STRESS, ANXIETY, AND HEART RATE VARIABILITY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

DISSERTATION

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By
Sooyeon Suh, M.A.
Graduate Program in Psychology

The Ohio State University
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Dissertation Committee:
Dr. Charles F. Emery, Adviser
Dr. Steven Beck
Dr. Julian Thayer
Dr. Michael Vasey
ABSTRACT

Elevated levels of anxiety and high prevalence of anxiety disorders have been observed among patients with Chronic Obstructive Pulmonary Disease (COPD). Decreased heart rate variability has been associated with COPD and poor emotional regulation, but there have not been any prior studies investigating decreased heart rate variability (HRV) as an underlying mechanism that may partially explain the high levels of anxiety in COPD patients. The current study utilized a 2 (disease status) X 2 (anxiety group) factorial design with four groups (COPD, COPD-ANX, HEA, HEA-ANX) examining heart rate variability associated with anxiety symptoms in COPD patients compared to healthy controls when exposed to a modified Trier Social Stress Task. Participants were evaluated with pulmonary function tests, HRV monitoring, and self-report questionnaires assessing health behaviors, psychological factors, and dispositional variables. 30 COPD patients were age- and gender-matched with 30 healthy controls. Mean age was 59.1 (±11.2) years and 50% of the participants were female. Participants with COPD presented with severe airway obstruction [mean forced expiratory volume in one second / forced vital capacity ratio (FEV₁/FVC) = 0.63 (± 0.15)].

There were no significant differences in resting HRV between groups.
Repeated measures ANOVA revealed that the COPD-ANX and HEA-ANX groups displayed a blunted response to a stressor. Although this study was unable to distinguish HRV differences unique to the COPD population, HRV responses to stress in anxious individuals with and without COPD were blunted. Future studies of COPD patients and healthy controls investigating HRV should measure anxiety to avoid the potential confound of high anxiety levels among patients with COPD. Results revealed that the COPD-ANX group had significantly lower psychological well-being and poorer sleep quality than the COPD group. Furthermore, individuals in the COPD-ANX group with pre-existing anxiety prior to their COPD diagnosis had lower resting vagal tone. Future studies should focus on distinguishing between individuals who have preexisting anxiety prior to their COPD diagnosis compared to individuals who developed anxiety after their COPD diagnosis.
Dedication

This document is dedicated to my family,

Thank you for all the support and love.
Acknowledgments

I gratefully recognize my advisor, Dr. Charles F. Emery, for his intellectual and emotional support which made this dissertation possible. Your knowledge, wisdom, support, guidance, editorial skills, and patience has built a strong foundation for my career and I will always be indebted to you.

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Vita

November 10, 1980.................................................................Born in Seoul, Korea

2003...........................................................................B.A. in Psychology
Korea University
Seoul, Korea

2003 – 2006.................................................................M.A. in Psychology
The Ohio State University, Columbus

2003 – 2009.............................................................Departmental Fellow
The Ohio State University, Columbus

2003 – 2004.........................................................Graduate Course Assistant
The Ohio State University, Columbus

2004 – 2007, 2008 – 2009...............................Graduate Teaching Associate
The Ohio State University, Columbus

2006.................................................................Recipient
Ray Travel Award
The Ohio State University, Columbus

2007.................................................................Recipient
Alumni Grants for Graduate Research and Scholarship
The Ohio State University, Columbus

2007.................................................................Recipient
Office of International Education Grant
The Ohio State University, Columbus

2009 – 2010.................................................................Clinical Psychology Intern
Rush University Medical Center
Chicago, IL
Publications


Fields of Study

Major Field: Psychology
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PSS-10

PSQW

RRS

PANAS

STAI-X1

STAI-X2

CESD

PSQI

Houston Non-Exercise Test

MCSD

ACS
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CHAPTER 1
INTRODUCTION

Chronic Obstructive Pulmonary Disease

Chronic Obstructive Pulmonary Disease (COPD) is currently the fourth most common cause of death in the U.S. and accounted for more than 122,283 deaths in 2003 (Mannino et al., 2000; American Lung Association, 2007). Currently, COPD affects over 17 million people in the U.S. and up to 600 million people worldwide, making it one of the leading causes of chronic morbidity and mortality in the world (Mannino et al., 2000; Global initiative for chronic obstructive lung disease, 2003). Direct costs for COPD are estimated to be over 18 billion dollars annually (Gross et al., 1999; Sullivan et al., 2000; Sin et al., 2002). In the United States during 2000, COPD was responsible for 8 million physician office and hospital outpatient visits, 1.5 million emergency department visits, 726,000 hospitalizations, and 119,000 deaths (Mannino et al., 2002). COPD also has an adverse impact on work force participation (Sin et al., 2002). Based on the Third National Health and Nutrition Examination Survey (NHANES III), it was estimated that in 1994, COPD was responsible for 9.9 billion dollars in lost work. Additionally, this survey demonstrated that severe COPD was estimated to be responsible for 14.4% of reduction in the labor force participation rate compared to those without COPD in the U.S. (Sin et al., 2002). In the U.S.,
COPD is the second leading cause of disability among the working population (Post and Collins, 1981 – 2). Between 1980 and 2000, there was an overall death rate increase of 67% due to COPD. COPD is currently the only major disease among the top five causes of death that is rising in prevalence and in mortality rates (Mannino et al., 2000).

Pathology of COPD

COPD is a medical condition characterized by expiratory airflow obstruction (Cugell, 1988; American Lung Association, 2007). It is a progressively deteriorating disease that is irreversible, with clinical manifestations of dyspnea, cough, wheezing, and mucus production. COPD is a diagnosis that encompasses both emphysema and chronic bronchitis. Although chronic bronchitis and emphysema have different pathophysiologies, patients often experience symptoms of both diseases, and COPD is the diagnosis incorporating both emphysema and chronic bronchitis.

Chronic bronchitis is characterized by inflammation and scarring of the lining of the bronchial tubes. Inflammation of the bronchi contributes to mucus and phlegm in the airways and reduces the size of airway passages (American Lung Association, 2007). Scarring of the bronchial tubes often causes bacterial infections within the airways, which may contribute to further inflammation and obstruction of airflow. Individuals who are diagnosed with chronic bronchitis most often manifest symptoms of chronic coughing, increased mucus production, and shortness of breath (American Thoracic Society, 1995).

Emphysema is characterized by the destruction of alveoli (air sacs) in the
lungs, where oxygen is exchanged with carbon dioxide in the blood. Damage to
the alveoli limits oxygen exchange in the bloodstream. The walls of the alveoli
lose their elasticity, causing the air spaces to get larger and air to get trapped
(American Lung Association, 2007). Individuals who are diagnosed with
dyspnea usually report dyspnea being the major physical complaint, with
emphysema patients do not report as many symptoms of coughing or sputum
production.

The primary cause of COPD is heavy cigarette smoking. Most patients
who have been diagnosed with COPD report a long history of regular cigarette
use. Other contributing factors include occupational exposure to pollutants, air
pollution, passive smoke exposure, respiratory virus infection, low socioeconomic
status, poor nutrition, alcohol ingestion, advancing age, gender, mucus
hypersecretion, and airway hyperresponsiveness (Rennard, 1998). In addition,
individuals with a congenital absence of the alpha-1-protease inhibitor in blood
serum may develop COPD (Cugell, 1988).

Clinical Manifestations of COPD

COPD affects many areas of the patient’s life as the illness progresses.
The initial stages of the illness are characterized by shortness of breath, which
later may lead to the use of supplemental oxygen and other mechanical
respiratory assistance as symptoms worsen (Global Initiative for COPD, 2003). A
survey by the American Lung Association revealed that in addition to limiting the
patient’s ability to work, COPD also limits normal physical exertion, household chores, social activities, sleeping, and recreational activities (McSweeny et al., 1982; Prigatano et al., 1984; American Lung Association, 2003).

COPD also may be associated with cognitive or neuropsychological decline which, in turn, affects ability to engage in daily activities. Data suggest that cognitive impairment is associated with hypoxemia (Grant et al., 1987). Areas of cognitive impairment may include short-term memory, psychomotor speed, attention, deductive thinking, and visual integration (Incalzi et al., 1993; Incalzi et al., 1997; Incalzi et al., 1998; Incalzi et al., 2003; Kass et al., 1975; Prigatano et al., 1983; Reeves et al., 1999).

Studies have documented effects of COPD on emotional well-being, with higher rates of emotional distress among COPD patients than in healthy adults. Studies have found that 58% of COPD patients may suffer from a psychiatric disorder (Yellowlees, 1987). The most common psychiatric disorder found in COPD patients is anxiety (Kim et al., 2000; Mikkelsen et al., 2004). Rates of anxiety disorders in patients with COPD vary across studies, revealing that up to 67% of COPD patients may meet diagnostic criteria for an anxiety disorder (Mikkelsen et al., 2004; Hynninen et al., 2005). There appears to be a substantial overrepresentation of anxiety in COPD patients, as evidenced by higher rates of generalized anxiety disorder (GAD) and panic disorder in COPD patients compared to the general public (Brenes, 2003). Prevalence of GAD among patients with COPD ranges from 10 – 15.8% when using standard diagnostic procedures compared with lifetime rates of 3.6 – 5.1% in the general population.
Thus, GAD is approximately three times more common in COPD patients than in the general U.S. population. Rates of panic disorder are also higher in this population, ranging from 8 – 32% for COPD patients, in contrast to 1.5 – 3.5% in the general population (Ayres et al., 1982; Robins et al., 1984; Karajgi et al., 1990; Rabinowitz, 1992; Pollack et al., 1996; Moore & Zebb, 1999). Porzelius et al. (1992) found that 37% of COPD patients reported a panic attack in the previous three weeks. In recent years, several studies have suggested that panic disorder is more highly associated with COPD than any of the other anxiety disorders (Rabinowitz, 1992). Emotional distress associated with COPD may also, in turn, exacerbate symptoms such as dyspnea, which may lead to panic (Fuhs, 1980; Burns & Howell, 1969). A recent study by Livermore et al. (2008) compared COPD patients with panic attacks or panic disorder to COPD patients without panic attacks or panic disorder and healthy age-matched controls using an inspiratory resistive load-testing protocol. Results revealed that patients with COPD with panic attacks showed heightened sensitivity to inspiratory loads, despite no differences found between groups on respiratory variables. Patients with COPD with panic attacks or panic disorder rated higher levels of subjective dyspnea compared to the other groups.

Psychological factors such as mood, attitudes, and beliefs may predict functional ability. Patients with both chronic medical illness and comorbid mental illness are more functionally disabled than patients who have either a chronic medical illness or a mental illness alone (Borson et al., 1992; Unutzer et al.,
1997). Also, patients who feel better about themselves, their situation, and the future will participate more actively in therapies and will subsequently benefit more, leading to better physiological outcomes (McSweeny, 1988). Thus, the patient’s functional performance is not solely related to physical symptoms, and several studies have suggested a relationship between psychiatric comorbidity and functional status among patients with COPD (McSweeny, 1988; Hynninen et al., 2005). For example, a study by Kim et al. (2000) found that symptoms of anxiety and depression explained more of the overall variance in functional status than did COPD severity and medical burden. Similarly, Beck et al. (1988) found that reduced activities of daily living were more consistently associated with psychological well-being than with illness severity.

Data suggest high comorbidity of anxiety disorders and COPD. Both illnesses share common symptoms, such as dyspnea and hyperventilation. The lifetime prevalence of respiratory disease is estimated to be approximately 47% in individuals who suffer from panic disorder, a higher rate of respiratory disease than in any other psychiatric diagnosis (Zandbergen et al., 1991). However, there have been few theoretical models proposed to explain mechanisms that may contribute to high comorbidity between anxiety and COPD. One model has suggested that hypoxemia, a symptom of COPD, may lead to structural changes in the brain that may cause neurocognitive deficits and impairment in mood regulation via alterations in the neurotransmitter systems (Borson, Claypoole, & McDonald, 1998). Repeated experiences of hypoxia may lead to sensitization of the brain circuitry involved in the control of fear responses to overreact to
subsequent episodes of hypoxia and hypercapnia or anxiety-associated shortness of breath (Sanders, Morzorati, Shekhar, 1995). Another model posits that hyperventilation in COPD patients is developed as a method to compensate for the patient’s hypercapnic states. However, hyperventilation also has counterproductive effects, such as airway narrowing and increased respiratory distress (Decramer et al., 2008).

The most widely embraced model to date is the cognitive-behavioral model, which proposes that COPD patients may misinterpret bodily sensations arising from dyspnea and hyperventilation as negative indicators of functioning, and the increased sensitivity to physiological arousal catalyzes a panic reaction, which, in turn, worsens the respiratory symptoms (Nutt et al., 1999). However, the research to date is inconclusive regarding the extent to which physiological and psychological factors are important in the development and maintenance of anxiety in COPD patients.

These three models have attempted to explain the high prevalence of anxiety in COPD patients as a result of COPD symptoms. This is a possible explanation, as anxiety symptoms have been observed in association with disease characteristics of COPD, including reduced forced vital capacity (FVC), chest pain, and dyspnea (Kellner et al., 1992; Borak et al., 1998; Gift & Cahill, 1990; Gift et al., 1990). However, it is possible that COPD and anxiety disorders may share a similar psychophysiological patholology that may explain the high prevalence of anxiety in the COPD population.

**Autonomic Dysregulation**
Disorders of affect such as anxiety disorders have been viewed as distorted emotional states in which an individual is not able to respond in an appropriate, flexible way to environmental demands (Friedman & Thayer, 1998; Thayer & Friedman, 1997). It has been suggested that when individuals respond to environmental demands in non-adaptive ways, it is not due to difficulty selecting an appropriate response, but more often due to inability to inhibit inappropriate responses (Thayer & Lane, 2000).

Consistent with this idea, Thayer and Lane (2000) have proposed an integrated model associated with autonomic nervous system dysregulation to explain a wide range of physical and psychological illnesses. The autonomic nervous system (ANS) is divided into two major branches – the sympathetic nervous system and the parasympathetic nervous system. The sympathetic nervous system functions primarily to rapidly activate bodily systems to meet threats or emergencies, eliciting a cascade of physiological responses that increase the amount of oxygen available to the brain and to the muscles to prepare an individual for fighting or fleeing from a perceived danger. The parasympathetic nervous system conserves and maintains physical resources, restoring bodily systems after an emergency and gradually returning body systems to a resting state. Although a wide range of physiologic factors determine heart rate, the two branches of the ANS work together in a dynamic manner and dually innervate the heart (Thayer & Lane, 2000; Thayer & Lane, 2006; Thayer & Friedman, 2004). The two autonomic branches innervate the sinoatrial node of the heart, via the stellate ganglia (sympathetic nervous system).
and the vagus nerve (parasympathetic nervous system). Past research has indicated that the heart is under tonic inhibitory control, with the parasympathetic nervous system dominating over the sympathetic nervous system. Supporting this idea, Jose and Colison (1970) found that when both cardiac vagal nerve and sympathetic influences are blocked pharmacologically (e.g., with atropine plus propranolol), intrinsic heart rate is higher compared with normal resting heart rate. Additionally, Saul (1990) found that due to differences in the temporal kinetics of the autonomic neuroeffectors, the sympathetic nervous system has a relatively slower influence on cardiac control compared to the parasympathetic nervous system. Thus, when parasympathetic nervous system functioning decreases, the organism is less able to adapt to rapid changes in the environment and organize an appropriate response (Thayer & Lane, 2000).

Dysregulation of the autonomic nervous system typically is observed when the sympathetic nervous system is hyperactive and the parasympathetic nervous system is hypoactive. Activation of the sympathetic nervous system enables the organism to organize an alarm reaction to respond appropriately to a stressful situation, also known as a “fight-or-flight” reaction. However, when the sympathetic nervous system dominates for prolonged periods of time, the energy demands on the body become excessive and eventually cannot be met, contributing to wear-and-tear of bodily systems (Thayer & Friedman, 2004). Dysregulation of the ANS is thus associated with a number of physical and psychological symptoms and diseases, and is associated with increased risk of all-cause mortality (Malliani et al., 1994; Thayer, 2009).
One of the main indices of cardiac autonomic function is heart rate variability (HRV), which refers to the beat-to-beat alterations in heart rate (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Wang et al., 2005). HRV can be measured in both time and frequency domains to index vagal nerve activity. In the time domain, the standard deviation of the interbeat intervals (IBI) is generally used, as well as the percentage of IBI differences greater than 50 ms, and the mean square of the successive differences in IBIs. In the frequency domain, both low frequency (LF: 0.04 – 0.15 hertz) and high frequency (HF: 0.15 – 0.40 hertz) spectral power have been used as indices of vagal activity (Task Force Guidelines, 1996). Short-term recordings and long-term recordings can be distinguished in the measurement of frequency domains of HRV. Short-term recordings consist of 2 to 5 minutes, and low frequency and high frequency values can be obtained. Long-term recordings are usually 24 hour recordings where ultra low frequency, very low frequency, low frequency, and high frequency values can be obtained.

The parasympathetic influences are pervasive over the frequency ranges of the heart rate power spectrum, whereas the sympathetic influences only exist below 0.15 hertz (Thayer, 2009). Thus, LF fluctuations in heart rate are jointly mediated by the sympathetic and parasympathetic nervous system, while HF fluctuations in heart rate are mediated solely by the parasympathetic nervous system. The sympathetic effects are slow (in seconds), whereas the parasympathetic effects are fast (milliseconds). Subsequently, only the
parasympathetic nervous system is capable of producing rapid beat-to-beat changes (Thayer, 2009). Another index of vagal tone that has been used in various studies is heart period variability (HPV), which is calculated on the interbeat interval data using 1 millisecond precision. This is in addition to HRV which is usually on the heart rate time series and is in beats-per-minute. Functionally these two indices are analogous, and only differ in how they are measured technically.

Autonomic dysregulation, indexed by reduced HRV, reflects a shift in parasympathetic control to sympathetic control of the heart rhythm. Reduced HRV has been shown to precede disease (Ziegler et al., 2001), as well as predict all-cause mortality, arrhythmic events, and sudden death after acute myocardial infarction (Bigger et al., 1993; Tsuji et al., 1996). Reduced HRV also has been associated with a wide range of metabolic, hemodynamic, trophic, and rheologic abnormalities that contribute to elevated cardiac morbidity and mortality (Brook & Julius, 2000). Additionally, decreased HRV has been associated with age (Craft & Schwartz, 1995). One pathway through which this happens is via increased production of proinflammatory cytokines, which occurs when the sympathetic branch is hyperactive. Research has indicated that release of proinflammatory cytokines is controlled by parasympathetic outflow (Das, 2000; Maier & Watkins, 1998; Tracey, 2002). Recent evidence indicates that cytokines communicate with the brain though the vagus nerve by sending afferent messages from the innervated organs to the brain. When the vagus nerve is severed in animals, cytokine-related functions cease, including pain responsivity, changes in
norepinephrine, glucocorticoid production, and conditioned taste aversions (Maier & Watkins, 1998). Thus, autonomic dysregulation may be a common pathway to increased morbidity and mortality due to a host of conditions and diseases (Thayer & Friedman, 2004).

Poor autonomic regulation also has been associated with diminished attentional control and the reduced ability to regulate emotions, which may be associated with the development of affective disorders and anxiety disorders. Research has indicated that reduced HRV is associated with negative affective states, predisposition to disease, ill health, and decreased positive affect (Friedman & Thayer, 1998; Kiecolt-Glaser et al., 2002; Krantz & McCeney, 2002; Musselman et al., 1998; Rozanski et al., 1999; Verrier & Mittleman, 2000; Bhattacharyya et al., 2008). In particular, panic disorder has been studied because the common symptoms of panic disorder (i.e., heart palpitations, tachychardia, shortness of breath, chest pain, perspiration) are consistent with ANS disturbances (Friedman & Thayer, 1996). Reduced HRV has been associated with elevated sympathetic heart rate activity in panic anxiety, poor attentional control, ineffective emotional regulation, and behavioral inflexibility (Friedman et al., 1993; Friedman, 2007; George et al., 1989; Klein et al., 1995; Middleton et al., 1994; Rechlin et al., 1994; Thayer & Friedman, 1993; Tyrrell et al., 1995; Yeragani et al., 1990; Yeragani et al., 1993; Yeragani et al., 1992; Yeragani et al., 1995; Yeragani et al., 1994). Additionally, reduced HRV has been found in panicogenic manipulations such as hyperventilation and infusions of isoproterenol, lactate, and yohimbine, suggesting that reduced HRV may be a
physiological marker linked with clinical anxiety (Ost et al., 1984). Friedman & Thayer (1998) compared panickers, blood phobics, and nonanxious controls on a variety of cardiovascular measures in response to laboratory stressors. Results indicated that panickers had the highest heart rates with the lowest HRV of the three groups. Blood phobics had slightly higher HRV than panickers, with nonanxious controls having the highest HRV (Friedman & Thayer, 1998). Thus, it appears that low HRV is associated with poorer emotion regulation and the presence of anxiety disorders. In contrast, positive affect has been associated with increased HRV. In a study in patients with coronary artery disease, positive affect was associated with increased HRV independent of age, gender, medication, CAD status, body mass index, smoking, and physical activity (Bhattacharyya et al., 2008).

