

Changes in cardiorespiratory fitness and coronary heart disease risk factors following 24 wk of moderate- or high-intensity exercise of equal energy cost

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Changes in cardiorespiratory fitness and coronary heart disease risk factors following 24 wk of moderate- or high-intensity exercise of equal energy cost. *J Appl Physiol* 98: 1619–1625, 2005. First published January 7, 2005; doi:10.1152/jappphysiol.01310.2004.—This study was designed to investigate the effect of exercise intensity on cardiorespiratory fitness and coronary heart disease risk factors. Maximum oxygen consumption ($\dot{V}O_{2\max}$), lipid, lipoprotein, and fibrinogen concentrations were measured in 64 previously sedentary men before random allocation to a nonexercise control group, a moderate-intensity exercise group (three 400-kcal sessions per week at 60% of $\dot{V}O_{2\max}$), or a high-intensity exercise group (three 400-kcal sessions per week at 80% of $\dot{V}O_{2\max}$). Subjects were instructed to maintain their normal dietary habits, and training heart rates were represcribed after monthly fitness tests. Forty-two men finished the study. After 24 wk, $\dot{V}O_{2\max}$ increased by 0.38 ± 0.14 l/min in the moderate-intensity group and by 0.55 ± 0.27 l/min in the high-intensity group. Repeated-measures analysis of variance identified a significant interaction between monthly $\dot{V}O_{2\max}$ score and exercise group ($F = 3.37$, $P < 0.05$), indicating that $\dot{V}O_{2\max}$ responded differently to moderate- and high-intensity exercise. Trend analysis showed that total cholesterol, low-density lipoprotein cholesterol, non-high-density lipoprotein cholesterol, and fibrinogen concentrations changed favorably across control, moderate-intensity, and high-intensity groups. However, significant changes in total cholesterol (-0.55 ± 0.81 mmol/l), low-density lipoprotein cholesterol (-0.52 ± 0.80 mmol/l), and non-high-density lipoprotein cholesterol (-0.54 ± 0.86 mmol/l) were only observed in the high-intensity group (all $P < 0.05$ vs. controls). These data suggest that high-intensity training is more effective in improving cardiorespiratory fitness than moderate-intensity training of equal energy cost. These data also suggest that changes in coronary heart disease risk factors are influenced by exercise intensity.

training; lipids; lipoproteins; non-high-density lipoprotein cholesterol; fibrinogen

LOW CARDIORESPIRATORY FITNESS is a powerful predictor of coronary heart disease (CHD) mortality, even among healthy middle-aged men (17, 33). While it is subject to a genetic component (5), cardiorespiratory fitness is increased by exercise training, regardless of age, gender, race, and initial fitness level (39). In a 5-yr study of 9,777 men, those who increased their physical fitness

enjoyed a substantial reduction in cardiovascular disease risk compared with men who remained inactive and unfit (4).

Meta-analyses suggest that CHD mortality is lower in highly fit individuals than in moderately fit individuals (15, 46). Because of this dose-response relationship, it is important that exercise guidelines explain how cardiorespiratory fitness is optimized. Current guidelines suggest that changes in cardiorespiratory fitness are similar in high-intensity interventions and in moderate-intensity interventions of longer duration if the energy cost of exercise is similar (3). However, those randomized controlled trials that have compared interventions of equal energy cost have concluded that high-intensity training is more effective in improving cardiorespiratory fitness (18, 23).

The comparison of isocaloric interventions is also appropriate when investigating the effect of exercise intensity on CHD risk factors. In contrast to observational designs, experimental studies have not identified dose-response relationships because of the relative homogeneity of interventions and because few authors have compared changes in CHD risk factors in interventions of different intensity (7, 19). In an uncontrolled trial, Crouse et al. (9) found similar changes in lipids and apolipoproteins following 24 wk of moderate- or high-intensity exercise. In a larger randomized controlled trial, Kraus et al. (23) also concluded that changes in lipoproteins were independent of exercise intensity. However, these studies did not regularly re-prescribe exercise to accommodate increases in cardiorespiratory fitness, which are rapid in previously sedentary men (20, 41).

