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Resistance training volume and nutrient intake on lean mass and strength in young women

Efeito do volume de treinamento de força e consumo de nutrientes na massa magra e força em mulheres jovens

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ABSTRACT

Introduction: Skeletal muscle plays a central role in resistance training adaptations and overall health, with hypertrophy and strength gains influenced by both genetic and external factors, including training volume (VT), protein intake and energy balance. Objective: This study aimed to explore the interplay effect of individual VT and dietary intake on muscle hypertrophy and strength responsiveness from resistance training. Methods: Forty-five untrained women including strict vegetarians (n = 25; 28.7 ± 4.6 years; 162.3 ± 9.3 cm) and non-vegetarians (n = 20; 30.7 ± 6.6 years; 162.7 ± 9.2 cm) performed a 16-week exercise intervention. Macronutrient intake was assessed through dietary record while individual VT was calculated by the sum of each exercise volume load (sets × repetitions × load). Muscular hypertrophy was estimated based on lower limb lean soft tissue (△ LST) measured via DXA, while strength gains were evaluated through maximal knee extension and flexion peak torque (△ SUM PT) at 60º/s using isokinetic dynamometry. The interaction between VT and macronutrient intake with hypertrophy and strength gains was evaluated using multiple polynomial regressions analyses. Results: The interaction between VT and protein intake (g/kg) significantly explained changes in △ LST ($p = 0.034$; $R^2 = 0.28$), while the interaction between VT and energy intake (kcal/kg) significantly explained changes in △ SUM PT ($p = 0.031$; $R^2 = 0.29$). Conclusion: Individual VT appeared to elicit greater effect on muscle hypertrophy when accompanied by protein intake exceeding 1.5 g/kg. High individual VT combined with low energy intake (15–20 kcal/kg) led to strength loss, whereas higher energy intake (35–45 kcal/kg) associated with greater VT supported more pronounced strength gains.

Keywords: Strength training; Weight lifting; Exercise volume; Muscle growth; Muscle hypertrophy; Protein.

RESUMO

Introdução: O músculo esquelético desempenha um papel central nas adaptações ao treinamento de força e na saúde geral, com os ganhos de hipertrofia e força sendo influenciados por fatores genéticos e externos, incluindo volume de treino (VT), ingestão de proteínas e balanço energético. Objetivo: Explorar a interação entre o volume de treino individual e a ingestão dietética na responsividade de hipertrofia muscular e ganho de força provenientes do treinamento de força. Métodos: Quarenta e cinco mulheres não treinadas, incluindo vegetarianas estritas (n = 25; 28,7 ± 4,6 anos; 162,3 ± 9,3 cm) e não vegetarianas (n = 20; 30,7 ± 6,6 anos; 162,7 ± 9,2 cm), participaram de uma intervenção de exercícios com duração de 16 semanas. A ingestão de macronutrientes foi registrada por diário alimentar, enquanto o VT foi calculado pela soma de séries × repetições × carga em cada exercício. A hipertrofia muscular foi estimada pela variação da massa livre de gordura dos membros inferiores (△ LST) via DXA, e os ganhos de força pela variação do pico de torque (△ SUM PT) na extensão e flexão de joelhos a 60º/s, por dinamometria isocinética. A interação entre VT e ingestão de macronutrientes foi analisada por regressões polinomiais múltiplas. Resultados: A interação entre VT e ingestão proteica (g/kg) explicou significativamente as mudanças em △ LST ($p = 0,034$; $R^2 = 0,28$). A interação entre VT e ingestão energética (kcal/kg) explicou significativamente △ SUM PT ($p = 0,031$; $R^2 = 0,29$). Conclusão: O VT pareceu promover maior hipertrofia muscular quando acompanhado de ~1,5 g/kg de ingesta proteica. Alto VT combinado com baixa ingestão energética levou à perda de força, enquanto uma ingestão energética mais elevada associada a alto VT favoreceu maiores ganhos de força.

Palavras-chave: *Treinamento de força; Levantamento de peso; Volume de exercício; Crescimento muscular; Hipertrofia muscular; Proteína.*

Introduction

The skeletal muscle is one of the most important organs for human health. Its significance extends beyond its structural role in supporting body mass and enabling movement; it also functions as an endocrine organ, capable of producing and secreting myokines[1].

These myokines play a crucial preventive role against chronic diseases such as cardiovascular disease, type 2 diabetes, cancer, and dementia[1].

Muscle hypertrophy, defined as the increase in muscle fiber size, is a key adaptation to resistance training (RT), typically noticeable after 8–12 weeks. It results from mechanical and metabolic stress activating pathways such as satellite cell proliferation, enhanced protein synthesis, and glycogen supercompensation[2-6]. In contrast, strength gains often occur earlier (4–8 weeks), primarily due to neural adaptations like improved motor unit recruitment and neuromuscular efficiency[7-8].

Hypertrophic responses vary individually, influenced by intrinsic (e.g., IGF-1, myogenin expression[9-10] and extrinsic factors. Among extrinsic variables, training volume (VT), the cumulative load across sessions, is a major determinant of hypertrophy, especially when using ~30–90% One-repetition maximum (1RM) loads taken near failure and performed for 3–6 sets per muscle group[11-13]. Conversely, maximal strength is best developed through high-load training (≥80% 1RM), with progressive volume and intensity[12,14,15].

Diet also modulates RT adaptations. Protein intake of 1.3–1.6 g/kg/day optimally supports muscle hypertrophy[16,17], though higher amounts offer no added benefit. Protein's effects on strength are training-dependent[18]. Energy balance further influences results; while strength may be preserved during caloric deficits, lean mass gains are impaired when intake is >500 kcal below needs[19].

Despite structured RT, some individuals show minimal improvements—so-called non-responders[10,20,21]. While individual RT responsiveness has been studied, the combined effects of VT and diet on strength and hypertrophy outcomes remain unclear

Methods

Trial design

This article is part of a non-randomized controlled trial, which recruited healthy young women who were not engaged in RT programs and had adopted a strict vegetarian diet or non-vegetarian diet for at least 6 months prior the study. The project was approved by the local ethics committee (registered number: 5.322.759), conducted according to the Declaration of Helsinki, and registered at Clinical Trials (NCT05576337).

