



Article

Vitamin D Inadequacy and Its Relation to Body Fat and Muscle Mass in Adult Women of Childbearing Age

Paula Moreira Magalhães ^{1,2,*}, Sabrina Pereira da Cruz ², Orion Araújo Carneiro ², Michelle Teixeira Teixeira ³
and Andréa Ramalho ^{2,4}

¹ Postgraduate Program of Clinical Medicine, Faculty of Medicine, Federal University of Rio de Janeiro (UFRJ), Rio de Janeiro 21044-020, Brazil

² Center for Research on Micronutrients (NPqM), Institute of Nutrition Josué de Castro, Federal University of Rio de Janeiro (UFRJ), Rio de Janeiro 21941-902, Brazil; sabrina.cruz.ufrj@gmail.com (S.P.d.C.); nutricionistaorion@uol.com.br (O.A.C.); aramalho.rj@gmail.com (A.R.)

³ Department of Public Health Nutrition, Nutrition School, Federal University of the State of Rio de Janeiro (UNIRIO), Rio de Janeiro 22290-250, Brazil; michelle.teixeira@unirio.br

⁴ Department of Social and Applied Nutrition, Institute of Nutrition Josué de Castro, Federal University of Rio de Janeiro (UFRJ), Rio de Janeiro 21941-902, Brazil

* Correspondence: paulammaga@yahoo.com

Abstract: To assess the correlation between vitamin D status and body composition variables in adult women of childbearing age, a cross-sectional study was conducted involving women aged 20–49 years. The participants were categorized based on their vitamin D status and further divided according to body mass index (BMI). Anthropometric and biochemical data were collected to compute body composition indices, specifically body fat and muscle mass. The sample included 124 women, with 63.70% exhibiting vitamin D inadequacy. Women with inadequate vitamin D status demonstrated a higher waist-to-height ratio (WHtR) and body adiposity index (BAI), along with a lower BMI-adjusted muscle mass index (SMI_{BMI}), compared to those with adequate levels of vitamin D ($p = 0.021$; $p = 0.019$; and $p = 0.039$, respectively). A positive correlation was observed between circulating concentrations of 25(OH)D and SMI_{BMI}, while a negative correlation existed between circulating concentrations of 25(OH)D and waist circumference (WC), WHtR, conicity index (CI), fat mass index (FMI), body fat percentage (% BF), and fat-to-muscle ratio (FMR). These findings suggest that inadequate vitamin D status may impact muscle tissue and contribute to higher body adiposity, including visceral adiposity. It is recommended that these variables be incorporated into clinical practice, with a particular emphasis on WHtR and SMI_{BMI}, to mitigate potential metabolic consequences associated with vitamin D inadequacy.

Keywords: vitamin D; body composition; women of childbearing age; body fat; muscle mass



Citation: Magalhães, P.M.; Cruz, S.P.d.; Carneiro, O.A.; Teixeira, M.T.; Ramalho, A. Vitamin D Inadequacy and Its Relation to Body Fat and Muscle Mass in Adult Women of Childbearing Age. *Nutrients* **2024**, *16*, 1267. <https://doi.org/10.3390/nu16091267>

Academic Editor: Michael F. Holick

Received: 28 February 2024

Revised: 3 April 2024

Accepted: 6 April 2024

Published: 25 April 2024



Copyright: © 2024 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

1. Introduction

The prevalence of vitamin D deficiency (VDD) in women of childbearing age is high [1–6], especially in those with obesity (88%) compared to those with a normal weight (51%) [7]. Evaluating the nutritional status of this vitamin is important not only for promoting bone, reproductive, immune, and mental health but also for understanding the influence of vitamin D on different tissues, such as adipose tissue and skeletal muscle, which have specific receptors for this vitamin [8–11].

In recent years, there has been a growing interest in understanding the relationship between vitamin D and body composition, as evidenced by the association between VDD and obesity [12]. However, most studies rely on conventional measures such as body mass index (BMI), waist circumference (WC), and body fat percentage (% BF) to assess body composition [13–18]. These studies often overlook other important variables related to the distribution of body fat.

Similarly, there is evidence of the association of vitamin D with skeletal muscle mass (SMM), where VDD can contribute to the reduction of muscle mass as well as decreased strength and muscle function, leading to sarcopenia [19]. Understanding these aspects is essential for a comprehensive assessment of the influence of vitamin D on body composition and muscle health in women of childbearing age.

Considering the crucial role of this vitamin in muscle mass during pregnancy and postpartum, influencing functional capacity and quality of life, this relationship promotes functional autonomy and reduces the risk of complications associated with muscle health throughout life. However, no studies related to the behavior of these variables in women of childbearing age have been found [20,21].

Considering the limited number of studies addressing the relationship between vitamin D status and body composition variables among adult women of childbearing age, especially in the context of body fat distribution and muscle mass, the present study aims to assess this association. These variables enable the measurement of both body fat and muscle mass in women within this age range.

2. Materials and Methods

This is a cross-sectional study with adult women of childbearing age assisted by a nutrition service located in the city of Rio de Janeiro, Brazil. Data collection occurred between August 2018 and August 2019. The participants were required to undergo clinical-nutritional anamnesis along with anthropometric and biochemical evaluation during the study period.

The sample size was calculated based on a prevalence study of VDD and its associated factors in women of childbearing age in Brazil [22]. To achieve a sample size with a 95% confidence interval, considering a prevalence of inadequacy of 72% with a margin of error of 5%, 86 women of childbearing age would be required in order to conduct the present study.

Women aged 20–49 years, of a normal weight and overweight (overweight and obesity) according to BMI and who provided information on the research variables, were included in the study. The exclusion criteria were pregnant women, nursing mothers, menopausal women, women who had undergone bariatric surgery, and those who had received vitamin D supplementation in the last 6 months.

Circulating concentrations of 25-hydroxyvitamin D (25(OH)D) were determined via high-performance liquid chromatography with ultraviolet detector (HPLC-UV) [23]; the reference values of Holick et al. (2011) [24] were adopted, and were evaluated according to circulating concentrations of 25(OH)D as sufficient (25(OH)D \geq 30 ng/mL), insufficient (25(OH)D \geq 20 and $<$ 30 ng/mL), and deficient (25(OH)D $<$ 20 ng/mL). In the current study, circulating concentrations of 25(OH)D \geq 30 ng/mL were considered adequate and circulating concentrations of 25(OH)D $<$ 30 ng/mL inadequate. In addition, triglycerides and high-density lipoprotein (HDL-cholesterol) serum concentrations were determined using a colorimetric enzymatic method, and their values were used to calculate one of the body variables.

Anthropometric evaluation consisted of measuring body weight in kilograms using an InBody270 electronic anthropometric scale [25]; height in meters using an Avanutri[®] (Avanutri, Três Rios, Brazil) stadiometer [25]; WC, hip circumference (HC) and arm circumference (AC) in centimeters measured with a Sanny[®] (Sanny, São Bernardo do Campo, Brazil) tape measure [25–27]; and triceps skinfold (TSF) in millimeters measured with a Lange[®] (US Chemical, Greenville, SC, USA) adipometer [25].

With these anthropometric data, the arm muscle circumference (AMC) [28] and the arm muscle area (AMA) [29] were calculated to evaluate muscle mass and body fat, BMI [26], waist-to-hip ratio (WHR) [30], conicity index (CI) [31], waist-to-height ratio (WHtR) [32], visceral adiposity index (VAI) [33], body adiposity index (BAI) [34], a body shape index (ABSI) [35], Clinical Universidad Navarre—body adiposity estimator (CUN-BAE) [36], and Belarmino-Waitzberg index (BeW) [37], according to their published formulas. BMI

was assessed according to the (2000) cut-off points for normal weight ($BMI \geq 18.5$ and $\leq 24.9 \text{ kg/m}^2$) and overweight ($BMI \geq 25.0 \text{ kg/m}^2$) recommended by WHO.

In addition, electric bioimpedance testing was performed with an InBody270[®] tetrapolar scale. Participants were informed about the pre-examination procedures to reduce measurement errors, obtaining fat mass (FM), fat-free mass (FFM), skeletal muscle mass (SMM) and % BF. With these data, the fat mass index (FMI) [38] and the fat-to-muscle ratio (FMR) [39] were obtained for body fat evaluation. For muscle mass evaluation, the fat-free mass index (FFMI) [38] and the muscle mass indices adjusted by height² (SMI_{height}) [40]; body weight (SMI_{weight}) [41] and BMI (SMI_{BMI}) [42] were considered according to their published formulas.

In Tables 1 and 2 are shown the overall indices and their formulas used to estimate body fat and muscle mass, respectively.

Table 1. Indices and formulas for estimating body fat.

