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Response monitoring and cognitive control in childhood obesity

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ABSTRACT

The ability to discern when actions deviate from goals and adjust behavior accordingly is crucial for efforts at self-regulation, including managing one's weight. We examined whether children with obesity differed from controls in *response monitoring*, an aspect of cognitive control that involves registering one's errors. Participants performed a cognitive interference task, responding to the colors of arrows while ignoring their orientations, and error-related neural activity was indexed via response-locked event-related potentials (ERPs). Compared to controls, participants with obesity exhibited significantly blunted "error-related negativity", an ERP component linked to response monitoring. Participants with obesity also exhibited a marginally blunted "error-related positivity", an ERP component linked to late-stage error processing, as well as in behavioral indices of cognitive control. These results suggest that childhood obesity may be associated with reduced response monitoring and that this aspect of cognitive control may play an important role in health-related self-regulatory behavior.

1. Introduction

Obesity is a mounting health concern among adults and children alike. To combat obesity, some prevention initiatives have begun facilitating access to better food choices and opportunities for physical activity. However, access to healthy alternatives works only so far as individuals actively choose to pursue them. The sustained lifestyle changes that are often necessary for combating obesity in themselves require immense efforts at self-regulation. The "simple" act of dieting is associated with diminished performance on a range of central executive tasks (Kemps et al., 2005), suggesting that such self-regulation requires and can exhaust cognitive control. This link between obesity and cognitive control raises the possibility that the struggle to manage one's weight might be exacerbated by atypicalities in mechanisms that underlie cognitive control. The purpose of the present study was to compare a sample of children with obesity to age- and sex-matched controls in order to identify cognitive control mechanisms that might be compromised.

The very general term "cognitive control" encompasses a diverse range of mechanisms that combine to enable people to guide their own behaviors and allocate cognitive resources in the service of a goal. Such mechanisms include response inhibition, task switching, and error monitoring, among others. Previous research has suggested relationships between obesity and aspects of cognitive performance, independent of related health problems (Elias et al., 2003). For example, Gunstad et al. (2007) have shown that detriments in executive functioning are more prevalent in obese adults, and Cserjési et al. (2009) found that obesity was associated with poor response inhibition and attentional control. The relationship between obesity and cognition is evident in both children and adolescents, with higher body mass indices (BMIs) associated with poor attention and task-switching abilities (Cserjési et al., 2007). Adolescents with excess weight exhibit greater difficulty with response monitoring and switching (Verdejo-García et al., 2010). Li et al. (2008) found that BMI negatively correlated with cognitive functioning even after controlling for mediating factors such as TV viewing and parental education level. Recent brain imaging work also suggests that cognitive control may be compromised in obesity. Grey matter volume in the orbital frontal cortex, a brain region involved in response inhibition, is reduced in obese individuals (Maayan et al., 2011) and higher BMI predicts decreased baseline activation of areas of the prefrontal cortex including the anterior cingulate cortex (ACC; Volkow et al., 2008; Willeumier et al., 2011).

The current study focused on the relationship between obesity and aspects of cognitive control linked with the ACC, which plays a role in response monitoring. ACC activity is thought to

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signal when a response deviates from goal-oriented intentions, and it is typically heightened after people make performance errors (e.g. Barch et al., 2000; Botvinick et al., 1999; Carter et al., 1998; Van Veen and Carter, 2002). Such neural activity can be examined by measuring event-related brain potentials (ERPs) following the execution of errors. An early ERP, the error-related negativity (ERN), peaks between 50 and 100 ms following the mistake and is thought to reflect the initial detection of conflict (e.g. Bernstein et al., 1995; Falkenstein et al., 1991; Gehring et al., 1993; Simons, 2010). Thus, the ERN may be conceptualized as a neural "red flag" that serves to alert control-related prefrontal brain regions, leading to a subsequent increase in cognitive control (Kerns et al., 2004; Ridderinkhof et al., 2004). In addition to the ERN, an error-related ERP waveform known as the Pe, a positive deflection recorded over parietal cortex that occurs around 300 ms post-response, was also examined. In contrast to the ERN, which can be robust even when people do not know that they have committed an error, the Pe component is typically larger when people are aware of their errors (Falkenstein et al., 2000; Nieuwenhuis et al., 2001). The Pe is thought to reflect additional, later-stage error processing that may represent the subjective assessment of an error or the mobilization of cognitive resources leading to adjustments in behavioral strategy (Falkenstein et al., 2000; Overbeek et al., 2005).

