Differential responses of adiposity, inflammation and autonomic function to aerobic versus resistance training in older adults

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Abstract

Background: Increased body fat, autonomic dysfunction and low-grade chronic inflammation are interrelated risk factors implicated in the etiology of several chronic conditions normally presented by older adults.

Objective: This study aims to assess the effectiveness of different training protocols on reducing body fat, improving autonomic function, and decreasing low-grade systemic inflammation in community-dwelling elderly adults.

Methods: Fifty participants (11 men, 68±5.5 years) were randomly allocated into resistance or aerobic training control groups. Evaluations were done at baseline and following the 8-month intervention period on their body composition (assessed by DXA), inflammatory biomarkers (high-sensitivity C-reactive protein [hs-CRP]), tumor necrosis-alpha [TNF-α], interferon-gamma [IFN-γ], interleukins-6 and -10 [IL-6, IL-10]), lipoprotein profile, fasting glycemia, blood pressure, heart rate variability (HRV; frequency and time domains) and aerobic fitness (assessed by six-minute walk distance [GMWD]). A paired t-test was used to detect changes (%Δ=(post-test score−pretest score)/pre-test score×100) within groups, while between-group differences were analyzed using the one-way ANOVA or General Linear Models.

Results: A significant change (%Δ) both in total (−5.4±6.3% and −3.3±2.9%, respectively) and central body fat (8.9±11.3% and −4.8±4.5%) was observed in resistance and aerobic training groups, respectively; along with a change in resting systolic and diastolic blood pressures (−9.2±9.8% and −8.5±9.6%), heart rate (−4.6±6.5%), hs-CRP (−18.6±60.6%), and GMWD (9.5±6.9%) in response to aerobic training.

Conclusions: The present findings provide further evidence for the benefits of aerobic and resistance training on reducing body fat. Aerobic training was demonstrated to reduce hs-CRP and blood pressure in community-dwelling elderly participants with no serious medical conditions.

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1. Introduction

Increased body fat, autonomic dysfunction, and low-grade chronic inflammation are interrelated risk factors (Marsland et al., 2007; Tracey, 2005) implicated in the etiology of diabetes (de Rekeneire et al., 2006), hypertension (Mathieu et al., 2009), atherosclerosis, and other cardiovascular diseases (CVD) (Heffernan et al., 2009).

Older adults normally present diminished autonomic nervous system function and higher circulating levels of inflammatory biomarkers; higher CVD morbidity and mortality is an unsurprising result in this population (Chodzko-Zajko et al., 2009; Newman et al., 2003). High circulating levels of inflammatory biomarkers are associated with poorer physical function (Newman et al., 2003), and consequently with a reduced quality of life in these older adults. The parasympathetic nervous system may inhibit inflammation by discharging acetylcholine (ACh) and suppressing synthesis and release of pro-inflammatory cytokines (Marsland et al., 2007; Tracey, 2005). This may have important implications for developing novel therapeutic strategies against subclinical chronic inflammation. Examples of such interventions include direct vagal nerve electrical stimulation, acupunture techniques, relaxation therapy, and the administration of nicotinic ACh receptor agonists (Johnston and Webster, 2009; Marsland et al., 2007). Regular exercise training induces positive adaptations in heart rate variability (HRV) (Tulppo et al., 1998), body composition (Henwood et al., 2008) and fitness (Tulppo et al., 1998), as well as a reduction in low-grade chronic inflammation (Petersen and Pedersen, 2005). Thus mortality and morbidity from cardiovascular disease are collectively decreased (Kokkinos et al., 2010).

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Exercise training is recommended for health promotion and disease prevention in older adults (Chodzko-Zajko et al., 2009). However, for this population there is conflicting evidence regarding the extent to which training prevents or reverses accumulation of visceral fat deposition (Heffernan et al., 2009), and whether it brings about changes in HRV (Loinmaa et al., 2000), and low-grade chronic inflammation (Markovitch et al., 2008).

Between-study discrepancies may be the result of measuring outcomes via indirect methods, such as the use of anthropometrics instead of dual-energy X-ray absorptiometry (DXA) to measure body composition. Heterogeneity in findings also stems from the use of cross-sectional studies including populations that are diverse in terms of age and disease status. Other limitations previously noted are: (i) a paucity of experimental studies using alternative types of exercise training instead of aerobic training to diminish inflammatory biomarkers and increase HRV, (ii) lack of control for habitual daily physical activity and dietary intake, and finally, (iii) use of either one or two biomarkers in most studies to assess low-grade chronic inflammation.