Index	Formula	Reference
Body mass index (BMI)	$BMI \text{ (kg/m}^2\text{)} = \frac{\text{weight (kg)}}{\text{height (m)}^2}$	WHO, 2000 [26]
Waist-to-hip ratio (WHR)	$WHR = \frac{WC \text{ (cm)}}{HC \text{ (cm)}}$	WHO, 2008 [30]
Waist-to-height ratio (WHtR)	$WHtR = \frac{WC \text{ (cm)}}{\text{height (cm)}}$	Ashwell; Lejeune; Mcpherson, 1996 [32]
Conicity index (CI)	$CI = \frac{WC \text{ (m)}}{0.109 \times \sqrt{\text{weight (kg)/height (m)}}$	Valdez, 1991 [31]
Body adiposity index (BAI)	$BAI \text{ (\%)} = \frac{HC \text{ (cm)}}{\text{height (m)}^{1.5}} - 18$	Bergman et al., 2011 [34]
A body shape index (ABSI)	$ABSI \text{ (m}^{11/6} \text{ kg}^{-2/3}\text{)} = \frac{WC \text{ (m)}}{BMI \text{ (kg/m}^2\text{)}^{2/3} \times \text{height (m)}^{1/2}}$	Krakauer; Krakauer, 2012 [35]
Clínica Universidad de Navarra—Body Adiposity Estimator (CUN-BAE)	$CUN-BAE \text{ (\%)} = -44.988 + (0.503 \times \text{age (years)}) + (10.689 \times \text{sex}) + (3172 \times BMI \text{ (kg/m}^2\text{)}) - (0.026 \times BMI \text{ (kg/m}^2\text{)}^2) + (0.181 \times BMI \text{ (kg/m}^2\text{)} \times \text{sex}) - (0.02 \times BMI \text{ (kg/m}^2\text{)} \times \text{age (years)}) - (0.005 \times BMI \text{ (kg/m}^2\text{)}^2 \times \text{sex}) + (0.00021 \times BMI \text{ (kg/m}^2\text{)}^2 \times \text{age (years)})$ Sex: female = 1	Gómez-Ambrosi et al., 2012 [36]
Belarmino–Watzberg index (BeW)	$Woman \text{ BeW (\%)} = -48.8 + 0.087 \times WC \text{ (cm)} + 1.147 \times HC \text{ (cm)} - 0.003 \times HC \text{ (cm)}^2$	Belarmino et al., 2018 [37]
Visceral adiposity index (VAI)	$VAI \text{ (women)} = \left(\frac{WC \text{ (cm)}}{39.58 + (1.89 \times IMC \text{ (kg/m}^2\text{)})} \right) \times \left(\frac{TG \text{ (mmol/L)}}{0.81} \right) \times \left(\frac{1.52}{HDL \text{ (mmol/l)}} \right)$	Amato et al., 2010 [33]
Fat mass index (FMI)	$FMI \text{ (kg/m}^2\text{)} = \frac{FM \text{ (kg)}}{\text{height (m)}^2}$	Schutz; Kyle; Pichard, 2002 [38]
Fat-to-muscle ratio (FMR)	$FMR = \frac{FM \text{ (kg)}}{MM \text{ (kg)}}$	Xu et al., 2018 [39]

Table 2. Indices and formulas for estimating muscle mass.

Index	Formula	Reference
Arm muscle circumference (AMC)	$AMC \text{ (cm)} = AC \text{ (cm)} - (DCT \text{ (mm)} \times 0.3142)$	Frisancho, 1974 [28]
Arm muscle area (AMA)	$AMA \text{ (cm}^2\text{)} = \frac{(AC \text{ (cm)} - [DCT \text{ (mm)} \times 0.3142])^2}{12.57}$	Gurney; Jelliffe, 1973 [29]
Fat-free mass index (FFMI)	$FFMI \text{ (kg/m}^2\text{)} = \frac{FFM \text{ (kg)}}{\text{height (m)}^2}$	Schutz; Kyle; Pichard, 2002 [38]
Muscle mass index adjusted by height ² (SMI_{height})	$SMI_{\text{height}} \text{ (kg/m}^2\text{)} = \frac{SMM \text{ (kg)}}{\text{height (m)}^2}$	Baumgartner et al., 1998 [40]
Muscle mass index adjusted by weight (SMI_{weight})	$SMI_{\text{weight}} = \frac{SMM \text{ (kg)}}{\text{weight (kg)}} \times 100$	Janssen; Heymsfield; Ross, 2002 [41]
Muscle mass index adjusted by BMI (SMI_{BMI})	$SMI_{\text{BMI}} = \frac{SMM \text{ (kg)}}{BMI \text{ (kg/m}^2\text{)}}$	Cawthon et al., 2014 [42]

The study was approved by the Research Ethics Committee of the Federal University of the State of Rio de Janeiro (UNIRIO), and the protocol was registered in the National Research Ethics System (CAEE 50063715.5.0000.5285).

The women were divided, as illustrated in Figure 1, according to circulating concentrations of 25(OH)D into sufficient, insufficient, and deficient, then into adequate and inadequate, vitamin D. The groups of women with adequate and inadequate vitamin D were then divided, according to BMI, into normal weight or overweight.

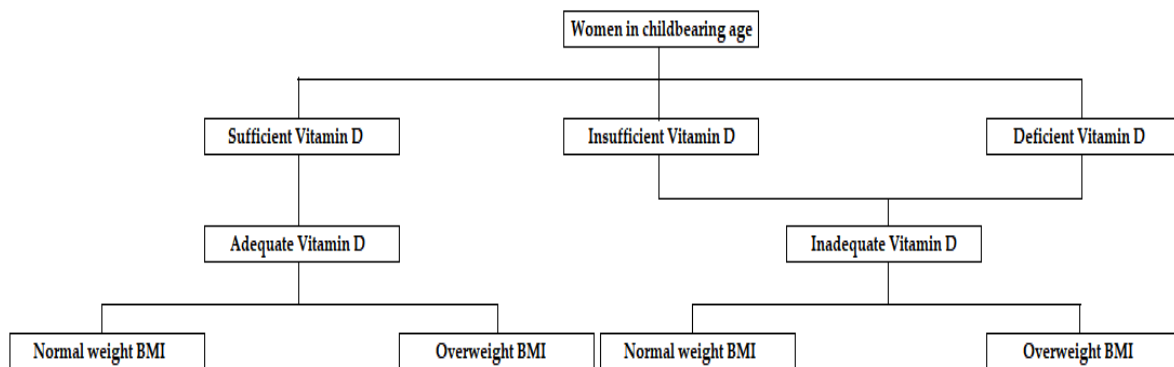


Figure 1. Division of women in the study. Legend: Women were divided according to circulating concentrations of 25(OH)D into sufficient ($25(\text{OH})\text{D} \geq 30 \text{ ng/mL}$), insufficient ($25(\text{OH})\text{D} \geq 20$ and $< 30 \text{ ng/mL}$) and deficient ($25(\text{OH})\text{D} < 20 \text{ ng/mL}$) vitamin D, and into adequate ($25(\text{OH})\text{D} \geq 30 \text{ ng/mL}$) and inadequate ($25(\text{OH})\text{D} < 30 \text{ ng/mL}$) vitamin D. Women with adequate and inadequate vitamin D were divided into normal weight ($\text{BMI} \geq 18.5$ and $\leq 24.9 \text{ kg/m}^2$) and overweight ($\text{BMI} \geq 25 \text{ kg/m}^2$) according to BMI.

Statistical tests were performed in the statistical package SPSS for windows version 21.0. The Kolmogorov–Smirnov test was performed to evaluate the normality of the sample. Measures of central tendency and dispersion (mean and standard deviation) were calculated for quantitative variables, and the Mann–Whitney and Kruskal–Wallis tests were used for comparisons. In comparisons of 2 groups, the Mann–Whitney test was applied, and in comparisons of 3 groups or more, the Kruskal–Wallis test was applied. In the case of comparisons of 3 groups, after the Kruskal–Wallis test, the Mann–Whitney test and Bonferroni correction was applied for separate post-test. The analysis of correlations between serum 25(OH)D concentrations and the analyzed variables was estimated using Spearman’s correlation coefficient. For all statistical tests, a probability of less than 5% ($p < 0.05$) was considered as the statistical significance level.

3. Results

3.1. General Characterization of the Sample

A total of 124 women with a mean age of 34.07 ± 7.13 years participated in the study. The mean BMI was $24.20 \pm 3.48 \text{ kg/m}^2$, with 66.93% classified as having a normal BMI, 23.39% as overweight, and 9.68% as obese. The mean of circulating concentrations of 25(OH)D was $28.83 \pm 10.35 \text{ ng/mL}$. Of the participants, 36.30% had vitamin D adequacy, while 63.70% had vitamin D inadequacy, including 44.35% with insufficiency and 19.35% with deficiency.

3.2. Body Composition According to Vitamin D Serum Concentrations

The means of 25(OH)D and the variables analyzed based on vitamin D status are presented in Table 3. Upon examining these results, we observed significantly lower BAI values in women with vitamin D sufficiency.

Table 3. General characteristics of the sample comprising 124 adult women of childbearing age, classified in accordance with Vitamin D status into sufficient, insufficient, deficient, adequate, and inadequate (mean ± sd).

