Clinical Psychology Review 71 (2019) 63–77

Contents lists available at ScienceDirect

Clinical Psychology Review

journal homepage: www.elsevier.com/locate/clinpsychrev

Review

Cognitive and attentional vulnerability to depression in youth: A review

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HIGHLIGHTS

- There is a need to better understand the development and maintenance of depression in youth.
- This review synthesizes research on cognitive and neurobiological factors in youth depression.
- Consistent with the model, attentional impairments appear greatest for negative information.
- There is limited evidence that attentional deficits are associated with rumination in youth.
- Other model predictions have mixed support or require additional study.

ARTICLE INFO

Keywords:
Attention control
Depression
Rumination
Youth
Stress

ABSTRACT

Although depressive disorders are among the most common disorders in youth, highly efficacious treatments for childhood affective disorders are lacking. There is significant need to better understand the factors that contribute to the development and maintenance of depression in youth so that treatments can be targeted at optimal mechanisms. The aim of the current paper was to synthesize research on cognitive and neurobiological factors associated with youth depression, guided by De Raedt and Koster's model (2010) for vulnerability to depression in adults. Consistent with model predictions, there is evidence that attentional impairments are greatest in the context of negative information, relative to positive or neutral information, and some evidence that attentional deficits are associated with rumination in depressed youth. However, we found little evidence for the model's assumption that attentional bias is an etiological and maintenance factor for depression. There are several other model predictions that require additional study as current data are lacking. Overall, De Raedt and Koster's (2010) integrative cognitive and biological framework has tremendous potential to move the field forward in understanding the development of depression in youth. Additional longitudinal studies incorporating measures across biological and cognitive levels of analysis are needed.

Depressive disorders are among the most common disorders in youth (e.g., Kessler et al., 2005). Depression emerges as early as preschool age (Luby et al., 2003) and the prevalence increases dramatically later in childhood and adolescence (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Merikangas et al., 2010). Depression in youth is often associated with a chronic or recurrent course (Costello et al., 2003; Kovacs, Obrosky, & George, 2016; Mian, Wainwright, Briggs-Gowan, & Carter, 2011) and predicts difficulty with interpersonal relationships, increased physical problems, and impairment in global functioning (Kovacs, Akiskal, Gatsonis, & Parrone, 1994; Nolen-Hoeksema, Girgus, & Seligman, 1992; Puig-Antich et al., 1985a,1985b; Puig-Antich et al., 1993; Rao et al., 1995; Rohde, Lewinsohn, & Seeley, 1994; Strober, Lampert, Schmidt, & Morrell, 1993).

Given the prevalence, persistence, and impairment associated with depressive symptoms in youth, more effective prevention and intervention efforts are necessary. Highly efficacious treatments for childhood affective disorders are lacking and existing treatments only yield moderate effect sizes and remission rates (e.g., Cartwright-Hatton, Roberts, Chitsabesan, Fothergill, & Harrington, 2004; Chorpita et al., 2011; Weisz, McCarty, & Valeri, 2006). Thus, there is significant need to better understand the factors that contribute to the development and maintenance of depression in youth so that treatments can be targeted at optimal mechanisms.

Our current understanding of the etiology and maintenance of depression may be limited in part because of a narrow focus on individual vulnerabilities for depression rather than an interaction of vulnerabilities (Hankin, 2012). Examining individual vulnerability factors with different measures and at different levels of analysis in separate

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https://doi.org/10.1016/j.cpr.2019.01.004
Received 29 December 2017; Received in revised form 8 January 2019; Accepted 22 January 2019
Available online 24 January 2019
0272-7358/

studies has resulted in findings that are difficult to aggregate; further, individual factors account for only small amounts of variance in depression (Hankin, 2012). Integration of vulnerability factors into a cohesive framework is likely to substantially improve our understanding of depression.

The current paper synthesizes research on cognitive and neurobiological factors associated with youth depression, guided by a model recently developed to explain the increasing vulnerability to depression in adults. De Raedt and Koster (2010) integrate previously disparate cognitive and neurobiological models of depression to provide a more comprehensive understanding of the development and maintenance of depression symptoms in adulthood. A key element of the model is that attentional control is progressively impaired with each depressive episode, increasing vulnerability for future recurrence. The authors build upon biological findings that depressive episodes are associated with hypercortisolism. These periods of increased cortisol are hypothesized to interfere with typical Hypothalamic-Pituitary-Adrenal axis (HPA axis) functioning, resulting in the system becoming increasingly reactive to stressors over time. Because the HPA axis also regulates serotonin transcription, which in turn regulates the dorsolateral prefrontal cortex (DLPFC), there are downstream negative effects on executive functioning and attentional control. Specifically, the DLPFC has decreased control over amygdala activity, resulting in prolonged periods of negative affect and deficits in inhibition. This combination of impaired inhibition and prolonged attention for negative material (as a result of increased amygdala activation) is hypothesized to provide a "gateway" for sustained elaborative processing (e.g., rumination). The model is unique in that it highlights the central role of attention in vulnerability for depression given its relationship with biological, cognitive (e.g., elaborative processing), and affective processes.

Examining a downward extension of De Raedt and Koster's (2010) model of depression to youth is critically important for several reasons. First, there is rapid cognitive development during school age and early adolescence when attention networks are established (Amso & Scerif, 2015; Crone & Steinbeis, 2017; Diamond, 2013). Given the key role of attention in the model, it is necessary to examine the literature from a developmental perspective. In turn, there may be developmental windows associated with attention and depression that represent ideal periods for prevention or intervention. Second, the chronicity of depressive episodes and findings that early depressive symptoms in youth are associated with later episodes in adulthood (Pine, Cohen, Cohen, & Brook, 1999) suggest that relevant cognitive and attentional processes may be impacted well before adulthood.

This paper is organized by first reviewing the prevalence and impact of depression in youth and the normative development of attentional control and attentional bias. The subsequent sections outline each of the major tenets of De Raedt and Koster's (2010) model and a brief review of the relevant literature focused on youth. The first section reviews literature on the hypothesized links between attentional control, attentional bias, and depression in youth. This is followed by a section reviewing the neurobiological underpinnings of attention deficits in youth depression, including dysregulation in the HPA axis, serotonergic system, prefrontal cortex, and amygdala. The final section reviews links between depression and stress-activated negative schema. We conclude with a summary on the state of the literature in terms of support for major predictions of the model and suggestions for future research.

1. Depression prevalence, course, and associated outcomes in youth

Clinically significant depression in youth emerges as young as age three (Luby et al., 2003; Luby, Belden, Pautsch, Si, & Spitznagel, 2009) and increases in prevalence across development. Approximately 2% of children and 12.5% of adolescents reported a major depressive disorder (MDD) in a one-year period (Center for Behavioral Health Statistics and Quality, 2016; Costello et al., 2002). Rates of MDD increase steadily through adolescence, rising from 5.4% at age 12 to 15% at age 17 (Center for Behavioral Health Statistics and Quality, 2016). The lifetime prevalence of MDD is 23.2%% (Kessler et al., 2005).

Identifying children in the earliest stages of a depressive episode may prevent the development of a detrimental long-term trajectory characterized by a relapsing and remitting course. Age at onset of first episode and number of depressive episodes both explain risk of depression recurrence (see Burcusa & Iacono, 2007 for review) and early onset has been linked with a chronic, episodic course of illness (Costello et al., 2003). Relapse of depression is common in youth. Estimates suggest that approximately 12% of children will experience a depression relapse within 1 year, 40% will relapse within 2 years, and 75% will experience a second episode within 5 years (Kovacs et al., 1984; Lewinsohn, Clarke, Seeley, & Rohde, 1994).

Depression in youth is associated with a number of additional negative concurrent and long-term effects. Children with depression experience significantly higher rates of comorbid disorders compared to children without the disorder. Approximately two-thirds of school-age children with depression have at least one comorbid disorder (Ford, Goodman, & Meltzer, 2003). Depressive disorders in youth are also linked with increased substance use and suicide risk (Cohen et al., 1993; Foley, Goldston, Costello, & Angold, 2006). Associated impairment in other domains is also common, as youth with depressive symptoms commonly experience significant difficulties in interpersonal relationships, low educational attainment, and impairment in global functioning (Asarnow, Jaycox, Duan, Laborde, Rea, Tang, et al., 2005; Nagar, Sherer, Chen, & Aparasu, 2010). Over the long term, adolescent depression is associated with a range of problems in young adulthood, including major depression, anxiety disorders, substance use disorders, poorer physical health, suicidality, and impairment in work, social, and family life (Dunn & Goodyer, 2006; Fergusson, Boden, & Horwood, 2007; Fergusson & Woodward, 2002; Keenan-Miller, Hammen, & Brennan, 2007; Mcleod, Horwood, & Fergusson, 2017); further, the detrimental effects seem to be greater for women (Hammen, Brennan, & Le Brocque, 2011).

