Lipoprotein–Cholesterol Distributions in Selected North American Populations: The Lipid Research Clinics Program Prevalence Study

GERARDO HEISS, M.D., ISRAEL TAMIR, M.D., CLARENCE E. DAVIS, PH.D., HERMAN A. TYROLER, M.D., BASIL M. RIFKIND, M.D., GUSTAV SCHONFELD, M.D., DAVID JACOBS, PH.D., AND IVAN D. FRANTZ, JR., M.D.

SUMMARY Total plasma lipid and lipoprotein-cholesterol distributions of 4756 white men and women ages 20–59 years are presented. Measurements were obtained during the visit-2 survey of the Lipid Research Clinics Program Prevalence Study and correspond to a 15% random sample of 35,748 white adults screened during the LRC visit-1 survey. Standardized examinations were carried out by 10 North American clinics using a common protocol, on diverse target populations chosen to include a range of sociodemographic characteristics. Age-specific means, medians and selected percentiles are given by sex, with stratification on exogenous sex hormone use in women.

Plasma lipid and lipoprotein concentrations in men and women vary with age. Differences in lipid and lipoprotein levels between the study populations are also present and manifest themselves as parallel trends of age-related changes in the 10 populations examined.

Higher total cholesterol values in men compared with women appear between the ages 20–50 years and higher LDL cholesterol between the ages 20–55 years. VLDL cholesterol levels are similar in both sexes at ages 20 and 59 years but higher in men than in women in all intermediate age groups. HDL cholesterol is higher in women than in men throughout the age range considered.

Women taking sex hormone preparations have higher total cholesterol than women not on hormones between ages 20–50 years, and higher LDL cholesterol between ages 20–40 years. From the third age decade onward, HDL cholesterol levels are progressively higher in women taking hormones than in women not taking sex hormones. Compared with women not taking exogenous sex hormones, women taking hormones have higher total plasma triglyceride values at all ages from 20–59 years. VLDL cholesterol values are higher in women on hormones compared with nonusers of hormones younger than 55 years.

THE DATA relating atherosclerotic coronary heart disease to plasma lipids have grown extensively in this century. Cumulative confirmatory evidence from animal experimental, clinical case series, and epidemiologic population studies have established the coronary heart disease risk-factor status of total plasma cholesterol. Early epidemiologic studies of plasma lipoproteins resulted in controversy regarding the information predictive of coronary heart disease provided by plasma ultracentrifugation in addition to that derived from total cholesterol measurement. The further development of concepts of plasma lipid transport mechanisms and simplified methods for measurement and classification of lipoprotein abnormalities stimulated numerous clinical and epidemiologic investigations. The negative-risk-factor status of HDL cholesterol (HDL-C) and the positive-risk-factor status of LDL cholesterol (LDL-C) have been shown in case-control studies, cross-sectional population studies, and cohort studies. These studies suggest an independent contribution of the plasma lipoprotein fractions to the risk of coronary heart disease; however, additional information regarding quantification of the coronary heart disease risk among populations with different distributions of other risk factors, genotypes, and lifestyle attributes is required.

The Lipid Research Clinics (LRC) Program was designed to contribute to knowledge of the determinants of plasma lipids and lipoproteins in populations and their cardiovascular sequelae. A series of studies was initiated that constituted a coronary primary prevention clinical trial and a program of population-based surveys. Epidemiologic surveys have been completed in 13 settings in the U.S., Canada, Israel, and the USSR; data collection for family studies and a mortality follow-up are in progress.

In this communication we present a description of the major characteristics of the lipid and lipoprotein distributions related to sex, age and sex hormones utilization by women among the 10 North American populations studied in the LRC Program. Further analyses of these data are in progress, and reports will be issued separately on the effects of genetic and environmental factors, and their relationship to mor-
bidity and mortality from atherosclerotic vascular disease.

**Methods**

The population studies component of the LRC Program has been described in detail elsewhere. Briefly, 10 LRCs in the United States and in Canada completed a series of population surveys between 1971–1976. The LRCs selected their study populations to ensure wide ethnic, geographic, socioeconomic and age variation; the collaborative LRC data are not, however, representative of the Canadian or U.S. populations. All clinics used a common protocol and highly standardized methods.

The Prevalence Study involved a brief visit-1 screen to determine plasma cholesterol and triglyceride and to obtain sociodemographic data and a medication history. For the second, more extensive examination, the participating LRCs first recalled a 15% random sample of visit-1 participants. Subsequently, the remaining visit-1 records were reviewed, and participants with elevated lipid levels or who were taking lipid-lowering medication were also invited to participate in the visit-2 screen.

Data collected at visit 2 included plasma lipids and lipoprotein-cholesterol quantification, resting and graded exercise electrocardiography, detailed dietary and medication history, nonlipid clinical chemistries, blood pressure and anthropometric indices.

A total of 12,595 white participants of both sexes were examined during the LRC visit-2 survey. Of these, 7055 constitute a 15% randomly sampled subset of LRC visit-1 participants and 5540 are visit-1 examinates recalled to the second examination on grounds of hypercholesterolemia or hypertriglyceridemia. The visit-2 survey also examined 1257 nonwhite participants of both sexes; these observations are not included in this report and are the topic of separate LRC publications.

