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The association of childhood obesity to neuroelectric indices of inhibition

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Abstract

To examine whether childhood obesity is associated with inhibitory control, we compared healthy weight and obese preadolescent children's task performance along with the N2 and P3 components during a Go/NoGo task. Results indicated that obese children exhibited lower response accuracy relative to healthy weight children during the NoGo task requiring greater amounts of inhibitory control, whereas no such difference was observed during the Go task. Neuroelectric data indicated that healthy weight children exhibited a more frontal distribution for the NoGo P3 relative to the Go P3, whereas obese children had similar topographic distributions between the Go P3 and NoGo P3. Further, obese children had larger NoGo N2 amplitude relative to the Go N2, whereas this difference was not observed for healthy weight children. These findings suggest that childhood obesity is negatively and selectively associated with prefrontal inhibitory control.

Descriptors: Childhood obesity, Inhibitory control, N2, P3, Go/NoGo

The epidemic of childhood obesity has become a worldwide public health concern (Ebbeling, Pawlak, & Ludwig, 2002; Swinburn et al., 2011), and approximately one fifth of preadolescent children now are considered obese in the United States (Ogden, Carroll, Kit, & Flegal, 2012). It has been well established that obesity is a major contributor to chronic diseases such as type 2 diabetes and cardiovascular disease not only for adults but also for children (Ebbeling et al., 2002). Recent studies have indicated that weight status is also negatively associated with academic achievement (Datar & Sturm, 2006; Donnelly et al., 2009; Hollar, Lombardo et al., 2010; Hollar, Messiah et al., 2010) and cognitive function in children (Kamijo et al., in press; Li, Dai, Jackson, & Zhang, 2008; Lokken, Boeka, Austin, Gunstad, & Harmon, 2009), suggesting that maintaining a healthy weight may be essential for healthy brain maturation and cognitive development during childhood.

Childhood Obesity and Inhibitory Control

Several longitudinal studies have indicated that school-based obesity prevention interventions including nutrition and physical activity programs can enhance appropriate weight gain during growth and improve academic achievement (Donnelly et al., 2009; Hollar, Lombardo et al., 2010; Hollar, Messiah et al., 2010). Further, recent cross-sectional studies have shown that weight status is negatively associated with cognitive performance during tasks requiring the upregulation of cognitive control (Kamijo et al., in press; Li et al., 2008; Lokken et al., 2009). Cognitive control describes a subset of goal-directed, self-regulatory operations that encompass the selection, scheduling, and coordination of computational processes underlying perception, memory, and action (Meyer & Kieras, 1997; Norman & Shallice, 1986), with inhibition, cognitive flexibility, and working memory thought to comprise core processes underlying such abilities (Diamond, 2006). Given that these aspects of cognition have been implicated in academic achievement (Agostino, Johnson, & Pascual-Leone, 2010; St Clair-Thompson & Gathercole, 2006), the negative association between weight status and cognitive control (Kamijo et al., in press; Li et al., 2008; Lokken et al., 2009) is consistent with findings of longitudinal studies indicating that higher body mass index (BMI) is associated with lower academic achievement scores (Datar & Sturm, 2006; Donnelly et al., 2009; Hollar, Lombardo et al., 2010; Hollar, Messiah et al., 2010).

Interestingly, recent studies have observed that cognitive control ability can predict future weight status. Graziano, Calkins, and Keane (2010) found that poorer inhibitory control, which is one aspect of cognitive control, in 2-year-old children predicted obesity/overweight status in 5.5-year-old children. A magnetic resonance imaging study revealed that smaller gray matter volume in brain regions associated with inhibitory control, such as the

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middle and superior frontal gyri, can predict the following year's weight gain in female adolescents, with smaller volume related to increases in BMI (Yokum, Ng, & Stice, 2012). Although the direction of the association between weight status and cognitive control remains unknown (see Smith, Hay, Campbell, & Trollor, 2011, for a review), inhibitory control appears to play a critical role in the cognition–weight status relationship.

Accordingly, we examined the association between weight status and task performance during a Go/NoGo task, which has been demonstrated to manipulate inhibitory control demands, in preadolescent children (Kamijo et al., in press). The Go task requires participants to respond to rare, target stimuli (e.g., 0.2 probability) and withhold their response to frequent, nontarget stimuli (e.g., 0.8 probability), whereas the NoGo task requires participants to respond to frequent, nontarget stimuli and withhold their response to rare, target stimuli. Although both the Go and NoGo tasks require attentional allocation to detect target stimuli amid the train of nontarget stimuli, the NoGo task requires greater amounts of cognitive control to inhibit the prepotent response on rare stimulus trials. We have previously found that children with higher BMI and fat mass, measured via dual energy X-ray absorptiometry (DXA), exhibited poorer task performance (i.e., lower response accuracy) on the NoGo, but not on the Go task, suggesting that childhood obesity is selectively associated with inhibitory control (Kamijo et al., in press). Given that no overt response is required for target stimuli in the NoGo task, the addition of neuroelectric measures reflecting a subset of covert processes involved in inhibition may lead to a deeper understanding of the association between weight status and cognitive control.

NoGo Event-Related Potentials (ERPs)

During the Go/NoGo task, frontocentral N2 and P3 are larger for NoGo relative to Go stimuli (e.g., Bruin & Wijers, 2002; Jodo & Kayama, 1992; Pfefferbaum, Ford, Weller, & Kopell, 1985; Smith, Johnstone, & Barry, 2008), suggesting that both the NoGo N2 and NoGo P3 reflect a subset of inhibitory processes. However, recent evidence has indicated that the processes reflected by the NoGo N2 and NoGo P3 may also be functionally dissociable.

