Hyperlipidemia in the Pacific Northwest
Bell Telephone Company Health Survey

Part 2. Lipoprotein Lipid Interrelationships

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The relationships of lipoprotein cholesterol and triglyceride among lipoprotein fractions have potential significance for understanding atherogenesis and distinguishing among different classes of hyperlipidemia. We have compared these relationships in normolipidemic, hypercholesterolemic, hypertriglyceridemic, and combined hyperlipidemic participants in the Pacific Northwest Bell Telephone Company Health Survey. The cholesterol/triglyceride ratio in each lipoprotein fraction was moderately higher (1% to 26%) in hypercholesterolemia but significantly lower (20% to 50%) in hypertriglyceridemia, compared to normolipidemia. In combined hyperlipidemia, the very low density lipoprotein ratios were lower than in normolipidemia, but larger than in hypertriglyceridemia. These changes were directionally the same, but differed quantitatively, in both men and women. Correlation coefficients between cholesterol and triglyceride within each fraction varied by gender and sex hormone use. The largest correlations were seen in combined hyperlipidemic men for very low density lipoproteins, normolipidemic men for low density lipoproteins, and combined hyperlipidemic women taking hormones for high density lipoproteins. The correlation of very low and low density lipoprotein cholesterol was generally negative and was strongest for hormone users \((r = -0.81)\) and weakest for nonusers \((r = -0.06)\). Very low density lipoprotein triglyceride and high density lipoprotein cholesterol correlations were generally negative and were strongest in hypertriglyceridemic women not taking hormones \((r = -0.55)\) and weakest in normolipidemic hormone users \((r = -0.10)\). This correlation was positive for hypertriglyceridemic and combined hyperlipidemic hormone users.

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Triglyceride, as well as cholesterol, is an important component of lipoprotein structure, and may be related to lipoprotein pathophysiology and atherogenesis.\(^1\)\(^2\) Proceeding from this rationale, we have reported the distributions of lipoprotein triglyceride and cholesterol and their relationships in a randomly selected sample of employees of the Pacific Northwest Bell Telephone Company in King County, Washington. We found that lipoprotein triglyceride concentrations differ among men, women not using hormones, and women using sex steroid hormones.\(^1\) The ratios of lipoprotein cholesterol and triglyceride were found to be altered by sex hormone use in women\(^2\) and specifically by oral contraceptives differing in chemical structure and relative potencies of their estrogen and progestin constituents.\(^3\) More recently, we have examined the data from hyperlipidemic subjects in our population for lipoprotein lipid differences associated with gender and sex hormone treatment.\(^4\) Three categories of hyperlipidemia were defined by age, sex, and hormone use-specific, population-based reference values of cholesterol and triglyceride. In addition to the expected very low density lipoprotein (VLDL) and low density
lipoprotein (LDL) abnormalities, other differences were observed in lipoprotein lipid concentrations in the hyperlipidemic subjects. These differences varied with gender and sex hormone use and were consistent with a greater atherosclerosis risk in men. Of special interest are comparisons of high density lipoprotein (HDL) cholesterol concentrations in subjects with combined elevations of triglyceride and cholesterol (combined hyperlipidemia) and those described for the familial form of this disorder.\textsuperscript{5,6}

The relationship of triglyceride and cholesterol within and among the lipoprotein fractions has not yet been examined in hyperlipidemic subjects of any defined population of which we are aware. These relationships are important for several reasons: 1) they may help to identify pathophysiologic abnormalities in hyperlipidemic subjects; 2) they may be altered by gender or sex hormone use which may exaggerate or diminish the potential for atherogenesis; 3) they may identify certain similarities between hyperlipidemias defined by population-based reference values, as done here, and qualitatively similar disorders defined on the basis of family studies, as done by others.\textsuperscript{5-7} The latter consideration is particularly important since familial combined hyperlipidemia can be distinguished from familial hypertriglyceridemia by certain relationships among the lipoprotein fractions.\textsuperscript{6,7}

