	0.21	[-0.004, 0.83]	.051
Rank (Parental)	-0.29	0.26	[-0.79, 0.22]	-.11	.26	-0.26	0.22	[-0.69, 0.18]	.24
White (Non-Hispanic)	-0.32	0.24	[-0.79, 0.14]	-.12	.17	-0.34	0.21	[-0.76, 0.08]	.11
LOC Eating Past Month	0.36	0.24	[-0.11, 0.83]	.14	.13	0.40	0.20	[-0.002, 0.80]	.051
STAI-C	0.01	0.02	[-0.03, 0.05]	.05	.58	0.003	0.02	[-0.03, 0.04]	.86
PSI-SF	0.01	0.01	[-0.004, 0.02]	.12	.20	0.01	0.01	[-0.01, 0.02]	.30
Interaction (PCS Total * PSI-SF)	0.07	0.13	[-0.18, 0.32]	.05	.60	0.11	0.11	[-0.10, 0.32]	.31

Note. DV = Allostatic Load Index (ALI); *Significant at $ps < .05$. Abbreviations: FRAS = Family Resilience Assessment Scale, PSI-SF = Parenting Stress Index - Short Form, Rank (Parental) = Warrant Officer/Officer vs. enlisted, Race/Ethnicity = Non-Hispanic White vs. Other, Loss-of-control (LOC) Eating Presence = Endorsed LOC eating at least on month prior; Sex = Male vs. Female, PCS moves = Permanent Change of Station moves, STAI-C = State Trait Anxiety Inventory – Children.

Table 6. Summary of Linear Regression Analyses between PCS and PSS

MODEL # (Valid Listwise)	Observed/Baseline Data					Multiple Imputation Data (Pooled)			
	R^2	Adjusted R^2	F	p		R^2	Adjusted R^2	F	p
MODEL 1 ($n = 127$)	.26	.21	5.01	< .001*		-	-	-	-
MODEL 2 ($n = 110$)	.27	.19	3.17	< .001*		-	-	-	-
MODEL 3 ($n = 123$)	.25	.18	3.73	< .001*		-	-	-	-
	b	SE(b)	95% CI	β	p	b	SE(b)	95% CI	p
MODEL 1									
PCS Total	-0.39	0.22	[-0.81,0.04]	-.15	.08	-0.40	0.20	[-0.80,0.004]	.053
Age (Months)	0.05	0.03	[-0.02,0.11]	.11	.18	0.05	0.03	[-0.01,0.12]	.09
Sex	-1.07	1.32	[-3.69,1.55]	-.07	.42	-0.40	1.17	[-2.70,1.89]	.73
Rank (Parental)	1.61	1.36	[-1.08,4.31]	.10	.24	2.11	1.48	[-0.90,5.12]	.16
White (Non-Hispanic)	-1.91	1.23	[-4.35,0.54]	-.13	.13	-1.67	1.10	[-3.83,0.49]	.13
LOC Eating Past Month	0.21	1.22	[-2.21,2.63]	.01	.87	0.19	1.18	[-2.15,2.53]	.87
BMIz	-2.59	1.70	[-5.96,0.77]	-.13	.13	-2.01	1.62	[-5.24,1.22]	.22
STAI-C	0.51	0.11	[0.29,0.73]	.38	< .001*	0.49	0.10	[0.30,0.68]	< .001*
MODEL 2									
PCS Total	-0.53	0.27	[-1.07,-0.01]	-.21	.048*	-0.42	0.21	[-0.83,-0.01]	.044*
Age (Months)	0.04	0.04	[-0.04,0.11]	.09	.33	0.05	0.03	[-0.01,0.11]	.11
Sex	-1.12	1.44	[-3.98,1.73]	-.07	.44	-0.42	1.15	[-2.68,1.84]	.72
Rank (Parental)	2.28	1.52	[-0.73,5.29]	.15	.14	2.14	1.43	[-0.74,5.03]	.14
White (Non-Hispanic)	-2.64	1.35	[-5.32,0.05]	-.17	.054	-1.62	1.11	[-3.80,0.55]	.14
LOC Eating Past Month	-0.56	1.35	[-3.23,2.11]	-.04	.68	0.20	1.23	[-2.24,2.63]	.87
BMIz	-2.02	1.83	[-5.65,1.62]	-.10	.27	-1.99	1.70	[-5.40,1.42]	.25
STAI-C	0.49	0.12	[0.25,0.73]	.37	< .001*	0.49	0.10	[0.30,0.69]	< .001*
FRAS	0.00	0.04	[-0.07,0.07]	.001	.99	0.001	0.04	[-0.07,0.08]	.98
Interaction (PCS Total * FRAS)	0.88	0.65	[-0.41,2.17]	.12	.18	0.43	0.58	[-0.71,1.58]	.45
MODEL 3									
PCS Total	-0.37	0.22	[-0.81,0.06]	-.15	.09	-0.40	0.20	[-0.80,0.003]	.052
Age (Months)	0.05	0.04	[-0.02,0.12]	.12	.17	0.06	0.03	[-0.01,0.12]	.08
Sex	-1.00	1.35	[-3.67,1.68]	-.06	.46	-0.33	1.15	[-2.59,1.93]	.77
Rank (Parental)	1.86	1.40	[-0.90,4.63]	.12	.19	2.04	1.41	[-0.79,4.87]	.15
White (Non-Hispanic)	-1.66	1.27	[-4.17,0.86]	-.11	.20	-1.66	1.10	[-3.83,0.50]	.13
LOC Eating Past Month	-0.06	1.27	[-2.57,2.46]	-.004	.97	0.11	1.24	[-2.37,2.59]	.93
BMIz	-3.12	1.78	[-6.64,0.40]	-.15	.08	-2.10	1.67	[-5.44,1.23]	.21
STAI-C	0.49	0.11	[0.26,0.71]	.37	< .001*	0.49	0.10	[0.30,0.69]	< .001*
PSI-SF	0.01	0.03	[-0.05,0.08]	.03	.71	0.02	0.04	[-0.07,0.10]	.70
Interaction (PCS Total * PSI)	-0.23	0.71	[-1.63,1.18]	-.03	.75	-0.20	0.60	[-1.38,0.98]	.74

Note. DV = PSS (Perceived Stress Scale); *Significant at $ps < .05$; Abbreviations: FRAS = Family resilience Assessment Scale, PSI-SF = Parenting Stress Index - Short Form, Rank (Parental) = Warrant Officer/Officer vs. enlisted, Race/Ethnicity = Non-Hispanic White vs. Other, Loss-of-control (LOC) Eating Presence = Endorsed LOC eating at least on month prior; Sex = Male vs. Female, PCS moves = Permanent Change of Station moves, STAI-C = State Trait Anxiety Inventory – Children, BMIz = Body Mass Index Z-scores.

Table 7. Summary of Linear Regression Analyses between PCS and BDI-II

MODEL # (Valid Listwise)	Observed/Baseline Data					Multiple Imputation Data (Pooled)			
	R^2	Adjusted R^2		F	p	R^2	Adjusted R^2	F	p
MODEL 1 ($n = 132$)	.25	.20		5.11	< .001*	-	-	-	-
MODEL 2 ($n = 114$)	.30	.23		4.34	< .001*	-	-	-	-
MODEL 3 ($n = 129$)	.25	.18		3.89	< .001*	-	-	-	-
	b	SE(b)	95% CI	β	p	b	SE(b)	95% CI	p
MODEL 1									
PCS Total	-0.04	0.20	[-0.43,0.36]	-.02	.86	-0.15	0.19	[-0.53,0.23]	.45
Age (Months)	-0.02	0.03	[-0.09,0.04]	-.06	.49	-0.03	0.03	[-0.09,0.03]	.26
Sex	-2.89	1.27	[-5.41,-0.38]	-.19	.03*	-3.53	1.21	[-5.93,-1.14]	.004*
Rank (Parental)	-0.53	1.29	[-3.09,2.02]	-.04	.68	-0.20	1.27	[-2.71,2.31]	.88
White (Non-Hispanic)	-0.61	1.18	[-2.95,1.73]	-.04	.61	-0.38	1.18	[-2.71,1.96]	.75
LOC Eating Past Month	0.61	1.19	[-1.74,2.95]	.04	.61	0.46	1.13	[-1.78,2.69]	.69
BMIz	-0.09	1.59	[-3.24,3.05]	-.01	.95	0.59	1.50	[-2.37,3.56]	.69
STAI-C	0.56	0.10	[0.36,0.76]	.44	<.001*	0.57	0.01	[0.39,0.76]	<.001*
MODEL 2									
PCS Total	0.03	0.24	[-0.45,0.51]	.13	.89	-0.12	0.21	[-0.53,0.30]	.57
Age (Months)	-0.02	0.04	[-0.09,0.05]	-.05	.61	-0.03	0.03	[-0.10,0.03]	.29
Sex	-2.27	1.35	[-4.95,0.40]	-.15	.10	-3.49	1.24	[-5.96,-1.02]	.01*
Rank (Parental)	-0.51	1.41	[-3.30,2.29]	-.03	.72	-0.28	1.29	[-2.84,2.28]	.83
White (Non-Hispanic)	-1.26	1.27	[-3.79,1.27]	-.09	.33	-0.42	1.19	[-2.78,1.94]	.72
LOC Eating Past Month	0.13	1.27	[-2.39,2.64]	.01	.92	0.40	1.14	[-1.84,2.63]	.73
BMIz	0.33	1.68	[-3.01,3.67]	.02	.85	0.52	1.59	[-2.65,3.68]	.75
STAI-C	0.60	0.11	[0.38,0.81]	.48	<.001*	0.57	0.10	[0.38,0.77]	<.001*
FRAS	-0.01	0.03	[-0.07,0.06]	-.02	.81	-0.01	0.03	[-0.08,0.06]	.76
Interaction (PCS Total * FRAS)	-0.90	0.60	[-2.10,0.29]	-.13	.14	-0.37	0.51	[-1.37,0.64]	.47
MODEL 3									
PCS Total	-0.03	0.21	[-0.43,0.38]	-.01	.90	-0.15	0.20	[-0.53,0.24]	.45
Age (Months)	-0.03	0.03	[-0.10,0.04]	-.08	.38	-0.03	0.03	[-0.09,0.03]	.27
Sex	-2.83	1.29	[-5.38,-0.28]	-.19	.03*	-3.52	1.22	[-5.94,-1.10]	.01*
Rank (Parental)	-0.27	1.32	[-2.88,2.35]	-.02	.84	-0.22	1.28	[-2.75,2.32]	.87
White (Non-Hispanic)	-0.72	1.21	[-3.11,1.67]	-.05	.55	-0.37	1.19	[-2.73,1.98]	.75
LOC Eating Past Month	0.69	1.22	[-1.73,3.10]	.05	.57	0.44	1.15	[-1.82,2.69]	.70
BMIz	-0.20	1.65	[-3.46,3.06]	-.01	.90	0.57	1.53	[-2.45,3.60]	.71
STAI-C	0.55	0.10	[0.35,0.76]	.44	<.001*	0.57	0.10	[0.39,0.76]	<.001*
PSI-SF	-0.002	0.03	[-0.06,0.06]	-.004	.96	0.003	0.03	[-0.05,0.06]	.90
Interaction (PCS Total * PSI)	0.54	0.64	[-0.72,1.81]	.069	.40	-0.05	0.56	[-1.14,1.05]	.93

Note. DV = BDI-II (Beck Depression Inventory-II); *Significant at $ps < .05$; FRAS = Family resilience Assessment Scale, PSI-SF = Parenting Stress Index – Short Form, Rank (Parental) = Warrant Officer/Officer vs. enlisted, Race/Ethnicity = Non-Hispanic White vs. Other, Loss-of-control (LOC) Eating Presence = Endorsed LOC eating at least on month prior; Sex = Male vs. Female, PCS moves = Permanent Change of Station moves, STAI-C = State Trait Anxiety Inventory – Children, BMIz = Body Mass Index Z-scores.

Table 8. Summary of Linear Regression Analyses between PCS and Adjusted ALI

MODEL # (Valid Listwise)	Observed/Baseline Data					Multiple Imputation Data (Pooled)			
	R^2	Adjusted R^2	F	p		R^2	Adjusted R^2	F	p
MODEL 1 ($n = 147$)	.01	.05	2.13	.045*		-	-	-	-
	b	SE(b)	95% CI	β	p	b	SE(b)	95% CI	p
MODEL 1									
PCS Total	0.06	0.05	[-0.04,0.16]	.10	.23	0.06	0.05	[-0.03,0.16]	.20
Age (Months)	0.01	0.01	[-0.01,0.03]	.11	.20	0.01	0.01	[-0.004,0.03]	.15
Sex	0.90	0.31	[0.30,1.51]	.25	.004*	1.14	0.29	[0.57,1.72]	<.001*
Rank (Parental)	-0.52	0.32	[-1.16,0.13]	-.14	.11	-0.59	0.31	[-1.19,0.01]	.05
White (Non-Hispanic)	-0.10	0.30	[-0.68,0.49]	-.03	.74	-0.01	0.28	[-0.65,0.46]	.73
LOC Eating Past Month	0.50	0.30	[-0.08,1.09]	.14	.09	0.47	0.28	[-0.07,1.01]	.09
STAI-C	-0.01	0.03	[-0.06,0.04]	-.03	.74	0.01	0.02	[-0.04,0.05]	.79

Note. DV = Adjusted ALI (Allostatic Load Index) *Significant at $ps < .05$; Abbreviations: FRAS = Family resilience Assessment Scale, PSI-SF = Parenting Stress Index - Short Form, Rank (Parental) = Warrant Officer/Officer vs. enlisted, Race/Ethnicity = Non-Hispanic White vs. Other, Loss-of-control (LOC) Eating Presence = Endorsed LOC eating at least on month prior; Sex = Male vs. Female, PCS moves = Permanent Change of Station moves, STAI-C = State Trait Anxiety Inventory – Children, BMIz = Body Mass Index Z-scores.

Table 9: Summary of Logistic Regression Analyses between PCS and MetS

	Observed/Baseline Data					Multiple Imputation Data (Pooled)				
	<i>B</i>	<i>SE(B)</i>	95% CI	<i>Exp(B)</i>	<i>p</i>	<i>B</i>	<i>SE(b)</i>	95% CI	<i>Exp(B)</i>	<i>p</i>
Model 1: MetS										
PCS Total	0.01	0.08	[0.89,1.24]	1.05	.55	0.01	0.08	[0.86,1.18]	1.01	.92
Age (Months)	-0.02	0.01	[0.97,1.02]	0.99	.56	-0.02	0.01	[0.96,1.01]	.98	.22
Sex	0.79	0.49	[0.91,6.18]	2.37	.08	0.79	0.46	[0.90,5.41]	2.21	.08
Rank (Parental)	0.19	0.54	[0.32,2.59]	0.90	.85	0.19	0.50	[0.45,3.23]	1.21	.71
White (Non-Hispanic)	-0.70	0.50	[0.24,1.72]	0.65	.38	-0.70	0.51	[0.18,1.36]	0.50	.17
LOC Eating Past Month	0.50	0.49	[0.50,3.35]	1.29	.60	0.50	0.46	[0.67,4.08]	1.65	.28
STAI-C	0.01	0.04	[0.90,1.07]	0.98	.67	0.01	0.04	[0.93,1.09]	1.01	.91

Note. DV = MetS (Metabolic Syndrome) *Significant at $ps < .05$; Abbreviations: FRAS = Family resilience Assessment Scale, PSI-SF = Parenting Stress Index - Short Form, Rank (Parental) = Warrant Officer/Officer vs. enlisted, Race/Ethnicity = Non-Hispanic White vs. Other, Loss-of-control (LOC) Eating Presence = Endorsed LOC eating at least on month prior; Sex = Male vs. Female, PCS moves = Permanent Change of Station moves, STAI-C = State Trait Anxiety Inventory – Children, BMIz = Body Mass Index Z-scores.