The sampling frames and number of study participants for the LRCs are presented in table 1. Overall participation rates were 74% for visit 1 and 85% for visit 2. The overall response rate for the data presented in this report, i.e., white visit-1 participants eligible for visit-2 examination, was 88%, with negligible differences by sex or age.

**Laboratory Methods**

Blood specimens were obtained from participants who had fasted for at least 12 hours. Venipuncture was done with the examinees in a sitting position; a tourniquet was used, but was released before collection of the blood sample. All samples were immediately placed on wet ice, and the standardized lipid laboratory procedures were initiated within 3 hours after venipuncture.

A detailed description of the laboratory processing procedures is provided in the LRC Laboratory Methods Manual. Briefly, plasma cholesterol and triglyceride levels were determined in each LRC core laboratory by use of Technicon Autoanalyzer I or Autoanalyzer II, adapted by the LRC Program. On frozen serum pools the AA-I instruments produced cholesterol values 2.1% higher than the manual Abell-Kendall target values; the AA-II instruments gave values 1.3% lower than target values. For plasma triglyceride concentrations the individual instrument biases varied from 4.9% below to 1.0% above reference values. Between-run variability was often less than within-run variability and interlaboratory variation was considerably less than intralaboratory variation.

All study determinations were done on fresh samples and the comparative data indicate differences even less than the values ascertained for frozen samples.

Uniform internal quality control and external surveillance systems were used in each LRC lipid laboratory in order to stabilize performance throughout the entire project and to achieve comparability of interlaboratory results. Control limits were established and were used to evaluate each day’s analysis. Cholesterol analyses of nine pools by the LRC laboratories over 12 months approximated the reference value (assigned by the CDC Lipid Standardization Laboratory, using the manual Abell-Kendall method) within 0.2%–2.1% over a range of 134–343 mg/dl. The overall coefficient of variation ranged from 3.4% at the lowest concentration to 1.9% at the highest concentration.

Lipoproteins were separated by ultracentrifugal flotation at saline density (d = 1.006 g/ml), to yield a supernatant fraction containing VLDL cholesterol (VLDL-C) and an infranatant fraction containing both LDL-C and HDL-C.

HDL-C was estimated in total plasma after precipitation of the apo-B–containing lipoproteins by means of heparin-manganese chloride. After direct estimation of HDL-C, LDL-C was observed by the formula: LDL-C = cholesterol in the 1.006 infranatant – HDL-C. VLDL-C was determined by the formula: VLDL-C = total plasma cholesterol –1.006 infranatant cholesterol.

In instances of incomplete precipitation of VLDL-C and LDL-C, the procedure was repeated on the infranatant fraction after ultracentrifugation.

To ensure interlaboratory comparability of ultracentrifugal results, 53 unfrozen plasma pools were distributed quarterly over a 3½-year period to each laboratory. The average coefficients of interclinic variability in lipoprotein-cholesterol analyses in these samples were 5% for LDL-C, 10–15% for HDL-C, and 10–40% for VLDL-C.

**Statistical Methods**

The aim of the statistical analysis was to describe the collaborative data, taking into account any possible differences between the populations surveyed. Multiple linear regression was used to construct a model of each lipid and lipoprotein as a function of age for each of three groups: males, females not taking hormones, and females taking hormones. The first step in the analysis was to fit a fifth-degree polynomial for age. Using a stepwise procedure, we determined that a linear quadratic equation was adequate for all
Table 1. Number of Eligible and Screened Examinees at Visit 1 and Visit 2 by Participating Lipid Research Clinics Program Prevalence Study, 1971–1976

<table>
<thead>
<tr>
<th>Sampling frame</th>
<th>Clinic</th>
<th>Visit 1 Number</th>
<th>Response rate (%)</th>
<th>Visit 2 Number</th>
<th>Response rate (%)</th>
<th>Visit 2, random sample,*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Eligible</td>
<td>Screened</td>
<td></td>
<td>Eligible</td>
<td>Screened</td>
</tr>
<tr>
<td>School children and their parents</td>
<td>Baylor</td>
<td>5,039</td>
<td>3,795</td>
<td>64</td>
<td>902</td>
<td>637</td>
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<tr>
<td></td>
<td>Cincinnati</td>
<td>15,571</td>
<td>10,803</td>
<td>70</td>
<td>3,049</td>
<td>2,652</td>
</tr>
<tr>
<td>Households</td>
<td>Iowa</td>
<td>5,594</td>
<td>3,918</td>
<td>70</td>
<td>1,999</td>
<td>999</td>
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<tr>
<td></td>
<td>Johns Hopkins</td>
<td>5,075</td>
<td>4,393</td>
<td>87</td>
<td>1,145</td>
<td>1,041</td>
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<td></td>
<td>La Jolla</td>
<td>7,791</td>
<td>6,087</td>
<td>78</td>
<td>1,644</td>
<td>1,459</td>
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<td></td>
<td>Minnesota</td>
<td>8,392</td>
<td>6,750</td>
<td>80</td>
<td>1,779</td>
<td>1,380</td>
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<tr>
<td></td>
<td>Oklahoma</td>
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<td>7,179</td>
<td>75</td>
<td>1,958</td>
<td>1,638</td>
</tr>
<tr>
<td>Occupational and industrial groups</td>
<td>Seattle</td>
<td>6,046</td>
<td>4,999</td>
<td>83</td>
<td>1,388</td>
<td>1,191</td>
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<tr>
<td></td>
<td>Stanford</td>
<td>6,028</td>
<td>4,280</td>
<td>71</td>
<td>1,189</td>
<td>1,074</td>
</tr>
<tr>
<td></td>
<td>Toronto-McMaster</td>
<td>11,968</td>
<td>8,208</td>
<td>60</td>
<td>2,182</td>
<td>1,781</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>81,926</td>
<td>60,502</td>
<td>74</td>
<td>16,335</td>
<td>13,832</td>
</tr>
</tbody>
</table>