Methods

Eleven North American clinics participated in the population studies of the Lipid Research Clinics Program under a common protocol.\textsuperscript{8} The study population of the Northwest Lipid Research Clinic (NWLC) consisted of Pacific Northwest Bell Telephone Company employees working in King County, Washington. An initial screening of 5000 eligible employees (Visit 1) provided information on fasting plasma lipid levels relative to age, sex, race, and gonadal hormone use.\textsuperscript{9} At a second visit (Visit 2) a subset (n = 1190) of the Visit 1 participants had determinations of cholesterol and triglyceride in whole plasma and of triglyceride and cholesterol in each lipoprotein fraction are presented with hyperlipidemia differ from those of normolipidemia. In combined hyperlipidemia, the VLDL (C/TG) ratio was lower than in normolipidemia. In hypercholesterolemia, the median VLDL (C/TG) ratios were slightly lower than in normolipidemia. In hypertriglyceridemia, all three groups of subjects exhibited the least percentage of difference.

The relationship of triglyceride and cholesterol in plasma and the lipoprotein fractions were measured using methods previously described for the Lipid Research Clinics Program.\textsuperscript{10} VLDL lipids were calculated as the difference between their concentrations in the plasma and in the d = 1.006 infranatant solution obtained by ultracentrifugation at 40,000 rpm (105,000 \times g) for 18 hours at 10\textdegree C. HDL lipids were measured in the heparin-manganese chloride supernatants after VLDL and LDL precipitation.\textsuperscript{10} LDL lipids were calculated as the difference between their concentrations in the d = 1.006 infranate and the heparin-manganese supernate. The analytical performance and quality control procedures associated with the measurements of triglyceride and cholesterol in the lipoprotein fractions are described by Wahl et al.\textsuperscript{1} The ratios of lipoprotein cholesterol to triglyceride within fractions were computed and were denoted as VLDL(C/TG), LDL(C/TG), and HDL(C/TG).

This analysis was restricted to whites 20 to 59 years of age. Eleven people who were taking lipid-lowering medications were excluded. The age, sex, and hormone-use specific 90th percentile values from the local Visit 1 cholesterol and triglyceride distributions\textsuperscript{9} were used to classify 1072 Visit 2 participants, on the basis of their Visit 2 lipid determinations, into the following conditions: 1) normolipidemia, 2) hypercholesterolemia, 3) hypertriglyceridemia, and 4) elevations of both lipids, or combined hyperlipidemia. Subjects were further classified according to gender and sex hormone use as follows: males, females not taking oral contraceptives or replacement estrogens (nonusers) and females taking such hormones (users).

Summary statistics including the mean, median, and standard deviation describe the lipoprotein ratios. The Kruskal-Wallis nonparametric one-way analysis of variance procedure\textsuperscript{11} and Dunn's multiple comparison test\textsuperscript{12} were used to determine the significance of differences among the lipid groups. Spearman correlation coefficients\textsuperscript{11} describe the linear relationships between cholesterol and triglyceride within each of the lipoprotein fractions.

The relationship of the lipoprotein ratios and the respective lipoprotein triglyceride (L-TG) is inversely, as previously shown for a random sample of this population.\textsuperscript{2} Nonlinear regression analysis yielded a model of the form:

\[
\text{Ratio} = \frac{A \times B}{A + L-TG}
\]

where A is a constant and B approximates the ratio when the L-TG is equal to zero. This is the same functional form presented by Myers et al.\textsuperscript{13} for describing the relationship of the LDL and HDL ratios with total triglyceride.

Results

Lipoprotein Lipid Ratios

The ratios of the concentrations of cholesterol and triglyceride in each lipoprotein fraction are presented in Table 1. The extent to which the ratios associated with hyperlipidemia differ from those of normolipidemia are presented in Figure 1.

