CHAPTER-I

INTRODUCTION

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 with high social, emotional and economic costs. Although there is 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 research in the literature which stress or examine particular symptoms and factors related to depression which have been recognized for thousands of years.

The concept of depression has been broadened to include milder forms. Clinicians and researchers have debated whether the concept of depression refers to a single disease that varies from mild to severe along a continuum or whether it consists of a set of discrete subtypes that differ in phenomenology, pathophysiology, and ultimately etiology (Kendell, 1976; Eysenck, 1970). This debate has yielded a number of different methods for subtyping depressive disorders, such as endogenous vs. reactive, psychotic vs. neurotic, and primary vs. secondary (Nelson & Charney, 1980; Akiskal, Rosenthal, Kashgarian, Khani, & Puzantian, 1979).

In spite of considerable agreement on the phenomenology of the clinical syndrome of depression, no complete satisfactory explanation has yet been offered to account for the mechanisms underlying the wide variations in symptomatology and course. The identification of psychosocial factors that may cause depression has proven to be an arduous task. The difficulty of demonstrating causal relationships in naturalistic research has been compounded by an overreliance on cross-sectional research which has been successful in demonstrating differences between depressed and non-depressed individuals; that is, it has identified abnormalities in the functioning of depressed individuals that are present during depressive episodes. Many of these abnormalities, such as dysfunctional cognitions, distressed relationships, anacritic personality types, and behaviors, have been implicated in the etiology of depression by theorists of various orientations. However, some of these problems in functioning may be symptoms, or concomitants of depression that appear with the onset of depressive episodes and disappear with remission.
Psychological Factors in Depression

Many different psychological factors may play a role in depressive disorders. In this section, the researcher discuss Freud’s psychoanalytic view, which emphasize the unconscious conflicts associated with grief and loss; personality factors, such as neuroticism and positive and negative affectivity, and cognitive factors, such as thoughts about the self and life events. These theories describe different diatheses to answer the question, “what are the characteristics of people who respond to negative life events with a depressive episode.”

Freud’s Theory In his celebrated paper “Mourning and Melancholia, “Freud (1917/1950) drew from clinical observations to develop a model of depression. He theorized that the potential for depression is created early in childhood, during the oral period. If the child’s needs are insufficiently or overly gratified, the person becomes fixated in the oral stage. This arrest in development may cause the person to become excessively dependent on other people for the maintenance of self-esteem.

Why do people with this childhood history come to suffer from depression? Freud hypothesized that after the loss of a loved one – whether by death, separation, or withdrawal of affection – the mourner identifies with the lost one – perhaps in a fruitless attempt to undo the loss. Freud asserted that the mourner unconsciously resents being deserted and feels anger toward the loved one for the loss. In addition, the mourner feels guilt for real or imagined sins against the lost person. According to the theory, the mourner’s anger toward the lost one becomes directed inward, developing into ongoing self-blame and depression. In this view, depression can be described as anger turned against oneself. Overly dependent persons are believed to be particularly susceptible to this process, and as noted above, people fixated in the oral stage are overly dependent on others.

Not much research has been carried out to test this theory, but the little information available does not strongly support it. Contrary to the idea that depression is a result of anger turned inward, people with depression express much more anger than do people without depression (Biglan et al., 1988). Despite this, some of Freud’s ideas continue to influence more recent models of depression. For example, Freud maintained that depression could be triggered by the loss of a loved one. A large body of evidence indicates that episodes of MDD are precipitated by stressful life events, which often involve losses. Researchers have consistently shown that people who are high in dependency are prone to depressive
symptoms after a rejection (Nietzel & Harris, 1990), a finding that also is congruent with Freud’s theory. Although some of Freud’s ideas still influence theories of depression, researchers have gone far beyond the clinical observations that were the foundation of his ideas.

