Heredity and Changes in Plasma Lipids and Lipoproteins after Short-term Exercise Training in Men

Jean-Pierre Després, Sitál Moorjani, Angelo Tremblay, Eric T. Poehlman, Paul J. Lupien, André Nadeau, and Claude Bouchard

The aims of this controlled experiment were to investigate the effects of short-term aerobic exercise training on plasma lipid and lipoprotein concentrations and the role of heredity in determining the individual variation observed in the lipoprotein-lipid response. Six pairs of male monozygotic (MZ) twins were subjected to an exercise training program that induced a 22,000 kcal energy deficit after 22 consecutive days of training. This program significantly reduced body weight, percent body fat, and subcutaneous fat and significantly increased maximal oxygen consumption (VO₂max) (p<0.005). The plasma insulin response to an oral glucose challenge was markedly reduced after training (p<0.001). Plasma triglyceride concentration decreased and the high density lipoprotein cholesterol (HDL-CHOL)/CHOL ratio increased with training (p<0.05). Subjects also displayed substantial individual variation in their response to exercise training, but the changes in plasma CHOL, apolipoprotein (apo) B low density lipoprotein cholesterol (LDL-CHOL), HDL-CHOL, and the HDL-CHOL/CHOL ratio tended to be similar within MZ twin pairs (0.67±0.92; 0.05>p<0.0001) thus indicating a significant effect of heredity on the sensitivity of plasma lipids and lipoproteins to exercise training. In addition, individual changes in plasma lipids and lipoproteins were correlated with neither changes in percent body fat nor changes in VO₂max, whereas changes in plasma CHOL, apo B, and LDL-CHOL were significantly correlated with changes in the proportion of trunk fat and with the Insulinogenic Index (0.63±0.74; 0.05>p<0.01). Control for initial level of body fat distribution or its alteration during exercise training eliminated the within-pair resemblance in CHOL, apo B, and LDL-CHOL responses. These findings indicate that some of the individual variation in the response of plasma lipoproteins to exercise training is influenced by heredity and is partly mediated by alterations in subcutaneous adipose tissue distribution and associated changes in insulin metabolism.

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with heredity has, to our knowledge, not been studied. To investigate this question, six pairs of MZ twins were subjected to a controlled experiment during which an aerobic exercise training program of 22 days was used to generate a negative energy balance of 22,000 kcal. The plasma lipoprotein-lipid profile, along with other potentially associated metabolic indices, was measured.

**Methods**

**Subjects**

Six pairs of young adult male MZ twins gave their written consent to participate in this study, which received the approval of the Laval University Medical Ethics Committee. The participants were subjected to a complete physical examination and were required to be healthy but sedentary. No subject had a history of recent illness, diabetes, hyperlipidemia, or other endocrine disorders. The zygosity was established from a questionnaire and on the basis of genetic concordance for 11 red blood cell antigens and enzymes (15 loci) and for the A, B, and C loci of the HLA system. Twins that were discordant for one of these loci were excluded from the study.

**Experimental Protocol**

A few weeks before the beginning of the experiment, the subjects were instructed to complete a dietary record for 3 days that included a weekend day as previously described.17 The reliability of the assessment of daily energy intake by this method has already been reported.17 During the experiment, the subjects were housed in an experimental station located in a park about 80 km from the laboratory. The men were instructed to refrain from exercise and their energy intake was carefully monitored by nutritionists during a 4-day baseline period. The daily caloric intake during the experimental protocol was determined from the mean energy intake obtained from the 3-day dietary journal and the 4-day observation period and corresponded to 14.4 ±0.8 MJ (3419 ±200 kcal) per day. This value represents the mean daily energy intake during the entire exercise training program. Once the assessment of basal energy needs was completed, the men were trained on bicycle ergometers for 22 consecutive days at 58% of their maximal oxygen uptake (VO2max). The exercise duration was 116 min/day and was calculated to induce a daily energy deficit of 4.2 MJ (1000 kcal). Two exercise sessions were performed each day to produce this energy deficit. During each exercise session, heart rate was recorded at every 3 minutes. All meals during the training program were prepared and measured by nutritionists and no additional food was available other than that distributed by nutritionists. The mean contribution of carbohydrate, fat, and protein intake corresponded to 45%, 38%, and 17% of the daily energy intake, respectively.

