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Interactions among obesity and age-related effects on the gait pattern and muscle activity across the ankle joint.

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Interactions among obesity and age-related effects on the gait pattern and muscle activity across the ankle joint.

Abstract:

Objective: The purposes of this study were to investigate the combined effects of age and obesity on gait and to analyze the relationship between age and obesity on ankle muscle activities during walking.

Materials and Methods: 4 groups; the young non-obese control group (CG, n=50, age=31.8±4.5 years; BMI=21.4±2.2 kg/m²), the young obese group (OB, n=30, age=35.4±4.1 years; BMI=38.6±3.5 kg/m²), the non-obese older adults group (OA, n=20, age=76.1±3.5 years; BMI=24.4±1.1 kg/m²) and the obese older adults group (OBOA, n=20, age=79.6±5.7 years; BMI=35.5±2.7 kg/m²) walked on an instrumented gait analysis treadmill at their preferred walking speed. Spatiotemporal parameters, walking cycle phases, Vertical ground reaction force (GRFv) and center of pressure (CoP) velocity were sampled from the treadmill software. Electromyography (EMG) activity of the gastrocnemius medialis (GM), the soleus (SOL) and tibialis anterior (TA) were also collected during the walking test. **A forward stepwise multiple regression analysis was performed to determine if body weight or age could predict ankle muscle activities during the different walking cycle phases.**

Results: Compared to OB, OBOA walked with higher CoP velocity, shorter stride, spending more time in support phase (p<0.05). These manifestations were associated with higher TA and SOL activities during the 1st double support (1st DS) and higher TA activity during the single support (SS) (p<0.05). Compared to OA, OBOA walked with lower GRFv, shorter and wider stride and spend more time in SU (p<0.05). Moreover, SOL, TA and GM activities of OBOA were higher compared to OAG during 1st DS, SS and 2nd Double support (2nd DS), respectively (p<0.05). **During the 1st DS, the stepwise multiple regression revealed that age accounted for 87% of the variance of TA activity. The addition of age contributed a further 16% to explain the variance TA activity. During the SS, age accounted for 64% and 46% of the variance of SOL and TA activity respectively. The addition of the body weight added further 15% and 66% of the variation of SOL and TA activity respectively. During the 2nd DS, body weight accounted for 86% of the variance and the addition of the body weight added a further 17% to explain the high level of GM.**

Conclusion: Age in obese adults and obesity in older adults should be considered separately to evaluate neuromuscular responses during walking and, subsequently, optimize the modality

of treatment and rehabilitation processes in obese individuals in order to reduce and/or prevent the risk of falls.

Keywords: aging, obesity, walking, electromyography, neuromuscular alterations

1. Introduction

Over the past decade, obesity has exhibited a dramatic increase in older adults and is known to accelerate older adults' risk for dependency (Hardy et al., 2013; Maktouf et al., 2019). Recent estimates suggest that obese older adults have a 1.5 – 5-fold risk of developing a walking limitation compared to normal weight older adults (Handrigan et al., 2017; Laroche et al., 2015). As an important prerequisite for autonomy, walking may play an important role in disease prevention and weight management in the obese older adults (Malatesta et al., 2009).

It is well established that obesity **in adults** is associated with altered postural control (Dutil et al., 2013; Handrigan et al., 2010; Hue et al., 2007; Maktouf et al., 2018 and 2020) **during quiet standing**. Recently, we point out these alterations could be related to high level of ankle muscle activities (Maktouf et al., 2018). Excessive muscle activity reduces the degrees of freedom in the postural control system (Tucker et al., 2008) and might inhibits smooth joint motion and restrict dynamic performance as walking (Allum et al., 2002; Wu, 2008). In this context, authors reported alterations in the spatiotemporal and kinetic gait parameters (Ko et al., 2010; Peyrot et al., 2009), the preferred walking speed and stride length being lower in obese adults than in normal weight adults (Ko et al., 2010). Additionally, obese adults walk with a wider stride, spending more time in double support but less time in the single support phases of gait (De Souza et al., 2005). These modifications are considered a self-optimization of their strength and functional capacities in order to increase stability and to minimize the energy cost of walking that is reported high in this population (Laroche et al., 2015; Iazzer et

al., 2003). In order to investigate the underlying mechanisms of this high level of energy cost during gait in obese adults, previous studies have investigated the external mechanical work which presents half of the total energy expenditure (Laroche et al., 2015; Peyrot et al., 2009). These authors reported that other biomechanical factors, other than the external work, might explain the origin of the high level of the energy cost in obese adults. They also suggested, but never showed, that the increased muscle activation required for both the stabilization of the center of pressure during the single support and the forward propulsion of the body may partly explain the high level of the energy cost in obese adults. Previous studies investigated the potential effect of obesity on lower limb muscle activity during walking (Hills and Parker, 1991) and adolescents (De Carvalho et al., 2012) and, subsequently, reported no difference between obese and non-obese individuals. Oppositely, other authors reported increased and prolonged activity of gastrocnemius medialis (GM) during the stance phase of gait in obese individuals with osteoarthritis (Amiri et al., 2015). However, the influence of obesity on the lower limb muscle activity and their implication in gait alterations in obese adults are still unclear.

A large proportion of age-associated falls are attributed to a decreased quality of gait due to age-related peripheral (e.g., postural control and muscle strength) (Gheno et al., 2012; Gimmon et al., 2015) and central impairments (e.g., atrophy of the motor cortical regions, and delayed muscular commands) (Geoffroy et al., 2016; Maslivec et al., 2018). In particular, the aging process strongly alters force production by ankle muscles (e.g., 16% and 38% loss of muscle mass of the dorsal and plantar flexors, respectively) (Kent-Braun, 1999; Rice et al., 1989). In response to neuro-musculoskeletal system alterations, a compensatory increase of muscle activities in the lower limbs occurs, mainly in the tibialis anterior (TA) and soleus (SOL) during the single support phase and in the GM during the first support phase in older adults (Marques et al., 2013; Schmitz et al., 2009).

Otherwise, one can assume that the combination of obesity and aging could dramatically impair the postural control of obese older adults, causing functional limitation, in particular gait ability, and possibly disposing to injuries (Handrigan et al., 2017; Wearing et al., 2006). However, literature has reported contradictory findings concerning the effect of obesity on postural control in obese older adults which could consequently affect walking. Pereira et al. (2017) reported that body mass index and fat mass do not affect balance in older adults **during a one-legged stance in older adults**, while other authors showed that obesity presents an additional constraints to age-related postural control deteriorations **during quiet standing** (Dutil et al., 2013; Mainenti et al., 2011; Maktouf et al., 2018 and 2019; Rossi-Izquierdo et al., 2015). To our knowledge, only two studies have characterized the gait pattern in obese older adults (Ko et al., 2010; Laroche et al., 2015) confirming that they spend less time in single support and more time in double support than older adults of normal weight. **These findings also appear to be contradictory to those reported by Pereira et al. (2017). This could be explained by the fact that postural control regulation during one-legged stance in quiet standing and one-legged stance during walking are different (Winter, 1995). Thus, the two-way interactions between age and obesity and the neuromuscular mechanisms by which gait alterations occur in obese older adults during walking require further investigation.**

Thus, the purpose of this study was to investigate the influence of age and/or obesity on gait parameters, with a focus on ankle muscle activities, during walking at the preferred walking speed in obese older adults. We hypothesized that: *i*) obesity-related impairments in gait pattern are associated with an increase in muscle activities across the ankle joint; *ii*) age increases the obesity-related gait and alterations in muscle activity patterns; and *iii*) obesity exacerbates the age-related gait and alteration in muscle activity patterns.

