

## Emotion Regulation in the Brain: Conceptual Issues and Directions for Developmental Research

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Emotion regulation cannot be temporally distinguished from emotion in the brain, but activation patterns in prefrontal cortex appear to mediate cognitive control during emotion episodes. Frontal event-related potentials (ERPs) can tap cognitive control hypothetically mediated by the anterior cingulate cortex, and developmentalists have used these to differentiate age, individual, and emotion-valence factors. Extending this approach, the present article outlines a research strategy for studying emotion regulation in children by combining emotion induction with a go/no-go task known to produce frontal ERPs. Preliminary results indicate that medial-frontal ERP amplitudes diminish with age but become more sensitive to anxiety, and internalizing children show higher amplitudes than noninternalizing children, especially when anxious. These results may reflect age and individual differences in the effortful regulation of negative emotion.

Psychologists have often looked to the nervous system to resolve ambiguities and uncertainties in their understanding of thought and behavior. But in the last 10 to 15 years, major advances in neuroscience have encouraged an unprecedented number of psychologists to explore brain function as an adjunct to their studies. With the rapid rise of imaging techniques, coupled with a huge body of anatomical data from animal and lesion research, and theoretical insights from cognitive science and connectionism, neuroscientists can now offer detailed models of the complex mental processes of interest to psychologists. Developmental psychologists, in particular, have been increasingly rewarded by their explorations of neural processes and the fresh perspectives these provide.

It seems that the question asked by Cole, Martin, and Dennis (this issue), about the scientific validity and precise meaning of emotion regulation, is particularly ripe for a neuroscientific perspective. It is a question that has been debated for a long time among emotion researchers and emotional developmentalists, without any real consensus. Cole et al. took the important step of identifying emotion regulation as an independent construct, one that refers to a process distinct from emotional activation, and

they reviewed evidence for its central role in child development. But if they are right, and if emotion regulation can be conceptualized and measured independently of emotion itself, this distinction should be apparent in the brain as well. The purpose of this article is to investigate this possibility, by (a) identifying problems intrinsic to a neural definition of emotion regulation, (b) showing how neuroscientists have tried to circumvent these problems by isolating the role of the prefrontal cortex in emotion processing, (c) describing a methodology that may be useful for assessing the regulatory activity of the prefrontal cortex, and (d) describing new directions for using this methodology to study emotion regulation in children and adolescents.

### Validity of Emotion Regulation in the Brain

Cole et al. (this issue) identified emotion regulation as an independent construct by differentiating it from emotional activation. They defined emotion regulation as “changes associated with activated emotions” independent of the emotions themselves (p. 317–333). These changes are conceptualized as a second stage of activity, following the initial stage of emotional activation. This seems a necessary distinction because emotion is defined by these authors as “a fluid and complex progression” (p. 317–333).

Yet, from a neural perspective, it is not so easy to determine when one stage ends and the other begins. This is not only because the active brain is in constant flux but because regulation is intrinsic to all neural activity related to emotion or motivation.

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How do neuroscientists identify emotion in the first place? Many emphasize the mediation of emotion by subcortical regions, but there is also some agreement that emotional processes involve synchronization across multiple neural subsystems, including the cortex. When people report emotional experiences, imaging studies demonstrate changes in blood flow across a large number of sites, ranging from the brain stem to the cortex (Damasio et al., 2000). In contrast, a relatively small number of sites are activated during conscious cognitive activity, and these are specifically cortical (Toga & Mazziotta, 2000; cf. Panksepp, in press). To make sense of these findings, some neuroscientists define emotion as the reciprocal recruitment of subsystems up and down the neural hierarchy, accompanied by endocrine and muscular changes, in a process of rapid self-organization (Freeman, 1999; Panksepp, 2000; Schore, 2000; Tucker, Derryberry, & Luu, 2000). These subsystems include the brain stem, mediating arousal and behavioral activation; the limbic system, mediating coarse perception, memory, learning, and affective feeling; and the cerebral cortex, subserving higher order perceptual processes, attention, working memory, and voluntary control. Subsystem synchronization is thought to produce a coherent neural gestalt—a unified brain—whose psychological features include a specific action readiness, a restricted attentional focus, a stable cognitive appraisal, and a distinct emotional feeling (Lewis, 2000; Scherer, 2000).

