

Positive and Negative Affect and the Mediating Effect of Perceived Stress on Health in  
Heart Failure Patients

by

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## ABSTRACT

Title of Thesis: Positive and Negative Affect and the Mediating Effect of Perceived Stress on Health in Heart Failure Patients

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**Objective:** Heart failure (HF) is a chronic and progressive disease with prevalence rates that continue to increase. Most research and treatment involving HF patients has focused on behavioral (e.g., smoking) and physiological risk factors (e.g., high blood pressure). Recently, the impact of various psychological factors such as anger, anxiety, depression, or perceived stress on HF patients have also been considered. In addition, positive and negative affect have been examined increasingly for potential influences on overall health. Both positive and negative affect can be viewed as either a state or trait. However, there has been little research examining the potential effects of affect on HF patients, and how perceived stress might impact this potential relationship. The aims of the present study are to: (1) evaluate if positive affect (PA) and negative affect (NA) are associated with psychological and physiological cardiovascular risk factors in HF patients; and (2) to examine the relationship between state and trait PA and NA on health

status of HF patients. (3) Finally, the present study will examine if perceived stress acts as a mediator upon the potential relationship between PA, NA, and health status in HF patients.

**Method:** HF patients ( $N = 147$ ) were assessed for PA, NA, perceived stress, and health status in relation to their HF symptoms at their baseline assessment and three-month follow-up assessment). The study sample was predominately African American (70.1%) and male (76.9%). Physiological variables (e.g., blood pressure, inflammatory markers) and other psychological variables (e.g., depression, anxiety, anger) were also measured during the baseline assessment period to assess for how affect was potentially correlated in HF patients.

**Results:** Partial correlational analyses, controlling for demographic factors, indicated that PA had a strong positive correlation with optimism and interpersonal support. PA also had a negative correlation with depression, anger, perceived stress and NA. NA had a strong positive correlation with depression, anger, and perceived stress and a negative correlation with optimism and interpersonal support. Surprisingly, PA had a positive correlation with anxiety only after controlling for perceived stress. Both “state” and “trait” PA and NA were examined by a mixed model analysis to create an average (i.e., “trait”) and deviation (i.e., “state”) variable from the PA and NA data at both the baseline and three-month follow-up assessments.

Mixed model analyses showed that both “state” and “trait” PA were predictive of better health as measured by the two different Kansas City Cardiomyopathy Questionnaire (KCCQ) scale scores. Both “state” and “trait” NA were predictive of worsening health as measured by KCCQ scores. Perceived Stress scores acted as a partial

mediator by weakening the relationship between NA, PA, and KCCQ scores. The impact of perceived stress on the relationship between NA, PA, and KCCQ scores was more significant for KCCQ Overall Summary scores. Additionally, when Perceived Stress Scale scores were controlled for then the relationship between “trait” PA, “trait” NA, and KCCQ scores were weakened and nonsignificant.

***Discussion:*** In summary, both PA and NA were related to changes in reported health as measured by KCCQ Overall and Clinical Summary scores. Perceived Stress scores only partially explained this relationship. This suggests other factors, such as methods of coping and interpersonal support, may also play a larger role in a HF patients’ reported health. Since self-efficacy is included in the Overall Summary score, self-efficacy may also play a role in these relationships.

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## **CHAPTER 1: Introduction to Heart Disease and Heart Failure**

Heart failure (HF), as defined by the American Heart Association (1), is a “chronic, progressive condition in which the heart muscle is unable to pump enough blood through to meet the body's needs for blood and oxygen.” This potentially debilitating condition is typically seen after the heart sustains damage from cardiovascular disease or coronary heart disease (1). When considering HF, the impacts of both cardiovascular disease and coronary heart disease should also be considered. CVD has been gradually declining in the past several years, but still remains one of the most prevalent diseases and a leading cause of death in the United States (3; 4). In 2010, CVD was responsible for approximately one in three deaths and CHD accounted for one in six deaths in the United States (5).

Mortality rates are not the only severe consequence associated with heart disease. Heart disease places a staggering economic burden on both an individual level and societal level, and this cost is only expected to triple in the next couple decades (6). One contributing cost to individuals with CVD is the large number of hospitalizations and rehospitalizations. Rehospitalization rates for HF patients are approximately between 15-37% in the United States and are only expected to rise (7; 8). Although most hospitalizations of HF patients are CVD related, HF alone tends to be the leading cause for these hospitalizations (9).

Additionally, a substantial amount of hospitalizations of HF patients are not cardiovascular related (9). The amount of non-cardiovascular hospitalizations seen in HF patients shows that comorbid and co-existing conditions should also be considered when

treating these individuals. In addition to direct medical care costs (e.g., hospitalizations), indirect costs such as loss of productivity, loss of wages, and decreased home productivity are expected to increase by approximately 61% by 2030 (6). Indirect costs place a burden on both the patient and their caretakers.

Both heart disease and heart failure are complex and many different factors are involved in the actual etiology. Most research conducted regarding CVD, CHD, and HF has been focused on behavioral risk factors (e.g., obesity, smoking, and stress) or physiological reactivity (e.g., heart rate and blood pressure; 10; 12). Potential psychosocial risk factors for the development of heart failure have also been examined, but have predominantly focused on identifying personality types and mental disorders that are correlated with increased risk (e.g., Type A personality, Major Depressive Disorder, anxiety, etc.; 13; 14).

In order to better understand risk factors for the development of HF, risk factors for the more general categories of CVD and CHD will be reviewed first. These risk factors include traditional factors (e.g., smoking, elevated cholesterol, hypertension), as well as psychosocial risk factors such as anger, anxiety, and depression. Therefore, protective factors such as social support and optimism will also be reviewed for their potential implications for preventing negative consequences of heart disease and heart failure such as comorbid health problems, hospitalizations, and death. Finally specific emotional styles, such as affect, will also be reviewed for their potential relationship with cardiovascular outcomes, risk factors, and protective factors.

## **CARDIOVASCULAR DISEASE, CORONARY HEART DISEASE, AND HEART FAILURE**

CVD has been utilized as a broad category to describe the various forms of heart disease (e.g., chronic rheumatic heart diseases, hypertensive heart disease, ischemic heart disease, myocardial infarction [i.e., heart attack], heart failure, stroke, arrhythmia) and the structures related to the heart that are affected (e.g., arteries, vessels, blood clot; 1; 15). As mentioned previously, CVD rates have been decreasing in the past several years but CVD still remains one of the most prevalent diseases in the United States (3-4). Some of the forms of CVD in particular are considered more likely to be fatal or result in a poorer prognosis, based on their mortality and hospitalization rates (1).

One particular form of CVD, coronary heart disease, involves the buildup of plaque in the heart's arteries (1). Due to the resulting obstruction in the heart's arteries, coronary heart disease (CHD) contributes to increased risk of adverse outcomes such as cardiac ischemia, heart attacks, and heart failure (1). The plaque buildup in the arteries (i.e., atherosclerosis) then deprives the heart of oxygenated blood (i.e., ischemia), which may lead to a heart attack or death in an individual (1). In the past several years CHD prevalence rates, similar to the broader CVD rates, have been decreasing (16). However, CHD continues to remain the most prevalent of CVD related mortalities by accounting for 48% of deaths in the United States (12).

The development of CHD can eventually lead to additional cardiovascular concerns, such as heart failure (1). During HF the heart's inability to pump enough oxygenated blood can cause the heart to compensate (e.g., enlarge, thicken the muscle walls, beat faster; 1). The heart's attempt to compensate for the lack of sufficient

oxygenated blood contributes to making this condition fatal for individuals. Potentially debilitating conditions, such as HF, are typically seen after the heart sustains damage from CVD or CHD (1). One possible contributing factor to why heart failure rates have continued to rise is the overall increase in an aging population due to improvements in health care overtime (6; 17).

HF and worsening progression overtime is generally monitored through several physiological measures. Beta- Natriuretic Peptide (BNP) is widely recognized biological marker for both the presence and severity of heart failure. BNP is particularly useful on measuring HF severity in individuals with comorbid conditions (18). Higher percentages of particular inflammation markers (e.g., Interleukin-1b, Tumor Necrosis Factor- $\alpha$ , Interleukin-6) in HF patients are associated with poorer prognosis and increased inflammation in HF patients (19; 20). A higher presence of Interleukin-10, however, is associated with a better prognosis in HF patients (20).

### **Adverse Outcomes of Heart Disease and Heart Failure**

Currently, mortality rates for HF patients are still considered high and a significant amount of these deaths are non-cardiovascular related (e.g., pulmonary disease, cancer, central nervous system disease, etc.; 9; 21). Mortality rates are not the only severe consequence associated with HF. HF hospitalizations and the associated economic burden have also been increasing (17). Rehospitalization rates for HF patients are as high as 37% and are only expected to rise (7; 8). Similar to deaths seen by HF patients, many of the hospitalizations experienced by HF patients are non-cardiac related (22). Hospitalization rates for non-cardiac conditions are hypothesized to also be high

because of the number of comorbid conditions associated with HF (e.g., diabetes, kidney disease infections, etc.; 22). Several risk factors have been identified to try to prevent mortality and rehospitalizations in HF patients, as well as to examine comorbid or coexisting conditions.

## **POTENTIAL RISK FACTORS FOR HEART DISEASE**

### **Health Behavior and Physiological Risk Factors**

#### ***Smoking***

Attempts to gain a better understanding of forms of CVD, particularly CHD, have resulted in a variety of theories and identification of risk factors. Smoking is both a standard and substantial risk factor due to the numerous effects it has on aspects of heart health (e.g., physiological changes, decreased ability to exercise, etc.; 1). Efforts to decrease smoking in the past several years, through smoking cessation programs and the usage of e-cigarettes, are believed to have contributed to decreased overall CVD rates (12). Despite reasonably successful efforts to decrease cigarette smoking, an extensive review found there are still individuals who continue to smoke because they perceive it as a coping mechanism for stress and negative emotions (23).

Although smoking tends to be perceived as a coping method to decrease stress, the chemicals in cigarettes have been shown to induce stress within the body on a physiological level (e.g., reduces lung capacity, builds plaque in arteries, increases risk of developing a blood clot, decreases HDL cholesterol, increases blood pressure, increases heart rate, narrows and thickens blood vessels, increases cortisol levels, damages the heart wall muscle; 2; 23). The physiological changes associated with smoking on the cardiovascular system are believed to then increase the individual's risk of experiencing a

heart attack (i.e., myocardial infarction) or sudden cardiac death (24). Furthermore, smoking is associated with known psychological risk factors for CHD, such as anxiety and anger (23; 25).

### ***Low-density Lipoprotein (LDL) cholesterol***

LDL cholesterol is essential for carrying proteins in the body; however, a higher quantity also increases the risk of developing problems associated with cardiovascular risk (27). The significance of LDL cholesterol on the development of heart disease and the progression of the disease has been examined by either isolating the LDL cholesterol from the total cholesterol amount or by viewing LDL in relation to High-Density Lipoprotein (HDL) Cholesterol (29). One concern in particular, is LDL helps produce plaque in the arteries and over time leads to Atherosclerosis (27). As described previously, Atherosclerosis ultimately deprives the heart of obtaining oxygenated blood and leads to additional adverse outcomes (e.g., heart attack, death) (1).

### ***Hypertension***

Another particular health problem that has been identified as a standard risk factor for heart disease is hypertension. Hypertension (i.e., high blood pressure) is described as either: Systolic blood pressure (i.e., blood pressure in the arteries when the heart contracts) at 160 mm Hg or higher or Diastolic blood pressure (i.e., pressure in the arteries when the heart is refilling with blood between heartbeats) at 90 mm Hg or higher (28). Combined with other risk factors (e.g., smoking, cholesterol, diabetes, obesity, physical inactivity), hypertension increases the odds of an individual to either initially develop cardiovascular disease or have a poorer progression of heart disease (2).

### ***Obesity***

Many of the health consequences associated with obesity are also present in CHD, which has made distinguishing health risk factors (e.g., decrease in HDL, hypertension, impaired glucose tolerance) between the two epidemics harder to isolate (10).

Considering the rising numbers of adults within the United States meeting criteria for obesity, the potential for increased cardiovascular risk is an additional concern (30).

68.8% of adult Americans were found to have either overweight or obese as recently as 2012 (30). Obesity is correlated with other health conditions such as sleep apnea, which may contribute to the development of CVD or even HF (31).

### ***Lack of Physical Activity***

A lack of physical activity, or physical inactivity, is defined as “physical activity levels less than those required for optimal health and prevention of premature death” (32). Overall, lack of physical activity has often been associated with the development of chronic diseases and conditions (e.g., heart disease, obesity, heart failure, hypertension, depression, etc.; 32; 33). The presence of physical activity, particularly vigorous physical activity, is inversely related with CHD risk (34).

Exercise has been widely known for not only helping potentially with weight loss but also for assisting in lowering resting blood pressure and heart rate. Higher levels of physical activity have also been associated with lower cardiovascular reactivity (e.g., heart rate) in response to stress (35). The presence of physical activity additionally is correlated with decreased anxiety and depression symptoms in individuals with a chronic condition such as dementia (36). Furthermore, physical activity is associated with

positive outcomes in cardiac rehabilitation settings by reducing both mortality and hospital admission rates (37).

### **Psychological Risk Factors**

Most research conducted regarding CHD and HF has focused on health behaviors (e.g., obesity, smoking, lack of physical activity) and stress (10; 12). Potential psychological risk factors involved in CHD and the development of HF have also been examined, but have predominantly identified particular personality types such as the Type A personality and psychological disorders that are correlated with increased risk (13; 14). However, there are several other psychological variables that have been correlated with CHD and HF.

### ***Stress***

There are several different approaches to the evaluation and description of stress. Different approaches emphasize various components of the stress process (e.g., physiological responses, the nature of the stressor stimulus, and cognitive appraisal of stressful events, an individual's capability to cope with stressful events; 23; 38; 39). Stress is generally defined as either a situation such as divorce or death that is appraised to require a large cognitive and emotional demand or the individual's evaluation of his or her own abilities to cope with particular demands (e.g., job loss, death, disease; 40). For the purpose of this review, stress will be defined as both a process that involves both the individual's appraisal of environmental demands (i.e., how difficult a situation seems) and their perceived capabilities of handling demands (i.e., how capable they feel of overcoming a particular situation).

Stress can be either acute or chronic. Acute stress affects the sympathetic nervous system and lead to physiological changes such as increased heart rate, increased blood pressure, and etc. (41). The impact of stress on the development of disease, to include CHD, has been widely explored but the exact physiological mechanisms are still a subject of study. Particular types of stressors, such as occupational environments and demands, have been associated with both cardiac events and the development of CAD (41).

Stress additionally increases both heart rate and blood pressure in individuals, which may contribute to increased mortality in HF patients (42). Both acute and chronic stress can lead to negative health behaviors such as smoking, poor diet, and lack of exercise (40; 23; 43). As described previously, the development and presence of negative health behaviors (e.g., smoking, poor diet, etc.) is related to adverse cardiovascular outcomes (e.g., hypertension, development of CVD, myocardial perfusion defects in CHD patients, and poorer prognosis in HF patients, etc.; 23; 31).

### ***Anger and Hostility***

Consistent with the definition described by Smith (as cited in 44), anger is “an unpleasant emotion ranging in intensity from irritation or annoyance to fury or rage.” Anger can then be further delineated into categories of state anger (i.e., temporary) and trait anger (i.e., chronic). There has been extensive literature on the potential correlation between the presence of anger and CHD. However, some of the research reviewing the association between anger and CHD has focused more specifically on aspects of hostility and “Type A” personality behavior (i.e., competitive drive, impatience, and hostility; 45).

