To compare the effects of strength training (ST) to those of aerobic training (AT) for coronary heart disease (CHD) risk factor intervention, we studied 37 previously untrained males (aged 50 ± 9 years, mean ± SD) before and after 20 weeks of either ST (N = 14), AT (walk/jog, N = 13), or no exercise (inactive controls, N = 10). Lipoprotein and lipid profiles, blood pressure, and glucose and insulin responses to an oral glucose tolerance test (OGTT) were assessed before and after the training period in all three groups. The ST program produced significant reductions in plasma glucose levels at 60, 90, and 120 minutes (P < .05) after glucose ingestion, whereas the AT program resulted in significant reductions only at 90 and 120 minutes (P < .05). ST also decreased insulin levels during fasting (P < .05) and at 90 and 120 minutes (P < .01) after glucose ingestion. AT decreased insulin levels at 90 and 120 minutes (P < .01) after glucose ingestion. Both training programs reduced the total area under the glucose tolerance curve for glucose (both P < .05) and insulin (both P < .05), but there were no significant differences in these changes between the two groups. None of the glucose or insulin values were significantly altered in the control group. There were no significant changes in lipoprotein and lipid profiles or blood pressure in any of the three groups. These results suggest that ST and AT have comparable effects on risk factors for CHD. Both ST and AT improve glucose tolerance and reduce insulin responses to oral glucose in middle-aged men with multiple risk factors for CHD.

Measurement of Aerobic Capacity and Body Composition

Aerobic capacity (VO2max) was measured during a continuous treadmill test as described previously.5 Oxygen uptake values were recorded every 30 seconds using the Medical Graphics Cardiopulmonary Exercise System 2001 (St Paul, MN) to analyze ventilation and gas fractions. VO2max was established when two of the following three criteria were met: (1) maximal heart rate was within 10 beats/min of age-predicted values; (2) respiratory exchange ratio values were greater than 1.10; and (3) a plateau in oxygen uptake was achieved, as evidenced by no more than a
150-mL · min⁻¹ or 2-mL · kg⁻¹ · min⁻¹ increase in oxygen uptake with an increase in workload. Subjects who did not meet these criteria the first time were retested; all subjects eventually met the criteria and were included in the data analysis.

Body composition was estimated from body density determined by hydrodensitometry. Underwater weight was adjusted for residual lung volume using the nitrogen washout technique on the Medical Graphics Pulmonary Function Test System 10/70. Percent body fat was calculated from body density values using the formula of Brozek et al.

Measurement of Blood Pressure

Resting blood pressure was measured in the seated position before each training session in both training programs. The mean of six blood pressure measurements obtained during the first 2 weeks of training was compared with six measurements taken during the last 2 weeks of training for both groups.

Dietary Records

All subjects were instructed by a registered dietitian regarding appropriate techniques for recording food records to ensure accuracy. They were also reminded on a regular basis not to change their dietary habits and alcohol intake throughout the program. Subjects were asked to abstain from alcohol consumption for at least 3 days before blood sampling. Food records were kept for 4 days before the initial blood sampling and OGTT, and the same diet was replicated for the 4-day period before the final blood sampling at the end of training. All food records were coded for nutritive value according to US Department of Agriculture guidelines and were analyzed for food composition and caloric content.

Plasma Lipoprotein and Lipid Determinations

Blood samples for lipid and lipoprotein measurements were obtained from each subject's antecubital vein after a 12- to 14-hour overnight fast. Samples were placed into chilled tubes containing EDTA on two separate mornings within 1 week apart. If values for either total cholesterol or HDL-C differed by more than 7%, a third blood sample was taken on another day; the baseline value reported is the mean of the two determinations that did not differ by more than 7%. After training, blood samples were drawn once in control subjects and 1 day (20 ± 5 hours) and 2 days (46 ± 7 hours) after the last exercise session in the training groups.

