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Effects of resistance training on resistin, leptin, cytokines, and muscle force in elderly post-menopausal women

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Abstract

It may be that resistance exercise can be used to prevent the degenerative processes and inflammation associated with ageing. Thus, the aim of the present study was to evaluate the effects of resistance training on cytokines, leptin, resistin, and muscle strength in post-menopausal women. Thirty-five sedentary women (mean age 63.18 years, $s = 4.8$; height 1.64 m, $s = 0.07$; body mass 57.84 kg, $s = 7.70$) were recruited. The 16 weeks of periodized resistance training consisted of two weekly sessions of three sets of 6–14 repetition maximum. Maximal strength was tested in bench press, 45° leg press, and arm curl. Plasma tumour necrosis factor-α, interleukin-6, interleukin-15, leptin, and resistin were determined by enzyme-linked immunosorbent assay. Maximal strength on all measures was increased after 16 weeks. There were minor or no modifications in tumour necrosis factor-α and interleukin-15. Interleukin-6 was decreased 48 h after compared with baseline and declined after 16 weeks. Leptin decreased 24 h after compared with baseline and was reduced at baseline and 48 h after compared with pre-training. There was a decrease in resistin after 24 and 48 h compared with baseline and a decline in baseline and immediately after levels compared with pre-training. A possible explanation of the results of the present study is a lower production of pro-inflammatory cytokines by the innate immune system. Periodized resistance training seems to be an important intervention to reduce systemic inflammation in this population.

Keywords: *Ageing, inflammation, systemic biomarkers, resistance training*

Introduction

The increasing proportion of elderly women and their increased life expectancy have been raising concerns for researchers and professionals in the health field, especially regarding the physiological changes involved in the post-menopausal period. Approximately 40% of women search for medical care to treat menopause-induced symptoms, including heat waves, night transpiration, vaginal dryness, and sleep disturbances (Nedrow et al., 2006). Another important biological dysfunction in post-menopausal women is the ''senile inflammation'', with a strong temporal relationship between ageing, inflammation, and menopause.

The gradual decrease in oestrogen release with ageing accompanied by the post-menopausal period induces increased concentrations of pro-inflammatory cytokines, including interleukin-6 (IL-6), interleukin-1 (IL-1), and tumour necrosis factor-alpha (TNF-α) (Pfeilschifter, Köditz, Pghol, & Schatz, 2002). Elevated pro-inflammatory cytokines are associated with increased risk of developing several types of cardiovascular disease (Reilly et al., 2005), osteoporosis (Pfeilschifter et al., 2002), diabetes mellitus (Kadoglou et al., 2007), and geriatric cachexia (Yeh et al., 2001) in later life. Several blood biomarkers are used as indicators of systemic inflammation, including interleukin-6, tumour necrosis factor-α, interleukin 1-beta (IL-1β), resistin, leptin, and C-reactive protein (CRP) (Reilly et al., 2005; Stewart et al., 2007; Tilg & Moschen 2006). Despite the known effects of leptin on appetite, it is also considered a pro-inflammatory mediator that activates the synthesis of pro-inflammatory cytokines as tumour necrosis factor-α, interleukin-6, and

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interleukin-12 by macrophages (Tilg & Moschen 2006; Zhao et al., 2005).

Hormone replacement therapy has been shown to decrease interleukin-6, interleukin-1, and tumour necrosis factor-α in healthy post-menopausal women (Cantatore et al., 1995) and prevent the increase of interleukin-6 in plasma in ovariectomized rats (Gregory, Duffner, Faunce, & Kovacs, 2000). However, hormone replacement therapy is not universally accepted, mainly because of contraindications and low adhesion of some patients (Zhang et al., 2007). The aversion by women regarding hormone replacement therapy is associated with collateral effects, and long-term risks of some types of cancers (Olson, Bandera, & Orlow, 2007; Zhang et al., 2007).

