CHAPTER II

REVIEW OF RELATED LITERATURE AND
FORMULATION OF HYPOTHESES

With a view to seek some guidelines* from the previous researches, which could be helpful in formulating the present investigation, the results of some of the representative studies are discussed below. The present review is by no means complete or exhaustive. It is an attempt to indicate the main trends in research and theory which have a direct or indirect bearing on the present research problem in the context of comorbidity among depression and anxiety.

Disorders in which anxiety or depression is the predominant feature constitute a large part of contemporary psychiatric practice. As such depression is a matter of social and public health concern. Although there is a considerable agreement regarding depression as a common and significant problem for the general population and the client in psychotherapy in particular, the literature regarding possible symptoms and correlates of depression is extensive and sometimes conflicting. There are theories and researches in the literature which stress or examine particular symptoms and factors related to depression which have been recognized for thousand of years. Depression accompanied with its debilitating effects on well-being is a highly treatable problem that often goes undiagnosed or is attributed to the stresses and strains of daily life (Kessler et al., 1994).

In recent years the psychoanalytic theory has lost ground in favor of the cognitive theories of depression (Lewinsohn & Rosenbaum, 1987). One of the early cognitivists, Becker (1962) postulated that depressives’ self-abusive cognitions and guilt feelings stem in part from their parents extensive use of guilt-inducing socialization practices. Beck (1967) advanced a cognitive theory of depression that attaches central importance to negative cognitive schemata that dominate depressed person’s evaluations of themselves, their environment and their future. Beck (1967) was explicit in stating that these schemata develop as a consequence of parental behaviour. That is, depressed persons are assumed to have parents who were critical and nonapproving of their self-worth. These evaluations were internalized and formed the building stones for a negative schema.

*What is already known, what others have attempted to find out, what problems remain to be solved, what methods of attack have been promising or disappointing, the techniques and methodology followed by earlier investigators, etc
Thus, both psychoanalytic theory and certain cognitive theories of depression view parental non-acceptance and rejection as precursors of adult depression (Lewinsohn & Rosenbaum, 1987). Indeed, one can logically infer from these theories that depressed persons, in comparison with non-depressed persons, will remember their parents as having been more rejecting and more negative in their behaviour, as Blatt, Wein, Chevron, & Quinlan (1979), arguing from the psychoanalytic point of view, and Crook, Raskin, & Eliot (1981), arguing from Beck’s cognitive-theory point of view, have done.

The cognitive view of behaviour assigns primary importance to the self-evident fact that people think. It assumes that nature and characteristics of thinking and resultant conclusions determine what people feel and do and how they act and react. This view of behaviour and psychopathology has a long history that bridges the disciplines of clinical psychiatry, clinical and academic psychology and philosophy (Wason & Johnson-Laird, 1972; Broadbent, 1971; Beck, 1967; Neisser, 1967; Kelly, 1955). Depression is one area of theory and research in which cognitive factors, that is, the manner of perceiving, construing and consequences have been emphasized. In this context, much of the impetus has come from the theoretical and empirical work of Beck (1967, 1974), Seligman (1975), Lewinsohn (1976). Indeed, the recent empirical literature on the psychology of depression is dominated by studies addressing Beck’s Cognitive theory, Seligman’s learned helplessness model or Lewinsohn’s theory, which attributes depressive state to a low rate of response contingent positive reinforcement.

Beck (1967, 1976) proposed that self-deprecating and negatively biased thinking styles are not only core features of adult depression but also may play a key role in the development and maintenance of this disorder. In addition to the overriding negative triad – negative view of self, current circumstances and future - and stereotypic schemas, premises or dysfunctional attitudes (shoulds and musts), a central theme of Beck’s cognitive model is that depressed individuals characteristically make specific dysphoria-provoking cognitive errors, collectively referred to as distortions, in response to ambiguous or negative life experiences. Beck, Rush, Shaw, & Emery (1979) described seven of these typical cognitive errors: generalization (believing that if a negative outcome occurred in one case, it will occur in any case that is even slightly similar); selective abstraction (attending exclusively to negative features of a situation
in the belief that only the negative features matter); assuming excessive responsibility or personal causality (seeing oneself as responsible for bad things, failures and so on); presuming temporal causality or predicting without sufficient evidence (believing that if something had happened in the past then it is always going to be true); making self references (believing oneself, especially one's bad performances, to be center of everyone's attention); catastrophizing (always thinking of the worst on the premise that it's most likely to happen to one) and thinking dichotomously (seeing everything as one extreme or other, black or white, good or bad). These cognitive errors or distortions are interpretations and predictions that are not usually justified by the information provided (Hammen, 1981). Even if there is partially realistic foundation for such interpretation and predictions in the lives of some depressed patients (Coyne & Gotlib, 1983), their repetitive self deprecating quality and extremely negative character can be still considered dysfunctional or maladaptive (Kovacs & Beck, 1978).

ANXIETY: CONCEPT AND ASSESSMENT

Fear and anxiety have long been regarded as fundamental human emotions. The concept of fear, according to Cohen (1969), is clearly reflected in ancient Egyptian hieroglyphics. James Kritzeck, of the Department of Oriental Studies at Princeton, noted a central concern with anxiety in the work of the medieval Arab Philosopher, Ala ibn Hazm, of Cordova. In a treatise entitled “A Philosophy of Character and Conduct”, written in the eleventh century, Ibn Hazm unequivocally asserts the universality of anxiety as a basic condition of human existence.

