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The Negative Association of Childhood Obesity to Cognitive Control of Action Monitoring

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The global epidemic of childhood obesity has become a major public health concern. Yet, evidence regarding the association between childhood obesity and cognitive health has remained scarce. This study examined the relationship between obesity and cognitive control using neuroelectric and behavioral measures of action monitoring in preadolescent children. Healthy weight and obese children performed compatible and incompatible stimulus–response conditions of a modified flanker task, while task performance and the error-related negativity (ERN) were assessed. Analyses revealed that obese children exhibited a longer reaction time (RT) relative to healthy weight children for the incompatible condition, whereas no such difference was observed for the compatible condition. Further, obese children had smaller ERN amplitude relative to healthy weight children with lower post-error response accuracy. In addition, healthy weight children maintained post-error response accuracy between the compatible and incompatible conditions with decreased ERN amplitude in the incompatible condition, whereas obese children exhibited lower post-error response accuracy for the incompatible relative to the compatible condition with no change in ERN amplitude between the compatibility conditions. These results suggest that childhood obesity is associated with a decreased ability to modulate the cognitive control network, involving the prefrontal cortex and anterior cingulate cortex, which supports action monitoring.

Keywords: anterior cingulate cortex, body mass index, cognitive health, error-related negativity, preadolescent children

Introduction

Recent studies have suggested that childhood obesity is inversely associated with cognitive control (Cserjesi et al. 2007; Li et al. 2008; Lokken et al. 2009; Kamijo et al. forthcoming). Cognitive control refers to "the ability to orchestrate thought and action in accord with internal goals" (Miller and Cohen 2001). The core cognitive processes, collectively termed "cognitive control," include inhibition, working memory, and cognitive flexibility (Diamond 2006). However, not all studies have uniformly shown an association between childhood obesity and poorer cognitive control. For instance, Gunstad et al. (2008) used several cognitive tasks requiring variable amounts of working memory and cognitive flexibility and indicated no association between weight status and cognitive performance in children. Thus, empirical evidence regarding the association between childhood obesity and cognitive control has remained inconclusive. Considering the current worldwide epidemic of childhood obesity (Ebbeling et al. 2002; Swinburn et al. 2011), further investigation is needed to elucidate whether obesity is associated with cognitive control in children. Positive findings would suggest that being obese is adversely related to cognitive development and brain health. Hence, the present study focused on a different aspect of cognitive control (i.e., action monitoring), which has not been previously investigated, to provide a new insight into the association of childhood obesity to cognitive and brain health.

Action monitoring is required to orchestrate goal-directed behaviors. That is, individuals must continuously monitor their correspondence between intended and executed actions, and correct response errors during subsequent environmental interaction for the maintenance and adaptation of successful performance. On laboratory tasks, it is well founded that individuals slow their response speed following an error of commission (i.e., post-error response slowing; Rabbitt 1966), possibly to prevent subsequent errors. Such action is believed to serve as a behavioral indicator of the increased recruitment and implementation of cognitive control (Botvinick et al. 2001; Kerns et al. 2004), since increases in post-error response slowing and post-error response accuracy are considered to reflect the upregulation of cognitive control.

Developmental studies have indicated that preadolescent children exhibit similar post-error response slowing as adolescent children and young adults (Davies et al. 2004; Santesso et al. 2006; Ladouceur et al. 2007; Wiersema et al. 2007), suggesting that this behavioral measure of action monitoring may not be sensitive enough to detect subtle developmental effects. Thus, an additional neuroelectric measure, which has been found to reflect the action monitoring system, error-related negativity (ERN; Gehring et al. 1993; or error negativity; Falkenstein et al. 1991) may lead to further understanding the association between childhood obesity and cognitive control. The ERN is a negative-going component of the response-locked event-related brain potential (ERP) occurring approximately 50–100 ms after errors of commission with a topographic maximum over fronto-central recording sites. The neural tissue underlying the generation of the ERN has been localized to the dorsal portion of the anterior cingulate cortex (ACC; Dehaene et al. 1994; Carter et al. 1998; van Veen and Carter 2002; Miltner et al. 2003; Ladouceur et al. 2007), which is a part of the neural circuit involved in action monitoring (Carter et al. 1998; Kerns et al. 2004).

The ERN has been theorized to reflect the detection of conflict in the ACC (Botvinick et al. 2001; Yeung et al. 2004), or the transmission of a negative reinforcement learning signal to the ACC (Holroyd and Coles 2002). Thus, although there is still a debate regarding the functional significance of the ERN; it is generally thought to reflect activation of action monitoring processes in response to erroneous behaviors, which

serve to initiate the upregulation of top-down compensatory processes to correct an individual's responses in support of subsequent environmental interaction (Falkenstein et al. 1991; Gehring et al. 1993; Gehring and Knight 2000). The ERN has been observed in various kinds of cognitive tasks, including flanker tasks (e.g., Gehring et al. 1993; Davies et al. 2004; Santesso et al. 2006; Ladouceur et al. 2007; Pontifex et al. 2011) and Go/NoGo tasks (e.g., Falkenstein et al. 1991; Scheffers et al. 1996; Wiersema et al. 2007; Torpey et al. 2012), which modulate inhibitory control requirements across task conditions. Further, several studies have indicated that the ERN shows similar characteristics irrespective of stimulus or response modality (e.g., Falkenstein et al. 1991; Holroyd et al. 1998; Nieuwenhuis et al. 2001), suggesting that the ERN reflects a generic action monitoring system.