*Of those screened, the following were excluded from analysis: nonwhite participants (n = 1257), Visit-2 examinees recalled on grounds of hyperlipidemia or lipid-lowering treatment (n = 5540); fasting less than 12 hours (n = 24); pregnant women (n = 13); ages less than 20 and greater than 59 years (n = 2251); missing information on sex hormone usage (n = 11).

We are reporting the distributions of plasma lipid and lipoprotein levels for 4756 white participants, ages 20–59 years, who were recalled to visit 2 as part of the 15% random sample. (Table 1 provides additional information on the study populations.) Mean and selected percentile values for plasma cholesterol and triglyceride, and lipoprotein-cholesterol fractions are given by 5-year age groupings in Appendix 1 (tables 1–6); means are also portrayed in figures 1–8. Given the high prevalence of sex hormone usage and the demonstrated influences of exogenous sex hormones on lipid and lipoprotein levels, definitions of sex hormone usage differ slightly in various publications, resulting in minor differences in the descriptive data.)

Not included in this report are white participants recalled to visit 2 on grounds of high plasma lipids (n = 5540);* nonwhite participants (n = 1257);* participants younger than 20 years or older than 59 years (n = 2251);* participants fasting less than 12 hours (n = 24); and women who were pregnant (n = 13) or for whom no data on sex hormone usage were available (n = 11).

The main features of multiple regression equations that best describe the plasma lipid and lipoprotein levels for the successive birth cohorts in the study population are presented in table 2. Females taking hormones have a linear relationship between their plasma lipid and lipoprotein levels and age. This is also true for HDL-C in females not taking hormones, while curvilinear relationships with age are the characteristic of all other lipid parameters in these females. Lipid and lipoprotein levels of males are best approximated by a curvilinear fit.

Plots of the best fit of lipid values on age are presented in figures 1–8 for the subpopulations defined by sex and hormone usage. The composition of these figures incorporates additional information in that the range of interclinic variation of lipid values is presented in each triplet of estimated curves. A lipid-age curve was computed for each of the participating LRCs; the lipid levels from the highest and the lowest clinics correspond to the upper and lower curves shown in the figures as interrupted lines. The middle curve in turn represents the lipid/lipoprotein levels for the median clinic.

*Separate reports will be issued for these visit-2 participants.
Cross-sectional Variation of Plasma Lipid and Lipoprotein Fractions with Age

**Plasma Cholesterol and Triglyceride Levels**

Figure 1 shows mean plasma cholesterol and triglyceride levels by age for white male LRC participants ages 20–59 years. Cholesterol values increase from 162 mg/dl in the age group 20–24 years to a maximum of 215 mg/dl in the 50–54-year age group. The slope of the cholesterol increment over successive age cohorts is greatest between 20–34 years; thereafter, the age-related increase in cholesterol levels is progressively less pronounced until a plateau is reached over the age strata 50–59 years. The triglyceride values of white males rise from 89 mg/dl in the 20–24-year age group to a peak between the ages 40–44 years, and decrease subsequently to a mean value of 134 mg/dl at ages 55–59 years.

Mean plasma cholesterol and triglyceride levels by age for white females not taking hormones are presented in figure 2. Plasma cholesterol levels show a curvilinear increase over the successive birth cohorts examined, from a mean value of 162 mg/dl at ages 20–24 years to 231 mg/dl at ages 55–59 years. Plasma triglycerides follow a similar pattern of increase with age, from 68 mg/dl to 132 mg/dl at the youngest and oldest age groups reported here.

Among white females on exogenous sex hormone preparations, both cholesterol and triglycerides show a linear increase with age (fig. 3). The mean cholesterol level of 20–24-year-olds is 178 mg/dl, and after a monotonic and linear increase over successive birth cohorts it reaches 222 mg/dl in the 55–59-year-olds. Fasting plasma triglyceride levels of females can be seen to increase with age, from 106 mg/dl at ages 20–24 years to 138 mg/dl at ages 55–59 years.

**Mean Lipoprotein-Cholesterol Levels**

Among white males, LDL-C shows a steep rise from ages 20–24 years (103 mg/dl) to ages 45–49

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**Figure 1.** Regression estimates of mean plasma lipid values by age and clinic, white males, ages 20–59 years. Lipid Research Clinics Prevalence Study, Visit 2, random sample.