Table 10. Summary of Linear Regression Analyses between PCS and Cardiovascular Biomarkers

	Observed/Baseline Data					Multiple Imputation Data (Pooled)			
	<i>b</i>	SE(<i>b</i>)	95% CI	β	<i>p</i>	<i>b</i>	SE(<i>b</i>)	95% CI	<i>p</i>
Systolic Blood Pressure (SBP)									
PCS Total	0.07	0.36	[-0.65,0.78]	0.02	.85	0.12	0.34	[-0.55,0.79]	.73
Age (Months)	0.20	0.06	[0.09,0.32]	0.31	<.001*	0.18	0.05	[0.08,0.29]	<.001*
Sex	4.39	2.14	[0.16,8.62]	0.17	.04*	5.29	2.03	[1.30,9.29]	.01*
Rank (Parental)	0.87	2.26	[-3.60,5.34]	0.03	.70	1.68	2.09	[-2.42,5.78]	.42
White (Non-Hispanic)	-1.88	2.06	[-5.95,2.19]	-0.08	.36	-1.90	1.96	[-5.74,1.95]	.33
LOC Eating Past Month	-1.21	2.06	[-5.28,2.87]	-0.05	.56	-1.15	1.91	[-4.88,2.58]	.55
STAI-C	-0.21	0.18	[-0.56,0.15]	-0.10	.25	-0.080	0.17	[-0.42,0.26]	.64
Diastolic Blood Pressure (DBP)									
PCS Total	-0.11	0.28	[-0.67,0.45]	-0.04	.70	-0.06	0.26	[-0.57,0.46]	.82
Age (Months)	0.03	0.05	[-0.06,0.12]	0.06	.49	0.04	0.04	[-0.04,0.12]	.32
Sex	0.84	1.68	[-2.48,4.15]	0.04	.62	1.79	1.56	[-1.27,4.86]	.25
Rank (Parental)	1.84	1.77	[-1.67,5.34]	0.10	.30	1.99	1.63	[-1.21,5.19]	.22
White (Non-Hispanic)	-2.93	1.61	[-6.12,0.26]	-0.16	.07	-2.46	1.55	[-5.50,0.59]	.11
LOC Eating Past Month	-0.10	1.61	[-3.29,3.09]	-0.01	.95	-0.47	1.48	[-3.37,2.43]	.75
STAI-C	0.19	0.14	[-0.09,0.47]	0.12	.18	0.18	0.13	[-0.07,0.44]	.16

Note. *Significant at $ps < .05$; Abbreviations: Rank (Parental) = Warrant Officer/Officer vs. enlisted, Race/Ethnicity = Non-Hispanic White vs. Other, Loss-of-control (LOC) Eating Presence = Endorsed LOC eating at least on month prior; Sex = Male vs. Female, PCS moves = Permanent Change of Station moves, STAI-C = State Trait Anxiety Inventory – Children.

Table 11. Summary of Linear Regression Analyses between PCS and Metabolic Biomarkers

	Observed/Baseline Data					Multiple Imputation Data (Pooled)			
	<i>b</i>	SE(<i>b</i>)	95% CI	β	<i>p</i>	<i>b</i>	SE(<i>b</i>)	95% CI	<i>p</i>
Hemoglobin A1c (HbA1c)									
PCS Total	0.01	0.01	[-0.01,0.03]	.09	.30	0.01	0.01	[-0.01,0.02]	.38
Age (Months)	-0.001	0.001	[-0.004,0.001]	-.08	.35	-0.001	0.001	[-0.004,0.001]	.35
Sex	0.02	0.05	[-0.09,0.12]	.03	.76	0.02	0.05	[-0.09,0.13]	.71
Rank (Parental)	-0.11	0.06	[-0.22,-0.001]	-.18	.048*	-0.12	0.05	[-0.22,-0.02]	.02*
White (Non-Hispanic)	-0.05	0.05	[-0.15,0.05]	-.08	.32	-0.06	0.05	[-0.15,0.04]	.25
LOC Eating Past Month	0.10	0.05	[0.003,0.21]	.17	.04*	0.09	0.05	[-0.01,0.18]	.07
STAI-C	-0.01	0.01	[-0.02,0.002]	-.14	.12	-0.01	0.004	[-0.01,0.003]	.19
High-Density Lipoproteins (HDL)									
PCS Total	-0.85	0.34	[-1.51,-0.18]	-0.21	.01*	-0.64	0.34	[-1.30,0.03]	.06
Age (Months)	-0.03	0.05	[-0.14,0.07]	-0.05	.54	-0.04	0.06	[-0.15,0.08]	.53
Sex	-6.62	2.02	[-10.63,-2.62]	-0.27	.001*	-7.24	2.04	[-11.26,-3.22]	<.001*
Rank (Parental)	8.47	2.11	[4.30,12.63]	0.34	<.001*	7.55	2.31	[2.97,12.12]	.001*
White (Non-Hispanic)	-1.68	1.93	[-5.50,2.14]	-0.07	.39	-1.90	1.96	[-5.74,1.95]	.33
LOC Eating Past Month	1.77	1.93	[-2.05,5.60]	0.07	.36	1.17	1.94	[-2.63,4.98]	.55
STAI-C	-0.13	0.17	[-0.47,0.20]	-0.06	.43	-0.11	0.17	[-0.43,0.22]	.53
Low-Density Lipoproteins (LDL)									
PCS Total	0.30	0.93	[-1.53,2.13]	.03	.74	-0.01	0.91	[-1.78,1.76]	.99
Age (Months)	0.06	0.15	[-0.24,0.35]	.04	.70	0.01	0.15	[-0.28,0.30]	.95
Sex	9.49	5.59	[-1.56,20.54]	.15	.09	7.61	5.52	[-3.26,18.47]	.17
Rank (Parental)	-3.98	5.83	[-15.50,7.55]	-.06	.50	-5.49	5.65	[-16.56,5.58]	.33
White (Non-Hispanic)	-5.00	5.36	[-15.59,5.60]	-.08	.35	-4.52	5.15	[-14.61,5.57]	.38
LOC Eating Past Month	-1.38	5.33	[-11.92,9.16]	-.02	.80	-0.81	5.37	[-11.37,9.74]	.88
STAI-C	-0.47	0.46	[-1.39,0.44]	-.09	.31	-0.22	0.46	[-1.11,0.68]	.64
Triglycerides (TRI)									
PCS Total	1.02	1.35	[-1.64,3.68]	.07	.45	0.55	1.38	[-2.15,3.25]	.69
Age (Months)	0.01	0.21	[-0.41,0.44]	.01	.95	0.00	0.22	[-0.43,0.44]	.999
Sex	18.48	8.06	[2.54,34.42]	.20	.02*	20.37	7.96	[4.77,35.97]	.01*
Rank (Parental)	-6.07	8.39	[-22.67,10.53]	-.07	.47	-8.67	8.37	[-25.10,7.71]	.30
White (Non-Hispanic)	3.34	7.70	[-11.88,18.57]	.04	.67	3.33	8.21	[-12.88,19.53]	.69
LOC Eating Past Month	6.14	7.70	[-9.09,21.37]	.07	.43	8.04	7.78	[-7.21,23.28]	.30
STAI-C	0.18	0.67	[-1.14,1.51]	.02	.79	0.46	0.70	[-0.93,1.84]	.52

Note. *Significant at $ps < .05$; Abbreviations: Rank (Parental) = Warrant Officer/Officer vs. enlisted, Race/Ethnicity = Non-Hispanic White vs. Other, Loss-of-control (LOC) Eating Presence = Endorsed LOC eating at least on month prior; Sex = Male vs. Female, PCS moves = Permanent Change of Station moves, STAI-C = State Trait Anxiety Inventory – Children.

Table 12. Summary of Linear Regression Analyses between PCS and Anthropometric Variables

	Observed/Baseline Data					Multiple Imputation Data (Pooled)			
	<i>b</i>	SE(<i>b</i>)	95% CI	β	<i>p</i>	<i>b</i>	SE(<i>b</i>)	95% CI	<i>p</i>
Body Mass Index Z-Scores (BMIz)									
PCS Total	0.01	0.01	[-0.01,0.03]	.06	.49	0.01	0.01	[-0.01,0.03]	.33
Age (Months)	0.00	0.002	[-0.004,0.003]	-.01	.95	0.00	0.002	[-0.004,0.003]	.89
Sex	0.17	0.07	[0.04,0.30]	.22	.01*	0.19	0.06	[0.07,0.31]	.002*
Rank (Parental)	-0.11	0.07	[-0.24,0.03]	-.14	.13	-0.11	0.07	[-0.25,0.02]	.10
White (Non-Hispanic)	-0.09	0.06	[-0.22,0.03]	-.12	.14	-0.10	0.06	[-0.22,0.03]	.12
LOC Eating Past Month	0.06	0.06	[-0.07,0.18]	.08	.35	0.10	0.06	[-0.02,0.22]	.11
STAI-C	0.004	0.01	[-0.01,0.02]	.06	.50	0.01	0.01	[-0.01,0.02]	.29
Waist Circumference (WC)									
PCS Total	0.14	0.34	[-0.53,0.81]	.04	.68	0.21	0.33	[-0.43,0.85]	.52
Age (Months)	0.17	0.05	[0.07,0.28]	.28	.002*	0.14	0.05	[0.04,0.24]	.01*
Sex	6.40	2.02	[2.40,10.39]	.27	.002*	6.30	1.89	[2.58,10.01]	<.001*
Rank (Parental)	-3.16	2.10	[-7.32,1.00]	-.13	.14	-3.33	2.03	[-7.31,0.67]	.10
White (Non-Hispanic)	-0.75	1.92	[-4.55,3.06]	-.03	.70	-0.56	1.91	[-4.32,3.19]	.77
LOC Eating Past Month	3.09	1.94	[-0.74,6.92]	.13	.11	3.96	1.86	[0.31,7.60]	.03*
STAI-C	0.14	0.17	[-0.20,0.47]	.07	.42	0.21	0.16	[-0.11,0.53]	.19

Note. *Significant at $ps < .05$; Abbreviations: Rank (Parental) = Warrant Officer/Officer vs. enlisted, Race/Ethnicity = Non-Hispanic White vs. Other, Loss-of-control (LOC) Eating Presence = Endorsed LOC eating at least on month prior; Sex = Male vs. Female, PCS Total = Permanent Change of Station move total, STAI-C = State Trait Anxiety Inventory – Children.

