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Relationships between lower limbs fatigability threshold and postural control in obese adults.

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France) and the students of Le Mans University for their acceptance to participate in this

study.

Abstract:

The objective of this study was to investigate the effect of obesity on markers of neuromuscular fatigability of ankle muscles and their potential relationships with postural control capacities.

Two groups: non-obese control group (CG; n=15; age = 40.1 ± 13.9 years; BMI = 23.9 ± 2.8 kg/m²) and obese group (OG; n=15; age = 44.2 ± 12.4 years; BMI = 42.2 ± 5.2 kg/m²), performed two postural tasks with and without vision in order to collect parameters of center of pressure (CoP) displacements; area (cm²), velocity (mm/s), length (cm) and the ratio length/area. Fatigability was estimated during 60 repeated maximal voluntary contractions of plantar (PF) and dorsal flexors (DF). The maximal force, critical force (F_{cr}) presenting the asymptote of the force-time evolution and rate of the force decrease (τ) were extracted. Results reported that CoP parameters were higher in OG compared to CG in all postural tasks (p<0.05). F_{cr} of PF and DF were 39% and 28% lower respectively in OG compared to CG (p<0.05). Only τ of PF was 12% lower in OG (p<0.05). F_{cr} of PF was negatively correlated with the ratio length/area in OG in all postural tasks (r = 0.86 and r = 0.85, respectively, p<0.05). We concluded that obesity is associated with a decreased F_{cr} of ankle muscles expressing the intensity threshold above which fatigue develops drastically. We also revealed that decreased fatigability threshold particularly that of PF, could partly explain postural control alterations in obese adults.

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 control in obese adults.
 3

4 **1. Introduction**

A growing body of evidence suggests that obesity affects negatively the postural 5 control regulation (Cieślińska-Świder and Błaszczyk, 2019; Corbeil et al., 2001; Handrigan et 6 al., 2017; Hue et al., 2007; Maktouf et al., 2018; Maktouf et al., 2019). Indeed, obesity 7 induces changes in body geometry (i.e., waist and hip circumferences) due to an excessive 8 9 accumulation of adipose tissue in body segments (e.g., waist and hip circumferences). The distribution of adipose tissue separates obesity into two groups; the android type (i.e., the fat 10 is localized in the abdominal area) and the gynoidal obesity type (i.e., the fat is localized on 11 the thighs and buttocks (WHO, 2008). Recently, Cieślińska-Świder et al. (2017) reported that 12 androidal obesity type exposed to a greater risk of instability than the gynoidal obesity type. 13 The androidal obesity is characterized by an excessive body fat at the abdominal level which 14 induces an anterior position of the center of Mass (CoM), and its relative position to the 15 center of foot pressure (CoP), from the axis of rotation (i.e., ankle joint assuming an inverted 16 pendulum model, where the CoM is the controlled variable and the CoP is the controlling 17 variable; Corbeil et al., 2001). This forward position of the CoM causes an increased 18 gravitational torque accelerating the body forward (Corbeil et al., 2001; Simoneau and 19 20 Teasdale, 2015). Consequently, individuals with androidal obesity have to develop an appropriate ankle torque with a higher rate of force development and velocity than normal 21 weighted individuals to counteract the gravitational torque in order to restore similar balance 22 level (Handrigan et al., 2012; Simoneau and Teasdale, 2015). This suggests that the altered 23 postural control abilities in adults with androidal obesity could be explained by a lack of 24 strength from the muscles mobilizing the ankle joint (Hue et al., 2007; Matrangola and 25 26 Madigan, 2009). However, Maktouf et al. (2018) reported that obese individuals showed

similar relative force capacities for the ankle plantar (PF) and dorsal flexors (DF) muscles
compared to normal weighted individuals, indicating that the primary source of imbalance in
obese individuals is rather associated with an increase in muscle activity of the PF than with a
lack of strength. These authors hypothesized that the increased muscle activity might be
responsive for an early fatigue in muscles, leading to postural alteration in obese individuals.
The aforementioned observations question the effects of the lower extremity muscle fatigue
development on postural control regulation in obese individuals.

