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Does Obesity Affect the Rate of Force Development in Plantar Flexor Muscles among Older Adults? A Cross-Sectional Study

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Does Obesity Affect the Rate of Force Development in Plantar Flexor Muscles Among Older Adults?

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Abstract: The literature offers limited information on the effect of obesity on the rate of force development, a critical parameter for mobility in older adults. The objectives of this study were; to explore the influence of obesity on RFD in older adults and to examine the association between this neuromuscular parameter and walking speed. 42 older adults were classified into two groups: the control group (CG, n=22; age=81.13±4.02 years; Body mass index (BMI)=25.13±3.35 kg/m²), and the obese group (OG, n=20; age=77.71±2.95 years; BMI=34.46±3.25 kg/m²). Walking speed (m/s) was measured using the 10-meter walking test. Neuromuscular parameters of the plantar flexors were evaluated during a maximal voluntary contraction test using a dynamometer. The RFD was calculated from the linear slop of the force – time curve in two phases: from the onset of the contraction to 50 ms ($RFD_{50\text{-}100}$) and from 100 to 200 ms ($RFD_{100\text{-}200}$). The gait speed was lower in the OG compared to the CG ($p<0.001$). $RFD_{50/100}$ and $RFD_{100/200}$ were lower in the OG ($p<0.001$). The $RFD_{50/100}$ emerged as the predominant influencer on gait speed, accounting for 51% of its variance ($R^2 = 0.89$, $p <0.05$). In conclusion, obesity negatively impacts the RFD in older adults and $RFD_{50\text{-}100}$ stands out as the primary factor among the studied parameters influencing gait speed.

Keywords: overweight; explosive force; triceps surae; walking

1. Introduction

The rate of force development (RFD) emerges as a pivotal determinant of mobility in older adults, potentially surpassing the influence of other muscular parameters [1–3]. The RFD parameter zeroes in on the force produced during the crucial initial 200 ms when a muscle is activated [4]. Intriguingly, this parameter exhibits heightened sensitivity to the aging process compared to maximal strength, underscoring its critical role in assessing age-related declines in muscle function [3,5]. Recent research underscores the RFD as a robust predictor of functional capabilities in older adults, notably its strong association with walking speed. Furthermore, a diminished ability in swift force generation within the 100-200 ms timeframe after a misstep might be a key factor in the diminished capacity of older adults to counteract falls [3]. Evaluations of RFD typically encompass various contraction intervals of muscles, specifically the early (0-50 ms) and the latter (100-200 ms) phases [6]. The preliminary phase correlates with the onset of motor unit activation and their respective firing sequences, as well as intrinsic muscle characteristics like fiber composition and

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calcium dynamics [7]. Conversely, the latter phase largely draws influence from elements such as peak strength and the overall muscular structure [8].

Prior research has documented age-related declines in maximal strength in lower limb muscles, including leg flexors [6], dorsiflexors [7], and plantar flexors (PF) [9]. Interestingly, it has been observed that the reduction in RFD during aging can be more pronounced (39–64%) than the decrease in maximal isometric strength (29–46%) [3,6]. When examining the impact of obesity on muscle strength in older adults, several factors contribute to understanding the potential reduction in muscle strength in older adults with obesity. The infiltration of fat within skeletal muscles reduces the contractile component of the overall muscle volume [10]. On other hand, chronic inflammation and a decrease in anabolic hormones such as insulin-like growth factor-1 (IGF-1), essential for muscle repair and growth [11,12], further exacerbate muscle tissue alterations, consequently diminishing the capacity of force production [13]. In this context, Maktouf et al. [9] showed that older adults with obesity possess lower relative maximal plantar flexor strength. However, it remains unknown whether obesity in older adults exacerbates the decline in RFD. This is particularly pertinent, as older adults with obesity are at a higher risk of falls [14].

Walking is an indicator of autonomy [15] and plays a key role in disease prevention and weight management in older adults [16]. It also serves as a crucial diagnostic criterion for conditions like sarcopenia and frailty [17]. Recent research indicates that obese older adults face a 1.5 to 5 times higher risk of developing walking limitations compared to their normal-weight peers [23]. In this context, Laroche et al. [19] showed that older adults spend less time in single support and more time in double support than older adult with normal weight. During the propulsion phase, the role of plantar flexors is pivotal for propelling the body forward and facilitating step transition [20], underscoring their notable association with walking speed [21]. In this context, Maktouf et al. [22] demonstrated that gait parameter alterations in older adults with obesity are attributed to increased activation of the PF, where body mass contributes to 87% of the variation in this heightened activation. However, there is a notable gap in research exploring the relationship between the RFD of PF and walking speed, especially among older adults with obesity. Addressing these research gaps is essential for developing targeted interventions and ensuring that therapeutic strategies are based on a thorough understanding of the underlying dynamics.