**Figure 2.** Regression estimates of mean plasma lipid values by age and clinic, white females not taking sex hormones, ages 20–59 years. Lipid Research Clinics Prevalence Study, Visit 2, random sample.

**Figure 3.** Regression estimates of mean plasma lipid values by age and clinic, white females taking sex hormones, ages 20–59 years. Lipid Research Clinics Prevalence Study, Visit 2, random sample.
years (144 mg/dl) (fig. 4). Thereafter, the observed increment with age is less pronounced, and a plateau appears at ages 50–59 years. HDL-C levels remain at a stable 44 mg/dl across age strata between the ages 20–54 years but rise to 48 mg/dl in the oldest age group (fig. 4).

VLDL-C levels in white males show a parabolic relationship to age at 20–59 years. The lowest value is observed among the 20–40-year-olds (14 mg/dl), an acme is reached between the ages 40–44 years (26 mg/dl), and is followed by a decrement (22 mg/dl at ages 55–59 years).

Mean levels of lipoprotein-cholesterol fractions by age for females not on hormones are shown in figure 5. LDL-C values rise with age, and have larger increments among middle-aged females compared with the slope of LDL-C increase among younger females. Mean LDL-C among 20–24-year-old females is 98 mg/dl and 150 mg/dl among the 55–59-year-olds.

HDL-C changes with age among females not on sex hormones are depicted in figure 5 as a moderate, linear increase from a low of 52 mg/dl (ages 20–24 years) to 60 mg/dl among the 55–59-year-olds. VLDL-C shows little change with age in the younger 5-year birth cohorts presented. A gradually steeper upward trend of VLDL-C levels with age is seen subsequently, leading to a mean of 21 mg/dl among the 50–59-year-olds.

Lipoprotein-cholesterol levels by age for females taking sex hormone preparations are presented in figure 6. All three lipoprotein-cholesterol fractions show a linear rise with age from ages 20–59 years. LDL-C levels increase from 108 mg/dl to 133 mg/dl, HDL-C from 55 mg/dl to 71 mg/dl, and VLDL-C from 15 mg/dl to 19 mg/dl.

**Comparisons by Age, Sex, and Hormone Use**

In figure 7 cholesterol and triglyceride levels by age are presented for males and for females identified by use of exogenous sex hormones.

The 20–24-year-old males and females not taking sex hormones have comparable mean cholesterol values; the age-dependent increase of plasma cholesterol levels occurs predominantly between ages 20–45 years in males, and in females it takes place also during middle age. Consequently, a crossover of plasma cholesterol levels of males and females takes place at approximately age 50 years.

The cholesterol levels of females taking hormones are higher than those of females not taking sex hormone preparations at all ages below the age stratum 50–54 years. A reversal occurs at this point, and females not on hormones have higher cholesterol levels in the older age groups.

The age distribution of plasma triglyceride levels shows marked differences among the three groups compared. Males and females not on hormones show inconspicuous differences in triglyceride levels at age 20 years; the plasma triglyceride of females follows an almost linear incremental tendency over the age strata.

FIGURE 7. Regression estimates of mean plasma lipid values by age for males, females not taking sex hormones and females taking sex hormone preparations. Lipid Research Clinics Prevalence Study, Visit 2, random sample.

FIGURE 8. Regression estimates of mean plasma lipoprotein-cholesterol values by age for males, females not taking sex hormones and females on sex hormone preparations. Lipid Research Clinics Prevalence Study, Visit 2, random sample.

considered, while the males reflect a more rapid increase of plasma triglyceride levels during young adulthood and a decrease during middle age. Thus, differences in plasma levels of triglyceride between males and females not taking hormones are maximal over the ages 35–44 years and minimal at both extremes of the age range presented. Females on hormone compounds have consistently, and markedly, higher total triglyceride levels than females not taking hormones at all ages.

Figure 8 summarizes the changes of lipoprotein-cholesterol levels by age, contrasting sex and hormone usage subgroups. The LDL-C of males and females not taking hormones increase with age, with the differential slope by sex already described. The crossover of LDL-C levels for the sexes occurs in the age group 50–54 years and leads to higher LDL-C values in females not taking hormones than in males in the age group 55–59 years.

In young females up to the age group 35–39 years, plasma LDL-C is higher among females characterized by use of exogenous sex hormones than in females not taking hormones. A reversal of this pattern is seen in females beyond age 40: higher LDL-C values are now characteristic of females not on hormones. This difference in LDL-C levels between females not on hormones and those taking hormones increases with increasing age.
The plasma levels of HDL-C are seen to be lower in males than in females at all ages between 20–59 years. In all age groups beyond age 30 years, females taking hormones have higher HDL-C levels than females not taking hormones, with an increasing difference over successively older examinees.

VLDL-C in females taking hormones is higher than in females not taking hormones among the LRC participants less than 50 years old. Females in their sixth age decade show little difference in VLDL-C levels by hormone use, and thereafter a reversal seems to take place in that females not on hormones show higher plasma levels of VLDL-C. Males approximate the female values of VLDL-C in the youngest and oldest age strata, and have higher VLDL-C levels in the intermediate age groups.

