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A Longitudinal Study of Risk Factors for Adolescent Depression: Gender Differences and Pathways of Risk

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A Longitudinal Study of Risk Factors for Adolescent Depression: Gender Differences and Pathways of Risk

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A Longitudinal Study of Risk Factors for Adolescent Depression: Gender Differences and Pathways of Risk

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Abstract: Despite consistent evidence that adolescent girls are at greater risk for developing depression than adolescent boys (Ge, Lorenz, Conger, & Elder, 1994; Nolen-Hoeksema, 1987, 1990; Weissman, Leaf, Holzer, Myers, & Tischler, 1984), and that women continue to predominate among depressed adults throughout the lifespan (Kessler, McGonagle, Swartz, Blazer & Nelson, 1993), few studies have examined the etiologic risk factors that predict depression for adolescent girls using a prospective design or examined differential processes of risk in a sample of adolescent girls and boys. Furthermore, although a number of variables have been implicated as risk factors for depressive symptoms or onset of depression among adolescents, some methodological limitations exist. The objective of this research was to examine a set of risk factors suspected to predict depression in adolescents, and to test whether gender moderates these relations.

Secondly, this study examined a set of risk factors proposed by the gender additive model of depression (Stice et al., 2000; Stice & Bearman, 2001) that attempts to partially explain the increased prevalence of depressive symptoms in adolescent girls compared to boys. Finally, exploratory classification tree analyses tested for interactions between risk factors that might signal differential pathways to depression. This research provides insight into the etiology of adolescent depression as well as the disparate rate of depression among adolescent girls versus boys, and also provides direction for identifying high-risk individuals and developing effective prevention programs.

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CHAPTER 1: INTRODUCTION

1.1 Epidemiology of Adolescent Depression

Major depression is one of the most common psychiatric problems confronted by adolescents, with lifetime prevalence rates estimated to range from 15 to 20% (Birmaher et al., 1996). Adolescent depression is characterized by a recurrent course and elevated psychiatric comorbidity, including anxiety disorders, disruptive behavior disorders, eating disorders and substance abuse (Angold & Costello, 1993; Fergusson & Woodward, 2002; Lewinsohn, Rohde, Klein, & Seeley, 1999; Newman et al., 1996; Pine, Cohen, Gurley, Brook & Ma, 1998). Depressed adolescents face increased risk for a host of adverse outcomes, including future suicide attempts, academic failure, marital difficulties, interpersonal problems, unemployment, substance abuse, and delinquency (Fergusson & Woodward, 2002; Gotlib, Lewinsohn, & Seeley, 1998; Birmaher et al., 1996). Even subdiagnostic depression during adolescence persists over time and predicts onset of psychiatric disorders, inpatient hospitalization, substance abuse, academic problems, impaired social functioning, and suicidal ideation (Capaldi & Stoolmiller, 1999; Gotlib et al., 1998; Nolen-Hoeksema, Girgus, & Seligman, 1992). Moreover, depression in adolescence may continue to impair functioning even after recovery (Kovacs & Golston, 1991; Puig-Antich et al., 1993), and is strong predictor of adult depression (Ge, Conger, & Elder, 2001; Lewinsohn, Rohde, Klein, & Seeley, 1999; Pine, Cohen, Cohen & Brook, 1999).

Adolescence appears to represent a period of considerable vulnerability for depression, with prevalence rates increasing from less than 3% in prepubertal children (aged 6-11) to 9% in children aged 12 to 16 years old (Fleming & Offord, 1990; Garrison, Addy, Jackson, McKeown, & Waller, 1992; Kaltiala-Heino, Rimpela, Rantanen, Laippala, 2001; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993), and rising dramatically from age 15 to 18 with regards to both onset of depression (Hankin & Abramson, 2001) as well as increases in depressive symptoms (Ge, Conger & Elder, 2001; Ge, Lorenz, Conger, Elder, & Simons, 1998). Although the precipitous rise of depression during adolescence and its deleterious impact have been well established, the factors that increase the risk for this disorder are less well understood.

While rates of depression increase from childhood to adolescence for all, a consistent finding is that adolescent girls are between 1.5 to 3 times more likely to develop depression than adolescent boys (Ge, Lorenz, Conger, & Elder, 1994; Nolen-Hoeksema, 1987, 1990; Weissman, Leaf, Holzer, Myers, & Tischler, 1984). This female preponderance is not seen in childhood; rather, some researchers have noted a slightly higher prevalence of depression among boys prior to adolescence (Brooks-Gunn & Petersen, 1991; Ge et. al., 1994; Rutter, 1986). This striking shift from a relatively balanced dispersion of depression among girls and boys towards a significant imbalance of depression in adolescent girls seems to occur between ages 13-16 (Hankin & Abramson, 2001; Hankin,

Abramson, Moffett, & Silva, 1998; Ge et. al., 1994). Once established, this phenomenon persists throughout most of adulthood (Kessler et al., 1993).

Given that the risk for depression increases for adolescents, and adolescent girls are at twice the risk for depression than adolescent boys, it is critical to examine both the variables that predict depression for this population as well as the variables that predict depression differentially for boys and girls. Furthermore, since the bulk of previous studies have examined risk factors in isolation, a study that examined possible interactions of theoretical risk factors for depression would also contribute to our understanding of the etiology of adolescent depression. Identifying variables that reliably predict depression is an essential step towards successfully intervening to prevent depression—an important goal given that fewer than 25% of depressed adolescents will ever receive treatment for this impairing disorder (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Newman et al., 1996). Risk factor research is helpful for prevention efforts because it identifies high-risk individuals who might benefit from intervention as well as informing the content of intervention efforts.

1.2 Risk Factors for Adolescent Depression

Although a number of risk factors have been identified as contributing to both the onset of major depression and increases in depressive symptomology, only a handful have been found to consistently predict depression above and beyond initial depression in prospective samples of adolescent boys and girls.

Indeed, the majority of studies that have investigated relations between putative risk factors and depression have relied on cross-sectional data (see Birmaher et al., 1996; Gladstone & Kaslow, 1995; Sheeber, Hops & Davis, 2001, for reviews). Unfortunately, cross-sectional data do not permit determination of whether potential risk factors are precursors, concomitants, or consequences of depression. Some studies that used prospective designs did not control for initial levels of depressive symptoms or initial diagnostic status (e.g., Duggal, Carlson, Sroufe, & Egeland, 2001; McFarlane, Bellissimo, & Norman, 1995; Spence, Najman, Bor, O'Callaghan, & Williams, 2002), without which we cannot rule out the possibility that changes in depression are simply the by-product of the correlation between the risk factor and depression at baseline for these studies.

Among the risk factors consistently found to prospectively predict depression above and beyond baseline depression in longitudinal examinations are stressful life events (Lewinsohn et al., 1994; Nolen-Hoeksema, Girgus, & Seligman, 1992; Windle, 1992), negative affectivity (Gjone & Stevenson, 1997; Leadbeater, Kuperminc, Blatt, & Hertzog, 1999), deficits in social support (Bennett & Bates, 1995; Lewinsohn et al., 1994; Windle, 1992), and externalizing behaviors (Fergussan, Wanner, Vitaro, Horwood, & Swain-Campbell, 2003; Lewinsohn et al., 1994). Other risk factors have received mixed support, such as attributional style (Lewinsohn et al., 1994; Bennett & Bates, 1995; Cole, Martin, Peeke, Seroczynski, & Hoffman, 1998), or have used inconsistent methodology, such as ethnic status. Finally, ruminative coping has yielded promising results in

two prospective studies of adolescent depressive symptoms (Abela, Brozina, & Haigh, 2002; Schwartz & Koenig, 1996); further evidence of this relation would more firmly establish this risk factor as a predictor of adolescent depression. The theoretical relation of these variables and depression in adolescents, as well as available evidence, will be discussed in detail below. Because previous literature has used the term *depression* in inconsistent ways, we will differentiate between those studies that measured depressive symptoms versus those that measured onset of clinically impairing depression diagnoses.

1.2.1 Stressful Life Events

One of the most widely accepted risk factors for onset of depression and increase in symptoms is the occurrence of one or more stressful life events (Birmaher et al., 1996). Theoretically, the occurrence of a stressful life event contributes to initial levels of negative affect and maladaptive cognitions in response to the stressor (Bennett & Bates, 1995; Hankin & Abramson, 2001). The negative mood and cognitive style, if they continue, are posited to lead to depressive symptoms such as difficulty sleeping or poor concentration. It has been suggested that stressful life events increase during adolescence because of the many important social and biological transitions that occur during this time (Ge, et al., 2001), and there is evidence that young adolescents experience more life events, particularly negative events, than their younger counterparts (Larson & Ham, 1993).

There is empirical evidence to support the relation between stressful events and depression in adolescents. Stressful life events have emerged as a robust predictor of adolescent depressive symptoms in numerous longitudinal studies (Bennett & Bates, 1995; Ge et al., 2001; Hilsman & Garber, 1995; Leadbetter, Kuperminc, Blatt, & Hertzog, 1999; Nolen-Hoeksema et al., 1992; Windle, 1992), and stressful life events also predict future onset of major depression in adolescent samples (Cohen, McGowen, Fooskas, & Rose, 1984; Goodyer, Herbert, Tamplin, & Altham, 2000; Lewinsohn et al., 1994; McFarlane, Bellissimo, Norman, & Lange, 1994). Furthermore, studies that examined changes in negative affect following stressors have found that negative affect rises initially for most people after a negative event occurs (Metalsky, Halberstadt, & Abramson, 1987; Nolen-Hoeksema & Morrow, 1991), and that negative affect predicted subsequent increases in depressive symptoms among late adolescents (Elliott, Marmarosh, & Pickelman, 1994).

The role of stressful life events in the gender difference in adolescent depression has received mixed support. Although some researchers have found that girls experience more stressful life events than boys during adolescence (Ge et al., 1994; Tubman & Windle, 1995), tests of gender moderation have yielded conflicting findings. Hankin, Abramson, & Siler (2001) found that gender moderated the relation of the interaction of stress and cognitive vulnerability to depressive symptoms, resulting in greater vulnerability for girls, and Rudolph (2002) found a significant interaction of interpersonal stress and gender on

depressive symptoms, causing greater increases for girls. Others reported that despite gender differences in levels of stress exposure, the relation of stressful events to depression was not moderated by gender (McFarlane, Bellissimo, Norman, & Lange, 1994; Spence, 2002).

1.2.2 Negative Affectivity

Some researchers have suggested that a propensity to experience negative affective states, such as anxious, angry, and depressive affects, may increase an individual's risk of developing depressive symptoms (Clark, Watson, & Mineka, 1994; Hankin & Abramson, 2001). Theoretically, individuals who are more negatively emotionally reactive are more likely to experience negative mood under general conditions, and this negative mood becomes more pronounced in stressful situations (Clark & Watson, 1991). These individuals are posited to experience more distress in the face of naturally occurring stressors, and to perceive events as more stressful than individuals who are less reactive. Although it is no longer believed that adolescence is universally a period of emotional turmoil for all, adolescence is associated with increases in negative affectivity (Larson & Richards, 1989) and mood lability (Larson, Czikszentmihali, & Graf, 1980), which may be partially rooted in biological processes (e.g., hormones). In support of the theoretical relation of negative affect and depression, negative affectivity has been found to predict adolescent depressive symptoms in

longitudinal studies (Elliott et al., 1994; Gjone & Stevenson, 1997; Leadbeater et al., 1999).

With regards to the gender differences in depression, Rudolph (2002) suggested that adolescent girls are more emotionally reactive to stress than adolescent boys, particularly when the stressors are of an interpersonal nature. In support of this claim, adolescent girls have been found to perceive negative interpersonal events as more stressful than boys (Wagner & Compas, 1990). Girls have also been found to experience higher levels of affective distress in response to stress in some studies (Ge et al, 1994), but other studies have not revealed gender differences in emotional reactivity to stress (Larson & Ham, 1993; Wagner & Compas, 1990). One longitudinal study that tested a mediational model found that girls' greater negative affect in the face of interpersonal stress partially accounted for gender differences in adolescent depressive symptoms (Rudolph, 2002). Conversely, a study that tested for gender moderation of the relation of negative affectivity to depressed behavior did not support this theoretical relation, and found that negative affect predicted this outcome for girls and boys alike (Gjone & Stevenson, 1999).

1.2.3 Social Support

Deficits in social support are another variable suggested to place adolescents at risk for depression. In theory, lack of support from family and friends may foster depression because the perception that one is not accepted or valued in

one's interpersonal environment may lead to decreases in self-esteem, confidence, and efficacy (Cohen & Wills, 1985). Whereas the belief that one is unconditionally supported by family and friends may mitigate the impact of daily hassles and stressful events, insufficient support may potentiate the influence of such incidents. During adolescence, it has been suggested that the parent-child relationship faces challenges associated with adolescent individuation (Windle, 1992). At the same time, peer influence becomes increasingly salient during adolescence (Inderbitzen, 1994; Kandel & Lesser, 1972). Thus, deficits in both peer and familial social support may confer particular risk for adolescents since it is during this time that these supports are both most precarious and critical.

A multitude of prospective studies have found that deficits in social support predicted future increases in depressive symptoms for adolescent boys and girls (Aseltine, Gore, & Colten, 1994; Bennett & Bates, 1995; Galambos, Leadbetter, & Barker, 2004; Kaltiala-Heino, Rimpelo, Rantanen & Laippala, 2001; Sheeber et al., 1997, Slavin & Rainer, 1990), as well as future onset of depression (Lewinsohn et al., 1994; McFarlane, Bellissimo, & Norman, 1995), although the theoretical assertion that support mitigates the impact of stressful life events has not been reliably supported (Burton, Stice & Seeley, in press). Interestingly, the studies that divided social support into separate constructs for familial support and friend support found that deficits in family support predicted future increases in depressive symptoms and major depression onset while deficits in support from

friends predicted neither (Lewinsohn et al., 1994; Stice, Ragan, & Randall, 2004; Windle, 1992).

The relation of social support to the gender difference in depression remains unclear. It has been proposed that girls are more sensitive to insufficient social support because they have stronger interpersonal affilliative needs than boys (Cryanowski, Frank, Young, & Shear, 2000). One study found that low familial support significantly predicted depressive symptoms for girls only, while friend support appeared to be an important protective factor for boys experiencing high levels of stress (Windle, 1992). Two studies found that social support deficits from both friends and family were related to increases in depressive symptoms for girls but not boys (Slavin & Rainer, 1990; Kaltiala-Heino, Rimpelo, Rantanen & Laippala, 2001); however, these studies examined boys and girls separately and did not appropriately test for gender moderation (Baron & Kenny, 1986). The studies we located that tested for gender moderation found no significant interaction for the effect of gender and inadequate social support variables, although social support deficits did predict depressive symptoms for both adolescent boys and girls (McFarlane, Bellissimo, Norman, & Lange, 1994; Pelkonen, Marttunen, & Aro, 2003).

1.2.4 Externalizing Behaviors

Theoretically, a causal relation between externalizing behaviors and depression stems from the adverse consequences that result from risk-taking

behaviors such as criminal acts, substance abuse, and sexual risk-taking (Fergussan et al., 2003). These adverse consequences may act as stressful life events that precipitate the onset of depression or increases in depressive symptoms. Furthermore, affiliation with deviant peers and delinquent behaviors may also result in alienation from family and friends, problems at school, and feelings of rejection, which have also been proposed to increase the risk for depression in adolescence (Fergussan & Woodward, 2002; Jaffee et al., 2002). Moffit (1993) has suggested that a relatively small group of boys and girls show persistent delinquent and antisocial behaviors throughout childhood, adolescence, and adulthood, but that a larger group of both genders will demonstrate such behaviors during adolescence only. As a result of this adolescence-limited trajectory, externalizing behaviors increase during early adolescence and then decrease substantially for most individuals in later adolescence (Bongers, Koot, van de Ende & Verhulst, 2003; Loeber, Stouthamer-Loeber, Van Kammen, & Farrington, 1989; Measelle, Stice, & Hogansen, in press; Moffitt, 1993), leading to consequent increases in depression during the adolescent period. In support of this model, adolescent externalizing behaviors predicted onset of depression (Fergussan et al., 2003; Lewinsohn et al., 1994) and future depressive symptoms (Capaldi, 1992; Pelkonen, Marttunen, & Aro, 2003). Another study found that association with deviant peers predicted depressive symptoms above and beyond initial symptoms for adolescent boys and girls (Fergusson et al., 2003).

