

Circuit resistance training in sedentary women: body composition and serum cytokine levels

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Abstract: Exercise can generate alterations in body composition and modulate the immune system. The objective of this study was to verify whether a circuit resistance training (CRT) protocol can increase lean body mass (LM), and reduce fat body mass (FM) and the percent of FM (%FM) of sedentary women, without inducing inflammatory responses, indicated by serum cytokine levels. The initial hypothesis was that CRT would improve body composition, without changing serum cytokine levels. The study consisted of 14 healthy, sedentary women, aged 33–45 years (mean \pm SD, 40.23 \pm 3.98 years), with a normal body mass index. They participated in 3 sessions per week of CRT, which included 2 rounds in 9 stations with 1 set of 8–12 repetition maximum at each station, for 10 weeks. During the 10-week CRT period, participants maintained their pretraining nutritional standard. Body composition was analysed with dual-energy X-ray absorptiometry both pre- and post-training. Blood samples were collected after 96 h of rest pre- and post-training, and 5 min, 24 h, and 48 h after the second and last training sessions to measure serum cytokine levels by flow cytometry. The nutritional standard was accompanied throughout the study period with 24-h dietary recall. Increases in LM (35.937 \pm 4.926 to 39.130 \pm 4.950 kg) and decreases in FM (21.911 \pm 8.150 to 17.824 \pm 4.235 kg) and %FM (37.10 \pm 10.84 to 31.19 \pm 6.06), without concurrent changes in serum cytokine levels, and in the nutritional standard (α = 0.05). The proposed CRT improved body composition and did not induce any changes in serum cytokine levels characteristic of the inflammatory response in women.

Key words: circuit resistance training, body composition, DXA, dietary recall, cytokines, inflammation, flow cytometry, women.

Résumé : L'exercice physique peut susciter des modifications de la composition corporelle et moduler le système immunitaire. Cette étude examine si un programme de circuits d'entraînement contre résistance (CRT) augmente la masse maigre corporelle (LM), diminue la masse adipeuse corporelle (FM) ainsi que le pourcentage de gras corporel (% FM) chez les femmes sédentaires, et ce, sans provoquer de réponses inflammatoires; lesquelles sont évaluées par la concentration sérique des cytokines. Selon l'hypothèse initiale, un CRT améliore la composition corporelle sans modifier la concentration sérique des cytokines. Quatorze femmes âgées de 33 à 45 ans (moyenne \pm écart type : 40,23 \pm 3,98 ans) sédentaires, en bonne santé et ayant un indice de masse corporelle normal participent à cette étude. Parcouru à 2 reprises, le circuit d'exercices comporte 9 stations constituées d'exercices de musculation répétés au maximum 8 à 12 fois (RM) à raison de 3 séances par semaine durant 10 semaines. Au cours du programme d'entraînement, on maintient le même apport alimentaire standard qu'au début du programme. Avant et après le programme d'entraînement, on évalue la composition corporelle par absorptiométrie à rayons-X en double énergie (DXA). On prélève des échantillons sanguins au repos, avant le début du programme d'entraînement, et 96 h après la fin du programme d'entraînement, puis 5 min, 24 h, 48 h de récupération après la deuxième et la dernière séance d'entraînement pour mesurer la concentration sérique des cytokines par cytométrie en flux. On vérifie tout au long de l'étude l'apport alimentaire standard au moyen de la méthode du rappel de la consommation des 24 dernières heures. On observe une augmentation de la LM (de 35,937 \pm 4,926 à 39,130 \pm 4,950 kg) et une diminution de la FM (de 21,911 \pm 8,150 à 17,824 \pm 4,235 kg) et du % FM (de 37,10 \pm 10,84 à 31,19 \pm 6,06) sans changements concomitants à la concentration sérique des cytokines et à l'apport alimentaire standard (α = 0,05). Le programme de circuits d'entraînement contre résistance améliore la composition corporelle, mais ne modifie pas la concentration sérique des cytokines, reflet de la réponse inflammatoire chez les femmes.

Mots-clés : circuits d'entraînement contre résistance, composition corporelle, DXA, méthode du rappel de la consommation des 24 dernières heures, cytokines, inflammation, cytométrie en flux, femmes.

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Introduction

Modern technologies have greatly reduced the need to exert force during daily activities (Bird et al. 2005). This sedentary lifestyle predisposes adults to a gradual loss of lean body mass with a reduction in basal metabolism. If this situation is coupled with a hypercaloric diet, a gain of fat mass may occur (Hill and Melanson 1999). An increase in fat body mass is associated with health problems, such as obesity and other chronic degenerative diseases, and with an increase in the general mortality rate (Layman et al. 2005; Pedersen and Saltin 2006; Slentz et al. 2004).