The effect of heredity in determining the sensitivity of various metabolic variables to exercise training and genotype-training interaction effects have been reported for carbohydrates18 and adipose tissue metabolisms,19 as well as for resting metabolic rate and dietary-induced thermogenesis.20 Thus, an extensive description of the experimental protocol has already been reported.20 The present study focuses on plasma lipid and lipoprotein changes and on the effect of heredity in determining the magnitude of their responses to exercise training.

**Laboratory Measurements**

The following measurements were made at the beginning and at the end of the experiment. VO2max was assessed by a progressive test to exhaustion on a bicycle ergometer. The initial work load was 50 watts and was increased by 25 watts until exhaustion. VO2 was recorded using an open gas circuit system and VO2max was considered as the highest VO2 recorded during the test for 1 minute.21 Subcutaneous skinfolds were measured on the left side of the body using the Harpenden skinfold caliper. Biceps, triceps, subscapular, calf, abdominal, and suprailiac skinfold thicknesses were measured by using the procedures of the International Biological Program.22 The sum of these six skinfolds was used as an indicator of subcutaneous fat,23 and the ratio of trunk skinfolds (subscapular, abdomen, suprailiac) divided by extremity skinfolds (biceps, triceps, calf) was used as an indicator of subcutaneous adipose tissue distribution.24,25 Body density was determined by underwater weighing26 and the percent body fat was calculated with the equation of Siri.27 The residual lung volume was assessed by the method of Wilmore et al.28 Blood samples were obtained in the morning after a 12-hour fast for the measurement of plasma lipids and lipoproteins. Plasma TG and CHOL were measured, whereas low density lipoprotein cholesterol (LDL-CHOL) was estimated by using the equation of Friedewald et al.21 Plasma apolipoprotein B (apo B) was measured by the electroimmunoassay of Laurell22 as previously described.18,32 An oral glucose tolerance test (OGTT) was also performed after an overnight fast, and plasma glucose and insulin concentrations were measured as previously described.18 Glucose and insulin areas were calculated by the trapezoid method. The effects of the present exercise training program on plasma glucose and insulin levels during the OGTT have already been reported.18 In the present study, these variables are used only as independent variables.

**Statistical Analysis**

The effect of exercise training was analyzed by using a two-way analysis of variance for repeated measures on one factor as previously described.20 The intraclass correlation coefficient for relative changes, which measures the intrapair resemblance in the response to exercise training, was calculated from the between- and the within-twin pairs variances as outlined by Haggard.34 Pearson product-moment coefficients were also computed to estimate the associations between the variables. As subjects of the same twin pair were not independent from each other, these correlations should be considered as trends, rather than as accurate measurements of the relations between the variables.
The exercise training produced a daily caloric deficit of 1000 kcal. Thus, changes in plasma CHOL ranged from a decrease of only 2%. A comparable variation in changes in plasma TG ranged from a decrease of 60% to +2% and in the change in the insulin/glucose areas (from -63% to +14%) was also noted.

The exercise training produced a daily caloric deficit of 1000 kcal. Thus, changes in plasma CHOL and LDL-CHOL (decreases of 11% and 16% respectively) and for an increase in plasma HDL-CHOL (increase of 16%) were not significant. However, these trends contributed to a significant increase in the HDL-CHOL/CHOL ratio (increase of 32%, p<0.05) in plasma apo B concentration did not change significantly.

Although only changes in plasma TG and in the HDL-CHOL/CHOL ratio were significant, the men again displayed individual variations in their responses to the exercise training prescription. A variation in response for the changes in insulin area after oral glucose ingestion (from −62% to +2%) and in the change in the insulin/glucose areas (from −63% to −14%) was also noted.