NoGo N2. The NoGo N2 is a negative-going component of an ERP occurring approximately 200 to 400 ms after stimulus onset, with a topographic maximum over frontocentral recording sites. Although earlier studies suggested that the NoGo N2 reflects inhibitory neural processes (Falkenstein, Hoormann, & Hohnsbein, 1999; Jodo & Kayama, 1992), recent studies have proposed that the NoGo N2 is associated with processes underlying conflict monitoring (Donkers & van Boxtel, 2004; Jonkman, 2006; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Randall & Smith, 2011). For example, Nieuwenhuis et al. (2003) manipulated Go/NoGo probability and indicated that frontocentral N2 amplitude was more strongly influenced by stimulus probability (i.e., rare > frequent) than response mapping (i.e., Go or NoGo), suggesting that the NoGo N2 might reflect conflict monitoring rather than response inhibition. The source of the NoGo N2 has been located in or near the anterior cingulate cortex (ACC) using dipole modeling, which is part of the neural circuit involved in conflict monitoring (Carter et al., 1998) in both adults (Bekker, Kenemans, & Verbaten, 2005; Nieuwenhuis et al., 2003) and children (Jonkman, Sniedt, & Kemner, 2007). Developmental studies have indicated that frontocentral NoGo N2 amplitude decreases with age during childhood (Ciesielski, Harris, & Cofer, 2004; Davis, Bruce, Snyder, & Nelson, 2003; Hämmerer, Li, Müller, & Lindenberger, 2010; Johnstone, Pleffer, Barry, Clarke, & Smith, 2005; Jonkman, 2006; Jonkman, Lansbergen, & Stauder, 2003). Based on the conflict monitoring theory of the N2, it is plausible that the decrease in NoGo N2 amplitude may reflect a reduction in conflict or a lower threshold with which to begin the cascade of processes involved in conflict monitoring (i.e., more efficient conflict resolution) during cognitive development.

NoGo P3. The NoGo P3 is a positive-going component occurring approximately 300 to 700 ms after stimulus onset, and displays a more frontal topographical distribution relative to the Go P3 (i.e., NoGo anteriorization; Bruin, Wijers, & van Staveren, 2001; Fallgatter & Strik, 1999; Pfefferbaum et al., 1985; Roberts, Rau, Lutzenberger, & Birbaumer, 1994), which typically has a topographic maximum over parietal scalp sites. Although there is still debate regarding the functional significance of the NoGo P3, accumulating evidence has demonstrated that the NoGo P3 is linked to response inhibition mechanisms (Bruin et al., 2001; Pfefferbaum et al., 1985; Randall & Smith, 2011; Roberts et al., 1994; Smith et al., 2008). For example, Bruin et al. (2001) used a cued-Go/NoGo task and observed larger NoGo P3 amplitudes for task conditions in which responses were primed more strongly by the cue (i.e., requiring greater amounts of inhibitory control), supporting the interpretation that the NoGo P3 reflects inhibitory processes. In contrast to the N2 component, it would appear that the effect of childhood development on the P3 component is task specific with associated alterations in scalp topography. Jonkman et al. (2003) indicated that adults exhibited larger P3 amplitude for a NoGo condition relative to a Go condition over frontocentral recording sites during an AX continuous performance test (AX-CPT), whereas children did not exhibit such differences, or even had larger P3 amplitude for the Go condition. Several studies have shown that adolescent and late-preadolescent children exhibit NoGo anteriorization, whereas this anteriorization is less pronounced or absent in early-preadolescent children (Hämmerer et al., 2010; Jonkman, 2006; Okazaki et al., 2004). Taken together, it would appear that increases in frontocentral NoGo P3 amplitude, which depict NoGo anteriorization, reflect development of inhibitory control during childhood.

Present Study

The aim of the present study was to examine whether childhood obesity is associated with inhibitory control. We compared task performance and the N2 and P3 components during a Go/NoGo task in healthy weight and obese preadolescent children. Based on previous findings, we hypothesized that obese children would exhibit lower response accuracy relative to healthy weight children and that this group difference would be disproportionately greater for the NoGo task relative to the Go task. We further predicted that obese children would exhibit larger N2 amplitude relative to healthy weight children. Given that we used the same target probability (0.2) across the Go and NoGo tasks, we predicted that N2 amplitude would not differ between these two tasks (Donkers & van Boxtel, 2004; Jonkman, 2006; Nieuwenhuis et al., 2003). Accordingly, we expected that a group difference in N2 amplitude would be observed across the Go and NoGo tasks. Lastly, we hypothesized that healthy weight children would exhibit a more frontal topographical distribution for the NoGo P3 relative to the Go P3, whereas such NoGo anteriorization would be less pronounced or absent for obese children.

Table 1. *Participant Demographics and Weight Status Data*

Measure	Healthy weight ≥5th to <85th BMI percentile	Obese ≥95th BMI percentile
No. of participants	37 (19 girls)	37 (19 girls)
Mean age (years)	8.9 (0.5)	9.0 (0.5)
Age range (years)	7.9–9.9	8.0–9.9
K-BIT composite score (IQ)	110.0 (13.8)	110.5 (11.1)
SES	1.7 (0.9)	1.8 (0.9)
VO_{2max} (ml/kg FFM/min)	51.2 (5.8)	50.5 (6.8)
BMI (kg/m^2)*	16.8 (1.3)	25.3 (2.8)
BMI percentile*	57.7 (20.0)	98.0 (1.3)
Whole-body percent fat (%)*	24.3 (6.5)	36.3 (6.2)

Notes. Data are expressed as mean (*SD*) unless otherwise specified. Participants were categorized using the Centers for Disease Control and Prevention BMI-for-age growth charts (Kuczmarski et al., 2000). K-BIT = Kaufman Brief Intelligence Test; SES = socioeconomic status; VO_{2max} = maximal oxygen consumption; FFM = fat free mass; BMI = body mass index.
*Significant difference, unpaired *t* test between groups, $p < .05$.

