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Effects of adiposity on postural control and cognition

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ABSTRACT

In the U.S., it is estimated that over one-third of adults are obese (Body Mass Index (BMI) > 30 kg/m^2). Previous studies suggest that obesity may be associated with deficits in cognitive performance and postural control. Increased BMI may challenge cognitive and postural performance in a variety of populations; however, most relevant studies have classified participants based on BMI values, which cannot be used to accurately assess the effects of adiposity on cognitive performance and postural control. The objective of the current study was to examine motor and cognitive responses for overweight and obese adults compared to normal weight individuals by using both BMI and adiposity measures. Ten normal weight (BMI = 18–24.9 kg/m^2), ten overweight (BMI = 25–29.9 kg/m^2), and ten obese (BMI = 30–40 kg/m^2) adults were evaluated (age: 24 ± 4 years). Participants were classified into three groups based on BMI values at the onset of the study, prior to body composition analysis. Participants performed (1) working memory task while maintaining upright stance, and (2) a battery of sensorimotor evaluations. Working memory reaction times, response accuracy, center-of-pressure (COP) path length, velocity, migration area, time to boundary values in anterior-posterior direction, and ankle-hip strategy-scores were calculated to evaluate cognitive-motor performance. No significant deficits in working memory performance were observed. Overall, measures of motor function deteriorated as BMI and body fat percentage increased. The relationship between deteriorating postural performance indices and body fat percentage were greater than those found between BMI and postural performance indices.

1. Introduction

The terms overweight and obesity are defined as abnormal/excessive fat accumulation with Body Mass Index (BMI) $\geq$ 25 kg/m^2 and 30 kg/m^2, respectively [1]. The World Health Organization (WHO) estimates that more than 1.9 billion adults are overweight worldwide; with 600 million obese adults in 2014 [1]. In the U.S., the situation is exacerbated with 78.6 million adults being classified as obese in 2012 [2].

Postural instability is defined as the inability to successfully respond to perturbations during upright stance [3] and is frequently associated with reduced sensorimotor function and increased fall risk [4,5]. Impaired motor function due to an increase in adiposity may severely impact quality of life and increase the risk of reduced postural stability and injury by falls [6,7]. Several studies have examined the relationship between obesity and postural control in adults. Hue et al. reported that increased body weight strongly correlated with decreased balance stability [8]. Similarly, increased body weight has been associated with increased anterior–posterior (AP) center of pressure (COP) movement [9]. Increased sway areas and an inability to modulate anticipatory actions suggests that obese participants use different postural strategies to maintain balance [10]. In contrast, Blaszczyk et al. suggested preserved postural control in obese adults [11], a notion later challenged in [12]. A primary limitation in these studies is that they used BMI as the primary classification method for identifying different weight groups; however, BMI only takes body mass and height into consideration. The exclusive use of BMI is flawed as a method to distinguish highly muscular persons from persons with high body fat percentages. Inconsistent outcomes from previous studies might have resulted from the use of BMI for classification. Using measures of fat amount may better illustrate the relationship between excessive adiposity and postural control.

Deficits in cognitive function have been reported as a powerful predictor of falls and correlate to dramatic increases in fall risks [13]. Recently, obesity has been linked with memory deficits and

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cognitive dysfunction in middle-aged and older adults [14,15]. Increased adiposity, resulting in obesity, may require additional attention for controlling posture [16]. Cognitive-motor interference, defined as decrements in performance that occur when cognitive and motor tasks are performed simultaneously (dual-task conditions), has been linked with falls [15]. A priori, we did not expect to see cognitive deficits in this study due to the narrow age range of participants in the current study; however, these data are the first step in preparing a larger scale evaluation of cognitive-motor deficits with respect to adiposity, aging, and neurological disease. Examining postural control during cognitive tasks will provide valuable information regarding the relationship among motor function, cognitive distraction, and excessive adiposity.

The objective of the current study was to examine responses during cognitive-motor tasks using different assessments of adiposity. The correlations among BMI, body composition, postural control, and cognitive performance were examined to clarify and explore the impact of adiposity on postural stability. We hypothesized that: (1) measures of postural control will deteriorate as indices of adiposity increase; and (2) whole body fat percentage ($\%Fat_{TOTAL}$) and trunk fat percentage ($\%Fat_{TRUNK}$) will exhibit more consistent relationships with postural control as compared to BMI. The results of this study advance our understanding of the true relationship of adiposity, body mass, and body fat distribution on postural control and cognitive performance.

