Calculation of LDL-cholesterol by using apolipoprotein B for classification of nonchylomicronemic dyslipemia

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In this paper we propose a calculation of LDL-cholesterol (LDL-C) not affected by hypertriglyceridemia by using lipid quantities directly measured in total serum. We also propose an algorithm for the classification of nonchylomicronemic dyslipemias. Plasma apolipoproteins (apo) A-I, B, total cholesterol (TC), triglycerides (TG), and cholesterol of lipoproteins were measured in a group of 38 normolipemic and 120 dyslipemic patients (42 phenotype IIa, 38 IIb, and 40 IV) classified according to TG and LDL-C values. Discriminant analysis was applied to obtain the best classification with the lowest number of quantities directly measured from total serum (TC, TG, and apo B), and multiple regression analysis was performed to find an equation to calculate LDL-C from these quantities. Apo B seems to be a useful discriminator between normolipemic and phenotype IIa patients, by using a cutoff value of 1.35 g/L obtained by ROC curve analysis. The proposed algorithm, based on lipid quantities measured by easily automated methods, is shown to be a good alternative for the classification of nonhyperchylomicronemic dyslipemia. LDL-C calculated from TC, TG, and apo B proved a better estimate of true LDL-C than the estimate obtained with Friedewald’s formula.

INDEXING TERMS: apolipoproteins • Friedewald formula • HDL-cholesterol • triglycerides • VLDL-cholesterol • discriminant analysis • multiple regression

Some genetic and secondary dyslipemias are asymptomatic and only identifiable by analysis of serum lipid constituents. International guidelines recommend serum/plasma measurement of total cholesterol (TC) and triglycerides (TG) for proper identification and control of dyslipemia [1]. Although phenotypic classification of dyslipemias can be obtained from these two quantities [2], plasma concentrations of HDL-cholesterol (HDL-C) and, especially, LDL-C, give more information about the lipid status and its relation to cardiovascular risk than does TC [3]. For this reason, clinical laboratories should measure LDL-C concentrations in a large number of samples.

A method based on immunoextraction of VLDL-C and HDL-C from plasma has recently been developed for direct LDL-C measurement [4, 5]. This method enables LDL-C to be measured exactly, even in hypertriglyceridemic and (or) phenotype III samples. Nevertheless, this method has some drawbacks [4, 5] that arouse caution about its use, such as a decrease of LDL-C values in frozen samples or in samples obtained under nonfasting conditions.

Reference methods proposed for LDL-C measurement are based on ultracentrifugation of serum [6, 7]. Because ultracentrifugation is a costly and impractical method, Friedewald proposed a formula to estimate LDL-C concentrations from TC, TG, and HDL-C [8]. However, Friedewald’s formula is unable to give true VLDL-C and LDL-C concentrations in sera with chylomicrons, with triglyceridemia >4.52 mmol/L, or with abnormal abundance of IDL [9].

Some immunological methods can easily measure apolipoproteins (apo), especially apo A-I and apo B. Because apo A-I and apo B are the major components of HDL and...
LDL particles, respectively, the first evaluations of their clinical interest suggested that they could replace HDL-C and LDL-C as cardiovascular risk parameters. All subjects with hyperapoipoproteinemia B have been shown to have a threefold increase in risk of coronary diseases [10–12]; thus, apo B concentrations appear to be useful for evaluation of lipid status. As mentioned above, most of the work done with apolipoproteins has been conducted to study their possible use in replacing HDL-C and (or) LDL-C measurements, but has not been focused on analyzing their role in the phenotypic classification of dyslipidemia or in improving LDL-C calculation.

The aims of this study were: (a) to develop a formula including apo B for the estimation of LDL-C in samples from dyslipemic patients (including grossly hypertriglycerideremic samples), and (b) to develop an algorithm for the classification of nonchylomicronemic dyslipidemia by means of direct and easily automated determinations of lipid quantities (i.e., TC and TG, HDL-C, and apolipoproteins A-I and B).