Figure 1. Relocation Factors contributing to Military Relocation Stress

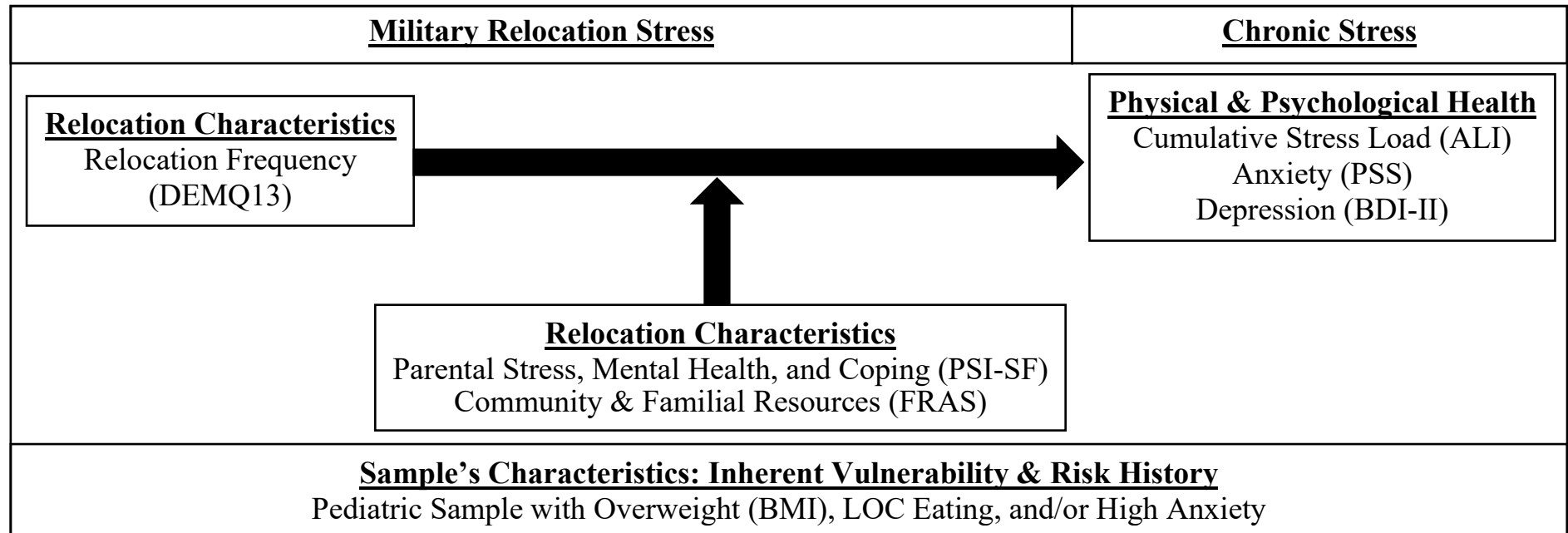


Figure 2. Two Stage Allostatic Model of Central and Peripheral Accommodation

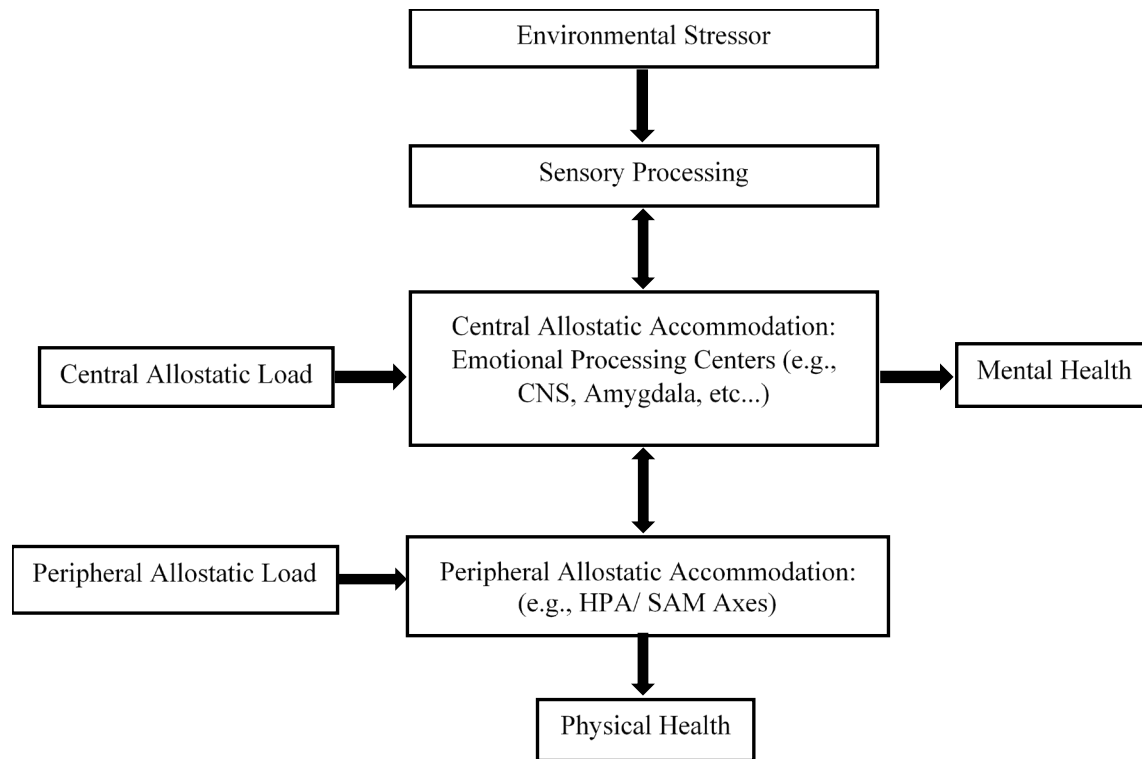


Figure 3. Model of Allostatic Adaptation

Adequate Functioning		Dysregulation		
Normal	Adaptation to Stress	Failed adaptation	Cumulative	Dysregulation
Homeostasis	Allostasis	Allostatic Load	Allostatic Overload	Health Outcomes
	Primary Mediators (e.g., epinephrine, cortisol)	Primary Effects (e.g., anxiety, depression)	Secondary Outcomes (e.g., metabolic dysregulation)	Tertiary outcomes (e.g., hypertension, MetS)

Figure 4. Compensatory and Protective Models of Resilience

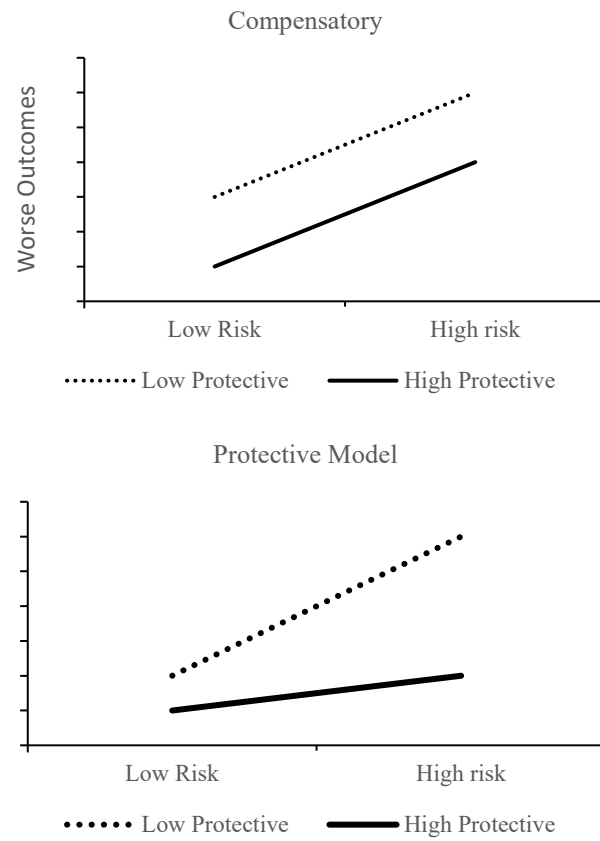
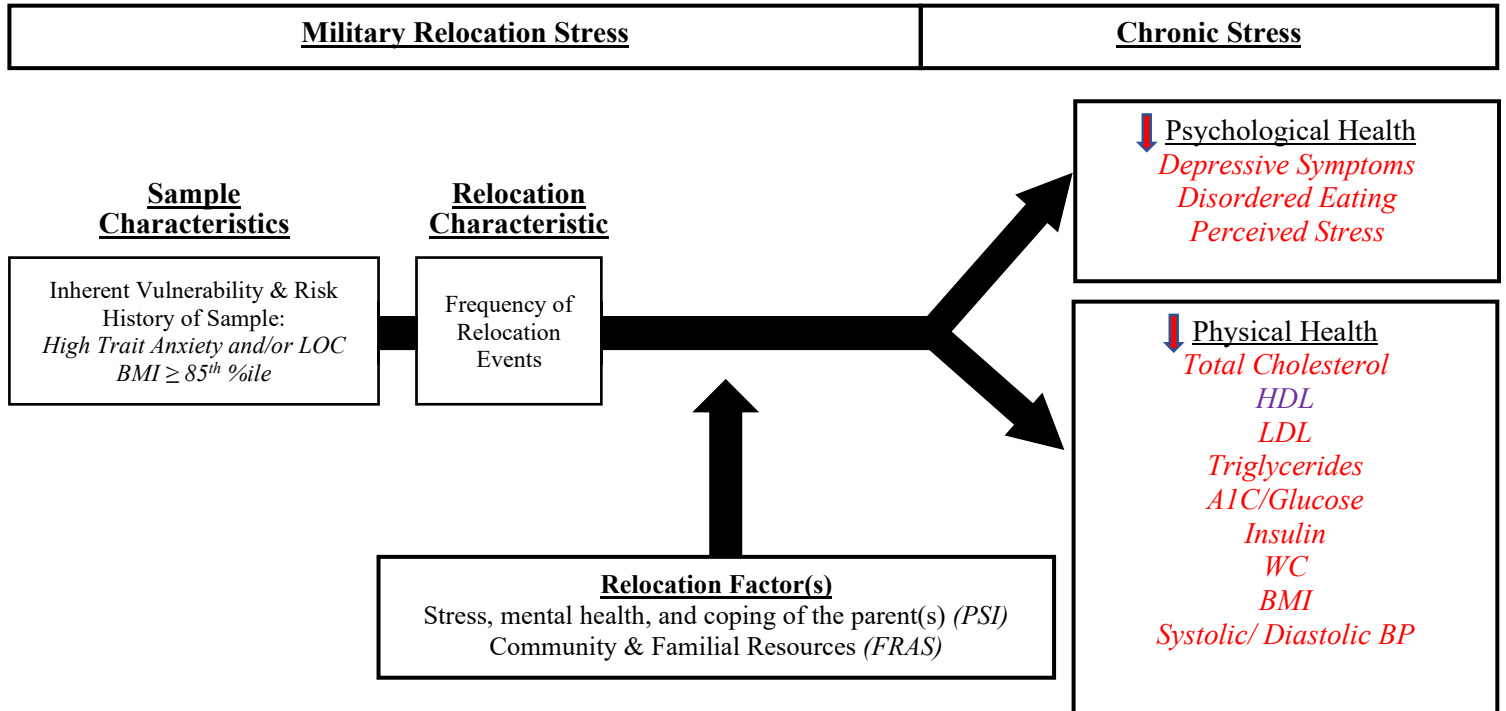


Figure 5. Proposed Relationships in the Military Relocation Stress Model for Aims 1 and 2



References

1. Abidin RR. 1990. *Parenting Stress Index (PSI)*. Pediatric Psychology Press Charlottesville, VA
2. Ackard DM, Neumark-Sztainer D, Story M, Perry C. 2003. Overeating among adolescents: prevalence and associations with weight-related characteristics and psychological health. *Pediatrics* 111:67-74
3. Adamec RE, Blundell J, Burton P. 2005. Neural circuit changes mediating lasting brain and behavioral response to predator stress. *Neurosci Biobehav Rev* 29:1225-41
4. Adameova A, Abdellatif Y, Dhalla NS. 2009. Role of the excessive amounts of circulating catecholamines and glucocorticoids in stress-induced heart disease. *Can J Physiol Pharmacol* 87:493-514
5. Agarwal V, Gupta B, Singhal U, Bajpai SK. 1997. Examination stress: changes in serum cholesterol, triglycerides and total lipids. *Indian J Physiol Pharmacol* 41:404-8
6. Aggleton JP, Young AW. 2000. The enigma of the amygdala: On its contribution to human emotion. In *Cognitive neuroscience of emotion*, pp. 106-28: Oxford University Press
7. Aguilera G, Kiss A, Luo X, Akbasak BS. 1995. The renin angiotensin system and the stress response. *Ann N Y Acad Sci* 771:173-86
8. Ahmed-Leitao F, Spies G, van den Heuvel L, Seedat S. 2016. Hippocampal and amygdala volumes in adults with posttraumatic stress disorder secondary to childhood abuse or maltreatment: A systematic review. *Psychiatry Res Neuroimaging* 256:33-43
9. Ahrens KA, Rossen LM, Simon AE. 2016. Relationship between mean leucocyte telomere length and measures of allostatic load in US reproductive-aged women, NHANES 1999-2002. *Paediatr Perinat Epidemiol* 30:325-35
10. Aina Y, Susman JL. 2006. Understanding comorbidity with depression and anxiety disorders. *J Am Osteopath Assoc* 106:9-14
11. Al-Hamad D, Raman V. 2017. Metabolic syndrome in children and adolescents. *Transl Pediatr* 6:397-407
12. Alvarez GE, Beske SD, Ballard TP, Davy KP. 2002. Sympathetic neural activation in visceral obesity. *Circulation* 106:2533-6
13. Amy NK, Aalborg A, Lyons P, Keranen L. 2006. Barriers to routine gynecological cancer screening for White and African-American obese women. *Int J Obes (Lond)* 30:147-55
14. Anderson S, Leventhal T. 2017. Residential mobility and adolescent achievement and behavior: understanding timing and extent of mobility. *J Res Adolesc* 27:328-43
15. Anderson SE, Cohen P, Naumova EN, Must A. 2006. Association of depression and anxiety disorders with weight change in a prospective community-based study of children followed up into adulthood. *Arch Pediatr Adolesc Med* 160:285-91
16. Andrews RC, Walker BR. 1999. Glucocorticoids and insulin resistance: old hormones, new targets. *Clin Sci (Lond)* 96:513-23
17. Andreyeva T, Puhl RM, Brownell KD. 2008. Changes in perceived weight discrimination among Americans, 1995-1996 through 2004-2006. *Obesity (Silver Spring)* 16:1129-34
18. Angers J, Beland R, Engelbaum M, Williams A, Silah M, Orsega S. 2020. The Joint Travel Regulations. ed. DoD. Alexandria, VA: The Per Diem, Travel, and Transportation Allowance Committee

19. Antczak AJ, Brininger TL. 2008. Diagnosed eating disorders in the U.S. Military: a nine year review. *Eat Disord* 16:363-77
20. APA. 2013. *Diagnostic and statistical manual of mental disorders (DSM-5)*. American Psychiatric Pub
21. Aronson KR, Kyler SJ, Moeller JD, Perkins DF. 2016. Understanding military families who have dependents with special health care and/or educational needs. *Disabil Health J* 9:423-30
22. Arrington EG, Wilson MN. 2000. A re-examination of risk and resilience during adolescence: incorporating culture and diversity. *Journal of Child and Family Studies* 9:221-30
23. Avitsur R, Levy S, Goren N, Grinshpahet R. 2015. Early adversity, immunity and infectious disease. *Stress* 18:289-96
24. Axelson DA, Birmaher B. 2001. Relation between anxiety and depressive disorders in childhood and adolescence. *Depress Anxiety* 14:67-78
25. Bagchi A, Bencio K, Kim J, Lee M, Schone E. 2007. Health care survey of DoD beneficiaries. *Mathematica Policy Research*
26. Bahreinian S, Ball GD, Vander Leek TK, Colman I, McNeil BJ, et al. 2013. Allostatic load biomarkers and asthma in adolescents. *Am J Respir Crit Care Med* 187:144-52
27. Balsevich G, Hausl AS, Meyer CW, Karamihalev S, Feng X, et al. 2017. Stress-responsive FKBP51 regulates AKT2-AS160 signaling and metabolic function. *Nat Commun* 8:1725
28. Barboza Solis C, Kelly-Irving M, Fantin R, Darnaudery M, Torrisani J, et al. 2015. Adverse childhood experiences and physiological wear-and-tear in midlife: Findings from the 1958 British birth cohort. *Proc Natl Acad Sci U S A* 112:738-46
29. Barlas FM, Higgins WB, Pflieger JC, Diecker K. 2013. 2011 health related behaviors survey of active duty military personnel, Department of Defense
30. Barlow DH. 2000. Unraveling the mysteries of anxiety and its disorders from the perspective of emotion theory. *Am Psychol* 55:1247-63
31. Barnes LLB, Harp D, Jung WS. 2016. Reliability generalization of scores on the spielberger state-trait anxiety inventory. *Educational and Psychological Measurement* 62:603-18
32. Beck AT, Steer RA, Brown GK. 1996. Beck depression inventory-II. *San Antonio* 78:490-8
33. Beck AT, Steer RA, Carbin MG. 1988. Psychometric properties of the beck depression inventory: twenty-five years of evaluation. *Clinical Psychology Review* 8:77-100
34. Beckie TM. 2012. A systematic review of allostatic load, health, and health disparities. *Biol Res Nurs* 14:311-46
35. Bennett DA. 2001. How can I deal with missing data in my study? *Aust N Z J Public Health* 25:464-9
36. Berden GF, Althaus M, Verhulst FC. 1990. Major life events and changes in the behavioural functioning of children. *J Child Psychol Psychiatry* 31:949-59
37. Berens AE, Jensen SKG, Nelson CA, 3rd. 2017. Biological embedding of childhood adversity: from physiological mechanisms to clinical implications. *BMC Med* 15:135
38. Berg KF. 2008. Easing transitions of military dependents into Hawaii public schools. *Journal of Invitational Theory and Practice* 14:41-55