The problem with defining emotion regulation as an independent process is that the synchronization of systems underlying emotion includes regulation of each system by the others. The brain stem and hypothalamus regulate the cortex by fueling it with a particular batch of neurotransmitters and neuropeptides, the limbic system coordinates brain stem activity while entraining the cortex to key sensory events, and the prefrontal cortex regulates all lower systems by delaying stereotypical responses and modulating arousal in the service of conscious evaluation and deliberate action (Tucker et al., 2000). These regulatory processes do not occur in discrete temporal stages. Rather, they coevolve rapidly as attention, perception, emotion, and motor output become spontaneously coordinated (Freeman, 1999; Schore, 2000). From a neural perspective, then, regulatory processes are intrinsic to the cascade of neural changes underlying emotion. This perspective integrates both of Cole et al.'s (this issue) categories—emotion as regulating and emotion as regulated—into one unified construct: emotion regulation as embedded in emotion.

### Prefrontal Cortex and Cognitive Control

If emotion regulation is embedded in emotion activation, must the concept of emotion regulation be abandoned as scientifically untenable? In fact, neuroscientists interested in emotion are as hooked on the idea of regulation as their behavioral counterparts. Their solution to the definitional problem of embeddedness is to hive off one aspect of emotion regulation—cognitive control—and study its relation to the remaining constituents of emotion. They do this by isolating the role of the prefrontal cortex, which is highly implicated in deliberate cognitive activity, and studying its patterns of activation when participants are experiencing varying degrees and types of emotions (e.g., Davidson, Putnam, & Larson, 2000; Hariri, Bookheimer, & Mazziotta, 2000; Thayer & Lane, 2000). There is rarely any attempt to fit prefrontal activity to a time window representing a later stage of emotional processing. Rather, the regulatory functions of prefrontal cortex are investigated during emotion elicitation by measuring the magnitude, location, and persistence of prefrontal activity, relative to other brain regions, as differentiated by eliciting events, task demands, types of emotion, and personality or clinical typologies.

Imaging studies using fMRI and PET techniques reveal prefrontal activation patterns that are reciprocally related to amygdala activity, suggesting a direct inhibitory effect on emotional responsiveness by prefrontally mediated cognitive processes (Davidson et al., 2000; Hariri et al., 2000). Fox and Davidson (e.g., 1987) have repeatedly shown characteristic asymmetries in prefrontal activation, beginning in infancy, that may tap temperamental differences in the cognitive regulation of anxiety. Within the prefrontal cortex itself, the more dorsal regions become more activated when participants are cognitively engaged and less activated when participants report experiences of anxiety or depression (Drevets & Raichle, 1998). Anxious and depressed participants characteristically show less dorsal activation and more activation of the ventral prefrontal cortex, an area richly connected to the limbic system. In addition, lateral and medial prefrontal sites become more activated when participants reappraise their negative emotions, whereas ventral prefrontal and amygdala sites become deactivated reciprocally (Ochsner, Bunge, Gross, & Gabrieli, 2002). These and other studies suggest that various regions of the prefrontal cortex subserve cognitive controls that can directly minimize the scope, intensity, or duration of negative emotions.

The anterior cingulate cortex (ACC) is particularly relevant to the cognitive control of emotion. The ACC is a region of more primitive prefrontal cortex, located along the medial surfaces of the two hemispheres, with strong links down to the limbic system and forward to other prefrontal regions. Because it is poised between motivational subcortical systems and prefrontal systems for planning and control, the ACC is credited with the executive functions of selective attention and response selection in challenging situations requiring deliberate monitoring. The ACC has also been singled out by child psychologists interested in self-regulation. Posner and Rothbart (1998, 2000) identified a rapid rise in what they termed *effortful control* at about age 4 years, which they attributed to the development of children's capacity to use the ACC to modulate impulsive behavior through deliberate selection of strategies. Based on the results of attentional tasks, these authors have proposed variations in ACC function corresponding with developmental and temperamental differences in directed attention. Thus, the regulatory function of the ACC may be an important window into the cognitive control of emotion in child development.

