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Both aerobic endurance and strength training programmes improve cardiovascular health in obese adults

Inga E. SCHJERVE*, Gjertrud A. TYLDUM*, Arnt E. TJØNNA*, Tomas STØLEN*, Jan P. LOENNECHEN†, Harald E. M. HANSEN*, Per M. HARAM‡, Garreth HEINRICH§, Anja BYE*, Sonia M. NAJJAR§, Godfrey L. SMITH*‖, Stig A. SLØRDAHL*†, Ole J. KEMI‖ and Ulrik WISLØFF*†

*Department of Circulation and Medical Imaging, Norwegian University of Science and Technology, Trondheim, Norway, †Department of Cardiology, St. Olav's Hospital, Trondheim, Norway, ‡Department of Cardiothoracic and Vascular Surgery, University Hospital North Norway, Tromsø, Norway, §Department of Physiology, Pharmacology, Metabolism and Cardiovascular Sciences, Medical University of Ohio, Toledo, OH, U.S.A., and ‖Institute of Biomedical and Life Sciences, University of Glasgow, Glasgow, U.K.

ABSTRACT

Regular exercise training is recognized as a powerful tool to improve work capacity, endothelial function and the cardiovascular risk profile in obesity, but it is unknown which of high-intensity aerobic exercise, moderate-intensity aerobic exercise or strength training is the optimal mode of exercise. In the present study, a total of 40 subjects were randomized to high-intensity interval aerobic training, continuous moderate-intensity aerobic training or maximal strength training programmes for 12 weeks, three times/week. The high-intensity group performed aerobic interval walking/running at 85–95% of maximal heart rate, whereas the moderate-intensity group exercised continuously at 60–70% of maximal heart rate; protocols were isocaloric. The strength training group performed 'high-intensity' leg press, abdominal and back strength training. Maximal oxygen uptake and endothelial function improved in all groups; the greatest improvement was observed after high-intensity training, and an equal improvement was observed after moderate-intensity aerobic training and strength training. High-intensity aerobic training and strength training were associated with increased PGC-1α (peroxisome-proliferator-activated receptor γ co-activator 1α) levels and improved Ca^{2+} transport in the skeletal muscle, whereas only strength training improved antioxidant status. Both strength training and moderate-intensity aerobic training decreased oxidized LDL (low-density lipoprotein) levels. Only aerobic training decreased body weight and diastolic blood pressure. In conclusion, high-intensity aerobic interval training was better than moderate-intensity aerobic training in improving aerobic work capacity and endothelial function. An important contribution towards improved aerobic work capacity, endothelial function and cardiovascular health originates from strength training, which may serve as a substitute when whole-body aerobic exercise is contra-indicated or difficult to perform.

Key words: calcium transport, cardiovascular health, endothelial function, exercise training, obesity.

Abbreviations: 1RM, one repetition maximum; ABTS, 2,2′-azinobis-(3-ethylbenzothiazoline-6-sulfonic acid); BMI, body mass index; BP, blood pressure; CRP, C-reactive protein; DBP, diastolic BP; FMD, flow-mediated dilation; HbA_{1c}, glycated haemoglobin; HDL, high-density lipoprotein; HR, heart rate; HR_{max}, maximal HR; LDL, low-density lipoprotein; NTG, nitroglycerine; PGC-1α, peroxisome-proliferator-activated receptor γ co-activator 1α; RER, respiratory exchange ratio; RPE, rate of perceived exertion; SERCA, sarcoplasmic/endoplasmic reticulum Ca^{2+} ATPase; $\dot{V}O_{2max}$, maximal oxygen uptake.

Correspondence: Dr Ulrik Wisløff (email ulrik.wisloff@ntnu.no).

INTRODUCTION

The global epidemic of overweight and obesity has become a major health, social and economical burden with 312 million people worldwide being obese [BMI (body mass index) $\geqslant 30\ kg/m^2$] and at least 1.1 billion people being overweight (BMI 25–29.9 kg/m^2) [1,2]. It has now been well established that obesity directly increases cardiometabolic risk by altering the secretion of adipokines and, indirectly, by promoting insulin resistance and its associated metabolic disorders, such as Type 2 diabetes. Moreover, obesity causes additional health problems as it is closely associated with the development and progression of coronary heart disease, certain forms of cancer, respiratory complications (e.g. obstructive sleep apnoea) and osteoarthritis [3]. Both overweight and obesity appear to be associated with low aerobic capacity and impaired endothelial function [4], of which both serve as strong and independent risk factors of mortality from cardiovascular and metabolic diseases [5–7]. Endurance training improves both aerobic capacity [8,9] and endothelial function [9,10], and is now increasingly recommended in the prevention and treatment of overweight and obesity [11].

Cardiovascular risk profiling attempts to establish the absence or presence of a number of risk factors that, together with overweight and obesity, contribute to the progression of cardiovascular disease, such as endothelial dysfunction, hypertension, inactivity and poor exercise capacity. Moreover, a number of well-established blood markers, such as cholesterol, triacylglycerols (triglycerides), creatinine, glucose and insulin resistance, are also used to complement the risk assessment. In general, exercise, in particular endurance exercise training, decreases cardiovascular risk, but an optimal training programme has not yet been identified. Similarly, criteria for the minimum protective exercise programme against overweight and obesity have not been established. Although the recommended exercise intensity spans the range 40–90 % of $\dot{V}O_{2max}$ (maximal oxygen uptake), most studies indicate that high-intensity exercise, i.e. toward the upper end of the range, results in larger aerobic and cardiovascular adaptations [8,12–14], and many rehabilitation programmes advocate the use of low-to-moderate-intensity exercise. Exercise training at an intensity of approx. 90 % of $\dot{V}O_{2max}$ is in the upper range of current guidelines for humans [11,15], and yields larger improvements in $\dot{V}O_{2max}$ than moderate-intensity exercise [8,9,16].