Affect and Neuroticism

There is accumulating evidence of the importance for personality in the onset of depression. Some researchers assume personality to be the most consistent and important predictor of individual differences in depression among adults, irrespective of age (Diener & Diener, 1996; Costa et al., 1987; Ormel, 1983). Other researchers propose that the very strong association between depression and physical impairment and related disablement overwhelms other factors in potential etiological significance (Prince et al., 1997). The most complete etiological model of depressive symptoms probably requires the inclusion of all three categories of influences: personality, health-related and social situational factors (Kendler et al., 1993).

Personality traits and life events are both potent risk factors for depression. Neuroticism is a term introduced by Eysenck (1967) to describe a higher-order factor in analyses of self-rated or observer-rated measures of personality, characterized by dysphoria, anxiety, tension, and emotional reactivity (e.g., DeNeve & Cooper, 1998; McCrae & Costa, 1985). Heritability is 40% to 50% (Jang et al., 1996; Floderus-Myrhed et al., 1980) and reasonably stable across adult life (Viken et al., 1994). High premorbid neuroticism scores are a robust predictor of future onset of MDD (Kendler et al., 1993b, 2004). Kendler et al. (1993b) estimated that 55% of the genetic risk of MDD was shared with neuroticism. There may be common genetic factors (presumably, specific DNA sequence variations) that can predispose to MDD, neuroticism, and generalized anxiety disorder, and less clearly to panic disorder and social phobia, while obsessive-compulsive disorder and simple phobias are more independent (Weissman et al., 2005; Mineka et al., 1998; also Kendler et al., 1995). Thus, some genetic studies incorporate depressive and anxiety symptoms into a single phenotype, either with trait scores such as neuroticism or using categorical diagnoses.

Among personality traits, high levels of neuroticism (Katz & McGuffin, 1987) or low levels of extraversion (Clark, Watson, & Mineka, 1994) have been associated with depression. Neuroticism refers to “a broad dimension of individual differences in the
tendency to experience negative, distressing emotions and to possess associated behavioral and cognitive traits" (Costa & Me Crae, 1987). Neuroticism is associated with a higher risk of onset of depression, with more severe episodes, and with a more unfavorable outcome (Ormel et al., 2004a; Klein et al., 2002). Although neuroticism has a state-dependent component, neuroticism scores of remitted individuals are still higher than those of never-depressed individuals (Ormel et al., 2004b; Maier et al., 1992), and also predict subsequent episodes (Surtees & Wainwright, 1996; Duggan et al., 1990; Faravelli et al., 1986).

Although elevated scores in neuroticism or lowered ones in extraversion might merely reflect depression's severity (Farmer, Redman, & Harris, 2002), many prospective studies further found that these personality traits predicted the onset of depressive symptoms (Block, Gjerde, & Block, 1991) and depressive disorders (Kendler, Kuhn, & Prescott, 2004). Personality may also play a role on the recurrence or relapse of depression. The relapse of depression in depressed adult patients was found to be associated with elevated scores in neuroticism, but not with lowered scores in extraversion (Berlanga, Heinze, & Torres, 1999), and the neuroticism scores of remitted adults remained higher than those of never-depressed individuals (Ormel, Oldehinkel, & Vollebergh, 2004). The differential associations of these two personality traits with depression or its recurrence were found in relatives of depressed adult patients as well (Duggan, Sham, & Lee et al., 1995). Another line of evidence deriving from twin studies suggested that neuroticism shared substantial genetic contributions with both first onset and recurrence of depression, whereas extraversion did not (Burcusa & Iacono, 2007; Kendler, Gatz, & Gardner, 2006; Clark, Watson, & Mineka, 1994). Yet, little is known of whether personality traits are associated with the recurrence of depression in adolescents.

