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ORIGINAL ARTICLE

Inhibitory control in otherwise healthy overweight 10-year-old children

S Reyes[1], P Peirano[1], P Peigneux[2], B Lozoff[3] and C Algarin[1]

BACKGROUND: Preventing obesity is a worldwide public health priority. In vulnerable children living in obesogenic environments, with easy access to high-caloric food, alterations in inhibitory control functions might favor excessive food intake and affect energy regulation. We hypothesized that overweight/obese children would present lower inhibitory control in comparison to normal weight children.

METHODS: We measured inhibitory control functions in 93 otherwise healthy overweight/obese and 92 normal weight 10-year-old children using the Stroop test and the Go/No-Go task. Event-related potentials were recorded during the Go/No-Go task.

RESULTS: Overweight/obese children showed slower reaction times (1248.6 ms (95% confidence interval (CI): 1182.9–1314.3) vs 1149.0 ms (95% CI: 1083.0–1215.1)) on the Stroop test, higher reaction time variability (0.25 (95% CI: 0.22–0.27) vs 0.21 (95% CI: 0.19–0.24)) on the Go/No-Go task and decreased P300 amplitude (4.1 μV (95% CI: 3.0–5.2) vs 6.4 μV (95% CI: 5.2–7.6)) on event-related potentials compared with normal weight children.

CONCLUSIONS: Our results indicate altered inhibitory control functions in otherwise healthy overweight/obese children, which might contribute to their excessive food consumption.

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INTRODUCTION

The impact of childhood obesity on non-communicable diseases makes its prevention an international public health priority.[1] An increasing proportion of the world's population is becoming obese, which is related to higher risks of morbidity and mortality. Childhood obesity has risen to significant levels globally and continues to increase in prevalence and severity.[2] Although it is clear that obesity is driven by multidimensional factors,[3] recent evidence indicates a relationship between obesity and neural circuits related to inhibitory control functions.[4]

Inhibitory control refers to the capacity to suppress a prepotent or dominant response that can be triggered by an external cue, stop an ongoing response or resist distracting stimuli.[5] Inhibitory processes are crucial for suppressing inappropriate/unwanted actions that can interfere with the completion of motor, cognitive or socio-emotional goals. They are critical for everyday life, allowing people to successfully complete actions, such as stopping at traffic lights, preventing impulsive behavior or resisting the temptation to eat.[6] Evidence suggests that inhibitory control processes are supported and regulated by multiple top–down neural connections[7] that may be compromised in obese individuals.[8]

In this respect, it has been shown that obese children exhibit deficits in inhibitory control,[9] attention[10] and impulsivity.[11] These neurocognitive skills are part of the executive functions system, which can be defined as the set of abilities that organize and direct cognitive and emotional responses to provide regulatory control over thought and behaviors, recognize errors and engage in planning.[12] Executive functions and their neural substrates are already present in infancy but continue to mature into adulthood.[13] These functions appear to be orchestrated by the prefrontal cortex, with dopamine being one of the main neurotransmitters.[14]

Easy access to large quantities of appealing food of poor nutritional quality requires the frequent need to inhibit eating in order to maintain normal weight (NW). Individual differences in inhibitory control could thus modulate the risk for overeating in environments with wide availability of high-caloric food.[15]

The mechanisms underlying the relationship between inhibitory control and obesity remain poorly understood. Obese human adults evaluated with positron emission tomography exhibited lower dopamine D2 receptor density in the striatum in comparison with NW controls. A reduction in D2 receptor availability was associated with higher metabolic activity in prefrontal regions, suggesting a mechanism that might contribute to overeating. This mechanism would be subtended by prefrontal pathways involved in inhibitory control.[16]

Studies on the association between inhibitory control and overweight/obesity (OW) in children have used various behavioral measures. Graziano *et al.*[9] found that measures of inhibition, emotion regulation and reward sensitivity at 2 years of age, through tasks of frustration and delay gratification in 57 toddlers, predicted variations in body mass index (BMI) and obesity at 5 years of age. On the basis of parent-report data ($n = 346$) for 6- to 13-year-olds, Van den Berg *et al.*[11] found that impulsivity predicted higher overeating scores, and overeating was significantly associated with BMI. Using the Go/No-Go and incompatibility tasks of the Attention Assessment Battery ($n = 177$) in 8- to 15-year-old children, Pauli-Pott *et al.*[17] reported a link between high impulsivity and higher BMI in younger participants.

