Hyperandrogenism Enhances Muscle Strength After Progressive Resistance Training, Independent of Body Composition, in Women With Polycystic Ovary Syndrome

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Abstract

Kogure, GS, Silva, RC, Miranda-Furtado, CL, Ribeiro, VB, Pedroso, DCC, Melo, AS, Ferriani, RA, and Reis, RMd. Hyperandrogenism enhances muscle strength after progressive resistance training, independent of body composition, in women with polycystic ovary syndrome. *J Strength Cond Res* 32(9): 2642–2651, 2018—The effects of resistance exercise on muscle strength, body composition, and increase in cross-sectional area of skeletal muscle (hypertrophy) were evaluated in women with polycystic ovary syndrome (PCOS). This case-control study included 45 PCOS and 52 non-PCOS women, with age between 18–37 years and body mass index of 18–39.9 $kg \cdot m^{-2}$. Subjects performed a program of progressive resistance training (PRT), 3 times per week for 4 months. Biochemical characteristics were measured before and after PRT. Muscle strength evaluated by 1 maximum repetition test and body composition and hypertrophy indicator, evaluated by anthropometry, were measured at baseline, at 8 weeks, and at 16 weeks after PRT. Progressive resistance training produced an increase in maximum strength (bench press, $p = 0.04$; leg extension, $p = 0.04$) in the PCOS group; however, no changes were observed in body composition between groups. Concentration of testosterone decreased in both PCOS and non-PCOS groups ($p < 0.01$, both) after PRT, as well as glycemia (PCOS, $p = 0.01$; non-PCOS, $p = 0.02$) and body fat percentage ($p < 0.01$, both). An increase in hypertrophy indicators, lean body mass (LBM), and maximum strength on all exercises was observed in both PCOS and non-PCOS groups ($p < 0.01$). This training protocol promoted increases in muscle strength in PCOS women, and improved hyperandrogenism and body composition by decreasing body fat and increasing LBM and muscle strength in both PCOS and non-PCOS groups. Therefore, it is suggested that resistance exercise programs could promote health and fitness in women of reproductive age, especially functional capacity of strength those with PCOS.

Key Words testosterone concentration, anthropometry, strength training

Introduction

Polycystic ovary syndrome (PCOS) is a common endocrine-metabolic disease, affecting 7–14% of women of reproductive age (26), and is usually characterized by chronic anovulation, hyperandrogenism, hyperinsulinemia, and obesity. It is associated with an increased risk of several metabolic complications, including insulin resistance (IR), type 2 diabetes, dyslipidemia and, possibly, cardiovascular disease (49). The excess androgen favors the deposition of abdominal fat in these women, which may exacerbate the adverse metabolic effects of PCOS (11).

Exercise can improve muscle strength and positively influence body composition, thereby increasing lean body mass (LBM) and decreasing visceral and subcutaneous adipose tissue in the abdominal region (44) in women with PCOS (21). In particular, increased strength and muscle mass, the primary outcomes of resistance training (RT), have been evidenced to reduce the risk of all-cause mortality (5); it is suggested that it can improve clinical outcomes in PCOS (9,24). However, these effects seem to be related, in part,

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with gains in muscle strength, rather than changes in muscle mass (17).

Although resistance exercise is used for all these purposes, effectiveness may be limited by decreased physical fitness levels, as individuals would be unable to meet the training load required for conditioning and hypertrophy (31), as well as by hormonal factors, with androgens promoting an intense physiological effect on body composition, including testosterone, which is considered as a physiological marker of the anabolic state of the body and of muscle strength (37). The development of strength with RT is also influenced by neural and morphological adaptations (13). In addition, a previous study by our team identified greater muscle strength in untrained women with PCOS than in a control group of women without PCOS (22). This higher baseline muscle strength associated with PCOS likely reflects the high androgen levels in these women (21).

Based on this context and knowing that improvements in muscle performance depends on the type, intensity, and duration of the exercise stimulus (28), the primary objective of our study was to evaluate the effects of 8 and 16 weeks of progressive resistance training (PRT) through linear periodization on body composition, indicators of hypertrophy, and muscle strength in women with PCOS compared with women with regular ovulatory cycles.

Methods

Experimental Approach to the Problem

A nonrandomized, therapeutic, open, single-arm case-control study of a 16-week program of PRT was performed for 1 hour per day, 3 times per week, on nonconsecutive days, for 4 months. The PRT program was based on the linear model of periodization, which involves continually increasing the level of muscle strength required as strength or endurance improve, with the aim of reducing the volume but increasing the intensity of training over time (41). Participants were screened clinically and biochemically for enrollment into a PCOS and non-PCOS control groups. The participants were not blinded to the PRT protocol. The PRT was initially supervised by a professional physical educator for all participants.

Biochemical measurements were measured at 2 time points: before and after PRT intervention. Body composition and hypertrophy indicators were measured by anthropometry and muscle strength using the 1 maximum repetition (1RM) method, at 3 time points: before, halfway through the intervention (8-weeks), and after PRT (16-week). Although this investigation was part of clinical trial that resulted in previous publications, none of the data of muscle strength measured after PRT in women with PCOS has previously been published.