2. Materials and methods

2.1. Study design

This study is an analytical cross-sectional study (**Figure. 1**). The experimental period lasted 3 months. The course of the study consisted of three periods: a 1-3 weeks recruitment period, 1-2 weeks screening period, and 7 weeks experimental testing. The experimental protocol lasted between 60-75 min and was composed of 4 assessments: (1) anthropometric measurements (2) walking test across a 20-meter corridor (3) the walk on the treadmill test (4) maximal voluntary contraction test.

-----**Figure. 1**-----

2.2. Participants

2.2.1. Estimation of sample size

The sample size was calculated using the freeware G*Power (version 3.1.9.4) (Faul et al., 2007). The ANOVA test (fixed effects, main effects and interactions) was predefined for power analysis. The estimation was based on predefined control of type I error ($\alpha = 0.05$) and Type II error ($\beta = 0.80$), with a moderate level of estimated effect size ($r = 0.5$). Under these settings, 48 participants were required as the minimum sample size.

2.2.2. Recruitment

One hundred and twenty volunteers were recruited in the current study, divided into 4 groups (**Table 1**) according to their age and their body mass index (BMI, kg/m^2): the young non-obese control group (CG, $n=50$, $\text{age}=31.8\pm 4.5$ years; $\text{BMI}=21.4\pm 2.2$ kg/m^2), the young obese group (OB, $n=30$, $\text{age}=35.4\pm 4.1$ years; $\text{BMI}=38.6\pm 3.5$ kg/m^2), the non-obese older adults group (OA, $n=20$, $\text{age}=76.1\pm 3.5$ years; $\text{BMI}=24.4\pm 1.1$ kg/m^2) and the obese older adults group (OBOA, $n=20$, $\text{age}=79.6\pm 5.7$ years; $\text{BMI}=35.5\pm 2.7$ kg/m^2). The CG was recruited from our university, the OB was recruited from an obesity treatment center and, the OA and OBOA were recruited from a retirement home. The 4 groups were matched for

average of age and sex ratio. All participants were invited to complete a survey questionnaire in order to verify comorbidities, chronic diseases, falls history, the use of medication, physical independence and levels of frailty. For the OB, OA and OBOA, these informations were also controlled by the medical staff of the structures. Physical activity level and cognitive score were assessed using the Ricci and Gagnon (Reichhart et al., 2016) and the Montreal Cognitive Assessment (MoCA) test (Ciesielska et al., 2016). The inclusion criteria for all groups were: $25 > \text{age} > 40$ years for CG and OB, and > 65 years for OA and OBOA, ability to verbally communicate with the experimenters and to be physical independent. The common exclusion criteria for all groups were: without neurological or cognitive impairments, without severe cardiovascular problems or severe musculoskeletal deformity and injuries of the lower limb, without comorbidities or chronic diseases, no use of medication that would interfere with testing, no falls history in the last three months, with a score < 26 in MoCA test. Before the experiment was initiated, each participant gave his written informed consent. The study was conducted according to the declaration of Helsinki and was approved by the local ethics committee including the head of staff and the medical staff of the structures (agreement: 2018/01/05).

2.3. Gait analysis

2.3.1. Procedure

Participants were asked first to complete three walking trials across a 20-meter corridor to determine their averaged preferred walking speed. The first and the last 5 meters were considered respectively as acceleration and deceleration distances. Thus, only the speed between the 5th and the 15th meters was measured in order to avoid to take into account the acceleration phase and to prevent any early deceleration from the participants. Then, they were familiarized to walk on an instrumented gait analysis treadmill (Zebris; FDM-T, sampling rate: 100 Hz; Zebris medical GmbH, Isny, Germany) for five minutes at their pre-

calculated preferred walking speed (Lavcanska et al., 2005), followed by a two-minute seated rest period. If participants had difficulties to walk normally on the treadmill, two additional minutes of training were proposed. At last, participants completed three one-minute trials, each separated by a five-minute seated rest period.

2.3.2. Data recording

The data were collected for a period of 10 seconds, starting at the 10th second of each trial. The gait parameters were sampled from the treadmill force plates at 100 Hz by the treadmill software (Zebris, Suite FDM-T) (Braun et al., 2015; Fan et al., 2016). Those parameters included the spatiotemporal parameters, the walking cycle phases, the vertical ground reaction force (GRFv) and the CoP displacements. The spatiotemporal parameters used in this study were the step length and width and the stride length (cm). The treadmill software divided the walking cycle into two phases, namely the support phase (SU, %) and the swing phase (SW, %). The SU consisted of the 1st double support (1st DS, %), a single support (SS, %) and the 2nd double support (2nd DS, %). Absolute GRFv peaks were used to calculate peaks 1 and 2 of the relative GRFv (P1, P2, N/kg, respectively) by dividing the absolute value of the GRFv peak by the body mass (**Fig. 1**). Displacements of the CoP data were used to extract the CoP length during the SU and SS (mm), the anteroposterior position of the CoP (mm) and the CoP velocity (cm/s) during walking.

-----**Figure. 2**-----

2.4. Electromyography recording

2.4.1. Procedure

Electromyographic (EMG) data from the ankle joint muscles were recorded firstly during the maximum voluntary isometric contraction (MVC) of the plantar and dorsal flexors, and then, during the walk on the treadmill test using the Powerlab 16/35 system (Powerlab 16/35; sampling rate: 1000 Hz, ADInstruments, Dunedin, New Zealand). The EMG recording

was time-synchronized with the treadmill data using a control device operating according to the principle of a synchronization switch system (ON/OFF). **Before the walk on the treadmill test, participants were instructed to perform maximal voluntary concentric contractions (MVC) of the plantar and dorsal flexors muscles.** For 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 (Maktouf et al., 2018). Strong verbal encouragement was given every contraction to promote maximal effort. For each condition, three 5-seconds trials were performed with a 2 min rest in-between.

During the MVC and walking on treadmill tests, two unipodal surface electrodes (Uni-gel Single Electrode-T3425, Thought Technology Ltd, Montreal, Canada) were placed on three ankle joint muscles: the gastrocnemius medialis (GM), the soleus (SOL) and the tibialis anterior (TA) of the dominant leg. Before attaching the electrodes, the skin was carefully shaved and cleaned using an abrasive cleaner and alcohol swabs to reduce impedance. The placement and location of the surface electrodes, on the belly of each muscle, in parallel to muscle fibers orientation and with an inter-electrode intervals of 20 mm, conformed to the recommendations of SENIAM (Surface EMG for Noninvasive Assessment of Muscles) (Hermens, 2000).

