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Cognitive Theories of Depression in Children and Adolescents: A Conceptual and Quantitative Review

Zia Lakdawalla,[1] Benjamin L. Hankin,[2,3] and Robin Mermelstein[1]

This paper quantitatively reviews longitudinal studies examining three central cognitive theories of depression—Beck's theory, Hopelessness theory, and the Response Styles theory—among children (age 8–12) and adolescents (age 13–19). We examine the effect sizes in 20 longitudinal studies, which investigated the relation between the cognitive vulnerability–stress interaction and its association with prospective elevations in depression after controlling for initial levels of depressive symptoms. The results of this review suggest that across theories there is a small relation between the vulnerability–stress interaction and elevations in depression among children (pr = 0.15) and a moderately larger effect (pr = 0.22) among adolescents. Despite these important findings, understanding their implications has been obscured by critical methodological, statistical, and theoretical limitations that bear on cognitive theories of depression. The evidence base has been limited by poor measurement of cognitive vulnerabilities and over reliance on null hypothesis significance testing; these have contributed to a field with many gaps and inconsistencies. The relative paucity of research on developmental applications of such theories reveals that surprisingly little is known about their hypothesized etiologic mechanisms in children and adolescents. Ways to advance knowledge in the area of cognitive theories of depression among youth are discussed.

KEY WORDS: cognitive vulnerability; depression; stress; youth

INTRODUCTION

Depression is among the most common of psychological disorders, such that is has been called the ''common cold'' of psychopathology (Gotlib and Hammen, 2002). According to the World Health Organization, depression is the number one cause of disability, and will be the second most important disorder by 2020 in terms of burden of disease (e.g., disability and mortality) (Murray and Lopez, 1996). It has been estimated that between 5 and 25% of the population will experience depression at some point in their life, and up to 15% of severely depressed individuals will commit suicide (Gotlib and Hammen, 2002).

These inescapable facts are especially true for young people because depression rises dramatically with the transition from childhood through adolescence and then remains at high prevalence levels throughout much of adulthood. For example, a 10-year prospective longitudinal study showed that rates of depression rise sixfold during adolescence (Hankin *et al.*, 1998): approximately 2% of 13 year olds are depressed, and these rates sky rocket to 17% at age 18 (Angold *et al.*, 2002; Hankin *et al.*, 1998; Lewinsohn *et al.*, 1994; Wade *et al.*, 2002). In addition, sex differences in depression begin to emerge and expand throughout this time. Researchers from

[1]Department of Psychology, University of Illinois at Chicago, Chicago, IL, USA
[2]Department of Psychology, Barnwell College, University of South Carolina, Columbia, SC, 29208, USA
[3]To whom correspondence should be addressed; e-mail: hankin@sc.edu

many camps have conducted studies and proposed theories to explain and predict depression; however, much of the knowledge on vulnerability to depression has utilized adult theories of depression without a consideration of developmental differences. The developmental nature of depression highlights the importance of identifying the factors that confer vulnerability to depression in childhood through adolescence.

Cognitive theories of depression have been hypothesized as way one way to understand the developmental etiology and maintenance of depression. These theories share the general hypothesis that the ways in which individuals attend to, interpret, and remember negative life events contribute to the likelihood that they will experience depression. Considerable research has examined the etiology of depression centering around three seminal cognitive theories: Beck's theory of depression (BT; Beck, 1987), the Hopelessness theory of depression (HT; Abramson *et al*., 1989), and the Response Styles theory (RST; Nolen-Hoeksema, 1991). Each of these theories identifies distinct cognitive vulnerability factors (dysfunctional attitudes, negative cognitive style, and a ruminative response style, respectively) that are hypothesized to contribute to the onset and/or maintenance of depression. In the past, these theories have received much empirical and theoretical attention, providing overall support for the central role that cognition may play in depression in adult populations (see Abramson *et al*., 2002; Ingram *et al*., 1998; Scher *et al*., 2005, for reviews).

These theories have been extended downward to youth in order to understand the etiology and development of depression. This downward extension of adult theories has been an important preliminary step in understanding depression developmentally; however, several facts may jeopardize the utility of cognitive theories when applied to child and adolescent populations. For example, it is possible that children do not have the cognitive abilities that are posited to play a role in adult depression (Garber *et al*., 1993; Rutter, 1987). Also, the structure and nature of depression may differ in children and adolescents (Weiss and Garber, 2003) and therefore the causes and/or consequences of depression may vary across the lifespan. Finally, measures of cognitive vulnerability, which were developed, originally for adult populations, have been applied and used with youth, yet they may be poorly adapted for younger populations. Numerous studies have examined cognitive theories of depression among youth, but many have failed to consider these issues, and as a result, there exists mixed support for cognitive theories of depression in children and adolescents.

Given the downward extensions of adult cognitive theories of depression, and tests of these theories, to youth without a careful consideration of developmental differences, the present state of knowledge is replete with many gaps and inconsistencies. Therefore, a primary goal of this review is to examine the extent to which cognitive theories of depression apply to children and adolescents. To answer this question, we will review the literature evaluating key constructs proposed by cognitive theories and their power to predict depression in children and adolescents. Each theory will be evaluated by examining the magnitude of the effect sizes for the interaction of cognitive vulnerabilities with stress in the prediction of future elevations of depression.

A secondary goal of this review is to provide a basis for theory development. A review of this nature is needed to provide a more objective analysis of the extent to which these theories apply to younger populations, and this goal has been obscured by poor methodological and statistical strategies in past research. The majority of studies used inadequate sample sizes and relied on statistical significance tests that can complicate interpretations and theory appraisal (Hunter and Schmidt, 2004). Most studies have low statistical power to detect the relationships posited by cognitive theories of depression, yet the prevailing decision rule has been that if a finding is statistically significant, then a relationship exists; and if not, there is no relationship. The lack of power and this method of analyzing and interpreting individual studies can lead to false conclusions—that is, accepting the null hypothesis—and may account for a literature with seemingly mixed support for cognitive theories.

Moreover, this practice may contribute to potentially unnecessary revisions of theory, based on the premise that equivocal and conflicting data need to be reformulated into newer theories, especially if the mixed evidence is based upon studies using null hypothesis significance testing in which the null hypothesis is incorrectly accepted. For example, a handful of studies have found that the interaction between a negative attributional style (Abramson *et al*., 1978) and stress was not statistically significantly associated with depressive symptoms in children (e.g., under 5th grade), whereas this cognitive vulnerability–stress interaction was observed to be

statistically significant starting in early adolescence (e.g., Nolen-Hoeksema *et al*., 1992; Turner and Cole, 1994). Based on this pattern of findings (i.e., no significant association in children, but a significant interaction in adolescents), which is grounded in null hypothesis significance testing, some theorists have put forward theoretical revisions to cognitive theories of depression to account for the lack of significant findings in children, and a significant finding in adolescents (e.g., Cole and Turner, 1993; Gibb and Coles, 2005). These theoretical reformulations may be accurate and represent an important advancement, yet the logical basis upon which such revisions are founded is flawed logically because such theoretical modifications are based on the practice and acceptance of the null hypothesis without knowledge of effect sizes. As Meehl argued (1978), theoretical revisions made on the basis of null hypothesis significance testing slow theoretical and empirical progress in psychological science. As such, it is imperative to know actual effect sizes at different ages to enable more rigorous empirical evaluations of the cognitive theories of depression as they relate to the development of depression among youth. The present review underscores the need to appraise cognitive theories by examining sizes of effect across studies and aggregating results derived from single studies to reveal underlying patterns of relations put forward by cognitive theories in order to provide a cumulative knowledge that comprehensively addresses scientific questions. Thus, with this review we aim to advance theory and knowledge on the role that cognitive factors and processes may play in the ontogeny of depression across development.

Given the prominence of cognitive theories of depression as explanations for the etiology of depression, it is surprising that the knowledge basis among youth has not been systematically and quantitatively reviewed for at least a decade. The last empirical syntheses of the evidence base among children and adolescents were conducted by Gladstone and Kaslow (1995) and Joiner and Wagner (1995). These reviews employed meta-analytic techniques to examine the relationship between cognitive vulnerability and *concurrent* levels of depression symptoms. These papers convincingly established that attributional style, increased negative cognitions about the self, and hopelessness, are correlated concurrently with depression in children and adolescents. However, the studies included in these meta-analyses linking such variables to depression employed cross-sectional designs. Cross-sectional designs lack the methodological strengths needed to establish temporal precedence and differentiate between causes, correlates, and consequences of depression (Barnett and Gotlib, 1988; Kraemer, 2003). As a result, at the time that these reviews were conducted, fewer studies had tested the *vulnerability–stress components* of cognitive theories of depression, and as such there was no strong evidence supporting this tenet of the theory. In addition, most of the studies in these reviews focused on one cognitive theory—HT—and evaluated only the attributional style composite, even though theory specifies three distinct inferential styles (for the self, causes, and consequences). In the past decade since these prior reviews, the research testing cognitive theories of depression has undergone considerable methodological, and theoretical advancements. For example, scientists in this area have conducted more rigorous tests of these theories by employing longitudinal prospective designs that control for initial levels of depression and include stress in order to evaluate the vulnerability–stress aspect of the theories. Thus, the current review integrates the latest research that provides the most powerful tests of cognitive theories' vulnerability–stress hypothesis. It is important and timely to review quantitatively the evidence concerning cognitive theories of depression among children and adolescents. We believe this review may advance knowledge in the field and may provide valuable information against which investigators can appraise the utility of cognitive theories of depression in their current formulations. The results from this review can provide information about possible modifications and revisions of these theories, if needed, to make them more developmentally sensitive to children and adolescents.