[1]Sleep and Functional Neurobiology Laboratory, Institute of Nutrition and Food Technology (INTA), University of Chile, Santiago, Chile; [2]UR2NF–Neuropsychology and Functional Neuroimaging Research Unit at CRCN–Centre de Recherches en Cognition et Neurosciences and UNI, Neurosciences Institute, Université Libre de Bruxelles (ULB), Belgium and [3]Center for Human Growth and Development and Department of Pediatrics and Communicable Diseases, University of Michigan, Ann Arbor, MI, USA. Correspondence: Professor C Algarin, Sleep and Functional Neurobiology Laboratory, INTA, University of Chile, Macul 5540, Macul, PO Box 138-11, Santiago, Region Metropolitana, Chile.
E-mail: calgarin@inta.uchile.cl
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Cserjési *et al.*[10] compared 12 obese with 12 NW 12-year-old children and found that performances on measures of sustained attention and perseveration indexes, as measured by the Wisconsin Card Sorting test, were lower in the obese group. In a cohort of 1000 subjects followed from birth to 32 years, Moffitt *et al.*[18] showed that self-control measures in childhood (observational ratings of children's lack of control and parent, teacher and self-reports of impulsive aggression, hyperactivity, lack of persistence, inattention and impulsivity) predicted adult health problems, including OW, even after controlling for initial socioeconomic status and intelligence quotient. We identified only one study that used electrophysiological measures of cognitive inhibitory control. Tascilar *et al.*[19] investigated electroencephalographic activity (latency and amplitude of P300 wave) using an auditory discriminated task paradigm ($n = 73$) in 10-year-old children and found reduced amplitude in the obese group compared with the NW group. Furthermore, obese children with insulin resistance had lower P300 amplitude than obese children without insulin resistance.[19]

We hypothesized that a lower ability to detect conflicts and a less efficient use of neural resources could underlie OW children's poorer performance on cognitive and motor inhibition tasks. We used behavioral and electrophysiological methods to assess aspects of inhibitory control in OW and NW children.

MATERIALS AND METHODS

Subjects

Children in the present study were participants in an ongoing longitudinal study in Chile about the long-term effects of iron-deficiency anemia (IDA) in infancy. As a subsample of those assessed at 10 years, there were 185 participants who performed the Stroop test and 132 who performed the Go/No-Go task. We included children who did or did not have IDA as infants.

Detailed descriptions of the population, design of the infancy study[20] and findings during infancy and childhood have been published elsewhere.[21–30] In brief, study participants were healthy full-term infants, with birth weight $\geqslant 3.0$ kg, living in the same geographical area and without perinatal complications or acute or chronic illnesses. For each infant with IDA identified at 6, 12 or 18 months, an infant of the same age who was clearly non-anemic (venous hemoglobin $\geqslant 115\ g\ l^{-1}$) was randomly selected and included in the control group. All participants were given oral iron for at least 3 months, and none had IDA on follow-ups in infancy or childhood.[29,30] Information on family background included parental education and occupation as well as indicators of socioeconomic status among others.[20]

The original and follow-up protocols were approved and reviewed annually by the Institutional Review Boards of the University of Michigan, Ann Arbor and the Institute of Nutrition and Food Technology (INTA), University of Chile. Parents provided signed informed consent and children signed assent.

Experimental tasks

We assessed cognitive inhibitory functions in NW and OW children using the Stroop and Go/No-Go tasks.