In his classic book, “The Meaning of Anxiety”, May (1950) surveys the evidence of the centrality of the problem of anxiety in contemporary literature, music, art and religion as well as in psychiatry, psychoanalysis and psychology. He also documents the concern with anxiety in current political and philosophical thought and examines in some detail the views of those philosophers who have most significantly influenced modern anxiety theory. For Spinoza, fear was essentially a state of mind or attitude, a subjective condition of uncertainty in which there was the expectation that something painful or unpleasant might happen. Spinoza held that to entertain fear was a sign of “weakness of the mind” and that fear could be overcome by “courageous dedication to reason [May, 1950, p.24].”
While Spinoza’s views were shared by most of the intellectual leaders of the seventeenth century, his faith in reason was questioned by Pascal who observed much irrationality and “perpetual restlessness” in himself and his fellow man. Pascal clearly recognized the power of the emotions to influence human behaviour, and the obvious insufficiency of reason in overcoming passion. Belief in the rational control of emotions was further challenged by nineteenth century philosophers such as Schelling, Nietzsche, Schopenhauer and especially, by Kierkegaard. All these existential thinkers “. . . insisted that reality can be approached and experienced only by the whole individual as a feeling and acting as well as a thinking organism [May, 1950, p.30].”

The nineteenth century also witnessed the increasing concern of biologists with fear and anxiety. Darwin believed that the potential for experiencing fear was an inherent characteristic of men and animals which had evolved as an adaptive mechanism over countless generations. The specific nature of fear reactions was presumably shaped through a process of natural selection of those who were successful in coping with or escaping the many dangers that imperiled their lives. “In The Expression of Emotion in Man and Animals”, first published in 1872, Darwin provides a vivid description of the typical manifestations of fear-rapid palpitation of the heart, trembling, increased perspiration, erection of the hair, dryness of the mouth, change in voice quality, dilation of the pupils and the like. An important characteristic of the expression of fear was that it varied in its level of intensity,” . . . in its gradations from mere attention to a start of surprise, into extreme terror and horror [Darwin, 1965, p.306].”

In the twentieth century, anxiety emerged as a central problem and predominant theme of modern life. This era is referred to as “the century of fear” by the French author, Albert Camus and as the Age of Anxiety in the title of a sensitive poetic work by Auden (May, 1950). The Age of Anxiety is also the title of Leonard Bernstein’s Second Symphony, as well as a modern Ballet choreographed by Robbins that was inspired by Bernstein’s music and Auden’s poem (Mason, 1954).

Sigmund Freud is undoubtedly the most important contributor to our present understanding of anxiety phenomena. In 1894, he conceptualized anxiety neurosis as a discrete clinical syndrome to be differentiated from neurasthenia (Freud, 1953), and subsequently came to regard anxiety as the fundamental problem in all neurotic symptom formation (Freud, 1936). Freud defined anxiety as “something felt,” an unpleasant emotional (affective) state that is universally experienced.
Thus, for Freud, anxiety was not only a central problem in neurosis but understanding anxiety was also essential to the development of a comprehensive theory of human behaviour. Freud’s theoretical views on fear and anxiety were continually modified over a period of nearly 50 years as he searched for the “right abstract ideas” with which to clarify the essential nature of these concepts.

Since the turn of the century, clinical studies of anxiety have appeared in the psychiatric literature with increasing regularity. Pavlov’s (1927) discovery of experimental neurosis also served to stimulate numerous investigations of fear and anxiety in animals. Prior to 1950, however, there were relatively few experimental investigations of anxiety in humans. The complexity of anxiety phenomena, the lack of appropriate instruments for assessing anxiety and ethical problems associated with inducing anxiety in the laboratory have all contributed to the paucity of research prior to 1950.

Theory and research on anxiety were greatly stimulated in 1950 by the publication of three important books-May’s “The Meaning of Anxiety”, Mowrer’s “Learning Theory and Personality Dynamics” and Dollard and Miller’s “Personality and Psychotherapy”. A collection of papers presented in a symposium sponsored by the American Psychopathological Association was also published that same year in a volume entitled Anxiety (Hoch & Zubin, 1950).

Interest in anxiety research at mid-century was further stimulated by the development of Taylor’s (1951,1953) Manifest Anxiety Scale & Mandler and Sarason’s (Sarason & Mandler, 1952) Test Anxiety Questionnaire, the first of a number of psychometric instruments designed to assess fear and anxiety in adults (Spielberger, Gorsuch, & Lushene, 1970; McReynolds, 1968; Zuckerman & Lubin, 1965; Endler, Hunt, & Rosenstein, 1962; Cattell, 1957; Freeman, 1953). Later on, self-report scales have been developed for measuring general and test anxiety in children (Spielberger, Edwards, Lushene, Montuori, & Platzeck, 1971; Sarason, Davidson, Lightfall, Waite, & Ruebush, 1960; Castaneda, McCandless, & Palermo, 1956).

RECENT TRENDS

Anxiety is one of the most fundamental of all constructs in psychology. The concept of anxiety is central in conceptualization of psychopathology, motivation and personality. Thus, there are wide ranges of points of view and perspectives on
anxiety; as normal facilitating, pathological debilitating, negative affect, emotional state or reaction, motivation, personality trait or disorder and syndrome. So, research on anxiety is one of the most active areas in psychology and it has been the focus of considerable study especially in the last two decades (Cox & Norton, 2000; Rapee, 1996; Cox, Wessel, Norton, Swinson, & Direnfield, 1995; Norton, Cox, Asmundson, & Maser, 1995; Wolman & Strieker, 1994).

It is one of the most common psychological disorders in school-aged children and adolescents worldwide (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). The prevalence rates range from 4.0% to 25.0% with an average rate of 8.0% (Boyd, Kostanski, Gullone, Ollendick, & Shek, 2000; Bernstein & Borchardt, 1991). These figures could be underestimated since anxiety among a large number of children and adolescents goes undiagnosed owing to the internalized nature of its symptoms (Tomb & Hunter, 2004).