Subjects

From February 2010 to December 2013, women with and without PCOS, 18–37 years of age, with a body mass index (BMI) in the normal (18–25 $kg \cdot m^{-2}$), overweight (25–29.0 $kg \cdot m^{-2}$), or first-degree obesity (>30 $kg \cdot m^{-2}$) ranged according to the World Health Organization (WHO) criteria, and who had not previously engaged in regular and systematic resistance training were recruited as previously described (21). The exclusion criteria were systemic diseases, hormonal contraceptive use, smoking, and pregnancy. Participants who did not complete the study were also excluded from the analysis.

Participants were assigned to the PCOS or non-PCOS groups based on results from transvaginal pelvic ultrasonography, performed using a Voluson 730 Expert machine (GE Medical Systems, Zipf, Austria), and assessment of clinical and biochemical hyperandrogenism. The PCOS group ($n = 45$) was classified using the Rotterdam consensus (10). The non-PCOS group ($n = 52$) consisted of healthy women with regular menstrual cycles of 24–32 days, with 3 to 7 days of duration. Before participation, participants were fully informed of the design of the study and the possible risks and discomforts related to the procedures to provide informed written consent and completed the physical activity readiness questionnaire (PAR-Q) (45). The study protocol was approved by the institutional review board of the University Hospital of the Ribeirao Preto Medical School, University of São Paulo (process number 13,475/2009). In addition, all ongoing and related trials for this intervention were registered in the Brazilian Clinical Trials Registry (ReBec).

Procedures

Adherence. The PRT protocol adherence was monitored through direct supervision, and data were recorded by physical education professionals through daily reminders and in individual training forms prepared by microcycles (21). The criterion of nonaccession was failure to attend at least 20% or 8 training sessions of the programed training sessions. In the case of consecutive absences (1 week), there was no evolution in the periodization for the following week (skipping sessions), maintaining the overload.

Biochemical Measurements. Serum concentrations of testosterone, androstenedione, and 17-hydroxyprogesterone (17-OHP) were measured using a radioimmunoassay (Immulite1000 Immunoassay System; Siemens). Sex-hormone–binding globulin (SHBG), fasting insulin, and thyroid stimulating hormone (TSH) were assessed using chemiluminescence (Immulite2000 Immunoassay System; Siemens, Santa Ana, CA, USA), and plasma glucose was determined with the glucose oxidase method. Serum concentrations of prolactin, TSH, and 17-OHP were assessed at baseline exclusion of other etiologies for hyperandrogenism. All other characteristics were performed before and after intervention under fasting conditions as previously described (21). The free androgen index (FAI) was determined using total testosterone ($nmol \cdot L^{-1}$)/SHBG ($nmol \cdot L^{-1}$) × 100 (8), and IR was quantified using the homeostatic model