Subjects and Methods

Study Protocol and Analytical Methods
Blood samples were obtained from subjects after they had fasted overnight (10–12 h). The blood was left at room temperature for 30 min and serum was separated by centrifuging for 15 min at 3000g. TC and TG were immediately analyzed. For lipoprotein analysis, a preservative solution was added to achieve a final concentration (mmol/L) of 1.0 of Na₂EDTA, 0.15 of gentamycin sulfate, 1.2 of cloramphenicol, and 10 of sodium azide (pH 7.2) [13].

TC and TG were measured by fully enzymatic methods (CHOD-PAP and GPO-PAP, refs. 1127578 and 1361155, respectively, from Boehringer Mannheim, Mannheim, Germany) in a RA-XT analyzer (Bayer Diagnostics, Leverkusen, Germany). The imprecision for TC and TG was, respectively, 1.55% and 2.18%. Lipoprotein fractions were separated by combined ultracentrifugation and precipitation [6]; VLDL-C was measured in the top fraction (d = 1.006 kg/L) and LDL+HDL in the bottom one (d >1.006 kg/L). HDL-C was measured by precipitation with phosphotungstate (Boehringer Mannheim, ref. 543004) from total serum. LDL-C was calculated by subtracting HDL-C values from the cholesterol fraction with a density >1.006 kg/L. The imprecisions for lipoprotein fractions were HDL-C 3.64%, LDL-C 2.91%, and VLDL-C 4.30%. Serum concentrations of apo A-I and B were determined by immunonephelometric methods, by using as antiserum a polyclonal specific antibody against each apolipoprotein (Behringwerke, Marburg, Germany, refs. OUED/15 and OSAN/15, respectively) and a Behring standard ref. OUPG/07 calibrated to an IFCC standard [14] in a Behring Nephelometer Analyzer. The imprecisions for apo A-I and apo B were, respectively, 2.10% and 1.91%.

Subjects
We studied 120 dyslipemic patients monitored in our lipid laboratory and 38 normolipemic healthy subjects. Patients with chylomicronemia or suspected dysbeta-ipoproteinemia [ratio VLDL-C (mmol/L):total TG (mmol/L) ≥0.68] were excluded. Patients were categorized as normo- or dyslipemic by using recommended cutoff points of 2.26 mmol/L for TG and (or) of 4.13 mmol/L for LDL-C obtained from ultracentrifugation [9]. According to these cutoffs, 38 individuals were classified as normolipemic and 120 as dyslipemic (42 of them as phenotype IIa, 38 as IIb, and 40 as IV).

Statistical Methods
Mean and SD of dyslipemic patients and normolipemic subjects were calculated for all quantities determined.

Apo B cutoff value of 1.35 g/L was reached by ROC curve analysis.

To obtain the minimum number of variables necessary for distinguishing between the groups studied, we used discriminant analysis to obtain the percentage of correctly classified individuals and the coefficients of the standardized discriminant function. We then removed the quantity with the lowest coefficient and repeated the discriminant analysis.

Once the best set of quantities had been selected by discriminant analysis, a multiple regression analysis was performed to study the correlation between LDL-C and those quantities directly measured in serum, and to find a function that allowed LDL-C to be calculated from TC, TG, and apo B.

Discriminant analysis and multiple regression of the quantities were performed with the statistical package Complete Statistical System (CSS:Statistica) version 3.1.

Because Friedewald’s or similar formulas can lead to gross errors in LDL-C calculation of hypertriglycerideremic patients, we used results obtained from samples containing phenotypes IIb and IV to find a formula that allowed LDL-C to be calculated. To validate this formula, the following criteria were used:

1) The formula was applied to normolipidemic and phenotype IIa samples not included in the population used to generate this formula.

2) Hypertriglycerideremic patients were randomly divided into two subsets of samples, and a similar formula was obtained from each subset. Afterwards, the formula obtained from one subset was applied to the other subset. Differences in LDL-C calculated from the two different subsets and in the LDL-C calculated from the entire set of hypertriglycerideremic patients were studied by means of Wilcoxon’s t-test.

