THE DEVELOPMENT AND MAINTENANCE OF ADOLESCENT DEPRESSION

AMY KERCHER

BPSYCH(HONS)

Centre for Emotional Health, Department of Psychology,
Faculty of Human Sciences, Macquarie University
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ABSTRACT

This research examined the longitudinal development of depressive symptoms among young adolescents (mean age 12 years). The first model examined depressive symptoms across 6 months in 315 young adolescents and their mothers, considering the mediation of perceived parenting and its influence on adolescent self-worth. Although parent-reported parental depression was not linked with child-reported perceived parenting, the child's perception of his or her mother as rejecting or less caring was associated with a lower sense of self-worth, which in turn predicted depressive symptoms 6 months later, controlling for initial depression. In the second model, tested across 12 months with 896 young adolescent girls, neuroticism served as a distal vulnerability for depression, conferring a risk of experiencing dependent stressors and negative automatic thoughts which fully mediated the effect of neuroticism on later depression. Initial depressive symptoms also followed this meditational pathway, in a possible maintenance and risk pathway for adolescent depression. Unexpectedly, independent stressors were also predicted by initial depressive symptoms, suggesting possible shared method or genuine environmental factors. Finally, it was proposed that young adolescents at risk of depression will not only display cognitive vulnerabilities contributing to increased depressive symptoms following stressors (cognitive diathesis-stress theory), but also be *more likely* to experience stressors at least partly dependent on their own behaviour (stress-generation theory). This model was supported with a large (N=756) sample of young adolescents across 6 months, controlling for initial depression. Taken together, this thesis extends previous theories about the aetiology of depression, providing evidence from family, personality and cognitive risk factors to better explain the development of depressive symptoms in early adolescence, with significant implications for intervention and treatment.

STATEMENT OF CANDIDATE

I certify that the work in this thesis entitled "The development and maintenance of adolescent depression" has not previously been submitted for a degree nor has it been submitted as part of requirements for a degree at any other university or institution other than Macquarie University.

I also certify that the thesis is an original piece of research and it has been written by me. Any help and assistance that I have received in my research work and the preparation of the thesis itself have been appropriately acknowledged. The additional authors included in Chapters 2, 3 and 4 were involved in the research at a supervisory level.

In addition, I certify that all information sources and literature used are indicated in the thesis.

The research presented in this thesis was approved by Macquarie University Ethics Review Committee, reference number: HE24MAR2006-D04582 on the 8th of September, 2006.

Amy Kercher (Student ID 30601630)

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CHAPTER ONE

INTRODUCTION

Adolescent Depression

Depression is a significant problem among adolescents, with point prevalence estimates of 1-2% in childhood increasing to 4-8% in adolescents over 13 years of age (Angold & Rutter, 1992; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). Initial increases in depressive symptoms are noted at around 13 years of age, with rates of depression increasing throughout adolescence (Costello et al., 2003; Fleming & Offord, 1990; Hankin et al., 1998). Adolescent depression includes symptoms of sadness, anhedonia, anger or irritability, social withdrawal and somatic complaints (Curry, 2001; Kovacs, 1996b), tends to last several months and is highly recurrent (Kovacs, Feinberg, Crouse-Novak, Paulauskas, & Finkelstein, 1984; Roberts & Bishop, 2005). Depression is expected to be the world's leading cause of death and disability by 2020 (WHO, 2001), when the current cohort of adolescents will be well into adulthood.

Adolescent depression is associated with significant impairments in social, academic and extracurricular functioning, suicide risk and with comorbid disruptive behavior, anxiety and substance use disorders (Curry, 2001; Puig-Antich et al., 1985a, 1985b; Weissman, Wolk, Goldstein, & al., 1999). A gender disparity in rates of depression first emerges in adolescence. By the age of 15 girls are twice as likely to suffer depression as boys (Angold & Rutter, 1992; Costello et al., 2003). Importantly, adolescent depression is strongly associated with depression throughout the lifespan, with a four-fold increase in the likelihood of suffering recurrent, severe depression in adulthood (Harrington, Fudge, Rutter, Pickles, & Hill, 1990). On this basis, research into the causes and origins of depression in adolescence is vital, with particular calls for research to begin in early adolescence, before the dramatic increases begin (Ge, Lorenz, Conger, Elder, & Simons, 1994; Roberts, 1999; Yap, Allen, & Sheeber, 2007).

Early adolescence is a critical time for the study of risk for depression, as it is the developmental period in which the first onset of depression occurs for many people (Cicchetti & Toth, 1998; Costello et al., 2003). It is a time of significant physiological, psychosocial and

biological changes, with the onset of puberty, academic and social demands in the transition to high school, and changing relationships with family possibly contributing to the onset of depressive symptoms (Ge et al., 1994; Schmidt & Maras, 2001; Yap et al., 2007).

Theories of Depressive Risk

Numerous theories have been proposed to explain the development of depression. Evidence supports a genetic component for depressive risk, with heritability estimates between 20-80% (see Donovan & Spence, 2001; Eley et al., 2008; Lopez-Leon et al., 2008; Rice, Harold, & Thaper, 2002). Strong evidence suggests that a combination of genetic vulnerability and environmental factors interact to predict depression (see Davey, Yücel, & Allen, 2008; Rutter, 2002; Shortt & Spence, 2006). Neurological theories focus on possible abnormalities in the functioning of serotonin, suggesting (among others) a model beginning with a particular polymorphism in the serotonin transporter gene interacting with the environment to predict depression (e.g., Caspi, Sugden, Moffitt, & al., 2003; Eley, Sugden, Corsico, & al., 2004).

However, not all children with a genetic or neurological susceptibility will become depressed, so research into psychosocial risk factors is crucial in considering early intervention and prevention (Cicchetti & Toth, 1998). The majority of such research in youth focuses on late adolescence and early adulthood (e.g., Abela, Brozina, & Seligman, 2004; Lakdawalla & Hankin, 2008) or early childhood (e.g., Najman et al., 2005). Relatively few studies examine the crucial early adolescent period in which prodromal depressive symptoms are often first exhibited, leading to chronic and recurrent depression throughout the lifespan if untreated (see Kovacs, 1996a; Roberts & Bishop, 2005; Roberts, 1999).

Parent Depression

Depression in parents has been well-established as a risk factor for depression in offspring, with up to a six-fold increased risk (e.g., Downey & Coyne, 1990; Garber, Keiley, & Martin, 2002). Genetic studies suggest that part of the risk may be hereditary, however

genetic explanations only partly explain the link, with twin studies also indicating a strong environmental component (Sullivan, Neale, & Kendler, 2000; Sussman, 2005; Thapar & McGuffin, 1994). Adolescents with depressed parents experience depressive episodes that are more frequent, longer in duration, had an earlier onset, and were more likely to have comorbid diagnoses than the offspring of healthy parents (Beardslee, Keller, Lavori, Staley, & Sacks, 1993). Maternal depression has been found to confer a particular increase in risk of depression among offspring (see Garber et al., 2002; Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Weissman & Jensen, 2002).

Children of depressed mothers are exposed to more stressful events and chronic daily stressors within the family than are children of non-depressed mothers (Hammen, 1991a). Increased stressors are suggested to result in higher levels of depression in children and parents alike, in turn potentially causing more interpersonal stress within the family (Roberts, 1999). Youth with depressed mothers have been shown to experience depressive symptoms in a similar timeframe to maternal episodes, supporting this shared-environment effect (Abela, Skitch, Adams, & Hankin, 2006; Hammen, 1991a). Assortative mating, wherein depressed persons tend to marry partners who also experience psychological difficulties, may increase the likelihood of psychopathology in both parents, thereby increasing the chances of depression in offspring (Feldman, Rubenstein, & Rubin, 1988; Wallerstein & Corbin, 1991). Children of divorced parents exhibit increased levels of depression and slower rates of recovery, such that the problems of depressed parents may directly influence offspring through such stressful life events (Brennan, Hammen, Katz, & Le Brocque, 2002; Cicchetti & Toth, 1998). Disrupted parent-child relationships (e.g., Harnish, Dodge, & Valente, 1995), marital conflict (e.g., Zahn-Waxler, Iannotti, Cummings, & Denham, 1990) and family stress (e.g., Hammen, Burge, & Adrian, 1991) have been shown to mediate the relationship between maternal depression and child outcomes.

Parenting

Substantial research suggests that the offspring of depressed parents may be at increased risk of depression due to the negative interactions they experience with their parents (e.g., Downey & Coyne, 1990), with depressed parents theorised to demonstrate altered parenting behaviours that may increase the risk of difficulties among children (McLeod, Weisz, & Wood, 2007; Rapee, 1997). Negative parenting has been proposed to mediate the relationship between parental depression and psychopathology in offspring (Garber, 2005). Parents with depressive symptoms such as anhedonia, sadness and lethargy (APA, 2000) may have a reduced ability to attend to the needs and behaviour of their children, and to respond to their child's needs in a contingent, affectionate and emotionally sensitive manner (Lovejoy et al., 2000). If depressed parents are more withdrawn, less positive and less warm than non-depressed parents, it has been suggested that their children will learn from this in regulating their own affect and behaviour (Rapee, 1997). This rejecting, uncaring parenting style, incorporating a spectrum from acceptance and warmth to rejection and criticism (Parker, 1990), has been particularly implicated in the development of depressive symptoms in offspring, with estimates of up to a two- to three-fold higher rate of depressive disorders among the offspring of rejecting parents (Jacquez, Cole, & Searle, 2004; Patton, Coffey, Posterino, Carlin, & Wolfe, 2001).

Observational studies of parental behaviour with infants provide strong evidence for the effect of parental depression on parent-child interactions. Compellingly, Cohn and Tronick (1983) demonstrated that when non-depressed mothers portrayed simulated depression, their infants' affective self-regulation was disrupted, and the infants displayed negative affect. Observational studies with infants also reported an increased maternal focus on failures (Cole & Rehm, 1986), reduced emotional expression and engagement (Tarullo, DeMulder, Martinez, & Radke-Yarrow, 1994), less positive responses (Downey & Coyne, 1990) and disrupted infant self-regulation and negative affect following simulated maternal

depression (Cohn & Tronick, 1983). Downey and Coyne (1990) surmise from these studies that parental depression results in harsh, inconsistent parenting, which contributes to depressive symptoms in children.

In addition to research considering the parenting behaviour of depressed parents, considerable research has examined the parenting environment of depressed offspring. Initial evidence came from studies with adults, wherein depressed individuals recalled negative, rejecting and unsupportive relationships and interactions with their parents (e.g., Garber, 2005; Parker, 1979). More recently, self-report research has considered the current parenting environment of depressed children and adolescents, with considerable evidence that depressed young people report negative parental interactions (McLeod et al., 2007). Depressed youth report parents who are rejecting, psychologically unavailable and controlling (Stein et al., 2000). Maternal criticism predicts depressive symptoms in offspring after 2-8 weeks, controlling for initial symptoms (Jacquez et al., 2004). Disturbed family relationships have been noted prior to, during and after major depressive episodes in youth (Reinherz, Paradis, Giaconia, Stashwick, & Fitzmaurice, 2003). Depressed children also report more family conflict, negative affective family tone and parents who are rejecting, psychologically unavailable and controlling (Stein et al., 2000). Lower family support and greater conflict predicted adolescent depression 12 months later, controlling for initial depression (Sheeber, Hops, Alpert, Davis, & Andrews, 1997). Dietz and colleagues' (2008) very recent observational study of problem-solving interactions showed that depressed children demonstrated more negativity and less positivity, with mother-child interactions characterised by maternal disengagement and low child positivity. Mothers of depressed adolescents were shown to be more facilitating in response to adolescent depressive behaviour, suggesting that mothers of depressed adolescents may inadvertently reinforce depressive behaviours in this way (Sheeber, Hops, Andrews, Alpert, & Davis, 1998).

Despite increasing evidence of an association between rejecting parenting and depression in offspring, the majority of research has used cross-sectional, adult retrospective or infant samples (McLeod et al., 2007). Adolescence is a crucial time for the consideration of depressive risk, with changing relationships with peers and parents (Garber, 2005), developmental changes and increasing environmental stressors (Schmidt & Maras, 2001) potentially altering the parent-offspring patterns seen in other age groups. Garber (2005) calls for more prospective studies with adolescents to directly examine parenting behaviour as a potential mediator between parent- and offspring-pathology.

In summary, maternal depression in particular has been clearly established as a risk factor for depression in offspring (McLeod et al., 2007), with negative, rejecting parenting implicated in its transmission (Downey & Coyne, 1990; Garber, 2005; McLeod et al., 2007). The actual process through which rejecting parenting may lead to adolescent depressive symptoms remains unknown, although it is possible that children may develop negative self-beliefs as a mediating factor (Garber, Robinson, & Valentiner, 1997).

Personality

As a higher order intrapersonal factor, personality has been long associated with depression, with particular emphasis on neuroticism (see Shea & Yen, 2005 for a review). Neuroticism, or negative emotionality, is described as a stable tendency to perceive and experience the world as threatening and distressing (Watson & Clark, 1992; Watson, Clark, & Harkness, 1994). It is associated with heightened sensitivity to negative stimuli, vulnerability to sadness, depression, anxiety and other negative emotions, as well as negative cognitions and appraisals, low self-esteem and life dissatisfaction, and is moderately heritable (Clark, 2005; Clark, Watson, & Mineka, 1994).

Empirical evidence with adults strongly associates neuroticism with depression, both concurrently (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1993; Krueger, 1999) and prospectively (Kendler, Gatz, Gardner, & Pederse, 2006; Lakdawalla & Hankin, 2008).

Although there is a well-established link between high levels of neuroticism and depression, the actual processes through which neuroticism may confer a risk of depression remain unclear (see Shea & Yen, 2005 for a review). Preliminary evidence suggests the involvement of stressful life events and cognitive processing styles (Lakdawalla & Hankin, 2008).

Initial evidence with adolescents suggests that personality characteristics may increase the risk of depression (Kendler, Gardner, & Prescott, 2003; Markey, 2002; Santor & Rosenbluth, 2005). However, very little empirical attention has considered the role of neuroticism as a risk for depression across time in early adolescence, or to the processes through which neuroticism may influence the development of depressive symptoms (Lakdawalla & Hankin, 2008).

In summary, as a known vulnerability for mood disorders, neuroticism has been theorised to confer a risk of depressive symptoms in adolescence (e.g., Kendler et al., 2003), however the actual process through which this takes place has not been established (Lakdawalla & Hankin, 2008). According to stress-generation theory (discussed below), neuroticism has been linked with the occurrence of dependent stressful life events, as the more neurotic individual is likely to behave in such a way as to cause interpersonal problems in particular (Hammen, 1991b), such as seeking excessive reassurance and irritating their peers (Hankin & Abramson, 2001). Neuroticism is also associated with depressogenic cognitive schema, such as a tendency to interpret events as threatening and distressing (Clark et al., 1994).

Cognitive Theories

Perhaps the most extensive current theories of depression focus on cognitive factors, stemming from the work of Aaron Beck in the mid-20th century. Beck (1967; 1976) observed that depressed individuals tended to display certain cognitive tendencies, holding a negative triad of views about the self, the world and the future. Beck (1967; 1976) suggested that the tendency to interpret information in such distorted ways maintained a negative belief system

that could underlie depression. Strong evidence supports the role of cognitive schema in depression among adults (e.g., Abela & D'Allesandro, 2002) and adolescents (e.g., Haley, Fine, Marriage, Moretti, & Freeman, 1985; Hankin & Abramson, 2002).

Attributional Style

Hopelessness Theory (Abramson, Metalsky, & Alloy, 1989) specified that depressed individuals tend to attribute negative events to stable and global causes, believing that the event would lead to further negative events and that it indicated a flaw with themselves. Such individuals blame themselves for negative life events, view the causes of those events as permanent, and over-generalise specific weaknesses as applying to many areas of their life (Watts & Markham, 2005). Extensive empirical research with adults has established that a negative attributional style confers a considerable risk of depression (e.g., Abramson et al., 1989; Abramson, Seligman, & Teasdale, 1978). Negative attributions are particularly cogent in the context of such stressful life events, as discussed below under cognitive diathesis-stress theories.

A negative attributional style has also been strongly implicated in the development and maintenance of depression in adolescent studies (e.g., Hankin, Abramson, & Siler, 2001; Southall & Roberts, 2002). For example, Garber and colleagues (2002) demonstrated that after controlling for gender and maternal depression, pessimistic attributional style predicted increases in depression among adolescents. It has also been suggested than the absence of a positive attributional style may be what places the individual at risk of depression (Spence & Reinecke, 2004). The majority of research considering attributional style has examined its involvement in the diathesis-stress model, discussed below.

Rumination

A tendency to ruminate in response to low mood has also been associated with depression (Abela, Brozina, & Haigh, 2002; Nolen-Hoeksema & Morrow, 1991). Nolen-Hoeksema (1998) described a ruminative response style as a tendency to respond to low mood

by focusing passively and repetitively on one's symptoms of depression and their possible causes and consequences, without taking action to relieve those symptoms. Self-focused rumination has been shown to be particularly detrimental (Watkins & Moulds, 2005; Watkins & Teasdale, 2001), when it is focused on the causes and experience of a problem rather than on the process of solving the problem (Watkins & Baracaia, 2002). Rumination is thought to enhance the effect of depressed mood on cognitive processing, by increasing access to negative interpretations and memories (Nolen-Hoeksema, 1998). In addition, a ruminative focus on the experience, cause and consequences of depressive symptoms may prevent the individual from taking appropriate action to solve social problems (Watkins & Baracaia, 2002) or to improve their mood (Nolen-Hoeksema, 1998), also impairing executive processing (Watkins & Brown, 2002).

Rumination following a stressful event has been shown to exacerbate an initial negative emotional response, worsening the symptoms of depression (Watkins, Moberly, & Moulds, 2008). Ruminative self-focus has been associated with negative future thinking among depressed individuals, which Lavender and Watkins (2004) suggest supports the exacerbating effect of rumination on cognitive biases in depression. Rumination predicts prospective increases in depressive symptoms in adolescence (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007), while adolescents who tend to ruminate in response to stress have been shown to report increased depressive symptoms following stressors (Skitch & Abela, 2008).

Self-worth

Self-worth, or beliefs about oneself and the value that one places on the self as a person (Harter, 1990), has also been strongly associated with depression (Angold & Worthman, 1993; Costello, Swendsen, Rose, & Dierker, 2008; Lewinsohn et al., 1994). Self-worth is proposed to develop during childhood, with perceived support and acceptance from significant others being important influences (Beck, 1967; Garber et al., 1997; Harter, 1986). Controlling for initial depressive symptoms, low self-worth predicts a vulnerability to

increased depressive symptoms among young adolescents (McCarty, Vander Stoep, & McCauley, 2007). Self-worth has also been shown to both mediate and moderate the relationship between negative life events and depressive symptoms in young adults, suggesting that it acts like other cognitive vulnerabilities in influencing the effect of stressors on depression (Uhrlass & Gibb, 2007). Initial research suggests that the association between self-worth and external feedback or success (self-worth contingencies) is an important predictor of depressive symptoms in adolescence (Burwell & Shirk, 2006), emphasising the importance of external reinforcement to self-worth and subsequent depressive symptoms. At a broader level, self-worth partly mediates the relationship between gender and depression in adolescence, such that girls may have higher levels of negative self-perceptions and depression (Eberhart, Shih, Hammen, & Brennan, 2006).

Despite considerable research linking self-worth and depression in childhood, relatively little evidence exists directly measuring these factors among adolescents. Researchers have called for further consideration of the role of cognitive schema such as self-worth in the development of depressive disorders (Paunesku et al., 2008), particularly in the formative early adolescent years (Roberts, 1999).

Cognitive vulnerabilities in adolescence

Overall, research examining cognitive factors among young adolescents has lagged behind that with adults or older adolescents. Developmental theories note that cognitive functioning changes around this time, with younger children viewing themselves in concrete, physical terms while at around this age young adolescents begin to view themselves in more abstract psychological terms, including stable personal characteristics (Damon & Hart, 1982). Young adolescents are beginning to engage in abstract reasoning and formal operational thought (see Hankin, 2006). It has been suggested that this shift in thinking could be potentially negative for children at risk of an affective disorder, as a cognitive vulnerability to depression may first emerge at this age while self-beliefs are really forming (e.g., Abela,

2001; Eley, 1997; Shortt & Spence, 2006). Krueger (1999) suggests that from this age and developmental stage, negative cognitions can interact with environmental stressors to contribute to depression. A longitudinal consideration of cognitive factors in the development of early adolescent depressive symptoms would provide important insights into the causes and maintenance of depression.

Stressful Life Events

A substantial body of literature implicates the occurrence of stressful or negative life events in the onset of depression (e.g., Goodyer & Altham, 1991a, 1991b; Rutter, 2000; Silberg et al., 1999). The majority of depressed individuals report recent stressful life events, with one in five occurrences of severely negative events leading to depression (Goodyer, 1991). Of course not all individuals who experience stressful life events will become depressed, leading to research focused on the factors unique to those individuals who do develop depression relative to those who do not (Goodyer, 1991; Silberg & Rutter, 2002).