Thus, there is growing interest in studying training protocols that are effective in the control and improvement of body composition. Among these, the popularity of resistance training protocols has grown immensely since the late 1970s (Deschenes and Kraemer 2002). This exercise modality normally stimulates a higher rate of muscular protein synthesis, compared with its degradation, resulting in muscle hypertrophy (Toigo and Boutellier 2006). Additionally, promoting an increase in bone and connective tissue (increasing lean body mass) and a decrease in fat mass induces an increase in the strength, power, and endurance of muscle, which may improve quality of life (Bird et al. 2005; Deschenes and Kraemer 2002; Kraemer et al. 2002; Marx et al. 2001; Haskell et al. 2007).

Data in the literature have shown a relationship in which regular physical activities with moderate intensity may benefit the immune response and the anti-inflammatory effect (Petersen and Pedersen 2005). In contrast, exhaustive exercise without adequate rest may generate exacerbated inflammatory responses, similar to those observed in situations like surgery, trauma, infection, or sepsis (Moldoveanu et al. 2001; Nieman 1997). Such responses, albeit transient, can result in immunosuppression, causing individuals to become more vulnerable to opportunistic infections, primarily in the upper airway tract (Nieman 1997), as occurs in overtraining syndrome (Smith 2004).

Cytokines are soluble proteins or glycoproteins of low molecular weight that are produced by and mediate communication between and within immune and nonimmune cells, and organs and organ systems throughout the body (Moldoveanu et al. 2001; Nieman et al. 2001). When produced in the muscular cells, they are called myokines (Pedersen and Febbraio 2005). The local response to an infection or tissue injury involves the production of cytokines, which are released at the site of inflammation. These cytokines facilitate an influx of lymphocytes, neutrophils, monocytes, and other cells, and these cells participate in the clearance of the antigen and the healing of the tissue (Pedersen and Hoffman-Goetz 2000). Thus, cytokines are being used as cellular and systemic indicators of inflammatory responses and the repair of injury that may be caused by physical effort (Moldoveanu et al. 2001; Nieman et al. 2001; Tomiya et al. 2004; Willoughby et al. 2003).

However, most of the data in the literature concerning inflammatory cytokines were derived from acute aerobic pro-

ocols; few were derived from resistance training (Moldoveanu et al. 2001). Among the available studies, investigators have used resistance exercise protocols as a means of inducing the inflammation, due to microtraumas, generated by the prioritization of the eccentric phases of movements (Moldoveanu et al. 2001; Tomiya et al. 2004; Willoughby et al. 2003).

The purpose of this research was to evaluate whether a 10-week circuit resistance training (CRT) protocol would increase lean body mass, and reduce fat body mass and the percent of fat body mass of adult women who are otherwise healthy and previously sedentary, without inducing inflammatory responses, as indicated by serum cytokine levels. The initial hypothesis was that the CRT would alter the body composition without changing serum cytokine levels.

Materials and methods

Subjects

Study participants were women aged 33 to 45 years who were in menopause, healthy (determined by medical consultation and clinical exams), had a normal body mass index (18.5 to 24.9 kg·m⁻²), were nonsmokers, were sedentary (did not participate in any physical activity), and who presented with peak oxygen uptake ($\dot{V}O_{2\text{ peak}}$) or maximum oxygen uptake ($\dot{V}O_{2\text{ max}}$) below 30 mL O₂·kg⁻¹·min⁻¹ (below the values considered normal for women in this age group) (Fletcher et al. 2001). Values of $\dot{V}O_{2\text{ peak}}$ were determined with a gas analyser (Aerograph VO2000, Medical Graphics Corp., St. Paul, Minn.) during a crescent-type ergospirometric test on a cycle ergometer (Ergo Cycle 167, Ergo-Fit, Pirmasens, Germany).

Fourteen subjects with the profile described above and characteristics listed in Table 1 participated in this study. This research was approved by the Ethics in Human Subjects Research Committee of the Federal University of São Carlos (São Carlos-SP, Brazil), in agreement with the ethical principles for medical research involving human subjects of the World Medical Association Declaration of Helsinki, and with the ethical, legal, and regulatory norms and standards for research involving human subjects in Brazil.

A Statement of Consent was read, understood, and freely signed by the subjects to participate in the research; they had the right to withdraw consent to participate at any time without reprisal.

Evaluation and maintenance of nutritional standards

Because a possible alteration in the nutritional standard of the subjects during the experimental period could directly interfere with body composition data, the nutritional standard of each subject was determined at the beginning of the study with an alimentary investigation that was conducted 3 days a week time (2 weekdays and 1 weekend day). DietWin software, version 2.0.23, was used to analyse the following nutritional variables: total caloric intake, caloric intake/body mass ratio, carbohydrate intake/body mass ratio,

Table 1. Subject characteristics.

