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Invited Review

Making sense of all the conflict: A theoretical review and critique of conflict-related ERPs

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ABSTRACT

Cognitive control theory suggests that goal-directed behavior is governed by a dynamic interplay between areas of the prefrontal cortex. Critical to cognitive control is the detection and resolution of competing stimulus or response representations (i.e., conflict). Event-related potential (ERP) research provides a window into the nature and precise temporal sequence of conflict monitoring. We critically review the research on conflict-related ERPs, including the error-related negativity (ERN), Flanker N2, Stroop N450 and conflict slow potential (conflict SP or negative slow wave [NSW]), and provide an analysis of how these ERPs inform conflict monitoring theory. Overall, there is considerable evidence that amplitude of the ERN is sensitive to the degree of response conflict, consistent with a role in conflict monitoring. It remains unclear, however, to what degree contextual, individual, affective, and motivational factors influence ERN amplitudes and how ERN amplitudes are related to regulative changes in behavior. The Flanker N2, Stroop N450, and conflict SP ERPs represent distinct conflict-monitoring processes that reflect conflict detection (N2, N450) and conflict adjustment or resolution processes (N2, conflict SP). The investigation of conflict adaptation effects (i.e., sequence or sequential trial effects) shows that the N2 and conflict SP reflect post-conflict adjustments in cognitive control, but the N450 generally does not. Conflict-related ERP research provides a promising avenue for understanding the effects of individual differences on cognitive control processes in healthy, neurologic and psychiatric populations. Comparisons between the major conflict-related ERPs and suggestions for future studies to clarify the nature of conflict-related neural processes are provided.

1. Introduction: cognitive control and conflict monitoring

Cognitive control theory provides an influential model for understanding the specific roles and neuroanatomical substrates of prefrontal cortex function (e.g., Botvinick et al., 2001; Miller and Cohen, 2001; O'Reilly et al., 2010; Shenhav et al., 2013). Cognitive control specifically refers to the ability to guide thoughts and actions in accord with internal intentions (Miller and Cohen, 2001). Two main component processes necessary for the accurate completion of goal-directed behavior are regulative and evaluative in nature (see Botvinick et al., 2001, 2004; Kerns et al., 2004; MacDonald et al., 2000; Perlstein et al., 2006). Regulative control processes include the implementation of top-down control to complete and adjust to task demands, the allocation of attention and control resources, maintenance of task and rule context, and preparation to override task-inappropriate response tendencies. Such regulative control functions are needed for correct task performance and implementation of dynamic adjustments in behavior. Evaluative control processes, in contrast, include monitoring performance for conflict or mistakes, signaling the need to change implementation of control, and providing ongoing feedback about the need for control. Anatomically, regulative control processes are theorized to occur in areas of the dorsolateral prefrontal cortex (dlPFC) and ventrolateral prefrontal cortex (vlPFC); evaluative control processes consistently seem to be implemented in the anterior cingulate cortex (ACC; Egner, 2011; Egner and Hirsch, 2005a,b; Kerns et al., 2004; MacDonald et al., 2000; Ridderinkhof et al., 2004).

Cognitive control theory is both lauded and criticized for its ability to account for prefrontal cortex activity and subsequent behaviors (e.g., Brown, 2011; Niendam et al., 2012; Schmidt, 2013; Shenhav et al., 2013). Studies testing specific hypotheses that stem from cognitive control theory are largely supportive (see Niendam et al., 2012 for an interesting meta-analysis), but questions remain about the precise interplay between dlPFC and vlPFC-mediated regulative control functions and ACC-mediated evaluative control functions and whether alternative explanations, such as time on task, reinforcement learning,

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priming, feature integration, or dynamic network adjustments can better account for findings that are thought to support cognitive control theory (e.g., Brown, 2011; Holroyd and Coles, 2002; Hommel et al., 2004; Mayr et al., 2003; Scherbaum et al., 2012; Schmidt, 2013).

Critical to cognitive control theory is the idea that the ACC detects conflict in performance or the environment and subsequently signals for increased implementation of control from the dlPFC or vlPFC. Conflict specifically refers to the simultaneous activation of competing stimulus or response options (Botvinick et al., 2001, 2004, 1999). For example, in the commonly-used Stroop task (Stroop, 1935), participants are instructed to identify the name of the color of the stimulus when presented with color–words where the word and color are congruent (e.g., the word RED written in red font) or where the word and color are incongruent (e.g., the word GREEN written in red font). Conflict occurs on the presentation of incongruent trials due to the simultaneous activation of the prepotent written word and the color. Similarly, in the traditional Eriksen Flanker Task (Eriksen and Eriksen, 1974), participants are told to respond to the direction of the middle 'target' stimulus while flanking stimuli are either congruent (e.g., <<<<<) or incongruent (e.g., <<><<) from the target. Similar to the Stroop task, conflict is induced in the incongruent condition by the simultaneous prompting of both correct and incorrect responses.

Conflict monitoring, therefore, refers to the process of monitoring performance for simultaneously competing response options (e.g., the simultaneous activation of error and correct response options or the simultaneous activation of two competing stimulus properties). If conflict is not adequately detected and subsequent adjustments in behavior and neural resources are not implemented, goal-directed behaviors will falter. For example, neuropsychologists working with individuals who have experienced moderate or severe traumatic brain injury (TBI) frequently report that these individuals make the same mistakes repeatedly even when corrective feedback is provided (Demery et al., 2010; Larson et al., 2009a, 2007a, 2007b; Slomine et al., 2002; Sozda et al., 2011). Studies from a conflict monitoring perspective suggest that the continued errors in those with TBI are due to poor detection and subsequent resolution of response conflict (Duncan et al., 2011; Larson et al., 2011a, 2009a, 2007a). Understanding specific deficits or excesses in conflict monitoring could lead to potential pharmacological or behavioral treatments focused on performance monitoring aspects of cognitive control in individuals with TBI or other psychiatric disorders such as autism or depressive and anxiety disorders (e.g., Clawson et al., 2013; Endrass et al., 2008; Larson et al., 2013a, 2012c; Weinberg et al., 2012, 2010). First, however, the field must develop a clear understanding of conflict-related control and the neural mechanisms associated with both healthy and dysfunctional conflict monitoring processes.

In a formative paper on cognitive control and conflict monitoring, Botvinick et al. (2001) used computational models to put forward the general mechanisms behind conflict monitoring. Specifically, they indicated that there is an ACC-mediated conflict monitoring (i.e., evaluative) mechanism that monitors information processing, makes an assessment of current demands, and signals for increased recruitment of control when information processing demands exceed the current level of control, such as in situations of high conflict (see Stroop example above). Botvinick and others have extrapolated some on this model (e.g., Shenhav et al., 2013), but the general concept of monitoring performance for conflict remains critical to cognitive control theory. At the conclusion of their paper, Botvinick et al. (2001) put forward several questions for future investigation of conflict monitoring processes. These questions included whether the ACC responds to conflict, how ACC-related activity impacts cognitive control, how conflict is measured, at what levels of processing conflict is measured, and how rich the information is that conflict monitoring provides to cognitive control. Subsequent theoretical and computational modeling papers, many by the Yeung et al. group (e.g., Yeung et al., 2004; Yeung and Cohen, 2006) have provided additional questions about conflict monitoring including whether conflict is increased with competing response representations, whether context biases the conflict monitoring system, how the strength of conflict-related signals changes with increased stimulus or response conflict, and whether conflict-related activity should vary based on the strength of regulative control implementation. Some of these questions have been addressed in the literature, whereas many remain unanswered.

How can these questions be addressed in a systematic way and what are the manifestations of conflict that can aid us in determining the underlying nature of conflict processes? Behaviorally, the presence of conflict in performance or the environment leads to slowing of responses (e.g., individuals respond slower to incongruent trials [high conflict] on the Stroop task relative to congruent trials [low conflict]) and/or increased numbers of errors (Shenhav et al., 2013). Increased response slowing or higher error rates could be indicative of increased conflict on incongruent trials compared to congruent trials. In contrast, response speeding or reduced error rates may indicate increased implementation of control associated with adjustments to previously high levels of conflict. However, such behavioral manifestations are non-specific and can often be accounted for by priming or other non-conflict explanations such as time on task or feature integration (Brown, 2011; Hommel et al., 2004; Mayr et al., 2003; Schmidt, 2013). Potentially more specific measures come from methodologies that directly examine conflict-related neural activity with excellent temporal resolution in order to identify conflicting options that occur within milliseconds of stimulus presentation or response selection. Scalp-recorded event-related potentials (ERPs) provide just such a tool and have been used extensively in the study of conflict. Event-related potentials are advantageous over spatially-oriented methods, such as functional magnetic resonance imaging (fMRI), due to the decreased time to acquire reliable conflict-related data and the temporal specificity of the results. Furthermore, the amplitude and latencies of conflict-related ERPs aid in determining the magnitude of the conflict in the stimulus or response as well as the onset of conflict recognition and resolution — data that would be obscured by the slower hemodynamic response in blood-flow based methodologies.

The purpose of this paper is to critically review the literature on conflict-related ERPs. We first summarize the available evidence from ERPs that addresses the key questions noted above. Then we highlight ERPs functionally thought to represent error-related conflict processing, stimulus-locked conflict processing, and conflict adaptation and indicate how these ERPs inform our knowledge of conflict-related control. We also briefly address the roles of affective context and neurologic or psychiatric impairment in conflict processing. Finally, we provide a comparison of commonly investigated stimulus- and response-locked ERPs and their functional significance. We specifically focus on the contributions of research on the error-related negativity (ERN), Flanker N2, Stroop N450, and conflict slow potential (also known as the conflict SP or negative slow wave [NSW]). Additional components could be included, but are non-specific, not clearly related to conflict, or are beyond the scope of this review (e.g., the P300, correct-related negativity [CRN], feedback-related negativity [FRN], or the error positivity [Pe]) and would have decreased the focus of the review on conflict-related ERPs. Thus, we chose to focus only on the aforementioned conflict-related ERPs. Specifically, we present information as to how these ERP components inform our knowledge of conflict-related processing. Throughout the review we also provide critiques and remaining questions that have not yet been thoroughly addressed in our understanding of conflict and conflict-related ERPs.