Developmental studies have consistently demonstrated that ERN amplitude increases with age during childhood (Davies et al. 2004; Santesso et al. 2006; Ladouceur et al. 2007; Wiersema et al. 2007; Torpey et al. 2012), suggesting that smaller ERN amplitude for younger children may reflect protracted maturation of the ACC and/or the neural circuit for action monitoring. That is, it would appear that smaller ERN amplitude reflects less effective action monitoring during childhood. Accordingly, if childhood obesity is inversely associated with cognitive control, obese children would be expected to exhibit smaller ERN amplitude relative to their healthy weight peers.

In the current study, we manipulated cognitive control demands using compatible and incompatible stimulus–response conditions of a modified flanker task (Eriksen and Eriksen 1974), consisting of congruent (e.g., >>>>>) and incongruent (e.g., <<><<) arrays. In the incompatible condition, participants were instructed to press a button that opposed the direction of the central target, which differs from the compatible condition in that the stimulus and response were consonant in directionality. Given that the incompatible condition increases response conflict due to the need to over-ride prepotent responses, this condition requires the upregulation of cognitive control—relative to the compatible condition—to manage the increased conflict (Friedman et al. 2009). Based on a previous ERN study using this manipulation (Pontifex et al. 2011), it would appear that decreased flexible modulation of cognitive control is reflected by a smaller change in ERN amplitude between the stimulus–response compatibility conditions and greater reduction in task performance for the incompatible condition.

The present study was designed to investigate the association between childhood obesity and cognitive control using the neuroelectric (i.e., ERN) and behavioral (post-trial RT and post-error response accuracy) measures of action monitoring, as well as overall task performance (i.e., RT, response accuracy), during compatible and incompatible stimulus–response conditions of a flanker task. This study employed a cross-sectional design, comparing these neuroelectric and task performance measures in healthy weight and obese preadolescent children. We predicted that obese children would exhibit a longer RT and lower response accuracy relative to healthy weight children. We also predicted that obese children would exhibit a shorter post-error RT, lower post-error response accuracy, and smaller ERN amplitude, reflecting less effective action monitoring and upregulation of cognitive control. Further, we predicted that these group differences would be disproportionately greater for the incompatible condition requiring greater upregulation of cognitive control relative to the compatible condition. Lastly, obese children were expected to exhibit a smaller increase in ERN amplitude and greater reduction in task performance between the compatible and incompatible conditions, suggesting decreased flexibility in the modulation of cognitive control.

Materials and Methods

Participants

Participants for this study were part of a larger ongoing longitudinal study (the FITKids study), investigating the effects of cardiorespiratory fitness on cognitive control and academic achievement. Preadolescent children between 7 and 9 years old were recruited from the East Central Illinois region. At pretest, 52 obese children (≥95th body mass index [BMI] percentile) classified according to the Centers for Disease Control and Prevention BMI-for-age growth charts (Kuczmarski et al. 2000), completed compatible and incompatible conditions of a modified flanker task, and underwent an assessment of body composition and cardiorespiratory fitness. After excluding participants who had either 1) high scores on the attention-deficit/hyperactivity disorder (ADHD) Rating Scale IV (≥90th percentile; DuPaul et al. 1998), 2) failed to meet criteria for maximal oxygen consumption (VO_{2max}; see below for details), 3) at or below chance performance (≤50% accuracy), or 4) an insufficient number of trials available for computing ERP average waveforms (<6 trials; Olvet and Hajcak 2009; Pontifex et al. 2010), we obtained 37 obese children for analyses. In the same manner, we yoked 37 healthy weight children (≥5th to <85th BMI percentile; Kuczmarski et al. 2000) from the pretest dataset who were matched for sex and cardiorespiratory fitness with obese children to exclude confounding effects of cardiorespiratory fitness on cognition (for a review, see Hillman et al. 2008). Thus, analyses were conducted on 74 participants (37 healthy weight and 37 obese). Demographic and weight status data for this sample are provided in Table 1. All weight status measures significantly differed between groups ($t_{72} \geq 8.1$; $P < 0.001$), whereas the other demographic measures did not differ ($t_{72} \leq 0.9$; $P \geq 0.36$). 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 and consent in accordance with the Institutional Review Board at the University of Illinois.

