ATTRIBUTIONAL STYLE AND ANXIETY SENSITIVITY AMONG ASTHMATICS

THESIS
SUBMITTED FOR THE AWARD OF THE DEGREE OF
Doctor of Philosophy
IN
PSYCHOLOGY
By
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Under the Supervision of
Dr. (Mrs.) Musaddiq Jahan

DEPARTMENT OF PSYCHOLOGY
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The relationship between physical illness and psychological factors is being increasingly understood and appreciated. Psychosomatic medicine has been an important concept for the last many years. Recent evidences have resulted in linking psychological factors to almost all physical disorders. Thus in the etiology and treatment of most diseases, even cancer and AIDS, psychological factors are being increasingly studied.

Asthma is an extremely common health problem that is caused or can be worsened by emotional and cognitive factors. Asthma is a psychophysiological respiratory disorder characterized by recurrent breathing problems and symptoms such as breathlessness, chest tightness, coughing and wheezing, together with cognitive dyscontrol (for example, dizziness and nervousness). It is not unusual for individuals with asthma to experience psychological sequela including anxiety, mood, behavioural disorders and depression. Evidences show that asthmatic population is at increased risk for affective comorbidity, specially anxiety and depression.

It appears that most of the researches focus on social (family), emotional and personality factors that in one way or the other contribute to asthma. But, emotional reactions do not occur without a stimulus. They are anchored in certain events. Therefore, it is important that we probe those factors which create an attitudinal perspective that triggers off emotions. An individual’s perceptions and views of the world in which s/he functions, determine his/her reactions, feelings, and emotions. In the present research an attempt is made to study two factors, which appear important, namely attributional style and anxiety sensitivity.

Attributional style is a multidimensional and individual differences variable that refers to the habitual ways in which people explain their positive and negative life experiences. Attributional style may also define as a tendency to make particular kind of causal inferences, rather than others, across different
situations and across time. Certain attributional style have been found to be associated with certain pathologies.

Anxiety sensitivity has been defined as the fear of anxiety and anxiety-related thoughts and bodily sensations, based on belief that they have harmful somatic, social or psychological consequences. Anxiety sensitivity is regarded as a pattern of thinking that can affect health and that some one who is more sensitive to internal bodily changes is going to be at greater risk for identifying benign internal symptoms as dangerous.

The following hypotheses were formulated, further enlarged by taking into consideration the duration of disease, age and gender, may be summarized as:

1. Asthmatics will depict an attributional style different from non-asthmatics.
2. Asthmatics with different duration of illness will differ in their attributional style.
3. Asthmatics of different age groups will differ in their attributional style.
4. Asthmatic males will depict an attributional style different from asthmatic females.
5. Asthmatics will have higher anxiety sensitivity than non-asthmatics.
6. Asthmatics with different duration of illness will differ in their level of anxiety sensitivity.
7. Asthmatics of different age groups will differ in their level of anxiety sensitivity.
8. Asthmatic males will differ from asthmatic females in their level of anxiety sensitivity.

The sample of the present investigation comprised of 150 subject, 75 asthmatic patients (38 males and 37 females) and 75 healthy counterparts, in the age range of 12 to 50 years. Asthmatic patients (who were diagnosed by
specialists) selected on the basis of purposive sampling technique, with emphasis on the fact that no bias should operate.

The Attributional style Questionnaire revised by Peterson and Seligman (1982), which comprised of 12 hypothetical events, was used for measuring attributional style. The 16-item Anxiety Sensitivity Index constructed by Reiss, Peterson, Gursky and McNally (1986) was used to measure anxiety sensitivity.

The t-test was applied to study the significance of difference between the means of various groups.

The main results of the present study are:

The asthmatics differ significantly from non-asthmatic normal counterparts in terms of attributional style and anxiety sensitivity. Global attributions for negative events emerged as a significant factor. Chronicity also emerged as an important mediating variable between attributional style and asthma, as chronicity increases the patients make more internal and unstable attributions for positive events and, global and stable attributions for negative events. Patients of different age groups also reveal significant differences in their attributional style. 20 to 35 years age group show most positive attributional style and 35 to 50 years age group show the most pathological attributional style. When gender differences were probed it emerged as a significant mediating variable in the relationship between attributional style and asthma. Women patients revealed a depressogenic attributional style.

When anxiety sensitivity is taken into consideration it is found that it is undoubtedly crucial variable that may enhance the probability of asthmatic attacks. Chronicity, age and gender mediate anxiety sensitivity and asthma relationship. Patients with shortest duration of illness report more physical concerns. As duration of illness increases patients exhibit more mental incapacitation and social concerns. Age of the patients seems to mediate only the lower order factors on anxiety sensitivity, as 12 to 20 years age group
expresses more physical concerns while the highest age group (35 to 50 years) reports more mental incapacitation and social concerns.

As regards the role of gender it is found that though male asthmatics as compared to non-asthmatic males have higher level of anxiety sensitivity but as compared to female asthmatic they are low on anxiety sensitivity with no specific concerns. Female asthmatic patients have high mental incapacitation concerns and social concerns.
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To My Parents
Certificate

This is to certify that the thesis entitled "ATTRIBUTIONAL STYLE AND ANXIETY SENSITIVITY AMONG ASTHMATICS" submitted for the degree of Doctor of Philosophy in Psychology of Aligarh Muslim University, Aligarh embodies the original research work carried out by Ms. Samreen Hasan Khan under my guidance and supervision and has not been submitted for the award of any other degree or diploma of this or any other university.

Dr. (Mrs.) Musaddiq Jahan
SUPERVISOR
Acknowledgement

All the praise to Almighty 'Allah' who bestowed my life with indefinite blessings and mercy, and showered his choicest blessing upon me in achieving this milestone. In utter gratitude, I bow my head before him with all humility and reverence.

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I would like to express my thanks to all colleagues and friends for extending their kind support and suggestions. Immense world of gratitude goes to Kamini Chauhan, Faheema Asmat and Fatima Jamal for their extraordinary cooperation and consistent inspiration.
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Samreen Hasan Khan
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Chapter – I

Introduction
ASTHMA

Definition, Symptoms and Types:

Asthma continues to present multiple challenges for affected individuals, their families, and health care professionals (Miller and Wood, 1991). Despite advances in medical treatment, the incidence, severity, and mortality of asthma have increased in recent years (Bender and Kliinnert, 1998; Weiss and Wagener, 1990). It is now the world’s most common long-term condition, according to the Global Burden of Asthma Report (Masali, Fabian, Holt and Beasley, 2004). The disease is estimated to affect as many as roughly 115 million people around the globe. It is also estimated that there may be an additional 100 million people with asthma by 2025 (Central Chronicle, 2005; Masoli et al., 2004).

Asthma is a Greek word which means ‘breathless’ or ‘to breath with open mouth’. Originally applied to shortness of breath of any cause, as in the description of the mode of death of metal miners (from the disease the Greeks call asthma) by Agricola in 1556, it has come to be applied particularly for episodic breathlessness due to bronchial disease. Like other common diseases, the definition of asthma has undergone several modifications with an increase in the knowledge of the disease. Clinicians have tended to prefer definitions based on a variety of symptoms and expiratory flow rates (Porter and Birch, 1971). According to Weiss and Wagener (1990), asthma is characterized by difficulty in breathing, shortness of breath when occurs due to increased
responsiveness of the trachea, major bronchi and peripheral bronchioles to various stimuli.

National Heart, Lung and Blood Institute (1997) define asthma as a chronic inflammatory disorder of airways resulting in recurrent episode of wheezing, breathlessness, chest tightness, and cough. Asthma is an extremely common disease of respiratory system which affects breathing by preventing air from flowing freely into the lung. In asthma the lining or walls of the airway passage of the asthmatic’s lung called bronchi and bronchioles become twitchy, thickened and swollen (due to the inflammation) in response to various stimuli (Taneja, 2004). These stimuli irritate and constrict the bronchi and bronchioles, that is, they become more narrow with excess mucus or phlegm or edema (accumulation of fluid in tissue) of the wall. Spasms of the bronchial muscles or collapse of the posterior walls of the trachea and bronchi during certain types of forced expiration block the passage of the airflow. The air becomes trapped in the airways and every breath becomes extremely laboured and wheezy (Taneja, 2004). Asthma resulting from imbalance of sympathetic and parasympathetic innervation, as suggested by Davison and Neale (1996), reflects a state of dominance of parasympathetic division of autonomic nervous system. The reactivity of the parasympathetic nervous system is responsible for contraction of bronchi. The narrowing is most marked in expiration, so the wheeze is usually in breathing out (exhalation), which the patient feels as tightness in the chest.
1. **Triggers**, including irritants (such as tobacco smoke, chemicals and cold air) or allergens (such as mold, animal dander, pollens and dust) are inhaled into the lungs.

2. When triggers aren't causing trouble, inhaling brings air through the bronchial tubes to fill elastic air sacs called **alveoli**. Oxygen passes through the thin alveolar walls into blood cells while carbon dioxide is diffused out of the blood and exhaled.

3. The triggers irritate the interior lining of the bronchioles, causing them to become inflamed and restricting the flow of air to and from the alveoli.

4. Triggers stimulate the airways to release mucus. As the mucus fills the airway, muscles surrounding the bronchial tube walls contract, further constricting and cutting off the flow of air in and out of the lungs.

Figure: The structure of respiratory system before and during an asthma attack.
Asthma can be allergic, occupational, seasonal, behavioural, early morning, nocturnal and so on (Taneja, 2004). Furthermore, severity of Asthma can have any of the four distinct levels:

(a) **Mild asthma:** Seasonal/sporadic condition - brief attacks occur a couple of times per month when triggered by events. Wheezing and breathlessness like symptoms are present only during the attacks.

(b) **Moderate asthma:** Occurs a couple of times per week, asthma symptoms like coughing and wheezing may be present at night and last for several days at a time. It may require emergency medical care.

(c) **Severe asthma:** Continuous symptoms and/or experience of frequent asthma attacks with occasional prolonged severe exacerbation (with fluctuations). Asthmatics must take preventive medication, as well as medications to treat attacks. Hospitalization and emergency care are common.

(d) **Brittle asthma:** Rare, unpredictable and most severe level of asthma that can be life threatening. Preventive and episodic medication is prescribed to help to control this condition.

Asthma is a changeable condition, so a mild case of asthma can become severe overtime, and from one asthma attack to the next. Asthma can be reversible or irreversible (Taneja, 2004). It is controllable and even curable speedily if it is caused by allergic and occupational factors. Early treatment prevents an asthma attack from becoming too severe.
**Classification of Asthma:** Asthma can be classified as follows:

a) **Extrinsic Asthma:** Starts early in life, has a familiar trend and there is a history of hay fever, eczema etc.

b) **Intrinsic Asthma:** Late onset associated with viral respiratory infections etc.

c) **Asthmatic Bronchitis:** Bronchitis associated with bronchospasm

d) **Status Asthma:** Continuous attacks not responding to bronchodilators. It is a medical emergency.

e) **Cardiac Asthma:** Patients of congestive heart failure who wheeze

f) **Exercise Induced Asthma (EIA):** After exercise, patients wheeze and experience short attack of bronchospasm.

g) **Sensitizing Chemicals:** Asthma due to Di-isocyanate, polyurethane etc.

h) **Occupational Asthma:** Asthma due to hay dust, cotton dust, silica etc.

i) **Cough Variant' Asthma:** Asthma presenting as cough symptom, diagnosed Pulmonary Function Test (P.F.T.).

Although asthma is relatively uncommon in infancy (Smyth, 1962), almost between 8 and 13% of the children (it depends on the countries) suffer from asthma. But, since symptoms usually improve with age, the condition is not too common among adults (Beeson and McDermott, 1977). It is common observation that 30 to 70 per cent children with mild asthma tend to improve about the time of adolescence. Again, the disease has increased in prevalence over a 50 years period. About 60 per cent asthma sufferers are below the age
of 17, and asthma occurs in boys twice as often as among girls, although the sex ratio evens out during the adult years. There are no well-documented explanations for this sex difference (Graham, Rutter, Yule and Pless, 1967; Purcell and Weiss, 1970). Thereafter, males probably again predominate among the elderly.

Asthma is a public health problem not only for developed countries, but its prevalence is increasing even in underdeveloped and developing countries. India accounts for one third of world's 115 million asthma patients (Central Chronicle, 2005). In India, rough estimate indicates prevalence between 10% and 15% in 5-11 years old children (World Health Organization, 2000). Due to environmental pollution, rapid industrialization and urbanization, and poor awareness, the prevalence of asthma is predicted to increase rapidly worldwide in the coming years. The increase is likely to be particularly dramatic in India, which is projected to become the world's most populous nation by 2050. An absolute 2% increase in the prevalence of asthma in India would result in an additional 20 million people to grapple with this respiratory disease (Rising asthma cases, May, 2005; Wilson, 2004).

Several studies have investigated mortality from asthma. The actual risk of an asthmatic patient dying of the disease clearly depends on the number of factors, including age; severity; availability; and quality of medical care and so on. Worldwide deaths from this condition have reached over 1,80,000 annually (World Health Organization, 2000). Anyone and everyone
can get asthma in any age including the extremes of life. Earlier the asthma begins the longer it is likely to last (Williams and McNicol, 1969).

Although many different allergic or infective stimuli may triggered an attack, asthma attacks occur intermittently and with variable severity and frequency from person to person. In an individual they may occur from hour to hour and day to day. The airways are not continuously blocked; rather the respiratory system turns to normal or near normal either spontaneously or after treatment, thus allowing asthma to be differentiated from chronic respiratory problems such as emphysema (Creer, 1982).

**The Etiology of Asthma**

The causative factors of asthma may be divided into three broad categories - allergic, infective, and psychological (Rees, 1964). Rees (1964) conducted an extensive study to determine the relative importance of these factors in the etiology of asthma. The principal results of Rees’ study demonstrated that asthma is a disease with multiple causes.

The table below shows relative importance of allergic, infective and psychological factors in the etiology of asthma.

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<th>Factors</th>
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<td></td>
<td>Dominant</td>
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<td>Allergic</td>
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This extensive study conducted by Rees showed that psychological factors emerged as a dominant cause in 37% of the cases, in 33% cases they were considered of subsidiary importance, and in 30% of the cases they were evaluated as totally unimportant—a conclusion at odds with the popular notion that asthma is always psychosomatic.

Clinical material can never constitute a definite proof of the etiology of the attack as most of the evidences are reconstructed from case histories, and many of the records observations are incidental and not reproducible. Asthma can have many causes, but it probably does not occur in the absence of biologically based predisposing factors; and the emotional factors involved in precipitating an attack tend to be quite idiosyncratic to the victim (Alexander, 1977, 1981; Knapp, 1989).

Allergy and Asthma:

In most persons, allergy is only one of many risk factors, including heredity, infection and emotional upsets, that play a major role in the etiology of their disease. The strongest risk factors for developing asthma are exposure especially in infancy, to indoor allergens (such as domestic mites in bedding, carpets and stuffed furniture, cots etc.) (World Health Organization, 2000). Many individuals report the symptoms of asthma on exposure to house dust, mites, low standards of domestic hygiene, fungal spores, molds, furs or pollens. Allergens may be encountered in the general, the domestic and the occupational environment. Timings of the symptoms may give an important
clue. For example, seasonal variation suggests allergy to the pollen or spores in the air (Seaton, Seaton and Leitch, 1995).

The cells of the respiratory tract may be especially sensitive to one or more substances or allergens bringing on asthma. The physical reactions are the result of allergen-antibody or antigen-antibody reactions of the tissue. That is, certain pollens are made up in part of chemical substances which have the property of penetrating the mucous layers of the respiratory tract and of diffusing into the tissue where they lead to the physical symptoms of asthma (Landis and Bolles, 1961).

**Air pollution/irritants and Asthma:**

Doctors cautioned that air pollution can play a part in the development and triggering of asthma; and can also worsen the symptoms (Masoli et al., 2004). Asthmatic patient may notice exacerbation in relation to episodes of air pollution. Photochemical smog in high traffic density areas, indoor air pollution from burning biomass, fuel for cooking, gas cookers, heating in poorly ventilated dwellings and side stream tobacco smoke also contributes to the burden of asthma (Seaton, Seaton and Leitch, 1995; Wilson, 2004). Exposure to chemical irritants in the work place are additional risk factors (World Health Organization, 2000).

**Respiratory infections and Asthma:**

Another factor that seems to be an important cause of asthma is the person’s history of respiratory infection. Respiratory infections, most often
acute bronchitis, can also make the respiratory system vulnerable to asthma (Davison and Neale, 1996). Studies have found that the individuals who contracted serious viral infections in infancy or early childhood are more likely to develop asthma than individuals who did not (Li and O'Connell, 1987). There is some evidence that viral infections in childhood may predispose the child to the development of asthma and bronchial hyperreactivity in later life (Burrows, Krudsen and Lebowitz, 1970). Infections damage the respiratory system, making it highly vulnerable and sensitive to certain triggering conditions; conflict, frustration and other emotional upsets can than interact with these damages to produce asthma (Lachman, 1972).

**Psychological factors producing Asthma**

There has been a paradigm shift in medicine and the ancient distinction between the psyche (the mind) and the body (physique) is no longer acceptable. It is suggested that any physical disease can have psychological roots. We see the human organism as integrated unit in which mind and body work together in a single living system. Disease, therefore, can have both physical and psychological causes, and the psychological component can be of greater or lesser importance (Hass, 1979). Most of the diseases are due to the interaction of physical and emotional (psychological) variables, that is, emotional factors probably play significant role in the onset or exacerbation of many disease.
At one time, asthma was considered exclusively an emotional disorder and referred to as 'asthma nervosa'. But, a small proportion of asthmatic attacks (and realistic estimates have varied from 5 to 20%) seem to be set off by emotional factor (Hass, 1979). Psychological factors (for example, stress, life event, emotion etc.) frequently stimulate autonomic nervous system activity that constricts the bronchiole and stimulates mucous secretion. Therefore, even when asthma is originally induced by an infection or allergy, psychological stress can precipitate attacks. Dividing asthmatic patients into categories based on whether asthma is due to specific allergens or psychological factors is not easy. Many asthmatic patients whose conditions seems to have clear allergic or infective roots also report attacks caused by strong emotions and other psychological events (Hass, 1979). In addition, research suggests that asthmatics are at increasing risk for affective comorbidity specifically anxiety, depression, and panic attacks. The role of emotional factors in asthma has been highlighted by many researchers (Knapp, 1989). Clinical and experimental studies carried out by Abramson (1951) and, Freeman, Feingold, Schlesinger and Gorman (1964) are extensive, and several psychological factors have been isolated as aggravating and etiological agents.

Most patients with asthma report that exacerbations are provided by psychological events, such as shock, bereavement, extreme emotional experience and excitement. Rarely, however, are such factors the dominant
cause of the disease (De Araujo, Van Arsdel, Holmes and Dudley, 1973; Rees, 1956; Seaton, Seaton and Leitch, 1995).

An extensive amount of research has been published on asthma and its psychological correlates. Asthma has been seen as the reaction to stress (Bengtsson, 1984; Gottschalk, 1975). Earlier Selye (1976) had expressed the notion that negative stress (distress) increases the vulnerability of an individual’s psychological and physical health and thus do more damage. Asthmatic patients are highly affected by psychological stressors (Carr, Lehrer, Hochron and Jackson, 1996) and respond to stress with greater increase in respiratory resistance, skin conductance, blood pressure, arousal, depression, and shortness of breath (Rietveld, Beest and Everaerd, 1999; Ritz, Steptoe, Dewilde and Costa, 2000). Even in young healthy college students stress can ‘promote at least some hallmark of inflammation associated with asthma’ (Liu and others, 2002).

Many studies have focused the role of pathogenic family patterns and parent-child interaction in the development of asthma. Parents of asthmatic children are found to have an ambivalent attitude towards children (Lipton, Steinschneider and Richmond, 1966; Olds, 1970). Children coming from the emotionally disturbed families are more susceptible to asthmatic attacks (Purcell et al., 1969). In many children overprotection by the mother plays a reinforcing role in the maintenance of these symptoms (Eiser, Eiser, Town and Tripp, 1991).
Attempts to find the role of personality factors in asthma have led to contradictory and inconclusive findings. A large body of research studies suggest that asthmatics have a common personality profile having unresolved dependency on the mother, unconscious fear of the loss of the mother (Alexander, French and Pollock, 1968; Herbert, 1965), and neurotic tendencies, like meekness, sensitivity, anxiety, meticulousness, perfectionism and obsessions (Rees, 1964).

In addition, researches suggest that asthmatics are at increased risk for affective comorbidity specifically anxiety, depression and panic attacks (Bennett, 1994; Goldney et al., 2003; Kashani, Konig, Shepperd, Wilfley and Morris, 1988; MacLean, Perrin, Gortmaker and Pierre, 1992; Opolski and Wilson, 2005). Asthmatics are generally more anxious than the normals. Whatever is the cause of asthma, anxiety is the chief characteristic of asthmatics (Lolas and VonRod, 1977; Mathe and Knapp, 1971; Pawar, 2003).

ATTRIBUTIONAL STYLE

The kind of explanations that people offer for events has been receiving considerable attention from psychologists. In an attempt to understand and explain the causative factors of human behavior, psychologists have found these explanations to be potentially significant. The causal explanations for events, termed attributions, help them explain many crucial areas of behavior like achievement, health, dysfunctional behaviors
and also incorporation of the principles of attribution facilitates the treatment process.

Attribution is a complex process through which we seek to understand the causes behind other’s behavior and occasionally the causes behind our behavior too (Kelley, 1972).

According to Weary, Stenley and Harvey (1989), an attribution is an inference about why an event occurred or about a person’s disposition or other psychological states. We make attributions about our own dispositions and experiences just as we make attributions about others. Hence, attributions may be perceptions and inferences about others or about self.

The attributions are on the one hand a person’s explanation of causation, on the other hand they gradually constitute his/her perspective and framework through which s/he views life. There are significant evidences which point towards the fact that causal explanation which the individual considers relevant with regard to various events experienced by him/her, has a marked effect on his/her action and behavior. Causal attribution processes are not only means of providing the individual with perceptions of reality about the world, but also of maintaining effective control in the world (Kelley, 1972; Stryker and Gottlieb, 1991).