2. The error-related negativity

The error negativity (Ne; Falkenstein et al., 1991) or error-related negativity (ERN; Gehring et al., 1993) is a response-locked ERP that occurs within 100 ms of an erroneous response and has been localized to the ACC (Brazdil et al., 2005; Bush et al., 2000; Stemmer et al., 2004; van Veen and Carter, 2002a; see Fig. 1). The ERN is relatively

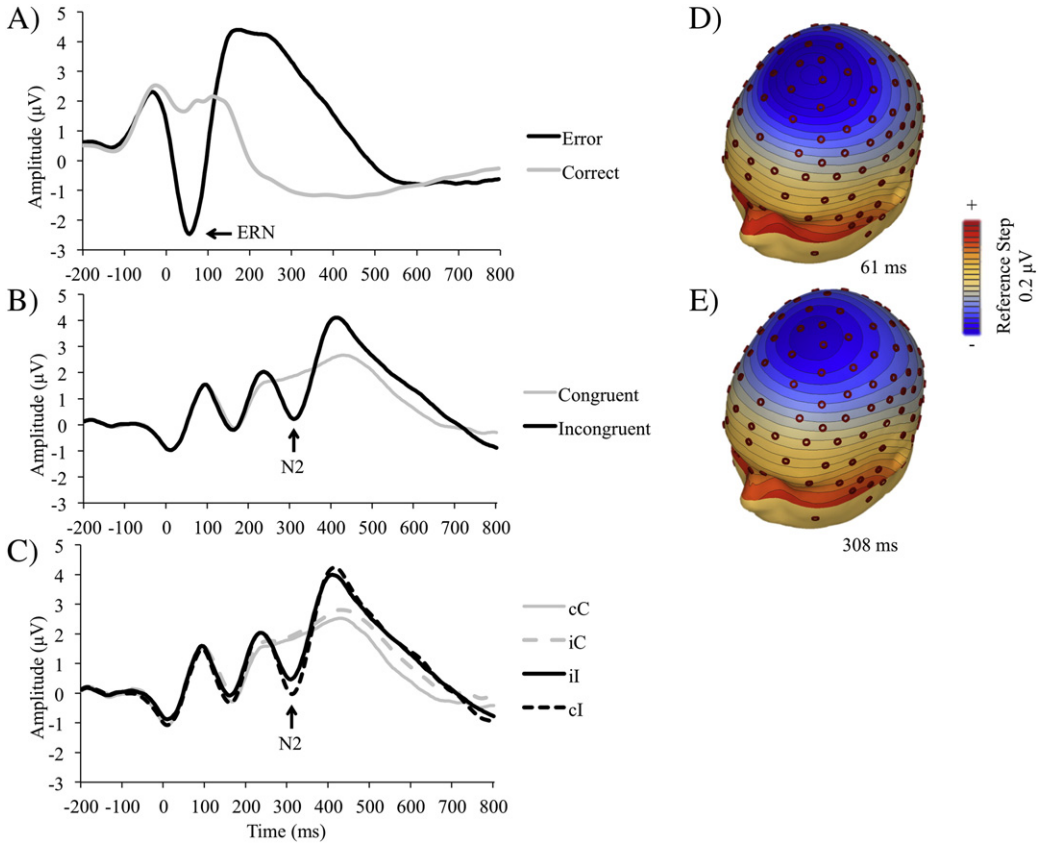

Fig. 1. A) Grand averaged response-locked ERP activity averaged from healthy participants across fronto-central electrode locations for the error-related negativity (ERN) recording during a modified Eriksen Flanker Task. B) Grand averaged stimulus-locked ERP waveforms from healthy participants averaged across fronto-central electrode locations for the N2 for congruent and incongruent trials from a traditional Flanker task. C) Grand averaged stimulus-locked ERP waveforms averaged across fronto-central electrode locations for the N2 for congruent trials preceded by congruent trials (cC), congruent trials preceded by incongruent trials (iC), incongruent trials preceded by incongruent trials (iI), and incongruent trials preceded by congruent trials (cI). D) Voltage map for response-locked difference activity (error minus correct) for the ERN. E) Voltage map for stimulus-locked difference activity (incongruent minus congruent) for the N2.

robust across multiple sensory modalities (Miltner et al., 1997) during tasks that involve the inhibition of prepotent or automatic responses (e.g., the Stroop task, Go/No-Go task) or interference caused by distracting irrelevant stimuli (e.g., the Eriksen Flanker Task). In order to elicit an ERN, target and task-irrelevant stimuli must be processed to a level of subjective awareness (Charles et al., 2013; Woodman, 2010), suggesting that the ERN represents higher-level cognitive processing that occurs after stimulus information has been brought to perceptual attention.

According to the conflict monitoring theory, the ERN is thought to be a marker of response conflict that occurs during error commission, often appearing immediately before or during the execution of an erroneous response. Response conflict occurs with the execution of an erroneous response, when the incorrect response conflicts with the correct response queued by the continued processing of the target stimulus (Yeung et al., 2004). Accordingly, the degree of competition between correct and incorrect response options should be reflected in the amplitude of the ERN, with greater conflict associated with more negative ERN amplitudes (Botvinick et al., 2001; Yeung et al., 2004). These alterations in response conflict theoretically act as a signal for other brain regions, such as the dlPFC or vlPFC, that are involved in implementing greater cognitive control and triggering behavioral adjustments (Kerns, 2006; Kerns et al., 2004; MacDonald et al., 2000). Consequently, response conflict signified by the ERN reflects performance monitoring processes wherein cognitive performance is monitored and adapted based on the task and performance context (e.g., task demands, cognitive goals; Botvinick et al., 2001; Yeung et al., 2004).

Several additional theories have been proposed regarding the ERN. One theory suggests that the ERN represents an error-detection or awareness signal resulting from a mismatch between the erroneous response and correct response (Falkenstein et al., 1991, 2000). Alternatively, the reinforcement learning theory posits that the ERN represents a negative reinforcement signal that occurs when events are worse than expected. Phasic dips in midbrain dopamine signal the occurrence of an error, disinhibiting the ACC in order to modify task performance (Holroyd and Coles, 2002). Other theories more explicitly acknowledge the role of emotion and motivation in the ERN, suggesting that errors in motivationally significant contexts (e.g., errors associated with greater loss of an incentive) may trigger a heightened emotional reaction, resulting in a larger-amplitude ERN (Chiu and Deldin, 2007; Vidal et al., 2000). We acknowledge the utility of these theories in the interpretation of the literature reviewed below and feel that further research contrasting and integrating these perspectives is requisite to more fully understand the significance of the ERN. However, for the sake of brevity and in keeping with the emphasis of this paper on ERPs associated with conflict monitoring, we will primarily focus our interpretations in terms of the conflict monitoring theory.

2.1. The role of conflict in ERN generation

A critical assumption of the conflict monitoring theory is that ERN amplitudes reflect dynamic changes in response conflict. A key assumption in cognitive control theory is the ability of the cognitive control system to flexibly recruit cognitive resources, including attention, to monitor the environment and signal the detection of conflict. Understanding how manipulations of attention to conflicting stimuli influence ERN amplitudes is an important step in determining whether the ERN is sensitive to the degree of conflict and whether ERN amplitudes are dynamic based on the task context.

There are several studies that support the assumption of dynamic alterations in cognitive control, including trial-by-trial alterations in ERN amplitude, which have been documented using traditional cognitive control tasks (e.g., Flanker, Stroop). For example, increased attentional focus on correct responses results in a larger ERN when accuracy is emphasized over speed (Gehring et al., 1993; Yeung et al., 2004). Similarly, ERN amplitudes are more negative for errors committed on incongruent trials preceded by incongruent trials compared to errors on incongruent trials that are preceded by congruent trials. This is purportedly due to the allocation of greater cognitive control resources following incongruent trials, leading to greater attention to the target stimulus to improve task performance (Larson et al., 2012b). This shift in attentional control facilitates greater detection of conflict between the correct target stimulus and committed erroneous response, resulting in larger ERN amplitudes. Thus, ERN amplitudes are larger during trials in which cognitive conflict is heightened, supporting the assumption that the ERN is sensitive to changes in cognitive conflict.

Several researchers have further examined this assumption by more directly manipulating response conflict. Hughes and Yeung (2011) compared trials during a conflict condition (using a traditional Flanker task) to trials during a masked condition, in which incongruent trials were replaced with masked congruent trials such that the masked trials produced the same proportion of errors and RTs as incongruent stimuli in the conflict condition. In this way, errors during the conflict condition were due to response conflict between competing correct/incorrect stimuli, but errors during the masked condition were due to stimulus degradation. ERN amplitudes were significantly greater for errors committed in the conflict condition compared to the masked condition, suggesting that degraded response stimuli reduced the level of correct response activation, resulting in overall reduced conflict activation. These findings suggest that the ERN is sensitive to the degree of activation of competing response options during the response selection process.

