Obesity, Visceral Adipose Tissue, and Cognitive Function in Childhood

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Objectives To evaluate the effects of a 9-month physical activity intervention on changes in adiposity and cognitive control based on pretrial weight status (ie, healthy weight vs obese) in children.

Study design Participants included obese (n = 77) and matched healthy-weight (n = 77) preadolescents (8-9 years) who participated in a 9-month physical activity randomized controlled trial. Cognitive function was assessed with an inhibitory control task (modified flanker task).

Results After the 9-month physical activity intervention, participants exhibited a reduction in adiposity. In contrast, children in the waitlist-control condition, particularly children identified as obese pretrial, gained visceral adipose tissue ($P = .008$). Changes in visceral adipose tissue were related to changes in cognitive performance, such that the degree of reduction in visceral adipose tissue directly related to greater gains in inhibitory control, particularly among obese intervention participants (CI −0.14, −0.04; $P = .001$).

Conclusions Participation in a daily physical activity program not only reduces adiposity but also improves children's cognitive function as demonstrated by an inhibitory control task. Furthermore, these findings reveal that the benefits of physical activity to improvements in cognitive function are particularly evident among children who are obese. *(J Pediatr 2017;187:134-40).*

Trial registration ClinicalTrials.gov: NCT01334359 and NCT01619826.

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Evidence indicates the deleterious effects of obesity and excess visceral adipose tissue (VAT) may extend beyond metabolic dysregulation and may impact cognitive function and brain health, with larger effects observed for tasks requiring cognitive control. Cognitive control refers to higher-order mental operations implicated in the regulation of goal-directed behaviors.[1-4] One aspect of cognitive control is inhibition,[5] or the ability to suppress irrelevant task information in the environment and override a prepotent or impulsive response in favor of a correct response.[5]

There are only a few investigations into the effects of chronic physical activity (PA) on cognitive control in children.[6-8] Research in children suggests that a 9-month PA intervention results in increased aerobic fitness and maintenance of body mass index (BMI) and enhanced task performance during tasks requiring greater amounts of cognitive control.[8] However, changes in fitness or body composition may not be necessary for beneficial changes in cognitive control. In an intervention with children who were obese, researchers did not observe changes in fitness or BMI but did observe improvements in cognitive processes.[7] Together, these findings suggest that PA interventions may be cognitively beneficial to children who are obese. Furthermore, no previous randomized controlled trials have examined the influence of baseline weight status on PA-derived cognitive benefits in children. Elucidating the extent to which pre-existing weight status limits or enhances benefits is important because current PA recommendations are targeted toward all children. However, it is plausible that some subgroups (eg, children who are obese) may disproportionately benefit from the same dose of activity.

The purpose of this study was to examine the differential relationship of types of whole body fat (%Fat) as well central adiposity (VAT and subcutaneous abdominal adipose tissue [SAT]) on cognitive control, as well as the changes that occur as a result of a PA intervention in children. Furthermore, we aimed to examine

%Fat	Whole-body fat
BMI	Body mass index
PA	Physical activity
SAT	Subcutaneous abdominal adipose tissue
SES	Socioeconomic status
VAT	Visceral adipose tissue
VO_2max	Maximum rate of oxygen consumption

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changes in cognitive function based on baseline weight status. We hypothesized that VAT would be selectively and negatively related to children's cognitive function and that children who were obese who participated in the intervention would exhibit the greatest cognitive benefits from PA-induced changes in VAT.

Methods

A total of 407 children between the ages of 8 and 10 years old were recruited to participate in the FITKids (n = 212) (ClinicalTrials.gov: NCT01334359), and FITKids2 (n = 195) (ClinicalTrials.gov: NCT01619826) research trials; the present study includes only a subset of these children. 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 (Champaign, Illinois). Participants were administered the Kaufman Brief Intelligence Test[9] or the Woodcock-Johnson III.[10] Socioeconomic status (SES) was determined with a trichotomous index based on participation in free or reduced-price lunch program at school, the highest level of education obtained by the mother and father, and the number of parents who worked full time.[11] Exclusion criteria included the presence of neurologic disorders and physical disabilities and other factors, found elsewhere.[8,12] After these exclusionary criteria were applied, 382 children were included, 92 obese children completed pretesting, and 77 of these children also completed post-testing; thus, these 77 children who were obese (43: treatment group; 34: control group) were matched to 77 children of healthy weight based on treatment allocation and demographic variables, including sex, age, IQ, SES, and fitness.