Variable	Pretraining period, mean±SD (95% CI)	Post-training period, mean±SD (95% CI)
Age (y)	40.220±3.990 (37.910–42.520)	40.450±3.990 (38.140–42.750)
Height (m)	1.636±0.067 (1.598–1.675)	1.636±0.067 (1.598–1.675)
Body mass (kg)	57.848±7.796 (53.347–62.350)	56.955±6.284 (53.327–60.583)
Body mass index (kg·m ⁻²)	21.530±1.770 (20.510–22.550)	21.230±1.460 (20.390–22.070)
$\dot{V}O_{2\text{ peak}}$ (mL·kg ⁻¹ ·min ⁻¹)	27.710±3.760 (25.540–29.880)	26.630±4.330 (24.130–29.130)

Note: $n = 14$ subjects. Student's t test was used to compare the pre- and post-training samples with distributions normal. $\dot{V}O_{2\text{ peak}}$, peak oxygen uptake; CI, confidence interval.

protein intake/body mass ratio, lipid intake/body mass ratio, percent of total caloric intake derived from carbohydrates, percent of total caloric intake derived from protein, and percent of total caloric intake derived from lipids. To isolate the effect of training on changes in body composition, subjects were encouraged to maintain their pretraining nutritional standards. The nutritional standards of the subjects were accompanied throughout the study period with a 24-h dietary recall.

Analysis of body composition

Body composition analysis was conducted before and after the training period for each subject, using dual-energy X-ray absorptiometry (DPX Plus No. 6243, Lunar, version 4.7e), with an in vivo variation coefficient of 0.9%–1.1%. The following variables were evaluated: body mass, lean body mass, fat body mass, percent of fat body mass, body bone mineral content, trunk fat mass, trunk lean mass, percent of trunk fat mass, upper limb fat mass, upper limb lean mass, percent of upper limb fat mass, lower limb fat mass, lower limb lean mass, and percent of lower limb fat mass.

Training protocol

The circuit of resistance exercises consisted of 9 stations, completed in the following order:

1. Smith-machine squat.
2. Wide-grip pulldown to front.
3. Bench press in the guided bar.
4. 45° leg press.
5. 1-arm dumbbell row (starting with the nondominant arm).
6. Incline-bench dumbbell press.
7. Leg curl.
8. Bar upright row.
9. Pulldown cable crunch.

Because the subjects were not accustomed to resistance training, most of the exercises were conducted with machinery that promotes greater security and joint stability than free loads. Personal coaches were present during training to track the subjects. At the beginning of each training session, subjects performed a warm-up standard, consisting of 1 set of 15 to 20 repetitions with light loads and without concentric failure at stations 1, 2, 3, and 7.

The 10-week training period consisted of 3 weekly sessions (Mondays, Wednesdays, and Fridays). In every training session, each subject performed 2 rounds in the circuit, with loads for 1 set of 8 to 12 repetition maximums (RMs) per station. Loads were adjusted to get the maximum effort from the subject in the appropriate execution technique of

movement until concentric failure was reached. One minute intervals were implemented between each station until the end of the 2 rounds.

The subjects completed the repetitions at a comfortable speed, with concentric and eccentric stages of the first repetitions lasting about 1.5 s each, and with an increase in duration in the concentric stage until concentric failure, due to gradual fatigue. The loads were updated when necessary to keep the number of repetitions within the same range of RMs and to provide a progressive overload for maximal muscle fibre recruitment and, consequently, muscle fibre hypertrophy (Kraemer et al. 2002). The total time of each training session did not exceed 40 min.

Analysis of overload of training

The overload of training for each station of CRT (OL) was calculated using the following equation:

$$OL = (nRM \times L)_{\text{round 1}} + (nRM \times L)_{\text{round 2}}$$

where nRM is the number of RMs and L is the load in kg.

Blood samples and serum cytokine levels

In total, 8 blood samples (3 mL) each were collected from the antecubital vein in vacutainers without anticoagulant. Four were collected at the beginning of training — 1 before starting training (pretraining rest period), and with 5 min, 24 h, and 48 h of recovery after the second training session; and 4 were collected at the end of training — with 5 min, 24 h, 48 h, and 96 h of recovery after the last training session (the 96-h recovery sample was the post-training rest period). Samples were centrifuged at 2400 r·min⁻¹ at 4 °C for 20 min, and the serum was stored at –80 °C until the day of the analysis. To prevent interference in the serum cytokine analysis, subjects were asked to avoid using any type of substance with anti-inflammatory action at least 72 h before the blood sample collection.

