THE ROLE OF GLUCOSE AND FATIGUE IN COGNITIVE CONTROL AND SUSTAINED ATTENTION: AN ELECTROENCEPHALOGRAPHIC ASSESSMENT

by

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ABSTRACT

Cognitive control mechanisms were examined through depletion and restoration manipulations common in self-regulation literature. In this within-subjects, double-blind placebo-controlled experiment, 33 participants performed three blocks of a high-congruency version of the flanker task while their EEG signals were recorded. Cognitive fatigue was induced in participants by lengthy flanker blocks, and they received either glucose or a placebo, so we could examine the effects of metabolic restoration. Overall, there was a main effect of cognitive fatigue on flanker accuracy and on error-related event related potentials (ERPs). Participants had more accurate responses in the third block of the flanker task when they drank glucose as compared to the placebo. Additionally, the error-related negativity (ERN) waveform increased in amplitude during the third block of the flanker task when participants drank glucose. These findings suggest that there is overlap between the bodies of literature on cognitive control and self-regulation, specifically when measuring the activity in the anterior cingulate cortex during conflict paradigms.

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INTRODUCTION

Directed attention has long been recognized to have a limited capacity, which draw on limited metabolic resources (Hockey, 1977; Wickens, 1991). Researchers have provided converging evidence that when cognitive demand increases, blood glucose levels decrease (Scholey, Harper, & Kennedy, 2001). Furthermore, when participants engage in higher-order cognitive functions for an extended period of time, they tend to feel fatigued. This effect is seen across many cognitive and social domains. The present experiment identifies the specific neural substrates involved in cognitive control and addresses which neural substrates are sensitive to resource availability and cognitive fatigue.

Cognitive control is a broad term used for the cognitive functions performed to monitor performance and to manipulate goal-directed cognitive information. Cognitive control allows humans to flexibly process information, to perform multiple tasks simultaneously, and to override prepotent responses. Braver (2012) provides a framework for two mechanisms of cognitive control; proactive and reactive control are subsets within a larger cognitive control mechanism. Proactive control is required for planning for future events, whereas reactive control is used for correcting errant behavior. Proactive control is generally more effective, but also demands more energy. Reactive control, though, is more susceptible to interference effects, but is also less demanding than proactive control. Two-process models of cognitive control suggest the prefrontal

cortex (PFC) supports goal maintenance, enabling on-task behavior, whereas the anterior cingulate cortex (ACC) plays a critical role in providing negative feedback to errors and strategically adjusting off-task behavior when necessary (Braver, Gray, & Burgess, 2007; Botvinick, Cohen, & Carter, 2004; Cohen, Botvinick, & Carter, 2000). The present study utilizes ERPs signatures in combination with the experimental manipulations of cognitive fatigue and resource availability (i.e., glucose vs. placebo) to better understand the role of glucose in modulating cognitive behavior.

One way of demonstrating how the brain processes information is as though there are two systems at work. Daniel Kahneman calls these two systems "system one" and "system two" (Kahneman, 2011). System one is incredibly quick, and acts as a primary attention filter to select for the relevant stimuli in the environment to attend to. This system needs to be quick, since there are copious amounts of information in the environment, most of which are irrelevant to the task at hand. System one requires no conscious thought and operates automatically, without the need for effort or metabolic resources. This is made possible by long-term memories – the library of experiences we have built up over our lifetime. System two, on the other hand, represents the system responsible for conscious thought, problem solving, and creative and critical thinking. System two requires effort (ergo, resources) and is slow and deliberate. Another difference between system one and system two is that system two exists within working memory, so system two is limited by individual differences in working memory capacity. The limits of working memory capacity are demonstrated by the classic seven plus-or-minus two experiment (Miller, 1956).

Expertise and learning new skills come down to the automation skills of system

one, developed through the painstaking deliberate practice of system two. Most of our day-to-day life is a stroll for system two with most tasks handled automatically by system one. Just as we spend a lot of our lives lounging around, our brains spend most of their time doing the mental equivalent. For repetitive tasks we have developed automatic ways of doing things, reserving system two's limited capacity for things that really need our attention. Just as it's hard to motivate someone to get off the couch and exercise, it's hard to get system two to give its full effort. There's an appeal to doing things you already know, for a musician to play the same familiar songs that system one has already automated, that feel and sound good. To always drive with the GPS on so you never get lost, but you also never learn the way. Because thinking takes effort, it involves fighting through confusion, and for most of us that's at least somewhat unpleasant because of the level of effort involved.

It is assumed that glucose is the primary metabolic resource utilized by higher order cognitive functions (Persson, Welsh, Jonides, & Reuter-Lorenz, 2007). Researchers have also proposed that the primary resources that are used by the brain are glucose and glycogen (Gailliot et al., 2007; Kennedy & Scholey, 2000; Scholey, Harper, & Kennedy, 2001). Similar findings have been found regarding resource models of self-regulation. Self-regulation is a controlled process, which requires resources. In one experiment, Baumeister and his colleagues demonstrated that participants who ate radishes and were tempted with chocolate chip cookies before attempting to complete a series of impossible tasks quickly realized that the problems they were being presented with were unsolvable (Baumeister Bratslavsky, Muraven, & Tice, 1998). In the other condition, participants who ate the cookies before the tasks persevered on the same tasks for a significantly

longer amount of time. The self-regulatory behavior in resisting the tempting food, in the form of chocolate chip cookies, took a cognitive toll on the participants who had to engage in self-regulation, affecting their decision-making skills and their higher-order cognitive functions.

According to Baumeister and other social psychology researchers (Bandura, 1991; Carver & Scheier, 1981), self-regulation is an inhibition control process in which individuals attempt to reign in unwanted behaviors and ignore distracting cues to keep the cues from impeding a particular social-based goal. In Baumeister's model, self-regulation is akin to a muscle that tires due to several factors, one of which is ego depletion (Baumeister, Vohs, & Tice, 2007). Researchers define ego depletion as the state of depletion following the use of self-control or willpower. Baumeister and colleagues argue that the self-regulation "muscle" can also be strengthened through training and cognitive-behavioral instruction. Baumeister suggests that when we become "depleted" of our self-regulatory resources, we tend to make more errors and fail to regulate behavior properly. Baumeister and colleagues (1998) also concluded that multiple cognitive and social functions such as choice, self-regulation, active response, and volition all share common resources. Moreover, consuming glucose can replenish participants' abilities to engage in self-regulatory behaviors and can increase the amount of cognitive control employed in oppositional logic tasks (Gailliot et al., 2007; Scholey, Harper, & Kennedy, 2001). Cognitive control and self-regulation, being higher order cognitive functions, both utilize similar neural substrates and resources.

Individuals depleted of glucose (including those on low-carbohydrate diets or diabetics) show decreased attentional capacity, diminished long term memory encoding,

decreased working memory efficiency, and experience a lower mood than those who are not on a diet (D'Anci, Watts, Kanarek, & Taylor, 2009). Studies have also found that self-regulation resources are replenished in depleted participants after they consume glucose (Gailliot et al., 2007). Thus, glucose metabolism is critical for engaging in higher-order cognitive processes such as self-regulation and attentional control effectively. Depletion of self-regulatory resources occurs when individuals are either lacking resources in the form of glucose or when competing processes deplete the shared resource. Making difficult choices (Vohs et al., 2009), negative affect (Heatherton & Wagner, 2011), mental fatigue (van der Linden, Frese, & Meijman, 2003), ego depletion (Baumeister, 2002; Hagger, Wood, Stiff, & Chatzisarantis, 2010; Inzlicht & Schmeichel, 2012), excessive stress (Cohen & Spacapan, 1978; Mendl, 1999; Muraven & Baumeister, 2000; Oaten & Cheng, 2005), and directing attention (Carver & Scheier 1981; Mann & Ward, 2007; Posner & Rothbart, 1998) all take their toll on the resources needed for self-regulation.

Mental fatigue can lead to a depleted state; Van der Linden and colleagues (2002) confirmed the effects of mental fatigue on planning. The authors used the Wisconsin Card Sorting Test and the Tower of London task, which measure cognitive flexibility and planning, respectively. The results demonstrated that participants who were fatigued showed more conservative behavior on the Wisconsin Card Sorting task and had a longer planning time on the Tower of London task. The results imply that mental fatigue compromised the executive function mechanism responsible for planning and cognitive flexibility. Researchers have also found that when self-regulation is utilized, blood glucose levels decrease (Gailliot et al., 2007). Therefore, the mental fatigue that

participants experienced in the experiment had considerable effects on not only their ability to self-regulate but also on their ability to plan for future events, because self-regulation requires planning and cognitive flexibility in order to mediate behavior effectively.

Self-regulation, a process similar to cognitive control, is a higher order process that both biases and is alerted by the lower level error-monitoring network. The error-monitoring network is involved in both multitasking and self-regulation, and plays a crucial role in self-regulatory behaviors and controlled cognition (Bush, Luu, & Posner, 2000). Error recognition and performance monitoring are the foundation for successful self-regulation. Without noticing that an error was committed, a compensatory behavior cannot be executed.

