Levels and Interrelationships of Serum and Lipoprotein Cholesterol and Triglycerides

Association with Adiposity and the Consumption of Ethanol, Tobacco, and Beverages Containing Caffeine

Nancy R. Phillips, Richard J. Havel, and John P. Kane

We have carried out a cross-sectional multivariate analysis of serum and lipoprotein lipid levels in white persons from an industrial population. Very low density lipoprotein triglyceride level was independent of ethanol consumption but increased with adiposity and cigarette smoking and decreased with coffee-tea drinking. Concomitant age variation in indices of adiposity accounted for only a small part of the sex-specific age trends in triglyceride level. Very low density lipoprotein cholesterol level was independent of all variables considered when controlled for very low density lipoprotein triglyceride. Low density lipoprotein cholesterol covaried with very low density lipoprotein triglyceride among normotriglyceridemic persons. Small increases in low density lipoprotein cholesterol level with adiposity and cigarette smoking appeared to reflect associated increases in very low density lipoprotein triglyceride. Increased low density lipoprotein cholesterol level with age, however, was largely independent of concomitant age variation in very low density lipoprotein triglyceride. High density lipoprotein cholesterol level increased with ethanol consumption and decreased with adiposity and cigarette smoking even after adjustment for its inverse relationship with very low density lipoprotein triglyceride.


This paper presents the results of a cross-sectional multivariate analysis of variation in the concentrations of cholesterol and triglyceride in blood serum and major lipoprotein classes associated with adiposity and the consumption of ethanol, tobacco, and beverages containing caffeine, based on a defined population sample of white men and women, aged 20-64 years. Plasma triglyceride levels have previously been found in cross-sectional data to increase with adiposity,4,6,7 and ethanol consumption.5,6,8 Plasma cholesterol levels have also been found to increase with adiposity10,11 and cigarette smoking.7,12,13 but to be unrelated to ethanol consumption.5 When lipoprotein classes have been considered, the cholesterol level in the low density lipoprotein (LDL) fraction has been found to be inversely related to ethanol consumption9 and positively related to cigarette smoking,7 whereas that in the high density lipoprotein (HDL) fraction has been found to be positively associated with ethanol consumption9 and negatively associated with both cigarette smoking14 and adiposity.3,6,14-17 In longitudinal data from the Multiple Risk Factor Intervention Trial,18 HDL-cholesterol level also tended to vary in the same direction with changes in reported alcohol intake and in the opposite direction with changes in cigarette use and body mass.

The purpose of the analysis presented here was to determine whether serum lipid levels were more highly correlated with skinfold thickness at the triceps and subscapular sites than with body weight adjusted for height; to determine the extent to which cross-sectional age trends in these indices of adiposity accounted for age trends in lipid levels; to evaluate the use of alcohol, tobacco, and beverages containing caffeine — three common and related behaviors — as sources of variation in lipid levels; and to determine whether variation in lipoprotein cholesterol levels associated with any variable within the set considered was independent of associated variation in very low density lipoprotein (VLDL) triglyceride.
Methods

Study Subjects

The study subjects were participants in a survey of lipoprotein concentration, composition, and properties, conducted between November, 1972 and August, 1976, among employees of the Southern Pacific Railroad working in the San Francisco Bay and Sacramento-Roseville areas of California. This population numbered approximately 9000 men and 1400 women. Of the 4719 men and 1285 women randomly selected for testing, 3678 men (78%) and 969 women (75%) gave blood samples.

Because this industrial population was multi-racial, a factor that could affect the variables under study, nonwhites (who comprised 20% of the male sample and 30% of the female sample) were excluded from the analyses. Women taking exogenous sex steroids, as well as a small number who were pregnant, were also excluded; they constituted 36% of the white female sample. Persons fasting less than 8 hours or aged 65 years and over were also excluded.

These exclusions left 2865 men and 419 women, of whom 663 men and 251 women were randomly selected for quantification of lipoprotein lipid levels. Only persons measured for height, weight, and skinfold thickness over the triceps and below the scapula were included in the analysis of variation in serum lipid levels. These persons numbered 2175 men and 280 women. Not all persons in the lipoprotein lipid subsample were measured on all four anthropometric variables; but to preserve the maximum number available, cases with incomplete anthropometric data were used in analyses not involving all four of these variables.

Lipid Measurements

The serum concentrations of total cholesterol and triglyceride were determined on duplicate extracts using standard AutoAnalyzer II procedure. Internal quality control procedures and external surveillance by the Lipid Standardization Laboratory at the Center for Disease Control, Atlanta, Georgia, resulted in stability of the population measurements over the course of the survey. The coefficient of variation in the mean concentration from 5.0% at levels below 50 mg/dl to 2.4% at levels between 200 and 300 mg/dl. Through a calibrator pool supplied by the Lipid Standardization Laboratory, the cholesterol readings were adjusted to Abell-Kendall equivalent units.

The three major lipoprotein classes were separated by preparative ultracentrifugation as described elsewhere and analyzed for component cholesterol and triglyceride. The loss of lipid material in centrifugation and/or tube-slicing was evaluated against a background of measurement variation. Evidence of a loss of lipid exceeding 5% and/or larger than average measurement error was observed in 2% of cases. These cases were discarded. In the remaining cases, assuming equal losses from the three fractions, we adjusted the lipoprotein measurements for each lipid proportionately so that their sum equaled the lipid's concentration in the whole serum.

Anthropometry

Height, weight, and thickness of the skinfolds over the triceps and below the scapula were measured when the blood specimen was collected. With the subject in stocking feet, height was measured to the nearest 0.5 cm with a free-standing rod having a sliding horizontal bar. Weight was measured to the nearest pound with an Accu-Weigh portable scale (Seca, West Germany). Men were stripped to the waist and wearing trousers with pockets emptied, while women wore a disposable paper gown over undergarments. An index of body mass, defined as weight in kilograms divided by the square of height in meters, was computed by machine. This index of body mass was independent of height in all age groups of both sexes.

The triceps and subscapular skinfolds were measured on the right side to the nearest millimeter with a Lange caliper (Cambridge Scientific Industries, Cambridge, Maryland) following established procedures. Two measurements were taken on each skinfold because their reproducibility is known to be lower than that of the other variables measured. Both determinations were recorded and the mean value computed by machine.

