CHAPTER-II

REVIEW OF LITERATURE AND FORMULATION OF HYPOTHESES

Borrowing from the description of hell in Milton's *Paradise Lost*, the American author Styron called his experience of deep clinical depression *darkness visible*, and severe depression certainly can be that: a suffocating, terrifying period of turmoil, extreme emotional pain, and perceived worthlessness. However, depression in the popular mind has become a catch-all expression for any psychological state from sadness and feeling blue to depressive psychosis and catatonia, and everything in between. Behavioural, cognitive, emotional, and physiological changes are typically evident in depression. The tendency for depressed people to withdraw and shut down is viewed as protective behaviours that serve to marshal limited cognitive resources toward the solution of complex problems. With the advent of antidepressant medication, there has been a tendency to see depression everywhere and to view any hint of prolonged sadness as a dysfunctional state to be rid of quickly and thoroughly [cf. Dunn, Whelton, & Sharpe, 2012].

In a recent paper, Andrews and Thomson (2009) suggested that the symptoms of depression are a highly adaptive strategy for conserving and focusing limited cognitive resources on complex social problems. Establishing and maintaining good social relationships is essential to most human activity. Many adaptations have evolved to promote social relations because these relations contribute in indispensable ways to human survival and reproduction (Buss, 2008). In turn, the need to belong and to be socially valued has adapted us to be hypersensitive to social indicators of social rank and status (Price, Sloman, Gardner, Gilbert, & Rohde, 1994; Gilbert, 1992), dominance or submission (Allan & Gilbert, 1997), attachment (Bowlby, 1969), and other relational cues.

Although many evolutionary psychology (EP) theorists have postulated some adaptive functions to depressive states (e.g., Sloman, 2009; Nesse, 2000; Price et al., 1994; Bowlby, 1980), only a few of these theorists have provided researchers with well-structured and testable models (Preti & Miotto, 2006). One of the most compelling of these EP models of depression is the social risk hypothesis formulated by Allen and Badcock (2003). Mild to moderate depression is thought to serve an adaptive purpose in our evolutionary history by decreasing the likelihood of exclusion from the all important social group. Allen and Badcock posited that socially relevant features of agency and affiliation are the determinants of
whether a depressive mechanism is activated or not. When this depressive mechanism is activated, a person will adopt a risk-averse strategy to social aspects of living in order to avoid ostracism from a valued group. The depressive mechanism triggers a cognitive hypersensitivity in the social domain prompting a reduction in an individual's propensity to engage in socially risky (e.g., confident, acquisitive) behaviors, submissive behaviors that diminish threat and the danger of conflict, and comfort-seeking behaviors that elicit social support from close and caring others.

The social risk hypothesis is an integrative evolutionary theory of depression that draws from three major Darwinian ideas: resource conservation (depression as a risk management strategy that inhibits behavior in suboptimal circumstances), social competition (depression as an involuntary de-escalating strategy signaling defeat in status conflicts), and attachment (depression as highlighting the importance of close and safe social bonds and the impact of their loss). The social risk hypothesis organizes these three ideas around a central principle of belonging and the need to avoid social exclusion. The dyad of defeat and social comparison are most representative of the social rank theories (e.g., Price, Solomon, Gardner, Gilbert, & Rohde, 1994; Gilbert, 1992) and have received much support in the field of evolutionary research.

In the context of the above presentation, it can be stated that depression is among the most common and debilitating psychological conditions afflicting modern society (Struijs et al., 2013; Ho et al., 2013; Alloy, Abramson, Keyser, Gerstein, & Sylvia, 2008; Kessler, 2002). With a lifetime prevalence estimated at 5–12% for men, and 10–25% for women, depression has negative consequences for millions of individuals and their families (APA, DSM-IV-TR, 2000). Classified as a mood disorder, depression is characterized by affective, physical, and motivation symptoms including low mood, insomnia, and impaired concentration. The impact of this disorder touches on many aspects of daily functioning, including interpersonal relationships, academic and vocational performance and physical health (DSM-IV-TR, 2000) [cf. Morley & Moran, 2011, p.1071].
The clinical and diagnostic features of the disorder are broadly similar in adolescents and adults (Thapar, Collishaw, Potter, & Thapar, 2010; Lewinsohn, Pettit, Joiner, & Seeley, 2003). The two main classification systems (international classification of diseases-10 [ICD-10] and the American diagnostic and statistical manual of mental disorders-IV [DSM-IV] define depression similarly, although DSM-IV makes one exception for children and adolescents, whereby irritable rather than depressed mood is allowed as a core diagnostic symptom (DSM-IV, APA, 1994). Nevertheless, depression in adolescents is more often missed than it is in adults (Leaf, Alegria, & Cohen et al., 1996), possibly because of the prominence of irritability, mood reactivity, and fluctuating symptoms in adolescents. Depression can also be missed if the primary presenting problems are unexplained physical symptoms, eating disorders, anxiety, refusal to attend school, decline in academic performance, substance misuse, or behavioural problems [cf. Thapar, Collishaw, Pine, & Thapar, 2012].

In the context of the above assertion, it can be stated that unipolar depressive disorder is a common mental health problem in adolescents worldwide (Lopez et al., 2006) with an estimated 1 year prevalence of 4–5% in mid to late adolescence (Costello, Erkanli, & Angold, 2006; Costello, Egger, & Angold, 2005). Depression in adolescents is a major risk factor for suicide, the second-to-third leading cause of death in this age group (Windfuhr, While, & Hunt et al., 2008), with more than half of adolescent suicide victims reported to have a depressive disorder at time of death (Hawton & Heerigen, 2009). Depression also leads to serious social and educational impairments (Fletcher, 2008; Lewinsohn, Rohde, & Seeley, 1988), and an increased rate of smoking, substance misuse, and obesity (Keenan, Hammen, & Brennan, 2007; Hasler, Pine, & Kleinbaum et al., 2005). Thus, to recognise and treat this disorder is important.

Criteria for ICD-10 depressive episode

Core symptoms (at least two must be present)

- Depressed mood present for most of the day and almost every day
- Loss of interest or pleasure in activities
- Decreased energy or increased susceptibility to fatigue
Associated symptoms

- Loss of confidence or self-esteem
- Unreasonable feelings of self-reproach or excessive inappropriate guilt
- Recurrent thoughts of death or suicide, or any suicidal behaviour
- Diminished ability to think or concentrate
- Change in psychomotor activity, agitation, or retardation
- Sleep disturbance
- Change in appetite with corresponding change in weight

At least four of these symptoms must be present for 2 weeks to diagnose a mild depressive episode, six to diagnose a moderate depressive episode, or eight for a severe depressive episode.

Epidemiology

Depression in prepubertal children is less common than depression in adolescents or adults, and seems to differ from these disorders with respect to some causative, epidemiological, and prognostic features (Harrington, 2001; Thapar & McGiffin, 1994).

The prevalence of depression in children is low (<1% in most studies) (Kessler, Avenevoli, & Reis, 2001) with no sex differences, and then rises substantially throughout adolescence (Green, McGinnity, Meltzer, Ford, & Goodness, 2005). Many factors could explain the recorded post-pubertal rise in prevalence because adolescence is a developmental period characterised by pronounced biological and social changes (Patton & Viner, 2007; Cyranowski, Frank, Young, & Shear, 2000). The most commonly postulated contributors are puberty and brain and cognitive maturation. They include enhanced social understanding and self-awareness (Blakemore, 2008), changes in brain circuits involved in responses to reward and danger, and increased reported stress levels, especially in girls (Nelson, Leibenluft, McClure, & Pine, 2005; Silberg, Pickles, & Rutter, 1999). Median 12-month prevalence estimates in mid to late adolescence are roughly similar to those seen in adulthood (4–5%) (Costello, Erkanli, & Angold, 2006; Costello, Egger, & Angold, 2005), with the cumulative probability of depression rising from around 5% in early adolescence to as high as 20% by the end of that time (Lewinsohn, Rohde, Klien, & Seeley, 1999; Hankin, Abramson, Moffitt, Silva, McGee, & Angell, 1998). However, population prevalence estimates vary widely across studies and in different countries, (Pillai, Patel, Cardozo, Goodman, Weiss, &
Patterns of service provision vary so much between studies and countries that comparisons of clinic-referred samples are not meaningful. Although some researchers suggest higher rates of depression in adolescents in low income and middle-income countries (Fleitich & Goodman, 2004), no consistent evidence of such systematic variation exists (Pillai, Patel, Cardozo, Goodman, Weiss, & Andrew, 2008; Lopez, Mathers, Ezzati, Jamison, & Murray, 2006; Fleitich & Goodman, 2004). Nevertheless, because of the young age distribution in low income and middle-income countries, the overall number of affected adolescents will be substantially greater in these countries than in high-income countries (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006). Despite this high burden, research has focused almost exclusively on high-income countries.

