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Developmental Origins of Cognitive Vulnerabilities to Depression: Review of Processes Contributing to Stability and Change Across Time

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Abstract

Cognitive theories of depression have been shown to be potent predictors of future increases in depressive symptoms and disorder in children, adolescents, and adults. This article focuses on potential developmental origins of the main cognitive vulnerabilities, including dysfunctional attitudes, negative cognitive style, and rumination. We selectively review processes and factors that have been hypothesized to contribute to the emergence and stabilization of these cognitive risk factors. This review focuses on genetic factors, temperament, parents and peers as salient interpersonal influences, and stressful life events. We end with suggestions for future theory development and research. In particular, we emphasize the need for additional conceptual and empirical work integrating these disparate processes together into a coherent, developmental psychopathological model, and we highlight the coexistence of both stability and change in the development of cognitive vulnerabilities to depression across the lifespan.

Depression is a substantial public health burden that shows dramatic increases from childhood into adolescence and adulthood. Rates of depression skyrocket from early to late adolescence (Hankin & Abela, 2005). Numerous etiological factors and processes have been proposed and studied to explain why individuals become depressed (Abela & Hankin, 2008a). Cognitive theories of depression are prominent etiological models that have been extensively studied in adults and are increasingly being applied to account for the development of depression across the lifespan (Hankin & Abela). Briefly, according to cognitive theories of depression, depressed and depression-vulnerable individuals are hypothesized to exhibit attention, interpretation, inferential, and memory biases for salient stimuli. The most prominent cognitive theories include Beck's cognitive theory (BT; Beck, 1987), hopelessness theory (HT; Abramson, Metalsky, & Alloy, 1989), and response styles theory (RST; Nolen-Hoeksema, 1991). Each theory highlights different cognitive vulnerabilities and processes hypothesized to contribute to depression. BT focuses on negative schema and dysfunctional attitudes, which are rigid and extreme cognitive structures that negatively filter and bias social and affective information. HT emphasizes a negative cognitive style, which comprises negative inferences about the cause of events (i.e., negative attributional style), self-implications, and consequences that individuals make in response to events. RST proposes rumination as a process of repeatedly focusing on symptoms of depression and the meaning, causes, and consequences of the symptoms.

Considerable evidence supports cognitive vulnerabilities from these main theories as prospective predictors of depression among adults, adolescents, and children. Both dysfunctional attitudes from BT and a negative cognitive style from HT moderate the longitudinal association between stressors and depression, consistent with the cognitive vulnerability-stress hypothesis of these theories, and baseline rumination has been shown to predict prospective elevations of depression (see Abela & Hankin, 2008b; Hankin & Abela, 2005; Lakdawalla, Hankin, & Mermelstein, for reviews). Despite considerable evidence supporting cognitive vulnerabilities and processes predicting depression, it is important to note that numerous factors and mechanisms contribute to the ontogeny of depression as it is a multifactorial disorder. As such, cognitive vulnerabilities are a contributory, but not a necessary, cause of depression, and so youth may become depressed without exhibiting cognitive vulnerability.

Given the successful predictive power of these central cognitive vulnerabilities in accounting for prospective increases in depression across the lifespan, investigators have increasingly turned attention to the potential developmental origins of these cognitive factors to understand when they emerge, consolidate, and stabilize into relatively enduring, trait-like risks to depression. The primary focus of this article is to review evidence for some of the processes and factors that have been postulated to contribute to the emergence and stabilization of cognitive vulnerabilities. In particular, we focus on genetic factors, temperament, parenting and peers as salient interpersonal influences, and stressful events and contexts (see Abela & Hankin, 2008b; Alloy, Abramson, Smith, Gibb, & Neeren, 2006; Ingram, 2001 for reviews).

