Demographic, Behavioral, Biochemical, and Dietary Correlates of Plasma Triglycerides

Lipid Research Clinics Program Prevalence Study

Linda D. Cowan, Timothy Wilcosky, Michael H. Criqui, Elizabeth Barrett-Connor, C.M. Suchindran, Robert Wallace, Peter Laskarzewski, and Carolyn Walden

Few studies have simultaneously examined the relationship of triglyceride levels with a wide variety of potential covariates. Thus, the present study was designed to assess in a large, free-living population the association of fasting plasma triglyceride values with selected demographic, behavioral, biochemical, and dietary measures. These analyses were done using data obtained from 5188 white men and women aged 20 to 69 years who participated in the Lipid Research Clinics Program Prevalence Study. Of the eight nondietary factors examined, age, Quetelet Index, fasting plasma glucose, and cigarette smoking were strongly, positively associated \((p < 0.0001)\) with triglycerides in men and in women not using gonadal hormones. Among women using oral contraceptives or estrogens, only Quetelet Index \((p < 0.01)\) and cigarette smoking \((p = 0.01)\) were significantly related to triglyceride values. Physical activity was inversely associated \((p < 0.0001)\) and use of diuretic medications was positively related \((p < 0.01)\) to triglycerides only in men. Results of analyses of triglycerides and six selected dietary measures varied by age, sex, and hormone-use subgroups. Although none of the dietary variables showed consistent associations with triglycerides across all of the subgroups, triglycerides tended to be inversely associated with total calories per kilogram of body weight and the percentage of calories as dietary fat. (Arteriosclerosis 5:466–480, September/October 1985)

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High triglyceride (TG) levels are generally associated with an increased risk of coronary heart disease in univariate analyses or after statistical adjustment for total cholesterol. However, whether the association between TG and coronary risk is independent of other known risk factors remains a subject of controversy. Some studies report TG to be a significant predictor of death from ischemic heart disease in men after adjustment for age, blood pressure, total cholesterol, body mass, and cigarette smoking, while others do not. In particular, the importance of high density lipoprotein cholesterol (HDL-C) as a covariate has been raised. Triglycerides have been more consistently linked with risk of coronary disease in women than in men.

Although information concerning the relationship of TG with other factors associated with risk of coronary disease would help to clarify the nature of the association between TG and heart disease, such interrelationships have not been well described in large, free-living populations. Increased TG levels have been observed in association with the use of a
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number of prescription medications, including digitalis and propranolol, diuretics, and oral contraceptives and estrogens. Triglycerides have also been positively associated with alcohol consumption, cigarette smoking, obesity, and diabetes. Most previous investigations have focused on the relationship of TG levels with only a few variables. Few studies have considered a wide variety of suspected correlates of TG both individually and simultaneously in the same population. The purpose of the present study was to examine in adult men and women the associations of TG values with selected demographic, behavioral, biochemical, and dietary measures and to identify those factors which were independently related to TG levels.

Methods

Design of the Prevalence Study

Participants were examined as part of the Lipid Research Clinics (LRC) Prevalence Study conducted between 1972 and 1976 by 10 North American clinics. Populations that would provide information from diverse geographical, occupational, socioeconomic, and age groups were chosen. Each LRC selected a defined population by sampling households, schools, various businesses, and one prepaid medical practice. The methodology used in the Prevalence Study has been reported previously, but is briefly described here.

A two-stage screening procedure was used. At Visit 1, participants were asked to answer a brief questionnaire and had their fasting plasma cholesterol and TG measured. A total of 81,926 persons were invited to the Visit 1 screening, and 60,502 (74%) participated.

Without knowledge of the results of the lipid measurements, each LRC invited a 15% random sample of its Visit 1 population for a second, more extensive evaluation. The entire range of lipid and lipoprotein values, including hyperlipidemic levels, was represented in the random sample. Among the remaining Visit 1 participants, persons with elevated lipids or who were taking lipid-lowering medications at the first screening were also invited to Visit 2, but are not considered in this report. Approximately 85% of the randomly selected participants (7733 of 9107) completed the Visit 2 examination. This second screening was conducted at each LRC clinic by use of a standardized protocol and included resting and stress electrocardiography; anthropometric measures; a detailed questionnaire concerning cardiovascular symptoms and morbidity, personal habits, and use of prescription medications within the 2 weeks before the interview; a 24-hour dietary recall; and fasting determinations of lipids, lipoprotein cholesterol, and eight additional clinical chemistries.

Lipid and Lipoprotein Determinations

Venous blood samples for the lipid and lipoprotein determinations were drawn after at least a 12-hour fast with participants in a sitting position. A tourniquet was used but was released before sampling to avoid artifactual increases in the concentrations of plasma lipids. Total plasma cholesterol, TG, and HDL-C, low density lipoprotein cholesterol (LDL-C), and very low density lipoprotein cholesterol (VLDL-C) were measured at each clinic by use of standardized LRC techniques. Plasma TG was analyzed fluorometrically by use of the Technicon Auto-Analyzer I or II, adapted for the LRC program.