Further, more than 70 percent of children and adolescents with depressive disorders or other serious mood disorders do not receive appropriate diagnosis and treatment (cf. Bhatia & Bhatia, 2007). Possible reasons for this may be the stigma attached to these disorders, an atypical presentation, a lack of adequate child mental health training for health care professionals, inadequate number of child psychiatrists, and inequalities in mental health care insurance.

Underdiagnosis and undertreatment are greater problems in children younger than seven years, in part because of this age group’s limited ability to communicate negative emotions and thoughts with language and consequent tendency toward somatization. Thus, young children with depression may present with general aches and pains, headaches, or stomachaches. Additionally, he or she may minimize the child’s depressive symptoms through a lack of awareness or an unwillingness to recognize symptoms that may be similar to his or her own.

**Risk Factors underlying Depressive Tendencies (Causes and Pathogenesis)**

**Biological Risk Factors**

Distal risks such as inherited factors and adversity in early life might directly and indirectly predispose to depression. Such risks could mediate their effects through temperament and personality attributes (negative emotionality, decreased positive emotionality and attentional control, behavioural inhibition, and neuroticism) and cognition
(Garber, 2006). However, consistent evidence is sparse as to whether such characteristics are risk factors, mediators, or consequences of depression.

Distal factors, together with hormonal and maturational changes can also alter individual sensitivity to stressors. These risk factors in turn affect proximal biological risk mechanisms such as changes in the neuroendocrine system and brain structure and function, although no one pathway has been identified as causal. Prevention and treatments have been targeted at reduction of early and later adversities, modification of ways of thinking and feeling, and reduction of core depression symptoms by alteration of biological pathways with medication.

**Depression and Supportive Attachments**

Numerous theorists have also suggested that life's earliest relationship experiences have important implications for depressive vulnerability (Moran, Neufeld-Bailey, & DeOliviera, 2008; Ingram, 2001, 2003; Bowlby, 1980; Beck, 1967, 1987). John Bowlby (1980, 1973, 1969) proposed that infants develop bonds, or attachments, through repeated interactions with primary caregivers over the first few years of life. These attachments – with deep evolutionary roots related to survival and reproductive fitness – have critical consequences for an individual's social and emotional functioning and associated developmental outcomes. In particular, Bowlby proposed that these experiences have implications for the later emergence of emotional disturbances such as depression: “The psychology and psychopathology of emotion is found in large part to be the psychology and psychopathology of affectional bonds” (Bowlby, 1977).

![Diagram](image-url)

**Figure 2.1:** Proposed pathway linking early attachment experiences to later depression (cf. Morley & Moran, 2011).
Consistent with Bowlby's hypothesis, research over the past few decades utilizing self-report measures with adult participants has revealed associations between depressive symptoms in adults and self-reported attachment style (Dozier, Stovall-McClough, & Albus, 2008; Williams & Riskind, 2004). Such findings suggest that those with stable and supportive attachments to important others are much less likely to suffer from depression than those with less supportive relationships.

This finding adds support to the recent work of Sloman and his colleagues (Sloman, 2008, Gilbert, & Hasey, 2003) who acknowledged not only the operation of social rank in depression but also the protective function of an attachment system which promotes closeness and proximity, sensitivity to distress, as well as the survival benefits of a secure attachment. Social isolation, feelings of being unloved, fears of abandonment and general negative relational patterns in the social realm have demonstrated a strong association between adult attachment style and depressive symptomatology in a broad range of directed research (e.g., Bifulco, Moran, Ball, & Bernazzani, 2002; Roberts, Gotlib, & Kassel, 1996). Similarly, Dunn et al. (2012) also found that strong affiliative bonds are linked to an absence of depressive features. Submission and interpersonal sensitivity initiates a feedback loop that operates to bolster one's social investment potential (or self-esteem) within the group, thus deflecting social ostracism and agonistic encounters through a gradual elicitation of support from higher ranking conspecifics.

**Cognitive Vulnerability to Depression**

Shortly after John Bowlby developed his theory of attachment, Aaron Beck began working on a hypothesis to account for individual variation in vulnerability to depression, i.e., a theory to account for the fact that some individuals become depressed following stressful life events, while others react less severely (Beck, 1967, 1987). Beck argued that depression should no more be thought of as an affective disorder than should scarlet fever be described as a disease of the skin, i.e., that external presentation should not be confused with the underlying pathology. Rather, he proposed that the affective, motivational and even physical symptoms of depression were primarily a product of the way an individual perceives himself, the world and the future — that depression is primarily a disorder of cognition.

Beck observed that his patients' negative cognitive patterns most often preceded unpleasant affective experiences and that the content of these cognitive distortions closely
paralleled that of the emotional response, suggesting a causal link. Beck's (1967) initial empirical observations were consistent with this inference: patients reported that maladaptive cognitions preceded congruent negative affective experiences. Beck subsequently developed an influential cognitive theory of differential susceptibility to depression following stressful life events (Beck, 1967, 1987). He (Beck, 1967, 1987) proposed that individuals develop a self-concept which reflects their representations of the self, world and future based on the attitudes and opinions communicated to them by important others during childhood. Subsequent experiences are then interpreted in ways that are consistent with the content of this self-concept. For example, an individual with a negative view of the self, world and future will tend to focus selectively on disappointing aspects of a situation and, in the process, further consolidate their negative self-concept. Through repeated experiences, such as this, the view of the self will solidify, becoming a more permanent cognitive structure, or a schema. While it may lay dormant, significant life stress can activate an individual's negative self-schema, influencing information processing, and causing external stimuli to be screened, coded and evaluated within the framework of this schema (Dozois & Beck, 2008).

Aspects of Beck's theory have been incorporated in a related but more specific account of vulnerability to depression: helplessness theory (Nolen-Hoeksema, Girgs, & Seligman, 1986; Abramson, Seligman, & Teasdale, 1978). The roots of helplessness theory can be traced to studies observing cognitive, emotional and motivational responses of dogs to repeated unavoidable electric shocks. Over time, the dogs passively accepted the shocks, making no attempts to escape even when it was possible to do so (Schueller & Seligman, 2008). Replication via parallel studies with human participants produced nearly analogous results but not all participants responded to uncontrollable events with helplessness (Abramson et al., 1978). They argued that helplessness arises where an individual makes stable, internal and global attributions about such negative experiences (Alloy et al., 2008; Nolen-Hoeksema et al., 1986). Various studies have found associations between these helpless explanatory styles and symptoms of depression in adults (Schueller & Seligman, 2008; Abramson et al., 1978) and in children and adolescents (LaGrange et al., 2008; Gladstone & Kaslow, 1995; Nolen-Hoeksema et al., 1986).
Self-esteem and Depression

Many theories of depression postulate that low self-esteem is a defining feature of depression (e.g., Abramson, Seligman, & Teasdale, 1978; Brown & Harris, 1978; Blatt, D’Afflitti, & Quinlan, 1976; Beck, 1967). Indeed, numerous studies have documented strong concurrent relations between low self-esteem and depression (Joiner, Katz, & Lew, 1999; Roberts & Monroe, 1992; Kernis, Grannemann, & Mathis, 1991; Lewinsohn, Hoberman, & Rosenbaum, 1988). However, the nature of this relation—specifically, the temporal order—remains unclear. Does low self-esteem lead to depression, does depression contribute to the development of low self-esteem or are they reciprocally related?

Two dominant models exist in the literature. The vulnerability model hypothesizes that low self-esteem serves as a risk factor for depression, especially in the face of major life stressors (e.g., Butler, Hokanson, & Flynn, 1994; Metalsky, Joiner, Hardin, & Abramson, 1993; Whisman and Kwon, 1993; Roberts & Monroe, 1992; Beck, 1967). For example, according to Beck’s (1967) cognitive theory of depression, negative beliefs about the self—one of three central components of depressive disorders—are not just symptomatic of depression but play a critical causal role in its etiology.

In contrast, the scar model hypothesizes that low self-esteem is an outcome of depression rather than a cause. Specifically, depression is assumed to persistently deteriorate personal resources such as self-esteem, even after remittance of a depressive episode; that is, episodes of depression may leave scars in the individual’s self-concept that progressively chip away at self-esteem over time (cf. Coyne, Gallo, Klinkman, & Calarco, 1998; Coyne & Whiffen, 1995, Rohde, Lewinsohn, & Seeley, 1990; Zeiss & Lewinsohn, 1988).