Given that experimental evidence is limited and in contrast to current guidelines (3), this study was designed to test the hypothesis that similar improvements in cardiorespiratory fitness are derived from moderate- or high-intensity exercise interventions of equal energy cost. Unlike previous randomized controlled trials, we prescribed exercise at baseline and after monthly fitness tests. This design also offered an opportunity to investigate the effect of exercise intensity on CHD risk factors.

MATERIALS AND METHODS

Subjects. Participants were recruited from large employers in the city of Canterbury, UK. With permission, a letter was sent to male

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employees soliciting their interest in taking part in a 24-wk study of the effects of exercise training on CHD risk factors. This letter stated that eligible men were aged 30–45 yr, nonsmokers or ex-smokers with at least 2 yr of abstinence, not regularly engaged in any sustained work- or leisure-time physical activity sufficient to induce sweating or heavy breathing, and willing to accept random assignment. Daily energy expenditure was estimated using a 7-day physical activity recall questionnaire (37), and volunteers were excluded if the questionnaire revealed any participation in very hard activities or >2-h participation in hard activities, as these are likely to induce a training effect (45). Volunteers were examined by a cardiologist and were excluded if there was evidence of cardiovascular disease or if two or more of the following risk factors were present: family history of heart disease or sudden death, fasting cholesterol >6.2 mmol/l, fasting glucose >6.1 mmol/l, systolic blood pressure >140 mmHg, or diastolic blood pressure >90 mmHg. The East Kent Hospitals Local Research Ethics Committee approved this study, and all men signed a statement of informed consent.

Interventions and randomization. After screening, subjects were randomly assigned to a nonexercise control group, a moderate-intensity exercise group, or a high-intensity exercise group. All participants were instructed not to change their dietary or lifestyle habits other than prescribed. In both exercise groups, cycling intensity and duration were gradually increased during the first 8 wk of training. Thereafter, the moderate-intensity group completed three 400-kcal sessions per week at 60% of maximum oxygen consumption ($\dot{V}O_{2\max}$), whereas the high-intensity group completed three 400-kcal sessions per week at 80% of $\dot{V}O_{2\max}$. The training volume was chosen because the expenditure of 1,200 kcal/wk is likely sufficient to improve lipids and lipoproteins (14). The intensity levels were chosen because they are in keeping with those recommended for the improvement of cardiorespiratory fitness (3). All exercise sessions were accompanied by a standardized 5-min warm-up and cool-down. Exercise testing took place at the exercise physiology laboratory of Canterbury Christ Church University College, and exercise training was undertaken ad libitum at the institution's supervised fitness center during normal opening hours.

Subjects were recruited by the principal investigator but were assigned to exercise or control groups by a third party using computer-generated sequences of random numbers. The principal investigator and the participants were blinded to the randomization sequence, and assignment was only divulged after baseline testing. Baseline and postintervention blood analyses were also undertaken by individuals blinded to group allocation.

Outcomes. At baseline and after 24 wk, venous blood was drawn following a 12-h overnight fast and 24-h abstinence from vigorous activity. Standard enzymatic measurements of total cholesterol, high-density lipoprotein cholesterol (HDL-C), and triglyceride concentrations were made on fresh serum samples using a Roche Integra 800 analyzer (Roche Diagnostics, Lewes, UK). Non-HDL-C concentration was determined by subtracting HDL-C concentration from total cholesterol concentration (10). Fibrinogen concentration was determined by Clauss estimation using commercial reagents (Biomerieux, Basingstoke, UK) on a Sysmex 7000 analyzer (Sysmex, Milton Keynes, UK). Low-density lipoprotein cholesterol (LDL-C) concentration was estimated using the Friedewald equation (16). All assays demonstrated a between-day coefficient of variation of <3% and were performed in laboratories holding Clinical Pathology Accreditation (UK).

The same trained investigator measured triceps, biceps, subscapular, and suprailiac skinfolds at baseline and postintervention using Harpenden callipers (British Indicators, Luton, UK). Percent body fat was estimated using a standard equation for men (13). Waist girth was measured with an inelastic tape in a horizontal plane at the narrowest part of the torso (6). Socioeconomic status was determined during an interview in accordance with the National Statistics Socio-Economic Classification manual (34) and 2000 coding index (35). The three-class version of the classification was used: managerial and profes-

sional occupations, intermediate occupations, and routine and manual occupations.

