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Obesity, Visceral Adipose Tissue, and Cognitive Function in Childhood

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Abstract

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

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

Results—Following 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 at pre-trial, gained visceral adipose tissue (95% confidence interval (CI) –58.58, –9.14; $p=0.008$). 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; $p=0.001$).

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. Further, these findings reveal that the benefits of physical activity to improvements in cognitive function are particularly evident among obese children.

Keywords

inhibitory control; physical activity; adiposity

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Trial registration [ClinicalTrials.gov](https://clinicaltrials.gov): NCT01334359 and NCT01619826

The authors declare no conflicts of interest.

Evidence indicates the deleterious effects of 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 (7–10). One aspect of cognitive control is inhibition (11), or the ability to suppress irrelevant task information in the environment, and over-ride a prepotent or impulsive response in favor of a correct response (11).

There are only a few investigations into the effects of chronic PA on cognitive control in children (21–23). Research in children suggests that a 9-month PA intervention results in increased aerobic fitness and maintenance of BMI; and enhanced task performance during tasks requiring greater amounts of cognitive control (24). However, changes in fitness or body composition may not be necessary for beneficial changes in cognitive control. In an intervention with obese children, researchers did not observe changes in fitness or BMI, but observed improvements in cognitive processes (22). Together, these findings suggest that PA interventions may be cognitively beneficial to obese children. 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 towards all children. However, it is plausible that some subgroups (e.g., obese children) 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 SAT) on cognitive control, as well as the changes that occur as a result of a PA intervention in children. Further, we aimed to examine 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 obese children who participated in the intervention would exhibit the greatest cognitive benefits from PA-induced changes in VAT.

Methods

407 children between the ages of 8 and 10 years old were recruited to participate in the FITKids (n=212) ([ClinicalTrials.gov](https://clinicaltrials.gov/ct2/show/study/NCT01334359): NCT01334359), and FITKids2 (n=195) ([ClinicalTrials.gov](https://clinicaltrials.gov/ct2/show/study/NCT01619826): 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 and Illinois at Urbana-Champaign. Participants were administered the Kaufman Brief Intelligence Test (K-BIT)(25) or the Woodcock Johnson (WJ III) (26). Socioeconomic status (SES) was determined using 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 (27). Exclusion criteria included the presence of neurological disorders and physical disabilities and other factors, found elsewhere (23,28). After these exclusionary criteria were applied, 382 children were included, 92 obese children completed pre-testing, and 77 of these children also completed post-testing, thus, these 77 obese children (43: treatment group; 34: control group) were

matched to 77 healthy weight children based on treatment allocation and demographic variables, including sex, age, IQ, SES, and fitness.

Participants completed a modified version of the Eriksen flanker task (29) 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 five fish facing the same direction, while incongruent trials consisted of the four flanking fish facing the opposite direction of the target (middle) fish. In addition, stimulus compatibility was manipulated, varying the amount 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 (23).

Adiposity measurements included BMI, %Fat, VAT, and SAT. The Centers for Disease Control growth charts were used to determine individual BMI-for-age percentiles (30). Whole body and regional soft tissue were measured by DXA using a Hologic Discovery bone densitometer (software version 12.7.3; Hologic, Bedford, Ma). Central adiposity (i.e., VAT, SAT) variables were generated from a 5cm wide section placed across the abdomen just above the iliac crest at a level approximately coinciding with the 4th lumbar vertebrae. The details can be found elsewhere (31).

Participants completed a VO_2max test on a treadmill; the protocol is described elsewhere (23). VO_2max relative to fat-free mass (ml/kg lean/min) was the primary fitness measure to isolate the influence of fat mass in the hierarchical regression modelling, and was calculated using absolute VO_2max and lean mass. Fat-free mass has been previously shown to be the primary contributor to aerobic capacity (32).