A review by Chida and Steptoe (2009) described hostility as a trait that is

characterized by a cognitive or attitude aspect directed toward others. Hostility is then separate from anger because anger focuses instead on the actual variation in emotional states within a range (46). In a study of older adults with HF, approximately half of the individuals were found to have higher levels of hostility (47). Higher levels of hostility are correlated with higher blood pressure and heart rate during stressful events or tasks; this places individuals at risk for further cardiovascular consequences (48). Overall, higher levels of hostility in HF patients is associated with longer hospitalization duration (49). The presence of hostility, particularly when combined with feelings of cynicism, is associated with both the development of CHD and mortality from cardiac related events in HF patients (50; 51).

Trait anger has also associated with both the development of CHD in healthy individuals and poorer prognosis for those already diagnosed with CHD (46). Trait anger (i.e., chronic) is correlated with higher incidences of CHD related events and the presence of HF (25; 52). In the evaluation of symptoms of CHD, anger has been correlated with the development of angina (i.e., chest pain) (53). Trait anger is also correlated with higher incidences of other CHD risk factors (e.g., smoking, alcohol usage, higher Body Mass Index; 25). In addition to etiology and health behavior risks, the presence of anger is related to the type of hospitalizations individuals report. Keith (2016) found that in heart failure patients, both hostility and anger were associated with all cause hospitalizations, but not with cardiac hospitalizations specifically.

HF patients experience acute anger, in addition to trait anger, more frequently than CHD patients due to the characteristics of the disease (i.e., living with a chronic illness that impacts multiple aspects of the individual's life; 54). Extensive reviews have

concluded that patients often experienced acute (i.e., state) anger prior to a cardiac event occurring (e.g., myocardial infarction, acute coronary syndrome, arrhythmia, etc.; 51; 55; 56). The duration of time that is considered the highest risk, for a cardiovascular event to occur, is within two hours after an acute anger event (i.e., anger outburst) occurs (55).

### ***Anxiety***

A review by Suls and Bunde (2005) noted that part of the difficulty in examining anxiety in cardiovascular disease populations is the lack of differentiation between state and trait anxiety in studies. Although feelings of anxiety can be described as being a temporary state, for the purposes of this review anxiety will be focused on as a trait (i.e., longer duration pattern). Similar to anger, higher levels of anxiety or worry have been associated with a variety of potential risk factors for CHD (e.g., smoking, higher alcohol usage, family history of CHD; 23; 57).

In addition to being associated with other risk factors, higher chronic anxiety levels are correlated with an overall increased risk of developing CHD and mortality (57; 58). When examining older HF patients (i.e., age sixty or older), higher levels of anxiety were reported in comparison to healthy older adults (47). Anxiety may also contribute to a poorer prognosis in HF patients because of its potential effects on cardiovascular pathophysiology (e.g., decreased heart rate variability, plaque rupture, increased blood pressure, etc.) and quality of life (47; 59).

### ***Depression***

The potential psychological, behavioral, and biological implications of depression have also been explored for individuals at risk of CHD. Depression has generally been

linked with inflammation processes (e.g., impaired coronary flow, autonomic nervous system dysfunction), which increase the risk of developing CVD or CHD (60). However, there have been some contradictory research claims that show depression is not associated with increased risk for inflammation in individuals with CHD (61).

Various aspects of depression pertain to a variety of characterizing symptoms (e.g., fatigue, sleep problems, reduced physical activity, inflammation), which also overlap with symptoms of CHD or HF (62). Depression has additionally been associated with health risk behaviors (e.g., smoking, lack of physical activity, lack of medication compliance) that increase risk of developing CHD or result in a poorer prognosis in patients with heart disease (23; 60). When examining acute depression (i.e., state depression), a review by Steptoe and Brydon (2009) concluded depression is correlated with both developing CHD and to a poorer prognosis following a cardiac event (e.g. myocardial infarction).

In addition to depression being a factor associated with other risk factors contributing to the development of CHD and HF, depression is a prevalent coexisting disorder among HF patients (14). Both meeting criteria for major depressive disorder (MDD) and mild depression have been seen to be associated with increased mortality risk in HF patients (63). Higher rates of depression have also been associated with mortality and frequent rehospitalizations in HF patients (14; 49; 63).

## **PROTECTIVE FACTORS**

To gain a more thorough understanding of epidemiology and potential prevention, protective factors should also be explored. One area of psychology that has made an

effort to focus more on the positive aspects of an individual instead of the negative pathological aspects (e.g., psychological disorders, disease, illness) is positive psychology. One component of positive psychology is the focus on positive emotions, such as happiness, that are also a component of positive affectivity (64). Positive affectivity has been examined for its effects upon various health outcomes, to include heart disease (65; 66). In addition to protective emotional factors within positive psychology, social support is an important factor within the literature while examining disease processes (64; 67). Positive psychology, optimism, and social support will be reviewed further within the following sections.

### **Social Support**

Social support has been proposed as a potential buffer to help protect individuals from negative outcomes by providing positive affect and stability (68). Overall, perceived social support is associated with decreased cardiac risk (69). Uchino, Cacioppo, and Kiecolt-Glaser (1996) found in their review that the presence of social support was associated with lower blood pressure levels, lower hypertension rates, and higher rates of adherence to health management behaviors such as medication compliance, smoking cessation, and healthier eating. Similarly, higher levels of perceived social support are associated with lower mortality rates after myocardial infarctions and better prognosis in CHD patients (69; 70).

### **Optimism and Positive Psychological Characteristics**

Particular areas within positive psychology that are related to health have been defined as coherence, optimism, benefit finding, and post-traumatic growth (71). Other areas typically encompassed within positive psychology involve positive emotion,

engagement in life, and having a purpose in life (72). The benefits of focusing on positive psychology have begun being utilized in treatment for acute cardiovascular disease to help reduce negative symptoms and overall risk factors (e.g., depression symptoms, anxiety; 73).

One area in particular that is vital within the health and positive psychology literature is the concept of optimism. Dispositional optimism is defined as a general trait that helps predict how an individual will cope with stress (71). Higher levels of optimism have also been correlated with a greater perception of social support, smaller increases in depression and stress, and utilization of coping strategies (74). Optimism is associated with an overall reduced risk of cardiovascular mortality, even when controlling for cardiovascular risk factors and depressive symptoms (75). Most recently, studies demonstrated that dispositional optimism is related to fewer incidents of HF and to fewer hospitalizations (76).

#### **POSITIVE AND NEGATIVE AFFECT**

In addition to the previously described risk factors and protective factors, affect has been explored more recently in relation to health and CHD. Consistent with the definition presented by Pressman and Cohen (2005), affect is defined as emotions and attitudes that are either positive or negative. Items that measure these attitudes include calm, depressed, cheerful, or nervous (65). There has been mixed outcomes in the literature in terms of effects of either positive or negative affect, partially because the wide range of adjectives used to define affective states and the variety of measures used to identify it (66).

Although positive affect (PA) and negative affect (NA) have been seen to be independent factors, they are not considered to be entirely independent of each other (66). The delineation between NA and PA becomes more difficult due to NA being periodically identified by lower PA levels instead of as a standalone factor (77). A review by Jacnicki-Deverts et al. (2007) found that low PA, in addition to high NA, contributes to the presence of clinical depression in individuals with life-threatening and chronic illnesses.

Additionally, Cohen et al. (2003) found that an increase in positive emotional styles (i.e., PA) is associated with decreased rates in illnesses such as the cold virus, but negative emotional style (i.e., NA) was not associated with clinical cold incidences. Negative emotional style was seen to be associated with increased somatic health complaints that were not indicative of actual disease. Although there are some aspects of affect that are similar to the previously mentioned psychological risk factors such as depression and anger, affect will be explored separately due to its overall focus on emotion and attitude states.

### **Negative Affect**

Negative affect is the “general dimension of subjective distress” and is comprised of negative emotions or feelings (e.g., anger, contempt, disgust; 78). Some research has compressed negative psychological traits (e.g., NA, depression, anxiety, increased CHD risk) and instead categorized them underneath a singular larger category (79). For the purpose of this review, NA will be considered separately from potentially overlapping diagnoses and symptoms perceived stress, and anxiety.

The presence of negative affect has been proposed to have severe adverse outcomes in regards to overall health (e.g., mortality, increased risk of developing disease, greater somatic complaints; 66; 80). A large sample study conducted by Nabi et al. (2008), found that individuals with higher measured levels of NA had a slightly elevated risk of having a coronary heart disease incident. The presence of NA, as an independent factor separate from PA, is also associated with CVD related hospitalizations and mortality (82).

### ***Health Behaviors and Physiological Risk Factors Associated with NA***

NA is also associated with risk-related health behaviors and physiological risk factors for CVD. Smokers tend to report experiencing more NA than nonsmokers (23). Negative affect, typically associated with perceived stress, is also seen to be elevated prior to smoking relapse in individuals attempting smoking cessation (83). The mechanism that leads to higher NA is proposed to be from the anticipation of there being a period of abstinence from smoking; therefore, leading to higher levels of NA as part of withdrawal prior to a relapse (84). Alcohol consumption is associated with NA and negative emotions, with alcohol consumption serving as a potential mechanism for coping with stress (85). Although alcohol consumption is not directly associated with the development of CVD or HF, if the consumption becomes chronic or in excess (i.e., binge drinking) then the risk for cardiovascular consequences is increased (86; Roerecke & Rehm, 2009).

High NA symptoms that are consistent with depression or anxiety are also associated with higher incidence rates of treated hypertension, especially in women (88;

89). NA is associated with changes in blood pressure over a prolonged amount of time of 18 months (89). Surprisingly, there has been little to no evidence found associating NA with cholesterol levels (90). In addition to standard risk factors for CHD, NA is also associated with lower immune functioning and greater susceptibility to viruses (65; 91).

### ***Psychological Risk Factors Associated with NA***

In addition to NA associations with physiological and health behavioral risk factors, by definition, negative affect is associated with several psychological risk factors (e.g., depression, anxiety, lower levels of optimism, pessimism, PTSD; 91-94). As described previously, a possible explanation for the correlation between NA and psychological risk factors is due to the overlapping adjectives and descriptors for emotions (e.g., sad, hostile, calm, etc.) and how often both are categorized together as one factor in various studies (66). The presence of NA is also associated with interpersonal risk factors for CHD such as low social support (93).

### **Positive Affect**

Contrary to negative outcome effects seen by higher levels of negative affectivity, PA is related to predominately positive health outcomes. Increased levels of PA, or positive emotional style (PES), have been associated with decreased rates of illness development and disease progression (65; 66). As mentioned previously, PA and NA are separate factors but they are not necessarily independent of each other. In female breast cancer survivors, higher rates of high-arousal positive affect (e.g., excited, active, alert) are associated with lower inflammatory markers at 6-12 month follow-ups following cancer treatment (95).

PA additionally has had mixed findings regarding its relationship with serious health outcomes, such as mortality. An extensive review by Pressman and Cohen (2005) found that PA was associated both with increased longevity of life and decreased longevity of life in a variety of different samples. The relationship between mortality and trait PA was only strengthened in individuals with diseases with a high mortality rate and poor short-term prognosis, such as cancer (66). Additionally, lower levels of PA in reaction to stressors are related to increased mortality risk (96). A possible explanation for conflicting findings within the literature is that PA is only beneficial in improving mortality rates in healthy older individuals (i.e., over 60 years of age; 66). Additionally, PA's measurement through a variety of scales has made interpreting findings more difficult (66).

Explanations for the possible relationship between PA, positive health outcomes, and a lower incidence of disease suggest that PA is associated with lower levels of stress and better adherence to better health practices (65). Some research has contradicted this theory and found that PA did not prevent stress, but instead it was other factors (e.g., coping mechanisms, social support presence, optimism) that decreased the risk for adverse health consequences (93; 97).

### ***Health Behaviors and Physiological Risk Factors Associated with PA***

A higher presence of PA is associated with better health outcomes (e.g., improved immune functioning, lower stress hormones, decreased inflammation, better sleep, less comorbidity, medication adherence, increased physical activity; 65; 66; 97; 98). Research

indicates that PA at baseline is predictive of better health behaviors such as not smoking, improved sleep, and lower alcohol use among individuals with CHD (98). The association between PA and improved health behaviors was also seen in the individuals with CHD up to five years after their initial baseline assessment (98).

Variability in PA has been generally examined for influences on physiological factors, including inflammation and heart rate variability. Lower PA, measured over a period of 6 days after individuals were exposed to a virus, is associated with increased inflammation as measured by the IL-6 biomarker (77). Higher PA is also associated with greater heart rate variability, which is a protective factor for individuals with suspected coronary heart disease (99). Higher PA, assessed by measuring positive behaviors such as smiling and positive verbal responses, is associated with decreased rates of incident CHD (i.e., heart failure) over a 10-year period (100). However, there have been limited studies overall that have examined associations between PA and cardiovascular risk factors in patients with heart failure.

### ***Psychological Risk Factors Associated with PA***

PA has been correlated with a higher reporting of positive psychological and interpersonal factors that may protect against disease development (e.g., higher levels of optimism, perceived social support, emotional support, healthier coping; 93; 101). Similarly, high levels of self-reported positive emotions (i.e., PA) have been seen in chronically ill patients with high levels of stress (102). Pressman & Cohen (2005) suggested in their review that PA acts as a buffer for individuals against stress. Lower PA is associated with symptoms of depression, especially when found in conjunction with

high NA (94). However, there has been less evidence linking PA with reduced anxiety, as opposed to the amount of research that demonstrates a positive relationship between anxiety and the presence of NA (94).

In summary, the literature suggests that both NA and PA are related to cardiovascular risk and protective factors as well as CVD outcomes. NA has predominately been associated with the presence of factors that increase CVD risk (e.g., anger, depression, anxiety, PTSD, smoking, alcohol consumption, lower immune functioning, etc.) (23; 65; 66; 85; 89). NA is associated with increased risk of incident coronary heart incident (e.g., heart attack), hospitalizations, and mortality (81; 82).

PA inversely is related to better health behaviors (e.g., not smoking, better diet, etc.) and protective factors (e.g., social support, healthy coping, etc.; 65; 66; 97; 98; 101). Higher levels of PA have additionally been associated with improved chance of survival, decreased mortality, and better immune functioning (66; 95). However, research also indicates that PA and NA may interact in their relationship with psychological and disease risk factors. A review by Jacnicki-Deverts et al. (2007) found that low PA, in addition to high NA, contributes to clinical depression in individuals with life-threatening and chronic illnesses. Low PA with higher NA is associated with poorer prognosis (e.g., hospitalizations and mortality) in CVD patients (82).

## **CHAPTER 2: Summary and Rationale for the Proposed Study**

Overall, the development and progression of coronary heart disease, including heart failure have been prospectively associated with numerous behavioral, physiological,

and psychological risk factors (e.g., LDL cholesterol, smoking, obesity, stress, anxiety, depression, etc.). NA and other negative emotions such as depression and anxiety have been implicated as risk factors for cardiovascular disorders. While PA has been related to protective factors for CHD such as positive health behaviors, increased social support, and higher levels of optimism.