Potential Correlates of Triglycerides

A variety of demographic, behavioral, biochemical, and dietary factors were assessed as potential covariates of TG. Age and education were ascertained at Visit 1. All other variables were measured as part of the Visit 2 screening. Demographic characteristics included age, sex, and years of education (<high school, high school, >high school). Behavioral characteristics were ascertained by questionnaire and included cigarette smoking, alcohol consumption, and physical activity. For the present study, cigarette smoking was categorized in four levels: never smoked, exsmoker, current smoker of less than 20 cigarettes per day, and current smoker of 20 cigarettes or more per day. Alcohol consumption during the week before the second screening was ascertained by a questionnaire, and ethanol was calculated in milliliters per day (ml/day) by using a formula that considered the differing amounts of ethanol in beer, wine, spirits, and liqueurs. Categories of alcohol consumption (ml/day) were: 0 (non-drinkers); 1–9; 10–19; 20–29; >30. A “yes” or “no” response to the question “Do you regularly engage in strenuous exercise or hard physical labor?” was used to characterize participants as active or sedentary. Use of diuretic medications in either sex, or oral contraceptives (OC) or estrogens in women, within the 2 weeks before the second screening was ascertained by questionnaire.

In addition to the above variables, fasting plasma glucose (FPG) levels and body mass were also ascertained as potential correlates of TG levels. Body mass was expressed as Quetelet’s Index (QI = [weight (kg) ÷ height (cm)2] × 1000) and categorized as lean (men <2.46; women <2.30), moderate (men 2.46–2.68; women 2.30–2.50) and obese (men ≥2.69; women ≥2.51). Cutpoints were derived from West and corresponded to <110% of “ideal” QI, 110% to 119% of “ideal,” and ≥120% of “ideal,” adapted for measurement of weight with participants wearing light clothing. The FPG levels were measured by Auto-Analyzer at one of two commercial laboratories by use of either the hexokinase method or a simultaneous dialysis technique, adapted from the method of Hoffman. Glucose values were categorized before analysis as: <80 mg/dl, 80 to 99
mg/dl, 100 to 109 mg/dl, and ≥110 mg/dl. Because of the small numbers of observations, it was not possible to further subdivide the highest FPG category.

The 24-hour dietary recalls were conducted at each clinic by dieticians who had been trained according to a common protocol. All dietary recalls were coded at the Nutrition Coding Center. With the use of standard food consumption tables, dietary intake was converted to represent the nutrient composition of the foods and beverages ingested. Selective dietary variables, derived from the 24-hour recall, were considered in these analyses. They included: sucrose (g/day), total calories per kilogram of body weight, alcohol (ml), and percentage of calories as total dietary fat, total carbohydrates, starch, and alcohol.

**Statistical Methods**

The present study was restricted to the 5189 white men and women aged 20 to 69 years who were selected in the 15% random sample of Visit 1 participants and who had fasted 12 hours or more, were not pregnant, and had a QI of less than 5.0. Five persons with a QI of more than 5.0 were excluded from those analyses that included QI as a covariate, since such extreme values would have had a disproportionate effect in the regression models. For most analyses, data are presented separately for two broad age groups, 20 to 44 and 45 to 69 years, and within those age groups for men, women not using gonadal hormones, and women using OC (aged 20–44 years) or estrogens (aged 45–69 years). Younger women using estrogens and older women using OC were excluded.

Because the distribution of TG values in this population is highly skewed, the natural logarithm (ln) transformation was used in all analyses. Age and sex-specific Pearson correlation coefficients of ln TG with the natural logarithms of total cholesterol, LDL-C, and HDL-C were computed for men, women not using gonadal hormones, and women using hormones. Natural log transformations of total cholesterol, LDL-C, and HDL-C were used since the associations of untransformed values with ln TG tended to be curvilinear. Prior to calculating the correlation coefficients, scatter diagrams of each TG-lipid pair were plotted by age to identify gross outliers and to check for linearity. Based on these scatter diagrams, values from seven persons, namely, five men (ln TG <2.7 or >5.9), one woman nonuser (ln TG >6.8), and one woman user (ln TG >6.0), were omitted as outliers from the correlation analyses. Age and clinic-specific correlations were computed for all strata with at least nine observations, and Fisher's transformation was used to test for the homogeneity of correlation coefficients across clinics. As this test indicated that data from the clinics could be reasonably combined within each age stratum, the clinic-specific transformed correlations were combined into a weighted average by using the inverse of the variance of the correlations as weights. The correlation of VLDL-C and TG was not assessed. The Visit 2 measurement of VLDL-C was an approximation derived by subtracting the sum of LDL-C and HDL-C values from total cholesterol, and negative values occasionally resulted. When VLDL-C is measured directly, the reported correlation of VLDL-C with TG is approximately 0.90.16

Except for the nutrient variables, age- and clinic-adjusted mean ln TG were computed for each category of the nonlipid covariates with the use of a general linear model. The midpoint of the appropriate age group, i.e., 32 or 57 years, and an arbitrarily selected clinic (Toronto) were used as the reference values for covariance adjustments. Adjusted values were estimated by using separate models for the six age, sex, hormone-use groups. The antilogs of these adjusted mean ln values are presented for ease of interpretation, and allow for visual comparison across categories of the covariates. However, the antilogs of the means from this analysis are not necessarily the same values that would be obtained from a similar analysis of untransformed TG values.