The objectives of this study were to: *i)* to investigate the influence of obesity on RFD in older adults with sarcopenia, and, *ii)* to examine the relationship between neuromuscular markers and gait speed in older adults with SO.

2. Materials and Methods

2.1. Study Design

The study was designed following an analytical cross-sectional approach, as depicted in **Figure 1**. The process involved a 4-week recruitment, a 3-week screening phase, and culminated in a 2-hour evaluation session that included health questionnaires, anthropometric measurements, a 10-meter walk test, and neuromuscular assessments.

2.2. Recruitment

Participants were recruited from various care centers through announcements, where medical staff gathered volunteer lists for researchers. Eligibility for older adults required being over 65 years old. Based on body mass index (BMI) criteria, those with a BMI under 25 kg/m² were placed in the control group (CG), and individuals with a BMI over 30 kg/m² were categorized into the older adults with obesity group (OG). Exclusion criteria included neurological or cognitive impairments, severe cardiovascular diseases, significant lower limb musculoskeletal problems, other major comorbidities or chronic diseases, use of medication affecting test outcomes, or a Montreal Cognitive Assessment (MoCA) score below 26.

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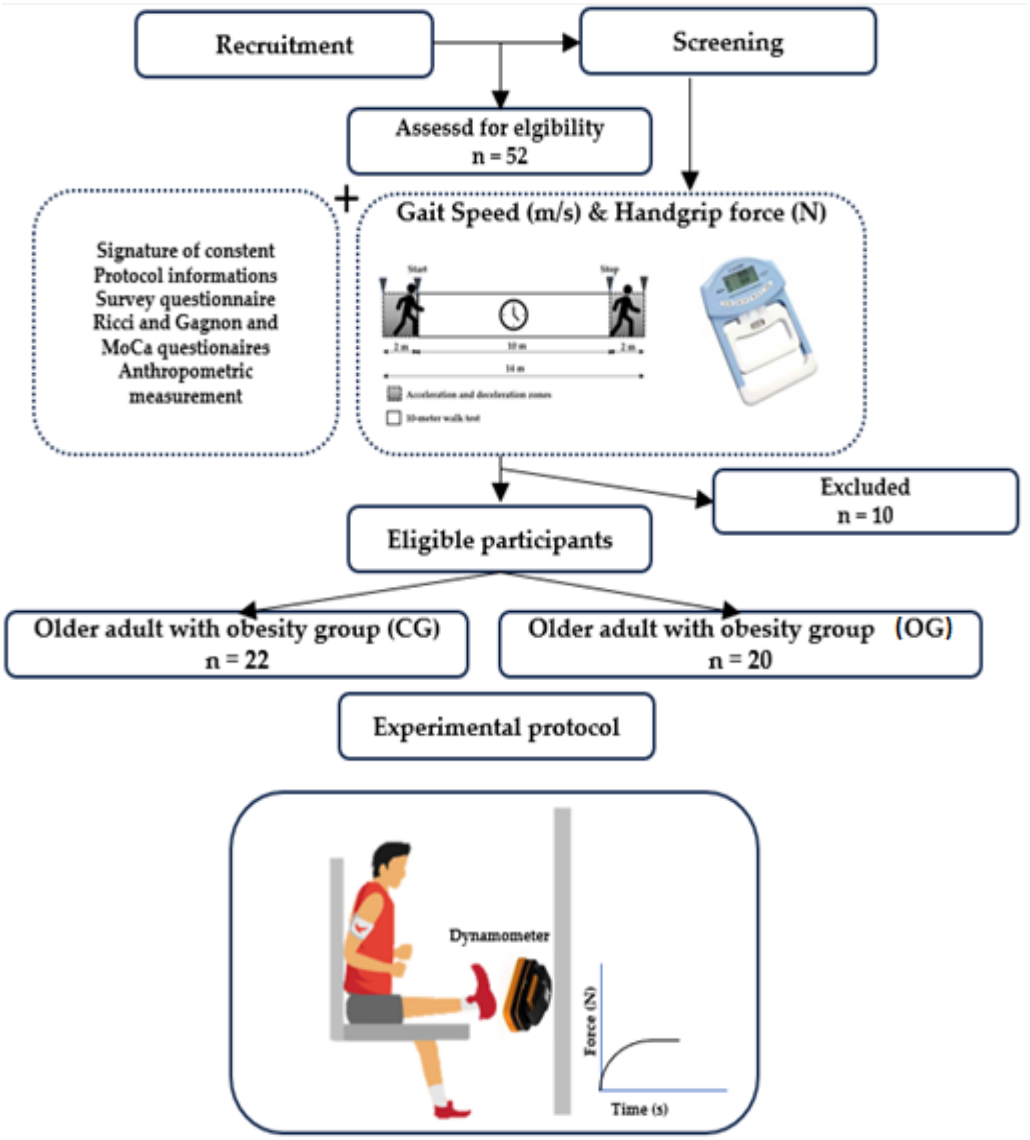