Laboratory Procedure

On the first visit to the laboratory, participants completed an informed assent, the Kaufman Brief Intelligence Test (Kaufman and Kaufman 1990) to assess intelligence quotient and had their height and weight measured. Concurrently, participants' legal

Table 1
Mean (SD) values for participant demographics and weight status data

Measure	Healthy weight	Obese
	≥5th to <85th BMI percentile	≥95th BMI percentile
No. of participants	37 (20 girls)	37 (20 girls)
Mean age (years)	8.8 (0.6)	8.9 (0.6)
Age range (years)	7.9–9.9	8.0–9.9
K-BIT composite score (IQ)	112.5 (13.2)	110.4 (10.9)
SES	1.9 (0.9)	1.8 (0.9)
VO_{2max} (mL/kg FFM/min)	50.9 (5.7)	49.5 (7.3)
BMI (kg/m^2)*	16.7 (1.4)	25.2 (2.9)
BMI percentile*	56.8 (19.9)	98.0 (1.4)
Whole-body percent fat (%)*	24.7 (6.6)	36.7 (6.1)

Note: Participants were categorized using the Centers for Disease Control and Prevention BMI-for-age growth charts (Kuczmarski et al. 2000). Whole-body percent fat was measured by dual-energy X-ray absorptiometry. Significant difference, unpaired *t*-test between groups, *$P < 0.05$.

K-BIT: Kaufman Brief Intelligence Test; SES: socioeconomic status; VO_{2max}: maximal oxygen consumption; FFM: fat-free mass; BMI: body mass index.

guardians completed an informed consent, health history, demographics questionnaire, and the ADHD Rating Scale IV (DuPaul et al. 1998). Further, given that socioeconomic status (SES) has been associated with cognitive control (Mezzacappa 2004) and adiposity (Shrewsbury and Wardle 2008), SES was assessed by creating a trichotomous index based on: 1) Participation in a free or reduced price meal program at school, 2) the highest level of education obtained by the mother and father, and 3) the 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 cardiorespiratory fitness.

On the second visit, participants were fitted with a 64-channel Quik-Cap (Compumedics Neuroscan, El Paso, TX, United States of America) and seated in a sound-attenuated room where the flanker task was administered. Participants were given instructions, afforded the opportunity to ask questions, and practiced the task prior to the start of testing. Upon completion of the flanker task, a dual-energy X-ray absorptiometry (DXA) measurement was performed to assess body composition.

Flanker Task

A modified flanker task asked participants to respond to a centrally presented target stimulus amid an array of 4 flanking stimuli, which were task irrelevant. Both the target and flanking stimuli were left- or right-oriented fish. The flanker task consisted of congruent trials, in which flanking fish faced in the same direction as the target fish; and incongruent trials, in which flanking fish faced in the opposite direction from the target fish. Congruent and incongruent trials were equiprobable and random. In the compatible condition, participants responded to the direction of the target fish with their consonant thumb. Next, participants completed the incompatible condition wherein they responded with a button press in the direction opposite to that of the target fish. After 40 practice trials, participants completed 150 trials (75 trials × 2 blocks) in each compatibility task. The viewing distance was 1 m and the stimuli subtended a horizontal visual angle between the 2 outside positions of 14.8° and a vertical visual angle of 3.2°. Stimulus duration was 200 ms, with a 1700-ms intertrial interval.

Task Performance

Behavioral data were collected on response latency for correct trials and response accuracy for all trials within each congruency and compatibility condition. Post-error RT was defined as the mean RT for correct trials following an error of commission trial minus the mean RT for the error of commission trials. In the same manner, post-matched-correct RT was also calculated as the mean RT for correct trials following a matched-correct trial minus the mean RT for matched-correct trials (i.e., the subset of correct trials matched to specific error trials based on RT). Post-error and post-matched-correct response accuracy were defined as the percentage of correct responses following errors of commission trials and matched-correct trials, respectively. Post-trial task performance was calculated across congruency due to an insufficient number of errors of commission in the congruent trials.

ERP Recording

Electroencephalographic (EEG) activity was measured from 64 electrode sites, referenced to a midline electrode placed at the midpoint between Cz and CPz, with AFz serving as the ground electrode, and interelectrode impedance at <10 kΩ. Additional electrodes were placed above and below the left orbit and the outer left and right canthi to monitor electro-oculogram activity with bipolar recording. Continuous data were digitized at a sampling rate of 500 Hz, amplified 500 times with a direct current to 70 Hz filter, and a 60-Hz notch filter using a Neuroscan Synamps2 amplifier (Neuro, Inc., Charlotte, NC, United States of America). Offline EEG processing included: Eye blink correction using a spatial filter (Compumedics Neuroscan 2003), re-referencing to average mastoids, creation of response-locked epochs (−600 to 1000 ms relative to response onset), baseline correction (−400 to −200 ms relative to response onset), bandpass filtering (1–12 Hz, 24 dB/octave), and artifact rejection (epochs with signals that exceeded ±75 μV were rejected). Average ERP waveforms were created for the error of commission trials (i.e., ERN) and correct trials (i.e., correct response negativity: CRN), which were individually matched (without replacement) to an error of commission trial with the closest possible RT latency (Coles et al. 2001) to account for potential artifacts that may exist due to differences in response latency between correct and incorrect trials (Falkenstein et al. 2001; Mathewson et al. 2005). Torpey et al. (2012) observed associations of age and task performance with the error-correct difference wave (i.e., ΔERN), but not ERN, amplitude in children, suggesting that ΔERN may be a valid measure to assess children's action monitoring processes. Thus, we also calculated the ΔERN amplitude for analyses. Trials with an error of omission were rejected and the waveforms were averaged across congruency. These response-locked ERP components were defined as the largest negative-going peak within a −50- to 100-ms latency window relative to response onset, and its amplitude was quantified as the mean voltage in the 30-ms interval around the peak.

