ORIGINAL ARTICLE Scandinavian Medicine and Science in Sport

Combined aerobic and resistance training decreases inflammation markers in healthy men

Ihalainen JK[1], Schumann M[1,2], Eklund D[1], Hämäläinen M[3], Moilanen E[3], Paulsen G[45], Häkkinen K[1], Mero AA[1]

[1]Neuromuscular Research Center, Faculty of Sport and Health Sciences, University of Jyväskylä, Finland

[2]Department of Molecular and Cellular Sport Medicine, German Sport University Cologne, Germany

[3] The Immunopharmacology Research Group, University of Tampere, Faculty of Medicine and Life Sciences and Tampere University Hospital, Tampere, Finland

[4]The Norwegian Olympic and Paralympic Committee and Confederation of Sports, Oslo, Norway

[5]Norwegian School of Sport Sciences, Oslo, Norway

Correspondence to: Johanna K. Ihalainen
Department of Biology of Physical Activity
University of Jyväskylä
P.O Box 35
40014 Jyväskylä, Finland
Tel. 358-40-8347106
Fax 358-14-2602071
Email: johanna.k.ihalainen@jyu.fi

Running title: Anti-inflammatory effects of exercise

ABSTRACT

Background and aims: Our primary aim was to study the effects of 24 weeks of combined aerobic and resistance training performed on the same day or on different days on inflammation markers.

Methods and results: Physically active, healthy young men were randomly divided into three groups that performed: aerobic and resistance training consecutively in the same training session (SS) 2-3 $d \cdot wk^{-1}$ or on alternating days (AD) 4-6 $d \cdot wk^{-1}$ as well as control (C). The total training volume was matched in the training groups. The control group was asked to maintain their habitual physical activity and exercise level. Maximal leg press strength (1RM) and peak oxygen uptake ($VO2_{peak}$) were measured. Abdominal fat mass was estimated with dual-energy absorptiometry (DXA). High-sensitivity C-reactive protein (hs-CRP), interleukin 6 (IL6), monocyte chemo attractant protein 1 (MCP-1), tumor necrosis factor alpha (TNF-α) and adipocytokines resistin, adiponectin and leptin were analyzed from plasma samples. Training significantly reduced circulating hs-CRP, leptin and resistin in both training groups ($P<0.05$), whereas MCP-1 and TNF-α decreased only in AD ($P<0.05$). Significant correlations were observed between changes in abdominal fat mass and corresponding changes in MCP-1, leptin, adiponectin and resistin.

Conclusion: Long-term combined aerobic and resistance training reduced markers of subclinical inflammation in healthy young men. The results indicate that a higher frequency of individual exercise sessions might be more beneficial with respect to the anti-inflammatory effects of physical activity. The decreases in inflammation markers seem to be related to decreases in abdominal fat mass.

Keywords: physical exercise, abdominal fat, adipokines, low-grade inflammation

1 Introduction

It is well recognized that the pathogenesis of chronic metabolic diseases such as type 2 diabetes (Pradhan et al., 2001) and atherosclerosis (Hansson, 2005) involve prolonged low-grade inflammation indicated by increased circulating levels of inflammatory mediators (Fantuzzi, 2005). Thus, previous studies have indicated an inverse association between physical activity and low-grade inflammation (Fischer et al., 2007; Lavie et al., 2011; Pinto et al., 2012). As such, lower inflammatory markers have been observed especially in individuals who report performing frequent moderate intensity physical activity (Beavers et al., 2010).

Both aerobic (AT) and resistance training (RT) have been shown to be important strategies for improving inflammatory profiles (Nassis et al., 2005). Interestingly, Nimmo et al. (2013) concluded that the most marked improvements in the inflammatory profile are probably achieved with a combination of high intensity AT and RT. While the effects of either AT or RT on inflammation are relatively well studied, data regarding the effects of combined AT and RT on inflammatory markers is sparse. Libardi et al. (2012) failed to observe significant reductions in inflammatory markers after combined training in sedentary middle-age men, while other studies have found significant improvements in inflammation markers in healthy untrained men and women (Donges et al., 2013;Stefanov et al., 2014) as well as in obese men (Brunelli et al., 2015) and in subjects with metabolic syndrome (Balducci et al., 2010). However, combined training can be performed in multiple ways, for example by performing AT and RT in the same session with different orders or separated on alternating days (Eklund et al., 2016).

Training intensity and frequency have been shown to affect inflammation markers in a dose-dependent manner (Fatouros et al., 2009). As changes in fat mass have previously been associated with alterations in low-grade inflammation (Gleeson et al., 2011a) it can be assumed that the mode of combined training could have a significant effect on the inflammatory profiles as well. A previous study from our group reported a significant reduction in fat mass after a training intervention, but only in a group that separated aerobic and resistance exercises on alternating days thus increasing the frequency of training while keeping the total training volume constant (Eklund et al., 2016). Thus, we hypothesized that the combined training mode with sufficient frequency may have a beneficial effect on inflammatory profiles. A secondary purpose was to assess whether training-induced changes in body composition and physical performance influence inflammation markers.