Studies also demonstrate that ERN amplitudes reflect the degree of competition between correct and incorrect response options. Stahl and Gibbbons (2007) measured ERN amplitude differences based on variations in the stimulus onset asynchrony on a stop-signal task, hypothesizing that conflict would increase with increased delays in the presentation of the stop signal due to increased attention to the correct response option that then conflicts with the incorrect response. The authors observed a larger ERN for correct response trials (long-delay stopped trails) relative to error trials (short-delay non-stopped trials), consistent with the hypothesis that the ERN is sensitive to changes in response conflict due to increased attention to the conflicting correct response option. Danielmeier et al. (2009) and Maier et al. (2012) similarly observed larger ERN amplitudes for errors in which flanker stimuli were spaced farther apart and when flankers were larger in size, respectively. When flankers were located far from the target stimulus or were smaller, less attention was directed to the flankers and more attention was directed to the correct-response target. Thus, when an error occurred there was greater conflict between the correct response and the incorrect response, leading to larger ERN amplitudes relative to trials in which target stimuli received less attention. Together, these studies demonstrate that increased attention to the correct response through parametric manipulations, which putatively led to greater activation of correct response options, resulted in greater cognitive conflict and a larger amplitude ERN following error commission.

In sum, research consistently indicates that the ERN is sensitive to changes in response conflict following dynamic adjustments of attention to correct and incorrect response representations. ERN amplitudes are largest when correct response representations are heightened, including situations with increased focus on correct stimuli (Danielmeier et al., 2009; Maier et al., 2012), situations when accuracy is emphasized (Gehring et al., 1993; Yeung et al., 2004), or situations in which both correct and incorrect response options are heightened (Hughes and Yeung, 2011). Whereas these studies are in-line with conflict monitoring theory explanations of error processing, plausible alternative explanations have not been ruled out and should be addressed in the future (e.g., response confidence, error awareness).

2.2. Affect and the ERN

Although the conflict monitoring theory does not explicitly account for the role of participant affect in cognitive control, understanding the impact of affect on these processes is essential to determine the nature of error processing and to rule out or integrate other theories of the ERN (e.g., the motivational salience theory). Several questions must be addressed to understand if and how affective context influences ERN amplitudes within the conflict monitoring theory. First, does affect influence the conflict monitoring system, or does affect change after conflict monitoring has occurred? If affective information is incorporated into conflict monitoring, at what level does this influence occur and does the affective information influence response conflict? Affective information may guide response selection at the attentional level, "biasing" attention to different sources of response information that are emotionally or motivationally relevant. Alternatively, affect may serve as an additional source of conflict that must be monitored during response selection. Understanding whether ERN amplitudes are modulated by changes in motivation or affect, and if or how this relates to cognitive conflict may lend insight into the type of information conveyed by this signal. Although extensive research has examined the role of affect and ERN generation, many of these questions have not yet been addressed to a level sufficient to fully understand the role of emotion in cognitive control. We review this literature in relation to conflict below.

2.2.1. The motivational significance of errors

Situations with heightened conflict are often aversive, particularly if they result in error commission or the loss of an incentive (Hajcak and Foti, 2008). Indeed, ERN amplitudes predicted startle potentiation following errors, possibly suggesting that the ERN reflects motivationally salient processes and may activate defensive systems (Hajcak and Foti, 2008). However, ERN amplitudes were not related to sympathetic nervous system arousal (e.g., changes in heart rate and skin conductance) following errors and may not be associated with autonomic responses to errors (Hajcak et al., 2003). If the ERN is related to the significance of performance outcomes, affect likely plays a role in ERN generation.

In order to understand the relationship between the ERN and the motivational significance of errors, several authors have examined whether the ERN is sensitive to changes in performance incentives. ERN amplitudes were larger following error trials when participants lost financial incentives (Gehring et al., 1993; Moser et al., 2005), suggesting that the ERN may reflect the motivational value of outcomes. Alternatively, individual characteristics may moderate the relationship between motivation and ERN amplitudes. Pailing and Segalowitz (2004) compared trials in which there were no incentives for accuracy to trials in which small payoffs were offered for accuracy. Differences in ERN amplitude based on task incentives were only present among individuals low in conscientiousness and high in neuroticism, possibly suggesting that individuals with a more external locus of control may selectively monitor their performance according to the consequences of the task (Pailing and Segalowitz, 2004). Supporting these findings, ERN amplitudes are related to satisfaction with life, suggesting that that individuals who are more satisfied with their life may view errors as less aversive or motivationally relevant (Larson et al., 2012b). Thus, the relationship between ERN and the motivational significance of outcomes may also reflect individual differences in motivation.

2.2.2. State versus trait affect

In order to further understand the relationship between ERN and affect, researchers have examined whether ERN amplitudes are

sensitive to changes in state affect or influenced by levels of trait affect. Manipulations of state affect may reveal whether ERN amplitudes reflect dynamic responses to the emotional context, while measures of trait affect determine whether more stable levels of affect underlie ERN generation. Again, few studies have directly manipulated the relationship between different levels of conflict and changes in state affect; however, current findings lend some insight into the role of emotion in ERN generation.

First, heightened levels of negative affect may be associated with increased ERN amplitudes. Hajcak et al. (2004) observed that participants with high self-reported levels of negative affect had larger ERN amplitudes and skin conductance responses following errors, although others have not shown a correlation between ERN amplitudes and levels of negative affect (Larson et al., 2010b). Luu et al. (2000) also observed enhanced ERN amplitudes in individuals with high levels of negative affect at the start of a task, but enhanced ERN amplitudes decreased later in the task, suggesting that individuals with heightened levels of negative affect may have initially over-attended to stimuli but later disengaged from the task. In line with these findings, Amodio et al. (2008) and Boksem et al. (2006) observed that individuals with heightened levels of the behavioral inhibition system (BIS) scores, but not the behavioral activation system (BAS) scores, displayed increased ERN amplitudes. The BIS is purportedly sensitive to levels of punishment and related to traits associated with negative affect, including neuroticism and anxiety (Amodio et al., 2008). Higher levels of negative affect may result in greater task engagement due to enhanced attention and vigilance (Gray, 1982), possibly leading to increased conflict representation and an increased ERN. Indeed, conflict activation in this regard appears to be greater among individuals with heightened BIS scores (Amodio et al., 2008; Boksem et al., 2006). Together, these studies suggest that levels of trait negative affect are associated with alterations in attention, resulting in greater conflict activation; however, in these studies little attention was simultaneously given to both state and trait affect.

The influence of changes in state affect on ERN amplitudes is less clear. Several studies reveal no differences in ERN amplitudes between different affective states, both when participants were given encouraging feedback relative to derogatory feedback (Clayson et al., 2012) or when affective state was examined orthogonally according to valence and arousal in calm, anxious, sad, and happy conditions (Larson et al., 2013b). In contrast, other studies indicate differences in ERN amplitudes based on manipulations of state affect (e.g., Compton et al., 2007; Larson et al., 2006; Wiswede et al., 2009a, 2009b), though the pattern of results is inconsistent across different affective states and the tasks used to elicit errors vary greatly, with Go/No-Go and Flanker tasks interspersed throughout the different studies (Compton et al., 2007; Inzlicht and Al-Khindi, 2012; Inzlicht and Tullett, 2010; Larson et al., 2006; Wiswede et al., 2009a, 2009b). The relationship between state affect and ERN amplitude remains unclear, with some evidence that state affect may influence ERN amplitudes.

Some discrepancies observed in studies of state affect may be explained by the influence of relevant trait characteristics that mediate subjective responses to induced affect. For example, neuroticism moderated the relationship between ERN amplitudes and changes in mood, with larger changes in mood associated with increases in ERN amplitudes (Olvet and Hajcak, 2012; Wiswede et al., 2009b). Similarly, though state-related manipulations did not influence changes in ERN amplitude, individuals with heightened levels of trait anxiety exhibited heightened ERN amplitudes during conditions with feedback (Olvet and Hajcak, 2009) and during the presence of a feared stimulus (Danielmeier et al., 2009). Again, negative affect may play a critical role in conflict-related ERN generation, pointing to the importance of examining how trait affect influences individual responses to state affect during error processing. Failing to account for these potential mediating variables may mischaracterize the relationship between affect and ERN generation.

2.2.3. Future directions in understanding the role of affect in error-related conflict monitoring

Results across studies indicate that individual characteristics or levels of trait affect may influence ERN amplitudes based on the motivational value of outcomes or levels of state anxiety. It is yet unclear, however, if and how affect influences conflict processing, as we cannot exclude explanations associated with graded levels of motivational or affective processing. Specifically, do affective variables lead to a preference toward a specific response representation, increasing attention to the target stimulus? This can only be determined by manipulating both affect and the degree of conflict between response representations. In addition, at what point does increased cognitive control become maladaptive? Understanding how contextual factors inform ERN amplitudes and conflict representation may assist in understanding at what point these processes are efficient and at what point they are ineffective.

2.3. The relationship between the ERN and performance change

The conflict monitoring theory posits that conflict signals are used to make adjustments in control to dynamically adapt to future conflict. The influence of this conflict signal can be measured by the degree of corresponding behavioral adjustments, as changes in behavior suggest that conflict signaling successfully marked the need for greater control. According to cognitive control theory, conflict monitoring is necessary but not sufficient for appropriate increases in control, as additional brain regions must respond to this conflict signal to implement control. Specifically, enhanced activity within the ACC and other prefrontal regions leads to decreases in activity in the motor system, resulting in post-error slowing (Danielmeier et al., 2011).

In line with this theory, Hajcak and Simons (2008) observed that conflict signaled by the ERN is not directly related to the implementation of remedial behaviors. The authors examined the association between ERN amplitudes and behavioral alterations following double-errors, a situation in which error commission has not been successfully avoided. The authors found no significant differences between ERN amplitudes following single-error trials relative to double-error trials, suggesting that double-errors occur due to a failure to implement control and not a failure of the action monitoring system to signal the need for greater control. Thus, control signified by ERN amplitudes may not be directly related to response activation, supporting the assumption that behavioral responses to conflict are implemented by other neural systems.