The primary analysis, reported in a separate article currently under peer review, compared muscle hypertrophy and strength outcomes between strict vegetarian and non-vegetarian groups and found no statistically significant differences. Based on these findings, the present study utilized the entire sample (strict vegetarian plus non vegetarian women) to explore individual responsiveness to RT adaptations. This approach aimed to assess within-individual variability and better understand predictors of hypertrophy and strength gains.

Participants

The study sample comprised healthy young women aged between 20 and 40 years, with body mass index of 18 to 29.9 kg/m^2. Participants were required to have no history of RT and no consumption of protein or amino acid-based supplements for at least six months prior to the study. Furthermore, they needed to follow either a strict vegetarian or non-vegetarian diet during that period. To ensure safety and reliable results, candidates were screened for any musculoskeletal disorders that might restrict their ability to participate in a RT program.

Before the study, all participants were informed about the purpose, procedures, potential benefits, and risks associated with participation. Written informed consent was obtained from each participant prior to their inclusion in the study.

Interventions

All participants were instructed to avoid consuming protein or amino acid supplements, engaging in additional physical training outside the study, and drinking alcohol on training days. Both groups were also advised not to alter their eating habits and to log their dietary intake over three days (two-week days and one weekend day) at three different time points: before the study (PRE), during week four, eight, and 12 and after the RT period (POST). At PRE and POST, the participants were invited to the university laboratory for assessments of body composition, avoiding the participant's menstrual period.

After baseline evaluation, all participants followed the same 16-week RT protocol, encompassing two sessions per week, with a linear periodization model. Exercise selection included knee extension, knee flexion, 45º leg press, hip abduction and calf raise. The weekly mesocycles were organized based 1RM zones as follows: weeks one and two consisted of two sets of 12-15 RM; weeks three, four, and five consisted of three sets of 10-12 RM; weeks six through nine involved three sets of 8-10 RM; weeks 10 through 13 involved four sets of 8-10 RM; and weeks 14 through 16 involved

four sets of 6-8 RM. The intensity of each exercise was adjusted individually, with the load increased whenever a participant could perform more repetitions than the target range for a given load. All training sessions were supervised by experienced instructors, with up to five participants per instructor. Apart from the nutritionist, all evaluators were blinded to the participants' group assignments.

Outcomes

- **Dual-energy X-ray absorptiometry**

The lean soft tissue (LST; kg) of the dominant lower limb (LL) of the participants, was assessed by dual-energy X-ray absorptiometry (DXA; Lunar Prodigy Advance, GE Healthcare, Chicago, USA) device according to the manufacturer's recommendations. All scans were conducted by the same evaluator. The obtained images were analyzed considering a defined LL region of interest (ROI), using the manufacturer's provided software. Thigh ROI was defined by the head of the femur to the tibial edge, while for the calf ROI was defined the lateral border of the tibia to the lateral malleolus of the ankle[22]. LL ROI was composed of the sum of both thigh and calf ROIs. Intervention muscle hypertrophy estimation (Δ LST; kg) was defined by (Post LST - Pre LST).

- **Muscle strength**

The dynamic peak torque (PT; N.m) of the dominant knee extensors (KE) and flexors (KF) was evaluated using an isokinetic dynamometer (Cybex Inc., Ronkonkoma, USA). Participants were familiarized during different sessions, each separated by at least 2 days. Before the test, participants underwent preparatory procedures: Following a warmup of 10 repetitions at 180º/s, participants performed a three-repetition familiarization at 60˚/s. After a 1-minute interval, participants performed five maximal concentric/concentric repetitions at 60˚/s for both extension and flexion[23]. Following another 1-minute rest, they were familiarized with three submaximal concentric/eccentric flexion repetitions at 60˚/s. After an additional 1-min interval, the test began, consisting of five maximal repetitions at 60º/s to assess maximal eccentric PT. The isokinetic PT was defined as the highest PT value within all 5 repetitions for each category of contraction. For strength analysis, baseline concentric extension, flexion and eccentric flexion PT were summed (PT SUM) and divided by body mass in order to normalize it. Intervention strength gains (Δ SUM PT; N.m/kg) was defined by (Post PT SUM - Pre PT SUM).

- **Individual training volume**

All exercises encompassing the LL muscles were included in individual volume-load calculation (i.e. knee extension, knee flexion, 45º leg press, hip abduction and calf raise).

The VT was defined by the sum of each LL exercise volume load, calculated as (sets × repetitions × load) for each participant, recorded during every RT session by instructors throughout the intervention; this method is likely the most appropriate method of accounting VT, as it accounts for all exercise main variables[24]. VT was normalized by body mass in order to account for intrinsic individual strength differences.

- **Dietary intake**

The 3-day food record, encompassing two typical days (week days) and one atypical day (weekend day), was collected at the PRE (week 0), week 4,8, and 12, and at POST (week 16) to evaluate relative macronutrient intake (g/kg). For each period under evaluation, the mean daily intake of energy and macronutrients (protein, carbohydrate and fat) was calculated.

To reduce potential bias, participants were trained to accurately complete their food logs. They were instructed to record each meal, food, and beverage consumed, providing both text descriptions and photographs. The records also included portion sizes (measured either with a kitchen scale or by household measurements), detailed ingredients for homemade dishes, and the brand of any commercial products consumed[25]. Evaluators remained in close contact with participants throughout the study, ensuring that the records were regularly reviewed, the questions were addressed, and participants remained consistent with their logs. The dietary records were analyzed using Webdiet Software (version 3.0).

Sample size

The sample size was estimated considering the muscle mass outcome, in order to find a non-negligible effect size (Cohen's d = 0.4 or f = 0.2; $\alpha = 0.05$; $1-\beta = 0.8$), for interaction (moment*intervention) in ANOVA, resulting in 54 participants (27 women per group)[26].

Statistical Method

Age, body mass, height, macronutrients intake, VT,

LST and PT were described as mean (standard deviation) while group differences were tested using independent samples t-test. Intervention repeated measures interaction for LST and PT was analyzed using linear mixed effect models accounting for group differences and random effects. For correlations, muscle hypertrophy (Δ LST) and strength gains (Δ SUM PT) were defined as Post-Pre values.