**Discussion**

The results presented, based on cross-sectional data, indicate that plasma lipid and lipoprotein levels vary with age in males, in females not taking hormones, and to a lesser degree in females who take sex hormones. The patterns of total plasma cholesterol and triglyceride by age, sex, and hormone use replicate the findings published by the LRC Program on visit-1 prevalence study participants. Based on 48,502 examinees, the latter findings provide descriptive data on total plasma lipids in 10 North American communities, but do not contain lipoprotein-fraction measurements. This study provides a comparable baseline of lipoprotein-cholesterol distributions in the 10 North American populations examined by the LRC Population Studies.

That lipoproteins have a role in atherogenesis has been recognized for many years. Recent clinical, laboratory and epidemiologic studies have gathered evidence suggestive of an independent contribution of the plasma lipoprotein fractions to atherogenesis and atherosclerotic cardiovascular disease. Most important, the risk of atherosclerotic heart disease increases with increasing levels of LDL-C, conversely, it decreases with increasing levels of HDL-C.

At a time of increased interest in lipoprotein metabolism it is relevant to describe population levels of the lipoprotein-cholesterol fractions, and to examine the determinants of their distributional patterns. Such data can add to comparison between populations, monitoring of secular trends, clinical decision making, and formulation of public-health policies.

The age- and sex-related changes in LDL-C levels documented in this study closely resemble those for total plasma cholesterol. This is not surprising, because almost 70% of total cholesterol is transported by lipoprotein in the ultracentrifugal low-density range. The male-female differences, however, are of greater magnitude for LDL-C.

The well-known higher HDL-C levels in females compared with males can be observed across the

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**Table 2. Multiple Linear Regression Analysis of Plasma Lipids and Lipoprotein-Cholesterol Fractions on Age, by Sex and Use of Exogenous Sex Hormones. Lipid Research Clinics Visit-2 Random Sample, Ages 20–59 Years**

<table>
<thead>
<tr>
<th></th>
<th>Intercept</th>
<th>Age (years)</th>
<th>Age²</th>
<th>Multiple R²</th>
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<tbody>
<tr>
<td><strong>Males (n = 2458)</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>71.0</td>
<td>5.17</td>
<td>-0.60</td>
<td>0.17</td>
<td>16.84</td>
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<tr>
<td>Triglyceride</td>
<td>65.9</td>
<td>9.24</td>
<td>-0.007</td>
<td>0.05</td>
<td>4.59</td>
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<tr>
<td>LDL cholesterol</td>
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<td>4.12</td>
<td>-0.042</td>
<td>0.14</td>
<td>13.30</td>
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<tr>
<td>HDL cholesterol</td>
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<td>0.23</td>
<td>-0.015</td>
<td>0.08</td>
<td>5.57</td>
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<tr>
<td>VLDL cholesterol</td>
<td>21.9</td>
<td>2.03</td>
<td>-0.022</td>
<td>0.04</td>
<td>3.66</td>
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<tr>
<td><strong>Females not taking hormones (n = 1652)</strong></td>
<td></td>
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<tr>
<td>Cholesterol</td>
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<td>0.32</td>
<td>0.024</td>
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<td>Triglyceride</td>
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<td>1.42</td>
<td>0.006</td>
<td>0.16</td>
<td>10.5</td>
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<tr>
<td>LDL cholesterol</td>
<td>75.7</td>
<td>0.61</td>
<td>0.014</td>
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<tr>
<td>HDL cholesterol</td>
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<td>0.10</td>
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<td>VLDL cholesterol</td>
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<td>0.61</td>
<td>0.012</td>
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<td><strong>Females taking hormones (n = 648)</strong></td>
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<tr>
<td>Cholesterol</td>
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<td>Triglyceride</td>
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<td>LDL cholesterol</td>
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</tr>
<tr>
<td>VLDL cholesterol</td>
<td>8.6</td>
<td>0.17</td>
<td></td>
<td>0.10</td>
<td>3.6</td>
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</tbody>
</table>

*Shown are coefficients with partial F of p < 0.05.
full age span covered in this report. Mean HDL-C values greater than 55 mg/dl are seen in most age strata of the females, contrasted to values of 45 mg/dl in age-comparable males. The HDL-C differences by sex increase over successive birth cohorts through middle age, by virtue of a small linear increment of HDL with age in females, while no changes with age are detectable on HDL-C values in males until the sixth age decade.

Exogenous estrogen increases HDL-C concentrations in women and men, and progestins appear to modify the response to estrogen and either decrease HDL-C concentrations or increase the heavier HDL₃ subclass. In contrast to estrogen, androgens appear to decrease HDL-C levels. Between 50–59 years of age, males have a tendency for HDL-C to increase with age. This characteristic has not been described before in population studies. On a speculative basis, age-related changes in gonadal hormone levels and selective survival may explain this phenomenon.

Women taking hormones compared with women not taking hormones have higher LDL-C levels between ages 20–40 years, higher HDL-C between ages 30–59 years, and higher VLDL-C between the ages 20–55 years. The differential effects of hormone usage on the lipoprotein-cholesterol fractions by age presumably reflect age-related changes in the type of exogenous sex hormone used. Sex hormones taken before menopause are likely to be oral contraceptive preparations, while estrogen replacement will be represented predominantly among the postmenopausal hormone users. The changes in lipoprotein-cholesterol levels associated with oral contraceptive and estrogen use in the LRC populations are presented in detail by Wallace et al.