No studies to date have examined the relationship between response monitoring, cognitive control, and childhood obesity. The current study examined the potential link among these variables using both behavioral and electrophysiological methods. Children and adolescents with obesity undergoing weight management treatment and a sample of age-matched, healthy-weight controls participated in a Simon-like cognitive interference task designed to elicit a substantial number of errors. Response-locked ERPs were recorded in order to examine error-related brain activity. Given previous findings linking obesity to poor cognitive control, we expected that weight management (WM) patients would exhibit diminished ERN and Pe amplitude when compared to healthy-weight (Control) children. Behavioral performance was also tracked to probe for overt indices of cognitive control.

2. Methods

2.1. Participants

28 obese children (22 female) and 32 control children (15 female) successfully completed the current study. All children were between 7 and 17 years of age with mean ages of 12.8 (±2.4) and 12.8 (±2.5) years for children in the obese and control groups respectively and the two groups did not differ significantly in age ($p = .92$). Children with obesity were recruited from a weight management clinic at Alfred I. DuPont Hospital for Children (an affiliate of Nemours Foundation) in Wilmington, Delaware. All weight management children were recruited during their first visit to the clinic and thus had not yet received any obesity treatment. Control children were also recruited from Alfred I. DuPont hospital and Nemours affiliated primary care clinics via flyers posted throughout the buildings.

All children in the control group had a body mass index (BMI) between the 5th and 85th percentile for their age and height, and were thus considered to be "healthy-weight." Children in the weight management group were determined to be "obese" by the weight management clinic, with BMIs greater than the 95th percentile for their height and weight. Participants were excluded from both groups for serious medical problems including cancer and genetic syndromes. Children with medical co-morbidities such as type II diabetes, hypertension, and sleep apnea or pre-existing cognitive dysfunctions, including autism and developmental delay, were also excluded from participating. We did not exclude for ADHD, but the number of children with ADHD was the same in each group: 3 participants in the weight management group and 3 children in the control group reported this diagnosis. All six of these participants had taken their prescribed medications before completing experimental procedures. Children in the control group were more likely to be Caucasian ($p < .05$) and tended to have families with somewhat higher socio-economic status ($p < .10$).

Parental consent and child assent were obtained either in the weight management clinic or upon arrival at the laboratory. Both parents and children were given compensation for participation. All study procedures were approved by the Institutional Review Boards of the University of Delaware and the Nemours (Alfred I. DuPont Hospital for Children) Office of Human Subjects Protection.

2.2. Arrow task

Each participant performed a variation of the classic Simon task (Simon, 1969). On each trial, participants made a speeded response based on the color of an arrow presented on a computer monitor while ignoring the direction in which the arrow was pointing. The task was composed of 576 trials arranged into 12 blocks of 48 trials each. Stimuli were arrows that could point left, right, or upwards and could be either red or green. Arrows were presented one at a time for 200 ms and were followed by a blank screen for an additional 800 ms. Participants could respond at any point during the entire 1000 ms interval. Each trial was followed by a 1000 ms inter-trial interval. Participants pressed the leftmost button on a response box on trials when the arrow was red and the rightmost button when the arrow was green. In "congruent" trials, the red arrow pointed left and the green arrow pointed right; in "incongruent" trials, the red arrow pointed right and the green arrow pointed left, resulting in conflict between the responses required by the arrow's color and direction. In "neutral" trials, the arrows pointed upwards and did not cause any directional interference. Trials were counterbalanced so that the same number of congruent, incongruent, and neutral stimuli appeared in each block. Participants completed two practice blocks of 50 trials each before data recording.

2.3. ERP recording, reduction and analysis

Brain activity was recorded using a 32-channel Waveguard electrode cap. EEG signals were sampled at 512 Hz using the ASA system (ANT; Advanced Neuro Technology, Enschede, The Netherlands), band-pass filtered (0.1–20 Hz), and referenced to electrodes placed at the mastoids. Impedances were kept below 10 kΩ. Waveforms were corrected for blinks, and signals that exceeded 75 mV were regarded as artifact and these trials were rejected. Response-locked ERPs were separately averaged for trials where participants responded correctly and those where participants committed an error. Trials where no response was recorded (i.e. omitted responses) were not included in the ERP analysis. ERPs were averaged for 900 ms following the response, with a 100 ms pre-response baseline.