Plasma total cholesterol and triglyceride concentrations were measured directly. HDL-C level was measured after precipitation of very-low-density lipoprotein (VLDL) and LDL by dextran sulfate. Since all subjects had triglyceride levels below 400 mg · dl⁻¹, LDL-C level was calculated using the formula LDL-C = total cholesterol − (HDL-C + triglyceride/5). All analyses were performed in duplicate as previously described.

Plasma Glucose and Insulin Determinations

Glucose and insulin concentrations were measured in the fasting blood sample and in blood samples drawn 30, 60, 90, and 120 minutes after ingestion of 40 g glucose/m² body surface area. Plasma glucose levels were measured by the glucose oxidase method with a glucose analyzer (Beckman Instruments, Fullerton, CA), and insulin levels were determined by radioimmunoassay. After training, OGTTs were performed again in the morning after a 12- to 14-hour fast, approximately 20 hours after the last training session.

Areas under the OGTT curves for glucose and insulin were computed by the trapezoidal method using both fasting concentrations (incremental area) and zero (total areas) as the baseline.

Training Programs

ST. Subjects trained three times a week on nonconsecutive days for approximately 20 weeks using Nautilus equipment (DeLand, FL). Before training, all subjects were shown the proper technique for each exercise by a qualified instructor. The training program consisted of two sets using the maximum amount of weight that could be lifted 12 to 15 times (repetitions maximum [RM]) per set, with 11 different exercises and modified sit-ups. Weights were adjusted throughout the training program as strength levels increased. All subjects engaged in a brief warm-up period consisting of static stretching and calisthenics before each training session. The following exercises were performed during each workout: duo squat, leg extension, leg curl, hip and back, decline press, pullover, arm cross, behind-the-neck pullover, overhead press, lateral raise, rowing torso, and sit-ups. The resting interval between sets was limited to 90 seconds. Every training session was carefully supervised and subjects were encouraged to exercise until they failed to complete the last repetition attempted. The exercises performed, amount of weight lifted (number of plates), and number of sets and repetitions completed for each exercise were recorded for every exercise session.

Strength was measured as the maximum load that could be successfully lifted for one repetition (1-RM). The 1-RM test for baseline upper- and lower-body strength was administered after three to five workout sessions to allow subjects to become accustomed to the exercise. The upper-body exercises included the pullover, decline press, and overhead press; the lower-body exercises included the duo squat, leg extension, and leg curl. The 1-RM was achieved by increasing the load by one plate after each successful lift until the maximum load was obtained for each exercise. Subjects were given a 2-minute rest interval after every three trials until the 1-RM was reached. These procedures were followed at both testing periods for both the ST and control groups.

AT. Following flexibility and warm-up exercises, subjects walked and/or jogged on a motorized treadmill for 30 min/d 3 days each week for approximately 20 weeks. During the first week, treadmill speed was adjusted to maintain an exercise intensity of 50% to 60% of each subject's maximal heart rate reserve. This relative intensity was increased to 60% to 70% during the second week and 75% to 85% thereafter. Heart rates were periodically monitored throughout the training program. Gradual increases in speed and/or elevation were administered throughout the training program to accommodate condition of the control group. Control subjects completed the identical procedures for testing at approximately the same two time periods as both of the training groups, but did not participate in any regular exercise for the entire 20-week period.

Statistical Analysis

Age, height, pretraining total body mass, percent body fat, fat-free mass, VO₂-max, and all blood variables were analyzed by one-way ANOVA to determine whether differences existed between the groups before training.

The chi-square test was used initially to determine whether there was an equal distribution of the three major risk factors among the groups. Within-group differences were analyzed collectively by MANOVA. When MANOVA revealed significant differences, univariate ANOVA was performed with planned comparisons to determine the effects of each training modality. Significant changes from before to after training were compared with changes from initial to final in the control group using the interactions derived from ANOVA. The mean ± within-subject SEM are presented on all figures to simplify interpretation of significant differences. All
data presented in tables are expressed as the mean ± SD and the
5% level of significance is used.