Physical fitness was indicated by the $\dot{V}O_{2\max}$ achieved while cycling to exhaustion during an incremental test lasting ~8–16 min. Respiratory gasses were measured by a mass spectrometer (model EX670, Morgan Medical, Gillingham, UK) that was validated periodically using a Gas Exchange System Validator (Medical Graphics), which was first described by Huszczuk et al. (21). Typically, high agreement was observed between expected and reported values. For example, in a recent validation test, oxygen consumption ($\dot{V}O_2$) varied by -3.1, -2.8, and -1.8% at low, medium, and high metabolic rates, respectively.

Maximum tests were also used to prescribe exercise. Maximum heart rate, $\dot{V}O_{2\max}$, and average values during the last 15 s of each 2-min stage were used to derive regression equations of heart rate against $\dot{V}O_2$ for each subject. Exercise programs cited a target heart rate associated with 60 or 80% of $\dot{V}O_{2\max}$, and training heart rates were prescribed on a monthly basis because improvements in fitness are rapid in previously sedentary men (20). To monitor adherence, exercise programs were also used as training diaries, which were signed by a fitness instructor after each completed session. Energy expenditure was determined from $\dot{V}O_2$, assuming an energy cost of 5 kcal/l of oxygen. For example, an individual with a $\dot{V}O_{2\max}$ of 3.0 l/min uses 9.0 kcal/min when exercising at 60% of $\dot{V}O_{2\max}$ [(3.0 × 60%) × 5] and will expend 400 kcal in 44.4 min (400/9). At 80% of $\dot{V}O_{2\max}$, the same individual uses 12 kcal/min and expends 400 kcal in 33.3 min.

Statistical analysis. Sample size was estimated using the nomogram described by Altman (2), with change in fitness as the primary outcome variable. In a 20-wk study of moderate-to-high-intensity endurance training, $\dot{V}O_{2\max}$ increased by 0.44 ± 0.23 l/min in 287 previously sedentary men (39). The nomogram suggests that there is greater than a 90% probability at the 5% level of significance of detecting the same change in fitness with groups of only 10 individuals. In anticipation of a 30% dropout (24), we aimed to recruit 20 individuals to each group. A sample of this size would also give a 60% probability at the 5% level of significance of detecting an exercise-induced change in LDL-C of 0.35 ± 0.50 mmol/l. Whereas such a change is greater than that commonly observed, the present intervention was more demanding than most randomized controlled trials (19).

Baseline group scores for socioeconomic status were compared using the χ^2 test. All other baseline-dependent variables were compared using general linear model (GLM) ANOVA and, where appropriate, Bonferroni post hoc tests. Because monthly data were available, the effect of exercise training on $\dot{V}O_{2\max}$ was determined using a repeated-measures GLM-ANOVA with group as the between-subjects factor. All other between-group 24-wk changes were analyzed using GLM-ANOVA and, where appropriate, Bonferroni post hoc tests. To identify dose-response relationships, polynomial trend analysis was used to determine whether mean changes in CHD risk factors were linear across control, moderate-intensity, and high-intensity groups. Normality was tested by examining normal plots of the residuals in ANOVA models. Residuals were regarded as normally distributed if Shapiro-Wilk tests were not significant (2). To better approach normality, a \log_{10} transformation was made of the fibrinogen data. Within-group comparisons were made using paired *t*-tests. Relationships between dependent variables were determined using Pearson's correlation coefficient. All data were analyzed using SPSS for Windows, version 11.0 (SPSS, Chicago, IL).