Figure 1. Study design.

2.3. Experimental Protocol

All evaluations were conducted in a designated clinical examination room under consistent environmental conditions, overseen by a singular, trained assessor. Partici-pants were given a standardized set of verbal instructions before the assessments to en-sure familiarity with the procedures.

2.3.1. Gait Speed Evaluation

Gait speed was assessed using a chronometer during the 10-meter walk test (10MWT), as outlined in **Figure 2**. Participants were instructed to walk along a 14-meter corridor, which included a 2-meter acceleration zone, a 10-meter measurement zone for the 10MWT, and a 2-meter deceleration zone [23]. To ensure accuracy, only the time taken to cover the distance between the 3th and 13th meters was recorded, effectively eliminating the influence of the acceleration and deceleration phases. The timing commenced as soon as the participant's toes crossed the 2nd meter mark and stopped when they crossed the 12th meter mark, in accordance with the American Physical Therapy Association Clinical Practice Guidelines [24]. Each participant performed three trials of the 10MWT, with a minimum rest period of 30 seconds between each trial.

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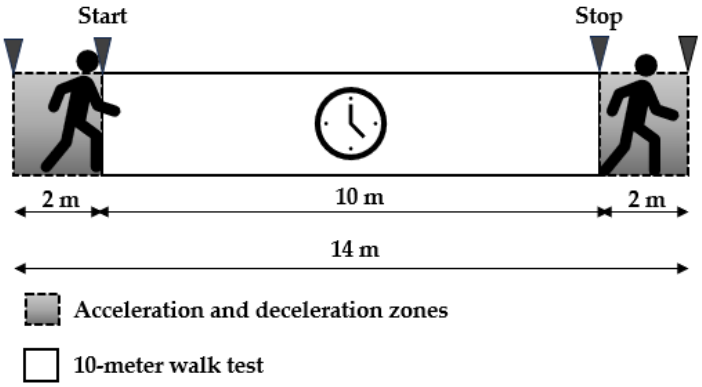

Figure 2. Configuration of the 10-m walking test in a 14-m corridor with 2-m zones for Acceleration and Deceleration.

For data processing, the average time from the three trials was used to calculate the 10MWT speed (m/s) as follows:

Gait speed (m/s) = time (s) / distance (m), where distance = 10 m

2.3.2. Neuromuscular Parameters Evaluation

Neuromuscular parameters of the PF of the dominant leg were measured using a dynamometer (K-Force, Kinvent, Montpellier, France) with sampling rate of 1000 Hz and accuracy of 100g. Participants were instructed to maintain contact between their back, buttocks, and thigh with the chair while keeping their leg stretched horizontally [9].

The protocol commenced with a dynamic plantar flexion of the ankle as a warm-up. Then, participants performed two explosive maximal isometric voluntary contractions (MVC), each lasting approximately 1 second, interspersed with 20-second rest periods. The absolute MVC from these two trials was recorded (Fmax, N). To calculate the relative force (R-Fmax, N/kg), the A-Fmax was normalized to the participant's body mass (A-Fmax/Body mass, N/kg).

For data processing, the numeric force signal from the dynamometer was filtered using a second-order zero-lag Butterworth low-pass filter with a 40-Hz cutoff frequency, employing Matlab (The MathWorks Inc., Natick, MA, USA) [25]. The onset of each contraction was identified using a second derivative method. The force signal analysis commenced from the onset, within a 200 ms time window (**Figure 3**). Absolute force values were extracted at 50 ms (F50), 100 ms (F100), and 200 ms (F200) intervals. These values were normalized to body mass (R-F50, R-F100, R-F200) and to Fmax (50-Fmax, 100-Fmax, 200-Fmax, %). The RFD–time curve was then calculated through the first derivative of the force signal (Δ force/Δ time) at each overlapping interval within the 200-ms continuum (**Figure 3**) [25]. This curve was also low-pass filtered at a 50-Hz cutoff frequency. The early RFD, from onset to 50 ms (RFD50-100), and the late RFD, from 100 to 200 ms (RFD100-200), were extracted.