The purpose of this study was to examine the effectiveness of two different training programs on reducing body fat, improving autonomic dysfunction, and decreasing low-grade inflammation in community-dwelling older adults.

2. Material and methods

2.1. Participants and study design

In this randomized controlled trial (RCT) study with a parallel three-group design, one hundred and eight community-dwelling and independent Portuguese Caucasian older adults were recruited from the Porto area (Portugal) via advertisement in newspapers.

Participants attended the Faculty of Sport (University of Porto) on four different days. According to the Helsinki Declaration, the nature, benefits, and risks of the study were explained to the volunteers, and their written informed consent was obtained. On the first day, one hundred and five volunteers (78 women and 27 men) completed a health history questionnaire to record past and present conditions and medications. Participants were included if they were older than 60 years and were not involved in supervised regular exercise training (performing moderate to vigorous exercise for 20 min or more at least twice a week) in the previous six months. Excluded were volunteers with acute or terminal illnesses, severe or uncontrolled hypertension or any cardiovascular and/or respiratory disorder, those with neurological, skeletal-muscle or joint disorders or disturbances that precluded participation in exercise, or who were undergoing pharmacological therapies that could reduce safety during exercise or influence the responses of cardiovascular function.

Participants underwent a battery of evaluations in the following order: resting HR and HRV, resting blood pressure then 6-min walk distance (6MWD). On a third visit, participants underwent anthropometric assessments, body composition measurements, and were instructed in use of accelerometers and 4-day food diary. Finally, >7 days after the third visit, participants returned the completed 4-day food diary and the accelerometer recordings. Venous blood was collected for further biochemical determination of lipid profile, glycemia, and circulating levels of inflammatory biomarkers.

The eighty-five participants who met inclusion criteria were randomly allocated (computer-generated block randomization) into aerobic training, resistance training or control for eight months. Completion of the training program was defined by an attendance rate >80% of scheduled sessions, while those absent from >7 consecutive sessions were also excluded from the analysis. Instructions were not to change the physical activity already included in their daily living routines or dietary patterns during the course of the study. All methods and procedures received ethical approval by the Institutional Review Board (IRB: PTDC/DES/108780/2008).

2.2. Intervention

For both training protocols, older adults trained three times per week (non-consecutive days) for eight months. Each exercise session lasted approximately 50 min and all sessions were performed under the supervision of a physical education teacher.

Aerobic training (AT) comprised a 10-min warm-up that included stretching, calisthenics, and low-intensity exercises (walking, cycling), 30 min of primarily walking aerobic exercise but also stepping and dancing, followed by a 10-min cool-down. The intensity of AT was calculated using the Karvonen formula, and it was gradually increased during the first month from 50–60% up to 70–80% of HRReserve. For the calculation of HRReserve, maximum heart rate was estimated as HRmax = 208 – 0.7 (age) as suggested by Tanaka et al. (2001). Polar heart rate monitors (Polar Team System, Finland) were worn during each exercise session to ensure that participants exercised at the targeted intensity.

Resistance training (RT) also comprised a 10-min warm-up including stretching, calisthenics, and low-intensity exercises (walking, biking). The warm-up was followed by nine resistance exercises (leg press, chest press, leg extension, seated row, seated leg curl, abdominal flexion, biceps curl, low-back extension, and triceps extension) covering all major muscle groups, and a 10-min cool-down. During the first week of RT, the participants were familiarized with the variable resistance machines (Nautilus Sports/Medical Industries, Independence, VA) by performing one set of 12–15 repetitions with no load. Participants were taught correct lifting techniques and safety precautions. To minimize excessive BP responses, individuals were told to avoid extended breath-holding (Valsalva maneuver), during repetitions (Braith and Stewart, 2006). One repetition maximum (1RM) for each exercise was calculated after which participants performed two sets of 12–15 reps at 50–60% of 1RM. After the first month, 1RM was re-evaluated and resistance increased to 80% of the new 1RM. Every consequent two months 1RM was measured and training load adjusted in order to maintain an adequate training stimulus. Concentric and eccentric movements were performed at a rate of 3 s (Henwood et al., 2008). Participants were allowed a 2-min rest between each set.

During the study period, individuals randomized to the Control group were contacted by phone every four months to certify that they were still interested in participating in the program. They were also asked not to change their lifestyle. After the 8-month observation period, they were invited to participate in specific exercise programs designed for seniors at the Faculty of Sport.