2.4.2. Data analysis

The analysis of EMG data collected during walking on the treadmill and MVC tests were post-processed using Matlab software (Matlab R2013a, MathWorks, Natick, USA), and the raw EMG signals were band-pass filtered at 15–500 Hz through a second-order Butterworth digital filter to remove noise or movement interference (Luca et al., 2010). The data from 10 strides were collected for a period of 10 seconds, in the 10th second of each trial

(i.e., the same period of the treadmill recording), and were rectified and smoothed using root mean square analysis (RMS) with a 20-ms window (Pouliquen et al., 2018; Ball and Scurr, 2010), calculated using the following equation (Hermens, 2000):

$$\text{RMS}_{(t_0)} = \sqrt{\frac{1}{T} \int_{t_0-T/2}^{t_0+T/2} (\text{EMG})^2 dt} \text{ , where T is the time of integration}$$

For the MVC tests, a moving window with a width of 20 ms was used to find the maximum RMS EMG activity resulting from the three efforts of MVC for each kind of contraction. Then, all RMS EMG data of walking on the treadmill test were normalized using following equation for each muscle:

$$\text{EMG RMS\%} = [(\text{RMS EMG during walking} / \text{RMS EMG MVC}) \times 100\%]$$

The normalized RMS of the GM (RMS GM), SOL (RMS SOL) and TA (EMG TA) of each walking cycle's phase were used in the study.

2.5. Statistical analyses

Statistical analyses were performed using Statistica Software 13.0 (Software, Inc., Tulsa, OK). The normality and homogeneity of variance were respectively determined using the Kolmogorov–Smirnov and Levene tests. Since parametric assumptions were met, a factorial 2×4 ANOVA (age × obesity) with Bonferonni post hoc test's (Bonferonni procedure) was applied. A forward stepwise multiple regression analysis was performed to determine if body weight or age could predict ankle muscle activities during the different walking cycle phases. Values are expressed as means (± standard deviation). The level of significance was set at the 5% critical level (p < 0.05). In order to determine the magnitude of changes, the effect size (r) was calculated based on the values of d (Glass & Hopkins, 1996) from group-comparisons and significance. The effect size was characterized by Cohen (1988) as weak,

moderate and strong effects, i.e., $d = 0.2$ is small, $d = 0.5$ medium and $d = 0.8$ large, respectively.

3. Results

3.1. Analysis of variance

3.1.1. Anthropometric measurements

Anthropometric characteristics are shown in **Table 1**. Obesity induces an increased body mass, waist and hip circumference, and FBM in young ($p < 0.001$; $r = 0.8$) and older adults ($p < 0.001$; $r = 0.7$). Age decreases LBM in non-obese and obese older adults ($p < 0.001$; $r > 0.7$). However, OB showed higher LBM than CG ($p < 0.05$; $r = 0.59$).

-----**Table 1**-----

3.1.2. Gait parameters

Spatiotemporal parameters and walking cycle phases are shown in **Table 2**. Step length and width, stride length, SU and Swing phases were lower in OB and OBOA ($p < 0.05$; $r > 0.6$) suggesting that obesity altered spatiotemporal parameters of gait in both young and older adults. These observations confirm previous studies reporting the same findings in young obese (Benedetti et al., 2009; DeVita and Hortobágyi, 2003) and in obese older adults (Ko et al., 2010; Laroche et al., 2015). When comparing CG vs. OA ($p < 0.01$; $r > 0.7$) and OB vs. OBOA ($p < 0.05$; $r > 0.6$), results showed that age was also associated with lower spatiotemporal parameters. Thus, age similarly altered spatiotemporal parameters in non-obese and obese older adults. Particularly, the SS and the 2nd DS phases were lower in OB vs. CG ($p < 0.05$; $r = 0.6$), in OBOA vs. OB ($p < 0.01$; $r = 0.8$) and in OBOA vs. OA ($p < 0.05$; $r > 0.5$) suggesting that these walking cycle phases were affected by both obesity and age (Laroche et al., 2015).

-----**Table 2**-----

Center of pressure displacements and vertical ground reaction force are shown in **Table 3**. Both age and obesity were associated with high CoP velocity when walking ($p < 0.05$; $r > 0.5$). This suggests these two phenomena increase postural instability not only during static postural control tasks (Dutil et al., 2013; Maktouf et al., 2018 and 2019) but also during gait. In addition, OBOA showed lower CoP velocity compared to CG ($p < 0.05$; $r > 0.9$) OA ($p < 0.05$; $r > 0.5$) and OB ($p < 0.01$; $r > 0.7$) suggesting that obese older adults were well disadvantaged by both age and obesity. Otherwise, the relative P1 and P2 of GRFv were lower in OA vs. CG ($p < 0.01$; $r > 0.7$) and in OBOA vs. OB ($p < 0.05$; $r > 0.6$) reflecting the negative effect of age on strength in obese and non-obese adults (Toda et al., 2015). However, obesity affects only the relative P2 and only in older adults ($p < 0.05$; $r = 0.5$).

-----**Table 3**-----

3.1.3. Electromyography recording

EMG muscle activities during walking phases are shown in **Table 4**. During the 1st DS, age increases EMG TA and EMG SOL in both obese and non-obese adults (Schmitz et al., 2009) ($p < 0.01$; $r > 0.6$). However, obesity increases EMG SOL only in older adults ($p < 0.05$; $r > 0.5$). Thus, the interaction of age and obesity induces only the increase of SOL activity during the 1st DS. During the SS, age increases EMG TA in both obese and non-obese adults ($p < 0.01$; $r > 0.6$) and EMG SOL only in non-obese adults ($p < 0.05$; $r > 0.5$) (Marques et al., 2013; Schmitz et al., 2009). Obesity also increases EMG SOL but only in young adults ($p < 0.01$; $r > 0.6$). In addition, the interaction of age and obesity was only observed on EMG TA ($p < 0.05$; $r > 0.6$) suggesting that the TA muscle is high solicited in obese older adults in the SS phase. During the 2nd DS, EMG GM was higher in OG vs. CG ($p < 0.05$; $r > 0.5$) and in OBOA vs. OA ($p < 0.05$; $r > 0.5$) suggesting that obesity is associated with an increased GM activity in both young (Amiri et al., 2015) and older adults. In addition, age and obesity separately induces a high EMG SOL ($p < 0.05$; $r > 0.4$) but there was no interaction of effects

of age and obesity. Finally, Age and obesity do not affect ankle muscle activities during the swing phase.

Table 4

3.2. Multiple regression analysis

Table 5 presents results of the stepwise multiple regression analyses for ankle muscle activities during the different walking cycle phases.

