RESEARCH LETTER

Levels of Plasma trans-Fatty Acids in Non-Hispanic White Adults in the United States in 2000 and 2009

To the Editor: Levels of trans-fatty acids (TFAs) in blood come from natural sources, such as milk, and industrial sources, such as partially hydrogenated vegetable oils. Dietary intake of TFAs increases low-density lipoprotein cholesterol (LDL-C) and has other adverse metabolic effects.1 Changing to a diet low in TFAs may lower the LDL-C level and decrease the risk for cardiovascular disease. To assist consumers, the Food and Drug Administration amended its regulations in 2003 to require that TFA content be declared on the nutrition label of foods and dietary supplements.2 Some community and state health departments have required restaurants to limit TFAs and reductions have been shown in supermarket and restaurant products.

The public health impact of these changes on TFA blood levels in the population is unknown. A preliminary study was conducted to determine plasma concentrations of TFAs in a subset of non-Hispanic white adults in the National Health and Nutrition Examination Survey (NHANES) in 2000 and 2009.

Methods. NHANES is a cross-sectional survey of the noninstitutionalized civilian population of the United States performed annually with a complex multistage probability design, weighted to be nationally representative.3 Half of the white persons aged 20 years or older who had a morning fasting blood sample in 2000 and 2009 were randomly selected. The protocol was approved by the National Center for Health Statistics Ethics Review Board and written informed consent was obtained.

Four TFAs (elaidic acid [C18:1n-9t], vaccenic acid [C18:1n-7t], linolelaidic acid [C18:2n-6t,9t], and palmitelaidic acid [C16:1n-7t]) were measured in plasma stored at −70°C, following previously described procedures.4,5 These are the 4 major TFAs and provide a reasonable representation of TFAs in blood.6 Their presence in blood cannot be used to distinguish food intake from natural vs industrial sources. There are no established reference ranges for TFA levels.

Statistical analysis was performed using SAS version 9.2 (SAS Institute Inc) and SUDAAN version 10.0 (Research Triangle Institute) by calculating weighted geometric means of the TFAs and the sum of the 4 TFAs using log-transformed data and single-year replicate NHANES examination weights. In addition, differences in the geometric means of the TFAs and the sum of the 4 TFAs from 2000 to 2009 and their 95% confidence intervals were calculated.

Table 1. Characteristics of Fasting Non-Hispanic Whites Aged 20 Years or Older

<table>
<thead>
<tr>
<th>NHANES Year of Specimen Collection</th>
<th>2000 (n = 229)</th>
<th>2009 (n = 292)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proportion of males, % (SE)</td>
<td>47.2 (2.7)</td>
<td>47.1 (2.7)</td>
</tr>
<tr>
<td>Weighted age, median (range), yr</td>
<td>45 (20-83)</td>
<td>46 (20-80)</td>
</tr>
<tr>
<td>Body mass index, median (interquartile range)</td>
<td>26.4 (22.4-30.9)</td>
<td>27.5 (23.9-31.6)</td>
</tr>
<tr>
<td>Cholesterol, mean (SE), mg/dL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low-density lipoprotein1d</td>
<td>128.2 (2.7)</td>
<td>119.2 (2.3)</td>
</tr>
<tr>
<td>High-density lipoproteind</td>
<td>49.6 (1.7)</td>
<td>55.8 (2.1)</td>
</tr>
<tr>
<td>Triglycerides, geometric mean (SE, mg/dL)</td>
<td>131.1 (5.2)</td>
<td>109.3 (5.8)</td>
</tr>
</tbody>
</table>

Abbreviation: NHANES, National Health and Nutrition Examination Survey.

Table 2. Levels of trans-Fatty Acids in Fasting Non-Hispanic Whites Aged 20 Years or Older

<table>
<thead>
<tr>
<th>No.</th>
<th>Geometric Mean (95% CI), µmol/L</th>
<th>No.</th>
<th>Geometric Mean (95% CI), µmol/L</th>
<th>Difference in Geometric Mean (95% CI), µmol/L</th>
<th>Decrease, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaccenic acid</td>
<td>229</td>
<td>43.7 (39.1-48.2)</td>
<td>291</td>
<td>19.4 (16.9-21.9)</td>
<td>24.3 (19.6-29.0)</td>
</tr>
<tr>
<td>Elaidic acid</td>
<td>229</td>
<td>38.2 (33.0-43.4)</td>
<td>292</td>
<td>14.0 (11.6-16.3)</td>
<td>24.2 (19.1-29.3)</td>
</tr>
<tr>
<td>Palmitelaidic acid</td>
<td>229</td>
<td>7.9 (7.3-8.5)</td>
<td>291</td>
<td>4.0 (3.6-4.5)</td>
<td>3.9 (3.2-4.6)</td>
</tr>
<tr>
<td>Linolelaidic acid</td>
<td>227</td>
<td>2.6 (2.2-2.9)</td>
<td>290</td>
<td>1.3 (1.2-1.5)</td>
<td>1.3 (1.0-1.6)</td>
</tr>
<tr>
<td>Sum of trans-fatty acids</td>
<td>222</td>
<td>93.1 (82.5-103.6)</td>
<td>292</td>
<td>38.0 (33.7-44.3)</td>
<td>54.1 (43.4-64.7)</td>
</tr>
</tbody>
</table>

