



Cognitive conflict adaptation in generalized anxiety disorder

Michael J. Larson^{a,b,*}, Ann Clawson^a, Peter E. Clayson^{a,c}, Scott A. Baldwin^a

^a Department of Psychology, Brigham Young University, Provo, UT 84602, United States

^b Neuroscience Center, Brigham Young University, Provo, UT 84602, United States

^c Department of Psychology, University of California, Los Angeles, CA, United States

ARTICLE INFO

Article history:

Received 7 May 2013

Accepted 26 August 2013

Available online 1 September 2013

Keywords:

Anxiety
Generalized anxiety disorder
Cognitive control
Conflict adaptation
Event-related potential
N2
Anterior cingulate (ACC)

ABSTRACT

Individuals with generalized anxiety disorder (GAD) display poor emotional conflict adaptation, a cognitive control process requiring the adjustment of performance based on previous-trial conflict. It is unclear whether GAD-related conflict adaptation difficulties are present during tasks without emotionally-salient stimuli. We examined conflict adaptation using the N2 component of the event-related potential (ERP) and behavioral responses on a Flanker task from 35 individuals with GAD and 35 controls. Groups did not differ on conflict adaptation accuracy; individuals with GAD also displayed intact RT conflict adaptation. In contrast, individuals with GAD showed decreased amplitude N2 principal component for conflict adaptation. Correlations showed increased anxiety and depressive symptoms were associated with longer RT conflict adaptation effects and lower ERP amplitudes, but not when separated by group. We conclude that individuals with GAD show reduced conflict-related component processes that may be influenced by compensatory activity, even in the absence of emotionally-salient stimuli.

© 2013 Elsevier B.V. All rights reserved.

1. 1 Introduction

There is an increasing focus on the role of cognitive and emotional control regulation processes on the mechanisms underlying psychiatric disorders. Understanding the nature of these processes may be useful in differentiating between disorders with similar pathophysiology and in understanding the mechanisms that contribute to psychopathology. Specifically, in generalized anxiety disorder (GAD) studies suggest that poor ability to detect emotional conflict and subsequently alter behavior may underlie dysfunctional emotion regulation behaviors and may be tied to altered attention and inhibitory control mechanisms (Etkin, Prater, Hoft, Menon, & Schatzberg, 2010). However, the preponderance of research to date has focused on the influence of emotional conflict on cognitive processing (i.e., when there are conflicting emotional stimuli). Due to the absence of research on cognitive control functioning in anxiety without emotional stimuli, it is unclear whether findings are due to generalized decrements in cognitive control or whether dysregulation is specific to the processing of emotional stimuli (Ernst, 2010). Thus, exploring conflict processing using non-emotional stimuli may help to elucidate the nature of deficits in cognitive processing in individuals with GAD.

A potential way to understand putative deficits in conflict processing in anxiety is through studies of emotional and cognitive conflict adaptation (also referred to as sequential-trial or Gratton effects; Gratton, Coles, & Donchin, 1992). Conflict adaptation requires both the accurate detection of conflict and the subsequent signaling for increased cognitive resources to adjust performance (Botvinick, Carter, Braver, Barch, & Cohen, 2001; Gratton et al., 1992). Conflict adaptation is typically seen during conflict-laden tasks, such as the Stroop or flanker, where conflict is created by similar (i.e., congruent) or differing (i.e., incongruent) target-stimulus properties and task-irrelevant information. Tasks that utilize cognitive or emotional target stimuli facilitate the examination of either cognitive or emotional processes (respectively) on conflict detection and resolution. Tasks that utilize task-irrelevant emotional distractors facilitate the examination of the impact of distracting emotional information on cognitive processes operating outside of the emotional system (Egner, Etkin, Gale, & Hirsch, 2008). Studies of emotional conflict adaptation in GAD suggest that poor abilities to detect emotional or cognitive conflict and subsequently adjust performance may play a role in clinical anxiety, as clinical levels of perseverative worry associated with GAD may place additional demands on cognitive systems in part by taxing attentional control systems (Etkin & Schatzberg, 2011). Differences in the processing of emotional information relative to “purely” cognitive information may elucidate cognitive processes that contribute to pathological levels of anxiety, including whether it is the cognitive processes

* Corresponding author. Tel.: +11 801 422 6125; fax: +11 801 422-0163.
E-mail address: michael.larson@byu.edu (M.J. Larson).

that are impaired in clinical anxiety, or whether it is the processing of irrelevant emotional information that interferes with cognitive processing.