Several population-based studies on lipoprotein distributions have been reported, although from a less extensive data base than the LRC Prevalence Study. A small sample of Swedish men and women was examined by Carlson and Ericsson to ascertain referent control values for their study on lipoproteins in coronary heart disease patients. Bang et al. studied lipid and lipoprotein subfraction levels in: 1) Eskimos in Greenland, 2) Eskimos in Denmark and 3) Caucasians in Denmark, and found differences by place of residence and by ethnicity. Tyrolean and co-workers described systematic differences between black and white adult residents of Evans County, Georgia. Similar black-white differences in children ages 5–14 years were noted by Srinivasan et al. in the Bogalusa Louisiana Study. Stanhope et al. described lower serum HDL-C levels in Maori compared with non-Maori New Zealand adolescents. They found that boys had lower HDL-C levels than girls.

Slack and co-workers reported on serum cholesterol and triglyceride and electrophoretically determined lipoproteins in residents of northwest London. The age trends by sex of total serum cholesterol and triglyceride presented by Slack et al. are similar to those we describe. The measurements reported by these authors for serum lipoprotein concentrations are also similar; however, the absolute values are not directly comparable because of differences in methods of lipoprotein quantification.

The age trends in LDL-C observed in the Cooperative Lipoprotein Phenotyping Study varied considerably from one population to another. No well-defined age trends were observed for HDL-C in the men and women ages 40 years and older. Interlaboratory variability may be partially responsible for these differences.

The Four City Study compared serum lipid and lipoprotein levels in four apparently healthy European populations ages 20–69 years. The sampling frames considered working populations in Geneva, London and Naples; in Uppsala a random sample was drawn from the population register. Considerable interpopulation differences were found in total serum lipid levels, with parallel trends for total cholesterol and triglycerides. Differences between the four populations in the total serum lipids were mostly accounted for by differences in LDL-C and VLDL-C concentrations. Little or no interpopulation differences in HDL-C were apparent.

Similarly, the 10 study populations surveyed by the North American LRCs reveal differences in their mean and median levels of total plasma lipids and lipoprotein-cholesterol fractions. These differences are statistically significant and are maintained as parallel trends across the age groups examined. The rigorous quality control of the LRC protocol makes it unlikely that interlaboratory variability is a source of this diversity among populations. The characteristics of the LRC study populations are being examined to ascertain the determinants of lipid and lipoprotein levels and the observed differences among population groups.

The difficulties in choosing normal cutoff values for use in clinical and public-health practice are well known. A corollary to the cross-sectional trends in total plasma lipids and lipoprotein-cholesterol concentrations presented in this report is the necessity to take into account differences by age, sex and idiosyncratic population characteristics when conventional limits are used.

Several factors other than sex, age and ethnicity have been found to influence the levels of lipoprotein-cholesterol fractions in human groups, although few of these associations are convincingly established. No attempt is made in this study to report on the correlates of the population distributions of lipid and lipoprotein-cholesterol fractions presented. Instead, this report represents a descriptive reference document for the forthcoming analytic studies of the LRC Population Studies.

References

2. Gofman JW, Lindgren FT, Elliott H: Ultracentrifugal studies
### APPENDIX 1

#### TABLE A1. Mean and Percentile Values of Plasma Cholesterol and Triglyceride (mg/dl) by Age—White Males 20–59 Years, Visit 2 Random Sample

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Missing values: cholesterol = 5; triglyceride = 1; inconsistent reporting of gonadal hormone use = 1.

Minimum cell size for display of percentiles: 5th and 95th ≥ 100; 10th and 90th ≥ 75; 50th ≥ 40.

#### TABLE A2. Mean and Percentile Values of Plasma Cholesterol and Triglyceride (mg/dl) by Age—White Females 20–59 Years, No Hormones*, Visit-2 Random Sample

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Missing values: cholesterol = 6; triglyceride = 1.

Minimum cell size for display of percentiles: 5th and 95th ≥ 100; 10th and 90th ≥ 75; 50th ≥ 40.

*Defined as congruent reporting of gonadal hormone usage during the previous 2 weeks.
TABLE A3. Mean and Percentile Values of Plasma Cholesterol and Triglyceride (mg/dl) by Age—White Females 20–59 Years, on Hormones*, Visit-2 Random Sample

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Missing values: cholesterol = 2; triglyceride = 1.
Minimum cell size for display of percentiles: 5th and 95th ≥ 100; 10th and 90th ≥ 75; 50th ≥ 40.
*Defined as congruent reporting of gonadal hormone usage during the previous 2 weeks.

TABLE A4. Mean and Percentile Values of LDL, HDL, and VLDL Cholesterol (mg/dl) by Age—White Males 20–59 Years, Visit-2 Random Sample

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</table>

Missing values: LDL = 4; HDL = 6; VLDL = 3; inconsistent reporting of gonadal hormone use = 1. Minimum cell size for display of percentiles: 5th and 95th ≥ 100; 10th and 90th ≥ 75; 50th ≥ 40.