The independent contribution of each factor to the variance in In TG was assessed with multiple linear regression models. Separate models were computed for men, women not using gonadal hormones, and hormone users. In these multivariable analyses, smoking, education, diuretic use, and physical activity were retained as categorical variables, while age, alcohol, QI, and fasting plasma glucose were treated as continuous variables.

Because of the large measurement error inherent in the use of a 24-hour recall to characterize an individual's usual dietary intake, nutrient values were not used as independent variables in the multiple regression model. Such models require the assumption that independent variables are measured without error. However, 24-hour nutrient data can be used to estimate the diet of a group of individuals (e.g., persons in TG quartiles), because the errors associated with individual measurements averaged over a large group of observations have less impact on the estimate obtained. Thus, rather than computing age and clinic-adjusted mean TG values by level of nutrient intake, as was done for the other covariates, mean values of each selected dietary parameter, adjusted for age and clinic, were computed by quartile of TG. For the analysis of nutrient data, TG quartile cutpoints were based on values from the Visit 1 population for 5-year age strata within sex and hormone-use groups.

**Results**

**Demographic Characteristics**

Median TG values in the Visit 1 population are shown for men, women using gonadal hormones, and women nonusers in Figure 1. In these cross-sectional data, TG values tended to increase with age until about age 45 years. This increase continued in women not using gonadal hormones. Howev-
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Figure 1. Age-specific median plasma triglyceride values in white men (○) and women using (▲) and not using (■) gonadal hormones. Visit 1 population.

In hormone users and in men, median TG values were approximately the same from ages 45 to 69, except for a decline in the median value for men aged 65 to 69 years. Triglyceride levels were generally higher in hormone users than in nonusers. Prior to age 50, where most of the hormone use was OC, median TG values in hormone users were closer to those observed in men than to values in nonusers.

Correlations with Other Lipoproteins

The age-specific correlation coefficients for ln TG with the natural logarithms of total cholesterol, LDL-C, and HDL-C are given in Table 1. Ln TG was positively correlated with ln total cholesterol in each age, sex, and hormone-use group, although the correlations were strongest before age 50 years. In women, TG and LDL-C were moderately, positively correlated. The weakest association between TG and HDL-C in women was observed in older estrogen users. Only younger men (aged 20–39) showed a similar strong positive association between these lipids. There was a strong inverse correlation between TG and HDL-C in men, women not using sex hormones, and in older women (aged 50–69 years) using estrogens. While still inversely related, the correlation between HDL-C and TG was weaker in young women using OC.

Age and clinic-adjusted mean TG values by category of mean alcohol consumption per day as determined from the Visit 2 interview are given in Figure 2. In both younger and older men, higher mean TG values were associated with greater levels of alcohol consumption. A consistent positive relationship between mean TG levels and quantity of alcohol used was generally not observed in women. Mean TG values in older women, both estrogen users and nonusers, tended to decrease with increasing levels of alcohol consumption greater than 19 ml/day.

The relationship between cigarette smoking and TG is shown in Figure 3. Except for older women using estrogens, persons smoking 20 or more cigarettes per day tended to have the highest TG values. Compared with nonsmokers, ex-smokers also generally had slightly higher mean TG levels. Among older women using estrogens, the highest TG values were observed in women smoking fewer than 20 cigarettes a day.

In both age groups of men and women not using hormones, Figure 4 shows an increase in TG values with increasing glucose level. This positive association was fairly consistent in these groups. However,

Table 1. Correlation Coefficients of the Logarithm (Ln) Triglycerides with Total Cholesterol, LDL Cholesterol, and HDL Cholesterol by Age, Sex, and Hormone Use