Implementation of control is typically measured by changes in behavior on trials immediately following error commission. Botvinick et al. (2001) hypothesized that these immediate behavioral adjustments reflect dynamic adjustments in cognitive control. If the correct response is strongly activated and chosen, response times (RTs) are short because the correct response representation overrides the incorrect response. If the incorrect response is strongly activated, either the correct response will override the error response, resulting in longer RTs, or the incorrect response will remain activated and an error will occur. Accordingly, post-error slowing is often observed on trials following error commission. The conflict monitoring theory suggests that post-error slowing signifies the successful implementation of compensatory adjustments to avoid error commission on trials following error trials (Rabbitt, 1966). Accordingly, an enhanced ERN signifying the need for greater control should be related to post-error slowing.

Several studies indicate that there is no relationship between post-error slowing and ERN amplitudes, suggesting that the ERN is not involved in remedial action following an error and may not be involved in sustaining adjustments in cognitive control (Dhar and Pourtois, 2011; Endrass et al., 2005, 2007; Hajcak et al., 2003; Larson and Clayson, 2011; Nieuwenhuis et al., 2001). These findings are supported by studies that indicate that changes in ERN amplitudes may not lead to changes in behavior, and that changes in behavior can occur independent of changes in ERN amplitude. For example, O'Connell et al. (2007) and

Olvet and Hajcak (2009) observed differences in RT slowing for aware and unaware trials and trials with feedback, respectively, despite a lack of differences in ERN amplitudes. In contrast, some researchers have observed that larger ERN amplitudes are associated with greater post-error slowing and may be more directly related to signaling changes in behavior, consistent with more of an error-recognition explanation of the ERN relative to a conflict-related explanation (Debener et al., 2005; Gehring et al., 1993; Hewig et al., 2011; Luu et al., 2000).

As noted previously, ERN amplitudes appear to mirror, to some degree, changes in response conflict. However, it is unclear how and to what extent the degree of conflict signaled by the ACC relates to adjustments in behavior. Although few studies have sought to address these questions, Dhar and Pourtois (2011) examined the malleability of this conflict processing system as related to changes in behavior. These authors observed that familiarization with task stimuli was related to changes in RTs but was unrelated to changes in ERN amplitudes, possibly suggesting that ERN amplitudes reflect an evaluative system that may remain consistent despite behavioral adaptation to conflict.

2.4. Is bigger better?

Although the conflict monitoring theory clearly outlines the relationship between ERN amplitudes and response conflict, the optimal threshold of conflict activation is unspecified and whether increased ERN amplitudes index better or worse performance is unclear. Some level of conflict activation is requisite to signify the presence of response conflict and guide response selection, but at what level does this activation become maladaptive? Assuming no differences in behavior, greater conflict activation, indicated by increased ERN amplitudes, may be indicative of greater conflict processing or greater attention to the environment. Alternatively, decreased ERN amplitudes in the absence of behavioral changes may indicate greater cognitive efficiency. These questions can only be partially answered until we understand what kind of information is conveyed by this system and how manipulations of conflict influence this system. Nevertheless, addressing this question may clarify discrepancies in the literature regarding the ERN and contribute to greater specificity in the conflict monitoring theory.

Work among individuals with pathology lends some insight into these questions. Individuals with clinically significant levels of anxiety (Ladouceur et al., 2006; Weinberg et al., 2010) and obsessive–compulsive disorder (Endrass et al., 2008; Hajcak et al., 2008) consistently display increased ERN amplitude, suggesting chronic overactive detection of conflict that may be tied to pathological worry or maladaptive perfectionism (Moser et al., 2012; Weinberg et al., 2010). Individuals with schizophrenia (Alain et al., 2002), autism spectrum disorders (Sokhadze et al., 2010; South et al., 2010), TBI (Larson et al., 2007a), and substance abuse (Franken et al., 2007; Smith and Mattick, 2013) display attenuated ERN amplitudes, possibly reflecting a reduced awareness of or attention to conflicting responses necessary to monitor performance. ERN amplitudes in major depressive disorder are somewhat inconsistent, with some studies revealing increased ERN amplitudes (Chiu and Deldin, 2007; Holmes and Pizzagalli, 2008, 2010) and some decreased ERN amplitudes (Schrijvers et al., 2009, 2008). Taken from a conflict monitoring perspective, it seems that individuals with heightened focus on errors in performance, such as those with perfectionistic or anxious tendencies, are those that most frequently exhibit the largest ERN amplitudes (Moser et al., 2012; Weinberg et al., 2010).

Differences among individuals without pathology also indicate the relevance of subtle differences in ERN amplitude. Increased attention/executive functioning ability is correlated with increased amplitude ERN, indicating that greater attention to performance may lead to increased attention to competing response options (Larson and Clayson, 2011). Similarly, enhanced ERN amplitudes are related to higher working memory span scores, suggesting that greater working memory may increase the overall capacity for the representation of conflicting response options (Miller et al., 2012). Individuals with higher levels of empathy also display larger ERN amplitudes (Larson et al., 2010a; Santesso and Segalowitz, 2009), possibly due to greater vigilance to the environment or greater concern for positive outcomes. Whereas these findings suggest that enhanced ERN amplitudes may be associated with characteristics that improve conflict monitoring, Moser et al. (2012) observed that larger ERN amplitudes and poor behavioral modification following errors were associated with anxious apprehension (worry) among healthy undergraduates, suggesting that heightened levels of worry may lead to chronic elevations of cognitive conflict that result in inefficient processing. Thus, it is unclear whether heightened ERN amplitudes reflect greater awareness of cognitive conflict, or overactive conflict monitoring.

In a similar vein, reduced ERN amplitudes may not necessarily reflect increased efficiency in processing or decreased awareness of conflict. Decreased ERN amplitudes are associated with high levels of impulsivity (Potts et al., 2006; Ruchsow et al., 2005) that may reduce appropriate alterations in stimulus and/or target processing. In addition, males display increased ERN amplitudes relative to females and may require greater conflict activation for similar performance, suggesting that males may be less efficient at monitoring conflict than females (Larson et al., 2011b). Interestingly, behavioral studies suggest that women may process task-irrelevant information (e.g., the flanking stimuli on a Flanker task) to a greater degree than men (Clayson et al., 2011; Judge and Taylor, 2012; Stoet, 2010), again supporting the theory that an attenuated ERN may signify less efficient conflict monitoring in women relative to men.

As a whole, the state of the current literature indicates that contextual factors must be considered when evaluating the significance of an enhanced/attenuated ERN. It is evident that individual differences in affect or cognitive capacity may constrain the cognitive control system. Larger ERN amplitudes may be adaptive in contexts in which conflict is heightened and greater attention is required, and smaller ERN amplitudes may signify increased efficiency when contextual demands are consistent. This system may then become maladaptive when ERN amplitudes are chronically elevated or attenuated regardless of environmental demands, either taxing resources unnecessarily or decreasing the likelihood of adaptive performance.

2.5. ERN summary, conclusions, and directions for future research

To summarize, the state of the current research lends support to the role of the ERN as an index or reflection of conflict detection as outlined by the conflict monitoring theory. The preponderance of research examining manipulations of conflict suggests that ERN amplitudes reflect dynamic changes in response conflict due to the degree of competition between correct and incorrect response options. Specifically, attention to correct response representations heightens response conflict, resulting in a greater need for conflict detection reflected by increases in ERN amplitudes. Future research should continue to examine the evaluative role of the ERN by testing the temporal nature of conflict processing and examining what amount of simultaneous co-occurring conflict or changes in conflict across time can be measured by the ERN. Though there is some variability regarding the relationship of the ERN and subsequent adjustments in behavior, multiple studies provide evidence that conflict detection signified by the ERN is independent of more regulative systems that implement control to change behavior.

Several questions may assist in further understanding the role of the ERN as an index of conflict detection and potential signal for subsequent cognitive control adjustments. First, if the ERN is not directly involved in implementing regulative control processes, what type of information is conveyed by this conflict signal? Is the conflict signal an unspecified cue of greater conflict, or does it also signal information about the magnitude of conflict? These questions may be addressed in part by understanding how neural or cognitive deficits lead to changes in ERN amplitudes and behavior, as observed in clinical populations or among dimensions of cognitive ability.

The relationship between affect, individual differences, and other contextual factors is largely unclear, making it difficult to draw definitive conclusions about the quality of information conveyed by the ERN. The conflict monitoring theory does not explicitly account for these variables, and future research should focus on determining the relationship between conflict and affective states/moods, affective disorders, and individual differences (e.g., differences in internalizing/externalizing personality traits). Also, determining if and when fluctuations in ERN amplitude represent improved behavioral performance may assist in understanding the adaptive nature of the conflict detection system and contribute to a greater understanding of pathological processes. Addressing these questions will aid in our understanding of the limits of the conflict monitoring system and help us to more precisely determine what information the conflict monitoring system contributes to cognitive control.

3. Stimulus-locked ERPs associated with conflict monitoring

Additional support for the role of conflict monitoring in the recruitment of cognitive control comes from stimulus-locked ERP research using paradigms such as the Eriksen Flanker Task (Eriksen and Eriksen, 1974) and the Stroop task (MacLeod, 1991; Stroop, 1935). For example, two functionally-related cognitive control indices of conflict monitoring are the N2 and N450, respectively. Both of these ERPs are thought to reflect ACC-mediated signaling for dynamic modulations of cognitive control (Folstein and Van Petten, 2008). Another commonly investigated ERP component elicited during the Stroop task is the conflict slow potential (conflict SP, also referred to as the negative slow wave [NSW]), which seems related to conflict monitoring and resolution (e.g., Larson et al., 2009b; Perlstein et al., 2006; West, 2003). We review these stimulus-locked ERPs and how they are thought to index response conflict below.

