Effect of Weight Loss in Moderate Obesity on Plasma Lipoprotein and Apolipoprotein Levels and on High Density Lipoprotein Composition

Joseph Zimmerman, Nathan A. Kaufmann, Menachem Fainaru, Shlomo Eisenberg, Yitzchak Oschry, Yechiel Friedlander, and Yechezkiel Stein

Plasma lipids, lipoprotein and apolipoprotein levels were determined in seven women and seven men with moderate obesity before, during 7 weeks of continuous weight loss (10.4% to 9.6% of body weight, 1000 kcal/day diet), and after 3 months at a stable, reduced weight. Plasma triglyceride levels decreased by 30.4% in men and by 39.4% in women (p < 0.0001) after 1 week of caloric restriction and remained at this level throughout the study period. The plasma cholesterol decreased by 19.0% in men (p < 0.001) and by 10.9% in women (p < 0.01) in the period of active weight loss, but returned to prediet values after stabilization at a leaner body mass. Similar changes were observed in LDL cholesterol levels. No change in high density lipoprotein (HDL) cholesterol levels occurred during active weight reduction, but after 3 months at a reduced weight, a significant increase in HDL cholesterol was evident, and the ratio of HDL cholesterol to plasma cholesterol increased over prediet values (p < 0.001, women). Separation of HDL subpopulations by zonal ultracentrifugation before and after weight reduction revealed that HDL₂ increased slightly in men and decreased slightly in women. In both genders, HDL₃ tended to decrease after weight reduction. Plasma levels of apolipoprotein A-I decreased during active weight loss, but this was significant only in women (p < 0.05). After 3 months of reduced weight, plasma apo A-I increased to prediet levels. No significant changes in plasma apo A-II or apo E were noted. Our results indicate that in moderately obese, but otherwise healthy, subjects weight reduction achieved by caloric restriction does not affect HDL composition or subpopulation distribution significantly. Moreover, maintenance of a leaner body mass has a beneficial long-term effect by increasing HDL cholesterol and decreasing plasma triglycerides. (Arteriosclerosis 4:115-123, March/April 1984)
Previous studies of the effects of weight reduction on plasma and lipoprotein lipid levels have yielded controversial results, mainly with regard to HDL-C levels. This is partly due to the lack of controlled dietary regimens and the heterogeneity of the subjects studied. Some of these investigations were conducted on subjects with morbid obesity, which may constitute a unique subset of patients incomparable to the more common "simple" obesity. The purpose of the present study was, therefore, to assess the impact of a controlled weight reduction program using a balanced, acceptable, hypocaloric diet on plasma and lipoprotein lipid and apolipoprotein levels in moderately obese subjects. Particular attention was focused on the effects of the diet on HDL-C, HDL subpopulation distribution, and HDL apolipoproteins during the phase of active weight reduction and after several months of stabilization at a leaner body mass.

**Methods**

Fourteen obese volunteers (seven women and seven men) were enrolled in the study. All were members of the hospital staff or their spouses. The study protocol was approved by the Hadassah University Hospital Helsinki Committee. All subjects gave informed consent to participate in the study prior to their enrollment.

The ages, weights, body mass indexes, and lipoprotein profiles of the subjects studied are summarized in Table 1. All subjects were in good health, as judged by medical history, physical examination, and laboratory tests, which included fasting serum glucose, electrolytes, urea, creatinine, albumin, liver and thyroid function tests and lipoprotein profile. None was taking any medication during the study period or in the 8 weeks preceding it. None of the women was or had been pregnant or treated with oral contraceptives during the study period or 12 months beforehand. Three subjects (two women and one man) smoked up to 20 cigarettes daily. None consumed alcohol regularly.

**Diet**

A diet of 1000 Kcal/day was prepared in the metabolic unit. Of total calories, 35% were supplied as carbohydrates, 30% as fat, and 35% as protein. The polyunsaturated/saturated (P/S) fatty acid ratio was 0.92, and the cholesterol content was 480 mg/day, values similar to those of a common Israeli diet. The diet was prepared by a certified dietician. Natural food commodities were used and the variety of items used was as small as possible to ensure consistency of the diet composition (i.e., only one kind of meat was used throughout the experiment). Menu variety was achieved by changing ways of cooking and seasoning. The contents of nutrients were calculated using the Israel Tables of Food Composition. The basic diet plan was constructed and a composite menu was sent to a central laboratory for chemical analysis. The chemically determined values were in good agreement with those calculated using the tables. Laboratory analysis of the percentages of protein and fat in the diet differed from calculated values by no more than 3%. For carbohydrates, similar differences were found, but on one occasion the difference was 4.9%. The difference between calculated and determined P/S ratios was less than 0.25.