Not all members of the field staff were trained to make skinfold measurements. Height and weight were not measured on persons working at isolated sites. Consequently, anthropometric data were missing or incomplete for one-fourth of the men and one-third of the women. There were no essential differences, however, between persons with and without complete anthropometric data with respect to lipid levels or the use of ethanol, tobacco, and beverages containing caffeine.

Behavioral Variables

Data on the use of alcohol, tobacco, and beverages containing caffeine were obtained by a self-administered questionnaire which, when completed, was reviewed by an interviewer. Persons...
were asked to report the quantity of beer, wine, and hard liquor—each separately—consumed in an average week. This information was recorded in ounces. If the quantity reported was in nonstandard units such as number of drinks or glasses, rather than a standard quantity (e.g., \( \frac{1}{2} \) pint, fifth, 6-pack), the interviewer attempted to assess how much liquor was in a cocktail, how much wine in a glass, etc. With the ethanol content assumed to be 3.2% for beer, 12% for wine, and 40% for hard liquor, an estimate of the quantity of ethanol ingested in an average week was computed by machine.

Tobacco was recorded as the separate number of cigarettes, cigars, and bowls of pipe tobacco smoked daily and the number of tins of snuff or chewing tobacco consumed weekly.

Consumption of beverages containing caffeine was recorded as the number of daily cups of coffee and/or tea, excluding decaffeinated products, and the daily number of 12-ounce cola drinks, sweetened either artificially or with sugar, habitually consumed.

The time and date the subject last ingested anything of caloric value were recorded together with the time and date of the venipuncture. The length of the fast was then computed by machine. An evaluation of serum triglyceride levels in relation to the length of the fast indicated that a fast of 8 hours was sufficient to exclude postprandial lipemia. The 2% of tested persons who had eaten within the preceding 8 hours were excluded from the analyses.

Statistical Methods

The data were analyzed by multiple linear regression within sex and age group. If the assumption of parallel regressions among age groups was tenable, pooled estimates of the sex-specific regression coefficients were computed. Terms for curvature were included in the regression model to evaluate the assumption of linear association. Curvilinear relationships were rectified when possible by transformation of scale. Terms for interaction were also included to test the assumption of additivity in the effects of the independent variables, but none was statistically significant.

To correct for the marked positive skew in the distribution and to stabilize the variance of the residuals about the regression, the triglyceride values for both the whole serum and VLDL fraction were transformed into logarithms. The distribution of the values in logarithmic scale was fairly symmetrical, and the antilog of the mean was within 2 or 3 mg/dl of the median observed value for each age group of both sexes. VLDL-cholesterol was analyzed as a power function of VLDL-triglyceride but with both lipids in logarithmic scale. The curvilinear inverse relationship between HDL-cholesterol and VLDL-triglyceride was also rectified with VLDL-triglyceride in logarithmic scale.

Results

Serum Lipid Levels

This section deals with variation among persons in the concentrations of cholesterol and triglyceride in the whole serum. The mean and standard deviation of the distributions of serum cholesterol and, in logarithmic scale, serum triglyceride among the study group are shown in table 1 by sex and age.

Compared with the Abell-Kendall-adjusted serum cholesterol levels of the national sample tested in the Health and Nutrition Examination Survey (HANES) of 1971–1974, the levels of

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**Table 1. Mean (\( \bar{x} \)) and Standard Deviation (sd) of the Distribution of Serum Cholesterol and Log10 Serum Triglyceride by Sex and Age**

<table>
<thead>
<tr>
<th>Sex and age (yrs)</th>
<th>No. of persons</th>
<th>Serum cholesterol (mg/dl)</th>
<th>Log10 serum triglyceride (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>( \bar{x} )</td>
<td>sd</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20–24</td>
<td>72</td>
<td>182.7</td>
<td>31.1</td>
</tr>
<tr>
<td>25–34</td>
<td>479</td>
<td>200.2</td>
<td>40.2</td>
</tr>
<tr>
<td>35–44</td>
<td>48</td>
<td>220.1</td>
<td>43.9</td>
</tr>
<tr>
<td>45–54</td>
<td>90</td>
<td>230.4</td>
<td>41.1</td>
</tr>
<tr>
<td>55–64</td>
<td>69</td>
<td>232.7</td>
<td>40.4</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20–24</td>
<td>13</td>
<td>190.2</td>
<td>39.9</td>
</tr>
<tr>
<td>25–34</td>
<td>60</td>
<td>189.4</td>
<td>36.7</td>
</tr>
<tr>
<td>35–44</td>
<td>90</td>
<td>204.3</td>
<td>38.2</td>
</tr>
<tr>
<td>45–54</td>
<td>93</td>
<td>228.3</td>
<td>33.0</td>
</tr>
<tr>
<td>55–64</td>
<td>69</td>
<td>241.6</td>
<td>37.1</td>
</tr>
</tbody>
</table>
Southern Pacific white women were consistently lower at all ages above 24 years, but the mean differences were small, ranging from 2 to 4 mg/dl. The mean levels of Southern Pacific white men were essentially identical to those of the national sample at ages 25-34 years and 35-44 years, but 2 to 3 mg/dl higher at ages 45-54 and 55-64 years.

Compared with white women not taking exogenous sex hormones tested in the Lipid Research Clinic (LRC) Prevalence Study, the observed triglyceride values of the Southern Pacific study group were generally 10 mg/dl lower, even though the LRC measurements were made on plasma that yields values approximately 3% lower than those obtained on serum. The observed age-specific serum levels of Southern Pacific men, on the other hand, were comparable to the plasma levels of LRC white men, except at ages 25-34 years where the values for Southern Pacific men averaged 10 mg/dl lower.

Indices of Adiposity

The means and standard deviations of the distributions of triceps skinfold thickness, subscapular skinfold thickness, and body mass index are shown in table 2 by sex and age. Women tended to have thicker skinfolds, particularly over the triceps, than men; but men were generally heavier for height than were women. The mean body mass index of men increased with age to a maximum at ages 35-44 years, leveled off, and then decreased slightly at ages 55-64 years. Their mean subscapular skinfold continued to increase until ages 45-54 years, after which it decreased slightly. Age variation among men in the thickness of the triceps skinfold was less marked, but decreased steadily with age after ages 35-44 years. The ratio of the subscapular skinfold to the triceps skinfold, however, increased monotonically with age from a mean of 1.21 at ages 20-24 years to 1.63 at ages 55-64 years. In women, all three indices of adiposity increased monotonically with age. The ratio of the subscapular skinfold to the triceps skinfold, which in women was generally thicker than the subscapular skinfold, varied little with age.

