CHAPTER - I

INTRODUCTION

Comorbidity has been the central concern of psychiatry since 1990s. The prevalence and extent of the comorbidity phenomenon has led to a very large literature documenting comorbidity across the range of psychopathology characterized in current classification system. The phenomenon poses a significant conceptual problem in both clinical and research work with psychopathology such as how to conceptualize cases that seem to exemplify distinct forms of psychopathology simultaneously, and how to choose cases to study when the typical case does not fit neatly into a specific diagnostic category. Previous research reviews (Clark et al., 1995; Mineka et al., 1998; Widiger & Sankis, 2000) have emphasized that comorbidity is a highly general phenomenon and clarified that no diagnostic grouping appears to be entirely “safe” from extensive comorbidity.

The concept comorbidity originated in literature on the epidemiology of medical diseases. The term comorbidity was coined in context of chronic disease (Feinstein, 1970) which refers to any distinct additional clinical entity that has existed or that may occur during the clinical course of a patient who has the index disease under study. Feinstein defined comorbidity as “any additional co-existing ailment”.

The comorbidity concept entered the psychiatric and psychological literature much later than 1970. Lilienfeld et al. (1994) indicated that the concept took root in the psychiatric and psychological literature starting around the late 1980s to early 1990s. Studies have documented extensive comorbidity across the entire spectrum of psychopathology (Clark et al., 1995; Kendall & Clarkin, 1992) and sparked numerous discussions on the meaning and implications of comorbidity in psychopathology.

In the epidemiological data on comorbidity, the main interest of the field is that a person who meets criteria for a specific mental disorder is much more likely to also meet criteria for other mental disorders than one would expect simply by chance. It shows that mental disorder constructs, as applied to groups of people, are correlated. The term comorbidity legitimately encompasses this correlational phenomenon—it allows people to have more than one diagnosis. The problem is that the term also encompasses the simple fact that a person who meets criteria
for a specific disorder could meet criteria for another disorder even if no correlation exists between the two disorders.

Some of the workers distinguish comorbidity in terms of cooccurrence and correlation. The distinction between co-occurrence and correlation is conceptually important, and a problem noted in recent literature is that the term comorbidity could legitimately refer to either phenomenon. Vella et al. (2000) mentioned that “comorbidity should be defined as two or more diseases, with distinct aetiopathogenesis (or, if the etiology is unknown, with distinct pathophysiology of organ or system), that are present in the same individual in a defined period of time.” This definition is closer to the idea of co-occurrence captured by Feinstein’s (1970) definition, although it also adds the provision that some evidence of underlying casual distinctiveness is needed for the term “comorbidity” to apply (Lilienfeld et al., 1994; Meehl, 2001). By way of contrast, in writing on the comorbidity of childhood psychopathologies, Lilienfeld (2003) restricts his use of the term to “covariation” among diagnoses across individuals rather than co-occurrence among diagnoses within individuals. None of the authors could be said to be wrong in the way they approach working with the term “comorbidity.” The problem is that the term itself is broad enough to encompass too many conceptually distinct phenomena.

A related concern emerging in recent literature relates to the prefix “co-” added to the word “morbidity.” That is, Feinstein’s (1970) definitions pertain to two diagnoses.

Yet the general tendency for mental disorders to be correlated means that three or more diagnoses are not uncommon. Should terms such as “trimorbidity” or “quadramorbidity” or multimorbidity be used to describe individuals with the requisite number of diagnoses? A number of commentators agreed with the need for this greater precision, but felt that abandoning the term “comorbidity” would be premature and even perhaps counterproductive. Similarly, Spitzer (1994) regarded comorbidity as a reasonable label for co-occurring entities that may not rise to the conceptual level of bona fide categories with clear cut etiologies and pathophysiologies—not only in psychiatry, but in medicine more generally. Maj (2005a, b) suggests that the fact that various mental disorders rarely occur in isolation could be viewed as evidence that comorbidity is an artifact of current diagnostic systems imposing categorical distinctions that do not exist in nature. Along these lines, Meehl (2001) suggest that the term
“comorbidity” would be most meaningfully applied to taxonic categorical conditions, where distinct and discrete latent structures underlie the two comorbid conditions. Bogenschutz and Nurnberg (2000) emphasize the importance of sorting through these issues to clarify thinking about diagnosis, noting that comorbidity among certain categorical mental disorders (e.g., major depression and posttraumatic stress disorder) may be better understood from a dimensional framework. Batstra et al. (2002) note that bivariate statistics such as odds ratios tend to be used in studying comorbidity among mental disorders, even though the phenomenon appears to be multivariate in nature and requires a statistical approach capable of mapping the concept of multimorbidity.

Indeed the basic phenomenon that has captured the interest of the field under the comorbidity rubric seems clear and important; mental disorders are significantly correlated. That is, meeting criteria for one disorder predicts meeting criteria for others.

COMORBIDITY OF ANXIETY AND DEPRESSION

Research on relationships between anxiety and depression has proceeded at a rapid pace since the 1980s. The similarities and differences between these two conditions, as well as many of the important features of the comorbidity of these disorders, are recognised.

Throughout the century, the anxiety and depressive disorders have been treated as separate diagnostic classes in official nosologies; many researchers have argued that these disorders are distinct entities (Akiskal, 1985; Cox et al., 1993). Nonetheless, other researchers have asserted that they represent a single underlying dimension, or together they form a more general class of mood disorders (Feldman, 1993; Hodges, 1990). This latter unitary construct view has been particularly prominent in Europe where even the term mood disorder subsumes both anxiety and depression. In turn, the former, dual construct, view may reflect a more general tendency on the part of American psychiatry toward diagnostic “splitting” rather than “lumping” (Frances et al., 1990).

More generally, researchers gradually have begun to realize that the controversy over the unitary versus dual models is both unnecessary and unproductive. These models increasingly are being replaced by a more nuanced view in which anxiety and depression are posited to have both shared, common components and specific, unique components (Clark & Watson, 1991c).
Another issue commonly raised is that the presence of the same symptom in two or more diagnoses (e.g. sleep disturbance in MDD and GAD) artifically raises the co-occurrence of the disorders. Excessive diagnostic splitting also has been noted as “cause” of comorbidity, usually with regard to highly similar disorders (e.g. overanxious disorder and GAD in children; Caron & Rutter, 1991). However, the separation of phenotypically diverse syndromes (e.g. MDD and GAD) into distinct categories may reflect the same phenomenon. Thus, the greatest challenge that the extensive comorbidity data pose to the current nosological system concerns the validity of the diagnostic categories themselves—do these disorders constitute distinct clinical entities?

**Symptoms Co-occurrence:**

The term comorbidity probably should be reserved to designate co-occurring disorders (or at least syndromes), but investigation of anxiety-depression comorbidity begins with the observation that key symptoms that define these theoretically distinct syndromes or disorders often co-occur. These symptoms can be divided into those that are unique to each type of disorder (e.g. panic attacks versus feelings of worthlessness) and those that are shared (e.g. difficulty concentrating). However, few of these symptoms clearly differentiate patients with one type of disorder versus the other (Clark, 1989). A similar picture is obtained at the syndromal level. Depression and anxiety frequently co-occur, especially in primary care settings. These co-occurrences manifest themselves in several ways and have different clinical courses. Hirschfeld (2001) suggested comorbid major depression and anxiety typically present as 1 of 4 clinical combinations (Figure 1). The patient may meet criteria for an anxiety disorder diagnosis, but suffer from only subsyndromal levels of depression symptoms (Figure 1, panel A). Alternatively, a patient may meet criteria for major depression, but suffer from only subsyndromal levels of anxiety symptoms (panel B). Thirdly, a patient may present with a full-fledged diagnosis of both an anxiety disorder and major depression (panel C). Finally, a patient may present with symptoms of both anxiety and depression, neither of which is severe enough to meet criteria for a diagnosis (panel D). This latter presentation is referred to as “mixed anxiety-depression.”
Figure 1: Depression and Anxiety Comorbidity: 4 Common Clinical Presentations

A. Anxiety Disorders With Depressive Symptoms

B. Major Depressive Disorder With Anxiety Symptoms

C. Coexisting Anxiety Disorder and Major Depressive Disorder
D. Anxiety Symptoms and Depressive Symptoms (subsyndromic).

Diagnostic comorbidity of anxiety and depression:

Lifetime diagnoses of anxiety and depression show extensive comorbidity. Clark’s (1989) meta-analysis, found that depressed patients had an overall rate of 57% for any anxiety disorder. Brady and Kendall (1992) reviewed anxiety and depression in children and adolescents including differential diagnosis, assessment of symptoms, family history data, developmental features, and clinical correlates. Findings indicated that 15.9% to 61.9% of children identified as anxious or depressed have comorbid anxiety and depressive disorders and the measures of anxiety and depression were found highly correlated. Lepine et al. and World Health Organization Field Trials (1993a) assessed lifetime and current comorbidity of anxiety and affective disorders in 543 subjects and revealed the strongest interrelationship with in anxiety disorders and moderate correlations of major depression with panic disorder, generalized anxiety disorder, agoraphobia, and obsessive compulsive Disorder. For the majority of Subjects with both anxiety and depression, depression followed anxiety.

The National Comorbidity Survey found a remarkably similar 58% lifetime prevalence rate (Kessler et al., 1996) and an only slightly lower (51.2%) 12-month prevalence rate. Social and simple phobias have both higher overall base rates and the highest rates of occurrence in depressed individuals, whereas panic disorder has the lowest rate in both cases. Nonetheless, the odds ratio (OR) in all cases far exceeds co-occurrence due simply to base rates (overall OR = 4.2; Kessler et al., 1996). Moras et al. (1996) reported a similar phenomenon but at lower levels
for strictly defined current comorbidity across 10 studies. As for depression comorbid with anxiety, the overall average is the same (e.g. 56% in Clark’s 1989 meta-analysis), but with rates widely varying by diagnosis: 67% in panic/agoraphobia, 33% in DSM-III GAD, 20% in social and simple phobias. The rates obtained for DSM-III-R GAD are considerably higher, however, in both the National Comorbidity Survey (NCS) data (62.4% for MDD, 39.5% for dysthymia; Wittchen et al., 1994) and the one study (73% MDD) reported in Clark (1989). The various anxiety disorders are also highly comorbid with each other (Brown & Barlow 1992; Brown et al., 1998), but anxiety disorders in the NCS were as or more comorbid with depression (M OR = 6.6) as among themselves (M OR = 6.2) (Kessler, 1997).