Stroop test. The Stroop test has been extensively used to evaluate cognitive inhibition.[31] Stimuli were presented on a computer monitor. Participants responded using the computer keyboard. The targets were the words 'red,' 'blue' and 'green' or a string of 'X's' displayed in red, blue or green color. Color words were stimuli in the incongruent trials (for example, the word 'red' displayed in blue), and strings of 'X's' were used in the control trials. Half of the items consisted of incongruent trials, and the other half were control trials. Each item was displayed in one of the three colors an equal number of times. Three keys from the computer keyboard were covered with a color patch, and participants were told to press the key that corresponded to the color of the target as soon as it appeared on the screen. Participants had a practice block of 12 trials, followed by an experimental block of 60 trials. Each trial began with a fixation point ('+' sign) presented for 500 ms in the center of the screen, after which the stimulus appeared on the monitor until subject's response. Reaction time (RT) for correct responses, error rate for control and incongruent trials and an interference index (that is, the RT difference between incongruent and control trials) were processed. In addition, the effect of the preceding trial's type on cognitive inhibitory processes[32] was assessed by comparing RTs for incongruent trials that followed one or two successive control trials (control–incongruent, control–control–incongruent: estimate of conflict adaptation) and RTs for control trials that followed one or two successive incongruent trials (incongruent–control, incongruent–incongruent–control: estimate of conflict inhibition).[33]

Go/No-Go task. The Go/No-Go task has been widely used to assess the inhibition capacity of a motor response when there is a previously learned or prepotent response.[34] We recorded event-related potentials (ERPs) during the Go/No-Go task to document obesity-related changes in the neural processes involved in motor response inhibition. More specifically, we focused on N200 and P300 components. N200 is an early negative deflection associated with inhibitory neural processes and attention.[29] The following positive P300 wave is associated with attentional assignment[35] and context renewing.[36] Its amplitude is considered to index the relevance of stimuli.[37]

Children were seated in front of a computer and asked to press a button as fast as possible when a letter stimulus (Go trial) was displayed on the screen. When an 'X' appeared (No-Go trial) they were told not to press the button. Each trial consisted of a single white upper-case letter presented in the center of a black computer screen. There were two blocks of trials. Block 1 consisted of 40 trials containing 100% Go trials. Block 2 was comprised of 40 Go trials and 40 No-Go trials that were intermixed randomly. Each trial lasted 1600 ms with 100 ms pre-stimulus presentation of a white '+' sign, 500 ms of stimulus presentation, and 1000 ms post-stimulus recording for the ERP. The interstimulus interval randomly varied from 1500 to 1700 ms. Behavioral measures were error rate and RT (mean and variability). RT variability was computed through s.d. and coefficient of variability (s.d. RT/mean RT).[38] Variation in RT is another relevant measure of performance, as higher values of variability are correlated with motor disinhibition.[38,39] Data were analyzed according to the three trial types: (a) block 1 Go trials in homogenous Go condition (Go simple), (b) block 2 Go trials in mixed-type trials condition (Go mixed) and (c) block 2 No-Go trials in mixed-type trials condition (No-Go mixed).[34]

ERPs were recorded from 32 scalp electrodes (Electro-Cap International, Eaton, OH, USA) using a modified 10–20 system. Scalp electrodes were referenced to Cz (central scalp electrode) during acquisition. To detect eye blinks, bipolar vertical electro-oculography activity was recorded from electrodes placed vertically above and below the right eye. Electroencephalographic and electro-oculographic activities were obtained using a Neurodata Acquisition System (Grass Instruments, West Warwick, RI, USA), amplified with Model 15 amplifiers with a gain of 50 000 for electroencephalographic channels and 5000 for electro-oculographic channels. The amplifier filter settings were 0.1–30.0 Hz with a 50.0 Hz notch filter. All channels were digitized at 200.0 Hz onto a hard drive with a 12-bit A/D converter (National Instruments, Austin, TX, USA).

