Plasma Lipid Distributions in Selected North American Populations: The Lipid Research Clinics Program Prevalence Study

THE LIPID RESEARCH CLINICS PROGRAM EPIDEMIOLOGY COMMITTEE

SUMMARY  Cross-sectional age- and sex-specific plasma lipid distributions (means, medians and selected percentiles) are given for 48,431 white participants in visit 1 of the Lipid Research Clinics (LRC) Prevalence Study. This study consisted of two visits in which 10 LRCs screened participants selected from well-defined North American target populations that included a broad range of sociodemographic subgroups.

These data confirm findings from earlier studies in developed countries, showing age-related differences in plasma lipid levels. However, for overall distributions, the LRC data showed slightly lower cholesterol and markedly higher triglyceride values than those previously reported for North America. Some variation in plasma lipid values was evident among the clinic populations.

The large number of participants within most subgroups permitted a variety of analytic and comparative studies. For example, data from the large pediatric population revealed a drop in plasma cholesterol levels in adolescent males and females. Males aged 20–50 years had higher cholesterol levels than females in the same age group, and higher triglyceride levels between ages 20–70 years. Numbers were also sufficient for meaningful comparisons between lipid distributions of females who were taking sex hormones and those who were not: In females taking sex hormones, cholesterol and triglyceride levels were higher for subjects younger than 45 years, but slightly lower after age 45, than lipid levels in females not taking hormones.

AN ASSOCIATION between serum cholesterol and coronary heart disease (CHD) is well established. It is based on a variety of evidence including: the cholesterol content and dynamics of the human atherosclerotic plaque; the production of atherosclerotic lesions in animals by regimens inducing hypercholesterolemia and the regression of these lesions during low-cholesterol regimens; the high prevalence of premature vascular disease in certain genetic hypercholesterolemias; the occurrence of hypercholesterolemia in subjects with clinically manifested atherosclerotic disease, especially in the younger age groups; and epidemiological studies of populations in a variety of settings. Of special importance is the observation, through prospective epidemiological studies, that total serum cholesterol concentration is an important predictor of CHD in North American adults. Although parallel studies have focused on serum triglyceride levels as an independent risk factor, the evidence has been contradictory and inconclusive.

The need to translate hyperlipidemia into hyperlipoproteinemia was advocated by Gofman in the early 1950s, and reemphasized by Frederickson, Levy, and Lees in 1967. These concepts led to the development of a system for classifying hyperlipoproteinemias into five (later six) types of patterns. This system has been widely adopted as a basis for characterizing lipoprotein disorders. Additional support for this view has recently come from observations that high-density lipoprotein (HDL) levels appear to be inversely and independently related to CHD risk.

Hyperlipoproteinemias are an important public health concern because they are frequently associated with CHD, especially in young persons. Many researchers have attempted to measure the prevalence and the extent of the relationship of hyperlipoproteinemias with CHD. However, few such studies have been population based or have involved sufficient numbers; therefore, a better understanding of the overall contribution of hyperlipoproteinemias to CHD is still needed. Moreover, an adequate interpretation of the significance of hyperlipoproteinemia requires a description of the distribution of lipids and lipoproteins within populations.

With these and related issues in mind, the National Heart and Lung Institute (NHLI) organized a Task Force in 1970 to develop a long-range plan to combat arteriosclerosis that would use existing knowledge and pinpoint research areas that require further study. The Task Force Panel on Hyperlipidemia and Premature Atherosclerosis was charged with recommending ways of preventing premature atherosclerosis through the diagnosis and treatment of hyperlipidemia. The panel reviewed research of the Molecular Disease Branch of the NHLI and elsewhere, which indicated that lipoprotein patterns could provide important information not provided by blood cholesterol and triglyceride levels alone. The identification of distinct lipid transport disorders, "hyperlipoproteinemias," which had formerly been grouped under the general heading of familial hyperlipidemia or hypercholesterolemia,

Appendix 1 is a list of principal investigators and key personnel for the study.


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offered a more systematic approach to the study and treatment of these conditions.

Among the recommendations of the Task Force was the creation of the Lipid Research Clinics (LRC) Program, as a logical extension of this research with the objective of improving the detection and clinical management of the hyperlipoproteinemias. Accordingly, 12 LRCs and five support facilities were established in the United States and Canada with three primary objectives:

1) to acquire data on the prevalence of different types of hyperlipoproteinemia in various age and ethnic groups, with special emphasis on the nature and frequency of genetic forms;

2) to collect reliable data on the prevalence and incidence of atherosclerosis in different types of hyperlipoproteinemia; and

3) to conduct an intervention trial to determine if lowering plasma lipid levels would reduce the risk of CHD.