The error-related negativity (ERN) and the positivity following an error (Pe) are the neurophysiological markers of error monitoring and error recognition, respectively. The ERN is a preconscious process which occurs before awareness of an error is established and peaks around 50 ms after an error response. Whereas the ERN indicates error detection and conflict monitoring, the Pe denotes error recognition and occurs anywhere from 200 to 500 ms after an error response (Falkenstein, M., Hoormann, Christ, & Hohnsbein, 2000). The ERN is a response-locked event-related potential (ERP) component associated with an erroneous response (Gehring, Coles, Meyer, & Donchin, 1990; Gehring & Fencsik, 2001; Rabbitt, 1966). Gehring, Goss, Coles, Meyer, and Donchin (1993) manipulated speed and accuracy stress during a flanker task and found that accuracy stress produced larger amplitude ERNs, while speed stress muted the effects of errors on the ERN. The authors posited that the modulation of the ERN was

due to the saliency of the error. When participants emphasized accuracy, erroneous responses were more salient and were more important to avoid. In this case, data suggest the task goal of being more accurate augmented the amplitude of the ERN. When the task emphasized speed, the avoidance of errors in order to complete the goal was no longer important and the errors produced a smaller ERN waveform. Additionally, Scheffers and Coles (2000) demonstrated that smaller ERNs were produced when the subjects did not recognize the error, while errors that were directly and overtly perceived produced the expected large ERN signatures.

Not all errors are equal; many are context driven. If there is not enough information to make a judgment, the error will not be perceived as an error. Yeung, Botvinick, and Cohen (2004) suggest that the ERN is an electrophysiological manifestation of conflict that occurs following an error as a result of continuous stimulus processing. According to the authors, the continuous posterror processing of the stimulus activates the correct response choice, while the conflict with the incorrect behavioral response produces the ERN. Researchers have used neuroimaging methodologies to identify the error detection network's neural substrates, the evidence of which suggests that the ERN originates within the anterior cingulate cortex (ACC) at around 50 ms posterror response (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Falkenstein et al., 2000; Hajcak, McDonald, & Simons, 2003; Herrmann et al., 2004; Kerns et al., 2004). A stimulus-response conflict is detected by the ACC when there is a mismatch of goal objectives and response.

The importance of the ACC's processes in human cognition, especially as related to the function of cognitive control and self-regulation, cannot be overstated. According

to Bush, Luu, and Posner (2000), the anterior cingulate is responsible for processing sensory, motor, cognitive, and emotional information, integrating input from various sources (including motivation, evaluation of error, and representations from cognitive and emotional networks), and modulating cognitive, motor, endocrine, and somatic responses.

Models of the neural substrates involved in human cognitive processes rarely overlook the ACC. In fact, the function of the ACC is one of the most important topics of research in modern cognitive neuroscience (Botvinick, Nystrom, Fissell, & Carter, 1999; Carter, Botvinick, & Cohen, 1999; Critchley, 2005; Peterson et al., 1999). Peterson and colleagues (1999) provided fMRI evidence linking performance on the Stroop task to ACC activation. The authors concluded that the ACC mediates both working memory and response selection. Converging evidence from MacDonald, Cohen, Stenger, and Carter (2000) suggests the ACC's involvement in cognitive control, a process that also heavily involves working memory. Largely, the ACC is responsible for monitoring stimuli in the environment that conflict with previously established expectations (Botvinik, Cohen, & Carter, 2000). When information in the external environment mismatches the internal cue, the ACC directs higher-order brain regions to pay more attention to the task.

Miller, Watson, and Strayer (2012) established that different WMC groups have notably differing electrophysiological responses to error trials, namely differences within the ACC, marked by the amplitude of ERN signatures. The authors conducted a study comparing individuals in upper and lower quartiles of the OSPAN (Unsworth, Heitz, Schrock, & Engle, 2005) distribution of performance on a spatial variant of the Simon task (Castel, Balota, Hutchinson, Logan, & Yap, 2007). Miller et al. (2012) concluded

that high WMC individuals had a more robust error detection network, marked by greater ERN amplitude. Because working memory influences the error-monitoring network, holding task goals in working memory must be vital for error detection, self-regulation, and cognitive control.

The Pe, or positivity following an error, originates within the posterior cingulate cortex (PCC; Falkenstein et al., 2000; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). Located behind the ACC in the limbic system, the PCC plays a central role in the default mode network (Fransson & Marrelec, 2008). The default mode network[1] has been shown to be responsible for episodic memory, autobiographical information, theory of mind, and social evaluations (Andrews-Hanna, 2012). According to the literature, the PCC is the culprit that drains cognitive resources in order to perform conscious thinking about the self and utilizes self-restraint in the form of self-regulation.

The Pe is an ERP component believed to be a manifestation of conscious awareness of an error. Overbeek et al. (2005) suggest three theories as to the function of the Pe: the affective-processing hypothesis, the behavior-adaptation hypothesis, and the error-awareness hypothesis. The affective-processing hypothesis suggests that the function of the Pe is to provide an emotional influence in making an erroneous response, or in other words, participants become emotionally upset by errors, allowing for a highly-salient case of behavioral correction. Furthermore, the affective-processing hypothesis suggest that the brain is using affect as a form of data to drive the salience of an error. Therefore, according to this hypothesis, the Pe is the brain's electrophysiological

[1] For more information on the default mode network, see Raichle, MacLeod, Snyder, Powers, Gusnard, & Schulman, 2001; Greicius, Krasnow, Reiss, & Menon, 2003; Hayden, Smith, & Platt, 2009.

manifestation of an emotional response to an error.

The behavior-adaptation hypothesis explains that the Pe provides a cue to the higher-order regions of the brain to modify the current behavior in order to stay on task. The ACC relays the error information to the PCC, which then biases the processing of the prefrontal cortex in order to stay on task. Deviations from the goal (i.e., errors) will create a larger Pe signal and result in a controlled reappropriation of attention back toward the task goal. This hypothesis is of particular interest because it supports the notion that cognitive self-regulation can be directly measured by the amplitude of the Pe. By measuring the degree to which the goal has deviated and measuring posterror slowing on the subsequent trial, researchers can quantify the amount of cognitive control and self-regulation exerted in order to reassert a goal-oriented state. The PCC unites the two seemingly disparate terms (cognitive control and self-regulation) in a quantifiable way.

The error-awareness hypothesis is much simpler than the previous two hypotheses. The error-awareness hypothesis suggests that the Pe reflects the participants' subjective awareness of the error they have committed. The Pe is responsible for one or all three of these hypotheses but is more likely that a combination of all three contribute to the generation of the Pe waveform. Together, the ACC and the PCC create an error-monitoring network associated with detecting and correcting goal-inconsistent behavior, which acts to update behavioral goals under the supervision of the prefrontal cortex (Botvinick, Cohen, & Carter, 2004).

Often, social and cognitive psychologists refer to comparable control mechanisms in their research, such as self-regulation and cognitive control, respectively. For instance, both fields discuss the importance of control in everyday behavior and how these self-

regulatory processes can be measured and predicted. This manuscript provides evidence for the resources used by cognitive control mechanisms under manipulations commonly used in self-regulation research, thereby bolstering the body of literature on cognitive control and the metabolic resources used in higher-order cognitive processes.

Fatigue and glucose manipulations typically are used in self-regulation literature, whereas error-related ERPs are used as dependent variables from the cognitive control literature. The present research brings these two ideas together to show that the manipulations have an effect in terms of self-regulatory behavior which manifests in the cognitive control and ERP components associated with cognitive control. Our goal is to determine if these two bodies of literature align. In order to address this research question, we induced cognitive fatigue, then gave participants either glucose or placebo drinks. We focused on the ERN and the Pe, ERP components specifically dealing with detecting and correcting errant behavior as part of the cognitive control network. Our prediction is that if there is a linkage between the two bodies of literature, then the self-regulatory manipulations (i.e., fatigue and glucose) should modulate the ERP components associated with cognitive control. Specifically, both the ERN and Pe will be modulated due to cognitive fatigue and glucose manipulations. We predict that both waveforms will decrease in amplitude due to cognitive fatigue, and that both will increase in amplitude when participants are given glucose. Finally, we predict that behavioral performance will diminish as cognitive fatigue sets in, but participants who receive glucose will have improved performance after glucose is metabolized and resources are made available.

METHODS

Participants

Thirty-three participants from the University of Utah were recruited to take part in this study. Two participants were excluded from analysis because they did not make enough errors to analyze error-related ERPs, and one participant dropped out of the study after the first session. The remaining 30 participants (16 female, $\bar{x}$=23.4 years old) were included in all subsequent analyses. Using a within-subjects design, participants were instructed to schedule two appointments anywhere from 2 days to 2 weeks apart. Participants who were left-handed, diagnosed with hypoglycemia, hyperglycemia, Type 1 or Type 2 diabetes, any neurological disorders or recent head trauma, or above the age of 45 were excluded from participation in the study. All participants provided informed consent and received course credit upon completion of the experiment. IRB approval for this experiment was granted by the University of Utah.

Materials and Procedures

Session One: In the first session, participants had an electrode cap placed on their scalps and electrodes applied to their faces in order to record electroencephalographic (EEG) and electrooculargraphic (EOG) signals. After situating participants with the electrodes, we tested participants' baseline blood glucose levels. Participants were instructed to fast and refrain from drinking any sugary or caffeinated drinks for at least 4

hours before their appointments. A questionnaire verified that participants did not eat for at least 4 hours before the study, and that they had a normal amount sleep the previous night. Having participants fast ensured that the participants were at their individual baseline blood glucose levels (Donohoe & Benton, 2000). We expected there to be fluctuations among individual baseline blood glucose levels, and the initial blood glucose measurement was used to statistically control for this variance. Blood glucose levels were measured by piercing the skin on the participant's finger using a SafeLan Pro lancing device. Approximately 0.4 μL of blood was collected using OneTouch Verio IQ test strips and analyzed using the OneTouch Verio IQ blood glucose analyzer.

