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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