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▶ To cite this version:

Waël Maktouf, Cerise Guilherme, S. Boyas, Bruno Beaune, Sylvain Durand. Relationships between lower limbs fatigability threshold and postural control in obese adults. Journal of Biomechanics, 2020, 105, pp.109819. 10.1016/j.jbiomech.2020.109819. hal-02954055

HAL Id: hal-02954055

https://hal.science/hal-02954055

Submitted on 20 May 2022

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

Wael MAKTOUF, Cerise GUILHEME C Sébastien BOYAS, Bruno BEAUNE and Sylvain DURAND

Le Mans University, Movement - Interactions, Performance, MIP, EA 4334 Faculty of Sciences and Technologies, Avenue Olivier Messiaen, 72000 Le Mans, France

Keywords: overweight; fatigue; strength, balance; ankle muscles

Corresponding author:

- **E-mail address:** waelmaktouf1@gmail.com
- Address: Avenue Olivier Messiaen, Laboratory "Movement, Interactions,
 Performance" (EA 4334), Faculty of Sciences and Technologies, Department of Sport Sciences, 72085 Le Mans, France.

E-mail addresses:

- Waelmaktouf1@gmail.com
- Cerise.Guilherme.Etu@univ-lemans.fr
- Sebastien.boyas@univ-lemans.fr
- Bruno.beaune@univ-lemans.fr
- Sylvain.durand@univ-lemans.fr

Address:

Le Mans University, Movement - Interactions, Performance, MIP, EA 4334
Faculty of Sciences and Technologies, Avenue Olivier Messiaen, 72000 Le Mans, France

Acknowledgements

We would like to thank the patients of the reeducation center François Gallouedec (Le Mans, 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.

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

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1. Introduction

A growing body of evidence suggests that obesity affects negatively the postural control regulation (Cieślińska-Świder and Błaszczyk, 2019; Corbeil et al., 2001; Handrigan et al., 2017; Hue et al., 2007; Maktouf et al., 2018; Maktouf et al., 2019). Indeed, obesity induces changes in body geometry (i.e., waist and hip circumferences) due to an excessive 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 is localized in the abdominal area) and the gynoidal obesity type (i.e., the fat is localized on the thighs and buttocks (WHO, 2008). Recently, Cieślińska-Świder et al. (2017) reported that androidal obesity type exposed to a greater risk of instability than the gynoidal obesity type. The androidal obesity is characterized by an excessive body fat at the abdominal level which induces an anterior position of the center of Mass (CoM), and its relative position to the center of foot pressure (CoP), from the axis of rotation (i.e., ankle joint assuming an inverted pendulum model, where the CoM is the controlled variable and the CoP is the controlling variable; Corbeil et al., 2001). This forward position of the CoM causes an increased gravitational torque accelerating the body forward (Corbeil et al., 2001; Simoneau and 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 weighted individuals to counteract the gravitational torque in order to restore similar balance level (Handrigan et al., 2012; Simoneau and Teasdale, 2015). This suggests that the altered postural control abilities in adults with androidal obesity could be explained by a lack of strength from the muscles mobilizing the ankle joint (Hue et al., 2007; Matrangola and Madigan, 2009). However, Maktouf et al. (2018) reported that obese individuals showed

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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.

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Several studies focused on fatigue development of the upper extremity muscles in obese adults, showing that obesity was associated with an early fatigue and a delayed recovery from isometric endurance exercise (Cavuoto and Nussbaum, 2014; Eksioglu, 2011; 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 increased body mass index altered endurance and central activation during maximal voluntary isometric contraction (MVC) of the tibialis anterior (TA) after a sustained isometric fatiguing 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 fatigability threshold of PF that are considered as the primary muscles responsible for the forward balance recovery in obese adults (Loram et al., 2004). Moreover, it is well established that a percentage of MVC did not refer to metabolic exercise intensity domains and thus to fatigue development (Kent-Braun et al., 1993; Saugen et al., 1997). The critical force (F_{cr}), which corresponds to the maximal exercise that still results in metabolic steady state, is considered as a potential alternative approach that allows identifying a threshold above which the fatigability is critically developed in healthy and pathological individuals (Poole et al., 2016; Veni et al., 2019). Thus, F_{cr} is the intensity threshold above which fatigue develops