The neural time course of these conflict adaptation processes can be measured using the conflict N2 component of the scalp-recorded event-related potential (ERP). The conflict N2 is a negative deflection in the ERP with a fronto-central scalp distribution that peaks approximately 250–350 ms after stimulus presentation (Nieuwenhuis, Yeung, Van Den Wildenberg, & Ridderinkhof, 2003; Yeung, Botvinick, & Cohen, 2004). The conflict N2 appears to be generated in the anterior cingulate cortex (ACC), and is more negative on incongruent than congruent trials, suggesting that it may reflect the allocation of top-down cognitive control to reduce conflict (van Veen & Carter, 2002a,b; Yeung et al., 2004). Conflict N2 amplitudes are less negative on incongruent trials preceded by incongruent trials (il) relative to incongruent trials following congruent trials (cl) and on congruent trials following incongruent trials (ic) relative to congruent following congruent trials (cc; Clayson & Larson, 2011a; Clayson & Larson, 2011b). This top-down biasing decreases levels of conflict on the following trial, resulting behaviorally in faster response times (RTs) and decreased error rates on il trials relative to cl trials (e.g., Clayson & Larson, 2011a; Clayson & Larson, 2011b; Forster, Cameron, Cohen, & Cho, 2011; Gratton et al., 1992; Ullsperger, Bylsma, & Botvinick, 2005). Alternatively, faster RTs and lower error rates on il relative to cl trials may be the result of the facilitative effects of repetition priming (Mayr, Awh, & Laurey, 2003), although this possibility remains controversial and studies using the precise task employed in the current study show conflict adaptation effects when repetition priming is controlled (Clayson & Larson, 2011a; Clayson & Larson, 2011b; Forster et al., 2011; Ullsperger, Bylsma, & Botvinick, 2005). Conversely, RTs and error rates are increased on ic trials relative to cc trials due to switching between congruencies (see Egner, 2007, for review). These neural and behavioral indices of conflict adaptation may be sensitive to subtle differences in cognitive processing, ideal for identifying the specific nature of cognitive processing deficits in anxiety disorders.

Research suggests that the neural processes implicated in conflict adaptation are altered in GAD. During conflict adaptation, the ACC detects conflict and subsequently signals the dorsolateral prefrontal cortex (PFC) and right ventromedial PFC to increase cognitive control (Egner, 2011; Egner & Hirsch, 2005; Kerns et al., 2004). In emotional conflict adaptation, activity increases within the rostral ACC, inhibiting activity within the amygdala in order to decrease emotional responsiveness to distracting affective stimuli (Egner, Etkin, Gale, & Hirsch, 2008). In a recent study conducted by Etkin, Prater, Hoefl, Menon, & Schatzberg (2010), individuals with GAD displayed decreased activation of the ACC that subsequently did not dampen amygdala response on an emotional conflict task in which emotionally salient words were overlaid on emotional faces (e.g., the word HAPPY overlaid on a fearful face). These results may suggest that GAD is associated with an attenuated response to conflict resulting in impaired top-down control and emotional dysregulation (Etkin et al., 2010). Similarly, individuals with panic disorder showed decreased conflict adaptation effects relative to psychiatrically-healthy controls during a similar emotional conflict task, including reduced dorsal ACC recruitment and increased amygdala activation during trials following incongruent trials (Chechko et al., 2009). Taken together, these findings suggest that abnormal patterns of activation observed within the ACC and amygdala during emotional conflict adaptation may be related to emotional dysregulation associated with anxiety.