Statistical analysis for ERN amplitude was performed at a medio-frontal scalp region of interest that included midline electrodes Cz and Fz and lateral electrodes FC1 and FC2. Pe amplitude was assessed at a centro-parietal region, including electrodes Cz, Pz, CP1 and CP2. Activation was measured by taking the mean amplitude for a time window between 20 and 80 ms post-response for the ERN and between 250 and 400 ms post-response for the Pe.

3. Results

3.1. ERP results

Data from 31 participants (16 WM, 15 controls) were entered into the ERP analysis. Six participants (4 weight management and 2 control) had error rates and/or rates of omitted responses greater than 2.5 standard deviations from the mean and were excluded from both behavioral and ERP analyses. Data from 3 additional control subjects were not used because their siblings also participated and served as better age-matches to the obese group. In addition, ERP data from 16 participants (6 WM, 10 controls) were excluded from the analysis due to excess movement artifact, and data from 4 additional participants were removed due to error rates that were too low for sufficient ERP averaging.

A 2 (trial type: error vs. correct) × 2 (group) ANOVA of ERN amplitude yielded a significant main effect for trial type, with a greater ERN on error trials ($M = -0.2042\ \mu V$, $SD = 0.2676\ \mu V$) than correct trials ($M = -0.0057\ \mu V$, $SD = 0.2118\ \mu V$) [$F(1,29) = 25.293$, $p < .001$]. The main effect for group was not significant, but a significant 2-way interaction confirmed that the difference in ERN amplitude for error and correct trials was larger among the control group than the WM group [$F(1,29) = 8.570$, $p = .007$; see Fig. 1]. A 2 (trial type: error vs. correct) × 2 (group) ANOVA revealed that the Pe was also significantly greater on error trials ($M = 1.0424\ \mu V$, $SD = 1.2840\ \mu V$) than correct trials ($M = -1.1802\ \mu V$, $SD = 0.8672\ \mu V$) [$F(1,29) = 90.541$, $p < .001$]. There was no significant main effect for group, but a marginally significant 2-way interaction emerged such that the difference in Pe amplitude for error and correct trials was larger among subjects in

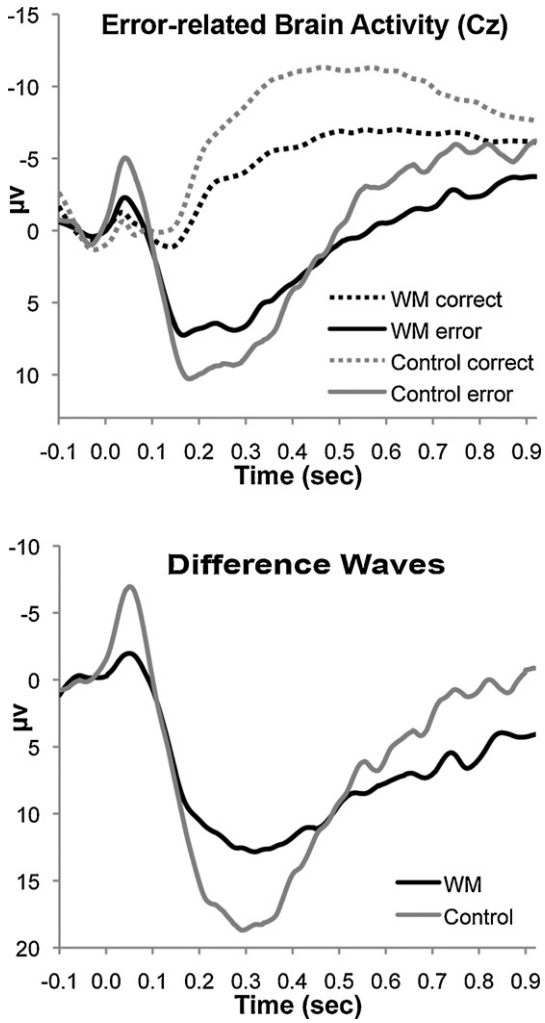

Fig. 1. (a) Error-related brain potentials recorded from electrode site Cz. Potentials are response-locked and the button press occurred at time 0. (b) Difference waves were calculated by subtracting brain response to correct trials from brain response to error trials.

