2003

Depression, Anxiety, And Locus Of Control In Asthmatic Women

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DEPRESSION, ANXIETY, AND LOCUS OF CONTROL IN ASTHMATIC WOMEN

BY

ELIZABETH ANNE SEEBODE

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Submitted in Partial Fulfillment of the Requirements for the Degree Doctor of Philosophy Seton Hall University 2003
Acknowledgments

I am greatly indebted to a number of individuals for their contributions and support during my dissertation process. I would like to extend special appreciation to my mentor, Dr. Sandra Lee, as well as my committee members, Dr. Cheryl Thompson-Sard and Dr. Thomas Massarelli, for their extensive knowledge and dedicated efforts. Each in their own way was a tremendous source of encouragement, inspiration, and guidance. I would also like to express my gratitude to Dr. Adriana Dunn, who was extremely influential during the initial conceptual phase of my research.

For their contributions to the data collection, I am especially grateful to Dr. Joseph Atanasio, Dr. Mercy Co, Dr. Chris Kokinakos, Dr. Frank Mazzarella, Dr. Nader Moaven, and Dr. George Zazzali. Along with the physicians, I am also extremely thankful to the nurses and office staff who tirelessly distributed the research materials in the medical offices. Without their assistance, this research would not have been possible.

I would like to express my appreciation to Dr. Rudolph Richichi, for his expert statistical consultation and data analysis. He was truly invaluable in the interpretation and clarification of the results of this study. I would also like to extend
my gratitude to Ms. Carol Gernat for her flawless proofreading and assistance in the preparation of the final manuscript.

For their unwavering support and devotion, I am forever indebted to my parents, Drs. Anna and Joseph Seebode, my husband, Dr. Peter Zazzali, and my children, Peter Joseph and Jonathan. I would also like to express my appreciation to Ms. Julia T. Dunne, my source of spiritual inspiration.

Finally, I would like to thank the women who participated in this study. Their honesty and openness regarding their unique physical and emotional experiences with asthma were essential to the present investigation, and will undoubtedly contribute to future directions in the field of asthma research.
Dedication

This dissertation is dedicated to my father, Joseph J. Seebode, M.D., a surgeon and scholar, who has always believed that knowledge is power, intellect is freedom, and education is the only true possession.
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Chapter I

Introduction

Bronchial asthma is a complex, chronic respiratory disorder which afflicts millions of people every year. Though widely recognized as a reversible obstructive airway disease, it is poorly understood in terms of its causality, etiology, and attributions. Considerable evidence now exists to suggest that emotional factors play a role in asthma, and much of the current research in this area has focused on understanding the intricate relationship between psychological factors and the physical components of the disease. In fact, the delineation of the role of such emotional variables may ultimately provide not only greater understanding of how asthma develops, but also insights into the care and management of the illness process.

Depression and anxiety are two of the more prominent emotional variables which have been studied in relationship to asthma. Like many patients with chronic medical illness, asthmatics are felt to be at risk for depression (Carlin, 1998; Chaney et al., 1999; Galil, 2000) and patients reporting clinical levels of depression have been found in some cases to have significantly higher levels of asthma (Bell, Jasnoski, Kagan, & King, 1991). The cause of such depression is generally considered to be multifaceted, ranging from difficulty in handling symptoms of fatigue and disability (Lehrer, Feldman, Giardino, Song & Schmaling, 2002), to complications resulting from high doses of asthma medications (Thompson & Thompson, 1984). Loss and extreme disappointment have
also been associated with the acute onset of asthma symptoms (Moran, 1995), and depressive conditions have been linked with negative asthma outcomes (Mancuso, Rincon, McCulloch, & Charlson, 2001). Some researchers have indicated that regardless of symptom presentation or improvement, asthma patients present with more depression than other physically ill individuals (Lyketsos, et al., 1984), leading a number of investigators to speculate that such patients may be genetically predisposed toward certain mood disorders (Wamboldt et al., 2000).

Depressive symptoms may also influence the way in which asthmatic patients interpret and respond to their physical disease. Miller and Wood (1994) noted that decreased energy levels often create such intense feelings of hopelessness and helplessness that the asthmatic fails to attend to the warning signs of an asthma attack. In fact, this disregard for the initial symptoms of an asthma attack not only places asthmatics at risk for higher morbidity, but also increases the potential for a fatal attack (Henry, Morera, Frugoni, & Gonzalez-Martin, 1993). Depression may also interfere with medical compliance (Cluley & Cochrane, 2001; Cochrane, 1995) and ultimately may affect the asthmatic patient’s ability to manage their disease effectively. The interrelationship between depression and asthma is therefore a crucial element in understanding and potentially controlling the illness.

A number of researchers have also identified anxiety as a risk factor for asthma (Carr, Lehrer, & Hochron, 1992; Moran, 1995; ten Thoren & Petermann, 2000). Anxiety disorders and asthma share many common characteristics such as breathlessness, choking, and a sensation of suffocation. Anxiety may result from perceived
physiological alterations during or before an asthma attack (Spinhoven, van Peski-Oosterbaan, VanderDoes, Willems, & Sterk, 1997) or from an interactive relationship between asthma, hyperventilation, and panic-fear (Lehrer, Isenberg, & Hochron, 1993). Greater levels of panic-fear have been associated with increased emergency room visits (Nouwen, Freeston, Labbe, & Boulet, 1999), longer hospitalizations, overmedication, and a preoccupation with symptoms (Carr, 1999; Kaptein, 1982; Lehrer et al., 1993), and may impact significantly upon the asthma patient’s level of compliance and quality of life (Hyland, Ley, Fisher, & Woodward, 1995).

Little research currently exists with respect to asthma and a sense of control over the illness. Some research has suggested that social and behavioral variables significantly influence illness control and ultimately effective management (Clark, Gong, & Kaciroti, 2001). One of the unique and complicating features of asthma is that even when patients adhere strictly to self-management regimens, they can still have severe asthma (Dupen, Higginbotham, Francis, Cruickshank, & Gibson, 1996). Nevertheless, like other chronic conditions, the management of asthma requires an increased perception of power over the illness (Crockett, 1993). In fact, Wilson, Scanagas, and German (1993) found that successful self-management programs can actually result in reduced health care dependence and greater life satisfaction. Thus, an asthmatic’s health locus of control not only becomes important in the intervention process, but also serves as a critical tool in the self-management of the disorder.

Much of the research in the field of asthma has concentrated on children and adolescents with considerably fewer studies conducted on adults. This is most likely
due to the fact that asthma frequently presents in childhood (Larsen, 2001) and has been documented to remit in up to 50% of adolescents by the time they reach adulthood (Rance et al., 2000). In addition, difficulty in obtaining random samples in adults, underdiagnosis of adult asthma, and the tendency to ascribe adult symptoms to emphysema, cardiac problems, or bronchitis (Expert Panel Report 2, 1997; Sears, 1991) have all contributed to the paucity of general adult asthma studies. Problems in determining adult asthma onset versus persistent asthma from childhood, have also limited research in this area (Reed, 1995). O'Byrne and Thomson (1995) noted that most cases of asthma which persist from childhood result from allergies, whereas asthma which develops in adults is rarely allergic and more often is occupational in nature. Also, patients with a long history of asthma have been found to have greater levels of obstruction than those with a more recent onset (Braman, Kaemmerlen, & Davis, 1991).

There actually appears to be a significant genetic link in asthmatics with symptoms that persist from childhood into adulthood (Stick, 1997). Therefore, the age of onset of asthma symptoms may be directly related to the physical health of the individual and as such, may contribute to the emotional factors associated with the disease process. Few studies have attempted to look specifically at these psychological issues in adult asthmatics and even fewer have directly addressed these issues in asthmatic women.

Many of the asthma studies that currently exist have also failed to examine the severity of illness, despite the fact that greater severity has been associated with increased levels of distress (Vamos & Kolbe, 1999), greater co-morbid psychiatric disorder (Garden & Ayres, 1993), and increased health care utilization (ten Brinke, Ouwerkerk,
Zwinderman, Spinhoven, & Bel, 2001). While recent attempts have been made to devise a classification system (Expert Panel Report 2, 1997), there is still no universally accepted measure of asthma (Wahlgren et al., 1997). Some investigators, such as Silverglade, Tosi, Wise, and D’Costa (1993), believe that this may be a reason for the enormous inconsistency in existing research.

The current research investigated the relationship between depression, anxiety, and locus of control in adult female asthma patients based on the level of severity of their illness and the age of onset of their disease. It is believed that understanding the correlates of asthma is an essential step in effectively treating and ultimately managing the illness.

Background of the Problem

Asthma is a chronic respiratory disease which is estimated to afflict close to 15 million Americans (Redd, 2002). Hersen and Van Hasselt (1990) noted that 5% to 10% of children will experience asthma at some time during their lives and in the adult population, it is the leading cause of activity restriction and loss of work productivity (Tehan, Conant-Sloane, Walsh-Robart, & Dyer-Chamberlain, 1989). Every year, complications from asthma result in millions of medical visits and approximately 5,500 deaths. In fact, despite advances in medical practice, the past 2 decades have witnessed more frequent hospitalizations of asthma patients, increased severity of asthma symptoms, and upward trends in reported mortality from asthma (Creer & Bender, 1993; Kaliner, Barnes, & Persson, 1991; Vaida, 1998). While the occurrence of asthma is
widespread, the disease process remains elusive. There is no commonly accepted
definition of asthma, either by the medical or psychological community. For this reason,
asthma is still a disease of clinical impressions, supported by laboratory findings (Fanta,
have characterized asthma as a chronic inflammatory disease of the intrapulmonary
airways, which causes episodic wheezing, chest tightness, dyspnea, and cough. Acute
airflow obstruction, followed by reversibility of symptoms, either spontaneously or with
treatment, is critical in formulating a diagnosis of asthma (Fabbri, Cogo, Cosma,
Guidoboni, & Ciaccia, 1995). In fact, despite problems in defining asthma, most agree on
the intermittent, variable, and reversible nature of the illness (Creer & Bender, 1995).

Many risk factors, such as a genetic predisposition, allergens, infections, smoke,
medications, and stress are felt to trigger attacks of asthma (Vaida, 1998). Thomson
(1995) also suggested that factors such as exercise or irritants may function as inciters of
asthma symptoms. A diagnosis of asthma requires a history, peak expiratory flow
records, and/or a reversibility test established through the use of a single dose of a
bronchodilator (O’Byrne & Thomson, 1995).

The clinical features of asthma are quite variable both between asthmatics and in
the individuals themselves (Crockett, 1993). In its mildest presentation, asthma may be
seen as only a minor aggravation that does not significantly interfere with daily activities
and lifestyle. However, in its most severe form, asthma remains a debilitating and life-
threatening disease (Hickey & Walters, 1995), requiring frequent utilization of health care
services (ten Brinke et al., 2001).
While many have attempted to categorize asthma severity, there remains no standardized classification system (Wahlgren et al., 1997). Generally, establishing the level of severity of asthma is based on history, an assessment of target symptoms, repeated measurements of lung functioning, and the amount of medication required for adequate control (O'Byrne & Thomson, 1995). Recently, the World Health Organization and National Institutes of Health (Expert Panel Report 2, 1997) devised a classification system which includes mild intermittent, mild persistent, moderate persistent, and severe persistent asthma. While these categories are somewhat arbitrary, they are essential elements in establishing an asthma diagnosis, prognosis, and treatment regimen. However, validation of such a classification system can only be established through ongoing studies.

In recent years, extensive interest has been generated regarding the interaction between psychological factors and asthma. Most of the historical research in this area stemmed from early psychosomatic medicine and psychoanalytical theory which attributed asthma to psychological causes (French & Alexander, 1941). Today, it is generally believed that the physical and emotional components of asthma are so interwoven that it is difficult to effectively treat or manage the disease without thoroughly examining the contributions of both factors (Rietveld, Everaerd, & Creer, 2000).

Interest in the psychological aspects of the illness has prompted many researchers to investigate specific emotional factors that influence the course of asthma. Numerous studies have attempted to link certain personality traits and types with susceptibility to asthma. While personality disorders have been associated with noncompliance
(Patterson, Greenberger, & Patterson, 1991), the majority of investigators have noted little evidence of any specific personality disorder in asthmatics (Bauer & Duijsens, 1998). These findings have caused most researchers to abandon the notion of a specific asthma personality in favor of a more multidimensional profile aimed at symptoms, emotions, and characteristics.

While there is little doubt that emotions and asthma are intricately intertwined, there has been no sufficient method of assessing emotionality. Creer and Kotses (as cited in Goreczny, 1995, p. 40) have pointed out that this dilemma is a result of difficulties in standardizing the stimuli that produce emotions, difficulty in quantitatively assessing emotional responses, problems in determining whether emotional responses are the cause or result of asthma, and problems created by the multifactorial relationship between asthma and emotions which is a “complex physiological enigma” (p. 40). Nevertheless, in spite of the difficulties in determining the influence of emotions on asthma, investigations have continued in an attempt to elucidate this multifaceted interaction.

Studies in the area of depression have suggested that the distress of adjusting to a chronic disease such as asthma, coupled with a long-term medication regimen, intensifies the depressive symptoms in asthmatic patients (Henry et al., 1993). Mullins, Chaney, Pace, and Hartman (1997) also contended that the unpredictable and uncertain nature of asthma may increase pessimistic attributions and actually impair the psychological adjustment of asthma sufferers. Kravis (1987) postulated that depression may cause asthmatics to neglect their health care, avoid treatment, and abuse medications, leading to a type of threatened or committed “suicide by asthma.” Similarly, Levitan (1983) noted
that asthma patients display a significantly greater degree of suicidal thoughts and behaviors.

Traditionally, anxiety has been perceived as the most common stigma or symptom in asthmatic patients (Kolbe, Fergusson, Vamos, & Garrett, 2002; Belloch et al., 1994). While levels of anxiety are not necessarily equated with the degree of actual respiratory distress, they are linked with feelings of decreased self-control (Henry et al., 1993), as well as asthma morbidity (Perna, Bertani, Politi, Colombo, & Bellodi, 1997). Studies aimed specifically at panic-fear and panic disorder have found that panic disorders are significantly more common in asthmatics than in the general population (Shavitt, Gentil, & Mandetta, 1992; Whittchen & Essau, 1993).

Disease control is another area of interest in the present study. Much of the existing research in this area stems from investigations of health locus of control on the illness experience and the notion of an internal versus an external control orientation. Wallston, Stein, and Smith (1994) believed that an individual’s health locus of control ultimately contributes to the types of health behaviors he or she will practice. Previous studies in the field of chronic disease have indicated that patients with a perception of greater internal control seek more information about their illness (Buckelew et al., 1990) and evidence substantial improvement over their symptoms (Reynaert, Janne, Vause, Zdanowicz, & Lejeune, 1995). Therefore, a sense of internal control appears to be an important factor both in the treatment and management of asthma.

Few studies on asthma and asthma psychopathology have looked specifically at adult asthmatics. Most studies have addressed asthma as a childhood disease. While
asthma does present most frequently in childhood, virtually two thirds of asthma cases
develop after the age of 17 (Weiss, 1994). In fact, it was not until 1990 that the first
guidelines for managing adult asthmatics were published (as cited in O’Byrne &
Thomson, 1995) suggesting a recognition that adult asthma might represent a unique
entity. In addition, in the child studies that currently exist, there has been little attempt to
draw parallels with adult asthma sufferers (Rocco, Barboni, & Balestrieri, 1998).

Even less attention has been given to female asthmatics as a group. In one study,
Suris, Parera, and Puig (1996) looked at chronic illness in adolescent females and found
that as a group, they had a much greater prevalence of sadness, depression, moodiness,
and suicidal ideation when compared to their normal peers. In a study of women with
chronic pulmonary disease, Sexton and Munro (1988) found that the patient’s entire
family was affected by the illness process. Centanni et al. (2000) noted that asthmatic
women had more significant levels of anxiety and depression than their male counterparts
and Tovt-Korshynska, Dew, Chokey, Spivak, and Lemko (2001) found that women
suffering from mild and moderate asthma revealed higher levels of distress compared to
other women. Despite the fact that women constitute close to 4% of the 7.2% of adults
suffering from asthma (Littlejohns, Ebrahim, & Anderson, 1989; Rhodes, Moorman,
Redd, & Mannino, 2003) and are hospitalized almost twice as often as asthmatic men
(Woods, Sorscher, King, & Hasselfeld, 2003), there are currently only a limited number
of studies which directly address this population. Therefore, a closer examination of
adult women with asthma would have tremendous implications for the field of asthma
research.
Need for the Study

Despite advances in the treatment of asthma, morbidity and mortality rates remain unacceptably high. Because poor compliance, management failure, and inadequate recognition of symptom exacerbation are associated with mortality (Vaida, 1998), clarifying the role of social and psychological factors may be critical in controlling and managing the disease. However, while there is evidence to suggest that emotional factors are involved in asthma, investigations aimed at uncovering the characteristics of this relationship have been rather scarce, with diverse interpretation and varying significance (Henry et al., 1993). Many have found existing studies to be inconsistent and inconclusive (Rocco et al., 1998; Silverglade et al., 1993).

As with most of the current studies on psychopathology and asthma, research in the area of depression has also yielded contradictory findings. While some studies have reported almost universal depressive symptoms in asthma patients (Chaney et al., 1999; Lehrer et al., 1993; Yellowlees, Haynes, Potts, & Ruffin, 1988), others have concluded that there is no pathological level of depression in asthmatics (Boulet, Deschesnes, Turcotte, & Gignac, 1991; Janson, Bjornsson, Hetta, & Boman, 1994). Similar findings have been evidenced in the anxiety literature. Anxiety was found in some cases to be an important psychopathological symptom in asthma (Belloch et al., 1994; Bussing, Burket, & Kelleher, 1996; Henry et al., 1993; ten Thoren & Petermann, 2000), whereas in other studies, anxiety was not determined to be a significant factor (Boulet et al., 1991; Janson et al., 1994).
Existing research has also failed to address the issue of locus of control in asthmatics, a critical variable in illness management. In addition, most studies have ignored the impact of childhood versus adult onset asthma, and there remains a paucity of studies which have directly addressed women asthmatics as a group.

Garden and Ayres (1993) postulated that many of the earlier studies were inherently inaccurate because they lacked standardized testing and diagnostic procedures. Others have suggested that most of the studies available are extremely difficult to interpret due to inconsistent classification, design flaws, and problems with patient selection (Moran, 1991). Some investigators have speculated that failure to look at severity of illness has led to such inconclusive findings (Garden & Ayres, 1993).

Because of these widespread limitations, there is a tremendous need for present research to utilize more regulated classification systems and methods of diagnosis, along with standardized measurement techniques. In addition, the selection and categorization of patients based on the severity of their asthma symptoms is crucial and may greatly contribute to an understanding of the unique qualities which define specific asthma groups.

At this point, scientific research has not answered many critical questions regarding emotional factors and asthma. If the relationship between asthma and emotions can be more clearly delineated, and if variables such as depression, anxiety, and locus of control can be more reliably and sufficiently measured, there could be substantial implications for the treatment and management of this disorder.
Theoretical Rationale and Significance of the Study

Asthma has long been regarded as one of the “psychosomatic” disorders, with the implication that both physical and emotional factors contribute to the disease process. Early theorists actually believed the etiology of asthma to be purely psychological in nature (Alexander, 1950). However, more contemporary models now suggest that physiological mechanisms are equally important as psychological factors (Knapp & Mathe, 1985) and that asthma is actually multideterminant in etiology (Stoudemire, 1995). Consistent with these models, Groen (1977) devised a multifactorial theory which perceived the development of disturbed ventilation in asthmatic patients to be the result of influences from particular emotional states. Similarly, the health belief model (Becker et al., 1978) contended that health behavior is directly related to beliefs and cognitions regarding severity of illness and vulnerability to disease.