These single-year replicate National Health and Nutrition Examination Survey (NHANES) weights were used because fasting subsample weights were not available for this analysis.8 Slight differences due to rounding.
56%: 24.3 µmol/L [95% CI, 19.6-29.0 µmol/L]). Similar changes were seen in elaidic acid, palmitelaidic acid, and linoelaidic acid. The weighted geometric mean of the difference for the sum of all 4 TFAs was 54.1 µmol/L (95% CI, 43.4-64.7 µmol/L) or 58% lower in samples from 2009 compared with samples from 2000 (Table 2). Levels of LDL-C were lower in the samples from 2009 (119.2 mg/dL [3.09 mmol/L]) compared with the samples from 2000 (128.2 mg/dL [3.32 mmol/L]; Table 1).

Comment. This study is, to our knowledge, the first time information on TFAs in white adults in the US population has been examined. Plasma levels of TFAs were substantially lower in 2009 than in 2000. This may lead to a decrease in risk for cardiovascular disease in this subpopulation. Because this study was limited to only 2 years and only a few parameters, it does not allow for detailed assessment of the association with LDL-C level or other factors. These findings provide preliminary data on white adults only and cannot be generalized to other racial/ethnic and age groups. Further studies to address these limitations are ongoing.

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Author Contributions: Dr Vesper had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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Drafting of the manuscript: Vesper, Kuiper.

Critical revision of the manuscript for important intellectual content: Vesper, Mirel, Johnson, Pirkle.

Statistical analysis: Mirel, Johnson.

Obtained funding: Johnson, Pirkle.

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Study supervision: Vesper, Johnson, Pirkle.

Conflict of Interest Disclosures: The authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none were reported.

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Disclaimer: The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention or the Agency for Toxic Substances and Disease Registry.

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CORRECTIONS

Clarification of Conflict of Interest Disclosures: In the Editorial entitled “Hospitalizations for Heart Failure in the United States—A Sign of Hope,” published in the October 19, 2011, issue of JAMA (2011;305[15]:1705-1706), and in the Commentary entitled “A Proposed Model for Initial Assessment and Management of Acute Heart Failure Syndromes,” published in the April 27, 2011, issue of JAMA (2011;305[16]:1702-1703), the conflict of interest disclosure information for Dr Braunwald should have read as follows: Dr Braunwald reported receiving research support from Merck, AstraZeneca, Johnson & Johnson, Beckman Coulter, Eli Lilly, Roche Diagnostics, sanofi-aventis, Daiichi Sankyo, GlaxoSmithKline, Bristol-Myers Squibb, and Pfizer as well as participating in symposia, advisory board meetings, and/or consultancies for the following companies: Eli Lilly, Merck, Genzyme, Amorcyte, CVRx (no compensation), The Medicines Company, CV Therapeutics, Daiichi Sankyo, MC Communications, Ortho McNeil, Ikaria, Menarini International, CardioRents, and sanofi-aventis. Also in the Commentary, the conflict of interest disclosure for Dr Gheorghiade should have read as follows: Dr Gheorghiade reported that he received consulting fees from Bayer, Novartis, Sigma Tau, Johnson & Johnson, Takeda, Otsuka, and Medtronic. These 2 articles were corrected online. A letter regarding the differences in Dr Braunwald’s conflict of interest disclosures appears in this issue.

Incorrect Work Group Name: In the Caring for the Critically Ill Patient article titled “Enteral Omega-3 Fatty Acid, γ-Linolenic Acid, and Antioxidant Supplementation in Acute Lung Injury” published in the October 12, 2011, issue of JAMA (2011;305[14]:1574-1581), the work group name was incorrectly stated. The group name “NHLBI ARDS Clinical Trials Network,” which appeared at the end of the byline, and the phrase “NHLBI ARDS Network Participants,” which appeared before the list of network personnel at the end of the article, should have read “NHLBI Acute Respiratory Distress Syndrome Network of Investigators.” This article has been corrected online.