Attribution theory is about how people make causal explanations, about how they answer questions beginning with “why?”. The theory deals with the information they use in making causal inferences, and with what they
do with these information to answer causal questions. The theory developed with in social psychology as a means of dealing with questions of social perception and also self-perception.

A number of influential theories, each of which has some similarities to and differences from the other, have been proposed to explain causal inferences developed by people. Heider (1958) first wrote about attribution theory in his book ‘The Psychology of Interpersonal Relationships’, which played a central role in the origination and definition of attribution theory. Heider’s theory was concerned with how we attempt to understand the meaning of other people’s behaviour—particularly how we identify the causes of their actions. He was first to create the dichotomy of situational (external) vs. dispositional (internal) factors. According to Heider’s analysis of social perception and phenomenal causality, attributional processes are inextricably intertwined with perceptual processes and are oriented towards the search for structure of dispositional properties (Weary, Stenley and Harvey, 1989). Heider (1958) also made the distinction between personal and impersonal causality. Personal causality is identified with intentionality and impersonal causality by multifinality. He asserted that people were naive/lay scientists who use rational processes to explain behaviour, that is, people act on the basis of their beliefs.

Jones and Davis (1965) employed attributional principles adopted from Heider, and developed the theory of correspondent Inferences. Correspondent inference means describing the individual’s intentions and
dispositions in terms of his/her behaviours. Jones and Davis focused primarily on how personal attributions are made. They described how an ‘alert perceiver’ might infer another’s intentions and personal dispositions (personality traits, attitudes etc) directly from or corresponds to his/her behaviour. Inferences are correspondent when the behaviour and the disposition can be assigned similar labels. We are likely to make dispositional rather than situational attribution about an individual (actor) when the behaviour is intentional (deliberate or voluntary), uncommon, and low in social desirability.

Another important theory, Kelley’s (1967, 1973) theory of External Attribution also grew out of Heider’s original work, and is not limited to interpersonal perception. Kelley defines attribution as the process of perceiving the dispositional properties of entities in the environment. His theory concerns the subjective experiences of attitudinal validity. He asks the question: “How do individuals establish the validity of their own or of other person’s impression of an object?” That is, his theory not only explains our perception of others, but also perception of our own behaviour.

Kelley suggested that perceivers examine three different kinds of informations in their efforts to establish validity (Ross and Fletcher, 1985) – (a) Consensus information - How other people react to the same stimulus, (b) Distinctiveness information – does the person react the same way or differently to different stimulus, and (c) Consistency - Is the person’s behaviour consistent over time.
Kelley maintains that when making an attribution of causality to personal/internal or environmental/external forces, a person draws on information concerning consensus, distinctiveness and consistency. If there is low consensus, low distinctiveness, and high consistency, a personal/internal attribution will be made. A combination of high consensus, high distinctiveness and high consistency determines a stimulus/external attribution. If consensus is low but distinctiveness and consistency are high, behaviour may be attributed to combination of internal and external factors.

These attributional theories have extended our understanding of how perception affects behaviour. According to these theories, we don’t observe traits, in fact, we observe behaviour and then attribute causes to it, that is, we attempt to explain why people behave as they do. Attribution theories stress the rational information-processing aspects of forming attributions about how people answer, questions beginning with ‘why’. They presume that all humans are rational, utilize the available information to draw certain causal inferences to seek the truth but if perceiver doesn’t process the information in an unbiased manner, the use of theories is restricted.

The term ‘attributional style’ emerged out of the theory of learned helplessness (Maier and Seligman, 1976, Seligman, 1972). Research interest in the concept of attributional style arose with the publication of Abramson, Seligman and Teasdale’s (1978) attributional reformulation of helplessness model.
While the original helplessness theory hypothesized that experience with uncontrollable events led to difficulties in motivation, cognition and emotion, the reformulated model postulated that people develop a characteristic causal explanation for events. The characteristic causal explanation termed as 'attributional style' by Abramsan et al. (1978) and as 'explanatory style' by Peterson and Seligman (1984), mediates the process by which uncontrollable events produce behavioral deficits (Peterson, Maier and Seligman, 1993). Peterson and Seligman (1984) defined explanatory style as a cognitive personality variable that reflects the tendency to explain bad events involving the self with causes that are internal in the self ("it's me"), stable across time ("it's going to last forever"), and global in effect ("it's going to undercut everything").

Attributional style is a multidimensional and individual differences variable that refers to the habitual ways in which people explain their positive and negative life experiences (Abramson, Seligman and Teasdale, 1978). Attributional style may be defined as: a tendency to make particular kind of causal inferences, rather than others, across different situations and across time (Metalsky and Abramson, 1981).

Ickes and Layden (1976) describe attributional style as consistent way of ascribing the causes of positive and negative events. It is the extent to attribute negative outcome to stable, internal causes such as their own traits versus specific, external causes. It may determine both individual’s susceptibility to learned helplessness and the extent to which they can be
protected against the occurrence by exposure to situation in which they can control negative events. Ramirez, Maldonado and Mortos (1992) have provided evidence for the conclusion.

In the original formulation of attribution theory, Heider (1958) had distinguished between perceived internal and external causes for events. Subsequently, Weiner (1972) drew a distinction between stable versus unstable causes, with stable attributions for failure being seen to contribute towards poor or low levels of motivation. The third dimension of helplessness was introduced by Kelley (1972) who focused on ascription of global versus specific causes for adverse events. Attributions to global causes are more likely to generalize across a variety of situations, thus engendering helplessness in the face of failure, whereas specific causality is likely to be restricted to particular situations and outcomes. These three causal explanatory dimensions (that is, permanence relating to stable versus unstable causes, personal relating to internal versus external causal statements, and pervasive relating to universal/global versus specific causal explanations) were incorporated into the reformulated attribution theory as accounting for habits of explanation rather than for single explanation of single failure as Weiner's attribution theory had done. These explanatory habits were seen to comprise a characteristic style of explanations which individuals impose on their world, allowing them to explain causes of events, at the same time as giving them a predisposition to view everyday interactions and events from a
predominating positive (that is, optimistic) or negative (that is, pessimistic) framework.

Attributional styles may be classified along three orthogonal basic attributional dimensions (Abramson, Garber and Seligman, 1980; Abramson, Seligman and Teasdale, 1978).

1. Internal-External Dimension: This internal/external distinction is reminiscent of Heider’s assumption that the outcome of an action depends on a combination of effective personal forces (ability factor and motivational factor) and an effective environmental force. Many psychologists have tried to categorise the causes in terms of internal and external. People search for a cause structure of events via reliance upon attributions to the environment (external attributions) or to something in the person involved in the event (internal attributions) (Heider, 1958).

When person tend to believe that the conducive cause is localized directly in themselves, that is due to their own doing (for example, ability, attitude, effort, emotional state, skill etc.) attributional style is said to be internal. On the other hand, attributional style is found to be external when person regard the environmental stimulus or factors (physical and social circumstances) (for example, chance, luck, task - difficulty etc.) as a cause of an event.

2. Stable-Unstable Dimension: Weiner, Frieze, Kulka, Reed and Rosenbaum (1971) added stability-unstability dimension of causal attribution; that was
also included in Heider’s analysis. This dimension refers to the persistence of a cause, whether the cause of the event is chronic (stable) or temporary (unstable).

Stability refers to the relative performance associated with an attribution. An assumption that cause does not change over a long period in similar situations is termed as stability. Contradictory to it, when person assumes that cause may change over a short time is termed as unstability. In other words, stable factors are thought to be long lived and recurrent, whereas unstable factors are short lived and intermittent. For example, ability, aptitude, task characteristics, interest etc. are stable causes, and chance, effort, mood, luck etc. are unstable causes.

3. Global-Specific Dimension: In addition to internality and stability, Abramson et al. (1978) and Miller and Norman (1979) added the globality dimension of attribution (Alloy et al., 1988). This dimension measures the extent to which a cause affects an individual’s whole life (global) or just a few areas (specific). Weiner (1986) considered that globality refers to consistency over situation. That is, in globality person generalizes the experience to a large variety of events or situations. Therefore global factors affect expectancy and performance in many situations. Whereas specific factors are unique to a particular context.

While global causes are relevant for a wide variety of outcomes, specific causes affect only a specific set of outcomes (that may result in helplessness) only in original situation.
Each dimension is thought to have specific consequences. Internal attributions for bad events influence an individual’s self-esteem in their wake, stable attributions result in motivation, and global attributions result in pervasive deficits. Somewhat different description of the dimensions of causal explanations have been proposed by Anderson (1983), Weiner (1986), and Stratton, Munton, Hanks, Heard, and Davidson (1989), among others, but all agree that causal attributions influence a wide variety of psychological outcomes.

The three attributional dimensions tend to be correlated, resulting in eight possible combinations regarding the attributional styles.

1. Internal-global-stable
2. Internal-global-unstable
3. Internal-specific-stable
4. Internal-specific-unstable
5. External-global-stable
6. External-global-unstable
7. External-specific-stable
8. External-specific-unstable

Later work has demonstrated that these combinations of causal dimensions are useful for the understanding of various sorts of attributional styles including depressogenic attributional style (Peterson and Seligman, 1984) and various causes of future expectations and actions of an individual.
Research suggests that people show consistency in the sort of causal explanations they typically offer, and thus we can speak of an attributional style (or explanatory style) with trait like properties (Peterson, 1991). Attributional style is not the only influence on actual causal explanations, of course, because people’s causal explanations are also shaped by the information that events afford as well as the degree of cognitive processing that they undertake (Gilbert, Pelham and Krull, 1988). But all other things being equal, attributional style predicts depression, achievement, and physical well-being (Buchanan and Seligman, 1995).

Few researches are available on gender differences in attributional style. However, Nolen-Hocksema et al. (1991), Yates and Afrassa (1994) and Yates et al. (1995) reported boys as evidencing a more negative pattern than girls. In the Yates et al. study the differences between the sexes achieved significance in both positive and negative subscales, while Nolen-Hoeksema et al. (1991) found significant results for boys predominantly on the negative subscale, with a significant difference being found only on those positive items that related to family interactions.

Attributional style is a significant variable linked to various domains of physiological as well as psychological health related functioning (Dua, 1994, 1995; Khan and Jahan, 2006; Michela and Wood, 1986). Over recent years, attribution theories have been applied to the study of health and health related behaviours. An emerging literature shows that explanatory style foreshadows poor health measured in a variety of ways: symptom report,
doctor visits, physician examination, immunosuppression, survival time with cancer, recurrence of heart disease, and untimely death (for example, Buchanan, 1995; Kamen-Siegel, Rodin, Seligman and Dwyer, 1991; Levy, Lee, Bagley and Lippman, 1988; Lin and Peterson, 1990; Peterson, 1988; Peterson, Seligman and Vaillant, 1988; Peterson, Seligman, Yurko, Martin, and Friedman, 1998). It was noted that those people who had a more pessimistic explanatory style got sick more often and had an impaired immune response (Kamen-Siegel et al, 1991). There was also preliminary evidence that showed that optimists were better able to fight serious diseases (Scheier and Carver, 1985), such as cancer. Pessimistic explanatory style predicted poor health after age 45 through 60 (Peterson, Seligman and Vaillant, 1988).

In the studies of great clinical interest, the attributional style of patients with bulimia (Goebel et al., 1989) or eating disorder (Tamara, Waller and Rachel, 2006); rheumatoid arthritis (Chaney et al., 2004; Hommel et al., 2001); diabetes, acute lymphocytic leukemia or sickle-cell syndrome (Schoenherr et al. 1992) etc. was examined, specifically in relation to depression, depressive symptoms or self rated/perceived disability. The relationship between health-illness indices and explanatory/attributional style in children and adolescents has also been explored, specifically among cancer patients and children with insulin-dependent diabetes mellitus.

Many researchers have also examined the mediating role of attribution in samples of cancer patients (for example, Taylor, Lichtman and
Wood, 1984; Timko and Janoff-Bulman, 1985); arthritis patients; female bulimic patients (Metalsky et al., 1997), as well as mothers of children with diabetes (Affleck et al., 1985) and infants at high risk for developmental disability (Affleck, Tennen and Gershman, 1985; Tennen et al., 1986).

Attributions may have a significant impact on smoking behavior. A small number of studies have examined the relationship between attribution and smoker’s ability to stop smoking or remain abstinent following smoking cessation treatment (Harackiewicz et al., 1987).

An additional area of interest to attribution-health research concerns the relationship between the Type A behavior patterns and coronary disease. In general, the studies conducted by Brunson and Mathews, 1981; Rhodewalt, 1984; Strube, 1985; Strube and Boland, 1986, suggest that attributional activity may have an important role in the relationship between Type A behavior and health-related functioning. However, the nature and meaning of this relationship is still somewhat unclear. Specifically, the nature of attributional activity in Type As seem inconsistent, and the degree to which desire for control (or some other motivation such as self appraisal) activates the type A pattern and influence attributions is unknown.

Individual differences in attributional style or desire to control among Type As may moderate the relationship between Type A behavior and coronary illness or may have an impact on compliance with medical regimens. As Strube (1987) noted, not all Type A individuals are coronary-
prone, and it may be that only Type As with certain attributional patterns (for example, a self-serving bias, a control bias) are more likely to experience coronary problems.

Patterns of attribution have also been related to depression. Negative attributional style has been suggested as a psychological correlate of depression. Attributing negative events to internal, stable and global causes plays a causal role in predisposing people to depression (Abramson et al., 1978). When people explain bad events in terms of “character flaws” (internal, stable, and global causations) they put themselves at risk, “for apathy, depression, failure, illness and even death. Those who blame themselves for bad events and feel powerless to change them find themselves in a particular stressful situation” (Peterson and Bossio, 1991).

Several studies as well as several comprehensive reviews of the literature (Alloy, Lipman and Abramson, 1992; Dixon and Ahrens, 1992; Greenberg, Pyszczynski, Burling and Tibbs, 1992; Hanger and Lund, 2002; Hull and Medolia, 1991; Joiner, Wagener and Diness, 1995; Sweeney, Anderson and Bailey, 1986) reveal much evidence for claiming an association between attributional style for negative events and depression in both clinical and non-clinical samples. Studies conducted on different diagnostic groups of schizophrenic like paranoid and non paranoid, and clinically depressed patients show significant differences in terms of attributional styles indicating internal, stable and global attribution for negative events as the characteristic of depressed patients as compared to schizophrenic non-depressed patients
(Bhojak et al., 1989; Raps et al., 1982; Silverman and Peterson, 1993). However, negative attributional style is associated with hopelessness depression symptoms rather than endogenous depression symptoms (Joiner, 2001). Causal explanations are front-and-center in many cognitive approaches and psychotherapy. For example, attributional retraining, or cognitive therapy which trains individuals to have more realistic attributions, has been shown to be related to cognitive outcomes such as increased expectations of success as well as behavioral outcomes such as enhanced task performance. Furthermore, causal attributions appear to be risk factors, not only for depression but also for a variety of difficulties such as anxiety, substance abuse, and eating disorders (Peterson, Maier and Seligman, 1993). Indeed, attributional retraining as a cognitive therapy has been shown to yield positive outcomes for these disorders as well (Fürsterling, 1985).

ANXIETY SENSITIVITY

In 1985 Steven Reiss and Richard McNally putforth the concept of ‘anxiety sensitivity’. Now it has become an established concept. Over the last two decades, the variable, anxiety sensitivity has attracted a great deal of attention from researchers, clinicians and professionals with more than 450 peer reviewed journal articles published. In addition, anxiety sensitivity has been the subject of numerous papers and posters at professional conventions; symposia; seminars etc.
Anxiety sensitivity has been defined as the fear of anxiety and anxiety-related sensations (Reiss and McNally, 1985), which arise from beliefs that these sensations have harmful somatic, social or psychological consequences.

However, anxiety sensitivity (an exaggerated response to anxious feeling) is different from the concept of anxiety (frequency of symptom occurrence). When we evaluate anxiety conditions, we need to consider not just the amount of anxiety shown by the person, but also the person’s sensibility to anxiety.

Fenichel (1945) observed that some people with anxiety disorders “develop a ‘fear of anxiety’ and simultaneously a readiness to become frightened very easily…” Evans (1972) reported the case history of a woman who feared recurrent panic attack whenever she had to eat in the presence of others.

Previously, the concept of the fear of fear was proposed by Goldstein and Chambless (1978) and by Reiss and McNally (1985) of Ohio University. Reiss and McNally (1985) have analyzed the fear of fear into two component processes called anxiety expectancy and anxiety sensitivity. Anxiety expectancy is primarily an associative learning process in which the individual has learned that a given stimulus arouses anxiety or fear. On the other hand, anxiety sensitivity is an individual difference variable consisting of beliefs that the experience of anxiety or fear causes illness, embarrassment
or additional anxiety. For example, the person may believe that a pounding heart is a sign of an impending heart attack or that it can be terribly embarrassing to have a growing stomach. Anxiety sensitivity should increased alertness to stimuli signaling the possibility of becoming anxious, increased worry about the possibility of becoming anxious, and increase motivation to avoid anxiety-provoking stimuli.

The Reiss and McNally position build upon the prior Goldstein and Chambless position but also departs from it. One difference concerns the role of panic experience in the fear of fear. Goldstein and Chambless regard the fear of fear as the consequence of panic experiences, whereas Reiss and McNally regard the fear of fear as the consequence of several factors, which include panic experiences, but also other factors like biological constitution and personality needs to avoid embarrassment, to avoid illness or to maintain control. A history of panic attacks may strengthen anxiety sensitivity by providing examples of frightening anxiety experiences. However, a history of panic experiences is not necessary for the acquisition of negative belief about the effects of anxiety.

Reiss and McNally (1985) first proposed the concept of anxiety sensitivity. A number of evidence suggests that the fear of anxiety is a secondary consequence of panic attacks. For example, many researchers accepted the hypothesis of introspective conditioning, which holds that a fear of anxiety develops when people who have initial panic attacks learn to fear the recurrence of those attacks (Goldstein and Chambles, 1978). In contrast,
Reiss and McNally (1985) proposed that the fear of anxiety (anxiety sensitivity) might constitute a cognitive risk factor for the development of panic disorder. It was also supported by Lau, Calamari, and Waraczynski (1996).

Although there are overlapping and similarities in the concept of anxiety sensitivity and panic disorder, and anxiety sensitivity and anxiety disorder, if we look at the picture critically we find that anxiety sensitivity has certain distinctive features, which set it apart from panic disorder and anxiety disorder. It may be a predisposition for both, it may come into existence as a part of the experiential impact of anxiety and panic but the cognitive component, which is so vitally related to anxiety sensitivity, sets it apart.

It is possible that the anxiety sensitivity is causally related to the development of anxiety disorders. Anxiety sensitivity should increase the negative valence (aversiveness) of anxiety experiences. For example, anxiety should be more likely to grow in magnitude for an individual who believes that anxiety causes heart attack than for someone who does not share this belief. Beck and Emery (1979) observed that, “as anxiety attacks recur, the victim becomes to dread the unpleasant symptoms of anxiety almost as much as the precipitating causes…”

Reiss and McNally (1985) outlined an expectancy model of fear based on a new concept of the fear of fear, called anxiety sensitivity. Because anxiety sensitivity was defined as a personality factor that enhances the
person's conditionability for fear, the concept has similarities to Eysenck's concept of neuroticism (Rachman, 1990). Because anxiety sensitivity was defined in terms of irrational beliefs, the concept has similarities to Ellis's (1979) concept of discomfort anxiety and to Clark's (1986) theory of panic. There also is some similarity between the concept of anxiety sensitivity and Rescorla and Wagner's (1972) concept of the "reinforcing effectiveness" of an Unconditioned Stimulus (UCS). Specifically anxiety sensitivity is seen as enhancing the reinforcing effectiveness of the sensations of anxiety.

The expectancy theory, developed in 1985 by psychologists Reiss, and McNally in collaboration with George Washington University psychologist Peterson argues that the person does not need to have a panic attack to develop a fear of anxiety symptoms.

Reiss expectancy theory holds that human motivation to avoid a feared object is a function of two classes of variables, called expectation and sensitivity. Expectation refers to what the person thinks will happen when the feared object/situation is encountered (example, "I expect the plane will crash", "I expect to have a panic attack during flight", "I expect other people will notice my fear of flying"). Sensitivity refers to the reason that a person holds for fearing the anticipated events (example, "I can't stand the thought of being handicapped", "panic attacks cause heart attacks"). Expectations (what one thinks will happen) and sensitivities (why one is afraid of the anticipated event) theoretically provide the key for understanding human fears.
Reiss expectancy model holds that there are three fundamental fears (called sensitivities): the fear of injury, the fear of anxiety, and the fear of negative evaluation. Thus this model has focused on the fear of anxiety (anxiety sensitivity). The model recognizes a wide range of individual differences in explanations regarding a particular object or situation (Gursky and Reiss, 1987; Rachman and Lopatka, 1986). For example, some people boarding an airplane will think that there is a chance that the plane will crash, whereas others think there is virtually no chance of a crash. Some people think there is a substantial likelihood that an airplane flight will cause them to have a panic attack, experience an upset stomach, or vomit; others dismiss the probability of such events as negligible.

The model also recognizes a wide range of individual differences in people’s sensitivities to fear-outcome events. Some people are terrified by fear-outcome events, whereas others do not care. Some people who expect to become anxious and stressed while flying in airplanes dismiss the bodily sensations of anxiety as harmless; other people think that anxiety experiences cause heart attacks and/or mental illness. Some people who anticipate the possibility of a plane crash dismiss the likely consequences of death or injury by telling themselves that God’s will is not to be feared.

Anxiety sensitivity is a pattern of thinking that can affect health, said Norman Schmidt (1998) associated Professor of Psychology at Ohio State University. “Just having this type of thinking pattern puts a person at greater risk for developing physical or mental impairment”. Schmidt conducted the
study with Darwin Lerew (1998). In addition to anxiety-sensitivity, the researchers evaluated two other psychological risk factors—body vigilance and discomfort intolerance—that could lead to psychological or physical impairment (Science daily, 1999).

