EXPLORING ASSOCIATIONS BETWEEN PROACTIVE AND REACTIVE CONTROL

by

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ABSTRACT

Cognitive control is the act of regulating, coordinating, and sequencing mental processes in accordance with internally maintained behavioral goals (Braver, 2012; Norman & Shallice, 1986). The Dual Mechanisms of Control (DMC) theory argues that variations in cognitive control are driven by two distinct operating modes, proactive control and reactive control (Braver et al., 2007). Proactive control is defined as an anticipatory and effortful attentional strategy that actively sustains task-relevant information before the occurrence of a cognitively demanding event (Miller & Cohen, 2001). In contrast, reactive control is an automatic process that is passively maintained and relies upon high-conflict, or trigger, events to reactivate task-relevant information after the occurrence of a cognitively demanding event (Jacoby, Kelley, & McElree, 1999). Traditional models of cognitive control focus on reactive control initiating proactive control (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999). Yet, recent research suggests the possibility of shifting to a predominantly proactive strategy with less reliance on reactive processing (Braver, Paxton, Locke, & Barch, 2009; Schmid, Kleiman, Amodio, 2015). However, little work has analyzed a direct relation between continuously sustained proactive control and reduced input from reactive control. In addition, affective variables might impact the ability to shift between proactive and reactive modes of control (Braver, Gray, & Burgess, 2007). Individuals high in trait levels of worry exhibit heightened reactive control and reduced proactive control compared to controls (Moser, Moran, Schroder, Donnellan, & Yeung, 2013). In the current study, participants performed a cognitively-demanding task while neural correlates of proactive and reactive control were measured. Self-reported levels of trait worry were also collected. In agreement with a proactive model of cognitive control, the results of this experiment indicated that greater levels of sustained proactive control predicted decreased reactive processing. However, this relation was moderated by trait-worry such that enhanced proactive control only predicted decreased reactive control when levels of trait worry were low.

INTRODUCTION

Throughout life, humans encounter situations that require the organization of thoughts and actions in order to meet shifting environmental demands. Managing these thoughts and actions requires cognitive control, broadly defined as the act of regulating, coordinating, and sequencing mental processes in accordance with internally maintained behavioral goals (Braver, 2012; Norman & Shallice, 1986). Cognitive control is important for a wide range of behaviors that require adaptability, such as overcoming stereotypical associations (Payne, 2005), controlling urges to eat unhealthy foods (Nederkoorn, Houben, Hofmann, Roefs, & Jansen, 2010), and attaining personal goals (Amodio et al., 2004). A core component of cognitive control is its variability across time, across situations, and between people. Understanding this variability is important for understanding how cognitive control becomes engaged, and may help to explain certain emotional processes (Braver, Gray, & Burgess, 2007).

The Dual Mechanisms of Control (DMC) theory offers an explanation for variability in cognitive control within and between people (Braver et al., 2007). This theory argues that variability in cognitive control is driven by two distinct operating modes: proactive control and reactive control. Proactive control is defined as an anticipatory and effortful attentional strategy that actively sustains task-relevant information before the occurrence of a cognitively demanding event (Miller & Cohen, 2001). In contrast, reactive control is an automatic process that is passively maintained and relies on high-conflict, or trigger, events to reactivate task-relevant information after the occurrence of a cognitively demanding event (Jacoby, Kelley, & McElree, 1999). For

example, imagine a film student analyzing a masterpiece by Truffaut with the goal to interpret which nuances are remarkable enough for a thesis subject. She is exerting proactive control when actively seeking out and making note of relevant material while watching the film. This process is advantageous because it allows for goals and behaviors to be continuously adjusted to lead to goal completion (i.e., not missing a crucial moment in the film). When she slips into indifference and forgets to monitor for task-relevant information, reactive control might be triggered when an important scene is cued by dramatic music. Consequently, reactive control produces a greater reliance upon the salience of trigger events. If an event is not salient enough, task-relevant information will not be reactivated and control will not be employed.

Variations within and between people in cognitive control are thought to arise across time and task situations because of divergences in the utilization of proactive and reactive control (Braver, et al., 2007). Divergences in the employment of proactive and reactive control rely, in part, on assumptions of the conflict-monitoring hypothesis, which states that conflict triggers compensatory adjustments in cognitive control in order to reduce subsequent conflict processing (Botvinick, Braver, Barch, Carter, & Cohen, 2001). Conflict-based adjustments are initiated when there is a divergence between higher-level goals and lower-level responses. The DMC theory adds a complementary explanation of the neurological adjustments that result from conflict detection, and explains how settings in the neurological environment can predict conflict sensitivity. Specifically, the DMC theory hypothesizes that reactive control occurs as the result of conflict detection and signals the need for the subsequent recruitment of proactive

control. Proactive control facilitates top-down biasing in preparation for future processing, which in turn decreases subsequent conflict-related activation (Corbetta & Shulman, 2002). The DMC theory fits well with assumptions posited by the conflict monitoring hypothesis as both argue that reactive control is the result of conflict-monitoring; however, the DMC theory further elaborates on a possible means through which conflict-monitoring can result in reduced subsequent conflict processing, specifically, through the engagement in proactive control. Top-down biasing acts to maintain internal representations of task-relevant information; consequently, there is less reliance upon bottom-up sensory information for task guidance (Botvinick et al., 2001). Proactive control thus diminishes conflict sensitivity through biasing information processing systems to rely on internal, rather than external, sources for guidance.

The conflict-monitoring hypothesis presents a model of cognitive control that focuses primarily on reactive mechanisms in that proactive control is posited to engage in reaction to conflict detection (Botvinick et al., 2001). That is, in high-conflict situations, conflict detection signals reactive control to activate top-down attentional resources. According to this theory, cognitive control is therefore argued to be driven by a feedback loop wherein reactive control activates proactive control, and enhanced proactive control results in less subsequent reactive processing. Alternatively, more recent research has proposed that cognitive control could also be driven preemptively by proactive control above and beyond reactive processes (Schmid, Kleiman, & Amodio, 2015). In such a scenario, the continuous sustained exertion of proactive control should diminish reactive processing, and the need for reactive conflict-monitoring processes to guide task

performance (Braver et al., 2007). However, more research is needed to verify whether the sustained activation of proactive control does indeed diminish reactive processing.

Intra-Individual Variation

Intra-individual variation in cognitive control might depend on whether proactive or reactive control is better suited for particular task. A proactive strategy is advantageous in situations where plans and behaviors need to be continuously adjusted (Miller & Cohen, 2001; Miller, Erickson, & Desimone, 1996). Exerting proactive control also helps to promote efficient performance through minimizing interference from distracting information (Miller, Erickson, & Desimone, 1996), such as not missing an important scene during a film even when a cell phone is ringing. The disadvantage of proactive control is that it requires the effortful maintenance of thoughts and actions. Actively maintaining task-relevant information, particularly over extended periods of time, is highly taxing on cognitive resources, leaving substantially reduced cognitive resources available for other goals (Braver et al., 2007). There are also capacity limitations concerning the amount of task-relevant information that can be held in focused attention at any given time (Cowan, 2010; Oberauer, 2002). As a result, in situations where multiple goals are being sought, people might increasingly rely on reactive control (Braver, 2012).

Under reactive control, goal relevant information is automatically activated as needed in a “just in time” manner. Reactive control is advantageous because it does not require effortful maintenance of thoughts and actions, which makes cognitive resources

available for other goals (Braver et al., 2007). For example, if someone knows they need to make a trip to the grocery store at the end of a work day, relying on reactive control to reactivate the goal to get groceries at the end of the day would be preferable over actively maintaining the goal throughout the entire day, which would leave less cognitive resources available for other work-related tasks. Relying on reactive control is thus an optimal strategy in situations where the duration between intention formation and task completion is long, as relying on proactive control would be impractical due to its consumption of cognitive resources. In such situations, reactive control is beneficial because a goal is only transiently activated at the time of intention and reactivated upon encountering a trigger event, such as seeing a grocery list on the car's dashboard. However, reactive control is disadvantageous in that it requires a trigger event salient enough to reactivate goal relevant information. If the trigger event does not occur or is not salient, goal-relevant information might not be reactivated and the probability of goal attainment is diminished.