Regular exercise can be used as a therapeutic modality to prevent age-associated degenerative processes and increases in systemic markers of inflammation (Fatouros et al., 2005; Greiwe, Cheng, Rubin, Yarasheski, & Semenkovich, 2001). However, with regard to inflammatory markers, the studies are controversial; for example, plasma leptin concentration was unchanged after acute aerobic exercise (Ferguson et al., 2004) and decreased (Nindl et al., 2002) with acute resistance exercise in young adults. Leptin was decreased approximately 24 h after acute resistance exercise, with no chronic training effect (no difference between pre-training and chronic) in type 2 diabetes patients (Kanaley et al., 2001).

Only one study has reported decreased leptin concentrations in older men after various resistance training protocols (Fatouros et al., 2005). Another study found a decrease in resistin and pro-inflammatory cytokines after 16 weeks of aerobic exercise consisting of four 45–60 min sessions per week [50–85% of maximum oxygen consumption ($\dot{V}O_{2max}$)] (Kadoglou et al., 2007). Frail elderly individuals present increased concentrations of tumour necrosis factor-α in the muscle compared with younger correlates, which is associated with muscle protein loss. After 3 months of resistance training, muscle tumour necrosis factor-α concentrations were decreased (Greiwe et al., 2001).

Active exercises that include resistance training can increase muscle strength, skeletal muscle mass, and bone mass (Barry & Carson, 2004). However, there is no consensus on the influence of a programme of resistance training on inflammatory marker levels in healthy and older individuals (Stewart et al., 2007). Recent evidence highlighted the need for further studies on the use of resistance exercise as an intervention in reducing inflammation in elderly individuals (Fatouros et al., 2005; Stewart et al., 2007).

Although previous research has targeted young males and females as well as elderly men, whether resistance training affects inflammatory markers in elderly post-menopausal women has not been addressed. Our hypothesis was that resistance exercise training would induce a decrease in systemic biomarkers of inflammation. Thus, the main objective of the present study was to assess the effects of periodized resistance training on resistin, leptin, cytokines, and muscle strength in older post-menopausal women.

Methods

Participants

Participants were recruited on a voluntary basis from the local community from posters and lectures about the study. Each volunteer underwent a thorough physical examination, which included a medical history, resting and exercise electrocardiogram (Tavel, 2001), blood pressure assessment, and orthopaedic evaluation before the resistance training programme. After completing the clinical examinations, 35 previously sedentary women (mean age 63.18 years, $s = 4.8$; height 1.64 m, $s = 0.07$; body mass 57.84 kg, $s = 7.70$) were selected. The inclusion criteria were: body mass index ≤ 26 kg · m^2, a sedentary lifestyle without any consistent exercise in the previous 6 months, no hormonal reposition, and no manifestation of cardiovascular or pulmonary disease. All participants signed an informed consent document and the study was approved by the Federal University of São Carlos Research Ethics Committee for Human Use (protocol #048/2007). The research procedures were in accordance with guidelines on the use of human participants by the American College of Sports Medicine (2006).

Maximal strength assessments

After 2 weeks of adaptation to the resistance exercise machines and clinical evaluations, one-repetition maximum (1-RM) strength tests were performed. The 1-RM tests were performed on the same day in the following order, with a minimum 10 min rest between tests: free weight barbell bench press, 45° leg press, and standing arm curl (Cybex International, Medway, MA). After a general warm-up (10 min of low-intensity treadmill running), individuals were required to perform eight repetitions at an estimated 50% of 1-RM (according to each participant's capacity), to rest for 1 min, and then perform a further three repetitions at an estimated 70% of 1-RM. After 3 min, subsequent trials were performed for one-repetition with progressively heavier weights until the one-repetition maximum was determined within three attempts, with 3–5 min rest between trials. Standardization of range of motion and performance of the exercises was conducted

according to Brown and Weir (2001). To ensure that the pre-training one-repetition maximum was stable before training began, the one-repetition maximum was determined on three separate days with 2 days' rest between them. A high interclass correlation was found between the second and the third 1-RM trials (barbell bench press, $r = 0.99$; 45° leg press, $r = 0.99$; standing arm curl, $r = 0.99$). The best one-repetition maximum determined from the last two trials was used as the baseline value.

