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Article

Sarcopenia and Functional Decline in Postmenopausal Women: The Roles of Type 2 Diabetes and Physical Activity

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Abstract

Background/Objectives: Postmenopausal women face an elevated risk of sarcopenia and functional decline, yet the distinct roles of type 2 diabetes mellitus (T2DM) and physical inactivity in these outcomes remain unclear. This study aimed to determine the independent and combined effects of T2DM and physical activity on sarcopenia and functional performance in postmenopausal women. **Methods:** Cross-sectional study of 175 postmenopausal women stratified by T2DM status and physical activity level (active ≥ 150 min/week vs. insufficiently active). Body composition was assessed via dual-energy X-ray absorptiometry, muscle strength by handgrip dynamometry, and functional performance by gait speed. Sarcopenia was diagnosed using the Asian Working Group for Sarcopenia 2019 criteria. Binary logistic regression calculated odds ratios (OR) for adverse outcomes. **Results:** Physical inactivity was the strongest predictor of functional decline, with insufficiently active women showing nearly four-fold increased odds of slow gait speed (<1.0 m/s) compared to active counterparts (OR: 3.93; 95% CI: 1.24–12.45). While T2DM appeared protective against sarcopenia in unadjusted analysis, multivariate adjustment revealed obesity (OR: 4.97; 95% CI: 1.62–15.20) and T2DM (OR: 3.80; 95% CI: 1.59–9.08) as independent sarcopenia predictors. **Conclusions:** Distinct etiological pathways underlie sarcopenia and functional decline in postmenopausal women. While T2DM and obesity drive sarcopenia through metabolic mechanisms, physical inactivity primarily determines functional impairment. These findings support targeted interventions: metabolic optimization for muscle mass preservation and structured physical activity, particularly resistance training, for maintaining functional independence in this high-risk population.

Keywords: sarcopenia; diabetes mellitus; Type 2; exercise; postmenopause; walking speed

1. Introduction

The menopausal transition is characterized by a substantial decline in estrogen, which promotes an unfavorable redistribution of body composition, including an increase in visceral fat and a

decrease in muscle mass [1]. These hormonal shifts contribute to a pro-inflammatory state and insulin resistance, significantly heightening the susceptibility of postmenopausal women to cardiometabolic diseases such as type 2 diabetes mellitus (T2DM) [2,3].

A critical consequence of the interplay between aging, hormonal changes, and metabolic disease is the acceleration of sarcopenia, a key geriatric syndrome characterized by progressive loss of muscle mass and function, which poses a considerable risk to independence in older adults [4–6]. Sarcopenia is particularly prevalent and severe in individuals with T2DM, where persistent hyperglycemia and associated metabolic alterations in muscle tissue exacerbate muscle deterioration. This decline in muscle health impairs the ability to perform activities of daily living (ADLs) and increases the risk of disability, falls, and functional dependence [4–7].

Adopting an active lifestyle, particularly through physical training, has been shown to significantly improve glycemic control and mitigate the physiological changes and symptoms associated with menopause. These practices contribute to maintaining or even increasing appendicular muscle mass, thereby enhancing muscle strength and overall physical fitness. In turn, this helps prevent the detrimental effects of sarcopenia and its functional impairments [8,9]. While recent research highlights the contribution of behavioral factors, such as emotional eating, to T2DM risk in younger adult populations [10], in older, postmenopausal women, the role of physical inactivity becomes paramount, directly influencing not only metabolic health but also the fundamental capacity for independent living.

However, although the effects of type 2 diabetes mellitus (T2DM) and physical inactivity on sarcopenia and functional outcomes, such as gait speed, are well-documented, their combined impact on these outcomes in postmenopausal women is less clear. This underscores the need for more comprehensive research to better understand the complex interactions between menopause and T2DM. Considering these findings and the intricate interplay of biological and behavioral factors, the present study aims to investigate the influence of physical activity on body composition and functional performance in postmenopausal women with T2DM.

2. Materials and Methods

2.1. Study Design and Ethical Approval

This cross-sectional study was designed and conducted in accordance with the translated version of the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [11]. This research received ethical approval from the Research Ethics Committee at the University of Pernambuco – UPE (Report Number. 2.332.880; CAAE: 72113417.2.0000.5192). All procedures were conducted in accordance with the ethical standards outlined in the Declaration of Helsinki (1964, revised in 2013) and complied with the Brazilian National Health Council Resolutions 466/2012 and 510/2016. Written informed consent was obtained from all individuals before their inclusion in the study.

