Personality and Social Psychology Review

http://psr.sagepub.com

# The Physiology of Willpower: Linking Blood Glucose to Self-Control

Matthew T. Gailliot and Roy F. Baumeister

*Pers Soc Psychol Rev* 2007; 11; 303
DOI: 10.1177/1088868307303030

The online version of this article can be found at:
http://psr.sagepub.com/cgi/content/abstract/11/4/303

Published by:
SAGE
http://www.sagepublications.com

On behalf of:
SPSP

Society for Personality and Social Psychology, Inc.

# The Physiology of Willpower: Linking Blood Glucose to Self-Control

**Matthew T. Gailliot**
**Roy F. Baumeister**
*Florida State University*

*Past research indicates that self-control relies on some sort of limited energy source. This review suggests that blood glucose is one important part of the energy source of self-control. Acts of self-control deplete relatively large amounts of glucose. Self-control failures are more likely when glucose is low or cannot be mobilized effectively to the brain (i.e., when insulin is low or insensitive). Restoring glucose to a sufficient level typically improves self-control. Numerous self-control behaviors fit this pattern, including controlling attention, regulating emotions, quitting smoking, coping with stress, resisting impulsivity, and refraining from criminal and aggressive behavior. Alcohol reduces glucose throughout the brain and body and likewise impairs many forms of self-control. Furthermore, self-control failure is most likely during times of the day when glucose is used least effectively. Self-control thus appears highly susceptible to glucose. Self-control benefits numerous social and interpersonal processes. Glucose might therefore be related to a broad range of social behavior.*

*Keywords:* *aggression; glucose; self-control; self-regulation; attention; emotion regulation; impulsivity; stress; crime; alcohol*

Self-control is the capacity to override one's impulses and automatic or habitual responses, and it is the conscious and effortful form of self-regulation. Self-control includes controlling thoughts, emotions, desires, and behavior, particularly so as to bring them into line with societal or personal standards such as moral rules, laws, ideals, social norms, and prescriptive expectations. From a biological perspective, self-control involves brain activity occurring in the prefrontal cortex, such as the anterior cingulate cortex (Banfield, Wyland, Macrae, Munte, & Heatherton, 2005; Kandel, Schwartz, & Jessell, 2000).

Self-control seems vital for the optimal functioning of humans individually and collectively. By exerting self-control, people can fulfill their personal aspirations, resist behaving in maladaptive or harmful ways, and abide by social rules, morals, laws, and other regulations. Self-control appears to be powerfully adaptive in many life domains, as indicated by its association with greater interpersonal popularity, healthier interpersonal relationships, superior school performance, and better mental health and coping skills, as well as less susceptibility to substance abuse problems, eating disorders, and criminality (Duckworth & Seligman, 2005; Finkel & Campbell, 2001; Gailliot, Schmeichel, & Baumeister, 2006; Gottfredson & Hirschi, 1990; Mischel, Shoda, & Peake, 1988; Muraven, Collins, & Nienhaus, 2002; Pratt & Cullen, 2000; Shoda, Mischel, & Peake, 1990; Tangney, Baumeister, & Boone, 2004). Self-control is particularly useful in facilitating interpersonal cooperation and group harmony, and it may have evolved largely for this purpose (Baumeister, 2005). For instance, self-control allows people to suppress stereotypes and prejudices (Gordijn, Hindriks, Koomen, Dijksterhuis, & Van Knippenberg, 2004; Richeson & Shelton, 2003; Richeson & Trawalter, 2005; Richeson, Trawalter, & Shelton, 2005), refrain from responding negatively toward their romantic partner and instead respond positively (Finkel & Campbell, 2001), and make a desirable impression on others (Vohs, Baumeister, & Ciarocco, 2005). Self-control is related to and benefits a wide range of social behavior.

**Authors' Note:** Please direct correspondence to Matthew T. Gailliot at gailliot@psy.fsu.edu.

*PSPR*, Vol. 11 No. 4, November 2007 303-327
DOI: 10.1177/1088868307303030

An accumulating body of research suggests that self-control relies on some sort of limited resource or energy, thus reviving the folk notion of willpower (for reviews, see Baumeister, Schmeichel, & Vohs, in press; Gailliot & Baumeister, in press; Muraven & Baumeister, 2000). A single act of self-control seems to deplete this resource, and subsequent efforts at self-control are impaired as a result. The idea that self-control relies on a limited resource or energy is, however, only a convenient metaphor at present. The precise nature of an energy source that might explain how self-control functions is unknown, and researchers have had to refer to this energy source in only vague or abstract terms.

This article reviews evidence suggesting that blood glucose plays an important role in self-control. Glucose is one physiological substrate on which self-control depends. The use and availability of glucose can help explain patterns of self-control success and failure. Blood glucose is an important, integral part of the body's energy processes, and its depletion and other dynamics could potentially help the energy models of self-control move from mere metaphor to physiological mechanisms.

Why focus on self-control? In fact, most mental and physical activity in the human organism depends on and consumes energy from glucose, and this raises broader questions about whether there is anything special about self-control and whether glucose energy dynamics should be incorporated into all psychological theories. To be sure, it would be presumptuous of us and far beyond the scope of this article to propose that all psychological theorists begin talking about glucose. There are at present many researchers whose excitement over brain research has led them to anticipate that soon all psychological theories will have to include some recognition of brain processes. If and when that happens, glucose, as fuel for brain processes, may also find itself mentioned in every theory. However, we think this is not imminent, and there are two important reasons that glucose may garner a more prominent place in self-control theory than in most other psychological theories.

The first reason is that self-control theory—very much unlike most currently active theories in social psychology—has been pushed by empirical findings to cultivate energy models in order to explain the observed behavior patterns. For the past decade, researchers have been finding that performance at diverse self-control tasks steadily declines even when participants move from one task to a seemingly unrelated one (e.g., Muraven, Tice, & Baumeister, 1998). The most parsimonious explanation is that some sort of energy resource is consumed in the process of exerting self-control. Dissonance theory, attribution theory, self-esteem theory, interdependence theory, and most other psychological theories have not had to incorporate energy models to account for their findings. They may or may not eventually incorporate brain processes and even glucose (as brain fuel) into their accounts, but so far it has not been necessary. In contrast, self-control theory has to explain some behavioral phenomena for which depletion of an energy resource is the leading explanation. The nature of that resource is therefore a pressing question for self-control researchers, whereas it is not for researchers working on other theories.

The second reason concerns what is unusual about self-control (and indeed what may explain why energy dynamics emerged as a central question earlier and more urgently than in other theories). As we have said, it is true that all brain processes and therefore all psychological events depend on the consumption of energy from blood glucose. But for most, apparently, this may involve small and unproblematic amounts of glucose. Indeed, one of the most heavily studied areas of socially cognitive processing today involves automatic information processing, which is defined in part by its low requirements in terms of mental effort and energy. Self-control may be unusual in its relatively high demands for energy. Recent evidence has suggested that the high effort involved in rational, intelligent decision making may deplete the same resource needed for self-control, as indicated by behavioral decrements in self-control after people have made many choices (Vohs et al., 2006). There may be other processes that have similarly large energy requirements. Among them, self-control leads the way not because of any unique reliance on energy but rather because of its central importance in daily life and widespread influence on a broad range of behaviors. But most psychological processes may be relatively low in energy requirements and therefore relatively unaffected by the body's glucose dynamics.

At a theoretical level, we think the self's executive function, also known as self as agent, is generally expensive in terms of energy and thus glucose. The self is the controller of controlled processes, the self-regulator, the decision maker, and the initiator of active as opposed to passive responding (Baumeister, 1998). That set of processes would presumably all involve high amounts of glucose. At present, we have no theoretical basis for predicting that any further processes would be similarly expensive, but there is no basis for ruling out the possibility that one or two may yet be found, perhaps especially relatively nonobvious ones such as rare but large responses.

By and large, social psychologists have not used energy models for decades. Indeed, initial evidence indicating that self-control relies on a limited energy source led to the term "ego depletion" to describe self-control processes (Baumeister, Bratslavsky, Muraven, & Tice, 1998), in homage to Freud, who was one of the last psychological theorists to describe the self in energy terms (Freud, 1923/1961a, 1933/1961b). Freud theorized that civilized behavior was possible because at some point in development the ego learned to take instinctual energy from the id and convert it into a superego, whereupon it could be used to restrain socially undesirable impulses. The ego also consumed and discharged energy while trying to carry out behavior under the conflicting demands of id, superego, and external reality. Although we are not espousing that antiquated model, we do understand self-control as a vital faculty that enables the individual to alter his or her behavior so as to conform to societal standards and values, and to forgo immediate rewards and pleasures in order to realize long-term benefits.

After Freud, energy models of the psyche fell into disuse among most psychological theorists, but recent advances in psychology have begun to integrate psychological processes with biological ones (e.g., brain activity, immune function, circadian rhythms). This line of theorizing seems bound to reawaken interest in energy models—if only because the human body is indisputably an energy system, whose very life is an uninterrupted cycle of ingesting and burning energy. Moreover, psychological processes almost certainly involve some energy requirements. For example, in explaining the constraints on the evolution of intelligence, Dunbar (1998) emphasized that the human brain constitutes only 2% of body mass but consumes 20% of the body's calories. Such expensive organs and processes will only be supported by natural selection to the extent that they pay for themselves in palpable, adaptive benefits. As noted above, self-control is hugely adaptive and beneficial for life in human cultural society, and so evolution might have repeatedly selected in favor of hominids with a progressively higher capacity for self-control, even if it did burn a lot of fuel.

The broader implication is that somehow the human organism manages to convert the caloric energy it takes in as food into the capacity for complex psychological processes. Self-control is a complex but crucial part of successful human functioning, and if it does indeed operate on the basis of energy, then some actual physiological energy resource will have to be integrated into self-control theory. Glucose, being an important component of the human energy system, therefore gained plausibility as one physiological substrate of self-control.

## THEORETICAL BASIS AND BACKGROUND

### Self-Control Relies on a Limited Energy Source

Previous research has indicated that self-control relies on a limited energy source that becomes depleted with use (Baumeister, 2005; Muraven & Baumeister, 2000). After exerting self-control, people are more prone to fail at later attempts at self-control. This suggests that an initial act of self-control consumes some energy source that is needed for later attempts at self-control. Moreover, this energy source seems to be a common stock used for many different forms of self-control, such that self-regulatory behaviors from several different domains (e.g., emotion regulation, attention control, impulse control, performance optimization) appear to deplete and rely on the same energy source.

Laboratory studies have found that after participants complete an initial self-control task, they perform worse on a second self-control task compared to participants whose initial task did not require self-control. For example, in one study, participants had to limit their intake of beer during a taste test because they anticipated a driving test, and participants who had previously engaged in a thought-suppression exercise drank more beer than those who had not previously exerted self-control (Muraven et al., 2002). In another study, resisting the temptation to eat cookies caused participants subsequently to give up faster on a frustrating task (Baumeister et al., 1998). Currently, more than 50 studies have supported the idea that self-control relies on a limited energy source, and these studies have examined a broad array of self-regulatory behaviors, such as stereotype suppression and the control of prejudicial reactions (Gordijn et al., 2004; Richeson & Shelton, 2003; Richeson & Trawalter, 2005), accommodating behaviors in romantic relationships (Finkel & Campbell, 2001), dietary restraint (Kahan, Polivy, & Herman, 2003; Vohs & Heatherton, 2000), and impression management (Vohs et al., 2005). These findings fit an energy model because they suggest that the first act of self-control consumes or depletes some resource, thereby impairing self-regulation on the subsequent task.

Evidence from outside the laboratory is also consistent with the view that self-control relies on some sort of finite energy. One study found that people who tried to limit their alcohol intake in situations outside of the laboratory were less able to do so after they had previously self-regulated (e.g., controlled their mood; Muraven, Collins, Shiffman, & Paty, 2005). Furthermore, coping with stress requires self-control (e.g., Muraven & Baumeister, 2000), and after coping with everyday stresses outside the laboratory, people are less successful at self-control (e.g., dieting; Cohen & Lichtenstein, 1990; Wadden & Letizia, 1992; Wevers, 1988). Controlling emotions requires self-control (e.g., Baumeister, Heatherton, & Tice, 1994), and after controlling emotions in their daily lives, people are less able to exert self-control (e.g., quit smoking; Ashton, 1982; Bradley, Phillips, Green, & Gossip, 1989; Brownell, Marlatt, Lichtenstein, & Wilson, 1986; Greeno & Wing, 1994; Hodgins, el Guebaly, & Armstrong, 1995; Marlatt & Gordon, 1985; Shiffman, 1982).

Thus, self-control appears to rely on a limited energy source, but it is not currently known what this energy source might be. The purpose of this review is to survey evidence suggesting that glucose is one important component of the energy source of self-control.

## Glucose and How It Operates

Glucose is the fuel for the brain. The brain's activities rely almost exclusively on glucose for energy (e.g., Siesjo, 1978; Weiss, 1986). Glucose from the bloodstream is metabolized in brain regions needed to carry out a particular task (e.g., light stimulation increases glucose metabolism in the visual cortex; McNay, McCarty, & Gold, 2001; Reivich & Alavi, 1983). Glucose enables cerebral functioning by providing the fuel for neurons to fire impulses. The brain must receive an adequate supply of glucose in order to function effectively.

All cerebral activity requires glucose as its fuel under normal conditions. Cerebral activity therefore consumes glucose and reduces available quantities of glucose in the brain and periphery (e.g., Benton, Owens, & Parker, 1994; Fairclough & Houston, 2004; McNay et al., 2001; McNay, Fries, & Gold, 2000). Glucose can be consumed in the brain faster than it can be replenished, however, and so cerebral activities can deplete the brain's supply of glucose (e.g., Benton, Parker, & Donohoe, 1996; Fairclough & Houston, 2004; McNay et al., 2001).

When glucose levels are severely low, cerebral functioning is profoundly disrupted, producing numerous cognitive and behavioral deficits (e.g., impaired coordination, blurred vision, amnesia, bizarre behavior and personality change, confusion, and anxiety). Relatively subtle changes in glucose can also have an important influence on thought and behavior (Benton, Brett, & Brain, 1987; Donohoe & Benton, 1999a; Fairclough & Houston, 2004; D. O. Kennedy & Scholey, 2000; Scholey, Harper, & Kennedy, 2001), though some scholars previously believed otherwise (e.g., Van den Berg, 1985).

Though the level of available glucose is important to cognitive functioning, so too is the body's ability to use glucose effectively (e.g., Benton et al., 1996). The body's quantity of glucose is normally increased by eating, and this shows up first as an increase in the blood glucose levels. The body then releases insulin to help store the glucose, and blood glucose levels return to baseline. In this fashion, the body is able to obtain and store glucose for future use. Some individuals, however, demonstrate poor glucose tolerance as a stable trait. Their bodies use glucose ineffectively, such that they are less able than other people to store and use glucose. After these individuals take in glucose, their blood glucose level either remains high or, after the body releases insulin, drops well below baseline. The body must use glucose effectively, which requires being able to store glucose and then to mobilize glucose from its stores as needed. Thus, people with good glucose tolerance seem more capable than others of transporting glucose to the brain in the face of cognitive demand. As a result, those with good glucose tolerance have been found to perform better on some cognitive tasks (e.g., tests of logical reasoning), compared to those with poor glucose tolerance (Benton et al., 1996).

In short, the brain relies on glucose to function. When the flow of glucose to the brain is inadequate—either because glucose is low or because glucose cannot be transported to the brain (i.e., poor glucose tolerance)—cerebral functioning is impaired.

## Self-Control Depletion and Glucose

Though nearly all cerebral functions rely primarily on glucose for fuel, some mental capacities appear more sensitive than others to changes in glucose. Specifically, the influence of glucose seems most pronounced on effortful, controlled, or executive processes (e.g., Benton et al., 1994; Benton et al., 1996; Donohoe & Benton, 1999a, 1999b; Fairclough & Houston, 2004; Korol & Gold, 1998; D. O. Kennedy & Scholey 2000; Owens & Benton, 1994; Parker, 1995; Scholey et al., 2001; Sünram-Lea, Foster, Durlach, & Perez, 2002; cf. Benton, 1990). Effortful, controlled, or executive processes require more glucose than simpler, less effortful, or automatic processes, and they are more likely to be impaired when glucose is low or cannot be used effectively. In this sense, effortful, controlled, or executive process are quantitatively different from other processes in that they require more glucose. It is plausible that effortful processes require more glucose because they are somehow qualitatively different from other processes, yet these qualitative differences are unknown at present. Regardless of whether the difference in biological process is quantitative or qualitative, however, the implication is that psychological theories about effortful, controlled, and executive processes, such as self-control, will have to incorporate energy dynamics much more centrally and prominently than will psychological theories that invoke the simpler and more automatic processes.

