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Differences in lower extremity muscular coactivation during postural control between healthy and obese adults.

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Abstract

Introduction: It is well established that obesity is associated with deterioration in postural control that may reduce obese adults' autonomy and increase risks of falls. However, neuromuscular mechanisms through which postural control alterations occur in obese adults remain unclear.

Objective: To investigate the effects of obesity on muscle coactivation at the ankle joint during static and dynamic postural control.

Materials and methods: A control group (CG; n = 20; $age=32.5\pm7.6$ years; BMI=22.4±2.2 Kg/m²) and an obese group (OG; n=20; $age=34.2\pm5.6$ years; BMI=38.6±4.1 Kg/m²) participated in this study. Static postural control was evaluated by center of pressure (CoP) displacements during quiet standing. Dynamic postural control was assessed by the maximal distance traveled by the CoP during a forward lean test. Electromyography activity data for the gastrocnemius medialis (GM), soleus (SOL) and tibialis anterior (TA) were collected during both quiet standing and forward lean tests. Muscle activities were used to calculate two separate coactivation indexes (CI) between ankle plantar and dorsal flexors (GM/TA and SOL/TA, respectively).

Results: CoP displacements were higher in the OG than in the CG for quiet standing (p<0.05). When leaning forward, the maximal distance of the CoP was higher in the CG than in the OG (p<0.05). Only the CI value calculated for SOL/TA was higher in the OG than in the CG for both static and dynamic tasks (p<0.05). The SOL/TA CI value in the OG was positively correlated with CoP displacements during quiet standing (r = 0.79; p<0.05).

Conclusion: Obesity increases muscle coactivation of the soleus and tibialis anterior muscles at the ankle joint during both static and dynamic postural control. This adaptive

neuromuscular response may represent a joint stiffening strategy for enhancing stability. Consequently, increased ankle muscle coactivation could not considered as a good adaptation in obese adults.

1. Introduction

During postural control, young adults produce a net torque at the ankle joint by activating both agonist and antagonist muscles to maintain body stabilization [1]. The simultaneous activation of agonist and antagonist muscles around a joint, which is defined as muscle coactivation [2], is designed to allow an agonist to work fluently [1] and to increase joint stabilization during refined motor performance [3]. In older adults, studies have reported increased levels of muscle coactivation between the soleus and the tibialis anterior during static postural control tests compared to young adults [4–6]. Increased muscle coactivation at the ankle level in older adults has been suggested to be a compensatory mechanism for enhancing postural control stability, resulting in increased joint stiffness [3,7,8]. However, other studies have reported a correlation between high muscle coactivation and low postural control stability [8,9], which may be explained by the following: first, strong muscle activation has been associated with an increased risk of excessive energy expenditure [7], resulting in fatigue, which could potentially lead to an increased risk of postural instability; and second, excessive muscle coactivation increases postural rigidity and might restrict dynamic postural control [8].

Several authors have shown that obesity is associated with a deterioration in postural control, resulting in an increase in center of pressure (CoP) oscillations during quiet standing [10,11]. Deteriorations in this functional capacity could lead to increase risks of falls, which may reduce obese adults' autonomy [12]. Postural control alterations in obese adults have been explained by the increased mechanical demands related to an increased body mass and

2

by the non-negligible proportion of body mass that is further from the axis of rotation (i.e., the ankle joint, assuming an inverted pendulum model) [11,13,14]. Recently, we reported that obese individuals showed similar relative force values for the ankle plantar and dorsal flexor muscles to those for non-obese individuals, suggesting that the primary source of imbalance in obese individuals is not associated with a lack of strength but is instead associated with increased muscle activity in the plantar flexors of the ankle [10]. This phenomenon is due to the increased stimulation of the plantar flexors in obese individuals, to counteract the anterior postural instability. However, we did not investigate ankle muscle coactivation in obese individuals, which may provide further indications regarding the underlying mechanisms through which postural control alterations occur in obese individuals.

