Received: 22 April 2020 | Revised: 22 June 2020 | Accepted: 14 July 2020
DOI: 10.1111/ijpo.12708

ORIGINAL RESEARCH

Pediatric OBESITY WILEY

The differential relationship of an afterschool physical activity intervention on brain function and cognition in children with obesity and their normal weight peers

Nicole E. Logan[1] | Lauren B. Raine[1] | Eric S. Drollette[2] | Darla M. Castelli[3] | Naiman A. Khan[4] | Arthur F. Kramer[1,5] | Charles H. Hillman[1,6]

[1]Northeastern University, Department of Psychology, Boston, MA
[2]University of North Carolina at Greensboro, Department of Kinesiology, Greensboro, North Carolina
[3]University of Texas at Austin, Department of Kinesiology and Health Education, Austin, Texas
[4]University of Illinois at Urbana-Champaign, Department of Kinesiology and Community Health, Urbana, Illinois
[5]Beckman Institute, University of Illinois at Urbana-Champaign, Urbana, Illinois
[6]Department of Physical Therapy, Movement, and Rehabilitation Sciences, Boston, MA

Correspondence
Nicole E. Logan Department of Psychology, Northeastern University, 635 ISEC
360 Huntington Ave, Boston, MA 02115.
Email: logan.n@northeastern.edu

Summary

Background: Physical activity (PA) is beneficial for cognitive and brain health during preadolescence. Given that childhood obesity (OB) is a public health concern, investigating this effect in children with OB is an important societal consideration.

Objectives: To identify the effects of weight status and PA on neuroelectric indices of executive function in preadolescence.

Methods: Children were randomly assigned to a PA intervention or a wait-list control group and completed a task that manipulated inhibitory control, while task performance and neuroelectric (P3 component) outcomes were assessed. About 103 children with OB were matched to a sample of 103 normal weight (NW) children based on treatment allocation and demographic variables.

Results: Children with OB in the control group demonstrated reduced P3 amplitude from pre- to post-test, meanwhile those with OB in the PA intervention maintained P3 amplitude at post-test compared to pre-test. Additionally, NW children in the PA intervention group showed that decreased visceral adipose tissue corresponded with faster task performance, a relationship not observed in children with OB.

Conclusions: These results suggest that a 9-month PA intervention may be particularly beneficial to the cognitive and brain health of children with OB. These results are important to consider given the public health concerns associated with childhood OB.

KEYWORDS
brain function, cognitive function, obesity, physical activity

1 | INTRODUCTION

Elevated weight status during childhood is an epidemic, with over 340 million children and adolescents aged 5 to 19 characterized as overweight or obese in 2016.[1] Childhood obesity (OB) has been linked to many health complications, including metabolic and cardiovascular dysfunction.[2] Recently, evidence has emerged suggesting that excess adiposity may also selectively influence cognitive and brain function in preadolescent children, particularly in those children with OB.[3,4]

1.1 | Adiposity and cognition

Adiposity is characterized by specific distributions of fat mass around the body; subcutaneous abdominal adipose tissue (SAAT) is the fat present directly under the skin in the abdominal area,[5] while visceral adipose tissue (VAT) is stored within the abdominal cavity, including around vital organs such as the liver, pancreas, and intestines.[6-8] Excess VAT is related to a higher risk of metabolic diseases and negative health outcomes as it produces inflammatory cytokines and hormones.[9] Additionally, total abdominal adipose tissue (TAAT) encompasses both SAAT and VAT.[10] Cross-sectional data have indicated that TAAT is a significant negative predictor of relational memory accuracy among preadolescent children with overweight and OB (7-9 years old).[10] Likewise, higher VAT values in children with OB have been associated with poorer intellectual and cognitive abilities, while normal weight (NW) children demonstrated a positive relationship between VAT and intellectual and cognitive abilities,[4] indicating that excess abdominal fat mass may negatively influence cognition. Furthermore, children with a higher body mass index (BMI) performed worse on reading and math achievement tests[4] as well as on measurements of inhibitory control.[3]

1.2 | Adiposity and brain function

Children with OB also demonstrate differential expression of the P3 event-related brain potential (ERP) when compared to NW children.[3] The P3 ERP is the positive peak in the time-locked EEG signal that occurs between 300 and700 ms after a stimulus is presented. The P3 is commonly used to assess brain activity associated with cognitive function. It is characterized by its amplitude and latency of stimuli in the environment, representing a neuroelectric index of attentional resource allocation (as defined by its amplitude) and processing speed (as defined by its latency), respectively.[11] Prior research using a Go/NoGo task to modulate response inhibition requirements[3] suggested that children with OB demonstrated similarity in the topographical distribution of P3 amplitude across the scalp regardless of inhibitory control requirement (ie, across task conditions modulating response inhibition demands). This finding stood in contrast to children with NW, who demonstrated the expected P3 scalp topography, with a more frontal distribution of the NoGo P3 (ie, greater response inhibition demands), relative to the Go P3 (ie, lesser response inhibition demands). Interestingly, children with NW also out-performed children with OB only on the NoGo task, as they exhibited significantly higher response accuracy.[3] The lack of a difference in P3 scalp distributions across task conditions in children with OB may reflect several health factors on the molecular and cellular level, such as inflammatory biomarkers that have been known to accompany excess adiposity.[10] Additionally, the error-related negativity (ERN) has also been assessed in relation to weight status and cognitive function in children.[12] Data from a modified flanker task indicate that ERN amplitude, a negative ERP component which reflects the action monitoring system and is modulated as a function of response conflict (ie, often elicited via incorrect responses), was smaller in children with OB relative to their NW counterparts. Developmental studies have consistently demonstrated that ERN amplitude increases with age during childhood, which suggests that a smaller ERN amplitude may reflect less effective maturation of the anterior cingulate cortex, the neural circuit related to action monitoring.[13-17] This reduction in ERN amplitude may suggest that childhood OB is associated with blunted modulation of the cognitive control network supporting action monitoring. Together, these data suggest that excess adiposity may be detrimental to specific aspects of cognition and brain function for children with OB.

1.3 | Physical activity and cognitive function: randomized controlled trials

Physical activity (PA) has demonstrated a positive impact on some of the complications associated with childhood OB, such as decreased risk of cardiovascular disease and type 2 diabetes.[18] Previous research has investigated the benefits of chronic PA participation on cognitive, brain, and physical health markers in the preadolescent population. For example, Khan et al[19] observed that the 9-month FITKids intervention (70 minutes of moderate-to-vigorous PA [MVPA] 5 days/week) in children 8 to 9 years old improved cardiorespiratory fitness, reduced fat mass, and prevented accumulation of central fat mass. The primary aim of the FITKids intervention was conducted irrespective of weight status and, as such, did not investigate the role of weight status on physical or cognitive outcomes. Further evidence suggests that children in the FITKids PA intervention demonstrated greater working memory performance from pre- to post-test.[20] In addition, all children regardless of weight status in the FITKids PA intervention also improved cardiorespiratory fitness relative to children in the wait-list control (CON) group. Notably, children who received the FITKids PA intervention regardless of weight status exhibited greater pre- to post-test improvements in inhibitory control and cognitive flexibility, during performance of a modified flanker task and a switch task, respectively.[21] Children in the PA intervention also increased attentional resources for tasks requiring inhibitory control and cognitive flexibility, as identified via increased P3 amplitude, indicating a greater ability to allocate attentional resources during cognitively demanding tasks. Specifically, increased P3 amplitude for incongruent relative to congruent trials was observed at electrode sites FCz, Cz, and CPz, meanwhile faster P3 latency was observed at the FCz site for post-test relative to pre-test. It was suggested that the PA intervention subsequently enhanced cognitive performance and brain function during tasks that require greater amounts of cognitive control.[21] Lastly, children who participated in the FITKids PA intervention displayed greater cardiorespiratory fitness and response accuracy on tasks that modulated inhibitory control demands (ie, a modified flanker task).[22] These data were further supported by the ERN during the modified flanker task.[22] That is, increased ERN amplitude was observed for children in the CON group from pre- to post-test. Alternatively, ERN amplitude remained stable from pre- to post-test for children in the PA intervention. Accordingly, the data indicate that daily PA may not only promote fitness, but subsequently it may benefit underlying neural processes associated with effective conflict monitoring during inhibitory control tasks.[22]

Similar effects on tasks of cognitive function have also been observed following 15-week PA interventions in preadolescent children with overweight.[23,24] Data indicate that following a high dose (40-minutes/day, 5 days/week) of aerobic MVPA (heart rate > 150 bpm), children demonstrated significantly greater post-test scores on the Cognitive Assessment System compared to children in the control group who did not receive MVPA, specifically in cognitively challenging tasks such as planning[23] and academic achievement math tasks.[24] Additionally, a small subset of this study also participated in functional magnetic resonance imaging (fMRI) consisting of baseline and post-test brain scans during an anti-saccade task of executive function. Analyses revealed that children who participated in the PA intervention (n = 11) demonstrated increased bilateral prefrontal cortex activity and decreased activity in bilateral posterior parietal cortex,[24] compared to controls (n = 9). In a separate, larger fMRI study, preadolescent children with overweight completed the same anti-saccade task, and a flanker task, at baseline and following an 8-month MVPA intervention (n = 43).[25] fMRI data suggest that children with overweight who received MVPA demonstrated decreased activation in several regions supporting anti-saccade performance, including the precentral gyrus and posterior parietal cortex, and increased activation in several regions supporting flanker performance including anterior cingulate and superior frontal gyrus.[25] Collectively, these data suggest that a 3-month, 8-month, and 9-month intervention of aerobic MVPA after-school is beneficial for the cognitive and brain function of children with overweight.