THE CURRENT REVIEW

In order to place the data derived from research on cognitive theories of depression within a theoretical context, we begin each section first with a brief conceptual review of the particular cognitive theory of depression and its primary hypotheses. We review each theory in the chronological order, according to the date that each was formulated; to reveal the number of published studies relative to the age of the theory. Next, we review quantitatively studies that have empirically examined the interaction of each form of cognitive vulnerability with stress to predict symptoms of depression over time after initial depressive symptoms were controlled. We report

effect sizes separately for children and adolescents whenever possible to evaluate the empirical status of these theories in youth at different ages. Finally, we discuss the implications for the cognitive depression theories and suggest avenues for enhancing future research.

In each section, we provide a brief review of the evidence for each cognitive theory among adults. We did this because we believe that it is important to have a sense of the degree of empirical support for a particular cognitive theory among adults given that each of the cognitive theories reviewed here were formulated originally with adults and have been studied most extensively with adults. Understanding the research base underlying a particular cognitive theory in adults provides a framework with which to evaluate the scope of support in children and adolescents that may be expected given the degree of support found in adult studies. Also, evaluating the evidence in adults first enables scientists to begin to elucidate how well a cognitive theory is supported by evidence across the lifespan, and as a consequence, informs the field whether any potential changes are needed to the theory overall across the lifespan or, alternatively, whether particular age-specific modification to theory are needed given plausible developmental differences. For example, if the evidence base shows that no support is found for a theory among children, whereas support is obtained among adolescents and adults, then this pattern has clear implications for the relevance of the theory, and may inform any modifications that may be needed to make the theory developmentally appropriate, if possible. On the other hand, if the preponderance of evidence across child, adolescent, and adult samples is relatively equivalent, then this suggests that the theory may be equally applicable across development and that any revisions, if needed, may be required across the lifespan and not to a particular age.

Originally, we intended to provide a meta-analytic review of cognitive theories of depression among youth; however, we were unable to conduct a rigorous meta analysis because the vast majority of identified studies did not lend themselves to meta-analytic techniques. More specifically, studies omitted essential information (e.g., beta weights, *t* values, and standard errors) in their results; this, in effect, precluded the computation of average effect sizes across studies for the interaction of cognitive vulnerability and stress. Therefore, the present review of the literature was conducted in a quantitative manner. We evaluated the meaningfulness of findings derived from empirical studies testing cognitive theories on the basis of sizes of effect rather than levels of significance, which are less affected by sample size.

We selected the partial correlation (pr) and standardized beta weight as the index of effect size because these were the most commonly reported statistics and are easily compared across studies. We used Cohen's (1988) criteria for small (pr = 0.10), medium (pr = 0.30), and large (pr = 0.50) effects and considered correlations less than 0.05 to be trivial. In cases where neither the partial correlation nor the beta weight was reported, a range of correlations was derived based on the probability value obtained and the number of subjects in the study.

This review only included studies that followed several methodologically rigorous criteria to provide the strongest test of cognitive theories of depression in children and adolescents. First, we confined our search to only prospective studies because, as mentioned previously, it is not possible to differentiate between causes, concomitants, and consequences of depression using cross-sectional data. Second, only studies that had follow-up periods of 2 weeks or greater were included in order to differentiate between initial reactions of negative affect and reasonably enduring symptoms of depression following negative life events (Hankin and Abramson, 2001). Third, only studies that controlled for initial levels of depression were included, because only this type of analysis can help to establish temporal precedence, and differentiate risk factors from concomitants or consequences of depression.

In order to locate studies adhering to these criteria that examined Beck, Hopelessness, and Response Styles theory in child and adolescent populations, computerized literature searches were conducted using PsycInfo and Web of Science for the years 1980–2005. The keywords used in this search were: depression, hopelessness theory, Beck's theory, dysfunctional attitudes, attributional style, cognitive style, explanatory style, rumination, response styles, children, prospective, adolescents, clinical, high-risk, and youth. The products of these searches were reviewed and pertinent articles were identified. In addition, we examined the reference sections of all identified articles to ensure that we did not miss any published studies in this area.

Before reporting the results, we wish to note that the our quantitative review provides a very conservative evaluation of cognitive theories of depression because many of the studies included in this analysis

incorporated other constructs proposed to be involved in the development of depression. Therefore, instead of examining the interaction of cognitive vulnerability with stress exclusively, as many of the cognitive theories originally postulated, other variables (e.g., self-esteem, social support) were also included in some studies. Consequently, it is possible that these other variables, which were not explicitly posited in cognitive theories of depression, may have accounted for depression-predicted variance that otherwise may have been associated with the constructs posited in cognitive theories of depression (e.g., dysfunctional attitudes and stress). As a result, the effect sizes for the cognitive vulnerability stress interactions may be under-estimated more than they would have been otherwise if these other variables had been excluded from the analysis.

BECK'S COGNITIVE TTHEORY OF DEPRESSION

Background and Evidence Among Adults

In Beck's cognitive theory of depression (BT; Beck, 1967, 1987), maladaptive self-schemata that include dysfunctional attitudes, involving themes of loss, inadequacy, failure, and worthlessness, constitute the cognitive vulnerability. These schemata consist of stored bodies of knowledge that affect encoding, comprehension, and retrieval of information. Consistent with vulnerability–stress models of depression, these dysfunctional attitudes are hypothesized to become activated following the occurrence of a negative life event, generating specific negative cognitions (e.g., negative thoughts about the self, world, and future), and lead to elevations of depressive symptoms. Depression arises as a result of inferences derived from distorted cognitions and schema-driven processes, whereas the inferences arrived at in nondepressives are based on relevant situational information.

Given that BT is a vulnerability–stress theory, without the occurrence of stress, individuals who possess depressogenic self-schemata are hypothesized to be no more likely to become depressed than those who do not possess such schemata. In addition, Beck posits that these self-schemata are typically latent in individuals vulnerable to depression and must be activated by a relevant stressor to trigger biased information-processing tendencies. The activation of the schema subsequently influences how the individual perceives, encodes, and retrieves information regarding the negative life event. Conversely, in the absence of stressful events, depressogenic self-schemata are hypothesized to remain inactive and not exert significant influence on cognitive processing. Taking these together, an adequate test of the etiological chain posited in BT requires that individuals' depressogenic schemata be assessed prior to the occurrence of stress and the onset of depressive symptoms in order to examine whether the schemata interacts with negative events to predict elevations in depressive symptoms.

Studies examining BT in adults using longitudinal designs have yielded mixed support for the theory (see Hankin and Abela, 2005; Scher *et al.*, 2005). Whereas the majority of studies have found support for the hypothesis that dysfunctional attitudes interact with negative life events to predict the onset and maintenance of depression (e.g., Brown *et al.*, 1995; Hankin *et al.*, 2004; Joiner *et al.*, 1999), other studies have found mixed support (e.g., Dykman and Joll, 1988; Voyer and Cappeliez, 2002), and some have found no support (e.g., Barnett and Gotlib, 1988; Kuiper and Dance, 1994). Thus, the evidence base in adult populations appears to generally support BT, but the extant evidence is not entirely supportive.

Empirical Evaluation of BT in Younger Populations

Whereas BT has been widely studied in adult populations, the research examining this theory in younger populations has lagged far behind. Only two studies with prospective designs were identified (see Table I).