Participants completed a modified version of the Eriksen flanker task[13] to assess inhibitory control. Congruent and incongruent trials required participants to respond based on the direction of the centrally presented stimuli. Congruent trials consisted of an array of 5 fish facing the same direction, whereas incongruent trials consisted of the 4 flanking fish facing the opposite direction of the target (middle) fish. In addition, stimulus compatibility was manipulated by varying the amount of inhibitory control needed to successfully execute the task. In the incompatible condition, the response mappings to each stimuli were reversed; for example, when the centrally presented fish faced right, a left button response was required. Research staff was blinded to treatment group, weight status, and experimental hypothesis. Additional details are described elsewhere.[8]

Adiposity measurements included BMI, %Fat, VAT, and SAT. The Centers for Disease Control and Prevention growth charts were used to determine individual BMI-for-age percentiles.[14] Whole-body and regional soft tissue were measured by dual-energy radiographic absorptiometry with a Hologic QDR 4500A bone densitometer (software version 13.4.2; Hologic, Bedford, Massachusetts). Central adiposity (ie, VAT, SAT) variables were generated from a 5-cm wide section placed across the abdomen just above the iliac crest at a level approximately coinciding with the fourth lumbar vertebrae. The details can be found elsewhere.[15]

Participants completed a maximum rate of oxygen consumption (VO_2max) test on a treadmill; the protocol is described elsewhere.[8] VO_2max relative to fat-free mass (ml/kg lean/min; VO_2max ff) was the primary fitness measure to isolate the influence of fat mass in the hierarchical regression modeling and was calculated with the use of absolute VO_2max and lean mass. Fat-free mass has been shown previously to be the primary contributor to aerobic capacity.[16]

The aforementioned measures were completed before and after randomization into a 9-month intervention/wait list control assignment. The intervention group received a 2-hour intervention (5 days/week for 9 months) based on the Child and Adolescent Trial for Cardiovascular Health curriculum, which is an evidence-based PA program that provides moderate-to-vigorous PA in a noncompetitive environment. Details of the intervention can be found elsewhere.[8,17] The control group was asked to maintain their regular after-school routine. They were not contacted again until post-testing.

Statistical Analyses

All statistical analyses were performed with SPSS 23 (IBM Corp, Armonk, New York) via a family-wise alpha threshold for all tests set at $P = .05$. Intervention analyses assessed group-wise differences over the course of 9 months. Analyses of adiposity (%Fat, VAT, SAT) were assessed by the use of separate 2 (weight status: healthy weight, obese) × 2 (group: intervention, control) × 2 (time: pretest, post-test) multivariate ANOVAs. Furthermore, change scores (Δ) were computed for fitness and adiposity measures by subtracting the pretest from the post-test measure. Follow-up analyses included univariate analyses on change scores for each adiposity measurement. Analyses of flanker accuracy and reaction time were assessed by the use of separate 2 (weight status: healthy weight, obese) × 2 (group: intervention, control) × 2 (time, pretest, post-test) × 2 (compatibility: compatible, incompatible) × 2 (congruency: congruent, incongruent) multivariate ANOVAs.

A second analytical approach used regression analyses to characterize relationships between the primary measures within and across groups. First, Pearson correlations (2-tailed) were used to assess bivariate relationships between changes in adiposity and changes in cognitive outcomes. Next, stepwise linear regressions were conducted across all participants to determine whether changes in %Fat, VAT, and SAT were associated with changes in flanker task performance. Finally, for significant relationships (ie, $P < .05$), additional analyses were conducted within each intervention group (intervention, control) and BMI group (healthy weight, control). In the first step, the dependent variables were regressed on significant demographic variables. Step 2 assessed Δ%Fat to account for changes in whole-body obesity. At step 3, ΔVAT and ΔSAT were inserted into the regression model to determine the contribution of changes in adiposity on changes in flanker performance. The change in R^2 values between steps was used to determine the contribution of these measures for explaining variance in the dependent variables of interest beyond that of

demographic variables. Note that because reaction time was not significantly associated with any variable of interest, only response accuracy data are reported in the results section.

Results

Participant demographics are presented in **Table I**. Children who were healthy weight and obese were matched for key demographic variables and did not differ in age, IQ, SES, or VO_2max, confirming efficacy of the participant matching procedure. As expected, children who were obese and healthy weight did differ in adiposity variables of %Fat, VAT, and SAT.