Body vigilance, that is the attention people give to bodily changes/sensations, provides a greater risk for identifying a benign internal symptom as dangerous. Schmidt (1998) said, “And someone who doesn’t tolerate unpleasant bodily sensations very well could be at risk for developing an anxiety disorder”. Schmidt said the fact that anxiety affected women more than men may have something to do with how males and females interpret stress. “Women are at greater risk for anxiety disorders than men and there is some evidence to suggest that gender differences in this particular type of thinking pattern (anxiety sensitivity) may be part of the reason why”, he said (Science daily, 1999).

Anxiety sensitivity is defined as a fear of anxiety-related thoughts and bodily sensations based on belief that they will be harmful. It has been characterized as a heightened anxious response to the perception of physiological sensations caused by a hypervigilant self-monitoring and attention focused on internal physical cues. According to this theory, individuals with a higher level of anxiety sensitivity show a greater proneness in assessing anxiety-related symptoms as threatening, alarming and dangerous. High anxiety sensitivity has also been discussed as a predisposing factor in the development and maintenance of anxiety disorders and it has
shown a strong relationship especially to panic disorder (Schmidt, Lerew and
to panic disorder (Schmidt, Lerew and Jackson, 1997; Taylor, Koch and McNally, 1992). Since individuals with
higher anxiety sensitivity seem to be more vigilant to subtle changes in
physiological sensations, an induction of intense bodily sensations should
cause more anxious responding in people who are higher in this trait.
Biological challenge producers such as inhalation of carbon-dioxide enriched
air as a panicogen trigger are widely used methods in physiological research
to investigate physiological and psychological responses in individuals with
raised levels of anxiety sensitivity (Zvolensky, Eifert, Lejuez and McNeil,
1999), as well as underlying pathogenic mechanism between different anxiety
disorders (Papp, Klein and Gorman, 1993).

Anxiety is a part of our lives. It is a normal and protective response
to events outside the range of everyday human experience. It helps us to
concentrate and focus on tasks. It helps us to avoid dangerous situations.
Anxiety also provides motivation to accomplish things that we may otherwise
tend to put off.

Since anxiety and anxiety disorder is a very common term, and
anxiety sensitivity is a new term so it would be apt to discuss the difference
between anxiety and anxiety sensitivity. The results and review of several
studies demonstrated that anxiety sensation is distinct from other measurable
aspects of anxiety.
Anxiety is a feeling of tension, fear or dread that occurs in response to a real or imagined threat. Anxiety sensitivity refers to individual differences in what people think will happen to them when they actually experience anxiety. Anxiety can be viewed as a momentary emotional response to life situations. Anxiety sensitivity is a fear of anxiety sensations, which arises from belief that these situations have harmful somatic, social, psychological consequences. The degree of anxiety depends on how serious or severe the person thinks a real or imaginary threat is. Anxiety sensitivity is an individual difference variable consisting of beliefs that the experience of anxiety/fear causes illness, embarrassment or additional anxiety.

Anxiety experience is related primarily with an anxiety provoking stimulus situation, anxiety-sensitivity is related to a cognitive framework which one has acquired, which can provoke a reaction of anxiety in absence of sufficiently powerful stimulus. Anxiety varies in intensity from mild to strong feelings of uneasiness and nervousness. Anxiety sensitivity is not the experience of anxiety, it is an increased alertness to stimuli (signaling the possibility of becoming anxious, increasing worry about the possibility of becoming anxious and increasing motivation to avoid anxiety-provoking stimuli). Anxiety is associated with a wide range of physical illness. On the other hand, anxiety-sensitivity may be a risk factor for the occurrence of anxiety disorders, particularly panic disorders. Therefore its relation to physical illness may be indirect.
It seems to be important also to distinguish anxiety sensitivity from trait anxiety. There is disagreement as to whether anxiety sensitivity is conceptually distinct from trait anxiety, and relatedly, whether anxiety sensitivity adds predictive utility beyond trait anxiety regarding the development of anxious symptoms (Lilienfield, 1996). Reiss (1997) discussed the conceptual and theoretical difference between trait anxiety and anxiety sensitivity. He said that, trait anxiety began as a psychodynamic concept, poorly tied to observable and requiring Freudian defense mechanism to explain recurrent anxiety episodes. McNally (1989, 1996a, 1996b) and Taylor (1996) distinguished anxiety sensitivity from trait anxiety by noting that, whereas trait anxiety predicts future anxiety generally, anxiety sensitivity predicts future fear to anxiety sensations specifically. An important difference is that the two constructs use different indicators to predict future anxiety and fear.

Among adults, researchers have addressed the criticism by demonstrating, that, anxiety sensitivity is factorically distinct from trait anxiety (Peterson and Heilbronner, 1987; Taylor, 1996), that anxiety sensitivity predicts anxious responding to challenge and stress beyond trait anxiety (for example, Rapee and Medoro, 1994; Schostak and Peterson, 1990), and that anxiety sensitivity prospectively predicts the development of panic beyond trait anxiety (Schmidt et al., 1997).

Chorpita and Daleiden (2000) examined anxiety sensitivity in context of the tripartite model of depression and anxiety. They noted that the tripartite
model conceptualizes fear as an index of arousal and trait anxiety as related to negative affect. In children, the Anxiety Sensitivity Index was associated with autonomic arousal more so than with trait anxiety and fear. In adolescents, however, the anxiety sensitivity related to trait anxiety more so than to fear or arousal. The distinction become less marked if we see the new theory of trait anxiety given by Eysenck, which appears to be inspired by the concept of anxiety sensitivity, in fact it has tried to assimilate anxiety sensitivity in the new version of trait anxiety.

Eysenck (1997) proposed a new theory of trait anxiety, this being a 4-factor theory of anxiety. According to this unified theory, there are four sources of information, which influence the level of anxiety experience (1) External stimulation, (2) Internal physiological activity, (3) Internal cognitions, (4) One’s own behaviour. The unified theory is essentially based on cognitive biases, and is more reflective of the concept of anxiety sensitivity (without actually using the term) than anxiety disorder as such.

According to McNally (1994), anxiety is similar to catastrophic misinterpretation. However, anxiety sensitivity is different because the person does not have to misinterpret anxiety symptoms such as shortness of breath as something else like an asthmatic attack to occur. They simply must believe that their arousal from anxiety can lead to heart attack or insanity. In addition, anxiety sensitivity is dispositional, while catastrophic misinterpretation is episodic (Fridhandler, 1986). The concept of anxiety sensitivity was established due in part to observations that intense bodily sensations do not
always lead to panic attacks. This fact is demonstrated in studies that found hyperventilation challenges and carbon dioxide inhalation to elicit responses form participants that ranged from terror to pleasure (Clark and Helmsley, 1985).

Anxiety sensitivity, or the idea that anxiety is not equally motivating to all people (Reiss and McNally, 1985), is a cognitive, individual difference variable consisting of belief that the experience of anxiety and fear causes illness, embarrassment or additional anxiety, and that these anxiety related sensations have harmful physical, psychological, or social consequences. People show important individual difference in how they react to anxious arousals. Most people who notice they are anxious they may notice a pounding heart, shortness of breath, or the ‘shakes’ – expect the anxiety to dissipate when the situation that is worrying them is resolved. A small percentage of people, however, misinterpret the signs of anxious arousal as threatening or dangerous. Those people believe that a pounding of heart can lead to a heart attack, or that shortness of breath can lead to an asthma attack, or that shaking is a sign to mental illness. This group is said to have “high anxiety sensitivity”. People with high anxiety sensitivity scores respond with alarm and may interpret an inability to concentrate on a task as a assign of mental illness etc. And, those with low anxiety sensitivity scores interpret these same symptoms as just unpleasant (Reiss & McNally, 1985). Furthermore, those with high anxiety sensitivity scores report more intense symptoms due to hyperventilation when the objective measure of heart rate
Anxiety sensitivity is a construct that denotes an individual difference in fear of anxiety. Just as people vary in their proneness to feel anxious, so they differ in their fear of feeling anxious — their anxiety sensitivity. Most recent research evidences suggest the factor structure of the ASI to be different for different ethnic/cultural groups among African (Carter, Miller, Sbrocco, Suchday and Lewis, 1999; Zvolensky et al., 2003).

Another concern involves the relation of anxiety sensitivity to depression. Among adults, anxiety sensitivity and depressive symptoms are correlated (Catanzaro, 1993; Otto, Pollack, Fava, Uccello, and Rosenbaum, 1995; Schmidt et al., 1997; Taylor, Koch, Woody and McLean, 1996), raising the question if anxiety sensitivity is specific to anxiety, or instead is associated with emotional distress in general. In response to this concern, Taylor et al. (1996) argued that two aspects of anxiety sensitivity — fear of bodily sensations and fear of publically observable symptoms — are specific to anxiety, whereas a third aspect — fear of loss of cognitive dyscontrol (i.e., phrenophobia) — is specific to depression not anxiety. Also in response to this concern, Schmidt, Lerew and Joiner (1998) demonstrated that nonphrenophobic aspects of anxiety sensitivity predicted future anxious symptoms controlling for depressive symptoms, but that phrenophobia predicted both depressive symptoms and anxious symptoms.
Anxiety sensitivity is also associated with depressive symptoms among youth (Hayward et al., 1997; Kearney et al., 1997), and Weems et al., (1997) reported this association even controlling for anxious symptoms. Few studies have examined factors of anxiety sensitivity and their relation to anxious versus depressive symptoms in children and adolescents (Chorpita and Daleiden, 2000; Laurent et al., 1998); Silverman, Ginsburg and Goehart, 1999, have reported factors of anxiety sensitivity among youth.

Another set of group differences that have been observed but much less extensively examined are gender differences. Females typically score significantly higher than males on the full 16-item version of ASI (Peterson and Phehm, 1999; Peterson and Reiss, 1992). For example, Stewart, Taylor and Baker (1997) examined gender differences in: (i) the lower — or higher - order factor structure of the ASI, and/or (2) pattern of ASI factor scores. 290 male and 528 female university students completed the ASI. Separate principle components analyses (PCAs) on the ASI items of the total sample, males, and females revealed nearly identical lower-order 3-factor structures for all groups, with factors pertaining to fears about the anticipated (a) physical, (b) psychological, and (c) social consequences of anxiety. PCAs on the lower-order factor scores of the 3 sample revealed similar unidimensional higher order solutions for all groups. Females scored higher on the physical concerns factor relative to their scores on the social and psychological concern factors, and males scored higher on the social and psychological concerns factors relative to their scores on the physical concerns factors.
Finally, females scored higher than males on the higher-order factor representing the global anxiety sensitivity construct.

Furthermore, it also seems that gender moderates some of the effects of anxiety sensitivity. For example, women high in anxiety sensitivity have been found not only to be more susceptible to pain (Keogh and Birkby, 1999) but also exhibit different coping biases when compared to men high in anxiety sensitivity (Stewart, Conrod, Gignae and Pihl, 1998).

The above discussion shows that most of the researches focus on social (family), emotional and personality factors that in one way or the other contribute to asthma. But, emotional reactions do not occur without a stimulus. They are anchored in certain events. Therefore, it is important that we probe those factors which create an attitudinal perspective that triggers off emotions. An individual’s perceptions and views of the world in which s/he functions determine his/her reactions, feelings, and emotions. In the present research an attempt is being made to study, two factors which appear important namely attributional style and anxiety sensitivity. Attributional styles are the beliefs about causations which are consistently expressed by the individual. Certain attributional styles have been found to be associated with certain pathologies. There may be some attributional style distinctive to the asthmatics and studying this can be an important contribution to psychological research. Anxiety sensitivity is also an important and relevant variable which may explain the negative emotions associated with asthma.
The major objectives of the present study were to find the attributional style and level of anxiety sensitivity of asthmatics. These objectives were further enlarged by taking into consideration the tenure of disease, age and gender. In this context the following hypotheses were formulated:

1. Asthmatics will depict an attributional style different from non-asthmatics.
   1.1 Asthmatic males will have an attributional style different from non-asthmatic males.
   1.2 Asthmatic females will have an attributional style different from non-asthmatic females.

2. Asthmatics with different duration of illness will differ in their attributional style.
   2.1 Asthmatics with illness duration of 1 year or less will differ in their attributional style from asthmatics with illness duration of 1 to 5 years.
   2.2 Asthmatics with illness duration of 1 year or less will differ in their attributional style from asthmatics with illness duration of more than 5 years.
   2.3 Asthmatics with illness duration of 1 to 5 year will differ in their attributional style from asthmatics with illness duration of more than 5 years.
3. Asthmatics of different age groups will differ in their attributional style.

3.1 Asthmatics of ages 12 to 20 years will have an attributional style different from those of ages 20 to 35 years.

3.2 Asthmatics of ages 12 to 20 years will have an attributional style different from those of ages 35 to 50 years.

3.3 Asthmatics of ages 20 to 35 years will have an attributional style different from those of ages 35 to 50 years.

4. Asthmatic males will depict an attributional style different from asthmatic females.

5. Asthmatics will have higher anxiety sensitivity than non-asthmatics.

5.1 Asthmatic males will have different level of anxiety sensitivity from non-asthmatic males.

5.2 Asthmatic females will have different level of anxiety sensitivity from non-asthmatic females.

6. Asthmatics with different duration of illness will differ in their level of anxiety sensitivity.

6.1 Asthmatics with illness duration of 1 year or less will differ in their level of anxiety sensitivity from asthmatics with illness duration of 1 to 5 years.

6.2 Asthmatics with illness duration of 1 year or less will differ in their level of anxiety sensitivity from asthmatics with illness duration of more than 5 years.
6.3 Asthmatics with illness duration of 1 to 5 year will differ in their anxiety sensitivity from asthmatics with illness duration of more than 5 years.

7. Asthmatics of different age groups will differ in their level of anxiety sensitivity.

7.1 Asthmatics of ages 12 to 20 years will have different level of anxiety sensitivity from asthmatics of ages 20 to 35 years.

7.2 Asthmatics of ages 12 to 20 years will have different level of anxiety sensitivity from asthmatics of ages 35 to 50 years.

7.3 Asthmatics of ages 20 to 35 years will have different level of anxiety sensitivity from asthmatics of ages 35 to 50 years.

8. Asthmatic males will differ from asthmatic females in their level of anxiety sensitivity.
Chapter – II

Review of Literature
Review of earlier researches conducted in the same field is an inseparable part of any study because every research/investigation contributes to the understanding of the field. No research can be seen as an isolated effort, but as part of a collective venture in search of the truth. It is quite essential that previous viewpoints and findings regarding the phenomena be taken into consideration.

The present research was conducted with the purpose of finding attributional styles and anxiety sensitivity among asthmatic. It is imperative that the investigator should explore the researches relating asthma to various psychological factors/variables. The second chapter is divided into three parts. In the first part studies relating psychological variables and asthma are reviewed. The second part concerns attributional style and health-related functioning. Third part deals with the studies relating to anxiety sensitivity in various samples. The study of the etiology of asthma dates back to at least to Hippocrates who is credited as being the first to describe asthma. He believed that asthma was caused by a disturbance of humors that had not been cleansed from the brain before birth. Further, in his ‘treatise on Asthma’, the renowned 12th century physician Moses Maimonides initially hypothesized that emotions are associated with asthma (Jones et al., 1999; Munster, 1968). The psychological factors that have been studied so far in relation to asthmatic symptoms are stress, pathogenic family relations, personality pattern, and affective comorbidity like depression, negative affectivity, anxiety and other anxiety related disorders.
The role of stressful life events in the etiology of various diseases has been a fertile field of research. Prior research endeavour established this point beyond doubt that there exists a positive relationship between stressful life events and the precipitation of the psycho-physiological diseases. The stress theory developed by a distinguished Canadian endocrinologist and biomedical researcher, Selye (1956, 1969) and his followers over the last generation (Dohrenwend and Dohrenwend, 1974; Rahe, 1974), that biochemical changes brought on by stress are eventually self damaging, has received wide acceptance. During the last two decades, investigators have shown that the recent life histories of hospitalized persons contain significantly more frequent and stressful life events than do histories of matched controls from the general population (for example, Paykel, 1974). Selye (1976) opined the notion that negative stress (distress) increases the vulnerability of the individual’s psychological and physical health and thus do more damage. There are plenty of evidences to show that the reactions to stress can lead to the development of asthma. A study conducted by Bengtsson (1984) revealed that stressful life events can even trigger an asthmatic attack in an individual already predisposed to develop this disease.

It is thought that emotional, environmental, and other personal stressors cumulatively leave biochemical scars that eventually result in ulcers, hypertension, asthma and other psychophysiological diseases (Gottschalk, 1975). Likewise, Aitken, Zeally and Rosenthal (1969) and, Plutchik et al. (1978) have suggested that emotions and life stress can affect the severity of
asthma symptoms, it can cause certain biochemical changes which in turn result in the development of asthma.

In a study conducted on stressful life events, personality and asthma, Pawar (2003) concluded that negative life events experienced by asthmatics have significant impact on the health of asthma patients. Other studies conducted on stress and asthma suggested that stress can produce bronchoconstriction and between 20 per cent and 40 per cent of subjects with asthma experience exacerbations of symptoms during periods of stress (Isenberg, Lahrer and Hochron, 1992).

Carr, Lehrer, Hochron and Jackson (1996) assessed airway impedance (that is, difficulty/hindrance in breathing) responses to psychological stressors among 113 individuals: 61 with asthma only (AS), 10 with asthma and panic disorder (ASPD), 24 with panic disorder only (PD), and 18 controls with neither condition (CON). They excavated that individuals with either AS or PD were affected by psychological stressors.

In a study conducted by Ritz, Steptoe, DeWilde and Costa (2000), 24 patients with mild to moderate asthma were compared to an equal number of age-matched controls. Both groups were exposed to stress in two ways: they were asked to complete subtraction problems while someone pressure them and they viewed emotionally charged films and slides. The asthma patients responded to stress with greater increase in respiratory resistance (the resistance offered to the passage of air through respiratory tract), blood
pressure etc. than control group experienced; they also reported high levels of depression, arousal, and shortness of breath.

On the basis of their observations, Rietveld, Beest and Everaerd (1999) argued that stress can be sufficient to induce breathlessness in asthmatic patients. Stress was induced by frustrating computer task in 30 adolescents with asthma and 20 normal controls, aged 14 to 19 years, stress measures were self-reported emotions, heart rate and blood pressure. High levels of negative emotions and stress were noticed and confirmed in asthmatic patients.

In a study, Liu and colleagues (2002) posited that the stress of school examination on otherwise apparently healthy young college student with no asthma had severe effect. The data strongly support the contention that stress promoted at least some hallmark of inflammation associated with asthma.

Among children with asthma, but without high level of chronic stress, acute psychological stress or stressful life events significantly had both an early and a late effect, in increasing the risk of new asthma attack on exacerbations. The early/immediate effect occurred in the first day or two after the stress and the late effect at weeks 5 to 7 (Archivist, 2005). Sandberg and colleague (2004) attempted to track the frequency and severity of asthma attacks in school aged children, none of the children in the study were felt to have chronic high stress at the start of the study. For 18 months, separate teams of researchers, who did not have access to the other team’s findings,
independently evaluated the children’s asthma and stressful life events. Children in the study averaged 2 major stressful life events during the study - events such as the death of a pet, a close friend’s family moving away, parent’s divorce or separation, the death of a grand parent, or becoming the victim of a serious bullying incident. The findings showed that these stressors had both immediate and delayed effects on the children’s asthma. Within two days of each stressful event, the risk of having an asthma attack skyrocked up nearly five-fold. After the first 48 hours, there was no increased risk - until 5 to 7 weeks later, when the risk of another attack nearly doubled.

Moreover, psychological distress in children has been associated with asthma that is more difficult to manage (Fritz and Overholser, 1989) with more frequent and lengthier admission to the hospital (Kaptein, 1982); and greater functional disability (Gutstadt et al., 1989). Some patients with asthma experience increased broncho-constriction in response to acutely distressful situations (Lehrer, 1998) (see Forsythe et al., 2004).

Archea et al. (2006) conducted a study on 189 adults with asthma. They analyzed responses to a self-completed questionnaire assessing negative life events and asthma related quality of life. They concluded that negative life events are associated with quality of life among adults with asthma.

Kleeman (1967) interviewed twenty-six patients over an eighteen month period. According to the reports of these patients, 69 per cent of their attacks began with an emotional disturbance.
Several studies that focused on the role of pathogenic family patterns have considered parent-child interactions important in the etiology of asthma. Mothers of asthmatic patients generally have an ambivalent feeling towards these children. They tend to reject them, while at the same time being overprotective and unduly restrictive of children's activities (Lipton, Steinschneider and Richmond, 1966; Olds, 1970). In one investigation Mrazek et al. (1991) began with 150 pregnant women who had asthma. Because asthma has a genetic component the investigators intended to study at risk offspring and assess parental characteristics as well. The parents were interviewed three weeks after the birth to determine their attitude towards the infant, their strategy for sharing parenting duties, and the presence of any emotional disturbance. The children were closely monitored over the next two years, and the frequency of asthma were than related to the parental characteristics noted earlier. Among the families who were rated as having problems, 25 per cent of the children developed asthma as compared with only 8 per cent of the children from the other families.

The mothers of asthmatic children have been seen as excessively controlling, and creating an emotionally tense home environment and thus attempting to prevent their children's growth and independence. This observation has had a very limited kind of verification from the frequent observation that when asthmatic children are removed from their homes, their symptoms almost always improve because this improvement might also be traced to being removed from hidden allergens or other extrinsic triggers.
To evaluate the role of mother, family and the home, Purcell et al. (1969) designed a complex experiment in which asthmatic children would stay in their own home, without their parents. Thirteen families in which emotional factors seemed to play a leading role in attack were selected. Another group of families in which children's attacks did not seem related to emotional circumstances was also chosen. Every family member but the asthmatic children moved out of the home for two weeks and a house-keeper was employed to care for the child. During the separation the asthmatic children for whom emotional factors were low showed no important change in their disease. The children for whom emotional factors were rated high demonstrated remarkable improvement. But when the two week experimental separation was over and the parents returned, the improved children returned to their old pattern.