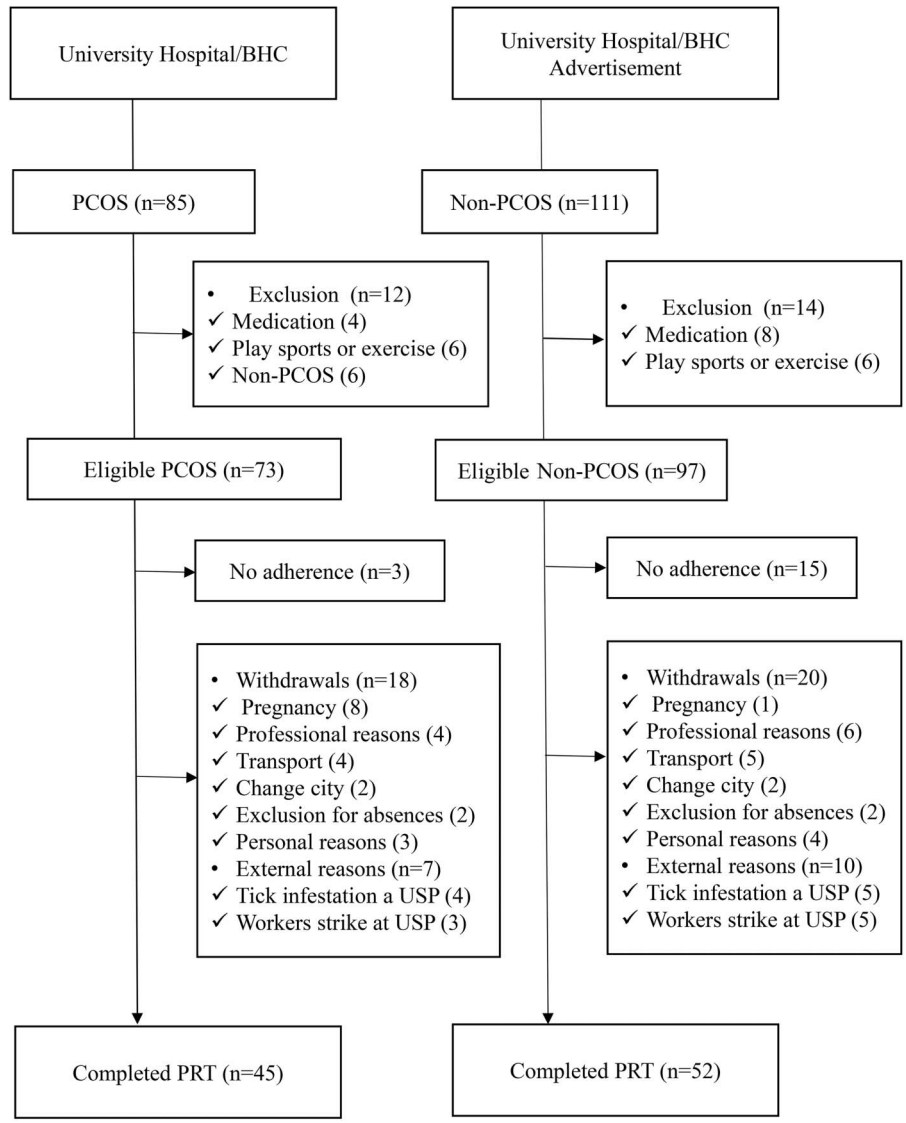

Figure 1. Study flowchart. BHC = basic health clinics; PCOS = polycystic ovary syndrome; PRT = physical resistance training.

assessment (HOMA-IR) [(fasting glycemia in $mg \cdot dl^{-1} \times 0.05551$) $\times$ fasting insulin in $\mu U \cdot ml^{-1}$]/(22.5) (29).

Body Composition and Hypertrophy Indicators. Anthropometric measurements were performed by certified anthropometrists, according to the protocols of the International Society for the Advancement of Kinanthropometry (18). Height and body mass were recorded to the nearest 0.1 cm and 0.5 kg, respectively, using a standing anthropometer and weight scale, incorporated in a balance platform (Filizola, São Paulo, Brazil). A nonelastic flexible measuring tape was used to measure upper arm and thigh circumferences. Skinfold thickness measurements were performed at standard sites (the biceps, triceps, subscapular, suprailiac, and thigh) using Sannyskinfold calipers (Sanny, Brazil). The skinfold measurements were used in age and sex equations to determine body density (19). Body density was used to estimate body fat percentage (%BF) as per the Siri (43) equation: % BF = (495/body density) −450. Fat mass was calculated by the transformation of %BF and the fat mass: fat mass = (body mass [kg] $\times$ %BF)/100. The absolute values of skinfolds or the sum of 4 skinfold thickness ($\sum$4SF) (trunk [subscapular and suprailiac], upper limb [triceps], and lower limb [thigh]) were calculated as an indicator of subcutaneous adipose tissue. The average of 3 measurements was used in the analysis. The same investigator made all the measurements.

Table 1. Biochemical features, body composition, hypertrophy indicators, and muscle strength in PCOS and non-PCOS.*