HRV has also been associated with prefrontal cortex activity (Thayer & Friedman, 2004; Lane et al., 2001). The prefrontal cortex generally governs higher executive cognitive processes. Research has indicated that prefrontal cortex activity is inversely related to activity in primitive emotional systems such as the amygdala (Davidson, 2000). When an organism is exposed to threat, prefrontal cortex activity is moderated and a relative sympathetic dominance is engaged, enabling a number of processes associated with the threat response to occur, including hypervigilance, fear, increased heart rate and blood pressure, all of which are associated with autonomic dysregulation. This allows the organism to organize automatic and prepotent processes to regulate behavior in order to adjust to rapid environmental changes in an adaptive way without the
interference of more deliberative and consciously guided processes (Arnsten & Goldman-Rakic, 1998). However, prolonged moderation of the prefrontal cortex will lead to symptoms that are often seen in anxiety disorders, such as hypervigilance, defensive behavioral disposition, and perseveration (Thayer & Siegle, 2002).

It has been suggested that when autonomic dysregulation takes place, the core cognitive toxic factor that prolongs psychological stress and eventually leads to physiological illness is increased rumination and worry, or perseverative cognition (Brosschot et al., 2006). Rumination is a specific cognitive vulnerability factor that has been defined as repetitive, intrusive, and negative cognitions that maintain depressive symptoms (Papageorgiou & Siegle, 2003; Roelofs et al., 2006). Worry has been defined as a chain of thoughts and images that provoke negative affect, and usually repetitive, abstract, and uncontrollable to the individual (Thayer & Friedman, 2002). Worry and rumination are key components of most anxiety disorders, especially GAD (American Psychiatric Association, 1994). Tallis and Eysenck (1994) propose a tripartite function of worry, first serving an alarm function by directing awareness to an issue that requires imminent action, then serving as a prompt function by continuously representing unresolved threatening situations to awareness, and finally as a preparation function, being vigilant about anticipated threat and readying the organism to respond to the perceived threat. Thus, perseverative cognition appears to be "a protracted state of psychophysiological action preparation without resolution", characterized by an inflexible way of thinking, marked by hypervigilance and
impaired habituation to non-threatening stimuli (Thayer & Friedman, 2004; Friedman et al., 2000; Thayer et al., 2000; Thayer & Friedman, 2002). In a study comparing individuals with GAD to non-anxious controls, GAD patients had lower vagally-mediated heart period variability compared to non-anxious controls across all experimental conditions including baseline. When asked to engage in a 10-minute worry period, worry in both the GAD and non-anxious control group was associated with a reduction in heart period variability. In addition, GAD patients failed to habituate to novel neutral stimuli whereas nonanxious controls showed habituation (Thayer & Lane, 2000). In a similar study, Lyonfields et al. (1995) exposed GAD patients and a nonanxious control group to periods of imagery and worry. GAD patients exhibited reduced HRV across all periods, with little change from one period to the next, demonstrating reduced habituation. Although nonanxious controls showed more variability of HRV across periods, they showed the greatest reduction of HRV during the worry period (Thayer & Friedman, 2004).

Worry and rumination are central cognitive components that prolong autonomic dysregulation because they extend acute psychological and physiological stressors of daily life into prolonged stressors through a cognitive representation. Perseverative cognition may eventually lead to physical health consequences via decreased vagally mediated HRV. Thus, a prolonged state of autonomic dysregulation perpetuates activation of several bodily systems that contribute to the wear and tear of the body. (Brosschet & Thayer, 1998; McEwen, 1998). It has also been suggested that perseverative cognition is a cognitive
manifestation of perceived uncontrollability, which has been associated with pathogenic physiological states and various health problems in stress-disease research (Brosschot et al., 1998; Everson et al., 1996; Frankenhäuser, 1980; Lundberg & Frankenhäuser, 1978; Steptoe and Appels, 1989; Ursin, 1987; Ursin & Hytten, 1992). Trait and state rumination and worry have been associated with illness symptoms (Brosschot & van den Doef, in press; Thomsen et al., 2004; Lok & Bishop, 1999; Emmons & King, 1988; Rector & Roger, 1996; Lacey et al., 2000). Chronic worry also may be a risk factor for coronary heart disease in older men. Compared with men reporting low levels of worry, men reporting high levels of worry exhibited 2.41 times more risk for nonfatal myocardial infarction, and 1.48 times more risk for coronary heart disease twenty years later (Kubzansky et al., 1997).

Stress may also lead indirectly to disease via poor lifestyle choices, such as lack of physical activity, smoking, alcohol consumption, drug use, and poor sleep quality. Research has indicated that lack of physical activity and substance abuse are associated with autonomic dysregulation and decreased vagal tone (Ingjaldsson et al., 2003; Nabors-Oberg et al., 2002; Reed et al., 1999; Rossy & Thayer, 1998; Weise et al., 1986). Research also indicates that the therapeutic effectiveness of smoking cessation, reduced alcohol consumption, and increased physical activity may be in part due to their ability to restore autonomic balance by increasing parasympathetic nervous system activity (Thayer & Friedman, 2004).

In summary, autonomic dysregulation indexed by reduced HRV is
associated with detrimental physical health outcomes as well as deficits in emotional regulation. This process is characterized by a core cognitive-emotional process involving worry and rumination, which causes stressors to be prolonged through a cognitive representation and therefore contributes to chronic hypervigilance and anticipation, eventually leading to negative physical consequences for the body. Reduced HRV may also contribute to poor health outcomes indirectly via negative health behaviors.

**Autonomic Dysregulation and COPD**

Autonomic dysregulation may be a main mechanism to explain elevated levels of anxiety and depression in COPD patients. As mentioned above, previous research indicates that the release of proinflammatory cytokines is regulated by parasympathetic outflow. COPD is a chronic illness with systemic consequences including deconditioning, exercise intolerance, skeletal muscle dysfunction, osteoporosis, metabolic impact, anxiety and depression, cardiovascular disease, and mortality (Decramer et al., 2008; Sin et al., 2006; Sevenoaks & Stockley, 2006). Literature suggests that the underlying mechanism of these systemic consequences involve a “spill-over” of inflammatory cells and cytokines from the lung into the systemic circulation which is most notable during exacerbations. During exacerbations, there are more circulating levels of cytokines, interleukin (IL)-6, IL-8, C-reactive protein (CRP), adhesion molecules, fibrinogen, and circulating activated inflammatory cells (Di Francia et al., 1994; Schols et al., 1996; Gan et al, 2004; Broekhuizen et al., 2006; Sauleda et al., 1999; Asin et al., 1998; Wedzicha et al., 2000; Spruit et al.,
There has also been suggestion that the elevated level of depression and anxiety in this population may be associated with the chronic inflammatory process in COPD. Depressed patients without COPD or other chronic medical illnesses have been shown to have high levels of inflammatory cytokines including IL-1, IL-6, and TNF-a (Musselman et al., 2001; Maes, 1999). In addition, when cytokines were administered to healthy individuals, they developed symptoms that were similar to depression, including fatigue, hypersomnia, irritability, decreased appetite, and cognitive deficits (Berkenbosch et al., 1987).

It has also been suggested that abnormal autonomic function may contribute to the symptomatology of COPD patients, including coughing, bronchospasms, and airway narrowing, via constriction of airway smooth muscle, bronchial vessels, and mucous glands in the bronchial wall (Volterrani et al., 1994; Barnes 1986; De Jongste et al., 1991). Afferent nerves in the respiratory tract can be divided into stretch-sensitive mechanosensors and the nocioceptors. Stretch-sensitive mechanosensors, which are mostly vagal afferent nerves, respond to nonharmful expansion of the lungs. Nocioceptors are nerves that are normally silent when in a healthy, nonpathological state, but evoke defensive reflexes when activated. Two nocioceptors of the respiratory tract are located in the larynx / large airways and bronchopulmonary C-fibers. When stimulated, nociceceptors of the larynx / large airway evoke a rapid cough reflex and evoke defensive reflexes including apnea, rapid shallow breathing, mucus secretion, bronchoconstriction, and cough (Undem & Kollarik, 2005; Coleridge & Coleridge,
Parasympathetic activity of the airways is tightly controlled by these stretch-sensitive afferent nerves and nociceptive nerves. Increased activation of stretch-sensitive afferent nerves and bronchopulmonary nociceptive activity lead to heightened parasympathetic nerve-mediated bronchial smooth muscle contractions and mucus secretion (Undem & Kollaric, 2005). Additionally, activation of nociceptors may lead to a pathological alteration of the cough reflex, leading to exaggerated sensitivity of the reflex pathways. Thus, the amount of afferent activation required to trigger cough may be decreased in COPD patients and may lead to a heightened urge to cough when unnecessary. Evidence also supports the hypothesis that vagal afferent activity may increase dyspneic sensations (Undem & Kollaric, 2005).

Several prior studies suggest decreased parasympathetic nervous system activity in COPD patients compared to healthy controls, although the results are equivocal. Volterrani et al. (1994) performed a brief manipulation with COPD patients and age-matched controls, including rest, controlled breathing, and passive orthostatism (a sympathetic maneuver), recording 600 heart cycles for each condition. Results indicated overall depressed HRV in COPD patients across all three conditions, indexed by reduced global HRV (standard deviation of the RR interval) in COPD patients compared to the controls. However, at rest, COPD patients demonstrated elevations in standardized HF measures, indicating increased parasympathetic activity, compared to controls. Volterrani et al. argued that increased vagal activity in COPD patients at rest could explain in part the reduction in forced expiratory volume in one second (FEV$_1$) and the increase in
bronchoconstriction observed in this population because the vagus nerve modulates the rate of sinoatrial discharge. Thus, an abnormality in the parasympathetic control of airway caliber may in turn result in a parallel change in heart rate. Results also indicated that COPD patients exhibited a lack of responsiveness to sympathetic stimulation compared to controls, indexed by a standardized LF index, and a much smaller decrease in the standardized HF index. Volterrani et al. suggested that autonomic nervous system activity of patients with COPD may be working at maximum capacity. Thus, although COPD patients exhibit higher vagal tone at rest, they may have less range of variation than healthy individuals and their autonomic pattern is more rigid.

Another study by Stein et al. (1998) investigated 24 hour long-term recording of HRV in COPD patients with marked α1-antitrypsin deficiency compared with matched healthy controls. This study utilized long-term recordings for daytime, nighttime, and 24 hour indexes of RMSSD and pNN50, and found that all indices of HRV were significantly decreased in COPD patients compared to healthy controls.

A third study by Bartels et al. (2003) compared short-term recordings of HRV in COPD patients and healthy controls at rest and during ramped bicycle ergometry to their volitional peak. COPD patients were not different in high frequency HRV at rest compared to healthy controls. However, the COPD patients had significantly increased ln-transformed high frequency band from rest to peak exercise while the HF band was unchanged for the control group. The LF/HF ratio was significantly decreased from rest to peak exercise in patients
with COPD while it was significantly increased in control subjects. The results of this study suggested that COPD patients may have a loss of ability to achieve a sympathetic response as the baseline sympathetic tone was elevated, or an abnormally higher level of parasympathetic tone in COPD patients compared to healthy controls.

Results from studies have also indicated that HRV was related to oxygenation status in COPD patients. Chen et al. (2006) found a significant negative correlation between a standardized HF measure (index of vagal activity) and PaO$_2$ (arterial partial pressure of O$_2$), and a significant positive correlation between standardized LF measures (indices of sympathetic activity) and PaO$_2$ in COPD patients. Stein et al. (1998) found a positive correlation between FEV$_1$ and most indices of HRV, reflecting mixed sympathetic and parasympathetic, and purely parasympathetic modulation of heart rate. Other studies have suggested that patients with hypoxemic COPD may have subclinical parasympathetic autonomic neuropathy, with autonomic neuropathy correlating significantly with PaO$_2$ in COPD patients (Stewart et al., 1991; Stewart et al., 1994; Stewart et al., 1995; Hjalmarsen et al., 1996; Scalvini et al., 1999).

In summary, autonomic dysregulation indexed by HRV has been associated with negative physical symptoms as well as diminished capacity for attentional control and emotional regulation. Although the research remains inconclusive about the presence of autonomic dysregulation in COPD patients, it is possible that autonomic dysregulation, if present, may be a component in the pathophysiology of COPD, contributing to the exacerbation of symptoms.
(coughing and dyspnea) as well as poor emotion regulation (symptoms of anxiety).

The Current Investigation

Elevated levels of anxiety and high prevalence of anxiety disorders, especially panic disorder and GAD, have been observed among patients with COPD. Furthermore, decreased heart rate variability has been linked to COPD and poor emotional regulation, but there have not been any studies investigating decreased heart rate variability as an underlying mechanism that may partially explain the high levels of anxiety in COPD patients.

This study examined heart rate variability associated with anxiety symptoms in COPD patients compared to healthy controls when exposed to a psychosocial stress task. The purpose of the study is to examine HRV in patients with COPD to evaluate the extent of autonomic dysregulation accompanying the disease, and examine psychological and dispositional variables such as worry and rumination and measures of distress such as anxiety in COPD patients. Although there have been no prior studies in this area, prior data suggest that the presence of both COPD and elevated anxiety may have an additive or synergistic effect reflected in abnormalities of HRV and other outcome measures.

To further clarify the relationship between HRV and anxiety in COPD patients, this study utilized a 2 X 2 factorial design comparing four groups of participants: COPD patients with elevated anxiety (COPD-ANX), COPD patients without elevated anxiety (COPD), healthy individuals with elevated anxiety who do not have COPD (HEA-ANX), and healthy individuals without elevated anxiety
(HEA). All participants completed a standardized psychosocial stress task and were assessed on measures of psychological distress, personality traits (worry and rumination), and pulmonary functioning before the stressor, as well as on psychological distress after the stressor. HRV was measured continuously throughout the baseline, task, and recovery phases.

The following hypotheses were examined in the present investigation:

(1) Poor health behaviors (current smoking, pack-years of smoking, alcohol consumption, poor sleep quality, and less physical activity) would be associated with lower HRV in all four groups.

(2) Higher HRV would be associated with better pulmonary functioning and lower state anxiety at baseline in all four groups.

(3) Depression, negative affectivity, and perceived stress would be negatively associated with HRV at baseline, and positive affectivity would be positively associated with HRV in all four groups.

(4) Exposure to a psychosocial stressor would be associated with decreased HRV and increased HR in all four groups.

(5) COPD-ANX participants would have lower HRV at baseline compared to individuals in the COPD, HEA, or HEA-ANX group.

(6) COPD-ANX participants would exhibit slower recovery in HRV following a psychosocial stressor compared to the other three groups.

(7) COPD-ANX participants would exhibit greater state anxiety following a psychological stressor compared to the other three groups.

(8) Individuals with high levels of worry and rumination would exhibit slower
recovery of HRV following a psychosocial stressor.
CHAPTER 2. METHODS

This study recruited individuals diagnosed with COPD and healthy individuals. Individuals with varying degrees of anxiety were recruited into both groups. All participants were exposed to a brief psychosocial stress task. Pulmonary and psychological functioning were assessed before the stressor task. HRV was monitored throughout the study. State anxiety was assessed after the stressor task.

Participants

Participants included 30 individuals diagnosed with COPD and 30 healthy individuals who were free from chronic lung disease. Participants with COPD were recruited from among participants in the Ohio State University (OSU) Pulmonary Rehabilitation Program located at the Center for Wellness and Prevention and OSU Hospital East, and also through flyers posted in the community. Healthy individuals were recruited via advertisements and flyers posted throughout the central Ohio community. All participants with COPD had a physician diagnosis of COPD for at least 3 months. All diagnostic procedures were consistent with the GOLD criteria [indicated by forced expiratory volume in one second / forced vital capacity < .70 (FEV_1/FVC < .70)]. Eligible participants were paid $50 for participation in the study. Participants ranged in age from 36 to 83 (mean = 59.1 ± 11.2 years). 50%
of the participants were female. The sample consisted of 40% Caucasian, 16% African American, 1% Hispanic, 2% Asian, and 1% Other. Twenty-two (73%) participants reported a diagnosis of COPD, five (17%) reported a diagnosis of emphysema, and three (10%) reported a diagnosis of chronic bronchitis. Participants had been diagnosed with their pulmonary condition for an average of 4.93 (± 3.51) years. Participants with COPD presented with severe airway obstruction, indicated by a mean FEV₁% predicted of 54.03 (± 22.11) and a FEV₁/FVC ratio of 0.63 (± 0.15). Five participants were receiving supplemental oxygen therapy on a daily basis. Additional demographic information and baseline pulmonary function values of the sample are summarized in Tables 1 and 2.

**Procedure**

This study was conducted at the Center for Wellness and Prevention located at the Ohio State University Medical Center. Exclusionary criteria included individuals who were pregnant, were currently on beta-antagonist medication, or had a history of cardiovascular disease (anyone who had been diagnosed with a cardiac disease such as congestive heart failure, prior myocardial infarction, stroke, etc.). Participants who expressed interest in the study were contacted by telephone or in person. Participants with COPD who were eligible for the study were matched by age (± 2 years) and gender to healthy individuals. After consenting to the study, all participants completed the State-Trait Anxiety Inventory – State version to identify high and low anxiety for analysis in four groups (COPD, COPD-ANX, HEA, HEA-ANX). The cut-off score
was a standard clinically relevant value of 39 (Spielberger, 1988). The target number of individuals in each group was 15, and individuals were deemed ineligible to participate after consenting to the study if the quota for a group had been met. Ineligible participants were paid $10 and were excused after the anxiety screening.

Individuals who qualified for the study were scheduled for a visit to complete the remainder of the study. All participants were asked to bring their list of medications to the visit. Additionally, all participants were asked to refrain from smoking, consuming alcohol or caffeine, or taking anxiolytic medication 24 hours prior to participating in the study to avoid confounding of HRV measurements. All participants with COPD were asked to refrain from taking β2-agonist inhalers six hours prior to the study to avoid confounding of HRV measurements.

Following consent to the investigational procedure, all participants were asked to complete a questionnaire measuring state anxiety (Spielberger, 1988). All participants with high levels of anxiety were asked about the duration of their anxiety symptoms. Blood pressure was measured for each individual to confirm that there were no hypertensive individuals in the sample. Following this procedure, height and weight was measured for each participant. Participants then completed pulmonary function testing using standard spirometry equipment (Koko Legend Spirometer). All spirometry testing was performed in accordance with the recommendations of the American Thoracic Society/European Respiratory Society (American Thoracic Society, 1995; European Respiratory Society, 1993). Spirometry testing measures inhaled and exhaled volumes or
flow of air as a function of time. Spirometry testing was conducted by a trained research assistant. All participants were seated in an upright position, and spirometry testing was explained as involving forceful breathing into a mouth piece to measure the largest volume of air that can either be breathed in or out from the lungs. Participants were asked to loosen tight fitting clothing such as belts or vests, which might limit chest expansion. The participant then placed the mouthpiece in his / her mouth and the nose clip on his / her nose. Participants were told to maintain a tight mouth seal throughout each test maneuver. Participants then inhaled rapidly and completely, positioned the mouthpiece, and exhaled with maximal force.

Following pulmonary function testing, participants were fitted with a polar watch and a thoracic band to measure HRV throughout the remainder of the experiment and oxygen saturation was measured to ensure participants had not desaturated.

The study was divided into three phases: Baseline, Task, and Recovery, as shown in Figure 1.