To meet these objectives, two major sets of collaborative studies were undertaken: the Population Studies and the Coronary Primary Prevention Trial. In this paper we describe the Population Studies component of the LRC Program and report our findings on the distributions of plasma total cholesterol and triglyceride in selected US and Canadian populations.

LRC Population Studies

The Population Studies encompass three major studies sharing the same general population base. The Prevalence Study focuses on the distribution of lipids and lipoproteins and their association with diet and coronary heart disease. It consisted of two screening visits in which each of the 10 participating North American LRCs evaluated subjects selected from well-defined populations. The Prevalence Study was expanded in 1972 as a result of the establishment of the US-USSR Joint Program in Cardiovascular Disease. Clinics were set up by the USSR government in Moscow and Leningrad, and their activities were closely integrated into the LRC program. In addition, in 1975 a LRC was established in Jerusalem, Israel.

The Family Study, started in 1975 and completed in 1978, was developed to measure the familial and genetic attributes of plasma lipids and lipoproteins. A subsample of those participating in the second Prevalence Study screen, including both normolipidemic and hyperlipidemic subjects, was chosen to participate as probands. In the Family Study, basic demographic information was collected and plasma lipids and lipoproteins were measured in all first-degree relatives and spouses of the probands. Nine of the North American LRCs participated in the Family Study (Baylor did not participate).

The Follow-up Study, initiated in mid-1977, is a prospective study that seeks to complement the cross-sectional observations made in the Prevalence Study. The follow-up study will determine the subsequent mortality experience of approximately 9000 males and females who were at least 30 years old at the time of their participation in the second Prevalence Study screen.

Methods

The North American Prevalence Study consisted of a series of studies conducted according to a standardized protocol. Subjects were selected according to approved procedures from well-defined target populations. Each study provided an independent contribution to the epidemiologic data base; none was required to be statistically representative of the entire North American population. The selected populations were deliberately diverse, covering a broad range of geographic, socioeconomic, occupational, age, sex, and ethnic groups.

The sampling strategies used for the populations were grouped into broad categories as follows:

School Children and their Parents

Baylor University: sophomore high school students in the Houston Independent School District between 1971–1975, and parents of students in high socioeconomic groups.


Households

University of Iowa: residents of Cedar County, Iowa and selected rural townships of neighboring counties as of July 1, 1973.

Johns Hopkins University: 1) East Baltimore, a random sample of residents of 11 census tracts of East Baltimore, Maryland, as of January 1, 1972; 2) Columbia: members of the Columbia Medical Plan in Columbia, Maryland.

University of California at San Diego (La Jolla): residents as of August 2, 1972 of Rancho Bernardo, California, ages 12 years and older.

University of Minnesota: residents of four census tracts, ages 10–59 years, of Richfield, Minnesota, a suburb of Minneapolis.

Oklahoma Medical Research Foundation: residents of four rural Oklahoma counties, age 19 years and older.

Occupational and Industrial Groups

University of Washington (Seattle): employees of Pacific Northwest Bell Telephone Company, ages 20–65 years, working in King County, Washington.

Stanford University: Stanford University employees, ages 26–70 years.

University of Toronto and McMaster University: employees of four firms: Canadian Bell Telephone Company, Dofasco Steel Foundry, Simpsons Department Store and Eatons Department Store, plus family members of 500 of these employees.
The Screening Visits — Visit 1 and Visit 2

The Prevalence Study involved two sequential examinations. The first, visit 1, was a brief screen to collect information on some sociodemographic variables and on utilization of five types of lipid-altering medication, and to measure fasting plasma cholesterol and triglyceride levels. Subjects who were part of a 15% randomly selected sample, or who had elevated lipid levels, or were taking lipid-altering medication, were asked to return for visit 2, a more extensive examination. At visit 2 we collected medical and family histories, including a detailed drug history, blood pressure measurements, lipid and lipoprotein determinations, nutrient intake evaluation by means of a 24-hour dietary recall, resting and exercise ECGs, clinical chemistries and anthropometric measurements. Those selected for visit 2 included approximately 25% of all subjects screened at visit 1.