After having collected the first blood sample for glucose measurements, participants were tested individually on a high-congruency version of the Eriksen and Eriksen (1974) flanker task created in E-prime 2.0. We instructed participants to respond based on the identity of the centrally presented letter in a series of five letter horizontal arrays. There were two types of stimuli: Congruent and incongruent. A congruent stimulus consisted of all identical letters (e.g., SSSSS or HHHHH) and an incongruent stimulus consisted of "flanking" letters that were associated with the opposite response (e.g., SSHSS or HHSHH). Each stimulus was preceded by a fixation cross, presented for 200 ms in the center of the display followed by a blank screen for 100 ms. Stimuli remained in view until the participant responded or 2000 ms had elapsed. The 5-letter horizontal array subtended 2.60 degrees of visual angle.

Participants first completed a practice block of 50 trials to familiarize themselves with the task. Average response time and accuracy statistics were presented to the participant at the end of the practice block as feedback. If participants finished the

practice task with accuracy lower than 75%, additional instructions were provided, and the participants attempted the practice again. Participants were instructed to respond to the target letter as quickly and as accurately as possible with the "Z" and "/" keys on a keyboard with their left and right index fingers, respectively. The mapping of response keys to the central letter identity was counterbalanced across participants. Once the practice block was completed and the participants felt comfortable with the task, participants performed one block of 800 trials of the flanker task.

After the first block of the flanker task, participants completed an automated version of the OSPAN task (Unsworth, Heitz, Schrock, & Engle, 2005) to reinforce the fatigue manipulation of the experiment. During the OSPAN task, participants were presented with simple math problems and the participant reported the veracity of the statement as either "true" or "false" (e.g., (8/2)+2=12…"False"). Following each math problem, a letter was presented for later recall. After sets of 3 to 7 math/letter pairs, the participants were prompted to recall the letters in the order in which they were presented. All OSPAN stimuli were presented on a computer screen and responses were made with a computer mouse.[2]

After participants completed the OSPAN task, another blood sample was taken to measure blood glucose levels. The purpose of this measurement was to determine how much glucose participants metabolized during the fatigue portion of the study. After blood glucose levels were recorded, participants consumed either 12 fluid ounces of lemon-lime Gatorade containing 21 grams of sugar (glucose), or 12 fluid ounces of a placebo version of lemon-lime Gatorade that did not contain glucose. Gatorade

[2] The OSPAN task was used as a fatigue manipulation and was not used in the data analysis.

manufactures this placebo for the purposes of blood-glucose experiments in their research and development department (Baker, Rollo, Stein, & Jeukendrup, 2015). The beverages were kept chilled and were drunk through a straw to minimize the taste profile, which reduced the chance that the participant could guess which beverage they were drinking. Random assignment to either the glucose condition or the placebo condition during the first session was determined by a coin flip, and the drinks were administered using a double-blind procedure. Fourteen participants drank the Gatorade with glucose during their first session, and 16 participants drank the placebo Gatorade during their first session.

While the glucose was being metabolized, participants completed another block of the flanker task. Prior studies have indicated that it takes around 15 minutes for liquid glucose to be metabolized and become available for cerebral functions when at baseline blood-glucose levels (Reivich et al., 1985). This block of the flanker task allowed for the glucose to be fully metabolized and to be available to use as a metabolic resource. Following the second block of the flanker task, participants had their blood glucose levels tested again. Once blood glucose levels were tested, participants completed the third and final block of the flanker task, followed by a final blood glucose level test.

Session Two: In session two, the same 30 participants completed the experiment 2 days to 2 weeks after the first session using the same experimental protocol outlined in session one. However, during the second session, the glucose conditions were switched. For example, if a participant was randomly assigned to drink the placebo Gatorade during the first session, they then consumed the Gatorade containing glucose during the second session, and vice versa. Upon completion of the second session of this experiment, all

participants were debriefed and received research credit for participating.

Design

Table 1 provides an overview of the protocol and design of the experiment. The study utilized a 2 (glucose vs. placebo condition) by 3 (flanker blocks) double blind placebo controlled within-subjects design. All participants completed three blocks of the flanker task per session, totaling six blocks per participant over the course of the two sessions. Each block consisted of 800 randomized trials, resulting in 2400 trials per participant per session. The congruent stimuli (e.g., HHHHH) comprised 75% of the trials, while the incongruent stimuli (e.g., SSHSS) comprised 25% of the trials, thereby creating a high-congruency variant of the paradigm. After every block of 800 trials, the program presented the participants with feedback on their average accuracy and response time for that block.

ERP Recording

For both sessions, participants had electrodes applied to their scalps and faces to record electroencephalographic (EEG) and electrooculargraphic (EOG) signals. For EEG/ERP data collection, we utilized a 36-channel SynAmps cap manufactured by Compumedics Neuroscan and placed the cap according to the International 10-20 placement guidelines (Jasper, 1958). We used a Compumedics Neuroscan NuAmps amplifier to digitize the signal for computer-based recording and processing. The amplifier sampled EOG and EEG signals at a rate of 250 Hz with a notch filter at 60 Hz and a low pass filter of 50 Hz. Research assistants cleaned participants' skin using a light

exfoliating gel on the sites where they applied 10-mm diameter Ag/AgCl biopotential electrodes external eye and mastoid electrodes. Mastoid and facial electrodes were applied using adhesive electrode collars and filled with saline-based gel. Electrodes in the cap were filled with sponges, and a saline solution was inserted into the electrodes until the sponge expanded and contacted the participant's scalp. Saline was applied until impedances on all electrodes was below 10 kOhms. HEOG and VEOG artifacts were corrected offline using Neuroscan's Scan 4.5 software. Trials with artifacts in the EEG signals were not included in the subsequent analysis (this excluded less than 4% of the data). Additionally, we employed an a-priori exclusion criterion for each participant having five or fewer accepted error trials per flanker block which, as mentioned above, resulted in the exclusion of two participants from the analysis. Error response events were epoched from -500 ms before the event to 1000 ms post event. A band pass zero phase shift filter from 0.1 Hz to 30 Hz was applied before rejecting artifacts that exceeded above 70 and below -70 microvolts. We created a final waveform by averaging the remaining accepted trials.

Table 1 Experimental protocol overview

		0h:00m	**0h:20m**	**0h:40m**	**0h:40m**	**0h:45m**	**1h:05m**	**1h:05m**	**1h:25m**
Glucose Condition	Blood Draw	Flanker Block 1	OSPAN	Blood Draw	Glucose Consumed	Flanker Block 2	Blood Draw	Flanker Block 3	Blood Draw
Placebo Condition	Blood Draw	Flanker Block 1	OSPAN	Blood Draw	Placebo Consumed	Flanker Block 2	Blood Draw	Flanker Block 3	Blood Draw

RESULTS

Blood Glucose Levels

Figure 1 shows the blood glucose levels throughout the experiment. When participants consume glucose, a spike in blood glucose is evident for the third blood draw. Blood glucose levels were analyzed using a 2 (condition) by 4 (time course) repeated measures ANOVA. The analysis revealed a main effect of time on blood glucose levels $F(3,87)= 56.69$ $p <0.01$, $\eta^2=0.66$. As expected, when participants drink glucose, their blood levels increased, and when they drank the placebo, their blood glucose was unchanged. The analysis also revealed a main effect of block on blood glucose levels $F(3,87)= 39.13$ $p <0.01$, $\eta^2=0.57$, and a significant interaction of condition by block on blood glucose $F(3,87)= 85.31$ $p <0.01$, $\eta^2=0.75$.

Behavioral Data

Perusal of Figures 2 and 3 indicate that both conditions had similar behavioral outcomes on the flanker task. Cumulative accuracy functions (CAFs) were calculated as a way of visualizing the behavioral data by plotting accuracy as a function of RT. CAFs were created for the glucose condition (see Figure 3) and the placebo condition (see Figure 2) for each of the three blocks of the flanker task by creating Vincentized deciles for participants in each of the experimental conditions. The CAFs reflect the average accuracy at each decile as a function of the average RT associated with that condition

(glucose vs. placebo), flanker block, and trial type (congruent vs. incongruent). Figure 4 shows the behavioral equivalency of the glucose and placebo groups during the third block of the flanker task.

The RT and accuracy data from the flanker task were analyzed using a 2 (glucose condition) by 3 (flanker block) by 2 (congruent vs. incongruent trial type) repeated measures ANOVA. Response time (RT) and accuracy for congruent and incongruent trial types are displayed in Figures 5 and 6, respectively. We considered trials outside the range of 200 to 2000 ms as outlier trials, and they were excluded from analysis. Additionally, trials where participants responded three standard deviations above or below their mean RT for that condition were also excluded from further analysis (less than 2% of trials). There was no main effect of condition on RT $F(1,28)= 0.002$, $p <0.97$, $\eta^2=0.00$, nor was there a main effect of block on RT $F(2,28)= 2.83$, $p <0.08$, $\eta^2=0.09$. This indicates that RT was not modulated by cognitive fatigue, nor was the glucose administration directly related to RT. However, there was a main effect of trial type on RT. Participants had significantly slower responses for incongruent trials than for congruent trials $F(1,28)= 174.25$, $p <0.00$, $\eta^2=0.68$. No other effects or interactions were significant for RT.

As shown in Figure 6, there was a main effect of block on flanker accuracy $F(2,28)= 7.71$, $p <0.05$, $\eta^2=0.35$. As the experiment progressed and fatigue set in, participants became less accurate. Participants were also less accurate on incongruent trials $F(2,28)=61.63$, $p <0.00$, $\eta^2=0.67$. There was no main effect of the glucose manipulation on flanker task accuracy.

Posterror Slowing

In Figure 7, posterror slowing was calculated on a participant-by-participant basis by subtracting the average response time of correct trials following a correct response from the average response time of correct trials following an error response. We analyzed posterror slowing using a 2 (condition) by 3 (block) repeated measures ANOVA. The analysis revealed a main effect of block $F(2,58)= 5.73$, $p <0.05$, $\eta^2=0.17$, but not of condition on posterror slowing $F(2,58)= 0.14$, $p=0.71$, $\eta^2=0.01$. These results indicate that posterror slowing is sensitive to fatigue manipulations, but not to glucose manipulations.