2.3. Assessments

Anthropometrics and body composition. Body weight was measured to the nearest 0.1 kg with an electronic weight scale (SECA 708). Participants were weighed barefoot wearing light clothing. Height was measured to the nearest 1 mm with a standard stadiometer. Body mass index (BMI) was determined as weight divided by height squared (kg/m²). Percent of total body fat (%BF), trunk fat (kg) and lean mass (kg) were determined using DXA (Hologic QDR-4500, software for windows XP, version 12.4) with participants in the supine position.

Biochemical analysis of plasma glucose, cholesterol (total cholesterol, HDL, LDL) and triglycerides was performed using standard enzymatic methods of measurement. High sensitivity C-reactive protein (hs-CRP) levels were determined by means of particle-enhanced immunonephelometry using a BN™II nephelometer (Dade Bhering).

Cytokine measurements were performed using a human multiplex immunoassay kit from Millipore according to the manufacturer’s protocol. Briefly, 10 µl of plasma was diluted and assayed for interleukins-6 and -10 (IL-6, IL-10), interferon-gamma (IFN-γ), and tumor necrosis factor-alpha (TNF-α) using specific antibodies (Abs) conjugated to beads and read on a Luminex 100 instrument (Luminex). The concentration of each cytokine was determined by preparation of a standard...

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The standard deviation of normal RR intervals (SDRR) was used as a measure of submaximal aerobic endurance in older adults (Kervio et al., 2003). This test measures the distance covered when participants are instructed to walk as far as possible in 6 min. Walks are conducted on a flat, indoor 50 m rectangular course marked off in 5 m segments. If necessary, participants are allowed to stop and rest.

Physical activity was assessed using accelerometers (Actigraph GT1M, Actigraph LLC, Pensacola, FL) as an objective measure of daily physical activity. The Actigraph accelerometer is a uniaxial monitor that measures the intensity of movement averaged over 1 min epochs (Mark and Janssen, 2008). Monitors were programmed to start recording at midday of the second meeting and for the following seven days. Participants were instructed to wear the accelerometer around their waist over their right leg, for a 7-day period (five weekdays and two weekend days). Exceptions included time spent sleeping, showering, and during water-based activities. Participants were asked to maintain their usual activities and record them in a diary. Data from each monitor were downloaded by the investigators and compared with data from a diary before the average counts/min had been calculated. Physical activity levels were therefore simply expressed as the average counts per minute (obtained from the Actigraph monitor) over the seven days. This unit of measurement (counts/min) is generated by magnitude and frequency of movement (Mark and Janssen, 2008).

Dietary intake was assessed by a 4-day dietary record (three weekdays and one weekend day) at two points (before and after the intervention). Participants were invited to record all foods and drinks consumed on each recording day, while trying not to change their eating habits, and then to bring the completed records to the research team. Trained nutritionists in the research center supervised the coding of records and data in accordance with uniform procedures. Dietary records were analyzed using an adapted Portuguese version of the software Food Processor Plus® (ESHA Research Inc., Salem, Oregon, USA).

2.4. Statistical analysis

Measures of IFN-γ, hs-CRP, IL-6, IL-10, SDRR, and HF, were log transformed before analysis. Between-group differences in baseline values were determined with one-way analysis of variance (ANOVA). When a significant main effect was detected at a significance level of $P<0.05$, the LSD correction was used for post hoc comparison. To detect changes within groups (before vs. after) the paired t-test was employed. The pre-to-post changes ($\Delta$) for each subject were related to the individual baseline level to define the relative change ($\%$ = [(post-test score – pretest score)/pre-test score] x 100) in all selected variables. General Linear Models (univariate) were used with relative change as the dependent variable and group as the fixed factor. Afterwards, in separate models, baseline values, gender, %BF and medication were inserted as covariates. When a significant main effect was detected at ($P<0.05$) an LSD correction was used. Results are shown as mean± standard deviations or as median with 25th and 75th percentiles. Linear regression analyses were employed to examine the associations between pre-to-post changes in body composition, blood pressure, HRV, and inflammation as well as the association between each baseline value of body composition, SBP, HRV and inflammation and each of the pre-to-post changes in SBP, HF and hs-CRP. Statistical significance was set at a $P<0.05$. All analyses were performed using the Statistical Package for the Social Sciences (SPSS, version 17.0 for Windows, Chicago, IL).