Methods

Participants

The present data stem from a larger ongoing longitudinal study (the FITKids Trial) investigating the effects of aerobic fitness on cognitive control and academic achievement. Preadolescent children between 7 and 9 years of age were recruited from the East Central Illinois region. Participants were paid $10/hour for their participation in this study. At pretest, 53 obese children (≥95th BMI percentile), classified according to the Centers for Disease Control and Prevention (CDC) BMI-for-age growth charts (Kuczmarski et al., 2000), completed the Go/NoGo task and underwent an assessment of body composition and aerobic fitness. After excluding participants who had either (a) high scores on the ADHD (attention deficit hyperactivity disorder) Rating Scale IV (≥90th percentile; DuPaul, Power, Anastopoulos, & Reid, 1998), (b) outliers in behavioral measures (±3 *SD*), (c) failing to meet criteria for maximal oxygen consumption (VO_{2max}; see below for details), or (d) excessive noise in the electroencephalogram (EEG) signal, we obtained 37 obese children for analyses. From a sample of 85 healthy weight children (≥5th to <85th BMI percentile; Kuczmarski et al., 2000) in the pretest data set, 37 participants who matched for sex and aerobic fitness with obese children were selected for analyses to exclude confounding effects of aerobic fitness on cognition (see Hillman, Erickson, & Kramer, 2008, for a review). Thus, analyses were conducted on 74 participants (37 healthy weight and 37 obese). Table 1 summarizes the participant demographics and weight status data for the healthy weight and obese groups. All weight status measures significantly differed between the healthy weight and obese groups, $ts(72) \geq 8.1$, $ps < .001$, whereas the other demographic measures did not differ between groups, $ts(72) \leq 0.7$, $ps \geq .51$. Prior to testing, legal guardians reported that their child was free of neurological diseases or physical disabilities, and indicated normal or corrected-to-normal vision. Participants and their legal guardians provided written informed assent/consent in accordance with the Institutional Review Board at the University of Illinois.

Laboratory Procedure

The experimental protocol occurred over two separate days for each participant. Participants were instructed not to consume any food or beverage containing caffeine prior to the experiment. On the first visit to the laboratory, informed assent/consent was obtained, participants completed the Kaufman Brief Intelligence Test (Kaufman & Kaufman, 1990) to assess IQ, and had their height and weight measured. Concurrently, participants' legal guardians completed a health history and demographics questionnaire, the ADHD Rating Scale IV (DuPaul et al., 1998), and the Physical Activity Readiness Questionnaire (Thomas, Reading, & Shephard, 1992) to screen for any previous health issues that might be exacerbated by exercise. Further, given that socioeconomic status (SES) has been associated with cognitive control (Mezzacappa, 2004) and adiposity (Shrewsbury & Wardle, 2008), SES was also assessed. SES was determined by creating a trichotomous index based on: (1) participation in a free or reduced-price meal program at school, (2) highest level of education obtained by the mother and father, and (3) number of parents who worked full time (Birnbaum et al., 2002). After completing all questionnaires, a graded exercise test on a motorized treadmill was performed to assess aerobic fitness. On the second visit, participants were fitted with a 64-channel Quik-Cap (Compumedics Neuroscan, El Paso, TX) and seated in a sound-attenuated room where the Go/NoGo task took place. The participant was given the instructions and practiced the task prior to the start of testing. Upon completion of the Go/NoGo task, all electrodes were removed, and then the DXA measurement was performed to assess body composition.

Go/NoGo Task

The Go/NoGo task used was a modification of a standard oddball task. The Go task asked participants to press a button with their right thumb to rare target stimuli (0.2 probability, a simple pictorial of a lion), which were presented within a train of frequent nontarget stimuli (0.8 probability, a simple pictorial of a tiger) that did not require a response. Following completion of the Go task, participants completed the NoGo task, which had them press a button to nontarget frequent stimuli (0.8 probability, tiger) and withheld a response to target rare stimuli (0.2 probability, lion). A fixed task order was used in this study to build a prepotent response to press a button to the rare stimulus during the Go task. This prepotent response would be further built by the NoGo instructions, which have the participants' respond to the frequent stimuli and inhibit their response to the rare stimuli. Thus, a fixed task order was deemed preferable to create conflict relative to inhibitory control during the NoGo task.

Participants were instructed to respond as quickly and accurately as possible. Before each task condition, the experimenter provided instructions and 20 practice trials were administered prior to the start of testing, with participants' performance checked to ensure that they achieved a sufficient level of accuracy (50%) prior to the start of the task. Participants then completed two experimental blocks of 125 trials of each task condition with a brief rest between blocks. The viewing distance was 1 m, and the stimuli subtended a horizontal visual angle of 2.6° and a vertical visual angle of 4.6°. Stimulus duration was 200 ms, with a 1,700-ms intertrial interval (ITI). Response accuracy was defined as the proportion of correct responses for the rare target stimuli for both the Go and NoGo task.

ERP Recording

EEG activity was measured from 64 electrode sites arranged in an extended montage based on the International 10-10 system

(Chatrian, Lettich, & Nelson, 1985), referenced to a midline electrode placed at the midpoint between Cz and CPz, with AFz serving as the ground electrode and interelectrode impedance at less than 10 kΩ. Electrooculographic activity was collected from electrodes placed above and below the right orbit and on the outer canthus of each eye to record bipolar eye movements. Continuous data were digitized at a sampling rate of 500 Hz, amplified 500 times with a DC to 70 Hz filter, and a 60 Hz notch filter using a Neuroscan Synamps2 amplifier (Neuro, Inc., Charlotte, NC). Offline EEG processing included: eye blink correction using a spatial filter (Compumedics Neuroscan), rereferencing to average mastoids, creation of stimulus-locked epochs (−100 to 1,000 ms relative to stimulus onset), baseline correction (−100 to 0 ms prestimulus period), low-pass filtering (30 Hz, 24 dB/octave), and artifact rejection (epochs with signals that exceeded ±75 μV were rejected). Trials with a response error were excluded from the ERP analyses. Across the Go and NoGo tasks, a mean of 33 and 34 trials were averaged for the healthy weight and obese group, respectively, which did not differ between groups, $t(72) = 0.4$, $p = .73$.