	PCOS		Non-PCOS		Non-PCOS − PCOS		M0–M2	
	M0	M2	M0	M2	M0	M2	PCOS	Non-PCOS
	Mean (*SD*)	Mean (*SD*)	Mean (*SD*)	Mean (*SD*)	Estimated difference (95% CI)	Estimated difference (95% CI)	Estimated difference (95% CI)	Estimated difference (95% CI)
Testosterone ($ng \cdot dl^{-1}$)	90.2 (35.2)	72.8 (24.4)	74.4 (29.4)	62.6 (22.0)	−13.6 (−25.6 to −1.5)†	−8.65 (−20.6–3.3)	17.1 (8.5–25.6)§	12.2 (4.6–19.7)‖
Androstenedione ($ng \cdot dl^{-1}$)	120.8 (43.6)	139.2 (54.7)	98.9 (32.6)	111.3 (33.4)	−18.2 (−36.0 to −0.8)†	−26.3 (−43.8–8.9)‡	−21.0 (−36.0 to −6.1)§	−13.0 (−26.3–0.4)
SHBG ($nmol \cdot L^{-1}$)	55.0 (37.8)	43.8 (24.4)	63.0 (35.7)	57.7 (35.5)	0.0 (−0.1–0.2)	0.1 (−0.1–0.3)‡	0.1 (0.0–0.2)§	0.0 (−0.0–0.1)
FAI	8.3 (6.3)	7.6 (5.1)	5.6 (4.6)	5.4 (4.6)	−1.8 (−3.9–0.2)	−1.2 (−3.2–0.8)	0.9 (−0.0–1.9)	0.3 (−0.5–1.2)
Fasting glucose ($mg \cdot dl^{-1}$)	96.2 (16.4)	91.1 (17.7)	95.7 (17.5)	90.6 (11.5)	3.4 (−2.7–9.5)	4.7 (−1.3–10.7)	6.7 (1.5–11.9)§	5.4 (0.69–10.1)‖
Insulin ($\mu IU \cdot ml^{-1}$)	9.2 (6.9)	10.1 (9.7)	5.2 (4.5)	5.7 (4.3)	−0.3 (−0.5 to −0.0)†	−0.0 (−0.3–0.1)	−0.1 (−0.2–0.0)	0.1 (−0.0–0.2)
HOMA-IR	2.3 (1.9)	2.4 (2.9)	1.2 (1.2)	1.3 (1.0)	−0.4 (−0.8 to −0.1)†	−0.2 (−0.6–0.0)	0.1 (−0.1–0.3)	−0.0 (−0.3–0.1)
Arm muscle area (cm)	23.1 (2.9)	24.2 (2.7)	22.3 (2.7)	23.1 (2.3)	−0.0 (−0.6–0.6)	−0.4 (−1.0–0.2)	1.1 (0.8–1.4)§	0.7 (0.5–1.0)‖
Thigh muscle area (cm)	48.9 (4.0)	50.0 (3.2)	46.9 (3.9)	48.0 (4.0)	−0.8 (−2.0–0.4)	−0.1 (−1.3–1.0)	1.2 (0.6–1.7)§	1.9 (1.4–2.4)‖
$\sum$4SF (mm)	117.6 (33.6)	107.0 (30.8)	105.3 (37.4)	93.7 (33.6)	−2.5 (−9.7–4.6)	−4.0 (−11.1–3.2)	−9.6 (−11.9 to −7.2)§	−11.0 (−13.0 to −9.0)‖
Body fat percentage (%)	32.8 (7.0)	29.6 (6.6)	30.9 (8.0)	28.2 (7.6)	0.1 (−1.6–1.9)	0.4 (−1.3–2.2)	−3.0 (−3.5 to −2.4)§	−2.7 (−3.2 to −2.1)‖
Body fat mass (kg)	24.8 (9.7)	22.2 (9.0)	22.0 (10.2)	20.9 (9.3)	0.6 (−0.7–1.9)	1.0 (−0.3–2.3)	−2.3 (−2.7 to −1.9)§	−1.9 (−2.2 to −1.5)‖
Lean body mass (kg)	48.3 (7.1)	50.3 (7.0)	46.0 (6.3)	47.8 (6.4)	0.3 (−1.4–2.0)	−0.1 (−1.8–1.6)	2.3 (2.0–2.8)§	2.0 (1.6–2.3)‖
Chest press (kg)	30.9 (5.3)	39.3 (5.4)	29.2 (5.6)	36.8 (6.6)	−1.3 (−3.8–1.2)	−2.5 (−5.0 to −0.0)‡	8.7 (7.8–9.6)§	7.5 (6.7–8.3)‖
Leg extension (kg)	26.6 (5.5)	36.2 (7.3)	24.7 (4.6)	33.6 (6.0)	−1.5 (−4.1–1.0)	−2.5 (−5.0 to −0.0)‡	9.9 (8.6–11.2)§	8.9 (7.7–10.0)‖
Arm curl (kg)	18.0 (3.2)	22.1 (3.5)	17.5 (3.1)	21.1 (3.5)	−0.8 (−2.2–0.6)	−1.0 (−2.5–0.3)	3.8 (3.3–4.4)§	3.5 (3.3–4.4)‖

*Data are presented as mean (*SD*) and CI (confidence interval). PCOS = polycystic ovary syndrome; M0 = baseline; M2 = 16-week follow-up; SHBG = sex-hormone–binding globulin; FAI = free androgen index; HOMA-IR = homeostasis model assessment–insulin resistance. Same letters represent statistical differences ($p < 0.05$).
†PCOS versus non-PCOS: Baseline.
‡PCOS versus non-PCOS: After.
§Baseline versus after: PCOS group.
‖Baseline versus after: Non-PCOS group.