We could only locate two studies that examined gender differences in the relation of adolescent externalizing behaviors and depression. Much of the research on externalizing behaviors has been conducted with boys, and there is evidence that boys with conduct problems in early adolescence were depressed in early adulthood (Capaldi & Stoolmiller, 1999). Although externalizing behaviors predicted depressive symptoms for boys and girls, no significant interactions with gender were found in either study (Pelkonen, Marttunen, & Aro, 2003; Weisner, 2003). A study by Leadbeater et al. (1999) found that externalizing symptoms did not predict internalizing symptoms for either gender.

1.2.5 Attributional Style

Negative attributional or explanatory style is central to a number of dominant etiologic models of depression. Beck (1976) postulates that a negative conception of oneself, one's experiences, and the future may promote a negative schema that biases the selection, encoding, and evaluation of information, producing a preference for information that confirms negative beliefs. The reformulated learned helplessness model posits that some individuals develop a tendency to attribute negative events to internal, stable, and global causes; and positive events to external, unstable, and specific causes (Abramson, Seligman, & Teasdale, 1978). This tendency leads them to blame themselves for negative events, and to view the causes of these events as unchangeable and pervasive. Conversely, these individuals do not attribute credit to themselves for positive

events. Adolescence is believed to be marked by heightened egocentrism and self-consciousness; this increased self-focus may make internal, stable, and global negative attributions more likely (Garber, Weiss, & Shanley, 1993).

Although the association between negative attributions and depression is widely accepted, this may be based on the high correlation observed between depression and negative attributions. A meta-analysis of this association was only able to locate four studies (of the 28 reviewed) that examined this relation prospectively in non-clinical populations (Gladstone & Kaslow, 1995). Among studies that have examined this relation prospectively to date, the support is inconsistent. Although attributional style predicted depressive symptoms (Hankin, Abramson, & Siler, 2001; Hilsman & Garber, 1995; Nolen-Hoeksema, Girgus, & Seligman, 1986; Robinson, Garber, & Hilsman, 1995) and onset of depression (Lewinsohn et al., 1994) in several prospective studies, some studies that examined the relation of attributional style to depressive symptoms found null effects (Bennett & Bates, 1995; Windle, 1992).

It has been proposed that girls are more likely to adopt a negative attributional style than boys (Hankin & Abramson, 1998), but this has not been borne out in studies that have examined gender differences in attributional style (see Gladstone, Kaslow, Seeley & Lewinsohn, 1997, for review). Whereas two studies that tested for gender differences in attributional style found null effects (Hankin et al., 2001; Lewinsohn et al., 1997), one study found that 10th-grade girls exhibited a more negative attributional style than boys (Nolen-Hoeksema &

Girgus, 1995). One cross-sectional study noted that girls' negative attributional style mediated the gender difference in depressive symptoms (Gladstone et al., 1997), while others have indicated that the relation between attributional style and depression is stronger for boys (Hops, Lewinsohn, Andrews and Roberts, 1990; Nolen-Hoeksema, Girgus, and Seligman, 1992). A prospective study that examined the relation of the interaction of stressors and attributional style to depressive symptoms did find that this relation was moderated by gender, and that girls were more vulnerable to the interaction of life stress and negative attributional style (Hankin et al., 2001). Another study found that negative attributions did not mediate the relation of gender to depressive symptoms for adolescents (Cole et al., 1999). In sum, there is conflicting evidence regarding whether girls are more likely to make negative attributions compared to boys, and whether the relation of negative attributions to depression differs for boys versus girls.

1.2.6 Ruminative Coping

Nolen-Hoeksema (1991) defined rumination as a stable, emotion-centered coping style that involves a passive, inward focus toward feelings of distress and the causes and consequences of these feelings. This focus on negative feelings is believed to foster depression because increasing attention to negative events or aspects of oneself enhances their accessibility and recall. In contrast, individuals who do not ruminate may be more likely to be distracted from their negative

emotional experience (Broderick, 1998). Because of cognitive developmental changes occurring during adolescence, the capacity for introspective self-reflection is heightened during this time (Keating, 1980), which is consistent with the self-focus rumination entails.

Although there is evidence that coping style predicts depression onset for both girls and boys (Lewinsohn et al., 1994), we could only locate two studies that examined ruminative coping as a predictor for depression in adolescents. In a study of prepubertal children and young adolescents, ruminative coping predicted increases in depressive symptoms over a six-week period (Abela, Brozina, & Haigh, 2002). A second study found that rumination predicted future depressive symptoms over six weeks in a sample of adolescents aged 14 to 18 (Schwartz & Koenig, 1996). Among college students, rumination has been found to predict depression (Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema et al., 1993).

Theoretically, girls are more likely to ruminate in response to negative events as a result of their subordinate social status and the subsequent belief that they must be wary of taking action to address the sources of their distress (Nolen-Hoeksema & Jackson, 2001). In a study of college students, women were more likely to choose an emotion-focused coping response than men, and that an emotion-focused coping response predicted depressed mood (Butler & Nolen-Hoeksema, 1994). Nolen-Hoeksema et al. (1993) also demonstrated that rumination mediated the relation of gender to depression, such that when rumination was controlled, gender no longer predicted the duration of depressed

mood. Although this variable has received less empirical examination among adolescent samples, two studies found that girls were more likely to endorse rumination as a coping mechanism in the face of stressors (Broderick, 1998; Schwartz & Koenig, 1996). Unfortunately, the first study was cross-sectional, and did not examine the proposed relation between rumination and depression. Although Schwartz & Koenig (1996) did examine gender differences in the predictive relation of rumination to depressive symptoms, they did not appropriately test for an interaction of gender and rumination in the complete sample, but instead looked at the relation of rumination to future depressive symptoms separately for girls and boys.

1.2.7 Ethnic Status

Cross-sectional investigations have found that minority adolescents report more depressive symptoms than Caucasian adolescents (Emslie, Weinberg, Rush, Adams, & Rintelmann, 1990; Roberts, Roberts, & Chen, 1997; Roberts & Sobhan, 1992; Siegal, Aneshensel, Taub, Cantwell, & Driscoll, 1998). However, other studies have not found ethnic differences in depression (Garrison, Addy, Jackson, McKeown, & Waller, 1992; Kandel & Davies, 1982). Two competing theoretical explanations have been put forth to explicate the link between ethnic status and depression. The first suggests that ethnic differences in depression are due primarily to social class effects, since positions of disadvantage as a result of economic status are associated with chronic stressors that lead to distress. The

second explanation assumes that different cultural patterns vary with regards to beliefs, values, and life-styles, and that psychological well-being is a consequence of these factors (Mirowsky & Ross, 1980). Roberts et al. (1997) and Seigel et al. (1998) found that ethnicity exerts a unique effect that cannot solely be accounted for by social class.

Unfortunately, it is difficult to draw any firm conclusions about the relation between ethnic status and depression, as the few studies that have examined this relation relied on different self-report measures of depression, and focused on different ethnic minority adolescents. We were unable to locate any studies that examined gender as a moderator of the effect of ethnic status on adolescent depression, although the study by Roberts et al. (1997) found that girls were at greater risk than boys across the different ethnic groups examined (African, Chinese, Anglo, and Mexican American).

Although some of the risk factors reviewed above appear to be robust predictors of adolescent depression, their specific role in the development of the gender disparity in adolescent depression is less clear. Whereas some studies have found gender differences in the predictive value of these risk factors, others have failed to show that gender moderates the relation between these risk factors and adolescent depression, or have examined the relations separately without expressly testing the moderation of gender and the purported risk factor (e.g., Gore, Aseltine, & Colton, 1992; Galaif, Sussman, Chou & Wills, 2003; Jenkins, Goodness & Buhrmester, 2002; Kaltiala-Heino, Rimpelo, Rantanen & Laippala,

2001; Leadbetter, Kuperminc, Blatt, & Hertzog,1999; Schwartz & Koenig, 1996; Slavin & Rainer, 1990). An explicit test of the gender-by-risk factor interaction is required to demonstrate that gender moderates the relation of the risk factor to depression, since examining these relations separately incorrectly assumes that depression has equal variance for both genders, resulting in a restriction in range (Baron & Kenny, 1986). In response to the inability of these established explanatory models to wholly account for the gender difference, several theorists have put forth the notion of a gender-specific model of depression as a way of explaining the abrupt rise of depression among adolescent girls. These models attempt to understand the process by which being female increases risk for development of depressive symptoms. Several influential models for explaining the gender differences in depression during adolescence will be subsequently be discussed.

1.3 Models of Gender Differences in Depression

1.3.1 Interactive Effects Model

Nolen-Hoeksema and Girgus (1994) propose that girls have characteristics that put them at risk for depression even prior to adolescence. The theorized risk characteristics include the propensity of girls to identify with feminine gender roles and thus less with instrumental (masculine) characteristics, less aggressive interaction styles, lower perceived competence, and more ruminative coping styles (Nolen-Hoeksema & Girgus, 1994). Nolen-Hoeksema asserts that girls

enter adolescence with a style of responding to stressors that is less efficacious than boys' style, and that this interacts with stressors to place girls at greater risk for depression (Nolen-Hoeksema, 1994). The specific stressors include pubertal development, the increased threat of sexual abuse, and the increased awareness of restrictive social expectations (Nolen-Hoeksema & Girgus, 1994; Nolen-Hoeksema, 1994).

Although the model proposed by Nolen-Hoeksema and Girgus (1994) is important in its developmental approach to understanding the increased prevalence of depression in adolescent girls, empirical support for some of the variables identified in this model has been inconsistent. To begin, components of sex-role identity did not interact with gender to predict depression in a prospective study of adolescent boys and girls (Petersen, Sarigiani, & Kennedy, 1991). Likewise, Cole et al. (1998; 1999) found that although increased depressive symptoms predicted children's tendencies to devalue their competencies, there was little evidence that lower perceived competence predicted future change in depressive symptoms.

As previously discussed, there is evidence that girls may be more likely to ruminate than boys. However, thus far the interaction of gender and rumination has not been appropriately tested in a sample of adolescents. Because this is a central component of this model, it will be important to test this relation.

Also problematic is the lack of empirical support for the developmental challenges suggested by Nolen-Hoeksema and Girgus (1994). Although they

point out the relation between sexual abuse and depression (Cutler & Nolen-Hoeksema, 1991), we were unable to locate any empirical evidence that adolescent girls fear the threat of sexual abuse or that this fear is associated with depressive symptoms. Similarly, we are not aware of any research demonstrating that increased awareness of social limitations among girls is related to increases in depression.

Only the third challenge delineated in Nolen-Hoeksema's model, early pubertal development, has been found to predict increased depression for girls relative to boys in prospective studies (Angold, Costello, & Worthman, 1998; Hayward et al., 1997; Petersen et al., 1991). It is important, however, to make a distinction between those studies that examine pubertal status and those that examined pubertal timing (i.e., the timing of puberty relative to one's peers). The effects of pubertal status are often confounded with age, making it difficult to interpret the predictive value of this variable. Therefore, we will discuss only those studies that examined pubertal timing as a risk factor for depressive symptoms.

Early puberty has been associated with elevated depressive symptoms for girls in several studies (Caspi & Moffitt, 1991; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Rierdan & Koff, 1991; Siegel, Yancey, Aneshensel, & Schuler, 1999; Wichstrom, 1999). However, other studies have not demonstrated the relation between pubertal timing and depressive symptoms for girls (Brooks-Gunn & Warren, 1989; Paikoff et al., 1991). What is more, the importance of

pubertal timing as a predictor of girls' depression has been questioned. In a longitudinal study that examined puberty as a risk factor for increases in depressive symptoms, early puberty accounted for less than one percent of the variance in depressive symptoms in a sample of adolescent girls (Stice, Presnell, & Bearman, 2001). Another longitudinal study of adolescent depression found that although early puberty had an initial effect on depressive symptoms, it was not significantly associated with depressive symptoms over time (Ge et al., 2001).

1.3.2 Extended Gender Intensification Model

Hill and Lynch (1983) suggested that the acceleration of gender-differential socialization during early adolescence leads to increased socialization of girls with feminine stereotypes and boys with masculine stereotypes. This process, thought to be triggered by pubertal development, theoretically leads to girls adapting a more helpless coping style compared to boys. In an elaboration of this model, Wichstrom (1999) suggested that this increased gender socialization places girls at greater risk for depression in the context of pubertal weight gain than boys because the stereotyped female sex role emphasizes attractiveness and because weight gain is at odds with the current ideal for female attractiveness. Wichstrom asserts that this dissatisfaction with appearance leads to negative assessment of global self worth which in turn leads to increased depressed mood for girls (Wichstrom, 1999).

Although theoretically appealing, some of the proposed risk factors highlighted in the Extended Gender Intensification Model have not been supported in longitudinal studies of depression among adolescents. In his test of this model, Wichstrom (1999) found that a lack of assertiveness and masculine instrumentality were not correlated with depression for adolescents. This is commensurate with previous studies that have also failed to find support for this relation (Allgood-Merten et al., 1990; Petersen et al., 1991). Although a number of studies have found support for both body dissatisfaction (Rierdon, Koff, & Stubbs, 1989; Siegal, 2002; Stice et al., 2000; Stice & Bearman, 2001) and self-esteem (Lewinsohn et al., 1994; Allgood-Merton et al., 1990) as risk factors for depression, the specific relationship whereby self-worth mediates the relation between body dissatisfaction and depression, proposed by Wichstrom (1999), has not been established.

1.3.3 Cultural Pressure to be Thin

A third theoretical model proposes that a cultural ideal of thinness for women directly causes increased body dissatisfaction in women, which then leads to depression among women at greater rates than among men (McCarthy, 1990). This model points to the similarity between the epidemiology of depression and eating disorders as evidence that body related concerns are related to depressive symptoms. According to McCarthy, these depressive symptoms are theoretically combated with eating pathology. McCarthy proposes that increased dieting or

bingeing initially alleviates the depressive symptoms, but that ultimately women who binge or fast become more depressed as a result of the disordered eating (McCarthy, 1990). McCarthy also notes that there is no culture that endorses the thin ideal that does not also have significantly more depression among women; likewise, all cultures that do not have more depression among women also do not have the thin ideal. The same is true of eating disorders (McCarthy, 1990).

Findings from longitudinal studies provide some support for this proposed model. Thin ideal internalization, body dissatisfaction, and bulimic behaviors have all been shown to prospectively predict the onset of depression (Stice et al., 2000) and depressive symptoms in girls (Stice & Bearman, 2001). Additionally, negative affect has emerged as a risk factor for future increases in bulimic symptoms (Cooley & Toray, 2001; Killen et al., 1996; Stice, 2001). Unfortunately, the complete model suggested by McCarthy (1990) has not been tested empirically to date.

1.3.4 Gender Additive Model

Drawing from the important theoretical contributions of the above models, the gender additive model suggests that as girls move further from the thin ideal during puberty, they subsequently develop body image and eating related risk factors for depression in addition to the risk factors they share with boys (Stice et al., 2000). Although some of these risk factors may also predict depression for boys, central to this theory is the notion that these gender specific variables exert

additional risk for girls, thus explaining the increase in depression for girls relative to boys during adolescence.

In theory, the idealization of the feminine body standard increases girls' risk for depression during adolescence, when pubertal weight gain increases the discrepancy between their own bodies and the ultra-slender ideal. For girls who have accepted that thinness is a valuable quality in society, growing increasingly deviant from this ideal may foster distress. In support of this model, initial levels of thin-ideal internalization as well as increases in thin-ideal internalization, but not body mass-index, predicted future increases in depressive symptoms for adolescent girls (Stice & Bearman, 2001).

In contrast to the thin ideal, evidence suggests that boys may subscribe to an ideal that emphasizes a mesomorphic build valuing muscularity over thinness (Jacobi & Cash, 1994; McCreary & Sasse, 2001; Moore, 1990; Smolak, Levine, & Thompson, 2001). Indeed, adolescent boys are more likely than girls to engage in behaviors to increase weight and musculature (McCabe, Ricciardelli, & Banfield, 2001). Theoretically, boys who have internalized this mesomorphic ideal would be vulnerable to depression when this ideal is not actualized. However, pubertal development in boys is characterized by increases in lean muscle mass, thus bringing them closer to their ideal physique. To our knowledge, the roles of ideal-body internalization and body mass in the development of depression have not been tested in a sample of adolescent boys.