The serum levels of cytokine interleukin (IL)-1 β , IL-6, IL-8, IL-10, and IL-12p70, and tumor necrosis factor (TNF) were determined by flow cytometry, with 50 μ L of each serum blood sample used for reading in a BD FACSCanto-Flow Cytometer, using BD CBA Human Inflammation Kit reagents produced by BD Biosciences, with the following detection limits: 7.2 pg·mL⁻¹ for IL-1 β ; 2.5 pg·mL⁻¹ for IL-6; 3.6 pg·mL⁻¹ for IL-8; 3.3 pg·mL⁻¹ for IL-10; and 1.9 pg·mL⁻¹ for IL-12p70; and 3.7 pg·mL⁻¹ for TNF.

Statistical analysis

An error α of 0.05 was adopted, and the significance level was set at $p < 0.05$. Statistica 6.0 software was used for all

statistical analyses. Tests of normality (Shapiro–Wilk test) and homocedasticity (Levene test) of distribution were applied to all sample variable analyses. The samples with normal and homocedastic distributions were compared using parametric methods (paired Student's *t* test or 1-way analysis of variance with Tukey's post hoc tests), and the samples with abnormal and (or) heterocedastic distributions were compared with nonparametric methods (paired Wilcoxon's test or Friedman with Tukey's post hoc tests).

Results

Nutritional standards

None of the analysed variables of nutritional standards showed significant alterations among the collected samples (Table 2).

Body composition

As originally hypothesized, Table 3 shows that the adopted CRT protocol with the maintenance of nutritional standards altered the body composition of these women, leading to a reduction in fat body mass and an increase in lean body mass, not only in the whole body but also in isolated body segments. This led to a reduction in the percentage of body fat, overall and by segment, without changing total body mass. However, the bone mineral content of subjects did not change in the 10 weeks of training.

Progressive overload of training

The data shown in Table 4 confirm a significant increase in overload training, and consequently an important increase in tensional stress.

Cytokines of the inflammatory response

As originally hypothesized, in all tests conducted, no significant alterations were observed in the serum levels of the 6 cytokines measured, either in acute comparisons between the different times evaluated at the beginning and the end of training (Table 5), or in comparisons between the respective moments at the beginning and end of the training period (Table 6).

Discussion

Nutritional standards

The maintenance of all variables of nutritional standards analysed during the experimental period allows for the correlation of alterations in body composition to the adopted CRT protocol.

Body composition

Despite the subjects having healthy body mass indexes, they had high percentages of average fat body mass (37.10%). This shows that an abnormally large proportion of the body mass consists of fat, together with a little lean body mass, probably because of previous physical inactivity.

Significant and beneficial results for body composition are presented in Table 3. In addition to reducing the percentage of fat mass in whole body and in all body segments evaluated, the importance of reducing more than 1.5 kg of fat mass trunk in just 10 weeks is emphasized, because the

Table 2. Results of nutritional standards.

Variable	Pretraining	Week 5 of training	Week 10 of training
Total caloric intake, kcal ^a	1887.90±544.39 (1573.59–2202.22)	1558.71±470.59 (1287.00–1830.42)	1646.36±501.59 (1356.75–1935.97)
% total caloric intake derived from carbohydrates ^a	51.58±7.23 (47.40–55.75)	49.14±6.01 (45.67–52.62)	55.27±7.01 (51.22–59.32)
% total caloric intake derived from protein ^a	18.25±3.43 (16.27–20.23)	21.33±2.48 (19.90–22.76)	18.50±4.18 (16.08–20.91)
% total caloric intake derived from lipids ^a	30.17±5.20 (27.17–33.17)	29.53±6.50 (25.77–33.28)	26.23±7.01 (22.18–30.28)
Caloric intake/body mass ratio, kcal·kg ^{-1b}	32.87±8.67 (27.87–37.88)	—	28.61±7.09 (24.52–32.71)
Carbohydrate intake/body mass ratio, g·kg ^{-1b}	4.24±1.28 (3.51–4.98)	—	3.95±1.06 (3.34–4.56)
Protein intake/body mass ratio, g·kg ^{-1b}	1.48±0.42 (1.24–1.72)	—	1.31±0.38 (1.09–1.53)
Lipid intake/body mass ratio, g·kg ^{-1c}	0.94±0.38 (0.63–1.68)	—	0.79±0.33 (0.40–1.64)

Note: *n* = 14 subjects. The intake/body mass ratio in week 5 of training was not calculated because dual-energy X-ray absorptiometry was not performed to obtain the body mass.

