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Glucose metabolism and self-regulation — Is insulin resistance a valid proxy of self-control?

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ABSTRACT

Glucose metabolism has been suggested as an underlying biological factor of self-control stimulating a range of studies exploring the associations between glucose and self-control. Research on interindividual trait-like differences in glucose metabolism and self-control is sparse, as most previous research has focused on associations between state self-control performance and momentary glucose levels. In two experiments in healthy participants ($n = 60$, mean age 35.2 ± 13.9, 58% women; $n = 103$, mean age 25.8 ± 6.3, 67% women) consisting of a baseline assessment and a laboratory session, we examined whether trait markers of glucose metabolism (fasting glucose levels, oral glucose tolerance, and insulin resistance) correlated with trait measures of self-control and state self-control performance measured in a self-control dual-task paradigm. We found only small to moderate associations with insulin resistance, indicating that higher trait self-control went together with lower insulin resistance. These associations were limited to self-reported measures only. For the association with objectively assessed self-control performance no consistent pattern was observed. Taken together, the present research does not provide support for a meaningful relationship between self-control and glucose metabolism.

1. Introduction

Glucose metabolism has been suggested as a biological correlate of self-control (Gailliot & Baumeister, 2007) and linked to the strength model of self-control (Muraven & Baumeister, 2000). According to the strength model of self-control, self-control is a limited resource, which is thought to be consumed during acts of self-control, that is overriding dominant responses, thoughts or behaviors (Muraven & Baumeister, 2000). This, in turn, may leave individuals in a putative state of ego depletion that may hamper consecutive self-control performance. Gailliot and Baumeister (2007) posit that glucose metabolism might be associated with self-control capacity and self-control failure. Drawing on evidence from research in diabetes, they assume that differences in the ability to metabolize glucose such as glucose tolerance should reflect differences in the self-control capacity. Furthermore, actual decreases in blood glucose levels after exerting self-control might mirror the temporary depletion of self-control resources (Gailliot & Baumeister, 2007) — an assumption that was supported by an initial series of laboratory studies (Gailliot et al., 2007) and also confirmed by a few other studies (e.g., DeWall, Baumeister, Gailliot, & Maner, 2008; Dvorak & Simons, 2009). However, current research challenges this assumption of a link between self-control and temporary changes in blood glucose levels (e.g., Job, Walton, Bernecker, & Dweck, 2013; Lange & Eggert, 2014; Molden et al., 2012; Sanders, Shirk, Burgin, & Martin, 2012).

The difficulties to replicate the initial findings of Gailliot & Baumeister may in part be explained by methodological shortcomings of the original studies (e.g., use of commercial glucose monitors instead of reliable laboratory quantification methods, insufficient fasting periods). Even more critical, some conceptual problems render the notion that changes in glucose levels reflect ego depletion implausible: Gailliot and Baumeister (2007) hypothesized that blood glucose levels decrease because controlled and complex cognitive processes, such as those involved in self-control consume more glucose in the brain than can be supplied via the blood stream in a timely fashion. This assumption seems intriguing at first glance: brain metabolism relies on a constant supply of glucose from the blood stream as its primary fuel (McCall, 2004) to ensure normal cognitive functioning. However, intraindividual variation in peripheral glucose levels may be a poor proxy of brain glucose consumption due to several reasons: (1) in healthy participants peripheral glucose levels are regulated effectively within narrow physiological ranges with glucose utilization being closely matched by glucose production, which ensures a constant supply of glucose to the

Abbreviations: BIS-15, Barratt Impulsiveness Scale; BIS-15-NI, Barratt Impulsiveness Scale subscale non-planning impulsivity; BIS-15-MI, Barratt Impulsiveness Scale subscale motor impulsivity; BIS-15-AI, Barratt Impulsiveness Scale subscale attentional impulsivity; FBG, fasting blood glucose; HDL, high-density lipoprotein; IR, insulin resistance; LDL, low-density lipoprotein; OGT, oral glucose tolerance; SCS, trait self-control scale; TFEQ, Three Factor Eating Questionnaire; TFEQ-HU, TFEQ hunger; TFEQ-DIS, TFEQ disinhibition; TFEQ-CR, TFEQ cognitive restraint.

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brain (Messier, 2004). As consequence, increased glucose consumption in the brain due to self-control can hardly afflict peripheral glucose levels in normal physiological states. (2) While cognitive demand is associated with increased glucose uptake in the brain, the amount of increase due to this cognitive demand is assumed to be only a relatively small amount of overall brain glucose uptake (Messier, 2004). (3) Challenging the assumption further, there is little direct evidence that specific cognitive processes such as those involved in self-control consume more glucose than other cognitive processes (see Kurzban, Duckworth, Kable, & Myers, 2013 for a critical discussion).