During the 1st DS, the stepwise multiple regression indicated that, age was the highest predictive factor of TA activity. It accounted for 87% of variation of TA activity. Adding body weight contributed to explain an additional fraction of 16% of the variance of TA activity. The final model explained 77% of the variance of TA activity ($r = 0.88$).

During the SS, the stepwise multiple regression indicated that, body weight was the highest predictive factor of TA activity. It accounted for 66% of variation of TA activity. Adding age contributed to explain an additional fraction of 46% of the variance of TA activity. The final model explained 63% of the variance of TA activity ($r = 0.79$). In the same walking phase, the stepwise multiple regression indicated that, age was the highest predictive factor of SOL activity (64%). Adding body weight contributed to explain an additional fraction of 15% of the variance of TA activity. The final model explained 43% of the variance of TA activity ($r = 0.65$).

During the 2nd DS, the stepwise multiple regression indicated that, body weight was the highest predictive factor of GM activity. It accounted for 86% of variation of GM activity. Adding age contributed to explain an additional fraction of 17% of the variance of GM activity. The final model explained 71% of the variance of TA activity ($r = 0.84$). In the same walking phase, the stepwise multiple regression indicated that, body weight was the highest predictive factor of SOL activity (62%). Adding age contributed to explain an additional

fraction of 51% of the variance of GM activity. The final model explained 62% of the variance of TA activity ($r = 0.79$).

-----**Table 5**-----

4. Discussion

Results point out that obesity-related impairments in the gait pattern are associated with an increase in ankle muscle activities, especially during the support phase. The combined effect of age and obesity was responsible for cumulative gait pattern alterations, but through different mechanisms at neuromuscular level compared to their individual effects.

4.1. Effect of obesity on gait pattern and neuromuscular adaptation

In contrast to previous studies investigating lower limb muscle activity in children and adolescents, that have reported no significant effect of obesity on EMG activity (Blakemore et al., 2013; de Carvalho et al., 2012; Hills and Parker, 1991), we identified that the kinematic alterations in obese adults were associated with increased muscle activity during the walking support phase. However, this findings were consistent with the study of Amiri et al. (2015) reporting a prolonged of GM during the same phase. The age categories in study populations and also the differences in analysis techniques, may explain our different findings. These adaptive neuromuscular responses could be explained by the biomechanical modifications of gait related to the mobilization of excessive body mass. Indeed, DeVita and Hortobágyi (2003) reported that joint torques and powers were 88% and 61% higher respectively in obese individuals compared to non-obese, suggesting that obese adults reorganize their neuromuscular function in response to the excessive body mass to mobilize during walking. The most significant finding of this study was the identification of body weight as a strong predictive factor of the high level of GM activity. Given that GM is engaged for forward propulsion of the body (Murray et al., 1978; Sutherland, 2002), an excessive body mass

induces an amplified stimulation of PF to ensure that GM will be able to manage a greater body mass during propulsion. This is in accordance with DeVita and Hortobágyi (2003) who also reported that knee flexion decreases by 12% and plantar flexion increases by 11% in obese adults when walking. Thus, taken all together, these results indicate that obese adults promote an adapted ankle gait strategy, which partly explains the high level of GM activity during the propulsion phase.

Furthermore, high ankle muscle activity could be also explained by failure mechanisms of postural control regulation during walking. Indeed, obesity is associated with increased postural instability during walking. SOL and TA are mainly considered as stabilizing muscles of the foot during the SS phase (Sutherland, 2001). During this phase, body weight was a strong predictor of the high level of TA activity. This finding gives evidence that obese adults increase the solicitation of ankle muscles in order to reduce postural instability during the unipodal position by enhancing dynamic joint stability, possibly as a response to an excessive body weight (McGraw et al., 2000; Menegoni et al., 2009). It has been reported previously that this postural instability is most likely related to deteriorations of sensory integration mechanisms and, consequently, proprioceptive capacities (Hills et al., 2001a; Hue et al., 2007; Wu and Madigan, 2014). Indeed, plantar mechanoreceptors participate to postural control feedback response to body oscillations. Obese individuals showed larger plantar contacts areas and greater mean pressure during postural control tasks than non-obese ones (Hills et al., 2001b). Thus, these manifestations may reduce the quality and/or quantity of the sensory information arising from the plantar mechanoreceptors (Hue et al., 2007; Wu and Madigan, 2014).

The observed adaptive neuromuscular strategies may be taken as a self-optimization by which obese adults can operate within their strength and functional capacities to manage the propulsion of the body and increase stability. However, higher muscle activity is associated

with higher energy cost (Gottschall and Kram, 2003; Peterson and Martin, 2010). This is in accordance to Lazzer et al. (2003) that reported a 71–84% higher energy cost of walking in obese adults than in normal weighted adults. In order to understand the origins of such a high energy cost, previous studies (Laroche et al., 2015; Peyrot et al., 2009) investigated the external mechanical work in obese adults and estimated that this parameter represented over one half of the total energy cost (Grabowski et al., 2005). Thus, these authors concluded that the high energy cost in obese adults might be explained rather by the increased muscle activities required for both the stabilization of the CoP during the SS phase or the forward propulsion of the body than external work only. The high level of muscle activities observed in obese adults in this study confirms such a hypothesis. All these results give evidence that obese adults walk with shorter steps in order to reduce the energy cost of walking probably induced by the high level of ankle muscle activities.

4.2. Effect of age on gait pattern and neuromuscular adaptation in obese individuals

To our knowledge, our study is the first one that explored the effect of age on gait pattern and neuromuscular adaptation in obese individuals. The most significant findings were: First, age similarly affects kinematics gait parameters in non-obese and obese older adults. Second, gait alterations in obese older adults were associated with neuromuscular adaptations different from those observed in non-obese older adults.

Generally, during the 1st DS, the TA is described as the most solicited muscle ensuring amortization of the body mass (Sutherland, 2001). During this phase, compared to obesity age was identified as a stronger predictor of the increase of TA activity. Thus, this adaptive neuromuscular response could be explained better by the age-related muscle weakness or detraining (Donath et al., 2015; Laughton et al., 2003), than by excessive body mass. Obese older adults may increase TA activity likely to counteract age-related muscle weakness or to

support excessive body mass. Accordingly, Kent-Braun (1999) reported a 16% loss of the ankle DF in older adults compared to young adults. However, studies showed that aging does not affect TA activity during the 1st DS compared to normal weighted individuals (Bailey et al., 2018; Schmitz et al., 2009). Thus, it seems that the increased TA activity during the 1st DS is a specific age-related neuromuscular adaptation in obese individuals. Furthermore, Schmitz et al. (2009) concluded that the age effect was more prominent for biarticular than uniaxial muscles. While results of this study report only differences in TA and SOL activities during the 1st DS and SS phases between obese adults and obese older adults, we suggest that monoarticular muscles may see larger age-related changes at preferred speed gait in obese individuals. As observed in obese older adults, it is likely that excess weight acts as an overload stimulus on the lower limb muscles, specifically the GM (Tomlinson et al., 2014). Theoretically, this stimulus is considered as a strength training stimulus, whereby the more the load increases, the more strength is gained (Erskine et al., 2010). As a whole, our results enforce the hypothesis that age-related adaptive neuromuscular responses differ between normal weighted and obese adults. They also suggest that the effects of aging and obesity on neuromuscular pattern during gait are not necessarily cumulative (Tomlinson et al., 2014).

