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Original Article

Does the Brain Consume Additional Glucose During Self-Control Tasks?

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Abstract: A currently popular model of self-control posits that the exertion of self-control relies on a resource, which is expended by acts of self-control, resulting in less of this resource being available for subsequent acts of self-control. Recently, glucose has been proposed as the resource in question. For this model to be correct, it must be the case that A) performing a self-control task reduces glucose levels relative to a control task and B) performing a self-control task reduces glucose relative to pre-task levels. Evidence from neurophysiology suggests that (A) is unlikely to be true, and the evidence surrounding (B) is mixed, and is unlikely to be true for subjects who have not recently fasted. From the standpoint of evolved function, glucose might better be thought of as an input to decision making systems rather than as a constraint on performance.

Keywords: self-control, glucose, brain metabolism, optimal foraging

Background and Introduction

A currently popular model of self-control posits that the exertion of self-control relies on a resource, which is expended by acts of self-control, resulting in less of this resource being available for subsequent acts of self-control (Baumeister, Vohs, and Tice, 2007; Muraven and Baumeister, 2000; Schmeichel and Baumeister, 2004; Vohs et al., 2008). The nature of this resource was previously left unspecified, referred to using metaphorical terms such as "willpower," or with the notion that self-control was "like a muscle" (Muraven and Baumeister, 2000).

Recently, Gailliot et al. (2007) reported the results of nine studies designed to make this model more concrete, specifying glucose as the resource necessary for, and depleted by, acts of self-control (see also Gailliot and Baumeister, 2007). Like much research in this literature, the studies in question used methods along the following lines. First, a "self-control" task was performed by subjects, after which a second "self-control" task was performed. In this literature, it is frequently reported that compared to relevant controls, performance on the second task tends to be lower (see Hagger, Wood, Stiff, and Chatzisarantis, in press, for a recent meta-analysis); the "resource" explanation of such

effects is that a resource was depleted by the first task, causing a reduction in performance on the second.

For the glucose account to be able to explain such results, both of the following propositions must be true.

> **Proposition 1**. Performing a self-control task reduces glucose levels *relative to a control task*.
> **Proposition 2.** Performing a self-control task reduces glucose levels *relative to glucose levels before the task.*

If Proposition 1 is not true, then the *difference* in performance observed in these studies cannot be due to a reduction in glucose. If Proposition 2 is not true, and glucose levels are as high or higher than they were before the task, then a reduction in glucose cannot be the cause of reductions in performance.

The requirement for Proposition 2 can be stated more strongly. Specifically*, for any subject in whom glucose does not go down relative to baseline, it cannot be the case that glucose is the resource in question*. That is, because the model specifies that it is the *reduction* in the resource that causes the *reduction* in performance; if there is no reduction in the resource, then this cannot explain the reduction in performance.

Note that the following two Propositions must also be true in order for the glucose model to be correct.

> **Proposition 3**. Performance on self-control tasks depends in some way on glucose.
> **Proposition 4**. Reduced performance on self-control tasks is due to reductions in glucose.

Note, however, that these both represent conditions of logical *necessity*, not logical *sufficiency*. If either of these two additional Propositions is false, then the model must be false. However, if either or both of them are true, that does not logically entail that the model is true.

This short piece is not intended as a complete review of the self-control literature (Baumeister, Schmeichel, and Vohs, 2007; Baumeister et al., 2007; Gailliot and Baumeister, 2007). Instead, it is intended to consider the plausibility of the glucose model given what is known about brain metabolism, and, using this analysis, consider what is arguably the best evidence in the social psychological literature that self-control tasks reduce blood glucose, the work by Gailliot et al. (2007). Note that the present paper reviews only five of the nine studies in Gailliot et al. (2007), and presents some novel analyses of the data collected in this paper, but not reported originally.

To be clear, the goal of this reanalysis is to investigate *only* Proposition 2, above. If Proposition 2 is false, then, because the truth of this Proposition is a *necessary* condition for the glucose model to be correct, then the glucose model of self-control cannot be correct, *independent of whether the other Propositions are true.*

When and why does peripheral glucose go down?