- 51 when performing isometric contractions of single agonist muscle (Poole et al., 2016).
- However, to the best of our knowledge, no study has examined the effect of obesity on F_{cr} of
- 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 the markers of neuromuscular fatigability of ankle muscles in order to better understand the neuromuscular fatigability threshold in obese adults. Second, we aim to examine the relationships between markers of the neuromuscular fatigability and postural control parameters to provide indications about the underlying mechanisms of postural control alterations in individuals with androidal obesity. We hypothesize that obese adults have a decreased fatigability threshold and an earlier fatigue development compared to normal weighted adults and that F_{cr} is negatively correlated with altered postural control parameters in obese adults.

2. Methods

2.1.Participants

Thirty men, 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; n=15; age = 40.1(13.9) years; BMI = 23.9(2.8) kg/m²) and the obese group (OG; n=15; age = 44.2(12.4) years; BMI = 42.2(5.2) kg/m²). Only adults with androidal obesity type (i.e., obese individuals with the waist to hip ratio greater or equal to 0.85; Cieślińska-Świder et al., 2017). 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. All participants were informed about the purpose and protocol of the study and 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. 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 240-Class 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).

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 low-pass filter (n = 4; cut-off frequency = 5Hz) in order to overcome the problems associated with the clean cut transition (Paillard and Noé, 2015). Then, the signal of CoP displacements 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%-confidence ellipse (area, cm²), the velocity (velocity, mm/s) and length (length, cm) of the CoP displacements (Donath et al., 2012; Paillard and Noé, 2015; Schubert and Kirchner, 2014). The average recorded measurements of the three trials for each condition were used in 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 accuracy of the postural system indicated by the area (Paillard and Noé, 2015).

2.2.3. Muscle evaluations

The force of the ankle plantar flexor (PF) and dorsal flexor (DF) muscles of the dominant leg were measured using a dynamometer (Sauter FL1K; Type: Force Gauge; Sauter 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 order to determine the warm up intensity, (2) warm up (10 contractions at 25% of the prewarm 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-specific experimental sessions lasted 10 minutes.

For PF testing, participants were instructed to keep their back, buttock and thigh in contact with a height-adjustable chair, to keep their leg stretched horizontally and to push 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 stand up, to keep their ankle at 90° and push up with the foot on the dynamometer (Nagai et al., 2013).

2.2.3.1. Maximal voluntary contraction testing

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

2.2.3.2. Fatigability intervention

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

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**).

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

2.2.4. Data analysis

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

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

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

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

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

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

3. Result

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	3.1.	Statistical	analy	vsis
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Statistical analysis was performed using Statistica Software 13.0 (Software, Inc., Tulsa, USA). The normality and homogeneity of variance were respectively determined using the Shapiro-wilk and Levene tests. The assumption of normality and homogeneity of variance was confirmed for all parameters. The student's t-test was used for markers of fatigability and anthropometric parameters, and the two-way ANOVA (obesity × postural task) for postural parameters with the Bonferroni post-hoc procedures were performed. Relationships between the postural parameters and markers of fatigability were evaluated using Pearson's correlations analysis. Values were expressed as means (standard deviation). The level of significance was set at the 5% critical level (p<0.05).

3.2. Anthropometric measurements

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Participants' characteristics are shown in **Table 1**. Body mass, BMI, body fat, hip and waist circumferences and the ratio waist/hip were higher in OG compared to CG (p<0.05). There are no significant differences concerning LBM, age and body height between groups.

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

3.3. Static postural control testing

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 $(F_{(1.56)} = 60.2, p < 0.01, \alpha^2 = 0.71, \beta$ 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

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

3.4. Muscle evaluations

The parameters of the neuromuscular fatigability are presented in **Table 3**. The absolute and the relative of PF and DF were not significantly different between OG and CG.

For PF, the relative F_{cr} to MVC was 21% lower in obese group (p < 0.01). The F_{cr} in Newton per body mass was significantly 39% lower in OG (p < 0.01). The τ value was 12% lower (p < 0.05) and the RMSSD was 47% higher in OG (p < 0.001). For FD, only the F_{cr} in Newton per body mass (p < 0.05) and the τ (p < 0.05) remained significantly lower in OG.