4.3. Effect of obesity on gait pattern and neuromuscular adaptation in older adults

Results of the previous study indicate that obese older adults are disadvantaged by both age and obesity presented two-fold more gait alterations than normal weight individuals. These alterations are associated with high level of ankle muscle activities, as indicated by higher SOL activity during the 1st DS. This finding could be explained by the results of Ko et al. (2010), stating that obese older adults have an higher ankle plantar flexion duration during the 1st DS to improve the absorption of additional impact imposed by excessive body mass. However, the stepwise multiple regression revealed that body weight accounted only for 20% of the variation of SOL activity and the addition of age contributed a further 36% to explain

the variation of SOL activity. Others variables such as the type of obesity (i.e., android and gynoid) could explain the increase of the SOL activity in obese older adults. Further investigations are needed in order to well understand the relationship between Body fat distribution and neuromuscular adaptation during walking. We were also surprised to find no difference in SOL activity between obese and non-obese adults, considering that obesity affects SOL in older adults but not in adults during the 1st DS. Therefore, we conclude that effects of obesity on the neuromuscular responses are different between young and older adults. In addition, the stepwise multiple regression revealed that age accounted for 87% of the variation of SOL activity and the addition of body weight contributed a further 16% to explain the variation of TA activity. This result could well explain the absence of a significant difference concerning the TA activity between obese and non-obese older adults during the 1st DS, suggesting that obesity in older adults do not affect neuromuscular capacity of TA during this walking phase.

Otherwise, obesity in older adults is associated with high TA activity and increased, CoP velocity during the SS phase. It seems that the solicitation of the ankle muscle, especially TA, is increased in obese older adults to reduce instability during walking. Since, age contributes to more than 87% of the variance of GM and only 16% for body weight, we can suggest that increased TA activity is rather related to age than excessive body mass. Consequently, this neuromuscular adaptation could be explained by age-related insufficient force production (Frontera, 2017).

During the propulsive phase of walking, the stepwise multiple regression revealed that body weight accounted for 87% of the variation of GM activity and the addition of age contributed a further 17% to explain the variation of GM activity. These findings point out that the solicitation of PF increases in obese older adults to manage the propulsive movement of greater body mass. In this context, results revealed that the interaction of age and obesity is

responsible for the decrease of the P2 of the GFRv. However, in a previous study (Maktouf et al., 2018), we did not observe any difference between obese and non-obese older adults concerning the maximal force of ankle muscles. It turns out that obese older adults were not able to produce an adequate force relative to their body mass during the forward propulsion. Thus, increased GM activity is rather related to excessive body mass than age. **Moreover, in the same phase both Age (51%) and body weight (62%) are predictors of the variation of SOL activity.** According to Rice et al. (1989) that noted a 38% loss of ankle PF mass in individuals over 65 years, we can propose that the increased SOL activity is explained by age-related insufficient force production (Frontera, 2017) **and also by the insufficient force production needed to stabilize the excessive body mass during the forward propulsion.** **However, no significant difference was revealed between obese and non-obese older adults, suggesting that obesity did not affect neuromuscular strategy during the propulsive phase in older adults as opposed to younger obese.**

All of the above results confirm that the age effect was more prominent for uniarticular muscles (e.g., TA during the 1st DS) than biarticular muscles (e.g., GM during the 2nd DS) (Schmitz et al., 2009). However, the neuromuscular adaptation related to the interaction of age and obesity effects induces greater changes in both monoarticular and biarticular muscles at preferred walking speed. These findings may explain why the duration of the SU phase is reduced in obese older adults. In fact, we can consider the gait modifications observed in this group as compensatory strategies to minimize the energy cost related to the high level of ankle muscle activities (Peterson and Martin, 2010).

4.4. Methodology for prescription

The prevalence of falls, fall-related injuries, and obesity has increased over the last decade (Handrigan et al., 2017). Although obese older adults have increased functional impairments, methodology of rehabilitation and treatment of this population remains unclear.

This could be explained by the lack of explanations of the underlying mechanisms of functional capacities impairments. This study may offer clinician insights into the methodology of rehabilitation and treatment of gait pattern alterations in order to enhance functional capacities. Consequently, this will help prevent the risk of falls in obese older adults. In addition, this study specifies that neuromuscular adaptation related to the combined effect of age and obesity seems to be different from those related to age or obesity separately. **Furthermore, the physical prescription** of gait rehabilitation in obese older adults should be different from the one set up for obese adults or non-obese older adults.

4.5. Limits of the study

The first critical caveat of this study is about the use of IMC as an obesity criteria. Indeed, the classification of obese individuals was made according to three evaluations: BMI, waist and hip circumferences, and bio-impedance meter measurements. The three methods gave consistent results, confirming the obese status of those classified as obese individuals. Second, it is well known that the quality of the myoelectric signal could be affected by thickness of the subcutaneous fat as observed in obese individuals (Farina et al., 2002). However, Minetto et al., (2013) confirmed that the detection of surface EMG is feasible in obese subjects presenting increased thickness of the subcutaneous fat tissue. **In addition, the normalization procedure as did in the present study help to control this bias and valid to comparison between different groups.**

5. Conclusion

Our findings indicate that both age and obesity result in cumulative gait pattern alterations, but through different mechanisms at neuromuscular level. EMG analyses of ankle muscle activities point out that the interaction of the influence of age and obesity during walking is different from those related to the individual effects of age and obesity. In other words, age in obese adults and obesity in **older adults** should be considered separately to

evaluate neuromuscular responses during walking and, subsequently, **optimize the modality of treatment and rehabilitation processes in obese older adults in order to reduce and/or prevent the risk of falls.**

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Table 1. Participants' physical characteristics