3. Results

After randomization, 11 individuals (8 RT, 3 AT) dropped out due to incompatibility with training timetable leaving 74 enrolled in the intervention. During the eight months 24 (~32%) participants were lost (8 RT, 4 AT and 12 controls). Fifty individuals remained in the study until 8 months. Similar attendance rate was observed for remaining participants at AT and RT ($83.8±3.5$% and $85.7±2.2$%, respectively). The flow of participants during the study is presented in Fig. 1. The final sample mean age was 68.0±5.5 years. Participants were mainly women ($n=39.78$). Fifty-six percent were controlled hypertensive, 42% had dyslipidemia, and 18% had diabetes controlled. According to BMI, 21% were classified as overweight ($25≤BMI<30$) and 28% as obese (BMI ≥30). None of the participants were current smokers. Finally, 4 (10.3%) out of 39 women were on hormone replacement therapy (HRT). At baseline, no differences between groups were observed for age, clinical conditions, body composition, BP, HRV, biomarkers of inflammation, glycemia, lipoprotein profile, or habitual physical activity. The control group had higher HR than the RT ($P = 0.02$), and the RT performed better on the 6MWD than AT and controls ($P = 0.01$). Controls also had a higher energy intake than AT ($P = 0.01$). Table 1 presents the main characteristics of the participants for each group at baseline.

After training, a significant decrease in total and central body fat was observed in RT and AT (see Table 2). In contrast, after the eight months, controls had a higher %BF ($P = 0.02$) and trunk fat ($P = 0.01$). Lean mass remained constant in all groups across the study period.

Except for a decrease in HDL cholesterol in AT ($P = 0.01$) and controls ($P = 0.01$), no differences were observed in lipoprotein profile or glycemia. AT demonstrated lower levels of hs-CRP ($P = 0.02$) after training whereas controls showed increased TNF-α levels ($P = 0.02$) at the end of the observation period (Table 2).

At baseline, 36% and 35% of the participants in RT and AT, respectively, were in the high-risk category for hs-CRP levels (> 3.0 mg/dL). Following training, there was a 50% (RT) and 85.7% (AT) reduction in the number of participants with high-risk hs-CRP.

Sub-analysis of the individuals classified as high-risk due to hs-CRP at baseline found that they also have higher levels of IL-6 ($P = 0.01$). The high-risk individuals in the AT experienced reduction in the IL-6 levels (− 43.5±32.0%; $P = 0.04$) following training. The AT demonstrated significant decreases in SBP ($P = 0.01$), DBP ($P = 0.01$) and HR$_{rest}$ ($P = 0.01$). HR$_{rest}$ diminished in controls as well ($P = 0.05$). There was a trend for AT to present a higher HF ($P = 0.06$). The AT demonstrated better performance in 6MWD ($P = 0.01$) after training. No differences were observed in the habitual daily living physical activity levels or energy intake of older adults across the interventions/observation.
General Linear Models (see Table 3) demonstrated significant differences in relative changes between groups for %BF, trunk fat, and 6MWD. Post hoc comparisons demonstrated greater differences in RT and AT than in controls for %BF (P < 0.01) and trunk fat (P < 0.01). There were greater improvements in 6MWD (P < 0.01) in the AT group compared with controls. Further adjustments for %BF, energy intake, gender, and medication use did not significantly change the overall results. Data from simple linear regressions showed that there were no associations between %BF, trunk fat, HR, SBP and DBP at rest, hs-CRP and HF absolute changes (pre-to-post changes—Δ). But, it was observed that hs-CRP baseline value related with ΔSBP (P = 0.03), ΔDBP (P = 0.04) and ΔHF (P = 0.03). Baseline %BF related with ΔHR at rest. Finally, baseline trunk fat related with ΔHF (P = 0.04) (Fig. 2).

4. Discussion

The present study showed that regular aerobic or resistance exercise training decreased total and central body fat. Aerobic exercise training also decreased resting blood pressure and hs-CRP, as well as improved the functional capacity of community-dwelling older adults. The observed mean changes in these variables were similar in AT and RT groups. Altogether, these results suggest that exercise training is an effective intervention in reducing total and central body fat as well as decreasing a marker of chronic inflammation. These findings support current recommendations that both aerobic and resistance training are effective interventions for health promotion and disease prevention in older, community-dwelling populations without serious medical conditions.