Subjects came to the metabolic ward once a day

<p>| Table 1. Physical Characteristics and Lipid Profiles of the Study Group before Weight Reduction |
|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|</p>
<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Initial body weight (kg)</th>
<th>Quetelet* Index</th>
<th>Initial plasma lipids (mg/dl), mean ± sd</th>
</tr>
</thead>
<tbody>
<tr>
<td>G.F.</td>
<td>33</td>
<td>M</td>
<td>86.0</td>
<td>30.1</td>
<td>143.6 ± 2.9</td>
</tr>
<tr>
<td>F.I.</td>
<td>32</td>
<td>M</td>
<td>98.8</td>
<td>32.0</td>
<td>215.5 ± 13.4</td>
</tr>
<tr>
<td>O.A.</td>
<td>30</td>
<td>M</td>
<td>110.3</td>
<td>35.9</td>
<td>183.0 ± 7.0</td>
</tr>
<tr>
<td>E.S.</td>
<td>50</td>
<td>M</td>
<td>119.0</td>
<td>31.1</td>
<td>259.3 ± 3.5</td>
</tr>
<tr>
<td>A.D.</td>
<td>48</td>
<td>M</td>
<td>99.8</td>
<td>35.1</td>
<td>214.0 ± 7.0</td>
</tr>
<tr>
<td>A.W.</td>
<td>30</td>
<td>M</td>
<td>107.5</td>
<td>30.7</td>
<td>180.0 ± 5.3</td>
</tr>
<tr>
<td>S.T.</td>
<td>35</td>
<td>M</td>
<td>99.5</td>
<td></td>
<td>202.0 ± 12.1</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>36.8 ± 3.2</td>
<td></td>
<td>102.9 ± 3.9</td>
<td>32.0 ± 0.9</td>
<td>199.6 ± 13.6</td>
</tr>
</tbody>
</table>

*Quetelet Index = body weight/height², kg/m²; normal values = 20–26 (see reference 47).
for either lunch or breakfast. The meals for the rest of the day were supplied in a container, and the subjects were asked to return food items that were not consumed on the following day. Usually, none was returned. A similar procedure was adopted for weekends; the only difference was that the subjects were supplied with food for 48, rather than 24, hours.

**Experimental Design**

Before the study began, three weekly 12-hour fasting blood samples were obtained from each subject. Throughout the study period, which lasted for 7 to 8 weeks, the subjects were ambulatory and continued their usual activities and smoking habits. Alcohol drinking was prohibited during the study period. The subjects agreed to consume all the food they were given and only this food. Body weight was monitored daily. During the study, 12-hour fasting blood samples were collected weekly from an antecubital vein into vacutainer tubes containing EDTA (1 mg/ml). The plasma was separated promptly by centrifugation at 2000 rpm for 20 minutes at 4°C. Aliquots of plasma were stored at −20°C for apolipoprotein quantification.

After completion of the 7 to 8-week controlled weight-reduction program, the patients were instructed to consume a weight-maintaining diet similar in composition to the experimental diet, and were examined again after 3 months. At that time, body weight was recorded and a fasting blood sample was drawn for plasma lipids, lipoprotein lipids, and plasma apolipoproteins.

**Analytical Methods**

Plasma cholesterol and triglycerides were analyzed with the Technicon Autoanalyzer II, following extraction with isopropranolol according to the Lipid Research Clinics protocol.29 The cholesterol content of the major lipoproteins, VLDL, LDL, and HDL, was measured using the same protocol.29 In short, 5 ml plasma samples were centrigfuged at a density of 1.006 g/ml for 18 hours at 39,000 rpm at 4°C in a Beckman 40.3 rotor. The VLDL supernatant was removed by the tube-slicing technique.30 HDL cholesterol was estimated after heparin-manganese precipitation of the 1.006 g/ml infranatant. LDL and VLDL cholesterol levels were calculated from the total plasma cholesterol, the cholesterol content in 1.006 g/ml infranatant (= LDL + HDL cholesterol), and the HDL cholesterol.