<table>
<thead>
<tr>
<th>Sex and hormone use</th>
<th>Age (yrs)</th>
<th>No.</th>
<th>Ln total cholesterol</th>
<th>Ln LDL-C</th>
<th>Ln HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>20–29</td>
<td>359</td>
<td>0.45</td>
<td>0.40</td>
<td>-0.42</td>
</tr>
<tr>
<td></td>
<td>30–39</td>
<td>774</td>
<td>0.47</td>
<td>0.20</td>
<td>-0.46</td>
</tr>
<tr>
<td></td>
<td>40–49</td>
<td>708</td>
<td>0.40</td>
<td>0.05</td>
<td>-0.40</td>
</tr>
<tr>
<td></td>
<td>50–59</td>
<td>601</td>
<td>0.32</td>
<td>0.05</td>
<td>-0.50</td>
</tr>
<tr>
<td></td>
<td>60–69</td>
<td>224</td>
<td>0.27</td>
<td>0.03</td>
<td>-0.49</td>
</tr>
<tr>
<td>Women nonusers</td>
<td>20–29</td>
<td>270</td>
<td>0.32</td>
<td>0.32</td>
<td>-0.31</td>
</tr>
<tr>
<td></td>
<td>30–39</td>
<td>481</td>
<td>0.47</td>
<td>0.43</td>
<td>-0.32</td>
</tr>
<tr>
<td></td>
<td>40–49</td>
<td>486</td>
<td>0.46</td>
<td>0.41</td>
<td>-0.41</td>
</tr>
<tr>
<td></td>
<td>50–59</td>
<td>331</td>
<td>0.38</td>
<td>0.31</td>
<td>-0.59</td>
</tr>
<tr>
<td></td>
<td>60–69</td>
<td>190</td>
<td>0.35</td>
<td>0.30</td>
<td>-0.60</td>
</tr>
<tr>
<td>Women users</td>
<td>20–29</td>
<td>216</td>
<td>0.44</td>
<td>0.35</td>
<td>-0.18</td>
</tr>
<tr>
<td></td>
<td>30–39</td>
<td>120</td>
<td>0.52</td>
<td>0.33</td>
<td>-0.10</td>
</tr>
<tr>
<td></td>
<td>40–49</td>
<td>104</td>
<td>0.42</td>
<td>0.30</td>
<td>-0.18</td>
</tr>
<tr>
<td></td>
<td>50–59</td>
<td>153</td>
<td>0.29</td>
<td>0.17</td>
<td>-0.30</td>
</tr>
<tr>
<td></td>
<td>60–69</td>
<td>70</td>
<td>0.18</td>
<td>0.21</td>
<td>-0.41</td>
</tr>
</tbody>
</table>
Figure 2. Age- and clinic-adjusted mean triglyceride values by category of alcohol consumption in milliliters of ethanol per day.

Figure 3. Age- and clinic-adjusted mean triglyceride values by category of cigarette smoking. (non = nonsmoking, ex = exsmoker; <20 = less than 20 cigarettes per day; ≥20 = 20 or more cigarettes per day).
Figure 4. Age- and clinic-adjusted mean triglyceride values by category of fasting plasma glucose (mg/dl).

Figure 5. Age- and clinic-adjusted mean triglyceride values by category of body mass (lean, moderate, and obese).
in both OC and estrogen users, this gradient was not observed, although highest TG means were seen in users with the highest glucose levels.

A comparison of age-adjusted mean TG values in lean, moderate, and obese participants is shown in Figure 5. In each age, sex, and hormone-use group, the highest mean TG values were observed in obese subjects. The positive association between TG and body mass was consistent in each group, with the exception of older women using estrogens.

Physical activity was associated with lower mean TG values (Figure 6). This relationship was consistently observed in each subgroup, although the magnitude of the difference in TG means between active and sedentary persons was large only among young men.

Figure 7 shows the relationship between TG levels and education. There was, in general, no consistent relationship between level of education and TG values among the groups examined. However, a positive association was observed in younger men and women not using gonadal hormones.

Mean TG values in persons using and not using diuretic medications are given in Figure 8. With the exception of young men, few of whom used diuretics, persons using diuretics had higher TG values compared with nonusers. This difference was most marked in women who were also taking OC and in older men.

All variables considered in Figures 2–8, as well as age and LRC population, were entered into a multiple linear regression model to identify those factors significantly associated with In TG values after adjustment for all others.

Results of multivariable analyses for men, women not using hormones, and hormone users are given in Table 2. In men, age, body mass, FPG, diuretic use, and cigarette smoking were significantly, positively associated with TG values. Physical activity was inversely related to TG as was the age² term, corresponding to the decline in TG values after age 55. Neither alcohol consumption nor education was significantly related to TG after adjustment for other variables.

Age, body mass, FPG, education, and cigarette smoking were significantly, positively associated with TG levels in women not using gonadal hormones. Again, alcohol consumption during the week before Visit 2 was not related to TG after multivariate adjustment.

In women using gonadal hormones, only body mass and cigarette smoking (≥20 per day) were significantly, independently associated with TG. The model R² values also indicate that this set of variables does not account for as much of the variation in TG among women using OC or estrogens as in men or in women not taking gonadal hormones.

Figure 6. Age- and clinic-adjusted mean triglyceride values by category of physical activity.
Figure 7. Age- and clinic-adjusted mean triglyceride values by category of education. HS = high school.

Figure 8. Age- and clinic-adjusted mean triglyceride values by category of diuretic medication use.
Table 2. Multiple Regression Coefficients for Selected Variables on the Logarithm (Ln) Triglycerides by Sex and Gonadal Hormone Use in Women