Although high-intensity exercise results in a lower percentage of fat oxidation during the exercise sessions, it is important to highlight that it is the total amount of fat oxidized that determines weight loss. In line with this, isocaloric training programmes at 45 and 85 % of $\dot{V}O_{2max}$ caused the same reductions in body fat and weight despite more fat (in percentage) being oxidized in the low-intensity group during the exercise sessions [17]. This is explained by the continued fat oxidation during the restitution phase; the higher the intensity of the exercise, the higher the fat oxidation post-exercise [18,19]. Interestingly, it has also been found that the resting metabolism is higher after strength training than endurance training with low or moderate intensity [19], but it is not known whether high-intensity endurance training yields the same effect on basal metabolism as strength training. Furthermore, little is known about the impact of strength training on cardiovascular health benefits and endothelial function compared with endurance training regimes, which have been found to improve the cardiovascular risk profile, including endothelial function [9].

Therefore the aim of the present study was to determine the efficiency of high-intensity aerobic training, moderate-intensity aerobic training and strength training in improving cardiovascular health in obese individuals.

MATERIALS AND METHODS

Subjects

A total of 40 subjects volunteered for the present study and underwent a thorough medical examination before inclusion. Inclusion criteria were males and females >20 years of age and who had a BMI >30 kg/m^2. Exclusion criteria were unstable angina pectoris, myocardial infarction within the last 12 months, decompensated heart failure, cardiomyopathy, severe valvular heart disease, considerable pulmonary disease, uncontrolled hypertension, kidney failure, orthopaedic and/or neurological limitations to exercise, surgery during the intervention period, drug or alcohol abuse, or participation in another research study. A compliance with the training programme of 70 % was also set as a criterion for completing the study.

The protocol was approved by the regional Ethical Committee for Medical Research, and the study conformed to the Declaration of Helsinki. Written informed consent was obtained from all subjects prior to inclusion in the study. For each individual, all pre- and post-tests were performed at the same time of the day.

Study design

The subjects were randomized to strength training ($n = 13$), continuous moderate-intensity aerobic training ($n = 13$) or high-intensity aerobic interval training ($n = 14$). Participants in all of the groups were encouraged to continue their normal nutritional habits during the study period. The procedures to make sure that the exercise programmes were as equal as possible with regard to energy expenditure have been described previously in detail by our group [8].

Over a 12-week period, the subjects performed three programmed exercise sessions per week; two supervised

by the study investigators in the research laboratory and one performed at home or in a gym, according to instructions. All tests and measurements described below were performed before (pre) and after (post) the training period.

Aerobic training

Exercise training in both the high-intensity and moderate-intensity groups was by treadmill walking or running. High-intensity training consisted of a 10 min warm-up period at 50–60 % of HR_{max} [maximal HR (heart rate)], followed by 4×4-min intervals at 85–95 % of HR_{max} with 3 min active breaks in between the intervals, consisting of walking or jogging at 50–60 % of HR_{max}. The exercise session was terminated by a 5 min cool-down period. The moderate-intensity group walked continuously for 47 min at 60–70 % of HR_{max} to ensure that the training protocols were isocaloric [8]. The subjects were instructed to control the intensity of the exercise by monitoring their HR and thereby adjusting the speed and/or incline of the treadmill to correspond to the preferred exercise intensity. For each session, HR, speed and incline were recorded. Participants were instructed to do the home training as outdoor uphill walking, in line with the laboratory-based training programme. The subjects were also instructed to register the intensity during their home sessions using the Borg RPE (rate of perceived exertion) 6–20 scale, whereby interval training should correspond to 16–18 and moderate training to 12–14 [9].

Strength training

The basis for the development of muscular strength is muscular hypertrophy and neural adaptations [20]. Before carrying out high-intensity strength training, subjects warmed up by treadmill walking for 15 min at 40–50 % of HR_{max}. In the present study, we chose a strength training regime of four series with five repetitions each, at approx. 90 % of 1RM (one repetition maximum), in a leg press apparatus to develop maximal strength mainly from neural adaptation with minimal weight gain due to muscular hypertrophy [20–22]. In addition, during each strength training session, the subjects performed additional abdominal and back exercises, consisting of three series of 30 repetitions with a 30 s break in between each series. At home or in the gym, the subjects warmed-up by walking and performed the abdominal and back strength programme on the floor and the leg strength programme in a leg press apparatus or as squats with appropriately loaded backpacks.