Studies investigated the presence of comorbid anxiety and depression disorders in children and adolescents with a depressive illness. Carey, Finch, and Imm (1989) reported a rate of 55.2% comorbid for anxiety and depression among hospitalized, depressed children. Kovacs et al. (1989) found the rate to be 31.5% in a sample of outpatients. The differing comorbidity rates between these two samples suggest that comorbidity is more frequent among more severely disturbed samples. Following debate over whether children even experience the symptoms associated with depression in adults (Cantwell & Carlson, 1983), there is general agreement that adult and childhood depression share some essential features (Cantwell & Carlson, 1983; Stark, 1990). Nilzon and Palmerus (1997) examined the relationship between anxiety and depression in school children. Results indicated that depressed children were generally anxious but were not identical to children suffering from overanxious disorder. Depressed girls were most likely to suffer from tension and somatic symptoms than depressed boys, who tend to be more generally anxious at this age. Findings supported the idea of depression and anxiety as comorbid concepts.

Depression and anxiety are however strongly associated. The onset of depression is often preceded by anxiety (Avenevoli et al., 2001; Cohen et al., 1993) and depression and anxiety are much more often comorbid than would be expected by chance (Angold, Costello, & Erkanli, 1999; Brown et al., 2001a; Mineka, Watson, & Clark, 1998; Williamson et al., 2005). Schatzberg and associates (1998) examined the temporal relationship between anxiety and depressive disorders in a cohort of patients with current major depression by using the Structured Clinical Interview for DSM-III-R Diagnosis. Results revealed 29% of the subjects met criteria for at least one current anxiety disorder and 34% had at least one anxiety disorder at some point in their
lives. In patients with major depression with comorbid anxiety disorders, both the social and simple phobia were more commonly reported to start at least 2 years prior to their major depression in contrast to depressives with comorbid panic or obsessive-compulsive. Thus, early-onset social and simple phobias are risk factors for later onset of major depression. Recently, Lamers et al. (2011) by examining comorbidity patterns and temporal sequencing of separate depressive and anxiety disorders in a large psychiatric cohort baseline data (N = 1,783) of the Netherlands Study of Depression and Anxiety reported that of those with a depressive disorder, 67% had a current and 75% had a lifetime comorbid anxiety disorder. Of persons with a current anxiety disorder, 63% had a current and 81% had a lifetime depressive disorder. In 57% of comorbid cases, anxiety preceded depression, and in 18%, depression preceded anxiety. Comorbidity with preceding depression compared to preceding anxiety was associated with a shorter duration of symptoms of depressive and/or anxiety symptoms (OR = 0.99), earlier age at first onset (OR = 0.46), and fewer fear symptoms (OR = 0.98).

Fava et al. (2000) examined the prevalence and clinical impact of anxiety disorder comorbidity in major depression among depressed outpatients and found that comorbid anxiety disorder diagnoses were present in 50.6% of these patients that included social phobia (27.0%), simple phobia (16.9%), panic disorder (14.5%), generalized anxiety disorder (10.6%), obsessive-compulsive disorder (6.3%), and agoraphobia (5.5%), social phobia and generalized anxiety preceded highly the 1\textsuperscript{st} episode of major depression in 65% and 63% of cases, respectively, than other anxiety disorders. Zimmerman et al. (2000) determined the frequency of anxiety disorders in depressed outpatients and reported that more than one-half of the patients met the full criteria for a current anxiety disorder, and more than one-half of the patients with an anxiety disorder had more than one. When partial remissions and anxiety disorder diagnoses classified as” not otherwise specified, two-thirds of the patients had a current anxiety disorder, and three-quarters had a lifetime history of an anxiety disorder. Ohayon et al. (2000) using the general population sample reported that overall, 13.2% of the sample had either a mood disorder or an anxiety disorder at the time of their interview. The prevalence was higher among women than among men. The comorbidity of mood and anxiety disorders was found in 3% of the sample. Goodwin (2002) by using data of community sample of adults reported that specific phobia [OR = 1.7 (1.6, 1.8)], agoraphobia [OR = 2.3 (2.2, 2.5)], obsessive–compulsive disorder [OR = 5.4 (5.0, 5.8)] and panic attack [OR = 1.9 (1.8, 2.1)] each made an independent contribution to the risk of major
depression. Each anxiety disorder and panic attacks appeared to confer an independent risk for the onset of major depression within 12-months among adults in the community. Moffit et al. (2007) reported the sequential and cumulative comorbidity between Generalized Anxiety Disorder and Major depressive Disorders. Anxiety began before or concurrently in 37% of depression cases, but depression began before or concurrently in 32% of anxiety cases. Cumulatively, 72% of lifetime anxiety cases had a history of depression, but 48% of lifetime depression cases had anxiety. During adulthood, 12% of the cohort had comorbid GAD-MDD, of whom 66% had recurrent MDD, 47% had recurrent GAD. The rate of comorbidity is particularly high for depression and anxiety.

Kessler (2005), in a major prevalence study involving 9,283 participants and all psychiatric diagnoses, found that 22% of those interviewed had 2 psychiatric diagnoses, and 23% had 3 or more diagnoses. Using data of community-dwelling adults in the United States from the National Comorbidity Survey-Replication public use dataset, Bellinda et al. (2009) found that more than half of respondents with a 12-month major depressive disorder (MDD) had a comorbid anxiety disorder or dysthymia. High rates of MDD were also found for those with anxiety disorders across both age groups, highest in the 18-64 years group for generalized anxiety disorder (28.5%) and highest in the 65 years and older group for panic disorder (36.7%). Onset of anxiety disorders preceded onset of depressive disorders for most of older adults (77.6%). Licanin (2011) analyzed the comorbidity between anxiety disorders patients with major depression in a clinical sample of a Bosnian population. Results reported that simple phobia was most prevalent in major depression (20.6%). The rate of panic disorder prevalence was 11.2 in major depression. Bipolar I patients showed a relatively low rate of comorbidity.

Other important features of comorbidity of Anxiety and depression:

In 1990, Alloy and Mineka and colleagues identified three “tentative” phenomena that any comprehensive theory of comorbidity in anxiety and depression should ideally be able to explain: (a) the sequential relationship between anxiety and depression, (b) the differential comorbidity of depression with various anxiety disorders, and (c) the relative infrequency of pure depression compared with pure anxiety (Alloy et al., 1990). These phenomena were labeled “tentative” because knowledge about comorbidity was relatively sparse at that time.
The sequential relationship between anxiety and depression has been observed both within episodes and across the lifetime. Within a single episode of illness, anxiety symptoms are more likely to precede depressive symptoms than the reverse (Alloy et al., 1990). Regarding lifetime comorbidity, Alloy et al. (1990) also reviewed evidence that an anxiety disorder is significantly more likely to precede a mood disorder than the reverse. This phenomenon is now well established. For example, in the NCS data, Kessler et al. (1997) found that all the anxiety disorder diagnoses are associated with an elevated risk of a later diagnosis of minor or major depression. The ORs were especially high for severe major depression (7-9 symptoms), ranging from 2.86 for social phobia to 12.87 for GAD.

Alloy et al. (1990) early review also suggested that individuals who received the diagnoses of panic disorder, agoraphobia, obsessive-compulsive disorder, and post-traumatic stress disorder were more likely to experience depression than were those with generalized anxiety disorder, social phobia, or simple phobia. The NCS data generally confirmed this conclusion through the use of OR that take into account the large base-rate differences for the different disorders (Kessler et al., 1996). Specifically, lifetime comorbidity data for major depression revealed OR of 2.9 for social phobia, 3.1 for simple phobia, 3.4 for agoraphobia, 4.0 for panic disorder and post-traumatic stress disorder, and 6.0 for GAD (results for obsessive-compulsive disorder are not presented). Thus, the one exception to the pattern observed by Alloy et al. (1990) was that GAD was the anxiety disorder most likely to co-occur with MDD. Alloy et al. (1990) also summarized evidence that cases of pure depression without concomitant anxiety were rarer than cases of pure anxiety without concomitant depression. The likelihood that someone with a mood disorder also will receive an anxiety-disorder diagnosis (either concurrently or subsequently) appears to be greater than the reverse. In the NCS data, 58% of those with a lifetime diagnosis of a depressive disorder also had an anxiety disorder (Kessler et al., 1996).

In order to understand unique features of comorbidity of anxiety and depression, the anxiety and depressive disorders given by the recognised classification systems of mental disorders should be understood first. Patients used in study were diagnosed as per diagnostic criteria of ICD-10 classification system of mental disorders. Therefore, anxiety and depressive disorders are described here as per diagnostic criteria of ICD-10 classification system.
Anxiety Disorders

Anxiety is multidimensional construct characterised by physiological, cognitive and behavioural responses (Lang, 1968). Importantly, anxiety is a normal emotional state that is experienced across the developmental spectrum and can be broadly defined as an emotional reaction to an actual or imagined danger (King, Hamilton & Ollendick, 1988). The term anxiety is used interchangeably with fear. In recent years, “anxiety” has been used to refer to emotional states such as doubt, boredom, mental conflict, disappointment, bashfulness, and feelings of unreality. Various cognitive deficits, such as lack of concentration, are also labelled as “anxiety”.

Symptoms of Anxiety Disorders:

There are several characteristic features or symptoms of an anxiety disorder diagnosis that are common across the diagnostic categories. These symptoms can be described as cognitive, physiological, and behavioural. Common cognitive symptoms include the presence of excessive or unreasonable fear or worry about performance or safety. Common physiological symptoms, which must occur immediately in response to the presence or threatened presence of the feared situation or stimuli, include elevated heart rate, blood pressure, muscle tension, sleep difficulties, restlessness, somatic complaints such as headaches or stomach aches, and feeling faint, dizzy, or nauseous. Common behavioural symptoms include avoidance, crying and tantrums, freezing, and clinging. All disorders require that the anxiety must have been present for a minimum duration of time, ranging from four weeks to six months.