Considerable research links stressful life events with depression among children and adolescents (e.g., Reinherz et al., 1989; Silberg & Rutter, 2002), with the impact of negative events particularly marked for adolescent girls (e.g., Silberg et al., 1999). Developmentally, early adolescence is a formative period during which increases in stressful life events mirror increases in depressive symptoms, and the gender disparity between boys and girls first emerges (e.g., Angold & Worthman, 1993; Hankin & Abela, 2005; Nolen-Hoeksema, Girgus, & Seligman, 1992). Longitudinal studies have demonstrated that the occurrence of stressors precedes the initial elevation, recurrence and exacerbation of depressive symptoms among adolescents (e.g., Ge, Conger, & Elder, 2001; Goodyer, Herbert, Tamplin, & Altham, 2000). However, the mechanisms through which stressful life events contribute to depressive symptoms for some adolescents, but not others, are the subject of ongoing theoretical and empirical attention. These processes have been the focus of two strong theories, cognitive diathesis-stress theory and stress-generation theory.

Cognitive Diathesis-Stress Theory

As an extension of cognitive theories of depression, Beck (1983) proposed that depressogenic cognitive styles, in combination with congruent stressful life events, would have a greater impact on the development of depression than cognitive styles alone. This became known as the diathesis-stress model of depression, emerging from the observation that not all individuals who are exposed to negative events develop depression – cognitive vulnerabilities determine the impact of stressful life events on the development of depressive symptoms (Abramson et al., 1989; Alloy, Hartlage, & Abramson, 1988; Beck, 1987). Diathesis-stress models of depression propose that individuals with certain cognitive tendencies will interpret the occurrence of stressors in such a way as to increase the likelihood of depressive symptoms (Bohon, Stice, Burton, Fudell, & Nolen-Hoeksema, 2008).

Extensive research supports an overall cognitive diathesis-stress model with adults, in which an overall negative attributional style interacts with the occurrence of a variety of stressful life events (see Abramson et al., 1999; Ingram, Miranda, & Segal, 1998 for reviews). Evidence also supports the cognitive diathesis-stress model with children and adolescents (e.g., Hankin et al., 2001; Nolen-Hoeksema et al., 1992). Longitudinal research with adolescents suggests that a negative attributional style interacts with the occurrence of stressful life events to predict later depression (e.g., Abela, 2001; Dixon & Ahrens, 1992; Weir & Jose, 2008). Very recent research has examined age and gender differences in the diathesis-stress pathway, suggesting that the diathesis-stress relationship is consistent across boys and girls and early- and middle-adolescence (Hankin, 2008). For example, an adolescent who tends to interpret academic failures as a sign of personal weakness (e.g,. “I’m stupid”), as commonly occurring to them, likely to happen in the future and to have extensive consequences (e.g., “I’ll never make it to university”) will become more depressed following such a failure. An adolescent with a depressogenic attributional style might perceive a friend as failing to acknowledge them, and assume that it is a personal slight, reflecting flaws in his

or herself and likely to have broad, ongoing negative consequences, and so experience low mood as a result.

In summary, diathesis-stress studies have established that individuals with negative cognitive styles are more likely to become depressed following stressful life events, as they interpret the event as a personal failing, likely to happen again and to affect them badly (Abramson et al., 1989; Beck, 1987). Evidence supports this theory in adolescence, establishing that a negative cognitive style interacts with stressors to predict later depression (e.g., Abela, 2001), however relatively few studies consider early adolescence and prodromal depressive symptoms in this age range (Roberts, 1999).

Stress-Generation Theory

Stress-generation theories proposed that the stressor-depression relationship is not uni-directional, but a bi-directional process (Hammen, 1991b). Stress-generation theory proposes that some individuals, based on trait (personality) or state (depressive affect) factors, will behave in such a way as to partly cause stressful events, which then lead to further increases in depression in a perpetuating cycle, maintaining depressive symptoms (Hammen, 1991b). These dependent stressful life events could include relationship difficulties, job losses or academic failures (Hammen, 1991b; Rudolph & Hammen, 1999). Cross-sectional evidence supports an increased report of dependent stressors among depressed adults (e.g., Rudolph & Hammen, 1999). However, such studies have been criticised for the simultaneous reporting of depressive symptoms and stressors (e.g., Harkness & Luther, 2001), given that depressed individuals have been shown to report more negative events than non-depressed controls (Monroe & Simons, 1991). Longitudinal research strengthens support for the stress-generation theory with adults, establishing that initial depression is associated with the generation of later dependent stressful life events (Waaktaar, Borge, Fundingsrud, Christie, & Torgersen, 2004; Wingate & Joiner, 2004). Stress-generation theory also suggests that events independent of the individual's actions, such as a death in the family or natural disasters,

should be unrelated to initial depressive symptoms (Hammen, 1991b), with some empirical support for this notion (e.g., Daley, Hammen, Burge, & Davila, 1997; Rudolph et al., 2000).

Personality has also been implicated in stress-generation theory, as a precursor to the occurrence of dependent stressors and subsequent depressive symptoms (Hammen, 1991b). Neuroticism has been associated with dependent negative life events in adults on a cross-sectional basis (Kendler et al., 1993), with more recent prospective studies showing that negative emotionality predicts the later occurrence of negative life events in adults (Kendler et al., 2003). However, most research has focused on depressive symptoms as predicting stress-generation, rather than personality, and to date, no research has been reported in this area with young adolescents.

Among adolescents, recent research has demonstrated that initial depressive symptoms predict later increases in stressors, which are associated with increases in depressive symptoms (e.g., Cole, Nolen-Hoeksema, Girgus, & Paul, 2006; Hankin, Roesch, & Mermelstein, 2004). However, again, relatively little research has considered early adolescence and emerging depressive symptoms.

The Elaborated Cognitive Vulnerability-Transactional Stress Theory

Hankin and Abramson (2001) proposed the elaborated cognitive vulnerability-transactional stress theory, including pre-existing vulnerabilities (genetic, personality, environmental adversity), which influence both cognitive vulnerabilities (negative attributional style, dysfunctional attitudes, ruminative tendency) and the occurrence of stressful life events (both dependent and independent). They propose that these stressors cause an initial response of negative affect, which may interact with a depressive cognitive style to predict increases in depressive symptoms. These symptoms may then lead to the occurrence of further interpersonal dependent stressful events (such as seeking excessive reassurance and being rejected). Although Hankin and Abramson's (2001) model considers possible variations

in the developmental pathways for boys and girls, gender differences are not a focus of this thesis, which instead focuses on overall models of risk for young adolescents.

Furthermore, the elaborated cognitive vulnerability-transactional stress theory considers multiple cognitive factors as forming an overall vulnerable cognitive style (Hankin & Abramson, 2001). Hankin and Abramson (2001) propose that dysfunctional attitudes, and/or a tendency to make negative attributions provides the individual with negative cognitive content, while rumination then facilitates the activation of this content – for example, the individual interprets a failure as a reflection of personal flaws, likely to happen repeatedly and to significantly affect their future, and dwells on this information repeatedly, along with ruminating about how badly they feel. Evidence supports this broader cognitive vulnerability, with rumination shown to interact with a negative inferential style in predicting depression (Alloy et al., 2000) and also to mediate the association between cognitive risk and depression (Spasojevic & Alloy, 2001). This is consistent with Watkins and colleagues' (2004; 2008) suggestions that it is not only biases toward negative information, but also a tendency to ruminate and to process that information negatively, that increases emotional reactivity to stressful events, and exacerbates the effect of negative cognitive content.

Preliminary research with adults has provided initial support for the elaborated cognitive vulnerability-transactional stress model, with negative emotionality shown to predict increases in negative life events and depressive symptoms across time (Lakdawalla & Hankin, 2008). Cognitive vulnerability interacted with negative life events to predict increases in depression, with cognition partly mediating the association between negative emotionality and increases in depressive symptoms (Lakdawalla & Hankin, 2008). Although this study supports this model in adults, it cannot be assumed that these risk factors operate in the same way in adolescents, when personality, cognitive and self-worth factors are still emerging, and social and life pressures are very different. Hankin (2006) reported a study of neuroticism, stressful life events and depression across time in adolescence, where initial levels of

neuroticism predicted the occurrence of additional stressors over later follow up. These stressors mediated the prospective association between neuroticism and increases in depression over time (Hankin, 2006). Therefore, this study suggests that personality and stressful life events may be important in the development of depressive symptoms in adolescence, however to date, the elaborated cognitive vulnerability-transactional stress model has not been extensively tested with adolescents.

Summary and Purpose of This Thesis

In summary, theories of depression propose an interaction between individual and environmental factors, which contribute to the development of depressive symptoms. Parent depression is proposed to confer a risk of depression in offspring, potentially through altered parenting behaviour and particularly through rejecting, less caring parenting. Such negative parenting may lead to negative self-beliefs in the adolescent, who develops a low sense of self-worth, placing them at risk of depression. More broadly, adolescents high on neuroticism are proposed to be vulnerable to depression, partly due to their negative cognitive style, and possibly also due to their tendency to generate dependent negative life events. Given that the occurrence of stressful life events is strongly associated with depressive episodes, but that not all who experience stressors become depressed, the processes through which stressors are related to depression is of particular importance. Cognitive diathesis-stress models propose that individuals with depressogenic cognitive tendencies will become depressed following stressful life events, while stress-generation theories propose that individuals at risk of depression may cause stressful life events.

Measurement bias has been noted as a flaw in previous, cross-sectional studies of risk factors and depression, because a depressed person is likely to provide a biased report (e.g., Ge et al., 1994). Studies examining the developmental trajectory of depression in early adolescence (12-13 years) are relatively rare (Roberts, 1999). Specifically, there is little research testing conceptual models for the development of depression across time in this age

group. Previous researchers have called for longitudinal analyses of depression in adolescence, particularly focusing on temporal mechanisms, with the hope of capturing risk factors specific to those individuals who develop depressive symptoms (e.g., Krueger, 1999; Roberts, 1999). Such research should begin in very early adolescence, a developmentally and clinically significant period, when children have an increased understanding of their own thinking processes and self-schemas are more stable, but when the increase in the prevalence of depression is only just beginning (Hammen, 1991b).

Therefore, the aim of the present research is to consider theoretically-derived models of depressive risk across time in early adolescence, building on previous theories of depressive risk, with the aim of identifying early risk factors and the processes through which they may contribute to the experience of depressive symptoms in adolescence. Heeding the call of previous researchers, the current work aimed to propose and test conceptual pathways potentially contributing to the development and maintenance of depressive symptoms over time. With important implications for the detection of children at risk, the early adolescent age range was targeted, providing initial prospective tests of conceptual pathways in this critical developmental stage. A range of theoretically important, yet previously rarely considered, risk factors were considered. Drawing together evidence from familial, personality, cognitive and life stress theories, three models were designed to examine the onset and maintenance of depressive symptoms in early adolescence.

The study reported in chapter two was designed to examine possible links between maternal and young adolescent depressive symptoms, and to consider the processes through which this vulnerability may be conferred, including perceived rejecting parenting and self-worth. In chapter three, elements of the elaborated cognitive vulnerability-transactional stress (Hankin & Abramson, 2001) and stress-generation theories (Hammen, 1991b) were considered, examining the possible mechanisms through which neuroticism may act as a risk for depression. Finally, chapter four examined an extension of previous theories considering

the pathways between stressful life events and depression and the possible convergence between cognitive diathesis-stress and stress-generation models (Abramson et al., 1989; Hammen, 1991b; Hankin & Abramson, 2001).

The results of these studies are presented in three self-contained papers, resulting in an inevitable degree of overlap.

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CHAPTER 2

PARENTING IN ADOLESCENT DEPRESSION:

THE MEDIATING ROLE OF SELF-WORTH IN A PROSPECTIVE TEST

Abstract

It is well recognised that depression in parents is associated with a risk for depression in offspring. The effect of parental depression on parenting behaviour has been established in research with infants, but rarely considered with adolescents. Previous research suggests that depressed parents may be perceived as more rejecting, contributing to a lower sense of self-worth among adolescents, placing them at risk of depression. A model was proposed to account for the association between maternal and early adolescent depression (mean age=12.8 years) 6 months later, considering the mediation of perceived rejecting parenting and its impact on adolescent self-worth. This model was partly supported, although mother-reported maternal depression was not associated with adolescent-reported parenting or depressive symptoms. Perceived maternal rejection was associated with higher concurrent depressive symptoms, partly through the mediation of lower self-worth, although only initial depression was associated with depressive symptoms 6 months later.

Parent depression has been established as a significant risk factor for the experience of depression among children and adolescents (e.g., Downey & Coyne, 1990; Garber, Keiley, & Martin, 2002; Weissman & Jensen, 2002), with some estimates suggesting as much as a six-fold increased risk (see Roberts, 1999, for a review). Previous research has considered a range of pathways through which this risk may be conferred, largely focusing on genetic factors (see Sullivan, Neale, & Kendler, 2000), shared environment (e.g., Thapar & McGuffin, 1994) and a combination of genetic vulnerability and increased environmental stressors in at-risk families (e.g., Thapar, Harold, & McGuffin, 1998). Many researchers have proposed that the children of depressed parents may be at increased risk of depression due to the negative interactions they experience with their parents, with depressed parents demonstrating altered parenting behaviours that may increase the risk of difficulties among children (see McLeod, Weisz, & Wood, 2007; Rapee, 1997 for reviews).

Although some research has examined the relationship between offspring depression and paternal relationships (e.g., Brennan, Hammen, Katz, & Le Brocque, 2002; Patel, 2003), the vast majority of research has focussed on the relationships with mothers (see Garber et al., 2002; Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Weissman & Jensen, 2002). Strong evidence for a link between maternal depression and parent-child interactions has been found in observational studies with infants, including an increased focus on failures (Cole & Rehm, 1986), reduced emotional expression and engagement (Tarullo, DeMulder, Martinez, & Radke-Yarrow, 1994), less positive responses (Downey & Coyne, 1990) and disrupted infant self-regulation and negative affect following simulated maternal depression (Cohn & Tronick, 1983). A considerable body of research demonstrates that depressed adolescents report negative parental interactions (see Garber, 2005; McLeod et al., 2007 for reviews). Mothers of depressed offspring, regardless of their own levels of depression, are more hostile (Dadds,

Sanders, Morrison, & Regbetz, 1992) and less warm and effective in their parenting (Ge, Best, Conger, & Simons, 1996). Depressed youth report parents who are rejecting, psychologically unavailable and controlling (Stein et al., 2000). Disturbed family relationships have been noted prior to, during and after major depressive episodes in youth (Reinherz, Paradis, Giaconia, Stashwick, & Fitzmaurice, 2003). Lower family support predicted adolescent depression 12 months later, controlling for initial depression (Sheeber, Hops, Alpert, Davis, & Andrews, 1997). In a summary of the literature, Garber (2005) suggests that negative parenting may mediate the relationship between parental depression and psychopathology in offspring, but cites mixed evidence, largely focusing on overt parental abuse (e.g., Bifulco et al., 2002), punitive parenting (e.g., Leionen, Solantaus, & Punamaki, 2003) and children's disruptive behaviours (Fendrich, Warner, & Weissman, 1990), calling for more prospective studies to consider parenting as a mediator between parent- and offspring-pathology.

Despite a strong body of literature with young children, relatively little research has considered mothers' interactions with their adolescents. Many of these studies also collapse developmentally diverse periods of time (e.g., childhood up to the age of 16 years) (Rapee, 1997), contrary to research establishing the importance of parenting at specific ages (e.g., Downey & Coyne, 1990). Developmentally, early adolescence is a critical time to study depression, with extensive research establishing a substantial increase in rates of depression beginning at around 13 years of age (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Fleming & Offord, 1990). In order to capture risk factors preceding the initial increase in depressive symptoms, research needs to examine children across this age range, with the hope of identifying particular risk factors specific to those children who develop depressive symptoms (Roberts, 1999). Given the known changes in adolescent social relationships, when

peer relationships take on increased importance and parental influence is known to reduce (Garber, 2005), it is important to consider adolescents' relationships with their parents separately from childhood studies of parenting and depression.

Much of the previous research examining parenting and depression has been limited by cross-sectional, self-report and retrospective methodology (McLeod et al., 2007). The current affective state of the reporter may influence their report, such that offspring-reported perceived parenting may not accurately reflect actual parenting behaviour (Garber, 2005). The majority of research has assessed perceived parenting, with the suggestion that the adolescent's perceptions of the family environment may be as predictive of depressive symptoms as the actual behaviour of family members (Garber, 2005). Perception has been found to be a critical element of the child's experience (Jacquez, Cole, & Searle, 2004), possibly mediating the link between parenting and child depression (Garber, 2005). Parker (1983) argues that the child's perception of parenting is more important than actual childrearing behaviours in the development of later psychopathology.

A rare observational study examined interactions between depressed and non-depressed adolescents and their parents, and found that mothers of depressed adolescents were more facilitating in response to adolescent depressive behaviour (Sheeber, Hops, Andrews, Alpert, & Davis, 1998). Sheeber and colleagues (1998) suggest that mothers of depressed adolescents may inadvertently reinforce depressive behaviours. However, other studies have reported no differences in parenting behaviour between depressed and non-depressed young adolescents (Hamilton, Asarnow, & Tompson, 1999).

In two reviews of research on parenting and youth psychopathology, Rapee (1997) and McLeod and colleagues (2007) each suggested that among a range of negative parenting behaviours, parental rejection may be specifically associated with depression in offspring.

The concept of parental rejection, or low parental care, has been described in terms of negative or hostile feelings and conveyed rejection and criticism by the parent toward the child (Parker, 1990; Rapee, 1997). Parental rejection has been strongly associated with depression in offspring (Asarnow, Tompson, Woo, & Cantwell, 2001; Jacquez et al., 2004; Puig-Antich et al., 1985a, 1985b). Previous research has shown an association between rejecting parenting and a two- to three-fold higher rate of depressive disorder in youth (Patton, Coffey, Posterino, Carlin, & Wolfe, 2001). It seems conceptually possible that depressed parents may be more rejecting. Given the evidence for biases in attention and attributions among adults with depression (e.g., Beck, 1987; Siegle, Ingram, & Matt, 2002), it is possible that depressed parents will perceive fewer positive characteristics in their adolescents and may be less likely to proffer praise and positive attention to their children, behaving in a more rejecting fashion. Lovejoy and colleagues (2000) suggested that depressive symptoms in mothers would be likely to affect their ability to attend to the needs and behaviour of their children and to respond in a contingent, affectionate and emotionally sensitive manner.

Several researchers have proposed that parental rejection may contribute to the development of depression by undermining the development of self-worth (e.g., Garber & Flynn, 1998; Garber, Robinson, & Valentiner, 1997; McLeod et al., 2007). Self-worth is defined as the value that one places on the self as a person (Harter, 1990), and has been strongly associated with depression in adolescents (Costello, Swendsen, Rose, & Dierker, 2008; Lewinsohn et al., 1994). Children's perceptions of support and acceptance from significant others have been implicated in the development of self-worth (Harter, 1986). Perceived rejecting parenting may influence self-worth through the internalisation of parental feedback, with the adolescent who perceives more parental rejection, less positivity and more

withdrawn parenting coming to believe that positive feedback is difficult to obtain and independent of their actions (Rapee, 1997). Beck (1967) proposed that children with critical or rejecting parents would be prone to developing negative self-schemas and to be vulnerable to depression. Bowlby's (1977) descriptions of ideal caregiving relationships suggest that parental rejection and control would lead a child to feel unloved and incompetent, which may contribute to a negative self-concept (Zemore & Rinholm, 1989). Garber and colleagues (1997, 1998) proposed that negative, rejecting parenting could be associated with the development of a negative self-image in children, leading to increased stress and conflict, impaired social skills, or heightened dependency, and placing the child at risk of depression.

Initial evidence links rejecting parenting with self-worth in children (see Garber, 2005, for a review). A high correspondence was demonstrated between depressed mothers and their toddlers in the affective tone of attributions and statements about the self, suggesting an increased vulnerability among these children for negative self-attributions (Radke-Yarrow, Belmont, Nottelmann, & Bottomly, 1990). Insecure attachment predicted prospective increases in depressive symptoms in college students, mediated by dysfunctional attitudes and low self-esteem (Hankin, Kassel, & Abela, 2005). In a long term prospective study, mothers' reports of parental rejection and control at 5 years of age predicted children's self-criticism at age 12 (Koestner, Zuroff, & Powers, 1991). Low maternal acceptance has been shown to predict children's subsequent low self-worth (Garber & Flynn, 2001). The clearest support came from Garber and colleagues (1997). Among mothers with a history of psychopathology, parental acceptance/rejection was cross-sectionally associated with depressive symptoms in young adolescents (mean age 11.9), a relationship partly mediated by the child's self-worth (Garber et al., 1997).

The aims of this study were to examine links between maternal and young adolescent depressive symptoms and consider the possible mediating roles of perceived rejecting parenting and self-worth (see Figure 1). It was expected that adolescents would perceive mothers high on depressive symptoms as more rejecting (or less caring) than less depressed mothers. In turn, adolescents' perceptions of maternal rejection were expected to predict lower overall self-worth, which in turn would predict depressive symptoms 6 months later, after controlling for initial symptoms of depression.