To our knowledge, only the study of Tomlinson et al. [15] has investigated ankle muscle coactivation during maximal isometric contraction in obese adults, indicating that obesity does not affect antagonist coactivation during this task. However, this study did not evaluate ankle muscle coactivation during postural control testing, nor did examine the relationships that may exist between ankle muscle coactivation and body weight which is a strong predictor of postural control stability [16]. Thus, the objectives of this study were to evaluate the effects of an excessive body weight on muscle coactivation at the ankle level during static and dynamic postural control and to analyze the relationships between ankle muscle coactivation, CoP parameters and body weight in obese adults. We hypothesize that an excessive body weight is associated with increased ankle muscle coactivation during static and dynamic postural control tasks, and the level of muscle coactivation and CoP parameters are positively correlated with body weight in obese adults.

2. Methods

2.1.Participants

Forty participants, recruited from our university and from an obesity treatment center, were divided into two groups (**Table 1**) according to their body mass index (BMI, Kg/m²). The non-obese control group (CG; age= 32.5 ± 7.6 years; BMI= 22.4 ± 2.2 Kg/m²) included 20 normal-weight adults and the obese group (OG; age= 34.2 ± 5.6 years; BMI= 38.6 ± 4.1 Kg/m²) included 20 obese adults. Participants were excluded from the study if they had a history of cardiovascular disorders, diabetes, uncorrected vision problems, severe musculoskeletal deformities, or injuries to their lower extremities that would interfere with testing. Participants were informed about the purpose and protocol of the study and all gave their written consent. The study was conducted according the Declaration of Helsinki 2004 and was approved by the local ethics committee on human research.

2.2.Experimental protocol

2.2.1. Static postural control testing

Static postural control during quiet standing was measured using a force platform (Zebris FDM-S; sampling rate: 100 Hz; Zebris Medical GmbH, Isny, Germany). Participants were instructed to stand barefoot on the platform with their feet together and their arms hanging alongside their bodies and to sway as little as possible. They were then asked to stare at a visual reference point located at eye level two meters in front of them and to participate in postural trials involving three conditions: with eyes open (EO), with eyes closed (EC), and with eyes open while standing on a foam (EOF). In all experimental conditions, the participants engaged in these three postural trials (30 s per trial) with rest intervals of 30 s to prevent fatigue or boredom. Nine trials were randomly presented to eliminate any order effects. Center-of-pressure (CoP) displacements were determined to extract three postural parameters: the area of the 95%-confidence ellipse (area, cm²) and the velocity (velocity,

mm.s⁻¹) and length (length, cm) of the CoP displacements. In this study, we used the average recorded measurements of the three trials for each condition.

2.2.2. Forward lean test

Participants were instructed to stand barefoot on the platform with their feet together and their arms hanging alongside their bodies and to lean forward. When leaning forward, participants were asked to keep their bodies rigid and to rotate around their ankle joints, to limit angular displacements of other joints and to keep their heels in contact with the floor. Participants performed three trials with one-minute rest intervals. The initial and final CoP coordinates of both legs (taken from the same trial) were used to determine the maximum forward distance traveled by the CoP [10]. The following equation was used to calculate the maximal distance performed:

Maximal distance =
$$\sqrt{(X_{\text{final}} - X_{\text{initial}})^2 + (Y_{\text{final}} - Y_{\text{initial}})^2}$$

2.2.3. Electromyography recording

Electromyographic (EMG) data of the dominant leg muscles were collected using a Powerlab 16/35 system (sampling rate: 1000 Hz, ADInstruments, Dunedin, New Zealand). Two unipolar surface electrodes (Uni-gel Single Electrode -T3425, Thought Technology Ltd, Montreal, Canada) were placed on three lower limb muscles: the gastrocnemius medialis (GM), soleus (SOL) and tibialis anterior (TA). Before attaching the electrodes, to reduce impedance, the skin was carefully shaved and cleaned using an abrasive cleaner and alcohol swabs. The placement and location of the surface electrodes conformed to the recommendations of SENIAM (Surface EMG for Noninvasive Assessment of Muscles) [17]. Electrodes were carefully positioned on the belly of each muscle parallel to the orientation of the muscle fibers at inter-electrode intervals of 20 mm. The EMG activity was recorded for the GM, SOL and TA while participants were performing maximal voluntary contractions (MVC) of their plantar and dorsal flexors to extract the maximal activity of each muscle and during the static postural control and forward lean tests. During the MVC of the plantar flexors muscles, participants were instructed to sit on the weight bench fitness chair, to keep their back, buttock and thigh in contact with the chair, to keep their leg stretched horizontally and to push with the tip of the foot on the wall. For the dorsal flexors muscles, participants were instructed to stand up, to keep their ankle at 90° and to push with the foot on the experimenter's hand [10].