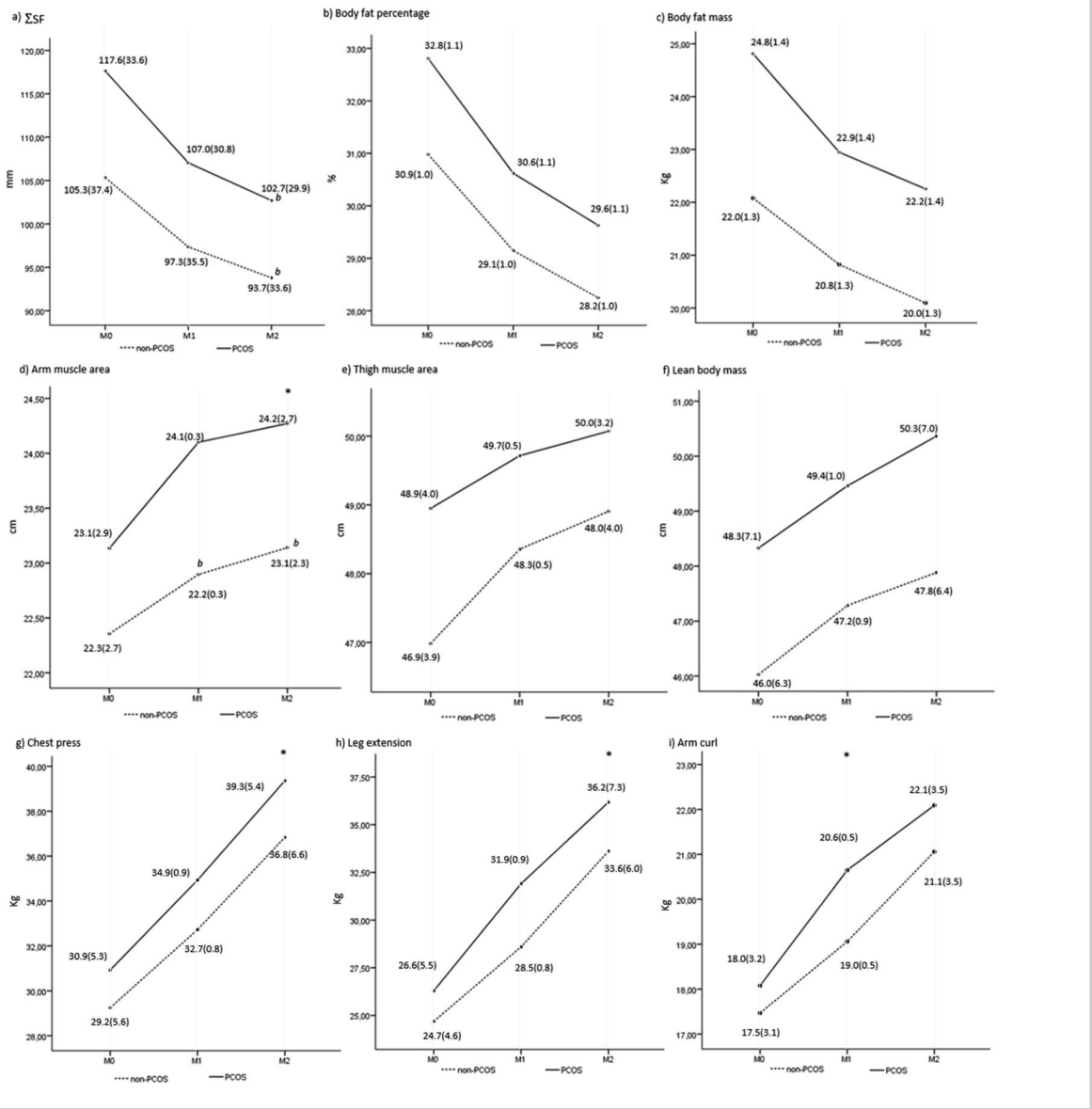

Figure 2. Mean between the stages of study of body composition, hypertrophy indicators, and muscle strength–between group. PCOS = polycystic ovary syndrome; M0 = baseline; M1 = halfway through the intervention; M2 = 16-week follow-up; kg = kilograms, cm = centimeters. *$p < 0.05$.

The hypertrophy indicators were represented by LBM and regional lean mass or corrected circumference, arm muscle area, and thigh muscle area. Lean body mass was determined by fractionation of the body mass into 2 components: LBM (kg) = body mass (kg) − fat mass (kg). The muscular circumference of the dominant limbs was calculated from the limb circumference (cm) obtained at the level corresponding to skinfold measurements, with the skinfold measured multiplied by π. The transformed skinfold measurement was subtracted from the limb circumference to obtain the muscular circumference, as follows: circumference $_{\text{muscular}}$ (cm) = circumference$_{\text{limb}}$ (cm) − π × skinfold (cm) (27).

Muscular Strength. Participants completed a familiarization protocol before the 1RM evaluation to observe protocol adaptation and to obtain maximal measures of muscular strength. The 1RM test was used as a noninvasive measurement of muscle strength and was conducted before the start of the program and used to determine initial loads for periodization. Four 1RM measurements, for different muscle groups, were obtained in one session, with a 48-hour rest period between 1RM sets for the same muscle group to avoid muscle damage and possible influences of other exercises on measurements. The initial overload for the 1RM test was based on the load used to perform 3 series of