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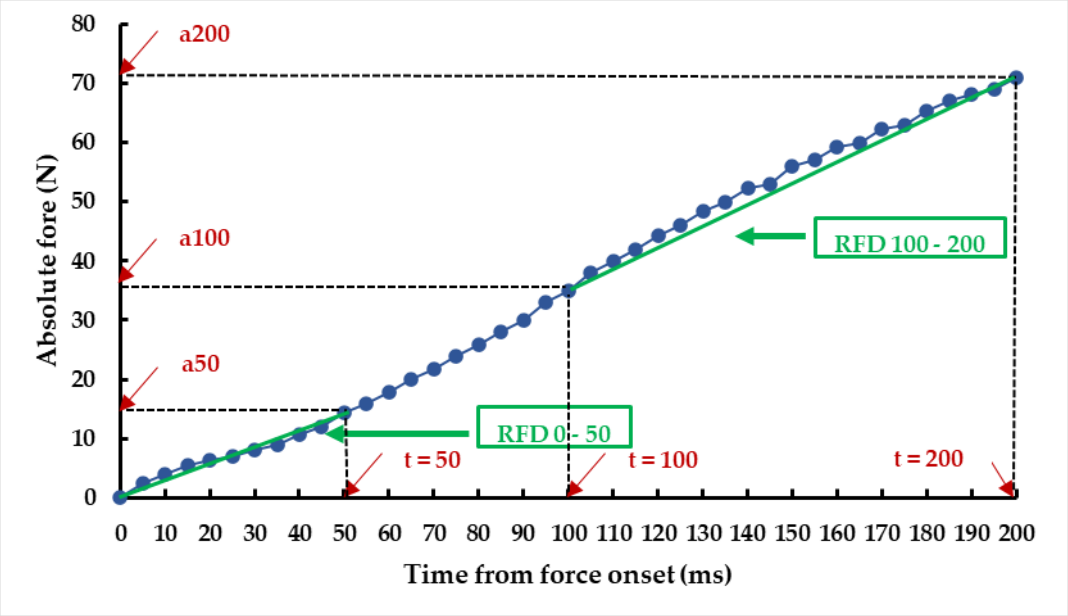

Figure 3. Force-time curve from force onset to 200 ms during maximal voluntary contraction of plantar flexors.

2.4. Statistical Analysis

The sample size was calculated using the freeware G*Power (version 3.1.9.4) [26]. The t-test was predefined for power analysis. The estimation was based on predefined control of type I error (alpha = 0.05) and Type II error (beta = 0.60), with a moderate level of estimated effect size (r = 0.30) [16]. Under these settings, 38 participants were required as the minimum sample size.

For statistical analysis, Jamovi software (version 2.3, Sydney, Australia) was utilized [27]. The Shapiro-Wilk test and the Levene test were employed to ascertain data normality and variance homogeneity, respectively. Upon confirming that the data adhered to these assumptions, an independent samples t-test was conducted to identify variances between the groups. Moreover, a Pearson correlation analysis was performed to pinpoint parameters strongly associated with gait speed in the OG. Correlation coefficients were categorized as follows: negligible for [0.00, 0.01), weak for [0.01, 0.16), moderate for [0.16, 0.49), strong for [0.49, 0.81), and very strong for [0.81, 1.00] [28]. Finally, multiple logistic regression models were employed, using parameters identified as having a strong correlation, to discern the model that exhibits the strongest correlation with gait speed in the OG.

3. Results

3.1. Participants

An initial group of 52 older adults was recruited. Following a thorough application of inclusion and exclusion criteria, 45 participants met the study's qualifications. Regrettably, three individuals could not meet the study requirements, resulting in a final cohort of 42 dedicated participants who completed the study in its entirety.

Statistical analysis showed no significant differences in age, body height and LBM between SOG and CG. Nonetheless, BM ($p < 0.001$), BMI ($p < 0.001$), body fat ($p < 0.001$) and FBM ($p < 0.001$) were notably higher in the OG compared to the CG **(Table 1)**.

Table 1. Anthropometric characteristics of groups.