The EMG analysis was post-processed using Matlab (Matlab R2013a, MathWorks, Natick, USA), and raw EMG signals were band-pass filtered at 15–500 Hz through a secondorder Butterworth digital filter to remove noise or movement interference [18]. Both the data collected during static postural control and forward lean tests were rectified and smoothed using root mean square analysis (RMS) with a 20-ms window. The RMS of each muscle was normalized to the maximal RMS values of each muscle obtained during MVC of each muscle. The normalized RMS of the GM (EMG GM), SOL (EMG SOL) and TA (EMG TA) were used in the study.

------Fig.1------

During static postural control testing, the coactivation levels of the GM/TA and SOL/TA were calculated for the dominant leg over the 30s of each test. During the forward lean test, the coactivation levels were calculated for the dominant leg and between the beginning of the forward movement and the return to the initial position. For all tasks, coactivation index (CI) was calculated using the following equation [19]:

$$CI = \frac{2 I \text{ antagonist}}{I \text{ total}} \times 100,$$

where *I antagonist* is the area of the total antagonistic activity and *I total* is the integral of the sum of (EMG TA + EMG SOL) during the task, which were calculated using the following equations:

(1) I antagonist =
$$\int_{T_1}^{T_2} EMG TA(t) dt + \int_{T_2}^{T_3} EMG SOL(t) dt$$
,

where T_1 to T_2 is the period in which the TA is working as an antagonist muscle (i.e., the EMG of the TA is less than that of the SOL), T_2 to T_3 is the period during which the SOL is working as an antagonist muscle, and *I antagonist* is the integral of the sum of these two periods.

(2)
$$I total = \int_{T_1}^{T_3} [EMG agonist + EMG antagonist](t) dt.$$

We noted that in these equations we used EMG SOL and EMG TA in order to calculate the CI of (SOL/TA). The same equations were used to calculate the CI of (GM/TA), where EMG SOL was replaced by EMG GM.

-----Fig.2-----

2.3. Statistical analysis

Statistical analyses were performed using Statistica Software 13.0 (Software, Inc., Tulsa, USA). The normality and homogeneity of variance were respectively determined using the Kolmogorov–Smirnov and Levene tests. When the parametric assumptions were met, we applied one way ANOVA (obesity) to compare participant's characteristics and maximal displacements of CoP during the forward lean test. A factorial ANOVA (obesity × postural task) with post hoc Fisher's least significant difference (LSD multiple comparison procedure) were applied to compare CI and CoP displacement during postural control testing. Relationships between the body weight, CI and CoP [16] were evaluated using Pearson's correlations analysis. Values are expressed as means (\pm 95% confidence interval). The level of significance was set at the 5% critical level (p<0.05). Partial eta-squared (η^2) and study power (β) were also reported.

3. Result

Participants' characteristics are shown in Table 1.

-----Table 1-----

3.1. Static postural control testing

The two-way ANOVA showed a significant interaction effect (obesity × postural task) on area (F _(2.114)=18.6, p<0.05, η^2 =0.15, β =0.98), velocity (F _(2.114)=36.5, p<0.001, η^2 =0.71, β =1) and CoP length (F _(2.114)=22.3, p<0.01, η^2 =0.54, β =1) (**Table 2**). Post hoc analysis revealed that OG exhibited significantly greater area, velocity and CoP length than CG in all conditions (p<0.05). In OG, area, velocity and CoP length were greater in EC than EO condition and in EOF than EC condition (p<0.05). In CG, only in EOF condition, parameters of CoP were higher than in other conditions (p<0.05).

-----Table 2-----

3.2. Forward lean test

The one-way ANOVA showed a significant obesity effect on CoP displacement when leaning forward ($F_{(1.18)}$ =74.9, p<0.001). The maximum distance traveled by the CoP was shorter in OG [6.4 cm (5.6; 9.2)] than in CG [(12.8 cm (8.2; 14.9)].