Method

Participants

Participants were a community sample of 209 seventh-grade students (122 boys, 87 girls) and their mothers, from high schools around Sydney, Australia, with a mean age of 12.8 years (*SD*=4 months) at Time 1. Sixty-nine percent of participants were from an Anglo-Australian background, 7% from Asian backgrounds, 25% European and the remainder from North America, Oceania and the Middle East. The median family income was $75-100 000, comparable with the average Australian family income (ABS, 2007). Students without consent were not further questioned, so it is not possible to estimate reasons for their non-participation. There were no significant differences on initial depression or on child gender, ethnicity, parental income or marital status between those who participated both times, and those who only participated at Time 1 (all *p*s>.05). Students were included from co-educational, boys' and girls' independent and Catholic schools.

Measures

The Centre for Epidemiological Studies Depression Scale (CES-D, Radloff, 1977) is a commonly used self-report scale. Respondents indicate how often they experienced symptoms

of depression over the past week, on a 4-point scale ranging from 0 (rarely or none of the time) to 3 (most or all of the time). The CES-D contains 20 statements, and has been standardised for high school populations, showing good psychometric properties among adolescents (Chabrol, Montovany, Chouicha, & Duconge, 2002; Radloff, 1991). Internal consistency in this sample was very good (Time 1 α=.85; Time 2 α=.82), and comparable with that of the full CES-D among junior high school students (α=.85, Radloff, 1991).

The Self-Perception Profile for Adolescents (SPPA, Harter, 1988) assesses perceived competence in various domains (scholastic/academic, social, athletic, physical appearance and behavioural conduct, plus three additional domains – job competence, romantic appeal and close friendships), and global self-worth. Adolescents report the extent to which they identify with one of two descriptions (for example, "Some teenagers are very happy being the way they are BUT Other teenagers wish they were different"), and items are rated on a 4-point Likert scale from 1="really true" for the negative descriptor, to 4="really true" for the positive. The self-worth subscale, used here, is calculated as the mean of the relevant items. The SPPA demonstrates good psychometric properties (Harter, 1988; Winters, Myers, & Proud, 2002). In the present sample, internal consistency for the self-worth subscale was assessed using mean inter-item correlations, as it contained fewer than 10 items (Briggs & Cheek, 1986), and was good (mean inter-item correlation =.60).

The Parental Bonding Instrument, Brief-Current form (PBI-BC, Klimidis, Minas, & Ata, 1992) was used to assess parenting style from the perspective of the adolescent, including two scales – 'care/rejection' and 'overprotection or control'. The care/rejection scale is the sum of two "care" items less the sum of two "rejection" items, and so will be high if the parent is perceived as more caring, and low if rejecting. Although the adolescent-report nature of this measure means that it cannot be assumed to be an accurate reflection of actual

parenting behaviour, scores on the PBI have been shown to correlate between pairs of twins (Mackinnon, Henderson, & Andrews, 1991; Parker, 1986), with interview-derived parent ratings (Parker, 1990) and sibling reports (Parker, 1983), which suggests that the PBI may indeed reflect actual childrearing behaviour (Rapee, 1997). The PBI-BC has strong psychometric properties (Klimidis et al., 1992), in the current sample internal consistency was again assessed using the mean inter-item correlation (Briggs & Cheek, 1986), and was very good (mean inter-item correlation=.39).

The Depression-Anxiety-Stress Scale - 21 (DASS, Lovibond & Lovibond, 1995), a 21-item questionnaire assessing symptoms of depression, anxiety and stress in adults, was used to assess the parents' own level of depressive symptoms in general. Factor analytic studies of the DASS report high reliability (depression subscale α=0.91), as well as good discriminant and concurrent validity (Antony, Bieling, Cox, Enns, & Swinson, 1998; Lovibond & Lovibond, 1995). Internal consistency in this sample was very good (α=.92).

Procedure

All procedures were approved by Macquarie University Ethics Committee and the Catholic Schools Office Parramatta Diocese. Students with parental consent completed surveys at school in group settings. Surveys were presented when adolescents were in 7th grade, and again 6 months later. To maximise sensitivity and specificity, a cut-off score of 24 on the CES-D was used to determine adolescents who may be at risk of depression (see Roberts, Lewinsohn, & Seeley, 1991). Parents and the school counsellor of students in this range were informed, and the families were provided with referral information.

Results

Descriptive Statistics

In a community sample, the skewness toward low depressive symptoms was to be expected, but was at a moderate level (all skewness values <3, Kline, 1998). It is important to consider whether the non-normality evident in the data affects the model and path significances. Bootstrapping (with 500 samples and 95% bias-corrected confidence intervals), which requires only that the data come from a random and large sample, was used to examine the model conservatively while addressing the deviation from normality. Given that in all tested models, bootstrapping provided the same pattern of results and path significances, it was apparent that the non-normal distributions did not markedly affect the conclusions that could be drawn from the path analyses. On this basis, maximum likelihood estimations were used for the final analyses (cf. Chou & Bentler, 1995).

Table 1.
Descriptive Statistics and Bivariate Correlations

Variable	CES-D – T1	DASS Depression – T1	PBI Care – T1	SPPA Self-worth – T1	CES-D – T2
CES-D-T1	-				
DASS Depression – T1	.01	-			
PBI Care-T1	-.43***	.09	-		
SPPA Self-worth-T1	-.61***	-.04	.43***	-	
CES-D-T2	.52***	.10	-.24***	-.39***	-
Mean	12.65	6.26	2.87	3.27	10.69
SD	8.89	7.61	1.47	.67	7.64
Minimum	0	0	-4.00	1.00	0
Maximum	56	40	4.00	4.00	34
Kurtosis (SD)	3.50 (.34)	7.56 (.34)	2.15 (.34)	.47 (.36)	.62 (.36)
Skewness (SD)	1.47 (.17)	2.40 (.17)	-1.48 (.17)	.89 (.18)	1.06 (.18)

Note. N=209. *** Correlations significant at $p<0.01$ (2-tailed Pearson's correlation). CES-D – T1 = Centre for Epidemiological Studies Depression Scale, Time 1. DASS Depression – T1 = Depression, Anxiety, Stress Scale, mother depression score Time 1. PBI Care – T1 = Parental Bonding Instrument (Brief, Current form), Care-Rejection Subscale (High = high care, low = high rejection), Time 1. SPPA Self-Worth – T1 = Self-Perception Profile for Adolescents, Self-Worth subscale, Time 1. CES-D – T2 = Centre for Epidemiological Studies Depression Scale, Time 2.

Correlations (see Table 1) were strong between the child-reported depressive symptoms Time 1, concurrent perceived parental care, self-worth and Time 2 child depressive symptoms ($p<.001$), but not significant between mother depressive symptoms and any of the child-reported factors.

Path Analyses

Given that there were multiple paths in the hypothesised model, path analysis was used to test the direct and indirect effects of each factor upon each outcome simultaneously, using Analysis of Moment Structures (AMOS) 16.0 (Arbuckle, 2007). This provides a conservative test of the hypothesised model, as testing each relationship separately in regressions would not account for the variance explained by the other relationships.

The standardised regression weights of both direct and indirect paths were of particular interest, given the conceptual nature of the relationships being tested. The overall model fit was considered using established goodness-of-fit indices. The comparative fit index (CFI, Bentler, 1990), relative fit index (RFI, Bollen, 1986), incremental fit index (IFI, Bollen, 1989) and the Tucker-Lewis Index (TLI, Tucker & Lewis, 1973) were examined, with cut-off values approaching .95 indicating a good fit to the model (Hu & Bentler, 1999). Additionally, to account for model complexity, the root mean square error of approximation (RMSEA, Steiger, 1990) was also examined, with its 90% confidence interval (Browne & Cudeck, 1993), where values of 0.06 or less indicate good fit (Hu & Bentler, 1999).

Model testing

The first hypothesised model (Figure 1) was designed to examine the paths from parental depression at Time 1 to concurrent child depression, considering perceived parenting and child self worth. This model replicated the study of Garber and colleagues (1997), in considering cross-sectional relationships between maternal acceptance/rejection and young adolescents' depressive symptoms, along with self-worth as a proposed mediator, and extended it by considering the possible link between maternal depressive symptoms and perceived rejecting parenting. This model was tested first, followed by the longitudinal model including Time 2 child depression as outcome, and the "trimmed", final model (Figure 2). Finally, bootstrapping was conducted to estimate the significance of the indirect paths.

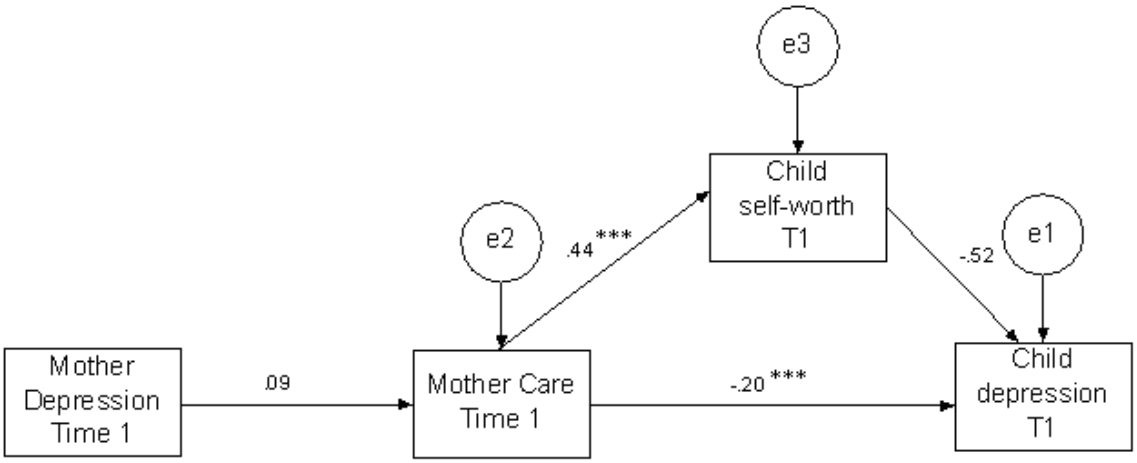

Figure 1. Cross-sectional, hypothesised model
*** Paths significant at $p<0.001$

As seen in Table 2, the hypothesised model provided a good fit for the data, with significant regression weights for all but one path (Figure 1). The path from parents' depressive symptoms to perceived parental care was not significant ($p=.216$). A partial

mediation was evident between perceived parental care and child depressive symptoms, with low care (or high rejection) associated with higher depressive symptoms (p<.001), and also with low self worth (p<.001). Low self worth, in turn, was associated with high depressive symptoms (p<.001). A direct path between parents' depressive symptoms and child depressive symptoms was also tested and was not significant in a model including perceived parenting and self-worth (p=.813), so it was excluded from the presented model.

Table 2.
Goodness-Of-Fit Indices

Model	χ^2/df	CFI	TLI	RFI	IFI	RMSEA	RMSEA (90% CI)
Model 1 (hypothesised, cross-sectional)	.769	1.000	1.018	.944	1.003	.000	.000-.127
Model 2 (hypothesised, longitudinal)	1.073	.999	.994	.918	.999	.019	.000-.120
Model 3	1.067	.999	.996	.944	.999	.018	.000-.149

Note. CFI= Comparative Fit Index, TLI= Tucker-Lewis Index, RFI= Relative Fit Index, IFI= Incremental Fit Index, RMSEA= Root Mean Square Error of Approximation and 90% confidence interval.

Secondly, the hypothesised model was extended to include longitudinal relationships, with all cross-sectional paths still included and initial depressive symptoms controlled (Figure 2). This model showed a close fit for the data (see Table 2). The paths from perceived parental care and self-worth to Time 1 child depressive symptoms were significant with a partial mediation evident ($p<.001$). However, longitudinal relationships between these factors and Time 2 child depressive symptoms were not apparent ($p>.05$) in a model including Time 1 child depressive symptoms. A direct path from Time 1 parents' depressive symptoms to child depressive symptoms at Time 2 was also tested, with no suggestion of a relationship evident ($p=.211$). No relationship was evident at a bivariate level, in Models 1 or 2 between parents' Time 1 depressive symptoms and child depressive symptoms (at Time 1 or 2) or perceived parental care, while significant paths were evident between perceived parental care, self-worth and Time 1 child depressive symptoms. For this reason, parents' depressive symptoms were dropped from the model.

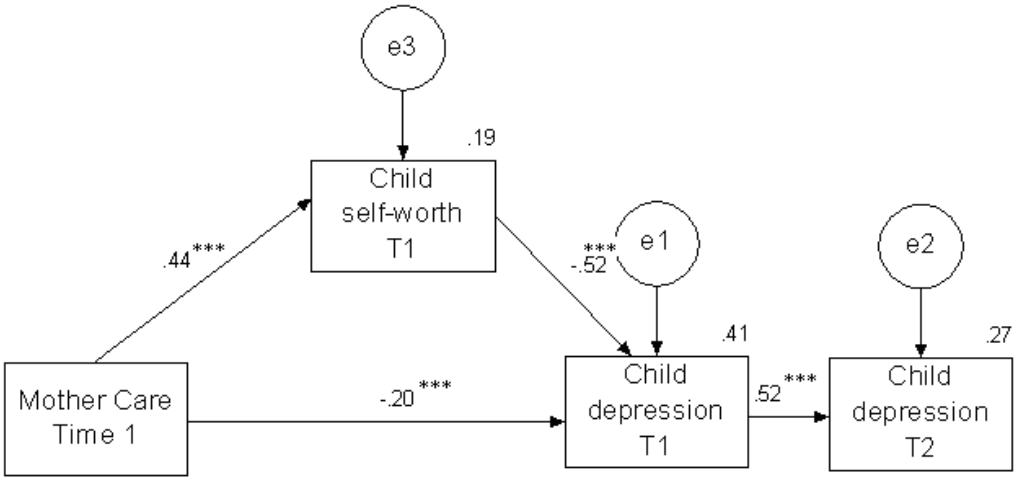

Figure 2. Trimmed longitudinal model
*** Paths significant at $p<0.001$.

Given that no longitudinal relationships were evident between perceived parenting, self-worth and later depressive symptoms, in a model controlling for initial depressive symptoms, these longitudinal paths were dropped in Model 3 (see Figure 2). Initial depressive symptoms were a good predictor of depressive symptoms 6 months later. The total proportion of the variance in Time 1 depressive symptoms explained by this model was 41%, and of Time 2 depressive symptoms was 27%.

Finally, the significance of the indirect paths was assessed in AMOS, using bootstrapping tests of indirect effects with two-tailed significance levels (using 500 samples and 95% bias-corrected confidence intervals). The indirect effect of perceived parental care on depressive symptoms at Time 1, via self-worth, was significant ($p<.01$), as was its effect on depressive symptoms at Time 2 via self-worth and initial depressive symptoms ($p<.01$).

Discussion

Perceived rejecting parenting by one's mother appears to be strongly linked with a young adolescent's depressive symptoms, both directly and through the partial mediation of lower levels of self-worth. However, perceived parenting and self-worth were not linked with changes in depressive symptoms after 6 months in this community sample, with Time 2 depressive symptoms predicted only by initial symptoms. Unexpectedly, maternal depressive symptoms were not associated with perceived rejecting parenting, nor with adolescent depressive symptoms.

Although perceived parenting and self-worth did not predict changes in depression across time, these factors may be important in the maintenance of adolescent depression, explaining 41% of the variance in concurrent symptoms. It appears that perceived rejecting parenting is a vital part of the adolescent's internal experience, associated with lower self-worth or negative self-beliefs, and increased depressive symptoms. It is possible that perceived maternal rejection and criticism (whether or not this is real) would lead an adolescent to develop and reinforce negative views of his or herself, as could a lack of encouragement, praise or warmth. The adolescent may come to believe that praise is difficult to obtain and independent of their actions, developing difficulties in regulating their own emotions and behaviour (Rapee, 1997). It might also be that adolescents who perceive less support from their mothers would be less likely to seek assistance with difficulties, or to seek encouragement and reassurance, possibly missing the opportunity to bolster their self-worth. Such negative self-beliefs or low self-worth would leave an adolescent vulnerable to depression (Costello et al., 2008; Lewinsohn et al., 1994). Accordingly, interventions should consider encouraging family communication and supportive parenting, as well as addressing the cognitive and interpretive biases the adolescent may be experiencing.

The lack of longitudinal relationships is interesting. It is possible that *earlier* childrearing behaviour may have influenced the child's vulnerability to depression over the lifetime. It may be that the causal impact of rejecting parenting on self-worth and depressive symptoms began much earlier in development, as evidenced by numerous studies considering parenting in early childhood (see Lovejoy et al., 2000). By adolescence, these factors may form part of the child's experience and may have already influenced a propensity to depressive symptoms (Najman et al., 2005), such that cross-sectional relationships are seen but longitudinal paths are not. Testing this possibility would require observational studies across childhood.

Given the cross-sectional relationship supported here, in which causation cannot be determined, future research should consider the possibility that the relationship between youth depressive symptoms and parenting in adolescence is bi-directional, with the adolescent's depressive behaviour possibly eliciting negative parental responses. For example, when the infants of depressed mothers interact with non-depressed strangers, they have been observed to elicit depression-like behaviour in the strangers (Field et al., 1988). A very recent observational study of problem-solving interactions showed that depressed children demonstrated more negativity and less positivity, with mother-child interactions characterised by maternal disengagement and low child positivity (Dietz et al., 2008). The bi-directional effects of such interactions may precede the onset of depression and serve as an ongoing risk for recurrent depression in childhood and adolescence (Rapee, 1997). Future studies may also consider the converse possibility that some parents may be *more* caring in response to adolescent depressive symptoms, attempting to comfort their children or to boost their mood, through reassurance, encouragement and praise (Sheeber et al., 1998).

Interestingly, it appears that the adolescent's perception of maternal negativity does not result from actual maternal depression. This may be an issue of measurement, with previous researchers suggesting that children may not provide an accurate report of parenting, particularly given known depressive biases (see McLeod et al., 2007; Rapee, 1997), and may only depict *perceived* parenting, as discussed here. It may be that measuring parenting through direct observation tasks would give a clearer picture, without susceptibility to reporter bias (Patton et al., 2001). Alternatively, it could be that some aspect of maternal personality, other than current depressive symptoms, predicts negativity. Parenting reflects long-term patterns and may be related to the mothers' mood and personality over time, but not necessarily to her current state at the time of reporting, particularly given the likelihood that her depression would have recurred throughout the adolescent's life (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). It was also particularly unexpected that maternal depressive symptoms did not relate to adolescent depressive symptoms in the current sample (cf. Cicchetti & Toth, 1998). This is especially surprising given previous demonstrations of a common genetic component (e.g., Rice, Harold, & Thaper, 2002; Rutter, 2002). In a community sample with low levels of depressive symptoms and relatively little variance across 6 months as seen here, it is possible that parent-offspring depression effects would be less evident than in the clinical samples considered in some previous research (e.g., Downey & Coyne, 1990; Garber et al., 1997; Sheeber, Davis, & Hops, 2002). On this basis, it may be that parental depression, parenting and self-worth factors are not significant contributors to the small changes in depressive symptoms seen here among young adolescents, but that they may be of greater significance for a clinical subgroup or across a longer time period. Alternatively, a relationship between depression and parenting behaviour may be most

applicable for parents who are severely depressed (Lovejoy et al., 2000), who may be unable to disguise their mood in interactions with their children.

This study contained several notable limitations. The established model was limited by the cross-sectional nature of the mediating paths (Cole & Maxwell, 2003), although it was more conservative than many in controlling for Time 1 depressive symptoms when examining longitudinal paths (Lakdawalla & Hankin, 2008). Other longitudinal studies of parenting and adolescent depression have also found evidence supporting only cross-sectional relationships, not longitudinal ones (e.g., Burt, Cohen, & Bjorck, 1988). Given the cross-sectional nature of the tested relationships, causality cannot be determined between perceived parenting, self-worth and depressive symptoms. Alternatively, all three constructs may be associated through a common relationship with a third factor such as negative affectivity. Although the current study focused on mothers as an initial test of the proposed model, given the strong empirical basis for an association between maternal and offspring depression (Garber et al., 2002), links between paternal and adolescent-psychopathology should also be considered (Brennan et al., 2002), along with possible gender-specific differences (Phares & Compas, 1992). Given that the influence of parental relationships, relative to peers, is known to shift during adolescence (Bednar & Fisher, 2003; Garber, 2005), future studies should consider the relative impact of parent and peer interaction styles on self-worth and internalising symptoms. Finally, a disadvantage inherent in studies using school samples is that they may unavoidably exclude students who have poor attendance, and therefore may under-represent children with depressive disorders (Fleming & Offord, 1990).

The current study provides important insights into the developmental trajectory of depression in early adolescence, considering the child's perception of their mothers and their self-worth. While maternal depression was not directly linked with adolescent depressive

symptoms in the model, the young adolescent's perception of a rejecting parenting style was strongly associated with lower self-worth and higher depressive symptoms, potentially explaining part of the maintenance of depression, and extending previous theories about parenting in the development of psychopathology (e.g., Garber et al., 1997; Rapee, 1997). Whether these children are "accurately" reporting their family relationships, or sadly perceiving less support than is actually available, it is possible that they will not seek parental support for emotional and other difficulties. This perception and their lower sense of self-worth may then be critical to the maintenance and chronicity of depressive symptoms. Given that perceived maternal rejection and lower self-worth did not predict increases in depression across 6 months, over and above initial depressive symptoms, it will be important to further investigate what facets of the adolescent's experience, in combination with other psychosocial risk factors, do contribute to increases in depression over time. This model should be considered in designing intervention programs for early adolescent depression, with initiatives aimed at improving family relationships and teaching pro-active coping and cognitive strategies to children and parents alike.