Resistance training

The resistance training was based on the linear periodization or ''classic'' model. In this model, the intensity of the training is increased in each microcycle (1–4 weeks) and the volume is decreased. The numbers of repetitions were reduced (maintaining the minimal zone established for each cycle) as the intensity increased. This periodization scheme was in accordance with our previous research (Prestes, De Lima, Frollini, Donatto, & Conte, 2009). Training loads were monitored in each session, according to the increase in muscle capacity of the participants. Before the start of periodization, participants completed 2 weeks of adaptation to resistance training. During this time, participants performed one exercise for each main muscle group with two sets of 15 repetitions in each exercise to allow for correct execution of movement and familiarization with resistance exercise types.

After the adaptation period, periodization was initiated. The order of the exercises in the training sessions is presented in Table I. In addition, at the end of each training session, three sets of 20–30 repetitions of abdominal crunches were performed. Training lasted for 4 months, with two weekly sessions; for each of the listed exercises, three sets to concentric failure were performed (inability to perform a repetition with the correct movement pattern). The number of repetitions and amount of rest between sets and exercises followed the weekly prescribed intensity, as presented in Table II. The mean duration of each session was 50 min. The duration of each repetition was 3–4 s, including the concentric and eccentric muscle actions. All sessions were supervised by an experienced strength training professional.

Table I. Order of the exercises in the training sessions over a 16-week resistance training programme.

1. Barbell bench press	6. Knee flexion
2. 45° leg press	7. Arm extension
3. Seated row	8. Hip adduction and abduction
4. Knee extension	9. Arm curl
5. Lateral raise	10. Standing calf raise

In the first 4 weeks, three sets of 12–14 RM were performed; from weeks 5–8, three sets of 10–12 RM were performed; from weeks 9–12, three sets of 8–10 RM were performed; and from weeks 13–16, three sets of 6–8 RM were performed. In all weeks, maximal repetitions to concentric failure were performed at the intensities indicated. The loads were monitored in each session. In each training session, three sets were performed, independently of the intensity; the rest interval was followed according to the intensity (Table II) and loads were adjusted to maintain the number of maximum repetitions.

Serum cytokines, leptin, and resistin determination

Blood samples of 3 ml were drawn from the antecubital vein in Vacutainer tubes (Becton Dickinson, Brazil). These samples were centrifuged at 2500 rev · min^{-1} at 4°C for 20 min. Samples were stored in aliquots in Eppendorff tubes at −80°C until analysis. Tumour necrosis factor-α, interleukin-6, interleukin-15, leptin, and resistin were determined by enzyme-linked immunosorbent assay (ELISA), according to the specifications of the manufacturer (Quantikine High Sensitivity Kit, R&D Systems, Minneapolis, MN). The results are presented in picograms per millilitre (pg · ml^{-1}). All samples were determined in duplicate to guarantee the precision of the results. Cytokines and biomarkers were evaluated before, immediately after, 24 and 48 h after the first resistance training session and at the same instances after 4 months of training. The intra-assay coefficient of variation was 3.1–9.7%, the inter-assay coefficient of variation was 6.3–7.0%, and the sensitivity was 0.0086 pg · ml^{-1}.

Statistical analysis

Data are presented as means standard errors of the mean (s_x). Initially, the Shapiro-Wilk test of normality and a test of homoscedasticity (Bartlet criteria) were performed. All variables presented a normal distribution and homoscedasticity, so a repeated-measures analysis of variance (ANOVA) was used. Tukey's *post hoc* test was applied where indicated by ANOVA. To test for significant differences between

Table II. Intensity and rest manipulation throughout a 16-week resistance training programme in elderly post-menopausal women ($n = 35$).

Sets × repetitions	Rest interval between sets and exercises
3 × 12–14 RM	60 s
3 × 10–12 RM	80 s
3 × 8–10 RM	100 s
3 × 6–8 RM	120 s

pre- and post-training 1-RM tests, we used a paired Student's *t*-test. For all analyses, statistical significance was set at $P \leq 0.05$. The software package used for all analyses was Statistica® 6.1 (StatSoft Inc., Tulsa, OK).