Error-Related Event Potentials

Figure 8 presents the average response-locked ERPs recorded at electrode site Cz for trials during the first block of the flanker task. ERPs for the second block are displayed in Figure 9, and ERPs for the third block are displayed in Figure 10. In each figure, the glucose and placebo conditions for error trials (top panel), correct trials (middle panel), and the difference waveforms (bottom panel) are plotted. For trials with an error, there is an initial negative component in the ERP that peaks at 75 ms (the ERN), followed by a positive component that peaks at 325 ms (the Pe).

Error-Related Negativity

The area under the curve measurements for the ERN is presented in Figure 11. To analyze the ERN, response-locked averages were generated for each condition, block, and participant. Averages were baseline corrected by subtracting the average value

between -500 ms and 0 ms from the waveforms. Difference waveforms were calculated from the average correct signals subtracted from the average error ERPs, and the difference between these two waveforms was analyzed. We quantified the ERN by integrating the area between 0 and 150 ms (see Coleman, Watson, & Strayer, 2018). Inferential statistics were generated using a 2 (condition) by 3 (block) repeated measures ANOVA. There was a significant interaction of condition by block $F(2,58)= 4.14$, $p <0.05$, $\eta^2=0.11$. Participants who consumed glucose had an increased ERN amplitude in the third block as compared to those who received the placebo. No other effects were significant.

Positivity Following an Error

Figure 12 presents the peak-to-peak measurement for both placebo and glucose conditions. To analyze the Pe, response-locked averages were generated for each condition, block, and participant. Averages were corrected by subtracting the average value at the peak of the ERN from the waveforms.[3] Difference waveforms were calculated from the average correct signals subtracted from the average error ERPs, and the difference between these two waveforms was analyzed. A 2 (condition) by 3 (block) repeated measures ANOVA revealed a main effect of block on Pe amplitude $F(2,28)= 6.87$, $p <0.05$, $\eta^2=0.19$, but no main effect of condition nor a significant interaction. These results indicate that there was no effect of glucose on the Pe, and that the Pe is

[3] The Pe waveforms was also analyzed by integrating the area between 150 and 700 ms (relative to a 150 ms baseline). A 2 (condition) by 3 (block) repeated measures ANOVA revealed a significant effect of block, $F(2,28)= 3.83$, $p =0.04$, $\eta^2=0.21$; however, neither the main effect of condition, nor the interaction was significant (all p's <.25).

susceptible to cognitive fatigue. The total number of errors per participant per block are listed in Table 3.

Table 2 Overall statistics

		df	*F* ratio	Significance
RT	Block	2,28	2.83	ns
	Condition	1,28	0.01	ns
	Block x Condition	2,58	0.83	ns
ACC	Block	2,28	7.71	*
	Condition	1,28	4.13	*
	Block x Condition	2,58	0.14	ns
PES	Block	2,28	5.73	**
	Condition	1,28	0.14	ns
	Block x Condition	2,58	1.85	ns
ERN	Block	2,28	2.87	ns
	Condition	1,28	1.47	ns
	Block x Condition	2,58	4.14	*
Pe	Block	2,28	6.87	**
	Condition	1,28	1.16	ns
	Block x Condition	2,58	0.94	ns

Table 2 provides the statistics for response time (RT), accuracy (ACC), posterror slowing (PES), error related negativity (ERN), and positivity following an error (Pe) measurements. Significance levels marked with a single star (*) are statistically significant at the $p<0.05$ level, p values marked with two stars (**) are statistically significant at the $p<0.01$ level, and "ns" refers to effects that were not statistically significant.

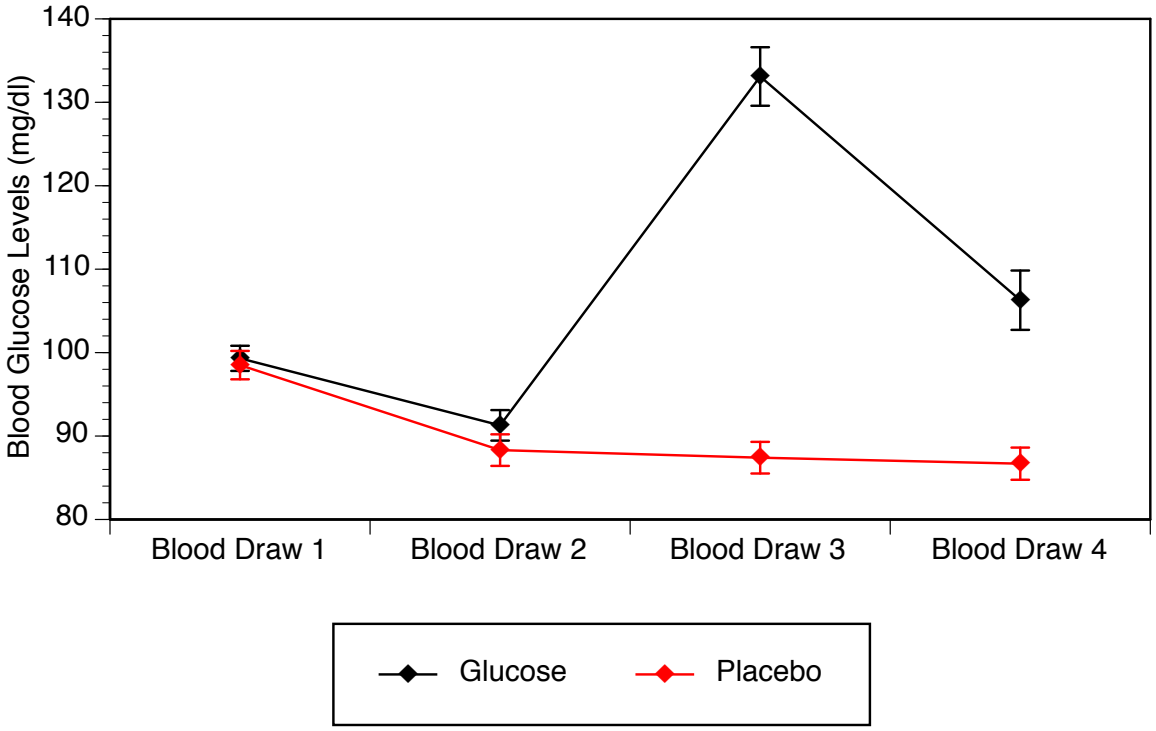

Figure 1. Average blood glucose levels in mg/dl for both glucose and placebo conditions across the four blood draw time points. Error bars represent the standard error of the mean.

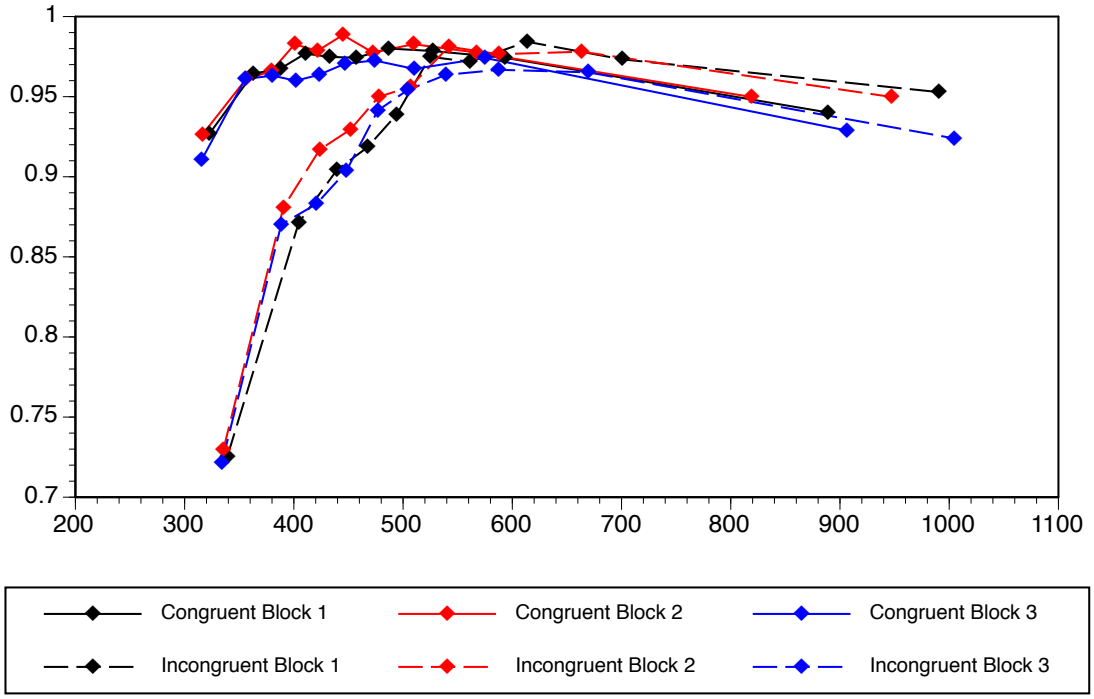

Figure 2. Cumulative accuracy functions for placebo condition.