1.4 | Current study

Collectively, there is evidence suggesting that excess adiposity negatively influences cognitive and brain function, and that PA positively influences cognitive and brain function in children. Although OB has been related to measures of cognition in children, the influence of OB on a long-term PA program *and* cognitive function remains poorly characterized among preadolescent children. Accordingly, the aim of the current study was to investigate the effects of OB and PA on cognitive and brain function during a task that modulated inhibitory control demands using a sample of preadolescent children with NW and OB who participated in the FITKids intervention. The current study incorporates the major component of weight status into the analysis of the effects of PA on P3 amplitude and latency, an important addition which distinguishes itself from previous FITKids papers, that examined a smaller group of children with OB (n = 54). Children with OB may respond differently to a PA intervention both in terms of physical and cognitive outcomes relative to their NW counterparts; thus, we hypothesized that (1) children with OB in the PA intervention will show reduced adiposity at post-test compared to children with OB in the wait-list CON intervention; (2) at pre-test, children with NW would demonstrate greater cognitive and neuroelectric function (ie, P3 amplitude) compared to children with OB; and (3) NW and children with OB in the PA intervention will improve their cognitive function and exhibit larger P3 amplitude during the inhibitory control task at post-test compared to pre-test, relative to their counterparts in the CON intervention.

2 | METHODS

2.1 | Participants

The present study includes a subset of the 407 children between the ages of 8 and 10 years old who were recruited to participate in the FITKids (n = 212) and FITKids2 (n = 195) research trials (ClinicalTrials.gov Identifier numbers: NCT01334359, NCT01619826, respectively) (Figure 1). All participants provided written assent and their legal guardians provided written informed consent in accordance with the Institutional Review Board of the University of Illinois at Urbana-Champaign. Participants were administered the Kaufman Brief Intelligence Test[26] or the Woodcock Johnson (III)[27] to assess IQ, a Tanner Staging System questionnaire to assess pubertal status,[28] and the Physical Activity Readiness Questionnaire to screen for health issues exacerbated by physical exercise.[29] Socioeconomic status (SES) was determined using a trichotomous index based on participation in free or reduced-price meal program at school, the highest level of education obtained by the mother and father, and the number of parents who worked full time.[30] Exclusion criteria included the presence of neurological disorders and physical disabilities, and other factors that precluded participation in the cognitive or physical aspects of the study, such as not completing either the aerobic fitness test, the body composition scan, or both pre- and post-test cognitive and brain function assessments.[31] After these exclusionary criteria were applied, 111 children with OB were identified: 8 of those children were further removed due to outlier detection on the cognitive task, while 103 children with OB who completed both pre- and post-testing remained in the dataset. Thus, 103 children with NW (56: PA group; 47: CON group) were matched to 103 children with OB based on treatment allocation and demographic variables, including sex, age, IQ, SES, and fitness (fat-free VO_2max). A coin was flipped by an independent researcher to determine treatment allocation. Matching the NW and OB samples based on treatment allocation and demographics was an important process to ensure there were no differences in variables such as IQ, fitness, and SES, as these have been known to influence flanker task performance.[32] Additionally, the matching process was blind to both flanker and P3 performance outcomes.

2.2 | Cognitive control task

Inhibitory control was assessed using a modified version of the Eriksen flanker task.[33,34] Participants completed two blocks (75 trials) of equiprobable congruent (eg, <<<<<, >>>>>) and incongruent (eg, <<> < <, >><>>) stimuli (3-cm tall goldfish on a blue background) presented using Neuroscan STIM software (Compumedics NeuroScan, Charlotte, NC) on a computer screen at a distance of approximately 1 m. Participants were instructed to respond as accurately and quickly as possible using a button box based in accordance with the directionality of a centrally located, target stimuli. Each trial was presented for 200 ms (FITKids) or 250 ms (FITKids2) with either a fixed intertrial interval (ITI) of 1700 ms (FITKids) or random ITI of 1600, 1800, and 2000 ms (FITKids2). Research staff were blinded to intervention group and experimental hypotheses. Outcomes of cognitive control task included mean response accuracy (percentage of correct responses) and reaction time (RT; the period of time from stimulus onset to response execution for correct trials). In addition, interference scores were calculated for accuracy and RT outcomes, which consisted of the absolute difference between congruent and incongruent trials.

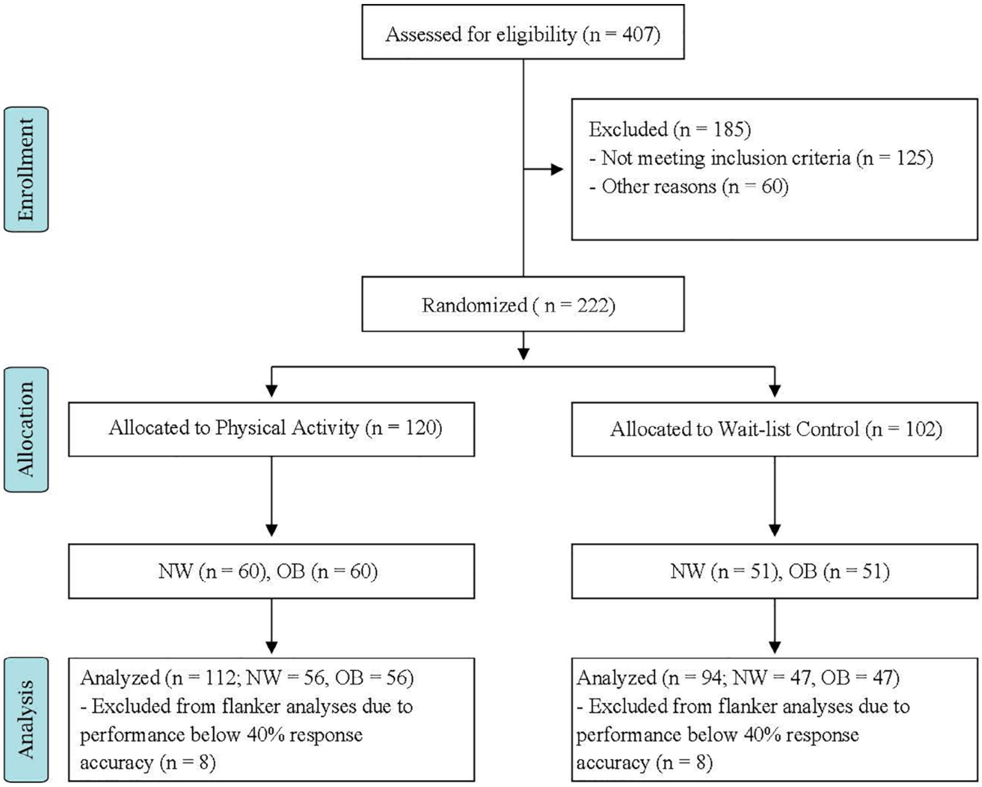

FIGURE 1 Flow diagram of the FITKids and FITKids2 intervention

TABLE 1 Participant demographics by intervention and weight status. Participants were matched on age, socioeconomic status (SES), IQ, fitness (VO2) across groups, and whole-body percent fat between weight status groups

	NW		OB	
	PA	CON	PA	CON
N (female)	56 (29)	47 (29)	56 (29)	47 (29)
Age (years)	8.64 ± 0.60	8.82 ± 0.67	8.73 ± 0.51	8.82 ± 0.54
SES (mothers' education)	1.82 ± 0.87	1.78 ± 0.81	1.82 ± 0.81	1.47 ± 0.72
IQ (standard score)	106.61 ± 15.93	108.17 ± 12.65	104.70 ± 11.87	108.11 ± 10.60
VO_2 Fat-free ($mL/kg_{(lean)}/min$)	57.15 ± 8.02	57.81 ± 5.11	54.33 ± 7.70	54.60 ± 7.07
Whole body fat (%)	28.30* ± 5.12	26.10* ± 4.31	40.00* ± 4.73	40.77* ± 4.84

Note: *Significant difference between BMI (NW, OB) groups ($p \leq 0.05$).

2.3 | Neuroelectric recording

EEG activity was recorded from 64 electrode sites arranged according to the international 10-10 system[35] using a Neuroscan Quik-cap (Compumedics Neuroscan), referenced to a midline electrode placed at the midpoint between Cz and CPz, with AFz serving as the ground electrode. Interelectrode impedance was <10 kΩ. Electrooculographic activity was collected from electrodes placed above and below the left 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, continuous EEG data were processed utilizing MATLAB (R2012b) and various toolbox plug-ins including EEGLAB [36] and ERPLAB,[37] re-referencing to average mastoids, creation of stimulus locked epochs (−200 to 1000 ms relative to stimulus onset), baseline correction (−200 to 0 ms pre-stimulus period), lowpass filtering (30 Hz, 24 dB/octave), and artifact rejection (epochs that exceeded ±75 μV were rejected). Independent component analyses (ICAs) were conducted to identify stereotypical eyeblink artifact[38] followed by an autocorrelation procedure developed for rejecting ICA components related to VEOG activity. This was accomplished by correlating point-by-point raw VEOG data with separate ICA activation waveforms (ie, EEG.icaact matrix generated by the ICA procedure). No more than two ICA components with a correlation coefficient greater than 0.30 were removed. Trials with a response error were excluded from the ERP analyses. The P3 component was quantified as the maximum positive deflections occurring within a 300 to 600 ms latency window.