RESULTS

Group Characteristics

There were no significant differences among the groups for age, height, total body mass, percent body fat, fat-free mass, VO\textsubscript{2}max, or any of the blood variables before training. Chi-square analysis demonstrated that the prevalence of hyperlipidemia, impaired glucose tolerance, and hypertension was the same among the groups.

Percent body fat decreased with AT (\(P < .05\), but not with ST; VO\textsubscript{2}max increased by 19% with AT (\(P < .0001\)), but did not change with ST. There were no significant changes in total body mass or fat-free mass in either the AT or ST groups. Body composition, total body mass, and VO\textsubscript{2}max also did not change in the control subjects (Table 1).

The ST program resulted in a 50% increase in upper-body strength (8 ± 2 v 12 ± 3 weight plates) and a 36% increase in lower-body strength when values for the 1-RM test were averaged for upper- and lower-body exercises, respectively. There were no significant changes in the 1-RM values for the control group.

Dietary Composition

Analysis of food records indicated that all subjects abstained from alcohol and ingested at least 250 g carbohydrates/d for 3 days immediately preceding each OGTT. There were no significant differences in either group between pretraining and posttraining food records with regard to total kilocalories consumed or percent of calories derived from carbohydrates, proteins, or fats. In addition, there were no differences in the amount of cholesterol, fiber, or fats derived from monounsaturated, polyunsaturated, or saturated fatty acids between the two recording periods (Table 2).

Lipoprotein and Lipid Profiles

The concentrations of triglycerides, total cholesterol, HDL-C, and LDL-C did not change with either training program (Table 3). These results were no different whether values were measured 1 day (20 ± 5 hours) or 2 days (46 ± 7 hours) after the last training session. Only three subjects from each training group showed an improvement in their lipid profiles as evidenced by a decrease in the ratio of total cholesterol to HDL-C by more than 0.5 mg \textsuperscript{-1} dL

There was no consistent relationship between changes in VO\textsubscript{2}max, weight, percent fat, or fat-free mass and changes in lipid profiles in either training group. In addition, there was no consistent relationship between changes in lipoprotein and lipids and family history of heart disease.

Blood Pressure

There were no significant changes in resting blood pressure in either of the training programs (Table 4); however, a definite trend toward a reduced mean arterial blood pressure response was observed among subjects with mild or moderate hypertension as a result of each training program. For example, there was an average decrease of 5 mm Hg in diastolic blood pressure (93 ± 8 v 88 ± 5) in the AT group (\(n = 4\)) and a 6-mm Hg decrease in systolic blood pressure (152 ± 17 v 146 ± 14) in the ST group (\(n = 4\)) among those subjects with hypertension. Nevertheless, there were too few subjects in this category to make any meaningful conclusions about the effects of training on blood pressure.

Glucose and Insulin Responses

The AT program resulted in a lower plasma glucose concentration at 90 (\(P < .05\)) and 120 minutes (\(P < .01\)) after glucose ingestion (Fig 1A), whereas the ST program resulted in decreased glucose concentrations at 60, 90, and 120 minutes (\(P < .06\); Fig 1B). The 90 and 120 minute reductions in AT and ST groups were both significantly different from changes in the control group. Although there was more than a 20% reduction from both training programs in total glucose incremental area (above basal) under the curve, these changes did not reach statistical significance in either the AT (\(P = .06\)) or ST (\(P = .09\)) groups (Fig 2A). However, there was a significant reduction in total glucose area under the curve in both groups (\(P < .05\)) when zero was used as the baseline (Fig 2B). These reductions were also significantly different from changes in the control group. There were no significant differences between the