In the first, Abela and Sullivan (2003) examined dysfunctional attitudes and stress within the context of high and low levels of self-esteem and social support in a community sample of seventh graders during a six-week interval. To place children in high and low social support and self-esteem groups, children were split dichotomously with children scoring at the median or higher placed in the high group and the remainder in the low group. Analyses were conducted separately for each of these groups (i.e., high social support, low social support, high self-esteem, low self-esteem). Significant interactions for two out of four vulnerability–stress interactions were found (high social support and high self-esteem) and results were provided only for these interactions. Results revealed that dysfunctional attitudes predicted depressive symptoms following the occurrence of a negative life event among children who possessed high levels of self-esteem and social support. To

Table I. Summary of Studies Testing Beck's Theory

Study	Sample Type	Follow-up	Vulnerability Measure	Stress Measure	Depression Measure	Variables in regression Analysis	*N*	*p*	Effect Size (pr)
Abela and Sullivan (2003)	Preadol	1.5 months	CDAS	CHAS	CDI	CDAS×stress (high social support)	184	< 0.05	–0.232
						CDAS×stress (low social support)		ns	
						CDAS×stress (high self-esteem)		< 0.05	–0.205
						CDAS×stress (low self-esteem)		ns	
Lewinsohn et al. (2001)	Adol	12 months	DAS	Modified LES and SRE	KSADS, LIFE	DAS×stress	1507	< 0.0859	β = 0.0234

Note: DAS, Dysfunctional Attitudes Scale; CDAS, Children's Dysfunctional Attitudes Scale; CDI, Child Depression Inventory; CHAS, Children's Hassle's Scale; K-SADS, Kiddie Schedule for Affective Disorders; LIFE, Longitudinal Interval Follow-Up Evaluation; LES, Life Events Schedule; SRE, Schedule of Recent Experiences. Preadol = Preadolescent (grade 7, age 12–13), Adol = Adolescent (ages 13–19)

explain these findings, Abela and Sullivan (2003) suggested that children with low levels of self-esteem and social support may have chronic negative perceptions of themselves and their relationships with others, resulting in little room for lability in these perceptions as compared to children in high social support and self-esteem groups. Due to the limited availability of statistical information, we derived partial correlations based on the probability value obtained and the number of subjects in the study, which are provided in Table I for significant interactions. These findings indicate that a moderate effect for the vulnerability–stress interaction may be present in preadolescents, but these are limited to individuals with high levels of social support and self-esteem.

In the second study Lewinsohn *et al.* (2001) reported results from the Oregon Adolescent Depression Project (Lewinsohn *et al.*, 1993), a representative sample of community adolescents examining the development of depression during a one-year interval. This study included a number of covariates (current depression, the presence of a nonmood disorder both prior and during the course of the study, and family history of depression and nonmood disorder) in order to provide a very conservative test of Beck's cognitive theory of depression. Whereas the analyses supported BT at the level of a trend, the obtained size of effect for the dysfunctional attitudes–stress interaction was negligible (pr = 0.0421).

Discussion and Recommendations

At the present time, no decisive comments can be made regarding the role of dysfunctional attitudes in depression in younger populations as too few studies have been conducted. Research by Abela and Sullivan (2003) seems to suggest that dysfunctional attitudes may play a role in the development of depression, but much more research is needed to examine this hypothesis. In addition, Lewinsohn and colleagues' (2001) findings provide a very conservative test of Beck's etiologic chain, as many potent predictors of depression were controlled in their analyses. Taking this into account, the size of effect for the interaction term, despite being quite small, adds incremental variance to understanding the development of depression in adolescence. Taken together, the inquiry into the role of dysfunctional attitudes and stress in the prediction of depression appears to be a promising avenue for research, but more research is needed to evaluate the basic tenets of

BT to understand whether and how the vulnerability–stress interaction functions in younger populations.

As research examining BT among youth accumulates a larger evidence base, it is important to consider some theoretical and methodological factors that may influence future directions for research. It may be that at these young ages other factors may be more robust and consistent predictors of elevations of depression. Interestingly, across both studies the magnitude for the main effect of stress was larger than the interaction of stress and dysfunctional attitudes or the main effect of dysfunctional attitudes. This finding is consistent with research and theory suggesting that at younger ages, before cognitive patterns have developed into stable, trait-like styles, depressive symptoms may be a direct result of reactions to current circumstances and stress (Shirk, 1988). Alternatively, it is possible that the small sizes of effect are a result of methodological limitations and an inadequate test of BT. Beck hypothesized that depressogenic schemata remain latent until activated by a negative life event or negative mood; therefore, more compelling results may come from studies that activate depressogenic schemata prior to their assessment (e.g., Taylor and Ingram, 1999). Future research should examine this possibility by conducting prospective studies that directly compare the predictive ability of primed versus unprimed schema in the development of depression in order to provide a stronger test of BT.

In addition to these concerns, a common critique of the downward extension of cognitive theories of depression to younger populations is how cognitive vulnerabilities are assessed, because most studies have tended to use age inappropriate measures with poor psychometric properties. Directly addressing this issue, Abela and Sullivan (2003) constructed a new measure to assess dysfunctional attitudes in children and included an analysis of its psychometric properties. The Children's Dysfunctional Attitudes Scale (CDAS) represents an age appropriate measure for the assessment of dysfunctional attitudes with high reliability ($\alpha = 0.92$) and validity (Abela and Sullivan, 2003) and provides a good foundation for research to examine BT using a more developmentally and age-appropriate measure. For example, we have recently completed a longitudinal study using the CDAS in a 4-wave prospective design with youth (6th–10th graders; $n = 320$ who reported on levels of depressive symptoms and occurrence of stressors every month for 4 months. Briefly, longitudinal analyses showed that the interaction of CDAS at baseline with stressors over the four time points predicted prospective elevations of depressive symptoms ($b = 0.027$, $t = 6.03$, $p < 0.001$; Hankin *et al.*, 2004). It is recommended that future research conducted in this area make use of this measure, in order advance knowledge on BT in a systematic manner.

In sum, at present there is simply not enough published research evidence with youth on BT to make any conclusive remarks regarding its application in younger populations. More research is needed to evaluate the basic tenets of BT in child and adolescent populations before inferences can be drawn regarding the strength of Beck's hypothesized etiologic chain.

HOPELESSNESS THEORY OF DEPRESSION

Background and Evidence Among Adults

Hopelessness theory (HT; Abramson *et al.*, 1989), a revision of the reformulated helplessness theory of depression (Abramson *et al.*, 1978), posits that some individuals exhibit a more depressogenic inferential style, and when confronted with a negative life event, are likely to develop symptoms of depression. Although the theory allows for other possible pathways to the development of depression (e.g., genetics, neurotransmitters, etc.), it postulates three types of negative inferences that individuals can make given the occurrence of negative events: causal inferences (inferences about why the event occurred including stable and global attributions), inferred consequences (inferences about what will result from the occurrence of the event), and inferences about the self (inferences about the self with respect to the event that occurred). Further, making such inferences increases the likelihood of developing hopelessness, and in turn, depression because hopelessness is posited to be a proximal sufficient cause of hopelessness depression, a theory-based subtype of depression.

The majority of studies examining HT in adults have provided support for the vulnerability–stress interaction (e.g., Alloy and Clements, 1998; Hankin *et al.*, 2004, studies 1, 2, and 3; Metalsky and Joiner, 1992, 1997; Reilly-Harrington *et al.*, 1999). In addition, the majority of studies examining HT's symptom component have provided support for the unique symptom profile (e.g., Alloy *et al.*, 1997; Metalsky and Joiner, 1997; Whisman and Pinto, 1997).

Table II. Summary of Studies Testing Hopelessness theory

Study	Sample Type	Follow-up	Vulnerability Measure	Stress Measure	Depression Measure	Variables in Regression Analysis	*N*	*p*	Effect Size (pr)
Abela and Payne (2003)	Child	1.5 months	CCSQ, CASQ	CHAS	CDI	CASQ weakest×stress (for girls)	314	ns	0.01
						CASQ weakest×stress (for boys)		ns	0.09
Abela and Sarin (2003)	Child	2.5 months	CASQ, CCSQ	CLES	CDI non-HD	CASQ GEN×stress	79	ns	0.153
						CASQ CONS×stress		ns	−0.172
						CASQ SELF×stress		ns	−0.138
						CASQ weakest×stress		ns	0.1
					CDI HD	CASQ GEN×stress		ns	0.232
						CASQ CONS×stress		ns	−0.088
						CASQ SELF×stress		ns	0.12
						CASQ weakest×stress		<0.01	0.386
Abela and Seligman (2000)	Adol	0.25–2 months	CSQ	University admission	MAACL	CSQ-SELF×stress	77	<0.001	0.303
						CSQ-CONS×stress		<0.001	0.376
						EASQ-GEN×stress		<0.001	0.387
Abela and Seligman (2000)	Adol	0.25–2 months	CSQ	fraternity/sorority admission	MAACL	CSQ-SELF×stress	77	<0.05	0.258
						CSQ-CONS×stress		<0.05	0.299
						EASQ-GEN×stress		<0.01	0.317
Abela (2001)	Child	1.5 months	CCSQ, CASQ	CLES	MAACL	CASQ×stress (7th grade)	382	<0.05	0.26
						CSQ-CONSx stress (3rd and 7th grade)		<0.05	0.14
						CSQ-SELFx stress (3rd and 7th grade)		<0.05	0.16
Conley *et al.* (2001)	Child	0.5–1 months	CASI, CASQ-R	DHQ	CDI	CASQ×stress×age	147	ns	0.12
						CASI×stress		ns	0.011
						CASI×stress×age		<0.05	−0.182
Dixon and Ahrens (1992)	Child	1 month	KASTAN-R	CDCEQ	CDI	KASTAN×stress	84	<0.05	0.2344
Hammen *et al.* (1988)	mixed	6 months	CASQ	Contextual Threat interview	K-SADS	CASQ×stress	79	ns	$R^2 = 0.32$
					CDI	CASQ×stress		ns	$R^2 = 0.16$
Hankin *et al.* (2001)	Adol	1.25 months	CASQ	APES	BDI	CASQ×stress	270	<0.01	0.16
					HDSQ-R	CASQ×stress		<0.001	0.23
					Non-HD	CASQ×stress		ns	0.08
					HD	CASQ×stress		<0.01	0.18
Joiner (2000)	Mixed[a]	2 months	CASQ	CAI	CDI	CASQ×stress	34	<0.05	0.43
Lewinsohn *et al.* (2001)	Adol	12 moonths	CASQ	Modified SRE and LES	KSADS, LIFE	CASQ×stress	1507	<0.0162	$\beta = -0.0875$
Nolen-Hoeksema *et al.* (1986)	child	12 months	CASQ	LEQ	CDI	CASQ×stress	168	0.06, 0.001	0.195–0.164
Nolen-Hoeksema *et al.* (1992)	Child	60 months	CASQ	LEQ	CDI	CASQ×stress	336	<0.01	
Panak and Garber (1992)	Child	12 months	CASQ	Peer rejection	CDI	CASQ×stress	512	<0.01	−0.14