Genetic Associations With Cognitive Vulnerabilities to Depression

Numerous studies have examined genetic influences on the development of depression, yet few have investigated associations between genes and cognitive vulnerabilities to depression. Hankin and Abramson (2001) hypothesized that genetic factors would operate as a distal risk to the development of cognitive vulnerabilities. Since then, both behavioral and molecular genetic studies in the last few years have found evidence supporting the association between genes and several cognitive vulnerabilities to depression (Hayden et al., 2008; Hilt, Sander, Nolen-Hoeksema, & Simen, 2007; Lau & Eley, 2008; Lau, Rijdsdijk, & Eley, 2006; Sheikh et al., 2008). These studies provide initial evidence suggesting that cognitive vulnerabilities may function as possible endophenotypes, or intermediate risk factors, connecting the distal etiological risk of genetics to the phenotype of depression.

Lau, Rijdsdijk, and Eley (2006) conducted the first study to investigate heritability of attributional style cross-sectionally in adolescent twin and sibling pairs. They found that the relationship between attributional style and depression had considerable genetic effects with heritability estimates ranging from .35–.47 for monozygotic (MZ) twins and .13 to .35 for other sibling pairs. Expanding on that initial study, Lau and Eley (2008) used a two-time point design to demonstrate genetic links between attributional style and depressive symptoms within each time point and across time. The authors replicated previous findings with heritability estimates in non-depressed MZ twins ranging from .40–.42 and other sibling pairs ranging from .15–.24 across the two time points. In sum, behavioral genetic studies show a moderate association between latent genetic factors and attributional style, although other cognitive risks have not been studied. Of note, behavioral genetic approaches cannot identify specific genes that may be associated with cognitive vulnerabilities to depression.

Recently, initial evidence identifying specific candidate genes associated with various cognitive vulnerabilities has emerged (Hayden et al., 2008; Hilt et al., 2007; Sheikh et al.,

2008). Hilt et al. found that rumination mediated the relationship between the Val/Val genotype of the brain-derived neurotrophic factor (BDNF) gene and depressive symptoms in adolescent girls. Variations in BDNF genotype are associated with worse performance on cognitive tasks of hippocampal functioning (e.g. memory tasks; Bath & Lee, 2006), and one feature of rumination involves biased recall of negative aspects of events. Therefore, the Val/Val genotype may be one genetic influence contributing to rumination. Two additional studies have examined the role of specific polymorphisms of the serotonin transporter promoter gene (5-HTTLPR) in cognitive vulnerabilities to depression in non-clinical samples of children (Hayden et al.; Sheikh et al.). Hayden and colleagues found greater biased recall of negative words in children possessing two copies of the S allele of the 5-HTTLPR genotype following a negative mood induction. Similarly, Sheikh and colleagues found that children who possessed either one or two copies of the S or L_G allele were more likely to make negative attributions for negative events than those homozygous for the long alleles.

Overall, these promising behavioral and molecular genetic studies provide burgeoning support for the idea that latent and specific genetic influences are associated with various forms of cognitive vulnerability, and cognitive risks may be an endophenotype of depression. However, caution is needed in concluding too much from these association studies, in particular, between candidate genes and cognitive vulnerabilities because the extant literature is based on small sample sizes (e.g., $N = 39$; Hayden et al., 2008). As these are early days in the investigation of genetic influences that may underlie cognitive vulnerabilities, it will be important for future research to replicate these initial findings. Additional research is needed to examine biologically plausible mechanisms through which candidate genes might be associated with cognitive risks.

Finally, investigating gene-cognitive vulnerability interplay will be important. First, are there gene-environment correlations (e.g., passive, evocative, active) that contribute to the development of cognitive vulnerabilities? For example, youth with susceptibility genes, which are associated with particular temperament traits, may evoke negative interactions from parents and peers. These negative social interchanges may be proximally involved with the emergence and formation of cognitive vulnerabilities to depression. Second, are there gene \times cognitive vulnerability or gene \times environment \times cognitive vulnerability interactions that predict the development of depression?

Temperament and its Relation to Depression and Cognitive Vulnerabilities

Individual temperamental traits, which are moderately heritable, are known to relate to depression in childhood and adolescence. Furthermore, temperament may play a role in the ontogeny of depression either directly or by increasing risk for the formation of cognitive vulnerabilities and other risk processes (Hankin & Abramson, 2001). Three major dimensions of temperament include negative emotionality (NE), positive emotionality (PE), and attentional control (AC; Compas, Connor-Smith, & Jasser, 2004). NE is conceptualized as an inclination toward experiencing intense discomfort, such as fear and anger, and reacting more easily to stress. PE is an inclination toward pleasure and reward from one's environment. Last, AC is defined as the ability to control emotions, behaviors, and focus.