Based on visual inspection of the grand average ERP waveforms for target stimuli (Figure 1A), earlier components (i.e., N1 and P2) appeared to distort the N2 amplitude. Thus, we employed P2-N2 peak-to-peak amplitude rather than N2 peak-to-baseline amplitude. The P2 and N2 components were defined as the largest positive going peak within a 150–300 ms latency window and the largest negative going peak within a 250–400 ms latency window, respectively. Given that P3 waveforms exhibited a less pronounced peak, the P3 was evaluated as the mean voltage within a 400–700 ms latency window. Additionally, visual inspection of the grand average waveforms (Figure 1A) suggested that a late positive potential (LPP), which was observed during a Go/NoGo task in children (Davis et al., 2003), might superimpose on the P3. Thus, the LPP was also evaluated as the mean voltage within a 700–900 ms latency window.

Weight Status and Body Composition Assessment

Standing height and weight measurements were completed with participants wearing light-weight clothing and no shoes. Height and weight were measured using a Tanita WB-300 Plus digital scale (Tanita Corp., Tokyo, Japan). BMI was calculated by dividing body mass (kg) by height (m) squared [(kg)/ht(m)^2]. As mentioned above, participants were classified according to the CDC BMI-for-age growth charts (Kuczmarski et al., 2000) for this study. Although there are several BMI metrics available for weight assessment in children (e.g., BMI, BMI z-score, and BMI-for-age percentile), expert consensus committees recommend using BMI-for-age percentile cut points to categorize children with increased risk for complications of excess body weight (Barlow, 2007). Further, in terms of ranking children, all BMI metrics have nearly identical correlations with percent body fat (Field et al., 2003). Although BMI is correlated with body fat (Pietrobelli et al., 1998), it provides no direct information about body composition. Therefore, whole-body composition was also measured by DXA using a Hologic Discovery A bone densitometer (software version 12.7.3; Hologic Inc., Bedford, MA). Precision for DXA measurements of interest are ~ 1–1.5% in our laboratory.

Aerobic Fitness Assessment

VO_{2max} was measured using a motor-driven treadmill and a modified Balke protocol (American College of Sports Medicine, 2006), which involved walking/running on a treadmill at a constant speed with increasing grade increments of 2.5% every 2 min until volitional exhaustion. Oxygen consumption was measured using a computerized indirect calorimetry system (ParvoMedics True Max 2400, Sandy, UT) with averages for VO_2 and respiratory exchange ratio assessed every 20 s. A Polar heart rate monitor (Polar WearLink+ 31; Polar Electro, Kempele, Finland) was used to measure heart rate throughout the test, and ratings of perceived exertion were assessed every 2 min using the children's OMNI scale (Utter, Robertson, Nieman, & Kang, 2002). VO_{2max} was based upon maximal effort as evidenced by (a) a peak heart rate ≥ 185 bpm (American College of Sports Medicine, 2006) and a heart rate plateau (Freedson & Goodman, 1993); (b) respiratory exchange ratio ≥ 1.0 (Bar-Or, 1983); (c) a score on the children's OMNI ratings of perceived exertion scale ≥ 8 (Utter et al., 2002); and/or (d) a plateau in oxygen consumption corresponding to an increase of less than 2 ml/kg/min despite an increase in workload. Given that VO_{2max} relative to fat free mass (ml/kg FFM/min) has been considered a more valid measure than VO_{2max} relative to total body weight (ml/kg/min) for comparing aerobic fitness in children of different body size (Goran, Fields, Hunter, Herd, & Weinsier, 2000), this approach was adopted for this study.

Statistical Analysis

Response accuracy for the target stimulus was analyzed using a 2 (Group: healthy weight, obese) × 2 (Task: Go, NoGo) repeated measures multivariate analysis of variance (MANOVA). Additionally, based on our hypotheses, unpaired t tests for response accuracy were conducted separately for each task as planned comparisons. Reaction time (RT) was analyzed separately for each task using an unpaired t test. P2-N2 amplitude was assessed at the Fz and FCz electrode sites (Jonkman, 2006) using a 2 (Group) × 2 (Task) × 2 (Site: Fz, FCz) repeated measures MANOVA. P3 and LPP voltages were analyzed using a 2 (Group) × 2 (Task) × 5 (Site: Fz, FCz, Cz, CPz, Pz) repeated measures MANOVA. When significant interactions including the site factor (i.e., difference in topographic distribution as a function of group and/or task) were found in the P3 analysis, the same repeated measures MANOVA was conducted for normalized P3 voltage data using the vector scaling procedure (McCarthy & Wood, 1985) to check whether the interaction remained significant after vector scaling. Post hoc analyses with three or more within-participants levels used the Scheffe test. All statistical analyses were conducted using a significance level of $p = .05$.

Results

Preliminary analyses revealed that sex was not related to differences in task performance or neuroelectric measures as a function of group, task, or site. Accordingly, all further analyses were collapsed across both sexes.