Ultimately, then, situational demands are expected to guide the extent to which one might select a proactive or reactive control strategy (Braver, 2012). According to the DMC theory, selecting a proactive strategy is plausible and, in fact, beneficial in certain scenarios, although its role in predicting the engagement of reactive control remains understudied. Work targeting whether cognitive control can be driven by the sustained engagement of proactive control could add support for an alternative proactive model of cognitive control that operates above and beyond the engagement of reactive processes.

Reactive Control and the Anterior Cingulate Cortex

Research has tied proactive and reactive control to activity in distinct neurophysiological substrates. For example, reactive control is linked with activity in the anterior cingulate cortex (ACC), a collar that surrounds the frontal part of the corpus callosum. Research has implicated ACC activation in conflict detection processes that initiate reactive control (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Kerns et al., 2004). In a laboratory setting, this effect is detectable when participants are engaging in a Stroop task (Stroop, 1935). For this task, participants see a color word that is either congruent with the ink color in which it is displayed (e.g., the word “blue” in blue ink) or that is incongruent with the ink color in which it is displayed (e.g., the word “blue” in red ink). Participants are asked to name either the color word or the ink color. Activity in the ACC increases during high-conflict (i.e., incongruent) Stroop trials, perhaps signifying the employment of reactive control due to detection of conflict between stimulus-driven response tendencies and higher order goals (Kerns et al., 2004; MacDonald, et al., 2000; Pardo, Pardo, Janer, & Raichle, 1990). Such reactive processing could also lead to enhanced subsequent proactive control. This possibility is supported by findings showing that faster response times are observed on high-conflict Stroop trials immediately preceded by a high-conflict trial, potentially indicating that ACC activation during the initial high-conflict trial signaled for the need for greater subsequent top-down control (Botvinick et al., 1999; Kerns et al., 2004).

Studies have also implicated the ACC in reactive control through work showing ACC activation during other high conflict situations, such as when participants attempt to make a correct choice from among a set of potential distracters. For example, ACC activation is greater during high-conflict trials relative to low-conflict trials on the Eriksen Flanker Task (Erkisen & Shultz, 1979; Larson, Clayson, & Baldwin, 2012). For a flanker task, each trial presents a series of either congruent or incongruent stimuli. For a flanker task using arrows, participants are asked to respond with a key press indicating the direction of the central arrow of a five-arrow array. Trials on which the central arrow is incongruent with the flanking arrows are considered high-conflict trials because of the need to suppress the conflicting information from flanker stimuli to correctly identify the direction of central stimulus. This interpretation of the flanker task is consistent with the suggestion of the conflict-monitoring hypothesis that increased ACC activation during high-conflict trials is due to the detection of conflict between a top-down goal to identify the central target and the bottom-up tendency to simply identify the distracting flanking stimuli. When conflict is detected, the ACC initiates a reactive control process that is thought to signal the need for the employment of proactive control, thus reactivating task-relevant information and initiating compensatory adjustments in behavior in order to enhance performance on subsequent trials (Botvinick et al., 1999; Kerns et al., 2004).

ACC activity is visible in real time using markers of neural processing. The N2 is a stimulus-locked neural marker in electroencephalograph (EEG) recordings that is visible as a negative deflection peaking roughly 200-350ms after stimulus presentation (Nieuwenhuis, Yeung, Wildenberg, & Ridderinkhof, 2003). Research has proposed that

the N2 indexes conflict-related processing (Yeung, Botvinick, & Cohen, 2004), and work showing associations between N2 amplitudes and conflict detection (Yeung et al., 2004) has led to beliefs that the N2 indicates ACC activity. For example, N2 amplitudes are enhanced during high-conflict flanker trials compared to low-conflict trials (Larson et al., 2012; Yeung et al., 2004). Because N2 amplitudes are modulated by the perception of conflict, such that high-conflict stimuli enhance the N2 compared to low-conflict stimuli, the N2 appears to be a valid marker of conflict detection and, thus, reactive processing. Importantly, an increase in this type of stimulus-related conflict processing (i.e., reactive control) is thought to inversely depend on an individual's expectancy of, and preparation for, upcoming conflict (i.e. proactive control). This notion is supported by research showing that an initial high-conflict flanker trial will recruit top-down control which, in turn, dampens conflict processing on a subsequent high-conflict trial; this shift in processing strategies is visible at the level of neural processing as diminished ACC activation (Botvinick et al., 1999), and decreased N2 amplitudes (Larson et al., 2012), on a subsequent high-conflict trial.

Proactive Control and the Prefrontal Cortex

In contrast to the grounding of reactive control in ACC activity, proactive control is thought to be associated with activity in the prefrontal cortex (PFC), an area of the brain within the frontal lobes responsible for maintaining and organizing task-relevant information (Davidson, 1992; Harmon-Jones, 2003; Schmid et al., 2015). The PFC is highly developed in organisms known for their diverse and flexible behavioral repertoire

and is in a suitable position for coordinating a wide variety of neurological processes, in part due to dense interconnections between the PFC and other neural areas (Miller & Cohen, 2001). A function of the PFC is to facilitate the representation and active maintenance of context information, a key aspect of proactive control (Braver et al., 2007; Fletcher, Shallice, & Dolan, 1998; MacDonald et al., 2000). *Context* is defined as internally represented task-relevant information that can alter cognitive processing related to task performance (Cohen & Servan-Schreiber, 1992). Context representations are short-lived mental representations of tasks that are flexible enough to bias action systems, perception, memory, attention, and emotion (Braver et al., 2007; MacDonald et al., 2000). Because context representations are internal, they can continuously influence processing without relying on input from external sources such as trigger events. The PFC uses context representations to protect against interference when conflicting task information is encountered, such as when target information is embedded within distracting stimuli (MacDonald et al., 2000).

Activity in the PFC has been linked to the active maintenance of context information (i.e. proactive control) in work using the AX-Continuous Performance Task (AX-CPT) paradigm (Braver & Cohen, 2000). This task is a delayed response paradigm where task-relevant information must be actively maintained over a retention interval in order to bias processing to a subsequent probe item. In target trials, participants must make one response for the letter X when it is preceded by a contextual cue, the letter A (AX condition). All other trials are non-target trials where either a valid cue is followed by an invalid probe (AY condition), or an invalid cue is presented (BX or BY condition).

The crucial aspect is that in some cases (BX condition), task-relevant contextual information that precedes the presentation of the probe must be used to inhibit a dominant response tendency.

The active representation and maintenance of task-relevant information during the AX-CPT retention interval is hypothesized to occur in the PFC (Braver & Cohen, 2000). The proactive role of the PFC in maintaining task-relevant information has been validated through brain-imaging studies demonstrating that PFC activity is increased when task-relevant information is being maintained during the AX-CPT task (Braver, Cohen, & Barch 2002). Similar conclusions have been drawn based on PFC activity measured during the Stroop (MacDonald et al., 2000), and flanker tasks (Schmid et al., 2015). Relatively greater activity in the left compared to right PFC, or left frontal asymmetry, might be particularly responsible for proactive engagement (Davidson, 1992; Harmon-Jones, 2003). For example, left frontal asymmetry is associated with better task performance on incongruent flanker trials relative to congruent trials (Schmid et al., 2015), an indication of increased proactive control. If left frontal asymmetry is visible when selecting a correct response during high-conflict trials, this is thought to signify that the PFC is maintaining context representations to guide top-down control and inhibit competing distractor information from the flankers, thus facilitating identification of the target stimuli.

Interactions between the PFC and the ACC

According to the conflict-monitoring theory, activation of the PFC is thought to be modulated by the ACC. That is, a reactive monitoring system, facilitated by the ACC, signals the need for PFC-mediated, proactive control as ongoing conflicts are processed (Botvinick et al., 2001; Kerns et al., 2004). For example, if an individual is not prepared to suppress distracting information when presented with a high-conflict stimulus, the ACC will reactively respond to the conflict by calling for the activation of proactive control (i.e. PFC activity) which results in heightened preparation for possible up-coming conflict and subsequently diminished conflict sensitivity. Stated differently, if an individual is not engaged in proactive control when a cognitively demanding event is encountered, reactive control will act as a corrective mechanism by reactivating task-relevant information, which consequently reduces conflict-processing.