2. Methods

2.1. Subjects

Thirty total individuals participated in the study. Participants were classified into three groups based upon their BMI scores at the onset of the study. The normal weight (BMI: 18.5–24.9 kg/m^2), the overweight (BMI: 25–29.9 kg/m^2) and the obese groups (BMI: 30–40 kg/m^2) each had five females and 5 males (Table 1). Prior to recruitment, participants completed a Physical Activity Readiness Questionnaire (PAR-Q) and the Modifiable Activity Questionnaire (MAQ). Exclusion criteria included: a history of neurological, musculo-skeletal or cardiovascular disorders; age below 18 or above 45 years old; and more than 90 min of exercise per week (indicating physical activity levels above moderate activity). The University of Houston Committee for the Protection of Human Subjects approved all procedures; all participants provided written informed consent.

2.2. Protocols

Each participant attended two testing sessions: (1) evaluation of postural and cognitive functions, and (2) body composition scanning. In (1), computerized dynamic posturography (NeuroCom International, Inc., Clackamas, OR) was used to record kinetic data at 100 Hz. A rectangular stability boundary was estimated by the outer extremes of the feet for each participant on NeuroCom force-plates; boundaries were marked and maintained in all conditions. In all conditions, participants stood upright with feet and body properly positioned, fitted with a safety harness and arms crossed in front of chest. Participants were tested under four conditions: (a) sensory organization test, (b) motor control test, (c) quiet stance, and (d) postural-cognitive evaluation (dual-task). All time series COP data were filtered using Butterworth low-pass filters with a cutoff frequency of 2 Hz, consistent with [17] using Matlab (The MathWorks Inc., 2013b, Natick, MA), verified using fast-Fourier transform analysis and consistent with the Nyquist sampling theorem.

Table 1
Participant age and anthropomorphic data.

	Normal weight	Overweight	Obese
N	10	10	10
Age (y)	24.4 ± 2.3	24.4 ± 3.0	23.8 ± 6.6
Mass (kg)	61.2 ± 10.0	80.0 ± 9.5	104.2 ± 20.4
Height (cm)	166.5 ± 10.8	167.9 ± 9.3	171.1 ± 11.4
BMI (kg/m^2)	21.9 ± 1.2	28.3 ± 1.5	35.3 ± 3.1
Trunk fat (%)	25.2 ± 5.3	37.2 ± 7.3	42.5 ± 4.2
Total fat (%)	25.3 ± 5.9	31.1 ± 6.6	37.2 ± 4.7

Values are mean ± SD. BMI = body mass index.

2.2.1. Sensory organization test (SOT)

SOT evaluations were performed in order to identify any potential sensory deficits. Participants experienced the six standard testing conditions in three 20 s trials: (1) eyes open with fixed platform, (2) eyes closed with fixed platform, (3) eyes open with sway-referenced vision, (4) eyes open with sway-referenced platform, (5) eyes closed with sway-referenced platform, and (6) eyes open with both sway-referenced vision and sway-referenced platform.

2.2.2. Motor control test (MCT)

The MCT was used to probe how participants responded to dynamic perturbations. Each participant underwent the six default perturbation conditions, applied via constant velocity force-plate translations. Three trials per condition were collected. The amplitude of perturbation was selected as small, medium, or large and the direction of translation included separate anterior and posterior conditions. In a set sequence, participants underwent each condition with eyes open: (1) posterior-small, (2) posterior-medium, (3) posterior-large, (4) anterior-small, (5) anterior-medium, and (6) anterior-large perturbation conditions.

2.3. Quiet stance testing

In the quiet stance condition, participants were instructed to cross their arms in front of their chest and keep their eyes open. Participants underwent three trials, lasting for 30 s each, consistent with the test duration in postural-cognitive evaluation.

2.3.1. Postural-cognitive evaluation

During postural-cognitive testing, participants underwent evaluation of working memory (N-back testing). Cognitive testing was only evaluated in this testing block and during no other evaluations. The N-back test is used to examine a participant's capacity to use short-term memory information in performing one or more tasks simultaneously [18]. The difficulty level of the N-back test is controlled by requiring participants to remember words further back in the presented series [19]. Each participant was assigned three levels of difficulty in auditory N-back tests (easiest to most difficult: 0-, 1-, and 2-back conditions) in a block randomized manner where task difficulty was the blocking factor. Participants were given a series of random words through a headphone-microphone device (Plantronics, Inc., Santa Cruz, CA), instructed to repeat the words, and at the same time maintain upright stance on the platform. Customized Visual C++ software was used to generate random words for this protocol (Microsoft, Corp., Redmond, WA). The rate of correct responses and verbal reaction time (how quickly the participant respond to the stimulus) was recorded by the software and extracted to evaluate cognitive performance.

