Effects of high- and low-intensity exercise training on aerobic capacity and blood lipids

GLENN A. GAESSER and ROBERT G. RICH

Department of Kinesiology, University of California, Los Angeles, CA 90024

ABSTRACT

GAESSER, GLENN A. and ROBERT G. RICH. Effects of high- and low-intensity exercise training on aerobic capacity and blood lipids. Med. Sci. Sports Exerc., Vol. 16, No. 3, pp. 269-274, 1984. Sixteen non-obese, non-smoking males, ages 20-30 yr, were assigned to one of two training groups, exercising on a cycle ergometer 3 d/wk for 18 wk: high-intensity (H; N=7, 50-85% \( \dot{V} \text{O}_{2\text{max}} \), 35 min/session) or low-intensity (L; N=9, 45% \( \dot{V} \text{O}_{2\text{max}} \), 50 min/session). Data were obtained at 3 wk intervals for \( \dot{V} \text{O}_{2\text{max}} \), body weight, percent body fat, and 12-h fasting blood levels of cholesterol (CHOL), triglycerides (TG), high-density lipoprotein cholesterol (HDLC), and low-density lipoprotein cholesterol (LDLC). The average post-training increase in \( \dot{V} \text{O}_{2\text{max}} \) for group H (0.58 l/min\(^{-1}\), 8.5 ml/min\(^{-1}\), kg\(^{-1}\)) was not significantly (P>0.05) greater than for group L (0.45 l/min\(^{-1}\), 6.5 ml/min\(^{-1}\), kg\(^{-1}\)). Significant reductions in percent body fat occurred in both groups, amounting to an average loss of approximately 1.35 kg. No statistically significant changes in CHOL, TG, HDLC, LDL-C, CHOL/HDL-C, or LDL-C/HDL-C occurred in either group. However, changes in HDLC after 18 wk of training were inversely correlated (r=-0.57, P<0.05) with pre-training levels. We conclude that 1) the minimum exercise intensity threshold for improving aerobic capacity is at least 45% \( \dot{V} \text{O}_{2\text{max}} \), 2) 18 wk of high- or low-intensity exercise training is ineffective in significantly altering CHOL, TG, HDLC, LDL-C, CHOL/HDL-C, and HDLC/LDL-C in young male subjects with low blood lipid levels, and 3) exercise training-induced changes in HDLC are dependent upon initial pre-training levels.

EXERCISE, AEROBIC CAPACITY, CORONARY HEART DISEASE, LIPOPROTEINS, LIPIDS

Improvement in aerobic capacity is directly related to the intensity, duration, and frequency of training (2,26). An exercise intensity of 50-85% \( \dot{V} \text{O}_{2\text{max}} \), a duration of 15-60 min of continuous activity, and a frequency of 3-5 d/wk have been recommended for developing and maintaining fitness in adults (2). Following these guidelines, exercise training invariably results in significant improvements in \( \dot{V} \text{O}_{2\text{max}} \) in previously unconditioned subjects.

In contrast to well-established effects of aerobic exercise training on \( \dot{V} \text{O}_{2\text{max}} \), literature dealing with the influence of chronic exercise training on blood lipids and lipoproteins is controversial. For example, exercise training has been reported to result in increases (3,9,12,14, 25,28), decreases (1,24), or no change (4,19,21,27) in the blood concentration of high-density lipoprotein cholesterol (HDLC). Blood HDLC levels are of importance because an inverse relationship between HDLC and coronary heart disease has been reported (23). At present, the contributions of exercise intensity, duration, and frequency in modifying blood HDLC levels are not well understood in individuals with lipid levels within the normal range. However, in hyperlipidemic subjects with low HDLC, exercise training which results in decreases in triglyceride levels frequently is accompanied by increases in HDLC (8).

The purpose of the present investigation was to evaluate the effects of high-intensity (50-85% \( \dot{V} \text{O}_{2\text{max}} \)) and low-intensity (45% \( \dot{V} \text{O}_{2\text{max}} \)) exercise training on the time course of changes in aerobic capacity and blood lipid constituents. We hypothesized that the low-intensity exercise training might result in minimal, if any, gains in aerobic capacity, while the high-intensity exercise training would result in a significant increase (10-25%) in aerobic capacity (2,26). If this was so, we could establish if improvements in aerobic capacity are necessary for changes in blood lipids and lipoproteins to occur.