The above measures were completed prior to and following randomization into 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 (CATCH) curriculum, which is an evidence based physical activity program that provides moderate to vigorous physical activity in a non-competitive environment. Details of the intervention can be found elsewhere (20,23). 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, Armonk, New York) using a family-wise alpha threshold for all tests set at $p=0.05$. Intervention analyses assessed group wise differences over the course of 9 months. Analyses of adiposity (%Fat, VAT, SAT) were assessed using separate 2 (Weight status: healthy weight, obese) $\times$ 2 (Group: Intervention, control) $\times$ 2 (Time: Pre-Test, Post-test) MANOVAs. Furthermore, change scores () were computed for fitness and adiposity measures by subtracting the pre-test from the post-test measure. Follow up analyses included univariate analyses on change scores for each adiposity measurement. Analyses of flanker accuracy and RT were assessed using separate 2

(Weight Status: healthy weight, obese) $\times$ 2 (Group: Intervention, control) $\times$ 2 (Time, Pre-Test, Post-Test) $\times$ 2 (Compatibility: compatible, incompatible) $\times$ 2 (Congruency: congruent, incongruent) MANOVAs.

A second analytical approach employed regression analyses to characterize relationships between the primary measures within and across groups. First, Pearson correlations (two-tailed) assessed bivariate relationships between changes in adiposity and changes in cognitive outcomes. Next, stepwise linear regressions were conducted across all participants to determine if changes in %Fat, VAT, and SAT were associated with changes in flanker task performance. Finally, for significant relationships (i.e., $p < 0.05$), 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 %Fat to account for changes in whole body obesity. At step 3, VAT and SAT 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 RT 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. Healthy weight and obese children were matched for key demographic variables and did not differ in age, IQ, SES, or VO₂max, confirming efficacy of the participant matching procedure. As expected, obese and healthy weight children did differ in adiposity variables of %Fat, VAT, and SAT.

The ANOVA revealed an effect of Time, $p = 0.01$, with a significant increase from pre-test (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.

The ANOVA revealed an effect of Time, $p = 0.01$, with %Fat decreasing from pre-test ($34.09 \pm 0.38\%$) to post-test ($33.65 \pm 0.41\%$); and BMI group, $p = 0.001$, with healthy weight children having less %Fat ($27.19 \pm 0.54\%$) than obese children ($40.55 \pm 0.55\%$). These effects were superseded by interactions of Treatment $\times$ Time, $p = 0.001$, with only the Treatment group decreasing %Fat from pre-test ($34.31 \pm 0.82\%$) to post-test ($33.26 \pm 0.82\%$), $t(84) = 4.435$, $p = 0.001$. In contrast, the control group maintained %Fat from pre-test ($33.81 \pm 1.08\%$) to post-test ($33.97 \pm 1.11\%$), $p = 0.53$. There was an interaction of Treatment $\times$ BMI group, $p = 0.009$, however, decomposition of this interaction revealed no significant effects. The univariate change score analysis revealed a main effect of Treatment, $p = 0.001$, with children in the Treatment group decreasing %Fat ($-1.05 \pm 0.24\%$) and children in the control group increasing %Fat ($0.16 \pm 0.26\%$).

The ANOVA revealed an effect of Time, $p = 0.002$, with VAT lower at pre-test (219.16 ± 7.24 g) compared with post-test (229.36 ± 7.90 g); and BMI group, $p = 0.001$, with

healthy weight children ($120.08 \pm 10.43\text{g}$) having lower VAT compared with obese children ($328.44 \pm 10.48\text{g}$). These effects were superseded by interactions of Treatment $\times$ Time, $p=0.002$, and Treatment $\times$ BMI group, $p=0.04$, which were superseded by a 3-way interaction of Treatment $\times$ BMI group $\times$ Time, $p=0.05$. Decomposition of the 3-way interaction assessed Treatment $\times$ Time within each BMI group, and revealed a significant interaction within the obese group, $p=0.008$; an increase in VAT was only observed for obese participants in the control group from pre-test ($332.59 \pm 120.25\text{g}$) to post-test ($365.83 \pm 112.06\text{g}$), $p=0.001$. The univariate change score analyses revealed an effect of Treatment, $p=0.002$, with children in the treatment group decreasing VAT ($-0.16 \pm 4.41\text{g}$) and children in the control group increasing VAT ($20.54 \pm 4.93\text{g}$). There was also an interaction of Treatment $\times$ BMI group, $p=0.04$; the obese intervention group lost VAT ($-0.62 \pm 9.36\text{g}$) and the obese control group gained VAT ($33.24 \pm 7.49\text{g}$), $p=0.008$. Further, the healthy weight control participants ($7.85 \pm 3.60\text{g}$) gained less VAT than the obese control participants ($33.23 \pm 7.49\text{g}$), $p=0.004$.