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CHAPTER THREE

NEUROTICISM, LIFE EVENTS AND NEGATIVE THOUGHTS IN THE DEVELOPMENT OF DEPRESSION IN ADOLESCENT GIRLS

Abstract

Theories of depression suggest that cognitive and environmental factors may explain the relationship between personality and depression. This study tested such a model in early adolescence, incorporating neuroticism, stress-generation and negative automatic thoughts in the development of depressive symptoms. Participants (896 girls, mean age 12.3 years) completed measures of personality and depressive symptoms, and 12 months later completed measures of depressive symptoms, recent stressors and negative automatic thoughts. Path analysis supported a model in which neuroticism serves as a distal vulnerability for depression, conferring a risk of experiencing dependent negative events and negative automatic thoughts, which fully mediate the effect of neuroticism on later depression. A second path supported a maintenance model for depression in adolescence, with initial levels of depression predicting dependent negative events, negative automatic thoughts and subsequent depressive symptoms. Unexpectedly, initial depression was also associated with later *independent* life events. This study establishes potential mechanisms through which personality contributes to the development of depression in adolescent girls.

The relationship between personality and depression has been long established, but the processes involved in the relationship are not well understood (see Shea & Yen, 2005 for a review). Neuroticism, or negative emotionality, is described as a stable tendency to perceive and experience the world as threatening and distressing (Tellegen, 1985; Watson & Clark, 1992; Watson, Clark, & Harkness, 1994). It is moderately heritable (Clark, 2005), and associated with heightened sensitivity to negative stimuli, vulnerability to sadness, depression, anxiety and other negative emotions, as well as negative cognitions and appraisals, low self-esteem and life dissatisfaction (Clark, Watson, & Mineka, 1994). In adults, elevated levels of negative emotionality or neuroticism have been strongly associated with depression (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1993; Krueger, 1999), and shown to prospectively predict increases in depression (Kendler, Gatz, Gardner, & Pederse, 2006; Lakdawalla & Hankin, 2008). Despite this established link between neuroticism and depression, the actual nature of the relationship remains unclear, and few studies have examined the processes by which neuroticism may transact with other vulnerability factors to contribute to increases in depression in adults (e.g., Lakdawalla & Hankin, 2008).

In adolescence, it has been suggested that personality characteristics may increase the risk of depression (Santor & Rosenbluth, 2005), but little prospective research has been undertaken to examine the development of depression from relevant risk factors (Lakdawalla & Hankin, 2008). There has been a lack of empirical attention paid to the role of neuroticism as a risk factor for depression across time in early adolescence, and to the mechanisms through which neuroticism may contribute to later depressive symptoms in this age range.

It has been proposed that neuroticism may act as a vulnerability factor in the development of depression through the generation of stressful life events (Angold & Worthman, 1993; Hammen, 1991; Luby et al., 2002). Hammen (1991) proposed the stress-generation theory of depression, wherein individuals currently experiencing symptoms of depression would be more likely to behave in such a way as to elicit negative reactions from

peers, or to actually generate stressful circumstances and additional events, which can then lead to further increases in depression (Rudolph & Hammen, 1999). Due to either trait (e.g. personality) or state (e.g. depressed mood) characteristics, some individuals would therefore be more likely to experience negative events that are at least partly dependent on their own behaviour (dependent negative life events, e.g. relationship difficulties or lob losses, Hammen, 1991). However, events independent of the individual's actions (independent negative life events), such as a death in the family or natural disasters, would not be expected to be associated with prior depression (Hammen, 1991).

Strong evidence supports the role of overall negative life events in predicting major depressive episodes in adults , with one in five occurrences of severely negative events found to lead to depression (Goodyer, 1991).Depression has been associated with the generation of dependent negative events (but not independent ones) in cross-sectional adult studies (e.g., Compas, Howell, Phares, & Williams, 1989; Daley, Hammen, Burge, & Davila, 1997; Wingate & Joiner, 2004). However, such studies have been criticised for the simultaneous reporting of depressive symptoms and stressors, given that depressed individuals have been shown to report more negative events than non-depressed controls. Prospective studies have demonstrated that depression does in fact predict subsequent dependent (but not independent) negative life events in adults, supporting the stress-generation theory across time (Daley et al., 1997). In adolescents, recent longitudinal studies also support the stress-generation hypothesis, stemming from initial levels of depression. For example, Hankin and colleagues (2004) reported that depressive symptoms predicted later increases in objectively assessed stressors, which were concurrently associated with depressive symptoms.

Negative emotionality, or neuroticism, has also been associated with dependent negative life events in adults on a cross-sectional basis (Kendler et al., 1993), supporting Hammen's (1991) stress generation theory. Santor and Rosenbluth (2005) suggest that personality traits such as neuroticism may either precipitate or worsen the interpersonal and

achievement difficulties associated with depression, as well as the experience of depressive symptoms. Prospective studies have demonstrated that negative emotionality predicts the later occurrence of negative life events in adults (Kendler, Gardner, & Prescott, 2003); however, most research has focused on specific symptoms of depression as predicting stress-generation, rather than personality more broadly. To date, no research has been reported in this area with young adolescents.

Another factor that may play a role in the developmental links between personality vulnerabilities, stressful life events and depression is negative thinking. Many theories of depression incorporate negative beliefs around personal failure, and feelings of loss and hopelessness (e.g., Abramson, Metalsky, & Alloy, 1989; Beck, 1967; 1976; Schniering & Rapee, 2002). Cognitive theories of psychopathology propose that individuals prone to depression will display a biased perception and negative automatic thoughts about loss and failure (Leahy, 2004; Schniering & Lyneham, 2007). Fundamental theories of depression incorporate thoughts about failure in response to stress as contributing to depressive symptoms. Beck's (1983) cognitive theory proposes that following a stressful event, one's information processing bias can lead to negative automatic thoughts, which in turn contribute to depressive symptoms. Furthermore, Abramson and colleagues' (1989) Hopelessness Theory proposes that negative inferences about the self, particularly around ideas of failure and worthlessness, are associated with increases in depression. Cognitive vulnerability has also been associated with negative emotionality (Clark et al., 1994).

Diathesis-stress models of depression incorporate certain thinking styles as a diathesis, that in the presence of negative life events increase vulnerability to depression (e.g., Watts & Markham, 2005). Only limited longitudinal research has been conducted supporting these models in adolescence (e.g., Bohon, Stice, Burton, Fudell, & Nolen-Hoeksema, 2008).

Hankin and Abramson (2001) proposed an elaborated cognitive vulnerability-transactional stress model incorporating cognitive diathesis-stress and stress-generation

theories of depression, including personality as a pre-existing vulnerability that increased the likelihood of later depression. They suggest that the link between personality and depression may be explained by an increased likelihood of experiencing negative events, in combination with a greater cognitive vulnerability to depression. Preliminary research by Lakdawalla and Hankin (2008) has provided initial support for this model in adults, demonstrating that negative emotionality predicted increases in depressive symptoms longitudinally. Furthermore, results showed that cognitive-vulnerability factors interacted with dependent negative life events to predict increases in depression, with these cognitive-diathesis stress factors partly mediating the association between negative emotionality and increases in depressive symptoms (Lakdawalla & Hankin, 2008). Such research has not been reported with adolescents.

Parallel research on the interrelationships between personality factors, negative life events and depression in youth has lagged behind that in adults. There is evidence of a link between stressful events and depression in children and adolescents (e.g., Eley & Stevenson, 2000; Goodyer, 2001; Goodyer, Wright, & Altham, 1990; Hammen, Burge, & Adrian, 1991; Reinherz et al., 1989; Silberg & Rutter, 2002; Tiet et al., 2001), with the impact of negative events particularly marked for adolescent girls (Silberg et al., 1999). Longitudinal studies have demonstrated that the experience of stressors precedes the initial elevation, recurrence and exacerbation of depressive symptoms (e.g., Ge, Conger, & Elder, 2001; Goodyer, Herbert, Tamplin, & Altham, 2000). However, to date there has been little research on the role of neuroticism in the temporal development of depression, and in association with negative life events. The generation of dependent negative life events in predicting depression has also rarely been examined in early adolescence, warranting further longitudinal research.

In one of the only studies to be conducted with adolescents, Hankin (2006) reports an examination of neuroticism, stressful life events and depression longitudinally in order to test the Hankin and Abramson (2001) model. He reports that initial levels of neuroticism

predicted the occurrence of additional stressors over later follow up, and that these stressors explained the prospective association between neuroticism and increases in depression over time (Hankin, 2006). Therefore, this study provides some hints that personality variables may serve as pre-existing vulnerabilities for the development of depression in youth. Although neuroticism has recently been implicated in such a model in the adult literature (Lakdawalla & Hankin, 2008), it cannot be assumed that this theory applies to the development of depression in children or young adolescents.

Measurement bias has often been noted as a flaw in cross-sectional studies of personality and depression, because a depressed individual is more likely to report high levels of negative emotionality (see Krueger, 1999). Krueger (1999) proposes that prospective studies would better address this question. Previous researchers have called for longitudinal analyses of depression in adolescence, particularly focusing on the temporal mechanisms and processes underlying individual differences in risk susceptibility (e.g. Roberts, 1999). Such research should begin in very early adolescence, a developmentally and clinically significant period, when children have an increased understanding of their own thinking processes and self-schemas are more stable, but when the increase in the prevalence of depression is only just beginning (Roberts, 1999).

Developmentally, early adolescence is a critical time for the study of risk factors, as it is the period during which the first onset of a depressive episode occurs for many individuals, leading to further problems into adulthood if left untreated. Few studies have examined the developmental trajectory of depression in early adolescence, although adult studies suggest that neuroticism, negative life events and negative thoughts may contribute to the onset of depression. No longitudinal research exists on this relationship in early adolescence. The current study was designed to address this deficit, by proposing and testing a model to explain the development and maintenance of depression in early adolescence (see Figure 1). Extending stress-generation (Hammen, 1991) and elaborated personality and cognitive-

vulnerability (Hankin & Abramson, 2001) theories of depression, the model was designed to examine the mechanisms through which neuroticism, as a pre-existing risk factor, may lead to the development and maintenance of depressive symptoms.

Given the higher prevalence of depression in adolescent girls (e.g., Birmaher, Ryan, Williamson, Brent, & Kaufman, 1996), and the decreased likelihood of floor effects, the current study focussed on adolescent girls as an initial exploration of the proposed model. It has also been established that some risk factors operate differently for boys and girls (e.g., Angold & Worthman, 1993), which warrants studying them separately. As studies of gender differences highlight the greater role of life events in adolescent girls' depression (e.g., Bennett, Ambrosini, Kudes, Metz, & Rabinovich, 2005; Shih, Eberhart, Hammen, & Brennan, 2006), and to provide maximum power for an initial test of the proposed model without adding gender as a further variable for consideration, a sample of young adolescent girls was chosen as the focus of this study.

It was expected that dependent negative life events and negative automatic thoughts would mediate the relationships between initial levels of neuroticism and depressive symptoms, and symptoms of depression 12 months later (see Figure 1). Independent negative events were included as a separate predictor. In this way, it was proposed that neuroticism would function as a distal vulnerability factor for depressive symptoms, by contributing to the likelihood that young adolescent girls will experience dependent negative life events and negative automatic thoughts, subsequently experiencing heightened symptoms of depression. It was also proposed that these same mediators would operate between initial and subsequent levels of depressive symptoms, explaining the maintenance and worsening of depressive symptoms in adolescence.

Method

Participants

The study involved a community sample of 896 adolescent girls in grade 7, recruited through high schools on Sydney's North Shore. The mean age of participants was 12.3 years (ranging from 10.7 - 13.8 years, SD=4.8 months) at time 1. The majority of participants came from and Caucasian and Australian backgrounds, with over 80% of children and their parents born in Australia or Western Europe, 7% from Asia, 3% from the Middle East, 3% from Oceania and a small number from the Americas and other countries. Most participants were from middle- or upper-middle class backgrounds, with only 2.9% of fathers and 3.4% of mothers unemployed, which is representative of the Australian population (ABS, 2006).

Measures

The Eysenck Personality Questionnaire (EPQ, Eysenck & Eysenck, 1992) is a personality trait measure often used with adolescents (e.g., Millikan, Wamboldt, & Bihun, 2002), assessing an individual's self-reported level of extroversion (vs. introversion) and neuroticism. Items include "Are you an irritable person?" and "Would you call yourself 'tense' or 'highly-strung'?", and are answered on a dichotomous yes/no basis. The shortened version of the EPQ, based on the strongest loading 12 items as reported by Grayson, shows good psychometric properties (Grayson, 1986), and was used here for speed of administration. In the current study, internal consistency was good (α=.82).

The Centre for Epidemiological Studies Depression Scale (CES-D, Radloff, 1977) is a commonly used self-report scale. Respondents indicate how often they experienced symptoms of depression over the past week, on a 4-point scale ranging from 0 (rarely or none of the time) to 3 (most or all of the time). The full CES-D contains 20 statements, and has been standardised for high school populations (Radloff, 1991), with a score of 19 or higher indicating depressed mood. The CES-D has established psychometric properties among adolescents (Radloff, 1991). A shortened version of the CES-D was used, based on a large

school study (Sheffield et al., 2006) and factor analysis extracting the top 12 loading items (see Appendix A). Internal consistency in this sample was good (α=.75), and comparable with that of the full CES-D among junior high school students (α=.85, Radloff, 1991).

Given that depressive symptoms and failure beliefs (CATS-PF, see below) were assessed concurrently, it was important to ensure that relationships between the two factors were not due to content similarity. Considering that the content overlap would be due to the cognitive symptoms of depression being similar to personal beliefs about failure, and following previous research (Jolly & Dykman, 1994; Schniering & Rapee, 2004), a "non-cognitive" version of the CES-D was derived. Two raters each reviewed the content of the items, and decided that three items broadly assessed overlapping content ("I felt that I was just as good as other people", "I felt hopeful about the future", "I felt that people disliked me"). Due to the content similarity of these items with the construct assessed by the CATS-PF (e.g. "I can't do anything right"), these items were removed from the CES-D factor, creating a symptom-based measure of depression without cognition-based content – the CES-D-9. In this sample, internal consistency for the CES-D-9 was good (α=.72).

The Child and Adolescent Survey of Experiences (CASE, Allen, 2005) is a checklist measure of life events, derived from the Psychosocial Assessment of Child Experiences (PACE) Interview (Sandberg et al., 1993). The CASE assesses both positive and negative life events, and focuses on acute rather than chronic experiences. CASE items have been divided into subscales of items independent of the respondent's behaviour (independent negative life events, iNLE, such as "Someone in my family died") and events at least partly dependent on the respondent's actions (dependent negative life events, dNLE, such as "I did badly in an important test or exam", Allen, 2005). The CASE effectively discriminates between clinical and control adolescents based on the number and reported impact of negative life events, and demonstrates moderate to good test-retest reliability (Allen, 2005).

The CASE is measured retrospectively, with respondents reporting the occurrence of life events (yes/no) over the previous 6 months (Allen, 2005). In this way, it allows for the assessment of life events reported to have occurred in the period leading up to the time at which depression is measured.

The Children's Automatic Thoughts Scale (CATS, Schniering & Rapee, 2002) is a self-report measure, designed to assess negative automatic thoughts and negative self-statements in children and adolescents, in response to external events. Previous research has demonstrated good internal consistency, with α values greater than .85 for all subscales, and satisfactory test–retest reliability, with a test–retest correlation of .91 for the CATS total score (Schniering & Rapee, 2002). The CATS has been shown to effectively discriminate between control and clinical groups, both on the total score and between clinical subgroups on the relevant subscales (Schniering & Rapee, 2002).

CATS items load onto four separate cognitive subscales, with the Personal Failure subscale of relevance here. The CATS Personal Failure Scale (CATS-PF) indicates the extent to which the respondent has thought of themselves as a failure, or attributed negative events to their own failings, in the preceding week (Schniering & Lyneham, 2007) . Items include "It's my fault that things have gone wrong" and "I can't do anything right", and are answered on a five-point Likert scale from "not at all" to "all the time". It has been shown to discriminate depressed from non-clinical, anxious and oppositional/conduct disorder children and adolescents (see Schniering & Rapee, 2002, for details of factor analyses). In the current study, internal consistency for the CATS-PF was very good (Time 1 α=.92).

Procedure

The procedures were approved by the Macquarie University Human Research Ethics committee. Informed, written consent was received from the school, parents and adolescents before participation in the study. Questionnaire batteries were provided to all participants as a

group, in a school setting. Research assistants were available to answer questions, but did not provide any information that could influence respondents. Data were collected when participants were in 7th grade, and again 12 months later, when 89.39% participated again (attrition rate = 10.61%), with a mean age of 13.3 years. Those who did not participate at both time points were excluded from these analyses. Participants missing more than 20% of items were excluded.

Results

Descriptive statistics and correlations

There were no significant differences on initial levels of variables in the model (independent group t-tests, all $p>.10$), between those who did not participate at Time 2 and those who participated both times. There were also no significant differences on the children's age or geographic location ($p>.10$); however, significant differences were evident for parental occupational status and marital status ($p<.01$), with those who did not participate at Time 2 more likely to have parents who were unemployed or employed in lower paid jobs, and less likely to have parents who were married. However, the model remained unchanged when these factors were controlled. No significant correlations were apparent between the participants' age and any of the factors measured.

Descriptive statistics and correlations for the observed scores on all factors represented in the models are reported in Table 1. To allow for a small number of missing items, the mean of the other items for that participant were substituted for the missing item, which is acceptable given the high internal consistency of each scale ($\alpha>.70$, Schafer & Graham, 2002). This was not done for the CASE, where internal consistency would not be expected. Means for the CES-D factors, adjusted to the reduced -12 and -9 item versions, were in the expected range seen in other non-clinical samples of Australian children (Allison, Roeger, Martin, & Keeves, 2001; Sawyer, Spence, & Pfeiffer, in preparation).

Table 1.
Descriptive Statistics and Bivariate Correlations

Variable	CES-D-12 – T1	CES-D-9 – T1	EPQ-N – T1	dNLE – T2	iNLE – T2	CATS-PF –T2	CES-D-12 – T2	CES-D-9 – T2
CES-D-12 – T1	-							
CES-D-9 – T1	.939**	-						
EPQ-N – T1	.590**	.586**	-					
dNLE – T2	.199**	.210**	.186**	-				
iNLE – T2	.149**	.154**	.136**	.325**	-			
CATS-PF – T2	.346**	.325**	.331**	.341**	.254**	-		
CES-D-12 – T2	.463**	.436**	.375**	.407**	.297**	.616**	-	
CES-D-9 – T2	.408**	.419**	.345**	.401**	.321**	.582**	.950**	-
M	7.92	4.50	4.21	.94	1.48	4.32	6.97	4.16
SD	5.81	4.40	3.17	1.13	1.37	7.04	6.03	4.50
Minimum	.00	.00	.00	.00	.00	.00	.00	.00
Maximum	34.00	25.00	13.00	4.00	7.00	40.00	36.00	27.00
Kurtosis (SD)	1.36 (.16)	2.17 (.16)	-.72 (.18)	-.18 (.17)	1.38 (.18)	7.94 (.18)	2.42 (.17)	3.33 (.17)
Skewness (SD)	1.14 (0.08)	1.44 (.08)	.49 (.09)	.96 (.09)	1.11 (.09)	2.67 (.09)	1.38 (.09)	1.66 (.09)

Note. N= 896. ** All correlations significant at p<0.01 (2-tailed Pearson's correlation). T1 = Time 1; T2 = Time 2. CES-D-12 = Centre for Epidemiological Studies, 12 item version (see Method); CES-D-9 = Centre for Epidemiological Studies, 9-item "non-cognitive" version (see Method); EPQ-N = Eysenck Personality Questionnaire Neuroticism Subscale; dNLE = dependent negative life events from the Child and Adolescent Survey of Experiences; iNLE = independent negative life events from the Child and Adolescent Survey of Experiences; CATS-PF = Children's Automatic Thoughts Scale – Personal Failure subscale.

Positive skewness and kurtosis were evident in the data, with the majority of responses clustering around low frequency of depressive symptoms, negative events and negative thoughts. This was to be expected in a non-clinical population, but was at a moderate level (all skewness values <3, Kline, 1998). Maximum likelihood estimation methods rely on an assumption of normality, yet in large samples it may still be the preferred method (Chou & Bentler, 1995). It is important to consider whether the non-normality evident in the data affects the model and path significances. Bootstrapping (with 500 samples and 95% bias-corrected confidence intervals), which requires only that the data come from a random and large sample, was used to examine the model conservatively while addressing the deviation from normality. Given that in all tested models, bootstrapping provided the same pattern of

results and path significances, it was apparent that the non-normal distributions did not markedly affect the conclusions that could be drawn from the path analyses. On this basis, maximum likelihood estimations were used for the final analyses.