The associations between increased body fat and chronic conditions such as diabetes, hypertension, dyslipidemia, and metabolic syndrome are well established. The need to decrease body fat and its associated comorbidities in the general population has motivated investigations to develop efficient interventions to achieve this goal. Similar to other research, the present study demonstrated that both resistance (Sillamäe et al., 2008) and aerobic (Oikta et al., 2004) training are effective in inducing significant reductions in body fat. On the contrary, many interventions fail to positively impact on body composition (Heffernan et al., 2009; Timmerman et al., 2008; Wanderley et al., 2010) due to insufficient dose of exercise (Heffernan et al., 2009; Wanderley et al., 2010) or by less accurate, proxy measures of body composition. Please cite this article as: Wanderley, F.A.C., et al., Differential responses of adiposity, inflammation and autonomic function to aerobic versus resistance training in older adults, Exp. Gerontol. (2013), http://dx.doi.org/10.1016/j.exger.2013.01.002
Changes in variables before and after training. Values normally distributed are represented by means with standard deviations and values not normally distributed are represented by medians with percentiles between 25 and 75 (25th–75th percentiles).

### Table 2

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Resistance training</th>
<th>Aerobic training</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td>Before</td>
</tr>
<tr>
<td>Body composition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.5 ± 5.0</td>
<td>28.1 ± 4.0</td>
<td>28.1 ± 4.1</td>
</tr>
<tr>
<td>%BF</td>
<td>34.5 ± 7.1</td>
<td>32.9 ± 8.3</td>
<td>38.4 ± 5.3</td>
</tr>
<tr>
<td>Trunk fat (kg)</td>
<td>11.6 ± 4.5</td>
<td>10.6 ± 4.4</td>
<td>12.4 ± 3.5</td>
</tr>
<tr>
<td>Lean mass (kg)</td>
<td>43.7 ± 9.8</td>
<td>43.8 ± 9.8</td>
<td>37.2 ± 5.5</td>
</tr>
<tr>
<td>Resting blood pressure and heart-rate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>131.0 ± 11.6</td>
<td>123.3 ± 14.2</td>
<td>137.5 ± 21.8</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>71.3 ± 7.0</td>
<td>67.4 ± 8.2</td>
<td>72.8 ± 9.3</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>62.2 ± 10.3</td>
<td>60.3 ± 8.4</td>
<td>67.5 ± 7.8</td>
</tr>
<tr>
<td>Resting heart-rate variability</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6MWD (m)b</td>
<td>36.8 ± 21.0</td>
<td>32.8 ± 22.3</td>
<td>24.0 ± 13.0</td>
</tr>
<tr>
<td>Functional capacity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI, body mass index; %BF, percentage of body fat; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; 6MWD, six-minute walk distance; LDL, low density lipoprotein; HDL, high density lipoprotein; hs-CRP, high sensitive C-reactive protein; TNF-α, tumor necrosis factor-alpha; IL-6, Interleukin-6; IL-10, Interleukin-10; IFN-γ, interferon-gamma; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; 6MWD, six-minute walk distance.</td>
<td>0.05.</td>
<td>0.05.</td>
<td>0.05.</td>
</tr>
</tbody>
</table>

### Table 3

General Linear Models: between-group comparisons of mean relative changes (%Δ) and relative amount of variance explained by models.