The glucose model of self-control is committed to the view that self-control tasks deplete glucose *because of energy consumption by the brain* (as opposed to organs in the

periphery, such as the heart). Gailliot et al. (2007) write "it seems likely that relatively few psychological processes are as expensive as self-control in terms of requiring large amounts of glucose" (p. 334), locating the expense of self-control in psychology and so, the brain. This makes relevant the question of the extent to which the brain consumes glucose as a function of task.

There is some evidence for a drop in peripheral glucose when subjects perform taxing cognitive tasks. Scholey, Harper, and Kennedy (2001) found in a small study (N=11) that compared to a control (key-pressing) task, subjects performing a "Serial Sevens" task showed reduction in glucose levels over a 5-minute interval. As the authors note, the Serial Sevens task is known to lead to an increase in heart rate (80 to 90 beats per minute; Kennedy and Scholey, 2000), making problematic the inference that the differences between the computational activity in the brain across conditions are responsible for the drop in glucose. (See also Scholey, Laing, and Kennedy, 2006; Wilkinson, Scholey, and Wesnes, 2002).

A second claim along similar lines is Fairclough and Houston (2004), who ran a study in which they measured subjects' glucose levels, and had them do one of two kinds of Stroop tasks for 45 minutes. One set of subjects did the Stroop task in which the word and the color of the word were the same – the "congruent" condition – and another group performed the task in which word and color were different – the "incongruent" condition. Glucose measurements were taken at 15, 30, and 45 minutes. Glucose did go down further in the "incongruent" condition. It bears mentioning that task performance did not decrease over time – unusual for self-control tasks – and that Marcora, Statiano, and Manning (2009) recently reported a study in which they also monitored subjects' blood glucose after a 45-minute task, the AX Continuous Performance Task, and this effect did not occur; glucose levels did not go down relative to the control task.

In short, the relationship between self-control performance and blood glucose level drops appears in some cases but not others. (See Gibson and Green, 2002, pp. 186-187 for a brief review and discussion of methodological difficulties with studies in this area.) Further, even if there are such drops, the reason for them is unclear, possibly having to do with what is going on in the rest of the body, rather than computations performed in the brain. This points to the importance of data surrounding the brain's metabolism of glucose across different types of tasks. If performing five-minute self-control tasks were found to reduce peripheral glucose levels, could this be due to brain metabolism?

Brain metabolism

Research on brain metabolism suggests that the relationship between blood glucose and mental function is complex, and not simply a matter of more "effortful" processes leading to the "soaking up" of more glucose by the brain. (See Gibson, 2007, and Messier, 2004, for recent excellent reviews.) This makes the intuition that "effortful" tasks straightforwardly lead to greater reduction in glucose misleading. Gibson and Green (2002), discussing the relationship between glucose and cognition, remarked that research in the area "…is based on the assumption that, since glucose is the major source of fuel for the brain (for example, see Sieber and Traystman, 1992), alterations in plasma levels of glucose will result in alterations in brain levels of glucose, and thus neuronal function. However, the strength of this notion lies in its common-sense plausibility, not in scientific evidence…" (p. 185).

Indeed, evidence suggests that the sorts of tasks in which subjects are engaging in this literature have very little effect on overall brain metabolism and, specifically, glucose use by the brain. Clarke and Sokoloff (1998) remarked that although "[a] common view equates concentrated mental effort with mental work…there appears to be no increased energy utilization by the brain during such processes" (p. 664), arguing that "…the areas that participate in the processes of such reasoning represent too small a fraction of the brain for changes in their functional and metabolic activities to be reflected in the energy metabolism of the brain…" (p. 675). Messier (2004) similarly concluded that it is "unlikely that the blood glucose changes observed during and after a difficult cognitive task are due to increased brain glucose uptake" (p. 39). This conclusion was recently echoed by Gibson (2007), who concluded that "task-induced changes in human peripheral blood glucose are unlikely to reflect changes in relevant areas of brain glucose supply" (p. 75).

Brain imaging data provide powerful evidence about how much glucose computational mechanisms consume. Recently, Raichle and Mintun (2006), in their review of brain imaging, suggested that "regional increases in absolute blood flow…rarely affect the overall rate of brain blood flow during *even the most arousing perceptual and vigorous motor activity*" (p. 467, emphasis added). They argue that relative to the continuous energy expenditure of the brain, *local* energy consumption due to a particular task "could be as little as 1%." Indeed, they conclude that the delivery of additional glucose is not the reason that blood flow increases to particular brain regions during particular tasks.