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men (nonusers)</th>
<th>Women (users)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β</td>
<td>SE(β)</td>
</tr>
<tr>
<td>Age</td>
<td>0.0485*</td>
<td>0.0059</td>
</tr>
<tr>
<td>Age²</td>
<td>-0.0005*</td>
<td>0.0001</td>
</tr>
<tr>
<td>Alcohol (ml/day)</td>
<td>0.0016</td>
<td>0.0008</td>
</tr>
<tr>
<td>Alcohol²</td>
<td>-0.0000</td>
<td>0.0000</td>
</tr>
<tr>
<td>Quetelet Index</td>
<td>0.4398*</td>
<td>0.0277</td>
</tr>
<tr>
<td>Fasting plasma glucose</td>
<td>0.0049*</td>
<td>0.0005</td>
</tr>
<tr>
<td>Physical activity (yes)</td>
<td>-0.0832*</td>
<td>0.0199</td>
</tr>
<tr>
<td>Diuretic use (yes)</td>
<td>0.1910†</td>
<td>0.0722</td>
</tr>
<tr>
<td>Education:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;HS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HS</td>
<td>0.0291</td>
<td>0.0225</td>
</tr>
<tr>
<td>≥HS</td>
<td>0.0155</td>
<td>0.0296</td>
</tr>
<tr>
<td>Smoking: smoking:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>never</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ex</td>
<td>0.3491†</td>
<td>0.0233</td>
</tr>
<tr>
<td>&lt;20/day</td>
<td>0.1444*</td>
<td>0.0343</td>
</tr>
<tr>
<td>≥20/day</td>
<td>0.1669*</td>
<td>0.0257</td>
</tr>
<tr>
<td>R²</td>
<td>0.244</td>
<td>0.344</td>
</tr>
</tbody>
</table>

*p < 0.0001.  
†p < 0.01.  
‡p < 0.05.

§For the categorical variables "education" and "smoking," the first category listed is the reference group to which other categories are compared. p values in parentheses are for the regression coefficient across all categories.

The multiple regression model also included terms that are not shown in the table to adjust for LRC populations. β is the beta coefficient derived from the multiple regression model.

Associations with Nutrient Intake

Mean values of selected nutrients, adjusted for age and LRC clinic population, are given in Table 3 by quartile of TG for men and women users and nonusers. The p values refer to the overall associations between TG and the individual nutrients, while the asterisks indicate significant differences in nutrient means between the highest TG quartile and other TG quartiles. If the overall association was not statistically significant, the differences between quartiles were not tested.

In younger men, the percentage of calories as starch, total calories per kilogram of body weight, and the percentage of calories as total fat were inversely related to TG, as the highest mean values tended to be observed in the lowest TG quartile. Both the percentage of calories as alcohol and milliliters of alcohol consumed in the previous 24 hours were positively associated with TG levels. The significance of these associations is apparently due to the high level of alcohol consumption in persons in the fourth quartile of TG, as there is little difference in either measure of alcohol use among Quartiles 1 through 3. In older men, only total calories per kilogram of body weight and the percentage of calories as total fat were significantly, inversely related to TG levels.

In younger women not using OC, total calories per kilogram of body weight and percentage of calories as total fat were inversely associated with TG values. In OC users, sucrose was positively, and percentage of calories as fat was inversely, related to TG. In women aged 45 to 69 years who were not using estrogens, total calories per kilogram of body weight was inversely related to TG, while the percentage of calories as starch and percentage of calories as carbohydrates were positively associated. This positive relationship between carbohydrate ingestion and TG was also observed in older women using estrogens and, as in nonusers, appeared to be largely due to lower mean levels of percentage of calories as carbohydrates in the first TG quartile.

The association between TG and alcohol consumption during the previous 24 hours was not the same in women as that seen in men. There was no significant relationship between alcohol and TG in younger women, regardless of hormone use. In both groups of older women, the percentage of calories as alcohol and milliliters of alcohol consumed were significantly, inversely associated with TG levels; the highest mean values of alcohol intake were observed among women in the lowest TG quartile. These results were not appreciably altered when mean values were also adjusted for HDL-C (Append...
Table 3. Age- and Clinic-Adjusted Mean Nutrient Values by Quartile of Triglycerides, Sex, Gonadal Hormone Use, and Age

<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Calories as starch (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>17.5</td>
<td>18.4</td>
<td>16.1</td>
<td>17.3*</td>
<td>16.3</td>
<td>16.6</td>
</tr>
<tr>
<td>2</td>
<td>18.0*</td>
<td>18.5</td>
<td>16.0</td>
<td>17.8*</td>
<td>15.9</td>
<td>17.1</td>
</tr>
<tr>
<td>3</td>
<td>17.9*</td>
<td>18.9</td>
<td>17.0</td>
<td>18.9</td>
<td>17.6</td>
<td>19.0</td>
</tr>
<tr>
<td>4</td>
<td>16.7</td>
<td>18.8</td>
<td>17.5</td>
<td>19.6</td>
<td>16.6</td>
<td>19.0</td>
</tr>
<tr>
<td>(p = 0.03)</td>
<td>(p = 0.80)</td>
<td></td>
<td>(p = 0.07)</td>
<td>(p = 0.006)</td>
<td>(p = 0.28)</td>
<td>(p = 0.06)</td>
</tr>
<tr>
<td>Sucrose (g)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>67.4</td>
<td>46.1</td>
<td>42.4</td>
<td>27.7</td>
<td>39.0</td>
<td>38.9</td>
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*pMean is significantly different (p ≤ 0.05) from mean of Quartile 4, after adjustment for multiple comparisons using Bonferroni's correction.