2.3.2. Body composition assessment

Body composition of each participant was measured via a whole body dual-energy X-ray absorptiometry (DEXA) scanning device (Discovery W, Hologic, Inc., Bedford, MA). Both $\%Fat_{TOTAL}$ and $\%Fat_{TRUNK}$ were extracted for further analysis.

2.4. Kinetic data analysis

COP time series data and ankle-hip strategy scores were directly obtained via NeuroCom. The following measures were calculated directly from COP data: AP path length, AP velocity, COP migration area, minimum time to boundary (TTB), and integrated time to boundary (iTTB). Path length indicates the total COP displacement along the anterior–posterior (AP) direction. Mean velocity was computed to quantify the average COP speed in the AP direction. The area of COP migration was estimated by fitting an ellipse to the COP data using principal component analysis methods [20]. The TTB quantity is the predicted time that the COP will cross the stability boundary as defined by the edges of the feet [21]. Minimum TTB was identified; smaller TTB values indicate instability in a particular direction. The iTTB was calculated below a 10 s threshold as an estimate of relative instability over an entire trial [22]. The ankle-hip strategy-score quantifies joint movement on the basis of shear force; a score near 100 indicates predominance of an ankle strategy, lower scores indicates shift toward a hip-dominant strategy [23].

2.5. Statistical analysis

Linear mixed model analyses were used to compare the effects of test conditions, BMI, $\%\text{Fat}_{\text{TOTAL}}$ and $\%\text{Fat}_{\text{TRUNK}}$. Separate models were created for each dependent variable: COP path length, COP velocity, COP migration area, minimum TTB, iTTB, ankle-hip strategy-score, correct N-back response rate, and N-back reaction time via SPSS version 21.0 (SPSS IBM, New York, NY, U.S.A.). A total of 18 models (6 postural measures × 3 adiposity measures) were computed for SOT, MCT, and N-back evaluations. Additional models were created to evaluate adiposity with respect to N-back accuracy and reaction time measures (3 models for each, 6 additional total models). The fixed factor of *Condition* included 4 levels for N-back testing (quiet stance, 0-back, 1-back, and 2-back), 6 levels for SOT (see previous), and 6 levels for MCT (see previous). Linear regression models with interaction terms were used to evaluate the associations of BMI, $\%\text{Fat}_{\text{TOTAL}}$, and $\%\text{Fat}_{\text{TRUNK}}$ with each dependent variable. Cook's D was used to identify and eliminate outliers. Significance was set at $p < 0.05$.

3. Results

Overall, the data showed that as BMI and %Fat increased, participants' postural performance decreased in N-back testing and SOT. However, neither BMI nor %Fat had negative effects on neither cognitive performance nor motor performance during the presence of physical external perturbations (MCT).

3.1. N-back response rate and reaction time

No measures of adiposity were found to be associated with reaction time or response rate of working memory in any of the statistical models used in this analysis. No differences in task performance were found among different N-back levels across the entire sample.

3.2. Quiet stance and N-back testing

Mixed model analysis indicated that N-back testing, BMI, and %Fat had generally negative effects on postural control. Overall, %Fat exhibited more consistent relationships with postural control as compared to BMI.

Compared to the quiet stance condition, increased N-back difficulty was associated with increased AP path length ($p < 0.005$, Fig. 1A), increased mean AP velocity ($p = 0.005$, Fig. 1B), reduced minimum TTB (MinAPTTB, $p < 0.05$, Fig. 1C), and increased iTTB (APiTTB, $p < 0.01$, Fig. 1D). Post hoc testing indicated AP path