Apolipoproteins A-I, A-II, and E in plasma were quantified by specific radioimmunoassay procedures described previously.31–34 Briefly, double antibody radioimmunoassays were performed using the purified apolipoproteins and their respective monospecific antibodies, prepared in rabbits. Antibodies against rabbit IgG were prepared in a goat, and used as a second antibody. The purified apolipoproteins were radio labeled with ^125I by the chloramine-T method.32 Plasma samples were assayed in appropriate dilutions in triplicates, and counted in a Gamma scintillation spectrometer (Packard). The intraassay and interassay coefficients of variations for apolipoprotein quantification were 5% and 9% respectively. All samples of an individual subject were analyzed in one assay, thus minimizing the interassay variation.

Separation and characterization of HDL subfraction was carried out before and after the weight-reducing diet; 10 ml of 12-hour fasting plasma was adjusted to density 1.4 g/ml with solid NaBr and applied to the bottom of a discontinuous gradient of 1.0–1.4 g/ml NaBr in a Ti-14 zonal rotor spinning at 3,000 rpm in a Beckman L5-50 Ultracentrifuge.30 The rotor was accelerated and spun at 41,000 rpm for 22 hours, after which it was decelerated and its contents were displaced with heavy salt solution. Zonal rotor effluent was continuously monitored at 280 nm (ISCO, Model UA-5 Absorbance Monitor, Instrumentation Specialties Company, Lincoln, Nebraska) and collected in 25 ml fractions. The HDL₂ and HDL₃ subfractions were identified, pooled, and concentrated by vacuum ultrafiltration. The total cholesterol and free cholesterol were determined in these fractions by the cholesterol oxidase method using a commercially available kit (Boehringer Mannheim Diagnostica, Mannheim, West Germany). Total protein was determined by the method of Lowry,35 and phospholipids were determined as organic phosphorus by the method of Bartlett.37

**Statistical Methods**

We used one-way analysis of variance for analyzing the data of these subjects; all had eight repeated measurements on the same variable at a fixed interval of time.36 This method enabled us to partition the total variability into three major components: 1) between-subject variation, 2) between-treatment variation, and 3) within-person residual variation. As the follow-up values of the subjects were not taken at fixed intervals, they were compared to the mean predicted values and to the last values on the in-unit diet by paired t test.

**Results**

Significant weight loss occurred in all subjects during the study period. The mean weight reduction (± SEM) after 7 weeks was 9.6 ± 2.1% of initial body weight in men and 10.4 ± 2.1% in women (p < 0.001 for both groups).

Plasma and lipoprotein lipid levels and plasma apolipoprotein levels during weight reduction are presented in Figures 1–7. In men, plasma triglyceride concentration decreased from a mean of three weekly prediet determinations of 129.6 to 90.2 mg/dl after 7 weeks of weight reduction, a fall of 30.4% (mean of percent individual change values); in women the concentration decreased from 110.7 to 63.7 mg/dl, a fall of 39.4% (Figure 1). In both men and women, the triglyceride level reduction was highly significant (p < 0.0001). It is noteworthy that the
A decrease in plasma triglycerides occurred during the first week of the diet, with no further change afterward, despite continuing weight loss throughout the whole dietary period. During the same period, the mean plasma total cholesterol in men decreased from 198.7 mg/dl (a mean of three weekly prediet determinations) to 159.4 mg/dl, a fall of 19.0% (p < 0.0001); in women, total cholesterol decreased from 172.4 to 153.8 mg/dl, a decrease of 10.9% (p < 0.01). However, plasma cholesterol levels decreased gradually and stabilized at lower levels only after 3 weeks of weight reduction (Figure 2). LDL-C levels showed a similar pattern to that of plasma total cholesterol (Figure 3). The decrease of LDL-C was highly significant in men (p < 0.0001), but not in women (p > 0.1). In spite of the marked decline of plasma triglyceride, plasma cholesterol, and LDL-C levels concomitant with weight loss, no significant...
changes occurred in HDL-C levels throughout the weight reduction period, in either women or men (Figure 4). Consequently, the ratio of HDL-C to plasma total cholesterol increased. This increase was significant only in male subjects (p < 0.01). In contrast to the absence of changes in HDL-C, plasma levels of apolipoprotein A-I decreased (Figure 5). However, this change was statistically significant only in female subjects (p < 0.05, paired t-test). No significant changes were observed in plasma apolipoprotein A-II and E levels (Figures 6 and 7).