the control group than that it was in subjects from the WM group [$F(1,29) = 3.695$, $p = .064$; see Fig. 1].[1]

Notably, there was a marginal group difference in error rates among this sample [WM: $M = 12.41\%$, $SD = 8.12\%$, Control: $M = 7.51\%$, $SD = 5.59\%$; $t(29) = -1.943$, $p = .062$], indicating that the WM group made more errors than controls. Because it is well established that ERN amplitude can vary substantially with error frequency (e.g., Gehring et al., 1993), we performed a 2 (trial type: error vs. correct) × 2 (group) ANCOVA on ERN amplitude using error rates as a covariate. The group × trial type interaction remained significant [$F(1,29) = 8.720$, $p = .006$] when removing the variance associated with error rates. Moreover, this interaction remained significant [$F(1,21) = 6.5$, $p = .019$] when comparing subsamples from both groups matched on number of errors (<15%; $N = 23$).

[1] Notably, when the six participants with ADHD (3 patients, 3 controls) were excluded from the analyses, both the main effect of trial type and the trial type × group interaction remained significant for the ERN ($p < .001$ and $p = .003$, respectively). For the identical analysis involving Pe, the main effect of trial type remained significant ($p < .001$) and the significance of the interaction with group became more robust ($p = .025$).

Table 1
Mean reaction times and standard deviations in milliseconds as a function of previous trial accuracy and current and previous trial congruence.

	WM	Control
Previous correct	462(100)	442(101)
Previous incorrect	496(115)	458(101)
Current congruent	448(99)	424(96)
Current neutral	467(106)	443(103)
Current incongruent	489(104)	465(103)
Previous congruent[a]	489(100)	465(100)
Previous neutral[a]	486(106)	474(100)
Previous incongruent[a]	481(111)	450(100)

[a] Reaction time average for current-incongruent trials only.

3.2. Behavioral results

Data from 51 participants (24 weight management and 27 controls) who successfully completed the task were entered into the behavioral analysis. These included the participants in the ERP analyses, as well as the 20 participants whose data were excluded from the ERP analysis due to artifact or low error rates.

Among this sample of participants, the weight management (WM) group had significantly higher error rates ($M = 12.37\%$, $SD = 7.14\%$) than the control group ($M = 7.89\%$, $SD = 6.32\%$) [$t(49) = 2.375$; $p = .022$]. Behavioral performance was further assessed using measures of response time (RT), with shorter RTs indicating better performance. In order to track dynamic adjustments in cognitive control, post-error effects were assessed by comparing performance following errors to performance following correct trials and congruency effects were assessed by analyzing performance as a function of both current-trial congruence and the congruence of the preceding trial.

Average RT following errors was compared to that following correct trials, and a 2 (previous trial response: error vs. correct) × 2 (group) repeated measures analysis of variance (ANOVA) revealed a main effect of previous trial response, with slower RTs following error trials ($M = 475.67$ ms, $SD = 108.72$ ms) than following correct trials ($M = 451.74$ ms, $SD = 100.07$ ms) [$F(1,49) = 15.04$, $p < .001$]. However, neither the main effect of group ($p = .32$) nor the 2-way interaction ($p = .15$) was significant, indicating that although both groups exhibited post-error slowing, they did not differ in their performance as a function of whether the preceding trial had been correct or incorrect. All RT data for the two groups separately are presented in Table 1.

Performance based on *current trial* congruence was measured as RT for correct responses and assessed using a 2 (congruence: incongruent vs. congruent) × 2 (group: WM vs. control) ANOVA, which revealed a main effect [$F(1,49) = 142.404$, $p < .001$] for congruence such that participants were slower to respond on incongruent trials ($M = 475.96$ ms, $SD = 103.22$ ms) than congruent trials ($M = 435.17$ ms, $SD = 97.22$ ms). There was no main effect ($p = .40$) or interaction with group ($p = .99$). To assess whether the congruency effect stemmed from interference on incongruent trials or facilitation on congruent trials, neutral trials were incorporated as a baseline, and two 2 (congruence: incongruent vs. neutral, congruent vs. neutral) × 2 (group: WM vs. control) ANOVAs were calculated to compare RTs on congruent and incongruent trials, respectively, to performance on neutral trials. RTs on congruent trials were significantly quicker than RTs on neutral trials ($M = 454.07$ ms, $SD = 104.22$ ms) [$F(1,49) = 56.257$, $p < .001$] and RTs on incongruent trials were significantly slower than RTs on neutral trials [$F(1,49) = 66.603$, $p < .001$]. However, RTs did not differ between groups for either comparison ($p = .40$, $p = .42$) and neither of the 2-way interactions was significant ($p = .93$, $p = .95$).