Interactions between the PFC and the ACC have been observed on the level of behavioral adjustments. For example, the Flanker effect, defined as the difference in reaction times between congruent and incongruent flanker trials, is modulated by previous trial congruency. A reduced Flanker effect, or the difference in processing between incongruent and congruent trials, is found for incongruent trials preceded by an incongruent trial (iI trials) compared to incongruent trials preceded by a congruent trial (cI trials; Gratton, Coles, & Donchin, 1992). That is, individuals tend to respond faster and more accurately on iI trials compared to cI trails. This pattern may be a product of increased top-down biasing of control (i.e. proactive control) following high-conflict

incongruent trials (Botvinick et al., 2007) facilitating responses to subsequent instances of conflict.

Reduced conflict processing due to enhanced proactive control is also discernible on the level of neural activity. For example, neuroimaging studies have found greater activation of the ACC on cI trials compared to iI trials and that ACC activity on initial high-conflict trials predicted greater PFC activation on the following trial (Kerns et al., 2004; Kerns, 2006). As noted above, work has also shown smaller N2 amplitudes on cI trials compared to iI trials (Larson et al., 2012). ACC activity was greater on cI trials compared to iI trials because for iI trials the initial high-conflict trial led to the recruitment of proactive control which subsequently reduced conflict processing for the following trial. Findings showing that greater ACC activity on the initial high-conflict trial predicted PFC activation on a following trial suggest a relation between ACC activity and subsequent PFC engagement, or that reactive processing signals for the recruitment of proactive control. These findings are consistent with predictions made by the conflict monitoring hypothesis that the ACC is responsible for conflict-monitoring and when activated the ACC signals for the recruitment of proactive control.

Worry and Interactions between the PFC and the ACC

The DMC theory is also able to explain instances where affective factors influence cognitive processing. As described earlier, proactive engagement aids in maximizing rewards through biasing thoughts and behaviors in a goal directed manner. Conversely, reactive processing is associated with conflict sensitivity and detection. Goal

seeking and conflict detection may relate to two affective dimensions of personality known as approach and avoidance, respectively (Braver et al., 2007). The reinforcement sensitivity theory formulated by Gray (1982) proposes that these two dimensions are part of constructs that represent two distinct motivational systems: the *Behavioral Activation System* (BAS) and the *Behavioral Inhibition System* (BIS). The BAS is a neurobiological system activated by goal pursuit that initiates approach behavior. The BIS is a neurobiological system activated by goal conflict that enhances conflict sensitivity and is positively associated with psychological problems such as anxiety and negative affect (Gray, 1982). As BIS activation is associated with a heightened sensitivity to conflict, it should also be associated with greater reactivity in the conflict-monitoring system that detects such conflicts, i.e. the reactive control system (Braver et al., 2007). Hyper-activity in the conflict-monitoring system also tends to be present in individuals relatively high in levels of the trait anxious apprehension, defined as excessive levels of worry and rumination, therefore, such individuals might exhibit heightened reactive control and reduced proactive control compared to those low in trait anxious apprehension (Moser, Moran, Schroder, Donnellan, & Yeung, 2013). Thus, trait level worry is one affective factor that could possibly interact with cognitive processing.

Braver (2012) has argued that although individuals typically alternate flexibly between proactive and reactive control to meet shifting environmental demands, high levels of trait worry can lead to worrisome thoughts occupying cognitive resources and interfering with the employment of more taxing, goal-directed proactive control. This form of cognitive depletion can result in a greater reliance upon more automatic, and less

efficient, reactive processes of cognitive control (Braver, 2012). Such compensation could be a potential “hidden cost” related to worry as normally simple tasks become difficult due to the consumption of cognitive resources that could otherwise be used for proactive control. Without the resources needed to engage proactive control, there might be a greater dependence in general on reactive processing (Berggren & Derakshan, 2013). However, a better understanding of dependence on reactive processing due to affective variables requires more work looking distinctly at affective influences on cognitive processing and task performance (Braver et al., 2007). Such work would aid in explaining how cognition and affect might interact.

The negative effects of increased reliance upon reactive control have been demonstrated in work linking levels of social anxiety, hallmarked by worry about social evaluation (American Psychiatric Association, 2013), with impaired proactive control. For example, Schmid and colleagues (2015) argued that individuals low in social anxiety tend to rely on proactive control to guide task performance whereas individuals high in social anxiety tend to rely on reactive control to guide performance. In this work, proactive and reactive control were indexed by left frontal asymmetry and N2 amplitudes respectively. In order to gain a continuous measure of sustained proactive control, left frontal asymmetry was measured throughout a flanker task during inter-trial intervals. N2 amplitudes were used to index trial-by-trial conflict related ACC activity. Whereas left frontal asymmetry predicted task performance for all individuals, N2 amplitudes predicted flanker task performance only for individuals high in levels of social anxiety.

The authors suggested this pattern of results emerged because contributions of proactive and reactive control to flanker task performance are modulated by levels of social anxiety. Individuals low in social anxiety were proposed to exhibit a proactive control profile characterized by the use of attentional focus and top-down control. Conversely, the predominant role of reactive control guiding task performance among individual high in social anxiety was thought to suggest a reactive profile wherein control was being guided primarily through reactive processes. This may be due to high-levels of social anxiety leading participants to attend more to socially-evaluative aspects of the experimental setting (e.g., concern about how their performance would lead them to be judged by others), taxing cognitive resources and consequently creating a greater reliance on less efficient reactive processing. Notably, those high in social anxiety also tended to perform worse on the flanker task, evidence of a potential "hidden cost" of social anxiety. Overall, this work provided initial evidence that individuals might use different cognitive control strategies to guide performance based on levels of social anxiety.

Thus, there is preliminary evidence that levels of social anxiety predict whether individuals tend to rely on proactive or reactive strategies to guide task performance during a flanker task (Schmid et al., 2015). However, it is not clear whether another trait related to conflict-processing, worry, also impacts the extent to which a proactive or reactive strategy is favored to guide task performance. Given worry's relation to reactive processing (Braver, 2012), whether an individual tends to rely on a proactive or reactive control strategy to guide task performance might be a function of worry. High levels of worry could possibly affect cognitive control in a similar fashion as social anxiety,

through leading to an increased reliance on reactive processing to guide task performance.

Recent work suggests that, although worry is linked primarily with General Anxiety Disorder (GAD), after controlling for a GAD diagnosis, worry is still relevant to a range of psychiatric disorders such as depression and bi-polar mania (Kertz, Bigda-Peyton, Rosmarin, & Björgvinsson, 2012). High levels of worry also reflect pre-clinical levels of symptoms, allowing investigations about the generalization of risk processes to non-clinical populations. Understanding how this prevalent and prognostic trait interacts with cognitive processing could help to explain how emotion interacts with cognition, and could shed light on why some individuals are more successful than others at regulating thoughts and actions in order to facilitate goal attainment.

Also unclear is whether there are direct associations between sustained proactive control and reactive processing at the level of neural functioning. Traditional examinations of cognitive control have focused almost entirely on reactive mechanisms (Schmid et al., 2015). However, according to the conflict-monitoring hypothesis, exhibiting sustained task-related proactive control (i.e. greater left frontal activity) should be associated with reduced subsequent conflict processing (i.e. smaller N2 amplitudes; Botvinick et al., 2001). If cognitive control can be maintained by the sustained enhancement of proactive control, this would offer evidence that proactive as well as reactive mechanisms are capable of driving control, thus adding a new perspective to control engagement. A proactive model of control would not only explain another means through which control can be maintained, but could also explain the presence of

differences in conflict sensitivity. Being that enhanced proactive control results in reduced subsequent conflict-processing (Botvinick et al., 1999), the sustained enhancement of proactive control could lead to muted conflict sensitivity. Given that worry is associated with impaired proactive control and increased conflict processing, variations in worry might modulate a relation between proactive and reactive control. Individuals high in trait worry might maintain a reliance on reactive control processes to guide task performance due to a depletion of cognitive resources needed to properly engage proactive control.

The Current Study

The current study aimed to investigate the relation between neural correlates of proactive (i.e. left frontal asymmetry) and reactive control (i.e. N2 Amplitude) and the degree to which their association might be moderated by trait-level individual differences in worry. Worry was chosen as a possible moderator because of its relation to reactive processing (Braver, 2012); additionally, worry was of interest because its prevalence among typically developing populations (Davey & Wells, 2006).