3.1. The N2 as an index of conflict

The N2 elicited during the Flanker task is a negative deflection in the stimulus-locked ERP with a fronto-central scalp distribution that peaks approximately 250–350 ms after stimulus presentation (Folstein and Van Petten, 2008; van Veen and Carter, 2002b; Yeung et al., 2004; see Fig. 2). The role of the N2 in conflict detection is supported by studies demonstrating that N2 amplitude is more negative (i.e., larger) on incongruent trials relative to congruent trials (van Veen and Carter, 2002a,b; Yeung et al., 2004). Research indicates that the N2 is sensitive to the degree of conflict prompted by a given stimulus and is sensitive to the extent to which individuals attend to task-irrelevant (flanker) information compared to task-relevant (target-stimulus) information (Danielmeier et al., 2009; Ullsperger et al., 2005; Yeung and Cohen, 2006; Yeung et al., 2007). Specifically, N2 amplitudes are enhanced with increased proximity of flankers (Danielmeier et al., 2009) and increased incongruity (Forster et al., 2011) but remain unaffected when the brightness of the target stimulus is manipulated (Yeung et al., 2007). Moreover, larger N2 amplitudes are related to longer RTs for correct trials (Beste, 2008; Yeung et al., 2004; Yeung and Nieuwenhuis, 2009), although some studies show this positive correlation only for incongruent trials (e.g., Clayson and Larson, 2011a), suggesting that the degree of conflict as indexed by N2 amplitude is associated with subsequent behavioral performance. The ACC has been in implicated

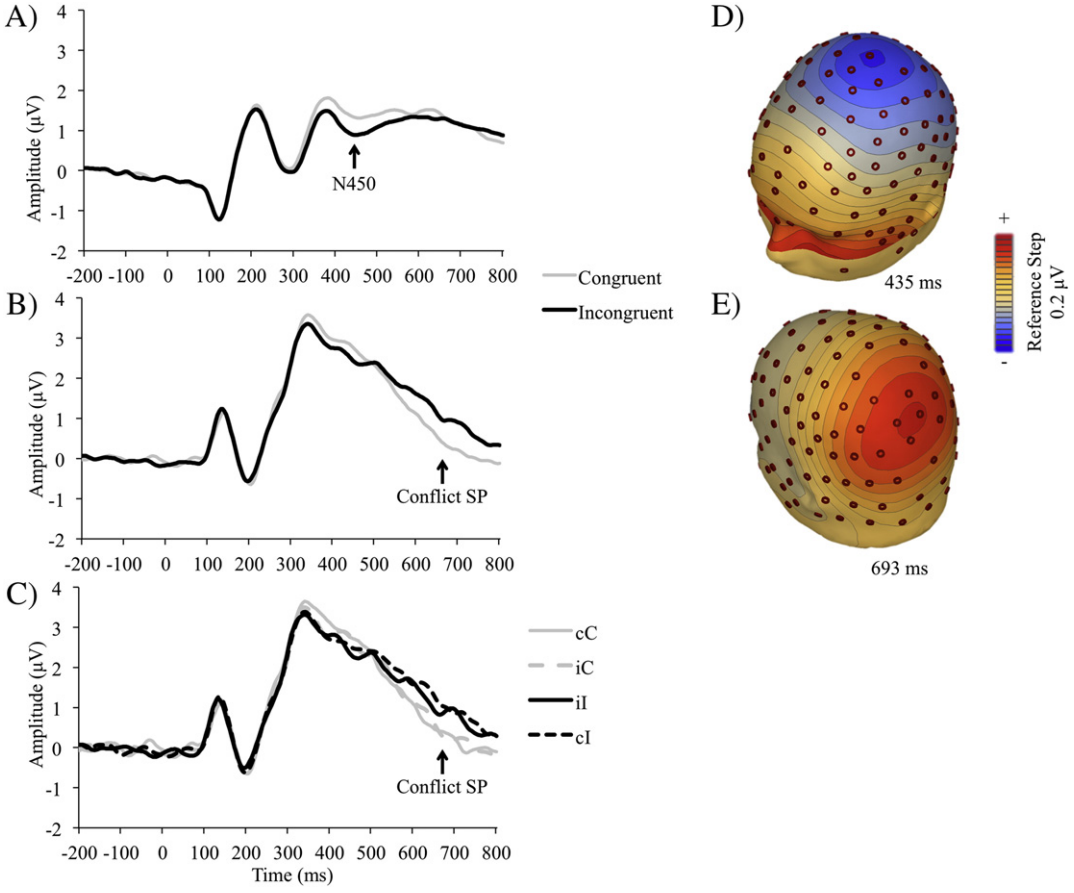

Fig. 2. A) Grand averaged stimulus-locked ERP waveforms from healthy participants on the color-naming condition of a traditional Stroop task averaged across fronto-central electrode locations for the N450. B) Grand averaged stimulus locked waveforms for the conflict slow potential (conflict SP) averaged across centro-parietal electrode locations. C) Grand averaged stimulus-locked ERP waveforms averaged across fronto-central electrode locations for the conflict SP for congruent trials preceded by congruent trials (cC), congruent trials preceded by incongruent trials (iC), incongruent trials preceded by incongruent trials (iI), and incongruent trials preceded by congruent trials (cI). D) Voltage map for stimulus-locked difference activity (incongruent minus congruent) for the N450. E) Voltage map for stimulus-locked difference activity (incongruent minus congruent) for the conflict SP.

in N2 generation based on findings using source localization (Ladouceur et al., 2007; van Veen and Carter, 2002a; Yeung et al., 2004), fMRI (Carter et al., 1998), and intracranial approaches (Wang et al., 2005), suggesting that the N2 is an index of ACC-mediated conflict monitoring.

As we have mentioned in previous studies (see Clayson and Larson, 2013), an N2 is elicited during various tasks, such as the Go/No-Go task, stop signal task, oddball paradigm, or sequential matching task to name a few (and not to mention the numerous variations of each of these tasks). Depending on the paradigm, the N2 component seems to reflect a host of different processes such as response inhibition, target probability, perceptual novelty, and mismatch (see Folstein and Van Petten, 2008, for review). As the primary purpose of this paper is to review conflict-monitoring ERPs and their relationship to the conflict monitoring model of cognitive control, we chose to focus on the N2 elicited during the Flanker task rather than the N2 elicited using other paradigms. A review of these other components and their potential meaning for the conflict monitoring model is outside the scope of this review and, indeed, remains a matter of considerable debate and interest (e.g., Randall and Smith, 2011; Smith, 2011; Smith et al., 2007, 2010). It is our opinion that "not all N2s are created equally", meaning that not all N2 ERP components necessarily index conflict monitoring processing. Thus, we recommend caution when attempting to compare N2 findings between paradigms.

3.1.1. Does the N2 index conflict or error likelihood?

Findings regarding the N2 have been influential in supporting the conflict monitoring theory. One model based on reinforcement learning that stands in contrast to the conflict monitoring theory suggests that the ACC uses dopaminergic signals to generate predictions of error likelihood (Brown and Braver, 2005). Yeung and Nieuwenhuis (2009) modeled contradictory predictions of the conflict monitoring and error likelihood accounts. According to the conflict monitoring model, ACC activity should be greatest for flanker trials with the longest RTs and lowest for flanker trials with the shortest RTs. The trials with the longest RTs should have lower error rates compared to trials with the shortest RTs. This conclusion of lower error rates for long-RT trials compared to short-RT trials is based on the notion that increased time attentively processing the task-relevant and task-irrelevant information should be associated with the greater conflict (i.e., greater ACC activation) but also lead to more accurate responses due to increased attention to the target. They concluded that incorrect responses tend to be the fastest responses due to insufficient time processing the trial information, which should be associated with reduced N2 amplitudes and increased error rates. However, the error-likelihood model predicts that higher error likelihood should be related to increased ACC activity, which in this case should be the trials with the shortest RTs. The empirical data were consistent with the conflict-monitoring model — N2 amplitude was enhanced on trials with long RTs compared to trials with short RTs, putatively reflecting greater ACC activation associated with conflict processing. These authors concluded that putative ACC activity reflected the current level of conflict rather than the retrospective coding of past performance, as the error likelihood model would predict.

3.2. The N450 and conflict SP

The N450, occasionally referred to as the medial frontal negativity (MFN, Chen et al., 2011; West, 2003; West and Alain, 2000; West and Bailey, 2012; West et al., 2012), is another stimulus-locked component that peaks approximately 450 ms after stimulus presentation, possesses a similar fronto-central scalp distribution as the N2, and is more negative following incongruent trials than following congruent trials, indicating a role in conflict detection (see Fig. 2 for example; Liotti et al., 2000; Perlstein et al., 2006; West, 2003; West and Alain, 1999, 2000; West et al., 2005). The N450 represents an index of conflict that is elicited during the Stroop task and likely represents activity of neural generators localized in the ACC (Hanslmayr et al., 2008; Liotti et al., 2000; West, 2003; West et al., 2004). Support for the role of the N450 in conflict monitoring comes from studies showing enhanced amplitude with increased incongruity (Chouiter et al., 2014) or increased interference effect (West and Alain, 2000).

In the Stroop task the conflict SP is a sustained centro-parietal positivity that follows the N450 beginning approximately 500 ms after stimulus presentation (Fig. 2; Bechara, 2005; Liotti et al., 2000). The conflict SP is more positive on incongruent trials than on congruent trials and likely reflects a signal for increased recruitment of cognitive control resources and subsequent compensatory adjustments for accurate task completion (Larson et al., 2009b; West and Alain, 1999, 2000). On incongruent trials more positive conflict SP amplitude has been associated with increased response times and accuracy providing support for the notion that the conflict SP reflects conflict resolution or possibly response selection (West et al., 2005). Source localization studies indicate that the conflict SP is likely generated from sources in the lateral frontal and posterior cortices (Hanslmayr et al., 2008; West, 2003).