Table II. Continued

Study	Sample Type	Follow-up	Vulnerability Measure	Stress Measure	Depression Measure	Variables in Regression Analysis	*N*	*p*	Effect Size (pr)
Prinstein and Aikens (2004)	Adol	17 months	CASQ	Peer rejection	CDI	CASQ-N×stress	158	< 0.001	β = −0.41
Robinson *et al.* (1995)	Child	4–5 months	CASQ	Combination of validated stres s measures	CDI	CASQ	371	ns	−0.009
Southall and Roberts (2002)	Adol	3.5months	CASQ	LES	CDI	CASQ×stress (whole sample)	115	ns	−0.05
						CASQ×stress (low initial depression)		ns	0.04
						CASQ×stress (high initial depression)		ns	−0.03
Spence *et al.* (2002)	Adol	12 months	CASQ-R	NLE	BDI	CASQ×stress	773	ns	β = −0.06

Note: CASQ, Children's Attributional Style Questionnaires; CASQ-R, Children's Attributional Style Quesionnaire—Revised; CCSQ, Children's Cognitive Style Questionnaire; CPQ, Cognitive Priming Questionnaire; CSQ, Cognitive Style Questionnaire; CASI, Children's Attributional Style Interview; CDI, Child Depression Inventory; BDI, Beck Depression Inventory; MAACL, Multiple Adjective Affect Checklist; K-SADS, Kiddie Schedule for Affective Disorders; LIFE, Longitudinal Interval Follow-Up Evaluation; EASQ, Extended Attributional Style Questionnaire; DAC, Depressive Adjective Checklist; CES-DC, Center for Epidemiological Studies Depression Scale for Children.
Child = Ages 8–12, Adolescent = 13–19.
[a]Clinical sample.

Empirical Evaluation of HT in Younger Populations

Of the three cognitive theories evaluated in this review, HT has received the most empirical attention among younger populations. Eighteen studies examining this theory in child and adolescent populations were identified (see Table II). Nine of these studies were conducted in child populations (age 8–12; Abela and Payne, 2003; Abela and Sarin, 2003; Abela, 2001; Conely *et al*., 2001; Dixon and Ahrens, 1992; Nolen-Hoeksema *et al*., 1986; Panak and Garber, 1992; Robinson *et al*., 1995; Nolen-Hoeksema *et al*., 1992), seven in adolescent populations (age 13–19; Abela and Seligman, 2000 (two studies); Hankin *et al*., 2001; Lewinsohn *et al*., 2001; Prinstein and Aikens, 2004; Southall and Roberts, 2002; Spence *et al*., 2002) and two used mixed child and adolescent samples (Hammen *et al*., 1988; Joiner, 2000). Only 17 studies are included in this discussion as the effect size for the cognitive vulnerability–stress interaction was not reported in one study and the available statistical information precluded the derivation of a parameter estimate (Nolen-Hoeksema *et al*., 1992).

Overall the average magnitude of the effect size for studies examining HT in both child and adolescent populations was in the small range. However, effect sizes were larger for studies using adolescent samples versus child samples. The magnitude for the average effect for the interaction of inferential style with stress predicting depression in child population was in the small range (pr = 0.15), whereas for adolescent populations the average effect size for the interaction term was relatively larger (pr = 0.22). Across both child and adolescent samples, the larger sizes of effect were seen in those studies that used the symptoms profile for hopelessness depression as the predictor variable (Abela and Sarin, 2003; Hankin *et al*., 2001), examined the three inferential styles in isolation rather than as an aggregate (Abela and Sarin, 2003), used a semi-structured interview to assess inferential style rather than child self-report questionnaire (Dixon and Ahrens, 1992), used diagnoses of depression via semi-structured interviews as the outcome (Hammen *et al*., 1988), and utilized a clinical sample (Joiner, 2000). At the same time, not all findings were consistent with these magnitudes as negligible sizes of effect were also found in studies using the symptoms profile for Hopelessness depression as a predictor variable (Abela and Payne, 2003), and when diagnoses of depression via semi-structured interview were used as the outcome (Lewinsohn *et al*., 2001).

For those studies using mixed child and adolescent samples the average magnitude for the interaction term was medium (pr = 0.30). Also, these studies used more rigorous methodologies, and this may have produced the larger effect sizes. The first (Hammen *et al*., 1988) made use of both mothers and children as sources of information about children and adolescents' negative life events and had teams of independent judges evaluate the objective event apart from children's own subjective report. This ‚contextual threat' method of evaluating stress (Brown and Harris, 1978) provides a more stringent approach to the examination of vulnerability–stress models. In addition, this study examined these effects in children experiencing clinically significant levels of depression symptoms. In the second study, Joiner (2000) used a sample of psychiatric inpatients, among whom more extreme forms of psychopathology were present. This may have resulted in a stronger association between depression and the cognitive vulnerability–stress component of HT.

Discussion and Recommendations

Based on this analysis, the negative cognitive style by stress interaction seems to be a relatively better predictor of depression in adolescent than child samples. Whereas this is consistent with the developmental hypothesis that cognitive vulnerability may not emerge until later stages of development when children develop the capacity for formal operational thought (e.g., Cole and Turner, 1993; Gibb and Coles, 2005), the effect sizes for the interaction term varied considerably across studies. A major shortcoming of studies investigating HT that may account for this variation is the way that inferential style was typically measured. The majority of studies (81%) used the Children's Attributional Style Questionnaire or the revised version (CASQ, CASQ-R; Seligman, 1984; Kaslow and Nolen-Hoeksema, 1991), both of which typically exhibit low reliability (e.g., α's of 0.45–0.61; Thompson *et al*., 1998). Utilizing a measure with low reliability increases the Type II error rate, and thus, limits researchers' ability to detect significant effects, even when they are present. As a result, effect sizes obtained may be smaller than if a more reliable measure had been used (Hunter and Schmidt, 2004). To address the low reliability of youth measures of negative cognitive style, various researchers have recently developed more reliable measures with good validity. For example, Hankin and Abramson (2002) developed the Adolescent

Cognitive Styles Questionnaire (ASCQ), which demonstrated excellent internal consistency reliability, good test–retest reliability, and factor structure consistent with HT. Also, Conley and colleagues (2001) developed the Children's Atributional Style Interview (CASI), a structured interview that exhibited good reliability. These more reliable and theoretically meaningful measures of HT's negative cognitive style in children and adolescents should be employed in future research to allow for a developmentally appropriate measure of HT's cognitive vulnerability.

Collectively, these findings suggest that interest in HT as it functions in younger populations is warranted but also lacking in several respects. First, more valid and reliable measures of negative cognitive style must be used in order to accurately assess the construct and facilitate the detection of significant findings. Second, little research has examined the symptom component of the HT. Future research examining the vulnerability–stress interaction component of this theory should include a test of the symptom component because the theory predicts that the vulnerability–stress interaction should predict increases in hopelessness, but not nonhopelessness, depressive symptoms (e.g., Hankin *et al*., 2001). Third, researchers should consider and evaluate alternative ways of conceptualizing and assessing how negative cognitive styles function across the lifespan. For example, the ‚weakest link' approach (Abela and Sarin, 2003) integrates principles of developmental psychopathology with HT to examine the development of the separate cognitive styles (i.e., cause, consequence, self-inferences) in HT that may be missed using aggregate scores. Briefly, the weakest link approach suggests that certain cognitive styles (e.g., self-implication inferences) may emerge and function earlier in development than others (e.g., causal inferences), so those first developing styles may be available and operating to predict depressive symptoms when children encounter stressors. As children develop cognitively throughout adolescence, all of the negative cognitive styles may be functioning and may begin to crystallize into a single, consolidated negative cognitive style, such as seen in adults (Hankin and Abela, 2005; Hankin *et al*., 2005).