As girls become increasingly invested in the thin-ideal and increase in adipose tissue following puberty, they also become increasingly body dissatisfied (Presnell, Bearman, & Stice, 2004). Because appearance is a central evaluative concern for girls in Western culture, this dissatisfaction with their weight and shape may have a consequent impact on their mood. Boys, by comparison, experience significantly lower levels of body dissatisfaction relative to girls, and become more satisfied as they progress through adolescence (Gardner, Friedman, & Jackson, 1999; Hargreaves & Tiggemann, 2002; Presnell, Bearman & Stice, 2004). Furthermore, boys' dissatisfaction appears to be related to being either under or overweight, while average-weight girls demonstrate dissatisfaction with their bodies (Bearman, Presnell, Martinez, & Stice, 2004; Richards, Boxer, Petersen, & Albrecht, 1990).

In support of the proposed relation between body dissatisfaction and depression for girls, body dissatisfaction predicted both depressive symptoms (Cole, Martin, Peeke, Seroczynski, & Hoffman, 1998; Rierdon et al., 1989; Siegal, 2002) and depressive onset (Stice et al., 2000) for adolescent girls. Furthermore, a randomized prevention trial indicated that a program focusing on reducing body dissatisfaction produced marked reductions in girls' depressive symptoms and negative affect (Bearman, Stice, & Chase, 2003). Only one study that we located examined this variable as a predictor of boys' depression, controlling for initial depression. Results from this study suggest that body

dissatisfaction did predict depressive symptoms for boys (Seigal, 2002), but to a lesser extent than for girls.

Because the ideal physique is virtually unattainable for most girls, they may embark on a frustrating endeavor to change their weight and shape by restricting their caloric intake. Dietary restraint in turn is thought to contribute to depression because of emotional distress caused by repeated dietary failures or because of the effect of caloric deprivation on mood. Because many boys want to gain weight and increase muscle tone to approximate the male ideal, it has been suggested that they may turn to exercise, rather than dieting (Furnham & Calnan, 1998; Ricciardelli & McCabe, 2004) thereby decreasing boys' risk for depression relative to girls. Girls have reported higher levels of caloric restraint than boys (Bearman, Presnell, Martinez & Stice, 2004), and self-reported dietary restraint predicted depressive symptoms (Stice & Bearman, 2001) and onset of major depression (Stice et al., 2000) for adolescent girls. To our knowledge, this relation has not been examined in a sample of adolescent boys.

Bulimic symptoms, theoretically resulting from body dissatisfaction and dieting, may further contribute to increases in depressive symptoms among adolescent girls because of the guilt and shame associated with losing control of one's eating behaviors. In general, boys are less likely to experience symptoms of eating pathology than girls, suggesting that this might exert differential risk for depression. Bulimic symptoms predicted adolescent girls' depressive symptoms

(Stice & Bearman, 2001) and onset of girls' depression (Stice et al., 2000), but have not previously been tested among adolescent boys.

Thus, there is a great deal of support that thin-ideal internalization, body dissatisfaction, dietary restraint, and eating pathology play a role in the development of depression for adolescent girls. Indeed, there is also evidence that these risk factors predict depressive symptoms for girls above and beyond the predictive value of other well-known risk factors, such as negative affectivity and social support (Stice & Bearman, 2001). However, this model has not yet been tested in sample that compares both girls and boys. Moreover, some of the most robust hypothesized shared risk factors, such as stressful life events, were not included in the previous research by Stice et al. In order to perform a more complete test of the gender additive model, it will be necessary to show that gender moderates the relation of the gender specific risk factors such that these risk factors increase the risk of depression for girls significantly more than for boys. Additionally, these risk factors must predict depressive symptoms for girls above and beyond the predictive value of the risk factors found to predict depression for both boys and girls, thus partially explaining the prevalence of depressive symptoms among adolescent girls.

CHAPTER 2: CURRENT STUDY

2.1 Study Aims

The overarching aims of this study are to corroborate and extend past research on the risk factors that predict adolescent depression in a longitudinal, community study of adolescent girls and boys. Therefore, among the goals of this study are to replicate the consistent finding that rates of depression increase during adolescence, and to establish the predictive relations of stressful life events, social support deficits, negative affectivity, and externalizing behaviors on depressive symptoms and onset of depression in adolescents. This study will also examine the roles of attributional style, ruminative coping, and ethnic status on the development of depressive symptoms and onset, as these variables have received less support in previous research [1].

A second goal of this study is to examine the gender differences in both depressive symptoms and onset of depression for adolescence. Specifically, this study intends to replicate the evidence of the gender difference in the rate of new onset and increases in depressive symptoms of adolescent girls and boys and verify that this gender difference occurs by age 14. This study also aims to examine the role of gender as a moderator of risk factors of adolescent depression. This study will examine whether gender interacts with a set of variables hypothesized to be shared risk factors for boys and girls. This study will also

expressly test one proposed theoretical model of the gender differences of depression, the gender additive model. This model suggests that body dissatisfaction, thin-ideal internalization, body mass index, dietary restraint and eating disorder symptoms are more potent predictors of depression for adolescent girls than for boys. Furthermore, this model posits that this constellation of risk factors operates above and beyond other risk factors that predict depression for both genders.

Finally, the majority of studies examining risk factors for depression in adolescents have either studied variables in isolation, or in specific combination in the search of support of a particular theoretical model. For example, it is possible that one pathway to depression might involve stress-buffering, in which the interaction of stressors and social support deficits theoretically foster susceptibility to depression, but life stressors in the face of adequate support should not (Cohen & Wills, 1985). A cognitive vulnerability transactional-stress model, on the other hand, proposes that the confluence of negative explanatory style and stressful events increase risk for depression, whereas the experience of life stressors with a more adaptive cognitive style would be benign. It is also possible that risk factors for depression interact with one another in ways that have not been previously considered in these theoretical models. When risk factors interact in complex ways with one another to predict an outcome—for

[1] Both ruminative coping and attributional style were assessed at T2 and T3, but not at baseline. Therefore, these variables will be examined in terms of their relation to T3 depressive symptoms,

example, as a sequence of mediators—addressing one link of that sequence may result in an effect with little statistical or real-world significance (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001).

One way to deal with the possibility of multiple risk factors that interact synergistically is to develop a risk classification based on multiple factors to define groups that face high versus low risk of the outcome (Kraemer et al., 1997). In light of this, a final goal of this study to explore interactions between the risk factors that identify subgroups of participants with unique risk factor profiles that might signal qualitatively different pathways to depression using classification tree analysis (CTA). This exploratory analytic technique is uniquely suited to explore the possibility that individuals take distinct pathways to depression because it does not assume linear effects, making it more sensitive than standard approaches (Breiman, Friedman, Olshen & Stone, 1984). The multivariate CTA models, which are particularly sensitive to detecting higher-order interactions, could provide a rich description of the relations between the hypothesized risk factors and depression.

2.2 Hypotheses

Hypothesis 1. Adolescent depressive symptoms and onset of depression will increase from age 13 to age 16 for both genders.

Hypothesis 2. Girls will show greater increases in depressive symptoms and depression onset from age 13 to age 16 relative to boys.

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but will not be included in the analyses examining all baseline risk factors.

Hypothesis 3. By age 14, girls will evidence significantly higher levels of depressive symptoms and onset of depression than boys.

Hypothesis 4. Stressful life events, deficits in social support, negative affectivity, externalizing behaviors, attributional style, ruminative coping, and ethnic status will predict depressive symptoms and onset of depression in univariate models of the combined sample of adolescent girls and boys (main effects).

Hypothesis 5. Body Dissatisfaction, ideal-body internalization, body-mass index, dietary restraint, and eating disorder pathology will predict depression in univariate models of the combined sample of adolescent girls and boys (main effects).

Hypothesis 6. Gender will moderate the relations of body dissatisfaction, dieting, and bulimic symptoms to future increases in depressive symptoms and onset of depression, but will not moderate the relations of stressful life events, deficits in social support, negative affectivity, externalizing behaviors, ruminative coping, attributional style, or ethnic status to future increases in depressive symptoms or onset of depression in separate models testing for the main effects of sex, each individual risk factor, and the interaction of sex and each risk factor.

Hypothesis 7. The gender specific variables will predict growth in depressive symptoms and onset of depression for girls even when the shared risk factors (stressful life events, deficits in social support, negative affectivity,

externalizing behaviors, attributional style, ruminative coping, and ethnic status) are entered into the multivariate model.

Hypothesis 8. The classification tree analyses will reveal interactions between the risk factors that indicate unique pathways for adolescent depression. We hypothesize that two main pathways will emerge. Based on a wealth of evidence demonstrating the cognitive vulnerability-transaction stress hypothesis, we expect that the interaction of stressful life events, externalizing behaviors and negative affectivity will increase the likelihood of depression for all adolescents. Next, sex will emerge, with girls facing an increased risk relative to boys. For girls with higher levels of body mass-index, thin-ideal internalization, body dissatisfaction, dietary restraint, and eating pathology, the probability of future depression will be higher than for girls with lower levels of these variables.

CHAPTER 3: METHODS

3.1 Participants

Participants were 247 adolescent girls and 181 adolescent boys from four public (89%) and four private (11%) middle schools in a large metropolitan area of the Southwestern United States. Adolescents ranged in age from 12 to 16 (Mean = 13.57) at baseline. The sample was composed of 2% Asian/Pacific Islanders, 4% African Americans, 64% Caucasians, 18% Latina, 1% Native Americans, and 5% who specified "other" or mixed racial heritage, which was representative of the ethnic composition of the schools from which we sampled (2% Asian/Pacific Islanders; 8% African Americans, 65% Caucasians, 21% Hispanics; 4% "other or mixed"). Highest educational attainment for parents ranged from grade school graduate (2%) to graduate degree (19%) with a mode of college graduate (42%), which was representative of the city from which we sampled (34% high school graduate or less; 25% some college; 26% college graduate; 15% graduate degree).

3.2 Recruitment

The study was presented to parents and participants as an investigation of adolescent mental and physical health behaviors. Parents of all eighth grade girls and boys from the participating schools were sent a description of the study along with an informed consent letter and stamped self-addressed return envelope. A second mailing was sent to non-responders after 2 weeks. Active parental consent

was obtained from all participants, and adolescent assent was also secured immediately before data collection took place. This resulted in an average participation rate of 53% of eligible students across schools. This participation rate was similar to that observed in other school-recruited samples that used active consent procedures and involved structured interviews (e.g., 61% for Lewinsohn et al., 1994).

Participants completed a self-report questionnaire, participated in a structured psychiatric interview, and had their weight and height measured by research assistants at baseline (T1) and at 1 and 2-year follow-up (T2 & T3). Assessments took place during elective courses during regular school hours or immediately after school on the school campus or in the participants' homes. Interviews were conducted by clinical assessors with a bachelors, masters, or doctoral degree in psychology. Clinical assessors attended extensive training sessions that focused on diagnostic criteria and delivery of structured interviews, and demonstrated an interrater agreement (kappa [k] > .80) with experts using tape-recorded interviews before collecting data. To prevent interviewer drift, assessors were recorded periodically throughout the study to document continued acceptable inter-rater agreement (k > .80) with experts. Participants received a $15 gift certificate to a local book and music store as compensation for participating in the study. This project received human subject's approval from the University of Texas Institutional Review Boards, as well as from the Austin Independent School District.

3.3 Design Considerations

Although using primarily self-report measures introduces the possibility that reporter bias accounts for the findings, many of the variables (e.g., body dissatisfaction, depressive symptoms, eating pathology,) would be unlikely to be accurately reported by others (Wilson, 1987). The use of structured interviews to assess depressive symptoms and bulimic symptoms, and the use of calibrated scales and stadiometers to assess body-mass index, should rule out the possibility that method variance accounts for the findings.

Other design considerations concern the age of the sample and the length of follow-up. Although it would have been preferable to follow participants from pre-adolescence until early adulthood, this was not possible given time and budgetary considerations. Since many researchers have found that gender differences in symptoms of depression emerge between 13-16 (Allgood-Merten et al., 1990; Ge et al., 1994; Girgus, Nolen-Hoeksema, & Seligman, 1989; Hankin et al., 1998), this seemed the optimal age on which to focus.

Additionally, we chose to include subclinical levels of major depression in our prospective analyses on depression onset. Given a sample of 429 participants, and the 4-6% annual onset rate that has been found in prior studies (Lewinsohn et al., 1993; Burton, Stice, & Seeley, 2004), we estimated that approximately 30 participants who were not depressed at baseline would develop a diagnosis of major depression over the two-year study period, and that the majority of these

would be girls. Including participants with subclinical depression increased our power to detect gender differences. Additionally, there is evidence that subclinical depression produces a similar level of functional impairment as is observed in full threshold major depression (Gotlib et al., 1995). In support of this assertion, only about half of adolescents referred for clinical services meet complete diagnostic criteria for depression (Compas, Ey, & Grant, 1993).

Finally, the question of how to optimally examine risk factors that may be implicated in the gender difference in adolescent depression must be considered from an analytic perspective. One the one hand, it is possible to consider these risk factors as mediators of the gender difference in depression. From this perspective, it would be necessary to establish the following: first, that gender predicts differences in the risk factors; second, that the risk factor predicts depression; and third, the relation of the risk factor to depression significantly reduces (or completely accounts for) the relation of gender to depression (Baron & Kenny, 1986). Conceptually, a mediational perspective also assumes a causal relationship between the independent variable and the proposed mediator—for example, that being female causes increased stressful life events or social support deficits (Baron & Kenny, 1986). Utilizing this conceptual and statistical framework, researchers have examined whether body image and self-esteem (Allgood-Merten, Lewinsohn, & Hops, 1994), self-competence (McCauley Ohannessian, Lerner, Lerner, & von Eye, 1999), negative attributions (Cole et al.,

1999) and interpersonal stress (Rudolph, 2002) mediated the gender difference in depression, with varying results.

In contrast, a moderator is a variable that affects the strength or direction of the relation between the risk factor and the outcome. From this perspective, it is necessary to examine first the relation of the proposed risk factor and the proposed moderator to the outcome, and then demonstrate that the interaction of the risk factor and the moderator predict the outcome. Conceptually, this perspective implies that the causal relationship between the risk factor—deficits in social support, for example—and future increases in depression varies as a function of the moderator variable, gender (Baron & Kenny, 1986). A handful of studies have used this analytic framework to examine the relation of body image (Seigel, 2002), stress and support (McFarlane, Bellissimo, Norman, & Lange, 1994) negative attributions (Hankin, Abramson, & Siler, 2001) and negative affectivity (Gjone & Stevenson, 1997) to depression outcomes in adolescents.

The gender additive model posits that certain risk factors exert unique predictive effects on girls versus boys, in addition to the risk factors that girls and boys share. Thus, it is not that being female causes an escalation in these risk factors, but rather that girls are uniquely vulnerable to the impact of certain risk factors. Given this theoretical position, it seemed optimal to test for the evidence that gender moderated the relation of the risk factors to future increases in depressive symptoms and future onset of depression.

3.4 Measures

3.4.1 Self Report Questionnaire

Stressful Life Events. The Major Life Events scale (Monroe, Rohde, Seeley, & Lewinsohn, 1999), which assesses the occurrence of 14 stressful life events during the past year (e.g., “Did your parents get divorced or separated?”), was used to tap this construct. The number of items endorsed is summed for analyses. Because they represented symptoms of psychopathology (i.e. "tried to commit suicide" or "had problems with drugs or alcohol"), 5 of the items were excluded from the measure. Major life events scales have demonstrated test-retest reliability (r = .83; Compas, Davise, Forsythe & Wagner, 1987), as well as predictive validity (r = .42, Andrews, Lewinsohn, Hops & Roberts, 1993) and convergent validity with measures of depression (r = .32; Andrews et al., 1993). Since this measure assesses the frequency of distinct negative events, there is no theoretical reason to expect internal consistency of these items. Pilot testing revealed a test-retest r = .90 for this measure of stressful life events.

Negative affectivity. Twelve items from Buss and Plomin’s (1984) Negative Affect Scale were used to assess a propensity toward becoming emotionally distressed. Items were averaged for analyses. Research has found this scale to possess acceptable internal consistency (α= .82), test-retest reliability (r = .80), and convergent and predictive validity (Buss & Plomin, 1984; Stice et al., 1998). This scale had a α=.79 at T1.

Social Support. Perceived social support was measured with items adapted from the Network of Relationships Inventory (Furman & Buhrmester, 1985) assessing companionship, guidance, intimacy, affection, admiration, and reliable alliance from parents and peers. Items are averaged for analyses to form separate scales of parental support and peer support. The internal consistency (M α=. 89), test-retest reliability (M 1-month r =. 69), and convergent and criterion validity of this measure have been documented (Furman & Buhrmester, 1985; Furman, 1996). This scale had a α•= .81 at T1 in this sample. Parental support had a •α= .87, while peer support had a α•= .89 at T1.