Results

Maximal strength

There was a significant increase in barbell bench press ($P = 0.01$), 45° leg press ($P = 0.01$), and standing arm curl ($P = 0.01$) maximal strength comparing the evaluation before with the evaluation after 16 weeks of training (Table III).

Cytokines, leptin and resistin

There were no statistically significant acute or chronic changes in plasma concentrations of tumour necrosis factor-α during the study (Figure 1). In contrast, plasma concentrations of interleukin-6 were acutely decreased after 48 h compared with baseline in the pre-training evaluation ($P = 0.04$). In addition, interleukin-6 was acutely decreased after 48 h compared with immediately after the post-training evaluation ($P = 0.04$). There was a chronic decrease in interleukin-6, evidenced by the difference between baseline values before and after training ($P = 0.01$) (Figure 2). The only change observed in plasma concentrations of interleukin-15 was an acute increase after 48 h compared with baseline in the pre-training evaluation ($P = 0.03$) (Figure 3). In the other periods, no acute or chronic differences were observed for interleukin-15.

Table III. Dynamic maximal strength tests (1-RM) before and after 16 weeks of training (mean ± s_x).

Maximal strength test (1-RM)	Evaluation before training	Evaluation after 16 weeks
Bench press (kg)	31.83 ± 0.80	38.34 ± 1.15*
45° leg press (kg)	172.21 ± 5.57	225.17 ± 7.06*
Arm curl (kg)	20.76 ± 0.45	22.75 ± 0.51*

*Statistically significant ($n = 35$): $P \leq 0.05$.

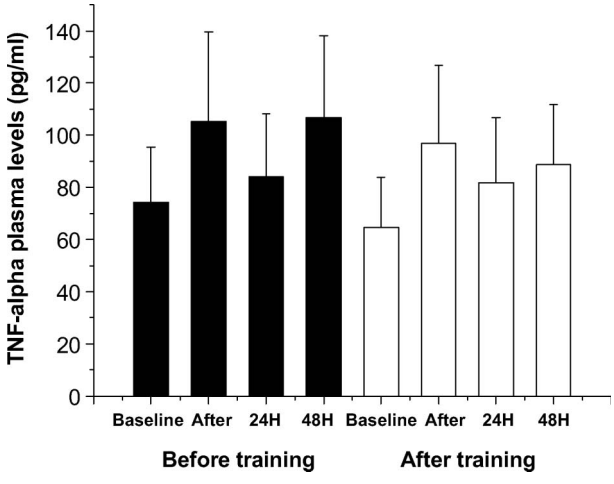

Figure 1. Plasma tumour necrosis factor-alpha (TNF-α) concentration (pg · ml^{-1}). TNF-α was evaluated at baseline, immediately after (After), 24 h (24H), and 48 h after (48H) the first resistance exercise session (black bars), and after 16 weeks of training (white bars). Values are mean and standard error ($n = 35$).

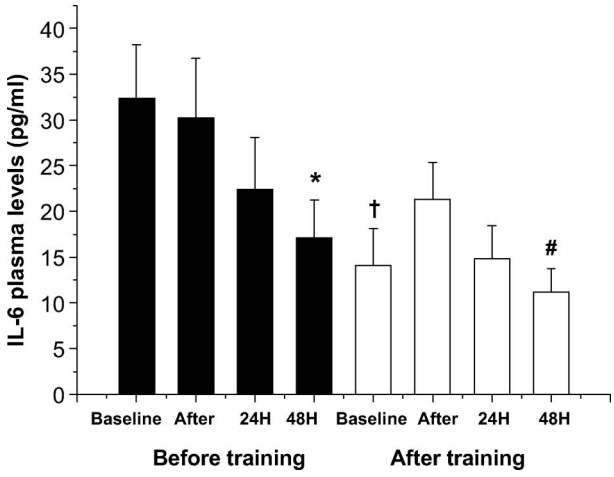

Figure 2. Plasma interleukin-6 (IL-6) concentration (pg · ml^{-1}). IL-6 was evaluated at baseline, immediately after (After), 24 h after (24H), and 48 hs after (48H) the first resistance exercise session (black bars), and after 16 weeks of training (white bars). Values are mean and standard error ($n = 35$). *Statistically significant difference compared with baseline ($P \leq 0.05$). #Statistically significant difference compared with immediately after ($P \leq 0.05$). †Statistically significant difference compared with the same period of evaluation before training ($P \leq 0.05$).