	Vitamin D (25(OH)D)			<i>p</i> ^a	Vitamin D (25(OH)D)		<i>p</i>
	Sufficient (n = 45)	Insufficient (n = 55)	Deficient (n = 24)		Adequate (n = 45)	Inadequate (n = 79)	
25(OH)D (ng/mL)	40.07 ± 8.24 ^{b,c}	24.56 ± 2.68 ^{b,d}	17.55 ± 1.60 ^{c,d}	<0.001	40.07 ± 8.24	22.43 ± 4.03	<0.001
Age (years)	33.78 ± 7.54	33.33 ± 6.50	36.33 ± 7.55	0.200	33.78 ± 7.54	34.24 ± 6.93	0.660
Weight (kg)	63.07 ± 7.87	66.62 ± 13.47	65.36 ± 9.87	0.496	63.07 ± 7.87	66.24 ± 12.44	0.239
WC (cm)	78.83 ± 7.02	81.89 ± 10.49	82.46 ± 8.88	0.249	78.83 ± 7.02	82.06 ± 9.97	0.114
HC (cm)	99.63 ± 6.14	102.67 ± 9.45	101.52 ± 8.08	0.382	99.63 ± 6.14	102.31 ± 9.01	0.181
AC (cm)	27.57 ± 2.43	27.32 ± 3.05	27.98 ± 2.67	0.607	27.56 ± 2.43	27.54 ± 2.93	0.900
TSF (mm)	26.75 ± 6.71	27.45 ± 7.67	27.68 ± 6.99	0.918	26.75 ± 6.71	27.53 ± 7.39	0.680
AMC (cm)	19.16 ± 1.22	18.70 ± 1.80	19.29 ± 1.48	0.166	19.16 ± 1.22	18.89 ± 1.71	0.256
AMA (cm ²)	29.34 ± 3.78	28.09 ± 5.54	29.78 ± 4.62	0.166	29.34 ± 3.78	28.65 ± 5.28	0.256
BMI (kg/m ²)	23.45 ± 2.85	24.80 ± 4.02	24.24 ± 3.03	0.284	23.44 ± 2.85	24.63 ± 3.73	0.115
WHR	0.79 ± 0.05	0.80 ± 0.06	0.81 ± 0.06	0.307	0.79 ± 0.05	0.80 ± 0.06	0.302
CI	1.17 ± 0.06	1.18 ± 0.07	1.20 ± 0.08	0.144	1.17 ± 0.058	1.19 ± 0.073	0.116
WHtR	0.48 ± 0.05 ^{b,c}	0.50 ± 0.06 ^b	0.50 ± 0.05 ^c	0.062	0.48 ± 0.05	0.50 ± 0.05	0.021
VAI	1.26 ± 0.88	1.00 ± 0.46	1.50 ± 1.12	0.291	1.26 ± 0.88	1.14 ± 0.74	0.688
BAI (%)	29.49 ± 3.44 ^b	31.24 ± 3.86 ^b	30.26 ± 3.60	0.034	29.49 ± 3.44	30.97 ± 3.78	0.019
ABSI (m ^{11/6} kg ^{-2/3})	0.12 ± 0.01	0.12 ± 0.01	0.13 ± 0.01	0.434	0.12 ± 0.01	0.12 ± 0.01	0.514
CUN-BAE (%)	32.12 ± 4.36	33.95 ± 5.79	33.68 ± 4.68	0.192	32.12 ± 4.36	33.87 ± 5.45	0.074
BeW (%)	42.45 ± 3.80	44.21 ± 5.72	43.71 ± 4.88	0.378	42.45 ± 3.80	44.06 ± 5.45	0.166
BF (kg)	19.51 ± 6.13	22.31 ± 9.36	22.21 ± 6.59	0.336	19.51 ± 6.13	22.29 ± 8.58	0.202
FFM (kg)	42.98 ± 4.31	43.12 ± 6.07	42.66 ± 4.73	0.984	42.98 ± 4.31	42.99 ± 5.67	0.958
SMM (kg)	23.45 ± 2.55	23.30 ± 4.14	23.16 ± 2.77	0.974	23.45 ± 2.55	23.25 ± 3.77	0.890
% BF (%)	30.73 ± 6.37	33.00 ± 7.53	33.69 ± 6.51	0.203	30.73 ± 6.37	33.20 ± 7.20	0.094
FMI (kg/m ²)	7.27 ± 2.34	8.33 ± 3.24	8.21 ± 2.45	0.270	7.27 ± 2.34	8.39 ± 3.01	0.132
FFMI (kg/m ²)	15.98 ± 1.25	16.17 ± 1.47	15.75 ± 1.16	0.511	15.98 ± 1.25	16.04 ± 1.39	0.692
FMR	0.84 ± 0.26	0.95 ± 0.32	0.96 ± 0.28	0.166	0.84 ± 0.26	0.95 ± 0.30	0.065
SMI _{height} (kg/m ²)	8.72 ± 0.76	8.73 ± 1.20	8.55 ± 0.71	0.522	8.72 ± 0.76	8.68 ± 1.08	0.846
SMI _{weight} (%)	37.78 ± 3.56	36.02 ± 4.69	35.97 ± 3.69	0.141	37.78 ± 3.56	36.01 ± 4.39	0.051
SMI _{BMI}	1.02 ± 0.13 ^b	0.96 ± 0.15 ^b	0.97 ± 0.12	0.106	1.02 ± 0.13	0.96 ± 0.14	0.039

Sufficient vitamin D (25(OH)D ≥ 30 ng/mL); Insufficient vitamin D (25(OH)D ≥ 20 and <30 ng/mL); Deficient vitamin D (25(OH)D < 20 ng/mL); adequate vitamin D (25(OH)D ≥ 30 ng/mL); inadequate vitamin D (25(OH)D < 30 ng/mL). WC: Waist circumference; HC: Hip circumference; AC: Arm circumference; TSF: Triceps skinfold; AMC: Arm muscle circumference; AMA: Arm muscle area; BMI: Body mass index; WHR: Waist-to-hip ratio; CI: Conicity index; WHtR: Waist-to-height ratio; VAI: Visceral adiposity index; BAI: Body adiposity index; ABSI: A body shape index; CUN-BAE: Clínica Universidad de Navarra—Body adiposity estimator; BeW: Belarmino–Waitzberg index; FM: Fat mass; FFM: Fat-free mass; SMM: Skeletal muscle mass; % BF: Body fat percentage; FMI: Fat mass index; FFMI: Fat-free mass index; FMR: Fat–muscle ratio; SMI_{height}: Height-adjusted muscle mass index²; SMI_{weight}: Weight-adjusted muscle mass index; SMI_{BMI}: BMI-adjusted muscle mass index. ^a Comparisons among the three groups analyzed by the Kruskal–Wallis test. ^b Separate post-test (Mann–Whitney) showed (*p* < 0.05) between sufficient and insufficient. ^c Separate post-test (Mann–Whitney) showed (*p* < 0.05) between sufficient and deficient. ^d Separate post-test (Mann–Whitney) showed (*p* < 0.05) between insufficient and deficient.

In the assessment of women with vitamin D inadequacy, significantly higher WHtR and BAI values were found compared to those with adequacy. Additionally, significantly higher SMI_{BMI} values were observed in women with inadequacy compared to those with inadequacy. Therefore, participants with some degree of impairment in vitamin D status exhibited higher values in body fat variables, including visceral fat, and lower values in muscle mass, in comparison to those with adequate vitamin D status.

3.3. Body Composition According to Vitamin D Serum Concentrations by BMI

When dividing participants with vitamin D adequacy by BMI, it was observed that 28.23% (n = 35) were of normal weight and 8.06% (n = 10) were overweight. Regarding vitamin D inadequacy, 38.71% (n = 48) were of normal weight and 25% (n = 31) were overweight.

Concerning the mean body composition variables, significant differences were found in weight, WC, HC, AC, TSF, AMC, AMA, BMI, WHR, WHtR, BeW, BAI, CI, FMI, CUN-BAE, SMM, FM, FFM, % BF, FMR, SMI_{height}, SMI_{weight}, and SMI_{BMI} among all women assessed (Table 4). It is noteworthy that the WHtR was significantly higher in women of normal weight and vitamin D inadequacy when compared to those of normal weight and vitamin D adequacy. Women who were overweight with vitamin D inadequacy had significantly higher mean values of weight, HC, and BeW than those who were overweight with vitamin D adequacy.

Table 4. General characteristics of the sample comprising 124 adult women of childbearing age, classified in accordance with their Vitamin D status as adequate or inadequate and BMI (mean ± sd).

	Adequate Vitamin D		Inadequate Vitamin D		p *
	BMI Normal Weight (n = 35)	BMI Overweight (n = 10)	BMI Normal Weight (n = 48)	BMI Overweight (n = 31)	
25(OH)D (ng/mL)	40.89 ± 8.12 ^{c,e}	37.17 ± 8.41 ^{d,f}	22.66 ± 4.16 ^{c,f}	22.08 ± 3.87 ^{d,e}	<0.001
Age (years)	33.14 ± 7.52	36.00 ± 7.59	34.63 ± 6.62	33.65 ± 7.45	0.619
Weight (kg)	60.79 ± 6.52 ^{a,e}	71.03 ± 7.18 ^{a,d,f}	58.60 ± 7.27 ^{b,f}	78.07 ± 9.01 ^{b,d,e}	<0.001
WC (cm)	76.03 ± 4.96 ^{a,e}	88.65 ± 3.16 ^{a,f}	76.33 ± 6.20 ^{b,f}	90.94 ± 8.03 ^{b,e}	<0.001
HC (cm)	97.77 ± 5.20 ^{a,e}	106.15 ± 4.66 ^{a,d,f}	96.59 ± 5.24 ^{b,f}	111.00 ± 6.08 ^{b,d,e}	<0.001
AC (cm)	26.79 ± 1.92 ^{a,e}	30.56 ± 1.83 ^{a,f}	26.23 ± 2.17 ^{b,f}	30.76 ± 1.86 ^{b,e}	<0.001
TSF (mm)	25.34 ± 6.37 ^{a,e}	32.22 ± 5.24 ^{a,f}	24.62 ± 6.02 ^{b,f}	34.74 ± 5.27 ^{b,e}	<0.001
AMC (cm)	18.83 ± 1.01 ^{a,e}	20.44 ± 1.18 ^{a,f}	18.50 ± 1.62 ^{b,f}	19.86 ± 1.59 ^{b,e}	<0.001
AMA (cm ²)	28.30 ± 3.02 ^{a,e}	33.35 ± 3.88 ^{a,f}	27.47 ± 4.95 ^{b,f}	31.58 ± 5.03 ^{b,e}	<0.001
BMI (kg/m ²)	22.20 ± 1.37 ^{a,e}	27.80 ± 2.36 ^{a,f}	22.15 ± 1.59 ^{b,f}	28.46 ± 2.70 ^{b,e}	<0.001
WHR	0.78 ± 0.05 ^{a,e}	0.84 ± 0.04 ^{a,f}	0.79 ± 0.05 ^b	0.82 ± 0.06 ^{b,e}	0.001
CI	1.15 ± 0.05 ^{a,e}	1.22 ± 0.05 ^{a,f}	1.17 ± 0.06 ^{b,f}	1.22 ± 0.08 ^{b,e}	<0.001
WHtR	0.46 ± 0.03 ^{a,c,e}	0.56 ± 0.03 ^{a,f}	0.47 ± 0.03 ^{b,c,f}	0.55 ± 0.05 ^{b,e}	<0.001
VAI	1.08 ± 0.55	1.83 ± 1.41	1.02 ± 0.61	1.30 ± 0.86	0.142
BAI (%)	28.04 ± 1.95 ^{a,e}	34.58 ± 2.55 ^{a,f}	28.86 ± 2.44 ^{b,f}	34.17 ± 3.17 ^{b,e}	<0.001
ABSI (m ^{11/6} kg ^{-2/3})	0.12 ± 0.01	0.12 ± 0.01	0.12 ± 0.01	0.13 ± 0.01	0.530
CUN-BAE (%)	30.23 ± 2.27 ^{a,e}	38.75 ± 3.30 ^{a,f}	30.33 ± 2.93 ^{b,f}	39.34 ± 3.59 ^{b,e}	<0.001
BeW (%)	41.20 ± 3.15 ^{a,e}	46.80 ± 2.44 ^{a,d,f}	40.56 ± 3.34 ^{b,f}	49.36 ± 3.29 ^{b,d,e}	<0.001
FM (kg)	1768 ± 4.41 ^{a,e}	27.10 ± 6.73 ^{a,f}	17.33 ± 4.23 ^{b,f}	31.72 ± 6.59 ^{b,e}	<0.001
AMC (kg)	42.74 ± 4.55 ^e	43.96 ± 3.18	41.33 ± 5.37 ^b	46.15 ± 4.92 ^{b,e}	0.005
SMM (kg)	23.28 ± 2.68 ^e	24.16 ± 1.91	22.14 ± 3.69 ^b	25.38 ± 2.96 ^{b,e}	0.003
% BF (%)	29.02 ± 5.39 ^{a,e}	37.77 ± 5.40 ^{a,f}	29.39 ± 5.18 ^{b,f}	40.47 ± 4.32 ^{b,e}	<0.001
FMI (kg/m ²)	6.48 ± 1.49 ^{a,e}	10.55 ± 2.44 ^{a,f}	6.53 ± 1.40 ^{b,f}	11.66 ± 2.28 ^{b,e}	<0.001
FFMI (kg/m ²)	15.70 ± 1.12 ^{a,e}	17.15 ± 1.15 ^{a,f}	15.58 ± 1.25 ^{b,f}	16.94 ± 1.21 ^{b,e}	<0.001
FMR	0.77 ± 0.21 ^{a,e}	1.12 ± 0.25 ^{a,f}	0.80 ± 0.20 ^{b,f}	1.25 ± 0.23 ^{b,e}	<0.001
SMI _{height} (kg/m ²)	8.55 ± 0.68 ^{a,e}	9.42 ± 0.72 ^{a,f}	8.35 ± 1.08 ^{b,f}	9.31 ± 0.77 ^{b,e}	<0.001
SMI _{weight} (%)	38.64 ± 3.13 ^{a,e}	34.19 ± 3.06 ^{a,f}	37.74 ± 4.22 ^{b,f}	32.71 ± 2.40 ^{b,e}	<0.001
SMI _{BMI}	1.05 ± 0.11 ^{a,e}	0.88 ± 0.08 ^{a,f}	1.00 ± 0.15 ^{b,f}	0.89 ± 0.09 ^{b,e}	<0.001