Another way to understand the magnitudes involved is to consider the numbers in terms of the familiar unit of calories. The brain as a whole consumes about one quarter of one calorie per minute (Clarke and Sokoloff, 1998). The consumption rate for the fraction of the brain involved in the particular computations associated with "self control" must, logically, be much smaller than .25 calories per minute. A one percent increase *across the entire brain* would, over the course of a 5-minute task, consume .01 X (5 minutes X .25 calories/minute) = .0125 calories. Suppose that one grants, for the sake of argument, an order of magnitude greater effect, a 10% increase (occasionally seen in regional changes using PET; Madsen et al., 1995). Suppose one also grants this increase in glucose consumption not just for the mechanisms involved in the self-control task – that is, the *marginal* computational mechanisms – but instead that this increase occurs across the entire brain. If one grants these very extreme assumptions, the caloric cost would still be well less than *.2 calories*.

That is, if one were to use this aggressive estimate – which could be off by multiple orders of magnitude – the brains of subjects categorized as "depleted" in this literature, have, relative to controls, used an additional amount of glucose equal to about 10% of a single Tic-Tac.

The magnitude of the plausible marginal glucose consumption by the brain is important not only in the context of evaluating the plausibility that self-control tasks, relative to control tasks, are having an effect on circulating glucose levels, but also in the context of controls used in this literature. The "placebo" drinks used, for example, in Gailliot et al. (2007), were sweetened with Splenda, which is marketed as a "No Calorie Sweetener," which the authors claim "does not increase blood glucose" (p. 330). However, the marketing slogan does not reflect the fact that while sucralose, which produces the sensation of sweetness, is not metabolized and so contains no calories (Roberts, Renwick, Sims, and Snodin, 2000), each packet of Splenda contains .8 grams of carbohydrates in

addition to sucralose, and so contains 3 calories (USDA National Nutrient Database for Standard Reference, 2009), or fifteen times the estimate above.

To put these values in further perspective, .2 calories is roughly the number of calories burned by running at a 9-minute-mile pace for about one second (Ainsworth et al., 2000). The claims regarding "depletion" turn on a minuscule, even trivial amount of glucose. Indeed, the conceptual difficulty is much worse. The glucose claim turns on the present concentration of glucose, not its recent history (see below), and not on the *reason* for its present level. So, if the glucose model were correct, performance on self-control tasks should be worse after vigorous exercise, which consumes orders of magnitude more glucose. (Indeed, if the glucose as resource model were taken seriously, researchers would not use self-control tasks to "deplete" subjects, given how little glucose these tasks consume.) However, exercise has consistently been found to *improve* performance on a large number of tasks, including "self-control" tasks such as the Stroop (Tomporowski, 2003). (See also Hillman, Erickson and Kramer, 2008). Recently, Hillman et al. (2009) found that 20 minutes of aerobic exercise, compared to resting quietly, leads to an *improvement* in performance on tasks requiring the cognitive control of attention among preadolescents.

Further, Madsen et al. (1995) directly measured the difference in blood glucose concentration of blood going into and coming out of the brain – hence measuring how much was removed from the blood by the brain – by comparing the concentration in arteries entering the brain compared to the veins leaving the brain. They measured this 41 times, before, during, and after subjects did a 10-minute Wisconsin Card Sort Task. They found no change in this difference from baseline compared to activation, suggesting that during the task, the brain was not differentially soaking up more glucose from the blood during this cognitively demanding task than before it.

In short, brain metabolism changes very little overall as a function of task (see also Lennie, 2003, p. 495). These lines of evidence converge to suggest that the brain does not use more glucose during activation when subjects are performing "self-control" tasks relative to other times and, even if the brain were using more glucose, it would be a minuscule amount. These ideas from the literature on brain metabolism point to the importance of looking carefully at claims that the brain is using more glucose during self-control tasks, the topic of the next section.