Importantly, the correlation between the Time 2 "non-cognitive" CES-D-9 and the CATS-PF ($r=.582$, $p<.001$) was significantly lower than that for the CES-D-12 ($r=.616$, $p<.001$; $t(893)=-4.078$, $p=.00005$, see Steiger, 1980) . This supported the use of the non-cognitive version of the CES-D, as the CES-D-9 appears to assess a construct that is more clearly distinct from that assessed by the CATS-PF. On this basis, the CES-D-9 was used in the analyses, to provide a more valid test of the hypothesised relationships by reducing variance that could be attributed to overlapping constructs in the concurrent measures.

Path Analyses

Given that there were multiple mediators in the hypothesised model, path analysis was used to test the direct and indirect effects of each factor upon each outcome simultaneously, using Analysis of Moment Structures (AMOS) 16.0 (Arbuckle, 2007). This provides a conservative test of the hypothesised model, as testing each relationship separately in regressions would not account for the variance explained by the other relationships.

Of particular interest were the standardised regression weights of the relevant paths, for both direct and total indirect relationships. The overall model fit was also examined, using established goodness-of-fit indices. The comparative fit index (CFI, Bentler, 1990), relative fit index (RFI, Bollen, 1986), incremental fit index (IFI, Bollen, 1989) and the Tucker-Lewis Index (TLI, Tucker & Lewis, 1973) were examined, with cut-off values approaching .95 said to indicate a good fit to the model (Hu & Bentler, 1999). Additionally, to account for model complexity, the root mean square error of approximation (RMSEA, Steiger, 1990) was also examined, with its 90% confidence interval (Browne & Cudeck, 1993), where values of 0.06 or less indicate good fit (Hu & Bentler, 1999).

Model testing

The hypothesised model (Figure 1) was designed to examine the paths from initial levels of neuroticism and depressive symptoms to depressive symptoms at time 2, considering mediating variables reported retrospectively for the intervening period (dependent and independent negative life events, and failure beliefs). The hypothesised model incorporated both the direct and indirect paths of interest, and all factors proposed, to provide a conservative test of the hypotheses. This model was tested, followed by modifications to improve the overall model fit. Finally, bootstrapping was conducted to estimate the significance of the indirect paths.

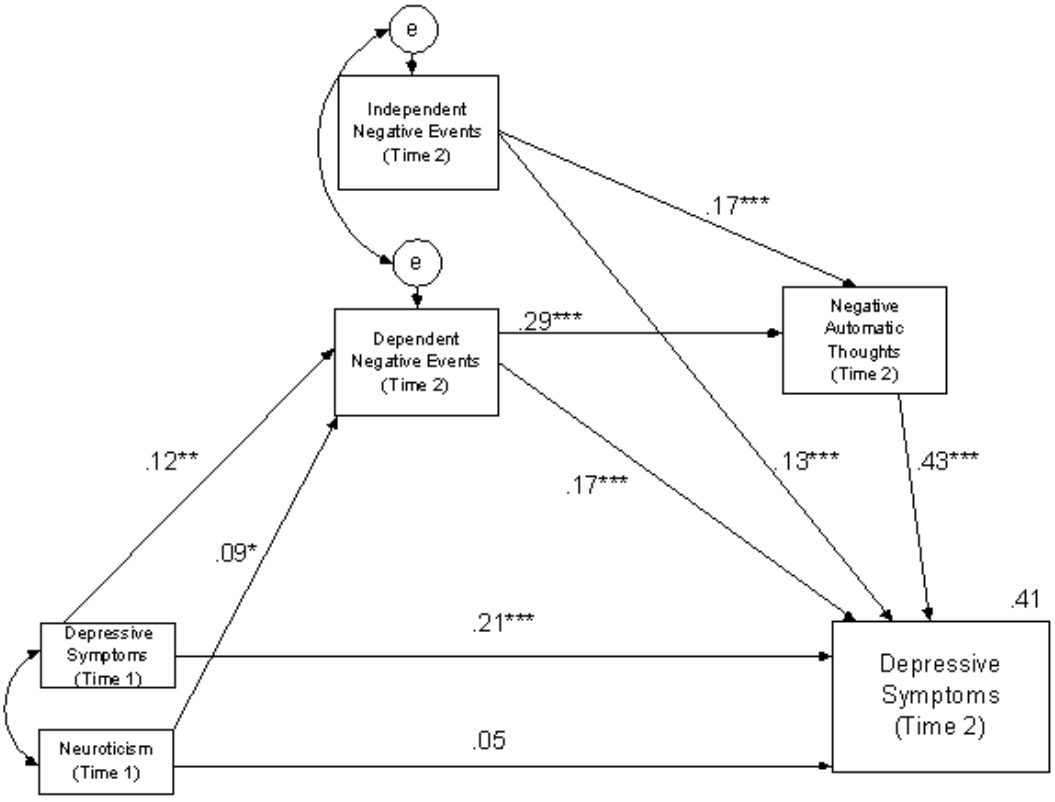

Figure 1. Hypothesised model
Note. ***=p<.001; **=p<.01; *=p<.05

As seen in Table 2, the hypothesised model did not provide a close fit for the data, despite significant regression weights for all but one path. Interestingly, the direct effect of Time 1 neuroticism on Time 2 depressive symptoms was not significant (p>.10), in a model

incorporating all the other factors. An exploration of step-wise regression analyses revealed that neuroticism was a significant predictor of Time 2 depressive symptoms at a bivariate level, when Time 1 depressive symptoms were covaried and when independent and dependent negative life events were included. However, when Time 2 CATS-PF was added, the relationship between neuroticism and later depressive symptoms became non-significant. The effect of initial levels of neuroticism on later depressive symptoms is thus fully mediated by dependent negative life events and failure beliefs. The hypothesised model explained 41% of the variance in Time 2 depressive symptoms.

Table 2.
Goodness-Of-Fit Indices

Model	χ^2/df	CFI	TLI	RFI	IFI	RMSEA	RMSEA (90% CI)
Model 1 (hypothesised)	25.54	.910	.527	.517	.911	.166	.139-.194
Model 1a	20.796	.909	.619	.607	.910	.149	.125-.174
Model 2 (modified)	1.50	.999	.990	.972	.999	.024	.000-.075

Note. CFI= Comparative Fit Index, TLI= Tucker-Lewis Index, RFI= Relative Fit Index, IFI= Incremental Fit Index, RMSEA= Root Mean Square Error of Approximation and 90% confidence interval.

Given the non-significance of the direct path from Time 1 neuroticism to Time 2 depressive symptoms, this path was removed from the model (Model 1a). Although this improved the model fit slightly, overall goodness-of-fit indices were still poor (see Table 2).

Finally, given the inadequate model fit, modifications were made at a post-hoc level (Model 2). Given the strong bivariate and conceptual associations between neuroticism and negative automatic thoughts, a direct path from Time 1 neuroticism to Time 2 CATS-PF was added. A path from Time 1 depression to Time 2 negative automatic thoughts was also added, as it would be sensible to assume that someone who has previously experienced higher levels of depressive symptoms would be likely to experience such thoughts. Modification indices were considered to examine other, non-predicted relationships within the data, in conjunction with consideration of the theoretical underpinnings for the model. Interestingly, modification

indices suggested the inclusion of a path from Time 1 depression to Time 2 *independent* negative life events.

This modified model (Figure 2) provided an excellent fit for the data (see Table 2), with all individual paths significant at $p<.001$, aside from the path from neuroticism to dependent negative events ($p<.05$). The total proportion of the variance in Time 2 depression accounted for by this model was 45%.

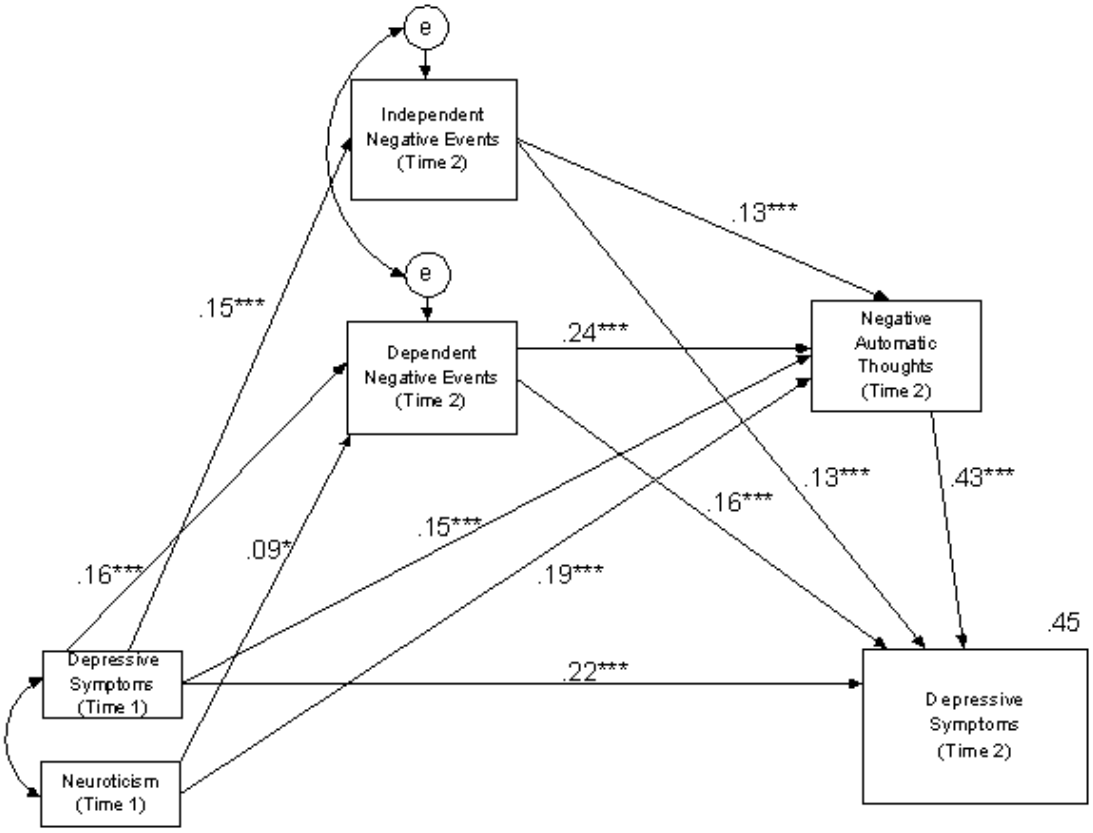

Figure 2. Modified model
Note. ***=$p<.001$; **=$p<.01$; *=$p<.05$

Finally, the significance of the indirect paths was assessed in AMOS, using bootstrapping tests of indirect effects with two-tailed significance levels (using 500 samples and 95% bias-corrected confidence intervals). All the indirect paths in the model were found to be significant. The indirect effect of initial neuroticism on Time 2 depressive symptoms, via dependent negative life events and negative automatic thoughts, was highly significant ($p<.01$), as was the indirect effect of initial levels of depressive symptoms on symptoms 12

months later, via these same mediators (p<.01). The indirect effects of neuroticism and initial depressive symptoms on negative automatic thoughts, via only dependent negative events, were also significant (p<.05); while the indirect effects of both independent and dependent negative events on Time 2 depressive symptoms, via only negative automatic thoughts, were also demonstrated (p<.01).

Overall, as seen in Table 2, the hypothesised model did not show a good fit for the data, while the modified model (Model 2) provided an excellent fit. As a distal vulnerability for depressive symptoms, the effect of neuroticism was mediated through increased negative events that were at least partly dependent on the individual's behaviour, and negative automatic thoughts about failure and loss. Similarly, the effect of initial depressive symptoms on the experience of depressive symptoms12 months later was partly mediated through dependent stressors as well as independent ones, and through negative automatic thoughts, although a direct effect was also evident.

Discussion

The results of the present study suggest that the developmental trajectory between neuroticism and depressive symptoms may be explained by an increased likelihood of dependent negative life events and failure beliefs. The demonstration that dependent negative life events and failure beliefs mediate the relationship between initial levels of neuroticism and later depressive symptoms, controlling for initial depressive symptoms, may explain the mechanisms through which personality serves as a distal vulnerability factor for depression. Adolescents with higher neuroticism, tending to perceive negativity and threat, are more likely to experience negative events that are at least partly dependent on their own behaviour, such as failing exams and fighting with friends. They are also more likely to have negative automatic thoughts around failure, loss and hopelessness, associated with an increase in depressive symptoms. The second path demonstrated, wherein dependent negative life events and negative thoughts partly mediated the relationship between initial and subsequent

depression symptoms, provides a possible explanation for the maintenance and worsening of depression in adolescence. The unexpected finding that independent negative life events are also associated with initial levels of depressive symptoms raises interesting questions about environmental and measurement issues, discussed below. Overall, the current study provides both a possible explanation for personality as a vulnerability to depression, and a maintenance pathway through which young adolescents who are already experiencing depressive symptoms may go on to experience chronic depression or depressive disorders.

The first path established here, wherein neuroticism confers vulnerability to later depressive symptoms, furthers both stress-generation (Hammen, 1991) and personality theories of depression (e.g., Hankin & Abramson, 2001). Hankin and Abramson's theory (2001) proposes that the link between personality and depression may be explained by an increase in negative events and a cognitive vulnerability to depression, with initial support found with adults very recently (Lakdawalla & Hankin, 2008). The developmental trajectory from a neurotic personality trait to depressive symptoms in early adolescence was here shown to be mediated by the propensity to experience negative life events that are at least partly dependent on behaviour, and by a tendency to engage in thoughts focussing on failure and loss. This highly significant pathway has never before been demonstrated with adolescents, and may provide insights into the development of depressive disorders. For example, a more neurotic teenager may be more likely to have difficulty concentrating, and so perform badly in an exam. They may then perceive this event as a sign of personal failure/inadequacy, and so experience increased symptoms of depression. This pattern of reacting to stressful life events could quickly develop into a negative feedback loop, whereby patterns of behaviours and thoughts lead to dependent negative life events and subsequent depressive symptoms, in an escalating cycle.

The second path demonstrated here, examining the maintenance of depressive symptoms over 12 months in early adolescence, supports and extends stress-generation

theories of depression (e.g., Hammen, 1991). In early adolescence, the relationship between initial levels of depressive symptoms and symptoms 12 months later was shown to be partly mediated by the occurrence of dependent negative life events, and also by negative beliefs about failure. Extending the stress-generation theory, the tendency of some adolescents to engage in negative thoughts about failure further increases the risk of depression. This important finding provides a possible explanation for increases from commonly experienced, low levels of depressed mood to clinical levels of depression among adolescents, which are known to confer a greatly increased risk of depressive disorders throughout life (Harrington, Fudge, Rutter, Pickles, & Hill, 1990).

One unexpected finding was the empirically derived path suggesting that initial levels of depressive symptoms predicted the reporting of *independent* negative life events 12 months later. Although stress generation theories (Hammen, 1991) predict that depressive symptoms may increase the generation of behaviour-dependent life events, there is little reason within these theories to expect an association with independent life events. One possible explanation for this results is that people high on depressive symptoms may be more likely to live in disadvantaged environments (Sawyer et al., 2000). Such environments are likely to increase the probability of experiencing both independent and dependent negative life events. It is also possible that children living in poor environments may develop negative thinking styles as a result of prior experiences. An explanation such as this, invoking the influence of a common third factor, is reflected in the identified shared variance between the error terms of independent and dependent negative events (in each model). For example, an individual in a poor environment, such as at socio-economic disadvantage, could report a higher rate of depressive symptoms at Time 1 and subsequently report more negative life events independent of their own behaviour (e.g., a parent losing their job) as well as partly dependent on it (e.g., poor school performance, where the child spends free time helping to support the family and so cannot study). Contrary to stress-generation theories, Sandberg and colleagues

(1998) have suggested that children with psychiatric diagnoses experience more negative life events and chronic adversities than non-diagnosed children, regardless of whether the events were dependent or independent of the child's behaviour. The current findings are consistent with this suggestion for early adolescent girls' depression.

Another possibility is that adolescents may have experienced events that are traditionally categorised as independent, but may not entirely be so (e.g., parental divorce may partly reflect family conflict including adolescent behaviour). Despite focusing on acute rather than chronic events, some CASE items may also incorporate ongoing events which would be reported – and influence mood – at both time points (e.g., death in the family following illness). Interview measures of life events would provide a more accurate assessment of the independence of stressors, and should be considered in future research. Finally, it is also possible that a reporting or shared method bias, where the respondent's current level of depressive symptoms influenced their reporting of negative events, may explain the relationship between depressive symptoms and independent negative events. Although items listed on the CASE were designed to reduce this potential limitation by including independent negative events that are relatively concrete and less open to reporting bias (e.g., "My parents split up", Allen, 2005), self-report measures of life events are limited by their assessment of the respondents' perspective, and are thus open to bias (Monroe & Roberts, 1990). Future research could extend this model and its impact on stress-generation theories in adolescence, by more closely assessing socio-economic circumstances and other aspects of the physical environment, including reports of negative events by impartial respondents, by utilizing interview-based assessment of negative events and by controlling for initial levels of independent and dependent negative events.

The developmental pathway from neuroticism to depressive symptoms, demonstrated here, is consistent with initial research in adults (Lakdawalla & Hankin, 2008), and supports newer, more comprehensive theories of depression (e.g., Hankin & Abramson, 2001). The

clinical and intervention implications of this model are considerable. If more neurotic adolescents are likely to generate stressful life events, and to experience negative automatic thoughts and increased depressive symptoms, this provides valuable insight into the need for preventative clinical efforts to focus on children and adolescents exhibiting neurotic tendencies. Training in social skills and cognitive restructuring might allow these adolescents to avoid the negative interactional pattern (Hammen, 1999) and cognitive style (Schniering & Rapee, 2004) implicated here in the translation of negative affectivity to depressive symptoms. Furthermore, training in problem solving skills would encourage teenagers to seek active solutions to their problems.

Developmentally, a shift in self concept in early adolescence may also play a role in the development of depression. Younger children view themselves in concrete, physical terms; while older children begin to view themselves in more abstract psychological terms, including stable personal characteristics (Damon & Hart, 1982). This shift in focus has been suggested to be potentially negative for children who are at risk of an affective disorder (Eley, 1997), where a cognitive vulnerability to depression may first emerge (e.g., Abela, 2001). It could be speculated that if a young adolescent perceives negative events as reflecting personal failure, they may then integrate this personal belief into their self concept at a formative developmental stage, undermining their sense of self-worth (Shortt & Spence, 2006) and placing them at further risk in the future. Early adolescence is a time of significant psychosocial and biological changes, with the onset of puberty, academic and social demands in the transition to high school and changing relationships with family possibly contributing to the onset of depressive symptoms (Ge, Lorenz, Conger, Elder, & Simons, 1994; Yap, Allen, & Sheeber, 2007). Although all adolescents in this study may be experiencing these changes, an assessment of pubertal timing would allow a closer consideration of possible biological factors.

The elaborated cognitive vulnerability-transactional stress model (Hankin & Abramson, 2001), here extended into a developmental pathway in adolescents, warrants more detailed examination, particularly regarding the cognitive processes involved in the development of depression. Future research would do well to examine these factors in more detail, considering cognitive schemas hypothesised to contribute to depressive symptoms, and the specific nature of negative events relative to the individual's self-beliefs. The important role of genetic vulnerability and parental psychopathology should also be considered, with particular attention paid to interpersonal and social difficulties.

The causal implications of the current study are limited by its half-longitudinal design (Cole & Maxwell, 2003), wherein negative life events, negative automatic thoughts and Time 2 depressive symptoms were measured concurrently. Future research exploring a fully longitudinal design would allow a clearer examination of causation (Maxwell & Cole, 2007). However, the retrospective nature of the life events measure, as well as controlling Time 1 depressive symptoms, provided a more conservative test of the model than the many cross-sectional mediational studies conducted to date (Lakdawalla & Hankin, 2008). Additionally, the current models were designed based on theoretically derived pathways and subsequent modifications, although numerous alternative models may exist. While path analysis allows the testing of a proposed conceptual model, alternative models could provide as good or better explanations of the data and can be tested in competition (Levy & Hancock, 2007). Future research may consider alternative pathways to explain the relationship between neuroticism and depressive symptoms in young adolescent girls, although the current study provides an important test of previous theories. The current study is also limited by its focus only on young adolescent girls, as an initial test of the proposed model. Future research should also replicate this model with boys, as although the current sample of early adolescent girls was chosen to maximise power and increase the likelihood of identifying crucial relationships, it cannot be assumed that the model applies equally to boys (cf. Hankin & Abramson, 2001).

In future, research may also extend the proposition that stress generation underlies comorbid anxiety and dysthymia (Harkness & Luther, 2001), by examining neuroticism as a possible common risk factor for both disorders, in a stress-generation model as established here. Finally, scores on the CES-D measures remained relatively stable across the 12 month time period (see Table 1), which may suggest a more chronic, low level of depressive symptoms across time in this age range. Future research may examine the exact trajectory of depressive symptoms in this early adolescent age range in more detail. This study was limited by its assessment of depressive symptoms, rather than clinical diagnoses of depression. Future research, employing interview-based assessments of depression diagnoses, will be an important extension of the present work.