One central hypothesis of this review is that low levels of glucose and the inability to transport glucose effectively to the brain (i.e., poor glucose tolerance) should be associated with impaired self-control. Conversely, restoring the amount of available glucose to higher and optimal levels should then improve self-control. This does not entail a linear relationship between glucose and self-control, such that a person who downs a large bag of candy will become a paragon of self-discipline for the next few hours. Rather, we hypothesize that the differences will be between having inadequate or depleted glucose and having enough. Getting more glucose beyond having enough will not yield further increments in self-control, whereas getting enough glucose to recover from a depleted state will yield improvements until the optimal level is reached.

## REVIEW OF EMPIRICAL FINDINGS

### Reviewing Strategy and Relevant Methods

To test the hypothesis that glucose is one important component of self-control energy, we reviewed a variety of studies that examined a broad set of self-control behaviors. Specifically, we reviewed studies on impulsivity, attention control, emotion regulation, crime and aggression, smoking cessation, the ability to cope with stress, alcohol consumption, and day and evening patterns of self-control. Each of these behaviors is related to self-control. Reviewing each of these areas of research allowed us to test whether problems with glucose impair the general capacity for self-control.

To provide multimethod converging evidence, we also reviewed evidence using a variety of techniques used to assess or manipulate blood glucose levels or assess the ability to use glucose effectively. A glucose testing meter or other similar device is oftentimes used to assess glucose levels. These devices intake a sample of blood and measure the amount of glucose in the blood sample. This method allows researchers to examine the relationship between glucose levels and other factors (e.g., aggressive behavior). There exists an equilibrium between glucose in the bloodstream and the brain (Lund-Anderson, 1979), and thus, it can be assumed that glucose in the bloodstream reflects that in the brain.

Another approach to assess the influence of glucose is to examine the behavior of people prone to experience low glucose levels in comparison to those who are less prone to experience low glucose. People with diabetes or hypoglycemia more frequently experience low glucose than other people. One can examine potential consequences of low glucose by comparing the two groups on various dimensions.

To manipulate glucose experimentally, researchers frequently use a glucose injection. This is accomplished either intravenously by injecting glucose directly into the bloodstream or orally by having participants consume a meal or glucose shake or tablet. Control participants in these studies typically receive a placebo treatment. The effects of increased glucose can be examined by comparing the consequences of receiving glucose versus a placebo. One should note, however, that glucose injections do not necessarily produce the same effects as regular meals outside of the laboratory.

A hyperinsulinaemic glucose clamp is sometimes used to experimentally decrease glucose. The clamp infuses insulin into the bloodstream, thereby decreasing glucose. A similar device that does not alter glucose levels is used among control participants. Any cognitive or behavioral changes that occur as a result of the glucose clamp are presumably attributable to the reduction in glucose.

Other studies have examined the consequences of skipping breakfast, and these studies are relevant to glucose insofar as skipping breakfast reduces available quantities of glucose or undermines the effective use of glucose. Participants are randomly assigned to skip breakfast or asked whether they have spontaneously done so. Skipping breakfast produces glucose levels that are lower than those of participants who eat breakfast because breakfast foods (typically high in carbohydrates) provide the body with glucose to counteract the low levels that occur after the nocturnal fast.

This review does not include studies on hyperglycemia (i.e., when levels of peripheral glucose are abnormally high). Hyperglycemia is not necessarily associated with increased levels of cerebral glucose, because the flow of glucose across the blood-brain barrier is restricted during a hyperglycemic state. In addition, the hypothesis that glucose partly underlies self-control energy implies that glucose levels below an optimal range should impair self-control, whereas glucose levels above the optimal range are not necessarily either detrimental or beneficial to self-control. Research on hyperglycemia therefore does not provide a clear test of the hypothesis that glucose partly underlies self-control energy.

To assess the ability to transport glucose effectively, researchers most frequently use a glucose tolerance test. For this test, glucose levels are assessed after an overnight fast. A glucose load (e.g., a glucose shake) is then administered and glucose levels are assessed at regular intervals afterwards. Being able to transport glucose effectively (i.e., good glucose tolerance) is indicated by glucose levels rising after the glucose load and then returning to baseline. Being unable to use glucose effectively (i.e., poor glucose tolerance) is indicated by glucose levels rising abnormally high after the glucose load or, upon their descent, by dropping well below baseline. One can measure differences in other domains (e.g., criminal behavior) between those who are able and those who are unable to transport glucose effectively.

Another means of examining the effects of being able to transport glucose effectively is to examine the behavior of people with diabetes. Diabetes is associated with poor glucose tolerance or being less effective at transporting glucose, in addition to frequent occurrences of low glucose (e.g., Goetsch, 1989). People with diabetes are less able to use glucose as an energy source than people without diabetes (Pohl, Gonder-Frederick, &

Cox, 1984). One can therefore compare a diabetic population to a nondiabetic population to assess possible consequences of glucose. As stated above, cerebral functioning requires the effective use of glucose so that one can transport peripheral glucose to the brain (e.g., Benton et al., 1996). The consequences of poor glucose utilization are therefore suggestive of the consequences of low (cerebral) glucose. In addition to research on diabetes, research on other disorders associated with poor glucose tolerance is also suggestive of the consequences of poor glucose mobilization.

Based on the hypothesis that glucose is one vital component of self-control energy, we predicted that low glucose levels and poor glucose tolerance would be associated with poor self-control. Diabetes and hypoglycemia should be linked with poor self-control. Self-control should deteriorate under conditions that reduce glucose and improve under conditions that increase glucose. People who are less able to tolerate glucose (e.g., diabetics) should exhibit lower self-control than those who are more able to tolerate glucose.

## Controlling Attention

Attention control is a pervasive and basic form of self-control. Executive control can dictate and choose what information is noticed and processed by the mind, as opposed to letting salience and the environment dictate. In a review of the literature on self-regulation, Baumeister et al. (1994) observed that attention appears to be the front line of control for many problem behaviors, and loss of attention control is a precursor to self-control failure in many different domains. Controlling attention requires self-control because attention automatically orients toward various stimuli in the environment (e.g., Shiffrin & Schneider, 1977). It takes self-control to override these automatic responses so as instead to remain focused on any single task or stimulus (Muraven & Baumeister, 2000). Consistent with the idea that attention control depletes the same energy needed for self-control, research has shown that people are less able to exert self-control after having controlled their attention and that they are less able to control their attention after having exerted self-control (e.g., Vohs et al., 2005; Gailliot & Baumeister, in press; Gailliot et al., 2006; Vohs & Faber, 2004).

One study found evidence consistent with the idea that attention control requires a relatively large amount of glucose (Gailliot, Baumeister, et al., 2007). Participants watched a 6-min video that required them either to control their attention by ignoring certain stimuli appearing on the screen or to watch the video as they would normally, thus not trying to control their attention. Among participants who had controlled their attention, glucose dropped after having watched the video. Among participants who watched without controlling attention, glucose levels did not change. The implication is that controlling attention resulted in the consumption of relatively large amounts of glucose. To be sure, it is more difficult to watch the video while controlling attention than while watching it without such effort, and participants' ratings of task difficulty confirmed that difference. Self-control requires effort, and that makes it difficult. From this and similar studies, it is not easy to infer whether the differences are due to self-control per se or to the general difficulty of performing the task.

The Stroop task is a classic psychological test of attention control. It requires participants to state the color ink in which words are displayed. On incongruent trials, the ink color and meaning of the word are different (e.g., *red* is presented in blue ink), and so participants must exert self-control by directing their attention away from the word's meaning and toward the color ink. One study found that participants whose glucose levels were dropping prior to the Stroop task performed worse (i.e., they had slower reaction times) on incongruent trials than participants whose glucose levels were rising (Benton et al., 1994). Presumably, the Stroop task required glucose, and so decreasing levels of glucose before the task hampered performance. Participants whose glucose levels were rising had more glucose becoming available and hence performed better.

In this same study by Benton et al. (1994), there was no relationship between changing glucose levels and performance on congruent Stroop trials (when the words' meaning and color ink were the same). These trials did not require attention control because participants did not have to override the meaning of the word in order to state the ink color. This finding suggests that the effect of glucose was specific to attention control and not to more general mental capacities, such as processing speed.

Another study assessed glucose levels before, during, and after the Stroop task (Fairclough & Houston, 2004). Participants who used less glucose during the Stroop task made more errors than other participants on the incongruent trials—but there was no difference on the congruent trials, which do not require self-control. Thus, it again seems that glucose is required for successful attention control. Exerting oneself at the self-regulation task led to performing well but required and consumed more glucose.

In another study, participants had to control their attention while watching a video (Gailliot et al., in press). They then completed the Stroop task. Higher glucose levels after having watched the video predicted better performance on the Stroop (responding faster and making somewhat fewer errors). The implication is that having more glucose enabled the participants to perform better on the Stroop. In a similar study, participants first either did or did not control their attention while watching a video. They then received either a glucose or placebo drink, after which they completed the Stroop task. Consistent with the idea that self-control relies on a limited energy source (glucose), doing the attention-control task led to poorer performance subsequently on the Stroop task—but only among participants who received the placebo. The impairments in Stroop performance after having controlled one's attention were eliminated among participants who received glucose. Thus, glucose benefited attention control after a difficult self-control task. The implication is that the first attention-control task depleted glucose, leading to poor performance on the Stroop task, unless a dose of glucose replenished the body's self-control energy.

Converging evidence supports the hypothesis that attention control is highly dependent on glucose. One study administered glucose drinks to participants and found that poor glucose tolerance (indicated by glucose levels remaining high after the person consumed the drink) was associated with poorer performance on a dichotic listening task, which is another classic attention-control task and requires participants to ignore information presented in one earphone in order to track and process the information coming in the other ear (Allen, Gross, Aloia, & Billingsley, 1996). Likewise, diabetes (which is marked by poor glucose tolerance or ineffective mobilization) is associated with increased distractibility and poor attention control among both children and adults (e.g., Lane, Stabler, Ross, Morris, Litton, & Surwitt, 1988; Lustman, Frank, & McGill, 1991; Rovet & Ehrlich, 1988), and a recent review concluded that low blood glucose causes these attentional impairments (Zhao & Liu, 1999).

A few studies have found that hypoglycemia leads to loss of control of attention and poor concentration (Smid et al., 1987; Tager & Shelton, 1943). In one study, participants were deprived of sleep for 1 night (Wu et al., 1991). Before and after the sleep deprivation, they completed an attention-control task that required them to press a button every 3 s. Sleep deprivation decreased glucose metabolism in brain regions associated with attention control and impaired performance on the task, and the reductions in glucose accounted for the task impairments. A lack of sleep thus impaired attention control, apparently by reducing glucose.

Vigilance is another important pattern requiring the control of attention. Experimentally increasing glucose improves attention control and vigilance, among both children and adults (e.g., Benton, 1990; Benton et al., 1987; Moser, Plum, & Buckmann, 1983; cf. Parker & Benton, 1995), as well as feelings of alertness (Wesnes, Pincock, Richardson, Helm, & Hails, 2003). In a study by Benton et al. (1987), children received a glucose or placebo drink and then completed a reaction-time test that required the children to wait for a cue to appear before responding. Later, they played a difficult computer game. The children who received the glucose drink were more successful at sustaining their attention than those who received the placebo, such that they responded faster during the reaction-time task and appeared more focused while playing the computer game.

In another study, participants consumed either a glucose or placebo drink (Benton, 1990). They then watched a screen for 25 min and had to press a button each time a specific digit appeared on the screen. The task thus required participants to control their attention and remain vigilantly focused on the screen. Participants who received the glucose drink performed better on the vigilance task than participants who received the placebo, such that their performance improved much earlier in the task, whereas placebo participants were slower to master the task.

The effects of reducing (rather than increasing) glucose levels on attention control were studied by Smid et al. (1997). Some participants were placed in a hypoglycemic state via a glucose clamp, whereas others were not. Before and after this procedure, participants completed an attention-control task that required them to respond to some information while ignoring other information. Participants in the hypoglycemic state performed worse on this task (as evident by slower reaction times) compared to their baseline performance. Control participants' final performance did not differ from baseline. Thus, experimentally decreasing glucose impaired attention control.

Conversely, interventions that increase glucose should result in improvements in attention control, at least among groups who are at risk for glucose deficiency. Schoolchildren who skip breakfast are presumably at such risk insofar as glucose levels drop overnight and breakfast would be required to replenish them. When grade schools provide free breakfast for their students, attentiveness during class increases as a result (Murphy et al., 2000; cf. Wesnes et al., 2003). Eating breakfast might improve attentiveness in part by increasing glucose or its mobilization.

Not all studies have found that glucose improves attention control (e.g., Flint & Turek, 2003; Manning, Hall, & Gold, 1990). One possible explanation for these seemingly contradictory findings is that glucose might benefit attention control primarily under difficult or demanding situations. In particular, our view is that a dose of glucose may be valuable to replenish a depleted state, but it will have little or no effect when the body already has enough glucose. In one study described by Benton (1990), for instance, a glucose drink (vs. placebo) improved attention during a driving-simulation task, but only toward the end of the task—when it was probably more difficult to sustain attention. In the Benton et al. (1994) study described above, the children completed the experiment toward the end of the school day. The children were probably fatigued by the end of the day and perhaps wanted to go home, and therefore the task may have been especially difficult. Likewise, studies that used very demanding attention-control tasks (e.g., the Rapid Information Processing Task) have typically found that glucose improves performance (e.g., Benton et al., 1994), whereas studies that have used less demanding attention-control tasks (e.g., a task requiring that participants indicated when a square approached a target) have not found any benefit of glucose (e.g., Flint & Turek, 2003). As we noted above, increasing glucose among research participants who already have enough is not likely to yield discernible benefits, and the effects of glucose interventions will be best observed with difficult tasks and other measures that require effortful control by the self.

In sum, a variety of evidence is consistent with the idea that glucose facilitates attention control. Both correlational and experimental evidence links impaired attention control with low glucose or poor glucose tolerance. Attention control is an important form of self-control (e.g., Muraven & Baumeister, 2000), and these results are therefore consistent with the hypothesis that glucose is the energy source of self-control. Furthermore, glucose might be especially likely to influence attention control under demanding situations. This too indicates that self-control is highly susceptible to glucose. Self-control demands should be highest when attention control is the most difficult, as one must exert additional controlled effort.

## Emotion Regulation

Emotions are powerful responses that can be highly aversive and disruptive, both to individual functioning (such as when emotions bias decision making) and interpersonal functioning (such as when inappropriate emotional displays offend or alienate interaction partners). Hence, many people seek to control their emotions, particularly in regard to concealing or stifling strong states of aversive emotion (Larsen, 2000). Emotion regulation relies on self-control energy such that suppressing or amplifying emotions has been shown to drain self-control resources, resulting in poorer self-control afterward (e.g., Baumeister et al., 1998; Finkel & Campbell, 2001; Muraven et al., 1998; Schmeichel, Demaree, Robinson, & Pu, in press; Vohs et al., 2005).

Emotion regulation appears to be especially sensitive to glucose. The flow of cerebral glucose increases during aversive emotional states (e.g., anxiety), and it has been argued that this occurs because additional glucose is required to cope with the aversive emotion (Barglow, Hatcher, Edidin, & Sloan-Rossiter, 1984; Kety, 1950). Coping with or regulating emotions may therefore require relatively large amounts of glucose.

Consistent with the idea that emotion regulation is especially sensitive to glucose, people with poor glucose tolerance exhibit signs of poor emotion regulation. For instance, diabetic patients with especially poor glucose tolerance are more likely to have a major depressive or anxiety disorder than diabetic patients with better glucose tolerance (e.g., Eren, Erdi, & Özcankaya, 2003; Fris & Nanjundappa, 1986; Lustman, Griffith, Clouse, & Cryer, 1986; Popkin, Callies, Lentz, Colon, & Sutherland, 1988; Wells, Golding, & Burnam, 1989). Diabetic patients are more likely than nondiabetic patients to throw temper tantrums (Fabrykant & Pacella, 1948; Wilson, 1951) and be emotionally labile (Barglow et al., 1984). Similar to diabetes, hypoglycemia is associated with high anxiety and low levels of happiness (Wredling, Theorell, Roll, Lins, & Adamson, 1992). These findings fit the overall pattern of low glucose or poor glucose tolerance being associated with poor regulation of mood and inability to escape from unpleasant feelings. Likewise, numerous studies demonstrate that depressed individuals exhibit poorer glucose tolerance than other individuals (e.g., Mueller, Heninger, & McDonal, 1968; van Pragg & Leijnse, 1965). A broad assortment of evidence suggests that depression stems in part from being unable to regulate moods effectively (Tomarken & Keener, 1998). It therefore seems plausible that poor glucose tolerance among depressed individuals might impair their ability to regulate mood, thereby prolonging their depressed state.