<table>
<thead>
<tr>
<th>Table 1. Characteristics of Training and Control Groups</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AT ((N = 13))</strong></td>
</tr>
<tr>
<td><strong>Before Training</strong></td>
</tr>
<tr>
<td><strong>Age (yr)</strong></td>
</tr>
<tr>
<td><strong>Height (cm)</strong></td>
</tr>
<tr>
<td><strong>Body mass (kg)</strong></td>
</tr>
<tr>
<td><strong>% Body fat</strong></td>
</tr>
<tr>
<td><strong>Fat-free mass (kg)</strong></td>
</tr>
<tr>
<td><strong>VO\textsubscript{2}max (ml \cdot kg\textsuperscript{-1} \cdot min\textsuperscript{-1})</strong></td>
</tr>
</tbody>
</table>

* \(P < .05\), significantly different from pretraining values.
* \(P < .001\), significantly different from pretraining values.
* \(P < .0001\), change from before training to after training significantly different from initial change to final change in control group.
* \(N = 12\) for % body fat, fat-free mass, and VO\textsubscript{2}max in AT; \(N = 13\) for VO\textsubscript{2}max in ST.
were no differences between the two training groups for or insulin values at any specific time points or total areas under the curve in the control group. There were no differences between initial and final glucose levels were not influenced by training-induced hemoconcentration or hemodilution. There was a significant reduction in the incremental insulin responses at any specific time points or for total insulin areas under the curve. In addition, the total insulin area under the curve resulted from the AT program, and a 21% reduction in the total insulin area under the curve for the AT group, but not for the ST group. These changes differed significantly from those of the control group. Peak insulin levels were attained at 90 minutes before training and 60 minutes after training in the AT group (Fig 3A), and 120 minutes before training and 60 minutes after training in the ST group (Fig 3B). There was a significant reduction in the incremental insulin area under the curve for the AT group, but not for the ST group (Fig 4A). These changes were not significantly different in either training group from those of controls. When using zero as baseline, a 24% reduction \((P < .01)\) in the total insulin area under the curve resulted from the AT program, and a 21% reduction \((P < .05)\) resulted from the ST program (Fig 4B). Only the results from the AT program were significantly different from controls. There were no differences between the two training groups for insulin responses at any specific time points or for total insulin areas under the curve (Fig 4A and B). In addition, there were no differences between initial and final glucose or insulin values at any specific time points or total areas under the curve in the control group.

Hematocrit levels were unchanged following both training modalities, suggesting that changes in glucose and insulin levels were not influenced by training-induced hemoconcentration or hemodilution.

Table 2. Dietary Analyses of Training and Control Groups

<table>
<thead>
<tr>
<th></th>
<th>AT (N = 13)</th>
<th>ST (N = 14)</th>
<th>Control Group (N = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before Training</td>
<td>After Training</td>
<td>Before Training</td>
</tr>
<tr>
<td>Total daily intake (kcal)</td>
<td>2,368 ± 436</td>
<td>2,727 ± 832</td>
<td>2,565 ± 639</td>
</tr>
<tr>
<td>Carbohydrate (%)</td>
<td>52 ± 12</td>
<td>53 ± 10</td>
<td>51 ± 12</td>
</tr>
<tr>
<td>Protein (%)</td>
<td>18 ± 4</td>
<td>17 ± 4</td>
<td>19 ± 3</td>
</tr>
<tr>
<td>Fat (%)</td>
<td>31 ± 10</td>
<td>30 ± 10</td>
<td>31 ± 11</td>
</tr>
<tr>
<td>Monounsaturated fats (g)</td>
<td>33 ± 17</td>
<td>31 ± 21</td>
<td>33 ± 12</td>
</tr>
<tr>
<td>Polyunsaturated fats (g)</td>
<td>11 ± 6</td>
<td>11 ± 7</td>
<td>13 ± 5</td>
</tr>
<tr>
<td>Saturated fats (g)</td>
<td>28 ± 15</td>
<td>27 ± 16</td>
<td>35 ± 11</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>323 ± 182</td>
<td>277 ± 133</td>
<td>427 ± 159</td>
</tr>
<tr>
<td>Fiber (g)</td>
<td>6 ± 4</td>
<td>5 ± 6</td>
<td>4 ± 2</td>
</tr>
</tbody>
</table>

NOTE. Values are means ± SD. There were no significant differences in dietary components between before training and after training within each of the training groups or between initial and final within the control group.