Performance based on *previous trial* congruence was also analyzed. The analysis was restricted to RTs on correct incongruent trials that followed other correct trials in order to isolate the effect, which is most robust on difficult (i.e., incongruent) trials. A 2 (previous trial congruence: congruent vs. incongruent) × 2 (group: WM vs. control) ANOVA yielded a significant main effect [$F(1,49) = 4.864$, $p = .032$] for congruence such that participants were quicker to respond following incongruent trials ($M = 464.86$ ms, $SD = 105.33$ ms) than congruent trials ($M = 476.54$ ms, $SD = 100.02$ ms). However, neither the main effect for group ($p = .35$) nor the 2-way interaction ($p = .49$) was significant. Additional 2-way ANOVAs that incorporated neutral trials as a baseline were conducted to assess whether the effect of previous trial congruence stemmed from a *speeding* of responses following incongruent trials or a *slowing* of responses following congruent trials, the latter of which would indicate a slackening of cognitive control. There were no significant main effects (trial $p = .51$; group $p = .55$) or significant interaction ($p = .17$) when comparing previous congruent trials to previous neutral trials in the 2 (previous trial congruence: congruent vs. neutral) × 2 (group) ANOVA. However, in the complementary 2 (previous trial congruence: incongruent vs. neutral) × 2 (group) ANOVA, RTs following incongruent trials were significantly faster than RTs following neutral trials ($M = 479.69$ ms, $SD = 108.96$ ms) [$F(1,49) = 8.92$, $p = .004$], and the 2-way interaction was marginally significant [$F(1,49) = 3.929$, $p = .053$], indicating that the control group increased the speed of their responses following incongruent trials more than did WM participants.

4. Discussion

In the current study, the weight management group exhibited smaller ERN amplitude following errors relative to the healthy-weight group. Reduced ERN amplitude in this context suggests a possible reduction in the efficiency of participants' response monitoring, which could hinder the executive regulation of cognitive control. The WM group also exhibited a smaller Pe component than the healthy-weight group, possibly indicating diminished awareness, motivation, or altering of response strategy. Although this effect was marginal, it provides converging evidence suggestive of inefficient error monitoring.

There are several theories to describe the functional significance of the ERN. Some accounts suggest that the ERN represents an error-detecting comparator process where the conception of the correct response and the actual (incorrect) response are pitted against one another (Coles et al., 2001; Falkenstein et al., 1991; Gehring et al., 1993), while other accounts suggest that the ERN is produced when two conflicting responses are activated, regardless of whether the brain discriminates which is "right" or "wrong" (Botvinick et al., 2001; Carter et al., 1998). The ERN may also have an affective component, as the strength of the signal differs with changes in mood (Larson et al., 2006) and motivational value of the task (Hajcak et al., 2005).

A "reinforcement-learning" hypothesis of the ERN may provide a neural mechanism linking obesity with reduced ERN. According to this account, the ERN involves the input of the midbrain dopaminergic "reward" circuitry, which indicates when outcomes are better or, in the case of errors, worse than expected (Holroyd and Coles, 2002), and this account is supported by the fact that dopamine appears to play a role in the strength of the ERN signal. The administration of haloperidol, a dopamine antagonist, results in reduced ERN amplitude (Zirnheld et al., 2004) while the dopamine agonist D-amphetamine increases ERN amplitude (de Bruijn et al., 2004). Importantly, obesity is linked to reduced striatal dopaminergic receptors (Wang et al., 2001) and variations in dopamine receptor genes that produce hyposensitivity (Noble, 2000). The current study did not test dopamine levels, but it is possible that this relationship could explain the blunted ERN signal observed among the weight management participants and provide a promising avenue for further research.