39. Bergmann N, Gyntelberg F, Faber J. 2014. The appraisal of chronic stress and the development of the metabolic syndrome: a systematic review of prospective cohort studies. *Endocr Connect* 3:55-80
40. Berthoud HR, Morrison C. 2008. The brain, appetite, and obesity. *Annu Rev Psychol* 59:55-92
41. Binder EB. 2009. The role of FKBP5, a co-chaperone of the glucocorticoid receptor in the pathogenesis and therapy of affective and anxiety disorders. *Psychoneuroendocrinology* 34:186-95
42. Binder EB, Salyakina D, Lichtner P, Wochnik GM, Ising M, et al. 2004. Polymorphisms in FKBP5 are associated with increased recurrence of depressive episodes and rapid response to antidepressant treatment. *Nat Genet* 36:1319-25
43. Bitsko RH, Holbrook JR, Ghandour RM, Blumberg SJ, Visser SN, et al. 2018. Epidemiology and impact of health care provider-diagnosed anxiety and depression among US children. *J Dev Behav Pediatr* 39:395-403
44. Blakely G, Hennessy C, Chung MC, Skirton H. 2012. A systematic review of the impact of foreign postings on accompanying spouses of military personnel. *Nurs Health Sci* 14:121-32
45. Bloch CA, Clemons P, Sperling MA. 1987. Puberty decreases insulin sensitivity. *J Pediatr* 110:481-7
46. Bourdeau I, Bard C, Noel B, Leclerc I, Cordeau MP, et al. 2002. Loss of brain volume in endogenous Cushing's syndrome and its reversibility after correction of hypercortisolism. *J Clin Endocrinol Metab* 87:1949-54
47. Bradshaw CP, Sudhinaraset M, Mmari K, Blum RW. 2010. School transitions among military adolescents: A qualitative study of stress and coping. *School Psychology Review* 39:84-105
48. Brand AH, Johnson JH, Johnson SB. 1986. Life stress and diabetic control in children and adolescents with insulin-dependent diabetes. *J Pediatr Psychol* 11:481-95
49. Bray RM, Pemberton MR, Hourani LL, Witt M, Olmsted KL, et al. 2009. Department of defense survey of health related behaviors among active duty military personnel, RTI International, Research Triangle Park, NC
50. Bray RM, Rae Olmsted KL, Williams J, Sanchez RP, Hartzell M. 2006. Progress toward healthy people 2000 objectives among U.S. military personnel. *Prev Med* 42:390-6
51. Brealey D, Singer M. 2009. Hyperglycemia in critical illness: a review. *J Diabetes Sci Technol* 3:1250-60
52. Breland JY, Donalson R, Nevedal A, Dinh JV, Maguen S. 2017. Military experience can influence womens' eating habits. *Appetite* 118:161-7
53. Buchanan T. 2008. Family resilience as a predictor of better adjustment among international adoptees.
54. Buckwalter J, Rizzo A, John B, Seeman T, Finlay L, et al. 2011. Analyzing the impact of stress: a comparison between a factor analytic and a composite measurement of allostatic load.
55. Buckwalter JG, Castellani B, McEwen B, Karlamangla AS, Rizzo AA, et al. 2016. Allostatic load as a complex clinical construct: a case-based computational modeling approach. *Complexity* 21:291-306

56. Bullock A. 2017. *Military stressors and the well-being of adolescents in Canadian armed forces families: the roles of relationships with parents and peers* Carleton University, Ottawa, Ontario
57. Bureau USC. 2015. Geographical mobility: 2014 to 2015. Washington DC: U.S. Census Bureau
58. Burt Solorzano CM, McCartney CR. 2010. Obesity and the pubertal transition in girls and boys. *Reproduction* 140:399-410
59. Byrne ME, LeMay-Russell S, Tanofsky-Kraff M. 2019. Loss-of-control eating and obesity among children and adolescents. *Curr Obes Rep* 8:33-42
60. Byrne ME, Tanofsky-Kraff M, Kelly NR, Grammer AC, Jaramillo M, et al. 2018. Pediatric Loss-of-Control Eating and Anxiety in Relation to Components of Metabolic Syndrome. *Journal of Pediatric Psychology* 44:220-8
61. Calcaterra V, Vinci F, Casari G, Pelizzo G, de Silvestri A, et al. 2019. Evaluation of allostatic load as a marker of chronic stress in children and the importance of excess weight. *Front Pediatr* 7:335
62. Campbell ND, Appelbaum JC, Martinson K, Martin E. 2000. Be all that we can be: lessons from the military for improving our nation's child care system. Washington D.C.: National Women's Law Center
63. Cardel M, Dulin-Keita A, Casazza K. 2011. Contributors to pediatric obesity in adolescence: more than just energy imbalance. *Open Obes J* 3:17-26
64. Caspi A, Sugden K, Moffitt TE, Taylor A, Craig IW, et al. 2003. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science* 301:386-9
65. Cecchini M, Sassi F, Lauer JA, Lee YY, Guajardo-Barron V, Chisholm D. 2010. Tackling of unhealthy diets, physical inactivity, and obesity: health effects and cost-effectiveness. *Lancet* 376:1775-184
66. Chaplin TM, Gillham JE, Seligman M. 2009. Gender, anxiety, and depressive symptoms. *The Journal of Early Adolescence* 29:307-27
67. Chen L, Xin X, Eckhart AD, Yang N, Faber JE. 1995. Regulation of vascular smooth muscle growth by alpha 1-adrenoreceptor subtypes in vitro and in situ. *J Biol Chem* 270:980-8
68. Chen Y, Baram TZ. 2016. Toward understanding how early-life stress reprograms cognitive and emotional brain networks. *Neuropsychopharmacology* 41:197-206
69. Chew J, Haase AM. 2016. Psychometric properties of the Family Resilience Assessment Scale: A Singaporean perspective. *Epilepsy Behav* 61:112-9
70. Chiodini I, Di Lembo S, Morelli V, Epaminonda P, Coletti F, et al. 2006. Hypothalamic-pituitary-adrenal activity in type 2 diabetes mellitus: role of autonomic imbalance. *Metabolism* 55:1135-40
71. Christian A, Parekh B, Koritzky G. 2020. Bias and discrimination against men with overweight in the military. *Health Psychol Open* 7
72. Chukwura CL, Santo TJ, Waters CN, Andrews A. 2019. 'Nutrition is out of our control': soldiers' perceptions of their local food environment. *Public Health Nutrition* 22:2766-76
73. Cifkova R, Frohlich J, Skodova Z, Lanska V, Adamkova V, et al. 2004. C-REACTIVE PROTEIN AND THE RISK OF DEVELOPING HYPERTENSION. A POPULATION STUDY: 5C.3. *Journal of Hypertension* 22:S149
74. Clark MS, Bond MJ, Hecker JR. 2007. Environmental stress, psychological stress and allostatic load. *Psychol Health Med* 12:18-30

75. Clever M, Segal DR. 2012. After conscription: the united states and the all-volunteer force. *Security and Peace* 30:9-18
76. Clever M, Segal DR. 2013. The demographics of military children and families. *Future Child* 23:13-39
77. Cohen S, Kamarck T, Mermelstein R. 1983. A global measure of perceived stress. *J Health Soc Behav* 24:385-96
78. Cohen S, Kessler RC, Gordon LU. 1995. *Measuring stress: A guide for health and social scientists*. New York, NY, US: Oxford University Press
79. Coker TR, Elliott MN, Wallander JL, Cuccaro P, Grunbaum JA, et al. 2011. Association of family stressful life-change events and health-related quality of life in fifth-grade children. *Arch Pediatr Adolesc Med* 165:354-9
80. Cole DA, Peeke LG, Martin JM, Truglio R, Seroczynski AD. 1998. A longitudinal look at the relation between depression and anxiety in children and adolescents. *J Consult Clin Psychol* 66:451-60
81. Cole DA, Truglio R, Peeke L. 1997. Relation between symptoms of anxiety and depression in children: a multitrait-multimethod-multigroup assessment. *J Consult Clin Psychol* 65:110-9
82. Cole DA, Turner JE, Jr. 1993. Models of cognitive mediation and moderation in child depression. *J Abnorm Psychol* 102:271-81
83. Conforte AM, DeLeon PH, Engel CC, Ling C, Bakalar JL, Tanofsky-Kraff M. 2017. Identifying policy implications and future research directions regarding military community support and child psychosocial adjustment. *Mil Med* 182:1572-80
84. Cooney R, Angelis K, Segal MW. 2011. Moving with the military: race, class, and gender differences in the employment consequences of tied migration. 18:360-84
85. Costello EJ, Mustillo S, Erkanli A, Keeler G, Angold A. 2003. Prevalence and development of psychiatric disorders in childhood and adolescence. *Arch Gen Psychiatry* 60:837-44
86. Cozza SJ, Lerner RM. 2013. Military children and families: introducing the issue. *Future Child* 23:3-11
87. Croll J, Neumark-Sztainer D, Story M, Ireland M. 2002. Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: relationship to gender and ethnicity. *J Adolesc Health* 31:166-75
88. Crow JR, Seybold AK. 2013. Discrepancies in military middle-school adolescents' and parents' perceptions of family functioning, social support, anger frequency, and concerns. *J Adolesc* 36:1-9
89. Cummings CM, Caporino NE, Kendall PC. 2014. Comorbidity of anxiety and depression in children and adolescents: 20 years after. *Psychol Bull* 140:816-45
90. Danese A, McEwen BS. 2012. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol Behav* 106:29-39
91. Danese A, Moffitt TE, Harrington H, Milne BJ, Polanczyk G, et al. 2009. Adverse childhood experiences and adult risk factors for age-related disease: depression, inflammation, and clustering of metabolic risk markers. *Arch Pediatr Adolesc Med* 163:1135-43
92. Danesh J, Wheeler JG, Hirschfield GM, Eda S, Eiriksdottir G, et al. 2004. C-Reactive Protein and Other Circulating Markers of Inflammation in the Prediction of Coronary Heart Disease. *New England Journal of Medicine* 350:1387-97

93. De Pedro KT, Astor RA, Gilreath T, Benbenishty R, Berkowitz R. 2016. Examining the relationship between school climate and peer victimization among students in military-connected public schools. *Violence Vict* 31:751-67
94. De Zwaan M. 2001. Binge eating disorder and obesity. *Int J Obes Relat Metab Disord* 25 Suppl 1:S51-5
95. Dimsdale JE, Herd JA. 1982. Variability of plasma lipids in response to emotional arousal. *Psychosom Med* 44:413-30
96. Dise-Lewis JE. 1988. The life events and coping inventory: an assessment of stress in children. *Psychosomatic medicine* 50:484-99
97. Diz-Chaves Y, Gil-Lozano M, Toba L, Fandino J, Ogando H, et al. 2016. Stressing diabetes? The hidden links between insulinotropic peptides and the HPA axis. *J Endocrinol* 230:77-94
98. DoD. 2012. Department of Defense Instruction. ed. ES Directorate. Washington, D.C.
99. DoD. 2018. 2018 Demographics Report: Profile of the Military Community, Office of the Deputy Assistant Secretary of Defense for Military Community and Family Policy
100. DoD. 2019. Defense Budget Overview, Department of Defense, Washington D.C.
101. Dong M, Anda RF, Felitti VJ, Williamson DF, Dube SR, et al. 2005. Childhood residential mobility and multiple health risks during adolescence and adulthood: the hidden role of adverse childhood experiences. *Arch Pediatr Adolesc Med* 159:1104-10
102. Dong Y, Peng CY. 2013. Principled missing data methods for researchers. *Springerplus* 2:222
103. Doron J, Thomas-Ollivier V, Vachon H, Fortes-Bourbousson M. 2013. Relationships between cognitive coping, self-esteem, anxiety and depression: A cluster-analysis approach. *Personality and Individual Differences* 55:515-20
104. Drotar D. 1997. Relating parent and family functioning to the psychological adjustment of children with chronic health conditions: what have we learned? What do we need to know? *J Pediatr Psychol* 22:149-65
105. Drummet AR, Coleman M, Cable S. 2003. Military families under stress: implications for family life education. *Family Relations* 52:279-87
106. Duchon LM, Weitzman BC, Shinn M. 1999. The relationship of residential instability to medical care utilization among poor mothers in new york city. *Medical Care* 37:1282-93
107. Duncan GE, Li SM, Zhou XH. 2004. Prevalence and trends of a metabolic syndrome phenotype among u.s. Adolescents, 1999-2000. *Diabetes Care* 27:2438-43
108. Dutko P, Ver Ploeg M, Farrigan T. 2012. Characteristics and influential factors of food deserts, USDA
109. Eilerman PA, Herzog CM, Luce BK, Chao SY, Walker SM, et al. 2014. A comparison of obesity prevalence: military health system and United States populations, 2009-2012. *Mil Med* 179:462-70
110. Eisenberg ME, Neumark-Sztainer D, Story M. 2003. Associations of weight-based teasing and emotional well-being among adolescents. *Arch Pediatr Adolesc Med* 157:733-8
111. Elliott CA, Tanofsky-Kraff M, Shomaker LB, Columbo KM, Wolkoff LE, et al. 2010. An examination of the interpersonal model of loss of control eating in children and adolescents. *Behav Res Ther* 48:424-8

112. Enger C, Jones ME, Kryzhanovskaya L, Doherty M, McAfee AT. 2013. Risk of developing diabetes and dyslipidemia among adolescents with bipolar disorder or schizophrenia. *Int J Adolesc Med Health* 25:3-11
113. Epel ES, Crosswell AD, Mayer SE, Prather AA, Slavich GM, et al. 2018. More than a feeling: a unified view of stress measurement for population science. *Front Neuroendocrinol* 49:146-69
114. Essex MJ, Boyce WT, Hertzman C, Lam LL, Armstrong JM, et al. 2013. Epigenetic vestiges of early developmental adversity: childhood stress exposure and DNA methylation in adolescence. *Child Dev* 84:58-75
115. Etheridge R. 1989. Family factors affecting retention: a review of the literature, U.S. Army Research Institute for the Behavioral and Social Sciences
116. Evans GW. 2003. A multimethodological analysis of cumulative risk and allostatic load among rural children. pp. 924-33: American Psychological Association
117. Evans GW, Kim P, Ting AH, Tesher HB, Shannis D. 2007. Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Dev Psychol* 43:341-51
118. Falkner B, Onesti G, Angelakos ET, Fernandes M, Langman C. 1979. Cardiovascular response to mental stress in normal adolescents with hypertensive parents. Hemodynamics and mental stress in adolescents. *Hypertension* 1:23-30
119. Falkner B, Onesti G, Hamstra B. 1981. Stress response characteristics of adolescents with high genetic risk for essential hypertension: a five year follow-up. *Clin Exp Hypertens* 3:583-91
120. Fallon EM, Tanofsky-Kraff M, Norman AC, McDuffie JR, Taylor ED, et al. 2005. Health-related quality of life in overweight and nonoverweight black and white adolescents. *J Pediatr* 147:443-50
121. Families BS. 2019. Blue Star Families 2019 Military Family Survey Results. In *Military Family Lifestyle Survey*. Encinitas, CA
122. Faul F, Erdfelder E, Lang AG, Buchner A. 2007. G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods* 39:175-91
123. Fauvel JP, M'Pio I, Quelin P, Rigaud JP, Laville M, Ducher M. 2003. Neither perceived job stress nor individual cardiovascular reactivity predict high blood pressure. *Hypertension* 42:1112-6
124. Feinstein BA. 2020. The rejection sensitivity model as a framework for understanding sexual minority mental health. *Arch Sex Behav* 49:2247-58
125. Fergus S, Zimmerman MA. 2005. Adolescent resilience: a framework for understanding healthy development in the face of risk. *Annu Rev Public Health* 26:399-419
126. Fergusson DM, Horwood LJ. 2003. Resilience to childhood adversity: Results of a 21-year study. *Resilience and vulnerability: Adaptation in the context of childhood adversities*:130-55
127. Fernandez JR, Redden DT, Pietrobelli A, Allison DB. 2004. Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. *J Pediatr* 145:439-44
128. Finkel LB, Kelley ML, Ashby J. 2003. Geographic mobility, family, and maternal variables as related to the psychosocial adjustment of military children. *Military Medicine* 168:1019-24
129. Fletcher D, Sarkar M. 2013. Psychological resilience. *European Psychologist* 18:12-23