METHODS

Subjects. Seventeen healthy, non-smoking males between the ages of 20 and 30 yr volunteered to be subjects in this study. None had been involved in any form of regular physical activity for 6 months prior to the study. Each person was instructed as to the nature of the project, and written informed consent was obtained. Subjects were randomly assigned to either a high-intensity (H, initially N=8) or low-intensity (L, N=9) exercise training group. One subject in the H group had to withdraw from the exercise program after 6 wk for medical reasons unrelated to the project; therefore, N=7 for H group for all data analysis. The other 16 subjects successfully completed the 18-wk training program. The mean age of subjects was 23.5 ± 0.6 yr for the H group and 25.7 ± 1.4 yr for the L group. Pre-training characteristics of subjects in each group are given in Tables 1-3. There were no dietary restrictions during the course of the study except for the 12-h abstinence from food, alcohol, and exercise prior to the withdrawal of blood at 3-wk intervals during
EXERCISE TRAINING, \( V_{O_{2,max}} \), AND HDL-C

Statistical procedures. A repeated-measures analysis of covariance for each variable was used in this study in order to account for initial differences between groups, using the initial values as the covariate to compare exercise groups. With this analysis it was possible to assess the influence of the training program on each of the dependent variables and to determine whether the H and L groups responded differently to the training stimuli of high- and low-intensity exercise. Correlations were also performed to see whether changes in CHOL, TG, HDL-C, and LDL-C after 18 wk were related to pre-training levels.

RESULTS

Both H and L groups increased aerobic capacity during the 18-wk training program (Table 1). The H group increased \( V_{O_{2,max}} \) 0.56 l·min\(^{-1} \) (18.7%) and 8.5 ml·min\(^{-1} \)·kg\(^{-1} \) (19.6%); the L group increased \( V_{O_{2,max}} \) 0.45 l·min\(^{-1} \) (15.1%) and 6.5 ml·min\(^{-1} \)·kg\(^{-1} \) (17.2%). These increases were statistically significant (P<0.05). Although H improved \( V_{O_{2,max}} \) more than L in both relative and absolute terms, the differences between H and L, with respect to the changes in \( V_{O_{2,max}} \) during the 18-wk training program, were not statistically significant (P>0.05).

Data on body composition are presented in Table 2. Body weight did not change significantly in either group during the training program; however, significant (P<0.05) reductions in percent body fat were demonstrated for both groups. The loss of body fat for both groups was approximately 1.3–1.4 kg, while total body weight remained essentially unchanged as a result of an approximate 1–2 kg gain in lean body mass for both groups.

Blood lipid and lipoprotein data are presented in Table 3. No statistically significant changes were observed in CHOL, TG, HDL-C, or LDL-C for either group H or L. As a result of slight reductions in CHOL and LDL-C and increases in HDL-C, a decrease in CHOL/HDL-C and an increase in HDL-C/LDL-C were observed. However, the changes in both of these parameters were not statistically significant (P>0.05). The ranges of pre-training lipid values (mg%) were: CHOL, 135–246; TG, 33–142; HDL-C, 17–64; and LDL-C, 82–162. The correlations between individual pre-training values and the changes observed after 18 wk of training were: CHOL = -0.38, (P>0.05); TG = 0.85, (P>0.05); HDL-C = -0.57, (P<0.05); and LDL-C = -0.43 (P>0.05).

DISCUSSION

The increases in \( V_{O_{2,max}} \) achieved by both groups were comparable to those reported by others utilizing exercise intensities eliciting greater than 50% of \( V_{O_{2,max}} \) (1,2,21, 26). We were surprised at the improvement in \( V_{O_{2,max}} \) demonstrated in group L, which may partially be explained by the fact that during the 50-min training sessions heart rate typically rose by 6–10 bpm, so that by the end of each training period exercise heart rates for group L were 70–71% of their measured \( HR_{max} \). Training intensities of 70% of \( HR_{max} \) have been documented to result in improvements in cardiovascular capacity (4,26). It is not likely that maximal testing every 3 wk contributed to the training effect observed in group L.