In hypercholesterolemia, the median VLDL (C/TG) ratios were slightly higher than in normolipidemia. In hypertriglyceridemia, all three groups of subjects had significantly lower VLDL (C/TG) ratios compared to normolipidemia. In combined hyperlipidemia, the VLDL (C/TG) ratio was lower than in normolipidemia but not as low as in hypertriglyceridemia. In both hypertriglyceridemia and combined hyperlipidemia, men exhibited the least percentage of difference.
HYPERLIPIDEMIA: LIPOPROTEIN LIPID INTERRELATIONSHIPS

Table 1. Lipoprotein Cholesterol (C) to Triglyceride (TG) Ratios by Lipid Classification

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Female nonusers</th>
<th>Female users</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Mean ± sd</td>
<td>Median</td>
</tr>
<tr>
<td>Normolipidemia</td>
<td>371</td>
<td>0.325 ± 0.166</td>
<td>0.292</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>86</td>
<td>0.344 ± 0.196</td>
<td>0.309</td>
</tr>
<tr>
<td>Hypertriglyceridemia</td>
<td>76</td>
<td>0.218 ± 0.066</td>
<td>0.207*†</td>
</tr>
<tr>
<td>Combined hyperlipidemia</td>
<td>38</td>
<td>0.258 ± 0.076</td>
<td>0.246†‡</td>
</tr>
<tr>
<td></td>
<td>370</td>
<td>8.98 ± 10.59</td>
<td>6.94</td>
</tr>
<tr>
<td></td>
<td>86</td>
<td>8.40 ± 3.84</td>
<td>7.54</td>
</tr>
<tr>
<td></td>
<td>76</td>
<td>5.18 ± 6.67</td>
<td>4.28*†</td>
</tr>
<tr>
<td></td>
<td>38</td>
<td>6.64 ± 4.85</td>
<td>5.59*†‡</td>
</tr>
</tbody>
</table>

*Significantly different from normolipidemia at \( p \leq 0.05 \).
†Significantly different from hypercholesterolemia at \( p \leq 0.05 \).
‡Significantly different from hypertriglyceridemia at \( p \leq 0.05 \).

from normolipidemia compared to women nonusers and users (Figure 1).

With respect to LDL, the (C/TG) ratio was 9% to 26% higher in hypercholesterolemia than in normolipidemia with the greatest difference (statistically significant) in women using hormones (Figure 1). In hypertriglyceridemia and combined hyperlipidemia, the LDL (C/TG) ratio was lower than in normolipidemia, more so in hypertriglyceridemia (18% to 38%) than in combined hyperlipidemia (5% to 20%). Women using hormones had the least percentage of reduction in both instances compared to men and women nonusers (Figure 1).

In HDL, the (C/TG) ratio was consistently, though nonsignificantly, higher (1% to 8%) in the hypercholesterolemic subjects. In hypertriglyceridemia, the (C/TG) ratio was significantly lower (38% to 48%) than normolipidemia, and was similar to combined hyperlipidemia (39% to 56%). In both these hyperlipidemias, women nonusers had a lesser percentage of difference in the (C/TG) ratio than men or hormone users.

Gender and sex hormone-use differences were apparent in the absolute (C/TG) ratios across all lipoprotein fractions to the extent that women not using hormones had the highest ratios and women using hormones the lowest. These relationships are generally true in both normolipidemia and hyperlipidemia.

**Lipoprotein Cholesterol/Triglyceride Ratios Controlling for Lipoprotein Triglyceride Concentration**

The cholesterol/triglyceride ratio of any lipoprotein fraction declined with increasing triglyceride concent-

![Figure 1](https://example.com/f1.png)
VLDL (C/TG) 1.20H 0.96- 0.72- 0.48- 0.24- 0
100 200 300 400 500 600 700 VLDL Triglyceride (mg/dl)

Figure 2. VLDL cholesterol/triglyceride ratio as a function of VLDL triglyceride concentration (---) for white males ages 20 to 59. Dashed line (-----) represents the area covered by data for normolipidemics. ○ = hypertriglyceridemics; • = combined hyperlipidemics.

Figure 3. LDL cholesterol/triglyceride ratio as a function of LDL triglyceride concentration (---) for white women not taking hormones ages 20 to 59. Dashed line (-----) represents the area covered by data for normolipidemics. ○ = hypertriglyceridemics; • = combined hyperlipidemics.

Figure 4. HDL cholesterol/triglyceride ratio as a function of HDL triglyceride concentration (---) for white women taking hormones ages 20 to 59. Dashed line (-----) represents the area covered by data for normolipidemics. ○ = hypertriglyceridemics; • = combined hyperlipidemics.
lowest VLDL cholesterol and triglyceride correlation ($r = 0.31$).