Several clues further imply that the personality traits and life events may not merely be two independent risk factors for depression. A prospective study in women found that the impact of neuroticism on the onset of depression was greater at high than at low levels of adversity (Kendler, Kuhn, & Prescott, 2004). One possibility is that there is common genetic vulnerability to both recurrent depression and accompanied psychosocial risk factors such as neuroticism, stressful life events, or social support (Burcusa & Iacono, 2007). Another possibility is that episodes of depression might leave scars that alter personality in ways that lowered the threshold of life stress required to precipitate depression recurrence (Ormel, Oldehinkel, & Brillman, 2001; Zeiss & Lewinsohn, 1988). In addition, individuals with
elevated neuroticism usually cope less efficiently with stress (McCleery & Goodwin, 2001; Uehara, Sakado, & Sakado, 1999). Thus, personality traits and life events tend to be intertwined (Kendler, Kuhn, & Prescott, 2004; Ormel, Oldehinkel, & Brillman, 2001) and have joint influences on the course of depression (Bolger & Zuckerman, 1995). However, whether nonsevere life events in adolescence also have such joint influences with personality traits on depression have been rarely explored.

There is also some evidence that neuroticism is related to a negative cognitive bias, although results are not consistent (Rusting, 1998). Of the 2 studies that specifically focus on the relation between neuroticism and emotion perception (faces), one reports no relation (McCown et al., 1989), while the other reports a positive relation with regard to the perception of ambiguous stimuli (Geers & Bouhuys, 1998). Finally, there is some evidence that neuroticism is linked with increased cortisol levels in response to stress (Zobel et al., 2004; Kirschbaum et al., 1995). Other studies, however, found the opposite (McCleery & Goodwin, 2001) or did not find any relation between neuroticism and cortisol at all (Roy, 1996).

Researchers have also amply demonstrated that major depression and anxiety disorders frequently co-occur. How can we differentiate anxiety and depression? One model conceptualizes depression and anxiety along three broad dimensions (see Table 1.1): negative affect (distress and worry), positive affect (happiness and contentment), and somatic arousal (sweaty palms, fast heart rate, etc.). Anxiety and depression are both expected to involve negative affect. Anxiety, but not depression, is expected to involve somatic arousal (Clark, Watson, & Mineka, 1994). And depression, but not anxiety, is expected to involve low levels of positive affect.

People who show high negative affect, low positive affect, and high somatic arousal may be at risk for comorbid anxiety and depressive disorders.

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<td>Anxiety Disorders</td>
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According to this model, depressive disorders should be differentiated from anxiety disorders by the lack of positive affect. Many of the symptoms of depression seem closely related to lack of positive affect – a loss of interest in pleasurable activities and even symptoms like lack of appetite and lack of interest in sex. Further, studies of responses to positive pictures and films suggest that people with MDD show fewer positive facial expressions, report less pleasant emotions, show less motivation, and demonstrate less psychophysiological activity in response to positive stimuli than do people without depression (Shestyuk et al., 2005; Solan, Struass, & Wisner, 2001; Henriques & Davidson, 2000; Berenbaum & Oltmanns, 1992). Research, then, does support the idea that episodes of MDD are characterized by high negative affect and low positive affect.

Can high negative affect and low positive affect be used to predict depression? Studies of personality help address this question. Several longitudinal studies suggest that neuroticism, a personality trait that involves the tendency to react to events with greater than average negative affect, predicts the onset of depression (Jorm et al., 2000). A major study of twins suggests that neuroticism explains at least part of the genetic vulnerability to depression (Fanous, Prescott, & Kendler, 2004). In fact, neuroticism is the personality trait most strongly associated with depression. Thus, there is good evidence that people who tend to experience negative affect are at elevated risk for developing depression.

The evidence for positive affect as a predictor is not clear. For example, extraversion is a personality trait associated with frequent experiences of positive affect. Some, but not all, studies suggest that low extraversion predicts the onset of depression (Klein et al., 2002). How would this fit with the idea that once a person is depressed, he or she experiences less positive affect? Some people might be fairly happy until the depressive symptoms kick in, at which point their level of happiness might decrease. In this way depression may suppress positive affect.

In sum, neuroticism predicts both anxiety and depression. But low extraversion doesn’t always precede depression. Once people are depressed, though, they seem to experience less positive affect than do people with no disorder.