Endothelial function

Endothelial function was measured as FMD (flow-mediated dilation) using high-resolution vascular ultrasound (14 MHz echo Doppler probe; Vivid 7 System; GE Vingmed Ultrasound) according to the current guidelines [23,24]. The measurements were done on the brachial artery approx. 4.5 cm above the antecubital fossa. All measurements were performed in the morning after an 8-h fast. In addition, subjects were not allowed to use nicotine and coffee, or any other caffeine-containing beverages, for 12 h preceding testing. After a rest of 10 min in the supine position in a quiet air-conditioned room with a stable temperature of $22 \pm 1\,^{\circ}C$, the internal diameter of the brachial artery was assessed. Thereafter we inflated a pneumatic cuff (Hokanson SC10) on the upper arm to 250 mmHg for 5 min and deflated it to create an ischaemia-induced hyperaemic-elevated blood flow. Data were recorded 10 s after cuff release to measure peak blood flow, whereas artery diameter was recorded every 30 s for 5 min. The subjects then rested for 5 min until the baseline diameter was restored. Subsequently, endothelium-independent dilation was measured by administrating 500 μg of NTG (nitroglycerine) sublingually. To avoid confounding effects of arterial compliance and cyclic changes in arterial dimension, all measurements were obtained at the peak of the R-wave in the ECG. Diameters were measured from intima to intima using calipers with a 0.1 mm resolution. The mean of three diameter measurements and flow measurements were used in the calculation of FMD and flow responses. Maximal dilation was in each case observed 1 min after cuff release in each group, and those data are therefore presented in the results. Shear rate was calculated as blood flow velocity (cm/s) divided by diameter (cm), as described by Pyke and Tschakovsky [25]. All ultrasound images were analysed in a random order, using EchoPAC^{TM} (GE Vingmed Ultrasound) by an investigator who was blinded to the group allocation of the subjects.

Blood profile

Blood samples were taken after 8 h of fasting. Citrated and EDTA venous plasma samples were centrifuged at 1500 *g* for 10 min at 4 °C, and stored at − 80 °C for later analysis. Serum ferritin, triacylglycerols, HDL (high-density lipoprotein)-cholesterol, total cholesterol, haemoglobin, high-sensitive CRP (C-reactive protein), Na^+, K^+, creatinine, HbA_{1c} (glycated haemoglobin), glucose and insulin C-peptide were measured according to standard procedures. Glucose and insulin C-peptide were re-measured 2 h after an OGTT (oral glucose tolerance test; 75 g of glucose in 3 dl of water within 5 min). Total antioxidant status was measured in frozen serum samples using the colorimetric total antioxidant status assay (Randox Laboratories). The method is based upon the incubation of ABTS [2,2′-azinobis-(3-ethylbenzothiazoline-6-sulfonic acid)] with metmyoglobin and H_2O_2 to produce the radical cation $ABTS^+$. This radical has a stable blue/green colour, which is measured at 600 nm. Antioxidants present in the sample weaken the intensity of the colour in proportion to the concentration. The assay was performed using an automated

system (Cobas Mira), according to the manufacturer's instructions. The concentration of oxidized LDL (low-density lipoprotein) was measured in plasma using an oxidized LDL ELISA kit (Mercodia), which is a solid-phase enzyme immunoassay modified from the original method [26]. All samples were analysed in duplicates.

$\dot{V}O_{2max}$

$\dot{V}O_{2max}$ was measured during uphill treadmill walking or running (Woodway PPS 55 Med) using the Metamax II system (Cortex), as described previously [9]. A warm-up period for 10 min (50–60 % of HR_{max}) preceded the test. A levelling off of $\dot{V}O_2$, despite increased work load, and RER (respiratory exchange ratio) $\geqslant 1.05$ were used as criteria for $\dot{V}O_{2max}$. HR was measured during the test (Polar type 610; Polar Electro), and HR_{max} was defined by adding 5 beats/min to the highest HR value obtained during the $\dot{V}O_{2max}$ test.

Maximal leg strength

The maximal leg strength test was performed in a leg press machine with the knee joints at 90°. After a 10 min warm-up by treadmill walking at 50–60 % of HR_{max} and 10–15 warm-up repetitions in the leg press machine, weights where added until 1RM was reached. The number of repetitions necessary to reach 1RM varied between three and ten. Subjects rested for at least 1 min, but often for 2–3 min, before the next trial, depending upon how hard they felt the previous repetition was.

Biochemistry of muscle biopsies

Muscle biopsies were obtained from musculus vastus lateralis using a sterile 5-mm-diameter biopsy needle (Bergström) [27] under local anaesthesia (2 % lidocaine). A 5–10 mm incision was made, the Bergström needle was introduced into the muscle tissue, without using suction, and three to four cuts were made. If present, superficial blood was quickly removed, and the biopsy was frozen in liquid nitrogen and stored at −80 °C for later analysis.

Muscle biopsies were homogenized in lysis buffer and equal amounts of lysates were analysed by SDS/PAGE and Western blot analysis with goat polyclonal antibodies against PGC-1α (peroxisome-proliferator-activated receptor γ co-activator 1α) (K-15; Santa Cruz Biotechnology). Gels were re-probed with a monoclonal antibody against α-actin (Sigma) for normalization. Protein levels were detected by chemiluminescence and quantified by densitometry.

Skeletal muscle SERCA (sarcoplasmic/endoplasmic reticulum Ca^{2+} ATPase) activity

Decreased maximal rate of Ca^{2+} re-uptake into the sarcoplasmic reticulum is inversely related to increased skeletal muscle fatigue in individuals with low aerobic capacity [9]. To measure this, Ca^{2+} (50 μmol/l) was added to skinned muscle fibres from the vastus lateralis muscle to induce a rapid increase in $[Ca^{2+}]$, and the kinetics of the subsequent decline in $[Ca^{2+}]$ were analysed with Fura-2 on an epifluorescence microscope (Diaphot-TMD; Nikon) to assess maximum SERCA-1 and -2 transport capacity, as described previously [28].

