Chapter-III

THE REVIEW OF
PREVIOUS LITERATURE
In its constitution the WHO offered a dynamic and positive definition of health, in terms of psychological and social as well as physical well being and emphasised that it is both dynamic and changeable. It is a common assumption if not a “cultural truism” that the experience of stress is necessarily associated with the impairment of health. It is increasingly being realised that many physical and psychological disorders arise directly and indirectly from what people do to themselves rather than solely from external sources such as viruses. There are a range of disorders which are often referred to collectively as “stress-induced” illnesses which seem to stem from the way people respond to adverse environmental factors.

Stress as a psychological concept has two meanings – a force imposed upon a system and the effect of a force upon the system. Personality and behavioural traits on the one hand and social environmental characteristics on the other determine the nature of one’s experience of stress and well being. Stress affects an individual in a variety of ways like cognitive deficits, inducing helplessness and irritability affecting physical and psychological health.

Reactions to stressful events have three elements – an emotional response, a physical response and psychological responses that reduce the impact of the stressor. The emotional response may be anxiety or depression or both. The physical response varies accordingly. The physical response associated with anxiety is autonomic arousal and increased muscle tension, the
physical response associated with depression is fatigue and lethargy. The psychological responses that serve to reduce the impact of stressful events are mechanisms of defence and coping strategies. Stress also has deleterious physiological consequences via the GAS which mediates via the adrenal cortex. The recognition of these effects serves to emphasize the importance of maintaining a psychosocial as well as a biological equilibrium in safeguarding health.

Hence, it may be deduced that stress results in many psychological changes within the personality that may as a consequence lead to SD, thus its impact needs to be reviewed accordingly as the study warrants it.

Many major changes in peoples' lives which necessitate physical or psychological adjustment now appear to be associated with a greater proneness to illness. The recognition of these effects serves to emphasise the importance of maintaining a psychosocial as well as a biological equilibrium in safeguarding health.

A more sophisticated approach is provided by so-called diathesis-stress models. The diathesis part of the model refers to a permanent physical vulnerability of the body which makes it more vulnerable to particular diseases. In psychiatric disorders the interaction between a permanent physical vulnerability and some form of physical or psycho-social stress forms the central
part of the model. The diathesis-stress models are common now not only in the biomedical approach but in a range of psychological (sensitivity to the experience of loss) or social level (a lack of social support from other individuals) rather than at a physical level. Various studies emphasise the fact that psychological factors play a major role towards initiating diseases.

A study by Aitken (1984) has also shown that the influence of psychological factors is more important whether the disease is of known or unknown etiology. Goldsmith (1983-1984) found that in psychiatric disorders there is always an implication of hidden psychological causes that antagonizes these patients needlessly. That the importance of psychological factors and somatic mechanisms is involved in practically all types of illnesses was stressed in the Presidential Address of the 30th Annual Meeting of the Academy of Psychosomatic Medicine 1983, where the presence physical factors involved in psychological conditions and disorders, traditionally considered to be psychological was discussed. It was also emphasizes that clinicians should become increasingly informed about somatic developments (Henker 1984). If the emotional response to life events is of sufficient intensity, the disorder may be “functional” and involve less organic pathology.

Studies have shown that stress affects somatizers most (Miranda, Pérez-Stable, Muñoz, Hargreaves et al. 1991), probably somatisation occurs due to severe life stress during early childhood (Reister, Tress, Schepank, Manz et al.)

In recent years there have been a number of studies investigating the relationship between changes in someone's life situation and the onset of illness. This follows from the work of Holmes and Rahe (1967) and others who maintain that life changes or life events require adjustments in the life pattern of an individual and that this will be stressful. They have focused on the impact of life changes which would be rated as negative (eg. divorce) as well as those which are positive (eg. promotion). These studies have shown that life events cluster significantly in a 2-year period preceding an illness and that the onset of an illness can be predicted by the total number of life events. Thus they maintain that it is possible to quantify the impact of life changes and that high levels of life change are correlated with the onset of disease.

This earlier work has been subject to a great deal of criticism on methodological grounds (Rabkin and Struening 1976, Cohen 1979). It was based on a rather simple stimulus-based approach to stress and has the problem that people have to report life events retrospectively which can introduce all sorts of biases. Also it emphasized the negative effects of life changes and ignores the fact that many people may directly benefit from a life
change or may undergo life events without developing a subsequent illness. As a result, recent work on these lines has concentrated more on the impact of events on the individual and this is seen to be a function of the appraisal and coping ability of the individual as well as the social support available (Craig and Brown 1984). Also there is now a greater focus on negative life events since these have been found to be more correlated with subsequent illness.

Research evidence on the impact of life events and life changes have been extensive. A theoretical model has been presented where illness is considered only one of several possible reactions to stress. The effects of stress depends on an individual’s adaptive capacity and on the presence of stressors. It was also found that individually determined effects of life changes often play a crucial role. The relevance of life changes should be considered in relation to the biopsychosocial environment and the individual’s psychological history (Mattila and Salokangas 1981). Hönmann and Schepank (1983) contends that life events are life changing events with a probable pathogenic effect. The concept of life events research is related to a “trigger” situation for the manifestation of symptoms. Readjustments in life events following a change in income raised the psychological distress levels of subjects relative to those of controls. Results were consistent with the life events hypothesis (Thoits and Hannan 1979). Fisher (1984, 1986) proposed that life events create changes in the level of demand together with reduction of control over various situations. A study by
Agrawal and Naidu (1988) contradicts the life events hypothesis because when a scale of stressful experiences were administered to 100 university students to determine the effect of stress (desirable and undesirable life events rated according to distress and change) on strains (mental, physical or disease). Results show that change was not a critical predictor of strains.