Evidence concerning emotional states less extreme than depression also suggests that glucose facilitates emotion regulation. One study, for instance, found that children with a glucose deficiency disorder (i.e., glucose-6-phosphate dehydrogenase deficiency) were rated by their teachers as having more mood swings than children without the disorder (Meijer, 1984). It is plausible that the glucose disorder increased the tendency to experience mood swings as a result of ineffective emotion regulation.

In addition to poor glucose tolerance, low levels of glucose have been linked with signs of poor emotion regulation. Several studies have found that when glucose is low, people are more likely to report feeling anxiety, irritability, tension, and other bad moods as compared to when glucose is high (e.g., Barglow et al., 1984; Benton & Owens, 1993; Hepburn, Deary, MacLeod, & Frier, 1996; Taylor & Rachman, 1988; Yaryura-Tobias & Neziroglu, 1975; cf. Reid & Hammersley, 1995; Scholey &

Kennedy, 2004). The relationship between low glucose and negative mood states might be especially strong among those with diabetes (Gonder-Frederick, Cox, Bobbitt, & Pennebaker, 1989). To the extent that people attempt to regulate their moods and avoid bad moods, low glucose can be seen as undermining mood regulation and hence contributing to both the unwarranted persistence and the inappropriate expression of emotional distress. People with diabetes are especially likely to experience low levels of glucose, and so they possibly have even greater difficulty regulating their moods when their glucose is low.

Additional evidence that glucose improves emotion regulation comes from studies that have manipulated glucose levels. In two separate studies, participants were placed under either a glucose clamp that reduced glucose levels or a control device that did not change glucose levels (Gold, MacLeod, Deary, & Frier, 1995; McCrimmon, Frier, & Deary, 1999). When participants were under the glucose clamp and their glucose had been reduced, they reported being in more negative and less positive mood states as compared to the control condition in which glucose levels were not reduced. Both the glucose clamp and the control devices penetrated the skin and therefore caused some discomfort. Participants therefore may have had to regulate their emotions in order to remain calm and relaxed. When the clamp reduced available glucose, they were less successful at regulating their moods in this way.

If low glucose reduces the likelihood of regulating negative mood states, then increasing available glucose should lead to more positive mood states. Consistent with that hypothesis, participants in one study reported feeling more positive emotionally after consuming a glucose drink than participants who consumed a placebo drink (Benton & Owens, 1993). Another study found that children who received a glucose drink (vs. a placebo) toward the end of the school day appeared less irritated and frustrated when playing an impossible computer game (Benton et al., 1987). Playing the impossible game was likely very frustrating for these children, perhaps especially at the end of the day, and so it should have required some self-regulatory effort to control their frustration. When they consumed the glucose shake, the children appeared more likely to do so.

A similar study found that adults who played an impossible computer game showed fewer signs of frustration when they had previously consumed a glucose shake rather than a placebo (Benton & Owens, 1993). This effect seemed to occur, however, primarily among participants who had been provoked by the experimenter (the experimenter told these participants that most people were better at playing the game by that time). The experimenter's remark presumably increased participants' frustration and other negative feelings, and so they had to self-regulate to a greater extent in order to control their frustration. The glucose shake seemed to facilitate such emotion regulation.

Work in our own laboratory has indicated that glucose improves coping with induced aversive emotional states, namely, death anxiety. There is some evidence that after thinking about their own death, people effortfully cope with thoughts of death (Arndt, Greenberg, Solomon, Pyszczynski, & Simon, 1997; Greenberg, Arndt, Schimel, Pyszczynski, & Solomon, 2001). This coping requires self-control and therefore impairs self-control afterwards (Gailliot & Baumeister, 2005). Hence, we found in one study that after thinking about death rather than a control topic, participants quit sooner on a task requiring effortful persistence (Gailliot & Baumeister, 2007). Drinking a glucose shake, however, eliminated this effect, whereas drinking a placebo did not. Coping with the thought of death therefore reduced self-control, but glucose replenished self-control.

Other research shows that death anxiety can increase defensiveness in the form of increased in-group favoritism and out-group derogation (e.g., Baldwin & Wesley, 1996). Consistent with this idea, we found that thinking about death rather than a control topic increased defensiveness about one's culture among participants who had consumed a placebo drink but not among participants who consumed a glucose drink (Gailliot, 2005). When participants had additional glucose, they presumably coped more easily with the aversive thought of death and therefore did not respond defensively.

The effects of skipping breakfast on mood were investigated by Smith, Clarka, and Gallaghera (1999). Participants were randomly assigned to eat or to skip breakfast. Participants who had eaten breakfast later reported being in a more positive mood than those who had skipped breakfast. Eating breakfast perhaps improved mood and hence self-regulation by increasing glucose or improving the effective use of glucose.

Thus, a review of the evidence on glucose and emotion suggests that glucose might improve emotion regulation. Correlational and experimental evidence shows that low levels of glucose and poor glucose tolerance are associated with a greater incidence of negative mood states. Alternatively, it is possible that glucose might improve mood directly, and some of the published evidence seems consistent with this possibility as well. Our hypothesis for future testing is that glucose will mainly elevate mood by means of self-control when people seek to regulate their moods. Despite the alternative possibility, findings on glucose and mood are consistent with the overall trend of glucose improving self-control. Glucose might benefit self-control in the form of emotion regulation.

## Crime, Aggression, and Violence

One of the most important functions of self-control is to enable people to conform to the rules of their society, including laws and moral restrictions that condemn violent, destructive, and exploitative actions. Conversely, low self-control is a leading cause of criminal and aggressive behavior (e.g., Baron, 1976, 1983; DeWall, Stillman, Baumeister, & Gailliot, in press; Gottfredson & Hirschi, 1990; Pratt & Cullen, 2000; Stucke & Baumeister, 2006; Tangney et al., 2004). Criminal acts provide immediate gratification, whereas the long-term costs (such as prison) generally outweigh the long-term benefits, and they require little to no planning. Criminal acts are therefore the opposite of behaviors that require good self-control, which typically entail delaying gratification to secure long-term benefits. Rather than working regular hours and saving money, for instance, a criminal might lounge around at home and then rob a convenience store to get money for paying rent or purchasing beer. Contrary to the impression one gets from the carefully planned crimes in popular films, most crimes are impulsive acts initiated on the spur of the moment when an opportunity was perceived, and so low self-control contributes to these impulsive acts (Gottfredson & Hirschi, 1990). Similar to criminal behavior, aggressive or violent acts are caused by low self-control because the individual experiences a desire to behave aggressively but fails to stifle that desire and refrain from behaving aggressively (DeWall et al., in press; Stucke & Baumeister, 2006). If glucose supports self-control, then glucose problems could release criminal and aggressive impulses.

Low glucose and poor glucose tolerance contribute to criminal and aggressive behavior. Numerous case studies have shown that hypoglycemia is commonly experienced among unlawful individuals (e.g., shoplifters; e.g., Hill & Sargant, 1943; Moyer, 1976; Wilder, 1947; Wolfgang & Ferracuti, 1967). Wilder (1940) found that individuals with low glucose committed a wide variety of crimes, including child or spouse abuse, false fire alarms, embezzlement, destruction of property, and traffic violations. Other case studies have found that hypoglycemia is common among those who commit illegal sex acts, such as public masturbation and exhibitionism (Elia & Goldstein, 1932; Powell, 1936). Socially inappropriate though legal behaviors, such as using excessive profanity in public, have been reported to be common during a hypoglycemic state (Greenwood, 1935; Powell, 1936). Though this evidence is largely anecdotal, it is consistent with the idea that low glucose contributes to criminal and socially undesirable behavior.

Stronger, more systematic evidence linking crime to low glucose was provided by Rojas and Sanchi (1941), who assessed glucose levels among delinquents shortly after they were apprehended. Approximately 90% of 129 delinquents exhibited glucose levels below the normal range. Such a high incidence of low glucose suggests that low glucose contributes to delinquency, even though it could be that glucose was depleted during the process of being arrested and possibly during the criminal activity that immediately preceded it.

Glucose tolerance tests have confirmed that adolescent criminals and delinquents have poorer glucose tolerance than noncriminals. Adjudicated adolescents tolerate glucose less effectively than nonadjudicated adolescents (Matykiewicz, La Grange, Vance, Mu, & Reyes, 1997), as do adolescent delinquents compared to nondelinquents (Gans et al., 1990). Many researchers have argued that this relationship is causal such that low glucose among delinquents causes unlawful and other impulsive behavior (e.g., Rojas & Ferrer, 1941).

Glucose has been linked to criminal behavior among adults, too. A history of illegal behavior is one criterion for a diagnosis of antisocial personality disorder (American Psychiatric Association, 1994), and people with antisocial personality disorder have been found to exhibit poorer glucose tolerance than those without any diagnosed disorders (Virkkunen, 1983). Poor glucose tolerance seems especially common among criminal offenders who are impulsive, such as among those who set fires (Roy, Virkkunen, Guthrie, & Linnoila, 1986; Virkkunen, 1984, 1986). It seems possible that poor glucose tolerance leads to criminal behavior among both arsonists and those with antisocial personality disorder. Consistent with this idea, hypoglycemia is unusually common among the Quolla inhabitants of Peru, and their society is marked by relatively high rates of criminal activities, such as throwing rocks and stealing (Bolton, 1979). Social norms among the Quolla clearly discourage such behavior, and rule breakers are punished, so individuals should have some incentive to refrain from criminal acts, yet hypoglycemia presumably undermines criminal restraint and increases criminal behavior.

Thus, a variety of evidence links glucose with increased criminality. To be sure, the data are only correlational, and so it is unclear whether problems with glucose cause criminal behavior or vice versa. Research on aggressive or violent illegal acts, however, suggests that problems with glucose might cause some forms of criminal and antisocial behavior.

Aggression has been linked to glucose in multiple studies. Among the Quolla (who exhibit high rates of hypoglycemia), fighting and other forms of aggression are especially frequent, and individuals with poor glucose tolerance are among the most aggressive (Bolton, 1973). Meijer (1984) found that Israeli students with an illness associated with low glucose (i.e., glucose-6-phosphate dehydrogenase deficiency) were rated by their teachers as being more aggressive than other students. Most likely, the students were more aggressive because the glucose illness undermined aggressive restraint. Thus, some evidence has linked low glucose to increased aggressive behavior.

Other evidence shows that poor glucose tolerance predicts aggressive behavior. Adolescents convicted of aggressive offenses, for instance, scored higher on a personality measure of aggression than their less aggressive peers, and they also performed worse on a glucose tolerance test (Matykiewicz, La Grange, Vance, et al., 1997). Their inability to tolerate glucose might have contributed to their aggressive tendencies. Likewise, adolescents with diabetes appear more aggressive than others (Swift, Seidman, & Stein, 1967). Among incarcerated individuals, the ability to tolerate glucose has been shown to predict who had versus had not committed violent and aggressive acts prior to incarceration (e.g., Virkkunen, 1982, 1984, 1986; Virkkunen & Huttunen, 1982).

Laboratory and scenario studies have likewise linked glucose to aggression. In two studies, participants completed a glucose tolerance test and then indicated how they would respond to several hypothetical scenarios describing an interpersonal conflict (Benton, Kumari, & Brain, 1982; Donohoe & Benton, 1999a). Participants with poor glucose tolerance were more inclined than others to favor aggressive responses. Another study associated poor glucose tolerance with increased fantasy aggression, as indicated by responding with more aggressive words on a sentence completion task (Bolton, 1979). Thus, individuals with poor glucose tolerance exhibit an aggressive temperament and seem predisposed toward violence.

Other evidence indicates a causal relationship between glucose and aggression. Participants in one study received either a glucose or placebo drink (Benton & Owens, 1993). They then played an impossible computer task. The task was sufficiently frustrating to cause some participants to bang on the computer equipment or curse aloud, but such aggressive responses were much reduced among participants who had had the glucose drink. An additional and very revealing finding emerged in a condition in which the experimenter provoked the participants' anger by suggesting that they should have performed better. The beneficial effect of glucose for reducing aggression was strongest in that condition. This suggests that glucose allowed participants to control their aggression, especially when the combination of frustrating failure and provoking criticism gave them the strongest urge to behave aggressively.

Evidence from case studies portrays a similar picture. There have been several reports of hypoglycemia being associated with aggressive acts, such as murder and torture (Moyer, 1976; Wolfgang & Ferracuti, 1967; for a review, see Wilder, 1947). In one instance, a hypoglycemic individual was reported to have pushed a woman off of a trolley car (Henner, 1936). In another, a hypoglycemic man stabbed his mother to death (Hill & Sargant, 1943). Partially through the police investigation, the researchers were able to determine that low glucose was one likely cause of the aggression. Hypoglycemic individuals seem to become aggressive when glucose levels are low, and they stop acting aggressively when glucose increases. Such patterns support the conclusion that glucose permits the self-control of aggressive impulses.

Glucose imbalances have also been linked with increased aggressive behavior, as assessed by reports from the participants, their families, and their psychiatrists (Yaryura-Tobias & Neziroglu, 1975). Treating the glucose imbalance with a low-carbohydrate, high-protein, high-fat diet helped reduce aggressive behavior, suggesting that the glucose imbalance was the cause of their aggression.

Several other studies have shown that improving the diet of incarcerated adolescents or adult prisoners reduces the incidence of violence in prison (e.g., "New Studies Show Strong Link," 2004). It is plausible that the reduction in prison violence by improved diet is partially attributable to improved glucose levels or glucose tolerance.

A remarkable longitudinal field study of criminal recidivism by Virkkunen, DeJong, Bartko, Goodwin, and Linnoila (1989) sought to predict violent criminal acts over several years after release from prison. It found that glucose tolerance correctly predicted further violence for 84% of criminals. In view of the many extraneous variables and error variance in measuring criminal behavior, it would be hard to ask for a stronger result. Prisoners who exhibited poor glucose tolerance were much more likely to commit violent acts years later as compared to prisoners with better glucose tolerance. This relationship did not appear to be attributable to any other relevant physiological or demographic factors. That glucose tolerance at one point predicted violent acts at a later point is consistent with the interpretation that poor glucose tolerance partially caused the violent acts. Individuals who exhibit poor glucose tolerance as a stable trait seem predisposed to behave violently when opportunities beckon.

Thus, a relatively wide body of evidence suggests that low glucose and poor glucose tolerance lead to aggressive, antisocial, and even criminal behavior. Low glucose and poor glucose tolerance are associated with increased aggression. This association is strengthened by the convergence across methods used to measure aggression, including self-report and objective measures of dispositional aggression, paper-and-pencil responses, and behavioral data. Both correlational and experimental evidence links glucose with aggression, thereby suggesting that problems with glucose cause aggressive behavior. The overall pattern of results is consistent with the hypothesis that glucose is the fuel for self-control. It is well established that low self-control causes crime and aggression (e.g., Gottfredson & Hirschi, 1990; Pratt & Cullen, 2000), and it seems that glucose improves self-control and thereby contributes to a reduced level of aggressive and criminal behavior.

## Impulsive Versus Socially Desirable Behavior

A central and ongoing task of self-control is to restrain impulses from causing problem behaviors. Most people experience problematic or inappropriate impulses occasionally, and self-control is therefore vital for restraining these. It takes self-control to resist immediate temptation and instead pursue long-term goals (e.g., Mischel et al., 1988; Shoda et al., 1990). If self-control depends on glucose, then problems with glucose should increase impulsivity, the essence of which is acting on the basis of here-and-now feelings, desires, and circumstances, even if at the expense of long-term goals, enlightened self-interest, and socially desirable standards.