Adequate vitamin D (25(OH)D ≥ 30 ng/mL); Inadequate vitamin D (25(OH)D < 30 ng/mL); Normal weight BMI (BMI ≥ 18.5 and ≤ 24.9 kg/m²); Overweight BMI (BMI ≥ 25.0 kg/m²). 25(OH)D: 25 hydroxy vitamin D; WC: Waist circumference; HC: Hip circumference; AC: Arm circumference; TSF: Triceps skinfold; AMC: Arm muscle circumference; AMA: Arm muscle area; BMI: Body mass index; WHR: Waist-to-hip ratio; CI: Conicity index; WHtR: Waist-to-height ratio; VAI: Visceral adiposity index; BAI: Body adiposity index; ABSI: Body shape

index; CUN-BAE: Clínica Universidad de Navarra—Body adiposity estimator; BeW: Belarmino–Waitzberg index; FM: Fat mass; FFM: Fat-free mass; SMM: Skeletal muscle mass; BF %: Percent Body fat percent; FMI: Fat mass index; FFMI: Fat-free mass index; FMR: Fat–muscle ratio; SMI_{height}: Height-adjusted muscle mass index²; SMI_{weight}: Weight-adjusted muscle mass index; SMI_{BMI}: BMI-adjusted muscle mass index. * Comparisons between the four groups analyzed by the Kruskal–Wallis test. ^a Separate post-test (Mann–Whitney) showed ($p < 0.05$) between adequate Vitamin D with normal-weight BMI and adequate Vitamin D with overweight BMI. ^b Separate post-test (Mann–Whitney) showed ($p < 0.05$) between inadequate Vitamin D with normal-weight BMI and inadequate Vitamin D with overweight BMI. ^c Separate post-test (Mann–Whitney) showed ($p < 0.05$) between adequate Vitamin D with normal-weight BMI and inadequate Vitamin D with normal-weight BMI. ^d Separate post-test (Mann–Whitney) showed ($p < 0.05$) between adequate Vitamin D with overweight BMI and inadequate Vitamin D with overweight BMI. ^e Separate post-test (Mann–Whitney) showed ($p < 0.05$) between adequate Vitamin D with normal-weight BMI and inadequate Vitamin D with overweight BMI. ^f Separate Post-test (Mann–Whitney) showed ($p < 0.05$) between adequate Vitamin D with overweight BMI and inadequate Vitamin D with normal-weight BMI.

3.4. Correlations of Vitamin D with Body Composition

There was a negative correlation between circulating concentrations of 25(OH)D and WC ($r = -0.194, p = 0.031$), WHtR ($r = -0.218, p = 0.015$), CI ($r = -0.207, p = 0.021$), FMI ($r = -0.210, p = 0.039$), % BF ($r = -0.214, p = 0.035$), FMR ($r = -0.226, p = 0.026$), and a positive correlation with SMI_{BMI} ($r = 0.219, p = 0.031$) (Table 5).

Table 5. Correlations between circulating concentrations of 25(OH)D, age, and body variables of 124 adult women of childbearing age.

	Correlation (r)	p
Age (years)	−0.068	0.451
Weight (kg)	−0.111	0.221
WC (cm)	−0.194	0.031
HC (cm)	−0.123	0.174
AC (cm)	−0.073	0.451
TSF (mm)	−0.132	0.170
AMC (cm)	0.063	0.514
AMA (cm ²)	0.050	0.605
BMI (kg/m ²)	−0.151	0.094
WHR	−0.175	0.053
CI	−0.207	0.021
WHtR	−0.218	0.015
VAI	−0.024	0.802
BAI (%)	−0.172	0.057
ABSI (m ^{11/6} kg ^{−2/3})	−0.093	0.304
CUN-BAE (%)	−0.161	0.073
BeW (%)	−0.132	0.145
FM (kg)	−0.189	0.063
FFM (kg)	0.050	0.627
SMM (kg)	−0.044	0.666
% BF (%)	−0.214	0.035
FMI (kg/m ²)	−0.210	0.039
FFMI (kg/m ²)	−0.008	0.940
FMR	−0.226	0.026
SMI _{height} (kg/m ²)	−0.003	0.980
SMI _{weight} (%)	0.199	0.051
SMI _{BMI}	0.219	0.031

WC: Waist circumference; HC: Hip circumference; AC: Arm circumference; TSF: Triceps skinfold; AMC: Arm muscle circumference; AMA: Arm muscle area; BMI: Body mass index; WHR: Waist-to-hip ratio; CI: Conicity index; WHtR: Waist-to-height ratio; VAI: Visceral adiposity index; BAI: Body adiposity index; ABSI: Body shape index; CUN-BAE: Clínica Universidad de Navarra—Body adiposity estimator; BeW: Belarmino–Waitzberg index; FM: Fat mass; FFM: Fat-free mass; SMM: Skeletal muscle mass; % BF: Percent body fat; FMI: Fat mass index; FFMI: Fat-free mass index; FMR: Fat–muscle ratio; SMI_{height}: Height-adjusted muscle mass index²; SMI_{weight}: Weight-adjusted muscle mass index; SMI_{BMI}: BMI-adjusted muscle mass index.

When only women with vitamin D inadequacy were analyzed, a negative correlation was found between circulating concentrations of 25(OH)D and VAI ($r = -0.359$, $p = 0.003$). This correlation was higher when only those with deficiency were analyzed ($r = -0.520$, $p = 0.022$).

Furthermore, women with insufficiency showed a negative correlation between circulating concentrations of 25(OH)D and % BF ($r = -0.336$, $p = 0.028$). Those with deficiency, in addition to the negative correlation of 25(OH)D with VAI, also had TSF ($r = -0.472$, $p = 0.027$).

4. Discussion

In this study, using different body composition assessment methods, a negative correlation of vitamin D status with body fat, especially abdominal fat, and a positive correlation with muscle mass were found. Among the indices showing correlation, the WHtR and SMI_{BMI} stand out and can be easily incorporated into clinical practice.

4.1. Prevalence of Vitamin D Inadequacy and Deficiency

The literature highlights a high prevalence of vitamin D inadequacy and deficiency in adult women of childbearing age, similar to what was found in the current study [2,5,22,43–45]. Variations in prevalence values may be attributed to different geographical locations of the studied populations, as well as seasonal changes and sun exposure.

A noteworthy recent meta-analysis that involved 4006 participants aimed to assess vitamin D deficiency in women of childbearing age in Brazil. This analysis revealed an overall prevalence of deficiency (35%, 95% CI: 34–37%), insufficiency (42%, 95% CI: 41–44%), and combined deficiency and insufficiency of vitamin D (72%, 95% CI: 71–74%) [22], similar to the findings in the present study.

4.2. Relationship between Serum Vitamin D Concentrations and Muscle Mass

Women with vitamin D adequacy exhibited higher muscle mass (SMI_{BMI}), and this was positively correlated with the vitamin. Recently, SMI_{BMI} was identified as the most effective adjustment method for SMM, demonstrating a stronger association with functional and disability measures (handgrip strength, gait speed, frailty) than adjustment based on height² and weight [46].

These findings provide important information for clinical assessment, as they suggest a possible influence of vitamin D on muscle tissue. Thus, maintaining sufficient concentrations of vitamin D may possibly ensure better muscle capacity and functionality for these women.

In agreement, other authors have also found a relationship between vitamin D status and muscle tissue. A study involving young (25 to 45 years) and middle-aged (46 to 60 years) adult women found positive correlations between 25(OH)D concentrations and muscle strength (1 repetition maximum in supine and leg press and handgrip) in both age groups assessed [47]. Another study in subjects with BMI ≥ 25 kg/m² of both sexes (60% women) observed that those with vitamin D sufficiency (25(OH)D > 30 ng/mL) had significantly higher SMI_{weight} (% SMM) (31.8 ± 4.9 vs. 36.7 ± 6.1 , $p < 0.001$). Moreover, serum vitamin D concentrations were positively associated with SMM ($r = 0.18$, $p = 0.03$) and maintained their levels after adjustment for fat mass and age ($p = 0.003$) [48].