Plasma HDL subpopulation distribution (HDL₂ and HDL₃) before and after weight reduction, was determined by centrifugation in a zonal rotor. A representative HDL elution profile of a female and a male subject is shown in Figure 8. In most male subjects, HDL₂ levels seemed to increase slightly, while no change or even some decrease was found in female subjects. HDL₃ levels showed a tendency to decrease in both genders. Measured levels of plasma HDL₂ protein, phospholipid, and cholesterol levels obtained after the zonal centrifugation confirmed these impressions, although none of the changes were statistically significant (Table 2).

Table 2. Composition of HDL₂ and HDL₃ Before and After Weight Reduction

<table>
<thead>
<tr>
<th>Plasmatic concentration</th>
<th>Men (n = 6)</th>
<th>Women (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before (mg/dl)</td>
<td>After (mg/dl)</td>
</tr>
<tr>
<td><strong>HDL₂</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>8.1 ± 2.2</td>
<td>11.4 ± 2.9</td>
</tr>
<tr>
<td>Phospholipid</td>
<td>6.4 ± 1.6</td>
<td>6.4 ± 2.2</td>
</tr>
<tr>
<td>Cholesterol ester</td>
<td>2.7 ± 1.0</td>
<td>4.1 ± 2.2</td>
</tr>
<tr>
<td>Free cholesterol</td>
<td>0.60 ± 0.3</td>
<td>0.92 ± 0.45</td>
</tr>
<tr>
<td><strong>HDL₃</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>76.2 ± 10.0</td>
<td>74.4 ± 11.6</td>
</tr>
<tr>
<td>Phospholipid</td>
<td>36.4 ± 3.6</td>
<td>32.3 ± 4.3</td>
</tr>
<tr>
<td>Cholesterol ester</td>
<td>27.8 ± 15.1</td>
<td>25.3 ± 14.2</td>
</tr>
<tr>
<td>Free cholesterol</td>
<td>3.0 ± 1.5</td>
<td>5.9 ± 3.9</td>
</tr>
</tbody>
</table>

Results are means ± SEM for seven women and six men.
Follow-up Studies

Five men and all seven women were available for re-examination 3 months after completion of the controlled diet period. After 3 months, all subjects maintained a reduced body weight, significantly lower than their initial body weight ($p < 0.01$) and not significantly different from their weight after 7 weeks of diet. The data of the follow-up study are presented in Table 3. After 3 months, plasma total cholesterol levels and LDL-C levels returned to prediet levels, but plasma triglycerides remained lower than prediet values. This was significant only in female subjects ($p < 0.01$). In both men and women, postdiet plasma HDL-C levels were significantly higher than the values after 7 weeks of weight reduction, and in female subjects the postdiet HDL-C levels were also significantly higher than prediet levels ($p < 0.02$). The ratio between HDL-C to plasma cholesterol increased significantly ($p < 0.001$) from prediet values in women, but not in men, after 3 months at a lower body weight.

Plasma apolipoprotein A-I decreased during the period of active weight reduction but returned toward pretreatment levels in parallel to the increase in HDL-C. No change was observed in plasma apolipoproteins A-II and E levels after 3 months of stable leaner body mass.

Discussion

In a recently published study, Wolf and Grundy demonstrated that active weight reduction in a heterogeneous group of female and male subjects (body weight 123% to 209% of ideal weight) results in a considerable decrease of plasma triglycerides, total cholesterol, and VLDL-C levels without a significant change of LDL-C and HDL-C levels. Our results generally agree with these observations although we studied separately women and men with moderate obesity. In the present study, moreover, investigations of HDL subpopulation distribution and apolipoprotein levels were included. In agreement with Wolf and Grundy and many other studies, we found the plasma triglyceride levels decreased with weight reduction, and remained lower than prediet values even 3 months after cessation of active weight loss. This change occurred promptly after the institution of the hypocaloric diet and was already established after 1 week on the diet. A transient decrease of plasma total cholesterol levels was observed during the period of active weight loss. This decrease was caused mainly by a decrease in LDL-C levels. Plasma cholesterol and LDL-C levels returned to prediet values 3 months after cessation of active weight loss. No significant change of HDL-C and HDL subpopulation distribution was observed during the period of active weight loss, although a tendency toward lower LDL values was found. As body weight stabilized at a leaner level, an increase in HDL-C levels became evident. Thus, during the acute phase of weight loss and caloric restriction, the well known reciprocal relationship between HDL-C and triglyceride levels was not observed.