Based on these models, the present study investigated the relationship between emotional symptoms and the physical disease of asthma, focusing on a number of implications which might be drawn from the findings. If the investigation proved to be significant, then it would lend greater support to existing theoretical frameworks which ascribe to a multideterminant model of disease. In spite of all the recent findings in this field, most asthma publications barely recognize emotional factors. This is a critical issue because even though psychologists are increasingly aware of the psychosocial influences on asthma, it must be a combined effort by both the medical and psychological communities if patients are to receive adequate treatment for their illness.
If indeed depression and anxiety are found to be related to the severity of asthma, then treatment of these emotional factors may assist in diminishing the symptoms and severity of the condition, decreasing the medical regimen required, and increasing overall compliance. Such findings could actually impact upon the astronomical cost of asthma by mitigating the amount of medications required, reducing the number of office visits, and lessening the length of hospitalizations. The benefits to the patient would be a much more normal lifestyle, with fewer restrictions and a greater sense of well-being.

Additionally, findings in the area of locus of control may have important implications in the self-management of asthma and ultimately may determine whether patients remain healthy or become chronically disabled by their symptoms.

Statement of the Problem

The purpose of the current study was to investigate the relationship between depression, anxiety, and locus of control in mild, moderate, and severe asthmatic women based on the age of onset of their asthma (childhood versus adult onset).

Hypotheses

The general prediction of this research was that a significant difference would exist between mild, moderate, and severe asthmatic women in their level of depression, anxiety, and locus of control. Included in this general hypothesis were the following specific hypotheses:

1. It was hypothesized that higher levels of depressive symptoms would be
associated with increased levels of asthma severity.

2. It was hypothesized that higher levels of state and trait anxiety would be associated with increased levels of asthma severity.

3. It was hypothesized that higher levels of external control would be associated with increased levels of asthma severity.

4. It was hypothesized that differentiation between mild, moderate, and severe asthma groups would be significant to the extent that group membership would be predicted.

5. It was hypothesized that the age of onset of asthma would be significantly related to levels of depression, anxiety, and locus of control.

Definition of Terms

The following were proposed as conceptual and theoretical definitions for the purpose of the present study:

1. Asthma: A chronic obstructive pulmonary disease characterized by airway inflammation, excess mucous production, and heightened airway responsiveness. Numerous stimuli may trigger asthma such as allergens, viral infections, exercise, smoke, cold air, medication, and psychological factors. Classic symptoms include dyspnea, wheezing, episodic cough, and chest tightness which are of an intermittent, reversible nature (Expert Panel Report 2, 1997).

The severity of asthma varies enormously, but for the purpose of this study, was categorized as mild, moderate, or severe based on a combination of symptoms,
treatment requirements, and objective measures of lung functioning. Asthma was operationally defined based on the physician’s assessment of history, clinical features, and daily medication requirements, as well as on the NIH guidelines for the classification of asthma severity (Expert Panel Report 2, 1997). Based on these guidelines, mild asthma was defined as asthma symptoms that occur less than once a day, require medications or bronchodilators only when symptoms increase, result in episodes which are brief, slightly influence activity, and produce nocturnal symptoms less than once a week. Moderate asthma was defined as asthma symptoms that occur every day, require a daily controller medication, affect activity, and result in nightly symptoms which present more than once a week. Severe asthma was defined as asthma symptoms which are continuous with frequent exacerbations, require multiple daily medications to maintain control, result in limited physical activity, and are often present at nighttime.

2. Depression: A unipolar mood disorder which consists of persistent feelings of dejection ranging from mild discouragement to despondency and despair. There are often feelings of listlessness, loss of initiative, decreased appetite, difficulty concentrating, and insomnia. Depression was operationally defined as the sum of scores on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

3. Anxiety: A pervasive feeling of apprehension and distress in response to an undefined or unknown threat. It is often a response to unconscious conflicts, insecurities, and impulses. For the purpose of this study, it referred to the following two constructs: state anxiety and trait anxiety (Spielberger, 1983).

State anxiety refers to a diffuse, situational state of fear in response to perceived
danger, resulting in persistent feelings of anxiety and fear which are not controllable. The perceived threat often has more influence on the anxiety state than the real danger. Trait anxiety refers to an enduring personality characteristic which leads to the perception that stressful situations are dangerous. The response to such situations is generally intense and fearful and often represents unconscious conflicts. Anxiety was operationally defined as the sum of scores on the State-Trait Anxiety Inventory, Form Y (Spielberger, 1983).

4. Locus of Control: The center of responsibility for the control of behavior, which may be either internal or external. Internal locus of control refers to the conviction that a person’s behavior can influence the outcome of a situation. External locus of control suggests that power lies outside of the individual and that outcomes are independent of their actions. Locus of control was operationally defined as the sum of scores on the Multidimensional Health Locus of Control Scale, Form C (Wallston, Wallston, & DeVellis, 1978).

5. Age of Onset: The age at which the patient first recalls suffering from asthma symptoms. This is not necessarily consistent with the age of diagnosis, which may have occurred sometime after the actual development of symptoms.

Limitations

The validity of the present study may be limited due to the following factors:

1. Due to the nature of the study, sample selection was based on voluntary participation from identified asthmatic patients and therefore cannot be considered random.
2. The use of self-report scales may have actually underestimated the level of emotional pathology in the subjects.

3. All subjects were given solicitation letters, instructions, and questionnaires by a nurse/secretary at the physician’s office. While these individuals were blind to the study, the perceived expectations and personality of the nurse/secretary could have affected subject responses.

4. Each participant completed three questionnaires during a single test session. While this controlled for the effects of history, it may have resulted in fatigue.

5. The fact that the subjects were aware of their participation in a research study, may have influenced their behavior.
Chapter II
Review of the Literature

Introduction

In order to clearly delineate existing studies in asthma research which are pertinent to the current investigation, this chapter will be divided into six sections. The first section will review the literature regarding the relationship between emotional factors and asthma. The second section will present an overview of the research on depression and asthma. The third section will concentrate specifically on research related to anxiety and asthma. The fourth section will focus on locus of control research and asthma. The fifth section will examine the existing research on the age of onset and asthma. Finally, the sixth section will look at investigations in the area of asthma severity.

Emotional Factors and Asthma

The assumption that emotional factors play a significant role in the physical expression and course of medical illness has existed since the time of Hippocrates (Stoudemire & Hales, 1995). Indeed, much of the research today stems from early psychosomatic medicine which was entrenched in the notion that physical disease was a product of psychological conflict. From the 1930s to the 1950s, asthma came to be seen as a psychological disease rooted in childhood trauma (French & Alexander, 1941), and treatment consisted of psychoanalysis or other talking cures. Today, the contention that
emotions cause disease is viewed by most researchers as not only arbitrary, but also incorrect (Lipowski, 1984). In fact, the failure to explain the actual manner in which the brain translates induced emotions into physiological experiences has been one of the leading criticisms of psychosomatic research (Weiner, 1980). Many investigators now believe that a unitary concept of disease is simply incomplete.

Such limitations in existing psychosomatic research have led investigators to explore other avenues into the mind-body relationship. While most clinicians now suspect a strong interconnection between psychological states and asthma symptoms, the actual emotional mechanisms remain elusive. Many of the early studies in the area of emotion and asthma actually sought to reveal a specific "asthma personality" with unique psychological traits (Alexander, French, & Pollack, 1968). However, such studies were generally inconsistent and the notion of a defining personality in asthma patients was not substantiated (Creer, 1978; Purcell, & Weiss, 1970).

Other researchers have attempted to identify particular personality characteristics and emotional profiles common to asthma patients. A noted strategy for investigating such emotions and asthma has been to compare asthmatics on a range of various emotional indices. One of the earliest investigations in this area was initiated by Rogerson (1937) who evaluated 30 children suffering from asthma-prurigo by subjecting them to a series of interviews and observations over a 1-year period. Twenty-two of the children were found to be excitable, restless, and overactive, while 21 of the children were described as afraid, insecure, over-anxious, and lacking in self-confidence. Also, 19 of them were found to be dominating, irritable, and aggressive. The consistent
personality findings in this study led to the conclusion that common personality factors may exist in asthma. Leigh and Marley (1956) administered the Cornell Medical Index to four separate adult groups: asthmatics seeing a physician, asthmatics seeing a psychiatrist, neurotic patients seeing a psychiatrist, and a control group. Particularly for the male asthmatics, the researchers uncovered more inadequacy, anger, and tension than in the control group, as well as greater levels of sensitivity, depression, and anxiety. Overall, they found the asthmatics to be more psychoneurotic and vulnerable, with increased rigidity and greater difficulty handling anxiety. Similarly, Rees (1956) investigated the role of personality characteristics such as obsessionality, anxiety, sensitivity, and timidity in a group of 441 asthmatics and 321 nonasthmatics. He reported that in 18% of asthmatics, psychological factors were the only determined cause for their asthma symptoms.

Consistent with earlier studies, Jones, Schum, Kinsman, and Resnikoff (1976) administered the Minnesota Multiphasic Personality Inventory (MMPI) to 155 hospitalized asthma patients in order to investigate the possibility of common personality characteristics. They found no stereotypic asthma personality. However, they did find that like many patients with chronic illness, asthmatics displayed significant elevations on the neurotic triad of the MMPI ($p < .001$), which they suggested might be a defensive stance in dealing with the illness itself. Similar findings were evidenced by Lyketsos et al. (1984). In assessing personality characteristics, they compared 35 adult asthma patients with 45 age-matched physically ill patients and measured their performance on the Personality Deviance Scale, The States of Anxiety and Depression Scale, and the
Social Readjustment Rating Scale. Overall, they found that the asthmatics were less
dominant ($t = 2.82$, $p < .005$), more introjective ($t = 3.57$, $p < .005$), more anxious ($t =
6.43$, $p < .0005$), and more depressed ($t = 5.59$, $p < .0005$) than the control group.
Huovinen, Kaprio, and Koskenvuo (2001) also concluded that while no specific
personality type could be linked to asthma onset, these patients appear to have diminished
life satisfaction as well as a higher level of neuroticism. In another study, Henry et al.
(1993) investigated the psychopathological profiles of 43 adult chronic asthma
outpatients using the SCL-90-R. Although their sample was quite small, and they failed
to find overall significance, they did observe elevated psychopathology in these patients
in areas of somatization ($M = 1.80$), depression ($M = 1.64$), obsession-compulsion ($M =
1.53$), and anxiety ($M = 1.45$). Other studies have investigated the impact of emotions on
asthma onset, morbidity, and mortality. Teiramaa (1981) studied 100 adult asthmatics by
dividing subjects into acute, subacute, and insidious groups based on the duration of the
prodromal phase of asthma. Using a semistructured interview and a number of
questionnaires, including the MMPI, the Wartegg Test, and the Beck Depression
Inventory, it was concluded that the vulnerability to the onset of asthma was directly
related to psychic characteristics in combination with psychosocial stress factors. Cluley
and Cochrane (2001) studied 103 adult asthma patients recruited from an outpatient clinic
and divided them into four subgroups: (a) those with well-controlled asthma, (b) those
with poorly controlled asthma who were medication compliant, (c) those with poor
control and non-adherence with medication, and (d) those who dropped out. Utilizing
both the Hospital Anxiety and Depression Scale (HADS) as well as a diagnostic
interview, they found significant differences between the groups in response to the HADS 
\( p < .002 \), with 46% of patients displaying a probable psychiatric disorder. In addition, 
30% of patients acknowledged a psychiatric problem in response to the diagnostic 
interview. They also found that psychiatric morbidity increased when control was poor, 
especially when there was poor control coupled with low adherence to medication.

In a study aimed at uncovering the psychiatric features of near fatal asthma, 
Yellowlees and Ruffin (1989) studied 25 patients (19 females, 6 males), who had suffered 
a near fatal attack. These patients were evaluated using a detailed clinical interview and 
two structured interviews, as well as numerous health and personality questionnaires. 
The findings indicated that 40% of the patients had psychiatric disorders at the time they 
were evaluated, as well as very high levels of denial. Denial was significantly higher in 
near fatal asthma patients \( (M = 4.6) \) when compared to control subjects \( (M = 2.3) \) \( p < 
.05 \). In addition, following the near fatal attack, patients seemed to decompensate 
psychiatically, showing greater symptoms of anxiety or increased levels of denial. It was 
concluded that denial, along with a psychiatric history, appear to be potential components 
in possible death from asthma. Similar and supportive findings were reported by 
Campbell et al. (1995). They evaluated 77 consecutive subjects who presented to a 
hospital with a near fatal asthma attack. Using a series of interview questionnaires, 
including the General Health Questionnaire, the denial scale from the Illness Behavior 
Questionnaire, and the stigma subscale from the Asthma Attitudes and Beliefs 
Questionnaire, the investigators found high levels of psychiatric morbidity (43%) in 
patients suffering a near fatal asthma attack. They also found high levels of denial
(57%), which in asthmatic patients, may well be life-threatening.

Certain personality types have also been associated with greater susceptibility to asthma. Sharma and Nandkumar (1980) considered patients with marked dependency needs, a high need for affection, covert aggression, extreme inhibition, and “simply neurosis” to be high-risk personality types. Similarly, Plutchik, Williams, Jerrett, Karnsu, and Kane (1978) suggested that anxiety, depression, and problems with self-esteem were all risk factors for asthma. Huovinen et al. (2001), in a review study of 11,540 asthmatic adults, noted that elevated extroversion scores, as demonstrated by a confidence interval of 1.58 to 5.64, were associated with increased vulnerability to asthma, particularly in women ($p < .05$).

While many studies have appeared to confirm the notion that emotions and asthma are intricately connected to asthma symptomatology, other studies have been less conclusive. Benjamin (1977) investigated 47 asthmatics and 43 matched non-asthmatic controls and compared them using respiratory assessments as well as standardized interviews. No major differences were demonstrated in the psychopathology of the groups; however, the index cases in this study had relatively mild asthma and the majority of the subjects had been symptom-free for a period of almost 3 years. Garden and Ayres (1993) compared 20 patients suffering from brittle asthma with a control group experiencing less severe asthma. The subjects were matched for age, sex, and duration of illness and were asked to complete the General Health Questionnaire, the Eysenck Personality Inventory, a structured clinical interview (SCID), and a screening test for psychiatric disorders. Though the results indicated that the brittle asthma patients had
more psychiatric morbidity, there was no difference in personality profiles between the two groups. The investigators point to the lack of specificity in the General Health Questionnaire instrument as a possible cause for their negative findings.

In an investigation of personality traits and disorders, Bauer and Duijsens (1998) compared a sample of 59 asthma and chronic obstructive pulmonary disease (COPD) patients with matched groups of psychiatric and normal subjects on a self-report instrument called the Questionnaire on Personality Traits. Their findings indicated that while there was a significant difference between the psychiatric patients and the pulmonary patients in terms of the number of personality disorders they reported ($p < .05$), there was no more prevalence of personality disorders in asthma and COPD patients than in the general population. Similar findings were reported by Rocco et al. (1998) who examined the presence of personality characteristics and psychiatric symptoms in 17 patients with asthma and 17 patients with near fatal asthma. Following baseline assessments and interviews, patients completed a series of psychodiagnostic tests including the MMPI, the Hamilton Depression and Anxiety Scale, and the Zung Anxiety and Depression Scale. Results indicated no difference in the groups in their psychodiagnostic scores and no significant psychopathological profile in asthma patients, with or without a near fatal attack. They also found no evidential connection between familial affective disorders and severe asthma, which directly contradicts the findings of Wamboldt, Weintraub, Krafchick, and Wamboldt (1996). These negative findings might be attributed to the limitations in the experimental design of this study, particularly the small sample size and lack of a structured diagnostic interview. Nevertheless, the results
are consistent with other studies which have failed to find a convincing level of psychopathology in asthma patients (Barboni, Peratoner, Rocco, & Sabadini, 1997; Boulet et al., 1991; Janson et al., 1994).

The conflicting findings in existing research on asthma and emotion were somewhat disturbing and could actually suggest that there is a weak relationship between emotionality and asthma. However, many of the early studies utilized small samples, lacked a comparison group, and had questionable generalizability. In addition, based on the substantial number of studies which have found a significant interaction between psychological factors and asthma, it is more likely that the relationship is complex, multifaceted, and as of yet, undetermined. Therefore, additional research is necessary not only to delineate the specific emotional variables that are present in asthmatic patients, but also to clarify the extent to which the severity of asthma symptoms impacts upon the psychological well-being of patients. As Stoudemire and Hales (1995) suggested, measuring the interaction between biological and psychosocial variables is one of the most essential challenges facing contemporary researchers.

**Depression and Asthma Research**

Much of the research on depression and asthma has developed from extensive investigations in the area of chronic illness. These studies have generally supported the contention that medical disease and depression are intricately interwoven (Guthrie, 1996; Hurwitz, & Morgenstern, 1999). For example, Coulehan, Schulberg, Block, Janosky, and Arena (1990) found that of 618 patients in a primary care setting, patients with major
depression not only had more physical illness than nondepressed patients \( (p = .057) \), but they also had greater functional disability related to their physical disorder. Studies of psychiatric patients have also indicated that those who have depression and a medical condition exhibit much greater chronicity and a lower recovery rate than patients who are depressed, but have no concurrent medical condition (Keitner, Ryan, Miller, Kohn, & Epstein, 1991).

Studies have also been conducted on medical patients who subsequently develop depression following the onset of their physical disability. Agle and Baum (1977) conducted psychiatric interviews on 23 patients with chronic obstructive pulmonary disease at a COPD rehabilitation project. After a year of studying the patients, they concluded that in addition to symptoms of jumpiness, tremulousness, tension, and a sense of impending doom, 17 of the 23 patients had debilitating depression, with symptoms of sadness, worthlessness, tearfulness, lack of motivation, and suicidal ideation. Loss of appetite and sleep disturbance were also common symptoms in these patients. Hodgkiss and Watson (1994) compared 29 patients with chronic pelvic pain prior to laparoscopy with a control group of 33 pain-free women. After completing four self-report scales, including the Hospital Anxiety and Depression Scale, the Illness Behaviour Inventory, the McGill Pain Questionnaire and the “disease conviction” scale of the Illness Behaviour Questionnaire, they found that 29 of the patients with chronic pain reported significantly higher levels of depression \( (M = 4.3, p = .04) \) and illness behavior \( (t = -6.2, p < .001) \) when compared to patients without pain. Silverstone (1990) studied 211 medical patients suffering from life-threatening illnesses such as myocardial infarction, acute upper
gastrointestinal hemorrhage, subarachnoid hemorrhage, and pulmonary embolism.

Patients who were depressed during their recovery period had poorer outcomes a month after their admission, with upwards of 33% of them suffering a life-threatening complication. They were also much more likely to die from complications subsequent to their illness ($p < .01$) than non-depressed patients.

Clinical depression has also been linked to poor recovery from strokes, with less improvement in functional status and cognitive performance (Morris, Raphael, & Robinson, 1992). In patients with myocardial infarction, depression has been found to cause a higher degree of chronicity and a poorer prognosis, when compared to patients with no history of myocardial infarction (Wells, Rogers, & Burnam, 1993). Major depression has also been found to significantly contribute to mortality following a myocardial infarction (Frasure-Smith, Lesperance, & Talajic, 1993). Therefore, the relationship between medical illness and depression is critical not only in terms of the treatment of acute episodes of illness, but also in terms of the long term maintenance of the disease process.