**Baseline.** The purpose of the baseline phase was to obtain a stable measure of the participant’s HRV at rest. All participants completed a packet of baseline questionnaires, including health behaviors, measures of distress, and dispositional variables. The questionnaires required 15 – 20 minutes for completion. Following the questionnaires, participants remained sitting quietly for another five minutes. After this quiet sitting period, participants read a neutral script out loud about doing laundry. Participants were asked to stop reading the
neutral script after one minute.

Task. Immediately after the baseline phase, participants were exposed to the stressor task. The Trier Social Stress Test (TSST; Kirschbaum et al., 1993) is a standardized procedure developed at the University of Trier to induce moderate psychosocial stress under laboratory conditions. In numerous studies, the TSST has proven to elicit significant changes in cardiovascular parameters and other endocrine markers (Kirschbaum et al., 1995; Kirschbaum et al., 1996; Kirschbaum et al., 1992a; Kirschbaum et al., 1992b). A modified version of the Trier Social Stress Test (speech task) was used for this study.

For the modified TSST, the research assistant first exposed the participant to a video camera, which had been previously hidden from view of the participant. The participant then listened to videotaped instructions indicating that the participant would need to deliver a speech, and would be given two minutes to prepare. The participant was informed that the speech would be videotaped and that the speech should be five minutes long. When the taped instructions said, “Please take 2 minutes now to construct your speech”, a stopwatch was set for two minutes. After two minutes, the participant was asked to stand in front of the video camera, and the video camera was switched on. The participant then delivered the speech. If the participant remained silent for more than 20 seconds during the speaking period, the participant was prompted to continue speaking until time was called.

Recovery. After the speech stressor task, the participant completed a post-stressor questionnaire of state anxiety. The experimenter then placed
headphones on the participant for the 20-minute recovery phase, during which the participant listened to relaxing music. After the recovery phase, oxygen saturation was measured, followed by removal of HRV equipment. Upon completion of the experimental procedures, participants were debriefed on the nature of the study and encouraged to ask any additional questions they might have regarding the study.

Measurement / Instrumentation

Health Behaviors

*Smoking History.* Participants were asked detailed questions about lifetime frequency, duration, and amount of cigarettes smoked. Pack-years of smoking was calculated as a cumulative measure of exposure to smoking (Pack years = Number of packs of cigarettes smoked per day X Number of years smoked this amount).

*Alcohol Intake.* Six items addressed alcohol use (e.g. “In the past month, how many alcoholic drinks per week did you consume?”). Responses range from 0 (I never drink beer / wine / mixed drinks) to 4 (6 or more beer / wine / mixed drinks a day), assessing for current and past use. One alcoholic drink is defined as 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of hard liquor.

*Physical Activity.* The Houston non-Exercise Index (Jackson et al., 1990) is a method to estimate $\text{VO}_{2\text{max}}$ based on self-reported physical activity rating (PAR) in combination with age, body mass index (BMI), and gender. The PAR ranges from a numeric value of 0 (avoids walking or exercise, for example, always uses elevators, drives whenever possible instead of walking) to 7 (runs
more than 10 miles per week or spends more than 3 hours per week in comparable physical activity). Calculation of the estimated VO$_{2\text{max}}$ uses the following equation: $VO_{2\text{max}} (\text{ml/kg/min}) = 56.363 + [1.921 \times (\text{PAR score})] - [0.381 \times (\text{age})] - [0.754 \times (\text{BMI})] + [10.987 \times (\text{gender}: 0 \text{ for women and } 1 \text{ for men})]$. This estimate has been shown to produce values that have a correlation of 0.78 with measured VO$_{2\text{max}}$. Accuracy is high in approximately 96% of the population with VO$_{2\text{max}}$ scores less than or equal to 55 ml/kg/min. The remaining 4% of the population are very highly fit individuals for which this model is less accurate (Ross & Jackson, 1990).

Sleep. The Pittsburgh Sleep Quality Index (PSQI; Buysse, D. J., et al., 1988) is a self-report questionnaire assessing sleep quality and disturbances over a one-month interval. It has good diagnostic sensitivity and specificity (a cut score of 6 or greater yielded a diagnostic sensitivity of 89.6% and specificity of 86.5%) in distinguishing good and poor sleepers. The scale yields a total score that ranges from 0 – 21, with higher scores indicating more difficulties with sleep. The questionnaire also has 7 subscales which include subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction.

Measures of Distress

Stress. The Perceived Stress Scale (PSS-10; Cohen, Kamarck, & Mermelstein, 1983) is a 10-item self-report measure assessing the degree to which situations in one’s life are appraised as stressful (e.g. “In the last week, how often have you been upset because of something that happened
Respondents are asked to rate each item on a scale of 0 (Never) to 4 (Very often). Higher scores on this measure are indicative of greater perceived stress. The internal consistency for this measure ranges from .84 - .86, while test-retest reliability ranges from .55 to .85 for time intervals ranging from two days to six weeks. Evidence for concurrent validity was found when this measure was correlated with the number and impact of life events, ranging from .24 to .49. The Cronbach’s alpha for this sample was 0.89 for the PSS-10.

Positive/Negative Affect. Trait positive and negative affect was measured with the Positive and Negative Affectivity Scales (PANAS; Watson, Clark & Tellegen, 1988). Negative affect describes a variety of negative moods including anxiety, sadness, guilt, hostility, and self-dissatisfaction. In contrast, positive affect depicts a number of positive mood states including joy, energy, enthusiasm, interest, and affiliation. The PANAS is a 20-item measure that assesses negative affect (PANAS-N, 10 items) and positive affect (PANAS-P, 10 items). Participants are asked to rate themselves on mood descriptors (i.e., “excited,” “upset,” and “inspired”) on a 5-point likert scale, ranging from (1) “very slightly or not at all” to (5) “extremely” based on how they generally felt. The Cronbach’s alpha for this sample was 0.48 for the PANAS-N, and 0.44 for PANAS-P.

Anxiety. The State-Trait Anxiety Inventory, state (STAI-X1; Spielberger, 1988) is a 20-item measure of state anxiety. The trait (STAI-X2, Spielberger, 1988) is a 20-item measure of trait anxiety. Both the state and trait scales of the STAI have been widely used for psychiatric and medical patients. Items on the
state version include statements such as “I feel upset” and “I am presently worrying over possible misfortunes” rated on a scale of 1 (almost never) to 4 (almost always). Items on the trait scale include statements such as “I am losing out on things because I can’t make up my mind soon enough” or “I try to avoid facing a crisis or difficulty” rated on a scale of 1 (almost never) to 4 (almost always). The instruction set asks respondents to indicate how they are feeling at the present moment for the state scale. For the trait scale, participants rate how they generally feel. The STAI has demonstrated adequate reliability in older adults (α = .94; Stanley et al., 1996). The measure also demonstrated adequate convergent reliability with the Fear Questionnaire, Worry Scale, and the Padua Inventory (Stanley et al., 1996). The Cronbach’s alpha for this sample was 0.94 on the STAI-X1 and 0.92 for the STAI-X2.

Depression. The Center for Epidemiological Studies Depression Scale (CESD; Radloff, 1977) is a 20-item measure of symptoms of depression including depressed mood, feelings of guilt, worthlessness, helplessness, hopelessness, loss of appetite, and sleep disturbance. Respondents indicate how they have been feeling during the past week using a scale of 0 (Rarely or none of the time, less than one day) to 3 (Most of or all of the time, five to seven days). The CESD demonstrates internal consistency with α = .85 for the general population and α = .90 for psychiatric populations. Test-retest reliability ranges from .51 to .67 (tested over two to eight weeks) and .32 to .54 (tested over 3 months to one year). This measure also demonstrates good concurrent validity with other depression and mood scales, and it has been useful in discriminating
between clinical and normal populations. The Cronbach’s alpha for this sample was 0.93 on the CESD.

Dispositional Variables

Worry. The Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) is a 16-item self-report measure assessing the trait of worry (e.g., “My worries overwhelm me”). Respondents are asked to rate each item on a scale of 1 (Not at all typical) to 5 (Very typical), with higher scores indicative of greater trait worry. The internal consistency for this measure is 0.9. Test-retest reliability for 8 – 10 weeks was demonstrated to be 0.92. This measure also has demonstrated adequate convergent validity, correlating with the State-Trait Anxiety Inventory, Trait version \((r=0.64, p<0.001)\) and the Beck Depression Inventory \((r=0.36, p<0.001)\). The Cronbach’s alpha for this sample was 0.91 on the PSWQ.

Ruminative Response Scale (RRS; Nolen-Hoeksema & Morrow, 1991) is a measure of rumination assessing responses to dysphoric mood that are focused on the self, symptoms, and possible causes and consequences of moods. The abbreviated 10-item measure will be used in this study. Respondents are asked to rate each item on a scale of 1 (Almost Never) to 4 (Almost Always) depending on how often they think or do something when they are feeling down, sad, or depressed (e.g., Think about a recent situation, wishing it had gone better). The RRS consists of two subscales – the reflection subscale measuring rumination and coping, and the brooding subscale measuring what people do when they are sad or depressed. Internal consistency for this measure ranged from .87 - .90 (Roelofs et al., 2006). Test-retest reliability
was assessed over a six month period and was adequate, ranging from .43 - .53.

The RRS also demonstrates adequate construct validity, moderately correlating with measures of the Zung depression scale \( r = .27 - .49 \), trait anxiety measured by the STAI \( r = .35 - .53 \), and Eysenck Personality Questionnaire – Neuroticism Scale \( r = .34 - .53 \). The Cronbach’s alpha for this sample was 0.82 for the RRS-B and 0.81 for the RRS-R.

**Social Desirability Scale.** The Marlowe-Crowne Social Desirability Scale (MCSD; Crowne & Marlowe, 1960) is a 13-item self-report measure assessing level of social desirability. Respondents are asked to answer true or false to a number of statements concerning personal attitudes and traits (i.e., “Sometimes I feel resentful when I don’t get my way”). This measure demonstrates an acceptable level of reliability at 0.76. The Cronbach’s alpha for this sample was 0.72 on the MCSD.

**Attentional Control.** The Attentional Control Scale (Derryberry & Reed, 2002) is a 20-item self-report questionnaire measuring the ability to focus and shift attention if necessary (e.g., “When concentrating, I can focus my attention so that I become unaware of what’s going on in the room around me”, “I can quickly switch from one task to another”). Items are scored on a 4-point scale with 0=never, 1=sometimes, 2=often, and 3=always. After recoding inversely formulated items, a total score can be computed by summing across relevant items, with higher scores reflect lower levels of attention control. The Cronbach’s alpha for this sample was 0.88 on the ACS.

**Physiological Indices**
**Heart Rate Variability.** The Polar S810 Fitness watch was used to measure continuous R to R interval mode using a thoracic band which transmits and stores interbeat intervals at 1000 hertz on a wristwatch. Interbeat interval time series for periods of interest were reduced and artifact rejected and processed using the KUBIOS Heart Rate Variability Package which calculates HRV parameters both in time and frequency domains using Task Force Guidelines for HRV. Task Force Guidelines for HRV recommend a short-term recording to be between 2 to 5 minutes to obtain a stable recording, or to avoid ‘stationarity.’ The five-minute resting segment of the baseline and the five-minute speech of the task phase were used. To avoid stationarity issues based on the Task Force Guidelines for HRV, a five-minute segment reflecting the most stable heart rate recording of the 20 minutes for the Recovery phase was identified and corrected for artifacts for each participant. The HRV parameters for the time domains that were used for analyses were mean heart rate (mean HR), mean IBI (Mean RR), the standard deviation of normal – normal beats (STDRR), and square root of the mean squared differences of successive N-N intervals (RMSSD). In the frequency domain, measures of HRV (HF-HRV, RMSSD) tend to be positively skewed; this deviation from normality negatively impacts statistical significance. Because of this, a natural log (Ln) transformed value of the LF and HF HRV was used, derived from the absolute values of HF and LF (e.g., Riniolo & Porges, 2000). The LF/HF ratio was also used in the frequency domain. Although past studies have used normalized HF and LF values, the equation from which these values are derived requires a very low frequency
value (VLF), representing the non-harmonic component which does not have coherent properties, as indicated in the following equation: \[ \frac{HF}{(TP-VLF)} \times 100 \] or \[ \frac{LF}{(TP-VLF)} \times 100 \]. However, it is difficult to obtain the VLF when the recording time is less than 5 minutes (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Thus, these values were not used in this study. As a measure of global HRV, the standard deviation of normal-normal beats will be used (STDRR), which has been used in previous studies with COPD patients (Volterrani et al., 1994).

**Pulmonary Functioning.** Standard pulmonary function testing equipment (Koko Legend Portable Spirometer) was used to determine the volume of expired air, after a maximum inspiration, as well as the flow rate of air. Spirometry measures the vital capacity (VC), which is the volume of air in a full expiration from the lungs. VC measured from a maximal forced exhalation is called the forced vital capacity (FVC). Common diagnostic measurements include both the FVC and FEV\(_1\), measured in liters. FEV\(_1\) is a primary indicator of the severity of COPD. Degree of COPD will be characterized for each patient by the percent predicted FEV\(_1\) (according to norms for age, race, sex, and height), and by the ratio FEV\(_1\)/FVC.

**Oxygen saturation.** Oxygen saturation was measured via a pulse oximeter finger-cusp throughout the baseline, task, and recovery phases. Oxygen saturation was not used for analyses, but was measured for safety precautions.
CHAPTER 3. DATA ANALYSIS

Pearson correlations were used to evaluate the intercorrelations between HRV and pulmonary functioning, health behaviors, state anxiety, depression, negative and positive affectivity, and perceived stress at baseline.

The primary mode of data analysis for HRV was a 2 (group: COPD-ANX group vs. COPD, HEA, HEA-ANX groups combined) X 3 (Phase) repeated measures analysis of variance (ANOVA) with group affiliation as the between subjects variables and phase as the within subjects variable.

Additional exploratory analyses were conducted using 2X2X3 (Disease State X Anxiety Group X Phase) repeated measures ANOVA with disease state (COPD vs. non-COPD) and anxiety group (anxious vs. nonanxious groups) as between subjects variables and phase (baseline, task, recovery) as the within subjects variable. A 2X2X2 repeated measures ANOVA was conducted for state anxiety with two levels of time (baseline and post-task). A one-way ANOVA was conducted for the following variables evaluating differences in disease state (COPD vs. non-COPD): pulmonary functioning (FEV₁, FVC, FEV₁/FVC), health behaviors (smoking status, pack-year history, alcohol consumption, sleep quality, physical activity), depression, trait anxiety, positive and negative affect, stress, worry, rumination, and attentional control.
CHAPTER 4. RESULTS

Sixty-nine individuals responded to recruitment efforts and consented to the procedure. However, six individuals were ineligible for the study because the quota had been met for the group they belonged in. Of the 63 remaining participants, two individuals completed the study but did not meet criteria for COPD and were excluded from analyses. One individual completed the study but was excluded from analyses due to HRV equipment malfunction. Therefore, 60 participants comprised the final sample of this study, with 15 participants in each of the four following groups: COPD, COPD-ANX, HEA, HEA-ANX. Participants with COPD (in the COPD and COPD-ANX group) did not differ from the healthy controls (in the HEA and HEA-ANX group) with regard to age, gender, marital status, living situation, income, and education. There was a significant difference between the COPD and healthy group in respect to ethnicity when including only Caucasian and African American participants ($X^2 = 4.84$, $p=0.03$) There was a significantly larger proportion of African Americans in the COPD group compared to the healthy group. As expected, there were differences in pulmonary functioning among the two groups. The average duration of anxiety for individuals in the high anxiety groups was 28.87 ($\pm 25.89$) years for the COPD-ANX group and 27.97 ($\pm 27.02$) years for the HEA-ANX group, and did not differ significantly between groups. Tables 1 summarizes demographic characteristics
in the COPD and healthy groups.

**Comparison of two groups: COPD groups (COPD and COPD-ANX) vs. Healthy Controls (HEA and HEA-ANX)**

Means, standard deviations, and percentages for all measures of health behaviors, distress, dispositional variables, and pulmonary functioning are included in Table 2.

**Health Behaviors**

- **Smoking History.** Mean pack-years for the COPD group [33.13 (±27.83)] was significantly greater than the healthy group [7.07 (±12.52); F (1, 59) =21.88, p=.000)].

- **Alcohol Intake.** Chi-square analyses revealed there were no differences in alcohol consumption between the COPD groups and the healthy groups (X² = 4.10, p=0.54).

- **Physical Activity.** V0₂max scores derived from the non-Exercise Houston questionnaire indicated mean scores of 20.7 (±11.0) for the COPD group and 23.9 (±10.4) for the healthy group. These scores were not significantly different between groups (F(1, 59) =1.35, p=0.25).

- **Sleep.** Mean scores on the PSQI indicated significantly higher levels of sleep difficulties for the COPD group [9.4 (±4.5)] than for the healthy group [6.4 (±3.8); F (1, 56) =7.2, p=0.01)]. Upon further investigation, the COPD group and healthy group were significantly different on subscales for subjective sleep quality (COPD vs. Healthy = 1.6 ± 0.9 vs. 1.1 ± 0.8; F (1, 57) = 5.30, p = 0.03), sleep disturbances (COPD vs. Healthy = 1.9 ± 0.7 vs. 1.4 ± 0.6; F (1, 59) = 6.09, p =
and use of sleep medications (COPD vs. Healthy = 1.1 ± 1.4 vs. 0.4 ± 0.9; F (1, 58) = 4.84, p = 0.03). Analyses of subscales indicated that individuals in the COPD group rated their sleep quality lower than the healthy group, with more sleep disturbances and more use of sleep medications compared to the healthy group.

**Measures of Distress**

**Stress.** Mean scores on the PSS-10 for the COPD group were not significantly different compared to the healthy group (F (1, 59) =0.02, p=0.87).

**Positive/Negative Affect.** The mean scores for the COPD group were not significantly different compared to the healthy group on either the PANAS-P or PANAS-N (F (1, 59) =0.6, p=4.3; F (1, 59) =0.30, p=0.58, respectively).

**Anxiety.** There was no difference in trait anxiety between the COPD group and the healthy group (F (1, 57) =0.04, p=0.85).

**Depression.** On the CESD, the mean for the COPD group was not significantly different compared to the healthy group (F (1, 57) =0.76, p=0.39).

**Dispositional Variables**

**Worry.** On the PSWQ, the mean for the COPD group was not significantly different compared to the healthy group (F (1, 56) =0.20, p=0.89).

**Rumination.** The means for the COPD group were not significantly different on either the RRS-R or RRS-B scales compared to the healthy group [F(1, 59) =0.15, p=0.70; F(1, 59) =0.24, p=0.62, respectively].

**Social Desirability Scale.** On the MCSD, the mean for the COPD group was not significantly different compared to the healthy group (F (1, 59) =0,
Attentional Control. On the ACS, the mean for the COPD group was not significantly different compared to the healthy group ($F(1, 59) = 0.42, p=0.52$).

**Pulmonary Functioning**

COPD participants had significantly lower $FEV_1$ [$F (1, 59) = 48.24, p <.000$], lower $FEV_1\%$predicted [$F (1, 59) = 55.90, p <.000$], lower $FVC$ [$F (1, 59) = 19.39, p <.000$], lower $FVC\%$ [$F (1, 59) = 21.65, p <.000$], and lower $FEV_1/FVC$ [$F (1, 59) = 37.30, p <.000$] compared to healthy participants.

**Manipulation Check**

To ensure that changes in heart rate were not due to production of speech during the task phase, all participants were asked to read aloud a neutral script out loud about doing laundry to obtain a reading baseline. Independent t-tests for HRV in both time and frequency domains between the resting baseline and baseline reading task revealed no significant differences in mean RR ($t= 0.38, p =.70$), STDRR ($t=-1.10, p=0.27$), mean HR ($t=-0.46, p = 0.65$), RMSSD ($t=-0.56 (p = 0.58)$), log LF ($t=-1.04, p=0.30$), log HF ($t=-0.30, p=0.76$), and LF / HF ratio ($t=-1.45, p=0.15$).

**Correlations between variables**

**Pulmonary functioning and HRV**

Correlations of pulmonary functioning variables with HRV can be found in Table 3.

For the complete sample, baseline mean heart rate was associated with FVC%predicted ($r=-.28, p=0.03$). When data for COPD and healthy controls were
analyzed separately, significant correlations between pulmonary functioning and HRV were found in the healthy participants but not the COPD participants. Specifically, in the healthy groups (HEA and HEA-ANX), baseline LF/HF ratio was negatively associated with FEV$_1$% ($r=-.36$, $p=.05$).