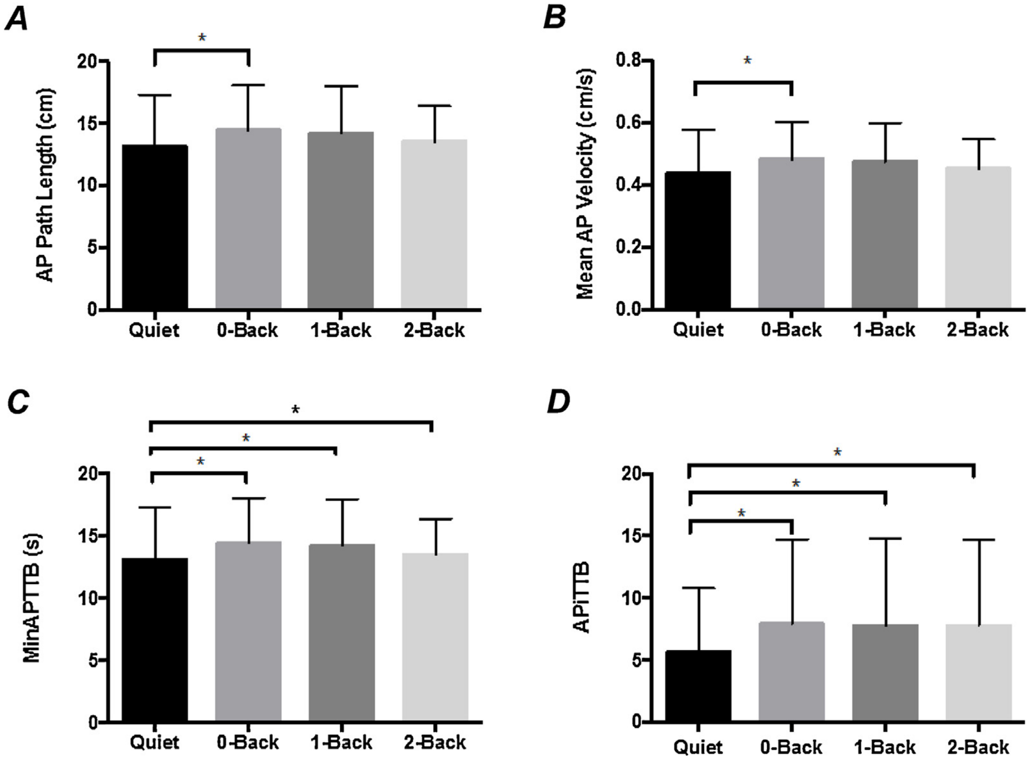

Fig. 1. Effects of working memory evaluation (N-back test) on postural performance measures. Mean values and standard deviations are shown. * Denotes significant post hoc differences with Bonferroni corrections applied. (A) Anterior–posterior (AP) path length. (B) Mean AP velocity. (C) Minimum time to boundary in the AP direction (MinAPTTB). (D) Integrated time to boundary in the AP direction (APiTTB).

Table 2
N-back testing, sensory organization test (SOT), and motor control test (MCT) statistical results.

Measures	N-back		BMI		$\%Fat_{TOTAL}$		$\%Fat_{TRUNK}$	
	$F_{3,76.3}$	p-value	$F_{1,27.0}$	p-value	$F_{1,26.0}$	p-value	$F_{1,25.9}$	p-value
MinAPTTB	3.972	0.011*	0.515	0.479	3.824	0.061	4.819	0.037*
APiTTB	4.537	0.006*	0.004	0.948	2.488	0.126	4.042	0.055
AP path length	5.597	0.002*	7.278	0.012*	4.819	0.037*	7.636	0.01*
AP velocity	4.438	0.005*	8.358	0.006*	4.468	0.042*	7.508	0.008*
Strategy score	1.802	0.154	36.565	$<$0.001**	6.577	0.016*	10.408	0.003*
COP migration area	2.202	0.094	2.123	0.157	4.888	0.036*	4.488	0.044*
Reaction time	1.116	0.335	0.415	0.525	0.96	0.336	1.514	0.229
	SOT		BMI		$\%Fat_{TOTAL}$		$\%Fat_{TRUNK}$	
	$F_{5,121.8}$	p-value	$F_{1,29.6}$	p-value	$F_{1,25.0}$	p-value	$F_{1,24.6}$	p-value
MinAPTTB	10.542	$<$0.001**	2.459	0.128	4.181	0.051	7.247	0.012*
APiTTB	21.727	$<$0.001**	0.117	0.735	1.555	0.224	3.659	0.067
AP path length	8.163	$<$0.001**	1.824	0.187	1.158	0.291	3.149	0.087
AP velocity	0.525	0.757	0.697	0.411	1.175	0.288	2.687	0.113
Strategy score	2.797	0.02*	11.587	0.002*	6.062	0.021*	10.305	0.004*
COP migration area	3.444	0.006*	0.061	0.807	2.786	0.107	3.745	0.064
	MCT		BMI		$\%Fat_{TOTAL}$		$\%Fat_{TRUNK}$	
	$F_{5,129.1}$	p-value	$F_{1,27.9}$	p-value	$F_{1,27.0}$	p-value	$F_{1,27.0}$	p-value
MinAPTTB	0.589	0.708	1.118	0.287	0.007	0.933	0.866	0.36
APiTTB	0.722	0.608	0.871	0.359	0.338	0.566	0.554	0.463
AP path length	2.306	0.048*	1.834	0.187	0.399	0.533	1.249	0.274
AP velocity	0.968	0.44	0.058	0.811	1.91	0.178	0.055	0.816
Strategy score	8.54	$<$0.001**	3.264	0.083	0.915	0.347	3.446	0.075
COP migration area	0.629	0.678	0.004	0.95	0.468	0.499	0.007	0.933