2 Methods

Participants. This study is a part of a larger research project (Eklund et al., 2016; Schumann et al., 2014). Participants were recruited through general advertisements in local newspapers as well as posters and emails that were delivered to local companies and institutions. A total of 150 people contacted us to express their interest towards the study (Figure 1). Of these, 93 people met the participation criteria: healthy non-obese (BMI <30 $kg \cdot m^{-2}$) men who were non-smokers, free of acute and chronic illness, disease or injury and did not report use of any medications (diabetes, cardiovascular diseases, cancer, hypertension, rheumatism, osteoporosis). Ultimately, a total of 48 healthy men completed pre- and post-measurements and were included in this study (age = 31 ± 6 yr, height = 1.79 ± 0.06 m, body mass = 80.9 ± 12.3 kg, BMI= 25.2 ± 3.5 $kg \cdot m^{-2}$). The subjects were moderately physically active as characterized by walking, cycling or occasionally participating in team sports at light to moderate intensity and a frequency of 3 $d \cdot wk^{-1}$. The subjects were informed about the possible risks of all study procedures before providing a written informed consent. A completed health questionnaire and resting ECG were reviewed by a cardiologist prior to participation. The study was conducted according to the declaration of Helsinki, and ethical approval was granted by the University of Jyväskylä Ethical Committee.

Study design. The subjects were assigned to either of the two training interventions or the control group: combined aerobic and resistance training performed in the same session (SS, n=16) or on alternating days (AD, n=16) or control group (C, n=16). In another data set from our research group, which was analyzed from the same group of previously untrained subjects, we did not observe significant changes in fat mass or performance variables between the participant who trained endurance and strength in a same session but with a

different order, thus we pooled the data of SS for the purpose of this study. The exercise order of SS training was randomized with half of the group performing aerobic immediately followed by resistance training and the other half performing the opposite exercise order. The overall training volume was equal in the two groups but SS consisted of only 2-3 combined training sessions per week, whereas AD performed 4 to 6 sessions per week (2-3 x aerobic and 2-3 x resistance, respectively) for 24 weeks. Measurements were performed before (PRE), during (i.e. after 12 weeks, MID) and after (i.e. after 24 weeks, POST) the training intervention. The control group was measured at PRE and POST. Participants were asked to keep their dietary intake constant and the dietary intake was examined by nutritional diaries.

Training. All training sessions were supervised and the detailed content has been described elsewhere (Eklund et al., 2016). Briefly, the endurance training was conducted on a cycle ergometer. During weeks 1-7 steady-state cycling of low to moderate intensity (below and above the aerobic threshold) was performed and during the remaining weeks, additional high-intensity interval sessions (below and above the anaerobic threshold) were incorporated into the training program. The duration of endurance cycling progressively increased from 30 to 50 minutes. During the second half of the study, training volume and intensity were further increased. The resistance training programme included exercises for all major muscle groups with a focus on lower extremities. During the first two weeks, training was performed as a circuit using low loads. Thereafter, protocols aiming for muscle hypertrophy and maximal strength were performed. During the last two weeks also protocols targeting explosive strength development were performed. During the subsequent 12-week period both training volume and frequency were slightly increased in an attempt to avoid a training plateau. The overall duration of each resistance training session was 30-50 min.

Abdominal fat. Whole body composition was estimated by Dual X-ray Absorptiometry (LUNAR Prodigy, GE Medical Systems, Madison, USA). The DXA-scans were performed in

the morning with the participant in a fasted (12h) state. Automatic analyses (Encore-software, version 14.10.022) provided total body fat mass and total body lean mass. Abdominal fat was calculated manually defining a range of interest confined cranially by the upper end plate of the first lumbar vertebra, laterally by the ribs and caudally by the iliac crest (Tallroth et al., 2013). This customized range was then copied to the DXA scans at MID and POST, respectively.

Cardiorespiratory performance. A graded protocol on a cycle ergometer (Ergometrics 800, Ergoline, Bitz, Germany) was used to determine $VO2_{peak}$ and metabolic thresholds for the aerobic training. The initial load for all subjects was 50 Watts and increased by 25 Watts every two minutes until volitional exhaustion. Oxygen uptake was determined continuously breath-by-breath using a gas analyzer (Oxycon Pro, Jaeger, Hoechberg, Germany). Peak oxygen consumption ($VO2_{peak}$) was averaged over 60 s periods during the test.

Maximal-strength performance. Maximal strength was measured by a one-repetition maximum (1RM) test of dynamic leg press exercise performed by a David 210 leg press device (David D210, David Health Solutions Ltd., Helsinki, Finland). The starting position (flexed) was at a knee angle of approximately 60 degrees, and 1RM was accepted as the highest loads the participants could lift to a full knee extension (180 degrees). Subjects performed three warm-up sets and 3 to 5 maximal trials, after which the highest load was accepted as the 1RM.