Higher NE (Wetter & Hankin, 2009), lower PE (Wetter & Hankin, 2009), and lower AC (Davies & Windle, 2001) relate to depression in youth (see Compas et al., 2004 for a review). More recently, research has progressed past main effect models of temperament and depression to investigate moderation and mediation models. NE has been shown to predict increases in depressive symptoms through stress generation, whereas low PE indirectly predicted depressive symptoms through reduced social support (Wetter & Hankin,

2009). Finally, AC may moderate other temperament and depression associations (Compas et al., 2004).

Hankin and Abramson (2001) postulate that preexisting vulnerabilities, such as NE, increase the likelihood of experiencing negative events and forming cognitive vulnerabilities. Hankin and Abramson also hypothesized that once formed, cognitive vulnerabilities would interact with stressors, which may be generated as a result of particular temperamental traits, to predict later depressive symptoms. Consistent with these hypotheses, Lakdawalla and Hankin (2008) found in young adults that NE predicted subsequent increases in depressive symptoms through the mediating process of stress generation and cognitive vulnerabilities (dysfunctional attitudes and negative cognitive style) that potentiated the association between stressors and depressive symptoms over time. In addition, Mezulis, Hyde, and Abramson (2006) found that temperament, specifically negative withdrawal, moderated the relationship between stressors and cognitive vulnerability. Together these findings suggest that temperament may play an important role in developing cognitive vulnerabilities and point out the need for more complex models of the relationship among these factors.

Given theory suggesting that certain temperamental traits, especially NE, may contribute to the development of cognitive vulnerabilities, this raises the question of the distinctiveness of temperament, especially NE, and cognitive vulnerabilities as constructs. Factor analytic evidence among young adults suggests that NE is moderately associated with, but factorially distinct from, cognitive vulnerabilities (Hankin, Lakdawalla, Carter, Abella, & Adams, 2007). However, the necessary factor analytic research has not been conducted with child and adolescent samples. An important hypothesis to examine is the extent to which particular temperamental traits, especially NE, and cognitive vulnerabilities separate and become factorially distinct as individuals mature cognitively and emotionally (e.g., see Abela & Hankin, 2008b). It is unlikely that the relations among temperament, cognitive vulnerabilities, and depression are isomorphic across development, and examining these associations across childhood and adolescence can inform the developmental origins of cognitive vulnerabilities.

Origins and Stability of Cognitive Vulnerabilities: The Role of Interpersonal Factors

Cognitive vulnerabilities for depression may begin to stabilize during adolescence (Hankin & Abela, 2005), but research suggests that fluctuations can still occur together with stability (Hankin, 2008). Adolescence is a period in which interactions with peers begins to increase while relations with parents maintain importance (Steinberg & Morris, 2001). Therefore, parental influences may play a considerable role in the formation of cognitive vulnerabilities in childhood, whereas experiences with peers may strongly contribute to changes of these vulnerabilities during adolescence. Very little research however has investigated the role of peers in the maintenance and exacerbation of cognitive vulnerabilities. Additionally, most of the evidence for parental influences is based on correlating self-reports of parent and child measures of cognitive vulnerabilities. Although informative, observational studies are needed to elucidate which social factors are related specifically to different cognitive vulnerabilities and explicate the mechanisms through which these interpersonal influences may lead to the development and exacerbation of cognitive vulnerabilities over time.

Parental modeling and inferential feedback

One way in which parents may contribute to the formation of cognitive vulnerabilities is by modeling negative cognitions about their own parental behavior to their children when negative events occur. Children may then observe the inferences their parents make and,

over time, learn to make similar maladaptive cognitions. Support for this modeling hypothesis comes from studies showing a correlation between self-reported causal inferences, dysfunctional attitudes, and negative cognitions made by mothers and their children (see Alloy et al., 2006 for a review). Associations were not found in these studies between negative cognitive styles of fathers and their children, suggesting that the modeling of maternal cognitive styles, but not paternal cognitive styles, may play a role in the development of children's cognitive vulnerabilities. One hypothesis is that associations are stronger between children and their mothers' cognitive styles, as compared with their fathers, because mothers are typically primary caregivers, and, therefore, children have more exposure to the negative cognitions they make about their own parental behavior.