This relationship can be explained by the presence of the VDR receptor in muscle tissue acting in two ways, as a nuclear receptor mediating genomic effects and through non-nuclear receptors mediating non-genomic actions. Thus, 1,25 dihydroxyvitamin D (1,25(OH)₂D) may play a role in muscle contraction by increasing calcium mobilization through enhancing the activities of calbindin D9K, a calcium-binding protein, in cellular sarcoplasm [49]. It also activates important molecules of the store-operated calcium entry (SOCE) and voltage-dependent calcium channel (VDCC) pathways, resulting in extracellular calcium influx [50].

Furthermore, 1,25(OH)₂D can function in cell proliferation, differentiation, and growth through the activation of different molecules present in cell signaling pathways, such as phosphoinositide 3-kinase (PI3K), mitogen-activated protein kinase (MAPK), protein kinase B (AKT), and mammalian target of rapamycin (mTOR) [51]. In addition to increasing the expression of growth factors, such as insulin-like growth factor 1 (IGF-1), insulin-like growth factor 2 (IGF-2), vascular endothelial growth factor A (VEGFa), and fibroblast growth factor 1 (FGF-1) [52–54]. Thus, vitamin D may play an important role in increasing muscle mass.

Finally, 1,25(OH)₂D can also increase mitochondrial oxygen consumption, the volume fraction of mitochondria, and mitochondrial branching [55], as well as exert an inhibitory effect on oxidative stress and mitochondrial dynamics by activating AMPK and sirtulin 1 (SIRT1) in muscle cells [56].

As a result, deficiency of the active form of vitamin D in skeletal muscle can lead to alterations in metabolic pathways, ultimately resulting in decreased protein synthesis, muscle atrophy, and mitochondrial dysfunction [57]. Therefore, it is important to maintain sufficient vitamin D status to ensure its functionality in skeletal muscle.

4.3. Relationship between Serum Vitamin D Concentrations and Body Fat

Women with vitamin D inadequacy exhibited higher percent body fat (%BF) and central obesity (WHtR). Additionally, vitamin D was negatively correlated with body fat (WC, WHtR, CI, FMI, %BF and FMR). These findings underscore the importance of assessing body fat through complementary methods that do not rely solely on body weight, as most formulas for these variables use WC or fat mass measured by bioimpedance. Furthermore, these results reinforce that abdominal fat, often more visceral, may be related to vitamin D.

Similarly, Shantavasinkul et al. (2015) observed that individuals with vitamin D adequacy had significantly lower % BF and this was negatively associated with 25(OH)D concentrations [48]. Another study, on the other hand, involving both sexes (40.5% women), found an association of 25(OH)D with % BF, even after adjusting for age and sex, and a negative correlation of 25(OH)D with % BF as well [58]. Nikolova et al. (2018) demonstrated that 25(OH)D was negatively correlated with WC, WHtR, and % BF in women aged 20–59 years [59].

In a study by Patriota et al. (2022) evaluating men and women aged 35–75 years, a negative correlation of 25(OH)D was observed with all anthropometric markers (weight, BMI, WC, HC, WHtR, WHtR, % BF, CI, and body roundness index (BRI)) except ABSI in women. In addition, % CG, WHtR, and BMI were negatively associated with 25(OH)D in women [60]. Another study, by Arazi et al. (2019), found a negative association of 25(OH)D with BMI in both young and adult women. These findings of Patriota et al. (2022) and Arazi et al. (2019) related to BMI, unlike what was found in our study, may have occurred because the overall BMI means of their samples corresponded to overweight BMI ($25.1 \pm 4.8 \text{ kg/m}^2$ and $26.91 \pm 4.59 \text{ kg/m}^2$, respectively), which did not occur in our study [47,60].

In women with inadequacy and deficiency of vitamin D, it correlated negatively with visceral fat (VAI). This index aims to assess the distribution and function of visceral fat, as it correlates with factors that make up metabolic syndrome and cardiovascular and cerebrovascular events [33]. Some studies indicate that visceral fat, as measured by the omental compartment, has higher vitamin D concentrations compared to subcutaneous fat [61,62]. Thus, visceral adipose may play a crucial role in the deficiency of this vitamin.

Given the above, it is assumed that vitamin D inadequacy shows a stronger relationship with visceral adiposity, which, in turn, is associated with an increased rate of macrophage infiltration and increased release of pro-inflammatory adipokines that directly enter the portal circulation, promoting inflammation [63]. In addition, the amount of adiponectin is lower in visceral adipose tissue, further contributing to this pro-inflammatory scenario [64]. These alterations may pose significant cardiometabolic risks to these women,

particularly hypertension, resistance to insulin action, type 2 diabetes mellitus, dyslipidemia, and cardiovascular diseases [65–70].

Furthermore, inadequate vitamin D status seem to be related to the way body fat is distributed, depending on BMI. In normal-weight women, a greater relationship with central obesity (WHtR) stands out; in women with obesity, the relationships with weight, HC, and body fat percentage (BeW) stand out.

All these findings indicate a relationship between vitamin D and adiposity, but caution should be exercised when interpreting these data as they can go both ways. Excess adipose tissue can lead to lower vitamin D concentrations; conversely, lower vitamin D concentrations can result in excess body fat.

In this context, the literature provides some possible explanations for such findings. The first would be the fact that adipose tissue acts as the main reservoir of vitamin D in the body, thus decreasing its bioavailability, in addition to generating the inability of this vitamin to return to the circulation as a substrate for 25-hydroxylase in the liver [71–73]. Volumetric dilution is noted as another possible cause of low vitamin D concentrations in individuals with obesity, as vitamin D is distributed in a greater volume of serum, muscle, fat, and liver (increased body compartments in this situation), requiring a greater amount of vitamin D to saturate these deposits, thus decreasing its serum concentrations [61,74,75].

More recently, a study provided evidence to support the existence of an additional mechanism for these findings. Specifically, levels of CYP2R1, the major hepatic 25-hydroxylase, were significantly reduced in mice with obesity. Thus, less 25(OH)D would be converted at the hepatic level, thus explaining lower plasma concentrations [76].

In addition, there are some studies that support the hypothesis that vitamin D may be involved in the pathogenesis of obesity, rather than just a consequence. 1,25(OH)2D plays a role in controlling adipogenesis, energy metabolism, adipokines expression and inflammation, and apoptosis of adipose cells. In mouse preadipocyte cells, 1,25(OH)2D has been shown to stimulate adipogenesis by decreasing the expression of regulatory proteins, leading to terminal differentiation of preadipocytes and other related molecules. However, in human cells there appears to be increased adipocyte differentiation and lipid accumulation in the presence of 1,25(OH)2D through increased expression of adipogenic markers. Therefore, its effects on adipogenesis are still uncertain [77].

Regarding energy metabolism, mice with targeted expression of human VDR in adipocytes develop obesity, due to reduced energy expenditure [78]. In the same animal model, overexpression of VDR in adipose tissue induced increased body weight and fat mass, in addition to a decline in energy metabolism and a state of resistance to insulin action, resulting from a decrease in the expression of molecules important for energy expenditure [10]. In human adipocytes, 1,25(OH)2D inhibited lipolysis and promoted lipogenesis by increasing the expression of adipogenic proteins [79].

Regarding inflammation and adipokine expression, low concentrations of vitamin D were associated with increased inflammatory markers and generation of reactive oxygen species in visceral adipose tissue samples from obese individuals. Furthermore, incubation with 1,25(OH)2D decreased oxidative stress in this tissue [80]. Treatment with 1,25(OH)2D reduced the production of different pro-inflammatory adipokines and attenuated the inflammatory response in adipose tissue through inhibition of signaling pathways, such as nuclear factor kappa B (NFκB) and mitogen-activated protein kinase (MAPK) [77,81]. Finally, in 3T3-L1 cells, 1,25(OH)2D at physiological doses inhibited apoptosis, and at high doses stimulated it [82]. Apoptosis is induced and mediated by calcium, and 1,25(OH)2D, at high doses, activates calcium-dependent apoptotic proteases in adipose tissue [83].

The present study employs various methods for assessing body composition, aiming to elucidate its relationship with serum vitamin D concentrations, particularly in women of childbearing age. This constitutes a strong point, allowing for a more comprehensive understanding of the interplay between vitamin D, body adiposity, and muscle mass. However, it is important to acknowledge some limitations of this research, such as the cross-sectional model, which restricts the ability to establish temporal relationships or

observe changes over time. Additionally, the absence of sun exposure assessment and the inability to generalize the results to other demographic groups beyond the specified age range are aspects to be considered.

5. Conclusions

We observed a high percentage of vitamin D status inadequacy related to lower muscle mass and higher body fat (total and visceral). We also observed a positive correlation between 25(OH)D and muscle mass and a negative correlation with body fat. These findings suggest that inadequate levels of this vitamin may affect muscle tissue and be involved with higher body adiposity, including visceral adiposity.

We suggest the inclusion of these body variables in clinical practice, especially WHtR and SMI_{BMI}, to minimize the metabolic consequences resulting from vitamin inadequacy.

Author Contributions: Conceptualization, A.R., M.T.T. and P.M.M.; methodology, A.R., M.T.T., P.M.M. and O.A.C.; validation, P.M.M. and S.P.d.C.; formal analysis, P.M.M. and S.P.d.C.; investigation, P.M.M. and O.A.C.; resources, A.R., M.T.T. and O.A.C.; writing—original draft preparation, A.R., P.M.M. and S.P.d.C.; writing—review and editing, A.R., M.T.T., P.M.M., S.P.d.C. and O.A.C.; visualization, P.M.M.; supervision, A.R.; project administration, A.R. and M.T.T. All authors have read and agreed to the published version of the manuscript.