Cholesterol and triglyceride in LDL were more weakly correlated in the three hyperlipidemic groups than in normolipidemics. Correlations were lowest among hypertriglyceridemic and combined hyperlipidemic men ($r = 0.08$ and 0.14, respectively). Less striking trends were observed in the two groups of women.

In HDL, the correlations between cholesterol and triglyceride were generally weakest among men and strongest among hormone users. The largest correlations were observed in combined hyperlipidemia: men, $r = 0.25$; women nonusers, $r = 0.54$; and women users, $r = 0.63$. No linear relationship was apparent for normolipidemic men or hypertriglyceridemic nonusers.

**Lipoprotein Lipid Correlations among Lipoprotein Fractions**

To examine the distinctions between hypertriglyceridemia and combined hyperlipidemia, we estimated the linear relationship of VLDL cholesterol with LDL cholesterol and VLDL triglyceride with HDL cholesterol, relationships previously reported as differing between familial combined hyperlipidemia and familial hypertriglyceridemia. Results are presented in Table 3. In normolipidemia and hypercholesterolemia, VLDL cholesterol and LDL cholesterol were not significantly correlated. However, in hypertriglyceridemic subjects a moderately inverse relationship was observed and an even stronger inverse relationship was observed in men ($r = -0.65$) and women using hormones ($r = -0.81$) with combined hyperlipidemia. Moderately negative correlations between VLDL triglyceride and HDL cholesterol were observed in men and women nonusers for all lipid groups. The negative correlations seen in combined hyperlipidemia were less than in hypertriglyceridemia for both men ($r = -0.13$ vs $-0.33$) and women nonusers ($r = -0.23$ vs $-0.55$). For hormone users the correlations were negative in normolipidemia and hypercholesterolemia, but moderately positive in hypertriglyceridemia ($r = 0.21$) and combined hyperlipidemia ($r = 0.16$).

**Table 2. Spearman Correlations between Cholesterol and Triglyceride within Each Lipoprotein Fraction by Lipid Classification, Gender, and Sex Hormone Use**

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Female nonusers</th>
<th>Female users</th>
</tr>
</thead>
<tbody>
<tr>
<td>VLDL-C vs VLDL-TG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normolipidemia</td>
<td>0.71</td>
<td>0.45</td>
<td>0.56</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>0.63</td>
<td>0.47</td>
<td>0.34</td>
</tr>
<tr>
<td>Hypertriglyceridemia</td>
<td>0.67</td>
<td>0.49</td>
<td>0.55</td>
</tr>
<tr>
<td>Combined hyperlipidemia</td>
<td>0.88</td>
<td>0.31</td>
<td>0.73</td>
</tr>
</tbody>
</table>

| LDL-C vs LDL-TG |              |                 |              |
| Normolipidemia  | 0.52         | 0.43            | 0.40         |
| Hypercholesterolemia | 0.44     | 0.34            | 0.14         |
| Hypertriglyceridemia | 0.08     | 0.23            | 0.33         |
| Combined hyperlipidemia | 0.14 | 0.27            | 0.24         |

| HDL-C vs HDL-TG |              |                 |              |
| Normolipidemia  | 0.06         | 0.15            | 0.34         |
| Hypercholesterolemia | 0.15     | 0.20            | 0.33         |
| Hypertriglyceridemia | 0.14     | 0.05            | 0.44         |
| Combined hyperlipidemia | 0.25 | 0.54            | 0.63         |

**Accounted for**

Atherosclerosis risk is greater in men than in women, and is more severe in familial hypercholesterolemia and familial combined hyperlipidemia than in familial hypertriglyceridemia. Since reliable markers for the genetically determined hyperlipidemias are still lacking, statistical definitions of hyperlipidemia (i.e., greater than the 90th or 95th percentile values in population-based samples) provide a practical method for the identification of hyperlipidemia. Using this pragmatic approach, we have examined three classes of hyperlipidemia arbitrarily de-