The ANOVA revealed an effect of Time, $p < .001$, with increases from pre-test ($1025.24 \pm 32.32\text{g}$) to post-test ($1072.97 \pm 35.68\text{g}$); and an effect of BMI group, $p=0.001$, with healthy weight participants having less SAT ($546.15 \pm 47.13\text{g}$) than obese participants ($1552.06 \pm 47.38\text{g}$). These main effects were superseded by interactions of Treatment $\times$ BMI group, $p=0.03$, with obese intervention participants having less SAT ($1416.69 \pm 87.36\text{g}$) than obese control participants ($1687.42 \pm 73.68\text{g}$), $p=0.02$; an interaction of Treatment $\times$ Time, $p=0.03$, with increases in SAT from pre-test ($1074.16 \pm 78.11\text{g}$) to post-test ($1150.36 \pm 87.82\text{g}$) only in the control group, $p=0.001$; and an interaction of BMI group $\times$ Time, $p=0.05$. Decomposition of this interaction revealed that at pre-test, obese children had greater SAT ($1509.29 \pm 57.19\text{g}$) than healthy weight children ($536.64 \pm 30.62\text{g}$), $p=0.001$, and at post-test, this pattern remained for obese ($1571.93 \pm 65.00\text{g}$) and healthy weight ($557.78 \pm 33.34\text{g}$) children, $p=0.001$. The univariate change score analyses revealed an effect of Treatment, $p=0.03$, with the treatment group gaining less SAT ($19.28 \pm 17.36\text{g}$) than the control group ($76.19 \pm 19.41\text{g}$); and an effect of BMI group, $p=0.05$, with healthy weight children gaining less SAT ($22.04 \pm 18.36\text{g}$) than obese children ($73.43 \pm 18.46\text{g}$).

Evaluation of cognitive outcomes included accuracy. The ANOVA revealed effects of Time, $p=0.001$, with greater accuracy at post-test ($80.42 \pm 0.89\%$) relative to pre-test ($75.02 \pm 0.95\%$), Compatibility, $p=0.001$, with increased accuracy in the compatible condition ($79.33 \pm 0.74\%$) relative to the incompatible condition ($76.11 \pm 0.74\%$), and Congruency, $p=0.001$, with higher accuracy for congruent ($79.98 \pm 0.79\%$) relative to incongruent ($75.45 \pm 0.83\%$) trials. Further, a 4-way interaction of Treatment $\times$ BMI group $\times$ Time $\times$ Compatibility was observed, $p=0.048$. The interaction was decomposed by assessing Treatment $\times$ BMI group $\times$ Compatibility at each time point and revealed no significant interactions, $ps > 0.14$. Additional attempts to deconstruct this 4-way interaction in a meaningful manner did not yield significant findings.

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. In order to account for pre-test adiposity and cognitive control, the pre-test values for each of the measures (VAT, SAT, %Fat, task performance) were also entered into Step 1. Demographic variables that were significantly correlated with changes in each specific cognitive measure were included in Step 1 (Table I). In order 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 (VAT and SAT) 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 flanker accuracy and SAT were non-significant in all cases and may be found in Tables VI and VII (available at www.jpeds.com).

The Step 1 regression analysis for compatible congruent accuracy was significant, adjusted $R^2=0.39$, $p < 0.001$. Although Step 2 was also significant, $R^2=0.40$, $p < 0.001$, the addition of %Fat did not account for an incremental amount of variance in compatible congruent accuracy beyond associated descriptive variables, $\beta=-0.11$, $p=0.09$. Step 3 was also significant, $R^2=0.40$, $p < 0.001$, however the addition of VAT ($\beta=-0.12$, $p=0.12$) did not account for an incremental amount of variance in compatible congruent accuracy beyond associated descriptive variables (Table III).