This indicates that the mechanism(s) responsible for the reduction of plasma triglyceride levels during caloric restriction do not involve increased catabo-
Lipoprotein lipase levels during caloric restriction are reportedly low.\textsuperscript{21,42} We therefore suggest that decreased synthesis is responsible for the lower plasma triglyceride levels. The increase of HDL-C levels after stabilization of body weight, when presumably VLDL synthesis and lipoprotein lipase activity return to normal, supports this hypothesis. The ratio of HDL-C to total cholesterol increased both during active weight loss and after stabilization at a leaner body mass, but the factors involved in this increase were different in the two instances: during active weight loss the increase was due to the decrease of total cholesterol levels, whereas after stabilization at a lower body weight an increase in HDL-C level was the important factor.

HDL-apoipoprotein levels have not been systematically studied during active weight loss in moderately obese subjects. We found that apolipoprotein A-I levels were reduced at the end of the active weight loss (significant only in women). This reduction in the absence of a parallel change in HDL-C reflects a change in the protein/lipid ratio, and can be explained by an increase of HDL\textsubscript{2} and/or a decrease in HDL\textsubscript{3}. As stated earlier, an increase of HDL\textsubscript{2} levels was found in men but not in women, and there was a decrease of HDL\textsubscript{3} in both men and women. Such changes, although of small magnitude, may be sufficient to explain the observations. The decrease of apolipoprotein A-I levels during caloric restriction possibly reflects decreased intestinal synthesis secondary to severe reduction of fat intake.\textsuperscript{43,44}

The changes in plasma lipid and lipoprotein lipid levels showed the same pattern in both genders. This was observed both during the active weight reduction period and after stabilization of body weight. This finding is in contrast to the study of Brownell and Stunkard,\textsuperscript{19} who reported no change of HDL-C, but a marked decrease of LDL-C in men, while in women HDL-C decreased and LDL-C changed only slightly.

Some of the recently published studies of the effects of weight reduction achieved by dietary restriction on plasma lipids are summarized in Table 4. The

**Table 4. Effects of Weight Reduction in Obesity on Plasma Lipids**

<table>
<thead>
<tr>
<th>Author (ref)</th>
<th>Energy intake (kCal/day)</th>
<th>Duration (wks)</th>
<th>Plasma triglyceride</th>
<th>Cholesterol</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Body weight</td>
<td>A</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LaRosa (18)</td>
<td>14</td>
<td>1656</td>
<td>8</td>
<td>96.8</td>
</tr>
<tr>
<td>Brownell (19)</td>
<td>11</td>
<td>~1500</td>
<td>16</td>
<td>106.7</td>
</tr>
<tr>
<td>Weltman (20)</td>
<td>11</td>
<td>10</td>
<td>92.0</td>
<td>-5.9\textsuperscript{∗}</td>
</tr>
<tr>
<td>Schwartz (17)</td>
<td>8</td>
<td>600</td>
<td>1</td>
<td>121.0</td>
</tr>
<tr>
<td>Taskinen (21)</td>
<td>10</td>
<td>400</td>
<td>1</td>
<td>-4.2</td>
</tr>
<tr>
<td>Friedman (25)</td>
<td>10</td>
<td>1166</td>
<td>8</td>
<td>86.7</td>
</tr>
<tr>
<td>Brownell (19)</td>
<td>10</td>
<td>~1200</td>
<td>16</td>
<td>89.8</td>
</tr>
<tr>
<td>Thompson (22)</td>
<td>15</td>
<td>1000</td>
<td>1</td>
<td>100.3</td>
</tr>
<tr>
<td>Taskinen (21)</td>
<td>10</td>
<td>400</td>
<td>1</td>
<td>-4.2</td>
</tr>
<tr>
<td>Friedman (25)</td>
<td>10</td>
<td>1166</td>
<td>8</td>
<td>86.7</td>
</tr>
<tr>
<td>Mixed groups</td>
<td></td>
<td></td>
<td></td>
<td>128.6</td>
</tr>
<tr>
<td>Sörbäck (15)</td>
<td>14.8-F</td>
<td>240</td>
<td>5</td>
<td>101.5</td>
</tr>
<tr>
<td>Wolf (24)</td>
<td>15.3-F</td>
<td>1000</td>
<td>0</td>
<td>118.0</td>
</tr>
<tr>
<td>Contaldo (16)</td>
<td>7.5-F</td>
<td>4-6</td>
<td>128.6</td>
<td>-10.0</td>
</tr>
<tr>
<td>Stroka (23)</td>
<td>13.10-F</td>
<td>600-F</td>
<td>0</td>
<td>101.5</td>
</tr>
<tr>
<td>Olefsky (45)</td>
<td>36.8-F</td>
<td>600-1600</td>
<td>8-40</td>
<td>89.3</td>
</tr>
<tr>
<td>Tokunaga (27)</td>
<td>13.9-F</td>
<td>600-800</td>
<td>12</td>
<td>97.0</td>
</tr>
<tr>
<td>Avogaro (48)</td>
<td>30.16-F</td>
<td>600-800</td>
<td>6-8</td>
<td>162</td>
</tr>
</tbody>
</table>

