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Practitioner Review: Dysphoria and its regulation in child and adolescent depression

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Abstract

Background—By emphasizing the importance of emotions, the "affect revolution" in how human behavior is conceptualized has inspired a new generation of studies on dysphoric experience and its regulation in clinical depression, and novel efforts to characterize the precursors of affective disorders in juveniles at familial risk for depression.

Method—We review clinical, behavioral, and functional neuroimaging studies of dysphoric experience and its regulation in depressed children and adolescents, and in juvenile offspring of parents with histories of clinical depression. We discuss the implication of the literature in the context of maternal depression.

Results—Findings confirm the high rate of clinically significant dysphoria in depressed children and adolescents and reveal notable affective lability in daily life as a function of context and activity. Findings also show that depressed youngsters have problems in attenuating dysphoria. Similarly, never-depressed offspring at familial risk for depression display problems in mood repair and impaired mood repair mechanisms. Brain neuroimaging findings indicate that, overall, depressed and high-risk youngsters differ from never depressed controls in neural functioning (activation, connectivity) both at rest and in response to emotion triggers.

Conclusion—The evaluation of depressed youngsters should include questions about reactivity of dysphoric mood to the changing contexts of daily life and about how they manage (respond to) their own sadness and distress. The resultant information may help the clinician to re-structure a young patient's day for the better and identify helpful mood repair responses. Evidence of impaired mood repair mechanisms in youngsters at high-risk for depression suggests the need for early intervention. But interventions must consider that many depressed and high-risk children have depressed mothers, who may be constrained in their ability to help offspring's emotion regulation efforts. To optimize treatment response of offspring, mothers of depressed children should therefore be routinely screened for depression and treated, as warranted.

Keywords

Dysphoric experience; emotion regulation; depression

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Introduction

The 'affect revolution' in how researchers think about human behavior has placed emotions at the center of normal and psychopathological functioning (Adrian, Zeman, & Veits, 2011; Dennis, 2010) and is reflected in the re-conceptualization of depression as a *disorder that derives from impaired emotion regulation* (e.g., Gross & Muñoz, 1995; Joormann & Gotlib, 2010; Tomarken & Keener, 1998). Correspondingly, there has been growing interest in studying how depressed individuals experience and self-regulate sadness and dysphoria, and the interplay of physiological and behavioral-psychological processes and their context, which support these affective processes.

Scope of this review

In this review, we focus on clinical, behavioral, and neuroimaging studies of dysphoric emotion experience and its regulation in pediatric depression, which were inspired by the affect revolution and published (with a few exceptions) since about the year 2000. We focus on two populations: (a) clinically depressed children and adolescents and (b) juveniles at high-risk for depression owing to having a parent with a history of depression. Because children of parents with depression histories will develop major depressive disorder at rates that reach 65% by the time they are in their 30's (Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997; Weissman, Wickramaratne et al., 2006), studying such offspring *before* they become depressed may yield clues about the precursors of mood disorder.

The goal of this review is to answer the following questions: What do we know about dysphoric emotion experience in clinically depressed and high-risk youngsters? Do depressed and high-risk youngsters differ from normal (control) peers in how they self-regulate dysphoria and the mechanisms that support such regulatory responses? Are there differences across depressed, high-risk, and control youths in how dysphoric experience, its regulation, and related mechanisms are represented in the brain? In the Discussion, we consider the results and implications of our review in the context of maternal depression and its impact on offspring's functioning and treatment response.

Some definitions

Sadness and dysphoria—Persistent and notable sadness and loss of joyfulness have long been regarded as cardinal symptoms of depressive illness (currently called major depressive disorder) and have been thus enshrined in recent psychiatric diagnostic systems (American Psychiatric Association, 1994, 2013; World Health Organization, 1992). In this review, we often refer to the predominant negative affect in clinical depression as "dysphoric experience" or "dysphoria," by which we mean the constellation of sadness, anhedonia, and associated emotional distress. Although the DSM system (e.g., American Psychiatric Association, 1994) has identified irritability as an acceptable manifestation of disordered mood in depression prior to adulthood, it is not included in our definition of dysphoria because we believe that its exclusively reactive nature and temporal characteristics render it different from sadness, anhedonia, and distress.

Emotion regulation—Experts in the field agree that the ability to regulate (modulate) emotion in ways that are appropriate to one's context is integral to psychological adjustment, and yet, no single definition of this construct has been uniformly accepted (e.g., Adrian et al., 2011; Fox, 1994; Gross, 1998). There also have been questions about whether an emotion and its regulation are separable phenomena (e.g., Goldsmith & Davidson, 2004; Gross & Feldman Barrett, 2011; Thompson, 2011), the goals of regulation (Carver & Scheier, 1990; Thompson, 1994, 2011), and whether regulation can be distinguished from culturally mandated overt expression of affect (display rules) and unconscious defense mechanisms (Gross, 1999; Thompson, 1994).

The most often cited definitions (Gross, 1998, 1999; Thompson, 1994, 2011) emphasize that emotion regulation is a multi-component process, which alters an emotional reaction, and has physiological, experiential, expressive, and behavioral features. According to Thompson (1994, 2011), emotion regulation entails "extrinsic and intrinsic" processes that are involved in "monitoring, evaluating, and modifying emotional reactions." The process-view of Gross (1998, 1999) has emphasized that emotion regulation can start before a given emotion has been instigated (antecedent-focused) or can target the emotion after it has onset (response focused). However, there is general agreement that, in addition to its physiological basis, emotion regulation: a) is intertwined with the context of the person experiencing the affect and is developmentally mediated (e.g., Bonanno, 2001; Cole, Martin, & Dennis, 2004; Fox, 1994; Kovacs, Joormann & Gotlib, 2008; Ochnser & Gross, 2007; Rothbart, Sheese, Rueda & Posner, 2011; Thompson, 2011), b) can involve cognitive processes, overt behaviors, interpersonal interactions, or the physical senses (e.g., Larsen, 2000; Parkinson & Totterdell, 1999), and c) can be automatic and without awareness or conscious and willful (Bargh & Williams, 2007; Mauss, Bunge & Gross, 2007).

In the present article, we build on the working definition of Cole, Martin and Dennis (2004), namely, that emotion regulation refers to *changes in an activated emotion*. Specifically, regardless of what triggered the affect, and regardless of its valence (positive or negative), regulation involves processes and responses (on multiple levels) that serve to attenuate, diminish, maintain, or prolong and strengthen the affect in question (Cole et al., 2004). However, as we now note, our focus is on one specific affect experience and its modulation.