Body composition and BP (blood pressure)

Dual-energy X-ray absorptiometry scanning (Hologic Discovery-A; Integrity Medical Systems) was used to measure body composition immediately after endothelial function was measured, i.e. after 8–9 h of fasting, to decrease large variations in hydration status. The waist/hip ratio was measured at the midpoint between the lower border of the ribs and the upper border of the pelvis (waist), and at the trochanter major (hip) [3,29]. BP was measured by a trained physiologist with a hand-held sphygmomanometer (Tycos) while the patient was sitting and had rested for at least 5 min in a quiet room. BP was measured at the same time of the day for each individual at pre- and post-test. The first reading was discarded and the mean of the next three consecutive readings with a coefficient of variation < 15 % was used in the present study, with additional readings if required.

Statistics

Before using parametric tests, the assumption of normality was verified using the Shapiro–Wilk W test. We used a two-way ANOVA to assess group–time interactions (group×time; 2×2). The Bonferroni post-hoc test adjusted for multiple comparisons was used to identify the statistical differences between the three groups. Pearson's correlation coefficient was used to determine potential relationships between FMD and parameters changing in parallel; only significant correlations are shown. Results are means ± S.E.M., and $P < 0.05$ indicates significant differences.

RESULTS

Baseline characteristics

The three groups did not differ significantly in any of the parameters at baseline (Table 1).

$\dot{V}O_{2max}$

A significant group–time interaction was found for $\dot{V}O_{2max}$ ($F = 26.4$, $P < 0.001$). $\dot{V}O_{2max}$ increased by 10, 16 and 33 % (all $P < 0.01$) in the strength training, moderate-intensity and high-intensity groups respectively, and thus high-intensity aerobic training had a greater effect than strength training or moderate-intensity aerobic training (Figure 1A). No difference in the increase in $\dot{V}O_{2max}$ occurred between the strength training and

Table 1 Baseline characteristics

Values are means ± S.E.M. No significant differences were observed between the groups. lbm, lean body mass.

Characteristic	Group		
	Strength training	Moderate-intensity training	High-intensity training
Age (years)	46.2 ± 2.9	44.4 ± 2.1	46.9 ± 2.2
Height (cm)	169.1 ± 2.6	167.7 ± 2.9	175.9 ± 2.2
Gender (*n*) (male/female)	2/11	3/10	3/11
Body weight (kg)	98.8 ± 4.5	104.1 ± 4.5	114.0 ± 5.7
BMI (kg/m^2)	34.5 ± 1.4	36.7 ± 1.4	36.6 ± 1.2
Body fat (%)	41.1 ± 1.3	43.6 ± 1.5	40.6 ± 1.4
Lean body mass (kg)	63.1 ± 5.7	66.3 ± 6.8	67.1 ± 3.8
Waist/hip ratio	0.90 ± 0.03	0.90 ± 0.03	1.00 ± 0.03
HR_{max} (beats/min)	173 ± 7	183 ± 2	170 ± 5
$\dot{V}o_{2max}$ (ml · lbm^{-1} · min^{-1})	38.3 ± 4.1	37.8 ± 4.8	39.7 ± 6.8
$\dot{V}o_{2max}$ (ml · kg^{-1} of body weight · min^{-1})	25.4 ± 1.9	25.1 ± 1.4	23.6 ± 1.3
RER	1.12 ± 0.04	1.14 ± 0.02	1.14 ± 0.03
1RM (kg)	174.1 ± 13.0	162.8 ± 19.1	180.6 ± 13.4
Glucose (mmol/l)	5.4 ± 0.3	6.2 ± 1.1	5.2 ± 0.1
Cholesterol (mmol/l)	6.3 ± 0.3	5.8 ± 0.3	6.3 ± 0.2
Triacylglycerols (mmol/l)	2.0 ± 0.3	1.5 ± 0.3	1.2 ± 0.1
HDL-cholesterol (mmol/l)	1.3 ± 0.1	1.4 ± 0.1	1.3 ± 0.1
Haemoglobin (g/dl)	14.4 ± 0.3	13.9 ± 0.3	14.5 ± 0.3