Another way parents may influence the development of cognitive vulnerabilities is by making negative attributions about their children's behavior after negative events (e.g. Fincham & Cain, 1986). In this variation of the modeling hypothesis, children internalize feedback communicated to them by their parents about the causes of events. Support for the inferential feedback hypothesis comes again from studies using self-report methods, specifically studies showing associations between self-reported inferences both parents make about their children and their children's own inferences about causes of events (see Alloy et al., 2006 for a review). Only one study (Mezulis et al., 2006) was located that employed observational methods to study the feedback hypothesis. They manipulated a math task so that all children received a poor score; then observational coders identified attributional inferences and negative statements mothers made about their children's performance during a parent-child discussion. Negative feedback from mothers during this task interacted with negative events to predict negative cognitive style, providing further support for the feedback hypothesis (Mezulis et al.). Future research would benefit from similar observational studies that clarify the role of fathers in the origin of cognitive vulnerabilities. Furthermore, research is needed on how both the gender of the child as well as the parent may influence cognitive vulnerabilities. There is some evidence that child depression is more strongly influenced by the behavior of the same-sex parent, possibly because of greater identification with that parent, but it is unknown whether this is also the case with child cognitive vulnerabilities (see Alloy et al. for a review).

Parenting styles

In addition to modeling and inferential feedback processes, general negative parenting may lead to the development of youths' negative cognitive styles. Parenting characterized by high levels of criticism, rejection, and control, as well as low levels of warmth and acceptance, may contribute to cognitive vulnerabilities (Beck, 1967; Blatt & Homann, 1992; Parker, 1983). A number of self-report studies support the link between negative parenting practices and cognitive vulnerabilities, including dysfunctional attitudes and negative attributional style (see Alloy et al., 2006 for a review). To date, no published observational study has examined associations between general parenting characteristics and cognitive vulnerabilities in children. We (Hankin, Oppenheimer, Flory, Maples, & Skiles, 2009) examined whether observed parental behaviors, such as authoritarian and critical parenting, coded from parent-child discussions, were associated with youths' self-reported cognitive vulnerabilities. Surprisingly, results showed no association between negative parenting and youth report of dysfunctional attitudes and cognitive style, although there was a significant association between negative parenting behaviors and depressive symptoms among youth. These findings suggest that more explicit communication of maladaptive cognitions, such as direct modeling and inferential feedback processes (e.g., Mezulis et al., 2006), may be necessary for the development of cognitive vulnerabilities, whereas general negative parenting alone may not be sufficient.

Although general negative parenting may not be associated with the development of explicit, self-reported cognitive vulnerabilities, evidence from our lab suggests that negative parenting is associated with biased information processing associated with risk to depression. Beck's theory of depression suggests that individuals who are depressed or vulnerable are likely to distort information and exhibit cognitive biases in information processing. In two independent samples of youth, we found that observed authoritarian and critical parenting was associated with youths' selective attention to angry faces assessed with an emotion-faces dot probe task (Hankin, Flory, Gibb, & Oppenheimer, 2009). This suggests that negative parenting may lead to particular biased information processing of socially relevant stimuli, which, in turn, may confer risk for depression.

Peer relationships

Very little research has examined the role that peers may play in the development of and changes in cognitive vulnerabilities. However, some studies suggest that peer experiences, such as rejection (Crick & Ladd, 1993; Panak & Garber, 1992) and peer victimization (Haines, Metalsky, Cardamone, & Joiner, 1999), may lead to the exacerbation of cognitive vulnerabilities. In a sample of young adults, Gibb, Abramson, and Alloy (2004) showed that peer victimization was associated with negative inferential style above and beyond parental maltreatment, and this association was largely due to victimization within romantic relationships.