Externalizing Behaviors. Adolescent's externalizing symptoms were assessed with items from the Child Behavior Checklist (Achenbach & Edelbrock, 1983). Adolescents reported on the frequency of 13 externalizing behaviors on a scale of 1 (never) to 5 (always). This scale has demonstrated acceptable test-retest reliability (r = .95; Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001) and predictive validity, as well as convergent validity with other measures of externalizing behaviors (Fischer, Barkley, Fletcher & Smallish, 1993). Our version of the CBCL evidenced appropriate internal consistency (α = .88) and 1-year test-retest reliability (r = .62; Stice, Barrera, & Chassin, 1998) and had a α•= .86 at T1.

Attributional Style. Items adapted from the Children's Attributional Style Questionnaire-Revised (CASQ-R; Kaslow & Nolen-Hoeksema, 1991) were used

to assess the extent to which participants made global, internal, and stable attributions about hypothetical negative and positive events. A composite score for negative and positive event attributions is generated to assess overall attributional style with lower scores reflecting a more depressive style. The internal consistency (α = .36-.63), test-retest reliability (r = .39-.64), and predictive validity of this scale with depression (r = .41) have been documented (Andrews et al., 1993; Metalsky & Joiner, 1992). This scale had a α = .49 at T1. This relatively low level of internal consistency is consistent with other studies that have used this scale (Andrews et al., 1993; Robins & Hinkley, 1989; Thompson, Kaslow, Weiss & Nolen-Hoeksema, 1998) and found that it correlated with self-reports of depression.

Ruminative Coping. Four items of the Ruminative Responses Scale (Jackson & Nolen-Hoeksema, 1998) were used to assess this construct. This scale asks participants to indicate their likelihood to ruminate in response to their own negative emotions on a scale of 1 (sometimes) to 4 (almost always). Items were averaged for analyses. This scale has acceptable internal consistency (α = .85, Nolen-Hoeksema & Jackson, 2001) and convergent and predictive validity (Butler & Nolen-Hoeksema, 1994). This scale had an α = .82 at T2 of this study, when it was first assessed.

Body dissatisfaction. Body dissatisfaction was assessed with an adapted form of the Satisfaction and Dissatisfaction with Body Parts Scale (Berscheid et

al., 1973). This scale asks participants to indicate their level of satisfaction with 9 body parts. Items are summed for analyses. This scale has acceptable internal consistency (α•= .94), test-retest reliability (r = .90), and predictive validity (Stice, 2001; Stice & Agras, 1998). Because some of the items on this scale appeared to assess satisfaction with body parts more relevant for females than males, four of the items were modified on the questionnaires completed by males. This scale had a α•= .93 at T1 (α•= .93 for girls and α•= .89 for boys).

Dieting. The Dutch Restrained Eating Scale (DRES; van Strien et al., 1986a) was used to assess dieting. Participants indicate the frequency of dieting behaviors using 5-point scales. Items are averaged for analyses. This scale has acceptable internal consistency (α•= .95), test-retest reliability (r = .82), convergent validity (with self-reported caloric intake), and predictive validity (Stice, 2001; van Strien et al., 1986b; Wardle & Beales, 1987). This scale had an α•= .92 at T1.

Body mass. The body mass index (BMI = Kg/M^2) was used to reflect adiposity (Pietrobelli et al., 1998). Height was measured to the nearest millimeter using a portable direct reading stadiometer. Participants were measured without shoes and with their bodies positioned such that the heels and buttocks were against the vertical support of the stadiometer and heads aligned so that the auditory canal and the lower rim of the orbit were in a horizontal plane. Body weight was assessed to the nearest 0.1 kg using digital scales with the participants

wearing light indoor clothing without shoes or coats. Two measures of height and weight were obtained and averaged for analyses to generate a T1 and T2 body mass index score. The BMI shows convergent validity (r .80 - .90) with direct measures of total body fat such as dual energy x-ray absorptiometry (Pietrobelli et al., 1998), and excellent test-retest reliability (9-month test-retest r = .92; Pietrobelli et al., 1998; Stice et al., 1999).

Ideal-body internalization. The Thinness and Restricting Expectancy Inventory (TREI; Hohlstein, Smith, & Atlas, 1998) assessed ideal-body internalization for the girls in this study. Items were selected from the TREI assessing agreement with statements concerning expected social and psychological benefits from achieving thinness using a 5-point response format ranging from 1 = *strongly disagree* to 5 = *strongly agree*. The TREI has adequate internal consistency (α = .98), test-retest reliability (r = .80), and convergent validity (Hohlstein et al., 1998). Because it has been demonstrated that the ideal body type for boys differs from that of girls (Labre, 2002; Smolak, Levine & Thompson, 2001) items were modified to reflect the expected benefits from achieving leanness and muscularity as well as thinness for males. This scale had a

3.4.2 Interview

Bulimic symptoms. The Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), a structured psychiatric interview assessing DSM-IV criteria for eating disorders, is used to generate an overall bulimic symptom composite. To streamline the interview, only the diagnostic items were administered. The EDE has acceptable internal consistency (M α = .80), inter-rater reliability (M k = .85), test-retest reliability (M r = .80), and discriminates between eating disordered individuals and controls (Fairburn & Cooper, 1993; Rizvi, Peterson, Crow, & Agras, 2000). All assessors are required to show a minimum (kappa) agreement with expert raters of .80 before starting data collection. The EDE has high inter-rater agreement (k = .88) and excellent test-retest reliability (k = 1.0). The 17 diagnostic items for bulimia were averaged to create an overall symptom composite, which demonstrated a three-week test-retest reliability of r = .86 and adequate internal consistency (α = .84; Stice & Bearman, 2001).

Depressive Symptoms and Depression Diagnoses. The ultimate outcome variables, depressive symptoms and diagnoses were assessed using an interview adapted from the DSM-IV criteria for major depression from the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Puig-Antich & Chambers, 1983). Our modified version of the K-SADS combined features of the epidemiological version with the present episode version. Clinical assessors attend 24 hours of training, wherein structured interview skills are

taught, diagnostic criteria for the disorders are reviewed, simulated interviews are observed, and interviews are role-played. Clinical assessors are required to show a minimum (kappa) agreement with expert raters of .80 before starting data collection. The K-SADS has been found to have acceptable test-retest reliability (k's = .60 - 1.00), inter-rater reliability (k's = .60 - 1.00) and internal consistency (α's = .68 - .84), and to discriminate between disordered and non-disordered adolescents (Ambrosini, 2000; Lewinsohn et al., 1993, 1995). A randomly selected subset of participants (5%) were interviewed within a three-day period by a second clinical assessor who was blinded to the initial interview, resulting in high inter-rater agreement (k = 1.0). Another randomly selected subset (5%) completed a second clinical interview with the same clinical assessor one week following their original interview, resulting in excellent test-retest reliability (k = 1.0).

The number of symptoms endorsed were summed and averaged to create a depressive symptom composite. The depression symptom composite had a α = .81 at T1 among a subgroup of this sample. For diagnoses of depression, DSM-IV criteria were applied, requiring at least five symptoms and interviewer-rated clinical impairment overlapping for duration of two weeks or longer (for full depression). Participants who reported the presence of at least five of the necessary overlapping symptoms and were rated by expert assessors as experiencing significant impairment, but who endorsed a subthreshold level on at

least one of these symptoms were given a subclinical diagnosis (DSM-IV Depressive Disorder Not Otherwise Specified).

CHAPTER 4: STATISTICAL ANALYSES

4.1 Analytic Strategy

4.1.1 Data Cleaning

Survey and interview data were double-entered to assure key punch accuracy, and were then carefully cleaned before analyses (e.g., checked for out-of-range values).

4.1.2 Preliminary Analyses

Prior to testing study hypotheses, analyses were conducted to characterize the sample. Independent t-tests were performed on all demographic variables and outcome measures to test for differences between boys and girls at baseline. Attrition analyses were conducted to determine the percentage of participants who did not provide data at all time points, and independent t-tests were performed to determine whether participants who withdrew from the study differed significantly from those who continued participation.

4.1.3 Descriptive Analyses

The relation of age and depression was examined using hierarchical linear modeling techniques (HLM; Bryk, Raudenbush, Cheong, & Congdon, 2000). For depressive symptoms, an unconditional Level 1 model was generated that estimated an equation for the slope of each participants' depressive symptoms over the three measurement periods as predicted by the intercept and slope

parameters of each individual's age, centered around the mean of the entire sample. For this model, the following equation was generated:

$$\textbf{Level 1:} \qquad \bullet = B_{0i} + B_{1i} * (AGE_i) + R_i$$

$$\textbf{Level 2:} \qquad B_{0i} = G_{00} + U_{0i}$$

$$_{1i} = G_{10} + U_{1i}$$

where B_{0i} represents depressive symptoms for individual i at the first data collection (T1); B_{1i} represents the linear trend in self-reported depression scores across the three data collections for individual i; AGE_i represents the linear trend for age across the three data collections for individual i; and R_i represents random error in depressive symptoms for individual i. For Level 2 of these models, G_{00} represents mean status of depressive symptoms for all participants at first data collection; U_{0i} represents random error in B_{0i} for individual i; G_{01} represents mean linear change in depressive symptoms for all participants; and U_{1i} represents random error in B_{1i} for individual i.

To investigate whether girls evidenced greater increases than boys in depressive symptoms as they increased in age over the course of the study (hypothesis 2), a Level 1 model was again fit to the data which estimated an equation for the slope of each participants depressive symptoms over the three measurement periods as predicted by the intercept and slope parameters of each individual's age, centered around the mean of the entire sample. Next, the uncentered intercept for sex was entered as a Level 2 covariate. The following

equation was generated to determine whether the slope generated by the Level 1 equation varied by the different levels of sex at Level 2 (0 = girls, 1= boys):

Level 1: $\bullet = B_{0i} + B_{1i}*(AGE_i) + R_i$

Level 2: $B_{0i} = G_{00} + U_{0i}$

$_{1i} = G_{10} + G_{11}*(SEX) + U_{1i}$

Similar models were generated to examine the relation of age and onset of depression. As above, we first examined the univariate relation of age to onset of major depression. Because depression onset is a dichotomous outcome, we used a Bernoulli sampling model and a logit-link function. To examine the relation of age to depression onset over the course of the study, the following equation was generated:

Level 1: $Prob(Y=1|\ ) = P$

$log[P/(1-P)] = B_{0i} + B_{1i}*(AGE)$

Where P is the log of the odds of depression onset, B_{0i} represents depression status for individual i at the first data collection (T1); B_{1i} represents the probability of depression onset across the three data collections for individual i; AGE_i represents the linear trend for age across the three data collections for individual i; and R_i represents random error in depressive onset for individual i. Level 2 random effects were excluded from these models because the number of iterations required in order for the analyses to converge exceeded the recommended value, suggesting that the estimated variances are likely to be close to zero (Raudenbush

& Byrk, 2002). To examine the hypothesis that girls would evidence higher rates of depression onset from age 12-18, the equation below was generated with gender entered as a Level 2 time-invariant covariate:

Level 1: $\mathbf{Prob(Y=1|\ B) = P}$

$\mathbf{\log[P/(1-P)] = B_{0i} + B_{1i} *(AGE)}$

Level 2: $\mathbf{B_0 = G_{00}}$

$\mathbf{B_1 = G_{10} + G_{11} *(SEX)}$

Finally, independent t-tests performed in SPSS compared the means of girls' and boys' depressive symptoms across all measurement periods at ages 13, 14, 15 and 16 to determine the age at which gender differences emerged (Hypothesis 3). For onset of depression, Chi-square analyses run in SPSS compared the rate of depression onset for girls versus boys at all measurement periods at ages 13, 14, 15 and 16 to determine the age at which gender differences emerged.

4.1.4 Prospective Analyses

Hierarchical linear modeling techniques were used to examine the prospective relations between study outcomes (growth in depressive symptoms, onset of depression) and the proposed risk factors at T1 (stressful life events, social support deficits, negative affectivity, externalizing behaviors, attributional style, ruminative coping, ethnic status, body dissatisfaction, ideal-body internalization, body mass index, dietary restraint, and eating disorder symptoms).

To aid in the interpretation of these analysis, time was anchored at baseline (T1=0) so that the intercepts would reflect the individual's level of depressive symptoms at baseline. All risk factor T1 intercepts were centered around the sample mean, producing an average of zero. In HLM, the T1 values for the outcome (T1 depressive symptoms, initial depression status) are used to estimate the parameters of change over time for each individual, thereby ensuring a truly prospective test. For all models, baseline levels of the risk factors were entered as Level 2 time-invariant covariates. For the outcome of depressive symptoms, the following equation was generated:

Level 1: $\bullet = B_{0i} + B_{1i}*(TIME_i) + R_i$

Level 2: $B_{0i} = G_{00} + U_{0i}$

$_{1i} = G_{10} + G_{11}*(RISK\ FACTOR) + U_{1i}$

where B_{0i} represents depressive symptoms for individual i at the first data collection (T1); B_{1i} represents the linear trend in self-reported depression scores across the three data collections for individual i; $TIME_i$ represents the linear trend for time across the three data collections for individual i; and R_i represents random error in depressive symptoms for individual i. For Level 2 of these models, G_{00} represents mean status of depressive symptoms for all participants at first data collection; U_{0i} represents random error in B_{0i} for individual i; G_{01} represents mean linear change in depressive symptoms for all participants, and G_{11} represents mean

linear change in depressive symptoms as predicted by baseline levels of each risk factor and U_{1i} represents random error in B_{1i} for individual i.

For the prospective analyses where depression onset was the outcome, we again utilized a Bernoulli sampling model and a logit-link to examine the relation of initial levels of each risk factor to future depression onset over the course of the study, as represented by the following equation:

Level 1: $\mathbf{Prob(Y=1|\ B) = P}$

$\mathbf{log[P/(1-P)] = B_{0i} + B_{1i}*(TIME)}$

Level 2: $\mathbf{B_0 = G_{00} + U_{0i}}$

$\mathbf{B_1 = G_{10} + G_{11}*(RISK\ FACTOR) + U_1}$

where P is the log of the odds of depression onset, B_{0i} represents depression status for individual i at the first data collection (T1); B_{1i} represents the probability of depression onset across the three data collections for individual i; $TIME_i$ represents the linear trend for time across the three data collections for individual i; and R_i represents random error in depressive onset for individual i. At level 2, G_{00} represents mean status of depression onset for all participants at first data collection; U_{0i} represents random error in B_{0i} for individual i; G_{01} represents mean linear change in probability for depression onset for all participants, G_{11} represents mean linear change in probability for depression onset as predicted by baseline levels of each risk factor and U_{1i} represents random error in B_{1i} for individual i.

4.1.5 Moderational Analyses

To assess whether gender moderated the relation between T1 risk factors and future increases in depressive symptoms or onset of depression, HLM models were generated to assess the effect of Level 2 time-invariant covariates of sex (0 or 1), T1 risk factor, and the interaction of sex and the T1 risk factor on the slope of the Level 1 unconditional model of depressive symptoms over time. For depressive symptoms, the following equation was generated:

Level 1: $\bullet = B_{0i} + B_{1i}*(TIME_i) + R_i$

Level 2: $B_{0i} = G_{00} + U_{0i}$

$_{1i} = G_{10} + G_{11}*(SEX) + G_{12}*(RISK\ FACTOR) + G_{11}*(SEX*RISK\ FACTOR) + U_{1i}$

Similar equations tested whether gender moderated the effect of initial risk factors on future onset of depression:

Level 1: $Prob(Y=1|\ B) = P$

$\log[P/(1-P)] = B_{0i} + B_{1i}*(TIME)$

Level 2: $B_0 = G_{00} + U_{0i}$

$B_1 = G_{10} + G_{11}*(SEX) + G_{12}*(RISK\ FACTOR) + G_{11}*(SEX*RISK\ FACTOR) + U_{1i}$

For those risk factors where the interaction with gender was a significant predictor of future increases in depressive symptoms or onset of depression,

follow up analyses of the simple effects of the T1 risk factor on depressive symptoms were conducted separately for boys and girls.

4.1.6 Mutivariate Prospective Analyses

To test whether the T1 risk factors identified by the gender-additional model continue to predict future increases in depressive symptoms and onset of depression above and beyond the predictive value of the T1 shared risk factors (hypothesis 7), depressive symptoms or probability of depression onset were modeled over time at Level 1 separately for girls and boys. T1 intercepts of gender-additional risk factors that prospectively predicted the outcomes were separately entered as Level 2 time-invariant covariates along with the intercepts of all shared risk factors that predicted future increases in depressive symptoms or onset of depression.