Parameters	Groups	Mean ± SD
Age (y)	OG	77.7 ± 2.9

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	CG	81.1 ± 4.0
Height (cm)	OG	162.9 ± 6.3
	CG	166.0 ± 7.5
Body mass (kg)	OG	91.0 ± 3.9***
	CG	68.74 ± 5.5
Body mass index (kg/h²)	OG	34.5 ± 3.2***
	CG	25.1 ± 3.4
Body fat (%)	OG	35.0 ± 6.34***
	CG	17.7 ± 1.98
Fat body mass (kg)	OG	32.0 ± 6.9***
	CG	12.2 ± 1.5
Lean body mass (kg)	OG	58.9 ± 4.1***
	CG	56.6 ± 5.0

OG: older adluts with obesity, CG: control group, * p < .05, ** p < 0.01, *** p < .001.

3.2. Gait Speed

The Gait speed was lower in the OG compared to the CG (p <0.001) (**Figure 3**).

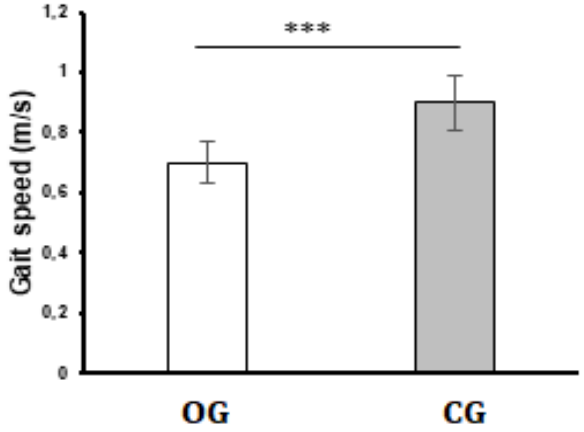

Figure 3. Comparison of the gait speed between groups.

3.3. Neuromuscular Parameters

Neuromuscular parameters during MVC test are detailed in **Table 2.** There was no significant difference between OG and CG in terms of Fmax. However, F50 (p <0.001), F100 (p = 0.015), and F200 (p <0.001) were lower in the OG. When normalized to BM, R-F50 (p <0.001; d = -1.80), R-F100 (p <0.001; d = -2.89), and R-F200 (p <0.001; d = -3.13) were also higher in the CG. In addition, $RFD_{50/100}$ and $RFD_{100/200}$ were lower in the OG compared to the CG (p <0.001 and p <0.001, respectively).

Table 2. Neuromuscular parameters of plantar flexors.

Parameters	Groups	Mean ± SD
Fmax (N)	OG	192.69 ± 15.41
	CG	194.89 ± 13.28
R-Fmax (N/kg)	OG	2.12 ± 0.22***
	CG	2.86 ± 0.36
F50 (N)	OG	31.18 ± 2.12***
	CG	33.80 ± 2.65
R-F50 (N/kg)	OG	0.37 ± 0.03***
	CG	0.45 ± 0.05
F100 (N)	OG	69.53 ± 5.45*

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	CG	73.51 ± 4.73
R-F100 (N/kg)	OG	0.76 ± 0.08***
	CG	1.07 ± 0.12
F200 (N)	OG	113.96 ± 8.93***
	CG	124.73 ± 8.48
R-F200 (N/kg)	OG	1.25 ± 0.13
	CG	1.82 ± 0.21
50-Fmax (%)	OG	17.60 ± 1.56
	CG	16.02 ± 0.96
100-Fmax (%)	OG	36.14 ± 2.01
	CG	37.81 ± 2.57
200-Fmax (%)	OG	59.40 ± 5.64*
	CG	64.16 ± 4.72
RFD $_{50/100}$ (N/ms)	OG	676.06 ± 53.00***
	CG	708.19 ± 38.21
RFD $_{100/200}$ (N/ms)	OG	444.26 ± 81.50***
	CG	512.11 ± 78.13

OG: older adults with obesity, CG: control group, RFD: rate of force development. F50, F100 and F200: absolute force at 50, 100 and 200ms. R-F50, R-F100, R-F200: relative force at 50, 100 and 200 ms. Fmax: absolute maximal force. R-Fmax: relative maximal force. SD: standard deviation. * p < .05, ** p < 0.1, *** p < .001.

3.4. Relationships between Neuromusclar Parameters and Gait Speed

Gait speed demonstrated significant correlations with neuromuscular parameters (**Table 3**). The strongest correlation was observed with RFD$_{50/100}$ (r = 0.84, p <0.001). The correlation with RFD$_{100/200}$ was also notably high (r = 0.83, p <0.001). Further correlations included R-F50 (r = 0.77, p <0.001), R-F100 (r = 0.66, p = 0.001), R-F200 (r = 0.75, p <0.001) were observed.

