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Magnitude and chronometry of neural mechanisms of emotion regulation in subtypes of aggressive children

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ABSTRACT

Emotion regulation is a key social skill and children who fail to master it are at risk for clinical disorders. Specific styles of emotion regulation have been associated with particular patterns of prefrontal activation. We investigated whether anxious aggressive children would reveal a different pattern of cortical activation than non-anxious aggressive children and normally-developing children. We examined the magnitude and timing of source activation underlying the N2—an ERP associated with inhibitory control—during a go/nogo task with a negative emotion induction component (loss of earned points). We estimated cortical activation for two regions of interest—a ventral prefrontal and a dorsomedial prefrontal region—for three 100-ms windows over the range of the N2 (200–500 ms). Anxious aggressive children showed high ventral prefrontal activation in the early window; non-anxious aggressive children showed high ventral prefrontal activation in the late window, but only for the duration of the emotion induction; and normally-developing children showed low ventral prefrontal activation throughout. There were no group differences in dorsomedial prefrontal activation. These results suggest that anxious aggressive children recruit ventral prefrontal activation quickly and indiscriminately, possibly giving rise to their rigid, threat-oriented approach to conflict. The late ventral prefrontal activation seen for non-anxious aggressive children may underlie a more delayed, situation-specific, but ineffective response to frustration.

1. Introduction

One of the most important tasks of childhood is learning to modify emotion-driven actions (e.g., hitting when angry or freezing when afraid) and the thoughts and feelings that go with them. This kind of cognitive control frequently falls under the rubric of emotion regulation. The development of emotion regulation is thought to be shaped by both biological factors and environmental transactions in infancy and childhood (see Gross & Thompson, 2007, for a review). With age, children acquire a repertoire of self-regulation strategies ranging from primitive methods, such as gaze aversion and distraction, to sophisticated methods requiring effortful regulation, such as reappraising emotional situations and inhibiting emotional impulses (for reviews see Bradley, 2000; Calkins & Hill, 2007; Eisenberg, Hofer, & Vaughan, 2007; Lewis, Todd, & Xu, 2010; Thompson, 1994). With the development of increasingly sophisticated emotion regulation strategies, children are better able to inhibit their negative feelings and impulsive behaviors in everyday social contexts.

However, not all children develop the same skill level when it comes to emotion regulation in general and response inhibition in particular. Inadequate regulatory capabilities can be seen all around us, for example, an angry outburst in a crowded train. Furthermore, the motivations underlying angry actions can vary greatly between people, requiring different emotion regulation styles even within the population of aggressive individuals. Examples of different motivations and their impact on aggressive behavior are discussed in the corpus of work focused on subtyping aggressive behavior problems (e.g., Barratt, Stanford, Felthous, & Kent, 1997; Crick & Dodge, 1996; Dodge, 1991; Hoving, Wallace, & LaForme, 1979; Moyer, 1968; Stieben et al., 2007; Vitiello & Stoff, 1997). As is evident from this body of work, there are many different ways of splitting the pie. Going back to our example, the angry outburst on the train may come from somebody retaliating for being accidentally bumped or from someone who is rigid with anxiety about being late for work again. Because of the high comorbidity found between anxiety disorders and

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disorders of conduct (Kessler, Chiu, Demler, & Walters, 2005), we use the terms *anxious aggressive* and *non-anxious aggressive* to subtype our clinical group.

Anxious aggressive children are often described as overcontrolled, cognitively inflexible, inhibited, and/or threat oriented, whereas purely aggressive children (without co-occurring anxiety) are often described as undercontrolled or impulsive (e.g., Eisenberg et al., 2005, 2007; Granic & Lamey, 2002; Lewis, Granic, & Lamm, 2006; Stieben et al., 2007). Thus, aggressive children with and without co-occurring anxiety problems may have very different emotion regulation styles. Because these regulatory differences can only be inferred on the basis of behavioral observations, we utilized a more direct measure of the cognitive resources recruited for emotion regulation. Specifically, we examined the real-time progression of frontocortical changes underlying emotion regulation in both groups of clinically-defined aggressive children as well as their age-matched normally-developing peers.

Researchers have started examining the neural mechanisms underlying emotion regulation with a variety of technologies (e.g., Busatto et al., 2000; Hajcak & Nieuwenhuis, 2006; Ochsner et al., 2004). However, electroencephalogram (EEG) recordings and the event-related potentials (ERPs) derived from them are particularly useful for studying self-regulation in children because they are relatively quick and nonintrusive (e.g., Hajcak & Dennis, 2009; Jonkman, Lansbergen, & Stauder, 2003; Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006). One ERP component, the mediofrontal N2, is observed between 200 and 500 ms post-stimulus and is generally associated with regulatory processes. These regulatory processes include inhibitory control (Dimoska, Johnstone, Barry, & Clarke, 2003; Falkenstein, Hoormann, & Hohnsbein, 1999; Jonkman et al., 2003; Overtoom et al., 1998; Schmajuk, Liotti, Busse, & Woldorff, 2006; Smith, Johnstone, & Barry, 2004) and conflict monitoring or response selection (Bartholow et al., 2005; Bekker, Kenemans, & Verbaten, 2004; Dimoska, Johnstone, & Barry, 2006; Nieuwenhuis, Yeung, & Cohen, 2004; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhob, 2003). Furthermore, a number of studies have shown greater N2 activation for nogo trials than go trials (e.g., Bekker, Kenemans, & Verbaten, 2005; Donkers & van Boxtel, 2004; Falkenstein et al., 1999; Nieuwenhuis et al., 2004), suggesting increased activation when prepotent responses need to be overcome, either through inhibition or conflict monitoring.