Several studies focused on fatigue development of the upper extremity muscles in 34 obese adults, showing that obesity was associated with an early fatigue and a delayed 35 recovery from isometric endurance exercise (Cavuoto and Nussbaum, 2014; Eksioglu, 2011; 36 37 Pajoutan et al., 2017). To our knowledge, only the study of Pajoutan et al. (2016) focused on the fatigue development of the lower extremity muscles in obese adults. They reported that 38 increased body mass index altered endurance and central activation during maximal voluntary 39 isometric contraction (MVC) of the tibialis anterior (TA) after a sustained isometric fatiguing 40 41 task (60% of MVC). However, they did not investigate the relationship between the early fatigue development and altered postural control parameters in obese adults, nor the 42 fatigability threshold of PF that are considered as the primary muscles responsible for the 43 forward balance recovery in obese adults (Loram et al., 2004). Moreover, it is well established 44 that a percentage of MVC did not refer to metabolic exercise intensity domains and thus to 45 fatigue development (Kent-Braun et al., 1993; Saugen et al., 1997). The critical force (F_{cr}), 46 which corresponds to the maximal exercise that still results in metabolic steady state, is 47 considered as a potential alternative approach that allows identifying a threshold above which 48 the fatigability is critically developed in healthy and pathological individuals (Poole et al., 49 2016; Veni et al., 2019). Thus, F_{cr} is the intensity threshold above which fatigue develops 50

51 when performing isometric contractions of single agonist muscle (Poole et al., 2016).

52 However, to the best of our knowledge, no study has examined the effect of obesity on F_{cr} of 53 muscles mobilizing ankle joint in order to identify the fatigability threshold.

Therefore, the main objective of this study was to investigate the effect of obesity on 54 the markers of neuromuscular fatigability of ankle muscles in order to better understand the 55 neuromuscular fatigability threshold in obese adults. Second, we aim to examine the 56 relationships between markers of the neuromuscular fatigability and postural control 57 parameters to provide indications about the underlying mechanisms of postural control 58 alterations in individuals with androidal obesity. We hypothesize that obese adults have a 59 decreased fatigability threshold and an earlier fatigue development compared to normal 60 61 weighted adults and that F_{cr} is negatively correlated with altered postural control parameters 62 in obese adults.

2. Methods

64 **2.1.Participants**

Thirty men, recruited from our university and from an obesity treatment center, were 65 divided into two groups (Table 1) according to their body mass index (BMI, kg/m²): the non-66 obese control group (CG; n=15; age = 40.1(13.9) years; BMI = 23.9(2.8) kg/m²) and the obese 67 group (OG; n=15; age = 44.2(12.4) years; BMI = 42.2(5.2) kg/m²). Only adults with androidal 68 obesity type (i.e., obese individuals with the waist to hip ratio greater or equal to 0.85; 69 Cieślińska-Świder et al., 2017). Participants were excluded from the study if they had a 70 history of cardiovascular disorders, diabetes, uncorrected vision problems, severe 71 musculoskeletal deformities, or injuries to their lower extremities that would interfere with 72 testing. All participants were informed about the purpose and protocol of the study and gave 73

- their written consent. The study was conducted according the Declaration of Helsinki 2004and was approved by the local ethics committee on human research.
- 76 **2.2.Experimental protocol**
- 77

2.2.1. Anthropometric measurements

Height, waist and hip circumferences were obtained using a measuring tape. Body
mass and percentage of body fat were measured using an impedance-meter (Tanita; SC 240Class III; Tanita Europe B. V., Amsterdam, The Netherlands). Fat body mass (FBM) and lean
body mass (LBM) were calculated using the equations from Gartner et al. (2004).

82

2.2.2. Static postural control testing

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 with eyes opened (EO) and with eyes closed (EC). In each experimental condition, participants performed three postural trials (30 s per trial) with rest intervals of 30 s to prevent fatigue or boredom. The six trials were randomly presented to eliminate any order effects.