*p values given in parentheses are for the regression coefficient across all four quartiles for the individual nutrients.

Discussion

In men and women not using gonadal hormones, those factors significantly, independently related to fasting TG levels were remarkably similar, and only minor differences were observed between the sexes in significant covariates of TG. However, women using either OC or estrogens did not, in general, show evidence of the same TG correlates as their peers not using hormones. In women using gonadal hormones, only body mass and cigarette smoking were significantly associated with TG in the multivariable analysis, and these two variables were relatively strong predictors of TG in all three sex and hormone-use groups.

The absence of a significant association between TG and FPG in women using OC or estrogens is especially interesting, since the relationship was strong and consistent in men and women nonusers in these data and has been reported by others.\textsuperscript{17, 31-33}
especially in diabetics.34–37 While the exact mechanism is unclear, these results suggest that gonadal hormone use may modify the TG-FPG association seen in nonusers and men, perhaps by the effects of these hormones on insulin secretion38,39 or resistance.40,41

Triglyceride levels have been reported to be positively associated with various indices of body mass in a number of other studies16,17,42,43 and a decrease in TG has been observed with weight loss.15 Using different measures of body fatness and fat distribution, Albrink and Meigs17 proposed that higher TG levels are more closely associated with "acquired" than with "hereditary" obesity. These authors and others have suggested that caloric excess may explain the association between TG levels and body mass, although TG metabolism may be directly influenced by obesity.

Phillips et al.18 observed a significant, positive association between cigarette smoking and TG, adjusted only for age. The positive association observed in the present study was independent of differences among smoking categories in age, alcohol consumption, body mass, FPG, physical activity, diuretic use, and education, and was most marked for persons smoking ≥20 cigarettes per day. The reasons for this association are unclear, but cigarette smoking has been shown to be independently, positively related to levels of LDL-C45 and inversely to HDL-C.46

In these data, physical activity was significantly, inversely associated with TG levels only in men. The absence of an independent relationship in women may be due to the lower prevalence of physically active women in this population or to differences between the sexes in intensity of activity measured by the question used. Lower TG values in active individuals have been reported from other cross-sectional data,47 and decreases in TG have been observed in association with physical conditioning.48–51 The mechanism by which physical activity may influence TG levels is unclear. In these data, the association was independent of differences between active and sedentary men in body mass, alcohol consumption, cigarette smoking, age, and FPG. While it has been suggested that negative caloric balance may explain the inverse association between exercise and TG levels, Gyntelberg et al.51 showed a reduction in TG levels in men participating in an exercise program, whether or not their increased caloric expenditure was compensated for by an increased caloric intake. A more likely explanation may be a direct, more chronic influence of activity on VLDL synthesis or catabolism.49

A positive association between use of diuretics and TG levels has been observed in other studies.8,9 Cutler52 recently reported some differences in the magnitude of change in TG levels after institution of diuretic therapy, depending on the drug used. There were too few users of diuretics in the present study to examine the relationship of TG with individual formulations. Ames and Hill53 observed close associations between changes in glucose, fasting insulin, and TG levels both during and after antihypertensive therapy and suggested that a common mechanism for these effects might be a change in insulin resistance during diuretic-based antihypertensive therapy.

Triglycerides were strongly, positively correlated with total cholesterol and LDL-C and inversely correlated with HDL-C in most age and sex subgroups. Two exceptions to these general trends were the absence of a correlation between TG and LDL-C in men aged 40 to 69 years and the low-order negative correlation between TG and HDL-C in OC users. The lack of correlation between TG and LDL-C in older men in these cross-sectional data may be due to selective survival in that those men with high TG and high LDL-C had already died of coronary heart disease. However, a positive correlation on the order of 0.27 between TG and LDL-C has been reported by Nicholson et al.54 in men aged 80 years and older. Such an explanation also requires the assumption that high TG and high LDL-C are a more "lethal" combination in men than high TG plus high total cholesterol or low HDL-C, because these two latter combinations did not show the same decline in magnitude of correlation with age. Phillips et al.18 reported a positive correlation between LDL-C and TG in men for TG levels up to about 100 mg/dl, after which the association was inverse. In persons with high TG values, as VLDL-TG increases, LDL-C tends to decrease.44 Thus, the low correlation of LDL-C and TG in men over 40 years of age may reflect an increasing proportion of men with high TG in this age group (Figure 1). The low correlation of TG with HDL-C in OC users, compared with that observed in nonusers and in men, may be a result of the variety of preparations used, as the influence of OC on these lipids varies dramatically depending on the relative estrogen-progesteron formulation.11

Triglycerides tended to be inversely associated with total calories per kilogram of body weight and percentage of calories as dietary fat in most age, sex, and hormone-use subgroups. The inverse association of total calories per kilogram and TG quartile may be explained in part by the observation that total calories consumed are inversely related to relative body weight,54 and body weight is positively related to TG values.22 Since an intake of more calories at a fixed body weight may reflect higher physical activity levels, the inverse trend of calories and TG might also be due to the effects of exercise. The inverse relationship of percentage of calories as total fat could be explained by a positive association of carbohydrate and TG, since these nutrients were expressed as a percentage of total calories, and as one increases, others must decrease. A more detailed analysis of the dietary correlates of TG in this population22 indicated that grams of total fat, saturated fat, and polyunsaturated fat were all inversely correlated with TG.