For the ERP analysis, data were processed offline using the ERP32 analysis software (New Boundary Technologies, Minneapolis, MN, USA). Participant data were included if there were at least 10 artifact-free trials for each trial type (Go simple, Go mixed and No-Go mixed).

Electrode groupings and time windows were selected on the basis of earlier reports of these components and through visual inspection. Components of interest (N200 and P300 waves) were identified relative to the number of major positive and negative peaks following stimulus onset. Electrophysiological measures were peak amplitude and latency for N200 and amplitude for P300. All latency values were based on stimulus onset, and peak amplitudes were calculated relative to the baseline amplitude.

Anthropometric measures

Trained personnel obtained anthropometric measurements using standardized protocols (without shoes, wearing underwear and in the Frankfurt position) on the same machine calibrated every day. Weight to the closest 0.1 kg and height to the closest 0.1 cm were measured using a SECA scale (model 700, Seca, Hamburg, Germany). BMI was calculated for each participant as the ratio of weight in kilograms divided by the square of height in meters. Using age-specific BMI *z*-scores,[40] participants were classified as NW (BMI *z*-score $\geqslant -2$ to < 1) or OW (BMI *z*-score $\geqslant 1$).

Data analysis

Repeated measures analyses of variance (ANOVA) were conducted using group (NW and OW) as a between-subject factor and inhibitory control parameters as a within-subject factor. For the Stroop test, separate ANOVAs were conducted on RTs and accuracy performance using the within-subject factors of trial type (two levels: control vs incongruent trials) or preceding trial types (two levels: conflict adaptation vs conflict inhibition sequences of trials). For the Go/No-Go task, separate ANOVAs were conducted on RTs and accuracy performance using the within-subject factor of trial type (three levels: Go simple vs Go mixed vs No-Go mixed). For electrophysiological data, electrode was also examined as a within-subject factor. *Post hoc* paired *t*-tests were conducted using the Bonferroni correction for multiple comparisons. The models also included the following covariates: sex and IDA status in infancy and birth weight. Statistical analyses were conducted with SPSS software version 19.0 (SPSS Inc., Chicago, IL, USA). All significance tests were two-tailed; a *P*-value $\leqslant 0.05$ was considered statistically significant.

RESULTS

Background characteristics are shown in Table 1. The groups were similar in the proportion of children with IDA or attention-deficit/hyperactivity disorder (ADHD). The OW group had higher birth weight. By design, anthropometric measurements at 10 years of age were different between groups.

Behavioral outcomes

Stroop test. There was a significant effect of trial type on RT ($F(1,180) = 25.7$, $P < 0.001$), with shorter RT for control trials than incongruent trials (1125.4 vs 1196.3 ms, $P < 0.001$). There was also a significant main effect of group for RT ($F(1,180) = 5.5$, $P < 0.05$) but not for error rate ($P = 0.56$), with longer RT for incongruent and control trials in the OW group than the NW group (Table 2). The interaction between trial type and group was not significant for RT or error rate (all $P > 0.73$).

There was a significant interaction between preceding trial type and group for RT ($F(1,153) = 6.0$, $P < 0.05$), with longer RT for conflict adaptation sequences in the OW group than the NW group (Table 2). *Post hoc* analyses showed longer RT in conflict adaptation compared with conflict inhibition in the OW group, whereas RT values were similar in the NW group (Table 2). These results were significant for RTs of stimuli followed by two successive trials (for example, control–control–incongruent). No covariates were significant in the final model.