Venous blood samples. Fasting venous blood samples were drawn from an antecubital vein in the morning (7:00-9:00 a.m.) after a 12 h overnight fast. Participants were instructed to abstain from strenuous physical activity for 48 h before the blood samples were taken. Venous blood was collected into EDTA tubes for analysis of inflammatory profiles. The samples were centrifuged for 10 min at +4°C with 2000 x g (Megafuge 1.0 R, Heraeus,

Germany). Plasma was kept at -80°C until analysed for high sensitive-C reactive protein (hs-CRP) and interleukin-6 (IL-6) using the Immulite 1000 and immunoassay kits (Immulite, Siemens, IL). Concentrations of monocyte chemoattractant protein-1 (MCP-1), adiponectin, leptin and resistin in plasma samples were determined by enzyme-linked immunosorbent assay (ELISA) with commercial reagents (R&D Systems, Europe Ltd, Abingdon, UK). The detection limits and inter-assay coefficients of variation, respectively, were 0.1 $pg \cdot ml^{-1}$ and 10 % for hs-CRP, 0.2 $pg \cdot ml^{-1}$ and 3.4 % for IL-6, 3.9 $pg \cdot ml^{-1}$ and 5.0 % for MCP-1, 19.5 $pg \cdot ml^{-1}$ and 2.2% for adiponectin, 15.6 $pg \cdot ml^{-1}$ and 4.0 % for resistin and 15.6$pg \cdot ml^{-1}$ and 5.1 % for leptin.

Statistical analysis. Data was analyzed using PASW statistic 22.0 (SPSS, Chicago, IL, USA). Data is presented as mean ± SD Before applying further statistical methods, the data was checked for sphericity and normality. If a specific variable violated the assumptions of parametric tests, log-transformation was used. This concerned values of adiponectin, leptin, IL-6, MCP-1 and hs-CRP. Absolute changes were analysed via two-way repeated analysis of variance for main (time) and interaction (group × time) effects. For each analysis, the baseline values were used as a covariate to control between-subject and between-group differences at baseline. This was followed by one-way repeated measures ANCOVA on each group to examine a main effect of time. If a significant main effect or interaction was observed, the change from pre-values for MID and POST was compared between groups using paired t-tests with Bonferroni correction. Effect sizes (ES) are given as Cohen's d with an effect size of ≥ 0.20 being considered small, ≥ 0.50 medium, and ≥ 0.80 large. Spearman's correlation coefficients were used to examine the associations between depending variables. The level of statistical significance was set at $p \leq 0.05$.

3 Results

Training adherence. The training adherence was 99±2% and 100±1% in SS and AD respectively. All subjects completed at least 90% of the overall training volume.

Circulating inflammatory markers. Circulating hs-CRP is presented in figure 2. For hs-CRP a significant main effect of time was observed ($p = 0.010$, ES = 0.785). Circulating concentrations of hs-CRP decreased significantly in the SS ($p = 0.021$) and in the AD ($p = 0.004$) from PRE to POST.

Figure 3 illustrates the changes in circulating adipocytokine and cytokine concentrations. A significant main effect of time ($p = 0.010$, ES = 0.942) was observed in concentrations of circulating resistin. Significant reductions in concentrations of circulating resistin were observed in SS ($p = 0.031$, ES = 0.582) and AD ($p = 0.022$, ES = 0.661) but remained unaltered in C. At POST, significant changes in concentrations of circulating leptin were observed in SS ($p = 0.031$) and AD ($p = 0.019$) at POST. Significant changes in adiponectin concentrations were not observed.

In the inflammatory cytokines, a significant main effect of time ($p = 0.02$, ES = 0.869) and interaction ($p = 0.027$, ES = 0.760) was observed in the levels of MCP-1. At POST a significant reduction was observed in AD ($p = 0.02$, ES = 0.840) but not in SS and the control groups. In addition, the reduced concentration of MCP-1 in AD was significantly lower than in SS and C ($p = 0.019$ and $p = 0.007$ respectively). A significant main effect of time was observed in circulating concentrations TNF-α ($p = 0.001$, ES = 0.926). Slight but statistically significant reduction in TNF-α concentration was observed in AD at POST ($p = 0.048$, ES = 0.418), while no changes in SS or C were found ($p = 0.056$ and $p = 0.218$, respectively). Significant main effects of time or interaction in IL-6 were not observed.

Body composition, aerobic performance and strength. Changes in body composition, 1RM and $VO2_{peak}$ are summarized in Table 1 and have been partly published elsewhere (Eklund et al. 2015; Eklund et al. 2016; Schumann et al. 2015). No significant changes were observed in body weight. A significant main effect of time ($p < 0.001$, ES = 0.974) and interaction ($p = 0.014$, ES = 0.789) was observed in abdominal fat mass. After 12 weeks of training, fat mass did not decrease in either of the two experimental groups. However, a significant decrease in abdominal fat mass from PRE to POST was observed in SS (-7.4 ± 15.4 %, $p = 0.041$, ES = 0.445) and AD (-21.1 ± 17.6 %, $p < 0.001$, ES = 0.997). No significant changes in abdominal fat mass was observed in C. Abdominal fat mass in AD at POST was significantly lower compared to SS and C group ($p = 0.050$, $p = 0.019$ respectively).

