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PHYSIOLOGICAL AND COGNITIVE FACTORS IN ASTHMA AND  
PANIC DISORDER: APPLICATION OF THE COGNITIVE  
AND DYSPNEA/SUFFOCATION FEAR THEORIES

By

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B. A., Oregon State University, 1995

A THESIS

Submitted in Partial Fulfillment of the  
Requirements for the Degree of  
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(in Psychology)

The Graduate School

The University of Maine

December, 2001

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Thesis Advisor: Dr. Sandra T. Sigmon

An Abstract of the Thesis Presented  
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Recently, the relationship of anxiety with asthma has been investigated in the psychological literature, revealing that individuals with asthma are more likely to develop panic disorder than are individuals without asthma (e.g., Carr, 1998). Two theories (dyspnea/suffocation fear theory and cognitive theory) have been used to explain how asthma symptoms are affected by panic attacks or panic disorder. The present study examines both theories, as well as a hypothesis developed by Carr (1998) that suggests that the presence of panic disorder in individuals with asthma may lead to better lung functioning in response to stressful stimuli than for individuals with asthma alone.

Sixty undergraduate females participated. They were divided into four groups based on history of asthma and panic disorder (Asthma only, Panic only, Asthma and Panic, Control). Participants completed questionnaires that assessed depressed mood, anxiety, anxiety sensitivity, self-focus on bodily sensations, fear of bodily sensations, and misinterpretation of bodily sensations. Each participant engaged in a 5 min baseline and three experimental tasks: breathing through a straw, turning one's head from side to side,

and relaxation. Following each task, spirometry measurements were taken, and participants completed measures of panic and asthma symptoms, panic attack related cognitions, and mood.

Women with panic disorder (with or without asthma) reported more depressed mood, trait anxiety, self-focus on bodily sensations, fear of bodily sensations, and anxiety sensitivity (on one of two measures of anxiety sensitivity) than women with asthma only or controls. Women with both asthma and panic disorder reported higher levels of asthma-related symptoms than women with asthma only. Little support was found for the application of cognitive theory to asthma alone, however, cognitive theory continued to explain results for individuals in the panic groups. Likewise, dyspnea/suffocation fear theory was not supported by the present study, nor was Carr's hypothesis. Implications of these results for the two theories and Carr's hypothesis are discussed. In addition, suggestions for future research are made.

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## Chapter 1

### INTRODUCTION

Asthma has long been viewed by both the medical and psychological community as a chronic respiratory illness that may be provoked or exacerbated by psychological factors. Knowledge of the relationship of asthma to psychological factors arose from an accumulation of case studies and anecdotes about the relation between emotional upset or exposure to benign objects and the exhibition of asthma symptoms (Dekker & Groen, 1956; Dekker, Pelser, & Groen, 1957; French & Alexander, 1941; Rees, 1956). For example, MacKenzie (1886) documented the case of a woman with asthma whose symptoms were precipitated by contact with roses. Interestingly, severe symptoms were also triggered by contact with an artificial rose. Over time, a variety of events (e.g., merely looking at dust, using an elevator, seeing a picture of a horse, or looking at a goldfish in a bowl) have been reported to lead to asthma symptoms (Dekker & Groen, 1956). Because asthma symptoms appear at times to be evoked or worsened by an individual's mental state, or by exposure to objects that should not physiologically lead to bronchoconstriction, psychological correlates of asthma continue to be widely researched.

In the medical and psychological literature, there are opposing views about whether or not asthma is indeed caused or exacerbated by psychological factors (e.g., Benjamin, 1977; Ibor, 1956; Purcell & Weiss, 1970). In the 1950s, researchers suggested that emotional factors played a role in the etiology of asthma symptoms (Dekker & Groen, 1956; Dekker et al., 1957; French & Alexander, 1941). However, at the time, it was unclear how much emotional factors contributed and how they operated in creating symptoms (Rees, 1956). Unfortunately, over 40 years later, there has been little progress

toward a definitive resolution of either of these points. Researchers continue to disagree about the role played by psychological factors in the experience of asthma.

A variety of contrasting views about the interaction between asthma and psychological factors have been presented. Psychosomatic models of asthma posit that the disease may be caused or exacerbated by intense emotions or particular personality types (e.g., dependent) or characteristics (e.g., emotional lability; Belloch et al., 1994; French & Alexander, 1941; Henry, Morera, Frugoni, & Gonzalez-Martin, 1993; Lyketsos et al., 1984). Some theorists suggest that asthma and specific psychological disorders (e.g., schizophrenia, agoraphobia, depression) are alternate manifestations of each other (Ibor, 1956; Kelly & Zeller, 1969). In contrast, other researchers propose that asthma is not caused by psychological phenomena at all, stating that the real task in studying asthma should be to examine the development of psychopathology as a consequence of having asthma (Maes & Schlosser, 1987). Conceivably, psychological disorders may occur as a result of living with a chronic illness, not vice versa (Plutchik, Williams, Jerrett, Karasu, & Kane, 1978). According to Maes and Schlosser (1987), it may be more productive to direct research toward questions of psychomaintenance. This area of focus might include such issues as how particular disorders (e.g., anxiety, depression) or methods of coping with asthma affect the course and outcome of the disease (Deenen & Klip, 1993; Janson-Bjerklie, Ferketich, & Benner, 1993; Staudenmayer, Kinsman, & Jones, 1978). Research about the relation between asthma and psychological factors leads to conclusions that lie somewhere between these diverse views. Although it would be inaccurate to state that the etiology of asthma is entirely psychological, it would be a mistake to discontinue researching the role that psychological factors play in the



experience of the disease. It is important to continue to investigate antecedent, concomitant, and consequential psychological phenomena in the hopes of ultimately reducing the symptoms, suffering, and costs associated with asthma.

#### Asthma: Relation to Symptoms and Theories of Panic Disorder

Some of the symptoms of asthma are similar to those of panic disorder (e.g., shortness of breath, choking sensation, chest pain, fear). This similarity may have strengthened the view of asthma as an illness with both biological and psychological causes (Yellowlees & Kalucy, 1990; Zandbergen et al., 1991). In addition, high comorbidity rates and the applicability of theories of panic disorder to asthma research have fostered conjoint study of the two disorders.

There is a particularly high rate of comorbidity between asthma and panic disorder. According to the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV; American Psychological Association [APA], 1994), the 1-year prevalence rate of panic disorder in the general population is 1% - 2%. For individuals who have asthma, however, it is estimated to be between 6.5% and 24% (Shavitt, Gentil, & Mandetta, 1992; Yellowlees, Alpers, Bowden, Bryant, & Ruffin, 1987). This high rate of comorbidity has led to much speculation about the possibility of common etiological factors that could lead to both disorders. Because symptoms of asthma are similar to symptoms of panic disorder, two theories (i.e., dyspnea-suffocation fear theory, cognitive theory) that have proved useful for understanding panic disorder have been applied to asthma.

The revised dyspnea/suffocation fear theory addresses the effects of the perception of difficulty in breathing on the production of panic symptoms (Ley, 1989). Dyspnea

refers to the sensation that breathing is impaired, whereas suffocation refers to a bodily condition that may lead to asphyxiation (Ley, 1994; 1998). Suffocation can occur with little or no discomfort or overt anxiety symptoms. This phenomenon has been observed in infants who suffer from Ondine's curse, a disorder wherein an infant temporarily stops breathing when asleep (Klein, 1993). Oddly, a child with this disorder does not exhibit symptoms of discomfort or distress when breathing ceases. In contrast, the dyspnea/suffocation fear theory posits that it is the experience of severe dyspnea in a context in which an individual believes he or she has little control over the causes of the sensation (e.g., having one's head forced under water; an uncued panic attack) that leads to the experience of panic. The theory's connection to asthma is reflected in the experience of dyspnea, which may lead to increased anxiety and further difficulty in breathing (Dirks, Kinsman, Staudenmayer, & Klieger, 1979; Jones, Kinsman, Dirks, & Dahlem, 1979; Purcell & Weiss, 1970).

Cognitive theory has been extensively discussed in relation to the understanding and treatment of panic disorder. Recently, cognitive theory has been applied to the experience of asthma (e.g., Carr, 1998; Carr, Lehrer, & Hochron, 1995; Carr, Lehrer, Hochron, & Jackson, 1996). According to cognitive theory, panic disorder develops as a result of the experience of ambiguous sensations that occur in the body. These sensations are then misinterpreted by an individual as having catastrophic consequences (e.g., "I am dying"; Clark, 1986). Next, the catastrophic cognitions lead to increased anxiety and anxiety-related sensations, continuing the cycle (Figure 1.1). In relation to asthma, it has been hypothesized that misinterpreting or catastrophizing about ambiguous or asthma-

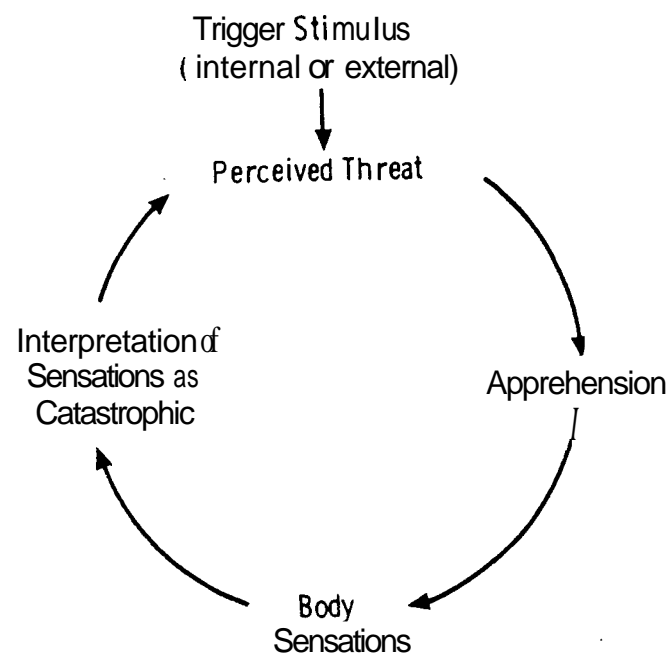


Figure 1.1 Representation of the sequence of events hypothesized to lead to panic attacks in Clark's (1986) cognitive theory of panic

related bodily symptoms may result in increased fear, that in turn may increase the severity of asthma symptoms (Dorhofer, Sigmon, & Boulard, 1998).

Investigations of the utility of these two theories in explaining asthma symptoms (as well as the experience of panic) have produced mixed findings. There has been some support for the dyspnea/suffocation fear theory in relation to asthma (e.g., Carr, Lehrer, & Hochron, 1992; see Ley, 1994 for criticisms). Support for cognitive theory has been mixed (e.g., Carr et al., 1995; Carr et al., 1996; Dorhofer et al., 1998; see Carr, 1998 for opposing viewpoint). The utility of these theories may be in helping researchers better understand how symptoms of asthma can be triggered or exacerbated by cognitive interpretations of physiological sensations, or the sensations themselves. In turn, this understanding could lead to interventions that could possibly reduce asthma symptoms, suffering, and expense (Klingelhofer & Gershwin, 1988; Lehrer, Sargunaraj, & Hochron, 1992; Rachelefsky, 1987). Thus, it may be important for the two theories to be tested together in an experimental paradigm in order to assess their applicability to the experience of asthma. These two theories are further discussed in a later section.

#### Asthma: General Characteristics

In order to discuss research pertinent to the psychological contributions to asthma symptoms, it is important to understand the basic characteristics of the disease. The physiological mechanisms and epidemiology of asthma have been studied for many years. It is now possible to understand trends in a number of epidemiological studies as well as the basic physiological changes that underlie an asthma attack.

### Asthma Prevalence and Demographics

Asthma represents one of three types of chronic obstructive pulmonary disease. Other types include chronic bronchitis and emphysema (Creer, 1979). Approximately 0.3% - 7.9% of the population of the United States suffers from asthma (Bonner, 1984; Cookson, 1987; Rees, 1980). Between 1965 and 1983, hospitalization rates for adults with asthma increased by 50%, whereas in children, the rates increased by 200% (Evans et al., 1987). This dramatic increase in asthma rates continued in the period between 1980 and 1987, when the prevalence of asthma in the United States increased by 29% (Expert Panel Report [EPR], 1991). In addition, asthma-related death rates increased by 40% between 1982 and 1991 (Center for Disease Control [CDC], 1995). Researchers have yet to discover why the mortality rates have steadily increased.

Approximately 63% of individuals with asthma experience symptom onset before the age of 15. In some studies, males appear to be slightly more prone to asthma (0.6% - 4.7% for men, 0.6 - 1.5% for women; Evans et al., 1987; Rees, 1980). However, other estimates of gender differences in asthma rates indicate that age may be an important factor as well. In one community sample, no significant gender differences were found for individuals younger than age 30 (Dodge & Burrows, 1980). However, for individuals between 30 and 50, the rates of active asthma for women was nearly double those for men (although this difference was not statistically significant). Beyond age 50, the rates for men again exceeded those for women (Pearlman & Bierman, 1988). Asthma is, therefore, a relatively common disease that is increasing in prevalence. The necessity of identifying factors that can reduce asthma morbidity and mortality is clear.

It is particularly important to investigate correlates of asthma in a female population for several reasons. First, as described above, asthma rates may be higher for women in certain age groups (Dodge & Burrows, 1980). Second, approximately 75% of adults hospitalized for asthma are female (Skobeloff et al., 1996). Third, a larger increase in mortality rates was observed between 1982 and 1991 for women than men (59% for women, 34% for men; CDC, 1995). These differences in hospitalization and death rates indicate that there are additional factors beyond prevalence alone that account for gender differences in asthma severity and use of health care resources.

Asthma remains a costly disorder. In 1976, total cost of asthma in the United States was estimated to be \$1.3 billion dollars. By 1992, that figure, comprising inpatient hospital services and lost productivity, reached \$6.2 billion (Weiss, Gergen, & Hodgson, 1992). With medical costs increasing yearly for the treatment of asthma, the prevention or curtailing of symptoms and attacks may serve to decrease these overall costs (Dirks & Kinsman, 1981; Kaptein, 1982).

In summary, asthma rates, deaths due to the disease, and societal costs of the disorder have been on the rise according to research conducted over the past 30 years. Because of specific increases in hospitalization and death rates for women, it is important to examine factors in addition to gender that may lead to differential asthma severity. These factors include the role of stress and anxiety, which may operate differentially for males and females.

#### Physical Alterations During an Asthma Attack

Asthma represents a chronic disorder characterized by three major elements: airway obstruction that is reversible either spontaneously or in response to treatment,

airway inflammation, and hyperresponsivity of the airways (American Thoracic Society [ATS], **1962; 1987**; Bailey, Higgins, Richards, & Richards, **1992**; EPR, **1991**; Pearlman, **1984**). Asthma generally occurs in discrete episodes or “attacks,” that are reversible with or without medical intervention (Hogg, **1984**). The severity and pattern of asthma attacks varies greatly from individual to individual (Purcell & Weiss, **1970**). Some individuals with asthma may only need to utilize occasional medication in the presence of a specific trigger, whereas others may not need to use medication at all (Alexander, **1981**). More severe asthma may not be completely reversible or controllable through use of regular medication (Creer, **1982**; Matts, **1984**). In this case, the individual is never completely without some asthma-related symptoms (American Medical Association [AMA], **1996**).

When asthma symptoms are triggered by a particular stimulus (e.g., exercise, overbreathing, airway cooling, allergic irritation, emotional factors), a variety of physiological changes occur in the respiratory system (Matts, **1984**). These changes include constriction of the smooth muscles in the airways and swelling or contraction of the bronchial walls, mucosal edema and secretion of thick mucus, and infiltration of the inflammatory cells (Creer, Reynolds, & Kotses, **1991**; Hogg, **1984**; Scanlon, **1984**). When lung obstruction increases, the sternocleidomastoid muscles maximize chest expansion, leading to breathing at a level near total lung capacity (EPR, **1991**).

Infiltration of the inflammatory cells involves the release of chemicals (e.g., histamine, prostaglandins) by mast cells into the mucosal, submucosal, and smooth muscle layers (McFadden, **1980a**). This infiltration results in increased permeability of the capillaries, leading to inflammation. The inflammatory response results in the secretion of substances (e.g., mucus, proteins) that may occlude the smaller airways

(ATS, 1987; Cross, 1994). In addition, the mucus secreted becomes more viscous, and during an asthma attack, the speed at which it is cleared from the airways decreases (McFadden, 1980b). During a severe attack, mucus plugs may form in the airways, further reducing an individual's ability to breathe (Nadel & Barnes, 1984).

Although individuals' symptom patterns may differ, the three major symptoms of hyperresponsivity, airway obstruction, and inflammation are generally observed across individuals with asthma. When symptoms are triggered, bronchial muscles constrict, the lungs may hyperinflate, and thick mucus may be secreted. Each of these physiological changes contributes to difficulty in breathing.

### Measures of Respiration

Several different measures of bronchial change have been used in asthma research. To some extent, each of these measures is related to the effort exerted by an individual. When asthma symptoms are present, the narrowing of the bronchial tubes leads to the reduction of vital capacity, expiratory flow rate, and maximal voluntary ventilation, as well as increased airway resistance (ATS, 1962; see Table 1.1). These symptoms are measurable via apparatus designed for the monitoring of lung function. Frequently, a spirometer or a peak flow meter is used. This process involves having an individual take a deep inhalation and then exhale as rapidly and completely as possible into the apparatus (Hyatt, Scanlon, & Nakamura, 1997). Less frequently, the more invasive procedure of forced oscillation pneumography is used.

### Spirometry

Spirometry assesses several different aspects of lung function, including forced vital capacity (FVC), forced expiratory volume, measurements during the middle of the



Table 1.1

Measures of lung function: increase vs. decrease during an asthma attack

Measures that Increase	Measures that Decrease
Total lung capacity	Vital capacity
Functional residual capacity	Forced vital capacity
Residual volume	Forced expiratory volume at 1 second
Total respiratory resistance	Maximal voluntary ventilation
Peak expiratory flow rate	Forced expiratory volume at 1 second
	Forced vital capacity

exhalation, and total respiratory resistance. The overall purpose is to measure changes in the rate at which the lungs change volume during forced exhalations (Hyatt et al., 1997).

As measured by a spirometer, FVC reflects the total amount of air exhaled forcefully after a complete inhalation. Vital capacity positively correlates with the level of lung hyperinflation that occurs during an asthma attack (EPR, 1991). However, disagreement exists about the sensitivity of FVC as a measure. Some researchers report that FVC is very sensitive to narrowing of the airways during an asthma attack (Hyatt et al., 1997), whereas others contend that this measure alone may not be reliably affected by asthmatic symptoms (Carr et al., 1992). In addition, FVC is effort-dependent and may not be sensitive to changes in total respiration (Miller & Kotses, 1995).

Forced expiratory volume at 1 sec ( $FEV_1$ ) represents the amount of air expelled in the first second after the start of exhalation. An estimation of the change in function of the large airways that narrow during an asthma attack is provided by this measure.  $FEV_1$  is sensitive to the level of airway obstruction (which reduces the speed at which air is exhaled), as well as disease severity (Hyatt et al., 1997; Laszlo, 1994). A decrease greater than or equal to 20% is considered clinically significant (Isenberg, Lehrer, & Hochron, 1992b).

These two measures (i.e.,  $FEV_1$  and FVC) are frequently combined in a ratio format ( $FEV_1/FVC$ ) to provide an index of the percent of an individual's full lung capacity expired after the first second. Generally, a normal adult is able to exhale between 75% - 85% of their total lung capacity after 1 sec, 94% after 2 sec, and 97% after 3 sec (Creer, 1979). This ratio is important for two reasons. First, it provides the interpreter with the ability to identify individuals who have airway obstruction as well as

reduced FVC, indicated by a low ratio (Hyatt et al., 1997; Laszlo, 1994). Second, the ratio assists in the identification of the cause of low  $FEV_1$ . If  $FEV_1$  is low, but the ratio is normal, this finding may indicate that restriction is occurring. Alternatively, a decreased overall ratio indicates that obstruction is likely (Carr et al., 1992).

Forced expiratory flow at 50% vital capacity ( $FEF_{50}$ ) represents the rates of air flow in the middle airways. It is much less effort dependent than  $FEV_1$ . However, a larger range of values are considered to be normal, leading to difficulty in interpretation (Asmundson & Stein, 1994; Carr et al., 1992; Hyatt et al., 1997).  $FEF_{25-75}$  reflects the average rate of air flow during the middle half of the exhalation. This rate represents the state of both large and small airways. Generally, values are greater than 150 L/min for men and 100 L/min for women (Creer, 1979).

Total respiratory resistance ( $R_t$ ) is a measure of the air flow resistance of the airways, lung tissue, and chest wall. When an individual experiences an asthma attack, constriction of the bronchioles leads to an increase in the measure of air flow resistance (Miller & Kotses, 1995). However, increased airway resistance may or may not be related to asthma symptoms. Normal individuals often exhibit temporary (less than 1 min in duration) responses to bronchoconstrictive suggestion (Isenberg, Lehrer, & Hochron, 1992a).

In summary, the use of spirometry in asthma research is reflected in several commonly used measures. Of these measures (e.g., FVC,  $FEV_1$ ,  $FEF_{50}$ ,  $R_t$ ), the most frequently used and most informative appear to be  $FEV_1$  and the ratio of  $FEV_1/FVC$  (e.g., Aitken, Zealley, & Rosenthal, 1969; Carr et al., 1992; Carr, Lehrer, Rausch, & Hochron,

1994; Isenberg et al., 1992b). In the present study, these two measures will be utilized as an index of respiratory change in response to experimental tasks.

### Peak Air Flow

Peak flow is measured by a simple apparatus that measures liters of air expelled per min, or an individual's peak expiratory flow rate (PEFR). Many peak flow meters are made of plastic, with a spring device that bumps a marker up to the appropriate number (Chiaramonte & Prabhu, 1982). Peak air flow reflects a relatively rough estimate, and is quite effort-dependent (Hyatt et al., 1997). This measure is less sensitive to small airways obstruction, but does correlate strongly with measures obtained via spirometry (Creer, 1979). PEFR also correlates with the level of lung hyperinflation (EPR, 1991). Overall, peak flow represents a simple measure of lung function, but it is highly effort-dependent, limiting its usefulness.

### Forced Oscillation Pneumography

Forced oscillation pneumography assesses the degree to which obstruction due to bronchoconstriction is occurring. Alternating multiple-frequency pressure oscillations are applied to the airways via a mouthpiece. This procedure results in a measure of total respiratory impedance ( $R_z$ ; Franetzki, Prestele, & Korn, 1979; Lehrer et al., 1996). Measurement of lung function via the forced oscillation method is not effort-dependent, and does not require maximum exertion on the part of the participant. However, this equipment is not easily available to non-medically trained researchers (Sirota, 1982).

### Conclusions about Measurement

Each of the above measures has documented strengths and weaknesses. Unfortunately, there has been no standardization of measurement within the asthma

literature. This lack of a common measurement standard, particularly in older articles, makes it difficult to compare results across studies. More recent studies examining the relationship between asthma and panic disorder have consistently utilized FVC, FEV<sub>1</sub>, FEF<sub>50</sub>, and the FEV<sub>1</sub>/FVC ratio (e.g., Carr et al., 1992; Carr et al., 1994; Isenberg et al., 1992b). The consistent use of these measures in future asthma research should facilitate building a knowledge base and enhance communication among researchers in the field.

### Psychological Approaches to Asthma

A large proportion of the published research in the psychological literature relating to asthma focuses on examining how, why, and when psychological events influence the elicitation of asthma symptoms. One line of research considers how airway changes may occur due to psychological manipulations. A second line of research involves measuring the respiratory effects of exposing an individual with asthma to a particular type of imaginal or real aversive stimuli (e.g., mental arithmetic, imagination of aversive scenes, viewing a graphic movie). A third line examines how suggestion can lead to bronchoconstriction or bronchodilation. These three areas are discussed next.

### Physiological Mechanisms

If psychological stimuli produce airway changes in individuals with asthma, it is important to consider how these changes might occur physiologically (Masuda, Notske, & Holmes, 1966). The autonomic nervous system regulates and controls alterations in lung function, and an increase in arousal may lead to a large change in airway smooth muscle tone (Erskine-Milliss & Schonell, 1981; McFadden, 1980a). Indeed, some researchers have conceptualized asthma as a malfunction of the autonomic nervous system (Holtzman, Sheller, Dimeo, Nadel, & Boushey, 1980; Nadel & Barnes, 1984). The

sympathetic and parasympathetic nervous systems appear to control different portions and functions of the airways. The sympathetic system controls the lower or small airways, whereas the parasympathetic system controls the upper or large airways (Nadel & Barnes, 1984). Similarly, sympathetic nervous system activation leads to bronchodilation, whereas the parasympathetic nervous system produces bronchoconstriction (Erskine-Milliss & Schonell, 1981).

The vagus nerve is one of the 12 cranial nerves, and represents a key component of the parasympathetic nervous system (Scanlon, 1984). Connecting the brain with the lungs, the vagus nerve may represent the specific pathway by which emotion may lead to asthma symptoms (Erskine-Milliss & Schonell, 1981). When this nerve is directly stimulated, airway constriction results (Nadel, Cabezas, & Austin, 1971; Widdicombe, 1975). Constriction occurs due to the release of acetylcholine at the end of the vagus nerve, near the lungs (McFadden, 1980a). The released acetylcholine is then received by receptors in the bronchial smooth muscle tissue, forming cyclic guanosine monophosphate. When an overabundance of guanosine monophosphate occurs, an imbalance with cyclic adenosine monophosphate results. It is this imbalance that leads to airway smooth muscle constriction (Scanlon, 1984). Release of acetylcholine from postganglionic cholinergic fibers of the vagus nerve may also trigger contraction of bronchial smooth muscle as well as mucus secretion (Harries, Parkes, & Lessof, 1981; Nadel & Barnes, 1984). Further evidence for the involvement of vagal activity is found in the cessation of bronchoconstriction by administration of substances that block the vagal efferent pathway (Mussell & Hartley, 1988; Widdicombe, 1975). Similarly, in animal research, cutting the vagus nerve results in bronchodilation (McFadden, 1980b).

Increases in vagal activity lead to more severe constriction of the upper airways than the lower airways (Nadel et al., 1971). Not all individuals with asthma exhibit alterations in lung function in response to psychological stimuli. Therefore, it has been suggested that those who do respond tend to experience more upper airway constriction (thus further implicating the parasympathetic nervous system; Lehrer, Isenberg, & Hochron, 1993).

Evidence for parasympathetic system involvement in bronchoconstriction has been found in a variety of studies investigating the effects of stress and relaxation on bronchoconstriction. In a typical study, peak air flow is measured in controls who have been conditioned to tense or relax their facial muscles in response to a particular cue (Glaus & Kotses, 1983). As the face and throat muscles constrict (measured via electromyography), airway constriction results, leading to a decrease in peak air flow. Alternatively, when participants are asked to relax their facial muscles, bronchodilation results. Interestingly, other types of muscular tension and relaxation (e.g., limb muscle) do not produce alterations in lung function. One explanation for these findings is that sensory information from the facial muscles affects the vagus nerve via trigeminal pathways (Kotses & Miller, 1987). However, it is possible that the role of the facial muscles in producing bronchoconstriction is a facet of the more general diving reflex. This reflex occurs in humans and other animals when the face is immersed in cold water, or cold pressure is applied to the forehead. As a result, the body conserves oxygen by slowing heart rate and limiting blood flow to all parts except the brain. In addition, bronchoconstriction occurs.

Further evidence implicating the vagus nerve in bronchoconstriction has been found in a study that used placebo bronchoconstrictor methodology. In this type of study, individuals with asthma are told that they will be inhaling a substance that provokes bronchoconstriction. In actuality, the substance is inert and should have no effect on lung function. Before the suggestion was given in this particular study, one group of participants was administered a substance known to block parasympathetic nervous system activity. By eliminating the action of the vagus nerve, the measurable effects of bronchoconstrictive suggestion were not observed (McFadden, Luparello, Lyons, & Bleecker, 1969). From these studies, evidence has accumulated suggesting that the vagus nerve and parasympathetic nervous system processes affect lung function.

Parasympathetic nervous system involvement, specifically through stimulation of the vagus nerve, appears to be the conduit by which psychological stimuli lead to airways changes (e.g., Isenberg et al., 1992a, Lehrer et al., 1993). Increased arousal, possibly reflected in increased face and throat muscular tension, stimulates the vagus nerve. In turn, the vagus nerve sends chemicals to the lungs, which increase bronchoconstriction of the upper airways. Therefore, psychological stimuli appear to be capable of affecting the physiological mechanisms underlying asthma symptoms. Research exploring different psychological stimuli that have been utilized to produce asthma symptoms is summarized next.

#### Imagined and In-Vivo Stressful Tasks

The role of imaginal or in-vivo exposure to aversive or stressful events in the production of bronchoconstriction is well documented (e.g., Isenberg et al., 1992a; Kotses et al., 1987). Individuals with asthma may exhibit increased air flow resistance when



imagining symptoms of asthma, imagining the experience of a cough, or imagining emotions such as fear and anger (e.g., Creer et al., 1991). Similarly, the performance of particular types of aversive tasks in a laboratory setting have been shown to lead to increased air flow resistance in individuals with or without asthma (Isenberg et al., 1992a).