Standardization of Procedures

The extensive standardization of all collaborative activities within the LRC Program was made possible by such basic features as a common protocol12 with detailed documentation of procedures, training and certification of all data collection personnel, and regular monitoring of data collection and quality. Edit-and-error correction procedures were instituted both at the clinics and the Central Patient Registry and Coordinating Center. A formal procedure designed to enhance data quality was executed by each clinic after the screening process was completed.

Plasma Lipid Determinations

Blood specimens, obtained by venipuncture, were taken from subjects who had been instructed to fast for at least 12 hours. Venipuncture was done with the participants in a sitting position; a tourniquet was used but was released before sampling to avoid significant increases in the concentration of plasma lipids. All samples were cooled immediately on wet ice, and within 3 hours after venipuncture the standardized processing procedures were initiated.13 Plasma cholesterol and triglyceride levels were determined in each clinic's core lipid laboratory with either the Technicon AutoAnalyzer I (AA-I) or AutoAnalyzer II (AA-II) analytical systems adapted for the program. The total cholesterol procedure adapted to the AA-I was the ferric chloride-sulfuric acid method; the total cholesterol procedure adapted to the AA-II was the Liebermann-Burchard procedure. Triglyceride was analyzed fluorimetrically for all samples, the same method of analysis being used with both instruments.13,14 Five laboratories — Baylor, Johns Hopkins, La Jolla, Seattle, and Stanford — used the AA-I system for all their Prevalence Study determinations. The other clinics used the AA-II system. (All clinics used the AA-II system for plasma lipid determinations for the Coronary Primary Prevention Trial.)

Lipid Laboratory Quality Control

A special emphasis on laboratory quality control was dictated by two features of the LRC Prevalence Study: the long period of time over which the tests were made and the fact that each clinic laboratory did the determinations for all samples collected by that clinic. Under these conditions, controlling both long-term drift or stability and interlaboratory variation were crucial. To accomplish this, identical systems of internal quality control and external surveillance were used in each laboratory.15-18 These included calibration of instruments with common solutions of cholesterol and triolein primary standards, and daily quality control assessment by the use of common serum pools of known cholesterol and triglyceride concentrations. The laboratories established control limits that were used to evaluate each day's analyses. Results obtained from "out-of-control" analyses were rejected, and those analyses were repeated. The cholesterol concentrations of the common serum pools were assigned by the Lipid Standardization Laboratory, by use of a reference Abell-Kendall method, and the triglyceride concentrations were assigned by use of a chromotrope acid colorimetric method. These samples were used to assess both within-run variability and overall precision.

Comparability of the AA-I and AA-II Systems

Because some of the Prevalence Study lipid determinations were done with the AA-I before the program's conversion to the AA-II system, the comparability of these two systems is an important issue for the Prevalence Study. On the average, for frozen serum pools the AA-I instruments tended to produce cholesterol values 2.1% higher than the manual Abell-Kendall target values. In contrast, the AA-II instruments tended to give values 1.3% lower than target values. However, all study determinations were done on fresh samples and the comparative data on fresh samples indicate differences even less than the values mentioned.

On quality control samples, the AA-II instruments showed excellent precision for cholesterol within runs, between runs and among instruments, as reported earlier.19 For analyses from common pools, the total standard deviation was small in AA-II instruments (3-4 mg/dl), with most of the variability due not to differences among instruments, but to variations within a single run on a single machine.15 Although the AA-I instruments did not appear quite as precise as the AA-IIs, the total standard deviation was still quite small, and the variability among instruments was similar to the run-to-run variability within a single instrument.

The standard deviations and accuracy of triglyceride determinations were similar for the two types of instruments on the frozen pools. As was true for cholesterol, most of the variability did not arise from differences between instruments, but from variation from sample to sample within a single laboratory.18
### Table 1. Number of Eligible and Screened for Visit 1 and Visit 2 of the LRC Program Prevalence Study, for Each Clinic Population, LRC Program, February 1978