Funding: This research was funded by Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES), protocol 88882.331804/2019-01 and Fundação Carlos Chagas Filho de Amparo à Pesquisa do Estado do Rio de Janeiro (FAPERJ), protocol E-26/200.876/2021-6 august 2021.

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki, and approved by the Research Ethics Committee of the Federal University of the State of Rio de Janeiro (UNIRIO) (protocol code CAEE 50063715.5.0000.5285 and date of approval was 23 November 2015).

Informed Consent Statement: Patient consent was waived due to research was carried out by consulting medical records.

Data Availability Statement: The data used to support the findings of this study are available from the corresponding author upon request. Data is not publicly available due to privacy.

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

References

- Alzaheb, R.A. The Prevalence of Hypovitaminosis D and Its Associated Risk Factors Among Women of Reproductive Age in Saudi Arabia: A Systematic Review and Meta-Analysis. *Clin. Med. Insights Women's Health* **2018**, *11*, 1179562X18767884. [[CrossRef](#)] [[PubMed](#)]
- Da Silveira, E.A.; Moura, L.d.A.N.E.; Castro, M.C.R.; Kac, G.; Hadler, M.C.C.M.; Noll, P.R.E.S.; Noll, M.; Rezende, A.T.d.O.; Delpino, F.M.; de Oliveira, C. Prevalence of Vitamin D and Calcium Deficiency and Insufficiency in Women of Childbearing Age and Associated Risk Factors: A Systematic Review and Meta-Analysis. *Nutrients* **2022**, *14*, 4351. [[CrossRef](#)] [[PubMed](#)]
- Sofi, N.Y.; Jain, M.; Kapil, U.; Seenu, V.; Ramakrishnan, L.; Yadav, C.P.; Pandey, R.M. Status of Serum Vitamin D and Calcium Levels in Women of Reproductive Age in National Capital Territory of India. *Indian J. Endocrinol. Metab.* **2017**, *21*, 731. [[CrossRef](#)] [[PubMed](#)]
- Ginde, A.A.; Sullivan, A.F.; Mansbach, J.M.; Camargo, C.A. Vitamin D Insufficiency in Pregnant and Nonpregnant Women of Childbearing Age in the United States. *Am. J. Obstet. Gynecol.* **2010**, *202*, 436.e1–436.e8. [[CrossRef](#)] [[PubMed](#)]
- Hedlund, L.; Brembeck, P.; Olausson, H. Determinants of Vitamin D Status in Fair-Skinned Women of Childbearing Age at Northern Latitudes. *PLoS ONE* **2013**, *8*, e60864. [[CrossRef](#)] [[PubMed](#)]
- Saeedian Kia, A.; Amani, R.; Cheraghian, B. The Association between the Risk of Premenstrual Syndrome and Vitamin D, Calcium, and Magnesium Status among University Students: A Case Control Study. *Health Promot. Perspect.* **2015**, *5*, 225–230. [[CrossRef](#)] [[PubMed](#)]
- Karlsson, T.; Osancevic, A.; Jansson, N.; Hulthén, L.; Holmäng, A.; Larsson, I. Increased Vitamin D-Binding Protein and Decreased Free 25(OH)D in Obese Women of Reproductive Age. *Eur. J. Nutr.* **2014**, *53*, 259–267. [[CrossRef](#)] [[PubMed](#)]
- Halfon, M.; Phan, O.; Teta, D. Vitamin D: A Review on Its Effects on Muscle Strength, the Risk of Fall, and Frailty. *BioMed. Res. Int.* **2015**, *2015*, 953241. [[CrossRef](#)]
- Nimitphong, H.; Holick, M.F.; Fried, S.K.; Lee, M.-J. 25-Hydroxyvitamin D3 and 1,25-Dihydroxyvitamin D3 Promote the Differentiation of Human Subcutaneous Preadipocytes. *PLoS ONE* **2012**, *7*, e52171. [[CrossRef](#)]

10. Xu, Y.; Lou, Y.; Kong, J. VDR Regulates Energy Metabolism by Modulating Remodeling in Adipose Tissue. *Eur. J. Pharmacol.* **2019**, *865*, 172761. [[CrossRef](#)]
11. Ceglia, L.; da Silva Morais, M.; Park, L.K.; Morris, E.; Harris, S.S.; Bischoff-Ferrari, H.A.; Fielding, R.A.; Dawson-Hughes, B. Multi-Step Immunofluorescent Analysis of Vitamin D Receptor Loci and Myosin Heavy Chain Isoforms in Human Skeletal Muscle. *J. Mol. Histol.* **2010**, *41*, 137–142. [[CrossRef](#)]
12. Bennour, I.; Haroun, N.; Sicard, F.; Mounien, L.; Landrier, J.-F. Vitamin D and Obesity/Adiposity—A Brief Overview of Recent Studies. *Nutrients* **2022**, *14*, 2049. [[CrossRef](#)] [[PubMed](#)]
13. Muscogiuri, G.; Barrea, L.; Somma, C.D.; Laudisio, D.; Salzano, C.; Pugliese, G.; de Alteriis, G.; Colao, A.; Savastano, S. Sex Differences of Vitamin D Status across BMI Classes: An Observational Prospective Cohort Study. *Nutrients* **2019**, *11*, 3034. [[CrossRef](#)] [[PubMed](#)]
14. Leiu, K.H.; Chin, Y.S.; Mohd Shariff, Z.; Arumugam, M.; Chan, Y.M. High Body Fat Percentage and Low Consumption of Dairy Products Were Associated with Vitamin D Inadequacy among Older Women in Malaysia. *PLoS ONE* **2020**, *15*, e0228803. [[CrossRef](#)] [[PubMed](#)]
15. De Pergola, G.; Martino, T.; Zupo, R.; Caccavo, D.; Pecorella, C.; Paradiso, S.; Silvestris, F.; Triggiani, V. 25 Hydroxyvitamin D Levels Are Negatively and Independently Associated with Fat Mass in a Cohort of Healthy Overweight and Obese Subjects. *Endocr. Metab. Immune Disord. Drug Targets* **2019**, *19*, 838–844. [[CrossRef](#)] [[PubMed](#)]
16. Rafiq, S.; Jeppesen, P.B. Body Mass Index, Vitamin D, and Type 2 Diabetes: A Systematic Review and Meta-Analysis. *Nutrients* **2018**, *10*, 1182. [[CrossRef](#)] [[PubMed](#)]
17. Al Hayek, S.; Matar Bou Mosleh, J.; Ghadieh, R.; El Hayek Fares, J. Vitamin D Status and Body Composition: A Cross-Sectional Study among Employees at a Private University in Lebanon. *BMC Nutr.* **2018**, *4*, 31. [[CrossRef](#)] [[PubMed](#)]
18. Delle Monache, S.; Di Fulvio, P.; Iannetti, E.; Valerii, L.; Capone, L.; Nespoli, M.G.; Bologna, M.; Angelucci, A. Body Mass Index Represents a Good Predictor of Vitamin D Status in Women Independently from Age. *Clin. Nutr. Edinb. Scotl.* **2019**, *38*, 829–834. [[CrossRef](#)] [[PubMed](#)]
19. Shoemaker, M.E.; Salmon, O.F.; Smith, C.M.; Duarte-Gardea, M.O.; Cramer, J.T. Influences of Vitamin D and Iron Status on Skeletal Muscle Health: A Narrative Review. *Nutrients* **2022**, *14*, 2717. [[CrossRef](#)]
20. Fernando, M.; Ellery, S.J.; Marquina, C.; Lim, S.; Naderpoor, N.; Mousa, A. Vitamin D-Binding Protein in Pregnancy and Reproductive Health. *Nutrients* **2020**, *12*, 1489. [[CrossRef](#)]
21. Pilz, S.; Zittermann, A.; Obeid, R.; Hahn, A.; Pludowski, P.; Trummer, C.; Lerchbaum, E.; Pérez-López, F.R.; Karras, S.N.; März, W. The Role of Vitamin D in Fertility and during Pregnancy and Lactation: A Review of Clinical Data. *Int. J. Environ. Res. Public Health* **2018**, *15*, 2241. [[CrossRef](#)] [[PubMed](#)]
22. Lucchetta, R.C.; Lemos, I.H.; Gini, A.L.R.; Cavicchioli, S.d.A.; Forgerini, M.; Varallo, F.R.; de Nadai, M.N.; Fernandez-Llimos, F.; Mastroianni, P. de C. Deficiency and Insufficiency of Vitamin D in Women of Childbearing Age: A Systematic Review and Meta-Analysis. *Rev. Bras. Ginecol. E Obstet. Rev. Fed. Bras. Soc. Ginecol. E Obstet.* **2022**, *44*, 409–424. [[CrossRef](#)]
23. Dorsey, J.G. Introduction to Modern Liquid Chromatography, 3rd ed. *J. Am. Chem. Soc.* **2010**, *132*, 9220. [[CrossRef](#)]
24. Holick, M.F.; Binkley, N.C.; Bischoff-Ferrari, H.A.; Gordon, C.M.; Hanley, D.A.; Heaney, R.P.; Murad, M.H.; Weaver, C.M. Endocrine Society Evaluation, Treatment, and Prevention of Vitamin D Deficiency: An Endocrine Society Clinical Practice Guideline. *J. Clin. Endocrinol. Metab.* **2011**, *96*, 1911–1930. [[CrossRef](#)] [[PubMed](#)]
25. Lohman, T.G.; Roche, A.F.; Martorell, R. *Anthropometric Standardization Reference Manual*; Human Kinetics Books: Champaign, IL, USA, 1988; ISBN 978-0-87322-121-4.
26. WHO. *Obesity: Preventing and Managing the Global Epidemic: Report of a WHO Consultation*; World Health Organization: Geneva, Switzerland, 2000.
27. Jelliffe, D.B.; WHO. *The Assessment of the Nutritional Status of the Community (with Special Reference to Field Surveys in Developing Regions of the World)*; World Health Organization: Geneva, Switzerland, 1966; ISBN 978-92-4-140053-4.
28. Frisancho, A.R. Triceps Skin Fold and Upper Arm Muscle Size Norms for Assessment of Nutrition Status. *Am. J. Clin. Nutr.* **1974**, *27*, 1052–1058. [[CrossRef](#)] [[PubMed](#)]
29. Gurney, J.M.; Jelliffe, D.B. Arm Anthropometry in Nutritional Assessment: Nomogram for Rapid Calculation of Muscle Circumference and Cross-Sectional Muscle and Fat Areas. *Am. J. Clin. Nutr.* **1973**, *26*, 912–915. [[CrossRef](#)] [[PubMed](#)]
30. WHO. *Waist Circumference and Waist-Hip Ratio: Report of a WHO Expert Consultation, Geneva, 8–11 December 2008*; World Health Organization: Geneva, Switzerland, 2008; ISBN 978-92-4-150149-1.
31. Valdez, R. A Simple Model-Based Index of Abdominal Adiposity. *J. Clin. Epidemiol.* **1991**, *44*, 955–956. [[CrossRef](#)]
32. Ashwell, M.; Lejeune, S.; McPherson, K. Ratio of Waist Circumference to Height May Be Better Indicator of Need for Weight Management. *BMJ* **1996**, *312*, 377. [[CrossRef](#)] [[PubMed](#)]
33. Amato, M.C.; Giordano, C.; Galia, M.; Criscimanna, A.; Vitabile, S.; Midiri, M.; Galluzzo, A. Visceral Adiposity Index. *Diabetes Care* **2010**, *33*, 920–922. [[CrossRef](#)]
34. Bergman, R.N.; Stefanovski, D.; Buchanan, T.A.; Sumner, A.E.; Reynolds, J.C.; Sebring, N.G.; Xiang, A.H.; Watanabe, R.M. A Better Index of Body Adiposity. *Obesity* **2011**, *19*, 1083–1089. [[CrossRef](#)]
35. Krakauer, N.Y.; Krakauer, J.C. A New Body Shape Index Predicts Mortality Hazard Independently of Body Mass Index. *PLoS ONE* **2012**, *7*, e39504. [[CrossRef](#)] [[PubMed](#)]