^aFor samples with normal and homocedastic distributions, 1-way analysis of variance with Tukey's post hoc test was used to compare the 3 samples (pretraining, week 5, week 10 — the final week of training) for total caloric intake and the percent of macronutrients; values are given as means ± SD (95% confidence interval (CI)).

^bFor samples with normal distributions, paired Student's *t* tests were used. Values are given as means ± SD and (95% CI).

^cFor samples with abnormal distributions, paired Wilcoxon's test was used. Values are presented as median ± SD (minimum–maximum).

Table 3. Pre- and post-training body composition.

Variable	Pretraining period	Post-training period	<i>p</i>
Body mass (kg) ^a	57.848±7.796 (53.347–62.350)	56.955±6.284 (53.327–60.583)	0.3703
Fat body mass (kg) ^a	21.911±8.150 (17.205–26.616)	17.824±4.235 (15.379–20.269) ^b	0.0213
Lean body mass (kg) ^a	35.937±4.926 (33.093–38.782)	39.130±4.950 (36.272–41.988) ^b	0.0006
Bone mineral content (kg) ^a	2.500±0.346 (2.300–2.700)	2.489±0.346 (2.290–2.689)	0.3591
% body fat mass ^a	37.10±10.84 (30.84–43.36)	31.19±6.06 (27.69–34.68) ^b	0.0142
Trunk fat mass (kg) ^a	8.859±2.999 (7.128–10.591)	7.327±1.920 (6.219–8.436) ^b	0.0171
Trunk lean mass (kg) ^a	16.764±2.342 (15.412–18.117)	17.809±1.861 (16.734–18.884) ^b	0.0365
% trunk fat mass ^c	36.50±10.31 (13.10–46.90)	31.30±6.88 (11.60–35.30) ^d	0.0219
Upper limb fat mass (kg) ^a	2.301±1.169 (1.626–2.976)	1.494±0.469 (1.223–1.765) ^b	0.0053
Upper limb lean mass (kg) ^a	4.199±0.727 (3.779–4.619)	4.375±0.822 (3.900–4.849) ^b	0.0198
% upper limb fat mass ^a	33.74±13.06 (26.20–41.27)	25.39±6.81 (21.46–29.33) ^b	0.0056
Lower limb fat mass (kg) ^a	9.221±3.776 (7.041–11.401)	7.735±2.124 (6.509–8.961)	0.0516
Lower limbs lean mass (kg) ^a	12.773±2.030 (11.601–13.945)	14.538±2.409 (13.147–15.929) ^b	0.0001
% lower limb fat mass ^a	40.69±10.87 (34.42–46.97)	34.50±5.90 (31.09–37.91) ^b	0.0130

Note: *n* = 14 subjects.

^aFor samples with normal distributions normal, values are means ± SD (95% confidence interval).

^b*p* < 0.05 using paired Student's *t* test, pre- vs. post-training.

^cFor samples with abnormal distributions, values are median ± SD (minimum–maximum).

^d*p* < 0.05 using paired Wilcoxon's test, pre- vs. post-training.

Table 4. Results of overload for the 9 stations at the beginning and end of the circuit resistance training period.

Training station	Overload		<i>p</i>
	Second training session	Last training session	
Smith-machine squat ^a	1069.43±255.98 (921.63–1217.22)	1383.36±351.49 (1180.41–1586.30) ^b	0.0008
Wide-grip pulldown to front ^a	585.71±157.84 (494.58–676.85)	742.29±173.29 (642.23–842.34) ^b	0.0007
Bench press in the guided bar ^c	374.00±104.07 (252.00–624.00)	448.50±77.12 (391.00–660.00) ^d	0.0110
45° leg press ^a	1952.50±626.84 (1590.57–2314.43)	2893.93±547.33 (2577.91–3209.95) ^b	0.0004
1-arm dumbbell row ^c	274.00±39.98 (190.00–336.00)	254.00±50.08 (210.00–352.00)	0.4326
Incline-bench dumbbell press ^a	152.07±41.78 (127.95–176.19)	192.57±28.75 (175.97–209.17) ^b	0.0050
Leg curl ^a	299.79±105.20 (239.05–360.52)	365.21±104.56 (304.84–425.58) ^b	0.0169
Bar upright row ^a	311.71±86.14 (261.98–361.45)	325.29±66.82 (286.70–363.87)	0.4361
Pulldown cable crunch ^c	472.50±110.12 (370.00–780.00)	840.00±159.54 (525.00–1080.00) ^d	0.0010

Note: *n* = 14 subjects.

^aFor samples with normal distributions normal, values are means ± SD (95% confidence interval).

^b*p* < 0.05 using paired Student's *t* test, pre- vs. post-training.

^cFor samples with abnormal distributions, values are median ± SD (minimum–maximum).