The raw center-of-pressure (CoP) signal of each trial was filtered by a Butterworth's 90 low-pass filter (n = 4; cut-off frequency = 5Hz) in order to overcome the problems associated 91 with the clean cut transition (Paillard and Noé, 2015). Then, the signal of CoP displacements 92 93 was pre-processed by a three-channel Anologic-to-Digital converter (100/16). Finally, data of CoP displacements was used to extract three postural parameters: the area of the 95%-94 confidence ellipse (area, cm²), the velocity (velocity, mm/s) and length (length, cm) of the 95 CoP displacements (Donath et al., 2012; Paillard and Noé, 2015; Schubert and Kirchner, 96 2014). The average recorded measurements of the three trials for each condition were used in 97 98 this study. The ratio between length and area of the CoP (L/A) for each condition was also

calculated in order to investigate the energy spend indicated by the length of CoP versus the 99 100 accuracy of the postural system indicated by the area (Paillard and Noé, 2015).

101

2.2.3. Muscle evaluations

The force of the ankle plantar flexor (PF) and dorsal flexor (DF) muscles of the 102 103 dominant leg were measured using a dynamometer (Sauter FL1K; Type: Force Gauge; Sauter 104 GmbH, Balingen, Germany). For each muscle group, a 20-min experimental session was dedicated as follows: (1) Familiarization and maximal voluntary contraction (MVC) testing in 105 order to determine the warm up intensity, (2) warm up (10 contractions at 25% of the pre-106 107 warm up MVC, five at 50% and two at 70%), (3) two-MVC with one-min rest between, (4) three-min of recovery, (5) exercise fatigability testing. The recovery between both muscle-108 specific experimental sessions lasted 10 minutes. 109

For PF testing, participants were instructed to keep their back, buttock and thigh in 110 contact with a height-adjustable chair, to keep their leg stretched horizontally and to push 111 112 with the tips of the foot on the dynamometer. A strap was placed around the dominant leg to secure it firmly to the foot plane (Maktouf et al., 2018). For DF, participants were asked to 113 stand up, to keep their ankle at 90° and push up with the foot on the dynamometer (Nagai et 114 115 al., 2013).

116

2.2.3.1. Maximal voluntary contraction testing

Prior to exercise fatigability measurement, two maximal voluntary contractions trials 117 were performed with a two-min rest in-between. Only the best trial was considered (absolute 118 force, N) and then normalized to LBM (relative force, N/kg). 119

120

2.2.3.2. Fatigability intervention

During the muscle fatigability testing, participants were invited to repeat 4-s maximal 121 contractions interspersed by 1 s for up to 5 min (i.e., 60 contractions). The follow-up was 122 assured by a smartphone's application which displayed a red color for contraction and green 123

color for recovery with an according audio signal. Strong verbal encouragement was given
during every contraction to promote maximal effort. In order to avoid pacing strategies,
participants were unaware of the precise duration of the test or of the remaining time (Tucker,
2009). The peak force (F _{peak}, expressed in percentage of MVC) of each contraction was used
in this study (Fig. 1).

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

130 **2.2.4. Data analysis**

131 The kinetics analysis of F _{peak} of both DF and PF was processed using Matlab (Matlab 132 R2013a, MathWorks, Natick, USA). The non-linear regression technique was used to trace 133 the exponential model of the kinetic of F _{peak} over the time of each participant and to extract 134 markers of fatigability according to Veni et al., (2019):

135
$$F_{peak} = F_{cr} + (100 - F_{cr}) \times e^{\frac{1}{\tau}}$$

136 Where F_{peak} is the maximal force expressed in percentage of the MVC, *t* is the 137 time of contraction, τ is the curvature constant in number of contraction 138 corresponding to the rate of the force decrease and F_{cr} is the force-time 139 asymptote relationship corresponding to the intensity threshold above which 140 fatigue develops.

141 The index of variability (RMSSD) was calculated for each participant using the following142 equation:

143

144
$$\sqrt{\frac{\sum_{i=1}^{n-1} (Fpeak (i) - Fpeak (i+1))^2}{n-1}}$$

145 Where *n* is the number of contractions (i.e., 60 contractions), $F_{peak}(i)$ is the 146 maximal force expressed in percentage of the MVC at *t* instant and F_{peak} 147 (i+1) is the F_{peak} at t+l instant.