It is important to note that because the participants had been fasting at least 12 hours, the associations...
of dietary intake and TG do not reflect the influence of acute ingestion of these nutrients on TG values. In addition, the associations were not adjusted for covariates other than age and LRC clinic. Although a number of nutrients identified from the 24-hour dietary recall were significantly related to TG levels, the differences in mean intake by TG quartile tended to be small. This observation suggests that dietary intake did not contribute appreciably to the variation in fasting TG.

Two measures of alcohol consumption were used in the present study; the alcohol consumed within the 24 hours before fasting for the Visit 2 lipid measurements (obtained from the 24-hour dietary recall) and the amount of alcohol drunk within the week before Visit 2 (obtained from the Visit 2 interview). In the analysis of dietary data, both percentage of calories as ethanol and milliliters of ethanol drunk in the previous 24 hours were positively associated with TG in men aged 20 to 44 years and inversely associated in 45- to 69-year-old women. No trend was discernible in younger women. After additional adjustment for HDL-C (Appendix A), both dietary measures of alcohol consumption were significantly, positively associated with TG in older, as well as younger, men. This difference between the sexes in the effect of alcohol exposure has been reported by others and may reflect sex differences in the accuracy of reporting dietary intake, a higher proportion of heavy drinkers in men, or the influence of factors that were not included in the statistical adjustment of dietary data but that are associated with both alcohol intake and TG.

The amount of alcohol consumed in the previous week may be a better indicator of patterns of usual use than that reported during the previous 24 hours. After multivariable adjustment, the average amount of ethanol consumed per day (based on reported weekly consumption) was not significantly associated with TG levels in either men or women. This finding is contrary to reports of a positive association between TG and frequency of drinking and ounces of alcohol consumed per week, but has been observed in another study that also used the amount of ethanol drunk per week as the measure of alcohol consumption. In the present study, when HDL-C was included as a covariate in the multivariate analyses (Appendix B), milliliters of ethanol per day was significantly, positively associated with TG in men (p < 0.0001) and was of borderline significance in women not using gonadal hormones (p = 0.06).

The relatively strong correlations among the various lipids and lipoproteins complicate attempts to evaluate the association of any single lipid with suspected covariates. In particular, the strong inverse correlation of TG and HDL-C may obfuscate the association of TG with other factors that are also related to HDL-C and with the risk of coronary disease. All analyses presented here were repeated, including a covariance adjustment for HDL-C (Appendixes A and B). While the HDL-C adjustment tended to reduce the magnitude of most observed associations, with few exceptions the significant relationships of demographic, behavioral, biochemical, and dietary factors with TG persisted. As mentioned previously, inclusion of HDL-C as a covariate in the multivariable analysis actually strengthened the association between regular alcohol consumption and TG in men and women nonusers.

The statistical independence of the association of TG with several important coronary heart disease (CHD) risk factors (age, smoking, body mass, fasting glucose, and alcohol consumption) is of interest, since failure to adjust for HDL-C has been cited as an explanation for the significant TG-CHD association observed in some studies. These results also suggest that while the factors associated with TG and HDL-C levels may be similar, these factors may influence TG and HDL-C through different mechanisms. Both dietary intervention and alcohol restriction have been shown to have somewhat differing and independent effects on TG and HDL-C in persons with hypertriglyceridemia. It has also been noted that HDL-C may not be a good measure of HDL mass in persons with high TG levels since the HDL in such persons tends to be enriched with TG at the expense of cholesterol esters.

The patterns of associations of TG with the known CHD risk factors investigated in the present study are consistent with the hypothesis that high levels of TG increase CHD risk. That is, TG were positively associated with several factors that are themselves positively associated with risk of CHD. The present study does not clarify the nature of the TG-CHD association, i.e., whether it is direct or indirect. However, it is clear that the relationships of TG with other lipids, age, hypertension, sedentary, and were more likely to be cigarette smokers. While controversy remains regarding the treatment of persons with hypertriglyceridemia, the results of this and other studies suggest that the presence of elevated TG levels increases the possibility that other CHD risk factors are present. In addition, the associations observed in the present study suggest areas for possible nonpharmacologic treatment of hypertriglyceridemia, several of which were mentioned in the recent National Institutes of Health Consensus Statement on Hypertriglyceridemia.
References


### Appendix A

Table A-1. Age-, Clinic-, and HDL-Adjusted Mean Nutrient Values by Quartile of Triglycerides, Sex, Gonadal Hormone Use, and Age