2. Development of attentional control and attentional bias

Because attentional control deficits are central to De Raedt and Koster's (2010) model of depression, applying a downward extension of the model to children should be informed in part by understanding how attentional control develops normatively. The terms executive function, cognitive control, and attentional control are at times used interchangeably in the literature. Attentional control includes a set of higher-order processes that guide and regulate lower-order processes to maintain goal-directed behavior (e.g., Miyake & Friedman, 2012). These higher-order processes are largely supported by frontoparietal cortices. Recent models suggest that executive functioning is comprised of three related but separable components (e.g., Miyake et al., 2000), including mental set shifting (the ability to switch between mental sets or tasks), working memory monitoring and updating (the ability to attend to incoming information relevant to current goals and updating previously but no longer relevant information with newer, relevant information), and inhibition (the ability to inhibit dominant, automatic, or prepotent responses). Attentional bias, also important in De Raedt and Koster's (2010) model, can be conceptualized as both an initial alerting mechanism toward incoming stimuli as well as sustained focusing of attention over longer periods. Attentional bias can be mapped onto the three components of attentional control by considering initial alerting problems as deficits in inhibition and sustained focusing as deficits in switching or updating working memory. For consistency with De Raedt and Koster's (2010) model, we use the term "attentional control" to refer to both executive function and attention throughout this review.

Attentional control emerges relatively early in life and increases from early childhood through late adolescence, although there are some differences in the trajectories of individual dimensions of attentional control (Amso & Scerif, 2015; Crone & Steinbeis, 2017; Diamond, 2013). The overall pattern of development in functional connectivity and grey-matter volume begins caudally and becomes more rostral over time (Amso & Scerif, 2015), with particularly slow development in childhood in the frontoparietal cortices (Gogtay et al., 2004; Sowell et al., 2004). Across the three dimensions of attentional control each domain follows a different developmental time course. Inhibition abilities have the most rapid developmental trajectory and appear fully developed by adolescence (Huizinga, Dolan, & van der Molen, 2006). Shifting abilities develop throughout adolescence (Cepeda, Kramer, & Gonzalez de Sather, 2001; Huizinga et al., 2006) while working memory skills continue to develop well into late adolescence and young adulthood (Carriedo, Corral, Montoro, Herrero, & Rucián, 2016; Cowan, 2016; Huizinga et al., 2006). Studies examining orienting behavior, which may also be relevant for attentional bias, suggest that there is ongoing development in visual orienting during childhood through adolescence (Konrad et al., 2005; Rueda et al., 2004).

There is also evidence that genetic and environmental effects differentially contribute to the variance in cortical thickness change over the course of childhood (see Amso & Scerif, 2015 for a comprehensive review). Environmental effects on DLPFC decrease rapidly in early to middle childhood with substantially decreased influence in late childhood throughout adolescence (Schmitt et al., 2014). By 16–17 years of age there appear to be virtually no environmental effects on individual differences in attentional control (Friedman et al., 2008). Taken together these findings suggest that attentional control abilities, across dimensions, are rapidly developing during school age and through adolescence and it may be helpful to time the delivery of early intervention toward this developmental window, when environmental influences are strongest.

3. Support for model components and predictions

3.1. Literature review

The focus of the review was on identifying literature relevant to the core predictions of De Raedt and Koster's (2010) model in youth. The search was restricted to youth between preschool age and early adulthood. The search was conducted between February and November 30th of 2017 in PSYCINFO and was limited to peer-reviewed articles published in English. Studies that included special diagnostic populations (e.g., youth with bipolar disorder or comorbid developmental delays and depression) were excluded. Given the complexity of De Raedt and Koster's (2010) model, separate searches with relevant combinations of the following search terms were conducted for each portion of the model. Search terms included "youth" "child" "adolescent" "depression" "major depressive disorder" "rumination" "attentional control" "executive function" "cognitive control" "attentional bias" "hypofrontality" "cognitive bias modification" "HPA dysregulation" "amygdala dysregulation" "amygdala hyperactivity" "amygdala volumetric difference" "serotonergic dysregulation" and "schema." Additional studies were identified and retrieved from reference lists.

3.2. Attentional control, attentional bias, and rumination in youth depression

De Raedt and Koster's (2010) model hypothesizes that attentional control plays a key role in linking biological and cognitive vulnerability to depression. The model predicts that stressors activate a cascade of biological processes (dysregulated HPA axis, serotonergic dysregulation, hypofrontality) and cognitive processes (activation of negative schema) that result in diminished attentional control. Decreased attentional control, especially inhibition and shifting, results in failure to prevent or disrupt negative elaborative processing (i.e., rumination). The model purports that such repetitive negative thinking leads to sustained negative mood and ultimately emotional disorders. Attentional control is thought to be mood congruent and the association between negative mood and rumination is strengthened after each mood episode. This association between attentional difficulty and rumination increases the likelihood that future negative moods will trigger similar negative thinking patterns. The literature examining these model predictions in youth is reviewed below.

3.2.1. Attentional control and depression

De Raedt and Koster's (2010) model predicts that stress and negative affect lead to diminished attentional control. Attentional control is expected to be impaired in the presence of negative mood (e.g., in a depressive episode) and is expected to be greater when processing negative information. Findings for neutral compared to emotional task stimuli, when available, are reviewed separately below.

In adults, depressive disorders have been linked consistently with moderate deficits in attentional control (Fossati, Coyette, Ergis, & Allilaire, 2002; Hammar & Årdal, 2009; Wagner, Doering, Helmreich, Lieb, & Tadić, 2012), although evidence in youth is less consistent. For example, both a meta-analysis and a review paper, both published in 2015, came to different conclusions about the association between attentional control and depressive disorders (Vilgis, Silk, & Vance, 2015; Wagner, Müller, Helmreich, Huss, & Tadić, 2015). The meta-analysis included 17 studies of 447 participants with a diagnosis of MDD and 1347 healthy controls, ranging in age from 9 to 15.8 years (Wagner et al., 2015). Of these, six examined shifting, six examined working memory, and three examined inhibition. Results suggested that across studies, youth with MDD performed more poorly on measures of attentional control compared to youth without MDD. There was a small effect of MDD on shifting ($d = 0.44$), a medium effect on working memory ($d = 0.49$), and a large effect on inhibition ($d = 0.77$). On other related measures of attention, there was a medium effect of MDD on sustained attention ($d = 0.52$), but not selective attention ($d = 0.19$). Overall, the effects of depression were strongest for inhibition, which is consistent with De Raedt and Koster's (2010) model prediction. Because the effects of neutral as compared to emotional information were not examined in this meta-analysis, the impact of valence on attentional control cannot be evaluated.

The qualitative review concluded that overall deficits in attentional control were not associated with depressive disorders in children and adolescents, although the authors note that negative stimuli may influence attention on some tasks (Vilgis et al., 2015). The authors reviewed 33 studies including children ages 6 to 19 years. Of note, 11 of these were also included in the Wagner et al. (2015) meta-analysis described above. Studies were grouped by findings related to set-shifting, working memory, and inhibition. "Hot" (affective) and "cold" (neutral) cognition were evaluated separately for each group. For tasks with neutral stimuli, only three of the ten studies examining shifting found evidence for shifting deficits in depression. Studies of working memory were mixed. For visuospatial working memory, results showed associations with depression on four estimates but failed to find differences on two others. For verbal working memory, two studies showed group differences, whereas five did not. Most results for tasks assessing response inhibition failed to find group differences; the authors reviewed 16 studies and noted that only three reported significant differences between depressed and healthy control groups. For tasks with emotional stimuli, results were mixed. Results from only one of four dot-probe tasks and five of nine other tasks assessing different components of attention found an association between depression group status and performance. Importantly, conclusions drawn from this review are based on the number of significant compared to non-significant findings. Such an approach does not allow for an evaluation of the magnitude of the findings, as in a meta-analysis. Therefore, results from Wagner and colleague's meta-analysis (2015) may provide a more precise estimate of the association between attentional control and MDD in youth.

Only one known study has used event-related potentials (ERPs) from electroencephalography (EEG) to estimate cognitive control deficits associated with MDD in youth. The error-related negativity (ERN) is a negative deflection in the ERP that follows an error on task performance and is associated with monitoring and regulating self-initiated actions (Ladouceur et al., 2012). The ERN is thought to be generated primarily by the anterior cingulate cortex. Associations between the ERN and MDD in adults have been inconsistent (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2010; Ruchsow et al., 2004, 2006). In the only known study with younger participants, results from a sample of youth ages 7 to 17 years indicated that those with MDD had smaller ERN amplitudes compared to healthy controls; further, youth with MDD failed to show the age-related increase in ERN demonstrated in the healthy sample (Ladouceur et al., 2012), suggesting deficits in cognitive control that persist over time.