36. Gómez-Ambrosi, J.; Silva, C.; Catalán, V.; Rodríguez, A.; Galofré, J.C.; Escalada, J.; Valentí, V.; Rotellar, F.; Romero, S.; Ramírez, B.; et al. Clinical Usefulness of a New Equation for Estimating Body Fat. *Diabetes Care* **2012**, *35*, 383–388. [[CrossRef](#)] [[PubMed](#)]
37. Belarmino, G.; Torrinhas, R.S.; Sala, P.; Horie, L.M.; Damiani, L.; Lopes, N.C.; Heymsfield, S.B.; Waitzberg, D.L. A New Anthropometric Index for Body Fat Estimation in Patients with Severe Obesity. *BMC Obes.* **2018**, *5*, 25. [[CrossRef](#)] [[PubMed](#)]
38. Schutz, Y.; Kyle, U.U.G.; Pichard, C. Fat-Free Mass Index and Fat Mass Index Percentiles in Caucasians Aged 18–98 y. *Int. J. Obes. Relat. Metab. Disord. J. Int. Assoc. Study Obes.* **2002**, *26*, 953–960. [[CrossRef](#)] [[PubMed](#)]
39. Xu, K.; Zhu, H.J.; Chen, S.; Chen, L.; Wang, X.; Zhang, L.Y.; Pan, L.; Wang, L.; Feng, K.; Wang, K.; et al. Fat-to-Muscle Ratio: A New Anthropometric Indicator for Predicting Metabolic Syndrome in the Han and Bouyei Populations from Guizhou Province, China. *Biomed. Environ. Sci. BES* **2018**, *31*, 261–271. [[CrossRef](#)] [[PubMed](#)]
40. Baumgartner, R.N.; Koehler, K.M.; Gallagher, D.; Romero, L.; Heymsfield, S.B.; Ross, R.R.; Garry, P.J.; Lindeman, R.D. Epidemiology of Sarcopenia among the Elderly in New Mexico. *Am. J. Epidemiol.* **1998**, *147*, 755–763. [[CrossRef](#)] [[PubMed](#)]
41. Janssen, I.; Heymsfield, S.B.; Ross, R. Low Relative Skeletal Muscle Mass (Sarcopenia) in Older Persons Is Associated with Functional Impairment and Physical Disability. *J. Am. Geriatr. Soc.* **2002**, *50*, 889–896. [[CrossRef](#)] [[PubMed](#)]
42. Cawthon, P.M.; Peters, K.W.; Shardell, M.D.; McLean, R.R.; Dam, T.-T.L.; Kenny, A.M.; Fragala, M.S.; Harris, T.B.; Kiel, D.P.; Guralnik, J.M.; et al. Cutpoints for Low Appendicular Lean Mass That Identify Older Adults with Clinically Significant Weakness. *J. Gerontol. A Biol. Sci. Med. Sci.* **2014**, *69*, 567–575. [[CrossRef](#)] [[PubMed](#)]
43. Becker, W.; Lindroos, A.K.; Nälsén, C.; Warensjö Lemming, E.; Öhrvik, V. Dietary Habits, Nutrient Intake and Biomarkers for Folate, Vitamin D, Iodine and Iron Status among Women of Childbearing Age in Sweden. *Ups. J. Med. Sci.* **2016**, *121*, 271–275. [[CrossRef](#)]
44. Burke, N.L.; Harville, E.W.; Wickliffe, J.K.; Shankar, A.; Lichtveld, M.Y.; McCaskill, M.L. Determinants of Vitamin D Status among Black and White Low-Income Pregnant and Non-Pregnant Reproductive-Aged Women from Southeast Louisiana. *BMC Pregnancy Childbirth* **2019**, *19*, 111. [[CrossRef](#)]
45. Lopes, V.M.; Lopes, J.R.C.; Brasileiro, J.P.B.; Oliveira, I.d.; Lacerda, R.P.; Andrade, M.R.D.; Tierno, N.I.Z.; Souza, R.C.C.d.; Motta, L.A.C.R. da Highly Prevalence of Vitamin D Deficiency among Brazilian Women of Reproductive Age. *Arch. Endocrinol. Metab.* **2017**, *61*, 21–27. [[CrossRef](#)] [[PubMed](#)]
46. Bahat, G.; Kilic, C.; Ilhan, B.; Karan, M.A.; Cruz-Jentoft, A. Association of Different Bioimpedanciometry Estimations of Muscle Mass with Functional Measures. *Geriatr. Gerontol. Int.* **2019**, *19*, 593–597. [[CrossRef](#)]
47. Arazi, H.; Eghbali, E. 25-Hydroxyvitamin D Levels and Its Relation to Muscle Strength, Maximal Oxygen Consumption, and Body Mass Index in Young and Middle Adulthood Women. *Int. J. Womens Health* **2019**, *11*, 57–64. [[CrossRef](#)] [[PubMed](#)]
48. Shantavasinkul, P.C.; Phanachet, P.; Puchaiwattananon, O.; Chailurkit, L.; Lapananon, T.; Chanprasertyotin, S.; Ongphiphadhanakul, B.; Warodomwicht, D. Vitamin D Status Is a Determinant of Skeletal Muscle Mass in Obesity According to Body Fat Percentage. *Nutrition* **2015**, *31*, 801–806. [[CrossRef](#)] [[PubMed](#)]
49. Lila, D.; Susana, Z.; Ricardo, B. Induction of a Calbindin-D9K-like Protein in Avian Muscle Cells by 1,25-Dihydroxy-Vitamin D3. *Biochem. Mol. Biol. Int.* **1994**, *32*, 859–867. [[PubMed](#)]
50. Girgis, C.M.; Clifton-Bligh, R.J.; Hamrick, M.W.; Holick, M.F.; Gunton, J.E. The Roles of Vitamin D in Skeletal Muscle: Form, Function, and Metabolism. *Endocr. Rev.* **2013**, *34*, 33–83. [[CrossRef](#)]
51. Domingues-Faria, C.; Boirie, Y.; Walrand, S. Vitamin D and Muscle Trophicity. *Curr. Opin. Clin. Nutr. Metab. Care* **2017**, *20*, 169–174. [[CrossRef](#)] [[PubMed](#)]
52. Garcia, L.A.; King, K.K.; Ferrini, M.G.; Norris, K.C.; Artaza, J.N. 1,25(OH)₂vitamin D₃ Stimulates Myogenic Differentiation by Inhibiting Cell Proliferation and Modulating the Expression of Promyogenic Growth Factors and Myostatin in C2C12 Skeletal Muscle Cells. *Endocrinology* **2011**, *152*, 2976–2986. [[CrossRef](#)] [[PubMed](#)]
53. Garcia, L.A.; Ferrini, M.G.; Norris, K.C.; Artaza, J.N. 1,25(OH)₂Vitamin D₃ Enhances Myogenic Differentiation by Modulating the Expression of Key Angiogenic Growth Factors and Angiogenic Inhibitors in C(2)C(12) Skeletal Muscle Cells. *J. Steroid Biochem. Mol. Biol.* **2013**, *133*, 1–11. [[CrossRef](#)]
54. Braga, M.; Simmons, Z.; Norris, K.C.; Ferrini, M.G.; Artaza, J.N. Vitamin D Induces Myogenic Differentiation in Skeletal Muscle Derived Stem Cells. *Endocr. Connect.* **2017**, *6*, 139–150. [[CrossRef](#)]
55. Ryan, Z.C.; Craig, T.A.; Folmes, C.D.; Wang, X.; Lanza, I.R.; Schaible, N.S.; Salisbury, J.L.; Nair, K.S.; Terzic, A.; Sieck, G.C.; et al. 1 α ,25-Dihydroxyvitamin D₃ Regulates Mitochondrial Oxygen Consumption and Dynamics in Human Skeletal Muscle Cells. *J. Biol. Chem.* **2016**, *291*, 1514–1528. [[CrossRef](#)] [[PubMed](#)]
56. Chang, E. 1,25-Dihydroxyvitamin D Decreases Tertiary Butyl-Hydrogen Peroxide-Induced Oxidative Stress and Increases AMPK/SIRT1 Activation in C2C12 Muscle Cells. *Molecules* **2019**, *24*, 3903. [[CrossRef](#)] [[PubMed](#)]
57. Dzik, K.P.; Kaczor, J.J. Mechanisms of Vitamin D on Skeletal Muscle Function: Oxidative Stress, Energy Metabolism and Anabolic State. *Eur. J. Appl. Physiol.* **2019**, *119*, 825–839. [[CrossRef](#)] [[PubMed](#)]
58. Mousa, A.; Naderpoor, N.; de Courten, M.P.J.; Scragg, R.; de Courten, B. 25-Hydroxyvitamin D Is Associated with Adiposity and Cardiometabolic Risk Factors in a Predominantly Vitamin D-Deficient and Overweight/Obese but Otherwise Healthy Cohort. *J. Steroid Biochem. Mol. Biol.* **2017**, *173*, 258–264. [[CrossRef](#)] [[PubMed](#)]