130. Flint SW, Cadek M, Codreanu SC, Ivic V, Zomer C, Gomoiu A. 2016. Obesity discrimination in the recruitment process: "you're not hired!". *Front Psychol* 7:647
131. Ford ES, Li C. 2008. Defining the metabolic syndrome in children and adolescents: will the real definition please stand up? *J Pediatr* 152:160-4
132. Fowler MG, Simpson GA, Schoendorf KC. 1993. Families on the move and children's health care. *Pediatrics* 91:934-40
133. Fox SE, Levitt P, Nelson CA, 3rd. 2010. How the timing and quality of early experiences influence the development of brain architecture. *Child Dev* 81:28-40
134. Francis KT. 1979. Psychologic correlates of serum indicators of stress in man: a longitudinal study. *Psychosom Med* 41:617-28
135. Frayling TM, Timpson NJ, Weedon MN, Zeggini E, Freathy RM, et al. 2007. A common variant in the FTO gene is associated with body mass index and predisposes to childhood and adult obesity. *Science* 316:889-94
136. Frey C, Patil S. 2002. Identification and Review of Sensitivity Analysis Methods. *Risk Analysis* 22:553-78
137. Fried EI, Nesse RM. 2015. Depression sum-scores don't add up: why analyzing specific depression symptoms is essential. *BMC Med* 13:72
138. Friend A, Craig L, Turner S. 2013. The prevalence of metabolic syndrome in children: a systematic review of the literature. *Metab Syndr Relat Disord* 11:71-80
139. Frost DM, Lehavot K, Meyer IH. 2015. Minority stress and physical health among sexual minority individuals. *J Behav Med* 38:1-8
140. Gallo LC, Fortmann AL, Mattei J. 2014. Allostatic load and the assessment of cumulative biological risk in biobehavioral medicine: challenges and opportunities. *Psychosom Med* 76:478-80
141. Ganzel BL, Kim P, Glover GH, Temple E. 2008. Resilience after 9/11: multimodal neuroimaging evidence for stress-related change in the healthy adult brain. *Neuroimage* 40:788-95
142. Ganzel BL, Morris PA, Wethington E. 2010. Allostasis and the human brain: Integrating models of stress from the social and life sciences. *Psychol Rev* 117:134-74
143. Garber J, Weersing VR. 2010. Comorbidity of anxiety and depression in youth: implications for treatment and prevention. *Clin Psychol (New York)* 17:293-306
144. Garboden P, Leventhal T, Newman S. 2017. Estimating the effects of residential mobility: a methodological note. *Journal of Social Service Research* 43:246-61
145. Gardner R, Feely A, Layte R, Williams J, McGavock J. 2019. Adverse childhood experiences are associated with an increased risk of obesity in early adolescence: a population-based prospective cohort study. *Pediatr Res* 86:522-8
146. Gee DG, Casey BJ. 2015. The impact of developmental timing for stress and recovery. *Neurobiol Stress* 1:184-94
147. Gerstein ED, Crnic KA, Blacher J, Baker BL. 2009. Resilience and the course of daily parenting stress in families of young children with intellectual disabilities. *J Intellect Disabil Res* 53:981-97
148. Gilreath TD, Astor RA, Cederbaum JA, Atuel H, Benbenishty R. 2014. Prevalence and correlates of victimization and weapon carrying among military- and nonmilitary-connected youth in Southern California. *Prev Med* 60:21-6
149. Gimeno D, Tabak AG, Ferrie JE, Shipley MJ, De Vogli R, et al. 2010. Justice at work and metabolic syndrome: the Whitehall II study. *Occup Environ Med* 67:256-62

150. Glasofer DR, Tanofsky-Kraff M, Eddy KT, Yanovski SZ, Theim KR, et al. 2007. Binge eating in overweight treatment-seeking adolescents. *J Pediatr Psychol* 32:95-105
151. Godoy LD, Rossignoli MT, Delfino-Pereira P, Garcia-Cairasco N, de Lima Umeoka EH. 2018. A comprehensive overview on stress neurobiology: basic concepts and clinical implications. *Front Behav Neurosci* 12
152. Gold SM, Dziobek I, Rogers K, Bayoumy A, McHugh PF, Convit A. 2005. Hypertension and hypothalamo-pituitary-adrenal axis hyperactivity affect frontal lobe integrity. *J Clin Endocrinol Metab* 90:3262-7
153. Goldstein-Piekarski AN, Korgaonkar MS, Green E, Suppes T, Schatzberg AF, et al. 2016. Human amygdala engagement moderated by early life stress exposure is a biobehavioral target for predicting recovery on antidepressants. *Proc Natl Acad Sci U S A* 113:11955-60
154. Gonzalez VR. 1970. *Psychiatry and the army brat*. Springfield, Ill: Thomas
155. Goodman E, Whitaker RC. 2002. A prospective study of the role of depression in the development and persistence of adolescent obesity. *Pediatrics* 110:497-504
156. Gruenewald TL, Seeman TE, Ryff CD, Karlamangla AS, Singer BH. 2006. Combinations of biomarkers predictive of later life mortality. *Proc Natl Acad Sci U S A* 103:14158-63
157. Guidi J, Lucente M, Sonino N, Fava GA. 2021. Allostatic load and its impact on health: a systematic review. *Psychother Psychosom* 90:11-27
158. Gunnar M, Quevedo K. 2007. The neurobiology of stress and development. *Annu Rev Psychol* 58:145-73
159. Hackett RA, Steptoe A. 2017. Type 2 diabetes mellitus and psychological stress - a modifiable risk factor. *Nat Rev Endocrinol* 13:547-60
160. Hammen C. 2005. Stress and depression. *Annu Rev Clin Psychol* 1:293-319
161. Harkness KL, Bruce AE, Lumley MN. 2006. The role of childhood abuse and neglect in the sensitization to stressful life events in adolescent depression. *J Abnorm Psychol* 115:730-41
162. Haskett ME, Ahern LS, Ward CS, Allaire JC. 2006. Factor structure and validity of the parenting stress index-short form. *J Clin Child Adolesc Psychol* 35:302-12
163. Haveman R, Wolfe B, Spaulding J. 1991. Childhood events and circumstances influencing high school completion. *Demography* 28:133-57
164. He J, Cai Z, Fan X. 2017. Prevalence of binge and loss of control eating among children and adolescents with overweight and obesity: an exploratory meta-analysis. *Int J Eat Disord* 50:91-103
165. Heim C, Binder EB. 2012. Current research trends in early life stress and depression: review of human studies on sensitive periods, gene-environment interactions, and epigenetics. *Exp Neurol* 233:102-11
166. Hendershott AB. 1989. Residential mobility, social support and adolescent self-concept. pp. 217-32. US: Libra Publishers
167. Herpertz-Dahlmann B, Wille N, Holling H, Vloet TD, Ravens-Sieberer U, group Bs. 2008. Disordered eating behaviour and attitudes, associated psychopathology and health-related quality of life: results of the BELLA study. *Eur Child Adolesc Psychiatry* 17 Suppl 1:82-91

168. Herpertz SC, Dietrich TM, Wenning B, Krings T, Erberich SG, et al. 2001. Evidence of abnormal amygdala functioning in borderline personality disorder: a functional MRI study. *Biol Psychiatry* 50:292-8
169. Hertzman C. 2012. Putting the concept of biological embedding in historical perspective. *Proc Natl Acad Sci* 109 Suppl 2:17160-7
170. Heslop P, Smith GD, Metcalfe C, Macleod J, Hart C. 2002. Change in job satisfaction, and its association with self-reported stress, cardiovascular risk factors and mortality. *Soc Sci Med* 54:1589-99
171. Hickman TB, Briefel RR, Carroll MD, Rifkind BM, Cleeman JI, et al. 1998. Distributions and trends of serum lipid levels among United States children and adolescents ages 4-19 years: data from the Third National Health and Nutrition Examination Survey. *Prev Med* 27:879-90
172. Higgins Neyland MK, Shank LM, Burke NL, Schvey NA, Pine A, et al. 2020. Parental deployment and distress, and adolescent disordered eating in prevention-seeking military dependents. *Int J Eat Disord* 53:201-9
173. Higgins Neyland MK, Shank LM, Lavender JM, Rice A, Schindler R, et al. 2021. Permanent change of station moves and disordered-eating attitudes and behaviors in prevention-seeking adolescent military-dependents. *Eat Behav* 40:101470
174. Hilbert A, Hartmann AS, Czaja J, Schoebi D. 2013. Natural course of preadolescent loss of control eating. *J Abnorm Psychol* 122:684-93
175. Hilliard ME, Yi-Frazier JP, Hessler D, Butler AM, Anderson BJ, Jaser S. 2016. Stress and A1c among people with diabetes across the lifespan. *Curr Diab Rep* 16:67
176. Hix WM, Shukiar H, Hanley JM, Kaplan RJ, Kawata JH, et al. 1998. Permanent change of station moves: trends and projections. In *Personnel Turbulence: The Policy Determinants of Permanent Change of Station Moves*. Santa Monica, CA: RAND Corporation
177. Holsboer F. 2000. The corticosteroid receptor hypothesis of depression. *Neuropsychopharmacology* 23:477-501
178. Hormigo S, Vega-Flores G, Castro-Alamancos MA. 2016. Basal Ganglia Output Controls Active Avoidance Behavior. *J Neurosci* 36:10274-84
179. Hotamisligil GS. 2006. Inflammation and metabolic disorders. *Nature* 444:860-7
180. Hutchings HA, Evans A, Barnes P, Demmler JC, Heaven M, et al. 2016. Residential Moving and Preventable Hospitalizations. *Pediatrics* 138
181. Huybrechts I, De Vriendt T, Breidenassel C, Rogiers J, Vanaelst B, et al. 2014. Mechanisms of stress, energy homeostasis and insulin resistance in European adolescents--the HELENA study. *Nutr Metab Cardiovasc Dis* 24:1082-9
182. Ihrke DK, Faber CS. 2012. Geographical mobility: 2005 to 2010, U.S. Census Bureau, Washington, DC.
183. Isnard P, Michel G, Frelut ML, Vila G, Falissard B, et al. 2003. Binge eating and psychopathology in severely obese adolescents. *Int J Eat Disord* 34:235-43
184. Jakobsen JC, Gluud C, Wetterslev J, Winkel P. 2017. When and how should multiple imputation be used for handling missing data in randomised clinical trials – a practical guide with flowcharts. *BMC Medical Research Methodology* 17
185. Jasik CB, Lustig RH. 2008. Adolescent obesity and puberty: the "perfect storm". *Ann N Y Acad Sci* 1135:265-79

186. Jelleyman T, Spencer N. 2008. Residential mobility in childhood and health outcomes: a systematic review. *J Epidemiol Community Health* 62:584-92
187. Jensen PS, Xenakis SN, Wolf P, Bain MW. 1991. The "military family syndrome" revisited: "by the numbers". *J Nerv Ment Dis* 179:102-7
188. Jiang S, Postovit L, Cattaneo A, Binder EB, Aitchison KJ. 2019. Epigenetic modifications in stress response genes associated with childhood trauma. *Front Psychiatry* 10
189. Juster RP, Marin MF, Sindi S, Nair NP, Ng YK, et al. 2011. Allostatic load associations to acute, 3-year and 6-year prospective depressive symptoms in healthy older adults. *Physiol Behav* 104:360-4
190. Juster RP, McEwen BS, Lupien SJ. 2010. Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neurosci Biobehav Rev* 35:2-16
191. Juster RP, Sasseville M, Giguere CE, Consortium S, Lupien SJ. 2018. Elevated allostatic load in individuals presenting at psychiatric emergency services. *J Psychosom Res* 115:101-9
192. Kaplan MS, Nunes A. 2003. The psychosocial determinants of hypertension. *Nutr Metab Cardiovasc Dis* 13:52-9
193. Karlamangla AS, Singer BH, McEwen BS, Rowe JW, Seeman TE. 2002. Allostatic load as a predictor of functional decline. MacArthur studies of successful aging. *J Clin Epidemiol* 55:696-710
194. Kelley ML, Hock E, Jarvis MS, Smith KM, Gaffney MA, Bonney JF. 2009. Psychological adjustment of navy mothers experiencing deployment. *Military Psychology* 14:199-216
195. Kendler KS, Karkowski LM, Corey LA, Neale MC. 1998. Longitudinal population-based twin study of retrospectively reported premenstrual symptoms and lifetime major depression. *Am J Psychiatry* 155:1234-40
196. Kessler RC. 1997. The effects of stressful life events on depression. *Annu Rev Psychol* 48:191-214
197. Kivimaki M, Head J, Ferrie JE, Shipley MJ, Steptoe A, et al. 2007. Hypertension is not the link between job strain and coronary heart disease in the Whitehall II study. *Am J Hypertens* 20:1146-53
198. Kivimaki M, Leino-Arjas P, Luukkonen R, Riihimaki H, Vahtera J, Kirjonen J. 2002. Work stress and risk of cardiovascular mortality: prospective cohort study of industrial employees. *BMJ* 325:857
199. Knorr U, Vinberg M, Kessing LV, Wetterslev J. 2010. Salivary cortisol in depressed patients versus control persons: a systematic review and meta-analysis. *Psychoneuroendocrinology* 35:1275-86
200. Kobrosly RW, van Wijngaarden E, Seplaki CL, Cory-Slechta DA, Moynihan J. 2014. Depressive symptoms are associated with allostatic load among community-dwelling older adults. *Physiol Behav* 123:223-30
201. Koehlmoos TP, Banaag A, Madsen CK, Adirim T. 2020. Child Health As A National Security Issue: Obesity And Behavioral Health Conditions Among Military Children. *Health Aff* 39:1719-27
202. Kraaij V, Kremers I, Arensman E. 1997. The relationship between stressful and traumatic life events and depression in the elderly. *Crisis* 18:86-8