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Δ% resistance</th>
<th>Δ% aerobic</th>
<th>Δ% controls</th>
<th>F</th>
<th>P</th>
<th>Partial eta squared (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body composition</td>
<td>-5.4 ± 6.3</td>
<td>-3.3 ± 2.9</td>
<td>2.1 ± 3.5</td>
<td>17.70</td>
<td>&lt;0.01</td>
<td>42.9</td>
</tr>
<tr>
<td>%BF</td>
<td>0.4 ± 4.8</td>
<td>0.1 ± 5.5</td>
<td>-0.6 ± 2.5</td>
<td>0.21</td>
<td>0.81</td>
<td>0.9</td>
</tr>
<tr>
<td>Trunk fat (kg)</td>
<td>-8.9 ± 11.3</td>
<td>-4.8 ± 4.5</td>
<td>4.9 ± 7.2</td>
<td>14.47</td>
<td>&lt;0.01</td>
<td>38.1</td>
</tr>
<tr>
<td>Lean mass (kg)</td>
<td>1.0 ± 13.3</td>
<td>0.2 ± 32.7</td>
<td>-2.0 ± 12.2</td>
<td>0.07</td>
<td>0.93</td>
<td>0.3</td>
</tr>
<tr>
<td>Cholesterol total (mg/dL)</td>
<td>0.1 ± 12.3</td>
<td>-3.4 ± 18.0</td>
<td>-3.1 ± 8.7</td>
<td>0.24</td>
<td>0.79</td>
<td>1.0</td>
</tr>
<tr>
<td>LDL (mg/dL)</td>
<td>10.1 ± 6.7</td>
<td>-5.4 ± 8.5</td>
<td>-6.5 ± 8.8</td>
<td>1.19</td>
<td>0.31</td>
<td>0.49</td>
</tr>
<tr>
<td>HDL (mg/dL)</td>
<td>0.9 ± 11.7</td>
<td>1.4 ± 25.5</td>
<td>19.0 ± 53.2</td>
<td>1.04</td>
<td>0.36</td>
<td>0.43</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>5.0 ± 26.3</td>
<td>5.6 ± 25.5</td>
<td>19.0 ± 53.2</td>
<td>1.04</td>
<td>0.36</td>
<td>0.43</td>
</tr>
<tr>
<td>Glycemia (mg/dL)</td>
<td>0.3 ± 11.9</td>
<td>1.1 ± 2.3</td>
<td>5.9 ± 16.1</td>
<td>1.65</td>
<td>0.20</td>
<td>0.67</td>
</tr>
<tr>
<td>hs-CRP (mg/dL)</td>
<td>93.8 ± 266.3</td>
<td>18.6 ± 60.6</td>
<td>212.2 ± 889.7</td>
<td>0.76</td>
<td>0.47</td>
<td>3.3</td>
</tr>
<tr>
<td>TNF-α (pg/mL)</td>
<td>22.5 ± 42.5</td>
<td>11.1 ± 26.4</td>
<td>23.9 ± 39.8</td>
<td>0.67</td>
<td>0.52</td>
<td>2.9</td>
</tr>
<tr>
<td>IL-6 (pg/mL)</td>
<td>49.8 ± 259.4</td>
<td>4.4 ± 65.4</td>
<td>21.0 ± 92.4</td>
<td>0.67</td>
<td>0.52</td>
<td>3.1</td>
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<tr>
<td>IL-10 (pg/mL)</td>
<td>-29.8 ± 40.3</td>
<td>-10.2 ± 41.3</td>
<td>-18.0 ± 39.1</td>
<td>0.78</td>
<td>0.47</td>
<td>3.8</td>
</tr>
<tr>
<td>IFN-γ (pg/mL)</td>
<td>29.5 ± 231.3</td>
<td>7.6 ± 100.2</td>
<td>-4.8 ± 20.2</td>
<td>0.50</td>
<td>0.61</td>
<td>2.2</td>
</tr>
<tr>
<td>Resting blood pressure (BP) and heart-rate (HR)</td>
<td></td>
<td></td>
<td>0.05.</td>
<td>0.05.</td>
<td>0.05.</td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>-5.4 ± 12.6</td>
<td>-9.2 ± 9.8</td>
<td>-3.1 ± 9.4</td>
<td>1.60</td>
<td>0.21</td>
<td>0.64</td>
</tr>
<tr>
<td>DBP</td>
<td>-5.0 ± 12.7</td>
<td>-8.5 ± 9.6</td>
<td>-2.0 ± 9.6</td>
<td>1.93</td>
<td>0.16</td>
<td>7.6</td>
</tr>
<tr>
<td>HRa</td>
<td>-2.5 ± 7.1</td>
<td>-4.6 ± 6.5</td>
<td>-4.0 ± 8.7</td>
<td>0.12</td>
<td>0.89</td>
<td>0.5</td>
</tr>
<tr>
<td>Resting heart-rate variability</td>
<td></td>
<td></td>
<td>0.05.</td>
<td>0.05.</td>
<td>0.05.</td>
<td></td>
</tr>
<tr>
<td>6MWDa</td>
<td>12.0 ± 81.0</td>
<td>27.0 ± 54.0</td>
<td>87.0 ± 317.0</td>
<td>0.61</td>
<td>0.55</td>
<td>2.6</td>
</tr>
<tr>
<td>Functional capacity</td>
<td>-100.0 ± 0.6</td>
<td>-100.0 ± 0.6</td>
<td>-99.0 ± 1.0</td>
<td>0.79</td>
<td>0.46</td>
<td>3.4</td>
</tr>
<tr>
<td>6MWDa*</td>
<td>4.6 ± 7.4</td>
<td>9.5 ± 6.9</td>
<td>-0.4 ± 9.7</td>
<td>6.86</td>
<td>&lt;0.01</td>
<td>23.8</td>
</tr>
</tbody>
</table>

BMI, body mass index; %BF, percentage of body fat; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; 6MWD, six-minute walk distance; LDL, low density lipoprotein; HDL, high density lipoprotein; hs-CRP, high sensitive C-reactive protein; TNF-α, tumor necrosis factor-alpha; IL-6, Interleukin-6; IL-10, Interleukin-10; IFN-γ, interferon-gamma; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; 6MWD, six-minute walk distance.