RESULTS

The flow of participants through the study is shown in Fig. 1. Subjects were recruited and enrolled in the study between March and October 2002. Ongoing recruitment and random assignment ensured that any seasonal variation in lipids and lipoproteins was distributed among groups. Dropout was sim-

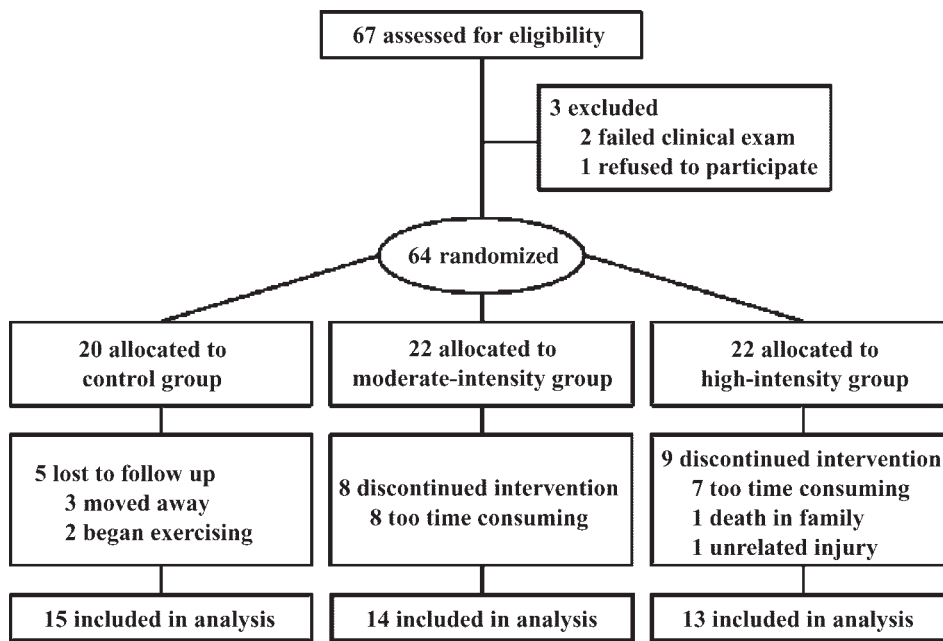


Fig. 1. Flow of participants through the study.

ilar in both exercise groups, and no within-group differences were observed between dropouts and men who finished the study for cardiorespiratory fitness, anthropometric variables, or CHD risk factors ($P \geq 0.05$). In men who finished the study, adherence was $99.4 \pm 1.0\%$ in the moderate-intensity group and $99.3 \pm 0.9\%$ in the high-intensity group.

The physical and physiological characteristics of those who finished the study are shown in Table 1. At baseline, exercise and control groups did not differ significantly in age, body composition, waist girth, or cardiorespiratory fitness. The groups were also of similar socioeconomic status: 80% of controls, 79% of moderate-intensity exercisers, and 85% of high-intensity exercisers were employed in managerial and professional occupations. Physical activity, as indicated by estimates of daily energy expenditure, was lower in the high-intensity group than in the control group, but this occurrence is likely explained by differences in weight. When expressed relative to weight, physical activity was no different in men allocated to control, moderate-intensity, and high-intensity groups (35.5 ± 1.4 , 36.6 ± 2.3 , and 35.2 ± 2.5 kcal·kg⁻¹·day⁻¹, respectively). After 24 wk, $\dot{V}O_{2\max}$ increased by 0.38 ± 0.14 l/min in the moderate-intensity group and by 0.55 ± 0.27 l/min in the high-intensity group (both $P < 0.001$ vs. controls). When expressed relative to body weight, $\dot{V}O_{2\max}$ increased by 4.85 ± 2.10 ml·kg⁻¹·min⁻¹ in the moderate-intensity group and by 7.14 ± 3.74 ml·kg⁻¹·min⁻¹ in the high-intensity group (both $P < 0.001$ vs. controls). The repeated-measures GLM-ANOVA identified a significant interaction between monthly $\dot{V}O_{2\max}$ score and exercise group ($F = 3.37$, $P < 0.05$), indicating that $\dot{V}O_{2\max}$ responded differently to moderate- and high-intensity exercise. Indeed, the graph of the improvement in $\dot{V}O_{2\max}$ shows a divergence between the groups after 8 wk, the point at which exercise intensity was increased to 80% of $\dot{V}O_{2\max}$ in the high-intensity group (Fig. 2). After 24 wk, there were modest but statistically significant changes in body fat in both exercise groups. High-intensity exercise was also accompanied by a significant change in waist girth. During the

intervention, physical activity increased significantly within both exercise groups. However, the change in energy expenditure was not significantly different between the moderate- and high-intensity groups (108 ± 78 vs. 135 ± 83 kcal/day, $P = 1.00$).