Like many chronic diseases, asthma has long been associated with depressive symptoms. In fact, the comorbidity of the two illnesses is thought to be common (Yellowlees et al., 1988) and the interconnection frequently leads to the worsening of both conditions (Rubin, 1993). The prevalence of major depression in asthmatic patients has been found to be close to 2½ times higher than rates found in the general population (Kessler et al., 1994) and up to 5 times greater than in other chronic medical conditions (Wells, Golding, & Burnam, 1988). Some of the earliest writings in the area of asthma
and affective disorders were based on the work of psychoanalysts and nonpsychiatric physicians who came to identify the wheeze in asthma as the suppressed cry of the child for the mother (French & Alexander, 1941). However, these preliminary case studies failed to provide any reliable conclusions regarding the association between depressive disorders and asthma symptoms.

Early research attempts to isolate depression as a factor in asthma were reported by Knapp and Nemetz (1957). They initially studied 40 adults suffering from chronic bronchial asthma using anamnestic studies, intensive treatment, and psychoanalysis, and found that episodic depression was the most common nonrespiratory symptom, evidenced in 83% of the patients. Most of the patients were found to have habitual feelings of sorrow and many attributed their depression to their chronic physical disability. Following this initial investigation, Knapp and Nemetz (1960) selected nine severe perennial asthmatics and asked them about concomitant feelings and fantasies which accompanied their attacks, as well as the antecedent events which occurred 48 hours prior to the onset of their asthma attacks. After studying the preliminary patterns of 406 attacks, the investigators concluded that by far, the most prominent manifestation during an asthma attack was depression, which was present in 43% of attacks. In addition, after dividing the subjects into "carefully studied" and "severe moderate" attack groups, they found depressive manifestations in almost two thirds of the attacks, which differed significantly from the main population of attacks (p < .01). They concluded that these patients had high levels of discouragement with frequent perceptions of being doomed and having a deep defect. They also found that the levels of hopelessness and
helplessness expressed by these patients were out of proportion to the degree of physiological distress they were experiencing. Knapp, Carr, Mushatt, and Nemetz (1965) utilized the same technique in reporting on six fatal asthma cases. Although biological factors were felt to play a role in the exacerbation of asthma, overt despair and hidden self-destructiveness were seen as contributing to the fatal course of the disease. While such early studies clearly suggested a connection between depression and asthma, they are cautiously interpreted, due to their small sample size, paucity of standardized instrumentation, and absence of diagnostic criteria.

More recently, Mancuso, Peterson, and Charlson (2000) examined 230 asthma outpatients suffering from moderate asthma and interviewed them using the Asthma Quality of Life Questionnaire (AQLQ) and the Medical Outcomes Study SF-36. They found that 45% of the patients reported depressive symptoms. In addition, when compared to patients who had a negative screening for depression, patients who initially tested positive for depression scored significantly worse on all of the composite measures ($p < .0001$). Similarly, Chaney et al. (1999) compared 39 longstanding asthma patients to age-matched healthy controls and found that the asthmatics were at much greater risk for major depression and learned helplessness deficits (21%) than their healthy cohorts (5%). In a related investigation, Goethe, Maljanian, Wolf, Hernandez, and Cabrera (2001) studied 317 participants in an inner-city asthma program. Patients were divided into two groups (depressed = 173, non-depressed = 41) based on their level of depressive symptoms as measured on the Center for Epidemiologic Studies Depression Scale (CES-D). They were then tested at 3 and 6-month follow-up periods on the Health Status
Questionnaire (HSQ) and a measure of functional health status (SF-36). Despite some improvement, the overall functional status and depressive symptoms were not transient. Indeed, the depressed patients had significantly lower functional scores compared to the non-depressed group both at baseline ($F = 23.13, p < .001$) and at the 3-month follow-up ($F = 77.07, p < .009$). Although the unequal sample size of the groups might have increased variability and decreased generalizability of the study, more than 55% of the subjects scored above the cut-off point for depression, suggesting a high incidence of depressive symptoms in asthma patients.

Belloch et al. (1994) examined the relationship between asthma and depression using separate questionnaires to measure anxiety, depression, self-consciousness, and subjective symptoms in 51 non-smoking asthma patients. They found that severity of asthma symptoms was not only related to advanced age and longer duration of illness, but also to high scores on depression ($p < .01$). Wamboldt et al. (1996) also noted that severity of asthma was associated with familial psychiatric history and relatives of asthma patients had significantly higher depression levels than relatives of non-asthmatic groups. Sibbald, White, Pharoah, Freeling, and Anderson (1988) suggested that patients with the highest morbidity had much greater feelings of stigma, fewer positive attitudes toward their physician, and less confidence in their ability to successfully manage their asthma symptoms. Similarly, Rimington, Davies, Lowe, and Pearson (2001) postulated that because depression is actually the best predictor of symptom levels, it may negatively impact the overall management of the illness.

Consistent with this recognized depressive symptomatology, suicidal thoughts and
behaviors have also been associated with asthma. In one study, Levitan (1983) reviewed the charts of 100 asthma patients, 75 COPD patients, 200 hypertension patients, and 200 cholelithiasis patients and found that the number of asthmatics demonstrating suicidal tendencies was significantly greater than both the COPD group ($p < .01$) and the cholelithiasis group ($p < .005$). In fact, 10.7% of the men and 12.5% of the women asthma patients were found to exhibit suicidal ideations or attempts. Such findings further reinforced the connection between the physical and emotional components of asthma.

While many investigators now believe that depression and asthma are interrelated, clues to the etiology and pathogenesis of this relationship have remained elusive. Klerman (1981) suggested that depression in asthma patients might result from a number of sources such as the continual threat of death, problems in self-esteem and self-image due to illness, and decreased capacity for sexual and interpersonal relationships. In fact, Thompson (1986) indicated that depression, with concomitant loss of desire, is not uncommon in individuals suffering from asthma. Such patients may develop depression due to high doses of corticosteroids, which are often used in the treatment of their asthma symptoms (Lewis & Smith, 1983; Thompson & Thompson, 1984). The self-perception of being chronically ill may also contribute to a sense of despair (Lehrer et al., 2002).

Despite speculation regarding the role of depression in asthma, researchers are still uncertain as to the actual connection between the two variables. A considerable number of studies have now been directed toward the role of depression in the onset of asthma, the maintenance of asthma symptoms, and the long term treatment and
management of the disease process.

Depression has been linked to the onset of asthma, particularly through the mechanism of loss. Levitan (1985) evaluated six patients whose asthma actually began during a period of highly intense mourning. He found that the magnitude of the grief reaction was an important psychological component to the onset of asthma symptoms. In a study of the acute or subacute onset of the disease, Teiramaa (1981) reviewed 100 case reports from a previous study, divided them into groups based on the prodromal phase of the illness, and evaluated past records from the MMPI, the Wartegg Test, and the Beck Depression Inventory. Although in the acute phase, disappointment prior to asthma was associated with a close friend ($p < .025$), in the subacute onset, it was more significantly related to work or economic matters ($p < .0001$). Teiramaa also noted that a diagnosis of anxiety neurosis, obsessive neurosis, or phobias was apparent in 43% of patients who had suffered a disappointment before the onset of asthma, as compared to 19% in the other groups ($p < .025$). Therefore, a sense of loss, disappointment, or sadness may actually trigger asthma symptoms in already vulnerable individuals. Other studies have reported crying (Weinstein, 1984), stressful events (Goreczny, Brantley, Buss, & Waters, 1988), and changes in mood (Hyland, 1990) as precipitating factors in asthma as well.

In studies comparing depressive factors with the physiological components of asthma, Allen, Hickie, Gandevia, and McKenzie (1994) examined the relationship between mood states and the voluntary drive to breathe. They assessed psychological distress using the Profile of Mood States questionnaire. Their findings indicated that depressed mood increased the risk of impaired voluntary activation of the diaphragm by
3.5 times. They concluded that asthmatic individuals suffering from depression would be at a heightened risk for ventilatory failure and possibly death if faced with severe airway narrowing. Support for these findings was demonstrated by Gift (1991). He evaluated 36 adult asthmatics in the emergency room, both during an acute episode of dyspnea, and again after their dyspnea had subsided as they were being discharged. The findings indicated that in addition to other emotional states, depression increased during times of high dyspnea. Miller (1987) determined that depression is responsible for increased central and peripheral cholinergic tone in depressed asthmatic children and in extreme cases, may be so destabilizing that it actually results in sudden death from asthma. In an attempt to elucidate this hypothesis, he gathered data from extensive telephone conversations with families of children who had died of asthma. He found that in at least four cases, depressive symptoms were clearly present and in three of these cases, there was a direct reference of a wish to die within the last week of life. In a follow-up study, Miller and Wood (1997) evaluated 24 children between the ages of 8 and 17, who suffered from moderate to severe asthma. While viewing the Movie E.T., the investigators recorded heart and respiration rates, as well as oxygen saturation. After identifying and preselecting scenes that evoked sadness, happiness, or both, they analyzed self-reports of emotion and indices of physiological responses. They concluded that while sadness evoked specific patterns of autonomic influence which resulted in significant airway constriction, happiness appeared to relieve airway constriction. Levitan (1983) also found that suicidal trends were 3 times greater in asthma patients after they were diagnosed.
There have been numerous reports which have indicated that depression directly contributes to death in asthma. Many of these investigations have looked specifically at fatalities in asthmatic children (Kravis, 1987; Lewiston & Rubinstein, 1986; Mascia et al., 1989; Strunk, Mrazek, Fuhrmann, & Labrecque, 1985). Though adult studies have been less common, they have generally yielded similar findings. Printgaret (1984), in a review of the literature, found that depression, self-destructive/suicidal intent, psychosis, and dependency on medical treatment, were all associated with adult asthma death.

Picado, Montserrat, dePablo, and Plaza (1989) examined the outcome of 49 adult asthma patients (20 males and 29 females), who had required mechanical ventilation following severe exacerbation of their asthma. Of the six fatalities in the group, all of whom were females, four required previous treatment for an anxiety-depression syndrome. Dirks and Kinsman (1982) analyzed the personality of a 20-year-old woman who died of asthma. They found that in addition to minimizing and disregarding her asthma symptoms, she consciously engaged in activities known to be harmful to her health and was noncompliant with therapeutic regimens.

Fritz, Rubinstein, and Lewinston (1987) noted that depression can cause patients to not only neglect their disease, but also to mislabel symptoms, often resulting in devastating consequences. Affleck et al. (2000) recognized that this misperception was in fact, related to mood states. After studying 48 adults with moderate to severe asthma, and reviewing mood states, asthma symptoms, and peak expiratory flow rates (PEFR), they concluded that changes in mood were directly linked not only to transitory changes in symptoms (happy-sad mood, $p < .05$; calm-nervous, $p < .01$), but to changes in PEFR.
as well (active-passive mood, $p < .05$; peppy-drowsy mood, $p < .001$). Asthmatics with major depression have also been found to possess poorer mental and physical health when compared to asthmatics not suffering from major depression, as well as exhibiting impaired health perception (Afari, Schmaling, Barnhart, & Buchwald, 2001). Depression can inhibit self-control, decrease adherence to conventional medical treatment, and cause medication abuse (Kravis, 1987). These findings support Miller’s (1987) contention that psychological disturbances are a significant risk factor in asthma death.

Depression has also been associated with poor compliance with treatment and poor compliance with treatment has been associated with morbidity and mortality. Bosley, Fosbury, and Cochrane (1995) recruited 102 asthma patients, ages 18 to 70, who required treatment with regular corticosteroids and beta-agonists. In addition to assessing the patients with the Hospital Anxiety and Depression Scale, the Inventory of Interpersonal Problems, and a semistructured interview, the patients were given an inhaler to use twice daily over 12 weeks. Of the 72 patients who completed the study, 37 were considered noncompliant, utilizing less than 70% of the prescribed dose of medication or omitting doses all together. An analysis indicated that the noncompliant group had significantly greater mean levels of depression (4.7%) when compared to the compliant group (3.2%) ($p < .05$). The investigators concluded that depression might be one of the psychological factors that significantly contribute to noncompliance and ultimately detracts from self-care and medical management of the disease. Indeed, studies have also shown that despite the prevalence of depression in asthmatics (41%), only a small percentage of them (7%) receive any type of treatment or medication for their symptoms
While numerous studies have reported the presence of depression in asthma, other studies have failed to find an association. Ringsberg, Loewhagen, and Sivik (1993) compared 15 patients with asthma to 13 patients with asthma-like symptoms on a battery of tests, including the MMPI and the Beck Depression Inventory. Their results indicated that the patients with asthma-like symptoms were more psychologically distressed, had greater levels of depression, and had a poorer quality of life than the asthma patients. Janson et al. (1994) compared 715 subjects with respiratory symptoms on a number of respiratory as well as psychological measures. In addition to a structured interview, psychological status was assessed using the Hospital Anxiety and Depression Scale (HAD). While they found a significant correlation between anxiety and depression and asthma-related symptoms ($p < .01$), they observed no correlation between anxiety and depression and the actual self-reported diagnosis of asthma. Creer (1986) critiqued the case-controlled study of 21 severe asthmatics conducted by Strunk et al. (1985) which established positive findings of depression as a cause for death from asthma, and contended that in fact, they had failed to establish such a relationship.

These inconsistencies in existing research regarding depression and asthma raised the possibility that depression is a broad construct, and that the influence on asthma may be extremely diverse. In addition, the role of the disease itself requires further examination. Certainly, there is a need for more controlled, prospective studies in this area.
Anxiety and Asthma Research

Researchers have long suspected a relationship between asthma and anxiety. This may be due in part to the fact that many physical symptoms which result in a sensation of breathlessness such as hyperventilation, shortness of breath, and difficulty in taking a deep breath are common to both disorders. However, beyond this perspective, there has been only minimal agreement on the causal connection between these disorders, with some investigators suggesting that anxiety actually precipitates the asthma symptoms and others inferring that the anxiety is more likely a consequence of the chronic asthma condition.

Numerous studies have identified the presence of anxiety in asthma patients (Baron et al., 1986; Bussing et al., 1996; Dirks, Kinsman, Staudenmayer, & Klieger, 1979; Kinsman, Dirks, Dahlem, & Heller, 1980; Silverglade et al., 1993; Thompson & Thompson, 1985). Shavitt et al. (1992) evaluated 107 asthmatic outpatients to determine the prevalence of anxiety disorders. They found that 13.1% of the asthmatics suffered from agoraphobia and 6.5% of the asthmatics suffered from panic disorders, suggesting much greater prevalence of phobic anxiety disorders than in the general population. In evaluating the occurrence of anxiety disorders in 86 asthmatic subjects, Nascimento et al. (2002) determined that 52.3% had an existing anxiety disorder, 13.9% had a panic disorder, and 26.8% had agoraphobia without a panic disorder. Similar findings were reported by Sreedhar (1989) who compared 50 asthmatic patients with matched groups of neurotic patients, general hospital outpatients, and a control group. Significantly greater
levels of anxiety were found in all three of the groups compared to the normal control group. Female asthmatics in particular were found to have much higher levels of anxiety when compared with the general hospital patients and control groups.

In patients suffering from life-threatening asthma, anxiety has also been found to be a significant factor. Yellowlees et al. (1988) compared 13 patients who had suffered a near fatal episode of asthma with 36 asthma patients who had not had such an experience. All subjects were given a series of interviews, along with the Diagnostic Interview Schedule. A psychiatric diagnosis was confirmed in 4 of the 13 near-miss cases (31%) and in 12 of the 36 asthma patients who had not experienced a life-threatening asthma episode (33%). Overall, they found that in addition to higher psychiatric morbidity, 87% of all asthmatic patients had anxiety disorders, a finding that is consistent with a study of patients suffering from chronic airway obstruction (Yellowlees, Alpers, Bowden, Bryant, & Ruffin, 1987). In a later study on life-threatening asthma attacks, Yellowlees and Ruffin (1989) evaluated 25 patients who had suffered a near fatal attack, using both structured interviews as well as a number of illness and health questionnaires. Of the 10 patients given a DSM-III diagnosis, nine were consistent with an anxiety disorder. Similar results were reported by Kolbe et al. (2002) who compared 77 asthmatics suffering from severe life-threatening asthma with 239 patients with acute asthma, matched only by the date of their attack. They were then compared with a random sample of community-based asthmatics. Although the patients with severe life-threatening asthma had less previous counseling (25%) when compared to the acute asthma group (35%) (p < .05), patients with acute asthma were found to have greater prevalence of
anxiety and depression than those in the community-based asthma group.

The notion that asthma symptoms are precipitated or influenced by anxiety has been studied by numerous investigators. Dirks, Jones, and Kinsman (1977) suggested that emotional factors may cause increased chronicity, medication dependence, and an intensified reliance on medical agencies. They studied 237 adult and adolescent inpatients and administered the Asthma Symptom Checklist and the MMPI. Both the MMPI panic-fear scale and the Asthma Symptom Checklist were related to chronicity in patients and were able to correctly assign high panic-fear asthmatics 85% of the time ($p < .001$). The inception of asthma may actually represent the culmination of synergic effects caused by various psychic factors (Teiramaa, 1976). Many researchers such as Kinsman, Dirks, and Jones (1982) have suggested that the course of asthma is significantly influenced by the personal style of the individual. This personal style, which is reflected in specific behaviors, may either assist or impede the individual in coping with their disease, and is therefore an essential element in the assessment and treatment of the asthmatic patient.

In determining the association between psychic factors and asthma, Teiramaa (1979) studied the original records of 100 asthmatic and 100 nonasthmatic subjects and compared them on a number of questionnaires, along with the Self-Image Test, the Wartegg Test, and the Beck Depression Inventory. When compared to the control group, asthmatics were found to have significantly more problems related to the handling of anxiety ($p < .01$), inhibition ($p < .0005$), disturbances in self-esteem ($p < .005$), depression ($p < .0005$), and fears (particularly in women) ($p < .0005$). The role of pre-
existing anxiety disturbances as a factor in asthma exacerbation have also been reported by Dirks and Kinsman (1982). In the case of a 20-year-old asthmatic woman, they noted that the severity of the patient's illness collided with her maladaptive personality style, ultimately resulting in her death. They concluded that a vicious cycle took place in the life of this young woman whereby "the asthma attack leads to unrecognized anxiety, the anxiety leads to physical activity, the activity exacerbates her asthma, the exacerbation leads to more anxiety, ad infinitum" (p. 182).

Consistent with these findings, Tietz, Kahlstrom, and Cardiff (1975) reported on three adolescent asthmatics who had subsequently died during acute asthmatic attacks. They suggested that a pervasive anxiety was characteristic of all three cases and resulted in an incapacity to cope with the asthma symptoms. They concluded that patients with significant psychopathology are particularly susceptible to asthma complications.

Reddihough, Jones, and Rickards (1978) interviewed 412 asthmatic children and adolescents and asked questions about anxieties, fears, and knowledge about asthma. They found that in addition to limited knowledge of their illness, subjects had significant anxieties related to death, decreased activity, separation issues, and long-term effects of medication.

Investigators have also analyzed trait anxiety as a factor in asthma. Stauder and Kovačs (2003) studied 646 consecutive patients who attended outpatient allergy clinics. Of these patients, 383 had rhinoconjunctivitis, 173 had asthma, and 90 presented with "other" allergies. All of the patients completed self-administered questionnaires which included the State-Trait Anxiety Inventory (STAI), and 60 of the patients also completed
structured interviews. After establishing the relationship between the STAI-Trait scores (STAI-T) and clinical diagnoses, it was determined that the STAI-T mean was higher in asthmatic patients than in those with rhinitis or other allergic conditions (45.6 vs. 41.7 and 43.2, $p < .01$). Dekker, Barendregt, and DeVries (1961) also found that adult asthmatic women scored higher on a test for neuroticism than a control group.