3.3. Coactivation index during static postural control and forward lean testing

During the static postural control tests, the two-way ANOVA showed a significant interaction effect (obesity × postural task) on CI of SOL/TA (F _(2.114)=25.1, p<0.01, η^2 =0.21, β =0.98). Post hoc analysis revealed that OG exhibited significantly greater CI of SOL/TA than CG in all conditions (p<0.05). In OG, CI of SOL/TA was greater in EOF than in EC condition and in EC than in EO condition. In CG, CI of SOL/TA was greater in EOF than in EC than in EO condition. In CG, CI of SOL/TA was greater in EOF than in EC and EO conditions (p<0.05) (**Fig. 3**).

-----Fig.3-----

During the static postural control tests, the two-way ANOVA showed no interaction effect (obesity × postural task) on CI of GM/TA. Only a significant effect on postural task was observed (F _(2.114)=154.1, p<0.001, η^2 =0.56, β =1). Post hoc analysis revealed that OG and

CG exhibited significantly greater CI of GM/TA in EOF than in EC condition and in EC than in EO condition (p<0.05) (**Fig. 4**).

------Fig.4------

During the forward lean test, the one-way ANOVA showed a significant obesity effect on CI of SOL/TA ($F_{(1.18)}$ =54.1, p<0.001). The CI of SOL/TA were higher in OG [38.8 (33.1; 46.4)] than in CG [25.9 (20.4; 30.4)].

The Pearson correlation's analysis revealed significant correlations between body weight and CI of SOL/TA during quiet standing in EO (r =0.61, p<0.05), EC (r =0.74, p<0.05) and EOF conditions (r =0.78, p<0.05) in OG (**Fig. 5**). Significant correlations were also revealed between CoP velocity and body weight in EO (r =0.68, p<0.05), EC (r =0.76, p<0.05) and EOF conditions (r =0.73, p<0.05) in OG (**Fig. 6**). During Forward lean test, CoP displacement was negatively correlated with body weight (The equation of the linear regression: CoP displacement = $0.35 \times Body$ weight + 131.8 (R² = 0.57, r = -0.75, p<0.05).

4. Discussion

This is the first study that measured the effect of obesity on muscle coactivation at the ankle joint under quiet standing and dynamic postural control capacities. The study provides three major findings. First, an excessive body weight is associated with altered CoP parameters during static and dynamic postural control trials. Second, obesity induces higher ankle muscle coactivation during upright standing tasks. Finally, during these tasks coactivation index of SOL/TA is significantly correlated with body weight in obese adults.

In the present study, velocity, length and area of CoP during upright standing tests were higher in OG compared CG. Since increased values of these parameters is repeatedly associated with higher risk of falls, data then confirm that obesity induces an important limitation factor of postural control regulation [10,11,20]. In addition, results point out that an excessive body weight was associated with higher CoP velocity (Fig. 2) confirming that an excessive body weight is a strong predictor of postural stability [16].

Several possibilities have been presented in order to explain the strong relationship between body weight and balance stability [10,11,21,22]. Alterations in postural control in obese individuals may be the consequence of a lack of quality and/or quantity of sensory information arising from plantar mechanoreceptors [22]. This is caused by decreased sensitivity resulting from hyper-activation of the plantar mechanoreceptors due to the greater pressure required to support a heavier body mass [23]. Another explanation is related to an increased solicitation of attentional resources in obese individuals for controlling posture [21]. In this context, Mignardot et al. [21] reported that postural oscillations and reaction time to sound stimulation increase in obese individuals when they performed multitasking. This suggests that situations that are cognitively challenging might place additional demands on maintaining balance and be additionally detrimental for postural control in obese individuals. Postural control regulation in obese individuals is related to higher motor control variability compared to lean individuals [11,16,24]. Indeed, when standing upright, the human body is likened to an inverted pendulum rotating around the ankle joint. An excessive body weight, in particular at the abdominal level, induces an anterior position of the CoP and a forward sway. This advanced position increases the gravitational torque at the ankle level and could lead to greater angular accelerations [11]. Thus, obese individuals require generating greater muscle torque in order to counteract this angular acceleration. Since greater ankle torque is related to high motor variability [24], increased muscle torque could add more noise to feedback postural control system and consequently larger balance motor commands variability. This could be explain why an excessive body weight is strongly correlated with increased CoP