Table 1. Physical and physiological characteristics at baseline and after 24 wk of moderate- or high-intensity training

	Control Group	Moderate Intensity	High Intensity
<i>n</i>	15	14	13
Age, yr			
Baseline	40±4	41±3	41±4
Height, cm			
Baseline	183±5	180±5	179±6
Weight, kg			
Baseline	101.5±20.6	88.6±18.5	83.6±10.1*
24 wk	102.8±21.1	87.5±18.1	83.1±10.8
Body fat, %			
Baseline	25.4±6.0	22.6±4.2	23.4±3.9
24 wk	26.4±5.9	22.3±4.5†	21.9±3.9‡§
Waist girth, cm			
Baseline	104±16	96±15	94±9
24 wk	106±17	95±14	92±9†
Physical activity, kcal/day			
Baseline	3,602±734	3,247±728	2,953±480*
24 wk	3,518±720	3,355±829	3,089±498†
$\dot{V}O_{2\max}$, l/min			
Baseline	2.88±0.36	2.69±0.47	2.62±0.43
24 wk	2.83±0.38	3.07±0.48‡	3.18±0.52‡
$\dot{V}O_{2\max}$, ml·kg ⁻¹ ·min ⁻¹			
Baseline	29.40±6.47	30.97±5.71	31.76±5.99
24 wk	28.40±5.39	35.82±6.28‡	38.90±8.08‡

Values are means ± SD; *n*, no. of subjects. $\dot{V}O_{2\max}$, maximum oxygen consumption. *Significantly different from control group at baseline, $P < 0.05$. †Change from baseline is significantly different than control group, $P < 0.05$. ‡Change from baseline is significantly different than control group, $P < 0.001$. §Change from baseline is significantly different than moderate-intensity group, $P < 0.05$.

Lipid, lipoprotein, and fibrinogen concentrations are shown in Table 2. At baseline, exercise and control groups did not differ significantly in CHD risk factors. After 24 wk, changes in risk factors were not significantly different in the moderate-intensity group compared with the control group. In contrast, 24-wk changes in total cholesterol (-0.55 ± 0.81 mmol/l), LDL-C (-0.52 ± 0.80 mmol/l), and non-HDL-C (-0.54 ± 0.86 mmol/l) concentrations were significantly different in the high-intensity group compared with the control group ($P < 0.01$, $P < 0.05$, and $P < 0.05$, respectively). Trend analysis showed that total cholesterol, LDL-C, non-HDL-C, and fibrinogen concentrations changed favorably across control, moderate-intensity, and high-intensity groups (Fig. 3). Data from both exercise groups were pooled to identify correlations between changes in outcome variables (Table 3). Apart from the relationship between change in waist girth and change in triglycerides, changes in risk factors were not related to changes in fitness, activity, body fat, or waist girth. These relationships were not changed when exercise groups were analyzed individually.

DISCUSSION

Current guidelines suggest that changes in cardiorespiratory fitness are independent of exercise intensity (3). However, few studies have tested this hypothesis by comparing changes in cardiorespiratory fitness in previously sedentary individuals following moderate- and high-intensity exercise interventions of equal energy cost. While the present study is the first randomized controlled trial to accommodate improvements in cardiorespiratory fitness by prescribing exercise after monthly fitness tests, it is in agreement with controlled (18, 23) and uncontrolled (9) trials demonstrating that high-intensity training is more effective in increasing $\dot{V}O_{2\max}$ than moderate-intensity training of the same energy cost. Collectively, these findings suggest that high-intensity exercise should be performed if the goal of training is to maximize cardiorespiratory fitness. The present study also suggests that changes in CHD risk factors are influenced by exercise intensity.