However, the majority of research on NA and PA has been conducted in healthy individuals and relatively little is known about relationships between NA, PA, and health outcomes in patients with cardiac disorders such as HF. Existing research on negative and positive affect has focused on specific elements of NA such as depression, anxiety, and anger in relation to cardiovascular outcomes rather than on NA as a global construct. The possible protective effects of PA, in patients with heart failure need further investigation.

Pressman and Cohen (2005) propose that PA acts as a buffer against the effects of stress on physiological responses and health behaviors. Stress is associated with affect reactivity, increased cardiovascular risk symptoms (e.g., increased heart rate, increased BP, etc.), and increased negative health behaviors that may serve as a form of coping (e.g., smoking, obesity, etc.; 23; 40; 103).

However, multiple factors (e.g., genetics, healthy lifestyle behaviors, inflammatory differences, general psychosocial factors) have also been considered as potential mediators between positive affect and health (97). Stress also needs to be further evaluated as a potential mediator on the relationships between NA, PA, and health outcomes, and buffering effects of PA in cardiac patients (40; 101).

Therefore, the present study will examine several issues related to associations between NA, PA, and health status in patients with heart failure. To better understand possible relationships between affect, risk factors, and outcomes in HF patients, we will first examine the relationship of positive and negative affect to traditional risk factors (e.g., smoking, lack of physical activity, etc.) and to related psychosocial HF risk factors (e.g., protective factors such as optimism and psychological risk factors such as stress). Following this, we will determine if NA and PA are prospectively related to health status changed in HF patients, and whether stress mediates these associations.

### **Specific Aim 1**

To determine the relationship between negative and positive affect and psychosocial and behavioral variables (e.g., anxiety, depression, anger, hostility, smoking, physically activity) in patients with HF, and whether these associations are independent of perceived stress.

### ***Hypothesis 1a***

NA will be positively correlated with perceived stress and other psychosocial risk factors (e.g., anxiety, depression, anger, hostility, history of smoking) and negatively correlated to protective factors such as social support, coping, physical activity, and optimism.

### ***Hypothesis 1b***

PA will be positively correlated with protective factors such as social support, coping, physical activity, and optimism. PA will be negatively correlated with anxiety, depression, anger, hostility, history of smoking, and perceived stress.

***Hypothesis 1c***

Correlations between NA, PA, psychosocial and behavioral risk factors will be weakened after controlling for perceived stress.

**Specific Aim 2**

To determine the relationship between affect, blood pressure (BP), BNP, and immune variables (e.g., IL-1B, IL-6, IL10, TNF-alpha), and whether these associations are independent of perceived stress.

***Hypothesis 2a***

NA will be positively correlated to BP, BNP, and immune markers, and PA will be negatively correlated to BP, BNP, and immune markers.

***Hypothesis 2b***

Perceived stress will be positively correlated with blood pressure, BNP, and immune markers.

***Hypothesis 2c***

When perceived stress is controlled for, then the correlations seen between affect and physiological factors will be weakened.

**Specific Aim 3**

To examine the relationships of “trait” negative and “trait” positive affect to symptoms and KCCQ self-reported health status scores over a 3-month follow-up period.

***Hypothesis 3a***

HF patients with a higher average level of NA will demonstrate more symptoms and poorer health status, as measured by the KCCQ, at a 3-month follow-up.

***Hypothesis 3b***

HF patients with a higher average level of PA will exhibit fewer symptoms and better self reported health status, as measured by the KCCQ, over a 3-month follow-up.

***Hypothesis 3c***

Perceived stress will be associated with HF symptoms and poorer health status in HF patients, as measured by the KCCQ.

***Hypothesis 3d***

Perceived stress will mediate the relationship between NA, PA, and self-reported health status in HF patients, such that the associations will be weakened.

**CHAPTER 3: Method**

## **PARTICIPANTS AND RECRUITMENT**

This study will draw data from a larger study, Behavioral Triggers of Heart Failure (BETRHEART), which was conducted at the Heart Failure Clinic at the University of Maryland Hospital in Baltimore, Maryland. The proposed study is based on the archival data collected from the previous BETRHEART study.

This study has received IRB approval through the Uniformed Services University of the Health Sciences (USUHS), the Baltimore Veterans Affairs (VA) Medical Center and the University of Maryland. The sample for this study consists of 150 participants, who were patients at the Heart Failure clinic at the time of the BETRHEART study recruitment. To meet eligibility criteria for study inclusion, all participants met diagnostic criteria for HF (left ventricular ejection fraction of less than or equal to 40% and New York Heart Association HF class II-IV for at least three months). A classification of II-IV by the New York Heart Association (NYHA) indicates that an individual has mild to severe limitations on physical activity (26).

All participants during recruitment were in stable condition, as determined by a board certified physician, and at least 21 years of age. Individuals were excluded from study participation if they had: (a) documented myocarditis for less than 6 months, (b) clinically significant mitral valve disease, (c) thyroid dysfunction, (d) current alcohol abuse or alcohol abuse within the past six months, (e) cognitive impairment that would interfere with consent and questionnaire completion, (f) implanted left ventricular assist device, (g) prior heart transplantation, (h) cancer treatment currently, (i) been living in a nursing home, or (j) currently pregnant.

## **PROCEDURES**

During a routine visit for heart failure, patients determined to be eligible by the inclusion criteria for the study were identified and informed consent was obtained. At the baseline assessment, participants completed various self-report measures to assess mood, depression symptoms, anger, anxiety, and perceived stress. Physiological measures, such as blood pressure, weight, and blood samples were also obtained at this time. After the conclusion of the baseline, patients were retested at a 3-month follow-up utilizing the same measures as the baseline assessment. A telephone interview was additionally conducted every two weeks, between the baseline and 3-month follow-up assessments. Additionally measures to evaluate self-efficacy, social support, and optimism were collected during these telephone interviews.

## **MEASURES**

### **Psychosocial Variables**

#### ***Measurement of Negative Affect and Positive Affect***

Patients were given a questionnaire called the “Mood and Health Practices Questionnaire” (MHPQ) comprised of 23 self-report items that examine negative mood states, positive mood states, and health behaviors in the past 24 hours. The 18 items pertaining to positive or negative mood states consist of adjectives that have previously been used for measuring negative affect, or negative emotional style, and positive affect (i.e., positive emotional style; 65). Patients completed the MHPQ at both the baseline and three-month follow-up assessments.

These eighteen items were adapted from the widely utilized Positive and Negative Affect Schedule (PANAS) created by Watson, Clark, & Tellegen (1988), but were not utilized by Cohen et al. (2003) because the PANAS lacks specific adjectives for

measuring affect such as “calm” or “happy” (66). The PANAS has also been noted to consist of adjectives (e.g., strong) that are not considered to be consistent with measuring mood or emotional states (66). This questionnaire has been found to have both validity and reliability, with Cronbach's alpha from 0.89 to 0.93 for the PA scales and from 0.87 to 0.92 for the NA scales (65).

### ***Perceived Stress Scale (PSS-10)***

The PSS is a measure of perceived psychological stress (104). The Perceived Stress Scale was originally a fourteen-item questionnaire but the current proposed study utilized the abbreviated ten-item questionnaire to reduce patient burden. The ten-item questionnaire consists of items concerning perception of stressful events, as evaluated in the past day. A five-point Likert scale is utilized with “0” indicating “never” and “4” indicating “very often.” The PSS-10 has good internal validity with a Cronbach’s alpha of .78 and .91 in two separate survey samples (105). Patients completed the PSS-10 at both the baseline and three-month follow-up assessments.

### ***Beck Depression Inventory (BDI-II)***

The Beck Depression Inventory-II (106) contains 21 statements depicting symptoms of depression within the past two weeks. A higher score on the scale indicates increasing severity in depression symptoms (106). Mild depression scores are within the 14-19 range, moderate depression is within the 20-28 range, and severe depression symptoms are indicated by a score of 29 and above (106). The BDI-II has been found to be valid with have an alpha of .91 with a sample of 140 psychiatric outpatients (107). The BDI-II has not only been extensively validated, but it has been one of the most widely utilized measures to assess for both the presence and severity of depression (108). The

questionnaire was administered during baseline and each follow-up visit over the 36-month follow-up period.

### ***State-Trait Anxiety Inventory (STAI)***

The STAI is a 40-item self-report instrument that utilizes items to evaluate an individual's self-reported experiences with anxiety symptoms, with each item coded on a 4-point Likert scale (109). The STAI has been found to be both valid and reliable with a Cronbach alpha of .86 when examining various populations (e.g., working adults, high school and college students, military recruits) (109; 110). The STAI contains two subscales to measure both state and trait anxiety, with higher scores correlated with higher symptomology of anxiety (109). The state-anxiety responses are coded as “not at all”, “somewhat”, “moderately so”, and “very much so.” The trait-anxiety responses are coded as “almost never”, “sometimes”, “often”, and “almost always.” Items assessing for the presence of state-anxiety include: “I am tense” and “I am worried.” Items assessing for the presence of trait-anxiety include: “I worry too much over something that really doesn't matter.” The STAI was administered to patients at the initial baseline assessment.

### ***State-Trait Anger Expression Inventory (STAXI-II)***

The STAXI-2 is a 57 item instruments that measures expressions of anger with each item coded on a 4-point Likert scale, with higher scores on the STAXI-2 correlated with greater symptomology of anger (111; 112). The STAXI-2 has been found to be both reliable and valid with a variety of individuals (e.g., heterogeneous normal populations, college undergraduates, etc.; 112). State-anger responses on the STAXI-2 are coded as “not at all”, “somewhat”, “moderately so” and “very much so.” Item statements that assess for state-anger include “I am furious” and “I feel irritated”. The trait-anger

responses are coded as “almost never”, “sometimes”, “often”, and “almost always.” Trait-anger is assessed per statements such as, “I am a hotheaded person” and “I am quick tempered.” The STAXI was administered at the initial baseline assessment.

### ***Cook-Medley Hostility Scale***

The Cook-Medley Hostility Scale is a 50-item scale from the larger Minnesota Multiphasic Personality Inventory (MMPI) that utilizes true or false responses to statements to measure hostility (113). The scale has been studied with a variety of populations, including individuals with documented coronary artery disease (114). The scale has convergent and discriminant validity and reliability with individuals with coronary artery disease and heart disease (115).

### ***Life Orientation Test (LOT)***

The LOT is a 16-item measure used to evaluate an individual’s level of optimism and pessimism by coding items on a 5-point Likert scale (i.e., ranging from strongly disagree to strongly agree; 116). Items on the LOT include such statements as, “In certain times, I usually expect the best” (116). The LOT has been found to be reliable with a Cronbach alpha of .76 (116).

### ***Interpersonal Support Evaluation List (ISEL)***

The ISEL is an instrument used to evaluate an individual’s perceived access to social support resources through 48 statements coded on a 4-point Likert Scale (i.e., definitely false to definitely true; 117). The ISEL-12 was used in this study to help reduce participant burden. The ISEL-12 utilizes three subscales to determine perceived support through appraisal (e.g., “I feel that there is no one I can share my most private worries

and fear with”), belonging (e.g., “If I decide one afternoon that I would like to go to a movie that evening, I could easily find someone to go with me”), and tangible (e.g., “If I were sick, I could easily find someone to help me with my daily chores”; 118). The ISEL-12 has been found to be reliable with a Cronbach alpha of at least between .77 (118) and it has been found to be a valid measure for a variety of racial and ethnic groups (119; 120).

## **Physiological Variables**

### ***Beta-Natriuretic Peptide (BNP)***

Blood samples for both BNP and immune and inflammatory markers were collected at both the initial baseline visit and three-month follow-up. Blood samples were collected using vacuum-sealed tubes (Ethylenediaminetetraacetic acid [EDTA] 4.5 mmol/l), then mixed gently for 30 seconds and then allowed to set at room temperature for 45 minutes. Blood samples were then placed in a port of the testing device to allow red blood cells to be separated from plasma through filtration. Plasma was then moved into a reaction chamber and allowed to incubate after forming a reaction mixture. The incubated reaction mixture is then analyzed through analyte and fluorescent antibody conjugates. All blood samples were stored at -80° C until the completion of the three-month follow-up assessment.

BNP samples were analyzed within one hour after collection utilizing the Triage® BNP assay by Biosite in San Diego, California. The Triage® BNP assay is a fluorescence immunoassay that determines the quantity of BNP within either a whole blood or plasma sample that has utilized EDTA as an anticoagulant. In addition to the blood sample collection to analyze BNP, participants were asked to complete a BNP

assessment questionnaire to help ensure the sample was viable at the time of collection. The questionnaire asked the participant for information on their sleep recently, perceived capability of completing potentially exerting activities (e.g., walking, biking, running, sexual intercourse, etc.), symptoms of anger, medication, and substance usage.

### ***Immune and Inflammatory Markers.***

Collection of immune and inflammatory markers occurred during the same blood sample collection conducted for BNP at both the initial baseline visit and three-month follow-up assessment. As described previously, blood samples were collected using vacuum-sealed tubes (Ethylenediaminetetraacetic acid [EDTA] 4.5 mmol/l), then mixed gently for 30 seconds and set at room temperature for 45 minutes. Blood samples were then mixed in a temperature-controlled centrifuge at 3000 g for 15 minutes to allow for separation of the plasma. Samples were again stored at -80° C until the completion of the three-month follow-up assessment. Tumor Necrotic Factor-alpha (TNF- $\alpha$ ), Interleukin 6 (IL-6), and Interleukin 10 (IL-10) levels within the blood samples were measured by Singulex® through the utilization of their Erenna® Immunoassay system. Singulex's® Erenna® Immunoassay system and specific procedures on their various kits for measuring IL-6, IL-10, and TNF- $\alpha$  can be downloaded from their website.

### **Outcome Variables**

#### ***Kansas City Cardiomyopathy Questionnaire (KCCQ)***

The KCCQ is a 23-item self-report measure that is utilized to evaluate the individual's perceived heart failure severity and symptoms, and has been found to be predictive of future hospitalizations and death in HF patients (121). The KCCQ includes

statements such as “my symptoms of heart failure have become...” and “how much has your heart failure limited your enjoyment of life?” Subjects respond to each question on a 5-point scale, with different responses ranging from “uncertainty” and “extreme limitation” to “certainty” and “feeling unlimited”. A score of 1 on a question includes such responses as the participant feeling “extremely limited” in the performance of specific activities, feeling their symptoms of HF have become “much worse” in the past two weeks, and feeling their symptoms are “extremely bothersome” or occurring “every night.” A score of five on a question includes such responses as feeling “not at all limited”, that in comparison with the past two weeks they are feeling “much better”, and that they feel “completely sure” they are capable of knowing who they would call or what to do if their HF symptoms are exacerbated. The KCCQ was collected at both the baseline and three-month follow-up assessments.

The KCCQ includes eight different domain scales (i.e., Physical Limitation, Symptom Stability, Symptoms as measured by NYHA class, Self-Efficacy, Social Limitation, Quality of Life, KCCQ functional status, and KCCQ Clinical Summary), with the potential to also look at an Overall Summary score (i.e., total score). However, the present study only utilized two of the domain scales on the KCCQ that represented overall HF health status. The Overall Summary Score of the KCCQ quantifies several domains, including physical limitations, symptoms (i.e., frequency, severity and recent change over time), self-efficacy, social limitations, and quality of life. The Clinical Summary Score includes the functional status score (i.e., physical limitations and symptoms), Quality of Life score, and the social limitation score, but does not include a measure of self-efficacy. This scale was utilized to avoid the possible confound of the

relationship of self-efficacy to PA and NA (see below). KCCQ scores range from 0 to 100, with fewer heart failure symptoms and better health status indicated by a higher score. The KCCQ has been found to be a reliable measure with a Cronbach's alpha for the Clinical Summary Score of .95 (121). The Overall Summary Score has been found to be reliable and correlated with NYHA status, in both anemic HF patients and non-anemic HF patients, with a Cronbach's alpha of .93 (122).

