Emotion (Dys)regulation and Links to Depressive Disorders

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ABSTRACT—***Clinical depression is a significant mental health problem that is associated with personal suffering and impaired functioning. These effects underscore the continuing need for new approaches that can inform researchers and clinicians when designing interventions. The present article proposes that individual differences in the self-regulation of sadness and distress provide an important link between stress, depressed mood, and the onset of depressive disorder, and that a better understanding of the ways children successfully manage dysphoric emotions will lead to improved prevention and treatment of pediatric depression. After examining the normative development of responses that children use to attenuate sadness, aspects of the neurobiological infrastructure that both enable and constrain such self-regulatory efforts, and affect regulation of children at familial risk for depressive disorders, this article concludes by calling for integrated, developmental, multidisciplinary studies of sadness self-regulation.***

KEYWORDS—*emotion regulation; depression; mood repair; development*

Clinical depression (such as major depressive disorder) in children and adolescents is a significant mental health problem and is a gateway to chronic affective difficulties and recurrent depressive episodes (e.g., Birmaher et al., 1996). Because highly effective treatments for pediatric depression have been elusive (Kennard et al., 2006; Weisz, McCarty, & Valeri, 2006), there is an urgent need for further conceptual and empirical approaches that can inform intervention efforts for this condition.

Researchers have typically regarded depression in youngsters as resulting from an interaction of personal vulnerabilities, such as negative cognitive biases (including the tendency to view the world through black glasses), social skill deficits or familial predisposition (diatheses), and adverse life events or circumstances (stressors; e.g., Cicchetti & Toth, 1998; Hammen, Brennan, & Shih, 2004; Joormann, Eugene, & Gotlib, in press). We propose that a key diathesis is an impaired ability to attenuate sad, dysphoric affect (which, in young children, typically manifests as distress) in a context-appropriate fashion, that this attribute should characterize vulnerable children before the onset of clinical depression, and hence that individual differences in the ability to regulate sadness represent a clinically meaningful link between stress and the emergence of depressed mood and depressive disorders. Focusing on affect regulation provides a ready bridge to intervention research (Kovacs, 2007). For example, if vulnerable children with poor affect regulation are identified early, it may be possible to prevent depressed mood and forestall depressive episodes. Also, characterizing typical and atypical developmental features of self-regulatory responses to sadness could help in designing more age-sensitive treatment strategies for depressed children.

THE SCOPE OF THIS ARTICLE

What do we know about the development and characteristics of individual differences in the ability to attenuate sadness, how these differences arise, and how they alter the risk of depression? To answer these questions, we summarize findings on typical responses to experimentally induced distress during the early years of life and consider how developmental trajectories can go awry; we also address the emerging literature on affect regulation among children at familial risk for

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depression. Then, we consider neurobiological factors, such as brain maturational processes, that enable normative regulatory responding, as well as other physiological systems that can facilitate or impede adaptive regulation of sadness. We conclude by noting that studies of sadness self-regulation that integrate psychosocial and neurobiological perspectives are best positioned to illuminate those pathways to pediatric depression that are preceded by dysregulated affect. We also highlight the need for studies of development over time and underscore the idea that studying children's self-regulatory responses can inform interventions for pediatric depression.

Being able to attenuate sadness is one facet of emotion regulation (ER). Although ER is a complex and global construct that has been defined in various ways (e.g., Cole, Martin, & Dennis, 2004; Eisenberg & Zhou, 2000), in this article we focus specifically on responses through which a child can change the intensity and duration of sadness (Cole et al., 2004; Thompson, 1994) and the neurobiological infrastructure that enables, but also developmentally constrains, such self-regulatory efforts. We focus on sad affect because it is a core feature of a depressive disorder (American Psychiatric Association, 1994); we focus on the ability to attenuate sadness because the *in*ability to do so is a key problem for clinically depressed people. In studies of adults, the process of attenuating sadness has been called "mood repair" (e.g., Joormann, Siemer, & Gotlib, 2007), meaning that a sensible way to cope with sadness or distress is to respond in ways that have a reparative effect and help one to feel better (Isen, 1985).