Weight Status and Body Composition Assessment

Standing height and weight measurements were completed with participants wearing lightweight 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 body mass (kg)/height (m^2). Although BMI is correlated with body fat (Pietrobelli et al. 1998), it does not provide direct information regarding 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, United States of America). Precision for DXA measurements of interest is approximately 1–1.5% in our laboratory.

Cardiorespiratory Fitness Assessment

VO_{2max} was measured using a computerized indirect calorimetry system (ParvoMedics True Max 2400, Sandy, UT, United States of America), with averages for VO_2 and respiratory exchange ratio (RER) assessed every 20 s. A modified Balke protocol (American College of Sports Medicine 2006) was employed using a motor-driven treadmill at a constant speed with increases in grade increments of 2.5% every 2 min until volitional exhaustion occurred. VO_{2max} was based on maximal effort as evidenced by 1) a peak heart rate ≥185 bpm (American College of Sports Medicine 2006) and a heart rate plateau (Freedson and Goodman 1993); 2) RER ≥1.0 (Bar-Or 1983); 3) a score on the children's OMNI ratings of perceived exertion scale ≥8 (Utter et al. 2002); and/or 4) a plateau in oxygen consumption corresponding to an increase of <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 cardiorespiratory fitness in children of different body size (Goran et al. 2000), this approach was adopted for this study.

Statistical Analysis

RT and response accuracy were analyzed using a 2 (Group: Healthy weight and obese) × 2 (Compatibility: Compatible and incompatible) × 2 (Congruency: Congruent and incongruent) repeated-measures multivariate analysis of variance (MANOVA). Post-error RT and post-error response accuracy were analyzed using a 2 (Group) × 2 (Compatibility) repeated-measures MANOVA. ERN and ΔERN amplitudes were assessed at the FCz electrode site, where it reached its topographic maximum, using a 2 (Group) × 2 (Compatibility) repeated-measures MANOVA. Post hoc analyses were conducted using Bonferroni-corrected *t*-tests. Additionally, based on our a priori hypotheses, planned comparisons were conducted to examine the group difference in the dependent variables within each compatible condition using unpaired *t*-tests. A second planned comparison examined the compatibility effect on dependent variables within each group using paired *t*-tests. Lastly, Pearson correlation analyses were conducted to examine whether ΔERN amplitude, which has been

more robustly associated with task performance relative to ERN amplitude (Torpey et al. 2012), is associated with post-error task performance for each group and compatibility condition. All statistical analyses were conducted using a significance level of $P = 0.05$ prior to Bonferroni correction.

Results

Table 2 provides mean (standard deviation, SD) values for neuroelectric and behavioral measures for each group and compatibility condition.

Reaction Time

Analysis revealed main effects for Group ($F_{1,72} = 5.4$, $P = 0.02$, $\eta_p^2 = 0.07$) and Compatibility ($F_{1,72} = 47.8$, $P < 0.001$, $\eta_p^2 = 0.40$), which were qualified by a Group × Compatibility interaction ($F_{1,72} = 4.5$, $P = 0.04$, $\eta_p^2 = 0.06$). Post hoc analyses comparing the group difference within each compatibility condition revealed no group difference for the compatible condition ($t_{72} = 1.5$, $P = 0.15$), whereas a longer RT was observed for the obese group relative to the healthy weight group for the incompatible condition ($t_{72} = 2.9$, $P = 0.005$; Fig. 1*A*). Secondary analyses comparing the compatibility effect within each group revealed a longer RT for the incompatible relative to the compatible condition within each group ($t_{36} \geq 4.0$, $P < 0.001$). A Congruency main effect was also observed ($F_{1,72} = 57.0$, $P < 0.001$, $\eta_p^2 = 0.44$) with a longer RT for the incongruent (mean = 572.5 ms, standard error, SE = 13.7) relative to the congruent (mean = 548.1 ms, SE = 13.0) trials.