Go/No-Go task. For behavioral results in the Go/No-Go task, the expected main effects of trial type were observed for RT ($F(1,97) = 5.6$, $P < 0.05$), s.d. ($F(1,97) = 12.1$, $P < 0.01$) and error rate ($F(1,97) = 3.8$, $P < 0.01$). There were shorter RTs for Go simple than Go mixed trials (506.9 vs 550.7 ms, $P < 0.001$), with lower s.d. for Go simple than Go mixed trials (119.0 vs 139.1 ms, $P < 0.01$) and higher error rate for No-Go mixed than Go simple (9.3% vs 1.8%, $P < 0.001$) and Go mixed (9.3% vs 1.7%, $P < 0.001$) trials. There were significant main effects of group for s.d. ($F(1,97) = 5.9$, $P < 0.05$) and coefficient of variability ($F(1,97) = 4.9$, $P < 0.05$), with higher values for Go simple trials in OW than in NW children (Table 2). Group by trial type interactions were not significant (all $P > 0.32$).

ERP outcomes

N200 wave was analyzed in the frontal region and had the greatest amplitude at the midline frontal electrode (Fz). There were main effects of trial type for amplitude ($F(2,123) = 17.5$, $P < 0.001$) and latency ($F(2,123) = 21.0$, $P < 0.001$). Amplitude was higher for Go mixed than Go simple (−10.8 vs −8.8 μV, $P < 0.001$) and No-Go mixed (−10.8 vs −7.96 μV, $P < 0.001$) trials. Latency was shorter for Go mixed than Go simple (354.4 vs 378.7 ms, $P < 0.001$) and No-Go mixed (354.4 vs 376.5 ms, $P < 0.001$) trials. There were no significant interactions between trial type and group for amplitude ($P = 0.572$) or latency ($P = 0.502$). Group differences for amplitude ($P = 0.655$) and latency ($P = 0.436$) were also not significant (Table 3). No covariates were significant in the final model.

P300 wave was analyzed at Fz, Cz and Pz leads and had the greatest amplitude at the midline parietal region. There was a main effect of trial type for peak amplitude ($F(2,111) = 33.0$, $P < 0.001$). Amplitude was higher in No-Go mixed than Go simple (7.1 vs 3.1 μV, $P < 0.001$) and Go mixed trials (7.1 vs 5.6 μV, $P < 0.01$). P300 amplitude was lower in the OW group compared with the NW group ($F(1,112) = 7.7$, $P < 0.01$; Figure 1). P300 amplitude was lower for Go simple and Go mixed trials in OW relative to NW children (Figure 2). The interaction between trial type and group was also significant ($F(2,111) = 4.3$, $P < 0.01$), with

Table 1. Characteristics of normal weight and overweight/obese children groups

	NW (n = *92*)	*OW* (n = *93*)	t-*test*	P-*value*
Female (%)[a]	42 (45.6)	41 (44.0)		0.83
Birth weight (g)	3474.4 ± 384.9	3617.0 ± 395.3	−2.4	0.014
Birth length (cm)	50.6 ± 1.9	50.9 ± 1.7	−1.1	0.265
Gestational age (week)	39.5 ± 1.0	39.2 ± 1.0	1.5	0.124
Age (years)	10.3 ± 0.1	10.2 ± 1.0	1.1	0.245
Body weight (kg)	32.8 ± 4.6	45.4 ± 7.3	−13.9	< 0.001
Body weight (*z*-score)	0.2 ± 0.1	2.3 ± 0.2	−83.8	< 0.001
Body height (cm)	137.6 ± 5.5	142.0 ± 5.6	−5.4	< 0.001
Body height (*z*-score)	0.4 ± 0.2	0.1 ± 0.6	4.2	< 0.001
BMI (kg m^{-2})	17.1 ± 1.3	22.5 ± 2.7	−17.3	< 0.001
BMI *z*-score	0.1 ± 0.5	1.9 ± 0.6	−19.8	< 0.001
Hemoglobin at 10 years (g l^{-1})	132.1 ± 7.2	132.7 ± 7.9	−0.41	0.678
IDA in infancy (%)[a]	53.3	49.5		0.605
Low SES (%)[a]	15.3	25.3		0.127
ADHD at 10 years (%)[a]	10.5	18.9		0.115
Mother education (years)	8.9 ± 2.8	9.5 ± 3.1		0.253
Mother self-reported prepregnancy weight (kg)	52.8 ± 9.2	55.6 ± 10.2	−1.7	0.078

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; BMI, body mass index; IDA, iron-deficiency anemia; NW, normal weight; OW, overweight/obese; SES, socioeconomic status. [a]X^2-test. Values are expressed as means ± s.d. SES: score < 27;[60] ADHD: score > 16 for males and > 12 for females.[61] Independent sample *t*-test.