<table>
<thead>
<tr>
<th>Sampling frame/clinic</th>
<th>Number Eligible</th>
<th>Screened</th>
<th>Response rate (%)</th>
<th>In analysis</th>
<th>Number Eligible</th>
<th>Screened</th>
<th>Response rate (%)</th>
</tr>
</thead>
<tbody>
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<td>Central city adults and their parents</td>
<td>Baylor</td>
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<td>3,795</td>
<td>64</td>
<td>2,295</td>
<td>902</td>
<td>637</td>
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<tr>
<td></td>
<td>Cincinnati</td>
<td>15,571</td>
<td>10,893</td>
<td>70</td>
<td>7,337</td>
<td>3,049</td>
<td>2,652</td>
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<td>Iowa</td>
<td>5,594</td>
<td>3,918</td>
<td>70</td>
<td>3,635</td>
<td>1,099</td>
<td>999</td>
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<td></td>
<td>Johns Hopkins</td>
<td>5,075</td>
<td>4,393</td>
<td>87</td>
<td>3,012</td>
<td>1,145</td>
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<td></td>
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<td>78</td>
<td>5,491</td>
<td>1,644</td>
<td>1,459</td>
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<td>5,966</td>
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<td></td>
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<td>Occupational and industrial groups</td>
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<td>83</td>
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<td></td>
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<td>4,280</td>
<td>71</td>
<td>3,426</td>
<td>1,189</td>
<td>1,074</td>
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<tr>
<td></td>
<td>Toronto-McMaster</td>
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<td>69</td>
<td>7,041</td>
<td>2,182</td>
<td>1,781</td>
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<td>Total</td>
<td>81,926</td>
<td>60,502</td>
<td>74</td>
<td>48,431</td>
<td>16,335</td>
<td>13,852</td>
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### Results

The data presented here are from the visit 1 screening for the 10 North American LRCs participating in the Prevalence Study. Screening for these visits was completed in 1976. General information about the response rates and the number of participants screened and included in this discussion is presented in table 1.

This report is intended to characterize the fasting plasma lipid levels of white LRC examinees of both sexes. Hence, 5945 nonwhite participants in the LRC visit 1 examination (table 1) are excluded from these analyses; their plasma lipid values are described in a separate communication.

To enhance measurement validity, comparability to other studies and usefulness of our results to researchers and practitioners alike, the following white examinees were also excluded from these analyses: 1127 participants whose blood specimens were frozen before determination of plasma lipids; 4640 participants who fasted less than 12 hours; 338 pregnant women, and 57 women for whom information on exogenous sex hormone usage was missing or uncertain. Data editing resulted in the exclusion of three other records due to invalid information on sex, race or age.

Consequently, of the 54,557 white participants screened at visit 1, 6126 records were excluded from analyses because of one or a combination of the exclusion criteria.

![Figure 1. Plasma cholesterol for white males, mean and percentile values for 5-year age groups, LRC Program Prevalence Study Visit 1.](source:LRC Visit 1 Survey Examination.)
Plasma Lipid Distributions

The plasma lipid distributions are presented in two formats. Figures 1 through 6 are plots of the means, medians, fifth and ninety-fifth empirical percentiles with points plotted for 5-year age groups. Plots are included for both cholesterol and triglyceride values for white males, white females not taking sex hormones, and white females taking sex hormones. The data for females have been separated according to whether they were taking hormones because of the previously demonstrated large impact of sex hormone usage on
plasma lipid levels. Also appearing on each plot are the highest and lowest clinic-specific medians for 10-year age groups. These values are included to show some of the interclinic variations in lipid levels. In addition, tables II-1 through II-6 in Appendix II include numbers of participants, means, standard deviations, and the fifth, tenth, median, ninetieth and ninety-fifth percentiles.

In general, the distributions of fasting plasma cholesterol for each age group tend to be only slightly skewed. Each distribution has a single mode, with the mean and median values fairly similar. In addition, the intervals from the fifth to fiftieth and from the fiftieth to ninety-fifth percentiles tend to be similar. However, the range does increase with increasing levels for the older age groups for both males and females.

In contrast, the total plasma triglyceride distributions are markedly skewed to the right, with large standard deviations relative to the mean, and markedly greater intervals between the fiftieth and ninety-fifth than those between the fifth and fiftieth percentiles. Also, the mean values for triglyceride are consistently (and in many instances markedly) higher than the median values. As with cholesterol, the standard deviations tend to be larger for the older age groups with their higher average levels.

**Plasma Cholesterol for White Males**

In fasting white males (fig. 1 and table II-1), the age-specific plasma cholesterol means closely parallel the medians and increase from 155 mg/dl for the 0-4-year age group to 160 mg/dl at 5-9 years. For ages 15-19 years, the mean is lower (150 mg/dl), then higher again for the older age groups, up to a level of 212-214 mg/dl for ages 45-69 years. There is a slight decrease after age 70 years. The ninety-fifth percentile is relatively constant at about 203 mg/dl through age 14 years, dips slightly in the 15-19-year age group, increases to 270 mg/dl for the age group 35-39 years and then varies between 261-277 mg/dl thereafter.