For aerobic fitness, the ANOVA revealed an effect of time, $P = .01$, with a significant increase from pretest (55.65 ± 0.58 ml/kg lean/min) to post-test (56.88 ± 0.57 ml/kg lean/min). There were no effects of treatment group or BMI group. The univariate change score analysis revealed no significant effects.

In terms of adiposity outcomes, the ANOVA revealed an effect of time, $P = .01$, with %Fat decreasing from pretest (34.09 ± 0.38%) to post-test (33.65 ± 0.41%) and BMI group, $P \leq .001$, with children of healthy weight having less %Fat (27.19 ± 0.54%) than children who were obese (40.55 ± 0.55%). These effects were superseded by interactions of treatment × time, $P = .001$, with only the treatment group decreasing %Fat from pretest (34.31 ± 0.82%) to post-test (33.26 ± 0.82%), $t(84) = 4.435$, $P \leq .001$. In contrast, the control group maintained %Fat from pretest (33.81 ± 1.08%) to post-test (33.97 ± 1.11%), $P = .53$. There was an interaction of treatment × BMI group, $P = .009$; however, decomposition of this interaction revealed no significant effects. The univariate change score analysis revealed a main effect of treatment, $P = .001$, with children in the treatment group decreasing their %Fat (–1.05 ± 0.24%) and children in the control group increasing their %Fat (0.16 ± 0.26%).

The ANOVA revealed an effect of time, $P = .002$, with VAT lower at pretest (219.16 ± 7.24 g) compared with post-test (229.36 ± 7.90 g) and BMI group, $P = .001$, with children of healthy weight (120.08 ± 10.43 g) having lower VAT compared with children who were obese (328.44 ± 10.48 g). These effects were superseded by interactions of treatment × time, $P = .002$, and treatment × BMI group, $P = .04$, which were superseded by a 3-way interaction of treatment × BMI group × time, $P = .05$, decomposition of the 3-way interaction assessed treatment × time within each BMI group, and revealed a significant interaction within the obese group, $P = .008$; an increase in VAT was only observed for participants who were obese in the control group from pretest (332.59 ± 120.25 g) to post-test (365.83 ± 112.06 g), $P \leq .001$. The univariate change score analyses revealed an effect of treatment, $P = .002$, with children in the treatment group decreasing VAT (–0.16 ± 4.41 g) and children in the control group increasing VAT (20.54 ± 4.93 g). There was also an interaction of treatment × BMI group, $P = .04$; the obese intervention group lost VAT (–0.62 ± 9.36 g) and the obese control group gained VAT (33.24 ± 7.49 g), $P = .008$. Furthermore, the control participants who were of healthy weight (7.85 ± 3.60 g) gained less VAT than the control participants who were obese (33.23 ± 7.49 g), $P = .004$.

The ANOVA revealed an effect of time, $P < .001$, with increases in SAT from pretest (1025.24 ± 32.32 g) to post-test (1072.97 ± 35.68 g) and an effect of BMI group, $P \leq .001$, with participants of healthy weight having less SAT (546.15 ± 47.13 g) than participants who were obese (1552.06 ± 47.38 g). These main effects were superseded by interactions of treatment × BMI group, $P = .03$, with intervention participants who were obese having less SAT (1416. 69 ± 87.36 g) than control participants who were obese (1687.42 ± 73.68 g), $P = .02$; an interaction of treatment × time, $P = .03$, with increases in SAT from pretest (1074.16 ± 78.11 g) to post-test (1150.36 ± 87.82 g) only in the control group, $P \leq .001$, and an interaction of BMI group × time, $P = .05$. Decomposition of this interaction revealed that at pretest, children who were obese had greater SAT (1509.29 ± 57.19g) than children of healthy weight (536.64 ± 30.62 g), $P = .001$, and at post-test, this pattern remained for children who were obese (1571.93 ± 65.00 g) and children of healthy weight (557.78 ± 33.34 g), $P = .001$. The univariate change score analyses revealed an effect of treatment, $P = .03$, with the treatment group gaining less SAT (19.28 ± 17.36 g) than the control group (76.19 ± 19.41 g), and an effect of BMI group, $P = .05$, with children of healthy weight gaining less SAT (22.04 ± 18.36 g) than children who were obese (73.43 ± 18.46 g).