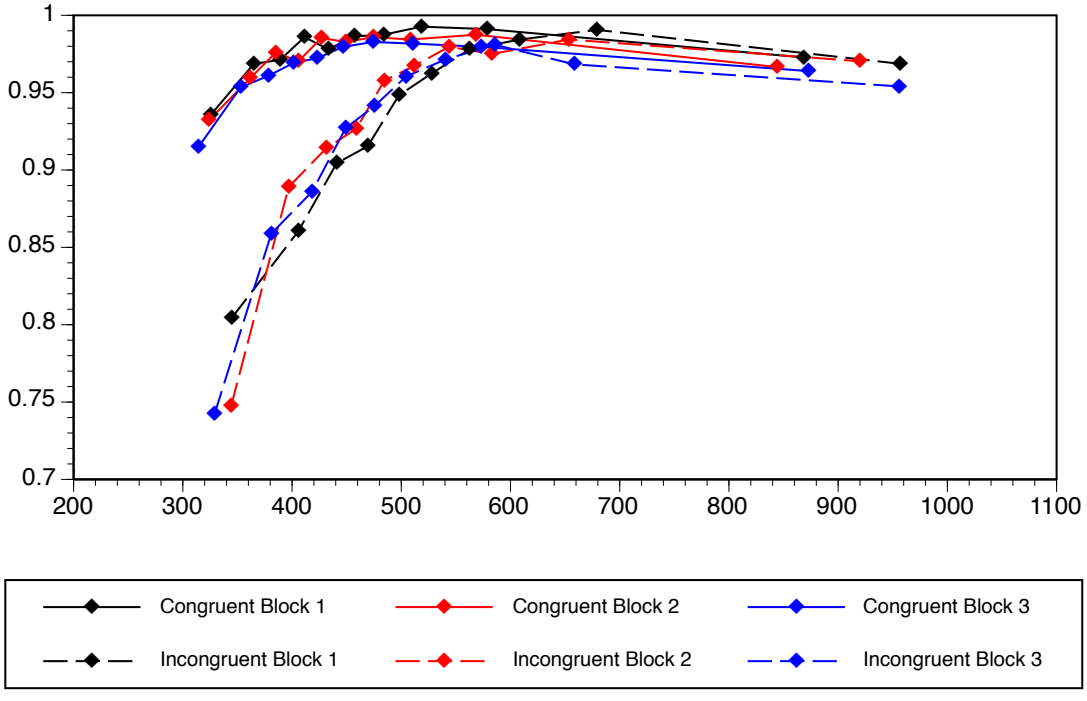

Figure 3. Cumulative accuracy functions for glucose condition.

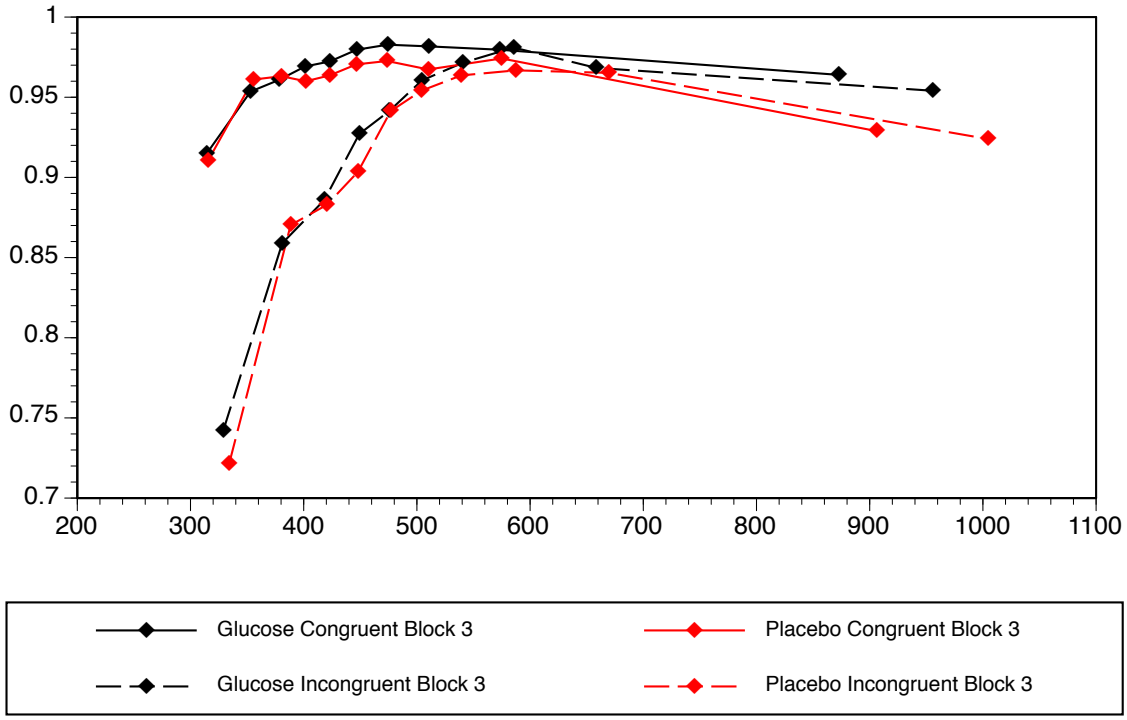

Figure 4. Cumulative accuracy function for block 3.

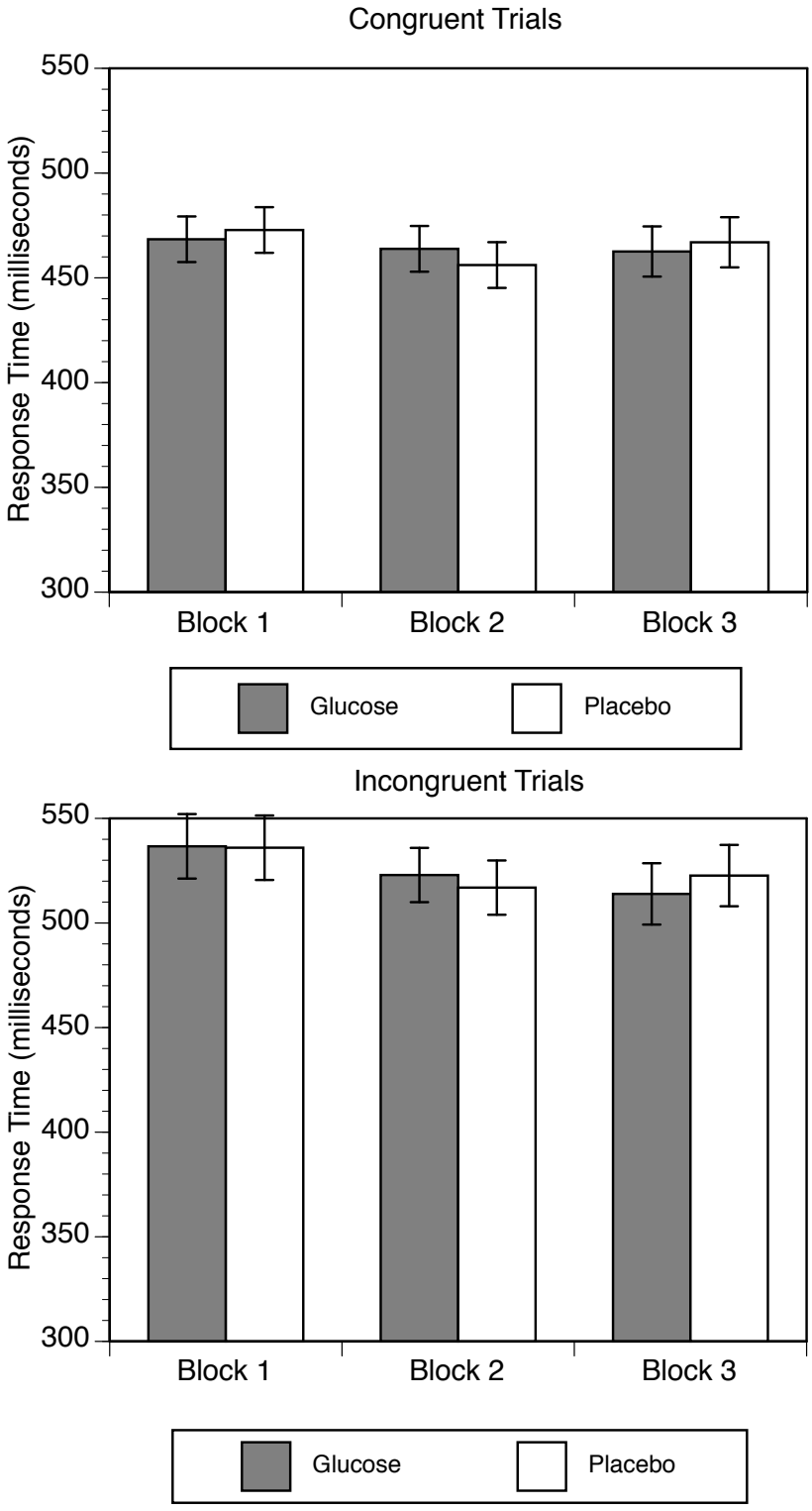

Figure 5. Mean response times in milliseconds for both glucose and placebo conditions for the three flanker blocks. Error bars represent the standard error of the mean.