Table 2
Mean (SD) values for neuroelectric and behavioral measures

Measure	Healthy weight		Obese	
	Compatible	Incompatible	Compatible	Incompatible
Reaction time (ms)	505.2 (115.5)	553.2 (124.6)	546.2 (122.9)	635.7 (123.0)
Response accuracy (%)	77.7 (9.3)	76.9 (10.5)	75.2 (10.1)	74.4 (10.1)
Post-error reaction time (ms)	124.2 (82.3)	100.6 (90.9)	142.6 (88.0)	112.2 (128.3)
Post-error response accuracy (%)	78.1 (16.2)	70.0 (17.6)	74.0 (19.6)	62.0 (17.0)
ERN amplitude (μV)	−7.1 (5.0)	−3.4 (5.6)	−4.5 (4.9)	−3.4 (5.2)
ΔERN amplitude (μV)	−10.0 (7.1)	−6.6 (8.3)	−6.6 (4.9)	−5.7 (4.1)

Note: Flanker congruency is collapsed within each compatibility condition.

Response Accuracy

Analysis revealed a main effect for Congruency ($F_{1,72} = 47.6$, $P < 0.001$, $\eta_p^2 = 0.40$), which was qualified by a Compatibility × Congruency interaction ($F_{1,72} = 17.9$, $P < 0.001$, $\eta_p^2 = 0.20$). Post hoc analyses revealed lower response accuracy for the incongruent (mean = 73.3%, SE = 1.1) relative to the congruent (mean = 79.6%, SE = 1.3) trials for the compatible condition ($t_{73} = 7.4$, $P < 0.001$), whereas no congruency effect for the incompatible condition was observed (congruent: Mean = 76.5%, SE = 1.3; incongruent: Mean = 74.8%, SE = 1.2; $t_{73} = 2.3$, $P = 0.03$, i.e., after Bonferroni correction). A Group × Congruency interaction was also observed ($F_{1,72} = 4.2$, $P = 0.05$, $\eta_p^2 = 0.06$). However, post hoc analyses revealed no group differences for either congruency conditions ($t_{72} \leq 1.7$, $P \geq 0.10$). Planned comparisons revealed no group differences across compatibility conditions ($t_{72} \leq 1.1$, $P \geq 0.29$) or compatibility effects across groups ($t_{36} \leq 0.5$, $P \geq 0.64$).

Post-Trial Reaction Time

Preliminary Bonferroni-corrected *t*-tests were conducted within each compatibility condition across groups comparing post-error RT with post-matched-correct RT. Results indicated longer post-error RT relative to post-matched-correct RT across compatibility conditions (compatible: Mean difference = 72.5 ms, SE = 10.4; incompatible: Mean difference = 77.8 ms, SE = 10.9; $t_{73} \geq 7.0$, $P < 0.001$), verifying the expected post-error response slowing. A Group × Compatibility MANOVA revealed no main effects or interactions ($F_{1,72} \leq 3.3$, $P \geq 0.07$, $\eta_p^2 \leq 0.04$). The planned comparisons revealed no group differences across compatibility conditions ($t_{72} \leq 0.9$, $P \geq 0.36$) or compatibility effects across groups ($t_{36} \leq 1.4$, $P \geq 0.18$). Note that, as may be seen in Table 2, the standard deviation appears to be much larger for the obese group during the incompatible relative to the compatible condition and the healthy weight group. However, the results remained unchanged even with nonparametric statistics.

Post-Trial Response Accuracy

Preliminary Bonferroni-corrected *t*-tests were conducted within each compatibility condition across groups comparing post-error response accuracy with post-matched-correct response accuracy. Results indicated no accuracy effect for the compatible

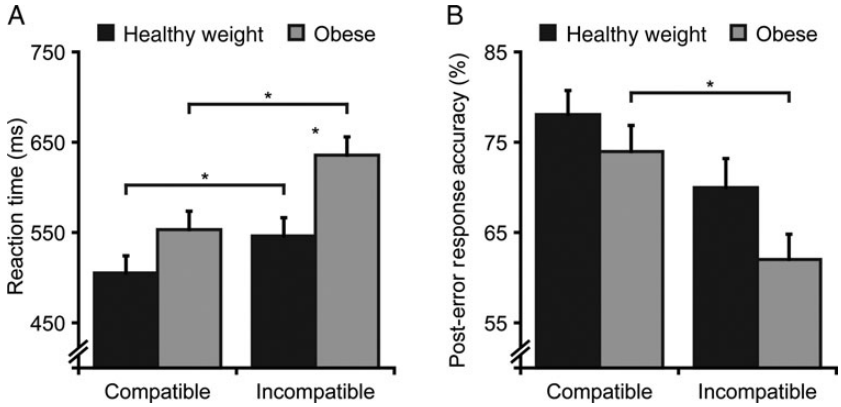

Figure 1. (*A*) Mean (SE) reaction time across congruency for each group and compatibility. (*B*) Mean (SE) post-error response accuracy for each group and compatibility condition.