Evaluation of cognitive outcomes included accuracy. The ANOVA revealed effects of time, $P \leq .001$, with greater accuracy at post-test (80.42 ± 0.89%) relative to pretest (75.02 ± 0.95%), compatibility, $P \leq .001$, with increased accuracy in the compatible condition (79.33 ± 0.74%) relative to the incompatible condition (76.11 ± 0.74%), and congruency, $P \leq .001$, with greater accuracy for congruent (79.98 ± 0.79%) relative to incongruent (75.45 ± 0.83%) trials. Furthermore, a 4-way interaction of treatment × BMI group × time × compatibility was observed, $P = .048$. The interaction was decomposed by assessing treatment × BMI group × compatibility at each time point and revealed no significant interactions, P values ≥ 0.14. Additional attempts to deconstruct this 4-way interaction in a meaningful manner did not yield significant findings.

Table I. Participant demographics and baseline fitness and adiposity characteristics

Demographics	Matched healthy weight	Obese	Entire sample
n	77 (49 female)	77 (49 female)	154 (94 female)
Age, y	8.88 ± 0.08	8.81 ± 0.06	8.84 ± 0.05
IQ	105.81 ± 1.51	108.14 ± 1.38	106.97 ± 1.03
SES	1.68 ± 0.08	1.73 ± 0.09	1.70 ± 0.061
VO_2max ff, ml/ kg lean/ min	56.63 ± 0.81	54.35 ± 0.86	55.49 ± 0.59
VAT,* g	119.40 ± 5.22	320.67 ± 13.45	220.04 ± 10.86
SAT,* g	536.65 ± 30.62	1509.29 ± 57.19	1022.97 ± 50.90
Whole body %Fat*	27.51 ± 0.54	40.78 ± 0.54	34.14 ± 0.66

VO_2max ff, fat free maximal oxygen volume.
*$P \leq .05$.

Table V. Regression analyses for ΔVAT predicting Δcompatible congruent and incongruent accuracy

	Congruent				Incongruent			
Steps	***B***	***SE B***	***β***	***t***	***B***	***SE B***	***β***	***t***
Step 1								
Treatment	−1.45	1.73	−0.05	−0.84	0.85	2.02	0.03	0.42
BMI group	0.49	1.54	0.04	0.32	0.64	1.79	0.05	0.36
Pretest compatible congruent accuracy	−0.71	0.07	−0.62	−9.54*	−0.59	0.09	−0.48	−6.54*
Pretest %Fat	−0.09	0.21	−0.05	−0.42	−0.08	0.25	−0.05	−0.32
Pretest VAT	0	0.01	0.04	0.36	0	0.01	−0.02	−0.12
Step 2								
Δ%Fat	−0.68	0.39	−0.11	−1.73	−0.24	0.47	−0.04	−0.51
Step 3								
ΔVAT	−0.04	0.02	−0.12	−1.55	−0.03	0.03	−0.08	−0.86

*$P \leq .05$.

Correlations were conducted across the entire sample for participant demographics and changes in cognitive control and adiposity (**Table II**; available at www.jpeds.com). Treatment group (intervention, control) and BMI group (healthy weight, obese) were entered into step 1 of the regression analyses. To account for pretest adiposity and cognitive control, the pretest values for each of the measures (VAT, SAT, %Fat, task performance) also were entered into step 1. Demographic variables that were correlated significantly with changes in each specific cognitive measure were included in step 1. To determine the unique contribution of specific types of fat beyond overall obesity, changes in %Fat were entered into step 2. Changes in adiposity variables (ΔVAT and ΔSAT) were entered into step 3. Each regression was first performed with all children, and subsequent regressions were conducted within each treatment and BMI group if the initial regression was significant. Regression analyses for Δflanker accuracy and ΔSAT were nonsignificant in all cases and may be found in **Tables III** and **IV** (available at www.jpeds.com).

The step 1 regression analysis for Δcompatible congruent accuracy was significant, adjusted $R^2 = 0.39$, $P \leq .001$. Although Step 2 was also significant, $R^2 = 0.40$, $P \leq .001$, the addition of Δ%Fat did not account for an incremental amount of variance in Δcompatible congruent accuracy beyond associated descriptive variables, $\beta = -0.11$, $P = .09$. Step 3 was also significant, $R^2 = 0.40$, $P \leq .001$; however, the addition of ΔVAT ($\beta = -0.12$, $P = .12$) did not account for an incremental amount of variance in Δcompatible congruent accuracy beyond associated descriptive variables (**Table V**).