Peer contagion factors may also contribute to cognitive vulnerabilities. Stevens and Prinstein (2005) found that the negative attributional style of reciprocal friends prospectively predicted changes in negative attributional style of adolescents. Associating with depressed peers may lead to the exacerbation of cognitive vulnerabilities because negative cognitions are reinforced and maintained through interactions with these peers.

Stressful Events and Negative Environmental Contexts

Maltreatment, such as abuse or neglect, is a severe environmental risk that may contribute to the development of cognitive vulnerabilities (Rose & Abramson, 1992). Children may attempt to understand the causes and consequences of maltreatment events, and when such events are recurrent and pervasive, they may be more likely to make depressogenic inferences about them. Over time, these children progress to form more general and stable negative cognitive styles. Research with young adults shows that those who experienced childhood maltreatment, especially emotional abuse, exhibit increases in depression. Cognitive vulnerability is one mediator of this relationship (e.g., Gibb, 2002; Hankin, 2005, see Alloy et al., 2006). Verbal victimization predicts prospective changes in negative attributional style among children (Gibb et al., 2006).

Less severe than maltreatment, negative life events predict depression, especially when moderated by cognitive vulnerabilities. In addition and consistent with a transactional model, greater stress exposure predicts changes in cognitive vulnerabilities. Among adolescents, greater stress levels predict more depressogenic attributions (Garber & Flynn, 2001), dysfunctional attitudes (Hankin, 2008), and the negative cognitive triad (Bruce et al., 2006).

The number of negative life events experienced by youth increases from childhood to adolescence (Ge, Lorenz, Conger, Elder, & Simons, 1994). Adolescence is a developmental period of marked transitions and stress across many domains (e.g., school transition, puberty, parental and peer support changes, dating, and sense of identity; Steinberg & Morris, 2001). A saturation effect has been hypothesized to occur culminating in adolescence, such that some adolescents experience an increase in stressors without

sufficient resources to cope with this increasing accumulation of events (Simmons, Burgeson, Carlton-Ford, & Blyth, 1987). One hypothesis, that has yet to be tested, is that the development of cognitive vulnerabilities may be an internal representation of experiencing accumulated negative events. Although the emergence of cognitive risk may occur at any age, it may not be until adolescence that cognitive vulnerabilities typically become manifest for those youth who have experienced a saturation of stressors. Providing some support for this notion, some research suggests that negative cognitive styles emerge in adolescence (Cole et al., 2008), whereas others found effects at younger ages. For example, Abela and Payne (2003) found that third graders were more pessimistic in their attributions than seventh graders and viewed themselves as more flawed following negative life events. This variation in results suggests that cognitive vulnerabilities to depression may emerge across childhood and adolescence rather than a specific, narrow age range. The age at which cognitive risks emerge may depend on youths' internalization of accumulating negative events over time.

Suggestions for Future Theory and Research

In conclusion, there are clearly several distal and proximal influences that contribute to the formation of cognitive vulnerabilities to depression. We close with final suggestions that we believe may advance knowledge of the developmental origins, emergence, and stabilization of cognitive vulnerabilities across the life course.

First, most of the empirical studies have focused on only one factor or process at a time to predict the emergence of one cognitive vulnerability in isolation of other known and possible mechanisms. Our review reflects this state of the empirical evidence. Yet, depression is a multifactorial disorder with numerous factors and processes that are posited and known to predict increases in symptoms and onset of disorder. It is most likely that explicating the emergence of cognitive vulnerabilities to depression will prove to be similarly complex and multidetermined with numerous processes transactionally contributing to their development and exacerbation throughout the life course. Additional conceptual work is needed to put forward explicit, developmentally sensitive, and integrative theories on the development of cognitive vulnerabilities based on developmental psychopathology principles and theoretical and empirical advances in genetics, brain development and function, hormonal changes, temperament, stressful environmental contexts, and social, cognitive, and emotional development. Moreover, considering how these and other influences affect formation of cognitive vulnerabilities for boys and girls, separately, will be important given sex differences in cognitive vulnerabilities and depression (Hankin & Abramson, 2001). Subsequently, more methodologically sophisticated empirical research can test these newer hypotheses. Knowledge on which processes are most predictive of emerging cognitive risks at different salient developmental periods can then be used to create relative risk profiles and multivariate prediction models. By expanding the theoretical and empirical scope of inquiry to include numerous risk processes, greater progress in understanding the developmental origins of cognitive vulnerabilities can be achieved.