Some evidence has linked impulsivity with glucose. Numerous case studies have suggested that hypoglycemia causes individuals to become more impulsive (e.g., Tager & Shelton, 1943). One study examined glucose levels while participants fasted, using questionnaire measures to assess past behavior (Svanborg, Mattila-Evenden, Gustavsson, Uvnäs-Moberg, & Åsberg, 2000). Among men, lower glucose levels were related to increased impulsivity across a broad range of behaviors, such as acting on the spur of the moment, lying, sexual promiscuity, arriving late to work, behaving recklessly, being selfish, and exploiting others. Glucose levels were not significantly related to impulsivity among women, though the failure may have been due to a floor effect insofar as the women reported relatively few impulsive behaviors generally.

The hypothesized link between glucose and impulsivity has also emerged in our own laboratory work. In one study, White participants discussed their views toward controversial racial issues with either a White or Black experimenter (Gailliot, Baumeister, et al., 2007). It takes self-control to refrain from impulsively expressing negative attitudes or prejudice during an interracial interaction (and to avoid saying anything that anyone else might erroneously but embarrassingly construe as prejudiced). Discussion with the Black experimenter caused glucose levels to drop. Talking to a White experimenter about the same topics had no discernible effect on glucose. Moreover, these effects were mainly found among participants whose habitual motivation to avoid stereotypes and prejudice was low, and so the task of discussing controversial racial issues with a Black experimenter was most onerous (see Gailliot, Plant, Butz, & Baumeister, 2007; Plant & Devine, 1998; Richeson & Shelton, 2003; Richeson & Trawalter, 2005; Richeson et al., 2005), hence depleting their available glucose. In contrast, participants who habitually stifle prejudicial or stereotyped thoughts have less difficulty doing so under highly charged circumstances of interracial discussion.

In three other studies in our lab, participants completed an initial self-control task by either controlling their attention, regulating their emotions, or suppressing their thoughts (Gailliot, Baumeister et al., 2007). They then completed a task requiring effortful persistence, namely, working on a figure-tracing task. In fact, the task was impossible, but participants could not ascertain this. As a measure of self-control, we assessed how long they persisted. The impulsive response would be to give up relatively early, and persistence therefore served as a measure of impulsivity. In each study, a higher level of glucose after completing the initial self-control task predicted longer persistence. Glucose presumably allowed participants to overcome the impulse to give up.

We have also found that ingesting glucose reduced impulsivity after an initial self-control task (Gailliot, Baumeister, et al., 2007). Participants first completed either a thought-suppression task (suppressing thoughts of a white bear) or a control task. They then received either a glucose drink or looked at magazines. Last, their self-control was measured by persistence on a frustrating (actually impossible) task. The thought-suppression task was designed to deplete self-control resources, and sure enough it did lead to quitting sooner on the impossible task as compared to a task that did not require thought suppression. However—and crucially—the glucose drink eliminated this effect. The implication is that the thought-suppression task depleted glucose, whereas getting a drink of glucose replenished this supply and thereby increased persistence.

Like low glucose levels, poor glucose tolerance has been linked to increased impulsivity. People with intermittent explosive disorder, which is marked by seemingly uncontrollable acts of destructive impulsivity such as smashing or breaking objects, seem worse at mobilizing glucose than others, as assessed by a glucose tolerance test (Virkkunen, Nuutila, Goodwin, & Linnoila, 1987). People with diabetes are more impulsive than those without diabetes (Lustman, Frank, & McGill, 1991). Diabetics are more likely than nondiabetics to display an opportunistic and explosive temperament style (as assessed by personality measures), defined by being less able to work toward long-term goals and persist on difficult or frustrating tasks. Arsonists likewise demonstrate poor glucose tolerance, and arson is viewed as a disorder of impulse control (Virkkunen, 1984).

Converging evidence linking impulsivity with glucose comes from studies on nutrition. Children who experience food insufficiency or hunger are more likely than others to behave in impulsive ways, such as being hyperactive in class, arriving late to class, and talking during inappropriate times (Murphy et al., 1998). It seems plausible that part of this relationship might be attributable to food insufficiency reducing or interfering with the use of glucose. Without a proper supply of glucose, these children seem less likely to follow norms of appropriate classroom behavior.

Thus, some studies have shown a link between glucose and impulsivity. These studies are limited, to be sure, insofar as most relied on correlational evidence. Although they are consistent with the hypothesis that problems with glucose cause impulsivity, they are also amenable to the reverse interpretation that impulsive action causes low glucose or to third-variable explanations. One review on impulsive behavior has, however, concluded that low glucose is probably a cause of impulsivity (Linnoila, Virkkunen, George, & Higley, 1993). It thus seems most likely that problems with glucose contribute to impulsivity, though more evidence demonstrating a causal relationship would be desirable.

The overall pattern of results is thus broadly consistent with the idea that self-control relies on glucose as an energy source. Low glucose and poor glucose tolerance are associated with increased impulsivity, often in the form of increased socially inappropriate behavior. The most likely explanation is that glucose problems cause impulsivity, although the data are not sufficient to support a strong causal explanation.

## Alcohol Use

Consuming alcohol clearly impairs most forms of self-control (for reviews, see Hull, 1981; also Baumeister et al., 1994). By and large, people fail to exert self-control when they drink. For example, people are more prone to overeat, overspend, and engage in illicit or inappropriate sexual acts when they have consumed alcohol. Alcohol causes people to indulge themselves in immediate gratifications and disregard long-term goals and plans. Might glucose at least partially mediate the effect of alcohol on impaired self-control?

Alcohol consumption reduces glucose metabolism throughout the body and brain (e.g., Altura, Altura, Zhang, & Zakhari, 1996; Wang et al., 2000; Zhu, Volkow, Ma, Fowler, Wang, & Gene-Jack, 2004). Kokavec and Crowe (2003) found that glucose dropped after participants had consumed white wine after a meal. Furthermore, alcohol is especially likely to reduce the flow of glucose to brain regions that underlie self-control (e.g., the frontal cortex; Volkow et al., 1990). It is therefore possible that alcohol consumption causes failures in self-control by reducing glucose in brain regions that underlie self-control. Indeed, some researchers has posited that impairments to overall cognitive functioning following alcohol consumption stem from reductions in glucose (Altura et al., 1996).

More direct evidence also suggests a link between alcohol, glucose, and self-control. Participants in one study consumed alcohol or a nonalcoholic placebo and then completed a divided-attention task (Haier et al., 1999). As noted above, controlling attention is highly demanding, especially under difficult circumstances, and requires self-control (e.g., Muraven & Baumeister, 2000). Performance on the divided-attention task can therefore be viewed as a measure of self-control. Consistent with the idea that alcohol impairs self-control because of lowered glucose levels, participants who drank alcohol performed worse on the divided-attention task than those who drank the placebo. This effect seemed attributable to reductions in cerebral glucose metabolism among those who drank alcohol.

Additional evidence indicates that low blood glucose and poor glucose tolerance are associated with alcohol abuse and violent or criminal behavior among both adolescents and adults (Linnoila & Virkkunen, 1992; Matykiewicz, La Grange, Reyes, Vance, & Wang, 1997). One possibility is that these individuals' drinking disrupts the transportation of glucose throughout the body and brain, thereby causing failures in self-control (e.g., violent and criminal behavior).

The ability to refrain from drinking should be directly dependent on self-control. Refraining from drinking requires self-control to break the habit of drinking and restrain the recurrent desires to drink. Low glucose seems to undermine drinking restraint. Individuals who exhibit poor glucose tolerance and criminal and violent behavior are more likely than others to abuse alcohol (e.g., Linnoila & Virkkunen, 1992). Poor glucose tolerance might therefore lead individuals both to abuse alcohol and to engage in criminal and violent behavior.

Likewise, individuals addicted to drugs and alcohol are more likely than others to experience low blood glucose (see Wright, 1977), and administering glucose to recovering alcoholics seems to reduce their alcohol consumption (Kissin & Gross, 1968). The implication is that low glucose makes it less likely that one will refrain from using drugs and alcohol, and therefore restoring glucose to higher and optimal levels increases drug and alcohol restraint.

Additional evidence that low glucose reduces drinking restraint comes from studies showing that people with diabetes are more likely to have alcohol-use disorder than people without diabetes (e.g., Goodwin, Hoven, & Spitzer, 2003; Rehm et al., 2003). To be sure, the link is a correlation, and the causal arrow could point in either direction. However, several studies suggest that alcohol use does not cause diabetes (e.g., Saremi, Hanson, Tulloch-Reid, Williams, & Knowler, 2004; cf. Wannamethee, Shaper, Perry, & Alberti, 2002), and so diabetes might lead to excessive alcohol use if, in fact, diabetes and alcohol use are causally related. Thus, low glucose and poor glucose tolerance might undermine efforts toward restraining alcohol consumption.

In summary, several studies have shown that alcohol reduces glucose. Given the link between self-control and alcohol, it seems reasonable to infer that alcohol might undermine self-control in part by reducing glucose or impairing the effective transportation of glucose. In addition, some research is consistent with the idea that low glucose or poor glucose tolerance reduces drinking restraint. This also suggests that self-control relies heavily on glucose: When a sufficient amount of glucose is not available, people might have a more difficult time limiting their consumption of alcohol. To be sure, the effects of alcohol are multifarious, and thus any link between self-control and alcohol likely could involve not only glucose but other factors (e.g., insulin release).

### Smoking Cigarettes

Quitting smoking requires self-control because a person must override the habit of smoking and ignore any urges or impulses to smoke (e.g., Baumeister et al., 1994). Several studies have examined the relationship between glucose and smoking cessation, and the majority of these studies have found that glucose increases the likelihood of successfully quitting smoking. For instance, research participants in one study who received glucose tablets for 1 month were more likely to remain abstinent during that month as compared to participants who received placebo tablets (West & Willis, 1998). In fact, that study found that glucose tablets were more effective than nicotine patches at helping smokers remain abstinent. Based on these and other findings, one recent and thorough review concluded that glucose tablets are an effective means of helping people to quit smoking (West, 2001).

Glucose has also been found to reduce the subjective urge to smoke (e.g., Berlin, Vorspan, Warot, Maneglier, & Spreux-Varoquaux, 2003; West, Courts, Beharry, May, & Hajek, 1999; cf. Jarvik, Olmstead, Schneider, Iwamoto-Schaap, & Madsen, 1998). This could be because glucose in some way directly reduces the actual bodily craving for nicotine, or it could reflect good self-control in the form of stifling one's urges. Past research seems unable to tease apart these two possibilities.

Though it is plausible that glucose directly reduces urges to smoke, independent of self-control, there is some evidence that the benefits of glucose to smoking cessation are not caused by direct reduction in physical cravings for nicotine. In one study examining smoking cessation over 4 weeks, participants received either glucose or placebo tablets and wore either a nicotine or placebo patch (West & Willis, 1998). Participants who received the glucose tablets smoked fewer cigarettes than participants who received the placebo tablets. The strength of the urge to smoke did not differ between those who received glucose and those who received the placebo tablets. Glucose, therefore, did not seem to improve smoking cessation by influencing the urge to smoke. Moreover, participants who received glucose tablets and a nicotine patch smoked fewer cigarettes than those who received the placebo tablets and a nicotine patch. The nicotine patch reduced participants' urges to smoke (as compared to the placebo), yet glucose reduced smoking even among participants who received the nicotine patch. Glucose improved smoking cessation even when the physical craving for nicotine had been minimized by the nicotine patch. Thus, although it is unclear whether glucose reduces the urge to smoke directly or indirectly by enhancing the ability to control such urges, glucose seems to improve smoking cessation regardless of whether the urge to smoke is reduced. It is therefore plausible that glucose facilitates the self-controlled ability or motivation to override the well-entrenched routine of smoking and refrain from lighting up.

Other explanations, aside from self-control, also seem unable to account for findings showing that glucose improves smoking cessation. For example, glucose does not appear to facilitate smoking cessation by reducing sick feelings or nausea (West et al., 1999). The effect of glucose on smoking cessation seems more pronounced among heavy than light smokers (e.g., Berlin et al., 2003; Harakas & Foulds, 2002), consistent with the idea that glucose facilitates smoking cessation by improving self-control. For heavy smokers, quitting smoking should be especially difficult and taxing because it involves overriding a well-entrenched routine of smoking and coping with relatively strong urges to smoke.

The idea that glucose reduces hunger cravings and consequently cigarette cravings is, however, another explanation that might account for the relationship between glucose and smoking cessation (e.g., McRobbie & Hajek, 2004; West, 2001). One possibility is that smokers learn to interpret hunger cravings as nicotine cravings, and so by reducing hunger, glucose reduces how often smokers believe they crave nicotine. This explanation can potentially account for some but not all of the findings. Some studies have found that glucose improves smoking cessation, but that this effect is not related to hunger cravings (e.g., Berlin et al., 2003). Self-control gains plausibility as an explanation insofar as reduced hunger cravings do not mediate between glucose and smoking cessation.

Thus, several studies suggest that glucose facilitates smoking cessation, although these findings are more subject to alternative explanations than with most of the other self-control phenomena we have reviewed. Despite these ambiguities, the findings are very consistent with the idea that glucose fosters greater self-control.

### Coping With Stress

Coping with stress requires self-control because it requires that people effortfully control their attention, thoughts, and emotions (Baumeister et al., 1994; Glass, Singer, & Friedman, 1969; Hancock & Warm, 1989; Lazarus & Folkman, 1984; Wegner & Pennebaker, 1993). People who more capably exert self-control in this fashion therefore cope with stress more effectively than others (Gailliot et al., 2006; Muraven & Baumeister, 2000). Glucose might contribute to the success or failure of such coping efforts.

In the face of stress, the body attempts to cope by converting stored energy into glucose and releasing it into the bloodstream, thereby increasing the flow of glucose to the brain, because stressful situations demand increased cognitive activity (e.g., Alvarez, Portilla, Gonzalez, & Ezcurra, 1989; Goetsch, 1989; Guyton, 1976; Hall & Brown, 1979; Schneiderman & Skyler, 1995). Glucose is thus mobilized to provide the brain with sufficient fuel to respond to and cope with stress. Stress is an inner signal of threat and hence of the need to cope, and so stress triggers the body to release more glucose.

Glucose has been shown to increase in response to stressful situations such as school examinations (e.g., Goetsch, Wiebe, Veltum, & Van Dorsten, 1990). In a study by Armario, Marti, Molina, de Pablo, and Valdes (1996), college students exhibited increased levels of glucose prior to taking an exam on 2 different days compared to a day in between the two exams. Presumably, the body increased its production and use of glucose to fuel efforts toward coping with the stressful exams. In further support of the hypothesized link between glucose and coping, self-reported measures of anxiety predicted levels of glucose. Students who reported being the most anxious also exhibited the highest levels of glucose. This suggests that the amount of available glucose increased in proportion to the effort required to cope with anxiety. Likewise, glucose levels tend to increase on days when people are stressed out compared to days when they are not (e.g., Aikens, Wallander, Bell, & McNorton, 1994; Goetsch, Abel, & Pope, 1994).

Some findings indicate that glucose is reduced after the person has coped with stress, consistent with the view that effective coping consumes glucose. Hall and Brown (1979) assessed college students' glycemic response to a stressful exam. Glucose was significantly higher 15 min before the exam compared to 1 week before or 3 weeks after the exam, and glucose decreased significantly by the end of the exam. It seems likely that coping with the stress of the exam contributed to the decline in glucose. This is consistent with the findings suggesting that self-control and executive function activities deplete a psychological resource.

Another study found that glucose levels initially rose during a stressful situation (listening to loud noise) and later dropped (Finkel & Poppen, 1948; cf. Edwards & Yates, 1985). Again, glucose probably increased in the face of the stressful situation and was consumed as participants had to cope.

Dispositional differences in stress have also been linked with glucose. People who frequently interpret events in stressful terms, such as those scoring high in neuroticism, exhibit increased levels of cerebral glucose, indicative of greater efforts toward coping (for a review, see Vitaliano, Scanlan, Krenz, & Fujimoto, 1996). Likewise, one study found that higher stress levels were associated with higher glucose levels among caregivers for people with Alzheimer's and also among matched controls (Vitaliano, Scanlan, Krenz, Schwartz, & Marcovina, 1996).

Converging evidence that coping with stress requires and consumes glucose comes from studies that assessed whether experimentally increasing glucose would facilitate coping with stress. If glucose is required to cope with stress, then increasing glucose should generally benefit coping. Participants in one study were given either a glucose or a water drink and then performed a cognitive task under stressful (noisy) or nonstressful conditions (Simpson, Cox, & Rothschild, 1974). The stress impaired performance on the cognitive task, but this impairment was reduced by the glucose drink. The additional glucose presumably facilitated coping with the stressful noise, and so participants performed better on the cognitive task when they had consumed glucose.