**Plasma Cholesterol for White Females Not Taking Sex Hormones**

For this group (fig. 2 and table II-2), the mean cholesterol rises from 156 to 164 mg/dl between ages 0-4 and 5-9 years, but falls to 157 mg/dl for the 15-19
APPENDIX 2

TABLE II-1. Plasma Total Cholesterol (mg/dl) for White Males—Lipid Research Clinics Program Prevalence Study, Visit 1

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>n</th>
<th>Mean</th>
<th>sd</th>
<th>5</th>
<th>10</th>
<th>50</th>
<th>90</th>
<th>95</th>
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<tr>
<td>0-4</td>
<td>238</td>
<td>154.6</td>
<td>27.2</td>
<td>114</td>
<td>125</td>
<td>151</td>
<td>186</td>
<td>203</td>
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<tr>
<td>5-9</td>
<td>1253</td>
<td>159.9</td>
<td>25.3</td>
<td>121</td>
<td>130</td>
<td>159</td>
<td>191</td>
<td>203</td>
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<tr>
<td>10-14</td>
<td>2278</td>
<td>157.6</td>
<td>25.6</td>
<td>119</td>
<td>127</td>
<td>155</td>
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<tr>
<td>15-19</td>
<td>1980</td>
<td>149.9</td>
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<td>113</td>
<td>120</td>
<td>146</td>
<td>183</td>
<td>197</td>
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<td>20-24</td>
<td>882</td>
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<td>124</td>
<td>130</td>
<td>165</td>
<td>204</td>
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<td>182.2</td>
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<td>133</td>
<td>143</td>
<td>178</td>
<td>227</td>
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<td>30-34</td>
<td>2444</td>
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<td>138</td>
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<td>190</td>
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<td>2428</td>
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<td>158</td>
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<td>42.9</td>
<td>144</td>
<td>150</td>
<td>202</td>
<td>263</td>
<td>275</td>
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</table>

Total 24,425

year age group. Thereafter, it increases regularly through ages 55-59 years to 231 mg/dl, remains generally at that level through age 79 years and declines slightly to 222 mg/dl after age 80 years. The ninety-fifth percentile varies between 200-205 mg/dl through age 19 years, and then rises regularly, reaching 300 mg/dl for the 55-59-year age group and remains at about that level through age 79 years. After age 80 years, the ninety-fifth percentile drops to 280 mg/dl.

TABLE II-2. Plasma Total Cholesterol (mg/dl) for White Females Not Taking Sex Hormones—Lipid Research Clinics Program Prevalence Study, Visit 1

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>n</th>
<th>Mean</th>
<th>sd</th>
<th>5</th>
<th>10</th>
<th>50</th>
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<td>172</td>
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<td>39.9</td>
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<td>228</td>
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<td>75-79</td>
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<td>229</td>
<td>282</td>
<td>291</td>
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<tr>
<td>80+</td>
<td>130</td>
<td>222.1</td>
<td>37.3</td>
<td>165</td>
<td>175</td>
<td>220</td>
<td>271</td>
<td>279</td>
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Total 18,108
### TABLE II-4. Plasma Total Cholesterol (mg/dl) for White Males—Lipid Research Clinics Program Prevalence Study, Visit 1

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<th>Age (years)</th>
<th>n</th>
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<th>sd</th>
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<th>Percentiles 10</th>
<th>Percentiles 50</th>
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<th>Percentiles 95</th>
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<td>59</td>
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<td>15-19</td>
<td>1980</td>
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<tr>
<td>20-24</td>
<td>882</td>
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<td>86</td>
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<td>201</td>
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<td>25-29</td>
<td>2042</td>
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<td>104.3</td>
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<td>104</td>
<td>213</td>
<td>266</td>
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<td>2138</td>
<td>151.8</td>
<td>117.8</td>
<td>58</td>
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<td>124</td>
<td>250</td>
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<td>136.7</td>
<td>142.2</td>
<td>57</td>
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<td>208</td>
<td>267</td>
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<td>70-74</td>
<td>454</td>
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<td>70.8</td>
<td>57</td>
<td>68</td>
<td>112</td>
<td>217</td>
<td>258</td>
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<tr>
<td>75-79</td>
<td>244</td>
<td>129.1</td>
<td>69.1</td>
<td>59</td>
<td>65</td>
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<td>120.6</td>
<td>55</td>
<td>64</td>
<td>105</td>
<td>182</td>
<td>255</td>
</tr>
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</table>