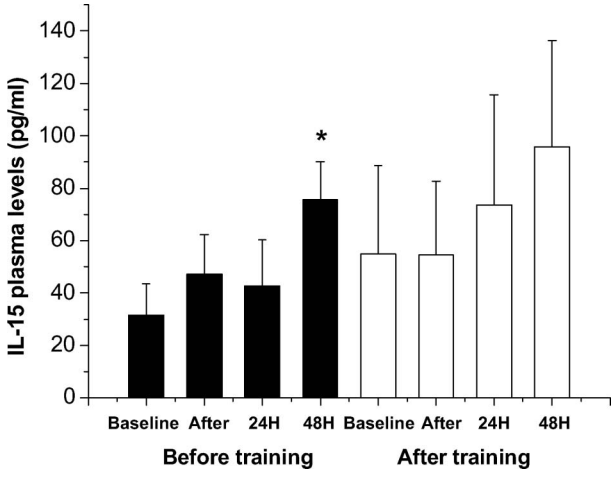

Figure 3. Plasma interleukin-15 (IL-15) concentration (pg · ml^{-1}). IL-15 was evaluated at baseline, immediately after (After), 24 h after (24H), and 48 h after (48H) the first resistance exercise session (black bars), and after 16 weeks of training (white bars). Values are mean and standard error ($n = 35$). *Statistically significant difference compared with baseline ($P \leq 0.05$).

Plasma leptin concentration exhibited a decrease 24 h after the first resistance exercise compared with baseline ($P = 0.04$). However, after 48 h its concentration increased compared with 24 h after in the pre-training evaluation ($P = 0.02$). Both baseline and 48-h values were chronically decreased compared with baseline and 48 h after the 16 weeks of training (both $P = 0.01$) (Figure 4). There was a significant decrease in resistin after 24 and 48 h compared with baseline (both $P = 0.01$) and after 24 h compared with immediately after in the pre-training evaluation ($P = 0.01$). Resistin also exhibited a chronic decrease in baseline and immediately after values in the post- compared with the pre-training evaluation (both $P = 0.01$) (Figure 5).

Discussion

The main findings of the present study were increased maximal strength in both the lower and upper body, markedly decreased plasma concentrations of interleukin-6, leptin, and resistin, with minor or no alterations in interleukin-15 and tumour necrosis factor-α after 16 weeks of resistance training. This is the first study to investigate these important inflammatory markers together in elderly women after resistance training. Thus, our initial hypothesis has been confirmed, since reduced markers of plasma inflammation were observed, together with an increase in muscle force.

Barry and Carson (2004) commented that older adults undertake resistance training programmes to combat substantial declines in muscular strength and power with a view to improving or maintaining functional capabilities. In the present study, there was a significant increase in upper and lower body maximal dynamic strength in elderly women after 16 weeks of periodized resistance training with loads between 6- and 14-repetition maximum.

According to Starkweather (2007), several biochemical markers of inflammation have been used in recent physical activity intervention studies. For example, interleukin-6 and tumour necrosis factor-α are pro-inflammatory cytokines defined as soluble mediators that are released from various cells (macrophage/monocyte lineage). In particular, interleukin-6 is a multi-functional cytokine that plays a pleiotropic role in immune regulation and inflammation, and its over-production with ageing is associated with cardiovascular disease, osteoporosis, rheumatoid arthritis, type II diabetes, and Alzheimer's disease (Kiecolt-Glaser et al., 2003). Elevated interleukin-6 concentrations are also associated with frailty, a decline in functional capacity, decreased muscle strength, and may be used as a predictor of future disability in older adults (Cohen, Pieper, Harris, Rao, & Currie, 1997; Ferrucci et al., 2002).