The N2 may also be sensitive to the processing of emotional information. In a number of studies, emotionally salient faces and words give rise to an N2-like component of greater magnitude for negative stimuli, such as fearful or angry faces, than positive or neutral stimuli (e.g., Lewis, Todd, & Honsberger, 2007; Li, Yuan, & Lin, 2008; Liddell, Williams, Rathjen, & Gordon, 2004; Tucker et al., 2003). It is not clear what functional networks contribute to this greater activation but it may be that negatively valenced stimuli generate fear or shame, and that either the experience or regulation of these emotions underlies increased activation. Several studies from our laboratory have shown emotion-related changes in N2 activation within a go/nogo task (e.g., Lamm & Lewis, 2010; Lewis et al., 2006; Stieben et al., 2007). This task consists of three blocks: the middle block is designed to induce negative emotion through the loss of valued points. According to a self-report measure administered directly after the task, participants felt increased negative emotions during the emotion induction block (which likely carried over into the final block). Results revealed greater nogo-N2 activation during and/or after the emotion induction block compared to the first block, which we interpreted as increases in emotion regulation over and above the regulatory demands of nogo trials themselves. Thus, not only does the N2 tap self-regulation, but it is also appears to reflect the regulation of emotional states or impulses.

The cortical generators most often associated with the N2 include prefrontal and cingulate regions implicated in inhibitory control. However, the N2, like other scalp activation patterns, is derived from the sum of all underlying cortical generators active at a particular moment in time. Activation in some of these regions may not be related to inhibitory control. A number of studies modeling the activation underlying the N2 (in visual tasks) have shown generators suggestive of occipital, ventral temporal, and parietal areas (e.g., Lamm & Lewis, 2010—see Fig. 6 for activation superimposed over an average MRI; Lamm, Zelazo, & Lewis, 2006; Lewis et al., 2006; Stieben et al., 2007), regions not directly related to inhibitory control. Given that activation from all these regions appear to contribute to the N2 scalp topography—either by enhancing its polarization (e.g., projecting negative current towards or near the mediofrontal scalp area) or diminishing its polarization (e.g., projecting positive current towards or near the mediofrontal scalp area)—much information can be gained by conducting a source-space analysis based on prespecified "regions of interest." This enables one to test the activation of cortical regions directly related to regulatory functions. By performing a source-space analysis, one can address specific questions of spatial localization as well as the chronometric (temporal) properties of that activation at the millisecond level.

Given the use of powerful computers and increased access to user-friendly programs, source-space analyses are becoming common practice. Recently, a few studies conducted with children and adults have modeled the generators underlying the N2 to areas suggestive of activation in the ventral prefrontal regions—including the orbitofrontal cortex (OFC), the rostral anterior cingulate cortex (ACC), and the ventromedial prefrontal cortex (vmPFC)—and/or the dorsal ACC (Bekker et al., 2005; Bokura, Yamaguchi, & Kobayashi, 2001; Lavric, Pizzagalli, & Forstmeier, 2004; Lamm & Lewis, 2010; Lamm et al., 2006; Lewis et al., 2006; Nieuwenhuis et al., 2003; Stieben et al., 2007). Ventral prefrontal activation has been linked with ongoing, non-deliberate or non-executive regulatory or evaluative processes, such as "in-the-moment" inhibitory control and the evaluation of positive or negative aspects of current or anticipated stimuli (Blasi et al., 2006; Drevets et al., 1992; Durston, Mulder, Casey, Ziermans, & van Engeland, 2006; Eshel, Nelson, Blair, Pine, & Ernst, 2007; Kaladjian et al., 2007; Ochsner et al., 2004; for reviews see Phillips, Ladouceur, & Drevets, 2008; Rolls, 2000). Increases in ventral prefrontal activation have also been associated with increased negative emotion (for example, Baumgartner, Lutz, Schmidt, & Jancke, 2006; Kawasaki et al., 2001) and more specifically emotion regulation (for example, Ochsner et al., 2004; for a review see Quirk & Beer, 2006). Dorsal ACC activation, on the other hand, has been linked to various deliberate or "executive" regulatory functions, such as attention regulation (Crottaz-Herbette & Menon, 2006), performance or conflict monitoring (Blasi et al., 2006; Santesso, Segalowitz, & Schmidt, 2005; for a review see Botvinick, Cohen, & Carter, 2004), and deliberate response inhibition (Blasi et al., 2006; Tamm, Menon, Ringel, & Reiss, 2004). Thus, the two prefrontal regions most consistently associated with the mediofrontal N2 are the ventral PFC and the dorsal ACC, thought to be linked with stimulus-bound and executive regulatory processes, respectively.

Variation in both ventral PFC and dorsal ACC activation has been observed in clinical populations, specifically those with anxiety problems and aggressive behavior problems. A number of studies have shown greater ventral prefrontal activation in anxious patients than normal controls (e.g., Busatto et al., 2000; Johanson, Smith, Risberg, Silfverskiold, & Tucker, 1992; Liotti et al., 2000; Raunch, Savage, Alpert, Fischman, & Jenike, 1997). This activation may be related to emotional arousal or ongoing (inefficient) efforts to regulate that arousal. Moreover, we recently reported decreases in ventral prefrontal activation, following treatment, correlated