* Differences within groups, P < 0.05.

b Variables show differences between groups at baseline, P < 0.05.
Unlike research showing positive effect of exercise training in HDL lipoprotein (Fahlman et al., 2002; Newman et al., 2003; Okita et al., 2004; Ring-Dimitriou et al., 2007) the present study failed to observe positive effects on blood lipid. Concerning the specific case of HDL cholesterol, it is possible that our participants had such high baseline HDL levels that little room was left for improvement. The following discussion will address each risk factor in turn and summarize the potential health implication of the changes seen in this study.

Fig. 2. Relation of hs-CRP baseline value with change in systolic blood pressure (A), diastolic blood pressure (B), and high frequency power (C), and relation between trunk fat baseline value with change in high frequency power (D).

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A significant mean decrease in hs-CRP was observed in participants who undertook aerobic training. High levels of hs-CRP are strongly associated with increased risk for coronary heart disease, stroke, and myocardial infarction (Kritchevsky et al., 2005). The large decreases (85.7% aerobic and 50% resistance) in number of participants classified as having a high-risk hs-CRP level indicate reduced CVD risk after aerobic and resistance exercise training. The subanalysis of the participants with hs-CRP levels >3.0 mg/dL showed that aerobic training also reduced IL-6 levels. As risk factors tend to cluster, this may suggest that high-risk individuals benefit more than those with lower risk, from aerobic training at least.

Adipose tissue contributes up to one-third of circulating IL-6 at rest (Thompson et al., 2010), which leads to the secretion of acute phase proteins by the liver; including CRP (Kritchevsky et al., 2005; Thompson et al., 2010). Free fatty acids released by adipocytes promote production of TNF-α. Exercise-induced loss of body fat may reduce serum IL-6 and TNF-α and as a consequence, CRP will be reduced (Mathieu et al., 2009). In our study, total and central body fat loss induced by aerobic training was accompanied by decreased hs-CRP and IL-6 (only for individuals previously classified as high-risk). Similar reductions in total and central body fat induced by resistance training showed no such reduction in any of the biomarkers investigated.

These findings added to the absence of association between the change in %BF or the change in trunk fat with the change in hs-CRP, suggesting that other training-induced mechanisms beyond body fat reduction may be necessary to decrease inflammation. It is possible that changes in anti-oxidant status (Ferreira et al., 2010) or endothelial function (Hambrecht et al., 2000) are related with the benefits observed in the inflammatory status following aerobic training. Despite the reduction in body fat, no changes in TNF-α were observed after either training protocols. Conversely, the controls demonstrated increases in levels of this biomarker at post-test. It is likely that if exercise training does not decrease TNF-α levels in older adults with no serious medical conditions, it at least avoids an increase.

Despite its important protective role in a number of inflammatory conditions, the effects of training on IL-10 serum levels have not been frequently assessed (Kritchevsky et al., 2005; Pedersen and Pedersen, 2005) suggested that the process of muscular contraction during exercise could stimulate the transcription of IL-6 mRNA, with consequent activation of the cascade of anti-inflammatory cytokines such as IL-10. Evidence supporting this hypothesis comes mainly from animal models (Nunes et al., 2008), acute effects of exercise (Zaldivar et al., 2006) or diseased populations (Goldhammer et al., 2005; Kadoglu et al., 2007). Nunes et al. (2008) showed that regular physical training improved the anti-inflammatory response by increasing plasmatic levels of IL-10 in a rat model of chronic heart failure. In humans, single bouts of exercise increase intracellular anti-inflammatory markers such as IL-4, IL-10 and growth hormone (Zaldivar et al., 2006). Acute moderate-intensity exercise has neither anti- nor pro-inflammatory effects when monitored for 7 days post-exercise (Markovitch et al., 2008). Study of the chronic effects of exercise on IL-10 is limited to patients with chronic conditions like diabetes (Kadoglu et al., 2007) and coronary artery disease (Goldhammer et al., 2005), in whom aerobic training increased circulating IL-10. It is important to take into account, however, that individuals diagnosed with such diseases often present high levels of pro-inflammatory and low levels of anti-inflammatory biomarkers. Such a status would, theoretically, allow greater exercise-induced improvements to be observed in such patients, compared to healthy individuals. Additional work is clearly needed to better understand the impact of exercise training on IL-10 in older adults free of clinical conditions.