Notwithstanding the universality of anxiety, the question can be raised as to whether there are cultural differences pertaining to the nature and level of anxiety. Sartorius (1990) stated that “the analysis of the origin of the words used to describe anxiety can help in understanding how the concept is conceived of in different cultures. In ancient Egyptian language, the word for acute fear is composed of two symbols, one indicating narrowness and the other showing a man prostate, as if dying. The latter is the same symbol used to describe a severely wounded person. In Arabic, anxiety’s meanings are similar to the Slavic idioms. There are a variety of words used to suggest a state of anxiety – some referring to restlessness, others to constriction of chest or stomach” (p. 5f). Anxiety is associated with substantial negative effects on children’s social, emotional and academic success (Essau, Conradt, & Petermann, 2000). Specific effects include poor social and coping skills, often leading to avoidance of social interactions (Weeks, Coplan, & Kingsbury, 2009; Albano, Chorpita, & Barlow, 2003), loneliness, low self-esteem, perception of social rejection, and difficulty in forming friendships (Weeks et al., 2009; Bokhorst, Goossens, & De Ruyter, 2001). Importantly, school avoidance decreased problem-solving abilities, and lower academic achievements have also been noted as consequences (McLoone, Hudson, & Rapee, 2006; Rapee, Kennedy, Ingram, Edwards, & Sweeney, 2005; Donovan & Spence, 2000). Anxiety is considered to be a universal phenomenon existing across cultures, although its contexts and
manifestations are influenced by cultural beliefs and practices (Guarnaccia, 1997; Good
& Kleinman, 1985).

In India, the main documented cause of anxiety among school children and
adolescents is parents’ high educational expectations and pressure for academic
achievement (Deb, 2001). In India, this is amplified in secondary school where all 16-
year old children attempt the class X first Board Examination, known as Secondary
Examination. Results of the Secondary Examination are vital for individuals since this
is the main determining criteria for future admission to a high quality senior secondary
school and a preferred academic stream. There is a fierce competition among students
since the number of places in these educational institutions is fewer than the number of
students. Therefore parents urge their children to perform well in the first Board
Examination and to this end they may appoint three to four private tutors or more for
special guidance. After the Secondary Examination, all students appear in the Class XII
Final Board Examination known as Senior Secondary Examination. Competition is
again ferocious as performance in this examination determines university entrance.
Admission to courses in Medicine, Engineering and Management are the most
preferred choices for parents because these qualifications are seen to guarantee future
job prospects. It is relevant to mention here that in one year alone in India, 2320
children or more than six children per day, committed suicide because of failure in
examinations (National Crime Records Bureau, Ministry of Home Affairs, Government
of India, 2000). This shocking figure underlines the seriousness of this problem and
its resounding social costs to communities.

Previous research on the sociodemographic correlates of anxiety reveals well
established relationships with gender and socioeconomic status but little evidence of
consideration of school type. Gender effects for anxiety disorders and symptoms have
been found in studies of children and adolescents in English-speaking countries.
Generally, more girls than boys develop anxiety disorders and symptoms. Adolescent
girls report a greater number of worries, more separation anxiety, and higher levels of
generalized anxiety (Costello et al., 2003; Poulton, Milne, Craske, & Menzies, 2001;
Weiss & Last, 2001; Campbell & Rapee, 1994). Socioeconomic status has been found
to be both related and unrelated to anxiety. Broadly, social disadvantage is associated
with increased stress (Goodman, Ewen, Dolan, Schafer-Kalkhoff, & Adler, 2005). In
studies of adults, socioeconomic status has been found to impact both directly on rates
of mental illness and indirectly via the influence of poverty and financial hardship on low and middle income groups (Hudson, Rapee, Deveney et al., 2009). While anxiety is known to affect both learning and performance (McDonald, 2001), no empirical research has explored the relationship between adolescent anxiety and school type, school choice or mode of instruction.

Anxiety is triggered by uncertain and novel situations that have the potential for adverse consequences (Brooks & Schweitzer, 2011). For example, an inexperienced homebuyer who is concerned about losing the opportunity to buy a desirable home may feel anxious when making an offer. Anxiety is a common emotion that signals the presence of a potential threat promotes pessimistic appraisals of future events and triggers psychological responses that help individuals reduce their vulnerability (Young, Klap, Shoai, & Wells, 2008; Shepperd, Grace, Cole, & Klein, 2005; Raghunathan & Pham, 1999; Savitsky, Medvec, Charlton, & Gilovich, 1998; Barlow, 1988; Butler & Mathews, 1983, 1987). Extant anxiety research has largely focused on trait anxiety (Kantor, Endler, Heslegrave, & Kocovski, 2001; Stober, 1997; Eysenck, 1992, 1997; Endler, 1980), a personality characteristic similar to neuroticism that reflects an individual’s susceptibility to anxious feelings (Spielberger, 1985). Individuals with high trait anxiety and those with anxiety disorders experience anxious feelings frequently. Unlike trait anxiety, state anxiety is relatively short-lived, often occurring for mere seconds or minutes. Consistent with prior research (Brooks & Schweitzer, 2011; Gray, 1991), we conceptualize anxiety to subsume fear, tension, worry, nervousness, stress and apprehension. Anxiety is an unpleasant and aversive emotion (Marks & Nesse, 1994) that is characterized by high activation (within Russell’s [1980] affective circumplex model), high uncertainty and low control (within Smith and Ellsworth’s [1985] appraisal framework). State anxiety can be directed or incidental. In contrast to directed emotions that are triggered by an aspect of the decision context itself (by the nature of the decision or the people involved), incidental emotions are triggered by a prior stimulus that is unrelated to the current decision (Lerner & Keltner, 2001). For example, an individual who plans to invest in the stock market might experience directed anxiety because he is worried about losing money in the market, incidental anxiety because he watched an anxiety-inducing movie before making an investment decision or both.
Further in the field of anxiety research, the assessment and measurement issues are of major interest (Antony, Orsillo, & Roemer, 2001; Maltby, Lewis, & Hill, 2000). In Arabic psychological studies on anxiety, there are two trends, first, a trend towards translating and adapting the Western (mainly American and British) scales and inventories, for example, the State-Trait Anxiety Inventory developed by Spielberger, Gorsuch, Lushene, Vagg, & Jacobs (1983) and adapted by Abdel-Khalek (1989). The KUAS (Abdel-Khalek, 2000, 2002, 2003, 2004) was developed originally in Arabic and has comparable forms and several results in English (Abdel-Khalek & Lester, 2002, 2003). Based on the Arabic version, results are available for college students from specific Arab countries, that is Lebanon (Abdel-Khalek & ElYahoufi, 2004), Saudi Arabia (Abdel-Khalek & Al-Damaty, 2003), and Kuwait (Alansari, 2002, 2004b), Syria (Abdel-Khalek & Rudwan, 2001).