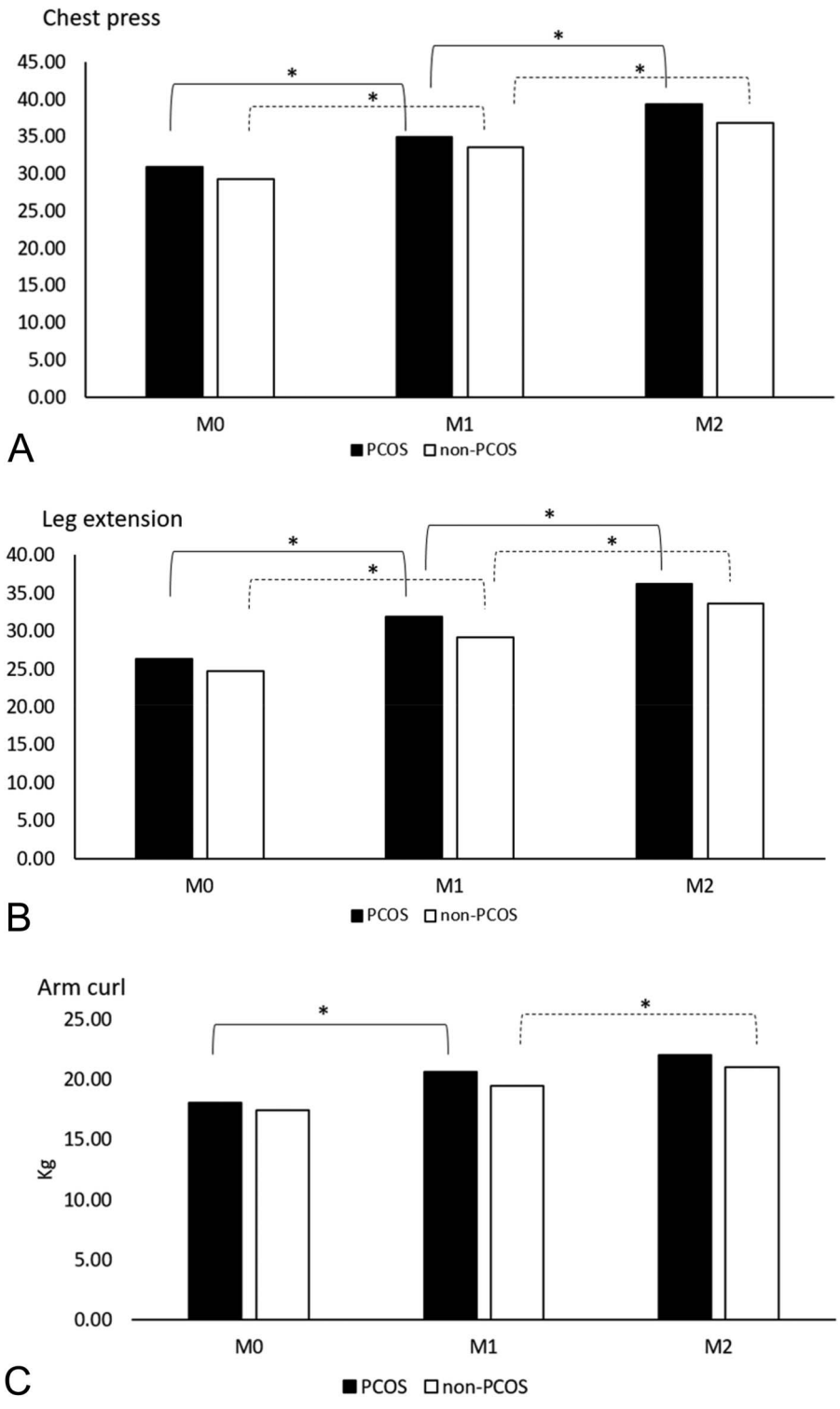

Figure 3. Mean between the stages of study of body composition, hypertrophy indicators, and muscle strength–within group. PCOS = polycystic ovary syndrome; M0 = baseline; M1 = halfway through the intervention; M2 = 16-week follow-up; kg = kilograms. *$p < 0.05$.

10 repetitions during the familiarization period. A method involving overload doubling was arbitrarily used; namely, the participant performed 3 sets of 10 repetitions with 30 kg and the initial overload for the execution of 1RM was 60 kg (40). Stretching exercises were performed for specific muscle groups, immediately followed by a series of 8 repetitions of each exercise with overloading used in the adaptation process and a series of 3 repetitions with a 10% increase in overload performed with a 1-minute interval between each series of 3 repetitions. For each exercise, overload was gradually increased by no more than 10% until the participant was able to perform the exercise with the maximum possible weight. The rest interval between each attempt (1RM) was 3 minutes and 3 attempts were needed to determine the maximum load (30). Testing of the 1RM was performed at the start (baseline or M0) and at week 8 (M1) and week 16 (M2) in both groups, providing 3 values for analysis. Anthropometric measures were obtained at the same time points, again providing 3 values for analysis.

Progressive Resistance Training Protocol. The PRT was based on the linear model of periodization, in which the training intensity is increased in each microcycle (1–4 weeks) and volume is decreased. The linear periodization scheme used was consistent with previous studies from our research group (21,34). The following exercises were included: bench press, leg extension, front latissimus pull-down, leg curl, lateral raise, leg press (45°), triceps pulley, calf leg press, arm curl, and abdominal exercise, executed in alternating segments. Participants were closely and individually supervised by a physical education for all testing and training sessions. As well, participants were instructed not to undertake any regular exercise during the experimental PRT program.

Statistical Analyses

A general linear mixed-model (random and fixed effects) was adjusted to verify the association of covariates (group, time, age, BMI, and HOMA-IR scores) with each measured outcome of strength, body composition, and hypertrophy indicators. Planned orthogonal contrasts were used for between-group comparisons at each time and within-group comparisons across time in these points of measurement. All statistical analyses were performed using SAS 9.0 (SAS Institute, Inc., University of North Carolina, Cary, NC, USA), with a level of significance of 5% ($p = 0.05$).