Re-analysis of data from Gailliot et al. (2007)

Gailliot et al. (2007) presented the first evidence that self-control tasks of the type used in the broader self-control literature lead to a reduction in peripheral glucose. In Study 1, subjects who were asked not to eat for three hours prior to the experiment had their glucose levels assessed as the first element of the procedure. Subjects were subsequently instructed to watch a film with common words appearing on the bottom corner of the screen. Half of the subjects were told to avoid looking at these words – this was the "self-control" condition – and half were not so instructed. Glucose levels were then measured. The prediction was that glucose levels would go down more in the self-control condition than in the control condition.

Because the above analysis makes the finding that blood glucose goes down when doing a 5-minute task very surprising in the context of the brain metabolism literature reviewed above, I requested the data from this study, in addition to Studies 2 through 6 (see

below). Gailliot (personal communication) indicated that "The data from Study 1 have been corrupted." The relevant results, from the original manuscript, are in the first two rows of Table 1, but I was obviously unable to independently verify these values.

Study 2 investigated the hypothesis that subjects "low in internal motivation to respond without prejudice" (IMS) would show reduction in glucose following an interaction with an opposite-race person as opposed to a same-race person. In contrast, no such reduction was predicted for those high in IMS because for these subjects, "suppressing prejudice should be a well practiced and hence presumably automatic way of acting" (p. 328). In this study, glucose drops are predicted as a function of an individual difference measure, rather than as a function of assignment to condition, so I do not analyze it further because it cannot speak as cleanly to the key issue at stake.

In Studies 3, 4, 5 and 6, a similar procedure to Study 1 was used. (Studies 7, 8 and 9 investigated whether consuming a glucose drink improves subsequent self-control performance relative to a placebo. These studies are not relevant to Proposition 2.) Subjects' glucose was measured and then subjects completed a self-control task, after which a glucose reading was taken. Subjects were then asked to perform a second self-control task because the researchers were interested in the relationship between performance on this second task and 1) subjects' absolute level of blood glucose, and 2) the level of glucose at the second reading, controlling for the subject's baseline glucose level.

The procedure up to performing the second self-control task in Studies 3 through 6 are replications of the attention condition in Study 1 with subjects who have not been asked to fast. The study was designed to look at performance as a function of the change in blood glucose levels. That is, given, for each subject, the drop in glucose, does this value predict performance?

These studies are the core of the reanalysis here, and it is important to take note of the logic that underlies these studies. First, this analysis *only* makes sense if one believed, before running the study, that the drop in glucose was a meaningful measure. Suppose that one thought that the glucose drop was *not* a meaningful measure for some reason, such as the concern that glucose might be entering into the blood stream, adding noise in sufficient magnitude to render the *difference* between glucose pre- and post- useless. In this case, an analysis investigating performance as a function of this *difference* would be of no value.

To be clear, if one *did* believe that this was a concern, one would not have conducted this study. That is, if the key measure, the glucose drop, is compromised because of glucose entering the blood, then the measure cannot be used to determine if the drop predicts performance. Note also that if one did have this concern – that endogenous glucose fluctuations swamp the putative change produced by the self-control task – then clearly one should not expect people who have not fasted to show a decrease in task 2 performance relative to those who do not do a self-control task. The noise from endogenous glucose entering the blood would, on this view, swamp any effect, making it just as likely that someone in the self-control treatment would have more, or less, blood glucose than someone in the control condition.

These points are important because the authors did not report the blood glucose drop in these four studies, arguing that because subjects didn't fast, glucose might be entering the blood stream. However, the reported analysis using the difference between pre- and post- readings means that they did not, in fact, believe that these readings were compromised. The fact that they reported an analysis *based on this relationship*, but not the

relationship itself, indicates that there was some reason, other than the argument offered in the text, that they did report these analyses.

Notice also that using the *drop* in glucose rather than the *absolute* measure as the predictor variable is inconsistent with the stated theory (though it might be consistent with a different theory). The stated theory is that glucose is a limited resource, so the prediction should be that performance depends on *the level of the resource*. The analysis presented, however, is based on the change, which means their implicit theory is that performance depends on the *recent change* in glucose. As an analogy, in essence, these four studies rely on a theory in which self-control is determined by the *recent drop in charge of a battery* rather than the *remaining charge* in the battery.