203. Kral TV, Rauh EM. 2010. Eating behaviors of children in the context of their family environment. *Physiol Behav* 100:567-73
204. Kreiser L. 1996. Belonging to the Army: Camp Followers and Community During the American Revolution. *Journal of American Culture* 19:125
205. Kuczmarski RJ, Flegal KM. 2000. Criteria for definition of overweight in transition: background and recommendations for the United States. *Am J Clin Nutr* 72:1074-81
206. Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, et al. 2002. 2000 CDC growth charts for the united states: methods and development. *Vital Health Stat* 11:1-190
207. Kumari M, Head J, Marmot M. 2004. Prospective study of social and other risk factors for incidence of type 2 diabetes in the Whitehall II study. *Arch Intern Med* 164:1873-80
208. Lagrone DM. 1978. The military family syndrome. *Am J Psychiatry* 135:1040-3
209. Lambert EA, Lambert GW. 2011. Stress and its role in sympathetic nervous system activation in hypertension and the metabolic syndrome. *Curr Hypertens Rep* 13:244-8
210. Law E, Fisher E, Eccleston C, Palermo TM. 2019. Psychological interventions for parents of children and adolescents with chronic illness. *Cochrane Database Syst Rev* 3
211. Le Grange D, Loeb KL. 2007. Early identification and treatment of eating disorders: prodrome to syndrome. *Early Interv Psychiatry* 1:27-39
212. Lederer WJ. 1950. *All the ship's at sea*. New York: Sloane
213. Ledesma J. 2014. Conceptual Frameworks and Research Models on Resilience in Leadership. *SAGE Open* 4:215824401454546
214. LeDoux JE. 1996. *The emotional brain: The mysterious underpinnings of emotional life*. New York, NY, US: Simon & Schuster. 384 pp.
215. Lee C, Tsenkova V, Carr D. 2014. Childhood trauma and metabolic syndrome in men and women. *Soc Sci Med* 105:122-30
216. Lee KH, Park SW, Ye SM, Kim SY, Kim SY, et al. 2013. Relationships between dietary habits and allostatic load index in metabolic syndrome patients. *Korean J Fam Med* 34:334-46
217. Lee L-C, Rebok GW. 2002. Anxiety and depression in children: a test of the positive-negative affect model. pp. 419-26. US: Lippincott Williams & Wilkins
218. Lerman LO, Chade AR, Sica V, Napoli C. 2005. Animal models of hypertension: an overview. *J Lab Clin Med* 146:160-73
219. Lester P, Saltzman WR, Woodward K, Glover D, Leskin GA, et al. 2012. Evaluation of a family-centered prevention intervention for military children and families facing wartime deployments. *Am J Public Health* 102 Suppl 1:S48-554
220. Lester P, Stein JA, Saltzman W, Woodward K, MacDermid SW, et al. 2013. Psychological health of military children: longitudinal evaluation of a family-centered prevention program to enhance family resilience. *Mil Med* 178:838-45
221. Lim CS, Espil FM, Viana AG, Janicke DM. 2015. Associations between anxiety symptoms and child and family factors in pediatric obesity. *J Dev Behav Pediatr* 36:664-72
222. Lin K-C, Twisk JWR, Huang H-C. 2012. Longitudinal impact of frequent geographic relocation from adolescence to adulthood on psychosocial stress and vital exhaustion at ages 32 and 42 years: the Amsterdam growth and health longitudinal study. *J Epidemiol* 22:469-76

223. Liu MY, Li N, Li WA, Khan H. 2017. Association between psychosocial stress and hypertension: a systematic review and meta-analysis. *Neurol Res* 39:573-80
224. Liu RT, Alloy LB. 2010. Stress generation in depression: a systematic review of the empirical literature and recommendations for future study. *Clin Psychol Rev* 30:582-93
225. London AS, Heflin CM. 2015. Supplemental nutrition assistance program (SNAP) use among active-duty military personnel, veterans, and reservists. *Population Research and Policy Review* 34:805-26
226. Lovallo WR, Enoch MA, Acheson A, Cohoon AJ, Sorocco KH, et al. 2016. Early-life adversity interacts with FKBP5 genotypes: altered working memory and cardiac stress reactivity in the oklahoma family health patterns project. *Neuropsychopharmacology* 41:1724-32
227. Lucassen EA, Cizza G. 2012. The hypothalamic-pituitary-adrenal axis, obesity, and chronic stress exposure: sleep and the HPA axis in obesity. *Curr Obes Rep* 1:208-15
228. Lupattelli G, Pirro M, Mannarino MR, Siepi D, Roscini AR, et al. 2012. Visceral fat positively correlates with cholesterol synthesis in dyslipidaemic patients. *Eur J Clin Invest* 42:164-70
229. Lupien SJ, Nair NP, Briere S, Maheu F, Tu MT, et al. 1999. Increased cortisol levels and impaired cognition in human aging: implication for depression and dementia in later life. *Rev Neurosci* 10:117-39
230. Luthar SS, Cicchetti D, Becker B. 2000. The construct of resilience: a critical evaluation and guidelines for future work. *Child Dev* 71:543-62
231. Lyons AM, Leon SC, Roecker Phelps CE, Dunleavy AM. 2009. The impact of child symptom severity on stress among parents of children with ASD: the moderating role of coping styles. *Journal of Child and Family Studies* 19:516-24
232. Majd Ara E, Talepasand S, Rezaei AM. 2017. A structural model of depression based on interpersonal relationships: the mediating role of coping strategies and loneliness. *Noro Psikiyatr Ars* 54:125-30
233. Mancina G, Grassi G. 2014. The autonomic nervous system and hypertension. *Circ Res* 114:1804-14
234. Marchant KH, Medway FJ. 1987. Adjustment and achievement associated with mobility in military families. *Psychology in the Schools* 24:289-94
235. Marcovecchio ML, Chiarelli F. 2012. The effects of acute and chronic stress on diabetes control. *Sci Signal* 5
236. Mattei J, Demissie S, Falcon LM, Ordovas JM, Tucker K. 2010. Allostatic load is associated with chronic conditions in the boston puerto rican health study. *Soc Sci Med* 70:1988-96
237. Mauss D, Li J, Schmidt B, Angerer P, Jarczok MN. 2015. Measuring allostatic load in the workforce: a systematic review. *Ind Health* 53:5-20
238. McCaffery JM, Marsland AL, Strohacker K, Muldoon MF, Manuck SB. 2012. Factor structure underlying components of allostatic load. *PLoS One* 7
239. McEwen BS. 2003. Mood disorders and allostatic load. *Biol Psychiatry* 54:200-7
240. McEwen BS. 2008. Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *Eur J Pharmacol* 583:174-85

241. McEwen BS, Seeman T. 1999. Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci* 896:30-47
242. McGonagle KA, Kessler RC. 1990. Chronic stress, acute stress, and depressive symptoms. *Am J Community Psychol* 18:681-706
243. McLeod BD, Wood JJ, Weisz JR. 2007. Examining the association between parenting and childhood anxiety: a meta-analysis. *Clin Psychol Rev* 27:155-72
244. McNulty PA. 2001. Prevalence and contributing factors of eating disorder behaviors in active duty service women in the Army, Navy, Air Force, and Marines. *Mil Med* 166:53-8
245. Meadows SO, Beckett MK, Bowling K, Golinelli D, Fisher MP, et al. 2016. Family resilience in the military: definitions, models, and policies. *Rand Health Q* 5:1-12
246. Medicine Io. 2003. *Weight management: state of the science and opportunities for military programs*. Washington, DC: The National Academies Press. 276 pp.
247. Menke A. 2019. Is the HPA Axis as target for depression outdated, or is there a new hope? *Front Psychiatry* 10:101
248. Meyer IH. 1995. Minority stress and mental health in gay men. *J Health Soc Behav* 36:38-56
249. Meyer IH. 2003. Prejudice, social stress, and mental health in lesbian, gay, and bisexual populations: conceptual issues and research evidence. *Psychol Bull* 129:674-97
250. Micali N, Solmi F, Horton NJ, Crosby RD, Eddy KT, et al. 2015. Adolescent eating disorders predict psychiatric, high-risk behaviors and weight outcomes in young adulthood. *J Am Acad Child Adolesc Psychiatry* 54:652-9
251. Mikolajczak M, Gross JJ, Roskam I. 2019. Parental burnout: what is it, and why does it matter? *Clinical Psychological Science* 7:1319-29
252. Milburn NG, Lightfoot M. 2013. Adolescents in wartime US military families: a developmental perspective on challenges and resources. *Clin Child Fam Psychol Rev* 16:266-77
253. Millegan J, McLay R, Engel C. 2014. The effect of geographic moves on mental healthcare utilization in children. *J Adolesc Health* 55:276-80
254. Miller GE, Chen E, Parker KJ. 2011. Psychological stress in childhood and susceptibility to the chronic diseases of aging: moving toward a model of behavioral and biological mechanisms. *Psychol Bull* 137:959-97
255. Mitchell KS, Porter B, Boyko EJ, Field AE. 2016. Longitudinal associations among posttraumatic stress disorder, disordered eating, and weight gain in military men and women. *Am J Epidemiol* 184:33-47
256. Mitra R, Jadhav S, McEwen BS, Vyas A, Chattarji S. 2005. Stress duration modulates the spatiotemporal patterns of spine formation in the basolateral amygdala. *Proc Natl Acad Sci* 102:9371-6
257. Moberg E, Kollind M, Lins PE, Adamson U. 1994. Acute mental stress impairs insulin sensitivity in IDDM patients. *Diabetologia* 37:247-51
258. Monheit AC, Grafova IB. 2018. Education and family health care spending. *Southern Economic Journal* 85:71-92
259. Moore BA. 2019. Understanding and working within the military culture. In *Treating PTSD in military personnel: A clinical handbook, 2nd ed.*, pp. 9-21: The Guilford Press

260. Morettini A, Schvey NA, Gillmore D, Tanofsky-Kraff M. 2020. Eating disorders and disordered eating in the military family. In *Adapting Evidence-Based Eating Disorder Treatments for Novel Populations and Settings*:194-215. Number of 194-215 pp.
261. Morey RA, Gold AL, LaBar KS, Beall SK, Brown VM, et al. 2012. Amygdala volume changes in posttraumatic stress disorder in a large case-controlled veterans group. *Arch Gen Psychiatry* 69:1169-78
262. Morris T, Manley D, Sabel CE. 2018. Residential mobility: towards progress in mobility health research. *Prog Hum Geogr* 42:112-33
263. Morrison Gutman L, McLoyd VC, Tokoyawa T. 2005. Financial strain, neighborhood stress, parenting behaviors, and adolescent adjustment in urban african american families. *Journal of Research on Adolescence* 15:425-49
264. Moyer AE, Rodin J, Grilo CM, Cummings N, Larson LM, Rebuffe-Scrive M. 1994. Stress-induced cortisol response and fat distribution in women. *Obes Res* 2:255-62
265. Muntner P, He J, Cutler JA, Wildman RP, Whelton PK. 2004. Trends in blood pressure among children and adolescents. *JAMA* 291:2107-13
266. Nagata JM, Garber AK, Tabler JL, Murray SB, Bibbins-Domingo K. 2018. Prevalence and correlates of disordered eating behaviors among young adults with overweight or obesity. *J Gen Intern Med* 33:1337-43
267. Narciso J, Silva AJ, Rodrigues V, Monteiro MJ, Almeida A, et al. 2019. Behavioral, contextual and biological factors associated with obesity during adolescence: A systematic review. *PLoS One* 14:214941
268. Narkiewicz K, Phillips BG, Kato M, Hering D, Bieniaszewski L, Somers VK. 2005. Gender-selective interaction between aging, blood pressure, and sympathetic nerve activity. *Hypertension* 45:522-5
269. Neumark-Sztainer D, Wall M, Guo J, Story M, Haines J, Eisenberg M. 2006. Obesity, disordered eating, and eating disorders in a longitudinal study of adolescents: how do dieters fare 5 years later? *J Am Diet Assoc* 106:559-68
270. Neumark-Sztainer DR, Wall MM, Haines JI, Story MT, Sherwood NE, van den Berg PA. 2007. Shared risk and protective factors for overweight and disordered eating in adolescents. *Am J Prev Med* 33:359-69
271. Niego SH, Pratt EM, Agras WS. 1997. Subjective or objective binge: Is the distinction valid? *International Journal of Eating Disorders* 22:291-8
272. Nieuwenhuizen AG, Rutters F. 2008. The hypothalamic-pituitary-adrenal-axis in the regulation of energy balance. *Physiol Behav* 94:169-77
273. Nolte R, Franckowiak SC, Crespo CJ, Andersen RE. 2002. U.S. military weight standards. *The American Journal of Medicine* 113:486-90
274. Novak M, Bjorck L, Giang KW, Heden-Stahl C, Wilhelmsen L, Rosengren A. 2013. Perceived stress and incidence of type 2 diabetes: a 35-year follow-up study of middle-aged swedish men. *Diabet Med* 30:8-16
275. Office GA. 2001. Longer time between moves related to higher satisfaction and retention, United States General Accounting Office, Washington, D.C.
276. Ogden CL, Carroll MD, Kit BK, Flegal KM. 2012. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999-2010. *JAMA* 307:483-90
277. Oksanen T, Kawachi I, Jokela M, Kouvonen A, Suzuki E, et al. 2012. Workplace social capital and risk of chronic and severe hypertension: a cohort study. *J Hypertens* 30:1129-36

278. Olive LS, Telford RM, Byrne DG, Abhayaratna WP, Telford RD. 2017. Symptoms of stress and depression effect percentage of body fat and insulin resistance in healthy youth: LOOK longitudinal study. *Health Psychol* 36:749-59
279. Orsi CM, Hale DE, Lynch JL. 2011. Pediatric obesity epidemiology. *Current Opinion in Endocrinology, Diabetes and Obesity* 18:14-22
280. Ortiz R, Joseph JJ, Lee R, Wand GS, Golden SH. 2018. Type 2 diabetes and cardiometabolic risk may be associated with increase in DNA methylation of FKBP5. *Clin Epigenetics* 10:82
281. Owen R, Combs T. 2017. Caring for military families: understanding their unique stressors. *Nurse Pract* 42:26-32
282. Pan A, Keum N, Okereke OI, Sun Q, Kivimaki M, et al. 2012. Bidirectional association between depression and metabolic syndrome: a systematic review and meta-analysis of epidemiological studies. *Diabetes Care* 35:1171-80
283. Parhofer KG. 2015. Interaction between glucose and lipid metabolism: more than diabetic dyslipidemia. *Diabetes Metab J* 39:353-62
284. Patterson JM. 2002. Integrating family resilience and family stress theory. *Journal of Marriage and Family* 64:349-60
285. Pearlman AT, Schvey NA, Neyland MK, Solomon S, Hennigan K, et al. 2019. Associations between Family Weight-Based Teasing, Eating Pathology, and Psychosocial Functioning among Adolescent Military Dependents. In *International journal of environmental research and public health*
286. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO, et al. 2003. Markers of Inflammation and Cardiovascular Disease. *Circulation* 107:499-511
287. Pereira CD, Azevedo I, Monteiro R, Martins MJ. 2012. 11beta-hydroxysteroid dehydrogenase type 1: relevance of its modulation in the pathophysiology of obesity, the metabolic syndrome and type 2 diabetes mellitus. *Diabetes Obes Metab* 14:869-81
288. Perreault K, McDuff P, Dion J. 2020. Impact of relocations on mental health and school functioning of adolescents from canadian military families. *Military Behavioral Health* 8:333-44
289. Perry RJ, Resch JM, Douglass AM, Madara JC, Rabin-Court A, et al. 2019. Leptin's hunger-suppressing effects are mediated by the hypothalamic-pituitary-adrenocortical axis in rodents. *Proc Natl Acad Sci U S A* 116:13670-9
290. Pessoa L. 2008. On the relationship between emotion and cognition. *Nat Rev Neurosci* 9:148-58
291. Peterson AL, Talcott GW, Kelleher WJ, Smith SD. 1995. Bulimic weight-loss behaviors in military versus civilian weight-management programs. *Mil Med* 160:616-20
292. Pine DS, Cohen P, Gurley D, Brook J, Ma Y. 1998. The risk for early-adulthood anxiety and depressive disorders in adolescents with anxiety and depressive disorders. *Arch Gen Psychiatry* 55:56-64
293. Piolanti A, Gostoli S, Gervasi J, Sonino N, Guidi J. 2019. A trial integrating different methods to assess psychosocial problems in primary care. *Psychother Psychosom* 88:30-6
294. Plumb J. 2011. THE IMPACT OF SOCIAL SUPPORT AND FAMILY RESILIENCE ON PARENTAL STRESS IN FAMILIES WITH A CHILD DIAGNOSED WITH AN AUTISM SPECTRUM DISORDER.