Table 2. Stroop test and Go/No-Go task results in normal weight and overweight/obese children groups

	NW	*OW*
Stroop test		
RT control trials (ms)	1084.1 (1032.6–1135.5)*	1174.1 (1123.0–1225.3)
RT incongruent trials (ms)	1149.0 (1083.0–1215.1)*	1248.6 (1182.9–1314.3)
RT conflict adaptation (ms)	1082.3 (989.6–1175.0)*	1211.4 (1118.1–1304.7)[a]
RT conflict inhibition (ms)	1117.9 (1046.4–1189.3)	1079.5 (1007.5–1151.4)
Error rate control trials (%)	10.5 (6.7–14.2)	11.9 (8.2–15.6)
Error rate incongruent trials (%)	11.1 (7.2–14.9)	12.7 (8.9–16.6)
Error rate conflict adaptation (%)	27.9 (20.5–35.2)	21.4 (14.4–28.3)
Error rate conflict inhibition (%)	21.4 (14.4–28.3)	22.2 (15.3–29.1)
Interference (ms)	68.8 (43.4–94.1)	73.0 (41.9–104.0)
Go/No-Go task		
RT Go simple (ms)	492.9 (457.8–528.0)	520.9 (489.1–552.7)
RT Go mixed (ms)	538.2 (511.6–564.7)	563.2 (539.1–587.2)
s.d. Go simple (ms)	107.2 (93.4–120.9)*	130.8 (118.4–143.2)
s.d. Go mixed (ms)	131.9 (117.2–146.7)	146.3 (132.9–159.6)
Coefficient variability Go simple	0.21 (0.19–0.24)*	0.25 (0.22–0.27)
Coefficient variability Go mixed	0.24 (0.22–0.26)	0.25 (0.24–0.27)
Error rate Go simple (%)	1.7 (0.9–2.5)	1.9 (1.2–2.6)
Error rate Go mixed (%)	1.9 (1.0–2.9)	1.5 (0.7–2.4)
Error rate No-Go mixed (%)	10.0 (7.3–12.8)	8.6 (6.1–11.1)

Abbreviations: NW, normal weight; OW, overweight/obese; RT, reaction time. ANOVA repeated measures values are expressed as means and confidence interval. *$P < 0.05$, difference between groups. [a]$P < 0.01$, different compared with RT conflict inhibition.

Table 3. ERP results for Go/No-Go task in normal weight and overweight/obese children groups

	NW (n = *63*)	*OW* (n = *69*)
N200		
Go simple amplitude (μV)	−8.8 (−10.4 to −7.3)	−8.9 (−10.3 to −7.4)
Go mixed amplitude (μV)	−11.1 (−12.7 to −9.5)	−10.4 (−12.0 to −8.9)
No-Go mixed amplitude (μV)	−8.2 (−9.9 to −6.6)	−7.6 (−9.2 to −6.0)
Go simple latency (ms)	374.4 (361.6–387.1)	383.0 (370.8–395.2)
Go mixed latency (ms)	351.7 (342.7–360.6)	357.1 (348.5–365.7)
No-Go mixed latency (ms)	376.1 (364.0 to −388.1)	376.9 (365.4–388.3)

Abbreviations: NW, normal weight; OW, overweight/obese. ANOVA repeated measures. Values are expressed as means and confidence intervals.