The notion that anxiety is a symptom or consequence of asthma had been studied extensively in the literature as well, particularly in the area of panic disorders (Carr et al., 1992; Carr, Lehrer, Rausch, & Hochron, 1994; Kinsman, Luparillo, O’Banion, & Spector, 1973; Porzelius, Vest, & Nochomovitz, 1992; Yellowlees et al., 1987; Yellowlees et al., 1988). Carr (1998) contended that anxiety and panic can actually exacerbate asthma symptoms, independent of objective pulmonary impairments, culminating in an increased need for steroid treatment, abuse of asthma medications, more frequent hospitalizations, and greater lengths of stay when hospitalized. Similarly, Kaptein (1982) indicated that the length of hospitalization following an asthma attack may be associated with the level of neuroticism and anxiety that occurred with the attack.

In an attempt to clarify this point, he administered the State-Trait Anxiety Inventory and the Panic-Fear Personality Scale to 40 Dutch asthmatics (16 to 60 years old) and found that the lengths of hospitalization as well as rehospitalization were associated both with anxiety and stigma related to being asthmatic. Consistent with these findings, Butz and Alexander (1993) examined the levels of state and trait anxiety in 155 asthmatic children immediately following an acute asthma attack. While the children were administered the State-Trait Anxiety Inventory for Children (STAIC), the State-Trait Anxiety Inventory-
Form C was simultaneously administered to their mother. Although there was no measurable relationship between mother and child anxiety, a significant association was found between the child state anxiety and “being upset” ($p < .05$) and between the child trait anxiety and “feeling panic” ($p < .01$) at the beginning of an asthma attack. Overall, they found that approximately two thirds of the children reported a feeling of “panic” at the onset of the asthma attack, suggesting that most children experience some form of anxiety during these episodes.

Indeed, many investigators have found higher rates of panic symptoms and panic disorders in patients with pulmonary diseases such as asthma (Carr, 1999; Karajgi, Rifkin, Doddii, & Kolli, 1990; Yellowlees et al., 1987), and it has been estimated that 1 in 10 asthma patients suffers from a panic disorder (Lehrer et al., 2002). In fact, states of panic-fear, irritability, and fatigue have been common symptom patterns among hospitalized asthma patients (Dirks et al., 1979; Kinsman et al., 1973). Zandbergen et al. (1991) found that compared to psychiatric patients, individuals with panic disorders had a higher occurrence of pulmonary disease in their lifetime.

In an attempt to elucidate this relationship, Perna et al. (1997) evaluated 51 asthmatic patients with respect to the prevalence of panic disorder and panic attacks, the temporal relationship between such disorders, and the familial risk for panic disorder. Clinical data was obtained through interviews and a psychiatric diagnosis was determined using the Diagnostic Interview Schedule. Family information was gathered using the Family History Method for Research. Overall, panic disorder (19.2%) and social phobia (9.8%) were the most prevalent psychiatric disorders found in the asthmatic patients. In
fact, the percentage of both lifetime panic disorder (20%) and sporadic unexpected panic attacks (26%) were much higher than in the general population. They found that in 90% of the asthmatics with panic disorder, the asthma had preceded the onset of the panic disorder. They also noted higher morbidity for panic disorder in families of asthmatics with panic disorders. These findings were not supported by Van Peski-Oosterbaan, Spinhoven, Van Der Does, Willems, and Sterk (1996). They evaluated the anxiety levels and panic disorders in 123 consecutive patients referred to a lung function laboratory using multiple self-report questionnaires, spirometry, and bronchial responsiveness. The findings indicated that panic disorder was higher in asthmatics than in the general population (9.0%); however, it was similar to nonasthmatic patients (8.9%). The investigators speculated that selection bias might be the cause for the higher prevalence of panic disorders reported in the asthma literature. Since the previous study by Perna et al. (1997) lacked a control group and dealt almost exclusively with allergic rather than intrinsic asthmatics, this may well be the case. Nevertheless, based on the majority of findings, it seems likely that there is a unique connection between asthma and panic and this connection is most likely bidirectional and nonspecific (Lehrer, et al., 2002).

Therefore, asthma and anxiety have been found to be intricately interwoven. Though a number of investigations have reported negative findings and suggest a lack of pathological levels of anxiety in asthma patients (Boulet et al., 1991; Janson et al., 1994; Rocco et al., 1998), the majority of studies point to a strong correlation between anxiety and asthma.
Locus of Control and Asthma Research

Control and a sense of mastery are critical elements in the pursuit of psychological and physical well-being. This is particularly true in patients suffering from potentially life-threatening illnesses such as asthma, where the unpredictable nature of the disease often prevents a feeling of competency and control. Research in the area of chronic illness and control has indicated that perceptions of control are often challenged by illness, and successful coping involves reestablishing a sense that one can gain desired outcomes (Janoff-Bulman & Frieze, 1983; Taylor, 1983).

Much of the research in this area has supported the contention that beliefs in control are associated with more successful health outcomes (Peterson & Stunkard, 1989; Wallston, 1992). Wallston, Maides, and Strudler-Wallston (1976) indicated that increased information about a life threatening condition leads to more positive steps to abolish the condition. In fact, Seeman and Seeman (1983) found that adults who feel more in control of their health actually participate in increased self-initiated preventive care. At the same time, individual differences greatly determine the level of perceived control patients experience with respect to their chronic illnesses.

The focus of a considerable amount of the work in the area of perceived health control has been in the area of locus of control (Rotter, 1966). According to Rotter’s social learning theory, people will engage in goal-directed behaviors only if they value the reinforcer, and only if they think their actions will lead to this valuable reinforcer. When people are suffering from a health threatening situation, they will seek information if they
value the outcome, and if they believe that their behavior will influence their health (Wallston et al., 1976).

These consistent beliefs which influence behavior are generalized expectancies. One of the most prominent general expectancies is internal versus external control, which refers to the extent to which a person feels that they have control over reinforcers that occur relative to their behavior (Rotter, 1966). External control suggests that a reinforcement is not seen by the individual as being entirely dependent upon their actions. Instead, luck, chance, fate, unpredictability, or even the control of others are believed to play a role. Internal control implies that the individual perceives what happens to be contingent upon their behavior or stable personality characteristics.

Most research has endorsed the notion that external locus of control is associated with higher levels of psychological distress and poorer personal functioning (Brannigan, Rosenberg, & Loprete, 1977; Lefcourt, 1982), as well as lower coping and management of affect (Vickers, Conway, & Haight, 1983). Holder and Levi (1988) studied 166 students using the SCL-90-R and Levenson’s Internal, Powerful Others, and Chance locus of control scales. They noted that both the externality scales were associated with greater psychological distress \( (p < .05). \) Similar findings were reported by Petrosky and Birkimer (1991) in a study of 102 psychology students. They found that externality was positively correlated with suppression \( (r = .29, p < .01) \) and negatively correlated with coping \( (r = .32, p < .01) \).

Conversely, internal control has been associated with emotional well-being and successful coping (Thompson & Spacepan, 1991). Patients with a high internal locus of
control have been found to have greater knowledge about their illnesses (Seeman & Evans, 1962), fewer hospitalizations and health care visits (Krantz, Baum, & Wideman, 1980), and better overall control of their condition (Surgenor, Horn, Hudson, Lunt, & Tennent, 2000).

Research on chronic illness and locus of control has underscored the adaptive value of a relationship between control and objective circumstances. Considerable research has been generated on cancer patients (Worchel, Copeland, & Barker, 1987; Taylor, Lichtman, & Wood, 1984), heart attack and kidney transplant patients (Krantz & Schultz, 1980), major depression patients (Reynaert et al., 1995), and stroke patients and their care providers (Thompson, Bundek, & Sobolew-Shubin, 1990; Thompson, Sobolew-Shubin, Graham, & Janigian, 1989) suggesting that a sense of personal control enhances coping in chronic illnesses.

In a recent study on heart disease, Dracup et al. (2003) investigated the relationship between perceived control and emotional distress in debilitating cardiac illness. Using a cross-section comparative design, they assessed 222 patients from a cardiac transplant center on a number of psychological questionnaires including the Control Attitudes Scale and the Multiple Affect Adjective Checklist. Findings indicated that patients with a greater sense of personal control over their illness had less anxiety ($t = -8.17, p = .0001$), less depression ($t = -7.81, p = .001$), and less hostility ($t = -5.30, p = .00$). In a study of health locus of control and depression, Christensen, Turner, Smith, and Holman (1991) evaluated 96 hemodialysis patients and found that the belief that the illness was under control was correlated with less depression among patients who had not
previously experienced renal failure. Similarly, Schiaffino and Revenson (1992) studied mediational and moderational relationships among perceived control, self-efficacy, and causal attributions in 64 adult rheumatoid arthritis patients. They found that self-efficacy was essential in mediating the connection between perceived control and physical disability. In addition, Helgeson (1992) studied perceived control in 80 adults hospitalized with their first cardiac event. Adjustment was evaluated during hospitalization and during a 3-month follow-up. The findings indicated that patients not only perceived that they had more control over their illness than others ($t = 5.85, p < .001$), but they also demonstrated levels of health locus of control that were related to less psychological stress ($r = -.24, p < .05$) and better psychosocial adjustment to illness ($r = -.31, p < .05$).

Although locus of control has been investigated extensively in the literature, there have been only a limited number of studies which have specifically addressed the relationship between control and asthma. Of these studies, the majority have investigated asthma in children. Hazzard and Angert (1986) evaluated knowledge, attitudes, and behaviors in 80 children with asthma, along with their parents. Their findings indicated that asthma knowledge and internal health locus of control were positively correlated ($r = .31, p = .0003$), as were self-concept scores and adaptive behaviors related to asthma ($r = .43, p = .001$). In a similar study, Gibson, Henry, Vimpani, and Halliday (1995) surveyed high school students on asthma knowledge, attitudes, and quality of life. Twenty-three percent of the students were found to have asthma. While the asthma students in this study were generally low on knowledge about
their disease (14.5%), those with higher internal control knew more about their asthma ($r = .22$, $p = .0001$). In a study of chronically and psychosomatically ill children and adolescents, Steinhausen (1982) found that all of the groups demonstrated greater levels of internal control except for the bronchial asthma group.

Studies on medication maintenance in asthma patients have also confirmed the role of control in fostering compliance. Weinberger (1988) noted that compliance in adolescents can actually be enhanced by including the patient in the decision making process in order to foster an internal locus of control belief.

Self-management programs have also been investigated in terms of control factors. Taggert et al. (1991) conducted pre-test and post-test evaluations on 40 hospitalized asthmatic children (age 6-12 years) following exposure to videotapes, written activity books, and discussions with nurses about their asthma. In addition to an increase in their knowledge of the early warning signs of asthma ($r = 2.53$, $p < .05$), the children also had significant increases in their level of internal control ($r = 2.05$, $p < .05$). These conclusions confirm the earlier findings reported by Taggert, Zuckerman, Lucas, Acty-Lindsey, and Bellanti (1987). In addition, Miles, Sawyer, and Kennedy (1995) found that in a study of 62 children and late adolescents (8 to 26 years of age), older children were more motivated regarding their health, and children who had parents who were knowledgeable about their asthma condition felt more capable of managing their own asthma. The lack of adult studies in this area suggests the need for further investigation of control and mastery issues in adult asthma populations. Many, such as Peterson and Stunkard (1989), believe that the programs aimed at promoting health related behaviors
must emphasize control and self-efficacy. This is particularly true with asthma, where the unpredictable nature of the illness has a direct impact upon the patient's perception of control and competency.

Age of Onset

While the natural history of asthma is variable and often unpredictable, there are some defining qualities of the disease which differentiate children and adolescents from adults. Asthma generally begins in childhood and frequently presents during the first year of life (Reed, 1995). Childhood asthma has been directly linked to allergens in the home (Sporik, Holgate, Platts-Mills, & Cogswell, 1990) and persistent cases have been attributed to mites, pets, and cockroaches (Brunekreef, Groot, & Hoek, 1992), as well as maternal cigarette smoking (Tager, 1986). While some children outgrow their asthma symptoms, others suffer with symptoms throughout their childhood and on into their adult years. Reed noted that while many studies report remission of the disease in up to half of all childhood sufferers, there is always the risk of recurrence of the illness later in life.

Adult onset asthma is difficult to establish since symptoms are often persistent from childhood. Reed (1995) contended that many adults with persistent asthma from childhood suffered from specific allergies to particular aeroallergens, whereas those who developed asthma later, were unlikely to be allergic and more prone to suffer from occupational asthma. Interestingly, those who developed non-allergic adult onset asthma often experienced greater symptoms as well as higher mortality rates (Ulrik, Backer, &
Dirksen, 1992). Kitch, Levy, and Fanta (2000) noted that one of the major problems with late onset asthma was that it may be mismanaged and misdiagnosed. They also indicated that it was difficult to determine if the same factors play a role in the adult disease process.

Conflicting information currently exists regarding the age of onset of asthma and the long term severity and prognosis of the disease (Bierman & Pearlman, 1988). Mrazek, Schuman, and Klinnert (1998) conducted a longitudinal investigation of 150 genetically at-risk asthmatic children and followed their development and asthma symptoms for 6 years. They compared them with 126 non-asthmatic children and found that those children diagnosed with asthma from an early age had significantly more behavioral problems including sleep disturbances ($p < .001$) and depressed mood ($p < .05$), as well as elevated fearfulness ($p = .12$) when compared to controls. Klinnert et al. (2001) studied 145 children who were at risk for asthma and followed them until they were eight years of age. They found that 28% of the children developed asthma symptoms by age eight and they concluded that psychosocial factors may contribute to asthma onset and persistence in childhood. Despite these findings, both studies were unclear as to the long-term consequences of such behavioral/social problems on adult symptoms. Indeed, there has been limited investigation into the actual differences between childhood sufferers and those with adult onset asthma, and virtually no studies which have addressed the emotional impact of early onset versus the adult onset of asthma symptoms. Therefore, while the age of onset of asthma has already been determined to play a significant role in the physical manifestation of the disease, it is also
quite possible that the length of time a patient suffers from asthma may affect their overall emotional and psychological well being.

_Asthma Severity_

In addition to the critical relationship between specific emotional factors and asthma, the severity of the actual asthma condition is influenced by numerous physiological determinants. Consistent with the acknowledgment of significant heterogeneity in asthma symptoms, there is currently no universally accepted measure for classifying asthma severity (Wahlgren et al., 1997). Recently, the Joint Task Force on Practice Parameters (1995) reviewed all of the existing classification systems focusing on pulmonary function, functional limitation, symptom measures, bronchial hyperresponsiveness, emergency room visits and hospitalizations, and use of prescribed asthma medications. They concluded that a single measure of asthma severity was impossible, because asthma patients varied so dramatically in their symptom presentation.

Many investigators however, believed that a consistent severity measure is critical both for proper epidemiological as well as clinical research (Wahlgren et al., 1997). Without such a standard, it is impossible to compare studies or even to replicate findings. Silverglade et al. (1993) have suggested that the failure to address the issue of severity in most of the existing research may be the reason for such dramatic inconsistencies in the findings.

The description of levels of asthma severity was based on a combination of symptoms of lung functioning and treatment requirements, as well as the length of illness
and frequency of exacerbations (O'Byrne & Thomson, 1995). The severity of asthma episodes may vary along a continuum from intermittent and mild, to severe and potentially life-threatening. Recently, the National Heart, Lung, and Blood Institute devised a set of guidelines for the diagnosis and treatment of asthma based on a specific system of classification (Expert Panel Report 2, 1997). This system categorized patients into mild intermittent, mild persistent, moderate persistent, and severe persistent asthma groups.

In addition to aiding in the classification of patients according to the physiological presentation of symptoms, asthma severity has also been evaluated in terms of the overall influence on the emotional adjustment of the patient. From a psychological perspective, there has been some research suggesting a connection between asthma severity and increased psychopathology. As mentioned previously, Garden and Ayres (1993) studied 20 brittle asthma patients and compared them with a matched group of 20 less severe asthmatics on a number of questionnaires and interviews. Findings indicated that the brittle asthma patients had greater psychiatric morbidity when compared to the control group ($p < .02$), and a significantly greater number of life events related to asthma ($p < .001$). Vilà, Nollet-Clemenccon, deBlieck, Mouren-Simeoni, & Scheinmann (1998) evaluated 94 severe asthmatic children who were paired with equal numbers of non-asthmatic children, and matched them for age, gender, and socioeconomic status. They found that in asthmatic children, those diagnosed with anxiety had significantly higher anxiety scores than either the nonanxious ($p = .0005$) or the nonasthmatic groups ($p = .0005$). Similarly, Mrazek (1992) found that severely asthmatic children had a greater
prevalence of depression and anxiety disorders. In a study of irrational beliefs and emotionality, Silverglade et al. (1993) divided 129 asthmatic adolescents into three groups based on the severity of their asthma symptoms, and compared them both with each other and with a group of 74 healthy, nonasthmatic adolescents. They found significant differences between the moderate and severe asthma group and the mild and non-asthmatic healthy group, $F(3, 187) = 1.71, \ p < .05$. Disease severity was found to be directly associated with levels of anxiety ($p < .0001$), depression ($p < .001$), and hostility ($p < .035$), as well as with a lack of control of emotions. These findings were confirmed in a study by Vamos and Kolbe (1999) which concluded that severe asthmatics had significantly more distress, with greater anxiety even between actual attacks. In an interesting study by Plutchik et al. (1978), 40 asthma clinic patients subjectively rated their asthma severity. Findings indicated that the subjective measures of asthma severity, not the objective measures by physicians, were highly correlated with a significant number of psychological variables such as fearfulness, anger, disgust, and sadness ($p < .05$).

Other investigators have postulated less support to the connection between asthma severity and psychological symptoms. ten Brinke, Ouwerkerk, Bel, and Spinboven (2001) investigated 90 severe asthmatic outpatients and 37 mild asthma outpatients and compared them on a number of psychological characteristics using self-report questionnaires. Overall, they found no significant difference in the psychopathology or personality of the two groups, except on an external control orientation ($p < .005$), which indicated less trust in physicians and medication regimes by the severe asthmatics when
compared to the mild asthma group. Between-group differences were again, only noted for the external locus of control orientation, $F (1, 111) = 4.41, p < .05$. Similarly, ten Brinke, Ouwerkerk, Zwinkerman, et al. (2001) compared severe asthmatic outpatients both with psychiatric disorder (cases) and without psychiatric disorder (noncases) in terms of their level of healthcare utilization. The level of psychological dysfunction was actually found to be more of a determining factor in increased emergency visits (71% cases versus 31% noncases, $p < .01$), exacerbation (92% cases versus 57% noncases, $p < .02$), and hospitalization (19% cases versus 5% noncases, $p < .04$), than the actual severity of the asthma symptoms. In addition, Wamboldt, Fritz, Mansell, McQuaid, and Klein (1998) found that severely asthmatic children do not report greater levels of anxiety than mild or moderate asthmatic children. They concluded that severe asthma in children may cause their parents increased stress and they in turn may report higher levels of internalizing symptoms in their children.

*Literature Conclusion*

It is evident that the relationship between emotional factors and asthma remains complex, multidimensional, and as of yet, not fully understood. Both depression and anxiety have been studied extensively in the asthma literature; however, much of the existing research is fraught with inconsistencies and limitations. While many investigators have indicated that depression and anxiety contribute significantly to the psychopathology in asthma patients (Henry et al., 1993; Rubin, 1993; Yellowlees et al., 1988), other researchers have found little relationship between psychiatric symptoms and
the presence of asthma (Janson et al., 1994; Rocco et al., 1998). These inconsistencies have been attributed to problems in classification, lack of standardization, flaws in design, as well as inappropriate patient selection (Garden & Ayres, 1993; Moran, 1991). In addition, many of the early studies conducted in the field of asthma research were aimed exclusively at children, making it virtually impossible to determine the unique relationship between emotions and asthma in adult populations. Nevertheless, most studies do suggest a relationship between emotions and asthma.