Mean amplitudes and peak latencies were outputted in ASCII format and analysed using SPSS 24.

2.4 | Weight status and adiposity assessment

Standing height and weight measurements were completed with participants wearing light-weight clothing and no shoes. Height and weight were measured using a stadiometer (Seca; model 240) and a Tanita WB-300 Plus digital scale (Tanita, Tokyo, Japan), respectively. Adiposity measurements included BMI, whole-body percent fat (%Fat), VAT, and SAAT. BMI was calculated by dividing body mass (kg) by height (m) squared [(kg)/ht(m)^2]. The Centers for Disease Control and Prevention growth charts were used to determine individual BMI and BMI percentiles for age and sex values. Whole-body and regional soft tissue were measured by dual-energy X-ray absorptiometry (DXA) using a Hologic QDR 4500A Discovery bone densitometer (software version 13.4.2; Hologic, Bedford, MA), as an accurate and valid measure of body composition in the pediatric population.[39] Central adiposity (ie, VAT, SAAT) was estimated using an algorithm that models SAAT at the fourth lumbar vertebra and subtracts it from the regional abdominal region fat.[19]

2.5 | Cardiorespiratory fitness testing

Maximal oxygen consumption was measured on a treadmill using a graded VO_2max test, with a computerized indirect calorimetry system (ParvoMedics true Max 2400). A modified Balke protocol was utilized, whereby participants walked or ran at a constant speed with increasing grade increments of 2.5% every 2 minutes until volitional exhaustion, with time interval averages of VO_2 and respiratory exchange rate assessed every 20 seconds. Heart rate was assessed throughout the test with a Polar Heart Rate Monitor. Additionally, the children's OMNI scale[40] was used to assess ratings of perceived exertion every 2 minutes. VO_2max qualification was based upon achieving at least three of the following criteria: (i) a peak heart rate ≥ 185 bpm and a heart rate plateau, (ii) respiratory exchange ratio ≥ 1.0, (iii) an OMNI rating of perceived exhaustion ≥8, and/or (iv) a plateau in oxygen consumption corresponding to an increase of less than 2 mL/kg/min despite an increase in intensity. Fat-free VO_{2max} (FF-VO_2max; ml/min/kg-lean mass) was calculated using absolute VO_2max (L/min) and lean mass (g) as the primary measure of fitness, as this measure has previously been shown to be the primary contributor to aerobic capacity in children of varying body sizes.[39]

2.6 | Physical activity intervention

The aforementioned measures were completed before and after randomization into a 9-month intervention program. Children received either a PA intervention or were placed in a wait-list CON group. Children in the PA intervention received a 2-hour intervention during 5 days per week for 9 months (150 days), based on the Child and Adolescent Trial for Cardiovascular Health curriculum. Children were encouraged to attend at least 80% of the intervention, and this adherence rate was successfully achieved (79.97% adherence). This intervention consisted of an evidence-based PA program, which provided MVPA via fitness stations, dance and motor skill development, and small-sided games such as 3v3 soccer, in an after-school non-competitive environment. The activities were aerobically challenging but simultaneously provided opportunities to refine motor skills.

The CON group was asked to maintain their regular after-school routine and was not contacted again until post-testing 9-months later. A more detailed description of the intervention has been published previously.[21]

2.7 | Statistical analysis

Fitness and adiposity measures were assessed using a 2 (weight status: NW, OB) × 2 (intervention: PA, CON) × 2 (time: pre, post) repeated measures multivariate analysis of variance (MANOVA). Cognitive outcomes of the flanker task were analysed using a 2 (weight status: NW, OB) × 2 (intervention: PA, CON), × 2 (congruency: congruent, incongruent) × 2 (time: pre, post) repeated measures MANOVA. The cognitive outcomes of interest included response accuracy and mean RT. Furthermore, interference effects of accuracy (congruent minus incongruent) and RT (incongruent minus congruent) were calculated and analysed using a 2 (weight status: NW, OB) × 2 (intervention: PA, CON) × 2 (time: pre, post) MANOVA. P3 ERP amplitude and latency were assessed using a 2 (weight status: NW, OB) × 2 (intervention: PA, CON), × 2 (congruency: congruent, incongruent), × 7 (electrode site: Fz, FCz, Cz, CPz, Pz, POz, Oz), × 2 (time: pre, post) repeated measures MANOVA. When significant interactions including the cognitive outcome of interest (ie, difference in behavioural responses as a function of weight status and/or intervention and/or task congruency and/or time) were found in the cognitive and/or P3 analysis, the same repeated measures MANOVA was conducted at the appropriate level. For simplicity, only significant interactions involving time were considered relevant and decomposed further. Differences between children with NW and OB in demographic, adiposity, fitness, and cognitive measures were assessed using a family-wise alpha threshold for all tests set at $P = 0.05$. Differences between groups were post hoc corrected using Tukey HSD corrections (HSD > 3.63 critical value, $k = 4$, $df = 202$, $P \leq 0.05$). Bivariate correlations between change scores (Δ: post–pre) for cognitive and ERP outcomes of the flanker task, fat-free fitness, and adiposity were also analysed, within each intervention and weight category (ie, NW PA; NW CON; OB PA; OB CON).

3 | RESULTS

Participant demographics are presented in Table 1. Children with NW were matched to the sample of children with OB for key demographic variables as well as intervention assignment and did not differ statistically in age, sex, IQ, SES, or FF-VO_2max, confirming efficacy of the participant matching procedure. As expected, children with NW and OB differed in adiposity variables of %Fat, $t(204) = 19.02$, $P \leq 0.001$ (NW: 27.42 ± 0.49%, OB: 40.35 ± 0.46%), VAT, $t(204) = 16.78$, $P \leq 0.001$ (NW: 122.01 ± 5.03 g, OB: 320.27 ± 10.68 g), and SAAT, $t(204) = 17.67$ $P \leq 0.001$ (NW: 510.54 ± 26.19 g, OB: 1488.53 ± 48.75 g), BMI, $t(204) = 24.83$, $P \leq 0.001$ (NW: 16.43 ± 0.14, OB: 25.34 ± 0.33%) and BMI percentile, $t(204) = 19.57$, $P \leq 0.001$ (NW: 52.15 ± 2.34%ile, OB: 98.05 ± 0.12%ile).

3.1 | Aerobic fitness

The ANOVA of fat-free VO_2max revealed a main effect of time, $F(1, 200) = 19.71$, $P \leq 0.001$, $\eta^2 = 0.99$, with a significant increase from pre-test (56.11 ± 0.51 mL/kg/min) to post-test (58.04 ± 0.53 mL/kg/min), and a main effect of weight status, $F(1, 200) = 153.48$, $P \leq 0.001$, $\eta^2 = 1.00$, where children with NW demonstrate a significantly greater fat-free VO_2max (58.57 ± 0.67 mL/kg/min) compared to children with OB (55.67 ± 0.78 mL/kg/min).

3.2 | Adiposity variables

Whole-body percent fat (%Fat): The ANOVA revealed a main effect of weight status, $F(1,200) = 342.89$, $P \leq 0.001$, $\eta^2 = 1.00$, where children with NW had significantly lower %Fat (27.26 ± 0.48%) compared to children with OB (40.13 ± 0.49%). These effects were superseded by an interaction of time × intervention, $F(1,200) = 10.24$, $P \leq 0.05$, $\eta^2 = 0.89$, whereby the PA intervention decreased %Fat from pre-test (33.88 ± 0.75%) to post-test (33.20 ± 0.72%), $t(113) = 3.19$, $P = 0.002$ (Figure 2). Such an effect was not observed in the CON group from pre-test (33.52 ± 0.95%) to post-test (33.82 ± 0.93%), $t(93) = 1.35$, $P > 0.05$.