Selye's General Adaptation Syndrome (GAS) lends itself as an explanatory model of accounting for the effects of life changes or events on health. Investigators of life changes have argued that they require readjustment by the individual and that this could result in physiological activation. Over time these could have a deleterious effect on the body and result in illness. This would explain why more frequent and more severe life changes have an increased likelihood of preceding the onset of disease. Thus it is argued that sustained and unsuccessful attempts at coping with life changes will lower bodily resistance and enhance the probability of disease occurrence.

Sargent (1983) describes the development of a theoretical model that studied the impact of contextual factors on an individual's physiologic and emotional functioning. Also in a paper presented at the Tenth International Round Table for the Advancement of Counseling, 1982, it was discussed that both the medical practitioner as well as the behavioural scientist should be aware of the capacity of the human body to respond to situational stress. When the fight or flight mechanism is activated in chronic situations, there is no
provision for the curtailed systems to be refurbished or to recuperate from the strain that has been placed on them. Repeated shut down and the associated deterioration results in physiological breakdown that manifests itself as complaints. Two factors appear to be critical in minimizing stress and its effects: (1) it is important for people to find an environment that is as close as possible to their personality and learn to control their environment rather than let the expectations of the circumstances that are around them control their life; (2) with the use of cognitive models the psychologist can recognize and help eliminate that cause of an individual's stress (Buckner 1984).

Pennebaker (1985) proposed a model to determine the relationship between traumatic experience and disease which included the following propositions. (1) To actively inhibit one's behaviour is stressful and disease-related; (2) When behavioural inhibition occurs there is an increased probability of obsessing about the event as well as long term illness consequences; (3) The act of confiding or otherwise translating the event into language reduces autonomic activity and disease rates. How the event is discussed, the possibility of ever coming to terms with the event and the ultimate consequences of discussing the experience are all variables that may influence the outcome of confiding, inhibition and ultimately health.

Wickramasekera (1986) hypothesized that there are 5 measurable high-risk factors that predispose people to develop functionally based somatic
disorders and that the factors compose a multidimensional model that encompasses variables involved in the predisposition, the precipitation and the buffering of stress-related symptoms. These high-risk factors are: (a) high or low hypnotic ability (b) habitual catastrophising cognitions and pessimistic belief systems (c) autonomic lability or neuroticism (d) multiple major life changes or minor hassles over a short period of time and (e) a deficit in support systems or coping skills or both. The same experimenter revised his theory in 1995 by studying 83 consecutive patients with chronic somatic complaints. They were tested on the 8 risk factors of the High Risk Model of Threat Perception. The model identified 3 predisposing factors (hypnotic ability, catastrophising and negative affectivity) that amplify the probability that 2 triggering variables (major life change and minor hassles) will generate psychological or somatic symptoms unless the impact of the triggers and predisposers are buffered.

Numerous studies have cited results in support of this theory (Nemiah 1981, Katon 1985, Miller and Ingham 1985, Starkman and Appelblatt 1984, Craig, Drake, Mills and Boardman 1994). Ironically certain studies have contradicted the theory directly or indirectly. Firstly, the theory has been criticized with respect to the availability of social supports. Sarason, Sarason, Potter and Antoni (1985) used a social support questionnaire to correlate positive and negative life events and social support with illness among 163 male school students aged 18-27 years. The results showed that negative life events
in the recent past were related to reports of illness. Although social support was not related to illness reports, the relation between negative life events and illness was stronger among subjects with low rather than high levels of social support. Another study assessed the predictive power gained by including measures of controllability and chronicity in a simple frequency count of stressors in 128 female and 100 male undergraduates. While available social support did not influence the stress-illness relationships, greater willingness to utilize social support was associated with lower levels of depression (Gannon and Pardie 1989).

Some studies have been conducted to determine the impact of hassles on reports of somatic symptoms, contradictions have arisen among the observed results. 88 undergraduates responded to questionnaires based on a 9 month period in a prospective study of the impact of hardiness, life events and hassles (daily mundane stressors) on reports of somatic symptoms. Data suggest that life events, hassles and symptoms were significantly related in an interactive manner. Although, life events shared a high degree of variance with hassles, hassles consistently contributed above and beyond life events in predicting somatic symptoms (Banks and Gannon 1988). Upadhyaya and Mastan (1988) explored the type, frequency and intensity of hassles and uplifts in normal daily living and found a positive correlation between hassles and gender. Male hassles and uplifts included work assignments and good food respectively.
Female hassles and uplifts included interactions and being with family respectively. Scaled scores indicated that most subjects irrespective of gender were concerned about the meaning of life and considered communicating well with others an uplift. Another study by Lepore, Palsane and Evans (1991) revealed that chronic strains including ongoing social and environmental conditions represented high intensity stressors that threatened survival, when compared to daily hassles. Chronic strains were associated with greater levels of symptomatology as well as lower perceived social support.

Pearlstone, Russell and Wells (1994) examined relationships among major life events, hassles, neuroticism and reported somatic symptoms for 131 subjects aged 15-83 years. Analyses of a hassles questionnaire revealed 4 factors – health, money, intimacy and work. Concerns about money did not correlate significantly with any of the other variables. Neuroticism and life events contributed differentially to the 3 hassles factors which in turn were related to somatic symptoms. It was argued that the hassles factor most strongly predictive of somatic symptoms – concerns about health was more likely to be a consequence than a cause of ill health. Minor stressors fall into a number of different classes that have different causes and effects. These results cast doubt on the concept of stress as a unitary phenomena, however results of other studies by Lazarus and Folkman 1984, Appley and Trumbell 1986, Evans (in press), Evans and Cohen (1987) and Lepore and Evans (1996) show that
certain specific stressors affect human health and behaviour. When Frost, Morgenthau, Riessman and Whalen (1988) examined physical symptoms of 173 undergraduate women who scored high or low on (1) a measure of the tendency to experience stress somatically and (2) a measure of current stress, results indicated that high somatic subjects reported greater number of symptoms than low somatic subjects regardless of stress level. However high somatic subjects who were experiencing high levels of current stress reported significantly more symptoms than high somatic subjects who were experiencing low levels of stress.