F- and p-values are provided for the fixed factors.
* Significant at $p < .05$.
** Significant at $p < .001$.

length and AP velocity were significantly lower in the quiet stance condition compared to 0-back condition (Fig. 1A and B, Table 2). Post hoc testing also indicated MinAPTTB and APiTTB were significantly lower in quiet stance compared to all N-back conditions (Fig. 1C and D, Table 2).

With respect to the effect of BMI on postural control, AP path length increased ($p < 0.05$, Fig. 2A) with increased BMI, and mean AP velocity ($p < 0.01$, Fig. 2D). Ankle-hip strategy-score ($p < 0.001$, Fig. 2J) was negatively correlated with increased BMI (Table 2). An interaction between N-back level and BMI was found in the measures of AP path length ($F_{3,76.3} = 4.77$, $p < 0.005$, Fig. 2A), AP velocity ($F_{3,76.3} = 3.40$, $p < 0.05$, Fig. 2D), minimum TTB (MinAPTTB, $F_{3,76.3} = 2.42$, $p < 0.05$), and iTTB (APiTTB, $F_{3,76.3} = 3.28$, $p < 0.05$).

When taking $\%Fat_{TOTAL}$ into consideration, increased AP path length ($p < 0.05$, Fig. 2B) and faster mean AP velocity ($p < 0.05$, Fig. 2E) persisted with higher $\%Fat_{TOTAL}$ (Table 2). Additionally, increased $\%Fat_{TOTAL}$ correlated positively with larger COP migration areas ($p < 0.05$, Fig. 2H), and reduced ankle-hip strategy-score ($p < 0.05$, Fig. 2K), reported in Table 2.

Analyses of kinetic measures indicated that increased $\%Fat_{TRUNK}$ was associated with increased AP path length ($p < 0.05$, Fig. 2C), increased mean AP velocity ($p < 0.01$, Fig. 2F), larger COP migration area ($p < 0.05$, Fig. 2I), and reduced ankle-hip strategy-score ($p < 0.005$, Fig. 2L), Table 2.

3.3. Sensory organization test (SOT)

In the SOT, as *Condition* became more challenging, more body sway was exhibited. Statistical results are presented in Table 2 and in Fig. 3. Analysis of both BMI and $\%Fat_{TOTAL}$ showed strong effects of both measures on ankle-hip strategy-score ($p < 0.005$, $p < 0.05$, respectively). Higher BMI and $\%Fat_{TOTAL}$ were associated with decreased ankle-hip strategy-score, illustrated in Fig. 2M and N. Increased $\%Fat_{TRUNK}$ was associated with lower ankle-hip strategy-scores ($p < 0.005$) and reduced MinAPTTB ($p < 0.05$), illustrated in Fig. 2O and 2R. BMI and $\%Fat_{TRUNK}$ interacted with *Condition* with respect to ankle-hip strategy-scores ($F_{5,130.1} = 2.93$, $p < 0.05$; $F_{5,130.1} = 2.80$, $p < 0.05$), shown in Fig. 2M and O.

3.4. Motor control test (MCT)

In the MCT, a significant effect of *Condition* was found for AP path length and ankle-hip strategy-score ($p < 0.05$, $p < 0.005$, respectively), such that AP path length increased and ankle-hip strategy-score decreased as perturbation size increased in forward/backward translations (Table 2). BMI, $\%Fat_{TOTAL}$, and $\%Fat_{TRUNK}$ did not show significant effects on the dependent variables measured within the MCT; no interactions were found.