Furthermore, participants who received glucose in the stressful condition used up more glucose than those who received glucose in the nonstressful condition. This suggests that glucose was used to cope with the stress, because the amount of glucose used matched the level of stress with which participants had to cope. The superior task performance of participants who drank the glucose (as compared to those who drank water) indicates that the glucose was indeed effective.

Similar evidence of the beneficial effects of glucose for coping were found in another study in which participants completed a visual-tracking task under stressful or nonstressful noise (e.g., Cox, Simpson, & Rothschild, 1973). Prior to this task, participants received a glucose or water drink. The stressful noise impaired task performance only among participants who received water. Those who received glucose performed relatively well on the task, even under the stressful noise. Moreover, completing the task reduced glucose levels, especially among participants under the stressful noise, and glucose dropped the most among participants under stressful noise who received glucose. Thus, several studies suggest that glucose facilitates coping with stress (counteracting its harmful effects) and is consumed in the process.

Additional evidence suggests that impairments in the use of glucose lead to less effective coping. Participants in one study were given a glucose tolerance test (Nagornev et al., 1999). One week later, they completed a psychoemotional test. Participants who exhibited poorer glucose tolerance performed worse on the psychoemotional test such that they exhibited more emotional instability. Furthermore, among individuals who are likely to be stressed out, those with good glucose tolerance experience fewer stressors compared to those with poor glucose tolerance (Peyrot & McMurry, 1992). It is possible that good glucose tolerance allows these individuals to cope better with stress and hence experience less stress. Consistent with this idea, diabetes is associated with high anxiety and difficulty coping with stressful events (e.g., Barglow, Hatcher, Berndt, & Phelps, 1985). Poor glucose tolerance among diabetics might undermine coping efforts.

Just as low glucose or poor glucose tolerance seems to undermine coping with stress, stress also appears to sap the effective use of glucose. Stressful conditions have been shown to undermine glucose tolerance (e.g., Schneiderman & Skyler, 1995), and stress has been shown to make glucose levels less stable among diabetics (Goetsch, 1989). In one study, pregnant woman with diabetes exhibited poorer glucose tolerance in the form of unstable glucose levels during days when they reported experiencing more stress (Barglow et al., 1985). In two other studies, participants were given a carbohydrate shake and then either sat quietly and relaxed or completed some competitive, stressful tasks (Wing, Blair, Epstein, & McDermott 1990; Wing, Epstein, Blair, & Nowalk, 1985). Participants who completed the stressful tasks were less able than others to metabolize the glucose from the shake.

In summary, glucose levels rise in the face of stress, glucose facilitates coping, glucose is reduced after having coped with stress, stress impairs the use of glucose, and poor glucose tolerance is associated with poorer coping. All of these findings converge upon the notion that coping with stress is highly contingent on glucose. Coping with stress requires self-control, and therefore this evidence demonstrates that self-control requires glucose as part of its fuel. Glucose facilitates self-control in the form of coping with stress.

## Diurnal Covariation of Glucose and Self-Control

Diurnal patterns are highly relevant to the hypothesized link between glucose and self-control. Failures at self-control are much more common in the evening and later at night than during the morning or midday (Baumeister et al., 1994). As the day progresses from morning to evening and night, the odds increase that people will break their diets, smoke cigarettes, drink alcohol, commit sexual acts they will later regret, and engage in violent or impulsive crimes. If failures in self-control are related to glucose, then glucose would be expected to be lower and/or used less effectively in the evening than earlier in the day.

Research has confirmed that glucose problems increase as the day progresses. A recent and extensive review demonstrated convincingly that glucose is used less effectively later in the evening than earlier during the day, and furthermore, that glucose use becomes increasingly inefficient as the night progresses (Van Cauter, Polonsky, & Scheen, 1997). More than two decades of empirical research have documented this effect. These studies have used a diverse range of measures and methods, such as oral and intravenous glucose injection, and morning and afternoon meal consumption. Indeed, the impairments in glucose use throughout the day have been observed so frequently that some researchers have used the term "afternoon diabetes" to describe the effect (Jarrett, Viberti, & Sayegh, 1978). Afternoon diabetes is likely the result of the time of day and not other factors, such as fasting duration (Carroll & Nestel, 1973; Jarrett, Baker, Keen, & Oakley, 1972), and it is probably attributable to changing circadian rhythms. Though glucose is used less effectively as the day progresses, glucose is used most effectively after sleep and upon waking in the morning. This pattern too mirrors patterns of self-control such that the ability to exert self-control is replenished by sleep and is strongest in the morning (Baumeister et al., 1994).

Because people are especially likely to fail at self-control in the evening or late at night, degraded utilization of glucose at those times is highly consistent with the idea that self-control relies on glucose as its fuel. It seems reasonable that the impaired use of glucose in the evening contributes to the failures of self-control. Poor glucose tolerance is associated with poor sleeping habits, antisocial behavior (e.g., violence), and impulsivity among the same individuals (e.g., Virkkunen, 1986). It is possible that these individuals do not get the sleep they need, resulting in impairments of their ability to use glucose and contributing to impulsive and antisocial behavior.

Thus, glucose is used less effectively as the day progresses, which parallels findings that self-control failures become more and more likely as the day wears on. People are likely to fail at self-control during the same times when they are less able to use glucose. We acknowledge that alternative explanations, unbeknownst to us, for self-control fluctuations throughout the day are possible. We only suggest that glucose dynamics might contribute to these fluctuations.

## GENERAL DISCUSSION

A growing body of research has suggested that effortful self-regulation depends on some kind of limited resource that operates like a strength or energy, especially insofar as it becomes depleted when used. Although the behavioral patterns of self-regulatory depletion have been well replicated (for review, see Baumeister, Gailliot, DeWall, & Oaten, 2006), the nature of the energy resource has remained a matter of metaphor and speculation. The purpose of this review was to evaluate the possibility that blood glucose may be one important aspect of that resource. We searched multiple patterns of findings to see whether blood glucose becomes depleted by acts of self-control and related phenomena and whether low levels of blood glucose contribute to failures at self-control.

Multiple findings have indicated that acts of self-control lead to low levels of glucose. Controlling attention, regulating emotions, resisting impulsivity, and coping with stress have all been found to consume relatively large amounts of glucose. Interacting with someone of a different race, performing the difficult Stroop task, coping with the stress of taking an exam, and working under stressful conditions have all been shown to produce drops in glucose as compared to performing tasks that did not require self-control. These findings all point to the conclusion that glucose is depleted by self-control processes.

There was also ample evidence that low glucose contributes to poor self-control. Controlling attention, regulating emotions, coping with stress, resisting impulsivity, and abstaining from cigarettes and alcohol have all been found to be impaired when glucose is low. Low glucose has been linked to less vigilance and greater distractibility (e.g., on a driving-simulation task and on the Rapid Information Processing Task), decreased attentiveness in class among schoolchildren, more negative mood states, a higher incidence of emotional disorders (e.g., depression and anxiety disorders), greater susceptibility to mood swings and temper tantrums, and impaired task performance under stressful conditions. Some evidence indicates that inadequate glucose renders recovering alcoholics less successful at remaining abstinent. Numerous studies have linked low glucose to a wide range of criminal, aggressive, and impulsive behaviors, including child or spouse abuse, false fire alarms, embezzlement, destruction of property, traffic violations, shoplifting, public masturbation, exhibitionism, throwing rocks and fighting, striking computer equipment and swearing, acting on the spur of the moment, lying, sexual promiscuity, tardiness at work and school, reckless and selfish behavior, and reduced effortful persistence. Studies on alcohol have produced additional evidence that low glucose impairs self-control such that alcohol has been found to produce a wide assortment of self-control failures and likewise to reduce glucose. In short, people seem more prone to fail at many forms of self-control when glucose is low.

Complementing the evidence that low glucose has been linked to poor self-control, we found substantial evidence that being unable to use glucose efficiently (poor glucose tolerance) is associated with poor self-control. Poor glucose tolerance has been linked to impaired attention control (e.g., poorer performance on dichotic listening tasks and greater distractibility), poor emotion regulation (e.g., an increased susceptibility to depressive disorder), and greater impulsivity (e.g., breaking objects, being opportunistic). People who are less able than others to tolerate glucose seem to cope less successfully with stress and to be more prone to abuse alcohol or engage in criminal and aggressive acts (e.g., arson, stealing, fighting). Glucose tolerance has even been found to predict violent acts years later. Moreover, self-control failures become increasingly common during the times when glucose is used less efficiently, especially later in the day and evening. Poor glucose tolerance presumably undermines the body's ability to transport requisite amounts of glucose to the brain, thereby impairing self-control.

In summary, a large body of evidence converges upon the hypothesis that self-control is highly susceptible to glucose. Self-control failure seems more likely when periphery glucose levels are too low or when glucose is not efficiently metabolized.

### Alternative Explanations and Issues for Future Research

Thus far, we have presented a broad range of evidence linking self-control to glucose. This evidence indicates that low glucose undermines self-control, thereby unleashing numerous undesirable urges and behaviors. There are, however, a few alternative explanations that could be suggested. One explanation might be that low glucose leads directly to undesirable urges or behaviors rather than undermining self-control. For example, low glucose might increase aggressive urges directly rather than by the indirect route of weakening the inner restraints on aggression.

There are several reasons to doubt the explanation that glucose directly increases undesirable urges. First, evidence indicates that low glucose impairs self-control by reducing metabolic activity in brain regions associated with self-control. For instance, sleep deprivation has been found to impair attention control by reducing glucose in brain regions that underlie attention control (Wu et al., 1991). Depressed individuals exhibit lower levels of cerebral glucose in brain regions responsible for emotion regulation (Drevets, 2000), and increasing the flow of glucose to these regions is associated with recovery from depression (Buchsbaum et al., 1997; S. H. Kennedy et al., 2001; Mayberg et al., 2000). Self-control deteriorates as the day progresses, and this effect might be partially attributable to reductions in the flow of glucose to brain regions that underlie self-control (e.g., Drummond, Gillin, & Brown, 2001; Horne, 1993; Wu et al., 1999). If low glucose increased the strength of people's urges or led directly to undesirable behaviors, then one would expect that low glucose would be associated with increased metabolic activity in other brain regions, but there is no evidence of this. That low glucose reduces activity in brain regions necessary for self-control strongly suggests that low glucose undermines self-control rather than directly increasing undesirable behavior or the strength of one's urges.

A second reason that the urge explanation is inadequate is that the effects of glucose on self-control often seem independent of people's urges. For instance, West and Willis (1998) found that glucose improved smoking cessation but that this effect was not attributable to glucose influencing the urge to smoke. Glucose improved smoking cessation even when participants wore a nicotine patch, which should have reduced the urge to smoke. If glucose improved smoking cessation by decreasing directly the strength of the urge to smoke, then one might expect that glucose would have had much less of an effect when participants' urges had been reduced by the nicotine patch.

Third, researchers have been largely unable to find any correlation between levels of glucose and the intensity of aggressive acts (Yaryura-Tobias, & Neziroglu, 1975). This suggests that glucose is not directly related to the strength of the urge to aggress but perhaps only to the ability to curb one's aggression (self-control). In support of this, Benton and Owens (1993) found that glucose decreased aggression primarily when participants had been provoked by the experimenter—that is, when the urge was externally provoked and the need for aggressive restraint would have been greatest. Similarly, glucose seems to benefit task performance primarily under strenuous circumstances when the most self-control would be needed, such as controlling one's attention toward the end (but not start) of a driving-simulation task (as cited in Keul, Huber, Lehman, Berg, & Jakob, 1982). The difficulty of the task should be inconsequential to glucose if glucose directly influenced urges or behavior.

Another alternative explanation is that in some cases, low glucose might impair self-control by increasing conscious or unconscious feelings of hunger, thereby distracting the participants from self-control tasks or creating a form of cognitive load. This possibility might account for some findings, but it seems unlikely that it accounts for the bulk of the findings. The finding that acts of self-control use up more glucose than acts that do not require self-control (e.g., Gailliot, Baumeister, et al., 2007) suggests that self-control relies heavily on glucose. It seems implausible that self-control tasks use up glucose because they activate thoughts of hunger. A study by Fairclough and Houston (2004) provided additional evidence that speaks against the hunger explanation. They found that using more glucose on the Stroop task was associated with better performance. If low glucose impairs self-control by increasing hunger, then participants who used more glucose (and therefore had lower glucose levels) should have performed worse (and not better) because they should have felt hungrier.

Might subjective feelings of mood or arousal account for the link between glucose and self-control? To be sure, this would not provide an alternative explanation for the link but rather would suggest a potential mediator for the link. Still, studies that have directly assessed mood and arousal have found that neither mood nor arousal accounts for the link between glucose and self-control performance (e.g., Gailliot, Baumeister, et al., 2007).

An issue for future research is whether the effect of low glucose operates by reducing the ability or the motivation for self-control. This may not be easy to establish, insofar as motivation and ability can complement each other. For example, physical tiredness may reduce the muscles' ability to perform but also affects motivation, insofar as tired people may become unwilling to do things that they are still capable of doing, and meanwhile a high motivational incentive may overcome physical tiredness. Similar patterns have been found with self-control: Depleted participants conserve their remaining resources (Muraven, Shmueli, & Burkley, 2006) but can be inspired to exert themselves if the motivational incentives are high (Muraven & Slessareva, 2003).

The most parsimonious conclusion for the findings reviewed here is that effective self-control requires sufficient glucose. Other explanations might account for some of the findings, but they cannot easily account for the bulk of evidence linking self-control to glucose.

One last issue to address is whether the effects of low glucose occur for all individuals from time to time or whether they occur primarily among a subset of individuals. The answer to this issue is unknown at present, and there is reason to suspect either. When glucose in the bloodstream is reduced, the liver should output more glucose into the bloodstream so as to maintain stable glucose levels (Campbell & Reece, 2002). Hence, the effects of glucose on self-control might occur primarily among individuals for whom the liver is ineffective at maintaining stable glucose levels (e.g., individuals with diabetes). However, it is possible that self-control tasks might consume glucose at a rate faster than even the healthiest of livers can output glucose into the bloodstream or among generally healthy individuals who happen to have low glycogen (stored glucose) in the liver for various reasons (e.g., having skipped a meal or two). Future work focusing on individual differences should be helpful in determining whether most people or only a select few people are prone to experience poor self-control as the result of low glucose.

### Implications

A broad range of social behaviors depends on or is affected by self-control processes—and, by extension, depends on or is affected by glucose. Self-control allows people to live in groups and helps provide the basis for sustaining modern culture, such as by facilitating adherence to morals, laws, social rules, and other regulations (Baumeister, 2005). Self-control allows people to suppress prejudice and stereotypes toward out-group members (Gailliot, Plant, et al., 2007; Gordijn et al., 2004; Richeson et al., 2005; Richeson & Shelton, 2003; Richeson & Trawalter, 2005), to avoid criticizing or responding negatively toward their romantic partner (Finkel & Campbell, 2001), to make a good impression on others (Vohs et al., 2005), and to limit their spending and intake of food and alcohol (Kahan et al., 2003; Muraven et al., 2002; Muraven et al., 2005; Vohs & Faber, 2004; Vohs & Heatherton, 2000). If self-control depends on glucose, then glucose should affect prejudice, interpersonal relationship maintenance, impression management, spending, and consumption—and probably many other behaviors too.

Another implication is that effective self-regulation may benefit from eating properly. It is possible that part of the well-established link between poor self-control and criminality (see Gottfredson & Hirschi, 1990; Pratt & Cullen, 2000) may be exacerbated by poor dietary habits. Autobiographical accounts have noted that youth gang members do not typically eat regular meals containing ample vitamins, protein, fruit, and vegetables, but rather they tend to eat unbalanced diets at erratic intervals at fast-food establishments (Shakur, 1993). More systematic studies at juvenile correctional institutions and prisons have found that replacing fried and sugary foods with healthier fare or even just adding vitamin supplements for the inmates resulted in significant and sometimes remarkably large drops in violent and disciplinary incidents in the prison (Gesch, Hammond, Hampson, Eves, & Crowder, 2002; "New Studies Show Link," 2004). It is possible that some foods reduce violence by fostering more efficient glucose use, which in turn may be conducive to effective self-control.