Stress has also been studied as a stimulus-response mechanism. Martin (1984) discusses the concept of stress in its relation to illness onset, focusing on how stimuli influence human functioning. The study concluded that the interaction between stimulus and response was the main arena of disease onset. Another study contradicts this finding. Dressler (1985) presented a model in which cultural context influenced the relationship between socio-cultural stressors and somatic symptoms, specifically with regard to modernization. Depending upon the social stratification context of the individual, the meaning of specific stressors changes and perception of stress may or may not be used to affect physiological outcomes. This model was considered to be particularly important in widening the scope of stress-disease research, which had focused primarily on a stimulus-response paradigm.
Retrospective studies of the occurrence of illnesses over quite long time periods (eg. 20 years) in someone's life have shown that illnesses do not appear to be distributed at random but often appear in clusters. Furthermore, these clusters appeared most often when a person was experiencing difficulties in adapting to various negatively rated life changes. Many different negative life events were found to be associated with an increase in the occurrence of major and minor illnesses. The association between negatively rated life events and illness is interesting and important but, again, it should not necessarily be thought of as causal. It might be the case that a third and as yet unknown factor could account for both the problems in adapting to life events and the susceptibility to illness. For the present, the most general interpretation of these findings is that negatively appraised life events will result in physiological and psychological changes, which in turn will directly or indirectly give rise to disease.

Most of the evidence reported have shown that there is an association between various psychosocial "stressors" and the onset of disease. These studies have provoked many criticisms and have also given rise to a number of further questions. The most obvious of these are concerned with the nature of the mechanisms which increase vulnerability of disease as the result of stress. From the outset it is probably fair to say that, as yet, medical science does not
have a clear answer to these questions and until such an answer exists, research evidence in this field will always be problematic.

The term "somatisation" was first used by Steckel (1943) who defined it as a bodily disorder that arises as an expression of a deep-seated neurosis, a method by which the body was translating into physiologic language the mental troubles of the individual. In the last two decades the term "somatisation" has also been used to describe the presentation of somatic symptoms that are not caused by physical disease or tissue damage, although, specific physiologic changes can be detected in some of these symptoms by special techniques (Kellner 1985). Somatisation indicates "one or more physical complaints, e.g., fatigue, loss of appetite, gastrointestinal or urinary complaints" and either "appropriate evaluation uncovers no organic pathology or pathophysiologic mechanism to account for the physical complaints" or "when there is related organic pathology, the physical complaints or resulting social or occupational impairment is grossly in excess of what would be expected from the physical findings". The term does not include conversion symptoms. Also it does not include psychosomatic disease in which tissue damage occurs such as peptic ulcers, bronchial asthma. Somatisation constitutes the core of somatoform disorder (Oxman, Harrigan & Kues 1983, Lipowski 1988). It is best to view somatisation as a continuum on which increasing levels of somatic symptoms are indicative of increasing distress, disability and maladaptive illness behaviour.
(Katon, Lin, von Korff, Russo et al. 1991). However, bodily complaints may be only one aspect of a psychiatric syndrome (such as depression) or of those who have been referred only after negative physical investigations (Mayou 1976).

There is a consistent association of somatisation with anxiety and depression, yet there are large differences among individuals in the ratios of somatic symptoms to affective ones. The interaction of emotion with bodily symptoms is complex: anxiety and depression frequently cause somatic symptoms; conversely, severe somatic symptoms may induce concerns, deprive a person of pleasure and, like other suffering, aggravate depression. At times of anxiety or depression, a person evaluates his/her somatic symptoms as having more ominous portent than at other times. Most of the bodily symptoms do not take place of emotions, they are not conversions but coexist with, result from, and may reinforce emotions. A common mechanism in somatisation, appears to be an affective disorder inducing somatic symptoms, followed by selective perception of bodily symptoms, motivated by fear of disease or other concerns, and a subsequent increase in anxiety with more somatic symptoms, forming links in a vicious circle. Anxiety and depression can be thought to be precipitating events for somatisation because both the disorders can cause distressing somatic symptoms. They can also serve as maintaining factors because bodily symptoms are evaluated in a fearful or pessimistic way at times of anxiety and depression. Somatic symptoms in turn may aggravate anxiety and depression.
because fears and false beliefs about bodily sensation can make a person more anxious and depressed. Since there is an intricate interaction and comorbidity of somatisation with anxiety and depression, it is best to analyse the effects of these affective components independently (Lader 1977, Ewald, Rogne, Ewald & Fink 1994, Iezzi, Stokes, Garnett, Adams, Pilon et al. 1994).