Although traditional models of cognitive control have focused on reactive control signaling the need for increased proactive control (Botvinick et al., 1999; Kerns et al., 2004; Larson, Clayson, & Baldwin, 2013), relatively little work has specifically investigated whether the sustained exertion of proactive control predicts levels of reactive control (Schimd et al., 2015). Understanding whether proactive control can guide task performance above and beyond reactive processing could explain another means through

which cognitive control is engaged. Cognitive control may not rely solely on reactive processes for activation but could be sustained through the effortful employment of proactive control (Braver et al., 2007). Evidence showing that the sustained employment of proactive control predicts reduced reactive control would add support for this hypothesis. In this scenario, the reactive control function to call for the recruitment of proactive control might be superseded by the continuous activation of proactive control. Such results would also indicate a complementary role between proactive and reactive control and imply that a proactive strategy in favor over a reactive strategy is being relied upon to guide task performance. The favoring of a strategy, proactive or reactive, could potentially explain variability in cognitive control within and between people.

If worry moderates associations between proactive and reactive control, this will inform trait-level differences that can lead to inter-individual variation in cognitive control strategies. Individuals high in levels of trait worry might tend to rely on reactive processing to guide cognitive control. Positive results would also provide evidence of an intersection between cognitive and affective processing.

Findings from this study will inform whether associations between proactive and reactive control are also associations between neural systems, and could provide converging evidence that left frontal asymmetry serves as a proxy for proactive control and N2 amplitude serves as a proxy for reactive control. Importantly, this study seeks to illumine the nature of the interplay between proactive and reactive control. By focusing on a relatively understudied proactive model of cognitive control, this investigation could

describe a role of proactive control that is not solely dependent on reactive control but also reciprocally guides the extent to which reactive control occurs.

Plan for Analysis

Based on the extant literature, proactive control was quantified during a flanker task using frontal asymmetry scores such that greater left frontal asymmetry indicated greater proactive engagement. In order to gain a measure of sustained proactive activity that was not confounded with immediate conflict-monitoring processes, proactive control was quantified during relatively conflict-free correct congruent flanker trials that preceded high-conflict correct incongruent trials. This study also sought to focus on proactive activity that was relatively unrelated to error processing, so only correct congruent trials were analyzed. This allowed for a measure of proactive activity during trials that were relatively low in both conflict and processes associated with error. In order to measure the relation between reactive activity and sustained proactive processes, reactive control was quantified during a flanker task by N2 amplitudes generated during high-conflict correct trials preceded by correct conflict-free trials.

Preliminary analyses included an investigation of where N2 was maximal in the current data. To do this, a repeated measures ANOVA was used to test for differences in N2 amplitudes across midline electrode sites. Then, an independent sample *t*-tests to test for possible sex differences in primary variables. Bivariate correlations were used to explore possible associations between primary variables. Finally, two-step hierarchical

regression models were used to investigate whether proactive control, measured as frontal asymmetry, interacted with worry to predict reactive control, measured as N2.

METHOD

Participants

This study was approved by the Montana State University Institutional Review Board (IRB #RB01014). Participants were recruited through the Montana State University psychology subject pool and participated in this experiment for partial fulfilment of course requirements. The sample included 123 participants (72 girls; M_{age} = 20.86; *SD* = 4.28). Eighty-one percent of participants identified themselves as Non-Hispanic Caucasian, 4% as African-American, 5% as American Indian or Alaskan Native, <1% as Native Hawaiian or Other Pacific Islander, 2% as Asian American, <1% as multiracial, <1% as other, and 5% declined to answer. Participants were free of neurological impairments and were not taking stimulant medications at the time of the study.

Procedure

Data for the current study were collected as part of a larger project that aimed to examine the role of emotion regulation strategies in association with putative costs of cognitive control. After participants arrived at the lab, written informed consent was obtained. Mental health symptoms, allostatic load, academic achievement information, demographic information and indicators of of socio-economic status (i.e., race/ethnicity, income, parent education, sex, employment status) were assessed via a computerized questionnaire. Following this, blood pressure was recorded and a saliva sample was

taken. A five-minute resting baseline measure of EEG activity was recorded. Participants completed computerized tasks while EEG data were recorded after which another five-minute resting baseline was recorded. A second saliva sample was taken at the end of the visit. Subsequent analyses focused on trait-worry and EEG data during the computerized flanker task as the remaining data were irrelevant for the aims of this project.

Trait Worry

In order to assess trait levels of worry, participants completed the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990), a 15-item self-report measure that asked participants to rate the degree to which statements describing excessive and/or uncontrollable worry (e.g., “I am always worrying about something”; “I've been a worrier all my life”) were typical or characteristic of themselves (1=not at all; 5= very typical). A mean score was generated for each participant to maximize all available data. The PSWQ demonstrated high internal consistency in this study (Chronbach’s α=.95), and has shown to be reliable over intervals as long as 8–10 weeks (Meyer et al., 1990).

EEG Assessment

Participants were fitted with a 64 channel nylon cap for EEG collection (ElectroCap International, Eaton, OH). For each baseline period, participants were asked to alternate between resting for 1 minute with eyes open and 1 minute with eyes closed for a total of 5 minutes.

After the initial baseline recording, participants completed the Eriksen flanker task (Eriksen & Eriksen, 1974), on a Dell computer using Presentation® stimulus delivery software (Neurobehavioral Systems, Inc.: Berkeley, Ca). Stimuli for the flanker task comprised two trial types with each trial type comprising approximately 50% of the task. On congruent trials, a central target arrow was flanked by four arrows that were directionally congruent (< < < < <; > > > > >). For incongruent trials, a central; target arrow was flanked by four arrows that were directionally incongruent (< < > < <; > > < > >).

One session of the flanker task consisted of four experimental blocks of 100 trials each. Participants were allowed a short break between blocks. Each trial began with the presentation of a fixation cross in the center of the screen for 1000 msec followed by a stimulus presentation (either congruent or incongruent) for 200 msec. Participants were allowed up to 1000 msec to respond after the stimulus presentation. Then, a blank inter-trial interval screen was presented for 1000-2000 msec after which the next trial began. The timing for the inter-trial interval screen was randomly varied between times of 1000,1250,1500,1750, or 2000msec. A full trial of the flanker task is depicted in Figure 1. Accuracy and reaction time were recorded for each trial.

EEG data were acquired during baselines and the flanker task using a BioSemi Active Two system with acquisition software BioSemi ActiView V705 (BioSemi, B.V.: Amsterdam, Netherlands). Data were sampled at a rate of 2,048 Hz with a gain of 1. All channels were referenced to a common mode sense active electrode during recording.

EEG data processing was conducted offline using a semi-automated procedure in Brain Vision Analyzer 2.0 (BVA; Brain Products: Gilching, Germany).

Reactive Control

Reactive control was quantified using the N2 event-related potential. To calculate N2, EEG data collected during the flanker task were re-referenced to an average of the two mastoid channels. EEG data were highpass filtered with a low cutoff 0.10 Hz (12dB rolloff) and submitted to an ocular correction in order to remove eye movement or eye blink artifacts (Gratton, Coles, & Donchin, 1983). EEG Data were then divided into 4,900 msec segments starting 2,900 msec before stimuli presentation. This extended pre-trial interval allowed previous trial types to be incorporated into the definition of the segment so that N2s could be calculated for both incongruent trials preceded by congruent trials (cI trials) and congruent trials preceded by congruent trials (cC trials). Initial trials were made to be congruent to gain a measure of conflict processing on subsequent trials that was not modulated by previous trial conflict processing. To ensure that participants were engaged, only correct trials were included in the data set. Segments were baseline corrected by subtracting the average activity from -200 to 0 msec prior to each stimulus presentation for the scored trial. Using a semiautomated procedure, artifacts were defined when one of the following conditions was met: a step of more than 75μV occurred between data points, a difference of 150 μV occurred within a single segment, an absolute voltage exceeded 200 μV within a single segment, or amplitudes less than 0.5 μV occurred within a 50 msec period. All segments were also visually inspected for any remaining artifacts.

Individual averages for artifact-free segments were generated and were lowpass filtered at 30 Hz. To control for individual differences in neural activity on cC trials, an N2 difference wave (ΔN2) was created by subtracting the cC trial amplitudes from cI trial amplitudes. The ΔN2 approach was chosen because of previous recommendations that this approach targets neural processes of interest while controlling for other simultaneously active yet unrelated neural activity (Luck, 2014). Calculated in this way, ΔN2 functions to control for stimulus-related activity that is also present on relatively conflict free cC trials and thus allows for a more specific investigation of neural activity related to conflict processing that occurs during high-conflict incongruent trials. As ΔN2 specifically targets conflict related neural activity, it is a measure that selectively responds to conflict processes that result in activating reactive control. Dampened responses to conflict produce less reactive control and are made visible as relatively smaller (more positive) ΔN2's whereas greater conflict processing results in greater reactive control and larger (more negative) ΔN2's.