RESPONSE STYLES THEORY

Background and Evidence Among Adults

The Response Styles Theory (RST; Nolen-Hoeksema, 1991) posits that the ways in which individuals respond to their depressive symptoms determines the severity and duration of such symptoms. The three main response styles proposed are: rumination, distraction and problem-solving. Rumination involves thoughts and behaviors that focus one's attention inward toward negative feelings and thoughts thereby intensifying and prolonging depressive symptoms. Nolen-Hoeksema (1991) outlined three mechanisms to explain how response styles, particularly rumination, may operate to influence depression. First, depressed mood is maintained through its effects on thinking and information processing, which in turn contribute to more depressed mood. Ruminative coping increases accessibility and recall of negative events (Bower, 1981); leads to more negative interpretations of behavior (Forgas *et al*., 1984); and causes individuals to feel like they have little control over outcomes (Alloy *et al*., 1981). Second, rumination interferes with instrumental behavior. Individuals who ruminate are less likely to engage in behaviors that provide any positive reinforcement and a sense of control. Finally, rumination interferes with effective problem solving. This is likely to occur because rumination makes negative cognitions more accessible and impedes the initiation of positive behaviors. There is evidence that ruminators have a more difficult time generating solutions to their problems and ultimately generate fewer and lower quality solutions to their problems (Morrow and Nolen-Hoeksema, 1990).

In contrast to rumination, problem solving, and distraction are assumed to alleviate depressive symptoms. Problem solving involves actively trying to change unfavorable situations or to resolve problems. This is often difficult as many factors associated with depression may hinder one's ability to engage in problem solving strategies. Distraction involves engaging in positively reinforcing activities to divert one's attention from symptoms of distress and depression. Such positive reinforcement often alleviates depressive affect (Fennell and Teasdale, 1984) and attenuates the duration of depressive symptoms (Nolen-Hoksema *et al*., 1993).

RST was originally developed, in part, to explain why women are more likely to develop depression than men. Nolen-Hoeksema (1991) theorized that there might be a sex difference in the tendency to employ these response styles such that women are more likely to adopt a ruminative response style and men more likely to engage in distracting responses. Later, Nolen-Hoeksema and colleagues (Nolen-Hoeksema, 1995; Nolen-Hoeksema and Girgus,

1994) extended RST into a vulnerability–stress model to help explain sex differences in depression and suggested that girls may carry a ruminative response style prior to adolescence, but it is during adolescence that it interacts with new stressors encountered in adolescence and is hypothesized to contribute to the substantial increase in depression for girls.

Research investigating the RST in adults has yielded strong support for some tenets of the theory, whereas support for others remains questionable. More specifically, an overwhelming majority of longitudinal studies have shown that individuals who ruminate report higher levels of depressive symptoms, even after controlling for initial levels of depression (e.g., Butler and Nolen-Hoeksema, 1994; Just and Alloy, 1997; Nolen-Hoeksema and Morrow, 1991; Nolen-Hoeksema *et al*., 1994). A ruminative response style has been shown to predict depressed moods that are of moderate severity as well as clinical depression (Nolen-Hoeksema *et al*., 1992; Nolen-Hoeksema, 2000). Moreover, a great deal of research has shown that more women tend to engage in a ruminative response style than men, consistent with the sex difference hypothesis of RST. In adults, the majority of studies examining a ruminative response style examined solely the main effect of rumination, excluding the role of stress. The small corpus of research examining the effects of distraction on duration and severity of depressed mood has been less convincing. Experimental studies examining this aspect of the theory have yielded the greatest support (Katz and Bertelson, 1993; Morrow and Nolen-Hoeksema, 1990; Nolen-Hoeksema *et al*., 1993; Trask and Sigmon, 1999). However, naturalistic studies examining this hypothesis have found mixed results (Butler and Nolen-Hoeksema, 1994; Just and Alloy, 1997; Nolen-Hoeksema and Morrow, 1991). Further, there has been mixed support for the hypothesis that men are more likely to distract than women; the majority of studies find that men and women are equally likely to engage in distracting activities (e.g., Butler and Nolen-Hoeksema, 1994; Strauss *et al*., 1997). Finally, little research has been conducted on problem-solving as a response to depressed mood.

Empirical Status of RST in Younger Populations

Whereas there has been an accumulation of support for some aspects of RST in adult populations, very few studies have examined this theory in younger populations. To our knowledge only two studies exist that have examined RST in younger populations (see Table III). The first study (Schwartz and Koenig, 1996) examined both the direct effect of rumination and distraction, and also their interaction with stress to predict increases in depressive symptoms 6 weeks later, in a community sample of adolescents. The main effects of rumination and distraction were in the small range (pr = 0.16 and pr = .1, respectively), whereas the magnitude for the interaction effects was negligible. In the second study, Abela and colleagues (2002) examined the three response styles using a short-term longitudinal design in a sample of 3rd and 7th grade children. The direct effect of rumination yielded a small effect size (pr = 0.17), whereas the effect for distraction and problem solving were negligible.

The limited research suggests that rumination as a vulnerability to depression has a small but consistent effect in younger populations; however, more studies using longitudinal prospective designs, which control for initial levels of depression, are needed to corroborate this notion. No support was found for the claim that distracting and problem-solving response styles should lead to decreases in depression. Without more studies, it is difficult to tell whether distraction and problem solving are not protective of depression and therefore revisions of the theory are necessary, or if there have been an insufficient number of studies with good methodology and measurement to test this premise adequately.

Discussion and Recommendations

Overall, few if any conclusive claims can be made about the status of RST in younger populations as there have simply not been enough studies conducted. Support for rumination as a vulnerability to depression appears to be a promising area of research, but more research is needed to understand when the vulnerability emerges and to test the mechanisms posited by Nolen-Hoeksema, which may account for how rumination contributes to the development of depression. More research is needed to replicate initial evidence supporting the developmental role of rumination in younger populations and for studies examining distraction and problem-solving response styles, both of which remain relatively untested tenets of RST at present. For example, it may be fruitful for future research to examine distracting response styles in experimental paradigms, as this approach has garnered support in adult populations.

As researchers continue to investigate RST in younger populations, several points merit attention

Table III. Summary of Studies Testing Response Styles Theory

Study	Sample Type	Follow-up	Vulnerability Measure	Stress Measure	Depression Measure	Variables in regression Analysis	*N*	*p*	Effect Size(pr)
Abela *et al.* (2002)	Child	1.5 months	CRSQ	None	CDI	Rumination	314	<0.01	0.17
						Distraction		ns	−0.01
						Problem Solving		ns	−0.07
Schwartz and Koenig (1996)	Adol	1.5 months	CRSQ	LEQ	CDI	Rumination	397	<0.05	$\beta = 0.16$
						Distraction		ns	$\beta = 0.1$
						Rumination×stress		ns	$\beta = 0.09$
						Distraction×stress		ns	$\beta = 0.02$

Note: CRSQ, Children's Response Style Questionnaire; CDI, Child Depression Inventory; LEQ, Life Events Questionnaire.
Child = Ages 8–12, Adolescent = 13–19.

and may serve as guidelines for future investigations in this area. Perhaps the most important issue, which we have highlighted throughout this review, is that of measurement. Little attention has been given to measures that characterize RST in children. First, most of the existing measures are modeled after the adult measure of response style and are constructed by making minor changes in wording to assure comprehension by younger participants. Whereas this seems like a good starting point, more careful thought needs to be given to developmental differences and how RST might apply to children, and as such, how developmental differences may affect measurement of a given style. A ruminative response style constitutes a self-focused attentional bias, which may or may not operate within the individual's awareness (Gotlib and Neubauer, 2000). In light of this, during early childhood it may be difficult for youth to reflect on their own thoughts and cognitive processes and report these in an accurate manner, simply because they do not yet possess higher level metacognitive abilities (Cole and Turner, 1993; Turner and Cole, 1994). Second, there seems to be a lack of consensus on which measure to use, with each study using a different measure of the response styles. Whereas it is likely that existing measures of response styles in children are highly overlapping, the use of different measures seriously limits generalizability of findings and makes comparison across studies somewhat difficult. Third, many of the measures that have been used have been criticized on the basis of problems with response bias and multiple items from the scale overlapping with depressive symptomatology. Several items on the rumination scale overlap with symptoms of depression (e.g., "Think about how hard it is to concentrate", "Think about how sad you feel"). Fourth, the small number of items assessing distraction on the Response Styles Questionnaire may be an inadequate measure of distracting responses. As Nolen-Hoeksema outlined in her original paper (1991), measures of distracting response styles should assess the number of distracters that people use, the degree of effort and concentration people use when they engage in the distracter, and the extent to which the distracter is engaging. Problems with measurement of the distraction scale may explain the lack of support. It is possible that individuals may engage in one or few distracting responses that successfully relieve their symptoms of depression. Consequently, high scores on the distraction subscales may reflect ineffective attempts at distracting, which might cause them to engage in

numerous distracting activities, but not alleviate depressive symptoms. An important avenue for research will be the development of improved assessment of ruminating and distracting response styles for use for children and adolescents.