In its position stand on exercise and cardiorespiratory fitness (3), the American College of Sports Medicine states that,

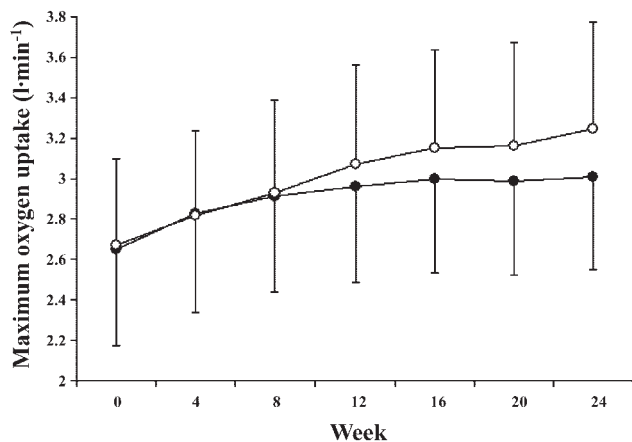


Fig. 2. Time course of the improvement in maximum oxygen consumption ($\dot{V}O_{2\max}$) among moderate-intensity exercisers (●, $n = 14$) and high-intensity exercisers (○, $n = 13$). Circles are means and error bars are SDs. Cardiorespiratory fitness did not change in the control group (-0.05 ± 0.16 l/min).

Table 2. Lipid, lipoprotein, and fibrinogen concentrations at baseline and after 24 wk of moderate- or high-intensity training

	Control Group	Moderate Intensity	High Intensity
<i>n</i>	15	14	13
Total cholesterol, mmol/l			
Baseline	5.41 ± 0.71	5.74 ± 0.82	6.02 ± 0.86
24 wk	5.71 ± 0.87	5.55 ± 0.94	5.48 ± 0.56*
LDL-C, mmol/l			
Baseline	3.44 ± 0.63	3.61 ± 0.89	4.04 ± 0.76
24 wk	3.61 ± 0.82	3.44 ± 0.82	3.52 ± 0.54†
HDL-C, mmol/l			
Baseline	1.20 ± 0.29	1.35 ± 0.30	1.44 ± 0.26
24 wk	1.28 ± 0.23	1.45 ± 0.42	1.43 ± 0.29
Non-HDL-C, mmol/l			
Baseline	4.20 ± 0.76	4.39 ± 0.85	4.58 ± 0.76
24 wk	4.43 ± 0.95	4.10 ± 0.90	4.04 ± 0.66†
Triglycerides, mmol/l			
Baseline	1.68 ± 0.77	1.72 ± 1.00	1.20 ± 0.38
24 wk	1.80 ± 0.84	1.45 ± 0.90	1.15 ± 0.48
Fibrinogen, g/l			
Baseline	2.98 ± 0.37	2.73 ± 0.50	2.88 ± 0.47
24 wk	3.17 ± 0.68 (11)	2.53 ± 0.40 (12)	2.58 ± 0.41 (12)

Values are means ± SD; *n*, no. of subjects. LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol. Numbers in parentheses denote different sample sizes. *Change from baseline is significantly different than control group, $P < 0.01$. †Change from baseline is significantly different than control group, $P < 0.05$.

“improvement will be similar for activities performed at a lower intensity-longer duration compared with higher intensity-shorter duration if the total energy cost of activities is similar.” However, the position stand cites only one study in which changes in fitness were compared following moderate- and high-intensity exercise interventions of similar energy cost (18). In that randomized controlled trial, Gossard et al. (18) compared changes in fitness in men who expended 350 kcal five times per week at 42–60 or 63–81% of $\dot{V}O_{2\max}$. After 12 wk, $\dot{V}O_{2\max}$ increased by 8% in the low-intensity group and by 17% in the high-intensity group (both $P < 0.001$ vs. controls). While the comparison of isocaloric interventions is rare, other authors have identified similar dose-response relationships. In an uncontrolled trial, Crouse et al. (9) reported that $\dot{V}O_{2\max}$ increased by 26% in men who expended 350 kcal 3 days/wk at 50% of $\dot{V}O_{2\max}$ and by 51% in men who completed the same 24-wk intervention at 80% of $\dot{V}O_{2\max}$. In a similar design as the present study, Kraus et al. (23) reported that $\dot{V}O_{2\max}$ increased by 0.16 ± 0.15 l/min in men and women who expended 1,200 kcal/wk at 40–55% of $\dot{V}O_{2\max}$ and by 0.41 ± 0.14 l/min in those who completed the same 6-mo intervention at 80% of $\dot{V}O_{2\max}$ ($P = 0.03$ and $P < 0.001$ vs. controls, respectively). These findings are in keeping with the present study and are not surprising, given that exercise intensity is the major determinant of the cardiovascular (30) and respiratory (8) responses to exercise.