Several researchers (Ammon 1978, Lefebvre 1980, Nguyen 1982, Katon, Ries & Kleinman 1984, Kellner et al. 1985, Chaturvedi & Upadhyaya 1989, Katon & Russo 1989, Brown, Golding & Smith 1990, Lipowski 1990, Mumford, Bavington, Bhatnagar, Hussain et al. 1991, Simon, von Korff 1991, Tomasson, Kent & Coryell 1991, Budd 1993, Viinamaki, Koskela & Niskanen 1993, Kirmayer, Robbins & Paris 1994) have shown the frequency of association between somatisation, emotional distress and depression. Research evidence for this association has been consistent regardless of the design of the study; depressed patients tend to have more somatic symptoms than non-depressed individuals, somatisers tend to be more depressed than patients with physical disease and within groups there is a consistent positive correlation between depressive and somatic symptoms (Cadoret et al. 1980, Katon et al. 1982, Kellner 1986). Correlational studies show a consistent and robust association of cognitive depression and somatic symptoms regardless of whether the studies were carried out in depressed patients, medical patients or normals (Kellner et al. 1985).
However, the association between somatisation and depression need to be viewed with caution (Lipowski 1988). This is due to the fact that some depressed patients are more or less convinced that their physical symptoms are due to somatic illness and steadfastly deny being emotionally depressed at all or only by the symptoms, while the majority seem to be aware of both bodily and affective distress but are unsure which of them is primary (Katon, Kleinman & Rosen 1982).

Other studies to determine the association between somatisation and depression have given contradictory results. In a study conducted on samples of "somatisers", "psychologisers" and normal controls recruited by epidemiological procedures the scores of the two groups on latent traits for depression indicated that between the two groups, somatizers as a group were less depressed (Bridges, Goldberg, Evans & Sharpe 1991). In another study where the association between chronic fatigue and somatisation was sought, the results indicated that mood disorders were less common in subjects with somatisation than those without this diagnosis (Manu, Lane & Matthews 1989). Another study by Wise & Rosenthal (1982) investigated the relationship of affective status to abnormal illness behaviour and severity of illness in 88 patients between the ages of 16-67 years. Results showed that there was no relationship between depression and the actual severity of illness and abnormal illness behaviour.
A variety of theories has been applied to the problem of the causal relationship between pain and depression (Romano & Turner 1985). Rudy et al. (1988) found that depression was the consequence of the extent to which increases in pain severity interfered with important life activities thereby limiting social rewards and reducing the patient's sense of control or personal mastery. However, studies by Garron & Leavitt (1983) and Kerns et al. (1983), show psychogenic pain and depression exist as separate phenomena and despite their capacity for mutual influence they are best seen as independent processes. Other studies also show that some patients fail to recognize affective distress and instead somatise their complaints using both pain and non-pain somatic complaints (Katon et al. 1982). A study by France et al. (1987) had established patients with chronic pain and depression and also depression with no pain complaints. A recent study by Aydin (1997) indicated a significant relationship between negative automatic thoughts and the occurrence of physical symptoms of illness. This confirms the expectation that depression symptoms may be expressed in physical illness but does not point to a causality between symptoms of physical illness and depression. Hence, from these above mentioned studies it may be deduced that if one accepts the notion of a somatisation trait (Escobar, Burnam, Karno et al. 1987, Escobar 1987) then those possessing it will tend, when depressed, to report primarily physical symptoms. Others may choose to present such symptoms not because they do not experience depressed mood but because they do not wish to be given
psychiatric diagnoses and hence be stigmatized (Prestidge, Lake 1987) or because they believe it inappropriate to discuss psychosocial issues with general practitioners (Parker, Abeshouse, Morey et al. 1984).

To sum up, it may be said that though previous research have highlighted the association between affective distress, somatisation and depression, other studies have given contradictory results, thereby paving the way for further research in this area. To remove these contradictions a need is felt to determine the extent of depression in an individual that may correspondingly contribute to the somatisation of affective distress.

Several authors have described somatisation as a "masked" depression (Oxman, Rosenberg, Schnurr and Tucker 1985, Fisch 1987, Westermeyer, Bonafuely, Neider & Callies 1989) or a depressive equivalent and others (Blumer & Heilbronn 1982) regard idiopathic chronic pain as a variant of depressive disorder or a "masked" depression. Another study (Tomasson, Kent and Coryell, 1991) reveals that depression is a common clinical phenomena and most depressives report with some somatic complaints in addition to affective and cognitive ones. The proportion of masked depressions is positively correlated with patient's tendency to somatise and negatively correlated to doctors ability to recognize depression that hide behind somatic complaints (Fisch 1987). However, there is no conclusive evidence that somatisation is a true depressive equivalent i.e. it has the same etiology course and response to
treatment as depression. This view of "masked" depression has been cogently criticized on the grounds that it lacks empirical support (Turk & Salovey 1984, Romano & Turner 1985, Large 1986). It generally implies a disturbance of affect, which is considered to be a central but guarded issue for some somatising patients. Usually some depressed patients emphasise somatic symptoms for various reasons, thereby they prevent revealing that they feel depressed. However, a strong correlation has been found between self-assessments of health and depressive affect (Oxman, Rosenberg, Schnurr & Tucker 1985, Lipowski 1987, Vassend 1989). Manifestation of grief are expressed principally in the somatic realm – physical responses to bereavement and mechanisms responsible for the transformation of grief into physical symptomatology and illness (Lewis 1988). As far as somatisation patients are concerned, in their language is indicated a confused negative self-identity which suggests that their somatic complaints serve a more complex internal and interpersonal function than that indicated by bodily preoccupation and depression (Oxman, Rosenberg, Schnurr & Tucker 1985). Functional somatic symptoms may occur after losses and stressful life events (Kellner et al. 1983) these individuals may respond to stresses largely with somatic symptoms rather than with overt emotional ones, but in most of these patients some affective symptoms are present.
In summary, it may be said that though numerous studies have been conducted on the association of "masked" depression with somatisation, many contradictions still prevail. This is due to the fact that a certain proportion of patients complain mainly of somatic symptoms thereby "masking" the underlying affective state that contributes to somatisation. Hence further research in this area is required to establish whether such a causal relationship exists at all.