An automated procedure was used to score the peak of the ΔN2 as the most negative peak occurring in the window of 250-300ms following stimulus presentation. Scored in this fashion, greater ΔN2 corresponds to greater negative amplitudes. Overall the mean number of cI trials used to calculate ΔN2 was 64.71 (*SD* = 24.55), and the mean number of cC trials used was 67.18 (*SD* = 25.83).

Proactive Control

Proactive control reflected by neural activity in the prefrontal cortex was quantified as hemispheric asymmetry in alpha power at frontal electrode sites. To calculate frontal asymmetry (i.e. proactive control), EEG data collected during the flanker task were re-referenced to an average of the two mastoid channels. EEG data were highpass filtered with a low cutoff 0.10 Hz (12dB rolloff) and then lowpass filtered with a high cutoff 30 Hz (12dB rolloff). Data from each participant were submitted to an ocular correction in order to remove eye movement or eye blink artifacts (Gratton, Coles, & Donchin, 1983). EEG data were divided into 1,000 msec segments starting 50 msec after the participant's response for the initial correct congruent trial. The data were segmented so that an initial congruent trial was followed by a subsequent incongruent trial (cI). Again, only correct trials were included.

The study aimed to acquire a measure of sustained proactive control that was not confounded with immediate conflict/error processing. With this in mind, proactive control was measured during correct congruent trials as these trials should contain the lowest levels of conflict/error processing. The progression of trial types were ordered so that frontal asymmetry segments could be extracted starting after the response for the initial correct congruent trial and before the presentation of the stimuli for the subsequent correct incongruent trial. Entire segments were baseline corrected. Using a semiautomated procedure, artifacts were defined when one of the following conditions was met: a step of more than 75μV occurred between data points, a difference of 150 μV occurred within a single segment, an absolute voltage exceeded 200 μV within a single

segment, or amplitudes less than 0.5 μV occurred within a 50 msec period. All segments were also visually inspected for any remaining artifacts.

Artifact-free data were submitted to a Fast-Fourier Transform using a Hamming window (50% overlap) and then aggregated. Power (μV^2) in the alpha (8–13 Hz) frequency band was extracted for each participant. Alpha power values were positively skewed and so were natural log transformed to correct for non-normal distribution. The resulting transformed values were then used to calculate frontal asymmetry by subtracting left frontal alpha power at electrode site F5 from right frontal alpha power at electrode site F6. As alpha power is thought to be an inverse of cortical activity, higher values on this metric signified greater left-sided PFC activity (Pizzagalli, Sherwood, Henriques, & Davidson, 2005) and thus indexed greater proactive control that is not dependent on conflict.

Missing Data

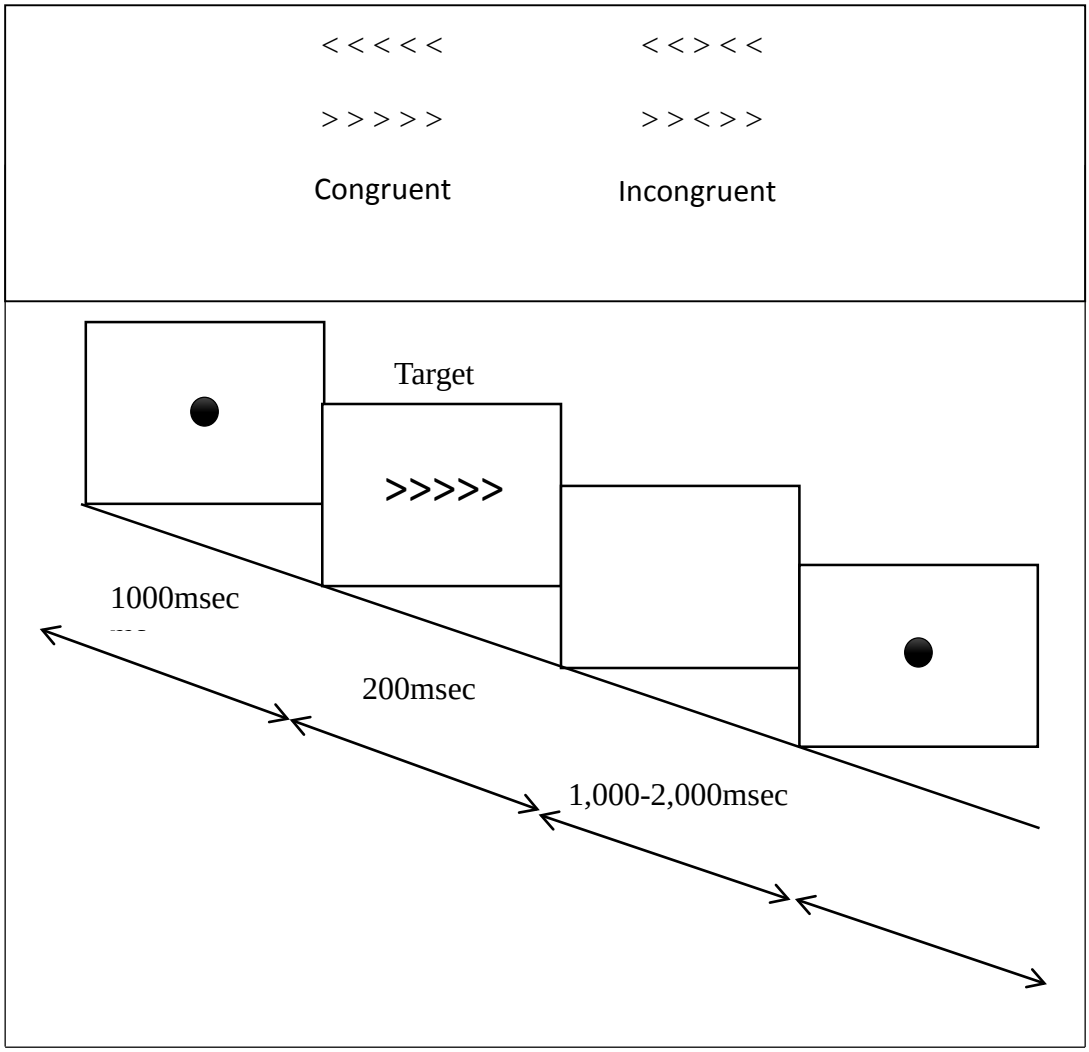
For the current analyses, a total of 45 participants did not provide usable EEG data either due technical error, task refusal, or excessive artifact. In addition, nine left-handed participants were excluded from the data set due to evidence that patterns of hemispheric activation may differ in left- and right-handed individuals (Bryden, 1982). A total of 34 participants did not provide usable ΔN2 data at FCz and a total of 33 participants did not provide usable ΔN2 data at Cz; either due technical error, task refusal, or excessive artifact. Five participants did not complete the PSWQ. Thus, a total of 65 participants were available to test study hypotheses.

FIGURES

Figure 1: Experimental procedure. (a) The four stimuli used in the present study, and (b) an overview of the procedure.

(a)

(b)



RESULTS

Preliminary Analyses

To investigate where ΔN2 was maximal, a repeated measures ANOVA was used to test for differences in ΔN2 amplitudes across midline electrode sites (Fz, FCz, Cz, and Pz). Greenhouse-Geisser corrections were applied when necessary to correct for violations of sphericity. Results showed no linear main effect of electrode site on ΔN2 scores (F (3, 88) = 2.058, p=.128). However, a significant quadratic main effect emerged (F (1, 88) = 9.437, p = .003, η^2_p=0.097).