Plasma interleukin-6 increases during acute exercise in proportion to intensity, duration, and level of fitness. Interleukin-6 has been suggested to be an important factor for the process of muscle repair and cell turnover, as well as for some of the beneficial health effects of exercise (Maggio, Guralnik, Longo, & Ferrucci, 2006). Additionally, interleukin-6 has

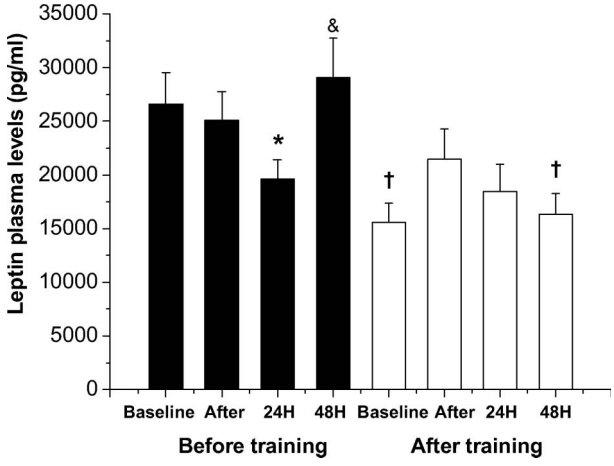

Figure 4. Plasma leptin concentration ($pg \cdot ml^{-1}$). Leptin was evaluated at baseline, immediately after (After), 24 h after (24H), and 48 h after (48H) the first resistance exercise session (black bars), and after 16 weeks of training (white bars). Values are mean and standard error ($n = 35$). *Statistically significant difference compared with baseline ($P \leq 0.05$). &Statistically significant difference compared with 24 h after ($P \leq 0.05$). †Statistically significant difference compared with the same period of evaluation before training ($P \leq 0.05$).

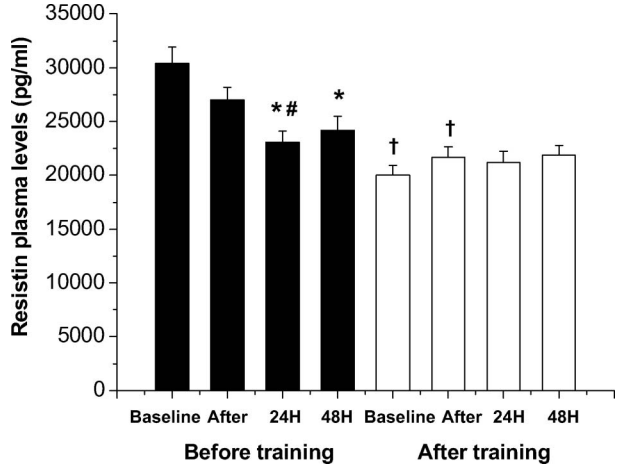

Figure 5. Plasma resistin concentration ($pg \cdot ml^{-1}$). Resistin was evaluated at baseline, immediately after (After), 24 h after (24H), and 48 h after (48H) the first resistance exercise session (black bars), and after 16 weeks of training (white bars). Values are mean and standard error ($n = 35$). *Statistically significant difference compared with baseline ($P \leq 0.05$). #Statistically significant difference compared with immediately after ($P \leq 0.05$). †Statistically significant difference compared with the same period of evaluation before training ($P \leq 0.05$).

been identified as myokine that is produced and released by skeletal muscle during acute exercise (Steensberg et al., 2000). This process is followed by increases in interleukin-1ra, interleukin-10 (anti-inflammatory cytokines), and soluble tumour necrosis factor-α receptor (sTNF-R), which inhibit tumour necrosis factor-α production (Pedersen & Febbraio, 2008). In contrast to these acute effects, chronically exercised individuals tend to have lower levels of interleukin-6 and other inflammatory markers (Elosua et al., 2005).