**GENDER DIFFERENCES IN DEPRESSION**

The gender difference in depression is among the most robust of findings in psychopathology research. Estimates are that, in adulthood, twice as many women as men are depressed (Lucht, Schaub, Meyer et al., 2003; Piccinelli & Wilkinson, 2000; Weissman, Bland, Canino et al., 1996; Kessler, Mignonage, Swartz, Blazer, & Nelson, 1993; Weissman & Klerman, 1977). Although the exact gender ratio varies slightly from culture to culture, most nations have reported a gender ratio close to 2.01 (Kuehner, 2003; Angst, Gamma, Gastpar et al., 2002). The World Health Organization has estimated that major depression is the leading cause of disease-related disability among women worldwide (Kessler, 2003). Research indicates that, although girls are no more depressed than boys in childhood (Cohen, Cohen, Kasen et al., 1993; Anderson, William, McGee, & Silva, 1987; Rutter, 1986), more girls than boys are depressed by ages 13 to 15 (Twenge & Nolen-Hoeksema, 2002; Wade, Cairney, & Pevalin, 2002; Hankin, Abramson, Moffitt et al., 1998; Kessler et al., 1993). In one well-sampled study of 15- to 24-year-olds, males had a lifetime incidence of major depressive disorder of 11%, compared with 21% for females (Kessler et al., 1993). If we are to understand the gender differences in depression in adulthood, we must understand its development in adolescence. The emergence of the gender differences in depression in adolescence has attracted the attention of medical and psychological researchers. The gender differences in depression has been attributed to a wide variety of factors including girls’ and women’s greater ruminative coping (Nolen-Hoeksema &
Girgus, 1994), dependence on relationships or affiliative needs (Cyranowski, Frank, Young, & Shear, 2000), ovarian and adrenal hormonal changes at puberty (Steiner, Dunn, & Born, 2003; Halbreich & Kahn, 2001; Goodyer, Herbert, Tamplin, & Altham, 2000), genetic factors (Zubenko, Hughes, Maher et al., 2002; Kendler, Gardner, Neale & Prescott, 2001; Jacobson & Rowe, 1999; Silberg, Pickles, Rutter et al., 1999; Kendler, Kessler, Neale, Heath, & Eaves, 1993), body dissatisfaction (Nolen-Hoeksema & Girgus, 1994), greater cognitive vulnerability (Hankin & Abramson, 2001), exposure to negative life events (Silberg et al., 1999; Kendler et al., 1993), experiences of rape and child sexual abuse (Kendler, Gardner, & Prescott, 2002), gender intensification and adherence to traditional gender roles (Aube, Fichman, Saltaris, & Koestner, 2000), and interactions among these factors (Hankin & Abramson, 2001; Petersen, Sarigiani, & Kennedy, 1991). The numerous theories and models of depression that have been proposed previously can be broadly categorized as proposing explanations for the emergence of the gender differences in depression emphasizing affective factors such as temperament or emotion regulation (Cyranowski et al., 2000; Kendler et al., 1993), biological factors such as genetics or pubertal hormones (Eley, Sugden, Corsico et al., 2004; Cyranowski et al., 2000), or cognitive factors such as cognitive style or rumination (Hankin & Abramson, 2001; Nolen-Hoeksema & Girgus, 1994). [c.f. Hyde, Mezulis, & Abramson, 2008, p.291].

Moreover, the evidence indicates that even subclinical or moderate levels of depressive symptoms are associated with diminished psychosocial functioning (Gotlib, Lewinsohn, & Seeley, 1995; Lewinsohn, Solomon, Seeley, & Zeiss, 2000), and the gender differences in depression is found whether assessed by symptom measures (Allgood-Merten, Lewinsohn, & Hops, 1990) or diagnoses (Kessler et al., 1993).

Three prior reviews have been particularly influential in the conceptualization of the causes of gender differences in depression: those by Nolen-Hoeksema & Girgus (1994), Hankin & Abramson (2001), and Cyranowski et al. (2000). Nolen-Hoeksema & Girgus (1994) considered the following etiological factors: personality (dependence on relationships, causal attributions and ruminative coping), biological factors (hormonal changes at puberty), body dissatisfaction, and social challenges (rape and sexual abuse, parental and peer enforcement of gender roles). They found some evidence for each of these factors in the etiology of the gender differences except that the data on hormonal influences were sparse and conflicting.
COMORBIDITY