THE DEVELOPMENT OF SELF-REGULATORY RESPONSES TO SADNESS AND DISTRESS

One key developmental task of childhood is to learn to attenuate (adaptively self-regulate) the emotion of sadness or distress so that it does not impair functioning (e.g., Cole et al., 2004; Eisenberg & Morris, 2002). We know that the responses that enable children to modulate distress start to emerge fairly early in life within a complex matrix of psychosocial variables, which interact with children's innate neurobiology (e.g., Goldsmith & Davidson, 2004; Morris, Silk, Steinberg, Myers, & Robinson, 2007), and that through various learning mechanisms, interactions between the child and family members shape the acquisition of such regulatory responses (e.g., Morris et al., 2007). Studies across the first few years of life (e.g., Cole et al., 2004; Fox, 1994) have also shown that development involves transitioning from caregiver-initiated ER in early infancy to self-initiated ER, that responses become increasingly sophisticated and effective, and that children's response repertoires expand to include mostly strategies that can attenuate distress, but also some that can exacerbate it (Kovacs, 2007). There also is a developmental sequencing of response types: The earliest to appear (around the age of 3 months) are "body-oriented" or *somatic-sensory* responses and include self-stroking, rocking, and hand clasping (e.g., Mangelsdorf, Shapiro, & Marzolf, 1995); the earliest form of a *cognitive response* category, namely gaze aversion, is favored at around 6 months (e.g., Mangelsdorf et al., 1995); by about 12 months of age, infants recruit caregivers as regulatory agents (e.g., Diener, Mangelsdorf, McHale, & Frosch, 2002), signifying *interpersonal ER responses*; and finally, *behavioral ER responses* (e.g., instrumental behaviors such as leaving the scene or solitary play) emerge between 12 and 24 months of age (e.g., Braungart & Stifter, 1991; Diener et al., 2002).

The postinfancy trajectories of these various ways of self-regulating sadness have not yet been fully described, and research has targeted mostly cognitive ER processes. For example, refocusing attention away from the source or experience of distress (the use of "executive attention"), which is a key component of ER (Posner & Rothbart, 2000), is clearly developmentally mediated: Gaze aversion in infants (e.g., Kopp, 2002) gives way to responsiveness to distracters between 3 and 6 months of age (e.g., Harman & Rothbart, 1997), which evolves into the use of attention refocusing to manage distress by 24 months of age (e.g., Grolnick, Bridges, & Connell, 1996). Although full control of attention emerges around 3 years of age, it is not entirely developed until adolescence (Posner & Rothbart, 2000). Cognitive ER responses that require abstract thinking (such as reinterpreting the meaning of one's emotion) are infrequent among children (Kovacs, 2007) and become typical only after midadolescence (Kuhn, 2000).

It is reasonable to expect that atypical development of self-regulatory responses to sadness should manifest in ways different from the patterns we noted above (Sroufe, 1997). For example, children with atypical ER development may fail to transition from caregiver-initiated to self-initiated regulatory responding, they may develop fewer "adaptive" responses than is optimal, or some of the expected transitions from rudimentary to more sophisticated responses may not take place. Any one of these scenarios would impair the child's ability to attenuate sad affect, especially under conditions of ongoing or recurrent distress-provoking conditions.

Although we lack information on how atypical sadness-related ER responses develop over time, studies of the young offspring of depressed mothers (see Goodman & Gotlib, 1999; Goodman, 2007) provide support for our proposal of impaired distress regulation being a diathesis in at-risk samples. For example, there is substantial evidence that such at-risk infants have more difficulty containing their distress, fuss more, smile less, and interact less than do infants of nondepressed mothers (e.g., Field et al., 2007; Weinberg, Olson, Beeghly, & Tronick, 2006). At the same time, compared with nondepressed mothers, depressed mothers provide their infants with less verbal interaction, less quality stimulation, less affectionate contact (Fleming, Ruble, Flett, & Shaul, 1988; Righetti-Veltema, Bousquet, & Manzano, 2003), and slower and less contingent responses—for example, the mother is far less likely to return

the child's smile or to return it after some delay (Field, Healy, Goldstein, & Guthertz, 1990).