2.2. Participants and Recruitment

A total of 175 postmenopausal women were recruited for this study. Participants were sourced through public advertisements on social media and posters, direct phone invitations facilitated by the UPE, and from an existing cohort in the “Doce Vida” Supervised Physical Exercise Program for Diabetics, an extension program of the university.

A sample size calculation was performed a priori using OpenEpi software (release 3.03, 2014)[12], determining that a minimum of 165 participants were required to achieve 80% statistical power with a 95% confidence level and a 5% alpha error.

Participants were required to meet the following criteria: (a) women aged 40 years and older, (b) residing in Recife or its metropolitan area, (c) who appeared to be healthy, (d) had no limitations for motor tests, (e) self-reported postmenopausal status, (f) not undergoing cancer treatment, and (g) did not use medications known to alter body composition or prosthetics. Exclusion criteria were women

who did not undergo Dual-Energy X-ray Absorptiometry (DXA) scans and/or did not complete the motor tests. Participants were considered ineligible for participation if they did not complete the Dual-Energy X-ray Absorptiometry (DXA) scan or any of the required motor function tests, and if they tested positive for Human Immunodeficiency Virus (HIV).

2.3. Group Stratification

Participants were stratified into four distinct groups based on their T2DM diagnosis and physical activity level:

1. Group 1 (G1): Physically active women with T2DM. These participants were enrolled in the “Doce Vida” program, engaging in supervised combined (resistance and aerobic) training sessions three times per week, with each session lasting approximately 90 minutes.
2. Group 2 (G2): Insufficiently active women with T2DM who did not engage in regular, structured physical activity.
3. Group 3 (G3): Physically active normoglycemic women. Their activity consisted of unsupervised walking (average 20 minutes/session) combined with a supervised water-based exercise program (approximately 50 minutes/session), performed three times per week.
4. Group 4 (G4): Insufficiently active normoglycemic women who did not participate in regular physical activity.

2.4. Procedures and Data Collection

Data collection was conducted in two phases at the Human Performance Assessment Laboratory (LapH) and Biomechanics Laboratory (LABi) from the UPE. In the first phase, participants were screened for eligibility and provided with instructions. In the second phase, a structured questionnaire was administered to collect sociodemographic data, medical history, T2DM diagnosis and duration, and physical activity details. Subsequently, a series of anthropometric and functional assessments were performed.

2.5. Outcome Measures and Definitions

The primary exposure variables were T2DM status, confirmed according to the Brazilian Diabetes Society guidelines [13], and physical activity level, dichotomized as physically active (≥ 150 minutes/week of moderate-intensity exercise) or insufficiently active (< 150 minutes/week). The primary outcomes were sarcopenia and slow gait speed [14].

2.5.1. Anthropometric and Body Composition

Participants' total body mass was measured in kilograms (kg), and height was measured in centimeters (cm) using a properly calibrated anthropometric scale (PL-200, Filizola S.A., São Paulo, SP, Brazil), adhering to the standards set by NBR ISO/IEC 17025:2005. Body mass index (BMI) was calculated by dividing body mass (kg) by the square of height (m^2).

Body composition was assessed using a DXA system Hologic Discovery Wi Bone Densitometry (Hologic, Inc., Marlborough, MA, United States of America [USA]). The following indices were calculated:

- (a) Appendicular Skeletal Muscle Index (ASMI): Calculated by dividing appendicular lean mass (kg) by the square height (m^2). Low muscle mass was defined as an ASMI < 5.5 kg/ m^2 .
- (b) Fat Mass Index (FMI): Calculated by dividing total fat mass (kg) by the square of height (m^2). Obesity was defined as an FMI ≥ 13.0 kg/ m^2 .
- (c) Fat Mass Percentage (FM%).

2.5.2. Functional Performance

Gait speed was assessed during a single trial of usual walking speed over a 5-meter walkway with designated acceleration and deceleration zones. Slow gait speed was defined as < 1.0 m/s [14].

Grip strength was measured using a Jamar hydraulic hand dynamometer model 5030 J1 (Sammons Preston Rolyan, Bolingbrook, IL, USA). Participants were tested while standing, and the maximum value from one measurement on each hand was recorded. Low muscle strength was defined as < 18 kgf [14].

2.5.3. Sarcopenia Diagnosis

Sarcopenia was diagnosed according to the Asian Working Group for Sarcopenia (AWGS) 2019 consensus criteria [14], defined as the presence of low muscle mass (ASMI < 5.5 kg/m²) combined with either low muscle strength (< 18 kgf) or slow gait speed (< 1.0 m/s).