**Health Behaviors and HRV**

Correlations of health behaviors with HRV can be found in Table 4.

Pack-year history was not significantly associated with heart rate variability measures.

Alcohol consumption was negatively associated with Baseline Mean RR ($\rho = -.34$, $p = 0.009$) and positively correlated with Baseline Mean HR ($\rho = .34$, $p = .009$).

The PSQI total score was not significantly associated with heart rate variability. However, sleep disturbances (subscale 5) on the PSQI was significantly associated with baseline log LF ($r=0.26$, $p = 0.05$).

Physical activity, indicated by VO$_{2\text{max}}$ scores calculated by the non-Exercise Houston questionnaire, was positively associated with baseline log LF ($r=0.29$, $p = 0.03$).

**Correlations between measures of distress, dispositional variables, and HRV**

Correlations between measures of distress, dispositional variables and HRV can be found in Table 4.

There were no significant associations between measures of distress and HRV, or between dispositional variables and HRV.

**Correlations between health behaviors, measures of distress and dispositional**
variables

Correlations between health behaviors, measures of distress, and dispositional variables can be found in Table 5.

There were no significant associations among health behaviors (PSQI, pack-year history, and VO$_{2\text{max}}$).

There were significant associations among measures of distress. PSS-10 was significantly correlated with PANAS-N ($r=0.27$, $p = 0.04$) and CESD ($r=0.74$, $p <0.001$). PANAS-P was significantly associated with PANAS-N ($r=0.74$, $p <0.001$). STAI-X2 was significantly associated with CESD ($r=0.84$, $p <0.001$).

There were significant associations among dispositional variables. PSWQ was significantly associated with RRS-R ($r=0.38$, $p = 0.003$), RRS-B ($r=0.62$, $p <0.001$), and ACS ($r=0.43$, $p = 0.001$). RRS-R was significantly associated with RRS-B ($r=0.57$, $p <0.001$) and MCSD ($r=-0.38$, $p = 0.002$). RRS-B was significantly associated with MCSD ($r=-0.28$, $p = 0.03$) and ACS ($r=0.49$, $p<0.001$). MCSD was significantly associated with ACS ($r=-0.30$, $p = 0.02$).

There were significant associations between health behaviors and measures of distress. PSQI was significantly associated with PSS-10 ($r=0.33$, $p = 0.01$), STAI-X2 ($r=0.41$, $p = 0.002$), and CESD ($r=0.51$, $p <0.001$).

There were significant associations between health behaviors and dispositional variables. PSQI was significantly associated with PSWQ ($r=0.33$, $p = 0.01$), RRS-R ($r=0.31$, $p = 0.02$), RRS-B ($r=0.38$, $p = 0.003$), and MCSD ($r=-0.28$, $p = 0.04$).

There were significant associations between measures of distress and
dispositional variables. PSS-10 was significantly associated with PSWQ (r=0.66, p <0.0001), RRS-R (r=0.46, p <0.001), and RRS-B (r=0.58, p <0.001). PANAS-N was significantly associated with PSWQ (r=0.33, p = 0.01). STAI-X2 was significantly associated with PSWQ (r=0.65, p <0.001), RRS-R (r=0.54, p <0.001), RRS-B (r=0.59, p <0.001), MCSD (r=-0.46, p <0.001), and ACS (r=0.54, p <0.001). CESD was significantly correlated with PSWQ (r=0.58, p <0.001), RRS-R (r=0.73, p <0.001), RRS-B (r=0.64, p <0.001), MCSD (r=-0.45, p <0.001), and ACS (r=0.29, p = 0.03).

Correlations of pulmonary functioning with health behaviors, measures of distress, and dispositional variables

Correlations of pulmonary functioning with health behaviors, measures of distress, and dispositional variables can be found in Table 6.

PANAS-P was significantly correlated with FEV₁% (r=0.27, p = 0.04) and FVC% (r=0.33, p = 0.01). Pack-year history was significantly correlated with FEV₁ (r= -0.49, p <0.001), FEV₁% (r= -0.60, p <0.001), FVC (r=-0.37, p = 0.004), FVC% (r=-0.50, p <0.01), and FEV₁/FVC(r=-0.50, p <0.001). VO₂max was significantly correlated with FEV₁ (r=0.53, p <0.001) and FVC (r=0.68, p <0.001).

Correlations of pulmonary functioning with health behaviors, measures of distress, and dispositional variables in only the COPD patients were also conducted. Pack-year history was associated with FEV₁ (r= -0.48, p=0.007), FEV₁/FVC (r=-0.36, p=0.05). Positive affectivity was associated with FEV₁% (r=0.38, p=0.04). VO₂max was associated with FEV₁ (r=0.38, p=0.04). MCSD was associated with FEV₁% (r=0.40, p=0.03). ACS was associated with FEV₁ (r= -
Comparisons of HRV between COPD-ANX vs. COPD, HEA, and HEA-ANX

Comparisons of Baseline values

Baseline HRV values in the COPD-ANX group and the other three groups combined can be found in Table 7.

Planned contrast analyses comparing the COPD-ANX group to the other three groups combined (COPD, HEA, and HEA-ANX) revealed significant differences in baseline HRV values for mean RR (t=-22.60, p<0.001), STDRR (t=-6.77, p<0.001), mean HR (t=-21.20, p<0.001), RMSSD (t=-5.11, p<0.001), log LF (t=-17.52, p<0.001), log HF (t=-10.97, p<0.001), and LF/HF ratio (t=-5.54, p<0.001). Mean RR, STDRR, log LF, and LF/HF ratio were significantly lower at baseline in the COPD-ANX group compared to the other three groups. In contrast, baseline mean HR, RMSSD, and log HF were significantly higher in the COPD-ANX group compared to the other three groups.

Comparisons across Phases

The COPD-ANX group was compared to the other three groups using repeated measures ANOVA, with group (COPD-ANX vs. COPD, HEA, HEA-ANX groups) as the between-subject factor and phase (Baseline, Task, Recovery) as the within-subject factor.

Mean RR. There was a significant phase effect [F(2, 114) = 107.42 p<.001], but no main effects of group status [F(1, 57) = 1.19, p=0.31] and no interactions of phase and group (p=0.76).

STDRR. There was a significant phase effect [F(2, 114) = 4.10, p=0.02], 
but no main effects of group status \( [F(1, 57) = 0.08, p=0.78] \) and no interactions of phase and group (p=0.53).

**Mean HR.** There was a significant phase effect \( [F(2, 114) = 106.82, p<.001] \), but no main effects of group status \( [F(1, 57) = 0, p=0.98] \) and no interactions of phase and group (p=0.68).

**RMSSD.** There was a significant phase effect \( [F(2, 114) = 5.82, p=0.004] \), but no main effects of group status \( [F(1, 57) = 0.30, p=0.58] \) and no interactions of phase and group (p=0.66).

**Log LF.** There was a significant phase effect \( [F(2, 104) = 6.04, p=0.003] \), but no main effects of group status \( [F(1, 52) = 0.02, p=0.88] \) and no interactions of phase and group (p=0.35).

**Log HF.** There was a significant phase effect \( [F(2, 110) = 6.79, p=0.002] \), but no main effects of group status \( [F(1, 55) = 0.12, p=0.73] \) and no interactions of phase and group (p=0.77).

**LF/HF ratio.** There was a significant phase effect \( [F(2, 110) = 9.38, p<0.001] \), but no main effects of group status \( [F(1, 55) = 1.64, p=0.21] \) and no interactions of phase and group (p=0.88).

**Comparisons of Heart Rate Variability and State Anxiety among 4 groups (COPD, COPD-ANX, HEA, HEA-ANX)**

Mean values for HRV measures for Baseline, Task, and Recovery phase can be found in Table 8. All HRV data were analyzed with 2 (disease status: COPD vs. Healthy) X 2 (Anxiety group: Anxious vs. Non-anxious) X 3 (Phase: Baseline vs. Task vs. Recovery) repeated measures ANOVA, with disease status...
and anxiety group as the between-subject factors and phase as the within-subject factor.

Comparisons of Baseline values

One-way ANOVA revealed no significant differences between groups for Mean RR, \( F(3, 60) = 0.36, p = 0.55 \), STDRR, \( F(3, 60) = 0.58, p = 0.63 \), Mean HR, \( F(3, 60) = 0.23, p = 0.87 \), RMSSD, \( F(3, 60) = 0.68, p = 0.57 \), Log LF, \( F(3, 60) = 0.59, p = 0.63 \), Log HF, \( F(3, 60) = 0.19, p = 0.90 \), and LF/HF ratio, \( F(3, 60) = 0.84, p = 0.48 \).

Repeated Measures ANOVA for HRV variables

**Mean RR**. There was a significant phase effect, \( F(2,110) = 139.59, p < .001 \), but no main effects of disease status, \( F(1, 55) = 0.23, p = 0.63 \), or anxiety group, \( F(1, 55) = 0.01, p = 0.92 \), and no interactions of disease status and anxiety group (p=0.95), phase and disease status (p=0.42), phase and anxiety group (p=0.14), or phase, disease status, and anxiety group (0.61). As expected, mean RR was decreased during the task and then increased during recovery.

**STDRR**. There was a significant phase effect, \( F(2,110) = 139.59, p = 0.03 \), but no main effects of disease status, \( F(1, 55) = 0.18, p = 0.67 \), or anxiety group, \( F(1, 55) = 0.34, p = 0.56 \), and no interactions of disease status and anxiety group (p=0.61), phase and disease status (p=0.43), or phase, disease status, and anxiety group (0.15). There was a statistical trend in the interaction of phase and anxiety group, \( F(2, 110) = 2.92, p = 0.07 \). STDRR for individuals in the anxious groups (COPD-ANX and HEA-ANX) increased from baseline to task to recovery. In contrast, STDRR decreased from baseline to task, and then increased from
task to recovery for the non-anxious group (COPD and HEA).

*Mean HR.* There was a significant phase effect [$F (2,110) = 140.16, p<.001$], but no main effects of disease status [$F(1, 55) = 0.28, p=0.60$] or anxiety group [$F(1, 55) = 0.02, p=0.89$], and no interactions of disease status and anxiety group ($p=0.72$), phase and disease status ($p=0.31$), phase and anxiety group ($p=0.35$) or phase, disease status, and anxiety group ($p=0.72$). As expected, mean HR increased during the task and then decreased during recovery.

*RMSSD.* There was a significant phase effect [$F(2,110) = 5.66, p=0.005$], but no main effects of disease status [$F(1, 55) = 1.05, p=0.31$] or anxiety group [$F(1, 55) = 0.02, p=0.90$], and no interactions of disease status and anxiety group ($p=0.84$), phase and disease status ($p=0.45$), phase and anxiety group ($p=0.13$) or phase, disease status, and anxiety group ($p=0.55$).

*Log LF.* There was a significant phase effect [$F (2,100) = 4.58, p=0.01$], but no main effects of disease status [$F (1, 50) = 0.11, p=0.75$] or anxiety group [$F(1, 50) = 0.38, p=0.54$], and no interactions of disease status and anxiety group ($p=0.63$), phase and disease status ($p=0.55$), or phase and anxiety group ($p=0.11$). There was, however, a significant three-way interaction of phase, disease status, and anxiety group [$F (2, 100) = 4.31, p = 0.02$]. Analysis of simple effects revealed a significant increase from Baseline to Recovery ($t = -2.63, p = 0.02$) for the COPD-ANX group. For the HEA group, there was a significant increase from Baseline to Task ($t = -3.56, p = 0.003$) and a significant decrease from Task to Recovery ($t=2.32, p = 0.04$). For the HEA-ANX group, there was a significant difference between the Baseline and Recovery phase ($t =$
Thus, it appears that both the anxious groups (COPD-ANX and HEA-ANX) had significant increases in log LF from Baseline to Recovery, and the HEA group exhibited the expected increase during the task. Figure 2 depicts Log LF values for the four groups across the Baseline, Task, and Recovery phases, plotted separately for disease status.

Log HF. There was a significant phase effect \[ F(2,106) = 8.84, p<0.001 \], but no main effects of disease status \[ F(1, 50) = 1.31, p=0.26 \] or anxiety group \[ F(1, 50) = 0.02, p=0.89 \], and no interactions for disease status and anxiety group \( p=0.65 \), phase and disease status \( p=0.37 \), or phase and anxiety group \( p=0.35 \). However, there was a significant three-way interaction of phase, disease status, and anxiety group \[ F (2, 106) = 3.78, p = 0.03 \]. Further investigation of the three-way interaction revealed a pattern similar to that observed in COPD-ANX and HEA-ANX groups. Analysis of simple effects revealed a significant decrease in log HF from baseline to task \( t=2.28, p=0.02 \) for the COPD group, and then subsequently an increase in parasympathetic activity from task to recovery \( t=-2.89, p=0.01 \). However, for both the COPD-ANX and HEA-ANX group, tests of simple effects revealed no significant difference in log HF from baseline to task, but a significant increase from task to recovery. For the HEA group, t-tests revealed no significant differences between the Baseline, Task, and Recovery phase. It appears that the HEA group did not respond to the psychological stressor, thus showing no changes in HRV activity. Figure 3 depicts Log HF values for the four groups across the Baseline, Task, and Recovery phase, plotted separately for disease status.
**LF/HF ratio.** There was a significant phase effect \([F(2, 106) = 13.23, p<0.001]\) and a significant main effect of disease status \([F(1, 53) = 4.72, p=0.03]\), but no significant effect of anxiety group \([F(1, 53) = 0.30, p=0.59]\). There were no interactions for disease status and anxiety group \((p=0.63)\), phase and disease status \((p=0.71)\), phase and anxiety group \((p=0.94)\) or phase, disease status, and anxiety group \((p=0.64)\). The COPD group had overall lower LF/HF ratios compared to the healthy group. As expected, LF/HF ratio was increased during the task and then increased during recovery.

**State Anxiety.** State anxiety was measured at baseline and immediately after the task. Repeated measures ANOVA revealed significant time \([F (3, 60) = 18.24, p<.001]\) and group \([F (3, 60) = 2.59, p<.001]\) effects, as well as a statistical trend for the Group X Time interaction \([F (3, 60) = 2.59, p = 0.06]\). Post-hoc analysis using Tukey’s HSD revealed significant differences between the anxious and non-anxious groups \((p<.001)\). Thus, state anxiety increased significantly from baseline to task following a psychological stressor in the COPD and HEA group (non-anxious groups), but the COPD-ANX and HEA-ANX groups (anxious groups) had elevated state anxiety at baseline and did not show an increase following the psychological stressor.

**Comparison of Anxiety Groups: COPD-ANX vs. HEA-ANX**

Means and standard deviations for all measures of health behaviors, measures of distress, dispositional variables, and pulmonary functioning are included in Table 9.

**Health Behaviors**
**Smoking.** There were no significant differences between the COPD-ANX and HEA-ANX groups for pack-year history \([F(1, 29) = 2.90, p=0.10]\).

**Alcohol intake.** There were no significant differences between the COPD-ANX (mode 1 – 5 alcoholic drinks) and HEA-ANX group (mode 1 – 5 alcoholic drinks) for alcohol intake \([X^2 = 2.20, p=0.70]\).

**Physical Activity.** There were no significant differences between the COPD-ANX and HEA-ANX group for VO2\text{max} \([F(1, 29) = 0.04, p=0.84]\).

**Sleep.** There were also significant differences on the total PSQI score between the COPD-ANX and HEA-ANX group, with the COPD-ANX group reporting lower sleep quality \([F (1, 27) = 7.53, p = 0.01]\). The COPD-ANX group had poorer subjective sleep quality than the HEA-ANX group \([F(1, 28) = 7.27, p = 0.01]\) as well as shorter sleep duration \([F(1, 29) = 4.07, p = 0.05]\).

**Measures of Distress**

**Stress.** There were no significant differences between the COPD-ANX and HEA-ANX group on the PSS-10 \([F (1, 29) = 0.17, p = 0.69]\).

**Positive/Negative Affect.** There were no significant differences between the COPD-ANX and HEA-ANX group for positive and negative affect \([F(1, 29) = 1.78, p=0.20; F(1, 29) = 3.48, p = 0.07, respectively]\).

**Anxiety.** There were no significant differences between the COPD-ANX and HEA-ANX group for trait anxiety on the STAI-X2 \([F(1, 29) = 0.10, p=0.75]\).

**Depression.** There were no significant differences between the COPD-ANX and HEA-ANX group for depression on the CESD \([F(1, 29) = 0.30, p=0.59]\).

**Dispositional Variables**
Worry. There were no significant differences between the COPD-ANX and HEA-ANX group for worry on the PSWQ [F(1, 29) = 0.01, p=0.92].

Rumination. There were no significant differences between the COPD-ANX and HEA-ANX group for rumination on the RRS-R or RRS-B [F(1, 29) = 0.06, p=0.92; F (1, 29) = 1.66, p = 0.21].

Social Desirability Scale. There were no significant differences between the COPD-ANX and HEA-ANX group for social desirability on the MCSD [F(1, 29) = 0.02, p = 0.89].

Attentional Control. There were no significant differences between the COPD-ANX and HEA-ANX group for attentional control on the ACS [F(1, 29) = 0.03, p = 0.87].

Heart Rate Variability

One-way ANOVA revealed no significant differences between groups at baseline for Mean RR, [F(1, 29) = 0.32, p = 0.58]), STDRR [F(1, 29) = 0.02, p=0.88]), Mean HR [F(1, 29) = 0.09, p=0.77]), RMSSD [F(1, 29) = 0.66, p=0.42]), Log LF [F(1, 29) = 1.11, p=0.30]), Log HF [F(1, 29) = 0.12, p=0.73]), and LF/HF ratio [F(1, 29) = 0.2.22, p=0.15]).

Comparisons between COPD vs. COPD-ANX group

Means and standard deviations for all measures of pulmonary functioning, measures of distress, dispositional variables, and pulmonary functioning are included in Table 10.

Analyses to compare only the COPD group and the COPD-ANX group were conducted. As indicated above, the two groups did not differ in disease
severity. The COPD group had higher pack-year history (t = 2.45, p=0.02) compared to the COPD-ANX group. The two groups did not differ in the number of medications taken (6.93 vs. 6.40, p = 0.75). The COPD group also had significantly lower scores on the PSS (t = -3.42, p = 0.002), PSWQ (t = -3.10, p = 0.004), RRS-R (t = -2.50, p = 0.02), RRS-B (t = -3.51, p = 0.002), STAI-X2 (t = -3.96, p=0.001), CESD (t = -4.18, p<.000), and lower scores on the ACS (t = -2.08, p=0.05) compared to the COPD-ANX group. The COPD group also had better subjective sleep quality (t = -3.78, p=0.001), more total sleep time (t = -2.19, p=0.04), and had less daytime dysfunction (t = -2.57, p=0.02) on the PSQI subscales, and had overall lower scores on the PSQI (t = -3.49, p = 0.002). Thus, although the COPD group had a greater smoking history, the COPD and COPD-ANX group did not differ in disease severity and the COPD-ANX group had significantly more stress, higher levels of worry and rumination, higher anxiety and depression, and decreased attentional control. The COPD-ANX group also had significantly worse sleep quality, including less sleep and had higher daytime dysfunction.

**Comparisons within the COPD-ANX group**

Further exploratory analyses were conducted for anxiety duration within the COPD-ANX group. Individuals in the COPD-ANX group were further divided into two groups depending on whether their anxiety was pre-existing (longer than diagnosis of COPD) or whether they started experiencing symptoms of anxiety when they were diagnosed with COPD. Six individuals reported having anxiety after their diagnosis of COPD, and nine individuals reported that their anxiety had
developed prior to their COPD diagnosis. There were no differences between the two groups for age, gender, income, marital status, and ethnicity. Individual t-tests revealed there were no differences between the two groups for measures of distress, dispositional variables, pulmonary functioning variables, or number of medications. Analyses of health behaviors revealed that there was a significant difference between the two groups for habitual sleep efficiency (0.60 vs. 2.22; t=-2.21, p<0.05). Means and standard deviations of these variables can be found in Table 1.

There were significant differences in baseline HRV between these two groups. Individuals who had preexisting anxiety prior to their COPD diagnosis had lower vagal tone at baseline, indicated by log HF (4.81 vs. 3.14; t = 2.69, p=0.02). There was also a trend approaching significance for baseline STDRR, a measure of global HRV (21.78 vs. 13.45; t=1.89, p=0.08), and also RMSSD, an HRV index measured in the time domain (24.79 vs. 11.12; t=2.05, p=0.06).