Are such infants emotionally dysregulated because they lack the developmentally appropriate responses, because they model their mothers' displays of emotion or mood, because their mothers have failed to initiate the ER process, and/or because their physiology does not favor such responses (as we discuss later)? It is possible that these infants lack the appropriate ER responses because they have been deprived of the needed learning opportunities. If such situations are recurrent or chronic, these offsprings' ER repertoires should be dominated by rudimentary somatic-sensory responses, along with early signs of atypical development in areas that are especially dependent on caregiver input, such as the strategic use of attention. The extent of atypical ER response development should depend on various factors including the timing of maternal depression (e.g., Maughan, Cicchetti, Toth, & Rogosch, 2007).

Indeed, impaired distress self-regulation and early problems in ER-related attention deployment appear to characterize preschool-age children of mothers with a history of depression. For example, both 3-year-old (Hoffman, Crnic, & Baker, 2006) and 4-year-old (Maughan et al., 2007) offspring of depressed mothers have been found to have more problems regulating their emotions than did control peers, although the depressed mothers were also less effective in helping their children's ER or problem-solving efforts. When they were subjected to a situation that should elicit some negative emotion, preschool-age, at-risk offspring have been found to have more difficulties than did their low-risk peers disengaging attention from the source of their distress and were less likely to use active self-distraction (Silk, Shaw, Skuban, Oland, & Kovacs, 2006). A related study of young at-risk children also reported problems in attention control but only when the children were affectively challenged (Perez-Edgar, Fox, Cohn, & Kovacs, 2006). Findings on older at-risk girls confirm that parental depression confers to offspring some impairment in attention control when faced with emotional stimuli (Joormann, Talbot, & Gotlib, 2007). However, longitudinal studies will be needed to link such putative ER deficits under conditions of stress to the development of clinical depression.

NEUROBIOLOGICAL ASPECTS OF SELF-REGULATORY RESPONDING TO SADNESS AND DISTRESS

How does a neurobiological perspective help us to understand the development of regulatory responses to sadness and the emergence of individual differences? At the very least, such a perspective highlights that the maturation of the brain and of various biological systems constrains the ER responses available to children at different ages. For example, brain regions mature in a sequence from "back to front," starting with areas associated with basic motor and sensory systems, followed by temporal and parietal association regions that underlie basic language skills and spatial attention, and last by the prefrontal cortex (PFC) and lateral temporal cortices that support higher order cognitive processes (Casey, Tottenham, Liston, & Durston, 2005). Maturational processes that continue into young adulthood include transitions from diffuse to focal recruitment of brain regions to accomplish specific tasks (Durston et al., 2006) and fine-tuning of neural connections that facilitate more efficient neural functioning (Casey et al., 2005).

Although studies of brain maturation have not empirically linked specific neural areas with ER skills, the sequence of maturational changes is consistent with the developmental trajectory of observable ER responses we have described. In early infancy, the brain is most ready to support rudimentary somatic-sensory ER responses (e.g., distress relief through gentle touch). Interpersonal ER responses that entail language and more complex social interaction (such as obtaining emotional support and help from a parent) come with the advent of toddlerhood, and cognitive ER responses that depend on higher order skills supported by the PFC (such as the ability to filter irrelevant information or to inhibit a dominant response) come to the forefront with adolescence (e.g., Rothbart & Bates, 2006). And the process of fine-tuning the neural connections in the brain may underlie observations that children's ability to attenuate sadness improves with age.

Studies of the neurobiology of sadness self-regulation (or mood repair) have focused almost exclusively on cognitive processes and mostly in adults; work in this area in children is just beginning. For example, functional neuroimaging of adults has shown that successful implementation of cognitive ER strategies, such as reappraisal (Ochsner, Bunge, Gross, & Gabrieli, 2002), is associated with increased activation in various prefrontal brain regions (e.g., dorsolateral PFC; Lévesque et al., 2003) and reduced activation in subcortical limbic structures (such as the amygdala) that enable affect and mood (e.g., Ochsner & Gross, 2005). Notably, when 8- to 10-year-old normal girls were asked to suppress experimentally induced sadness (a cognitive ER response), they activated the same prefrontal brain regions as did adults with the same task, although the pattern of activation in the children included other, more diffuse regions as well (Lévesque et al., 2004). Such results confirm that the efficient use of cognitive processes for ER is closely linked to the maturation of the brain.