3) A further validation was, in hypertriglycerideremic patients, the correlation analysis of VLDL-C obtained by ultracentrifugation with VLDL-C obtained from TC, HDL-C, and LDL-C calculated according to formula (Eq. 1 in Results).

LDL-C results calculated in such a way were correlated
to those obtained by ultracentrifugation and to those obtained from Friedewald’s formula by using Passing–Bablok regression analysis [15, 16]. Wilcoxon’s t-test was used to analyze differences between LDL-C values calculated and those obtained by ultracentrifugation.

The variances of LDL-C values calculated with the new formula and LDL-C values calculated with Friedewald’s formula were obtained through the formula for the propagation of errors [17].

**Results**

Table 1 shows descriptive statistics of lipid constituents from the 158 samples analyzed, including the ratio VLDL-C:TG. In Fig. 1, the values obtained for TG, HDL-C, and apo A-I and apo B of all individuals studied are shown. From the graphical representation of results, one can deduce that HDL-C and apo A-I values do not allow for differentiation between normo- and hyperlipemic patients. Also, the apo B cutoff value of 1.35 g/L allows for accurate separation of all normolipemic subjects from those with phenotype IIa in the normotriglyceridemic group.

To distinguish between phenotypes IIb and IV in the hypertriglyceridemic group, a discriminant analysis was applied to this set of samples by using all lipid quantities. LDL-C obtained by ultracentrifugation was the most efficient to separate the two phenotypes. This value allowed for correct classification of 93.6% of hypertriglyceridemic patients (92.1% of phenotype IIb and 95% of phenotype IV). To comply with the cutoff value recommended by National Cholesterol Education Program (NCEP) guidelines for LDL-C (4.13 mmol/L), a constant of −0.27 was added to the equation:

\[
\text{LDL-C} = 0.41 \text{TC} - 0.32 \text{TG} + 1.70 \text{apoB} - 0.27 \quad (2)
\]

Figure 2 shows the graphical representation of LDL-C values calculated by this equation in all individuals studied. When Eq. 2, based on hypertriglyceridemic patients, was applied to normolipemic and phenotype IIa patients, the values of calculated LDL-C correctly classified 100% of these patients.

Column 1: With Wilcoxon’s t-test did not show significant differences (P = 0.17) between LDL-C values calculated according to Eq. 2 and those obtained by ultracentrifugation. The same comparison between LDL-C by ultracentrifugation and those with Friedewald’s formula showed a significant difference (P < 0.05), mainly due to differences in IIb (P = 0.04) and IV (P = 0.05) phenotype patients (Table 4). In addition, association between LDL-C obtained by ultracentrifugation and LDL-C calculated from Eq. 2 or Friedewald’s formula was 2.97%.

\[
\text{ANOVA of multiple regression shows } F = 3213.253 \text{ and } P < 0.001.
\]

A calculated LDL-C cutoff value of 4.40 mmol/L was used as the dependent variable, and TC, TG, and apo B as independent ones. The coefficients for each variable, with the standard error, confidence intervals, and significance level are shown in Table 2.

A multiple regression coefficient of 0.9961 and the following equation (all quantities in mmol/L, except for apo B in g/L) were obtained:

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\]
wald’s formula was studied by using Passing–Bablok regression analysis. Fig. 3 shows regression lines, correlation coefficients, and parameters of regression lines for all patients: normotriglyceridemic (normolipemic and phenotype IIa) and hypertriglyceridemic ones (phenotypes IIb and IV).

After the validation study, no statistically significant differences were found between LDL-C calculated from the two different subsets \((P = 0.39)\), nor when comparing these with LDL-C calculated (Eq. 1) from the entire set of hypertriglyceridemic patients \((P = 0.66 \text{ and } P = 0.65)\).