In order to evaluate the nonlinear interaction between VT and dietary intake into RT adaptation variables (hypertrophy and strength), several polynomial regressions accounting for two predictor variables, their quadratic terms and their interaction were performed utilizing ordinary least squares method through scikit-learn and statsmodels Python libraries[27]. The first predictor variable was held constant as VT for all models, while the second was alternated for each dietary intake variable (DI) (protein intake, carbohydrate intake, fat intake and energy intake). The basic structure of each polynomial regression was:

$$RTA=\beta_0+\beta_1 VT+\beta_2 VT^2+\beta_3 DI+\beta_4 DI^2+\beta_5 VTDI+e$$

Overall models and coefficients individually significance was assessed through ANOVA omnibus F test and Wald's t-test respectively (alpha = 0.05). Normality was assessed through residuals plot inspection. Multicollinearity was assessed and variance inflation factor (VIF) while autocorrelation was assessed though Durbin Watson test. Individual VT and dietary intake were expressed as deviations around their means in order to reduce multicollinearity[27]. Post hoc achieved power was calculated utilizing Gpower 3.1.9.7.

In order to verify possible bias to our main analysis interpretation, two-tailed spearman correlation heatmap adjusted with Benjamini-Hochberg correction was utilized in order to assess monotonic correlations between (Δ LST, Δ SUM PT) x (VT, protein intake, carbohydrate intake, fat intake, energy intake, age). Separate spearman tests were utilized to assess correlation between (VT) x (protein intake, carbohydrate intake, fat intake, energy intake) and (hypertrophy) x (Δ SUM PT, baseline SUM PT). This non-parametric test was chosen for its robustness in detecting associations while not assuming linearity, given the physiological uneven dose-response patterns.

In order to calculate the LST hypertrophy responsiveness cut-off point, we utilized a similar previous test–retest scans method[21]. Responsiveness was defined by two times the standard error of measurement (SEM), calculated by the equation SEM = SDdiff/√2, where SDdiff is the standard deviation of the differences between two evaluations separated by a 7-day interval[28]. Additionally, the intraclass correlation coefficient was calculated in order to estimate the reliability of the measure[29].

Results

Baseline data

Seventy-one participants began the training program, with thirty-seven assigned to a strict vegetarian group and thirty-four to a non-vegetarian group. Twenty-six participants dropped out of the intervention due to lack of time, while only one participant from the vegetarian group withdrew after beginning to consume eggs during the intervention. With the final sample composed of 25 strict vegetarian and 20 non-vegetarian participants. Therefore, forty-five participants were included in the main analysis. Descriptive results are displayed at Table 1.

Intervention effect

Intervention effects on strength and hypertrophy are presented at Table 1. Of the sample, 21 participants (46%) did not achieve Δ LST responsiveness, and eight participants (17%) did not achieve Δ SUM PT responsiveness. Among those who did not achieve responsiveness, four individuals (8% of the sample) failed to achieve concomitant responsiveness in both Δ LST and Δ SUM PT. Most variables showed no significant group differences ($p > 0.05$), except for protein (vegetarian: 1.00 ± 0.3 g/kg, non-vegetarian: 1.20 ± 0.3 g/kg; $p = 0.003$) and carbohydrate intake (vegetarian: 4.41 ± 0.3 g/kg, non-vegetarian: 3.20 ± 0.3 g/kg; $p < 0.001$). Despite these differences, both groups exhibited significant improvements in LST and performance over time ($p < 0.001$), with no group-time interaction ($p > 0.05$).

Spearman correlation

Spearman correlation heatmap analysis is displayed at Figure 1. The correlation between Energy intake and Δ SUM PT was the only test which achieved significance ($p = 0.002$ $R = 0.560$). For the individual tests, protein intake did not demonstrate a significant correlation with VT ($p = 0.667$; $R = -0.070$), while Δ LST demonstrated a significant correlation with Δ SUM PT ($p = 0.002$; $R = 0.439$). Correlation statistics for individual models can be found in Appendix 1.

Table 1 – Descriptive results and resistance training repeated measures separated by group.

Descriptive	Strict vegetarian	Not vegetarian	Whole sample mean	Group p-value	
	Mean (Standard deviation)	Mean (Standard deviation)	Mean (Standard deviation)		
Age (years)	28.7 (4.6)	30.7 (6.6)	29.5 (5.5)	0.220	
Height (cm)	62.3 (9.3)	62.7 (9.2)	162.0 (7.0)	0.864	
Body mass (kg)	163.2 (7.0)	161.9 (8.3)	62.6 (9.1)	0.553	
Individual training volume (a.u.)	10186.8 (2348.3)	10217.3 (2260.3)	10221.0 (2305.0)	0.967	
Energy (kcal/kg)	29.7 (8.0)	27.1 (5.7)	28.5 (7.0)	0.225	
Protein (g/kg)	1.0 (0.3)*	1.2 (0.2)*	1.1 (0.3)	0.003*	
Carbohydrate (g/kg)	4.4 (1.2)*	3.2 (0.9)*	3.8 (1.2)	< 0.001*	
Fat (g/kg)	1.0 (0.3)	1.1 (0.2)	1.0 (0.3)	0.326	
Linear mixed model	Pre	Post	Time p-value	Group p-value	Time x Group p-value
Strict vegetarian - Lean soft tissue (kg)	7.0 (1.0)	7.3 (1.0)	< 0.001*	0.680	0.586
Not vegetarian - Lean soft tissue (kg)	6.9 (1.0)	7.1 (1.0)			
Strict vegetarian - Sum Peak Torque (N.m/kg)	4.2 (0.8)	4.8 (0.8)	< 0.001*	0.764	0.538
Not vegetarian - Sum Peak Torque (N.m/kg)	4.3 (0.8)	4.9 (0.8)			

* Significant interaction ~ p < 0.05.