Characteristics	CG	OB	OA	OBOA	Age effect		Obesity effect		Interaction age × obesity	
	(n = 50) Mean ± SD	(n = 30) Mean ± SD	(n = 20) Mean ± SD	(n = 20) Mean ± SD	F	p	F	p	F	p
Age (years)	31.8 ± 4.5	35.4 ± 4.1	76.1 ± 3.5*	79.6 ± 5.7*	256.1	p<0.001	-	NS	-	NS
Body height (cm)	171.0 ± 6.5	162.3 ± 5.8	165.7 ± 4.9	160.3 ± 4.5	-	NS	-	NS	-	NS
Body mass (kg)	64.7 ± 5.9*	109.0 ± 10.5*	67.4 ± 4.9*	91.7 ± 6.7*+	89.7	p<0.001	226.9	p<0.001	149.5	p<0.001
BMI (kg/m ²)	21.4 ± 2.2	38.6 ± 3.7*	24.4 ± 1.1	35.8 ± 2.7*+	110.7	NS	202.9	p<0.001	129.9	p<0.001
Body fat (%)	13.8 ± 3.4	34.8 ± 6.7*	24.4 ± 3.4*	38.6 ± 3.5*+	99.5	p<0.01	248.7	p<0.001	89.5	p<0.001
FBM (kg)	8.9 ± 2.9	37.9 ± 7.4*	16.5 ± 4.2*	34.6 ± 3.8*+	91.9	p<0.001	221.9	p<0.001	79.5	p<0.001
LBM (kg)	55.8 ± 5.5	71.1 ± 5.1*	50.9 ± 6.4*	53.1 ± 4.4* ϵ	119.5	p<0.001	19.7	p<0.05	-	NS
Waist circumference (cm)	71.4 ± 8.9	109.8 ± 6.0 *	68.7 ± 3.7	96.8 ± 6.8*+	87.5	p<0.001	189.7	p<0.001	119.5	p<0.001
Hip circumference (cm)	81.7 ± 6.7	104.4 ± 8.9*	76.5 ± 4.5	99.8 ± 5.1*+	-	NS	109.4	p<0.001	-	NS

Comparison between 4 groups based on two-way ANOVA for each parameter with additional post hoc analysis (Bonferroni procedure) when there is a main effect of obesity, age and an interaction (age × obesity). CG: control group, OB: obese group, OA: older adults group, OBOA: obese older adults group, BMI: body mass index, FBM: fat body mass, LBM: lean body mass, SD: standard deviation.

NS: no significant difference.

*= Significant difference between CG and other groups (p < 0.05).

+ = Significant difference between OA and OBOA (p < 0.05).

ϵ = Significant difference between OB and OBOA (p < 0.05).

Table 2. Participants' spatiotemporal parameters.

	CG (n = 50) Mean ± SD	OB (n = 30) Mean ± SD	OA (n = 20) Mean ± SD	OBOA (n = 20) Mean ± SD	Age effect				Obesity effect				Interaction age × obesity				
					F	p	α^2	β	F	p	α^2	β	F	p	α^2	β	
Step length (cm)																	
Left	67.5±5.5	53.8±11.1*	38.3±13.0*	22.1±5.4*+€	417.3	p<.001	0.80	1	138.4	p<.001	0.56	1	18.1	p<.001	0.14	0.98	
Right	66.5±5.3	50.4±11.1*	39.4±13.4*	24.6±4.6*+€	420.7	p<.001	0.81	1	133.5	p<.001	0.57	1	18.5	p<.001	0.15	0.98	
Stride length (cm)	133.8±10.5	106.6±13.7*	77.3±26.1*	46.6±9.4*+€	423.5	p<.001	0.81	1	135.6	p<.001	0.59	1	19.0	p<.001	0.14	0.98	
Step width (cm)	8.6±2.6	12.0±3.0*	10.7±3.9*	13.2±2.3*+€	116.4	p<.001	0.52	1	159.5	p<.001	0.60	1	58.8	p<.001	0.35	1	
Walking cycle																	
Support phase (%)																	
Left	64.7±1.9	66.8±1.9*	68.9±5.2*	74.9±2.9*+€	93.6	p<.001	0.46	1	40.5	p<.001	0.27	1	9.8	p<.01	0.10	0.80	
Right	64.6±1.9	67.3±1.9*	69.3±5.8*	74.3±5.2*+€	83.9	p<.001	0.52	1	48.5	p<.001	0.29	1	10.6	p<.01	0.15	0.81	
1 st double support (%)																	
Left	14.6±2.0	16.9±1.6	21.0±2.0*	26.1±4.7*€	17.5	p<.001	0.14	1	-	NS	-	-	-	NS	-	-	
Right	14.7±1.8	17.1±1.8	21.3±3.6*	25.9±3.6*€	17.1	p<.001	0.15	1	-	NS	-	-	-	NS	-	-	
Single support (%)																	
Left	35.4±1.9	32.7±1.9*	26.9±2.7*	23.6±6.9*+€	93.6	p<.001	0.46	1	40.5	p<.001	0.28	1	9.9	p<.05	0.10	0.87	
Right	35.3±1.9	33.2±2.0*	26.4±3.4*	23.5±5.6*+€	91.5	p<.001	0.45	1	41.2	p<.001	0.26	1	10.1	p<.05	0.12	0.86	
2 nd double support (%)																	
Left	14.8±1.8*+	17.1±1.8*	21.5±4.3*	24.0±3.8*+€	227.3	p<.001	0.68	1	52.2	p<.001	0.21	1	6.9	p<.01	0.10	0.74	
Right	14.6±2.0*+	17.0±1.6*	21.1±3.7*	24.7±4.5*+€	220.5	p<.001	0.67	1	51.7	p<.001	0.22	1	7.1	p<.01	0.09	0.73	
Swing phase (%)																	
Left	35.3±1.9	33.2±1.9*	30.9±5.6*	25.1±4.0*+€	172.5	p<.001	0.28	1	20.1	p<.001	0.17	1	7.5	p<.01	0.11	0.78	
Right	35.4±2.0	32.7±1.9*	29.7±5.9*	25.7±5.1*+€	170.4	p<.001	0.30	1	20.5	p<.001	0.16	1	8.1	p<.01	0.15	0.74	

Comparison between 4 groups based on two-way ANOVA for each parameter with additional post hoc analysis (Bonferroni procedure) when there is a main effect of obesity, age and an interaction (age × obesity). CG: control group, OB: obese group, OA: older adults group, OBOA: obese older adults group, SD: standard deviation.

NS: no significant difference.

*= Significant difference between CG and other groups (p < 0.05).

+ = Significant difference between OA and OBOA (p < 0.05).

€ = Significant difference between OB and OBOA (p < 0.05).

Table 3. Center of pressure and vertical ground reaction force parameters.