Are these brain regions that are critical in responding to and regulating sadness compromised in youngsters at risk for depression? Preliminary evidence suggests that the answer is "yes." For example, children of mothers with mood disorders exhibit greater activation in some limbic structures when presented with fearful faces, but lesser activation in response to happy faces than do control peers (Monk et al., 2008). Atypical brain activity, usually hypoactivation in some frontal sites (assessed by scalp electroencephalography), has been

reported among offspring of depressed mothers, including infants (e.g., Diego et al., 2004), preschoolers (e.g., Dawson et al., 2003), and adolescents (Tomarken, Dichter, Garber, & Simien, 2004). And a recent study found that when at-risk youngsters performed a task under emotional stress, a subtle deficit in selective attention led them to recruit more anterior brain processing resources than did control peers (Perez-Edgar et al., 2006). Thus, it appears that compared to their low-risk peers, young children at familial risk for depression are more reactive neurally to negative emotional triggers (which could strain their ER resources) and that stress may elicit ER-related attention deficits. Such features, in turn, may adversely affect these children's further ER development.

Researchers also have examined the functioning of two other systems—the parasympathetic nervous system (PNS) and the hypothalamic–pituitary–adrenal (HPA) system—that may be particularly critical to ER across the first few years of life because they mirror physiological flexibility in responding to environmental demands. One index of PNS functioning is cardiac vagal tone (Porges, 1996), which reflects the ability to alter heart rate in response to external demands, allocate attention, and thus regulate distress and arousal. Notable maturation of vagal tone takes place between 7 weeks and 2 years of age (Bornstein & Suess, 2000) and, by early childhood, individual differences in vagal tone are associated with differences in stress reactivity, regulation/soothability, and attention (Calkins, 1997). Preschoolers with different behavior problem profiles display distinct patterns of vagal regulation in response to laboratory challenges (Calkins, Graziano, & Keane, 2007). Furthermore, in a sample of young children at risk for depression, low vagal tone (a potential indicator of poor affect regulation) was associated with internalizing problems (Forbes, Fox, Cohn, Galles, & Kovacs, 2006), and among such offspring, there was also an association between less resilient vagal responses (evidenced in low vagal recovery) and dysfunctional ways of self-regulating distress (Santucci et al., 2008).

Researchers have also examined physiological flexibility by studying the HPA system that orchestrates the human stress response. HPA functioning is typically indexed by the production of cortisol (Stansbury & Gunnar, 1994) and is believed to mirror individual differences in the ability to self-regulate and cope (Gunnar & Quevedo, 2007). Cortisol levels across 24-hr cycles and in response to stress undergo maturational changes (e.g., Gunnar, 1992) and increase at puberty (for a review, see Gunnar & Vazquez, 2006), although these changes have not yet been linked to ER. However, some HPA dysfunction is evident in pediatric depression risk. For example, elevated levels of basal cortisol have been found to predict the onset and recurrence of depression, along with suicidality (e.g., Mathew et al., 2003), as well as increased depressive symptoms (Luby et al., 2003). Compared to low-risk peers, children of depressed mothers appear to have higher and more variable morning cortisol levels (Halligan, Herbert, Goodyer, & Murray, 2004) and greater HPA reactivity to laboratory stressors (Ashman, Dawson, Panagiotides, Yamada, & Wilkinson, 2002). And finally, in one study, cortisol levels among preschoolers predicted higher levels of symptoms of psychological problems later in life (Essex, Klein, Cho, & Kalin, 2002).

CONCLUSIONS AND FUTURE DIRECTIONS

The central role of protracted despondent, unhappy mood in clinical depression (American Psychiatric Association, 1994) underscores the idea that an impaired ability to regulate dysphoric affect is a crucial feature of this condition. We proposed that individual differences in the ability to attenuate sadness and distress in context-appropriate ways represent a developmentally meaningful link among stress, depressed mood, and depressive disorders, and that impairment in self-regulatory skills predates the onset of a depressive disorder. For example, clinical experience suggests that, for a variety of reasons, some children are not able to regulate negative affect that ordinary stressful events or atypical circumstances can trigger. If the provocations persist and the child's intrinsic regulatory responses fail to bring emotional relief, what may have started out as a temporary downcast affect may become protracted depressed mood. Downcast mood often is one of the first signs of an impending depressive episode; such mood typically elicits negative social feedback and can also directly trigger a cascade of other depressive symptoms including loss of interest and motivation, tiredness, and sleep problems, all of which then contribute to a downward spiral of worsening symptoms.