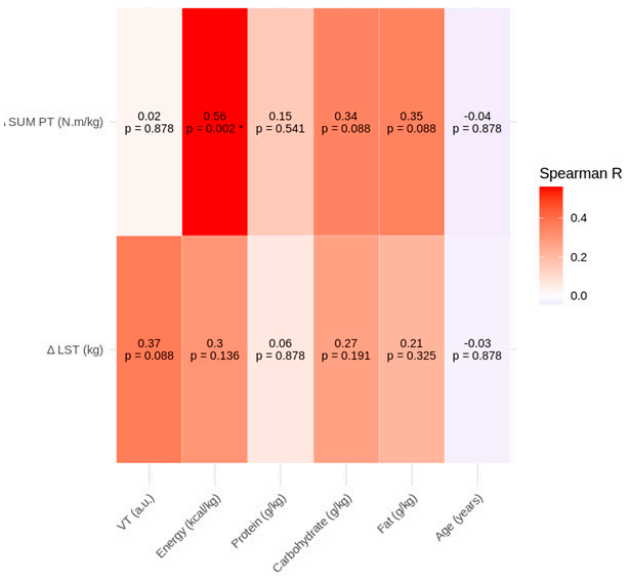

Figure 1 – Spearman correlation heatmap between resistance training adaptations (y axis: strength gains and muscle hypertrophy) and extrinsic factors (x axis: individual training volume, dietary intake variables and age).
Colour gradient demonstrates the strength of the correlation.
Δ SUM PT = Strength gains; Δ LST = Muscle hypertrophy; VT = training volume; Spearman R = spearman rank correlation coefficient

Multiple Polynomial regression

Polynomial model fitting statistics are presented at Table 2 and Appendix 2. The only polynomial model which achieved significance in explaining Δ LST was the (protein intake x VT) (R^2 = 0.288; p = 0.034). Likewise, the only polynomial model which achieved significance in explaining Δ SUM PT was the (energy intake x VT) (R^2 = 0.286; p = 0.035) Model statistics are detailed at Table 2. The hypertrophy prediction model is presented in Figure 2, while the strength predictive model is visually presented in Figure 3.

Responsiveness threshold

Test-retest scan method was applied in a subsample of 6 participants, which resulted in an excellent intraclass correlation coefficient of 0.997 (0.982-1.00 C.I.)[29] and a SEM of 92 g for the LST, resulting in a responsiveness cutt-off point of 184 g for LST.

Discussion

This article explores how individual VT and dietetic intake are associated with muscular adaptations induced by RT in the context of our sample. The individual VT exerted a positive logarithmic relationship with hypertrophy, suggesting additional volume provides no further meaningful benefits beyond a certain threshold and may even have detrimental effects. Nonetheless, this relationship was modulated by protein intake to further enhance muscle hypertrophic stimulus (Figure 2). Conversely, the relationship between individual VT and strength adaptations shifted entirely depending on overall caloric intake. When caloric intake was low, higher individual VT seemed to exert a detrimental effect on strength adaptations. However, in a high-caloric intake environment, increased individual VT significantly enhanced strength adaptations (Figure 3).

Our results corroborate a logarithmic relationship between individual VT and hypertrophy when protein

Table 2 – Results of overall polynomial regression models predicting resistance training adaptations (muscle hypertrophy and strength gains) based on dietary variables (protein, carbohydrate, fat and energy), with individual training volume as a fixed variable through all models.

Resistance training adaptation	Dietary (+ individual training volume)	R^2	Adj. R^2	F	p	AIC	BIC	Durbin Watson	Achieved Power
△ Lean soft tissue (kg)	Protein (g/kg)	0.288	0.183	2.746	0.034*	16.04	26.18	1.319	0.831
△ Lean soft tissue (kg)	Carbohydrate (g/kg)	0.156	0.032	1.260	0.303	22.81	32.94	1.141	0.454
△ Lean soft tissue (kg)	Fat (g/kg)	0.144	0.019	1.148	0.354	23.37	33.50	1.239	0.415
△ Lean soft tissue (kg)	Energy (kcal/kg)	0.184	0.064	1.537	0.204	21.46	31.59	1.211	0.545
△ Sum Peak Torque (N.m/kg)	Protein (g/kg)	0.115	-0.015	0.882	0.503	84.29	94.42	2.031	0.323
△ Sum Peak Torque (N.m/kg)	Carbohydrate (g/kg)	0.216	0.100	1.879	0.126	79.45	89.59	1.756	0.645
△ Sum Peak Torque (N.m/kg)	Fat (g/kg)	0.123	-0.006	0.953	0.460	83.93	94.06	1.949	0.348
△ Sum Peak Torque (N.m/kg)	Energy (kcal/kg)	0.293	0.189	2.817	0.031*	75.31	85.44	1.679	0.842

* Significant interaction ~ $p < 0.05$. R^2 = coefficient of determination; Adj R^2 = coefficient of determination adjusted for multiple predictors ; F = F-statistic; p = p-value; AIC = Akaike information criterion; BIC = Bayesian information criteriom.

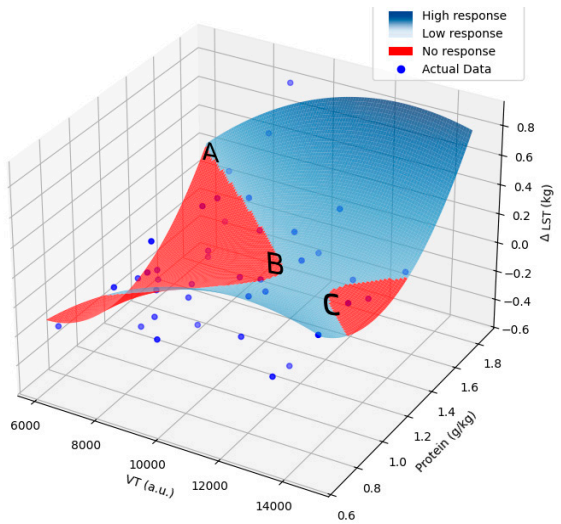

Figure 2 – Three-dimensional polynomial model fit between muscle hypertrophy (△ LST; kg), individual training volume (VT; a.u.) and protein intake (g/kg). Red plane indicates either negative or no hypertrophic responsiveness. (A) Minimum individual volume with maximum protein intake resulted in barely hypertrophy responsiveness. (B) Average individual volume with ~1.1 g/kg of protein intake resulted in barely hypertrophy threshold, which could be improved with higher protein intakes. (C) Maximum individual volume hypertrophy with low protein intake compromise hypertrophy. Hypertrophy threshold was derived from SEM, set as 184 g.