In a typical experimental paradigm, individuals with or without asthma would view three films that varied in emotional content (i.e., children with asthma in the hospital, an industrial accident, and a mother deciding whether or not to put her infant up for adoption). In comparison to controls, individuals with asthma tend to respond to each of the three films with increased  $R_t$ , particularly during extremely emotional scenes (Levenson, 1979). More recently, Dorhofer, Sigmon, and Boulard (1997) found that women with asthma who listened to scenes depicting the experience of asthma and negative asthma-related situations (e.g., “You decide to go out with a friend. You go to a local bar, where most people are smoking. You feel your lungs tighten as you breathe in the smoky air.”) exhibited decreased peak air flow and higher levels of anxiety in comparison to controls. These studies demonstrate that exposing individuals with asthma to emotional or asthma-related stimuli consistently reduces lung function compared to controls.

Other types of aversive cognitive tasks have been utilized in asthma research. In one study, male college controls were assigned to perform either an easy or difficult arithmetic task (Kotses, Westlund, & Creer, 1987). The group that performed the difficult calculations exhibited significantly higher  $R_t$ . It should be noted that controls exhibit transient increased lung resistance as well when engaging in arithmetic tasks.

Therefore, it appears that physiological responses of individuals with asthma are not qualitatively different from that of individuals without asthma. This pattern of lung resistance effects indicates that asthma studies that do not include controls may simply be measuring normal human responses, not a particular response caused by the pathology of asthma (Kotses, Hindi-Alexander, & Creer, 1989). Individuals who have asthma do, however, exhibit greater and more consistent increases in  $R_t$  in response to a variety of stimuli than controls. According to some researchers, a quantitative (i.e., occurring along a continuum), rather than qualitative, difference in response may occur (Isenberg et al., 1992a).

The type of aversive task used may result in different types of lung response. In a study of individuals with and without asthma (Lehrer et al., 1996), the effects on lung function, measured via forced oscillation, of active (e.g., mental arithmetic and reaction time tasks) versus passive (e.g., watching 2 gory films) tasks were compared.

Interestingly, when both groups engaged in active tasks,  $R_z$  decreased (resulting in better lung function) in comparison to the passive tasks. According to the researchers, active tasks may activate the sympathetic nervous system, which is responsible for the “fight or flight” response. Activation of this system may balance or override the effects of the parasympathetic system, leading to overall bronchodilation. In contrast, passive tasks may lead to increased parasympathetic activation that results in bronchoconstriction. Thus, the observed alterations in lung function may depend on the nature of the task utilized as a stressor.

In general, it appears that a wide variety of aversive tasks lead to alterations in lung function. In many cases, these procedures lead to decreases in lung function, but in

some cases, an increase may be noted. Further research would be helpful in order to make more definitive statements about the conditions under which bronchoconstriction versus bronchodilation will occur.

### Placebo Bronchoconstrictor Studies

The role that placebos (neutral substances that an individual believes will have a real effect) may play in the development of bronchoconstriction has been widely studied. It is well known in both the medical and psychological literature that placebos are frequently capable of creating measurable psychological or physical alterations in an individual (e.g., Edmeads, **1984**). In the study of asthma, a specific type of research has evolved wherein bronchoconstriction is induced in response to a placebo. In a typical research protocol, participants inhale a substance that is presented as either a neutral substance (control condition) or as a strong bronchoconstrictor (e.g., Kotses et al., **1989**; Luparello, Lyons, Bleecker, & McFadden, **1968**; McFadden et al., **1969**; Miller & Kotses, **1990**; Wigal, Kotses, Rawson, & Creer, **1988**). In reality, the inhaled substance is inert (e.g., saline solution, distilled water, room air) in both conditions. At baseline and after the inhalation(s), airway function is measured via spirometry or peak flow. One early report indicated that full-blown asthma attacks were both induceable and reversible with the use of this methodology (Luparello et al., **1968**). In this type of study, researchers hypothesize that the physiological mechanism responsible for the airway response is the vagus nerve, as the body responds to heightened arousal (Spector, Luparello, Kopetzky, Souhrada, & Kinsman, **1976**).

Subsequent studies, however, have not reported the occurrence of full-blown asthma attacks. More frequently, less severe but measurable alterations in lung function

occur. For example, a group of 29 individuals with asthma inhaled a saline solution on five occasions (Janson-Bjerklie, Boushey, Carrieri, & Lindsey, 1986). During the first (control) trial, participants were informed that they were inhaling an inert substance. During the second through fifth (suggestion) trials, participants were informed that they were inhaling a chemical in progressively increasing doses that would cause chest tightness and wheezing. Although no full-blown asthma attacks were noted, 34% of the participants responded to the suggestion with a clinically significant increase in airway resistance (i.e., greater than 20% above baseline).

Similar studies have been conducted using variants of this basic methodology. Alterations in the focus of the experiments have led to the use of different types of inert substances (e.g., distilled water, saline) and different orders of control versus suggestion trials. Reviewing the use of saline as an inert substance, Isenberg et al. (1992a) calculated that approximately 48% of participants responded to the suggestion trial with a clinically significant deterioration in lung function (20% decrease, e.g., Luparello et al., 1968; Luparello, Leist, Lourie, & Sweet, 1970; McFadden et al., 1969; Philipp, Wilde, & Day, 1972). In comparison, only 17% responded to the control condition.

A large number of placebo bronchoconstrictor studies have been reported in the asthma literature. Most studies report that a proportion of the participants exhibit some bronchoconstriction in comparison to baseline (e.g., Butler & Steptoe, 1986; Janson-Bjerklie et al., 1986; McFadden et al., 1969). According to a review of studies that used bronchoconstrictive suggestion, approximately 30 to 40% of participants exhibit temporary decreases in lung function in response to the suggestion (Isenberg et al., 1992a). However, these authors have suggested a more conservative average of 20%

participant response, taking into account serious methodological problems. This low rate of responsiveness to the experimental manipulation greatly reduces the value of data obtained with this type of methodology for drawing conclusions about the contribution of psychological factors to asthma.

A related methodology involves using a substance that will cause actual bronchoconstriction or bronchodilation. These substances are inhaled with either suggestion of bronchoconstriction or bronchodilation (e.g., receive bronchoconstrictor but told it is a bronchodilator; Luparello et al., 1970; Strupp et al., 1974). One experiment of this type involved participants inhaling methacholine, a known bronchoconstrictor, during two trials. During the first trial, participants were told that they were inhaling either a bronchodilator or a bronchoconstrictor. During the second trial, participants were given the opposite suggestion. For example, one participant was given methacholine during two trials (Pastorello et al., 1987). During the first trial, he or she was told that the inhaled substance would produce difficulty in breathing. During the second trial, the participant was told that the inhaled substance would lead to easier breathing. Thus, individuals were given the same substance but were given two different suggestions about its possible effect. Airway responses to the trials were mixed. In comparison to baseline lung function, statistically significant changes did not occur after the trials. Normally, it would be expected that an individual would experience significant bronchoconstriction when inhaling methacholine. According to the authors, the absence of significant changes indicates that the suggestion prevents the expected bronchoconstriction. On the other hand, the lack of significant changes could be due to inconsistent physiological responses or heterogeneity within the sample.

Many studies that utilize bronchoconstrictive suggestion use small samples and have other serious methodological flaws that reduce the impact of the manipulation. For example, individuals with asthma have hyperreactive airways that are irritated by a wide variety of stimuli. The effects of a particular methodology may not be directly attributable to the experimental manipulation. In addition, simply taking a deep breath before exhaling into a spirometer or other apparatus can cause transient air flow obstruction in some individuals (Gayrard, Orehek, Grimaud, & Charpin, 1975; Gayrard, Orehek, Grimaud, & Charpin, 1979; Orehek, Gayrard, Grimaud, & Charpin, 1975). Therefore, it is important to consider the effects of these flaws or confounds on the results presented in this literature.

Confounds from the type of substance inhaled are frequently observed in placebo bronchoconstrictor studies. The use of nebulized saline as a control substance raises particular difficulties. Inhalation of saline causes bronchoconstriction for some participants, confounding the results (Lewis, Lewis, & Tattersfield, 1983). In some cases, participants respond with bronchoconstriction to both control and suggestion conditions as a result of warm saline's effects on the lungs. Unfortunately, response to the control substance in some cases eliminates any measurable placebo effect (Pastorello et al., 1987). Indeed, particular concentrations of saline have been suggested for use as a challenge task for measurement of airway hyperreactivity in individuals with asthma (Boulet, Legris, Thibault, & Turcotte, 1987; Schoeffel, Anderson, & Altounyan, 1981).

Cooling of the airways has also been shown to cause bronchoconstriction (Deal, McFadden, Ingram, Breslin, & Jaeger, 1980; Spector & Farr, 1974). Room temperature saline (20° C) and warm saline at 37° C produces different results. Bronchoconstriction

as a result of suggestion only occurs when saline is cool, not when it is at room temperature (Lewis et al., 1983). Thus, when the type of substance inhaled in a control condition causes bronchoconstriction, the level of airway change due to the experimental manipulation is difficult to estimate.

Another flaw commonly found in placebo bronchoconstrictor studies reflects the lack of estimates of the clinical significance of the reduction in airway function. A reduction of approximately 20% of peak air flow is considered to be indicative of impairment, and a reduction of 50% or more is likely to require emergency medical intervention (Isenberg et al., 1992a). However, few studies use these guidelines in assessing the degree to which participants responded.

Control groups are rarely used in placebo bronchoconstrictor studies (e.g., Butler & Steptoe, 1986; Horton, Suda, Kinsman, Souhrada, & Spector, 1978; Janson-Bjerklie et al., 1986; Neild & Cameron, 1985; Pastorello et al. 1987). This methodological oversight restricts the conclusions that can be drawn about responses of individuals with asthma versus individuals without asthma. Further research has shown that responses to suggestion occur in a similar manner in individuals without asthma (as described above), throwing further doubt on the utility of the placebo bronchoconstrictor methodology (Wigal et al., 1988).

In summary, although widely used, the placebo bronchoconstrictor/bronchodilator studies suffer from a lack of methodological soundness. Although a moderate percentage of individuals with asthma do appear to respond to these manipulations, the extent to which these findings are valid and generalizable is questionable. Thus, the results of this type of study must be interpreted cautiously.

Currently, due to the variety of problems that commonly occur in placebo bronchoconstrictor studies, asthma researchers have been less likely to utilize this methodology. Rather, less intrusive stimuli (e.g., listening to aversive scenes, mental arithmetic) have been advocated when a task that affects lung function is to be studied (Dorhofer & Sigmon, in press). These stimuli may be more ecologically valid, similar to types of events that trigger asthma symptoms in everyday life.

### Emotional States and Asthma Symptoms

Positive and negative emotional states may accompany or lead to symptoms of asthma. The relation between naturally occurring mood states (i.e., measured several times during the course of the day) and peak expiratory air flow has been investigated in several studies. Mood states and peak air flow are often measured at the same time during an individual's usual daily activities. However, studies of this nature are generally correlational in nature.

Frequently, individuals record their observations several times per day. In one study, boys in a treatment facility wore FM radio transmitters for 4 hours per day (Miklich, Chai, Purcell, Weiss, & Bradley, 1974). Approximately every 20 minutes, each boy was instructed via a transmitter to measure and record peak flow, as well as note mood. For 38% of the boys, significant negative correlations between emotional intensity and peak flow were found.

More recent studies have generally corroborated the finding that peak flow and naturally occurring negative mood states are significantly correlated for some individuals with asthma (e.g., Hyland, 1990; Steptoe & Holmes, 1985). However, not all participants exhibit a negative correlation between mood state intensity and peak flow (as the mood



becomes more intense, peak flow decreases). In addition, the type of mood state (e.g., anger, sadness, joy) accompanied by decreased peak flow may vary from individual to individual. For example, some individuals may exhibit decreased air flow while experiencing anger, whereas other individuals may exhibit decreased air flow while sad, happy, or during other emotional states (Steptoe & Holmes, 1985). A review of studies that induce emotional states in participants in the laboratory identified a subgroup of individuals with asthma (40%) who respond to emotional stimuli (e.g., induction of anger, fear, anxiety) with increased airway resistance (Isenberg et al., 1992a). The authors, however, did not hypothesize why only a subset of individuals with asthma exhibit lung function that correlates with negative mood states.

An additional difficulty in interpreting the results of studies examining the mood-peak flow relation reflects the cyclical nature of this relation (i.e., mood-peak flow relationship is stronger in the evening; Hyland, 1990). Studies of circadian rhythms in asthma have shown that for some individuals, asthma symptoms occur more frequently at night, with increased severity (Barnes, 1985; Clark, 1985; Hetzel, 1981; Hetzel & Clark, 1980). Thus, it is possible that time of day should be carefully considered in studies examining the relations between mood, suggestion, and bronchoconstriction.

Overall, some evidence has been found for a negative correlation between a few types of positive and negative mood states and lung function. However, this research needs to be replicated and extended before stronger conclusions can be made. The heterogeneity of mood states correlated with reduced lung function warrants further investigation. Possibly, mood state may be another avenue by which facial muscle tension or general arousal affects the vagus nerve, leading to bronchoconstriction. One

mood state in particular (e.g., anxiety) has been studied extensively in individuals with asthma.

### The Role of Anxiety in Asthma

#### Asthma-Related Panic-Fear

In individuals with asthma, panic-fear is hypothesized to be an anxious state experienced in response to asthma symptoms. The first type, illness-specific panic-fear, refers to the anxiety experienced in response to asthma symptoms (Kinsman, Dirks, Dahlem, & Heller, 1980). It is measured by a subscale of the Asthma Symptom Checklist (ASC; Kinsman, Dahlem, Spector, & Staudenmayer, 1977; Kinsman, Luparello, O'Banion & Spector, 1973). The second type, generalized panic-fear, is more stable and trait-like, and is measured by a subscale of the Minnesota Multiphasic Personality Inventory (MMPI; Carr et al., 1995; Dirks, Jones, & Kinsman, 1977). According to Dirks et al. (1977), the panic-fear trait scale identifies individuals with asthma who are sensitive, emotionally labile, and either unable or unwilling to persist during stressful life events.

For individuals with asthma, scores on the illness-specific panic-fear scale have been found to be positively correlated with a diagnosis of panic disorder and the tendency to report symptoms of panic in response to asthma symptoms, whereas scores on the trait scale are not (Carr et al., 1995). In addition, the illness-specific scale is related to measures of catastrophic cognitions (e.g., the Agoraphobic Cognitions Questionnaire and the Anxiety Sensitivity Index; Chambless, Caputo, Bright, & Gallagher 1984; Reiss, Peterson, Gursky, & McNally, 1986; Carr et al., 1995). Although both measures have been widely used in the asthma literature, the illness-specific scale appears to be the more

valid, reliable measure. Therefore, discussion of the panic-fear construct below will refer only to findings relevant to this scale.

Research on the panic-fear construct has revealed that high panic-fear levels are related to several negative outcomes for individuals with asthma. Panic-fear levels are positively correlated with the intractability of asthma, increased steroid medication needs for maintenance and overuse of as-needed medication (Carr et al., 1995; Dirks, Fross, & Evans, 1977; Dirks, Jones et al., 1977). Prescription decisions by physicians tend to be influenced by the distress or anxiety level of the patient, not actual respiratory function (Hyland, Kenyon, Taylor, & Morice, 1993). In addition, requests for as-needed medications are more frequent for inpatients with high panic-fear as compared to those with low panic-fear (Dahlem, Kinsman, & Horton, 1979). These requests were independent of lung function for those with high panic-fear (Dahlem, Kinsman, & Horton, 1977).

Although it may be maladaptive to have a high level of panic-fear, it may be just as maladaptive to have low panic-fear. Individuals with low panic-fear levels are often discharged from the hospital earlier than individuals with moderate or high levels (Dirks, Kinsman, Horton, Fross, & Jones, 1978). However, individuals with either very high or very low panic-fear levels are more likely to be rehospitalized than are individuals with moderate levels of panic-fear (Dirks, Kleiger, & Evans, 1978; Kaptein, 1982). Rehospitalization rates have been found to be independent of objective asthma severity (Dirks, Fross, & Paley, 1978). To explain these rates, one hypothesis posits that individuals with high levels of panic-fear tend to overreact to symptoms of asthma and overmedicate themselves during the attack (Dahlem et al., 1977). After the attack has

subsided, these individuals then sharply decrease their medication in order to reduce the medication's side effects. According to these authors, undermedication may lead to the development of symptom recurrence leading to hospitalization or rehospitalization.

In contrast, low levels of panic-fear in individuals with asthma may lead to discounting or minimization of symptoms until they are severe enough to require hospitalization (Dirks, Kinsman et al., 1978). Once individuals with low panic-fear are hospitalized, they may be anxious to be discharged and hurry their recovery. However, they may be discharged too early, leading to the necessity for rehospitalization (Carr et al., 1995). Thus, both high and low levels of panic-fear may lead to behaviors that can be detrimental to an individual's health.

One conceptualization of the panic-fear construct posits that it represents self-focused attention to breathing difficulties. The more attention paid to respiratory symptoms, the higher the resultant level of panic-fear (Jones et al., 1979). Two recent studies found that for individuals with asthma, anxiety sensitivity levels predicted the experience of panic-fear (Carr et al., 1995; Dorhofer et al., 1998). It is likely that these two constructs are related aspects of attention to bodily sensations, and may be important in further understanding how psychological factors affect asthma.

### Anxiety Sensitivity

Anxiety sensitivity, the fear of bodily sensations related to the experience of fear (Reiss et al., 1986), represents an important construct in both anxiety and asthma research. High levels of anxiety sensitivity are positively related to anxiety disorders in general, but particularly to the experience of panic attacks. According to expectancy theory (Reiss, 1991), there are three fundamental fears: fear of injury, fear of anxiety, and

fear of negative evaluation. Anxiety sensitivity relates to fear of anxiety. More specifically, fear of anxiety refers to the belief that experiencing anxiety-related sensations may lead to harmful bodily, social, or psychological consequences (Taylor & Cox, 1998). Individuals with high levels of anxiety sensitivity report increased levels of fear of anxiety symptoms, whereas individuals with low anxiety sensitivity levels do not fear the experience of anxiety (Shostak & Peterson, 1990).

Explanations of anxiety sensitivity have changed over the years. When the fear of anxiety or “fear of fear” concept was first developed, it was posited that fear of anxiety symptoms resulted from interoceptive conditioning related to the experience of panic attacks (Goldstein & Chambless, 1978). Some researchers posit that this hypothesis is unlikely given that some individuals who have never experienced a panic attack report high levels of anxiety sensitivity (Maller & Reiss, 1992). Anxiety sensitivity may thus function as an antecedent, rather than a consequence, of panic attacks. Further research indicates that anxiety sensitivity may develop as a result of childhood learning experiences. A recent study of undergraduates revealed that if an individual’s parents reinforced his or her sick-role behavior, the individual was more likely to have a high level of anxiety sensitivity (Watt, Stewart, & Cox, 1998). Thus, learning to catastrophize about general types of bodily sensations may lead to anxiety sensitivity levels that are higher than normal. Increased anxiety sensitivity may also result from environmental stressors such as trauma or relationship problems (Maller & Reiss, 1992).

Anxiety sensitivity appears to function as a risk factor that is necessary but not sufficient for the development of panic attacks (Maller & Reiss, 1992). High levels of anxiety sensitivity may facilitate conditioning of anxious responses due to a vicious cycle

of amplification and interpretation of anxiety symptoms (Taylor, Koch, & McNally, 1992). The experience of panic attacks increases anxiety sensitivity further, resulting in an escalation of panic due to sensitivity to body sensations (Reiss, 1987). Indeed, anxiety sensitivity levels have been found to predict the development of panic attacks over a three-year period (Maller & Reiss, 1992). A more recent line of research has investigated the effects of differing levels of anxiety sensitivity on participants' responses to experimental tasks. Both individuals with and without asthma have been studied utilizing this methodology.

Anxiety sensitivity levels have been found to predict responses to a variety of stressful tasks in individuals without asthma. After engaging in a hyperventilation task, undergraduates with high anxiety sensitivity levels reported experiencing more bodily sensations and more anxiety than participants with low anxiety sensitivity (Holloway & McNally, 1987). A second study (Donnell & McNally, 1989) compared responses to a hyperventilation task in individuals with or without panic, who differed in anxiety sensitivity levels. Regardless of history of panic, high anxiety sensitivity participants reported more anxiety and more bodily sensations in response to hyperventilation than individuals with low anxiety sensitivity. Highest levels of bodily sensations were found in individuals who were high in anxiety sensitivity and had experienced at least one panic attack. It is important to note that individuals with a history of panic and low anxiety sensitivity responded similarly to individuals with low anxiety sensitivity and no history of panic. Thus, it appears that anxiety sensitivity levels, not history of panic attacks, influences anxiety and reports of bodily sensations in response to hyperventilation.

Less intrusive stressful tasks (e.g., mental arithmetic) employed with individuals varying in levels of anxiety sensitivity have led to mixed results. Anxiety sensitivity does not account for physiological arousal level after an arithmetic task, but does relate to self-reported anxiety after the task (Shostak & Peterson, 1990). Individuals with high anxiety sensitivity levels reported experiencing more anxiety symptoms and a greater level of cognitively experienced anxiety after engaging in mental arithmetic. These results indicate that although actual physiological changes may not occur during a stressful task, individuals with high anxiety sensitivity may erroneously perceive that threatening physiological changes have occurred. A second study examined physiological (EMG, heart rate, skin temperature, epidermal activity) reactions and self-reports of cognitions in response to relaxation and a mental arithmetic task (Borden & Lister, **1994**) in individuals varying in anxiety sensitivity levels and history of panic versus no panic. Results indicated that only panic attack history, not anxiety sensitivity level, predicted reactions to physiological changes in response to the stressful task. The authors concluded that physiological and cognitive/subjective responses in individuals with high anxiety sensitivity are in some cases desynchronous. It is also possible that in this case, the stressful task did not activate sufficient arousal to activate fearful cognitions in participants.

The relation between anxiety sensitivity and asthma is just beginning to be explored. Dorhofer and Sigmon (in press) found that women with asthma alone, with a history of panic attacks, or with asthma and panic attacks had similar levels of anxiety sensitivity. The anxiety sensitivity scores of the asthma alone and asthma and panic history groups were significantly higher than those of controls. In addition, researchers

have hypothesized that observed differences in anxiety sensitivity levels may be a partial cause of increased rates of panic disorder among individuals with asthma. The presence of panic disorder in individuals with asthma is not thought to be due to dyspnea or other asthma-related symptoms (Carr, 1998). Moreover, panic disorder in individuals with asthma appears to be influenced by catastrophic misinterpretation of bodily sensations, similar to individuals who do not have asthma but do have panic disorder (Carr et al., 1994). According to proponents of the cognitive theory of panic disorder, without catastrophic misinterpretation, panic disorder does not occur (e.g., Clark, 1986).

Overall, a rapidly growing body of literature has provided general support for the existence of anxiety sensitivity as an independent, valid construct. In addition to its key role in panic disorder research, recent studies have shown the importance of anxiety sensitivity in individuals' responses to stressful tasks. Currently, the promising relation between anxiety sensitivity and the experience of asthma may, with further research, assist in the development of new theories about the relation between psychological variables and asthma symptoms. Because of the relation between anxiety sensitivity and both asthma and the development of panic disorder, it is important to further explore the nature of the relation between asthma and panic disorder.

### Panic Disorder

Panic attacks involve a sudden sensation of intense fear or discomfort with a variety of accompanying symptoms (e.g., increased heart rate, sweating, shortness of breath, dizziness, nausea, numbness or tingling, and fear of losing control, going crazy, or dying; APA, 1994). Estimates of the lifetime prevalence of panic disorder in the general population range from 1.5% - 3.5%, with one-year prevalence rates of 1% - 2% (APA,



1994). For women, the rate of panic disorder is approximately twice that of men (APA, 1994; McNally, 1990). Interestingly, the experience of panic attack symptoms can closely resemble symptoms of asthma. Indeed, asthma and panic disorder frequently coexist and may be exacerbated by one another (Shavitt et al., 1992; van Peski-Oosterbaan, Spinhoven, van der Does, Willems, & Sterk, 1996). Current models of panic disorder are currently being applied to conceptualizations of asthma in the hope of identifying common factors and points of intervention.

Panic disorder has been the focus of an explosion of research interest since the publication of the 3<sup>rd</sup> version of the DSM, which clearly delineated panic disorder as an independent diagnosis (APA, 1980; Barlow et al., 1985). Previous to DSM-III, panic attacks generally were of little interest except as a manifestation of other disorders (Rapee & Barlow, 1993). Since that time, disagreement has arisen concerning the possibility of truly “spontaneous” panic attacks (Margraf, Ehlers, & Roth, 1986a). Panic attacks with all their accompanying symptoms are now reproducible in the clinical or laboratory setting, indicating that perhaps panic attacks are not solely spontaneous as was once thought. A number of different panic challenge tasks have been developed that can elicit panic symptoms for the purpose of research and treatment.

Although individuals who experience panic attacks may report that their attacks have no cue, findings suggest that there may be external precipitants to the occurrence of an attack (e.g., stress, exposure to a feared situation, Hibbert, 1984). In addition, internal events (e.g., the experience of bodily sensations) followed by catastrophic cognitions have been shown to be a frequent precursor of panic attacks (Clark, 1986; Ley, 1985).

Because panic attacks frequently have an identifiable and replicable precursor, it is possible to create symptoms similar to those of panic attacks in the laboratory.

Panic challenge tasks. Many types of biological challenges have been utilized in order to study panic attacks in the laboratory setting or as aids in treatment studies. This type of study has investigated the effects of inducing panic-related physiological sensations in individuals with high anxiety sensitivity, panic attacks, panic disorder, and controls. These challenges typically involve an activity that produces bodily sensations similar to those experienced in a panic attack. In a typical challenge task, an individual is infused with sodium lactate, norepinephrine, or isoproterenol, given an oral dose of yohimbine or caffeine, or asked to inhale a mixture of carbon dioxide and oxygen (McNally, 1990). Each of these substances has led to self-reports of panic attack symptoms and/or attacks in most individuals with either a history of panic attacks or a diagnosis of panic disorder. However, other activities that do not involve ingestion of a chemical or other substance have been utilized in research on and treatment of panic disorder (e.g., voluntary hyperventilation, spinning in a chair).

In the treatment of panic disorder, one common component includes exposure to panic sensations (e.g., Barlow & Craske, 1994; Hecker & Thorpe, 1992). Producing these sensations in a therapy setting is accomplished via such activities as voluntary hyperventilation, shaking the head from side to side, spinning in a chair, and breathing through a straw (Clark, 1993; Hecker & Thorpe, 1992). For example, turning one's head from side to side produces sensations that often accompany a panic attack such as dizziness and nausea, whereas breathing through a straw can produce feelings of restricted air flow or dyspnea (Barlow & Craske, 1994).

Individuals who have never experienced a panic attack generally do not respond with panic or increased anxiety symptoms to panic challenge tasks (e.g., Balon, Yergani, Pohl, Muench, & Berchou, 1990; Donnell & McNally, 1989; Fyer et al., 1987; van den Hout, van der Molen, Griez, Lousberg, & Nansen, 1987). Interestingly, individuals without panic report the same bodily sensations as those with panic, but they do not report the accompanying anxiety (Gaffney, Fenton, Lane, & Lake, 1988; Margraf, Ehlers, & Roth, 1986b). According to some researchers, it is a fearful response to the induced bodily sensations that leads to a panic attack (e.g., Clark, 1986; McNally, 1990). However, other researchers posit that although catastrophic cognitions in some cases can result in a panic attack, cognitions do not invariably accompany or lead to the experience of a panic attack (Rachman, Lopatka, & Levitt, 1988; Westling & Ost, 1993). Panic may occur in the reported absence of these thoughts in approximately 27% of attacks (Rachman et al., 1988). An additional study found that nearly all of the full-blown panic attacks (91%) were accompanied by catastrophic cognitions, with only 57% of the limited symptom attacks characterized by this type of thinking (Westling & Ost, 1993). Thus, the evidence for catastrophic cognitions as causal agents in panic disorder has been mixed and warrants further investigation.

Comorbidity of Asthma and Panic Disorder. A number of studies have examined comorbidity rates of asthma and panic disorder. Including estimates for asthma as well as other types of chronic obstructive pulmonary disease, these rates yield a conservative median of estimates of 9.7% (van Peski-Oosterbaan et al., 1996). Rates of panic attack history are higher, with approximately 45% of individuals with asthma experiencing at least one unexpected panic attack (Perna, Bertani, Politi, Colombo, & Bellodi, 1997).

The presence of panic disorder in individuals with asthma is not thought to be related to the degree of pulmonary impairment experienced (Carr et al., 1994). That is, more severe asthma is not necessarily accompanied by higher rates of panic disorder. However, panic attacks are more common for individuals with asthma if they have high levels of anxiety sensitivity. Among a sample of individuals with asthma who reported high anxiety sensitivity, 35.3% were diagnosed with panic disorder (Carr et al., 1994).

It has been argued that these high comorbidity rates may be in part due to selection bias (van Peski-Oosterbaan et al., 1996). Individuals who experience both a chronic illness and panic disorder are more likely to seek treatment for either or both conditions. In turn, these treatment-seeking individuals are often recruited for study participation. The method by which the diagnosis is obtained also may have an effect on comorbidity rates. Different interview schedules have resulted in greatly discrepant estimates (van Peski-Oosterbaan et al., 1996). Therefore, it is important to consider carefully the type of measure used to diagnose panic disorder when estimating the prevalence of panic in asthma. However, even the lowest estimate of panic disorder found in individuals with asthma is much higher than the rate in the general population. This high rate of comorbidity suggests that there is a particular connection between asthma and panic disorder that merits further examination.