There was also a trend toward a group effect for log HF [F (1, 13) = 3.69, p=0.08]. Change scores (Task log HF – Baseline log HF) were -0.66 for individuals with preexisting anxiety and 0.61 for individuals whose anxiety developed after the diagnosis (p=0.05). This indicates that individuals with preexisting anxiety experienced an increase in parasympathetic response during the task phase, whereas individuals without preexisting anxiety experienced a more typical decrease in parasympathetic response. Figure 3 depicts log HF responses of the groups.
CHAPTER 5. DISCUSSION

The current investigation was the first to examine the effects of a psychological stressor on HRV comparing 30 COPD patients with 30 age- and gender-matched healthy individuals who presented as both anxious and non-anxious. Additional factors, including health behaviors, measures of distress, and dispositional variables were also measured to compare differences between the groups. Results of the study addressed both physiological and psychological functioning in the groups studied.

For physiological variables, the primary results were (1) no baseline differences in HRV between groups, (2) a blunted HRV response found in the anxious groups compared to the COPD group, (3) an increase in parasympathetic nervous system activity for the anxious groups when exposed to a stressor and when the stressor was removed, a pattern that was not found in non-anxious groups, and (4) decreased parasympathetic nervous system activity in COPD patients with pre-existing anxiety prior to diagnosis compared to COPD patients whose anxiety symptoms started with their diagnosis.

COPD was expected to be associated with lower resting HRV compared to healthy controls, and the combination of both COPD and high levels of anxiety was expected to be associated with the lowest HRV compared to the other three groups (COPD, HEA, and HEA-ANX). However, results of this study revealed
that resting HRV values for the COPD-ANX group were significantly higher in RMSSD and log HF rather than lower when compared to the other three groups (COPD, HEA, and HEA-ANX).

There were no differences in resting HRV when comparing the four groups, or between the COPD and healthy controls. Although this is consistent with one prior study showing no differences (Bartels et al., 2003), other studies have found decreased global HRV in COPD patients compared to healthy controls (Volterrani et al., 1994; Stein et al., 1998). The differences found across studies may be associated with short-term and long-term recordings of HRV used in the studies. In the Volterrani et al. (1994) study, there was elevated HRV in short-term recordings of standardized HF measures among COPD participants compared to healthy controls at rest and in response to a passive sympathetic stimulation. Additionally, Bartels et al. (2003) found COPD patients displayed no resting differences in short-term recordings of parasympathetic nervous system activity, but found elevated parasympathetic activity in response to exercise when compared to healthy controls. On the other hand, in long-term recordings, Stein et al. (1998) found decreased global HRV when COPD patients were compared to healthy controls. Thus, it is possible that COPD patients may have overall decreased global HRV, or cardiac vagal tone, when long-term recordings of HRV are used. However, when short-term recordings of HRV are used, as in this study, COPD patients appear to have increased HRV compared to healthy controls, especially in response to sympathetic stimulation such as exercise or a stressor. This may be explained, in part, by the influence of breathing against
resistance or obstructed breathing affecting cardiac function in COPD patients (Buda et al., 1979).

Second, similar patterns were also found across the COPD-ANX and HEA-ANX groups in their responses to the speech task. There was an interesting difference between the COPD group compared to the pattern of responses from the COPD-ANX and HEA-ANX group. The COPD group did not exhibit increased sympathetic activity as reflected in the lack of change in log LF across the Baseline, Task, and Recovery phases. In contrast, the COPD-ANX and HEA-ANX group showed a significantly increasing pattern of sympathetic activity, demonstrated by changes between Baseline and Recovery.

There were also different patterns between groups in parasympathetic nervous system activity. The COPD group displayed a decrease in parasympathetic nervous system activity, indicated by log HF, when exposed to a psychological stressor, and then subsequently an increase in parasympathetic activity following the task phase. In contrast, the COPD-ANX and HEA-ANX groups did not exhibit change in HRV in response to the speech task. This is consistent with past literature indicating that anxious individuals display a blunted response to a stressor as they tend to have more anticipatory anxiety at baseline (Hoen-Saric & McLeod, 1988). Although there were no differences in resting HRV between the four groups in this sample, the response of individuals in the COPD-ANX and HEA-ANX to stress appeared to be blunted compared to the COPD group. However, because all three groups (COPD, COPD-ANX, and HEA-ANX) were at similar levels at recovery, further examination of baseline
differences may be important.

Similar patterns that reflected blunted responses in the COPD-ANX group and HEA-ANX group compared to the COPD and HEA group were also found in state anxiety before and after exposure to the stressor. Individuals in the COPD and HEA group had significant increases from baseline to task in state anxiety, while the COPD-ANX and HEA-ANX groups did not. Consistent with the HRV data, this suggests that individuals in the COPD-ANX group and HEA-ANX group had higher levels of anticipatory anxiety prior to the stressor, reflected by their lack of change in state anxiety prior to and after the stressor.

There was a similar HRV response pattern found across phases for the COPD-ANX and HEA-ANX groups, with both groups exhibiting increased STDRR across phases. In contrast, the COPD and HEA groups exhibited an initial decrease at task and an increase at recovery, which would be expected in this paradigm, as individuals with flexible responses should display a decrease in parasympathetic activity when exposed to a stressor, and subsequently an increase when the stressor is removed. A similar pattern was found with log HF indices. Log HF continued to increase across phases in both COPD-ANX and HEA-ANX groups, while the COPD group exhibited an initial decrease at task and an increase at recovery, the expected response in this paradigm. Past studies have reported that intrathoracic pressure swings that occur during obstructed breathing may cause fluctuation in cardiac performance (Buda et al., 1979). The unexpected increasing parasympathetic response pattern found in the COPD-ANX and HEA-ANX groups could be explained in part by increased
dyspnea accompanying anxiety, regardless of COPD status. Past literature has suggested that increased parasympathetic activity may be potentiated during dynamic hyperinflation and increased end-expiratory lung volumes (Marin et al., 2001). It is possible that exposure to a psychological stressor may have caused the anxious individuals (COPD-ANX and HEA-ANX groups) to breathe in a manner that caused dynamic hyperinflation and increased end-expiratory lung volumes and a prolonged increase in parasympathetic activity (log HF).

Increasing parasympathetic activity following termination of a stressor may also reflect the fact that anxious individuals have a diminished ability to flexibly respond to a stressor (Thayer, Friedman, & Borkovec, 1996; Thayer & Lane, 2002).

Individuals who reported pre-existing high levels of anxiety prior to their COPD diagnosis exhibited reduced vagal activity at rest compared to individuals whose anxiety reportedly started after their COPD diagnosis. Analyses revealed that the COPD-ANX individuals with pre-existing anxiety had a similar increasing linear pattern in parasympathetic response from Baseline to Task to Recovery phase, whereas the COPD-ANX individuals without pre-existing anxiety displayed a quadratic pattern that was similar to the COPD and HEA group. This suggests that there may be relevant subgroups within the COPD population, and that individuals who experience anxiety following their diagnosis of COPD may be more similar to COPD patients without anxiety, as it may be a systemic consequence of their illness. In contrast, individuals with pre-existing anxiety prior to their COPD diagnosis may exhibit patterns reflecting greater rigidity in
responding to their environment. This can be explained, in part, by the cognitive behavioral model. The cognitive behavioral model proposes that COPD patients may misinterpret bodily sensations arising from dyspnea and hyperventilation as harmful, which subsequently leads to increased sensitivity to physiological arousal that escalates into a panic reaction, and worsens their respiratory symptoms. Individuals with a rigid pattern of responding may be more prone to misinterpret bodily sensations through repeated experiences of dyspnea, thus making them more likely to develop panic disorder.

With regard to health behaviors, measures of distress, and dispositional traits, there were significant differences between the four groups in sleep, stress, trait anxiety, depression, worry, and rumination. Consistent with past literature, individuals in the COPD-ANX and HEA-ANX group reported higher levels of stress, trait anxiety, depression, and worry. Additionally, the COPD-ANX group reported significantly higher levels of rumination compared to the COPD, HEA, and HEA-ANX groups. Additionally, the COPD-ANX participants reported significantly higher levels of sleep disturbances, especially worse subjective sleep quality, compared to the COPD, HEA, and HEA-ANX groups. Thus, it appears that having both COPD and high levels of anxiety negatively impacts sleep above and beyond having COPD or high levels of anxiety alone. Additionally, individuals with both COPD and high levels of anxiety have significantly higher levels of rumination compared to the other three groups, indicating they may be at higher risk for prolonging a stressor and maintaining symptoms of depression and anxiety.
The COPD-ANX group reported worse emotional functioning than the COPD group as indicated by higher levels of stress, worry, rumination, anxiety, depression, and decreased attentional control. Thus, COPD patients with anxiety may be at higher risk for increased functional disability, despite having similar disease severity compared to COPD patients without anxiety. Additionally, research indicates that patients with better emotional functioning tend to participate more actively in treatment and gain more benefit from treatment, leading to better treatment outcomes (McSweeny, 1988). Thus, among patients with COPD, it will be important to identify individuals with comorbid anxiety, and focus on concurrent treatment of anxiety.

Sleep was a health domain in which the COPD-ANX group differed significantly from the other groups. The COPD-ANX group reported significantly lower subjective sleep quality, less total sleep time, and higher daytime dysfunction than the COPD group. Individuals with pre-existing anxiety prior to COPD diagnosis appeared to be especially affected. Individuals with pre-existing anxiety reported spending more time in bed despite similar total sleep times, resulting in reduced sleep quality. Past research has found that persistent insomnia is associated with significant impairments of daytime functioning, reduced quality of life, and heightened risks for psychiatric illness when not treated (Morin et al., 2009). Additionally, research has indicated that behavioral treatment of insomnia is helpful in alleviating comorbid psychiatric illnesses (Manber et al., 2008). A component of behavioral treatment of insomnia is to restrict time spent in bed to increase sleep efficiency [(Time asleep / Time spent
in bed) X 100]. Because COPD-ANX patients reported spending more time in bed despite total sleep times that were similar to non-anxious patients, COPD patients with anxiety may be especially candidates for receiving behavioral treatments of insomnia.

This is the first investigation examining HRV as a possible common physiological mechanism in high levels of anxiety and COPD. Although the HRV pattern for individuals with both COPD and anxiety did not differ from that of other study participants (i.e., COPD patients without anxiety, healthy individuals, and healthy anxious individuals, results suggested that anxious individuals displayed smaller responses to stressful stimuli. It is possible that past studies investigating HRV in patients with COPD may have included a heterogeneous sample with and without anxiety, and previous studies of HRV in COPD patients may have been affected by the high prevalence of anxiety in this population, as none of the previous studies in this area measured anxiety in COPD patients.

Additionally, the COPD group in this sample appeared to be relatively high functioning. With the exception of pulmonary functioning, sleep and pack-year history, the COPD patients did not differ from healthy controls in terms of physical activity, measures of distress, and dispositional variables. This may be explained, in part, by the fact that many COPD patients were recruited from a pulmonary rehabilitation program and may have been more active and had greater access to health care resources compared to the typical COPD patient. Thus, the COPD sample in this study may not have been representative of the COPD population.
The lack of HRV differences between the COPD group and the healthy control groups may be due to long-term use of beta-agonist inhalers in COPD patients. Unfortunately, duration of drug use was not measured in this study, although use of beta-agonist inhalers is relatively common among COPD patients. Also, it is common for COPD patients to be treated with cycles of short-term steroids, which may alter cardiovascular response. Thus, although patients were asked to refrain from using medication prior to participating, medication use may have been a confound in this investigation.

Correlations among pulmonary functioning variables and measures of distress were low. Overall, the lack of association between pulmonary functioning variables and measures of distress is not surprising in light of past research revealing minimal correlations of mood status with pulmonary function (Engstrom et al., 1996). These results suggest that emotional well-being does not appear to be related to clinical indicators of lung function, but may be more closely related to strategies utilized to cope with a chronic illness.

An additional limitation is the small sample size employed in the current study. Experimental hypotheses were tested utilizing a sample of 60 participants, with 15 participants in each group. With a limited sample size, there is a smaller probability of detecting true relationships that may exist among study outcomes. Additionally, the sample may have been further compromised as the COPD-ANX group included individuals with pre-existing anxiety prior to COPD diagnosis as well as individuals whose anxiety had developed after their COPD diagnosis. A larger sample size would allow for greater opportunity to identify the effects of
stress on HRV in COPD patients presenting with and without anxiety.
CHAPTER 6. CONCLUSION

This study evaluated physiological and psychological factors associated with elevated anxiety symptoms in COPD patients. From a physiological perspective, no unique HRV effects were observed in the COPD population. However, the HRV response to stress in anxious individuals with and without COPD appeared to be blunted. Future studies of COPD patients and healthy controls investigating HRV should account for anxiety to avoid the potential confound of high anxiety levels among patients with COPD. Furthermore, it may be important to distinguish between individuals who have pre-existing anxiety prior to their COPD diagnosis and individuals who developed anxiety after their COPD diagnosis, as these two groups may have heterogeneous characteristics. Additionally, it will be important to ensure that psychological stressors in future studies elicit a stress response in both healthy and COPD patients.

From a psychological perspective, COPD and anxiety in combination was associated with higher levels of stress, trait anxiety, depression, rumination and worry compared to COPD patients without anxiety. COPD patients with anxiety also had poorer sleep quality and higher levels of rumination than patients with COPD or anxiety alone. This indicates that having both COPD and anxiety may negatively impact the patient’s quality of life. Although this study did not specifically investigate the effect of anxiety on pulmonary functioning and
dyspnea when exposed to a stressor, past research has reported that COPD patients with anxiety may experience increased dyspnea, which may escalate into panic. Furthermore, past literature has indicated that having both a pre-existing mental illness and a comorbid chronic illness may decrease functional ability more than the effect of either condition alone (Borson et al., 1992; Unutzer et al., 1997). Thus, it is important to identify anxious individuals within the COPD population and provide appropriate therapeutic interventions.

The findings from this study demonstrated that COPD patients with anxiety displayed a blunted response to a stressor, which was similar to healthy, anxious individuals without COPD. Although this pattern of findings was not unique to COPD patients, evidence of inflexible autonomic response may suggest the need for therapeutic interventions that can restore autonomic balance. Interventions such as stress management and cognitive behavioral therapy are techniques that may be effective for COPD patients with anxiety not only to improve perception of pulmonary functioning but also to improve psychological well-being by decreasing anxiety (Livermore et al., 2008).
<table>
<thead>
<tr>
<th></th>
<th>COPD (n=30)</th>
<th></th>
<th>HEALTHY (n=30)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>N(%)</td>
<td>M (SD)</td>
<td>N(%)</td>
</tr>
<tr>
<td>Age</td>
<td>59.1 (11.24)</td>
<td>15 (50%)</td>
<td>59.2 (11.28)</td>
<td>15 (50%)</td>
</tr>
<tr>
<td>Years of Education</td>
<td>14.37 (2.7)</td>
<td>12 (40%)</td>
<td>16.0 (3.5)</td>
<td>4 (13.33%)</td>
</tr>
<tr>
<td>Years since Diagnosis</td>
<td>4.9 (3.5)</td>
<td>2 (6.7%)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>26600 (21500)</td>
<td>11 (36.7%)</td>
<td>31700 (27600)</td>
<td>11 (36.7%)</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Gender</th>
<th>COPD (n=30)</th>
<th>HEALTHY (n=30)</th>
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</thead>
<tbody>
<tr>
<td>Male</td>
<td>15 (50%)</td>
<td>15 (50%)</td>
</tr>
<tr>
<td>Female</td>
<td>15 (50%)</td>
<td>15 (50%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Race</th>
<th>COPD (n=30)</th>
<th>HEALTHY (n=30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CA</td>
<td>17 (56.67%)</td>
<td>23 (76.67%)</td>
</tr>
<tr>
<td>AA</td>
<td>12 (40%)</td>
<td>4 (13.33%)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>0 (0%)</td>
<td>1 (3.33%)</td>
</tr>
<tr>
<td>Asian</td>
<td>1 (3.33%)</td>
<td>2 (6.67%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Marital Status</th>
<th>COPD (n=30)</th>
<th>HEALTHY (n=30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single</td>
<td>6 (20.7%)</td>
<td>5 (16.7%)</td>
</tr>
<tr>
<td>Married</td>
<td>10 (34.5%)</td>
<td>9 (30%)</td>
</tr>
<tr>
<td>Divorced/Separated</td>
<td>10 (34.5%)</td>
<td>13 (43.3%)</td>
</tr>
<tr>
<td>Widowed</td>
<td>3 (10.3%)</td>
<td>3 (10%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Living Arrangements</th>
<th>COPD (n=30)</th>
<th>HEALTHY (n=30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alone</td>
<td>11 (36.7%)</td>
<td>11 (36.7%)</td>
</tr>
<tr>
<td>With Spouse / SO</td>
<td>12 (40%)</td>
<td>10 (33.3%)</td>
</tr>
<tr>
<td>With Children</td>
<td>2 (6.7%)</td>
<td>1 (3.3%)</td>
</tr>
<tr>
<td>With children and spouse</td>
<td>2 (6.7%)</td>
<td>6 (20%)</td>
</tr>
<tr>
<td>With parents</td>
<td>0 (0%)</td>
<td>1 (3.3%)</td>
</tr>
<tr>
<td>With friends</td>
<td>3 (10%)</td>
<td>1 (3.3%)</td>
</tr>
</tbody>
</table>

Note.  
M = Mean  
SD = Standard Deviation

**Table 1: Demographic Characteristics of Participants**
<table>
<thead>
<tr>
<th></th>
<th>COPD (n=30) M (SD)</th>
<th>HEALTHY (n=30) M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV$_1$</td>
<td>1.45 (0.67)</td>
<td>2.79 (0.81) ***</td>
</tr>
<tr>
<td>FEV$_1$/FVC</td>
<td>54.03 (22.11)</td>
<td>94.73 (19.99) ***</td>
</tr>
<tr>
<td></td>
<td>2.29 (.094)</td>
<td>3.4 (1.01) ***</td>
</tr>
<tr>
<td></td>
<td>65.40 (20.77)</td>
<td>88.80 (18.10) ***</td>
</tr>
<tr>
<td></td>
<td>0.63 (0.15)</td>
<td>0.82 (0.06) ***</td>
</tr>
</tbody>
</table>

**Health Behaviors**

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pack Year</td>
<td>33.13 (27.83)</td>
<td>7.07 (12.52) ***</td>
</tr>
<tr>
<td>V0$_{max}$</td>
<td>20.71 (10.96)</td>
<td>23.92 (10.39)</td>
</tr>
<tr>
<td>PSQI</td>
<td>9.37 (4.5)</td>
<td>6.43 (3.76) **</td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>8 (26.7%)</td>
<td>6 (20%)</td>
</tr>
<tr>
<td>1</td>
<td>6 (20%)</td>
<td>6 (20%)</td>
</tr>
<tr>
<td>1-5</td>
<td>11 (36.7%)</td>
<td>15 (50%)</td>
</tr>
<tr>
<td>6-10</td>
<td>3 (10%)</td>
<td>2 (6.7%)</td>
</tr>
<tr>
<td>11-15</td>
<td>0 (0%)</td>
<td>1 (3.3%)</td>
</tr>
<tr>
<td>16-20</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>20&gt;</td>
<td>2 (6.7%)</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

**Distress**

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>PSS-10</td>
<td>18.33 (7.62)</td>
<td>17.06 (6.23)</td>
</tr>
<tr>
<td>PANAS-POS</td>
<td>28.4 (3.73)</td>
<td>29.23 (4.37)</td>
</tr>
<tr>
<td>PANAS-NEG</td>
<td>25.9 (4.48)</td>
<td>26.53 (4.43)</td>
</tr>
<tr>
<td>STAI-X2</td>
<td>46.83 (12.68)</td>
<td>44.6 (14.93)</td>
</tr>
<tr>
<td>CESD</td>
<td>18.47 (11.92)</td>
<td>15.71 (12.09)</td>
</tr>
</tbody>
</table>