2.6. Outcome Measures and Definitions

Data were double-entered and analyzed using the SPSS Statistics for Windows (IBM Corp., Armonk, NY, USA, release 22.0, 2013). The normality of continuous variables was assessed using the Kolmogorov-Smirnov test, while Levene’s test was employed to examine the homogeneity of variances. Continuous variables were summarized with means and standard deviations (SD), while categorical variables were presented as absolute (n) and relative (%) frequencies. Baseline characteristics between the four investigated groups were compared using one-way Analysis of Variance (ANOVA). Pearson’s chi-square test (χ^2) or Fisher’s exact test was employed as appropriate for categorical variables. Binary logistic regression analyses were conducted to calculate odds ratios (OR) and 95% confidence intervals (CI) to evaluate the association between risk factors (T2DM, physical inactivity, obesity) and dichotomous outcomes (sarcopenia, slow gait speed). All p-values and 95% CI were calculated and reported with exact values. A two-tailed significance level of 5% ($p \leq 0.05$) was adopted for all statistical tests.

3. Results

3.1. Baseline Characteristics of the Study Sample

The final study sample comprised 175 postmenopausal women, of whom 74 (42.3%) had a diagnosis of T2DM. The sample was stratified into four groups based on T2DM status and physical activity level. Among postmenopausal women with T2DM, a majority (60.8%) were classified as physically active, whereas among normoglycemic women, the majority (58.4%) were classified as insufficiently active.

The baseline anthropometric, body composition, and physical performance characteristics of the four groups are detailed in Table 1. Significant between-group differences were observed for the ASMI ($p = 0.002$), handgrip strength ($p = 0.008$), and gait speed ($p = 0.002$).

Table 1. Baseline characteristics of postmenopausal women by diabetes status and physical activity level (n = 175).

Variables	G1 (n = 45)	G2 (n = 29)	G3 (n = 42)	G4 (n=59)	p
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
Age, years	64.5 ± 9.5	65.1 ± 9.2	61.3 ± 10.4	61.3 ± 10.4	0.200
Body mass index, kg/m ²	27.6 ± 4.2	28.7 ± 4.4	26.7 ± 4.1	26.7 ± 4.1	0.170
Appendicular skeletal muscle index, kg/m ²	6.5 ± 0.8	6.1 ± 0.8	5.7 ± 0.8	5.7 ± 0.8	0.002
Fat mass index, kg/m ²	11.0 ± 3.0	11.9 ± 2.8	11.2 ± 4.9	11.2 ± 4.9	0.670
Fat mass, %	39.6 ± 5.7	41.6 ± 3.6	41.6 ± 7.2	41.6 ± 7.2	0.200
Handgrip strength, kgf	21.9 ± 3.7	22.9 ± 5.1	23.8 ± 4.2	23.8 ± 4.2	0.008
Gait speed, m/s	1.4 ± 0.3	1.1 ± 0.3	1.4 ± 0.3	1.4 ± 0.3	0.002

Abbreviations: G1, physically active women with T2DM; G2, insufficiently active women with T2DM; G3, physically active normoglycemic women; G4, insufficiently active normoglycemic women; SD, standard deviation; T2DM, Type 2 Diabetes Mellitus.

The prevalence of adverse clinical outcomes is presented in Table 2. Notably, there were significant differences in the prevalence of slow gait speed ($p < 0.001$) and sarcopenia diagnosis ($p = 0.008$) across the groups.

Table 2. Prevalence of sarcopenia, obesity, and functional impairment by diabetes status and physical activity level (n = 175).

Variables	G1 (n = 45) n (%)	G2 (n = 29) n (%)	G3 (n = 42) n (%)	G4 (n=59) n (%)	p
Obesity (FMI > 13.0 kg/m ²)	13 (28.9%)	8 (27.8%)	10 (23.8%)	18 (30.5%)	0.900
Low muscle mass (ASMI < 5.5 kg/m ²)	7 (15.6%)	6 (20.7%)	14 (33.3%)	19 (32.2%)	0.150
Low handgrip strength (< 18 kgf)	15 (33.3%)	6 (20.7%)	5 (11.9%)	18 (30.5%)	0.080
Slow gait speed (< 1.0 m/s)	0 (0.0%)	10 (34.5%)	4 (9.5%)	4 (6.8%)	< 0.001
Sarcopenia diagnosis [14]	5 (11.1%)	3 (10.3%)	11 (26.2%)	21 (35.6%)	0.008

Abbreviations: G1, physically active women with T2DM; G2, insufficiently active women with T2DM; G3, physically active normoglycemic women; G4, insufficiently active normoglycemic women; ASMI, Appendicular Skeletal Muscle Index; FMI, Fat Mass Index; T2DM, Type 2 Diabetes Mellitus.