Results

Participant Recruitment

In total, 170 women, 73 with PCOS (PCOS group) and 97 without PCOS (non-PCOS group) were eligible at baseline. Three participants in the PCOS and 15 in the non-PCOS group did not adhere to the PRT program, and 18 participants in the PCOS and 20 in the non-PCOS group either violated the protocol or withdrew during the planned period of training, as previously described (21). In total, 97 women, 45 with PCOS (PCOS group) and 52 without PCOS (non-PCOS group), completed the 16-week PRT program, without violation of the training protocol. Adherence to training excluding these was 46.4% for the PCOS group and 53.60% for the non-PCOS group ($p = 0.47$). The study flowchart is shown in Figure 1.

Adverse Events

No adverse events were reported by any study participant.

Baseline Characteristics

There were no differences at baseline between the groups in age (PCOS 28.13 ± 5.45 vs. non-PCOS 29.62 ± 5.28 years, $p = 0.22$), height (PCOS 1.60 ± 0.05 vs. non-PCOS 1.61 ± 0.06 m, $p = 0.65$), BMI (PCOS 28.48 ± 6.02 vs. non-PCOS 26.6 ± 5.77 $kg \cdot m^{-2}$, $p = 0.06$), and body mass (PCOS 73.14 ± 15.63 vs. non-PCOS 68.11 ± 15.47, $p = 0.11$). However, concentrations of testosterone, androstenedione, insulin, and HOMA-IR scores were higher among women in the PCOS than in non-PCOS group (Table 1). Analysis of body composition indicated that women in the PCOS and non-PCOS groups had similar body fat and hypertrophy indicators, as well as the maximum strength test in bench press, leg extension, and arm curl (Table 1).

Progressive Resistance Training Intervention

There were no group differences in body composition with this training protocol at some point evaluation. Hypertrophy indicators, such as arm muscle area, was higher in PCOS group in M2 (Table 1) and no difference was observed in other indicators. An increase in maximum strength was observed on the bench press and leg extension measures in M2 (Table 1) and arm curl in M1 (Figure 2) in the PCOS group.

Regarding body composition, a decrease in $\sum$4SF, fat mass, and %BF was identified at M2 from M0, along with an increase in LBM and hypertrophy indicators in both groups. Improvements in strength were also identified in all test exercises for both groups at M2 (Table 1). However, in PCOS group, the muscle strength increased in all exercises at M1 from M0, and in exercises such as chest press and leg extension, at M2 from M1. In non-PCOS group, the muscle strength increased in chest press and leg extension at M1 from M0, and increased in all exercises at M2 from M1 (Table 1 and Figure 3).

Discussion

We found that the 16-week PRT program improved muscle strength of the trunk and lower limbs in PCOS. We also reinforce the positive effects of PRT on body composition, hypertrophy indicators, and muscle strength, regardless of PCOS status. In addition, periods shorter than 16 weeks of PRT seem to be sufficient for increases in muscle strength, but do not promote changes in body composition in women of reproductive age. Previous studies investigating the effect of exercise in women with PCOS have observed clinically

important outcomes such as changes in body composition, circulating androgens, fasting glycemia, sexual function, depression, and anxiety (4,21,25,34,46,47).

At baseline, women with PCOS showed hyperandrogenism and low insulin sensitivity, consistent with the characteristics of the disease (26). Unlike Thomson et al. (46), we have previously reported that increased muscle strength without intervention of any treatment/exercise in women with PCOS may be associated with BMI in addition to hyperandrogenism (23). Therefore, along with other modifying covariables such as HOMA-IR and age, the BMI was controlled in the adjustments in our statistical modeling. This suggests that the largest increases in muscle strength in trunk and lower body observed in women with PCOS than in those without PCOS after PRT might be related to the prevalent hyperandrogenism at baseline associated with the syndrome.

Interindividual differences such as testosterone concentration and FAI in PCOS women could be indicative of the potential muscle strength gain with an intensive short-term RT (14). Studies in other populations have reported a positive correlation between serum levels of testosterone and muscle strength (7), and when submitted to the same volume and intensity of RT, individuals with a higher level of testosterone achieve greater gains in muscle strength (2). Therefore, it is suggested that the serum concentration of this androgen can be an important indicator of an individual's trainability (14). In addition, the between-group differences in strength gains in our study could not be explained by effects of the total volume of each exercise (28) over the 16-week program, which was comparable between the 2 groups. The gain in muscle strength observed in our study among all women in our study group regardless of PCOS status was expected because it is one of the main adaptations associated with this RT, regardless of sex and age (2,15,36), or PCOS factor (47). Morphological factors, such as the angle of pennation, muscle fiber type composition, fiber length, and physiological cross-sectional area, as well as neural factors, such as recruitment and frequency of discharge of motor units, intramuscular synchronization or coordination, muscle synergies, and co-contraction of the antagonist muscles (13), contribute to the increase in muscle strength with a training stimulus.

We did not find that the baseline higher levels of testosterone in women with PCOS were associated with changes in body composition and hypertrophy indicators after the 16-week PRT program, except for arm muscle area. The possible explanation is that upper body muscles may have more androgen receptors than lower body muscles, because the hypertrophy response of arm muscles is greater in men than that in women (20). However, previous reports indicate that the mechanisms of resistance-exercise–mediated muscle hypertrophy are solely dependent on intrinsic processes of increases in muscle work, which may not be influenced by transient changes in circulating hormones (35).