condition (post-error: Mean = 76.0%, SE = 2.0; post-matched-correct: Mean = 73.6%, SE = 1.4; $t_{73} = 1.1$, $P = 0.29$), whereas lower post-error response accuracy (mean = 66.0%, SE = 2.2) was observed relative to post-matched-correct response accuracy (mean = 76.0%, SE = 1.4) for the incompatible condition ($t_{73} = 4.2$, $P < 0.001$). Given that we expected that post-error response accuracy would be higher than post-matched-correct response accuracy, an additional analysis was conducted using the mean distance of error runs (i.e., the number of sequential errors of commission) to seek the cause of the opposite effect. Analysis for the mean distance of error runs revealed a trend for a larger number of sequential errors of commission for the incompatible (mean = 2.1 trials, SE = 0.1) relative to the compatible (mean = 1.7 trials, SE = 0.1) condition ($t_{73} = 1.9$, $P = 0.06$). That is, the observed lower post-error response accuracy for the incompatible condition appears to be attributed to the larger number of sequential errors of commission. Thus, participants exhibited longer lapses in sustained attention during the incompatible condition, likely due to the increased cognitive control demands in this task condition.

A Group × Compatibility MANOVA revealed main effects for Group ($F_{1,72} = 4.1$, $P = 0.05$, $\eta_p^2 = 0.05$) with lower post-error response accuracy for the obese relative to the healthy weight group, and Compatibility ($F_{1,72} = 12.5$, $P = 0.001$, $\eta_p^2 = 0.15$) with lower post-error response accuracy for the incompatible relative to the compatible condition. Planned comparisons revealed no group differences across compatibility conditions ($t_{72} \leq 1.9$, $P \geq 0.07$). However, the planned comparisons revealed lower post-error response accuracy for the incompatible relative to the compatible condition for the obese group after Bonferroni correction ($t_{36} = 3.0$, $P = 0.005$; Fig. 1*B*). No such compatibility effect was observed for the healthy weight group ($t_{36} = 2.0$, $P = 0.05$).

Error-Related Negativity

Figure 2*A*,*B* illustrates grand averaged response-locked ERP waveforms for error and matched-correct trials, respectively, for each group and compatibility condition at the FCz electrode site. Preliminary Bonferroni-corrected *t*-tests were conducted within each compatibility condition across groups comparing ERN amplitude with CRN amplitude. Results indicated larger ERN amplitude relative to CRN amplitude across compatibility conditions (compatible: Mean difference = 3.6 μV, SE = 0.6; incompatible: Mean difference = 2.7 μV, SE = 0.8; $t_{73} \geq 3.4$, $P \leq 0.001$), verifying the expected accuracy effect. A Group × Compatibility MANOVA revealed a main effect for Compatibility ($F_{1,72} = 10.1$, $P = 0.002$, $\eta_p^2 = 0.12$) with smaller ERN amplitude for the incompatible relative to the compatible condition. Planned comparisons revealed smaller ERN amplitude for the obese group relative to the healthy weight group for the compatible condition ($t_{72} = 2.3$, $P = 0.02$), whereas no group difference was observed for the incompatible condition ($t_{72} = 0.0$, $P = 0.98$; Fig. 3*A*). Further, planned comparisons revealed smaller ERN amplitude for the incompatible relative to the compatible condition for the healthy weight group ($t_{36} = 4.4$, $P < 0.001$), whereas no compatibility effect was observed for the obese group ($t_{36} = 0.9$, $P = 0.39$; Fig. 3*A*).

Δ Error-Related Negativity

Figure 2*C* illustrates grand averaged error-matched-correct difference waveforms for each group and compatibility condition at the FCz electrode site. A Group × Compatibility MANOVA revealed a main effect for Compatibility ($F_{1,72} = 5.5$, $P = 0.02$, $\eta_p^2 = 0.07$) with smaller ΔERN amplitude for the incompatible relative to the compatible condition. A trend for a Group main effect was also observed ($F_{1,72} = 3.6$, $P = 0.06$, $\eta_p^2 = 0.05$) with smaller ΔERN amplitude for the obese relative to the healthy weight group. Planned comparisons revealed smaller ΔERN amplitude for the obese relative to the healthy weight group for the compatible condition ($t_{72} = 2.4$, $P = 0.02$), whereas no group difference was observed for the incompatible condition ($t_{72} = 0.6$, $P = 0.53$; Fig. 3*B*). Further, planned comparisons revealed a trend for smaller ERN amplitude for the incompatible relative to the compatible condition for the healthy weight group ($t_{36} = 2.2$, $P = 0.04$), whereas no compatibility effect was observed for the obese group ($t_{36} = 0.9$, $P = 0.36$; Fig. 3*B*).