Overall, the model demonstrated here is of great importance to our understanding of the development of depression in adolescence, and the relationship between personality traits and depression. While many adolescents experience depressed mood at times (Kessler, Avenevoli, & Merikangas, 2001), the question of why some continue along a developmental trajectory to depressive disorders is not well understood. Here, the role of personality as a distal vulnerability factor was considered in the development and maintenance of depression, along with initial levels of depression. Given that neuroticism was only found to predict later depressive symptoms through the generation of dependent negative life events and the tendency to experience negative automatic thoughts about failure, future clinical efforts should focus on alleviating the behavioural and cognitive consequences of neurotic tendencies, particularly in generating negative events and maladaptive negative thoughts. The developmental and maintenance models shown here are consistent with suggestions that neuroticism, as a distal vulnerability factor, influences the development of depression in adolescence, and that initial levels of depressive symptoms could develop into more serious depressive disorders through cognitive and stress-generation factors. Future research, extending the current model to adolescent boys and considering pre-existing and impartially

reported life events, will further establish the developmental pathway from vulnerable personality traits to depressive disorders.

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APPENDIX A

CES-D-12 Items

1. I did not feel like eating; my appetite was poor.
2. I felt that I could not shake off the blues, even with help from family or friends.
3. I felt that I was just as good as other people. * #
4. I felt depressed.
5. I felt hopeful about the future. * #
6. I was happy.
7. I felt lonely.
8. People were unfriendly.
9. I had crying spells.
10. I felt sad.
11. I felt that people disliked me. #
12. I could not get “going”.

* Recoded

Removed in non-cognitive version (CES-D-9)

CHAPTER FOUR

A TEST OF A COGNITIVE DIATHESIS – STRESS GENERATION PATHWAY

IN EARLY ADOLESCENT DEPRESSION

Abstract

This study evaluates a pathway for depressive risk that integrates cognitive diathesis-stress and stress-generation theories, following Hankin and Abramson's (2001) elaborated cognitive-diathesis transactional stress model. In this model, young adolescents with initial depressive symptoms would be hypothesised to experience later stressors that were at least partly dependent on their behaviour. The interaction of cognitive vulnerability, a tendency to make depressogenic attributions and to ruminate, with these dependent stressors would then predict depressive symptoms after six months. This model was supported in a sample of 756 young adolescents, with cognitive style and dependent stressors partly mediating the relationship between initial and subsequent depressive symptoms. Cognitive vulnerability was also linked with an increased likelihood of dependent stressors.

An extensive literature has demonstrated an association between recent stressful life events and symptoms of depression in adolescents, yet the occurrence of stressors does not always lead to depression (e.g., Rutter, 2000; Silberg & Rutter, 2002). In recent years, two theories have been proposed to further describe the relationships between life events and depression. Cognitive diathesis-stress theory proposes that an individual with certain cognitive tendencies will interpret the occurrence of stressors in such a way as to increase the likelihood of depressive symptoms (Abramson, Metalsky, & Alloy, 1989; Beck, 1987). Stress-generation theory suggests that at-risk individuals will behave in such a way as to at least partly cause the occurrence of stressful life events, subsequently becoming more depressed (Hammen, 1991). In a logical combination of these theories, Hankin and Abramson (2001) proposed an elaborated cognitive vulnerability-transactional stress model. They suggest that cognitively vulnerable individuals will respond to the occurrence of negative events with increased depressive symptoms, which may partly feed back to contribute to the occurrence of interpersonal stressful life events (Hankin & Abramson, 2001).

Cognitive theories of depression emerged following observations that depressed individuals portrayed certain cognitive tendencies (Beck, 1967; 1976). Noting that not all individuals who are exposed to negative events develop depression, the cognitive diathesis-stress model of depression suggested that cognitive vulnerabilities determine the impact of stressful life events on the development of depressive symptoms (Abramson et al., 1989; Beck, 1987). Extensive research supports a generic cognitive diathesis-stress model, in which an overall negative cognitive style interacts with the occurrence of a variety of stressors (e.g., Allen, de L Horne, & Trinder, 1996; Fresco, Sampson, Craighead, & Koons, 2001).

Different types of cognitive processing have been implicated in diathesis-stress models. Abramson and colleagues (1989) proposed Hopelessness Theory, implicating a cognitive style characterised by attributing stressful events to stable and global causes, the belief that the event will lead to further negative events and indicate that something is wrong

with them, predicting hopelessness symptoms of depression. For example, an adolescent who tends to interpret academic failures as a sign of personal weakness, as commonly occurring to them, likely to happen in the future and to have extensive consequences will become more depressed following such a failure. Extensive research supports this model with adults and adolescents (e.g., Abela, 2001; Abramson et al., 1999).

A tendency to ruminate in response to low mood has also been implicated as a cognitive vulnerability among adults and adolescents (Abela, Brozina, & Haigh, 2002; Nolen-Hoeksema & Morrow, 1991). Individuals with a ruminative response style are said to respond to initial low mood by focusing passively and repetitively on their symptoms, and the possible causes and consequences of their symptoms, without taking action to relieve them (Nolen-Hoeksema, 1998). Watkins and colleagues (2001; 2008) describe rumination as a processing mode which is most detrimental when self-focused (Watkins & Moulds, 2005; Watkins & Teasdale, 2001) or focused on the causes and experience of a problem, rather than on the process of how to solve the problem (Watkins & Baracaia, 2002). Nolen-Hoeksema (1998) suggests that rumination enhances the effect of depressed mood on thinking by making negative interpretations and memories more accessible and more likely. Rumination may prevent the individual from taking appropriate action to solve their problems or improve their mood (Nolen-Hoeksema, 1998), reducing the use of social problem-solving in particular (Watkins & Baracaia, 2002) and impairing executive processing (Watkins & Brown, 2002). Watkins and colleagues (2008) suggest that following a stressful event, rumination exacerbates an initial negative emotional response, worsening symptoms of depression. Evidence supports the role of rumination in the development of adolescent depression (e.g., Park, Goodyer, & Teasdale, 2004). Depressive rumination has been shown to worsen emotional responses to a stressful event in experimental studies (Thomsen, Jørgensen, Mehlsen, & Zachariae, 2004; Watkins et al., 2008) and to interact with stress (Guastella & Moulds, 2007) thus suggesting a diathesis-stress process.

Hankin and Abramson (2001) integrated a negative attributional style along with a tendency to ruminate as an overall vulnerable cognitive style in the development of depression. They proposed that negative attributional tendencies or dysfunctional attitudes provide negative cognitive content, while rumination facilitates the accessibility to or activation of this content, such that the two processes provide a combined cognitive vulnerability to depression (Hankin & Abramson, 2001). Rumination has been found to interact with a negative inferential style in predicting depression (Alloy et al., 2000) and to mediate the association between cognitive risk and depression (Spasojevic & Alloy, 2001). This is consistent with the suggestion that it is not only biases toward negative information, but also a tendency to process emotion negatively, or to ruminate, that can increase emotional reactivity in response to stressful events (Watkins et al., 2008). For this reason, an overall cognitive vulnerability is considered in the present study, to provide a broad test of the proposed model, considering an adolescent's tendency both to ruminate and to make negative attributions.

Stress-generation theory proposes that some individuals, based on trait (personality) or state (depressive affect) factors, behave in such a way as to generate stressful circumstances or events for themselves (dependent stressors), which then lead to further increases in depression in a self-perpetuating maintenance cycle (Hammen, 1991). Cross-sectional and longitudinal research supports this theory with adults (e.g., Daley, Hammen, Burge, & Davila, 1997; Rudolph & Hammen, 1999; Wingate & Joiner, 2004). Recent research has demonstrated that among adolescents, initial depressive symptoms predict later increases in stressors, which are associated with increases in depressive symptoms (Cole, Nolen-Hoeksema, Girgus, & Paul, 2006; Hankin, Roesch, & Mermelstein, 2004).

While cognitive diathesis-stress theories focus on processes leading to increased depressive symptoms, stress-generation theory notes the importance of dependent stressors in maintaining depression. The elaborated cognitive vulnerability-transactional stress theory

sensibly incorporates elements of both these processes, including pre-existing vulnerabilities, cognitive vulnerabilities, negative events (independent and dependent), initial depressive affect and increases in depression (Hankin & Abramson, 2001). Hankin and Abramson (2001) propose that individuals may be vulnerable to depression on the basis of genetic, personality or environmental characteristics, which will influence both cognitive vulnerabilities (negative attributional style, dysfunctional attitudes, ruminative tendency) and the occurrence of negative events (both dependent and independent of their actions). They propose that these negative events drive an initial response of negative affect, which interacts with a depressive cognitive style to predict increases in depressive symptoms. These symptoms can then feed back into *interpersonal* dependent stressors, where the individual may seek excessive reassurance and be rejected by peers. Initial support for this theory in the association between personality, cognitive style and stressful life events in predicting later depression has recently been demonstrated with adults (Lakdawalla & Hankin, 2008) and adolescents (Kercher, Rapee, & Schniering, under review).

Young adolescence (from 13 years of age) is a time of increases in stressful life events mirroring increases in depressive symptoms (Ge, Lorenz, Conger, Elder, & Simons, 1994). The developmental hypothesis suggests that a cognitive vulnerability to depression emerges only during the transition from childhood to adolescence, when children begin to engage in abstract reasoning and formal operational thought (see Cole & Turner, 1993; Hankin & Abela, 2005 for discussion). Few longitudinal studies of risk for depression exist in this age range, which is vital as it is a time when initial increases in depressive symptoms first emerge, potentially leading to lifelong difficulties if left untreated (see Roberts, 1999). An improved understanding of early risk factors in this age range will be vital to improving prevention and early intervention efforts (Roberts, 1999). It has been suggested that from early adolescence, such negative cognitions can interact with environmental stressors to contribute to depression

(Hankin, 2006). For these reasons, early adolescence is a critical developmental stage at which to examine early depressive symptoms which may be emerging for the first time.

The current study aims to consider the possible convergence between cognitive diathesis-stress and stress-generation theories, testing Hankin and Abramson's (2001) model. It was hypothesised that initial depressive symptoms would predict increases in stressors that are at least partly dependent on the individual's behaviour – for example, the depressed adolescent may have difficulty concentrating and so perform badly in an exam, or make negative interpretations of interpersonal cues and so argue with friends or family. These dependent stressors, if combined with a cognitive vulnerability to make negative attributions (Hankin & Abramson, 2002) and a tendency to ruminate in response to low mood (Nolen-Hoeksema & Morrow, 1991), were hypothesised to predict increases in depression over six months. The interaction of dependent stressors and cognitive vulnerability was hypothesised to explain further unique variance in later depressive symptoms. As such, an adolescent who displays a vulnerable cognitive style and who experiences dependent stressors will develop more depressive symptoms than an adolescent who experiences only one of these risk factors, or than would be expected based on the sum of the two risks. To provide a full test of both elements of the model, depressive symptoms at Time 1 will be controlled (See Figure 1).

Method

Participants

Participants were a community sample of 756 seventh-grade students (50.6% boys), from high schools around Sydney, Australia, with a mean age of 12.8 years (*SD*=4.7 months) at Time 1. Schools were chosen to represent each sector of the New South Wales education system, including co-educational, boys' and girls' government, independent and Catholic schools, and from geographically and demographically diverse regions of Sydney. The majority (75%) of participants were from an Anglo-Australian background, with 7% from

Asian backgrounds, 11% European and the remainder from North America, Oceania and the Middle East. The median family income was $75-100 000, comparable with the average Australian family income (ABS, 2007). All students in 7th grade were invited to participate, although students without consent were not further questioned, so it is not possible to estimate reasons for their non-participation. At time 2, 46 students were absent (and therefore excluded from analyses), an attrition rate of 6%. Adolescents who were absent at Time 2 were slightly more likely to report depressive symptoms at Time 1 (t=3.47, p<.01). However, there was no difference on gender, school, ethnicity, family income or parents' marital status (ps>.05) between those who participated at both times, and those who did not participate at Time 2.

Procedure

Questionnaires were presented when students were in the seventh grade, and again six months later. Students with parental consent completed surveys at school in group settings. All procedures were approved by Macquarie University Ethics Committee, the Catholic Schools Office Parramatta Diocese and the NSW Department of Education and Training. To maximise sensitivity and specificity, a cut-off score of 24 on the CES-D was used to determine adolescents who may be at risk of depression (see Roberts, Lewinsohn, & Seeley, 1991). Parents and the school counsellor of students in this range (n=99) were informed, and the families were provided with referral information.

Measures

The Centre for Epidemiological Studies Depression Scale (CES-D, Radloff, 1977) is a commonly used self-report scale, and was administered at Times 1 and 2. Respondents indicate how often they experienced symptoms of depression over the past week, on a 4-point scale ranging from 0 (rarely or none of the time) to 3 (most or all of the time). The CES-D contains 20 statements, and has been standardised for high school populations, showing good psychometric properties among adolescents (Radloff, 1991). Internal consistency in this

sample was good (Time 1 α=.88; Time 2 α=.84), and comparable with that of the full CES-D among junior high school students (α=.85, Radloff, 1991).

The Adolescent Cognitive Style Questionnaire (ACSQ, Hankin & Abramson, 2002) was administered at Time 1. The ACSQ traditionally contains 12 hypothetical events, such as "You take a test and get a bad grade", for which participants rate the degree to which the cause of such an event would be stable or global, indicated a flaw with themselves and the likelihood of further negative consequences resulting from the event. Scores on each item range from 1 to 7, with high scores indicating a negative attributional style, and were averaged to form a scale score indicating the overall attributional tendency. For the younger, Australian sample, items were slightly amended (items relating to college, dating and work were removed), such that 7 scenarios remained relating to school performance, extracurricular achievements, appearance, trouble with parents and teacher, and social situations. The ACSQ has established internal consistency (α=.92), two-week test-retest reliability (*r*=.73) and concurrent validity with depressive symptoms (*r*=.82, Hankin & Abramson, 2002). In the current sample using 7 scenarios, internal consistency was very good (α=.94).

The Ruminative Responses Scale of the Response Styles Questionnaire (RRSQ, Nolen-Hoeksema & Morrow, 1991) was administered to assess children's tendencies to ruminate in response to their experience of negative emotion, focusing on the causes, meaning and consequences of low mood. The RRSQ includes 22 items (e. g., "Think about how sad you feel"), which respondents rate on a four-point scale from almost never to almost always. Previous research has established strong internal consistency (α=.90) and psychometric properties for the RRSQ (Nolen-Hoeksema, 2000; Nolen-Hoeksema & Morrow, 1991). In the current sample, the RRSQ showed excellent internal consistency (α=.93).

A composite "cognitive vulnerability" score was created following Hankin and Abramson's (2001) elaborated cognitive theory, by summing the standardised scores of the ACSQ and RRSQ at Time 1. High scores on the cognitive vulnerability factor reflected the

adolescent's tendency to attribute negative events in a depressogenic pattern (high ACSQ) and to ruminate in response to depressed mood (high RRSQ).

The Adolescent Life Events Questionnaire (ALEQ, Hankin & Abramson, 2002) assesses a range of stressful life events common among adolescents, including school and achievement problems, social difficulties and family problems. Items include "got a bad report card" to assess academic difficulties, "had an argument with a close friend" for social difficulties and "your parents grounded you" for family difficulties (Hankin, & Abramson, 2002). Again, the wording of some items was slightly modified for a younger, Australian audience (items regarding the honour roll and dating were removed). Participants rated how often particular events happened to them over the preceding six months at Time 2, to cover the intervening period between assessments. Items were rated on a 5-point Likert scale from never (0) to always (4). The ALEQ has established test-retest reliability over two weeks (r=.65, Hankin & Abramson, 2002).

For the current study, stressful life events that were likely to be at least partly dependent on the adolescent's actions (e.g., poor school results, arguments) were summed into a "dependent stressor" factor. First, items that were ambiguous or involved the adolescent's interpretation (e.g., "had to do chores/work when you don't want to") were excluded, based on ratings made by the two authors. The two authors then independently rated whether the remaining items may be partly dependent on the adolescent's behaviour (n=21). Three additional psychologists then independently rated these items, to examine inter-rater reliability. The intra-class correlation coefficient was r=.91 for ratings of dependent stressors (p<.001).

Results

Descriptive Statistics and Bivariate Relationships

In a community sample, the skewness toward low depressive symptoms was to be expected (see Table 1), but was at a moderate level (all skewness values <3, Kline, 1998).

However, it is important to consider whether non-normality in the data affected the model and path significances. Bootstrapping (with 500 samples and 95% bias-corrected confidence intervals), which requires only that the data come from a random and large sample, was used to examine the model conservatively while addressing the deviation from normality. Given that in the proposed models, bootstrapping provided the same pattern of results, it was apparent that the non-normal distributions did not markedly affect the conclusions that could be drawn from the path analyses. On this basis, maximum likelihood estimations were used for the final analyses (cf. Chou & Bentler, 1995).

Table 1.
Descriptive Statistics and Bivariate Correlations

Variable	CES-D T1	ACSQ – T1	RRSQ – T1	Cognitive Vulnerability T1	Dependent Stressors T2	CES-D T2
CES-D T1	-	-	-	-	-	-
ACSQ – T1	.525***	-	-	-	-	-
RRSQ – T1	.743***	.535***	-	-	-	-
Cognitive Vulnerability – T1	.729***	.879***	.873***	-	-	-
Dependent Stressors – T2	.484***	.375***	.469***	.488***	-	-
CES-D T2	.579***	.380***	.541***	.530***	.620***	-
M	12.92	2.95	33.93	-.02	14.97	11.64
SD	9.57	1.05	10.86	1.73	9.97	8.55
Minimum	0.00	1.00	22.00	-2.96	0.00	0.00
Maximum	56.00	7.00	87.00	6.85	57.00	49.00
Kurtosis (SD)	1.81 (.18)	.33 (.20)	1.39 (.18)	.90 (.20)	.86 (.20)	1.99 (.19)
Skewness (SD)	1.27 (.09)	.35 (.10)	1.24 (.09)	.83 (.10)	.96 (.09)	1.32 (.09)

Note: N=756. *** Correlations significant at $p<0.001$ (2-tailed Pearson's correlation). CES-D – T1 = Centre for Epidemiological Studies Depression Scale, Time 1. ACSQ – T1 = Adolescent Cognitive Style Questionnaire, Time 1. RRSQ – T1 = Ruminative Response Style Questionnaire, Time 1. Cognitive Vulnerability – T1 = sum of standardised ACSQ-T1 and RRSQ-T1 scores. Dependent Stressors T2 = Total number of dependent stressors reported in the 6 months preceding Time 2 data collection. CES-D – T2 = CES-D, Time 2.

Bivariate Pearson correlations (see Table 1) were strong between all measured variables, most notably between Time 1 depressive symptoms and ruminative tendency

(r=.743, p<.001). The main effect variables (cognitive vulnerability Time 1 and dependent stressors Time 2) were centred before path analyses were conducted.

Path Analyses

Given that there were multiple paths of interest in the hypothesised model, with both mediation and moderation analyses planned, path analysis was used to test the direct and indirect relationships simultaneously (Klem, 2000), using Analysis of Moment Structures (AMOS) 16.0 (Arbuckle, 2007).

The standardised regression weights of both direct and indirect paths were of particular interest, given the conceptual nature of the relationships being tested. The overall fit of the model to the variation in the data was considered using established goodness-of-fit indices. Each fit index considers the ability of the model to reproduce the data, or to explain the variance seen in the data, where in each case values approaching .95 are said to indicate a good fit to the model (Hu & Bentler, 1999). The comparative fit index (CFI, Bentler, 1990), relative fit index (RFI, Bollen, 1986), incremental fit index (IFI, Bollen, 1989) and the Tucker-Lewis Index (TLI, Tucker & Lewis, 1973) were examined, each of which considers the fit of the tested model relative to a null model (see Kenny, 2008). Additionally, to account for model complexity, the root mean square error of approximation (RMSEA, Steiger, 1990) was also examined, with its 90% confidence interval, where values of 0.06 or less indicate good fit (Hu & Bentler, 1999).

Model testing

The hypothesised model was tested first (Figure 1). Both moderating and mediating relationships were considered in the hypothesised model, by drawing paths between initial depressive symptoms, cognitive vulnerability and dependent stressor main effects and their interaction term, and subsequent depressive symptoms, controlling for the path between initial and later depressive symptoms. To account for the known covariance between the main effects and interaction term in testing the proposed model, these factors were covaried (with

the residual of dependent stressors Time 2, as an endogenous variable), although the covariance was not to be interpreted. Modifications were then considered to improve the overall model fit and consider further, non-hypothesised paths (Figure 2). Bootstrapping was conducted to estimate the significance of the indirect (mediational) path, and finally, the interaction was investigated.

Hypothesised Model

The hypothesised model (Figure 1) did not fit the data well (see Table 2), but the proposed paths were supported. Initial depressive symptoms covaried significantly with cognitive vulnerability at Time 1 ($p<.001$), and predicted dependent stressors six months later ($p<.001$). Dependent stressors in turn predicted depressive symptoms at Time 2 ($p<.001$), as a partial mediator of the relationship between initial and subsequent depressive symptoms. Importantly, the interaction of initial cognitive vulnerability and later dependent stressors explained unique variance in Time 2 depressive symptoms ($p<.01$), in a model containing main effects and initial depressive symptoms. Cognitive vulnerability did not directly predict Time 2 depressive symptoms in this model ($p>.05$).