ICD-10 Anxiety Disorders

The ICD-10 (International Classification of Diseases-10) is one of the most widely used systems for the current classifications of mental and behavioural disorders. In ICD-10 anxiety disorders are placed in block F40 - F48 (Neurotic, stress-related and somatoform disorders). Anxiety disorders are Phobic Anxiety Disorders (F40), other anxiety disorders (F41), Obsessive-compulsive disorder (F42), Reaction to severe stress, and adjustment disorders.

Phobic Anxiety Disorders (F40) consists of group of disorders such as Agoraphobia (F40.0), Social phobias, Specific (isolated) phobias, Other phobic anxiety disorders, Phobic anxiety disorder, unspecified. In this group of disorders, anxiety is evoked only, or predominantly, by
certain well-defined situations or objects (external to the individual) which are not currently
dangerous; As a result, these situations or objects are characteristically avoided or endured with
dread.

**Agoraphobia (40.0)**

The term “agoraphobia” refers to an interrelated and often overlapping cluster of phobias
embracing fears of leaving home: fear of entering shops, crowds, and public places, or of
travelling alone in trains, buses, or planes. Although the severity of the anxiety and the extent of
avoidance behaviour are variable, this is the most incapacitating of the phobic disorders and
some sufferers become completely housebound; many are terrified by the thought of collapsing
and being left helpless in public. The lack of an immediately available exit is one of the key
features of many of these agoraphobic situations. Most sufferers are women and the onset is
usually early in adult life. Depressive and obsessional symptoms and social phobias may also be
present but do not dominate the clinical picture.

As per ICD-10 Criteria, For the definite diagnoses of agoraphobia, the psychological or
autonomic symptoms must be primarily manifestations of anxiety and not secondary to other
symptoms, such as delusions or obsessional thoughts; The anxiety must be restricted to (or occur
mainly in) at least two of the following situations: crowds, public places, travelling away from
home, and travelling alone; and Avoidance of the phobic situation must be, or have been, a
prominent feature.

**Social Anxiety Disorder**

Social anxiety disorder [Social Phobias (40.1)] is characterized by fear of embarrassment and
humiliation in social and performance situations. Social Anxiety Disorder (Social Phobia) is the
most common type of anxiety disorder and the third most common mental disorder in the
population.

As per ICD-10 Diagnostic Criteria, for diagnosis of specific Phobias, “First, the psychological,
behavioural, or autonomic symptoms must be primarily manifestations of anxiety, and not
secondary to other symptoms such as delusions or obsessional thought; secondly, the anxiety
must be restricted to or predominate in particular social situations; and the phobic situation is
avoided whenever possible”. 

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Specific (isolated) Phobias (F40.2)

These are phobias restricted to highly specific situations such as proximity to particular animals, heights, thunder, darkness, flying, closed spaces, urinating or defecating in public toilets, eating certain foods, dentistry, the sight of blood or injury, and the fear of exposure to specific diseases. Although the triggering situation is discrete, contact with it can evoke panic as in agoraphobia or social phobias. Fear of the phobic situation tends not to fluctuate, in contrast to agoraphobia.

For diagnosis of specific Phobias, First, the psychological or autonomic symptoms must be primary manifestations of anxiety, and not secondary to other symptoms such as delusion or obsessional thought. Secondly, the anxiety must be restricted to the presence of the particular phobic object or situation; and the phobic situation is avoided whenever possible.

Other Anxiety Disorders:

Generalized Anxiety Disorder

Generalized Anxiety Disorder was known by many names. Freud called it free floating anxiety. In DSM-II nd it was called anxiety reactions. It is also labelled as trait anxiety”. The essential feature of a Generalized Anxiety Disorder (GAD) is anxiety, and apprehension (worry) which is generalized and persistent but not restricted to, or even strongly predominating in, any particular environmental circumstances. (i. e. it is “free-floating”). The focus of the apprehension may be future events, past events, performance, evaluation by others, or world issues.

As per ICD-10 Diagnostic Criteria, The sufferer exhibits primary symptoms of anxiety i. e. apprehension (worries about future misfortunes, feeling “on edge” difficulty in concentrating, etc.); motor tension (restless fidgeting, tension headaches, trembling, inability to relax); and automatic overactivity (lightheadedness, sweating, tachycardia or tachypnoea, epigastric discomfort, dizziness, dry mouth, etc.) most days for at least several weeks at a time, and usually for several months.

Panic Disorder

The idea of Panic Disorder has its roots in the concept of irritable heart syndrome given by “Jacob Mendas Dacosta”. In 1895, Sigmund Freud introduced the concept of ‘anxiety neurosis’,
Freud’s acute anxiety neurosis was similar to panic disorder. The essential features of panic disorders are recurrent attacks of severe anxiety (panic) which are not restricted to any particular situation or set of circumstances, and which are therefore unpredictable. Possible physiological symptoms of panic include sudden onset of palpitations, sweating, trembling, shaking, chest pain, choking sensations, dizziness, and feelings of unreality (depersonalization or derealisation). This invariably involves a secondary fear of dying, losing control, or going mad.

As per ICD-10 Diagnostic Criteria, for a diagnosis of PD, several severe attacks of autonomic anxiety should have occurred within a period of about 1 month, in circumstances where no objective danger happens; without being confined to predictable situations; and with comparative freedom from anxiety symptoms between attacks. PD may be diagnosed as occurring with or without Agoraphobia (anxiety about being in places or situations from which escape might be difficult or embarrassing, or help may not be readily available.

**Obsessive Compulsive Disorder (OCD)**

Obsessive Compulsive Disorder is an anxiety disorder characterized by either obsessional thoughts or compulsive acts or both. Obsessional thoughts are idea, images or impulses that enter the individual’s mind again and again in a stereotyped form. They are being violent, obscene, senseless cause distress, and the sufferer often tries, unsuccessfully to resist them. Compulsive acts or rituals are stereotyped behaviours that are repeated again and again. They are not inherently enjoyable, nor do they result in the completion of inherently useful tasks. Compulsive acts are commonly performed by the sufferer in response to an obsessional thought and as an attempt to neutralise the anxiety that the obsessional thought evokes.

As per ICD-10 criteria, the obsessional symptoms or compulsive acts, or both, are present on most days for at least two successive weeks and is a source of distress or interference with activities. They are, however, recognised as the individual’s own thoughts, even though they are involuntary and often repugnant; There must be at least one thought or act that is still resisted unsuccessfully, even though others may be present which the sufferer no longer resists; the thought of carrying out the act must not in itself be pleasurable; the thoughts, images, or impulses must be unpleasantly repetitive.
ICD-10 Depressive Disorders

Although broadly described as a mood disorder, depression is a multidimensional construct characterised by affective, cognitive, motivational, and behavioural symptoms. As far as the diagnosis of clinical depression is concerned, “clinical depression is diagnosed through the presence, duration, and severity of a set of symptoms such as depressed mood that lasts at least two weeks, changes in weight, changes in sleep and activity patterns, loss of energy, feelings of worthlessness, and recurrent thoughts of suicide or death” (Conger & Galambos, 1997).

Depression includes a variety of emotional, physiological, behavioural, and cognitive symptoms. The most common emotion in depression is sadness but rather than the usual type, it is a more sustaining and painful one. The patient loses interest in everything. This symptom is referred to as anhedonia. In depression many bodily functions are disrupted and changes in appetite, sleep, and activity level occur. Behaviourally, many depressed people are slowed down a condition called psychomotor retardation. The people suffering from depression lack energy and feel chronically fatigued. They cannot react to crisis as quickly as necessary to avoid them. Similarly, there is a subset of depressed people who exhibit psychomotor agitation. As far as the cognitive symptoms of depressed people are concerned, their thought patterns are often filled with themes of worthlessness, guilt, hopelessness, and even suicide. They usually have trouble concentrating and making decisions.

Depressive Episodes

The ICD-10 categorizes depression under the three categories of mild (F32.0), moderate (F32.1) and severe (F32.2 and 32.3) depressive episode. In all these three kinds of depressive episodes (i.e. mild, moderate, and severe), the sufferer usually shows the symptoms of depressed mood, loss of interest and enjoyment, and reduced energy leading to increased fatiguability and diminished activity. Marked tiredness after only slight effort is common.

However, some other common symptoms are also involved such as reduced concentration and attention, reduced self-esteem, and self-confidence; ideas of guilt and unworthiness, bleak and pessimistic views of the future, ideas or acts of self-harm or suicide, disturbed sleep, diminished appetite. For depressive episode of all three grades of severity, a duration of at least 2 weeks is
usually required for diagnosis, but shorter periods may be reasonable if symptoms are unusually severe and of rapid onset.

**Mild depressive episode (32.0):** Depressed mood, loss of interest and enjoyment, and increased fatiguability are usually regarded as the most typical symptoms of depression, and at least two of these, plus at least two of the other symptoms described above should be present, for a diagnosis of mild depressive episode. None of the symptoms should be present to an intense degree.

**Moderate Depressive Episode (32.1):** At least two of the three most typical symptoms (Depressed mood, loss of interest and enjoyment, increased fatiguability, and diminished activity) should be present, plus at least three or four of the other symptoms. Several symptoms are likely to be present at marked degree, but this is not essential if a particularly wide variety of symptoms is present overall.

**Severe depressive episode without psychotic symptoms (32.2):** In a severe depressive episode, the sufferer usually shows considerable distress or agitation, unless retardation is a marked feature. Loss of self-esteem or feelings of uselessness or guilt are likely to be prominent, and suicide is a distinct danger in particularly severe cases. It is presumed here that the somatic syndrome will almost always be present in a severe depressive episode.

**Severe depressive episode with psychotic symptoms (32.3):** A severe depressive episode with psychotic symptoms is a severe depressive episode which involves all the symptoms noted for severe depressive episode without psychotic symptoms, and in which delusions, hallucination, or depressive stupors are present.

**Other depressive episode:** These episodes do not fit the descriptions/criterion given for any of depressive episode, but for which the overall diagnostic impression indicates that they are depressive in nature.

**Recurrent Depressive Disorder**

The disorder is characterized by repeated episodes of depression as specified in depressive episode Mild, Moderate, or Severe, without any history of independent episodes of mood elevation and overactivity that fulfil the criteria of mania.