As a further test, the ratio VLDL-C:TG with the VLDL-C obtained by subtraction of HDL-C and LDL-C calculated (Eq. 1) from TC was checked in hypertriglyc-

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**Table 2. Multiple regression analysis.**

<table>
<thead>
<tr>
<th></th>
<th>Coefficients</th>
<th>Standard errors</th>
<th>Confidence intervals</th>
<th>(P) level</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC</td>
<td>0.409702</td>
<td>0.068015</td>
<td>0.2763926</td>
<td>0.00000</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.5430094</td>
<td></td>
</tr>
<tr>
<td>TG</td>
<td>−0.324762</td>
<td>0.028077</td>
<td>0.269731</td>
<td>0.00000</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.3797929</td>
<td></td>
</tr>
<tr>
<td>Apo B</td>
<td>1.700713</td>
<td>0.252654</td>
<td>1.2055112</td>
<td>0.00000</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2.1959148</td>
<td></td>
</tr>
</tbody>
</table>

LDL-C obtained by ultracentrifugation was used as dependent variable and TC, TG, and apo B as independent ones.
eridemic patients. All patients had a ratio <0.68. A regression analysis between VLDL-C obtained by ultracentrifugation and VLDL-C obtained by subtraction led to a regression coefficient of 0.910 and a regression line defined by the equation 

\[ y = 2.15 + 0.957x \]

with confidence intervals for \( y \)-intercept and for slope comprising 0 and 1, respectively.

As a consequence of all the results described above, a sequential algorithm for classification of the patients studied was developed (Fig. 4). The first step was to identify phenotypes I and V by storing samples at 4 °C. The next step consisted of separating phenotypes IIb and IV from the others by a cutoff TG value of 2.26 mmol/L. Normotriglyceridemic (normolipemics and phenotype IIa) patients could be differentiated by a cutoff value of apo B of 1.35 g/L, and hypertriglyceridemic patients could be separated into IIb and IV phenotypes by using LDL-C calculated according to Eq. 2. By using this algorithm, 153 of 158 patients (96.8%) were correctly classified (100% normolipemic and IIa phenotype patients, 92.1% phenotype IIb, and 95% phenotype IV).

**Discussion**

Diagnosis and control of dyslipemia are major concerns of strategies for both primary and secondary prevention of cardiovascular diseases. So-called “opportunistic” screening for cardiovascular diseases in subjects over age 20, seen for any medical reason, is recommended by international guidelines [1, 18]. Lipid quantities to be included in this “opportunistic” screening are TC and HDL-C [18]; from these two values and TG, LDL-C values can be derived [8]. This screening, along with monitoring of known dyslipemic patients, implies a large number of samples to be analyzed for LDL-C measurement. Furthermore, LDL-C should be preferred to TC as a marker of cardiovascular risk, because TC can appear as “normal” when high LDL-C values coexist with low HDL-C values.

Friedewald’s formula allows for estimation of LDL-C from TC, TG (used to estimate VLDL-C), and HDL-C. This calculation multiplies the errors derived from TC, TG, and HDL-C measurements [19]. Pitfalls of LDL-C estimation by this formula, which means that ultracentrifugation must be used, occur in gross hypertriglyceridemic patients (>4.52 mmol/L) [13].

According to internationally accepted guidelines for LDL-C measurement [9], in our population, this constituent should be measured by ultracentrifugation in 39.7% of hypertriglyceridemic patients (12 of 41 phenotype IIb, 19 of 40 phenotype IV) who had total TG concentrations >4.52 mmol/L.