Mood repair—While research articles about depressed or depression-prone individuals usually refer to emotion regulation problems in general, the studies themselves focus on depressed mood and responses than can attenuate or exacerbate it (e.g., Ehring, Tuschen-Caffier, Schnulle, Fischer & Gross, 2010; Joormann, Siemer & Gotlib, 2007). This research focus is consistent with the fact that, while depressed patients are likely to vary in how easily they manage various discreet emotions, impaired ability *to reduce sadness and dysphoria* is a key problem for *every depressed individual.* The process of reducing or overcoming sadness and distress has been called *mood repair* (Isen, 1985; Josephson, Singer, & Salovey, 1996; Morris & Reilly, 1987), which is the term we use in this article.

According to the functionalist view, a person's goal in a given context determines whether a given emotion regulation strategy is regarded as good or bad, helpful or not, adaptive or maladaptive (e.g., Gross & Thompson, 2007). However, because we focus on emotion

processes in *depression*, we have taken the position that regulatory responses, which allow a person to attenuate or decrease dysphoria are *adaptive*, while responses that maintain or exacerbate dysphoria are *maladaptive* (Kovacs et al., 2008; Kovacs, Rottenberg, & George, 2009). Further, adaptive mood repair responses have to facilitate the person's functioning both in the short and long run. Here are some examples of adaptive responses to dysphoria that can help a youth to attenuate it: seeking parental (physical or verbal) comforting (Morris, Silk, Steinberg, Myers, & Robinson, 2007), engaging in physical exercise (Augustine & Hemenover, 2009), refocusing attention in various ways away from the distress experience (Erber & Erber, 2000), or cognitively re-appraising in a less depressogenic fashion the meaning of a dysphoric trigger or experience (e.g., Perlman et al., 2012). An extensively researched *maladaptive response* to sadness is depressive ruminating (being preoccupied with and mulling over one's feelings of misery, its causes, and implications), which invariably prolongs dysphoric mood (reviewed by Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Rood, Roelofs, Bogels, Nolen-Hoeksema, & Schouten, 2009). According to experimental studies of children and youngsters, dysphoria also tends to be prolonged when young subjects become behaviorally passive or socially withdrawn in distress provoking situations (e.g., Reijntjes, Stegge, Terwogt & Hurkens, 2007; Silk et al., 2011).

Mood repair mechanisms—There are many different ways in which human beings can repair their own negative mood (Parkinson & Totterdell, 1999; Rippere, 1977; Thayer, Newman, & McClain, 1994), but the underlying mechanisms are likely to be limited in number. One mechanism that appears to underpin a variety of mood repair responses is refocusing of attention away from the experience of distress (Kovacs & Lopez-Duran, 2010). Indeed, Erber and Erber (2000) have maintained that the easiest way to attenuate sadness is to get involved in something that engages attention, whether "doing crossword puzzles or yard work" (p. 144). There also is substantial evidence of the key role of attention in the developmental unfolding of self regulation mechanisms during the early years of life (Rothbart et al., 2011).

Another mechanism that appears to subserve various mood repair responses is the ability to draw upon one's capacity for positive affect such as joy and pleasure (Kovacs & Lopez-Duran, 2010). The capacity for positive affect is regarded as an important and highly adaptive trait (e.g., Fredrickson, 2001) and harnessing it for mood repair should be adaptive. Mood repair responses in which hedonic capacity or hedonic reserve plays a role include engaging in play activity in order to feel better, positive humor in the face of distress, or recalling happy personal memories (e.g., Joormann, Cooney, Henry, & Gotlib, 2012; Samson & Gross, 2012). In this article, we use the terms *hedonic capacity* or *hedonic reserve* to refer to a person's innate capacity to experience a continuum of positive emotion, with maximal pleasurable (hedonic) experiences representing one end-point and total lack of positive emotions (anhedonia) the other end-point.

We now turn to the literature on dysphoric affect experience, its regulation, and related mechanisms in depressed and high-risk youngsters, and then examine information about how these constructs are represented in the brain.

Dysphoria and mood repair: clinical and behavioral perspectives

One source of information regarding affect experience in pediatric depression has been standardized, clinical, usually multi-informant diagnostic interviews that query about the frequency and intensity of mood symptoms. Experience sampling methods (ESM), also called ecologic momentary assessment (EMA) (aan het Rot, Hogenelst & Schoevers, 2012; Shiffman, Stone & Hufford, 2008) have provided a more nuanced picture of affect landscapes and the temporal dynamics of specific emotions. ESM or EMA involves repeated collection of data on affect and related matters in daily life by contacting subjects (usually electronically) at random times, typically 8 to 10 times per day, for several days (e.g., Merrick, 1992).

Mood repair has been assessed in various ways: having youngsters respond verbally to vignettes of negative affect experience, creating a distressing scenario in the laboratory and observing subjects' responses, instructing youngsters to engage in the laboratory in a particular repair strategy after negative mood induction, or self- or parent-rated questionnaires. Likewise, multiple strategies have been used to assess the mechanisms of interest (attention deployment and hedonic capacity) including: laboratory-based behavioral observation, self- or parent-rated questionnaires, performance on standardized tests, or experiment-specific tasks, that have included gambling or other types of rewards to assess hedonic response.

Depressed children and adolescents

Affect experience—According to ratings based on diagnostic interviews, affect experience in clinically referred (7- to 17-year-old) youngsters with major depressive disorder (MDD) is characterized by pervasive dysphoria: from 69% to 78% experience clinically significant depressed mood and 45% have anhedonia (n=559, Baji et al., 2009; n=135, Barbe et al., 2005). Anhedonia also was quite prevalent (near 60%) among pre-schoolers (n=55) with MDD (Luby et al., 2003). The rates of these mood symptoms apparently rise in older teenagers, as 98% of MDD cases in a community-based study (n=1,709; aged 14- to 18-years) had depressed mood and 77% had anhedonia (Lewinsohn, Rohde & Seeley, 1998). Irritability, a criterion mood symptom for major depression in children and adolescents (but not in adults) starting with the DSM-III (American Psychiatric Association, 1980), was present in 65% to 80% of various samples of young patients with MDD (Baji et al., 2009; Barbe et al., 2005).

Studies using EMA have uncovered interesting information about the temporal dynamics of affects. There is convergent evidence that depressed youngsters (aged 7- to 19-years) experience more frequent, intense, and enduring states of sadness, dysphoria, and anger, and less happiness, than do non-depressed controls (Merrick, 1992; Sheeber, Allen, Davis, & Sorensen, 2000; Sheeber et al., 2009; Silk, Steinberg, & Morris, 2003; Silk et al., 2011). One particularly important finding has been that, in everyday life, negative affect among diagnosed depressed youths is labile and dependent on context and activity. Merrick (1992) reported that, while sadness and anhedonia were experienced on a daily basis more often and more intensely by clinically depressed 11- to 18-year olds (n=7) than remitted depressed youths (n=7) or normal control peers (n=14), mood tended to fluctuate in all three groups.