It is also possible that many mental health difficulties might be alleviated by glucose, and indeed, numerous mental disorders have been linked to problems with glucose (e.g., Altschule, Grunebaum, Parkhurst, & Siegel, 1953; Catalano, 1949). Self-control benefits mental health in multiple ways, such as by allowing more effective coping with stress and better emotion regulation (e.g., Gailliot et al., 2006; Tangney et al., 2004). Poor self-control can be a major obstacle to successful therapy insofar as improving one's mental health often requires one to exert self-control by overriding maladaptive thought patterns and behavior. Effective glucose use might be highly beneficial in developing and maintaining good mental health.

The connection between self-control and glucose also suggests that self-control depletion is likely caused in part by reduced glucose. An initial act of self-control has been shown to impair subsequent attempts at self-control (Baumeister et al., 2006). It is possible that an initial act of self-control depletes glucose needed for future attempts at self-control, thereby impairing other self-control efforts. Indeed, recent evidence in our laboratory is consistent with this possibility (Gailliot, Baumeister, et al., 2007). Across seven studies, we found that an initial act of self-control reduced glucose, that lower glucose levels after an initial self-control task predicted poorer performance on a second self-control task, and that consuming a glucose drink eliminated the detrimental effects of an initial self-control task. In two other studies, we found that completing an initial self-control task reduced helping toward strangers compared to a task that did not require self-control. Drinking glucose reduced or eliminated this effect by increasing helping behavior among participants whose self-control had been depleted. The effects of temporary self-control depletion appear partly attributable to glucose.

Though important, glucose is certainly not the only influential factor in self-control processes. Biological and psychological factors aside from glucose (e.g., self-efficacy, socialization, personality) undoubtedly influence self-control as well. Sleep and rest, for instance, have been found to replenish the ability to exert self-control (Baumeister et al., 1994), and it seems likely that the benefits of sleep and rest are somewhat independent from glucose (Van Cauter et al., 1997). For example, a person who has sufficient glucose stores and uses it efficiently but gets little sleep will probably not be very effective at self-control. We thus do not claim that glucose provides a full explanation of self-control processes, but glucose is an important part.

Why might self-control be highly susceptible to glucose? The answer to this question is controversial, and we can only speculate here. One reason, as noted above, is that self-control processes are costly and require relatively large amounts of glucose. Processes that require the most glucose are probably more likely than others to be impaired when glucose is low. When glucose is low, brain regions that carry out operations more central to survival (e.g., breathing, physical coordination) might have first dibs on available glucose and so there is not enough glucose to allow for more advanced mental operations. This is consistent with the idea that abilities that developed last ontogenetically are the first to become impaired when cognitive resources are compromised—the "last-in, first-out rule" (Wilder, 1947).

Another plausible yet speculative reason is that the body might curtail more glucose-demanding endeavors, such as self-control, when glucose is low. An analogy would be how body metabolism slows when people are hungry so as to conserve caloric energy. From an evolutionary perspective, any organism that could effectively reduce its use of glucose when glucose was low and thus conserve this vital energy source could be at a major advantage and therefore more likely than others to survive and reproduce, provided that any temporary loss of higher order cognitive functions stemming from the reduction in glucose was not overly detrimental.

The link between self-control and glucose might also be attributable to the reliance of self-control on the prefrontal cortex (Banfield et al., 2005). Processes that rely heavily on the prefrontal cortex might require more glucose than processes that rely more on other brain regions, because the prefrontal cortex generally underlies effortful, controlled, and executive processes, whereas it is less influential in other processes (e.g., Banfield et al., 2005; Kandel et al., 2000). Indeed, acts that rely on processing in the prefrontal cortex (i.e., decision making, logical reasoning) have been found to rely on a limited energy source such that they either impair executive functioning later on or are impaired by previous acts requiring executive functioning (Baumeister et al., 1998; Schmeichel, Vohs, & Baumeister, 2003; Vohs et al., 2006). It appears that this energy source might be glucose such that high glucose levels have been linked to better performance on tasks relying heavily on the prefrontal cortex. Parker (1995) found that high glucose was associated with superior performance on tasks that required some components of working memory (i.e., the visuospatial scratch pad and the articulatory loop) only when the central executive had to oversee the operation of those tasks. Increased glucose has been shown to facilitate performance on difficult but not easy maze tasks (Donohoe & Benton, 1999b), working memory tasks (D. O. Kennedy & Scholey, 2000), and reaction-time tasks (Owens & Benton, 1994). Keul et al. (1982) showed that giving participants glucose improved their performance near the end but not the beginning of a difficult driving-simulation task, which suggests that the dose of glucose only helped when available fuel had already been depleted. Low or hypoglycemic levels of glucose have been linked with impaired decision making (Blackman, Towle, Lewis, Spire, & Polonsky, 1990), poor planning and behavioral initiation (Lee & Bernicky, 1999), and inflexible thinking (e.g., as needed on the Water Jars test; Donohoe & Benton, 1999b), all of which likely rely on the prefrontal cortex. In contrast, tests of simple psychomotor abilities, which presumably make minimal demands on the prefrontal cortex, seem relatively unaffected by glucose levels (Pramming, Thorsteinsson, Theilgaard, Pinner, & Binder, 1986).

Further evidence for the prefrontal cortex in the link between self-control and glucose is based on the principle that the prefrontal cortex is more important for learning new skills than for executing familiar ones. Haier, Siegel, Tang, Abel, and Buchsbaum (1992) found that playing the challenging video game Tetris at first consumed a relatively large amount of glucose when participants were first learning how to play and hence relying on processing by the prefrontal cortex. After practicing for 1 to 2 months, however, participants consumed less glucose when playing Tetris, and participants who performed the best showed the greatest decreases in glucose use. Thus, after learning the task and hence relying less on processing by the prefrontal cortex, the task used less glucose. We reiterate that the precise physiological mechanisms through which glucose levels influence self-control are unknown at present. Pinpointing these mechanisms might be a fruitful avenue for future research.

## Concluding Remarks

Glucose provides energy for nearly all of the brain's activities, and it is plausible that self-control, as a particularly expensive process in terms of complex brain activity, is especially dependent on glucose. The current review indicates that fluctuations in glucose, including even subtle or minor fluctuations, can have a profound impact on self-control. Self-control successes and failures are driven in part by the quantity of glucose and the ability to use it efficiently.

Glucose is not the only aspect of self-control, or else we would be advocating the patently absurd suggestion that consuming ever greater quantities of sugar snacks is the path to virtuous self-control (and slimness!). But the fluctuations in self-control capacity within an individual do seem to depend on the depletion and replenishment of some resource. Glucose seems a promising candidate for the resource on which willpower seems to depend.

## REFERENCES


Aikens, J. E., Wallander, J. L., Bell, D. S. H., & McNorton, A. (1994). A nomothetic-idiographic study of daily psychological stress and blood glucose in women with Type I diabetes mellitus. *Journal of Behavioral Medicine*, *17*, 535-548.

Allen, J. B., Gross, A. M., Aloia, M. S. & Billingsley, C. (1996). The effects of glucose on nonmemory cognitive functioning in the elderly. *Neuropsychologia*, 34, 459-465.

Altschule, M. D., Grunebaum, H. U., Parkhurst, B. H., & Siegel, E. P. (1953). Mobilization of glucose by phlorhizin in patients with mental disorders. *Archives of Neurology & Psychiatry*, *70*, 235-239.

Altura, B., Altura, B., Zhang, A., & Zakhari, S. (1996). Effects of alcohol on overall brain metabolism. In H. Begleiter & B. Kissin (Eds.), *The pharmacology of alcohol and alcohol dependence. Alcohol and alcoholism* (pp. 145-180). New York: Oxford.

Alvarez, M. A., Portilla, L., Gonzalez, R., & Ezcurra, E. (1989). Insulin response to a short stress period. *Psychoneuroendocrinology*, *14*, 241-244.

Armario, A., Marti, O., Molina, T., de Pablo, J., & Valdes, M. (1996). Acute stress markers in humans: Response of plasma glucose, cortisol and prolactin to two examinations differing in the anxiety they provoke. *Psychoneuroendocrinology*, *21*, 17-24.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Arndt, J., Greenberg, J., Solomon, S., Pyszczynski, T., & Simon, L. (1997). Suppression, accessibility of death-related thoughts, and cultural worldview defense: Exploring the psychodynamics of terror management. *Journal of Personality and Social Psychology*, *73*, 5–18.

Ashton, H. (1982). *Smoking: Psychology and pharmacology*. London: Tavistock.

Baldwin, M. W., & Wesley, R. (1996). Effects of existential anxiety and self-esteem on the perception of others. *Basic and Applied Social Psychology*, *18*, 75-95.

Banfield, J., Wyland, C. L., Macrae, C. N., Munte, T. F., & Heatherton, T. F. (2005). The cognitive neuroscience of self-regulation. In R. F. Baumeister & K.D. Vohs (Eds.), *The handbook of self-regulation* (pp. 63-83). New York: Guilford.

Barglow, P., Hatcher, R., Berndt, D., & Phelps, D. (1985). Psychosocial childbearing stress and metabolic control in pregnant diabetics. *Journal of Nervous & Mental Disease*, *173*, 615-620.

Barglow, P., Hatcher, R., Edidin, D. V., & Sloan-Rossiter, D. (1984). Stress and metabolic control in diabetes: Psychosomatic evidence and evaluation of methods. *Psychosomatic Medicine*, *46*, 127-144.

Baron, R. A. (1976). The reduction of human aggression: A field study of the influence of incompatible reactions. *Journal of Applied Social Psychology*, *6*, 260-274.

Baron, R. A. (1983). The control of human aggression: An optimistic overview. *Journal of Social & Clinical Psychology*, *1*, 97-119.

Baumeister, R. F. (1998). The self. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *Handbook of social psychology* (4th ed.; pp. 680-740). New York: McGraw-Hill.

Baumeister, R. F. (2005). *The cultural animal: Human nature, meaning, and social life*. New York: Oxford University Press.

Baumeister, R. F., Bratslavsky, E., Muraven, M., & Tice, D. M. (1998). Self-control depletion: Is the active self a limited resource? *Journal of Personality and Social Psychology*, *74*, 1252-1265.

Baumeister, R. F., Gailliot, M., DeWall, C. N., & Oaten, M. (2006). Self-regulation and personality: How interventions increase regulatory success, and how depletion moderates the effects of traits on behavior. *Journal of Personality*, *74*, 1773-1801.

Baumeister, R. F., Heatherton, T. F., & Tice, D.M. (1994). *Losing control: How and why people fail at self-regulation*. San Diego, CA: Academic Press.

Baumeister, R. F., Schmeichel, B. J., & Vohs, K. D. (in press). Self-regulation. In E. T. Higgins & A. W. Kruglanski (Eds.), *Social psychology: Handbook of basic principles*.

Benton, D. (1990). The impact of increasing blood glucose on psychological functioning. *Biological Psychology*, *30*, 13-19.

Benton, D., Brett, V., & Brain, P. F. (1987). Glucose improves attention and reaction to frustration in children. *Biological Psychology*, *24*, 95-100.

Benton, D., Kumari, N., & Brain, P. F. (1982). Mild hypoglycaemia and questionnaire measures of aggression. *Biological Psychology*, *14*, 129-135.

Benton, D., & Owens, D. (1993). Is raised blood glucose associated with the relief of tension? *Journal of Psychosomatic Research*, *37*, 1-13.

Benton, D., Owens, D. S., & Parker, P. Y. (1994). Blood glucose influences memory and attention in young adults. *Neuropsychologia*, *32*, 595-607.

Benton, D., Parker, P. Y., & Donohoe, R. T. (1996). The supply of glucose to the brain and cognitive functioning. *Journal of Biosocial Science*, *28*, 463–479.

Berlin, I., Vorspan, F., Warot, D., Maneglier, B., & Spreux-Varoquaux, O. (2003). Effect of glucose on tobacco craving. Is it mediated by tryptophan and serotonin? *Psychopharmacology*, *178*, 27-34.

Blackman, J. D., Towle, V. L., Lewis, G. F., Spire, J-P., & Polonsky, K. S. (1990). Hypoglycemic thresholds for cognitive dysfunction in humans. *Diabetes*, *39*, 828-835.

Bolton, R. (1973). Aggression and hypoglycemia among the Quolla: A study in psycho-biological anthropology. *Ethology*, *12*, 227-257.

Bolton, R. (1979). Hostility in fantasy: A further test of the hypoglycemia-aggression hypothesis. *Aggressive Behavior*, *2*, 257-274.

Bradley, B. P., Phillips, G., Green, L., & Gossip, M. (1989). Circumstances surrounding the initial lapse to opiate use following detoxification. *British Journal of Psychiatry*, *154*, 354-359.

Brownell, K. D., Marlatt, G. A., Lichtenstein, E., & Wilson, G. T. (1986). Understanding and preventing relapse. *American Psychologist*, *41*, 765-782.

Buchsbaum, M. S., Wu, J., Siegel, B .V., Hackett, E., Trenary, M., Abel, L., et al. (1997). Effect of sertraline on regional metabolic rate in patients with affective disorder. *Biological Psychiatry*, *41*, 15-22.

Campbell, N. A., & Reece, J. B. (2002). *Biology*. New York: Benjamin Cummings.

Carroll, K., & Nestel, P. (1973). Diurnal variation in glucose tolerance and in insulin secretion in man. *Diabetes*, *22*, 333-348.

Catalano, A. (1949). High dosage of glucose in the treatment of mental diseases. *Archivio di Psicologia, Neurologia e Psichiatria*, *10*, 87-88.

Cohen, S., & Lichtenstein, E. (1990). Perceived stress, quitting smoking, and smoking relapse. *Health Psychology*, *9*, 466-478.

Cox, T., Simpson, G. C., & Rothschild, D. R. (1973). The blood glucose level and skilled performance under stress. *Journal of International Research Communications*, *1*, 30.

DeWall, C. N., Stillman, T., Baumeister, R. F., & Gailliot, M. T. (in press). Violence restrained: Effects of self-regulation and its depletion on aggression. *Journal of Experimental Social Psychology*.

Donohoe, R. T., & Benton, D. (1999a). Blood glucose control and aggressiveness in females. *Personality and Individual Differences*, *26*, 905-911.

Donohoe, R. T., & Benton, D. (1999b). Cognitive functioning is susceptible to the level of blood glucose. *Psychopharmacology*, *145*, 378-385.

Drevets, W. C. (2000). Neuroimaging studies of mood disorders. *Biological Psychiatry*, *48*, 813-829.

Drummond, S. P. A., Gillin, J. C., & Brown, G. G. (2001). Increased cerebral response during a dived attention task following sleep deprivation. *Journal of Sleep Research*, *10*, 85-92.

Duckworth, A. L., & Seligman, M. E. P. (2005). Self-discipline outdoes IQ in predicting academic performance of adolescents. *Psychological Science*, *16*, 939-944.

Dunbar, R. I. M. (1998). The social brain hypothesis. *Evolutionary Anthropology*, *6*, 178-190.

Edwards, C., & Yates, A. J. (1985). The effects of cognitive task demand on subjective stress and blood glucose levels in diabetics and non-diabetics. *Journal of Psychosomatic Research*, *29*, 59-69.