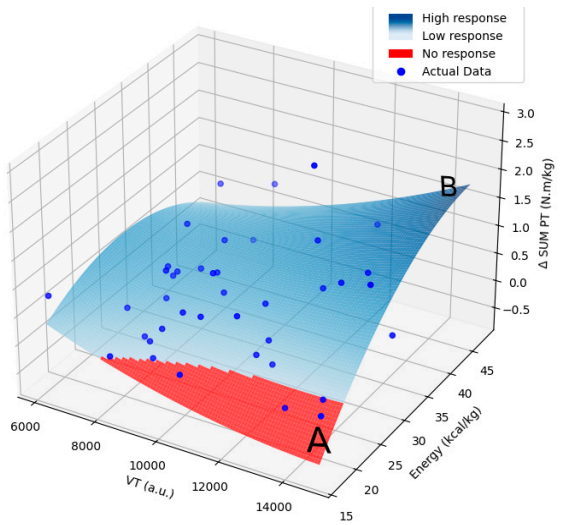

Figure 3 – Three-dimensional polynomial model fit between strength gains (△ SUM PT; N.m/kg), individual training volume (VT; a.u) and energy intake (Energy; kcal/kg). (A) Maximum individual volume with low energy intake (15-20 kcal/kg) resulted in loss of strength. (B) Maximum individual volume with high energy intake (35-45 kcal/kg) resulted in greater strength adaptations. Red plane indicates either negative or no strength responsiveness. Blue plane indicates positive strength responsiveness. Strength threshold set as 0 N.m/kg.

intake is low (~0.6 g/kg). However, when protein intake was high (~1.8 g/kg), the hypertrophic response followed a linear relationship with individual VT. Accordingly, minimal levels of VT appeared to elicit no response in hypertrophy, regardless of the protein intake (Figure 2-A). In contrast, participants with average VT and moderated protein intake (~1.2 g/kg) demonstrated a minimal hypertrophy responsiveness (Figure 2-B). Additionally, the effect of the average VT could be improved by further enhancing protein intake (~1.4 - 2.0 g/kg). Finally, maximum VT appeared to promote hypertrophic response both in low and moderate protein intakes (~0.6 - 1.2 g/kg). However, at those lower protein intakes, the hypertrophic response was low and barely differentiated from the minimal responsive threshold, corroborating to an inverted U-shaped relationship[30]. Thus, enhancing protein intake to around 1.4 to 2.0 g/kg appeared to be substantial in order to efficiently improve the hypertrophic response when reaching the maximum individual dose-response volume plateau (Figure 2-C). It is important to note that DXA measures lean soft tissue, including non-con-

tractile components such skin, connective tissue, and water, potentially complicating the interpretation of muscle hypertrophy. However, all scans were standardized with test–retest reliability checks to control hydration status and participant positioning. Given the long term duration of our intervention (i.e. 16 weeks) and the nature of RT itself, substantial non-contractile changes are unlikely; thus, increases in LST are likely to reflect improvements in muscle tissue. Nevertheless, glycogen-related water shifts could transiently elevate lean mass values, representing a limitation when interpreting adaptations in terms of sarcoplasmic or myofibrillar hypertrophy.

Consuming enough protein is essential for health and physical activity, as it supports not only muscle maintenance but also immune function, hormone production, and tissue repair, with both essential and nonessential amino acids needed in the diet to fully support these roles[31]. Since women who consumed higher amounts of protein showed enhanced hypertrophic responses, our results align with existing recommendations that suggest increased protein intake (beyond the general recomendation of 0.8 g/kg/day) is beneficial for individuals engaged in resistance training. This supports the broader evidence that protein needs are elevated in physically active populations, particularly for promoting muscle growth, recovery, and overall health[31].

Even though our 16-week intervention qualifies as long-term, we observed discrepancies in the expected relationship between strength and muscle hypertrophy aligned with VT, whereas greater VT favoured hypertrophy but not strength if caloric intake was not high. However, these findings are supported by Schoenfeld et al.[32], who compared VT by manipulating weekly sets between groups and found that higher volume led to greater hypertrophy gains but did not significantly impact strength. Additionally, meta-analytical data also suggest an interplay effect of training to volitional failure on muscle hypertrophy and strength, whereas greater VT may enhance the hypertrophic response[33] but also may compromise strength gains[33,34]. This effect of training to volitional failure is particularly relevant since it justifies the increased individual VT in our intervention as we will discuss further.

To our best knowledge, no study has yet accounted for the complex interplay between caloric intake and VT, a relationship that may affect the magnitude of strength adaptations elicited by RT. Our results suggest that, regardless of VT, lower caloric intakes lead to poor strength adaptations, with the effects worsening as VT increases (Figure 3-A). This is further supported by studies showing that caloric restriction can elevate plasma cortisol[35], impair strength[36], and reduce RT knee extensor strength adaptations in overweight population[37]. However, in an caloric abundant environment, VT appeared to enhance strength instead of impairing it (Figure 3-B), which can be explained by higher volumes having a long term positive effect on cortisol, testosterone and IGF-1 resting serum[38]. However, these benefits likely depend on stress being acutely induced by training rather than by dietary restriction. It is important to note that summing distinct isokinetic contraction modes may not accurately reflect functional strength adaptations due to their specific physiological characteristics, fixed angular velocities, and isolated-joint kinetics. These factors limit both generalization and practical applicability.

For muscle hypertrophy, our polynomial model (protein intake x VT) significantly explained 28.8% (18.3% adjusted; 83.1% achieved power) of the variance, while for strength, our polynomial model (energy intake x VT) significantly explained 29.3% (18.9% adjusted; 84.2% achieved power). A possible reason for this relatively low R^2 may reflect methodological factors like DXA's inclusion of non-contractile tissue, multiple participants per instructor and self-reported diets, which may contribute to residual variance. Despite this, our models remained significant ($p<0.05$) and powered (>80%). Likewise, while this R^2 may seem modest, it aligns with the understanding that the complex interplay of genetics (hormonal expression, muscle fiber composition) and environmental factors (work, stress, sleep habits) can make higher R^2 values unrealistic or unlikely[39]. Thus, our model's R^2's may be considered meaningful in clinical research[39].

We propose that individual tolerance and response to perceived effort during sets to failure primarily influenced VT. While training to failure can impair recovery and hinder muscle growth and strength due to increased muscle damage[40,41], it also acutely elevates growth hormone and IGF-1 levels[42], enhancing hypertrophic and strength adaptations. This dual effect underscores the complexity of VT and suggests a third nutritional variable may help explain variability in RT outcomes. Our findings suggest that protein intake plays a key role in muscle hypertrophy, while energy intake is a critical factor in strength adaptation.