3.2.2. Attentional control and rumination

The De Raedt and Koster (2010) model predicts that attentional impairments are greater following a stressor (which can include an internal stressor [i.e., rumination] or external stressor) and that rumination is an important mechanism linking attentional control and affective symptoms. These predictions are important because they may explain the inconsistent associations between attentional control and depression reviewed above. Specifically, attentional control may interact with rumination to predict depression. This interaction has also been outlined in the attentional scope model of rumination (Whitmer & Gotlib, 2013). The attentional scope model highlights the importance of mood, proposing that negative mood narrows the number of thoughts, percepts, and actions activated in working memory and/or available for selection from long term memory (Whitmer & Gotlib, 2013). It is thought that negative mood signals potential danger in the environment, narrowing attentional selection and thereby decreasing the likelihood of exploration of the environment (e.g. Fredrickson, 1998, 2001). It follows that a narrowed attentional scope will result in encoding information at the center of attention more deeply and that these representations will be maintained more robustly. Because the amount of information available has become limited, such thoughts and actions are more likely to be repeated, resulting in rumination. Thus, the attentional scope model posits that the association between mood and repetitive thoughts is mediated by attentional breadth. This model allows that people high in rumination may not have broad deficits in cognitive control or attentional control (or inhibition, as posited by De Raedt and Koster's (2010) model). Consistent with this model, higher rumination in adults has been associated with attentional narrowing for self-related information relative to other-related information (Grol, Hertel, Koster, & De Raedt, 2015) and impaired switching in the context of emotional information (De Lissnyder, Koster, & De Raedt, 2012; Koster, De Lissnyder, & De Raedt, 2013). Other studies in adults have also linked higher rumination with better performance on the Stroop and Flanker task (used specifically to measure breadth of attention), suggesting less distraction and more stable maintenance of goal-relevant information (Altamirano, Miyake, & Whitmer, 2010; Zetsche & Joormann, 2011). Of note, these findings contrast with De Raedt and Koster's (2010) model that highlights the importance of inhibition deficits specifically, which is thought to open the "gateway" to rumination.

Although the attentional scope model provides an alternative lens through which to interpret the attentional control findings, support for the model is inconsistent and few studies have examined rumination and attentional control deficits in youth specifically. One study linked rumination with shifting deficits in adolescent boys, but not girls (Wagner, Alloy, & Abramson, 2014), whereas others have failed to find an association with shifting in a mixed sample of boys and girls (Hilt, Leitzke, & Pollak, 2014). Findings from the qualitative review discussed previously (Vilgis et al., 2015) are somewhat consistent with predictions from the attentional scope model in terms of depression (although notably rumination was not included in the review). The authors found mixed support for deficits in working memory and little support for inhibition deficits associated with depressive disorders. Further, only three of ten studies found evidence for deficits in shifting, which contradicts predictions from the attentional scope model. However, given that the studies reviewed by the authors focused on the effects of depression broadly and not rumination specifically, it may be premature to draw conclusions about the fit of the model in youth.

There are several methodological and conceptual issues in this area that may explain the inconsistent findings. First, the influence of mood or negative affect on attentional control remains understudied. This issue has also been discussed as the effect of "hot" compared to "cold" executive function deficits. The term "hot" can refer to affective processing either in the form of emotional stimuli following a stressor or in the context of a negative mood. However, mood and information type are separable variables and the attentional scope model suggest the distinction is critical, as mood narrows attentional scope but the type of information in the center of attention is irrelevant and processed the same (i.e., repetitively). Vilgis et al.' (2015) finding that the associations between depression and hot and cold attentional control did not differ supports the model hypothesis in general (but not the expected deficits in shifting). Additional research examining the effect of mood specifically on attentional scope is needed.

Additionally, results are complicated by the heterogeneity of the "depressed" groups, which sometimes included those with a prior, but not current diagnosis. Medication status also appears to influence findings (Vilgis et al., 2015) and has not been measured in most studies. Future studies would benefit from assessing continuous depression and disorder status variables, as well as medication usage.

3.2.3. Attentional bias and depression

De Raedt and Koster's framework suggests that deficits in attentional control result in increased rumination, which in turn leads to sustained negative affect (2010). In this framework, attentional bias is considered a form of attentional control. Attentional bias studies have significant variability in terms of stimuli format (words vs. faces), exposure duration (150 ms to 20s), emotional content (sad vs. angry), and measurement method, which makes aggregation across studies difficult. The subsequent sections review the current literature on associations between attentional bias and concurrent depression, attentional bias in children at-risk for depression, and the efficacy of attentional bias modification for decreasing symptoms of depression.

Current research suggests that in youth ages 5 to 21 years, attentional bias toward sad stimuli is associated with concurrent depression symptoms and diagnosis (see Platt, Waters, Schulte-Koerne, Engelman, & Salemink, 2017 for a review). Platt et al. (2017) reviewed 21 studies including 2993 children and concluded that youth depression is characterized by attentional biases for negative stimuli. This correlation between attentional bias and youth depression provides necessary but not sufficient support for a causal role of attentional bias.

The association between attentional bias and childhood risk for depression is also supported by current research (Platt et al., 2017) though several factors moderate the nature of the negative bias (toward vs. away) and its association with depression. Some have found main effects for attentional bias away from negative stimuli on depression onset in at risk adolescent girls (Joormann, Talbot, & Gotlib, 2007) and depression symptoms two years later in children age 9 to 13 years (Price et al., 2016). However, others have found that the effect is moderated by other variables. For example, children of depressed mothers with low suppression (avoidance of emotional expression) showed attentional bias *toward* sad faces (Connell, Patton, Klostermann, & Hughes-Scalise, 2013) whereas children at genetic risk (homozygous for the S allele, possession of the S or L_G alleles) of depressed mothers with high suppression showed attentional bias *away* from negative faces (sad or angry; Connell et al., 2013; Gibb, Benas, Grassia, & McGeary, 2009; Jenness, Young, Hankin, 2017). Longer stimulus presentations (greater than 1250 ms) are also associated with greater avoidance of sad faces (Harrison & Gibb, 2015). Thus, although there is evidence that youth with depression and those at risk for depression exhibit attentional bias, the nature of the bias (toward or away stimuli) is moderated by additional variables (Platt et al., 2017).

A key prediction of De Raedt and Koster's (2010) model is that attentional bias is an etiological and maintenance factor for depression, but is not epiphenomenal. However, evidence for this hypothesis is inconsistent. Cognitive Bias Modification (CBM)-attention interventions appear to have no effect on reducing depression in youth. Two meta-analyses of CBM in youth found no statistically significant effect of CBM-attention (Mogoaşe, David, & Koster, 2014) or CBM (attention and interpretation) on depression or other mental health outcomes (Cristea, Mogoașe, David, and Cuijpers, 2015b). Cristea, Mogoașe et al. (2015) concluded that although the training resulted in changes in bias, these changes were not converted into clinical gains. A subsequently published study however, showed that CBM-attention alters both attentional bias (i.e. toward happy faces and away from sad faces) and decreases psychophysiological responses to stress (LeMoult, Joormann, Kircanski, Gotlib, 2016). Although evidence to date suggests that attentional bias modification trainings have largely failed to alleviate depression, this does not rule out the possibility of attentional bias as an etiological and maintenance factor for depression. For example, the limited efficacy of CBM may be due to significant methodological problems (discussed extensively elsewhere; Platt et al., 2017), insufficient intervention dose, and the limited inhibitory control of prefrontal areas over subcortical areas (De Raedt, Vanderhasselt & Baeken, 2015; Phillips, Ladouceur, & Drevets, 2008). Specifically, limited prefrontal cortex resources may interfere with the ability to engage with CBM-attention tasks, reducing the impact of CBM interventions and therefore limiting symptom reduction.

De Raedt and Koster's (2010) hypothesis that attentional control (i.e., attention bias) results in increased rumination has some support in youth. Rumination, induced in young adults ($M = 22.09$ years; $SD = 6.25$) via a negative mood manipulation, was associated with decreased positive attentional bias (i.e. decreased attention toward positive stimuli) but not associated with negative attentional bias on a dot-probe task (Morrison and O'Connor, 2008). The lack of association between attentional bias and rumination may be explained in part by the proposed two-factor structure of rumination (reflection and brooding). When examining brooding specifically, there is a significant relationship between negative depression outcomes (current and future depression; Treynor, Gonzalez, & Nolen-Hoeksema, 2003) and attentional bias for sad faces (Joormann, Dkane, & Gotlib, 2006).

Overall, current research suggests that attentional biases are evident in children with current MDD and/or depression symptoms. Further, attentional biases are associated with depression risk and subsequent depression symptoms/disorder status (Platt et al., 2017). However, literature to date does not support the prediction that the changes in attentional bias after CBM are associated with depression symptom change in youth (Cristea, Mogoașe, et al., 2015). Taken together, CBM-attention findings raise questions about the role of attentional bias in the etiology of depression in youth.

3.3. Neurobiological underpinnings of attention deficits in youth depression

De Raedt and Koster's (2010) model proposes that several biological factors underpin the biological stress reactivity associated with vulnerability for depression. Specifically, the model predicts that HPA axis impairment leads to depression by way of dysregulated serotonergic function, which in turn acts on attentional control. The following sections review research in youth in each of these areas.