^d*p* < 0.05 using paired Wilcoxon's test, pre- vs. post-training.

accumulation of visceral fat in this region is associated with metabolic disorders like insulin resistance, independent of fat mass total (Ross et al. 2002; Janssen et al. 2002).

The maintenance of the bone mineral content was expected because of the lower plasticity of bone tissue, which probably requires longer periods of training to suffer significant alterations. Thus, the significant gain in lean body mass was largely due to hypertrophy of the muscle and (or) connective tissue, corroborating results in reviews that recognize that the best results for muscle hypertrophy are achieved in programs with loads between 70% and 85% of 1 RM (corresponding to 8–12 RMs), with 3 weekly sessions for beginners, and intervals between sets of 1 to 2 min for a better response of anabolic hormones (Deschenes and Kraemer 2002; Kraemer et al. 2002; Marx et al. 2001; Haskell et al. 2007).

In this CRT protocol was emphasized multiple-joint exercises, which allowed for both the training of more than

1 muscle group at a time and the use of higher loads for greater gains in muscular strength (Kraemer et al. 2002). Furthermore, the increase of overload training (Table 4), verified by the evolution of workload in performing a series of 8 to 12 RMs to concentric failure, produced significant tensional stress, which stimulates a higher rate of muscle protein synthesis than degradation by promoting muscle hypertrophy (Kraemer et al. 2002; Toigo and Boutellier 2006), and, therefore, contributes to the increased lean body mass observed in this study.

Another factor that may have contributed to the significant changes in body composition was the initial sedentary condition of the women, because, in this condition, the adaptive mechanisms mentioned above are expressed more efficiently, resulting in large gains in physical capacity at the beginning of a training.

High-volume resistance-training protocols, performance of multiple sets and multiple exercises in sequence for each

Table 5. Results of acute analysis of serum cytokine levels after training sessions at the beginning and the end of the training period.

Cytokine levels in training sessions (pg·mL ⁻¹)	96 h pre- or 96 h post-training rest		5 min after training session		24 h after training session		48 h after training session	
	Median±SD (minimum–maximum)	CV (no. detectible values; detectible % of total <i>n</i>)	Median±SD (minimum–maximum)	CV (no. detectible values; detectible % of total <i>n</i>)	Median±SD (minimum–maximum)	CV (no. detectible values; detectible % of total <i>n</i>)	Median±SD (minimum–maximum)	CV (no. detectible values; detectible % of total <i>n</i>)
IL-1β								
Second	—	—	—	—	—	—	—	—
Last	—	—	—	—	—	—	—	—
IL-6								
Second	ND	ND	ND	ND	ND	ND	ND	ND
Last	ND	ND	ND	ND	ND	ND	ND	ND
IL-8								
Second ^a	6.27±2.43 (3.60–10.66)	38.78 (9; 64.3%)	6.84±1.86 (4.38–11.58)	26.50 (14; 100.0%)	6.86±1.57 (5.03–10.34)	22.35 (14; 100.0%)	6.15±1.11 (4.32–8.28)	17.64 (14; 100.0%)
Last ^b	6.32±1.61 (3.73–8.34)	26.28 (13; 92.9%)	6.11±2.89 (3.60–11.65)	43.36 (12; 85.7%)	6.12±1.52 (4.11–9.79)	24.35 (14; 100.0%)	5.86±1.49 (3.73–9.44)	25.41 (12; 85.7%)
IL-10								
Second	—	—	—	—	—	—	—	—
Last	—	—	—	—	—	—	—	—
IL-12p70								
Second	—	—	—	—	—	—	—	—
Last	—	—	—	—	—	—	—	—
TNF								
Second	—	—	—	—	—	—	—	—
Last	—	—	—	—	—	—	—	—

Note: Total *n* = 14. Detection level of the kit used are as follows: 7.2 pg·mL⁻¹ for IL-1β; 2.5 pg·mL⁻¹ for IL-6; 3.6 pg·mL⁻¹ for IL-8; 3.3 pg·mL⁻¹ for IL-10; and 1.9 pg·mL⁻¹ for IL-12p70; and 3.7 pg·mL⁻¹ for tumor necrosis factor (TNF). Friedman's test with Tukey's post hoc test were used to compare the different times (rest; 5 min; 24 h; 48 h). CV, coefficient of variation; ND, not determined; —, values below the detection level of the used kit or nondetectable.

^aNumber of detectible values considered for statistical analysis = 9; % of total *n* = 64.3%; *p* = 0.48244.

^bNumber of detectible values considered for statistical analysis = 11; % of total *n* = 78.6%; *p* = 0.53703.

Table 6. Results of serum cytokine levels after training sessions at the beginning and end of the training period.