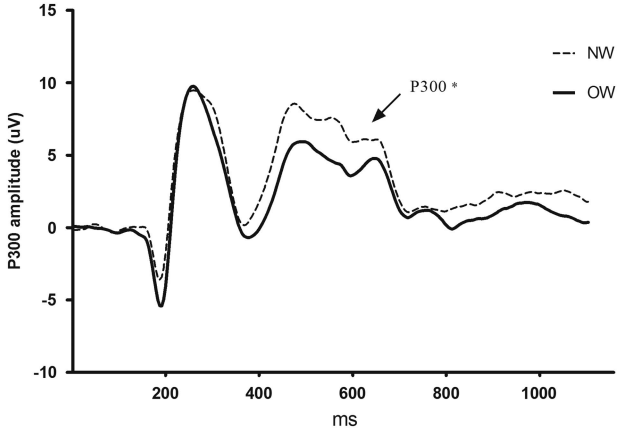

Figure 1. Grand average for the mean amplitude of P300 wave for Go/No-Go task at ERP midline electrodes in NW and OW children groups. *$P < 0.05$ indicates the statistically significant group difference.

the OW group showing lower amplitude in Go mixed compared with No-Go mixed trials, whereas the NW group exhibited similar amplitudes. For both groups, the amplitude in Go simple was lower than in No-Go mixed trials (Figure 2). No covariates were significant in the final model.

DISCUSSION

We showed here that OW children had longer RTs in the Stroop test and higher RT variability and lower P300 amplitude in the Go/No-Go task. These results indicate a reduced capacity in cognitive and motor inhibition compared with NW children. Furthermore, the electrophysiological result of lower P300 amplitude suggests greater difficulties in allocating cognitive resources in OW children when the tasks are more complex. These results are consistent with the hypothesis that differences in inhibitory control and the capacity to efficiently recruit cognitive resources modulate the risk for overeating and obesity in environments with easy access to high-caloric food.[15]

Between-group differences in RTs on the Stroop test are indicative of altered cognitive inhibitory control.[41] Simultaneous and incompatible representations give rise to cognitive conflict,[42] and successful adaptation is the result of abilities to detect the conflict and to do the necessary cognitive adjustment as a function of trial sequence.[42,43] Here we provide evidence that impaired inhibitory control functions are associated with OW in childhood. In the Go/No-Go task, we found increased RT variability in OW children. Although available evidence is limited,[41] a reduction in RT variability is considered an indicator of response efficiency for successful Go as well as No-Go trials.[39] It is thought that individuals with higher RT variability less efficiently use premotor brain regions needed to guide response selection, and consequently, must involve higher-order prefrontal brain regions to produce appropriate motor responses.[6,39] This suggests that OW children would need to recruit higher-order brain circuits in order to select appropriate responses.

Considering electrophysiological responses, lower amplitude of P300 wave in OW children was driven mainly by Go mixed trials. The expected response to stimuli requiring greater inhibitory

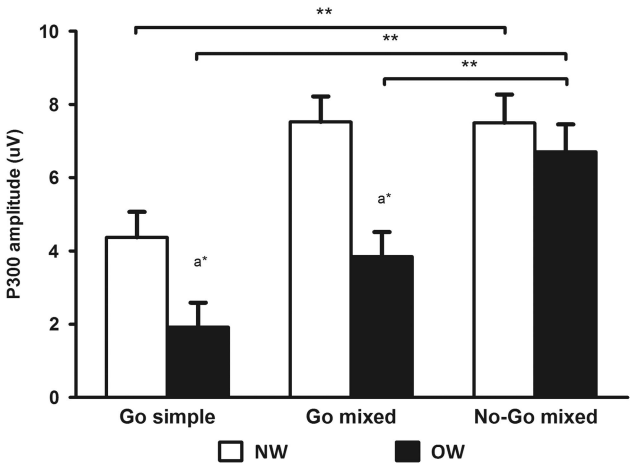

Figure 2. Amplitude of P300 wave of the Go/No-Go task at ERP midline electrodes for three trial types in NW and OW children. Each bar represents the amplitude mean and standard error. Horizontal lines indicate a significant difference within group. Letter 'a' shows a significant difference between groups. Statistical differences were determined by repeated measures ANOVA. $*P < 0.05$; $**P < 0.01$.