295. Poljicanin T, Ajdukovic D, Sekerija M, Pibernik-Okanovic M, Metelko Z, Vuletic-Mavrinac G. 2010. Diabetes mellitus and hypertension have comparable adverse effects on health-related quality of life. *10:12*
296. Pouwer F, Kupper N, Adriaanse MC. 2010. Does emotional stress cause type 2 diabetes mellitus? A review from the European Depression in Diabetes (EDID) Research Consortium. *Discov Med* 9:112-8
297. Prpic-Krizevac I, Canecki-Varzic S, Bilic-Curcic I. 2012. Hyperactivity of the hypothalamic-pituitary-adrenal axis in patients with type 2 diabetes and relations with insulin resistance and chronic complications. *Wien Klin Wochenschr* 124:403-11
298. Puhl RM, Brownell KD. 2006. Confronting and coping with weight stigma: an investigation of overweight and obese adults. *Obesity* 14:1802-15
299. Puhl RM, Heuer CA. 2009. The stigma of obesity: a review and update. *Obesity* 17:941-64
300. Puhl RM, Heuer CA. 2010. Obesity stigma: important considerations for public health. *Am J Public Health* 100:1019-28
301. Pulgaron ER. 2013. Childhood obesity: a review of increased risk for physical and psychological comorbidities. *Clin Ther* 35:18-32
302. Puustinen PJ, Koponen H, Kautiainen H, Mantyselka P, Vanhala M. 2011. Psychological distress predicts the development of the metabolic syndrome: a prospective population-based study. *Psychosom Med* 73:158-65
303. Quattlebaum M, Burke NL, Higgins Neyland MK, Leu W, Schvey NA, et al. 2019. Sex differences in eating related behaviors and psychopathology among adolescent military dependents at risk for adult obesity and eating disorders. *Eat Behav* 33:73-7
304. Quek YH, Tam W, Zhang M, Ho R. 2017. Exploring the association between childhood and adolescent obesity and depression: a meta-analysis. *Obes Rev* 18:742-54
305. Radin RM, Tanofsky-Kraff M, Shomaker LB, Kelly NR, Pickworth CK, et al. 2015. Metabolic characteristics of youth with loss of control eating. *Eat Behav* 19:86-9
306. Ranzenhofer LM, Engel SG, Crosby RD, Anderson M, Vannucci A, et al. 2014. Using ecological momentary assessment to examine interpersonal and affective predictors of loss of control eating in adolescent girls. *Int J Eat Disord* 47:748-57
307. Ranzenhofer LM, Hannallah L, Field SE, Shomaker LB, Stephens M, et al. 2013. Pre-meal affective state and laboratory test meal intake in adolescent girls with loss of control eating. *Appetite* 68:30-7
308. Raz N, Rodrigue KM, Acker JD. 2003. Hypertension and the brain: vulnerability of the prefrontal regions and executive functions. *Behav Neurosci* 117:1169-80
309. Reckelhoff JF. 2001. Gender differences in the regulation of blood pressure. *Hypertension* 37:1199-208
310. Reinehr T, de Sousa G, Toschke AM, Andler W. 2007. Comparison of metabolic syndrome prevalence using eight different definitions: a critical approach. *Archives of disease in childhood* 92:1067-72
311. Reitman D, Currier RO, Stickle TR. 2002. A critical evaluation of the Parenting Stress Index-Short Form (PSI-SF) in a head start population. *J Clin Child Adolesc Psychol* 31:384-92
312. Reynaud E, Guedj E, Trousselard M, El Khoury-Malhame M, Zendjidjian X, et al. 2015. Acute stress disorder modifies cerebral activity of amygdala and prefrontal cortex. *Cogn Neurosci* 6:39-43

313. Richardson AS, Nicosia N, Ghosh-Dastidar MB, Datar A. 2020. School food and beverage availability and children's diet, purchasing, and obesity: evidence from a natural experiment. *J Adolesc Health* 67:804-13
314. Richardson EW, Mallette JK, O'Neal CW, Mancini JA. 2016. Do youth development programs matter? an examination of transitions and well-being among military youth. *Journal of Child and Family Studies* 25:1765-76
315. Riley WJ. 2012. Health disparities: gaps in access, quality and affordability of medical care. *Trans Am Clin Climatol Assoc* 123:167-72
316. Rippe JK. 2012. The impact of low, moderate, and high military family mobility school district transfer rates on graduating senior high school dependents' achievement and school engagement. *Student Work* 42
317. Roccella E. 1996. Update on the 1987 task force report on high blood pressure in children and adolescents: A working group report from the national high blood pressure education program. *Pediatrics* 98:649-58
318. Rod NH, Gronbaek M, Schnohr P, Prescott E, Kristensen TS. 2009. Perceived stress as a risk factor for changes in health behaviour and cardiac risk profile: a longitudinal study. *J Intern Med* 266:467-75
319. Rofey DL, Kolko RP, Iosif AM, Silk JS, Bost JE, et al. 2009. A longitudinal study of childhood depression and anxiety in relation to weight gain. *Child Psychiatry Hum Dev* 40:517-26
320. Rogosch FA, Dackis MN, Cicchetti D. 2011. Child maltreatment and allostatic load: consequences for physical and mental health in children from low-income families. *Dev Psychopathol* 23:1107-24
321. Romeo RD, McEwen BS. 2006. Stress and the adolescent brain. *Ann N Y Acad Sci* 1094:202-14
322. Rosen JB, Schulkin J. 2004. Adaptive fear, allostasis, and the pathology of anxiety and depression. In *Allostasis, homeostasis, and the costs of physiological adaptation.*, pp. 164-227. New York, NY, US: Cambridge University Press
323. Roth TL, Sweatt JD. 2011. Annual research review: epigenetic mechanisms and environmental shaping of the brain during sensitive periods of development. *J Child Psychol Psychiatry* 52:398-408
324. Rubin D. 1976. Inference and missing data. *Biometrika* 63:581-92
325. Rubin DB. 1996. Multiple Imputation After 18+ Years. *Journal of the American Statistical Association* 91:473
326. Russo TJ, Fallon MA. 2014. Coping with stress: supporting the needs of military families and their children. *Early Childhood Education Journal* 43:407-16
327. Saab PG, Llabre MM, Ma M, DiLillo V, McCalla JR, et al. 2001. Cardiovascular responsivity to stress in adolescents with and without persistently elevated blood pressure. *Journal of Hypertension* 19
328. Saaddine JB, Fagot-Campagna A, Rolka D, Narayan KM, Geiss L, et al. 2002. Distribution of HbA(1c) levels for children and young adults in the U.S.: Third National Health and Nutrition Examination Survey. *Diabetes Care* 25:1326-30
329. Sadacca R, McCloy RA, DiFazio AS. 1993. The impact of army and family factors on individual readiness, Army Research Institute, Alexandria, VA
330. Saklayen MG. 2018. The global epidemic of the metabolic syndrome. *Curr Hypertens Rep* 20:12

331. Salleh MR. 2008. Life event, stress and illness. *Malays J Med Sci* 15:9-18
332. Saltzman WR. 2016. The FOCUS family resilience program: an innovative family intervention for trauma and loss. *Fam Process* 55:647-59
333. Saltzman WR, Lester P, Beardslee WR, Layne CM, Woodward K, Nash WP. 2011. Mechanisms of risk and resilience in military families: theoretical and empirical basis of a family-focused resilience enhancement program. *Clin Child Fam Psychol Rev* 14:213-30
334. Sanderson PW, Clemes SA, Biddle SJ. 2011. The correlates and treatment of obesity in military populations: a systematic review. *Obes Facts* 4:229-37
335. Sanghez V, Razzoli M, Carobbio S, Campbell M, McCallum J, et al. 2013. Psychosocial stress induces hyperphagia and exacerbates diet-induced insulin resistance and the manifestations of the Metabolic Syndrome. *Psychoneuroendocrinology* 38:2933-42
336. Santos-Lozada AR, Howard JT. 2018. Using allostatic load to validate self-rated health for racial/ethnic groups in the united states. *Biodemography Soc Biol* 64:1-14
337. Sapolsky RM. 2004. *Why zebras don't get ulcers: the acclaimed guide to stress, stress-related diseases, and coping*. Henry Holt and Company
338. Sarwer DB, Polonsky HM. 2016. The psychosocial burden of obesity. *Endocrinol Metab Clin North Am* 45:677-88
339. Sayers SL, Farrow VA, Ross J, Oslin DW. 2009. Family problems among recently returned military veterans referred for a mental health evaluation. *J Clin Psychiatry* 70:163-70
340. Scanlon E, Devine K. 2001. Residential mobility and youth well-being: research, policy and practice issues. *Journal of Sociology and Social Welfare* 28:119-38
341. Schlotz W, Yim IS, Zoccola PM, Jansen L, Schulz P. 2011. The perceived stress reactivity scale: Measurement invariance, stability, and validity in three countries. *Psychological Assessment* 23:80-94
342. Schlund MW, Cataldo MF. 2010. Amygdala involvement in human avoidance, escape and approach behavior. *Neuroimage* 53:769-76
343. Schluter N, Schmidt R, Kittel R, Tetzlaff A, Hilbert A. 2016. Loss of control eating in adolescents from the community. *Int J Eat Disord* 49:413-20
344. Schumm W, Bell BD, Tran G. 1994. Family adaptation to the demands of army life: a reivew of findings, Army Research Instititue, Alexandria, VA
345. Schvey NA, Barmine M, Bates D, Oldham K, Bakalar JL, et al. 2017. Weight stigma among active duty U.S. military personnel with overweight and obesity. *Stigma and Health* 2:281-91
346. Schvey NA, Klein DA, Pearlman AT, Riggs DS. 2020. A descriptive study of transgender active duty service members in the U.S. military. *Transgend Health* 5:149-57
347. Schvey NA, Marwitz SE, Mi SJ, Galescu OA, Broadney MM, et al. 2019. Weight-based teasing is associated with gain in BMI and fat mass among children and adolescents at-risk for obesity: A longitudinal study. *Pediatr Obes* 14:12538
348. Schvey NA, Sbrocco T, Stephens M, Bryant EJ, Ress R, et al. 2015. Comparison of overweight and obese military-dependent and civilian adolescent girls with loss-of-control eating. *Int J Eat Disord* 48:790-4
349. Sciences NAO. 2019. Strengthening the Military Family Readiness System for a Changing American Society, National Academies Press, Washington, DC

350. Seeman T, Gleit D, Goldman N, Weinstein M, Singer B, Lin YH. 2004. Social relationships and allostatic load in taiwanese elderly and near elderly. *Soc Sci Med* 59:2245-57
351. Seeman TE, Crimmins E, Huang MH, Singer B, Bucur A, et al. 2004. Cumulative biological risk and socio-economic differences in mortality: MacArthur studies of successful aging. *Soc Sci Med* 58:1985-97
352. Seeman TE, McEwen BS, Singer BH, Albert MS, Rowe JW. 1997. Increase in urinary cortisol excretion and memory declines: MacArthur studies of successful aging. *J Clin Endocrinol Metab* 82:2458-65
353. Segal M. 1986. The Military And the Family As Greedy Institutions. *Armed Forces & Society* 13:9-38
354. Seidell JC, Cigolini M, Charzewska J, Ellsinger BM, Björntorp P, et al. 1991. Fat distribution and gender differences in serum lipids in men and women from four European communities. *Atherosclerosis* 87:203-10
355. Selye H. 1956. *The stress of life*. New York, NY, US: McGraw-Hill. 324 pp.
356. Shank LM, Crosby RD, Grammer AC, Shomaker LB, Vannucci A, et al. 2017. Examination of the interpersonal model of loss of control eating in the laboratory. *Compr Psychiatry* 76:36-44
357. Shank LM, Schvey NA, Ekundayo K, Schreiber-Gregory D, Bates D, et al. 2019. The relationship between weight stigma, weight bias internalization, and physical health in military personnel with or at high-risk of overweight/obesity. *Body Image* 28:25-33
358. Shank LM, Tanofsky-Kraff M, Kelly NR, Schvey NA, Marwitz SE, et al. 2017. Pediatric Loss of Control Eating and High-Sensitivity C-Reactive Protein Concentrations. *Child Obes* 13:1-8
359. Shank LM, Tanofsky-Kraff M, Radin RM, Shomaker LB, Wilfley DE, et al. 2018. Remission of loss of control eating and changes in components of the metabolic syndrome. *International Journal of Eating Disorders* 51:565-73
360. Sharma AN, Wigham J, Veldhuis JD. 2014. Corticotrophic axis drive of overnight cortisol secretion is suppressed in adolescents and young adults with type 1 diabetes mellitus. *Pediatr Diabetes* 15:444-52
361. Shih JH, Eberhart NK. 2008. Understanding the impact of prior depression on stress generation: examining the roles of current depressive symptoms and interpersonal behaviours. *Br J Psychol* 99:413-26
362. Shinseki EK. 2003. The Army Family. *Rep. 70-84-1*, United States Army
363. Shomaker LB, Tanofsky-Kraff M, Elliott C, Wolkoff LE, Columbo KM, et al. 2010. Salience of loss of control for pediatric binge episodes: does size really matter? *Int J Eat Disord* 43:707-16
364. Silva Í, Cunha K, Ramos E, Pontes F, Silva S. 2020. Family resilience and parenting stress in poor families. *Estudos de Psicologia (Campinas)* 38
365. Simpson GA, Fowler MG. 1994. Geographic mobility and children's emotional/behavioral adjustment and school functioning. *Pediatrics* 93:303-9
366. Singh A, Singh S, Singh N, Agrawal N, Gopal K. 2011. Obesity and dyslipidemia. *Int J Biol Med Res* 2:824-8
367. Singh JP, Larson MG, Tsuji H, Evans JC, O'Donnell CJ, Levy D. 1998. Reduced heart rate variability and new-onset hypertension: insights into pathogenesis of hypertension: the Framingham Heart Study. *Hypertension* 32:293-7