4. Discussion

The goal of the current study was to investigate the relationship between adiposity and cognitive and motor functions. Of the two hypotheses formulated in the introduction, the first hypothesis was partially confirmed and the second hypothesis was confirmed. Compared to BMI, $\%Fat_{TOTAL}$ and $\%Fat_{TRUNK}$ exhibited more consistent trends regarding postural performance.

4.1. Adiposity and motor performance

In the current study, significant decrements in postural performance were observed with increased adiposity on AP path length, AP mean velocity, and ankle-hip strategy-score during cognitive loading. Our results support findings from previous studies such that obesity is associated with increased body sway and postural instability [8–10]. Several factors may contribute to postural control deficits associated with increased adiposity. A large amount of body fat distributed about the abdomen leads to an exaggerated anterior position of the full-body center-of-mass (COM) with respect to the ankle joint [24]. The combination of increased body mass and increased horizontal COM distance may

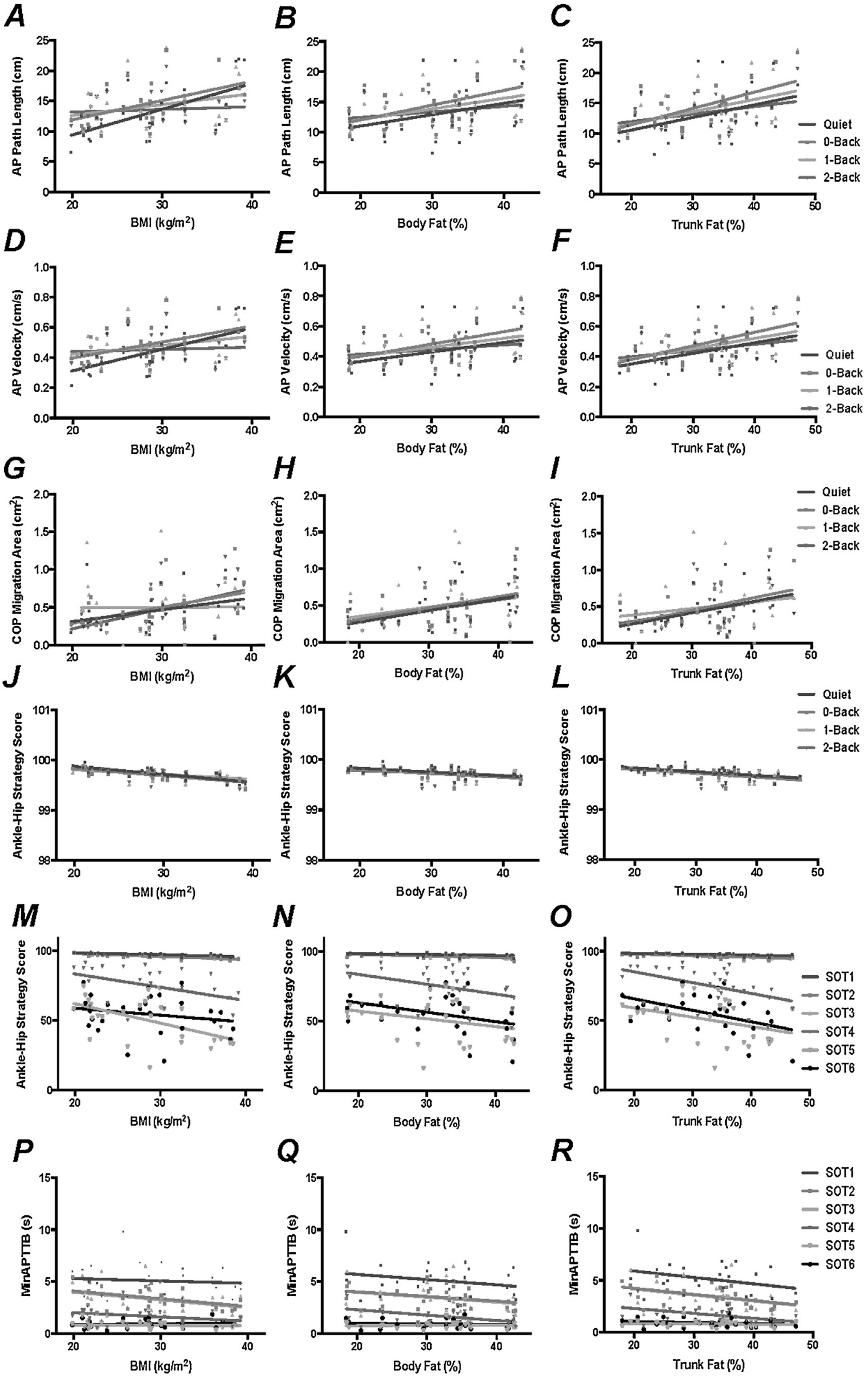

Fig. 2. Correlations between adiposity and postural performance measures under N-back testing and the sensory organization test (SOT). In N-back testing (panels A–L), postural measures generally increased with increased adiposity measures, with the exception of strategy-score. In SOT evaluation (panels M-R), postural measures decreased with increased adiposity as conditions became more challenging. In both tests, interaction effects were noted in analyses using BMI measures. (A) AP path length versus BMI. (B) AP path length versus %Fat$_{TOTAL}$. (C) AP path length versus %Fat$_{TRUNK}$. (D) AP velocity versus BMI. (E) AP velocity versus %Fat$_{TOTAL}$. (F) AP velocity versus %Fat$_{TRUNK}$. (G) COP area versus BMI. (H) COP area versus %Fat$_{TOTAL}$. (I) AP velocity %Fat$_{TRUNK}$. (J) Strategy-score versus BMI. (K) Strategy-score versus %Fat$_{TOTAL}$. (L) Strategy-score versus %Fat$_{TRUNK}$. (M) Strategy-score versus BMI. (N) Strategy-score versus %Fat$_{TOTAL}$. (O) Strategy-score versus %Fat$_{TRUNK}$. (P) MinAPTTB versus BMI. (Q) MinAPTTB versus %Fat$_{TOTAL}$. (R) MinAPTTB versus %Fat$_{TRUNK}$.