| Table 1: Effects of 18 wk of either high-intensity (H) or low-intensity (L) exercise on \( V_{O_{2,max}} \) |
|---|---|---|---|---|---|---|---|
| Group | \( V_{O_{2,max}} \) | 0 | 3 | 5 | 9 | 12 | 15 |
| H | l·min\(^{-1} \) | 2.99 ± 0.21 | 3.26 ± 0.18 | 3.36 ± 0.16 | 3.37 ± 0.13 | 3.39 ± 0.11 | 3.55 ± 0.18 | 3.47 ± 0.11 |
| L | l·min\(^{-1} \) | 2.71 ± 0.11 | 2.84 ± 0.09 | 2.84 ± 0.08 | 3.00 ± 0.11 | 3.16 ± 0.09 | 2.99 ± 0.13 | 3.03 ± 0.10 |
| H | ml·min\(^{-1} \)·kg\(^{-1} \) | 43.3 ± 2.4 | 47.2 ± 2.4 | 49.0 ± 2.3 | 48.9 ± 2.2 | 48.3 ± 1.7 | 51.8 ± 2.2 | 50.1 ± 1.5 |
| L | ml·min\(^{-1} \)·kg\(^{-1} \) | 37.1 ± 1.6 | 39.8 ± 1.0 | 33.7 ± 1.3 | 41.5 ± 1.7 | 44.2 ± 1.5 | 41.8 ± 1.5 | 42.9 ± 1.4 |

Values are means ± SEM; N = 7 for H, N = 9 for L.

| Table 2: Total body weight, percent body fat, and lean body mass during 18 wk of either high-intensity (H) or low-intensity (L) exercise training |
|---|---|---|---|---|---|---|---|
| Parameter | Group | 0 | 3 | 5 | 9 | 12 | 15 |
| Body weight (kg) | H | 69.3 ± 4.0 | 69.8 ± 4.0 | 68.6 ± 4.1 | 70.0 ± 4.0 | 68.5 ± 3.6 | 68.1 ± 3.5 | 68.9 ± 3.5 |
| L | 72.5 ± 2.6 | 72.2 ± 2.7 | 72.3 ± 2.8 | 72.8 ± 2.9 | 72.2 ± 2.8 | 71.9 ± 2.8 | 72.2 ± 2.8 |
| Percent body fat | H | 18.0 ± 1.6 | 17.5 ± 1.5 | 17.2 ± 1.4 | 16.7 ± 1.4 | 16.6 ± 1.4 | 15.8 ± 1.2 | 15.9 ± 0.9 |
| L | 19.0 ± 1.7 | 17.9 ± 1.7 | 17.8 ± 1.5 | 18.0 ± 1.5 | 17.2 ± 1.5 | 17.2 ± 1.4 | 17.2 ± 1.4 |
| Lean body mass (kg) | H | 56.5 ± 3.1 | 57.4 ± 3.0 | 57.5 ± 3.2 | 58.1 ± 3.1 | 57.8 ± 3.0 | 58.0 ± 3.0 | 58.7 ± 2.9 |
| L | 58.4 ± 1.8 | 59.1 ± 1.7 | 59.2 ± 1.9 | 59.7 ± 1.8 | 59.5 ± 1.9 | 59.3 ± 1.8 | 58.6 ± 1.9 |

Values are means ± SEM; N = 7 for H, N = 9 for L.
the program. Subjects were encouraged to continue with their normal dietary habits.

**Training program.** Both groups of subjects pedaled at 50 rpm, 3 d/wk for 18 consecutive wk on a cycle ergometer (Monark, Model 668). Each subject in the high-intensity group exercised for 25 min per session at an intensity calculated to elicit 80–85% of his measured VO\(_{2\max}\), and each subject in the low-intensity group exercised for 50 min per session at an intensity calculated to elicit 45% of his measured VO\(_{2\max}\). The duration of the exercise sessions was chosen so that the total estimated energy expenditure of the two groups was approximately the same. The estimated caloric expenditure for the H and L groups was approximately 300 kcal/session at the beginning of the study; however, at the end of the study, the estimated caloric expenditure for both groups was approximately 350 kcal/session. All exercise sessions were supervised by laboratory personnel who monitored exercise conditions to ensure that the intensity remained constant during the entire 25- or 50-min exercise bout. In addition, heart rates were recorded (carotid palpation) every 5 min and 10 min for the H and L groups, respectively. After the first 5 min (group H) or 10 min (group L) of the exercise training sessions, heart rates ranged between 148–185 (85–90% HR\(_{max}\)) for group H and between 126–134 (65–67% HR\(_{max}\)) for group L. By the end of each training session, exercise heart rates had risen by 6–10 bpm, to about 95–96% HR\(_{max}\) for group H and 70–71% HR\(_{max}\) for group L.

Attendance at training sessions averaged 97%, with no subject missing more than two sessions (out of a possible 54). The subjects were randomly assigned to one of three starting dates, which were staggered 1 wk apart to distribute the collection of data evenly. This allowed measurements to be taken on approximately one-third of the subjects (representing both H and L groups) each week.