Body mass index was correlated with both skinfolds, but more so with the subscapular than with the triceps skinfold ($r = 0.71$ vs $0.52$ in men and $0.74$ vs $0.62$ in women). The correlation between the two skinfolds was greater in women ($r = 0.74$) than in men ($r = 0.60$).

Table 3 gives the product moment coefficients of correlation between the indices of adiposity and serum lipid levels by sex and age. The subscapular skinfold was more highly correlated with both serum lipids than was the triceps skinfold. The sum of the two skinfolds showed a lower order of correlation with serum lipid level than was shown by the subscapular skinfold alone. The coefficients for body mass index were of the same order of magnitude as those for the subscapular skinfold. All indices of adiposity were more highly correlated with triglyceride than with cholesterol.

The slope of the regression of serum cholesterol level on each index of adiposity decreased with age in both sexes. There was no apparent age trend in the slope of the regression of the logarithm of VLDL-triglyceride level on either subscapular skinfold thickness or body mass index in either sex; but the variation in the age-specific coefficients for the subscapular skinfold among men was statistically significant, due to a larger than average coefficient at ages 35-44 years.

Table 2. Mean ($\bar{x}$) and Standard Deviation (sd) of the Distribution of Skinfold Thickness and Body Mass Index by Sex and Age

<table>
<thead>
<tr>
<th>Sex and age (yrs)</th>
<th>Triceps skinfold (mm)</th>
<th>Subscapular skinfold (mm)</th>
<th>Body mass index</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\bar{x}$</td>
<td>sd</td>
<td>$\bar{x}$</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-24</td>
<td>13.0</td>
<td>6.0</td>
<td>15.6</td>
</tr>
<tr>
<td>25-34</td>
<td>12.4</td>
<td>5.3</td>
<td>17.2</td>
</tr>
<tr>
<td>35-44</td>
<td>13.4</td>
<td>5.6</td>
<td>19.5</td>
</tr>
<tr>
<td>45-54</td>
<td>12.8</td>
<td>5.0</td>
<td>19.9</td>
</tr>
<tr>
<td>55-64</td>
<td>12.1</td>
<td>4.8</td>
<td>19.6</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-24</td>
<td>16.3</td>
<td>11.0</td>
<td>16.6</td>
</tr>
<tr>
<td>25-34</td>
<td>17.8</td>
<td>5.8</td>
<td>16.2</td>
</tr>
<tr>
<td>35-44</td>
<td>22.3</td>
<td>9.4</td>
<td>21.0</td>
</tr>
<tr>
<td>45-54</td>
<td>22.7</td>
<td>7.3</td>
<td>21.7</td>
</tr>
<tr>
<td>55-64</td>
<td>23.0</td>
<td>6.2</td>
<td>21.9</td>
</tr>
</tbody>
</table>
### Table 3. Product Moment Coefficients of Correlation Between Serum Lipid Level and Indices of Adiposity

<table>
<thead>
<tr>
<th>Sex and age (yrs)</th>
<th>Index of adiposity</th>
<th>Triceps skinfold</th>
<th>Subscapular skinfold</th>
<th>Sum of skinfolds</th>
<th>Body mass index</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum cholesterol</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-24</td>
<td></td>
<td>0.159</td>
<td>0.292</td>
<td>0.249</td>
<td>0.302</td>
</tr>
<tr>
<td>25-34</td>
<td></td>
<td>0.082</td>
<td>0.210</td>
<td>0.173</td>
<td>0.228</td>
</tr>
<tr>
<td>35-44</td>
<td></td>
<td>0.093</td>
<td>0.174</td>
<td>0.157</td>
<td>0.139</td>
</tr>
<tr>
<td>45-54</td>
<td></td>
<td>0.076</td>
<td>0.095</td>
<td>0.100</td>
<td>0.150</td>
</tr>
<tr>
<td>55-64</td>
<td></td>
<td>0.064</td>
<td>0.087</td>
<td>0.087</td>
<td>0.126</td>
</tr>
<tr>
<td>Log10 serum triglyceride</td>
<td></td>
<td>0.237</td>
<td>0.436</td>
<td>0.371</td>
<td>0.425</td>
</tr>
<tr>
<td>20-24</td>
<td></td>
<td>0.114</td>
<td>0.300</td>
<td>0.246</td>
<td>0.300</td>
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<tr>
<td>25-34</td>
<td></td>
<td>0.247</td>
<td>0.435</td>
<td>0.397</td>
<td>0.415</td>
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<tr>
<td>35-44</td>
<td></td>
<td>0.125</td>
<td>0.270</td>
<td>0.242</td>
<td>0.332</td>
</tr>
<tr>
<td>55-64</td>
<td></td>
<td>0.076</td>
<td>0.243</td>
<td>0.197</td>
<td>0.278</td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum cholesterol</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-24</td>
<td></td>
<td>0.076</td>
<td>0.139</td>
<td>0.112</td>
<td>0.186</td>
</tr>
<tr>
<td>25-34</td>
<td></td>
<td>0.163</td>
<td>0.164</td>
<td>0.176</td>
<td>0.166</td>
</tr>
<tr>
<td>35-44</td>
<td></td>
<td>0.114</td>
<td>0.207</td>
<td>0.172</td>
<td>0.171</td>
</tr>
<tr>
<td>45-54</td>
<td></td>
<td>-0.050</td>
<td>0.044</td>
<td>0.004</td>
<td>0.093</td>
</tr>
<tr>
<td>55-64</td>
<td></td>
<td>-0.042</td>
<td>-0.036</td>
<td>-0.044</td>
<td>-0.077</td>
</tr>
<tr>
<td>Log10 serum triglyceride</td>
<td></td>
<td>0.184</td>
<td>0.234</td>
<td>0.214</td>
<td>0.290</td>
</tr>
<tr>
<td>20-24</td>
<td></td>
<td>0.161</td>
<td>0.400</td>
<td>0.308</td>
<td>0.343</td>
</tr>
<tr>
<td>25-34</td>
<td></td>
<td>-0.067</td>
<td>0.257</td>
<td>0.105</td>
<td>0.258</td>
</tr>
<tr>
<td>35-44</td>
<td></td>
<td>0.085</td>
<td>0.248</td>
<td>0.192</td>
<td>0.364</td>
</tr>
<tr>
<td>55-64</td>
<td></td>
<td>-0.041</td>
<td>0.144</td>
<td>0.075</td>
<td>0.190</td>
</tr>
</tbody>
</table>