4.1.7 Exploratory Classification Tree Analyses

We used classification tree analysis (CTA) to test for interactions among the risk factors that identify subgroups with unique risk factor profiles that would suggest qualitatively distinct pathways to onset of depression. CTA uses an empirically based recursive partitioning approach that selects the optimal cut-point on the optimal predictor (of all possible cut-points and predictors) for generating subgroups with differential risk for a dichotomous outcome. This procedure is then repeated in each successive subgroup until there are no remaining predictors that identify subgroups at significantly differential risk or the

node sizes become too small. When different risk factors emerge for two branches from the same fork (i.e., the optimal predictor for the outcome in one subgroup is different than that for another subgroup), this signifies an interaction.

We used the Classification Tree procedure in S-Plus (S-PLUS 6 Guide to Statistics, 1998) to conduct the CTA. We entered all of the T1 risk factors as potential predictors of T2 or T3 depression onset among participants who did not meet criteria for full or subclinical depression at T1. We set the minimum terminal node size to be 20 participants to minimize the impact of influential outliers and used an alpha of .005 to reduce the risk of chance findings.

4.2 Results

4.2.1 Power Calculations

Primary power calculations are based on an N of 428 because HLM can accommodate missing data (Bryk, Raudenbush, Cheong, & Congdon, 2000). Power calculations focused on our ability to detect small effect sizes because the average effect size for risk factors in this study was small (M d = .26) according to Cohen's criteria. Assuming an N = 428, an alpha of .05, and directional tests, the power to detect a small effect size (h = .50) is greater than .88 for the univariate and multivariate HLM models (Cohen, 1988). Because we also examined the boys and girls separately in order to test for the simple effects of significant interactions, ability to detect small effects was calculated for these subgroups. For the girls, assuming an N = 247, and alpha = .05, and directional tests, the power to detect a small effect was .72 (Cohen, 1988). For the boys (N =

181), power to detect a small effect using the same criteria was .60. Because this is less than optimal power, tests of the simple effects should be interpreted cautiously.

4.2.2 Preliminary Analyses

In preliminary analyses we tested for attrition biases that might compromise the generalizability of the findings. Of the original 429 participants, 15 did not provide data at T2 (3.5%), and 19 did not provide data at T3 (4.4%), although only 10 participants did not provide data at both T2 and T3 (2.3%). Participants who dropped out of the study did not differ significantly from those who provided complete data on any of the variables considered in this study at T1. Because hierarchical linear modeling requires complete data for the dependent variables and covariates for each individual *at* a specific time point, but does not require complete data *across* all time points, even those participants who provided only baseline data are included in these analyses.

Independent t-tests were performed to determine gender differences in depressive symptoms and all risk factors at baseline. Girls reported higher levels of body dissatisfaction, peer social support, negative affectivity and dietary restraint at baseline, and higher levels of negative attributions and ruminative coping at T2. Boys reported higher levels of stressful life events, externalizing behaviors, and depressive symptoms at baseline. Of the 428 participants, 33 met initial criteria for full or subclinical major depression (24 girls, 9 boys) at

baseline. Means and standard deviations for all baseline variables, and the correlations among them, are provided in Table 1. Means and standard deviation for all baseline measures by gender are reported in Table 2.

4.2.3 Descriptive Analyses

The univariate relation between age and depressive symptoms was first investigated in an individual model to test whether depressive symptoms and onset increased from age 13 to 16 (Hypothesis 1). As predicted, increases in age predicted increases in depressive symptoms (= 0.22, t = 2.41, p < .001). Results of the analyses where sex was entered at Level 2 indicated that the cross-level interaction of age and gender was significant, with girls showing significantly greater increases in depressive symptoms over time than boys (=-0.08, t = -4.50, p < .001), as hypothesized (Hypothesis 2). In SPSS, independent t-tests compared the means of girls' and boys' depressive symptoms across all measurement periods at ages 13, 14, 15 and 16 to determine the age at which this gender difference emerged, hypothesized to be evident by age 14 (Hypothesis 3). Boys reported higher levels of depressive symptoms (M = 1.44) at age 13 than girls (M = 1.30, t = -3.191, p < .005), although this difference was no longer significant at age 14. By age 15, however, girls reported higher levels of depressive symptoms (M = 1.43) compared to boys (M = 1.34, t = 2.42, p < .05). By age 16, this effect was no longer significant (girls' M = 1.44; boys' M = 1.37). Figure 1 illustrates the effect of age on depressive symptoms for boys and girls.

Increases in age were also significantly associated with increases in depression onset (= 0.77, t = 4.74, p < .001), and results indicated that the cross-level interaction of age and gender increased the probability of depression onset, although it was only a trend, increasing the probability for girls more than for boys (=-0.63, t = -1.92, p < .06). Chi-square analyses run in SPSS compared the rate of depression onset for girls versus boys at all measurement periods at ages 13, 14, 15 and 16 to determine the age at which this gender difference emerged. Although there were no significant differences in onset at age 13 or 14, the gender difference was evident by age fifteen for the current sample (^{2} [1, N = 411] = 9.380, p = .002) and at age 16 (^{2} [1, N = 217] = 9.594, p = .002). Figure 2 illustrates the effect of age on depression onset for girls and boys.

4.2.4 Prospective Analyses

Hypotheses 4 and 5 assert that stressful life events, deficits in social support, negative affectivity, externalizing behaviors, ethnic status, body dissatisfaction, ideal-body internalization, body-mass index, dietary restraint, and eating pathology would predict future increases in depression in the full sample. To test these main effects, the baseline levels of the 10 proposed risk factors were included as Level 2 time-invariant covariates in individual univariate models. The following risk factors were significant predictors of future increases in depressive symptoms over the study period: negative affectivity (= 0.04, t = 2.49, p < .05, d = .13), deficits in parental social support (=- 0.04, t = -3.02, p <

.005, d = .13), externalizing behaviors (= 0.04, t = 2.77, p < .01, d = .16), and eating pathology (=0.04, t = 1.98, p < .05, d = .14). These models are reported in Table 3. Stressful life events, deficits in peer support, ethnic status, ideal-body internalization, body-mass index, body dissatisfaction, and dietary restraint did not prospectively predict future increases in depressive symptoms for the combined sample.

Similar univariate models, also reported in Table 3, were generated to examine the relation between the risk factors introduced at T2 (attributional style and ruminative coping) and future increases in depressive symptoms from T2 to T3. Negative attributional style was a significant predictor of future increases in depressive symptoms for the combined sample (= -0.03, t = -3.08, p < .005, d = .16). Ruminative coping was also a significant predictor of future increases in depressive symptoms from T2 to T3 (= 0.28, t = 7.14, p < .001, d = 1.42).

For depression onset, the following T1 risk factors were significant predictors of depression onset over the study period: negative affectivity (= 0.43, t = 4.11, p < .001, OR = 1.54), deficits in parental social support (=- 0.35, t = -4.66, p < .001, OR = -1.42), externalizing behaviors (= 0.30, t = 3.24, p < .005 OR = 1.35), body dissatisfaction (= 0.25, t = 3.17, p < .005, OR = 1.28), dietary restraint (= 0.18, t = 3.11, p < .005, OR = 1.20), and eating pathology (=0.30, t = 3.54, p < .005, OR = 1.35). Stressful life events did not significantly predict onset of depression, but results indicated a trend (= 0.07, t = 1.82, p = .07, OR = 1.06). These models are reported in Table 4.

4.2.5 Tests of Gender Moderation

Hypothesis 6 proposed that gender would moderate the relations of the gender additive, but not the shared, risk factors to depression outcomes. To test whether gender moderated any of the univariate effects of the risk factors, T1 risk factors, sex, and the interaction of sex and the risk factor were entered as Level 2 variables to the models described above. Results indicated that the interaction of sex and the T1 risk factors significantly predicted future increases in depressive symptoms for the following variables: deficits in peer social support (= 0.07, t = 2.65, p < .01, d = .28) and negative attributions (=0.04, t = 2.50, p < .05 d = .17). Post-hoc analyses demonstrated that both variables significantly predicted future increases in depressive symptoms for girls, but not boys. The interaction of sex and both body dissatisfaction (=-0.04, t = -1.88, p = .06, d = .14), and dietary restraint (=-0.03, t = -1.90, p = .06, d = .12) approached significance at the trend level. The same procedure was followed for the models predicting onset of depression. For these models, results indicated that the interaction of sex and T1 risk factors significantly predicted future depression onset for the following: body dissatisfaction (=-0.40, t = -1.98, p < .05, OR = 1.49) and dietary restraint (=-0.39, t = -2.79, p < .01, OR = 1.48). Post-hoc analyses revealed that body dissatisfaction and dietary restraint predicted onset of girls', but not boys' depression.

4.2.6 Test of the Gender Additive Model

The baseline values of the T1 shared risk factors and the significant gender additive risk factors were included in separate models of depressive symptoms or onset in order to test Hypothesis 7, that the gender additive risk factors would demonstrate incremental predictive utility for girls, above and beyond the shared risk factors. Stressful life events, deficits in peer social support, and ethnic status were not included in the analysis of depressive symptoms, since they did not predict depressive symptoms for the combined sample. Likewise, ideal-body internalization and body-mass index were excluded from these models because they did not predict depressive symptoms in previous models. Body dissatisfaction, dietary restraint, and eating pathology were no longer significant predictors of future increases in depressive symptoms for girls when negative affectivity, deficits in parental social support, and externalizing behaviors were included in the model.

For depression onset, deficits in peers social support and ethnic status were not included in the set of shared risk factors, since they did not predict depression onset for the combined sample. Ideal-body internalization and body mass index were also excluded. Body dissatisfaction remained a significant predictor of future onset of depression for girls, even when stressful life events, negative affectivity, deficits in parental social support, and externalizing behaviors were included in the model (=-0.21, t = 2.29, $p < .05$, OR = 1.22). Dietary restraint

and eating pathology did not demonstrate a predictive effect on future onset of depression for girls when the shared risk factors were included in the model. These results are reported in Table 5.

4.2.7 Classification Tree Analyses

We used classification tree analysis (CTA) to test for interactions among the risk factors that identify subgroups with unique risk factor profiles that would suggest qualitatively distinct pathways to onset of depression (Hypothesis 8). CTA uses an empirically based recursive partitioning approach that selects the optimal cut-point on the optimal predictor (of all possible cut-points and predictors) for generating subgroups with differential risk for a dichotomous outcome. This procedure is then repeated in each successive subgroup until there are no remaining predictors that identify subgroups at significantly differential risk or the node sizes become too small. When different risk factors emerge for two branches from the same fork (i.e., the optimal predictor for the outcome in one subgroup is different than that for another subgroup), this signifies an interaction.

We used the Classification Tree procedure in S-Plus (S-PLUS 6 Guide to Statistics, 1998) to conduct the CTA. We entered all of the T1 risk factors (age, sex, stressful life events, negative affectivity, deficits in parental support, deficits in peer support, externalizing behaviors, ethnic status, body-mass index, ideal-body internalization, body dissatisfaction, dietary restraint, and eating pathology)

as potential predictors of T2 or T3 depression onset among male and female participants who did not meet criteria for full or subclinical depression at T1. We set the minimum terminal node size to be 20 participants to minimize the impact of influential outliers and used an alpha of .005 to reduce the risk of chance findings. The CTA produced a tree with three forks and four terminal nodes (Figure 3). Participants who reported that they were moderately or extremely dissatisfied with their bodies were more likely to become depressed (^{2} [1, N = 358] = 31.175, $p < .001$, $r = .28$) than their more body satisfied counterparts. Participants with higher body dissatisfaction scores at T1 showed a .40 probability of developing depression, whereas participants with less body dissatisfaction showed a .08 probability of onset.

Stressful life events emerged as the next predictor, but only among the participants with lower T1 body dissatisfaction scores (^{2} [1, N = 323] = 10.95, $p = .001$, $r = .17$). In this subgroup, participants who reported more than 2.5 stressful life events at T1 showed a .20 probability of depression onset, whereas participants with fewer stressful life events showed a .06 probability of onset. Ethnic status emerged as the final significant predictor, but only among the participants with greater than 2.5 T1 stressful life events (^{2} [1, N = 49] = 7.98, $p = .005$, $r = .37$). In this subgroup, participants who were an ethnic minority showed a .40 probability of becoming depressed, whereas Caucasian participants showed a .07 probability of onset.

CHAPTER 5: DISCUSSION

The first aim of this study was to demonstrate that depressive symptoms and rates of depression onset increased from ages 13 to 16. Results were consistent with this well-documented finding (Ge, Conger & Elder, 2001; Ge et al., 1994; Kaltiala-Heino, Rimpela, Rantanen, Laippala, 2001; Hankin et al., 1998), as both depressive symptoms and onset of depression increased as a function of age increases over the course of the study. Among adolescents who did not meet initial criteria for depression, 13 adolescents experienced onset of major depression by T2 (3%). The rate of new cases doubled between T2 and T3, with 25 adolescents who had not previously met diagnostic criteria for depression experiencing an onset (6%). Age did not exert the same effect on both boys and girls in the sample, however. For girls, increases in age were associated with increased depressive symptoms and future onset, while for boys the reverse was true. This parallels the study by Ge et al. (2001) that documented girls' depressive symptoms increased from 7th to 12th grade while boys' depressive symptoms were highest in 7th grade. Although we hypothesized girls would evidence higher levels of depressive symptoms and onset by age 14, this difference did not become evident until age 15. The same pattern was observed for onset of depression.

A second aim of this study was to examine a set of risk factors that have been suggested to increase risk for adolescent depression. It has been posited that

adolescence represents a particularly vulnerable phase of development, in part because adolescents are presented with a number of unique life challenges (Ge et al., 1994). To begin, adolescents are confronted with dramatic physical transformations—both physical growth as well as the onset of pubertal change. A number of pivotal transitions also occur during the course of adolescence, among them transition from elementary school to middle school, and from middle school to high school (Kandel & Davis, 1982). Furthermore, life stressors may become more salient during adolescence as the result of the myriad psychosocial changes that occur during this time. Nonetheless, a majority of adolescents are able to successfully traverse this developmental phase. Therefore, in the hope of clarifying the etiology of this highly impairing disorder, we examined the set of risk factors addressed below.

5.1 Relation of Shared Risk Factors to Depression

5.1.1 Stressful Life Events

Stressful life events emerged as a prospective predictor of future onset of depression for boys and girls at the trend level. Stressful life events at baseline did not predict future increases in depressive symptoms, despite evidence from previous research suggesting that experiencing life stressors leads to increases in symptoms of depression (Ge et al., 2001; Nolen-Hoeksema et al., 1992; Windle, 1992). This same pattern of results, whereby stressful life events predicted future depression onset but not growth in symptoms was also found in a study of

adolescent girls (Burton, Stice, & Seeley, in press). In theory, experiencing a significant negative incident, such as the death of a loved one, parental divorce, or a major illness is thought to lead to elevations in negative mood and maladaptive beliefs (Bennett & Bates, 1995; Hankin & Abramson, 2001). It has been suggested that adolescents are at greatest risk when they incur multiple stressful events (Petersen, Sargiani, & Kennedy, 1991), and as we have previously noted, the number of reported stressful events increases over the course of adolescence (Larson & Ham, 1993). In the current study, adolescent boys reported significantly greater increases in stressful life events relative to girls. Although previous research has noted that girls experience higher rates of stressful life events than boys (Davies & Windle, 1997; Ge et al., 1994), studies have also demonstrated that girls reported more interpersonal negative events while boys reported more non-interpersonal events (Larson & Ham, 1993; Rudolph & Hammen, 1999; Windle, 1992). The current study used a measure that assessed more non-interpersonal events than stressors resulting from relationships. However, other studies utilizing the same measure of stressful life events have found that it was a robust predictor of future depression among adolescents (Lewinsohn et al., 1994), suggesting that the current study's weaker support for this relation is not due to our assessment tool.

5.1.2 Negative Affectivity

As expected, negative affectivity predicted both future increases in depressive symptoms and onset of depression in the current study. The role of negative affectivity as a predictor of depressive symptoms has been well documented (Elliott et al., 1994; Gjone & Stevenson, 1997; Leadbeater et al., 1999). Theoretically, individuals with emotionally labile temperaments are generally more vulnerable to experiencing sadness, anxiety, and anger than those with a less emotionally reactive temperament (Clark & Watson, 1991). These individuals may be more susceptible to perceptions of stress, and are posited to experience more distress in the face of this stress. As adolescence is associated with increases in negative affectivity (Larson & Richards, 1989) as well as an ever-increasing range of stressors (Ge et al., 1994), this represents a particularly challenging phase for individuals who are predisposed to experience negative emotions. We are confident that this predictive effect is not due to a criterion confound, since the content of the negative affectivity scale is distinct from that of the measure of depression, and since the T1 measure of depression was included in the univariate models, thus controlling for this correlation.