Along with affective distress and somatic complaints there patients also complain of dysphoria and a feeling of being "emotionally low". Studies on mood states of somatic patients have come to the conclusion that moods are the most consistent predictors of later physical symptom (Leventhal, Hansell, Diefenbach, Leventhal et al. 1996).

Ironically, some somatising depressed patients may be aware of their low mood but do not report it. Others are either unable to label and report the cognitive aspects of emotions or attribute their mood to subjectively experienced bodily distress (Lipowski 1990). Clinical depression is regarded by many as a disease, however there is no consensus regarding exactly how depression should be distinguished from unhappiness, many regard the clinical state as the extreme end of a continuum (Arieti & Bemporad 1978, Kendell 1976). Depression may be a cause of somatisers' complaints, but they do not complain of sadness, but of weakness, fatigue or somatic manifestations of depression (Oken 1984). Various correlational studies on depression and somatisation
reveals that depressed people usually report significantly more physical complaints, increased disability, functional limitations, increased use of health care services and a variety of physical complaints in all organ systems (Betrus, Elmore & Hamilton 1995). However, in contrast somatisation patients usually accept the presence of dysphoric affect in themselves. They occupy a sick role, consider themselves psychologically unwell, avoid blame for mental illness and feel less depressed than they might as sufferers of mental illness (Goldberg & Bridges 1988, Ewald, Rogne, Ewald & Fink 1994), while others believe that lowered mood and increased impairment are related to a greater number of somatic complaints (Bacon, Bacon, Atkinson, Slater et al. 1994). Usually, the paralysed dissatisfaction with one's life is generated through moods for different people and consequently these negative moods manifest themselves in physical symptoms (Budd 1993). Another study of patients with depression and somatisation reveal differences between the two groups in the sense that it is found that patients with depressive disorder show more dysfunction in the cognitive area whereas patients with somatisation show dysfunction in vocational, personal and familial areas (Chadda, Bhatia, Shome & Thakur 1993, Wetzel, Guze, Cloninger, Martin et al. 1994, Scicchitano, Lovell, Pearce, Marley et al. 1996). A recent study on negative affect (NA) comprising of depressive affect (DA) and anxious affect (AA) brought about interesting results. The study showed that NA and its two constituent variables predicted somatic complaints associated with acute illness. Interestingly, AA and not DA
were associated with subsequent somatic complaints (Leventhal, Hansell, Diefenbach, Leventhal et al. 1996).

To sum up: Studies so far reported show several contradictions in the obtained results. However, majority of the depressed people usually report of somatic symptoms, while somatisation patients emphasize more on dysphoric affect. Somatic complaints that reflect somatisation in depressive disorders may have different origins (Blacker, Clare 1987). Some are vegetative symptoms concomitant with depressed mood, while others may be either somatic metaphors with which patients communicate their emotional distress or exacerbations of coexistent physical illnesses. The depressed mood may also influence cognition in the direction of augmented perception of bodily sensations and their interpretation in terms of physical illness. Recent experimental work (Croyle, Uretsky 1987) indicates that such a mood fosters illness related memories and a negative view of one's health. Hence, the question that still remains unanswered is that whether somatic symptoms or dysphoric affect has a direct relationship with the onset of somatisation.

Somatisation may arise from many precipitating factors (Mallouh, Abbey & Gillies 1995). Precipitating factors include life events and situations that are personally stressful because of their subjective meaning of loss or threat for the individual. Bereavement, physical illness or injury, breakup of a relationship, and witnessing death appear to be common precipitants (Kellner 1986).
events usually are relatively recent and are of acute onset in cases of somatisation. Psychophysiologically, psychological states such as hopelessness, helplessness following stress can lead to increased levels of cortisol which reduces the efficacy of the immune system breakdown of the coping mechanism and resultant somatisation (Eysenck 1995). In another study it was found that high-risk individuals respond to excessive stress, tension and denied dependency needs through "acceptable" disability (Weinstern 1978). It is considered that dependency and self-criticism along with self-devaluation are important dimensions in depression. They represent different presentations of both clinical and normal depression as well as aspects of character that predispose individuals to the development of depression following stressful events. Results support the buffer-hypothesis that social support protects against the development of mental disorders only when the individual is exposed to stressors like negative life events. This buffering effect was especially strong for depression (Dalgard, Bjork & Tambs 1995).

Other studies on personality aspects of somatisation patients have established the fact that at times these patients show an inability or an unwillingness to express emotional feelings (Hollingshead & Redlich 1958). There is evidence to suggest that the inhibition of emotions and emotional expression particularly of hostility and anger is associated with physiologic arousal and somatisation (Gringes & Dawson 1978, Pennebaker 1985). In a
clinical study depressed women who appeared to hold in their anger were judged to be more prone to somatic symptoms than those who were not angry (Harris 1951). In a study of several populations, somatic symptoms were positively correlated with self-rated anger-hostility scales and negatively correlated with feelings of friendliness, somatic symptoms however tended to be associated more strongly with symptoms of anxiety and depression than with those of hostility (Kellner et al. 1985). Hostile subjects show greater change in skin conductance (Hokanson 1961), so the physiological concomitants of hostility could be pathways by which anger produces somatic symptoms. Therefore the results suggest that depression is the link between inhibited anger and somatisation (Kellner, Hernandez & Pathak 1992). Anger and hostility cause arousal with physiological over activity, they may be concomitants of certain kinds of depression (Fava et al. 1982, Overall et al. 1966, Weissman et al. 1971) or the consequences of chronic distress. Although anxiety and depression appear to be more common causes of somatization, anger particularly restrained anger probably plays a major part in a few. There is also evidence that repression of threats is associated with increased autonomic arousal (Hare 1966, Parsons et al. 1969). Deliberate suppression of facial expressive responses is generally associated with autonomic arousal but some results have been conflicting (Lanzetta et al. 1976, Pennebaker 1985). A survey of studies suggests an association of repression, denial, the inhibition of emotion and the lack of confiding on one hand and physiological arousal,
somatic symptoms and the incidence of disease on the other (Pennebaker 1985), however a few of the findings have been inconsistent.