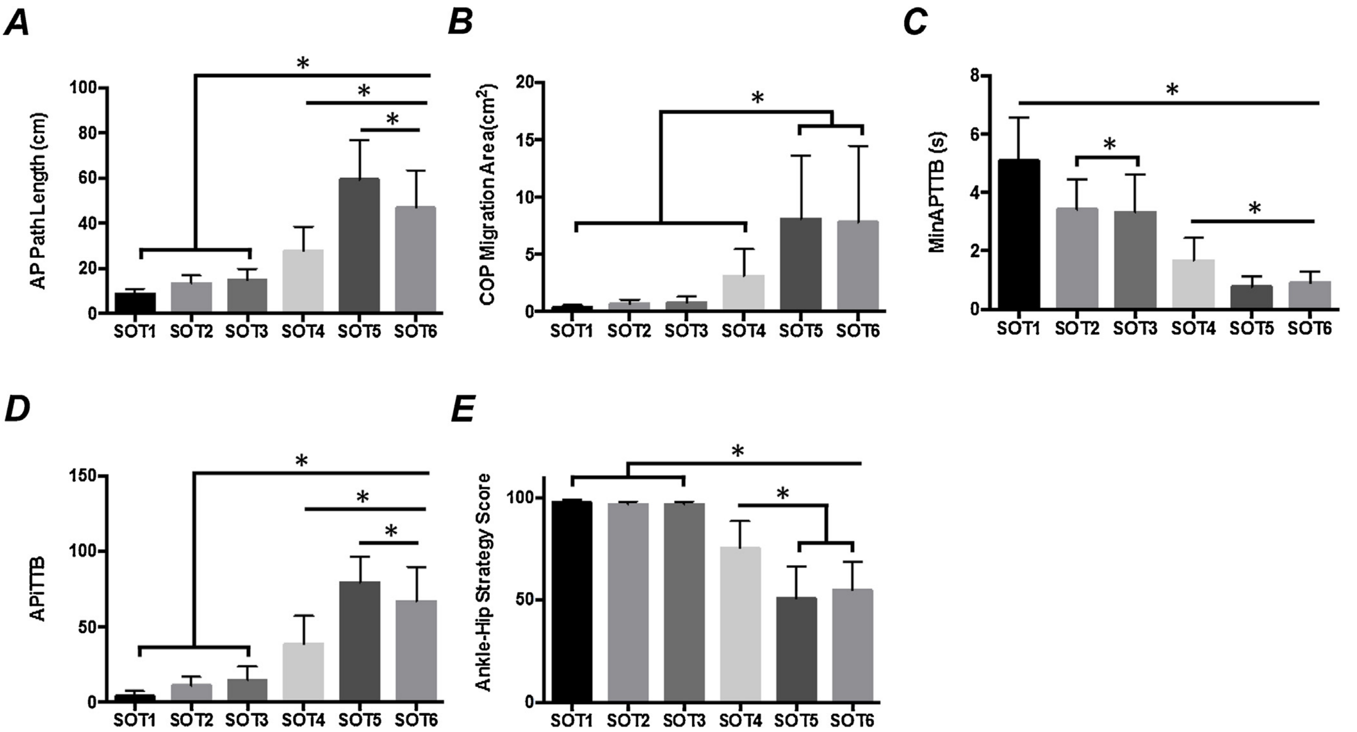

Fig. 3. Effects of SOT *Condition* on postural performance measures. Mean values and standard deviations are shown. Generally, postural control measures worsened as *Condition* became more challenging. (A) Anterior–posterior (AP) path length. (B) COP area. (C) Minimum time to boundary in the AP direction (MinAPTTB). (D) Integrated time to boundary in the AP direction (APiTTB). (E) Strategy-score.

result in the necessity of increased ankle torque generation to maintain postural stability. As an extension of this hypothesis, a recent study suggests that motor commands of obese individuals are more variable than those of lean individuals, leading to impaired motor control with obesity [25]. Obese individuals are known to generate higher maximal strength values; however, when this strength has been expressed as relative volume with respect to body mass, obese individuals have lower relative maximal force [26]. Relative weaker muscular strength may also contribute to difficulty in generating the ankle torques required to maintain postural stability, particularly in older adults when confronted with externally-generated perturbations. Postural control also depends on the ability to integrate information from multiple sensory systems. Accurate plantar sensation may play a vital role in maintaining upright stance [27]. Due to prolonged exposure of heavy loads on the feet, planar cutaneous sensation may be impaired in overweight and obese individuals [27,28]; however, this is beyond the scope of the current study.

Most previous obesity-related studies have not directly examined the link between adiposity and postural control. While BMI has been widely used for its convenience, this metric remains unable to differentiate body composition [29]; likely leading to discrepancies in reported postural evaluation results. In the current study, we measured both %Fat$_{TOTAL}$ and %Fat$_{TRUNK}$, as fat distribution may be directly related to postural control. Use of BMI was associated with the emergence of confounding interaction effects within the postural data that were not present when the same data were assessed using both %Fat measurements. These results suggest that %Fat is a better predictor of postural control as compared to BMI. Further, these data suggest that increased adiposity negatively impacts postural control under both cognitive-posture and posture-only tests.