### **DATA ANALYSES**

SPSS 24.0 was used for data analysis. To analyze the first two hypotheses, a partial correlation was conducted to determine if PA and NA are correlated with standard cardiovascular risk factors (e.g., BP, BNP, inflammation and immune factors, smoking, reported physical activity, socioeconomic status [SES],) and psychosocial risk factors (e.g., stress, anxiety, depression, social support, optimism, anger and hostility). A partial correlation was conducted to control for the potential influence of standard cardiovascular factors (e.g., gender, race, SES (i.e., household income). Covariates were selected based on their impact historically within the larger cardiovascular literature. An additional partial correlation was conducted to determine if the relationship between affect is weakened after controlling for perceived stress. An additional partial correlation was conducted to determine if the relationship between affect is weakened after controlling for perceived stress.

To examine the third aim, a general linear mixed model (GLMM) was conducted to determine if affect (i.e., NA and PA) is predictive of scores on the KCCQ. The advantage of the GLMM is that it allows for mean (i.e., "trait-like") and deviation ("state like") effects to be examined as well. The mixed model utilized the mean and standard

deviations of the variables (i.e., affect and KCCQ) to calculate a cluster variable that utilizes scores from both the baseline assessment and the three-month follow-up assessment. The cluster variables for NA and PA were utilized in the linear mixed model to determine if there are within subject (i.e., deviation) or between subject (i.e., average) differences between the two groups across the two separate time points (i.e., baseline and the three month follow-up) on reported KCCQ scores.

To examine health status of individuals with respect to various domains (i.e., symptoms, functional status, quality of life, self-efficacy, etc.), a linear mixed model was conducted as separate analyses to examine any differences seen between the KCCQ Overall Summary Score and the KCCQ Clinical Summary score. PSS scores was also calculated utilizing a cluster variable to similarly evaluate between and within subject effects of PSS-10 scores on KCCQ scores, and determine if perceived stress is a mediator for the relationship between affect (i.e., NA and PA) and KCCQ scores on the two separate scales. Potential mediation was determined using the approach described by Baron and Kenny( 1986).

## **POWER**

The initial validity study for the development of the KCCQ consisted of 129 participants with congestive heart failure (121). As previously stated, the current study is derived from the BETRHEART study, which was comprised of 150 participants. Since the current study sample is larger than the one used to initially validate the KCCQ and if the independent variables for the current study are found to be significant, then the study can be assumed to have appropriate power.

Additionally, power was calculated using G\*Power for a repeated measures ANOVA (124). Although a repeated measures ANOVA was not utilized in the current study, the power analysis is similar enough to be used to calculate an adequate sample size. G\*Power for an ANOVA, repeated measures with a within factor, determined that a total sample of at least 29 participants was necessary to yield the desired calculated effect size of 0.71 while maintaining adequate power at 95%.

## **CHAPTER 4: Results**

### **SAMPLE CHARACTERISTICS**

A sample of 150 participants was initially recruited at the University of Maryland

Medical Center and Baltimore VA heart failure clinic. Of the 150 recruited, only 147 participants completed the baseline demographic information and were included in the demographic information. 70.1% of the participants identified as African American (n=103) and approximately 29.3% identified as White. The mean (SD) age of participants was 57.41 (11.46) years and ranged from 23-87 years. Demographic data for the sample are provided in Table 1. The sample was predominately within a lower socioeconomic status and received a household annual income below \$15,000. The majority of participants (n=104) indicated that they had a history of smoking. On average, participants spent approximately 9.97 months (3.47) within the study total. Data was only utilized up to the three-month follow-up period due to the hypotheses focusing on shorter-term outcomes for HF patients, and particular measures only being utilized within this follow-up time period for the larger BETRHEART study.

### **Bivariate Demographic and Behavioral Risk Factor Correlations**

Baseline PA was only negatively correlated with NA [ $r(147) = -0.44, p \leq .001$ ] and being physically active [ $r(147) = -0.24, p < .01$ ]. Baseline NA was not significantly correlated with any of the demographic variables. However; due to the current study's proposed covariates (e.g., race, gender, SES (i.e. household income), age) being used consistently throughout the larger cardiovascular literature, the covariates were still included within data analyses. Demographic data and bivariate correlations of demographic variables are included in tables 1 and 2 below.

### **CROSS-SECTIONAL ANALYSES**

#### **Aim 1: Associations Between Psychological Risk Factors, PA, and NA**

Baseline PA was found to have a strong positive correlation with several factors after controlling for demographic factors (e.g., gender, age, race, and SES as determined by income). Baseline PA was positively correlated with reported optimism [ $r(122) = .33, p \leq .001$ ] and perceived interpersonal support [ $r(122) = .33, p \leq .001$ ]. Baseline PA was also negatively correlated with baseline NA [ $r(122) = -.42, p \leq .001$ ], baseline depression [ $r(122) = -.48, p \leq .001$ ], baseline state anger [ $r(122) = -.26, p < .01$ ], and baseline perceived stress [ $r(122) = -.59, p \leq .001$ ]. Baseline NA was found to have a strong positive correlation with baseline depression symptoms [ $r(122) = .54, p \leq .001$ ], baseline state anger [ $r(122) = .40, p \leq .001$ ], and baseline perceived stress [ $r(122) = .70, p \leq .001$ ]. Baseline NA also had a small to medium negative correlation with optimism [ $r(122) = -.31, p < .01$ ] and perceived interpersonal support [ $r(122) = -.22, p < .05$ ].

When controlling for perceived stress, partial correlation analysis showed that the relationship between affect (e.g., NA and PA) and the previously tested psychosocial variables was significantly weakened. PA was now positively correlated with baseline state anxiety [ $r(122) = .19, p < .05$ ]. No other significant correlations were seen between baseline PA and psychosocial variables. Baseline NA was still positively correlated with baseline depression symptoms [ $r(122) = .20, p < .05$ ] and baseline state anger symptoms [ $r(122) = .26, p < .01$ ], but the size of this correlation was weakened. Refer to table 3 for more details on the partial correlations between affect and the psychosocial variables.

## **Aim 2: Associations Between PA, NA, and Physiological Variables**

The partial correlation analysis, while controlling for demographic variables such as gender, age, race and income, found that there were no significant correlations

between positive affect, negative affect, and physiological variables. All of the correlations within the analysis were small in magnitude and nonsignificant. The only other significant correlations seen were between inflammatory biomarkers, and between diastolic and systolic blood pressure. Refer to table 4 for more detailed information on the correlations between affect and physiological variables. When these analyses were conducted again controlling also for perceived stress, these results were relatively unchanged.

## **LONGITUDINAL ANALYSES**

### **Aim 3a: Mixed Model Analyses of PA**

Cluster variables were utilized to examine potential within (i.e., deviation) PA and between (i.e., mean or “trait like”) differences in relation to effect on both KCCQ Overall Summary scores and KCCQ Clinical Summary scores. Results of the linear mixed model analysis indicated that deviation PA (i.e., within-subject positive affect) was a significant predictor of increased KCCQ Overall Summary scores (i.e., better health status),  $b = 0.57$ ,  $t(122.97) = 3.02$ ,  $p = .003$ , 95% CI [0.20, 0.94]. Mean (i.e., between-subject) PA did not significantly predict health status as measured by KCCQ Overall Summary scores,  $b = -0.11$ ,  $t(142.88) = -0.12$ ,  $p >.05$ , 95% CI [-2.08, 1.85]. After controlling for PSS, Mean PA was still not significantly predictive of health status as determined by the KCCQ Overall Summary Scores,  $b = -0.89$ ,  $t(141.13) = -0.71$ ,  $p >.05$ , 95% CI [-3.37, 1.60].

Results of the linear mixed model analysis also indicated that deviation PA was a significant predictor of increased Clinical Summary KCCQ scores (i.e., indicated better health status),  $b = 0.68$ ,  $t(122.98) = 0.68$ ,  $p \leq .001$ , 95% CI [0.31, 1.1]. Mean PA (i.e.,

between-subject or trait like affect) was also significantly predictive of increased Clinical Summary KCCQ scores, better health status,  $b = 0.90$ ,  $t(138.72) = 0.68$ ,  $p \leq .001$ , 95% CI [0.52, 1.3]. After controlling for Perceived Stress Scale scores, mean PA was no longer a significant predictor of Clinical Summary KCCQ scores,  $b = 0.20$ ,  $t(138.04) = 0.89$ ,  $p > .05$ , 95% CI [-0.24, 0.64]. Deviation PA was still a significant predictor of Clinical Summary KCCQ scores but the size of the effect was weakened,  $b = 0.47$ ,  $t(121.80) = 0.89$ ,  $p \leq .05$ , 95% CI [0.08, 0.85].

The findings overall for Aim 3a show that the presence of self-efficacy within the KCCQ scale has an impact on the relationship seen between affect and health status in HF patients. When examining the Clinical Summary KCCQ scores, both increases in “state like” and “trait like” PA were associated with a better health status. Perceived Stress had a small impact on the relationship between affect and health status, when examining “state like” (i.e., deviation) PA. When only utilizing KCCQ Overall Summary scores, only “state like” increases in PA were seen to be associated with increased reported health status in HF patients.

### **Aim 3b: Mixed Model Analyses of NA**

Cluster variables were utilized again to examine within (i.e., deviation) NA and between (i.e., mean or “trait like”) differences in relation to effect on both KCCQ Overall Summary scores and KCCQ Clinical Summary scores. Deviation NA (i.e., within-subject) was a significant predictor of decreased KCCQ Overall Summary scores, which indicated poorer health,  $b = -0.92$ ,  $t(123.24) = -5.67$ ,  $p \leq .001$ , 95% CI [-1.24, -0.60]. Mean NA (i.e., between-subject) did not significantly predict KCCQ Overall Summary Scores on the KCCQ,  $b = -1.35$ ,  $t(123.24) = -5.67$ ,  $p > .05$ , 95% CI [-3.63, 0.94]. After

controlling for PSS scores, deviation NA was still a significant predictor of decreased KCCQ Overall Summary scores,  $b = -0.74$ ,  $t(122.33) = -4.12$ ,  $p \leq .001$ , 95% CI [-1.10, -0.38]. After controlling for PSS scores, mean NA was still not a significant predictor of decreased KCCQ Overall Summary scores,  $b = -1.54$ ,  $t(141.50) = -0.95$ ,  $p > .05$ , 95% CI [-4.74, -1.67].

Deviation NA (i.e., within-subject) was a significant predictor of decreased Clinical Summary KCCQ scores, which indicated poorer health,  $b = -0.92$ ,  $t(123.82) = -5.58$ ,  $p \leq .001$ , 95% CI [-1.25, -0.59]. Mean NA (i.e., between-subject or “trait like”) also significantly predicted a decrease in Clinical Summary KCCQ scores,  $b = -1.24$ ,  $t(58.47) = -5.12$ ,  $p \leq .001$ , 95% CI [-1.73, -0.76]. After controlling for PSS scores, the between-subjects effect for mean NA on Clinical Summary KCCQ scores was no longer significant,  $b = -0.75$ ,  $t(80.23) = -1.14$ ,  $p > .05$ , 95% CI [-0.97, 0.26]. However, deviation NA was still a significant predictor of Clinical Summary KCCQ scores, but the size of the effect was weakened,  $b = -0.74$ ,  $t(122.57) = -4.07$ ,  $p \leq .001$ , 95% CI [-1.11, -0.38].

Similar to the results seen when examining PA, self-efficacy appeared to have an impact upon the relationship between affect and health status as measured by the two different KCCQ scores. When self-efficacy was included, as part of the KCCQ Overall Summary score, only “state like” NA was seen to be a negative predictor of decreased health status. After controlling for PSS scores, the relationship between “state like” NA and both KCCQ scores was weakened. The relationship between “trait like” NA was only seen to be significant when examining its effect on KCCQ Clinical Summary scores, without PSS scores being controlled for.

### **Aim 3c: Mixed Model Analyses of PSS**

Similar to the previous two discussed mixed model analyses, “cluster” variables were again utilized to examine “state like” and “trait like” effects of perceived stress on KCCQ scores. Deviation Perceived Stress (i.e., within-subject) significantly predicted Overall Summary Scores on the KCCQ,  $b = -0.77$ ,  $t(123.09) = -4.31$ ,  $p \leq .001$ , 95% CI [-1.12, -0.41], with increases in deviation PSS significantly predicting decreased KCCQ scores (i.e., poorer health). Mean Perceived Stress scores (i.e., between-subject) did not significantly predict Overall Summary Scores on the KCCQ,  $b = -0.71$ ,  $t(142.27) = -0.71$ ,  $p > .05$ , 95% CI [-2.70, 1.27]. Deviation Perceived Stress scores (i.e., within-subject) significantly predicted Clinical Summary Scores on the KCCQ,  $b = -0.75$ ,  $t(122.67) = -4.15$ ,  $p \leq .001$ , 95% CI [-1.11, -0.39], with increases in deviation Perceived Stress Scale scores predicting decreased KCCQ scores (i.e., poorer health). Mean Perceived Stress Scale scores (i.e., between-subject) also significantly predicted Clinical Summary Scores on the KCCQ,  $b = -1.29$ ,  $t(143.68) = -7.23$ ,  $p \leq .001$ , 95% CI [-1.11, -0.39].

### **Aim 3d: Mediation Analyses**

A linear mixed model analysis was utilized to determine if Perceived Stress Scale scores mediated the relationship between affect (i.e., PA and NA) and HF symptoms as measured by the KCCQ (i.e., Overall Summary score and Clinical Summary score). For the mediation analyses, PA and NA data from both the baseline time point and the 3-month follow-up time point were simultaneously utilized as independent variables. Similarly, Perceived Stress Scale scores from the baseline and 3 month follow-up were utilized within the mediation model simultaneously as independent potential mediator variables.

### ***PA and Overall Summary KCCQ Scores***

For positive affect, results indicated that PA was a significant negative predictor of PSS scores,  $b = -0.51$ ,  $SE = 0.05$ ,  $p \leq .001$ , and PSS was a significant predictor of Overall Summary KCCQ scores,  $b = -0.97$ ,  $SE = 0.14$ ,  $p \leq .001$ . PA was a significant predictor of Overall Summary KCCQ scores,  $b = 0.85$ ,  $SE = 0.13$ ,  $p < .001$ . PA was also a significant predictor of Overall Summary KCCQ scores after controlling for the mediator, PSS,  $b = 0.39$ ,  $SE = 0.14$ ,  $p < .05$ . These results supported the hypothesis that after controlling for Perceived Stress scale scores, the relationship between PA and Overall Summary KCCQ would be weakened which is consistent with partial mediation.

### ***NA and Overall Summary KCCQ Scores***

For negative affect, the mixed model analysis determined that Perceived Stress also mediated the relationship between NA and heart failure symptoms as measured by the Overall Summary KCCQ scores. NA was found to be a significant predictor on Overall Summary KCCQ scores,  $b = -1.11$ ,  $SE = 0.03$ ,  $p \leq .001$ , with increases in NA implying decreased Overall Summary KCCQ scores and lower reported health status. Results indicated that NA was a significant positive predictor of Perceived Stress,  $b = 0.65$ ,  $SE = 0.05$ ,  $p \leq .001$ , and that Perceived Stress was a significant predictor of KCCQ scores,  $b = -0.77$ ,  $SE = 0.14$ ,  $p \leq .001$ . These results again support the meditational hypothesis. NA was a significant predictor of Overall Summary KCCQ scores after controlling for the mediator, PSS,  $b = -0.67$ ,  $SE = 0.15$ ,  $p \leq .001$ .