Background

Comorbidity is currently one of the hot topics in psychopathology research (Kendall & Clarkin, 1992). According to Klerman (1990), a neo-Kraepelinian belief in the existence of discrete mental disorders clearly reemerged in the Third Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) (American Psychiatric Association, 1980), fueled in part by the success of research into diagnostic classification afforded by such developments as the Research Diagnostic Criteria (RDC) (Spitzer, Endicott, & Robins, 1978). Consistent with this belief, the framers of DSM-III included extensive exclusionary criteria. However, criticism of these exclusionary rules soon followed on both conceptual and empirical grounds (First, Spitzer, & Williams, 1990), and they were largely eliminated in DSM-III-R (American Psychiatric Association, 1987). Freed from these exclusionary rules, ensuing research documented extensive comorbidity across the entire spectrum of psychopathology (Clark, Watson, & Reynolds, 1995; Kendall & Clarkin, 1992) and sparked numerous discussions on the meaning and implications of comorbidity in psychopathology. With regard to anxiety and depression, the modern history of their classification (Clark & Watson, 1991a,b; Clark, 1989) and the surrounding controversies (Cole, Truglio, & Peeke, 1997; Lonigan, Carey, & Finch, 1994; Feldman 1993; King, Ollendick, & Gullone, 1991) have been reviewed previously. 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 (Cox, Swinson, Kuch, & Reichman, 1993; Akiskal, 1985). Nonetheless, other researchers have asserted that they represent a single underlying dimension or that 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 disorders” 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). In DSM-III exclusion rules limited co-diagnosis both within and across the anxiety and depressive disorders, but as mentioned, early research that ignored these exclusion rules revealed extensive comorbidity. Moreover, research based on the DSM-III-R and DSM-IV (American
Psychiatric Association, 1994), in which the exclusion rules were largely eliminated, documented that the anxiety and depressive disorders were among the most notable examples of overlapping disorders (Clark et al., 1995). Ironically, in so far as this research is leading some researchers to argue that diagnostic splitting has gone too far and that certain disorders should be recollapsed into a single category, it may serve ultimately to justify the view of the DSM-III framers that certain coexisting syndromes actually reflect the same underlying disorder and should not be diagnosed separately. 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, 1991b). The term comorbidity was coined in the context of chronic disease (Feinstein, 1970) to refer 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" (pp. 456-57). Attempts to extend this definition of comorbidity to psychiatric disorder, however, quickly run into difficulties in delineating the concept of a "distinct clinical entity" (Lilienfield, Waldman, & Israel, 1994).

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 (panic attacks versus feelings of worthlessness) and those that are shared (difficulty concentrating). However, few of these symptoms clearly differentiate patients with one type of disorder versus the other (Clark, 1989). When rated by clinicians, panic attacks, agoraphobic avoidance and overall autonomic symptoms (but, surprisingly, not anxious mood) tend to be found more frequently in anxiety disorder patients whereas depressed mood, anhedonia, psychomotor retardation, suicidal behaviour, early-morning awakening and pessimism (but not loss of libido, loss of appetite, feelings of worthlessness or guilt) are generally found to be more frequent in depressed patients. However, when self-ratings are compared, depressed patients tend to report more symptoms of both types than do those with anxiety disorders (Clark, 1989). A similar picture is obtained at the syndromal level. The psychometric properties of measures assessing syndromal
depression and anxiety are generally good in terms of convergent validity for both self- and clinician ratings but the discriminant validity of self-ratings is poor in both adults (Clark & Watson, 1991b) and children (Brady & Kendall, 1992). Clinician ratings are notably more discriminating, suggesting that clinicians give more weight to factors that distinguish anxiety from depression than do patients. It is unclear, however, whether this represents (a) sensitivity to subtle cues that patients discount or are unaware of or (b) rating biases on the part of clinicians. However, ratings of anxiety and depression in children by clinicians, teachers, and parents also show poor discrimination: Analyses of behavioural and observational rating scales typically yield a single anxiety-depression factor in children. It is unclear whether (a) the syndromes are less differentiated in children or (b) the scales used to assess them are less adequate than those available for use with adults (Brady & Kendall, 1992).

**IMPLICATIONS OF COMORBIDITY**

One result of the division of psychopathology into multiple distinct and "separate" categories or syndromes and more generally the reliance on the neo-Kraepelinian paradigm (a tradition exemplified by the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders), has been the observation that multiple disorders tend to co occur within the same individual. This overlap has often been termed comorbidity. Comorbidity may imply either the co-occurrence of two or more disorders in an individual at a given time, or the manifestation of multiple disorders during the lifetime of the individual. Understood in this way, an extensive body of literature documents the high prevalence of comorbidity among childhood disorders in general and between anxiety and depression in children and adolescents in particular. Despite the popular notion that comorbidity may be observed in clinical practice because of the self-selection processes that occur in clinic-referred samples, evidence suggests that comorbidity between anxiety and affective disorders is a phenomenon that exists in both help-seeking and non-help-seeking samples. Clearly, comorbid presentations of anxiety and affective disorders are common. However, the meaning and implications of comorbidity go beyond its utility as a purely descriptive phenomenon. For example, children with comorbid anxiety and depression are typically more severely impaired than children with either disorder alone (Nottleman & Jensen, 1994). Additionally, comorbidity may have implications for the course of psychopathology. In a sample
of adolescents in the Dunedin investigation, anxiety comorbid with depression at age 15 was associated with one of the highest rates of any disorder at age 18 (Feehan, McGee, & Williams, 1993).

Thus, although comorbidity appears to have a significant impact on disordered individuals, conceptual and practical implications of its presence have been largely ignored. In treatment outcome studies, comorbid conditions often serve to exclude individuals from entry. As such, we know that these individuals represent more severely impaired and disordered populations but we know little about how our treatments impact upon them. More importantly, perhaps, little is known about the pathways to comorbidity or in other words, the conditions under which comorbidity occurs or why it occurs with such frequency. Surprisingly, little effort has been put forth to explicate the meaning behind the high comorbidity of anxiety and affective disorders in youth. As Rutter (1994) acknowledged: the observation of comorbidity or co-occurrence has to be regarded as a starting point from which to undertake research to investigate the mechanisms that might be involved. It does not in any way provide an explanation of the processes and for this reason, it can only be regarded as a stimulus to further investigative work (p. 101). In this vein, four principal explanations for high rates of comorbidity can be deduced from the literature (Frances et al., 1990). First, although two disorders such as anxiety and depression may be indicative of two distinct underlying constructs, the extent of overlapping definitional criteria may produce high rates of comorbidity. Second, it may be that comorbidity is caused by the splitting of a unitary latent construct into two (or more) categories. Third, two disorders can be comorbid because they share some underlying causal or risk factors. Last, one disorder may cause or put an individual at increased risk for a second disorder. Thus, these first two explanations suggest that comorbidity of anxiety and depression is an artifact caused by the way in which the disorders are defined whereas the last two explanations suggest that comorbidity is due to the nature of the disorders (etiology and associated outcomes).