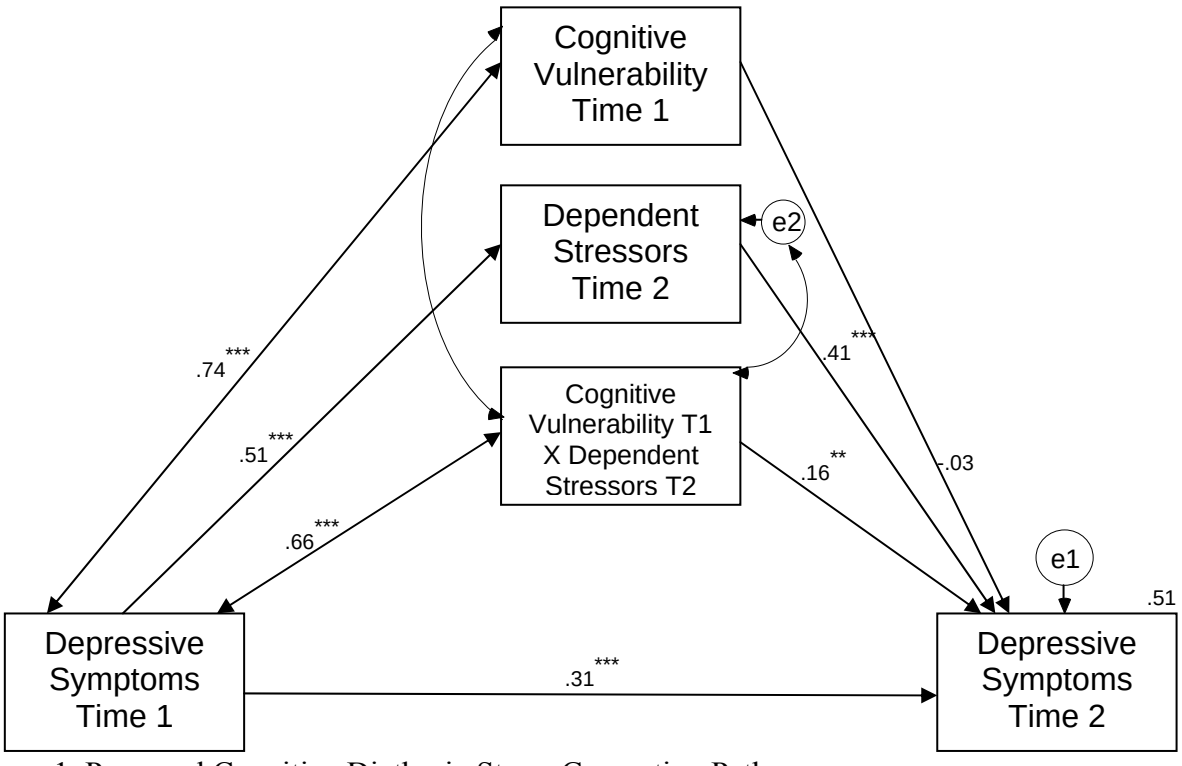

Figure 1. Proposed Cognitive Diathesis-Stress Generation Pathway
Note. ***=$p<.001$; **=$p<.01$.

Table 2.
Goodness-Of-Fit Indices

Model	χ^2/df	CFI	TLI	RFI	IFI	RMSEA	RMSEA (90% CI)
1. Hypothesised diathesis-stress generation pathway	27.606***	.985	.775	.769	.985	.188	.131-.251
2. Trimmed diathesis-stress generation pathway	0.237	1.000	1.006	.998	1.000	.000	.000-.076

Note: *** p<.001. CFI= Comparative Fit Index, TLI= Tucker-Lewis Index, RFI= Relative Fit Index, IFI= Incremental Fit Index, RMSEA= Root Mean Square Error of Approximation and 90% confidence interval.

Modified model

Given the inadequate fit of the hypothesised model, it was apparent that additional paths should be considered. In considering the theory of stress-generation, it would be sensible to consider that an individual displaying cognitive vulnerabilities might be more likely to generate stressors, in addition to the variance explained by initial depressive symptoms. On this basis, Time 1 cognitive vulnerability was covaried with the residual of Time 2 dependent stressors (p<.001). Additionally, the non-significant path between cognitive vulnerability and later depressive symptoms was removed (Figure 2). This trimmed model provided a near-perfect fit for the data (see Table 2), presenting interesting pathways from initial depressive symptoms and cognitive tendencies to later depressive symptoms, considering dependent stressors and the interaction between dependent stressors and cognitive vulnerability[1].

[1] To ensure the applicability of this model to each individual cognitive vulnerability factor, the same model was run separately with the ACSQ and its interaction with dependent stressors, and the RRSQ and its interaction term. The former showed the same pattern of results, although the interaction term was then significant at p<.05 and the fit indices slightly poorer. The latter also showed the same patterns and excellent fit indices, with the interaction term significant at p<.001.

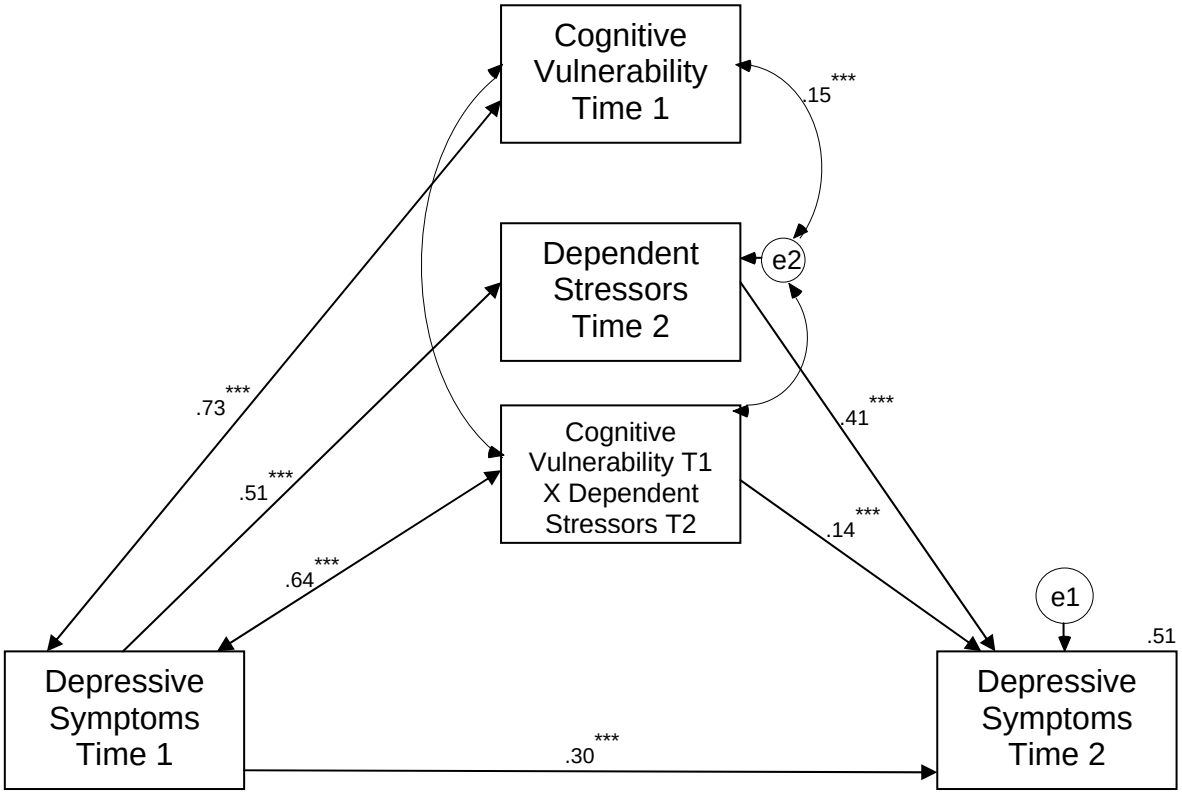

Figure 2. Modified Cognitive Diathesis-Stress Generation model
Note. ***=p<.001.

The significance of the indirect path was then determined, using bootstrapping tests of indirect effects with two-tailed significance levels (using 500 samples and 95% bias-corrected confidence intervals). The indirect effect of initial depressive symptoms on subsequent depressive symptoms via dependent stressors was significant (standardised regression coefficient= .211, p<.01), that is, dependent stressors and the interaction term partly mediated the relationship between initial and subsequent depressive symptoms.

Finally, the significant interaction of cognitive vulnerability with dependent stressors in predicting subsequent depressive symptoms was further examined. In order to more clearly interpret the nature of the interaction, the sample was divided into two dichotomies based on median splits of the number of dependent stressors and cognitive vulnerability. As can be seen in Figure 3, the effect of experiencing a dependent stressor on subsequent depression scores was greater for the more cognitively vulnerable group, than for the less vulnerable

group. Separate ANOVAs were conducted for participants who reported low cognitive vulnerability and those who reported high vulnerability, to compare Time 2 depressive symptoms between groups who experienced low vs high numbers of dependent stressors.

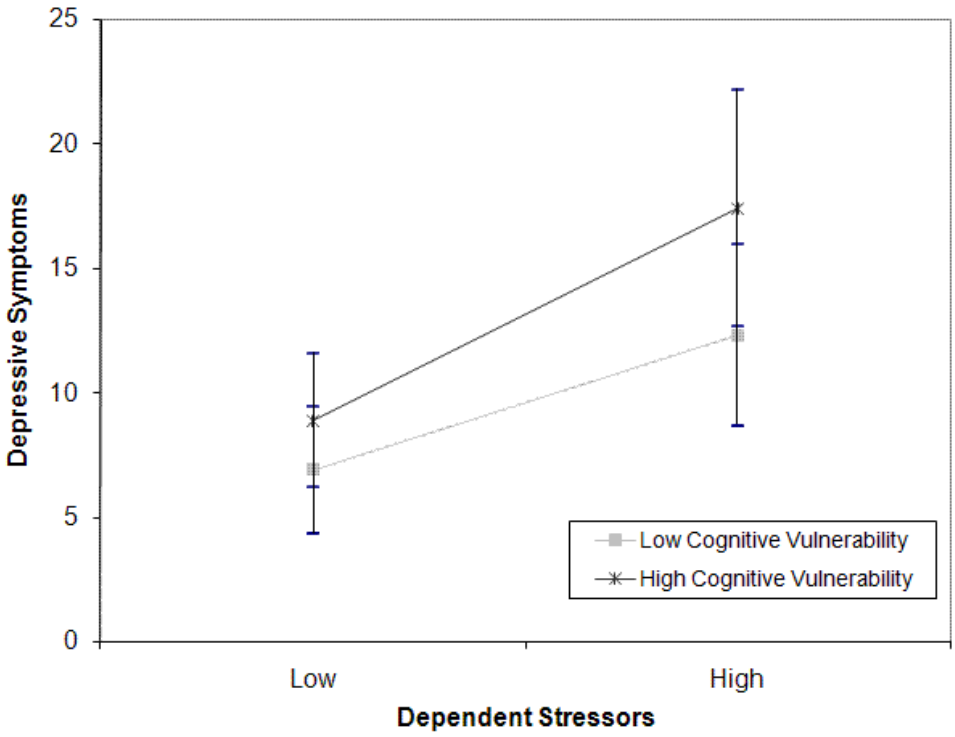

Figure 3. The interaction of cognitive vulnerability with dependent stressors, predicting Time 2 depressive symptoms

Among participants who reported a lower cognitive vulnerability, those with low levels of dependent stressors had a mean CES-D at Time 2 of 6.87 (SD=5.12), significantly lower than participants who experienced a high level of dependent stressors (mean=12.29, SD=8.31; F=50.26, $p<.001$, Cohen's $d=0.86$). Among participants who reported high rates of cognitive vulnerability, those who experienced less dependent stressors had a mean CES-D score at Time 2 of 8.87 (SD=5.35), while those with high dependent stressors reported significantly more depressive symptoms (mean=17.39, SD=9.51, F=59.638, $p<.001$, Cohen's $d=1.10$). As can be seen, the difference in depression between participants who experienced high vs low levels of dependent life events showed a larger effect size for participants with high cognitive vulnerability, than for those with low cognitive vulnerability.

Discussion

This study tested a cognitive diathesis-stress generation pathway to examine the development and maintenance of early adolescent depression. Initial depressive symptoms and cognitive vulnerability predicted the occurrence of stressful life events that were at least partly dependent on the adolescent's behaviour, which in turn predicted increases in depressive symptoms six months later. The interaction of cognitive vulnerability factors (negative attributional style and tendency to ruminate) with dependent stressors predicted unique variance in Time 2 depressive symptoms. Importantly, this suggests that adolescents who both possess a vulnerable cognitive style, and who experience stressful events at least partly based on their own behaviour, will be more likely to become depressed than an adolescent who reported only one of these risk factors, or than would be expected based only on the sum of these risks. Cognitive vulnerability factors, dependent stressors and their interaction partly mediated the relationship between initial and subsequent depressive symptoms, in a model potentially explaining both increases and the maintenance of depressive symptoms in early adolescence.

While the cognitive diathesis-stress model essentially explains the possible cause of depression (e.g., Metalsky & Joiner, 1992), stress-generation models suggest more about its maintenance and chronicity as a self-perpetuating cycle (Pettit & Joiner, 2006; Rudolph et al., 2000). Among adolescents, depressive symptoms tend to be persistent , particularly at the level of low mood or dysthymia (Kovacs, Akiskal, Gatsonis, & Parrone, 1994). Despite this, models of risk tend to focus on causal factors leading to increases in symptoms, rather than maintenance models (cf. Abela & Hankin, 2008). Following Hankin and Abramson (2001), the model supported here combines the possible causal path seen in diathesis-stress models, wherein individuals with a negative cognitive style are more at risk following stressful life events; with the maintenance pathway of stress-generation, wherein individuals at risk are more likely to report stressors that are at least partly dependent on their behaviour. This model

suggests a cycle that perpetuates across time, hinting at the mechanisms that may both initiate and maintain or worsen depressive symptoms in adolescence.

The association between initial and subsequent depressive symptoms was mediated through several potential contributing mechanisms. Firstly, initial depressive symptoms correlated with concurrent cognitive vulnerability – a tendency to make negative attributions in response to negative events (Abramson et al., 1989), and a tendency to ruminate in response to low mood (Nolen-Hoeksema, 1998). This overall cognitive vulnerability in turn predicted the occurrence of dependent stressors, as did initial depressive symptoms directly. The findings also extended the stress-generation model (Hammen, 1991) by implicating cognitive factors as a basis for stress-generation. For example, an adolescent tending to attribute an academic failure to internal, stable and global causes might become preoccupied with such thoughts and unable to concentrate, so performing badly in an exam. Adolescents who ruminate about their low mood may be less likely to take proactive, positive coping actions, so increasing their likelihood of dependent negative events. Hankin and Abramson's (2001) theory also incorporates dysfunctional attitudes as a cognitive vulnerability factor, based on the work of Beck (1987), including such beliefs as basing one's self-worth on others' approval that could be activated if an acquaintance does not say hello. Dysfunctional attitudes were not assessed in the current study, but could be an interesting extension in future research.

Secondly, the interaction between cognitive vulnerability and dependent stressors also partly mediated the link between initial and subsequent depression, explaining unique variance in depressive symptoms at Time 2 over and above the main effects and initial depressive symptoms. This suggests that adolescents who make negative attributions, tend to ruminate and experience *dependent* stressors are more likely to become depressed than expected based on the added risks of cognitive style and stressors. The adolescent who is

already experiencing depressive symptoms and/or cognitive vulnerability will report more symptoms of depression following such dependent stressors.

The model demonstrated here carries important implications for intervention and prevention in adolescence. Consistent with the fundamental tenets of cognitive behavioural therapy (e.g., Clark & Beck, 1990), it appears that initial negative cognitive style and depressive symptoms predict later maladaptive behaviour among young adolescents. Given the established risk among young adolescents with a negative attributional style and tendency to ruminate both to develop depressive symptoms and to generate stressful life events, screening programs could target these individuals for early intervention. Such intervention programs should aim to shift the adolescents' negative attributional style and to reduce ruminative responses, also increasing active problem solving skills in an effort to prevent dependent stressors and subsequent depressive symptoms. Given that initial evidence links cognitive schema with parenting (Kercher & Rapee, under review), and specifically associates parent and child rumination as a possible learned process (Kercher & Rapee, in preparation), such intervention programs should also consider the possibility that cognitive vulnerability factors are influenced by the home environment, incorporating family therapy and support.

Several limitations should be considered in designing future studies of this model. Firstly, the use of self-report measurement has been criticised in the past for the possible bias in reporting, especially regarding life events as discussed below (Monroe & Simons, 1991). Initial depressive symptoms were controlled throughout the models, to control systematic bias due to depressive symptoms. However, when assessing internalising constructs such as depressive symptoms and cognitions, many researchers argue that self-report is the preferred method, as these are subjective phenomena where the validity of external report is debatable (cf. Clarke, Lewinsohn, Hops, & Seeley, 1992). A multi-method study, including interview assessments of life events in greater detail, clinical interview assessment of depression and possibly third-party reports, is needed in future research to support the current findings. For

ethical and duty of care reasons, the parents and school counsellors of adolescents reporting high CES-D scores were informed of their risk of depression, and referred to appropriate support services. Of these, 6 participants reported having received treatment for depression at Time 2. This raises two possible limitations given the self-report methodology. Firstly, adolescents may be less likely to self-report at Time 2, if their report at Time 1 led to parental contact (although parents were strongly encouraged to gently investigate their child's need for support, without referring to the study). Secondly, the adolescents who received treatment between Times 1 and 2 may have altered trajectories of their depressive symptoms, although the number of such participants was too small to have much impact on the results.

Secondly, the ALEQ does not have an established protocol for distinguishing between dependent and independent items – such a dichotomy would be difficult to validate without supporting interview and/or third party reports of independent events and surrounding circumstances. For example, although parental divorce may appear to be independent of the adolescent, it cannot be conclusively established that adolescent rebellious behaviour did not influence his or her parents' conflict. Future research should consider the relative role of events independent of the adolescents' actions, using an interview, third party or established dichotomised measure. The ALEQ also did not contain sufficient items to separately consider interpersonal from achievement events. Studies should consider the known differences between girls and boys on the impact of different types of negative events, testing this model with each gender and each type of stressor (see Hankin & Abramson, 2001). Third, the partial-longitudinal design did not allow the most conservative test of mediation, as proposed by Cole and Maxwell (2003), nor a test of the direction of effects or of causation. Ideally, initial depressive symptoms, cognitive vulnerability factors, intervening life events and later depressive symptoms would all be assessed at separate time points, with auto-correlations controlled in the tested model (Cole & Maxwell, 2003). Future research could also consider third-party reports of stressful life events, to eliminate the possibility of shared-reporter biases

(Monroe & Simons, 1991). Additionally, experimental studies could help to establish the causal pathways between these factors, particularly by examining initial depressive symptoms and cognitive style, and subsequent behaviour in possibly causing negative outcomes in behavioural tasks, as well as considering the interaction of these factors in predicting later depression. Finally, the current study examined a community sample of adolescents, in an effort to capture a naturalistic range of depressive symptoms and those adolescents who may be developing prodromal symptoms of depression. School studies carry the unavoidable possibility of absenteeism, with depressed students potentially more likely to be absent (Fleming & Offord, 1990), possibly seen here with more depressed students more likely to be absent at Time 2. In this sample and age range, the variance in depressive symptoms over six months was relatively small, leaving little to be explained by the proposed model and adding weight to the fact that significant paths were found. Replication with clinical samples and diagnostic assessments would strengthen the proposed model.

Overall, the cognitive diathesis-stress generation pathway demonstrated here provides an important extension of our understanding of the development and maintenance of depression in adolescence. Integrating cognitive diathesis-stress and stress-generation theories, it was established that initial depressive symptoms and cognitive vulnerabilities may lead to the occurrence of stressful life events that are at least partly dependent on the individual's behaviour, which in turn contribute to increases in depressive symptoms over six months. The interaction of cognitive vulnerability and dependent stressors predicted further variance in depressive symptoms across time, suggesting that individuals who make negative attributions, tend to ruminate and behave in such a way as to generate further stressors are more likely to become depressed than individuals experiencing only one of these risk factors. Cognitive vulnerability factors, dependent stressors and their interaction all partly mediated the link between initial and subsequent symptoms of depression in young adolescents, potentially explaining both the development and maintenance of depression.

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CHAPTER FIVE

GENERAL DISCUSSION

Summary of Results

The study reported in Chapter Two demonstrated that the adolescent's perception of rejecting parenting by his or her mother was strongly associated with depressive symptoms, an effect partly mediated by its relationship with low self-worth. However, across time, only initial depressive symptoms accounted for significant independent variance in symptoms of depression 6 months later. This suggests that perceived parental support or rejection is a vital part of the adolescent's internal experience, possibly involved in the maintenance of depression along with negative self-beliefs. It is possible that a causal relationship between perceived parenting, self-worth and increased depressive symptoms would be seen earlier in childhood, when such cognitive and emotional states are first developing, and that these relationships are already established by adolescence. Unexpectedly, maternal depressive symptoms were not associated with adolescent symptoms nor with perceived parenting, possibly due to the low rates of depression in the community sample or to the self-report methodology. Consistent with the parenting literature (e.g. Bowlby, 1980; Garber, Robinson, & Valentiner, 1997), this study suggests that a lack of encouragement/praise or criticism from one's mother is associated with negative self-beliefs and depressive symptoms in offspring. However, it is important to emphasise that the adolescent's perception of parenting may not reflect actual parenting behaviour (e.g. Rapee, 1997). Importantly from a clinical perspective, this perception of parenting provides a vital insight into the adolescent's internal experience, such that the depressed adolescent with low self-worth may not seek their parent's support or reassurance, if they do not perceive it to be readily available. Clinical interventions should consider family environment factors and provide strategies aimed at improving the parent-child relationship.

