ASSESSMENT OF GLUCOSE’S ROLE IN COGNITIVE CONTROL  
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Applied Cognition

Introduction

Cognitive control describes the processes key to task-centered behavior and maintaining it. These processes involve overcoming innate reactions, shifting goals, and flexibly interpreting information. Research has also suggested (e.g., Kaplan & Berman, 2010) “self-regulation”, a process by which planned actions are maintained, is also linked to cognitive control. The processes of cognitive control and self-regulation are both essential to goal-oriented behavior (Schmitt & Kray, 2018). Both of which share glucose as a common limiting factor, which may elucidate the connection between the two facets of goal behavior (Kaplan & Berman, 2010).

Over the past two semesters, we in the Applied Cognition lab have explored this link. While the glucose and self-regulation remains better understood, we seek to expand what is known about the effect of glucose on cognitive control. Using EEG and ERP (electroencephalography and event-related potential) to elucidate the connection between these two constructs through the well-known ERN signal (error-related negativity; Gehring, W. Goss, B, Meyer, D. Donchin, E. 1993, Vocat et al. 2018).

Background

The glucose depletion hypothesis of self-regulation states that cognitive tasks use the limited amount of glucose we possess (Persson, Welsh, Jonides, & Reuter-Lorenz, 2007; Gailliot et al., 2007; Galliott, Peruche, Plant, & Baumeister, 2009; Kennedy & Scholey, 2000; Scholey, Harper and Kennedy, 2001). When depleted, this results in the “fatigue” of self-regulation (Burmeister, Vohs & Tice, 2007), and participants experiences lapses in self-control, such as failing to restrain oneself from making an inappropriate comment.

The ERN is a response-locked ERP component linked to an erroneous response (Falkenstein, 1991; Gehring, Coles, Meyer, &; Donchin, 1990; Gehring & Fencsik, 2001; Coleman, Watson, &; Strayer, 2017). Motivation is known to relate to Error Related Negativity, whose amplitude increases when participants seek the greatest accuracy or monetary rewards (Gehring & Willoughby, 2002). Source localization and convergent fMRI studies strongly suggest that the ERN is generated in the Anterior Cingulate Cortex (ACC; Botvinick et al., 2001; Falkenstein et al., 2000; Hajcak, McDonald, & Simons, 2003; Herrmann et al., 2004; Kerns et al., 2004).

Motivation is imperative for ERN, self-regulation and cognitive control (Baumeister & Vohs, 2007). Decrease in ERN signals have been linked to self-regulatory failure, now we’re trying to understand if supplementary glucose would raise the peak of the ERN, preventing a fatigued response.

Noted deficits in self-regulation have been shown to cause deficits in cognitive control, and there is preliminary evidence to support cognitive control and self-regulatory processes sharing the same neural pathways and resources (e.g., von Hippel &amp; Gonsalkorale, 2005; Schmeichel et al., 2006; Blair, C &amp; Razza, 2007). There is a
relationship involving glucose, which depletes conversely with cognitive control and self-regulation. When participating in high-level cognitive tasks, many reach a state of fatigue in which performance becomes increasingly detrimental (Parasuraman, Warm, & See, 1998).

Literature shows that as the cognitive task becomes more challenging, glucose concentration decreases within the bloodstream (Scholey, Harper, & Kennedy, 2001; Gailot et al., 2007). We will assess our hypothesis through electroencephalographic methods, (i.e, ERP), in a double blind, placebo-controlled crossover experiment. Cognitive control is a decidedly important component of our higher cognition, as it is responsible not just for the driving our goal-centered behavior, but additionally maintaining them (Miller & Wallis, 2009).

Using ERP to isolate error-related negativity from the extraneous noise commonly associated with EEG. Therefore, we’re able to detect if glucose and not the placebo, is able to “restore” the error monitoring system of cognitive control, which will raise the amplitude of ERN signals. As we finish our testing phase, we will be using statistical methods such as ANOVA (analysis of variance) to support the significance of the results.

Method

Participants
Participants were recruited from the university of Utah and the surrounding area (N = 30, male = 12, female = 18, nonbinary/other = 1). All participants were between the ages of 18 and 64, have normal neurological functioning, and no history of blood disorders. Participants were all either compensated at a rate of 15$ an hour or equivalent class credit if they chose.

Materials
We used a 32 channel EEG cap with flexible montage, 5 additional eye-electrodes were used in conjunction with approximately half of participants, but was dropped for continuing participants because of the ability to get the same effect from data pre-processing using Matlab.

Procedures
Participants each came in for two 3-hour time slots a week apart at the same time. Each visit, they filled out an information form which collected data about caffeine, sleep and other confounds. At each session participants did three flanker task blocks and a single OSPAN, blood was drawn a total of four times from a single finger (middle). Participants were also given both a glucose beverage and a placebo (Gatorade and sugar free Gatorade), which were relabeled so that no experimenter was aware of which, and additionally counter balanced so that participants received either glucose or placebo first. A short motivation form was given at the end of every flanker, and a single form in which they guessed which order they had placebo and glucose to validate our double-blind aspect, and also what they thought possible effects of glucose could be on their cognition at the end of the finals session.

Expected results:
We expect that the application of glucose prevents the peaks of ERN signals from shrinking with fatigue related with cognitively strenuous tasks.

References


Falkenstein, 1991; Gehring, Coles, Meyer, & Donchin, 1990; Gehring & Fencsik, 2001; Coleman, Watson, & Strayer, 2017