Vulnerability Model

The vulnerability model states that low self-esteem is a risk factor for future depression. The underlying assumption of the vulnerability model is that self-esteem, like other personality traits, is a diathesis exerting causal influence in the onset and maintenance of depression. Low self-esteem might contribute to depression through both interpersonal and intrapersonal pathways. One interpersonal pathway is that some individuals with low self-esteem excessively seek reassurance about their personal worth from friends and relationship partners, increasing the risk of being rejected by their support partners, increasing the risk of depression (Joiner, 2000; Joiner et al., 1999; Joiner, Alfano, &
Metalsky, 1992). A second interpersonal pathway is that individuals with low self-esteem seek negative feedback from their relationship partners to verify their negative self-concept, which may further degrade their self-concept (Giesler, Josephs, & Swann, 1996; Joiner, 1995; Swann, Wenzlaff, & Tafarodi, 1992). A third interpersonal pathway is that low self-esteem motivates social avoidance, thereby impeding social support, which has been linked to depression (cf. Ottenbreit & Dobson, 2004). Relatedly, individuals with low self-esteem are more sensitive to rejection and tend to perceive their relationship partner’s behavior more negatively, thereby undermining attachment and satisfaction in close relationships (Murray, Rose, Bellavia, Holmes, & Kusche, 2002; Murray, Holmes, & Griffin 2000). A fourth interpersonal pathway is that individuals with low self-esteem engage in antisocial behaviors, such as aggression and substance abuse that might contribute to their feeling excluded and alienated from others (Donnellan, Trzesniewski, Robins, Moffitt, & Caspi, 2005).

An intrapersonal pathway explaining how low self-esteem contributes to depression might operate through rumination. The tendency to ruminate about negative aspect of the self is closely linked to depression (Spasojevic & Alloy, 2001; Tapnell & Campbell, 1999; Nolen-Hoeksema, 1991, 2000; Nolen-Hoeksema & Morrow, 1991; Morrow & Nolen-Hoeksema, 1990). Mor and Winquist’s (2002) meta-analysis of correlational and experimental studies showed that self-focused attention strongly and causally influences negative affect (including depression) and, moreover, that a ruminative self-focus has a particularly strong impact (compared with a reflective self-focus).

**Scar Model**

The scar model, in contrast to the vulnerability model, states that low self-esteem, like other correlates of depression such as negative attributional style, might be a consequence of depression rather than a causal factor (Rohde et al., 1990; Lewinsohn, Stinmetz, Larson, & Franklin, 1981). Indeed, it is conceivable that the self-concept and self-esteem are permanently changed by the experience of depression, especially after major depressive episodes. Again, depression might impair an individual’s self-esteem through both intrapersonal and interpersonal pathways. A possible intrapersonal pathway is that the experience of depression might influence self-esteem by persistently altering the way in which individuals process self-relevant information; for example, the chronic negative mood associated with depression may lead the individual to selectively attend to, encode, and retrieve negative information about the self, resulting in the formation of...
more negative self-evaluations. One interpersonal pathway is that depressive episodes may damage important sources of self-esteem such as close relationships or social networks. Another interpersonal pathway is that depression might change how the individual is perceived by others.

These representations may be relatively persistent and cause the individual to be treated by others with low regard or in ways that minimize the individual's self-esteem, even if the depression has already remitted (Joiner, 2000). Another pathway is that the disclosure of depression to others may not only shape how others perceive the individual, but also amplify the intrapersonal effects of depression on the self-concept (cf. Tice, 1992), thereby combining interpersonal and intrapersonal processes. Of course, the vulnerability model and the scar model are not mutually exclusive because both processes (i.e., low self-esteem contributing to depression and depression eroding self-esteem) might operate simultaneously.

The Diathesis-Stress Model of Low Self-Esteem and Stressful Events

A growing body of research suggests that low self-esteem contributes to the development of depression. Overall, the findings support the "vulnerability model," which states that low self-esteem operates as a risk factor for depression (Metalsky, Joiner, Hardin, & Abramson, 1993; Beck, 1967). Many previous studies used prospective designs and controlled for prior levels of both self-esteem and depression (e.g., Orth, Robins, & Roberts, 2008; Orth, Robins, Trzesniewski, Maes, & Schmitt, 2008; Kernis et al., 1998; Roberts & Monroe, 1992). The effect of low self-esteem on depression holds for men and women, from adolescence to old age, and after controlling for content overlap between self-esteem and depression scales (Orth et al., 2008). Moreover, prior research has failed to support an alternative model of the relation between self-esteem and depression – the "scar model" – which hypothesizes that low self-esteem is an outcome rather than a cause of depression (Ormel, Oldehinkel, & Vollebergh, 2004).

Many diathesis-stress models of depression consider low self-esteem to be a predisposing factor for the development of depression (e.g., Hammen, 2005; Metalsky et al., 1993; Brown & Harris, 1978; Beck, 1967). In the face of challenging life circumstance, individuals with low self-esteem are assumed to have fewer coping resources and thus are prone to depression, whereas those with high self-esteem are assumed to have better coping resources and thus avoid spiraling downward into depression. In other words, the experience
of stressful events generally contributes to depression, but individuals with relatively high self-esteem are buffered against this effect (and, conversely, individuals with relatively low self-esteem are more vulnerable to this effect). If self-esteem buffers individuals against the deleterious consequences of stressful life events, then low self-esteem and stressful events should have an interactive effect on subsequent depression (Hankin, Fraley, Lahey, & Waldman, 2005; Prisciandaro & Roberts, 2005; Lewinsohn, Solomon, Seeley, & Zeiss, 2000; Ruscio & Ruscio, 2000). The buffering hypothesis is a commonly accepted view of the causal relationship among self-esteem, stressful events, and depression. For example, Abela, Webb, Wagner, Ho, & Adams (2006) state that following negative events, “protective factors, such as high self-esteem, may prevent the outcome of depressive symptoms by decreasing the negative impact of depressogenic thoughts on the affective, cognitive, behavioral, and physiological symptoms of depression” and Roberts (2006), in a review on self-esteem from a clinical perspective, states that one of the themes that emerges from the depression literature is that self-esteem “interacts with other risk factors, such as life stress and attributional style, in the prediction of depression”.

The self-esteem buffering hypothesis has been tested in numerous studies, that is, whether the effects of stressful events on subsequent depression (controlling for prior levels of depression) were stronger for low versus high self-esteem individuals. Four studies confirmed the hypothesized interaction (Abela, 2002; Frenandez, Mutran, & Reitzes, 1998; Ralph & Mineka, 1998; Metalsky, 1993); seven studies failed to find the hypothesized interaction (Southall & Roberts, 2002; Kernis et al., 1998; Cheng & Lam, 1997; Butler, Hokanson, & Flyn, 1994; Roberts & Morone, 1992; Murrell, Meeks, & Walker, 1991; Lewinsohn, Hoberman, & Rosenbaum, 1988); three studies failed to find the hypothesized two-way interaction but found evidence of a three-way interaction of self-esteem, stressful events, and third variable such as dysfunctional attitudes (Abela & Skitch, 2007; Abela et al., 2006; Robinson, Garber, & Hilsman, 1995); and one study reported an interaction effect showing the opposite pattern (the effect of stressful events on depression was stronger for high vs. low self-esteem individuals; Whisman & Kwon, 1993).
Hypothesis 1

Self-Esteem

Stressful Events

Depression

Hypothesis 2

Self-Esteem

Stressful Events

Depression

Hypothesis 3

Stressful Events

Self-Esteem

Depression

Hypothesis 4

Self-Esteem

Depression

Stressful Events
**Hypothesis 5**

![Figure 2.2: Hypotheses concerning the relations among self-esteem, stressful events, and depression. Hypothesis 1: ("self-esteem buffering"); Self esteem buffers the effects of stressful events on depression. Hypothesis 2: stressful events mediate the effects of low self-esteem on depression. Hypothesis 3: Low Self-esteem mediates the effects of stressful events on depression. Hypothesis 4: Stressful events account for the relation between low self-esteem and depression. Hypothesis 5: Low self-esteem accounts for the relation between stressful events and depression.](image)

**Depression in Relation to Hostility and Locus of Control**

Ever since Abramowitz (1969) reported a relation between scores on the Rotter Internal–External Scale (I-E) and the Guilford Depression Scales (externals scoring significantly higher than internals), researchers and theorists have shown much interest in clarifying the relationship (Becker & Lesiak, 1977; Fogg, Kohaut, & Gayton, 1977; Naditch, Gargan, & Michael, 1975; Calhoun, Cheney, & Dawes, 1974; Goss & Morosko, 1970). Several studies have yielded significant positive correlations between external-control scores and depression scores although Fogg et al. (1977) points out that the relation appears to hold for males but not females. Reviews of this literature can be found in Lefcourt (1966, 1972) and Joe (1971). Thus, some evidence exists for Abramowitz’s interpretation that depression is associated with external control. Also, the external control-depression interpretation is in line with Seligman’s theory of helplessness (Miller & Seligman, 1975; Seligman, 1975), which postulates depression as a function of the person’s inability to control reinforcement outcomes.