### TABLE A5. Mean and Percentile Values of LDL, HDL, and VLDL Cholesterol (mg/dl) by Age—White Females 20–59 Years, No Hormones*, Visit-2 Random Sample

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>n</th>
<th>Mean</th>
<th>SEM</th>
<th>Percentiles</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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<td>5</td>
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<tr>
<td><strong>LDL cholesterol</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>20-24</td>
<td>96</td>
<td>98.1</td>
<td>2.8</td>
<td>—</td>
</tr>
<tr>
<td>25-29</td>
<td>181</td>
<td>106.0</td>
<td>2.0</td>
<td>70</td>
</tr>
<tr>
<td>30-34</td>
<td>232</td>
<td>108.9</td>
<td>1.7</td>
<td>68</td>
</tr>
<tr>
<td>35-39</td>
<td>249</td>
<td>118.8</td>
<td>2.1</td>
<td>76</td>
</tr>
<tr>
<td>40-44</td>
<td>254</td>
<td>125.1</td>
<td>1.9</td>
<td>76</td>
</tr>
<tr>
<td>45-49</td>
<td>255</td>
<td>130.0</td>
<td>2.1</td>
<td>81</td>
</tr>
<tr>
<td>50-54</td>
<td>186</td>
<td>145.0</td>
<td>2.8</td>
<td>90</td>
</tr>
<tr>
<td>55-59</td>
<td>189</td>
<td>150.4</td>
<td>2.7</td>
<td>93</td>
</tr>
<tr>
<td>Total</td>
<td>1642</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| HDL cholesterol | | | | | | | | |
| 20-24          | 96 | 52.2  | 1.5 | — | 37 | 50 | 68 | — |
| 25-29          | 181 | 55.9  | 1.0 | 37 | 39 | 55 | 73 | 81 |
| 30-34          | 233 | 55.4  | 0.8 | 38 | 40 | 55 | 71 | 75 |
| 35-39          | 248 | 54.5  | 0.9 | 34 | 38 | 52 | 74 | 82 |
| 40-44          | 254 | 57.0  | 1.0 | 34 | 39 | 55 | 78 | 87 |
| 45-49          | 257 | 57.7  | 1.1 | 33 | 39 | 56 | 78 | 86 |
| 50-54          | 186 | 60.0  | 1.1 | 37 | 40 | 58 | 77 | 89 |
| 55-59          | 189 | 59.5  | 1.2 | 36 | 39 | 58 | 82 | 86 |
| Total          | 1644|       |     |   |    |    |    |    |

| VLDL cholesterol | | | | | | | | |
| 20-24           | 96 | 11.9  | 0.7 | — | 3  | 10 | 22 | — |
| 25-29           | 181 | 12.0  | 0.6 | 2  | 4  | 11 | 22 | 24 |
| 30-34           | 233 | 10.5  | 0.5 | 0  | 2  | 9  | 19 | 24 |
| 35-39           | 247 | 14.4  | 0.7 | 1  | 3  | 13 | 26 | 35 |
| 40-44           | 254 | 14.1  | 0.5 | 3  | 5  | 12 | 26 | 29 |
| 45-49           | 255 | 16.9  | 0.8 | 2  | 4  | 14 | 32 | 40 |
| 50-54           | 186 | 16.6  | 1.2 | 0  | 4  | 14 | 33 | 37 |
| 55-59           | 189 | 21.3  | 0.9 | 2  | 4  | 18 | 39 | 49 |
| Total           | 1641|       |     |   |    |    |    |    |

Missing values: LDL = 10; HDL = 8; VLDL = 11. Minimum cell size for display of percentiles: 5th and 95th ≥ 100; 10th and 90th ≥ 75; 50th ≥ 40. *Defined as congruent reporting of gonadal hormone usage during the previous 2 weeks.
### Table A6. Mean and Percentile Values of LDL, HDL, and VLDL Cholesterol (mg/dl) by Age-White Females 20–59 Years, on Hormones*, Visit-2 Random Sample

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>n</th>
<th>Mean</th>
<th>SEM</th>
<th>5</th>
<th>10</th>
<th>50</th>
<th>90</th>
<th>95</th>
</tr>
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<td>LDL cholesterol</td>
<td></td>
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<td></td>
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<tr>
<td>20–24</td>
<td>101</td>
<td>108.3</td>
<td>3.3</td>
<td>62</td>
<td>66</td>
<td>106</td>
<td>149</td>
<td>163</td>
</tr>
<tr>
<td>25–29</td>
<td>132</td>
<td>115.9</td>
<td>2.5</td>
<td>72</td>
<td>80</td>
<td>111</td>
<td>157</td>
<td>169</td>
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<tr>
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<td>95</td>
<td>117.4</td>
<td>3.1</td>
<td>—</td>
<td>—</td>
<td>110</td>
<td>155</td>
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<td>5.4</td>
<td>—</td>
<td>—</td>
<td>114</td>
<td>—</td>
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<td>63</td>
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<td>—</td>
<td>124</td>
<td>—</td>
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<tr>
<td>45–49</td>
<td>71</td>
<td>127.3</td>
<td>4.0</td>
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<td>—</td>
<td>127</td>
<td>—</td>
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<td>HDL cholesterol</td>
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<td>53</td>
<td>74</td>
<td>79</td>
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<td>38</td>
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<td>75</td>
<td>83</td>
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<td>57</td>
<td>77</td>
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<tr>
<td>Total</td>
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<td>VLDL cholesterol</td>
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<td>1.1</td>
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<td>17</td>
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<tr>
<td>Total</td>
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<td></td>
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<td></td>
</tr>
</tbody>
</table>

Missing values: LDL = 4; HDL = 2; VLDL = 3.
Minimum cell size for display of percentiles: 5th and 95th > 100; 10th and 90th > 75; 50th > 40.
*Defined as congruent reporting of gonadal hormone usage during the previous 2 weeks.