Cognitive Theories: In some theories, negative thoughts and beliefs are seen as major causes of depression. One can easily think of people who interpret life events differently; some people seem to see the downside of events much more easily than others do.
Beck’s theory: The most important cognitive theory of depression is that of Aaron Beck (1967). His thesis is that people develop depression because their thinking is negative (see Figure 1.1). That is, **Beck proposed that depression is associated with the negative triad: negative views of the self, the world, and the future.** The “world” part of the depressive triad refers to the person’s own corner of the world – the situations he or she faces. For example, the person might think, “I cannot possibly cope with all these demands and responsibilities” as opposed to worrying about problems in the broader world outside of their life.

![Diagram](attachment:image.png)

**Figure 1.1**: The interrelationships among different kinds of cognition in Beck’s theory of depression.

According to this model, in childhood, people with depression acquired negative schemata through experiences such as loss of a parent, the social rejection of peers, or the depressive attitude of a parent. Schemata are different from conscious thoughts – they are an underlying set of beliefs that operate outside of a person’s awareness to shape the way a person makes sense of his or her experiences. The negative schema is activated whenever the person encounters situations similar to those that originally caused the schema to form.

Once activated, negative schemata are believed to cause cognitive biases, or tendencies to process information in certain negative ways (Kendall & Ingram, 1989). That is, Beck suggested that people with depression might be overly attentive to negative feedback about themselves and more likely to remember such negative information than other people are. Likewise, they might fail to notice or to remember positive feedback about themselves. People with an underlying ineptness schema might readily notice signs that they are inept and remember feedback that they are inept. Signs that they are competent, though, are not noted
or remembered. Overall, people who are depressed make certain cognitive errors to arrive at biased conclusions. Their conclusions are consistent with the underlying schema, which then maintains the schema (a vicious cycle).

Despite the clear evidence that thinking is negative during a depressive episode, the greatest challenge for cognitive theories of depression is to resolve questions of cause and effect. That is, can certain cognitive style cause depression, or do depressive symptoms cause those cognitive styles? Some studies suggest that people with negative cognitive styles are at elevated risk for developing depression. For example, in a study of 1,507 adolescents, very high scores on the Dysfunctional Attitude Scale (DAS) in combination with negative life events predicted the onset of MDD (Lewinsohn, Joiner, & Rohde, 2001). Other researchers found that high scores on the DAS predicted relapse for several years after treatment for depression (Segal et al., 2006). On the other hand, in a study of 770 women followed for 3 years, the DAS did not predict first episodes of depression, nor did the DAS scores predict recurrent episodes of depression once history of depression was controlled (Otto et al., 2007). Hence, findings are not consistent regarding the DAS.

Hopelessness Theory: The initial version of this theory was called learned helplessness; it was then modified to incorporate attributions and then modified again to emphasize hopelessness (see Figure 1.2).

Martin Seligman (1974) formulated learned helplessness theory to explain the behavior of dogs given electric shocks. He compared two groups of dogs that both received repeated shocks: one group could escape from the shocks, and the other could not. The dogs that received inescapable shocks seemed to give up. Later, when the experimental conditions were changed and the shocks could be avoided, these dogs were less likely to learn an avoidance response than dogs that had been able to escape from shocks. Rather, after a shock, most of them would lay down in a corner and whimper. Seligman proposed that animals acquire a sense of helplessness when confronted with uncontrollable aversive situations. This sense of helplessness then impairs their performance even when aversive situations are controllable. Animals exposed to uncontrollable shocks also developed symptoms that look like depressive symptoms, such as decreased appetite. On the basis of neurobiological and behavioral studies on the effects of uncontrollable stress, Seligman concluded that learned helplessness in animals could provide a model for human depression.
Researchers revised the learned helplessness model to incorporate cognition (Abramson, Seligman, & Teasdale, 1978). The revised theory focused on three key dimensions of attributions – the explanations a person forms about why a stressor has occurred (Weiner et al., 1971):

1. Internal (personal) versus external (environmental) causes
2. Stable (permanent) versus unstable (temporary) causes
3. Global (relevant to many life domains) versus specific (limited to one area) causes

Attributional style predicts increases in depressive symptoms (Peterson, Maier, & Seligman, 1993), but it is unclear whether attributional style predicts full diagnoses of MDD. For instance, attributional style has predicted the onset of MDD among children (Nolen-Hoeksema, Girgus, & Seligman, 1986), but some studies have found that attributional style...
did not predict the onset of diagnosable MDD in adolescents (Lewinsohn, Joiner, & Rohde, 2001) or adults (Barnett & Gotlib, 1988).