	CG (n = 50) Mean ± SD	OB (n = 30) Mean ± SD	OA (n = 20) Mean ± SD	OBOA (n = 20) Mean ± SD	Age effect				Obesity effect				Interaction age × obesity			
					F	p	α ²	β	F	p	α ²	β	F	p	α ²	β
CoP parameters																
Length in SU (mm)																
Left	161.1±21.3	184.7±32.4	147.9±38.2	117.7±28.8	44.5	p<.001	0.30	1	-	NS	-	-	19.9	p<.001	0.16	0.98
Right	161.4±15.1	182.5±13.8	152.1±40.5	120.4±15.1	45.5	p<.001	0.29	1	-	NS	-	-	20.1	p<.001	0.15	0.98
Length in SS (mm)																
Left	113.5±15.3	97.5±24.6*	67.7±39.5*	39.6±14.1*+€	116.1	p<.001	0.51	1	34.6	p<.001	0.24	1	-	NS	-	-
Right	113.8±13.3	97.2±24.4*	79.6±42.2*	39.4±14.1*+€	114.2	p<.001	0.52	1	159.5	p<.001	0.60	1	-	NS	-	-
Anteroposterior position (mm)	169.1±14.5	150.2±17.6*	168.5±35.0	147.8±25.1*+	-	NS	-	-	19.6	p<.001	0.15	1	-	NS	-	-
Maximal velocity (cm/s)	65.5±15.5	97.7±31.2*	117.8±27.2*	158.5±26.5*+€	121.7	p<.001	0.53	1	57.9	p<.001	0.34	1	12.2	P<.05	0.17	0.89
GRFv parameters																
Relative P1 (N/kg)																
Left	10.4±1.1	10.1±1.2	9.2±2.3*	9.4±1.5*	32.5	p<.001	0.28	0.91	-	NS	-	-	-	NS	-	-
Right	10.2±1.0	9.9±1.2	9.4±2.7*	9.5±1.4*	31.5	p<.001	0.29	0.95	-	NS	-	-	-	NS	-	-
Relative P2 (N/kg)																
Left	11.0±2.7	10.5±1.2	10.0±2.1*	9.3±1.2*+€	7.3	p<.01	0.10	0.75	-	NS	-	-	10.1	p<.01	0.18	0.78
Right	10.7±2.5	10.2±1.6	9.5±2.2*	9.0±1.5*+€	7.5	p<.01	0.11	0.76	-	NS	-	-	10.2	p<.01	0.19	0.68

Comparison between 4 groups based on two-way ANOVA for each parameter with additional post hoc analysis (Bonferroni procedure) when there is a main effect of obesity, age and an interaction (age × obesity). CG: control group, OB: obese group, OA: older adults group, OBOA: CoP: center of pressure, GRFv: vertical ground reaction force, Sph: support phase, SS: single support, SD: standard deviation.

NS: no significant difference.

*= Significant difference between CG and other groups (p < 0.05).

+ = Significant difference between OA and OBOA (p < 0.05).

€ = Significant difference between OB and OBOA (p < 0.05).

Table 4. Normalized RMS EMG of ankle muscle joint during walking cycle phases.

Walking cycle phases	Muscle	CG	OB	OA	OBOA	Age effect				Obesity effect				Interaction age × obesity			
		(n = 50)	(n = 30)	(n = 20)	(n = 20)	<i>F</i>	<i>p</i>	α^2	β	<i>F</i>	<i>p</i>	α^2	β	<i>F</i>	<i>p</i>	α^2	β
1st double support	GM	10.4±2.2	11.7±3.8	12.2±3.3	12.0±2.9	-	NS	-	-	-	NS	-	-	-	NS	-	-
	SOL	9.4±1.8	10.6±3.3	13.2±4.5*	17.1±5.9*+€	20.5	p<.001	0.31	0.95	-	NS	-	-	10.5	p<.01	0.11	0.89
	TA	18.1±2.7	19.8±4.2	25.7±5.7*	26.3±6.2*€	10.5	p<.01	0.12	0.89	-	NS	-	-	-	NS	-	-
Single support	GM	10.9±2.3	11.8±3.8	12.9±3.8	12.8±5.2	-	NS	-	-	-	NS	-	-	-	NS	-	-
	SOL	10.2±3.0	16.6±3.9*	17.8±5.1*	18.2±3.5*	20.0	p<.001	0.33	1	18.4	p<.001	0.18	0.98	-	NS	-	-
	TA	11.2±2.9	15.6±5.1*	16.5±3.7*	19.1±3.1*+€	39.4	p<.001	0.32	1	20.1	p<.001	0.25	0.96	40.1	p<.001	0.29	0.88
2nd double support	GM	18.8±3.5	28.6±3.9*	20.1±2.9	30.1±5.4*+	-	NS	-	-	86.3	p<.001	0.51	1	14.9	p<.05	0.10	0.60
	SOL	24.1±5.4	29.4±5.9*	30.6±8.6*	31.1±6.7*	15.0	p<.01	0.15	0.96	9.9	p<.01	0.10	0.84	-	NS	-	-
	TA	7.6±2.2	8.8±4.9	9.3±3.9	10.1±3.5	-	NS	-	-	-	NS	-	-	-	NS	-	-
Swing phase	GM	6.1±4.5	7.1±3.2	7.5±3.2	7.7±3.6	-	NS	-	-	-	NS	-	-	-	NS	-	-
	SOL	7.1±2.8	8.5±3.5	12.5±3.4	12.9±6.4	-	NS	-	-	-	NS	-	-	-	-	-	-
	TA	13.5±3.1	14.5±5.2	14.5±5.2	15.5±3.3	-	NS	-	-	-	NS	-	-	-	NS	-	-

Comparison between 4 groups based on two-way ANOVA for each parameter with additional post hoc analysis (Bonferroni procedure) when there is a main effect of obesity, age and an interaction (age × obesity). CG: control group, OB: obese group, OA: older adults group, OBOA: obese older adults group, GM: gastrocnemius medialis, TA: tibialis anterior, SOL: soleus SD: standard deviation.

NS: no significant difference.

*= Significant difference between CG and other groups ($p < 0.05$).

+ = Significant difference between OA and OBOA ($p < 0.05$).

€ = Significant difference between OB and OBOA ($p < 0.05$).

Table 5. Summary of the forward stepwise multiple regression analysis examining the effect of body weight and age on ankle muscle activity during the different walking cycle phases

Walking cycle phases	Muscle	Independent variables	Final solution of the model					
			β	<i>S.E.</i> β	<i>r</i>	<i>R</i> ²	Adjusted <i>R</i> ²	<i>p</i> -level
1 st Double support	GM	Body weight	0.12	0.034	0.35	0.14	0.14	NS
		Age	0.14	0.052	0.34	0.15	0.14	NS
		Final solution of the model (Body weight + Age)			0.39	0.15	0.14	<0.05
	SOL	Body weight	0.20	0.042	0.40	0.16	0.15	<0.05
		Age	0.36	0.069	0.43	0.17	0.16	<0.05
		Final solution of the model (Body weight + Age)			0.42	0.18	0.17	<0.01
	TA	Body weight	0.16	0.064	0.86	0.76	0.75	<0.05
		Age	0.87	0.395	0.87	0.76	0.75	<0.001
		Final solution of the model (Body weight + Age)			0.88	0.77	0.77	<0.001
Single support	GM	Body weight	0.13	0.093	0.37	0.18	0.17	NS
		age	0.12	0.036	0.36	0.17	0.17	NS
		Final solution of the model (Body weight + Age)			0.39	0.21	0.18	<0.05
	SOL	Body weight	0.15	0.029	0.35	0.19	0.18	<0.05
		Age	0.64	0.11	0.64	0.41	0.40	<0.001
		Final solution of the model (Body weight + Age)			0.65	0.43	0.41	<0.001
	TA	Body weight	0.66	0.139	0.78	0.61	0.60	<0.001
		Age	0.46	0.220	0.74	0.62	0.61	<0.001
		Final solution of the model (Body weight + Age)			0.79	0.63	0.62	<0.001
2 nd double support	GM	Body weight	0.86	0.385	0.84	0.71	0.69	<0.001
		Age	0.17	0.061	0.35	0.18	0.17	<0.05
		Final solution of the model (Body weight + Age)			0.84	0.71	0.69	<0.001
	SOL	Body weight	0.62	0.315	0.78	0.61	0.60	<0.01
		Age	0.51	0.235	0.78	0.61	0.60	<0.01
		Final solution of the model (Body weight + Age)			0.79	0.62	0.61	<0.01
	TA	Body weight	0.03	0.08	0.10	0.03	0.03	NS
		Age	0.22	0.08	0.38	0.17	0.16	<0.05
		Final solution of the model (Body weight + Age)			0.39	0.18	0.16	<0.05

β : mathematical weightenings of the explanatory variables in the equation, *S.E.* β : estimated precision of the coefficient, *r* : the correlation coefficient, *R*²: the multiple correlation coefficient, adjusted *R*²: the adjusted multiple correlation coefficient, *p*: the *p*-level, GM: gastrocnemius medial, SOL: soleus, TA: tibialis anterior. Significant explanatory factor at the level: *p* < 0.05.