#### **Event-Related Potential (ERP) Methods for Studying Cognitive Control of Impulse and Action**

The role of the prefrontal cortex in cognitive control of impulse and action—if not emotion per se—has perhaps been most extensively studied through EEG techniques, particularly the analysis of ERPs. ERPs tap characteristic task-related changes in the electrical activity of a region of cortex by averaging across EEG segments that are synchronized to a repeated stimulus or response. Two ERPs have been associated with cognitive control in challenging circumstances: the inhibitory N2 and the error-related negativity (ERN). Both are negative deflections recorded above the frontal midline region and thought to be generated by activity in the dorsal ACC (Gehring & Willoughby, 2002; van Veen & Carter, 2002). The inhibitory N2 is recorded 200 to 350 ms following a challenging stimulus, and the ERN is recorded about 80 ms following an erroneous response (though its generation begins well before the response, when the brain first “perceives” its own mistake). Classically, the inhibitory N2 was thought to tap the successful inhibition of a prepotent response. Thus, in go/no-go tasks, where participants press a button on go cues but withhold the button-press on no-go cues, the N2 is reliably evoked on correct no-go trials (Eimer, 1993; Jodo & Kayama, 1992). Classically, the ERN was thought to tap the

recognition of a mismatch error in responding, as when one becomes aware that one has pressed the wrong button in a choice task (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991). However, both ERPs are now thought to tap more global aspects of deliberate cognitive control, exemplified by effortful evaluation, conflict monitoring, selection among competing responses, action monitoring, feedback monitoring, and adjusting to violated expectancies (e.g., Luu, Flaisch, & Tucker, 2000; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; van Veen & Carter, 2002). Because self-monitoring and action-regulation are central to this family of operations, Luu and Tucker (2002) have characterized both ERPs as tapping self-regulation, a description very close to Posner and Rothbart's (1998, 2000) construct, *effortful control*.

Could the operations indexed by the N2 and ERN, and dubbed self-regulation by Luu and colleagues, tap emotion regulation per se? Two lines of research suggest that they could. First, Tucker, Luu, and colleagues have shown that motivational engagement is necessary to produce these ERPs and that negative emotion is particularly relevant. Tucker, Hartry-Speiser, McDougal, Luu, and deGrandpre (1999) found greater mediofrontal activity at about 350 ms when participants received critical feedback on their performance, an effect replicated by Luu, Tucker, Derryberry, Reed, and Poulsen (2003) independent of the participant's actual recent performance. Tucker et al. (in press) found a similar N2-like response differentiating between good trait and bad trait descriptive words (e.g., “generous” vs. “mean”), potentially tapping the regulation of induced negative emotion. Second, individual differences in the ERN have been related to dysfunctions in emotion regulation. Undersocialized individuals who cannot control their aggressive impulses show lower amplitude ERNs (Dikman & Allen, 2000). Conversely, obsessive-compulsive individuals show higher amplitude ERNs than normal individuals, with the effects localized to the ACC (Gehring, Himle, & Nisenson, 2000; Hajcak & Simons, 2002). Higher amplitude ERNs have also been associated with lower scores on impulsivity (Pailing, Segalowitz, Dywan, & Davies, 2002), and ERNs diminish in magnitude when participants are given anxiety-reducing drugs (Johannes, Wieringa, Nager, Dengler, & Münte, 2001). Finally, Luu, Collins, and Tucker (2000) found that participants with higher scores on trait negative affect produced higher amplitude ERNs, suggesting more effortful or more consistent cognitive regulation of emotion. These studies indicate a fine line between impulse control and emotion regulation.

In sum, the inhibitory N2 and ERN appear to tap links between motivational processes and cognitive regulation that are mediated by the ACC. However, the studies reviewed here have not measured emotion regulation directly. To apply ERP methods to the study of emotion regulation, it would be necessary to devise experiments in which negative emotion is specifically induced, changes in frontal ERPs are measured, and these changes are related to participants' capacities or efforts to cognitively control their emotions. In the following section we outline a research strategy for accomplishing this goal with children and adolescents, and we describe preliminary work using this approach with children from 6 to 16 years of age.

### Approach to Developmental ERP Research on Emotion Regulation

Before describing our own approach, we briefly mention some relevant work in child ERP studies. Nelson and colleagues have conducted numerous ERP studies on children's recognition of emotional expressions. Some of these bear on issues of emotional perception and perhaps emotion regulation. Most relevant here, 4- to 6-year-old children showed an N2-like deflection at central sites in response to angry rather than happy faces, interpreted as an attentional response to affective information (Nelson & Nugent, 1990). Children also showed higher amplitude ERPs to angry than happy faces as compared with adults (Kestenbaum & Nelson, 1992). In terms of individual differences, children who showed greater amplitude responses to familiar faces at frontal and central electrodes were more emotionally attuned and had lower levels of cortisol (Gunnar & Nelson, 1994). These studies indicate that ERP research with children is capable of tapping cognitive processes that vary systematically with type of emotional stimulus and emotional disposition.