The current version of the theory, hopelessness theory (Abramson, Metalsky, & Alloy, 1989), suggests that cognitive processes explain only one type of depression (hopelessness depression). Symptoms of hopelessness depression include decreased motivation, sadness, suicidality, decreased energy, psychomotor retardation, sleep disturbances, poor concentration, and negative cognitions. In this view, the most important trigger of this type of depression is hopelessness, which is defined as an expectation that 1) desirable outcomes will not occur and that 2) the person has no responses available to change the situation. As in the revised model incorporating attributions, hopelessness can be triggered by stable and global attributions about the causes of stressors. But the model also suggest that there are other ways in which a person can become helpless, including through low self-esteem or through the sometimes accurate recognition that life events will have severe negative consequences.

Gerald Metalsky and colleagues conducted the first test of hopelessness theory. Early in the semester, college students completed the Attributional Style Questionnaire (ASQ), as well as questionnaires to assess their grade aspirations, their depressive symptoms, hopelessness and self-esteem. These measures were used to predict the persistence of depressive symptoms after a test among the students whose grades were below their expectations. Those who attributed poor grades to global and stable factors experienced more hopelessness, but this pattern was found only among students whose self esteem was low. Hopelessness predicted depressive symptoms. Clearly, these results support the hopelessness theory. Also, a similar study conducted with children in the sixth and seventh grades yielded almost identical results (Robinon, Garber, & Hillsman, 1995).

Cognitive vulnerability to depression

Several recent reviews have articulated the general cognitive vulnerability–stress model of depression (Abramson & Alloy, 2006; Abramson et al., 2006; Hankin & Abramson, 2001). Cognitive vulnerability–stress models of depression posit that individuals with certain negative cognitive styles have greater chances of developing depression when they encounter negative or stressful life events. Two of the most well-examined cognitive vulnerability
models of depression are the hopelessness theory and the ruminative response styles theory (Nolen-Hoeksema, 2000; Abramson, Metalsky, & Alloy, 1989).

The hopelessness theory of depression proposes that individuals who make negative inferences about causality, self, and consequences in response to negative events will be most likely to develop depression in the wake of negative events (Abramson et al., 1989). Retrospective and prospective tests of the hopelessness theory in adults and adolescents have supported the hypothesis that these cognitive styles do in fact confer vulnerability to depression (Hankin, Abramson, & Siler, 2001; Alloy et al., 2000; Schwartz, Kaslow, Seeley, & Lewinsohn, 2000; Abramson et al., 1999; Metalsky, Joiner, Hardin, & Abramson, 1993; Alloy & Clements, 1998; Alloy, Lipman, & Abramson, 1992; Metalsky & Joiner, 1992).

Perhaps the most rigorous examination of the cognitive vulnerability-stress model in young adults occurred in the Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project (Alloy et al., 2000). In this two-site study, college freshmen were selected on the basis of being high or low on standardized measures of cognitive vulnerability. Stressful life events and depression were assessed every 6 weeks over a 2.5-year period and then every 4 months for 3 additional years. Participants high in cognitive vulnerability who experienced more stressful life events were significantly more likely to develop depression over the course of the study. The cognitive vulnerability-stress model predicted both first episodes of depression (for participants high in cognitive vulnerability with no prior history of depression) and recurrences of depression (Alloy et al., 2000). To date, this is the most rigorous prospective study testing the core vulnerability-stress interaction predicted by the cognitive vulnerability-stress model of depression.