In the area of locus of control, there have been a limited number of studies which have addressed asthma, particularly in adults. This is significant because internal locus of control has been associated not only with better health outcomes (Peterson & Stunkard, 1989), but also with greater information seeking, increased medication compliance, and more self-initiated preventive care (Seeman & Seeman, 1983; Wallston & Wallston, 1978). Because a sense of internal control may ultimately determine whether an individual stays healthy or becomes ill (Wallston, 1992), research in the area of locus of control may provide a critical link in terms of self-management, prevention, and quality of life in asthma patients.

Therefore, the integration of all of the psychological and physical aspects of asthma is an essential element of the research. The challenge is to identify this unique connection through the use of well-controlled, adult studies. Additionally, such studies must take into consideration the severity of illness, with emphasis on the classification of individuals by their distinctive symptoms.
Chapter III

Methodology and Procedures

This chapter on methods and procedures is divided into five sections. The first section describes the eligibility of subjects for the study, with emphasis on selection criteria and group assignment. The second section includes a description of the research procedures and details the data collection process. The third section on psychometric tools reviews the instrumentation used to measure the dependent variables and focuses on the reliability and validity of the scales. The fourth section outlines the actual research design that was employed in the study. The final section presents an overview of the statistical analysis that was used to test each hypothesis proposed in the study.

Subjects

The subjects in the present study consisted of 150 adult female asthma patients between the ages of 18 and 55. Based on the recommendations of Stevens (1996), the sample size reflects an expectation of adequate power (.70 - .80), as well as the anticipation of a moderate effect size. Subjects were selected based on their history of asthma and were subsequently classified into mild, moderate, and severe asthma groups based on the severity of their asthma symptoms. The classification of subjects into asthma groups was conducted by a licensed pulmonologist who followed the National Institutes of Health guidelines for the “Classification of Asthma Severity” (Expert Panel
Report 2, 1997). This classification system allowed for the categorization of patients into mild intermittent, mild persistent, moderate persistent, and severe persistent asthma groups. For the purpose of the present research, the mild intermittent and mild persistent groups were combined to form one category, since patients with mild intermittent asthma rarely present to a physician’s office and therefore would have been extremely limited in number. In addition, throughout the study, the asthma groups were referred to as mild, moderate, and severe.

Each asthma group consisted of 50 subjects. The mild asthma group was composed of adults who had asthma symptoms less than once a day, used prescribed medications or a bronchodilator only when symptoms escalated, had exacerbations which were brief and may have only slightly affected their activity level, and experienced nocturnal asthma symptoms less than twice a month. The moderate asthma group included adults who had daily asthma symptoms, had exacerbations which affected their activity level, required a daily controller medication, and experienced nocturnal symptoms more than once a week. The severe asthma group consisted of adults who had continuous asthma symptoms and frequent exacerbations, required multiple daily medications to maintain control, had limited physical activity, and experienced frequent nighttime symptoms.

The sample was screened for any physical factors which might have precluded their completion of the study such as severely impaired vision, neurological limitations, or any psychiatric illness which might have rendered them incapable of understanding the purpose of the research. All subjects were required to have a minimum of a high school
education to ensure adequate comprehension of the study forms and questionnaires. Subjects determined to have less than a high school education were not included in the study. In addition, all subjects spoke English as their primary language. Subjects determined to have another language as their primary language were not included in the study.

The treating physicians were located in several suburban communities in Northeastern New Jersey. A total of six physicians participated in the study. All subjects were active patients in their practices.

Procedure

The physicians were contacted by the investigator, and a meeting was arranged with each of them to discuss the proposed research. All physicians were given a letter which specified the purpose and format of the study (Appendix A), as well as a copy of the research proposal. If a physician decided that they were interested and wanted to participate, a letter of agreement (Appendix B) was signed and returned to the researcher, and study packets were distributed to the physician. The physician subsequently presented all study packets to the office nurse/secretary who was responsible for their distribution. In order to preserve each subject's right to privacy and to protect the confidentiality of the data, all subjects were identified by a random code number only, which appeared at the top of each study packet, as well as at the top of each of the enclosed forms and questionnaires.

During a regularly scheduled office visit, female asthma patients were asked by
the office nurse/secretary if they would be interested in participating in a research study
about asthma. At that time, subjects were presented with a letter of solicitation
(Appendix C), which provided them with specific information about the study. If the
patient agreed, they were then given a packet which they were asked to fill out while in
the waiting room. Subjects first read a set of instructions regarding the materials
presented to them (Appendix D). Next, they completed the background information form
(Appendix E). Subjects then completed the questionnaires which included the Beck
Depression Inventory (Beck et al., 1961), the State-Trait Anxiety Inventory (Spielberger,
1983), and the Multidimensional Health Locus of Control Scale, Form C (Wallston et al.,
1994). Once the subject had finished filling out all forms and questionnaires, they were
asked to place the sealed study packet into a drop box which was available in the waiting
room and labeled “Research Drop Box”. Each of the drop boxes was emptied on a daily
basis by the researcher.

After the study packets had been collected, subjects were classified into one of
the three asthma groups. In order to ensure uniformity of diagnosis across physicians, a
standardized classification form (Appendix F) was filled out by a licensed pulmonologist
who was blind to the number of subjects required for each of the asthma groups. The
classification form was representative of the system proposed by the National Institutes of
Health (Expert Panel Report 2, 1997). Based on the patient’s asthma history as recorded
on the background information form, the physician classified each of the subjects into
either the mild, moderate, or severe asthma groups and then placed the classification form
in the subject’s study packet. Once the study packet was completed by the subject,
scored by the researcher, and classified by the physician, the packet was sealed and stored in a locked filing cabinet in the researcher’s office.

The study continued until the required number of study packets was completed for each group. Partially completed study packets and study packets with missing data were eliminated from the final analysis.

*Research Instruments*

The following instruments were employed in the study and administered according to the standardized instructions:

1. Beck Depression Inventory (BDI)
2. State-Trait Anxiety Inventory, Form Y (STAI)
3. Multidimensional Health Locus of Control Scale, Form C (MHLC)

*Beck Depression Inventory*

The Beck Depression Inventory (BDI) (Beck et al., 1961) is a self-report method of assessing the severity of depression. It was initially aimed at measuring the symptoms and attitudes presented by depressed psychiatric patients. Since that time, it has been used extensively across a variety of cultures (Shaw, Vallis, & McCabe, 1985) and in various clinical settings (Enns, Cox, Parker, & Guertin, 1998). It has been used to measure depression in normal individuals (Steer, Beck, & Garrison, 1986), as well as in psychiatric populations (Piotrowski, Sherry, & Keller, 1985). The BDI takes only 10 to 15 minutes to administer and is currently one of the most widely used measures of depression. Although the Beck Depression Inventory-II (Beck, Steer, & Brown, 1996)
has emerged in recent years as an updated version of the BDI, for the purposes of the current study, the original BDI was employed because it continues to be more widely used on chronically ill populations (Aben, Verhey, Lousberg, Lodder, & Honig, 2002; Cassidy, Tomkins, Hardiman, & O'Keane, 2003; McKellar, Clark, & Shriner, 2003) and more specifically, with asthmatics (Gillaspy, Hoff, Mullins, Van Pelt, & Chaney, 2002; Kovacs, Stauder, & Szemak, 2003). In addition, the BDI-II, which is highly correlated with earlier versions of the inventory, is believed to produce no meaningful interpretive differences (Beck, Steer, Ball, & Ranieri, 1996).

The BDI is a 21-item inventory, with four self-evaluative statements per item, which respondents rate on a scale from 0 to 3. The rated responses yield a total score which can range from 0 to 63. The scores are then categorized as follows: 0-9 ($M = 10.9$, $SD = 8.1$) indicates no depression or minimal depression, 10-18 ($M = 18.7$, $SD = 10.2$) suggests mild to moderate depression, 19-29 ($M = 25.4$, $SD = 9.6$) reflects moderate to severe depression, and 30-63 ($M = 30.0$, $SD = 10.4$) indicates severe depression (Beck, 1967).

The BDI is a clinically derived scale which was originally tested on a random sample of 226 psychiatric patients (Beck et al., 1961). Of the sample, 33.6% were inpatients, 66.4% were outpatients, 40.7% were males, and 59.3% were females. Patients with organic brain damage and mental deficiency were automatically excluded. A second sample, consisting of 183 patients, was used as a replication group. This sample was composed of 34.4% inpatients and 65.6% outpatients, 37.2% males and 62.8% females. Following administration of the BDI, patients were rated by independent psychiatrists.
regarding the “Depth of Depression” (none, mild, moderate, or severe).

*Reliability of the Beck Depression Inventory.* The initial study of the BDI (Beck et al., 1961) yielded high levels of internal consistency, with all categories showing a significant relationship to the overall score of the inventory ($p < .001$) except for weight loss which was significant at the .01 level. In a second evaluation of internal consistency, 97 cases in the first sample were selected and analyzed. Split-half reliability with the Pearson $r$ yielded a reliability coefficient of .86, and this rose to .93 with the Spearman-Brown correction. A variation on the test-retest method was employed using a group of 38 patients at two separate times, and it was found that changes in inventory scores paralleled changes in the clinical depth of depression.

More recent studies of the reliability of the BDI have generally found the inventory to possess good internal consistency, with alpha coefficients higher than .75 (Richter, Werner, Heerlein, Kraus, & Sauer, 1998). A meta analysis of established internal consistency research has yielded ranges from .73 to .92 with a mean of .86 (Beck, Steer, & Garbin, 1988). Split-half reliability coefficients have been found to range from .58 to .93 (Gallager, Nies, & Thompson, 1982; Reynolds & Gould, 1981; Strober, Green, & Carlson, 1981). Studies of test-retest reliability have reported ranges from .48 to .86, depending on the retesting interval (Beck et al., 1988). Beck (1970) has always contended that the test-retest reliability is not an adequate method for testing the reliability of the BDI since large temporal distances may underestimate reliability due to therapeutic changes, and short distances may overestimate reliability due to memory effects.

A few recent efforts using multiple administrations have yielded less stability over
time. Ahava, Iannone, Grebstein, and Schirling (1998) studied 197 undergraduate students during 7 weekly administrations over an 8-week period and found that with multiple administrations, BDI scores systematically decreased over time, $F(6, 894) = 17.39, p < .001$. Although statistical problems, such as regression to the mean, along with methodological limitations, have been sighted as the possible cause for their findings, similar instability of scores over time has been noted by other investigators (Hatzenbuehler, Parpal, & Matthews, 1983; Richter et al., 1998; Zimmerman, 1986) and suggest a potential shortcoming of the inventory. However, for the most part, studies have found the scores on the BDI to be relatively stable and reliable over time.

*Validity of the Beck Depression Inventory.* Validity studies have generally been supportive of the BDI. In terms of content validity, the composition of the BDI was achieved by clinician consensus regarding common symptoms of depressed patients, and six of the nine criteria categories in the DSM-IV are included in the inventory. In the initial evaluation of the BDI, Beck et al. (1961) found that for each increment in depression, there was a progressively higher mean score, with differences at the < .001 level of significance. The power of the inventory to discriminate between specific “Depth of Depression” categories yielded $p$ values of < .01 in the original group and < .02 in the replication group. More recently, Schotte, Maes, Cluydts, DeDoncker, and Cosyns (1997) studied 338 unipolar depressed inpatients who had been diagnosed using the DSM-III. After establishing subcategories of depression, they found significant effects for the BDI, $F(2, 310) = 13.30, p < .00001$, the BDI/somatic scale, $F(2, 318) = 19.73, p < .000001$, and to a lesser extent the BDI/psychological scale, $F(2, 316) = 3.59, p < .03$,
suggesting similar support for the construct validity of the BDI.

Initial tests to establish concurrent validity obtained highly significant correlations between the scores on the BDI and the clinical ratings on the “Depth of Depression”. The original group yielded correlations at the .65 level and the replication group at the .67 level, both significant at the <.01 level. Recent studies of concurrent validity with other self-rating scales have generally reported moderate to high correlation coefficients. Robinson and Kelley (1996) administered the BDI, the State-Trait Anxiety Inventory, and the Nowicki-Strickland Locus of Control Scale to 211 undergraduate students and found that the BDI significantly correlated with generalized anxiety (.69, p < .05), locus of control (.49, p < .05) and self-concept (.35, p < .05). Similarly, Schneider (1998) recently studied 54 cancer patients in an attempt to establish the association between the Multidimensional Fatigue Inventory-20 and the BDI. The findings indicated that scores on the BDI significantly correlated with scores on the Multidimensional Fatigue Inventory, with correlations ranging from .52 to .61 (p < .001). Validity coefficients have been found to be in the range of .38 to .76 when BDI scores were correlated with other measures of depression as well as measures of hopelessness (Robinson & Kelley, 1996). Dinning and Evans (1977) also found .46 to .70 correlations when the BDI was compared with the Symptom Checklist-90 (SCL-90).

In general, the BDI has been shown to possess high internal consistency in psychiatric and nonpsychiatric populations, high content and convergent validity, and a capacity for good sensitivity to change. Although a number of shortcomings such as high item difficulty, insufficient norming data, and questionable factorial and discriminant
validity have been noted in the literature (Richter et al., 1998), overall, the BDI has been shown to possess adequate psychometric properties.

*State-Trait Anxiety Inventory*

The State-Trait Anxiety Inventory (STAI) (Spielberger, 1983) is a self-report scale based on the principles of state and trait anxiety first proposed by Cattell (1966). Emotional states are perceived as rather transitory and are characterized by apprehension, tension, and worry. Personality traits are seen as relatively enduring differences among people that influence not only the way they view the world, but also the way in which they respond to it. The STAI has been used extensively in clinical practice and has frequently been enlisted in medical, surgical, and psychosomatic research (Spielberger, 1983). It has also been employed previously to investigate the role of anxiety in asthma (Alexander, 1972; Kurata, Glousky, Newcomb, & Easton, 1976).

The STAI consists of two separate 20-item scales designed to measure transient and enduring levels of anxiety, respectively. The State-Anxiety scale asks respondents to indicate how they feel at the moment using a 4-point scale to answer the 20 items. The Trait-Anxiety scale asks respondents to indicate how they generally feel with respect to each of the 20 items, again using a four-point scale. The original STAI, known as Form X, was replaced by Form Y in 1980. Form Y is apparently a purer measure of anxiety, includes fewer items related to depression, and has an improved factor structure compared to Form X (Okun, Stein, Bauman, & Johnson-Silver, 1996). While much of the research has been based on Form X, correlations between Form X and Form Y were uniformly high, ranging from .96 to .98.
The STAI (Form Y) was standardized and validated on more than 5,000 subjects, including working adults, college students, high school students, and military recruits. The working adults were represented by 1,838 Federal Aviation employees (1,387 males; 451 females). The college students consisted of 855 students in an introductory psychology class (324 males; 531 females). The high-school students consisted of 424 tenth-grade students (202 males; 222 females). The military recruits were comprised of two samples: 1,701 male Air Force recruits and 263 Navy recruits (192 males; 71 females).

Reliability of the State-Trait Anxiety Inventory. The STAI has been found to be a relatively reliable and internally consistent measure (Spielberger, 1983). The stability coefficients for the STAI were based on two groups of more than 300 high school students who were administered the inventory in a classroom situation. The test-retest reliability for the Trait-Anxiety scale ranged from .65 to .75, with a median reliability coefficient of .695. For the State-Anxiety scale, the stability coefficients were relatively low, ranging from .34 to .62, with a median reliability coefficient of only .33. This low stability coefficient is expected for the State-Anxiety scale because situational factors may influence the response at the time of testing.

Since anxiety states are transitory, alpha coefficients are actually considered to be more reliable measures of State-Anxiety than test-retest correlations. With the exception of one score, all State-Anxiety alpha coefficients for the normative samples were above .90 with a median coefficient of .93. Alpha coefficients for Trait-Anxiety were also relatively high, with a median coefficient of .90.
Additional evidence of internal consistency was derived from item-remainder correlations computed for the normative sample. The median State-Anxiety correlations were .55 for high school students, .59 for college students, .61 for military recruits, and .63 for working adults. The Trait-Anxiety correlations were .54, .57, .52, and .56 respectively. Therefore, the stability and internal consistency of the STAI was quite high.

Validity of the State-Trait Anxiety Inventory. The construct validity of the Trait-Anxiety scale was tested by comparing normal subjects from the normative sample with 461 neuropsychiatric patients (Spielberger, 1983). With the exception of one group (character disorder), all other neuropsychiatric groups had higher Trait-Anxiety scores ($M = 46.62$) than the normal subjects ($M = 38.39$), suggesting that the STAI sufficiently discriminated between normals and psychiatric patients with anxiety. The Trait-Anxiety scores were notably lower, particularly in the character disordered group ($M = 40.32$), where the lack of anxiety was actually an essential component in defining the condition. The STAI was also found to identify nonpsychiatric patients with emotional problems.

The construct validity of the State-Anxiety scale was further evaluated in two studies that employed high and low stress conditions to undergraduate students (Spielberger, Gorsuch, & Lushene, 1970). In the first study, more than 900 undergraduates were first given the State-Anxiety scale and then were asked to respond according to the way they would feel “just prior to the final exam in an important course.” The mean State-Anxiety scores achieved critical ratios of 24.14 for males and 42.13 for females, with point biserials of .60 and .73 respectively, suggesting that the level of State-Anxiety was directly associated with the experimental conditions. In a second study, 197
undergraduate students were given the State-Anxiety scale under four different experimental conditions including: (a) a normal test condition, (b) following a 10-minute relaxation session, (c) after working on what was described as a “relatively easy IQ test”, and (d) following a stressful movie. The scores increased based on the amount of stress in each of the experimental conditions, with the lowest mean scores (males = 32.70, females = 29.60) attained during the relaxation condition, and the highest mean scores (males = 50.03, females = 60.94) achieved after viewing the stressful movie. The fact that some items were sensitive to State-Anxiety at low levels of stress and some to higher levels of stress is consistent with the test-theory concept of item-intensity specificity (Spielberger et al., 1970).

In terms of the content validity of the STAI, Okun, et al. (1996) compared the inventory with the criteria for generalized anxiety disorder and major depressive episode. The STAI measured five out of the eight areas for generalized anxiety disorder, supporting its applicability and validity.

The STAI has been found to have relatively good concurrent validity. The Trait-Anxiety scale correlated well with the IPAT Anxiety Scale (Cattell & Scheier, 1963) and the Taylor Manifest Anxiety Scale (Taylor, 1953), with correlations ranging from .85 to .73. In addition, the brevity of the Trait-Anxiety scale was a major advantage over the other two scales.