Focusing on interindividual differences in glucose regulation rather than within person fluctuations in glucose levels, may be a more promising approach to shed light on the glucose hypothesis of self-control: interindividual differences in glucose regulation are well documented — even in healthy individuals (Sacks, 2011). Gailliot and Baumeister (2007) assumed that differences in the ability to transport and use glucose afflict the availability of glucose in the brain and, thus, might influence self-control performance in cognitive tasks as well as indicators of trait-self-control. This hypothesis is supported by evidence that glucose regulation affects cognitive functions (Lamport, Lawton, Mansfield, & Dye, 2009) by facilitating the glucose transport to the brain via the blood brain barrier (McCall, 2004). Furthermore, data suggests that peripheral insulin resistance is accompanied by central insulin resistance which negatively impacts brain-insulin signaling and, in turn, cognitive functioning (McNay & Recknagel, 2011). Surprisingly, interindividual differences in glucose metabolism have hardly ever been focused on in research on the self-regulatory strength model. To our best knowledge, there is only one study examining a direct link between glucose metabolism and trait self-control: Eriksson et al. (2012) found that men with normal glucose tolerance as measured by a standard oral glucose tolerance test had significantly lower scores in impulsivity, a facet of the factor conscientiousness, than men with newly diagnosed type 2 diabetes or prediabetes (Eriksson et al., 2012). The actual effect size was rather small, though, with $d = .13$, and the relationship was not found in women or after adjustment for potential confounders. Other studies that tried to examine the association of self-control and glucose metabolism exist (DeWall, Pond, & Bushman, 2010; DeWall, Deckman, Gailliot, & Bushman, 2011) but suffer from methodological problems: objective indicators of glucose metabolism were not assessed (DeWall, Pond et al., 2010; DeWall, Deckman et al., 2011) or the studies reported only correlations on a population level, for example that countries with high diabetes rates have high rates of violent crimes, which should be linked to deficient self-control (DeWall et al., 2011). In addition, little is known on how interindividual differences in glucose metabolism impact self-control performance in a given (laboratory) task. Only one study found that higher fasting blood glucose was associated with worse performance in the Stroop test (Gluck et al., 2013). As for the different indicators of glucose metabolism, despite the evidence linking insulin resistance to cognitive functioning, insulin resistance has never been studied in the context of self-control.

Thus, the aim of our study was to investigate if glucose metabolism covaries with measures of trait self-control and self-control performance in a healthy adult sample as predicted by the glucose hypothesis of self-control. In a pilot experiment and a conceptual replication experiment, we assessed self-control performance, different measures of trait-self-control, and three different indicators of glucose metabolism (insulin resistance, oral glucose tolerance, and fasting glucose). We hypothesized that lower insulin resistance, better oral glucose tolerance and lower fasting glucose are related to better trait self-control and better performance in a self-control task.

2. Materials and methods

Both experiments comprised two parts: a baseline examination to assess trait indicators of glucose metabolism, measures of trait self-control, and medical and demographic confounders, and a laboratory session consisting of a computer-based dual-task paradigm, the current standard procedure to assess self-control performance and ego depletion. The study was approved by the ethics committee of the Medical Association of the Federal State of Rhineland-Palatinate and conducted in accordance with the Declaration of Helsinki (no. 837.059.12-8160).

2.1. Participants

We consecutively recruited participants for both experiments between July 2012 and April 2014 via advertisements on university campus and via e-mail advertisements in leisure clubs in the region. Inclusion criteria were age between 18 and 65 years and sufficient German language skills. Participants with known diabetes mellitus or impaired glucose tolerance, cognitive impairment, current mental disorders, color blindness, and other medical conditions that may influence glucose metabolism (e.g., current pregnancy, chronic inflammatory diseases, current treatment with immune suppressive drugs, or thyroid disorders), cardiovascular diseases except hypertension, and treatment with anticoagulants other than acetylsalicylic acid were excluded. Students of psychology were not eligible for participation as they may be acquainted with the strength model of self-control, possibly rendering the experimental manipulation too obvious. Sports students were excluded as they may have above average physical fitness which is associated with enhanced insulin sensitivity and, thus, may probably lead to variance reduction in markers of glucose metabolism.

Eligible participants received written information that the study aimed at investigating self-regulation and its biological correlates and comprised two sessions: a baseline assessment and a laboratory session with two computer-based cognitive tasks. After providing written informed consent, participants first underwent the baseline assessment. To ensure that only participants with normal blood glucose metabolism were included in the laboratory experiment, the laboratory session was scheduled approximately four to six weeks later when the blood analysis results of the baseline assessment were available. After the laboratory session, participants were debriefed about the aim of the experimental manipulation and were asked not to share the information with other participants. Participants received a compensation of 25 EUR for the baseline assessment and another 10 EUR for the laboratory session.

2.2. Materials and methods Experiment 1

2.2.1. Baseline assessment

Participants were examined at 7:30 a.m. after an overnight fast of at least eight hours. They were also instructed to refrain from moderate to heavy exercise, alcohol, coffee and nicotine for at least eight hours before examination.

2.2.1.1. Blood glucose metabolism. We determined three different indicators of glucose metabolism: (1) insulin resistance reflects reduced sensitivity of the cells to insulin. In case of insulin resistance, larger amounts of insulin are required to ensure adequate glucose uptake (Mendez, Goldberg, & McCabe, 2010). (2) Oral glucose tolerance indicates the ability to utilize consumed glucose in an effective manner. While in case of normal glucose tolerance, glucose levels return to the normal range within two hours, impaired oral glucose tolerance leads to elevated glucose levels after meals (postprandial glucose). (3) Fasting glucose levels reflect glucose levels in the fasting state, i.e. rather independent from glucose consumption. Reduced sensitivity to insulin can be temporarily compensated by increased insulin production of the beta-cells. In consequence, impaired oral glucose tolerance and abnormal fasting glucose develop later on when the compensatory insulin production fails (Mendez et al., 2010).

Insulin resistance was determined with the validated Homeostatic Model Assessment 2 (HOMA 2, Wallace, Levy, & Matthews, 2004)