**Measurements.** The following parameters were determined in each subject prior to initiating the training program and at 3-wk intervals thereafter: VO\(_{2\max}\), body weight, percent body fat, cholesterol (CHOL), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). The 3-wk interval was selected based upon the data of Hickson et al. (13) which demonstrated that the adaptations to constant-intensity endurance exercise of the systems that limit VO\(_{2\max}\) were complete after 3 wk. On the basis of VO\(_{2\max}\) measurements, training conditions for the subsequent 3 wk were adjusted accordingly for each subject.

The cycle ergometer test for determination of the aerobic capacity was performed on the Friday ending each 3-wk period. Maximum aerobic capacity was determined utilizing an incremental cycle ergometer test. The VO\(_{2\max}\) test was performed by subjects on two occasions prior to initiating the training program in order to ensure a reliable baseline and familiarize the subjects to the cycle ergometer. Pedaling at a constant 50 rpm, the test began with 2 min at 49 W (300 kpm-min\(^{-1}\)). After the initial 2 min, the work rate was increased by 49 W every 2 min until a work rate of 196 W was reached. Thereafter, the work rate was increased by 24.5 W every 2 min until exhaustion. Exhaustion was defined as the point where the subjects could no longer move the pedals on the cycle ergometer.

Oxygen uptake was calculated according to open-circuit, indirect calorimetric methods (6). Subjects inspired room air through a Daniel's low-resistance valve, and expired air was passed through a flow integrator and digital pneumotachograph (Hewlett-Packard model 47303 A) for volume determination. Expired air was sampled continuously at a flow rate of 500 ml-min\(^{-1}\) from a mixing chamber, in series with the pneumotachograph, passing through anhydrous CaSO\(_4\) (for drying) prior to analysis of O\(_2\) and CO\(_2\) content (Beckman OM-11 and LB-2 medical gas analyzers, respectively). The VO\(_{2}\) for each intensity was calculated by using data from the last 30 s at each condition. All instruments were calibrated prior to and checked again after each experimental session. The concentration of gases in the calibration tank used to calibrate the O\(_2\) and CO\(_2\) analyzers were verified by Schoonder analysis. The highest VO\(_{2}\) recorded during the exercise test was determined to be VO\(_{2\max}\). The "leveling off" criterion was met in all VO\(_{2\max}\) tests. In fact, in many cases the VO\(_{2}\) value at the final test condition was slightly lower than that of the preceding condition due to the pedaling rate approaching zero.

Throughout the exercise tests, heart rate was monitored continuously via a three-lead ECG using a Grass polygraph (model 7D).

Body density was determined by hydrostatic weighing, using a Chatillon autopsy scale. Residual volume was assessed by the modified oxygen dilution technique of Wilmore et al. (29), and percent body fat was calculated according to Brozek et al. (5). The hydrostatic weighing was performed on the day preceding the exercise test.

Blood was sampled in the morning of the day of the exercise test, following a 12-h overnight abstention from food (water permitted) and alcohol, 44–48 h after their most recent session. Approximately 10 ml of blood was drawn from an antecubital vein in a vacuum-sealed tube containing ethylenediaminetetraacetic acid (EDTA). The blood was centrifuged and the plasma was analyzed for CHOL, TG, HDL-C, and LDL-C the same day. Plasma CHOL and TG were determined on the AutoAnalyzer II (Technicon Instruments Corp., Tarrytown, NY) using an isopropanol extract treated with a zeolite mixture, following the procedures described by Kessler (15) and the Lipid Research Clinic (LRC) program (20). Determination of HDL-C was achieved by the heparin-manganese precipitation procedure as described by the LRC program. Week-to-week variation in "standards" for CHOL, TG, and HDL-C was <1.5%. Low-density lipoprotein cholesterol was estimated by using the indirect method of Friedewald et al. (10).
and Quinney (27) reported no significant changes in \( V_{\text{O}_{2\max}} \) in subjects who were maximally tested every 3 wk for 18 wk. Our results demonstrate that the minimum intensity threshold for improving aerobic capacity is at least 45% \( V_{\text{O}_{2\max}} \); provided the duration and frequency of exercise training are 50 min/session and 3 d/wk, respectively.

Reductions in body fat content and increases in lean body mass consequent to training have also been reported (2,19,26). In the present study it is probable that a major portion of the increase in lean body mass resulted from an expansion of total blood volume (7). Body composition changes during training are related to the total caloric expenditure (26). Our data are consistent with this finding in that both groups reduced body fat significantly but did not differ with respect to the absolute amount of fat lost.

A major purpose of this study was to see if blood lipids and lipoproteins could be altered while exercising at an intensity below that which has been previously reported to be necessary to improve aerobic capacity. We were unable to find evidence of an intensity threshold for inducing changes in HDL-C, or any other blood lipid parameter, because none of the blood lipid constituents we measured changed significantly in either group H or L during the 18 wk of training.