The cognitive vulnerability-stress interaction has been demonstrated in adolescents as well. For example, Hankin and colleagues (Hankin & Abramson, 2002; Hankin et al., 2001) found, in two studies, that cognitive style interacted significantly with negative events to predict depressive symptoms in high school students.

Overall, attempts to apply the cognitive vulnerability-stress model to predicting depression in children and younger adolescents have yielded inconsistent results that the authors believe, reflect a particular pattern. According to two meta-analyses of studies of children and adolescents, attributional style correlates with depressive symptoms ($r = .41$, $p < .001$).
Gladstone & Kaslow, 1995; and \( r = .50 \), Joiner & Wagner, 1995). These correlations, however, test simple main effects and do not test the interaction of cognitive style and stress that is implied by the theory. Tests of this interaction have yielded more mixed results (Joiner & Wagner, 1995). Some prospective studies have found that negative attributional style, in interaction with negative events, predicts increases in depressive symptoms in children and adolescents (Prinstein & Aikins, 2004; Abela & Sarin, 2002; Abela, 2001; Schwartz et al., 2000; Hilsman & Garber, 1995; Robinson, Garber, & Hilsman, 1995; Turner & Cole, 1994; Dixon & Ahrens, 1992; Nolen-Hoeksema, Girgus, & Seligman, 1992; Panak & Garber, 1992). However, others have not found a significant cognitive vulnerability–stress interaction (Bennett & Bates, 1995; Cole & Turner, 1993; Hammen, Adrian, & Hiroto, 1988). Analysis by age indicates that negative attributional style, in interaction with negative events, typically predicts depression in children only after age 11 (Garber, Keiley, & Martin, 2002; Abela, 2001; Lewinsohn, Joiner, & Rohde, 2001; Schwartz et al., 2000; Turner & Cole, 1994; Nolen-Hoeksema et al., 1992), and these same studies failed to find this predicted interaction in children prior to age 11.

Research with children under 11 indicates no gender difference in attributional style or that boys may have the more negative style (Mezulis, Hyde, & Abramson, 2006; Mezulis, 2004; Abela, 2001; Thompson, Kaslow, Weiss, & Nolen-Hoeksema, 1998; Nolen-Hoeksema, Girgus, & Seligman, 1991). However, research with adolescents shows girls having more negative styles (Hankin & Abramson, 2002; Nolen-Hoeksema & Girgus, 1994).

There is some indication, in a study of high school students, that negative attributional style is more strongly linked to depression for girls than for boys (Gladstone, Kaslow, Seeley, & Lewinsohn, 1997). Another high school study found that negative cognitive style mediates the gender difference in depressive symptoms, reducing the gender–depression association by 53% (Hankin & Abramson, 2002). There is also evidence of a more potent cognitive vulnerability–stress interaction for adolescent girls compared with adolescent boys. Prinstein and Aikins (2004), beginning with 10th graders (16-year-olds), administered measures of attributional style, peer acceptance/rejection (a stressor), and depressive symptoms. They recontacted the students 17 months later and again assessed depressive symptoms. They found a three-way interaction between attributional style, peer rejection, and gender. For girls, the combination of a negative attributional style and peer rejection at Time 1 predicted
depressive symptoms at Time 2, controlling for Time 1 depressive symptoms. The effect was not significant for boys.

Gender Differences

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 et al., 2003; Piccinelli & Wilkinson, 2000; Weissman et al., 1996; Kessler, McGonagle, 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 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 et al., 1993; Anderson, Williams, McGee, & Silva, 1987; Rutter, 1986), more girls than boys are depressed by ages 13 to 15 (Wade, Cairney, & Pevalin, 2002; Twenge & Nolen-Hoeksema, 2002; Hankin et al., 1998; Kessler et al., 1993).

The emergence of the gender difference in depression in adolescence has attracted the attention of medical and psychological researchers. The gender difference 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 (Cyranoowski, 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 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 (Kendler et al., 1993; Silberg et al., 1999), 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; A. Petersen, Sarigiani, & Kennedy, 1991).