Follow up analyses using paired-samples t-tests to examine ΔN2 effects at each electrode site revealed that ΔN2 was significantly greater at FCz (M=-3.08, SD=3.10) compared to Fz (M=-2.66, SD=3.22; t(89)=-2.407, p = .018, d = -.133), ΔN2 was marginally greater at Cz (M=-3.00, SD=3.07) compared to Fz (M=-2.55, SD=3.07; t(88)=-1.804, p = .075, d = -.147), and ΔN2 was marginally greater at Cz (M=-3.00, SD=3.07) compared to Pz (M=-2.68, SD=2.94; t(88)=-1.903, p = .06, d = -.106). There were no significant differences in ΔN2 at remaining electrode sites: ΔN2 at FCz (M=-2.97, SD=2.95) was not different from Cz (M=-3.00, SD=3.07; t(88)=0.163, p = .871), ΔN2 at FCz (M=-3.08, SD=3.10) was not different from Pz (M=-2.79, SD=3.11; t(89)=-1.295, p =.199), and ΔN2 at Fz (M=-2.66, SD=3.22) was not different from Pz (M=-2.79, SD=3.11; t(89)=.490, p =.625). Results from these analyses suggest that ΔN2 was greatest ΔN2 at electrode sites FCz and Cz compared to both the frontal and parietal

electrode sites. Thus, subsequent analyses focused on ΔN2 at the fronto-central electrode sites (FCz and Cz).

Independent sample *t*-tests were used to explore for possible sex differences in primary variables. Results indicated no significant sex differences in any primary variables (all *t*s ≤ 2.03, *p*s > 0.05) except worry (*t* (106.97) =-6.70, *p* = 0, *d* = -1.25). On average, men (*M*=2.47, *SD*=0.68) reported lower levels of worry than did women (*M*=3.40, *SD*=0.80). Therefore, subsequent analyses involving levels of worry controlled for sex.

Bivariate correlations exploring possible associations between primary variable revealed no significant correlations. Descriptive statistics are reported in Table 1. Bivariate correlations between primary variables are reported in Table 2.

A paired sample t-test indicated that, as expected, reaction times during the flanker task were significantly longer for cI trials (*M* = 536.78 msec, *SD* = 61.19) than for cC trials (*M* = 463.56 msec, *SD* = 55.42; *t*(106) = 16.32, *p* < .001, *d* =1.587). These results confirm that greater conflict processing was successfully elicited during cI trials.

Frontal Asymmetry and Worry as Predictors of ΔN2

Two-step hierarchical regression models were used to investigate whether proactive control, measured as frontal asymmetry, interacted with worry to predict reactive control, measured as ΔN2. In order to minimize possible problems with multicollinearity and to provide the opportunity for an internal replication of results, ΔN2 at FCz and ΔN2 at Cz were tested as dependent variables in separate models. The same

statistical procedures were used for each model: the main effects for sex, frontal asymmetry, and worry were entered in the first step and the interaction between frontal asymmetry and worry was entered in the second step. Consistent with the suggestions of Aiken & colleagues (1991), all predictor variables were centered prior to the creation of interaction terms.

The results of the analysis predicting ΔN2 at FCz are presented in Table 3. Neither frontal asymmetry nor worry significantly predicted ΔN2 at FCz. However, consistent with study hypotheses, frontal asymmetry and worry interacted to predict ΔN2 amplitudes ($\Delta R^2 = 0.08$, $\beta = 0.31$, $p = .02$). Per the suggestions of Aiken & colleagues (1991), this interaction was probed by recentering worry at high (+1 SD) and low (-1 SD) values. This method allowed for a specification of the effects of frontal asymmetry on ΔN2 at varying levels of worry while conserving statistical power through using a continuous probe of the interaction. Results indicated that when levels of worry were high, frontal asymmetry was not associated with ΔN2 ($\beta = -0.27$, $p = .20$). Contrastingly, when levels of worry were low, greater left frontal asymmetry was associated a smaller (more positive) ΔN2 ($\beta = 0.31$, $p = .04$).

The results of the analysis predicting ΔN2 at Cz are presented in Table 4. Analysis results at Cz revealed what was essentially an internal replication of the results found at electrode site FCz. Similar to the pattern of results at FCz, neither frontal asymmetry nor worry significantly predicted ΔN2 at Cz, but frontal asymmetry and worry interacted to predict ΔN2 ($\Delta R^2 = 0.07$, $\beta = 0.29$, $p = .03$). Once again, this interaction was probed by recentering worry at high (+1 SD) and low (-1 SD) values.

Although frontal asymmetry was associated ΔN2 at high and low levels, neither of the simple slopes were significant (high: β = -0.32, *p* = .14, low: β = .22, *p* = .14)

Follow-up Analyses

Initially, the effects of frontal asymmetry on ΔN2 were examined at moderate levels of worry (+/- 1 SD). In order specify the effects of frontal asymmetry on ΔN2 at levels of worry comparable to those who qualified for a GAD (GAD; Meyer, Miller, Metzger, & Borkovec, 1990), the interaction was re-probed with ΔN2 recentered at very high (+2 SD) and very low (-2 SD) levels.

For the model predicting ΔN2 at FCz, results indicated that when levels of worry were very high (+2 SD), greater left frontal asymmetry was marginally associated with a larger (more negative) ΔN2 at FCz (β = -0.55, *p* = .08). Contrastingly, when levels of worry were very low (-2 SD), greater left frontal asymmetry was significantly associated a smaller ΔN2 at FCz (β = 0.60, *p* = .01).

For the model predicting ΔN2 at Cz, results similarly showed that when levels of worry were very high (+2 SD), greater left frontal asymmetry was marginally associated with a larger ΔN2 at Cz (β = -0.56, *p* = .08). Alternatively, when levels of worry were very low (-2SD), greater left frontal asymmetry was significantly associated with a smaller ΔN2 at Cz (β = 0.48, *p* = .04).

TABLES

Table 1: Descriptive Statistics Using t-tests to Compare Differences in Sex

	N	Mean	*SD*	*t*-test
ΔN2 Fz	90	-2.65	3.22	0.46
Men	37	-2.85	4.26	
Women	52	-2.49	2.28	
ΔN2 FCz	90	-3.08	3.10	0.64
Men	37	-3.29	3.76	
Women	52	-2.84	2.51	
ΔN2 Cz	89	-3.00	3.07	1.37
Men	36	-3.49	3.62	
Women	52	-2.58	2.56	
ΔN2 Pz	90	-2.79	3.11	2.03
Men	37	-3.52	3.55	
Women	52	-2.19	2.63	
Frontal Asymmetry	69	0.15	0.25	-1.63
Men	24	0.22	0.25	
Women	44	0.11	0.26	
Worry	118	3.03	0.88	6.47*
Men	46	2.47	0.68	
Women	70	3.40	0.80	

Note. *$p < 0.05$.

Table 2: Bivariate Correlations among Primary Variables

	1.	2.	3.	4.	5.	6.
1. ΔN2 Fz						
2. ΔN2 FCz	.861*					
3. ΔN2 Cz	.696*	.818*				
4. ΔN2 Pz	.655*	.770*	.860*			
5. Frontal Asymmetry	.090	.124	.028	-.076	-.219	
6. Worry	.061	.055	.025	.010	.063	-.074

Note. *$p < 0.01$.

Table 3: Hierarchical Regression Predicting ΔN2 at FCz from Frontal Asymmetry, Worry, and the interaction between Frontal Asymmetry and Worry While Controlling for Sex

	Step 1				**Step 2**			
	B	***SE(B)***	**β**	**95% *CI***	***B***	***SE(B)***	**β**	**95% CI**
Sex	-.03	.84	-0.01	[-1.72, 1.65]	-.250	0.82	-.05	[-1.89, 1.39]
Frontal Asymmetry	1.37	1.33	.13	[-1.30, 4.03]	.22	1.37	.02	[-2.53, 2.96]
Worry	.20	.45	.07	[-0.70, 1.10]	.24	.43	.08	[-0.62, 1.11]
Asymmetry * Worry					3.41	1.43	.31*	[0.55, 6.27]
	R^2 = 0.02				R^2 = 0.10			

Note. $*p < 0.05$.