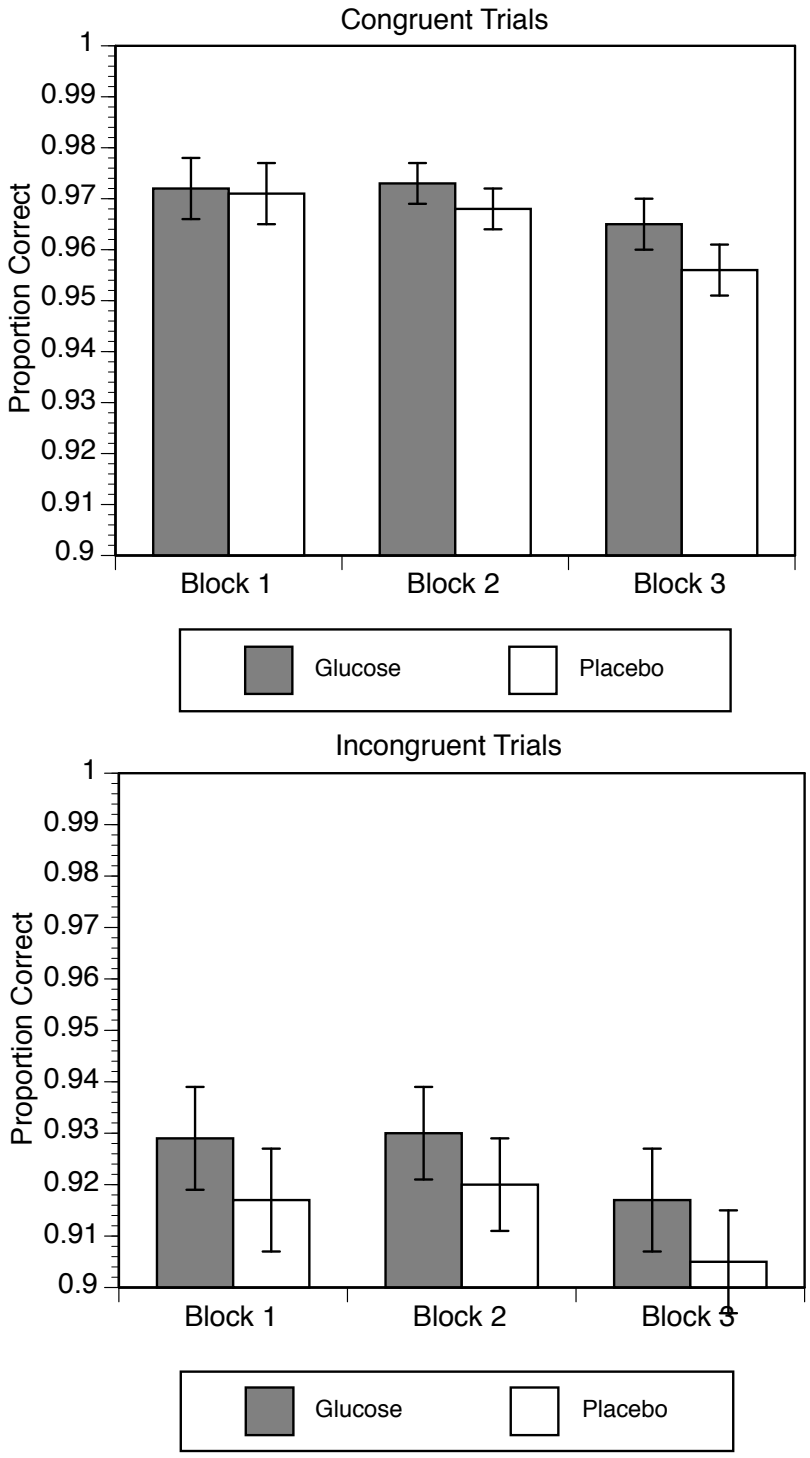

Figure 6. Mean accuracy for both glucose and placebo conditions for the three flanker blocks. Error bars represent the standard error of the mean.

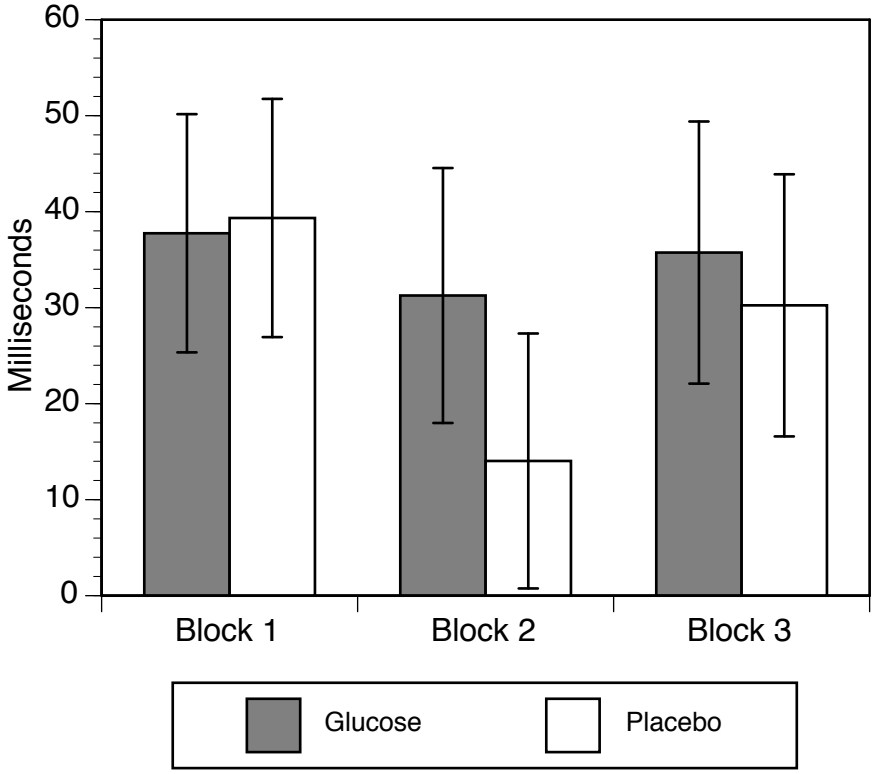

Figure 7. Posterror slowing in milliseconds for both glucose and placebo conditions for the three flanker blocks. Error bars represent the standard error of the mean.

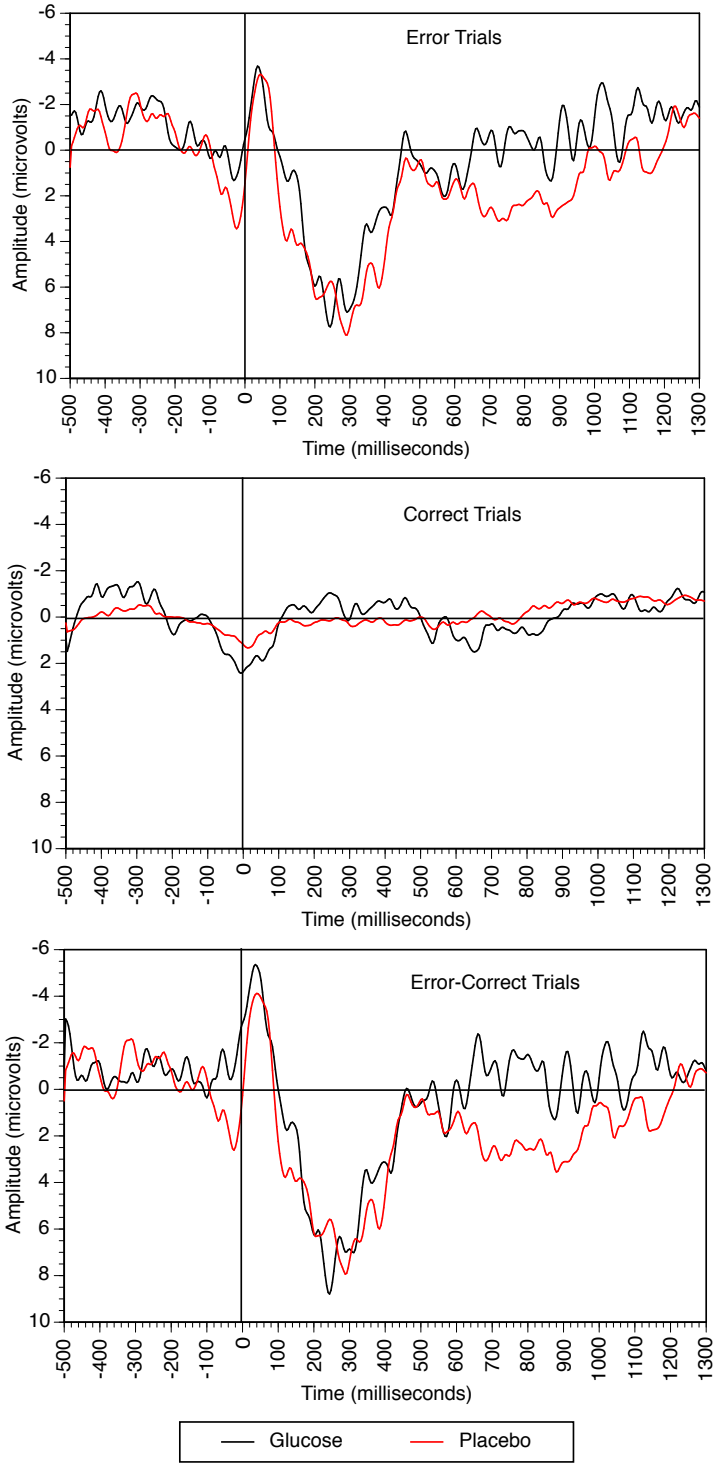

Figure 8. Block 1 ERPs consist of error trials, correct trials, and error-correct trials, respectively, for the first block of the flanker task. Waveforms are response-locked averages for electrode site Cz.