SUMMARY OF VULNERABILITY–STRESS FINDINGS IN CHILDREN AND ADOLESCENTS

Across theories, the preponderance of research indicates that the magnitude of effect for the cognitive vulnerability–stress interaction is in the small range in child populations and is moderately larger in adolescent populations. We believe these are important findings based on this quantitative synthesis of the published research examining vulnerability–stress components of cognitive theories using longitudinal designs controlling for initial depression. These findings augment previous cross-sectional reviews (Joiner and Wagner, 1995; Gladstone and Kaslow 1995; Haaga *et al*., 1991) that demonstrated a concurrent association between negative attributional style and depressive symptoms. However, despite the advances the present review makes over past reviews, the overall lack of studies combined with a history of poor measurement of cognitive vulnerability factors makes a challenging evaluation of the empirical status of cognitive theories of depression among youth. Given such limitations, it is likely that the present review of the empirical status of these theories may be somewhat limited and the effect sizes are likely attenuated, particularly among samples of children, for whom the empirical attention and methodology is most clearly lacking. Still, the current review takes an important first step by demonstrating clearly that positive, albeit relatively small to modest, effect sizes are observed for cognitive vulnerabilities interacting with stressors to predict prospective elevations of depression.

With the exception of HT, there have been very few longitudinal prospective studies investigating cognitive theories of depression in child and adolescent populations. Due to the paucity of research investigating BT and RST, few conclusions can be drawn from the extant empirical database. In addition, questions regarding the age at which cognitive vulnerabilities become detectable and operate as putative causal risk factors (e.g., Cole and Turner, 1993; Gibb and Coles, 2005; Hankin and Abela, 2005) and whether these cognitive risks can help account for the emergence of the sex difference in depression (e.g., Hankin and Abramson, 2001; Nolen-Hoeksema and Girgus, 1994) cannot be answered due to the lack of research in this area.

GENERAL DISCUSSION: RECOMMENDATIONS FOR FUTURE RESEARCH AND IMPLICATIONS FOR A DEVELOPMENTAL UNDERSTANDING OF THE ONTOGENY OF DEPRESSION

Developmental Implications

The results of this review reveal that the average magnitude of the effect for the vulnerability–stress interaction is smaller for children than for adolescents. Whereas we caution against making major developmental assertions based on our review, this pattern may be consistent with a developmental hypothesis suggesting that negative cognitive styles are acquired in the transition from childhood to adolescence when children develop the capacity for abstract reasoning and formal operational thought (Cole and Turner, 1993). To support this notion, there is evidence that children's ability to make internal and stable attributions increases across development (Shirk, 1988). Further, theorists have hypothesized that cognitive capacities may become more generalized and rigid across development (Crick and Dodge, 1994; Gotlib and MacLeod, 1997). Indeed, there may be a point in children's cognitive development when cognitive patterns of thinking have not yet stabilized into trait-like styles (Cole and Turner, 1993). According to Cole and Turner (1993), at these younger ages depression results most directly from encountering negative life events and subsequent environmental feedback rather than from the interaction of negative attributional style with stress. As youth mature cognitively in early adolescence, their model posits that a cognitive vulnerability–stress interaction will be observed.

At the same time, however, other researchers have hypothesized that cognitive vulnerabilities to depression may emerge at a much earlier age than researchers had previously believed. Hankin and Abela (2005) elucidated several methodological reasons (see next section) why there may be inconsistent findings in child and adolescent populations and suggested that a history of poor measurement of cognitive vulnerability factors has likely contributed to mixed findings. They argue that such methodological issues, in turn, have contributed to the mixed findings and led to theoretical modifications (e.g., Cole and Turner, 1993; Gibb and Coles, 2005) to account for the equivocal findings (see next section

for greater discussion of these issues). In sum, the findings regarding the small effects size among children and the moderately larger effect size among adolescents are consistent with, but cannot be used to refute or clearly support, Cole and Turner's (1993) developmental hypothesis, or other developmental modifications of basic cognitive theories of depression.

Thus, although it is uncertain presently why the strength of the association between the cognitive vulnerability–stress interaction and depression is smaller in children compared with adolescents, it is clear from this review that: (1) negative cognitive style from HT interacts with stress to predict prospective levels of depressive symptoms in children and adolescents; (2) the main effect of rumination is associated with prospective elevations in depressive symptoms; and (3) no clear conclusion can be reached concerning BT's dysfunctional attitudes–stress interaction. It seems reasonable, at the present time, to infer that the research in this area is generally consistent with hypotheses from cognitive theories of depression, and that these theories warrant further investigation. As research progresses, it is important to note some of the shortcomings of previous research that were apparent from reviewing the extant studies. In the next section we outline some of the limitations that may have impeded advancement of this field and raise some critical issues that may improve our understanding of how cognitive vulnerability confers risk for depression among youth.

Methodological Limitations of Past Research

Since the last empirical syntheses of the literature (e.g., Gladstone and Kaslow, 1995; Joiner and Wagner, 1995), the field has undergone considerable methodological advancements. As mentioned earlier, the most accurate tests of cognitive theories of depression are those that employ longitudinal prospective designs, control for initial depression, and include stress as a main effect and in interaction with cognitive vulnerability. Whereas studies of this nature can be costly and laborious, investigators in this area of research have largely met these criteria and significantly increased knowledge on cognitive theories of depression. However, the current review reveals several methodological concerns that have affected the study of cognitive theories of depression in child and adolescent populations. These limitations suggest some methodological advancements for future research.

First, it is likely that studies using measures with unestablished or poor reliability and validity produce smaller effect sizes. Many researchers have tended to assess cognitive vulnerability using age-inappropriate measures with poor psychometric properties. As mentioned earlier, one example of this is that the vast majority of studies examining HT have used the Children's Attributional Style Questionnaire (CASQ; Seligman *et al*., 1984), or the revised version (CASQ-R; Thompson *et al*., 1998), both of which demonstrate poor internal consistency alphas typically ranging from 0.4 to 0.6 (Gladstone and Kalsow, 1995; Thompson *et al*., 1998). Further, there appears to be little consensus on which assessment tools to use for measuring cognitive vulnerability and stress. Thus, it is not clear whether the link between cognitive vulnerability and depression is weaker earlier in development, or if this is an artifact of poor measurement. It is recommended that measures demonstrating good psychometric properties, such as the ACSQ (Hankin and Abramson, 2002) or the CDAS (Abela and Sullivan, 2003), be used in future research to allow for a more reliable and valid assessment of cognitive vulnerabilities to depression. In light of this review, research may benefit from developing and utilizing measures that are specific to child and adolescent populations separately as the strength of the cognitive vulnerability–stress interaction appears to differ, albeit in a moderate fashion, across age. However, more research is needed to understand the dynamics between cognitive development and cognitive vulnerability in order to construct age appropriate measures that accurately represent cognitive theories of depression across development.

Second, cognitive theories of depression posit that individuals who are cognitively vulnerable to depression are more likely to become depressed following the occurrence of stress. Examining these theories developmentally raises some critical issues concerning the time frame within which to examine the development of depression. Little research has considered whether the optimal time frame for examining these mechanisms differs developmentally. With widespread use of self-report questionnaires and two time point panel designs, it may be that children, and perhaps early adolescents exhibit reduced capabilities to recall stress and symptoms of depression accurately over lengthy follow-up intervals. Further, after the stressor has occurred, little consideration is given to trajectories of depression after this point. It is possible, as Weiner and Graham (1985) has emphasized, that many individuals expe-

rience a primitive emotional response after perceiving an event, which is determined by the attainment or nonattainment of a given goal, rather than by cognitive processes. Based on this, Hankin and Abramson (2001) hypothesized that all individuals experience rises in initial negative affect after a stressor occurs, but it is only those who are cognitively vulnerable to depression who experience enduring elevations in depression. In light of this information, the use of two time point designs, which predominate the field currently, may not accurately depict the experience of depression in childhood and adolescence, and may not capture subtle fluctuations in depressive symptoms after stress. Thus, it will be important for future studies to follow participants through the period of greatest risk for the development of depression and use multiple assessments points to provide an optimal test of the etiologic processes posited in cognitive theories of depression. Indeed, developmental methodologists (e.g., Curran and Willoughby, 2003) encourage a minimum of three time points to test developmental hypotheses and processes, and they argue that two time points are not much of an improvement over cross-sectional designs.