**Discussion**

Atherosclerosis risk is greater in men than in women, and is more severe in familial hypercholesterolemia and familial combined hyperlipidemia than in familial hypertriglyceridemia. Since reliable markers for the genetically determined hyperlipidemias are still lacking, statistical definitions of hyperlipidemia (i.e., greater than the 90th or 95th percentile values in population-based samples) provide a practical method for the identification of hyperlipidemia. Using this pragmatic approach, we have examined three classes of hyperlipidemia arbitrarily de-

**Table 3. Selected Correlations* among Lipoprotein Lipids in Normolipidemic and Hyperlipidemic Groups by Gender and Sex Hormone Use**

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Female nonusers</th>
<th>Female users</th>
</tr>
</thead>
<tbody>
<tr>
<td>VLDL-C vs LDL-C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normolipidemia</td>
<td>0.01</td>
<td>0.11</td>
<td>-0.09</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>-0.16</td>
<td>-0.04</td>
<td>0.11</td>
</tr>
<tr>
<td>Hypertriglyceridemia</td>
<td>-0.52</td>
<td>-0.20</td>
<td>-0.26</td>
</tr>
<tr>
<td>Combined hyperlipidemia</td>
<td>-0.65</td>
<td>-0.06</td>
<td>-0.81</td>
</tr>
</tbody>
</table>

| VLDL-TG vs HDL-C  |              |                 |              |
| Normolipidemia    | -0.47        | -0.31           | -0.10        |
| Hypercholesterolemia | -0.23 | -0.41           | -0.29        |
| Hypertriglyceridemia | -0.33   | -0.55           | 0.21         |
| Combined hyperlipidemia | -0.13  | -0.23           | 0.16         |

* Spearman correlations.
Another variable potentially affecting atherosclerosis risk is the lipoprotein cholesterol/triglyceride ratio. Our previous study of lipoprotein cholesterol/triglyceride ratios in a general population shows no differences between men and women not using hormones when the ratios are adjusted for triglyceride concentrations. However, in women using hormones the LDL triglyceride concentration is increased disproportionately to the LDL cholesterol concentration resulting in a decrease in the LDL cholesterol/triglyceride ratio.2 This effect is seen in both premenopausal oral contraceptive users and in postmenopausal estrogen users.3 The lipoprotein cholesterol/triglyceride ratio has not been described in hyperlipidemic subjects and is the focus of this paper. In addition, we took the opportunity to examine the relationships of lipoprotein lipids among the different lipoprotein fractions, with emphasis on hypertriglyceridemia and combined hyperlipidemia, to determine if there are distinctions similar to those described for the familial forms of these conditions.

In hypercholesterolemia, slightly higher cholesterol/triglyceride ratios are present in each of the lipoprotein fractions, accompanying absolute increases in the concentrations of lipoprotein cholesterol that we previously observed.4 With respect to VLDL and LDL, an increase in the cholesterol/triglyceride ratio could signify an increase in lipoprotein remnants in the VLDL fraction or a more smectic, less liquid crystalline structure in LDL. Ratio increases in both lipoprotein fractions could enhance the potential for atherogenesis on the basis of the human model of “remnant removal disease”18 and primate models of the varied atherogenicity of LDL.18

In hypertriglyceridemia, the cholesterol/triglyceride ratio is low, reflecting the higher concentration of triglyceride in each fraction.4 Compared to normolipidemia, men experience the least decrease in the VLDL ratio, a potentially atherogenic bias. However, men also have the greatest reduction in the LDL ratio, a potentially antiatherogenic bias (figure 1). Hormone users experience the largest decrement in the VLDL ratio and the smallest in the LDL ratio. It is not known if these order differences reflect hormonal effects, but the question deserves more study. There is no apparent effect of gender or sex-hormone on the order differences in HDL.