Table 2 presents the effects of the training program on the concentration of plasma lipids and lipoproteins. Plasma TG decreased significantly in response to the exercise program, whereas the trends for decreases in plasma CHOL and LDL-CHOL (decreases of 11% and 16% respectively) and for an increase in plasma HDL-CHOL (increase of 16%) were not significant. However, these trends contributed to a significant increase in the HDL-CHOL/CHOL ratio (increase of 32%, p<0.05). Plasma apo B concentration did not change significantly.

Table 2 presents the effects of the training program on Carbohydrate Metabolism In Six Pairs of Male MZ Twins. The exercise training produced a daily caloric deficit of 1000 kcal. Thus, changes in plasma CHOL and LDL-CHOL (decreases of 11% and 16% respectively) and for an increase in plasma HDL-CHOL (increase of 16%) were not significant. However, these trends contributed to a significant increase in the HDL-CHOL/CHOL ratio (increase of 32%, p<0.05). Plasma apo B concentration did not change significantly.

Table 1. Effects of 22 Days of Exercise Training on Body Fatness, Maximal Aerobic Power, and Carbohydrate Metabolism In Six Pairs of Male MZ Twins

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before</th>
<th>After</th>
<th>% change (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>77.3±7.0</td>
<td>74.9±7.1†</td>
<td>−3% (−6% to −1%)</td>
</tr>
<tr>
<td>% body fat (%)</td>
<td>18.3±3.2</td>
<td>15.9±3.5*</td>
<td>−18% (−49% to −1%)</td>
</tr>
<tr>
<td>Sum of six skinfolds (mm)</td>
<td>67.7±12.7</td>
<td>60.5±12.6*</td>
<td>−13% (−21% to −2%)</td>
</tr>
<tr>
<td>T/E skinfolds ratio</td>
<td>1.58±0.29</td>
<td>1.61±0.24**</td>
<td>+3% (−4% to +16%)</td>
</tr>
<tr>
<td>VO2max (l/min)</td>
<td>3.53±0.13</td>
<td>3.78±0.11*</td>
<td>+7% (0% to +14%)</td>
</tr>
<tr>
<td>VO2max (ml/kg/min)</td>
<td>48.4±2.8</td>
<td>53.1±3.1*</td>
<td>+10% (−1% to +17%)</td>
</tr>
<tr>
<td>Glucose area</td>
<td>16.410±664</td>
<td>17.073±493**</td>
<td>+5% (−17% to +27%)</td>
</tr>
<tr>
<td>Insulin area</td>
<td>13.569±1397</td>
<td>7.556±1336†</td>
<td>−46% (−62% to +2%)</td>
</tr>
<tr>
<td>Insulin/glucose areas</td>
<td>0.84±0.09</td>
<td>0.45±0.09‡</td>
<td>−49% (−63% to −14%)</td>
</tr>
</tbody>
</table>

The values are means ± SEM.

The effect of training was assessed by a two-way analysis of variance for repeated measures on one factor (exercise training) with twins nested in pairs. T/E skinfolds ratio = trunk skinfolds (subscapular, abdomen, and suprailiac)/extremity skinfolds (triceps, biceps, and calf) ratio; Sum of six skinfolds = sum of trunk and extremity skinfolds; VO2max = maximal oxygen consumption; Glucose area = sum of plasma glucose concentrations (mg/dl/180 min) during the oral glucose tolerance test; Insulin area = sum of plasma insulin concentrations (μU/ml/180 min) during the oral glucose tolerance test; Insulin/glucose areas = ratio of the insulin area over the glucose area.

The exercise training produced a daily caloric deficit of 1000 kcal.

Values are given as mg/dl and are the means ± SEM.

The effect of training was assessed by a two-way analysis of variance for repeated measures on one factor (exercise training) with twins nested in pairs. CHOL = cholesterol, TG = triglyceride, apo = apoprotein, LDL = low density lipoprotein, HDL = high density lipoprotein.

The exercise training produced a daily caloric deficit of 1000 kcal.