However, no internal load variable, such as the rate of perceived exertion, was objectively recorded in this study. Therefore, our interpretation of the relationship between individual external VT and the observed RT adaptations remains inconclusive.

This study has several methodological limitations that should be considered when interpreting the findings. First, the non-randomized design increases the risk of selection bias and residual confounding, as participants were allocated to groups based on pre-existing dietary habits. Third, dietary intake was self-reported via 3-day food records, which are prone to recall bias and underreporting despite nutritionist supervision and photographic verification. Fourth, internal training load variables such as rate of perceived exertion were not recorded, limiting interpretation of individual responsiveness. Additionally, the menstrual cycle phase was not biochemically verified, and training supervision occurred at a ratio of up to five participants per instructor, potentially affecting exercise fidelity. The responsiveness threshold was based on a small subsample, which may affect generalizability. Given these limitations and the exploratory nature of this analysis, the results should be interpreted with caution and confirmed in future randomized controlled trials.

In this non-randomized exploratory study, we observed that individual VT played a primary role in initiating muscle hypertrophy, while adequate protein intake appeared to amplify this response, particularly at higher volumes. The relationship between VT and hypertrophy followed a logarithmic dose–response pattern, in which moderate volumes produced optimal growth, and further increases in volume did not yield additional benefits. Adequate protein intake enhanced the hypertrophic effect of high VTs but did not independently stimulate growth in low-volume contexts. For strength adaptations, VT interacted with caloric intake, with higher energy availability supporting greater gains, while lower caloric intake was associated with impaired strength development. These findings highlight the importance of aligning VT with sufficient protein and energy intake to support not only performance goals but also long-term health and functional capacity through regular physical activity.

However, given the non-randomized design, reliance on DXA for hypertrophy assessment, self-reported dietary intake, absence of internal load measures, and the modest proportion of variance explained by our models, these findings should be interpreted with caution. Future randomized controlled trials using more precise measurement tools and larger, more diverse samples are warranted to confirm and extend these observations.

Conflict of interest

The authors declare no conflict of interest.

Author's contributions

Strey BM: Conceptualization; Methodology; Software; Validation; Formal analysis; Investigation; Visualization; Writing – original draft; Writing – review & editing; Approval of the final version. Martini GL and Schemes MB: Conceptualization; Methodology; Software; Validation; Investigation; Data curation; Project administration; Visualization; Writing – original draft; Writing – review & editing; Approval of the final version. Irigoyen A: Methodology; Software; Validation; Formal analysis; Visualization; Writing – original draft; Writing – review & editing; Approval of the final version. Souza CG: Conceptualization; Methodology; Investigation; Resources; Supervision; Writing – original draft; Writing – review & editing; Approval of the final version. Pinto RS: Conceptualization; Methodology; Investigation; Resources; Supervision; Project administration; Funding acquisition; Writing – original draft; Writing – review & editing; Approval of the final version.

Declaration regarding the use of artificial intelligence tools in the article writing process

For the development of this manuscript, the artificial intelligence tool DEEPSEEK was used for the following activity(ies): Text organization and revision. The authors declare that all material derived from such process has been reviewed, and the authors assume full responsibility for all the content of the manuscript.

Availability of research data and other materials

The data of this study is available on demand from referees

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Appendix

Appendix 1 – Ancilatory spearman correlations statistics

Dependent variable	Predictor variable	R	p
△ Lean soft tissue (kg)	Baseline strength gains (N.m/kg)	-0.185	0.223
△ Lean soft tissue (kg)	△ Sum Peak Torque (N.m/kg)	0.439	0.002*
Individual training volume (a.u.)	Baseline strength gains (N.m/kg)	0.153	0.344
Individual training volume (a.u.)	Energy (g/kg)	0.089	0.0583
Individual training volume (a.u.)	Protein (g/kg)	-0.070	0.667
Individual training volume (a.u.)	Carbohydrate (g/kg)	0.207	0.201
Individual training volume (a.u.)	Fat (g/kg)	0.125	0.444
Individual training volume (a.u.)	Age (years)	-0.233	0.147

* Significant interaction ~ $p < 0.05$.

Appendix 2 - Results of individual coefficients from significant polynomial regression models predicting hypertrophy and strength based on individual training volume, protein intake (for hypertrophy) and energy intake (for strength).

Dependent variable	Predictors	βi estimate	SD	t	p	VIF
△ lean soft tissue (kg)	$\beta_{0\ (kg)}$	0.125	0.070	1.799	0.081	
	Individual training volume (a.u.)	6e-05	2.3e-05	2.750	0.009*	1.400
	Individual training volume² (a.u.)	-1.4e-08	8.9e-09	-1.590	0.121	1.322
	Protein (g/kg)	-0.034	0.176	-0.194	0.847	1.290
	Protein² (g/kg)	1.207	0.470	2.561	0.015*	1.279
	Individual training volume.Protein(a.u.)	1.207	7.8-e05	0.151	0.881	1.519
△ Sum Peak Torque (N.m/kg)	$\beta_{0\ (kg)}$	1.065	2.668	4.336	> .001*	
	Training volume (a.u.)	-5e-04	0.000	-0.105	0.917	1.336
	Training volume² (a.u.)	1.0e-08	1.9e-08	0.574	0.577	1.310
	Energy (kcal/kg)	0.095	0.098	3.574	0.001*	1.330
	Energy² (kcal/kg)	-0.002	0.001	-1.879	0.069	1.271
	Training volume.Energy(a.u.)	9.7e-06	6.1e-06	1.568	0.126	1.060

* Significant interaction ~ $p < 0.05$; β_0 = Intercept. Coefficients (βi) were estimated for predictor variables centered around their means for inference purposes: SD = standard deviation; t = t-value; p = p-value; VIF = Variance inflation factor

Reviewers' assessment

The reviews of this article were originally conducted in Portuguese. This version has been translated using ChatGPT and subsequently reviewed by the Chief Editors.