Hypothesized Effects of Comorbidity. When individuals with asthma sense that they may be starting to have an asthma attack, they may experience sensations of fear or panic. This increased anxiety may make asthma symptoms worse through an increase in parasympathetic nervous system activation (Rumbak, Kelso, Arheart, & Self, 1993). However, for individuals with asthma, it has also been hypothesized that having

symptoms of panic disorder may actually be adaptive, rather than maladaptive (Carr, 1998). To test this hypothesis, individuals with asthma, panic disorder, or both conditions engaged in stressful tasks (i.e., arithmetic task, reaction time task, viewing gory films; Carr et al., 1996). In response to these tasks, individuals with either asthma or panic disorder exhibited decreased airway resistance. In addition, individuals with panic disorder, with or without asthma, exhibited lower airway impedance at baseline than those with asthma only at baseline. Surprisingly, individuals with asthma and panic disorder exhibited lower airway impedance at baseline than individuals with asthma. After the tasks, no significant results were found for the interaction between asthma, panic, and task. However, at baseline, individuals with asthma were found to have significantly higher airway impedance (more difficulty breathing) in comparison with those with asthma and panic disorder.

A nonsignificant trend for better lung function for the asthma and panic disorder group was noted following the film tasks. This contrast between the lung function of the asthma and asthma and panic disorder groups suggests that having both disorders may make it easier to breathe. At present, it is unclear what role psychological or physiological processes play in this phenomenon. Because only one published study reported this facilitation, replication is necessary. According to the researchers, these results are congruent with previous research indicating that low levels of panic-fear in asthma result in higher levels of asthma-related mortality. These deaths are thought to be a result of decreased sensitivity to bodily symptoms that can be fatal if ignored (Carr et al., 1996).

Individuals with asthma may suffer from several different types of anxiety, including illness-specific panic-fear, high anxiety sensitivity, panic attacks, and panic disorder. Some researchers hypothesize that excessive or insufficient panic-fear levels are detrimental to an individual's ability to accurately assess their asthma symptoms, leading to increased medication intake or hospitalization (Carr et al., 1995). Possibly, panic-fear resembles anxiety sensitivity in that both constructs describe attention to and fear of bodily sensations. In addition, individuals who have asthma and high levels of anxiety sensitivity may be more vulnerable to the development of panic disorder. Preliminary research indicates that having both disorders may actually facilitate breathing during stressful tasks rather than impeding it (Carr et al., 1996). Therefore, it is important to explore the practical and theoretical implications of the comorbidity of panic and asthma.

### Relevance of Psychological Theories to Asthma

#### Psychosomatic Theories of Asthma

Historically, an influential literature review on psychogenic factors in asthma (French & Alexander, 1941) strengthened the view of asthma as psychosomatic in nature. Using data from case studies, French and Alexander concluded that allergic and psychological factors worked in concert to produce asthma symptoms. In addition, they cited evidence of the efficacy of hypnosis in preventing asthma attacks in response to known allergens. According to psychoanalytic theory, individuals with asthma may be experiencing separation anxiety (expressed as a suppressed cry) that may lead to an attack (Purcell & Weiss, 1970; Turnbull, 1962). According to this view, treatment based on psychoanalytic theory could assist individuals with asthma in working through their

unconscious conflicts, resulting in reduced numbers of asthma attacks (French & Alexander, 1941). However, empirical studies have failed to confirm the tenets of the psychosomatic theory of asthma (Creer, 1982).

Over time, this view has been generally superseded by a biopsychosocial view which emphasizes genetic and biological components that may be responsible for the initial development of asthma (e.g., Matts, 1984; Rees, 1980). However, as discussed above, psychological and social stimuli may also have an effect on the severity of symptoms and the manner in which treatment is conducted. Two psychological models, dyspnea/suffocation fear theory and cognitive theory, have been extended to the conceptualization of asthma severity and frequency.

#### Dyspnea/Suffocation Fear Theory

Ley (1989) proposed the dyspnea/suffocation fear theory to explain the occurrence of panic attacks. In later articles, three types of panic attacks were described in order to account for individual differences in panic symptoms (Ley, 1992a; 1992b). Currently, the dyspnea/suffocation fear theory is most applicable to one of the three types of panic attacks, or hyperventilatory panic attacks (Ley, 1998). Because of the similarity of symptom experience between panic attacks and asthma, this theory has since been extended to asthma.

Dyspnea is a commonly reported sensation during a panic attack (de Ruiter, Garssen, Rijken, & Kraaimaat, 1992; Ley, 1989). According to the dyspnea/suffocation fear theory, the fear that accompanies a panic attack results from severe dyspnea that is induced by hyperventilation. The panic attack itself occurs when an individual feels that he or she has little or no control over the dyspnea (Ley, 1998).

Further evidence for the role of dyspnea in panic comes from observations of the frenzied activity that can occur during a panic attack (Ley, 1989). Ley hypothesizes that this activity may serve to increase metabolic  $\text{CO}_2$  levels, thus reducing the sensation of dyspnea. However, recent studies involving panic challenge tasks have utilized  $\text{CO}_2$ -enriched air to provoke panic-related sensations. Conversely, during relaxation, metabolic  $\text{CO}_2$  levels are known to decrease. This decrease may account for the experience of panic cued by relaxation or those occurring during sleep (Ley, 1988a; 1988b). Although these findings appear contradictory, Ley describes how  $\text{CO}_2$  inhalation may produce both panic-related sensations and pleasant sensations (Ley, 1989). For approximately 1 minute after inhaling  $\text{CO}_2$ -enriched air, participants generally report experiencing the sensation that breathing is uncontrollable, leading to acute anxiety. During this time, heart rate increases, dissipating the  $\text{CO}_2$ . After the first minute, when  $\text{CO}_2$  levels decrease, participants may report a pleasant relaxation state. Excluding catastrophic cognitions, dyspnea and any resultant experience of fear are posited to be the sole triggers for hyperventilatory panic attacks. According to Ley (1989), common cognitions reported during panic (that are of central importance in the cognitive theory of panic) may actually be due to cerebral hypoxia that occurs as a result of hyperventilation.

In contrast, other researchers argue that dyspnea, hyperventilation, and tachycardia (frequently observed in response to hyperventilation) are produced by cooling or water loss that occurs during overbreathing (de Ruiter et al., 1992). De Ruiter et al. suggest that it is just as likely that dyspnea leads to hyperventilation as vice versa. Regardless of which explanation is correct, individuals who experience panic attacks report that they notice dyspnea and/or other physiological symptoms before experiencing fear (Ley,



1985). According to the dyspnea/suffocation fear theory, the proposed sequence of events in a panic attack is as follows: Hyperventilation → fear → sensations → catastrophic cognitions.

To test the tenets of the dyspnea/suffocation fear theory, ~~Carr~~ et al. (1992) investigated dyspnea in individuals with panic disorder, asthma, and controls. The researchers hypothesized that the high rate of concordance of panic disorder in individuals who have respiratory disorders may be due to the experience of dyspnea. Tests of pulmonary function and self-reports of mood during and after a relaxation task revealed that compared to controls, individuals with asthma and individuals with panic disorder exhibited similar levels of dyspnea and similar levels of psychopathology. Results indicated that although individuals with asthma or panic disorder report comparable levels of dyspnea, panic symptoms are accounted for by different factors. A significant amount of variance in panic symptoms in individuals with asthma is explained by dyspnea, but not for individuals with panic disorder. In other words, panic symptoms in asthma may be due to dyspnea, but in panic disorder, they are not due to dyspnea levels. This difference in the contribution of dyspnea to the experience of panic symptoms suggests that the dyspnea/suffocation fear theory (rather than explaining panic attacks) may be better utilized to more fully understand dyspnea and its relation to asthma.

According to Ley (1994), however, individuals in the above study were selected according to DSM-III-R criteria for panic disorder, not for their experience of hyperventilatory panic attacks. Thus, Ley argues, the lack of support for the dyspnea/suffocation fear theory in relation to panic disorder is due to this methodological

flaw. In rebuttal, Carr and Lehrer (1994) point out that Ley's (1992a; 1992b) subtypes of panic attacks have yet to be empirically validated. Furthermore, Carr and Lehrer (1994) argue that individuals in both the panic disorder and asthma groups reported a level of dyspnea that was significantly greater than controls. Thus, individuals in the panic disorder group did indeed experience a high level of dyspnea. It is possible, however, that this sample was generally composed of individuals experiencing hyperventilatory panic attacks. Overall, the results of these studies must be replicated and extended in order to determine the specific role of dyspnea/suffocation fear in asthma and/or panic disorder.

### Cognitive Theory

The cognitive theory of panic proposes that catastrophic misinterpretation of benign bodily sensations results in the experience of a panic attack (Clark, 1986). Extending this theory to asthma, it is possible that individuals with asthma and a tendency to misinterpret symptoms may experience more frequent and more severe asthmatic episodes. Mislabeling non-asthma related bodily sensations as symptoms of asthma may have serious consequences. In studies of asthma inpatients, 19% - 27% consistently mislabeled relatively benign bodily symptoms (e.g., fatigue, worry, irritability, anxiety) as an asthma attack. Those individuals who exhibited this tendency to mislabel symptoms were more likely to be rehospitalized for asthma at a 6-month follow-up. Forty to 84% of the mislabelers were rehospitalized by 6 months, whereas only 29% - 40% of the non-mislabelers were rehospitalized. There was no measured difference between symptom mislabelers and non-mislabelers in lung function (Dirks & Schraa, 1983). In individuals with asthma, the consequences of symptom misinterpretation may be due to the anxiety-

producing effects of the catastrophic interpretation of benign sensations that may lead to increased bronchoconstriction.

According to Clark (1993), there are three criteria that must be fulfilled in order to demonstrate cognitive mediation of panic attacks induced by biological challenge tasks:

- 1) panic disorder patients have a stronger tendency to misinterpret certain bodily sensations than controls;
- 2) that thoughts based on the misinterpretation of bodily sensations accompany challenge induced panic attacks; and
- 3) that experimental manipulations of cognitive variables have an influence on whether or not an individual panics during a biological challenge test (Clark, 1993, p. 76).

Clark (1993) provides evidence that these three criteria have been fulfilled in the research on panic disorder. Thus, strong support for cognitive mediation of panic attacks during biological challenge tests has been reported in the panic literature.

Criterion One. Criterion One has been confirmed through research utilizing the modified Interpretations Questionnaire (Butler & Matthews, 1983). This questionnaire assesses the extent to which individuals interpret ambiguous internal and external stimuli as threatening. Individuals with panic disorder tend to mistakenly interpret bodily sensations as symptoms of an approaching physical or mental catastrophe more than individuals with other types of anxiety disorders or controls (Clark, 1993). Research with expanded versions of the Interpretations Questionnaire revealed that individuals with panic disorder are less likely to be able to reinterpret stimuli as nonthreatening once they have made an anxiety-related response (e.g., Kamieniecki, Wade, & Tsourtos, 1997).

Criterion Two. The second criterion (thoughts based on the misinterpretation of bodily sensations are present during challenge induced panic attacks) has also been supported. Individuals with panic disorder were exposed to feared situations (e.g., walking alone into a supermarket). Participants completed measures of their level of fear, a checklist of bodily sensations, and a checklist of fearful cognitions after each trial. Approximately **73%** of the panic attacks experienced by the participants were accompanied by self-reports of fearful cognitions (Rachman et al., **1988**). Significantly more fearful cognitions were reported on the trials that led to panic than those that did not, indicating that panic is generally accompanied by these types of catastrophic cognitions.

Criterion Three. The third criterion (manipulating cognitions either leads to or prevents panic attacks in experimental settings) is perhaps the most important of the three criteria. Yet, this hypothesis has been more difficult to unambiguously support. Many studies have revealed that the manipulation of cognitive variables can affect the likelihood of panic symptoms or attacks during challenge tests (Margraf, **1993**; Salkovskis & Clark, **1990**; van der Molen, van den Hout, Vroemen, Lousberg, & Griez, **1986**). To test this hypothesis, studies alter the information participants are given before a challenge task begins.

Instructing two groups of controls that the sensations of hyperventilation are either symptoms of impending fainting or signs of a higher state of consciousness produces very different results (van der Molen et al., **1986**). Although the same bodily sensations and changes in heart rate and pCO<sub>2</sub> were produced in both groups, these sensations were rated as either pleasant or unpleasant, depending entirely on the instructions given (Salkovskis

& Clark, 1990). A similar study examined the effects of instructions on self-reported mood in response to sodium lactate infusion. Two groups of controls were informed that they would either experience anxious tension or pleasant excitement in response to the infusion. As was expected, the group that received the anxious tension suggestion reported significantly higher levels of negative, anxious mood.

Expectations about what symptoms a CO<sub>2</sub>/O<sub>2</sub> challenge task will produce also alter the likelihood of experiencing a panic attack. Individuals with either panic disorder or social phobia were given one of two different explanations of the symptoms they would experience as a result of CO<sub>2</sub>/O<sub>2</sub> inhalation (e.g., Rapee, Mattick, & Murell, 1986). The first explanation gave the individual no expectation about what he or she would experience, whereas the second explanation listed symptoms that are commonly reported during a panic attack. After the inhalation trial, participants were given a list of 10 neutral and panic related cognitions and were asked to indicate what went through their mind when they began to feel the bodily sensations associated with the gas. Individuals in both conditions reported similar levels and types of bodily sensations. However, many more of the individuals who were in the “no explanation” condition experienced a panic attack. The cognitions reported by this group were significantly more likely to be catastrophic in nature (Rapee et al., 1986). The results of this study provide strong evidence that an individual’s fearful cognitions in an ambiguous situation can lead to panic symptoms. Conversely, if an individual can attribute ambiguous bodily sensations to a known cause, then he or she may be less likely to have a panic attack.

Cognitions about situational control may also alter the anxiety responses of individuals with panic disorder. In one study, participants were allowed to “turn down”

the level of CO<sub>2</sub> they were receiving if a light turned on (for half the participants, the light never turned on; for the others, the light was turned on during the entire procedure; Sanderson, Rapee, & Barlow, 1989). In actuality, the dial did not alter the CO<sub>2</sub> level in any way. However, participants who believed they had control over the gas reported fewer and less severe symptoms of panic, less resemblance to panic sensations, and fewer occurrences of actual panic attacks (Sanderson et al., 1989). Studies of this type provide support for the role that cognitions play in the production and/or reduction of panic symptoms and attacks.

In addition, anticipating the negative effect of CO<sub>2</sub> inhalation can produce increased self-reports of physiological responses that indicate increasing levels of anxiety. In comparison, when neutral expectations are fostered for participants with panic disorder, hyperventilation does not result in strong physiological responses (Margraf, 1993). Thus, it appears that it is negative interpretations of bodily sensations that lead to the experience of panic. The symptoms of hyperventilation or any other experience alone do not result in a panic attack.

#### summary

The relationship of asthma with psychological factors has been studied for many years (e.g., French & Alexander, 1941; MacKenzie 1886). A variety of methodologies, including exposure to real or imagined aversive events (Creer et al., 1991; Dorhofer et al., 1997), suggestion of bronchoconstriction (Kotses et al., 1989; Luparello et al., 1968; Janson-Bjerklie et al., 1986), and naturalistic observation of mood states (Hyland, 1990; Miklich et al., 1974; Steptoe & Holmes, 1985), have been used to identify factors that are related to alterations in lung function. These factors include type of task (active versus

passive) and mood (positive versus negative). Classical conditioning and stress provocation have both been suggested as possible causal factors in alterations in lung function (Dekker et al., 1956; Miller & Kotses, 1995).

Recently, the relationship of anxiety with asthma has been investigated in the psychological literature (e.g., Carr, 1998). High panic-fear levels have been linked to negative outcomes (e.g., medication overuse, rehospitalization) for individuals with asthma (Carr et al., 1995; Dahlem et al., 1979; Dirks, Fross et al., 1977; Dirks, Jones et al., 1977; Hyland et al., 1993). High anxiety sensitivity levels also appear to be related to asthma, and may pose **an** additional risk factor in the development of panic disorder (Dorhofer et al., 1997). In addition, it is well established that individuals with asthma are more likely to develop panic disorder than are individuals without asthma (Carr et al., 1994; van Peski-Oosterbaan et al., 1996). Further, the mechanisms and consequences surrounding the frequent comorbidity of asthma and panic disorder have been of interest to researchers (e.g., Carr et al., 1996; van Peski-Oosterbaan et al., 1996). Specifically, the presence of both panic disorder and asthma may lead to better lung functioning in response to stressful stimuli than for individuals with asthma alone (Carr, 1998). At present, it remains unclear what role psychological or physiological processes play in this phenomenon.

Because of the similarity and frequent comorbidity of asthma and panic disorder, two theories (dyspnea/suffocation fear theory and cognitive theory) have been used to explain how asthma symptoms are affected by psychological factors. New studies examining these theories in relation to asthma have resulted in some support for each (Carr et al., 1992; Carr, 1998; Dorhofer et al., 1997). At the present time, no published

research has examined both theories within the same study. These theories may contribute to further understanding the mechanisms underlying the comorbidity and experience of asthma and panic disorder.

### Statement of Purpose

The present research project was designed to investigate dyspnea/suffocation fear theory and cognitive theory in relation to asthma. In addition, Carr's hypothesis regarding the possible beneficial effects of comorbid panic disorder and asthma on lung function was tested. Using experimental challenge tasks that produce asthma and/or panic-like sensations, tenets of these theories and Carr's hypothesis were investigated in women with asthma. Lung function and self-reports of cognitions, bodily sensations, and dyspnea levels were assessed. For individuals with asthma, the presence of panic disorder may actually assist breathing efforts (Carr et al., 1996). Therefore, it may be important to identify what elements of the panic experience are associated with increased lung function (e.g., changes in level of catastrophic cognitions, perception of dyspnea).

In the present study, comparison groups will include individuals with asthma, asthma and panic disorder, panic disorder alone, and controls. The utilization of these four groups allowed comparisons of individuals who may 1) experience aversive bodily sensations and anxiety on a regular basis and 2) may be able to pinpoint or explain the source of the bodily sensations (e.g., individuals with asthma). In addition, comparison of these groups allowed for replication and extension of dyspnea/suffocation fear theory, cognitive theory, and Carr's hypothesis in relation to asthma.

The dyspnea/suffocation fear theory was tested in a manner similar to that of a previous study that used a passive relaxation condition (Carr et al., 1992). In a replication



of previous research, individuals relaxed as deeply as possible with their eyes closed for 5 minutes. In an extension of dyspnea/suffocation fear theory, changes in dyspnea level were assessed before and after engaging in breathing through a straw and head turning. Breathing through a straw and turning one's head from side to side are active tasks designed to induce sensations that are dyspneic (affect perception of lung function) and panic-related (dizziness), respectively (Barlow & Craske, 1994; Hecker & Thorpe, 1992). According to dyspnea/suffocation fear theory, it was expected that individuals with asthma and/or panic disorder would report greater changes in dyspnea levels than controls after a passive task. Similarly, after the head turning task, individuals with asthma and/or panic disorder were expected to report greater changes in dyspnea levels than controls. In contrast, greater changes in dyspnea levels were expected for controls after the straw task, as this group may not have experience with shortness of breath.

To test Carr's hypothesis that having both asthma and panic disorder is adaptive for lung function, an active task was utilized. According to Carr, producing panic-like sensations led to an increase in lung function observed for the comorbid group in previous research. To replicate and extend this hypothesis, individuals with and without asthma and panic disorder engaged in the head turning task (producing panic-like sensations) as well as Carr's relaxation task.

An adaptation of Clark's cognitive model of panic (extended to asthma) and the criteria for evidence of a cognitive component to panic disorder were used to investigate differences in cognitions experienced after the tasks for individuals with or without asthma and panic disorder (Clark, 1986). In addition, changes in panic-related cognitions were assessed after each task.

Extending Clark's model to asthma included the following hypotheses:

- 1) compared to controls, individuals who have asthma fear and misinterpret certain bodily sensations more;
- 2) thoughts based on the misinterpretation of bodily sensations accompany challenge induced asthma symptoms or attacks; and
- 3) experimental manipulations of cognitive variables have an influence on whether or not an individual has asthma symptoms, increased air flow resistance, or an asthma attack during a challenge task.

Unlike the panic disorder literature, the asthma literature is less clear on whether or not these hypotheses have been supported. Hypothesis one, that individuals with asthma fear and misinterpret bodily sensations more than controls, has not yet been investigated. However, previous research (Dorhofer & Sigmon, in press) indicates that as a group, individuals with asthma report higher levels of anxiety sensitivity or fear of anxiety sensations. The present study investigated this hypothesis by having participants complete questionnaires that assessed an individual's tendency to fear, focus on, and misinterpret ambiguous situations or sensations.

Hypothesis two, that thoughts based on the misinterpretation of sensations accompany induced asthma symptoms, has not been investigated in the asthma literature. The present study examined this hypothesis by assessing panic-related cognitions after the experimental tasks.

Hypothesis three, that experimental manipulations of cognitive variables have an influence on whether or not an individual has asthma symptoms, increased air flow resistance, or an asthma attack during a biological challenge test, has been supported

through use of placebo bronchoconstrictor methodology. As discussed previously, this type of study generally produces alterations in lung function depending on the suggestion given (e.g., Isenberg et al., 1992a). For individuals with asthma, it appears that specific cognitions (e.g., expecting that lung function will decrease) do indeed affect the likelihood of changes in airway function.

### Hypotheses

#### Hypothesis One

Compared to baseline, individuals with asthma, panic disorder, and asthma and panic will experience a greater decrease in dyspnea level after the relaxation task and head turning task than controls. After the straw task, controls are expected to experience a greater decrease in dyspnea level than the other three groups.

#### Hypothesis Two

After the relaxation task, the panic and asthma and panic groups will report a greater increase in panic-related symptoms from baseline than the asthma group. In turn, the asthma group will report a greater increase in these symptoms than the control group. After the head turning task, the panic and asthma and panic groups are expected to experience a greater increase in panic sensations than the asthma and control groups. Panic-related symptoms will be equivalent for all groups after the straw task.

#### Hypothesis Three

Individuals in the asthma and panic group will experience an increase in lung function after the relaxation and head turning tasks in comparison to baseline. In contrast, the control group will exhibit no change in lung function. The asthma and panic groups will exhibit a decrease in lung function after these tasks. Following the straw

task, it is expected that the asthma and asthma and panic groups will both show an increase in lung function. No change is expected for the control group, and a decrease is expected for the panic group.

#### Hypothesis Four

After the relaxation and head turning tasks, individuals with panic disorder and asthma and panic will report an increase in panic-related cognitions in comparison to the asthma and control groups. After the straw task, the asthma and asthma and panic groups will report a greater increase in panic-related cognitions than the panic group, with the control group reporting no increase.

#### Hypothesis Five

Individuals in the asthma, panic, and asthma and panic groups are expected to report more self-focus on bodily sensations than the control group.

#### Hypothesis Six

Individuals in the asthma, panic, and asthma and panic groups will report more misinterpretation of bodily sensations than the control group.

#### Hypothesis Seven

Individuals in the panic and asthma and panic groups are expected to report more fear of anxiety sensations than the asthma group. In turn, the asthma group will report a higher level than the control group. Panic-fear levels will be higher for the asthma and panic group than the asthma group, and the panic group's level will be higher than that of the control group.

## Chapter 2

### METHOD

#### Participants

Sixty female participants over the age of 18 were recruited on the University of Maine campus and in the community. The sample included four groups of 15 participants who were diagnosed with asthma, panic disorder, asthma and comorbid panic disorder, or had no diagnosis (control group). A power analysis (based on an effect size calculated utilizing group means from previous research; Carr et al., 1996; see Appendix A) revealed that 15 participants per group would result in a power of .94.

Participants were recruited through psychology classes and through flyers posted at the University of Maine and at stores in Bangor, Maine. For undergraduates in psychology classes, extra course credit was offered. Other participants were offered payment for their participation. All participants read and signed an informed consent form (Appendix B). At the conclusion of the experiment, they were debriefed and received a referral list for psychological treatment.

Participants with asthma and panic disorder were selected based on criteria similar to that used by Carr et al. (1995). An attempt to verify that participants had been diagnosed with asthma was made via contact with the participant's physician. Participants signed a release of information form (Appendix C) that was sent to their physician. The physician completed the form, indicating the individual's diagnosis and age at diagnosis, asthma severity, and any medications that may have been prescribed. Participants had to indicate during the diagnostic interview that their panic symptoms and their asthma symptoms occurred independently (e.g., panic has occurred at times when an

individual's asthma was well controlled). The panic disorder section of a structured diagnostic interview (Anxiety Disorders Interview Schedule for DSM-IV [ADIS-IV]; Brown, Di Nardo, & Barlow, 1994) was utilized to diagnose the presence of panic disorder according to DSM-IV criteria.

### Experimenters

The experiment was primarily conducted by the principal investigator and another advanced graduate student in clinical psychology. Interrater reliability for the ADIS-IV interviews was established by a second rater (an advanced graduate student in clinical psychology). This individual was blind to the experimental conditions. He or she listened to audiotapes of the diagnostic interviews and provided a second rating for each participant. The second rater was trained by the principal investigator in ADIS-IV scoring procedures.

### Dependent Measures

The Anxiety Disorders Interview for DSM-IV. The Anxiety Disorders Interview for DSM-IV (ADIS-IV; Brown et al., 1994) was utilized to establish a diagnosis of panic disorder. Reliability and validity information on the ADIS-IV is not yet available, however, these data are available for the previous versions, the ADIS (Di Nardo, O'Brien, Barlow, Waddell, & Blanchard, 1983), based on DSM-III criteria (APA, 1980), and the ADIS-R (Di Nardo & Barlow, 1988), which was based on DSM-III-R criteria (APA, 1987). Interrater reliability for the ADIS has been examined in several studies. Two outpatient samples resulted in a kappa value of .68 for anxiety disorders in general and kappas for specific disorders ranged between .56 and .90 (Di Nardo et al., 1983). A third study comparing independent diagnoses made by psychiatrists and lay interviewers

utilizing the ADIS led to somewhat lower kappa values (range = .47 - .67). The interrater reliability of the ADIS-R is generally acceptable; kappas range from .57 to .82 (Di Nardo, Moras, Barlow, Rapee, & Brown, 1993).

Anxiety Sensitivity Index. The Anxiety Sensitivity Index (ASI; Reiss et al., 1986; Appendix D) is a 16-item self-report measure that assesses the fear of experiencing anxiety symptoms. Items on the ASI take the form of statements about the possible negative consequences of anxiety symptoms (e.g., “When I notice that my heart is beating rapidly, I worry that I might have a heart attack”). Each item is rated on a 5-point Likert scale (0=very little; 4=very much), and scores range from 0 to 64. High scores on the ASI are highly correlated with agoraphobia, panic disorder, or other anxiety disorders (Peterson & Reiss, 1992). The ASI has acceptable reliability (test-retest at two weeks:  $r = .75$ ; at three years:  $r = .71$ ; coefficient alpha = .88; Maller & Reiss, 1992; Peterson & Reiss, 1992). Validity studies indicate that the ASI is positively related to the Taylor Manifest Anxiety Scale (Taylor, 1953) and the Anxiety Frequency Checklist (Reiss et al., 1986). However, the ASI accounts for a significant portion of variance in Fear Survey Schedule-II (Geer, 1965) scores above and beyond the other two measures (Reiss et al., 1986). In addition, the ASI predicts anxiety levels after hyperventilation, whereas measures of trait anxiety do not (McNally, 1989). Thus, it appears that anxiety sensitivity assesses a component of anxiety not tapped by trait anxiety measures.

Scores on the ASI reliably differentiate between individuals diagnosed with agoraphobia or panic disorder and those with other types of anxiety disorders. In addition, ASI scores discriminate between controls and individuals with anxiety disorders (Peterson & Heilbronner, 1987; Reiss et al., 1986). Thus, it appears that the ASI has

clinical utility for differentiation of individuals with anxiety disorders. This measure is particularly relevant for panic disorder.

Several studies of the factor structure of the ASI have led to the identification of at least three factors. These factors include fear of somatic sensations, fear of cognitive dyscontrol, and fear of publicly observable symptoms. Recently, a new instrument, the Anxiety Sensitivity Profile (see below) has been developed to further explore these factors (Taylor & Cox, 1998). In this study, the ASI was utilized as a measure of group differences in level of fear of anxiety sensations and resulted in a coefficient alpha of .91.

Anxiety Sensitivity Profile. The Anxiety Sensitivity Profile (ASP; Taylor & Cox, 1998; Appendix E) is a 60-item self-report measure that was developed in response to criticisms of the ASI's factor structure. The ASP includes 60 items that question 6 domains of anxiety symptoms and their perceived dangerousness: cardiovascular, respiratory, gastrointestinal, publicly observable anxiety reactions, dissociative and neurological symptoms, and cognitive dyscontrol. Each item is rated with regard to the likelihood that the sensation would lead to a negative outcome for the individual on a 1 (not at all likely) to 7 (extremely likely) Likert scale.

Coefficient alpha for the six subscales of the ASP range from .88 to .94. The ASP subscales correlate significantly with both the ASI ( $r$ s between .41 and .66) and the Trait version of the State-Trait Anxiety Inventory ( $r$ s between .11 and .29; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1982; Taylor & Cox, 1998). Further reliability and validity studies on the ASP have yet to be published. In the present study, only the respiratory subscale of the ASP was utilized as a measure of the perceived dangerousness of respiratory symptoms.