Additionally, sensitivity to social context was greatest among the depressed youths: they were the most dysphoric when alone and notably less sad when with family or friends. These findings were confirmed and extended by Silk and colleagues (2011), who used EMA with 7- to 17-year-old MDD patients (n=47) being treated for depression. While, overall, depressed patients were sadder than were controls, they reported notably less sadness when they were with peers and engaged in some type of activity, compared to when they were alone or with their families, and when they did nothing. Although treatment led to increased positive affect and reduced negative affect, depressed patients *continued* to report a 2:1 ratio of these affect states (Silk et al., 2011) whereas 3:1 is optimal. The later ratio derives from work by Fredrickson and colleagues, who found that the 3 to 1 ratio of positive to negative emotions in daily life is the "tipping point" at or above which people experience excellent mental health and "optimal functioning" (Fredrickson & Losada, 2005).

Mood repair—The high rates of clinically significant sadness in depressed youngsters indicate that they are having difficulties shifting out of the depressed state (Sheeber et al., 2000), which suggest problems in mood repair. Indeed, when tested via vignettes of distressing scenarios, depressed children are less likely than are non-depressed peers to choose mood repair responses that alleviate negative affect, are more likely to select responses that worsen or intensify it, or not even attempt mood repair (e.g., Garber, Braafladt, & Weiss, 1995; Reijntjes et al., 2007). Young patients with MDD (aged 7- to 14-years) also have been found to have significantly larger maladaptive mood repair response repertoires than age-matched controls (Tamas et al., 2007). However, one study of children (aged 7- to 11-years; n=26) with prior MDD (in apparent remission) reported that they did not differ from high-risk peers or normal controls on a 12-item questionnaire of sadness management, which focused on overt expression, reactivity, and a few specific regulatory responses (Luking et al., 2011).

What do we know about *specific* mood repair response use among depressed youngsters? There is some evidence that more severe depression makes it less likely that youngsters will employ adaptive cognitive strategies to alleviate distress, such as neutral reappraisal of a depressogenic stimulus (Betts, Gullone & Allen, 2009). Depressive rumination, which is often seen in clinical depression (Nolen-Hoeksema et al., 2008) has been shown to increase depressed mood in already depressed teenagers (Park, Goodyer, & Teasdale, 2004). "Co-ruminating" with peers, which entails mutual dwelling on problems and negative emotions to the exclusion of other matters, also is more common among children with depression histories (aged 9- to 14-years; n=19; 83% in remission) than among typical peers (Stone, Uhrlass, & Gibb, 2010) and has been found to predict a new depressive episode among 11- to 15-year olds (Stone, Hankin, Gibb, & Abela, 2011).

To provide further information about mood repair in pediatric depression for the present article, we updated and extended two of our previous reports (Mayer et al., 2009; Tamas et al., 2007). We accessed the most up-to-date information on 614 patients (aged 7- to 14-years) with MDD (44% girls, mean age: 11.66 years), who were recruited from various mental health settings across Hungary for a study of the genetics and correlates of pediatric depression, and underwent a rigorous, standardized procedure to verify the presence of DSM-IV major depression (for more detail, see Tamas et al., 2007). We then compared

these patients to 1,228 age- and sex-matched controls (using a 1:2 ratio), who were recruited from several urban schools in Hungary (see Mayer et al., 2009 on a report for a smaller sample). All youngsters completed a questionnaire we designed, which queried about the typical use of a variety of adaptive (30 items) and maladaptive (24 items) mood repair responses (see Tamas et al., 2007); parents completed a parallel questionnaire about their children.

According to both self- and parental-ratings, depressed youths were found to have about twice as large *maladaptive* mood repair response repertoires than controls (*p*s<.0001; Cohen's *d*=1.54 for parental-rating and 1.06 for self-rating), which is reflected in the rates of the various responses. For example, depressed youngsters reported being significantly more likely than controls (*p*<.0001 for each contrast) to respond to sadness by ruminating (58% vs. 25%, respectively), becoming passive, laying down, and just feeling bad (60% versus 37%, respectively), throwing or kicking things (60% versus 32%, respectively), yelling and screaming at family (48% versus 22%, respectively), and just eating and eating (41% versus 19%, respectively).

However, surprisingly, according to their parents, the adaptive mood repair response repertoires of depressed youngsters were only marginally smaller than that of controls (*p*<.05; Cohen's *d*= 0.12), while, by their own self-report, depressed youngsters had larger adaptive response repertories than control peers (*p*<.001; Cohen's *d*=0.38). For example, significantly more of the depressed than control youngsters reported responding to being sad by seeking physical comforting (56% versus 48%, respectively, *p*<.002) or to distract themselves by thinking about projects and things to do (60% versus 49%, respectively, *p*<.001). According to post-hoc analyses, the higher endorsement rates (compared to controls) were most characteristic of younger depressed patients. While these findings have to be replicated, they are consistent with a laboratory-based study of adolescents with MDD histories (aged 11- to 18-years; n=210), most of whom (n=180) were remitted from depression, and never depressed controls (n=161): After exposure to a sad film clip, all groups were similarly successful in mood repair regardless of whether they were instructed to engage in attention refocusing or to recall happy personal memories (Kovacs, Yaroslavsky et al., 2013). Results of these two studies suggest that: a) differences in mood repair outcomes across depressed and control youths primarily mirror the presence and impact of maladaptive (rather than the absence of adaptive) mood repair strategies and b) although depressed youngsters appear to be able to deploy adaptive mood repair responses, these responses do not seem to bring them the needed emotional relief in everyday life.

Mood repair mechanisms—Do differences in mood repair outcomes across depression-prone and control youths reflect problems in underlying mechanisms? We were not able to locate behavioral studies of attention processes in depressed youngsters during mood repair. However, the information processing literature suggests that depressed youths deploy their attention less flexibly than unaffected peers. Compared to typically developing youths, depressed youngsters manifest biased attention toward negatively valenced stimuli and are slower to switch their attention from such stimuli. In a series of studies, Ladouceur and colleagues (2006) found that depressed children and adolescents (n=16) responded to sad faces faster than did controls (n=18). Such biased attention to sad faces in currently

depressed and remitted youths (n=29) was likewise reported by Hankin, Gibb, Abela and Flory (2010). Compared to controls, depressed adolescents also were slower to respond when shifting attention from negatively to positively valenced words (Maalouf et al., 2012), were slower to respond to neutral stimuli in the presence of negatively valenced background images (Ladouceur et al., 2005), and were more accurate in responding to sad target words (Kyte, Goodyer, & Sahakian, 2005). However, some studies reported that depressed and control youths did not differ in attention disengagement or attention shifting when faced with sad words as experimental stimuli (Dagleish et al., 2003; Kyte et al., 2005; Neshat-Doost, Moradi, Taghavi, Yule, & Dalgleish, 2000).

