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The neural correlates of emotion-based cognitive control in adults with early childhood behavioral inhibition

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ABSTRACT

The present study is the first to assess whether the neural correlates of cognitive control processes differ in adults with and without a behaviorally inhibited temperament during early childhood. Adults with and without childhood behavioral inhibition completed an emotional conflict task while undergoing functional magnetic resonance imaging scanning. While no group differences in behavior were observed, adults with childhood behavioral inhibition, relative to adults without childhood behavioral inhibition, exhibited greater dorsomedial prefrontal cortex activity during conflict detection and greater putamen activity during conflict adaptation. Lifetime psychopathology predicted behavioral, but not brain-related, differences in conflict adaptation. These data suggest that the brain regions underlying cognitive control processes are differentially influenced by childhood behavioral inhibition, and may be differently related to psychopathology.

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1. Introduction

Effective emotion regulation requires the engagement of multiple cognitive processes that modulate the expression and/or experience of negative affect. Cognitive control is one such process that facilitates the ability to filter out, and inhibit, prepotent responses to irrelevant stimuli (Botvinick et al., 2001). The capacity to exert cognitive control increases with development, and coincides with the maturation of brain regions in the prefrontal cortex that have been implicated in cognitive control (reviewed by Casey et al., 2000). Emotion regulation is particularly critical in the presence of irrelevant stimuli that signal danger or fear, which are known to interfere with normative cognitive processes (reviewed by LeDoux, 2000; Vuilleumier, 2005). Hence, delineating the neural mechanisms of cognitive control utilized in the service of emotion regulation is important, particularly when considering development and psychiatric disorders related to anxiety, where symptoms are associated with affective processing.

Etkin and co-workers have recently described procedures that can be used to map brain regions engaged by emotional stimuli in the context of a cognitive control task (Egner et al., 2008; Etkin et al., 2006, 2010; Etkin and Schatzberg, 2011). This emotional conflict task requires subjects to identify the expression of an emotional face while ignoring an emotion word superimposed upon the face (see Fig. 1A). Some trials include congruent word-face emotions, whereas others include incongruent word-face emotions. The emotional conflict task can be used to assess two aspects of cognitive control: conflict detection and inhibitory control in the form of conflict adaptation. Enhanced *emotional conflict detection* results in response time interference, or slowing, for incongruent, relative to congruent trials (Fig. 1B). This interference is inhibited when an emotionally incongruent trial follows an incongruent trial, but not when it follows a congruent trial—an effect called *emotional conflict adaptation* (Fig. 1C). This adaptation to emotional conflict reflects the activation of regulatory mechanisms, triggered by conflict on the previous trial, which then improves performance on the current incongruent trial (Egner et al., 2008; Etkin et al., 2006, 2010; Etkin and Schatzberg, 2011). Unlike healthy adults, patients with generalized anxiety disorder fail to adapt to emotional conflict (Etkin et al., 2010; Etkin and Schatzberg, 2011). This failure to adapt has

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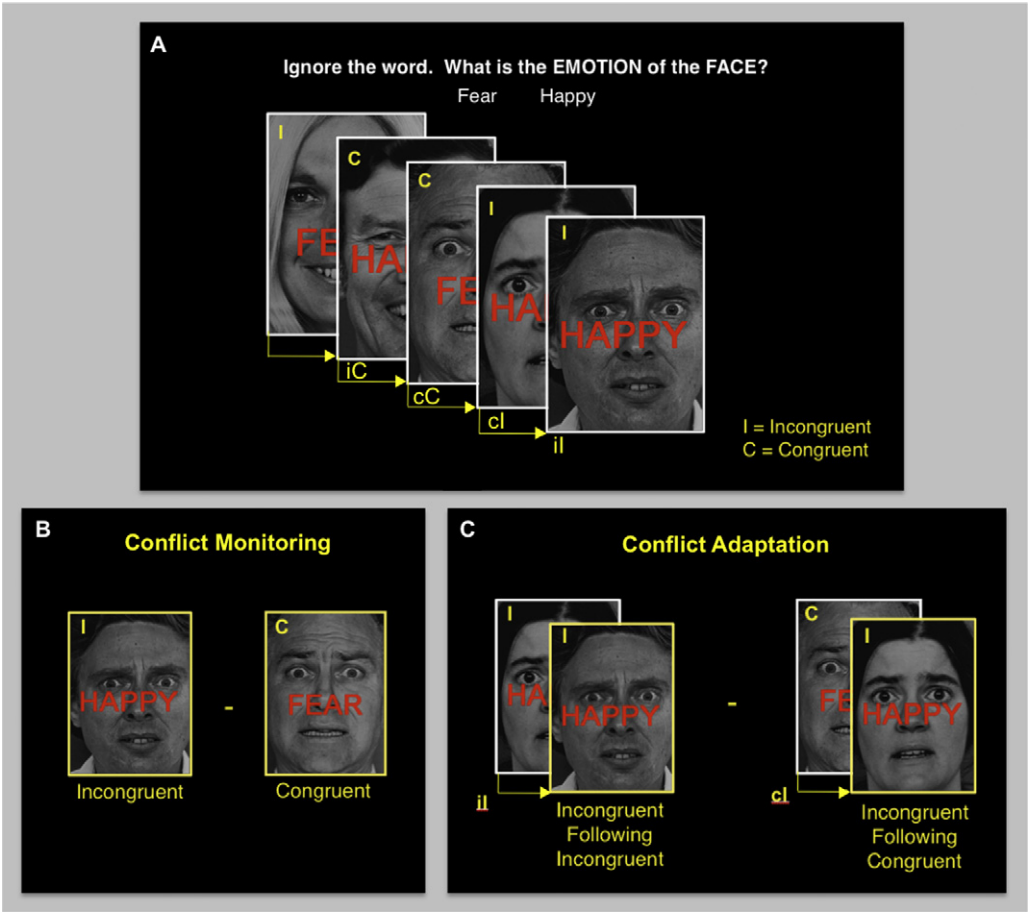