The parents of the asthmatic child also frequently become anxious and tend to be overprotective towards the child. Occasionally, family disputes or marital problems may be found to be major factors in the etiology of intractable asthma. Because individuals coming out of such family backgrounds tend to be overdependent and insecure, it would hardly be surprising if they should react with chronic emotional mobilization to problems that do not seem threatening to most people. It is likely that such psycho-social factors in a family will make their contribution to the severity of asthma.
Although, these researches suggest that the home life of some asthmatic children may play a role in their disease. Some researchers do not find any difference in the adjustment level and disciplinary practices of the mother's of asthmatic and normal children (Gauthier et al., 1977, 1978; Eiser, Eiser, Town and Tripp, 1991). In such cases asthma which has allergic or infectious causes may just be perpetuated by family attitudes. Parents of these children may unwittingly reward various symptoms of the syndrome by catering to asthmatic children and treating them especially because of the asthma. Their mothers tend to become ambivalent, protective, and restrictive after the asthma appears.

A large body of literature suggests a common personality profile descriptive of asthmatic patients or of a specific type of nuclear conflict, for example, unresolved dependency on the mother, or unconscious fear of the loss of their mother and mothering image. The suggestion that unresolved dependency conflicts are central in the development of asthma is contained in the influential writings of Alexander, Fench and Pollock (1968), they explained how personality traits give rise to particular psychosomatic symptoms by using the concept of regression. According to Alexander et al. (1968), adult emotions stir up childhood organ fixations. A child deprived of sufficient mothering (nursing) during the oral stage fixates his or her need. French and Alexander (1941) hypothesized that asthma may often be viewed as a “suppressed cry”, with the stated implications that asthmatic children cry less than non-asthmatics, particularly around critical periods of separation.
conflict. Dynamic theorists have attributed asthma to the relationship between an overly fond mother and clinging child. Most of them can trace their explanations of psychosomatic disorder to Freud (1931), who believed that psychophysiological symptoms were "organ neuroses". The symptoms symbolically expressed the patient's hidden emotional needs. The wheezing or coughing, for example, were seen as a 'repressed cry for help'. The patients wanted to be an infant again and was metamorphically looking for mother.

Many authors have implied that certain personality characteristics bear a causal relationship or the development of the asthmatic symptom; others have noted that they appear secondary, resulting from symptom and its effects on the patient and family. Asthmatics, particularly children are anxious, dependent, conforming, insecure, lacking in self-confidence and hypersensitive. According to Alexander, French and Pollock (1968) and Gottschalk (1975) asthmatics are overdependent, infantile, want to be cared for, have ambivalent feeling towards self and others. Several investigators have found that asthmatic individuals have many neurotic symptoms such as dependency (Herbert, 1965), meekness, sensitivity, anxiety, meticulousness, perfectionism and obsessions (Rees, 1964).

First, Neuhaus (1958) compared the personality test scores of asthmatic children with those of a group of normal children and a group of children with cardiac conditions, and found that both asthmatic and cardiac patients (children) were significantly more maladjusted (anxious, insecure,
and dependent) than a normal control group. While the asthmatics were found to be more neurotic than the other two groups.

In a recent study, Pawar (2003) studied specific personality patterns of asthmatics and observed that the asthmatic patients scored high on such personality variables viz. as irrationality, locus of control, repression-sensitization and anxiety. Asthmatics possessed more field-dependent style of personality as compared to the normals.

To conclude, attempts to study particular constellation of personality traits linked to asthma as yet gave results that are inconclusive.

Asthmatics are also at an increased risk for affective comorbidity specifically anxiety, depression and panic disorder. Considerable evidence exists that asthma symptoms continue well into adolescence and early adulthood for 90 per cent of patients (Kelly, Hudson, Phelan, Pain and Olinsky, 1987). Therefore, asthmatic patients usually experience psychological sequela (Beuder and Klinnert, 1998; Creer, Harm and Marion, 1988; Lehrer, Isenberg and Hochron, 1993; Silverglade, Tosi, Wise, and D’Costa, 1994), including anxiety, mood and behavioural disorders, and poor self-esteem and social competence (Vila, Nollet-Clemenccon, Vera, et al., 1999). Several researches evidence that asthma and these psychological states and traits may mutually potentiate each other through asthma triggers and inaccuracy of asthma symptom perception etc.
Jones, Wagener, Lando and Feldman (1999) conducted a study to examine the association of symptoms of anxiety and depression with increased risk of developing asthma. The association between asthma incidence and anxiety and depression were tested among 5,231 baseline nonasthmatics (aged 25-74 years) using C. Cox proportional hazards regression. A clear risk gradient was observed for both anxiety and depression symptomatology. The effects of anxiety and depression were particularly strong among nonsmokers without respiratory symptoms.

Bussing, Burket and Kelleher (1996) compared 37 asthmatic children with 31 matched healthy controls for DSM-III-R anxiety disorders and observed that the asthma group had significantly more total anxiety disorders, past school problems, past psychiatric illness, and intrafamilial stress, and there was also more family history of emotional problems.

Nascimento et al. (2002) evaluated the frequency of anxiety disorders in 86 asthmatic outpatients (aged 13-80 years). Psychiatric diagnosis were assessed with the Mini-International Neuropsychiatric Interview 4.4 version. 45 asthmatic patients reported at least one current anxiety disorder. The frequency of panic disorder with or without agoraphobia was 13.9% and that of agoraphobia without panic disorder was 26.8%. Social anxiety and generalized anxiety disorders occurred in 9.3% and 24.4% of the sample, respectively. 29 patients reported a major depressive episode. The psychiatric morbidity of the sample was 61.6%. The results supported the high morbidity
of anxiety disorders, particularly panic/agoraphobic spectrum disorders, in asthmatic outpatients.

Carr (1998, 1999) opined that the presence of asthma is a risk factor for the development of panic disorder. The occurrence of panic disorder in asthma was greater than would be expected based on their individual prevalence rates. This may be due in part to the important role of respiratory factors in panic disorder. Panic and anxiety can directly exacerbate asthma symptoms through hyperventilation.

Gillaspy et al. (2002) revealed that adolescents with asthma experienced higher levels of anxiety, depression and global psychological distress than normals. Asthmatic adolescents, already at risk for adjustment problems, with secondary to lower economic strata and educational or vocational failure, may be more likely to experience psychological distress than normals.

Silverglade, Tosi, Wise and D’Costa (1994) studied 129 asthmatic adolescents (aged 12-18 years) and a group of 74 healthy, non-asthmatic adolescents. Differences in selective (irrational beliefs) and emotional (anxiety, depression and hostility) characteristics were examined. Multivariate analysis indicated that irrational beliefs in the importance of approval and the lack of control of emotions, along with self-reported anxiety, depression, or hostility, were strongly associated with asthma severity.
Wamboldt et al. (1998) carried out a study in which subjects were 337 children (aged 7-19 years). Children's asthma severity was rated by experienced paediatric asthma specialists. Children filled out the Children's Manifest Anxiety scale and the Weinberger Adjustment Inventory. Child rated anxiety symptoms were unrelated to asthma severity or to markers of asthma functional morbidity. Results revealed that children with severe asthma did not rate themselves as having higher level of anxiety than those with mild or moderate asthma or than standardized norms.

Hommel et al. (2003) tested the differential condition of illness uncertainty of self-reported anxiety and depression in the sample of 56 adolescents (aged 18-21 years) with childhood-onset asthma. Measures of illness uncertainty, anxiety and depression were completed by the subjects and objective assessments of illness severity were obtained with the help of semi-structured interview and pulmonary function test. Results revealed that illness uncertainty contributed significant variance to anxiety after statistically controlling the effects of demographic and disease parameters and depressive symptomatology; illness uncertainty did not contribute significant variance to depression. Earlier, results obtained by Hommel et al. (2002) indicated that the combination of anxiety and depression severity contributed significant variance to asthma quality of life after statistically controlling demographic and disease covariates. Moreover, anxiety demonstrated a significant main effect on asthma quality of life.
Ten Thoren and Petermann (2000) argued that the main characteristic of asthma is sudden and unexpected attacks of impaired breathing. Both the attacks themselves and the prospect of attacks generate much anxiety amongst patients. Several different forms of anxiety can be identified which vary in intensity and the situations in which they appear. Anxiety disorders are more common in asthmatics and have a considerable influence on asthma management because they influence symptom perception. Excessive anxiety about asthma symptoms can affect the patient's response to an attack; anxiety related to asthma triggers can reduce the patient's quality of life and anxiety related to medical treatment can influence compliance.

Deshmukh, Toelle, Usherwood, O'Grady and Jankins (2007) reviewed researches concerning asthma and anxiety disorders. They suggested the increased probability of the prevalence of anxiety disorders, and particularly panic disorder and panic attacks in patients with asthma, as compared to a normal population. Research also indicates significant levels of co-morbidity between asthma and anxiety as measured on dimensional scales of anxiety and panic. Clinical anxiety and panic manifestations affect symptom perception and asthma management through the effects of anxiety symptoms such as hyperventilation, and indirectly through self-management behaviour and physician response.

Krommydas and others (2004) examined the relation between depression, anxiety and pulmonary function in asthmatics. Thirty eight adult asthmatic patients underwent psychometric evaluation with DSSI/sAD
questionnaire, filled in asthma questionnaire and underwent spirometry. The majority of patients suffered from mild-persistent asthma. Twenty-six reported symptoms of anxiety and 25 reported symptoms of depression. These findings indicate a high frequency of depression and anxiety in adult asthma patients.

Phillipp and his associates (1972) demonstrated that the expectancy of asthmatic attack is an important factor in this respiratory disorder. He compared the reactions to bronchospasmatic and neutral substances of a group of allergic asthmatics (based on skin test reactivity) to a group of non-allergic asthmatics. The findings, based on measures of breathing capacity were that the non-allergic group had more asthmatic attacks than the allergic group in response to both the bronchospasm-inducing and the neutral inhalants. The investigators interpreted these results as indicating that the psychogenic group’s greater fear or expectancy of asthmatic attacks were responsible for the differences. They also learned that the non-allergic or psychogenic group also benefited more from relaxation training than the allergic group, suggesting again that fear of attack may be important in triggering the asthma attack itself.

Specific emotional states particularly negative affectivity in relation to asthma has also been extensively studied by many investigator. Miller and Wood (1997) studied twenty four children aged 8-17 years with moderate to severe asthma. The subjects viewed the movie E.T., the Extra-Terrestrial while having their heart and respiration rate and oxygen saturation
continuously recorded. Specific scenes were identified and preselected to evoke sadness, happiness, and a mixture of happiness and sadness. Self-report of emotion and indices of physiological response were analyzed for these targeted scenes. Results indicated that sadness was associated with greater heart rate variability and instability of oxygen saturation compared with happiness, with mixed result for happiness and sadness. They concluded that results support sadness as evoking patterns of autonomic influence consistent with cholinergically mediated airway constriction. Happiness appears to effect autonomic patterns that would tend to relieve airway constriction.

Opolski and Wilson (2005) carried out a review of the researches conducted on depression and asthma. The main findings from this review included that sadness and depression can produce respiratory effects consistent with asthma exacerbations.

Put and others (2004) investigated the effect of suggestion on subjective and objective asthma symptoms as a function of negative affectivity. Findings showed that asthmatics (N=32) with high negative affectivity and overall more intense asthma symptoms. They also reported more airway obstruction after suggested bronchoconstriction and less after suggested bronchodilation, whereas person with low negative affectivity did not show such variation. These effects were unrelated to social desirability. They concluded that self-reported symptoms of asthmatics with high negative affectivity are more influenced by suggestion than those of patients with low negative affectivity.
Smith and Nicholson (2001) conducted a longitudinal study of 92 asthmatic adults to investigate the role of psychosocial factors in exacerbations of asthma in adults induced by upper respiratory tract infections (URTIs). The results showed that those who experienced at least one episode reported more negative life events, high negative affectivity and low social support.

Put, Demedts, Van Den Berge, Demyttenaere and Verleden (1999) carried out an empirical study on 116 asthmatic patients and hypothesized that the symptom reporting in asthmatics does not necessarily correspond to clinical status, but may be directly or indirectly mediated by personality, such as negative affectivity.

Priel, Heimer, Robinowitz and Hendler (1994) studied the role of negative affectivity on patients’ perceptions of behaviour during asthma attacks among 47 asthma subjects. Patients completed 17 to 30 daily questionnaires assessing negative affect, asthma perception, additional drug intake, search for medical assistance, and peak-flow measures of respiratory distress. Asthma perceptions were correlated with negative affect and educational level; the perception of the asthma severity, but not negative affect, did predict behaviour during an attack.

Attributional theories have been used as a framework for understanding diverse issues and topics, most intriguing of which are the problems of health—physical as well as psychological. An emerging body of
literature has addressed the relationship between attributional style and various health-related functioning supporting the contention that the way people attribute the causes of negative and positive events/situations has an important link with the physical and psychological well-being of the individuals.

Since the attributional theory emerged out of the learned helplessness model of depression its role in depression is well documented.

Sweeney, Anderson and Bailey (1986) reported that for negative events, attributions to internal, stable, and global causes had reliable and significant association with depression. The relation between attribution factors of ability and luck was also significant but it was stronger for negative events.

Greenberg, Pyszczynski, Burling and Tibbs (1992) examined whether depressive self focusing style account for the lack of self serving attributional bias in depressed person. They found that conditions analogous to non depressed patterns of attributional focus led to self serving attributional bias for all subjects.

Dixon and Ahrens (1992) carried out a longitudinal study to assess the ability to interaction of attributional style and daily negative events to predict self reported depression in 84 children. The self reported depression symptoms were assessed before and after exposure to stressful event. It was found that attributional style did not predict change in self reported depression
symptoms following stressful events, the interaction of attributional style with stress did predict them. Stress predicted depression symptoms as well.

Hanger and Lund (2002) investigated how self-concept and attributional style are related to depression. On the basis of an inventory, 166 teacher students (mean age 25.3 years) were scored on general and academic self-esteem, attribution for positive and negative events and depression. The two self-esteem variables were found to constitute important predictors of depression, while the contributions of the attributional variables were of minor importance. In addition, pessimistic attributions to both positive and negative events resulted in higher depression than pessimistic attributions to either kind of events, and to neither kind of events. Finally, factor analysis resulted in interpretable solutions.

Studies focusing efforts to explore relationship between attributional style and anxiety yield conflicting results.

Kenardy, Evans and Oei (1990) investigated the relationship between the development of panic disorder and attributional style by administering the Attributional Style Questionnaire (ASQ) to 28 subjects with panic disorder with agoraphobia and 21 subjects with other anxiety disorders who had experienced a panic attack at some time. No significant differences were found between the groups suggesting that cognitive style as assessed by the ASQ may not predispose to the development of panic disorder. However, evidences from a variety of sources suggest that early experience with
diminished control may foster a cognitive style characterized by an increased probability of interpreting or processing subsequent events as out of one’s control, which may represent a psychological vulnerability for anxiety (Chorpita and Barlow, 1998).

Ahrens and Haaga (1993) conducted a study in which 94 undergraduate students completed measures of trait positive and negative affectivity, anxiety, depression, optimism, hopelessness, and attributional style (ATS). After writing about negative events or hearing a tape describing a positive academic experience, subjects completed measures of state positive and negative affect and self-efficacy expectancies. Positive affectivity was associated with ATS for positive, but not negative, events. Negative affectivity was associated with ATS for negative, but not for positive, events. Negative event ATS was specifically associated with anxiety expectancies and positive event ATS was associated with depression. ATS predicted state positive affect following the positive tape. Effects of ATS on affect were partially independent of expectations.

Bell-Dolan and Wessler (1994) postulated that, although the role of causal attributions in children’s anxiety is important from theoretical and practical standpoints, knowledge of anxious children’s attributions and incorporation of knowledge into specific treatments is quite limited. Attributional style is included in several theories of anxiety, with particular reference to external locus of control and stable attributions for negative situations. Adult literature support a relationship between anxiety and
negative attributional style, with negative attributions most strongly related to social anxiety. Additionally, the stability dimension seems most consistently related to anxiety. Although the child literature is less developed, it suggests that similar relationships may hold for child anxiety and attributional style.

Lynd-Stevenson and Rigano (1996) proposed that research by Ahrens and Haaga failed to support the prediction the expectancy mediates the relationship between attributional style for negative outcomes and anxiety because the measure of expectancy failed to evaluate the type of expectations directly involved in the etiology of anxiety (i.e., threat expectancy). 104 college students (aged 18-48 years) were interviewed, and their scores on measures of attributional style for positive and negative outcomes, threat expectancy regarding the prospect of future unemployments, and anxiety about future unemployment (unemployment anxiety) were obtained. Findings support the prediction that threat expectancy mediates the relationship between attributional style for negative outcomes and unemployment anxiety. There was also evidence consistent with the tripartite model of anxiety and depression that attributional style for positive outcome is unrelated to the cognitive processes that generate anxiety.

Luten, Ralph and Mineka (1997) carried out two studies with college students and explored the relationship of a pessimistic attributional style to positive and negative affect, as well as to depressed and anxious mood. Both studies revealed that a pessimistic attributional style was correlated with negative affect and depressed mood, but was unrelated to low levels of
positive affect. The second study also showed a correlation with anxiety, and that the association of pessimistic attributional style with emotional distress occurs for both depression-relevant (that is, loss/failure) as well as anxiety-relevant (that is, threatening) events. Results supported the hypothesis that pessimistic attributional style is a nonspecific diathesis for symptoms of both anxiety and depression.

Swendson (1997) applied the experience sampling method to test the helplessness-hopelessness theory of relationship between patterns of anxiety and depression (L.B. Alloy et al., 1990) in 44 undergraduates (mean age 19 years). 22 subjects categorized as having low depression and anxiety and "low risk" attributional style provided 5 daily self-reports of negative events, attributions, and anxious and depressed mood immediately after negative events. Attributional style predicted these causal attributions, but did not directly explain changes in post event depressed mood. Despite support for more established components of the theory, no support was found for newer aspects concerning the relationships of control attributions to anxious mood.

Rodriguez and Pehi (1998) examined the pattern of relationships among attributional style, depression, and anxiety in a sample of 69 New Zealand Children (aged 8-14 years), and evaluated the specificity of maladaptive attributional cognition to depression. Subjects responded to 3 self-report measures: the Children Depression Inventory, the Children's Manifest Anxiety Scale-Revised and Children's Attributional Style Questionnaire. Both depression and anxiety scores were significantly
correlated with attributional style. However, multiple regression analysis revealed that depression but not anxiety significantly predicted overall attributional style. Thus anxiety was no longer significantly correlated with maladaptive explanatory style upon controlling for depression.

Waschbusch, Sellers, LeBlanc and Kelley (2003) evaluated whether anxiety influences the relationship between helpless attributions and depression. Results showed that male adolescents with anxiety only had helpless attributions style that were similar to male adolescents with depression, but the same was not true for female adolescents. Results also suggest that helpless attributions may be related to both anxiety and depression in males.

An extensive body of literature has addressed the relationship between attributional style and physical or psychological health.

In Virginia Polytechnic study, Peterson (1988) found that individuals who believed that stable plus global factors caused bad events, experienced more days of illness in a month and visited physicians more frequently in a year. They also reported more unhealthy habits, lower efficacy to change the habits, and more stressful occurrences than subjects who experienced bad events with unstable plus specific causes. Optimistic individuals who explain bad events with external, unstable and, specific causes experience better health than the pessimists, who explain bad events with internal, stable and global causes (Peterson, 1995).
Dua (1994) determined the comparative predictive value of attributional style in predicting self-reported physical and psychological health. He observed that global attributions for bad events were better predictor of health than those for good events.

Pessimistic explanatory style is a risk factor for illness, but the factor linking explanatory style and illness are unknown. One’s characteristic response to poor health may mediate this relationship. Perhaps pessimistic individuals act helplessly in the face of their symptoms, thereby exacerbating disease. In a study, Lin and Peterson (1990) investigated this possibility and observed that subjects who explained bad events pessimistically (with internal, stable, and global causes) reported more frequent illness during the past year and rated their overall health more poorly than those who habitually favour external, unstable and specific explanations. When ill, pessimistic subjects were less likely than their optimistic counterparts to take active steps to combat their illness. Results suggested that one pathway leading from pessimistic explanatory style to poor health is mundane: passivity in the face of disease.

A study of Harvard University graduate assessing pessimistic explanatory style at age 25 found that these men had significantly poorer health or were more likely to have died when they were assessed 20 to 35 years later. Explanatory style was extracted from open-ended questionnaires filled out by 99 graduates of the Harvard University classes of 1942-1944 at age 25. Physical health from ages 30 to 60 as measured by physician
examination was related to earlier explanatory style. Pessimistic explanatory style predicted poor health at ages 45 through 60, even when physical and mental health at age 25 was controlled. Pessimism in early adulthood appears to be a risk factor from poor health in middle and late adulthood (Peterson, Seligman and Vaillant, 1988).

Metalsky et al. (1997) examined whether the negative attributional style featured in helplessness/hopelessness theory would moderate the exhibition of depressive symptoms in 22 bulimic females as compared with 14 depressed patients. Results indicated that clinically bulimic subjects with a negative attributional style exhibited depressed symptoms whereas clinical bulimics without a negative attributional style did not. Bulimic subjects with a negative attributinal style feel severe range of symptom severity as opposed to bulimic subjects with negative style who feel in normal range of severity. Additionally, attributional style moderate severity of depressed symptoms as much in bulimic as in depressed subjects.

Goebel, Spalthoff, Schulze and Florin (1989) studied a group of 44 bulimic women as well as 38 women with no indication of eating disorder, compared with respect to age, weight and height, and observed that bulimics showed significantly higher ASQ (bad negative events) scores. The findings provide first evidence that dysfunctional attributes and depressive attributional style are predominant in bulimic women but at the same time are not necessarily predictive of the severity of the disease.
Tamara, Waller and Rachel (2006) examined attributional style in the eating disorders for positive and negative events, independent of covariant effects of depression. Twenty-five eating-disordered women and 26 nonclinical women each completed measures of attributonal style, depressed mood, and eating pathology. They also completed a measure of verbal intelligence (to ensure comparability of groups). Women with an eating disorder had a greater tendency to attribute negative situations to the self when compared with nonclinical women, even when differences in depressed mood were controlled for. There were no comparable differences in positive attributional biases. Women with an eating disorder adopted a self-blaming style when evaluating negative events, and such self-blame was contributed to the maintenance of an eating disorder.