**Dispositional**

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>PSWQ</td>
<td>45.10 (12.13)</td>
<td>44.63 (12.69)</td>
</tr>
<tr>
<td>RRS-R</td>
<td>9.67 (3.34)</td>
<td>10.0 (3.3)</td>
</tr>
<tr>
<td>RRS-B</td>
<td>10.43 (3.62)</td>
<td>10.03 (2.57)</td>
</tr>
<tr>
<td>MCSD</td>
<td>7.13 (2.70)</td>
<td>7.13 (3.29)</td>
</tr>
<tr>
<td>ACS</td>
<td>53.47 (9.07)</td>
<td>51.97 (8.92)</td>
</tr>
</tbody>
</table>

**Abbreviations:**
FEV$_1$: Forced Expiratory Volume in 1 second; FVC: Forced Vital Capacity; PSQI: Pittsburgh Sleep Quality Index; PSS-10: Perceived Stress Scale – 10 Items; PANAS-POS: Positive and Negative Affectivity Scales, Positive Subscale; PANAS-NEG: Positive and Negative Affectivity Scales, Negative Subscale; STAI-X2: State-Trait Anxiety Inventory – Trait version; CESD: Center for Epidemiology Studies – Depression; PSWQ: Penn State Worry Questionnaire; RRS-R: Ruminative Response Scale – Ruminative Subscale; RRS-B: Ruminative Response Scale – Brooding Subscale; MCSD: Marlowe-Crowne Social Desirability Subscale; ACS: Attentional Control Scale; *p<0.05, **p<0.01, ***p<0.001

**Table 2:** Pulmonary Functioning and health behaviors, measures of distress, and dispositional variables of Participants
<table>
<thead>
<tr>
<th></th>
<th>FEV₁</th>
<th>FEV₁%</th>
<th>FVC</th>
<th>FVC%</th>
<th>FEV₁/FVC</th>
</tr>
</thead>
<tbody>
<tr>
<td>STDRR</td>
<td>-0.03</td>
<td>-0.05</td>
<td>0.03</td>
<td>0.02</td>
<td>-0.14</td>
</tr>
<tr>
<td>Mean HR</td>
<td>-0.13</td>
<td>-0.21</td>
<td>-0.18</td>
<td>-0.28*</td>
<td>-0.02</td>
</tr>
<tr>
<td>Mean RR</td>
<td>0.12</td>
<td>0.17</td>
<td>0.17</td>
<td>0.25</td>
<td>-0.03</td>
</tr>
<tr>
<td>RMSSD</td>
<td>-0.10</td>
<td>-0.05</td>
<td>-0.04</td>
<td>0.03</td>
<td>-0.18</td>
</tr>
<tr>
<td>log LF</td>
<td>0.07</td>
<td>-0.03</td>
<td>0.11</td>
<td>0.02</td>
<td>-0.08</td>
</tr>
<tr>
<td>log HF</td>
<td>-0.001</td>
<td>-0.06</td>
<td>0.05</td>
<td>-0.02</td>
<td>-0.16</td>
</tr>
<tr>
<td>LF/HF</td>
<td>0.07</td>
<td>0.05</td>
<td>0.02</td>
<td>0.004</td>
<td>0.17</td>
</tr>
</tbody>
</table>

**Abbreviations:** FEV₁: Forced Expiratory Volume in 1 second; FVC: Forced Vital Capacity; Mean RR= mean interbeat intervals; Mean HR= mean heart rate; STDRR= standard deviation of normal – normal beats; RMSSD= square root of the mean squared differences of successive normal – normal intervals; Log LF= natural ln transformed low frequency value; Log HF= natural ln transformed high frequency value; LF/HF= low frequency / high frequency ratio; *p<0.05

Table 3. Correlation coefficients between resting heart rate variability and pulmonary functioning variables
<table>
<thead>
<tr>
<th></th>
<th>STDRR</th>
<th>Mean RR</th>
<th>Mean HR</th>
<th>RMSSD</th>
<th>logLF</th>
<th>logHF</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSS-10</td>
<td>.08</td>
<td>.009</td>
<td>-.07</td>
<td>.05</td>
<td>.10</td>
<td>.11</td>
<td>.001</td>
</tr>
<tr>
<td>PANAS-P</td>
<td>-.10</td>
<td>.08</td>
<td>-.04</td>
<td>-.04</td>
<td>-.13</td>
<td>-.09</td>
<td>.09</td>
</tr>
<tr>
<td>PANAS-N</td>
<td>-.21</td>
<td>-.14</td>
<td>.16</td>
<td>-.19</td>
<td>-.18</td>
<td>-.21</td>
<td>.16</td>
</tr>
<tr>
<td>STAI-X2</td>
<td>.05</td>
<td>.01</td>
<td>-.06</td>
<td>-.03</td>
<td>.15</td>
<td>.06</td>
<td>-.10</td>
</tr>
<tr>
<td>CESD</td>
<td>.14</td>
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**Abbreviations:** Mean RR= mean interbeat intervals; Mean HR= mean heart rate; STDRR= standard deviation of normal – normal beats; RMSSD= square root of the mean squared differences of successive normal – normal intervals; Log LF= natural ln transformed low frequency value; Log HF= natural ln transformed high frequency value; LF/HF= low frequency / high frequency ratio; ; PSS-10: Perceived Stress Scale – 10 Items; PANAS-POS: Positive and Negative Affectivity Scales, Positive Subscale; PANAS-NEG: Positive and Negative Affectivity Scales, Negative Subscale; STAI-X2: State-Trait Anxiety Inventory – Trait version; CESD: Center for Epidemiology Studies – Depression; PSWQ: Penn State Worry Questionnaire; RRS-R: Ruminative Response Scale – Ruminative Subscale; RRS-B: Ruminative Response Scale – Brooding Subscale; MCSD: Marlow-Crowne Social Desirability Subscale; ACS: Attentional Control Scale; PSQI = Pittsburgh Sleep Quality Inventory; VO₂max: maximal oxygen consumption. * p<0.05, ** p< 0.01

**Table 4. Correlation coefficients between resting heart rate variability and health behaviors, measures of distress, and dispositional variables**
<table>
<thead>
<tr>
<th>PSS-10</th>
<th>PANAS-P</th>
<th>PANAS-N</th>
<th>STAI-X2</th>
<th>CESD</th>
<th>PSWQ</th>
<th>RRS-R</th>
<th>RRS-B</th>
<th>MCSD</th>
<th>ACS</th>
<th>PSQI</th>
<th>PY</th>
<th>VO&lt;sub&gt;2max&lt;/sub&gt;</th>
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<td>VO&lt;sub&gt;2max&lt;/sub&gt;</td>
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**Abbreviations:** PSS-10: Perceived Stress Scale – 10 Items; PANAS-P: Positive and Negative Affectivity Scales, Positive Subscale; PANAS-N: Positive and Negative Affectivity Scales, Negative Subscale; STAI-X2: State-Trait Anxiety Inventory – Trait version; CESD: Center for Epidemiology Studies – Depression; PSWQ: Penn State Worry Questionnaire; RRS-R: Ruminative Response Scale – Ruminative Subscale; RRS-B: Ruminative Response Scale – Brooding Subscale; MCSD: Marlow-Crowne Social Desirability Subscale; ACS: Attentional Control Scale; PSQI: Pittsburgh Sleep Quality Index; PY: Pack Year; VO<sub>2max</sub>: maximal oxygen consumption.

* p<0.05, ** p<0.01

**Table 5. Correlations among health behaviors, measures of distress, and dispositional variables**
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<tr>
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<th>FEV&lt;sub&gt;1&lt;/sub&gt;%</th>
<th>FVC</th>
<th>FVC%</th>
<th>FEV&lt;sub&gt;1&lt;/sub&gt;/FVC</th>
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<td>0.68**</td>
<td>0.25</td>
<td>-0.06</td>
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**Abbreviations:** FEV<sub>1</sub>: Forced Expiratory Volume in 1 second; FVC: Forced Vital Capacity; PSQI: Pittsburgh Sleep Quality Index; PSS-10: Perceived Stress Scale – 10 Items; PANAS-POS: Positive and Negative Affectivity Scales, Positive Subscale; PANAS-NEG: Positive and Negative Affectivity Scales, Negative Subscale; STAI-X2: State-Trait Anxiety Inventory – Trait version; CESD: Center for Epidemiology Studies – Depression; PSWQ: Penn State Worry Questionnaire; RRS-R: Ruminative Response Scale – Ruminative Subscale; RRS-B: Ruminative Response Scale – Brooding Subscale; MCSD: Marlow-Crowne Social Desirability Subscale; ACS: Attentional Control Scale; Pittsburgh Sleep Quality Index; VO<sub>2</sub>max: maximal oxygen consumption.

* p<0.05, ** p<0.01

**Table 6:** Correlation coefficients between health behaviors, measures of distress, dispositional variables and pulmonary functioning variables
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<td>9 (20%)</td>
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<td>1&lt;</td>
<td>2 (13.3%)</td>
<td>10 (22.2%)</td>
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<tr>
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<td>11-15</td>
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<td>1 (2.2%)</td>
</tr>
<tr>
<td>16-20</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>20&gt;</td>
<td>1 (6.7%)</td>
<td>1 (2.2%)</td>
</tr>
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<td>16.13 (6.59)*</td>
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<td>29.27 (4.13)</td>
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<td>PANAS-NEG</td>
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<td>25.27 (3.9)</td>
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<td>STAI-X2</td>
<td>48.62 (9.61)</td>
<td>38.73 (11.26)**</td>
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<tr>
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<td>25.73 (14.14)</td>
<td>14.14 (10.87)**</td>
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<td>PSWQ</td>
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<td>42.64 (13.09)*</td>
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<td>9.51 (2.43)**</td>
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<td>Baseline HRV</td>
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<td>Log HF</td>
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<td>LF / HF ratio</td>
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<td>5.22 (4.22)</td>
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* p<0.05, ** p< 0.01, *** p<0.001

Table 7. Differences in COPD-ANX group compared to COPD, HEA, and HEA-ANX group combined on health behaviors, measures of distress, dispositional variables, and HRV
<table>
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<th>HEA-ANX (n=30)</th>
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<td>COPD-ANX M (SD)</td>
<td>HEA M (SD)</td>
<td>HEA-ANX M (SD)</td>
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<tr>
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<td>803.07(128.28)</td>
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<td>704.75(120.54)</td>
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<tr>
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<td>834.14(144.04)</td>
<td>845.58(134.76)</td>
<td>847.98(148.17)</td>
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<tr>
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<tr>
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<td>794.48 (128.60)</td>
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<td>703.63 (124.06)</td>
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<td>74.08 (12.62)</td>
<td>72.82 (13.31)</td>
<td>73.13 (14.15)</td>
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<td>21.93 (14.05)</td>
<td>20.20 (10.55)</td>
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<tr>
<td>Baseline</td>
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<td>16.59 (14.01)</td>
<td>14.12 (5.47)</td>
<td>13.29 (7.17)</td>
</tr>
<tr>
<td>Task</td>
<td>14.82 (12.69)</td>
<td>14.68 (8.20)</td>
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<td>RMSSD</td>
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<tr>
<td>Recovery</td>
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<tr>
<td>Log LF</td>
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<tr>
<td>Baseline</td>
<td>5.31 (1.65)</td>
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<td>Task</td>
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<td>5.49 (1.24)</td>
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<td>5.47 (1.24)</td>
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<tr>
<td>Recovery</td>
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<td>5.80 (1.20)</td>
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<td>Task</td>
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<td>Recovery</td>
<td>5.39 (1.40)</td>
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<td>Baseline</td>
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<td>3.92 (1.18)</td>
<td>3.90 (1.57)</td>
<td>3.48 (1.46)</td>
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<td>Recovery</td>
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<td>4.46 (1.41)</td>
<td>3.72 (1.25)</td>
<td>4.43 (1.48)</td>
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continued
Table 8 continued

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<th>Baseline</th>
<th>Task</th>
<th>Recovery</th>
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<tr>
<td>Log HF</td>
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<td>3.63 (1.15)</td>
<td>3.68 (1.50)</td>
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<td>4.20 (1.48)</td>
<td>4.08 (1.40)</td>
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<td>3.73 (1.37)</td>
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<tr>
<td>LF/HF</td>
<td>4.41 (3.99)</td>
<td>3.97 (3.59)</td>
<td>4.87 (4.36)</td>
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<td>6.53 (5.16)</td>
<td>6.13 (4.71)</td>
<td>7.99 (6.37)</td>
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<td>2.42 (2.13)</td>
<td>3.44 (2.82)</td>
<td>5.08 (4.27)</td>
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<td>47.40 (7.37)</td>
<td>28.40 (6.40)</td>
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<tr>
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<td>42.00 (11.23)</td>
<td>51.67 (12.52)</td>
<td>38.33 (12.84)</td>
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<tr>
<td>STAI X1</td>
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<td>39.17 (13.07)</td>
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<td></td>
<td>46.83 (12.68)</td>
<td></td>
<td>44.60 (14.93)</td>
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</tbody>
</table>

**Abbreviations**: Mean RR= mean interbeat intervals; Mean HR= mean heart rate; STDRR= standard deviation of normal – normal beats; RMSSD= square root of the mean squared differences of successive normal – normal intervals; Log LF= natural ln transformed low frequency value; Log HF= natural ln transformed high frequency value; LF/HF= low frequency / high frequency ratio; STAI-X1= State-Trait Anxiety Inventory, State version

**Table 8: HRV measures and state anxiety of Participants**
<table>
<thead>
<tr>
<th></th>
<th>COPD-ANX (n=15) M (SD)</th>
<th>HEA-ANX (n = 15) M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Health Behaviors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pack Year</td>
<td>21.63 (22.36)</td>
<td>9.62 (15.67)</td>
</tr>
<tr>
<td>V0₂ max</td>
<td>23.60 (9.66)</td>
<td>24.39 (11.18)</td>
</tr>
<tr>
<td>PSQI</td>
<td>12.0 (4.06)</td>
<td>7.8 (4.02) **</td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>5 (33.3%)</td>
<td>4 (26.7%)</td>
</tr>
<tr>
<td>1&lt;</td>
<td>2 (13.3%)</td>
<td>4 (26.7%)</td>
</tr>
<tr>
<td>1 - 5</td>
<td>5 (33.3%)</td>
<td>6 (40%)</td>
</tr>
<tr>
<td>6-10</td>
<td>2 (13.3%)</td>
<td>1 (6.7%)</td>
</tr>
<tr>
<td>11-15</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>16-20</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>20&gt;</td>
<td>1 (6.7%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td><strong>Distress</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSS-10</td>
<td>22.4 (5.84)</td>
<td>21.67 (3.77)</td>
</tr>
<tr>
<td>PANAS-POS</td>
<td>27.47 (3.60)</td>
<td>25.07 (4.37)</td>
</tr>
<tr>
<td>PANAS-NEG</td>
<td>25.27 (3.90)</td>
<td>28.0 (4.12)</td>
</tr>
<tr>
<td>STAI-X2</td>
<td>48.62 (9.61)</td>
<td>49.73 (8.70)</td>
</tr>
<tr>
<td>CESD</td>
<td>25.73 (11.11)</td>
<td>23.43 (11.56)</td>
</tr>
<tr>
<td><strong>Dispositional</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSWQ</td>
<td>51.13 (6.72)</td>
<td>51.54 (12.82)</td>
</tr>
<tr>
<td>RRS-R</td>
<td>11.07 (3.86)</td>
<td>10.73 (3.56)</td>
</tr>
<tr>
<td>RRS-B</td>
<td>12.4 (3.96)</td>
<td>10.73 (3.08)</td>
</tr>
<tr>
<td>MCSD</td>
<td>6.73 (1.94)</td>
<td>6.60 (3.16)</td>
</tr>
<tr>
<td>ACS</td>
<td>50.20 (7.59)</td>
<td>49.67 (9.89)</td>
</tr>
</tbody>
</table>

* p<0.05. **p<0.01

Table 9: Health Behaviors, Measures of Distress, and Dispositional variables in the COPD-ANX and HEA-ANX group
<table>
<thead>
<tr>
<th>COPD (n=30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>COPD                               COPD-ANX</td>
</tr>
<tr>
<td>M (SD)                             M (SD)</td>
</tr>
<tr>
<td>FEV(_1)                           1.31 (0.69)    1.59 (0.63)</td>
</tr>
<tr>
<td>FEV(_1)%                         52.4 (26.29)    55.67 (17.78)</td>
</tr>
<tr>
<td>FVC                                2.06 (1.05)     2.52 (0.78)</td>
</tr>
<tr>
<td>FVC%                               62.2 (22.83)    68.6 (18.71)</td>
</tr>
<tr>
<td>FEV(_1)/FVC                      0.64 (0.14)     0.63 (0.17)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Health Behaviors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pack Year                         44.63 (28.66)    21.63 (22.36) **</td>
</tr>
<tr>
<td>V(_0)max                         17.81 (11.72)    23.60 (9.66)</td>
</tr>
<tr>
<td>PSQI                               6.93 (3.47)      12.0 (4.06) **</td>
</tr>
<tr>
<td>Alcohol</td>
</tr>
<tr>
<td>0                                 3 (20%)         5 (33.3%)</td>
</tr>
<tr>
<td>1&lt;                                4 (26.7)        2 (13.3%)</td>
</tr>
<tr>
<td>1-5                               6 (40%)         5 (33.3%)</td>
</tr>
<tr>
<td>6-10                              1 (20%)         2 (13.3%)</td>
</tr>
<tr>
<td>11-15                             0 (0%)          0 (0%)</td>
</tr>
<tr>
<td>16-20                             0 (0%)          0 (0%)</td>
</tr>
<tr>
<td>20+                               1 (6.7%)        1 (6.7%)</td>
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</table>

<table>
<thead>
<tr>
<th>Distress</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSS-10                            14.26 (7.12)    22.4 (5.84) **</td>
</tr>
<tr>
<td>PANAS-POS                          29.33 (3.74)    27.47 (3.60)</td>
</tr>
<tr>
<td>PANAS-NEG                          26.53 (5.03)    25.27 (3.90)</td>
</tr>
<tr>
<td>STAI-X2                           34.87 (8.76)    48.62 (9.61) **</td>
</tr>
<tr>
<td>CESD                              11.20 (7.62)    25.73 (11.11) ***</td>
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</table>

<table>
<thead>
<tr>
<th>Dispositional</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSWQ                              39.06 (13.48)   51.13 (6.72) **</td>
</tr>
<tr>
<td>RRS-R                             8.27 (1.98)     11.07 (3.86) *</td>
</tr>
<tr>
<td>RRS-B                             8.47 (1.76)     12.4 (3.96) **</td>
</tr>
<tr>
<td>MCSD                              7.53 (3.31)     6.73 (1.94)</td>
</tr>
<tr>
<td>ACS                               56.73 (9.48)    50.20 (7.59)</td>
</tr>
</tbody>
</table>

* p<0.05, **p<0.01, ***p<0.001

Table 10: Pulmonary functioning, Health Behaviors, Measures of Distress, and Dispositional variables in the COPD and COPD-ANX group
<table>
<thead>
<tr>
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<th>COPD-ANX (n=15)</th>
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<tr>
<td></td>
<td>Anxiety after diagnosis (n=9)</td>
<td>Pre-existing anxiety (n=9)</td>
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<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
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<tr>
<td><strong>FEV₁</strong></td>
<td>1.61 (0.54)</td>
<td>1.58 (0.73)</td>
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<tr>
<td><strong>FEV₁%</strong></td>
<td>60.0 (18.87)</td>
<td>52.78 (17.53)</td>
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<tr>
<td><strong>FEV₁/FVC</strong></td>
<td>86.50 (11.15)</td>
<td>79.0 (26.53)</td>
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<tr>
<td><strong>Health Behaviors</strong></td>
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</tr>
<tr>
<td><strong>Pack Year</strong></td>
<td>16.0 (12.96)</td>
<td>25.39 (27.01)</td>
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<tr>
<td><strong>V0₂max</strong></td>
<td>22.65 (6.29)</td>
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<td>10.5 (3.42)</td>
<td>12.67 (4.33)</td>
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<tr>
<td><strong>Alcohol</strong></td>
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<td>3 (33.3%)</td>
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<td>1 (11.1%)</td>
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<td>1 (16.7%)</td>
<td>1 (11.1%)</td>
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<td>16-20</td>
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<td>0 (0%)</td>
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<td>20&gt;</td>
<td>0 (0%)</td>
<td>1 (11.1%)</td>
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<tr>
<td><strong>Distress</strong></td>
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<tr>
<td><strong>PSS-10</strong></td>
<td>25.33 (6.59)</td>
<td>20.44 (4.67)</td>
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<tr>
<td><strong>PANAS-POS</strong></td>
<td>27.50 (4.51)</td>
<td>27.44 (3.17)</td>
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<tr>
<td><strong>PANAS-NEG</strong></td>
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<td>25.22 (4.02)</td>
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<td><strong>STAI-X2</strong></td>
<td>47.67 (11.69)</td>
<td>49.43 (11.69)</td>
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<tr>
<td><strong>CESD</strong></td>
<td>25.17 (11.55)</td>
<td>26.11 (11.49)</td>
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<td><strong>Dispositional</strong></td>
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<tr>
<td><strong>PSWQ</strong></td>
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<td><strong>RRS-B</strong></td>
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<td>12.83 (4.22)</td>
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<td>7.17 (2.23)</td>
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</tr>
<tr>
<td><strong>ACS</strong></td>
<td>51.0 (5.14)</td>
<td>49.67 (9.14)</td>
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</tr>
</tbody>
</table>

Table 11. Pulmonary functioning, health behaviors, measures of distress, and dispositional variables within COPD-ANX individuals divided by pre-existing anxiety and anxiety developed after diagnosis
Figure 1. Study Design

Consent

Screening

- STAI-X1
- Blood Pressure screening

Pulmonary Function Testing

- STAI-X1
- Measures of Distress
- Dispositional Variables

Fitted with HRV equipment

Baseline Questionnaires

- Health Behaviors

Quiet Sitting Period (5 minutes)

Baseline Speech (Neutral Script: 1 minute)

Task (Trier Social Stress Task)

- STAI-X1
- 20 minutes of relaxing music

Recovery

Baseline Phase

Task Phase

Recovery Phase
Figure 2. Log LF for Baseline, Task, and Recovery plotted by disease state
Figure 3. Log HF for Baseline, Task, and Recovery plotted by disease state
Figure 4. Log HF for Baseline, Task, and Recovery plotted for the COPD-ANX group divided by individuals with / without preexisting anxiety


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APPENDIX A
APPENDIX A. The Ohio State University Consent to Participate in Research

Study Title: Stress, Anxiety, and Heart Rate Variability in Patients with Chronic Obstructive Pulmonary Disease
Principal Investigator: Charles F. Emery, Ph.D.
Sponsor: Alumni Grants for Graduate Research and Studies (AGGRS)

- This is a consent form for research participation. It contains important information about this study and what to expect if you decide to participate. Please consider the information carefully. Feel free to discuss the study with your friends and family and to ask questions before making your decision whether or not to participate.