For instance, older men and women between 60 and 90 years submitted to 30 min of walking, five times per week at 60% of their maximal heart rate for 10 weeks, exhibited significant decreases in serum interleukin-6, and improvements in stress state, mood, and several quality-of-life indices (Starkweather, 2007). A study with 3075 adults aged 70–79 years found that individuals who reported higher levels of physical activity had significantly lower interleukin-6 (Colbert et al., 2004). Literature shows support for either exercise-induced reductions (Castaneda et al., 2004) or unchanged concentrations (White, Castellano, & McCoy, 2006) of plasma/serum proinflammatory cytokines.

However, studies addressing the effects of resistance training in the elderly are sparse and report contrasting results. Bautmans et al. (2005) found a trend for a decrease in concentrations of circulating interleukin-6 after 6 weeks of periodized resistance training in elderly individuals. There were no significant alterations in circulating interleukin-1β, tumour necrosis factor-α, interleukin-2 or interleukin-6 after 12 weeks of progressive resistance training in elderly individuals (Kapasi, Ouslander, Schnelle, Kutner, & Fahey, 2003). In contrast, in the present study there was a marked decrease in plasma concentrations of interleukin-6 after 16 weeks of resistance training. It is possible that a longer period (>12 weeks) might be necessary for marked alterations in plasma interleukin-6 to occur. There was no chronic effect of 16 weeks of resistance training on plasma tumour necrosis factor-α, consistent with the above studies.

Interleukin-15 was initially identified as a T-cell growth factor and shares many properties and functions with interleukin-2 (Giri et al., 1995). Besides interleukin-15 immune functions, this cytokine serves as a growth factor, which is highly expressed in skeletal muscle resulting in tissue hypertrophy (Nielsen & Pedersen, 2007). An *in vivo* study indicated that systemic administration of interleukin-15 in muscle-wasting rats with cancer cachexia decreases muscle protein degradation, preserving lean body mass (Carbo et al., 2000). Ostrowski et al. (1998) found no alterations in plasma interleukin-15 (measured up to 6 h after exercise) in response to 2.5 h of treadmill running at 75% of $\dot{V}O_{2max}$. After 10 weeks of resistance training (three sessions per week with loads set at 80% of 1-RM), there was an acute increase immediately after exercise but not chronic increase in plasma interleukin-15 in young individuals (Riechman, Balasekaran, Roth, & Ferrell, 2004). In the present study, we found an increase in plasma concentrations of interleukin-15 48 h after the first resistance exercise session compared with baseline. In line with the above-mentioned studies, no chronic effect of training was observed. It is possible that this cytokine is involved in acute muscle repair and growth after resistance training sessions, acting more locally than presenting chronic plasma alterations.

Leptin is a hormone/cytokine released from adipose tissue, which may have an effect on appetite. Circulating leptin increases with weight gain and decrease with weight loss (Fatouros et al., 2005). Leptin typically exerts pro-inflammatory effects on the immune system. There is evidence that leptin may trigger the growth of several cancer cells, including pancreatic, ovarian, prostate cells, pulmonary carcinomas and gastric cells (Tilg & Moschen 2006). Leptin signals directly to OBR_b receptors on the surface of macrophage membranes, inducing the synthesis of pro-inflammatory cytokines such as tumour necrosis factor-α, interleukin-6, and interleukin-12 (Tilg & Moschen, 2006; Zhao et al., 2005).

Exercise alters the homeostasis of energy balance, sympathoadrenal discharge, as well as hormonal and metabolic responses, which may influence leptin concentrations at rest or during exercise. Some previous studies examined the effects of acute, but not chronic exercise on plasma leptin. For example, type 2 diabetes individuals exhibited a significant 30% reduction in resting leptin 24 h after a single bout of resistance exercise, with no chronic training effect after 6 weeks (Kanaley et al., 2001). Leptin exhibited a delayed reduction 9–13 h in the systemic circulation after an acute resistance-exercise protocol for the main muscle groups with loads between 70 and 85% of 1-RM (Nindl et al., 2002). The acute alterations due to different resistance exercise intensities on plasma leptin concentration, maximal strength (4 sets of five repetitions at 88% of 1-RM with 3 min rest between sets), muscular hypertrophy (4 sets of 10 repetitions at 75% of 1-RM with 2 min rest between sets), and strength endurance (4 sets of 15 repetitions at 60% of 1-RM with 1 min rest between sets) were tested in young individuals; the results showed a significant decrease in leptin 30 min after exercise, with no differences between the protocols (Zafeiridis, Smilios, Considine, & Tokmakidis, 2003). Similarly, in the present study, leptin decreased 24 h after the first acute resistance exercise

session and returned to pre-exercise values after 48 h in older post-menopausal women.