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<th>45-69 yrs</th>
<th>20-44 yrs</th>
<th>45-69 yrs</th>
<th>20-44 yrs</th>
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<td>4.6*</td>
<td>4.2</td>
<td>2.3</td>
<td>1.0</td>
<td>2.1</td>
<td>0.31</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>7.7</td>
<td>5.0</td>
<td>2.7</td>
<td>1.8</td>
<td>3.1</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(p = 0.0001)</td>
<td>(p = 0.0001)</td>
<td>(p = 0.45)</td>
<td>(p = 0.07)</td>
<td>(p = 0.61)</td>
<td>(p = 0.05)</td>
</tr>
<tr>
<td>Ethanol (ml/24 hrs)</td>
<td>1</td>
<td>22.4*</td>
<td>10.3*</td>
<td>6.9</td>
<td>5.4</td>
<td>8.0</td>
<td>10.1</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>28.6*</td>
<td>16.2*</td>
<td>7.5</td>
<td>1.3*</td>
<td>7.8</td>
<td>0.37</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>27.3*</td>
<td>19.6</td>
<td>8.1</td>
<td>2.5</td>
<td>6.3</td>
<td>0.48</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>49.2</td>
<td>24.0</td>
<td>10.9</td>
<td>6.4</td>
<td>10.2</td>
<td>2.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(p = 0.0001)</td>
<td>(p = 0.001)</td>
<td>(p = 0.23)</td>
<td>(p = 0.03)</td>
<td>(p = 0.73)</td>
<td>(p = 0.05)</td>
</tr>
</tbody>
</table>

*Mean is significantly different (p ≤ 0.05) from mean of Quartile 4, after adjustment for multiple comparisons by using Bonferroni’s correction.

p values given in parentheses are for the regression coefficient across all four quartiles for the individual nutrients.
### Appendix B

Table B-1. Multiple Regression Coefficients for Selected Variables on Logarithm (Ln) Triglycerides by Sex and Gonadal Hormone Use in Women, Including Adjustment for High Density Lipoprotein Cholesterol (HDL-C)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th></th>
<th>Women (nonusers)</th>
<th></th>
<th>Women (users)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.0397*</td>
<td>0.0054</td>
<td>0.0159†</td>
<td>0.0056</td>
<td>0.0204‡</td>
</tr>
<tr>
<td>Age²</td>
<td>-0.0004*</td>
<td>0.0001</td>
<td>-0.0000</td>
<td>0.0001</td>
<td>-0.0001</td>
</tr>
<tr>
<td>Alcohol (ml/day)</td>
<td>0.0049*</td>
<td>0.0008</td>
<td>0.0032</td>
<td>0.0017</td>
<td>0.0006</td>
</tr>
<tr>
<td>Alcohol²</td>
<td>-0.000002†</td>
<td>0.0000</td>
<td>-0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
<tr>
<td>Quetelet Index</td>
<td>0.3062*</td>
<td>0.0259</td>
<td>0.1793*</td>
<td>0.0222</td>
<td>0.1523†</td>
</tr>
<tr>
<td>Fasting plasma glucose</td>
<td>0.0043*</td>
<td>0.0005</td>
<td>0.0040*</td>
<td>0.0006</td>
<td>0.0010</td>
</tr>
<tr>
<td>Physical activity (yes)</td>
<td>-0.0502†</td>
<td>0.0182</td>
<td>-0.0010</td>
<td>0.0267</td>
<td>-0.0147</td>
</tr>
<tr>
<td>Diuretic use (yes)</td>
<td>0.1468§</td>
<td>0.0659</td>
<td>0.0281</td>
<td>0.0362</td>
<td>0.0653</td>
</tr>
</tbody>
</table>

**Education:**

| < HS                         | 0.0210    | 0.0205       | 0.3218          | 0.0217       | -0.0289       | 0.0344       |
| ≥ HS                         | 0.0216    | 0.0270       | 0.0797†         | 0.0311       | -0.0152       | 0.0590       |

*(p = 0.53) (p = 0.04) (p = 0.70)*

**Smoking:**

| Never                        | $§$        | $§$          | $§$             | $§$          |
| ex                           | 0.0411    | 0.0213       | 0.0046          | 0.0255       | -0.0173       | 0.0415       |
| < 20/day                     | 0.1057†   | 0.0313       | 0.0637†         | 0.0296       | 0.0919        | 0.0495       |
| ≥ 20/day                     | 0.0722†   | 0.0238       | 0.1210*         | 0.0278       | 0.0670        | 0.0454       |

*(p = 0.002) (p < 0.0001) (p = 0.12)*

| R²                           | 0.371     | 0.412        | 0.157           |              |

* *p < 0.0001.
† *p < 0.01.
‡ *p ≤ 0.05.
§ For the categorical variables “education” and “smoking,” the first category listed is the reference group to which other categories are compared. p values in parentheses are for the regression coefficient across all categories.

The multiple regression model also included terms that are not shown in the table to adjust for HDL-C, HDL-C², and for Lipid Research Clinics populations.

### Acknowledgments

**Epidemiology Analysis Executive Committee**


**Lipid Research Clinics Epidemiology Committee**


**Lipid Research Clinics Directors Committee**


Index Terms: triglycerides • diet • smoking • exercise • alcohol • body mass • glucose
L D Cowan, T Wilcosky, M H Criqui, E Barrett-Connor, C M Suchindran, R Wallace, P Laskarzewski and C Walden

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