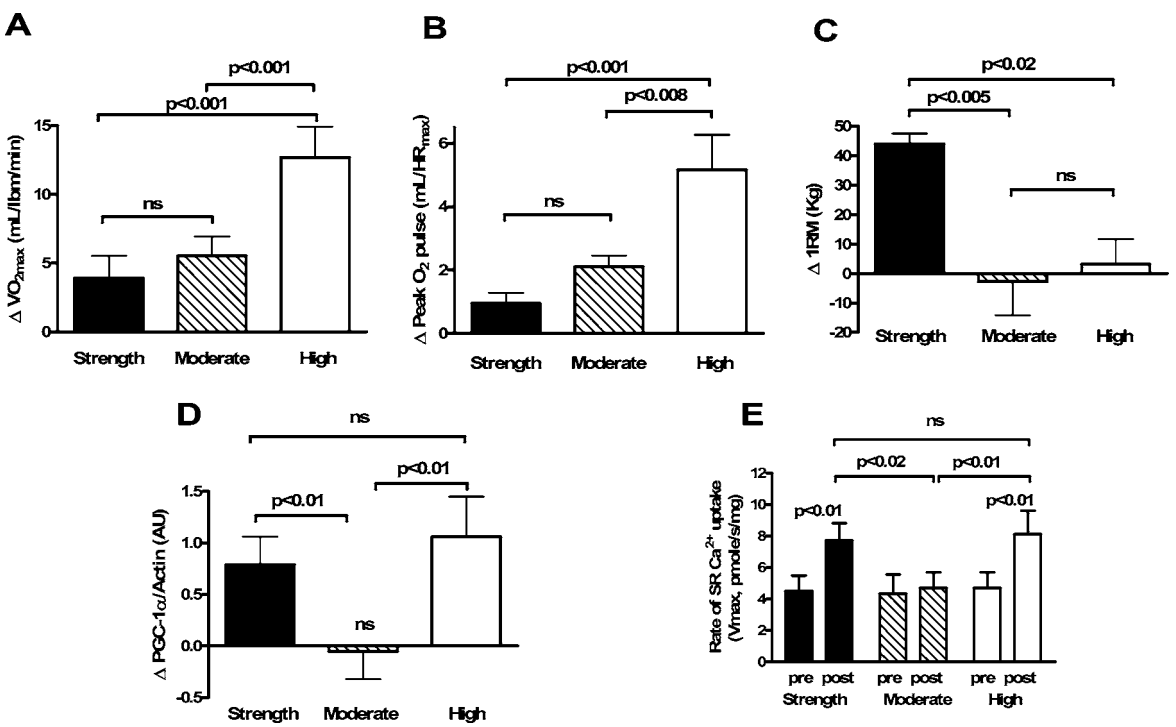

Figure 1 $\dot{V}o_{2max}$ **(A), peak O_2 pulse (B), 1RM (C), PGC-1α level (D) and SERCA activity (E) in subjects undergoing high-intensity aerobic exercise, moderate-intensity aerobic exercise or strength training**

Values are means ± S.E.M. ns, not significant. lbm, lean body mass.

moderate-intensity groups (Figure 1). The RER was not significantly different from that measured at pre-test in any of the groups (Table 1). Post-test RERs were 1.10 ± 0.02, 1.12 ± 0.01, and 1.10 ± 0.03 in the strength training, moderate-intensity and high-intensity groups respectively. All subjects satisfied the criteria for $\dot{V}O_{2max}$, i.e. a levelling-off despite increased work load and a RER > 1.05, as well as being < 5 beats from actual HR_{max}. HR_{max} was reached both at pre-test (Table 1) and post-test (172 ± 4,181 ± 4 and 171 ± 3 beats/min for the strength training, moderate-intensity and high-intensity groups respectively).

A significant group–time interaction was found for the O_2 pulse ($F = 22.3$, $P < 0.001$). Peak O_2 pulse (in ml/HR_{max}) improved in all groups ($P < 0.01$), indicating that the maximal stroke volume increased [30]. No difference was observed in the increase in the O_2 pulse between the strength training and moderate-intensity groups, but the high-intensity group had a greater improvement in peak O_2 pulse compared with the other two groups (Figure 1B).

Maximal strength

A significant time–group interaction was observed for maximal strength ($F = 8.1$, $P < 0.01$). The strength training group improved 1RM by 25 % ($P < 0.001$), whereas there were no changes in the moderate-intensity or high-intensity groups (Figure 1C).

Skeletal muscle PGC-1α and SERCA

PGC-1α is a master regulator of mitochondrial biogenesis and enzymes of fatty acid metabolism [31,32]. A time–group interaction was observed for the level of PGC-1α ($F = 6.1$, $P < 0.01$).

Both strength training and high-intensity aerobic training increased PGC-1α protein levels ($P < 0.01$), but moderate-intensity aerobic training did not (Figure 1D). The maximal rate of Ca^{2+} re-uptake into the sarcoplasmic reticulum by SERCA in skeletal muscles increased by 73 and 72 % after high-intensity aerobic training and strength training respectively, but moderate-intensity aerobic training had no effect (Figure 1E).

Endothelial function

A significant time–group interaction was found for FMD ($F = 5.9$, $P < 0.01$). FMD improved significantly ($P < 0.001$) in all of the groups (Figures 2A, 2C and 2E). High-intensity aerobic training had a significantly greater effect on endothelial function compared with strength training and moderate-intensity groups ($P < 0.05$), although there was no statistical difference between the latter two groups (Figures 2A, 2C and 2E). The resting diameter of the brachial artery was similar in all three groups and did not change during the experimental period (Table 2). Additionally, peak blood flow did not change (Figures 2B, 2D and 2F), so that shear rates were similar between the three groups and were not influenced by the training regimens (Table 2). Therefore the observed changes in FMD were not due to a change in artery diameter or shear rate, as the same group differences were seen after normalizing FMD for potential differences in shear rate (Figures 2B, 2D and 2F). Exercise training had no impact on endothelium-independent dilation induced by NTG (Table 2, and Figures 2A, 2C and 2E).