**Total** 24,425

---

**Plasma Cholesterol for White Females Taking Sex Hormones**

The age-specific mean values for cholesterol for white females taking sex hormones (fig. 3 and table II-3) increase regularly from 169 mg/dl for ages 15-19 years to 218 mg/dl at ages 50-59 years. For ages 60-64 years the mean is 224 mg/dl and then declines to 216 mg/dl at ages 70-79 years. The ninety-fifth percentile for ages 15-19 years is 231 mg/dl, rises to 236 mg/dl at ages 20-29 years, and slowly increases to 258 mg/dl at ages 40-44 years. For ages 45-49 years, there is a sharp increase to 276 mg/dl, then a more gradual increase to 285 mg/dl by ages 60-64 years, followed by a decline to 271 mg/dl for the 70-74-year age group.
Plasma Triglyceride for White Males

For fasting white males (fig. 4 and table II-4), the age-specific plasma triglyceride means rise from 56 mg/dl for ages 0–9 years to a high of 151 mg/dl for ages 40–54 years. For the older age groups, the mean values gradually decline to about 130 mg/dl. Because of the skewed triglyceride distributions, comparable median values are lower. The age-specific medians rise from 51 mg/dl for ages 0–9 years to 124 mg/dl for ages 45–54 years, then decrease to 105 mg/dl at age 80 years. The ninety-fifth percentile rises from approximately 100 mg/dl for ages 0–9 years to a value of 318 mg/dl for ages 45–49 years, and then declines somewhat irregularly to 255–267 mg/dl above age 65 years.

Plasma Triglyceride for White Females Not Taking Sex Hormones

The age-specific mean values (fig. 5 and table II-5) rise slightly but consistently from 60–64 mg/dl for ages 0–9 years to levels of 125–135 mg/dl for ages 55 years and over. Age-specific medians generally parallel the mean values but at a lower level, with the maximum difference being 23 mg/dl for ages 80 years and older. After an initial decline from 112 mg/dl at ages 0–4 years to 105 mg/dl at ages 0–9 years, the ninety-fifth percentile gradually rises to a peak of 262 mg/dl for ages 55–59 years and then declines somewhat thereafter.

Plasma Triglyceride for White Females Taking Sex Hormones

For white females taking sex hormones (fig. 6 and table II-6), the age-specific triglyceride means rise steadily from about 106 mg/dl for ages 15–24 years to 126 mg/dl at ages 35–39 years and remain at 125–130 mg/dl thereafter. The median values parallel those of the means at a lower level, with the largest difference being 20 mg/dl for ages 50–54 years and 65–69 years. The ninety-fifth percentile declines from 199 mg/dl for ages 15–19 years to 176 mg/dl for ages 20–24 years, then increases substantially to a peak of 259 mg/dl at ages 45–49 years, after which it decreases to 224 mg/dl for ages 70–74 years.

Discussion

The LRC data confirm the rise in cholesterol levels among males and females from the teenage years to middle age, a characteristic of findings in developed countries.18 The LRC data also show a substantial rise in triglyceride levels between the late teens and middle age for males. A steady rise also occurs in females, although the levels are consistently much higher in women taking sex hormones.

Males and Females

Our data permit comparisons of lipid distributions between males and females. Such comparisons should allow for the substantial impact of sex hormone usage on lipid distributions. Accordingly, in figure 7, data on females are confined to the group not taking sex hormones. These females have slightly higher median plasma cholesterol levels than males in the first 2 decades, lower levels until about age 50 years, and considerably higher levels thereafter. The median female triglyceride levels are slightly higher until about age 20 years; after that, the male levels are markedly higher.
TABLE II-6. Plasma Triglyceride (mg/dl) for White Females Taking Sex Hormones—Lipid Research Clinics Program Prevalence Study, Visit 1

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>n</th>
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<th>sd</th>
<th>Percentiles</th>
</tr>
</thead>
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<td>17.4</td>
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<td>106.3</td>
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<td>788</td>
<td>105.3</td>
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<td>35-39</td>
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<td>90.9</td>
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<td>50-54</td>
<td>729</td>
<td>130.0</td>
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<td>55-59</td>
<td>577</td>
<td>126.5</td>
<td>65.7</td>
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<td>70-74</td>
<td>95</td>
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<td>55.3</td>
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<td>26</td>
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<td>34.4</td>
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<td>115.9</td>
<td>41.1</td>
<td></td>
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</tbody>
</table>

Total 5,898

until about age 70 years, when the two levels become similar.