The term 'alexithymia' (Sifneos 1973) describes a trait common in patients with somatoform disorders. Alexthymic patients have difficulties in expressing emotions in words, they do not have fantasies expressive of feelings, and their thought content is dominated by details of events in their environment (Nemiah 1977). There are a few empirical studies that link alexithmia to somatisation and to psychosomatic disorders. The trait is more common in somatising patients than in patients with psychosomatic diseases (Shipko 1982) but it is not limited to the former, it has also been found in other psychiatric patients as well as in medical patients (Freyberger 1977, Lesser et al. 1979, Lesser & Lesser 1983). Somatisation is associated with certain personality traits such as neuroticism, but little is known about the relationship of alexithymia to these traits.

Alexithymia is a behaviour that probably has various roots. In some patients it appears to be the manifestation of a state. In others it appears to be predominantly a cognitive style and people with this trait would attend to somatic distress rather than psychological events. Alexithymia appears to be one of the several processes that are involved in the ways people orient to and interpret bodily sensations which define their magnitude and meaning. In some patients it
is perhaps a manifestation of repression or denial or reflects an inability to become aware of their emotions.

In summary it may be said that life events and their appraisal as well as certain personality traits play a crucial role in the presentation of somatic symptoms. In certain cases it seems that there is a direct relationship between negative life events and personality traits and the production of corresponding somatic symptoms. Though numerous research work has been conducted in these areas, they also reveal many contradictions. Hence further study has to be taken up with a view to reducing the contradictions present in the previous studies.

Somatosensory amplification i.e. the tendency to experience bodily sensations as intense, noxious and disturbing may serve as a pathogenic mechanism for many psychiatric disorders with somatic features. It also explains the variability in somatic symptomatology (Barsky 1992). Some somatoform disorders are mainly characterised by somatic symptoms, somatosensory amplification plays an important role. Developmental learning factors appear to be crucially important to explain somatisation as learned abnormal perceptions or as a cognitive style. Learning to focus attention on somatic perceptions, to interpret them as threatening, to express them verbally and nonverbally, and to use such communication as an idiom of psychological distress and needs take place in the family context (Mechanic 1980, Ford 1983, Hartvig, Sterner 1985,
Kellner 1986, Shapiro, Rosenfeld 1986, Kriechman 1987). Since learning takes place from an early age, events in adult life can contribute to the precipitation of symptoms as well as to their maintenance (Kellner 1985). Due to this, somatosensory amplification results in social and vocational disability and is closely related to three measures of dysphoria – depression, anxiety and hostility. This tendency to amplify bodily sensations by somatisation patients may be an important factor in experiencing, reporting and functioning with an acute and relatively mild medical illness (Barsky et al. 1988), thereby resulting in exacerbations and exaggerations of mild somatic symptoms.

In spite of the previous mentioned studies on somatosensory amplification and its ill-effects, the question still remains, whether somatisation has to be differentiated psychopathologically, because it is a neurobio-logical core symptom of melancholia because of the perception of abnormal somatic symptoms or whether it is a culture bound symptom in the sense of being abnormally concerned with somatic symptoms.

Thus, it may be concluded that though quite a good number of research works has been conducted on various aspects of depression and its contribution to the production of somatic symptoms the studies reveal contradictions. Hence to undertake a study on the extent of depression that may contribute to somatisation more studies on these aspects of depression ought to be undertaken and is deemed necessary to reach definite conclusions.
Anxiety may be defined as a complex emotion that is accompanied by a wide variety of psychophysiological changes, many of which underline symptoms in patients (Lader 1977). However must patients show a mixture of such somatic symptoms with psychological symptoms as a result of which Mayou (1976) regarded anxiety as a reaction ranging from normality to totally disabling severity.

Spielberger (1966, 1972, 1985) considers anxiety a personality trait that determines how external or internal stimuli are appraised. In other words, the appraisal of threat in Spielberger's model will be a function of one's level of trait anxiety. He distinguishes trait anxiety from what he terms state anxiety, considering the latter to be a transitory emotional state. The personality trait of anxiety may lead the person to be anxiety-prone. According, to Spielberger's state-trait model, if the individual frequently experiences state anxiety then she has a strong trait anxiety. This model enables one to distinguish between common anxiety reactions and more intense and consistent anxiety. The model also supports to some extent Barlow's (1988) notion of the “anxious personality” being more vulnerable to triggers that produce acute anxiety or panic. However, the causes of individual differences in trait anxiety remain unexplained.

Psychodynamic theorists postulate that anxious feelings arise from internal conflicts or impulses of which the individual is not consciously aware. Thus origin of anxiety is in the unconscious and the feeling is triggered in the
present by a variety of associations. **Freud in 1926** saw anxiety as the psychic reaction to danger. A situation can be defined as dangerous if it threatens a person with helplessness in the face of hazard. Anxiety is self defeating or pathological when it is noticeable, intense, disruptive and paralyzing or when it triggers self defeating defensive processes. **Bowlby 1973** proposes that individuals have an innate propensity to make emotional bonds: once attachment is made it determines our emotional state. If the attachment is a secure one and we do not lose it at a vulnerable stage, then we are more likely to be healthy emotionally. More recently **Bowlby (1999)** states that change can go throughout the life cycle. **Eysenck (1976)** advocated the interactive role of learning and conditioning in the formation and expression of biologically based or innate emotions such as anxiety.