### ***PA and Clinical Summary KCCQ Scores***

When examining PA and KCCQ Clinical Summary scores, there was still a significant effect seen between PA and Clinical Summary KCCQ scores after controlling for PSS scores,  $b=0.39$ ,  $SE=0.14$ ,  $p > .05$ . The effect seen between PA and PSS was equivalent to the previously described findings when examining the KCCQ Clinical Summary scores. Additionally, PSS was seen to have a negative effect upon KCCQ Clinical Summary scores.  $b=-0.83$ ,  $SE=0.14$ ,  $p \leq .001$ . When only examining the effect of PA upon Clinical Summary KCCQ scores, PA was seen to be predictive of greater health status (i.e., increases in Clinical Summary KCCQ scores)  $b=0.79$ ,  $SE=0.13$ ,  $p \leq .001$ .

#### ***NA and Clinical Summary KCCQ Scores***

Examination of NA and Clinical Summary KCCQ scores with PSS as a potential mediator yielded similar results to PA. PSS was a significant mediator of the relationship between NA and Clinical Summary KCCQ scores,  $b=-0.66$ ,  $SE=0.16$ ,  $p \leq .001$ . The effect between NA and PSS was equal to the previous analysis conducted with Overall Summary KCCQ scores because no other variables were altered. Similarly PSS scores was seen to be significantly predictive of decreased health status as measured by the Clinical Summary KCCQ scores,  $b=-0.66$ ,  $SE=0.15$ ,  $p \leq .001$ . When only examining the total effect of NA on Clinical Summary KCCQ scores,  $b=-1.03$ ,  $SE=0.13$ ,  $p \leq .001$ , NA was seen to significantly predict decreases in health status.

#### ***Mediation Results Overall***

The results indicated overall that NA had a larger impact on KCCQ scores than PA, regardless of which score was utilized as the outcome (i.e., Overall Summary or Clinical Summary). Additionally, PSS scores acted a partial mediator upon the

relationship between PA, NA, and both scales of KCCQ scores. Cluster variables were not utilized in the mediation analyses. The reason for this is that hypothesis 3a is focused on relationships between overall Positive and Negative Affect and KCCQ scores, and does not involve examining within and between subject differences. See Figures 1 and 2 for more detailed information regarding these mediation analyses.

## TABLES

Table 1. Sample Characteristics (N=147)

| Variable |      | Mean $\pm$ SD or %     |
|----------|------|------------------------|
| Gender   | Male | 113 (76.9%)            |
| Age      |      | 57.41 $\pm$ 11.46 (SD) |

|                     |                   |             |
|---------------------|-------------------|-------------|
| Race                | African American  | 103 (70.1%) |
|                     | Caucasian         | 43 (29.2%)  |
|                     | Other             | 1 (.7%)     |
| Household Income    | <\$15,000         | 51 (34.9%)  |
|                     | \$15-30,000       | 39 (26.7%)  |
|                     | \$30-70,000       | 43 (29.5%)  |
|                     | >\$70,000         | 13 (8.9%)   |
| Baseline Creatinine | 1.38 ± .71 (SD)   |             |
| Ejection fraction   | 23.14 ± 7.48 (SD) |             |
| History of Smoking  | 104 (70.7%)       |             |
| Months in Study     | 9.97 ± 3.47 (SD)  |             |

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*Note:* \* p < .05, \*\*p < .01, \*\*\*p ≤ .001

Table 2. Bivariate Correlations of Standard Demographic and Behavioral Risk Factors (N=147)

|                      | Baseline<br>PA    | Baseline<br>NA    | Gender            | Age               | Race             | Household<br>Income | Hx of<br>Smoking  | Physically<br>Active |
|----------------------|-------------------|-------------------|-------------------|-------------------|------------------|---------------------|-------------------|----------------------|
|                      | M=22.8<br>SD=8.40 | M=8.10<br>SD=7.70 | M=1.20<br>SD=0.42 | M=57.4<br>SD=11.5 | M=1.72<br>SD=9.5 | M=2.12<br>SD=1.00   | M=1.29<br>SD=0.46 | M=1.37<br>SD=0.48    |
| Baseline PA          | 1                 | -0.44***          | 0.12              | 0.02              | 0.16             | -0.04               | 0.13              | -0.24**              |
| Baseline NA          | --                | 1                 | -0.06             | -0.10             | -0.03            | -0.12               | -0.00             | 0.06                 |
| Gender               | --                | --                | 1                 | -0.12             | 0.21**           | -0.00               | 0.11              | -0.05                |
| Age                  | --                | --                | --                | 1                 | -0.36***         | 0.09                | -0.03             | 0.17*                |
| Race                 | --                | --                | --                | --                | 1                | -0.16               | -0.06             | -0.17*               |
| Household<br>Income  | --                | --                | --                | --                | --               | 1                   | 0.16              | 0.16                 |
| Hx of<br>Smoking     | --                | --                | --                | --                | --               | --                  | 1                 | -0.06                |
| Physically<br>Active | --                | --                | --                | --                | --               | --                  | --                | 1                    |

*Note:* \*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p \leq .001$ . Hx is being used as an abbreviation for history. Physically active is defined as being active for greater than 30 minutes, at least three times a week, for the past month. Both history of smoking and physically active were categorized as binary variables with “1” meaning “yes” and “2” meaning “no.” Gender and race were also binary variables with a “1” notating “Male” and “not Hispanic/Latino” and a “2” meaning “Female” and “Hispanic/Latino.”

Table 3. Partial Correlations of Psychosocial Variables (N=122)

|                                 | Baseline<br>PA   | Baseline<br>NA  | Optimism         | Interpers.<br>Support | Baseline<br>Depression | Baseline<br>State<br>Anxiety | Baseline<br>State<br>Anger | Hostility<br>Symptoms | Baseline<br>Perceived<br>Stress |
|---------------------------------|------------------|-----------------|------------------|-----------------------|------------------------|------------------------------|----------------------------|-----------------------|---------------------------------|
|                                 | M=22.8<br>SD=8.6 | M=7.9<br>SD=7.7 | M=21.5<br>SD=5.7 | M=38.2<br>SD=6.4      | M=11.9<br>SD=9.5       | M=21.6<br>SD=93.7            | M=16.7<br>SD=5.7           | M=2.8<br>SD=130.1     | M=13.1<br>SD=8.0                |
| Baseline PA                     | 1                | -0.42***        | 0.33***          | 0.33***               | -0.48***               | 0.09                         | -0.26**                    | -0.14                 | -0.59***                        |
| Baseline NA                     | --               | 1               | -0.31**          | -0.22*                | 0.54***                | 0.17                         | 0.40***                    | 0.07                  | 0.70***                         |
| Optimism                        | --               | --              | 1                | 0.32***               | -0.34***               | -0.47***                     | -0.18*                     | -0.35***              | -0.59***                        |
| Interpersonal<br>Support        | --               | --              | --               | 1                     | -0.38***               | -0.39***                     | -0.20                      | -0.25                 | -0.48*                          |
| Baseline<br>Depression          | --               | --              | --               | --                    | 1                      | 0.79***                      | 0.52***                    | 0.52**                | 0.72**                          |
| Baseline State<br>Anxiety       | --               | --              | --               | --                    | --                     | 1                            | 0.53***                    | 0.45***               | 0.79***                         |
| Baseline State<br>Anger         | --               | --              | --               | --                    | --                     | --                           | 1                          | 0.36***               | 0.49***                         |
| Hostility<br>Symptoms           | --               | --              | --               | --                    | --                     | --                           | --                         | 1                     | 0.44***                         |
| Baseline<br>Perceived<br>Stress | --               | --              | --               | --                    | --                     | --                           | --                         | --                    | 1                               |

Note: \* p<.05, \*\*p<.01, \*\*\*p≤.001. Covariates controlled for in the partial correlation include: age, gender, race, and household income.

Table 4. Partial Correlations of Physiological Variables; after controlling for covariates (N=122)

|               | PA               | NA              | BNP                 | IL-1b           | IL-6              | IL-10           | TNF- $\alpha$   | SBP                | DBP               |
|---------------|------------------|-----------------|---------------------|-----------------|-------------------|-----------------|-----------------|--------------------|-------------------|
|               | M=22.9<br>SD=8.1 | M=8.5<br>SD=7.9 | M=501.5<br>SD=693.7 | M=0.2<br>SD=0.1 | M = 6.4<br>SD=9.2 | M=1.6<br>SD=1.1 | M=5.7<br>SD=4.0 | M=120.2<br>SD=20.0 | M=75.4<br>SD=12.4 |
| PA            | 1                | -0.41***        | -0.10               | -0.10           | -0.08             | -0.13           | -0.06           | 0.01               | 0.10              |
| NA            | --               | 1               | 0.03                | 0.03            | 0.11              | 0.17            | 0.11            | 0.01               | 0.06              |
| BNP           | --               | --              | 1                   | 0.20*           | 0.35***           | 0.06            | 0.24*           | 0.08               | 0.13              |
| IL-1b         | --               | --              | --                  | 1               | 0.09              | 0.23*           | 0.22*           | 0.03               | 0.03              |
| IL-6          | --               | --              | --                  | --              | 1                 | 0.48***         | 0.27**          | -0.04              | -0.10             |
| IL-10         | --               | --              | --                  | --              | --                | 1               | 0.37***         | 0.02               | 0.00              |
| TNF- $\alpha$ | --               | --              | --                  | --              | --                | --              | 1               | -0.37              | -0.37             |
| SBP           | --               | --              | --                  | --              | --                | --              | --              | 1                  | 0.76***           |
| DBP           | --               | --              | --                  | --              | --                | --              | --              | --                 | 1                 |

*Note:* \*  $p < .05$ , \*\* $p < .01$ , \*\*\* $p \leq .001$ . Positive Affect (PA), Negative Affect (NA), Beta-Natriuretic Peptide (BNP), Tumor Necrotic Factor-alpha (TNF- $\alpha$ ), Interleukin inflammation variables (IL-1b, IL-6, and IL-10), Systolic Blood pressure (SBP), and Diastolic blood pressure (DBP).

FIGURES

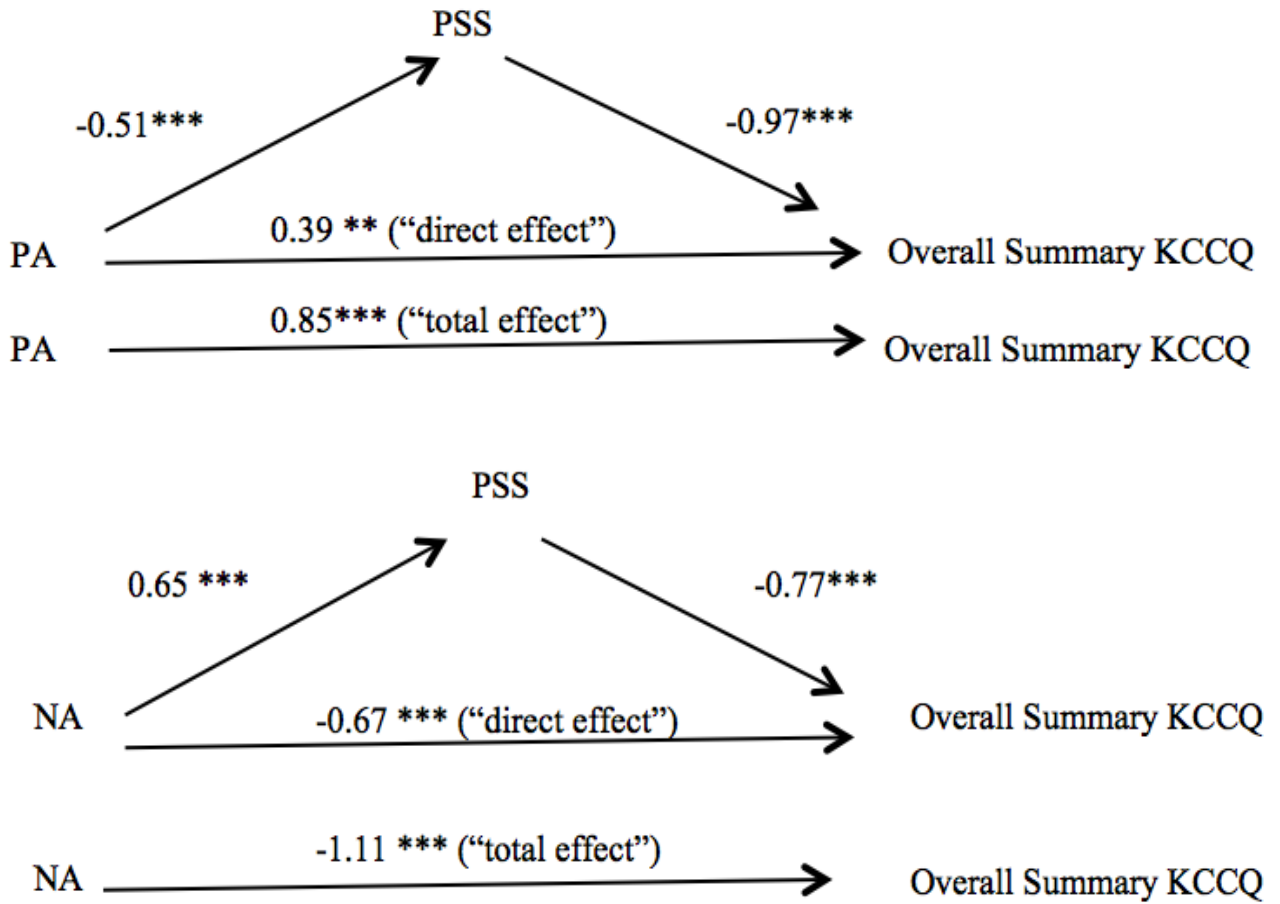


Figure 1. NA and PA mediation analyses showing a significant relationship between both forms of affect and Overall Summary KCCQ scores, with PSS scores as a significant partial mediator upon the relationship. Values represent estimates of fixed effects with \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