5.1.3 Social Support Deficits

Deficits in parental social support also significantly predicted future increases in depressive symptoms as well as onset of depression for the adolescent

sample. Adolescents are considered to be particularly sensitive to social processes, perhaps as a result of the heightened value placed on interpersonal relationships and the increasing levels of conflict with peers and parents that are common in adolescence (Sheeber, Hops, Alpert, Davis, & Andrews, 1997). Whereas adequate support from important relationships is thought to promote feelings of self-esteem and efficacy that may buffer the impact of life stressors and negative mood, adolescents who lack this support are thought to be at heightened risk of depressive symptoms. Interestingly, deficits in peer support did not predict either depressive outcome for the combined sample, although there was evidence that gender moderated this effect, discussed below. The null effects for the combined sample dovetails with the study by Lewinsohn et al. (1994) where parental but not peer support deficits were a significant predictor of adolescent depression onset in a coed sample. Although adolescents spend an increasing amount of time with friends outside the home, there is evidence that family relationships retain their salience throughout adolescence and remain more reliable predictors of depressive symptoms than peer relationships (Gore, Aseltine, & Colten, 1993; McFarlane et al., 1994; Stice, Ragan, & Randall, 2004). Another possibility is that lack of peer support is more often a consequence of depression rather than a causal factor, as there is evidence that depressed individuals experience erosions in their social support network as a result of their depressed affect and the strains this places on relationships (Bell-Dolan, Reaven, & Peterson, 1993). Many previous studies have failed to separate the constructs

of social support from peers versus parents; the results of this study suggest that there may be important differences in the impact of these two types of support on depression in adolescents, and that researchers should make an effort to examine them separately.

5.1.4 Externalizing Behaviors

Externalizing behaviors also predicted both depressive symptoms and onset of depression for the adolescent sample, mirroring the previous results that found support for this relation in studies examining depressive symptoms (Fergussan et al., 2003) and onset of depression (Fergussan et al., 2003; Lewinsohn et al., 1994). It is presumed that adolescents who exhibit these behaviors are at greater risk of negative costs such as difficulty with peers and parents, school problems, and legal problems (Fergussan et al., 2003). Thus, externalizing behaviors may increase the number of stressful life events for these adolescents, as well as disrupt the interpersonal relationships that might otherwise allay the effect of these events. Delinquent behaviors show increases in early adolescence and then decrease as individuals move through this developmental period (Moffitt, 1993), although there is evidence that these behaviors increase throughout later adolescence when they co-occur with depression (Beyers & Loeber, 2003). The brief but striking rise of adolescence-limited externalizing behaviors increases the risk for subsequent depressive symptoms and onset of depression during early adolescence. Although boys have been shown to demonstrate more externalizing

behaviors than girls during both prior to and during adolescence (Bongers et al., 2003; Broidy et al., 2003; Keiley, Bates, Dodge, & Pettit, 2000; Moffitt, 1993), the current study suggests that girls who exhibit these types of risk-taking behaviors are at equal risk of developing depressive symptoms and onset of depression as boys.

5.1.5 Ruminative Coping

Ruminative coping at T2 also predicted future increases in depressive symptoms in our sample, consistent with previous research that examined this prospective relation in adolescents (Abela, Brozina, & Haigh, 2002; Schwartz & Koenig, 1996). Nolen-Hoeksema (1994) proposed that ruminative coping leads to depressive symptoms for girls more than boys because of girls' greater propensity to passively focus on feelings of distress, thereby strengthening the cognitive accessibility and recall of negative feelings and beliefs. In contrast, boys are believed to take more active steps towards coping with their distress (Nolen-Hoeksema, 1994). However, in the current study, gender did not moderate the relation of rumination to depressive symptoms. It may be that this variable is better conceived as a potential mediator of the gender difference in depression, since it may not be that rumination is more potent for girls, but rather that girls are more likely to ruminate. Thus, rumination may operate similarly in girls and boys, but the latter show less of a preference for this coping style.

5.1.6 Ethnic Status

Although a number of cross-sectional investigations have noted that minority adolescents report more depressive symptoms than Caucasian adolescents (Emslie, Weinberg, Rush, Adams, & Rintelmann, 1990; Roberts, Roberts, & Chen, 1997; Roberts & Sobhan, 1992; Siegal, Aneshensel, Taub, Cantwell, & Driscoll, 1998), ethnic status was not a significant predictor of adolescent depression in the HLM models. It has been proposed that ethnic minority status may be associated with lower socioeconomic status. Therefore, ethnic differences in depression may be the result of stressful events that occur as a function of social class effects. Alternatively, it is possible that cultural beliefs, values, and lifestyles associated with different ethnic minority groups may increase the likelihood of depression (Mirowsky & Ross, 1980). The emergence of minority status as a variable that signaled risk of depression onset in the classification tree analysis, discussed later, lends some support to the notion that the interaction of minority status and stressful events increases the probability of developing depression.

5.2 Interaction of Gender and Shared Risk Factors on Depression

Whereas the majority of the shared risk factors showed similar relations to adolescent depression regardless of gender, two of the hypothesized shared risk factors relations' to depressive symptoms were moderated by gender: peer support deficits and attributional style. Both variables significantly predicted increases in

depressive symptoms for girls, but not for boys. The finding that peer support deficits predicted future growth in girls' depressive symptoms is consistent with the evidence that girls report more peer-related stressors than boys, and place more emphasis on the value of interpersonal relationships than boys (Hankin & Abramson, 2001; Rudolph, 2002). Rudolph (2002) found that friendship stress mediated the gender difference in depressive symptoms for boys and girls, further supporting the notion that the impact of peer relations on depression may differ for boys and girls.

Although other studies examining the predictive value of attributional style on depressive symptoms have supported its relation to adolescent depression (Hankin, Abramson, & Siler, 2001; Robinson, Garber, & Hilsman, 1995), previous cross-sectional studies reported that a negative attributional style was associated more with boys' depression than girls' (Hops, Lewinsohn, Andrews & Roberts, 1990; Nolen-Hoeksema, Girgus, & Seligman, 1992), and the evidence for gender moderation from prospective studies has been equivocal (Cole et al., 1999; Hankin et al., 2001). The tendency to attribute negative events to internal, stable, and global causes and positive events to external, unstable, and specific causes is thought to lead to depression because it fosters a negative view of oneself and the world (Abramson, Seligman, & Teasdale, 1978). These negative beliefs in turn are thought to advance a negative schema that influences the processing of information, resulting in a preference for information that confirms negative beliefs. The increases in self-absorption that are characteristic of

adolescence may promote negative attributions during this time (Garber, Weiss, & Shanley, 1993). Girls' greater cognitive vulnerability in particular may be related to more detailed encoding of life events by girls than boys (Davis, 1999; Seidlitz & Diener, 1998), resulting in better recall of emotional incidents for girls than boys. This is particularly resonant given that in this study girls experienced fewer stressful life events than boys. This strengthens the argument that it is not the frequency of negative events, but rather the meaning attached to those events, which accounts for the increased depressive symptoms for girls.

5.3 Relation of the Gender Additive Variables to Depression

In addition to these shared risk factors, we examined the predictive utility of a set of body image and eating related risk factors theorized to be stronger predictors of depression in girls than boys. The prominent increase of depression for girls relative to boys during adolescence underscores the importance of examining models that can account for this phenomenon. The gender additive model of depression proposes that the constellation of body-mass, ideal-body internalization, body dissatisfaction, dietary restraint, and eating pathology are more robust predictors of girls' depression than boys', and that during adolescence these risk factors accumulate and predict girls' depression above and beyond the value of the shared risk factors.

5.3.1 Body Mass Index

Body mass index did not predict future increases in depressive symptoms or future depression onset for girls or boys in the study, and this relation was not moderated by gender. This dovetails with previous studies that found null effects predicting girls' depressive symptoms (Galambos, Leadbetter, & Barker, 2004; Stice & Bearman, 2001) and depression onset (Galambos, Leadbetter, & Barker, 2004; Stice et al., 2000). In theory, girls' risk for depression increases during adolescence in part because pubertal weight gain makes them increasingly disparate from the ultra-slender ideal for females whereas boys may aspire towards a muscular ideal that is more attainable given their developmental trajectory of increased lean muscle mass during puberty (Jacobi & Cash, 1994; McCreary & Sasse, 2001; Moore, 1990; Smolak, Levine, & Thompson, 2001). However, the failure of this variable to predict differing depression outcomes in three independent studies is compelling evidence that body weight does not increase the risk for depression. The absence of this effect is striking, given the well-documented prejudice directed at obese individuals and the grim health outcomes that are associated with obesity (Marcus, 1993; Wadden, Womble, Stunkard, & Anderson, 2002)

One possibility is that body-mass index is too broad a measure of body size and shape, and other measurements of body change might be more sensitive to the physical changes adolescent girls experience as the result of pubertal

development. For example, changes in waist or chest measurements might more accurately reflect the developmental changes believed to trigger weight-related distress. It may also be the case that girls' impressions of their size and shape, rather than physical weight per se, are related to increases in depression. Interestingly, body mass at time one was also not a significant predictor of increases in body dissatisfaction over the course of the study, and this effect was also not moderated by gender. This, in conjunction with previous null findings for body mass, suggests that the gender differences in depression for girls and boys stem from attitudinal, rather than physical, differences.

5.3.2 Ideal-Body Internalization

Ideal-body internalization also did not predict increases in depressive symptoms or future depression onset for either gender in the adolescent sample. Although ideal-body internalization has been examined as a prospective predictor of depression in previous studies of adolescent girls (Stice & Bearman, 2001; Stice et al., 2000), to our knowledge this is the first study to test this relation among adolescent boys. As posited by the gender additive model, individuals who have accepted that attaining the ideal physique is a valuable quality in society may become increasingly frustrated and demoralized as their body grows away from this ideal during puberty. One possibility for the null results is that internalization of the ideal body interacts with other variables to increase vulnerability to depression. A study by Stice, Presnell, and Spangler (2002)

found that very slender girls' overvaluation of appearance did not significantly increase their risk for binge eating, while for their heavier counterparts it did; perhaps a similar interaction exists for the prediction of depression. It may be that the cost of "buying into" the importance of an ideal body is only relevant when an individual is deviant from this ideal.

5.3.4 Eating Pathology

Finally, eating pathology predicted increases in depressive symptoms and depression onset equally for girls and boys in the current study, in contrast to study hypotheses. According to the model put forth by McCarthy (1990), individuals may binge eat to provide comfort and distraction from adverse emotions. People may also use radical compensatory behaviors, such as fasting, to reduce anxiety about impending weight gain or because they believe it serves as an emotional catharsis. It has been suggested that extreme caloric restraint leads to decreased levels of tryptophan, an amino acid precursor of serotonin, which increases the likelihood of binge eating high-carbohydrate food to restore serotonin levels. Depression is also characterized by serotonin dysregulation, suggesting another possible link between eating pathology and onset of depression (Wurtman, 1993). Girls are more likely to experience bulimic symptoms than boys (Ricciardelli & McCabe, 2004); however, results of this study suggest that boys who do engage in bulimic behaviors likewise face an increased risk for depressive symptoms. Rates of eating disorders are increasing

among boys (Ricciardelli & McCabe, 2004); this may likewise increase the risk of onset of adolescent depression.

5.4 Interaction of Gender and the Additive Risk Factors on Depression

Body dissatisfaction interacted with gender to predict increases in depressive symptoms for the combined sample, although this was only a trend, and body dissatisfaction was a significant predictor of girls' future increases in depressive symptoms, but not boys'. In terms of depression onset, the interaction of body dissatisfaction and gender significantly predicted future onset, and the relation of body dissatisfaction to future depression onset was significant for girls, but not boys. In the past, body dissatisfaction has been found to be a significant predictor of depressive symptoms (Cole, Martin, Peeke, Seroczynski, & Hoffman, 1998; Rierdon et al., 1989; Siegal, 2002) and depressive onset (Stice et al., 2000) for adolescent girls. Because physical appearance is a central evaluative concern for girls in Western culture, feelings of displeasure with their physique may have a consequent impact of their mood. In comparison, boys may place more emphasis on other domains, such as sports ability or academic achievement (Hankin & Abramson, 1998). Whereas average-weight girls have been shown to experience high levels of body dissatisfaction, boys are more vulnerable to body dissatisfaction in response to being either under or overweight (Presnell, Bearman, & Stice, in press; Richards, Boxer, Petersen, & Albrecht, 1990). Thus, boys and girls alike may encounter the physical transformations of adolescence

but with gravely different consequences: as girls naturally develop they may be more likely to experience body dissatisfaction and its adverse consequences, such as depression.

Self-reported dietary restraint interacted with gender to predict increases in depressive symptoms for the combined sample, although this effect was only a trend. Follow-up tests demonstrated that this effect was significant for girls, but not for boys. The interaction of dietary restraint and gender significantly predicted future depression onset, and dietary restraint emerged as a significant predictor of girls' future depression only. Stice et al. (2000) also found evidence for this relation in adolescent girls. In theory, dieting may represent an attempt to alter one's size and shape in response to feelings of body dissatisfaction. However, because self-reported dietary restraint has been shown to predict weight gain rather than weight loss, as well as bulimic symptoms such as bingeing and purging (Field et al., 1999; Stice & Agras, 1998; Stice et al., 2002), it may set in motion a cycle of disordered eating, increases in weight, and feelings of shame and guilt. Boys may be less likely to diet in response to body dissatisfaction since they may strive to increase muscle mass, more effectively accomplished via increases in exercise (McCabe & Ricciardelli, 2001). Exercise has been shown to have a positive impact on mood (Calfas & Taylor, 1994), whereas the frustration associated with unsuccessful dieting may leave girls more susceptible to depression than boys.

Body dissatisfaction and dietary restraint were both stronger predictors of girls' future depression onset compared to boys, and similar trends were found for gender moderation of these risk factors on increases in depressive symptoms. These results provide partial support for the gender additive model of depression, which further posits that the body-image and eating related risk factors will predict depression outcomes for girls above and beyond the effect of the shared risk factors. As anticipated, body dissatisfaction continued to be significant predictor for onset of future depression among girls, even when stressful life events, negative affectivity, deficits in parental social support, and externalizing behaviors were included in the models. However, dietary restraint and eating pathology no longer remained significant when the shared risk factors were included. This suggests that body dissatisfaction is the most robust of the risk factors identified by this theoretical model, exerting unique effects for girls beyond the predictive value of other risk factors that generally increase adolescents' risk for depression. The evidence for the relative importance of body dissatisfaction in the etiology of depression is bolstered by the results of the classification tree analyses, discussed in the next section.

With the exception of body dissatisfaction, the variables from the gender additive model failed to show unique effects when the shared risk factors were included in the multivariate models of girls' future depression onset. The reverse was also true: when body dissatisfaction, dietary restraint and eating pathology were separately included in the multivariate models with the shared risk factors,

the unique effects for stressful life events and negative affectivity disappeared in all three tests, and externalizing behaviors failed to retain unique effects when dietary restraint and eating pathology were entered into the model. Only deficits in parental social support consistently remained a robust predictor of girls' depression onset when the complete set of both shared and gender additive risk factors were included. It is possible that the null effects for the majority of the risk factors is an indication that mediational relations exist among many of the risk factors, in which case one would not expect to see unique predictive effects since mediators account for the variance of the independent variable on the dependent variable (Baron & Kenny, 1986). On the other hand, some of these risk factors are highly correlated, so it is also possible that the multicollinearity precludes unique effects because these variables share variance.

It is worth noting that we appeared to have greater sensitivity for detecting effects when the outcome of interest was future onset of depression versus the continuous measure of symptom increases. Nearly all of the effects that demonstrated a trend in the latter analyses were significant in the former, including the hypothesized moderational effects of gender. Whereas the continuous outcome reflected a wide range of individuals who likely varied from clinically depressed to those who merely endorsed difficulty with somatic symptoms (e.g., change in weight or difficulty concentrating), the dichotomous outcome divided the participants into two qualitatively distinct groups based on expert diagnoses of clinical impairment and a quantitative threshold of symptoms.

Thus, there was greater homogeneity across the participants within the criterion reference group, possibly reducing the error variance as compared to the continuous outcome and providing a more powerful test of our hypotheses.

5.5 Pathways to Adolescent Depression

The final aim of this study was to identify subgroups with unique risk factor profiles that might signal alternative pathways to adolescent depression. The multivariate CTA models, which are sensitive to detecting non-linear effects, provided a somewhat different picture of the relations between the risk factors and depression onset, identifying interactions between the T1 risk factors that increased risk for onset of depression at T2 or T3.