The significant correlations between external locus of control and depression are not consistent with the general psychoanalytic view of the depressed person as highly self-critical, accepting of responsibility for the effects of his actions, and overly sensitive to...
approval by others (Cameron, 1963). Likewise, Schwartz (1964) has suggested that the
depressed person experiences a strong sense of power and responsibility. Golin & Terrell
(1977) found mildly depressed college students set high standards of success and had higher
levels of aspiration than the non-depressed samples. And, another study on self-
reinforcement (Rozensky, Rehm, Pry, & Roth, 1977) indicates that the depressed individual
sets higher standards for self-reinforcement and is more self-punishing for failure.

Naditch, Gargan, & Michael (1975) studied denial, anxiety, locus of control and the
discrepancy between aspirations and achievements as components of depression. The
subjects were 547 men in their second week of army basic training at Forts Monmouth and
Dix in New Jersey. Depression was negatively correlated with denial and positively
correlated with anxiety, locus of control, and the discrepancy between aspirations and
achievements (discontent). Locus of control was positively correlated with discontent and
anxiety, and negatively correlated with denial. There were interaction effects between locus
of control and discontent, between locus of control and anxiety, and between anxiety and
denial when these terms were regressed on depression. The importance of denial and anxiety,
and the interaction effect of locus of control and discontent were discussed as important
components of depression.

Peterson, Sushinsky, & Demask (1978) administered the Rotter's Locus of Control
Scale and the MMPI to 39 inpatients at a Private Psychiatric Hospital. The results do not
show consistent differences in external locus of control scores between depressed subjects
and non-depressed subjects across the varying methods of defining depression. The
correlations between locus of control and the MMPI Scales, and a review of the
literature suggests that the types or degree of depression may have different dynamics
and some types may be related to locus of control while other types of depression are
not related to locus of control.

The studies which report a simple relation between external scores and depression
have generally limited psychopathology to depression and have studied college student
populations (Fogg, Kohaut, & Gayton, 1977; Calhoun, Cheney, & Dawes, 1974). There are
also some inconsistencies in the findings of an external-depression relation for college
students as Golin & Terrell (1977) failed to find a significant correlation between locus of
control and depression in their college sample.
Further, there is the issue of the comparability of mildly depressed college students with depressed psychiatric patients. This literature suggests that the relation between depression and external control is not a simple one to one relation; a great deal more information is needed to understand the relation. It might help to adopt the view that multiple causes and types of depression may exist and to assess varying definitions and populations, it may well be that both general interpretations of depression, i.e., lack of internal locus of control and presence of internal locus of control with high standards for self-approval, are correct but for different groups of depressed people. Until additional research is carried out the relation between locus of control and depression will remain unclear, just as will the status of the differing theoretical interpretations of depression.

Further, many formulations of psychopathology maintain that underlying hostility is a central component of depression. Psychodynamic theorists (Rubinfine, 1968; Alexander, 1948) have described the role of introjected hostility in depression, while behavioral theorists (Ferster, 1973) have noted the suppression of anger and aggressive acts by depressed individuals.

Empirical investigations have confirmed the association between hostility and depression, although there has been disagreement regarding the exact nature of this association. As reviewed by Akiskal & McKinney (1975) the Abrahamn-Freud model viewed depression as the turning of one’s aggression inside rather than directing it at the appropriate object. This hypothesis was supported by Becker & Lesiak’s study (1977) which showed that depression, measured by the Beck Depression Inventory, was correlated positively with both covert hostility and external control. Overt forms of hostility were unrelated to depression. Gershon, Cromer, & Klerman (1968) found that inwardly directed hostility called “hostility-in” was positively correlated with depression as expected by Abraham and Freud; they also found another pattern of hostility. This pattern was marked by outwardly directed hostility called “hostility-out” in patients with hysterical symptoms. Patients so characterized tended to show an increase in outward hostility with deepening depression. Such findings may support Bibring’s (1953) idea that underlying process in depression does not necessarily involve the aggressive impulses but the vicissitudes of ego functioning.

Most studies (Kendell, 1970; Gershon, Cramer, & Klerman, 1968; Foulds, Caine, & Creasey, 1960) have concluded that hostility is directed inwardly by depressed individuals.
However, other studies have indicated either that hostility is directed outwardly by depressed individuals (Foulds, 1965; Wessman, Ricks, & Tyl, 1960) or that both inwardly and outwardly directed hostility occur in depression (Friedman, 1970; Grinker; Miller, Sabshin, Nunn & Nunnally, 1961).

Moore & Paolillo (1984) examined the relationships of the criterion variable, depression, to seven predictor variables, hopelessness, external locus of control, personal responsibility of external locus of control, general hostility, overt hostility, covert hostility, and length of treatment. The data were measures of depression, hopelessness, locus of control, personal responsibility of locus of control, general hostility, overt hostility, and covert hostility from 317 outpatients of a neural mental health center in southeastern Wyoming. Length of treatment and descriptive data were also collected. The descriptive data included date of birth, gender, diagnosis according to the DSM II of the APA. Over 50% of the variance of depression was explained by hopelessness, 7% of the remaining variance was explained by covert hostility and approximately 1% was explained by length of treatment. These findings support the views of depression in which hopelessness and covert hostility were regarded as important correlates of depression. No relationship between depression and overt hostility was noted, suggesting that advocating outward expression of hostility will not alleviate depression.

Contradictory findings regarding the relationship of hostility and depression may reflect a failure to consider the dynamic relationship between hostility and depression.

**Gender Differences in Depression**

The prevalence of depression in children and adolescents is on the rise, and depressive illness in these developmental periods is associated with significant impairment in multiple social domains. Elevated risk for the disorder begins in the early teens and continues to rise in a linear fashion throughout adolescence, with lifetime rates estimated to range from 15% to 25% by late adolescence (Rao, 2006; Kessler, Berglund, Demler, Jin Merikangas, & Walters, 2005). There is evidence that early depressive episodes recur and persist into adult life along with ongoing psychosocial difficulties, including disruption in interpersonal relationships, early pregnancy, low educational attainment, poor occupational functioning, and unemployment, as well as elevated risk for suicidal behavior (Rao, 2006).
Depressive symptoms among youths are often attributed to the normal stress of adolescence; misdiagnosed as primarily conduct, attentional, or substance abuse disorders; or seen as a stage the youths are going through. Overlooking MDD among youths can have tragic consequences. Depression is associated with an increased risk of suicide (Birmaher, Ryan, & Williamson et. al., 1996), and teen suicide rates have nearly tripled in the last 50 years (cf. Saluja, Iachan, Scheidt, Mary, Overpeck, Wenyu, Jay, & Giedd, 2006). Those who experience depression at an early age often struggle with depression throughout their lives (Lewinsohn, Rohde, Klein, & Seeley, 1999) and in many cases, early onset of depression predicts more severe depression during adulthood (Weissman, Wolk, & Goldstein, 1999). Even subclinical depression during adolescence increases the risk of MDD as an adult 2- to 3-fold (Pine, Cohen, Gurley, Brook, & Ma, 1998).

One of the most consistent psychiatric epidemiological findings is that depression is twice as common in women as in men (Kessler et al., 1994). Numerous theories, ranging from biologically based (Blehar & Oren, 1995; Wolk & Weissman, 1995) to psychosocial theories (Nolen-Hoeksema, Larson, & Grayson, 1999; Nolen-Hoeksema, 1995), have been proposed to account for women’s vulnerability to depression. However, in recent years, the accuracy of these theories has been disputed. Data suggest that the sex gap in depression may not be due to actual differences in prevalence rates but rather to a number of other factors, such as men’s unwillingness to seek help for depression, men’s tendency to underreport symptoms of depression (Angst et al., 2001; Courtenay, 2000), and clinician sex bias in depression diagnoses (Potts, Burnam, & Wells, 1991). Another proposed explanation for the gap in prevalence rates of depression is that men may exhibit depressive symptomatology in ways that differ from women. A preponderance of anecdotal evidence suggests that men manifest depression differently than women and that this atypical symptom presentation is even more evident in men who adhere to restrictive masculine norms (Coehran & Rabinowitz, 2000). Another study (Magovecevic & Addis, 2008) found that men who adhered strongly to masculine norms were more likely to endorse externalizing symptoms than prototypic symptoms of depression, as captured by existing measures of depression.