Recurrent depressive episode may be subdivided by specifying first the type of the current episode and then the type that predominates in all the episodes. Almost in every recurrent
depressive disorder, at least two episodes should have lasted for a minimum of 2 weeks and should have been separated by several months without significant mood disturbance.

I) Recurrent depressive disorder, current episode mild (33.0).
II) Recurrent depressive disorder, current episode moderate (33.1).
III) Recurrent depressive disorder, current episode severe without psychotic symptoms (33.2).
IV) Recurrent depressive disorder, current episode severe with psychotic symptoms (33.3).

Dysthymia is a chronic depression of mood. Basically this is a mild persistent depression. Sufferers usually have periods of days or weeks when they describe themselves as well, but most of the time (often for months at a time) they feel tired and depressed; everything is an effort and nothing is enjoyed. They brood and complain, sleep badly and feel inadequate, but are usually able to cope with the basic demands of life. Dysthymia therefore has much in common with the concepts of depressive neurosis and neurotic depression. It usually begins early in adult life and lasts for at least several years, sometimes indefinitely.

Besides the above distinctions, there is also a distinction within the depression between reactive and endogenous depression.

Reactive depression also known as exogenous depression, results from events occurring in one’s life. Some stressful event(s) or an uncontrollable loss such as death of a close friend, relative, or unemployment causes it. The event triggers an episode of depression.

Endogenous depression results from ‘internal’ i.e. physiological (e.g. hormonal imbalance) and psychological factors. The term is used clinically, when there is no apparent precipitant, although many prefer not to use it at all on the grounds that it implies that there are no precipitating events.

Comorbidity between Generalized anxiety and Depression

Kessler (1999) reported that the majority of respondents with generalized anxiety disorder at 12 months in the National Comorbidity Survey (58.1%) and the Midlife Development in the United States Survey (69.7%) also met the criteria for major depression at 12 months. Community
epidemiological studies using DSM-III-R and DSM-IV criteria continued to find strong comorbidity between GAD and MD (Grant et al., 2005; Kessler et al., 2005; Kessler et al., 1996). Attempts to explore the factor structure of comorbidity among Axis I disorders concluded that GAD and MD are both strongly related to a general “distress” factor (Krueger, 1999; Krueger et al., 1998; Vollebergh et al., 2001). However, longitudinal analysis showed meaningful divergence between GAD and MD both in risk factors (Moffitt et al., 2007).

The prospective evidence of somewhat different environmental risk factors for GAD and MD is consistent with the results of genetic epidemiological studies, which suggest that while the genes for GAD and MD are very similar or possibly even identical, the environmental determinants are less strongly related (Kendler, 1996; Kendler et al., 2007; Kendler et al., 1992; Roy et al., 1995). Taken together with the prospective evidence for differential risk factors, these data suggests that, despite strong similarities that might lead them to be placed in the same diagnostic category in the upcoming revisions of the DSM and ICD systems (Watson, 2005), GAD and MD are distinct disorders.

Epidemiological studies have consistently identified a high level of co-morbidity between DSM-IV major depressive episode (MDE) and generalized anxiety disorder (GAD) in large community-based samples (Judd et al., 1998; Hunt et al., 2002; Hunt et al., 2004; Grant et al., 2005; Kessler et al., 2005).

**Comorbidity of Panic Disorders with Depression**

The diagnosis of panic disorder rarely occurs in isolation, over half of individuals with PDA present with comorbid psychological disorders (60%, Brown et al., 2001a; 51%, Brown, Antony, & Barlow, 1995), and this has been a consistent finding. Common psychological disorders comorbid with panic disorder include other anxiety disorders (Goisman, 1995; Sanderson et al., 1990), mood disorders (Chen & Dilsaver, 1995; Lesser et al., 1988), substance use disorders (Cox, 1990), and personality disorders (Chambless et al., 1992; Diaferia et al., 1993).

Andrade et al. (1994) examined rates of comorbidity of panic attacks and major depressive disorder in a population based sample of 12,668 adults from 4 sites of the National Institute of Mental Health Epidemiologic Catchment Area Program. Data showed that the co-occurrence of Panic Attacks and Major Depression over the lifetime was 11 times higher than expected by
chance. Andrade and associates (1996) assessed Lifetime comorbidity of panic attacks and Major depression and its suspected effects of comorbidity of Major depression and panic attack on the age of onset of each of these disorders in a population-based sample of 12,668 Ss. Results indicated that 262 of these Subjects had lifetime comorbidity of DIS/DSM-III major depressive disorder and panic attacks. The Disorders begin earlier in the life when they were comorbid than when they occurred singly. Occurrence of one disorder increased the chance of the other.

Kessler et al. (1998) predicted lifetime panic-depression comorbidity in the National Comorbidity Survey of 8,098 respondents (aged 15-54yrs). Findings reported about half of Subjects with lifetime panic attack and panic disorders also met life time criteria for depression, whereas about one fifth of Subjects with lifetime depression reported a lifetime panic attacks and one tenth met lifetime criteria for panic disorder. Goodwin et al. (2004) determined the association between panic attacks and the risk of major depression among young adults (age 15-21 years) in the community by drawing data from a 21-year longitudinal birth cohort study (n = 1, 265). Results reported that having a panic attack in the preceding 3 years was significantly associated with elevated risk of current major depression (past month) at the ages of 18 and 21. This association persisted after adjusting for a range of fixed social, family and individual risk factors for psychopathology, history of major depression, comorbid psychiatric disorder and life adversity among both males (OR = 8.9) and females (OR = 2.3).

Brown et al. (2001a) obtained patterns of comorbidity for 360 patients carefully diagnosed with PDA according to the Anxiety Disorders Interview Schedule for DSM-IV: Lifetime (ADIS-IV-L), and found 59% with a comorbid anxiety or mood disorder and 46% with a comorbid anxiety disorder alone. Among specific disorders, 23% presented with a comorbid major depressive disorder, 16% with generalized anxiety disorder, and 15% each with social phobia or specific phobia. Other studies have found similar high rates of comorbidity (First et al., 1996).

**Comorbidity between Obsessive-Compulsive Disorder (OCD) and Depression**

Individuals with obsessive-compulsive disorder often have depressive symptoms, and patients suffering from recurrent depressive disorder (F33.-) may develop obsessional thoughts during their episodes of depression. In either situation, increases or decreases in the severity of the
depressive symptoms are generally accompanied by parallel changes in the severity of the obsessional symptoms.

Nowhere is the relationship of anxiety and depression stronger than for OCD. Up to 80% of patients with OCD may be currently depressed according to measures that assess severity of depressed mood. In addition, a diagnosable major depressive episode is also present in approximately one–quarter to one–third of cases (Barlow et al., 1986; Crino & Andrews, 1996; Weissman et al., 1994), with 22% meeting criteria. Dysthymia accompanies OCD somewhat less frequently, with 10% meeting Criteria. According to a report on a large cohort of patients, the likelihood of developing a mood disorder after the onset of OCD is second only to the likelihood of developing another type of anxiety disorder (Yaryura-Tobias et al., 2000). This relationship between OCD and depression is particularly evident if one examines lifetime diagnoses—that is, whether patients presenting with OCD have ever met criteria for another diagnosis during their lifetimes. It is found that 61% of patients experienced a major depressive disorder at some point in their lives, far exceeding the next most prevalent lifetime diagnosis (social phobia, at 27%).

Overbeek et al. (2002) investigated the co-occurrence of depressive disorders in obsessive-compulsive disorder (OCD). A retrospective chart analysis was performed on baseline ratings of 120 OCD patients and post treatment ratings of 72 of these patients. Result revealed that one third of the OCD patients in our sample were found to be depressed. Symptom severity on OCD symptoms at baseline did not differ between depressed and nondepressed OCD patients; on general anxiety symptoms, the comorbid group was more severely affected. Because of this high frequency of depression, some investigators have questioned whether OCD is simply a variant of a mood disorder (Insel, Zahn, & Murphy, 1985), and should be classified with mood rather than anxiety disorders. Furthermore, most studies that chronicle the onset of OCD (Rasmussen & Eisen, 1992b; Welner, et al., 1976), although there are clearly some expectations to this rule.

Bhattacharyya et al. (2005) investigated anxiety and depressive comorbidity in OCD among adult Indian sample of 218 OCD (146 males and 72 females) patients seen in the OCD clinic at the National Institute of Mental Health and Neurosciences, Bangalore, these patients were evaluated using the OPCRIT criteria for ICD-10 for the presence of comorbid depressive and anxiety disorders. Their mean age at OCD onset was 21.32 ± 0.64 years. Results reported that thirty-six (16.5%) patients had depressive episodes, 12 (5.5%) dysthymia and 15 (6.9%) any
anxiety disorder. However, findings also revealed that female OCD patients were more likely than males to have comorbid major depressive disorder.

**Comorbidity of social anxiety disorder with Depression**

Studies of comorbidity in patients with social phobia, most with the generalized subtype, evidence high levels of anxiety and depression. Kessler et al. (1999) studied co-morbidities between lifetime social phobia and mood disorders by using normal population data from the US National Comorbidity Survey (NCS). Results reported that strong associations exist between lifetime social phobia and major depressive disorder (odds ratio 2.9), dysthymia (2.7) and bipolar disorder (5.9). Odds ratios increase in magnitude with number of social fears. Temporally-primary social phobia predicts subsequent onset of mood disorders, with population attributable risk proportions of 10-15%. Social phobia is also associated with severity and persistence of co-morbid mood disorders. In the study reported by Brown et al. (2001a), 46% of 186 carefully diagnosed patients with social phobia met criteria for an additional Axis I disorder. More specifically, 45% met criteria for either an anxiety or mood disorder, 28% for an anxiety disorder alone, and 29% for a mood disorder alone (Major depressive disorder or dysthymia). In individual disorders, the most frequent comorbid disorder by a substantial margin was one of the mood disorders, with 14% meeting criteria for major depressive disorder and 13% meeting criteria for dysthymia. If one looks at the comorbidity of mood disorders over a lifetime, fully 44% presenting with a principal diagnosis of social phobia had met criteria for a mood disorder at some point in their lives, with 44% meeting criteria for major depressive disorder and 17% for dysthymia. These data underscore the close relationship between social phobia and depression. Charvira et al. (2004) studied comorbidity between social anxiety and major depression among youth randomly selected (from enrollees in a pediatric primary care clinic) sample of 190 families with children. Results reported that the generalized type of social anxiety disorder was highly comorbid with major depression, generalized anxiety disorder, specific phobias, and ADHD, while little comorbidity was present for the nongeneralized subtype of social anxiety disorder.
MODELING COMORBIDITY

A large number of models representing comorbidity have been proposed Klein and Riso (1993) originally described a comprehensive set of comorbidity models, which Neale and Kendler (1995) later elaborated and formalized in a quantitative manner. These models are referred to as KRNK models which are bivariate i.e. they involve only two disorders occurring simultaneously. However, the comorbidity phenomenon is better described as a multimorbidity phenomenon in the sense that patterns of association among mental disorders involve multiple disorders across the current nosology (Batstra et al., 2002). For this reason, it is also important to consider multivariate models, which involve more than two disorders simultaneously.