Do the mood repair problems of depressed youths reflect inability to access their hedonic reserve? While we were unable to locate direct tests of this question, studies of reward processing (which has been linked to hedonic capacity) (e.g., Kringelback, 2005) have yielded relevant findings. The indications are that depressed youths do not pursue potential rewards to the extent that normal peers do. On gambling tasks used to index hedonic capacity, depressed adolescents (n=19) displayed low reward seeking; they bet far more conservatively than controls even under conditions of very high (i.e., 80–90%) reward probability, and their low reward seeking behavior was significantly associated with self-rated anhedonia (Rawal, Collishaw, Thapar, & Rice, 2013). In another study, when depressed and remitted 11-year-olds were faced with a high probability of winning (i.e., 66%), they failed to adjust their betting relative to control peers, regardless of the magnitude of the rewards (Forbes, Shaw, Dahl, 2007). Impaired sensitivity to reward magnitudes at age 11, in turn, predicted depression levels and internalizing disorders a year later (Forbes et al., 2007). Not surprisingly, these findings are indicative of reduced hedonic capacity while youngsters are in a depressive episode, which may persist even during periods of remission.

High-risk children and adolescents

Affect experience—Given their family histories, one could expect that youngsters at high risk for depression (but not yet depressed) would evidence *clinically significant* dysphoric symptoms at slightly higher rates than healthy controls, but at notably lower rates than diagnosed depressed peers. Because we were unable to locate any publications with the needed information, we again turned to our studies. One study has involved the follow-up of young offspring of USA parents, who had been previously ascertained as having had juvenile-onset mood disorder, and control parents, who had been ascertained as having had no lifetime major psychiatric disorder (e.g., Gentzler, Rottenberg, Kovacs, George & Morey, 2012; Lopez-Duran, Kuhlman, George & Kovacs, 2013). For the present article, we examined the most up-to-date data from this ongoing study on: a) n=77 high-risk never-depressed offspring (49% girls; ages 8- to 19-years) and n=109 control offspring (45% girls; ages 8- to 19-years). Based on standardized clinical interviews, the point prevalence of clinically significant mood symptoms was quite low among high-risk youths, but consistently exceeded the rates among controls. Specifically, the rates for the high-risk and control groups, respectively, were as follows: 4% versus 0% for sad/depressed mood (p=.07), 14% versus 4% for irritability (p<.009), and 1% versus 0% for anhedonia (n.s.).

One phase of the previously described study of young, depressed, Hungarian probands included their biological siblings (a group at high risk for familial depression owing to their depressed brothers or sisters) and normal control peers (Kovacs, Bylsma et al., 2013); the controls, recruited from neighborhood schools, were prescreened for mental health via clinical interviews. For the present article, we compared n=205 never depressed siblings (54% girls, aged 11- to 19-years) of probands with MDD histories and n=180 never depressed control peers (36% girls, aged 11- to 19-years). Based on standardized interviews, the point prevalence of clinically significant sadness in the sibling high-risk and control low-risk groups was 2% versus 0.6% (N.S.), anhedonia was present in 0.5% versus 0% (n.s.), and irritability was found in 1% versus 1%, respectively (n.s.). Thus, in independent studies, high-risk youths (defined either by parental or sibling depression history) displayed low rates of mood symptoms, which tended to slightly exceed the rates among control peers, but were substantially lower than the rates reported for age mates with depressive illness (reviewed in the beginning of this article).

Mood repair—Observational studies have found that high-risk and low-risk offspring differ in aspects of mood repair very early in life. In a study by Manian and Bornstein (2009) of 5-month-old offspring, mothers with (n=48) and without (n=68) histories of depression were taught to display a facial expression that invariably elicits distress in infants (both groups of mothers performed equally well). High-risk infants were not as successful as were control infants in using gaze aversion to reduce their distress when confronted with the unexpected maternal expression, but compensated by using the more elementary and "self-directed" strategy of self-soothing. In a study by Maughan, Cicchetti, Toth and Rogosch (2007) of how 4-year-olds respond to simulated anger between adults, 30% more of the high-risk (n=93) than the control (n=58) children ($p<.001$) had dysregulated emotion profiles.

Based on parental ratings, Gentzler, Santucci, Kovacs and Fox (2009) found that 6- to 13-year-old high-risk offspring (n=39) had fewer adaptive mood repair responses ($p<.01$) and a greater number of maladaptive responses ($p<.05$) than offspring of control parents (n=26). Using a partially overlapping offspring sample, Silk, Shaw, Skuban, Oland, and Kovacs (2006) induced distress by having the children wait for a visible but physically un-reachable prize: They found that high-risk children (n= 49), especially girls, were significantly more likely than control children (n=37) to continue looking at the source of distress and to display behavioral passivity; both of those responses have been associated with the maintenance or exacerbation of dysphoria. However, in one dissenting study, when asked to recall positive personal memories for mood repair, 9- to 14-year-old high-risk girls (n=20) improved their mood just as much as low-risk girls did (Joormann et al., 2012).

Mood repair mechanisms—There is some evidence of impaired attention mechanisms in high- risk youngsters during mood repair. In their study, Manian and Bornstein (2009) observed that high-risk, 5-month-old infants did not have the "appropriate attentional regulatory capacities" to attenuate distress. As also noted previously, 4- to 7-year old high-risk children, particularly girls, displayed more difficulties in re-focusing attention under experimentally induced distress than did low-risk peers (Silk et al., 2006). In a related study,

these high-risk offspring manifested subtle deficits in selective attention (compared to low-risk control peers) when confronted with an emotionally distressing task (Perez-Edgar, Fox, Cohn, & Kovacs, 2006).

However, information processing studies have yielded equivocal findings regarding putative attention anomalies in high-risk youths. On the one hand, two studies found that after negative mood induction, 5- to 7-year-old (n=15) and 9- to 14-year old high-risk girls (n= 21) responded faster than did control peers (n=20–27) to sad faces (Kujawa et al., 2011; Joormann, Talbot, & Gotlib, 2007). Further, whereas low-risk girls were more engaged with happy faces (n=20), high-risk girls were more likely to selectively attend to sad faces (Joormann, Talbot et al., 2007). On the other hand, Gibb and colleagues (2009) found no group differences in a study of 8- to 12-year-old boys and girls. Further, while Kujawa et al. (2011) found negative attention bias to sad faces only among high-risk girls but not boys, Lopez-Duran et al. (2013) found such attention bias only among high-risk boys but not girls.