Psychological and social factors are major determinants of the presentation of illness to doctors. They may also have a primary role in the onset and course of physical illness.

The psychological precursors of illness are:

**Personality traits** — Clinicians and research workers continue to try to identify personality traits that predispose to illness. The chief difficulty with this approach is that the links almost always have to be made retrospectively. Most interest has focused on type "A" personality — a group of traits characterized by
impatience, hostility and time urgency. Type "B" personality traits are thought to encourage the appraisal of situations as more stressful or to prompt behaviours that produce elevated sympathetic and neuroendocrine responses.

**Life events** – More recently research has concentrated on the nature of the situation or event preceding the onset of illness along with long term personality factors that might render an individual especially sensitive to that situation. Current theories propose a series of chains of circumstances each linking the external situation to the particular disorder via the specific characteristics and responses of the individual.

**Proposed model linking external stressors (life events) to a disorder via specific predispositions**

**Model 1**

Social roles & long term personality dispositions → Life events difficulties → Emotional response → Psychological disorder

If the emotional response to life events is of sufficient intensity the disorder may be "functional" and involve less organic pathology. The most persuasive evidence for such a link is the association between, stress and the onset of a variety of "functional" illnesses and the recognition that the stressors involved are very similar to those implicated in the onset of emotional disorders.
Model 2

Proposed link between life events, psychological distress and the perception of bodily sensations

Events & Difficulties \(\rightarrow\) Emotional Response \(\rightarrow\) Increased sensitivity to and awareness of physical abnormality / pain without gross organic damage

In another model, it is less the emotional response and its intensity than the physiological concomitants of this response leading to organic changes that play the critical intervening role in bringing the subject into treatment.

Model 3

Proposed link between life events, psychological distress and tissue damage e.g. mental stress can promote ventricular ectopy

Events & Difficulties \(\rightarrow\) Emotional Response \(\rightarrow\) Physiological Concomitants \(\rightarrow\) Changes/Damage to bodily tissues

Though these models are crude, they illustrate the crucial interaction between social factors, personality dispositions and life events that influence the experience and presentation of illness. There is also strong evidence for the effects of psychological and social variables on the course and recovery from illnesses. There is little doubt that emotional factors can exert a powerful influence on the onset and course of illnesses. In some instances these may outweigh the extent and influence of any coexisting physical disorder.
Research evidence shows stressful events can enhance or perpetuate pain or reduce the individual's capacity to tolerate pain (Sternbach 1974, Weisenberg 1977). Sternbach 1996b reported that daily life "hassles" are strongly associated with increased pain. Emotional stress may increase pain by precipitating activity in the psycho-physiological system. Anxiety, depression, anger and other emotions provoke substantial autonomic, visceral and skeletal activity. The pain-anxiety-tension cycle is activated (Keefe & Gil 1986). The greater the anxiety the greater the perception of events as painful appears warranted (Weisenberg 1977, Sternbach 1986b). Anxiety disorders in children and adults are accompanied by an increase incidence of somatic symptoms and pain complaints (Beidel et al. 1991). However a clear empirical basis for the simplest proposition that anxiety increases pain is not available. Arntz et al. (1991) reviewed studies that varyingly indicated that anxiety enhances, relieves or has no impact on pain. Contradictions in findings concerning the relationship between pain and anxiety may reflect difficulties is clearly defining and measuring both pain and anxiety, response biases as people become less or more willing to complain of pain when anxious (Malow et al. 1989) and the moderating role that attention has on both anxiety and pain (Arntz et al. 1991). There is also the issue of the direction of causality. Does the high level of anxiety make the experience of pain more severe or does a high level of pain provoke high levels of anxiety? Thus it can be said that pain and resulting anxiety can contribute to further deterioration. Emotional distress serves not only
as a component of psychological pain but it may be a consequence of pain; a cause of pain or a concurrent problem with independent sources, all possibilities deserve attention (Feurerstein & Skjei 1979). There is also debate and confusion whether emotional processes should be conceptualized as causes or consequences of pain (Beutler et al. 1986).

Research evidence (Russo, Katon, Sullivan, Clark et al. 1994) shows that severity of somatisation was greater for subjects with greater number of current and past anxiety. Numerous authors have described somatic symptoms in anxiety. Several have expressed the view that somatic sensations are mainly the bodily manifestations of anxiety states.