Of interest, we analyzed the effect sizes of the studies included in our review as a function of the reported length of follow-up period. A curious pattern emerged in which the cognitive vulnerability–stress interaction remained relatively stable for children across follow-up intervals; however, for adolescents the size of effect appeared to diminish somewhat over time. Although these results speak against the developmental hypothesis, we caution against making any firm conclusions at this point as this pattern was based on a limited number of studies, each of which employed a variety of measurement tools and study designs. More studies are needed using consistent methodology to shed light on the potential effects of the length of follow-up period on the examination of cognitive theories in younger populations.

Third, understanding how children and adolescents become depressed, within the context of cognitive theories of depression, may benefit from multimethod, multi-informant designs. To date, the vast majority of studies examining vulnerabilities to depression in children and adolescents have utilized self-report questionnaires or interviews as the primary methods of assessment. Whereas this makes sense given that many of the symptom criteria for diagnosing depression are subjective, these methods may have limitations specific to younger children. First, self-report methods are not suitable for children below a certain level of reading and cognitive capacity (Kovacs, 1986) because metacognitive abilities (e.g., planning, monitoring, and evaluating) are needed for children to be able to evaluate how often and intensely they experience maladaptive thinking styles in order to reflect upon their symptoms of depression. Second, self-report measures have generally had difficulty differentiating depression from other forms of negative affect (Wolfe *et al*., 1987). Third, a dominant theme in depressive disorders is the tendency to perceive things in a more pessimistic manner than is necessarily the case (Beck, 1967). Therefore, it is possible that self-report questionnaires allow youth to overestimate their degree of emotional distress. We acknowledge the logistical difficulties that come with conducting more rigorous tests of cognitive theories; however, much knowledge can be gained from using multiple collateral informants and multiple methods (e.g., observational procedures) to minimize the effects of these limitations.

Fourth, some have expressed concern about the discriminant validity of the etiological constructs posited in cognitive vulnerability theories of depression (e.g., self-criticism and dependency; Coyne and Whiffen, 1995). However, in contrast to this hypothesis, factor analytic research has produced initial evidence, at least among older adolescents, suggesting that each of the cognitive vulnerabilities featured in BT, HT, and RST are indeed distinct from each other and from neuroticism, depressive symptoms, and low self-esteem (Hankin *et al*., 2005). Still, in this study many items from the rumination scale loaded highly onto a factor consisting of depression, self-esteem and neuroticism, whereas the remaining items from the rumination scale loaded onto their own factor. Interestingly, previous studies examining the factor structure of the rumination scale have found that, after confounding depression items were removed, a two factor model of rumination emerged, labeled Brooding and Reflection (Treynor *et al*., 2003). These initial factor analytic studies providing evidence for discriminant validity have been conducted entirely with older adolescents and adults, so there is an important need for future factor analytic studies to evaluate the discriminant validity of cognitive vulnerability measures in childhood and adolescence separately. Currently, there is little consensus on what measures should be used to examine each of the key vulnerabilities posited by cognitive

theories of depression in youth. Still, discriminant validity should be considered when developing and validating appropriate measures for children and adolescents.

Statistical Limitations of Past Research

With the advent of more rigorous methodologies and designs to examine cognitive theories of depression, statistical strategies to analyze and interpret the data derived from such enhanced studies are crucial for the advancement of the field. The current review has highlighted some of the statistical limitations of past research, and we now suggest some ways for future investigations to advance knowledge in this area.

First, as previously mentioned, we originally intended to provide a meta-analytic review of the empirical status of cognitive theories of depression among youth. However, very few studies provided the necessary statistical indices (beta weights and their associated T and standard error values), so this precluded a meta-analytic approach. Moreover, some studies failed to provide sizes of effect for the cognitive vulnerability–stress interaction term, which constitutes the core of cognitive theories of depression. To move the field forward, it is essential that authors report means, standard deviations, and correlations among the main measures in their studies. In addition, authors should report unstandardized and standardized regression coefficients, associated t values and standard errors, partial correlations, R^2, adjusted R, F values, and exact p values for all variables examined in statistical models.

Second, many studies have not taken into account powerful techniques for modeling longitudinal data (e.g., Collins and Sayer, 2003). All of the studies included in this investigation used standard multiple regression, which is limited by its ability to use only two time points and to consider only cases with complete data. As stated earlier, more powerful tests of cognitive theories of depression require longitudinal data with multiple assessments to model fluctuations of each of the key variables over time. Analytic techniques, such as hierarchical linear modeling and latent growth modeling, can take advantage of such rigorous study designs as they flexibly handle missing data, and offer a more sensitive approach for modeling longitudinal data (Raudenbush, 2001). Further, such an approach allows for a more theoretically sound test of cognitive theories as the strength of the association between individuals' fluctuations in stress and depressive symptoms over time can be modeled as a function of their cognitive vulnerability levels (i.e., an idiographic approach). In contrast, the use of multiple regression techniques only enable nomothetic tests of how individuals' changes in stressors and depression vary from the sample average, not from how the individuals' levels of stress vary within an individual over time (i.e., idiographic) (see Abela and Hankin, in press, for greater discussion of this issue).

Finally, and perhaps most importantly, null hypothesis significance testing is widely employed, whereas its implications are largely misunderstood (see Chow, 1988; Morgan, 2003, for reviews). In this next section we review the fundamentals and logic behind null hypothesis significance testing and present some of the problems associated with this statistical approach.

Null hypothesis significance testing represents a broad set of quantitative techniques for evaluating a research hypothesis under the assumption that the null hypothesis is true. The null hypothesis states that there are no systematic differences between the two populations—that is, the population used in the research study and the hypothetical statistical population. In null hypothesis significance testing, the hypothetical statistical population represents the distribution of sample means one might expect to observe in the research population. Based on this distribution, the expected difference between the hypothetical statistical population mean and the mean observed in the research population are compared when the sampling error can be estimated. This information is used to make a binary decision about whether the null hypothesis is a viable explanation for the study results. If the probability of observing the mean derived from the research population under these assumptions is small, the null hypothesis is rejected. By convention if the probability of observing a particular mean in the research population is less than 5% ($p < 0.05$), the null hypothesis is rejected and the study results are deemed "statistically significant". More practically, rejection of the null hypothesis eliminates chance as a plausible explanation for the observed difference between the research and statistical populations. Conversely, if the probability of observing the mean from the research population under these assumptions is large (greater than 5%, $p > 0.05$), the null hypothesis is accepted. Accepting the null hypothesis implies that chance cannot be discounted as an explanation for the differences between the two populations.

Whereas many philosophical and practical problems are associated with the use of null hypothesis significance testing, we review a few key problems that may readily create an awareness of the misconceptions associated with this statistical approach. First, null hypothesis significance testing does not allow researchers to make any claims regarding their research hypothesis. Null hypothesis significance testing is only capable of determining the probability of observing the data in the research population, given that the null hypothesis is true. Therefore, the research hypothesis is, in effect, never explicitly tested, and as a result it is impossible to ascertain its validity. Moreover, null hypothesis significance testing does not allow the researcher to evaluate whether alternative hypotheses, that are different from the research hypotheses, may lead to the observed difference between the research and statistical populations (Morgan, 2003).

Second, the *p*-value is commonly misunderstood as being indicative of meaningfulness or importance of a finding. Little attention is paid to the difference between theory, statistics, and data, and researchers often mistake null hypothesis significance testing as a test for theoretical hypotheses as opposed to statistical hypotheses. Null hypothesis significance testing is minimally useful, in that it provides criteria for judging whether the results are likely, based on the premise that they are unlikely (Morgan, 2003). It is important to emphasize that this statistical approach is silent on the practical, and theoretical importance of a finding. Theory evaluation can only be accomplished by examining the size of effect, which represents a quantitative index of the strength of association between variables (Berger and Berry, 1988; Chow, 1988; Schmidt, 1996). Correlations, the percentage of variance explained, or some other index of effect size provide a more objective approach to evaluating theories, and should be routinely reported and considered when interpreting findings.

Finally, statistical power is rarely taken into account when conducting null hypothesis significance testing. Statistical power is defined as the probability of correctly rejecting the null hypothesis and is strongly influenced by sample size. Sample size and rejection of the null hypothesis are inversely proportional such that a large sample size requires a smaller difference between the two populations to reject the null hypothesis (Morgan, 2003). Therefore, an evaluation of theories based on levels of significance may overlook meaningful associations because studies often use insufficient power to detect such effects. Additionally, sizes of effect can have large or small *p*-values, depending on the sample size. Researchers should take the statistical power of their planned analyses into account when selecting sample sizes to diminish the Type II error rate.

A final comment raises a critical issue regarding interpretation of findings and theory evaluation. The over reliance on null hypothesis significance testing has led many researchers to conclude that the evidence supporting cognitive theories of depression in child and adolescent populations is mixed. However, by using sizes of effect as the evaluating criterion, it is apparent that the cognitive vulnerability–stress interaction across theories and populations does account for incremental variance in explaining prospective increases in depressive symptoms, albeit in some cases to a small degree. The central issue worth considering is, significance testing aside, how much incremental variance is meaningful and can be considered as evidence supporting, or refuting, hypotheses derived from cognitive theories of depression.