In any case, change in glucose was not reported for these four studies in the original manuscript. However, as noted, if the glucose model is to explain performance decreases for non-fasting subjects, then, *even against the backdrop of endogenous glucose variation*, glucose levels of subjects who have performed a self-control task must go down. If they do not – whether for this or some other reason – then the glucose model cannot explain performance reductions among such subjects because the model is that reduced performance is caused by lower levels of glucose in the blood.

Indeed, the question of whether glucose drops among subjects who have not fasted is actually *more* relevant than the data from subjects who have fasted (as in Study 1) because this methodological requirement is not frequently used in previous research in this literature (Hagger et al., in press). The glucose model is not supposed to explain self-control failures *only* for people who have not eaten for three hours and so have lower and/or more stable glucose levels. For this reason, the data from these four studies speak directly to the model.

The raw data from Studies 3 through 6, and the results are shown in Table 1. Note that because the raw data for Study 1 were "corrupted," I report data in the first two rows from the original manuscript. For Studies 5 and 6, the sample size reflects the raw data provided, which differs by one from what is reported in the manuscript. In evaluating these values, it is important to bear in mind that the accuracy (SD) of readings by the Accu-Check compact meters used for the blood glucose levels in the range reported is around 3.6 mg/dL (Roche Diagnostics, 2006). Across the three tasks, including the attention task (Studies 3 and 4), and two different self-control tasks (Studies 5 and 6), in none of the four cases did the reduction in glucose replicate. The mean glucose levels were statistically indistinguishable from before the task to after the task. Collapsing across the four new studies, again glucose levels do not change statistically. The same is true if one combines all the data from all five studies.

We can also simply ask what proportion of subjects in each condition experienced a drop in glucose, which obviously reduces the impact of outliers. This is indicated in the last column of Table 1, and none of these is different from chance. The distribution of changes in glucose from baseline to post-task across Studies 3 through 6 is shown in Figure 1 and can be seen to resemble a normal distribution centered at zero.

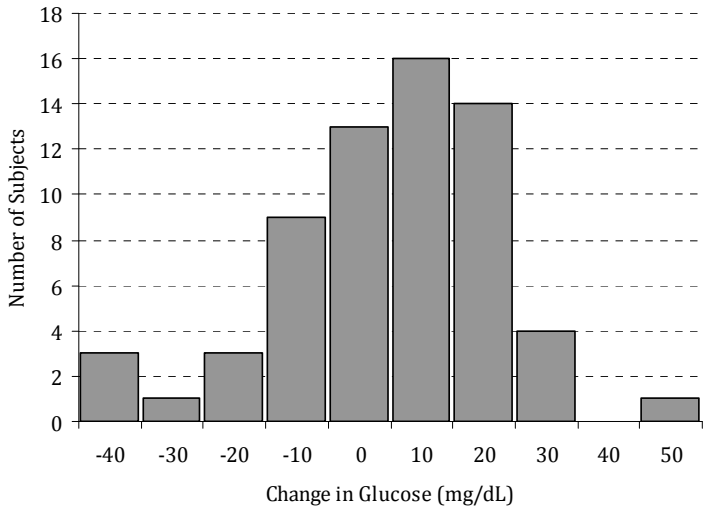
Table 1. Sample size, mean (*SD*) absolute levels of and changes in glucose levels (in mg/dL) in five of the studies reported in Gailliot et al. (2007). Significance tests are one-tailed, following the original manuscript.

Study	Task	*n*	Glucose time 1	Glucose time 2	Δ	*p*	Fraction Increase
1	Attention Control	52	107.10 (21.02)	101.22 (18.34)	-5.88	< .05	?
1	Control	51	102.24 (21.20)	103.24 (18.71)	+1.00	*ns*	?
3	Attention Control	15	96.07 (22.08)	91.67 (17.77)	-4.40 (17.23)	0.17	6/15
4	Attention Control	12	99.42 (18.15)	105.75 (21.18)	+6.33 (27.75)	na	7/12
5	Stroop	22	100.32 (12.83)	104.32 (14.38)	+4.00 (19.00)	na	15/22
6	Emotion Regulation	16	104.75 (25.00)	105.06 (19.79)	+0.31 (17.68)	na	8/16
3-6	Various	65	100.26 (19.31)	101.85 (18.37)	+1.58 (20.10)	na	36/65

In summary, although Gailliot et al. (2007) reported only those data consistent with their prediction, the complete dataset undermines the hypothesis that these tasks reduce the level of peripheral glucose. Obviously, if performing a short self-control task does not reduce glucose among subjects who have not fasted, then the glucose account of the effect on task 2 performance cannot be correct for such subjects.