The lipid distributions for all females show marked differences between those who take sex hormones and those who do not. The magnitude of this change, when coupled with the observed high percentage of sex hormone usage, indicates that the rate of sex hormone usage may be an important determinant of female lipid levels.17

Pediatric Age

These data, which include some 20,000 participants ages 0-19 years, show a drop in plasma cholesterol levels in the teenage years. This relationship persists when individual populations are examined and when analyses are based on individual years of age rather than the broad age groups used in this paper.18 A similar relationship has been noted in other investigations.18

Comparison with Other Studies

The aggregated LRC cholesterol levels are consistently lower than those found in previous surveys of the US populations. For example, comparisons of the LRC plasma cholesterol levels of adults with the serum cholesterol levels reported by the National

![Figure 7. Plasma cholesterol and triglyceride for white males and females not taking sex hormones, median values for 5-year age groups. LRC Program Prevalence Study Visit 1.](http://circ.ahajournals.org/content/60/2/436/F7.large.jpg)
Center for Health Statistics for 1960–1962 and 1971–1974 (fig. 8) show that LRC values are consistently lower, even when a 3% plasma-serum difference is considered. 20

In contrast, the triglyceride distributions show values that are considerably higher than those generally believed to be present in the North American populations. For example, ninety-fifth percentiles for males ages 30 years and above range between 250–300 mg/dl, in contrast to the values of 150–200 mg/dl commonly used. 7 Whether these differences are a function of laboratory procedures, sampling methods, the aggregation of diverse populations, or actually represent a shift in the population distributions, is not yet known.

The primary purpose of this paper is to provide a baseline of descriptive data, both for the information it provides directly and as a preparation for subsequent publications. The plasma lipid and lipoprotein distributions and related data from the LRC Prevalence Study are significant for several reasons. The wide range of sociodemographic subgroups provides a broad basis for investigating relationships among these groups, with respect to both lipid and lipoprotein levels and to the associations of these factors with other risk factors for CHD.

The large numbers of participants in most subgroups permit such investigations to be unusually detailed. Further, the fact that participants were screened according to common and highly standardized methods facilitates comparisons among the participating study populations, and provides a basis for useful comparisons with other studies. Some aspects, such as the relationship between sex hormone usage and lipid levels, have not been previously investigated on such a large scale. However, certain issues should be considered carefully in evaluating these data.

Variation Among Populations

Each LRC carried out a standardized survey in a population selected on criteria other than medical characteristics; hence, in all but the Johns Hopkins University/Columbia population, membership was independent of health status, medical card usage, or hospitalization. Therefore, each local survey can be referred to a defined, noninstitutionalized population.

For the purpose of this presentation, the results of lipid distributions were aggregated across all participating clinics. The results are not those which would necessarily result from a random sample of the US population.

In fact, there is evidence of considerable interclinic variation, as shown by the highest and lowest clinic-specific median values shown in figures 1–6. The range of the cholesterol median values among the clinics is large and different for different age-race-sex groups; it reaches 18 mg/dl for white males ages 40–49 years and 24 mg/dl for females ages 50–59 years not taking hormones. The range of clinic-specific median values for triglycerides is even greater, being as much as 40 mg/dl for some of the subgroups.

The observed interclinic difference may be attributed in part to sampling variation. It could also be due to clinic differences in the distribution of lipid-influencing factors such as genetic, dietary, behavioral, and comorbidity characteristics. The point to be emphasized is that the summary information, including means, medians and percentiles, does not represent fixed biologic attributes; rather, it is highly influenced by a large set of both known and unknown genetic and environmental factors. Therefore, the aggregated LRC lipid distributions presented here should not be used as fixed reference values, but as the combined results of contemporary surveys of a diverse set of populations. The lipid data are also presented as the baseline for descriptive and analytic epidemiologic LRC studies to be reported subsequently.