Asthma Symptom Checklist. The Asthma Symptom Checklist (ASC; Kinsman et al., 1977; Kinsman et al., 1973; Appendix F) is a 36-item list of somatic and mood symptoms that are related to asthma. Each item is rated on frequency of occurrence during asthma attacks (1 = never; 5 = always). The ASC is composed of five factors: Panic-fear, irritability, fatigue, ~~hyperventilation-hypocapnia~~, and bronchoconstriction. The ASC provides an index of the specific symptoms an individual experiences during an attack, and may be related to measures of bother, anxiety, and depression. Cluster internal consistency reliabilities for the subscales range from .78 to .92 (Kinsman et al., 1977).

The panic-fear subscale (ASC-PF) has been extensively used to estimate an individual's level of anxiety in response to asthma symptoms. This subscale is hypothesized to measure illness-specific panic-fear (Carr et al., 1995). ASC-PF scores are not related to pulmonary function, indicating that an individual's level of anxiety about their symptoms is not related to the severity of their asthma. However, scores are positively correlated with level of prescribed steroids, excessive use of as-needed medications, and more frequent hospitalizations and rehospitalizations (Dirks, Fross et al., 1977). The ASC was used in two ways. First, it was used to assess the general level of symptoms typically experienced by individuals with asthma during an attack. Second, it was used with all four groups as a state measure after each task to assess the current level of asthma-related symptoms.

Body Vigilance Scale. The Body Vigilance Scale (BVS; Schmidt, Lerew, & Trakowski, 1997; Appendix G) is an 18-item measure that assesses the degree to which individuals focus conscious attention on internal bodily sensations. Each item is rated on

a 1-10 Likert scale (1 = No attention paid to this sensation, 10 = Extreme amount of attention paid to this sensation). Test-retest reliability over a 5-week period is .58 in a sample of individuals with panic disorder. Internal consistency of the BVS is high, with Cronbach's  $\alpha = .82$  for both a sample of undergraduates and a sample of individuals with panic disorder (Schmidt et al., 1997). In a sample of individuals diagnosed with social phobia, Cronbach's  $\alpha$  was slightly lower (.74; Schmidt et al., 1997). In the present study, Cronbach's  $\alpha$  was .82. Body vigilance is significantly correlated with anxiety sensitivity as well as anxiety measured by the Beck Anxiety Inventory, but not STAI Trait anxiety or depression symptoms (Schmidt et al., 1997). In this study, the BVS provided a measure of participants' focus on their bodily sensations.

Borg Category Scale. The Borg Category Scale (Borg, 1982; Appendix H) is a visual analog scale that assesses an individual's level of dyspnea. This measure ranges from 0 (nothing at all) to 10 (maximal) ratings of difficulty breathing. The Borg Scale was utilized between tasks to assess participants' level of dyspnea. This provided a self-report measure of the level of breathing difficulty participants perceive that they are experiencing.

Brief Body Sensations Interpretation Questionnaire. The Brief Body Sensations Interpretation Questionnaire (BBSIQ; Clark et al., 1997; Appendix I) is a 14-item measure based on the Interpretation Questionnaire (McNally and Foa, 1987). The BBSIQ includes two categories of ambiguous events: panic body sensations and external events. There are several steps to completing the BBSIQ. Participants first read each item and then write down "why" the event occurred. Next, they turn the page and are given three explanations (negative, positive, or neutral) to rank-order. Finally, participants return to

the beginning of the measure and rate how much they would believe each explanation if they were in that situation. The ratings range from 0 (not at all likely to be true) to 8 (extremely likely to be true). Clark et al. (1997) note that the written “why” responses are very time consuming to score and do not provide additional information above and beyond the ranking information. Indeed, in the second study in the Clark et al. (1997) article, the “why” responses are not scored at all. Following this convention, these responses were not scored for the purposes of this study.

Internal consistency is satisfactory (Cronbach’s  $\alpha = .86$  for ranking of panic body sensations, .90 for ratings of panic body sensations, .74 for ranking of external events, and .80 for external event belief ratings). For individuals diagnosed with panic disorder, the BBSIQ panic body sensation items correlate significantly with the ACQ Physical Concerns factor, but not with the ACQ Social-Behavioral Consequences factor or the STAI. BBSIQ external event items correlate with the ACQ Social-Behavioral Consequences factor and STAI Trait anxiety, but not with the ACQ Physical Concerns factor. Test-retest reliability has not yet been examined for the BBSIQ. In this study, the BBSIQ provided a measure of the degree to which participants tend to interpret ambiguous situations in a threatening way.

General History. The General History (GH; Appendix J) questionnaire was constructed specifically for the purpose of this study. It assesses use of prescription medications, known triggers of asthma symptoms, involvement in sports or other types of physical exercise, and cigarette smoke exposure. The GH provided information about environmental stimuli that the individual is exposed to on a daily basis.

Panic Attack Cognitions Questionnaire. The Panic Attack Cognitions Questionnaire (PACQ; Clum, Broyles, Borden, & Watkins, 1990; Appendix K) is a 35-item self-report checklist that assesses negative cognitions associated with panic attacks. The PACQ was utilized as a state measure of catastrophic, panic-related cognitions between tasks. The degree of preoccupation with each cognition during a panic attack is rated on a 1 (not at all) to 4 (totally dominated) Likert scale.

The PACQ has been found to reliably differentiate between individuals who experience panic attacks versus individuals diagnosed with other anxiety disorders (Clum et al., 1990). Cronbach's alpha for the PACQ was .88. In addition, the PACQ was found to contribute uniquely to the differentiation between individuals with and without panic attacks (depression, state and trait anxiety measures were also included; Clum et al., 1990).

Profile of Mood States. The Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1971; Appendix L) is a 63-item self-report questionnaire that assesses transient mood states. It was utilized throughout the experiment to detect alterations in mood due to the experimental tasks. The POMS is composed of 6 subscales: Depression-Dejection, Tension-Anxiety, Anger-Hostility, Fatigue-Inertia, Vigor-Activity, and Confusion-Bewilderment. Each item is rated with regard to how much an individual is currently experiencing that emotion on a 0 (not at all) to 4 (extremely) Likert scale. For this study, only the Tension-Anxiety subscale was used. This subscale assesses somatic tension. Internal consistency for this scale is .90, and a high degree of test-retest reliability in a sample of outpatients has been found ( $r = .70$ ; McNair et al., 1971).

State-Trait Anxiety Inventory. The State-Trait Anxiety Inventory (STAI; Spielberger et al., 1982, Appendix M) is a 40-item self-report measure that assesses two aspects of anxiety. Twenty items measure an individual's general or "trait" level of anxiety, and 20 items assess an individual's current or "state" level of anxiety. For the State version, items are rated according to how the individual feels at the moment, from 0 (not at all) to 4 (very much so). The items on the Trait version are rated according to how the individual generally feels (0 = almost never, 4 = almost always). The test-retest reliability for the state version in college students (.16 to .54) is lower than that of the trait version (.73 to .86), as may be expected (Spielberger et al., 1982). In this study, the STAI provided measures of the general and current levels of anxious mood that participants experience. Coefficient alpha in the present study was .80 for the state version and .74 for the trait version.

Symptom Checklist. Because no single measure of both asthma and panic symptoms exists in the published literature, a new measure was devised as a state measure of both types of symptoms. The Symptom Checklist (Appendix N) comprises all the items of the ASC (five subscales) plus a sixth subscale comprised of 14 items from the Panic Attack Symptoms Questionnaire (PASQ; Clum et al., 1990). PASQ items were selected that did not overlap with items already on the ASC. The PASQ itself is a **36-**item checklist of symptoms frequently experienced during a panic attack. It reliably differentiates between individuals with panic attacks and individuals diagnosed with other anxiety disorders (Clum et al., 1990). In addition, the PASQ contributes uniquely to the differentiation between individuals with and without panic attacks (depression, state, and trait anxiety measures were also included; Clum et al., 1990).

Visual Analog Scales. A 7-point Likert scale (Appendix O) was utilized between tasks to assess the similarity of sensations experienced during the task to those experienced during a panic attack. The scale ranges from 0 (not at all similar) to 7 (extremely similar). Because the sensations that individuals with panic disorder experience differ from individual to individual (Barlow & Craske, 1994), some challenge tasks may not be as salient as others. This visual analog scale provided a measure of how similar sensations during the tasks were to those experienced during panic attacks.

A second 7-point Likert scale (Appendix P) assessed how stressful the task was for the participant. The scale ranges from 0 (not at all stressful) to 7 (extremely stressful). Because some researchers have proposed that stress reactivity causes increased air flow resistance (Kotses et al., 1989), it is important to measure the participant's perception of stress level.

A third 7-point Likert scale (Appendix Q) assessed how similar the sensations produced during the tasks were to an asthma attack. The nature of symptoms experienced by individuals with asthma varies from individual to individual (Purcell & Weiss, 1970). Therefore, it was important to utilize a self-report measure of how similar the sensations experienced during the tasks were to an asthma attack.

Participants completed a fourth 7-point Likert scale (Appendix R) that assessed how much control they believed they had over the sensations experienced during each task. According to Ley (1998), dyspnea produces panic sensations when the individual believes he or she has little or no control over the dyspnea.

Measurement of Lung Function. Pulmonary function was assessed via spirometry, using methodology similar to previous studies (e.g., Carr et al., 1992; Carr et

al., 1994; Isenberg et al., 1992b). At each assessment, participants exhaled forcefully 3 times at maximal lung capacity, or until at least 2 equivalent readings ( $\pm 5\%$ ) are obtained. No more than 5 exhalations were attempted at any assessment. A nose clip was utilized to reduce air escaping through the nasal passages. Measures examined included  $FEV_1$  and the ratio of  $FEV_1$  to FVC ( $FEV_1/FVC$ ). Because the level of expected lung capacity differs based on an individual's body height, age, weight, and gender,  $FEV_1$  was analyzed as percent predicted based on the participant's characteristics, calculated by the spirometer.

### Procedure

Participant Selection. Students in numerous Psychology courses completed screening questionnaires that assessed presence or absence of asthma and history of panic attacks. Approximately 1,500 women were screened over a 1.5 year period. Interested participants who met the initial screening criteria for one of the groups were contacted by telephone and asked a series of short questions relating to their medical and psychiatric history. If the responses to these questions indicated that the individual continued to meet criteria, the individual was asked to take part in the structured interview (ADIS-IV). Those who did not meet the inclusion criteria were provided with a brief explanation of why they were not be able to take part in the study (e.g., "We are looking for people who did not experience the onset of both panic and asthma within a year's time.") and thanked for their interest in the experiment. Women who met initial inclusion criteria were scheduled for the experiment during the first 7 days of their menstrual cycle in order to control for hormonal levels that may affect lung function (e.g., Chandler, Shuldheisz, Phillips, & Muse, 1997; Cross, 1994; Settupane & Simon, 1989).

Upon arrival at the laboratory, the experimenter verbally explained the information in the consent form, and participants were asked to read and sign the consent form. Individuals were informed that they would be engaging in tasks not expected to be more stressful than those encountered in daily living, and that they could discontinue participation in the experiment at any time without penalty. After signing the informed consent form, each participant was interviewed utilizing the panic disorder section of the ADIS-IV.

Exclusionary Conditions. In addition to assessment for panic disorder, participants were screened for history of schizophrenia or psychotic episode(s), and alcohol and drug abuse or dependence within the past year. Medical conditions that resulted in exclusion included vestibular disorders and other chronic diseases (i.e., diabetes, hypo or hyperthyroidism, etc.).

Following the structured interview, those who met criteria for participation immediately began the experimental session. Individuals who did not meet criteria were debriefed, thanked for their participation, paid or given course credit, and given a list of treatment referral information.

Questionnaire Assessment. During the experimental session, participants completed the packet of questionnaires and engaged in the experimental tasks. Questionnaires included the ACQ, ASI, respiratory subscale, ASC, BBSIQ, BVS, GH, and STAI, and were presented in random order to minimize the possibility of order effects. Participants were asked to read the instructions for each questionnaire carefully before completing it.



Tasks. Once the questionnaire packet was completed, participants engaged in the three experimental tasks: turning head from side to side, breathing through a straw, and relaxation. To control for order effects, the tasks were counterbalanced. A 5-minute baseline preceded each task. During each baseline, participants sat in a comfortable chair and were told:

“Now we are going to take a short, 5-minute break. During this time, simply sit back and rest. After 5 minutes, we will measure your lung function and you will have a short set of questionnaires to complete.”

After each baseline and after each task, spirometry measures were taken, and each participant completed the anxiety subscale of the POMS, PACQ, PASQ, ASC, Borg Scale, and the visual analog scales.

Head Turning, Task. The head turning task involved the participant turning her head from side to side while seated in a chair for 2 minutes. This task was designed to cause symptoms reminiscent of a panic attack (i.e., dizziness), without affecting lung function or increasing heart rate (Barlow & Craske, 1994). It is particularly important to avoid activity that affects heart rate, because heart rate is related to respiratory rate (Lehrer, Hochron, Rausch, & Carr, 1994a). If heart rate is inadvertently increased, then respiratory rate also increases.

Participants were seated in a chair and told:

“During this task, please turn your head from side to side (experimenter demonstrates). This task will last 2 minutes. Your task is to focus on your thoughts, feelings, and the sensations you experience while turning your head. After the minute is up, I will measure your lung function, and have you will complete the short questionnaire

packet again. If it becomes absolutely necessary for you to stop this activity before the 2 minutes are up, that is okay, but please do your best to continue for as long as you can.”

To avoid the loss of participants due to possible inability to complete 2 full minutes of the head turning task, the length of time the participant continued was recorded. Data for participants completing at least 30 seconds of the task was retained.

Straw Task. Breathing through a straw lasted 2 minutes. This task was designed to mimic the sensations of dyspnea experienced during an asthma attack (Barlow & Craske, 1994).

Participants were seated in a comfortable chair and a nose clip was placed on their nose. They were told:

“In this task, you will be breathing through this straw for 2 minutes. Your task is to focus on your thoughts, feelings, and the sensations you experience while breathing through the straw. Again, after this task, I will measure your lung function and ask you to complete the short questionnaire packet. If it becomes absolutely necessary for you to stop this activity before the 2 minutes are up, that is okay, but please do your best to continue for as long as you can.”

Again, to reduce participant attrition, the length of time participants continued was recorded. Data for participants who continued the straw task for at least 30 seconds was retained.

Relaxation Task. Relaxation was 5 minutes in length. This task served as a comparison and also replicated the findings of Carr et al. (1992).

Participants were seated in a comfortable chair and told:

“During this task, please relax as completely as possible with your eyes closed. You will have 5 minutes to simply sit back and relax. After the task, I will measure your lung function and have you complete the short questionnaire packet.”

Spirometry Procedure. Instructions for the participants regarding how their lung function was measured were modeled after American Thoracic Society guidelines (ATS, 1995). Participants were shown the equipment and the disposable mouthpiece they exhaled into. The following instructions were provided:

“This is a spirometer. It is a machine that measures how well your lungs are working. You will be exhaling into this mouthpiece (show mouthpiece). I ~~am~~ going to put this nose clip on your nose in order to prevent air from escaping. It is very important that you inhale completely before you put the mouthpiece in your mouth. If you inhale into the mouthpiece, this will alter the readings. When you put the mouthpiece in, make sure you have made a good seal with your lips to prevent air from escaping on the sides (experimenter demonstrates with another mouthpiece). When you exhale, it is important that you blast the air from your lungs and then continue exhaling as fully as possible (experimenter demonstrates). We will be doing at least 3 exhalations after each task. Do you have any questions?”

The experimenter demonstrated the procedure and, as the participant exhaled, encourage her to exhale as fully as possible. At least 3 acceptable ( $<0.2$  L variation; ATS, 1995) maneuvers were necessary. No more than 5 total maneuvers were attempted at any task interval.

Debriefing. After the completion of the series of tasks, lung function assessments, and questionnaire measures, participants were thanked for their participation and debriefed. Participants not receiving experimental credit were paid \$10.00 to assist in defraying travel costs associated with their participation in the study.

Modifications to Procedure. Because of the difficulty in obtaining participants, two of the constraints that had originally been placed on participation had to be abandoned. It was hoped that women would be able to be scheduled during the first week of their menstrual cycle in order to hold hormonal factors constant. It is well-established that hormone fluctuations during the menstrual cycle may lead to exacerbations for some women during the premenstrual phase (e.g., Chandler et al., 1997; Cross, 1994; Setticone & Simon, 1989). Furthermore, the menstrual cycle appears to impact frequency and severity of panic attacks (e.g., Cameron, Kuttlesch, McPhee, & Curtis, 1988; Cook et al., 1990; Kaspi, Otto, Pollack, Eppinger, & Rosenbaum, 1994; Sigmon et al., 2000). However, potential participants were less likely to follow through with scheduled assessments when planned dates were more than a few weeks away. Instead, women indicated which phase of the menstrual cycle they were in and that information was recorded as part of data collection.

Several women in each of the groups smoked cigarettes on a regular basis. Previous research in the asthma and panic area has excluded individuals who smoke in order to rule out reasons for lung dysfunction other than asthma (e.g., Carr et al., 1992; Carr et al., 1996). If the study had been conducted with a larger sample size, it is possible that a more homogeneous sample without confounds (i.e., smoking status, menstrual cycle phase) might have been obtained.

## Chapter 3

### RESULTS

In this section, participant characteristics, interrater reliability statistics for diagnostic interviews using the panic section of the Anxiety Disorders Interview for DSM-IV (ADIS-IV; Brown et al., 1994), and results for each hypothesis are presented. Additional related findings are included in the results for each hypothesis as well as at the end of the Results section. All data analyses were conducted with SPSS version 10.0 software for Windows. Where possible, power and effect size ( $\eta^2$ ) were calculated for each analysis. According to Cohen (1988), effect sizes of .10, .25, and .40 are considered to be small, medium, and large, respectively. Post hoc comparisons were conducted using Tukey's HSD test with alpha set at .05.

#### Sample Characteristics

##### Demographic Information

Female participants were 93% Caucasian, 3% Hispanic, and 3% Native American. All but one participant was an undergraduate at the University of Maine (99%). The four groups of participants (Asthma only, Panic only, Asthma and Panic, Control) did not significantly differ in age,  $F(3,55) = .43$ , *ns*. The mean age of participants by group was as follows: Asthma only,  $M = 19.47$ ,  $SD = 1.81$ ; Asthma and Panic,  $M = 22.80$ ,  $SD = 9.24$ ; Panic,  $M = 20.93$ ,  $SD = 3.08$ ; Control,  $M = 21.07$ ,  $SD = 4.67$ .

##### Asthma Groups

Women in the Asthma only and Asthma and Panic groups were compared on several variables that related to their asthma condition. Women in the Asthma only group were first diagnosed with the disease at a mean age of 11.38 years ( $SD = 5.84$ ), whereas

women in the Asthma and Panic group were diagnosed at a mean age of 16.83 years ( $SD = 12.59$ ). However, these means were not significantly different  $F(1,23) = .56$ , ns.

Women in the two asthma groups did not significantly differ with regard to the number of medications taken. Women in the Asthma group reported taking an average of 1.8 medications, and women in the Asthma and Panic group reported taking 3.33 medications,  $F(1,28) = 2.48$ , ns. Types of medications taken by both groups were similar, most frequently beta agonists (60% of Asthma group, 47% of Asthma and Panic group), followed by corticosteroids (13% of Asthma group, 13% of Asthma and Panic group) and allergy medications (13% of Asthma group, 20% of Asthma and Panic group). Similarly, there was no significant difference in length of time from last asthma attack in weeks between the Asthma ( $M = 46.38$ ,  $SD = 71.53$ ) and Asthma and Panic groups ( $M = 32.87$ ,  $SD = 83.66$ ),  $F(1,26) = .00$ , ns.

Several  $t$  tests were performed on the subscales of the Asthma Symptom Checklist (ASC) comparing the responses of women in the Asthma only and Asthma and Panic groups (see Table 3.1). Women in the Asthma and Panic group reported higher levels of asthma symptoms than the Asthma only group on total scale score,  $t(1,28) = -2.67$ ,  $p < .01$ , the Panic Fear  $t(1,28) = -2.96$ ,  $p < .006$ , Irritability,  $t(1,28) = -2.41$ ,  $p < .02$ , and Fatigue  $t(1,28) = -2.17$ ,  $p < .03$  subscales. Means for the Hyperventilation  $t(1,28) = -.58$ , ns, and Bronchoconstriction  $t(1,28) = -1.74$ , ns, subscales were not significantly different between the Asthma only and Asthma and Panic groups.

### Panic Groups

Women in the Asthma and Panic and Panic only groups were compared on panic-related variables assessed on the Anxiety Disorders Interview Schedule for DSM-IV

Table 3.1

Means and Standard Deviations for Asthma Symptom Checklist Subscales

	Asthma	Asthma & Panic	
Measure	<u>M (SD)</u>	<u>M (SD)</u>	<u>t (1,28)</u>
Total Score	98.26 (27.57)	122.78 (18.95)	-2.67*
Panic Fear	19.06 (8.08)	27.20 (6.87)	-2.96**
Irritability	14.00 (5.50)	19.00 (5.85)	-2.41*
Fatigue	11.53 (4.89)	15.28 (4.32)	-2.17*
Hyperventilation	20.60 (6.25)	21.73 (4.25)	-.58
Bronchoconstriction	33.06 (8.97)	38.00 (7.43)	-1.74

\*  $p < .05$ . \*\*  $p < .01$ .

(Brown et al., 1994). Both groups reported having experienced panic attacks for a similar length of time (Asthma only:  $\underline{M} = 78.93$  months,  $\underline{SD} = 54.43$ ; Asthma and Panic:  $\underline{M} = 68.40$  months,  $\underline{SD} = 65.00$ ),  $\underline{F}(1,27) = .538$ , ns. Women in the Panic only group reported an average of 7.33 panic attacks ( $\underline{SD} = 9.63$ ) over the past month, and 36.73 attacks ( $\underline{SD} = 59.84$ ) over the past 6 months. These values were not significantly different from those reported by the Asthma and Panic group, who reported an average of 7.93 attacks ( $\underline{SD} = 7.90$ ) in the past month,  $\underline{F}(1,28) = .001$ , ns, and 39.00 attacks ( $\underline{SD} = 43.09$ ) over the past 6 months,  $\underline{F}(1,28) = .32$ , ns. When asked to rate the amount they worried about having another panic attack during the past month on a 0 (no worry/apprehension) to 8 (constantly worried/extreme apprehension) Likert scale, the average response of the women in the Panic only group was 4.60 ( $\underline{SD} = 2.56$ ), whereas women in the Asthma and Panic group reported less worry, 3.73 ( $\underline{SD} = 2.60$ ). These means, however, were not significantly different,  $\underline{F}(1,28) = .001$ , ns. Similarly, no significant differences were found between the Panic only and Asthma and Panic groups on measures of panic attack life interference and distress,  $\underline{F}(1,28) = 2.89$ , ns, and  $\underline{F}(1,28) = .89$ , ns, respectively.

An advanced clinical graduate student reviewed audiotapes of the ADIS-IV interviews and rated which group criteria the participant met. These ratings were compared with the principal investigator. Unfortunately, 13 out of 60 interviews were not recorded due to equipment failure. On the remaining 47 participant interviews, the principal investigator and the advanced clinical graduate student agreed 100% that women in the Panic only and Asthma and Panic groups met DSM-IV criteria for Panic Disorder and that women in the Asthma and Control groups did not meet criteria for Panic Disorder.



### Questionnaire Measures

Oneway ANOVAs were performed on total scores for the following questionnaire measures. Means and standard deviations for these measures appear in Table 3.2.

On the State version of the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1983), no significant differences between groups were found,  $F(3, 56) = 1.68$ , *ns*. On the Trait version, however, a significant effect for group was found,  $F(3, 56) = 9.71$ ,  $p < .0001$  (see Figure 3.1). Women in the Panic only group reported significantly more trait anxiety than women in the Asthma only and Control groups, and women in the Asthma and Panic group reported more trait anxiety than the Asthma only group. Mean scores for the Asthma only and Control groups did not significantly differ, nor did the Asthma and Panic and Panic only groups.

### Profile of Mood States

A oneway ANOVA was conducted on baseline Profile of Mood States (POMS; McNair et al., 1971) Tension-Anxiety subscale scores to identify group differences,  $F(3, 56) = 5.41$ ,  $p < .002$  (see Table 3.3). Post hoc analyses revealed that the Panic only group reported significantly more anxiety at baseline than the Asthma only and Control groups. The Asthma and Panic group did not differ from any other group, and the Asthma only group did not differ from the Control group. To calculate POMS scores for each task taking into account baseline differences, change scores were derived for each task from baseline. A MANOVA on POMS change scores from all three tasks revealed a significant overall effect for group, Wilks' lambda  $F(9, 129) = 2.58$ ,  $p < .009$ , power = .85,  $\eta^2 = .12$ . Followup oneway ANOVAs and post hoc tests indicated that on the head turning task,  $F(3, 55) = 5.67$ ,  $p < .002$ , women in the Asthma and Panic group reported

Table 3.2

Means and Standard Deviations for Questionnaires

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u> (3,52)
State Anxiety	48.60 (4.13)	52.66 (8.53)	53.20 (8.94)	49.26 (4.05)	.18
Trait Anxiety	47.60 <sub>b,c</sub> (3.73)	55.26 <sub>a</sub> (6.78)	57.06 <sub>a,d</sub> (5.90)	50.93, (4.21)	9.71***

Note: Groups are significantly different from means subscripted.

\*\*\*  $p < .0001$ .

Figure 3.1. Mean trait anxiety score by group

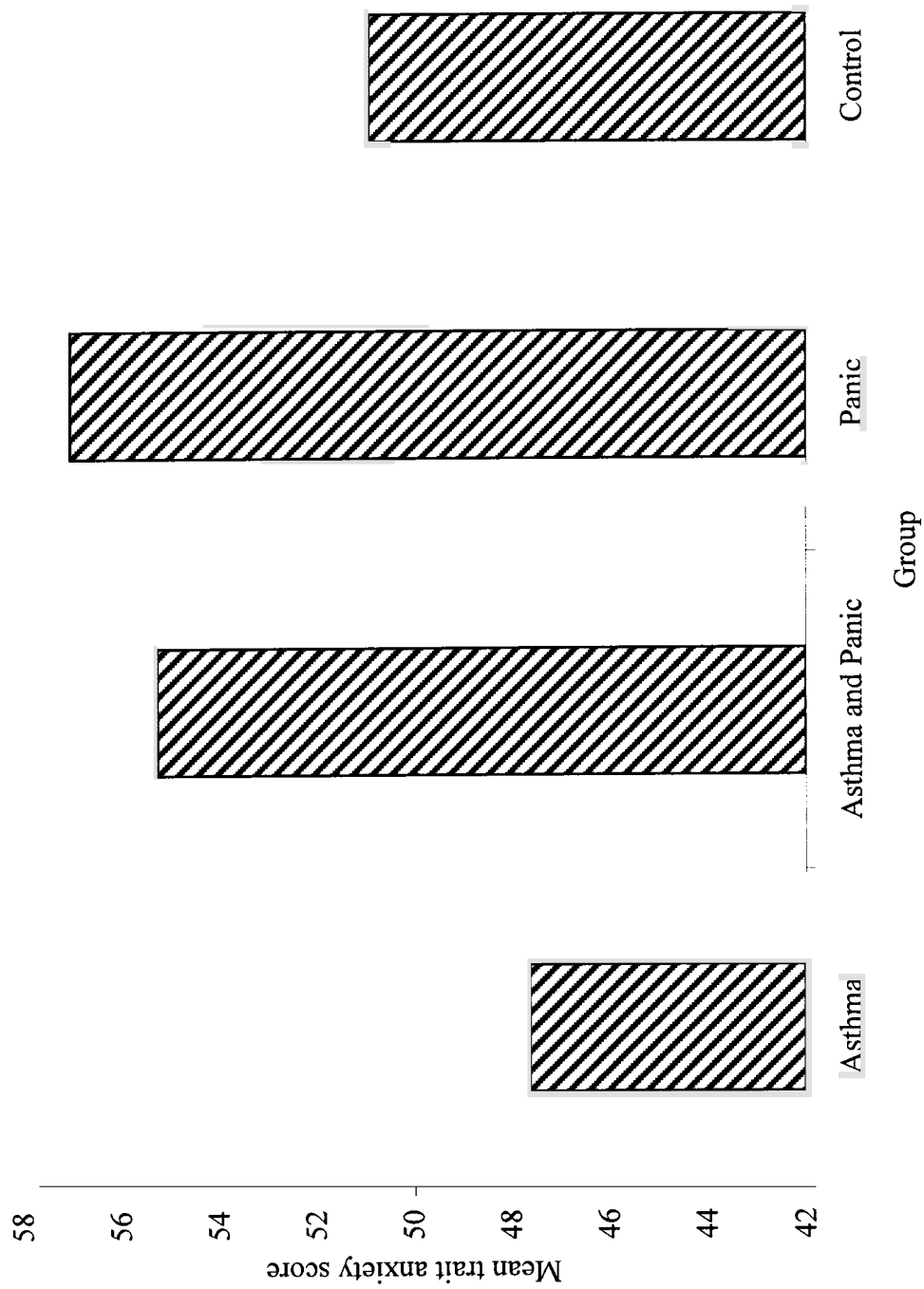


Table 3.3

Means and Standard Deviations for Profile of Mood States Tension-Anxiety Scores

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u>
Baseline	5.26, (3.41)	9.73 (7.73)	14.46 <sub>a,d</sub> (10.90)	4.93, (5.70)	<u>F</u> (3,56) = 5.41**
Head Turning	6.26 <sub>b</sub> (4.74)	17.13 <sub>a,c,d</sub> (9.53)	17.06 <sub>b</sub> (11.48)	5.80 <sub>b</sub> (5.26)	<u>F</u> (3,55) = 5.67**
Change	-1.00	-7.40	-2.60	-.87	
Straw	7.46 (6.73)	16.86 <sub>d</sub> (9.01)	17.93 (11.91)	5.86 <sub>b</sub> (5.04)	<u>F</u> (3,55) = 2.62
Change	-2.20	-7.13	-3.47	-.93	
Relaxation	3.33 (3.15)	6.06 (5.50)	11.66 (10.93)	3.21 (4.67)	<u>F</u> (3,55) = .31
Change	+1.93	+3.67	+2.80	+1.72	

Note. Negative change scores reflect intensification of anxiety relative to baseline; positive change scores reflect decrease in anxious

mood relative to baseline. Groups are significantly different from means subscripted.