Cytokine levels in the times of sample collection (pg·mL ⁻¹)	Second session ^a		Last session ^a	
	Median±SD (minimum–maximum)	CV (no. detectible values; detectible % of total <i>n</i>)	Median±SD (minimum–maximum)	CV (no. detectible values; detectible % of total <i>n</i>)
IL-1β				
96 h of rest	—	—	—	—
5 min after training session	—	—	—	—
24 h after training session	—	—	—	—
48 h after training session	—	—	—	—
IL-6				
96 h of rest	ND	ND	ND	ND
5 min after training session ^b	3.43±0.82 (2.81–5.20)	23.37 (7; 50.0%)	2.93±0.68 (2.81–4.42)	20.86 (5; 35.7%)
24 h after training session	ND	ND	ND	ND
48 h after training session	ND	ND	ND	ND
IL-8				
96 h of rest ^c	6.27±2.43 (3.60–10.66)	38.78 (9; 64.3%)	6.32±1.61 (3.73–8.34)	26.28 (13; 92.9%)
5 min after training session ^d	6.84±1.86 (4.38–11.58)	26.50 (14; 100.0%)	6.11±2.89 (3.60–11.65)	43.36 (12; 85.7%)
24 h after training session ^e	6.86±1.57 (5.03–10.34)	22.35 (14; 100.0%)	6.12±1.52 (4.11–9.79)	24.35 (14; 100.0%)
48 h after training session ^f	6.15±1.11 (4.32–8.28)	17.64 (14; 100.0%)	5.86±1.49 (3.73–9.44)	25.41 (12; 85.7%)
IL-10				
96 h of rest	—	—	—	—
5 min after training session	—	—	—	—
24 h after training session	—	—	—	—
48 h after training session	—	—	—	—
IL-12p70				
96 h of rest	—	—	—	—
5 min after training session	—	—	—	—
24 h after training session	—	—	—	—
48 h after training session	—	—	—	—
TNF				
96 h of rest	—	—	—	—
5 min after training session	—	—	—	—
24 h after training session	—	—	—	—
48 h after training session	—	—	—	—

Note: Total *n* = 14. Detection level of the kit used kitare as follows: 7.2 pg·mL⁻¹ for IL-1β; 2.5 pg·mL⁻¹ for IL-6; 3.6 pg·mL⁻¹ for IL-8; 3.3 pg·mL⁻¹ for IL-10; and 1.9 pg·mL⁻¹ for IL-12p70; and 3.7 pg·mL⁻¹ for tumor necrosis factor (TNF). CV, coefficient of variation; ND, not determined; —, values below the detection level of the used kit or nondetectable.

^aPaired Wilcoxon's test was used for comparison of serum cytokine levels between the second vs. last training sessions for each different time.

^bNumber of detectible values considered for statistical analysis = 5; % of total *n* = 35.7%; *p* = 0.1088.

^cNumber of detectible values considered for statistical analysis = 9; % of total *n* = 64.3%; *p* = 0.3139.

^dNumber of detectible values considered for statistical analysis = 12; % of total *n* = 85.7%; *p* = 0.5829.

^eNumber of detectible values considered for statistical analysis = 14; % of total *n* = 100.0%; *p* = 0.0843.

^fNumber of detectible values considered for statistical analysis = 12; % of total *n* = 85.7%; *p* = 0.2860.

muscle group are recommended for the achievement of better muscle hypertrophy, whereas in CRT protocols, which have been recommended for respiratory and cardiovascular improvement, using short intervals of rest (15–30 s) and lighter loads (40%–60% of 1 RM) allows for a higher maximum number of repetitions and aerobic work (Kraemer et al. 2002; Gotshalk et al. 2004; Braith and Stewart 2006). However, our results demonstrate that low-volume circuit training may also have its variable acute training geared toward improvement of body composition, with increased lean body mass (muscle and (or) connective tissue hypertrophy), in a period of 10 weeks, at least in women who were previously sedentary.

Since there was no change in nutritional standards (Table 2), the significant reductions in fat body mass and percentage of fat body mass pretraining observed in this study were due mainly to the greater energy expenditure generated by the training protocol during the training sessions, and to the increased lean body mass observed, which generates a higher resting energy expenditure for its daily maintenance. These data corroborated the results of reviews carried out by Pedersen and Saltin (2006) and Epstein and Goldfield (1999), when they found that physical training increases energy expenditure and induces lipolysis, provided that the energy expenditure is not compensated for by an increase in caloric intake.