Table 3. Plasma Lipid and Lipoprotein Levels in Training and Control Groups

<table>
<thead>
<tr>
<th></th>
<th>AT (N = 13)</th>
<th>ST (N = 14)</th>
<th>Control Group (N = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before Training</td>
<td>After Training</td>
<td>Before Training</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>167 ± 103</td>
<td>172 ± 153</td>
<td>160 ± 110</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>212 ± 40</td>
<td>210 ± 38</td>
<td>200 ± 26</td>
</tr>
<tr>
<td>LDL-C</td>
<td>135 ± 29</td>
<td>138 ± 29</td>
<td>130 ± 22</td>
</tr>
<tr>
<td>HDL-C</td>
<td>41 ± 9</td>
<td>43 ± 7</td>
<td>37 ± 7</td>
</tr>
</tbody>
</table>

NOTE. Values are means ± SD in mg/dL. There were no significant differences in lipid levels between before training and after training within each of the training groups or between initial and final within the control groups.

* \(N = 12\) for LDL-C and HDL-C in AT; \(N = 9\) for triglycerides in control groups. There were no significant differences between groups.

DISCUSSION

The results of this study demonstrate that ST has effects on risk factors for CHD similar to those of AT in middle-aged men. Both ST and AT reduced glucose and insulin responses to glucose ingestion during an OGTT, suggesting that both programs are equally effective for improving glucose tolerance and insulin response. Such changes in regulation of glucose metabolism should protect against the development of non-insulin-dependent diabetes mellitus (NIDDM) and possibly atherosclerosis.

Previous investigations show that AT decreases both plasma glucose and insulin concentrations in response to OGTT. ST also reduces the insulin response to glucose ingestion, but glucose tolerance did not change in these studies; however, only normoglycemic subjects were studied. In the present study, all subjects either closely approached or met criteria for impaired glucose tolerance or NIDDM. In this regard, although none of the NIDDM patients changed their disease status, in three of four subjects in the ST group with impaired glucose tolerance, glucose tolerance normalized following training. To our knowledge, this is the first report that demonstrates an improved glucose tolerance from ST with responses similar to those resulting from AT.

There appears to be an empirical basis for the finding that ST is just as effective as AT in improving the regulation of glucose metabolism. Studies in isolated muscle indicate that muscle contraction produces an insulin-like effect on glucose uptake, regardless of whether the contractions are performed isometrically or isotonically. Although no infor-
Table 4. Blood Pressure in Training and Control Groups

<table>
<thead>
<tr>
<th></th>
<th>AT (N = 13)</th>
<th>ST (N = 14)</th>
<th>Control Group (N = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>After</td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Training</td>
<td>Training</td>
<td>Training</td>
<td>Initial Final</td>
</tr>
<tr>
<td>Systolic</td>
<td>133 ± 8</td>
<td>132 ± 9</td>
<td>137 ± 17 135 ± 15</td>
</tr>
<tr>
<td>Diastolic</td>
<td>86 ± 6</td>
<td>84 ± 7</td>
<td>85 ± 6 83 ± 7</td>
</tr>
<tr>
<td>Mean arterial*</td>
<td>102 ± 5 100 ± 3</td>
<td>102 ± 9 100 ± 10</td>
<td>99 ± 7 99 ± 9</td>
</tr>
</tbody>
</table>

NOTE: Values are means ± SD in mm Hg.

*Mean arterial blood pressure = diastolic + (systolic - diastolic)/3; none of the values are significantly different.

information was obtained in the present study to explain the mechanism by which either AT or ST reduced glucose or insulin responses after glucose ingestion, other investigators suggest that AT programs might reduce insulin secretion by increasing the number of glucose transporters, specifically the GLUT 4 transporter.