Blood markers

A group–time interaction was observed for oxidized LDL ($F = 4.2$, $P < 0.05$). Oxidized LDL decreased significantly after strength training ($P < 0.005$) and moderate-intensity aerobic training ($P < 0.04$), but not after high-intensity aerobic training (Figure 3A). Only strength training increased total antioxidant status ($P < 0.03$; Figure 3B), but no group–time interaction was observed. None of the traditional blood markers were affected by any of the training programmes, as serum ferritin, triacylglycerols, HDL-cholesterol, total cholesterol, haemoglobin, high-sensitive CRP, Na^+, K^+, creatinine, HbA_{1c}, glucose and insulin C-peptide remained unchanged in all of the groups.

Body composition

A small, but significant, group–time interaction for body weight ($F = 4.4$, $P < 0.05$) was observed. Body weight decreased by 3 % ($P < 0.005$) and 2 % ($P < 0.04$) after moderate-intensity and high-intensity aerobic training respectively, whereas no change was observed in the strength training group (Figure 4A). A decrease in BMI was observed in both the moderate-intensity (from 36.7 ± 1.4 to 35.6 ± 1.4 kg/m^2; $P < 0.007$) and high-intensity (from 36.6 ± 1.2 to 36.0 ± 1.2 kg/m^2; $P < 0.04$) groups, whereas strength training had no effect on BMI. Body fat decreased 2.5 % ($P < 0.03$) and 2.2 % ($P < 0.02$) in the moderate-intensity and high-intensity groups respectively, but not in the strength training group (Figure 4B). There were no changes in the waist/hip ratio in any of the groups (results not shown).

BP

No changes were observed for SBP (systolic BP) (results not shown). In contrast, DBP (diastolic BP) decreased by 9 % ($P < 0.02$) in the moderate-intensity group and by 7 % ($P < 0.002$) in the high-intensity group, whereas no change was observed in the strength training group (Figure 4C). The group–time interaction was significant for DBP ($F = 2.0$, $P < 0.05$).

Correlations

We observed a low, but significant, correlation between FMD and DBP ($R = -0.4$, $P = 0.044$), and a correlation between FMD and $\dot{V}O_{2max}$ ($R = 0.54$, $P < 0.001$).

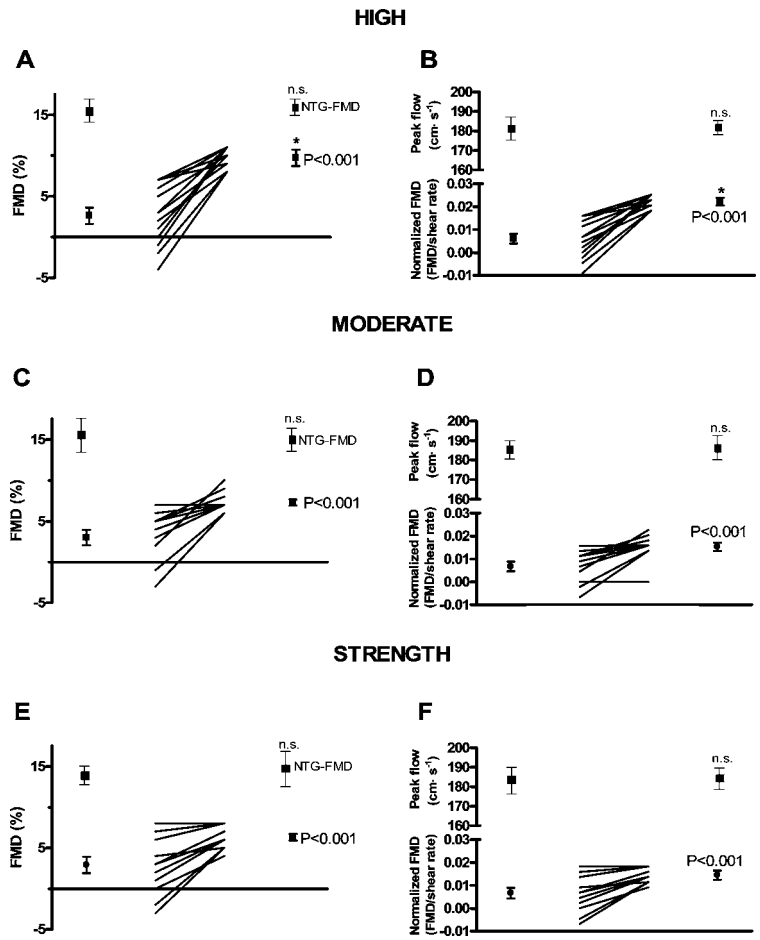

Figure 2 Endothelial function in subjects undergoing high-intensity aerobic training, moderate-intensity aerobic training or strength training

Results are means ± S.E.M. (A, C and E) FMD, as a measure of endothelial function, presented as the percentage increase from baseline diameter of the vessel. (B, D and F) FMD normalized for shear rate. *Significantly greater improvement ($P < 0.05$) than after strength training or moderate-intensity aerobic exercise. NTG-FMD, NTG-induced FMD; n.s., not significant.

Table 2 Baseline, peak diameter and shear rate in the brachial artery

Values are means ± S.E.M. Shear rate is calculated as flow (cm/s) divided by artery diameter (cm).