3.3.1. Dysregulated HPA system

The HPA axis includes the hypothalamus, pituitary gland, and adrenal glands and plays an important role in regulation stress responses. The hypothalamus secretes the corticotropin releasing hormone (CRH) and arginine-vasopressin (AVP) which travels to the anterior pituitary and causes corticotropic cells to release adrenocorticotropic hormone (ACTH). ACTH then travels via the blood to the adrenal glands. The adrenal cortex releases corticosteroids, including cortisol and dehydroepiandrosterone (DHEA), which spread throughout the body. When cortisol reaches the blood-brain barrier the hypothalamus inhibits secretion of CRH (for an overview see Guerry and Hastings, 2011).

There is significant empirical evidence that HPA dysregulation is associated with major depression in adults (Guerry & Hastings, 2011; Nestler et al., 2002) and a growing body of literature in youth also suggest such an association. Dysregulation within the HPA system has been examined using several methods (Lopez-Duran, Kovacs, & George, 2009) including the Dexamethasone Suppression Test (DST), assessing basal HPA-axis functioning, the corticotrophin releasing hormone (CRH) challenge, psychological probes, and assessing HPA axis functioning in children of depressed mothers (Guerry & Hastings, 2011; Lopez-Duran et al. 2009).

The Dexamethasone Suppression Test (DST) consists of injecting dexamethasone, which suppresses the secretion of CRH, and evaluating the HPA's ability to suppress CRH production. A meta-analysis of 17 studies ($N = 926$) found that youth with MDD experience less suppression of cortisol after the DST compared to controls (Lopez-Duran et al., 2009). Furthermore, inpatient depressed youth have twice the cortisol non-suppression as that outpatient samples (Guerry and Hastings, 2011). Thus, HPA hyperactivity in youth depression may be partially a result of insensitivity to cortisol inhibition or escape from cortisol suppression (Guerry and Hastings, 2011).

In addition to decreased suppression of cortisol, depressed youth are also theorized to have increased basal levels of cortisol. In a meta-analysis of 17 studies ($N = 1332$) basal cortisol levels were significantly higher for depressed children and adolescents than non-MDD controls (Lopez-Duran et al., 2009). Although there is considerable heterogeneity in the methods and findings relating to basal levels of cortisol, evidence from several longitudinal studies suggests that dysregulated HPA both precedes and predicts depression in adolescence (see Adam et al. 2010; Goodyer, Herbert, & Tamplin, 2003; Mathew et al., 2003). For example, basal cortisol levels measured in the morning after awakening are associated with MDD (Adam et al., 2010). Research suggests that an elevated Cortisol Awakening Response in youth precedes and predicts future depression (Adam et al., 2010; Dedovic & Ngiam, 2015; Ulrike, Reinhold, & Dirk, 2013; Vrshek-Schallhorn et al., 2013). Therefore, the Cortisol Awakening Response may represent a risk factor for depression.

Another method for measuring HPA axis activity and cortisol response specifically, is administering CRH. Administering CRH causes the release of ACTH from the pituitary gland and cortisol from the adrenal gland (Lopez-Duran et al., 2009). A blunted response suggests the pituitary gland is under-sensitive to CRH (Lopez-Duran et al., 2009). A recent review found very few studies have used CRH in youth, and those that did found no significant differences between MDD and non-MDD samples response to CRH (Lopez-Duran et al., 2009). More research is needed to further evaluate CRH's potential contribution to examining the dysregulation of the HPA to depression.

Studies evaluating the effect of psychological probes on HPA-reactivity in youth are scarce, however findings suggest differences between children and adolescents with and without MDD (Lopez-Duran et al., 2009). One study found that depressed preschoolers (3 to 5.6 years-old) displayed an increased pattern of cortisol response when using a combination of two stressors (frustration and separation stressors), compared to control children (Luby et al., 2003). All preschoolers were distressed by frustration stressors (e.g. being unable to unlock a transparent box with a desired toy); however, preschoolers who met criteria for MDD (except the duration criterion), exhibited a pattern of increased cortisol production following both the frustration and parental separations stressors separately (Luby et al., 2003). In contrast, the comparison group displayed decreased cortisol levels in response to the separation stressor. These findings provide limited information on the differences in frustration stress response between depressed and nondepressed youth given higher cortisol levels in preschoolers due to the stress of the experiment (Luby et al., 2003; Lopez-Duran et al., 2009). Importantly, the study did not find elevated cortisol reactivity in children with MDD compared to health children despite findings differences in the pattern of response (Luby et al., 2003). In contrast, a study of adolescents diagnosed with MDD found that depressed participants displayed elevated and more prolonged cortisol production in response to a stressor (Trier Social Stress Test) compared to a nondepressed control group (Rao, Hammen, Ortiz, Chen, & Poland, 2008). Contrasting study findings may reflect the differences in the two ages ranges. Puberty may mark a developmental switch in reactivity to stress (Hankin, Badanes, Abela, & Watamura, 2010), given that the prepubertal children displayed cortisol hyporeactivity whereas postpubertal youth exhibited cortisol hyperreactivity (Hankin et al., 2010; Rao et al., 2008).

In conclusion, there is a substantial body of literature to support this tenet of the De Raedt and Koster (2010) model. Data suggest that youth with depression experience a dysregulated HPA axis as evidenced by atypical responses to Dexamethasone Suppression Test, higher baseline cortisol levels, and hyperactive responses to psychosocial stressors. Furthermore, based on the longitudinal studies reviewed above (e.g. Adam et al. 2010; Goodyer et al. 2003), HPA axis hyperactivity is postulated to be a risk factor for, rather than a consequence of, depression in adolescence and children, in concordance with adult findings (Pariante & Lightman, 2008).

3.3.2. Serotonergic dysregulation

De Raedt and Koster's model (2010) predicts that impairments in HPA axis activity result in dysregulated serotonin metabolism, which in turn negatively effects the control of frontal brain regions over amygdala activity. Stressors then trigger amygdala activity, which is not adequately down regulated by the DLPFC which is mediated by the anterior cingulate cortex, resulting in prolonged amygdala activation and sustained negative affect. These components are reviewed in more detail below.

5-HTTLPR, stress, and depression. Evidence suggests that the association between stress and depression is moderated by a polymorphism in the promoter region of the transporter of the serotonin gene (5-HTTLPR; Karg, Burmeister, Shedden, & Sen, 2011). This polymorphism affects the rate of transcription with the short allele transcribing less efficiently than the long allele (Karg et al., 2011). Although HTTLPR is often considered biallelic other variations occur, some of which are important for transcription (Hu et al., 2006). For example, a single-base substitution at the sixth nucleotide has created the L_A and L_G allele subtypes (Hu et al., 2006). Although L_A has high transcription efficiency, L_G has approximately equivalent efficiency as S (Hu et al., 2006). Thus, serotonin transcription efficiency can be categorized into low (SS, SL_G or L_GL_G), medium (SL_A or L_AL_G), and high (L_AL_A; Pérez-Edgar et al., 2010).

A recent meta-analysis including child and adult samples found that 5-HTTLPR moderated the relationship between stress and depression such that the short allele was associated with greater depression risk (Karg et al., 2011). Furthermore, an updated meta-analysis including both child and adult samples also found overall support the moderation of 5-HTTLPR on the relationship between stress and depression (Sharpley, Palanisamy, Glyde, Dillingham, & Agnew, 2014). However, it should be noted that approximately 26% of studies failed to find an effect (Sharpley et al., 2014). Importantly, mean age did not moderate the study's findings (Sharpley et al., 2014). Thus, although fairly robust evidence suggests that 5-HTTLPR moderates the association between stress and depression, other variables also likely influence the association.

The HPA axis and 5-HTTLPR. Although there is strong evidence for the moderating effect of 5-HTTLPR on the relationship between stress and depression (Sharpley et al., 2014), little is known about the mechanisms by which 5-HTTLPR enhances risk for depression in the presence of stressors (Gotlib, Joormann, Minor, & Hallmayer, 2008). To address the mechanisms underlying the risk of depression, studies have investigated the association between 5-HTTLPR and HPA activity. One such study using healthy girls (9 to 14 years of age) found that girls at risk for depression (i.e. homozygous for the short allele) displayed elevated and prolonged cortisol production in response to a stressor compared to girls with at least one long allele (Gotlib et al., 2008). Another study of youth (9 to 15 years of age) reported that cortisol reactivity was more stable over time in youth who were homozygous for the short 5-HTTLPR allele compared to those with a long allele (Hankin, Badanes, Smolen, & Young, 2015). Together these findings suggest older children and adolescents homozygous for the 5-HTTLPR short allele of 5-HTTLPR are more likely to exhibit an elevated and stable response to stress due to a dysregulated HPA activity as measured by cortisol reactivity to stress (Hankin et al., 2015), which may explain how 5-HTTLPR confers risk for depression. In summary, children that are homozygous for the short 5-HTTLPR allele and have a dysregulated HPA axis may be at increased risk for depression. Overall, although there is cross-sectional evidence of an association between the HPA axis and 5-HTTLPR, it is unclear at this time if dysfunctional HPA axis activity mediates the effect of the 5-HTTLPR on depressive symptoms. Additional longitudinal studies are needed.