The step 1 regression analysis for Δcompatible incongruent accuracy was significant, adjusted $R^2 = 0.21$, $P \leq .001$. Although step 2 was also significant, $R^2 = 0.21$, $P \leq .001$, the addition of Δ%Fat did not account for an incremental amount of variance in Δcompatible incongruent accuracy beyond associated descriptive variables, $\beta = -0.04$, $P = .61$. Step 3 was also significant, $R^2 = 0.21$, $P \leq .001$; however, the addition of ΔVAT ($\beta = -0.08$, $P = .39$) did not account for an incremental amount of variance in Δcompatible incongruent accuracy beyond associated descriptive variables (**Table V**).

The step 1 regression analysis for Δincompatible congruent accuracy was significant, adjusted $R^2 = 0.38$, $P \leq .001$. Although step 2 was also significant, $R^2 = 0.38$, $P \leq .001$, the addition of Δ%Fat did not account for an incremental amount of variance in Δincompatible congruent accuracy beyond associated descriptive variables, $\beta = -0.03$, $P = .64$. Step 3 was also significant, $R^2 = 0.42$, $P \leq .001$, with the addition of ΔVAT accounting for an incremental amount of variance in Δincompatible congruent accuracy beyond associated descriptive variables, $\beta = -0.26$ (**Table VI** and **Figure**; **Figure** available at www.jpeds.com).

Next, this regression was performed separately for each treatment and BMI group. Three groups (all but obese controls) demonstrated a significant step 1 effect, adjusted R^2 values ≥ 0.40, P values ≤ .001. Although step 2 was significant, $R^{2'}$ values ≥ 0.40,

Table VI. Regression analyses for ΔVAT predicting Δincompatible congruent accuracy

	All				Obese intervention			
Steps	***B***	***SE B***	***β***	***t***	***B***	***SE B***	***β***	***t***
Step 1								
Treatment	−2.72	1.86	−0.09	−1.46				
BMI group	−0.08	1.66	−0.01	−0.05				
Pretest incompatible congruent accuracy	−0.60	0.06	−0.63	−9.76	−0.62	0.11	−0.65	−5.888*
Pretest %Fat	0.19	0.23	0.11	0.85	0.40	0.51	0.13	0.77
Pretest VAT	−0.01	0.01	−0.08	−0.71	−0.04	0.02	−0.35	−2.098*
Step 2								
Δ%Fat	−0.20	0.44	−0.03	−0.46	−0.60	0.61	−0.11	−0.99
Step 3								
ΔVAT	−0.09	0.03	−0.26	−3.38*	−0.08	0.03	−0.36	−2.578*

*$P \leq .05$.

Table VII. Regression analyses for ΔVAT predicting Δincompatible incongruent accuracy

	All				Obese intervention			
Steps	***B***	***SE B***	***β***	***t***	***B***	***SE B***	***β***	***t***
Step 1								
Trt	–2.88	2.01	–0.09	–1.43				
BMI class	0.13	1.83	0.01	0.07				
Pretest incompatible incongruent response accuracy	–0.64	0.07	–0.64	–9.25*	–0.75	0.09	–0.77	–7.96*
Age	1.89	1.81	0.07	1.04	0.82	2.95	0.03	0.28
Pretest %Fat	–0.01	0.25	–0.01	–0.05	–0.07	0.48	–0.02	–0.14
Pretest VAT	0.00	0.01	–0.03	–0.31	–0.03	0.02	–0.24	–1.70
Step 2								
Δ%Fat	0.20	0.48	0.03	0.41	–0.39	0.57	–0.06	–0.68
Step 3								
ΔVAT	–0.08	0.03	–0.23	–2.92*	–0.08	0.03	–0.32	–2.70*

Trt, treatment.
*$P \leq .05$.

$P \leq .001$, the addition of Δ%Fat did not account for an incremental amount of variance in Δincompatible congruent accuracy beyond associated descriptive variables, β values ≤ 0.14, P values ≥ .20. For step 3, only the obese intervention group showed a significant relationship, adjusted $R^2 = 0.58$, $P \leq .001$, such that greater ΔVAT was associated with smaller Δincompatible congruent accuracy, with ΔVAT accounting for an incremental amount of variance in Δcompatible congruent accuracy beyond associated descriptive variables, $\beta = –0.36$, $P = .01$ (**Table VI** and **Figure**).