Studies of high school students (Brooks, Harris, Thrall, & Woods, 2002; Lewinsohn, Rohde, Klein, & Seeley, 1999; Lewinsohn, Rohde, & Seeley, 1998) have shown that similar to depression among adults, the incidence of depression among adolescents is greater for
females than males. These sex differences have been reported to emerge between the ages of 13 and 15 years, and prior to this age, rates of depression appear to be similar for young boys and girls (West, 2003; Sweeting, Bron, Shea, & Steiner, 2002; Hankin & Abramson, 1999; Petersen, Compas, Brooks-Gunn, & Stemmier, 1993).

Although studies indicate that there are no overall sex differences in depressive symptom presentation, severity, mean number of symptoms, duration, or prevalence of a single depressive episode (Hildebrandt, Stage, & Kragh-Sørensen, 2003), some research reveals that qualitative differences do emerge when specific depressive symptoms are examined. For example, men diagnosed with depression are more likely to have comorbid alcohol abuse problems (Angst et al., 2002; Roecloff, Funk, Unutzer, Tang, & Well, 2001). Men are also more likely than women to report engaging in activity to avoid thinking about depression and to report somatic complaints, concerns regarding work success, inability to make decisions and general social withdrawal (Kessing, 2006; Winkler et al., 2004; Kornstein et al., 2000; Vredenburg, Krames, & Flett, 1986; Kleinke, Stanneski, & Mason, 1982; Padesky & Hammen, 1981; Hammen & Padesky, 1977). On the other hand, men are less likely than women to report crying (Salokangas, Pacriev, Sohlman, & Lehtinen, 2002; Hammen & Padesky, 1977).

The qualitative sex differences that emerge in research examining depressive symptoms seem to indicate that men in general report symptoms more acceptable to dominant masculine norms, such as preoccupation with work failure, social withdrawal, avoidance of affect, and self-medication (Kessing, 2006; Winkler et al., 2004; Kornstein et al., 2000; Vredenburg et al., 1986; Kleinke et al., 1982; Padesky & Hammen, 1981; Hammen & Padesky, 1977). However, findings related to sex differences are often inconsistent because of their focus on mean differences between the two sexes. For example, some studies have found that women are more likely than men to endorse somatic complaints (Wenzel, Steer, & Beck, 2005; Silverstein, 2002).

Scholars in masculinity theory and research have speculated about the role of gender socialization as a contributing factor to the qualitative differences in depressive symptom presentation. It has been postulated that masculine gender socialization and adherence to dominant masculine norms may have an impact on men’s acknowledgement and presentation of depressive affect (Hart, 2001; Cochran & Rabinowitz, 2000; Pollack, 1998; Real, 1997). Masculine gender socialization emphasizes characteristics such as stoicism, independence,
physical toughness, restrictive emotional expression, competition, and the avoidance of anything “feminine” (O’Neil, Good, & Homes, 1995; Pleck, 1981; Brannon, 1976). The masculine gender socialization framework is an individual differences framework that assumes that the degree to which individual men adhere to dominant masculine norms may differ. This framework may account for differences among individual men’s symptom presentation. For example, depressive affect and behaviors that accompany depression (e.g., crying) have been stereotyped as feminine (Warren, 1983) and may be particularly aversive to a man who adheres to the norms of stoicism and toughness. Such affect may not be aversive to another man who does not adhere to these particular norms.

In recent years, clinicians have theorized, based on their experience with men in clinical practice, that men who adhere more strongly to masculine norms may exhibit their depression in ways that are more congruent with those norms (Cochran & Rabinowitz, 2000; Pollack, 1998; Real, 1997). Clinicians have postulated that these men may tend to mask their distress by engaging in behaviors such as drinking or using drugs, aggression, reckless behavior, withdrawing from family and friends, and overfocusing on work. These theoretical and clinical speculations imply that men experience depression in the prototypic way (as described by DSM-IV-TR criteria, American Psychiatric Association, 2000) but tend to avoid or deny the experience because it is incongruent with the male role. It has also been suggested that some men may substantially “mask” an underlying depression with the externalizing behaviors described above (Real, 1997).

Although the concept of a “masked” depression in men is theoretically appealing, it is difficult to examine empirically and challenging to validate. Put simple, if depression is present in an individual, but “masked” in some way, it is virtually impossible to establish the presence of the disorder independent of the presence of prototypic symptoms (as described by DSM-IV-TR criteria). Nonetheless, a number of studies have attempted to validate the construct of a male depressive syndrome and have developed measures aimed at capturing depressive symptomatology (Diamond, 2005; Moller-Leimkuhler, Bottlender, Straub, & Rutz, 2004; Rutz, 1996). Unfortunately, there are a number of methodological concerns regarding the reliability and validity of these measures. For example, the more widely used Gotland Scale for Male Depression (Rutz, 1996), was developed to capture symptoms of depression as exhibited by suicidal men on the Island of Gotland in Sweden. The purpose of this measure was to help primary care clinicians diagnose depression in men in an attempt to
decrease male suicide rates on the island (Rutz, 2001). In terms of reliability, the items are exceptionally lengthy, and a number of statements are double-barreled (DeVellis, 1991).

The association between the risk behaviors and depressive symptoms in this study are consistent with previous research on older adolescents. Youths who reported using substances were more likely to report depressive symptoms than other youths, suggesting that those who use substances might be self-medicating, even at this young age. We should also consider the possibility that substance use may actually precede the onset of depressive symptoms, as suggested by previous research (Degenhardt, Hall, & Lynskey, 2003). Furthermore, youths who reported suffering from frequent somatic symptoms were more likely to report depressive symptoms than other youths. Previous research has found that girls are more likely than boys to admit to internalizing symptoms such as depression and anxiety. In contrast, boys have a greater tendency to externalize symptoms through “acting out” (Ostrov, Offer, & Howard, 1989). Thus, the higher proportion of females reporting somatic symptoms is not surprising.

Results from this study also suggest that depression is a substantial problem facing young adolescents. The study highlights the need for more intervention at the middle school level. Because depressive symptoms are likely to coexist with other adolescent problem behaviors, such as bullying and substance use, young adolescents who are involved in such behaviors might be depressed as well. Youths who suffer from frequent somatic symptoms (e.g., frequent absenteeism and/or complaints of headaches and other physical symptoms) are more likely to be depressed. Practitioners who work with youths who have such symptoms/behaviors should consider screening them for depression; successful treatment of depression could lead to many other positive health and behavioral outcomes for these youths. Increased awareness of depression among this age group is the first step toward implementing effective intervention.

Saluja, Iachan, Scheidt, Overpeck, Sun, & Giedd, (2006) examined prevalence of and risk factors for depressive symptoms among young adolescents in a nationally representative, cross-sectional sample of young adolescents. 9863 students in grades 6, 8, and 10 (average ages, 11, 13, and 15) were the participants. The study revealed that eighteen percent of youth reported symptoms of depression. A higher proportion of females (25%) reported depressive symptoms than males (10%). Prevalence of depressive symptoms increased by age for both males and females. Among American Indian youths, 29% reported depressive symptoms, as
compared with 22% of Hispanic, 18% of white, 17% of Asian American, and 15% of African American youths. Youths who were frequently involved in bullying, either as perpetrators or as victims, were more than twice as likely to report depressive symptoms than those who were not involved in bullying. A significantly higher percentage of youths who reported using substances reported depressive symptoms as compared with other youths. Similarly, youths who reported experiencing somatic symptoms also reported significantly higher proportions of depressive symptoms than other youths.

A recent study by Nilsen et al. (2013) found that girls reported higher levels of social skills and depressive symptoms compared to boys, which is well in line with earlier findings (Fossum et al., 2007; Eberhart & Hammen, 2006; Eberhart et al., 2006; Rose & Rudolph, 2006; Zahn-Waxler et al., 2006). Social skills predicted social support from most sources, which also support former findings (Galambos et al., 2004; Rubin et al., 2004; Cauce, 1986).