*p<0.005, †p<0.001, ‡p<0.0001, ns = not significant.

Results

The 22-day exercise training program significantly increased VO2max expressed in absolute values (l/min) or as per kilogram of body weight (Table 1). Body fatness was also significantly reduced as indicated by the significant decreases in body weight, percent of body fat, and in the sum of skinfolds. The proportion of subcutaneous fat in the trunk did not change significantly, as reflected by the lack of change in the sum of trunk/skinfolds ratio. There was no effect of exercise training on the plasma glucose area during the glucose tolerance test; Insulin area = sum of plasma insulin concentrations (μU/ml/180 min) during the oral glucose tolerance test; Insulin/glucose areas = ratio of the insulin area over the glucose area.

The exercise training produced a daily caloric deficit of 1000 kcal.

Values are given as mg/dl and are the means ± SEM.

The effect of training was assessed by a two-way analysis of variance for repeated measures on one factor (exercise training) with twins nested in pairs. CHOL = cholesterol, TG = triglyceride, apo = apoprotein, LDL = low density lipoprotein, HDL = high density lipoprotein.

The exercise training produced a daily caloric deficit of 1000 kcal.

*p<0.005, †p<0.001, ‡p<0.0001, ns = not significant.
Table 3. *F* Ratios for within-Twin Pair Resemblance for Effect of Exercise Training, and within-Pair Similarity in Response to Exercise Training

<table>
<thead>
<tr>
<th>Variable</th>
<th>Resemblance before exercise training</th>
<th>Exercise training effect</th>
<th>Genotype-exercise training interaction</th>
<th>Intraclass coefficient for relative changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma CHOL</td>
<td>0.90§</td>
<td>3.0**</td>
<td>23.8</td>
<td></td>
</tr>
<tr>
<td>Plasma TG</td>
<td>0.67†</td>
<td>23.2‡</td>
<td>0.6*</td>
<td>−0.26*</td>
</tr>
<tr>
<td>Plasma apo B</td>
<td>0.87§</td>
<td>4.4**</td>
<td>16.0§</td>
<td>0.88§</td>
</tr>
<tr>
<td>Plasma LDL-CHOL</td>
<td>0.89§</td>
<td>3.7**</td>
<td>18.3§</td>
<td>0.90§</td>
</tr>
<tr>
<td>Plasma HDL-CHOL</td>
<td>0.48*</td>
<td>1.7**</td>
<td>5.1†</td>
<td>0.67†</td>
</tr>
<tr>
<td>HDL-CHOL/CHOL</td>
<td>0.65†</td>
<td>6.3†</td>
<td>10.9‡</td>
<td>0.83‡</td>
</tr>
</tbody>
</table>

*This *F* ratio is for within-MZ pair similarity in response to overfeeding.

CHOL = cholesterol, TG = triglyceride, apo = apoprotein, LDL = low density lipoprotein, HDL = high density lipoprotein.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Relative changes in VO$_{\text{max}}$</th>
<th>% Body fat</th>
<th>Trunk fat</th>
<th>Extremity fat</th>
<th>T/E ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma CHOL</td>
<td>−0.24</td>
<td>0.15</td>
<td>0.70†</td>
<td>0.20</td>
<td>0.74†</td>
</tr>
<tr>
<td>Plasma TG</td>
<td>−0.05</td>
<td>−0.27</td>
<td>−0.04</td>
<td>0.12</td>
<td>−0.19</td>
</tr>
<tr>
<td>Plasma apo B</td>
<td>−0.15</td>
<td>0.28</td>
<td>0.79‡</td>
<td>0.33</td>
<td>0.72‡</td>
</tr>
<tr>
<td>Plasma LDL-CHOL</td>
<td>−0.18</td>
<td>0.33</td>
<td>0.78‡</td>
<td>0.35</td>
<td>0.68*</td>
</tr>
<tr>
<td>Plasma HDL-CHOL</td>
<td>−0.04</td>
<td>0.14</td>
<td>0.18</td>
<td>−0.15</td>
<td>0.40</td>
</tr>
<tr>
<td>HDL-CHOL/CHOL</td>
<td>0.17</td>
<td>−0.24</td>
<td>−0.39</td>
<td>−0.29</td>
<td>−0.22</td>
</tr>
<tr>
<td>Plasma glucose area</td>
<td>0.00</td>
<td>−0.17</td>
<td>−0.19</td>
<td>−0.37</td>
<td>0.15</td>
</tr>
<tr>
<td>Plasma Insulin area</td>
<td>−0.09</td>
<td>−0.20</td>
<td>0.39</td>
<td>−0.16</td>
<td>0.73†</td>
</tr>
<tr>
<td>Insulin/glucose areas</td>
<td>−0.12</td>
<td>−0.08</td>
<td>0.53</td>
<td>0.06</td>
<td>0.66*</td>
</tr>
</tbody>
</table>