The Step 1 regression analysis for compatible incongruent accuracy was significant, adjusted $R^2=0.21$, $p < 0.001$. Although Step 2 was also significant, $R^2=0.21$, $p < 0.001$, the addition of %Fat did not account for an incremental amount of variance in compatible incongruent accuracy beyond associated descriptive variables, $\beta=-0.04$, $p=0.61$. Step 3 was also significant, $R^2=0.21$, $p < 0.001$, however the addition of VAT ($\beta=-0.08$, $p=0.39$) did not account for an incremental amount of variance in compatible incongruent accuracy beyond associated descriptive variables (Table III).

The Step 1 regression analysis for incompatible congruent accuracy was significant, adjusted $R^2=0.38$, $p < 0.001$. Although Step 2 was also significant, $R^2=0.38$, $p < 0.001$, the addition of %Fat did not account for an incremental amount of variance in incompatible congruent accuracy beyond associated descriptive variables, $\beta=-0.03$, $p=0.64$. Step 3 was also significant, $R^2=0.42$, $p < 0.001$, with the addition of VAT accounting for an incremental amount of variance in incompatible congruent accuracy beyond associated descriptive variables, $\beta=-0.26$ (Table IV 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 's > 0.40 , p 's < 0.001 . Although Step 2 was significant, R^2 's > 0.40 , $p < 0.001$, the addition of %Fat did not account for an incremental amount of variance in incompatible congruent accuracy beyond associated descriptive variables, β 's > 0.14 , p 's > 0.20 . For Step 3, only the obese intervention group showed a significant relationship, adjusted $R^2=0.58$, $p < 0.001$, such that greater VAT was associated with smaller incompatible congruent accuracy, with VAT accounting for an incremental amount of variance in compatible congruent accuracy beyond associated descriptive variables, $\beta=-0.36$, $p=0.01$ (Table IV and Figure).

The Step 1 regression analysis for incompatible incongruent accuracy was significant, adjusted $R^2=0.38$, $p=0.001$. Although Step 2 was significant, $R^2=0.37$, $p=0.001$, the addition of %Fat did not account for an incremental amount of variance in incompatible incongruent accuracy beyond associated descriptive variables, $\beta=-0.03$, $p=0.64$. Step 3 was also significant, $R^2=0.41$, $p=0.001$, with the addition of VAT accounting for an incremental amount of variance in incompatible incongruent accuracy beyond associated descriptive variables, $\beta=-0.23$, $p=0.004$ (Table V 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 's 0.30 , p 's 0.006 . Although Step 2 was significant, R^2 's 0.33 , $p=0.005$, the addition of %Fat did not account for an incremental amount of variance in incompatible incongruent accuracy beyond associated descriptive variables, β 's 0.25 , p 's 0.14 . For Step 3, only the obese intervention group showed a significant relationship, adjusted $R^2=0.69$, $p=0.001$, such that greater VAT was associated with smaller incompatible incongruent accuracy, with VAT accounting for an incremental amount of variance in incompatible incongruent accuracy beyond associated descriptive variables, $\beta=-0.32$, $p=0.01$ (Table V and Figure).

Discussion

Previous research has established cross-sectional links between increased adiposity and measures of cognitive control in preadolescent children (12–16). Our study extends this by establishing the relationships between changes in adiposity and cognitive control using a 9-month PA randomized-controlled trial. Further, 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 was particularly 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 obese intervention participants.

This study examined the extent to which a PA intervention influenced changes in adiposity and subsequent improvements in inhibition across healthy weight and obese children. 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 following 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 over eat (34,35). 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 (36–

41). We extend the knowledge in this area by demonstrating a beneficial effect on cognitive control performance following PA intervention (23,42). Among sedentary obese children, previous research suggests a beneficial effect of PA on cognitive control (21,22). Previous research indicates that PA has been shown to increase concentration, memory, self-discipline, and classroom behavior (5, 19). In addition, regular PA participation has been linked to enhancement of brain function and cognition (5, 19). Although the present study and others (24,22,21) 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 (e.g., depression, anxiety) have been shown to differ between HW and OB children. For example, a relationship has been observed between sleep duration and disruption with obesity (2,66). In addition, Obstructive Sleep Apnea has also been related to deficits in cognitive control (67). Thus, it is possible that these health factors may account, in part, for the findings observed (66,68,69). 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 obese children, 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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Abbreviations and Acronyms