As with depression, the association of anxiety and somatic symptoms is consistent (Tyrer 1976) and this applies also to studies in which the anxiety or depression scale no somatic items, thus the correlations were not spurious (Kellner et al. 1972). Somatic symptoms are invariably more numerous in patients with anxiety disorders than in normal subjects. Correlation studies show robust positive correlations of anxiety symptoms and somatic symptoms in patients as well as in normal subjects (Kellner 1988). The study of the relationship of somatic symptoms on the one hand and anxiety and depression on the other is complicated by the coexistence of anxiety and depression in a large proportion of patients (McNair & Fisher 1988, Von Zerssen 1986). In a correlational study with neurotic patients, somatic symptoms were somewhat
Studies highlighting personality factors of patients with somatisation have been very scarce. Self concept refers to the composite of ideas, feelings and attitudes people have about themselves (Hilgard, Atkinson & Atkinson 1999). Self concept is our attempt to explain ourselves to ourselves, to build a scheme that organizes our impressions, feelings and attitudes about ourselves. But this model or scheme is not permanent, unified or unchanging. Our self perceptions vary from situation to situation and from one phase of our lives to another. Psychiatric patients in general have reported to possess an external locus of control (Steinhausen 1982), however the cognitive aspects of patients play an important role in terms of internality / externality. But patients who view life events as primarily due to circumstances beyond their control are more likely to report maladaptive pain and psychological distress than those having internal control (Crisson & Keefe 1988). Peterson & De Avila (1995) reported that patients with optimistic explanatory cognitive styles, who explain bad events with specific causes, were less at risk and more able to prevent health problems. Internal show better psychological adjustment and physical wellbeing compared to externals (Strassberg 1973). However, encountering failures or other negative outcomes in life lead internals to harbour strong feelings of personal responsibility to get depressed and show health problems (Phares 1972).
144, 17 year old students completed a battery of measures, including the STAI to assess anxiety and behaviour control in coping with stressful situations. Highly anxious subjects reported low behaviour control and a greater tendency to use an escape coping strategy (ESCS) in stressful situations. Girls had a significantly higher level of trait anxiety than did boys. The greatest difference in anxiety levels was in situations provoking unrealistic anxiety. Boys had a higher level of behaviour control in situations of physical injury caused by accident, animals or dangerous people. Girls performed significantly higher control situations involving possible punishment by parents. Anxiety was related to ESCS and was used by girls with higher trait and state anxiety level. The choice of ESCS used by girls varied with the situation (Prokopcaková 1992).

A study was conducted to determine the relationship between locus of control and SES by exploring whether LOC reflects real resources or internal strengths and by investigating the relationship with depression. Data were obtained from a stratified random sample of 150 Israeli widows under the age of 54 years. Results indicate that both LOC and SES are related to depression and life situations independently. Internal LOC seemed to be crucial in decreasing depression and increasing life satisfaction in general. Subjects with internal LOC were more task and achievement oriented, less complaint and less conforming to social influences (Landau 1995). A study reveals high beliefs in lack of internality is associated with higher levels of depressive symptomatology. High
scores on chance and powerful others scales were uniquely associated with higher scores on measures of depressive symptomatology (Presson & Benassi 1996).

Dalgard, Bjork and Tambs 1995 study results support the buffer hypothesis that social support protects against the development of mental disorders only when the individual is exposed to stressors like negative life events. This buffering effect was especially strong for depression and applied to subjects with an external LOC orientation. Studies (Bates & Rankin-Hill 1994) show that an increased sense of control may contribute to an increased ability to cope successfully with the chronic pain experience. It is suggested that it may be possible to alter patients sense of control through the development of deliberate culturally appropriate and personally relevant programmes that help establish a sense of control over their lives and pain.

Self esteem is our evaluation of our own self concept. The developing of self concept of the child is influenced by parents and other family members in the early years and by friends, schoolmates and teachers as the child grows. The self concept evolves through constant self evaluation in different situations (Shavelson & Bolus 1982).

In terms of self-perception, people experiencing emotional distress have lowered self concepts and lack of competence in dealing with emotional
problems resulting in reduction of physical, mental and social functioning (Ezeilo 1983). High self esteem is associated with internal locus of control (Abdallah 1989) while external locus of control and low self esteem shows the highest psychological maladjustment (Kliwer & Sandler 1992). Contradictorily, Tómasson & Kent (1994) concluded that some somatisation disorder patients have stable self concepts and show no difference in social relations when compared to patients with conversion disorder.

A study investigated levels of self esteem and self reported depressive symptomatology in 42 children hospitalized for medical symptoms. Fifteen children (aged 8-14 years) with a somatoform disorder, 14 children (average age 11-41 years) with a depressive disorder and 13 children (average age 9-91 years) with no DSM-III-R diagnosis completed the Childrens’ Depression Inventory and the Piers-Harris Children's Self-concept Scale. Significant differences between the somatoform groups and the other groups were found for behavioural self-esteem (Kronenberger, Laite & LaClave 1995). Kellner, Hernandez & Pathak's (1992) study reveals depression to be the link between inhibited anger and somatisation. Philpot, Holliman, Bruce & Madonna's (1995) study reports strong intercorrelations between negative self statements and self-esteem. A study (Ezeilo, 1983) administered the Tennessee Self Concept Scale to 30 male undergraduates suffering from severe anxiety and somatic symptoms and to 47 normal subjects. Results confirm findings from
other cultures that indicate that people who experience emotional distress also have lowered self concepts which is probably related to lack of competence in dealing with the emotional problem and consequent reduction in physical, mental and social functioning.

In sum, it can be said that many psychological factors contribute conjointly in the etiology of somatisation disorder. No particular factor gains precedence and each makes its own contribution. Research work in this field needs to be made extensively, in order to arrive at definite and accurate conclusions.

**General Summary of the previous literature and implications of the present study**

To sum up, reviewing previous literature in the area of somatisation, an impression can be drawn that there is much lack of comparable data, i.e. lack of similarity in the techniques employed, lack of homogeneity and error in sample selection and other methodological problems found in the previous studies, it is rather difficult to draw a picture regarding the psychological personality characteristics involved in the disease processes. The present study thus intends to measure the personality characteristics and arousal level of some such patients and to compare them with another similar group of patients i.e. the conversion disorder patients. Such a comparative study is required in view of the fact that both these groups are similar in various aspects, but their perceptions of the environment differ resulting into two distinct disease processes with
accompanying separate symptomatic features. Apart from this, in terms of the Indian context this study seems necessary because accurate diagnosis and differentiation of the patients in terms of their personality will lead to proper therapeutic aid to the patients.