3.5. Correlations analyses

Pearson correlation's analysis revealed significant correlations between the (L/A) ratio of CoP displacements and the F_{cr} PF expressed in percentage of MVC in EO (r = 0.86, p < 0.05) and EC (r = 0.85, p < 0.05) in OG (**Fig. 2**).

4. Discussion

Results point out that obesity decreases the F_{cr} expressing the intensity threshold above which fatigue develops drastically, and an early fatigue development in ankle muscles. Moreover, results indicate that F_{cr} of PF was negatively correlated with altered postural 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 non-obese adults have similar neuromuscular functioning characteristics (e.g., function of cross-section 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 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 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 fatigue development. Obese adults may thus often exceed their F_{cr} even during daily living activities especially when ankle muscles are considerably solicited (e.g., walking, balance...). In the same way, the curvature of the force-time relationship of the PF was 12% lower in OG confirming that obesity causes not only a reduced fatigability threshold but also an earlier 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 al., 2019).

It has been also reported that older adults have greater peak force variability (RMSSD) than young adults (Poole et al., 2016) due to a decreased of maximal force (Sosnoff and Newell, 2006). The present study shows that despite obese adults develop similar relative force to non-obese ones, they have higher peak force variability of PF (+47%). Hence, the force variability seems to be independent of the maximal force level in obese adults. It is 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 with the findings of Pajoutan et al. (2017) reporting that fatigue in obese adults originates rather from the central level than the peripheral level. The precise nature of this interference and the level it occurs need further investigations.

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. Indeed, DF contains approximately 27% of fast twitch fibers characterized by their low fatigue resistance (Wåhlin Larsson et al., 2008), while PF, particularly gastrocnemius 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 development, particularly at PF level. This observation confirms results of Maffiuletti et al. (2007) pointed out that obese individuals demonstrated lower fatigue resistance during voluntary extensions of quadriceps muscle.

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Otherwise, Pajoutan et al. (2016) showed a significant effect of obesity on central activation failure of dorsiflexor muscles of the ankle after a fatiguing tasks suggesting that 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 linear correlation has been evidenced between the ratio (L/A), i.e. the level of the energy spend versus the accuracy of the postural system (Paillard and Noé, 2015), and the F_{cr} expressed in percentage of MVC in EO and EC conditions (Fig.2). The more patients reported higher energy spent to regulate balance, the lower F_{cr} were found. This observation suggests that postural control alterations in obese adults could be partly explained by a decreased fatigability threshold. Similar relationship has previously been observed between increased fatigability and poor functional mobility and balance in obese subjects. (Ercan et al., 2019; Resnick et al., 2006). A possible explanation is related to mechanical changes in obese 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 rotation. This might cause a greater gravitational torque that accelerates the body in the anteroposterior axis (Corbeil et al., 2001). To maintain upright stance, this gravitational

torque must be countered by increasing muscular torque (Handrigan et al., 2012; Teasdale et 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 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 not related to an insufficient ankle muscle force production but rather to an increase in PF muscle activity (Maktouf et al., 2018). Thus, the high level of muscle activity may increase 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 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 fatigue but rather to a decreased fatigability threshold of PF (i.e. decreased Fcr). In fact, the 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 consequently lower extremity muscle fatigue development (Pajoutan et al., 2016). Accordingly, it is evident that the more the fatigability threshold of PF is decreased, the higher risk of instability could be observed in obese adults. Moreover, it is possible that the lower fatigability threshold in obese individuals, when combined with age-related neuromuscular changes, may increase the risk of fall in obese elderly (Handrigan et al., 2017; Maktouf et al., 2018; Melzer and Oddsson, 2016). Further investigations are needed to clarify the interactions between obesity and age-related effects on the fatigability threshold and the relationship between lower limbs fatigability and altered postural control regulation in obese elderly (Maktouf et al., 2018).