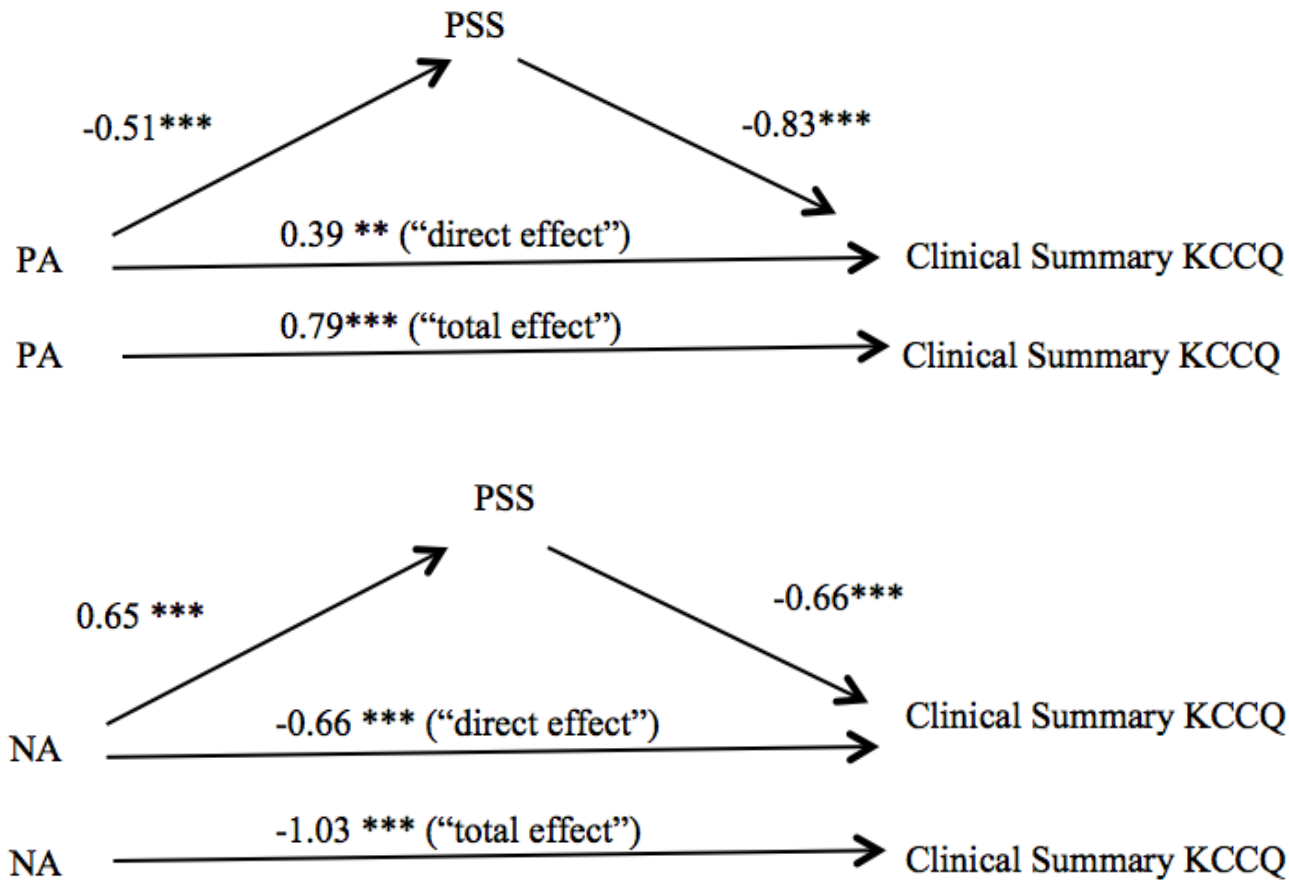


Figure 2. NA and PA mediation analyses showing a significant relationship between both forms of affect and Clinical Summary KCCQ scores, with PSS scores as a significant partial mediator upon the relationship. Values represent estimates of fixed effects with \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

## **CHAPTER 5: Discussion**

The current study confirms that PA and NA are correlated with various psychosocial variables such as optimism, interpersonal support, depression, perceived stress, and anger. After PSS scores were controlled for, associations between affect and psychosocial variables were weakened. The weakened correlations seen after controlling for PSS scores show that an individual's perceived stress has an impact on the relationship between affect (i.e., PA and NA) and psychosocial factors such as depression or anger. Surprisingly, there were few relationships among NA and PA and physiological variables related to heart failure risk. In longitudinal analyses, however, NA and PA were both predictive of HF symptoms and health status, and these relationships were partially mediated by Perceived Stress Scale scores. Similar to the findings from the correlation analyses, the relationship between affect and health status was weakened after controlling for PSS scores.

### **AFFECT ASSOCIATIONS WITH PSYCHOLOGICAL VARIABLES**

Surprisingly, PA was not significantly correlated with anxiety until after PSS was controlled for in the analyses. Previous literature has found a correlation between anxiety, NA, and higher levels of physiological arousal (125). A review of studies examining PA and anxiety found small inverse correlations between the two (126), which directly contrasts the positive correlation between PA and anxiety for the current study. The positive correlation seen between PA and anxiety then could be in part due to an effort to enhance PA, as a form of coping, despite having high levels of anxiety (127; 128). Additionally, the correlation seen between PA and anxiety may be due to

unforeseen collinearity between constructs. A more detailed factor analysis would need to be conducted to determine if some of the findings associated with affect were due instead to overlaps in constructs.

Because of the correlational nature of the present analyses, we cannot conclude whether higher levels of PA are a cause of increased interpersonal support and optimism, or if these factors (i.e., interpersonal support and optimism) instead cause HF patients to report having higher levels of PA. However, the strong positive correlations seen between PA and a variety of health behaviors (e.g., medication adherence; 129) suggest that it is important to at least consider levels of PA when treating HF patients. The strong correlations seen with NA indicate an overlap in the general measurement of NA, anger, and depression. The strong correlations between NA and psychosocial risk factors also suggest the importance of addressing a HF patient's symptoms related to anger and depression and to help reduce their general level of NA.

#### **AFFECT ASSOCIATIONS WITH PHYSIOLOGICAL VARIABLES**

Previous studies have shown mixed findings regarding affect being associated with physiological variables such as BNP or IL-6 (93; 130; 131). PA is associated with lower IL-6 and TNF- alpha, but the association is weakened after controlling for factors such as depression or the specific type of affect measure used in the study (i.e., PANAS or Global Mood Scale; 130). One previous study only found PA to be associated with lower IL-6 levels in female participants (93). The presence of optimism was not associated with lower inflammation levels in a study that was comprised of predominately white males (132). Both state anger and anxiety have also been associated with higher BNP (130), IL-6, and TNF-alpha (132). Considering the strong correlation

seen between NA and anger in the current study, it is surprising that NA did not also show also show a correlation with at least BNP in the current study's results.

Although there were no significant correlations seen between NA and physiological factors, the potential impact of perceived stress was still accounted for. There were still no significant correlations seen between affect and physiological factors after controlling for PSS scores. Zawadski et al. (2017) found that individuals with higher affectivity did not necessarily have higher physiological reactivity, even after being exposed to a task designed to elicit a particular emotion such as anger. The reason for the lack of a significant correlation between affect and physiology may be due to other factors within the current study's sample.

Although demographic factors were controlled for, the sample for the current study was predominately male and African American. Previous findings show an increase in NA overtime in individuals with chronic medical conditions, but the connection between NA and chronic medical conditions was seen to be less prevalent in African Americans (134). Assari and Lankarani (2016) proposed that the difference seen between Caucasians and African Americans, when examining NA and chronic medical conditions, could be partially explained by culture differences related to resilience. Overall, there has been very little research on racial differences when examining the association between affect, physiological reactivity, and heart failure.

The previously described mixed findings between affect, psychosocial variables, and physiological factors then may be partially explained by the demographic characteristics and potential lack of demographic generalizability of the current study. This study also focused on HF patients specifically while some of the previous studies

involving inflammation markers have focused on a range from health in the general population (93) to myocardial infarction (132).

## **MIXED MODEL AND MEDIATION ANALYSES**

### **State versus Trait Effects**

Differences seen between affect and KCCQ scores, when examining “state like” and “trait like” affect, reflects some of the previous research conducted. State PA has previously been associated with slower progression of several disorders, to include cardiovascular disease (66). Trait PA is associated with influencing disease outcomes when the disease processes occur over a longer period of time (66). In our study sample then, when examining the Clinical Summary KCCQ Scores, the significant finding of state PA after controlling for PSS suggests that PA is more predictive of the health status in individuals through looking at the actual progression of the disease and potential disease related incidences (e.g., hospitalizations) as suggested by the previous Pressman & Cohen (2005) findings.

There are differing views regarding how state and trait NA are related to various health outcomes. State NA is associated with increased objective disease outcomes (i.e., actual worsening of heart condition) while trait NA is related to increased subjective complaints (40). The current study’s findings suggest that individuals with higher state NA are actually experiencing increased health concerns after controlling for PSS scores, as indicated by their Clinical Summary KCCQ scores. As stated previously, the present results indicate that trait NA was not a significant predictor of Clinical Summary KCCQ scores. This lack of a significant finding suggests that trait NA has less of an impact on health status and this may be from potentially less actual subjective complaints (40).

### **The Role of Perceived Stress as a Mediator**

Previous studies have suggested that NA acts as a moderator on the relationship between social support and perceived stress and that PA does not have a significant influence upon this relationship (135). Çivitci (2015) finding regarding the moderation effect of affect suggests that in the current study we would expect the relationship between NA, PA, and KCCQ scores to remain after accounting for PSS scores. As the present results indicated, this was not the case in this study sample, and perceived stress instead acted as a partial mediator. The current study's findings suggest that factors, other than perceived stress (e.g., interpersonal support or coping style), related to affect are not being accounted for when considering potential contributions and moderating effects on KCCQ scores.

In the mixed model analyses, the greater differences seen in the associations of NA, PA, and KCCQ scores on Overall Summary scores than Clinical Summary scores after controlling for PSS, further suggests that stress has an impact upon self-efficacy in HF patients. Self-efficacy is utilized in computing the Overall Summary Score but not in the Clinical Summary Score. Both "trait like" and "state like" Perceived Stress scores were predictive of decreased health status and lower KCCQ Clinical Summary scores. This was not the case when looking at KCCQ Overall Summary scores and suggests that self-efficacy may instead act as a buffer between stress and reported health status in HF patients.

### **The Role of Self-Efficacy**

If it is the case that HF patients with higher PA and/or lower NA have a greater sense of self-efficacy and therefore report a greater level of health, the present research

adds to the literature indicating that self-efficacy is associated with better self-care in HF patients and improved reported health (136). However, generally there has been little to no prior research on the relationship between affect and self-efficacy in HF patients. Most research has focused on the association between affect in either healthy populations (137) or with cancer survivors (138). Prior findings indicate that there is a significant positive association between physical functioning and self-efficacy, and a significant negative association between self-efficacy and illness severity (139). The positive associations seen between PA and positive health behaviors (e.g., self-efficacy, self-care) in HF patients (136; 139) suggest that clinicians should further consider the role of PA when providing care for these individuals.

#### **STUDY LIMITATIONS**

As stated previously, the sample for the current study was predominately male and African American. As a result, the findings may not be as representative to the wider general population but they convey more insight on HF with this particular demographic group. African Americans are at increased risk for developing heart disease, and ultimately experiencing heart failure, due in part to a higher prevalence of comorbid diseases and conditions such as diabetes (140). Although women have been reported to have a better prognosis after being diagnosed with HF, the potential impact on their quality of life should be explored further and could be partially explained by differing levels in affect (141; 142). Furthermore, higher levels of positive affect or positive emotions have been found to be protective against negative affect in women (143).

As noted by Endrighi et al. (2016), the exact influence of PSS and the various changes between baseline and the three-month follow-up may have also been impacted

by various factors such as decreased stress due to frequent contact from study staff.

Additionally, the measurement points used in the current study were limited to those used in the larger BETRHEART study. The proposed outcomes of the current study would be stronger if there had been more time points available for assessment measures or if there were other potential outcomes measures considered in conjunction (i.e., the number of adverse cardiac events) to determine how strong of an effect PA and NA have upon HF patients. It is also important to note that the current study measures PA and NA through items that have not been more widely utilized throughout research, therefore exact comparison to other studies may be more difficult.

Future research should further examine how affect is being measured and the correlations between various instruments. As several previous studies have indicated, there is still inconsistency in how affect is being defined and measured (65; 66; 133). As stated previously, affect items for the current study were adopted from the items utilized by Cohen et al. (2003) because the more widely utilized PANAS lacks specific adjectives (e.g., calm, happy) that reflect the range in activation associated with various affective states. Pressman and Cohen (2005) also noted that particular adjectives that are not considered accurate measures of mood or emotional states (e.g., strong; 66) are included in the PANAS. However, the usage of affect items from Cohen et al. (2003) should then also be further examined in relation to other instruments for examining affect to help maintain greater consistency and generalizability.

#### **CLINICAL IMPLICATIONS AND FUTURE DIRECTIONS**

Future studies should further examine the role of PA and NA upon other psychological variables aspects (e.g., coping, self-efficacy, optimism) that are related to

health in HF patients. The evidence for whether there is a predictive relationship between PA, NA, and health status in HF patients would be increased by examining other outcome measures pertaining to cardiovascular outcomes in HF (e.g., hospitalizations, physiological measures, myocardial infarction rates, death). Another direction for future research in the area of affect and health would be to create a more universal and standardized definition and measurement of affect. Additionally, it would be important to determine whether the present findings can be replicated in other demographic groups.

The unexpected positive correlation seen between PA and anxiety after controlling for PSS scores, in the present study's sample of HF patients, should also be examined further in other populations to determine greater generalizability. The exact influence of self-efficacy and PA upon an individual's health status could also be explored further, since this relationship can potentially be capitalized upon to help improve health in individuals with a chronic disease such as heart failure. HF patients who are able to experience more frequent daily PA could be expected to have increased feelings of self-efficacy, better adherence to positive health behaviors, and ultimately a better prognosis.

In summary, the current study contributes to the general literature on emphasizing aspects of positive psychology within clinical care. The findings suggest that "state like" changes in affect, both NA and PA, have a significant impact on how individuals with heart failure perceive and experience their health status. When coordinating care with HF patients, providers may want to emphasize the association between positive mood states and the patient's perceived health. Similarly, treatment interventions could be designed to

further reduce HF patients' negative affect and mood states while they are undergoing treatment.

## REFERENCES

1. American Heart Association. 2015. *About Heart Failure*. Dallas, TX: American Heart Association.  
[http://www.heart.org/HEARTORG/Conditions/HeartFailure/AboutHeartFailure/About-Heart-Failure\\_UCM\\_002044\\_Article.jsp#.Vj0MSYT5NUQ](http://www.heart.org/HEARTORG/Conditions/HeartFailure/AboutHeartFailure/About-Heart-Failure_UCM_002044_Article.jsp#.Vj0MSYT5NUQ)
2. American Heart Association. 2014. *Smoking & Cardiovascular Disease (Heart Disease)*. Dallas, TX: American Heart Association.  
[http://www.heart.org/HEARTORG/HealthyLiving/QuitSmoking/QuittingResources/Smoking-Cardiovascular-Disease\\_UCM\\_305187\\_Article.jsp#.V5kmbldWLjI](http://www.heart.org/HEARTORG/HealthyLiving/QuitSmoking/QuittingResources/Smoking-Cardiovascular-Disease_UCM_305187_Article.jsp#.V5kmbldWLjI)
3. Ford, ES, Ajani, UA, Croft, JB, Critchley, JA, Labarthe, DR, Kottke, TE, ... Capewell, S. 2007. The decrease in US deaths from coronary disease, 1980-2000. *New England Journal of Medicine*. 356:2388-2398.
4. Murphy, LS, Xu, J, Kochanek, KD. 2013. Deaths: Final Data for 2010. *National Vital Statistics Reports*. 61:1-117.
5. Go, AS, Mozaffarian, D, Roger, VL, Benjamin, EJ, Berry, JD, ... Turner, MB. 2013. Heart disease and stroke statistics—2013 update: A report from the American Heart Association. *Circulation*. 127:e6-e245.
6. Heidenreich, PA, Trogon, JG, Khavjou, OA, Butler, J, Dracup, K, Ezekowitz, MD, ... Lloyd-Jones, DM. 2011. Forecasting the future of cardiovascular disease in the United States a policy statement from the American heart association. *Circulation*. 123:933-944.
7. Gheorghide, M, Vaduganathan, M, Fonarow, GC, Bonow, RO. 2013. Rehospitalization for heart failure: Problems and perspectives. *Journal of the American College of Cardiology*. 61:391-403.
8. Keenan, PS, Normand, SLT, Lin, Z, Drye, EE, Bhat, KR, Ross, JS, ... Wang, Y. 2008. An administrative claims measure suitable for profiling hospital performance on the basis of 30-day all-cause readmission rates among patients with heart failure. *Circulation: Cardiovascular Quality and Outcomes*. 1:29-37.
9. O'Connor, CM, Miller, AB, Blair, JE, Konstam, MA, Wedge, P, Bahit, MC, ... Oren, RM. 2010. Causes of death and rehospitalization in patients hospitalized with worsening heart failure and reduced left ventricular ejection fraction: Results from efficacy of vasopressin antagonism in heart failure outcome study with Tolvaptan (EVEREST) program. *American Heart Journal*. 159:841-849.
10. Eckel, RH, & Nutrition Committee. 1997. Obesity and Heart Disease: A statement for healthcare professionals from the nutrition committee, American Heart Association. *Circulation*. 96: 3248-3250.