The helplessness-hopelessness theory of anxiety and depression (Alloy et al., 1990) is the most specific psychological theory that attempts to explain the comorbidity of these disorders as well as the patterns of their relationship. The helplessness-hopelessness perspective is an extension of the revised hopelessness
theory of depression (Abramson, Metalsky, & Alloy, 1989) and follows a series of increasingly complex theoretical formulations that have described the role of attribution and life events in the etiology of depression. However, although many of the tenets of the helplessness-hopelessness theory build on earlier formulations that have much empirical support (for reviews and commentaries on this literature Robins, 1988; Sweeney, Anderson, & Bailey, 1986; Peterson, Villanova, & Raps, 1985), the role of attribution in anxiety and depression comorbidity has not been extensively studied.

According to the helplessness-hopelessness model, once negative life events have occurred an individual then determines the degree to which the event is within his or her control, as well as the degree to which the cause of the event is internal (due to self), stable (enduring over time) and global (affecting outcomes in many life domains). The theory views anxiety and depression as both being characterized by helplessness (i.e., the expectation that future negative outcomes, should they happen, would be uncontrollable), but differing in that only depression is characterized by hopelessness (an expectation that future negative outcomes will occur due to attributions that the causes of negative events are stable and global). The high frequency of pure anxiety is explained by the theory in that helplessness (which causes anxiety) can occur without hopelessness or hopelessness depression. However, as hopelessness cannot occur without helplessness, depressive syndromes commonly have significant anxiety symptoms. Similarly, the evolution of anxiety into depression (and not the reverse) is also explained by a progression through the cognitive continuum from helplessness to hopelessness. In this way, anxiety is seen as the initial mood response to negative events and helpless attributions but may evolve into hopelessness depression as the individual becomes more certain that the causes of negative events are stable and global. In addition to specific attributions, the helplessness-hopelessness theory also integrates attributional style and the diathesis-stress concept that has been presented in earlier formulations of the theory (Abramson et al., 1989). In this way, the theory views attributional style as a distal contributory cause of hopelessness depression as mediated by specific causal attributions. Thus, individuals with depressogenic attributional styles should be more likely to attribute negative events to stable or global causes and therefore be more likely to experience hopelessness depression. The most recent theoretical formulations have also described non-attributional causes of hopelessness as
The helplessness-hopelessness theory and earlier formulations indicate an attributional basis for both clinical and subclinical mood states but very little research has examined the more basic question of anxiety and depressive symptom co-occurrence, especially on a day-to-day basis (Dohrenwend, 1990). The description of anxiety and depressive mood co-occurrence is an important starting point for understanding more complex associations at the diagnostic level and patients with coexisting depression and anxiety disorders show a greater overlap at the symptom level than at the diagnostic level (Hiller, Zaudig, & Bose, 1989). However, although symptom co-occurrence may often exist at the subclinical level, the examination of clinical theories using non-patient populations remains controversial. Whereas some authors have reviewed evidence indicating that clinical theories of depression can be appropriately tested using normal college samples (Vrendenburg, Flett, & Krames, 1993), others have argued that clinical depression is conceptually and empirically different from the distress or negative affect commonly reported in college student samples (Coyne, 1994). While this issue remains actively debated, the hopelessness subtype of depression is in itself conceptualized to exist on a continuum as a function of negative event and attribution severity. In this way, a less severe analog to clinical depression should exist when individuals exhibit a negative outcome and helplessness expectancy about only a limited life domain (Abramson et al., 1989).

ANXIETY AND DEPRESSIVE DISORDERS

Depression and anxiety frequently co-occur, especially in primary care settings. These co-occurrences manifest themselves in several ways and have different clinical courses. The scope and impact of depression and anxiety disorders worldwide are overwhelming. The Watershed Global Burden of Disease study found that major depression ranked fourth among all medical illnesses in terms of its disabling impact on the world population. The authors estimated that by the year 2020 depression would be second only to ischemic heart disease. Anxiety disorders rank close behind major depression, contributing additional disability. The prevalence of depressive and anxiety disorders in primary care settings is high. Between 10% and 20% of adults in any given 12-month period will visit their primary care physician during an episode of mental illness (although frequently not because of the episode). Depression and anxiety
disorders contribute to the majority of those visits. If unrecognized and undiagnosed, depression and anxiety disorders contribute to high medical utilization in the primary care setting. Twenty-four percent of high utilizers (the top 10%) have been found to suffer from current major depression, and 22% from an anxiety disorder.