3.3.3. Serotonin, prefrontal cortex, and attention

De Raedt and Koster's model (2010) proposes that the serotonin system affects emotion regulation via attentional control to negative stimuli and there is evidence of this association in adult samples. For example, healthy adults with a short 5-HTTLPR allele found it more difficult to disengage from sad and happy stimuli compared to adults with two long alleles (Beevers, Wells, Ellis, & McGeary, 2009). Using a dot-probe paradigm, another study of healthy adults reported biased processing of positive stimuli and avoidance of negative stimuli in adults with the LL allele (Fox, Ridgewell, & Ashwin, 2009). Those with the SL and SS allele did not display this pattern (Fox et al., 2009). Another study of female adults found that a stress induction was associated with impaired inhibition of negative emotional information, but only in participants with two short alleles; participants with two long alleles actually showed an improvement in inhibition after stress exposure (Markus & De Raedt, 2011). Thus, the long alleles may increase resilience to depression by facilitating a protective bias toward positive information (Fox et al., 2009; Markus & De Raedt, 2011).

A similar relationship between short 5-HTTLPR alleles and attentional biases to faces has also been demonstrated. One study found that 5-HTTLPR genotype was associated with attentional bias to angry faces using a dot probe task (Pérez-Edgar et al., 2010). Transcription efficiency of 5-HTT—low (SS, SL_G or L_GL_G), medium (SL_A or L_AL_G), and high (L_AL_A) was negatively related to vigilance to angry faces (Pérez-Edgar et al., 2010). The low and medium transcription groups were associated with vigilance to angry faces whereas the high transcription group was not (i.e. lower 5-HTT transcription efficiency displayed greater bias for angry faces). Attentional bias to angry faces has been demonstrated in people who report abuse and those subjected to criticism (Gibb et al., 2011). Angry faces may indicate social rejection which is theorized to promote depression onset in adults and adolescents who may be especially sensitive to social rejection. Furthermore, the opposite pattern was evident for happy faces; children (mean age of 15) with low transcription efficiency exhibited the least bias for happy faces (Pérez-Edgar et al., 2010). Others have also shown that, compared to children with two long alleles, children with one or two short alleles displayed greater attentional bias to angry faces (ages 8 to 12 years; Gibb et al., 2011) and greater negative schematic processing when exposed to a negative mood induction (aged 7 years; Hayden et al.,

2008) than children with two long alleles. In summary, lower transcription efficiency is correlated with greater attentional bias to angry faces in children suggesting lower transcription efficiency may increase depressive symptoms by increasing attentional bias to negative stimuli.

Maternal history of depression may also impact the relationship between 5-HTTLPR and attentional bias. For example, children with maternal history of depression displayed greater attentional avoidance of sad faces (Gibb et al., 2009). Although this may appear contradictory, some evidence suggests adolescents first attend to negative stimuli before engaging in later avoidance to regulate emotions (Harrison & Gibb, 2015). Furthermore, evidence demonstrating a stronger relationship between maternal history of depression and attentional avoidance of sad faces in children with short or L_G 5-HTTLPR (versus a L_A allele) approached significance, $p = .07$ (Gibb et al., 2009). Thus, transcription efficiency may affect neurobiological and information processing responses to threat (Pérez-Edgar et al., 2010).

Given the important role of stress in the development of depression it is also important to examine the potential moderating role of 5-HTTLPR on stress and depression development. A study on a specific type of childhood stress (i.e., maternal criticism) on attentional bias found a moderating relationship of 5-HTTLPR such that greater stress was associated with attentional bias for angry faces but only for children with at least one short allele (Gibb et al., 2011). In summary, serotonin is associated with attentional biases toward negative stimuli in addition to HPA axis dysregulation, which suggests that serotonin transcript efficiency may contribute to both elevated and sustained stress response and attentional biases toward negative stimuli.

3.3.4. Hypofrontality

A number of neuroimaging studies have examined the link between executive function deficits and depression in youth and findings overall suggest that executive functioning deficits are associated with depression in youth. For example, a meta-analysis compared 246 youth with MDD) to 274 healthy controls (age ranged from 4 to 24 years; $M = 18.36$ years; Miller et al., 2015). Youth with depression showed hyperactivity and hypoactivity in brain activity in both task-general and task-specific ways. In negatively valenced tasks, youth with depression showed hyperactivity in the DLPFC, a region strongly associated with cognitive control. Across aggregated tasks, youth with depression also showed hyperactivity in subgenual anterior cingulate cortex (sgACC) and ventrolateral prefrontal cortex across aggregated tasks; hyperactivity was also found in thalamus and parahippocampal gyrus during affective tasks. Conversely, youth with depression showed hypoactivation in the caudate across aggregated tasks and in the cuneus, dorsal cingulate cortex, and dorsal anterior insula during executive functioning tasks. There was also evidence of hypoactivity in the posterior insula during positive valence tasks.

The pattern of neural activation demonstrated by Miller and colleagues' meta-analysis maps onto the Default Mode Network (DMN), a system comprised of two alternating circuits: the Task Positive and Task Negative Networks (Marchetti, Koster, Sonuga-Barke, & De Raedt, 2012). The Task Positive network is associated with external, vigilant attention while the Task Negative Network is associated with internal, self-focused thinking, such as rumination (Buckner et al., 2008; see Marchetti et al., 2012, for full review of the DMN and associations with depression in adults).

Results from Miller et al. (2015) suggest that youth with MDD are characterized by over activation of the Task Negative Network and associated increases in elaborative, self-referential processing. Underactivity in the Task Positive Network, in combination with the overactivity of the Task Negative Network, suggests that youth with MDD may have difficulty deactivating self-focused Task Negative neural activity in order to shift of attention away from rumination toward active processing (Miller et al., 2015). The pattern of hyperactivation in DLPFC in depressed youth suggests that a third network might also be important to consider. The DLPFC is a key region in the central-executive network, which regulates cognitive control. Miller and colleagues conclude that youth with depression have difficulties with emotion regulation despite over activity in DLPFC and VLPFC, which may reflect a compensatory mechanism. Additional study of if these patterns alter as a function of development or symptom course require further study.

3.3.5. Amygdala hyperactivity

Several studies have found associations between amygdala hyperactivity and youth depression using emotional face-matching and facial memory paradigms. A review of emotional face-matching paradigms studies found that youth (13–18 years old) and young adults (19–25 years old) displayed elevated amygdala activation in response to angry and fearful faces compared to healthy controls (Kerestes, Davey, Stephanou, Whittle, & Harrison, 2014). The majority of studies of youth and young adults (12 to 24.5 years old) with depression using the emotional face-matching paradigm reported left-side amygdala hyperactivity (Mingtian et al., 2012; Tao et al., 2012; Yang et al., 2010; Zhong et al., 2011) although another study reported elevated bilateral amygdala activity (Kerestes et al., 2014; Matthews et al., 2008). Additionally, a recent fMRI study, which replicated these previous study findings of elevated bilateral amygdale activity, reported that medication-naïve adolescents with MDD exhibited elevated bilateral amygdala activity in response to fearful faces compared to healthy controls (Hall et al., 2014). Thus, medication history may be an important variable to measure when examining amygdala activity.

In addition to the emotional face-matching paradigm, the link between amygdala hyperactivity and depression has been observed using a facial encoding memory paradigm. Specifically, one study found that adolescent MDD patients displayed heightened left amygdala activation during facial encoding of emotional faces (Roberson-Nay et al., 2006). Adolescents with MDD also show amygdala hyperactivity while engaging in an emotional regulation task, relative to controls (Perlman et al., 2012). Specifically, youth with MDD exhibited greater right amygdala activation while maintaining their emotional response to an evocative image versus attempting to reduce their reaction, relative to healthy controls (Perlman et al., 2012). Similarly, others have found that adolescents with MDD passively viewing fearful faces display greater left amygdala activation than healthy peers (Beesdo et al., 2009). Findings are divergent regarding whether depressed youth display increased right vs left activation while viewing emotional faces; however, more research is needed to understand why such differences might exist.

Importantly, in addition to findings suggesting concurrent associations between depression and amygdala hyperactivity, there is some evidence that such hyperactivity may represent a vulnerability to depression (Kerestes et al., 2014; Pilhatsch et al., 2014). For example, currently non-depressed children of parents with MDD have shown greater amygdala activity, compared to children of non-depressed parents, in response to passively viewing fearful faces (Monk et al., 2008). Similarly, a recent study found that healthy adolescents with a family history of depression displayed greater left amygdala activity to negative stimuli compared to adolescents without a family history of depression (Pilhatsch et al., 2014). Finally, a review of twenty-eight functional brain imaging studies found an association between major depression in youth and abnormal activation in the amygdala and anterior cingulate (Kerestes et al., 2014). In summary, the bulk of the evidence suggests that adolescents and youth with depression display increased amygdala activity to negative stimuli, suggesting greater emotional reactivity; however, additional well-powered studies using consistent methodologies are warranted.