The difference between the Study 1 results and the results of Study 3 through 6 could be due to the fact that subjects fasted before Study 1, a Type 1 error in Study 1, or Type II errors in Studies 3-6. If the result is due to fasting, then the glucose model does not apply to subjects who have not recently fasted, and therefore cannot explain the results of the studies the model is intended to explain.

Figure 1. Histogram of changes in blood glucose levels between baseline and the completion of the first self-control task in Studies 3 through 6 in Gailliot et al (2007). Values on the x-axis reflect the top value of the bin (e.g., "20" corresponds to the values 11 through 20). One observation (75) is not pictured.



If the result is not due to fasting, then the possibility of a Type 1 error in Study 1 seems likely. In Study 1, mean glucose for subjects in the key cell in which glucose was predicted to go down started quite high, 107.1 mg/dL, even though these subjects were asked to fast for three hours before the experiment. Taking a weighted mean across all cells in all conditions, the mean of subjects' glucose coming into the lab was 103.0 mg/dL. Estimating the standard deviation of 20, given $n = 168$, we can estimate the standard error of the mean to be roughly 1.5 mg/dL. If we assume this estimate, a mean of 107.1 mg/dL is more than two standard errors away.

So, the mean glucose level in the key cell in which glucose had to decline to see an effect was well above the mean of the population. If changes in glucose levels over the period of the study are a random walk (see Figure 1), then regression to the mean could have led to the appearance of a reduction in glucose due to the task. The single case in which glucose was found to decline, then, might be simply to an unlucky draw of 52 subjects whose glucose was particularly high.

Dvorak and Simons (2009) have recently provided additional evidence of a glucose drop among subjects performing a self-control task. In this work, subjects were asked to fast for three hours, as in Study 1 of Gailliot et al. (2007). Subjects watched videos, one humorous and one disgusting, and then given anagrams to solve, some of which were solvable, others of which were not. Some subjects were told to suppress their emotions while watching the video, while control subjects were not so instructed. Supporting the view that the initial 107.1 mg/dL measure in Gailliot et al (2007) was anomalous, the average glucose level for the 180 subjects in this study who had fasted for three hours was 97.7 mg/dl, with a *SD* of 11.0, yielding a standard error of about .8. It is important to note that blood glucose did fall more in the suppression condition (97 mg/dl to 92 mg/dl) than in the control condition (98 mg/dl to 95 mg/dl). These results suggest that among subjects

who have fasted, emotion suppression does lead to a larger glucose drop, though the possibility that this effect is driven by what is going on in the periphery, rather than the brain, when one is suppressing emotion cannot be excluded.

Glucose level: constraint or input?

The data in Gailliot et al. (2007) do not support the claim that, at least for non-fasting subjects, self-control tasks reduce the level of glucose in the blood. The level of glucose – or the recent change in level of glucose – might, nonetheless, affect performance on many kinds of tasks, including, perhaps, "self-control" tasks (e.g., Donohoe, and Benton, 1999; Scholey et al., 2001). This issue is, however, subject to a certain amount of debate. Gibson (2007) suggested that the evidence surrounding this relationship is "divergent" and that there is "no clear mechanistic model that satisfactorily explains" the diversity of results. He concludes that "…overall brain function appears to be quite insensitive to such fluctuations in blood glucose…" (p. 80), with clear benefits being "limited to hippocampally mediated verbal memory" (p. 71). Green, Elliman, and Rogers (1997) found no relationship between glucose levels and their measures of cognitive function, and concluded that "the brain is, therefore, relatively invulnerable to short-term deprivation," suggesting that effects might be seen only "after several days of total food deprivation" (p. 89; see also Amiel, 1994).