There are a number of symptoms that are common to both anxiety and depression. However, there are also a number of symptoms that are not. Common symptoms include sleep disturbances and alterations in appetite and libido. On the other hand, symptoms that help differentiate the 2 conditions include hypervigilance, which is characteristic of anxiety and anhedonia or low mood symptoms, which are found in patients with depression. These differences in symptoms suggest that anxiety disorders and depression are distinct diagnostic entities. Roy-Byrne and Katon (1997) developed a useful way to depict the symptom overlap in GAD and depression. While depression symptoms include changes in mood, interest, appetite, esteem and suicidality, patients with GAD may also experience additional symptoms of agitation, dysphoria, sleep problems, fatigue, concentration problems, restlessness, irritability, worry, anxiety and tension. In addition, depressed patients with anxiety symptoms, compared to depressed patients without anxiety, called anxious depression, are more ill at presentation, have more psychological and social impairment, are more chronically ill and have a poorer response to treatment. Another way to look at the issue of the prevalence of anxiety symptoms in patients with depression was developed by Gorman. Comorbid depression symptoms or MDD occur in up to 90% of patients with anxiety disorders and 85% of patients with depression also experience significant symptoms of anxiety. Coexisting anxiety in patients with depression is associated with increased symptom severity, more chronic course and poorer outcome. Numerous studies have documented the negative effects of anxiety-depression comorbidity on various aspects of psychopathology such as course, chronicity, recovery and relapse rates (Brown et al., 1996; Rief, Hiller, Geissner, & Fichter, 1995; however, a few studies report no effects, Hoffart & Martinsen 1993), treatment seeking (Lewinsohn et al., 1995; Sartorius, Ustun, Lecrubier, & Wittchen, 1996), and psychosocial functioning (Lewinsohn et al., 1995; Reich et al., 1993). In so far as comorbidity rates tend to be substantially higher in individuals with more severe conditions (Kendall, Kortlander, Chansky, & Brady, 1992, Kessler et al., 1994), and severity of disorder is also a negative prognostic indicator (Keller, Lavori, Mueller et
al., 1992), it may be impossible at this stage of our knowledge to disentangle the issues of comorbidity and severity of disorder (Clark et al., 1995).

SUICIDE POTENTIAL

Multiple studies in a range of settings have found increased rates of suicidal ideation, suicide attempts and completed suicide in cases with comorbid anxiety and depression compared to those with a single disorder. In general, the risk of suicide is greater in patients with depression than any other single diagnosis (Wilson, Nathan, O’Leary, & Clark, 1996) with the possible exception of substance abuse (Conwell, 1996). However, the increased risk associated with comorbidity cannot be attributed simply to the presence of depression (Clark et al., 1995). For instance, the co-presence of anxiety in patients with depression or vice versa, increases the risk of suicide over the risk associated with pure depression (Bronisch & Wittchen, 1994, Reich et al., 1993). Moreover, the direction of causality is not simply from comorbidity to increased suicide risk: Newly abstinent substance abuse patients were more likely to develop a comorbid anxiety disorder if they had a history of suicidal ideation (Westermeyer, Tucker, & Nugent, 1995). Increased rates of suicide attempts in individuals with comorbid depression and anxiety disorders have been reported in community samples (Bronisch & Wittchen, 1994, Lewinsohn et al., 1995; Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992) as well as patient samples (Fawcett, Clark, & Busch, 1993; Reich et al., 1993). Many studies have also examined comorbidity of anxiety and depression with other disorders and found increased suicide risk regardless of the comorbid disorder (personality disorder, Zisook, Goff, Sledge, & Shuchter, 1994). Similarly, age does not appear to be a factor: Similar findings have been reported in children (Shafii, Steltz-Lenarsky, Derrick, Beckner, & Whittinghill, 1988), adolescents (Lewinsohn et al., 1995), and the elderly (Kunik, 1993).

WHAT ARE THE CONSEQUENCES OF COMORBIDITY?

Patients who have depression and anxiety comorbidity have higher severity of illness, higher chronicity and significantly greater impairment in work functioning, psychosocial functioning and quality of life than patients not suffering from comorbidity. One of the most important clinical reasons to screen for comorbidity is that unrecognized depression/anxiety comorbidity is associated with an increased rate of psychiatric hospitalization and an increased rate of suicide attempts. For example,
suicide attempt rates are 70% higher in patients with comorbid major depression and panic disorder than in those with major depression alone and more than 4 times higher than in patients suffering from uncomplicated panic disorder. Among anxiety disorders, post-traumatic stress disorder (PTSD) has the highest rate of comorbid psychiatric disorders, including alcohol abuse. Comorbid depression and anxiety have also been shown to significantly increase the suicide attempt risk above what is contributed by major depression alone. The results of a large national survey found that anxiety comorbidity was associated with a 2.5 fold increased likelihood of hospitalization, with patients suffering from concurrent panic disorder being most at risk (odds ratio = 3.2). The presence of comorbidity increases the chronicity of each disorder, slows recovery and increases the likelihood of a recurrence once the patient has recovered. Patients who suffer from comorbid anxiety disorder and depression tend to have more severe and chronic anxiety. They also have greater social and vocational impairment, higher rates of alcohol and substance abuse, and are at an increased risk of suicide. In addition, they demonstrate a poorer response to acute as well as long term therapy. Elderly patients ≥60 years who are depressed and also anxious have worse outcomes. Alexopoulos, Meyers, Young et al., 1996 in the PROSPECT study, “compared the outcomes of depressed elderly primary care patients in practices that implemented an intervention based on care management versus practices that offered usual care.” They concluded, “Regardless of the remission criterion, depressed patients with comorbid anxiety disorders, hopelessness and limitations in physical and emotional functioning were associated with low remission rates in primary care elderly patients receiving either the intervention or usual care.”

Most recently, the study of comorbidity has begun to emerge as an important task in itself, with the recognition that understanding how comorbidity arises may inform our understanding of the development of psychopathology. In effect, rather than being seen simply as a bothersome problem to be ignored, or an embarrassment to categorical diagnosis, or something to be defined away by the use of combined diagnostic categories, comorbidity has emerged as an opportunity for understanding better the development of psychopathology and as a potential tool for improving nosology. A number of review articles (Rutter, 1997; Achenbach, 1995; Loeb & Keenan, 1994; Klein & Riso, 1993; Angold & Costello, 1993; Abikoff & Klein, 1992; Kendall et al., 1992; Caron & Rutter, 1991) have detailed the
importance of taking comorbidity into account for understanding the etiology, course and treatment of psychiatric disorders.