In contrast, the findings consistently point to reduced hedonic capacity in high-risk but not-yet-depressed youths. In a longitudinal study of 1- to 9-year-old high-risk offspring (n=117) and low-risk peers (n=95), the groups displayed similar developmental trajectories of negative affect across the years, but high-risk offspring had *consistently lower* levels of positive affect than controls (Olino, Lopez-Duran et al., 2011). This finding complements results from cross-sectional studies, which showed lower levels of positive affect among high-risk offspring than among controls in experimental situations, including in samples of: 1- to 8-year-olds (Shaw et al., 2006), 3- to 4-year-olds (Durbin, Klein, Hayden, Buckley, & Moerk, 2005), 9- to 14-year-olds (McMakin, Burkhouse et al., 2011), and 8- to 17-year-olds (Dietz et al., 2008). Importantly, (compared to affect in low-risk offspring), attenuated positive affect persisted across different laboratory contexts according to high-risk 9- to 14-year-olds and their mothers (McMakin, Burkhouse et al., 2011). We have also found that, across several hedonic stimuli (e.g., watching a humorous film clip, receiving a desired prize), self-rated positive affect was consistently attenuated (relative to affect in control peers) among 11- to 19-year-old high-risk, never depressed siblings of young depressed probands (Kovacs, Bylsma et al., 2013).

Summary—The affective landscape of pediatric depression is defined by high rates of clinically significant sadness, anhedonia, and irritability, along with daily fluctuations of affect experience as a function of children's social context and activity level. Impaired mood repair is likely to be one important factor that accounts for the ongoing dysphoria in pediatric depression. The mood repair problems of depressed youngsters reflect the fact that most of their responses to dysphoria exacerbate (rather than diminish) that affect. The difficulties of depressed youngsters in attenuating sadness also may be secondary to some impairment in attention and hedonic processes that underpin many adaptive mood repair responses.

Affect experience in youngsters at high familial risk for depression appears to be similar to that of typical youths, as evidenced by low rates of mood symptoms. However, it is of considerable concern that atypical mood repair can be detected among high-risk offspring as early as infancy. A further cause for concern is that attention and hedonic processes, which

fuel a variety of mood repair responses, appear to be compromised in young high-risk youths. Because a large portion of youngsters at familial risk for depression eventually will develop depressive disorders, any impairment related to emotion experience could be a viable target for early intervention.

The next issue we address is how dysphoric experience, its regulation, and the mood repair mechanisms of interest are represented on a neural level, and if there are differences in those representations across depressed, depression-prone, and control youths.

Dysphoria and mood repair: their representation in the brain

Drevets, Price, and Furey (2008) have provided an overview of the neural structures and circuits implicated in mood disorders, including depression, while Dalgleish, Dunn, and Mobbs (2009) have evaluated recent work on brain regions that appear to be crucial in the representation, processing, and experience of affect, in general. In brief, the experience and regulation of emotions are subserved by interactive neocortical and subcortical brain regions. The prefrontal cortical (PFC) regions of interest have included the dorsolateral areas (dlPFC), the orbitofrontal cortex (OFC), the medial PFC, and the anterior cingulate cortex (ACC), each of which is believed to subserve a multitude of related functions that concern cognition, attention, decision making, and behavioral control. The subcortical regions of interest have included the limbic network (e.g., amygdala, hippocampus) and the striatum (e.g., nucleus accumbens, caudate nucleus, and putamen), which have been implicated in the recognition, experience, and expression of emotions (e.g., Drevets et al., 2008).

There is agreement that the dorsolateral regions of the PFC subserve executive functions and decision making (Dalgleish et al., 2009). During the process of mood repair, the dlPFC is believed to exert an inhibitory effect on the activities of the orbito- and medial-frontal cortices and the anterior cingulate cortex (Grabenhorst & Rolls, 2011), which together comprise the ventromedial prefrontal cortex (vmPFC). While serving a number of diverse functions, the vmPFC is involved in evaluating various parameters of reward and punishment (Grabenhorst & Rolls, 2011; Kringlebach, 2005) and in integrating input from visceral, attention, and emotion systems (Dalgleish et al., 2009; Grabenhorst & Rolls, 2011; Holroyd & Coles, 2008; Phillips, Drevets, Rauch, & Lane, 2003). The vmPFC and dlPFC connect with subcortical limbic regions, including the amygdala and hippocampus, and the ventral striatum. Amygdala activity has been associated with the perception of a variety of emotional stimuli and with hippocampal-related memory consolidation and learning (Phelps & LeDoux, 2005), while the ventral striatum has been of interest in connection with reward-related and hedonic experiences (Haber & Knutson, 2010; Knutson & Greer, 2008).

The interface between cortical and subcortical brain structures during the experience and regulation of emotions has been framed in various ways. As reflected in overviews of neuroimaging studies (e.g., Der-Avakian & Markou, 2012; Forbes & Dahl, 2012; Hulvershorn, Cullen, & Anand, 2011; Perlman & Pelphrey, 2011), emotion regulation has been typically conceptualized as the brain's PFC regions exerting neural control over limbic-amygdalar regions. Some researchers have referred to "top-down" versus "bottom-up" control processes, usually meaning the influence of prefrontal on subcortical structures,

and vice versa (e.g., Corbetta & Shulman, 2002; Dalgleish et al., 2009; Ochsner & Gross, 2007; Thompson, 2011). Using a related perspective, Phillips et al. (2003) proposed that emotion regulation is subserved by a dorsal cortical system (dlPFC, ACC, and hippocampus) that supports executive functions, while emotion perception and processing is subserved by a ventral system (OFC, limbic, and striatum).

Depressed children and adolescents

Affect experience—Neuroimaging studies typically assess subjects at rest (believed to reflect trait affectivity) and during exposure to emotionally salient stimuli (reactivity) such as photos of sad faces or depressing pictorial scenes. One study found that while viewing pictures of sad faces, depressed children (n=11, mean age=4.5 years) displayed increased right amygdala activity as compared to healthy controls (Gaffery et al., 2011). In another study that involved watching a sad film clip, depressed children (n=24, mean age = 9.8 years; 87% in remission) had reduced left dorsolateral PFC activity relative to control peers and increased amygdala activity as a function of depression severity (Pagliaccio et al., 2012). In two overlapping depressed samples (who were compared to controls), resting neuroimaging revealed reduced functional connections of the dorsal PFC to the right ventral ACC (n=17, mean age=9.6 years) (Gaffery et al., 2010) and the amygdala (n=13, mean age = 8.9 years) (Luking et al., 2011). Similarly, compared to controls, depressed adolescents at rest (n=12, aged 15- to 19-years) displayed diminished prefrontal control of the subgenual ACC, which, on a structural level, showed reduced white matter connections between dorsal and ventral regions (Cullen et al., 2009; 2010). Thus, neural representation of dysphoric experience in depressed youngsters differs from that in controls: it is associated with lower levels of dorsal/prefrontal activity, higher activity in some subcortical areas, and reduced connectivity to ACC regions.