Consistent with the HLM models, the CTA provided evidence that one pathway for the development of depression was through body dissatisfaction. Adolescents whose body dissatisfaction scores corresponded with *moderately dissatisfied* or *extremely dissatisfied* showed a 5-fold increase in the risk for depression onset relative to adolescents who reported greater satisfaction with their size and shape. Although body dissatisfaction is more ubiquitous for girls than boys (Ricciardelli & McCabe, 2001; Wood, Becker, & Thompson, 1996), the evidence that body dissatisfaction increased the likelihood of depression onset for the entire sample suggests that any adolescent experiencing body dissatisfaction is vulnerable to depression, regardless of gender.

For the participants who evidenced greater satisfaction with their bodies, number of stressful life events increased the risk for depression onset. Adolescents who reported greater than 2.5 stressful events were at three times the risk of their counterparts for developing depression. This risk increased again for those participants who experienced more than 2.5 stressful events and reported ethnic minority status. These adolescents faced a six-fold risk of onset for depression, compared to the risk faced by Caucasian adolescents who had experienced the same number of stressful events. This is consistent with prior research that has suggested that adolescents who accumulate a number of stressful life events are vulnerable to depression (Cohen & Wills, 1985). Specifically, previous studies documented that among adolescents who had no history of depression, steep increases in depression onset occurred for those individuals who had three or more stressful life events (Brown & Harris, 1978; Lewinsohn, Allen, Seeley & Gotlib, 1999). The interaction of stressful life events and ethnic status, moreover, lends support to the notion that minority adolescents are at greater risk for depression, above and beyond the impact of stressful life events (Mirowsky & Ross, 1980).

Overall, the results of the CTA suggest that there are two main pathways to depression. Body dissatisfaction clearly emerged as one route, and stressful life events as another—particularly for adolescents who have ethnic minority status. In contrast to our predictions, sex did not emerge as a variable that increases risk of depression onset. The fact that body dissatisfaction emerged as the first

pathway, however, is not devoid of gendered implications, since girls were considerably more body-dissatisfied than boys in this sample. We should also note that it was not possible to include negative attributional style and ruminative coping in this analysis, since they were assessed at a later time point than the other potential risk factors. Thus, we cannot rule out the possibility that inclusion of these variables might have resulted in different pathways toward onset of depression.

5.6 Strengths and Limitations

This study attempted to improve upon previous research of the etiology of adolescent depression by using a prospective design, employing structured diagnostic interviews and direct assessment of body mass, controlling for initial depression, and appropriately testing for gender moderation. Certain methodological limitations should nonetheless be acknowledged. To begin, our recruitment rate (53%) suggests that nearly half of the participants who were invited chose not to participate in the current study. Although our sample was representative of the community with regards to ethnicity and parental education, it is possible that other substantial differences exist between study participants and adolescents who did not participate. It would be useful for future studies to include a non-sample control group in order to increase confidence in the generalizability of the results. Second, two of the risk factors—ruminative coping and attributional style—were assessed only at T2 for this sample. We were

therefore unable to include them in the multivariate models with the risk factors assessed at baseline. Nonetheless, our results indicate that these variables did predict depressive symptoms for adolescents, and that a negative attributional style was a significant predictor for girls in particular.

A third limitation concerns the assessment procedures of this study. Because the measure of body dissatisfaction and thin-ideal internalization were modified for boys in order to include items relevant for both genders, it is possible that this may have introduced some measurement artifact. However, both of these modified measures demonstrated rates and correlates of body dissatisfaction and ideal-body internalization that are commensurate with previous studies of these constructs, increasing our confidence that they were adequately assessed. In the current study, boys' responses indicated the degree to which they had internalized a "male ideal" that emphasized leanness and muscularity, as well as thinness. Much less is known about the ways in which placing value on this ideal may impact males, although drive for muscularity has been found to correlate with low self-esteem and efforts to increase body mass in males (McCreary & Sasse, 2000). It would be useful for future studies to separate the effects of thin-ideal internalization from internalization of the muscular male ideal, since it is possible that they exert different effects on boys' mental health outcomes.

Another potential measurement artifact may exist because girls were assessed for the first time the year prior to the boys as a part of a larger, adjunct study of adolescent girls. As a result, the first assessment of the current study is

actually the second time girls completed this questionnaire. This may have introduced some measurement desensitization, perhaps explaining the anomalous finding that girls had lower levels of depressive symptoms compared to boys at T1 (Windle, 1955). However, other baseline measurements were consistent with prior research we have conducted examining adolescent girls, suggesting that these measurements may be judged reliable.

Another shortcoming of the current study regards the exclusion of certain risk factors that have been implicated in the development of adolescent depression. For example, research has suggested that competence may play an important role in the development of depression (Cole et al., 1999; McCauley Ohannessian, Lerner, Lerner, & von Eye, 1999). Pubertal timing has also been implicated in girls' higher rates of depression (Angold, Costello, & Worthman, 1998), but was not measured in this study. The way in which adolescents regulate negative affect may also be an important factor in the etiology of depression (Garber, Braafladt, & Weiss, 1995). Unfortunately, it was not possible to include all possible risk factors in this study because of constraints on participant time. We acknowledge, however, that the set of risk factors reviewed in this study is not exhaustive. Finally, whereas longitudinal data provide information regarding temporal precedence, third-variable explanations cannot be ruled out with a non-experimental design. Therefore, it is possible that some shared causal variable increases both the risk factors and depression.

As we have noted, future research might include other general risk factors that may be relevant for adolescent depression to more completely test the hypothesis that body image and eating related risk factors operate in addition to other variables. It would also be useful if future studies began with a younger aged cohort, in order to better delineate when gender differences emerge in the risk factors that predict adolescent depression, such as ruminative coping or body dissatisfaction. A younger cohort would also allow researchers to examine other potentially influential processes, such as pubertal development and transition from elementary school to secondary education. A study that incorporated assessment of parents and friends might also be helpful, in particular to disentangle constructs that are necessarily subjective, such as number of stressful life events or parental or peer support.

5.7 Theoretical Implications

The majority of risk factors consistently found to predict depression in prior research were also significant predictors of depression onset in the current study, although deficits in peer support and negative attributional style only predicted increases in depressive symptoms for girls. Furthermore, this study found partial support for the gender additive model of depression, as body dissatisfaction and dietary restraint emerged as significant predictors of girls', but not boys', depression. Contrary to the theoretical model, body mass index and ideal-body internalization did not predict depression for adolescents, and these relations were

not gender moderated. Eating pathology was a significant predictor of depression, but predicted equally for girls and boys. In the tests of incremental predictive utility, only body dissatisfaction remained a unique predictor of girls' future onset of depression when the effects of stressful life events, negative affectivity, parental social support deficits, and externalizing behaviors were included in the model. The role of body dissatisfaction as a robust predictor of depression was also highlighted by the CTA, wherein body dissatisfaction emerged as a variable that significantly increased the probability of developing depression. Experiencing more than 2.5 stressful life events also emerged as a predictor of depression onset, and the ability of the CTA to identify this previously documented threshold increases our confidence in this analytic technique. Among individuals who have experienced life stressors, minority status further increased the likelihood of developing depression.

The current study is the third in a series (Stice et al., 1994, Stice & Bearman, 2001) to demonstrate that body dissatisfaction, dietary restraint, and eating pathology contribute to the development of adolescent depression for girls, and the first to demonstrate that some of these risk factors exert unique risk for adolescent girls relative to boys. This study also identified other potential contributors to the emergence of gender differences in depression during adolescence. Because negative attributional style and peer support deficits are conceptually distinct from the body-image and eating related variables originally proposed by the gender additive model, this model must be reconsidered in order

to include these variables. An integrative model utilizing the results of the current study follows.

5.7.1 Revised Gender Additive Model

From a theoretical perspective, adolescent girls may develop negative attributional style as a result of the increasing self-consciousness and self-involvement that adolescence entails (Garber, Weiss, & Shanley, 1993), and this may be more salient for girls due to more detailed encoding for negative events compared to boys (Davis, 1999; Seidlitz & Diener, 1998). As we have previously discussed, negative attributional style has been directly linked to onset of depression. Additionally, negative explanatory style might foster consequent deficits in social support, since the dysphoria engendered by self-critical and pessimistic attributions is thought to interfere with interpersonal relationships (Joiner, Coyne, & Blalock, 1999). It is also possible that a maladaptive explanatory style might make girls more likely to perceive peers as unsupportive, even when they lack confirming evidence. A study that included measures of both perceived and actual support demonstrated that perceived support was a better predictor of mood disturbances in adolescence (Kistner, Balthazor, Risi, & Burton, 1999), theoretically because of the negative lens through which these children interpret ambiguous social cues. For girls, inadequate social support—whether accurately perceived or not—is believed to foster depression because of

the heightened importance placed on social relationships relative to boys (Cryanowski, Frank, Young, & Shear, 2000).

Deficits in peer social support may also lead to increases in body dissatisfaction for girls, since the perception that they are accepted and valued by their social environment is thought to help girls feel more positively about themselves (Stice, Spangler, & Agras, 2001). In support of this theoretical link, social support deficits predicted girls' body dissatisfaction in a prospective study (Stice & Whitenton, 2002). A negative attributional style might likewise lead to body dissatisfaction because maladaptive cognitions about one's physical appearance, as well as a tendency to recall negative body experiences and increased attention to feedback that confirms negative body beliefs would likely increase one's feelings of displeasure with their body (Cash & Grant, 1996). This body dissatisfaction may cultivate feelings of depression because of the importance placed on appearance for girls in western culture and pressure to conform to an unrealistic ideal of female beauty (Stice et al., 2000). As previously discussed, dieting may appeal to body-dissatisfied girls as a means of improving their physical appearance and, consequently, their social status. Yet this may ultimately result in increased demoralization since self-reported dieting has been associated with increased weight gain as well as disordered eating behaviors (Field et al., 1999; Stice & Agras, 1998; Stice et al., 2002). A schematic of the revised model appears in Figure 4. Such a revised model has the benefit of incorporating elements of a cognitive vulnerability-stress model, which

has garnered considerable empirical support for predicting increases in depression among children and adolescents (see Hankin & Abramson, 2001, for a review) with relevant eating and body image related concerns, all of which demonstrated that they increased girls' risk of adolescent depression. It will be important to test this revised model in future prospective studies of girls' depression.

5.8 Clinical Implications

In addition to improving our understanding of the etiology of adolescent depression and the gender differences that emerge, the results of this study might also provide future direction for prevention efforts. Depressive disorders appear in a substantial proportion of adolescents who are referred for clinical services (Petersen, Compas, & Brooks-Gunn, 1992), and there is evidence of a secular increase in rates of adolescent depression (Ryan et al., 1992). Furthermore, Ryan and colleagues (1992) assert that the observed secular increase in child and adolescent onset of depression is unlikely the result of genetic changes in the population, suggesting that environmental variables such as those identified in this study may play a role in increasing rates of this disorder. Longitudinal research has documented that adolescent onset of depression predicts a pattern of recurrent episodes during adolescence and into adulthood, as well as a worse course of the disorder compared to later onset (Compas et al., 1993). Perhaps most discouraging, research has demonstrated that the few adolescents who receive treatment for depression show little improvement (Weersing & Weisz,

2002). Clearly, preventing the development of depression is a compelling goal, given the tremendous costs of this impairing disorder both to individuals and society at large.

This study identified a number of malleable risk factors for adolescent depression that are already targeted in existing empirically supported adolescent depression prevention programs, such as negative attributional style and negative affectivity (Clarke et al., 1995; Spence, Sheffield, & Donovan, 2003). Still, a number of the variables that emerged as predictors of depression for boys and girls in this investigation are potentially malleable, and to our knowledge are not currently included in extant prevention programs. These include deficits in parental support, externalizing behaviors, ruminative coping, and eating pathology. Prevention interventions, when examined in randomized trials, also allow a test of etiologic relations identified in prospective research, since in theory the reduction of a causal risk factor should result in a decrease in the criterion variable (Kraemer et al., 1997). Examining the relations of potential risk factors to outcomes with this type of design is especially useful because it addresses the limitations of prospective studies by more effectively controlling for third-variable explanations.

This study also identified variables that increase boys' and girls' probability of becoming depressed. This has important clinical implications, since the identification of high-risk individuals is a vital component of successful prevention. It has been shown that preventive interventions for depression have

the largest impact on youth who face elevated risk of that outcome (Cardemil, Reivich, & Seligman, 2002; Clarke, Hornsbrook, Lynch, Polen, Gale, Beardslee, et al., 2001; Spence et al., 2003). This may be because these individuals are more motivated to experience the improvements promised by such an intervention, or because they are more able to apply newly learned skills to relevant domains in their lives. This study provides additional justification for existing prevention efforts targeting minority adolescents (Cardemil et al., 2002) and individuals who have experienced life stressors (Beardslee et al., 1997; Wolchik, West, Westover & Sandler, 1993) and suggests that adolescents with elevated body dissatisfaction might be an appropriate population for selective prevention efforts.

The current study also adds to the wealth of evidence that demonstrates girls' increased risk for depression during adolescence, and identified potentially influential variables that may play a role in this gender difference. Thus, girls should be a particular focus of preventive efforts for depressive disorders, with an emphasis on challenging negative beliefs, building interpersonal skills, increasing self and body esteem, and encouraging beneficial weight management behaviors, such as physical activity. Concerted efforts to focus on empirically supported risk factors, both in the design of prevention interventions and in the selection of high-risk recipients of prevention interventions, could help adolescent girls—as well as boys—successfully negotiate this potentially perilous developmental period.

TABLES AND FIGURES

Table 1. Correlations among the T1 Putative Risk Factors and T1 Depression, along with Means and Standard Deviation for Boys and Girls

	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	M	SD
1. Stressful Life Events	.23	-.25	-.06	.44	-.17	.06	.08	.11	.07	.10	.38	.22	.23	.03	1.40	1.43
2. Negative Affectivity		-.19	-.02	.48	-.10	.09	.12	.32	.25	.26	.40	.31	-.18	-.08	2.69	0.61
3. Support-Parent			.10	-.39	.08	-.06	.01	-.32	-.08	-.25	-.29	-.29	.05	-.01	3.96	0.84
4. Support-Peer				-.08	-.04	-.04	-.00	-.10	-.08	-.03	-.19	-.11	-.29	.05	4.16	0.77
5. Externalizing					-.11	.10	.13	.10	.14	.19	.44	.30	.17	.06	1.77	0.61
6. Ethnic Status						-.30	.04	-.09	-.18	-.14	-.14	-.03	.03	.08	4.42	1.12
7. Body Mass Index							-.06	.30	.42	.29	.08	-.03	.02	.07	21.66	4.69
8. Ideal-body Intern.								.11	.16	.17	.13	.08	.00	.00	3.22	0.75
9. Body Dissatisfaction									.38	.42	.22	.13	-.11	-.02	2.66	0.88
10. Dietary Restraint										.38	.20	.02	-.15	.01	2.02	0.87
11. Eating pathology											.36	.20	-.09	.08	0.32	0.50
12. Depressive Sxs.												.61	.11	.03	1.37	0.34
13. Depression Dx.													-.09	.09	0.78	0.27
14. Gender														.09	0.43	0.50
15. Age															13.56	0.55

Note. Absolute correlations greater than .09 are significant at $p < .05$

Table 2. Means and Standard Deviation for each T1 Variable by Gender

	Girls		Boys	
Variable	M	SD	M	SD
Stressful Life Events	1.12_a	1.22	1.77_b	1.59
Negative Affectivity	2.78_a	0.60	2.57_b	0.59
Support-Parent	3.92	0.90	4.00	0.74
Support-Peer	4.35_a	0.72	3.91_b	0.75
Externalizing	1.68_a	0.61	1.88_b	0.61
Ethnic Status	4.39	1.15	4.45	1.09
Body Mass Index	21.57	4.74	21.78	4.64
Ideal-body Intern.	3.22	0.67	3.22	0.84
Body Dissatisfaction	2.74_a	0.95	2.55_b	0.76
Dietary Restraint	2.13_a	0.91	1.87_b	0.80
Eating pathology	0.35	0.53	0.27	0.44
Depressive Sxs.	1.33_a	0.34	1.41_b	0.33
Depression Dx.	0.10	0.30	0.05	0.22
Age	13.52	0.53	13.62	0.57
T2 Ruminative Coping	2.24_a	0.65	2.08_b	0.62
T2 Attributional Style	2.45_a	2.39	1.72_b	2.39

Note. Means having different subscripts are significantly different at $p < .05$.