VAT: The ANOVA revealed a main effect of time, $F(1,200) = 12.79$, $P \leq 0.001$, $\eta^2 = 0.95$, with increases in VAT from pre-test (221.89 ± 6.30 g) to post-test (232.36 ± 6.98 g), and an effect of weight status, $F(1,200) = 250.84$, $P \leq 0.001$, $\eta^2 = 1.00$, where children with NW had lower VAT (124.34 ± 9.11 g) compared to children with OB (329.81 ± 9.24 g). These effects were superseded by an interaction of time × weight status, $F(1, 202) = 7.54$, $P = 0.007$, $\eta^2 = 0.78$, whereby children with OB increased in VAT from pre-test (320.62 ± 11.67 g) to post-test (337.98 ± 12.20 g), $t(92) = 3.36$, $P \leq 0.001$, an effect not seen in children with NW from pre-test (124.58 ± 4.98 g) to post-test (126.17 ± 4.92 g). Additionally, children with OB had significantly greater VAT at pre-test (320.27 ± 8.52 g) compared to children with NW (121.65 ± 8.39 g), $F(1,202) = 275.48$, $P \leq 0.001$, $\eta^2 = 1.00$, and similar patterns were seen for children with OB at post-test (336.77 ± 9.79 g) compared to children with NW (126.27 ± 9.69 g), $F(1, 202)$, $P \leq 0.001$, $\eta^2 = 1.00$. There was also an interaction of time × intervention, $F(1,202) = 9.92$, $P = 0.002$, $\eta^2 = 0.880$, whereby children in the CON group increased in VAT from pre-test (218.24 ± 14.53 g) to post-test (237.88 ± 15.56 g), $t(92) = 4.86$, $P \leq 0.001$, an effect not

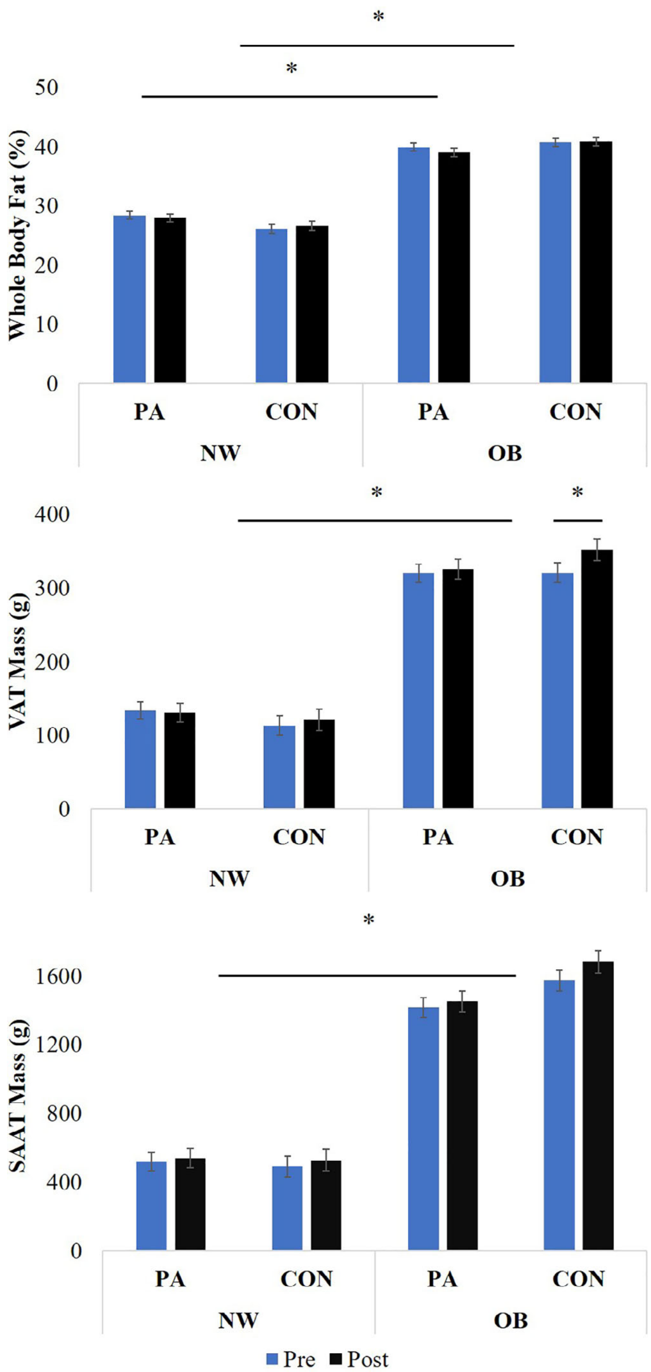

FIGURE 2 Means (standard errors) of adiposity: (i) whole-body percent fat (%Fat); (ii) visceral adipose tissue (VAT, g); (iii) subcutaneous adipose tissue (SAAT, g); split by intervention (PA, CON) and weight status (NW, OB). Blue bars indicate levels at pre-test, black bars indicate levels at post-test. Note: * Significant difference, paired (time: pre, post) comparisons ($P \leq 0.05$) within groups, and unpaired (weight status: NW, OB; intervention: PA, CON) comparisons between groups ($P \leq 0.05$)

seen in the PA group from pre-test (222.29 ± 12.53 g) to post-test (223.34 ± 13.89 g), $t(113) = 0.249$, $P > 0.05$ (Figure 2).

SAAT: The ANOVA revealed a main effect of time, $F(1,200) = 19.53$, $P \leq 0.001$, $\eta^2 = 0.99$, with increases in SAAT from pre-test (998.43

± 29.22 g) to post-test (1049.17 ± 30.66 g), and an effect of weight status, $F(1,200) = 296.58$, $P \leq 0.001$, $\eta^2 = 1.00$, where children with NW had lower SAAT (517.65 ± 41.26 g) compared to children with OB (1529 ± 41.87 g) (Figure 2).

3.3 | Cognitive outcomes

Response accuracy: The ANOVA revealed a main effect of time, $F(1,198) = 57.27$, $P \leq 0.001$, $\eta^2 = 0.17$, with an increase from pre-test (74.980 ± 0.93%) to post-test (81.76 ± 0.81%), and an effect of congruency, $F(1,198) = 283.19$, $P \leq 0.001$, $\eta^2 = 0.62$, with congruent responses (81.88 ± 0.76) having greater accuracy than incongruent responses (74.86 ± 0.79) (Table 2). No effects of intervention or weight status were noted.

Mean RT: The ANOVA revealed a main effect of time, $F(1,198) = 24.81$, $P \leq 0.001$, $\eta^2 = 0.14$, with longer RT at pre-test (546.67 ± 8.87 ms) compared to post-test (506.28 ± 7.45 ms), and an effect of congruency, $F(1,198) = 201.37$, $P \leq 0.001$, $\eta^2 = 0.58$, with congruent responses (508.63 ± 7.04 ms) having a shorter RT than incongruent responses (544.22 ± 7.73 ms) (Table 2). No effects of intervention or weight status were noted.

Interference effects: The ANOVA revealed no main effects of time, weight status, or intervention, for interference accuracy or interference RT (Table 2).

P3 ERP: P3 mean amplitude: The ANOVA revealed a main effect of congruency, $F(6,191)$,=34.48, $P \leq 0.001$, $\eta^2 = 1.00$, with congruent (7.79 ± 0.39 μV) trials having lower amplitudes than incongruent trials (9.27 ± 0.47 μV). There was a main effect of electrode site, $F(6,191) = 125.86$, $P \leq 0.001$, $\eta^2 = 1.00$, with the maximal mean amplitude occurring over the central electrodes (Fz: 4.20 ± 0.43 μV, FCz: 8.01 ± 0.46 μV, Cz: 11.69 ± 0.44 μV, CPz: 11.76 ± 0.43 μV, Pz: 10.18 ± 0.42 μV, POz: 9.51 ± 0.42 μV, Oz: 5.20 ± 0.45 μV). There was no main effect of time, $F(6,191) = 0.01$, $P > 0.05$, $\eta^2 = .05$, or weight status, $F(6,191) = 1.56$. $P > 0.05$, $\eta^2 = 0.08$. Main effects were superseded by an interaction of weight status × intervention × electrode site × time, $F(6,191) = 3.37$, $P = 0.022$, $\eta^2 = 0.74$. This four-way interaction was decomposed into four 3-way ANOVAs, whereby significant interactions involving time were considered relevant. ERP mean amplitude responses were averaged across congruency for further analyses. Notably, post hoc comparisons using the Tukey HSD test indicated that for children in the control group, there was a significant interaction between time × weight status at electrode Cz, $F(1,92) = 9.28$, $P = 0.003$ $\eta^2 = 0.85$, whereby mean amplitude for children with OB declined from pre-test (11.83 ± 1.17 μV) to post-test (10.10 ± 0.86 μV), t (46)=1.96, $P = 0.05$, and similarly at electrode CPz, $F(1,92) = 4.98$, $P = 0.027$, $\eta^2 = 0.60$, pre-test (12.41 ± 1.11 μV), post-test (10.34 ± 0.93 μV) t (46)=2.32, $P = 0.025$. In contrast, within the control group, P3 amplitude was maintained in children with NW, t (55)=1.76, p's > 0.05, from pre-test (electrode Cz: 10.64 ± 0.95 μV; electrode CPz: 10.64 ± 0.91 μV) to post-test (electrode Cz: 12.70 ± 0.94 μV; electrode CPz: 12.31 ± 0.93 μV). Within the PA group, P3 amplitude was maintained in children with NW from pre-test

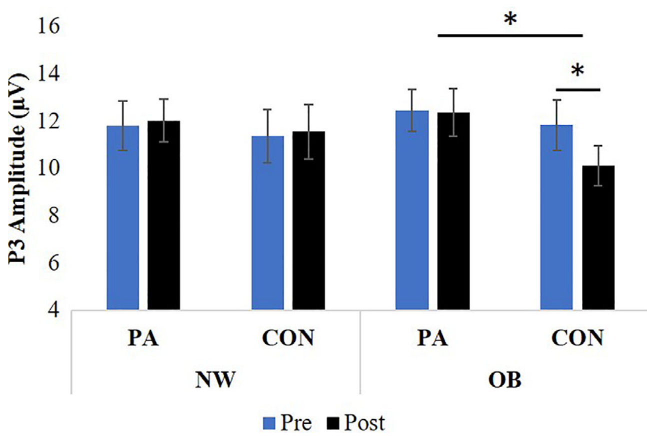

FIGURE 3 ERP means (standard errors) of the modified flanker task. Mean amplitude at electrode Cz for (i) normal weight children (NW, μV) and (ii) children with obesity (OB, μV) for each intervention (PA, CON). Blue bars indicate levels at pre-test, black bars indicate levels at post-test. Note: * Significant difference, paired (time: pre, post) comparisons ($P \leq 0.05$) within groups, and unpaired (intervention: PA, CON) comparisons between groups ($P \leq 0.05$)

(electrode Cz: 11.61 ± 1.12 μV, Figure 3; electrode CPz: 11.67 ± 1.12 μV) to post-test (electrode Cz: 12.01 ± 0.90 μV, Figure 3; electrode CPz: 11.90 ± 0.92 μV), t (56)=0.35, p's > 0.05. This effect was also seen in children with OB in the PA group, whereby mean amplitude was maintained from pre-test (electrode Cz: 12.44 ± 0.99 μV, Figure 3; electrode CPz: 13.29 ± 0.83 μV) to post-test (electrode Cz: 12.48 ± 1.01 μV, Figure 3; electrode CPz: 12.79 ± 0.90 μV), t (45) =0.66, p's > 0.6 (Figure 4).