3.2. The Confounding Role of Obesity in the Association Between T2DM and Low Muscle Mass

In the unadjusted analysis, a paradoxical association was observed between T2DM and muscle mass. Women with T2DM had a significantly lower prevalence of low muscle mass compared to their normoglycemic counterparts (17.6% vs. 32.7%; $p = 0.03$), exhibiting 56% lower odds of this outcome (OR: 0.44; 95% CI: 0.21 – 0.91), as shown in Table 3.

Table 3. Risk of low muscle mass associated with type 2 diabetes mellitus (n = 175).

Variables	Low Muscle Mass		p	OR (95% CI)
	No (n = 129) n (%)	Yes (n = 46) n (%)		
<i>Type 2 Diabetes Mellitus</i>				
No (n = 101)	68 (67.3)	33 (32.7)	0.025	0.44 (0.21 – 0.91)
Yes (n = 74)	61 (82.4)	13 (17.6)		

Abbreviations: CI, confidence interval; OR, odds ratio.

However, to investigate this relationship further, a binary logistic regression model was performed, adjusting for glycemic status, obesity (defined by FMI), and physical activity level. In this adjusted model, the protective association of T2DM was no longer present. Instead, obesity emerged as the strongest predictor, significantly increasing the odds of low muscle mass by more than five-fold (OR: 5.48; 95% CI: 1.82 – 16.47; $p = 0.002$). T2DM showed a non-significant trend towards increased odds (OR: 2.05; 95% CI: 0.94 – 4.46; $p = 0.07$), while physical activity level was not significantly associated with low muscle mass (OR: 0.74; 95% CI: 0.36 – 1.52; $p = 0.41$). These results suggest that obesity acts as a critical confounding variable, masking the underlying detrimental relationship between T2DM and muscle mass.

3.3. Physical Inactivity as a Primary Driver of Functional Decline

Physical activity level was strongly associated with functional performance. As detailed in Table 4, postmenopausal women who were insufficiently active had nearly four times greater odds of

exhibiting clinically slow gait speed (< 1.0 m/s) compared to their physically active peers (OR: 3.93; 95% CI: 1.24 – 12.45; p = 0.01).

Table 4. Risk of slow gait speed associated with physical inactivity (n = 175).

Variables	Slow Gait Speed		p	OR (95% CI)
	No (n = 157) n (%)	Yes (n = 18) n (%)		
<i>Physical Activity Level</i>				
Physically active (n = 87)	83 (95.4)	4 (4.6)	0.014	3.93 (1.24 – 12.45)
Insufficiently active (n = 88)	74 (84.1)	14 (15.9)		

Abbreviations: CI, confidence interval; OR, odds ratio.

3.4. Independent Predictors of Sarcopenia

Similar to the findings for low muscle mass, the unadjusted analysis revealed that sarcopenia was significantly less prevalent among women with T2DM compared to normoglycemic women (10.8% vs. 31.7%; p < 0.001). This corresponded to 74% lower odds of sarcopenia in the T2DM group (OR: 0.26; 95% CI: 0.11 – 0.61), as shown in Table 5.

Table 5. Risk of sarcopenia associated with type 2 diabetes mellitus (n = 175).

Variables	Sarcopenia Diagnosis		p	OR (95% CI)
	No (n = 135) n (%)	Yes (n = 40) n (%)		
<i>Type 2 Diabetes Mellitus</i>				
Yes (n = 101)	69 (68.3)	32 (31.7)	< 0.001	0.26 (0.11 – 0.61)
No (n = 74)	66 (89.2)	8 (10.8)		

Abbreviations: CI, confidence interval; OR, odds ratio.

To elucidate the independent predictors of sarcopenia, a binary logistic regression was conducted, adjusting for glycemic status, obesity, and physical activity. In the adjusted model, the relationship was reversed. Both T2DM (OR: 3.80; 95% CI: 1.59 – 9.08; p = 0.003) and obesity (OR: 4.97; 95% CI: 1.62 – 15.20; p = 0.005) were identified as significant and independent predictors of increased odds of sarcopenia. Physical inactivity was not significantly associated with sarcopenia in this model (OR: 0.67; 95% CI: 0.31 – 1.45; p = 0.31). These findings confirm that both diabetes and obesity independently contribute to the risk of sarcopenia, a relationship that was obscured in the unadjusted analysis.