59. Nikolova, M.G.; Boyanov, M.A.; Tsakova, A.D. Correlations of serum vitamin D with metabolic parameters in adult outpatients with different degrees of overweight/obesity coming from an urban community. *Acta Endocrinol.* **2018**, *14*, 375–383. [[CrossRef](#)] [[PubMed](#)]
60. Patriota, P.; Rezzi, S.; Guessous, I.; Marques-Vidal, P. Association between Anthropometric Markers of Adiposity, Adipokines and Vitamin D Levels. *Sci. Rep.* **2022**, *12*, 15435. [[CrossRef](#)] [[PubMed](#)]
61. Carrelli, A.; Bucovsky, M.; Horst, R.; Cremers, S.; Zhang, C.; Bessler, M.; Schrope, B.; Evanko, J.; Blanco, J.; Silverberg, S.J.; et al. Vitamin D Storage in Adipose Tissue of Obese and Normal Weight Women. *J. Bone Miner. Res. Off. J. Am. Soc. Bone Miner. Res.* **2017**, *32*, 237–242. [[CrossRef](#)] [[PubMed](#)]
62. Beckman, L.M.; Earthman, C.P.; Thomas, W.; Compher, C.W.; Muniz, J.; Horst, R.L.; Ikramuddin, S.; Kellogg, T.A.; Sibley, S.D. Serum 25(OH) Vitamin D Concentration Changes after Roux-En-Y Gastric Bypass Surgery. *Obesity* **2013**, *21*, E599–E606. [[CrossRef](#)] [[PubMed](#)]
63. Fontana, L.; Eagon, J.C.; Trujillo, M.E.; Scherer, P.E.; Klein, S. Visceral Fat Adipokine Secretion Is Associated with Systemic Inflammation in Obese Humans. *Diabetes* **2007**, *56*, 1010–1013. [[CrossRef](#)]
64. Wronska, A.; Kmiec, Z. Structural and Biochemical Characteristics of Various White Adipose Tissue Depots. *Acta Physiol.* **2012**, *205*, 194–208. [[CrossRef](#)]
65. Le Jemtel, T.H.; Samson, R.; Milligan, G.; Jaiswal, A.; Oparil, S. Visceral Adipose Tissue Accumulation and Residual Cardiovascular Risk. *Curr. Hypertens. Rep.* **2018**, *20*, 77. [[CrossRef](#)] [[PubMed](#)]
66. Matsha, T.E.; Ismail, S.; Speelman, A.; Hon, G.M.; Davids, S.; Erasmus, R.T.; Kengne, A.P. Visceral and Subcutaneous Adipose Tissue Association with Metabolic Syndrome and Its Components in a South African Population. *Clin. Nutr. ESPEN* **2019**, *32*, 76–81. [[CrossRef](#)] [[PubMed](#)]
67. Sato, F.; Maeda, N.; Yamada, T.; Namazui, H.; Fukuda, S.; Natsukawa, T.; Nagao, H.; Murai, J.; Masuda, S.; Tanaka, Y.; et al. Association of Epicardial, Visceral, and Subcutaneous Fat with Cardiometabolic Diseases. *Circ. J.* **2018**, *82*, 502–508. [[CrossRef](#)] [[PubMed](#)]
68. Jung, U.J.; Choi, M.-S. Obesity and Its Metabolic Complications: The Role of Adipokines and the Relationship between Obesity, Inflammation, Insulin Resistance, Dyslipidemia and Nonalcoholic Fatty Liver Disease. *Int. J. Mol. Sci.* **2014**, *15*, 6184–6223. [[CrossRef](#)] [[PubMed](#)]
69. Neeland, I.J.; Turer, A.T.; Ayers, C.R.; Powell-Wiley, T.M.; Vega, G.L.; Farzaneh-Far, R.; Grundy, S.M.; Khera, A.; McGuire, D.K.; de Lemos, J.A. Dysfunctional Adiposity and the Risk of Prediabetes and Type 2 Diabetes in Obese Adults. *JAMA* **2012**, *308*, 1150–1159. [[CrossRef](#)] [[PubMed](#)]
70. Fox, C.S.; Massaro, J.M.; Hoffmann, U.; Pou, K.M.; Maurovich-Horvat, P.; Liu, C.-Y.; Vasan, R.S.; Murabito, J.M.; Meigs, J.B.; Cupples, L.A.; et al. Abdominal Visceral and Subcutaneous Adipose Tissue Compartments: Association with Metabolic Risk Factors in the Framingham Heart Study. *Circulation* **2007**, *116*, 39–48. [[CrossRef](#)] [[PubMed](#)]
71. Wortsman, J.; Matsuoka, L.Y.; Chen, T.C.; Lu, Z.; Holick, M.F. Decreased Bioavailability of Vitamin D in Obesity. *Am. J. Clin. Nutr.* **2000**, *72*, 690–693. [[CrossRef](#)] [[PubMed](#)]
72. Heaney, R.P.; Recker, R.R.; Grote, J.; Horst, R.L.; Armas, L.A.G. Vitamin D(3) Is More Potent than Vitamin D(2) in Humans. *J. Clin. Endocrinol. Metab.* **2011**, *96*, E447–E452. [[CrossRef](#)] [[PubMed](#)]
73. Brouwer, D.A.; van Beek, J.; Ferwerda, H.; Brugman, A.M.; van der Klis, F.R.; van der Heiden, H.J.; Muskiet, F.A. Rat Adipose Tissue Rapidly Accumulates and Slowly Releases an Orally-Administered High Vitamin D Dose. *Br. J. Nutr.* **1998**, *79*, 527–532. [[CrossRef](#)]
74. Drincic, A.T.; Armas, L.A.G.; Van Diest, E.E.; Heaney, R.P. Volumetric Dilution, Rather than Sequestration Best Explains the Low Vitamin D Status of Obesity. *Obesity* **2012**, *20*, 1444–1448. [[CrossRef](#)]
75. Walsh, J.S.; Bowles, S.; Evans, A.L. Vitamin D in Obesity. *Curr. Opin. Endocrinol. Diabetes Obes.* **2017**, *24*, 389–394. [[CrossRef](#)] [[PubMed](#)]
76. Roizen, J.D.; Long, C.; Casella, A.; O’Lear, L.; Caplan, I.; Lai, M.; Sasson, I.; Singh, R.; Makowski, A.J.; Simmons, R.; et al. Obesity Decreases Hepatic 25-Hydroxylase Activity Causing Low Serum 25-Hydroxyvitamin D. *J. Bone Miner. Res. Off. J. Am. Soc. Bone Miner. Res.* **2019**, *34*, 1068–1073. [[CrossRef](#)] [[PubMed](#)]
77. Mutt, S.J.; Hyppönen, E.; Saarnio, J.; Järvelin, M.-R.; Herzig, K.-H. Vitamin D and Adipose Tissue—More than Storage. *Front. Physiol.* **2014**, *5*, 228. [[CrossRef](#)] [[PubMed](#)]
78. Wong, K.E.; Kong, J.; Zhang, W.; Szeto, F.L.; Ye, H.; Deb, D.K.; Brady, M.J.; Li, Y.C. Targeted Expression of Human Vitamin D Receptor in Adipocytes Decreases Energy Expenditure and Induces Obesity in Mice. *J. Biol. Chem.* **2011**, *286*, 33804–33810. [[CrossRef](#)] [[PubMed](#)]
79. Shi, H.; Norman, A.W.; Okamura, W.H.; Sen, A.; Zemel, M.B. 1 α ,25-Dihydroxyvitamin D₃ Modulates Human Adipocyte Metabolism via Nongenomic Action. *FASEB J. Off. Publ. Fed. Am. Soc. Exp. Biol.* **2001**, *15*, 2751–2753. [[CrossRef](#)]
80. Ionica, M.; Aburel, O.M.; Vaduva, A.; Petrus, A.; Rațiu, S.; Olariu, S.; Sturza, A.; Muntean, D.M. Vitamin D Alleviates Oxidative Stress in Adipose Tissue and Mesenteric Vessels from Obese Patients with Subclinical Inflammation. *Can. J. Physiol. Pharmacol.* **2020**, *98*, 85–92. [[CrossRef](#)] [[PubMed](#)]
81. Park, C.Y.; Kim, T.Y.; Yoo, J.S.; Seo, Y.; Pae, M.; Han, S.N. Effects of 1,25-Dihydroxyvitamin D₃ on the Inflammatory Responses of Stromal Vascular Cells and Adipocytes from Lean and Obese Mice. *Nutrients* **2020**, *12*, 364. [[CrossRef](#)] [[PubMed](#)]

82. Sun, X.; Zemel, M.B. Role of Uncoupling Protein 2 (UCP2) Expression and 1alpha, 25-Dihydroxyvitamin D3 in Modulating Adipocyte Apoptosis. *FASEB J. Off. Publ. Fed. Am. Soc. Exp. Biol.* **2004**, *18*, 1430–1432. [[CrossRef](#)]
83. Sergeev, I.N.; Song, Q. High Vitamin D and Calcium Intakes Reduce Diet-Induced Obesity in Mice by Increasing Adipose Tissue Apoptosis. *Mol. Nutr. Food Res.* **2014**, *58*, 1342–1348. [[CrossRef](#)]

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.