10

velocity [16] as this latter parameter results from larger variability in the balance motor commands [24]. Recently, we reported that the relative strength produced by obese adults at the ankle level is similar to non-obese adults. Thus, altered postural control capacities in obese individuals may be not related to an insufficient ankle muscle force production but likely to an increase in muscle activity of plantar flexors [10]. We suggest that the increase of muscle activity could be an adaptive response of the neuromuscular system to maintain body posture.

In the present study, OG demonstrated higher muscle coactivation of the ankle joint (SOL/TA) than CG during EO (+22%), EC (+27%) and EOF (+29) conditions. These differences indicate that obesity induces not only a decline in postural control ability (Table. 2) but also a change in central nervous system control of postural regulation. Additionally, the high level of coactivation index of SOL/TA was positively correlated with the high body mass weight in obese group (Fig. 5) and this latter parameter was also correlated with increased CoP velocity (Fig. 6). These observations support the hypothesis that an excessive body weight is associated with higher ankle muscle coactivation of SOL/TA and this could substantially explain the increased CoP velocity which represents the overall amount of activity necessary to maintain stability [16]. Higher muscle coactivation can be interpreted as a postural control strategy that stiffens the joints of lower limbs [25]. Thus, increased muscle coactivation may be a necessary neuromuscular adaptation to compensate for altered postural control in obese individuals related to mechanical changes (e.g., anterior position of CoP) [11], deficiency in the proprioception process [22], and higher attentional cost during balance [21]. However, previous studies indicate that excessive muscle coactivation decreases the performance of agonist muscles in single joint movement [26] and increases postural rigidity [27]. This may reduce the degree of freedom of the postural control system [27]. Generally, postural control uses the same neuromuscular control mechanisms during quiet standing and

dynamic postural control [28]. Therefore, results related to the lower distance when leaning forward in OG as compared to CG could suggest that the restricted flexibility in the ankle joint may be related to an excessive muscle coactivation (+50%) may reduce voluntary movements of the CoP when leaning forward [8]. Additionally, CoP displacement was negatively correlated with body weight indicating that a heavier weight induces not only a decline in static balance but also in dynamic postural control ability. However, we did not observe correlations between body weight and coactivation index when leaning forward. Although further studies are required, the absence of correlation between body weight and muscle activities could be partly explained by the fear of falling.

Otherwise, studies reported that high muscle coactivation induces a greater energetic cost [28] and may induce an early fatigue [29]. Ledin et al. [30] indicated that muscular fatigue of the triceps substantially altered postural control in individuals with 20% additional body weight. Muscular fatigue could also restrict the ability to respond to postural perturbation [31] due to the inability to produce the required force output, and to decreased functionality of the proprioceptive system [32,33]. All of these observations suggest that the high muscle coactivation during postural control for counteracting obesity-related constraints may accelerate development of fatigue. In fact, it is possible that the appearance of early fatigue may lead to restrict the ability to respond to postural perturbation in obese adults, that's why increased muscle coactivation could not be considered as a good adaptation since it has a negative consequence that may also impact daily activities as the gait ability, and increase consequently the risk of falls. However, these suggestions need to be confirmed experimentally.

Contrary to what we found for the SOL/TA coactivation index, results of the present study reported no significant differences between CG and OG concerning the GM/TA

12

coactivation index. Possible explanations may be related to the higher level of GM activity in obese adults. While GM activity is always higher than TA activity, GM is described as being an agonist muscle during movement. Consequently, the level of coactivation depends on the level of TA activity. First, obese individuals require higher GM activity (relative to non-obese individuals) in order to counteract the anterior position of CoP. However, no significant differences in TA activity were observed [10]. These observations indicate that in obese adults, the GM is principally activated as an agonist muscle and TA as an antagonist muscle during balance regulation. Therefore, similar levels of TA activity may explain our observation concerning the GM/TA coactivation values. Second, the GM is a biarticular muscle that acts not only as a plantar flexor but also as a knee flexor, meaning that it functions as an antagonist muscle during knee extension. In contrast, the SOL is a monoarticular plantar flexor. Based on this anatomical difference, the GM should be highly activated in order to simultaneously regulate the angle of the ankle and knee during quiet standing [34]. Consequently, the high level of GM activity caused by simultaneous stimulation during quiet standing may impact the level of the GM/TA coactivation index.