\*\*  $p < .01$ .

more anxiety compared to baseline than the other three groups (see Figure 3.2). Women in the Asthma only group did not score differently from women in the Panic only or Control groups, and the Panic only and Control groups did not differ on their self-report of anxious mood. On the straw task,  $F(3,55) = 2.62, p < .06$ , a trend toward significance was identified. The Asthma and Panic group exhibited a larger increase in anxiety during the straw task than the other three groups, although this was not statistically significant. Finally, no significant mood changes were identified for the relaxation task,  $F(3,55) = .31, ns$ .

### Results for Hypotheses

#### Hypothesis One

According to Hypothesis One, in comparison to baseline scores, individuals in the Asthma only, Panic only, and Asthma and Panic groups would experience a greater increase in dyspnea level after the relaxation and head turning tasks than the Control group. Controls were expected to experience greater dyspnea after the straw task. Baseline Borg Dyspnea Scale (BDS; Borg, 1982) scores were compared via a oneway ANOVA (see Table 3.4). No baseline group differences were found,  $F(3,56) = 2.03, ns$ . In order to compare BDS scores after each task to baseline, change scores were calculated for each task. BDS scores from the head turning, straw, and relaxation tasks were subtracted from baseline scores. A oneway ANOVA revealed no significant differences between groups on BDS change scores following the straw,  $F(3,56) = 1.66, ns$ , or relaxation tasks,  $F(3,56) = .44, ns$ . Group differences on the head turning task, however, approached significance,  $F(3,56) = 2.57, p < .06$ . Post hoc analyses, however, did not identify significant differences between any pairs of groups. During the head turning

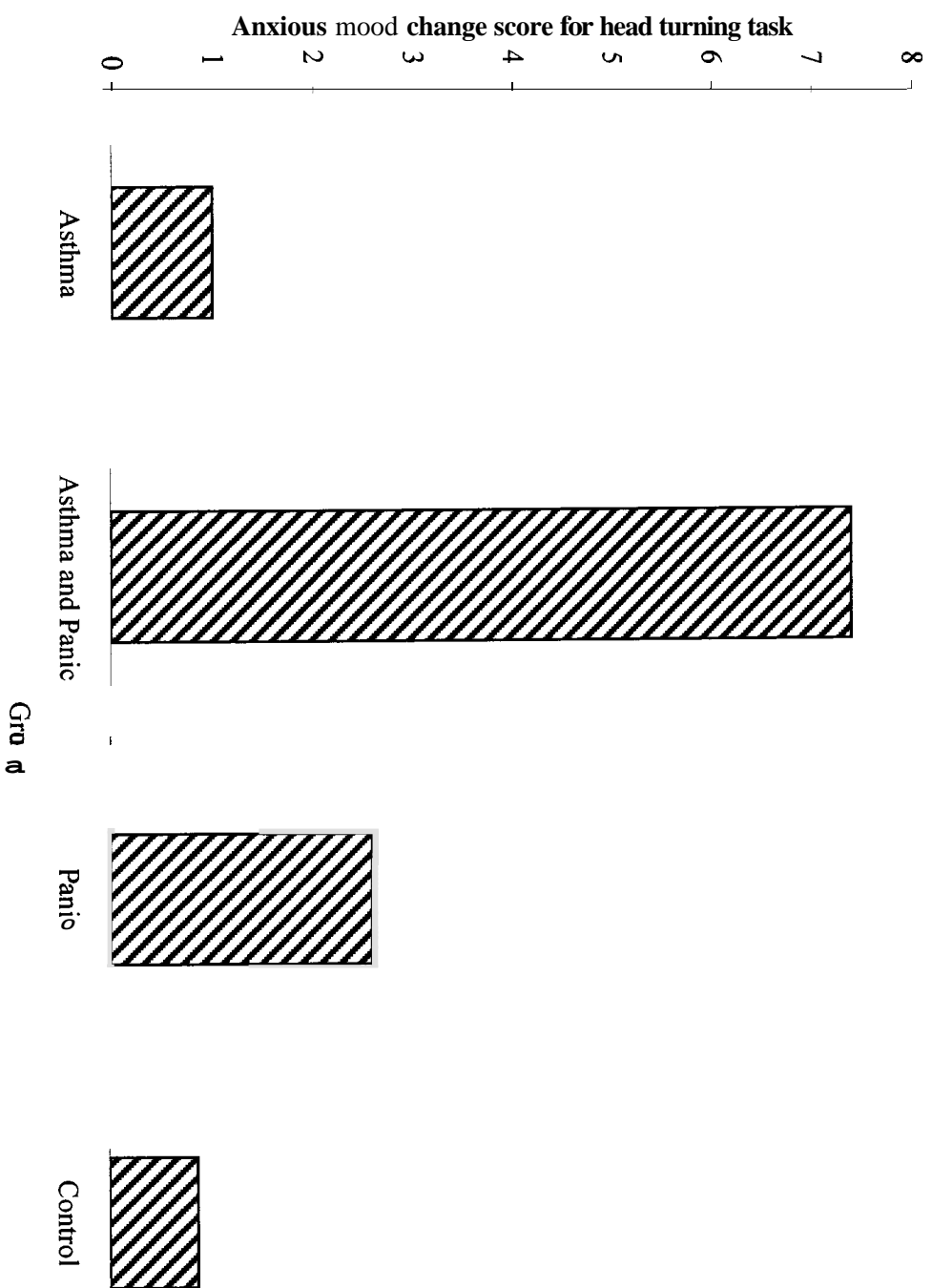


Figure 3.2. Anxious mood change scores for head turning task

Table 3.4

Means and Standard Deviations for Borg Dyspnea Scale Scores

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u> (3,55)
Baseline	1.67 (1.23)	1.87 (1.67)	1.53 (1.25)	.80 (1.26)	2.03
Head Turning	1.87 (1.19)	2.80 (1.37)	2.27 (1.75)	.47 (.52)	2.57
Change	-.20	-.93	-.74	-.33	
Straw	2.53 (1.19)	4.00 (1.13)	3.40 (2.56)	2.13 (1.64)	1.66
Change	-.86	-2.13	-1.87	-1.33	
Relaxation	1.13 (1.19)	1.13 (1.19)	.87 (1.19)	.14 (.36)	.44
Change	-.54	-.74	+.66	+.66	

Note. Negative change scores reflect intensification of dyspnea relative to baseline; positive change scores reflect decrease in dyspnea relative to baseline. Groups are significantly different from means subscripted.

task, women in the Asthma and Panic and Panic only groups reported more anxiety than women in the Asthma and Control groups.

### Hypothesis Two

According to Hypothesis Two, after the relaxation task, the Panic only and Asthma and Panic groups would report a greater increase in panic-related symptoms from baseline than the Asthma only group. In turn, the Asthma only group was expected to report a greater increase in these symptoms than the Control group. After the head turning task, the Panic only and Asthma and Panic groups were expected to report a greater increase in panic sensations than the Asthma only and Control groups. After the straw task, panic-related symptoms were expected to be equivalent for all groups.

Baseline scores from the six subscales of the Symptom Checklist (five subscales of the ASC, plus panic specific symptoms) were analyzed via ANOVA. Five out of the six subscales resulted in a significant oneway ANOVA and significant group differences with post hoc testing (see Table 3.5). On the Panic-Fear,  $F(3, 56) = 4.09, p < .01$ , Fatigue,  $F(3, 56) = 4.38, p < .008$ , and Hyperventilation subscales,  $F(3, 56) = 4.77, p < .005$ , an identical pattern of scores was identified. For these three subscales, women in the Panic only group scored higher than women in the Asthma only and Control groups. The Asthma and Panic group did not significantly differ from any other group. On the Irritability subscale,  $F(3, 56) = 4.65, p < .006$ , women in the Panic only group scored significantly higher than women in the Asthma only group. No differences were found for the Asthma and Panic or Control groups. On the Bronchoconstriction subscale,  $F(3, 56) = 1.92, ns$ , no significant group differences were identified. Finally, on the Panic-Specific subscale, women in the Panic only group scored higher than women in the



Table 3.5

Means and Standard Deviations for Baseline Symptom Checklist Subscales

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u> (3,56)
Panic-Fear	.73 <sub>c</sub> (1.09)	3.53 (5.15)	5.53 <sub>a,d</sub> (6.10)	1.27, (4.54)	4.09**
Irritability	1.06, (.79)	3.60 (2.94)	5.20 <sub>a</sub> (5.14)	2.06 (2.52)	4.65**
Fatigue	2.26, (3.01)	3.26 (3.63)	5.80 <sub>a,d</sub> (4.58)	1.33, (2.71)	4.38**
Hyperventilation	1.73, (2.12)	1.86 (2.94)	4.53 <sub>a,d</sub> (4.08)	.80 <sub>c</sub> (1.56)	4.77**
Bronchoconstriction	6.00 (5.27)	5.13 (5.89)	7.06 (6.70)	2.40 (4.11)	1.92
Panic Symptoms	2.26 (1.75)	5.60 (7.51)	7.40 <sub>d</sub> (7.51)	2.00, (2.07)	3.45*

Note: Groups are significantly different from means subscripted.

\*  $p < .05$ . \*\*  $p < .01$ .

Control group. No significant differences were identified for the Asthma and Panic or Asthma only groups.

In order to compare Symptom Checklist subscale scores while taking into account baseline differences, change scores for each task were calculated (see Table 3.6). For the head turning task, a MANOVA on the six subscale scores did not reveal any significant group differences, Wilks' lambda  $F(18,144) = 1.39$ , ns, power = .82, effect size = .13. Likewise, the overall MANOVA on the relaxation task was nonsignificant, Wilks' lambda  $F(18,144) = 1.81$ , ns, power = .93, effect size = .17. No followup ANOVAs were conducted for these two tasks. On the straw task, however, a significant Wilks' lambda was revealed,  $F(18,144) = 2.76$ ,  $p < .0001$ , power = .99, effect size = .24. Followup oneway ANOVAs were significant for two of the six subscales, Hyperventilation,  $F(3,56) = 5.26$ ,  $p < .003$ , and Bronchoconstriction,  $F(3,56) = 3.17$ ,  $p < .03$ . Post hoc testing for the Hyperventilation subscale revealed that the Panic only group scored significantly higher than the Asthma only and Control groups. The Asthma and Panic group did not differ from any other group. On the Bronchoconstriction subscale, women in the Asthma and Panic group scored higher than the Asthma only group. No significant differences were found for the Panic only or Control groups. Post hoc tests on the remaining subscale scores, Panic-Fear,  $F(3,56) = 1.07$ , ns, Irritability,  $F(3,56) = 1.58$ , ns, Fatigue,  $F(3,56) = .87$ , ns, and Panic Specific,  $F(3,56) = 2.10$ , ns, revealed no significant findings.

Hypothesis Three

According to Hypothesis Three, individuals in the Asthma and Panic group were expected to experience an increase in lung function after the relaxation and head turning tasks in comparison to baseline. In contrast, the Control group was expected to exhibit no

Table 3.6

Means and Standard Deviations for Task Symptom Checklist Subscales

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u> (3,56)
Head Turning Task					
Panic-Fear	.80 (2.07)	4.60 (5.66)	7.93 (7.88)	.40 (.91)	1.83
Change	-.06	-1.06	+.86	-.66	
Irritability	1.00 (1.41)	5.66 (4.46)	6.80 (6.23)	3.00 (3.09)	2.00
Change	-.06	-2.06	-1.60	-.93	
Fatigue	2.00 (2.44)	4.60 (4.89)	8.20 (5.65)	1.60 (2.50)	1.83
Change	+.26	-1.33	-2.40	-.26	
Hyperventilation	3.46, (2.53)	7.13 (5.39)	11.26 <sub>a,d</sub> (8.25)	2.73, (3.71)	4.76**
Change	-1.73	-5.26	-6.73	-1.93	

Table 3.6 Continued

Measure	Asthma (a)		Asthma & Panic (b)		Panic (c)		Control (d)	
	<u>M</u>	( <u>SD</u> )	<u>M</u>	( <u>SD</u> )	<u>M</u>	( <u>SD</u> )	<u>M</u>	( <u>SD</u> )
Head Turning Task								
Bronchoconstriction	4.93	(4.63)	8.60	(7.93)	9.13	(9.45)	.66	(.33) 3.25*
Change	+1.06		-3.46		-2.06		+1.73	
Panic Symptoms	4.60	(5.26)	10.73	(9.45)	14.66	(11.77)	3.26	(2.11) 3.17 *
Change	-2.33		-5.13		-7.26		-1.26	
Straw Task								
Panic-Fear	1.46	(2.85)	6.26	(5.17)	8.00	(9.00)	1.00	(1.64) 1.07
Change	-.73		-2.73		-2.46		+.26	
Irritability	1.33	(1.98)	5.53	(3.13)	7.66	(6.07)	3.13	(2.82) 1.58
Change	-.26		-1.93		-2.46		-1.06	

Table 3.6 Continued

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u> (3,56)
Straw Task					
Fatigue	2.13 (2.32)	4.86 (4.32)	7.00 (5.43)	2.00 (2.39)	.87
Change	+.13	-1.60	-1.20	-.66	
Hyperventilation	3.06, (1.98)	5.00 (3.56)	10.33 <sub>a,d</sub> (8.38)	1.40, (3.26)	5.26**
Change	-1.33	-3.13	-5.80	-.60	
Bronchoconstriction	8.20 <sub>b</sub> (5.33)	13.93, (6.47)	5.93 (3.30)	10.25 (8.05)	3.17*
Change	-2.20	-8.80	-5.86	-3.53	
Panic Symptoms	4.60 (4.17)	10.66 (6.84)	14.33 (13.81)	3.40 (3.13)	2.10
Change	-2.33	-5.06	-6.93	-1.40	

Table 3.6 Continued

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u> (3,56)
Relaxation Task					
Panic-Fear	.33 (.89)	1.13 (1.68)	4.66 (6.91)	.35 (.92)	.62
Change	+.40	+2.40	+.86	+1.00	
Irritability	.73 (1.27)	2.06 (2.52)	4.73 (6.50)	1.64 (3.27)	.61
Change	+.33	+1.53	+.46	+.57	
Fatigue	1.93 (2.49)	4.20 (3.83)	5.73 (5.50)	2.42 (2.56)	.55
Change	+.33	-.93	+.06	-1.00	
Hyperventilation	1.26 (1.38)	1.40 (1.68)	6.73 (7.31)	.42 (1.34)	2.00
Change	+.46	+.46	-2.20	+.42	

Table 3.6 Continued

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u> (3,56)
Relaxation Task					
Bronchoconstriction	2.80 (2.95)	3.40 (5.67)	6.20 (11.30)	.64 (1.15)	.77
Change	+3.20	+1.73	+.86	+.78	
Panic Symptoms	1.80 (2.04)	4.13 (3.64)	9.06 (12.31)	4.10 (7.17)	.75
Change	+.46	+1.46	-1.66	+.85	

Note. Negative change scores reflect intensification of symptoms relative to baseline; positive change scores reflect decrease in symptoms relative to baseline. Groups are significantly different from means subscripted.

$p < .05$ . \*\*  $p < .01$ .

change in lung function. The Asthma only and Panic only groups were expected to exhibit a decrease in lung function after these tasks. Following the straw task, it was expected that the Asthma only and Asthma and Panic groups would both show an increase in lung function. No change was expected for the Control group, and a decrease was expected for the Panic only group.

The spirometric equipment calculates the degree to which the three exhalation trials are consistent with each other, and selects the “best” exhalation (i.e., values as large as possible, but also consistent with the other two exhalations) as the one to use for interpretation. The accepted level of reliability is less than 0.5 L variation between the three trials. If no two of the three trials are within 0.5 L of each other, additional measurements are made (Hyatt, Scanlon, & Nakamura, 1997). Because some of the participants were not able to provide spirometric values that were reliable even after four or five exhalations, a number of readings were not included in analyses. The pattern of unreliable spirometric performance is in and of itself interesting. The Asthma only group’s percentage of unreliable measurements was low compared to that of the other groups (12%), Asthma and Panic group 30%, Panic only group 29%, and the Control group 16%. In order to use as much data as possible, ANCOVA was utilized with the spirometric percent variance as a covariate. In addition, eliminating data that fell below the 5% variance cutoff would have decreased the cell sizes for the Asthma and Panic and Panic only groups in a disproportional manner.

An ANCOVA on baseline forced vital capacity (FVC) percent predicted data using percent variance as a covariate revealed no significant group differences,  $F(3,55) = .84$ , ns. A similar analysis on baseline forced expiratory volume at 1 second ( $FEV_1$ )



percent predicted data was also nonsignificant,  $F(3,55) = 1.24, \underline{ns}$ . Because of the lack of significant baseline differences between groups on any of these measures, change scores were not calculated. Rather, each measurement was analyzed via ANCOVA with the percent variance used as a covariate (see Table 3.7).

No significant differences were identified for any spirometric measure following any task. ANCOVAs for FVC,  $F(3,55) = .90, ns$ , FEV<sub>1</sub>,  $F(3,53) = .91, \underline{ns}$ , and an ANOVA on the ratio of FEV<sub>1</sub>/FVC,  $F(3,55) = .89, \underline{ns}$ , for the head turning task failed to reach significance. Likewise, for the straw task, no significant differences were identified via ANCOVAs on FVC,  $F(3,55) = 1.95, \underline{ns}$ , FEV<sub>1</sub>,  $F(3,55) = 1.63, \underline{ns}$ , or an ANOVA on FEV<sub>1</sub>/FVC,  $F(3,56) = 1.80, \underline{ns}$ . Relaxation task results were similar, with an ANCOVA on FVC,  $F(3,54) = .60, ns$ , and FEV<sub>1</sub>,  $F(3,54) = 1.28, \underline{ns}$ , resulting in nonsignificance. Likewise, an ANOVA on relaxation FEV<sub>1</sub>/FVC,  $F(3,55) = 1.38, ns$ , was not significant.

#### Hypothesis Four

After the relaxation and head turning tasks, individuals with Panic Disorder and both Asthma and Panic were expected to report an increase in panic-related cognitions in comparison to the Asthma only and Control groups. After the straw task, the Asthma only and Asthma and Panic groups were expected to report a greater increase in panic-related cognitions than the Panic only group, with the Control group reporting no increase.

Total scores for the Panic Attack Cognitions Questionnaire (PACQ; Clum et al., 1990) were compared at baseline (see Table 3.8). Results of an ANOVA on the PACQ scores revealed a significant group effect,  $F(3,56) = 6.24, p < .001$ . Post hoc tests indicated that women in the Panic only group reported significantly more panic related

Table 3.7

Means and Standard Deviations for Spirometry

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u>
Forced Vital Capacity					
Baseline	93.40 (9.27)	91.06 (10.91)	94.06 (14.58)	98.33 (16.64)	$\underline{F}(3,55) = .84$
Head Turning	94.93 (7.90)	89.20 (16.98)	92.06 (13.64)	97.00 (17.79)	$\underline{F}(3,55) = .90$
Straw	93.06 (10.61)	86.00 (12.29)	92.86 (11.64)	97.66 (17.22)	$\underline{F}(3,55) = 1.95$
Relaxation	93.86 (10.39)	90.20 (10.21)	94.40 (14.84)	97.57 (17.24)	$\underline{F}(3,54) = .60$
Forced Expiratory Volume at 1 second					
Baseline	97.00 (9.77)	90.33 (19.42)	92.93 (13.16)	100.26 (16.64)	$\underline{F}(3,55) = 1.24$
Head Turning	99.14 (7.24)	88.86 (18.69)	90.46 (17.49)	97.46 (17.77)	$\underline{F}(3,53) = .91$
Straw	95.93 (10.29)	86.86 (18.50)	86.86 (14.59)	96.80 (19.45)	$\underline{F}(3,55) = 1.63$

Table 3.7 Continued

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u>
Forced Expiratory Volume at 1 second					
Relaxation	95.66 (10.44)	87.73 (17.60)	89.40 (16.72)	98.57 (19.63)	$\underline{F}(3,54) = 1.28$
FEV 1/FVC					
Baseline	.92 (.05)	1.13 (.88)	.87 (.07)	.90 (.05)	$\underline{E}(3,56) = .38$
Head Turning	.92 (.04)	.88 (.15)	.86 (.09)	.89 (.05)	$\underline{E}(3,55) = .89$
Straw	.91 (.02)	.88 (.13)	.83 (.12)	.88 (.09)	$\underline{E}(3,56) = 1.80$
Relaxation	.91 (.06)	.85 (.15)	.84 (.11)	.90 (.07)	$\underline{E}(3,55) = 1.38$

Table 3.8

Means and Standard Deviations for Panic Attack Cognitions Questionnaire Scores

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u>
Baseline	25.73, (1.27)	28.86 (4.06)	25.86 <sub>a,d</sub> (1.80)	26.26, (1.80)	$\underline{F}(3,56) = 6.24^{***}$
Head Turning	25.33 (7.57)	32.13 (5.12)	36.86 (12.27)	26.26 (1.16)	$\underline{F}(3,55) = 2.29$
Change	+.40	-3.20	-11.00	0.00	
Straw	27.13 (3.75)	32.33 (5.89)	37.86 (17.04)	26.26 (0.96)	$\underline{F}(3,55) = .93$
Change	-1.40	-3.47	-.12	0.00	
Relaxation	25.40 (0.73)	27.13 (2.32)	32.13 (10.34)	25.42 (1.15)	$\underline{F}(3,55) = .24$
Change	+.33	+1.73	-6.27	+.84	

Note. Negative change scores reflect intensification of symptoms relative to baseline; positive change scores reflect decrease in

symptoms relative to baseline. Groups are significantly different from means subscripted.

\*\*\*  $p < .001$ .

thoughts than women in the Asthma only and Control groups. Interestingly, although the PACQ mean for the Asthma and Panic group appeared to be higher than the Panic only group mean, it was not significantly different at the .05 level. No significant baseline differences were found for the Asthma and Panic group.

Change scores were calculated for each task in comparison to baseline. A MANOVA on change scores revealed an overall significant Wilks' lambda,  $F(9,129) = 2.32, p < .01$ , power = .80,  $\eta^2 = .11$ . Oneway ANOVAs revealed no significant differences on the straw  $F(3,55) = .93$ , ns, power = .24,  $\eta^2 = .04$ , or relaxation tasks  $F(3, 55) = .24$ , ns, power = .09,  $\eta^2 = .01$ . However, on the head turning task, a trend toward significance was found,  $F(3,55) = 2.29, p < .08$ , power = .54,  $\eta^2 = .11$ . Although post hoc tests did not identify any group differences for the head turning task, visual inspection of the PACQ change scores for this task suggests that women in the Asthma and Panic and Panic only groups reported more panic-related thoughts than women in the Asthma only and Control groups.

#### Hypothesis Five

Women in the Asthma only, Panic only, and Asthma and Panic groups were expected to report more self-focus on bodily sensations than women in the Control group. Analysis of the total scores on the Body Vigilance Scale (Schmidt et al., 1997; see Table 3.9) revealed significant group differences,  $F(3,56) = 10.08, p < .0001$ . On this measure, women in the Asthma and Panic and Panic only groups reported more bodily self-focus than women in the Asthma and Control groups.

Table 3.9

Means and Standard Deviations for Anxiety Sensitivity Measures

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u> (3,56)
Anxiety Sensitivity Index	16.86 <sub>b,c</sub> (6.67)	33.06 <sub>a,d</sub> (11.39)	30.26 <sub>a,d</sub> (15.62)	15.53 <sub>b,c</sub> (6.64)	10.55***
Body Vigilance Scale	15.54 <sub>b,c</sub> (5.93)	23.63 <sub>a,d</sub> (9.76)	23.53 <sub>a,d</sub> (6.95)	11.40 <sub>b,c</sub> (6.36)	10.08***
Anxiety Sensitivity Profile					
Cardiovascular	26.00 <sub>b</sub> (7.74)	39.93 <sub>a,d</sub> (13.36)	35.53 (13.88)	25.60 <sub>b</sub> (9.30)	5.87***
Respiratory	25.20 <sub>b</sub> (9.25)	41.00 <sub>a,d</sub> (14.81)	37.93 <sub>d</sub> (16.00)	23.60 <sub>b,c</sub> (11.72)	6.66***
Gastrointestinal	26.93 <sub>b</sub> (8.37)	39.33 <sub>a,d</sub> (12.65)	35.20 (14.98)	25.13 <sub>b</sub> (11.65)	4.61**
Public anxiety	25.20 <sub>b</sub> (7.08)	38.06 <sub>a,d</sub> (12.73)	34.86 (13.34)	26.60 <sub>b</sub> (11.83)	4.44**

Table 3.9 Continued

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u> (3,56)
Anxiety Sensitivity Profile					
Dissociative/Neurological	27.60 <sub>b</sub> (8.97)	40.73 <sub>a,d</sub> (13.72)	36.60 (14.89)	28.00 <sub>b</sub> (12.66)	3.89**
Cognitive Dyscontrol	25.53 <sub>b</sub> (8.67)	40.20 <sub>a,d</sub> (12.41)	34.66 <sub>d</sub> (13.41)	22.93 <sub>b,c</sub> (10.80)	7.33***

Note: Groups are significantly different from means subscripted.

\*\*  $p < .01$ . \*\*\*  $p < .001$ .

### Hypothesis Six

Individuals in the Asthma only, Panic only, and Asthma and Panic groups were expected to report more misinterpretation of bodily sensations than women in the Control group. Because three participants failed *to* complete the Brief Body Sensations Interpretation Questionnaire (BBSIQ; Clark et al., 1997), the Asthma and Panic group totaled 13 and the Panic only group totaled 14 for the following analyses. A MANOVA on the BBSIQ subscales revealed an overall significant group effect Wilks' lambda,  $F(18,136) = 2.37, p < .003, \text{power} = .98, \eta^2 = .22$  (see Table 3.10). Followup ANOVAs indicated no significant group differences on measures of external explanation rankings or neutral explanations for either the panic or external scenes,  $F(3,53) = .25, \text{ns}$ , and  $F(3,53) = .32, \text{ns}$ , respectively. The panic explanations were rated higher than the neutral explanations by the Panic only group in comparison to the Control group,  $F(3,53) = 3.81, p < .01$ . No differences were found for this subscale for the Asthma only or Asthma and Panic groups. The panic explanations were rated higher by women in the Asthma and Panic and Panic only groups compared to women in the Asthma only and Control groups,  $F(3,53) = 10.71, p < .0001$ . Finally, women in the Panic only group rated the external explanations as more likely than the Asthma only group,  $F(3,53) = 3.32, p < .02$ . No differences were found for the Control or Asthma and Panic groups on this subscale.

A MANOVA on Anxiety Sensitivity Profile (ASP; Taylor & Cox, 1998) subscale scores (see Table 3.9) revealed a significant overall group effect, Wilks' lambda  $F(3,56) = 1.73, p < .04, \text{power} = .92, \text{effect size} = .41$ . Followup oneway ANOVAs and post hoc comparisons identified two patterns of significant differences. On the Cardiovascular,  $F(3,56) = 5.87, p < .001$ , Gastrointestinal,  $F(3,56) = 4.61, p < .006$ , Public Anxiety,



Table 3.10

Means and Standard Deviations for Brief Body Sensations Interpretation Questionnaire

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u> (3,56)
Panic explanation rank	2.76 (.28)	2.48 (.51)	2.36 <sub>d</sub> (.54)	2.81 <sub>c</sub> (.28)	3.18**
External explanation rank	2.63 (.52)	2.48 (.59)	2.33 (.59)	2.58 (.43)	.86
Panic explanation rating	1.47 <sub>b,c</sub> (1.17)	3.60 <sub>a,d</sub> (1.95)	3.00 <sub>a,d</sub> (1.41)	.89 <sub>b,c</sub> (1.21)	10.71***
External explanation rating	1.90, (1.07)	3.36 (2.01)	3.48 <sub>a</sub> (1.62)	2.46 (1.47)	3.32*
Neutral explanations- panic scenes	5.71 (1.04)	5.65 (1.23)	5.98 (.78)	5.65 (1.12)	.32
Neutral explanations-external scenes	6.21 (.97)	6.09 (1.51)	6.05 (1.02)	6.04 (1.02)	.07

Note: Groups are significantly different from means subscripted.