Summary data from recently published studies on the effects of weight reduction in obese human subjects on plasma lipid and lipoprotein lipid levels. The studies are identified by the first author and by number in the list of references. Diet composition, cholesterol contents, and P/S ratios are not included. Data are calculated to express the percentage of change of mean prediet values, and the significance of the change is calculated by t test. Negative changes are presented by the symbol (−); positive changes are unmarked. Values are means.

A = the mean of prediet value, mg/dl; B = change in % from A.

*Original values (in SI units) converted to mg/dl.

\textsuperscript{∗}p < 0.05.

\textsuperscript{†}p < 0.01.

\textsuperscript{‡}p < 0.001.
most consistent finding is the tendency of plasma triglycerides to decrease, and this is in accord with our findings. Obesity is associated with an increase in triglyceride production rate, and weight reduction in these subjects may cause a decrease in VLDL synthesis.\(^\text{15}\) Plasma cholesterol levels also tended to fall with weight reduction but this is less consistent than the decrease of plasma triglycerides. Changes of LDL cholesterol levels during weight loss were closely correlated with changes in plasma total cholesterol levels (\(r = 0.91, p < 0.001\)). The data on the effect of weight reduction by caloric restriction on HDL-C levels are controversial. Seven studies show a significant decrease in HDL-C during weight reduction.\(^\text{18-22, 26-27}\) Four of these studies were performed on female subjects, and in one\(^\text{19}\) the pattern of response of HDL-C levels between men and women was significantly different. The data compiled in Table 4 and our results do not show gender-specific effects of weight reduction on plasma lipid and lipoprotein lipids. Six other studies\(^\text{15-17, 23, 24, 48}\) show an increase in HDL-C during weight reduction and in some of these studies,\(^\text{16, 23, 24, 48}\) the elevated HDL-C levels persisted after the period of active weight loss, during which time the subjects maintained a leaner body weight. Our results lend support to this latter observation.

The results presented in Table 4 should be interpreted with caution. First, some of these studies were conducted on patients with extreme morbid obesity.\(^\text{15-17, 23, 24}^\text{25}\) These may constitute a separate group, incomparable to subjects with moderate obesity, although no consistent trend related to this factor is evident in the table. The subjects in our study could be classified as moderately obese, as the highest Quetelet Index did not exceed 36. Second, in most of the studies, the dietary protocol was not completely specified as to its caloric content, composition, cholesterol content, or P/S ratio. Therefore, it is possible that dietary perturbations unrelated to caloric restriction per se contributed to the change in plasma lipids. In our study, the composition of the diet was kept constant and all subjects were given the same menu, thus permitting a comparison between male and female subjects. Moreover, the P/S ratio and the cholesterol content in the diet did not differ from that of the average Israeli diet,\(^\text{46}\) thus making caloric restriction the major determinant of the observed changes in plasma lipids. Third, in some of the studies compiled in Table 4, nondietary factors that are known to affect lipoprotein lipid levels, such as physical exercise and alcohol consumption, were not similar and were not kept constant throughout the experiment, and therefore might have contributed to the observed changes in plasma lipoproteins.

Epidemiologic surveys have repeatedly confirmed a significant negative association between HDL cholesterol levels and coronary heart disease.\(^\text{8, 14}\) Our results, as well as those of others, suggest that weight reduction by caloric restriction in moderately obese subjects has a salutary effect on plasma lipids by the immediate lowering of VLDL triglycerides and by the slow but sustained increase of HDL cholesterol on a leaner body mass. Theoretically, these changes may be beneficial in the goal to prevent coronary heart disease.

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**References**

19. Brownell KD, Stunkard AJ. Differential changes in plasma high-density lipoprotein-cholesterol levels in obese men and...
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