To evaluate further the combined power of both skinfold sites to predict serum triglyceride level, the triceps skinfold was entered as a separate term in a multiple regression equation with the subscapular skinfold. Although not generally significant within an age group, the partial coefficient for the triceps skinfold was negative in sign in all age groups of both sexes. In women, the assumption of homogeneity in the regression plane among age groups was tenable (F = 1.01) and the inclusion of the triceps skinfold significantly reduced the residual sum of squares (F = 11.99; p < 0.001) in the pooled data. In men, however, the assumption of parallelism was not tenable, due again to the larger than average coefficient for the subscapular skinfold at ages 35–44 years.

When adjusted for body mass index, the variation in serum triglyceride level among age groups was reduced, but remained statistically very highly significant (p < 0.0001) in both sexes.

### Alcohol, Tobacco, and Beverages Containing Caffeine

Table 4 describes the study group with respect to the weekly consumption of ethanol, number of cigarettes smoked daily by cigarette users, and daily consumption of beverages containing caffeine. Abstainers from alcohol included 26% of the men and 28% of the women; an additional 2% to 3% were infrequent or sporadic users of alcohol whose weekly consumption was typically zero. Cigarette smokers included 40% of the men and 38% of the women; another 7% of the men used tobacco in forms other than cigarettes.

Smokers of both sexes drank an average of 1½ to 2 more cups of coffee and/or tea per day than nonsmokers. The quantity of ethanol ingested weekly was also greater among smokers than nonsmokers, the mean difference being 1.9 oz for men and 1.5 oz for women. The quantities of ethanol and cigarettes consumed by persons who both smoked cigarettes and used ethanol tended to covary (r = 0.20) in both sexes.

Body mass index covaried with cola consumption in all age groups of both sexes (r = 0.12 in men and 0.27 in women) and tended to vary inversely with ethanol consumption, cigarette smoking, and coffee-tea drinking, but not significantly so.

Table 5 gives the sex-specific age-adjusted partial coefficients of the regression of the logarithm of serum triglyceride level on body mass index and the use of ethanol, tobacco in specific forms, and beverages containing caffeine.
Table 4. Percentage Distribution of Use of Ethanol, Cigarettes, and Caffeine Drinks

<table>
<thead>
<tr>
<th>Ounces of ethanol weekly</th>
<th>Men (%)</th>
<th>Women (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>none</td>
<td>28.8</td>
<td>31.3</td>
</tr>
<tr>
<td>0.1–1.99</td>
<td>14.6</td>
<td>24.6</td>
</tr>
<tr>
<td>2.0–3.99</td>
<td>16.4</td>
<td>16.7</td>
</tr>
<tr>
<td>4.0–5.99</td>
<td>10.7</td>
<td>9.6</td>
</tr>
<tr>
<td>6.0–7.99</td>
<td>8.5</td>
<td>6.0</td>
</tr>
<tr>
<td>8.0–9.99</td>
<td>5.8</td>
<td>4.6</td>
</tr>
<tr>
<td>10.0–14.99</td>
<td>6.1</td>
<td>5.0</td>
</tr>
<tr>
<td>15.0–19.99</td>
<td>3.6</td>
<td>0.4</td>
</tr>
<tr>
<td>≥20.00</td>
<td>3.6</td>
<td>1.8</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Cigarettes smoked daily</th>
<th>Men (%)</th>
<th>Women (%)</th>
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<tbody>
<tr>
<td>1–10</td>
<td>11.4</td>
<td>23.4</td>
</tr>
<tr>
<td>11–20</td>
<td>38.2</td>
<td>44.9</td>
</tr>
<tr>
<td>21–30</td>
<td>26.1</td>
<td>21.5</td>
</tr>
<tr>
<td>31–40</td>
<td>20.1</td>
<td>9.3</td>
</tr>
<tr>
<td>≥41</td>
<td>4.1</td>
<td>0.9</td>
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<table>
<thead>
<tr>
<th>Daily cups of coffee-tea</th>
<th>Men (%)</th>
<th>Women (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>none</td>
<td>11.6</td>
<td>8.2</td>
</tr>
<tr>
<td>1–2</td>
<td>20.1</td>
<td>26.0</td>
</tr>
<tr>
<td>3–4</td>
<td>29.4</td>
<td>31.7</td>
</tr>
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<td>5–6</td>
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<td>25.3</td>
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<tr>
<td>7–8</td>
<td>9.3</td>
<td>4.6</td>
</tr>
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<td>9–10</td>
<td>5.8</td>
<td>2.8</td>
</tr>
<tr>
<td>≥11</td>
<td>2.5</td>
<td>1.4</td>
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</tbody>
</table>

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<thead>
<tr>
<th>Daily cola drinks</th>
<th>Men (%)</th>
<th>Women (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>none</td>
<td>75.1</td>
<td>69.7</td>
</tr>
<tr>
<td>1</td>
<td>16.8</td>
<td>21.4</td>
</tr>
<tr>
<td>2</td>
<td>5.9</td>
<td>7.1</td>
</tr>
<tr>
<td>3</td>
<td>1.3</td>
<td>1.1</td>
</tr>
<tr>
<td>≥4</td>
<td>0.9</td>
<td>0.7</td>
</tr>
</tbody>
</table>

In both sexes, the serum triglyceride level increased with the number of cigarettes smoked daily and decreased with the number of cups of coffee-tea taken daily. The variation associated with both variables tended to be more marked in women than in men, but the difference in the sex-specific coefficients was statistically significant only for cigarettes (t = 3.4; p < 0.001). Although not statistically significant, the coefficients for other forms of tobacco among men were also positive in sign. The female coefficient for cola drinks was negative in sign but not statistically significant. In men, the coefficient was positive rather than negative, and statistically significant. Weekly ethanol consumption was unrelated to serum triglyceride level.