The lack of change in CHOL and TG was expected. The mean pre-training values for CHOL (182–183 mg%) and TG (58–83 mg%) were low (see Table 3). It is known that exercise training is unlikely to induce changes in CHOL and TG when pre-training values are low at the beginning of the study (9,18).

The effect of exercise training on plasma HDL-C concentrations is controversial (8). Although some authors of longitudinal studies (3,9,12,14,25,26) have reported increases in HDL-C after exercise training, others have found decreases (12,24) or no change (4,19,21,27). These inconsistent results are in contrast to the data of a rather substantial number of cross-sectional studies, as in the study of Dufaux et al. (8), which indicate that HDL-C levels are significantly higher in habitually active people. If physical activity has a dependable influence on increasing HDL-C levels, then longitudinal studies of exercise training should confirm the consistent results of cross-sectional studies. Factors which may influence the results (and therefore enhance the possibility of discrepancies) of longitudinal studies are numerous. Among the most important would be exercise intensity, duration, frequency, mode, length of training program, initial fitness level, pre-training lipid and lipoprotein levels, weight, body fat content, age and gender of subjects, diet, smoking habits, and socioeconomic factors (8). It is with these multiple factors in mind that we must limit our conclusions to the population our subjects represent and the specific conditions of our experiment.

The composition of the diet is known to influence lipoprotein levels (8). Although we did not analyze the diets of our subjects, each subject informed us that he/she made no conscious attempt to change his dietary habits during the course of the study. It has been reported previously that exercise training does not result in changes in nutrient intake or ratios of dietary fats, carbohydrates, and protein (4,16,17). We believe it is unlikely that dietary factors prevented significant changes in lipoprotein levels in our subjects throughout the 18 wk of training.

There is evidence to suggest that significant decreases in TG must accompany exercise training for increases in HDL-C to occur (1,3,26). Allison et al. (1) and Sutherland and Woodhouse (28) reported that exercise training-induced increases in HDL-C occurred only in subjects who showed significant reductions in TG. As a consequence, selection of a sample of subjects with very low initial TG levels, such as the case in the present study, may preclude an exercise training-induced increase in
HDL-C. However, increases in HDL-C after training have been reported in the absence of significant decreases in TG (18). Additionally, in the present study individual changes in TG after 18 wk of training were not significantly correlated with changes in HDL-C ($r = -0.17$, $P>0.05$). Further research, utilizing a larger sample with a wider range of initial TG values, is necessary to clarify this issue.

Increases in HDL-C have been reported in the absence of decreases in CHOL (9,12,18) or body weight (9,12, 14,22,28). Therefore, the inability to show a training-induced increase in mean HDL-C levels in our subjects is not likely due to a lack of change in CHOL or body weight. Despite the lack of changes in mean HDL-C levels for both groups during the 18-wk training program, individual changes in HDL-C after 18 wk were significantly correlated to initial pre-training values, which suggests that with exercise training it is easier to demonstrate increases in HDL-C in subjects with low initial values. This is consistent with the results of Sutherland and Woodhouse (28), who reported that changes in HDL-C were dependent upon pre-training values. In their study, the subjects with the lowest initial values increased HDL-C values the most after training.

A lack of change in total HDL-C does not necessarily mean that no alterations occurred in this lipoprotein fraction. Increases in the HDL-2 subfraction with either no change or a decrease in the HDL-3 subfraction, have been reported (3,25,30). Several investigators have shown that it is HDL-2 that is the critical subfraction that changes with physical activity (3,25,30). Thus, in the present study there is the possibility that the lack of a significant increase in total HDL-C observed after 18 wk did not reflect important changes in the subclasses of HDL that may have occurred as a result of training.

Significant changes in CHOL/HDL-C and HDL-C/LDL-C may occur despite nonsignificant changes in the concentrations of lipids and lipoproteins (19). The ratios of CHOL to HDL-C and of HDL-C to LDL-C help to estimate the risk for coronary heart disease (8,11). In our subjects the pattern of changes in CHOL/HDL-C and HDL-C/LDL-C during the 18 wk of training could be considered favorable in reducing the risk of CHD. However, the changes were not statistically significant. Our data suggest that exercise training for 18 wk will not alter the percentages of circulating CHOL in the form of HDL-C and LDL-C in young male subjects with low blood lipid levels.

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REFERENCES


MEDICINE AND SCIENCE IN SPORTS AND EXERCISE