	Group					
	Strength training		Moderate-intensity training		High-intensity training	
Measurement	Pre-	Post-	Pre-	Post-	Pre-	Post-
Baseline diameter (cm)	0.420 ± 0.03	0.410 ± 0.04	0.400 ± 0.05	0.401 ± 0.04	0.402 ± 0.04	0.391 ± 0.03
Peak diameter (cm)	0.432 ± 0.04	0.436 ± 0.03	0.412 ± 0.03	0.430 ± 0.02	0.411 ± 0.03	0.429 ± 0.02
Peak NTG diameter (cm)	0.481 ± 0.06	0.470 ± 0.05	0.461 ± 0.06	0.472 ± 0.07	0.461 ± 0.04	0.462 ± 0.05
Shear rate (flow/diameter)	441 ± 37	438 ± 40	446 ± 29	442 ± 36	438 ± 39	437 ± 38

DISCUSSION

The major findings of the present study are that (i) high-intensity aerobic interval training was better at improving endothelial function than either continuous moderate-intensity aerobic training or strength training, and (ii) strength training and moderate-intensity aerobic training were equally efficient in improving endothelial function in obese adults, albeit less efficiently than high-intensity aerobic interval training. This demonstrates that

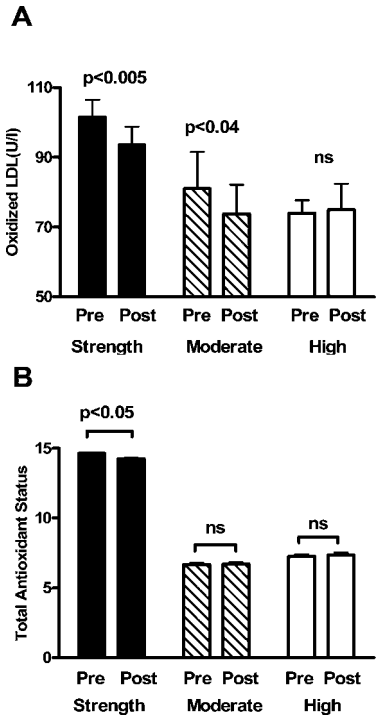

Figure 3 Oxidized LDL (A) and total antioxidant status (B) in subjects undergoing high-intensity aerobic exercise, moderate-intensity aerobic exercise or strength training

Values are means ± S.E.M. *P* values indicate a significant difference between pre- and post-exercise. ns, not significant.

it is possible to reverse impaired endothelial function in subjects that are hindered from performing whole-body endurance training.

FMD (endothelial function)

Shear stress to the arterial wall stimulates endothelial production of NO which subsequently induces vessel dilation. The dependence of exercise intensity suggests that high-intensity aerobic training induces a greater shear stress during exercise compared with moderate-intensity aerobic training, consistent with previous results [9]. Results from the present study suggest that strength training appears to induce a shear stress similar to that associated with moderate-intensity aerobic training. It cannot be ruled out that the improvement in endothelial function in the strength training group is caused by the 15 min warm-up periods; however, if this was the case, it would mean that 15 min of walking at 40–50 % of HR_{max} would equal 47 min at 60–70 % of HR_{max} but, because of the dose–response relationship between moderate- and high-intensity aerobic training reported in the present study and elsewhere [8,9,14,16] in improving endothelial function, this assumption is unlikely, although it should be tested in future studies. In fact, it has been demonstrated that 1 year of strength training improves endothelial function in overweight women, independently of changes in major cardiovascular risk factors such as BMI, body composition, BP, fasting blood lipids, and fasting blood glucose and insulin [33]. In contrast, 12 weeks of whole-body resistance training in healthy young men did not change endothelial function (FMD) or shear rate, but increased arterial diameter and, hence, blood flow [34]. Thus it seems likely that strength training may improve endothelial function in overweight and obese subjects, but not in healthy subjects. The underlying mechanisms for why strength training should affect endothelial function in these populations remain largely unknown, although improved antioxidant status after strength training may indicate that oxidative stress by ROS (reactive oxygen species) and oxidized LDL is decreased, which would enhance the bioavailability of NO. However, these findings should be interpreted cautiously as, for unknown reasons, the baseline values for total antioxidant status in the strength training group were twice that in the other two groups at baseline (and at post-test), and this may well be the reason that only strength training improved antioxidant status. Further studies are needed to determine whether improved FMD due to strength training involves actual changes in antioxidant status. Antioxidative effects of aerobic exercise training have been reported previously in patients

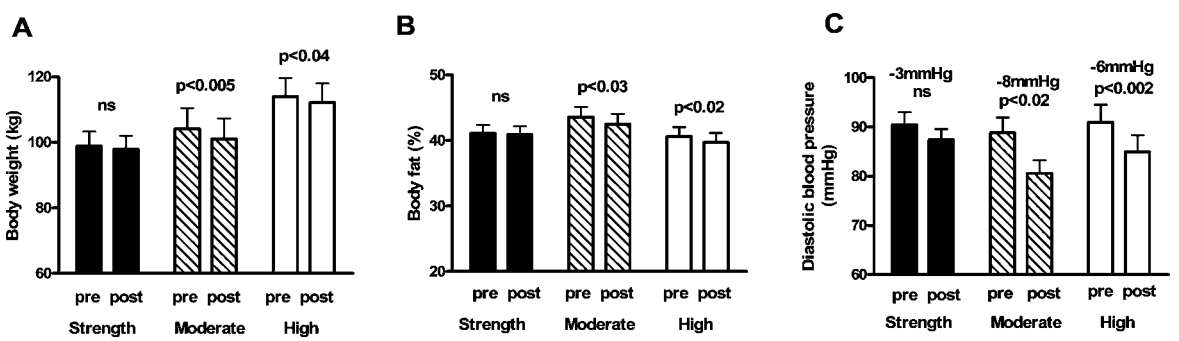

Figure 4 Body weight (A), body fat (B), expressed as a percentage of total body weight, and DBP (C) in subjects undergoing high-intensity aerobic exercise, moderate-intensity aerobic exercise or strength training

Values are means ± S.E.M. *P* values indicate a significant difference between pre- and post-exercise. ns, not significant.

with heart failure [9,35]; however, why no effects of high-intensity or moderate-intensity aerobic training were found in the present study is unknown, but may be linked to the different study populations.