It has been suggested that improvements in glucose regulation from exercise training are secondary to reductions in body fat. The findings from the ST program refute this conclusion, since percent body fat did not change. An increase in muscle mass is postulated as one mechanism by which ST might improve glucose regulation; however, there were no changes in fat-free mass determined by hydrodensitometry in the present study. Nevertheless, this does not necessarily mean that muscle mass did not change, because we have recently observed increases in regional muscle mass with ST despite changes in total fat-free mass measured by hydrodensitometry.

The possibility that improvements in glucose tolerance and reductions in insulin responses in the present study were due to the short-term effects of the last exercise session cannot be excluded. The residual effects of the last exercise session may play a role in the increase in insulin sensitivity observed following training. Short-term exercise can increase both glucose disposal and insulin responses. However, the elapsed time between the last exercise session and the OGTT was the same for all subjects in the present study.

The finding that neither training modality affected lipoprotein and lipid profiles was unexpected. Although several investigators report no improvements in lipid levels with
either AT\textsuperscript{33,34} or ST programs,\textsuperscript{35} others studies do show significant changes in lipid profiles with both training modalities.\textsuperscript{36} An insufficient training intensity or duration,\textsuperscript{37} the initial VO\textsubscript{2}max being too high,\textsuperscript{38} HDL-C level being too low,\textsuperscript{39} no weight loss,\textsuperscript{40} or the absence of a decrease in body fat are some of the most common reasons for the lack of change in lipid profiles. After careful examination for the effects of each of these factors, it does not appear that any of them could explain the discrepancy between the findings in the present study and those of studies that found improved lipid profiles after either AT or ST. Although the ST group did have relatively low initial HDL-C levels and neither group changed their weight with training, other studies have reported improvements in lipid profiles with similar initial HDL-C levels\textsuperscript{41,42} and without weight loss.\textsuperscript{34,43,44} Furthermore, there was a significant decrease in body fat in the AT group, a factor that appears to be important to the improvement in lipid profiles with AT.\textsuperscript{43} It is conceivable that the initial level of adiposity was a factor inhibiting a significant improvement in lipid profiles. In this regard, Coon et al\textsuperscript{45} also reported no change in lipoprotein and lipid profiles in older obese men after AT. Most of the subjects in our study were obese; however, after examination of the results in responders (n = 3 for each group) versus nonresponders, we were unable to identify any factors that could explain the lack of improvement in lipoprotein and lipid profiles with either AT or ST. Thus, we do not have an explanation for the conflicting results between the present study and those that have found favorable alterations in lipid profiles with either AT or ST. However, in some of the previous studies that reported improvements in lipid profiles with ST, there was a lack of control for normal variations of lipid and lipoprotein levels by not having a control group, drawing only one blood sample, and using subjects who had no risk factors for CHD, and there were also alterations in body weight or composition.\textsuperscript{44} The results of the present study show that when these limitations are considered, neither ST nor AT improves lipoprotein and lipid profiles in middle-aged men. The finding of no significant changes in resting blood
pressure with either training modality also was unexpected. Several investigators have found significant reductions in blood pressure with either AT or ST. In the present study, there was a trend toward a reduction in diastolic blood pressure with AT and a decrease in systolic blood pressure with ST in the hypertensive men. It is conceivable that the lack of effect from either training modality in normotensive subjects prevented a statistically significant decrease that may have occurred if all subjects were hypertensive. This possibility is currently being investigated in hypertensive subjects.

In summary, 20 weeks of ST had the same effects as 20 weeks of AT (walking/jogging) on risk factors for CHD. Although both training modalities reduced glucose and insulin responses to an OGTT, neither program altered lipoprotein and lipid profiles or resting blood pressure.

ACKNOWLEDGMENT

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