3.3.6. Volumetric differences in the amygdala

In addition to amygdala hyperactivity, recent studies suggest there are volumetric differences in amygdala in pediatric affective disorders. A recent meta-analysis of adult and child unipolar and bipolar mood disorders found that children with bipolar disorder displayed significantly smaller left amygdala volumes than healthy controls and trending smaller right amygdala volumes (Hajek et al., 2009). However, there were no significant differences between pediatric amygdala volumes for unipolar depression compared to healthy controls. Of note, inpatient status was associated with larger left amygdala volumes compared to health controls (Hajek et al., 2009). Medication, severity, or diagnostic comorbidity may explain the association between inpatient status and greater amygdala volumes (Hajek et al., 2009). By contrast, a review of neuroimaging findings in children with MDD suggested smaller amygdala volumes compared to controls (Hulvershorn, Cullen, & Anand, 2011). Although much evidence for associating amygdala volume and depression has been cross-sectional, there is initial longitudinal evidence linking amygdala growth and the onset of a depressive disorder (Whittle et al., 2014). Specifically, reduced normative amygdala growth from ages 12–16 was associated with depressive disorder onset in males, whereas accelerated growth was associated with depression onset in females.

The mixed findings in this area may be due in part to methodological issues. For, example, measurement difficulties limit the power to detect amygdala volumetric differences, and suggested null effects might partially be due to these limitations (Hajek et al., 2009; MacMaster et al., 2008). More specifically, discrepancies may be due to differences in image acquisition, measurement, analysis, and sample characteristics such as medication status and comorbidity (MacMaster et al., 2008). In summary, although some research suggests reduced amygdala volume in pediatric depression, findings have been mixed and additional research with large, adequately powered studies is needed.

3.3.7. Amygdala connectivity

Studies of the sgACC (associated with regulation of emotional behavior), amygdala, and connections to other regions involved in emotional processing and regulation largely suggest aberrant connectivity is associated with pediatric depression (Henje Blom et al., 2016). Studies have demonstrated significant reductions in connectivity between the sgACC and the following network of cortical areas: supragenual, right medial frontal cortex, left superior and inferior cortex, left superior temporal cortex, and insular cortex in depressed adolescents (Cullen et al., 2009). This network is associated with the regulation of emotional behavior (Drevets, Savitz, & Trimble, 2008).

Medication experience may be an important moderating factor affecting amygdala connectivity. A study using medication-naïve adolescents experiencing their first-episode of MDD found elevated functional connectivity between the sgACC and right amygdala compared to healthy controls (Connolly et al., 2013). In contrast, a recent study of adolescents (73% medication-naïve) with MDD without substance abuse disorders reported that adolescents with MDD displayed lower positive resting-state functional connectivity between the amygdala, left hippocampus, and parahippocampus (Cullen et al., 2014). Furthermore, adolescents with MDD had reduced resting-state functional connectivity between the amygdala and brainstem but increased connectivity between the amygdala and precuneus, relative to healthy controls (Cullen et al., 2014). The precuneus is often associated with self-referential processing (Northoff et al., 2006), thus increased connectivity between the amygdala and precuneus may serve to associate "negative" emotions with a person's view of themselves. Although the hypothesis that decreased resting-state functional connectivity between the amygdala and frontal cortex in adolescents with depression was not supported, connectivity deficits between the amygdala and subcortical regions (e.G. *hippocampus*, parahippocampus, and brainstem) were reported (Cullen et al., 2014). The authors suggest the lack of amygdala-frontal connectivity findings in children, in contrast to adults, may be explained by the continued growth of the frontal cortex during childhood (Cullen et al., 2014). Although results from these studies are not conclusive as to the role of medication, some evidence exists that medication may alter functional connectivity which might confound studies of connectivity in youth with a history of medication use (Connolly et al., 2013).

Diffusion tensor imaging (DTI) has also been used to support abnormal amygdala in depression. For example, one study found youth (15 to 19 years of age) with depression displayed significantly lower fractional anisotropy (FA) in the tract connecting the right sgACC to the right amygdala compared to healthy controls (Cullen et al., 2010). Lower FA was also evident in the left hemisphere tract connecting the left sgACC to the left amygdala; however, this was not statistically significant (Cullen et al., 2010). In addition, no voxel-wise comparisons were significant after accounting for whole-brain multiple comparisons (Cullen et al., 2010). It is important to note the study's sample included comorbid mental health issues and participants were not all medication-naïve. These findings provide some evidence for diminished organization of the white matter microstructure in the pathway connecting the sgACC, which plays a role in the modulation of emotional behavior (Drevets et al., 2008), and the amygdala suggesting that the regulatory input from the anterior cingulate cortex to amygdala is deficient (Cullen et al., 2010). This diminished regulation from the anterior cingulate cortex allows for prolonged negative affect characteristic of depression.

A recent review of the neuroscience of adolescent depression summarized the extant literature by suggesting adolescent MDD is marked by hyperactivity of the amygdala and sgACC and deviant connectivity between these two regions and other regions involved in emotional processing and regulation (Henje Blom et al., 2016). However, null findings do exist, including a study which found no differences in functional connectivity between the (sgACC), a prefrontal cortex region, and the amygdala using a seed-based approach to resting-state fMRI between adolescents (15 to 19 years of age) with depression (and comorbid disorders, i.e. 80% comorbid anxiety) and healthy controls (Cullen et al., 2009). Furthermore, there were no correlations between measures of functional connectivity and symptom severity (BDI-II), illness duration, medication status, or comorbid anxiety diagnosis (Cullen et al., 2009). However, most studies suggest sgACC appears to be an important part of altered brain networks associated with pediatric depression (Cullen et al., 2009).

Although there is some evidence of resting-state functional connectivity differences between the sgACC and amygdala, few studies have addressed connectivity differences during emotional processing. For example, one study using a negative emotional processing task, found that medication-naïve adolescents with MDD had increased sgACC to amygdala functional connectivity (right sgACC to left amygdala) compared to healthy controls (Ho et al., 2014). The authors conclude that adolescents with MDD display biased processing of negative stimuli, which may lead to larger imbalances in the cognitive-executive, salience, and resting-state functional brain networks (Ho et al., 2014). However, results from a study using dynamic causal modeling found adolescents (12 to 19 years old) with MDD displayed weaker bottom-up connectivity between the amygdala and sgACC during an emotional face matching paradigm compared to healthy controls (Musgrove et al., 2015). Although these findings contrast with other study findings on connectivity (reduced vs enhanced; Connolly et al., 2013; Ho et al., 2014) they provide further evidence of abnormality in the amygdala-sgACC connection in adolescents with MDD. Overall, across methods, the literature suggests that amygdala to sgACC connectivity is altered in youth with depression. However, most studies to date have been cross-sectional and have not established temporal precedence of depression or changes in brain functioning. Therefore, it is unclear if brain changes are a cause or a consequence of depression.

To understand the etiology of depression, studies are also needed to identify possible connectivity differences for children at-risk for depression. One such study reported that children with a history of MDD, a maternal history of an affective disorder, or both a personal and maternal history of MDD, displayed reduced connectivity between the amygdala and brain regions which are positively correlated with amygdala activity (i.e. limbic regions), compared to healthy controls (Luking et al., 2011). Furthermore, children with a history of MDD or a maternal history of an affective disorder had reduced connectivity between the amygdala and brains regions which are negatively correlated with amygdala activity, (i.e. dorsal prefrontal and parietal cortices), compared to controls (Luking et al., 2011). Reduced connectivity between the amygdala and areas important for cognitive control and emotional regulation (Kanske, Heissler, Schönfelder, Bongers, & Wessa, 2011) may suggest that depressed children are less able to down regulate emotions. The implications of reduced connectivity between the amygdala and other limbic regions is less clear; however, it has been hypothesized that the reduced connectivity between the amygdala and hypothalamus may represent a deficiency in integrating memory and emotional experiences (Luking et al., 2011). This deficiency could contribute to the chronicity of depression as new positive contingencies may not be integrated into memory. Thus, reduced connectivity between the amygdala and prefrontal regions, including the sgACC, may be an important risk factor, and biological marker, of pediatric depression.

4. Activation of negative schema in response to stress

De Raedt and Koster's (2010) integrated framework expands upon Beck's cognitive schema theory to include neurobiological cognitive processes that contribute to depression. Specifically, stressors are thought to activate both latent schemas and the HPA axis. Thus, schemas are activated when stressors arise. These schemas, paired with dysregulated biological stress response, attentional deficits, and rumination are thought to contribute to depression.