A significant main effect of time ($p = 0.015$, ES = 0.748) and interaction ($p = 0.007$, ES = 0.877) was observed in $VO2_{peak}$. Both the SS and AD groups increased $VO2_{peak}$ significantly from PRE to MID (6.80 ± 8.28 % $p = 0.001$ and 13.2 ± 11.9 % $p < 0.001$, respectively) and from PRE to POST (9.3 ± 8.85 % $p < 0.001$ and 18 ± 10.3 % $p < 0.001$, respectively), while no significant change was observed in C ($p = 0.637$, ES = 0.081). A significant main effect of time ($p < 0.001$, ES = 0.989) and interaction ($p = 0.003$, ES = 0.918) in 1RM was observed. 1RM increased in all groups ($p < 0.001$). Both training groups as well as C increased 1RM from PRE to MID ($p < 0.001$) and from PRE to POST ($p < 0.001$). The increase in 1RM was significantly larger in SS and AD groups (+14.1 ± 11.4 %, $p<0.01$ and +12.7 ± 7.24 %, $p<0.01$; respectively) than in C group (+4.7 ± 4.65 %).

Associations between changes in performance, body composition and inflammatory markers.

Leptin correlated significantly with abdominal fat mass at all measurement points (PRE R = 0.732, p<0.001, MID R = 0.650, P<0.001 and POST R = 0.522 p < 0.001) when all the subjects were pooled. In addition, in the pooled data, the changes from PRE to POST in abdominal fat mass correlated positively with the change in leptin (R = 0.433, p = 0.002), MCP-1 (R = 0.581, P = 0.023) and resistin (R = 0.343, P = 0.016) and negatively with adiponectin (R = -0.290, p = 0.043). Changes in inflammation markers and performance variables were not associated but a significant negative correlation was observed between TNF-α and $VO2_{peak}$ as well as between leptin and $VO2_{peak}$ at PRE (R = -0.389, R = 0.018 and p = -0.654, all p < 0.05). In the experimental groups, an inverse relationship between change in concentration of circulating adiponectin and change in maximal strength from PRE to POST was observed (R = -0.459, p = 0.014).

4 Discussion

The present study assessed the effects of 24 weeks of combined aerobic and resistance training on inflammation markers in young, healthy men. Herein, we provide evidence that combined AT and RT reduces inflammation as demonstrated by lowered circulating concentrations of hs-CRP, leptin and resistin. The special focus of the present study, however, was to investigate whether the performing AT and RT in the same session (SS) or on alternating days (AD) affected the inflammation markers differently. The main finding of the study was that combined training performed on alternating days elicited the largest reductions in circulating levels of TNF-αand MCP-1. Furthermore, the beneficial effects of exercise on inflammation markers were achieved without concomitant weight loss, however, a decrease in abdominal fat mass was associated with reductions in the inflammation markers, which emphasizes meaningfulness of this change in body composition.

In the present study, we showed that the baseline levels of hs-CRP allowed us to classify the participants as having “moderate cardiovascular risk” (1.0 to 3.0 $mg \cdot L^{-1}$) prior to commencement of the study in all groups. At POST the mean if hs-CRP was reduced to the level of “low cardiovascular risk” (< 1.0 $mg \cdot L^{-1}$) in both experimental groups (Pearson et al. 2003). These findings are in line with a study by Stewart et al. (Stewart et al., 2007a), who suggested that a combination of AT and RT reduced the risk of cardiovascular disease development, as defined by a decrease in hs-CRP concentrations in healthy populations. While C-reactive protein (CRP) concentrations are generally determined by genetic factors, centrally located adiposity is also considered to be a major determinant of CRP levels (Perry et al., 2008). Cross-sectional studies have found an inverse relationship between physical

activity and CRP (Ford, 2002) and training studies have reported reductions in CRP (Stewart et al., 2007a). Interestingly, Libardi et al. (2012) did not find any significant differences in CRP, IL-6 or TNF-α in sedentary middle age men after 16 weeks of concurrent training in which AT and RT were performed in the same session, three times a week. These findings were opposed to those of Stewart et al. (Stewart et al., 2007b), who found a significant improvement in CRP concentrations after a 12-wk concurrent training period in young and old sedentary subject. Interestingly, in the present study we did not observe any significant changes in circulating inflammation markers after 12 weeks, but only after 24 weeks of training. In contrast to the studies by Stewart et al. (2007) and Libardi et al. (2012), the subjects in the present study were young and healthy and reported to be moderately active. Thus, our findings indicate that even moderately active young healthy subjects benefit from prolonged combined AT and RT, but adaptations may be delayed in comparison to inactive and/or elderly subjects. However, it is notable that the training in the present study was progressive as both training volume and frequency were increased during the training intervention. Therefore, it is also possible that the training was not intensive enough to elicit anti-inflammatory effect during the first 12 weeks of training.

