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 Klinnert, 1998; Weiss and Wagener, 1990). It is now the world’s most common long-term condition, according to the Global Burden of Asthma Report (Masoli, 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 in and out of the lungs.

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.

<table>
<thead>
<tr>
<th>Factors</th>
<th>Relative importance, %</th>
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<tr>
<td></td>
<td>Dominant</td>
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<tr>
<td>Allergic</td>
<td>23</td>
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<tr>
<td>Infective</td>
<td>38</td>
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<tr>
<td>Psychological</td>
<td>37</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 and 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-Hoeksema 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 (Försterling, 1985).

ANXIETY SENSITIVITY

In 1985 Steven Reiss and Richard McNally put forth 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 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 elevated 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 (Lilenfield, 1996). Reiss (1997) discussed the conceptual and theoretical difference between trait anxiety and anxiety sensitivity. He said that, trait anxiety begans as a psychodynamic concept, poorly tied to observable and requiring Freudian defense mechanism to explain recurrent anxiety episodes. McNally (1989, 1996a, 1966b) 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 tripartile model of depression and anxiety. They noted that the tripartile
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 Eysenk, 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 from 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 sign 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
are the same as for those with low anxiety sensitivity scores (Asmundson et al., 1994).

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.