If, however, some relationship exists, it need not necessarily be the one implied by a resource account. (See Gibson, 2007, for a list of possibilities.) Glucose, like water and oxygen, is central to biological activities. The level of each of these is monitored by appropriate systems, and these levels probably influence behavior. In particular, it seems likely that readings of these levels computed by some mechanisms can act as *inputs* to other mechanisms that influence the deployment of computational resources.

Consider, for example, research in the animal literature, in particular on risk-sensitive foraging. The level of risk that an organism will endure depends on its current state (Stephens, 1981). Organisms in a low caloric state will engage in more risky foraging behavior. A resource construal of this effect would be that blood glucose is necessary in order for the organism to suppress risky foraging behavior. An input account, which seems more likely, would be that there are mechanisms designed to monitor calories, and that the information produced by this mechanism acts as an input to a decision-making system regarding foraging. When calories are low, foraging is up-regulated.

In the same way that a low reading on a putative "sociometer" mechanism (Leary and Baumeister, 2000), for example, might motivate behavior to increase one's social value (at the expense of other priorities), mechanisms that monitor material needs might have similar effects. Messier (2004) reviewed evidence that is consistent with such an account in the context of memory effects – though similar pathways might explain effects in other domains – suggesting that "a number of observations suggest the existence of one or more peripheral mechanisms that mediate the effects of glucose and other sugars as well as of other nutrients. Possible mechanisms include glucose-responsive neurons in the liver or elsewhere that transmit a neural message via the vagus nerve and a hormonal mediator released by the gut which could act in the periphery or centrally to influence brain activity related to memory processing" (p. 48). Gibson and Green (2002) similarly summarize the data relevant to this claim: "The change in cognitive performance after administration of glucose, and other foods, may depend on the level of sympathetic activation, glucocorticoid

secretion, and pancreatic β-cell function, rather than simple fuelling of neural activity" (pp. 169-170), and, more directly, they argue that "behavioural effects of glucose ingestion should not be seen as resulting from simple fuelling of neural activity" (p. 198; see Rogers and Lloyd, 1994, and Green et al., 1997, for similar remarks regarding the role of sympathetic activity).

Why might the level of glucose in the blood be relevant to tasks requiring attention? Organisms have mechanisms designed to make adaptive decisions about how their computational resources – such as those frequently discussed using the rubric of "attention" – ought to be deployed. In the same way that sensory information recruits attention – threats from predators, mating opportunities, and so on – internal variables probably influence the deployment of resources. Thirsty organisms' attention is probably deployed around the task of acquiring water in the same way that hungry organisms' attention is allocated to determining how to find food. Given that computation is finite, there are necessary tradeoffs. An organism computing means of gaining food, acquiring water, impressing a mate, or evading a predator, is to some extent not using computational resources for the other tasks.

To the extent that glucose levels influence such computations, the degree of computational resources allocated to the particular cognitive task assigned by an experimenter might well vary as a function of, perhaps, both absolute levels of glucose and recent changes in glucose. Indeed, Scholey, Sünram-Lea, Greer, Elliott, and Kennedy (2009) recently took such a view, suggesting: "One mechanism through which glucose acts as a cognition enhancer is through allocation of attentional resources." (p. 549). (See Messier, 2004, for proposals regarding detection mechanisms in the periphery that influence central nervous system performance.) Wang and Dvorak (2010) recently presented a related idea, suggesting that their "findings suggest an adaptive mechanism linking human decision making to metabolic cues" (p. 4).

Mechanisms of these types might explain the relationship between peripheral glucose and performance. In this context, consider physical exercise performance, an area in which a resource or fuel model would seem to be a very likely model to explain performance. That is, one might think that in the context of physical exercise, the idea that glucose is a constraint on performance would be even more likely to be the case than in the context of mental activity. If glucose is in fact an *input* in the context of exercise, the possibility that it is an input in the context of self control would be particularly plausible.