CHOL = cholesterol, TG = triglyceride, apo = apoprotein, LDL = low density lipoprotein, HDL = high density lipoprotein, Glucose area = sum of plasma glucose concentrations (mg/dl/180 min) during the oral glucose tolerance test, Insulin area = sum of plasma insulin concentrations (µU/ml/180 min) during the oral glucose tolerance test, Insulin/glucose area = ratio of the insulin area over the glucose area in plasma during the oral glucose tolerance test, VO$_{\text{max}}$ = maximal oxygen consumption, Trunk fat = sum of subscapular, abdominal, and suprailiacl skinfolds; Extremity fat = sum of biceps, triceps, and calf skinfolds; T/E ratio = trunk/extremity skinfolds ratio.

*p<0.05, †p<0.01, ‡p<0.005, ||p<0.0001, ns = not significant.

Table 4. Correlation Coefficients between Relative Changes in VO$_{\text{max}}$, Percent Body Fat, Subcutaneous Fat Distribution, and Relative Changes in Carbohydrate Metabolism, as well as Relative Changes in Plasma Lipids and Lipoproteins after Exercise Training.

Table 3 suggests that this variation in response to exercise training was associated with the genotypic characteristics of the subjects. Before training, there was a significant within-pair resemblance for all plasma variables (0.65≤r≤0.90, 0.05>p>0.005) except plasma HDL-CHOL. There was also a significant intrainpair resemblance in the relative changes in plasma CHOL, apo B, LDL-CHOL, HDL-CHOL, and the HDL-CHOL/CHOL ratio (0.67≤r≤0.92, 0.05>p>0.0001), indicating a high level of similarity within MZ twin pairs in the magnitude of changes in plasma lipoprotein profile in response to training.

Table 4 shows that changes in plasma lipids and lipoproteins were not correlated with changes in the percent of body fat nor with changes in VO$_{\text{max}}$. However, when the relative loss of trunk fat was considered, significant correlations were observed between the individual changes in the relative proportion of trunk fat and changes in plasma CHOL, LDL-CHOL, and apo B. We previously reported that this training protocol significantly reduced the insulin response to an oral glucose challenge.18 In the present study, the indicators of carbohydrate metabolism were analyzed as independent variables in an attempt to further understand the mechanisms by which alterations in body fat distribution are associated with changes in plasma lipoproteins. Table 4 indicates that changes in the plasma glucose and insulin areas during the glucose tolerance test, as well as the changes in the ratio of insulin/glucose areas, were not correlated with the magnitude of changes in percent body fat nor with the changes in VO$_{\text{max}}$. Changes in the trunk/extremity skinfolds ratio were, however, significantly correlated with alterations in both the insulin area (r=0.73, p<0.01) and the ratio of insulin/glucose areas (r=0.66, p<0.05). These results indicate that subjects who lost more trunk fat compared to other individuals were those who also showed the greatest improvements in their carbohydrate metabolism.