**Depression and general aggression**

Prima facie, there appears to be little resemblance between depression and aggression (see for example, Zuravin, 1989). Depression connotes lethargy and sluggishness, making a person too listless to aggress. The Diagnostic and Statistical Manual of Mental Disorders DSM-IV-TR (2000) lists the following as criteria for a Major Depressive Episode: “feelings of sadness, diminished interest in activities, fatigue and diminished ability to think or concentrate, indecisiveness, recurrent thoughts of death” (DSM-IV-TR, 2000, p. 366). These seem counterindicative of aggression. Furthermore, the attributional biases of depressed and aggressive persons seem contradictory. Depressed people attribute negative events to an internal cause (i.e., blame themselves), a cause that is immutable and chronic (Joormann, 2009; Burns & Seligman, 1989). Burns and Seligman (1989) call this an “explanatory style” and show it to be stable across a lifespan and constituting an “enduring risk for depression” (p. 475). On the other hand, aggressive people externalize blame, perceive negative events as produced by traits in others, and see these others as having hostile intent (Dutton, 2006; Dodge, Pettit, Bates, & Valente, 1995; Dodge, 1993). The contradiction between an internal and external attribution for a negative event implies that depression and anger may be mutually exclusive consequences of different cognitive sequences. Despite these seemingly contradictory attributions, however, evidence exists to indicate that depression is linked to and is a risk factor for aggression. In part, the different views of depression and aggression may stem from a common view of all forms of depression as vegetative when
it is not, and of overlooking a common feature of depression, that of irritability (Kessler & Wang, 2009).

A number of studies have reported increased likelihood of aggression in samples reporting depression. Weiss and Catron (1994) examined 350 public school children (M ages=10.5) using a self-report measure of depression (The Children's Depression Inventory: Kovacs & Beck, 1977) and anxiety, and of self, peer and teacher reports of aggression. The authors found a significant relationship between depression and aggression (r=.35) which they construed as a “more broadband relation between externalizing and internalizing”. In fact, they found a significant positive correlation between externalizing (consisting of hyperactivity/attentional problems and aggression) and internalizing (depression and anxiety) of .46. Comparisons of confirmatory factor analysis models suggested that rather than a specific link between aggression and depression, persons with comorbid depression and aggression may have broadband associations of externalizing and internalizing problems of which aggression and depression are subsets. Both internalizing and externalizing contain other sub factors besides depression and aggression. For example both withdrawn and aggressive boys had fewer peer contacts and self and teacher reported anxiety and externalizing children had attentional problems. They cited other studies as indicating that aggression may cause peer rejection which in turn would cause internalizing problems. They also concluded that the broadband set of relationships found did not support the theory that depression directly causes aggression (because depression was not related to hyperactivity), but was more consistent with the theory that the consequences of aggression cause depression. They did not explore the role of anxiety as a common aspect of both depression and aggression, although in their factor analysis, anxiety had a significant correlation to both internalizing (.91) and externalizing (.41).

Taft et al. (2009) examined the relationship of depression using the Beck Depression Inventory (BDI: Beck, Steer, & Carbin, 1988) to both general aggression (i.e., aggression to another other than a relationship partner) and spousal aggression in a war veterans group. The authors found significant correlations of both PTSD and BDI scores to both forms of aggression. The BDI measures of depression were for the two-week period prior to assessment, while the aggression measures were for the year preceding the assessment. The authors acknowledged that the cross-sectional design used limited causal conclusions about the role of depression and PTSD on aggression.
Sher et al. (2005) assessed lifetime aggression with the Aggression History Scale (Brown & Goodwin, 1986), impulsivity with the Barrat Impulsivity Scale (Barratt, 1965), suicidality with the Columbia Suicide History Form (Oquendo, Halberstam, & Mann, 2003), and depression using the Structured Clinical Interview 11 (SCID-11). General lifetime aggression and lifetime impulsivity were significantly higher in the depressed+alcoholism group. Again, depression measures were for current depression while aggression and suicidality measures were for past incidences.

Community sample studies have yielded a positive correlation between depressive symptomatology and male physical aggression towards spouses. Bland and Orn (1986) presented data from a random digit dialled survey of urban dwellers. Using the Diagnostic Interview Schedule, they assessed for anti-sociality, major recurrent depression, alcohol dependence, and spouse assault. For respondents with no diagnoses, the baseline reported spouse assault rate was 15.5%. When depression was diagnosed, it increased to 54.5%. When both depression and alcoholism were diagnosed, the rate increased by 84%. Using “nonordered multinomial logistic models”, Pan, Neidig, & O'Leary (1994) assessed the possibilities of mild and severe husband to wife physical aggression in 11,870 white men. They found that depressive symptomatology increased the odds of mild (non-injurious—typically pushing, shoving or slapping) wife assault by 30% and severe (typically punching, kicking or beating up) wife assault by 74%.

In a college student sample, verbal and physical aggression towards an intimate partner (intimate partner violence: IPV) was found to be significantly related to moderate depression in perpetrators (Brooks, Harris, Thrall, & Woods, 2002; Patten, Choi, Vickers, & Pierce, 2001). Comorbid depression in veterans with PTSD was among the strongest risk factors for spouse assault perpetration (Taft et al., 2005). Taft et al. (2009) examined correlates of IPV and general aggression in male combat veterans suffering from PTSD and found a direct correlation between depression and aggression. In Cogan & Fennell's (2007) college sample study on sexuality and physical violence, those who perpetrated violence to partners had higher scores on depression than those who did not. Vivian & Langhinrichsen-Rohling (1994) assessed depression (BDI) and spouse assault aggression for both men and women in a couples' therapy clinic. Reported depression spouse assault perpetration was significantly correlated for both men and women in the sample.
Other studies have also noted the association of anger and depression. Anger outbursts were found to be positively correlated with depression (Painuly, Grover, Gupta, & Mattoo, 2011), suggesting that depression can also moderate affect which can bring about aggressive behaviour (more on this below). Additionally, the clinical literature has suggested that “males who are depressed display aggression and violence, but do not report depressed affect” (Roberts, Gold, Kim, & Hounchell, 2010, p. 373), proposing that expression of depressive symptoms of IPV perpetrators may be impeded in or masked by aggressive symptoms. Finally, borderline personality, which is also predictive of anger and IPV (Dutton, 2006), has a depressive component. Reich & Zanarini (2001) found that “sadness or emotional pain” and depression were the two most frequently reported childhood symptoms by adults diagnosed with Borderline PD.

**Depression and Isolation**

One of the negative consequences of depression is loss of social support due to: 1) social withdrawal, 2) perceived alienation, and 3) actual reduced support due to affective instability and pessimistic dispositions. Social support encompasses close, personal relationships with others, the degree of embeddedness or social integration in one’s community, and the degree of deep and abiding social resources (Berkman, 2000), and can be measured as “quantitative versus qualitative”, and “instrumental versus expressive” (Wallston, Alagna, DeVellis, & DeVellis, 1983, p. 369). Depressed individuals may generally receive less social support, either actual or perceived, than non-depressed individuals as a result of their negative attributions and mood, which, in turn, deprives them of the beneficial effects of social support on dealing with aggression issues. Depressed individuals exhibit symptoms, such as “negative self-statements, dependency, reassurance seeking, inappropriate or premature disclosures, passivity, [and] social withdrawal” that significantly reduce social support, particularly in the form of friend support (Stice, Rohde, Gau, & Ochner, 2011, p. 361). Both Pope, Bierman, & Mumma (1991) and Hymel, Rubin, Rowden, & LeMare (1990) found that aggression produced peer rejection and subsequent internalizing problems including depression. In a study investigating interpersonal consequences of depressed behavior by examining reactions to subjects who enacted either a depressed or nondepressed role, depressed subjects elicited more negative reactions and were rejected at a higher rate than nondepressed ones (Hammen & Peters, 1978). In particular, nondepressed subjects expressed a general lack of interest for further contact with
depressed subjects, especially if they perceived them as being psychologically impaired and unable to function in various roles, factors that further contribute to depression and social withdrawn, maintaining the vicious cycle of depression–isolation.

In a group of 258 adolescents, Auerbach, Bigda-Peyton, Eberhart, Webb, and Ho (2011) found that deficits in social support resulted in interpersonal stress and depressive symptoms. Similarly, when examining the relations between social contact, perceived social support, depression, and alcohol use, Peirce, Frone, Russell, Cooper, & Mudar (2000) found that social contact was negatively correlated to depression, emphasizing the benefits of social engagement. Social deficits resulting from depression can bring about social isolation or alienation, a sense of separation or estrangement from society, one's self, or significant others. Alienation, in turn, can minimize access to available community resources that may help reduce aggression such as support groups, conflict resolution, skill building and anger management workshops, and relationship counselling. Furthermore, the isolated/alienated individual is more likely to ruminate and rumination can be another link to aggression (Carpara, 1986).