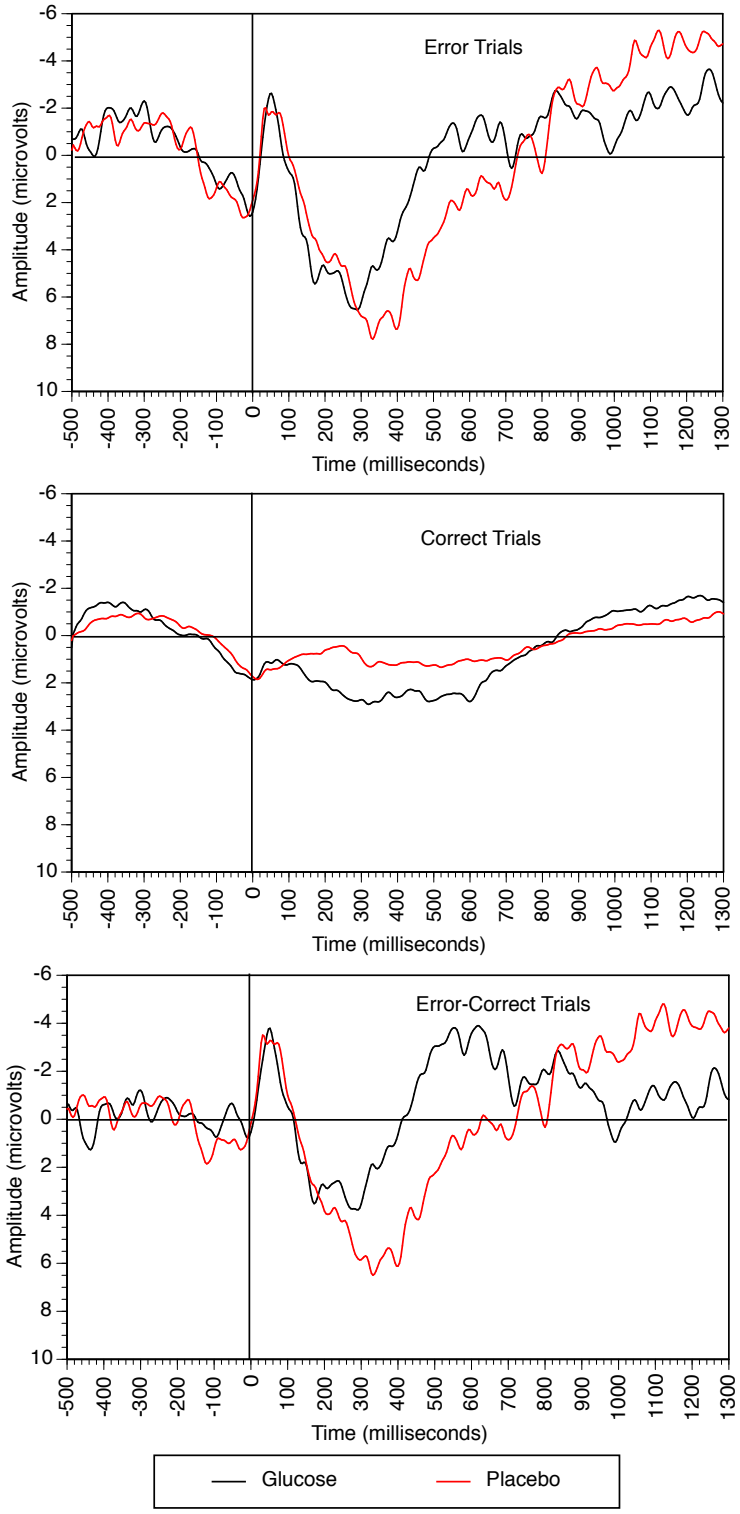

Figure 9. Block 2 ERPs consist of error trials, correct trials, and error-correct trials, respectively, for the second block of the flanker task. Waveforms are response-locked averages for electrode site Cz.

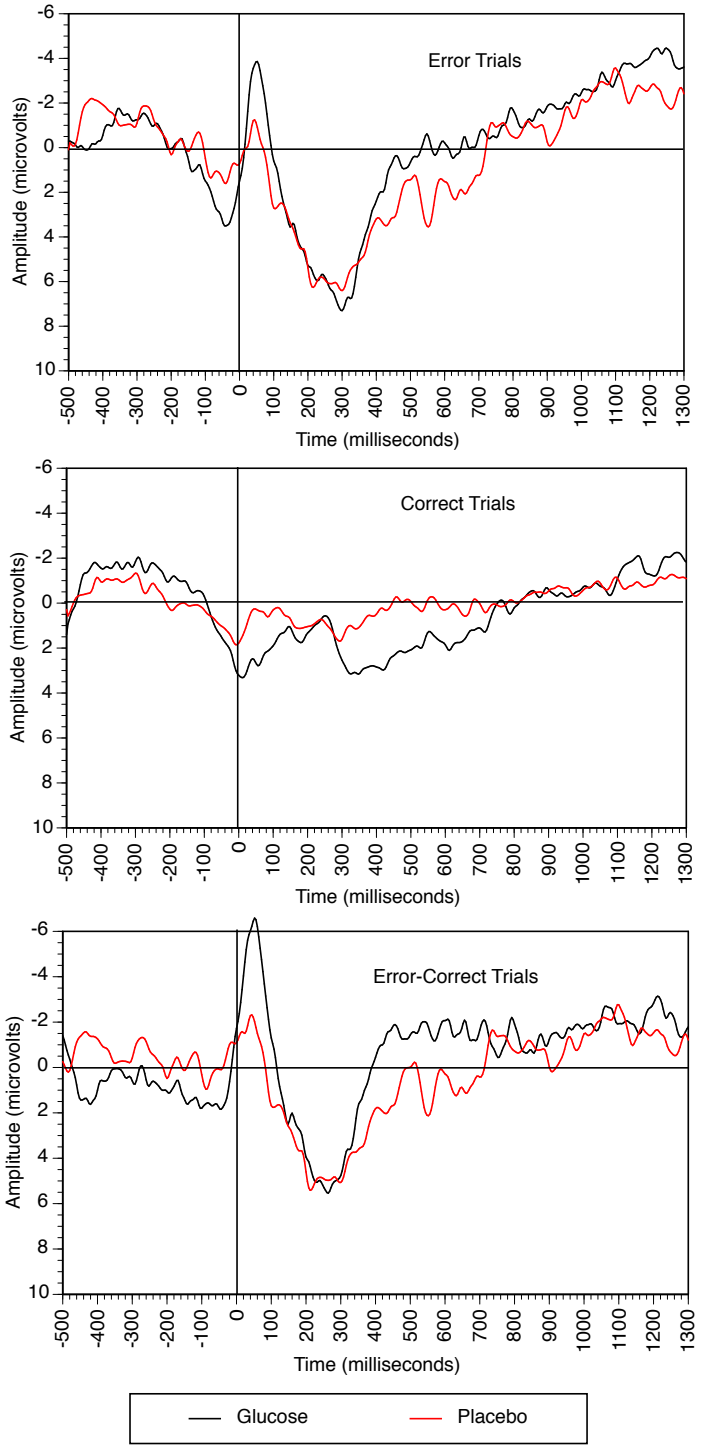

Figure 10. Block 3 ERPs consist of error trials, correct trials, and error-correct trials, respectively, for the third block of the flanker task. Waveforms are response-locked averages for electrode site Cz.

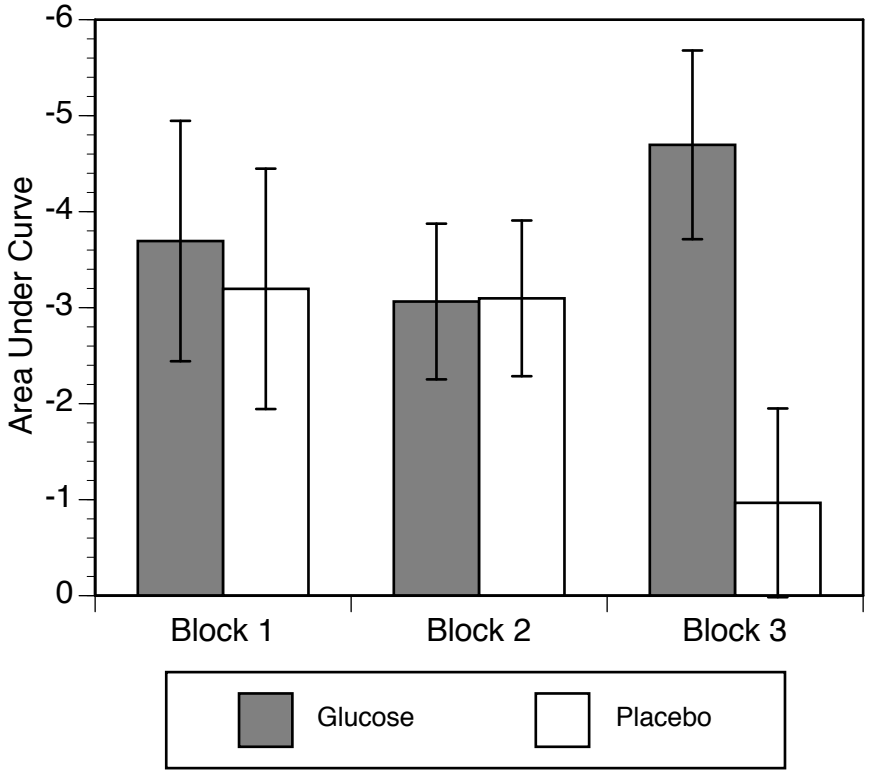

Figure 11. ERN area under the curve averages for both glucose and placebo conditions for the three flanker blocks. Error bars represent the standard error of the mean.