control is an increased amplitude, reflecting the relevance of stimuli[34] and greater activation of the neural circuits involved in these processes.[44] There is evidence that decreased P300 amplitude is a characteristic of psychiatric and drug abuse disorders,[45] including alcoholism,[46] schizophrenia,[47] depression[48] and ADHD.[49] This electrophysiological pattern was interpreted as being a greater difficulty in processing stimuli and an inability to allocate resources when the demands are higher.[50] Furthermore, lower P300 amplitude during response execution and inhibition may indicate deficient cognitive processing mechanisms and the activation of different/inappropriate brain circuits.[46] It may also represent an endophenotype for neurocognitive dysfunctions in relation to a variety of complex behavioral disorders.[51] Smaller P300 amplitude has been found in children of one or both alcoholic parents, a condition in which dopaminergic genetic determinants are key players.[52] In line with reports in healthy children,[34,53] the NW group showed similar amplitudes for Go mixed and No-Go mixed trials, but the OW group did not. The pattern in OW children may represent lower efficiency to allocate neural resources to process stimuli type and control the motor response. Hence, inhibitory control impairment might precede the tendency for overeating behavior, which would then be followed by weight gain.

The neurophysiological mechanisms underlying the relationships between inhibitory control and BMI deserve more attention. As compared with lean subjects, obese adolescents are characterized by reduced gray matter volume in the orbitofrontal cortex, a cortical area involved in regulating inhibition of inappropriate responses.[54] Higher BMI is associated with lower metabolic activity in prefrontal and cingulate regions, which participate in regulating impulse control.[8] Obese individuals exhibit a decreased availability of striatal D2 receptor, which may promote overeating via modulation of striatal prefrontal pathways related to inhibitory control.[16] Greater BMI was associated with decreased cerebral blood flow in the prefrontal cortex in otherwise healthy adults.[55] In line with this evidence, P300 wave is associated with activity of the dopaminergic pathways and dopaminergic genetic influences.[52] We did not find an association between OW and N200 in the Go/No-Go task, suggesting similar early conflict detection processes in OW and NW children. However, the difference in P300 amplitude suggests greater difficulties in processing cognitive demands, which is supported by the behavioral results of greater variability of RT in OW group.[41] Previous studies showing no differences in inhibitory control did not use electrophysiological measures.[4,56]

There are inherent limitations with the present study. The cross-sectional nature of the study precluded inferences regarding causality. We relied on BMI to establish OW status, but measures like body composition or biochemical indices might classify individuals more accurately. The absence of time pressure to respond in cognitive tasks might have influenced the performance of subjects.[57] Additional response modality (oral, for instance) or trial types for the Stroop test could help explore further interference and/or facilitation effects.[31] Finally, IDA in infancy appears to affect inhibitory control at the age of 10 years.[29] Nonetheless, we included children with IDA in infancy in both groups (OW and NW). Also, a link between ADHD in children and obesity in adults has been described.[58] The neurobiological mechanism that could explain this relationship is the dysfunction of fronto-striatal dopaminergic pathways involved in both conditions, which regulate executive functions.[59] However, the proportion of IDA or ADHD subjects were not different between the groups.

Our findings suggest that OW children have reduced inhibition capacity and more difficulties allocating the appropriate resources to solve cognitive conflicts than NW children. We propose here that reduced inhibitory control exposes children to weight gain. Prospective studies are needed to determine if such an alteration in these cognitive aspects and/or other executive functions precede and predict overeating, overweight and the progression to obesity.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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