**Depression and impulsivity**

Depression has negative implications for general self-regulation, influencing cognitions, behavior, and affect. Although not a DSM criterion for depression, impulsivity is frequently found in depressed samples and correlates positively with aggressive behavior (Perroud, Baud, Mouthon, Courtet, & Malafosse, 2011; Peluso et al., 2007; Semple, Zians, Strathdee, & Patterson, 2007). “Impulsivity is the neurophysiologically based inability to conform behavior to its context or consequences” (Peluso et al., 2007, p. 227). Impulsive individuals can be careless and spontaneous, making unplanned, rapid decisions, and acting recklessly (Magid, MacLean, & Colder, 2007). Their inhibitory functions to potential consequences or punishment are not properly activated (Newman, 1987), making them more likely to engage in illegal or sensation-seeking activities due to poor self-control. Generally, impulsive individuals are overly sensitive to rewards (Gray, 1987) demonstrating poor delayed gratification. Barratt and Patton (cf. Peluso et al., 2007) studied and measured impulsivity as a stable characteristic persisting across time and situations. Patton et al. (cf. Peluso et al., 2007) specified three subscales of impulsivity and their implications to cognition and behavior: “attentional (rapid shifts of attention and impatience with complexity), motor (impetuous action), and non-planning (lack of future orientation)” (p.
229), with the attentional and motor subscale being the primary risk factors in compromising interpersonal relationships. Attentional and motor subscales of impulsivity are representative of hostile aggression, a type of “affective, impulsive, or reactive aggression”, characterized by lack of planning and driven by anger, and where the intention is to harm the target (Anderson & Bushman, 2002, p. 29). Sher et al. (2005) study of clients with mood disorders showed lifetime impulsivity (measured by the Barratt Impulsivity Scale) and suicidality to be related. The aggressive/suicidal group had histories of both depression and alcoholism.

A large (n=188) sample of patients diagnosed with Major Depressive Disorder (MDD) and who had previous suicide attempts showed high impulsivity scores and severe lifetime aggression compared to MDD patients without prior suicide attempts, demonstrating the relationship between impulsive and aggressive (albeit self-aggression) traits (Perroud et al., 2011). Increased state and trait impulsivity levels have also been found in depressed bipolar, euthymic bipolar, and depressed unipolar patients contributing to poorer compliance to treatment (Peluso et al., 2007).

**Social Support and Depression**

Measures of both received support and perceived available support typically assess respondents' perceptions of several support functions that are provided through social relationships. In this regard, there are various types of support functions that may be differentially useful for different types of problems or stressors (Cutrona & Russell, 1990). Wills and Shinar (2000) described the most common support functions, including emotional support; instrumental support; informational support; companionship support; and feedback. Emotional support (also known as esteem support) refers to the availability of people who listen sympathetically and communicate care and acceptance when an individual is undergoing life difficulties. Instrumental support (also known as tangible support or practical support) refers to the provision of practical help when necessary (e.g., helping with transportation, household chores, and financial aid). Informational support (i.e., advice or guidance) refers to the provision of knowledge and information that may be useful for solving problems (e.g., providing information about community resources, assisting with decision making, or providing advice). Companionship support (socializing) refers to the availability of people with whom individuals can participate in social, leisure, and recreational activities (e.g., having a partner for sports, movies, and shopping). Finally, feedback refers to the provision of information about the appropriateness and normativeness of social behaviors.
At the broadest level, social support has been defined* as any process whereby social relationships promote health and well-being (Cohen, Gottlieb, & Underwood, 2000). The various types of social relationships that have been studied include romantic partners, friends, and family members. Deficits in social support have been found to affect a variety of mental health constructs, including depression (Lakey & Cronin, 2008).

Considerable research has shown that depression is associated with difficulties in interpersonal relationships. Individuals with major depressive disorder are significantly more distressed by interpersonal problems than normative samples (Barrett & Barber, 2007). Depressed individuals also enjoy their social interactions less, and find them to be less intimate, when compared with non-depressed individuals (Nezlek, Hampton, & Shean, 2000; Nezlek, Imbrie, & Shean, 1994). Among the various interpersonal difficulties experienced by depressed individuals, deficits in social support represent a major deterrent to well-being. As one illustration, just over half of a large sample of adults with major depression reported that social support issues complicated their depression, or made it difficult to treat (Gladstone, Parker, Malhi, & Wilhelm, 2007). This study also found that depressed patients' perceptions of poor support from friends, as well as their perception that poor support impeded recovery, were shown to be related to a longer duration of their current depressive episode. Taken together, these types of findings provide persuasive evidence for the critical role of social support in depression.

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*Social support has been defined as relationships that can provide material and interpersonal resources (Thompson, 1995). However, several types of relations, formed within different groups, might be important for depressive symptoms in different ways during development. Parents are one of the main sources of social support during childhood, providing in most cases both material and emotional support, while relationships with, and social support from, friends become increasingly important in the transition from childhood to adolescence (Rubin et al., 2006).
Considerable research, thus, points to strong and consistent relationships between social connectedness and depression. For example, in a series of studies, Cacioppo et al. (Cacioppo, Hawkley, & Thisted, 2010; Cacioppo, Fowler, & Christakis, 2009; Cacioppo, Hughes, Waite, Hawkley, & Thisted, 2006) have demonstrated that perceived social isolation is a strong longitudinal predictor of depressive symptomatology within general community samples, even when controlling for an array of demographic and social factors that might account for such a link.

Furthermore, low social support predicts poor response to depression treatment and early drop-out (Trivedi, Morris, Pan, Grannemann, & Rush, 2005) and low social functioning increases the risk of relapse (Backs-Dermott, Dobson, & Jones, 2010; George, Blazer, Hughes, & Fowler, 1989; Paykel, Emms, Fletcher, & Rassaby, 1980). There is also evidence that interventions aimed at increasing social interaction can reduce depression symptoms (Cattan, White, Bond, & Learmouth, 2005; Perese & Wolf, 2005). The association between social isolation and depression has proved to be robust, despite considerable variation in the measures that researchers use (e.g., assessing a person's number of friends, intensity of social activity, perceived loneliness; Kikuchi & Coleman, 2012; Harpham, Grant, & Thomas, 2002).

It is now generally accepted that deficiencies in social support increase the risk for depression. However, processes or mechanisms that may be involved are still not well understood. In particular, the matching hypothesis states that social support is effective in promoting coping and reducing the negative effects of stressful life events, insofar as the type of support matches the demands of the stressors. As one illustration, having a friend lend you money may be more beneficial when faced with a job loss than when dealing with the loss of a relative. Similarly, receiving emotional support from a friend may be more beneficial when dealing with the loss of a relative than when faced with a job loss.

When applied to depression, the buffering hypothesis posits that social support reduces the association between negative life events and the severity of depression levels, or the risk for development of depression. Past research has suggested that social support may buffer the negative effects of stress, and thus protect against depression, by enhancing individuals’ self-esteem, self-confidence, and self-efficacy (Nezlek, Kowalski, Leary, Blevins, & Holgate, 1997).
In general, evidence for the stress-buffering effects of social support on depression is mixed. Some studies have found support for the buffering hypothesis, typically in the form of a significant interaction. Here, the positive impact of greater social support on reducing depression levels is most evident when the number of negative life events is high (Nezlek & Allen, 2006; Burton, Stice, & Seeley, 2004). Other studies, however, have provided contrary evidence, with no interaction effects being evident (Nezlek & Allen, 2006; Choenarom, Williams, & Hagerty, 2005; Wade & Kendler, 2000). In turn, this mixed pattern of findings suggests that there could also be more pervasive and direct positive effects of social relationships on the well-being of depressed individuals; and that these direct effects are not necessarily limited to the occurrence of negative life events.

In addition to being associated with depressive symptoms, findings indicate that social support is linked to social skills (Galambos et al., 2004; Rubin et al., 2004; Cauce, 1986). It is suggested that social skills protect people from developing internalizing problems because of more positive interpersonal interactions (Letcher et al., 2009; Lewinsohn, 1974). Better quality of interpersonal interactions is associated with social support, which in turn is negatively associated with depressive symptoms (Rubin et al., 2006, 2004). Still, few studies have examined the interplay between these three factors. Two recent studies yield support for this model. Segrin and Rynes (2009) reported that young adults’ positive relations with others significantly mediated the prospective relationship between social skills and depressive symptoms. Also, Lee et al. (2010) reported that the prospective association between social skills and depressive symptoms among 11–17 year-olds was mediated by conflicting relationships to parents, but not by conflicting relationships with friends, suggesting that different sources of support play a different role. Neither of these studies examined support from friends, parents, and teachers nor gender-specific mechanisms.

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/attempt), 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, which did not find a significant relationship between developmental level and depressive symptoms, may be because of the fact that the study had participants with a larger age range.