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

$\underline{F}(3,56) = 4.44, p < .007$ , and Dissociative/Neurological subscales,  $\underline{F}(3,56) = 3.89, p < .01$ , the Asthma and Panic group scored significantly higher than the Asthma and Control groups. No differences were identified on these subscales for the Panic only group. For the Respiratory,  $\underline{F}(3,56) = 6.66, p < .001$ , and Cognitive Dyscontrol,  $\underline{F}(3,56) = 7.33, p < .001$ , subscales, the Asthma and Panic group scored higher than the Asthma only and Control groups. In addition, the Panic only group scored higher than the Control group on the above subscales.

### Hypothesis Seven

Women in the Panic only and Asthma and Panic groups were expected to report more fear of anxiety sensations than the Asthma group. In turn, the Asthma only group was expected to report a higher level than the Control group. Panic-fear levels were expected to be higher for the Asthma and Panic group than the Asthma only group. Significant differences between groups (see Table 3.9, Figure 3.3) were identified on the Anxiety Sensitivity Index (ASI; Reiss et al., 1986),  $\underline{F}(3, 56) = 10.55, p < .0001$ . Women in the Asthma and Panic and Panic only groups scored higher than women in the Asthma only and Control groups. However, women in the Asthma only group did not report greater fear of anxiety sensations than women in the Control group. As discussed above, women in the Asthma and Panic group scored higher than women in the Asthma only group on four of six subscales of the ASC, including the Panic-Fear subscale.

### Ancillary Analyses

#### Visual Analog Scales

Several women did not to complete one or more of the visual analog scales (VAS; asthma attack similarity, panic attack similarity, control over sensations, stressfulness of

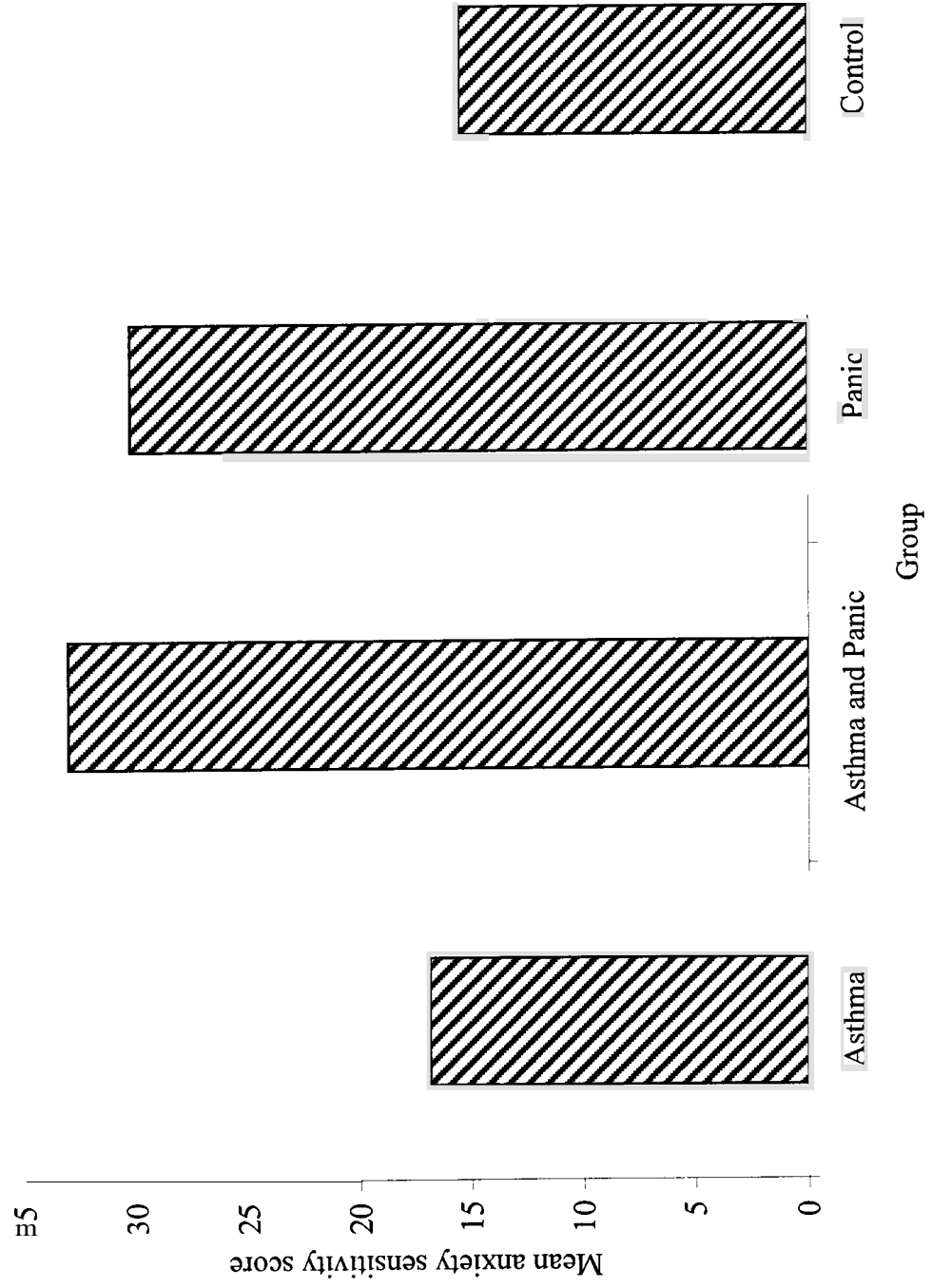


Figure 3.3. Mean anxiety sensitivity score by group

task). As a result, cell sizes are not consistent for these measures. In cases where cell sizes differ, the associated  $F$  test degrees of freedom have been listed along with means and standard deviations in Table 3.11. Oneway ANOVAs on baseline VAS scores were calculated. No significant group differences were identified for the panic VAS  $F(3,56) = 2.16$ , ns or asthma VAS,  $F(3,46) = 2.12$ , ns. On the stress VAS, a trend approaching significance was found,  $F(3,56) = 2.57$ ,  $p < .06$ . Women in the Panic only group rated the baseline as more stressful than women in the Control group. No differences were identified for the Asthma only or Asthma and Panic groups. On the control VAS, a similar pattern was identified, with women in the Panic only group reporting that they had less control over the sensations experienced at baseline than women in the Control group,  $F(3,56) = 3.81$ ,  $p < .01$ . Again, no significant differences were identified for the Asthma only or Asthma and Panic groups.

In order to control for baseline VAS scores, change scores for each VAS from baseline were calculated for each of the three tasks. A MANOVA on the VAS for each task was conducted. For the panic VAS, Wilks' lambda  $F(9,129) = 1.78$ ,  $p < .07$ , power = .66,  $\eta^2 = .09$ , a trend toward significance was identified. Post hoc tests, however, revealed no significant group differences on the head turning  $F(3,55) = 2.27$ , ns and relaxation tasks  $F(3,55) = .29$ , ns. On the straw task,  $F(3,55) = 5.22$ ,  $p < .003$ , women in the Asthma and Panic group reported that the task was much more like a panic attack than women in the Asthma only or Control groups. No differences were identified for the Panic only group.

For the MANOVA on the asthma VAS, a group main effect was significant, Wilks' lambda,  $F(9,99) = 2.58$ ,  $p < .01$ , power = .84,  $\eta^2 = .15$ . Followup ANOVAs

Table 3.11

Means and Standard Deviations for Visual Analog Scales

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u>
Panic Visual Analog Scale					
Baseline	2.13 (1.51)	1.67 (.62)	2.73 (1.79)	1.67 (1.11)	<u>F</u> (3,56) 2.16
Head Turning	2.40 (1.50)	3.20 (1.42)	3.73 (2.12)	2.47 (1.41)	<u>F</u> (3,55) = 2.27
Straw	2.53 <sub>b</sub> (1.19)	3.73 <sub>a,d</sub> (1.03)	4.20 (1.78)	2.47 <sub>b</sub> (1.25)	<u>F</u> (3,55) = 5.22**
Relaxation	1.47 (1.13)	1.33 (.62)	2.13 (1.85)	1.14 (.36)	<u>F</u> (3,55) = .29
Asthma Visual Analog Scale					
Baseline	2.67 (1.50)	1.80 (1.21)	3.00 (2.14)	1.67 (1.30)	<u>F</u> (3,46) = 2.12
Head Turning	3.00 (1.56)	2.80 (1.15)	4.38 (1.77)	2.09 (1.22)	<u>F</u> (3,43) = 1.17
Straw	3.73 <sub>b</sub> (1.58)	4.67 <sub>a,d</sub> (1.11)	5.14 (1.21)	3.00 <sub>b</sub> (2.32)	<u>F</u> (3,43) = 4.80**
Relaxation	1.73 (1.44)	1.33 (.82)	2.00 (1.55)	1.09 (.30)	<u>F</u> (3,43) = 1.16

Table 3.11 Continued

	Asthma (a)	Asthma & Panic (b)	Panic (c)	Control (d)	
Measure	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )	<u>F</u>
Stress Visual Analog Scale					
Baseline	1.93 (1.22)	2.07 (1.44)	2.47 (.83)	1.40 (.51)	$\underline{F}(3,56) = 2.57$
Head Turning	2.47 (1.30)	3.40 (1.88)	3.93 (1.91)	2.33 (1.29)	$\underline{F}(3,55) = 1.53$
Straw	2.87 (1.46)	3.80 (1.15)	4.20 (1.70)	2.80 (1.52)	$\underline{F}(3,55) = 1.24$
Relaxation	1.67 (1.29)	1.73 (.88)	2.40 (1.84)	1.50 (1.34)	$\underline{F}(3,55) = .28$
Control Visual Analog Scale					
Baseline	4.67 (1.72)	5.27 (1.39)	4.33 <sub>d</sub> (1.84)	6.13, (1.25)	$\underline{F}(3,56) = 3.81^{**}$
Head Turning	4.27 (1.87)	3.73 (1.39)	3.07 (1.98)	4.60 (1.55)	$\underline{F}(3,55) = 2.27$
Straw	3.87 (1.60)	3.27 (1.03)	3.27 (1.75)	4.40 (1.87)	$\underline{F}(3,55) = 2.25$
Relaxation	4.93 (1.67)	5.53 (1.68)	5.13 (1.88)	5.64 (1.98)	$\underline{F}(3,55) = .22$

Note: Groups are significantly different from means subscripted.  $^{**} p < .01$ .

indicated no significant differences on the head turning,  $F(3,46) = 1.17, \underline{ns}$ , or relaxation tasks,  $F(3,43) = 1.16, \underline{ns}$ . On the straw task, the ANOVA was significant,  $F(3,43) = 4.80, p < .006$ . Post hoc tests revealed that the Asthma and Panic group rated the straw task as more like an asthma attack than the Asthma only and Control groups. No differences were identified for the Panic only group.

On the stress **VAS**, a MANOVA revealed no group effect, Wilks' lambda  $F(9, 129) = .89, \underline{ns}$ , power = .34,  $\eta^2 = .04$ . Similarly, the analysis for the control VAS was not significant, Wilks' lambda  $F(9, 129) = 1.60, \underline{ns}$ , power = .61,  $\eta^2 = .08$ . Hence, no followup ANOVAs were calculated for these two measures.

#### Correlations between Questionnaire Measures

Correlations between several of the questionnaire measures (ASI, STAI-S, STAI-T, BVS) were calculated (see Table 3.12). Significant positive correlations were found between all four of these measures. Each measure was significantly correlated with each other measure.

Additional correlational analyses were completed to examine relationships between the ASI and subscales of the ASP and ASC. For the ASI and ASC correlations, data from the Asthma only and Asthma and Panic groups was used, as the other two groups did not complete the ASC (Tables 3.13 and 3.14). Interestingly, for women in the Asthma only group, the ASI was not significantly correlated with any of the ASP subscales (Cardiovascular, Respiratory, Gastrointestinal, Public Anxiety, Dissociative/Neurological, and Cognitive Dyscontrol), but for women in the Asthma and Panic group, the ASI was positively correlated with every subscale of the ASP. Subscales of the ASC (Panic-Fear, Irritability, Fatigue, Hyperventilation, Bronchoconstriction) that were

Table 3.12

Correlations between Questionnaire Measures

	Body Vigilance Scale	State Trait Anxiety Inventory-State	State-Trait Anxiety Inventory-Trait
Anxiety Susceptibility	.81 ***	.50 **	.60 **
Inventory			
Body Vigilance		.40* *	.52 **
Scale			
State-Trait Anxiety			.59***
Inventory-State			

\*  $p < .01$  \*\*  $p < .001$



Table 3.13

Correlations between Asthma Symptom Checklist Subscales and Anxiety Sensitivity Measures for the Asthma Only Group

	Anxiety Sensitivity Inventory	Panic- Fear	Irritability	Fatigue	Hyperventilation	Bronchoconstriction
Anxiety Sensitivity Inventory		.62*	.62*	.30	.60*	.46
Cardiovascular	.47	.50	.50	.28	.51*	.27
Respiratory	.39	.34	.45	.33	.51	.17
Gastrointestinal	.35	.57*	.58*	.39	.61*	.44
Public Anxiety	.32	.42	.48	.12	.37	.19
Dissociative/Neurological	.32	.50	.47	.34	.44	.34
Cognitive Dyscontrol	.25	.27	.46	.22	.36	.11

\*  $p < .05$ .

Table 3.14

Correlations between Asthma Symptom Checklist Subscales and Anxiety Sensitivity Measures for the Asthma and Panic Group

	Anxiety Sensitivity Inventory	Panic- Fear	Irritability	Fatigue	Hyperventilation	Bronchoconstriction
Anxiety Sensitivity Inventory		.80**	.32	.36	.06	.30
Cardiovascular	.74**	.61*	.45	.22	.16	-.07
Respiratory	.81**	.67**	.47	.35	.30	.03
Gastrointestinal	.90**	.72**	.30	.37	.02	.05
Public Anxiety	.82**	.82**	.36	.43	.05	.02
Dissociative/Neurological	.93**	.74**	.38	.33	.08	.05
Cognitive Dyscontrol	.85**	.68**	.37	.31	.05	.09

\*  $p < .05$ . \*\*  $p < .01$

significantly correlated with the ASI for the Asthma only group included the Panic-Fear, Irritability, and Hyperventilation subscales. For the Asthma and Panic group, only the Panic-Fear subscale correlated significantly with the ASI. In examining correlations between ASC and ASP subscales, for women in the Asthma only group, the Hyperventilation and Cardiovascular subscales were positively correlated. The Gastrointestinal subscale was significantly correlated with the Panic-Fear, Irritability, and Hyperventilation subscales. For women in the Asthma and Panic group, the only subscale that was correlated with any subscale of the ASP was the Panic-Fear subscale. This subscale was significantly positively correlated with all six ASP subscales (see Table 3.14).

For the Panic only and Control groups, correlations between the ASI and the subscales of the ASP were calculated (see Table 3.15). For the Panic only group, the ASI was significantly positively correlated at  $p < .01$  for all six ASP subscales. For the Control group however, no ASP subscale was significantly correlated with the ASI.

#### Exploratory Within-Subject Analyses

The three experimental tasks in the present study were conceptualized as being different with regard to the testing of three key theories. The hypotheses were phrased so that between-groups variables were the primary variables of interest. For this reason, when this study was in its design phase, repeated measures MANOVA was not considered as an analysis tool. However, given that so few of the predicted relations emerged, further exploratory analyses using repeated measures MANOVA were conducted in order to explore within groups effects. Investigating changes in lung function and the questionnaire measures for each group across tasks allows for the

Table 3.15

Correlations between subscales of the Anxiety Sensitivity Profile and the Anxiety Sensitivity Index

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	Panic	Control
Cardiovascular	.69**	.50
Respiratory	.67**	.31
Gastrointestinal	.71**	.32
Public Anxiety	.67**	.41
Dissociative/Neurological	.75**	.37
Cognitive Dyscontrol	.70**	.30

---

\*\*  $p < .01$ .

identification of tasks that might elicit more asthma/panic related symptoms or changes in lung function for a particular group. The questionnaire and spirometric data following the tasks were re-analyzed with **4** (Group: Asthma only, Panic only, Asthma and Panic, Control) x **3** (Task Change Score: Head Turning, Straw, Relaxation) MANOVAs. The task change score represents the repeated measures and the change from baseline for each dependent variable for each task. No significant Group main effects were found for any of the post-task measures. To test within-groups differences across tasks, paired t-tests were performed with alpha set at .05. A summary of the results is presented in Table 3.16.

Profile of Mood States. Although a significant Task main effect was found for anxious mood, Wilks' lambda  $F(2,54) = 35.94, p < .0001, \eta^2 = .57$ , power = 1.00, no significant Group main effect was found, Wilks' lambda  $F(3, 122) = 1.88, ns$ . However, a significant Group x Task interaction was found, Wilks' lambda  $F(6,108) = 3.37, p < .004, \eta^2 = .15$ , power = .92. No significant Task simple main effect was found for the Control group,  $F(2,54) = 1.93, ns$ . However, significant Task simple main effects were found for the Asthma only,  $F(2,54) = 3.98, p < .024$ , Panic only,  $F(2,54) = 9.64, p < .0001$ , and Asthma and Panic groups,  $F(2,54) = 32.00, p < .0001$ .

Post hoc t-tests indicated that participants in the Asthma only group reported more anxious mood from baseline to the straw task than from baseline to head turning task or relaxation task (see Table 3.3). No significant differences were identified for the head turning and relaxation tasks for the Panic only group. For the Panic only group, anxious mood scores increased more from baseline to straw task and head turning task than from

Table 3.16

Summary of Within-Subjects Results

Measure	Asthma	Asthma & Panic	Panic	Control
Profile of Mood States – Anxiety Subscale	ST > HT = RL	ST = HT > RL	ST = HT > RL	<u>ns</u>
Panic Attack Cognitions Questionnaire	<u>ns</u>	ST = HT > RL	ST = HT > RL	<u>ns</u>
Borg Category Scale	ST > HT > RL	ST > HT > RL	ST > HT > RL	ST > HT > RL
FEV <sub>1</sub>	<u>ns</u>	<u>ns</u>	<u>ns</u>	<u>ns</u>
FVC	<u>ns</u>	<u>ns</u>	<u>ns</u>	<u>ns</u>
FEV <sub>1</sub> /FVC	<u>ns</u>	HT > ST RL = HT, ST	<u>ns</u>	<u>ns</u>

Note. ST = straw task, HT = head turning task, RL = relaxation task.

Table 3.16 Continued

Measure	Asthma	Asthma & Panic	Panic	Control
Symptom Checklist Subscales				
Panic-Fear	<u>ns</u>	ST = HT > RL	ST = HT > RL	<u>ns</u>
Irritability	<u>ns</u>	ST > RL	ST = HT > RL	<u>ns</u>
		HT = ST, RL		
Bronchoconstriction	ST > HT > RL	ST > HT > RL	ST > HT > RL	ST > HT > RL
Fatigue	<u>ns</u>	HT > RL	<u>ns</u>	<u>ns</u>
		ST = HT, RL		
Hyperventilation	ST = HT > RL	ST = HT > RL	<u>ns</u>	<u>ns</u>
Panic Specific	ST = HT > RL	ST > HT > RL	ST > HT > RL	<u>ns</u>

Note. ST = straw task, HT = head turning task, RL = relaxation task.

baseline to relaxation task. No differences were found between the straw and head turning tasks for the Panic only group. For the Asthma and Panic group, participants reported more anxious mood from baseline to head turning task and straw task than from baseline to relaxation task. No significant differences were identified for the Asthma and Panic group's head turning and straw task anxious mood change scores.

Panic Attack Cognitions Questionnaire. For the PACQ, a significant Task main effect, Wilks' lambda  $F(2,54) = 19.19, p < .0001, \eta^2 = .41, \text{power} = 1.00$ , and Group x Task interaction, Wilks' lambda  $F(6,108) = 3.01, p < .009, \eta^2 = .14, \text{power} = .89$ , was found. No significant Group main effect was identified,  $F(3,55) = .79, p < .50, \eta^2 = .04, \text{power} = .21$ . Breaking down the interaction, no significant simple main effect was found for the Control group,  $F(2,54) = .34, \text{ns}$ , or the Asthma only group,  $F(2,54) = 1.10, \text{ns}$ . For the Panic only group, a significant simple main effect for Task was found,  $F(2,54) = 14.55, p < .0001$ , as well as for the Asthma and Panic group,  $F(2,54) = 13.35, p < .0001$  (see Table 3.8). Post hoc tests revealed that women in the Panic only and Asthma and Panic groups reported more panic related cognitions from baseline to the head turning and straw tasks relative to the relaxation task. No significant differences were identified between change scores for the head turning and straw tasks for either group.

Born Category Scale. The BCS analyses resulted in a significant Task main effect, Wilks' lambda  $F(2,54) = 19.19, p < .0001, \eta^2 = .41, \text{power} = 1.00$ , and a significant Group x Task interaction, Wilks' lambda  $F(6,108) = 3.01, p < .009, \eta^2 = .14, \text{power} = .89$ . No significant Group main effect was found, Wilks' lambda  $F(3,55) = .79, \text{ns}, \eta^2 = .04, \text{power} = .21$ . Breaking down the interaction, significant simple main effects were found for the Control, Wilks' lambda  $F(2,54) = 13.38, p < .0001$ , Panic only,



Wilks' lambda  $F(2,54) = 28.88, p < .0001$ , Asthma and Panic, Wilks' lambda  $F(2,54) = 38.26, p < .0001$ , and Asthma only groups, Wilks' lambda  $F(2,54) = 8.53, p < .001$ . For all four groups, post hoc tests revealed that the straw task produced greater reports of dyspnea symptoms than the head turning task, and that the straw and head turning tasks resulted in greater reports of dyspnea than the relaxation task (see Table 3.4).

Spirometric Measures. Percent predicted values for FEV<sub>1</sub> and FVC were utilized, as well as the ratio of FEV<sub>1</sub> /FVC. For FEV<sub>1</sub>, no significant main effects or interactions were identified for the change scores. For Task, Wilks' lambda  $F(2,53) = 2.46, ns, \eta^2 = .08$ , power = .47, for Group, Wilks' lambda  $F(3,54) = 1.49, ns, \eta^2 = .07$ , power = .37, and for Group x Task, Wilks' lambda  $F(6,106) = .46, ns, \eta^2 = .02$ , power = .18.

For FVC, a Task main effect was found, Wilks' lambda  $F(2,54) = 3.21, p < .04$ ,  $\eta^2 = .10$ , power = .59. No Group main effect, Wilks' lambda  $F(3,55) = .99, ns, \eta^2 = .05$ , power = .25, or Group x Task interaction was found, Wilks' lambda  $F(6,108) = .93, ns, \eta^2 = .04$ , power = .35. In order to follow up the Task main effect, paired t-tests with a more conservative  $p$  value of .01 were utilized to control for Type I errors. No significant simple main effects were identified for Task, head turning vs. straw,  $t(1,58) = -2.25, ns$ , head turning vs. relaxation,  $t(1,57) = -.85, ns$ , or straw vs. relaxation change scores,  $t(1,58) = 1.27, ns$ .

For the ratio of FEV<sub>1</sub> /FVC, a significant Task main effect was found, Wilks' lambda  $F(2,53) = 4.44, p < .01, \eta^2 = .14$ , power = .73. No significant Group main effect, Wilks' lambda  $F(3,54) = 1.24, ns, \eta^2 = .06$ , power = .37, or Group x Task interaction was identified, Wilks' lambda  $F(6,106) = 1.22, ns, \eta^2 = .06$ , power = .46. Again, following up the Task main effect, no significant simple main effects were found

for head turning vs. straw,  $t(1,58) = -2.35$ , ns, head turning vs. relaxation,  $t(1,57) = -2.51$ , ns, or straw vs. relaxation change scores,  $t(1,58) = -.29$ , ns.

Symptom Checklist Subscales. For the subscales of the SCL, no Group main effect was identified, Wilks' lambda  $F(3,55) = 2.09$ , ns,  $\eta^2 = .10$ , power = .50. A significant Task main effect Wilks' lambda  $F(2,54) = 65.14$ ,  $p < .0001$ ,  $\eta^2 = .70$ , power = 1.00, and a significant Group x Task interaction was found, Wilks' lambda  $F(6,108) = 4.20$ ,  $p < .001$ ,  $\eta^2 = .19$ , power = .97. See Table 3.6 for means, standard deviations, and change scores for the subscales of the SCL.

Breaking down the interaction, no significant differences were found for the Panic-Fear subscale for the Control group, Wilks' lambda  $F(2,54) = .40$ , ns, or the Asthma and Panic group, Wilks' lambda  $F(2,54) = 1.03$ , ns. However, for the Panic only and Asthma and Panic groups, significant differences were found, Wilks' lambda  $F(2,54) = 12.77$ ,  $p < .0001$ , Wilks' lambda  $F(2,54) = 23.68$ ,  $p < .0001$ . Post hoc tests revealed no significant differences in Panic-Fear change scores between the straw and head turning tasks for the Asthma only and Panic only groups. Women in both the Asthma only and Panic only groups reported greater changes in Panic-Fear ratings for the head turning and straw tasks than the relaxation task.

On the Irritability subscale, no significant differences were found for the Control group, Wilks' lambda  $F(2,54) = 2.28$ , ns, or the Asthma only group, Wilks' lambda  $F(2,54) = .37$ , ns. However, significant differences were identified for the Panic only group, Wilks' lambda  $F(2,54) = 13.36$ ,  $p < .0001$ , as well as for the Asthma and Panic group, Wilks' lambda  $F(2,54) = 8.55$ ,  $p < .001$ . Post hoc tests revealed no significant differences in irritability ratings between the straw and head turning tasks for the Asthma

and Panic group and Panic only groups. However, women in the Asthma and Panic group reported greater irritability for the head turning and straw tasks from baseline than the relaxation task. No significant difference was found for the Panic only group between the head turning task and relaxation tasks although a significant difference emerged between the straw task and relaxation task, with the straw task producing greater self-reports of irritability from baseline.

For the Bronchoconstriction subscale, significant differences for all four groups were found, Control, Wilks'  $\lambda F(2,54) = 10.12, p < .0001$ , Panic only, Wilks'  $\lambda F(2,54) = 17.89, p < .0001$ , Asthma and Panic, Wilks'  $\lambda F(2,54) = 45.76, p < .0001$ , Asthma only, Wilks'  $\lambda F(2,54) = 11.22, p < .0001$ . Women in the Asthma only, Panic only, and Asthma and Panic groups reported more bronchoconstriction after the straw task than the head turning task, and women in these groups reported more bronchoconstriction after the head turning task and the straw task compared to the relaxation task. Participants in the control group reported more bronchoconstriction on the straw task than the head turning and relaxation tasks. No significant difference was found for the head turning and relaxation tasks for the Control group.

Analysis of the Fatigue subscale resulted in significance for the Panic only group, Wilks'  $\lambda F(2,54) = 5.20, p < .009$ . No significant effect was found for the Control group, Wilks'  $\lambda F(2,54) = .42, ns$ , Asthma and Panic group, Wilks'  $\lambda F(2,54) = .59, p < .55$ , or the Asthma only group, Wilks'  $\lambda F(2,54) = .06, p < .93$ . Post hoc tests for the Panic only group revealed that these women reported more fatigue for the head turning task than the relaxation task. No significant differences between the straw and head turning or straw and relaxation task were found.

The Hyperventilation subscale analyses revealed a significant effect for the Panic only, Wilks' lambda  $F(2,54) = 13.19, p < .0001$ , Asthma only, Wilks' lambda  $F(2,54) = 3.20, p < .04$ , and Asthma and Panic groups, Wilks' lambda  $F(2,54) = 17.59, p < .0001$ . No significant effect was found for the Control group, Wilks' lambda  $F(2,54) = 2.88, ns$ . Post hoc tests for the Asthma only and Panic only groups revealed no significant difference between the head turning and straw tasks, but the head turning and straw tasks produced greater reports of hyperventilation than the relaxation task. Women in the Asthma and Panic group reported more hyperventilation for the head turning task than the straw task. In addition, the head turning and straw tasks resulted in greater self-reports of hyperventilation than the relaxation task.

Finally, for the Panic Specific subscale, no significant differences were identified for the Control group, Wilks' lambda  $F(2,54) = 2.22, ns$ . However, a significant effect was found for the Panic only, Wilks' lambda  $F(2,54) = 12.47, p < .0001$ , Asthma and Panic, Wilks' lambda  $F(2,54) = 18.60, p < .0001$ , and Asthma only groups, Wilks' lambda  $F(2,54) = 3.39, p < .04$ . Post hoc testing revealed no differences between the head turning and straw tasks for the Panic only, Asthma only, and Asthma and Panic groups, but head turning and straw tasks resulted in increased reports of panic specific symptoms compared to the relaxation task.

## Chapter 4

### DISCUSSION

In this study, three theories that relate to the comorbidity of asthma and panic disorder were tested. Women with or without asthma and with or without panic disorder were assessed on both spirometric and self-report measures of their responses to three tasks designed to test aspects of each theory.

#### Self-Report

##### State and Trait Anxiety

Previous research conducted by Dorhofer & Sigmon (in press), utilizing groups of women with or without asthma, and with or without panic attacks, found differences in state anxiety. In the Dorhofer & Sigmon (in press) study, women with both asthma and a history of panic attacks reported significantly more state anxiety than controls. They endorsed less state anxiety overall than women in the present study. The differences in the means for the two studies may explain the apparent discrepancy in results. Both controls and women with both asthma and panic disorder endorsed more state anxiety than in the previous study. It is possible that anticipation of the experimental tasks in this study contributed to increases in self-reports of state anxiety for participants compared to participants in the previous study.