Although debate exists regarding the functional significance of the ERN, it is widely believed that the signal is produced at least partly by the ACC, a brain region involved in the executive regulation of cognitive control (Bernstein et al., 1995; Falkenstein et al., 1991; Gehring et al., 1993). Diminished cognitive, or inhibitory, control is associated with impulsivity and there have been a number of studies that have established relationships between reduced ERN amplitude and impulsive characteristics such as quickened response times (Pailing et al., 2003; Ruchsow et al., 2005), reduced punishment sensitivity (Boksem et al., 2006; Potts et al., 2006), externalizing (Hall et al., 2007), and risk-taking propensity (Santesso and Segalowitz, 2009). Blunted ERN components are also observed among children with ADHD (Albrecht et al., 2008; Liotti et al., 2005) and individuals with substance abuse disorders (Franken et al., 2007). Likewise, obesity has been linked with temperament and personality characteristics associated with impulsive behavior (Fassino et al., 2002). For example, children with obesity responded more impulsively on a cognitive task (Braet et al., 2007) and higher BMIs were associated with more impulsive responding and less activation in brain regions involved in impulse control (Batterink et al., 2010). Considering such findings, it is possible that the attenuated ERN associated with the obese group could correspond to a heightened susceptibility to impulse control problems.

Efforts to understand the relationship between cognitive control and obesity can be facilitated by noting that "cognitive control" itself is not a unitary construct and has different aspects. For example, it has been proposed that individuals engage both *proactive* and *reactive* cognitive control strategies to carry out goal-oriented behavior (Braver et al., 2007). To illustrate, if a smoker who is trying to quit knows in advance that she will be at a party with many smokers, she can summon the means to control her impulses ahead of time–this is what is referred to as *proactive* cognitive control. In contrast, if she unexpectedly encounters a roomful of smokers, only then can she attempt to apply the effort to overcome an impulse to smoke, thereby relying on *reactive* control.

The present study contained a potential means for isolating the contributions of reactive and proactive forms of control, which involves examining task performance as a function of both the congruency of the current trial (reactive control) and the congruency of the preceding trial ($n - 1$; proactive control; Botvinick et al., 2001). Participants are typically slower to respond to incongruent vs. congruent trials (with RTs for neutral trials falling in between), but quicker to respond on trials *following* incongruent vs. congruent trials, an effect known as *conflict adaptation* (Botvinick et al., 2001, 2004; Gratton et al., 1992; Kerns et al., 2004). In the current study, RT trends did not differ between obese and healthy weight children when examining performance based on current trial congruence (reactive control). However, marginal differences emerged when examining performance based on preceding trial congruence. The healthy weight children exhibited speeded responses following incongruent trials (relative to following neutral trials), but the obese group did not. Although this effect fell slightly short of significance and the experiment design did not equate the number of previous-congruent and previous-incongruent trials, it highlights the possibility that the self-regulatory deficits observed among obese individuals may stem from differences in the ability to apply executive control proactively. Future research into this relationship may help elucidate the role that proactive cognitive control plays in weight regulation.

The current findings link obesity with decreased ERN amplitude relative to that in a healthy-weight group. Smaller ERNs could index

reduced response monitoring, which may have deleterious consequences for subsequent recruitment of cognitive control. However, it is important to note that there are limitations in the current study. For example, there is no way to infer causation from the present results. It is, as of yet, unclear whether obesity is detrimental to cognitive functioning or whether pre-existing response monitoring differences themselves contribute to behaviors that lead to obesity. Furthermore, the current sample was somewhat idiosyncratic. As noted above, three of the subjects in the weight management group were also taking medication for ADHD. Because obesity and ADHD are highly co-morbid (Cortese and Peñalver, 2010), we chose not to eliminate subjects with ADHD; rather, we chose to include three subjects in the control group who were also medicated for ADHD. We also did not control for factors such as IQ, psychological co-morbidities, socio-economic status, and parental education level, which themselves have an impact on cognition. Further research, in which such factors are better balanced, is needed to verify and establish the role of response monitoring and cognitive control in obesity.

Obesity is a complex problem that encompasses more than simple metabolic disadvantage. Understanding how obesity is tied to cognitive inefficiencies deemphasizes the popular view that obesity is the "fault" of the individual and has a simple solution. Rather, it may be that current intervention programs achieve limited success because they do not target related underlying cognitive processes. Although cognitive behavioral therapy is routinely used in obesity treatment (Barlow, 2007), current interventions may not adequately isolate specific cognitive mechanisms associated with obesity. Through greater precision in our understanding of such cognitive mechanisms, it may be possible to design more efficient cognitive control interventions to supplement nutritional counseling. The present findings may constitute a brick of the path toward that goal.

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