Figure 1 shows that changes in the ratio of insulin/glucose areas after training were correlated with changes in plasma apo B (−27% to +18%), HDL-CHOL (−35% to +50%), and in the HDL-CHOL/CHOL ratio (−22% to +80%) was observed.
plasma CHOL (Figure 1A) but not with changes in plasma TG (Figure 1B). Figure 2 indicates that changes in the insulin/glucose areas were correlated with changes in plasma apo B (Figure 2A) and in plasma LDL-CHOL (Figure 2B), suggesting a synchronized response to exercise training for body fat distribution, carbohydrate metabolism, and some plasma lipoproteins.

Because changes in plasma lipoproteins were correlated with changes in body fat distribution (Table 4) and with the initial level of body fat distribution (for CHOL, apo B, and LDL-CHOL, respectively, p<0.01), the within-pair similarity in lipoprotein response was studied after control for relative change in body fat distribution or for the initial level of adipose tissue distribution (Table 5). After these controls, the within-pair similarity in lipoprotein response was no longer observed, suggesting that the genetic effect on the response of these plasma variables was mediated by a similar initial level or by response in body fat distribution.

The within-pair resemblance in HDL-CHOL/CHOL response to training remained, however, statistically significant, suggesting that this genotype-training interaction effect is not mediated by the within-pair resemblance in body fat distribution.

Discussion

The results of this study indicate that biological inheritance is an important determinant of the heterogeneous response of plasma lipids and lipoproteins to an exercise training program that generates a chronic negative energy balance for 22 days. Indeed, whereas only plasma TG concentration and the HDL-CHOL/CHOL ratio were significantly modified by this short-term, exercise training program, the subjects showed substantial individual variations in plasma lipid and lipoprotein responses to the program. Thus, with the exception of serum TG, changes in CHOL, LDL-CHOL, HDL-CHOL, and apo B tended to be similar within twin pairs (0.05>p<0.0001). The responses of plasma CHOL, apo B, and LDL-CHOL were correlated with changes in body fat distribution (Table 4) and with the initial level of body fat distribution (for CHOL, apo B, and LDL-CHOL, respectively, p<0.01).
were, however, correlated with the initial level and with the change in body fat distribution. After statistical control for body fat distribution (initial level or change), the within-pair similarity in CHOL, apo B, and LDL-CHOL responses was no longer observed. These results suggest that the genotype-training interaction effect noted for the response of CHOL, apo B, and LDL-CHOL is mediated by the effect of heredity on body fat distribution. The response of the HDL-CHOL/CHOL ratio to exercise training, which was also influenced by heredity, was not due to the twins' similarity in initial levels nor to changes in body fat distribution. However, with only six different genotypes studied, these statistical controls for resemblance in body fat distribution should be interpreted with caution.

Numerous exercise training studies have reported changes in plasma lipids and lipoproteins, and these changes in the lipoprotein-lipid profile are considered to be favorable in reducing the potential risk of CVD. In the present study, along with a decrease in serum triglyceride levels, we also noted a significant increase in the proportion of plasma CHOL carried in the HDL fraction (HDL-CHOL/CHOL). Although the atherogenicity of high serum TG concentrations is still a subject of controversy, its reduction after training indicates a decreased secretion of TG-rich particles by the liver combined with a higher capacity of the endothelium to catabolize TG in circulating lipoproteins. The increase in the HDL-CHOL/CHOL ratio is undoubtedly a beneficial effect of the exercise program, because this ratio is considered a foremost predictor of cardiovascular disease events.

Many studies have reported reduction in plasma CHOL and an increase in HDL-CHOL after exercise training, although not all studies are concordant. In the present experiment, we also observed similar trends for these two variables, but they did not attain statistical significance. The magnitude of decreases in the concentrations of plasma CHOL (11%), apo B (12%), and LDL-CHOL (16%) and the concomitant increase in HDL-CHOL concentration (16%) is comparable to the exercise training effects generally reported. The non-significance of these changes could be attributed partly to the small number of subjects. In addition, although severe (2 hours of aerobic exercise per day), the exercise program was of short duration. In addition, the initial condition of the men in this study (young, nonobese, with normal lipoprotein profiles) was undoubtedly a factor in minimizing the effect of exercise training on plasma lipoproteins. The initial levels of lipoproteins and lipids have already been reported to be an important variable in the determination of the magnitude of the lipoprotein responses to an exercise training program.