BMI	Body mass index
DXA	Dual Energy X-Ray Absorptiometry
SAT	subcutaneous abdominal adipose tissue
VAT	visceral adipose tissue
PA	physical activity
%Fat	whole body fat

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Table 1

Participant demographics and baseline fitness and adiposity characteristics

	Matched Healthy Weight	Obese	Entire Sample
n	77 (49 females)	77 (49 females)	154 (94 females)
Age (years)	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 ₂ (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
SAAT [*] (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

Note: IQ = intelligent quotient; SES = socioeconomic status; VO₂ = maximal oxygen volume; VAT = visceral adipose tissue; SAAT = subcutaneous adipose tissue;

^{*}
p = 0.05

Table 2

Correlations Between Demographics and Changes in Cognition and Adiposity.

	Compatible Congruent Accuracy	Compatible Incongruent Accuracy	Incompatible Incongruent Accuracy	Compatible Congruent Reaction Time	Compatible Incongruent Reaction Time	Incompatible Congruent Reaction Time	Incompatible Incongruent Reaction Time	%Fat	VAT	SAT
Age	−0.05	−0.08	−0.16	−0.06	−0.07	−0.09	−0.08	−0.07	−0.01	−0.03
Sex	0.04	0.08	0.00	0.09	0.15	0.12	0.15	−0.07	−0.04	−0.06
IQ	−0.02	0.02	−0.02	0.05	0.06	0.10	0.00	0.00	0.08	0.00
SES	−0.03	−0.02	−0.05	0.06	0.04	0.03	0.00	−0.01	−0.04	−0.11
Pre-Test VO2 FF	−0.15	−0.06	0.00	−0.08	−0.07	−0.07	−0.05	−.16 [*]	−0.10	−.19 [*]

* Correlation is significant at the 0.05 level (2-tailed).

Table 3
Regression Analyses for VAT predicting Compatible Congruent and Incongruent Accuracy

Step 1	Congruent					Incongruent				
	B	SE B	β	t	Step 1	B	SE B	β	t	
Treatment	-1.45	1.73	-0.05	-0.84	Treatment	0.85	2.02	0.03	0.42	
BMI Group	0.49	1.54	0.04	0.32	BMI Group	0.64	1.79	0.05	0.36	
Pre-Test Compatible Congruent Accuracy	-0.71	0.07	-0.62	-9.54*	Pre-Test Compatible Incongruent Accuracy	-0.59	0.09	-0.48	-6.54*	
Pre-Test %Fat	-0.09	0.21	-0.05	-0.42	Pre-Test %Fat	-0.08	0.25	-0.05	-0.32	
Pre-Test VAT	0	0.01	0.04	0.36	Pre-Test VAT	0	0.01	-0.02	-0.12	
Step 2					Step 2					
%Fat	-0.68	0.39	-0.11	-1.73	%Fat	-0.24	0.47	-0.04	-0.51	
Step 3					Step 3					
VAT	-0.04	0.02	-0.12	-1.55	VAT	-0.03	0.03	-0.08	-0.86	

Table 4

Regression Analyses for VAT predicting Incompatible Congruent Accuracy

Step 1	All				Obese Intervention			
	B	SE B	β	t	B	SE B	β	t
Treatment	-2.72	1.86	-0.09	-1.46				
BMI Group	-0.08	1.66	-0.01	-0.05				
Pre-Test Incompatible Congruent Accuracy	-0.60	0.06	-0.63	-9.76	-0.62	0.11	-0.65	-5.88*
Pre-Test %Fat	0.19	0.23	0.11	0.85	0.40	0.51	0.13	0.77
Pre-Test VAT	-0.01	0.01	-0.08	-0.71	-0.04	0.02	-0.35	-2.09*
Step 2								
%Fat	-0.20	0.44	-0.03	-0.46	-0.60	0.61	-0.11	-0.99
Step 3								
VAT	-0.09	0.03	-0.26	-3.38	-0.08	0.03	-0.36	-2.57*