Cytokines of the inflammatory response

If physical activity is of sufficient vigour to induce an inflammatory response, there is an increase in the serum levels of the pro-inflammatory cytokines TNF- α and IL-1 β , and of the inflammation-responsive cytokine IL-6, followed by a regulatory increase in the serum levels of the anti-inflammatory cytokine IL-10 and of the pro-inflammatory cytokine inhibitors, such as IL-1ra, sTNF-r1, and sTNF-r2 (Petersen and Pedersen 2005; Moldoveanu et al. 2001; Nieman 1997; Nieman et al. 2001; Pedersen and Hoffman-Goetz 2000; Ostrowski et al. 1999; Fischer 2006). The serum levels of the chemokine IL-8 also increase after strenuous exercise, such as marathon races (Pedersen and Hoffman-Goetz 2000), since its primary functions are to attract neutrophils to the sites of inflammation or infection and to mediate their activation to initiate the clearance and repair of damaged tissue (Petersen and Pedersen 2005; Moldoveanu et al. 2001; Nieman et al. 2001; Ostrowski et al. 1999). These findings suggest that cytokine inhibitors and anti-inflammatory cytokines restrict the magnitude and duration of the inflammatory response to exercise (Pedersen and Hoffman-Goetz 2000; Ostrowski et al. 1999).

Thus, the type of exercise, its intensity, and its duration are key factors in the profile of the response of pro-inflammatory cytokines produced after exercise (e.g., the release of IL-1 appears to be more sensitive to the intensity of exercise, whereas human TNF and IL-6 are more sensitive to its duration) (Moldoveanu et al. 2001; Fischer 2006). Therefore, resistance exercises with emphasis in the eccentric phase, using high loads, have shown changes in the profile of cytokines through the generation of microlesions (Moldoveanu et al. 2001; Paulsen et al. 2005).

In contrast, the CRT adopted in this study allowed the maintenance of low serum cytokine levels, probably because this protocol aimed to improve body composition and to minimize the harmful and inflammatory potential of the training. Thus, the sequence of exercises (alternating muscle group agonists) and the frequency of the CRT (with a minimum recovery period of 48 h between the sessions) were implemented to provide an adequate time for physical recovery and for the adaptation of the subjects, and consequently to prevent the development of chronic inflammation associated with excessive training and insufficient rest and recovery, as occurs in athletes with overtraining syndrome (Smith 2004). Moreover, the low volume of training (4 sets) for any particular muscle group, with a total duration of 2 rounds in the circuit that did not exceed 40 min, was implemented because both the volume and duration of training positively influence the increasing amounts of serum cytokines, including IL-6, which reaches its peak serum level at the end of the exercise or shortly thereafter (Fischer 2006; Brenner et al. 1999), but which, in this research, did not present alterations in the 5 min after the training sessions. The loads corresponding to 8–12 RMs (70%–85% of 1 RM) were adopted to maximize muscle hypertrophy, instead of supra-maximal loads and the predominance of the eccentric phase of movement, which are related to injuries and changes in levels of cytokines (Willoughby et al. 2003).

The maintenance of serum cytokine levels at the pretraining rest period after the second training session (5 min, 24 h, and 48 h of recovery), and also at the post-training period

after the last training session (same point times), demonstrates that these acute sessions did not induce an inflammatory response. These results corroborate the data found by Brenner et al. (1999), which showed no changes in plasma concentrations of IL-6, IL-10, or TNF- α in samples collected immediately, 3 h, 24 h, and 72 h after an acute session of resistance training. The results also agree with those of Simonson (2001), who showed that acute sessions of resistance exercise do not necessarily debilitate the immune function.

Additionally, the maintenance of serum cytokine levels between the pre- and post-training rest samples and between the respective samples after the second and last training sessions demonstrates that 10 weeks of the CRT protocol did not induce an exacerbated inflammatory response or immunosuppression, which occur in exhaustive exercises (Nieman 1997) and in overtraining syndrome (Smith 2004).

Conclusions

The CRT protocol examined here did not elicit the inflammatory response indicated by the serum levels of cytokines, but it did improve the body composition of the subjects by increasing lean body mass (muscle and (or) connective tissue hypertrophy) and reducing fat body mass. These conclusions agree with those of Malm et al. (2000), indicating that it is possible that muscle adaptation occurs in response to physical training without muscle inflammation.

The absence of an additional group for comparison of these results is a limitation of this study; however, this research contributes to the area of resistance training, showing that it is possible to improve the body composition of healthy, sedentary adult women in a short period of time (10 weeks) without inducing inflammatory responses, as evidenced by serum cytokines levels, even in the absence of previous periods of adaptation to training using lighter loads.

In the future, a possible extension of this research could be the study of CRT as a viable alternative for improving the body composition of individuals with any degree of chronic inflammatory disease, such as rheumatism, obesity, diabetes, or cardiopathy.

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