However, little information is available about the chronic effects of resistance training on leptin in older adults. Fatouros et al. (2005) tested the effect of different resistance exercise intensities in overweight and inactive elderly individuals, at low intensity (45–50% of 1-RM), moderate intensity (60–65% of 1-RM), and high intensity (80–85% of 1-RM). The training protocol was performed three times a week for 24 weeks. All groups presented a significant chronic decrease in plasma leptin concentration, with greater reductions for the high-intensity group. After 24 weeks of detraining, leptin concentration increased again. In our study, a significant chronic decrease in serum leptin was observed after 16 weeks of resistance training. Possible explanations for a decline in leptin induced by resistance exercise include elevated glucose uptake by peripheral tissues in the presence of lactate, induced acidosis, augmented sympathoadrenal discharge and energy expenditure, glycogen depletion, and glycolysis inhibition (Fatouros et al., 2005).

Another inflammatory biomarker and risk factor of atherosclerotic cardiovascular disease is resistin (Reilly et al., 2005). Jamurtas et al. (2006) showed no acute alteration in plasma resistin concentration up to 48 h after a 45-min bout of cycle ergometer exercise at 65% of $\dot{V}O_{2max}$ in middle-aged overweight males. Another study did not find chronic alterations in resistin concentration after 14 weeks of aerobic training that consisted of a supervised walking programme 3–4 times per week for 60 min at 65–70% of $\dot{V}O_{2peak}$ in diabetic post-menopausal women (Giannopoulou et al., 2005). In conrrast, older diabetic individuals submitted to 16 weeks of aerobic exercise training with four 45–60 min sessions per week at 50–85% of $\dot{V}O_{2max}$ exhibited a significant chronic decrease in resistin (Kadoglou et al., 2007). A recent study found a significant decrease in resistin after 8 weeks of a supervised aerobic training in overweight adolescents (Jones, Basilio, Brophy, McCammon, & Hickner, 2009). The present study is the first to examine the effects of acute and chronic resistance training on plasma resistin concentration in older post-menopausal women. The results point to a decrease after 24 and 48 h after the acute exercise session compared with baseline, and significant reductions after 16 weeks of training compared with baseline and immediately after values.

Resistin may be affected by the type and intensity of exercise, aerobic versus resistance training, duration of the study, and population analysed. Possible explanations for the results of the present study include lowered production of pro-inflammatory cytokines by the innate immune system, decreased production of pro-inflammatory mediators by adipose tissue and liver, and increased production of anti-inflammatory mediators by adipose tissue (Nicklas, You, & Pahor, 2005). The reduction in systemic pro-inflammatory markers is associated with the decrease of chronic degenerative diseases in post-menopausal women (Colbert et al., 2004).

The present study has some limitations, including a small number of participants ($n = 35$), the absence of a control group, and a short 16-week training period. Longer studies with a control group should be conducted.

In summary, the present study highlights important clinical effects of periodized resistance exercise training on muscle force and inflammatory biomarkers in post-menopausal elderly women. Sixteen weeks of resistance training induced increase in upper and lower body muscle force, a chronic decline in serum concentrations of interleukin-6, leptin, and resistin, with minor or no effects on interleukin-15 and tumour necrosis factor-α. These results outline the importance of resistance exercise as a non-pharmacological tool to reduce systemic inflammation in this population. We suggest that future studies compare the effects of different periodization models, such as undulating and reverse linear on inflammatory markers. Comparisons among aerobic and resistance training are also important.

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