Behavioral studies of individuals with clinical levels of anxiety corroborate neuroimaging findings by demonstrating decreased control following conflict, highlighting important differences in top-down regulation. Across multiple studies using an emotional conflict task, individuals with GAD did not display increased RTs

relative to controls on il trials relative to cl trials (Etkin et al., 2010; Etkin & Schatzberg, 2011), evidencing deficits specific to emotional conflict adaptation. Further, the degree of impairment in emotional conflict adaptation was correlated with levels of anxiety among patients with GAD, adding support to a relationship between conflict processing deficits and symptoms of clinical anxiety (Etkin et al., 2010). Together, these findings suggest that pathological anxiety may differentially alter cognitive control mechanisms during emotional processing, resulting in decreased cognitive flexibility that may contribute to the pervasive maintenance of worry characteristic of clinical anxiety.

Though it is clear that both neural and behavioral indices of conflict adaptation are altered in anxiety disorders, it is uncertain whether conflict processing deficits are primary, secondary, or independent of deficits in emotional processing. In one behavioral study, adults with obsessive compulsive disorder (OCD) and unipolar depression displayed response repetition slowing during a non-emotional Stroop task, possibly due to increased levels of rumination in these individuals (Merian, Diamond, Toder, & Nemets, 2010). These findings lend support to possible conflict adaptation deficits in the absence of emotional stimuli in anxiety disorders. However, no studies to date have examined neural and behavioral indices of conflict adaptation in GAD without emotional stimuli. If differences are present on purely cognitive tasks, it may be that anxiety disorders are associated with deficits in conflict adaptation independent of emotional processing.

Of note, conflict adaptation processes may differ among anxiety disorders and other comorbid conditions. Whereas GAD shares many characteristics with other anxiety disorders, the nature of the pervasive worry in GAD may uniquely affect cognitive systems involved in conflict adaptation by reducing the available resources needed to complete the task (Etkin & Schatzberg, 2011). It may also be that differences in compensatory processes associated with anxiety are related to distinct cognitive processes that distinguish anxiety disorders (Armstrong, Zald, & Olatunji, 2011). While some similarities in behavioral performance and neural activation have been observed among anxiety disorders and other comorbid conditions (e.g., depression), distinct patterns of activation and behavior have also been observed, again pointing to important differences in cognitive processing and neural activation (Etkin & Schatzberg, 2011). Thus, our primary focus was to determine the nature of cognitive control processing in individuals with GAD.

Based on the absence of neural and behavioral research in anxiety disorders, we aimed to compare electrophysiological (conflict N2) and behavioral (RTs, error rates) indices of conflict adaptation between individuals with GAD and controls using a non-emotional modified flanker task (Eriksen & Eriksen, 1974). Based on previous research, we predicted that participants with clinical levels of anxiety would display reduced behavioral modification and electrophysiological activation based on previous trial conflict, suggestive of deficits in basic cognitive processing. Specifically, we hypothesized that individuals with GAD would display attenuated N2 amplitudes relative to controls and would not display modulations in N2 amplitude based on previous-trial conflict. Similarly, we predicted that individuals with GAD would not display increased RTs on il trials relative to cl trials, again indicating deficits in cognitive control following conflict.

2. Method

2.1. Participants

All participants provided written informed consent as approved by the Brigham Young University Institutional Review Board. Individuals with GAD were recruited via referral from university and

متن کامل مقاله

دریافت فوری ←

ISIArticles

مرجع مقالات تخصصی ایران

- ✓ امکان دانلود نسخه تمام متن مقالات انگلیسی
- ✓ امکان دانلود نسخه ترجمه شده مقالات
- ✓ پذیرش سفارش ترجمه تخصصی
- ✓ امکان جستجو در آرشیو جامعی از صدها موضوع و هزاران مقاله
- ✓ امکان دانلود رایگان ۲ صفحه اول هر مقاله
- ✓ امکان پرداخت اینترنتی با کلیه کارت های عضو شتاب
- ✓ دانلود فوری مقاله پس از پرداخت آنلاین
- ✓ پشتیبانی کامل خرید با بهره مندی از سیستم هوشمند رهگیری سفارشات