The step 1 regression analysis for Δincompatible incongruent accuracy was significant, adjusted $R^2 = 0.38$, $P \leq .001$. Although step 2 was significant, $R^2 = 0.37$, $P \leq .001$, the addition of Δ%Fat did not account for an incremental amount of variance in Δincompatible incongruent accuracy beyond associated descriptive variables, $\beta = –0.03$, $P = .64$. Step 3 was also significant, $R^2 = 0.41$, $P \leq .001$, with the addition of ΔVAT accounting for an incremental amount of variance in Δincompatible incongruent accuracy beyond associated descriptive variables, $\beta = –0.23$, $P = .004$ (**Table VII** and **Figure**).

Next, this regression was conducted separately for each treatment and BMI group. Three groups (all but obese controls) demonstrated a significant step 1 effect, adjusted R^2 values ≥ 0.30, *P* values ≤ .006. Although step 2 was significant, R^2 values ≥ 0.33, $P = .005$, the addition of Δ%Fat did not account for an incremental amount of variance in Δincompatible incongruent accuracy beyond associated descriptive variables, β values ≤ 0.25, *P* values ≥ .14. For step 3, only the obese intervention group showed a significant relationship, adjusted $R^2 = 0.69$, $P \leq .001$, such that greater ΔVAT was associated with smaller Δincompatible incongruent accuracy, with ΔVAT accounting for an incremental amount of variance in Δincompatible incongruent accuracy beyond associated descriptive variables, $\beta = –0.32$, $P = .01$ (**Table VII** and **Figure**).

Discussion

Previous research has established cross-sectional links between increased adiposity and measures of cognitive control in preadolescent children.[18-22] Our study extends this by establishing the relationships between changes in adiposity and cognitive control using a 9-month PA randomized controlled trial. Furthermore, the current work identified the impact of baseline weight status as a predictor of potential benefits derived from participation in a 9-month PA program. Indeed, our findings revealed that participation in the PA program particularly was beneficial for children who were obese at baseline, indicated by greater reductions in fat mass. In addition, the reductions in VAT were associated with greater gains in cognitive performance, independent of changes in whole-body fat, particularly among intervention participants who were obese.

This study examined the extent to which a PA intervention influenced changes in adiposity and subsequent improvements in inhibition across children who were healthy weight and obese. Smaller changes in VAT, a metabolically pathogenic fat depot, were associated with larger changes in incompatible flanker performance, the task requiring greater upregulation of cognitive control. These findings suggest that changes in VAT, independent of whole-body adiposity, have a selective impact on children's cognitive control. These findings are critical, as they suggest that changes in VAT may be a link between changes in cognitive control after a PA intervention. Cognitive control has a critical role in planning and organizing goal-directed thoughts and actions; thus, effective cognitive control is essential for activities of everyday life, including the prevention of impulsive behavior, or resisting the temptation to overeat.[23,24] These data further suggest that children who are most in need of PA intervention also benefit the most in terms of both VAT reduction and cognitive gains.

These findings are in concert with previous data that have shown PA to exhibit a greater influence on cognitively demanding tasks with larger inhibitory control requirements.[25-30] We extend the knowledge in this area by demonstrating a beneficial effect on cognitive control performance after PA intervention.[8,31] Among sedentary children who are obese, previous research suggests a beneficial effect of PA on cognitive control.[6,7] Previous research indicates that PA has been shown to increase concentration, memory, self-discipline, and classroom behavior.[32,33] In addition, regular PA participation has

been linked to enhancement of brain function and cognition.[32,33] Although the present study and others[6-8] highlight the importance of PA interventions for optimizing brain health during preadolescence, continued research is needed to better understand the potential causal relationship and mechanisms underlying PA effects on cognitive control in children. Therefore, the present study, along with others, highlights the importance of PA interventions for optimizing brain health during preadolescence.

This preliminary study has limitations. Differences in factors such as diet, sleep, and mental health (eg, depression, anxiety) have been shown to differ between children who are of healthy weight and are obese. For example, a relationship has been observed between sleep duration and disruption with obesity.[34,35] In addition, obstructive sleep apnea also has been related to deficits in cognitive control.[36] Thus, it is possible that these health factors may account, in part, for the findings observed.[35,37,38] Future work should account for these other health factors to better understand the relationship between obesity and cognitive control.