Chambers, Bridge, and Jones (2009) recently investigated the effect of a glucose-rich beverage compared to placebo placed in one's mouth and then spit out, rather than ingested, on real, rather than metaphorical, muscles, studying people engaged in physical exercise. They found that simply putting the glucose in the mouth of cyclists improved their performance. They conclude that "improvement in exercise performance that is observed when carbohydrate is present in the mouth may be due to the activation of brain regions believed to be involved in reward…" (p. 1779). Carter, Jeukendrup, and Jones (2004a) showed a similar result, concluding that "the mechanism responsible for the improvement in high-intensity exercise performance with exogenous carbohydrate appears to involve an increase in central drive or motivation rather than having any metabolic cause" (p. 2107). Further, provocatively, simply infusing glucose into the blood does not improve exercise performance (Carter, Jeukendrup, Mann, and Jones, 2004b), strongly suggesting that it is the signal, rather than the glucose itself, that drives performance

increases.

Pottier, Bouckaert, Gilis, Roels, and Derave (2010) obtained a similar result and suggested that the improvement observed when subjects rinsed with carbohydrate (CHO) solutions derive from the possibility that "fatigue signals from the muscles to the brain are being suppressed unconsciously by afferent CHO signals from the CHO receptors in the oral cavity to certain areas of the brain" (p. 109). This idea, that sugar in the mouth reduces sensations of effort, in turn influencing behavior, resonates closely with the idea that glucose is an input, rather than constraint.

In short, persistence on "self-control" tasks might depend on representations of costs and benefits of continuing (Kurzban, in press). The perception of high glucose concentrations in the mouth might activate reward systems, leading to increased effort on tasks. If physical effort can be explained by virtue of understanding glucose as an input or reward, mental effort might be similarly explained. This changes glucose in the blood or the digestive tract from a resource tethering performance to an input that feeds into decision-making systems that ultimately determine the allocation of continued effort, whether physical or mental.

Conclusion

The data from Studies 3, 4, 5 and 6 of Gailliot et al. (2007), which replicate the procedure in Study 1 and are directly relevant to the hypothesis that self-control tasks reduce levels of glucose in the blood, lead to a conclusion opposite to the ones that the authors reached: exerting self control does not reduce levels of glucose in the bloodstream. If one accepts this conclusion, because the glucose drop is a logically necessary component of the glucose account, the data reported in the manuscript undermine, rather than support, this account. This conclusion is bolstered by the fact that subjects in Study 1 were instructed to fast. If glucose drops only among subjects who fasted before studies of this nature, then the glucose model cannot explain data from all those studies in which subjects did not fast. Data from Dvorak and Simons (2009) support the view that relative glucose drops occur in subjects who have fasted for the particular task they used.

The failure to find the effect predicted by the glucose model of self-control is not surprising given what is known about brain metabolism. Even very different computational tasks result in very similar glucose consumption by the brain. The neuroscience literature very strongly implies that the marginal difference in glucose consumption by five minutes of "self-control" tasks is unlikely in the extreme to be of any significant size. The research on exercise shows that burning calories through physical activity, which should have much greater "depleting" effects, in fact shows the reverse pattern. Even if it were the case that self-control reduced levels of peripheral glucose, the cause of the reduction is not in line with a "resource" account in which self-control mechanisms burn through glucose in the brain and are impaired by the subsequent lack of glucose, but rather a cascade of effects mediated through peripheral systems.

Further, holding aside failures to replicate the basic effects (Murtaugh and Todd, 2004), it would seem that the glucose model – or any "resource" model – is undermined by demonstrations that "depleted" subjects can do just as well as non-depleted subjects on a subsequent self-control task if they are given a gift (Tice, Baumeister, Shmueli, and Muraven, 2007). To the extent that such findings are taken to support the resource account, it is only because the model is sufficiently protean to accommodate essentially any

findings.

The weight of evidence implies that the glucose model of self-control in particular – and perhaps the resource model in general – ought to be carefully rethought. If there is no plausible candidate for the putative resource, then the resource metaphor ought to be abandoned. From a computational perspective, a "resource" account is the wrong kind of explanation for performance decrements to begin with. No one whose computer is performing slowly would think that the fault lies in the power supply – or the fact that running Excel for five minutes drained the battery – even though no one would deny that electricity is necessary for computers. The correct explanation for changes in performance might well lie in domain of computation (e.g., Dewitte, Bruyneel, and Geyskens, 2009).

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