Bivariate Comorbidity Models

Associated Liabilities Models: A number of the KRNK models can be treated as subtypes of a single, general model which is referred as the associated liabilities model. A liability is an indirectly observed or latent propensity to develop observed or manifest disorders. This model, which is illustrated in Figure a, posits that each disorder is influenced by a latent liability factor and that these liability factors are correlated. This figure defines three distinct subtypes of associated liabilities models.

Multiformity Models: Multiformity models represent heterogeneity in the expression of liability, i.e., the possibility of multiple pathways from same liability to different manifestations of that liability. These models (represented in Figure b) posit that the liability factors A and B are independent and uncorrelated, but that both liability factors can cause symptoms of both disorders 1 and 2.

Causation Models: The KRNK causation models posit that one disorder may directly cause another disorder (Figure c). Under causation models, comorbidity results because of the direct influence of one disorder on the development of the other disorder. In directional causation models, one disorder causes the other. In the reciprocal causation model, in contrast, both disorders may cause one another.

Independence Model: Under the independence model, comorbid disorder reflects an independent condition, separate from the other disorders. Each disorder is influenced by its own
liability factor, and comorbid disorder is itself also influenced by its own liability factor, distinct from the liability factors influencing other disorders. Under this model, comorbidity does not represent the combined presence of two disorders, but rather a third distinct disorder.

**Spurious Association Models:** Under these models, some external variable or set of variables creates spurious associations between disorders. In the sampling bias model, there is bias selection of sampling methods of comorbid cases. This could occur if comorbid cases are oversampled because individuals with a greater number of disorders have more opportunities to be selected for study (Berkson, 1946).

**Multivariate Comorbidity Models**

In multivariate models, more than two disorders are considered simultaneously. Multivariate models are more comprehensive in providing explanations of comorbidity than are bivariate models. Rhee and colleagues (Rhee et al., 2004; Rhee et al., 2005) have evaluated the statistical power to distinguish between different bivariate comorbidity models, and have concluded that different comorbidity models can be distinguished well in many circumstances, with some caveats. Most multivariate comorbidity models can be treated as extensions of bivariate models to a greater number of disorders. In extending such models to include more disorders, however, hybrid models can be specified that would not be possible in the bivariate case. This flexibility allows multivariate models to provide more comprehensive explanations of comorbidity than bivariate models. Figure e illustrates this point. The comorbidity model represented by the path diagram in Figure e is a hybrid of the associated liabilities and multiformity models. The general point is that the multivariate model represented in Figure e unifies a number of bivariate models into a single integrated, albeit rather complex, account of the multimorbidity among six disorders. Multivariate models often elucidate the importance of parsimony in a way that bivariate models do not.
**Application of comorbidity models:** Although there is an extremely large literature on bivariate relationships between disorders, relatively few studies have explicitly compared multiple models of comorbidity within a KRNK framework.

Among different forms of comorbidity in psychiatry, comorbidity between depression and anxiety disorder seems to be the most frequently modeled. Perhaps the most comprehensive review of models of comorbidity between depression and anxiety disorders was provided by Middeldorp et al. (2005), who reviewed twin and family studies of depression and anxiety disorder in the framework of KRNK models. Middledorp et al. (2005) concluded that shared genetic liability can explain much of the comorbidity between depression and the anxiety disorders. Another example of a study comparing bivariate KRNK models was provided by Johnson et al. (2004), who compared various KRNK models of comorbidity between smoking and depression.

**Structural Models of Comorbidity of Anxiety and Depression**

Beginning in the 1980s, numerous studies have examined the genetic links between anxiety and depression, primarily investigating whether a common genetic diathesis renders certain individuals vulnerable to the development of both types of disorder. The first major analysis
investigated self-reported anxious and depressive symptoms in a large, community-based sample of Australian twins (Jardine et al., 1984). These analyses indicated that the observed phenotypic covariation between the two types of symptoms was largely due to a single common genetic factor (Kendler et al., 1987).

In contrast, anxiety disorders other than GAD are more modestly related to depression. Panic disorder, for example, is genetically distinguishable from both GAD and depression (Kendler, 1996; Kendler et al., 1995; Woodman, 1993), a finding that replicates the symptom-level results. Kendler et al. (1995) have reported the most comprehensive analysis of the genetic architecture of depression and the anxiety disorders, examining the associations among major depression, GAD, panic, and the phobias. They found evidence of two significant genetic factors. The genetic evidence has important implications for our understanding of the mood and anxiety disorders. First, it is clear that the anxiety disorders themselves are genetically heterogeneous (Kendler, 1996; Kendler et al., 1995).

Phenotypic models increasingly have emphasized that depression and anxiety are characterized by both common and distinctive features. An early example of this approach was a “two-factor model” based on the seminal work of Tellegen (1985) that emphasized the role of basic dimensions of affect. Extensive research has demonstrated that affective experience is characterized by two general factors: Negative Affect and Positive Affect (Tellegen, 1985; Watson & Clark, 1997; Watson & Tellegen, 1985). Negative Affect reflects the extent to which a person is experiencing negative mood states such as fear, sadness, anger, and guilt, whereas Positive Affect reflects the extent to which one reports positive feelings such as joy, enthusiasm, energy, and alertness.

These two general dimensions are differentially related to depression and anxiety. Specifically, depression and anxiety both are strongly related to measures of General Negative Affect. In contrast, measures of Positive Affect are consistently negatively correlated with depressed mood and symptomatology but are largely unrelated to anxious mood and symptomatology (Dyck et al., 1994; Jolly et al., 1994; Tellegen, 1985; Watson et al., 1988a). Thus, in this two factor model Negative Affect represents a nonspecific factor common to depression and anxiety, whereas Positive Affect is a specific factor that is related primarily to depression.
TRIPARTITE MODEL

Clark and Watson (1991c) extended the earlier (two factor model) model by proposing a second specific factor-physiological hyperarousal-that is relatively specific to anxiety. They argued that a “tripartite model” offered a more accurate characterization of anxious and depressive phenomena. In this model, symptoms of depression and anxiety can be grouped into three basic subtypes. First, many symptoms are strong indicators of a general distress or Negative Affect factor. This nonspecific group includes both anxious and depressed mood, as well as other symptoms (insomnia, poor concentration, etc) that are prevalent in both types of disorder. However, each syndrome is characterized by its own cluster of symptoms; somatic tension and hyperarousal (e.g. shortness of breath, dizziness and lightheadedness, dry mouth) are relatively specific to anxiety, whereas manifestations of anhedonia and the absence of Positive Affect (e.g. loss of interest, feeling that nothing is interesting or enjoyable) are relatively specific to depression. In other words, according to the tripartite model, depression is specifically characterized by low positive affect (PA), anxiety is specifically characterized by physiological hyperarousal (PH), and negative affect (NA) is a nonspecific factor that relates to both depression and anxiety.

NA represent displeasurable engagement with the environment and a sense of high subjective distress, it is a broad general factor of emotional distress that includes mood such as fear, sadness, anger, and guilt (Watson & Clark, 1984; Watson & Tellegen, 1985). Clark, Watson, and Mineka (1994a) further define NA as a temperamental sensitivity to negative stimuli resulting in feelings of fear, anxiety, depression, guilt, and self-dissatisfaction. NA has been described generally as “a stable, heritable trait tendency to experience a broad range of negative feelings such as worry, anxiety, self criticisms, and a negative self-view” (Keogh & Reidy, 2000). NA is seen as a common underlying factor contributing to both anxiety and mood disorders. It has been proposed that this common factor may explain the high rates of comorbidity and similarity between mood disorders and anxiety disorders, particularly generalized anxiety disorder (GAD) (Clark et al., 1994a), which is frequently seen as most closely reflecting the NA construct (Brown, Barlow, & Liebowitz, 1994; Brown, Chorpita, & Barlow, 1998).

The potential to differentiate anxiety and depressive disorders has rested, in part, on the Positive Affect (PA) construct (Watson & Clark, 1984; Watson & Tellegen, 1985). PA is seen as similar
in nature to NA in that it is stable and heritable. PA refers to a predisposition to experience a wide range of positive emotions. PA reflects an individual’s pleasurable engagement with the environment and the extent to which that person feels enthusiastic, active, and alert (Watson, Clark, & Carey, 1988a). Individuals experiencing anxiety and depression may exhibit similar, elevated scores on measures of NA. However, the distinguishing characteristic is that depressed individuals also score low on measures of PA (Watson et al., 1988a; Watson, Clark & Tellegen, 1988b). Clark and Watson (1991c) considered low PA to be characteristic of depressive disorder and unrelated to anxiety. Likewise, some other studies have reported support for low PA as a personality risk factor underlying depression (Brown et al., 1998; Keogh & Reidy, 2000).

Physiological Hyperarousal (PH) also termed as autonomic arousal, was originally conceptualized as a higher-order temperamental trait specifically underlying anxiety. PH is characterized by a predisposition to experience sympathetic nervous system reactivity, including elevated heart rate, respiration, and muscular tension. The somatic and physiological symptoms that characterize PH (such as shortness of breath, shaky hands, light-headedness or dizziness, sweating) are not components of depressive disorders but of anxiety disorders only. However, more evidence suggested that PH actually represents a lower-order factor arising as a consequence of an anxiety disorder (Brown et al., 1998; Clark et al., 1994a).