Mood repair—In one study, depressed and healthy adolescents (n=28, aged 13- to 18-years) were asked either to reduce (via cognitive reappraisal) or maintain their emotional reactions to negatively valenced images (Perlman et al., 2012). Compared to controls, depressed youngsters showed increased right amygdala activity while they engaged in cognitive reappraisal for mood repair and reduced association in activity between medial PFC and amygdalar regions while they maintained emotional arousal (Perlman et al., 2012). This response pattern to emotional challenge (possibly reflecting the inability to down regulate emotional arousal) has been associated with increased depressive symptoms and functional impairment (Perlman et al., 2012; Yang et al., 2009). In a parallel vein, resting-state hyperactivity in ventral brain regions correlated with self-rated difficulties in regulating dysphoric emotions among children remitted from depression (Gaffery et al., 2010; Luking et al., 2011).

Mood repair mechanisms—The likelihood that successful attention refocusing in mood repair is subserved by increased dorsal PFC activity and reduced activity in ventral brain regions is suggested by a study of healthy *adults* that examined focusing attention through mindfulness (Dickenson, Berkman, Arch, & Lieberman, 2013) and a study of the brain correlates of mindfulness during exposure to negative images (Taylor et al., 2011). While in pediatric depression, the neural substrates of attention during mood repair are yet to be

explored, one study examined the brain correlates of traditional attention tasks among 14- to 17-year-olds: The results revealed that depressed youths (n=21) had lower levels of activation in dlPFC and ACC regions than age- and sex-matched controls (n=21) as they were completing a series of attention and behavioral inhibition tasks (Halari et al., 2009).

The neural substrates of hedonic processes during mood repair have not yet been examined in depressed children and adolescents, but studies of reward related processes have yielded relevant findings. Compared to healthy peers, depressed youngsters failed to show activation in ventromedial PFC and striatal regions in the face of monetary rewards (n=20, aged 13- to 18-years) (Chantiluke et al., 2012). In a series of studies with overlapping samples, Forbes and colleagues (2006) likewise found reduced striatal activity in depressed youths (n=14, aged 9- to 17-years) while they anticipated winning and then won a gambling task, while they (n=15, aged 8- to 17-years) participated in a card guessing game for monetary reward (Forbes et al., 2009), and while they (n=10, aged 8- to 16-years) anticipated winning subsequent game trials (Olino, McMakin et al., 2011). Lending external validity to some of these findings, hypo-activation in striatal regions predicted low positive affect outside the laboratory (Forbes et al., 2009) and reduced treatment response in an 8-week clinical trial with depressed adolescents (n=13, ages 10- to 16-years) (Forbes et al., 2010). Thus, the indications are that, in depressed youngsters, potentially rewarding stimuli elicit atypical hypoactivation of striatal regions.

High-risk children and adolescents

Affect experience—Research also indicates that offspring at familial risk for depression (but not yet depressed) tend to display atypical neural activity in regions implicated in emotion processing. Unlike control peers (n=14), high-risk 7- to 11-year-olds (n=11) showed an attenuated relationship between ventral (amygdalar) and dorsal system activity during rest (Luking et al., 2011). Hypoactivation in the left dorsolateral PFC also was reported in high-risk, 16- to 21-year-olds (n=29) during exposure to fearful faces (Mannie, Taylor, Harmer, Cowen, & Norbury, 2011). Further, compared to controls, high-risk children tended (p = .06) to display reduced co-activation of amygdalar and hippocampal regions (Luking et al., 2011). Reduced co-activation in these youths correlated with self-reported difficulties in managing sadness (Luking et al., 2011). And in a study of 15- to 17-year-olds, which used EEG to index brain activation, never depressed high-risk girls (n=37) showed greater emotional reactivity to depressogenic stimuli than did control peers (n=44) (Foti, Kotov, Klein & Hajcak, 2011).

Mood repair—Research on the neural correlates of mood repair in youths is still in its early stages. In a pioneering study, never depressed, high-risk girls (n= 20, aged 9- to 14-years) and controls received negative mood induction via film clips and then were instructed to recall positive autobiographical memories as the mood repair strategy (Joormann et al., 2012). Compared to their baseline patterns of neural activation, high-risk girls showed greater amygdalar activity and no change in dorsolateral PFC activity during mood repair, while controls displayed greater PFC activity and no significant change in amygdalar activity. Thus, in high-risk girls, the mood repair response of interest failed to elicit the expected, presumably normative and adaptive dorso-ventral neural activation pattern.

Mood repair mechanisms—We were unable to locate neuroimaging studies of attention re-focusing in high-risk subjects while engaging in mood repair. However, there is growing evidence of blunted neural activation of hedonic circuitry in response to pleasurable experimental stimuli in this population. Specifically, compared to their low-risk counterparts, high-risk offspring (n=13, ages 10- to 14-years old) displayed reduced striatal activity while engaging in pleasant tasks such as playing games that could result in prizes (Gotlib et al., 2010) and while viewing happy faces (n=17; aged 10- to 18-years) (Monk et al., 2008). Likewise, in the study of 15- to 17-year-olds, which used EEG to index neural activation, never depressed, high risk girls (n=37) showed blunted responses to monetary rewards compared to n=44 low-risk controls (Foti et al., 2011). Thus, hedonic capacity appears to be compromised in young, high-risk offspring.

Summary—There is accumulating evidence that neural activity in depressed, remitted, and high-risk youngsters differs from that in control cases during dysphoric experience and its regulation. While few studies have reported on all the brain regions of interest from the same scans, the overall findings on depressed and depression-prone youths suggest that dysphoria is associated with reduced connectivity across fronto-limbic structures, less than expected regulation of various ventral brain regions by prefrontal cortices, and atypical functioning of neural regions that subserve hedonic-related experiences. While there is impressive evidence of hedonic impairment, little is known about the neural correlates of attention-refocusing during mood repair in depressed and high-risk youngsters.

Despite their value, the implications of functional neuroimaging studies of dysphoria and its regulation are not straightforward. Results are inconsistent regarding which specific sub-regions of the PFC and limbic system account for core disparities between depressed and healthy children, and whether such differences are bilateral (e.g., Luking et al., 2011) or unilateral (e.g., Beesdo et al., 2009; Perlman et al., 2012). There are inconsistencies as to whether a region of interest (e.g., amygdala) shows hyperactivation (e.g., Perlman et al., 2012) or hypoactivation (e.g., Beesdo et al., 2009) when depressed adolescents and controls are compared. In some studies, neural findings do not correlate with depressive symptom severity (e.g., Luking et al., 2011), while in other studies, symptoms correlate with brain regional activity that does not differentiate affected and un-affected youngsters (e.g., Pagliaccio et al., 2012). Finally, the apparent lack of specificity of various functional neuroimaging results to depression constrains the practical implications of this body of research.