It is logical to speculate that such a training program in subjects with abnormal lipoprotein profiles would have produced more substantial changes. Despite the controlled environment (restricted caloric intake and exercise training), an Individual variation in the response to training was clearly evident. However, further work is required to identify the mechanisms responsible for the presence of low and high responders to intervention measures aimed at reduction of the CVD risk.

It is noteworthy that individual changes in plasma lipoproteins and lipids were dissociated from changes in percent body fat and VO2max. A reduction in the proportion of subcutaneous trunk fat, however, appeared to be associated with the response of postglucose plasma insulin and of plasma lipoproteins to exercise training. Indeed, the results indicate that subjects who responded with the greatest reduction in their proportion of trunk fat as reflected by the trunk/extremity skinfolds ratio were the ones who showed the greatest reductions in plasma CHOL, LDL-CHOL, and apo B levels and in whom there were the largest reductions in postglucose plasma insulin and insulin/glucose areas. These results suggest a dynamic association between fat distribution and plasma lipoproteins, as well as between changes in body fat topography and changes in the carbohydrate metabolism. Associations between body fat distribution and indicators of carbohydrate metabolism have been reported, and recent data have also demonstrated that body fat topography is associated with plasma lipoprotein and lipid concentrations. Therefore, the association between body fat distribution and metabolic complications could help to explain the relationship between adipose tissue localization and CVD events in recent prospective studies.

To our knowledge, the relationship between body fat localization and CVD risk factors has not been studied in a controlled study where weight loss occurs. Thus, we wondered whether the subjects showing the greatest loss of trunk fat were also those showing the most substantial changes in insulin metabolism and in lipoprotein-lipid profile. Our results show that this was, indeed, the case, as indicated by significant correlations between changes in the trunk/extremity skinfolds ratio and changes in the insulinogenic index (postglucose plasma insulin/glucose areas). Furthermore, decreases in the insulinogenic index were associated with reductions in plasma CHOL, LDL-CHOL, and apo B concentrations. All these changes were also associated with alterations in fat distribution but were dissociated from changes in total body fat. The importance of body fat distribution in the response of CHOL,
LDL-CHOL, and apo B to exercise training was such that, after control for initial level or change in body fat distribution, no within-pair similarity was observed in the response of these plasma variables. Because the insulinogenic index was closely associated with changes in CHOL, LDL-CHOL, apo B, and in body fat distribution, these results suggest that the genetically determined response of CHOL, LDL-CHOL and apo B was due to the within-pair resemblance in body fat distribution and was mediated by changes in plasma insulin levels. The mechanism relating body fat distribution to insulin metabolism has been recently reviewed. 

The mechanism by which body fat localization is correlated with plasma lipoprotein lipids remains to be established. However, the present data suggest that some of the effects of fat distribution on plasma lipoproteins are mediated by changes in the metabolism of insulin. The relationship between TG-rich lipoproteins and plasma insulin levels is well documented, but the effects on CHOL-rich lipoproteins are less well understood. The significant correlation observed between changes in the insulin/glucose areas and LDL-CHOL suggests an augmented uptake of LDL particles by the apolipoprotein B-E receptor because it has been shown that insulin can modulate the receptor uptake and catabolism of LDL. Therefore, it is possible that in situations of improved insulin sensitivity such as in exercise trained subjects, the LDL receptor activity is enhanced. The similar correlation between insulin/glucose areas and plasma apo B levels also supports the direct removal of LDL particles, but does not rule out a similar removal mechanism for the particles of intermediary density that are produced during increased TG hydrolysis. These changes could also be attributed to exercise-training-associated variations in plasma insulin levels. Further work is required to substantiate this hypothesis.

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- monozygotic twins  aerobic exercise  insulin

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