Body composition and BP

High BP is associated with increased risk of stroke and ischaemic heart disease [36]. We found that both high-intensity and moderate-intensity aerobic training, but not strength training, significantly decreased DBP by 6–8 mmHg. On the basis of a meta-analysis of 1 million adults, this would translate to a 30% lower risk of premature deaths [36]. The observed correlation between FMD and DBP are consistent with other studies [37,38] and indicate that improved endothelial function after the endurance training regimens contributes to the decreased DBP. On the other hand, endothelial function was also improved after strength training, which was not linked to a decrease in DBP. This suggests that other factors may be more important in the regulation of BP than changes in FMD and should be studied further.

In the present study, both of the aerobic training regimens caused small, but significant, decreases in body weight. Although both obesity and aerobic capacity are strong and independent prognostic markers of cardiovascular mortality, the link between aerobic capacity and mortality appears to be stronger [39], and it has therefore been suggested that improving aerobic capacity is more important than losing weight [40].

Oxygen uptake

As expected, the greatest improvement in $\dot{V}O_{2max}$ was observed after high-intensity aerobic interval training, but, surprisingly, strength training increased $\dot{V}O_{2max}$ to a smaller, but not statistically different, extent compared with moderate-intensity aerobic training. High intensity yielding a higher effect than moderate intensity during an aerobic training programme confirms previous studies in both healthy subjects [16] and patients with post-infarction heart failure [9]. Previous studies involving patients with coronary artery disease employing aerobic interval exercise with elements of high intensity, as in the present study, have also indicated that the development of $\dot{V}O_{2max}$ depends on exercise intensity [8,41]. Although the various studies are not directly comparable due to different exercise protocols, they demonstrate however that high-intensity aerobic exercise is associated with the greatest improvements in $\dot{V}O_{2max}$. The present study also suggests that the stroke volume of the heart is a mediator of $\dot{V}O_{2max}$, as indicated by a greater O_2 pulse after high-intensity aerobic interval training compared with the other two groups.

The reason as to why strength training increases $\dot{V}O_{2max}$ is not fully understood, but it may be that a greater 1RM allows for more ordinary daily activities, such as walking, and thereby permits an increase in general activity levels. This was, however, not controlled for in the present study. In addition, the possibility remains that the improvement in $\dot{V}O_{2max}$ after strength training was caused by the 15 min low-intensity warm-up period, although this would be unlikely, as discussed above.

Skeletal muscle

In line with our recent studies in patients with heart failure [9] or the metabolic syndrome [42], PGC-1α, a master regulator of energy metabolism [31,32,41], increased after high-intensity aerobic training, but not after moderate-intensity aerobic training. The observation that strength training also increased PGC-1α levels is, however, novel. The reason for the differences in training response is unknown, but it is conceivable that the ischaemic conditions in skeletal muscle during high-intensity aerobic interval training and strength training are a considerable stimulus for the up-regulation of muscle mechanisms that improve aerobic metabolism. Our hypothesis was supported by the findings that exercise with restricted, rather than non-restricted, blood flow induced a greater increase in PGC-1α mRNA levels [43]. High-intensity interval and maximal strength training would also restrict blood flow and/or induce local hypoxia in the skeletal muscles. This may well be the mechanism for strength training improving $\dot{V}O_{2max}$ in the present study and resting metabolism in other studies [44].

Interestingly, decreased maximal rate of Ca^{2+} re-uptake into the sarcoplasmic reticulum increased only after strength training and high-intensity aerobic interval training. As those were the only training programmes that also increased PGC-1α levels, it may suggest that increased metabolic and ATP-producing capacity [32] allows for a concomitant increase in the capacity of the SERCA to transport Ca^{2+}, as it is an ATPase ('ATP user'). Although not investigated in the present study, this may suggest that high-intensity aerobic interval and strength training programmes improve overall contractile performance in the skeletal muscle.

Conclusions

The present study demonstrates that both aerobic exercise training at either high or moderate intensities and high-intensity strength training improve endothelial function and decrease the cardiovascular risk profile in obese adults. However, high-intensity aerobic interval training results in a greater improvement in endothelial function and a decrease in the cardiovascular risk profile in these subjects than moderate-intensity aerobic training or strength training. Maximal strength training improved endothelial function and $\dot{V}O_{2max}$ equally as efficiently as moderate-intensity aerobic training. Improved endothelial function after maximal strength training occurred in conjunction with improved antioxidant status and decreased levels of oxidized LDL, indicating a possible mechanistic explanation. Moreover, enhanced $\dot{V}O_{2max}$

after strength training and high-intensity aerobic interval training was associated with higher expression of PGC-1α and improved SERCA activity in the skeletal muscle. These observations demonstrate that it might be possible to reverse impaired endothelial function, decrease cardiovascular risk and improve exercise capacity in subjects that have difficulty performing whole-body aerobic training.

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