Dietary fat consumption and incidence of type 2 diabetes

Dietary fat and type 2 diabetes

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Data analysis will begin upon the approval. A draft of the manuscript will be distributed for internal circulation by January 2004.

It’s been hypothesized that dietary fat consumption may be important in the development of type 2 diabetes. Possible mechanisms include various fatty acids’ influence on insulin secretion, insulin signaling, and expressions of genes which encode the proteins related to lipogenesis. Insulin secretion may be impaired by lipotoxicity on beta-cell in the pancreas by high-fat diet. Insulin signaling can differ because dietary fatty acids and cholesterol change cell membrane composition, resulting in change of fluidity and insulin receptors. The genes known to be down-regulated by dietary fatty acids include pyruvate kinase, glucose 6-phosphatase, fatty acid synthase, and leptin and those known to be up-regulated include lipoprotein lipase and phosphoenolpyruvate carboxykinase.

Several prospective epidemiological studies, including the Nurse Health Study, the Iowa Women’s Health Study, and the Health Professionals Follow-up Study, have examined the association between dietary fat intake and the risk of developing abnormal glucose tolerance. However, the findings have been inconsistent as shown in the table below. Although the ARIC Study has a smaller sample size than the three previously mentioned comparable studies, the inclusion of a more comprehensive type 2 diabetes assessment, African-American participants, and both men and women in a community-based setting can contribute to the existing research in this area. Therefore, we propose to examine the association between baseline dietary fat consumption and the risk of developing type 2 diabetes. Furthermore, we propose to examine the association between baseline dietary fat consumption and the change of markers of insulin resistance among those who did not develop type 2 diabetes by Visit 4 of the ARIC study.
Table. Adjusted odds ratios for diabetes for those with the highest vs. the lowest quintile of intake

<table>
<thead>
<tr>
<th></th>
<th>Nurse Health Study^4</th>
<th>Iowa women’s Health Study^5</th>
<th>Health Professional Follow-up Study^6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total fat</td>
<td>n.s.</td>
<td>--</td>
<td>1.63 (1.35, 1.96)</td>
</tr>
<tr>
<td>Total animal fat</td>
<td>1.25 (1.08, 1.45)</td>
<td>n.s.</td>
<td>--</td>
</tr>
<tr>
<td>Total vegetable fat</td>
<td>0.67 (0.59, 0.78)</td>
<td>0.82 (0.70, 0.97)</td>
<td>--</td>
</tr>
<tr>
<td>Total saturated fatty acids</td>
<td>n.s.</td>
<td>1.74 (1.43, 2.11)</td>
<td></td>
</tr>
<tr>
<td>Monosaturated fatty acids</td>
<td>n.s.</td>
<td>n.s.</td>
<td>--</td>
</tr>
<tr>
<td>Polyunsaturated fatty acids</td>
<td>0.85 (0.75, 0.97)</td>
<td>0.85 (0.71, 0.98)</td>
<td>--</td>
</tr>
<tr>
<td>Long-chain n-3 fatty acids</td>
<td>--</td>
<td>n.s.</td>
<td>n.s.</td>
</tr>
<tr>
<td>Trans-unsaturated fatty acids</td>
<td>n.s.</td>
<td>n.s.</td>
<td>1.28 (1.07, 1.53)</td>
</tr>
<tr>
<td>Dietary cholesterol</td>
<td>1.42 (1.23, 1.65)</td>
<td>n.s.</td>
<td>--</td>
</tr>
</tbody>
</table>

5. **Main Issues/Hypotheses to be addressed:**
The following components of dietary fat intake independently increase the risk of incident type 2 diabetes and insulin resistance;
  a) Higher energy intake from total fat
  b) Higher ratio of animal fat to vegetable fat intake
  c) Lower polyunsaturated fatty acids intake, especially long-chain n-3 fatty acids
  d) Trans-unsaturated fatty acids

6. **Data (variables, timeline, source, inclusion/exclusion):**
Individuals with prevalent type 2 diabetes, missing diabetes status at baseline, and missing information on dietary fat consumption will be excluded from this analysis. Other variables needed are already available at the local center are: center, age, gender, race, dietary intake of nutrients, diabetes status and the time of its development, insulin, glucose, anthropometry, blood pressure, lipid profiles. Cox proportional hazards regression models will be constructed for multivariate analysis. Potential interaction with whole grain intake, dietary fiber intake, and total calorie intake will be examined for all hypotheses.

7.a. Will the data be used for non-CVD analysis in this manuscript? _X_ Yes  _No_

7.b. If Yes, is the author aware that the file ICTER02 must be used to exclude persons with a value RES_OTH = “CVD Research” for non-DNA analysis, and for DNA analysis RES_DNA = ‘CVD Research” would be used? _X_ Yes  _No_  
(This file ICTDER01 has been distributed to ARIC PIs, and contains the responses to consent updates related to stored sample use for research.)

8.a. Will the DNA data be used in this manuscript? _Yes  _No_

8.b. If Yes, is the author aware that either DNA data distributed by the Coordinating Center must be used, or the file ICTDER01 must be used to exclude those with the value RES_DNA = “No use/storage DNA”? _Yes  _No_

9. The lead author of this manuscript proposal has reviewed the list of existing ARIC Study manuscript proposals and has found no overlap between this proposal and previously approved manuscript proposals either published or still in active status. ARIC
Investigators have access to the publications lists under the Study Members Area of the web site at: [http://bios.unc.edu/units/cscc/ARIC/stdy/studymem.html](http://bios.unc.edu/units/cscc/ARIC/stdy/studymem.html)

Yes ☒  No ☐

MS282 proposed the study of association between dietary fatty acids intake and insulin.
Reference List