The present study provides important information on the adaptive responses of the neuromuscular system in obese adults but it is worth noting that there is a limitation. It is difficult to confirm that the high ankle muscle coactivation observed in obese individuals is only caused by postural alterations related to an excessive body mass. This neuromuscular adaptation could be also related to the mechanical constraints of the position (feet together) during the postural control testing, or at least, to the combined effect of both phenomena. Further studies are needed to verify these hypotheses.

13

Finally, results of this study may offer to clinician a complementary approach that could be used in the development of rehabilitation and training procedures. Comprehensive strength training of ankle joint muscles and proprioceptive abilities could be an appropriate management to decrease muscle coactivation of lower limbs and energy expenditure that may delay the development of fatigue. Future research should test the impact of this type of physical activity training, which may be a relevant modality of management, both to confirm our hypotheses and to ameliorate balance regulation in obese adults.

5. Conclusion

Obesity increases muscle coactivation of the soleus and tibialis anterior muscles at the ankle joint to counteract obesity-related constraints during postural regulation. However, high levels of muscle coactivation may reduce the performance of the agonist muscle, increase postural rigidity and could confound future development of fatigue. For these reasons, muscle coactivation could not be considered as a good adaptation since it is associated with negative consequences that could impact other daily activities. Consequently, this alteration should be taken in consortium for the development of rehabilitation and training procedures.

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Fig. 1. Typical recordings of EMG activities of gastrocnemius medialis (A), soleus (B) and tibialis anterior (C) during postural control of a non-obese (CG) and an obese participant (OG).



Fig. 2. Model of agonist and antagonist activities during postural control testing (adapted from Falconer and Winter, 1985). From T_1 to T_2 , TA was defined as agonist and SOL as an antagonist muscle. From T_2 to T_3 , SOL was defined as an agonist and TA as an antagonist muscle





CG: control group, OG: obese group, * = Significant difference between CG and OG (p<0.05), ¥ = Significant difference between EO and other conditions in the same group (p<0.05), + = Significant difference between EC and EOF condition in the same group (p<0.05)



Fig. 4. Coactivation index of the gastrocnemius medialis and tibialis anterior of obese and control groups

CG: control group, OG: obese group, $\mathbf{Y} =$ Significant difference between EO and other conditions in the same group (p<0.05), + = Significant difference between EC and EOF condition in the same group (p<0.05)



Fig. 5. Correlations between body weight and coactivation index of the soleus and tibialis during static postural control trials in obese individuals

Black circles present the linear relationship between the body weight and the coactivation index (SOL/TA) in eyes opened condition. The equation of the linear regression: $CI = 0.07 \times Body$ weight + 6.9 ($R^2 = 0.37$, r = 0.61, p<0.05)

White circles present the linear relationship between the body weight and the coactivation index (SOL/TA) in eyes closed condition. The equation of the linear regression: $CI = 0.16 \times Body$ weight + 1.8 ($R^2 = 0.55$, r = 0.74, p<0.05)

Black squares present the linear relationship between the body weight and the coactivation index (SOL/TA) in eyes opened standing on a foam condition. The equation of the linear regression: $CI = 0.24 \times Body$ weight -2.6 ($R^2 = 0.60$, r = 0.78, p<0.05)



Fig. 6. Correlations between body weight and the center of pressure velocity during static postural control trials in obese individuals

Black circles present the linear relationship between the body weight and the center of pressure velocity in eyes opened condition. The equation of the linear regression: Velocity = $0.11 \times Body$ weight - 1.6 (R² = 0.42, r = 0.68, p<0.05)

White circles present the linear relationship between the body weight and the center of pressure velocity in eyes closed condition. The equation of the linear regression: Velocity = $0.15 \times \text{Body}$ weight + 9 (R² = 0.42, r = 0.76, p<0.05)