The STAI also correlated well with other personality measures, suggesting good convergent and divergent validity. Stanley, Beck, and Zebb (1996) studied 50 older adults with a diagnosis of general anxiety disorder and compared them with a subgroup of
normal controls on the STAI, the Worry Scale, and the Padua Inventory. They found positive correlations on measures of trait anxiety (.40, \( p < .01 \)) worries, (.46, \( p < .001 \)), and obsessive compulsive traits (.44, \( p < .01 \)) in older individuals diagnosed with anxiety. Correlations of the STAI scales with the MMPI were relatively consistent as well. In a two-part study comparing separate groups of hospitalized psychiatric patients (Spielberger, 1983), findings revealed that the correlations between the STAI scale and the individual MMPI scales were comparable. In the first study, 129 patients revealed correlations ranging from -.64 to .79 with the State-Anxiety scale and -.63 to .81 with the Trait-Anxiety scale. In the second study, 79 patients revealed correlations of -.46 to .48 with the State-Anxiety scale and -.60 to .68 with the Trait-Anxiety scale. The Cornell Medical Index also correlated (.70) with both the State-Anxiety and Trait-Anxiety scales, suggesting an association between numerous medical symptoms and high STAI scores. Novy, Nelson, Goodwin, and Rowzee (1993) also examined the correlation of the STAI with the MMPI in an investigation of 285 men and women from different ethnic groups. They found that the STAI scales were highly correlated with the MMPI basic scales, particularly the Depression (D) scale (.52 - .72) and the Psychasthenia (Pt) scale (.50 - .76). The STAI has also been found to be significantly correlated with the Mooney Problem Checklist (Mooney & Gordon, 1950), the Jackson Personality Research Form (Jackson, 1967), and the Edwards Personal Preference Schedule (Edwards, 1954).

In general, the STAI has been found to possess sufficient internal consistency, as well as adequate construct, concurrent, convergent, and divergent validity.
The Multidimensional Health Locus of Control Scale

The Multidimensional Health Locus of Control Scale, MHLC (Form C) (Wallston et al., 1994) is a self-report, general purpose scale, based on the original Multidimensional Health Locus of Control scales (Wallston et al., 1978). Health locus of control refers to an individual's belief regarding the center of control of their illness. Those with internal locus of control believe that their own behaviors significantly influence their health, whereas those with external locus of control feel that their health is influenced by other people or is a result of fate, chance, or luck. The original MHLC maintained the internality subscale, and split externality into Powerful Others and Chance subscales.

Unlike the original MHLC scales which measured general health behavior, Form C was designed to measure condition-specific locus of control. Wallston et al. (1994) found that individuals with chronic conditions had significant difficulty answering the more general scales and needed a scale which was able to address their specific conditions. Form C has been adapted for this purpose and can be utilized with any pre-existing medical or health-related condition.

The initial version of MHLC (Form C) consisted of 24 items, with eight Internality, eight Chance, and eight Powerful Other items. Following a factor analysis, the scale was reduced to two 6-item scales for Internality and Chance, and two distinct three-item scales for Powerful Others, which included Doctors and Other People. Form C now consists of 18 items designed to target specific health problems. In order to test for an identified medical condition, the word "condition" in each of the 18 items was
replaced by the medical condition under study. Subjects are asked to answer each item along a scale which ranges from strongly disagree to strongly agree.

The norming data for the MHLC (Form C) was achieved from 588 patients with four different medical conditions (Wallston et al., 1994). These included 273 rheumatoid arthritis patients, 111 chronic pain patients, 111 Type I and II diabetic patients, and 93 cancer patients.

Reliability of the Multidimensional Health Locus of Control Scale. The MHLC (Form C) has been found to have adequate internal consistency, with alpha reliabilities ranging from .71 to .87 for the Internality and Chance subscales, and from .70 to .85 for the Doctors and Other People subscales (Wallston et al., 1994). While these second set of alphas were lower due to shorter subscales, they still achieved acceptable levels for a three-item subscale.

Test-retest reliabilities were found to yield moderate to high stability coefficients for the arthritis and chronic pain samples, with coefficients ranging from .40 to .80. However, for the pain sample, there were lower stability coefficients on the Internality, Chance, and Doctors subscales, particularly during an intervention period.

Validity of the Multidimensional Health Locus of Control Scale. The construct validity of the MHLC was tested by the presence of an intervention in the chronic pain sample, which consisted of a behaviorally-oriented pain management program (Wallston et al., 1994). This program was designed to weaken the subjects' beliefs about pain helplessness, with the expectation that internal beliefs would be strengthened and external beliefs would be weakened. The arthritis patients were given no such intervention.
Findings indicated that the mean internality scores for the pain patients increased, whereas, their mean externality scores decreased. Raja, Williams, and McGee (1994) also studied the external validity of the MHLC by evaluating 772 mothers of children taking part in a longitudinal study of health, development, and behavior. Their findings indicated that MHLC clusters can be identified and are positively correlated ($p < .01$).

Concurrent validity of the MHLC (Form C) was demonstrated by moderate correlations with MHLC Form B subscales. Internality, Chance, and the two Powerful Other subscales demonstrated modest correlations ranging from .38 to .65 when compared to Form B. The MHLC (Form C) Other People and Form B Chance subscale also showed a significant correlation of .30. MHLC (Form C) did not tend to correlate significantly with noncounterparts from Form B, thus demonstrating the discriminant and convergent validity of the instrument. A significant relationship was also noted between MHLC (Form C) and the Levenson’s I, P, and C scales for Internality, Other People, and Chance items, with modest correlations ranging from .35 to .50. Overall, the validity evidence strongly suggested that the MHLC (Form C) is a valid measure of condition-specific locus of control beliefs.

Research Design

Since the current investigation had more than one level of the independent variable and multiple dependent variables, a factorial research design (Campbell & Stanley, 1963) was employed. This design allowed for the analysis of the relationship between the levels of the independent variable (mild, moderate, and severe asthma
groups) and the degree of depression, anxiety, and locus of control as assessed by the dependent measures (BDI, STAI, and MHLC).

Statistical Procedures

A number of statistical procedures were utilized in the analysis of the data. First, basic descriptive statistics were generated on the group variables. A Multivariate Analysis of Variance (MANOVA) was then computed to analyze the relationship between the three treatment groups and the dependent variables. The treatment groups were identified as mild, moderate, and severe asthmatics and the dependent variables were scores on the BDI, the STAI, and the MHLC. Univariate $F$ tests for significant group differences were calculated, along with multiple comparison tests to clarify exactly which means were different.

Second, a Discriminant Function Analysis was performed to examine the degree to which the asthma groups differentiated between the BDI, the STAI, and the MHLC. Discriminant Function Analysis was also utilized as a means of classification so that subjects could be accurately placed into the groups of the dependent variable from a given combination of the independent variable.

Third, following descriptive statistics for the 'age of onset' variable, a MANOVA was computed to analyze the relationship between the age of onset variable and the dependent measures. The age of onset variable was divided into four groups which were identified as: Age 1 to 12, Age 13 to 18, Age 19 to 24, and Age 25 and up. Again, the dependent measures were scores on the BDI, the STAI, and the MHLC. Univariate $F$
tests as well as post-hoc comparison tests were also computed.

An alpha level of .05 was used for all statistical tests.
Chapter IV

Results

The current chapter presents a summary of the data analysis and is divided into six sections. The first section provides descriptive statistics on the Beck Depression Inventory (BDI), the State-Trait Anxiety Inventory (STAI), and the Multidimensional Health Locus of Control Scale (MHLC) for the mild, moderate, and severe asthma treatment groups. The second section is a review of the MANOVA of the differences between the asthma groups on the dependent measures and includes the results of the Univariate $F$ tests on each of the groups. Multiple comparison tests are also presented. The third section examines the discriminant function analysis for the differentiation and classification of the variables. The fourth section presents descriptive statistics for the ‘age of onset’ variable on the BDI, the STAI, and the MHLC. The fifth section is a review of the MANOVA for the ‘age of onset’ variable. This section also presents Univariate $F$ tests for the age of onset variable, along with post-hoc comparison tests. The final section is a summary of the proposed hypotheses and the subsequent results.

Descriptive Statistics for the Three Asthma Groups on the Dependent Measures

Descriptive statistics for the mild, moderate, and severe asthma groups on the BDI, the STAI, and the MHLC are presented in Table 1. The results revealed that with respect to the BDI, subjects from the severe asthma group had the highest average,
<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>n</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BDI</strong></td>
<td>Mild</td>
<td>50</td>
<td>9.20</td>
<td>8.09</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>50</td>
<td>6.78</td>
<td>5.87</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>50</td>
<td>10.48</td>
<td>8.99</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>8.82</td>
<td>7.86</td>
</tr>
<tr>
<td><strong>STAI-State Scale</strong></td>
<td>Mild</td>
<td>50</td>
<td>41.52</td>
<td>13.27</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>50</td>
<td>36.28</td>
<td>10.62</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>50</td>
<td>43.14</td>
<td>11.72</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>40.31</td>
<td>12.20</td>
</tr>
<tr>
<td><strong>STAI-Trait Scale</strong></td>
<td>Mild</td>
<td>50</td>
<td>41.72</td>
<td>12.29</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>50</td>
<td>36.76</td>
<td>9.42</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>50</td>
<td>41.36</td>
<td>11.66</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>39.95</td>
<td>11.34</td>
</tr>
<tr>
<td><strong>MHLC-Internal Scale</strong></td>
<td>Mild</td>
<td>50</td>
<td>21.68</td>
<td>5.86</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>50</td>
<td>23.52</td>
<td>6.07</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>50</td>
<td>20.54</td>
<td>6.90</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>21.91</td>
<td>6.37</td>
</tr>
<tr>
<td><strong>MHLC-Chance Scale</strong></td>
<td>Mild</td>
<td>50</td>
<td>14.18</td>
<td>5.33</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>50</td>
<td>14.12</td>
<td>6.71</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>50</td>
<td>14.50</td>
<td>5.55</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>14.27</td>
<td>5.86</td>
</tr>
<tr>
<td><strong>MHLC-Doctors Scale</strong></td>
<td>Mild</td>
<td>50</td>
<td>13.56</td>
<td>3.11</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>50</td>
<td>14.98</td>
<td>2.69</td>
</tr>
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<td></td>
<td>Severe</td>
<td>50</td>
<td>13.74</td>
<td>3.65</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>14.09</td>
<td>3.22</td>
</tr>
<tr>
<td><strong>MHLC-Other People Scale</strong></td>
<td>Mild</td>
<td>50</td>
<td>6.84</td>
<td>3.41</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>50</td>
<td>6.94</td>
<td>3.50</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>50</td>
<td>7.86</td>
<td>3.95</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>7.21</td>
<td>3.63</td>
</tr>
</tbody>
</table>
followed by the mild asthma group and moderate asthma group respectively. The difference between the severe asthma group and the moderate asthma group suggested greater depressive feelings in those subjects suffering from more severe symptoms. On the STAI-State Scale, the severe asthma group achieved the highest average score, followed by the mild and moderate groups. On the STAI-Trait Scale, the mild asthma group had the largest overall average, followed by the severe asthma and moderate asthma groups respectively. Therefore, while the severe asthma group reported the highest level of state anxiety, they did not score the highest on trait anxiety.

Scores on the MHLC varied considerably among the different MHLC subscales. On the MHLC-Internal Scale, the moderate asthma group had the highest overall score, followed by the mild and severe asthma groups. The severe asthma group had the largest mean score on the MHLC-Chance Scale, however, it was only slightly higher than the mean scores achieved by the mild and moderate groups. On the MHLC-Doctors Scale, the moderate asthma group yielded the highest score, followed by the severe asthma group, and the mild asthma group. Finally, on the MHLC-Other People Scale, the severe asthma group had the highest average, followed by the moderate and mild asthma groups respectively. Overall, the largest differences in the group means occurred between the severe asthma group and the mild asthma group. While the severe asthmatics had the highest levels of chance and other people locus of control, it was the moderate asthmatics who scored the highest on the internal and doctors locus of control.
Multivariate Analysis of Variance Between Three Treatment Groups on the Dependent Measures

A MANOVA was used to test for differences between groups (mild, moderate, and severe asthma) on all dependent variables simultaneously (BDI, STAI, and MHL.C). A Box's M test was used to test the assumption of equal variance. The Box's M test was statistically significant, (Box's M = 88.35, F = 1.47, p < .013) which suggests that the assumption of equal variance had been violated. However, because the sample sizes within each group were identical, the corresponding Multivariate F test should be robust to violations of this assumption. Therefore, no transformation of the data was deemed necessary.

Overall Multivariate tests, which are presented in Table 2, were significant. This suggests that there were significant between group differences among the mild, moderate, and severe asthma groups when all of the variables (BDI, STAI-State Scale, STAI-Trait Scale, MHL.C-Internal Scale, Chance Scale, Doctors Scale, and Other People Scale) were considered simultaneously.

Table 2
Multivariate Analysis of Variance: Differences Between Asthma Groups on the Depression, Anxiety, and Locus of Control Measures

<table>
<thead>
<tr>
<th>Test Name</th>
<th>Value</th>
<th>F</th>
<th>Hypothesis df</th>
<th>Error df</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilks' Lambda</td>
<td>.850</td>
<td>1.71</td>
<td>14.00</td>
<td>282.00</td>
<td>.053*</td>
</tr>
</tbody>
</table>

Note. * p < .05
Univariate F tests are presented in Table 3 and were used to examine between
groups differences on each dependent variable in isolation (BDI, STAI-State Scale,
STAI-Trait Scale, and MHLC-Internal Scale, Chance Scale, Doctors Scale, and Other
People Scale). The Univariate F test was significant for the STAI-State Scale and the
STAI-Trait Scale suggesting that the individual groups scored significantly different on
these variables. Although the BDI and the MHLC-Doctors Scale were not statistically
significant at the .05 level, they were quite close.

Post hoc tests (Tukey’s LSD comparisons) were performed to clarify exactly
which means were statistically different from each other and are presented in Table 4. In
addition to the STAI State and Trait variables, multiple comparison results for the BDI as
well as the MHLC-Doctors Scale were also displayed since they fell close to the .05 level
of significance. Statistically significant mean differences on the STAI-State Scale were
found between the moderate and severe asthma groups. On the STAI-Trait Scale,
statistically significant mean differences were found between the mild and moderate
asthma groups, as well as the moderate and severe asthma groups.

Overall, severe asthmatics were higher in their level of state anxiety than
moderate asthmatics and those with mild asthma were higher than moderate asthmatics in
their level of trait anxiety. Severe asthmatics were found to have significantly higher
levels of trait anxiety than moderate asthmatics. No statistically significant differences
were found on the BDI or the MHLC-Doctors Scale.
Table 3

Univariate Analysis of Variance: Between-Subjects Effects on the Depression, Anxiety, and Locus of Control Measures

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI</td>
<td>353.08</td>
<td>2</td>
<td>176.54</td>
<td>2.93</td>
<td>.057</td>
</tr>
<tr>
<td>STAI- State Scale</td>
<td>1285.69</td>
<td>2</td>
<td>642.85</td>
<td>4.53</td>
<td>.012*</td>
</tr>
<tr>
<td>STAI- Trait Scale</td>
<td>764.85</td>
<td>2</td>
<td>382.43</td>
<td>3.05</td>
<td>.050*</td>
</tr>
<tr>
<td>MHLC- Internal Scale</td>
<td>226.09</td>
<td>2</td>
<td>113.05</td>
<td>2.85</td>
<td>.061</td>
</tr>
<tr>
<td>MHLC- Chance Scale</td>
<td>4.17</td>
<td>2</td>
<td>2.09</td>
<td>.06</td>
<td>.942</td>
</tr>
<tr>
<td>MHLC- Doctors Scale</td>
<td>59.77</td>
<td>2</td>
<td>29.89</td>
<td>2.97</td>
<td>.055</td>
</tr>
<tr>
<td>MHLC- Other People Scale</td>
<td>31.61</td>
<td>2</td>
<td>15.81</td>
<td>1.20</td>
<td>.304</td>
</tr>
</tbody>
</table>

Note. *p < .05
Table 4

Multiple Comparisons of the Mean Differences Between Asthma Treatment Groups

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Group Classification (I)</th>
<th>Group Classification (J)</th>
<th>Mean Difference (I-J)</th>
<th>SE</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI</td>
<td>Mild Asthma</td>
<td>Moderate Asthma</td>
<td>2.42</td>
<td>1.55</td>
<td>.121</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe Asthma</td>
<td>-1.28</td>
<td>1.55</td>
<td>.411</td>
</tr>
<tr>
<td></td>
<td>Moderate Asthma</td>
<td>Mild Asthma</td>
<td>-2.42</td>
<td>1.55</td>
<td>.121</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe Asthma</td>
<td>-3.70</td>
<td>1.55</td>
<td>.018</td>
</tr>
<tr>
<td></td>
<td>Severe Asthma</td>
<td>Mild Asthma</td>
<td>1.28</td>
<td>1.55</td>
<td>.411</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moderate Asthma</td>
<td>3.70</td>
<td>1.55</td>
<td>.018</td>
</tr>
<tr>
<td>STAI-State Scale</td>
<td>Mild Asthma</td>
<td>Moderate Asthma</td>
<td>5.24</td>
<td>2.38</td>
<td>.029</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe Asthma</td>
<td>-1.62</td>
<td>2.38</td>
<td>.498</td>
</tr>
<tr>
<td></td>
<td>Moderate Asthma</td>
<td>Mild Asthma</td>
<td>-5.24</td>
<td>2.38</td>
<td>.029</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe Asthma</td>
<td>-6.86</td>
<td>2.38</td>
<td>.005*</td>
</tr>
<tr>
<td></td>
<td>Severe Asthma</td>
<td>Mild Asthma</td>
<td>1.62</td>
<td>2.38</td>
<td>.498</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moderate Asthma</td>
<td>6.86</td>
<td>2.38</td>
<td>.005</td>
</tr>
<tr>
<td>STAI-Trait Scale</td>
<td>Mild Asthma</td>
<td>Moderate Asthma</td>
<td>4.96</td>
<td>2.24</td>
<td>.028*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe Asthma</td>
<td>.36</td>
<td>2.24</td>
<td>.872</td>
</tr>
<tr>
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<td>Moderate Asthma</td>
<td>Mild asthma</td>
<td>-4.96</td>
<td>2.24</td>
<td>.028</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe Asthma</td>
<td>-4.60</td>
<td>2.24</td>
<td>.042*</td>
</tr>
<tr>
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<td>Severe Asthma</td>
<td>Mild Asthma</td>
<td>-.36</td>
<td>2.24</td>
<td>.872</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moderate Asthma</td>
<td>4.60</td>
<td>2.24</td>
<td>.042</td>
</tr>
<tr>
<td>MHLC-Doctors Scale</td>
<td>Mild Asthma</td>
<td>Moderate Asthma</td>
<td>-1.42</td>
<td>.64</td>
<td>.027</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe Asthma</td>
<td>-.18</td>
<td>.64</td>
<td>.777</td>
</tr>
<tr>
<td></td>
<td>Moderate Asthma</td>
<td>Mild Asthma</td>
<td>1.42</td>
<td>.64</td>
<td>.027</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe Asthma</td>
<td>1.24</td>
<td>.64</td>
<td>.053</td>
</tr>
<tr>
<td></td>
<td>Severe Asthma</td>
<td>Mild Asthma</td>
<td>.18</td>
<td>.64</td>
<td>.777</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moderate Asthma</td>
<td>-1.24</td>
<td>.64</td>
<td>.053</td>
</tr>
</tbody>
</table>

Note. *p < .05
Discriminant Function Analysis of the Between Group Differences 
and Classification of Three Asthma Groups

Based on the significant Multivariate tests on the group variable, a discriminant function analysis was performed to clarify which variables (BDI, STAI, MHLC) contributed most to the discrimination between groups (mild, moderate, and severe asthma). It was also used as a method of accurately classifying subjects into the dependent variable groups. The results are presented in Table 5 and showed that the first discriminant function accounted for a majority of the variance. The corresponding canonical correlation of .346 indicated that only a moderate relationship existed between the first discriminant function and the dependent variable. Wilks' Lambda results for the first two discriminant functions were significant at .053. This suggests that both discriminant functions were needed to account for the differentiation between groups, because the residual associated with the second discriminant function was not significant.