Table 3. Correlation analysis between neuromuscular parameters and gait speed.

Neuromuscular parameters	**Gait speed (m/s)**	
	r	p
RFD50/100 (N/ms)	0.84	<0.001
RFD100/200 (N/ms)	0.83	<0.001
R-F50 (N/kg)	0.77	<0.001
R-F100 (N/kg)	0.66	<0.001
R-F200 (N/kg)	0.75	<0.001
R-Fmax (N/kg)	0.60	<0.05
50-Fmax (%)	0.32	0.164
100-Fmax (%)	0.26	0.236
200-Fmax (%)	0.35	0.129

RFD: rate of force development. F50, F100 and F200: absolute force at 50, 100 and 200ms. R-F50, R-F100, R-F200: relative force at 50, 100 and 200 ms. Fmax: absolute maximal force. R-Fmax: relative maximal force. Green: strong correlations. Blue: very strong correlations.

The logistic regression analysis (**Table 4**) assessed the relationship between gait speed and neuromuscular parameters of PF using parameters that identified as having a strong correlation as shown in **Table 3**.

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Table 4. Logistic regression analysis assessed the impact of various neuromuscular parameters on gait speed in older adults with obesity.

			Unstard. Coef.	Standard. Coef.			Model summary			
	Model		B	Beta	SD	t	p	r	R^2	Adj R^2	SD
Gait speed (m/s)	1	(Constant)	-0.46		0.18	-2.58	0.019	0,84	0,7	0,69	0,06
		$RFD_{50/100}$ (N/ms)	0	0.84	0	6.55	<0.001				
	2	(Constant)	-0,28		0,15	-1,89	,076	0,91	0,82	0,8	0,05
		$RFD_{50/100}$ (N/ms)	0	0,51	0	3,61	,002				
		$RFD_{100/200}$ (N/ms)	0	0,48	0	3,39	,004				
	3	(Constant)	-0,51		0,14	-3,55	,003	0,94	0,89	0,87	0,04
		$RFD_{50/100}$ (N/ms)	0	0,51	0	4,45	<.001				
		$RFD_{100/200}$ (N/ms)	0	0,35	0	2,8	,013				
		R-Fmax (N)	0,14	0,29	0,05	3,06	,008				
	4	(Constant)	-0,47		0,16	-3,01	,009	0,95	0,89	0,86	0,04
		$RFD_{50/100}$ (N/ms)	0	0,46	0	3,19	,006				
		$RFD_{100/200}$ (N/ms)	0	0,38	0	2,58	,022				
		R-F50 (N)	0,3	0,1	0,42	0,71	,487				
		R-F100 (N)	-0,15	-0,1	0,25	-0,57	,575				
	5	(Constant)	-0,5		0,14	-3,52	,003	0,95	0,89	0,87	0,04
		$RFD_{50/100}$ (N/ms)	0	0,43	0	3,04	,008				
		$RFD_{100/200}$ (N/ms)	0	0,34	0	2,77	,014				
		R-F200 (N)	0,12	0,14	0,11	1,07	,302				
		R-Fmax (N)	0,13	0,26	0,05	2,64	,019				

RFD: rate of force development. F50, F100 and F200: absolute force at 50, 100 and 200ms. R-F50, R-F100, R-F200: relative force at 50, 100 and 200 ms. Fmax: absolute maximal force. R-Fmax: relative maximal force.

In the first model, with $RFD_{50/100}$ as the sole parameter, the equation was: Gait speed = - 0.46 + 0.84 $RFD_{50/100}$ with R^2 = 0.7, indicating that approximately 70% of the variance in gait speed could be explained by $RFD_{50/100}$ alone ($p < 0.001$).

The second model combined $RFD_{50/100}$ and $RFD_{100/200}$. In this model, the equation was: Gait speed = - 0.28 + 0.51 $RFD_{50/100}$ + 0.48 $RFD_{100/200}$, with R^2 = 0.82, showing an improved explanatory power over the first model and both predictors were statistically significant ($p < 0.05$). $RFD_{50/100}$ was the predominant influencer on gait speed, accounting for 51% of its variance.

The third model included $RFD_{50/100}$, $RFD_{100/200}$ and F-Fmax. the equation was: Gait Speed = - 0.51 + 0.51 $RFD_{50/100}$ + 0.35 $RFD_{100/200}$ + 0.29 R-Fmax, with an R^2 = 0.89. All variables had a significant influence on gait speed ($p < 0.001$) indicating that $RFD_{50/100}$ was the predominant influencer on gait speed, accounting for 51% of its variance.