Task Performance

Table 2 provides mean (*SD*) values for task performance for each group. Figure 2A illustrates overall response accuracy for each group, and Figure 2B illustrates the mean response accuracy for each group and task. Response accuracy was lower for the obese relative to the healthy weight group, $F(1,72) = 6.0$, $p = .02$, $\eta_p^2 = .08$, (see Figure 2A), and for the NoGo task relative to the Go task, $F(1,72) = 155.5$, $p < .001$, $\eta_p^2 = .68$. The planned comparisons

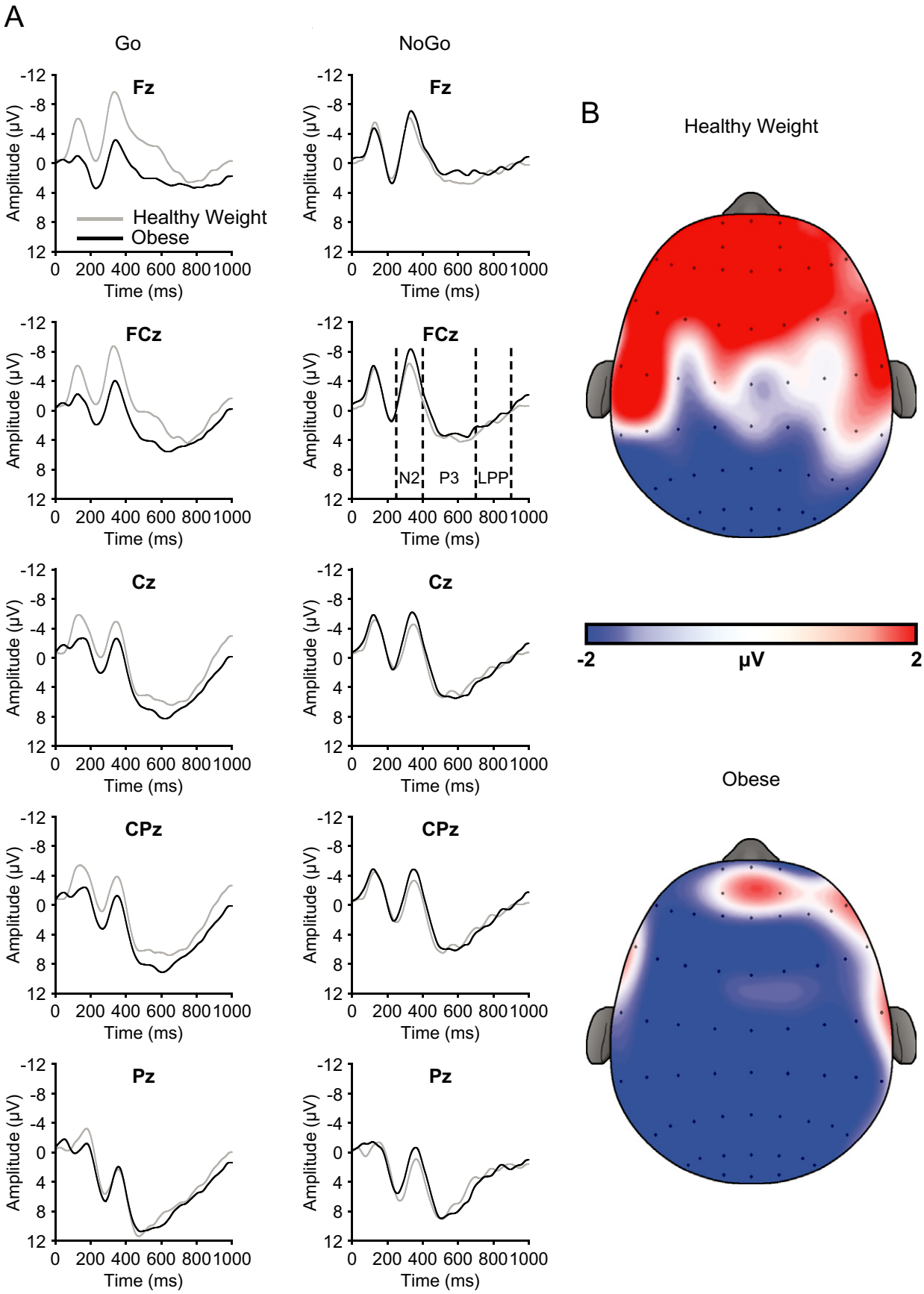

Figure 1. A: Grand average ERP waveforms of target stimuli for each group and task at each electrode site. Vertical lines indicate the latency windows for each component. B: Topographical maps of the mean P3 voltage of the NoGo–Go difference waves for each group.

Table 2. *Mean (SD) Values for Task Performance*

Measure	Healthy weight	Obese
Go response accuracy (%)	95.4 (6.1)	94.3 (5.7)
NoGo response accuracy (%)	77.9 (11.0)	71.6 (13.0)
Go reaction time (ms)	514.2 (75.2)	531.7 (83.2)
NoGo reaction time (ms)	429.5 (69.9)	422.8 (82.1)

(unpaired *t* tests for each task) revealed that response accuracy did not differ between groups for the Go task, $t(72) = 0.7$, $p = .46$, whereas response accuracy for the obese group was significantly lower than the healthy weight group for the NoGo task, $t(72) = 2.3$, $p = .03$ (see Figure 2B). Although the use of bimodal healthy weight and obese groups designates BMI as a noncontinuous variable, scatter plots are shown in Figure 2C to visualize the negative association between weight status and inhibitory control. RT analysis revealed no significant difference between groups for both the Go and NoGo task, $t(72) \leq 1.0$, $p \geq .35$.

N2 Component

Figure 1A illustrates grand average ERP waveforms for target stimuli for each group and task at each electrode site. Analysis for P2-N2 amplitude revealed a Group × Task interaction, $F(1,72) = 5.9$, $p = .02$, $\eta_p^2 = .08$. Figure 3 illustrates the Group × Task interaction, which demonstrated no difference between the Go and NoGo tasks for the healthy weight group, $t(36) = 0.4$, $p = .71$, whereas larger P2-N2 amplitude was observed for the NoGo task relative to the Go task for the obese group, $t(36) = 3.0$, $p = .005$. Secondary analyses comparing groups within each task revealed no significant between-group difference for both the Go and NoGo task, $t(72) \leq 1.4$, $p \geq .18$. A Task × Site interaction was also significant, $F(1,72) = 6.6$, $p = .01$, $\eta_p^2 = .08$. Post hoc analyses revealed that P2-N2 amplitude did not differ between the Go and NoGo tasks at Fz, $t(73) = 1.0$, $p = .31$, whereas P2-N2 amplitude for the NoGo task was larger than the Go task at FCz, $t(73) = 2.5$, $p = .01$.

