The HS-Omega-3 Index Is Favorably Associated with Triglycerides and Inflammatory Markers: Data from 100,000 Patients

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Synopsis: The HS-Omega-3 Index (RBC eicosapentaenoic acid + docosahexaenoic acid; EPA + DHA) has been shown to be independently and inversely related to risk for sudden cardiac death and for acute coronary syndromes (ACS). This relationship could be mediated by effects of omega-3 fatty acids (O3FA) on other coronary heart disease (CHD) risk markers.

Purpose: The purpose of this study was to examine the cross-sectional relationships between the HS-Omega-3 Index and selected lipid/lipoprotein fractions and inflammatory markers in a large clinical dataset.

Methods: Data on the HS-Omega-3 Index and lipid (low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides) and inflammatory markers (C-reactive protein [CRP] and Lipoprotein-associated phospholipase A2 [LpPLA2]) were collected from tests performed at HDL. No information other than age and sex were available for these clinical samples. Nonparametric splines with 95% confidence bands were used for exploratory analysis. Partial (adjusted for age) Pearson's correlations were calculated cross-sectionally between the Index and the biomarkers; non-normally distributed variables were log-transformed.

Results: The most significant difference between genders was that in women the omega-3 index had a direct relation with HDL-C; however, in men they were independent (Table 1 and Figure 1). The other biomarkers had similar magnitudes between genders. The inflammatory markers had the strongest (inverse) correlation with the omega-3 index and LDL-C had the weakest.

Conclusions: The inverse association between the HS-Omega-3 Index and coronary heart disease could be, in part, mediated by favorable relations with known risk markers.

Table 1 Age-adjusted correlations with Ln(Omega-3 Index)

<table>
<thead>
<tr>
<th>Biomarker</th>
<th>Male</th>
<th>N</th>
<th>Female</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ln(TGs)</td>
<td>−0.095</td>
<td>52,944</td>
<td>−0.139</td>
<td>55,566</td>
</tr>
<tr>
<td>HDL-C</td>
<td>0.006 *</td>
<td>52,930</td>
<td>0.108</td>
<td>55,539</td>
</tr>
<tr>
<td>LDL-C</td>
<td>−0.085</td>
<td>52,933</td>
<td>−0.062</td>
<td>55,542</td>
</tr>
<tr>
<td>Ln(CRP)</td>
<td>−0.165</td>
<td>47,596</td>
<td>−0.146</td>
<td>49,334</td>
</tr>
<tr>
<td>Lp-PLA2</td>
<td>−0.147</td>
<td>52,772</td>
<td>−0.121</td>
<td>55,264</td>
</tr>
</tbody>
</table>

TGs, triglycerides.
*P = .18, all other P < .0001.

Figure 1 Relationship between the omega-3 index (mean [SD] of 5.2% [2.0%]) and biomarkers shown using splines with 95% confidence bands. Upper bounds of biomarkers are about their mean + 3SD.

Heterogeneity of Low-Density Lipoprotein Particle Number at Normal Concentrations of Lipoprotein (a) in Hyperlipidemia Patients

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Synopsis: Lipoprotein (a) [Lp(a)] is used commercially as a marker to predict cardiovascular disease. The clinical utility of routine measurement of Lp(a) is unclear. Furthermore, limited data are available that can be used to evaluate the relationship between low-density lipoprotein particle number (LDL-P) and Lp(a) concentration.

Purpose: To determine the correlation between LDL-P and Lp(a) in an unselected population of patients undergoing lipid analysis.

Methods: Cases comprised subjects with at least one of the following diagnoses in the database as reported by the ordering physician: hyperlipidemia, clinical coronary heart disease, diabetes mellitus, or symptomatic carotid artery disease. LDL-P levels were analyzed by nuclear magnetic resonance (NMR) spectroscopy. Lipids and Lp(a) were measured by standardized automated methods, and LDL cholesterol was calculated by the Friedewald equation. The query for the NMR and Lp(a) were obtained from January 1, 2009, through October 31, 2011, with a sample size of 3024 subjects.

Results: Among the 3024 subjects, the mean age was 60 ± 16 years (51% female). The mean Lp(a) concentration was 83 mg/dL, and the mean LDL-P concentration was 1389 nmol/L. There was no correlation between Lp(a) and LDL-P levels ($r^2 = 0.0012$). At Lp(a) levels of less than