Of the variables considered, body mass index was the most important source of variation in serum triglyceride level among men. Cigarette smoking was a somewhat more important source of variation in triglyceride level among women than was body mass index.

Table 4 gives the age-adjusted partial coefficients of the regression of serum cholesterol level on the same set of variables within broad age ranges where the coefficients were relatively homogeneous. The increase in serum cholesterol level with increasing body mass index was statistically significant in all age groups of men, but less marked at older ages. The increase among women was smaller and not statistically significant with these sample sizes. The coefficients for cigarette usage were positive at all ages but statistically significant only for younger men and older women. No consistent association with either ethanol or caffeine beverages was evident, although the coefficient for ethanol was statistically significant for older men. Because, as will be shown in the following section, cholesterol levels within lipoprotein classes often had differential associations with the same variables, analysis of the level in the whole serum may be confounded.

Lipoprotein Lipid Levels

This section deals with the level of triglyceride in the VLDL fraction and the levels of cholesterol

Table 5. Partial Coefficients of the Regression of the Logarithm of Serum Triglyceride Level (mg/dl) on the Behavioral Variables and Body Mass Index*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethanol: oz/week</td>
<td>-0.0004</td>
<td>0.0002</td>
</tr>
<tr>
<td>Cigarettes: ½ pack/day</td>
<td>0.0177</td>
<td>0.0535</td>
</tr>
<tr>
<td>Cigar: no./day</td>
<td>0.0085</td>
<td>-</td>
</tr>
<tr>
<td>Pipe: bowl/day</td>
<td>0.0018</td>
<td>-</td>
</tr>
<tr>
<td>Snuff: tin/week</td>
<td>0.0018</td>
<td>-</td>
</tr>
<tr>
<td>Coffee-tea: cup/day</td>
<td>-0.0035</td>
<td>-0.0109</td>
</tr>
<tr>
<td>Cola drinks: 12 oz/day</td>
<td>0.0137</td>
<td>-0.0178</td>
</tr>
<tr>
<td>Body mass index: unit</td>
<td>0.2247</td>
<td>0.0124</td>
</tr>
</tbody>
</table>

*Adjusted for age within sex.
†b = slope; se = standard error of b.
‡p ≤ 0.001.
§p ≤ 0.05.
||p ≤ 0.01.
Table 6. Partial Coefficients of the Regression of Serum Cholesterol Level (mg/dl) on the Behavioral Variables and Body Mass Index

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men (20-34 yrs)</th>
<th>Men (35-64 yrs)</th>
<th>Women (20-44 yrs)</th>
<th>Women (45-64 yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(b) SE(f)</td>
<td>(b) SE</td>
<td>(b) SE</td>
<td>(b) SE</td>
</tr>
<tr>
<td>Ethanol: oz/week</td>
<td>0.22 0.24</td>
<td>0.55 0.17(\d)</td>
<td>-1.36 0.80</td>
<td>0.33 0.67</td>
</tr>
<tr>
<td>Cigarettes: % pack/day</td>
<td>3.45 1.34(\d)</td>
<td>0.49 0.74</td>
<td>3.83 3.08</td>
<td>7.24 2.65(\d)</td>
</tr>
<tr>
<td>Cigar: no./day</td>
<td>— —</td>
<td>-0.58 1.12</td>
<td>— —</td>
<td>— —</td>
</tr>
<tr>
<td>Pipe: bowl/day</td>
<td>— —</td>
<td>-0.74 0.47</td>
<td>— —</td>
<td>— —</td>
</tr>
<tr>
<td>Snuff: tin/week</td>
<td>— —</td>
<td>0.60 2.88</td>
<td>— —</td>
<td>— —</td>
</tr>
<tr>
<td>Coffee-tea: cup/day</td>
<td>0.68 0.56</td>
<td>0.71 0.36(\d)</td>
<td>-0.33 1.34</td>
<td>-1.41 1.11</td>
</tr>
<tr>
<td>Cola drinks: 12 oz/day</td>
<td>-2.24 1.95</td>
<td>1.16 1.31</td>
<td>4.48 4.04</td>
<td>-3.93 5.40</td>
</tr>
<tr>
<td>Body mass index: unit</td>
<td>2.70 0.47(\d)</td>
<td>1.58 0.29(\d)</td>
<td>1.12 0.74</td>
<td>0.22 0.64</td>
</tr>
</tbody>
</table>

*Adjusted for age within sex.
\(\d\) = slope; SE = standard error of \(b\).
\(p < 0.001\).
\(\d\) = standard error of \(b\).
\(p < 0.01\).
\(\d\) = standard error of \(b\).
\(p < 0.05\).

in the VLDL, LDL, and HDL fractions. Table 7 gives the sex- and age-specific mean levels of the study group.

VLDL Triglyceride

Among men, 60% of the triglyceride in the whole serum was found, on the average, in the VLDL fraction. Among women, with their generally lower serum levels, the proportion carried by VLDL averaged 50%. The product moment coefficient of correlation between the levels of triglyceride in the whole serum and VLDL fraction was 0.99 in both sexes.

Results of the analysis of VLDL triglyceride in relation to the three indices of adiposity and the consumption of ethanol, tobacco, and caffeine-containing beverages were essentially the same as those obtained in the analysis of serum triglyceride. Like the level in the whole serum, the triglyceride level in the VLDL fraction was more highly correlated with the subscapular skinfold than with the triceps skinfold. Its correlation with body mass index was comparable to its correlation with the subscapular skinfold. All three indices of adiposity tended to be more highly correlated with the VLDL-triglyceride level in men than in women.