Elia, H., & Goldstein, J. (1932). Insulin-shock und zentralnervensystem. *Med Klin*, *1*, 542.
Eren, I., Erdi, Ö., & Özcankaya, R. (2003). Relationship between blood glucose control and psychiatric disorders in type II diabetic patients. *Turk Psikiyatri Dergisi*, *14*, 184-191.
Fabrykant, M., & Pacella, B. (1948). Labile diabetes: Electroencephalographic status and effect of anticonvulsive therapy. *Annals of Internal Medicine*, *29*, 860-877.
Fairclough, S. H., & Houston, K. (2004). A metabolic measure of mental effort. *Biological Psychology*, *66*, 177-190.
Finkel, E. J., & Campbell, W. K. (2001). Self-control and accommodation in close relationships: An interdependence analysis. *Journal of Personality and Social Psychology*, *81*, 263-277.
Finkel, A. L., & Poppen, J. R. (1948). Clinical effects of noise and mechanical vibrations of a jet engine on man. *Journal of Applied Physiology*, *1*, 183-203.
Flint, R. W., & Turek, C. (2003). Glucose effects on a continuous performance test of attention in adults. *Behavioural Brain Research*, *142*, 217-228.
Freud, S. (1961a). The ego and the id. In J. Strachey (Ed. & Trans.), *The standard edition of the complete psychological works of Sigmund Freud* (Vol. 19, pp. 12–66). London: Hogarth Press. (Original work published 1923)
Freud, S. (1961b). New introductory lectures on psycho-analysis. In J. Strachey (Ed. & Trans.), *The standard edition of the complete psychological works of Sigmund Freud* (Vol. 22, pp. 7–182). London: Hogarth Press. (Original work published 1933)
Fris, R., & Nanjundappa, G. (1986). Diabetes, depression, and employment status. *Social Science and Medicine*, *23*, 471-475.
Gailliot, M. T. (2005). *A glucose drink reduces defensive reactions to thoughts about death*. Manuscript in preparation.
Gailliot, M. T., & Baumeister, R. F. (2005). *Glucose eliminates worldview defense after mortality salience*. Manuscript in preparation.
Gailliot, M. T., & Baumeister, R. F. (2007). Self-regulation and sexual restraint: Dispositionally and temporarily poor self-regulatory abilities contribute to failures at restraining sexual behavior. *Personality and Social Psychology Bulletin*, *33*, 173-186.
Gailliot, M. T., & Baumeister, R. F. (in press). Self-control and business ethics: How strengthening the self benefits the corporation and the individual. In R. A. Giacalone, C. L. Jurkiewicz, & C. Dunn (Eds.), *Positive psychology, ethics, and social responsibility in organizational life*. Greenwich, CT: Information Age.
Gailliot, M. T., Baumeister, R. F., DeWall, C. N., Maner, J. K., Plant, E. A., Tice, D. M., et al. (2007). Self-control relies on glucose as a limited energy source: Willpower is more than a metaphor. *Journal of Personality and Social Psychology*, *92*, 325-336.
Gailliot, M. T., Plant, E. A., Butz, D. A., & Baumeister, R. F. (2007). Increasing self-regulatory strength can reduce the depleting effect of suppressing stereotypes. *Personality and Social Psychology Bulletin*, *33*, 281-294.
Gailliot, M. T., Schmeichel, B. J., & Baumeister, R. F. (2006). Self-regulatory processes defend against the threat of death: Effects of self-control depletion and trait self-control on thoughts and fears of dying. *Journal of Personality and Social Psychology*, *91*, 49-62.
Gans, D., Harper, A., Bachorowski, J., Newman, J., Shrago, E., & Taylor, S. (1990). Sucrose and delinquency: Oral sucrose tolerance test and nutritional assessment. *Pediatrics*, *86*, 254-262.
Gesch, C. B., Hammond, S. M., Hampson, S. E., Eves, A., & Crowder, M. J. (2002). Influence of supplementary vitamins, minerals and essential fatty acids on the antisocial behaviour of young adult prisoners: Randomised, placebo-controlled trial. *British Journal of Psychiatry*, *181*, 22-28.
Glass, D. C., Singer, J. E., & Friedman, L. N. (1969). Psychic cost of adaptation to an environmental stressor. *Journal of Personality and Social Psychology*, *12*, 200-210.
Goetsch, V. L. (1989). Stress and blood glucose in diabetes mellitus: A review and methodological commentary. *Annals of Behavioral Medicine*, *11*, 102-107.
Goetsch, V. L., Abel, J. L., & Pope, M. K. (1994). The effects of stress, mood, and coping on blood glucose in NIDDM: A prospective pilot evaluation. *Behaviour Research & Therapy*, *32*, 503-510.
Goetsch, V. L., Wiebe, D. J., Veltum L. G., & Van Dorsten, B. (1990). Stress and blood glucose in type II diabetes mellitus. *Behavioral Research Therapy*, *28*, 531–537.
Gold, A. E., MacLeod, K. M., Deary, I. J., & Frier, B. M. (1995). Hypoglycemia-induced cognitive dysfunction in diabetes mellitus: Effect of hypoglycemia unawareness. *Physiology and Behavior*, *58*, 501-511.
Gonder-Frederick, L. A., Cox, D. J., Bobbitt, S. A., & Pennebaker, J. W. (1989). Mood changes associated with blood glucose fluctuations in insulin-dependent diabetes mellitus. *Health Psychology*, *8*, 45-49.
Goodwin, R. D., Hoven, C. W., & Spitzer, R. L. (2003). Diabetes and eating disorders in primary care. *International Journal of Eating Disorders*, *33*, 85-91.
Gordijn, E. H., Hindriks, I., Koomen, W., Dijksterhuis, A., & Van Knippenberg, A. (2004). Consequences of stereotype suppression and internal suppression motivation: A self-regulation approach. *Personality and Social Psychology Bulletin*, *30*, 212-224.
Gottfredson, M. R., & Hirschi, T. (1990). *A general theory of crime*. Stanford, CA: Stanford University Press.
Greenberg, J., Arndt, J., Schimel, J., Pyszczynski, T., & Solomon, S. (2001). Clarifying the function of mortality salience-induced worldview defense: Renewed suppression or reduced accessibility of death-related thoughts? *Journal of Experimental Social Psychology*, *37*, 70-76.
Greeno, C. G., & Wing, R. R. (1994). Stress-induced eating. *Psychological Bulletin*, *115*, 444-464.
Greenwood, J. (1935). Hypoglycemia as a cause of mental symptoms. *Pennsylvania Medical Journal*, *39*, 12.
Guyton, A. C. (1976). *Textbook of medical physiology*. Philadelphia: Saunders.
Haier, R. J., Schandler, S. L., MacLachlan, A., Soderling, E., Buchsbaum, M. S., & Cohen, M. (1999). Alcohol induced changes in regional cerebral glucose metabolic rate during divided attention. *Personality & Individual Differences*, *26*, 425-439.
Haier, R. J., Siegel, B., Tang, C., Abel, L., & Buchsbaum, M. S. (1992). Intelligence and changes in regional cerebral metabolic rate following learning. *Intelligence*, *16*, 415-426.
Hall, J. B., & Brown, D. A. (1979). Plasma glucose and lactic acid alterations in response to a stressful exam. *Biological Psychology*, *8*, 179-188.
Hancock, P. A., & Warm, J. S. (1989). A dynamic model of stress and sustained attention. *Human Factors*, *31*, 519-537.
Harakas, P., & Foulds, J. (2002). Acute effects of glucose tablets on craving, withdrawal symptoms, and sustained attention in 12-h abstinent tobacco smokers. *Psychopharmacology*, *161*, 271-277.
Henner, E. (1936). Hypoglykaemie, podminena adenomen obruvku Langerhansovych brisni slinivky. *Casop Lek Cesk*, *75*, 177.
Hepburn, D. A., Deary, I. J., MacLeod, K. M., & Frier, B. M. (1996). Adrenaline and psychometric mood factors: A controlled case study of two patients with bilateral adrenalectomy. *Personality and Individual Differences*, *20*, 451-455.
Hill, D., & Sargant, W. (1943). A case of matricide. *Lancet*, *1*, 526.
Hodgins, D. C., el Guebaly, N., & Armstrong, S. (1995). Prospective and retrospective reports of mood states before relapse to substance use. *Journal of Consulting and Clinical Psychology*, *63*, 400-407.
Horne, J. A. (1993). Human sleep, sleep loss and behavior: Implications for the prefrontal cortex and psychiatric disorder. *British Journal of Psychiatry*, *162*, 413-419.
Hull, J. G. (1981). A self-awareness model of the causes and effects of alcohol consumption. *Journal of Abnormal Psychology*, *90*, 586-600.
Jarrett, R. J., Baker, I. A., Keen, H., & Oakley, N. W. (1972). Diurnal variation in oral glucose tolerance: Blood sugar and plasma insulin levels morning, afternoon, and evening. *British Medical Journal*, *1*, 199-201.
Jarrett, R. J., Viberti, G. C., & Sayegh, H. A. (1978). Does "afternoon diabetes" predict diabetes? *British Medical Journal*, *1*, 548-549.
Jarvik, M. E., Olmstead, R. E., Schneider, N. G., Iwamoto-Schaap, P. N., & Madsen, D. C. (1998). Sweeteners and cigarette craving: Glucose, aspartame, sorbitol. *American Journal of Health Behavior*, *22*, 130-140.

Kahan, D., Polivy, J., & Herman, C. P. (2003). Conformity and dietary disinhibition: A test of the ego-strength model of self-regulation. *International Journal of Eating Disorders*, *32*, 165-171.

Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (2000). *Principles of neural science*. New York: McGraw Hill.

Kennedy, D. O., & Scholey, A. B. (2000). Glucose administration, heart rate and cognitive performance: Effects of increasing mental effort. *Psychopharmacology*, *149*, 63-71.

Kennedy, S. H., Evans, K. R., Kruger, S., Mayberg, H. S., Meyer, J. H., McCann, S., et al. (2001). Changes in regional blood flow metabolism measured with positron emission tomography after paroxetine treatment of major depression. *American Journal of Psychiatry*, *158*, 899-905.

Kety, S. S. (1950). Circulation and metabolism of the human brain in health and disease. *American Journal of Medicine*, *8*, 205-217.

Keul, J., Huber, G., Lehman, M., Berg, A., & Jakob, E. F. (1982) Einfluss von Dex-trose auf Fahrleistung, Konzentrationsfaehigkeit, Kreislauf und Stoff-wechsel in Kraftfahrzeug-simulator (Doppelblindstudie im Cross-over Design) [Influence of dextrose on driving performance, concentration ability, and cycle and metabolism in a motor vehicle simulator (double-blind, crossover design)]. *Akt Ernahr Mad*, *7*, 7-14.

Kissin, B., & Gross, M. M. (1968). Drug therapy in alcoholism. *American Journal of Psychiatry*, *125*, 31-41.

Kokavec, A., & Crowe, S. F. (2003). Effect on plasma insulin and plasma glucose of consuming white wine alone after a meal. *Alcoholism: Clinical and Experimental Research*, *27,* 1718-1723.

Korol, D. L., & Gold, P. E. (1998) Glucose, memory, and aging. *American Journal of Clinical Nutrition*, *67*, 764-771.

Lane, J. D., Stabler, B., Ross, S. L., Morris, M. A., Litton, J. C., & Surwitt, R. S. (1988). Psychological predictors of glucose control in patients with IDDM. *Diabetes Care*, *11*, 798-800.

Larsen, R. J. (2000). Toward a science of mood regulation. *Psychological Inquiry*, *11*, 129-141.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Lee, P. S., & Bernicky, G. R. (1999). Effects of blood glucose levels on performance in activities of daily living: A case example of a diabetic man with an acquired brain injury. *Brain Injury*, *13*, 381-385.

Linnoila, V. M., & Virkkunen, M. (1992). Aggression, suicidality, and serotonin. *Journal of Clinical Psychiatry*, *53*, 46-51.

Linnoila, V. M., Virkkunen, M., George, T., & Higley, D. (1993). Impulse control disorders. *International Clinical Psychopharmacology*, *8*, 53-56.

Lund-Anderson, H. (1979). Transport of glucose from blood to brain. *Physiological Review*, *59*, 305-352.

Lustman, P. J., Frank, B. L., & McGill, J. B. (1991). Relationship of personality characteristics to glucose regulation in adults with diabetes. *Psychosomatic Medicine*, *53*, 305-312.

Lustman, P. J., Griffith, L. S., Clouse, R. E., & Cryer, P. E. (1986). Psychiatric illness in diabetes mellitus: Relationship to symptoms and glucose control. *Journal of Nervous and Mental Disease*, *174*, 736-742.

Manning, C. A., Hall, J. L., & Gold, P. E. (1990). Glucose effects on memory and other neuropsychological tests in elderly humans. *Psychological Science*, *1*, 307-311.

Marlatt, G. A., & Gordon, J. R. (1985). *Relapse prevention*. New York: Guilford.

Matykiewicz, L., La Grange, L., Reyes, E., Vance, P., & Wang, M. (1997). Adolescent males, impulsive/aggressive behavior, and alcohol abuse: Biological correlates. *Journal of Child and Adolescent Substance Abuse*, *6*, 27-37.

Matykiewicz, L., La Grange, L., Vance, P., Mu, M., & Reyes, E. (1997). Adjudicated adolescent males: Measures of urinary 5-hydroxyindoleacetic acid and reactive hypoglycemia. *Personality and Individual Differences*, *22*, 327-332.

Mayberg, H. S., Brannan, S. K., Tekell, J. L., Silva, J. A., Mahurin, R. K., McGinnis, S., et al. (2000). Regional metabolic effects of fluoxetine in major depression: Serial changes and relationship to clinical response. *Biological Psychiatry*, *48*, 1830-1843.

McCrimmon, R. J., Frier, B. M., & Deary, I. J. (1999). Appraisal of mood and personality during hypoglycemia in human subjects. *Physiology & Behavior*, *67*, 27-33.

McNay, E. C., Fries, T. M., & Gold, P. E. (2000). Decreases in rat extracellular hippocampal glucose concentration associated with cognitive demand during a spatial task. *Proceedings of the National Academy of Sciences*, *97*, 2881-2885.

McNay, E. C., McCarty, R. C., & Gold, P. E. (2001). Fluctuations in brain glucose concentration during behavioral testing: Dissociations between brain areas and between brain and blood. *Neurobiology of Learning and Memory*, *75*, 325-327.

McRobbie, H., & Hajek, P. (2004). Effect of glucose on tobacco withdrawal symptoms in recent quitters using bupropion or nicotine replacement. *Human Psychopharmacology: Clinical and Experimental*, *19*, 57-61.

Meijer, A. (1984). Psychiatric problems of children with glucose-6-phosphate dehydrogenase deficiency. *International Journal of Psychiatry in Medicine*, *14*, 207-214.

Mischel, W., Shoda, Y., & Peake, P. K. (1988). The nature of adolescent competencies predicted by preschool delay of gratification. *Journal of Personality and Social Psychology*, *54*, 687-696.

Moser, L., Plum, H., & Buckmann, M. (1983) Der Einfluß von dextrose auf die psychophysische leistungsfähigkeit des autofahrers [The influence of dextrose on the psychological efficiency of the driver]. *Aktuelle Ernaehrungsmedizin*, *8*, 247-249.

Moyer, K. E. (1976). *The psychobiology of aggression*. New York: Harper & Row.

Mueller, P. S., Heninger, G. R., McDonal, R. K. (1968). Intravenous glucose tolerance test in depression. *Archives of General Psychiatry*, *21*, 470-477.

Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin*, *126*, 247-259.

Muraven, M., Collins, R. L., & Nienhaus, K. (2002). Self-control and alcohol restraint: An initial application of the Self-Control Strength Model. *Psychology of Addictive Behaviors*, *16*, 113-120.

Muraven, M., Collins, R. L., Shiffman, S., & Paty, J. A. (2005). Daily fluctuations in self-control demands and alcohol intake. *Psychology of Addictive Behaviors*, *19*, 140-147.

Muraven, M., Shmueli, D., & Burkley, E. (2006). Conserving self-control strength. *Journal of Personality and Social Psychology*, *3*, 524-537.

Muraven, M., & Slessareva, E. (2003). Mechanisms of self-control failure: Motivation and limited resources. *Personality and Social Psychology Bulletin*, *29*, 894-906.

Muraven, M., Tice, D. M., & Baumeister, R. F. (1998). Self-control as a limited resource: Regulatory depletion patterns. *Journal of Personality and Social Psychology*, *74*, 774-789.

Murphy, J. M., Pagano, M. E., Patton, K., Hall, S., Marinaccio, J., & Kleinman, R. (2000). *The Boston Public Schools universal breakfast program: Final evaluation report*. Boston: Massachusetts General Hospital.

Murphy, J. M., Wehler, C. A., Pagano, M. E., Little, M., Kleinman, R. F., & Jellinek, M. S. (1998). Relationship between hunger and psychosocial functioning in low-income American children. *Journal of the American Academy of Child & Adolescent Psychiatry*, *37*, 163-170.

Nagornev, S. N., Bobrovnitsky, I. P., Ryguin, N. N., Orlova, T. A., Dlusskaya, I. G., Petrova, T. V., et al. (1999). Informational assets of the glucose tolerance test in evaluation of endurance of psychoemotional stresses. *Aviakosmicheskaya i Ecologicheskaya Meditsina*, *33*, 22-26.

New studies show strong link between diet and behavior. (2004). Retrieved October 17, 2004, from http://www.kidscanlearn.net/artcri.htm

Owens, D. S., & Benton, D. (1994). The impact of raising blood glucose on reaction times. *Neuropsychobiology*, *30*, 106-113.

Parker, P. Y. (1995). *The influence of blood glucose on the cognitive functioning of young adults*. Doctoral thesis, University of Wales, Swansea.

Parker, P. Y., & Benton, D. (1995). Blood glucose levels selectively influences memory for word lists dichotically presently to the right ear. *Neuropsychologia*, *33*, 843-854.