Ginicola (2007) also examined children’s unique experience of depression, using a developmental approach to predict variation in symptomatology. Participants were 252 psychiatric inpatients aged 4 to 16 with a diagnosed depressive disorder. Children were only included in the study if they had been given an IQ test and were diagnosed with a depressive disorder; there were no other exclusion criteria. The author argued that although inpatient children are not representative of all children due to the severity of their symptoms and subsequent functioning difficulties, they were utilized because there were fully experiencing severe depression. Additionally, a large quantity of detailed records (including symptom notes from staff and clinicians, parent or guardian reports, observable child behavior and survey scores) can be used within this population. Given these reasons, using an inpatient sample provides a good degree of power to identify the relationship between developmental level and patterning of depressive symptoms.
All children were diagnosed by trained clinicians using DSM criteria. Patients were predominantly male (61%) with varied ethnic backgrounds (Caucasian 54%; African American 22%, Hispanic 19%; Other 5%). Participants’ ages ranged from 4 to 16 years old, with an average of 10.23 (S.D. = 3.07) years. The majority of participants were diagnosed with Depressive Disorder NOS (38.1%) or Mood Disorder with psychotic features (12.7%), Major Depressive Disorder (11.5%), Major Depressive Episode (3.2%) and Dysthemia (2.8%). The majority of participants (66%) were comorbid for an anxiety or conduct disorder (25% for an anxiety disorder, 29% for a conduct disorder, and 12% for both an anxiety disorder and a conduct disorder). A few symptoms, such as feelings of guilt (8%) and diminished ability to concentrate (16%) were not very prevalent within this sample. The average IQ score of this sample was 92.65 (S.D. = 16.5) and was normally distributed. It is worthy to note that only 5% of the sample was diagnosed with mental retardation, indicating that the vast majority of the sample was cognitively normative. All diagnoses were made by experienced clinicians using DSM-IV TR criteria. Children were given an IQ test (KBIT or WISC) while within the hospital. Mental age was calculated by using the child’s IQ score and chronological age. Four trained raters reviewed children’s records for depressive symptoms as defined by the DSM-IV TR. Additionally, a ratio score was calculated to indicate the number of internalizing symptoms to total symptoms.

The first hypothesis, that mental age would be positively correlated with internalizing symptoms was tested with a more stringent alpha level of <.01 in order to account for inflated error. The results indicated that mental age was strong correlate of internalizing depressive symptoms, r = .51, p < .0001. It is interesting to note that the correlation was slightly larger than that between chronological age and the internalizing ratio score, r = .48, p = .0001, and much larger than that between IQ and the ratio score, r = .249, p = .0001. The hypothesis that mental age would be associated with symptom presentation of depression was supported. Mental age served as a correlate of depressive symptoms and the relationship can be seen across developmental time periods. Mental age was found to be a much better predictor than IQ; however, in this predominantly developmentally-normative sample, CA was also a good predictor, indicating that CA has value as a predictor of symptoms as well. These results are supported by previous research and theory on depression across different ages and IQs, as well as normative developmental theory (Bowen & John, 2001). The findings from this study differ from the results of the Kovacs and Paulauskas (1984) study, which 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.

The hypothesis that mental age would correspond to the type of comorbid diagnoses was also supported. Comorbidity was related almost linearly with mental age and presentation (internalized only or mixed presentation), which supports the numerous studies reporting the emergence of conduct disorders before anxiety disorders (Weiss & Catron, 1994). This relationship could be explained in that anxiety disorders may be less prevalent in very young children because certain cognitive structures need to have developed to express anxiety in a traditional manner. Conduct Disorders, on the contrary, may be completely action-based in nature and may not require the development of certain cognitive functions. Another reason for this finding could be that depression and these other disorders are not comorbid at all; rather they are extended symptoms of depression.

Although there was no main effect for ethnicity, there was a significant interaction between ethnicity and mental age. At a low developmental level, there was no significant difference between minorities and Caucasians on the internalizing ratio score – they were both quite low. However, in the high mental age group, minorities were significantly lower on the internalizing ratio score than their Caucasian counterparts. This indicates that the relationship between developmental level and the internalizing ration score is buffered by a cultural variable. Familial and cultural socialization has been shown to promote aggression and eschew suicide in minority populations (Guerra & Williams, 2006). Perhaps then, this socialization, although individually not stronger than mental age, dampens the relationship between developmental level and symptoms in higher mental age periods, such as adolescence.

The findings presented within this study indicate that a developmental approach is useful in understanding children’s depressive symptoms. Within the context of gender and culture, children’s symptom presentation was significantly related to their age. This indicates that as a child develops, their experience of depression changes in important ways. These differences can complicate both the diagnosis and treatment of depression in children. By increasing the knowledge of how depressive symptoms change across the course of childhood, earlier diagnoses of depression in children can be made and the best treatment options can be selected. The author emphasized that subsequent research should focus on replication of this study, using both M.A. and a broader measure of developmental level, either in an outpatient or community sample.
OVERVIEW

1. 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.

2. 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.

3. Depression is a heterogeneous disorder, both in its manifestation and its etiology. In spite of this heterogeneity, research on depressive disorder is most often monodisciplinary. Research that crosses boundaries between disciplines is rare, which precludes an integrated understanding of depression (Costello et al., 2002).

4. Adolescence is a period when depressive symptoms start to occur with increased frequencies (Yang, Soong, & Kuo, 2004; Oldehinkel, Wittehen, & Shuster, 1999) and depressive symptoms emerging in this period usually persist into later life (Harrington, Fudge, & Rutter et al., 1990) and more likely stay on the course (Ge, Conger, & Lorenz et al., 1995).

5. According to the 2001 World Health Organization (WHO) report, depression is the most common disease suffered by women when compared with other diseases. In the WHO’s Global Burden of Disease Indexes, the point prevalence of unipolar depressive episodes is 1.9% for men and 3.2% for women; 5% of men and 9.5% of women experience a depressive episode in a 12-month period. Starting in adolescence, girls are two to three times more likely than boys to report depression – a gender difference that persists throughout the life span (Zahn-Waxler, Crick, Shirtcliff, & Woods, 2006; Hankin et al., 1998). Several studies suggest that this preponderance of depression in girls might be due to interpersonal vulnerability (Cyranowski, Frank, Young, & Shear, 2000; Petersen et al., 1993). Both lack of social skills and social support are reported to be risk factors for future depressive symptoms (Segrin & Rynes, 2009; Thompson, Flood, & Goodvin, 2006; Segrin, 2000), and several findings indicate that there are gender-specific mechanisms behind these relations (Eberhart, Shih, Hammen, & Brennan, 2006; Rose & Rudolph, 2006).

6. Several recent reviews have articulated the general cognitive vulnerability–stress model of depression (Abramson & Alloy, 2006; Abramson et al., 2001; Hankin & Abramson,
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.

7. Depressed individuals may generally receive less social support, either actual or perceived, than non-depressed individuals as a result of their negative attributions and mood, which, in turn, deprives them of the beneficial effects of social support on dealing with aggression issues.

8. 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, may be because of the fact that the study had participants with a larger age range.

CONCLUSION

A careful review of the overview reveals that with respect to the examination of pathogenesis of depression among adolescents, numerous variables viz. Cognition, personality, affect, self-esteem and aggression have gained the lion’s share of research attention with a consequent neglect of the developmental approach to predict variation in depression symptomatology. There is paucity of research to date which has directly tested the hypothesis of a relation between developmental level and depressive symptom patterns in the non-clinical population. In this context, two studies by Kovacs & Paulaskas (1984) and Ginicola (2007) are noteworthy. Participants in these studies were psychiatric inpatients aged 4 to 16 with a diagnosed depressive disorder. All children were diagnosed by trained clinicians using DSM criteria. Four trained raters reviewed children’s records for depressive symptoms as defined by the DSM-IV TR (A.P.A., 2000). Although the results of
these studies indicate that a developmental approach is useful in understanding children’s depressive symptoms, the results are limited to psychiatric inpatients and cannot be generalised to non-clinical population. Even in non-clinical population, depression can be found in a wide range of individuals, from infants to the elderly.

In the context of the above observation, the current study expanded on the existing limited literature by examining adolescents’ unique experience of depression, using a developmental approach to predict variation in symptomatology. The purpose of the current study was to refine and replicate the association between developmental level and depressive symptoms in non-clinical sample of male and female adolescents.

HYPOTHESIS

The following hypotheses were formed on the basis of above mentioned review of literature:

1. It is expected that internalizing symptoms would be more prevalent at high developmental level.

2. It is expected that externalizing symptoms would be more prevalent at low developmental levels and gradually change to internalizing behavior over time

3. Male and female adolescents differ on the relevance of internalizing as well as externalizing symptoms in relation to developmental level.