4. Discussion

This cross-sectional study reveals a fundamental dichotomy in the pathophysiology of musculoskeletal decline in postmenopausal women: while T2DM and obesity independently drive the development of sarcopenia through metabolic pathways, physical inactivity emerges as the predominant determinant of functional impairment. This distinction has profound implications for clinical practice, suggesting that preserving muscle mass and maintaining functional capacity may require distinct therapeutic strategies.

4.1. The Sarcopenia Paradox: Unmasking the Role of Obesity and Exercise Modality

Our most striking finding was the apparent protective association of T2DM against low muscle mass in unadjusted analyses—a paradox that was resolved only after controlling for body composition. This methodological insight highlights a critical issue in sarcopenia research: the masking effect of obesity in populations with high adiposity prevalence [15]. After adjusting for FMI, not only did the protective association disappear, but T2DM showed a trend toward increased

sarcopenia risk, consistent with the well-established detrimental effects of chronic hyperglycemia on muscle tissue [16–19].

The mechanistic basis for this relationship likely involves the accumulation of advanced glycation end-products (AGEs) in diabetic muscle tissue, as documented by Mori et al. [19], leading to impaired contractile function and accelerated protein degradation. Our findings align with recent evidence showing that T2DM accelerates age-related muscle loss through multiple pathways, including insulin resistance, mitochondrial dysfunction, and chronic inflammation [16,17].

Equally important is the role of exercise modality in explaining our initial paradoxical findings. The physically active T2DM group (G1) engaged in combined resistance and aerobic training, while normoglycemic active women (G3) performed predominantly aerobic activities. This difference is crucial, as resistance training provides the primary anabolic stimulus for muscle protein synthesis and hypertrophy [20]. Rossi et al. [21] demonstrated that combined training produces superior body composition outcomes compared to aerobic-only exercise in postmenopausal women—findings that directly support our observations and suggest that exercise prescription specificity may partially compensate for the catabolic effects of T2DM.

4.2. Physical Inactivity: The Primary Driver of Functional Decline

While the relationships between T2DM, obesity, and sarcopenia were complex and required multivariate adjustment to elucidate, the impact of physical inactivity on functional performance was unequivocal. Insufficiently active women demonstrated nearly four-fold higher odds of clinically slow gait speed (<1.0 m/s), a threshold associated with increased mortality, hospitalization, and loss of independence [19,22–25].

The strength of this association underscores a critical concept: functional capacity reflects not merely muscle mass but the integrated performance of neuromuscular, cardiovascular, and metabolic systems [24,25]. Our findings align with Sardinha et al. [24], who demonstrated that fitness parameters predict physical independence more accurately than body composition measures alone. This suggests that while sarcopenia represents structural deterioration at the tissue level, gait speed captures the functional consequences of systemic deconditioning.

The clinical significance of reduced gait speed extends beyond mobility. Recent evidence links slow gait speed with cognitive decline [23], increased depression risk [25], and higher T2DM incidence [26], creating a vicious cycle of functional and metabolic deterioration. Our data support the growing consensus that gait speed should be considered the “sixth vital sign” in geriatric assessment [25,27–29].

4.3. Integrating Metabolic and Functional Perspectives

The dissociation between sarcopenia risk factors (T2DM and obesity) and functional decline determinants (physical inactivity) revealed in our study challenges the traditional conflation of these outcomes. While previous research has often treated sarcopenia and functional impairment as parallel consequences of aging and disease [4–6], our findings suggest they may represent distinct phenotypes requiring targeted interventions.

This distinction aligns with emerging evidence from longitudinal studies. Hu et al. [4] demonstrated that sarcopenia predicts cognitive impairment independently of physical activity levels, while Li et al. [5] found associations between sarcopenia and depression that persisted after controlling for functional status. These observations, combined with our findings, suggest that metabolic drivers of muscle loss may operate through pathways distinct from those governing functional capacity.

4.4. Clinical and Public Health Implications

Our findings necessitate a paradigm shift in managing postmenopausal women with T2DM. Current clinical guidelines focus predominantly on glycemic control, with insufficient attention to

functional outcomes that directly impact quality of life and independence [30,31]. The dissociation between metabolic and functional risk factors identified in our study calls for comprehensive care models that address both dimensions simultaneously.