368. Sixbey MT. 2005. *Development of the family resilience assessment scale to identify family resilience constructs*. University of Florida
369. Smith C, Klosterbuer A, Levine AS. 2009. Military experience strongly influences post-service eating behavior and BMI status in American veterans. *Appetite* 52:280-9
370. Smith MM, Minson CT. 2012. Obesity and adipokines: effects on sympathetic overactivity. *J Physiol* 590:1787-801
371. Smith SM, Vale WW. 2006. The role of the hypothalamic-pituitary-adrenal axis in neuroendocrine responses to stress. *Dialogues Clin Neurosci* 8:383-95
372. Smrekar CE, Owens DE. 2003. "It's a way of life for us": high mobility and high achievement in department of defense schools. *The Journal of Negro Education* 72:165-77
373. Sonnevile KR, Horton NJ, Micali N, Crosby RD, Swanson SA, et al. 2013. Longitudinal associations between binge eating and overeating and adverse outcomes among adolescents and young adults: does loss of control matter? *JAMA Pediatr* 167:149-55
374. Spielberger CD, Gorsuch RL, Lushene R, Vagg PR, Jacobs GA. 1983. *Manual for the state-trait anxiety inventory*. Palo Alto, CA: Consulting Psychologists Press
375. Spoehr T, Handy B. 2018. The looming national security crisis: young Americans unable to serve in the military. *Backgrounder* 3282
376. Stanley J, Segal MW, Laughton CJ. 1990. Grass roots family action and military policy responses. *Marriage & Family Review* 15:207-23
377. Stark KD, Laurent J. 2001. Joint factor analysis of the children's depression inventory and the revised children's manifest anxiety scale. *J Clin Child Psychol* 30:552-67
378. Steinberg L, Morris AS. 2001. Adolescent development. *Annu Rev Psychol* 52:83-110
379. Steinberg N, Nemet D, Pantanowitz M, Eliakim A. 2018. Gait Pattern, Impact to the Skeleton and Postural Balance in Overweight and Obese Children: A Review. *Sports (Basel)* 6:75
380. Steinberger J, Kelly AS. 2008. Challenges of existing pediatric dyslipidemia guidelines: call for reappraisal. *Circulation* 117:9-10
381. Steinberger J, Moran A, Hong CP, Jacobs DR, Jr., Sinaiko AR. 2001. Adiposity in childhood predicts obesity and insulin resistance in young adulthood. *J Pediatr* 138:469-73
382. Sterling P, Eyer J. 1988. Allostasis: A new paradigm to explain arousal pathology. In *Handbook of life stress, cognition and health*:629-49. Oxford, England: John Wiley & Sons. Number of 629-49 pp.
383. Strobino J, Salvaterra M. 2000. School Transitions among Adolescent Children of Military Personnel: A Strengths Perspective. *Children & Schools* 22:95-107
384. Sumner J, Boisvert D, Andersen JP. 2015. The effects of stress and social support on externalizing behaviors among children in military families. *Deviant Behavior* 37:246-62
385. Survey MFL. 2018. Summary of Trend & Resulting Impact Report 2009-2018, Blue Star Families, Encinitas, CA
386. Surwit RS, Schneider MS, Feinglos MN. 1992. Stress and diabetes mellitus. *Diabetes Care* 15:1413-22
387. Sutin AR, Stephan Y, Terracciano A. 2015. Weight discrimination and risk of mortality. *Psychol Sci* 26:1803-11
388. Swaab DF, Bao AM, Lucassen PJ. 2005. The stress system in the human brain in depression and neurodegeneration. *Ageing Res Rev* 4:141-94

389. Swanson SA, Crow SJ, Le Grange D, Swendsen J, Merikangas KR. 2011. Prevalence and correlates of eating disorders in adolescents. Results from the national comorbidity survey replication adolescent supplement. *Arch Gen Psychiatry* 68:714-23
390. Szyf M, Weaver IC, Champagne FA, Diorio J, Meaney MJ. 2005. Maternal programming of steroid receptor expression and phenotype through DNA methylation in the rat. *Front Neuroendocrinol* 26:139-62
391. Tabachnick BG, Fidell LS. 2013. *Using multivariate statistics*. Pearson Education
392. Tamashiro KL, Sakai RR, Shively CA, Karatsoreos IN, Reagan LP. 2011. Chronic stress, metabolism, and metabolic syndrome. *Stress* 14:468-74
393. Tanofsky-Kraff M. 2009. Binge Eating Among Children and Adolescents. In *Handbook of Childhood and Adolescent Obesity*, ed. E Jelalian, RG Steele:43-59. Boston, MA: Springer US. Number of 43-59 pp.
394. Tanofsky-Kraff M, Sbrocco T, Theim KR, Cohen LA, Mackey ER, et al. 2013. Obesity and the US military family. *Obesity* 21:2205-20
395. Tanofsky-Kraff M, Shomaker LB, Olsen C, Roza CA, Wolkoff LE, et al. 2011. A prospective study of pediatric loss of control eating and psychological outcomes. *J Abnorm Psychol* 120:108-18
396. Tanofsky-Kraff M, Wilfley DE, Young JF, Mufson L, Yanovski SZ, et al. 2007. Preventing excessive weight gain in adolescents: interpersonal psychotherapy for binge eating. *Obesity* 15:1345-55
397. Tanofsky-Kraff M, Wilfley DE, Young JF, Mufson L, Yanovski SZ, et al. 2007. Preventing Excessive Weight Gain in Adolescents: Interpersonal Psychotherapy for Binge Eating*. *Obesity* 15:1345-55
398. Tanofsky-Kraff M, Yanovski SZ, Schvey NA, Olsen CH, Gustafson J, Yanovski JA. 2009. A prospective study of loss of control eating for body weight gain in children at high risk for adult obesity. *Int J Eat Disord* 42:26-30
399. Tenk J, Matrai P, Hegyi P, Rostas I, Garami A, et al. 2018. Perceived stress correlates with visceral obesity and lipid parameters of the metabolic syndrome: A systematic review and meta-analysis. *Psychoneuroendocrinology* 95:63-73
400. Tennant C. 2002. Life events, stress and depression: a review of recent findings. *Aust N Z J Psychiatry* 36:173-82
401. Thaker VV, Osganian SK, deFerranti SD, Sonnevile KR, Cheng JK, et al. 2020. Psychosocial, behavioral and clinical correlates of children with overweight and obesity. *BMC Pediatrics* 20:291
402. Thase ME, Jindal R, Howland RH. 2002. *Biological aspects of depression*. New York, NY, US: The Guilford Press. 192-218 pp.
403. Thompson M. 2016. Here's Why the US Military Is a Family Business. *Time Magazine*. Retrieved from <http://time.com/4254696/military-family-business>
404. Tilling K, Williamson EJ, Spratt M, Sterne JAC, Carpenter JR. 2016. Appropriate inclusion of interactions was needed to avoid bias in multiple imputation. *Journal of Clinical Epidemiology* 80:107-15
405. Tomba E, Offidani E. 2012. A clinimetric evaluation of allostatic overload in the general population. *Psychother Psychosom* 81:378-9
406. Tomeleri CM, Ronque ER, Silva DR, Cardoso Junior CG, Fernandes RA, et al. 2015. Prevalence of dyslipidemia in adolescents: comparison between definitions. *Rev Port Cardiol* 34:103-9

407. Tomiyama AJ. 2019. Stress and Obesity. *Annu Rev Psychol* 70:703-18
408. Tong PK, Payne LA, Bond CA, Meadows SO, Lewis JL, et al. 2018. Enhancing family stability during a permanent change of station: a review of disruptions and policies, RAND Corporation, Santa Monica, CA
409. Torres SJ, Nowson CA. 2007. Relationship between stress, eating behavior, and obesity. *Nutrition* 23:887-94
410. Tozzi L, Farrell C, Booij L, Doolin K, Nemoda Z, et al. 2018. Epigenetic Changes of FKBP5 as a Link Connecting Genetic and Environmental Risk Factors with Structural and Functional Brain Changes in Major Depression. *Neuropsychopharmacology* 43:1138-45
411. Tu W, Eckert GJ, Saha C, Pratt JH. 2009. Synchronization of adolescent blood pressure and pubertal somatic growth. *J Clin Endocrinol Metab* 94:5019-22
412. Tunstall H, Cabieses B, Shaw R. 2012. The characteristics of mobile families with young children in England and the impact of their moves on neighbourhood inequalities in maternal and child health. *Health Place* 18:657-70
413. van Dijk G, Buwalda B. 2008. Neurobiology of the metabolic syndrome: an allostatic perspective. *Eur J Pharmacol* 585:137-46
414. van Reedt Dortland AK, Giltay EJ, van Veen T, van Pelt J, Zitman FG, Penninx BW. 2010. Associations between serum lipids and major depressive disorder: results from the Netherlands Study of Depression and Anxiety (NESDA). *J Clin Psychiatry* 71:729-36
415. Vanaelst B, Huybrechts I, De Bourdeaudhuij I, Bammann K, Hadjigeorgiou C, et al. 2012. Prevalence of negative life events and chronic adversities in European pre- and primary-school children: results from the IDEFICS study. *Arch Public Health* 70:26
416. Vencill JA, Tebbe EA, Garos S. 2015. It's not the size of the boat or the motion of the ocean: the role of self-objectification, appearance anxiety, and depression in female sexual functioning. *Psychology of Women Quarterly* 39:471-83
417. Vernez G, Zellman GL. 1987. Families and mission: a review of the effects of family factors on army attrition, retention, and readiness. *Rep. MDA93-86-C-0059*, RAND Corporation, Santa Monica, CA
418. Verster JC, Sandalova E, Garssen J, Bruce G. 2021. The Use of Single-Item Ratings Versus Traditional Multiple-Item Questionnaires to Assess Mood and Health. *European Journal of Investigation in Health, Psychology and Education* 11:183-98
419. Vgontzas AN, Pejovic S, Zoumakis E, Lin HM, Bentley CM, et al. 2007. Hypothalamic-pituitary-adrenal axis activity in obese men with and without sleep apnea: effects of continuous positive airway pressure therapy. *J Clin Endocrinol Metab* 92:4199-207
420. Vindas MA, Helland-Riise SH, Nilsson GE, Øverli Ø. 2019. Depression-like state behavioural outputs may confer beneficial outcomes in risky environments. *Scientific Reports* 9
421. Vining M, Hacker BC. 2001. From camp follower to lady in uniform: women, social class and military institutions before 1920. *Contemporary European History* 10:353-73
422. Waasdorp CE, Caboot JB, Robinson CA, Abraham AA, Adelman WP. 2007. Screening military dependent adolescent females for disordered eating. *Mil Med* 172:962-7
423. Wagner IV, Sabin MA, Pfäffle RW, Hiemisch A, Sergeyev E, et al. 2012. Effects of obesity on human sexual development. *Nat Rev Endocrinol* 8:246-54

424. Waldstein SR, Manuck SB, Ryan CM, Muldoon MF. 1991. Neuropsychological correlates of hypertension: review and methodologic considerations. *Psychol Bull* 110:451-68
425. Walsh F. 2006. *Strengthening family resilience*. New York, NY, US: The Guilford Press. 384 pp.
426. Wang J, Johnson DE. 2019. An Examination of Discrepancies in Multiple Imputation Procedures Between SAS® and SPSS®. *The American Statistician* 73:80-8
427. Wang Q, Verweij EW, Krugers HJ, Joels M, Swaab DF, Lucassen PJ. 2014. Distribution of the glucocorticoid receptor in the human amygdala; changes in mood disorder patients. *Brain Struct Funct* 219:1615-26
428. Wardle J, Chida Y, Gibson EL, Whitaker KL, Steptoe A. 2011. Stress and adiposity: a meta-analysis of longitudinal studies. *Obesity* 19:771-8
429. Warner C, Warner C, Matuszak T, Rachal J, Flynn J, Grieger TA. 2007. Disordered eating in entry-level military personnel. *Mil Med* 172:147-51
430. Watanabe HK, Jensen PS, Newby J, Cortes RM. 1995. The exceptional family member program: perceptions of active duty enrollees. *Mil Med* 160:639-43
431. Webb RT, Pedersen CB, Mok PLH. 2016. Adverse Outcomes to Early Middle Age Linked With Childhood Residential Mobility. *Am J Prev Med* 51:291-300
432. Weber EG, Weber DK. 2005. Geographic relocation frequency, resilience, and military adolescent behavior. *Mil Med* 170:638-42
433. Weber EG, Weber DK. 2005. Geographic Relocation Frequency, Resilience, and Military Adolescent Behavior. *Military Medicine* 170:638-42
434. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, et al. 2004. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med* 350:2362-74
435. Wickham JA. 1983. The Army Family, Department of the Army
436. Wilcox G. 2005. Insulin and insulin resistance. *Clin Biochem Rev* 26:19-39
437. Wiley JF, Gruenewald TL, Karlamangla AS, Seeman TE. 2016. Modeling Multisystem Physiological Dysregulation. *Psychosom Med* 78:290-301
438. Williams VF, Stahlman S, Taubman SB. 2018. Diagnoses of eating disorders, active component service members, U.S. armed forces, 2013–2017, Washington, D.C.
439. Williamson V, Stevelink SAM, Da Silva E, Fear NT. 2018. A systematic review of wellbeing in children: a comparison of military and civilian families. *Child Adolesc Psychiatry Ment Health* 12:46
440. Wills TA, Vaughan R. 1989. Social support and substance use in early adolescence. *J Behav Med* 12:321-39
441. Wood DL, Sheps SG, Elveback LR, Schirger A. 1984. Cold pressor test as a predictor of hypertension. *Hypertension* 6:301-6
442. Wright BJ, O'Brien S, Hazi A, Kent S. 2014. Increased systolic blood pressure reactivity to acute stress is related with better self-reported health. *Sci Rep* 4:6882
443. Wu YE, Zhang CL, Zhen Q. 2016. Metabolic syndrome in children. *Exp Ther Med* 12:2390-4
444. Yang BZ, Zhang H, Ge W, Weder N, Douglas-Palumberi H, et al. 2013. Child abuse and epigenetic mechanisms of disease risk. *Am J Prev Med* 44:101-7

- 445. Yang Y, Kozloski M. 2011. Sex differences in age trajectories of physiological dysregulation: inflammation, metabolic syndrome, and allostatic load. *J Gerontol A Biol Sci Med Sci* 66:493-500
- 446. Yorbik O, Birmaher B, Axelson D, Williamson DE, Ryan ND. 2004. Clinical characteristics of depressive symptoms in children and adolescents with major depressive disorder. *J Clin Psychiatry* 65:1654-9
- 447. Zannas AS, Wiechmann T, Gassen NC, Binder EB. 2016. Gene-stress-epigenetic regulation of FKBP5: clinical and translational implications. *Neuropsychopharmacology* 41:261-74