Limits of study

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The present study provides interesting information about the possible links between 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 type were taken into account. Nevertheless, the androidal obesity type exposed to a greater risk of instability than the gynoidal obesity type (Cieślińska-Świder et al., 2017). This choice allows us to provide indications about the underlying mechanisms of postural control alterations in obese most exposed to risk of instability. Furthermore, we are aware that the 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 naturally (Cieślińska-Świder and Błaszczyk, 2019). However, the effects of muscle fatigue noticed in the present work during an unnatural position can also be manifested in everyday life, where regaining stability requires more effort in the obese. Moreover, we used the standardized position described by Maktouf et al. (2018) in order to check their hypothesis regarding the relationship between neuromuscular fatigability and postural instability

Conclusion

Obesity decreased fatigability threshold and induces an early fatigue development at the ankle level in obese adults. Decreased fatigability threshold could partly explain postural control instability in this population when exposed to difficult standing. This also confirms that altered postural control regulation during difficult tasks of daily living is not related to an 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 be a relevant indicator to detect symptoms of fatigue in obese adults that clinician should strongly consider in order to prevent functional limitations that may develop at an advanced age. This possibility should be explored in future research.

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Acknowledgements

- We would like to thank patients of the reeducation center François Gallouedec (Le Mans,
- France) for their acceptance to participate in this study. We also thank the managers of this
- 298 institution for their collaborations.

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Conflict of interest

301 The authors declare no conflict of interest.

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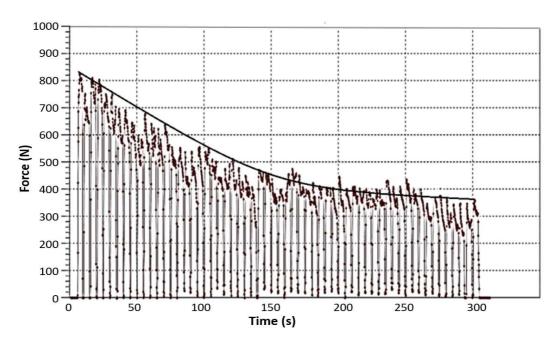


Figure 1. Peak force decrease over the time during fatiguing exercise of the plantar flexor muscles.

The black line represents the exponential model of the kinetic of F $_{\text{peak}}$ over the time.

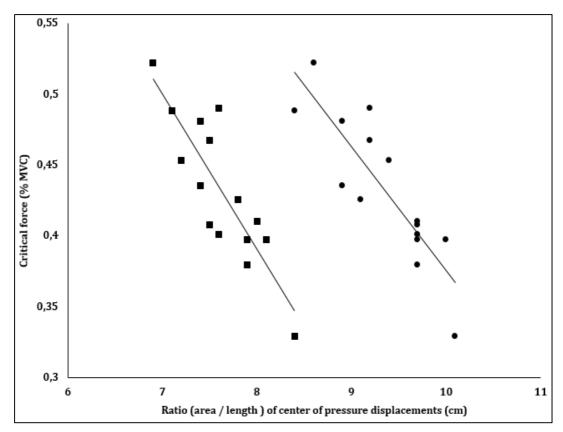


Figure 2. Correlations between Ratio (length/area) of center of pressure displacements and the critical force of plantar flexors expressed in percentage of MVC.

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× 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) = $-0.11 \times$ Ratio (length/area) + 1.26 (R² = 0.74, r = 0.85, p<0.05).

Table 1. Participants' physical characteristics.

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

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.

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

	CG Mean(SD)	OG Mean(SD)	Obesity effect Postural task effect		Obesity × Postural task			
			F-values	p-Level	F-values	p-level	F-values	p-level
Area EO (cm²) Area EC (cm²)	2.9(1.5) 3.8(1.9)+	3.7(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) Velocity EC (mm/s)	6.2(2.2) 9.1(3.1)+	11.3(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) Length EC (cm)	18.7(4.2) 27.2(6.4)+	34.0(5.1)* 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) Ratio L/A EC (cm)	6.5(1.4) 7.2(2.4)	9.2(2.6)* 7.7(2.1)*+	$F_{(1.56)} = 24.2$	p<0.05	-	-	$F_{(2.56)} = 15.9$	p<0.05

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
$\tau(s)$	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
$\tau(s)$	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.