Theoretical Limitations of Past Research

As the research accumulates in this literature, a number of theoretical issues have emerged. First, a significant limitation of previous research examining HT with children and adolescents is their sole focus on negative attributional style as the cognitive vulnerability. According to HT, a negative cognitive style also includes negative inferences for the consequences and implications for the self, following the occurrence of a negative life event. Therefore, it is important to study the entire construct of cognitive vulnerability to depression. To date only a fraction of HT has been investigated, primarily due to the lack of assessment tools needed to measure all three inferential styles. Researchers should take advantage of the recent improvements in developmentally sensitive measures to evaluate all tenets of HT.

Second, theory and initial evidence suggests that various forms of cognitive vulnerability may develop at different rates for different children (Abela and Payne, 2003; Abela and Sarin, 2003). The majority of research in younger populations has examined global levels of cognitive vulnerability and has not taken into account the relationships among the different cognitive vulnerability elements (e.g., negative inferences for cause, consequence, and self), all of which together are summed to create a global score of overall cognitive vulnerability. Abela and Sarin's (2003) "weakest link" hypothesis suggests that until

different forms of cognitive vulnerability emerge and consolidate to form a global negative cognitive style, children's cognitive vulnerability to depression will be determined by their most specific negative cognitive style (e.g., inferences for cause, consequences, or self-characteristics). Research with adults shows that the three negative inferential styles coalesce to form one latent factor and each of these components is not factorially distinguishable (Hankin *et al*., 2005). However, at younger ages, inferences about the self are more likely to be the weakest link, and causal inferences become more consolidated starting in early adolescence (e.g., Abela and Payne, 2003; Abela and Sarin, 2003). Therefore, the research with younger populations suggests that different facets of the negative cognitive style are separable. This approach has important implications for studying depression developmentally as different cognitive vulnerabilities may emerge over time.

Third, research investigating cognitive theories of depression is likely to benefit from examining the effects of cognitive vulnerability on individual depressive symptoms as well as overall levels of depressive symptoms because depression may manifest itself differently at various stages of development (Hammen and Rudolph, 2003; Weiss and Garber, 2003). Developmental psychopathologists suggest that the structure and nature of depression, and the causes or consequences related to depression, may differ between children and adolescents such that manifestations of depression may comprise a different set of specific symptoms because children may not yet possess the associated capacities to experience the symptoms that are typical of adult depression. Although there is little research on this topic (Weiss and Garber, 2003), preliminary evidence appears to support this notion and shows that very young children tend not to report hopelessness and depressed mood, but rather they tend to endorse somatic symptoms of depression (e.g., Carlson and Kashani, 1988; Kovacs, 1996). These types of symptoms decrease with age, whereas psychomotor retardation and anhedonia become more common with the transition from childhood to adolescence (Weiss and Garber, 2003). Another important reason to study the prediction of individual symptoms of depression comes from cognitive theorists who have postulated that cognitive vulnerability factors may lead to a specific subtype of depression, such as hopelessness depression, characterized by a unique symptom profile. Future research examining individual depressive symptoms may shed light on the structure and nature of depression across development and will inform how vulnerabilities interact with stress to affect the development of depression across the lifespan.

Last, few published studies have examined the priming hypothesis in youth. Many theorists have hypothesized that cognitive vulnerability factors are typically latent and that relevant cognitive structures or processes must be activated or primed in order to be assessed accurately (Gotlib *et al*., 2004; Ingram *et al*., 1998; Persons and Miranda, 1992; Scher *et al*., 2005). Timbremont and Braet (2004) found that never-depressed children exhibited biased recall of positive words after a negative mood induction, whereas currently depressed children showed biased recall of negative words. Children diagnosed with major depression were found to attend more to negative emotional pictures compared with control children who attended to positive pictures (Ladouceur *et al*., 2005). Last, Taylor and Ingram (1999) found that children of depressed parents who experienced a negative mood induction exhibited reduced processing of positive self-referent words and greater recall of negative words compared with control children. Of interest, these priming effects were only seen when a negative mood induction was used: half of the children received no negative mood induction, and no significant priming effects were observed among these children. It has been suggested that priming procedures and/or a negative mood induction can activate the latent negative schema hypothesized in cognitive models in much the same way that stressors are postulated to activate these negative cognitive structures to contribute to increases in depression. Overall, despite a handful of priming studies, the lack of research examining the priming hypothesis represents a key lacuna in the literature and suggests that current studies may not be providing an accurate test of the most basic tenets posited by cognitive theories of depression. It is possible that the mixed findings in younger populations may be the result of a failure to activate the schema prior to its assessment.

CONCLUSION

Research in this area presents a promising avenue for research as relatively little is known about how cognitive theories of depression function in children and adolescents. This is somewhat surprising given that Beck articulated the first cognitive theory of depression approximately 40 years ago (1967), yet as this review revealed, only two studies have

prospectively tested BT's basic vulnerability–stress hypothesis in youth, let alone the other aspects of his model (e.g., domain matches between vulnerability and stress). The cognitive theories provide rich frameworks upon which to begin understanding the role of cognition in the etiology and maintenance of depression, but more rigorous studies using more sophisticated designs, statistical approaches, and developmentally sensitive measures to assess entire cognitive vulnerabilities are needed.

A number of conclusions emerged from this review on cognitive theories of depression in youth. Most importantly, as the field currently stands, the effect size magnitude for the average cognitive vulnerability–stress interaction in children falls in the small range, whereas for adolescents this effect is moderately larger. However, the empirical status of cognitive theories of depression in younger populations is at present unclear and obscured by various methodological, statistical, and theoretical limitations. Thus, these results should be considered as tentative conclusions because few studies have adequately tested the etiologic chains proposed by cognitive theories of depression. Such limitations have impeded advances in understanding how cognitive factors and processes confer risk for depression developmentally.

The findings from this review should be interpreted with caution for the following reasons. First, our method of averaging across studies to determine the strength of association between the cognitive vulnerability–stress interaction and depression among children and adolescents is limited because it does not take into account the sample size in each study. It is likely that studies with larger sample sizes provide more robust findings. Second, it is important to consider the outlined methodological, statistical, and theoretical limitations when interpreting our findings. Our review reveals that the average effect size for the cognitive vulnerability–stress interaction is relatively larger in adolescents than children, yet it is important to take into account the current state of the field in order to overstate any major developmental assertions. Finally, as noted earlier, we wish to highlight that our review presents a very conservative evaluation of cognitive theories of depression because all of the studies controlled for initial levels of depressive symptoms, and this likely is an overly cautious statistical control. Further, many of the studies included in this analysis incorporated other constructs proposed to be involved in the development of depression. It is likely that the effect sizes for the cognitive-vulnerability–stress interactions may be an under-estimated.

Several directions for future research were suggested by this review. First, researchers examining cognitive theories of depression should make greater use of developmentally appropriate, validated measures of cognitive vulnerability and conduct research using multiple assessments to maximize the chances of detecting the effect posited by each of these theories. Studies should also use multiple methods of data collection, multiple informants, and more rigorous analytic techniques to model longitudinal data more thoroughly. Second, more prospective studies are needed to investigate BT and RST, in particular, as only a few preliminary studies exist. More studies are needed to evaluate all cognitive theories using psychometrically strong measures. This information is crucial for understanding how cognitive vulnerabilities may function in younger populations and for disentangling subtle differences delineated by each theory on the role that cognitive processes may play in depression developmentally. It would be ideal for these studies to investigate these effects in community as well as clinical populations, especially because findings from only clinical samples are limited in terms of generalizability (Goodman *et al.*, 1997). Third, more studies are needed that focus on child and adolescent populations separately to understand developmental differences in depression, cognitive vulnerability, and the experience of stress. Knowledge of these distinctions may inform much needed research focusing on the developmental period when rates of depression rise with the transition from childhood to adolescence. Finally, studies would benefit from including several theoretically important moderators (e.g., age, ethnicity, and sex) to examine whether these operate differently within the context of each theory.

In closing, cognitive theories of depression originated with adult research in the 1960s and 1970s and have been extended downward to youth more recently. Prospective research with adults has been largely supportive, and as demonstrated in this review, the evidence mostly supports cognitive vulnerability–stress models in youth as well. We believe that there is substantial room for developmental psychopathologists to continue testing cognitive factors and processes using enhanced methods, designs, and statistics in order to test newer, more advanced developmentally sensitive aspects of these cognitive theories. We are excited by the future of research testing developmentally minded cognitive theories of

depression to understand the development of depression over the lifespan, and we look forward to new and stronger tests of cognitive theories of depression and the ensuing accumulation of knowledge.

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