Studies have shown structure models consistent with tripartite model, for example, Brown et al. (1998) examined structural relationships among dimensions of Anxiety and Mood Disorders and dimensions of Negative Affect, Positive Affect and Autonomic Arousal among various model evaluated, the best fitted model reflected a structure consistent with the tripartite model. For instance, the higher order factors, negative affect and positive affect, influenced emotional disorder factors in the expected manner.
Structure of anxiety and depression

Hypothesized Model (1) (Figure-I)
Figure- I & II Structural models of DSM-IV disorders and tripartite model factors

The authors provided a hypothesized model which was viewed to be consistent with the following: This model was viewed to be consistent with the following: (a) The general distress symptoms shared by the various anxiety and mood disorders are best conceptualized as trait NA, a chronic feature that represents a vulnerability dimension for the development of emotional disorders; (b) the influence of trait PA is specific to Depression (DEP); and (c) the anxiety disorders, but not Depression (DEP), influence AA (i.e., the absence of a path from DEP to AA would not strain model fit because AA is specific to anxiety disorders). Although the hypothesized model contained a path from GAD to AA, it was uncertain whether this path would be significant given evidence that GAD and worry may be associated with autonomic suppression (Borkovec et al., 1993; Hoehn- Saric, McLeod, & Zimmerli, 1989), however, other data indicate that patients with GAD endorse autonomic symptoms frequently (Brown, Marten, & Barlow, 1995). This hypothesized model proved as best fit model. The hypothesized model was compared to competing model-2 which also proved the best fit model.
A designed model to conceptually represent the hypothesized relations between anxiety and depression according to tripartite conceptualization (Figure-A) have been presented. Model illustrated the NA, PA, and PH constructs of the Tripartite Model.

(Figure-A)

Note: PH = physiological hyperarousal; NA = Negative Affectivity; PA = Low Positive Affect.
Many studies related to Clark and Watson’s (1991c) tripartite model of anxiety and depression provide a mean for understanding the relation between anxiety and depression disorders and symptoms. Consistent with the tripartite model, Lonigan et al. (1994) found that measures indicative of low P.A. (i.e. low interest or low motivation) discriminated between children diagnosed with a depressive disorder and children diagnosed with anxiety disorder; in contrast, the anxious children and depressed children were indistinguishable on indexes of NA. Joiner, Catanzaro, and Laurent (1996) found support for the hypothesized three-factor structure of tripartite model among child and adolescent psychiatric inpatients.

In addition to its potential for refining measurement instruments and depression among both children and adults, the tripartite model highlights the potential role of temperament or personality factors that may predispose individuals for depressive disorders, anxiety disorders, or both disorders. Evidence has accumulated to suggest that NA and PA operate in children as outlined in the tripartite model with adults. For example, Lonigan et al. (1994) found that measures related to low PA best discriminated children with depressive disorders from those with anxiety disorders. More recently, Lonigan et al. (1999) examined the relations of PA and NA measures with anxiety and depression measures in a school sample of 365 children and adolescents and found that NA and PA measures performed in a manner consistent with findings from adult samples and that such findings were uniform across children and adolescents. More recently, studies have also provided support for tripartite model of anxiety and depression in children and adolescent’s clinical sample. For example, in a clinical sample of children and adolescents in Korea, Yang et al. (2006) found that the model fit of a three factor model was superior to one- and two-factor models showing the application of tripartite model to ethnic and cultural groups. Non clinical sample of children and adolescents have also shown consistency with the tripartite model. For instance, Chorpita (2002) evaluated the structural relationship among tripartite factors and dimensions representing selected anxiety disorders and depression in a large multiethnic school sample of children and adolescents. Results reported that Negative Affect was positively related with all anxiety and depression scales, and Positive Affect was negatively correlated with the depression scale. Physiological Hyperarousal was positively related with Panic only, and was not significantly positively correlated with other anxiety syndromes.
Research with adults has demonstrated that the tripartite model is useful for distinguishing between anxious and depressive states. For instance, Watson et al. (1988a) examined the overlap of anxiety and depressive disorders in adults and found that they could be discriminated by relatively pure measures of affect. Individuals with either an anxiety or a depressive disorder reported high levels of NA; however, only individuals with anxiety and depressive disorder reported low levels of PA. Studies have shown impressive support for tripartite models of anxiety and depression that include a common factor of negative affect, and the unique factors, positive affect and somatic arousal, among clinically-based samples of adults. For example, a study conducted on older adult psychiatric outpatients revealed that a three-factor model provided an adequate fit (Cook et al., 2004). Nonclinical-based samples of adults have also shown best fit for the tripartite model, for instance, Philipp et al. (2008) determined the generalizability of the tripartite model using a highly diverse sample of undergraduate students. The results of the study suggested that the model fits generally for each group, but the study did not find cross-group equivalence in the relationships between constructs that indicates the implication of tripartite model cross-culturally.

Previous studies with clinical and nonclinical samples have provided evidences supporting tripartite model, Clark et al. (1994b) investigated common and specific symptom dimensions of anxiety and depression proposed by the tripartite model and cognitive models. Results revealed a common underlying large factor, i.e., negative affect. However, the specific depression and anxiety dimensions were also apparent. The cognitive and motivational symptoms were specific to depression and the physiological arousal symptoms that were found to be unique to anxiety. Watson et al. (1995a) firstly tested the key prediction of the tripartite model that anxiety and depression can be differentiated better by de-emphasizing the largely nonspecific symptoms, i.e., negative affect and by focusing more sharply on the two specific clusters, low positive affect and physiological hyperarousal, in 5 samples (3 student, 1 adult, and 1 patient sample) by using the Mood and Anxiety Symptom Questionnaire (Watson & Clark, 1991). Consistent with the tripartite model, the MASQ Anxious Arousal and Anhedonic Depression scales both differentiated anxiety and depression well and also showed excellent convergent validity. In subsequent study (Watson, et al., 1995b), item analysis of the Mood and Anxiety Symptom Questionnaire from the same data sets (3 students, 1 adult, 1 patient) revealed the same 3 factors (General Distress, Anhedonia vs. Positive Affect, Somatic Anxiety) emerged in each data set.
suggesting that the symptom structure in this domain is highly convergent across diverse samples. Moreover, these factors broadly corresponded to the symptom groups proposed by the tripartite model. Findings by Reidy and Keogh (1997) also support those of previous studies in that although anxiety and depression share a general distress element, they can be discriminated by concentrating on specific symptoms. The factor analysis of the data collected on British students revealed 3 factors relating to general distress, positive affect, and anxious arousal which were consistent with three factor suggested by tripartite model. Brown et al. (1998) tested several models of the structural relationships of dimensions of key features of selected emotional disorders and dimensions of the tripartite model of anxiety and depression. Of various structural models evaluated, the best fitting involved a structure consistent with the tripartite model. Consistent with the predictions of the tripartite view and with previous work on North American samples (Joiner, 1996). Joiner et al. (1998) also provided the best fit of the tripartite model. Findings revealed a three-factor model with Positive Affect, Physiological Hyperarousal, and Negative Affect as factors. These findings also contribute to an emerging literature on the validity of the tripartite model, to indicate that the model and its implications may be applied cross-culturally.

Recent studies with clinically-based sample have also provided good fit for the tripartite model of anxiety and depression, As, Grant et al. (2003) using data of depression either or hypertension drawn from the Medical Outcomes Study (Tarlov et al., 1989) examined the structure of self-reported symptoms representative of the tripartite model. Results were broadly consistent with the tripartite model (Clark & Watson, 1991c). Factors emerged corresponding to each of the 3 posited first-order dimensions of negative affect, positive affect, and physiologic arousal. Likewise, Nitschke et al. (2001) have contributed for key prediction of tripartite model by suggesting that anxious arousal (somatic anxiety) and anxious apprehension (worry) represent distinct affective dimensions that can be distinguished from depression and negative affect. According to tripartite model, physiological hyperarousal (PH, i.e. autonomic hyperactivity) is specific for anxiety and not depression. Research on the relation between anxiety, depression and physiological measures representing arousal is lacking. Findings by Greaves-Lord et al. (2007) pointed towards high arousal in anxiety. As expected, low arousal was reported in depression which is consistent with the model’s predictions.
Community based epidemiological studies have also shown consistency with the key prediction of tripartite model of anxiety and depression. For instance, Lambert and associates (2004) examined the validity of the tripartite model of anxiety and depression in a community epidemiological sample of 467 urban African American youth. Results indicated that a 3-factor model representing the tripartite model fit the data well and better than competing models. Cannon and Weems (2006) using cluster analysis in a community sample of youth reported that four groups would emerge (primarily elevated on anxiety symptoms only, elevated on depression symptoms only, elevated on both anxiety and depression symptoms, and a low symptom group). Analyses using specific tripartite model variables and parent report of internalizing symptoms provided additional support for the groupings and tripartite model predictions. Findings added support for the tripartite model in youth, and support the idea that anxiety and depression do represent unique syndromes in youth. Recently, the tripartite model has also been validated in a community-based sample of African American adolescents (Gaylord-Harden, 2011). Community based sample with adults and older population have also shown support for the hypothesis of tripartite model. For instance, In order to examine the structure of neuroticism, anxiety, and depressive symptoms in young, middle, and older adult cohorts, Teachman et al. (2007) evaluated negative affect in a cross-sectional community sample of adults. As expected, the tripartite model fit best for all age groups. Further, multigroup analyses indicated age invariance for the tripartite model, suggesting the model can be effectively applied with older populations.