Discussion

As various reviews and meta-analyses indicate, research on pediatric depression has continued to flourish along well-established paths, including prevention (Horowitz & Garber, 2006; Stice, Shaw, Bohon, Marti, & Rohde, 2009; Vitiello, 2011), pharmacological and psychological treatment (Tsapakis, 2008; Usala, Clavenna, Zuddas & Bonati, 2008; Watanabe, Hunot, Omori, Churchill, & Furukawa, 2007; Weisz, McCarty & Valeri, 2006), and psychosocial risk and potentiating factors such as stress generation (Hammen, 2009; Liu & Alloy, 2010).

Multi-disciplinary investigations of the experience and regulation of dysphoric affect in clinical depression represent a comparatively new research area, reflecting an upsurge of interest in the role of emotions in healthy and atypical human functioning. The resultant studies are re-confirming some clinical observations about depressed youngsters, provide new insights about the experience of dysphoria and its regulation and their neural representation in the brain, and reveal that atypical aspects of mood repair can be detected at a very young age in not-yet-depressed children at familial risk for depressive disorders. Some of these findings have useful implications for clinicians, but their practical value can be appreciated only in the context of children's lives: We provide some context by addressing recent findings on the impact of maternal depression on children's functioning.

Information on the point prevalence of mood symptoms in psychiatrically diagnosed children and young adolescents suggests that clinically significant sadness and irritability occur at similarly high rates (up to about 80% of cases), while anhedonia is not as frequent (less than 60% of cases). However, the rates appear to rise by the late teens. The comparatively lower rate of anhedonia in younger ages lends some support for the broader DSM mood criteria for a depressive disorder in childhood (sadness, anhedonia, irritability) as compared to adulthood (sadness, anhedonia).

An important and underappreciated finding from EMA studies of mood has been that, in daily life, dysphoria in depressed youths is labile: it varies as a function of social context (which has been replicated) and type of activity (which needs replication). In fact, diurnal changes in affect, in tandem with changes in context, seem to be the rule both in depressed and typically developing youngsters. The lessening of depressed mood in depressed youngsters in the presence of others, compared to when being alone (Merrick, 1992; Silk et al., 2011), and as a function of doing something versus doing nothing (Silk et al., 2011) is notable. Importantly, similar diurnal variations of dysphoric mood have been reported in depressed adults (Thompson et al., 2012). These findings suggest that the DSM symptom criterion that requires dysphoric mood "most of the day" and "nearly every day" for a depressive episode is not a valid reflection of mood experience in clinical depression. All in all, clinicians should query young depressed patients (and their parents) about the patients' affect and mood in various social contexts and in connection with different levels of activity. The resultant information may help the clinician to suggest ways to re-structure a young patient's daily schedule in order to bring some degree of emotional relief. Moreover, given that variability in dysphoric mood across contexts appears to be the norm, lack of daily variability in a depressed child's affect should give rise to concern because it may signal a particularly severe depression.

Studies of mood repair have yielded converging evidence that depressed youngsters often respond to their own distress in ways that unwittingly maintain or exacerbate that affect. Depressed youngsters also have more extensive repertoires of such maladaptive regulatory responses than do emotionally healthy peers, which would be expected. However, one surprising finding has been that, both by parental- and self-report, depressed children and adolescents do *not* appear to have a deficiency of adaptive mood repair responses. On the contrary, depressed youngsters in our study (especially younger ones) report larger repertoires of adaptive ways to attenuate dysphoria than controls, which is counter-intuitive

and needs to be replicated. However, impaired attentional and hedonic capacity, well documented in high-risk children, could render adaptive regulatory responses less effective and may partly explain why youngsters who have many “adaptive” ways to attenuate sadness still develop clinical depression.

Possibly therefore, the overall mood repair difficulties of depressed and depression-prone youngsters can be accounted for (to an unknown extent) by an excess of maladaptive mood repair responses *and* adaptive responses that fail to bring the expected emotional relief in daily life. The latter proposition receives some support from a study in which depressed adults and healthy controls were similarly able to cognitively down-regulate distress, but this effect was not sustained in the patients (Erk et al., 2010). It also has been reported that adaptive mood repair responses can have adverse physiological correlates in vulnerable samples. For example, when depressed adults engaged in neutral re-appraisal of a distressing stimulus (an adaptive cognitive mood repair response), this led to an *increase* (rather than the expected decrease) in neural activity in subcortical circuits associated with emotional arousal (Goldsmith, Pollak & Davidson, 2008; Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007). The existence of such contrary effects in depressed youths is yet to be explored. Further work also is needed to uncover how presumably helpful (adaptive) mood repair responses fail to protect depression-prone youths from clinical depression.

The research findings on mood repair in depressed youngsters suggest that responses to sadness are viable targets for intervention. Although mood repair responses typically are automatic (but not necessarily unconscious, see Davidson, Jackson & Kalin, 2000; Goldsmith et al., 2008; Mauss et al., 2007), older children are able to report on them, as are parents. Therefore, at the minimum, clinicians should try to identify how a given young patient responds to dysphoria and distress and assure that the child’s caregivers do not reinforce maladaptive strategies. It may also be useful to identify key person(s) that can best help the young patient to reduce dysphoria (see Marroquín, 2011 for a review of interpersonal sources of mood regulation) and enlist their assistance with the goal of replacing a child’s maladaptive responses by adaptive ones.

For example, a child who becomes passive when dysphoric (which is likely to maintain the distress) may be encouraged to undertake a prescribed physical activity as a response, which should improve the child’s mood. Or, for example, a youngster who tends to ruminate when distressed may be taught to focus instead on a specific behavioral task or sensory process. Admittedly, reduced attentional or hedonic capacity may constrain the level of mood repair that can be achieved with some youngsters. However, one laboratory study reported similar levels of emotional relief after instructed mood repair in normal control adolescents and adolescents with histories of major depression (Kovacs, Yaroslavsky et al., 2013); another study likewise found comparable mood repair benefits in controls and high-risk but never depressed adolescents after they deployed a given regulatory response (Joormann et al., 2012). These findings suggest that very specific mood repair instructions in structured settings may be needed as the first step to remediate mood repair problems in depression-prone youths.

Based on the literature, we proposed that attention refocusing and hedonic capacity are two mechanisms that enable many forms of mood repair. Indeed, recently introduced novel interventions for depression emphasize the importance of these processes: One "brain training" program has been designed to increase executive (prefrontal cortical) functioning (Siegle, Ghinassi & Thase, 2007), while another program seeks to stimulate and maintain positive affect (e.g., McMakin, Siegle, & Shirk, 2011). However, because these programs rely almost exclusively on abstract cognitive operations and decision making skills, which are still developing in childhood and adolescence, they may not be appropriate to younger depressed patients. Mood improvement may be achieved more easily in younger ages by focusing on, and teaching specific mood repair responses. For example, as a substitute for "top-down" attention processes, a parent and depressed child may be taught a concrete "bottom-up" mood repair strategy that utilizes some somatic-sensory process (e.g., getting a hug, listen/dance to music) to attenuate distress. (For suggestions, see Kovacs & Lopez-Duran, 2012). For depression-prone children with low hedonic reserve, attempts to increase hedonic experiences may be less successful than what Meehl (1975) called "hedonic bookkeeping." According to Meehl (1975), low hedonic capacity accounts for one form of depression: to help individuals with this trait, activities and hobbies should be planned in ways that maintain hedonic reserve.