However, in spite of exclusion of chylomicronemic and phenotype III sera from our set of samples, ultracentrifu-

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**Table 3. Phenotypic classification of individuals (n = 158) according to Friedewald’s formula (F) and newly developed formula (N)*

<table>
<thead>
<tr>
<th>True phenotype by ultracentrifugation</th>
<th>Predicted phenotype by total TG and LDL-C by F and N</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normolipemic (n = 38)</td>
</tr>
<tr>
<td></td>
<td>Phenotype IIa (n = 42)</td>
</tr>
<tr>
<td></td>
<td>Phenotype IIb (n = 38)</td>
</tr>
<tr>
<td></td>
<td>Phenotype IV (n = 40)</td>
</tr>
<tr>
<td>Normolipemic (n = 38)</td>
<td>F/N</td>
</tr>
<tr>
<td></td>
<td>37/38</td>
</tr>
<tr>
<td>Phenotype IIa (n = 42)</td>
<td>F/N</td>
</tr>
<tr>
<td></td>
<td>1/0</td>
</tr>
<tr>
<td>Phenotype IIb (n = 38)</td>
<td>F/N</td>
</tr>
<tr>
<td></td>
<td>0/0</td>
</tr>
<tr>
<td>Phenotype IV (n = 40)</td>
<td>F/N</td>
</tr>
<tr>
<td></td>
<td>0/0</td>
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</tbody>
</table>

* LDL-C = 0.41TC - 0.32TG + 1.70apo B - 0.27

**Table 4. LDL-C values (mean ± SD) obtained by ultracentrifugation (UC), Friedewald’s (F), and newly developed (N) formula in the different groups and in the whole set of samples analyzed.**

<table>
<thead>
<tr>
<th></th>
<th>Normolipemic (n = 38)</th>
<th>Phenotype IIa (n = 42)</th>
<th>Phenotype IIb (n = 38)</th>
<th>Phenotype IV (n = 40)</th>
<th>Whole group (n = 158)</th>
</tr>
</thead>
<tbody>
<tr>
<td>UC</td>
<td>3.19 ± 0.56</td>
<td>6.46 ± 2.21</td>
<td>5.27 ± 0.93</td>
<td>3.40 ± 0.68</td>
<td>4.61 ± 1.88</td>
</tr>
<tr>
<td>F</td>
<td>2.99 ± 0.60</td>
<td>6.08 ± 2.14</td>
<td>4.84 ± 0.97*</td>
<td>3.07 ± 0.82*</td>
<td>4.27 ± 1.84*</td>
</tr>
<tr>
<td>N</td>
<td>3.11 ± 0.59</td>
<td>5.82 ± 1.78*</td>
<td>4.91 ± 0.81</td>
<td>3.26 ± 0.79</td>
<td>4.30 ± 1.60</td>
</tr>
</tbody>
</table>

* P <0.05 with respect to UC values by Wilcoxon’s t-test.
gation was still required in 19.6% of patients (31 of 158) (TG >4.52 mmol/L). Because 19.6% represents a large number of samples, any approach that avoids ultracentrifugation in LDL-C measurement or any improvement regarding its calculation would be useful for most clinical laboratories, even in hypertriglyceridemic samples.

The apo B cutoff value of 1.35 g/L, which separates normolipemic from phenotype IIa samples, was obtained by ROC curve analysis, showing a sensitivity and a specificity of 1.00. Although it differs from the reported value in other studies using the same or different methods [20], the international standard of apo B (SP3- specificity of 1.0007 International Reference Material) was not available and was not analyzed by different methods until 1994 [14]. For this reason, comparison of results among different authors should be interpreted cautiously. Whatever the case, apo B values cannot differentiate between phenotypes IIb and IV in hypertriglyceridemic patients.
This is not surprising because hypertriglyceridemic patients are known to be heterogeneous with respect to apo B values [21].

With Eq. 2 obtained from hypertriglyceridemic patients, LDL-C correctly classified 100% of patients in normolipemic and phenotype IIa groups, representing an indirect confirmation of the usefulness of such a calculation. After randomly dividing hypertriglyceridemic patients into two subsets, coefficients of the new equations obtained were between the confidence limits of the coefficients of Eq. 1. Moreover, the percentage of individuals correctly classified was exactly the same and there were no statistically significant differences between the LDL-C calculated with the two new equations, nor with the LDL-C calculated with Eq. 1.