P3 peak latency: The ANOVA revealed a main effect of congruency, $F(6,191) = 17.176$, $P \leq 0.001$, $\eta^2 = 0.99$, with congruent trials (537.96 ± 4.63 ms) having shorter latencies than incongruent trials (550.14 ± 4.10 ms). There was a main effect of electrode site, $F(6,191) = 113.61$, $P \leq 0.001$, $\eta^2 = 1.00$, with peak latency decreasing from frontal to occipital electrodes (Fz: 600.25 ± 5.93 ms, FCz: 576.49 ± 5.68 ms, Cz: 550.31 ± 5.39 ms, CPz: 542.88 ± 4.85 ms, Pz: 520.29 ± 4.55 ms, POz: 514.60 ± 4.64 ms, Oz: 503.53 ± 4.71 ms). Lastly, a main effect of time, $F(6,191)$ $P \leq 0.001$, $\eta^2 = 1.00$, indicated that post-test latencies (530.58 ± 4.59 ms) were shorter than pre-test latencies (557.62 ± 4.74 ms).

3.4 | Correlations

Results of the bivariate correlations (Table 3) across participants within each intervention and weight category (ie, NW PA; NW CON; OB PA; OB CON) revealed the following:

NW PA: Δ flanker congruent mean RT was significantly correlated with Δ VAT ($r = 0.305$, $P = 0.025$) and Δ SAAT ($r = -0.271$, $P = 0.047$), such that faster RT was associated with reduced VAT and faster RT was associated with increased SAAT; Δ flanker incongruent mean RT was also significantly correlated with Δ VAT ($r = 0.321$, $P = 0.018$), such that faster RT was associated with reduced VAT.

TABLE 2 Means (standard errors) for the modified flanker task: (i) response accuracy (%); (ii) mean reaction time (ms); (iii) interference accuracy and reaction time

		NW				OB			
		PA		CON		PA		CON	
		Pre Mean ± SE	Post Mean ± SE	Pre Mean ± SE	Post Mean ± SE	Pre Mean ± SE	Post Mean ± SE	Pre Mean ± SE	Post Mean ± SE
Response accuracy (%)	*Congruent*	78.09 ± 1.90	87.09 ± 1.50	81.09 ± 2.16	85.42 ± 1.70	75.05 ± 1.87	83.71 ± 1.47	79.82 ± 2.06	84.79 ± 1.622
	Incongruent	70.45 ± 1.81	79.66 ± 1.73	73.59 ± 2.05	78.62 ± 1.96	69.13 ± 1.78	76.02 ± 1.70	72.65 ± 1.96	78.77 ± 1.873
Mean RT (ms)	*Congruent*	520.79 ± 15.99	486.14 ± 14.15	529.97 ± 18.13	496.23 ± 16.04	536.28 ± 15.70	486.94 ± 13.89	529.36 ± 17.32	483.31 ± 15.325
	Incongruent	559.57 ± 18.18	529.38 ± 14.61	561.24 ± 20.61	521.80 ± 16.57	569.44 ± 17.85	525.18 ± 14.35	566.68 ± 19.70	520.44 ± 15.833
Interference accuracy (%)		7.43 ± 0.96	7.64 ± 1.10	6.80 ± 1.09	7.50 ± 1.24	7.68 ± 0.94	5.92 ± 1.08	6.02 ± 1.04	7.16 ± 1.187
Interference RT (ms)		43.25 ± 4.83	38.78 ± 5.87	25.57 ± 5.48	31.28 ± 6.66	38.24 ± 4.75	33.16 ± 5.77	37.13 ± 5.24	37.32 ± 6.361

Note: Paired (time: pre, post; and congruency: congruent, incongruent) comparisons within groups for response accuracy and mean RT are all significant (*p*'s ≤ 0.05).

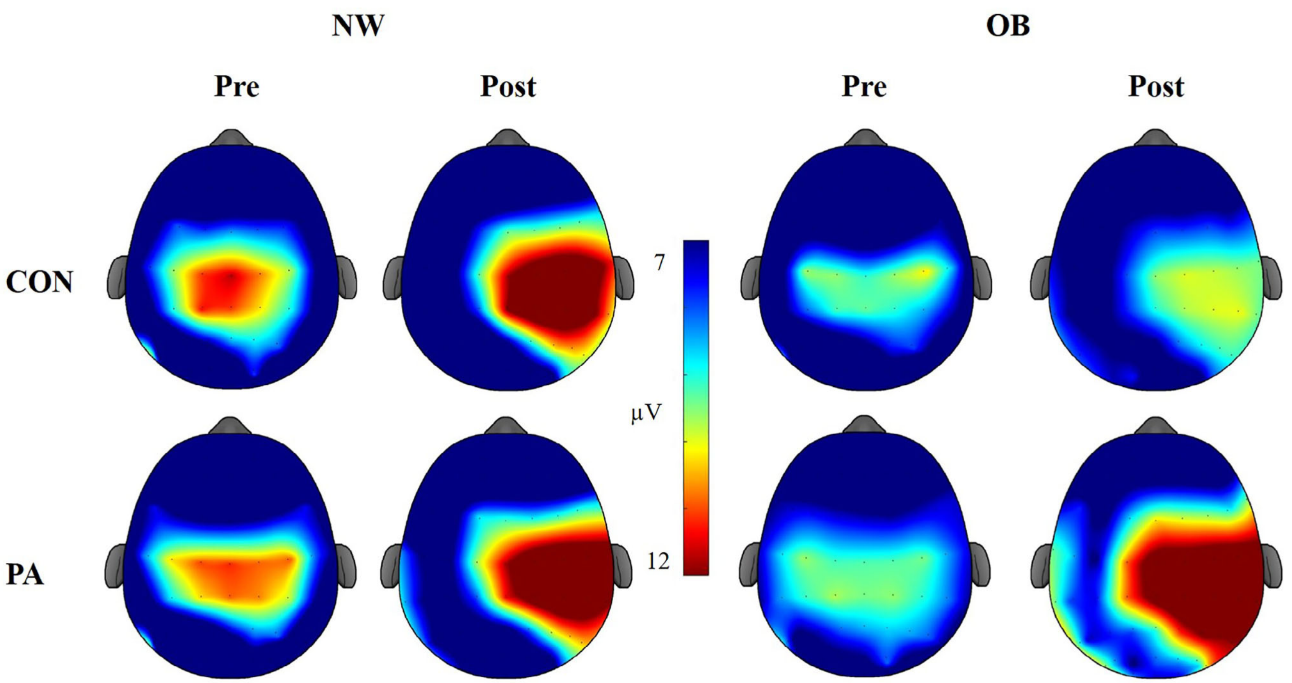

FIGURE 4 Topographic scalp plots of mean P3 amplitude (μV) of the modified flanker task for each intervention (PA, CON) and weight status (NW, OB)

NW CON: no significant correlations.

OB PA: Δ flanker congruent response accuracy was negatively correlated with Δ VAT (r = –0.314, P = 0.026) and Δ SAAT (r = –0.308, P = 0.029), such that improved accuracy was associated with decreases in adiposity (VAT, SAAT).

OB CON: Δ flanker incongruent P3 mean amplitude at electrode site FCz was significantly correlated with Δ SAAT (r = –0.333, P = 0.031), such that decreased P3 amplitude was associated with increases in SAAT.