However, the above noted intervention suggestions have to be considered in the context of maternal cooperation, because it is typically mothers who bring their children for mental health care. Of particular relevance to the present article, findings show that from 14% to 30% of mothers who seek treatment for their depressed offspring are themselves clinically depressed, a further 59% have sub-threshold symptoms (Ferro, Verdeli, Pierre, & Weissman, 2000; Hammen, Rudolph, Weisz, Rao, & Burge, 1999), and close to 70% have a lifetime history of depressive disorder (Ferro et al., 2000; Hammen et al., 1999). Even sub-syndromal parental depression has been linked to depression symptoms in offspring (Campbell, Morgan-Lopez, Cox, & McLoyd, 2009; Cortes, Fleming, Catalano, & Brown, 2006).

While maternal depression has long been known to have wide-ranging negative repercussions for young offspring (for reviews, see Goodman, 2007; Goodman & Gotlib, 1999; Hammen, 2009), interest in its effects on children's mood repair is relatively recent. Goodman and Gotlib (1999) proposed that depression during pregnancy directly affects offspring's emotion regulation by disrupting neuro-regulatory mechanisms that subserve it. The effects of maternal depression also can persist well beyond the post-partum period because mothers play a key role in the development of their children's emotion regulation (e.g., Kopp, 1989; Morris et al., 2007): they typically initiate and assist children's mood repair during the first few years of life, model mood repair responses through their own behavior, and continuously shape children's mood repair competence by supporting and reinforcing some responses, while discouraging some other responses. Importantly, mothers with depression histories may have unwittingly modeled maladaptive mood repair responses for their offspring or may have failed to sufficiently reinforce offspring's deployment of adaptive responses. The impact of such learning histories may pose a particular challenge to the clinician seeking to remediate a depressed child's mood repair deficits. Further, some of our recommendations may not be feasible in the treatment of a given depressed child if

mother has an ongoing depression, which undermines her ability to assist her offspring's attempts at improved mood repair.

Depression in the mother also has other adverse implications because it has been associated with treatment non-completion of depressed offspring (Kovacs et al., 2006) and a low rate of treatment response (Kennard et al., 2008). In one study of depressed youths, the effectiveness of cognitive behavioral therapy "plummeted in the face of maternal depressive symptoms" (Brent et al., 1998, p. 192). However, offspring clearly benefit when depressed parents receive appropriate interventions (see a review by Gunlicks & Weissman, 2008; see also Garber, Ciesla, McCauley, Diamond, & Schloredt, 2011; Pilowsky et al., 2008; Swartz et al., 2008; Weissman, Pilowsky et al., 2006). Thus, a *depression screen focusing on the mother* should be a standard part of the evaluation of a *depressed child*. If there is evidence of maternal depression, the clinician should do whatever is possible to assure the availability of services.

Functional brain neuroimaging studies of dysphoric experience and its regulation in youngsters have confirmed that prefrontal cortices are associated with self-regulatory processes. The overall evidence suggests that depressed and high-risk youngsters usually (but not consistently) differ from typical peers in the activity of prefrontal, limbic, and striatal regions while at rest, and in response to emotion triggers. Reduced connectivity and atypical activation patterns between fronto-limbic and fronto-striatal regions in depression-prone youngsters may well mirror the neural underpinnings of overt mood repair problems and sub-optimal attention and hedonic/reward related mechanisms. Overall, therefore, neuroimaging findings parallel in many respects the results of clinical and behavioral studies, and underscore that depressed and depression-prone youngsters differ from healthy age mates in various aspects of dysphoric experience, its regulation, and some underlying mechanisms. However, the significance of the neuroimaging literature on pediatric depression must be tempered by limitations inherent in the very small sample sizes of most imaging studies and equivocal findings regarding the specific neural sub-regions that account for across-group differences in emotion processing and regulation. Also, activity in the brain regions of interest has not consistently correlated with clinical symptoms. Thus, while affective neuroscience research has inspired the development of experimental interventions (McMakin, Siegle et al., 2011; Siegle et al., 2007), the implications for everyday clinical practice are still rather limited.

We end by highlighting what we believe to be the most striking aspect of the literature that we reviewed, namely, the convergence of behavioral and functional neuroimaging findings on mood repair and related mechanisms in *young offspring at familial risk for depression.* With few exceptions, the studies uniformly point to some impairment in attentional and hedonic processes that enable a variety of mood repair responses, and thus put constraints on high-risk children's mood repair attempts. The impairments have been reported at various levels of functioning, in independent pediatric samples, by different investigators, and typically *prior* to manifest depression in the offspring. Admittedly, impairment in the putative mood repair mechanisms across the early years of development and adolescence in these high-risk groups has not yet been linked to later depressive illness. However, follow-up has shown that by young adulthood, about two-thirds of high risk offspring will develop

major depression (Weissman, Wickramaratne et al., 2006). Thus, impaired mood regulatory mechanisms in such high-risk populations can be regarded as candidate risk factors for eventual clinical depression and should be considered in efforts to prevent depression.

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Key Points

- Pediatric depression is characterized by high rates of dysphoric emotions (sadness, anhedonia, distress) but these experiences are labile and vary in daily life as a function of social context and degree of activity. Clinicians may therefore wish to include in their diagnostic assessment of young depressed patients questions about diurnal changes in affect in various contexts and levels of activity. Lack of affect variability may indicate severe depression.
- Depressed youths, and those at high familial risk for depression, have difficulties in attenuating dysphoria (mood repair). They differ most dramatically from typically developing peers by having far more maladaptive mood repair responses (responses that exacerbate dysphoria). Clinicians may therefore wish to identify how young patients respond to upswings in dysphoria and the affective sequelae of those responses.
- There is increasing evidence that high-risk, but not yet depressed youths display subtle and early signs of impairment in attention processes and hedonic capacity that underpin many adaptive mood repair responses. These processes may therefore be potential targets in depression prevention trials.
- Functional neuroimaging studies of depressed and high-risk youngsters yielded some evidence that brain regional activation and connectivity patterns during dysphoric experience and mood repair, and during tasks probing attention and hedonic processes, can generally differentiate depression-prone youths from control peers.
- Depression screen focusing on mothers should be a part of the evaluation of depressed children. Treating maternal depression will benefit the child's response to his or her own therapy and enable the mother to be a more effective agent in facilitating the child's mood repair attempts.