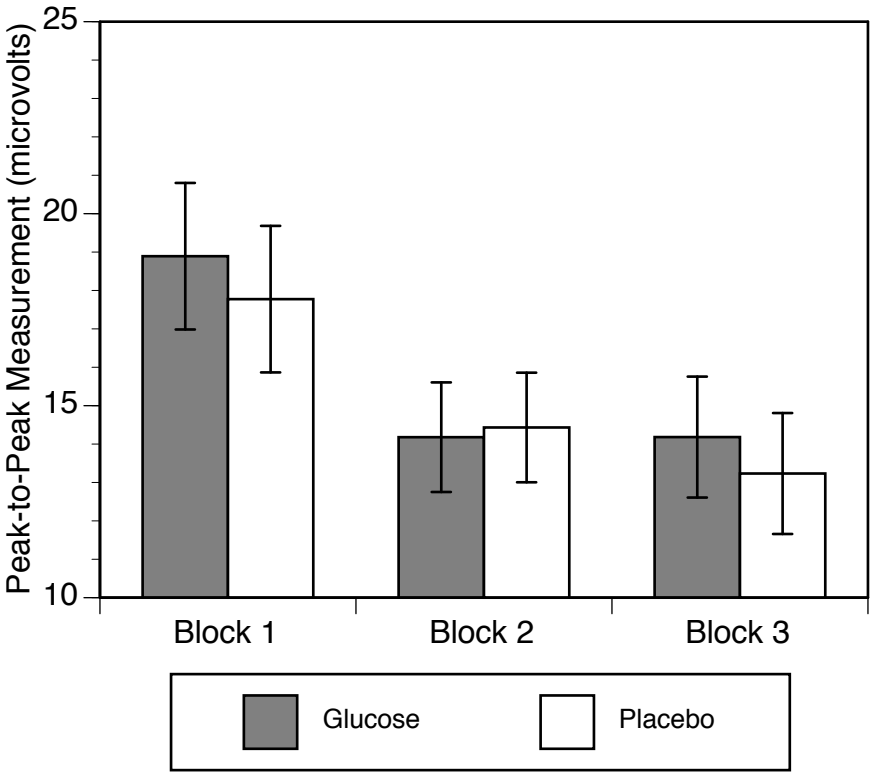

Figure 12. Peak to peak measurement averages for both glucose and placebo conditions for the three flanker blocks. Error bars represent the standard error of the mean.

Table 3. Errors per participant per block

Participant	Glucose Block 1	Glucose Block 2	Glucose Block 3	Placebo Block 1	Placebo Block 2	Placebo Block 3
01	53	49	65	56	59	45
02	103	103	103	84	106	103
03	13	16	22	16	15	21
04	35	39	63	92	26	26
05	26	32	36	33	28	110
06	10	15	25	9	11	24
07	40	29	39	37	28	61
08	19	33	57	11	8	18
09	9	10	13	21	12	18
10	6	37	29	24	30	36
11	26	24	42	25	35	45
12	5	8	14	11	12	13
13	23	18	35	19	17	26
14	57	43	76	101	104	97
15	59	69	63	40	46	56
16	12	21	28	20	25	29
17	12	12	13	12	26	19
18	31	38	37	43	46	45
19	44	31	59	63	49	107
20	18	24	50	45	45	43
21	52	34	33	31	37	35
22	25	43	40	21	28	31
23	28	28	54	66	31	68
24	37	35	42	14	46	38
25	8	6	5	11	9	12
26	48	55	58	43	42	43
27	6	10	7	14	27	25
28	25	50	7	24	41	41
29	26	51	50	32	34	30
30	27	21	18	9	9	10

DISCUSSION

The purpose of this research was to explore the relationship between self-regulation and cognitive control. In the self-regulation literature, manipulations of glucose and fatigue are often used to measure changes in overt behavior (Baumeister et al., 1998; Scholey, Harper, & Kennedy, 2001). In the cognitive control literature, ERPs recorded in conflict paradigms are often used to determine the mechanisms involved in cognitive control (e.g., Coleman, Watson, & Strayer, 2018). The current research merged the two literatures by examining error-related ERPs elicited in a conflict paradigm while using the glucose and fatigue manipulations to explore the overlap between the two approaches. Utilizing a double-blind placebo controlled experimental design, we determined the impact of fatigue and glucose on ERP components associated with cognitive control affording inferences on the underlying neural substrates.

Figure 13 provides a representation of the observed effects of this experiment. There were significant effects of glucose and fatigue on the ERN. By contrast, fatigue modulated the Pe, but glucose did not. The dissociative effect of glucose on the ERN and the Pe helps to link the cognitive control and self-regulation literatures. Given what is known about the neurophysiology associated with error detection, the ERN, and the Pe (Falkenstein et al., 2000; Gehring, Coles, Meyer, & Donchin, 1990; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005; Rabbitt, 1966), the current data suggest that the ACC utilizes glucose as a metabolite when available, whereas the PCC does not take

advantage of this resource (at least within the timing of the current study).

Based upon the overlap in the self-regulation and cognitive control literatures, we hypothesized that the effects of glucose and fatigue would be observed in both the ERN and Pe. It would appear that the link between the literatures is through the ERN, an error-related ERP component thought to be generated in the ACC. The ERN is thought to be a preconscious waveform initiated by a mismatch in stimulus expectation and behavioral response (Gehring, Coles, Meyer, & Donchin, 1990; Rabbitt, 1966). This waveform is associated with error detection after an erroneous response is committed. The interaction of fatigue and glucose on the ERN suggests that the behavioral outcomes observed in self-regulation literature are due in part to the metabolic changes in the ACC.

The Pe, an error-related ERP component thought to be generated in the PCC, was not sensitive to manipulations of blood glucose. As such, the link between this component and the literature on self-regulation and cognitive control is more tenuous. The Pe is thought to signal either a change in behavior, an affective response, or the initial processing of error saliency (Overbeek et al., 2005). However, we did not find any evidence that supports any of these interpretations. The Pe in this experiment was sensitive to the fatigue manipulation, suggesting that the Pe is not a direct measure for self-regulatory processing.

Our findings are consentient with the notion that the ERN is a preconscious signal in that we found no behavioral changes in terms of response time, accuracy, or posterror slowing between placebo and glucose groups. We did, however, see an increase in the ERN. Interestingly, what this may suggest is that self-regulation processes are also preconscious or unconscious. This explanation aligns with the results from self-regulation

experiments; there is often an overt change in behavior due to a subtle glucose or fatigue manipulation. Participants are often unaware of the behavioral changes in these experiments. For example, when participants ate the cookies in the Baumeister et al. experiment (1998) they were not aware that they were spending more time on the difficult problems than the group that ate radishes. These overt behavioral changes were due to preconscious mechanism being manipulated by glucose, much like we saw in this experiment.

With respect to the initial research question, the ERN provides a link between the cognitive control and self-regulation literatures. Manipulations commonly used in the self-regulation literature modulate the ERN, a component associated with error regulation in the cognitive control literature. This implicates the ACC as the common neural link. The link between the literatures is more tenuous with the Pe, given the lack of changes in posterror slowing associated with the glucose manipulation. PET and fMRI studies on cerebral blood flow provide evidence that blood flow to the PCC is increased when the default mode is active (Raichle et al., 2001). During visual attention tasks, however, the blood flow to the ACC increases (Paus, Koski, Caramanos, & Westbury, 1998; Shulman et al., 1997). When attentional control networks are active, there appears to be decreased blood flow to the PCC. This literature helps to explain the effect of glucose on the ERN but not the Pe. By engaging the ACC in the flanker task, participants may have increased blood flow and higher levels of cerebral metabolism in this brain region. Conversely, because blood flow to the PCC was reduced, the glucose manipulation was ineffective.

One future direction could be to use a more difficult task using the same manipulations in order to observe behavioral effects of glucose and fatigue. The flanker

task we used in this experiment produced reliable ERPs, but as a ceiling effect was evident. Another future experiment could use a protocol that had a more dynamic time interval for blood glucose testing. A final future direction could look at other mechanisms thought to have restorative effects. For example, prior studies have shown that creative thinking, working memory, and problem-solving skills all improve after spending time in a natural environment without technology for an extended period of time (Atchley, Strayer, & Atchley, 2012; Berman, Jonides, & Kaplan, 2008; Kaplan, 1995). The mechanism for the restorative effect of nature is thought to be the activating of the default mode network. When the default mode network is active, it gives the prefrontal cortex time to rest which decreases cerebral metabolism and increases the availability of glucose and glycogen. Based on the results of this study, glucose has a restorative effect on the ERN. It would be theoretically informative to see if the same results are obtained from a natural environment as with a restorative chemical (i.e., glucose).

In conclusion, this experiment demonstrated that cognitive control mechanisms are indeed influenced by the depletion and resource manipulations commonly used in the self-regulation literature. This is the first study that utilizes the fatigue and glucose manipulations to dissociate the functions of the ERN and Pe, and links the cognitive control and self-regulation literatures. The neurophysiological measures utilized in this experiment can be used to explain the loading factors and states that determine one's ability to self-regulate and engage in cognitive control processes. The current research dissociated the the ERN and the Pe and suggests that the ACC utilizes glucose for processing of erroneous behavior, whereas the PCC does not take advantage this resource

to control and correct errant behavior. The results from this experiment indicate that there is a link between the self-regulation and cognitive control literatures.

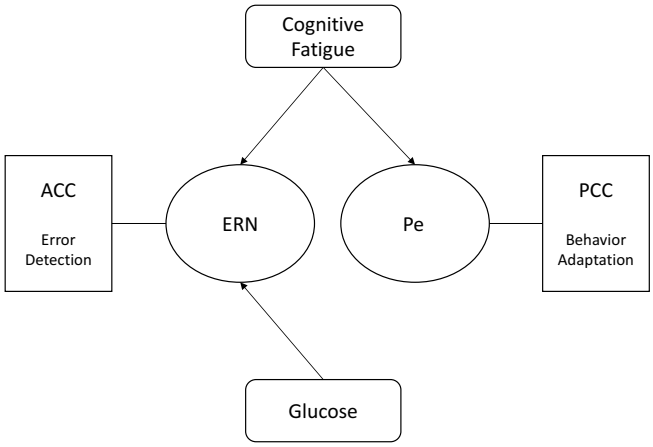

Figure 13. Diagram of observed effects.

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