4 | DISCUSSION

This investigation provides insight into the differential relationship on the effects of a 9-month PA intervention on brain and cognition as a function of weight status and adiposity. That is, the PA intervention is particularly efficacious for maintaining P3 amplitude and decreasing adiposity in children with OB, while decline in P3 amplitude and increased adiposity were observed for children with OB in the CON group. These changes, which differed from the trajectory of children

TABLE 3 Correlation table between aerobic fitness and adiposity, and outcome measures (behavioural accuracy and reaction time, and P3 amplitude)

	NW PA				NW CON				OB PA				OB CON			
	Δ VAT	Δ SAAT	Δ %Fat	Δ VO_2max	Δ VAT	Δ SAAT	Δ %Fat	Δ VO_2max	Δ VAT	Δ SAAT	Δ %Fat	Δ VO_2max	Δ VAT	Δ SAAT	Δ %Fat	Δ VO_2max
Behavioural																
Δ Flanker congruent mean RT	0.305*	-0.271*	0.170	0.148	0.26	0.12	0.20	0.07	0.034	-0.112	-0.126	-0.001	0.085	-0.002	-0.093	0.063
Δ Flanker incongruent mean RT	0.321*	-0.247	0.174	0.176	0.29	0.17	0.26	0.02	0.002	-0.197	-0.111	-0.028	0.207	0.137	0.085	0.079
Δ Flanker congruent response accuracy	0.032	-0.124	0.110	0.140	0.13	0.20	0.21	-0.07	-0.314*	-0.308*	-0.273	0.037	-0.202	0.112	-0.022	-0.028
Δ Flanker incongruent response accuracy	0.120	-0.220	0.121	0.235	0.06	0.16	0.18	-0.10	-0.202	-0.173	-0.060	0.034	-0.109	-0.154	-0.033	0.017
ERPs																
Congruent																
Δ Flanker P3 mean amplitude Fz	-0.074	-0.126	0.038	0.118	0.09	-0.01	0.15	0.28	-0.190	0.054	-0.010	0.021	0.100	-0.040	-0.006	-0.247
Δ Flanker P3 mean amplitude FCz	-0.087	-0.194	0.080	0.258	0.19	0.06	0.18	-0.05	-0.177	-0.046	-0.071	0.062	0.073	-0.166	-0.145	-0.007
Δ Flanker P3 mean amplitude Cz	-0.114	-0.079	0.103	0.227	0.32	0.08	0.27	-0.20	-0.153	0.013	0.114	-0.070	-0.047	0.020	-0.095	0.007
Δ Flanker P3 mean amplitude CPz	-0.193	-0.118	0.020	0.201	0.29	0.09	0.26	-0.08	-0.126	-0.004	0.022	-0.058	-0.069	0.063	-0.124	0.057
Δ Flanker P3 mean amplitude Pz	-0.189	-0.043	0.098	0.193	0.18	-0.04	-0.10	0.04	-0.122	-0.051	0.107	-0.004	-0.104	0.083	-0.090	0.095
Incongruent																
Δ Flanker P3 mean amplitude Fz	-0.145	0.013	-0.045	-0.037	0.09	0.04	-0.04	-0.29	-0.150	0.162	0.104	-0.105	-0.108	-0.196	-0.166	-0.160
Δ Flanker P3 mean amplitude FCz	-0.210	-0.031	-0.013	0.046	0.11	0.06	-0.01	-0.24	-0.135	0.078	0.019	-0.107	-0.226	-0.333*	-0.268	-0.090
Δ Flanker P3 mean amplitude Cz	-0.197	-0.011	-0.023	0.076	0.21	0.14	0.06	-0.22	-0.176	0.053	0.040	-0.196	-0.203	-0.025	-0.121	-0.150
Δ Flanker P3 mean amplitude CPz	-0.186	-0.041	-0.004	0.093	0.20	0.14	0.07	-0.17	-0.151	0.006	-0.040	-0.170	-0.260	0.073	-0.131	-0.110
Δ Flanker P3 mean amplitude Pz	-0.209	0.032	-0.063	0.061	0.26	0.09	0.05	-0.19	-0.160	-0.052	0.026	-0.083	-0.266	0.179	-0.118	-0.218

with NW, indicate a differential effect on cognition and brain function after PA, as a function of weight status.

Overall, children with NW in the PA intervention demonstrated significantly lower %Fat at the completion of the 9-month intervention, compared to children with NW in the CON group, indicating that the PA intervention was efficacious in decreasing adiposity. Additionally, both children with NW and OB in the CON group showed a significant increase in VAT at post-test, compared to their counterparts in the PA group, who did not show significant increases in adiposity at post-test. Furthermore, at both pre-test and post-test, children with OB demonstrated significantly greater VAT and SAAT compared to children with NW.

Behavioural analyses resulted in the expected, confirmatory effects across congruency and time, with children performing better on congruent relative to incongruent trials and at post-test relative to pre-test. Additionally, the results demonstrated a significant relationship between adiposity and the PA intervention. Specifically, children with NW in the PA intervention demonstrated a positive relationship between Δ congruent flanker RT and Δ VAT, such that a decrease in VAT corresponded with shorter RT. Additionally, children with OB in the PA intervention demonstrated a negative relationship between Δ flanker response accuracy and Δ VAT, such that decreased adiposity was associated with increased accuracy. Contrary to our initial hypothesis, we did not see an effect of intervention of weight status on flanker performance; however, we did see significant relationships between VAT and flanker performance, dependent upon weight status. The implication of adiposity in this relationship suggests that the internal metabolic changes in VAT over 9 months are important in observing behavioural differences, potentially more so than participation in a PA intervention. These findings corroborate recent studies by providing novel prospective support for the benefits of reducing adiposity for cognitive benefits among preadolescent children.[4]

ERP analyses revealed children with OB in the CON group demonstrated a reduction in P3 amplitude from pre- to post-test, reflecting a reduction in the allocation of attentional resources during task conditions that require variable amounts of inhibitory control.[41] Furthermore, children with OB in the PA intervention maintained P3 amplitude from pre- to post-test. However, this trajectory appears to differ for children with NW. Regardless of PA participation, children with NW also maintained P3 amplitude from pre- to post-test. Such a pattern of results suggests that carrying excess body fat (without participating in a prescribed PA program during childhood) may have negative consequences for brain function, as measured via the P3 potential. Meanwhile PA, particularly for children with excess body fat, may positively maintain brain function during preadolescent development. In addition, the lack of excess body fat in children with NW may also positively maintain brain function during typical development. Excess body fat can be stored in the viscera, giving way to VAT.[8] VAT is stored within the abdominal cavity and around vital organs. It is a metabolically active endocrine tissue, which produces inflammatory cytokines and hormones.[9] Excess VAT may lead to altered metabolic and inflammatory states as well as cardiovascular disease, demonstrating an important link between adiposity and the cardiovascular system,[42] an essential factor to evaluate the beneficial effect of PA on cognitive and brain function.

Additionally, MRI evidence suggests that lean mass index (LMI) among children with overweight or OB is positively associated with regions overlapping white matter tracts, which are related to executive function and planning, emotion and conflict processing, visual processing, attention, memory, and motor function, independent of fat mass index or BMI.[43] These data contribute to previously mixed findings in the field of OB with grey and white matter, such that LMI is suggested to be a positive predictor of regional white matter volumes in children with overweight or OB, indicating a relationship between brain structure and OB. Furthermore, MRI evidence indicates a negative influence of adiposity on brain function in older children.[44] Adolescents (15-18 years old) demonstrated significant associations between a greater visceral fat ratio and increased cortical thickness throughout the brain.[44] As increased cortical thickness has also shown associations with decreased cognitive ability in children,[45,46] the relationship observed between cortical thickness and VAT, but not weight status, is crucial in evaluating the mechanistic nature of differences in cognitive performance in children with excess adiposity. As such, the PA intervention in the current study may serve to positively influence the cardiovascular system, metabolic, and inflammatory states in children with OB and, in turn, promote maintenance of cognitive function as assessed by flanker performance.

Interestingly, behavioural correlational analyses revealed that children in the PA intervention exhibited differential positive effects on cognitive function, dependent upon weight status. Specifically, children with NW demonstrated associations between lower adiposity and shorter RTs; meanwhile, children with OB demonstrated associations between decreased adiposity and improved performance accuracy. The relationship between shorter RTs and positive health states has been observed previously. Westfall et al.[47] identified that higher fitness in adolescents was associated with shorter RT in the flanker task, and Hwang, Castelli, and Gonzalez-Lima[48] demonstrated that young adults with excellent or superior levels of aerobic fitness maintained faster speed performance during tasks of sustained attention, and shorter yet accurate memory retrieval during working memory tasks. Meanwhile, the relationship between longer RTs and increased weight status in adolescents has also been observed. Huang et al.[49] showed that higher waist circumferences, as measured with anthropometric tape at the level of the umbilicus, were associated with a longer RTs on a modified flanker task. Collectively, the pattern of results highlights the beneficial differential effects which both weight status and PA have on preadolescent cognition and brain function.

Notably, the pattern of results after the PA intervention indicates maintenance of brain function with associations to changes in cognitive performance. The strength of these findings is important to consider, as changes to the biomarkers of brain function, such as P3 amplitude, may in fact proceed changes in behavioural performance, such that changes in brain structure may lead to changes in brain function, before changes in behavioural performance are observed.[50]

Additionally, changes to behavioural outcomes in children may be apparent during other cognitive tasks that further upregulate inhibitory control demands, or which may be present during other tasks that tap different aspects of executive function (ie, working memory[20], cognitive flexibility[21]). The current data indicate that, while behavioural outcomes of the flanker task were not overly sensitive to the relationship between PA and weight status, neuroelectric markers, such as the P3 ERP, may have the requisite sensitivity to illuminate the effects. Similar patterns have also been observed in a cross-sectional MRI study of preadolescent children,[50] whereby children with higher aerobic fitness levels also had large hippocampal volumes, compared to their lower-fit counterparts, yet no association between aerobic fitness, hippocampal volume, and memory performance was observed.[50] These results support the finding that neural correlates of the PA and cognition relationship, such as P3 amplitude and hippocampal volume, may precede changes in performance in children.