Beavers et al. (Beavers et al., 2010) concluded that AT interventions for healthy individuals are beneficial for reducing inflammatory biomarkers, although reductions in body weight are small. In the present study, we did not observe significant reductions in body weight. Interestingly, the abdominal fat mass decreased significantly only when combined training was performed on alternating days as opposed to AT and RT in the same session. This group difference in abdominal fat mass could be due to the greater frequency of exercise that probably resulted in increased overall energy expenditure (Almuzaini et al., 1998). Intra-abdominal obesity has been shown to be an important risk factor for low-grade inflammation.

The distribution of excess fat in the abdominal region is known to modify the health risk profile, whereas excess adiposity in the periphery does not appear to increase the risk of developing cardiovascular disease (Strasser et al., 2012). In the present study, we observed a significant association between the change in abdominal fat mass and all measured circulating adipocytokine concentrations. Previous studies suggest that physically active individuals or subjects with higher fitness level have more favorable adipocytokine profiles compared to sedentary populations (Lavie et al., 2011). This was supported by our findings as the initial $VO2_{peak}$ was significantly associated with circulating leptin concentration at baseline. However, we did not observe a significant correlation between changes in $VO2_{peak}$ and changes in adipocytokine concentrations. Interestingly, we observed a significant reduction in circulating MCP-1 concentrations after 24 weeks when the training was separated into alternating days as opposed to AT and RT in the same session. Moreover, reductions in MCP-1 are associated with the changes in abdominal fat mass, irrespective of intervention group, which indicates that fat mass in the abdominal area has a significant effect on MCP-1 concentration.

We observed that the circulating resistin levels were reduced in both experimental groups after 24 weeks of training, even if we did not observe a significant reduction in visceral fat mass in SS group. Resistin is a signaling protein that has been linked to inflammation and coronary heart disease (Zhang et al., 2010), and, consequently, a reduction in resistin concentrations may be interpreted as a beneficial biological adaptation. Our data indicate that long-term combined AT and RT alters the concentrations of circulating resistin regardless of changes in abdominal fat mass. Gleeson et al. (Gleeson et al., 2011b) suggested that both the reduction of visceral fat mass and the anti-inflammatory environment induced by each exercise session might elicit long-term anti-inflammatory effects. One of the possible

mechanisms behind the anti-inflammatory effect of exercise has been suggested to be the acute IL-6 release following an exercise session, possibly stimulating the accumulation of anti-inflammatory cytokines, such as interleukin-10 and interleukin-1 receptor antagonist (Gleeson et al., 2011c). IL-6 has been shown to be related to circulating resistin levels, but if IL-6 releases are mechanistically linked to reductions in circulating resistin levels awaits further investigation. Nevertheless, we observed no significant changes in circulating IL-6 concentration in the experimental groups.

Changes in body composition, or more precisely, changes in abdominal fat mass seem to be an important factor when an exercise intervention for reducing inflammation markers is planned. In the present study we showed that a significant reduction in adipokines is possible also in the absence of change in abdominal fat mass, as seen in the decrease in resistin levels. However, significant reductions in leptin levels seem to be dependent on a significant reduction in fat mass (Baile et al., 2000). There are several mechanisms involved in the beneficial effects of exercise on immunological function, and recent research has focused on its role in the improvement of the inflammatory profile. However, further studies are needed to identify the molecular mechanisms underlying the anti-inflammatory effect of exercise and what the role of skeletal muscle is in this action.

The strengths of this study include its careful measurement of a wide range of potential confounding variables and a prolonged supervised training intervention. However, several limitations should be considered when interpreting our results. First, the participants in this study were young healthy men and therefore a generalization of our results to other populations might be problematic. Secondly, although in the present study several different

factors are suggested to be important markers and/or regulators of inflammation, there are many other pro- or anti-inflammatory factors that could have been measured. Nevertheless, CRP, in particular, has proven to be a relatively useful marker of systemic inflammation and predictor of clinically relevant outcomes and is the most commonly measured inflammatory marker (Pearson et al. 2003). Lastly, we cannot determine the directions of the associations nor causality observed in this study with absolute certainty.

4.1 Perspectives

Combined AT and RT without concomitant body weight loss may induce anti-inflammatory effects, leading to improvements in levels of circulating inflammation markers in men. These effects could be enhanced with a reduction in visceral fat mass that was observed only when AT and RT were performed on alternating days. The findings of this study indicate that a higher frequency of exercise sessions should be recommended in the prevention of inflammation related diseases. The improvement in the inflammatory profile achieved in the present study may be an effective strategy for reduction in low-grade systemic inflammation and improving the health trajectory of young men.

ACKNOWLEDGEMENTS

This project was partly funded by Ellen and Artturi Nyyssönen Foundation, Juho Vainio Foundation, Jenny and Antti Wihuri Foundation, Yrjö Jahnssons Foundation, Department of Biology of Physical Activity, University of Jyväskylä and the competitive research funding of Pirkanmaa Hospital District. The authors would like to thank Ms Terhi Salonen for excellent technical assistance in the laboratory analyses and all the subjects and research assistants involved in the implementation of the study.

CONFLICT OF INTEREST

The authors do not have conflicts of interests and state that the results of the present study do not constitute endorsement by ACSM. The authors alone are responsible for the content and writing of the manuscript.