The VLDL-triglyceride level also increased with the consumption of cigarettes and decreased with the intake of coffee-tea in both sexes. The coefficient for cigarettes was again significantly larger in women than in men \((t = 3.3; p < 0.001)\). The coefficient for coffee-tea was also larger for women, but the difference in the sex-specific coefficients was not statistically significant. The coefficient for cola drinks in women was not statistically significant but was also negative in sign; but in men, the coefficient for cola drinks was again positive in sign and statistically

Table 7. Mean Lipid Levels of White Persons Randomly Selected for Separation of Lipoprotein Classes by Sex and Age

<table>
<thead>
<tr>
<th>Sex and age (yrs)</th>
<th>No. of Persons</th>
<th>Cholesterol (mg/dl)</th>
<th>Log_{10} triglyceride (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Whole serum</td>
<td>VLDL</td>
<td>LDL</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-24</td>
<td>19</td>
<td>180.0</td>
<td>12.8</td>
</tr>
<tr>
<td>25-34</td>
<td>150</td>
<td>197.0</td>
<td>17.4</td>
</tr>
<tr>
<td>35-44</td>
<td>115</td>
<td>219.0</td>
<td>23.5</td>
</tr>
<tr>
<td>45-54</td>
<td>204</td>
<td>225.9</td>
<td>24.8</td>
</tr>
<tr>
<td>55-64</td>
<td>175</td>
<td>228.6</td>
<td>23.3</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-24</td>
<td>11</td>
<td>183.5</td>
<td>9.2</td>
</tr>
<tr>
<td>25-34</td>
<td>53</td>
<td>189.6</td>
<td>8.0</td>
</tr>
<tr>
<td>35-44</td>
<td>30</td>
<td>209.2</td>
<td>13.6</td>
</tr>
<tr>
<td>45-54</td>
<td>86</td>
<td>227.6</td>
<td>12.9</td>
</tr>
<tr>
<td>55-64</td>
<td>71</td>
<td>240.7</td>
<td>18.7</td>
</tr>
</tbody>
</table>

VLDL = very low density lipoprotein; LDL = low density lipoprotein; HDL = high density lipoprotein.
significant. There was no demonstrable relationship between weekly ethanol consumption and VLDL-triglyceride level in either sex when cigarette usage was taken into account.

VLDL Cholesterol
The levels of cholesterol and triglyceride in the VLDL fraction were highly correlated, the product moment coefficient of correlation being approximately 0.90. Like VLDL triglyceride, VLDL cholesterol increased with body mass index and cigarette smoking and decreased with coffee-tea drinking. The VLDL cholesterol level was unrelated to ethanol consumption in both sexes, but increased with cola consumption in men. When adjusted for VLDL triglyceride, however, VLDL cholesterol was independent of these variables.

LDL Cholesterol
Approximately two-thirds of the cholesterol in the whole serum was found, on the average, in the LDL fraction. The product moment coefficient of correlation between the levels of cholesterol in the whole serum and LDL fraction was somewhat higher for women \((r = 0.92)\) than for men \((r = 0.85)\) in whom hypercholesterolemia from marked elevations in VLDL is more common.

The coefficients for the slope of the regression of LDL-cholesterol level on body mass index decreased with age in both sexes. The only coefficient significantly different from zero was that for men aged 25-34 years.

Table 8 gives the sex-specific age-adjusted partial coefficients of the regression of LDL-cholesterol level on the use of ethanol, cigarettes, and beverages containing caffeine. Men who used tobacco in forms other than cigarettes were excluded from the computations because their number was too small to give useful information. In both sexes, LDL cholesterol level decreased as ethanol consumption increased and increased as cigarette usage and the intake of coffee-tea increased; but the only coefficients significantly different from zero at the 0.05 level were that for ethanol in men and that for cigarettes in women.

**Table 8. Partial Coefficients of the Regression of LDL Cholesterol Level (mg/dl) on the Behavioral Variables***

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men ((n = 609))</th>
<th>Women ((n = 251))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethanol: oz/week</td>
<td>(-0.47 \pm 0.22)</td>
<td>(-0.73 \pm 0.50)</td>
</tr>
<tr>
<td>Cigarettes: (1/4) pack/day</td>
<td>1.94 \pm 1.04</td>
<td>4.91 \pm 1.73§</td>
</tr>
<tr>
<td>Coffee-tea: cup/day</td>
<td>0.97 \pm 0.52</td>
<td>0.89 \pm 0.74</td>
</tr>
<tr>
<td>Cola drinks: 12 oz/day</td>
<td>(-0.93 \pm 1.88)</td>
<td>4.06 \pm 2.67</td>
</tr>
</tbody>
</table>

*Adjusted for age within sex.
†b = slope; se = standard error of b.
‡p ≤ 0.05.
§p ≤ 0.01.

Figure 1. Age-adjusted mean level of LDL cholesterol by VLDL triglyceride level of persons aged 25-64 years. The age adjustment was made by weighting the observed age-specific mean levels of each sex by the proportionate representation of that age group in the total unselected white subsample of men and women aged 25-64 years. Persons less than 25 years were omitted because their number was too small, and their VLDL triglyceride levels too low, for adequate representation at VLDL triglyceride levels above 60 mg/dl. White men selected for quantitation of lipoprotein lipid levels were included in the computations of mean LDL cholesterol levels at VLDL triglyceride concentrations \(\geq 120\) mg/dl to increase the sample sizes at the higher concentrations, their LDL cholesterol levels being comparable to those of unselected men. LDL triglyceride levels \(\geq 120\) mg/dl were infrequent among women below 55 years of age not taking exogenous sex steroids. The sample sizes for all data points shown exceeded 45 persons except for the last 3 data points of each sex, which were 37, 15, and 14 men and 22, 21, and 7 women.
Table 9. Partial Coefficients of the Regression of HDL Cholesterol Level (mg/dl) on the Behavioral Variables*

<table>
<thead>
<tr>
<th>Sex and variable</th>
<th>Adjusted for log₁₀ VLDL triglyceride</th>
<th>Without adjustment for log₁₀ VLDL triglyceride</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b</td>
<td>se</td>
</tr>
<tr>
<td>Men (n = 609)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethanol: oz/week</td>
<td>0.613</td>
<td>0.067elijke $</td>
</tr>
<tr>
<td>Cigarettes: ½ pack/day</td>
<td>-0.710</td>
<td>0.324elijke $</td>
</tr>
<tr>
<td>Coffee-tea: cup/day</td>
<td>0.166</td>
<td>0.162elijke $</td>
</tr>
<tr>
<td>Cola drink: 12 oz/day</td>
<td>-0.548</td>
<td>0.586elijke $</td>
</tr>
<tr>
<td>Women (n = 251)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethanol: oz/week</td>
<td>0.974</td>
<td>0.187elige $</td>
</tr>
<tr>
<td>Cigarettes: ½ pack/day</td>
<td>-2.271</td>
<td>0.696elige $</td>
</tr>
<tr>
<td>Coffee-tea: cup/day</td>
<td>0.106</td>
<td>0.286elige $</td>
</tr>
<tr>
<td>Cola drink: 12 oz/day</td>
<td>-0.588</td>
<td>1.012elige $</td>
</tr>
</tbody>
</table>

*Adjusted for age within sex.