Another consideration of the current study is the robust changes to P3 amplitude in children with OB in the CON group, despite the absence of changes to P3 latency. Previous literature indicates that the FITKids PA intervention was beneficial for reducing P3 latency during the incongruent condition of the flanker task in children (however, BMI and/or adiposity were not considered in the model, with a sample of 54 children with OB out of a total 221 children).[21] This finding was not supported in the current study, suggesting that, when weight status is considered in the model, P3 latency is less susceptible than P3 amplitude to change after a PA intervention. This idea is supported by previous research in children with OB on tasks of attention. Specifically, results indicated that P3 latency was not sensitive to weight status, and that the time needed for target stimulus evaluation and detection was comparable between weight groups.[51] Evidently, further identification into the specificity of P3 amplitude and latency in children with OB is needed. The findings indicate that the PA intervention may be particularly beneficial for children with OB, as they maintained P3 amplitude from pre- to post-test over 9 months, compared to children with OB in the CON group. Thus, the current findings suggest a negative relationship between elevated weight status and P3 amplitude, and further, this relationship is mitigated by a 9-month PA intervention. Additionally, the current study shows the detrimental effects of adiposity on brain indices of executive function over a 9-month period as children with OB in the CON group exhibited decreased P3 amplitude across time, which appears to differ from the trajectory of regular development in children with NW and OB engaged in regular PA. The results of the current study complement previous findings in children investigating cross-sectional associations between: OB and amplitude of the N2 and P3[3]; adiposity, intellectual, and cognitive abilities[4]; OB and memory performance[10]; and RCT investigations assessing: adiposity changes after a PA intervention[19]; changes in cognitive performance and P3 amplitude after a PA intervention[21]; and OB and cognitive functioning after a PA intervention.[23,24]

The interaction between PA and adiposity is important to consider when evaluating cognitive and brain function in preadolescent children with NW and OB. Following prior literature,[3,4,10,52] the current data provide additional support for a selective relationship between these constructs, with regular PA uniquely influencing and protecting brain function, especially for typically developing children who carry excess adiposity. The excess accumulation of VAT in children with OB may be linked to reduced brain indices of executive function during stimulus engagement, which has been shown to be mitigated by PA. The current data indicate that children with OB who received daily PA intervention reduced VAT and improved brain indices of executive function, compared to their CON counterparts, who increased VAT and exhibited a reduction in P3 amplitude. The notion that VAT may be implicated in cognitive and brain function supports previous research, such that the metabolic, inflammatory, and insulin resistant by-products associated with excess VAT may be related to diminished cognitive and brain structure and function.[3,4,19,23-25,44,52-54] However, critically, PA plays a novel role in mitigating VAT and cognitive function during a period of the lifespan characterized by rapid development of the brain. Further research is needed to determine how VAT relates to cognitive performance and brain function during childhood, and how it can be manipulated via lifestyle interventions such as PA.

PA interventions continually benefit the physical and mental health of typically developing children.[3,4,19-22,52,55,56] Given the findings from the current study, PA appears increasingly important to cognitive function, particularly in those who need it most. Specifically, children with OB, whose physical development deviates from the typical height and weight curve, have been associated with comparatively poorer brain function, relative to NW children, under certain conditions. Such a premise is supported by previous research, which demonstrated that acute bouts of exercise had a greater benefit on brain and cognitive function in preadolescent children, who performed more poorly (albeit still within the normal range) on a cognitive task that modulated inhibitory control requirements.[57] Although this research investigated the transient benefits stemming from an acute bout of aerobic exercise,[57] the current (chronic) findings of a 9-month PA intervention appear consistent, wherein children with NW and OB maintained P3 amplitude from pre- to post-test, while children with OB without PA demonstrated reduced benefits in brain functioning, as indicated by a decrease in P3 amplitude at post-test.

4.1 | Limitations and future directions

Although recent advances in software technology permit the estimation of VAT using DXA,[58] its application to developing children requires caution as the strength of the correlation between DXA generated estimates and CT- and MRI-derived visceral fat range between moderate to strong (ie, 0.62-0.90).[59] Additionally, although this study accounted for key demographic and fitness covariates, this study did not address metabolic, molecular, or cellular markers of inflammation, which should be noted for future investigations. It is also worth noting that this study specifically examined children with OB and did not account for children with overweight in the sample, due to an a priori focus on the more extreme weight group. Therefore, the relationship between adiposity, PA, cognition, and brain function in the overweight group remains unknown. Lastly, childhood OB itself is further classified into three classes: BMI ≥95th percentile as Class I; BMI ≥120% of the 95th percentile as Class II; and BMI ≥140% of the 95th percentile as Class III. Therefore, future investigations should consider using this classification for children with OB to further detect potential differences between OB classes, in comparison to their NW counterparts. Additionally, assessing the mechanisms behind the beneficial effects of PA on cognition should be considered. Specifically, how this relationship is moderated by factors such as IQ, or mediated by factors such as P3 amplitude, are important avenues to address in future studies.

4.2 | Conclusion

The complex relationship between PA and the neuroelectric indices of cognition in children is particularly interesting when accounting for weight status and changes in adiposity. Regular engagement in PA over a 9-month period enabled a reduction in whole-body percent fat for both NW and children with OB. Reduced adiposity in children as a result of PA may be linked to healthier physiological and psychological outcomes. Specifically, P3 amplitude has been shown to be overwhelmingly partial to the effects of PA in children. The results have repeatedly shown that greater P3 amplitudes, induced via regular PA or greater aerobic fitness, are associated with improved cognitive control. This study extended those findings to demonstrate differential effects of PA on P3 as a function of excess adiposity. The selective influence of adiposity and PA on P3 amplitude is therefore important to consider given the health complications associated with OB. The results from this study suggest a detrimental effect of excess adiposity in children with OB, which can be improved with PA, on tasks that manipulate inhibitory control demands. As greater cognitive capacity is optimal for overall mental and cognitive functioning, these results have important implications for the health and development of preadolescent children.

ACKNOWLEDGEMENTS

Support for this project was provided by National Institute of Health Human Development grants HD055352 and HD069381 awarded to Drs. Hillman and Kramer. These sources did not play a role in the study design, collection, analysis, or interpretation of the data, nor in the writing of the report and decision to submit the article for publication. All authors were involved in designing, carrying out, data interpretation or writing the paper, and had final approval of the submitted and published versions.

CONFLICT OF INTEREST

The authors of this document report no conflicts of interest associated with the collection, dissemination, or interpretation of this research. No patents, copyrights, or royalties are involved or included in this work. The results of this study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

ORCID

Nicole E. Logan https://orcid.org/0000-0003-4016-2146
Naiman A. Khan https://orcid.org/0000-0002-6135-9389

REFERENCES

1. [WHO] WHO. Obesity and Overweight. 2020; https://www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight#.
2. Ebbeling CB, Pawlak DB, Ludwig DS. Childhood obesity: public-health crisis, common sense cure. *The Lancet*. 2002;360(9331):473-482.
3. Kamijo K, Pontifex MB, Khan NA, et al. The association of childhood obesity to neuroelectric indices of inhibition. *Psychophysiology*. 2012b;49(10):1361-1371.
4. Raine LB, Drollette ES, Kao S-C, et al. The associations between adiposity, cognitive function, and achievement in children. *Med Sci Sports Exerc*. 2018;50:1868-1874.
5. Freedland ES. Role of a critical visceral adipose tissue threshold (CVATT) in metabolic syndrome: implications for controlling dietary carbohydrates: a review. *Nutr Metab*. 2004;1(1):12.
6. Arner P. Obesity and the adipocyte: regional adipocity in man. *J Endocrinol*. 1997;155:191-192.
7. Ibrahim MM. Subcutaneous and visceral adipose tissue: structural and functional differences. *Obes Rev*. 2010;11(1):11-18.
8. Wajchenberg BL. Subcutaneous and visceral adipose tissue: their relation to the metabolic syndrome. *Endocr Rev*. 2000;21(6):697-738.
9. Chaldakov GN, Stankulov IS, Hristova M, Ghenev PI. Adipobiology of disease: adipokines and adipokine-targeted pharmacology. *Curr Pharm des*. 2003;9(12):1023-1031.
10. Khan NA, Baym CL, Monti JM, et al. Central adiposity is negatively associated with hippocampal-dependent relational memory among overweight and obese children. *J Pediatr*. 2015;166:302-308.e1.
11. Polich J. Updating P300: an integrative theory of P3a and P3b. *J Clin Neurophysiol*. 2007;118(10):2128-2148.
12. Kamijo K, Pontifex MB, Khan NA, et al. The negative association of childhood obesity to cognitive control of action monitoring. *Cereb Cortex*. 2014;24(3):654-662.
13. Davies PL, Segalowitz SJ, Gavin WJ. Development of response-monitoring ERPs in 7-to 25-year-olds. *Dev Neuropsychol*. 2004;25(3):355-376.
14. Ladouceur CD, Dahl RE, Carter CS. Development of action monitoring through adolescence into adulthood: ERP and source localization. *Dev Sci*. 2007;10(6):874-891.
15. Santesso DL, Segalowitz SJ, Schmidt LA. Error-related electrocortical responses in 10-year-old children and young adults. *Dev Sci*. 2006;9(5):473-481.
16. Torpey DC, Hajcak G, Kim J, Kujawa A, Klein DN. Electrocortical and behavioral measures of response monitoring in young children during a Go/No-Go task. *Dev Psychobiol*. 2012;54(2):139-150.
17. Wiersema JR, van der Meere JJ, Roeyers H. Developmental changes in error monitoring: an event-related potential study. *Neuropsychologia*. 2007;45(8):1649-1657.
18. Ogden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index in US children and adolescents, 2007-2008. *J Am Med Assoc*. 2010;303(3):242-249.
19. Khan NA, Raine LB, Drollette ES, et al. Impact of the FITKids physical activity intervention on adiposity in prepubertal children. *Pediatrics*. 2014;133(4):875-883.
20. Kamijo K, Pontifex MB, O'Leary KC, et al. The effects of an afterschool physical activity program on working memory in preadolescent children. *Dev Sci*. 2011;14(5):1046-1058.