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TABLES WITH HEADINGS

Table 1. Physical fitness and body composition at before (pre) after 12 weeks (mid) and after 24 weeks (post) of training. AD = Different-day training, SS = Same-session training, C = Controls. *=difference from PRE value ($p<0.05$) #=difference between the AD and SS. Mean ± SD.

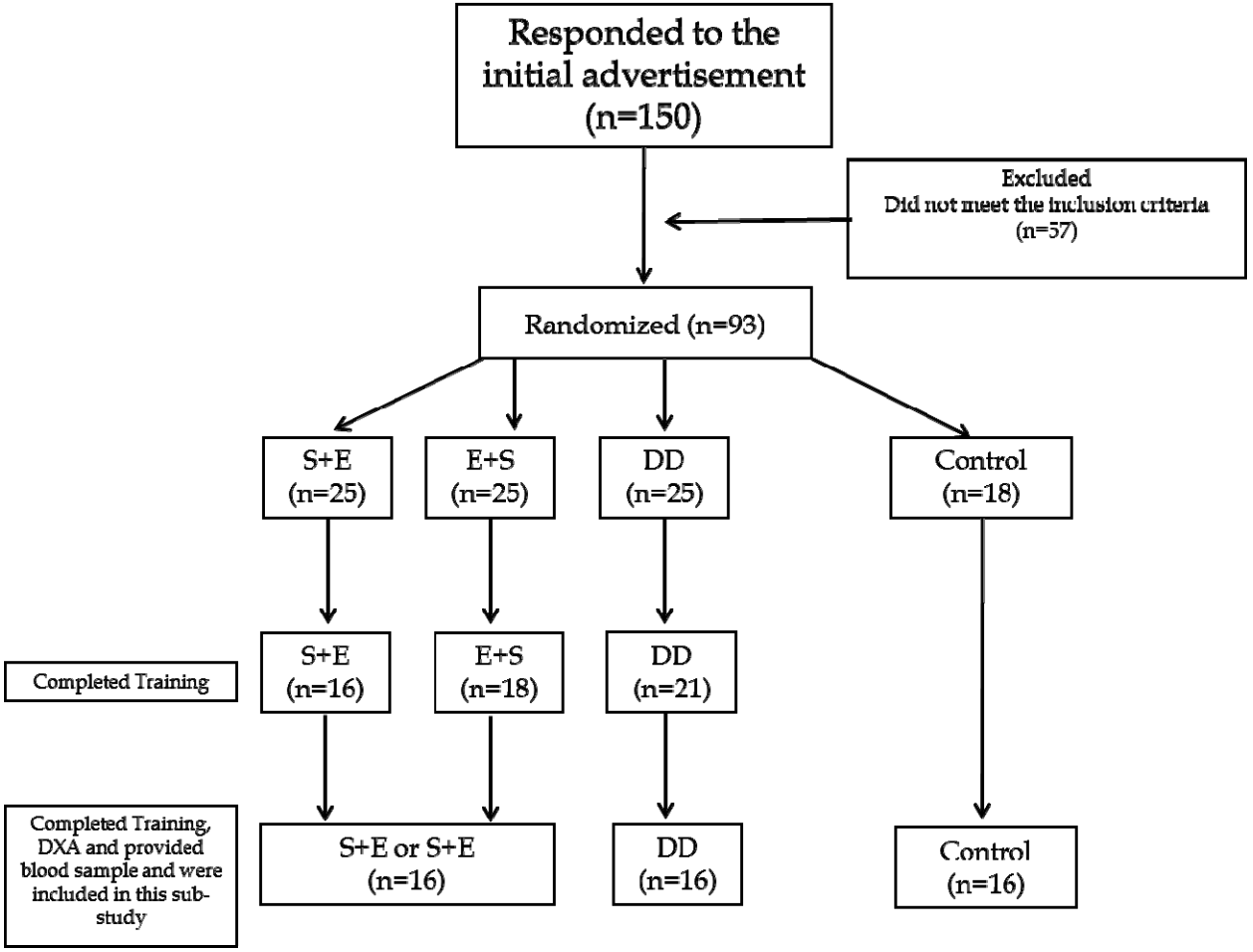
	PRE			MID		POST		
	SS (n=16)	AD (n=15)	CONT (n=18)	SS (n=16)	AD (n=15)	SS (n=16)	AD (n=15)	CONT (n= 18)
Physical fitness								
1RM (kg)	151 ± 32.2	145 ± 18.3	159 ± 29.9	164 ± 26.5	159 ± 16.7	170 ± 26.2	163 ± 16.0	167 ± 28. 5
$VO2_{peak}$ ($L \cdot min^{-1}$)	3.13 ± 0.40	2.82 ± 0.32	3.07 ± 0.53	3.33 ± 0.42	3.17 ± 0.26	3.41 ± 0.49	3.34 ± 0.36	3.11 ± 0.53
Body composition								
Height (m)	1.78 ± 0.06	1.80 ± 0.08	1.78 ± 0.06	1.78 ± 0.06	1.80 ± 0.08	1.78 ± 0.06	1.80 ± 0.08	1.78 ± 0.06
Body weight (kg)	80.1 ± 13.2	81.8 ± 10.3	80.7 ± 11.7	80.1 ± 11.9	81.9 ± 10.3	80.4 ± 11.1	80.6 ± 10.4	81.7 ± 11,5
BMI ($kg \cdot m^{-2}$)	25.2 ± 3.00	25.3 ± 2.60	25.2 ± 3.9	25.2 ± 2.50	25.3 ± 2.93	25.4 ± 2.34	24.9 ± 2.85	25.5 ± 3.8 9
Body fat mass (kg)	20.8 ± 8.12	22.9 ± 6.11	19.2 ± 7.42	20.0 ± 7.27	21.6 ± 6.67	19.0 ± 7.00	19.5 ± 7.28	20.4 ± 7.66
Body Fat-% (%)	25.4 ± 7.1	27.0 ± 4.3	23.1 ± 8.3	24.5 ± 6.6*	27.6 ± 4.4	23.2 ± 6.2 **	25.9 ± 5.5 **	24.4 ± 8.9
Abdominal fat mass (g)	2571 ± 1190	3060 ± 993	2310 ± 1210	2340 ± 1060	2810 ± 1040**	2330 ± 1080	2490 ± 1120***	2450 ± 1361
Lean mass (kg)	53.3 ± 6.13	55.9 ± 5.12	59.5 ± 5.85	54.1 ± 5.74	57.2 ± 5.73	54.8 ± 5.93*	58.0 ± 5.22*	58.7 ± 5.87