Figures 1 and 2 summarise the prevalence and comorbidity data found in the survey. Among those individuals with mental disorders, marginally more women than men had at least one other comorbid mental disorder (28% of women as against 24% of men with any of these mental disorders). The patterns of comorbidity differed between men and women, reflecting the differences in prevalence within the sexes for the individual disorders. Among women, affective and anxiety disorders most often occurred together, accounting for three quarters of women who had more than one mental disorder. Among men, comorbid disorders more often involved an anxiety or an affective disorder in combination with a substance use disorder. These combinations of disorders affected two thirds of men who had more than one mental disorder. A small proportion of men (0.8%) and women (0.8%) had all three types of disorder (i.e., an anxiety, affective and substance use disorder) (Andrews, Hall, Teesson, & Henderson, 1999).

Figure 1. Prevalence (%) of single and comorbid affective, anxiety and substance use disorders among Australian men in the past year.
CONCLUSION

Thus, the co-occurrence of depression and anxiety disorders is extremely common in primary care. The clinical implications of depression/anxiety comorbidity include increased risk of suicide, increased risk of psychiatric hospitalization, increased disability, decreased compliance with treatment of medical illness, and markedly increased utilization of medical services. Patients with depression/anxiety comorbidity tend to have more chronic and recurrent forms of illness that require long-term treatment. This puts a premium on medications, such as the SSRIs, that have broad-spectrum efficacy (notably sertraline and paroxetine), wide therapeutic windows, and favorable pharmacokinetic and drug-interaction profiles (such as sertraline and citalopram) and are well tolerated in terms of side effects (such as weight gain), contributing to compliance with long-term treatment. Despite the high prevalence and important clinical implications of depression/anxiety comorbidity, very little prospective treatment research is available. The appropriate recognition and treatment
of depression and anxiety disorder comorbidity truly represent the last therapeutic frontier in the management of psychiatric illness in the primary care setting.

OVERVIEW (Gaps and Limitations)

1. Comorbidity has been defined as the presence of more than one disorder in a person, for a defined period of time (Wittchen, 1996). As the adherence to diagnosis according to diagnostic criteria has been widely accepted, comorbidity has by default become the rule, rather than the exception (Van Praag, 1996).

The recent literature on psychopathology in childhood and adolescence reflects the considerable attention that has been given to the issue of comorbidity, commonly defined as the joint occurrence of two or more disorders. Such findings are crucial to the evaluation of the validity and utility of diagnostic schema (c.f. Burke et al., 2005, p.1200).

2. There are three main types of comorbidity: comorbidity of physical and psychiatric disorders, e.g. depression and hyperthyroidism; comorbidity of related disorders, e.g. anxiety and depression and comorbidity of disorders indirectly related, e.g. psychotic depression and substance abuse.

3. Methodological issues implicated in the research of comorbidity include the question of whether the two diagnoses being studied are truly independent of one another. The current underlying hypothesis is that they are indeed independent, but this may not be entirely clear from the viewpoint of depressive and anxiety disorders.

4. Comorbidity is usually associated with poorer prognosis and also may present a challenge for the treating psychiatrist. For example, while treating one condition (such as anxiety disorder) in patients with depressive disorder, it might also lead to a hypomanic or even to a manic episode.

5. Theoretically comorbidity may advance our knowledge. For example, if two disorders frequently coexist (e.g. depression and anxiety), this may shed some light on a possible common psychopathological pathway, therefore providing an important lead for innovative research and therapeutic approaches.
6. Anxiety disorders such as panic disorder share a chronic and debilitating course with affective disorders such as major depression. **Mounting evidence suggests, however, that patients who have coexisting panic disorder and depression can have an even worse prognosis than those with either condition alone.** Comorbid patients may be more resistant to standard treatment, but many are successfully treated with cognitive or behavioral therapy and antidepressants medication.

7. Depressed patients with comorbid panic disorder may have more severe illness with greater vocational and psychotic social impairment (Coryell et al., 1988; Van Valkenburg et al., 1984), may make more suicide risk attempts (Fawcett, 1992; Fawcett, Schefter, & Fogg, 1990; Johnson, Weissman, & Klerman, 1990) and may be at greater risk of poor treatment outcome (Grunhaus, Rabin, & Greden, 1986).

8. Comorbidity among affective disorders, and particularly between anxiety and depression, has been repeatedly demonstrated (Wittchen et al., 2003; Angold et al., 1999), including some examination of developmental sequencing and heterotypic continuity. De Graaf, Bijl, Spijker, Beekman, & Vollebergh (2003) found that the onset of anxiety tended to precede depression. Costello et al. (2003) found evidence of both homotypic and heterotypic continuity for depression and anxiety. In a review, Wittchen et al. (2003) concluded that anxiety disorders typically precede the onset of depression when the two co-occur, and that the number and type of anxiety disorders present influence the risk for later depression. Further, some evidence exists to suggest that the presence of behavioural inhibition in contrasts to social withdrawn, predicts more favourable outcomes from comorbid disruptive behaviour and anxiety (Maughan & Rutter, 1998; Kerr, Tremblay, Pagani, & Vitaro, 1997). The overview provided the guidelines for the formulation of the following hypotheses.

**HYPOTHESES**

The following hypotheses were formulated:

1. Adolescents with Comorbidity of anxiety and depressive disorders would exhibit more of negative cognition as revealed by Self-deprecation,
Hopelessness and Dysfunctional attitudes than adolescents with pure anxiety and pure depression.

2. Adolescents with Comorbidity of anxiety and depressive disorders will be high on Suicide ideation than adolescents with pure anxiety and pure depression.

3. Adolescents with Comorbidity of anxiety and depressive disorders will be low on Extraversion than adolescents with pure anxiety and pure depression.

4. Adolescents with Comorbidity of anxiety and depressive disorders would score higher on Psychoticism than adolescents with pure anxiety and pure depression.

5. Adolescents with Comorbidity of anxiety and depressive disorders will show lower levels of cohesion, expressiveness and higher levels of conflict in the family environment than adolescents with pure anxiety and pure depression.