P3 Component

The omnibus analysis for P3 voltage revealed a main effect for site, $F(4,69) = 21.5$, $p < .001$, $\eta_p^2 = .56$, and a Task × Site interaction, $F(4,69) = 9.7$, $p < .001$, $\eta_p^2 = .36$, which were superseded by a Group × Task × Site interaction, $F(4,69) = 4.5$, $p = .003$, $\eta_p^2 = .21$. The three-way interaction remained significant after vector scaling, $F(4,69) = 5.5$, $p = .001$, $\eta_p^2 = .24$. Figure 4 illustrates the Group × Task × Site interaction. Decomposition of the three-way interaction examined Task × Site within each group and revealed a Task × Site interaction only for the healthy weight group, $F(4,33) = 11.0$,

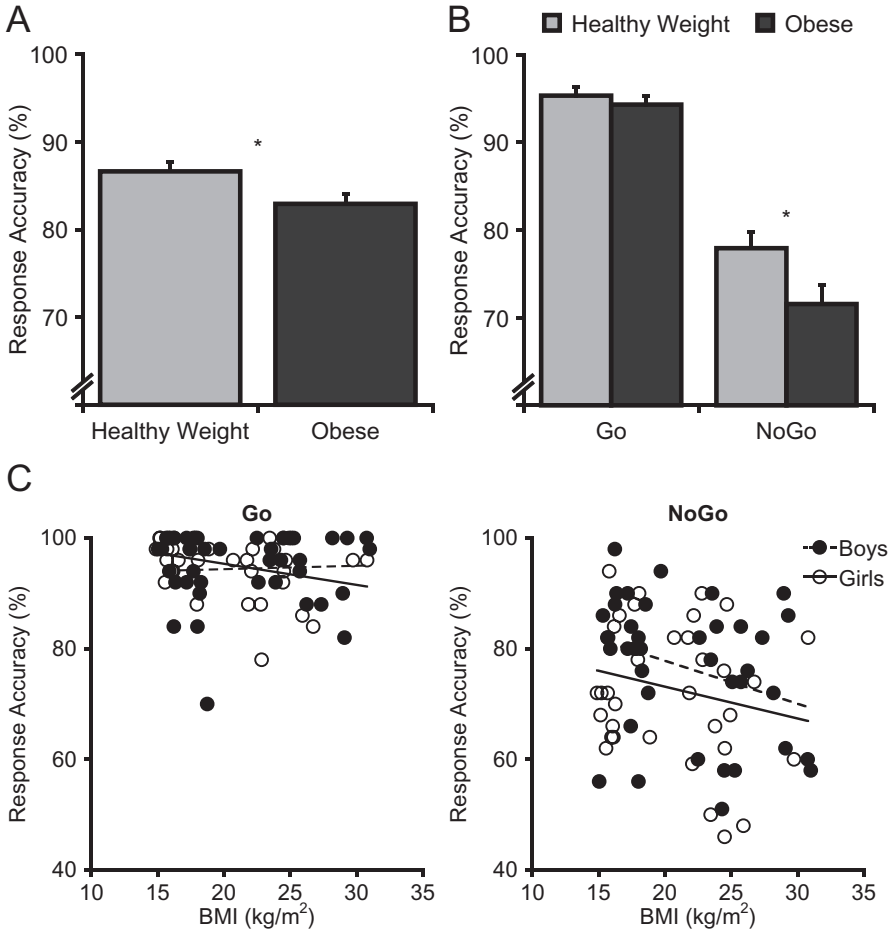

Figure 2. A: Mean (*SE*) response accuracy for each group collapsed over task. B: Mean (*SE*) response accuracy for each group and task. C: Scatter plots for the relationship between body mass index and response accuracy for each task.

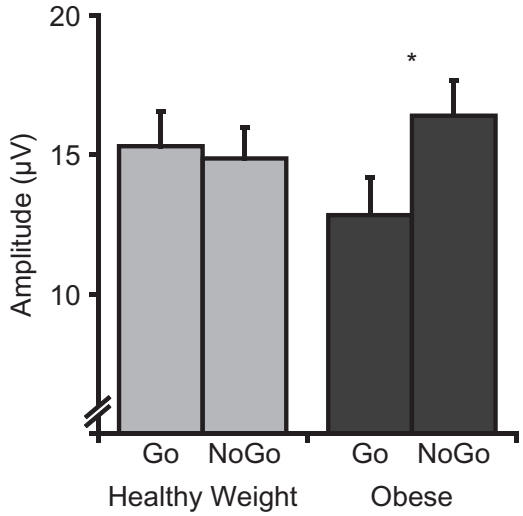

Figure 3. Mean (*SE*) P2-N2 peak-to-peak amplitude for each group and task collapsed over electrode.

$p < .001$, $\eta_p^2 = .57$. The two-way interaction remained significant after vector scaling, $F(4,33) = 7.4$, $p < .001$, $\eta_p^2 = .47$. Scheffe post hoc analyses for the healthy weight group revealed larger Go P3 voltage at Pz relative to the other four sites, $p \leq .01$; CPz relative to Fz, FCz, $p < .001$; Cz relative to Fz, FCz, $p < .001$; and FCz relative to Fz, $p = .02$; whereas larger NoGo P3 voltage was only observed at Pz relative to Fz, FCz, Cz, $p \leq .02$, and CPz relative to Fz, $p = .007$ (see Figure 4).

For the obese group, although a site main effect was found, $F(4,33) = 11.1$, $p < .001$, $\eta_p^2 = .57$, no such interaction was observed, $F(4,33) = 1.6$, $p = .20$, $\eta_p^2 = .16$. Scheffe post hoc analyses of site for the obese group revealed larger P3 voltage at Pz relative to Fz, FCz, Cz, $p \leq .01$; CPz relative to Fz, FCz, $p \leq .01$; and Cz relative to Fz, $p < .001$, across the Go and NoGo tasks (see Figure 4). These analyses indicate that the healthy weight group had a more frontal distribution for the NoGo P3 relative to the Go P3 (i.e., NoGo anteriorization), whereas obese children had similar topographic distributions between the Go P3 and NoGo P3. To visualize the topographic difference in P3 voltage as a function of group and task, the NoGo–Go difference waveforms were calculated (see Figure 1B).