Standardized discriminant function coefficients for the first and second discriminant function are presented in Table 6. For the first discriminant function, the MHLC-Internal Scale had the greatest ability to discriminate between groups. This was followed by the MHLC-Other People Scale and the STAI-State Scale. On the second discriminant function, the BDI, the STAI-Trait Scale, the MHLC-Doctor Scale, and the MHLC-Other People Scale had large standardized discriminant function coefficients. The STAI-Trait Scale contributed the most to the between group discrimination, followed by the BDI. The MHLC-Doctors Scale and the MHLC-Other People Scale also contributed; however, the STAI-Trait Scale had a substantially larger influence on the
Table 5

Canonical Discriminant Function Analysis: Differentiation Between Three Asthma

Groups on Categories of the Dependent Variables

<table>
<thead>
<tr>
<th>Function</th>
<th>Eigenvalue</th>
<th>% of Variance</th>
<th>Cumulative %</th>
<th>Canonical Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>.136</td>
<td>79.2</td>
<td>79.2</td>
<td>.346</td>
</tr>
<tr>
<td>2</td>
<td>.036</td>
<td>20.8</td>
<td>100.0</td>
<td>.186</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>After Function</th>
<th>Wilks’ Lambda</th>
<th>Chi Square</th>
<th>df</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 through 2</td>
<td>.850</td>
<td>23.45</td>
<td>14</td>
<td>.053*</td>
</tr>
<tr>
<td>2</td>
<td>.965</td>
<td>5.06</td>
<td>6</td>
<td>.536</td>
</tr>
</tbody>
</table>

Note. *p < .05
Table 6

*Standardized Canonical Coefficients for the Beck Depression Inventory, the State-Trait Anxiety Inventory, and the Multidimensional Health Locus of Control Scale*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Function 1</th>
<th>Function 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI</td>
<td>.352</td>
<td>.821</td>
</tr>
<tr>
<td>STAI-State Scale</td>
<td>.479</td>
<td>.167</td>
</tr>
<tr>
<td>STAI-Trait Scale</td>
<td>-.153</td>
<td>-1.215</td>
</tr>
<tr>
<td>MHLC-Internal Scale</td>
<td>-.553</td>
<td>-.255</td>
</tr>
<tr>
<td>MHLC-Chance Scale</td>
<td>-.207</td>
<td>.060</td>
</tr>
<tr>
<td>MHLC-Doctors Scale</td>
<td>-.340</td>
<td>.476</td>
</tr>
<tr>
<td>MHLC-Other People Scale</td>
<td>.488</td>
<td>.540</td>
</tr>
</tbody>
</table>
group discrimination.

Correlations between each of the variables and the discriminant functions are presented in Table 7. The results indicated moderate correlations with the first discriminant function on the BDI, the STAI-State Scale, the STAI-Trait Scale, the MHLC-Internal Scale, and the MHLC-Doctors Scale. On the second discriminant function, moderate correlations were found on the STAI-Trait Scale, MHLC-Doctors Scale, and the MHLC-Other People Scale. As is evident in Table 7, the MHLC-Chance Scale had a weak relationship with both the first (0.067) and second (0.075) discriminant function and therefore, had little influence on the findings.

Results of the classification analysis are presented in Table 8. Overall, less than half of the cases were classified correctly. There was a moderate ability to classify subjects into the mild, moderate, and severe asthma groups.

*Descriptive Statistics for the 'Age of Onset' Variable on the Dependent Measures*

Descriptive statistics for the ‘age of onset’ variable on the BDI, the STAI, and the MHLC are presented in Table 9. The age of onset variable represented the age at which subjects first recalled suffering from asthma symptoms and was divided into four groups: Ages 1 to 12, ages 13 to 18, ages 19 to 24, and ages 25 and up. Table 9 revealed that on the BDI, subjects who had asthma by the age of 1 to 12 had the highest mean score, followed by the 13 to 18 age group and the 19 to 24 age group respectively. Those who first displayed asthma symptoms between the ages of 19 and 24 had the lowest mean
Table 7

*Correlations Between the Beck Depression Inventory, the State-Trait Anxiety Inventory, and the Multidimensional Health Locus of Control Scale and the Discriminant Functions*

<table>
<thead>
<tr>
<th>Structure Matrix</th>
<th>Function 1</th>
<th>Function 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>STAI-State Scale</td>
<td>.664</td>
<td>-.204</td>
</tr>
<tr>
<td>BDI</td>
<td>.541</td>
<td>-.038</td>
</tr>
<tr>
<td>MHLC-Internal Scale</td>
<td>-.534</td>
<td>-.005</td>
</tr>
<tr>
<td>STAI-Trait Scale</td>
<td>.498</td>
<td>-.468</td>
</tr>
<tr>
<td>MHLC-Doctors Scale</td>
<td>-.478</td>
<td>.510</td>
</tr>
<tr>
<td>MHLC-Other People Scale</td>
<td>.253</td>
<td>.462</td>
</tr>
<tr>
<td>MHLC-Chance Scale</td>
<td>.067</td>
<td>.075</td>
</tr>
</tbody>
</table>
Table 8

Classification of Subjects into Mild, Moderate, and Severe Asthma Groups

<table>
<thead>
<tr>
<th>Group Classification</th>
<th>Predicted Asthma Group Membership</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild</td>
<td>Moderate</td>
</tr>
<tr>
<td>Original Count</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild Asthma</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>Moderate Asthma</td>
<td>11</td>
<td>28</td>
</tr>
<tr>
<td>Severe Asthma</td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>Percentage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild Asthma</td>
<td>30</td>
<td>40</td>
</tr>
<tr>
<td>Moderate Asthma</td>
<td>22</td>
<td>56</td>
</tr>
<tr>
<td>Severe Asthma</td>
<td>30</td>
<td>26</td>
</tr>
</tbody>
</table>
Table 9

Means and Standard Deviations for the 'Age of Onset' Variable on the Depression, Anxiety, and Locus of Control Measures

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>n</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 to 12</td>
<td>54</td>
<td>9.74</td>
<td>8.48</td>
</tr>
<tr>
<td></td>
<td>13 to 18</td>
<td>32</td>
<td>8.47</td>
<td>6.65</td>
</tr>
<tr>
<td></td>
<td>19 to 24</td>
<td>18</td>
<td>6.67</td>
<td>7.67</td>
</tr>
<tr>
<td></td>
<td>25 and up</td>
<td>46</td>
<td>8.83</td>
<td>8.02</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>8.82</td>
<td>7.86</td>
</tr>
<tr>
<td>STAI-State Scale</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 to 12</td>
<td>54</td>
<td>41.63</td>
<td>12.86</td>
</tr>
<tr>
<td></td>
<td>13 to 18</td>
<td>32</td>
<td>39.69</td>
<td>11.49</td>
</tr>
<tr>
<td></td>
<td>19 to 24</td>
<td>18</td>
<td>37.83</td>
<td>11.31</td>
</tr>
<tr>
<td></td>
<td>25 and up</td>
<td>46</td>
<td>40.17</td>
<td>12.40</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>40.31</td>
<td>12.20</td>
</tr>
<tr>
<td>STAI-Trait Scale</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 to 12</td>
<td>54</td>
<td>41.15</td>
<td>11.63</td>
</tr>
<tr>
<td></td>
<td>13 to 18</td>
<td>32</td>
<td>40.28</td>
<td>11.12</td>
</tr>
<tr>
<td></td>
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<td>13.64</td>
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<td>25 and up</td>
<td>46</td>
<td>38.43</td>
<td>10.34</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>39.95</td>
<td>11.34</td>
</tr>
<tr>
<td>MHLC-Internal Scale</td>
<td>Age</td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 to 12</td>
<td>54</td>
<td>23.70</td>
<td>6.25</td>
</tr>
<tr>
<td></td>
<td>13 to 18</td>
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<td>19.25</td>
<td>6.44</td>
</tr>
<tr>
<td></td>
<td>19 to 24</td>
<td>18</td>
<td>21.28</td>
<td>6.13</td>
</tr>
<tr>
<td></td>
<td>25 and up</td>
<td>46</td>
<td>21.91</td>
<td>6.04</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>21.91</td>
<td>6.37</td>
</tr>
<tr>
<td>MHLC-Chance Scale</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 to 12</td>
<td>54</td>
<td>13.85</td>
<td>4.98</td>
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<td>13 to 18</td>
<td>32</td>
<td>13.97</td>
<td>6.06</td>
</tr>
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<tr>
<td></td>
<td>25 and up</td>
<td>46</td>
<td>15.02</td>
<td>6.62</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>14.27</td>
<td>5.86</td>
</tr>
<tr>
<td>MHLC-Doctors Scale</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 to 12</td>
<td>54</td>
<td>13.83</td>
<td>3.02</td>
</tr>
<tr>
<td></td>
<td>13 to 18</td>
<td>32</td>
<td>13.47</td>
<td>3.67</td>
</tr>
<tr>
<td></td>
<td>19 to 24</td>
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<tr>
<td></td>
<td>25 and up</td>
<td>46</td>
<td>14.17</td>
<td>3.32</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>14.09</td>
<td>3.22</td>
</tr>
<tr>
<td>MHLC-Other People Scale</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 to 12</td>
<td>54</td>
<td>6.83</td>
<td>3.16</td>
</tr>
<tr>
<td></td>
<td>13 to 18</td>
<td>32</td>
<td>7.44</td>
<td>3.56</td>
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<td>8.11</td>
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<tr>
<td></td>
<td>25 and up</td>
<td>46</td>
<td>7.15</td>
<td>4.19</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>150</td>
<td>7.21</td>
<td>3.63</td>
</tr>
</tbody>
</table>
score. The 1 to 12 age group also achieved the highest mean score on the STAI-State Scale. They were followed by the 25 and up age group, the 13 to 18 age group, and the 19 to 24 age group. On the STAI-Trait Scale, once again, subjects who developed asthma symptoms between the ages of 1 and 12 had the highest overall mean score, followed by the 13 to 18 and 19 to 24 age groups respectively. The 25 and up age group had the lowest mean score.

On the MHLC-Internal Scale, the 1 to 12 age group again demonstrated the highest score, followed by the 25 and up age group, the 19 to 24 age group, and the 13 to 18 age group respectively. On the MHLC-Chance Scale, the highest score was achieved by the 25 and up age group, followed by the 19 to 24 age group, the 13 to 18 age group, and finally, the 1 to 12 age group. The 19 to 24 age group achieved the highest mean score on the MHLC-Doctors Scale. They were followed by the 25 and up age group and the 1 to 12 age group. The lowest Doctors Scale mean score was displayed by the 13 to 18 age group. Finally, on the MHLC-Other People Scale, the 19 to 24 age group achieved the highest score, followed by the 13 to 18 age group, the 25 and up age group, and the 1 to 12 age group respectively.

**Multivariate Analysis of Variance Between Four ‘Age of Onset’ Groups**

* on the Dependent Measures

A MANOVA was used to test the difference between the age of onset groups on all dependent variables (BDI, STAI, and MHLC) simultaneously. Prior to running the multivariate analysis on the age of onset variable, a Box’s M test was performed and was
found to be nonsignificant, suggesting that the assumption of equal variance was not violated (Box’s M = 105.96, $F = 1.13$, $p > .204$). Results of the MANOVA are presented in Table 10 and yielded conflicting findings. Although the Roy’s Largest Root suggested significance, the Wilks’ Lambda clearly did not show significant results. In most cases, researchers are satisfied at this point to use either the Wilks’ Lambda, Hotelling’s trace criterion, or Pillai’s criterion, all of which are pooled statistics. The Wilks’ Lambda which represents the pooled ratio of error variance to effect variance plus error variance is the most commonly used criterion (Tabachnick & Fidell, 1989).

However, because the Roy’s Largest Root combines the dependent variables along only the first dimension, some researchers actually prefer it over the pooled statistics (Harris, 1975). Nevertheless, based on the overall outcomes in the present study, it is likely that any difference which existed on the age of onset variable was relatively small.

Table 10

<table>
<thead>
<tr>
<th>Test Name</th>
<th>Value</th>
<th>$F$</th>
<th>Hypothesis df</th>
<th>Error df</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilks’ Lambda</td>
<td>.809</td>
<td>1.47</td>
<td>21.00</td>
<td>402.56</td>
<td>.083</td>
</tr>
<tr>
<td>Roy’s Largest Root</td>
<td>.137</td>
<td>2.78</td>
<td>7.00</td>
<td>142.00</td>
<td>.010*</td>
</tr>
</tbody>
</table>

*Note. *$p < .05$
The results of the Univariate F tests for the 'age of onset' variable are presented in Table 11. The only variable with a significant difference between the age of onset groups was the MHLC-Internal Scale. Based on these results and the lack of overall significance on the Multivariate tests, a discriminant function analysis was not performed.

Results of the Tukey's LSD comparison test on the age of onset variable are presented in Table 12. Findings indicated that the only significant mean difference was between ages 1 to 12 and ages 13 to 18 ($M = 4.45, p < .002$).

**Summary of the Hypotheses**

The general hypothesis that a significant difference would exist between mild, moderate, and severe asthmatic women in their level of depression, anxiety, and locus of control was supported by the data. Included in this general hypothesis were the following specific hypotheses:

1. The hypothesis that higher levels of depressive symptoms would be associated with increased levels of asthma severity was only partially supported. Although severe asthmatics had the highest level of depressive symptoms, those with mild asthma were found to express more depressive feelings than those with moderate asthma symptoms.

2. The hypothesis that higher levels of state and trait anxiety would be associated with increased levels of asthma severity was also partially supported by the data. This finding indicated that women with severe levels of asthma had higher state anxiety when compared to those with
Table 11

*Univariate Analysis of Variance: Between-Subjects Effects on the 'Age of Onset'*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sum of Squares</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI</td>
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*Note. *p < .05*
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<td>1.73</td>
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Note. *p < .05
moderate or mild symptoms. However, women with mild asthma expressed the highest level of trait anxiety. Women with moderate anxiety had the lowest degree of trait anxiety.

3. The hypothesis that higher levels of external control would be associated with increased levels of asthma severity was partially supported. On both the MHLC-Doctors Scale and the MHLC-Other People Scale, moderate and severe asthmatics scored higher than mild asthmatic women, with severe asthmatics exhibiting the strongest belief that their health was influenced by other people.

4. The hypothesis that group membership would be predicted by depression, anxiety, and locus of control was moderately supported. The BDI, the STAI, and the MHLC were found to be only reasonably accurate predictors of membership in the mild, moderate, and severe asthma groups.

5. The hypothesis that the 'age of onset' of asthma symptoms would be significantly related to levels of depression, anxiety, and locus of control was not supported by the data. Despite the conflicting results on the multivariate tests and the presence of one significant univariate test, it was unlikely that any absolute difference existed between the age of onset groups.
Chapter V
Discussion

Overview of the Study

The purpose of this study was to investigate the relationship between depression, anxiety, and locus of control in mild, moderate, and severe asthmatic women. The initial analysis focused on the relationship between the severity of illness as a major factor in emotional responding. The secondary analysis examined the age at which subjects first experienced asthma symptoms and the effect of the duration of illness on levels of depression, anxiety, and locus of control.

In order to investigate these relationships, the Beck Depression Inventory, the State-Trait Anxiety Inventory, and the Multidimensional Health Locus of Control Scale were administered to 150 asthmatic women between the ages of 18 and 55, who were being seen for a general medical appointment. All of the subjects voluntarily participated and were subsequently classified into mild, moderate, and severe asthma groups. The hypotheses were then examined using Multivariate Analysis of Variance, Discriminant Function Analysis, and multiple comparison tests.

Summary of Findings

The results of the current investigation revealed a significant association between asthma severity and psychological functioning. The findings supported the general hypothesis of the study that a significant difference would exist between mild, moderate, and severe asthmatic women in their levels of depression, anxiety, and locus of control.
Indeed, it appeared that the severity of asthma symptoms was a contributing factor in the level of psychological impairment and dysfunction suffered by patients. These findings add to the existing research supporting an association between asthma severity and prevalence of psychopathology (Mrazek, 1992; Vila, et al., 1998). Many investigators now believe that there is a well-established connection between the degree of emotional distress, the level of asthma exacerbation, and the potential for severity of symptoms. The implication of the present research is not only that differences exist in the level of psychopathology in asthmatics, but that each group of asthmatics represents a unique category of illness. This is a critical point because previous research that reported contradictory findings when examining emotions and asthma often neglected to classify asthmatics into discrete subgroups. Part of this failure was due to the lack of a reliable system of classification. As mentioned earlier, a consistent method of classification did not exist until recently (Expert Panel Report 2, 1997). In some current studies (ten Brinke, Ouwerkerk, Bel, et al., 2001) the failure to find significant differences between groups may have been a result of using different criteria for classification. Wamboldt et al. (1998) also speculated that the lack of adequate adult studies as well as the paucity of investigations on women may be factors in the contradictory findings of existing research. Therefore, while the present study reinforces the importance of proper classification of asthmatics, further research aimed at clarifying the unique components of these groups as well as their specific emotional difficulties is imperative.

Following the general prediction of the research, a number of additional hypotheses were postulated. The first hypothesis predicted that higher levels of depressive symptoms would be associated with increased levels of asthma severity. In
fact, those women with the most severe asthma did express greater degrees of depressive feelings, thus complementing existing evidence of a well-established relationship between asthma and depression (Galil, 2000). This association of asthma severity and higher scores on depressive measures is consistent with previous research (Belloch et al., 1994; Mancuso et al., 2000) which has indicated that more severe asthmatic patients suffer from greater dysthymic feelings. However, contradictory to existing research was the current finding that mild asthmatics had greater levels of reported depression than those with moderate symptoms. One possible explanation for this is symptom perception (Lehrer et al., 2002). Much of the research on symptom perception has addressed the issue of subjective perception of symptoms (Apter et al., 1997), where major discrepancies exist between actual and perceived symptoms of illness. It is possible that those subjects classified with mild asthma in the current study perceived their symptoms to be much more severe and therefore, were prone to more depressive responding. Also, while some of the existing studies have failed to establish a relationship between asthma severity and depression (Afari, et al., 2001), it is feasible that the current lack of empirical data determining the role of negative emotions in asthma exacerbation is a significant factor (Lehrer, 1998). Certainly, the findings of the current research are consistent with the growing consensus that depression and asthma are intricately related and point to the need for greater understanding of this complex, bidirectional interrelationship.

The second hypothesis which predicted that higher levels of state and trait anxiety would be associated with increased levels of asthma severity was only partially supported. Women with severe asthma were in fact, more likely to have higher state or
transitory anxiety. Though these findings are somewhat consistent with existing research, in actuality, there has been considerable debate in the literature regarding the relationship between asthma severity and anxiety. While some researchers have postulated that more severe asthmatics suffer from greater anxiety (Mrazek, 1992; Vamos & Kolbe, 1999), others have noted that elevated anxiety levels are not positively associated with asthma severity (Afari et al., 2001). Studies in the area of panic-fear research have also reported inconsistent findings, with some researchers indicating greater degrees of generalized panic-fear in asthmatics with higher levels of asthma morbidity (Nouwen et al., 1999) and others suggesting a poor correlation between asthma severity and panic symptoms (ten Brinke, Ouwerkerk, Bel, et al., 2001).

Although evidence regarding the high comorbidity between anxiety and asthma severity remains inconclusive, it is possible that asthmatics with continual episodes of breathlessness, frequent nighttime symptoms, decreased activity, and medication dependence develop an almost "classically conditioned" vulnerability to asthma. As such, they may manifest a situational fear of the potential attack, which is reinforced by the intermittent and unpredictable nature of the disease. In addition, numerous possible environmental triggers may also serve as a perceived threat or danger for severe asthma patients, as does the very real possibility of a fatal attack.