21. Hillman CH, Pontifex MB, Castelli DM, et al. Effects of the FITKids randomized controlled trial on executive control and brain function. *Pediatrics*. 2014;134:e1063-e1071.
22. Drollette ES, Pontifex MB, Raine LB, et al. Effects of the FITKids physical activity randomized controlled trial on conflict monitoring in youth. *Psychophysiology*. 2017;55(3):1-15.
23. Davis CL, Tomporowski PD, Boyle CA, et al. Effects of aerobic exercise on overweight children's cognitive functioning: a randomized controlled trial. *Res Q Exerc Sport*. 2007;78(5):510-519.
24. Davis CL, Tomporowski PD, McDowell JE, et al. Exercise improves executive function and achievement and alters brain activation in overweight children: a randomized, controlled trial. *Health Psychol*. 2011;30(1):91-98.
25. Krafft CE, Schwarz NF, Chi L, et al. An 8-month randomized controlled exercise trial alters brain activation during cognitive tasks in overweight children. *Obesity*. 2014;22(1):232-242.
26. Kaufman AS. *Kaufman brief intelligence test: KBIT. AGS*. Circle Pines, MN: American Guidance Service; 1990.
27. Schrank FA, Mather N, McGrew KS, Woodcock-Johnson IV. *Tests of Achievement*. Rolling Meadows, IL: Riverside Publishing; 2014.
28. Taylor S, Whincup P, Hindmarsh P, Lampe F, Odoki K, Cook D. Performance of a new pubertal self-assessment questionnaire: a preliminary study. *J Paediatr Perinat Epidemiol*. 2001;15(1):88-94.
29. Thomas S, Reading J, Shephard RJ. Revision of the physical activity readiness questionnaire (PAR-Q). *Can J Sport Sci*. 1992;17(4): 338-345.
30. Birnbaum AS, Lytle LA, Murray DM, Story M, Perry CL, Boutelle KN. Survey development for assessing correlates of young adolescents' eating. *Am J Health Behav*. 2002;26(4):284-295.
31. DuPaul GJ, Power TJ, Anastopoulos AD, Reid R. *ADHD rating scale—IV: checklists, Norms, and Clinical Interpretation*. New York, NY, US: Guilford Press; 1998.
32. Raine LB, Kao S-C, Pindus D, et al. A large-scale reanalysis of childhood fitness and inhibitory control. *J Cogn Enhanc*. 2018;2(2): 170-192.
33. Eriksen BA, Eriksen CW. Effects of noise letters upon the identification of a target letter in a nonsearch task. *Percept Psychophy*. 1974;16 (1):143-149.
34. Pontifex MB, Saliba BJ, Raine LB, Picchietti DL, Hillman CH. Exercise improves behavioral, neurocognitive, and scholastic performance in children with attention-deficit/hyperactivity disorder. *J Pediatr*. 2013; 162(3):543-551.
35. Chatrian GE, Lettich E, Nelson PL. Ten percent electrode system for topographic studies of spontaneous and evoked EEG activities. *Am J*. 1985;25(2):83-92.
36. Delorme A, Makeif S. EEGLAB: An open source tool-box for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*. 2004;134:9-21.
37. Lopez-Calderon J, Luck SJ. *ERPLAB (Version 1.0. 0.33 a) (Computer Software)*. CA: UC-Davis Center for Mind, Brain. 2010.
38. Comon P. Independent component analysis, a new concept? *Signal Process*. 1994;36(3):287-314.
39. Goran M, Fields DA, Hunter GR, Herd SL, Weinsier RL. Total body fat does not influence maximal aerobic capacity. *Int J Obes (Lond)*. 2000; 24(7):841-848.
40. Utter AC, Robertson RJ, Nieman DC, Kang J. Children's OMNI scale of perceived exertion: walking/running evaluation. *Med Sci Sports Exerc*. 2002;34:139-144.
41. Kao SC, Cadenas-Sanchez C, Shigeta TT, et al. A systematic review of physical activity and cardiorespiratory fitness on P3b. *Psychophysiology*. 2019;57:e13425.
42. Després J-P, Lemieux I, Bergeron J, et al. Abdominal obesity and the metabolic syndrome: contribution to global cardiometabolic risk. *Arterioscler Thromb Vasc Biol*. 2008;28(6):1039-1049.
43. Gracia-Marco L, Esteban-Cornejo I, Ubago-Guisado E, et al. Lean mass index is positively associated with white matter volumes in several brain regions in children with overweight/obesity. *Pediatr Obes*. 2020;15:e12604.
44. Saute R, Soder R, Alves Filho J, Baldisserotto M, Franco A. Increased brain cortical thickness associated with visceral fat in adolescents. *Pediatr Obes*. 2018;13(1):74-77.
45. Porter JN, Collins PF, Muetzel RL, Lim KO, Luciana M. Associations between cortical thickness and verbal fluency in childhood, adolescence, and young adulthood. *Neuroimage*. 2011;55(4):1865-1877.
46. Shaw P, Greenstein D, Lerch J, et al. Intellectual ability and cortical development in children and adolescents. *Nature*. 2006;440(7084): 676-679.
47. Westfall DR, Gejl AK, Tarp J, et al. Associations between aerobic fitness and cognitive control in adolescents. *Front Psychol*. 2018;9:1298.
48. Hwang J, Castelli DM, Gonzalez-Lima F. The positive cognitive impact of aerobic fitness is associated with peripheral inflammatory and brain-derived neurotrophic biomarkers in young adults. *Physiol Behav*. 2017;179:75-89.
49. Huang T, Tarp J, Domazet SL, et al. Associations of adiposity and aerobic fitness with executive function and math performance in danish adolescents. *J Pediatr*. 2015;167(4):810-815.
50. Chaddock L, Erickson KI, Prakash RS, et al. A neuroimaging investigation of the association between aerobic fitness, hippocampal volume, and memory performance in preadolescent children. *J Brain Res*. 2010;1358:172-183.
51. Tsai C-L, Chen F-C, Pan C-Y, Tseng Y-T. The neurocognitive performance of visuospatial attention in children with obesity. *Front Psychol*. 2016;7:1033.
52. Kamijo K, Khan NA, Pontifex MB, et al. The relation of adiposity to cognitive control and scholastic achievement in preadolescent children. *Obesity (Silver Spring)*. 2012a;20(12):2406-2411.
53. Mora-Gonzalez J, Esteban-Cornejo I, Cadenas-Sanchez C, et al. Physical fitness, physical activity, and the executive function in children with overweight and obesity. *J Pediatr*. 2019;208:50-56. e51.
54. Schwartz DH, Leonard G, Perron M, et al. Visceral fat is associated with lower executive functioning in adolescents. *Int J Obes (Lond)*. 2013;37(10):1336-1343.
55. Hillman CH, Pontifex MB, Raine LB, Castelli DM, Hall EE, Kramer AF. The effect of acute treadmill walking on cognitive control and academic achievement in preadolescent children. *Neuroscience*. 2009; 159(3):1044-1054.
56. Pontifex MB, Raine LB, Johnson CR, et al. Cardiorespiratory fitness and the flexible modulation of cognitive control in preadolescent children. *J Cogn Neurosci*. 2011;23(6):1332-1345.
57. Drollette ES, Scudder MR, Raine LB, et al. Acute exercise facilitates brain function and cognition in children who need it most: an ERP study of individual differences in inhibitory control capacity. *Dev Cogn Neurosci*. 2014;7:53-64.
58. Weber DR, Leonard MB, Zemel BS. Body composition analysis in the pediatric population. *Pediatr Endocrinol Rev*. 2012;10(1):130-139.
59. Micklesfield LK, Goedecke JH, Punyanitya M, Wilson KE, Kelly TLJO. Dual-energy X-ray performs as well as clinical computed tomography for the measurement of visceral fat. 2012;20(5):1109–1114.

How to cite this article: Logan NE, Raine LB, Drollette ES, et al. The differential relationship of an afterschool physical activity intervention on brain function and cognition in children with obesity and their normal weight peers. *Pediatric Obesity*. 2020;e12708. https://doi.org/10.1111/ijpo.12708