11. Krantz, DS, Manuck, SB. 1984. Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodologic critique. *Psychological Bulletin*. 96:435-464.
12. U.S. Department of Health and Human Services (USDHSS), Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. 2014. *The Health Consequences of Smoking: 50 Years of Progress. A Report of the Surgeon General*. Rockville, MD: USDHSS.  
<https://www.surgeongeneral.gov/library/reports/50-years-of-progress/exec-summary.pdf>
13. MacMahon, KA, Lip GH. 2002. Psychological factors in heart failure: A review of the literature. *Archives of Internal Medicine*. 62:509-516.
14. Rutledge, T, Reis, VA, Linke, SE, Greenberg, BH, Mills, PJ. 2006. Depression in heart failure: A meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *Journal of the American College of Cardiology*. 48:1527-1537.
15. Xu, J, Murphy, S, Kochanek, K, Bastian, B. 2016. Deaths: Final Data for 2013. *National Vital Statistics Reports*. 64:1-118.
16. Centers for Disease Control and Prevention (CDC). 2011. Prevalence of coronary heart disease--United States, 2006-2010. *MMWR: Morbidity and Mortality Weekly Report*. 60:1377-1412.
17. Bui, AL, Horwich, TB, Fonarow, GC. 2011. Epidemiology and risk profile of heart failure. *Nature Reviews Cardiology*. 8:30-41.
18. Kim, HN, Januzzi, JL. 2011. Natriuretic peptide testing in heart failure. *Circulation*. 123:2015-2019.
19. Van Tassel, BW, Raleigh, JMV, Abbate, A. 2015. Targeting interleukin-1 in heart failure and inflammatory heart disease. *Current Heart Failure Reports*. 12:33-41.
20. Stenvinkel, P, Ketteler, M, Johnson, RJ, Lindholm, B, Pecoits-Filho, R, Riella, M, ... Girndt, M. 2005. IL-10, IL-6, and TNF- $\alpha$ : central factors in the altered cytokine network of uremia—the good, the bad, and the ugly. *Kidney International*. 67:1216-1233.
21. Henkel, DM, Redfield, MM, Weston, SA, Gerber, Y, Roger, VL. 2008. Death in heart failure a community perspective. *Circulation: Heart Failure*. 1:91-97.
22. Blecker, S, Paul, M, Taksler, G, Ogedegbe, G, Katz, S. 2013. Heart failure—associated hospitalizations in the United States. *Journal of the American College of Cardiology*. 61:1259-1267.

23. Kassel, JD, Stroud, LR, Paronis, CA. 2003. Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychological Bulletin*. 129:270-304.
24. Allan R. 2012. The Evolution of Cardiac Psychology. In *Heart and Mind*, ed. R Allan, J Fisher, pp. 3-11. Washington, DC: American Psychological Association
25. Williams, JE, Paton, CC, Siegler, IC, Eigenbrodt, ML, Nieto, FJ, Tyroler, HA. 2000. Anger proneness predicts coronary heart disease risk prospective analysis from the Atherosclerosis Risk in Communities (ARIC) study. *Circulation*. 101:2034-2039.
26. American Heart Association. 2016. *Classes of Heart Failure*. Dallas, TX: American Heart Association.  
[http://www.heart.org/HEARTORG/Conditions/HeartFailure/AboutHeartFailure/Classes-of-Heart-Failure\\_UCM\\_306328\\_Article.jsp#.VsDI-Db5NUQ](http://www.heart.org/HEARTORG/Conditions/HeartFailure/AboutHeartFailure/Classes-of-Heart-Failure_UCM_306328_Article.jsp#.VsDI-Db5NUQ)
27. American Heart Association. 2016. *About Cholesterol*. Dallas, TX: American Heart Association.  
[http://www.heart.org/HEARTORG/Conditions/Cholesterol/AboutCholesterol/Good-vs-Bad-Cholesterol\\_UCM\\_305561\\_Article.jsp#.V1k49VdWLBE](http://www.heart.org/HEARTORG/Conditions/Cholesterol/AboutCholesterol/Good-vs-Bad-Cholesterol_UCM_305561_Article.jsp#.V1k49VdWLBE)
28. American Heart Association. 2016. *Understanding Blood Pressure Readings*. Dallas, TX: American Heart Association.  
[http://www.heart.org/HEARTORG/Conditions/HighBloodPressure/KnowYourNumbers/Understanding-Blood-Pressure-Readings\\_UCM\\_301764\\_Article.jsp#.WgRk78bMyRs](http://www.heart.org/HEARTORG/Conditions/HighBloodPressure/KnowYourNumbers/Understanding-Blood-Pressure-Readings_UCM_301764_Article.jsp#.WgRk78bMyRs)
29. Wilson, PW, D'Agostino, RB, Levy, D, Belanger, AM, Silbershatz, H, Kannel, WB. 1998. Prediction of coronary heart disease using risk factor categories. *Circulation*. 97:1837-1847.
30. Flegal, KM, Carroll, MD, Kit, BK, Ogden, CL. 2012. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. *Jama*. 307:491-497.
31. Poirier, P, Giles, TD, Bray, GA, Hong, Y, Stern, JS, Pi-Sunyer, FX, Eckel, RH. 2006. Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss an update of the 1997 American Heart Association Scientific statement on obesity and heart disease from the obesity committee of the council on nutrition, physical activity, and metabolism. *Circulation*. 113:898-918
32. Booth, F.W, Roberts, CK, Laye, MJ. 2012. Lack of exercise is a major cause of chronic diseases. *Comprehensive Physiology*. 2:1143-1211
33. Ladabaum, U, Mannalithara, A, Myer, PA, Singh, G. 2014. Obesity, abdominal obesity, physical activity, and caloric intake in US adults: 1988 to 2010. *The American Journal of Medicine*, 127:717-727.

34. Sesso, HD, Paffenbarger, RS, Lee, IM. 2000. Physical activity and coronary heart disease in men the Harvard Alumni Health Study. *Circulation*. 102: 975-980.
35. Huang, CJ, Webb, HE, Zourdos, MC, Acevedo, EO. 2013. Cardiovascular reactivity, stress, and physical activity. *Frontiers in Physiology*. 4:1-13.
36. Edwards, N, Gardiner, M, Ritchie, DM, Baldwin, K, Sands, L. 2008. Effect of exercise on negative affect in residents in special care units with moderate to severe dementia. *Alzheimer Disease & Associated Disorders*. 22:362-368
37. Heran, BS, Chen, JM, Ebrahim, S, Moxham, T, Oldridge, N, Rees, K, ... Taylor, RS. 2011. Exercise-based cardiac rehabilitation for coronary heart disease. *Cochrane Database of Systematic Reviews*, 7:1-73.
38. Folkman, S, Lazarus, RS, Dunkel-Schetter, C, DeLongis, A, Gruen, RJ. 1986. Dynamics of a stressful encounter: Cognitive appraisal, coping, and encounter outcomes. *Journal of Personality and Social Psychology*. 50:992-1003.
39. Krantz, DS, Whittaker, KS, Sheps, DS. 2012. Psychosocial Risk Factors for Coronary Heart Disease: Pathophysiologic Mechanisms. In *Heart and Mind* ed. R Allan, J Fisher, pp. 91-105. Washington, DC: American Psychological Association
40. Cohen, S, Kessler, RC, Gordon LU. 1995. Strategies for measuring stress in studies of psychiatric and physical disorders. In *Measuring Stress: A Guide for Health and Social Scientists* ed. S Cohen, R Kessler, L Gordon, pp. 3-25. Oxford, NY: Oxford University Press.
41. Rozanski, A, Blumenthal, JA, Kaplan, J. 1999. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*. 99:2192-2217.
42. Kupper, N, Denollet, J, Widdershoven, J, Kop, WJ. 2015. Cardiovascular reactivity to mental stress and mortality in patients with heart failure. *JACC: Heart Failure*. 3:373-382.
43. Krueger, PM, Chang, VW. 2008. Being poor and coping with stress: health behaviors and the risk of death. *American Journal of Public Health*. 98:889-896.
44. Smith, TW, Traupman, EK. 2012. Anger, Hostility, and Aggressiveness in Coronary Heart Disease: Clinical Applications of an Interpersonal Perspective. In *Heart and Mind* ed. R Allan, J. Fisher, pp. 91-105. Washington, DC: American Psychological Association
45. Diamond, EL. 1982. The role of anger and hostility in essential hypertension and coronary heart disease. *Psychological Bulletin*. 92:410-433.

46. Chida, Y, Steptoe, A. 2009. The association of anger and hostility with future coronary heart disease: A meta-analytic review of prospective evidence. *Journal of the American College of Cardiology*. 53:936-946.
47. Moser, DK, Dracup, K, Evangelista, LS, Zambroski, CH, Lennie, TA, Chung, ML, ... Heo, S. 2010. Comparison of prevalence of symptoms of depression, anxiety, and hostility in elderly patients with heart failure, myocardial infarction, and a coronary artery bypass graft. *Heart & Lung: The Journal of Acute and Critical Care*. 39:378-385.
48. Guerrero, C, & Palmero, F. 2010. Impact of defensive hostility in cardiovascular disease. *Behavioral Medicine*. 36:77-84.
49. Jenner, RC, Schweitzer, RD, Strodl, ES. 2009. Anger and depression predict hospital use among chronic heart failure patients. *Australian Health Review*. 33:541-548.
50. Keith, F. 2016. Behaviors and Cognitions as Mediators of Psychosocial Variables and Re-hospitalizations in Patients with Heart Failure: A Structural Equation Modeling Approach. PhD Thesis, Uniformed Services University of the Health Sciences, Bethesda, MD.
51. Suls, J, Bunde, J. 2005. Anger, Anxiety, and Depression as Risk Factors for Cardiovascular Disease: The Problems and Implications of Overlapping Affective Dispositions. *Psychological Bulletin*. 131:260-300.
52. Kucharska-Newton, AM, Williams, JE, Chang, PP, Stearns, SC, Sueta, CA, Blecker, SB, Mosley, TH. 2014. Anger proneness, gender, and the risk of heart failure. *Journal of Cardiac Failure*. 20:1020-1026.
53. Kubzansky, LD, Cole, SR, Kawachi, I, Vokonas, P, Sparrow, D. 2006. Shared and unique contributions of anger, anxiety, and depression to coronary heart disease: A prospective study in the normative aging study. *Annals Of Behavioral Medicine: A Publication Of The Society Of Behavioral Medicine*. 31:21-29.
54. Rafanelli, C, Gostoli, S, Tully, PJ, Roncuzzi, R. 2016. Hostility and the clinical course of outpatients with congestive heart failure. *Psychology & Health*. 31:228-238.
55. Mostofsky, E, Penner, EA, & Mittleman, MA. 2014. Outbursts of anger as a trigger of acute cardiovascular events: a systematic review and meta-analysis. *European Heart Journal*. 35:1404-10
56. Steptoe, A, Brydon, L. 2009. Emotional triggering of cardiac events. *Neuroscience & Biobehavioral Reviews*. 33:63-70.

57. Kubzansky, LD, Kawachi, I, Spiro, A, Weiss, ST, Vokonas, PS, Sparrow, D. 1997. Is worrying bad for your heart? A prospective study of worry and coronary heart disease in the Normative Aging Study. *Circulation*. 95:818-824.
58. Roest, AM, Martens, EJ, de Jonge, P, Denollet, J. 2010. Anxiety and risk of incident coronary heart disease: A meta-analysis. *Journal of the American College of Cardiology*. 56:38-46.
59. Konstam, V, Moser, DK, De Jong, MJ. 2005. Depression and anxiety in heart failure. *Journal of Cardiac Failure*. 11:455-463.
60. Cohen, BE, Edmondson, D, Kronish, IM. 2015. State of the art review: depression, stress, anxiety, and cardiovascular disease. *American Journal of Hypertension*. 28:1295-1302.
61. Whooley, MA, Caska, CM, Hendrickson, BE, Rourke, MA, Ho, J, & Ali, S. 2007. Depression and inflammation in patients with coronary heart disease: Findings from the Heart and Soul Study. *Biological Psychiatry*. 62:314-320.
62. Kop, WJ, Synowski, SJ, Gottlieb, SS. 2011. Depression in heart failure: Biobehavioral Mechanisms. *Heart Failure Clinics*. 7: 23-38.
63. Jiang, W, Alexander, J, Christopher, E, Kuchibhatla, M, Gauden, LH, Cuffe, M S, ... O'Connor, CM. 2001. Relationship of depression to increased risk of mortality and rehospitalization in patients with congestive heart failure. *Archives of Internal Medicine*. 161:1849-1856.
64. Fredrickson, BL. 2001. The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist*. 56:218-226.
65. Cohen, S, Doyle, WJ, Turner, RB, Alper, CM, Skoner, DP. 2003. Emotional style and susceptibility to the common cold. *Psychosomatic Medicine*. 65:652-657.
66. Pressman, SD, Cohen, S. 2005. Does positive affect influence health? *Psychological Bulletin*. 131:925-971.
67. Uchino, BN, Cacioppo, JT, Kiecolt-Glaser, JK. 1996. The relationship between social support and physiological processes: a review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin*. 119:488-531.
68. Cohen, S, Wills, TA. 1985. Stress, social support, and the buffering hypothesis. *Psychological Bulletin*. 98:310-357.
69. Barth, J, Schneider, S, von Känel, R. 2010. Lack of social support in the etiology and the prognosis of coronary heart disease: a systematic review and meta-analysis. *Psychosomatic Medicine*. 72:229-238.