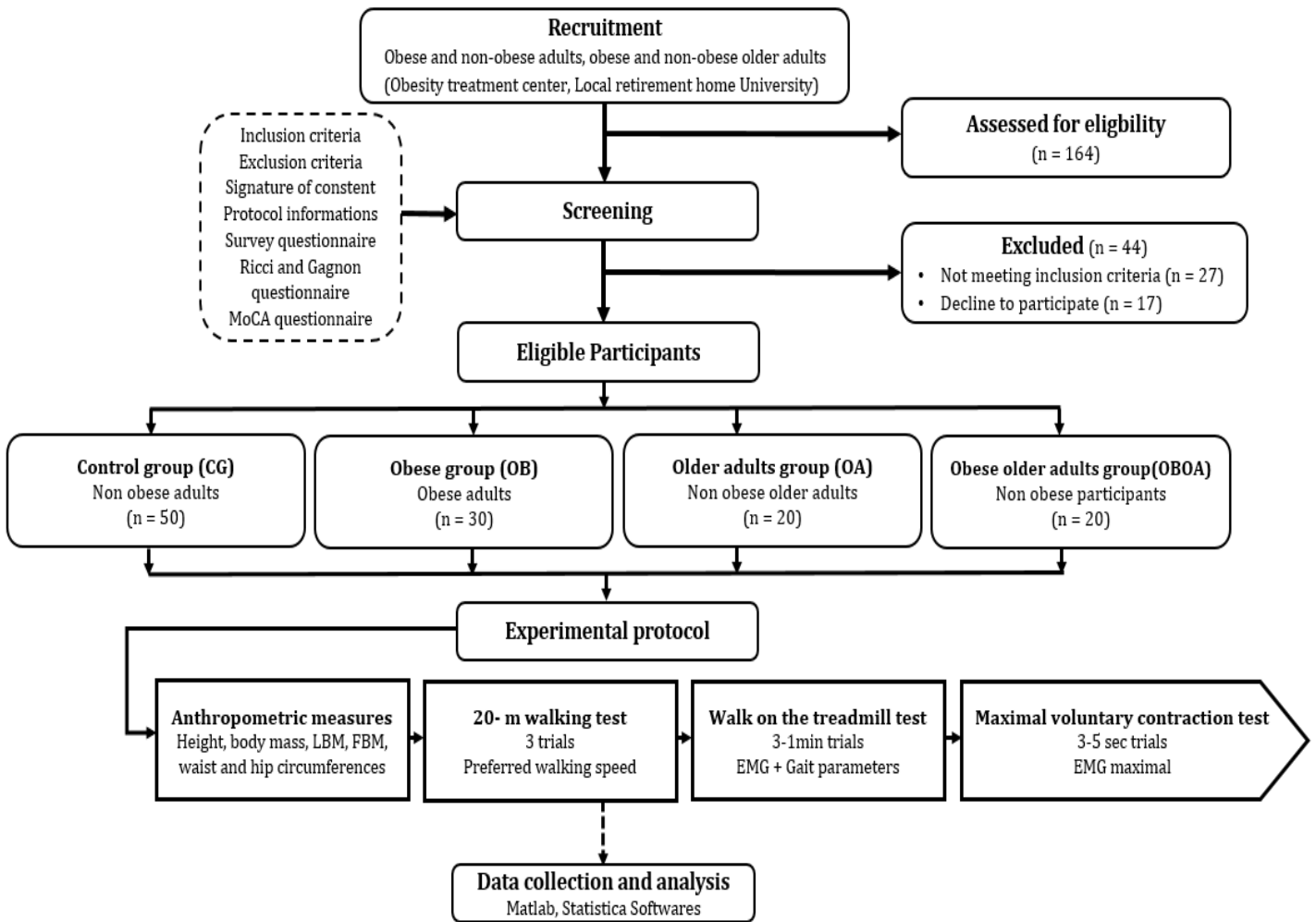


Figure. 1: Flow diagram illustrating the experimental procedure design.

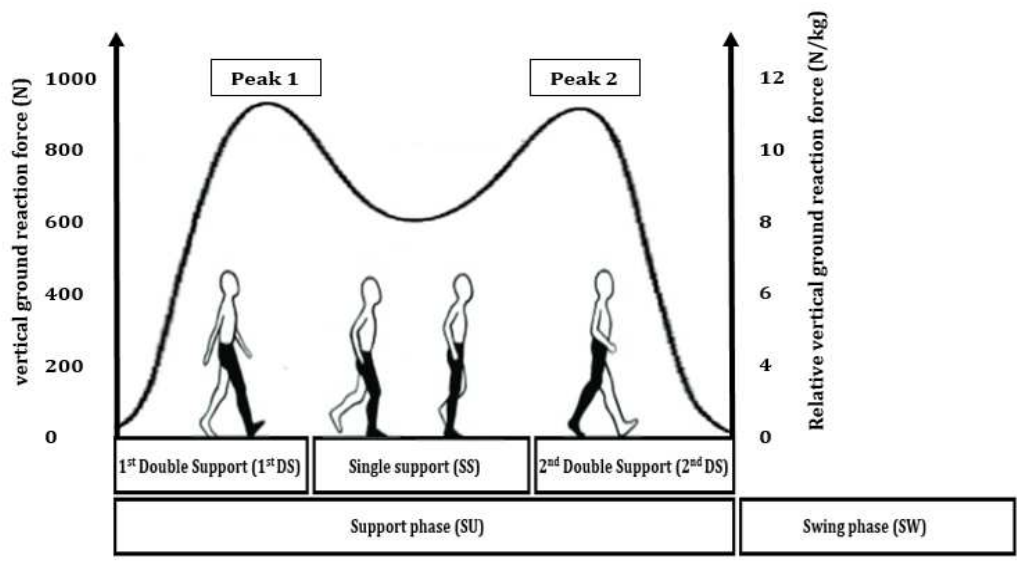


Figure 1. Vertical ground reaction force during walking cycle.