The fourth model included R-F50, R-F100, $RFD_{50/100}$ and $RFD_{100/200}$. The equation was: Gait Speed = - 0.47 + 0.46 $RFD_{50/100}$ + 0.38 $RFD_{100/200}$ + 0.1 R-F100 - 0.1 R-F100, with an R^2 = 0.89. In this model, only $RFD_{50/100}$ and $RFD_{100/200}$ remained significant ($p < 0.05$). $RFD_{50/100}$ emerged as the predominant influencer on gait speed, accounting for 46% of its variance.

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The fifth model included R-Fmax, R-F200, $RFD_{50/100}$ and $RFD_{100/200}$. The equation was: Gait Speed = - 0.5 + 0.43 $RFD_{50/100}$ + 0.34 $RFD_{100/200}$ + 0.13 R-Fmax + 0.12 r200, with an R^2 = 0.89. In this configuration, only $RFD_{50/100}$, $RFD_{100/200}$, and R-Fmax were significant predictors ($p<0.05$) where RFD50/100 emerged as the predominant influencer on gait speed, accounting for 43% of its variance.

4. Discussion

The primary aim of this study was to discern the impact of obesity on neuromuscular parameters of plantar flexors in older adults. The findings underscored the significant influence of obesity on neuromuscular parameters, particularly the RFD. Our secondary objective delved into the relationship between these neuromuscular parameters and gait speed in older adults with obesity. Notably, RFD of plantar flexors stood out as the predominant factor affecting gait speed, contributing to a notable 51% of its variability, which notably surpassed the impact of the relative maximal force of plantar flexors.

Our study found that obesity had no significant impact on the absolute maximal force generated by the plantar flexors, a finding consistent with the results of Maktouf et al. who also observed no differences between older adults with normal weight and their obese counterparts [29]. However, when forces were normalized to body mass, both maximal and submaximal forces of plantar flexors were notably lower in the OG. This is in alignment with multiple studies examining the effects of obesity on muscle strength in young adults [30–32]. This phenomenon may be partially explained by the observations of Tomlinson et al. [12], who noted that the most significant impact of combined aging and adiposity was evident in the rate of muscle volume loss. One plausible mechanism underlying this accelerated muscle loss is that obesity exacerbates the challenges posed by sarcopenia [13,33]. It does so by exerting additional mechanical stress on the musculoskeletal system, particularly due to the need to support elevated adipose tissue weight [34]. Beyond serving as mere energy storage, adipose tissue is a dynamic endocrine organ that secretes an array of hormones and pro-inflammatory cytokines, thereby amplifying biochemical stress in the body [35]. Chronic adiposity results in elevated levels of circulating pro-inflammatory cytokines such as TNF-α, IL-1α, IL-6, and CRP, which contribute to both acute and chronic systemic inflammation [36]. These inflammatory agents negatively impact skeletal muscle by promoting protein degradation over synthesis, ultimately leading to muscle wasting or atrophy [11]. Further complicating the scenario is the association between obesity and a decline in anabolic hormones, specifically insulin-like growth factor-1 (IGF-1), which plays a crucial role in muscle repair and growth [37].

The innovative aspect of this study centers on the assessment of the RFD, identified as a crucial parameter for evaluating mobility in vulnerable populations. Our findings reveal a marked influence of obesity on RFD parameters. Specifically, the values for $RFD_{50/100}$ and $RFD_{100/200}$ decreased by 5% and 13%, respectively, in the OG when compared to the CG. In a related vein, Olmos et al. [1] highlighted that late rapid force parameters are disproportionately compromised in older adults without obesity. Based on these observations, we suggest that the detrimental effects on early rapid force are likely obesity-specific in older adults, while the impact on late rapid force seems to exacerbate existing sarcopenia-related impairments. The impact of obesity on both early and late rapid force in older adults can be elucidated by examining multiple factors, such as motor unit recruitment [38], intrinsic muscle properties [12,39], and systemic inflammation [11,13]. Early rapid force is predominantly associated with initial motor unit recruitment and firing rates, as well as intrinsic muscle attributes such as fiber type composition and calcium kinetics [7]. Obesity might specifically affect the early RFD due to the increased mechanical stress it places on the musculoskeletal system, thereby affecting the efficiency of motor unit recruitment and the contractile properties of muscle fibers. Moreover, adipose tissue in older adults with obesity functions as a dynamic endocrine organ, secreting an array of hormones and pro-inflammatory cytokines, like TNF-a, IL-1a, IL-6, and CRP [11]. This heightened state of systemic inflammation could adversely affect early RFD by causing protein degradation to outpace synthesis, leading to muscle atrophy and reduced contractile capabilities. On the other hand, late rapid force measures are more strongly influenced by factors such as maximal strength, muscle size, tendon stiffness, and