The results from this study suggest a beneficial effect PA, particularly in children who are obese, in terms of improving adiposity and cognitive control, especially in demanding cognitive tasks. The current findings contribute to a greater understanding of the relationship between PA and cognitive function, indicating the beneficial impact of PA on aspects of cognitive control requiring extensive amounts of inhibition. ■

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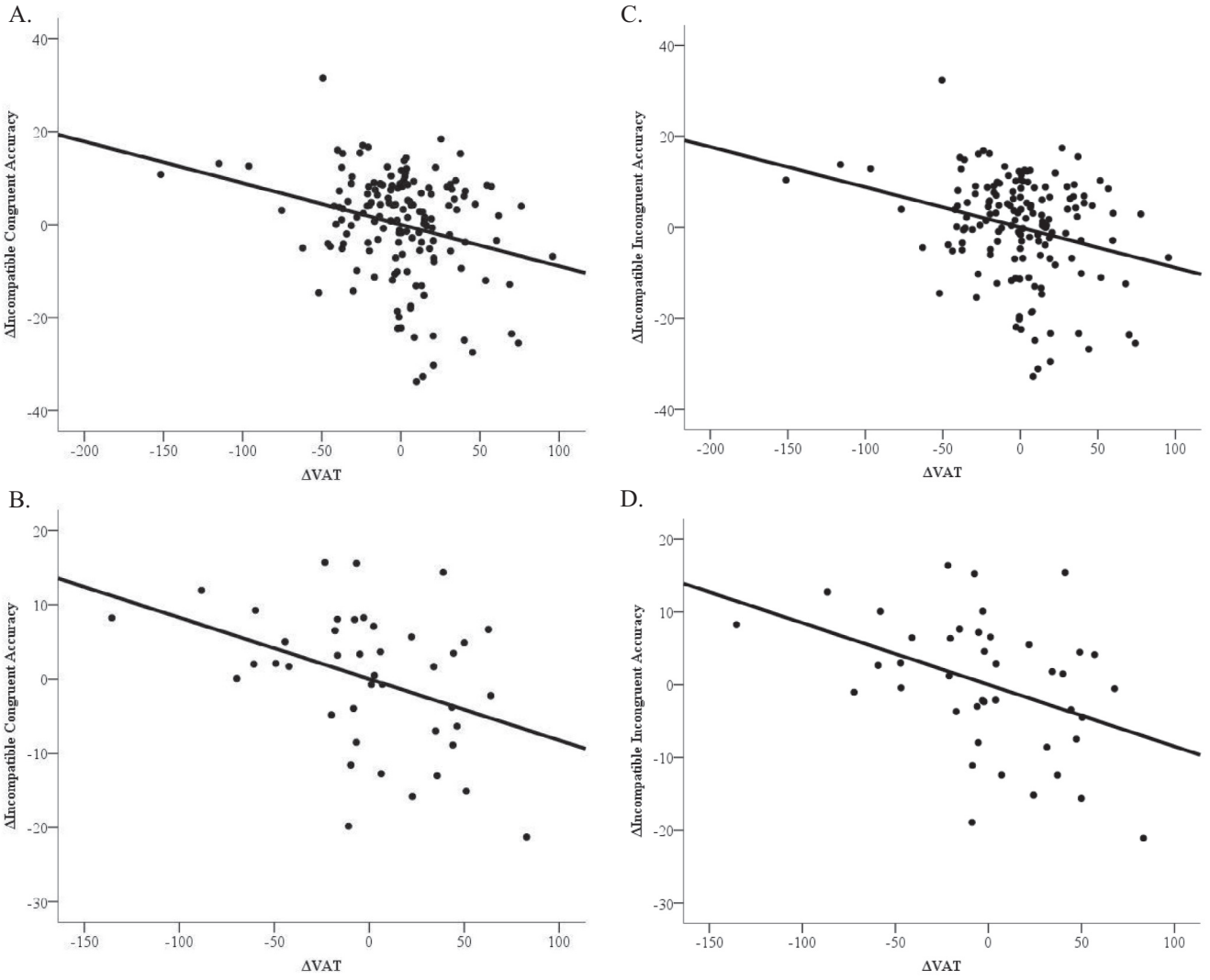

Figure. Partial regression plots for ΔVAT predicting Δincompatible congruent and incongruent accuracy. **A,** All participants: ΔVAT predicting Δincompatible congruent. **B,** Intervention participants who were obese: ΔVAT predicting Δincompatible congruent. **C,** All participants: ΔVAT predicting Δincompatible incongruent. **D,** Intervention participants who were obese: ΔVAT predicting Δincompatible incongruent.