As stated by the Working Group of Lipoprotein Measurements of NCEP [9], the proportion of phenotype III patients is very low (from 0.02% to 0.2%), and the prevalence of this phenotype in dyslipemic patients ranges between 0.1% and 1%. Furthermore, because most phenotype III patients have high concentrations of TG [22] and because no patients in this group had a ratio VLDL-C:TG >0.68, when VLDL-C was obtained by subtraction, phenotype III patients could be excluded. This VLDL-C estimation was significantly associated with VLDL-C obtained by ultracentrifugation. Therefore, these results represent indirect but strong evidence of the expected behavior of LDL-C as calculated in hypertriglyceridemic samples.

There was also a close association between LDL-C calculated by Eq. 2 and LDL-C obtained by ultracentrifugation in hypertriglyceridemic sera. This association was better than the association between LDL-C obtained by Friedewald’s formula and LDL-C obtained by ultracentrifugation.

Various authors have tried to improve the classification power of Friedewald’s formula in two ways: (a) by modifying the numerical value used to estimate VLDL-C from TG [4, 23–25], or (b) by considering a specific cholesterol:TG ratio for each plasmatic lipoprotein and incorporating it in the calculation of LDL-C [24]. With the first approach, no essential improvements in reaching true LDL-C values have been obtained: The factor used in Friedewald’s formula appears to be the best one for estimating LDL-C [4]. As for the second approach, the use of a unique numerical value to calculate VLDL-C from TG values is based on the assumption that all TG from plasma comes from VLDL particles and that cholesterol:TG in VLDL is constant. However, from our study, the mean VLDL-C:TG ratios of the different phenotypes ranged from 0.234 to 0.409. Similar results have been described elsewhere [26], and variation of the cholesterol:TG ratio in VLDL particles has been shown to increase the pitfalls in calculating LDL-C from 4% to 60% [27]. Improvement of the classification power of Friedewald’s formula, especially in hypertriglyceridemic patients, seems far from ideal.

Moreover, apo B values are far from representative of the number of plasmatic apo B-containing particles, even with alteration of their lipid content, as occurs in patients with abnormal abundance of small, dense LDL particles [10]; LDL+IDL account for >90% of atherogenic apo B-containing particles, even in hypertriglyceridemic subjects [21], and IDL are the minor components of this pair of particles. Thus, as demonstrated by our results, inclusion of apo B values in the calculation of LDL-C produces an estimate of LDL-C that approaches true LDL-C concentrations obtained by ultracentrifugation.

The formula developed offers several advantages over traditional approaches to measuring LDL-C. First, ultracentrifugation can be avoided even in hypertriglyceridemic samples. Second, LDL-C can be derived from quantities such as TC, TG, and apo B easily measured by automated methods. Thus, if not necessary for cardiovascular risk evaluation, HDL-C measurement by precipitation methods can be avoided. Third, apo B values on their own offer an additional predictor of cardiovascular risk as demonstrated by prospective studies, showing a relation between hyperapolipoproteinemia B and increased risk of cardiovascular diseases in normolipemic, hypercholesterolemic, and hypertriglyceridemic patients [11, 27]. Furthermore, in patients with apo B concentrations higher than reference ranges, such as those with hypertriglyceridemia, a pharmacological treatment based on apo B concentrations [28], rather than the classical treatment, should be recommended. Finally, by using three measured constituents (TC, TG, and apo B) and a derived one (calculated LDL-C), nonchylomicronemic patients can easily be classified in their respective phenotypes.

It is advisable that all laboratories recalculate their formulas (coefficients of each variable) to solve the differences caused by method and instrumentation.

In conclusion, our findings support the hypothesis that the application of an algorithm that calculates LDL-C by means of a new formula gives an accurate calculation of LDL-C, based on easy automated determinations of lipid quantities, and allows a good phenotypic classification of
nonchylomicronemic dyslipemia. The introduction of apo B in the set of determinations enables hypertriglycerideemic samples with high apo B values to be recognized. According to these results, derivation of LDL-C values with the reported formula offers several advantages over values calculated with Friedewald's formula.

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References