In clinical practice, the integration of functional assessments into routine diabetes care emerges as an immediate priority. Gait speed measurement, requiring less than two minutes to perform, provides a powerful screening tool for disability risk that complements traditional metabolic monitoring [19,25,32]. This simple assessment could identify women at the highest risk for functional decline before irreversible disability occurs. Furthermore, our findings emphasize the need for precision in exercise prescription. Rather than generic recommendations to “be more active,” clinicians should specify resistance training components for sarcopenia prevention, as supported by recent meta-analyses demonstrating superior outcomes with combined training modalities [20,33]. The identification of sarcopenic obesity in our postmenopausal women groups also highlights the inadequacy of BMI as a sole anthropometric measure, supporting the implementation of body composition assessment through DXA or bioimpedance analysis in high-risk populations [15].

From a public health perspective, these findings argue for fundamental revisions to diabetes management guidelines. The incorporation of functional targets alongside glycemic goals would acknowledge the equal importance of maintaining independence in this population. The development of accessible, supervised exercise programs that include resistance training components represents a critical infrastructure need, particularly given that our physically active T2DM participants who engaged in structured combined training showed better muscle mass preservation than their aerobically active normoglycemic counterparts. Early screening initiatives for sarcopenia and functional decline in postmenopausal women with metabolic disease could enable timely interventions before the onset of disability. Recent evidence from successful community-based programs demonstrates the feasibility and cost-effectiveness of such approaches [34,35], suggesting that implementation barriers are surmountable with appropriate resource allocation and policy support.

4.5. Strengths, Limitations, and Future Directions

This study's strengths include objective body composition assessment via DXA, clear group stratification, and comprehensive functional evaluation. The identification of obesity as a confounding variable in the T2DM-sarcopenia relationship represents an important methodological contribution.

However, several limitations warrant consideration. The cross-sectional design precludes causal inference; longitudinal studies are needed to establish temporal relationships between T2DM, physical activity, and musculoskeletal outcomes. Self-reported physical activity assessment introduces potential recall and social desirability bias—future research should employ accelerometry for objective measurement. The heterogeneity in exercise modalities between active groups, while revealing limits to direct comparisons and warrants randomized controlled trials comparing training types.

Additionally, we did not assess dietary factors, vitamin D status, or inflammatory markers, which may modulate the relationships observed [7,36,37]. Future studies should incorporate these variables to develop more comprehensive predictive models.

5. Conclusions

This study elucidates the distinct etiological pathways underlying sarcopenia and functional decline in postmenopausal women. While T2DM and obesity drive sarcopenia through metabolic mechanisms, physical inactivity remains the primary modifiable determinant of functional impairment. These findings underscore the necessity of dual-targeted interventions: metabolic optimization to preserve muscle mass and structured physical activity—particularly resistance training—to maintain functional independence.

For the growing population of postmenopausal women with T2DM, our results advocate for comprehensive care models that extend beyond glycemic control to encompass functional preservation. The implementation of routine functional assessments and precision exercise prescriptions represents not merely an enhancement of current practice but an essential evolution in diabetes care. As the global burden of T2DM continues to rise, preventing functional decline and maintaining quality of life must become equivalent priorities to traditional metabolic targets.

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Data Availability Statement: The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding author.

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

ADLs	Activities of Daily Living
ANOVA	Analysis of Variance
ASMI	Appendicular Skeletal Muscle Index
AWGS	Asian Working Group for Sarcopenia
BMI	Body Mass Index
CI	Confidence Interval
Doce Vida	Supervised Physical Exercise Program for Diabetics
DXA	Dual-Energy X-ray Absorptiometry
FM%	Fat Mass Percentage
FMI	Fat Mass Index
G1	Physically Active Postmenopausal Women with Type 2 Diabetes Mellitus

G2	Insufficiently Active Postmenopausal Women with Type 2 Diabetes Mellitus
G3	Physically Active Normoglycemic Postmenopausal Women
G4	Insufficiently Active Normoglycemic Postmenopausal Women
HIV	Human Immunodeficiency Virus
LABi	Biomechanics Laboratory
LapH	Human Performance Assessment Laboratory
OR	Odds Ratio
SD	Standard Deviation
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
T2DM	Type 2 Diabetes Mellitus
UPE	University of Pernambuco
χ ²	Chi-squared test

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