Differences in trait anxiety are consistent with previous research that found that individuals with both asthma and panic disorder reported significantly higher levels of trait anxiety than individuals with asthma only (van Peski-Oosterbaan et al., 1996). In addition, using the Taylor Manifest Anxiety Scale (Taylor, 1953) to assess trait anxiety, Carr et al. (1996) found that individuals with both asthma and panic disorder reported

more trait anxiety than individuals with asthma only or controls, and that individuals with panic disorder reported more trait anxiety than individuals with asthma only. Thus, converging lines of evidence suggest that having panic disorder with or without asthma leads to greater reports of trait anxiety in general, in addition to having episodes of panic. One possible sequelae regarding the effectiveness of cognitive-behavioral treatment for panic disorder may lie in its ability to not only provide skills for coping with panic attacks, but to lower levels of trait anxiety through cognitive restructuring as well.

### Asthma Symptoms

It is unclear why women in the Asthma and Panic group would report more irritability and fatigue but not more hyperventilation or bronchoconstriction symptoms than women with asthma alone. Hyperventilation and bronchoconstriction are symptoms that may be characteristic of a panic attack, as well as an asthma attack. It may be that questionnaire instructions were specific enough to limit participants' ratings to actual asthma attack and not panic attack symptoms ("Please rate the following symptoms you may experience when you are having an asthma attack."). To date, no other published study has compared respiratory symptoms for individuals who have asthma with or without panic disorder. Future research should investigate more thoroughly and with a more diverse sample the effects that having comorbid asthma and panic disorder may have on self-reports of asthma symptoms.

### Focus on Bodily Sensations

It was predicted that women in the Asthma only group would report more self-focusing than women in the Control group. However, this was not the case. It appears that meeting diagnostic criteria for panic disorder is concomitant with increased focus on

bodily sensations, but a diagnosis of asthma does not necessarily lead to greater reports of bodily self-focus.

Previous research has utilized the Body Sensations Questionnaire (BSQ; Chambless et al., 1984) as a measure of fear of specific bodily sensations in populations with or without asthma and with or without panic disorder (Carr et al., 1994). The Carr et al. (1994) study found no significant differences in fear of bodily sensations between individuals with or without asthma only. Participants with panic disorder (with or without asthma) reported higher levels of fear of bodily sensations. These results parallel current findings. However, no published research has specifically examined the amount of focus on bodily sensations in a population with asthma. Future research should develop measures that can assess the amount of focus on bodily sensations and fear of bodily sensations that includes symptoms that are specific to asthma.

#### Anxiety Sensitivity

Extensive research has documented the role that anxiety sensitivity plays in the development and maintenance of panic disorder (Maller & Reiss, 1992; Reiss, 1987; Taylor et al., 1992), consistent with higher self-reports of anxiety sensitivity of women in the two panic groups. Consistent with some previous research, no anxiety sensitivity differences were identified between individuals with and without asthma. For example, Carr et al. (1994) found no anxiety sensitivity differences between groups with asthma alone and controls. However, it should be noted that the Carr et al. (1994) study had uneven groupings and some groups had a small sample size. This limitation may have prevented the identification of significant findings for the anxiety sensitivity construct.

Although the present study's lack of significant differences in anxiety sensitivity level between the Asthma only and Control groups is consistent with some research, the results are in sharp contrast to Dorhofer & Sigmon (in press). Dorhofer and Sigmon (in press) did find clear differences between women with asthma only and controls. It is not clear why these two studies, conducted with similar numbers of participants drawn from a similar student population, have inconsistent results. It is possible that the criteria used to determine group inclusion in the Dorhofer & Sigmon (in press) study may not have adequately divided individuals with and without a history of panic attacks. The authors used a time criterion, panic attacks within the last year. Women with a history of panic attacks longer than 1 year previous to the study may have been included in the Asthma only group. This inclusion may have led to a higher mean level of anxiety sensitivity for the Asthma group. Further research in this area is necessary to provide additional evidence as to whether or not individuals with asthma alone have higher levels of anxiety sensitivity than individuals who do not have asthma. In addition, if high levels of anxiety sensitivity are found for individuals with asthma only, the implications for the development of panic disorder in this population will need to be thoroughly examined.

Interestingly, although it is logical to expect that women with both asthma and panic disorder would score higher in comparison to women with asthma only on measures of fear of respiratory, dissociative/ neurological, and cognitive dyscontrol symptoms, these women also scored higher on scales that assess severity of fear of gastrointestinal, public anxiety, and cardiovascular symptoms. In contrast, women with panic disorder only scored higher on the respiratory and cognitive dyscontrol scales. It may be that women with both asthma and panic disorder learn to fear a wider array of



symptoms because they are vigilant about two different types of attacks- panic and asthma attacks. Anecdotally, women with asthma and panic disorder report that they frequently have trouble identifying whether sensations are asthma or panic related. It may be that these women fear any bodily sensation, regardless of the cause of the sensation. Again, it is not known whether this fear arises as a consequence of having a chronic illness, or if it is a separate phenomenon.

Future research should focus on helping individuals who have both asthma and panic disorder identify the source of anxiety producing bodily sensations. This skill may be crucial, because what an individual does to cope with the sensations will greatly differ depending on the origin of the sensations. If the sensations are perceived as being asthma-related, the individual may use a short-acting inhaler (i.e., albuterol) to treat the symptoms. If the sensations are perceived as panic-related, the individual may use cognitive, relaxation, or other techniques to address the sensations. Unfortunately, because short-acting inhalers often cause anxiety symptoms, mistaking panic sensations for asthma symptoms and treating oneself with an inhaler may actually lead to increased anxiety. Treatment involving exposure to asthma or panic sensations, as currently practiced in cognitive-behavioral therapy for panic disorder, may also be beneficial to reduce anxiety about these sensations.

Results are consistent with previous findings that panic-fear scale is positively correlated with a diagnosis of panic disorder (~~Car~~ et al., 1995). Given that panic-fear levels are associated with a variety of negative outcomes for individuals with asthma, the difference in women with or without panic disorder is an important one. Future research should examine whether a diagnosis of panic disorder in individuals with asthma is linked

to higher numbers of visits to primary health care practitioners, increased numbers of hospitalizations, and higher numbers of prescriptions. Overall health care costs may be higher for individuals with both diagnoses. Furthermore, treatment of panic disorder in individuals with asthma may be able to reduce numbers of visits, hospitalizations, prescriptions, and overall health care costs.

### Mood After Tasks

Previous research has identified correlational differences in mood that are related to peak air flow measurements for individuals diagnosed with asthma (Hyland, 1990). No consensus has been reached as to which specific emotions are associated with decreases in peak air flow, but it is suspected that negative mood states are more likely to be linked to reduced lung function (Steptoe & Holmes, 1985). Although individuals with asthma have been found to exhibit greater physiological changes in response to stressful activities than controls, controls also show some changes in oscillatory resistance that are correlated with self-reports of intense emotions (Ritz, Steptoe, DeWilde, & Costa, 2000). These findings were supported by the present study. For the sample as a whole, as anxious mood increased, lung capacity decreased.

Although the combination of both asthma and panic disorder may lead to a similar level of anxiety on a general basis, having both disorders did not result in greater self-reports of transitory anxious mood during this particular baseline situation. These results differ from baseline anxiety mood scores for women in the Dorhofer & Sigmon (in press) study, where the group with both asthma and a history of panic attacks reported significantly higher baseline anxious mood than the Control group. Again, it may be that differences in group assignment criteria led to the discrepant results between these two

studies. Nevertheless, taking the results of both studies together, it appears that having panic attacks or panic disorder with or without asthma leads to reports of higher baseline anxious mood.

The present study is limited in its ability to more thoroughly examine correlations between anxious mood and lung function for each group separately because of its small sample size and problems with reliability of the lung function measures. In addition, because of high correlations between several measures, multiple regressions predicting lung function measures could not be conducted. Thus, only mood and lung function correlations were calculated for the entire sample, and were not broken down by group. To date, no published study has specifically examined how the relation between mood and lung function may differ for individuals who have asthma with or without panic disorder. It is important that future research investigate potential differences in the correlation between anxious mood and lung function utilizing these two groups of individuals.

### Cognitive Theory

Several hypotheses addressed the role that cognitions may play in the relation between asthma and panic disorder. An adaptation of Clark's cognitive theory of panic to asthma suggests that thoughts about bodily sensations may lead to a worsening of asthma symptoms or a panic attack. Mislabeling non-asthma related sensations as asthma-related has been found to be related to increases in rehospitalization (Dirks & Schraa, 1983), and may be connected to other negative outcomes for individuals with asthma (e.g., overuse of medications).

Clark (1993) outlines three criteria that must be fulfilled in order for cognitive mediation of panic attacks to be supported by research. These criteria were adapted to the asthma literature for the purposes of the present study.

#### Criterion One

The adaptation of Criterion One stated that individuals with asthma fear and misinterpret certain bodily sensations more than controls. Taking the results of the three anxiety sensitivity questionnaire measures together, it appears that women who have asthma without panic disorder do not tend to misinterpret bodily sensations more than women without asthma. Therefore, Criterion One was not supported by the results of this study as it pertains to individuals with asthma only. However, women with both panic disorder and asthma misinterpret, fear, and focus on various types of bodily sensations more than both women with asthma only and controls.

Future research should investigate the types of sensations that individuals with both asthma and panic disorder fear, and how these sensations differ from those feared by individuals with asthma or panic disorder only. As discussed above, research examining how individuals with both asthma and panic disorder interpret and differentiate physical symptoms is crucial. It will be important in the future to develop treatments to assist individuals in learning how to discriminate between various types of bodily sensations, and how to react to them in a more adaptive manner.

It should be noted that results for women with panic disorder were in the predicted direction and support Clark's (1993) original theory as it pertains to panic. Clark (1993) predicted that individuals with panic disorder would interpret ambiguous internal sensations as threatening. Women in the Panic only group reported more negative

interpretations of bodily sensations than controls, more fear of respiratory and cognitive dyscontrol symptoms, and focused more on bodily sensations than women without panic disorder. These results clearly support Clark's cognitive theory of panic disorder.

### Criterion Two

Criterion Two stated that thoughts based on the misinterpretation of bodily sensations accompany challenge induced asthma symptoms or attacks. Criterion Two was not supported in relation to individuals with asthma only, but was supported as it relates to individuals with panic disorder. In addition, increases in panic-related cognitions for the Panic only and Asthma and Panic groups were significantly greater than those produced by the relaxation task. This is consistent with previous research that suggests that for individuals with panic disorder, experimentally induced bodily sensations are accompanied by self-reports of increased fearful cognitions (e.g., Rachman et al., 1988).

A number of reasons may have contributed to the lack of significant findings for women with asthma only. It may be that participants were aware of the source of the sensations (i.e., that the sensations were induced by the task, not an unknown internal source) and were less likely to misinterpret them as being catastrophic in nature. Sensations that are produced in the laboratory, with an experimenter in the room, might not be as frightening as if they are experienced "out of the blue" and with no one else nearby.

It is also possible that the tasks were not salient enough to produce sensations that these women fear or are likely to misinterpret. Of the 60 women who participated in the study, only two stopped the tasks before the 2 minute time period was completed. Both

participants had been diagnosed with panic disorder (one in the Asthma and Panic group, one in the Panic only group) and were unable to complete either the head turning or straw task. Given that half the sample had been diagnosed with panic disorder, and therefore the tasks should have been extremely aversive, it is surprising that nearly all women were able to complete the tasks. However, not all individuals with panic disorder fear the same sensations. During the exposure component of cognitive-behavior therapy for panic disorder, tasks that are specific to sensations that an individual fears are selected as exposure assignments (Barlow & Craske, 1994). It may be that the head turning and straw tasks produced sensations that were simply not feared by women in this study. Future research in this area could investigate tailoring tasks used for exposing participants to the individual, rather than expecting that any single task will induce panic-related cognitions and sensations in all participants.

### Criterion Three

The third criterion stated that manipulations of cognitive variables have an influence on whether or not an individual experiences asthma symptoms, increased air flow resistance, or an asthma attack during a challenge task. This criterion was not directly examined. However, as discussed in Chapter 1, previous research has supported this hypothesis both for individuals with panic disorder and individuals with asthma only. Manipulation of cognitions for individuals with panic disorder leads to increased likelihood of panic symptoms or attacks during panic challenge tasks (e.g., Margraf, 1993; Salkovskis & Clark, 1990; van der Molen et al., 1986).

### Conclusions about Cognitive Theory

Overall, both of the criteria proposed by Clark (1993) that were extended to asthma and tested in this study were not supported. It appears that women who have asthma and no history of panic disorder do not interpret bodily sensations more negatively than women who do not have asthma, nor do they focus more on bodily sensations, or report more fear of these sensations than women without asthma. Women with asthma did not report more catastrophic cognitions during the tasks than women without asthma. It should be noted, however, that women with panic disorder (with or without asthma) reported more fear of bodily sensations, more bodily self-focus, and at baseline, more panic-related cognitions than the other groups. In addition, the head turning and straw tasks produced greater increases in panic-related cognitions than the relaxation task for the Panic only and Asthma and Panic groups. These results are consistent with previous research and Clark's (1993) original theory.

### Dyspnea/Suffocation Fear Theory

In testing Ley's (1989) dyspnea/suffocation fear theory, four aspects of the theory were addressed. First, the relaxation task represented an attempt to replicate the findings of Carr et al. (1992). In the Carr et al. (1992) study, individuals with asthma only and individuals with panic disorder reported similar levels of dyspnea and similar levels of psychopathology. The authors noted that variance in panic symptoms in individuals with asthma is better accounted for by dyspnea scores compared to individuals with panic disorder. In other words, panic symptoms in asthma may be due to dyspnea, but in panic disorder, they are not due to dyspnea levels.

It was predicted that women in the Asthma only, Panic only, and Asthma and Panic groups would report similar levels of change in dyspnea scores from baseline to after the relaxation task. These levels were predicted to be significantly higher than those reported by women in the Control group. All four groups reported similar levels of dyspnea both at baseline and after the relaxation task. However, when within-subjects differences were examined, the straw task produced the greatest increase in dyspnea reports, followed by the head turning task for all four groups. The relaxation task produced the smallest changes in dyspnea reports. These results suggest that the straw task did produce increased dyspneic sensations, but that all groups, including the Control group, reported similar levels of dyspnea after the tasks.

A number of differences between the Carr et al. (1992) study and the present study may have led to the discrepant between-groups results for the relaxation task. In the previous study, participants recruited from the community included both males and females with an average age of 45. Furthermore, in the Carr et al. (1992) study, participants were seated in a reclining chair with the lights off for the relaxation task. In the present study, participants sat upright in the same chair that was used for the other tasks, and lights remained on during the relaxation task. It is possible that sample and methodological differences contributed to the lack of significant findings for the relaxation task in the present study.

Women in the three experimental groups were expected to report significantly greater dyspnea levels after the head turning task in comparison to controls. Although between-group differences were not found for the head turning task, it is clear that the task itself did produce an increase in dyspnea reports relative to baseline for all groups.



Due to their unfamiliarity with the sensation, women in the Control group were expected to report more dyspnea during the straw task than women in the other three groups. However, no significant group differences were identified. Within-subjects analyses revealed that for all four groups, the straw task produced the greatest increase in dyspnea relative to the other two tasks.

There are several possible reasons that may have contributed to the lack of between-groups differences in the present study. It may be that individuals who have not experienced dyspnea on a regular basis (as individuals with asthma or panic disorder might) pay less attention to the sensation of dyspnea. As noted above, women in both of the panic disorder groups focus more on bodily sensations and might therefore be expected to notice more subtle changes in dyspnea level in comparison to controls. Again, examination of dyspnea change scores for the straw task shows that all four groups reported an increase in dyspnea during the straw task in comparison to the other two tasks. The task itself may have been strong enough to produce uniform changes in perceived dyspnea. Future research should use more than one task specifically designed to induce dyspneic sensations and compare self-reports across these tasks, rather than relying on one task that appears to produce similar changes in all participants.

A fourth hypothesis testing the dyspnea/suffocation fear theory suggested that differences would be identified in panic sensation levels after each task. The head turning and relaxation tasks did not produce changes in panic symptoms. After the straw task, women in the Panic only group reported more hyperventilation symptoms than women in the Asthma only and Control groups, and women in the Asthma and Panic group reported more bronchoconstriction than women in the Asthma only group. These results may

appear to conflict with the findings discussed above, where no group differences in dyspnea levels were identified. However, examination of the measures used may explain the differences. The Borg scale is a 0-10 Likert scale that asks individuals to rate the amount of difficulty breathing that they experienced during the task. The hyperventilation and bronchoconstriction subscales of the ASC ask about much more specific symptoms (i.e., “shallow breathing,” “tingly in spots,” “chest tightening,” “mucous congestion”). When asked to rate difficulty breathing in general, participants may tend to minimize their symptoms, or not think of symptoms like “shallow breathing,” as constituting “difficulty breathing”. When participants are asked specifically about ways their breathing may be impaired, this may elicit increased self-reports of hyperventilation and bronchoconstriction.

These results suggest that it is important for future research to use more specific measures of breathing difficulty and dyspnea than the Borg scale. Respiratory symptoms such as perceived hyperventilation and bronchoconstriction are hypothesized to play an important role in the onset of panic attacks (Ley, 1989). Therefore, it is particularly important to use measures of specific respiratory symptoms rather than a global rating of breathing difficulty when studying individuals with both asthma and panic disorder.

When within-subjects differences were examined for the Bronchoconstriction subscale, it was evident that the straw task produced the greatest increase, followed by the head turning task, and lastly the relaxation task. These findings applied to the Asthma only, Panic only, and Asthma and Panic groups. Women in the Control group exhibited a slightly different pattern, with the straw task producing the largest increase in self-reported bronchoconstriction in comparison to the head turning and relaxation tasks,

which were not significantly different from each other. These results are very similar to those for the dyspnea self-reports. Furthermore, examination of within-subjects differences on sensations specific to panic, the Asthma only, Panic only, and Asthma and Panic groups reported equivalent levels of change from baseline for the head turning and straw tasks. The levels of change reported for both the head turning and straw task were equivalent for these groups. This finding indicates that the head turning and straw tasks did indeed produce changes in panic sensation levels, but that these changes did not necessarily differ across groups.

Overall, the between-groups hypotheses related to the dyspnea/suffocation theory were generally not supported in this study. Dyspnea levels did not differ for any of the groups at baseline or after any of the tasks. Self-reports of asthma-related and panic sensations were greater for women in the Panic only group at baseline. No significant differences between groups were identified for the head turning or relaxation tasks on bodily sensations measures. After the straw task, however, women with panic disorder only reported more hyperventilation symptoms than women in the Asthma only and Control groups. In addition, women in the Asthma and Panic group reported more bronchoconstriction during the straw task than women with asthma only. Therefore, the hypothesis put forth by Carr et al. (1992) that suggested that the high rate of concordance of panic disorder in individuals who have respiratory disorders may be due to the experience of dyspnea was not supported by the results of the present study.

Arguments by Ley (1992a; 1992b) regarding using individuals who meet DSM criteria for panic disorder also apply to the present study. Ley notes that his theory applies to individuals who experience hyperventilatory panic attacks only. The use of

DSM-IV criteria rather than Ley's criteria for hyperventilatory panic attacks in selecting participants in the present study may account for the lack of significant group differences. Selection of participants who have hyperventilatory panic attacks for studies similar to the present study might lead to results that would be more in line with the tenets of dyspnea/suffocation fear theory.

### Carr's Hypothesis

Carr et al. (1996) conducted an experiment that involved having individuals with asthma, panic disorder, or both disorders engage in baseline and stressful tasks. The authors found that at baseline, individuals with asthma had more difficulty breathing than individuals with both asthma and panic disorder. Furthermore, a nonsignificant trend was identified that suggested that individuals with both asthma and panic disorder experienced improved lung function after a stressful task. Thus, Carr (1998) concluded that it is possible that having both asthma and panic disorder may actually improve lung function and in some cases may serve as a protective function for individuals with asthma.

The present study attempted to replicate the findings of Carr et al. (1996) by measuring lung function after each task. However, no between-groups differences after any of the tasks were identified for the spirometry measures. Similarly, when within-subjects analyses were conducted, no differences in FEV<sub>1</sub> change scores were found for any of the groups. For FVC however, the Asthma and Panic group's change scores for the relaxation task were greater than for the straw task. There were no significant differences for the straw and head turning tasks nor for the head turning and relaxation tasks. These results indicate that women in the Asthma and Panic group experienced a

decrease in lung function after the straw task in comparison to the relaxation task. This finding directly contradicts Carr's hypothesis that having both asthma and panic disorder serves as a protective factor.

For the ratio of  $FEV_1/FVC$ , no within-subjects differences were found for the Asthma only, Asthma and Panic, and Control groups. However, women in the Panic only group experienced decreased lung function during the head turning task compared to the straw task. There were no significant differences between the head turning and relaxation tasks nor for the straw and relaxation tasks for this group. This finding suggests that for the Panic group, the straw task produced greater lung obstruction than did the head turning task.

It must be noted that the problems with the reliability of the spirometry readings led to the need for different analyses than were originally proposed. Because many of the participants' spirometric efforts produced readings that were less reliable than would be ideal, the variability readings for the spirometry measurements were used as covariates rather than excluding measurements that did not fall within the cutoff for reliability. Future research should utilize much larger samples than was possible in the present study, and ensure as much as possible that spirometry efforts are consistent. Furthermore, the type of lung function measurement equipment used in these two studies was not the same. Carr et al. (1996) used forced oscillation pneumography, whereas the present study utilized spirometry. This difference in equipment may have led to disparate findings. Spirometry involves 3 separate blasts of air after the task and is effort-dependent. Forced oscillation pneumography can continuously monitor breathing via a mouthpiece, and is not effort-dependent. It is possible that use of forced oscillation pneumography might

have identified brief alterations in lung function during the tasks, and it would have eliminated the problems with participant effort that the spirometry caused.

Interestingly, there have been no published studies to date that clearly confirm Carr's hypothesis that having both asthma and panic disorder may lead to better lung function. Dorhofer & Sigmon (in press) measured participants' lung function after they listened to neutral and asthma-related audiotaped scenes. In that study, peak air flow measurements were taken with a simple plastic device. The authors found a non-significant trend that suggested that having both asthma and panic attacks might protect against a drop in peak flow experienced by individuals with asthma only. It is also possible that Carr's original finding was an anomaly that is not replicable in further research. However, studies that replicate these findings are needed before a definitive statement is made regarding the possible beneficial effects of panic attacks or panic disorder on the lung function of individuals diagnosed with asthma.

#### Study Limitations

Numerous limitations to the present study are noteworthy. The small sample size of 60 participants across four groups may have led to a lack of significant findings for some analyses. Comparisons between subsets of individuals within each group were not possible because of the small sample size. Although several reasons led to the use of women only in this study, the lack of men in this sample limits the generalizability of study results. Future research should make a point of comparing groups of men and women who suffer from both asthma and panic disorder.

The use of primarily Caucasian undergraduates as experimental participants raises various concerns in and of itself. It is unclear how generalizable these results are to other

populations such as males, middle-aged individuals, the elderly, or minority groups. Research has indicated that changes in the prevalence of both asthma and panic disorder occur with age (Dodge & Burrows, 1980; Pearlman & Bierman, 1988). In particular, following women as they go through menopause and documenting how their asthma and/or panic disorder symptoms change will be important for future research to investigate.

One serious limitation is that for some individuals with asthma, simply taking a deep breath before exhaling into a spirometer can cause short-lived lung function decreases (Gayrard et al., 1975; Gayrard et al., 1979; Orehek et al., 1975). Unfortunately, spirometry is the most widely used, least expensive, and least intrusive of a number of measures of lung function. Until alternative ways of measuring lung function become more affordable and less intrusive, the use of spirometry is likely to continue.

The tasks that were used in the present study may not have been salient enough to lead to increases in anxiety for the majority of the participants. It is also possible that the tasks were either too long or too short to produce an anxiety experience that would lead to group differences. For example, if the tasks were too short, the threshold for anxiety may not have been reached. If the tasks were too long, participants may have habituated to the sensations. Anecdotally, several participants mentioned that the straw task bothered them when they first began the task, but after a few seconds they relaxed and became less anxious as they became used to the sensations. Using a smaller straw (a coffee stirrer rather than a drinking straw) may also lead to an increase in sensations and anxiety during this task. In addition, standardizing the speed at which participants turn their heads

during the other experimental task could ensure that the task was more consistent between participants.

### Future Directions

It is only within the last 10 years that interest has emerged in researching the effects of comorbidity of asthma and panic disorder. Therefore, there are numerous areas that future research can and should address. Many of these have been discussed above, but deserve additional attention here.

First, the relation between the anxiety sensitivity construct and the onset and maintenance of panic disorder in individuals with and without asthma needs to be examined more thoroughly. Conflicting findings have been reported regarding whether or not individuals with asthma only report higher levels of anxiety sensitivity than controls. Further study is required with a more demographically diverse sample than the present study used to help shed light on this issue. If individuals with asthma only do indeed report higher levels of anxiety sensitivity, longitudinal studies could examine the rates with which high anxiety sensitivity individuals with asthma develop panic disorder. The onset and course of panic disorder in individuals with asthma has not been investigated to date. Longitudinal studies will be able to address this issue as well.

Treatment for individuals with comorbid asthma and panic disorder represents another important area for future research. The results of the present study suggest that using panic challenge tasks with these individuals will not be detrimental to lung function in the short term. However, it remains unknown how effective and safe behavioral treatments are for use with individuals with asthma over the long term.



In general, the results of the present study indicate that having asthma and panic disorder leads to increases in trait anxiety, self-focus on bodily sensations, fear of bodily sensations, and anxiety sensitivity (on one of two measures of anxiety sensitivity) in comparison to controls or individuals with asthma only. Cognitive theory was supported for individuals in both panic groups. Finally, the head turning and straw tasks were successful in producing increases in anxious mood, increases in panic and asthma sensations, self-reported dyspnea, and panic related cognitions in comparison to the relaxation task.

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## APPENDIX A

## Power Analysis

Power analyses were calculated based on statistics presented in ~~Carr~~ et al. (1996):

$$F(3,109) = 20.77$$

$$k = 4$$

$$k*n = 109$$

$$w^2 = \frac{(k - 1)(F - 1)}{(k - 1)(F - 1) + kn}$$

$$w^2 = \frac{(4 - 1)(20.77 - 1)}{(4 - 1)(20.77 - 1) + 109}$$

$$w^2 = .352$$

$$\Phi = \frac{n' w^2}{1 - w^2}$$

$$\Phi = \frac{n' .352}{.648}$$

$$\Phi = .737 \quad n'$$

Substituting 15 participants per cell in for  $n'$ ,  $\Phi = 2.85$ . Looking these values up on charts from Pearson and Hartley (1951), a power value of .94 is obtained.

## APPENDIX B

Asthma and Anxiety  
Informed Consent Form I

I have been asked to participate in this study because I am at least 18 years old and responded to media recruitment efforts for a study on asthma and anxiety. The purpose of this study is to gain more information about the relation between psychological events and physical health. My participation in this study will help further the understanding of the psychological processes involved in asthma and anxiety disorders. First, I will undergo a psychological assessment to see if I qualify for the experiment. This assessment will involve completion of a diagnostic interview (e.g., Do you currently have times when you feel a sudden rush of intense fear or discomfort?). If I qualify, I will participate in an experimental session. During this session, I will complete some questionnaires. If a question makes me feel uncomfortable, I don't have to answer it. I will be asked to respond to various questions assessing:

- How I feel about various bodily sensations (e.g., "It scares me when I feel faint", "Your heart is pounding")
- Symptoms of asthma that I may experience (e.g., "Coughing," "Chest congestion")
- My mood (e.g., "I do not feel sad," "I am worried")
- My interpretations of everyday situations

In addition to the interview and questionnaires, I will complete three tasks. In one task, I will briefly turn my head from side to side. In another task, I will breathe through a straw for a short period of time. A third task involves relaxing. Between these tasks, I will complete some questionnaires and have my lung function measured. Lung function will be assessed by a spirometer. I will be asked to take a deep breath and then forcefully exhale into a device that measures how well my lungs are working.

I understand that I will be paid for my participation in this study. If I qualify, I will receive \$10 for the interview and experimental session, which will require a maximum of 2 hours.

The risks associated with this project are minimal. However, if I experience any distress associated with completing questionnaires or the experimental session, I may speak with a licensed psychologist (Dr. Sandy Sigmon, 207-581-2052). I may also contact Dr. Sigmon long-distance by calling her collect. If I want more information about this study, I may contact the experimenter, Diana Dorhofer, at 581-2031 (301 Little Hall, University of Maine, Orono, ME **04469**). If I live long-distance from the University and have a question for Diana, I may call Dr. Sigmon collect and leave a message for her. At any point in the study, I may request a referral for treatment. I may terminate my participation at any point without penalty. If I desire, a *summary* of the results of this

project will be made available to me at the end of the study. If I wish to receive a summary of the results, I may indicate so at the bottom of this form.

My responses will remain anonymous and all of the information I provide will be stored in a locked laboratory. My participation in this study is strictly voluntary and I may withdraw at any time without penalty.

I acknowledge that I have received a copy of this consent form.