Recently it was suggested that exercise training-related improvements in HRV may also play a role mediating the inhibition of inflammatory response observed in trained individuals (Hamer and Steptoe, 2007). The latest findings showed increased vagal heart rate control to be associated with decreased peripheral inflammatory responses, measured by TNF-α, IL-6 and hs-CRP, which may reduce the risk of inflammatory diseases (Heffernan et al., 2009). This immunomodulatory function of the vagus nerve, whereby activation of effenter arm results in regulation of cytokine production, was termed the ‘Cholinergic anti-inflammatory pathway’ (Johnston and Webster, 2009). The trend to an improvement in parasympathetic tonus followed by reduced hs-CRP levels that were observed after aerobic training seems to reinforce the idea of a cholinergic anti-inflammatory pathway, and at the same time give insight into the role of regular aerobic exercise in prevention of diseases linked with low-grade inflammation. However, in the present study, no significant association was found between change in hs-CRP and change in HF. But, it was observed that subclinical inflammation assessed by hs-CRP at baseline was associated with changes in HF and blood pressure after training. These findings suggest that individuals with greater subclinical inflammation benefit more from the effects of training on parasympathetic tonus and blood pressure.

Concerning BP, although others (Carter et al., 2003; Melo et al., 2008) have reported decreases in BP following resistance training, in the current study only the aerobic group demonstrated lower levels of SBP and DBP after intervention. Despite the fact that no significant differences were found in the resistance group, it is important to refer to the reduction in about 8 and 4 mm Hg in the SBP and DBP means of this group, respectively, which was almost two-fold the reduction observed in controls. Once hypertension is closely related with autonomic dysfunction (Mathieu et al., 2009) and chronic inflammation (Kritchevsky et al., 2005) it is possible that the reduction of inflammation added to an increased parasympathetic activity observed in the aerobic group could, at least partially explain the BP reduction in this group. Likewise, aerobic training was a unique intervention to improve the aerobic fitness of older adults. The aerobic exercise-induced improvements in BP and aerobic fitness have been systematically verified by other investigators (Audette et al., 2006; Okita et al., 2004; Perini et al., 2002; Sillanpaa et al., 2008; Thompson et al., 2010).

Although the aerobic group demonstrated a greater number of positive changes to the CVD risk profile than the resistance group, the comparison of relative changes between groups suggests that the relative mean changes observed over the eight months were not significantly different between these groups. Therefore, we have to assume that both training protocols induce changes of similar magnitude in the variables under investigation.

4.1. Limitations and strengths

The sample size may have been insufficient to achieve statistical power when analyzing dynamic outcomes as HRV and some inflammatory biomarkers. Difficulties in making a priori decision stem from a lack of knowledge about reference values to mean significant changes as well as normative values including the full age spectrum (Nunan et al., 2010). Moreover, biomarkers of inflammation and HRV indices presented great measures of dispersion indicating a large inter-subject variation. Breathing frequency was not controlled when analyzing HRV; however, it was demonstrated that measures of HRV using a metronome had a similar consistency with time (Sinnaeve et al., 1998) and a similar reproducibility (Dionne et al., 2002) to free breathing conditions. The high dropout rate observed in the resistance-training group might be considered a limitation. Nevertheless, previous studies (Ring-Dimitriou et al., 2007) with long-duration exercise programs like that in the present study have reported a similar withdrawal rate. In spite of these limitations, this study has important strengths. A key strength is that to our knowledge, this is the first study aimed at assessing training effects on both autonomic function and inflammation in community-dwelling older adults free of clinical heart disease. Additionally, the study is based on well-validated methods for assessing body composition, as well as for controlling physical activity and energy intake. Thus it is our belief that the methodology used in the present study may offer more reliable information and provide insight beyond.
any possible confounders. Lastly, the assessment of several inflammatory biomarkers led us to a broader and deeper evaluation of the effects of exercise in the studied groups.

5. Conclusion

The present findings provide further evidence for the benefits, by different mechanisms, of aerobic and resistance training on reducing total body and trunk fat, and the effectiveness of aerobic training on decreasing blood pressure and hs-CRP in community-dwelling older adults. Although there are a limited number of investigations aimed at assessing the effects of training beyond aerobic exercise on inflammation and autonomic function of older adults, our results emphasize the need to promote and prescribe resistance training as a therapeutic strategy against increased body fat and its related morbidity.

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