Correlation Analysis

Table 3 provides the correlation coefficients between ΔERN amplitude and post-error task performance measures for each group and compatibility condition. For the healthy weight group, analyses revealed that ΔERN amplitude was positively correlated with post-error RT and negatively correlated with post-error response accuracy for the incompatible condition, but not for the compatible condition. For the obese group, no

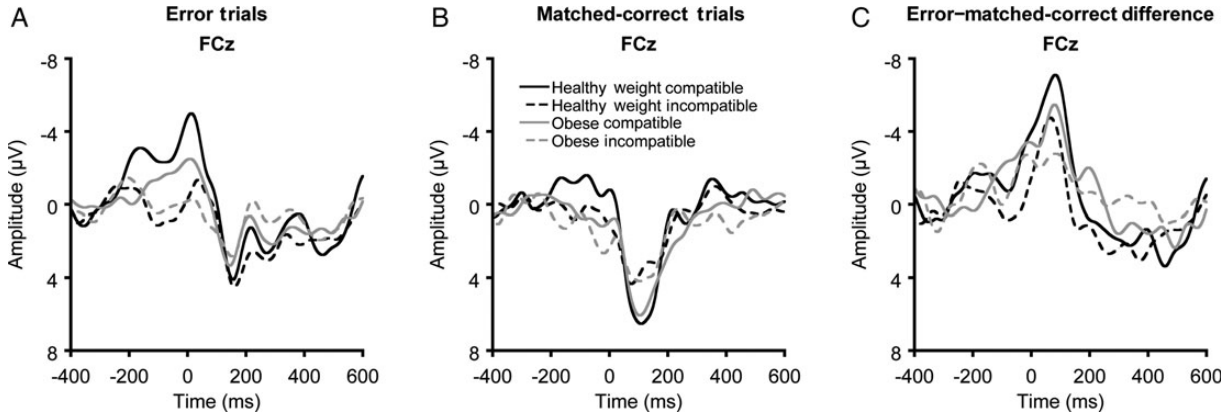

Figure 2. Grand averaged response-locked ERP waveforms for error trials (*A*) and matched-correct trials (*B*), and error-matched-correct difference waveforms (*C*) for each group and compatibility condition at the FCz electrode site.

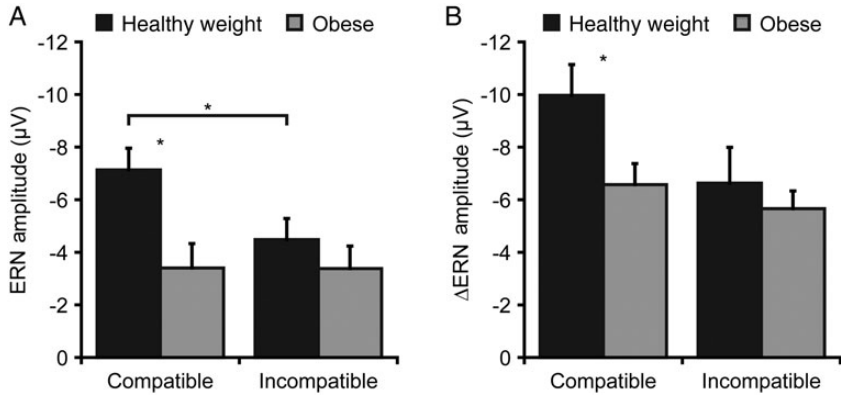

Figure 3. (*A*) Mean (SE) ERN amplitude for each group and compatibility condition. (*B*) Mean (SE) ΔERN amplitude for each group and compatibility condition.

Table 3
Correlation coefficients between ΔERN amplitude and post-error task performance measures

Measure	Healthy weight ΔERN amplitude		Obese ΔERN amplitude	
	Compatible	Incompatible	Compatible	Incompatible
Post-error reaction time	0.18	0.38*	−0.13	−0.02
Post-error response accuracy	−0.21	−0.60*	−0.26	−0.03

*$P \leq 0.05$.

such relationship was observed for either the compatible or the incompatible conditions.

Discussion

This study was conducted to provide the new insight into the negative association between childhood obesity and cognitive control using neuroelectric and behavioral measures of action monitoring. As hypothesized, obese children exhibited a longer RT relative to healthy weight children for the incompatible condition, whereas no such group difference was observed for the compatible condition. The incompatible condition requires the upregulation of cognitive control to inhibit and over-ride prepotent responses relative to the compatible condition (Friedman et al. 2009). Thus, the current RT findings suggest that healthy weight children can flexibly upregulate cognitive control to maintain task performance in the incompatible condition, whereas obese children demonstrated less ability to flexibly modulate cognitive control to meet the increased task demands.

Inconsistent with a priori predictions, planned comparisons indicated that obese children had smaller ERN amplitude relative to healthy weight children only for the compatible condition; an effect not found for the incompatible condition. Such a pattern of findings was due to the decreased ERN amplitude in healthy weight children from the compatible to the incompatible condition. Previous studies have shown a consistent developmental difference in ERN with younger children exhibiting smaller amplitude using the compatible condition of the flanker task (Davies et al. 2004; Santesso et al. 2006; Ladouceur et al. 2007), suggesting that smaller ERN amplitude may reflect protracted maturation of the ACC and/or the neural circuitry involving the ACC during childhood. Thus, it is likely that the smaller ERN amplitude observed in the obese group for the compatible condition may reflect relatively delayed development of the ACC. The decreased ERN amplitude for the incompatible condition for healthy weight children may support the dual mechanisms of control theory (Braver et al. 2007; Braver 2012), in which cognitive control operates along 2 qualitatively distinct strategies referred to as proactive control and reactive control. Proactive control is characterized by future-oriented early selection, which is associated with sustained lateral prefrontal cortex (PFC) activation prior to the imperative stimulus to actively maintain goal-relevant information (i.e., top-down processes). Alternatively, reactive control is characterized by past-oriented late correction, which is associated with transient activation of lateral PFC and a wider brain network including the ACC to reactivate task goals only as needed (i.e., bottom-up processes). That is, "proactive control relies upon the anticipation and prevention of interference before it occurs, whereas reactive control relies upon the detection and resolution of interference after its onset" (Braver 2012).