Table 4: Hierarchical Regression Predicting ΔN2 at Cz from Frontal Asymmetry, Worry, and the interaction between Frontal Asymmetry and Worry While Controlling for Sex

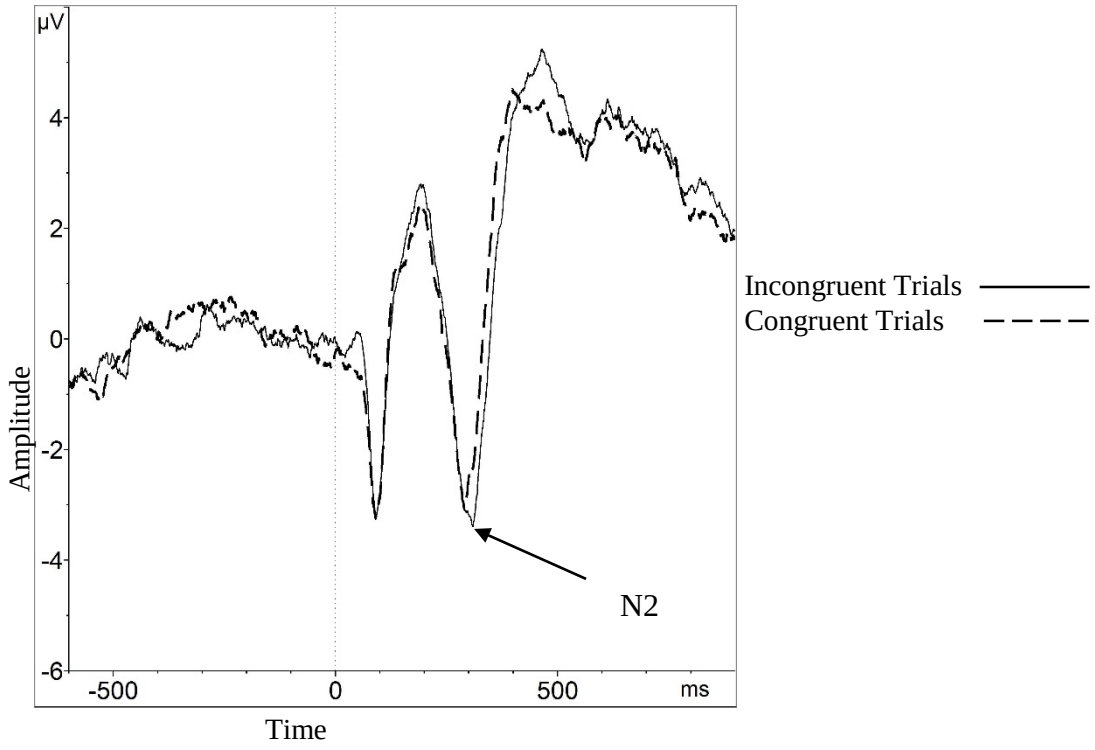
	Step 1				**Step 2**			
	B	***SE(B)***	**β**	**95% *CI***	***B***	***SE(B)***	**β**	**95% CI**
Sex	.66	.94	.11	[-1.21, 2.53]	.44	.92	.07	[-1.39, 2.27]
Frontal Asymmetry	.60	1.48	.05	[-2.36, 3.56]	-.57	1.53	-.05	[-3.64, 2.50]
Worry	- .06	.50	-.02	[-1.06, .93]	- .02	.49	-0.01	[-.99, .95]
Asymmetry *Worry					3.49	1.60	0.29*	[.30, 6.68]
	R^2 = 0.01				R^2 = 0.08			

Note. $*p < 0.05$.

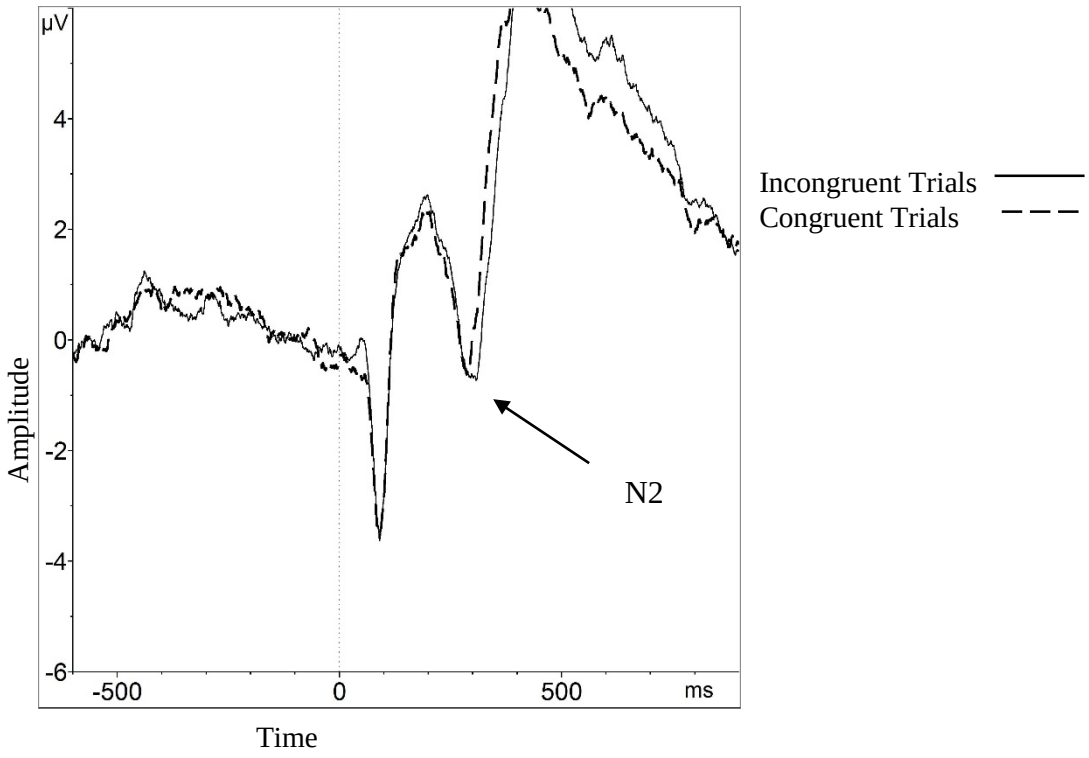
FIGURES

Figure 2. Grand Average Waveforms at (A) FCz and (B) Cz

A. FCz



B. Cz


μV
4
2
0
-2
-4
-6
-500
0
500
ms
Amplitude
Time
N2
Incongruent Trials
Congruent Trials

DISCUSSION

The present experiment provided evidence for a moderated relation between the two modes of cognitive control put forth by the DMC theory. Results indicated an association between a neural correlate of proactive control (i.e., left frontal asymmetry) and a neural correlate of reactive control (i.e., ΔN2 amplitude) that was dependent on trait levels of worry. Specifically, when levels of worry were low, greater left frontal asymmetry predicted smaller N2 amplitudes. Conversely, when levels of worry were high, proactive and reactive control appeared to be unrelated. Of additional interest, at levels of worry comparable to those found in individuals diagnosed with GAD, greater left frontal asymmetry predicted somewhat increased reactive processing.

The findings from this study elaborated on theoretical assumptions put forth by the DMC theory by providing direct neural evidence that a neurological component signifying proactive control predicts less activation of a neurological component signifying reactive control. Notably, the effect of sustained proactive control on reactive processing was moderated by levels of worry. The DMC theory argues that proactive and reactive control are dissociable, but that an interaction between the two is feasible. Theoretically, proactive and reactive control could interact such that enhanced proactive control produces less reliance on reactive processing (Braver et al., 2007). This is the pattern of results found in the current study when levels of worry were low. At low levels of worry greater left frontal asymmetry, a measure associated with the maintenance of proactive control, was associated with smaller ΔN2 amplitudes, a measure associated with reactive processing.

This work offers support for previous assertions that proactive control can play a role in cognitive processing, above and beyond reactive control (Schmid et al., 2015). Evidence that sustained heightened levels of proactive control predict reduced conflict-related processing suggests that proactive control is taking a role in task guidance. Thus, the sustained engagement of proactive processing may not necessarily rely on persistent input from reactive processing, but might actually dampen neural processes associated with reactive control. Considering that proactive control is a more efficient form of cognitive processing, understanding the feasibility of the sustained engagement of proactive control could help to inform interventions seeking to improve cognitive functioning.

The current results also offer support for theoretical assertions of the conflict-monitoring hypothesis, which states that when a divergence between higher-level goals and lower level responses (i.e., conflict) is detected, subsequent conflict related processing is diminished. In terms of the DMC theory, an assumption posited by the conflict-monitoring hypothesis is that a reduction of conflict related processing depends on enhancements in proactive control. Findings from this study are in line with that assumption, in that greater proactive control predicted reduced conflict processing, or diminished reactive control. Notably, this association was only visible when levels of worry were low. Such a moderated interaction holds implications for shifting between proactive and reactive strategies; namely, shifting away from a reliance on reactive control might be difficult at high levels of worry. This hypothesis is supported by the present findings. When levels of worry were high, the relation between proactive control

and reactive processing was either non-significant or enhanced proactive control was marginally associated with greater reactive processing. This presents a potential "hidden cost" of worry due to trouble with shifting to a primarily proactive mode of control. Lacking the cognitive resources needed to shift cognitive strategies may mean not reaping a potential benefit associated with proactive control, less interference from conflict-processing, and greater reliance on less efficient reactive processing for task guidance.