- Your participation is voluntary. You may refuse to participate in this study. If you decide to take part in the study, you may leave the study at any time. No matter what decision you make, there will be no penalty to you and you will not lose any of your usual benefits. Your decision will not affect your future relationship with The Ohio State University. If you are a student or employee at Ohio State, your decision will not affect your grades or employment status.

- You may or may not benefit as a result of participating in this study. Also, as explained below, your participation may result in unintended or harmful effects for you that may be minor or may be serious depending on the nature of the research.

- You will be provided with any new information that develops during the study that may affect your decision whether or not to continue to participate. If you decide to participate, you will be asked to sign this form and will receive a copy of the form. You are being asked to consider participating in this study for the reasons explained below.

1. Why is this study being done?

Chronic Obstructive Pulmonary Disease (COPD) is a lung disease in which the lungs are damaged, making it difficult to breathe. COPD is a term that refers to
individuals who have emphysema and / or chronic bronchitis. Research shows that approximately 40% of individuals who have Chronic Obstructive Pulmonary Disease (COPD) experience elevated levels of anxiety. The study is designed to provide information on anxiety symptoms in COPD patients.

2. How many people will take part in this study?

45 COPD patients and 45 healthy adults will be recruited for participation in this study.

3. What will happen if I take part in this study?

Prior to participating in the study, you will be asked to refrain from smoking and consuming caffeine and alcohol for 24 hours. If you are using a Beta-2 agonist inhaler (e.g., Albuterol, Nebulizer, Xopenex, Maxair, Serevent), you will be asked to refrain from using it six hours prior to participating in the study. You will also be asked to refrain from using anti-anxiety medication (e.g., Xanax, Valium, Paxil, etc.). You will also be asked to bring in your list of medications with you on your day of the study.

You will be asked to participate in a public-speaking task. You will be asked to complete pulmonary functioning tests and questionnaires regarding depression, anxiety, stress, personality factors, sleep, exercise, alcohol consumption and smoking history. You will then participate in a speech task, and complete a questionnaire on anxiety after the task. Throughout this study, your heart rate and the level of oxygen in your blood will be monitored.

This study is divided into three phases – baseline, task, and recovery. You will be required to complete two packets of questionnaires during the course of the study – at baseline and at recovery.

The baseline phase will take approximately 25 – 30 minutes. Upon consenting to the study, you will be asked to complete a questionnaire measuring your anxiety level to determine eligibility of the study. If it is determined that you are not eligible, you will be paid $10 and will not proceed with the study. If you are a premenopausal woman, you will be asked to take a pregnancy test provided by the research staff. Pregnant women will be excluded from the study. All participants will undergo blood pressure measurement, administered by a trained technician. Participants who meet the criteria for hypertension (140 / 90) will be determined to be ineligible to participate in the study and will not proceed with the study. Following this procedure, height and weight will be measured. You will then complete pulmonary function tests, which will involve taking deep breaths and blowing all of your air out into a machine that measures your lung capacity and air flow. Following this measurement, you will be fitted with a wrist watch device and a chest band to measure heart rate, and a finger clip device to measure the level of oxygen in your blood periodically through the three phases of the study. Following this procedure, you will complete a baseline self-report questionnaire packet that will ask you questions about your level of depression, anxiety, stress, personality factors, and lifestyle (e.g. alcohol consumption, sleep, exercise, smoking history). You will then sit quietly for five minutes, after which you will be asked to read a paragraph out loud.
The task phase will take approximately 15 minutes. During this phase, you will be asked to prepare and give a brief speech on an assigned topic. Following the task, you will complete a self-report questionnaire of anxiety.

During the recovery phase, you will listen to relaxing music for 20 minutes.

4. How long will I be in the study?

This procedure will require approximately 1.5 hours of your time.

5. Can I stop being in the study?

You may leave the study at any time. If you decide to stop participating in the study, there will be no penalty to you, and you will not lose any benefits to which you are otherwise entitled. Your decision will not affect your future relationship with The Ohio State University.

6. What risks, side effects or discomforts can I expect from being in the study?

Overall, the study has few serious risks.

You may experience minor physical discomfort if you participate in this study. Pulmonary function testing involves breathing forcefully into a machine, and occasionally people develop light-headedness during this procedure. You will also be asked to refrain from using your beta-blocker inhaler six hours prior to the study, and as a result you may experience shortness of breath.

You may also experience heightened anxiety from refraining from consuming caffeine, not smoking, and not taking anti-anxiety medication.

The questionnaires that you will be completing may elicit feelings of distress or discomfort. There is minimal risk of injury during the speech task, and the recording of heart rate variability involves no greater risk than that associated with normal everyday activity. There may be a risk to your reputation as you will be questioned about your history of alcohol consumption and tobacco use.

7. What benefits can I expect from being in the study?

You will be contributing to society’s knowledge of anxiety symptoms in COPD patients. The knowledge gained concerning the relationship of heart rate variability, anxiety, and COPD will help identify factors that may improve quality of life among patients. You will not be receiving a direct benefit as a result of participation in this study.

8. What other choices do I have if I do not take part in the study?

You may choose not to participate without penalty or loss of benefits to which you are otherwise entitled.
9. Will my study-related information be kept confidential?

Efforts will be made to keep your study-related information confidential. However, there may be circumstances where this information must be released. For example, personal information regarding your participation in this study may be disclosed if required by state law. Also, your records may be reviewed by the following groups (as applicable to the research):

- Office for Human Research Protections or other federal, state, or international regulatory agencies;
- U.S. Food and Drug Administration;
- The Ohio State University Institutional Review Board or Office of Responsible Research Practices;
- The sponsor supporting the study, their agents or study monitors; and
- Your insurance company (if charges are billed to insurance).

If the study involves the use of your protected health information, you may also be asked to sign a separate Health Insurance Portability and Accountability Act (HIPAA) research authorization form.

All the data will be coded by subject number, and all identifying information will be removed immediately after receiving the completed questionnaires. All data will be stored in a locked filing cabinet under the supervision of the principal investigator.

10. What are the costs of taking part in this study?

There are no costs related to participation in this study.

11. Will I be paid for taking part in this study?

A payment of $50 will be made upon completion of the study. If it is determined that you are ineligible to participate in the study after consenting to participate, you will be paid $10.

By law, payments to subjects are considered taxable income. If you are an OSU employee, any compensation you receive as a result of participating in the study will be made through the payroll system and applicable taxes will be deducted.

12. What happens if I am injured because I took part in this study?

If you suffer an injury from participating in this study, you should notify the researcher or study doctor immediately, who will determine if you should obtain medical treatment at The Ohio State University Medical Center.

The cost for this treatment will be billed to you or your medical or hospital insurance. The Ohio State University has no funds set aside for the payment of health care expenses for this study.
13. What are my rights if I take part in this study?

If you choose to participate in the study, you may discontinue participation at any
time without penalty or loss of benefits. By signing this form, you do not give up any
personal legal rights you may have as a participant in this study.

You will be provided with any new information that develops during the course of the
research that may affect your decision whether or not to continue participation in the
study.

You may refuse to participate in this study without penalty or loss of benefits to which
you are otherwise entitled.

An Institutional Review Board responsible for human subjects research at The Ohio
State University reviewed this research project and found it to be acceptable,
according to applicable state and federal regulations and University policies
designed to protect the rights and welfare of participants in research.

14. Who can answer my questions about the study?

For questions, concerns, or complaints about the study you may contact the
Principal Investigator, Charles F. Emery, Ph.D. at 145 Psychology Building,
1835 Neil Avenue, Ohio State University, Columbus OH 43210. . 
For questions about your rights as a participant in this study or to discuss other
study-related concerns or complaints with someone who is not part of the research
team, you may contact Ms. Sandra Meadows in the Office of Responsible Research
Practices at 1-800-678-6251.

If you are injured as a result of participating in this study or for questions about a
study-related injury, you may contact Dr. Charles F. Emery or Alyssa Suh at 614-
292-6527.

Signing the consent form

I have read (or someone has read to me) this form and I am aware that I am being
asked to participate in a research study. I have had the opportunity to ask questions and
have had them answered to my satisfaction. I voluntarily agree to participate in this
study.
I am not giving up any legal rights by signing this form. I will be given a copy of this form.

Printed name of subject

Signature of subject

Date and time

Printed name of person authorized to consent for subject (when applicable)

Signature of person authorized to consent for subject (when applicable)

Date and time

Relationship to the subject

Date and time

**Investigator/Research Staff**

I have explained the research to the participant or his/her representative before requesting the signature(s) above. There are no blanks in this document. A copy of this form has been given to the participant or his/her representative.

Printed name of person obtaining consent

Signature of person obtaining consent

Date and time

*Witness(es)* - *May be left blank if not required by the IRB*

Printed name of witness

Signature of witness

Date and time

Printed name of witness

Signature of witness

Date and time
APPENDIX B
Beginning April 14, 2003, the new HIPAA Privacy Rule requires that Ohio State University Principal Investigators (PIs) provide research subjects with greater detail than what is currently included in the IRB-approved consent form concerning how a subject’s past, present and future health-related information (collectively, Protected Health Information or PHI) will be used, shared and protected during the research. Specifically, the Privacy Rule now requires that PIs inform subjects of the following: 1) what specific kinds of information will be used or disclosed to others during the course of the research; 2) the specific identities of collaborating investigators, sponsor companies or sponsor agencies that will potentially receive copies of subjects’ PHI during the research; 3) that subjects have a right to review their research-related PHI; and 4) that subjects have the express right to revoke their authorizations for the release of PHI at any time.

To meet these new requirements, PIs using PHI obtained from medical or research records from the Ohio State University Hospitals, The Arthur G. James Cancer Hospital and Richard J. Solove Research Institute, OSU & Harding Behavioral Health Care & Medicine, the Ohio State University Hospitals East and the Primary Care Network (the University Health System), or other University operated health centers or clinics, must now complete and receive a signed copy of the University’s “Authorization to Use Personal Health Information in Research” form (the Authorization) below from subjects enrolling in research studies on or after April 14th (or be granted a waiver by a HIPAA Privacy Board) in addition to obtaining a signed IRB-approved consent form. The form will need to be carefully prepared by PIs to ensure that the Authorization covers ALL of the necessary uses and disclosures of personal health information used in clinical research. Failure to do so may violate the Privacy Rule and result in penalties against the University as well as individual civil and criminal penalties against the Principal Investigator.

INSTRUCTIONS TO RESEARCHERS
FOR PREPARING THE RESEARCH AUTHORIZATION FORM

1. Complete the first section of the Authorization form with title of the study, the OSU IRB protocol number, and PI name. Add subject name at the time of authorization. Do not include these instructions as part of the completed Authorization form.

2. “Uses and Disclosures Covered by this Authorization” – List every known non-OSU person, class of persons, or organizations (including the sponsor agency or company, known subsidiaries of the sponsor, cooperative data groups, etc.) that may create, disclose, receive, and/or use the information in connection with the study. Fill in the blanks on the form (and delete the instructions in italics as well as inapplicable
3. bulleted sections) as appropriate. If information will not be disclosed outside of The Ohio State University, delete all bullets and insert “None”. Note: if a person(s) or organization is not listed on the form, they may not create, disclose, receive or use PHI in connection with the study.

3a. “HIPAA Privacy Contact” – If the research involves the use of medical records from the University Health System, where applicable, insert the contact and address: HIPAA Privacy Manager, the Ohio State University Medical Center, 140 Doan Hall, 410 W. Tenth Avenue, Columbus, Ohio 43210.

3b. If the research solely involves the use of personal health records at non-University Health System clinics or health care facilities (for example, the Dental School, Optometry School, Nisonger Center, Younkin Center, Psychological Services Center, Anxiety and Stress Disorder Clinic, Marriage & Family Therapy Clinic, Camera Center or faculty practice group such as OSU-P) insert the name and address of the appropriate Privacy Contact for the center, school, clinic or practice group. If unknown, contact the director of the health center, school, clinic or practice group or the Office of Legal Affairs at (614) 292-0611 for the contact and address of the applicable Privacy Contact.

4. The Authorization must be presented to all newly enrolled or “re-consented” subjects in IRB-approved research beginning April 14, 2003 at the time the IRB-approved consent form is signed. The subject or his/her legally authorized representative must be provided with a copy of this form after it has been signed. The original, signed copy must be retained in the research file for a period of six years from the date the Authorization was signed (or longer, according to sponsor requirements). Prior IRB approval of the Authorization is not required; however, the Privacy Contact and/or HIPAA Privacy Board may conduct audits of the Authorization to ensure completeness.

5a. “Notice of Privacy Practices” – Each subject who receives health care services at the University on or after April 14, 2003 should receive a copy of a Notice of Privacy Practices (NPP) and sign an acknowledgement (NPP Acknowledgement form) that (s)he obtained the NPP.

5b. If the research involves the use of health and/or medical records from the University Health System and the subject has not received a copy of the University Health System’s NPP, provide the subject with a copy of the NPP. The subject should sign a copy of the University Health System’s NPP Acknowledgement form. The original, signed copy of the NPP Acknowledgement form must be retained in the research file for a period of six years from the date the NPP Acknowledgement was signed (or longer, according to sponsor requirements). The University Health System’s NPP and NPP Acknowledgement form are available in electronic format on the Office of Responsible Research Practices (ORRP) website at http://www.orrp.ohio-state.edu/ as well as the Medical Center’s website at http://www.osumedcenter.edu.

5c. If the research involves the use of health records at other non-University Health System clinics or facilities (including the sites listed above in item 3b.) and the subject has not received a copy of the facility or clinic’s individual NPP, provide the subject with a copy of the NPP. Contact the director of the applicable health center,
school, clinic or practice group to obtain a copy of the NPP and the NPP Acknowledgement form. The original, signed copy of the NPP Acknowledgement form must be retained in the research file for a period of six years from the date the NPP Acknowledgement was signed (or longer, according to sponsor requirements).

**AUTHORIZATION TO USE**

**PERSONAL HEALTH INFORMATION IN RESEARCH**

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**Title of the Study:** Stress, Anxiety, and Heart Rate Variability in Patients with Chronic Obstructive Pulmonary Disease

**OSU Protocol Number:** 2007H0304

**Principal Investigator:** Charles F. Emery, Ph.D.

**Subject Name**

Before researchers use or share any health information about you as part of this study, The Ohio State University is required to obtain your authorization. This helps explain to you how this information will be used or shared with others involved in the study.

- The Ohio State University and its hospitals, clinics, health-care providers and researchers are required to protect the privacy of your health information.

- You should have received a Notice of Privacy Practices when you received health care services here. If not, let us know and a copy will be given to you. Please carefully review this information. Ask if you have any questions or do not understand any parts of this notice.

- If you agree to take part in this study your health information will be used and shared with others involved in this study. Also, any new health information about you that comes from tests or other parts of this study will be shared with those involved in this study.

- Health information about you that will be used or shared with others involved in this study may include your research record and any health care records at the Ohio State University. For example, this may include your medical records, x-ray or laboratory results. Psychotherapy notes in your health records (if any) will not, however, be shared or used. Use of these notes requires a separate, signed authorization.

- Please read the information carefully before signing this form. Please ask if you have any questions about this authorization, the University’s Notice of Privacy Practices or the study before signing this form.

**Initials/Date:** ________________
Those Who May Use, Share And Receive Your Information As Part Of This Study

- Researchers and staff at The Ohio State University will use, share and receive your personal health information for this research study. Other Ohio State University staff not involved in the study but who may become involved in your care for study-related treatment will have access to your information.

- Those who oversee the study will have access to your information, including:
  - Members and staff of the Ohio State University’s Institutional Review Boards, including the Western Institutional Review Board
  - The Office for Responsible Research Practices
  - University data safety monitoring committees
  - The Ohio State University Research Foundation

- Your health information may also be shared with federal and state agencies that have oversight of the study or to whom access is required under the law. These may include:
  - The Food and Drug Administration
  - The Office for Human Research Protections
  - The National Institutes of Health
  - The Ohio Department of Human Services

The information that is shared with those listed above may no longer be protected by federal privacy rules.

Initials/Date________

Authorization Period

This authorization will not expire unless you change your mind and revoke it in writing. There is no set date at which your information will be destroyed or no longer used. This is because the information used and created during the study may be analyzed for many years, and it is not possible to know when this will be complete.

Signing the Authorization

- You have the right to refuse to sign this authorization. Your health care outside of the study, payment for your health care, and your health care benefits will not be affected if you choose not to sign this form.

- You will not be able to take part in this study and will not receive any study treatments if you do not sign this form.

- If you sign this authorization, you may change your mind at any time. Researchers may continue to use information collected up until the time that you formally changed your mind. If you change your mind, your authorization must be revoked in writing.
To revoke your authorization, please write to:

- Charles F. Emery, Ph.D., 145 Psychology Building, 1835 Neil Avenue, Ohio State University, Columbus, OH 43210, 292-3016 or Steven Beck, Ph.D., 1835 Neil Avenue Mall, Columbus, OH 43201, 292-6849.

- Signing this authorization also means that you will not be able to see or copy your study-related information until the study is completed. This includes any portion of your medical records that describes study treatment.

Contacts for Questions

- If you have any questions relating to your privacy rights, please contact Steven Beck, Ph.D., 1835 Neil Avenue Mall, Columbus, OH 43201, 292-6849.

- If you have any questions relating to the research, please contact Charles F. Emery, Ph.D., 145 Psychology Building, 1835 Neil Avenue, Ohio State University, Columbus, OH 43210, 292-3016.

Signature

I have read (or someone has read to me) this form and have been able to ask questions. All of my questions about this form have been answered to my satisfaction. By signing below, I permit Charles F. Emery, Ph.D. and the others listed on this form to use and share my personal health information for this study. I will be given a copy of this signed form.

Signature________________________________________________________
(Subject or Legally Authorized Representative)

Name ________________________________
(Print name above)
(If legal representative, also print relationship to subject.)