A second relevant line of research concerns the elicitation of the ERN and N2 in children. Davies, Segalowitz, and Gavin (2003, submitted) examined changes in the form and amplitude of the ERN in participants aged 7 to 20 years. They found that the amplitude and apparently the consistency of the ERN increased with age, with the increase most evident at 17 to 20 years. This trend may reflect a developing capacity for the cognitive control of impulsive action (cf. Henderson, 2003), but this interpretation remains speculative until task factors are better understood. Regarding individual differences, a marked reduction of the inhibitory N2 has been observed in children with attention deficit hyper-

activity disorder (ADHD), attributed to their inability to initiate or maintain response inhibition (Pliszka, Liotti, & Woldorff, 2000; Yong-Liang et al., 2000). These studies suggest that prefrontal ERPs can be sensitive to developmental factors as well as individual differences in children's cognitive control.

Our approach to the neurocognitive study of emotion regulation in children relies on an emotion-induction procedure inserted into a classical paradigm for eliciting cognitive control. The paradigm we have chosen is the go/no-go task, in which no-go (or lure) trials are relatively infrequent (approximately 30% of all trials). Children are asked to push a button each time a letter appears on the screen, as quickly as possible. However, when a letter appears twice in a row, they are required to withhold their response (Garavan, Ross, & Stein, 1999). This kind of task is particularly useful for looking at cognitive control. When participants successfully withhold a prepotent response on a go/no-go task, their control is indexed by an inhibitory N2. When they fail to withhold their response, the error gives rise to an ERN. In the first case, cognitive control may be directed at response selection. In the second case, it may be directed at response evaluation. Or, as noted earlier, both activities can be construed in terms of self-regulation and effortful control. In the task we have developed, the error rate is adjusted dynamically online by increasing the speed of stimulus presentation to increase errors or reducing it to decrease errors, producing an error rate that stays close to 50% (Garavan et al., 1999; S. J. Segalowitz, personal communication, June 2003). We can thus end up with an equivalent number of N2s and ERNs.

In our task, children are rewarded with points, shown periodically onscreen, for successful performance, and they are told that a high number of points is needed to win a "big prize" at the end of the experiment. After the EEG net is placed on their heads, the task proceeds through three blocks: A, B, and C. These represent an A-B-A design, with the A and C blocks being identical, and the B block intended to elicit negative emotion. In the A block, the children see their points climb steadily, suggesting they will win the prize. In the B block, because of a change in the point-adjustment algorithm, their points plummet to zero. Post-task interviews indicate that participants experienced anxiety and disappointment during this block. In the C block, they see their points advance once again, and they end up winning the prize, but we think that the anxiety induced in the B block remains present during the C block, an assumption we are now testing. Our expectation has been that the elicitation

of negative emotion in the B block will introduce an (additional?) element of emotion regulation into the cognitive control processes normally tapped by the go/no-go paradigm. If we are right, children who apply more effortful cognitive controls to regulate their emotions, whether for developmental or individual-difference reasons, will show higher amplitude or more consistent N2s or ERNs, or both, in the B and C blocks than in the A block, that is, following negative emotion induction.

So far, we have tested 53 children with this procedure, ranging in age from 6 to 16 years. Our preliminary analyses have revealed a clear inhibitory N2 and ERN waveform on correct and incorrect no-go trials, respectively. N2 amplitudes decreased significantly with age and were significantly differentiated by block (with B and C block amplitudes approximately double A block amplitudes, for 13- to 16-year-olds only). Our working hypothesis is that younger children expend more effort controlling their responses in general, whereas older children recruit effortful self-control more under anxious conditions. Children who scored in the top quartile on a measure of internalizing showed greater mean amplitudes for both ERPs, with the increase in ERN amplitude significant only in Block B. Internalizers may have exerted more effortful control in anticipation of responding on all blocks, whereas their regulation efforts following errors increased with the anxiety induction. These findings, though preliminary, suggest that frontal ERPs can pick up developmental and individual differences in cognitive processes dedicated to the regulation of negative emotion.

In conclusion, emotion regulation may not correspond to a particular stage of emotion activation, either in brain or in behavior. Rather, regulatory processes may be embedded in the interacting constituents of emotions as they self-organize. Yet, the cognitive control of emotional outcomes can be isolated and studied online, as demonstrated in research on cortical activities. Using electrophysiological and other methods, one can examine prefrontal processes that underlie the cognitive regulation of emotion as they vary with the type of emotion experienced, individual differences in emotional and cognitive characteristics, and developmental changes in regulatory processes. We encourage developmental researchers to explore this new terrain, as it can greatly enrich our perspective on emotion regulation across the lifespan.

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