The promulgation of moderate activity is also justified on the basis that vigorous activity is accompanied by increased risk of injury and dropout (3). While moderate interventions are often more successful (12), the present study is in agreement with those of others who found no difference in injury or dropout

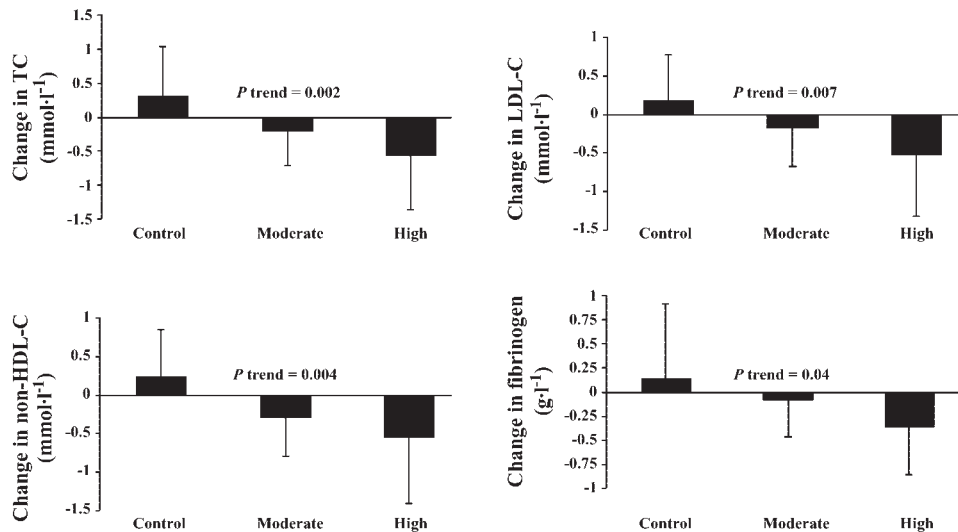


Fig. 3. Changes in total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), non-high-density lipoprotein cholesterol (HDL-C), and fibrinogen concentrations following 24 wk of moderate- or high-intensity exercise. Columns are means and bars are SDs.

rates between moderate- and high-intensity exercise groups (9, 18, 23). In the present study, the high-intensity group was able to expend 1,200 kcal/wk with three 30- to 40-min visits to the gym. To achieve the same energy expenditure while walking briskly, these men would have to walk for ~30 min/day, 7 days/wk (1).

Although it is often suggested that the genetic component of physical fitness undermines its prognostic power, genetic differences explain only ~25–47% of the individual variation in $\dot{V}O_{2\max}$ (5). Indeed, while there are high and low responders, vigorous exercise (75% of $\dot{V}O_{2\max}$) has been shown to increase cardiorespiratory fitness by ~20% in sedentary individuals, regardless of age, gender, race, and initial fitness level (39). For these reasons, it has been concluded that “the level of aerobic fitness that is needed for reducing the risk of CHD is not limited by the heredity of most people” (26). In the present study, the fitness level of the moderate-intensity group increased from “fair to average” to “average to good,” while the fitness level of the high-intensity group increased from “fair” to “good” (38). It is reasonable to suggest, therefore, that both groups enjoyed increases in fitness that are likely to confer long-term health benefits. Although health benefits were not obvious in the moderate-intensity group, recent evidence suggests that moderate- to high-intensity training reduces CHD

risk independent of conventional risk factors by improving endothelial function (36, 40).