149 **3. Result**

150 **3.1. Statistical analysis**

Statistical analysis was performed using Statistica Software 13.0 (Software, Inc., 151 Tulsa, USA). The normality and homogeneity of variance were respectively determined using 152 the Shapiro-wilk and Levene tests. The assumption of normality and homogeneity of variance 153 was confirmed for all parameters. The student's t-test was used for markers of fatigability and 154 anthropometric parameters, and the two-way ANOVA (obesity × postural task) for postural 155 parameters with the Bonferroni post-hoc procedures were performed. Relationships between 156 the postural parameters and markers of fatigability were evaluated using Pearson's 157 correlations analysis. Values were expressed as means (standard deviation). The level of 158 significance was set at the 5% critical level (p < 0.05). 159 **3.2.** Anthropometric measurements 160 161 Participants' characteristics are shown in Table 1. Body mass, BMI, body fat, hip and 162 waist circumferences and the ratio waist/hip were higher in OG compared to CG (p < 0.05). 163 There are no significant differences concerning LBM, age and body height between groups. 164 ------Table 1------165 **3.3. Static postural control testing** 166 The two-way ANOVA showed a significant obesity × postural task interaction effect 167 on area ($F_{(1.56)} = 11.1$, p < 0.05, $\alpha^2 = 0.15$, $\beta = 0.98$), velocity ($F_{(1.56)} = 60.2$, p < 0.01, $\alpha^2 = 0.71$, β 168 = 1), CoP length ($F_{(1.56)}$ = 25.2, p < 0.01, α^2 = 0.54, β = 1) and L/A ration ($F_{(2.56)}$ = 15.9, p < 0.05, 169 $\alpha^2 = 0.27$, $\beta = 0.98$) (**Table 2**). Post hoc analysis revealed that OG exhibited significantly 170 171 greater area, velocity, CoP length and L/A ratio than CG in all conditions (p < 0.05). In the two groups, area, velocity and CoP length were greater in EC than EO condition (p < 0.05). 172 The L/A ratio was higher in EC than EO condition only in OG (p < 0.05). 173

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175

3.4. Muscle evaluations

176	The parameters of the neuromuscular fatigability are presented in Table 3. The
177	absolute and the relative of PF and DF were not significantly different between OG and CG.
178	For PF, the relative F_{cr} to MVC was 21% lower in obese group (p < 0.01). The F_{cr} in
179	Newton per body mass was significantly 39% lower in OG (p < 0.01). The τ value was 12%
180	lower (p < 0.05) and the RMSSD was 47% higher in OG (p < 0.001). For FD, only the F_{cr} in

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

181 Newton per body mass (p < 0.05) and the τ (p < 0.05) remained significantly lower in OG.

3.5. Correlations analyses

183 Pearson correlation's analysis revealed significant correlations between the (L/A) ratio

184 of CoP displacements and the F_{cr} PF expressed in percentage of MVC in EO (r = 0.86,

185 $p \le 0.05$) and EC (r = 0.85, $p \le 0.05$) in OG (**Fig. 2**).

186 **4. Discussion**

187 Results point out that obesity decreases the F_{cr} expressing the intensity threshold above
188 which fatigue develops drastically, and an early fatigue development in ankle muscles.
189 Moreover, results indicate that F_{cr} of PF was negatively correlated with altered postural
190 control parameters in obese adults.

The absolute and relative maximal forces during the MVC testing are statistically similar in CG and OG (**Table 3**). This observation provides evidence that obesity does not affect maximal force production capacities at the ankle level and suggests that obese and nonobese adults have similar neuromuscular functioning characteristics (e.g., function of crosssection area, muscle typology, motor recruitment) during MVC. However, the MVC evaluation alone cannot inform about the mechanisms of muscles fatigability in obese adults. Consequently, we focused on the F_{cr} concepts in this study. Physiologically, F_{cr} can be

determined to discriminate poor and heavy intensities and is defined as an intensity threshold 198 199 above which fatigue develops drastically (Burnley et al., 2012; Ferguson et al., 2007). Interestingly, F_{cr} expressed in percentage of the MVC and F_{cr} expressed in Newton per 200 201 kilogram of body mass of the PF were 21% and 39% lower in OG than CG, respectively. This reveals that obesity lowers the fatigability threshold of ankle muscles promoting an early 202 fatigue development. Obese adults may thus often exceed their F_{cr} even during daily living 203 activities especially when ankle muscles are considerably solicited (e.g., walking, balance...). 204 In the same way, the curvature of the force-time relationship of the PF was 12% lower in OG 205 confirming that obesity causes not only a reduced fatigability threshold but also an earlier 206 207 fatigue at the ankle level compared to non-obese adults. These neuromuscular alterations may contribute to the development and persistence of chronic fatigue in this population (Veni et 208 al., 2019). 209