The attributional reformulation of helplessness theory predicts that stress coupled with a pessimistic explanatory style leads to negative outcomes, including physical illness, among at risk individuals. The longitudinal study of 198 college students examined whether pessimistic explanatory style interacts with perceived stress to predict subsequent illness, even when controlling for baseline illness. Results confirmed this hypothesis (Jackson, Sellers, and Peterson, 2002).

Uomoto and Fann (2004) have examined the perception of injury and explanatory style in symptomatic mild traumatic brain injury (MTBI). Participants were 22 adults with MTBI and 11 with moderate/severe traumatic brain injury (TBI). Results revealed that MTBI patients reported greater
injury severity and poorer cognitive recovery and rated their brain injury as affecting more areas of life than the moderate/severe TBI group. Pessimistic explanatory style was associated with poorer perceived recovery.

In Recurrent Coronary Prevention Pessimism study, Buchanan (1995) found that pessimism predicted death from coronary events over a period of 8½ years.

Hommel, Wagner, Chaney & Mullins (2001) examined the prospective contribution of attributional style in rheumatoid arthritis. 42 patients were followed over the course of 1 year and completed various measures at time 1 and time 2. Results revealed that a pessimistic attributional style at Time 1 significantly predicted lower self-rated disability at Time 2. Chaney et al. (2004) examined longitudinal relationships between causal attributions and depression symptoms in adults with rheumatoid arthritis. Cross-lagged penal correlations tested the temporal precedence of attributions relative to depression symptoms over 1 year. 42 participants completed self-report instruments on 21 occasions. Results showed that Time 1 attributions predicted increased levels of depression symptoms at Time 2 after perceived pain and disability were controlled; Time 1 depression symptoms were unrelated to Time 2 attributions. Cross-lagged correlation comparisons revealed statistical dominance for attribution-depression relationships relative to depression attribution relationships.

Love (1988) studied the attributional styles of depressed and non-depressed chronic low back pain patients (N=91) in order to test the Revised
Learned Helplessness model’s prediction of differences between the two. The results partly supported the hypothesis; an internal, stable, global style for negative events distinguished the depressed group from the non-depressed, but there were no differences in attributional style for positive events.

Buckelew and his colleagues (1990) studied locus of control beliefs among 160 subjects (67 males and 93 females) referred to a comprehensive pain rehabilitation program, and found that the younger male patients reported a strong internal attributional style. Older male patients relied more heavily on both chance and powerful other factors. Among women, cluster assignment was related to the use of coping strategies. It appears that the presence of both Internal and Powerful other health attributional styles is associated with less frequent use of cognitive self-management techniques.

In a study McGuigan (1995) examined the attributional style and depression in men receiving treatment for chronic back pain. 122 subjects (aged 22-55 years) completed the Attributional Style Questionnaire and the Beck Depression Inventory. Finding showed that there was no significant correlation between depression and negative attributional style among back pain patients.

Schoenherr, Brown, Baldwin and Kaslow (1992) focused on attributional style by examining 96 youth (aged 7 years to 16 years 11 months) diagnosed with insulin-dependent diabetes mellitus (IDDM), acute lymphocytic leukemia, or sickell-cell syndromes (SCSs). Disease/disability
parameters, including duration of diseases and age of disease onset, were examined to determine their relation to attributional style. Attributional style was an efficient predictor of youth’s self-reports of depressive symptoms, when controlling for demographic and disease-reports of depressive symptoms.

Cheng and Furnhaum (2001, 2003) examined to what extent attributional style (internal, stable, and global) predicts positive affect, self-reported happiness, mental health or psychological well-being in college students. Regression analysis showed that the Attributional Style Questionnaire was the significant predictor of happiness and mental health. Results indicated that optimistic attributional style in positive situations was a stronger predictor of self-reported happiness than mental health and pessimistic attributional style in negative situations was a predictor of both happiness and mental health.

Khan and Jahan (2006) in a study found that persons experiencing high sense of well-being differed from those experiencing low sense of well-being on attributional style. Persons having high sense of well-being had more internal attributions for the positive events and, more unstable and specific attributions for the negative events. On the other hand, the attribution of the persons having low sense of well-being were found more external for positive events and, more stable and global for negative events.
Few studies have reported gender differences in the role of attributional style. Previously, Rim (1990) suggested that men and women differ in the coping styles related to attribution. The coping styles most related to attribution in men were suppression, replacement and reversal and in women, the coping styles were blame seeking, succorance, replacement and reversal.

Rim (1991) investigated the relationship between neuroticism and extraversion and three attributional styles: internality stability and globality, for good and bad events. Results showed that women scoring low on neuroticism have significantly higher scores on good events than on bad events on all three attributional styles. Men scoring low on neuroticism attributed good events to more stable factors, whereas those scoring high on neuroticism attributed good events to more global factors. With regard to extraversion, low scoring men and women scored higher on good than on bad events on internality, and whether high or low on extraversion – men and women scored higher on good than on bad events for stability.

Bunce and Peterson (1997) investigated the links between explanatory style and established personality variables as measured by the California Psychological Inventory (CPI). Correlations with a pessimistic explanatory style were heavily concentrated among the class I variables of CPI for men, where as women’s pessimistic explanatory style was linked with well-being and good impression. Two major scales (sociability and socialization) showed significant sex differences with respect to their
correlation with explanatory style for negative events. The differential pattern of correlations suggests that explanatory style may be relevant to different personality domains for males and females.

Gladstone, Kaslow, Seeley and Lewinson (1997) examined attributional style, sex and depressive symptoms and diagnosis in high school students. The results revealed that (1) for females and males, higher levels of depressive symptoms correlated with a more depressive attributional style; (2) females and males who met diagnostic criteria for a current depressive disorder evidenced more depressogenic attributions than psychiatric controls, and never and past depressed adolescents; (3) although no sex difference in terms of attribution pattern for positive events, negative events, or for positive and negative events combined emerged, sex differences were revealed on a number of dimensional scores; (4) across the Children’s Attribution Style Questionnaire (CASQ) subscale and dimensional scores, the relation between attribution and current self reported depressive symptoms was stronger for females than males; and (5) no sex X Diagnostic Group status interaction effects emerged for CASQ subscale or dimensional scores.

Poropat (2002) suggested that attributonal style is one of the cognitive-affective system and has been shown to be related to a number of different patterns for men and women. His study examined the relationship between attributional style as assessed by the Attributional Style Questionnaire, gender, and the FFM (five factor model) Mini-Markers, using a sample of students (aged 17-53 years). The patterns of correlations between
attributional Style Questionnaire and FFM dimensions appeared different for men and women, and 3 significant gender interactions were observed using multiple regression. Both internal attributional style for positive events and overall attributional style interacted with gender to predict openness and hopefulness interacted with gender to predict extraversion.

ANXIETY SENSITIVITY

Accumulated research evidences suggest that anxiety sensitivity is a risk factor for anxiety pathology and plays a prominent role in the maintenance and genesis of the anxiety disorder in general and panic disorder in particular (for example, Barlow, 1991; McNally, 1990; Reiss, 1991). According to this theorizing, the elevated anxiety and/or physiological arousal that all of us are prone to during stressful times become stimuli capable of triggering a vicious cycle of ever-heightening anxiety and even panic for people high in anxiety sensitivity (Zinbarg et al., 2001). Jensen’s study (as cited in Joiner et al., 2002) illustrated that children who scored significantly higher on the Childhood Anxiety Sensitivity Index are more often diagnosed as having anxiety disorders.

In a four year longitudinal investigation using high school students, Weems, Hayward, Killen and Taylor (2002) found high anxiety sensitivity group reporting the experience of panic attacks as compared to low anxiety sensitivity group. Comparable findings were obtained by Lau, Calamari and Waraczynski (1996). They observed significant correlations between the
scores of Panic Attack Questionnaire and Childhood Anxiety Sensitivity Index.

The role of anxiety sensitivity in pain disorders is also well documented. Asmundson and Norton (1995) investigated pain reports in a group of chronic back pain patients (N=70, age range = 17-58 years) that were classified as high, medium, or low in anxiety sensitivity. Although no difference were found in the intensity of experienced pain, those high in anxiety sensitivity reported greater fear of negative consequences of pain, and also greater cognitive disruption and anxiety in response to pain than low-anxious individuals. This suggests that those high in anxiety sensitivity tend to report a greater fear of pain sensations, irrespective of reported pain intensity.

Keogh and Birkby (1999) reported that anxiety sensitivity may play a role in mediating negative experience and sensations, associated with pain. Measure of pain threshold and tolerance were taken, as were self-reported measure of affective and sensory experiences. Because differences between males and females have been found with both anxiety sensitivity and pain experience, gender differences were also investigated. As expected, gender was found to mediate the association of anxiety sensitivity and sensory pain. While, high anxiety sensitive females reported greater pain than low anxiety sensitive females, no effect of anxiety sensitivity on sensory pain was found among males.
Keogh and Cochrane (2002) conducted a study to determine the mechanism by which the relationship between anxiety sensitivity and pain experience exists. Selective attentional and interpretative biases for negative material were compared as potential mediator of the anxiety sensitivity-pain relationship. With the cold pressure task, the study found that high anxiety sensitive participants exhibited a greater interpretative bias and reported more negative pain experiences than those low in anxiety sensitivity. A negative interpretative bias was also related to higher affective pain experiences. However, most important was the tendency to interpret innocuous bodily sensations related to pain that mediated the association between anxiety sensitivity and affective pain experiences. These findings not only confirm that anxiety sensitivity plays an important role in the perception of experimental pain but also identify a potential cognitive mechanism by which the relationship exists.

Keogh, Hamid, Hamid and Ellery (2004) investigated the effect of anxiety sensitivity, gender, and negative interpreting bias on the perception of chest pain and suggested that anxiety sensitivity may be an important component in the negative response to pain sensations, especially those with cardiopulmonary origin. Furthermore, they suggested that such effects may be stronger in women than men. The primary aim of this investigation was to determine the relative roles that anxiety sensitivity and gender have in the pain reports of patients referred to a hospital clinic with chest pain. A total of 78 females and 76 male adults were recruited on entry to a Rapid Access
Medical Clinic. All patients had been referred with chest pain; and were administered a range of pain and anxiety measures prior to diagnosis. This investigation confirmed that both anxiety sensitivity and gender were important factors in the experience of pain. This study not only provides an explanation of how anxiety sensitivity is related to pain, but also for whom. Negative interpretative biases were found to mediate the association between anxiety sensitivity and pain in women, but not men, indicated the existence of a gender-specific cognitive mechanism, which may be an important determinant of pain experience. These results not only confirm that anxiety sensitivity is related to greater negative pain responses in women, but that this may be due to increased tendency to negatively interpret sensations.

Interestingly, anxiety sensitivity is not only related with pain experience but also associated with pain-related coping behaviours. Asmundson and Taylor (1996) investigated the role of anxiety sensitivity in pain fear and avoidance in a group of 259 patients with chronic back pain. Through structural equation modeling, they not only found that anxiety sensitivity significantly contributed to the fear of pain, but that it might in turn lead to increased pain avoidance behaviours. In other words, anxiety sensitivity exacerbated the fear of pain, which in turn was found to lead to avoidance behaviours, that is, negative coping strategy. This suggests that anxiety sensitivity may mediate both the perception of pain and the way in which pair is dealt with.
Norman and Lang (2005) explored the role of anxiety sensitivity on functioning in the chronically physically ill. Participants were 267 primary care patients. Logistic regression showed that physical anxiety sensitivity (but not social or psychological), controlling for age, gender, and negative affect, was associated with hypertension, been disease, and high cholesterol ($p < .01$). Higher anxiety sensitivity was associated with poor vitality, mental functioning, and social functioning ($p < .05$). They also predicted that anxiety sensitivity may be correlate of poorer adjustment to chronic illness.

Cognitive variables as assessed by Anxiety Sensitivity Index (ASI) and Agoraphobic Cognition Questionnaire (ACQ) were significantly related to illness-specific panic fear (that is, panic fear in response to symptoms of asthma) among asthmatics. In a study conducted in 1995 on 86 asthmatics Carr, Lehrer and Hochron concluded that cognitive variables predicted significant variances in both panic-fear scales (illness-specific and generalized panic fear) after controlling for the effects of demographic and asthma variables. By contrast, the asthma variables were not associated with generalized panic-fear when the cognitive measures were controlled.

In an earlier study, Carr, Lehrer, Rausch and Hochron (1994) proved the relationship among anxiety sensitivity, frequent spontaneous panic attacks and pulmonary function is 93 asthmatic adults. 22.6% of the asthmatics reported a history of spontaneous panic attacks with 9.7% reporting attacks that were severe and frequent enough to meet the DSM-III-R criteria for panic disorder. Anxiety sensitivity but not pulmonary function was significantly
related to panic disorder. In this study, asthmatics (with or without panic disorder) were compared with 10 panic disorder patients without asthma and with 32 nonanxious, non asthmatic controls on the Anxiety Sensitivity Index (ASI), the Bodily Sensation Questionnaire, and Agoraphobic Cognitions Questionnaire. Whereas subjects with panic disorder (asthmatic and nonasthmatic) displayed significant elevations on these measures, the presence of asthma alone had no effect.

In a study Delvaux, Fontaine and Bartsch (1999), with 66 subjects, showed that anxiety sensitivity correctly discriminates the subjects with panic disorder, who hyperventilate after a stress (mental imagery), subjects who hyperventilate but have no panic disorder, and healthy control subjects. The results were compared with those of a study including asthmatics who experienced hyperventilation before or during their bronchoconstriction. The results showed that asthma didn’t arise anxiety sensitivity compared to subjects with hyperventilation only. Subjects prone to hyperventilation who present a panic disorder showed the highest level of anxiety sensitivity.

Perhaps more remarkable, however, is that females report high levels of anxiety sensitivity than males (for example, Peterson and Phehm, 1999; Peterson and Reiss, 1992, Stewart, Taylor and Baker, 1997). Nevertheless, sex differences on the levels of anxiety sensitivity have also been observed with respect to the reporting of the experience of pain. Anxiety sensitivity was related to pain in women but not men (for example, Keogh and Birkby, 1999; Keogh, Hamid, Hamid and Ellergy, 2004).
The above review reveals that though attributional style and anxiety sensitivity have been explored in relation to various disorders, no effort yet has been made to find the role of these variables among asthmatics. Asthma is an intermittent, unpredictable and uncontrollable disease. There are numerous body sensations (for example, wheezing, shortness of breath etc.) that asthmatics must negotiate, including the variable functional limitations and decreased ability to engage in normal daily activities. The feelings of helplessness and lack of control over the situations, which may ultimately alter the availability of certain cognitive and physiological resources, predict increased levels of anxiety and depression in this population. Furthermore, due to increased unpredictability of disease exacerbations, individuals with asthma must attend vigilantly to internal cues (for example, tightness in one’s chest), which may lead to increased anxious behaviour related to their illness (Carr, 1999; Celano and Geller, 1993). Though biological susceptibility is at the root cause of asthma, but precipitation and exacerbation of asthmatic symptoms have links to psychological factors. On the one hand, asthma has been found to be linked to stress, personality factors and pathogenic family interactions, and on the other hand, it has comorbidity with depression, negative affectivity, anxiety and other anxiety disorders particularly agoraphobia and panic attacks. Also not all biologically susceptible individuals develop asthma. It is therefore pertinent to explore the psychological factors related to asthma.
Chapter – III

Methodology
Before undertaking any research it is important that the researcher examine his/her problem, aims and objectives, so that it can be appropriately planned as to how these objectives can best be achieved. Taking into consideration the requirement of a scientific study, the present research has also been planned.

The purpose and objective of present study was to investigate the attributional style and anxiety sensitivity among asthmatic patients. The main concern of present research was to find out whether asthmatics and non-asthmatics have different attributional style and different levels of anxiety sensitivity or not. It was hypothesized that the attributional style and the level of anxiety sensitivity of asthmatics would differ from those of the non-asthmatic healthy normals. Further, these differences will be studied in the context of gender, age and tenure of disease. These comparisons will explain the phenomenon in a more intensive and exhaustive manner. Appropriate research hypotheses have been formed and are given in Chapter-I.

Sample

Participants of the present study comprised of 150 subjects: 75 asthmatics and 75 normal counterparts (non-asthmatic controls). The age of the participants ranged between 12 to 50 years. Asthmatic patients were undergoing investigations and treatments in Out Patient Department (OPD) of Jawaharlal Nehru Medical College of Aligarh Muslim University, Aligarh and other clinics in Aligarh city. Participation in the study was purely voluntary.
Asthmatic patients (who were diagnosed by specialists as asthmatics) were selected on the basis of purposive sampling (that is, judgemental sampling) technique, which is based on typicality of the cases to be included in the sample.

Today, purposive sampling remains the primary method for selecting large, representative samples for social science and business researches. In this sampling method the researcher purposively chooses persons who, in his/her judgement about some appropriate characteristic required for the sample members, are thought to be relevant to the research topic and are easily available to him/her.

Ideally, random sample procedure which is totally free from bias and permit each and every element of the population an equal chance of being part of sample should be followed. Randomization is necessary to ensure validity of independence assumptions but practical difficulties do not allow pure random sample. However, it is imperative that the element of bias should be controlled. This was kept in mind by the present researcher.

The purposive selection of the experimental group of asthmatic patients did not restrict the choice in the matter of their sex, educational level or socioeconomic status except age (that is, in the range of 12 to 50 years). A group of normal subjects was selected to serve as a control group. These normal subjects (both male and female) were of the same age group and matched with asthmatic subjects for such variables as educational level and
socioeconomic background. None of them reported any serious medical complication.

Table: Showing categorization of the sample and number of subjects in each group.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Total</th>
<th>Ages (in years)</th>
<th>Duration of Disease (in years)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>12-20</td>
<td>20-35</td>
</tr>
<tr>
<td>Asthmatics N=75</td>
<td>Male</td>
<td>38</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>37</td>
<td>10</td>
</tr>
<tr>
<td>Non-Asthmatics N=75</td>
<td>Male</td>
<td>38</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>37</td>
<td>10</td>
</tr>
</tbody>
</table>

Tools of Study

The following measures were employed for collecting information regarding the subject's attributional style and anxiety sensitivity:

Attributional Style Questionnaire (ASQ): The attributional style of the subject was measured with the help of Attributional Style Questionnaire (ASQ) developed by Peterson, Semmel, von Baeyer, Abramson, Metalsky and Seligman in 1982 and revised by Peterson and Seligman in 1984.

ASQ is most widely known and used dimensional measure of attributional style. This measure was developed to test predictions from the reformulated theory of learned helplessness/depression (Abramson, Seligman and Teasdale, 1978), which holds that attributing uncontrollable bad events to internal, stable, and global factors leads to depression. The ASQ has proven to
be a valid measure of attributional style and it assesses habitual tendencies in the attribution of causes (Peterson et al., 1982).

This 12 item measure contains 6 items assessing the causal dimensions related to the interpersonal/affiliative events and 6 items to the achievement related events. In addition to this, out of 12 hypothetical events, half describes positive events (‘you become very rich’) and other half describes negative events (‘you can’t get all the work done that others expect of you’).

It was observed that 2 out of 12 items of ASQ were highly uncomfortable and irrelevant for the Indian surroundings because they are not part of experience of Indian population. Therefore, the phrase ‘spouse (boy friend/girl friend)’ has been changed by the word ‘friend’ in the statement ‘your spouse (boy friend/girl friend) has been treating you more lovingly’. In other statement ‘you out on a date and it goes badly’, the term ‘date’ has been substituted by ‘tour’. This practice was already carried out by Siddiq (1997) during her M.Phil. work, through pilot study.

The printed instructions of ASQ are self explanatory. Each item presents the individual with a statement to imagine an event and then requires the respondent to generate its one major cause. On the following 3 questions, that are always in the same order, subjects have to rate each cause along a 7-point bipolar scale (for instance, ‘totally due to other people or circumstances’
Peterson et al. (1982) suggested that the three attributional dimension rating scales associated with each event description are scored in the directions of increasing internality, stability, and globality. Composite scores are created simply by summing the appropriate item scores and dividing the sum by the number of items in the composite. Scores are derived by simply averaging within dimension and across events for individual dimension scores or across dimensions and across events for composite scores. Each individual dimension ranges from 1 to 7. Therefore, composite scores (composite positive and composite negative) range from 3 to 21. High score on any dimension of attributional style denotes internality, stability and globality and, on the other hand, low score on any attributional style dimension shows externality, unstability and specificity.

ASQ promises to be a reliable and valid instrument. It assumes a modest degree of cross-situational consistency in the type of attribution people make. Peterson et al. (1982) observed that the three scales, that is, locus, stability and globality have modest reliability with Cronbach’s alpha ranging from .44 to .69 (mean reliability of .54). Peterson and Seligman (1984) found Cronback’s alpha coefficient of revised ASQ range from .66 to .88.

A number of studies have explored the criterion and construct validity of ASQ. Peterson et al. (1982) followed correlational approach and
devised several methods of demonstrating the criterion validity of ASQ. The results of study conducted by Peterson, Bettes and Seligman (1982) demonstrated the construct validity for the ASQ in that it both taps spontaneously generated attributions and relate to theoretically relevant symptomatology. Other studies conducted by Zullow and Seligman (1985), Kamen and Seligman (1985) and Seligman and Shulman (1986) have further supported the construct validity of ASQ.

**Anxiety Sensitivity Index (ASI)**: (Age limit: 12 & more; time limit: 2-3 minutes) The Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky and McNally, 1986) is an extensively developed, established, most widely used, tested instrument to measure the fear of autonomic arousal and shown to have good psychometric properties (Peterson and Reiss, 1992). ASI has popularity and utility in researches and clinical practices throughout the world and can be employed for the following purposes:

- To determine the level of fear of anxiety sensations.
- To assess Panic Disorder and Post Traumatic Stress Disorder.
- To assess dual diagnosis (substance abuse and anxiety disorder) in psychopathological researches.
- It prospectively predicts relapse vs durability of improvement.