Teaching at-risk children how to adaptively modulate sadness could be a practical way to prevent protracted despondent moods and thereby reduce the likelihood of depressive episodes. But to design comprehensive remediation programs, we need more information about the unfolding of ER responses (especially after the first few years of life) and their interface with brain maturation and other neurobiological and psychological systems, and about how a child's individual learning history and transactions within his or her social context affect ER response development. It would be particularly useful to have data about which types of ER responses are most effective at distress relief at various ages.

As summarized above, we know that very young offspring at familial risk for depression already display patterns of dysregulated affect and that the ability to refocus attention to repair mood may be compromised in posttoddler age, at-risk youngsters. We also know that when they are affectively challenged, at-risk youngsters tend to be neurally more reactive and physiologically less flexible than are their comparison peers, which clearly should influence the success of their ER efforts. But we know little about how various aspects of ER response development other than attention may have been impaired in these children or if competence in one (e.g., interpersonal)

regulatory domain may have offset deficiency in other (e.g., cognitive) regulatory responses; we also know little about the extent to which a vulnerable biological infrastructure could historically have constrained the development and effectiveness of ER responses. Furthermore, although there are indications that repertories for sadness self-regulation continue to develop as children grow, there is scant information about these trajectories beyond very early childhood, especially in children at risk for depression.

Are some children unable to attenuate distress because their ER strategies are not sufficiently effective or because they unwittingly use strategies that exacerbate their negative mood (e.g., Nolen-Hoeksema, 1991)? Do individual differences in neurophysiologic reactivity or flexibility predispose children to intense distress, which then renders their self-regulatory responses ineffective? To what extent do individual differences in the development of the PFC explain the effectiveness of cognitive mood-repair strategies such as reappraisal? And under which conditions does impaired self-regulation of sadness increase the risk of depressive disorder? Answers that have practical implications for intervention are most likely to be generated by studies that integrate psychological, social, and neurophysiologic aspects of mood repair.

Indeed, investigating links among different functional systems is essential if we want to understand why experiencing sadness and distress leads to sustained negative affect and clinical depression in some children but not in others. For example, as we noted, there are indications (from unrelated studies) that elevated basal cortisol levels and impaired attention under conditions of emotional challenge characterize young offspring at risk for depression. For children who have both of these attributes, the link to depression may be the effect of stress hormones on the brain; specifically, increased cortisol secretion secondary to stress can have especially detrimental effects on those parts of the brain that are involved in emotions (e.g., Bremner et al., 2003) and their regulation.

Thus, when a young child is exposed to a chronic stressor-like maternal depression, the associated high cortisol levels may compromise the subsequent development of certain cognitive regulatory responses to distress. It is important to note, however that (a) sensitive caregiver behavior (which encompasses appropriate responsivity to the child's ER needs and may be provided by alternate caregivers) can buffer neuroendocrine responses to stress in infants (for a recent review, see Gunnar & Quevedo, 2007), and (b) even if the unfolding of cognitive ER responses is compromised, other (i.e., interpersonal, behavioral and somatic sensory) ways of regulating sadness remain available to the child. Hence, it may be possible to intervene in various ways to facilitate the development of better ER responses throughout children's lives.

To conclude, we note that there is a disconnect between studies of the development of ER and studies of ER responses among older children, adolescents, and young adults. Studies of the latter age groups have focused almost exclusively on the use of cognition to regulate mood, although regulatory repertories include a much broader array of responses. Attention to interpersonal, behavioral, and somatic-sensory means of self-regulating sadness and their neurobiological underpinnings should broaden our knowledge of individual differences that contribute to the resolution (or continuation) of episodes of sadness. Investigating what type of ER responses predict the onset of a depressive episode in high-risk youth could yield particularly valuable information for prevention and treatment. Better characterization of the developmental unfolding of diverse, adaptive ways to self-regulate sadness, and the reinforcement contingencies that maintain maladaptive ER responses, may help in the design of novel age-appropriate intervention strategies for pediatric depression.

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