Table II. Correlations between demographics and changes in cognition and adiposity

Demographics	ΔCompatible congruent accuracy	ΔCompatible incongruent accuracy	ΔIncompatible congruent accuracy	ΔIncompatible incongruent accuracy	ΔCompatible congruent reaction time	ΔCompatible incongruent reaction time	ΔIncompatible congruent reaction time	ΔIncompatible incongruent reaction time	Δ%Fat	ΔVAT	ΔSAT
Age	–0.05	–0.08	–0.16	–0.16*	–0.06	–0.07	–0.09	–0.08	–0.07	–0.01	–0.03
Sex	0.04	0.08	0.00	0.07	0.09	0.15	0.12	0.15	–0.07	–0.04	–0.06
IQ	–0.02	0.02	–0.02	–0.14	0.05	0.06	0.10	0.00	0.00	0.08	0.00
SES	–0.03	–0.02	–0.05	–0.03	0.06	0.04	0.03	0.00	–0.01	–0.04	–0.11
Pretest VO_2 FF	–0.15	–0.06	0.00	–0.01	–0.08	–0.07	–0.07	–0.05	–0.16*	–0.10	–0.19*

VO_2max ff, fat free maximal oxygen volume.
*Correlation is significant at the 0.05 level (2-tailed).

Table III. Regression analyses for ΔSAT predicting Δcompatible congruent and incongruent accuracy

	Congruent				Incongruent			
Steps	***B***	***SE B***	***β***	***t***	***B***	***SE B***	***β***	***t***
Step 1								
Treatment	−1.43	1.81	−0.05	−0.79	0.97	2.1	0.04	0.46
BMI group	0.63	1.48	0.05	0.43	0.61	1.73	0.04	0.35
Pretest compatible congruent accuracy	−0.71	0.08	−0.62	−9.48*	−0.59	0.09	−0.49	−6.53*
Pretest %Fat	−0.05	0.31	−0.03	−0.16	−0.03	0.37	−0.02	−0.09
Pretest SAT	0	0	0	0	0	0	−0.04	−0.21
Step 2								
Δ%Fat	−0.7	0.4	−0.12	−1.75	−0.23	0.48	−0.04	−0.48
Step 3								
ΔSAT	0	0.01	−0.05	−0.59	0	0.01	−0.05	−0.48

*$P \leq .05$.
ΔCompatible congruent accuracy and ΔSAT Step 1 adjusted $R^2 = 0.39$, $P \leq .001$; step 2 $\Delta R^2 = 0.01$, $P = .09$; step 3 $\Delta R^2 = 0.01$, $P = .56$. ΔCompatible incongruent accuracy and ΔSAT step 1 adjusted $R^2 = 0.21$, $P \leq .001$; step 2 was also significant, $\Delta R^2 = 0.01$, $P = .63$; step 3 $\Delta R^2 = 0.01$, $P = .63$.

Table IV. Regression analyses for ΔSAT predicting Δincompatible congruent and incongruent accuracy

	Congruent				Incongruent			
Steps	***B***	***SE B***	***β***	***t***	***B***	***SE B***	***β***	***t***
Step 1								
Treatment	−1.49	1.9	−0.05	−0.78	−1.13	2.03	−0.04	−0.56
BMI group	−0.12	1.57	−0.01	−0.08	0.43	1.71	0.03	0.25
Pretest incompatible congruent accuracy	−0.61	0.06	−0.64	−10.11*	−0.67	0.07	−0.66	−9.85*
Pretest %Fat	0.75	0.33	0.42	2.28*	2.25	1.75	0.09	1.29
Pretest SAT	−0.01	0	−0.4	−2.40*	0.82	0.35	0.43	2.34*
					−0.01	0	−0.52	−3.09*
Step 2								
Δ%Fat	−0.03	0.44	0	−0.07	0.44	0.47	0.06	0.95
Step 3								
ΔSAT	−0.01	0.01	−0.12	−1.4	−.006	.008	−.066	−.781

*$P \leq .05$.
ΔIncompatible congruent accuracy and ΔSAT step 1 adjusted $R^2 = 0.40$, $P \leq .001$; step 2 $\Delta R^2 = 0.01$, $P = .94$; step 3 $\Delta R^2 = 0.02$, $P = .16$. ΔIncompatible incongruent accuracy and ΔSAT step 1 adjusted $R^2 = 0.42$, $P \leq .001$; step 2 $\Delta R^2 = 0.01$, $P = .35$; step 3 $\Delta R^2 = 0.01$, $P = .44$.