Fig. 1. Emotion conflict task. (A) A sample of the experimental paradigm's stimulus presentation sequence. (B) Emotional conflict detection is assessed by subtracting congruent trials (C) from incongruent trials (I). (C) Emotional conflict adaptation is assessed by subtracting incongruent trials that follow congruent trials (cI), from incongruent trials that follow incongruent trials (iI).

been associated with dysregulation in a neural circuit comprised of medial prefrontal cortex (mPFC) and amygdala, which facilitates emotion regulation in healthy individuals (Etkin et al., 2010; Etkin and Schatzberg, 2011)

Most research on the neural correlates of emotion regulation has examined adults who are healthy, or affected by psychopathology. To date, no study has assessed the degree to which behavioral characteristics during childhood prospectively predict the neural correlates of emotion regulation in adults. Such work is of major theoretical importance, since clinical factors during childhood predict risk for anxiety in adulthood (Pine et al., 1998). This raises vital questions about the degree to which such clinical factors also predict neural mechanisms engaged by emotion regulation, over and above any potential association with adult clinical factors. In the present study, we examined one such childhood behavioral characteristic, childhood temperament. Specifically, we examined adults who, during childhood, were characterized based on their expression of behavioral inhibition (BI). BI is a temperament that can be identified in infancy, and presents in children as extreme fear of social novelty that persists across early development (reviewed by Fox et al., 2005). Social reticence, a primary characteristic of children with BI (Henderson et al., 2004), is associated with low self-esteem, and high rates of peer victimization (reviewed by Rubin et al., 2009). Moreover, emerging fMRI research suggests that BI during childhood predicts brain function in adolescents and adults. However, only six such studies exist, and all six examine the relationship between early childhood BI and emotional reactivity (Bar-Haim et al., 2009; Guyer et al., 2006; Helfinstein et al., 2011; Perez-Edgar et al., 2007; Schwartz et al., 2011, 2003). The current study is the first to examine the relationship between childhood BI and the neural mechanisms engaged by emotion regulation. Given that leading theories (Derryberry and Rothbart, 1997; Rothbart et al., 1994) propose that two central constructs contribute to temperament, emotional reactivity, as modeled in the six prior imaging studies, and emotional regulation, which has yet to be tested, this research is a critical extension of prior work.

Although some data suggest that children with BI are at risk for developing anxiety disorders (Chronis-Tuscano et al., 2009; Fox et al., 2005), the strength of this prediction varies as a function of the sample, the methods used to assess BI, and the type of anxiety disorder. Indeed, there is substantial discontinuity in the expression of the BI phenotype with maturation into adolescence and adulthood. Thus, there is a great interest in identifying factors associated with both continuity and discontinuity in the expression of BI across development (e.g., Calkins et al., 1996; Fox et al., 2001). The current study sought to address this issue. Here we report on the first study to compare brain regions engaged by cognitive control in adults with and without childhood BI.

There is evidence to suggest dysregulation in the neural mechanisms associated with cognitive control in individuals with a history of BI. For example, during a flanker task, adolescents with childhood BI exhibited heightened error-related negativity (ERN) response, relative to those without childhood BI (McDermott et al., 2009). The ERN has been attributed to activity in mPFC, and is thought to reflect heightened attention to, and detection of errors in performance (e.g., Yeung et al., 2004). Similarly, Henderson (2010)