ASI has been translated into 24 languages and more than 450 articles have used it (Anxiety Sensitivity Index, 2005).
ASI is a 16-item self-report questionnaire that taps content related to fears, worries, and concerns about not only anxiety but also a person’s fear of anxiety related somatic sensations. This questionnaire asks people to rate their fear of such symptoms as rapid heart beat, shortness of breath, nervousness and even stomach growling.

ASI broadly measures—(1) psychological concerns, that is fear of mental incapacitation concerns (called “phrenophobia” by Taylor et al., 1996); (2) social concerns that is, fear of publically observable anxiety reactions; (3) physical concerns, that is, (a) fear of cardiopulmonary sensation, (b) fear of respiratory sensations, (c) fear of gastro-intestinal sensations. A number of factor analytic studies and critical review of literature suggest that anxiety sensitivity has three lower-order factors that all load on a single higher-order (Zinbarg, Mohlman and Hong, 1999). The lower-order factors represent Physical Concerns, Mental Incapacitation Concerns, and Social Concerns, and the higher order factor represents the global anxiety sensitivity construct (Zinbarg, Barlow, and Brown, 1997; Zinbarg et al., 1999; Rodriguez et al., 2004; Dehon et al., 2005).

The instructions of ASI are brief, clear and self-explanatory, which are printed at the top of the questionnaire—“Circle the one phrase the best represents the extent to which you agree with the item. If any of the items concerns something that is not part of your experience (for example, “It scares me when I feel shaky” for someone who has never trampled or had the ‘shakes’), answer on the basis of how you think you might feel if you had
such an experience. Otherwise, answer all items on the basis of your own experience”.

All the 16 items are presented with five phrase (5 point Likert type) answer format ranging from 0 (very little) through 4 (very much) used for record the responses. The subject choose the one phrase that best represent how much they generally agree with statements such as “It scares me when I feel faint” or “It scares me when I become short of breath”. Once the test is completed, scoring involves a highly simple system for which each item is scored on a 0 to 4 point scale: very little (scored as 0), a little (1), some (2), much (3), and very much (4).

Following the recommendations of recent factor analytic studies (for example, Zinbarg et al., 1997; Zvolensky and Forsyth, 2002), the three subscales involving ‘Physical Concerns’ (Items 3, 4, 6, 8, 9, 10, 11 and 14), ‘Mental Incapacitation Concerns’ (Item 2, 12, 15 and 16), and ‘Social Concerns’ (Items 1, 5, 7 and 13) were utilized. Although not without controversy, this solution has arguably been found to replicate most consistently across different populations (Zinbarg et al., 1999). However, the total ASI scores (that is, sum of all the points for all 16 items) was also used.

ASI is the most commonly used and most studied measure of anxiety sensitivity. It’s measurement issues, that is, norms, reliability and validity are also well studied (Peterson and Phehm, 1999).
The official norms compiled by Peterson and Reiss in 1991 (as cited in Anxiety Sensitivity Index, 2005) are based on studies that assessed 5459 nonclinical subjects and 1821 clinical (diagnosed) subjects. But, these norms are still valid today. The psychometric properties of ASI are well established. It has satisfactory degree of internal consistency and test-retest reliability. The internal consistency of the ASI is good with Chronbach’s alpha ranged from .82 to .91 (Peterson and Phahm, 1999). Previously, Peterson and Heilbronner (1987) have obtained the alpha coefficient of .88 and the Guttman split half reliability of .85, for a sample of 119 college students who had identified themselves as being anxious. Alpha coefficient for this scale was reported .82 for a sample of 840 college students (Telch, Shermis and Lucas, 1989); .87 for combined sample of 275 college students and 52 patients with panic disorder or agoraphobia (Cox, Endler, Swinson and Norton, 1991); and .91 for 93 psychiatric out patients and .84 for 142 spider phobic college students (Taylor, Koch and Crockett, 1991).

The ASI is a highly reliable measure. Reiss et al. (1986) calculated two week test-retest reliability of 0.75 for a sample of 127 college students. Further, the test-retest reliability has been reported to be .71 for college students over a 3-year period (Maller and Reiss, 1992; Peterson and Phelm, 1999). The reliabilities of the lower order scale were .86 for physical concerns; .83 for mental incapacitation concerns; and .65 for social concerns (Zinbarg and Barlow, 1996; Zvolensky and Forsyth, 2002).
Both factor validity and validity by criterion group comparison was determined certain group comparisons. A number of students have found that ASI scores are associated with diagnostic conditions in accordance with theoretical expectations (Reiss and McNally, 1985; Reiss, 1991). The criterion validity of ASI is exceptional (McNally and Lorenz, 1987; Reiss et al. 1986; Cox, Endler and Swinson, 1991; Rapee, Brown, Antony and Barlow, 1992; Taylor, Koch and McNally, 1992 etc.).

**Procedure**

The researcher first of all visited the out patient department for T.B. and Chest diseases, of J.N. Medical College, A.M.U. and other clinics in Aligarh. The data were collected individually in a separate room provided by the department for this purpose. Patients diagnosed with asthma were referred by the doctor for psychological investigation.

Normal control group consisted mostly of the relatives of the asthmatics who were free of the problem (any serious medical complication).

Subjects were presented with a brief description about the objective of the study. They were instructed adequately along with the assurance of confidentiality. The researcher established a harmonious relationship with the subjects. The subjects who were reluctant to participate were not tested and allowed to go. Following their agreement to participate, both the questionnaires (ASQ and ASI) were administered individually to the participants of the study. The researcher helped those subjects who faced
difficulty is understanding some of the items of the questionnaire because many subjects were either less educated or illiterate.

After data collection scoring was done and the data were processed/reduced which mainly involves various processes necessary for preparing the data for analysis (for instance - checking, editing, categorizing etc.).

**Statistical Analysis**

Means, S.D.s and t-values (two tailed probability) were computed to analyse the data for finding out the significance of differences on eight dimensions of attributional style, and anxiety sensitivity among the various groups.
Chapter – IV

Results
Table 1. Showing Attributional Style scores of Asthmatics and Non-asthmatics

<table>
<thead>
<tr>
<th>Attributional Style Dimensions</th>
<th>Asthmatics (N=75)</th>
<th>Non-asthmatics (N=75)</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>For Positive Events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>14.61</td>
<td>1.64</td>
<td>15.25</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.88</td>
<td>0.76</td>
<td>5.28</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.99</td>
<td>0.71</td>
<td>5.16</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.73</td>
<td>0.42</td>
<td>4.81</td>
</tr>
<tr>
<td>For Negative Events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>13.43</td>
<td>2.05</td>
<td>12.75</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.33</td>
<td>0.99</td>
<td>4.24</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.60</td>
<td>0.88</td>
<td>4.41</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.47</td>
<td>0.85</td>
<td>4.01</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; **significant at 0.01 level

In table 1, it is displayed that there is significant difference between asthmatics and non-asthmatics on 4 out of 8 dimensions of attributional style. Non-asthmatics score high on composite for positive events while asthmatics score high on composite for negative events. Asthmatics as compared to normal counterparts are more external on positive dimension and more global on negative dimensions of attributional style.

Therefore, hypothesis 1 which postulates that asthmatics depict an attributional style different from non-asthmatics is supported by our result(s).
Figure 1.1. Composite scores of Asthmatics and Non-asthmatics for Positive and Negative Events on Attributional Style Questionnaire (ASQ)

Figure 1.2. Scores of Asthmatics and Non-asthmatics on different dimensions for Positive Events on Attributional Style Questionnaire (ASQ)

Figure 1.3. Scores of Asthmatics and Non-asthmatics on different dimensions for Negative Events on Attributional Style Questionnaire (ASQ)
Table 2. Showing Attributional Style scores of Asthmatic and Non-asthmatic Males

<table>
<thead>
<tr>
<th>Attributional Style Dimensions</th>
<th>Asthmatic Males (N=38) year</th>
<th>Non-asthmatic Males (N=38)</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>For Positive Events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>15.51</td>
<td>1.27</td>
<td>15.46</td>
</tr>
<tr>
<td>Internal-External</td>
<td>5.19</td>
<td>0.74</td>
<td>5.30</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>5.29</td>
<td>0.56</td>
<td>5.22</td>
</tr>
<tr>
<td>Global- Specific</td>
<td>5.03</td>
<td>0.73</td>
<td>5.14</td>
</tr>
<tr>
<td>For Negative Events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>12.73</td>
<td>1.97</td>
<td>12.54</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.09</td>
<td>1.07</td>
<td>4.25</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.38</td>
<td>0.88</td>
<td>4.31</td>
</tr>
<tr>
<td>Global- Specific</td>
<td>4.27</td>
<td>0.92</td>
<td>3.86</td>
</tr>
</tbody>
</table>

*S Significant at 0.05 level; **significant at 0.01 level; ***significant at 0.001 level

Table 9 indicates that asthmatic males and normal males do not differ significantly in their attributional style, on both positive and negative dimensions.

Thus, hypothesis 1.1 stands rejected.
Figure 2.1. Composite scores of Asthmatic and Non-asthmatic Males for Positive and Negative Events on Attributional Style Questionnaire (ASQ)

Figure 2.2. Scores of Asthmatic and Non-asthmatic Males on different dimensions for Positive Events on Attributional Style Questionnaire (ASQ)

Figure 2.3. Scores of Asthmatic and Non-asthmatic Males on different dimensions for Negative Events on Attributional Style Questionnaire (ASQ)
Table 3. Showing Attributional Style scores of Asthmatic and Non-asthmatic Females

<table>
<thead>
<tr>
<th>Attributional Style Dimensions</th>
<th>Asthmatic Females (N=37)</th>
<th>Non-asthmatic Females (N=37)</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>For Positive Events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>13.71</td>
<td>2.02</td>
<td>15.04</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.57</td>
<td>0.79</td>
<td>5.26</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.70</td>
<td>0.86</td>
<td>5.14</td>
</tr>
<tr>
<td>Global- Specific</td>
<td>4.43</td>
<td>0.85</td>
<td>4.67</td>
</tr>
<tr>
<td>For Negative Events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>14.14</td>
<td>2.14</td>
<td>12.91</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.58</td>
<td>0.91</td>
<td>4.24</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.83</td>
<td>0.89</td>
<td>4.51</td>
</tr>
<tr>
<td>Global- Specific</td>
<td>4.78</td>
<td>0.79</td>
<td>4.71</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; **significant at 0.01 level; ***significant at 0.001 level

Significant difference is observed between asthmatic and non-asthmatic females on 4 out of 8 dimensions of attributional style. They differ on composite, internal-external and stable-unstable dimensions for positive events and on composite for negative events. Asthmatic females as compared to non-asthmatic females scored significantly higher on composite scale for positive events and attributed these events to external and unstable causes. For negative events asthmatic females scored significantly higher on composite scale only.

Thus, hypothesis 1.2 is borne out by our result.
Figure 3.1. Composite scores of Asthmatic and Non-asthmatic Females for Positive and Negative Events on Attributional Style Questionnaire (ASQ)

Figure 3.2. Scores of Asthmatic and Non-asthmatic Females on different dimensions for Positive Events on Attributional Style Questionnaire (ASQ)

Figure 3.3. Scores of Asthmatic and Non-asthmatic Females on different dimensions for Negative Events on Attributional Style Questionnaire (ASQ)
Table 4. Showing Attributional Style scores of Asthmatic with illness duration of 1 year or less and with illness duration of 1 to 5 year

<table>
<thead>
<tr>
<th>Attributional Style Dimensions</th>
<th>Asthmatics (N=30)</th>
<th></th>
<th>Asthmatics (N=21)</th>
<th></th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
<td>S.D.</td>
<td></td>
</tr>
<tr>
<td>For Positive Events</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>14.67</td>
<td>1.23</td>
<td>14.64</td>
<td>2.11</td>
<td>0.06</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.60</td>
<td>0.51</td>
<td>5.01</td>
<td>0.82</td>
<td>2.05*</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>5.16</td>
<td>0.46</td>
<td>4.96</td>
<td>1.08</td>
<td>0.80</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.90</td>
<td>0.76</td>
<td>4.75</td>
<td>0.89</td>
<td>0.62</td>
</tr>
<tr>
<td>For Negative Events</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>12.62</td>
<td>1.46</td>
<td>14.14</td>
<td>2.27</td>
<td>2.71**</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.28</td>
<td>1.03</td>
<td>4.42</td>
<td>0.81</td>
<td>0.54</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.38</td>
<td>0.67</td>
<td>4.46</td>
<td>1.03</td>
<td>0.31</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>3.96</td>
<td>0.74</td>
<td>5.16</td>
<td>1.20</td>
<td>4.14***</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; **significant at 0.01 level; ***significant at 0.001 level

Table 2 indicates that there is significant difference between asthmatics with illness duration of 1 year or less and those with illness duration of 1 to 5 year on 3 out of 8 dimensions of attributional style. They differ on internal-external dimension for positive events and, on composite and global-specific dimensions for negative events. Asthmatics with asthma duration of 1 year or less are more external on positive dimension and are more specific on negative dimension of attributional style. While patients with 1 to 5 years illness duration made internal attributions for positive events and global attributions for negative events.

Hence, hypothesis 2.1 is confirmed by our result.
Table 5. Showing Attributional Style scores of Asthmatic with illness duration of 1 year or less and with illness duration of more than 5 year

<table>
<thead>
<tr>
<th>Attributional Style Dimensions</th>
<th>Asthmatics (N=30) Duration of Disease: 1 year or less</th>
<th>Asthmatics (N=24) Duration of Disease: more than 5 year</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>For Positive Events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>14.67</td>
<td>1.23</td>
<td>14.65</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.60</td>
<td>0.51</td>
<td>5.36</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>5.16</td>
<td>0.49</td>
<td>4.80</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.90</td>
<td>0.76</td>
<td>4.58</td>
</tr>
<tr>
<td>For Negative Events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>12.62</td>
<td>1.46</td>
<td>13.50</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.28</td>
<td>1.03</td>
<td>4.14</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.38</td>
<td>0.67</td>
<td>4.99</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>3.96</td>
<td>0.74</td>
<td>4.52</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; **significant at 0.01 level

Table 3 shows significant difference between asthmatics with illness duration of more than 5 years and those with illness duration of 1 year or less on 5 out of 8 dimensions of attributional style. These are internal-external and stable-unstable for positive events and composite, stable-unstable and global-specific for negative events. Asthmatics with illness duration of more than 5 year are more internal and unstable on positive dimensions and are more stable and global on negative dimensions of attributional style.

Therefore, hypothesis 2.2 is retained.
Table 6. Showing Attributional Style scores of Asthmatic with illness duration of 1 to 5 year and with illness duration of more than 5 year

<table>
<thead>
<tr>
<th>Attributional Style Dimensions</th>
<th>Asthmatics (N=21) Duration of Disease: 1 to 5 year</th>
<th>Asthmatics (N=24) Duration of Disease: more than 5 year</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>For Positive Events</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>Mean: 14.68  S.D.: 2.11</td>
<td>Mean: 14.65  S.D.: 1.39</td>
<td>0.06</td>
</tr>
<tr>
<td>Internal-External</td>
<td>Mean: 5.01  S.D.: 0.82</td>
<td>Mean: 5.36  S.D.: 0.97</td>
<td>1.29</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>Mean: 4.96  S.D.: 1.08</td>
<td>Mean: 4.80  S.D.: 0.51</td>
<td>0.66</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>Mean: 4.75  S.D.: 0.89</td>
<td>Mean: 4.58  S.D.: 0.69</td>
<td>0.71</td>
</tr>
<tr>
<td><strong>For Negative Events</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>Mean: 14.14  S.D.: 2.27</td>
<td>Mean: 13.50  S.D.: 1.62</td>
<td>1.10</td>
</tr>
<tr>
<td>Internal-External</td>
<td>Mean: 4.42  S.D.: 0.81</td>
<td>Mean: 4.14  S.D.: 1.11</td>
<td>0.96</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>Mean: 4.46  S.D.: 1.03</td>
<td>Mean: 4.99  S.D.: 1.02</td>
<td>1.71</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>Mean: 5.16  S.D.: 1.20</td>
<td>Mean: 4.52  S.D.: 0.91</td>
<td>2.02*</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level

From table 4, it is observed that asthmatics with illness duration of 1 to 5 year differ significantly from asthmatics with illness duration of more than 5 year on only global-specific dimensions for negative events. Asthmatics with illness duration of 1 to 5 year as compared to more than 5 year duration are more global on negative dimension of attributional style.

Therefore, hypothesis 2.3 is partly accepted.

Since, it may be seen from the tables 4, 5 and 6 that asthmatics with different duration of illness differ in their attributional style. Hence, hypothesis 2 is supported by our results.
Asthmatics (DoD*=1 year or less)
Asthmatics (DoD*= 1 to 5 year)
Asthmatics (DoD*= more than 5 year)

Figure 4.1. Composite scores of different illness duration of Asthmatics for Positive and Negative Events on Attributional Style Questionnaire (ASQ)

Asthmatics (DoD*=1 year or less)
Asthmatics (DoD*= 1 to 5 year)
Asthmatics (DoD*= more than 5 year)

Figure 4.2. Scores of different illness duration groups of Asthmatics on different dimensions for Positive Events on Attributional Style Questionnaire (ASQ)

Asthmatics (DoD*=1 year or less)
Asthmatics (DoD*= 1 to 5 year)
Asthmatics (DoD*= more than 5 year)

Figure 4.3. Scores of different illness duration groups of Asthmatics on different dimensions for Negative Events on Attributional Style Questionnaire (ASQ)

*DoD = Duration of disease
Table 7. Showing Attributional Style scores of Asthmatics of age 12 to 20 years and of ages 20 to 35 years

<table>
<thead>
<tr>
<th>Attributional Style Dimensions</th>
<th>Asthmatics (N=20) Ages: 12 to 20 years</th>
<th>Asthmatics (N=32) Ages: 20 to 35 years</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td><strong>For Positive Events</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>14.74</td>
<td>1.87</td>
<td>15.29</td>
</tr>
<tr>
<td>Internal-External</td>
<td>5.04</td>
<td>0.98</td>
<td>5.13</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.96</td>
<td>0.66</td>
<td>5.39</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.75</td>
<td>0.84</td>
<td>4.51</td>
</tr>
<tr>
<td><strong>For Negative Events</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>12.77</td>
<td>2.05</td>
<td>13.23</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.34</td>
<td>0.67</td>
<td>4.35</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.06</td>
<td>0.98</td>
<td>4.53</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.36</td>
<td>1.06</td>
<td>4.35</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level

From table 5, it is observed that there is no significant difference between the asthmatics of ages 12 to 20 years and of ages 20 to 35 years except on stable-unstable dimension for positive events, on which asthmatics of ages 12 to 20 years are more unstable.

Thus hypothesis 3.1 is partly supported by our findings.
Table 8. Showing Attributional Style scores of Asthmatic of age 12 to 20 years and of ages 35 to 50 years

<table>
<thead>
<tr>
<th>Attributional Style Dimensions</th>
<th>For Positive Events</th>
<th>For Negative Events</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Asthmatics (N=20)</td>
<td>Asthmatics (N=23)</td>
</tr>
<tr>
<td></td>
<td>Ages: 12 to 20 years</td>
<td>Ages: 35 to 50 years</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
</tr>
<tr>
<td>Composite</td>
<td>14.74</td>
<td>1.87</td>
</tr>
<tr>
<td>Internal-External</td>
<td>5.04</td>
<td>0.98</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.96</td>
<td>0.66</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.74</td>
<td>0.84</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>12.77</td>
<td>2.06</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.34</td>
<td>0.67</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.06</td>
<td>0.98</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.36</td>
<td>1.06</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; ***Significant at 0.001 level

From table 6, it is depicted that asthmatics of ages 12 to 20 years and of ages 35 to 50 years differ significantly on attributional style for negative events. Asthmatics of ages 35 to 50 years as compared to the other group scored higher on both composite and stable-unstable dimensions of negative events.

Therefore, hypothesis 3.2 stands accepted.
Table 9. Showing Attributional Style scores of Asthmatics of ages 20 to 35 years and of ages 35 to 50 years

<table>
<thead>
<tr>
<th>Attributional Style Dimensions</th>
<th>Asthmatics (N=32) Ages: 20 to 35 years</th>
<th>Asthmatics (N=23) Ages: 35 to 50 years</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td><strong>For Positive Events</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>15.29</td>
<td>1.62</td>
<td>14.08</td>
</tr>
<tr>
<td>Internal-External</td>
<td>5.13</td>
<td>0.63</td>
<td>4.66</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>5.39</td>
<td>0.51</td>
<td>4.64</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.51</td>
<td>0.80</td>
<td>4.93</td>
</tr>
<tr>
<td><strong>For Negative Events</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>13.23</td>
<td>2.28</td>
<td>14.32</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.35</td>
<td>0.87</td>
<td>4.31</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.53</td>
<td>0.81</td>
<td>5.32</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.35</td>
<td>0.71</td>
<td>4.71</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; **Significant at 0.01 level

Table 7 indicates that asthmatics of ages 20 to 35 year differ significantly from asthmatics of ages 35 to 50 years on 5 out of 8 dimensions of attributional style. These are composite, internal-external, and stable-unstable dimensions for positive events and composite and stable-unstable dimension for negative events. Asthmatics of ages 20 to 35 years as compared to 35 to 50 years age group are more internal and stable on positive dimensions, but are unstable on negative dimension of attributional style.

Therefore, hypothesis 3.3 is confirmed.