Three prior reviews have been particularly influential in the conceptualization of the causes of gender differences in depression: those by Nolen-Hoeksema & Girgus (1994),
Nolen-Hoeksema and Girgus (1994) considered the following etiological factors: personality (e.g., 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 difference, except that the data on hormonal influences were sparse and conflicting.

Nolen-Hoeksema and Girgus (1994) further proposed three basic developmental models of how the gender difference might appear. According to Model 1, the same factors cause depression in girls and in boys, but one or more of these factors become more prevalent for girls than boys in early adolescence. Model 2 posits that different factors cause depression in girls compared with boys and that the factors for girls become more common in early adolescence, whereas the factors for boys do not become more common or increase only slightly. Model 3 rests on an interactive model that hypothesizes that, even before puberty, girls have more vulnerability factors for depression than boys do but that it is not until early adolescence, when major new challenges arise, that the vulnerability factors interact with the challenges to produce more depression. Nolen-Hoeksema and Girgus concluded that the evidence best supported Model 3 and the interactions specified by it.

Hankin and Abramson (2001), beginning with a generic cognitive vulnerability-stress model, articulated a host of elaborations that incorporated aspects of research on rumination, interpersonal theories of depression, genetic vulnerability for depression, and developmental factors (pubertal onset). These added factors were hypothesized to occur both proximally and distally. Among the distal factors they considered were genetic vulnerability and early environmental adversity. The conceptualization of cognitive vulnerability, a proximal factor, was elaborated to include ruminative response styles and negative cognitions about body image. Initial negative affect was introduced into the cognitive vulnerability-stress causal chain. In addition, Hankin and Abramson elaborated the types and frequencies of stressful negative events that may interact with vulnerabilities to produce depression. Stimulated by interpersonal theories of depression (Hammen, 1991), Hankin and Abramson considered stress-generation processes and specifically interpersonal stress generation. Distinguishing between independent negative events (those outside the
individual’s control) and dependent events (those to which the individual contributes), they noted the evidence that depression itself can lead to increases in interpersonal negative events because the person may seek reassurance excessively or withdraw from others, leading to rejection.

In contrast to the cognitive vulnerability–stress model proposed by Hankin and Abramson (2001), Cyranowski and colleagues proposed a model emphasizing the role of pubertal hormones in interaction with gender role intensification in adolescence. They suggested that pubertal hormones “sensitize females to the depressogenic effects of negative events” (Cyranowski et al., 2000). According to their model, this happens through the mechanism of increased affiliative needs driven by intensified gender role socialization as well as pubertal increases in oxytocin. This pubertal intensification in affiliative needs combines with other depressogenic vulnerability factors, such as a highly anxious temperament, insecure attachment, and poor coping skills, to create an overall depressogenic diathesis that interacts with stressors in the adolescent transition to produce depression.

The ABC model incorporates many elements of the previous models, but is distinctive in the following ways: (a) It is an integrative model, that is, it integrates key categories of factors. For example, genetic vulnerability (a biological factor) is presumed to influence temperament (an affective vulnerability), and temperament is presumed to contribute to negative cognitive style (a cognitive vulnerability), (b) The ABC model incorporates cutting-edge research not available at the time the other models were proposed. Examples are new research on genetic polymorphisms that confer vulnerability to depression, research on objectified body consciousness (OBC), and research on peer sexual harassment. The combination of these two primary differences allows the ABC model to provide greater elaboration on the processes by which depression vulnerability develops than was evident in prior reviews.