70. Weiss-Faratici, N, Lurie, I, Neumark, Y, Malowany, M, Cohen, G, Benyamini, Y, ... Gerber, Y. 2016. Perceived social support at different times after myocardial infarction and long-term mortality risk: a prospective cohort study. *Annals of Epidemiology*. 26:424-428.
71. Aspinwall LG, Tedeschi RG. 2010. The value of positive psychology for health psychology: Progress and pitfalls in examining the relation of positive phenomena to health. *Annals of Behavioral Medicine*. 39:4-15.
72. Seligman, ME. 2008. Positive health. *Applied Psychology*. 57:3-18.
73. Huffman, JC, Mastromauro, CA, Boehm, JK, Seabrook, R, Fricchione, GL, Denninger, JW, Lyubomirsky, S. 2011. Development of a positive psychology intervention for patients with acute cardiovascular disease. *Heart International*. 6:47-52.
74. Brissette, I, Scheier, MF, Carver, CS. 2002. The role of optimism in social network development, coping, and psychological adjustment during a life transition. *Journal of Personality and Social Psychology*, 82:102-111.
75. Giltay, EJ, Kamphuis, M. H., Kalmijn, S., Zitman, F. G., & Kromhout, D. (2006). Dispositional optimism and the risk of cardiovascular death: the Zutphen Elderly Study. *Archives of Internal Medicine*. 166:431-436.
76. Kim, ES, Smith, J, Kubzansky, LD. 2014. Prospective study of the association between dispositional optimism and incident heart failure. *Circulation: Heart Failure*. 7:394-400.
77. Janicki-Deverts, D, Cohen, S, Doyle, WJ, Turner, RB, Treanor, JJ. 2007. Infection-induced proinflammatory cytokines are associated with decreases in positive affect, but not increases in negative affect. *Brain, Behavior, and Immunity*. 21:301-307.
78. Watson, D, Clark, LA, Tellegen, A. 1988. Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology*. 54: 1063-1070.
79. Chida, Y, Steptoe, A. 2008. Positive psychological well-being and mortality: A quantitative review of prospective observational studies. *Psychosomatic Medicine*. 70:741-756.
80. Watson, D, Pennebaker, JW. 1989. Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review*. 96:234-254.
81. Nabi, H, Kivimaki, M, De Vogli R, Marmot MG, Singh-Manoux, A. 2008. Positive and negative affect and risk of coronary heart disease: Whitehall II prospective cohort study. *British Medical Journal*. 337:1-7.

82. Meyer, FA, von Känel, R, Saner, H, Schmid, JP, Stauber, S. 2015. Positive affect moderates the effect of negative affect on cardiovascular disease-related hospitalizations and all-cause mortality after cardiac rehabilitation. *European Journal of Preventive Cardiology*. 22:1247-1253.
83. Shiffman, S, Waters, AJ. (2004). Negative Affect and Smoking Lapses: A Prospective Analysis. *Journal Of Consulting And Clinical Psychology*. 72:192-201.
84. Leventhal, AM, Greenberg, JB, Trujillo, MA, Ameringer, KJ, Lisha, NE, Pang, RD, Monterosso, J. 2013. Positive and negative affect as predictors of urge to smoke: Temporal factors and mediational pathways. *Psychology Of Addictive Behaviors*. 27:262-267.
85. Cooper, ML, Frone, MR, Russell, M, Mudar, P. 1995. Drinking to regulate positive and negative emotions: A motivational model of alcohol use. *Journal of Personality and Social Psychology*. 69:990-1005.
86. Urbano-Marquez, A, Estruch, R, Navarro-Lopez, F, Grau, JM, Mont, L, Rubin, E. 1989. The effects of alcoholism on skeletal and cardiac muscle. *New England Journal of Medicine*. 320:409-415.
87. Roerecke, M., & Rehm, J. (2010). Irregular heavy drinking occasions and risk of ischemic heart disease: a systematic review and meta-analysis. *American Journal of Epidemiology*. 171:633-644.
88. Jonas, BS, Lando, JF. 2000. Negative affect as a prospective risk factor for hypertension. *Psychosomatic Medicine*. 62:188-196.
89. Pollard, TM, Schwartz, JE. 2003. Are changes in blood pressure and total cholesterol related to changes in mood? An 18-month study of men and women. *Health Psychology*. 22:47-53.
90. Ryff, CD, Dienberg Love, G, Urry, HL, Muller, D, Rosenkranz, MA, Friedman, EM, Davidson, RJ, Singer, B. 2006. Psychological well-being and ill-being: do they have distinct or mirrored biological correlates? *Psychotherapy and Psychosomatics*. 75:85-95.
91. Kiecolt-Glaser, JK, McGuire, L, Robles, TF, Glaser, R. 2002. Psychoneuroimmunology: psychological influences on immune function and health. *Journal of Consulting Clinical Psychology*. 70:537-547.
92. Beckham, JC, Vrana, SR, Barefoot, JC, Feldman, ME, Fairbank, J, Moore, SD. 2002. Magnitude and duration of cardiovascular response to anger in Vietnam veterans with and without posttraumatic stress disorder. *Journal Of Consulting And Clinical Psychology*. 70:228-234.

93. Steptoe, A, O'Donnell, K, Marmot, M, Wardle, J. 2008. Positive affect and psychosocial processes related to health. *British Journal of Psychology*. 99:211-227.
94. Watson, D, Clark, LA, Carey, G. 1988. Positive and negative affectivity and their relation to anxiety and depressive disorders. *Journal of Abnormal Psychology*. 97:346-353.
95. Moreno, PI, Moskowitz, AL, Ganz, PA, Bower, JE. 2016. Positive Affect and Inflammatory Activity in Breast Cancer Survivors: Examining the Role of Affective Arousal. *Psychosomatic Medicine*. 78:532-541.
96. Mroczek, DK, Stawski, RS, Turiano, NA, Chan, W, Almeida, DM, Neupert, SD, Spiro, A. 2015. Emotional reactivity and mortality: Longitudinal findings from the VA Normative Aging Study. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*. 70:398-406.
97. Steptoe, A, Dockray, S, Wardle, J. 2009. Positive affect and psychobiological processes relevant to health. *Journal of Personality*. 77:1747-1776.
98. Sin, NL, Moskowitz, JT, Whooley, MA. 2015. Positive affect and health behaviors across 5 years in patients with coronary heart disease: the heart and soul study. *Psychosomatic Medicine*. 77:1058-1066.
99. Bhattacharyya, MR, Whitehead, DL, Rakhit, R, Steptoe, A. 2008. Depressed mood, positive affect, and heart rate variability in patients with suspected coronary artery disease. *Psychosomatic Medicine*. 70:1020-1027.
100. Davidson, KW, Mostofsky, E, Whang, W. 2010. Don't worry, be happy: positive affect and reduced 10-year incident coronary heart disease: the Canadian Nova Scotia Health Survey. *European Heart Journal*. 31:1065-1070.
101. Tugade, MM, Fredrickson, BL, Feldman Barrett, L. 2004. Psychological resilience and positive emotional granularity: Examining the benefits of positive emotions on coping and health. *Journal of Personality*. 72:1161-1190.
102. Folkman, S, Moskowitz, JT. 2000. Positive affect and the other side of coping. *American Psychologist*. 55:647-654.
103. Piazza, JR, Charles, ST, Sliwinski, MJ, Mogle, J, Almeida, DM. 2013. Affective reactivity to daily stressors and long-term risk of reporting a chronic physical health condition. *Annals of Behavioral Medicine*. 45:110- 120.
104. Cohen, S, Kamarck, T, Mermelstein, R. 1983. A global measure of perceived stress. *Journal of health and social behavior*, 24:385-396.

105. Cohen, S, Janicki-Deverts, D. 2012. Who's stressed? Distributions of psychological stress in the United States in probability samples from 1983, 2006, and 2009. *Journal of Applied Social Psychology*. 42:1320-1334.
106. Beck, AT, Steer, RA, Brown, G. 1996. *Manual for the Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.
107. Beck, AT, Steer, RA, Ball, R, Ranieri, W. 1996. Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *Journal of Personality Assessment*. 63:588-597.
108. Richter, P., Werner, J., Heerlein, A., Kraus, A., & Sauer, H. (1998). On the validity of the Beck Depression Inventory. A review. *Psychopathology*, 31:160-168.
109. Spielberger, CD, Gorsuch, RL, Lushene, R, Vagg, PR, Jacobs, GA. 1983. *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.
110. Spielberger, CD, Reheiser, E. C. (2004). Measuring anxiety, anger, depression, and curiosity as emotional states and personality traits with the STAI, STAXI, and STPI. In *Comprehensive Handbook of Psychological Assessment* ed. MJ Hilsenroth, DL Segal, M Hersen, pp. 70-86. Hoboken, New Jersey: John Wiley & Sons, Inc.
111. Spielberger, CD, Johnson, EH, Russell, SF, Crane, RJ, Jacobs, GA, Worden, TJ. 1985. The experience and expression of anger: Construction and validation of an anger expression scale. In *Anger and Hostility in Cardiovascular Behavioral Disorder*, ed. MA Chesney, RH Rosenman, pp. 5-30. Washington, DC: Hemisphere.
112. Spielberger, CD. 1988. *Manual for the State-Trait Anger Expression Inventory (STAXI)*. Odessa, FL: Psychological Assessment Resources.
113. Cook WW, Medley, DM. 1954. Proposed hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology*. 38:414-418.
114. Barefoot, JC, Dodge, KA, Peterson, BL, Dahlstrom, WG, Williams, RB. 1989. The Cook–Medley Hostility Scale: Item content and ability to predict survival. *Psychosomatic Medicine*. 51:46–57.
115. Smith, TW, Frohm, KD. 1985. What's so unhealthy about hostility? Construct validity and psychosocial correlates of the Cook and Medley Ho scale. *Health Psychology*. 4:503-520.
116. Scheier, MF, Carver, CS. 1985. Optimism, coping, and health: Assessment and implications of generalized outcome expectancies. *Health Psychology*. 4:219-247.

117. Cohen, S, Hoberman, HM. 1983. Positive events and social supports as buffers of life change stress. *Journal of Applied Social Psychology*. 13:99-125.
118. Cohen, S, Mermelstein, R, Kamarck, T, Hoberman, HM. 1985. Measuring the functional components of social support. In *Social support: Theory, research and applications* (pp. 73-94). Springer Netherlands.
119. Merz, EL, Roesch, SC, Malcarne, VL, Penedo, FJ, Llabre, MM, Weitzman, OB, ... Johnson, TP. 2014. Validation of interpersonal support evaluation list-12 (ISEL-12) scores among English-and Spanish-speaking Hispanics/Latinos from the HCHS/SOL Sociocultural Ancillary Study. *Psychological Assessment*. 26:384-394.
120. Payne, TJ, Andrew, M, Butler, KR, Wyatt, SB, Dubbert, PM, Mosley, TH. 2012. Psychometric evaluation of the Interpersonal Support Evaluation List–Short Form in the ARIC study cohort. *Sage Open*. 2:1-12.
121. Green, CP, Porter, CB, Bresnahan, DR, Spertus, JA. 2000. Development and evaluation of the KCCQ: A new health status measure for heart failure. *Journal of the American College of Cardiology*. 35:1245-1255.
122. Spertus, JA, Jones, PG, Kim, J, Globe, D. 2008. Validity, reliability, and responsiveness of the Kansas City Cardiomyopathy Questionnaire in anemic heart failure patients. *Quality of Life Research*. 7:291-298.
123. Baron, RM, Kenny, DA. 1986. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology*. 51:1173-1182.
124. Faul, F, Erdfelder, E, Buchner, A, Lang, AG. 2009. Statistical power analyses using G\*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods*. 41:1149-1160.
125. Brown, TA, Chorpita, BF, Barlow, DH. 1998. Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology*. 107:179-192.
126. Kashdan, TB, Weeks, JW, Savostyanova, AA. 2011. Whether, how, and when social anxiety shapes positive experiences and events: A self-regulatory framework and treatment implications. *Clinical Psychology Review*. 31:786-799.
127. Eisner, LR, Johnson, SL, Carver, CS. 2009. Positive affect regulation in anxiety disorders. *Journal of Anxiety Disorders*. 23:645-649.
128. Schiffrin, HH, Nelson, SK. 2010. Stressed and happy? Investigating the relationship between happiness and perceived stress. *Journal of Happiness Studies*. 11:33-39.

129. Pressman, SD, Cohen, S. 2012. Positive emotion word use and longevity in famous deceased psychologists. *Health Psychology*. 31:297-305.
130. Brouwers, C, Mommersteeg, PM, Nyklíček, I, Pelle, AJ, Westerhuis, BL, Szabó, BM, Denollet, J. 2013. Positive affect dimensions and their association with inflammatory biomarkers in patients with chronic heart failure. *Biological Psychology*. 92:220-226.
131. Laederach-Hofmann, K, Roher-Gubeli, R, Messerli, N, Meyer, K. 2007. Comprehensive rehabilitation in chronic heart failure-Better psycho-emotional status related to quality of life, brain natriuretic peptide concentrations, and clinical severity of disease. *Clinical & Investigative Medicine*. 30:54-62.
132. Celano, CM, Huffman, JC. 2011. Depression and cardiac disease: a review. *Cardiology In Review*. 19:130-142.
133. Zawadzki, M, Smyth, J, Sliwinski, M, Ruiz, J, Gerin, W. 2017. Revisiting the lack of association between affect and physiology: Contrasting between-person and within-person analyses. *Health Psychology*. 36:811-818.
134. Assari, S, Lankarani, MM. 2016. Chronic Medical conditions and negative affect; racial Variation in reciprocal associations over time. *Frontiers In Psychiatry*. 7:1-8.
135. Çivitci, A. 2015. The Moderating Role of Positive and Negative Affect on the Relationship between Perceived Social Support and Stress in College Students. *Educational Sciences: Theory and Practice*. 15:565-573.
136. Chen, AM, Yehle, KS, Albert, NM, Ferraro, KF, Mason, HL, Murawski, MM, Plake, KS. 2014. Relationships between health literacy and heart failure knowledge, self-efficacy, and self-care adherence. *Research in Social and Administrative Pharmacy*. 10:378-386.
137. Moè, A, Pazzaglia, F, Ronconi, L. 2010. When being able is not enough. The combined value of positive affect and self-efficacy for job satisfaction in teaching. *Teaching and Teacher Education*. 26:1145-1153.
138. Yu, Y, Peng, L, Tang, T, Chen, L, Li, M, Wang, T. 2014. Effects of emotion regulation and general self-efficacy on posttraumatic growth in Chinese cancer survivors: assessing the mediating effect of positive affect. *Psycho-Oncology*. 23:473-478.
139. Arnold R, Ranchor AV, DeJongste MJ, Köeter, GH, Ten Hacken NH, Aalbers R, Sanderman R. 2005. The relationship between self-efficacy and self-reported physical functioning in chronic obstructive pulmonary disease and chronic heart failure. *Behavioral Medicine*. 31:107-115.

140. Saab KR, Kendrick J, Yracheta JM, Lanaspá MA, Pollard M, Johnson RJ. 2015. New Insights on the Risk for Cardiovascular Disease in African Americans: The Role of Added Sugars. *Journal of the American Society of Nephrology : JASN*. 26:247-257.
141. Hopper, I, Kotecha, D, Chin, KL, Mentz, RJ, von Lueder, TG. 2016. Comorbidities in Heart Failure: Are There Gender Differences? *Current Heart Failure Report*. 13:1-12.
142. Riedinger, MS, Dracup, KA, Brecht, ML, Padilla, G, Sarna, L, Ganz, PA. 2001. Quality of life in patients with heart failure: do gender differences exist? *Heart & Lung: The Journal of Acute and Critical Care*. 30:105-116.
143. Fujita, F, Diener, E, Sandvik, E. 1991. Gender differences in negative affect and well-being: The case for emotional intensity. *Journal of Personality and Social Psychology*. 61:427-434.
144. Endrighi, R, Waters, AJ, Gottlieb, SS, Harris, KM, Wawrzyniak, AJ, Bekkouche, NS, Yisheng, L, Willem, JK, Krantz, DS. 2016. Psychological stress and short-term hospitalisations or death in patients with heart failure. *Heart*.