Second, given that there are likely numerous factors and processes that contribute to the development of cognitive vulnerabilities across development, there is almost certainly some degree of stability and change in these processes and exposure to these environmental contexts (e.g., increasing salience of peers with transition from childhood into adolescence). With change and stability in the processes hypothesized to contribute to these cognitive factors, one would expect to find both continuity and change in the pattern of test-retest correlations of cognitive vulnerabilities across time. The available evidence is consistent with coexistence of both stability and change. For example, the average test-retest

correlation of negative cognitive style across four waves of data among early to middle adolescents was $r = .52$ (Hankin, 2008), and the average stability estimate in a daily diary study of negative cognitions made to daily stressors among young adults was $r = .38$ (Hankin & Abela, 2005). In both studies, structural equation modeling showed that there was an enduring, stable force that organized the pattern of test-retest correlations across time, and this finding is consistent with the interpretation that the dynamics giving rise to the test-retest correlations in cognitive risks are fairly trait-like and enduring. Yet, it is also clear from these studies that there is considerable change in cognitive vulnerabilities over time as well.

Evidence for both continuity and change in cognitive vulnerabilities is consistent with recent conceptualizations and evidence from the temperament and personality literatures. Research in these areas has concluded that traits, which had often been hypothesized and characterized to be stable across time and place (e.g., “set in plaster”; Costa & McCrae, 1994), likewise exhibit both continuity and change (Caspi & Roberts, 1999). For example, a meta analysis of the degree of stability for personality traits from childhood to old age revealed average test-retest correlations of $r = .31$ in childhood that increased to $r = .64$ during adulthood for major personality traits (Roberts & DelVecchio, 2000). Thus, cognitive vulnerabilities to depression, like temperament and personality traits, exhibit both continuity and change and demonstrate fairly similar stability estimates across different developmental periods. Moreover, it is likely that cognitive vulnerabilities, like personality traits, will show increasing stability estimates from childhood into adulthood while still exhibiting some change over time.

One implication that follows from the perspective that both continuity and change coexist and underlie the development of cognitive vulnerabilities is that there is no definitive endpoint at any particular age when cognitive vulnerabilities would be expected to finally emerge and stabilize into unchanging, trait vulnerabilities that are “set in plaster.” Indeed, asking the question of which factors and processes predict the developmental origins of cognitive vulnerabilities, which has been the aim of this article, seems to imply that there is an endpoint to the formation of cognitive vulnerabilities and that a specific set of mechanisms will be found that contribute to the final manifestation of cognitive vulnerabilities at some point in development. Yet, the conceptualization of cognitive vulnerabilities, like personality traits, as exhibiting both continuity and change across time reframes the scope of inquiry from searching for specific factors and processes that predict the ultimate formation of cognitive vulnerabilities, at some particular endpoint, into investigating various mechanisms that may contribute to both stability and change in cognitive risks at different points across development. The degree of stability in cognitive vulnerabilities likely increases with age, much as seen with personality traits, and there is change that coexists alongside this increasing continuity. Change in cognitive risks would be expected to be more rapid at younger ages, especially during childhood and early adolescence, but change would be anticipated, and likely varies across individuals, depending on the degree of variation in one’s experience, environment, and contexts across time. As individuals experience different positive or negative circumstances, levels of cognitive vulnerabilities would likely alter. Greater exposure to and accretion of these experiences likely would be required to produce change as individuals mature. Cognitions may worsen with accumulating negative experiences (e.g., experiencing ongoing intimate partner violence as an adult), or they may improve in response to positive experiences (e.g., efficacious psychotherapy). Clearly, various forms of effective psychotherapy (e.g., cognitive-behavioral therapy) exist that have been shown to ameliorate youths’ depression (Weisz, McCarty, & Valeri, 2006), and modifying youths’ cognitive vulnerabilities and enhancing positive experiences both appear to be processes through which efficacious psychotherapy work.

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