Large prospective studies of Harvard alumni (28), British civil servants (31, 32), US health professionals (42), and Finnish twins (25) have shown that cardiovascular and all-cause mortality are lower among vigorously active men than among moderately active men. In the present study, trend analysis suggested that changes in CHD risk factors are also influenced by exercise intensity. However, no changes in lipid or lipoprotein concentrations were found in the Studies of a Targeted Risk Reduction Intervention through Defined Exercise (STRRIDE) among 111 overweight, dyslipidemic adults allocated to one of four groups for 6 mo (24): nonexercise control; low-volume/moderate-intensity exercise (1,200 kcal/wk at 40–55% of $\dot{V}O_{2\max}$); low-volume/high-intensity exercise (1,200 kcal/wk at 65–80% of $\dot{V}O_{2\max}$); or high-volume/high-intensity exercise (2,000 kcal/wk at 65–80% of $\dot{V}O_{2\max}$). Given the statistical power of STRRIDE (24), it is difficult to explain why the exercise interventions were unsuccessful. The authors suggested that lipid and lipoprotein concentrations were unchanged because participants were encouraged not to lose weight (23). However, exercise-induced changes in CHD risk factors are often observed in the absence of weight loss (44). Another explanation is the fact that STRRIDE did little to accommodate changes in fitness: $\dot{V}O_{2\max}$ was determined at baseline and after a 2- to 3-mo ramp period but not again during 6 mo of training (24). In the present study, training heart rates were prescribed after monthly fitness tests, and the high-intensity group enjoyed a reduction in total cholesterol sufficient to decrease CHD incidence by 54% (27). Concurrently, high-intensity training was accompanied by a significant reduction in non-HDL-C, a stronger predictor of cardiovascular disease mortality than total cholesterol or LDL-C (10). The modest changes in total cholesterol and LDL-C observed in the moderate-intensity group were similar to those reported in a meta-analysis of 31 randomized controlled trials with an average intensity of 62.9% of $\dot{V}O_{2\max}$ (19). We are not aware of any other study that has reported the effect of exercise training on non-HDL-C.

A number of risk factors were unchanged by exercise in the present study. The apparent resistance of HDL-C to training is

Table 3. Correlation matrix for changes in outcome variables

Variable	$\Delta\dot{V}O_{2\max}$, l/min	Δ Activity, kcal/day	Δ Body Fat, %	Δ Waist Girth, cm
$\Delta\dot{V}O_{2\max}$, l/min		0.25	-0.45*	-0.20
Δ Activity, kcal/day	0.25		-0.07	0.49*
Δ Body fat, %	-0.45*	-0.07		0.30
Δ Waist girth, cm	-0.20	0.49*	0.30	
Δ Total cholesterol, mmol/l	-0.15	-0.13	0.15	0.05
Δ LDL-C, mmol/l	-0.13	0.09	0.07	0.02
Δ HDL-C, mmol/l	-0.15	-0.33	0.34	-0.33
Δ Non-HDL-C, mmol/l	-0.11	-0.03	0.04	0.15
Δ Triglycerides, mmol/l	0.05	-0.34	-0.07	0.39*
Δ Fibrinogen, g/l	-0.04	0.25	0.19	0.00

Data were derived both exercise groups; $n = 27$. Δ , Change. *Two-tailed correlation is significant, $P < 0.05$.

unusual, given that an increase in HDL-C is the most common lipid change (29). This intransigence may be explained by the fact that, unlike LDL-C, change in HDL-C is related to baseline concentration (29). It is also possible that any change in HDL-C is an acute response to recent exercise (43). In the present study, men were instructed to observe a 24-h abstinence from vigorous activity before providing blood. However, as blood was drawn early in the morning, it is more likely that subjects avoided exercise for ~36 h. Similar circumstances may explain the apparent intransigence of triglyceride, because its decrease normally parallels HDL-C in onset and disappearance (43). It is also possible that changes in triglyceride and HDL-C concentrations are dependent on substantial reductions in abdominal adiposity (11). In the present study, exercise training was accompanied by modest reductions in abdominal adiposity, as indicated by changes in waist girth. In agreement with other randomized controlled trials (19), changes in body fat did not correlate with changes in lipids and lipoproteins. Although neither exercise group enjoyed a significant reduction in fibrinogen concentration compared with the control group, a dose-response relationship was evident in trend analysis. It is well documented that exercise training reduces fibrinogen concentration (22), and our null findings are likely explained by the small sample size and the large intragroup variability.

In conclusion, this study of previously sedentary men has shown that a high-intensity exercise intervention is more effective in improving cardiorespiratory fitness than a moderate-intensity intervention of equal energy cost. While dose-response relationships were evident in the present study, future research is required to determine the effect of exercise intensity on CHD risk factors.

GRANTS

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