Unlike the N2 there is little research on the N450 and the conflict SP as indices of conflict monitoring. One area, however, that is informative about the role of conflict in the N450 and conflict SP is research on conflict adaptation. We discuss the N450 and conflict SP and how these are influenced by previous-trial conflict below.

3.3. Evidence of conflict adjustment from studies of conflict adaptation

According to the conflict monitoring theory, detection of high conflict on incongruent trials leads to the recruitment of cognitive resources to enhance subsequent performance (Botvinick et al., 2001, 2004; Carter and van Veen, 2007). This model is supported by findings that the ACC is involved in evaluative processes and is activated by conflict (Botvinick et al., 1999; Carter et al., 1998) that in turn signals the recruitment of additional cognitive resources to reduce conflict activation on the subsequent trial (Botvinick et al., 2001; Cohen et al., 2000; Kerns et al., 2004). Two areas recruited by the ACC to improve performance are the dlPFC (Botvinick et al., 2001; Durston et al., 2003; Egner and Hirsch, 2005a,b; Kerns et al., 2004) and the vlPFC (Egner, 2011). The dlPFC minimizes conflict by biasing posterior systems, such as the parietal cortex (Corbetta and Shulman, 2002; Desimone and Duncan, 1995) that consequently reduces conflict on the following trial (Rainer et al., 1998) and enhances attentional focus (Egner and Hirsch, 2005a). The enhanced attentional focus filters out distracting, task-irrelevant features, such as the flankers in the Eriksen Flanker Task or the printed word in the Stroop color–word task. In short, the conflict monitoring theory implicates brain regions such as the ACC, as the conflict evaluator, and dlPFC, as the cognitive control implementer, in the strategic deployment of cognitive control following conflict. This link between the detection of conflict in the ACC and the subsequent enhancement of cognitive control by the dlPFC is known as the conflict-control loop (Carter and van Veen, 2007).

Behavioral evidence of this trial-by-trial recruitment of cognitive control in response to conflict has been shown in the Flanker task (e.g., Botvinick et al., 1999; Gratton et al., 1992; Ullsperger et al., 2005), Stroop color–word task (e.g., Kerns et al., 2004; Larson et al., 2009b), and spatial Stroop task (Verbruggen et al., 2005). Conflict adaptation effects have also been shown in numerous N2 ERP studies using the Flanker task (Clawson et al., 2013; Clayson et al., 2011; Clayson and Larson, 2011a,b, 2012, 2013; Forster et al., 2011; Freitas et al., 2009; Kamijo and Takeda, 2013; Larson et al., 2013a, 2012b, 2012c; Rustamov et al., 2014; Sozda et al., 2011). Specifically, incongruent (high-conflict) trials preceded by other incongruent trials (iI trials) are related to faster RTs, reduced error rates, and attenuated N2 amplitudes compared to incongruent trials preceded by congruent (low-conflict) trials (cI trials), putatively reflecting the enhancement of cognitive control following high conflict. For congruent trials preceded by incongruent trials (iC), individuals tend to respond more slowly and less accurately than for congruent trials preceded by congruent trials (cC)

due to switching between congruencies and strategies (see Egner, 2007, for review). N2 amplitudes tend to be larger (i.e., more negative) for cC trials in comparison to iC trials due to reduced control following low conflict and increased processing of task-irrelevant (flanker) information (e.g., Clayson and Larson, 2011a,b, 2013; Larson et al., 2013a); however, larger N2 amplitude on cC trials compared to iC trials is subtle and not always present (e.g., Clawson et al., 2013; Clayson and Larson, 2012; Larson et al., 2012c), likely due to the need for large samples to detect the subtle differences between iC and cC trials. Recent N2 empirical findings also indicate that N2 conflict adaptation effects represent psychometrically-dependable measures as indicated by reliable internal consistency when at least 30 trials were retained for averaging and test–retest stability over a two week period (Clayson and Larson, 2013). These findings indicate that the N2 conflict adaptation effects are consistently observed across numerous studies and represent a dependable, stable measure of conflict adaptation effects, thus providing a promising approach to the study of trial-by-trial adaptations of cognitive control in response to conflict.

3.3.1. N2 conflict adaptation support for the conflict monitoring theory

N2 studies have been influential in examining predictions of the conflict monitoring theory of cognitive control. The conflict monitoring theory predicts that the ACC should be activated in response to high conflict trials leading to the enhancement of cognitive control to reduce subsequent conflict activation (e.g., Corbetta and Shulman, 2002). Not only have incongruent-trial N2 amplitudes been shown to be reduced following another incongruent trial (as noted above), but N2 amplitudes also decrease across up to four consecutive incongruent trials, suggesting that the N2 putatively reflects reduced ACC conflict activation following enhancement of control processes (Clayson and Larson, 2011a; see Durston et al., 2003, for similar fMRI findings). During a modified Flanker task with varying levels of stimulus conflict, higher levels of previous-trial conflict were associated with lower current-trial N2 amplitudes indicating that the recruitment of control is sensitive to the degree of conflict on the preceding trial (Forster et al., 2011). However, this effect was only observed in a subset of participants who showed larger cI compared to iI N2 amplitude; thus in support of the conflict monitoring model the extent to which control was implemented was contingent on the level of conflict on the previous trial in those participants that showed reliable N2 conflict adaptation effects. In these instances the N2 has been used as a direct index of ACC-mediated conflict activation in accordance with predictions of the conflict monitoring theory (Clayson and Larson, 2011a; Yeung et al., 2004; Yeung and Cohen, 2006).

The N2 has also been used as an indirect index of the current level of adaptation of cognitive control present in a trial. Error trials were preceded by incremental reductions of N2 amplitude across five preceding trials, possibly indicating that error commission may be the result of trial-by-trial reductions of cognitive control (Eichele et al., 2010). Errors most frequently occurred to incompatible trials and were frequently preceded by a high number of congruent trials (Eichele et al., 2010). A follow-up study by Steinhauser et al. (2012) observed that these trial-by-trial reductions of N2 amplitude could be adequately predicted using the original connectionist model of conflict adaptation proposed by Botvinick et al. (2001). Steinhauser et al. (2012) in essence concluded that when adapting to consecutive congruent trials a participant may adopt the strategy to utilize information from flanker stimuli to improve performance, as the target-stimulus and flankers queue identical responses (see also Clayson and Larson, 2011a; Yeung and Cohen, 2006; Yeung et al., 2007). However, the adaptation of this strategy and consequent decrease in control are detrimental when an incongruent trial is presented, as a different response strategy is requisite. Taken together, the N2 represents an empirical marker of both a control mechanism to handle conflict and a reflection of the level of cognitive control implemented during the Flanker task.

3.3.2. Is conflict adaptation really just priming? Evidence from the N2

An alternative explanation of conflict adaptation effects suggests that conflict adaptation effects are primarily the result of the facilitative effects of repetition priming (Logan et al., 2007; Mayr et al., 2003; Verbruggen et al., 2005). For example, Mayr et al. (2003) showed that behavioral conflict adaptation effects were present only for trial pairs that included stimulus–response (S–R) repetitions (i.e., two consecutive stimuli requiring identical responses) but not trial pairs omitting S–R repetitions. Thus, they concluded that conflict adaptation effects were due to S–R repetitions, which bypass the need for increased cognitive control. Nieuwenhuis et al. (2006) did not show reliable conflict-sensitive control modulations across five different behavioral experiments with varying degrees of conflict when S–R repetitions were not present. After the removal of S–R repetitions in a conventional Flanker task, N2 amplitudes were sensitive to conflict adaptation effects as indicated by reduced N2 amplitude to iI compared to cI trials (Clayson and Larson, 2011a,b) as well as a linear reduction across three consecutive incongruent trials (Clayson and Larson, 2011a), suggesting that S–R repetitions do not fully account for the post-conflict recruitment of cognitive control. When examining the effects of S–R repetitions on N2 amplitude, N2 amplitudes were more negative overall for data omitting S–R repetitions compared to trial pairs exclusively examining response repetitions (Clayson and Larson, 2011b). These findings indicate that the post-conflict recruitment of cognitive control is not accounted for by the facilitative effects of repetition priming as indexed by the N2; although, it should be noted that S–R repetitions are a special case of feature integration effects,[2] which were not fully excluded in the abovementioned flanker studies.

3.3.3. N450 and conflict SP in understanding conflict adaptation

With regard to the Stroop task, the conflict SP is sensitive to conflict adaptation effects in the color–word Stroop task (Larson et al., 2012a, 2011a, 2009a,b), an auditory Stroop task (Donohue et al., 2012), and a counting Stroop task (West and Bailey, 2012). The N450 has been shown to be sensitive to conflict adaptation effects in the counting Stroop task (West and Bailey, 2012) but not the color–word Stroop task (Larson et al., 2011a, 2009b) or the auditory Stroop task (Donohue et al., 2012). Considering that both the conflict N2 and Stroop N450 putatively reflect ACC conflict-monitoring processes, it remains unclear as to why the conflict N2 seems consistently sensitive to the post-conflict recruitment of cognitive control while the Stroop N450 exhibits nonsignificant effects of previous-trial conflict in two of three kinds of Stroop tasks. Therefore, the nature and functional significance of the similarities and differences between the conflict N2 and Stroop N450 in ACC-mediated conflict monitoring remain to be explored. Indeed, we see within-subjects studies of these two conflict-related components as one area for future research. Clarifying the relationship between these two components will help elucidate the task demands under which conflict activation recruits cognitive control.