West et al. (48) demonstrated that healthy women can attain similar gains in muscle hypertrophy with RT as men, despite having an approximately 10-fold lower level of testosterone concentration. In addition, the muscular hypertrophy does not occur uniformly across different muscle groups, which included differences in indicators of muscle hypertrophy, and that it may be related to the dimension of the structures involved in the execution of the movements and the volume of muscle mass recruited by the exercise (1).

Although we were limited to the use anthropometry (skinfold and circumference measures), we did demonstrate that our 16-week PRT program improved body composition and hypertrophy indicators among all women in our study group, regardless of PCOS status, in agreement with other findings in PCOS, though with distinct methodologies of body composition evaluation (5,47). Our findings of a decrease in body fat with a gain in LBM is consistent with previously reported findings (28), after a 12-week RT program in young women (36), and a 12-month PRT program in older women (6). The increase in fat-free mass parallel to the decrease in fat mass without reduction in body mass after strength training in women with PCOS is also in line with our previous data (4). The absence of a reduction in body mass (12) or the weight gain in PCOS group (47) after strength training may be due to this increase in LBM or muscle hypertrophy.

In our study, the gain in muscle strength in both groups was observed in the first 8 weeks of the intervention, whereas an increase in indicators of hypertrophy and LBM was observed after 16 weeks of the training protocol. In sedentary individuals, the initial phase of training is characterized by a predominance of neural adaptations in muscles (42), i.e., it is before the hypertrophic muscular response that demand certain time of training sessions to become evident (38), consistent with our results. Subsequently, at this stage, muscle hypertrophy progressively exerts a greater share of contribution in increases in muscle strength (38). Although there is a tendency to associate muscle strength levels with the size of the cross-sectional area of the muscle, this relationship seems to be true only when the neural adaptations have already been largely manifested. As the effect of neural adaptations decrease over time, the contribution of morphological adaptations increases. Optimization of these parameters to maximize strength gains, including the time required for these adaptations to occur and the magnitude of these changes, remains to be clearly defined, particularly in women.

Data on the endocrine metabolic variables related with characteristics and with the diagnosis of PCOS after 16 weeks of PRT have been discussed previously by our group (21). Nonetheless, we observed a reduction in serum concentration of testosterone and glycemia after 16 weeks of PRT among all participants (PCOS and non-PCOS). No previous information about the basal anabolic hormonal response to RT among women with PCOS is available.

Interestingly, animal studies have linked short-term swimming exercise training to decreased serum androgen concentrations in female sex, suggesting that the therapeutic application of exercise could lead to a reduction in symptoms associated with hyperandrogenism in women with PCOS (39). In other populations, this change in basal testosterone concentration among women who perform RT have also been inconsistent (3,14,36). The reduction of glycemia after RT may be related to increased muscle mass, contributing to decrease in the secretion of insulin, which is necessary to maintain a normal glucose tolerance (33). However, changes in GLUT4 muscle content and in various expressions and activity of insulin signaling proteins are parts of the mechanism behind the improvement in insulin action (16), interesting for PCOS (9).

Some methodological limitations (e.g., methods with greater accuracy such as the quantification of the signal amplitude of the surface electromyography, muscular biopsy, computed tomography, magnetic resonance imaging, or ultrasound) raise questions regarding the efficacy of PRT in stimulating strength and hypertrophy increases. In addition, in this study, we used immunoassays for the measurement of steroid sex hormones, which may not be as sensitive as mass spectrometry (32) for detecting androgen levels in women. However, the linear periodization, with manipulation of volume and intensity of the training to avoid the establishment of a plateau of adaptations (12), seems to have favored the increase in muscle strength among our study participants.

In summary, PRT increases muscular strength among women with PCOS and these changes are related to the intrinsic hyperandrogenism associated with PCOS. In addition, PRT promoted positive changes in body composition and indicators of hypertrophy, in addition to progressive increases in muscle strength, among all participants, regardless of PCOS status. Effects of PRT were more evident in the first 8 weeks of the program due to a reduction in testosterone level and improvements in the hyperandrogenic pattern of PCOS.

Practical Applications

Progressive resistance training can be effective to improve body composition and increase strength in women of reproductive age, regardless of PCOS status (20). We show for the first time that in PCOS specifically, there was a more profound impact in muscle strength after exercise than seen in non-PCOS women. Progressive resistance training is an anabolic exercise modality that can improve metabolic and endocrine disorders and reduce body fat, as well as improve skeletal muscle mass and strength muscle (12). Because PCOS shares common characteristic with various metabolic and endocrine disorders (25), similar results of PRT would be expected in women without PCOS, but with other metabolic complications. Despite the effectiveness of PRT in PCOS women, this exercise is not currently recommended or routinely prescribed in the treatment of this syndrome (9). Our data may reinforce the use of the PRT in PCOS lifestyle improvements and encourage future investigations comparing different variables of training to better understand the functional, morphological, and endocrine metabolic changes that could be promoted by PRT in this disease.

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