Secondary post hoc analyses examining Group × Task within each site revealed the Group × Task interactions at Fz and FCz, $F(1,72) \geq 6.2$, $p \leq .02$, $\eta_p^2 \geq .08$, but not at Cz, CPz, and Pz, $F(1,72) \leq 1.5$, $p \geq .22$, $\eta_p^2 \leq .02$. However, post hoc analyses comparing groups within each task revealed no significant difference for both the Go and NoGo tasks at Fz and FCz, $t(72) \leq 2.1$, $p \geq .04$ (i.e., after Bonferroni correction).

LPP

Analysis for LPP voltage revealed main effects for task, $F(1,72) = 12.3$, $p = .001$, $\eta_p^2 = .15$, and site, $F(4,69) = 5.0$, $p = .001$, $\eta_p^2 = .23$, which were superseded by a Task × Site interaction, $F(4,69) = 3.0$, $p = .02$, $\eta_p^2 = .15$. Post hoc analyses revealed that LPP voltage did not differ between the Go and NoGo tasks at Fz, $t(73) = 1.9$, $p = .06$, whereas LPP voltage for the Go task was larger than the NoGo task at the other four sites, $t(73) \geq 2.8$, $p \leq .007$. There were no main effects or interactions involving the group factor.

Discussion

Task Performance

Consistent with *a priori* predictions, obese children exhibited lower response accuracy relative to healthy weight children, and this group difference was disproportionately greater for the NoGo task relative to the Go task. Developmental studies have reported no difference in response accuracy between preadolescent children and young adults for a Go task, whereas lower response accuracy has been found for children relative to young adults for a NoGo task (Casey et al., 1997; Ciesielski et al., 2004). These prior findings imply that cognitive performance during childhood differs based on the aspects of cognition studied. That is, preadolescent children might be able to detect target stimuli at the same level of young adults, whereas their ability to inhibit a prepotent response remains relatively immature. It has been proposed that the late development of inhibitory control is associated with protracted maturation of the prefrontal cortex (Diamond, 2002). Thus, the present findings suggest that childhood obesity is selectively associated with poorer prefrontal inhibitory control, which may differ from the developmental trajectory of healthy weight children.

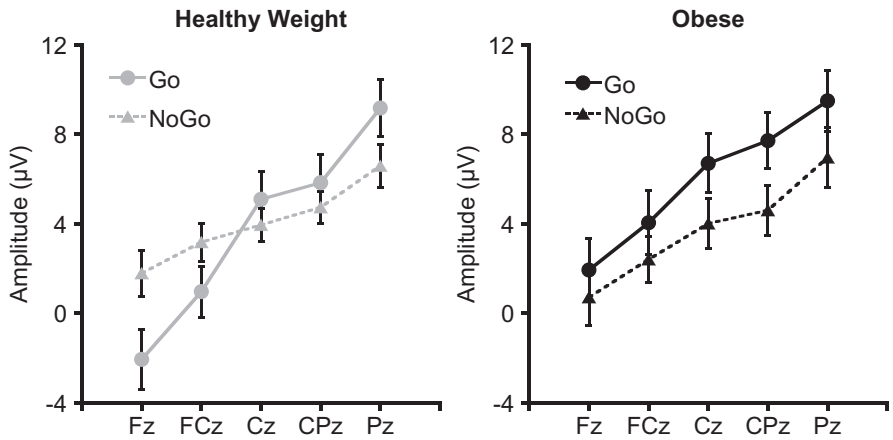

Figure 4. Mean (*SE*) P3 voltage for each group and task at each electrode.

N2 Component

The difference in N2 amplitude as a function of group and task was unexpected; obese children had larger N2 amplitude for the NoGo task relative to the Go task, whereas this difference was not observed for healthy weight children. Given that the target probability (0.2) was similar between the Go and NoGo tasks, we predicted that N2 amplitude would not differ between tasks based on the conflict monitoring theory of the N2 (Donkers & van Boxtel, 2004; Jonkman, 2006; Nieuwenhuis et al., 2003). Several studies have shown larger NoGo N2 in adults, relative to Go N2, even when the stimulus probability was consonant (Bruin & Wijers, 2002; Nieuwenhuis et al., 2003). Nieuwenhuis et al. (2003) explained this N2 effect as resulting from an increased bias toward the Go response due to task instructions that emphasized response speed, thereby leading to increased conflict on NoGo trials. However, in the present study, task instructions emphasized both response speed and accuracy. It has been suggested that childhood obesity is associated with impulsivity, with obese children responding more impulsively relative to healthy weight children (Braet, Claus, Verbeken, & Van Vlierberghe, 2007; van den Berg et al., 2011). It is plausible that the impulsive performance tendencies in obese children may be naturally biased toward a Go response, resulting in more response conflict on NoGo trials, despite task instructions emphasizing both response speed and accuracy. If this assumption is valid, larger N2 amplitude in obese children during the NoGo task relative to the Go task demonstrates that they may experience more conflict during the NoGo task.

By contrast, N2 amplitude did not differ between the Go and NoGo tasks for the healthy weight group. It has been proposed that the ACC, which is a neural generator of the N2 (Bekker et al., 2005; Jonkman et al., 2007; Nieuwenhuis et al., 2003), serves to evaluate for the presence of response conflict and signals other areas of the brain, such as the dorsolateral prefrontal cortex, which act to upregulate cognitive control, to reduce conflict and to prevent future performance decrements (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Carter & van Veen, 2007). As such, a decrease in N2 amplitude may reflect reduced conflict (i.e., reduced ACC activation) due to the upregulation of cognitive control. Based on this assumption, and coupled with the fact that healthy weight children showed greater response accuracy during the NoGo task, the pattern of N2 findings in healthy weight children implies that they might flexibly upregulate cognitive control to meet task demands during the NoGo task requiring greater amounts of cognitive control. Conversely, obese children might not be capable of flexibly modulating cognitive control operations based on task demands.