On the basis of results displayed in table 7, 8 and 9, it is clear that hypothesis 3 is retained.
Figure 5.1. Composite scores of different age groups of Asthmatics for Positive and Negative Events on Attributional Style Questionnaire (ASQ)

Figure 5.2. Scores of different age groups of Asthmatics on different dimensions for Positive Events on Attributional Style Questionnaire (ASQ)

Figure 5.3. Scores of different age groups of Asthmatics on different dimensions for Negative Events on Attributional Style Questionnaire (ASQ)
### Table 10. Showing Attributional Style scores of Asthmatic males and Asthmatic females

<table>
<thead>
<tr>
<th>Attributional Style Dimensions</th>
<th>Asthmatic Males (N=38)</th>
<th>Asthmatic Females (N=37)</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td><strong>For Positive Events</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>15.51</td>
<td>1.27</td>
<td>13.71</td>
</tr>
<tr>
<td>Internal-External</td>
<td>5.19</td>
<td>0.74</td>
<td>4.57</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>5.29</td>
<td>0.56</td>
<td>4.70</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>5.03</td>
<td>0.73</td>
<td>4.43</td>
</tr>
<tr>
<td><strong>For Negative Events</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>12.73</td>
<td>1.97</td>
<td>14.14</td>
</tr>
<tr>
<td>Internal-External</td>
<td>4.09</td>
<td>1.07</td>
<td>4.58</td>
</tr>
<tr>
<td>Stable-Unstable</td>
<td>4.38</td>
<td>0.88</td>
<td>4.83</td>
</tr>
<tr>
<td>Global-Specific</td>
<td>4.27</td>
<td>0.92</td>
<td>4.78</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; **significant at 0.01 level; ***significant at 0.001 level

From Table 8 it is palpable that asthmatic males and asthmatic females differ significantly on 5 out of 8 dimensions of attributional style. The difference is found for composite and internal-external dimensions for positive events. While for negative events the difference was found for composite score, and internal-external and stable-unstable dimensions. For positive events asthmatic females as compared to asthmatic males scored significantly lower on composite score and attributed these events to external causes. Conversely, for negative events asthmatic females scored higher on composite scale and attributed these events to internal and stable causes.

Thus hypothesis 4 is borne out by our results.
Figure 6.1. Composite scores of Asthmatic Males and Asthmatic Females for Positive and Negative Events on Attributional Style Questionnaire (ASQ)

Figure 6.2. Scores of Asthmatic Males and Asthmatic Females on different dimensions for Positive Events on Attributional Style Questionnaire (ASQ)

Figure 6.3. Scores of Asthmatic Males and Asthmatic Females on different dimensions for Negative Events on Attributional Style Questionnaire (ASQ)
Table 11. Showing Anxiety Sensitivity scores of Asthmatics and Non-asthmatics

<table>
<thead>
<tr>
<th>Anxiety Sensitivity Concerns</th>
<th>Asthmatics (N=75)</th>
<th>Non-asthmatics (N=75)</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Total Scores</td>
<td>26.91</td>
<td>5.46</td>
<td>21.15</td>
</tr>
<tr>
<td>Physical concerns</td>
<td>12.18</td>
<td>2.09</td>
<td>11.22</td>
</tr>
<tr>
<td>Mental Incapacitation concerns</td>
<td>7.62</td>
<td>2.33</td>
<td>4.84</td>
</tr>
<tr>
<td>Social concerns</td>
<td>7.08</td>
<td>1.96</td>
<td>5.07</td>
</tr>
</tbody>
</table>

**Significant at 0.05 level; ***significant at 0.001 level

The perusal of table 11 indicates that asthmatics as compared to non-asthmatics score significantly higher on anxiety sensitivity index. Asthmatics endorse significantly higher physical, mental incapacitation and social concerns than those of their normal counterparts.

Therefore hypothesis 5 is confirmed.
Figure 7.1. Total Scores of Asthmatics and Non-asthmatics on Anxiety Sensitivity Index (ASI)

Figure 7.2. Scores of Asthmatics and Non-asthmatics on different concerns on Anxiety Sensitivity Index (ASI)
Table 12. Showing Anxiety Sensitivity scores of Asthmatic and Non-asthmatic Males

<table>
<thead>
<tr>
<th>Anxiety Sensitivity Concerns</th>
<th>Asthmatic Males (N=38)</th>
<th>Normal Males (N=38)</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Total Scores</td>
<td>23.32</td>
<td>3.92</td>
<td>18.28</td>
</tr>
<tr>
<td>Physical concerns</td>
<td>12.52</td>
<td>1.54</td>
<td>11.71</td>
</tr>
<tr>
<td>Mental Incapacitation concerns</td>
<td>5.79</td>
<td>1.80</td>
<td>3.25</td>
</tr>
<tr>
<td>Social concerns</td>
<td>5.01</td>
<td>1.58</td>
<td>3.22</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; ***significant at 0.001 level

It is clear from table 19 that asthmatic males experience elevated degree of anxiety sensitivity in comparison to their male healthy counterparts. Asthmatic males depict significantly higher physical, mental incapacitation and social concerns as compared to their normal counterparts.

Thus hypothesis 5.1 is supported by our results.
Figure 8.1. Total Scores of Asthmatic and Non-asthmatic Males on Anxiety Sensitivity Index (ASI)

Figure 8.2. Scores of Asthmatic and Non-asthmatic Males on different concerns on Anxiety Sensitivity Index (ASI)
Table 13. Showing Anxiety Sensitivity scores of Asthmatic and non-asthmatic Females

<table>
<thead>
<tr>
<th>Anxiety Sensitivity Concerns</th>
<th>Asthmatic Females (N=37)</th>
<th>Normal Females (N=37)</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Total Scores</td>
<td>30.49</td>
<td>7.01</td>
<td>24.07</td>
</tr>
<tr>
<td>Physical concerns</td>
<td>11.84</td>
<td>2.65</td>
<td>10.73</td>
</tr>
<tr>
<td>Mental Incapacitation concerns</td>
<td>9.45</td>
<td>2.86</td>
<td>6.43</td>
</tr>
<tr>
<td>Social concerns</td>
<td>9.18</td>
<td>2.33</td>
<td>6.92</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; ***significant at 0.001 level

In table 20, it is displayed that asthmatic females in comparison to their counterparts endorse significantly higher anxiety sensitivity. Asthmatic females score significantly higher on physical, mental-incapacitation and social concerns as compared to their healthy counterparts.

Therefore, hypothesis 5.2 is confirmed.
Figure 9.1. Total scores of Asthmatic and Non-asthmatic Females on Anxiety Sensitivity Index (ASI)

Figure 9.2. Scores of Asthmatic and Non-asthmatic Females on different concerns on Anxiety Sensitivity Index (ASI)
Table 14. Showing Anxiety Sensitivity scores of Asthmatics with illness duration of 1 year or less and with illness duration of 1 to 5 year

<table>
<thead>
<tr>
<th>Anxiety Sensitivity Concerns</th>
<th>Asthmatics (N=30)</th>
<th></th>
<th>Asthmatics (N=21)</th>
<th></th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Duration of Disease: 1 year or less</td>
<td></td>
<td>Duration of Disease: 1 to 5 year</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
<td>S.D.</td>
<td></td>
</tr>
<tr>
<td>Total Scores</td>
<td>26.50</td>
<td>5.51</td>
<td>24.75</td>
<td>6.00</td>
<td>1.06</td>
</tr>
<tr>
<td>Physical concerns</td>
<td>13.63</td>
<td>2.12</td>
<td>10.45</td>
<td>2.29</td>
<td>5.04***</td>
</tr>
<tr>
<td>Mental Incapacitation concerns</td>
<td>5.47</td>
<td>1.82</td>
<td>8.05</td>
<td>2.35</td>
<td>4.23***</td>
</tr>
<tr>
<td>Social concerns</td>
<td>7.46</td>
<td>2.01</td>
<td>6.30</td>
<td>1.86</td>
<td>2.11*</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; ***significant at 0.001 level

Table 12 depicts that no significant difference between asthmatics with illness duration of 1 year or less and those with illness duration of 1 to 5 year is observed when the total scores obtained on Anxiety Sensitivity Index are compared when lower order factors are considered, asthmatics with illness duration of 1 year or less report higher level of physical and social concerns as compared to those having asthma from 1 to 5 year. While asthmatics with illness duration of 1 to 5 year endorse significantly higher mental incapacitation concerns as compared to those with illness duration of 1 year or less.

Therefore, hypothesis 6.1 is retained.
Table 15. Showing Anxiety Sensitivity scores of Asthmatics with illness duration of 1 year or less and with illness duration of more than 5 year

<table>
<thead>
<tr>
<th>Anxiety Sensitivity Concerns</th>
<th>Asthmatics (N=21) Duration of Disease: 1 year or less</th>
<th>Asthmatics (N=24) Duration of Disease: more than 5 year</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Total Scores</td>
<td>28.50</td>
<td>5.51</td>
<td>29.48</td>
</tr>
<tr>
<td>Physical concerns</td>
<td>13.63</td>
<td>2.12</td>
<td>12.50</td>
</tr>
<tr>
<td>Mental Incapacitation</td>
<td>5.47</td>
<td>1.82</td>
<td>9.35</td>
</tr>
<tr>
<td>Social concerns</td>
<td>7.46</td>
<td>2.01</td>
<td>8.15</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; **significant at 0.001 level

From table 13 it is clear that asthmatics with illness duration of more than 5 year as compared to those with illness duration of 1 year or less experience high degree of anxiety sensitivity. But, asthmatics with asthma duration of 1 year or less reports significantly higher physical concerns as compared to those with illness duration of more than 5 year. While, asthmatics with illness duration of more than 5 year endorse higher mental incapacitation concerns as compared to those with illness duration of 1 year or less.

Thus hypothesis 6.2 is confirmed by our results.
Table 16. Showing Anxiety Sensitivity scores of Asthmatic with illness duration of 1 to 5 year and with illness duration of more than 5 year

<table>
<thead>
<tr>
<th>Anxiety Sensitivity Concerns</th>
<th>Asthmatics (N=21) Duration of Disease: 1 to 5 year</th>
<th>Asthmatics (N=24) Duration of Disease: more than 5 year</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Total Scores</td>
<td>24.75</td>
<td>6.00</td>
<td>29.48</td>
</tr>
<tr>
<td>Physical concerns</td>
<td>10.45</td>
<td>2.29</td>
<td>12.50</td>
</tr>
<tr>
<td>Mental Incapacitation concerns</td>
<td>8.05</td>
<td>2.35</td>
<td>9.35</td>
</tr>
<tr>
<td>Social concerns</td>
<td>6.30</td>
<td>1.86</td>
<td>8.15</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; ***significant at 0.001 level

The perusal of table 14 shows that asthmatics with illness duration of more than 5 year differ significantly from those with illness duration of 1 to 5 year on anxiety sensitivity total score as well as on physical, mental incapacitation and social concerns.

Thus hypothesis 6.3 is borne out by our results.

Therefore, hypothesis 6 is retained which postulates that asthmatics with different duration of illness differ in their level of anxiety sensitivity.
Figure 10.1. Total scores of different illness duration groups of Asthmatics on Anxiety Sensitivity Index (ASI).

Figure 10.2. Scores of different illness duration groups of Asthmatics on different concerns on Anxiety Sensitivity Index (ASI).

*DoD = Duration of disease
Table 17. Showing Anxiety Sensitivity scores of Asthmatics of ages 12 to 20 years and of ages 20 to 35 years.

<table>
<thead>
<tr>
<th>Anxiety Sensitivity Concerns</th>
<th>Asthmatics (N=20) Ages: 12 to 20 years</th>
<th>Asthmatics (N=32) Ages: 20 to 35 years</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Total Scores</td>
<td>25.58</td>
<td>4.38</td>
<td>27.98</td>
</tr>
<tr>
<td>Physical concerns</td>
<td>13.26</td>
<td>1.68</td>
<td>12.03</td>
</tr>
<tr>
<td>Mental Incapacitation concerns</td>
<td>5.96</td>
<td>1.85</td>
<td>8.82</td>
</tr>
<tr>
<td>Social concerns</td>
<td>6.34</td>
<td>1.57</td>
<td>7.13</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; ***significant at 0.001 level

Table 15 illustrates that asthmatics of ages 12 to 20 years differed significantly from asthmatics of ages 20 to 35 years on two of the three lower order factors of anxiety sensitivity. Asthmatics of ages 12 to 20 years had significantly higher physical concerns, while asthmatics of ages 20 to 35 years showed significantly higher mental incapacitation concerns.

Thus hypothesis 7.1 stands accepted.
Table 18. Showing Anxiety Sensitivity scores of Asthmatics of ages 12 to 30 years and of ages 35 to 50 years

<table>
<thead>
<tr>
<th>Anxiety Sensitivity Concerns</th>
<th>Asthmatics (N=20) Ages: 12 to 20 years</th>
<th>Asthmatics (N=23) Ages: 35 to 50 years</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Total Scores</td>
<td>25.58</td>
<td>4.38</td>
<td>27.16</td>
</tr>
<tr>
<td>Physical concerns</td>
<td>13.26</td>
<td>1.68</td>
<td>11.29</td>
</tr>
<tr>
<td>Mental Incapacitation concerns</td>
<td>5.96</td>
<td>1.85</td>
<td>8.09</td>
</tr>
<tr>
<td>Social concerns</td>
<td>6.34</td>
<td>1.57</td>
<td>7.78</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level; **significant at 0.001 level

Table 16 indicates that asthmatics of ages 35 to 50 years scored significantly higher in comparison to asthmatics of ages 12 to 20 years. Asthmatics of ages 35 to 50 years had significantly higher mental incapacitation and social concerns while asthmatics of ages 12 to 20 years had significantly higher physical concerns.

Therefore, hypothesis 7.2 is retained.
Table 19. Showing Anxiety Sensitivity scores of Asthmatics of ages 20 to 35 years and of ages 35 to 50 years

<table>
<thead>
<tr>
<th>Anxiety Sensitivity Concerns</th>
<th>Asthmatics (N=32) Ages: 20 to 35 years</th>
<th>Asthmatics (N=23) Ages: 35 to 50 years</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Total Scores</td>
<td>27.98</td>
<td>6.42</td>
<td>27.16</td>
</tr>
<tr>
<td>Physical concerns</td>
<td>12.03</td>
<td>2.22</td>
<td>11.29</td>
</tr>
<tr>
<td>Mental Incapacitation concerns</td>
<td>8.82</td>
<td>3.18</td>
<td>8.09</td>
</tr>
<tr>
<td>Social concerns</td>
<td>7.13</td>
<td>2.11</td>
<td>7.78</td>
</tr>
</tbody>
</table>

Table 17 depicts no significant difference between the asthmatics of ages 20 to 35 years and of ages 35 to 50 years, in terms of their scores obtained on Anxiety Sensitivity Index.

Thus hypothesis 7.3 stands rejected.

Therefore, hypothesis 7 is partly borne out by our results.
**Figure 11.1.** Total scores of different age groups of Asthmatics on Anxiety Sensitivity Index (ASI).

**Figure 11.2.** Scores of different age groups of Asthmatics on different concerns on Anxiety Sensitivity Index (ASI).
### Table 20. Showing Anxiety Sensitivity scores of Male and Female Asthmatics

<table>
<thead>
<tr>
<th>Anxiety Sensitivity Concerns</th>
<th>Asthmatic Males (N=38)</th>
<th>Asthmatic Females (N=37)</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Total Scores</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>23.32</td>
<td>3.92</td>
<td>30.49</td>
</tr>
<tr>
<td>Physical concerns</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>12.52</td>
<td>1.54</td>
<td>11.84</td>
</tr>
<tr>
<td>Mental Incapacitation concerns</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5.79</td>
<td>1.80</td>
<td>9.45</td>
</tr>
<tr>
<td>Social concerns</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5.01</td>
<td>1.58</td>
<td>9.18</td>
</tr>
</tbody>
</table>

***significant at 0.001 level

From table 18, it is clear that asthmatic females experience significantly higher degree of anxiety sensitivity as compared to asthmatic males. Asthmatic females had higher mental incapacitation and social concerns as compared to their male counterparts.

Thus hypothesis 8 is confirmed.
Figure 12.1. Total Scores of Asthmatic Males and Asthmatic Females on Anxiety Sensitivity Index (ASI)

Figure 12.2. Scores of Asthmatic Males and Asthmatic Females on different concerns on Anxiety Sensitivity Index (ASI)
Chapter – V

Discussion
The objectives of the present investigation were to explore the attributional style and anxiety sensitivity among asthmatics. In view of the objectives certain research hypotheses were formulated. The results of the present study are discussed in the light of the ‘attributional style’ concept as evolved in the reformulated model of learned helplessness (Abramson et al., 1978) and termed as explanatory style by Peterson and Seligman (1984) and the anxiety sensitivity concept as presented by Reiss and McNally (1985). Abramson et al. (1978, 1980) proposed that causal attributions can be classified along internal-external, stable-unstable and specific-global dimensions. Attributions to these dimensions have implications for future expectations of noncontingency and symptoms of helplessness. Abramson et al. (1978) provided evidence of a depressive attributional style. These authors suggested that the particular attribution that depressed people choose for failure is probably irrationally distorted towards global, stable and external factors. Large number of studies indicated that internal, stable and global attribution to negative events and external, unstable and specific attribution to positive events (pessimistic attributional style) is a risk factor for various disorders. While the reverse (optimistic attributional style) is related to good health and psychological well-being (Khan and Jahan, 2006).

Our first hypothesis stated that asthmatics would have an attributional style different from non-asthmatics. It was observed that for positive events on Attributional Style Questionnaire (ASQ), asthmatics
obtained significantly lower composite score as compared to non-asthmatic normals. They also attributed positive events to more external rather than internal causes. For negative events, however, asthmatics obtained significantly higher composite score and attributed them to more global than specific factors.

Our findings are corroborated by the results of the earlier studies. Peterson (1988) found that a dimension that combines stable and global attributions was a better predictor of physical health than the internal dimension. Dua (1994) also found that of the attributions for good and bad events made along three dimensions, global attributions for bad events were the best predictors of self-reported emotional and physical health.

Peterson (1995) suggested biological, emotional, behavioural and interpersonal pathways between explanatory style and health. At the biological level the uncontrollability may compromise the immune system. At the emotional level the route between explanatory style and physical well-being runs through a wide range of negative feelings. At the behavioural level, explanatory style is associated with health relevant behaviour. Individuals with a pessimistic explanatory style do not do the sort of things that lead to long term well being. Unlike their more optimistic counterparts, they smoke, drink, and refrain from exercise (Peterson, 1988), when they happen to fall ill, they respond in a passive and helpless manner (Lin and Peterson, 1990). In contrast, individuals
with a more optimistic explanatory style take active steps in order to feel better (Peterson et al., 1992). As regards interpersonal pathways Peterson (1995) discusses that those with a pessimistic explanatory style are more likely to experience loneliness (Anderson and Arnoult, 1985) as they initiate fewer attempts to be friends with others.

Our second hypothesis that the patients with different duration of illness differ in their attributional style was only partly confirmed. Though the three groups (1 year or less, 1 to 5 year and above 5 years illness) did not differ significantly from each other on composite scores on ASQ for positive events, yet some differences appeared on internal-external and stable-unstable dimensions. Patients with 1 year or less duration of asthma were more external and stable for positive events, while patients between 1 to 5 year history of asthma made more internal attributions. Patients with above 5 year history of asthma onset made internal (scoring highest among three groups), but unstable attributions for positive events. Patients, in their earlier phase of illness, have more external and stable attributions for positive events. While the chronic patient’s attribution for positive events is more internal but remains unstable.

For negative events of ASQ the three groups differed significantly from each other depicting the highest composite score for 1 to 5 year illness duration group and lowest for 1 year or less illness duration group. Illness duration of 1 to 5 year group also made global attributions for
negative events while more than 5 year asthma duration group was both stable and global for making attributions to negative events.

Most researches evidence that stable and global attributions for negative events is the characteristic of depressive persons (for example, Sweeney et al., 1986). Researches also reveal that negative affects particularly depression cohere the asthmatic symptoms. Hence, the results of the present investigation suggest that as asthma prolongs/persists, it may make the patient more vulnerable to depression. Therefore, probably, patients with the longest duration of illness show the depressogenic attributional style.

Our next hypothesis was that patients of different age groups would exhibit different attributional style. The results did not reveal any significantly different attributional style for 12 to 20 years age group while for the group of 20 to 35 years significantly positive attributional style emerged. They made internal and stable attributions for positive events and unstable attributions for negative events. However, the age group 35 to 50 years had more negative attributional style specifically attributing negative events to stable causes. The present findings have some support from the results of an earlier longitudinal study conducted by Peterson (1995), which indicated that pessimistic attributions for negative events had strongest relationship with health-related problems at age 45 and above. He found that optimistic explanations for bad events at age 25 were related to good health and pessimistic style was unrelated to
health at age 30 to 40 years. But, thereafter a relationship emerged, reaching its most robust level at age 45 showing marked deterioration in their health.

A comparison of male and female asthmatics also revealed significant differences, leading to the acceptance of the hypothesis regarding gender differences. Asthmatic males obtained higher composite score for positive events, particularly attributing positive events to internal causes.

On the other hand, female asthmatics as compared to male asthmatics scored significantly higher composite score for negative events, especially attributing negative events to internal and stable causes. This showed that female asthmatics as compared to male asthmatics have more maladaptive attributional style.

One significant finding that the present investigation revealed is that asthmatic males did not differ from non-asthmatic normal males in terms of their attributional style. While, on the other hand, asthmatic females significantly differed from non-asthmatic normal females, particularly on attribution for positive events. Asthmatic females obtained significantly low composite score for positive events and made more external and unstable attributions. Whereas, for negative events no significant difference emerged on internal-external, stable-unstable or global-specific dimensions except composite score. These findings
suggest that though attributional style plays a significant role in women's asthma, it is, perhaps, not relevant for explaining asthma among males.

Another variable of the present investigation was anxiety sensitivity. Anxiety sensitivity as conceptualized by Reiss and McNally (1985) in their expectancy model is the fear of anxiety related thoughts and bodily sensations based on belief that they will be harmful. It is a cognitive risk factor/pattern of thinking that can affect health, "Just having this type of thinking pattern puts a person at greater risk for developing physical or mental impairment" (Schmidt, 1998). High anxiety sensitivity has also been a predisposing factor in the development and maintenance of anxiety disorders and has a strong relationship especially to panic disorder (Schmidt, Lerew and Jackson, 1997). The result of the present study also insonance with the earlier findings. It was hypothesized that asthmatics and non-asthmatics would differ in the level of anxiety sensitivity. The results obtained were in accordance with the hypothesis as asthmatics were found to have significantly higher level of anxiety sensitivity when compared to non-asthmatic normal group. The present results are endorsed by the findings of a pilot study carried out by Khan and Jahan (2005) which found that asthmatics show higher level of anxiety sensitivity when compared to nonasthmatics. Asthmatics also scored high on lower order factors, pointing to high level of physical, mental incapacitation as well as social concerns. The same findings were obtained when comparisons between asthmatic and normal males, and asthmatic and normal females were made. These findings were partly supported by the
findings of earlier studies (Carr, Lehrer, and Hochron, 1995; Carr, Lehrer, Rausch and Hochron, 1994, Khan and Jahan, 2005) conducted on asthmatics, which found anxiety sensitivity to be significantly related to panic disorder among asthmatics. They also found that cognitive variables predicted both illness specific panic fear and generalized panic fear.