In combined hyperlipidemia, the percent difference from normolipidemia of the cholesterol/triglyceride ratios in VLDL and LDL is notably less than in hypertriglyceridemia. The HDL ratio is similarly lower in combined hyperlipidemia and hypertriglyceridemia. There is no apparent order effect of gender and sex hormone use. The ratio differences in VLDL and LDL are not a function of a lower concentration of triglyceride in these lipoprotein fractions because the ratios remain elevated in combined hyperlipidemia compared to pure hypertriglyceridemia, even when triglyceride concentration is taken into account (Figures 2 to 4). A larger VLDL and LDL cholesterol/triglyceride ratio in combined hyperlipidemia could have greater atherogenicity compared to pure hypertriglyceridemia,18,19 as explained above. A higher VLDL(C/TG) ratio has been described in familial combined hyperlipidemia compared to familial hypertriglyceridemia.6,7 The parallel differences between hypertriglyceridemia and combined hyperlipidemia found in this study suggest that the familial forms of these disorders may be present in our empirically defined groups of hyperlipidemias.

Comparing the Spearman correlations of lipoprotein lipid concentrations in normolipidemia and hyperlipidemia, there are differences observed according to gender or hormone-use group as well as category of hyperlipidemia. The order of correlations by gender and hormone-use group differs in each lipoprotein fraction. The interpretation of these observations is not known, but the results suggest differing degrees of influence by gender or sex hormone use on lipoprotein lipid structure. Perhaps more significant are the high correlations in VLDL and HDL and low correlations in LDL observed in combined hyperlipidemia.

Relationships of VLDL cholesterol to LDL cholesterol and VLDL triglyceride to HDL cholesterol were examined to determine if the characteristics that distinguish hypertriglyceridemia and combined hyperlipidemia resemble those reported for the familial forms of these disorders.6,7 This hypothesis is largely borne out in men. The relationship between VLDL cholesterol and LDL cholesterol was more strongly inverse in combined hyperlipidemia than in familial hypertriglyceridemia in men and women using hormones, as previously described for predominantly male subjects with familial disorders.6,7 Women not using hormones apparently do not fit this pattern. In addition, the presence of a strongly negative correlation in male hypertriglyceridemic subjects contrasts with the observations made by Chait et al.8 in whose study the subjects had no negative association between VLDL cholesterol and HDL cholesterol. However, their subjects were more markedly hypertriglyceridemic than our group. There is no assurance that our hypertriglyceridemic group is not comprised of a large proportion of subjects with familial combined hyperlipidemia manifesting phenotypically as hypertriglyceridemia. Indeed, the trait for familial combined hyperlipidemia is expressed in 1% to 2% of the population.5 In the future, apolipoprotein B measurements may be helpful as a marker for familial combined hyperlipidemia in all of its manifestations.20

Many features that reportedly distinguish between familial hypertriglyceridemia and familial combined hyperlipidemia are found in our empirically defined hypertriglyceridemic and combined hyperlipidemic male subjects.6,7,19 These include: 1) an HDL cholesterol concentration intermediate between normolipidemia and hypertriglyceridemia; 2) a higher VLDL cholesterol/triglyceride ratio than in hypertriglyceridemia; 3) a more strongly inverse relationship between VLDL and LDL cholesterol than in hypertri-
glyceridemia, at least in men and female hormone users; 4) a weaker inverse relationship between VLDL triglyceride and HDL cholesterol (in men and women not using hormones only). Previously undescribed is the inverse relationship between VLDL and LDL cholesterol in hypertriglyceridemia and the inconsistency observed for women in conforming to these VLDL and LDL or VLDL and HDL interrelationships which are most clearly expressed in men. It is noteworthy that there is little inverse relationship between VLDL and LDL cholesterol in normolipidemic and hypercholesterolemic subjects. There appear to be more features in common between hypertriglyceridemia and combined hyperlipidemia than between either of these two and hypercholesterolemia.

The present study shows that the lipoprotein cholesterol/triglyceride ratios are larger in certain hyperlipemias and that differences from normolipidemia vary with gender and sex hormone use, but in a manner insufficient to suggest a unifying theory. Obviously, these lipoprotein ratios do not permit distinctions among altered composition, altered mass, or both. Nonetheless, a consistent finding is the presence of a number of alterations in lipoprotein lipid composition and concentration in combined hyperlipidemia, most clearly expressed in men, which is consistent with a higher degree of atherogenicity in this disorder.

Acknowledgments

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References


Index Terms. hypercholesterolemia • hypertriglyceridemia • combined hyperlipidemia • lipoprotein ratio • cholesterol • triglyceride
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