Reviewer A

Anonymous

- Was any evidence of plagiarism observed in the manuscript?
  No
- Did the authors provide clarification regarding the ethical procedures adopted for conducting the research?
  Yes

Comments to the authors:

- The study aimed to verify the moderating effects of training volume and protein intake on muscle hypertrophy in young adult women. The study presents an interesting proposal with good practical applicability. However, several issues need to be addressed to improve the overall flow of the manuscript. Please see the comments below:
- It is necessary to standardize the Portuguese term referring to the exercise modality. For example, the title uses "treinamento de força" (strength training), but in the abstract introduction, it is described as "treinamento de resistência" (resistance training). It is recommended to use a single term consistently, such as "strength training" or "resistance training."
- In the abstract, indicate that the participants were previously untrained women.
- The terms massa magra and lean mass are not entirely appropriate. Since the measurement was obtained via DXA, the correct term would be fat- and bone-free mass (MIGO in Portuguese) or lean soft tissue (LST) in English. Please standardize this terminology in the title, abstract, and throughout the text.
- It seems that this study is part of a larger project that aimed to compare vegan and non-vegan diets. If that is correct, please indicate that this is an exploratory study that forms part of a broader project with a different primary objective. Otherwise, it would be interesting to present comparative results between the vegan and non-vegan groups.
- In the Intervention section, describe which exercises were performed during training.
- Page 6, line 19: "Individual training volume section." This sentence appears out of context. Please rewrite it.
- Page 7, line 8: The most common English term is lean soft tissue (LST).
- It is not clear what the responsiveness analysis added to the study. Moreover, the cutoff point applied—although used in other experiments—may be too strict. It is at least questionable that in a sample of untrained, healthy individuals, and therefore with a large adaptive window, almost half of the sample did not show hypertrophy after a hypertrophy training program. In this sense, one could question the application, monitoring, and control of the training, since it appeared effective for only half of the participants. Although responsiveness is an important topic of study, it is unclear how much this analysis contributes to the main purpose of this investigation.
- Figure 1: It would be more informative for readers if the exact p-values were reported, even for non-significant correlations.

Final Decision

- Substantial revisions required

Reviewer B

Charles Phillipe de Lucena Alves
Federal University of Pelotas, Rio Grande do Sul, Brazil

- Was any evidence of plagiarism observed in the manuscript?
  Not applicable
- Did the authors provide clarification regarding the ethical procedures adopted for conducting the research?
- Yes

Comments to the authors

- I would like to thank the authors for submitting the manuscript entitled "Resistance Training Volume and Nutrient Intake on Lean Mass and Strength in Young Women." The topic is relevant and timely, and the study presents interesting hypotheses regarding the interaction between training volume and dietary intake. However, despite these

strengths, the manuscript has several important methodological limitations that, in my view, prevent its acceptance in its current form. I hope that my comments and suggestions may be helpful to the authors in improving future research efforts.

- In the methods section, it is described that the study was a non-randomized controlled trial involving healthy young women who had adhered to either a strict vegetarian or non-vegetarian diet for at least six months. While this design allowed for a controlled comparison between diet groups, the absence of random allocation introduces several methodological concerns that should be acknowledged. From my point of view, most notably, the lack of randomization increases the risk of selection bias, as participants were allocated to groups based on pre-existing dietary patterns. This compromises baseline comparability and opens the possibility that observed differences or lack there of may be influenced by unmeasured confounding factors, such as differences in health behaviors, nutritional status, or lifestyle characteristics that correlate with diet choice. Furthermore, from an epidemiological and statistical perspective, confounding is a major concern in non-randomized studies. Even when efforts are made to statistically adjust for known variables, residual confounding may persist, especially from factors that were not measured or inadequately captured. This limits the internal validity of the study and reduces the strength of causal inference. It is also important to consider that non-randomized trials generally require more complex statistical handling (e.g., matching techniques, stratification, or multivariate adjustments) to attempt to mitigate bias, approaches that may still fall short of the balance achieved through randomization. While the current study adopted a within-subject analysis to explore individual responsiveness, which may reduce some sources of between-group variability, the initial non-randomized design remains a limitation that should be carefully considered when interpreting the findings. In summary, again, from my point of view, although the study provides relevant insights into resistance training adaptations in different dietary groups, the lack of randomization limits the strength of the conclusions, particularly regarding causality and generalizability. These findings should be interpreted with caution and ideally confirmed in future randomized controlled trials.
- Regarding the intervention protocol, several methodological aspects warrant further consideration. Although the study provided a structured and supervised RT program, some elements may introduce bias or limit the interpretability of the findings. First, participants were instructed to refrain from consuming protein or amino acid supplements, engaging in additional training, and drinking alcohol on training days. However, it is not explicitly stated how adherence to these recommendations was monitored or verified. Without objective measures of compliance, these behaviors may introduce uncontrolled variability, potentially influencing training outcomes and weakening the internal validity of the study. Additionally, dietary intake was assessed using self-reported food records collected at multiple time points. While this is a commonly used method, it is subject to recall bias and underreporting, especially in nutritional interventions related to muscle hypertrophy and strength. The accuracy of dietary data is particularly relevant in this context, as differences in protein intake and overall diet quality between groups could confound the results. Another relevant point concerns the control of hormonal variation. Another point is that: although body composition assessments were scheduled to avoid the menstrual period, fluctuations throughout the menstrual cycle may still have affected variables such as strength performance or hydration status, introducing potential biological noise in outcome measurements. Furthermore, although a standardized 16-week RT protocol ensures consistency across participants, it may not fully account for individual variation in training responsiveness or recovery capacity, particularly between groups with distinct dietary backgrounds. A uniform protocol might overlook subtle differences in adaptation potential, which could influence the interpretation of group-level comparisons. Lastly, although training sessions were supervised by experienced instructors, the supervision ratio (up to five participants per instructor) may limit the ability to closely monitor exercise execution and ensure progression based on individual performance. This could affect the fidelity of the intervention and contribute to variability in training stimuli across participants. Taken together, these aspects highlight the importance of considering adherence, measurement precision, biological variability, and intervention fidelity when

interpreting the results. While the intervention was well structured and systematically described, these factors may influence the robustness and generalizability of the findings.