The third chapter examined the role of neuroticism in the development of young adolescent girls' depression. Neuroticism appears to serve as a distal vulnerability factor for the development of depressive symptoms, predicting dependent stressful life events consistent

with stress-generation theory (Hammen, 1991). These dependent stressors fully mediated the association between neuroticism and depressive symptoms 12 months later, after controlling for initial depressive symptoms. Secondly, negative automatic thoughts about failure and loss partly mediated the relationship between dependent stressful life events and later depressive symptoms, following the elaborated cognitive vulnerability transactional-stress model (Hankin & Abramson, 2001). More neurotic adolescents appear more likely to experience stressful life events that are at least partly dependent on their own actions, and to interpret such events as personal failures, subsequently becoming more depressed. Initial depressive symptoms also influenced later symptoms partly through these same mediators, such that dependent stressors and cognitive interpreting style appear to partly explain the maintenance and worsening of depression itself. Interestingly, initial depressive symptoms were also associated with *independent* stressful life events 12 months later. Although it is possible that this is an artefact of the method, with adolescents reporting independent and dependent life events in the same measure at Time 2, it is unlikely to have been influenced by their depressive symptoms, as both Time 1 and 2 depressive symptoms were controlled in the model. It may be that depressed adolescents genuinely live in more disadvantaged environments (Sawyer et al., 2000), such that they might report both independent (e.g. parent losing a job) and dependent (e.g. poor school results, if the child was too busy helping at home to study) stressors.

The final study (Chapter Four) examined a possible convergence of cognitive diathesis-stress (Abramson, Metalsky, & Alloy, 1989) and stress-generation (Hammen, 1991) theories, specifying and testing a pathway suggested in Hankin and Abramson's (2001) elaborated cognitive vulnerability transactional-stress theory. Overall cognitive vulnerability, comprised of a tendency to make negative attributions and to ruminate, was included along with its interaction with dependent stressful life events, as potential mediators of the relationship between initial and subsequent depressive symptoms. This model was fully

supported, such that young adolescents with initial symptoms of depression were more likely to report a negative cognitive style, to generate dependent stressors and to report more subsequent depressive symptoms, with the relationship between initial and later depressive symptoms partly mediated by these factors. Adolescents who displayed a negative cognitive style and who generated dependent stressors were more likely to report increases in depressive symptoms than those who experienced only one of these factors, or than would be expected based on the sum of these two risks.

Theoretical Implications

The studies reported here carry important theoretical implications for our understanding of the development and maintenance of depressive symptoms in adolescence. Given the developmental factors highlighted in adolescence, it cannot be assumed that theories of depression in childhood or adulthood apply equally to young adolescents, or to the initial onset of depression (Roberts, 1999). Combining elements of previous theories, this thesis emphasises the role of perceived parenting, personality, negative automatic thoughts, attributional and ruminative tendencies and self-worth, along with dependent and independent stressors in predicting increases and the maintenance of depressive symptoms among young Australian adolescents. Broadly, it appears that more distal risk factors (perceived parental rejection, stressful life events and underlying personality style) are critically associated with cognitive factors (negative automatic thoughts, attributional style, rumination and self-worth) in contributing to depressive symptoms. Adolescents who report stressful events at least partly based on their behaviour (dependent stressful life events) are most affected by these when they also possess a negative cognitive style (hopelessness-style attributional tendencies, ruminative tendency). In turn, some forms of adversity such as rejecting parental style, also appear to increase the occurrence of a negative cognitive style. Moreover, adolescents with initial depressive symptoms are likely to perpetuate their experience of stress, generating dependent stressful life events, and possibly perceiving more rejecting parenting. It may be

that depressed adolescents' overall negative cognitive style leads them to misinterpret the behaviour of parents, family and friends, and to enhance negative interactions, as well as to underperform in academic and extracurricular contexts due to their negative cognitive framework. Rumination may exacerbate this effect (Watkins, Moberly, & Moulds, 2008), both by intensifying the cognitive focus on negative content (Nolen-Hoeksema, 1998), and by preventing the individual from undertaking pro-active coping strategies to resolve their difficulties, as they are preoccupied with negative thoughts (Nolen-Hoeksema, 1998; Watkins & Baracaia, 2002).

Importantly, not only were pathways to increased depression demonstrated, but the maintenance of depressive symptoms was also considered. Given that adolescents with greater depression were more likely to generate dependent stressors, to make negative attributions and to ruminate, to report negative automatic thoughts about failure and loss, to perceive parental rejection and to hold negative self-beliefs, this thesis provides an improved understanding of the continuation of depression from initial symptoms to chronic disorder. Previous models of depression in adolescence tend to focus on risk and causal pathways (e.g. Abela & Hankin, 2008; Metalsky & Joiner, 1992). Given that depressive symptoms in adolescence tend to be persistent (Birmaher et al., 1996; Kovacs, Akiskal, Gatsonis, & Parrone, 1994; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993), it is important to consider the pathways through which initial depressive symptoms may lead to chronic, recurrent and more severe depressive disorders in adolescence. The current body of work suggests several vital pathways in this regard. Firstly, following Hankin and Abramson (2001), the third model combines the possible causal path seen in diathesis-stress models, wherein individuals with a negative cognitive style are more at risk following stressful life events; with the circular or maintenance element of a stress-generation model wherein individuals at risk are more likely to report stressors that are at least partly dependent on their behaviour, leading to further increases in depression. Additionally, the second study suggests

that adolescents at risk are more likely to report (and/or to experience) independent stressors. Adolescents at risk of depression appear to engage in negative cognitive processes including depressogenic attributional and ruminative tendencies, a tendency to report automatic negative thoughts about failure and loss and to hold negative self beliefs. These adolescents are also likely to perceive that their mothers are rejecting and unsupportive. In addition to generating dependent stressful life events, it is possible that at-risk adolescents may be less likely to seek active solutions to their problems (Nolen-Hoeksema, 1998; Watkins & Baracaia, 2002), and possibly to seek less support or encouragement from parents they perceive to be less supportive.

Clinical Implications

In highlighting the significant impact of stressful life events in conjunction with psychological vulnerabilities for the development of depressive symptoms among young adolescents, the current thesis provides strong directions for the screening, prevention and early intervention of depression. Given the particularly poor prognosis for early onset depression (Lewinsohn et al., 1993), it is vital that interventions target the earliest risk factors predictive of depression (Roberts, 1999). With the aim of preventing prodromal adolescent depressive symptoms from developing into chronic depressive disorders, researchers and clinicians must work toward an improved understanding of early risk factors as intended in the current research.

Early risk factors for depression appear to include high neuroticism, low self-worth, and negative cognitive styles including attributional style, negative automatic thoughts and a tendency to rumination. Cognitive therapy should be directed at children displaying these tendencies, with the possibility of screening for vulnerability or of teaching “critical thinking” (cognitive restructuring) to school groups (e.g. Rooney et al., 2006). If young adolescents were able to think more realistically or positively about the occurrence of stressful life events, it might be that the translation of stressors into depressive symptoms could be interrupted. In

addition, adolescents at risk of depression appear to perceive their mothers as less supportive and caring, whether or not this reflects actual parenting behaviour. Nevertheless, it may be that these adolescents would be less likely to seek support from their parents. In this way, they may miss the opportunity to receive encouragement and praise that would bolster their self worth, and teach them to think more positively about themselves and their world. Interventions could be designed to target young adolescents and their parents, encouraging open and supportive family communication.

Finally, the repeated demonstration that young adolescents at risk of depression report more dependent stressful life events should be considered a priority. Whether directly based on cognitive biases (for example, misinterpreting a friend's comment and initiating an argument) or indirectly based on low mood (for example, lethargy and impaired concentration leading to poor school performances), intervention programs could target young adolescent behaviour directly. Consistent with the basis of cognitive-behavioural therapy (e.g. Clark & Beck, 1990), it appears that adolescents at risk of depression and displaying negative cognitive tendencies are more likely to behave in maladaptive ways, reporting more dependent stressors. As well as encouraging increased positive behaviour (behavioural activation, Jacobson, Martell, & Dimidjian, 2001), early intervention or prevention programs could teach pro-active problem solving skills, in an effort to counter ruminative/passive responses to stress, and to allow adolescents to take positive actions. In combination with cognitive therapy (e.g. Roberts, Kane, Thomson, Bishop, & Hart, 2003; Ryan, 2005), problem solving could encourage adolescents to make more helpful assessments of everyday events, and to take positive, instead of negative, actions.

Implications for parenting

Given that study one relies on the adolescents' perception of their mothers' parenting behaviour, it should be noted that one cannot assume that this accurately reflects the mothers' behaviour. However, whether through genuine maternal rejection or simply biased perception,

it is possible that depressed adolescents will not seek maternal support or assistance, if they do not believe it is readily available, as suggested by their reports of rejecting parenting. Future research should try to determine whether the mothers of depressed adolescents are genuinely rejecting, or whether this is a perceptual bias, as the two hold different implications for prevention. If actual maternal behaviours are involved, programs should focus on teaching parents new ways of interacting with their child. However, even biased perceptions could have serious ramifications for the adolescent's wellbeing, if the adolescent misinterprets caring, supportive parents and disengages from them, such that the parents are unable to either assist the adolescent or to direct them toward appropriate support services. Consistent with previous studies (e.g. Garber et al., 1997), the adolescent's perception of rejecting parenting is associated with a lower sense of self-worth. Adolescents who do not perceive (or possibly, receive) encouragement, praise and reassurance from their mothers appear to develop more negative self-beliefs, and possibly to miss the opportunity to learn to make more positive interpretations of negative experiences. Given that the present study associated both rejecting parenting and low-self worth with depressive symptoms in adolescence, it appears that these factors are a vital part of the adolescent's internal experience. However, actual maternal depressive symptoms were not associated with adolescent symptoms nor with adolescent self-worth, which might be due to the low overall levels of pathology seen in this community sample.

Implications for the role of cognition in the development of depression

The studies presented here provide strong evidence for the vital role of cognitive factors in the development of early adolescent depressive symptoms. Cognitive factors appear to mediate the relationship between more distal risk factors (perceived parenting, personality, stressful life events) and increases in depressive symptoms. Importantly, cognition was assessed in a variety of ways – considering overall self-worth or beliefs about oneself; a tendency to attribute negative events to internal, stable and global causes consistent with

Hopelessness Theory; a tendency to ruminate about one's depressive symptoms and their causes and consequences; and a tendency to engage in automatic negative thoughts about events reflecting personal failures. Comprehensive theories of depression suggest that multiple cognitive factors form vulnerabilities to depression, with slightly different possible pathways for each. Each factor was strongly associated with early adolescent depressive symptoms, and with stressful life events.

Negative Automatic Thoughts were considered here as the extent to which the respondent has thought of themselves as a failure, or attributed negative events to their own failings, in the preceding week (Schniering & Lyneham, 2007). These thoughts were strongly associated with young adolescents' level of neuroticism and depressive symptoms. In the proposed model, they mediated the link between dependent stressful life events and depressive symptoms – conceptualising a negative event as reflecting a personal failure (e.g. "It's my fault things have gone wrong"), overgeneralising ("I can't do anything right") and indicating hopelessness about the future ("I will never overcome my problems"). This cognitive style could also indicate an overall negative attributional or interpretive style (Schniering & Rapee, 2002). Consistent with the conceptualisation of neuroticism as a tendency to perceive the world as a negative, threatening place (Watson, Clark, & Harkness, 1994), the demonstration that more neurotic adolescents report negative automatic thoughts about failure and loss potentially explains part of the developmental trajectory between trait personality and state depression. In addition, the association between negative automatic thoughts and dependent stressors suggests that adolescents who make such negative judgements are also inclined to behave in a maladaptive manner, consistent with the tenets of cognitive-behavioural therapy (discussed above).

At a broader level, an overall negative cognitive style was conceptualised in the third study, encompassing attributions consistent with Hopelessness Theory (Abramson et al., 1989), along with a tendency to ruminate in response to low mood (Nolen-Hoeksema, 1998).

Comprehensive theories of depressive cognition (Hankin & Abramson, 2001) suggest that these two factors may operate together. (Hankin and Abramson (2001) also suggest that dysfunctional attitudes may form part of a broader cognitive vulnerability to depression, which was not assessed in the current study but should be considered in future research.) It has been suggested that while a negative attributional style would provide negative cognitive content (e.g. "I'm stupid, I'll always fail, I'll never be successful"), rumination facilitates the access to and activation of this content, thus exacerbating its effect on mood (Hankin & Abramson, 2001; Watkins et al., 2008). That this combined cognitive vulnerability served as a potential pathway between initial and subsequent depressive symptoms, in interaction with dependent stressful life events, suggests that this overall cognitive pattern serves to maintain and worsen depression in adolescence.

The process of rumination in response to low mood has garnered increasing empirical attention in recent years, both as a stand-alone vulnerability factor and in combination with stressors or other cognitive factors as demonstrated here (e.g. Nolen-Hoeksema, 1998; Watkins & Baracaia, 2001; Watkins et al., 2008). More broadly, initial research links an adolescent's tendency to ruminate with the same tendency in their parents (Kercher & Rapee, in preparation). In combination with the finding in Chapter Two that perceived parenting relates to self-beliefs, the question of shared parent-child cognitive style would be of great interest in future research.

As a reflection of overall thoughts about oneself, self-worth has strong and obvious associations with depression (Costello, Swendsen, Rose, & Dierker, 2008; Lewinsohn et al., 1994). In associating self-worth with the adolescent's perceptions of their mother as rejecting, the current study raises interesting questions about the possible pathways from parent mood and behaviour to child disorders. Although the association between maternal depressive symptoms and adolescent-reported perceived parenting was not significant in this community sample, previous clinical samples have demonstrated that depressed mothers are more

rejecting, critical and less supportive of their children (see Lovejoy, Graczyk, O'Hare, & Neuman, 2000 for a review). If these youths then come to internalise their mothers' negative feedback, it makes conceptual sense that they would develop a low sense of self-worth and subsequent depressive symptoms, as demonstrated here. Although the longitudinal pathway between perceived parental rejection and self-worth and later depression was not supported, it may be that in a clinical sample or across a longer timeframe (with more variance in Time 2 depressive symptoms), this relationship would be upheld. Nevertheless, the adolescents' perceptions of their mothers as rejecting or uncaring appears to be a vital component of their internal experience, with low self-worth forming a significant risk factor for depressive symptoms.

Life Events in Adolescent Depression

Overall, it appears that stressful life events have a considerable effect on the development of depressive symptoms in young adolescents, in combination with other vulnerabilities. As the consistent link between initial and subsequent depressive symptoms in studies 2 and 3, dependent stressful life events appear to play a vital role in the continuation and maintenance of depressive symptoms in adolescence. Whether driven by personality, initial depressive symptoms, cognitive style or all three, the generation of stressful life events that are at least partly dependent on one's behaviour appears to be a strong component of adolescent depression. Consistent with diathesis-stress models, dependent stressors appear most influential when combined with a vulnerable cognitive framework, a tendency to interpret negative events personally and to ruminate negatively on them.

Interestingly, study 2 highlighted links between independent stressors and prior depressive symptoms, contrary to stress-generation theories of depression which would suggest that these events will occur irrespective of one's level of depressive symptoms (Hammen, 1991). Several potential explanations could be made for this phenomenon, given also the demonstrated covariance between dependent and independent stressors. Firstly,

dependent and independent stressors were assessed within the same life events questionnaire. Psychometric studies suggest that respondents tend to show consistency within questionnaires, irrespective of content (Nunnally, 1978). However, in the CASE questionnaire, independent events should be difficult to misreport (Allen, 2005), as they are very concrete and objective (e.g. divorce or death items), a matter discussed by Brewin and colleagues (1993). Secondly, and perhaps more likely, this finding may reveal something of the adolescents' environments. Previous researchers have suggested that depressed adolescents have disadvantaged backgrounds (Sawyer et al., 2000). If these adolescents, for example, tend to live in negative environments such as impoverished or conflicted families, they could be exposed to both more independent (e.g. having to move house, parent unemployment) and dependent (e.g. social difficulties, school underperformance if time spent caring for siblings/working/helping at home) stressors. Finally, it is possible that the association between depressive symptoms and independent stressors may partly be due to chronic life events. Although the CASE was designed to assess acute rather than chronic stressors (e.g. "someone in my family died", "I did badly in an important test or exam", Allen, 2005), it does include items which could reflect ongoing sources of stress for adolescents (e.g. illness within the family), or present different yet related negative life events at both time points (e.g. parental illness at Time 1, recent parental death at Time 2; parental separation or conflict at Time 1, divorce at Time 2).

Developmental Considerations

Importantly, the current thesis provided longitudinal examinations of the development of depressive symptoms in early adolescence, a time of increasing social, academic and physiological challenges and of initial increases in depressive symptoms (Ge, Lorenz, Conger, Elder, & Simons, 1994; Roberts, 1999). Although adolescents in each sample were measured simultaneously during their first year of Australian high school, the timing of pubertal, developmental and social challenges can vary, with the possibility of hormonal and

physiological changes influencing mood and cognitive functioning (Angold, Costello, & Worthman, 1998). Future research should consider assessing pubertal timing or adrenarche, in examinations of depressive risk in adolescence. Further, the dramatic changes in social, academic, cognitive and physiological factors across adolescence suggest that models of risk for 12-13 year olds may not fit for older adolescents. While this was vital to the heuristic value of the current work, future research should also consider the manifestation of these risk factors at different ages and developmental stages.

Limitations

Several noteworthy limitations should be considered in designing future studies of this kind. Firstly, in each sample, there was a relatively small amount of variance between initial adolescent depressive symptoms, and those 6 or 12 months later. While this was to be expected in community samples of young adolescents (cf. Hankin, 2008), it left relatively little variance to explain, adding potency to the significant effects seen after controlling for initial depression. However, it may be that the pathways demonstrated would be even stronger among clinical samples of young adolescents, particularly with an increased likelihood of correlation between clinical parent- and offspring-depressive symptoms (e.g., Downey & Coyne, 1990; Sheeber, Davis, & Hops, 2002). It may also be that a longer time period, upwards of 12 months, would reveal further changes in depressive symptoms, and thus, better indicate early risk factors. However, this was beyond the scope of the current research, given significant and uncontrollable delays in arranging school participation. Further measurement points would also allow a more meaningful examination of potential risk pathways, allowing a fully longitudinal design and more conservative analyses consistent with the recommendations of Cole and Maxwell (2003). Experimental paradigms may also allow a closer examination of the potential causal pathways suggested here. The reliance of the current research on self-report measurements, necessitated by the large, community-based samples, may be a further limitation. The lack of observational measurement and independent

corroboration may limit the interpretation of the current findings, due to possible biased reporting by adolescents (see Monroe & Roberts, 1990; Rapee, 1997), although initial depressive symptoms were controlled throughout each model.

Future Directions

This research provides a strong foundation for the consideration of depressive risk trajectories in young adolescents. In applying strong conceptual theories to this age range, it has been demonstrated that young adolescents appear most vulnerable following stressful life events, particularly when combined with psychological vulnerabilities such as neuroticism, negative self-beliefs, negative attributional style, a tendency to ruminate and negative perceptions of parental support. Given the developmental changes occurring throughout childhood and adolescence, and the known increases in depressive symptoms in later adolescence, future research should replicate the demonstrated models in other age groups. The current research did not consider the known gender differences that emerge around early adolescence (e.g. Angold & Worthman, 1993; Hankin et al., 1998), partly due to limited numbers of participants, limited measures and the scarcity of father participants. Given the known differences between boys and girls in susceptibility to certain stressors and in relation to their parents (Hankin & Abramson, 2001; Nolen-Hoeksema, Larson, & Grayson, 1999; Sheeber et al., 2002), this will be a vital extension of the current work.

Concluding Remarks

Overall, the current thesis provides a strong insight into the developmental trajectory of depressive risk in early adolescence. From parenting to personality, attributions to self-worth to rumination, independent to dependent stressors, a range of developmental pathways were provided to explain both the development of prodromal depressive symptoms, and also the progression of early symptoms into chronic disorders. The majority of studies of depressive risk consider causal factors, but less often refer to the continuation of depression, which is a vital consideration given its chronicity and recurrence among adolescents. The overall

outcomes of the current research suggest that young adolescents with initial vulnerabilities (neuroticism, depressive symptoms, cognitive style) are at risk of generating more stressful life events, of negative cognitive interpretations and processing, and of ongoing depressive symptoms. If replicated across longer time periods, with clinical samples and with consideration of gender and pubertal status, the models demonstrated here will have substantial implications for the early identification of children at risk, and ultimately, provide important directions for the prevention of depression in adolescence.

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