Black squares present the linear relationship between the body weight and the center of pressure velocity in eyes opened standing on a foam condition. The equation of the linear regression: Velocity = $0.24 \times Body$ weight + 2.6 (R² = 0.53, r = 0.73, p<0.05)

Characteristics	CG	06	Obesity affect	
Characteristics	(r - 20)	(1 20)	Obesity effect	
	(n = 20)	(n = 20)		
	(Mean \pm 95% confidence	$(Mean \pm 95\%)$	p - level	
	interval)	confidence interval)		
Age (years)	33.5 (30.7 ; 36.2)	, 36.2) 34.2 (30.3 ; 35.7)		
Body height (cm)	170.3 (166.3 ; 174.2)	171.6 (168.0 ; 175.2)	NS	
Body mass (kg)	69.7 (66.2 ; 73.1)	108.9 (104.2 ; 113.8)	p<0.001	
BMI (kg/m²)	23.9 (23.4 ; 24.5)	37.0 (35.9 ; 38.5)	p<0.001	
Body fat (%)	17.2 (15.9 ; 18.5)	41.4 (40.2 ; 42.6)	p<0.001	
Waist circumference	71.0 (68.8 ; 73.1)	112.1 (106.9 ; 117.2)	p<0.001	
(cm)				
Hip circumference (cm)	76. 2 (73.2 ; 79.2)	100.2 (97.2 ; 103.3)	p<0.001	

Table 1. Participants' physical characteristics.

Comparison between two groups based on one-way ANOVA for each parameter. CG: control group, OG: obese group, BMI: body mass index, NS: no significant effect.

	CG	OG	Obesity effect		Postural task effect		Obesity imes Postural task			
	$(Mean \pm 95\%)$	(Mean ± 95%								
	confidence interval)	confidence interval)								
			F-values	p-Level	F-values	p-level	F-values	p-level		
Area EO (cm ²)	1.9 (1.7 ;2.0)	2.5 (2.3 ; 2.6)								
Area EC (cm ²)	3.8 (3.6; 4.1)+	5.3 (4.9; 5.8)*+	$F_{(1.114)} = 56.9$	p<0.001	$F_{(2.114)} = 200.1$	p<0.001	$F_{(2.114)} = 11.1$	p<0.05		
Area EOF(cm ²)	4.0 (3.8 ; 4.2)+	6.0 (5.6 ; 6.5)*+¥	. ,	-				-		
Velocity EO (mm/s)	8.0 (7.7; 8.3)	11.0 (10.3 ;11.7)*								
Velocity EC (mm/s)	18.2 (17.2; 19.2)+	24.9 (23.4 ; 26.3)*+	$F_{(1,114)} = 170.2$	p<0.001	$F_{(2.114)} = 473.5$	p<0.001	$F_{(2.114)} = 60.2$	p<0.001		
Velocity EOF (mm/s)	21.7 (20.9 ; 22.5)+¥	29.4 (27.8 ; 30.1)*+¥		•		•		•		
Length EO (cm)	18.7 (17.4 ; 19.9)	24.0 (22.5 ; 25.5)*								
Length EC (cm)	27.2 (26.1 ; 28.3)+	45.4 (41.9 ; 48.9)*+	$F_{(1,114)} = 155.2$	p<0.001	$F_{(2.114)} = 273.9$	p<0.001	$F_{(2.114)} = 25.2$	p<0.01		
Length EOF (cm)	46.3 (44.4 ; 48.2)+¥	56.5 (53.2 ; 59.8)*+¥		-		-		-		

 Table 2. Postural parameters of participants during postural control testing.

Comparison between two groups based on a factorial ANOVA [obesity \times postural task] for each parameter with additional post hoc analysis where there is a main effect of Obesity, Postural task and an interaction (Obesity \times Postural task). CG: control group, OG: obese group, EO: eyes opened, EC: eyes closed, EOF: eyes opened standing on a foam.

*= Significant difference between OG and CG (p<0.05)

+ =Significant difference between EOF or EC and EO (p<0.05)

 $\mathbf{Y} = \mathbf{Significant}$ difference between EC and EOF