Peyrot, M. F., & McMurry, J. F. (1992). Stress buffering and glycemic control. *Diabetes Care*, *15*, 842-846.

Plant, E. A., & Devine, P. G. (1998). Internal and external motivation to respond without prejudice. *Journal of Personality and Social Psychology*, *75*, 811-832.

Pohl, S. L., Gonder-Frederick, L., Cox, D. J. (1984). Diabetes mellitus: An overview. *Behavioral Medicine Update*, *6*, 3-7.

Popkin, M. K., Callies, A. L., Lentz, R. D., Colon, E. A., & Sutherland, D. E. (1988). Prevalence of major depression, simple phobia, and other psychiatric disorders in patients with long standing type I diabetes mellitus. *Archives of General Psychiatry*, *45*, 64-68.

Powell, E. (1936). The role of the diet in the etiology and treatment of mental disorders resulting from hyperinsulinism. *Tri-State Medical Journal*, *8*, 1612.

Pramming, S., Thorsteinsson, B., Theilgaard, A., Pinner, E. M., & Binder, C. (1986). Cognitive function during hypoglycaemia in type I diabetes mellitus. *British Medical Journal*, *292*, 647-650.

Pratt, T. C., & Cullen, F. T. (2000). The empirical status of Gottfredson and Hirschi's general theory of crime: A meta-analysis. *Criminology*, *38*, 931-964.

Rehm, J., Room, R., Graham, K., Monteiro, M., Gmel, G. & Sempos, C. T. (2003). The relationship of average volume of alcohol consumption and patterns of drinking to burden of disease-an overview. *Addiction*, *98*, 1209-1228.

Reid, M., & Hammersley, R. (1995) Effects of carbohydrate intake on subsequent food intake and mood state. *Physiology and Behavior*, *58*, 421-427.

Reivich, M., & Alavi, A. (1983) Positron emission tomographic studies of local cerebral glucose metabolism in humans in physiological and pathological conditions. *Advances in Metabolic Disorders*, *10*, 135–176.

Richeson, J. A., & Shelton, J. N. (2003). When prejudice does not pay: Effects of interracial contact on executive function. *Psychological Science*, *14*, 287-290.

Richeson, J. A., & Trawalter, S. (2005). Why do interracial interactions impair executive function? A resource depletion account. *Journal of Personality and Social Psychology*, *88*, 934-947.

Richeson, J. A., Trawalter, S., & Shelton, J. N. (2005). African Americans' implicit racial attitudes and the depletion of executive function after interracial interactions. *Social Cognition*, *23*, 336-352.

Rojas, N., & Ferrer, S. A. (1941). Hypoglycemia in delinquents. *Archivos de Medicina Legal*, *11*, 29-35.

Rojas, N., & Sanchi, A. F. (1941). Hipoglucemia en delincuentes [Hypoglycemia in delinquents]. *Archivos Medicales Legal Identificacion*, *11*, 29.

Rovet, J. F., & Ehrlich, R.M. (1988). Effects of temperament on metabolic control in children with diabetes mellitus. *Diabetes Care*, *11*, 77-82.

Roy, A., Virkkunen, M., Guthrie, S., & Linnoila, M. (1986). Indices of serotonin and glucose metabolism in violent offenders, arsonists and alcoholics. In J. J. Mann & M. Stanley (Eds.), *Psychobiology of suicidal behavior* (pp. 202-220). New York: New York Academy of Sciences.

Saremi, A., Hanson, R. L., Tulloch-Reid, M., Williams, D. E., & Knowler, W. C. (2004). Alcohol consumption predicts hypertension but not diabetes. *Journal of Studies on Alcohol*, *65*, 184-190.

Schmeichel, B. J., Demaree, H. A., Robinson, J. L., & Pu, J. (in press). Ego depletion by response exaggeration. *Journal of Experimental Social Psychology*.

Schmeichel, B. J., Vohs, K. D., & Baumeister, R. F. (2003). Intellectual performance and ego depletion: Role of the self in logical reasoning and other information processing. *Journal of Personality and Social Psychology*, *85*, 33-46.

Schneiderman, N., & Skyler, J. S. (1995). Insulin metabolism, sympathetic nervous system regulation, and coronary heart disease prevention. In K. Orth-Gomer, & N. Schneiderman (Eds.), *Behavioral medicine approaches to cardiovascular disease prevention*. Hillsdale, NJ: Lawrence Erlbaum.

Scholey, A. B., Harper, S., & Kennedy. D. O. (2001). Cognitive demand and blood glucose. *Physiology & Behavior*, *73*, 585-592.

Scholey, A. B., & Kennedy, D. O. (2004). Cognitive and physiological effects of an "energy drink": An evaluation of the whole drink and of glucose, caffeine and herbal flavouring fractions. *Psychopharmacology*, *17*, 320-330.

Shakur, S. (1993). *Monster: The autobiography of an L.A. gang member*. New York: Atlantic Monthly Press.

Shiffman, S. M. (1982). Relapse following smoking cessation: A situational analysis. *Journal of Consulting and Clinical Psychology*, *50*, 71-86.

Shiffrin, R. M., & Schneider, W. (1977). Controlled and automatic human information processing: I. Detection, search, and attention. *Psychological Review*, *84*, 1-66.

Shoda, Y., Mischel, W., & Peake, P. K. (1990). Predicting adolescent cognitive and self-regulatory competencies from preschool delay of gratification: Identifying diagnostic conditions. *Developmental Psychology*, *26*, 978-986.

Siesjo, B. K. (1978). *Brain energy metabolism*. Chichester, UK: Wiley.

Simpson, G. C., Cox, T., & Rothschild, D. R. (1974). The effects of noise stress on blood glucose level and skilled performance. *Ergonomics*, *17*, 481-487.

Smid, H., Trumper, B., Pottag, G., Wagner, K., Lodmann, R., Scheich, H., et al. (1997). Differentiation of hypoglycaemia induced cognitive impairments: An electrophysiological approach. *Brain; a Journal of Neurology*, *120*, 1041-1056.

Smith, A. P., Clarka, R., & Gallaghera, J. (1999). Breakfast cereal and caffeinated coffee effects on working memory, attention, mood, and cardiovascular function. *Physiology & Behavior*, *67*, 9-17.

Stucke, T. S., & Baumeister, R. F. (2006). Ego depletion and aggressive behavior: Is the inhibition of aggression a limited resource? *European Journal of Social Psychology*, *36*, 1-13.

Sünram-Lea, S. I., Foster, J. K., Durlach, P., & Perez, C. (2002). Investigation into the significance of task difficulty and divided allocation of resources on the glucose memory facilitation effect. *Psychopharmacology*, *160*, 387-397.

Svanborg, P., Mattila-Evenden, M., Gustavsson, P. J., Uvnäs-Moberg, K., & Åsberg, M. (2000). Associations between plasma glucose and DSM-III-R cluster B personality traits in psychiatric outpatients. *Neuropsychobiology*, *41*, 79-87.

Swift, C. R., Seidman, F., & Stein, H. (1967). Adjustment problems in juvenile diabetes. *Psychosomatic Medicine*, *29*, 555-571.

Tager, B. N., & Shelton, E. K. (1943). Personality changes in endocrine disorders; with a note on 'symptomatic hypoglycemia.' *Journal of Clinical Endocrinology*, *3*, 239-242.

Tangney, J. P., Baumeister, R. F., & Boone, A. L. (2004). High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. *Journal of Personality*, *72*, 271-322.

Taylor, L. A., & Rachman. S. J. (1988). The effects of blood sugar level changes on cognitive function, affective state, and somatic symptoms. *Journal of Behavioral Medicine*, *11*, 279-291.

Tomarken, A. J., & Keener, A. D. (1998). Frontal brain asymmetry and depression: A self-regulatory perspective. *Cognition and Emotion*, *12*, 387-420.

Van Cauter, E., Polonsky, K. S., & Scheen, A. J. (1997). Roles of circadian and rhythmicity and sleep in human glucose regulation. *Endocrine Reviews*, *18*, 716-738.

Van den Berg, C. J. (1985). On the relation between energy transformations in the brain and mental activities. In G. R. J. Hockey, A. W. K. Gaillard, & M. H. G. Coles (Eds.), *Energetics and human information processing* (pp. 131-138). Dordrecht, Netherlands: Martinus Nijhoff.

van Pragg, H. M., & Leijnse, B. (1965). Depression, glucose tolerance, peripheral glucose uptake and their alterations under the influence of anti-depressive drugs of the hydrazine type. *Psychopharmacologica*, *8*, 67-78.

Virkkunen, M. (1982). Reactive hypoglycemic tendency among habitually violent offenders. *Neuropsychobiology*, *8*, 35-50.

Virkkunen, M. (1983). Insulin secretion during the glucose tolerance test in antisocial personality. *British Journal of Psychiatry*, *142*, 598-604.

Virkkunen, M. (1984). Reactive hypoglycemic tendency among arsonists. *Acta Psychiatrica Scandinavica*, *69*, 445-452.

Virkkunen, M. (1986). Insulin secretion during the glucose tolerance test among habitually violent and impulsive offenders. *Aggressive Behavior*, *12*, 303-310.

Virkkunen, M., DeJong, J., Bartko, J., Goodwin, F. K., & Linnoila, M. (1989). Relationship of psychobiological variables to recidivism in violent offenders and impulsive fire setters: A follow-up study. *Archives of General Psychiatry*, *46*, 600-603.

Virkkunen, M., & Huttunen, M. O. (1982). Evidence for abnormal glucose tolerance test among violent offenders. *Neuropsychobiology*, *8*, 30-34.

Virkkunen, M., Nuutila, A., Goodwin, F. K., & Linnoila, M. (1987). Cerebrospinal fluid monoamine metabolites in male arsonists. *Archives of General Psychiatry*, *44*, 241-247.

Vitaliano, P. P., Scanlan, J. M., Krenz, C., & Fujimoto, W. (1996). Insulin and glucose: Relationships with hassles, anger, and hostility in non-diabetic older adults. *Psychosomatic Medicine*, *58*, 489-499.

Vitaliano, P. P., Scanlan, J. M., Krenz, C., Schwartz, R. S., & Marcovina, S. M. (1996). Psychological distress, caregiving, and metabolic variables. *Journal of Gerontology: Psychological Sciences*, *51*, 290-297.

Vohs, K. D., Baumeister, R. F., & Ciarocco, N. J. (2005). Self-regulation and self-presentation: Regulatory resource depletion impairs impression management and effortful self-presentation depletes regulatory resources. *Journal of Personality and Social Psychology*, *88*, 632-657.

Vohs, K. D., Baumeister, R. F., Twenge, J. M., Schmeichel, B. J., Tice, D. M, & Crocker, J. (2006). *Decision fatigue: Making multiple decisions depletes the self*. Manuscript submitted for publication.

Vohs, K. D., & Faber, R. J. (2004). To buy or not to buy? Self-control and self-regulatory failure in purchase behavior. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 509-524). New York: Guilford.

Vohs, K. D., & Heatherton, T. F. (2000). Self-regulatory failure: A resource-depletion approach. *Psychological Science*, *11*, 249-254.

Volkow, N. D., Hitzemann, R., Wolf, A. P., Logan, J., Fowler, J. S., Christman, D., et al. (1990). Acute effects of ethanol on regional brain glucose metabolism and transport. *Psychiatry Research*, *35*, 39–48.

Wadden, T. A., & Letizia, K. A. (1992). Predictors of attrition and weight loss in patients treated by moderate and caloric restriction. In T. A. Wadden & T. B. VanItallie (Eds.), *Treatment of the seriously obese patient* (pp. 383–410). New York: Guilford.

Wang, G.-J., Volkow, N. D., Franceschi, D., Fowler, J. S., Thanos, P. K., Panayotis, K., et al. (2000). Regional brain metabolism during alcohol intoxication: Alcohol effects on the fetus, brain, liver, and other organ systems. *Alcoholism: Clinical & Experimental Research*, *24*, 822-829.

Wannamethee, S. G., Shaper, A. G., Perry, I. J., & Alberti, K. G. M. M. (2002). Alcohol consumption and incidence of type II diabetes. *Journal of Epidemiology & Community Health*, *56*, 542-548.

Wegner, D. M., & Pennebaker, J. W. (1993). Changing our minds: An introduction to mental control. In D. M. Wegner & J. W. Pennebaker (Eds.), *Handbook of mental control* (pp. 1–12). Englewood Cliffs, NJ: Prentice Hall.

Weiss, V. (1986). From memory span to the quantum mechanics of intelligence. *Personality and Individual Differences*, *7*, 737–749.

Wells, K. B., Golding, J. M., & Burnam, M. A. (1989). Affective substance use, and anxiety disorders in persons with arthritis, diabetes, heart disease, high blood pressure, or chronic lung conditions. *General Hospital Psychiatry*, *11*, 320-327.

Wesnes, K. A., Pincock, C., Richardson, D., Helm, O. & Hails, S. (2003). Breakfast reduces declines in attention and memory over the morning in schoolchildren. *Appetite*, *41*, 329-331.

West, R. (2001). Glucose for smoking cessation: Does it have a role? *CNS Drags*, *15*, 261-265.

West, R., & Willis, N. (1998) Double-blind placebo controlled trial of dextrose tablets and nicotine patch in smoking cessation. *Psychopharmacology*, *136*, 201-204.

West, R., Courts, S., Beharry, S., May, S., & Hajek, P. (1999). Acute effect of glucose tablets on desire to smoke. *Psychopharmacology*, *147*, 319-321.

Wevers, M. E. (1988). The role of postcessation factors in tobacco abstinence: Stressful events and coping responses. *Addictive Behaviors*, *13*, 297-302.

Wilder, J. (1940). Problems of criminal psychology related to hypoglycemic states. *Journal of Criminology and Psychopathology*, *1*, 219.

Wilder, J. (1947). Sugar metabolism and its relation to criminology. In S. Linder & B. J. Selinger (Eds.), *Handbook of correctional psychology*. New York: Philosophical Library.

Wilson, D. R. (1951). Electroencephalographic studies in diabetes mellitus. *Canadian Medical Association Journal*, *65*, 462-465.

Wing, R. R., Blair, E. H., Epstein, L. H., & McDermott, M. D. (1990). Psychological stress and glucose metabolism in obese and normal-weight subjects: A possible mechanism for differences in stress-induced eating. *Health Psychology*, *9*, 693-700.

Wing, R. R., Epstein, L. H., Blair, E., & Nowalk, M. P. (1985). Psychologic stress and blood glucose levels in nondiabetic subjects. *Psychosomatic Medicine*, *47*, 558-564.

Wolfgang, M. E., & Ferracuti, F. (1967). *The subculture of violence: Towards an integrated theory in criminology*. London: Tavistock.

Wredling, R. A. M., Theorell, P. G. T., Roll, M. H., Lins, P. E. S., & Adamson, U. K. C. (1992). Psychosocial state of patients with IDDM prone to recurrent episodes of severe hypoglycemia. *Diabetes Care*, *75*, 518-521.

Wright, J. S. (1977). The psychology and personality of addicts. *Adolescence*, *12*, 399-403.

Wu, J., Buchsbaum, M. S., Gillin, J. C., Tang, C., Cadwell, S., Wiegand, M., et al. (1999). Prediction of antidepressant effects of sleep deprivation by metabolic rates in the ventral anterior cingulated and medial prefrontal cortex. *American Journal of Psychiatry*, *156*, 1149-1158.

Wu, J. C., Gillin, J. C., Buchsbaum, M. S., Hershey, T., Hazlett, E., Sicotte, N., et al. (1991). The effect of sleep deprivation on cerebral glucose metabolic rate in normal humans assessed with positron emission tomography. *Sleep: Journal of Sleep Research & Sleep Medicine*, *14*, 155-162.

Yaryura-Tobias, J. A., & Neziroglu, F. A. (1975). Violent behavior, brain dysrhythmia, and glucose dysfunction: A new syndrome. *Journal of Orthomolecular Psychiatry*, *4*, 182-188.

Zhao, Y., & Liu, F. (1999). Cognitive function induced by diabetes and relevant studies. *Chinese Mental Health Journal*, *13*, 314-316.

Zhu, W., Volkow, N. D., Ma, Y., Fowler, J. S., & Wang, G.-J. (2004). Relationships between ethanol-induced changes in brain regional metabolism and its motor, behavioural and cognitive effects. *Alcohol and Alcoholism*, *39,* 53-58.