It should be noted that the pattern of N2 findings in healthy weight children is inconsistent with previous studies that have indicated larger NoGo N2 relative to Go N2 for adults even when the stimulus probability was held constant across tasks (Bruin & Wijers, 2002; Nieuwenhuis et al., 2003). One possible source of this discrepancy is the difference in task instructions as discussed above. That is, the lack of difference in N2 amplitude between Go and NoGo tasks for healthy weight children may simply be due to task instructions rather than the upregulation of cognitive control. More important, obese children exhibited larger NoGo N2 relative to Go N2 even when task instructions emphasized both response speed and accuracy. Further studies are required to support current arguments using manipulation of task instructions and neuroimaging techniques.

P3 Component

The observed topographic difference in P3 as a function of group and task is consistent with expectation of the negative association between obesity and inhibitory control. As hypothesized, healthy weight children exhibited a more anterior distribution for the NoGo P3 relative to the Go P3, whereas obese children had similar topographic distributions across tasks. Jonkman (2006) indicated that, although late-preadolescent children (9–10 years) had smaller NoGo P3 voltage relative to young adults (19–23 years) across frontal and parietal recording sites, both groups exhibited similar topographic distributions of the NoGo P3. By contrast, the topographic distribution of the NoGo P3 was more parietally dominant for early-preadolescent children (6–7 years) relative to young adults (Jonkman, 2006). Thus, it would appear that the NoGo anteriorization may start around 9–10 years, which corresponds to the age of the current participants. The NoGo anteriorization has been hypothesized to reflect prefrontal inhibitory control (Fallgatter & Strik, 1999; Strik, Fallgatter, Brandeis, & Pascual-Marqui, 1998). In fact, Fallgatter and his colleagues have reported that patients who generally have deficits of inhibitory control, such as ADHD and obsessive-compulsive disorder, exhibited reduced NoGo anteriorization relative to healthy control participants (Fallgatter et al., 2004; Herrmann, Jacob, Unterecker, & Fallgatter, 2003). Collectively, it is plausible that the less pronounced NoGo anteriorization for obese relative to healthy weight children may reflect inferior inhibitory control and/or relatively delayed development of the prefrontal cortex.

Note that, as may be seen in the grand average ERP waveforms, the topographic difference in P3 as a function of group and task appears to primarily be due to group differences in the Go P3 rather than the NoGo P3. Based on an inhibition hypothesis of the P3, suggesting that P3 amplitude is proportional to the amount of attentional resources allocated toward the suppression of extraneous neuronal activity (Polich, 2007), numerically larger Go P3 for obese children relative to healthy weight children may reflect greater inhibition. However, given that task performance did not differ between groups during the Go task, it is plausible that these group differences may indicate less efficient attentional allocation for obese children. Conversely, the upregulation of cognitive control in healthy weight children during the NoGo task might increase frontocentral NoGo P3, and, consequently, similar frontocentral NoGo P3 voltages were observed between groups.

Limitations

Despite demonstrating a negative association between childhood obesity and inhibitory control, some caution is needed in interpreting the current findings. First, given that we used a fixed ITI (1,700 ms), the contingent negative variation (CNV) was elicited during the ITI. To exclude possibility of the effects of CNV on the N2 and P3 components, we assessed the CNV. The mean CNV voltage was calculated from the 200-ms period prior to the target onset, and analyzed in a similar manner to the P3. The analysis revealed that there were no main effects or interactions involving the group factor. Thus, it is plausible that the CNV did not influence the current result. However, we cannot deny the possibility that movement-related potentials, which may be elicited after target onset, might influence the current ERP findings.

Second, it should be noted that the LPP was assessed as it might superimpose on the P3 component. Davis et al. (2003) observed the LPP for children, but not for adults, during a Go/NoGo task, suggesting that children may involve additional neural sources to

perform the Go/NoGo task. In addition, given that the LPP for the Go condition was larger than the NoGo condition in children, they suggested that the component may reflect processes involved in maintaining response inhibition or the evaluation of a response. In the present study, the LPP was larger for the Go task relative to the NoGo task, which replicates the findings of Davis et al. across both groups. Thus, although the functional significance of the LPP remains an open question, the processes reflected by the LPP do not appear to be associated with childhood obesity. More important, given that the LPP did not differ between groups, it is plausible that the LPP did not influence the current findings of topographic difference in P3 between groups.

Lastly, we should emphasize again that recent findings of longitudinal studies suggest bidirectional associations between weight status and cognitive control in children (Datar & Sturm, 2006; Donnelly et al., 2009; Graziano et al., 2010; Hollar, Lombardo et al., 2010, Hollar, Messiah et al., 2010; Yokum et al., 2012; see also Smith et al., 2011, for a review). Since the present study was cross-sectional, the current findings simply indicate an association between childhood obesity and inhibitory control, but not the direction of the association. It is highly plausible that reduced inhibitory control may lead to obesity due to the inability to regulate specific behaviors.

Summary

The present study provides new insight into the negative association between childhood obesity and cognitive control using neuroelectric measures of inhibition. In summary, childhood obesity was associated with poorer inhibitory control, as reflected by lower response accuracy during the NoGo task and less pronounced NoGo P3 anteriorization. Further, childhood obesity might also be associated with a decreased ability to flexibly modulate cognitive control operations based on task demands, as reflected by larger NoGo, relative to Go, N2 amplitude. These findings suggest that childhood obesity is negatively and selectively associated with prefrontal inhibitory control.

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