Keogh and his associates (for example, Keogh and Cochrane, 2002; Keogh, Ellery, Hunt and Hannert, 2001) on the basis of their extensive and intensive researches on anxiety sensitivity and pain postulated that anxiety sensitivity is a trait susceptibility associated with the fear of anxiety related sensations. The reason why such fears exist may be because of the cognitive/information processing, such as attentional bias and interpretative bias. Those high in anxiety sensitivity selectively attend toward such sensations (for example, physical or social) and they have a tendency to misinterpret ambiguous events and sensations in a negative manner. Evidence exists that anxiety sensitivity is associated with both attentional and interpretative biases. Negative emotions such as anxiety and depression are characterized by negative processing biases. The biased cognitive processing not only helps in maintaining such mood states in the clinical group but also makes the nonclinical persons to be vulnerable for negative emotions. They further argued that as pain patients often report high level of anxiety and depression, it is likely that they will also exhibit cognitive processing biases. The cognitive processing biases mediate the anxiety sensitivity-pain relationships.
Borden and Lister (1994) had also suggested that individuals with high levels of anxiety sensitivity believe the experience of anxiety as harmful and monitor their physiological responses by focusing attention to their internal stimuli.

The same explanation seems to be true for asthma condition. Given that patients with asthma also report negative emotional states like depression and anxiety (Bennett, 1994; Goldney et al., 2003; Kashani, Konig, Shepperd, Wilfley and Morris, 1988; MacLean, Perrin, Gortmaker and Pierre, 1992, Opolski and Wilson, 2005) it is likely that they will exhibit biased cognitive processing. Asthma is a chronic lung disease characterized by recurrent breathing problems and symptoms such as breathlessness and chest tightness, together with cognitive dyscontrol (for example, dizziness and nervousness). These symptoms vary over times, and also differ in severity. Asthma often leads to hospitalization, missed work, school absenteeism, limitations on physical activities, sleepless nights and in some cases even death. Therefore, asthmatic is expected to be highly attentive toward those visceral/bodily cues which occur prior to and during an asthma attack. Anxiety sensitivity amplifies attention to fear related physiological sensations through a process of vigilance and hypervigilance. This in turn leads to increased anxious behaviour among high anxiety sensitivity patients and thus it makes a vicious circle.

It was also hypothesized that asthmatics with different durations of the disease would exhibit different levels of anxiety sensitivity. The results of
the present study confirmed the hypothesis, as patients with the longest duration of illness, that is more than 5 years, reported the highest level of anxiety sensitivity. These findings were obtained by Khan and Jahan (2005) in a pilot study, indicating that patients with different illness duration (that is less than 1 year, 1 to 5 year and more than 5 year) vary in their anxiety sensitivity. It was observed that anxiety sensitivity was high in the beginning, followed by a decrease, and again an increase in its level. They also reported high mental incapacitation concern followed by social concerns. Conversely, patients with illness duration of 1 year or less as compared to other groups had the highest level of physical concerns and the least mental incapacitation concerns. However patients with illness duration of 1 to 5 year reported/exhibited the lowest level of anxiety sensitivity.

Our next hypothesis stated that patients in different age groups would differ in their level of anxiety sensitivity. The hypothesis was only partly confirmed as patients in different age groups did not differ in their total anxiety sensitivity score. Yet they were significantly different on lower order factors. 12 to 20 years age group as compared to other groups had significantly highest level of physical concerns, significantly lower social concerns and least mental incapacitation concerns. 20 to 35 years group did not differ significantly from 35 to 50 years group. But as compared to 12 to 20 years group, 35 to 50 years group had significantly lowest physical concerns, but highest mental incapacitation and social concerns.
The above findings get support from earlier studies which reveal that mental incapacitation concerns had the strongest positive linear relation with depressed mood (Schmidt, Lerew and Joiner, 1998; Taylor, Koch, Woody and McLean, 1996) while physical concerns showed the strongest positive relation with panic related phenomena (Zinbarg, Brown, Barlow and Rapee, 2001) — fear responses to hyperventilation challenge and 5.5% CO2.

It seems that during early phase of the illness the patient reacts panic related symptoms to asthmatic attack with panic like symptoms and, therefore, show more physical concerns. As duration of illness increases and asthma becomes chronic, affecting the person’s almost all life activities, it leads to depression and therefore the patients in the later stage of life with long standing problem of asthma experience highest level of mental incapacitation concerns.

Our next hypothesis concerned the gender differences and it was expected that male and female asthmatics would differ in their level of anxiety sensitivity. The results of the study confirmed the hypothesis revealing significant differences between the two groups on anxiety sensitivity. Asthmatic females as compared to asthmatic males scored significantly higher on Anxiety Sensitivity Index. Same finding was also observed by Khan and Jahan (2005), revealing that female asthma patients reported greater anxiety sensitivity than male asthma patients. Though males scored a little higher on physical concerns, the difference between the means
was not statistically significant. On the other hand, females expressed significantly higher mental incapacitation as well as social concerns.

The results of the present investigation are partly supported by the anxiety sensitivity-pain relationship studies conducted by Keogh and associates (Keogh and Birkby, 1999; Keogh, Hamid, Hamid and Ellery, 2004). They found that both anxiety sensitivity and gender were related with the experience of pain. Females high in anxiety sensitivity were found to report greater sensory pain compared than females low in anxiety sensitivity. Anxiety sensitivity was, however, not found to significantly mediate the sensory pain responses of males.
Chapter – VI

Conclusions, Implications and Suggestions for Future Research
CONCLUSIONS

From the discussion it is concluded that the asthmatics differ significantly from non-asthmatic normal counterparts in terms of attributional style and anxiety sensitivity. Global attributions for negative events emerged as a significant factor. Chronicity also emerged as an important mediating variable between attributional style and asthma, as chronicity increases the patients make more internal and unstable attributions for positive events and global and stable attributions for negative events. Patients of different age groups also reveal significant differences in their attributional style. 20 to 35 years age group show most positive attributional style, making internal and stable attribution for positive events and unstable attribution for negative events. 35 to 50 years age group show the most pathological attributional style making stable attribution for negative events. When gender differences were probed it emerged as a significant mediating variable in the relationship between attributional style and asthma. Women patients revealed a depressogenic attributional style.

When anxiety sensitivity is taken into consideration it is found that it is undoubtedly crucial variable that may enhance the probability of asthmatic attacks. Chronicity, age and gender mediate anxiety sensitivity and asthma relationship. Patients with shortest duration of illness report more physical concerns. As duration of illness increases patients exhibit more mental incapacitation and social concerns. Age of the patients seems to mediate only the lower order factors on anxiety sensitivity as 12 to 20 years age group
expresses more physical concerns while the highest age group (35 to 50 years) reports more mental incapacitation and social concerns.

As regards the role of gender it is found that though male asthmatics as compared to non-asthmatic males have higher level of anxiety sensitivity but as compared to women asthmatic they are low on anxiety sensitivity with no specific concerns. Women asthmatics have high mental incapacitation concerns and social concerns.

IMPLICATIONS

The paradigm shift in psychology and medicine led its professionals to adopt a bio-psycho-social perspective which resulted in an increased interest of the researcher in exploring psychological aspects of various physical ailments. This perspective maintaining that many illnesses are associated with structural damage, and may require chemical or surgical intervention. However, the presence of structural damage does not rule out the possibility that psychological variables were critical in precipitating the disorder. The conditions that maintain a disorder need not be the same as the condition that led to the disorder. Thus, although psychological manipulations may not be effective in curing an established disorder, they may be effectively used to reduce the overall incidence of the disease though the implementation of preventive medicine programs.

The present study is an humble effort in the same direction. As the results of the present investigation advocate the role of two of the important
psychological variables - attributional style and anxiety sensitivity - in asthma, the study will have the following implications:

1. It will contribute to the repertory of researches in the field of medical psychology.

2. It will help the health-care professionals to understand the role of two important cognitive variables, namely attributional style and anxiety sensitivity, among asthmatics.

3. The results of the present study, to a great extent, may also have implications for asthma management. Interventions may be used which would target the attributional style and anxiety sensitivity. Professionals can think of changing these cognitive states of mind of asthmatics to help them recover early and improve their health. There are evidences that cognitive therapy can change explanatory style from pessimistic to optimistic (Seligman et al., 1988). It is also hoped that anxiety sensitivity may also be reduced by using certain cognitive therapy techniques.

4. Understanding of these dynamics may also help parents to think about fostering in their children those cognitive states/styles which will be helpful to them in later life. In the words of Peterson, “It seems unlikely that anyone would be rescued from death’s door by changing his or her explanatory style. But it is not so far-fetched to think that the deliberate encouraging of an optimistic way of explaining events, started early in
life, might later pay dividends in terms of increased quantity and quality of life. And it is not far-fetched to think that the acquisition of an optimistic explanatory style might help a person recover more quickly and more fully from a less than fatal illness” (Peterson, 1995).

SUGGESTIONS FOR THE FUTURE RESEARCH

Despite of voluminous work on bio-psycho-social variables among the psychosomatic patients, there are many other areas that need to be explored further in order to gain better understanding of the phenomenon. Whatever may be the result of the investigation it does not mean that it blocks the way to future researches.

- Future researches should investigate the attributional style of asthmatic patients with or without depression, in order to reflect how they explain the events that befall them (in depressive or in efficacious manner).

- In future, in the same way, asthmatic with or without panic-related symptomatology may be taken to find out the differences in their level of anxiety sensitivity.

- Better result would also be obtained if we take into consideration other factors, such as- disease expectations, illness uncertainty, and the biographical characteristics of the patients.

- Although the present investigation had taken into consideration the age, duration of disease and gender, additional measures of subjective and
objective disease severity (including Pulmonary Function Test) should also be included in the future investigation.

- Researcher may use the revised version of Attributional Style Questionnaire and Anxiety Sensitivity Index.

- Finally, the measure included in the present investigation were all self-reported in nature, introspective reports given by asthmatic patients may add potential in the investigation.
Summary
The entire empirical research work on the problem “Attributional Style and Anxiety Sensitivity among Asthmatics” has been presented systematically in six different Chapters.

In Chapter-I, the first part deals with the concept, definition, physiology, types, etiology and psychological correlates of asthma. The next part of this chapter describes the concept and definition of attribution; various attribution theories; the origin, definitions, terminology, classification, gender and individual differences, physiological as well as psychological health-related functioning and other related concepts of attributional style. The last part of the chapter-I was concerned with the concept, theories, definition and nature of anxiety sensitivity. In addition to gender and individual differences on anxiety sensitivity, the relation of anxiety sensitivity to panic, depression and other disorders was also discussed. In the light of the aims and objectives of the present investigation, eight hypotheses were formulated. These hypotheses took into consideration attributional style, anxiety sensitivity, duration of illness, age and gender.

Chapter-II deals with the review of the literature so that the issues and problems related to the phenomenon were clarified and highlighted. The second chapter was divided into three parts. The first part included a review of research studies of etiology of asthma; negative affectivity, stress, anxiety and depression among asthmatics. The second part was especially concerned with the review of researches conducted in the field of attributional style and health related functioning such as anxiety, depression and other disorders. The third part, more remarkably, dealt with the review of studies on anxiety
sensitivity in various samples (for example- panic patients, pain-patients and asthmatics). Moreover, the gender differences on anxiety sensitivity were also reviewed.

Chapter-III was designed for describing methodology, where the sample, tools, procedure and the statistical analysis opted in carrying out the investigation has been comprehensively enumerated. The sample consisted of 150 subjects (75 asthmatics and 75 non-asthmatics) between the ages of 12 to 50 years, selected on the basis of purposive sampling technique from J.N. Medical College, Aligarh Muslim University and other clinics in Aligarh city. The Attributional Style Questionnaire (Peterson and Seligman, 1984) and the Anxiety Sensitivity Index (Reiss, Peterson, Gursky and McNally, 1986) were used.

In chapter-IV, the results have been presented systematically in various tables. Each table displayed the Means, S.D.s, t-values and the level of significance. Comparisons were made between various groups.

Chapter-V was meant to discuss the obtained findings. The emerged overall pictures were discussed comprehensively and extensively. Beside this, the obtained results were supported and justified in the light of earlier empirical studies.

In chapter-VI, conclusions, suggestions and, implications have been presented avariciously. Having described outcomes of the study, in brief, some suggestions for the future investigations, as well as the implications of the present research work have also been putforth.
References


Rees, L. (1956). The physical and emotional factors in bronchial asthma. Journal of Psychosomatic Research, 1, 98.


Appendix – I

Attributional Style Questionnaire

Name: ........................................................................................................ Gender: .................Age.............

Disease (if any) .................................................................................. Duration of disease: ........................................

INSTRUCTIONS:
1. Read each situation and vividly imagine it happening to you.
2. Decide what you believe would be the one major cause of the situation if it happened to you.
3. Write this cause in the blank provided.
4. Answer three questions about the cause by circling one number per question. Do not circle the words.
5. Go on the next situation.

SITUATIONS:

You meet a friend who compliments you on your appearance.

1. Write down the one major cause:

2. Is the cause of your friend’s compliment due to something about you or something about other people or circumstances?
   Totally due to other people or circumstances? 1 2 3 4 5 6 7 Totally due to me

3. In the future when you are with your friend, will this cause again be present?
   Will never again be present 1 2 3 4 5 6 7 Will always be present

4. Is the cause something that just affects interacting with friends, or does it also influence other areas of your life?
   Influence just this particular situation 1 2 3 4 5 6 7 Influence all situation in my life

You have been looking for a job unsuccessfully for some time.

5. Write down the one major cause:

6. Is the cause of your unsuccessful job search due to something about you or something about other people or circumstances?
   Totally due to other people or circumstances? 1 2 3 4 5 6 7 Totally due to me
7. In the future when you look for a job, will this cause again be present?

Will never again be present 1 2 3 4 5 6 7 Will always be present

8. Is the cause something that just influences looking for a job, or does it also influence other areas of your life?

Influences just this particular situation 1 2 3 4 5 6 7 Influences all situations in my life

You become very rich.

9. Write down the one major cause:

____________________________________________________

10. Is the cause of your becoming rich due to something about you or something about other people or circumstances?

Totally due to other people or circumstance 1 2 3 4 5 6 7 Totally due to me

11. In your financial future, will this cause again be present?

Will never again be present 1 2 3 4 5 6 7 Will always be present

12. Is the cause something that just affects obtaining money, or does it also influence other areas of your life?

Influences just this particular situation 1 2 3 4 5 6 7 Influences all situations in my life

A friend comes to you with a problem and you don’t try to help him/her.

13. Write down the one major cause:

____________________________________________________

14. Is the cause of your not helping your friend due to something about you or something about other people or circumstances?

Totally due to other people or circumstances 1 2 3 4 5 6 7 Totally due to me

15. In the future when a friend comes to you with a problem, will this cause again present?

Will never again be present 1 2 3 4 5 6 7 Will always be present
16. Is the cause something that just affects what happens when a friend comes to you with a problem, or does it also influence other areas of your life?

Influences just this particular situation 1 2 3 4 5 6 7  
Influences all situations in my life

You give an important talk in front of a group and the audience reacts negatively.

17. Write down the one major cause:


18. Is the cause of the audience's negative reaction due to something about you or something about other people or circumstances?

Totally due to other people or circumstances 1 2 3 4 5 6 7  
Totally due to me

19. In the future when you give talks, will this cause again be present?

Will never again be present 1 2 3 4 5 6 7  
Will always be present

20. Is the cause something that just influences giving talks, or does it also influence other areas of your life?

Influences just this particular situation 1 2 3 4 5 6 7  
Influences all situations in my life

You do a project which is highly praised.

21. Write down the one major cause:


22. Is the cause of your being praised due to something about you or something about other people or circumstances?

Totally due to other people or circumstances 1 2 3 4 5 6 7  
Totally due to me

23. In the future when you do a project, will this cause again be present?

Will never again be present 1 2 3 4 5 6 7  
Will always be present

24. Is the cause something that just affects doing projects, or does it also influence other areas of your life?

Influences just this particular situation 1 2 3 4 5 6 7  
Influences all situations in my life
You meet a friend who acts hostilely towards you.

25. Write down the one major cause:

________________________________________________________________________

26. Is the cause of your friend acting hostile due to something about you or something about people or circumstances?

<table>
<thead>
<tr>
<th>Totally due to other people or circumstances</th>
<th>1 2 3 4 5 6 7</th>
<th>Totally due to me</th>
</tr>
</thead>
</table>

27. In the future when interacting with friends, will this cause again be present?

<table>
<thead>
<tr>
<th>Will never again be present</th>
<th>1 2 3 4 5 6 7</th>
<th>Will always be present</th>
</tr>
</thead>
</table>

28. Is the cause something that just influences interacting with friends, or does it also influence other areas of your life?

| Influences just this particular situation | 1 2 3 4 5 6 7 | Influences all situations in my life |

You can't get all the work done that others expect of you.

29. Write down the one major cause:

________________________________________________________________________

30. Is the cause of your not getting the work done due to something about you or something about other people or circumstances?

<table>
<thead>
<tr>
<th>Totally due to other people or circumstances</th>
<th>1 2 3 4 5 6 7</th>
<th>Totally due to me</th>
</tr>
</thead>
</table>

31. In the future when doing work that others except, will this cause again be present?

<table>
<thead>
<tr>
<th>Will never again be present</th>
<th>1 2 3 4 5 6 7</th>
<th>Will always be present</th>
</tr>
</thead>
</table>

32. Is the cause something that just affects doing work that others except of you, or does it also influence other areas of your life?

<table>
<thead>
<tr>
<th>Influences just this particular situation</th>
<th>1 2 3 4 5 6 7</th>
<th>Influences all situation in my life</th>
</tr>
</thead>
</table>
Your friend has been treating you more lovingly.

33. Write down the one major cause:

34. Is the cause of your friend treating you more lovingly due to something about you or something about other people or circumstances?

| Totally due to other people or circumstances | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Totally due to me |

35. In future interactions with your friend, will this cause again be present?

| Will never again be present | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Will always be present |

36. Is the cause something that just affects how your friend (boyfriend/girlfriend) treats you, or does it also influence other areas of your life?

| Influences just this particular situation | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Influences all situations in my life |

You apply for a position that you want very badly (e.g. important job, graduate school admission, etc.) and you get it.

37. Write down the one major cause:

38. Is the cause of your getting the position due to something about you or some thing about other people or circumstances?

| Totally due to other people or circumstances | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Totally due to me |

39. In the future when you apply for a position, will this cause again be present?

| Will be never again be present | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Will always be present |

40. Is the cause something that just influences applying for a position, or does it also influence other areas of your life?

| Influences just this particular situation | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Influences all situations in my life |
You go out on a tour and it goes badly.

41. Write down the one major cause:

42. Is the cause of the tour going badly due to something about you or something about other people or circumstances?
   
   Totally due to other peoples or circumstances 1 2 3 4 5 6 7

43. In the future when you go out on a tour, will this cause again be present?
   
   Will never again be present 1 2 3 4 5 6 7

44. Is the cause something that just influences tour, or does it also influence other areas of your life?
   
   Influences just this particular situation 1 2 3 4 5 6 7

You are awarded a prestigious scholarship.

45. Write down the one major cause:

46. Is the cause of your getting a scholarship due to something about you or something about other people or circumstances?
   
   Totally due to other people or circumstances 1 2 3 4 5 6 7

47. In the future in your academic career, will this cause again be present?
   
   Will never again be present 1 2 3 4 5 6 7

48. Is this cause something that just affects getting a scholarship, or does it also influence other areas of your life?
   
   Influences just this particular situation 1 2 3 4 5 6 7

Appendix – II

Anxiety Sensitivity Index

Circle the one phrase that best represents the extent to which you agree with the item. If any of the items concerns something that is not part of your experience (e.g., “It scares me when I feel shaky” for someone who has never trembled or had the “shakes”), answer on the basis of how you think you might feel if you had such an experience. Otherwise, answer all items on the basis of your own experience.

1. It is important to me not to appear nervous.
   Very little  A little  Some  Much  Very much

2. When I cannot keep my mind on a task, I worry that I might be going crazy
   Very little  A little  Some  Much  Very much

3. It scares me when I feel “shaky” (trembling)
   Very little  A little  Some  Much  Very much

4. It scares me when I feel faint.
   Very little  A little  Some  Much  Very much

5. It is important to me to stay in control of my emotions.
   Very little  A little  Some  Much  Very much

6. It scares me when my heart beats rapidly.
   Very little  A little  Some  Much  Very much

7. It embarrasses me when my stomach growls.
   Very little  A little  Some  Much  Very much

8. It scares me when I am nauseous.
   Very little  A little  Some  Much  Very much

9. When I notice that my heart is beating rapidly, I worry that I might have a heart attack.
   Very little  A little  Some  Much  Very much

10. It scares me when I become short of breath.
    Very little  A little  Some  Much  Very much

11. When my stomach is upset, I worry that I might be seriously ill.
    Very little  A little  Some  Much  Very much

12. It scares me when I am unable to keep my mind on a task.
    Very little  A little  Some  Much  Very much

13. Other people notice when I feel shaky.
    Very little  A little  Some  Much  Very much

14. Unusual body sensations scare me.
    Very little  A little  Some  Much  Very much

15. When I am nervous, I worry that I might be mentally ill.
    Very little  A little  Some  Much  Very much

16. It scares me when I am nervous.
    Very little  A little  Some  Much  Very much