### Appendix 2

**Lipid Research Clinics**

**North American Clinics**

- Baylor College of Medicine, Houston, Texas
  - Director: William Insull, M.D.
  - Past Director: Antonio Gotto, M.D.
- *George Washington University, Washington, D.C.
  - Director: John LaRosa, M.D.
- Johns Hopkins University, Baltimore, Maryland
  - Director: Peter Kwiterovich, M.D.
- Oklahoma Medical Research Foundation, Oklahoma City, Oklahoma
  - Director: Reagan Bradford, M.D.
- Stanford University, Palo Alto, California
  - Director: John Farquhar, M.D.
- University of California at San Diego, La Jolla, California
  - Director: Fred Mattson, Ph.D.
  - Co-director: Daniel Steinberg, M.D.
  - Past Director: Virgil Brown, M.D.
- University of Cincinnati, Cincinnati, Ohio
  - Director: Charles Glueck, M.D.
- University of Minnesota, Minneapolis, Minnesota
  - Director: Ivan D. Frantz, Jr., M.D.
- University of Toronto/McMaster University at Hamilton, Ontario, Canada
  - Director: Francois Abboud, M.D.
  - Past Director: William Connor, M.D.
PLASMA LIPOPROTEIN-CHOLESTEROL DISTRIBUTIONS/Heiss et al.

Director: Alick Little, M.D.
Co-Directors: Maurice Mishkel, M.D. and George Steiner, M.D.
University of Washington, Seattle, Washington
Director: Robert Knopp, M.D.
Past Directors: Edward Bierman, M.D., William Hazzard, M.D.

*Washington University, St. Louis, Missouri
Director: Gustav Schonfeld, M.D.
Past Director: Robert Shank, M.D.

USSR Clinics
All Union Cardiologic Institute, Moscow
Director: Elena Gerasimova, M.D.
Institute of Experimental Medicine, Leningrad
Director: Anatoli Klimov, M.D.

Israeli Clinic
Hadassah Medical School and Hebrew University, Jerusalem
Director: Yechezkiel Stein, M.D.

Support Agencies
Central Clinical Chemistry Laboratory
Bio-Science Laboratories, Van Nuys, California
Director: Frank Ibbott, Ph.D.
Past Director: Donald Wybenga, B.M.

Central Electrocardiographic Laboratory
University of Alabama, Birmingham, Alabama
Director: L. Thomas Sheffield, M.D.

Central Patient Registry and Coordinating Center
University of North Carolina, Chapel Hill, North Carolina
Director: O. Dale Williams, Ph.D.
Past Director: James E. Grizzle, Ph.D.

*Drug Supply and Distribution Center
Mead Johnson, Evansville, Indiana
Director: John Boenigk, Ph.D.

Lipid Standardization Laboratory
Center for Disease Control, Atlanta, Georgia
Director: Gerald Cooper, M.D., Ph.D.

Nutrition Coding Center
University of Minnesota, Minneapolis, Minnesota
Director: P. Victor Grambsch

Program Office
Lipid Metabolism Branch, National Heart, Lung, and Blood Institute
Chief: Basil Rifkind, M.D.
Past Chief: Robert Levy, M.D.

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Gerasimova, M.D.; Igor Glasunov, M.D.; Gaetan Godin, M.S.;
S. T. Halfon, M.D.; Robin Harris, M.P.H.; William Haskell,
Ph.D.; Gerardo Heiss, M.D., Ph.D.; David Hewitt, M.A.; Judith
Hill, M.S.; Joanne Hoover, M.D.; David Jacobs, Ph.D.; Kathe
Kelly, M.S.; Anatoli Klimov, M.D.; Alick Little, M.D.; Arden
Mackenthun, Ph.D.; Irma Mebane, M.S.; Richard Mowery,
M.S.P.H.; John Morrison, Ph.D.; Basil Rifkind, M.D.; Dimitri
Shestov, M.D.; Israel Tamir, M.D.; Henry Taylor, Ph.D.; Pearl
Van Natta, Ph.D.; Robert Wallace, M.D.; O. Dale Williams,
Ph.D.; Andrei Zadoja, M.D.

*Did not participate in the LRC Prevalence Study.
Lipoprotein-cholesterol distributions in selected North American populations: the lipid research clinics program prevalence study.
G Heiss, I Tamir, C E Davis, H A Tyrold, B M Rifkand, G Schonfeld, D Jacobs and I D Frantz, Jr

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