- From my point of view, I must strongly emphasize, using DXA to assess hypertrophy has some clear limitations. While it's a widely used method to estimate body composition, it doesn't measure muscle mass directly. DXA captures total lean mass, which includes other tissues like skin, connective tissue, and even fluid. So, changes in lean mass might not reflect true muscle growth. Also, small or localized muscle changes, like those expected after a few months of resistance training, can be hard to detect with DXA, especially when using regional analysis like the lower limb only. Factors like hydration, glycogen levels, and limb positioning during the scan can also affect the results. In short, DXA is not the most accurate method for measuring hypertrophy, and complementary tools like ultrasound or MRI would offer more specific insights into actual muscle growth.
- In my view, using an isokinetic dynamometer to assess strength is a solid approach, but there are still a few important points to consider. First, although participants were familiarized over multiple sessions, the testing protocol was quite complex, involving both concentric and eccentric contractions, at different speeds and modes. This increases the risk of learning effects or inconsistent effort, especially if participants were not used to this type of evaluation. Another point is that the strength outcome was expressed as a sum of different contraction modes (concentric extension, flexion, and eccentric flexion), which are physiologically distinct. Summing them into a single variable (PT SUM) may reduce specificity and make it harder to interpret which type of strength actually changed with training. Also, since strength was normalized by body mass, this could introduce some variability if participants had changes in weight unrelated to muscle function. Finally, even though isokinetic testing is objective, it doesn't fully reflect functional or sport-specific strength, which may limit the generalizability of the findings. Overall, it's a reliable method, but these factors should be kept in mind when interpreting the results.
- About training volume, I think the method used here is appropriate overall, but it still has some limitations. Calculating volume-load as sets × reps × load is a common and accepted approach. However, it mainly captures the external load, and doesn't account for internal factors like perceived effort, time under tension, or rest intervals, all of which can influence training adaptations. Also, the focus on lower-limb exercises only is justified by the study aim, but it's worth noting that global training load (e.g., including core or upper body work, if performed) can still impact recovery and overall adaptation.
- Normalizing volume by body mass is a good attempt to reduce interindividual variability, but it doesn't fully capture differences in muscle mass, neuromuscular efficiency, or training history, especially in a heterogeneous sample in terms of diet and possibly baseline strength. Regarding dietary intake, the use of 3-day food records across different time points adds value, and it's good that participants were trained and supervised throughout the process. Still, self-reported dietary data always come with limitations. Underreporting is a well-known issue, especially for total energy and protein intake, and even more so in populations with specific goals (like muscle gain). Also, although participants were instructed to report everything in detail, accuracy still depends heavily on their motivation, memory, and attention to detail. From my point of view, using both written and photographic records was a strength, but without biomarkers or controlled feeding conditions, we can't be entirely confident in the dietary data. This is important considering that diet is a major factor influencing hypertrophy and strength outcomes.
- The sample size calculation was based on muscle mass as the primary outcome, aiming to detect a moderate effect size with adequate power. However, the actual outcomes analyzed were hypertrophy and strength, which are related but distinct measures. This mismatch raises concerns about whether the study was sufficiently powered to detect meaningful differences in these specific outcomes. Additionally, no justification was provided for not recalculating the sample size based on strength or hypertrophy measures, which could have affected the validity of the findings. Ideally, sample size estimation should align directly with the primary outcomes to ensure robust and reliable results.
- From my point of view, the statistical methods used in this study are quite comprehensive and appropri-

ate for exploring the complex relationships between training volume, dietary intake, and adaptations like hypertrophy and strength. I appreciate the use of polynomial regression models to capture potential nonlinear interactions, which is often overlooked in similar research. The thorough checks for assumptions, such as normality, multicollinearity, and autocorrelation, add credibility to the analysis. However, I find the choice of paired samples t-test to analyze intervention effects on lean mass and peak torque somewhat limited, especially since the study design involves repeated measures with multiple variables. More advanced methods like mixed-effects models could have provided a more flexible framework to account for individual variability and potential confounders. Additionally, although the use of Spearman correlation for monotonic relationships is justified given physiological nonlinearities, there is no mention of adjustment for multiple comparisons, which might increase the risk of type I errors when testing many correlations. Overall, while the statistical approach is solid and detailed, a few alternative or supplementary methods could improve robustness and interpretability from my perspective.

- The study started with 71 participants, but a high dropout rate (26 individuals, or 37%) reduced the final sample to 45. This considerable attrition raises concerns about potential bias and the representativeness of the analyzed group. The baseline characteristics are presented only as overall means and standard deviations, but a detailed descriptive table is missing. This omission is significant, as it prevents a clear understanding of the sample composition, possible differences between subgroups, or factors related to dropout. Such information is crucial for interpreting the results and assessing external validity. Regarding the intervention effects, nearly half of the participants did not show muscle hypertrophy responsiveness, and 17% did not respond in strength gains. Macronutrient intake and training volume were reported, but the analysis showed limited significant correlations: only training volume correlated significantly with hypertrophy, and protein intake did not correlate with volume. Polynomial regression models identified interaction effects between protein intake and training volume on hypertrophy, and energy intake and training volume on strength, but these models explained less than 30% of the variance, indicating other factors are involved. The responsiveness threshold was determined using a small subsample (n=6), which yielded excellent reliability, but the small size limits the robustness of this estimate. Overall, the lack of detailed sample description and the high dropout limit confidence in the findings. More comprehensive reporting and larger samples would strengthen the study's conclusions.
- The discussion offers valuable insights into how training volume and diet interact to affect muscle adaptations, especially highlighting protein's role in hypertrophy and calories in strength gains. However, limitations in study design and methods reduce confidence in the findings. The non-randomized design raises concerns about confounding and selection bias that affect causal interpretation. Using DXA to measure hypertrophy and summing distinct strength measures limit the precision of outcomes. Self-reported dietary data may be inaccurate despite supervision. The dropout rate was high, and lack of detailed baseline data weakens representativeness. While the statistical approach is appropriate, some choices could be improved for robustness. Overall, the study contributes useful observations but its conclusions should be viewed cautiously given these methodological constraints.
- The conclusion appropriately summarizes the main findings about training volume and nutrition's role in muscle adaptations. However, given the study's methodological limitations, especially the non-randomized design, measurement constraints, and high dropout, these conclusions should be interpreted with caution. The observed relationships are interesting but require confirmation in more rigorous trials before being generalized.

Final Decision

- Rejection