A second interesting outcome in the current study was that women with mild asthma reported greater levels of trait anxiety than moderate or severe asthmatic women. This is a curious finding which suggests that those with mild symptoms had the most enduring anxiety traits. One possibility for this finding is the tendency for milder asthma symptoms to be confused more easily with other problems than severe asthma symptoms.
In fact, Tiller, Pain, and Biddle (1987) found that inaccurate perceptions of regular breathing patterns actually lead to greater dyspnea in asthmatics with high levels of trait anxiety. Another possibility is that the women classified as mild asthmatics may have had an underlying anxiety/panic disorder which contributed to their asthma symptoms and ultimately their diagnosis. Therefore, inconsistencies in the current and existing research may suggest that anxiety levels in adults are not necessarily related to the severity of illness but to the comorbid personality of the asthmatic. While the present results did not entirely support the proposed direction of the research, the findings were consistent with research which has determined anxiety to be a complex, significant factor in need of further examination.

The third hypothesis predicted that higher levels of external control would be associated with increased levels of asthma severity. More severe asthmatics were in fact found to rely on external locus of control factors such as other people, instead of internalizing their sense of personal control to cope more effectively with their illness. This was true of moderate asthmatic women as well, who relied more on doctors than those with mild asthma symptoms. While most of the existing studies in the area of locus of control support the contention that internal control is essential to successful health outcomes (Seeman & Seeman, 1983), few studies have addressed the issue of asthma and locus of control and most have investigated children and not adult populations. In addition, severity of illness has not been examined with respect to the impact on internal and external control factors. Since it has been shown that medication compliance (Weinberger, 1988) and successful asthma self-management programs (Miles et al., 1995) are related to levels of personal control, it is important that internal health
locus of control is recognized as an essential psychological tool in coping with and gaining mastery over asthma. Therefore, the implications of the current investigation may be broad-reaching in terms of locus of control research. In addition to addressing asthma in a population that has to date been underrepresented, the present study also examined the severity of asthma symptoms as a potential contributing factor in perceived control, self efficacy, and competence.

The fourth hypothesis that levels of depression, anxiety, and locus of control would predict group membership was moderately supported, indicating that the designated emotional factors appeared to be only reasonably accurate predictors of mild, moderate, or severe asthma. These findings suggest that while the assessment measures used in the study possessed adequate reliability and validity, there is the possibility that the instruments were not able to appropriately measure the asthmatic groups. Consistent with these findings, Yellowlees et al. (1988) found that severe asthmatics have a tendency to both deny and minimize their symptoms. ten Brinke, Ouwerkerk, Bel, et al. (2001) also speculated that attitudes of denial are harder to detect by self-report questionnaires. In addition, despite the utilization of the guidelines specified by the National Institutes of Health (Expert Panel Report 2, 1997), the classification of asthmatics into specific groups remains difficult. Past literature has suggested that because of the dramatic differences in asthma symptoms, it is impossible to accurately classify subjects. However, most researchers agree that a standard and consistent measure of asthma severity is essential for comparative research. Therefore, the implication of the present study is not only to clarify the illness spectrum of asthma, but also to establish a more consistent procedure of measurement. Further research which
takes into consideration these issues of measurement and classification will help to
expand the functional understanding of the psychological forces affecting asthma.

Finally, although the fifth hypothesis predicted that the “age of onset” would be
significantly related to levels of depression, anxiety, and locus of control, no relationship
was established. These findings are inconsistent with recent investigations which have
suggested a relationship between the early onset of asthma symptoms and significant
behavioral problems, including depressed mood (Mrazek et al., 1998). It has also been
speculated that psychosocial factors may affect asthma onset and persistence, although
the long term consequences on adult etiology of the disease are unclear (Klinnert et al.,
2001). It is possible that the current research failed to establish the actual onset of
asthma symptoms since it has been determined that the inception of symptoms,
particularly in adults, is difficult to establish due to the frequent persistence from
childhood. Clearly, present and past research indicates the difficulty in determining the
onset of the disease process. Therefore, in order to clarify these findings and establish
the connection between the age of onset and emotional factors, further research will be
needed.

Theoretical and Clinical Implications

There are a number of theoretical and clinical implications which can be
postulated from the current findings. From a theoretical perspective, the present
investigation lends support to the multifactorial theory (Groen, 1977) which is consistent
with a multideterminant model of health (Stoudemire, 1995). Such models not only
suggest an interplay of physiological and psychological mechanisms in the illness
process, but also concede that certain emotional states influence the development of disturbed patterns of ventilation. These findings also support the health belief model (Becker et al., 1978) which recognizes the constant interplay of cognition and belief in the asthmatic’s behavior toward their illness. The current investigation substantiates the role of both emotional and physical factors in asthma and reinforces the complex, bidirectional relationship between psychological influences and asthma presentation. Certainly, the level of asthma severity, coupled with the emotional impact of depression, anxiety, and locus of control factors reinforce an integrative model of disease.

In terms of the clinical impact of the research, the present findings have numerous applications. First, the finding that higher levels of emotional disturbance were noted in more severe asthmatics may facilitate the development of specific psychological therapies to target particular comorbid conditions. By understanding the elements of asthma severity, as well as the spectrum of psychological factors which might accompany the illness process, clinicians and health care providers can target cognitive strategies, individualized self-management programs, and even symptom perception training for asthmatic patients. Second, the classification of asthma patients into discrete groups may actually assist in the understanding of the unique treatments and interventions necessary for specific levels of the disease. It may be that more complex and comprehensive strategies are required for particular types of asthmatic patients. Finally, the exclusive observation of women in the study suggests a unique opportunity to explore the psychopathological aspects of asthma as they relate to gender. Because women in general are known to experience higher levels of anxiety and depression than
their male counterparts, the interconnection with asthma may present an interesting challenge in clinical practice.

Limitations of the Study

The present results are qualified by a number of potential methodological limitations. First, although the sample size was adequate, the participants were from a rather select group of adults in a specific geographical location. There was not only assumed similarity in socioeconomic status, but there was a lack of ethnic diversity as well. Thus, the results may reflect relatively homogeneous characteristics with limited generalizability. Second, the classification of subjects into asthma treatment groups was based on the 1997 guidelines suggested by the World Health Organization and National Institutes of Health (Expert Panel Report 2, 1997). Because classification was reliant upon self-report, it is possible that subjects either overperceived symptoms or underestimated their condition and were incorrectly classified. This was particularly true of the mild asthma group who may have been misdiagnosed with asthma, when in fact, their symptoms were more consistent with an anxiety disorder. In addition, although pulmonary function tests were not used in the classification process and are in fact known to be unreliable, more accurate measures of symptom intensity may be required. Third, the lack of a comparison group consisting of healthy subjects or other chronically ill individuals may have limited the interpretation of the data since all of the participants were suffering from asthma.
Recommendations for Future Research

The findings of the present study provide a number of important perspectives for the field of asthma research on both asthma severity and the psychological components of the disease process as assessed by the Beck Depression Inventory, the State-Trait Anxiety Inventory, and the Multidimensional Health Locus of Control Scale. However, while the study established some of the underlying emotional factors associated with mild, moderate, and severe asthma, the need for further research is apparent. Therefore, the following potential directions are proposed for future investigation:

1. Since the current study examined only asthmatic subjects, it would be important to evaluate the emotional components of depression, anxiety, and locus of control using both other chronically ill groups and normal comparison groups.

2. It would be interesting to compare the present outpatient population with a hospitalized asthma group, particularly in terms of levels of psychopathology.

3. The use of other assessment devices aimed at uncovering components of depression, anxiety, and locus of control require more research.

4. Classification of subjects into asthma severity groups utilizing both pulmonary function tests as well as symptom self-report may be helpful in more accurately measuring the level of symptom intensity.

5. The effects of psychopathology on the perception and interpretation of symptoms as well as the direct psychophysiological impact of psychopathology on respiratory health needs to be examined.
6. The influence of gender on levels of psychopathology and severity of asthma symptoms would be an important area to explore, particularly in light of what is already known about women and emotional illness.

7. The effects of diverse treatment modalities on the psychological disturbances in patients with different levels of asthma severity need to be examined. Along with this, the evaluation of asthma education programs and cognitive interventions require more research.

The relationship between psychological manifestations and respiratory symptoms in asthma remains complex, multifaceted, and interdependent. Consistent with previous research, the current investigation provides substantial evidence of the impact of asthma severity on levels of emotional impairment and dysfunction. While the assessment of depression, anxiety, and locus of control in mild, moderate, and severe asthmatic women was elucidated by present findings, it is essential that future research be directed toward the use of diverse comparison groups, more reliable classification of asthma symptoms, and greater awareness of the effects of psychopathology. Despite apparent improvements in treatment strategies and interventions, asthma continues to be widespread, with upward trends in hospitalizations, levels of severity, and mortality. However, with the design of well-controlled, prospective studies, future asthma research may be able to provide a greater understanding of the complex physiological and psychological dimensions of this chronic and often debilitating disease.
References


Larsen, G. L. (2001). Differences between adult and childhood asthma. Disease a Month, 47 (1), 34-44.


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Appendix A
Introductory Letter to Physicians
INTRODUCTORY LETTER TO PHYSICIANS

Dear Physician:

I am a doctoral candidate in clinical psychology at Seton Hall University, College of Education and Human Services, and I am currently collecting data for my dissertation. The purpose of my research study is to examine the relationship between emotional factors and asthma in women. I am specifically interested in depression, anxiety, and locus of control, and the presence of these variables in female patients with mild, moderate, and severe asthma symptoms.

I am requesting your assistance in allowing patients from your practice to be asked to participate in this study. I am also requesting assistance from the nurse/secretary in your office in distributing study information to the patients. A total of approximately 30 female asthma patients between the ages of 18 and 55 would be asked by the nurse/secretary if they would like to participate in a research study on asthma. If they agree to participate, they will be given a study packet which they will fill out while they are in the waiting room. Participation will last for approximately 30 minutes. During that time, patients will be requested to fill out a background information form, the Beck Depression Inventory, the State-Trait Anxiety Inventory, and the Multidimensional Health Locus of Control Scale. After the patient has completed the form and questionnaires, they will seal the packet and drop it into a “Research Drop Box” which will be available in the waiting room.

Participation in the study will be entirely voluntary and patients may discontinue their involvement at any time. There are no foreseeable risks or discomforts to the patients for participating in this study and there are no alternative procedures available. There are no short term benefits to the patients for their participation, however, the long term benefit may be the discovery of new information regarding the emotional factors related to asthma. Such findings may ultimately aid in the treatment and management of their asthma symptoms. No compensation will be provided to patients for their participation. All patient responses will be anonymous and confidentiality will be strictly maintained.

This project has been reviewed and approved by the Seton Hall University Institutional Review Board for Human Subjects Research. The IRB believes that the research procedures adequately safeguard the subject’s privacy, welfare, civil liberties, and rights. The Chairperson of the IRB may be reached through the office of Grants and Research Services. The telephone number of the office is (973) 275-2974.

Your assistance in this study would be greatly appreciated. If you choose to participate, please sign and date the letter of agreement and return it to me in the stamp addressed envelope. If you have any questions, please do not hesitate to call me at (973) 680-8782. Thank you in advance for your time and consideration.

Sincerely,

Elizabeth A. Seebode, M.A.
Appendix B
Letter of Agreement
LETTER OF AGREEMENT

I have met with Ms. Elizabeth A. Seebode, a doctoral candidate at Seton Hall University in the College of Education and Human Services. She has informed me that she is currently collecting data for her dissertation on “Depression, Anxiety, and Locus of Control in Asthmatic Women.” In addition to our discussion, I have also been given a letter describing the research procedure as well as a copy of the dissertation proposal.

I understand fully the level of involvement being requested of my patients as well as the degree of participation being requested of my office staff. At this time, I agree to allow patients from my medical practice to be asked to participate in this research study. I also have discussed the procedures with my office staff who are comfortable handing out study information.

Physician’s Signature: ___________________ Date: ___________________
Appendix C
Informational Letter to Patients
INFORMATIONAL LETTER TO PATIENTS

Dear Patient:

I am a doctoral candidate in clinical psychology at Seton Hall University, College of Education and Human Services. I am currently collecting data for my dissertation on asthma in women. The purpose of my research is to look at the relationship between asthma and the many factors which may influence the disease. It is hoped that the findings in this study will provide a greater understanding of asthma both to patients as well as their physicians.

If you decide you would like to participate, it will take about 30 minutes. During that time, you will be asked to fill out a background information form and three brief questionnaires. The first two questionnaires will ask you questions about your thoughts and feelings. The third questionnaire will ask questions regarding your asthma.

Your participation in this study is completely voluntary and you may withdraw from the study at any time. All information obtained during the study will remain confidential and anonymous and will be used strictly for research purposes. This study is separate and apart from your physician's medical practice. In addition, the research information obtained will not become part of your medical record.

Your decision to complete and return the materials provided will be recognized as an understanding of the study and a willingness to participate. In the unlikely event that your participation in the study results in any undue stress, you are strongly advised to speak to your doctor or to a family member. If you have any pertinent questions about the research and/or about your rights as a research subject, you can contact me at (973) 759-5040.

This project has been reviewed and approved by the Seton Hall University Institutional Review Board for Human Subjects Research. The IRB believes that the research procedures adequately safeguard the subject's privacy, welfare, civil liberties, and rights. The Chairperson of the IRB may be reached through the office of Grants and Research Services. The telephone number of the Office is (973) 275-2974.

If you are interested in participating, please see the nurse and she will give you a study packet which contains all of the necessary information. Thank you for your time and consideration.

Sincerely,

Elizabeth A. Seebode, M.A.
Appendix D
Instructions for Study Packet
INSTRUCTIONS

This study packet contains the following items:

i. Background Information Form
ii. Questionnaire I
iii. Questionnaire II
iv. Questionnaire III

Before beginning, please note that you are not to put your name on any of the forms or questionnaires. You may begin by filling out the Background Information Form. Once this is completed, you may fill out the questionnaires. Each of the questionnaires has a set of instructions at the top of the page. Please read the instructions carefully and begin by filling out questionnaire I. Once this is completed, fill out questionnaire II. Finally complete questionnaire III.

Once you have completed the background form and all questionnaires, please seal your study packet. The entire packet is then to be placed in the box labeled “Research Drop Box” which is in the waiting room.

Thank you in advance for your cooperation.
Appendix E
Background Information Form
BACKGROUND INFORMATION FORM

The following are a list of general background questions, along with specific questions regarding your asthma history. Please read each question carefully and circle the most appropriate answer. Try to answer every question.

1. Age________

2. Highest level of Education
   a. Grammar School
   b. Some High School
   c. High School Graduate
   d. College Graduate
   e. Graduate/Professional Degree

3. Please circle any medical conditions which apply:
   a. Arthritis
   b. Cancer
   c. Drug Addiction
   d. Alcohol problems
   e. High blood pressure
   f. AIDS
   g. Tuberculosis
   h. Kidney disease
   i. Heart Disease
   j. Migraine headaches
   k. Neurological Disease
   l. COPD
   m. Hepatitis
   n. Diabetes
   o. Seizures
   p. Other

4. Do you have a psychiatric history?
   a. Yes
   b. No

If yes, please circle any psychiatric conditions which apply:
   a. Depression
   b. Anxiety Disorder
   c. Bipolar Disorder
   d. Psychotic Disorder
   e. Personality Disorder
   f. Other
5. When did you first develop asthma symptoms?
   a. Age 1 to 12
   b. Age 13 to 18
   c. Age 19 to 24
   d. Age 25 to 55

6. When were you diagnosed with asthma?
   a. Age 1 to 12
   b. Age 13 to 18
   c. Age 19 to 24
   d. Age 25 to 55

7. Does anyone else in your family suffer from asthma?
   a. Mother
   b. Father
   c. Sister
   d. Brother
   e. Other, specify

8. Please circle if you suffer from any of the following problems:
   a. Eczema
   b. Hay Fever
   c. Urticaria
   d. Wheezy Bronchitis
   e. Drug allergies

9. Are you a smoker?
   a. Yes
   b. No
   If yes, how many packs a day? ____

10. Please circle any of the following which have triggered an asthma attack:
    a. Exercise
    b. Cold air
    c. Respiratory infection
    d. Emotions
    e. Stress
    f. Cough/Sneeze
    g. Pollen
    h. Food
    i. Allergies
    j. Animals
    k. Other

11. How often do you have asthma attacks?
    a. Once a year
    b. 2 to 3 times a year
    c. Once a season
    d. Once a month
    e. Once a week
    f. Everyday

12. Have you ever been hospitalized for asthma?
    a. Yes
    b. No
    If yes, how many times ____

13. How often do you take asthma medication?
    a. Never
    b. Occasionally/during an attack
    c. Everyday
    If on medication, specify ______

14. Which of the following best describes your activity level?
    a. no limitations due to asthma
    b. Asthma symptoms may affect activity
    c. Asthma symptoms affect activity
    d. Limited physical activity due to asthma

15. When do you usually have asthma attacks?
    a. Early morning
    b. Afternoon
    c. Evening
    d. Bedtime

16. How often do you have nighttime symptoms?
    a. Never
    b. Less than twice a month
    c. More than twice a month
    d. More than four times a month

17. Overall, how would you describe your asthma?
    a. Intermittent symptoms
    b. Frequent symptoms
    c. Daily symptoms
    d. Continual symptoms
Appendix F
Classification Form
CLASSIFICATION OF ASTHMA SEVERITY

After assessing the patient’s asthma condition based on the Background Information Form, please follow the NIH guidelines below and circle the level of severity which is appropriate for this patient. Because of the nature of the data collection, information on lung function is not available. As noted in the guidelines, the presence of one of the features of severity is considered sufficient in order to place them into a particular category. Patients should be assigned to the most severe category in which any feature occurs.

I. Mild Intermittent Asthma
   SYMPTOMS
   - Asthma symptoms < 2 times a week
   - Asymptomatic and normal PEF between exacerbations
   - Exacerbations brief (from a few hours to a few days; intensity may vary)
   NIGHTTIME SYMPTOMS
   - Nocturnal asthma < 2 times a month
   LUNG FUNCTION (not available)
   - FEV1 or PEF > 80% predicted
   - PEF variability < 20%

II. Mild Persistent Asthma
   SYMPTOMS
   - Symptoms > 2 times per week, but < 1 time a day
   - Exacerbations may affect activity
   NIGHTTIME SYMPTOMS
   - Nocturnal asthma > 2 times a month
   LUNG FUNCTION (not available)
   - FEV1 or PEF > 80% predicted
   - PEF variability 20-30%

III. Moderate Persistent Asthma
   SYMPTOMS
   - Daily symptoms
   - Daily use of inhaled short acting beta 2- agonist
   - Exacerbations affect activity
   - Exacerbations > 2 times a week; may last days
   NIGHTTIME SYMPTOMS
   - Nocturnal symptoms > 1 time a week
   LUNG FUNCTION (not available)
   - FEV1 or PEF > 60% - < 80% predicted
IV. Severe Persistent Asthma

SYMPTOMS
- Continual symptoms
- Limited physical activity
- Frequent exacerbations

NIGHTTIME SYMPTOMS
- Frequent nocturnal symptoms

LUNG FUNCTION (not available)
- FEV1 or PEF < 60% predicted
- PEF variability > 30%

| MILD INTERRMITTENT | MILD PERSISTENT | MODERATE PERSISTENT | SEVERE PERSISTENT |