The ABC model is a vulnerability–stress model of the emergence of the gender difference in depression insofar as the authors posit that affective, biological, and cognitive vulnerabilities converge in early adolescence to form a depressogenic vulnerability that in the presence of stress, produces depression. An important component of this model is the cognitive vulnerability–stress pathway, as articulated by Hankin and Abramson (2001). However, affective vulnerability (temperament) is also integrated into this pathway. The authors provide conceptual and empirical evidence that temperament contributes to the development of cognitive vulnerability and, in addition, is a vulnerability factor itself to depressive outcomes. Biological vulnerability (genetic vulnerability, puberty) is also
integrated into this pathway. Again, the authors provide conceptual and empirical evidence that genetic vulnerability is a contributing factor and that it can be integrated into this pathway and that pubertal processes contribute to the development of cognitive vulnerability (particularly in the body image domain), exacerbate affective vulnerability, and evoke unique stressors that interact with other vulnerability factors. A conceptual diagram of the model is shown in Figure 1.3.

Figure 1.3: A conceptual diagram of the ABC model of the emergence of gender differences in depression in adolescence. (Depressogenic Vulnerability represents the collection of all the vulnerabilities, all of which are hypothesized to interact with negative events to yield gender differences in depression).
Developmental level and Depressive Symptoms

Depression can be found in a wide range of individuals, from infants to the elderly (Ginicola, 2007; DSM-IV-TR, 2000). However, research indicates that children’s experience of depression differs significantly from that evidenced in adults. The current perspective on depression, as indicated in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR, 2000) and the National Institute for Clinical Excellence (cf. Ginicola, 2007), suggests that although children and adults can have similar symptoms, their presentation may vary. Past reports have indicated the presence of irritability and aggression as symptoms of depression in children, whereas the symptoms of depression are not listed as evident in depressed adults (Weiss & Catron, 1994).

One possible reason for these findings is that children normatively develop cognitive and emotional skills over the course of their childhood (Zigler & Glick, 2001). Prior to reaching some of these cognitive milestones, they normatively present with a more behavioral and less cognitive orientation to their environment. Developmental research indicates that externalizing behaviors are present at low developmental levels (young children) and gradually change to internalizing behaviors over time.

Based upon the conceptualizations of behavior proposed by Achenbach (1978, 1991) and the action-thought theory (Zigler, 1961), depressive symptoms could be delineated into internalizing and externalizing symptoms. Internalizing symptoms are those that are more thought or emotions oriented (depressed mood, feelings of worthlessness/hopelessness, feelings of guilt, suicidal ideations/Attempts), whereas externalizing symptoms are behavioral and action oriented (irritability, aggressive behavior, changes in psychomotor patterns). Although aggressive behavior is not listed among the DSM-IV criteria, it is found in research reports on depression in young children (Shaffi & Shaffii, 1992). The remaining symptoms can be seen as physiological symptoms of depression with no visible differences across developmental levels. Kovacs & Paulauskas (1984) study, did not find a significant relationship between developmental level and depressive symptoms, which may be because this study had participants with a larger age range.

Ginicola (2007) examined symptomatology of depressive experiences among children and found that mental age is positively correlated (r=0.51) with an internalizing/total symptom ratio score and delineated between several individual symptoms. Mental age also predicted comorbidity with anxiety and conduct disorders, whereas children with a higher...
mental age presented more often with anxiety disorders. Gender was independently related to depressive symptoms, but minority status interacted with mental age. A developmental approach is useful in understanding children's depressive symptoms and has implications for both diagnosis and treatment of depression. If children experience depression differently, it follows that treatment options may also differ from that which is effective in adults.

Keeping in view what has been said in the preceding paragraphs, it can be noted that there is extreme paucity of research based on the association between developmental level and expression of depressive symptomatology in terms of absence/presence of a depressive symptom. The purpose of the present study is to refine and expand the relationship between developmental level and depressive symptomatology in non-clinical sample of male and female adolescents.

OBJECTIVES OF THE STUDY

The study starts with the following objectives:

1. To examine the role of developmental level in the experience of internalizing symptoms of depression.

2. To examine the role of developmental level in the experience of externalizing symptoms of depression.

3. To examine gender differences in the experience of internalizing symptoms in relation to developmental level.

4. To examine gender differences in the experience of externalizing symptoms in relation to developmental level.