It has been also reported that older adults have greater peak force variability (RMSSD) 210 than young adults (Poole et al., 2016) due to a decreased of maximal force (Sosnoff and 211 Newell, 2006). The present study shows that despite obese adults develop similar relative 212 force to non-obese ones, they have higher peak force variability of PF (+47%). Hence, the 213 force variability seems to be independent of the maximal force level in obese adults. It is 214 215 possible that the high force variability observed in obese adults is related to an altered central mechanisms driving motor behavior (Lorist et al., 2002). This hypothesis is in accordance 216 with the findings of Pajoutan et al. (2017) reporting that fatigue in obese adults originates 217 218 rather from the central level than the peripheral level. The precise nature of this interference and the level it occurs need further investigations. 219

Otherwise, only the F_{cr} of DF normalized to body mass was lower in OG compared to
 CG. This may be surprising since obese adults present the same relative MVC of DF. It is

likely that DF and PF do not present the same neuromuscular mechanisms of fatigability. 222 223 Indeed, DF contains approximately 27% of fast twitch fibers characterized by their low fatigue resistance (Wåhlin Larsson et al., 2008), while PF, particularly gastrocnemius 224 225 muscles, contains over 43% of fast twitch fibers (Gollnick et al., 1974). For all these reasons, we can assume that obesity decreases the fatigability threshold and induces an early fatigue 226 development, particularly at PF level. This observation confirms results of Maffiuletti et al. 227 228 (2007) pointed out that obese individuals demonstrated lower fatigue resistance during voluntary extensions of quadriceps muscle. 229

Otherwise, Pajoutan et al. (2016) showed a significant effect of obesity on central 230 activation failure of dorsiflexor muscles of the ankle after a fatiguing tasks suggesting that 231 232 obese individuals may be at higher risk of falls due to weaker ankle dorsiflexor as the prime controllers of the balance recovery. In this context, results of this study reported a strong 233 linear correlation has been evidenced between the ratio (L/A), i.e. the level of the energy 234 spend versus the accuracy of the postural system (Paillard and Noé, 2015), and the F_{cr} 235 expressed in percentage of MVC in EO and EC conditions (Fig.2). The more patients reported 236 higher energy spent to regulate balance, the lower F_{cr} were found. This observation suggests 237 that postural control alterations in obese adults could be partly explained by a decreased 238 fatigability threshold. Similar relationship has previously been observed between increased 239 fatigability and poor functional mobility and balance in obese subjects. (Ercan et al., 2019; 240 Resnick et al., 2006). A possible explanation is related to mechanical changes in obese 241 242 individuals caused by their wide body mass per se. Indeed, a non-negligible proportion of body mass, particularly in individuals with androidal obesity, is further away from the axis of 243 rotation. This might cause a greater gravitational torque that accelerates the body in the 244 anteroposterior axis (Corbeil et al., 2001). To maintain upright stance, this gravitational 245