No significant effects of adiposity were observed in tasks with external postural perturbations (e.g. MCT). MCT results may differ from SOT results as MCT is used to assess one's ability to quickly and automatically recover from unexpected external perturbations. One potential reason why no adverse responses to postural perturbations emerged may be due to the population evaluated. Here, only young healthy controls with no history of neurological and/or musculoskeletal disorders were evaluated. Given their health state, all participants may have retained the ability to adequately respond to external perturbations, independent of their amount of adiposity. It is possible that such responses may differ in aged overweight and obese individuals with neurological and/or musculoskeletal disorders. The results of this study imply that clinical and rehabilitation studies should take into account of the effects of adiposity in recruiting patients. In future studies, researchers should be careful in selecting obesity classification methods to avoid confounding outcomes. Finally, future studies should expand this study to older adults as a means to examine the mechanisms leading to postural instability and falls in older adults.

4.2. Cognitive-motor performance

Previous studies have attempted to establish a link between obesity and cognitive impairment. Higher BMI has been associated with reduced cognitive function [14]; however, the exact mechanism contributing to reduced cognitive function with obesity remains unknown. Furthermore, obesity may require additional attention for controlling posture [16]. In our study, cognitive performance was not associated with adiposity in any of the tested groups. As the average participant age was 24 years old, frequent brain activity may offset the negative effects of adiposity on cognitive performance in adults. In the current study, we did not specifically recruit adults who experienced childhood obesity, leaving us unable to comment on the longitudinal effects of obesity within the current data set; further investigation is needed in this area.

5. Conclusions

Overall, our findings indicate that obesity is associated with reduced postural control, where %Fat is a better predictor of postural control as compared to BMI, indicating that DEXA analysis is a superior method of obesity classification. The mechanism underlying the association between adiposity and postural control remains unclear and requires further investigation.

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Conflict of interest statement

None of the authors have financial or personal relationships causing conflicts of interest to report.

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