Schemas are cognitive structures that help to organize information from the environment into attitudes and assumptions about the self, others, and the world. Schemas develop from experiences and impact the development of a worldview, interpretation of events, and response (Beck, 1967). Though developed early in life, schemas may not become activated until exposure to a stressor later in life, suggesting a diathesis-stress model (Ingram, Miranda, & Segal, 1998). Childhood and adolescence are periods with substantial interactions with friends and family members and as a result, are also peak times for the development of cognitive schemas. Beck's cognitive schema theory suggests the activation of schemas leads to biased information processing such that negative schemas lead to an attentional bias for negative aspects of events, negative interpretations of experiences, a lack of attention toward positive events (Beck, 1967). The role of schemas in adult depression has generated considerable research support (Abramson et al., 2002; Joormann, 2009). Namely negative beliefs about the self, others, and the world are thought to be an etiological factor for depression (Beck, 1967; Dozois & Beck, 2008).

Negative schemas are also associated with youth depression. For example, children as early as school age report negative beliefs about themselves, others and the world, with depressed children showing similar negative thinking patterns as adults with depression (e.g., Kaslow & Racusin, 1990). Further, children with higher levels of distress report greater negative beliefs about themselves, others, and the world (Kaslow, Stark, Printz, Livingston, and Ling Tsai, 1992). Negative schemas in childhood include unhappiness with self, negative beliefs about the future, and negative beliefs about problem solving ability (Weisz, Sweeney, Proffitt, & Carr, 1993; Asarnow and Bates, 1988; McCauley, Mitchell, Burke, & Moss, 1988). For example, depressed children were more likely to view themselves as less academically competent than nondepressed peers, despite equivalent cognitive and achievement levels (Asarnow, Carlson, Guthrie, 1987). Longitudinally, there is evidence, in a community sample of children followed from fourth to sixth grade, that self-reported depressive symptoms predicted an increase in negative self-schemas and underestimates of competence (McGrath & Repetti, 2002). A review (Hankin, 2012) of cognitive styles and dysfunctional attitudes in children, found strong support for negative cognitive/inferential style, dysfunctional attitudes, and rumination as risk factors that interact with stress to predict depressive symptoms in youth. Additionally, some research suggests that cognitive vulnerabilities for depression produce larger effects in adolescents compared to children, possibly due to continued cognitive/neurological development during childhood including the ability for abstract reasoning and formal operational thought (Cole and Turner, 1993; Lakdawalla, Hankin, & Mermelstein, 2007), though this theory remains understudied.

Beck's original cognitive model of depression proposed that severe life events (death of a loved one or loss of a job) precipitate depression (Beck, 1967); however, De Raedt and Koster's model (2010) suggests that milder stressful life events provide an alternate pathway to depression onset and maintenance in vulnerable adults (Kendler, Kuhn, Vittum, Prescott, & Riley, 2005). Importantly, progressively milder stressful events may trigger successive episodes of depression, suggesting a “kindling” effect (Kendler, Thornton, & Gardner, 2000; Monroe & Harkness, 2005). This effect has been shown in children (Jacobs, Reinecke, Gollan, & Kane, 2008; Scher, Ingram, & Segal, 2005) and adolescents (e.g., Technow, Hazel, Abela, & Hankin, 2015). For example, one study of adolescents followed over two years found that dependent stressors (e.g. relationship problems) were stronger predictors of depressive symptoms following the onset of a depressive disorder (Technow et al., 2015). This kindling effect, central to De Raedt and Koster's model (2010), can be attributed to the differential activation hypothesis (Teasdale, 1988), which suggests that each depressive episode strengthens the association between negative mood and negative thinking patterns. This hypothesis has been supported in depressed adults, with reduced monitoring of schema-associated thoughts and feelings linked to chronic negative thinking patterns (Sheppard & Teasdale, 2000). Further, in adults, the association between decreased dysfunctional thinking and partial remission of depression is mediated by increased metacognitive monitoring of negative schemas (Sheppard & Teasdale, 2004). The absence of metacognitive monitoring also predicts MDD and depression severity in children and adolescents (Rawal, Collishaw, Thapar, & Rice, 2013).

Relatedly, although the influence of schemas and HPA axis dysregulation on depression has been described as temporally synchronous yet distinct (De Raedt & Koster, 2010), the pairing of high and sustained cortisol with negative schemas may create and maintain an association between stress response and negative thought content. Thus, negative thought content and stress response may serve as retrieval cues for one another and influence their chronicity. Furthermore, although stress moderates the relationship between schemas and depression, there is also a bidirectional relationship between stress and depressive symptoms in adults (Grant, Compas, Thurm, McMahon, & Gipson, 2004).

Gender differences in depression prevalence may be partially explained by types of stress. One study found that the increased level of depressive symptoms in adolescent girls is partially accounted for by more self-reported interpersonal dependent stressors (e.g. end of relationships, disagreements with friends, etc.) which were also associated with prospective rumination (Hamilton, Stange, Abramson, & Alloy, 2015). Thus, interpersonal dependent stressors are a risk factor for the development of cognitive vulnerabilities to depression in adolescence. Although this vulnerability affected both boys and girls equally, girls experienced more perceived interpersonal dependent stressors. Furthermore, greater response to interpersonal dependent stressors in adolescence paired with a ruminative response style may partially explain higher rates of depressive symptoms in girls than boys (Hamilton et al., 2015).

Importantly, individual cognitive and neurobiological vulnerability factors (e.g. latent schemas or a ruminative response style) have limited explanatory power for depression, suggesting that cognitive vulnerability variables should be examined at multiple levels of analysis (Hankin, 2012; Jacobs et al., 2008). For example, although schemas interact with stress to produce depression (Hankin, 2008), there are various other moderating factors affecting stress and depression including HPA axis hyperactivity, a ruminative response style, and serotonin transcription efficiency. Although studies focused on the contribution of negative schemas, ruminative response styles, and interactions with stress are useful, a deeper understanding of the neurobiology will increase the understanding of the etiology and maintenance of depression. For example, as previously reviewed, HPA axis hyperactivity is evident in children at-risk for depression suggesting it is not only the presence of stressors, but a child's existing stress response, which contributes to depression. Thus, an existing dysregulated HPA axis, in addition to low serotonin transcription efficiency, in children at risk for depression, produces an exaggerated stress response. This stress response is then paired with the depressogenic schema during stressors.

5. Summary

Depression is one of the most common mental health conditions in children and adolescents and is characterized by a relapsing and remitting course that often begins in youth (e.g., Center for Behavioral Health Statistics and Quality, 2016; Costello et al., 2002). Therefore, it is critically important to identify factors early in development that may explain an increasing vulnerability for depression over the lifespan. The current review was designed to integrate the literature on cognitive and neurobiological factors associated with youth depression, focused specifically on the model proposed by De Raedt and Koster (2010).

Overall, there is evidence to support the core tenet of De Raedt and Koster's (2010) model. In general, evidence suggests that youth with MDD perform more poorly on measures of attentional control than those without MDD (Wagner et al., 2015). This observation, in combination with the expected normative increase in attentional control from early childhood to adolescence in healthy individuals (Amso & Scerif, 2015; Crone & Steinbeis, 2017; Diamond, 2013) suggests that a neurocognitive scar associated with depression in youth may begin to develop early. However, there are only a few longitudinal studies of the development of depression in youth and more research is needed to specifically examine the relationship between depressive episodes and attentional control.

A specific prediction of the model is that attentional impairments are greatest in the context of negative information relative to positive or neutral information. More specifically, the model proposes that attentional control deficits are characterized by a failure of the DLPFC to down-regulate amygdala activity via the anterior cingulate cortex. The bulk of the evidence indicates that youth with depression show deficits in attentional control and attentional bias when processing negatively valenced information (Ho et al., 2014; Kerestes et al., 2014; Luking et al., 2011; Platt et al., 2017; Miller et al., 2015; Wagner et al., 2015) and altered connectivity between amygdala and attentional control regions when processing negative emotions (Ho et al., 2014; Musgrove et al., 2015) and at rest (Luking et al., 2011). Therefore, the preponderance of the literature supports the basic tenet that youth with depression have attentional deficits associated with negative information (see Vilgis et al., 2015 for exceptions).

Evidence for the model prediction that rumination is an important mechanism linking attentional control and affective symptoms via impaired inhibition is weak. Both De Raedt and Koster's model (2010) and the attentional scope model (Whitmer & Gotlib, 2013) predict that attentional control is altered by negative mood/stress and increases the likelihood of rumination. The models differ slightly in that De Raedt and Koster (2010) conceptualize deficits in inhibition as a central “gateway” (p. 60) that fails to prevent negative information from entering working memory, which increases rumination. On the other hand, the attentional scope model proposes that deficits in shifting and disengaging attention, but not deficits in inhibition or maintaining information in working memory, increase rumination. We are unaware of any studies of rumination as a mediator of the association between attentional control and depression in youth (although there is evidence of such an association in adults (e.g., Demeyer, De Lissnyder, Koster, & De Raedt, 2012) and so this prediction cannot yet be evaluated in youth. Cross-sectional neuroimaging findings suggest that youth depression is associated with greater DMN activity during negative tasks and less activity in task-positive networks (Miller et al., 2015), indicating difficulty disengaging attention from rumination and shifting attention toward external demands. However, behavioral evidence suggests that associations between inhibition and rumination are gender-specific to boys (Hilt et al., 2014) or nonsignificant (Wagner et al., 2015). The limited amount of available literature in this area suggests that although negative information, attention, and rumination may be related, their causal relationships are unclear.