†b = slope; se = standard error of b.

$R² = proportion of variation in HDL cholesterol due to regression on statistically significant variables and, when included, VLDL triglyceride.

§p ≤ 0.001.

¶p ≤ 0.05.

¶¶p ≤ 0.01.

VLDL = very low density lipoprotein; HDL = high density lipoprotein.

The age-specific partial coefficients for body mass index, controlled for the use of ethanol, cigarettes, and caffeine-containing beverages, were little different from the simple coefficients. Inclusion of body mass index in the regression equation had essentially no effect on either the direction or strength of the association between LDL cholesterol and the other variables.

Age and VLDL triglyceride level were both important sources of cross-sectional variation in LDL cholesterol. The age-adjusted variation in LDL cholesterol associated with VLDL triglyceride level is illustrated in figure 1. LDL cholesterol generally increased as the level of VLDL triglyceride increased, up to about 100 mg/dl, and then decreased with increasing VLDL triglyceride concentration. The age-related sex-specific increase in LDL cholesterol (table 7) was largely independent of concomitant age variation in VLDL triglyceride. When its relationship with VLDL triglyceride was controlled with a second degree polynomial in the 72% of men with VLDL triglyceride below 100 mg/dl, LDL cholesterol increased curvilinearly with age, the partial coefficients of the regression being 3.64 mg/dl (se = 1.02) for age in years and -0.032 mg/dl (se = 0.012) for the square of age. Thus, the predicted increase in LDL cholesterol from 25 to 55 years of age in normotriglyceridemic men of like VLDL triglyceride level was 32 mg/dl. Among the 90% of women with VLDL triglyceride levels below 100 mg/dl, LDL cholesterol, adjusted for VLDL triglyceride, increased linearly at the rate of 1.11 mg/dl (se = 0.18) per year of age.

With adjustment for VLDL triglyceride level, LDL cholesterol level was unrelated to cigarette usage. The coefficient for the regression of LDL cholesterol on body mass index in men aged 25–34 years, however, remained statistically significant when controlled for VLDL triglyceride.

HDL Cholesterol

The proportion of the whole serum cholesterol found in the HDL fraction averaged 23% in men and 29% in women. Although the proportion typically carried by HDL was substantial, the cholesterol levels in the whole serum and HDL fraction were generally unrelated in men (r = 0.027) and only weakly related in women (r = 0.144).

Table 9 gives the sex-specific partial coefficients of the regression of HDL cholesterol level on the use of ethanol, cigarettes, and caffeine-containing beverages computed for persons who were either nonsmokers or exclusive users of cigarettes. Table 10 gives the coefficients for body mass index and the behavioral variables computed on persons measured for both height and weight, exclusive of those who used tobacco in forms other than cigarettes. In both tables, the coefficients are shown with and without adjustment for VLDL triglyceride in logarithmic scale. Because HDL cholesterol and VLDL triglyceride were inversely related (r = -0.5 with VLDL tri-
Table 10. Partial Coefficients of the Regression of HDL Cholesterol Level (mg/dl) on Body Mass Index and the Behavioral Variables*  

<table>
<thead>
<tr>
<th>Sex and variable</th>
<th>Adjusted for log_{10} VLDL triglyceride</th>
<th>Without adjustment for log_{10} VLDL triglyceride</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b</td>
<td>se†</td>
</tr>
<tr>
<td>Men (n = 518)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass index: unit</td>
<td>-0.534</td>
<td>0.131§</td>
</tr>
<tr>
<td>Ethanol: oz/week</td>
<td>0.069</td>
<td>0.348</td>
</tr>
<tr>
<td>Cigarettes: ½ pack/day</td>
<td>-0.754</td>
<td>0.518</td>
</tr>
<tr>
<td>Coffee-tea: cup/day</td>
<td>0.194</td>
<td>0.173</td>
</tr>
<tr>
<td>Cola drinks: 12 oz/day</td>
<td>-0.518</td>
<td>0.618</td>
</tr>
<tr>
<td></td>
<td>0.37</td>
<td></td>
</tr>
<tr>
<td>Women (n = 200)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass index: unit</td>
<td>-0.485</td>
<td>0.200‖</td>
</tr>
<tr>
<td>Ethanol: oz/week</td>
<td>0.748</td>
<td>0.833§</td>
</tr>
<tr>
<td>Cigarettes: ½ pack/day</td>
<td>-2.968</td>
<td>0.382</td>
</tr>
<tr>
<td>Coffee-tea: cup/day</td>
<td>0.209</td>
<td>1.272</td>
</tr>
<tr>
<td>Cola drinks: 12 oz/day</td>
<td>0.081</td>
<td>1.272</td>
</tr>
</tbody>
</table>

*Adjusted for age within sex.
†b = slope; se = standard error of b.
‡R2 = proportion of variation in HDL cholesterol due to regression on statistically significant variables and, when included, VLDL triglyceride.
§p ≤ 0.001.
‖p ≤ 0.05.
¶p ≤ 0.01.

VLDL = very low density lipoprotein; HDL = high density lipoprotein.

Glyceride in logarithmic scale), VLDL triglyceride was held constant to remove associations between HDL cholesterol and other variables possibly mediated through VLDL triglyceride. HDL cholesterol level increased in both sexes with the quantity of ethanol typically ingested weekly. The sex-specific coefficients for the slope of the regression of HDL cholesterol on ethanol consumption were essentially unaltered when adjusted for VLDL triglyceride. HDL cholesterol level decreased in both sexes as body mass index and the number of cigarettes smoked daily increased. When the increase in VLDL triglyceride level associated with body mass index and cigarette smoking was taken into account, the decrease in HDL cholesterol level associated with each variable was reduced but remained statistically significant for both sexes. The decrease associated with cigarette usage was more marked in women than in men, even with adjustment for VLDL triglyceride (t = 2.03; p < 0.05). There was no statistically significant variation in HDL cholesterol level associated with the consumption of beverages containing caffeine when the inverse relationship between HDL cholesterol and VLDL triglyceride was taken into account.