Some studies have also provided inconsistent results in the verification of the tripartite model. However, inconsistent results have been found in both clinical and non clinical samples. Burns and Eidelson (1998) tested the tripartite model of anxiety and depression with taking self-report data on three different samples, outpatients seeking treatment for (1) mood disorders, (2) substance abuse disorders and (3) college students. Analyses of data by using structural equation modeling reported that that tripartite did not proved good fit for any of the group because the nonspecific symptoms of depression and anxiety could not be adequately represented by a single General Distress factor. Similarly, Ollendick et al. (2003) in a study on children and adolescents, the goodness of fit of single-factor (i.e., negative affectivity), two-factor (i.e., anxiety and depression), and three-factor models was examined for boys and girls studying in fourth, seventh, and tenth grade. Results failed to support the tripartite theory; rather, the findings supported a two-factor model in all cases. One another study, Meeks et al. (2003) examined the
structural relationship of anxiety and depression in two samples of older adults, a large probability sample \((N = 1429)\) and a smaller convenience sample \((N = 210)\). Across all analyses, a correlated, two-factor, psychometric model was the most parsimonious. The tripartite model could be fit to the data, but added no explanatory power; in some cases, a one-factor model also fit. Buckby et al. (2008) examined the validity of the tripartite model using scale level data from the Mood and Anxiety Symptoms Questionnaire among help-seeking community sample of older adolescents and young adults. Results reported that a 2-factor model demonstrated superior model fit and parsimony compared to 1- or 3-factor models. These broad factors represented depression and anxiety and were highly correlated \((r = .88)\). It indicates that the tripartite model does not adequately explain the relationship between anxiety and depression in all clinical populations. Recently, Stevanovic and Lakic (2011) using a preliminary data from Serbia reported that PA correlated negatively with the depression only, while NA significantly correlated with the depression and all anxiety, except the separation anxiety significantly correlated with the depression, panic-somatic and separation anxiety and school avoidance indicating that the relationships between Tripartite Model of Emotion and symptoms of anxiety and depression in children and adolescents could be partially supported.

The fact that anxiety and depression may be discrete disorders and temporally linked, is not mutually exclusive propositions. Presently, it is unclear the extent to which symptoms of anxiety and depression accompany other disorders among children. Nonetheless, it is important to differentiate anxiety like or depression like symptoms from indicators of the syndromal disorder. For example, on the symptom level, anxiety and depression may have a high overlap; at the disorder level, however, they are not necessarily manifestations of a single disorder (Dobson, 1985b). It is often found that high NA is common to both anxiety and depression, thus contributing to the high correlation between measures of these two constructs. Low PA, on other hand, appears to be a unique feature of depression. Results suggest that NA and PA are useful constructs for understanding the distinctive and overlapping features of anxiety and depression (Kendall & Watson, 1989).

Brown et al. (1998) reported evidence supporting the tripartite model in LISREL analyses using both self-report and interview-based data. Psychophysiological analyses offer further support for the tripartite model by demonstrating that the three hypothesized symptom groups
reflect highly distinctive patterns of brain activity. Specifically, individuals reporting elevated levels of general Negative Affect consistently show augmented base startle reactivity (Cook et al., 1991; Lang et al., 1993). Other evidence has linked heightened levels of Negative Affect to increased activity in the right frontal cortex (Bruder et al., 1997; Tomarken & Keener, 1997).

Investigators in some of the earlier studies extracted highly correlated lower order factors corresponding to depression and anxiety, which then gave rise to a second order dimension of general distress or Negative Affect (Clark et al., 1994a; Steer et al., 1995). These results suggest a hierarchical three-factor model in which the traditional syndromes of anxiety and depression represent narrow, lower order constructs that are highly interrelated; in this hierarchical model, the Negative Affect dimension emerges as a broader, more general construct that represents the strong degree of overlap between the lower order syndromes. In other studies, however, general distress, anhedonia/low Positive Affect, and somatic arousal have emerged as three separable first order factors. In some analyses, the somatic anxiety and Negative Affect factors are moderately to strongly interrelated (Brown et al., 1998; Chorpita et al., 1998; Joiner, 1996), but in other cases, all three factors are largely independent of one another (Joiner et al., 1996; Watson et al., 1995b). These data suggest a nonhierarchical model in which the three hypothesized symptom factors exist at the same basic level of generality.

Brown et al. (1998) reported the most compelling analysis. They tested various alternative structural models and found evidence of two higher order factors; one of these (Positive Affect) was specifically related to depression, whereas the other (Negative Affect) was nonspecific. The third component of the tripartite model-anxious–arousal emerged as a specific lower order factor.

Differentiating depression and anxiety at the mood, symptom and syndromal levels has been a long-standing problem for researchers in psychopathology (Breier, Charney, & Heninger, 1985; Stavrakaki & Vargo, 1986). A number of studies have found that mood and symptoms measures of depression and anxiety correlate almost as highly between constructs as within constructs (Block, 1991; Dobson, 1985a; Gotlib, 1984; Johnstone et al., 1980). After reviewing this literature, various researchers have concluded that self report measures of depressive and anxious symptoms on average correlate .62 to .70, whereas clinical rating scales show better discrimination, with between-constructs correlations that range from .40 to .45 (Clark & Watson, 1991c; Dobson, 1985b; Gotlib & Cane, 1989).
Clark and Watson (1991a) argued that cognitive factors like negative attributional style, negative self-referent thinking, and dysfunctional beliefs are not specific to depression but instead represent the cognitive aspect of NA. However, it is proposed that anxiety can be distinguished from depression by the presence of physiological hyperarousal symptoms, and depression can be differentiated from anxiety by symptoms indicative of low PA (Clark & Watson, 1991a, b, c; Zoven & Tellegen, 1982; Tellegen, 1985). Empirical support for the model can be found in factor-analytic studies that have shown that various self-report anxiety and depression measures load on a single general distress factor (Block, 1991; Clark & Watson, 1991b; Gotlib et al., 1984).

Subsequent modeling has suggested limitations of the tripartite model that subjected the model to new revisions. It became increasingly apparent that the anxiety disorders are quite heterogeneous and subsume a diverse array of symptoms (Mineka et al., 1998; Zinbarg & Barlow, 1996). This heterogeneity reflected in two ways. First, anxiety disorders were differentially related to depression, with some disorders showing much greater comorbidity than others (Mineka et al., 1998). Secondly, it was established that a single specific factor such as the anxious arousal or somatic anxiety component of the tripartite model was insufficient to capture the diversity of symptoms subsumed within these disorders. In fact, Brown et al. (1998) found that anxious arousal was not generally characteristic of the anxiety disorders but instead represented the specific, unique element of panic disorder; subsequent evidence also has linked it to posttraumatic stress disorder (Brown et al., 2001a).

**An Integrative Hierarchical Model**

Basically, Barlow and colleagues first proposed a hierarchical model of the anxiety disorders (Barlow, 1991; Brown & Barlow, 1992; Zinbarg & Barlow, 1996). They asserted that each anxiety disorder contains a shared component that represents the higher-order factor of general distress/negative affectivity in a two-level hierarchical scheme. Accordingly, this higher-order factor not only is common across the anxiety disorders, but also is shared with depression; it therefore is largely responsible for the observed overlap both (a) among the individual anxiety disorders and (b) between depression and anxiety. In addition to this shared component, however, each of the anxiety disorders also contains a unique component that distinguishes it from the others.
Subsequently, Mineka et al. (1998) suggested a more accurate and comprehensive structural model. Basically, they proposed an integrative hierarchical model that incorporated key elements from both the tripartite model (Clark & Watson, 1991c) and Barlow’s hierarchical structure (Barlow, 1991; Brown & Barlow, 1992; Zinbarg & Barlow, 1996). In this integrative, hierarchical model, each individual syndrome is hypothesized to contain both a common and a unique component. Consistent with earlier models, the shared component represents broad individual differences in general distress/ negative affectivity; it is a pervasive higher-order factor that is common to both the anxiety and mood disorders and primarily is responsible for the overlap/comorbidity between these disorders. In addition, each disorder also includes unique features that differentiate it from all of the others. That is to say, each disorder is specifically related to their unique features in the manner that other disorder could not share (Kotov et al., 2007). Thus, Anhedonia, and the absence of Positive Affect comprise the specific, unique component of depression. These propositions are fully consistent with the original tripartite model. The major change which is required by the marked heterogeneity of the anxiety disorders is that anxious arousal is no longer viewed as broadly characteristic of all anxiety disorders, rather is considered as a specific element in syndromes such as panic disorder.

Further, Mineka et al. (1998) proposed that this integrative model can be enhanced with three additional considerations that are worth noting. First, the size of these general and specific components differs markedly across disorders. For instance, major depression and generalized anxiety disorder (GAD) both are distress-based disorders clearly containing an enormous amount of variance of general Negative Affect, in contrast, most of the other anxiety disorders contain a more modest component of nonspecific negative affectivity (Brown, et al., 1998; Kendler, 1996; Watson et al., 2005). Second, Mineka et al. (1998) reviewing previous studies (Hinden et al., 1997; Krueger et al., 1996; Watson & Clark, 1994a) suggested that this general Negative Affect dimension was not confined to the mood and anxiety disorders but also characterized many other types of pathology.

Finally, third, symptom specificity must be viewed in relative terms rather than absolute terms (Mineka et al., 1998). They basically believed that “it is highly unlikely that any group of symptoms will be found to be unique to a single disorder across the entire DSM” (p. 398). Such as, low positive affectivity was not unique to depression but also characterized schizophrenia,
social phobia, and other disorders (Brown et al., 1998; Watson & Clark, 1995a; Watson et al.,
1988a).

Seeing that Zinbard and Barlow (1996) have suggested, all anxiety and depressive disorders can
be classified as the emotional disorders. These emotional disorders should be collapsed together
into an overarching class of emotional disorders which can be decomposed into 3 subclasses: the
bipolar disorders (bipolar I, bipolar II, cyclothymia), the distress disorders (major depression,
dysthymic disorder, generalized anxiety disorder, posttraumatic stress disorder), and the fear
disorders (panic disorder, agoraphobia, social phobia, specific phobia), [Figure -1]. This new
model includes the bipolar disorders too, which were excluded from the previous anxiety-
depression models (Watson, 2005). Within the class of Distress disorders, together with Major
Depression (MD) and Dysthymic Disorder (DD), are now placed Generalized Anxiety Disorder
(GAD) and Post-traumatic Stress Disorder (PTSD).

(Figure-1, Watson, 2005)

Note: BPD I = bipolar I disorder; BPD II = bipolar II disorder, CT = cyclothymia; MDD = major
depressive disorder; DD = dysthymic disorder; GAD = generalized anxiety disorder; PTSD =
posttraumatic stress disorder.