Participant's signature: \_\_\_\_\_ Date: \_\_\_\_\_

Printed name: \_\_\_\_\_ Phone: \_\_\_\_\_

Please indicate if you would like a summary of the results of this study:

\_\_\_\_\_ yes \_\_\_\_\_ no

If yes, please write your permanent mailing address below:

## Asthma and Anxiety Informed Consent Form II

I have been asked to participate in this study because I am at least 18 years old and responded to media recruitment efforts for a study on asthma and anxiety. The purpose of this study is to gain more information about the relation between psychological events and physical health. My participation in this study will help further the understanding of the psychological processes involved in asthma and anxiety disorders. First, I will undergo a psychological assessment to see if I qualify for the experiment. This assessment will involve completion of a diagnostic interview (e.g., Do you currently have times when you feel a sudden rush of intense fear or discomfort?). If I qualify, I will participate in an experimental session. During this session, I will complete some questionnaires. If a question makes me feel uncomfortable, I don't have to answer it. I will be asked to respond to various questions assessing:

- How I feel about various bodily sensations (e.g., "It scares me when I feel faint", "Your heart is pounding")
- Symptoms of asthma that I may experience (e.g., "Coughing," "Chest congestion")
- My mood (e.g., "I do not feel sad," "I am worried")
- My interpretations of everyday situations

In addition to the interview and questionnaires, I will complete three tasks. In one task, I will briefly turn my head from side to side. In another task, I will breathe through a straw for a short period of time. A third task involves relaxing. Between these tasks, I will complete some questionnaires and have my lung function measured. Lung function will be assessed by a spirometer. I will be asked to take a deep breath and then forcefully exhale into a device that measures how well my lungs are working.

I understand that I will receive experimental credit for my participation in this study. If I qualify, I will receive 2 credits for the interview and experimental session, which will require a maximum of 2 hours. If I do not qualify, I will receive 1 credit for the interview portion of the study.

The risks associated with this project are minimal. However, if I experience any distress associated with completing questionnaires or the experimental session, I may speak with a licensed psychologist (Dr. Sandy Sigmon, 207-581-2052). I may also contact Dr. Sigmon long-distance by calling her collect. If I want more information about this study, I may contact the experimenter, Diana Dorhofer, at 581-2031 (301 Little Hall, University of Maine, Orono, ME 04469). If I live long-distance from the University and have a question for Diana, I may call Dr. Sigmon collect and leave a message for her. At any point in the study, I may request a referral for treatment. I may terminate my participation at any point without penalty. If I desire, a *summary* of the results of this project will be made available to me at the end of the study. If I wish to receive a summary of the results, I may indicate so at the bottom of this form.

My responses will remain anonymous and all of the information I provide will be stored in a locked laboratory. My participation in this study is strictly voluntary and I may withdraw at any time without penalty.

I acknowledge that I have received a copy of this consent form.

Participant's signature: \_\_\_\_\_ Date: \_\_\_\_\_

Printed name: \_\_\_\_\_ Phone: \_\_\_\_\_

Please indicate if you would like a summary of the results of this study:

\_\_\_\_\_ yes \_\_\_\_\_ no

If yes, please write your permanent mailing address below:

## APPENDIX C

## Physician Release of Information

Physician name: \_\_\_\_\_

Physician address: \_\_\_\_\_  
\_\_\_\_\_

I, \_\_\_\_\_ have participated in an experiment at the University of Maine that examines psychological factors that may influence asthma symptoms. This experiment requires that participants have been diagnosed with asthma. Please complete the following questions and return this form in the enclosed self-addressed stamped envelope. I give permission for you to release information regarding my diagnosis of asthma only to Diana Dorhofer, the primary experimenter for this study.

\_\_\_\_\_  
Participant signature\_\_\_\_\_  
Date\_\_\_\_\_  
Print name

Please complete the following questions:

Has \_\_\_\_\_ been diagnosed with asthma? YES \_\_\_\_\_ NO \_\_\_\_\_

At approximately what age was she diagnosed? \_\_\_\_\_

What medications have you prescribed for her for her asthma symptoms (please include dosage information)? \_\_\_\_\_  
\_\_\_\_\_Please rate the severity of her asthma: Mild \_\_\_\_\_ Moderate \_\_\_\_\_  
Severe \_\_\_\_\_Is her asthma: Well-controlled \_\_\_\_\_ Moderately well-controlled \_\_\_\_\_  
Not controlled well \_\_\_\_\_

Thank you for completing this information. Please return this form in the enclosed envelope. If you have any questions, please contact Diana Dorhofer (207-581-2031), 301 Little Hall, Orono, ME **04469**.



## APPENDIX D

## Anxiety Sensitivity Index

Respond to each item by indicating the number of the phrase which best represents the extent to which you agree with the item. If any of the items addresses something that is not part of your experience (i.e., "it scares me when I feel shaky" for someone who has never trembled or had the "shakes"), answer on the basis of how you think you might feel if you had such an experience. Otherwise, answer all items on the basis of your own experience. Be careful to make only one choice for each item and please answer all items.

0 = Very little

1 = A little

2 = Some

3 = Much

4 = Very much

- \_\_\_ 1. It is important to me not to appear nervous
- 2. When I cannot keep my mind on a task, I worry that I might be going crazy
- 3. It scares me when I feel "shaky" (trembling)
- 4. It scares me when I feel faint
- 5. It is important to me to stay in control of my emotions
- 6. It scares me when my heart beats rapidly
- \_\_\_ 7. It embarrasses me when my stomach growls
- 8. It scares me when I am nauseous
- 9. When I notice that my heart is beating rapidly, I worry that I might have a heart attack
- 10. It scares me when I am short of breath
- 11. When my stomach is upset, I worry that I might be seriously ill
- 12. It scares me when I am unable to keep my mind on a task
- 13. Other people notice when I feel shaky
- 14. Unusual body sensations scare me
- 15. When I am nervous, I worry that I might be mentally ill
- \_\_\_ 16. It scares me when I am nervous

## APPENDIX E

## Anxiety Sensitivity Profile

Instructions: It is very important that you read these instructions carefully so that you will be able to answer the questions that follow. The purpose of this questionnaire is to measure your level of fear of anxiety-related sensations. There are many anxiety-related sensations, including the following: palpitations (pounding heart or accelerated heart rate), sweating, trembling, shortness of breath, chest pain or discomfort, nausea, dizziness, feelings of unreality, chills, and hot flashes. People differ in their fears of these sensations: some people have little or no fear, others have mild or moderately severe fears, while others have very strong fears.

Anxiety sensations are feared if a person believes that these sensations have bad consequences. For example, people are frightened of palpitations if they believe these sensations could lead to a heart attack. People are frightened of dizziness if they believe that this sensation could mean that they are going crazy. People are frightened of publicly observable anxiety reactions (e.g., blushing or trembling) if they believe these reactions could cause others to ridicule or reject them.

We would like you to do two things for each of the items on the following pages:

1. Imagine that you are experiencing the sensation. Try to imagine this as vividly **as** possible.
2. Using the scale provided, rate the likelihood that if **YOU** experienced the sensation, it would lead to something bad happening to you, such **as** dying, going crazy, losing control, or being ridiculed or rejected by others. There are no right or wrong answers, and all responses will remain anonymous. Please note: We are not assessing whether or not you experience these sensations **as** a result of being anxious. We want to assess whether you believe that anxiety-related sensations would lead to something bad happening to you.

Practice item:

Imagine that you're experiencing the following sensation. What is the likelihood that this sensation would **LEAD** to something **BAD** happening to **YOU**? Circle the number that best indicates your choice:

	Not at all likely			Somewhat likely			Extremely likely	
0. Your legs feel unsteady	1	2	3	4	5	6	7	

Before you answer the following questions, please place a check mark here if you fully understand the instructions you have read: \_\_\_\_\_

If you don't understand the instructions, please ask for clarification.

What is the likelihood that this sensation would LEAD to something BAD happening to YOU?

1 = Not at all likely

2

3 = Somewhat likely

4

5 = Extremely likely

- \_\_\_\_\_ 1. Your heart is pounding
- \_\_\_\_\_ 2. Your thoughts seem slower than usual
- \_\_\_\_\_ 3. You feel like you can't take a deep breath
- \_\_\_\_\_ 4. Your stomach is making loud noises
- \_\_\_\_\_ 5. You have tingling sensations in your hands
- \_\_\_\_\_ 6. You have pain in your chest
- \_\_\_\_\_ 7. Your thoughts seem jumbled
- \_\_\_\_\_ 8. Your heart is beating so loud that you can hear it
- \_\_\_\_\_ 9. You feel like you're in a fog
- \_\_\_\_\_ 10. Hot flushes sweep over you
- \_\_\_\_\_ 11. You have diarrhea
- \_\_\_\_\_ 12. You are "jumpy" or easily startled
- \_\_\_\_\_ 13. You keep getting distracted by unwanted thoughts
- \_\_\_\_\_ 14. Your heart beats rapidly
- \_\_\_\_\_ 15. You feel like you're suffocating
- \_\_\_\_\_ 16. You have a knot in your stomach
- \_\_\_\_\_ 17. You feel numb all over
- \_\_\_\_\_ 18. Thoughts seem to race through your mind
- \_\_\_\_\_ 19. You feel out of breath even though you haven't been exerting yourself
- \_\_\_\_\_ 20. Your heart pounds in your ears
- \_\_\_\_\_ 21. You feel like something is stuck in your throat
- \_\_\_\_\_ 22. Your body feels strange or different in some way
- \_\_\_\_\_ 23. Your face sweats even though you're not hot
- \_\_\_\_\_ 24. Your voice quavers (trembles or sounds shaky)
- \_\_\_\_\_ 25. You can't keep your mind on a task
- \_\_\_\_\_ 26. You have difficulty swallowing
- \_\_\_\_\_ 27. Your stomach aches
- \_\_\_\_\_ 28. You have burning sensations in your chest (heartburn)
- \_\_\_\_\_ 29. Familiar surroundings seem strange or unreal to you
- \_\_\_\_\_ 30. You feel like you're choking
- \_\_\_\_\_ 31. You feel your heartbeat pulsing in your neck
- \_\_\_\_\_ 32. You are constipated
- \_\_\_\_\_ 33. You feel faint or lightheaded
- \_\_\_\_\_ 34. Your heart starts beating slower
- \_\_\_\_\_ 35. You shiver even though you're not cold
- \_\_\_\_\_ 36. You have trouble thinking clearly
- \_\_\_\_\_ 37. You feel that there's a lump in your throat
- \_\_\_\_\_ 38. You feel like you're about to vomit

- 1 = Not at all likely  
2  
3 = Somewhat likely  
4  
5 = Extremely likely

- \_\_\_\_\_ 39. You're awake but you feel like you're in a daze
- \_\_\_\_\_ 40. Your stomach is upset
- \_\_\_\_\_ 41. You have trouble remembering things
- \_\_\_\_\_ 42. Your heart beats erratically
- \_\_\_\_\_ 43. You have tingling sensations in your lips
- \_\_\_\_\_ 44. Your mind goes blank
- \_\_\_\_\_ 45. Your throat feels tight
- \_\_\_\_\_ 46. You feel "spacey" or spaced out
- \_\_\_\_\_ 47. You feel like you're not getting enough air
- \_\_\_\_\_ 48. Your face blushes red
- \_\_\_\_\_ 49. You feel bloated (gassy)
- \_\_\_\_\_ 50. You feel sick in your stomach (nausea)
- \_\_\_\_\_ 51. Your heart skips a beat
- \_\_\_\_\_ 52. Your face feels numb
- \_\_\_\_\_ 53. The muscles in your face twitch
- \_\_\_\_\_ 54. You are easily distracted
- \_\_\_\_\_ 56. You have difficulty concentrating
- \_\_\_\_\_ 57. You have to urinate more frequently than usual
- \_\_\_\_\_ 58. Your hands are trembling
- \_\_\_\_\_ 59. You feel like you can't breathe properly
- \_\_\_\_\_ 60. You feel like things are spinning around you

## APPENDIX F

## Asthma Symptom Checklist

Please rate the following symptoms you may experience when you are having an asthma attack.

1= Never  
 2= Rarely  
 3= Sometimes  
 4= Frequently  
 5= Always

- |                                 |                             |
|---------------------------------|-----------------------------|
| _____ 1. Shallow breathing      | _____ 19. Worried           |
| _____ 2. Choking                | _____ 20. Headache          |
| _____ 3. Pins & needles feeling | _____ 21. Afraid of dying   |
| _____ 4. Anxious                | _____ 22. Itchy skin        |
| _____ 5. Edgy                   | _____ 23. Panicky           |
| _____ 6. Coughing               | _____ 24. Short of breath   |
| _____ 7. Itchy throat           | _____ 25. Chest filling up  |
| _____ 8. worn out               | _____ 26. Chest congestion  |
| _____ 9. Frustrated with things | _____ 27. Mucous congestion |
| _____ 10. Worried about myself  | _____ 28. Cranky            |
| _____ 11. Worried about attack  | _____ 29. Numb              |
| _____ 12. Dizzy                 | _____ 30. Fatigued          |
| _____ 13. Itchy lungs           | _____ 31. Irritable         |
| _____ 14. Nauseated             | _____ 32. Rapid breathing   |
| _____ 15. No energy             | _____ 33. Chest tightening  |
| _____ 16. Tingly in spots       | _____ 34. Frightened        |
| _____ 17. Scared                | _____ 35. Weak              |
| _____ 18. Short tempered        | _____ 36. Hard to breathe   |



## APPENDIX H

## Borg Category Scale

Please rate the amount of difficulty breathing that you experienced during the task you just completed.

- 0 Nothing at all
- 0.5 Very, very slight (just noticeable)
- 1 Very slight
- 2 Slight
- 3
- 4
- 5 Moderate
- 6 Somewhat severe
- 7 Severe
- 8 Very severe
- 9 Very, very severe (almost maximal)
- 10 Maximal

## APPENDIX I

## Brief Body Sensations Interpretation Questionnaire

Here are some outline descriptions of situations in which it is not quite clear what is happening. Read each one, and then answer the question below it very briefly. Write down the first thing that comes into your mind without thinking too long about it. Please write what you think is happening before you turn over the page. Be as specific as possible.

When you have done that, turn the page over and you will see three possible explanations for the situation. Arrange these in the order in which they would most likely come to your mind if you found yourself in a similar situation. So the one that you would consider most likely to be true should come first, and the one that you would consider least likely to be true should come third. Do not think too long before deciding. We want your first impressions, and do not worry if none of them fits with what you actually did think.

1a. You have visitors over for a meal and they leave sooner than you expected.

Why?

- 1b. a) They did not wish to outstay their welcome.
- b) They had another pressing engagement to go to.
- c) They did not enjoy the visit and were bored with your company.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

2a. You feel short of breath.

Why?

- 2b. a) You are developing the flu.
- b) You are about to suffocate or stop breathing.
- c) You are physically "out of shape."

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

3a. Your vision has become slightly blurred.

Why?



- 3b. a) You have strained your eyes slightly.  
 b) You need to get glasses or change your existing glasses.  
 c) This is a sign of a serious illness.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 4a. You go into a shop and the clerk ignores you.

Why?

- 4b. a) They are bored with their job, and this makes them rude.  
 b) They are concentrating very hard on something else.  
 c) They find you irritating and resent your presence.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 5a. You feel lightheaded and weak.

Why?

- 5b. a) You are about to faint.  
 b) You need to get something to eat.  
 c) You didn't get enough sleep last night.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 6a. You smell smoke.

Why?

- 6b. a) Your house is on fire.  
 b) Some food is burning.  
 c) Someone is smoking a cigarette.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 7a. A friend suggests that you change the way that you're doing a job in your own house.

Why?

- 7b. a) They are trying to be helpful.  
b) They think you're incompetent.  
c) They have done the job more often and know an easier way.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 8a. Your chest feels uncomfortable and tight.

Why?

- 8b. a) You have indigestion.  
b) You have a sore muscle.  
c) Something is wrong with your heart.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 9a. You wake with a start in the middle of the night, thinking you heard a noise, but all is quiet.

What woke you up?

- 9b. a) You were woken by a dream.  
b) A burglar broke into your house.  
c) A door or window rattled in the wind.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 10a. You are introduced to someone at a party who fails to reply to a question you ask them.

Why?

- 10b. a) They did not hear the question.  
b) They think you are uninteresting and boring.  
c) They were preoccupied with something else at the time.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 11a. You notice your heart is beating quickly and pounding.

Why?

- 11b. a) Because you have been physically active.  
 b) Because there is something wrong with your heart.  
 c) Because you are feeling excited.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 12a. You suddenly feel confused and are having difficulty thinking straight.

Why?

- 12b. a) You are going out of your mind.  
 b) You are coming down with a cold.  
 c) You've been working too hard and need a rest.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 13a. A letter marked "URGENT" arrives.

What is in the letter?

- 13b. a) It is an advertisement designed to attract your attention.  
 b) You forgot to pay a bill.  
 c) News that someone you know has died or is seriously ill.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_

- 14a. You notice that your heart is pounding, you feel breathless, dizzy and unreal.

Why?

- 14b. a) You have been overdoing it and are overtired.  
 b) Something you ate disagreed with you.  
 c) You are dangerously ill or are going mad.

1<sup>st</sup> \_\_\_\_\_ 2<sup>nd</sup> \_\_\_\_\_ 3<sup>rd</sup> \_\_\_\_\_



## APPENDIX J

## General History

AGE \_\_\_\_\_ HEIGHT \_\_\_\_\_ WEIGHT \_\_\_\_\_ DATE \_\_\_\_\_ TIME \_\_\_\_\_

At what age were you diagnosed with asthma? \_\_\_\_\_

Have you been diagnosed with any other chronic illness? (e.g., diabetes, high blood pressure, etc.)

\_\_\_\_\_ yes \_\_\_\_\_ no

If yes, what illness? \_\_\_\_\_

What medications do you take, at what dosages, and how often? Please include birth control pills.

Do you take these medications as prescribed \_\_\_\_\_ or \_\_\_\_\_ less frequently  
\_\_\_\_\_ more frequently

Describe increase or decrease \_\_\_\_\_

How often do you have asthma attacks?

- \_\_\_\_\_ Once per day or more
- \_\_\_\_\_ Once per week or more
- \_\_\_\_\_ Once per month
- \_\_\_\_\_ Once per year
- \_\_\_\_\_ Less than once every couple of years

How often do you have asthma symptoms (e.g., wheezing, coughing, tightness of chest, mucus, etc.)?

- \_\_\_\_\_ Once per day or more
- \_\_\_\_\_ Once per week or more
- \_\_\_\_\_ Once per month
- \_\_\_\_\_ Once per year
- \_\_\_\_\_ Less than once every couple of years

When was your last asthma attack? \_\_\_\_\_

Is your asthma \_\_\_\_\_ exercise induced, \_\_\_\_\_ allergy induced, or \_\_\_\_\_ both?

If allergy induced, list things that trigger symptoms or **an** attack \_\_\_\_\_

During what months do you usually have asthma symptoms?

☐ All months

☐ January

☐ February

☐ March

☐ April

☐ May

☐ June

☐ July

☐ August

☐ September

☐ October

☐ November

☐ December

Do you use a peak flow meter at home? Y ☐ N ☐

If yes, how often do you use it? \_\_\_\_\_

If yes, what is your usual peak flow? \_\_\_\_\_

What sport(s) or other types of exercise do you regularly engage in, if any?

How often and for how long?

Do you smoke? Y ☐ N ☐ If yes, how many cigarettes per day? \_\_\_\_\_

Do you live with someone who smokes? Y ☐ N ☐

If yes, how many hours per day are you exposed to household smoke?

\_\_\_\_\_

## APPENDIX K

## Panic Attack Cognitions Questionnaire

Instructions: Using the scale below, rate each one of the following thoughts according to the degree to which you thought it during and after the task you just completed. Remember to rate each thought twice, once for during and once for after the task.

- 1 = Not at all  
 2 = Some, but not much  
 3 = Quite a lot  
 4 = Totally dominated your thoughts

	During
1. I am going to die.	_____
2. I am going insane.	_____
3. I am losing control.	_____
4. This will never end.	_____
5. I am really scared.	_____
6. I am having a heart attack.	_____
7. I am going to pass out.	_____
8. I don't know what people will think.	_____
9. I won't be able to get out of here.	_____
10. I don't understand what is happening to me.	_____
11. People will think I am crazy.	_____
12. I will always be this way.	_____
13. I am going to throw up.	_____
14. I must have a brain tumor.	_____
15. I will choke to death.	_____
16. I <del>am</del> going to act foolish.	_____
17. I am going blind.	_____
18. I will hurt someone.	_____
19. I am going to have a stroke.	_____
20. I am going to scream.	_____
21. I am going to babble or talk funny.	_____
22. I will be paralyzed by fear.	_____
23. Something is really physically wrong with me.	_____
24. I will not be able to breathe.	_____
25. Something terrible will happen.	_____

## APPENDIX L

## Profile of Mood States

Instructions: Below is a list of words that describe feelings that people have. Please read each one carefully. Then select one of the numbered descriptors that best describes how you feel right now. Place that number on the small line to the left of each word. Do not skip any items, and print your numbers clearly.

0 = Not at all  
 1 = A little  
 2 = Moderately  
 3 = Quite a bit  
 4 = Extremely

- |                                       |                       |
|---------------------------------------|-----------------------|
| _____ 1. Tense                        | _____ 13. Restless    |
| _____ 2. Unhappy                      | _____ 14. Discouraged |
| _____ 3. <i>Sorry</i> for things done | _____ 15. Nervous     |
| _____ 4. Shaky                        | _____ 16. Lonely      |
| _____ 5. Sad                          | _____ 17. Miserable   |
| _____ 6. On edge                      | <b>18.</b> Anxious    |
| _____ 7. Blue                         | _____ 19. Gloomy      |
| <b>8.</b> Panicky                     | _____ 20. Desperate   |
| _____ 9. Hopeless                     | _____ 21. Helpless    |
| _____ 10. Relaxed                     | _____ 22. Worthless   |
| _____ 11. Unworthy                    | _____ 23. Terrified   |
| _____ 12. Uneasy                      | _____ 24. Guilty      |



## APPENDIX M

## State-Trait Anxiety Inventory- State

Instructions: A number of statements which people have used to describe themselves are given below. Read each statement and then, using the scale below, place the number that indicates how you feel right now, that is, at this moment, in the blank before the number. 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.

1 = Not at all  
 2 = Somewhat  
 3 = Moderately so  
 4 = Very much so

1. I feel calm
2. I feel secure
3. I am tense
4. I feel strained
- 5. I feel at ease
6. I feel upset
7. I am presently worrying over possible misfortunes
8. I am satisfied
9. I feel frightened
10. I feel comfortable
11. I feel self-confident
12. I feel nervous
13. I am jittery
14. I feel indecisive
15. I am relaxed
16. I feel content
17. I am worried
18. I feel confused
19. I feel steady
20. I feel pleasant

## State-Trait Anxiety Inventory- Trait

Instructions: A number of statements which people have used to describe themselves are given below. Read each statement and then, using the scale below, place the number that indicates how you generally feel in the blank before the number. 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 how you generally feel.

1 = Almost never

2 = Sometimes

3 = Often

4 = Almost always

- \_\_\_ 21. I feel pleasant
- \_\_\_ 22. I feel nervous and restless
- \_\_\_ 23. I feel satisfied with myself
- 24. I wish I could be as happy as others seem to be
- 25. I feel like a failure
- 26. I feel rested
- 27. I am "calm, cool, and collected"
- 28. I feel that difficulties are piling up so that I cannot overcome them
- 29. I worry too much over something that really doesn't matter
- \_\_\_ 30. I am happy
- \_\_\_ 31. I have disturbing thoughts
- 32. I lack self-confidence
- \_\_\_ 33. I feel secure
- \_\_\_ 34. I make decisions easily
- 35. I feel inadequate
- 36. I am content
- 37. Some unimportant thought runs through my mind and bothers me
- 38. I take disappointments so keenly that I can't put them out of my mind
- \_\_\_ 39. I am a steady person
- \_\_\_ 40. I get in a state of tension or turmoil as I think over my recent concerns and interests

## APPENDIX N

## Symptom Checklist

Below is a list of symptoms that people experience. Please rate each item by writing the number in each slot that best describes how you felt **during** the task you just completed.

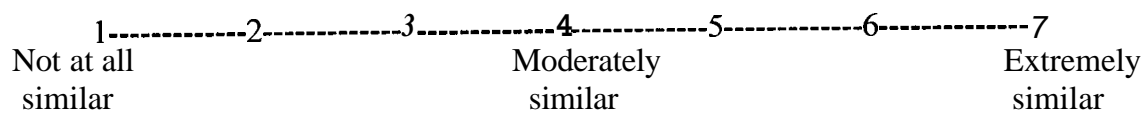
0 = None  
 1 = Mild  
 2 = Moderate  
 3 = Severe  
 4 = Very severe

- |   |   |
|---|---|
| _____ 1. Shallow breathing                                      | _____ 26. Worried   |
| _____ 2. Choking  | _____ 27. Headache  |
| _____ 3. Pins & needles feeling                                 | _____ 28. Afraid of dying                                 |
| _____ 4. Anxious  | _____ 29. Itchy skin                                      |
| _____ 5. Edgy   | _____ 30. Panicky   |
| _____ 6. Coughing   | _____ 31. Short of breath                                 |
| _____ 7. Itchy throat   | _____ 32. Chest filling up                                |
| _____ 8. worn out   | _____ 33. Chest congestion                                |
| _____ 9. Frustrated with things                                 | _____ 34. Mucous congestions                              |
| _____ 10. Worried about myself                                  | _____ 35. Cranky  |
| _____ 11. Worried about attack                                  | _____ 36. Numb  |
| _____ 12. Dizzy   | _____ 37. Fatigued  |
| _____ 13. Itchy lungs   | _____ 38. Irritable                                       |
| _____ 14. Nauseated   | _____ 39. Rapid breathing                                 |
| _____ 15. No energy   | _____ 40. Chest tightening                                |
| _____ 16. Tingly in spots                                       | _____ 41. Frightened                                      |
| _____ 17. Scared  | _____ 42. Weak  |
| _____ 18. Short tempered  | _____ 43. Hard to breathe                                 |
| _____ 19. Racing/pounding heart                                 | _____ 44. Trembling or shaking                            |
| _____ 20. Sweating  | _____ 45. Chest pain/discomfort                           |
| _____ 21. Hot flashes or chills                                 | _____ 46. Feeling that things aren't real                 |
| _____ 22. Fear of going crazy or<br>losing control              | _____ 47. Visual difficulties (blurred,<br>tunnel vision) |
| _____ 23. Hearing difficulties (ringing,<br>difficulty hearing) | _____ 48. Difficulty concentrating                        |
| _____ 24. Desire to escape or run out                           | _____ 49. Difficulty keeping calm                         |
| _____ 25. Feelings of embarrassment                             | _____ 50. Dry mouth                                       |

## APPENDIX O

## Visual Analog Panic Scale

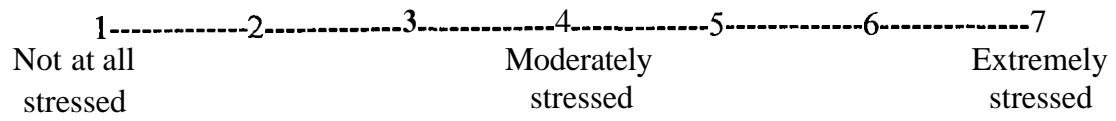
Please rate the similarity of the sensations you experienced during the task to those of a panic attack.



## APPENDIX P

## Visual Analog Stress Scale

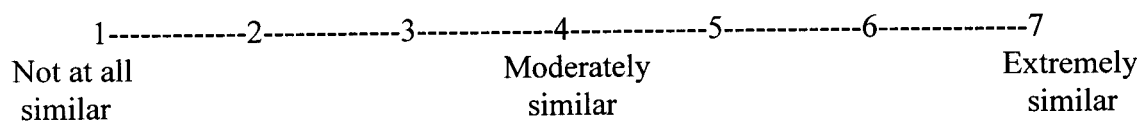
Please rate the level of stress you felt during the **task**.



## APPENDIX Q

## Visual Analog Asthma Scale

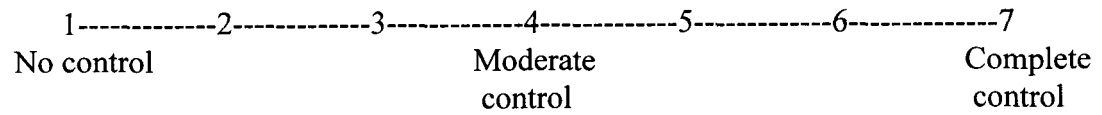
Please rate the similarity of the sensations you experienced during the task to those of **an** asthma attack.



## APPENDIX R

## Visual Analog Control Scale

Please rate how much you believed you had control over the sensations you experienced during the task.



## BIOGRAPHY OF THE AUTHOR

Diana Dorhofer was born in Everett, Washington and lived in numerous towns in Washington and Oregon as a child. She graduated from Seaside High School in 1991 and Oregon State University in 1995 with a B.A. in Psychology. In 1995, Diana entered the Clinical Psychology Doctoral Program at the University of Maine where she has been involved in research focusing on women's health and the relation between asthma and panic disorder. While working with Dr. Sandra Sigmon, Diana has co-authored 5 publications and has made numerous presentations to professional societies. Diana is a student member of the Association for the Advancement of Behavior Therapy, American Psychological Association, and the Society of Behavioral Medicine.

Diana completed her predoctoral internship at the VA Palo Alto Health Care System and will be staying on as a Behavioral Medicine postdoctoral fellow. Currently, she is extending her research to the treatment of individuals with comorbid respiratory diseases and panic disorder, and the impact of neuropsychological deficits on health behavior. Diana is a candidate for the Doctor of Philosophy degree in Psychology from The University of Maine in December, 2001.