3.3.4. The influence of individual differences and affect on ERP indices of conflict adaptation

On a group level, the investigation of ERP conflict adaptation effects may elucidate nuanced differences in cognitive control functioning. For

[2] Another possible interpretation of conflict adaptation comes from the feature integration account (Hommel et al., 2004; Notebaert et al., 2001; Wendt et al., 2006). This account suggests that a response and a stimulus co-occurring in time are integrated into an "event file." Any exact repetition of a stimulus or complete alternation of stimulus information (e.g., in a color–word Stroop, neither the color nor word from the previous trial matches the current trial) should result in faster RTs (Hommel, 1998; Notebaert et al., 2001), because the information is bound together in the event file so that by activating the stimulus, for example, the other response is easily activated. A partial repetition where only one feature from the event file repeats should result in slow RTs, because of encoding interference. However, the conflict monitoring ERP research using the Stroop has done little to examine the feature integration account as after removing repetitions there are too few trials left for analysis due to a low signal-to-noise ratio.

example, in an investigation of sex differences in conflict monitoring and adaptation processes, males showed significantly larger (i.e., more negative) incongruent-trial N2 amplitude compared to females (Clayson et al., 2011). When examining conflict adaptation, however, males and females did not differ on behavioral or N2 conflict adaptation effects. These findings mirror ERN findings noted above and indicate that females may show more efficient recruitment of cognitive control following conflict, as less putative ACC activation was required to implement similar cognitive resources. Furthermore children (mean age of 9.7 years) and young adults (mean age of 22.3 years) show similar conflict SP conflict adaptation effects (Larson et al., 2012a). In another study using the Flanker paradigm youth with a mean age of 14 similarly showed intact N2 conflict adaptation effects (Larson et al., 2012c). Thus, children seem to show intact reactive control adjustments to conflict as indicated by findings from the Stroop and Flanker tasks indicating that cognitive control mechanisms effectively regulate conflict adaptation processes in children. Although no published studies to date have used ERPs to examine conflict adaptation in older adults recent preliminary findings from our lab suggest similar N2 conflict adaptation processes for older adults (mean age of 69) and younger adults (mean age of 21) during a Flanker task (Larson et al., 2013c). Taken together ERP conflict adaptation processes seem reliably intact across the lifespan.

In addition to the study of development, ERP conflict adaptation research provides a method for examining cognitive control in neurologic and psychiatric populations. For example, individuals with mild traumatic brain injury show intact current-trial conflict monitoring processes as indicated by the N450, while demonstrating impaired conflict regulation processes as measured by the conflict SP (Larson et al., 2011a). These findings are particularly important as research on mild TBI outcome indicates inconsistent behavioral (e.g., Hartikainen et al., 2010; Pontifex et al., 2009) and neuropsychological findings (e.g., Binder et al., 1997; Frencham et al., 2005; Pertab et al., 2009; Vanderploeg et al., 2007). In concert with impaired conflict regulation process in the conflict SP shown in severe TBI (Larson et al., 2009a), nonsignificant conflict SP conflict adaptation effects may index control deficits across the range of TBI severities. However, research examining this possibility across a range of TBI severity is requisite to support this notion. These findings have implications for understanding functional impairments in TBI, as impairments associated with the recruitment of cognitive control may underlie cognitive rigidity and perseverative errors in TBI. Indeed, as a result of these and other such findings, treatment approaches have begun to emphasize cognitive control deficits in rehabilitation (e.g., Ownsworth et al., 2006, 2010).

To examine the dimensional relationship between the post-conflict recruitment of cognitive control and individual differences, the magnitude of conflict adaptation effects and the mean conflict adaptation scores prove helpful. The magnitude of conflict adaptation effects refers to the difference between iI and cI trials which reflects the influence of the enhancement of cognitive control following a high-conflict trial compared to a low-conflict trial (e.g., Clayson and Larson, 2012; Larson et al., 2012c). Previous research also uses a common formula, (cI − cC) − (iI − iC), to assess mean conflict adaptation scores as a measure of the efficiency of cognitive control processes (Nieuwenhuis et al., 2006; van Steenbergen et al., 2009, 2010). The inverse of these metrics is commonly employed to make the data comparable to RT and error rate mean conflict adaptation scores, such that larger scores are associated with larger adjustments of control.

The use of these indices to characterize the pattern of fluctuations in cognitive control has been influential in highlighting specific individual differences. For example, after the removal of S–R repetitions, a larger magnitude of N2 conflict adaptation effects and larger mean N2 conflict adaptation scores were related to higher scores on neuropsychological measures of attention and executive functioning in the absence of a relationship between either RTs or error rates and other measures of neuropsychological functioning (Clayson and Larson, 2012). In this case a smaller magnitude of N2 conflict adaptation scores and reduced mean N2 conflict adaptation effects may represent more efficient post-conflict recruitment of cognitive control in individuals with higher scores on measures of attention and executive functioning, as these individuals maintained a similar level of performance to those with low scores on these measures. These results were also observed in the absence of a significant relationship between simple, current-trial N2 amplitude and indices of cognitive functioning in the same sample of participants (Larson and Clayson, 2011), indicating that the relationship between cognitive functioning and changes in N2 amplitude is specific to post-conflict modulation of ACC-mediated conflict monitoring reflected in the N2. These findings support the notion that larger conflict adaptation effects are associated with improved cognitive control functioning.

The examination of the magnitude of conflict adaptation effects and mean conflict adaptation scores has also been influential in examining the dimensional role of symptom severity in neurologic and psychiatric populations. Youth with autism spectrum disorders showed nonsignificant N2 conflict adaptation processes, and higher scores on the Autism Diagnostic Observation Schedules-Generic (Lord et al., 2000) were related to lower magnitudes of N2 conflict adaptation effects, suggesting that increased autism severity may be related to worse cognitive control functioning (Larson et al., 2012c). Although one study observed that individuals with major depressive disorder showed intact N2 conflict adaptation effects, higher scores on the Beck Depression Inventory, 2nd Edition (BDI-II; Beck et al., 1996) were associated with smaller mean N2 conflict adaptation effects, possibly indicating that individuals with higher depression symptom severity show diminished recruitment of cognitive control following conflict (Clawson et al., 2013). Impaired N2 conflict adaptation effects were also observed in individuals with generalized anxiety disorder (Larson et al., 2013a). Increased depression symptom severity, measured by the BDI-II, and increased trait anxiety symptom severity, measured by the State-Trait Anxiety Inventory (Spielberger et al., 1983), were related to lower mean N2 conflict adaptation scores. These studies highlight the importance of examining conflict adaptation effects in individuals with psychopathology in a dimensional fashion. In these studies increased symptom severity, whether it was autism severity, anxiety symptom severity or depressive symptom severity, was related to worse cognitive control functioning indexed by the magnitude of conflict adaptation effects or mean conflict adaptation scores.

Investigations of conflict adaptation in psychiatric and neurologic populations hold specific promise for targeting deficits with treatment approaches such as those that emphasize ameliorating cognitive control impairments. For example, treatment approaches have begun to emphasize cognitive control deficits in rehabilitation in TBI (e.g., Ownsworth et al., 2006, 2010) and in major depressive disorder (see Siegle et al., 2007 for review). These findings also support the interpretation of larger conflict adaptation effects as a measure of cognitive flexibility and the use of post-conflict recruitment of control as an indication of frontally-mediated cognitive control dysfunction. In sum, conflict adaptation research provides a promising avenue for investigating cognitive control functioning in psychiatric, neurologic, and developmental populations with future research geared toward treatment of cognitive control deficits.

4. Comparing conflict-related ERPs

4.1. Comparing the N2 and N450 stimulus-locked ERPs

The N2 and N450 belong to the conflict-monitoring family of ERPs and seem to represent similar cognitive control signals related to conflict monitoring (see Folstein and Van Petten, 2008). However, nuanced differences between the two components may shed light on the distinction between the functional significance of the N2 and N450. Comparing tasks with various congruent/incongruent trial probability ratios may be helpful in distinguishing these two components. Previous research

indicates that conflict is greatest when incongruent trials are rare compared to when they are frequent (e.g., Botvinick et al., 2004; Braver et al., 2001; Gratton et al., 1992). The N450 seems to generally show an increased congruency effect when incongruent trials are rare compared to when they are frequent (Lansbergen et al., 2007; West and Alain, 2000). Notably, however, the N2 measured frontally (at Fz) showed a larger congruency effect during a congruent probable condition compared to an incongruent probable condition, and the N2 measured at Cz was insensitive to incongruent-trial probability (Bartholow et al., 2005).

A recent study by Tillman and Wiens (2011) used a within-subjects design to assess the sensitivity of the N2 and N450 to paradigmatic manipulations of congruent/incongruent trial probability. The Stroop N450 showed the expected larger congruency effect in the congruent-probable task compared to the incongruent-probable task, whereas larger congruency effects were observed for the incongruent-probable task compared to the congruent-probable task for the N2. They proposed that the N2 represents processes associated with response inhibition or the biasing of attentional focus on target-stimulus information, rather than a role as a conflict signal. Although speculative (decompositions of significant interactions were not presented in the Tillman and Wiens paper), upon closer examination of the findings it appears that N2 amplitude was numerically larger (i.e., more negative) for congruent trials in the congruent-probable condition compared to the incongruent-probable condition. As mentioned previously, N2 amplitude is sensitive not only to the degree of conflict for a given stimulus but also to the extent to which task-irrelevant information is processed. This notion is further supported by findings that N2 amplitudes increase over consecutive congruent trials (Clayson and Larson, 2011a), as cognitive control subtly relaxes over consecutive congruent trials and more flanker information is processed. It seems more accurate to suggest that decreased congruency effects in the N2 in the congruent-probable condition compared to the incongruent probable condition are likely the result of enhanced N2 amplitude to congruent trials rather than significant decreases in N2 amplitude to incongruent trials. The difference in N2 congruency effects in the two conditions is likely the result of increased processing of flanker stimuli on congruent trials during the congruent-probable condition, as opposed to a decrease in conflict detection processes as suggested by Tillman and Wiens. Thus contrary to their conclusions, their findings do not impugn the role of the N2 in conflict detection as outlined by the conflict monitoring model.