High correlations with NA, genotypic (Kendler, 1996; Watson, 2005) and phenotypic similarities
(Krueger, 1999; Watson, 2005) between GAD and depression are taken as evidence for the
placement of GAD and PTSD within the same class with MD and DD. Other anxiety-phobic
disorders from the current classifications of disorders are now placed within the third group of
disorders labelled Fear disorders. This new classification reflects the idea that physiological
hyperarousal is not related to all forms of anxiety disorders. Thus, this new model implies that
each disorder has a specific set of characteristics, for example, low PA in depression and
physiological hyperarousal in panic disorder.
Unfortunately, this integrative hierarchical model has also run into problems. Most notably, the comorbidities among the unipolar mood and anxiety disorders cannot be adequately captured using a single nonspecific factor. That is, the integrative hierarchical model predicts (a) a high level of comorbidity between disorders that have strong components of negative affectivity, but (b) weaker overlap between syndromes containing less of this general factor variance. This former proposition has received substantial support in the literature. Most notably, major depression and GAD—two disorders that are strongly saturated with general factor variance—are highly comorbid. The latter prediction has proven to be more problematic, however. For instance, specific phobia and social phobia—disorders containing a lesser amount of nonspecific variance—also are strongly comorbid (Watson, 2005).

**Quadripartite Model**

Watson, (2009), recently, has suggested that accuracy of this integrative hierarchical model could be enhanced by positing additional nonspecific dimensions. In order to create a new structural model that captures these mood and anxiety disorders as precisely as possible, they analyzed the comorbidity of DSM mood and anxiety disorder diagnosed across four large national epidemiological samples (Kessler et al., 2005; Krueger, 1999; Slade & Watson, 2006) and proposed a new structural model.

Basically, the previous structure (hierarchical structure) was based on disorder-based analysis. The disorder-based analyses have proven good to illustrate the relations between the mood and anxiety disorder. However, several daunting problems were faced when conducting analyses based on diagnosis. Watson, (2009) have discussed five general problems that complicate disorder-based analyses (Brown et al., 2001 a. b; Watson, 2005).

1. Many *DSM* disorders show very **low base rates** in nonclinical samples and even in some patient samples. These low prevalence rates can render statistical analyses problematic or impossible.

2. **Changes in diagnostic criteria** may influence the strength of a disorder’s associations with other diagnoses. More generally, changes in symptom criteria make it hazardous to collapse findings across different editions of the DSM.
(3) **Diagnostic Inconsistencies across Studies** is also a major problem. Diagnostic criteria may be applied inconsistently across sites and studies. Diagnosticians may use different severity thresholds for judging clinically significant distress and dysfunction. Timing and duration requirements may be applied idiosyncratically.

(4) **Diagnostic Unreliability** is also another issue among diagnoses of disorders. Disorder-based approaches are largely dependent on dichotomous indicators (i.e., the presence versus absence of *DSM* diagnoses). It is believed that continuous scores are more stable and more reliable than dichotomous measures (Brown & Barlow 2005; Watson, 2003; Widiger & Clark, 2000).

(5) Finally, Diagnosis-based analyses fail to capture the marked heterogeneity of many anxiety and mood disorders (Watson, 2005) i.e. **Diagnostic Heterogeneity** is not found among these disorders. Consequently, symptom-based analyses provide a more precise and nuanced view of the associations between depression and the anxiety disorders.

Given these problems, Watson (2009) tried to supplement disorder-based framework of Hierarchical approach by moving to a new approach (examining specific symptoms within disorders) to understand the relations between the mood and anxiety disorder. This new approach focuses on examining relations among the specific symptom dimensions that make up the major disorders within these diagnostic classes (e.g., specific symptoms within PTSD).

Watson (2009) proposed a new a quadripartite model that represents a synthesis of the earlier tripartite and integrative hierarchical models. This new structural combines (a) the symptom focus of the tripartite model with (b) the quantitative elements and (c) the disorder-based framework of the integrative hierarchical model.

First, the model demonstrated that the anxiety disorders correlate quite differently with major depression and dysthymic disorder. Specifically, these mood disorders are strongly comorbid with GAD; show more moderate associations with PTSD, panic disorder, OCD, and social phobia; and are weakly related to agoraphobia and specific phobia. Second, GAD and PTSD are more strongly linked to the unipolar mood disorders than to the other anxiety disorders.

Consequently, structural analyses consistently indicate that major depression, dysthymic disorder, GAD, and PTSD mark one factor, whereas panic disorder, agoraphobia, social phobia, and specific phobia define another (Cox et al., 2002; Vollebergh et al., 2001; Watson, 2005; Slade & Watson, 2006).
These results demonstrate a fundamental problem with the current organization of these disorders in *DSM-IV*; consequently, it makes more sense to reorganize them into distress disorders and fear disorders, respectively (Clark & Watson, 2006; Watson, 2005). This new model also demonstrated that two quantitative dimensions/elements need to be considered when analyzing the properties of symptoms - the magnitude of their general distress component and their level of specificity vis-à-vis depression versus anxiety. These quantitative dimensions can be used to organize these relevant symptoms into $2 \times 2$ classification scheme which produces four basic groups of symptoms (i.e., a quadripartite model) that reflect varying combinations of distress and specificity- (1) High distress symptoms with limited specificity. (2) High distress symptoms with greater specificity. (3) Low distress symptoms with greater specificity. (4) Low distress symptoms with limited specificity.

Thus, this new model establishes the quasi-independence of the specificity and distress elements of the model and that differentiates this new scheme from the tripartite model (in which nonspecific symptoms were assumed to have a strong component of general distress): Even symptoms that have a relatively weak distress component can show little or no specificity.

To illustrate the value of this approach and to clarify the nature of the relations between the mood and anxiety disorders, the properties of major symptoms dimensions within three heterogeneous disorders; posttraumatic stress disorder, obsessive–compulsive disorder, and major depression was reviewed. Most notably, the evidence of marked heterogeneity both between and within disorders was found. For example, although PTSD symptoms correlate moderately to strongly with indicators of depression, the magnitude of these relations varies widely across the individual symptom dimensions. In contrast, although specific OCD symptom dimensions also show different levels of association with depression, the magnitude of these relations tends to be much lower (Watson, 2009). The value of this approach is best illustrated within depression. For instance, the data used have identified two different symptom dimensions with links to sleep disturbance: lassitude (which includes hypersomnia) and insomnia. Findings demonstrated that lassitude symptoms contain a moderately large distress component but still show a reasonable level of specificity; in contrast, insomnia is less strongly saturated with negative affectivity but displays virtually no specificity (Koffel & Watson, 2009).
Rationale for the study

Comorbidity among psychiatric disorders has now become a topic of active research. It is established from a number of recent reviews that there is no specific mental disorders which occur in isolation (Lilienfeld, 2003; Maj, 2005a, b; Widiger & Sankis, 2000). Research on relationships between anxiety and depression has proceeded at a rapid pace since the 1980s. From the Krapelin’s classification, the anxiety and depressive disorders have been treated as separate diagnostic classes in official nosologies; many researchers have argued that these disorders are distinct entities. There is proved to be some relationship between both these disorders. The sequential relationship between anxiety and depression has been observed both within episodes and across the lifetime. Within a single episode of illness, anxiety symptoms are more likely to precede depressive symptoms than the reverse (Alloy et al., 1990). Regarding lifetime comorbidity, Alloy et al. (1990) also reviewed evidence that an anxiety disorder is significantly more likely to precede a mood disorder than the reverse. Similarly, Kessler et al. (1997) found that all the anxiety disorder diagnoses are associated with an elevated risk of a later diagnosis of minor or major depression. The onset of depression is often preceded by anxiety (Avenevoli et al., 2001; Cohen et al., 1993) and depression and anxiety are much more often comorbid than would be expected by chance (Angold, Costello, & Erkanli, 1999; Brown et al., 2001a; Mineka, Watson, & Clark, 1998; Williamson et al., 2005). Recently, Lamers et al. (2011) by examining comorbidity patterns and temporal sequencing of separate depressive and anxiety reported that of those with a depressive disorder, 67% had a current and 75% had a lifetime comorbid anxiety disorder. Of persons with a current anxiety disorder, 63% had a current and 81% had a lifetime depressive disorder. In 57% of comorbid cases, anxiety preceded depression, and in 18%, depression preceded anxiety. Models depicting relationship of anxiety and depression have emphasized that depression and anxiety are characterized by both common and distinctive features. For example, “tripartite model” offered a more accurate characterization of anxious and depressive phenomena. According to this model, depression is specifically characterized by low positive affect (PA), anxiety is specifically characterized by physiological hyperarousal (PH), and negative affect (NA) is a nonspecific factor that relates to both depression and anxiety. Many studies have provided impressive support for the validation of tripartite model of anxiety and depression (Cannon & Weems, 2006; cook et al., 2004; Gaylord-
Harden, 2011; Lambert et al., 2002; Reidy & Keogh, 1997). Some studies of comorbidity of anxiety and depression have been found on Indian Population. For instance, Bhattacharya, Reddy and Khanna (2005) reported a study investigating anxiety and depressive comorbidity in Indian adult OCD. Results reported that 16.5% OCD patients had depressive episodes, 12 (5.5%) dysthemia and 15 (6.9%) any anxiety disorder. Recently Sahoo and Khess (2010) in an Indian study reported that comorbid anxiety and depression was high, with about 87% of college going students having depression also suffering from anxiety disorder. But, it is apparent from the review of related research studies that there is very less number of studies regarding the relationship between the anxiety and depression especially in India. And, there is no reported study regarding the investigation of tripartite model in clinical & non-clinical sample of Indian population. Most of the studies are related with the prevalence rate or the comorbidity of either anxiety or depressive symptoms with any physical illness. Therefore, the proposed work is aimed at studying the comorbidity among anxiety and depression, and also to replicate the tripartite model given by & Clark & Watson (1991c). In the light of earlier study, the present study was planned to examine the tripartite model of anxiety and depression in clinical sample of Indian Adult population.

The problem of the study may be stated as:

AN EXAMINATION OF THE TRIPARTITE MODEL OF COMORBIDITY BETWEEN ANXIETY AND DEPRESSION IN ADULTS

MAIN OBJECTIVES OF THE STUDY:

1. To examine the comorbidity between anxiety and depression.

2. To study the association of Positive and Negative Affectivity with symptoms of anxiety and depression.

3. To examine the relationship between various other symptoms of the Anxiety and Depression disorders.

4. To examine the tripartite model of anxiety and depression on Indian adult sample.