torque must be countered by increasing muscular torque (Handrigan et al., 2012; Teasdale et 246 247 al., 2007). Consequently, obese individuals need to generate greater muscle force at the ankle level to reach a level of balance similar to non-obese individuals. However, results of this 248 249 study point out that the relative strength produced by obese adults at the ankle level is similar to non-obese adults confirming that altered postural control capacities in obese individuals are 250 not related to an insufficient ankle muscle force production but rather to an increase in PF 251 muscle activity (Maktouf et al., 2018). Thus, the high level of muscle activity may increase 252 253 the development of an early extremity muscle fatigue (Pajoutan et al., 2017). Therefore, our results fit with the hypothesis made by Maktouf et al. (2018) suggesting that the postural 254 255 control instability in obese adults can be explained by the neuromuscular fatigability of ankle muscles. We also reveal that postural control alterations are not mainly related to an early 256 fatigue but rather to a decreased fatigability threshold of PF (i.e. decreased Fcr). In fact, the 257 258 higher muscle activity of PF needed to counteract obesity related constraints (i.e., mechanical and/or proprioceptive) could be responsive for a higher energy cost (Marques et al., 2013) and 259 consequently lower extremity muscle fatigue development (Pajoutan et al., 2016). 260 Accordingly, it is evident that the more the fatigability threshold of PF is decreased, the 261 higher risk of instability could be observed in obese adults. Moreover, it is possible that the 262 lower fatigability threshold in obese individuals, when combined with age-related 263 neuromuscular changes, may increase the risk of fall in obese elderly (Handrigan et al., 2017; 264 Maktouf et al., 2018; Melzer and Oddsson, 2016). Further investigations are needed to clarify 265 the interactions between obesity and age-related effects on the fatigability threshold and the 266 relationship between lower limbs fatigability and altered postural control regulation in obese 267 elderly (Maktouf et al., 2018). 268

269 Limits of study

The present study provides interesting information about the possible links between 270 271 markers of the neuromuscular fatigability and postural parameters in obese adults but it is worth noting that there are limitations. First of all, only individuals with androidal obesity 272 type were taken into account. Nevertheless, the androidal obesity type exposed to a greater 273 risk of instability than the gynoidal obesity type (Cieślińska-Świder et al., 2017). This choice 274 allows us to provide indications about the underlying mechanisms of postural control 275 alterations in obese most exposed to risk of instability. Furthermore, we are aware that the 276 277 standardized position of the feet during postural control test in this study (feet together) may influence the test results, since healthy young obese show a stable position when standing 278 naturally (Cieślińska-Świder and Błaszczyk, 2019). However, the effects of muscle fatigue 279 noticed in the present work during an unnatural position can also be manifested in everyday 280 life, where regaining stability requires more effort in the obese. Moreover, we used the 281 282 standardized position described by Maktouf et al. (2018) in order to check their hypothesis regarding the relationship between neuromuscular fatigability and postural instability 283

284 Conclusion

Obesity decreased fatigability threshold and induces an early fatigue development at the ankle 285 level in obese adults. Decreased fatigability threshold could partly explain postural control 286 instability in this population when exposed to difficult standing. This also confirms that 287 altered postural control regulation during difficult tasks of daily living is not related to an 288 289 insufficient ankle muscle force production but rather to a decreased F_{cr} expressing the intensity threshold above which fatigue develops drastically in obese adults. Finally, F_{cr} could 290 be a relevant indicator to detect symptoms of fatigue in obese adults that clinician should 291 292 strongly consider in order to prevent functional limitations that may develop at an advanced age. This possibility should be explored in future research. 293

294

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299

300 Conflict of interest

- 301 The authors declare no conflict of interest.
- 302

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Figure 1. Peak force decrease over the time during fatiguing exercise of the plantar flexor muscles. The black line represents the exponential model of the kinetic of F_{peak} over the time.





Black squares present the linear relationship between the ration (length /area) and critical force in eyes opened condition. The equation of the linear regression: Fcr (%MVC) = $-0.08 \times \text{Ratio}$ (length/area) + 1.25 (R² = 0.75, r = 0.86, p<0.05).

Black circles present the linear relationship between the ration (length/ area) and critical force in eyes closed condition. The equation of the linear regression: Fcr (%MVC) = $-0.11 \times \text{Ratio}$ (length/area) + 1.26 (R² = 0.74, r = 0.85, p<0.05).