Ethanol consumption, cigarette smoking, body mass index, and VLDL triglyceride level together accounted for a substantial proportion of the variation in HDL cholesterol level among persons (tables 9 and 10). Of these variables, VLDL triglyceride was the largest source of variation.

Discussion

The triglyceride level in the whole serum and VLDL fraction was related to all three indices of adiposity used in this study. Of the two skinfold sites measured, the subscapular site was the more predictive of triglyceride level in all age groups of both sexes. When the triceps and subscapular skinfolds were combined as separate terms in a multiple regression equation, the partial coefficient for the triceps skinfold was negative in sign in all age groups of both sexes. This result suggests that, like earlier findings by Albrink and Meigs,25 centripetal fat distribution is a correlate of hypertriglyceridemia. The association between centripetal fat distribution and triglyceride level was more clearly displayed among women, who, in contrast to men, typically have thicker skinfolds over the triceps than below the scapula.

Body mass index was as predictive of triglyceride level as was the subscapular skinfold in men and the weighted combination of both skinfolds in women. That the predictive power of the skinfolds was not greater may indicate that the precision of the measurements was less than optimal. However, height-weight ratios have been found to be as accurate as the subscapular skinfold in predicting total body fat in adults.26 27

When adjusted for age differences in body mass index, the variation among age groups in triglyceride level remained statistically very
highly significant in both sexes. Only a small part of the observed age variation in triglyceride level was accounted for by concomitant age variation in body mass index.

The triglyceride level in the whole serum and VLDL fraction increased with the number of cigarettes smoked daily, more markedly so in women than in men, and decreased somewhat as the consumption of coffee-tea increased. Although not statistically significant, the partial coefficient for the consumption of cola drinks among women was also negative in sign. Among men, however, the partial coefficient for cola drinks was positive in sign and statistically significant. If caffeine does in fact tend to decrease triglyceride level, this contrary result for cola drinks among men may reflect an opposing effect from carbohydrate consumption. If it is assumed that women are much more likely than men to use artificially sweetened cola drinks, then the caffeine might be displayed in women but not in men.

Clinical opinion holds that abstinence from alcohol is fundamental to the control of hypertriglyceridemia, and positive associations between the use of alcohol and triglyceride level have been observed in a number of population samples. Our data, however, showed no relationship between ethanol consumption and triglyceride level when their mutual covariance with cigarette smoking was taken into account. The positive associations found in other population data were not usually controlled for cigarette usage. Although sustained ingestion of alcohol may exacerbate hypertriglyceridemia stemming from certain causes, it may have little or no effect on fasting triglyceride level in the average person.

VLDL cholesterol, like VLDL triglyceride, increased with body mass index and cigarette smoking and tended to decrease as the consumption of coffee-tea increased. In men, VLDL cholesterol level also increased with the consumption of cola drinks. When adjusted for VLDL triglyceride level, VLDL cholesterol was independent of these variables. Thus, these data showed no evidence of an accumulation in the VLDL fraction of remnant particles, which are relatively rich in cholesterol, in association with any of the variables considered.

The ambient level of VLDL triglyceride appears to be highly correlated with the rate of VLDL triglyceride synthesis. LDL is largely the product of VLDL catabolism. To the extent that increased hepatic secretion of VLDL triglyceride involves an increase in the number of VLDL particles, it must be presumed that more particles of LDL will be formed. That the levels of VLDL triglyceride and LDL cholesterol covaried in normotriglyceridemic members of our population is consistent with this concept. The covariance of these two lipoprotein lipids did not apply as the concentration of VLDL triglyceride increased above approximately 100 mg/dl; rather, the relationship became inverse. Whereas almost all VLDL particles are thought to be converted to LDL particles in normotriglyceridemic persons, this does not appear to be the case in persons with hypertriglyceridemia. A progressive fall in the fraction of VLDL particles converted to LDL as triglyceride levels increase above a critical level could explain the inverse relationship that was observed at higher concentrations. Our data raise the possibility that the coupling of VLDL secretion to LDL production is incomplete in most persons with even mild hypertriglyceridemia. The quasiparabolic rather than linear relationship between LDL cholesterol and VLDL triglyceride levels found in our data may explain why Grundy and his associates found no correlation between the level of LDL cholesterol and the rate of VLDL triglyceride transport in a group of 59 persons, most of whose VLDL triglyceride levels exceeded 100 mg/dl.

Among normotriglyceridemic persons, the LDL cholesterol level, adjusted for VLDL triglyceride, increased with age in both sexes. Thus, the sex-specific age trends in LDL cholesterol level observed in this and other North American populations were not accounted for by concomitant age variation in VLDL triglyceride level. With the exception of men aged 25–34 years, the small increase in LDL cholesterol level with increased body mass index appeared to reflect the related increase in VLDL triglyceride level. The trend toward higher LDL cholesterol levels with cigarette consumption also appeared to be due to the higher VLDL triglyceride levels of cigarette smokers.

The level of cholesterol in the HDL fraction increased with the quantity of ethanol habitually ingested weekly and decreased with increasing body mass index and the number of cigarettes smoked daily. Only part of the decrease associated with body mass index and cigarette smoking was accounted for by concomitant increase in VLDL triglyceride. These results suggest that the concentration of HDL particles, as well as their cholesterol content, may decrease with increasing adiposity and cigarette usage. The magnitude of the cross-sectional variation in HDL cholesterol associated with adiposity, ethanol consumption, cigarette smoking, and VLDL triglyceride level indicates that all of these variables should be taken into account in assessing other environmental and genetic sources of variation. These data also suggest that substantial increases in HDL cholesterol level might be realized by persons who reduce body fatness, stop smoking, and lower their VLDL triglyceride levels through dietary modifications.
Acknowledgments

This research was made possible by the cooperation of the Southern Pacific Transportation Company and its employees.

References

13. Plaque smoking, serum-cholesterol, blood-pressure, and body fatness; observations in Finland. Lancet 1959;1:283-292

Index Terms: adiposity • cholesterol • cigarettes • ethanol • lipoproteins • triglycerides
Levels and interrelationships of serum and lipoprotein cholesterol and triglycerides. Association with adiposity and the consumption of ethanol, tobacco, and beverages containing caffeine.

N R Phillips, R J Havel and J P Kane