When most trials are incompatible within a block, participants should be biased toward adoption of a proactive control strategy to optimize task performance within the environment, which reduces conflict with increased sustained lateral PFC activation and decreased transient ACC activation (De Pisapia and Braver 2006). That is, the decrease in ACC activation may reflect a reduction in conflict or a lower threshold for the detection of conflict (i.e., more efficient conflict resolution) due to increases in top-down proactive control. Accordingly, it is speculated that the decreased ERN amplitude (i.e., decreased transient ACC activation) for the incompatible condition observed in healthy weight children might reflect a strategic shift from bottom-up reactive control to top-down proactive control, which would provide a more effective strategy for the demands of this condition. In fact, healthy weight children exhibited a smaller increase in RT from the compatible to the incompatible condition and maintained post-error response accuracy across compatibility conditions, providing support for this argument. Conversely, obese children might be unable to flexibly enact this strategy shift, as reflected by a lack of change in ERN amplitude between the compatibility conditions, and a larger increase in RT and decreased post-error response accuracy from the compatible to the incompatible condition. Developmental studies have suggested that the neural systems underlying proactive control exhibit prolonged development relative to reactive control and

continue to mature from childhood to young adulthood (Chatham et al. 2009; Andrews-Hanna et al. 2011). Accordingly, it is possible that childhood obesity may be inversely associated with development of such PFC–ACC interactions underlying top-down proactive control. However, considering the current flanker task included both congruent and incongruent trials, further investigation is necessary to support this argument through the manipulation of cognitive control strategy.

It should be noted that correlation analyses indicated that ERN amplitude (i.e., ACC activity on error trials) was only associated with post-error task performance for healthy weight children during the incompatible condition. The conflict monitoring theory (Botvinick et al. 2001; Carter and van Veen 2007) suggests that the ACC evaluates conflict and signals the PFC to upregulate cognitive control in support of the subsequent environmental interaction. Based on this theory, and coupled with the fact that post-error response accuracy was higher for the compatible relative to incompatible condition across groups, participants appeared to be able to correct their responses with relatively less activation in the network involving the PFC and ACC during the compatible condition. As such, ERN amplitude might not be related to post-error task performance in this condition across groups. By contrast, this cognitive control network might be more critical for the maintenance of task performance during all trials in the incompatible condition, due to the need for greater amounts of cognitive control, particularly among this age group. Based on this speculation, obese children might be unable to flexibly adjust their performance during the incompatible condition due to the decreased ability to modulate the cognitive control network, and consequently, no relationship between ERN amplitude and post-error task performance was observed. Although such an interpretation is speculative at this time; it is consistent with group differences in task performance, which were disproportionately greater for the incompatible condition.

In sum, childhood obesity was associated with a decreased ability to modulate the cognitive control network involving the PFC and ACC in order to optimize behavioral interactions within the environment. The present study replicates previous findings, indicating the negative association between childhood obesity and cognitive control (Cserjesi et al. 2007; Li et al. 2008; Lokken et al. 2009; Kamijo et al. forthcoming), and extends this area of research by using neuroelectric and behavioral measures of action monitoring. It has been well established that cognitive control is closely associated with academic achievement (Bull and Scerif 2001; Blair and Razza 2007; Latzman et al. 2010; Hillman et al. 2012). Further, a recent ERN study suggested that the ERN component may be a biomarker for academic achievement (Hirsh and Inzlicht 2010). Thus, this study supports previous findings, indicating the negative association between weight status and academic achievement (Datar and Sturm 2006; Castelli et al. 2007; Donnelly et al. 2009; Hollar, Lombardo, et al. 2010; Hollar, Messiah, et al. 2010; Roberts et al. 2010; Kamijo et al. forthcoming). Given the cross-sectional design used herein, the current findings do not necessarily imply that childhood obesity can negatively impact cognitive control. Recent studies have indicated that poorer cognitive control can be a risk factor for an increase in BMI, suggesting bidirectional associations between weight status and cognitive control in children (for a review, see Smith et al. 2011). Cognitive control can be improved by such factors as regular physical activity and by the implementation of cognitive control training (for a review, see Diamond and Lee 2011). Accordingly, we conclude that both maintaining a healthy weight and improving cognitive control may be essential for cognitive and brain health, and effective functioning throughout development.

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Notes

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