Characteristics	CG	OG	
	(n = 15)	(n = 15)	
	Mean(SD)	Mean(SD)	p - level
Age (years)	40.1 (13.9)	44.2 (12.4)	NS
Body height (m)	1.79 (0.13)	1.70 (0.09)	NS
Body mass (kg)	68.1 (13.5)	118.2 (22.6)	p<0.001
BMI (kg/m²)	21.3 (2.8)	39.1 (5.2)	p<0.001
Body fat (%)	23.6 (5.1)	49.2 (7.1)	p<0.001
Lean body mass (kg)	54.0 (11.8)	60.2 (15.1)	NS
Fat body mass (kg)	16.0 (3.9)	58.0 (13.1)	p<0.001
Waist circumference (cm)	95.4 (11.5)	138.1 (11.8)	p<0.001
Hip circumference (cm)	94.7 (6.2)	122.8 (10.9)	p<0.001
Ratio Waist / hip	1.0 (0.1)	1.1 (0.1)	p<0.05

 Table 1. Participants' physical characteristics.

Comparison between two groups based on the student's t-test for each parameter. CG: control group, OG: obese group, BMI: body mass index, NS: not significant.

10010 201 0000	Tuble 2. Tostatal parameters of participants daming postatal control testing.							
	CG	OG	Obesity	effect	Postural to	ask effect	Obesity $\times Pc$	ostural task
	Mean(SD)	Mean(SD)						
			<i>F</i> -values	p-Level	F-values	p-level	F-values	p-level
Area EO (cm ²)	2.9(1.5)	3.7(1.9)*						
Area EC (cm ²)	3.8(1.9)+	5.9(2.1)*+	$F_{(1.56)} = 56.9$	p<0.01	$F_{(1.56)} = 200.1$	p<0.001	$F_{(1.56)} = 11.1$	p<0.05
Velocity EO (mm/s)	6.2(2.2)	11.3(3.1)*						
Velocity EC (mm/s)	9.1(3.1)+	15.1(3.4)*+	$F_{(1.56)} = 170.2$	p<0.001	$F_{(1.56)} = 473.5$	p<0.001	$F_{(1.56)} = 60.2$	p<0.01
Length EO (cm)	18.7(4.2)	34.0(5.1)*						
Length EC (cm)	27.2(6.4)+	45.4(9.2)*+	$F_{(1.56)} = 155.2$	p<0.001	$F_{(1.56)} = 273.9$	p<0.001	$F_{(1.56)} = 25.2$	p<0.01
Ratio L/A EO (cm)	6.5(1.4)	9.2(2.6)*						
Ratio L/A EC (cm)	7.2(2.4)	7.7(2.1)*+	$F_{(1.56)} = 24.2$	p<0.05	-	-	$F_{(2.56)} = 15.9$	p<0.05

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

Comparison between two groups based on two-way ANOVA (group × postural task) for each parameter with additional post hoc analysis where there is a main effect of Obesity, Postural task and an interaction (Obesity × Postural task). CG: control group, OG: obese group, EO: eyes opened, EC: eyes closed, Ratio L/A: length/area

* Significant difference between CG and OG (p<0.05).
+ Significant difference between EO and EC conditions (p<0.05).

Table 3. Parameters of the neuromuscular fatigability during fatiguing exercise.

	CG	OG	
	Mean(SD)	Mean(SD)	p-Level
PF			
Absolute Force (N)	626.6(193.6)	683.0(115.6)	NS
Relative Force (N/kg)	11.6(2.0)	11.3(2.0)	NS
Fcr (%)	56.2(5.5)	44.4(6.6)	p<0.01
Fcr (N/kg)	7.0(1.6)	4.3(1.0)	p<0.01
RMSSD (N)	45.8(5.5)	67.3(9.2)	p<0.001
τ (s)	96.0(8.1)	84.7(8.1)	p<0.05
DF			
Absolute Force (N)	209.5(66.2)	237.8(60.8)	NS
Relative Force (N/kg)	3.9(0.9)	3.9(0.5)	NS
Fcr (%)	65.9(17.6)	54.4(9.6)	NS
Fcr (N/kg)	2.5(0.4)	1.8(0.3)	p<0.05
RMSSD (N)	19.3(2.4)	18.1(2.0)	NS
τ (s)	78.0(4.0)	58.4(4.3)	P<0.05

Comparison between two groups based on t-student's test for each parameter.

CG: control group, OG: obese group, Fcr (%): critical force relative to maximal voluntary contraction, Fcr (N/kg): critical force in Newton per body mass, RMSSD: index of variability, τ : rate of force decrease, NS: not significant.