High levels of worry could be associated with an increased reliance upon a reactive strategy to compensate for an inability to use reactive control adaptively (Inzlicht et al., 2015). That is, heightened worry may lead to excessive monitoring for conflict in both the task and in their environment, which could lead to a competition between task-relevant and task-irrelevant reactive signals, thus making it difficult to discern how to best allocate cognitive resources. The source of such task-irrelevant information, conflict detection, is an automatic process (Braver et al., 2007). Because this process is automatic, the actual generation of signals requires little effort. However, the proactive control system could still be overwhelmed by a flood of input as it relies on effort to operate and is limited in its capacity to coordinate information (Braver, 2012).

This work supports the existence of an interaction between cognitive control and affect. In the past, theorists have argued that affective states undermine the facilitation of cognitive control (Metcalfe & Mischel, 1999). Recently however, scientists are espousing the view that affect and cognition are not separately organized faculties, but are the product of broadly distributed functional networks that interact to create both cognition

and affect (Inzlicht et al., 2015; Lindquist & Barrett, 2012). The DMC theory offers a potential explanation for how such networks interact to impact cognition and affective processing. For example, the theory argues that the monitoring of conflict produces a mode of cognitive control, reactive processing. Enhanced conflict monitoring has been shown to relate to emotional outcomes via increased ACC activation. For example, the ACC is tonically enhanced for individuals relatively high in conflict sensitivity, a trait associated with affective processing (Braver et al., 2007; Zald, Mattson, & Pardo, 2001). Because ACC activation is closely linked with reactive processing, the priming of this system through enhanced conflict-sensitivity could predict the utilization of the conflict-monitoring function of the ACC during cognitive processing. The current study supports this prediction, as individuals high in trait worry did not exhibit a reduction in reactive processing due to enhanced proactive engagement, a finding that contrasts the relation that was apparent for individuals low in trait-worry.

Conflict monitoring is one function of the ACC that is closely tied to cognitive processes such as reactive control. Besides its relation to cognitive control, perceiving conflict is also associated with emotional states such as negative affect (Carver & Scheier, 2011). The experience of a negative affective state, such as worry, is thought to motivate proactive control (Bradley, Codispoti, Cuthbert, & Lang, 2001). In this sense, negative affect could be an essential component of control, operating to signal the need to exert top-down proactive employment. The relation between negative affect and cognition may become maladaptive when, despite enhanced motivation, negative affect is not down-regulated and reactive processing remains robust. This scenario might be

reflected in worriers. Ideally, conflict detection leads to a recruitment of proactive control, which then adjusts and diminishes subsequent reactive processing and interference from high-conflict bottom up stimuli (Botvinick et al., 1999). When levels of worry are high, however, increased proactive control does not predict decreased reactive control, possibly because of problems with correctly allocating cognitive resources and appropriately utilizing conflict-monitoring processes.

The current research is limited in its ability to determine the boundaries between cognition and affect but does shed light on how cognitive control is influenced by an affective state. In the present sample, worry was a moderating factor. The intersection between cognitive control and worry could be tied to the perception of conflict. If high levels of worry reduce one's ability to properly engage proactive control this may be because of malfunctioning in the feedback loop that regulates conflict sensitivity. The regulation of conflict-sensitivity should be a product of enhanced proactive control (Botvinick et al., 1999; Braver et al., 2007), however high levels of worry might interfere with the regulation of conflict sensitivity through cognitive depletion. This posits an association wherein adaptively switching between proactive and reactive modes of control hinges on the ability to reduce conflict sensitivity and vice versa. Optimally, approaching a high-conflict event through mobilizing proactive control should effectively decrease conflict sensitivity and negative affect (Inzlicht et al., 2015). However, if high levels of worry prevent any reduction of conflict sensitivity due to the mobilization of proactive control, as implied by findings from this study, then such a strategy, and any relation between proactive and reactive control, might hinge on affective variables that

impact conflict processing and/or interfere with top-down control.

The study demonstrated the current findings through revealing associations between proactive and reactive control on the level of neural activity. The current work discovered a moderated interaction between frontal asymmetry and N2 amplitudes, thus providing converging evidence that shifting between proactive and reactive control might require coordination among distinct neural networks. Importantly, in the present study, the relation between proactive and reactive control changes depending on levels of worry; there was no main effect between sustained proactive control and reduced reactive processing. However, it is not uncommon for a main effect to be obscured by a moderator (Aiken, et al., 1991), and often the relation between a predictor and an outcome depends on individual differences that influence the strength of that relation (Cohen & Edwards, 1989). Therefore, the theory-based notion that these two modes of cognitive control interact remains plausible, but should not necessarily be assumed given that individual-difference characteristics appear to modulate the strength and direction of this association. This offers a crucial and relatively novel component of the theoretical assumptions posited by the DMC theory; that the strength and direction of the relation between proactive and reactive control depends on levels of trait worry. Thus, the interaction between two fundamental cognitive processes, proactive and reactive control, might be contingent on emotional states, thus supporting an interaction between affect and cognition.

Limitations

The main limitation of this study is that the analyses were unable to address causality, and either direction of causality is possible. It is possible that diminished reactive processing produces enhanced proactive control when levels of worry are low. For example, at low levels of worry individuals who tend to rely on proactive control only do so because of a reduced ability to monitor for conflict. Follow-up studies will need to be performed to better address directionality. Another limitation of this study was its inability to determine the temporal nature of the relation between proactive and reactive control. Due to the nature of EEG data, any neurological processes of interest are often of such a small signal embedded within a larger expanse of noise that multiple trials are required to generate a stable measure. Therefore, analyses could not provide information concerning trial-by-trial relationships between frontal asymmetry and N2 amplitude, only differences within grand averages were analyzed.

Another limitation of the present research is the relatively small sample size, which may have limited the detection of smaller effects while increasing the chances that any observed effects are spurious. However, the current study was powerful enough to detect an interaction and the predicted effects. The current sample also consisted of typically developing college students; further research is needed to investigate if the present results extend to atypically developing or at-risk populations.

CONCLUSIONS

This research offers a novel outlook on the corresponding relation between proactive and reactive control through demonstrating an association between sustained proactive engagement and reduced reactive processing at low levels of worry. Previous work focusing on cognitive control almost exclusively examined a primarily reactive model wherein reactive processes signal for the recruitment of proactive control. The current work focused on and found evidence of a complementary proactive model of control through showing a moderated relation between the two modes of control put forth by the DMC theory. Importantly, the relation between proactive and reactive control depended on levels trait worry. When levels of worry were low, the sustained enhancement of proactive control predicted reduced conflict processing. However, when levels of worry were high this relation was non-significant, and at levels of worry comparable to those who qualify for a diagnosis of GAD, enhanced levels of proactive control marginally predicted increased conflict processing. The present pattern of effects might be due to worry influencing the allocation of cognitive resources and interfering with the ability to effectively switch between proactive and reactive strategies for task guidance.

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APPENDIX A

THE PENN STATE WORRY QUESTIONAIRE

Enter the number that best describes how typical or characteristic each item is of you, putting the number next to the item.

1	2	3	4	5
not at all typical		somewhat typical		very typical

_____ 1. If I don't have enough time to do everything I don't worry about it.

_____ 2. My worries overwhelm me.

_____ 3. I don't tend to worry about things.

_____ 4. Many situations make me worry.

_____ 5. I know I shouldn't worry about things, but I just can't help it.

_____ 6. When I am under pressure I worry a lot.

_____ 7. I am always worrying about something.

_____ 8. I find it easy to dismiss worrisome thoughts.

_____ 9. As soon as I finish one task, I start to worry about everything else I have to do.

_____ 10. I never worry about anything.

_____ 11. When there is nothing more I can do about a concern, I don't worry about it anymore.

_____ 12. I've been a worrier all my life.

_____ 13. I notice that I have been worrying about things.

_____ 14. Once I start worrying, I can't stop

_____ 15. I worry all the time