Table 3. Univariate Relations of Each Risk Factor to Adolescent Boys' and Girls' Depressive Symptoms

Fixed Effect						Random Effect				
Effect	*Parameter*	*Coefficient*	*Se*	*t*	*p*	*Parameter*	*Variance*	^{2}	*d.f.*	*p*
Stressful Life	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.74	420	<.001
Events	$_1$	0.00	0.005	0.95	ns	U_1	0.015	683.65	419	<.001
Negative	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.49	420	<.001
Affectivity	$_1$	0.04	0.014	2.49	<.05	U_1	0.015	697.48	419	<.001
Parental	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.77	420	<.001
Social Support	$_1$	-0.04	0.012	-3.02	<.001	U_1	0.015	691.318	419	<.001
Peer Social	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.78	420	<.001
Support	$_1$	0.00	0.014	0.03	ns	U_1	0.014	675.36	419	<.001
Externalizing	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.46	420	<.001
Behaviors	$_1$	0.04	0.016	2.77	<.01	U_1	0.016	702.36	419	<.001
Ethnic Status	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.74	420	<.001
	$_1$	-0.00	0.007	-0.66	ns	U_1	0.014	677.45	419	<.001
Body-Mass	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.45	420	<.001
Index	$_1$	0.00	0.002	1.08	ns	U_1	0.014	676.17	419	<.001
Thin-Ideal	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.40	420	<.001
Internalization	$_1$	0.00	0.010	0.24	ns	U_1	0.014	676.06	419	<.001
Body	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.78	420	<.001
Dissatisfaction	$_1$	0.02	0.011	1.61	ns	U_1	0.015	684.30	419	<.001
Dietary	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.76	420	<.001
Restraint	$_1$	0.01	0.009	1.02	ns	U_1	0.014	679.59	419	<.001
Bulimic	$_0$	1.36	0.016	83.95	<.001	U_0	0.072	1212.44	420	<.001
Pathology	$_1$	0.04	0.019	1.98	<.05	U_1	0.015	691.92	419	<.001
Negative	$_0$	1.36	0.016	85.11	<.001	U_0	0.072	1212.76	405	<.001
Attributions	$_1$	-0.03	0.009	-3.08	<.005	U_1	0.056	621.51	405	<.001
Ruminative	$_0$	1.36	0.016	85.11	<.001	U_0	0.072	1212.44	405	<.001
Coping	$_1$	0.28	0.039	7.14	<.001	U_1	0.035	540.85	405	<.001

Table 4. Univariate Relations of Each Risk Factor to Adolescent Boys' and Girls' Depressive Onset

	Fixed Effect					Random Effect				
Effect	*Parameter*	*Coefficient*	*se*	*t*	*p*	*Parameter*	*Variance*	^{2}	*d.f.*	*p*
Stressful Life	$_0$	-2.44	0.105	-23.18	<.001	U_0	1.346	267.27	415	ns
Events	$_1$	0.07	0.036	1.82	<.070	U_1	0.029	226.49	414	ns
Negative	$_0$	-2.45	0.110	-22.19	<.001	U_0	1.193	273.52	415	ns
Affectivity	$_1$	0.43	0.106	4.11	<.001	U_1	0.058	281.02	414	ns
Parental	$_0$	-2.45	0.110	-22.33	<.001	U_0	1.201	271.82	415	ns
Social Support	$_1$	-0.35	0.074	-4.66	<.001	U_1	0.051	266.68	414	ns
Peer Social	$_0$	-2.43	0.103	-23.46	<.001	U_0	1.413	265.51	415	ns
Support	$_1$	-0.06	0.081	-0.80	ns	U_1	0.178	221.10	414	ns
Externalizing	$_0$	-2.44	0.108	-22.73	<.001	U_0	1.272	269.62	415	ns
Behaviors	$_1$	0.30	0.092	3.24	<.005	U_1	0.040	247.51	414	ns
Ethnic Status	$_0$	-2.43	0.103	-23.55	<.001	U_0	1.429	264.50	415	ns
	$_1$	-0.04	0.048	-0.82	ns	U_1	0.029	214.21	414	ns
Body-Mass	$_0$	-2.43	0.103	-23.63	<.001	U_0	1.444	264.06	415	ns
Index	$_1$	0.01	0.012	0.97	ns	U_1	0.029	211.74	414	ns
Thin-Ideal	$_0$	-2.43	0.103	-23.46	<.001	U_0	1.413	265.27	415	ns
Internalization	$_1$	0.08	0.075	1.11	ns	U_1	0.030	218.76	414	ns
Body	$_0$	-2.44	0.106	-23.04	<.001	U_0	1.328	267.57	415	ns
Dissatisfaction	$_1$	0.25	0.080	3.17	<.005	U_1	0.036	241.42	414	ns
Dietary	$_0$	-2.42	0.103	-23.47	<.001	U_0	1.424	264.66	415	ns
Restraint	$_1$	0.18	0.058	3.11	<.005	U_1	0.030	216.76	414	ns
Bulimic	$_0$	-2.45	0.107	-22.81	<.001	U_0	1.256	270.51	415	ns
Pathology	$_1$	0.30	0.085	3.54	<.005	U_1	0.033	232.44	414	ns

Table 5. Multivariate Models of Gender Additive and Shared Risk Factors: Girls' Depression Onset

Fixed Effect						Random Effect				
Effect	*Parameter*	*Coefficient*	*se*	*T*	*p*	*Parameter*	*Variance*	^{2}	*d.f.*	*p*
Depression	B_0	-2.27	0.141	-16.05	<.001	U_0	1.313	170.88	240	ns
Body Dissatisfaction	G_1	0.21	0.091	2.29	<.05	U_1	0.130	169.62	235	ns
Stress. Events	G_2	-0.04	0.074	-0.52	ns					
Neg. Affect	G_3	-0.01	0.179	-0.08	ns					
Ext. Behavior	G_4	0.35	0.166	2.12	<.05					
Par. Support	G_5	-0.21	0.090	-2.39	<.05					
Depression	B_0	-2.27	0.141	-16.13	<.001	U_0	1.336	170.43	240	ns
Dietary Restraint	G_1	0.13	0.085	1.55	ns	U_1	0.133	159.15	235	ns
Stress. Events	G_2	-0.03	0.068	-0.43	ns					
Neg. Affect	G_3	0.07	0.174	0.42	ns					
Ext. Behavior	G_4	0.21	0.159	1.33	ns					
Par. Support	G_5	-0.28	0.088	-3.24	<.005					
Depression	B_0	-2.27	0.141	-16.12	<.001	U_0	1.328	170.10	240	ns
ED Pathology	G_1	0.07	0.124	0.56	ns	U_1	0.176	164.70	235	ns
Stress. Events	G_2	-0.03	0.070	-0.41	ns					
Neg. Affect	G_3	0.11	0.172	0.62	ns					
Ext. Behavior	G_4	0.23	0.154	1.51	ns					
Par. Support	G_5	-0.28	0.086	-3.30	<.005					

Figure 1. Mean Scores for Depressive Symptoms by Age and Gender

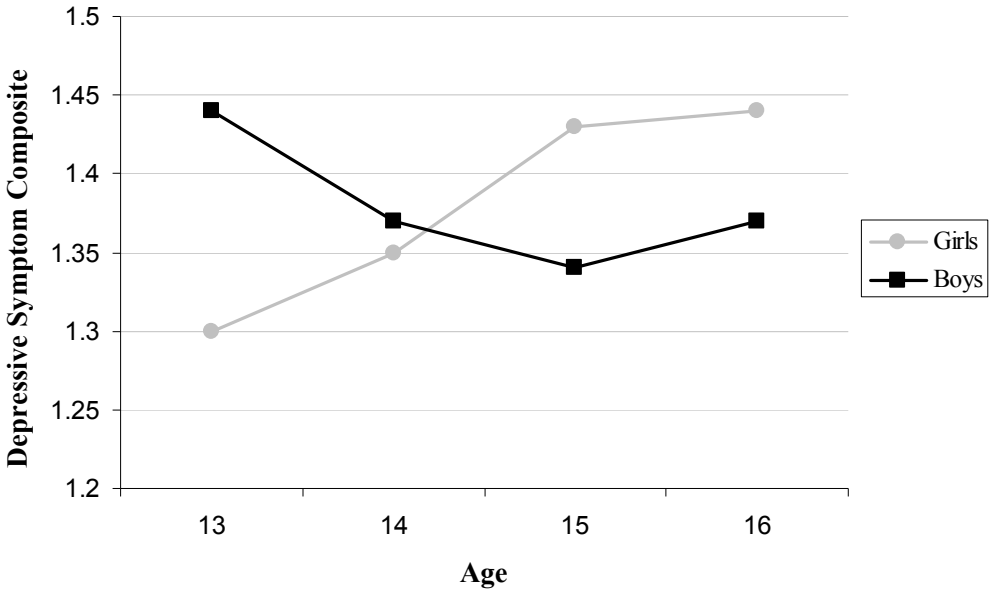


Figure 2. Depression Onset by Age and Gender

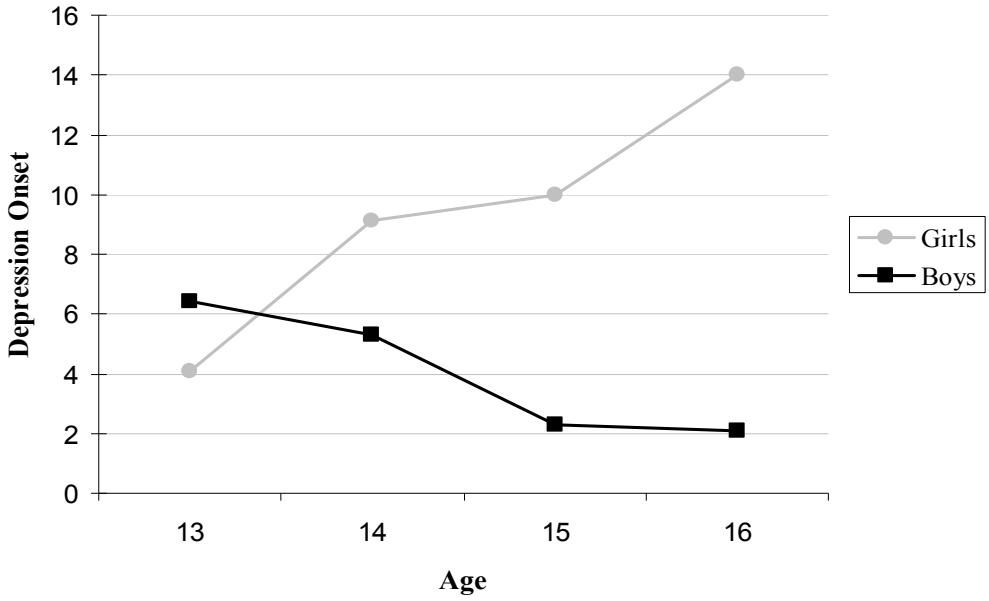


Figure 3. Results of Classification Tree Analyses for Depression Onset

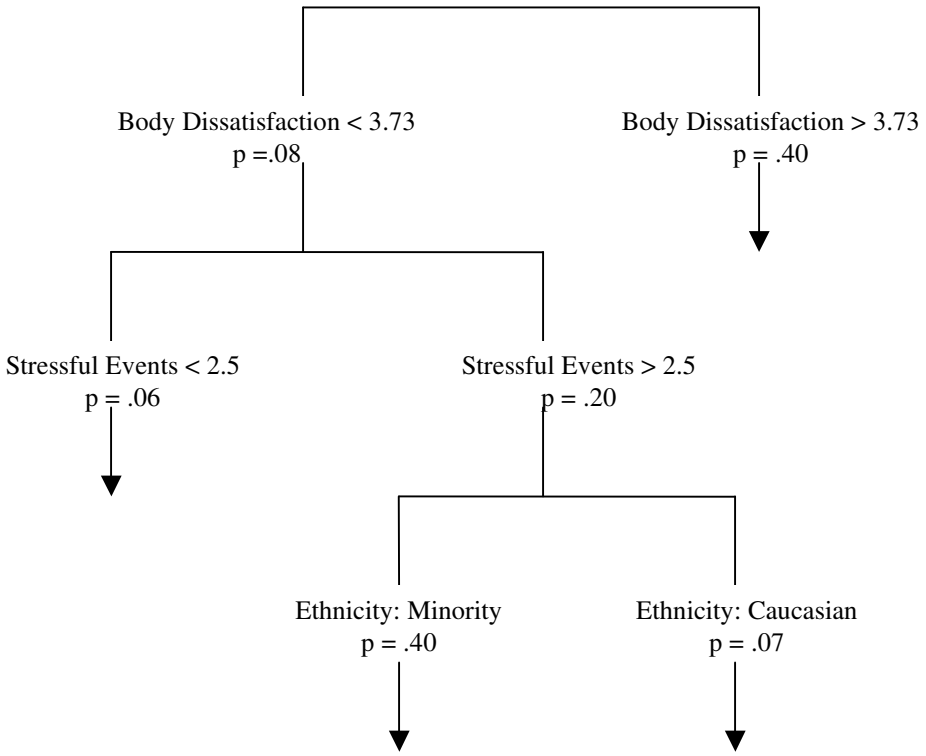
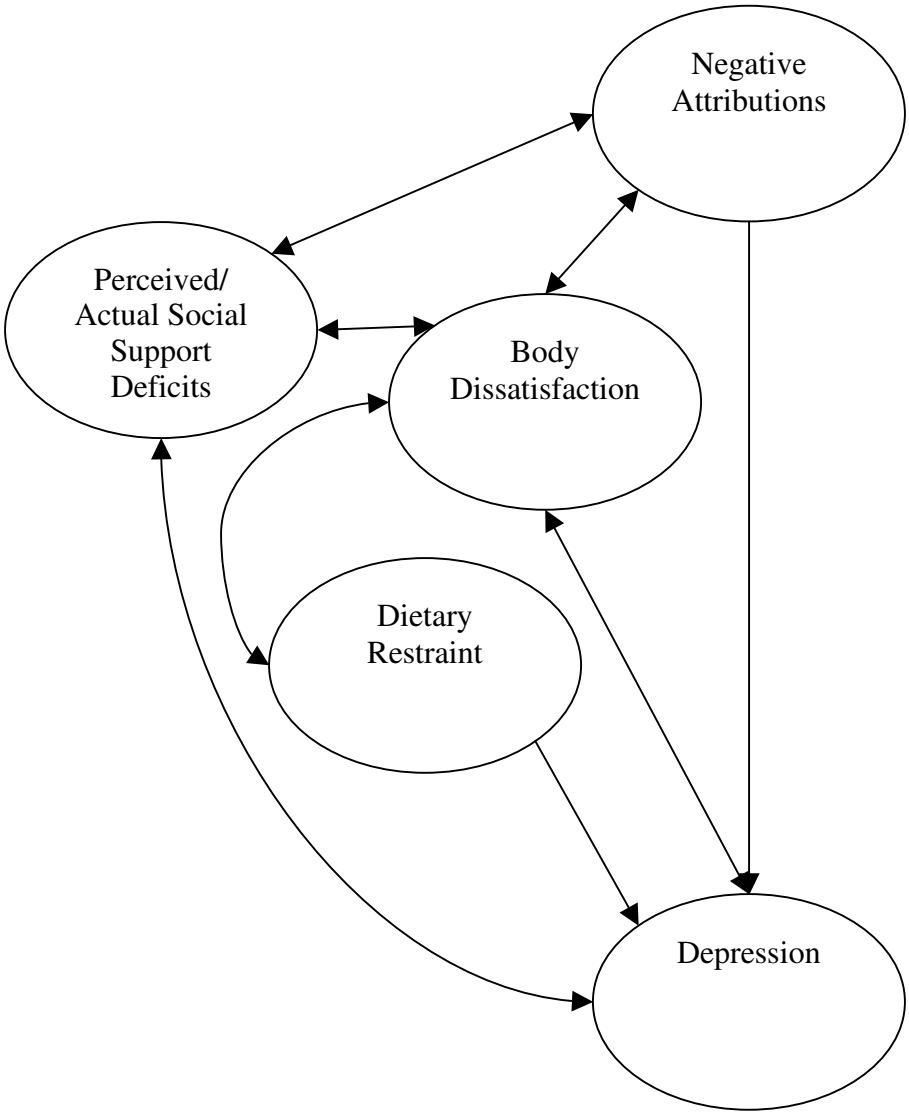


Figure 4. The Revised Gender Additive Model of Depression among Adolescent Girls



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