Overall, there is little evidence for De Raedt and Koster's (2010) model assumption that attentional bias is an etiological and maintaining factor for depression, not epiphenomenal to the disorder. Although evidence in adult samples is mixed (Browning, Blackwell, & Holmes, 2012; Cristea, Kok, & Cuijpers, 2015), a meta-analysis of CBM in youth found no effect on attentional bias on depressive symptoms (Mogoaşe, David, & Koster, 2014), indicating that any effect of CBM on biases are not converted into clinical gains. It has been suggested that changes in bias are not a sufficient, and possibly not necessary, condition for symptom change (Cristea, Kok, and Cuijpers, 2015a; Cristea, Mogoaşe, David, and Cuijpers, 2015b). This finding casts doubt on the hypothesis that attentional bias is causally related to depressions symptoms. However, De Raedt and Koster's model (2010) also allows that attention bias may not play a role in the etiology of all depressive episodes, and may interact with attentional control deficits to predict depression. Although we are not aware of any research examining the moderating effect of attentional control on attention bias and its association with depression in youth, other moderating variables have been proposed to explain statistically insignificant main effects between CBM procedures and depression reduction. Thus, although there is not clear evidence to support this component of the model, it is possible that other moderating effects may be masking the causal influence of cognitive biases on depression.

De Raedt and Koster's model (2010) makes several predictions that require additional study. First, the model predicts that attentional impairments are greater following a stressor, which can include an internal stressor (i.e., rumination) or external stressor. Very few studies have tested this hypothesis. One study found that undergraduates reported worse working memory following a stressor and that post-stress working memory deficits interacted with rumination to predict depression symptoms (Quinn & Joormann, 2015). Others have shown that stress interferes with inhibition in adult females with two short 5-HTTPLR alleles (Markus & De Raedt, 2011). However, to our knowledge this hypothesis has not been directly tested in youth by either measuring attention pre- and post- experimental induction of a stressor/rumination or by longitudinally assessing attention before and after a naturally occurring stressor. Second, we were unable to identify any studies that addressed the prediction that episodes of depression are associated with increasing attentional impairments that persist after remission. Although there is evidence that youth with MDD failed to show normative age-related increase in cognitive control (Ladouceur et al., 2012), this study was not longitudinal and the effects of depressive episodes on subsequent cognitive control were not examined. There is also evidence from related studies that prior episodes of depression are associated with both dysregulated cortisol responses to stress (Mazurka, Wynne-Edwards, & Harkness, 2016) and increase the likelihood of subsequent depression following stress Technow et al., 2015), these studies did not examine the role of attention or cognitive control. Thus, it is possible that deficits in attention explain the effects of a prior episode on dysregulated stress response and subsequent depression, however, this has yet to be shown empirically in youth. Third, we were unable to evaluate the prediction that the influence of attention bias on depression increases as attentional control diminishes with each depressive episode. This would require longitudinal studies of the interaction of both attentional control and attention bias on depression in youth. There is evidence that cognitive control and attention bias are associated with depression cross-sectionally (Platt et al., 2017; Wagner et al., 2015); however, no known studies have examined their relative contributions or interaction effects on the prediction of depression over time.

6. Clinical implications

The findings of the current review suggest that clinical intervention studies designed to target both cognitive and biological (i.e., stress reactivity) variables in youth may be especially helpful in decreasing vulnerability to depression. Delivering cognitive interventions during an early developmental window may take advantage of high levels of natural neuroplasticity given that attentional abilities are still developing throughout adolescence and trait-like rumination has not yet stabilized (Abela, Brozina, & Haigh, 2002). Although evidence for the overall efficacy of CBM appears mixed (e.g. Cristea, Kok et al., 2015; Cristea, Mogoașe et al., 2015), cognitive control training may offer another approach to cognitive training. Cognitive control training packages that include the Paced Auditory Serial Addition Task (PASAT; Hoorelbeke, Koster, Vanderhasselt, Callewaert, & Demeyer, 2015) or a combination of the PASAT and the Attention Training Technique (Knowles, Foden, El-Deredy, & Wells, 2016; Siegle, Ghinassi, & Thase, 2007) have been shown to decrease depression and rumination in adults and may be effectively delivered to youth as well. Whether this training will show efficacy as a stand-alone intervention or if it is best used as an adjunct treatment to optimize other cognitive behavioral interventions for youth depression requires additional study.

Findings from the current review require continued empirical investigation and support the value of a highly integrated view of cognitive and biological vulnerabilities to depression. This view attempts to bridge the gap between seemingly discrepant theories of depression with limited upper ends of explanatory power (Hankin, 2012). Thus, for clinicians to be most helpful to children with or at-risk for depression, treatments should ideally target each component of the model. A therapy based only on reducing negative schemas, based on this review, may not be as effective as a multi-faceted one that targets schemas, attentional bias, HPA dysregulation, perceived stressors, and/or amygdala dysregulation.

7. Limitations and future directions

There are several important areas for future research. First, much of the research included in this review is cross-sectional and therefore unable to address questions about the effects of depression on normative development of attentional control and vice versa. Therefore, it is not clear if cognitive control deficits represent a causal vulnerability factor to the development of depression, as predicted by De Raedt and Koster's model (2010), or if the associated deficits are the result of depression or rumination, as suggested by other models, such as the resource depletion account (Hertel, 1998; 2004; Levens, Muhtadie, & Gotlib, 2009). Further, it is unclear if such deficits, even if initially the result of depression, go on to represent a vulnerability for later depression. For example, one study found that executive functioning deficits predict later depressive symptoms and the strength of the effect increased with age (Snyder & Hankin, 2016), suggesting there may be longer-term effects of attentional control on affective symptoms not captured by cross-section studies. Conversely, others have shown that depression was associated with stunted development of later reactive control (Vijayakumar et al., 2016) and failure to show the age-related increases in the ERN demonstrated by the healthy group (Ladouceur, Slifka, Dahl, Birmaher, Axelson, and Ryan, 2012). Thus, the effects of attentional control deficits on depression over time, and their mutual risk for worsening one another, remains an understudied but important area for future research.

Second, much of the work to date has focused on a single level of analysis. To have a more complete understanding of the vulnerability for depression in youth, studies that begin early in life and follow children across development while measuring multiple constructs are necessary. As Hankin's (2012) review points out, what appear to be upper limits on our ability to predict and explain variance in depression may reflect an overly narrow focus on a small set of variables or one level of analysis. Progress is more likely if models integrating across levels and incorporating several constructs, across the lifespan, are proposed and tested. De Raedt and Koster's model (2010) is a promising model; however, it is by no means the only model. Addition frameworks should also be developed, tested, and compared in order to move the field forward.

Also of note, is the striking lack of diversity in the currently available research. As is the case across much of the literature, most of the studies reviewed here focused on non-Hispanic, White, and middle to upper-middle class samples from urban and suburban areas. There is a need for additional study of underserved and under-represented groups, including racial, ethnic, and sexual minority groups and those living in poverty and in rural areas. Not only are these groups understudied, which is problematic in general, but there is evidence that these groups are at even greater risk than majority groups. For example, rates of depression are greater in members of ethnic and racial minority groups in adult samples (Dunlop, Song, Lyons, Manheim, & Chang, 2003) and rates of suicidal behavior are greater in sexual minority groups (Cramer et al., 2017). Further, rates of suicide are nearly twice as high for rural compared to urban youth (Fontanella et al., 2015). Given these disparities, it is important to study factors that may explain increasing vulnerability early in life.

Third, the proposed model may be applicable transdiagnostically to other affective disorders, especially anxiety disorders (De Raedt & Koster, 2010). At the heart of the model is the prediction that deficits in attention allow for repetitive negative thinking (i.e., rumination) which results in sustained negative affect and ultimately disorder status. Although rumination is a key feature of depression, a growing body of work with adult samples indicates that repetitive negative thinking is a transdiagnostic construct relevant to as many as 13 disorders including depression, the anxiety and related disorders, eating disorders, psychosis, and alcohol use disorder (Ehring & Watkins, 2008). There is also evidence that many disorders are characterized by attentional control deficits, including schizophrenia, post-traumatic stress disorder, attention deficit hyperactivity disorder, substance use disorder, and anxiety disorders (Snyder, Miyake, & Hankin, 2015). Anxiety disorders are also associated with dysregulated stress responses (e.g., (Faravelli et al., 2012), similar to those outlined in the model. Therefore, there is reason to think the core tenets of the cognitive aspects of the model may be applicable to a broad range of symptoms although the biological predictions may require additional study.

Declarations of interest

None.

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