Date___________ Time __________ AM / PM
APPENDIX C
APPENDIX C. Experimenter Reading Instructions for Reading Task

Next, we need to get a measure of your heart rate while you read out-loud. Please take 60 seconds to read this passage in your normal speaking voice. If you finish before 60 seconds is complete, please start from the beginning and read again.

1. Collect all your dirty clothes from the bathroom floor, or wherever you keep them. Keep in mind that your socks may be in the living room or under your desk. Make sure you find everything.

2. Sort the clothes into piles. There are two important considerations here: what material your clothes are, and what color.

3. Read all the labels, and pick out the clothes that (a) cannot be washed (need to be taken to a dry cleaner), (b) all clothes that say delicate, or have other restrictions. These are commonly materials such as silk, wool, and certain synthetic fibers.

4. Remember delicates may have to be washed by hand. This means using a sink or a bucket, and adding water (read labels to verify the temperature) and detergent. The water should feel slippery. Warning: remember to sort the colors (see point five below).

5. Sort the remaining clothes (generally materials such as cotton, linen, synthetic fibers) according to color. All whites should go with only whites (or very light colors if the item has been washed many times). Then put all the reds, pinks, and oranges in a separate pile (never keep this close to the white pile as you will be wearing pink shirts for a long time). Then, depending on how much more you have left, put the other colors together, possibly into a lighter (e.g. greys, yellows, light blues) and a darker pile (blacks, dark blues, browns, greens, purples).

6. Know that, each pile is its own load. It is recommended to start with the highest priority pile, usually the one with socks and underwear.

7. Put the first pile into the washer. Add detergent (the bottle/box should say how much you need).

8. Read all instructions on the washer carefully, and examine all the knobs to make sure you are washing the clothes the way you are planning to. Whites and underwear usually go on hot temperatures. Colored clothes and sheets usually go on warm or cold temperatures.

9. Close the door and push the on button.

10. Come back when the washer is done and take the clothes out. Shake them gently, and either put into a dryer (remember to read the label) or hang them up to dry.
APPENDIX D
APPENDIX D. VIDEO SCRIPT

You will now have to deliver a speech for a job application, for which you will have two minutes to prepare. This speech will be filmed, and someone who is trained in behavioral observation will be viewing the tape and his / her behavior will be accordingly documented. As for the speech, you should imagine that you have applied for a position and you have been invited by that institution to introduce yourself. The speech should take five minutes. Please take 2 minutes now to construct your speech.
APPENDIX E
APPENDIX E. Questionnaires
Demographic Information

Age: ____________________

Please circle one numbered response for each of the questions below.

1. What is your gender?  a. Male           b. Female

2. With what ethnic group do you primarily identify?
   a. White           b. Black           c. Hispanic
   d. Asian           e. American Indian
   f. Other (Please list) ________________________

3. What is your marital status?

4. What are your living arrangements?
   a. Living alone  b. Living with spouse or “significant other”
   c. Living with children  d. Living with children and spouse or “significant other”
   e. Living with parents  f. Living with friend(s)

5. What are your total annual gross wages or income (pre-tax)? $ _________________

6. How many years of formal education do you have? _______________ years

7. What is your current height? ___________ inches

8. What is your current weight? ___________ pounds

<for COPD patients only>

9. What is your current pulmonary diagnosis? ____________________

10. When were you diagnosed? ___________ / ___________
    (YYYY) / (MM)
HEALTH BEHAVIOR QUESTIONNAIRE

1. Do you smoke cigarettes now?
   1… Yes (Continue)
   2… NO (Skip to #6)

2. On the average, how many cigarettes do you smoke a day?
   1… Less than a pack a week
   2… ½ a pack a day or less
   3… 1 pack a day
   4… 1 ½ pack a day
   5… 2 packs a day
   6… 2 ½ packs a day
   7… 3 or more packs a day

3. How many years have you smoked this amount? ___________ years

4. How strongly do you desire to quit smoking now? (Circle one number)
   1 ----- 2 ----- 3 ----- 4 ----- 5 ----- 6 ----- 7
   Not at all                                    Very, very much

5. How likely is it that you will actually quit if you make a serious attempt? (circle one number)
   1 ----- 2 ----- 3 ----- 4 ----- 5 ----- 6 ----- 7
   Not at all                                    Very, very much

6. Have you ever used cigarettes on a regular basis?
   1… Yes (continue)
   2… No (skip to #10)

7. Prior to quitting, on the average, how many cigarettes did you smoke a day?
   1… Less than a pack a week
   2… ½ a pack a day or less
   3… 1 pack a day
   4… 1 ½ pack a day
   5… 2 packs a day
   6… 2 ½ packs a day
   7… 3 or more packs a day

8. How many years did you smoke? ___________ years

9. How long has it been since you quit smoking? ___________ years
10. Do you consume alcohol?
   1... YES
   2... NO (Skip to # 13)

11. In the past month, about how many alcoholic drinks did you consume per week? (A drink is 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of hard liquor)
   1... Less than 1 drink per week
   2... 1 to 5 drinks per week
   3... 6 to 10 drinks per week
   4... 11 to 15 drinks per week
   5... 16 to 20 drinks per week
   6... 20 to more drinks per week

12. How long have you consumed this amount of alcohol? ________________ (Answer and skip to #17)

13. Have you ever consumed alcohol?
   1... YES
   2... NO (Skip to #17)

14. Prior to quitting, on the average, how many alcoholic drinks did you consume per week? (A drink is 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of hard liquor)
   1... Less than 1 drink per week
   2... 1 to 5 drinks per week
   3... 6 to 10 drinks per week
   4... 11 to 15 drinks per week
   5... 16 to 20 drinks per week
   6... 20 to more drinks per week

15. How long did you consume this amount of alcohol? ______________

16. How long has it been since you quit consuming? ______________
PSS-10
Thoughts and Feelings During the Last Week

The questions in this scale ask you about your feelings and thoughts during the past month. In each case, you will be asked to indicate how often you felt or thought in a certain way. The best approach is to answer each question fairly quickly. That is, don’t try to count up the number of times you felt a particular way, but rather indicate the answer that seems like a reasonable estimate. Circle the appropriate number.

Almost Never Some Never Often Very

1. In the last month, how often have you been upset because of something that happened unexpectedly?
   0 1 2 3 4

2. In the last month, how often have you felt that you were unable to control the important things in your life?
   0 1 2 3 4

3. In the last month, how often have you felt stressed?
   0 1 2 3 4

4. In the last month, how often have you felt confident in your ability to handle your personal problems?
   0 1 2 3 4

5. In the last month, how often have you felt that things were going your way?
   0 1 2 3 4

6. In the last month, how often have you found that you could not cope with all the things that you had to do?
   0 1 2 3 4

7. In the last month, how often have you been able to control irritations in your life?
   0 1 2 3 4

8. In the last month, how often have you felt that you were on top of things?
   0 1 2 3 4

9. In the last month, how often have you been angered because of things that happened that were outside of your control?
   0 1 2 3 4

10. In the last month, how often have you felt difficulties piling up so high that you could not overcome them?
   0 1 2 3 4
### PSQW

Enter the number that best describes how typical or characteristic each item is of you, circling the number next to the item.

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Not at all Typical</td>
<td>Somewhat Typical</td>
<td>Very Typical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. If I don’t have enough time to do everything, I don’t worry about it.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>2. My worries overwhelm me.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>3. I do not tend to worry about things.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>4. Many situations make me worry.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>5. I know I shouldn’t worry about things, but I just cannot help it.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>6. When I am under pressure I worry a lot.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>7. I am always worrying about something.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>8. I find it easy to dismiss worrisome thoughts.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>9. As soon as I finish one task, I start to worry about everything else I have to do.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>10. I never worry about anything.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>11. When there is nothing more I can do about a concern, I don’t worry about it anymore.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>12. I’ve been a worrier all my life.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>13. I notice that I have been worrying about things.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>14. Once I start worrying, I can’t stop.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>15. I worry all the time.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>16. I worry about projects until they are done.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>
RRS

People think and do many different things when they feel sad, blue, or depressed. Below is a list of possibilities. Please indicate if you never, sometimes, often, or always think or do each one when you feel down, sad, or depressed. Please indicate what you generally do, not what you think you should do. Please circle your response.

1. Think "What am I doing to deserve this?"
   - Almost Never
   - Sometimes
   - Often
   - Almost Always

2. Analyze recent events to try to understand why you are depressed.
   - Almost Never
   - Sometimes
   - Often
   - Almost Always

3. Think "Why do I always react this way?"
   - Almost Never
   - Sometimes
   - Often
   - Almost Always

4. Go away by yourself and think about why you feel this way.
   - Almost Never
   - Sometimes
   - Often
   - Almost Always

5. Write down what you are thinking and analyze it.
   - Almost Never
   - Sometimes
   - Often
   - Almost Always

6. Think about a recent situation, wishing it had gone better.
   - Almost Never
   - Sometimes
   - Often
   - Almost Always

7. Think "Why do I have problems other people don't have?"
   - Almost Never
   - Sometimes
   - Often
   - Almost Always

8. Think "Why can't I handle things better?"
   - Almost Never
   - Sometimes
   - Often
   - Almost Always

9. Analyze your personality to try to understand why you are depressed.
   - Almost Never
   - Sometimes
   - Often
   - Almost Always

10. Go someplace alone to think about your feelings.
    - Almost Never
    - Sometimes
    - Often
    - Almost Always
The PANAS – General

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you generally feel this way, that is, how you feel on the average. Use the following scale to record your answers.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>very slightly or not at all</td>
<td>a little</td>
<td>moderately</td>
<td>quite a bit</td>
<td>extremely</td>
</tr>
</tbody>
</table>

___ interested   ___ irritable
___ distressed   ___ alert
___ excited     ___ ashamed
___ upset        ___ inspired
___ strong       ___ nervous
___ guilty       ___ determined
___ scared       ___ attentive
___ hostile      ___ jittery
___ enthusiastic ___ active
___ proud        ___ afraid
**STAI-X1**

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate answer to the right of the statement to indicate how you feel right now, that is, AT THIS MOMENT. There are no right or wrong answers. Do not spend too much time on any one statement, but give the answer which seems to describe your present feelings best.

<table>
<thead>
<tr>
<th></th>
<th>NOT AT ALL</th>
<th>SOMEWHAT</th>
<th>MODERATELY SO</th>
<th>VERY MUCH SO</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I feel calm</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2. I feel secure</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3. I am tense</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4. I am regretful</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. I feel at ease</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. I feel upset</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7. I am presently worrying over possible misfortunes</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8. I feel rested</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9. I feel anxious</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10. I feel comfortable</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11. I feel self-confident</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12. I feel nervous</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13. I am jittery</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14. I feel “high strung”</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15. I am relaxed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16. I feel content</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17. I am worried</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18. I feel over-excited and “rattled”</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19. I feel joyful</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20. I feel pleasant</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
**STAI-X2**

**DIRECTIONS:** A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate answer to the right of the statement to indicate how you GENERALLY feel. There are no right or wrong answers. Do not spend too much time on any one statement, but give the answer which seems to describe your present feelings best.

<table>
<thead>
<tr>
<th></th>
<th>ALMOST NEVER</th>
<th>SOMETIMES</th>
<th>OFTEN</th>
<th>ALMOST ALWAYS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I feel pleasant</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2. I tire quickly</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3. I feel like crying</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4. I wish I could be as happy as others seem to be</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. I am losing out on things because I can't make up my mind soon enough</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. I feel rested</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7. I am “calm, cool, and collected”</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8. I feel that difficulties are piling up high that I cannot overcome them</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9. I worry too much over something that really doesn't matter</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10. I am happy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11. I am inclined to take things hard</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12. I lack self-confidence</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13. I feel secure</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14. I try to avoid facing a crisis or difficulty</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15. I feel blue</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16. I am content</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17. Some unimportant thought runs through my mind and bothers me</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18. I take disappointments so keenly that I can't put them out of my mind</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19. I am a steady person</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20. I get in a state of tension or turmoil as I think over my recent concerns and interests</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
**CES-D**

Circle the number for each statement which best describes how often you felt or behaved this way – **DURING THE PAST WEEK.**

<table>
<thead>
<tr>
<th>DURING THE PAST WEEK:</th>
<th>Rarely or none of the time (less than 1 day)</th>
<th>Some or a little of the time (1-2 days)</th>
<th>Occasionally or a moderate amount of time (3-4 days)</th>
<th>Most of or all of the time (5-7 days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I was bothered by things that usually don’t bother me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. I did not feel like eating; my appetite was poor.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. I felt that I could not shake off the blues even with help from my family or friends.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. I felt that I was just as good as other people.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. I had trouble keeping my mind on what I was doing.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. I felt depressed</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. I felt that everything I did was an effort.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8. I felt hopeful about the future.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9. I thought my life has been a failure.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10. I felt fearful.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11. My sleep was restless.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12. I was happy.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13. I talked less than usual</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14. I felt lonely.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>15. People were unfriendly.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>16. I enjoyed life.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>17. I had crying spells.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>18. I felt sad.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>19. I felt that people disliked me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>20. I could not get “going.”</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
PITTSBURGH SLEEP QUALITY INDEX (PSQI)

Instructions:
The following questions relate to your usual sleep habits during the past month ONLY. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, when have you usually gone to bed at night?
USUAL BED TIME_________________________

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?
NUMBER OF MINUTES_____________________

3. During the past month, when have you usually gotten up in the morning?
USUAL GETTING UP TIME________________

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spend in bed.)
HOURS OF SLEEP PER NIGHT____________

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you........
   (a) cannot get to sleep within 30 minutes
   Not during the Less than Once or Three or more past month________ once a week_______ twice a week_______ times a week______
   (b) Wake up in the middle of the night or early morning
   Not during the Less than Once or Three or more past month________ once a week_______ twice a week_______ times a week______
   (c) Have to get up to use the bathroom.
   Not during the Less than Once or Three or more past month________ once a week_______ twice a week_______ times a week______
   (d) Cannot breathe comfortably.
   Not during the Less than Once or Three or more past month________ once a week_______ twice a week_______ times a week______
   (e) Cough or snore loudly.
   Not during the Less than Once or Three or more past month________ once a week_______ twice a week_______ times a week______
   (f) Feel too cold.
   Not during the Less than Once or Three or more past month________ once a week_______ twice a week_______ times a week______
   (g) Feel too hot.
   Not during the Less than Once or Three or more Past month________ once a week_______ twice a week_______ times a week______
   (h) Had bad dreams.
   Not during the Less than Once or Three or more Past month________ once a week_______ twice a week_______ times a week______
   (i) Have pain.
Not during the Less than Once or Three or more
Past month_______ once a week_______ twice a week_______ times a week_______
(j) Other reason(s), please describe__________________________________________
How often during the past month have you had trouble sleeping because of this?
Not during the Less than Once or Three or more
Past month_______ once a week_______ twice a week_______ times a week_______
6. During the past month, how would you rate your sleep quality overall?
Very good __________________
Fairly good _______________
Fairly bad ________________
Very bad __________________
7. During the past month, how often have you taken medicine (Prescribed or "over
the
counter") to help you sleep?
Not during the Less than Once or Three or more
Past month_______ once a week_______ twice a week_______ times a week_______
8. During the past month, how often have you had trouble staying awake while
driving, eating
meals, or engaging in social activity?
Not during the Less than Once or Three or more
Past month_______ once a week_______ twice a week_______ times a week_______
9. During the past month, how much of a problem has it been for you to keep up
efficiently
enthusiasm to get things done?
No problem at all __________
Only a very slight problem __________
Somewhat of a problem __________
A very big problem __________
Houston Non-Exercise Test

Determine your Physical Activity Rating (PAR)

Directions: Circle the appropriate PAR score (0-7) based on the following scale.

I. Does not participate regularly in programmed recreation, sport, or physical activity.

0 points: Avoids walking or exercise (for example, always uses elevators, drives whenever possible instead of walking).

1 point: Walks for pleasure, routinely uses stairs, occasionally exercises sufficiently to cause heavy breathing or perspiration.

II. Participates regularly in recreation or work requiring modest physical activity (such as golf, horseback riding, calisthenics, gymnastics, table tennis, bowling, weight lifting, or yard work).

2 points: 10 – 60 minutes per week

3 points: Over 1 hour per week

III. Participates regularly in heavy physical exercise (such as running or jogging, swimming, cycling, rowing, skipping rope, running in place) or engages in vigorous aerobic type activity (such as tennis, basketball, or handball)

4 points: Runs less than 1 mile per week or spends less than 30 minutes per week in comparable physical activity

5 points: Runs 1 – 5 miles per week or spends 30 – 60 minutes per week in comparable physical activity

6 points: Runs 5 – 10 miles per week or spends 1 – 3 hours per week in comparable physical activity.

7 points: Runs more than 10 miles per week or spends more than 3 hours per week in comparable physical activity.
MCSD

On this page are a number of statements concerning personal attitudes and traits. Read each item and decide whether the statement is TRUE or FALSE, as it pertains to you personally. Use the scale below and write the number of your response on the blank line.

\[0 = \text{FALSE} \quad 1 = \text{TRUE}\]

1. It is sometimes hard for me to go on with my work if I am not encouraged.  
2. Sometimes I feel resentful if I don’t get my way.  
3. On a few occasions, I have given up doing something because I thought too little of my ability.  
4. There have been times when I felt like rebelling against people in authority even though I knew they were right.  
5. No matter who I’m talking to, I’m always a good listener.  
6. There have been occasions when I took advantage of someone.  
7. I’m always willing to admit it when I made a mistake.  
8. I sometimes try to get even rather than forgive and forget.  
9. I’m always courteous, even to people who are disagreeable.  
10. I have never been irked when people expressed ideas very different from my own.  
11. There have been times when I was quite jealous of the good fortune of others.  
12. I am sometimes irritated by people who ask favors of me.  
13. I have never deliberately said something that hurt someone’s feelings.
Please answer each item, indicating how often it is true for you on the scale below each question.

1. It’s very hard for me to concentrate on a difficult task when there are noises around.

   1  Almost never  2  Sometimes  3  Often  4  Always

2. When I need to concentrate and solve a problem, I have trouble focusing my attention.

   1  Almost never  2  Sometimes  3  Often  4  Always

3. When I am working hard on something, I still get distracted by events around me.

   1  Almost never  2  Sometimes  3  Often  4  Always

4. My concentration is good even if there is music in the room around me.

   1  Almost never  2  Sometimes  3  Often  4  Always

5. When concentrating, I can focus my attention so that I become unaware of what’s going on in the room around me.

   1  Almost never  2  Sometimes  3  Often  4  Always

6. When I am reading or studying, I am easily distracted if there are people talking in the same room.

   1  Almost never  2  Sometimes  3  Often  4  Always
7. When trying to focus my attention on something, I have difficulty blocking out distracting thoughts.

1 2 3 4
Almost never Sometimes Often Always

8. I have a hard time concentrating when I’m excited about something.

1 2 3 4
Almost never Sometimes Often Always

9. When concentrating I ignore feelings of hunger or thirst.

1 2 3 4
Almost never Sometimes Often Always

10. I can quickly switch from one task to another.

1 2 3 4
Almost never Sometimes Often Always

11. It takes me a while to get really involved in a new task.

1 2 3 4
Almost never Sometimes Often Always

12. It is difficult for me to coordinate my attention between the listening and writing required when taking notes during lectures.

1 2 3 4
Almost never Sometimes Often Always

13. I can become interested in a new topic very quickly when I need to.

1 2 3 4
Almost never Sometimes Often Always

14. It is easy for me to read or write while I’m also talking on the phone.

1 2 3 4
Almost never Sometimes Often Always

15. I have trouble carrying on two conversations at once.

1 2 3 4
Almost never Sometimes Often Always
16. I have a hard time coming up with new ideas quickly.

   1           2           3           4
Almost never Sometimes Often Always

17. After being interrupted or distracted, I can easily shift my attention back to what I was doing before.

   1           2           3           4
Almost never Sometimes Often Always

18. When a distracting thought comes to mind, it is easy for me to shift my attention away from it.

   1           2           3           4
Almost never Sometimes Often Always

19. It is easy for me to alternate between two different tasks.

   1           2           3           4
Almost never Sometimes Often Always

20. It is hard for me to break from one way of thinking about something and look at it from another point of view.

   1           2           3           4
Almost never Sometimes Often Always