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pennation angle [8]. Obesity can exacerbate the loss of muscle mass—often referred to as sarcopenia—which is already compromised in older adults. The compounded effect of obesity and aging leads to a decline in maximal strength and muscle size, which in turn, significantly impacts the late RFD. The accelerated muscle loss may be further exacerbated by chronic inflammation and a decline in anabolic hormones like insulin-like growth factor-1 (IGF-1), which are crucial for muscle repair and growth [11,12].

Our study brings forth compelling evidence that the RFD is an invaluable predictor of gait speed among older adults with obesity. When integrating variables such as $RFD_{50/100}$, $RFD_{100/200}$, and R-Fmax, we obtained a high adjusted R^2 value of 0.89. Strikingly, the early RFD, represented by $RFD_{50/100}$, was the dominant determinant, accounting for 51% of the variation in gait speed within the OG. Additionally, $RFD_{100/200}$ and R-Fmax contributed to the explanatory power by 35% and 29%, respectively. These results are in harmony with earlier studies on frail populations, which have demonstrated the RFD to be an independent predictor of an array of physical functions [39,40]. These include abilities as varied as rising from a chair, completing timed "up and go" tests, and achieving both casual and maximal walking speeds in older adults with regular weight [2]. Moreover, our findings echo other research that emphasizes the significant role of muscle power, rather than mere muscle strength, in affecting walking velocity and, by extension, susceptibility to falls [1,2].

A critical question that warrants discussion is why RFD is a more salient predictor of gait speed than maximal force. One plausible explanation is that quick force generation could constrain an individual's capacity to engage in rapid movements, especially in activities requiring sequential agonist and antagonist muscle contractions, such as walking [3]. Many daily activities necessitate the rapid application of force over a short duration (i.e., RFD capacity). For instance, a noticeable surge in force within approximately 200 milliseconds is essential when an older adult stands up from a seated position [1]. Furthermore, the ability to avert a fall is not solely reliant on the production of maximal force but is also contingent on the speed of motor response [41]. This underscores the functional significance of the rate at which submaximal force can be generated (i.e., RFD). Hence, RFD serves a dual role: it is not only a performance determinant in functional tasks that demand more power than force but also a pivotal metric for assessing fall risk [3,5].

4.1. Limitations and Perspectives

Our study has several limitations that warrant attention. First and foremost, the small cohort size restricts the generalizability of our findings, particularly given the heterogeneity often observed in older adults with obesity. These populations can sometimes present a complex interplay of factors, resulting in characteristics that may not be wholly reflective of those in our study sample. Another key limitation lies in the non-utilization of electromyography, which could have provided further insights into the neurological mechanisms involved in force development and muscle function. The exclusion of this analytical tool leaves certain questions unanswered and calls for additional research to elucidate these mechanisms more comprehensively. These limitations pave the way for future studies that could contribute to a more holistic view of how obesity influences mobility and fall risks among older adults.

4.2. Practical Recommendations

Considering our study's findings, which emphasize the critical role of the RFD in physical function, clinicians are urged to integrate RFD measurements into their standard neuromuscular assessments for a more nuanced understanding, particularly in vulnerable populations like older adults with obesity. Based on this comprehensive evaluation, tailored intervention programs should be designed to improve RFD and thereby enhance essential functional capacities, such as walking speed. Instead of solely focusing on maximal force, exercise regimens should prioritize muscle power, incorporating high-intensity, explosive movements that mimic real-world scenarios requiring rapid force generation. This approach is particularly pertinent for fall prevention programs, as RFD is a significant predictor of fall risks.

5. Conclusions

Our findings reveal that obesity has a pronounced negative impact on the RFD. Specifically, the adverse effects on early RFD appear to be obesity-specific in older adults with obesity, while also exacerbating the existing impairments related to sarcopenia in late RFD. Significantly, early RFD emerges as the dominant factor influencing gait speed, accounting for an impressive 51% of its variability, far surpassing the impact of relative maximal force of plantar flexors. This underscores the critical diagnostic and prognostic role that RFD could play in the management of this vulnerable population. furthermore, rather than concentrating exclusively on improving maximal force, physical exercise regimens should prioritize boosting muscle power to better address the nuanced challenges faced by older adults with obesity.

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