FIGURE LEGENDS

FIGURE 1. Flowchart of study participants.

FIGURE 2. Mean (SD) in hs-CRP at weeks 0, 12 and 24. * significant within-group change. AD = alternating days training, SS = same session training, C = controls.

FIGURE 3. Mean (SD) changes in adipocytokines (left) and cytokines (right). * significant within-group change. SS = same session training, AD = alternating days training, C = Controls.



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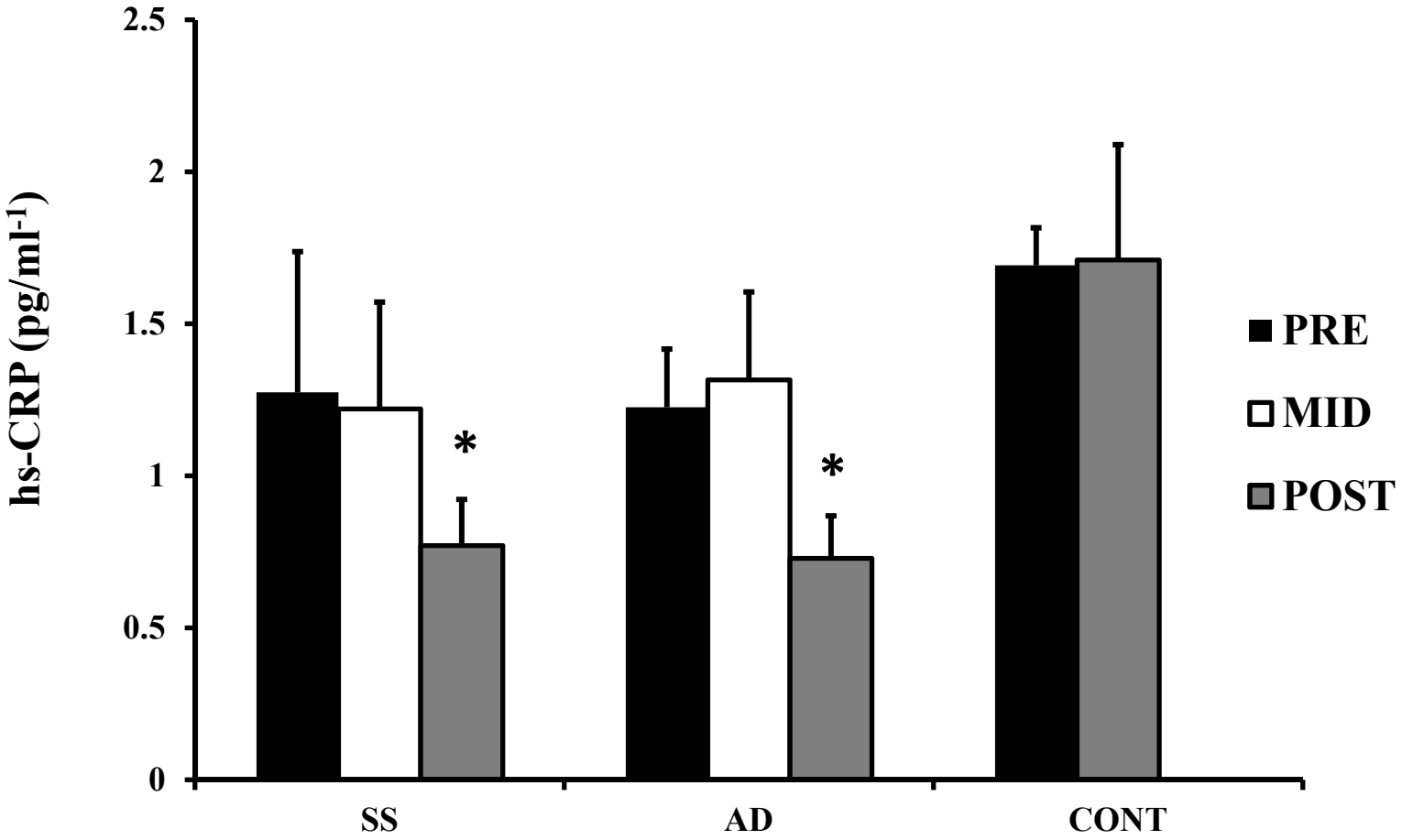