Table 5
Regression Analyses for VAT predicting Incompatible Incongruent Accuracy

Step 1	All				Obese Intervention			
	B	SE	B	t	B	SE	B	t
Trt	-2.88	2.01	-0.09	-1.43				
BMI Class	0.13	1.83	0.01	0.07				
Pre-Test 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
Pre-Test %Fat	-0.01	0.25	-0.01	-0.05	-0.07	0.48	-0.02	-0.14
Pre-Test VAT	0.00	0.01	-0.03	-0.31	-0.03	0.02	-0.24	-1.70
Step 2								
%Fat	0.20	0.48	0.03	0.41	-0.39	0.57	-0.06	-0.68
Step 3								
VAT	-0.08	0.03	-0.23	-2.92*	-0.08	0.03	-0.32	-2.70*

Table 6
Regression Analyses for SAAT predicting Compatible Congruent and Incongruent Accuracy

Step 1	Congruent					Incongruent				
	B	SE B	β	t	Step 1	B	SE B	β	t	
Treatment	-1.43	1.81	-0.05	-0.79	Treatment	0.97	2.1	0.04	0.46	
BMI Group	0.63	1.48	0.05	0.43	BMI Group	0.61	1.73	0.04	0.35	
Pre-Test Compatible Congruent Accuracy	-0.71	0.08	-0.62	-9.48*	Pre-Test Compatible Incongruent Accuracy	-0.59	0.09	-0.49	-6.53*	
Pre-Test %Fat	-0.05	0.31	-0.03	-0.16	Pre-Test %Fat	-0.03	0.37	-0.02	-0.09	
Pre-Test SAAT	0	0	0	0	Pre-Test SAAT	0	0	-0.04	-0.21	
Step 2					Step 2					
%Fat	-0.7	0.4	-0.12	-1.75	%Fat	-0.23	0.48	-0.04	-0.48	
Step 3					Step 3					
SAAT	0	0.01	-0.05	-0.59	SAAT	0	0.01	-0.05	-0.48	

Note: Compatible congruent accuracy and SAAT Step 1 adjusted $R^2=0.39$, p 0.001; Step 2 $R^2=0.01$, $p=0.09$; Step 3 $R^2=0.01$, $p=0.56$. Compatible incongruent accuracy and SAAT Step 1 adjusted $R^2=0.21$, p 0.001; Step 2 was also significant, $R^2=0.01$, $p=0.63$; Step 3 $R^2=0.01$, $p=0.63$.

Table 7
Regression Analyses for SAAT predicting Incompatible Congruent and Incongruent Accuracy

Step 1	Congruent					Incongruent				
	B	SE B	β	t	Step 1	B	SE B	β	t	
Treatment	-1.49	1.9	-0.05	-0.78	Treatment	-1.13	2.03	-0.04	-0.56	
BMI Group	-0.12	1.57	-0.01	-0.08	BMI Group	0.43	1.71	0.03	0.25	
Pre-Test Incompatible Congruent Accuracy	-0.61	0.06	-0.64	-10.11*	Pre-Test Incompatible Incongruent Accuracy	-0.67	0.07	-0.66	-9.85*	
Pre-Test %Fat	0.75	0.33	0.42	2.28*	Age	2.25	1.75	0.09	1.29	
Pre-Test SAAT	-0.01	0	-0.4	-2.40*	Pre-Test %Fat	0.82	0.35	0.43	2.34*	
Step 2					Pre-Test SAAT	-0.01	0	-0.52	-3.09*	
%Fat	-0.03	0.44	0	-0.07	Step 2					
Step 3					%Fat	0.44	0.47	0.06	0.95	
SAAT	-0.01	0.01	-0.12	-1.4	Step 3					
					SAAT	-0.06	.008	-0.066	-7.81	

Note: Incompatible congruent accuracy and SAAT Step 1 adjusted $R^2=0.40$, p 0.001; Step 2 $R^2=0.01$, $p=0.94$; Step 3 $R^2=0.02$, $p=0.16$. Incompatible incongruent accuracy and SAAT Step 1 adjusted $R^2=0.42$, p 0.001; Step 2 $R^2=0.01$, $p=0.35$; Step 3 $R^2=0.01$, $p=0.44$.