Cross-sectional Data

These data are cross-sectional, and the cholesterol and triglyceride values are from samples of birth cohorts drawn from the several populations. Each of these defined populations is a biological unit, with definite limits to the variation that may occur in that population. Therefore, the relationships of lipid levels and age are likely to be a combination of actual changes with age, selective survival or participation, and cohort effects.

Response Rates

More than 60,000 subjects were examined at visit 1, with an overall response rate of 74%; however, there was substantial variation in response rates for each age, race, sex, and clinic subgroup. Although it compares favorably to several large-scale, population-based studies, participation should be taken into account when considering the validity of the results. It is also pertinent that over 90% of all participants
screened had fasted for 12 hours or longer. This report includes data only for those fasting 12 hours or more.

**Bias Considerations**

A possible bias in these data is selective mortality, which is a potential flaw common to all cross-sectional surveys. (If plasma lipid levels are associated with increased mortality, spuriously lower population values of plasma lipids could be obtained in a cross-sectional examination of survivors.) The magnitude of such bias, if present, cannot be readily ascertained.

A second source of potential bias is selective response, if factors associated with response are also determinants of lipid levels. Differential response rates by clinic are shown in table 1. Variations in response by age are discernible within clinics, with lower than average response rates in 20-25-year-olds and in those older than 75 years. In two clinics, response is also influenced by sex. Each of these characteristics is found to influence plasma lipid values in the LRC data and is controlled for in the analysis.

Some degree of bias cannot be ruled out if the response is less than complete, which is the characteristic common to population-based survey research. To estimate the magnitude of this potential bias is, however, speculative, since the attributes of the nonresponders are unmeasured. Short of recalling nonresponders, estimation of possible bias remains inconclusive. While we cannot be certain the plasma lipid levels were the same in examined and nonexamined eligibles, there is no obvious reason to suspect differences in each age, sex and clinic group.

**The Lipid Laboratories**

Measurements of cholesterol and triglyceride levels were made by lipid laboratories located at each LRC. Further, five of the clinics, Baylor, Johns Hopkins, La Jolla, Seattle, and Stanford, which started a year earlier than the others, used the AutoAnalyzer I system. The other clinics used the AutoAnalyzer II system, since it was available by the time they started screening. The AA-II system represented a considerable advance in the speed and ease of operation and in the precision of results. The first five clinics used the AA-I system throughout the study to maintain intraclinc comparability.

**Plasma vs Serum**

The LRC Prevalence Study evaluated plasma cholesterol and triglyceride, in contrast to most previous studies, which used serum. Plasma determinations were used at visit 1 in order to maintain intrastudy comparability, since the measurements of lipoproteins at visit 2 required plasma. A related study indicated that values for serum average about 3% higher than corresponding plasma values. This difference must be noted when LRC results are compared with those of other studies.

**Other LRC Investigations**

The determinants of the distributions of plasma lipids include the composite effect of a variety of environmental and genetic traits. More detailed investigations of the effects of the different individual factors and their interactions are in progress. Specific factors being investigated are behavioral characteristics such as diet and exercise, social characteristics that are reflective of lifestyles, obesity and physique, drug usage, and familial and genetic traits.

**References**

17. Wallace RB, Hoover J, Sandler D, Tyroier HA, Rifkind BM: Altered plasma lipids associated with oral contraceptive or
oestrogen consumption. The Lipid Research Clinic Program. Lancet 2: 11, 1977

Appendix 1

Lipid Research Clinics

North American Clinics

Baylor College of Medicine, Houston, Texas
Director: William Insull, M.D.
Past Director: Antonio Gatto, 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.
Past Directors: Virgil Brown, M.D., Daniel Steinberg, M.D.

University of Cincinnati, Cincinnati, Ohio
Director: Charles Glueck, M.D.

University of Iowa, Iowa City, Iowa
Director: Francois Abboud, M.D.
Past Director: William Conner, M.D.

University of Minnesota, Minneapolis, Minnesota
Director: Ivan D. Frantz, Jr., M.D.

University of Toronto and McMaster University at Toronto and Hamilton, respectively, Ontario, Canada
Director: J. 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 Schönfeld, M.D.
Past Director: Robert Shank, M.D.

USSR Clinics

All-Union Cardiological Research Center, 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.

Past Prevalence Study Coordinators: Gary Fisher, M.D., Barry Greenberg, M.D., Robin Harris, M.P.H.

LRC Epidemiology Committee

Chairperson: H.A. Tyroler, M.D.

*Did not participate in the North American LRC Prevalence Study.

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