Fig 2. Mean (SD) in hsCRP at weeks 0, 12 and 24. * significant within-group change. AD = alternating days training, SS = same session training, C = controls

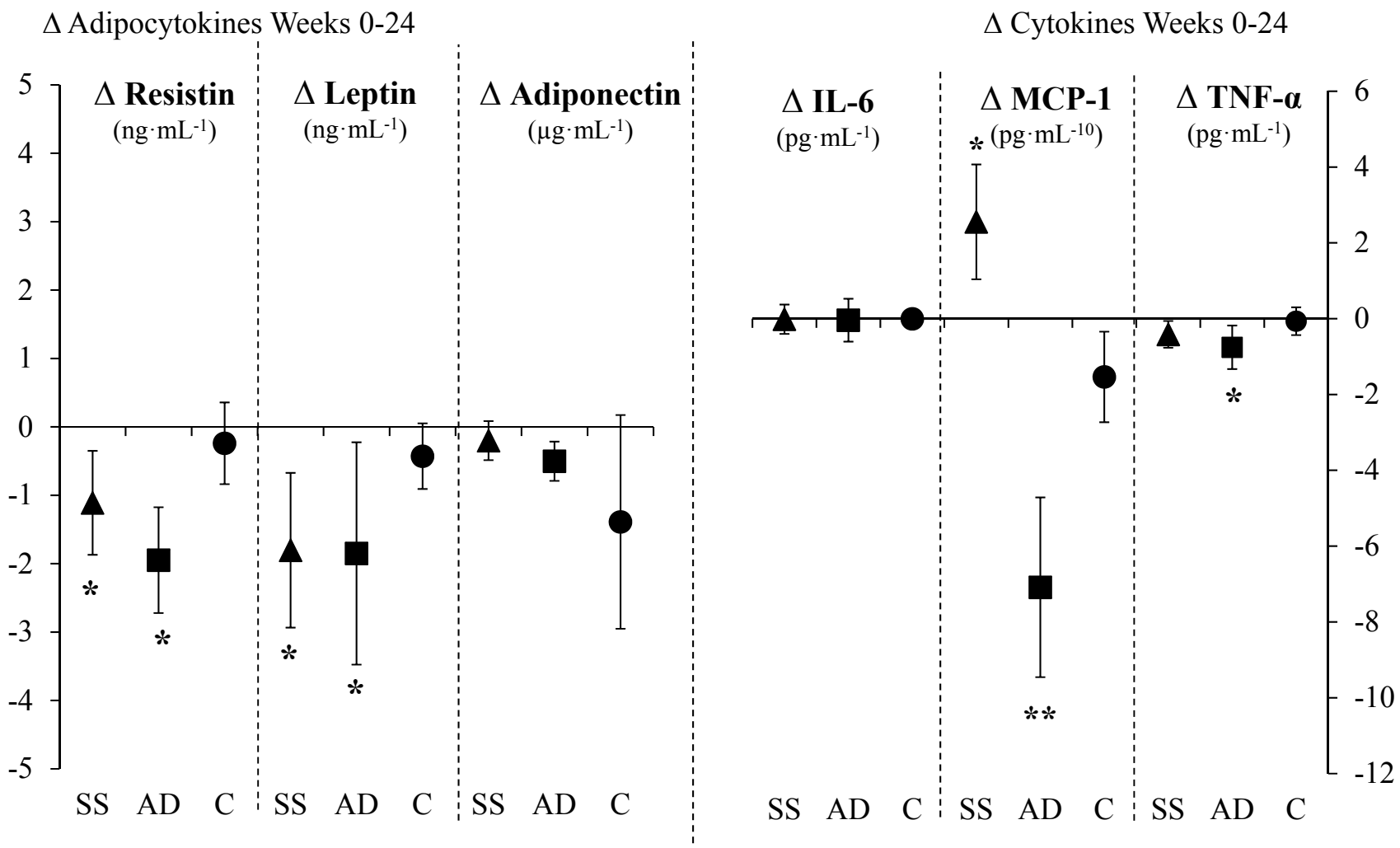

Fig 3. Mean (SD) changes in adipocytokines (left) and cytokines (right). * significant within-group change. SS = same session training, AD = alternating days training, C = Controls