Another notable N2/N450 difference is that the N2 is sensitive to conflict adaptation processes (e.g., Clayson and Larson, 2011a,b; Forster et al., 2011), whereas the N450 is not generally altered by the post-conflict recruitment of control (Larson et al., 2011a, 2009b; see Conflict Adaptation section 3.3). One simple, potential explanation may be differences in sample sizes between Stroop and Flanker studies. For example, studies of conflict adaptation using the color–word Stroop task used overall sample sizes of 36 adults (Larson et al., 2009b) or 36 healthy adults (65 when including individuals with mild TBI; Larson et al., 2011a). Conflict adaptation studies using the Flanker task generally have much larger sample sizes such as 181 adults (Clayson and Larson, 2011a), 89 adults (Larson et al., 2012c), and 49 adults (Forster et al., 2011); however, this is not always the case as some studies have utilized smaller samples such as 36 youth (Larson et al., 2012c) and 19 adults (Kamijo and Takeda, 2013). Before drawing definite conclusions that the N450 is not sensitive to conflict adaptation effects it may be helpful to examine N450 conflict adaptation in larger samples comparable to some of the flanker conflict adaptation studies.

In short, the N450 and N2 seem to reflect a similar conflict detection signal; however, there is some evidence against this conclusion in that the N2 is also sensitive to the degree to which flanker information is processed. It is very rare that the N450 and N2 are compared within the same participants and justification for the functional relationship between the two components is largely theoretical and somewhat circumstantial. We recommend further research to examine the N450 in a large sample comparable to the N2 studies mentioned above to assess whether the N450 is sensitive to conflict adaptation effects, which would provide support for the N450 as a conflict signal that functions in a manner consistent with predictions of the conflict monitoring model of cognitive control. In addition, it would be helpful to compare the trial-by-trial recruitment of cognitive control indexed by the N2 and N450 within-subjects to clarify whether the magnitude of conflict adaptation effects is comparable and the potential for comparing and contrasting conflict adaptation findings from the Flanker and Stroop paradigms. As more studies examine the role of the same individual differences in N2 and N450 processing, the functional similarity between the N2 and N450 may be clarified or questioned.

4.2. Comparing the N2 and ERN ERPs

As noted above, according to the conflict monitoring model (Carter and van Veen, 2007; Yeung et al., 2004; Yeung and Cohen, 2006), the ERN represents post-response conflict generated between a competing mental representation of an erroneous response and a subsequent corrective response prompted by the target stimulus, whereas the N2 indexes pre-response conflict generated between the activation of response options queued by the target stimulus and flanker stimuli. Although both the ERN and N2 putatively reflect a conflict signal generated by the ACC (Yeung et al., 2004), the conflict monitoring model makes separable predictions about these two components: the ERN is contingent upon continued processing of the target stimulus following error commission and the N2 is closely related to the processing of the task-irrelevant (flanker) stimuli (see Yeung and Cohen, 2006). These separate predictions are consistent with empirical studies showing distinctions between ERN and N2 amplitude, such as an attenuated ERN and enhanced N2 in a participant with a left-ACC lesion (Swick and Turken, 2002), an attenuated ERN and normal N2 after the consumption of alcohol (Ridderinkhof et al., 2002), an attenuated ERN and an enhanced N2 with increased proximity of flanker stimuli to the target stimulus (Danielmeier et al., 2009), an enhanced ERN and normal N2 during a flanker trial with a bright target stimulus compared to dim target stimulus (Yeung et al., 2007), an increased ERN and reduced N2 following enhancements of post-conflict cognitive control (Larson et al., 2012b), a reduced ERN and normal N2 in children reared in deprived environments (Loman et al., 2013), and a decreased ERN and enhanced N2 following sleep deprivation (Tsai et al., 2005). These studies simply provide evidence that the ERN and N2 represent distinct, separable processes. The ERN is susceptible to alterations of attention to the target stimulus following error commission, whereas pre-response levels of attention to flanker stimuli alter the N2. Taken together, the ERN and N2 represent distinct, ACC-mediated conflict signals associated with cognitive control that can show dissociable differences associated with ongoing, post-response stimulus processing and pre-response flanker processing, respectively.

The dissociation between the ERN and N2 provides a framework for understanding conflict processing not only in studies of individual differences but also in psychiatric and neurologic populations — that is, specifically which aspect of conflict processing is compromised or exaggerated. For example, a recent meta-analysis indicated a small-to-medium relationship between increased trait anxiety and increased ERN amplitude; however, the Flanker N2 was not examined and seems to be largely ignored in ERP studies of trait anxiety (Moser et al., 2013). In studies of generalized anxiety disorder (GAD), individuals with GAD showed enhanced ERN amplitude (Weinberg et al., 2012, 2010; Xiao et al., 2011) and reduced N2 amplitude (Larson et al., 2013a). A synthesis of these findings indicates that individuals with GAD show exaggerated processing of the correct target-stimulus following error commission and compromised processing of pre-response conflict associated with flanker processing. A recent account of the relationship between heightened ERN and increased anxiety may clarify the significance of the

enhanced-target stimulus processing in GAD (Proudfit et al., 2013). To summarize briefly, Proudfit et al. argued that errors signal threat and that differences in ERN amplitude can reflect trait-like differences in early threat sensitivity. They suggested that enhanced ERN in individuals with high trait anxiety indicates a disposition to react more substantially to an uncertain threat. In this context, individuals with GAD may show exaggerated attention to the correct target stimulus following error commission as a result of an overreaction to uncertain threat associated with erroneous responding. This theory needs future testing; however, thinking of the ERN and N2 as dissociable components representing similar but distinct conflict signals will hopefully improve understanding of abnormal activity in psychiatric and neurologic populations.

In summary, the ERN represents an index of post-response conflict that is contingent upon the continued processing of an erroneous response and the subsequent corrective response. The N2 represents pre-response conflict between the activation of multiple response options by the target stimulus and flanker stimuli. The dissociation between these two components is evident in the numerous studies mentioned above. Although the ERN and N2 provide distinct information regarding conflict-monitoring processes, it does not seem common practice to analyze both components when using the Flanker task. In order to understand the paradigmatic and individual differences captured in the ERN and N2, it would be beneficial for studies using the Flanker task to assess both components to provide more information regarding these components. In consideration of their similarities and distinctions, analyzing both the ERN and N2 should provide valuable information regarding cognitive control processes for any study. Thus, we recommend that moving forward studies examine the ERN and N2 in concert rather than separately. This seems particularly important given the burgeoning interest in assessing cognitive control processes related to conflict monitoring in psychiatric, neurologic, and developmental populations.

5. Summary and conclusions

Summarizing this large body of information provides interesting insights into the role of ERPs in our understanding of conflict-related processing. Returning to the original questions summarized from the early Botvinick et al. (2001) and Yeung et al. (2004, 2006) papers, current ERP evidence provides some interesting topics for discussion. First, the ERN and N2 ERPs provide some index of conflict-related processing. However, the precise functional significance of the N450 and conflict SP ERPs remains unclear. Future research utilizing within-subjects designs directly comparing these ERPs will help to elucidate their role in conflict processing. Second, the level of conflict in a stimulus or response is detectable via the amplitude of both stimulus- and response-locked ERPs. Research questions examining the magnitude of the conflict response and the temporal sequence of cognitive control adjustments are areas well suited for ERP-related research. One primary question in this regard, however, is the relationship (or general absence thereof) between conflict-related ERPs and behavioral performance. Third, evidence from ERPs and a corpus of additional findings from fMRI studies (Banich, 2009; Niendam et al., 2012; Shackman et al., 2011; Shenhav et al., 2013; Silton et al., 2011; Silton et al., 2010) show that ACC is involved in conflict monitoring and that ACC-mediated monitoring, and specifically conflict monitoring, plays a large role in cognitive control processes. Perhaps the strongest evidence on this front comes from the studies summarized above regarding conflict adaptation. Clearly, the magnitude of conflict on previous trials influences performance on subsequent trials. Conflict-related changes, at least from an ERP perspective, do not appear to be accounted for solely by priming processes. Conflict adaptation differs according to individual differences and affective functioning, suggesting that there is more to conflict adaptation than simple conflict detection and control adjustments. Nevertheless, existing studies indicate that context biases conflict-monitoring processes and that the strength of conflict-related processes is identifiable through stimulus-locked ERPs. Fourth, there is a paucity of information about what happens after conflict is detected. Only the conflict SP and, to some degree, the P3 (not reviewed here, but see Clayson and Larson, 2011a,b) have been examined as clear indices of post-conflict processing of stimuli and subsequent adjustments in control. Thus, an area of future research is on how post-conflict processes are manifest, how they vary as a function of conflict magnitude, and how they influence manifestations of regulative control. Finally, the relationships between behavioral performance, affect, and indices of conflict monitoring are extremely unclear. Studies manipulating both level of conflict and affect are needed. We are hopeful that this review provides the impetus for better understanding of conflict-related processes and that, as we noted in the introductory section, our increased understanding of conflict processes can begin to transition into clinical disorders such as TBI or autism where disrupted cognitive control and conflict monitoring play such a large role in maladaptive daily functioning.

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