excluded on that basis). Like Carlsson et al. (1), we also excluded subjects whose body mass index had decreased from ≥25 to <25 between any two time points used in the analysis (n = 125) and subjects known to have diabetes (n = 153). The average age of the 1,664 subjects included in our analysis was 54 years (range, 42–78 years); 94 percent were White, and the majority of the remaining 6 percent were Black. Of the 1,664 subjects, 1,345 had normal glucose tolerance, 205 had impaired glucose tolerance, and 114 were found to be diabetic.

Regarding the relation of body mass index and waist circumference to risk of impaired glucose tolerance or diabetes mellitus, our results (table 1) generally agreed with those presented by Carlsson et al. (1) in their table 1. However, we found little to suggest that duration of overweight was independently related to risk. Although our cell sizes were limited, they exceeded many of those on which Carlsson et al. based their analysis. Further adjustment of our results for serum dioxin level or military occupation had essentially no effect (not shown).

The younger age of the Swedish subjects (35–56 years) compared with our subjects (42–78 years) might have accounted for the difference in findings. Therefore, we fitted models similar to those shown in table 1 (lower panel) with an interaction term for duration of overweight (ordinal variable) × age (continuous variable). The only suggestion of a stronger effect of weight history among younger subjects was for the risk of diabetes, where the p value for interaction was 0.20. Therefore, the reason for the differing results in the two studies remains unclear.

### Table 1. Odds ratios for the associations of overweight with impaired glucose tolerance and diabetes mellitus, Air Force Health Study, 1992*

<table>
<thead>
<tr>
<th>Variable</th>
<th>No family history of diabetes</th>
<th>Family history of diabetes</th>
<th>Diabetes mellitus†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. with normal GT</td>
<td>No. with impaired GT</td>
<td>Odds ratio</td>
</tr>
<tr>
<td>Present BMI‡,§</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤24.9</td>
<td>219</td>
<td>17</td>
<td>1</td>
</tr>
<tr>
<td>25.0–27.9</td>
<td>381</td>
<td>35</td>
<td>1.2</td>
</tr>
<tr>
<td>≥28.0</td>
<td>464</td>
<td>101</td>
<td>2.8</td>
</tr>
<tr>
<td>Present waist circumference (cm)¶</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;92</td>
<td>282</td>
<td>17</td>
<td>1</td>
</tr>
<tr>
<td>92–105</td>
<td>588</td>
<td>71</td>
<td>1.4</td>
</tr>
<tr>
<td>≥106</td>
<td>194</td>
<td>65</td>
<td>2.4</td>
</tr>
<tr>
<td>Duration of overweight</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(BMI ≥25.0) (years)¶</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0‡</td>
<td>219</td>
<td>17</td>
<td>1</td>
</tr>
<tr>
<td>&gt;0–9</td>
<td>190</td>
<td>20</td>
<td>0.9</td>
</tr>
<tr>
<td>10–20</td>
<td>267</td>
<td>29</td>
<td>0.6</td>
</tr>
<tr>
<td>&gt;20</td>
<td>388</td>
<td>87</td>
<td>0.9</td>
</tr>
</tbody>
</table>

* All odds ratios were adjusted for age (continuous variable) and race (White vs. Nonwhite).
† Results for diabetes were adjusted for family history of diabetes.
‡ GT, glucose tolerance; CI, confidence interval; BMI, body mass index.
§ Weight (kg)/height (m)^2.
¶ Odds ratios for waist circumference and duration of overweight were adjusted for 1992 body mass index as a continuous measure.
# Body mass index less than 25.0 at all three time points.

REFERENCES

Matthew P. Longnecker  
Epidemiology Branch  
National Institute of Environmental Health Sciences  
Research Triangle Park, NC 27709

Joel E. Michalek  
Air Force Research Laboratory  
Brooks Air Force Base  
San Antonio, TX 78235

THE AUTHORS REPLY

We thank Drs. Longnecker and Michalek (1) for their interest in our work. We also appreciate the opportunity to compare our results (2) with theirs. We have no clear-cut explanation for the discrepancy between the results of the two studies; perhaps chance is the most likely explanation.

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However, there are some differences between the studies that could have been potentially important to the outcomes. First, Longnecker and Michalek performed a prospective cohort study (1), whereas our study (2) was cross-sectional. Consequently, while previous weight in the US study was based on measurements made in the past, we used retrospective information based on self-reports. Second, as the authors pointed out, they investigated subjects in an older age range (42–78 years compared with 35–56 years in our study), which resulted in a prevalence of diabetes mellitus and impaired glucose tolerance almost twice that found in our study. Third, Longnecker and Michalek compared subjects with durations of overweight of <10, 10–20, and ≥20 years, whereas shorter duration periods were compared in our study: 0–4, 5–9, and ≥10 years. Fourth, we adjusted for confounding due to degree of obesity by including present weight in the regression model; in the study by Longnecker and Michalek, body mass index was included. Together, these differences may not account entirely for the discrepancy between results, but they do contribute to the explanation to some extent.

The concept of an influence of weight history is not new. Previous studies in Pima Indians (3) and in Israelis (4) and a recent Japanese study (5), in accordance with our study (2), have indicated an independent effect of duration of obesity on the risk of type 2 diabetes. However, further research is needed to determine the effect of long-term obesity, especially considering the lack of association presented in this study of US military veterans (1). We plan to begin a follow-up study by the year 2002, which should add important information to our findings. Additionally, we have recently finalized data collection for a corresponding study of 5,000 women; thus, we will soon have the opportunity to analyze this issue among women.

REFERENCES


Sofia Carlsson
Per-Gunnar Persson
Division of Epidemiology
Stockholm County Council
Karolinska University Hospital
S-171 76 Stockholm
Sweden

Valdemar Grill
Department of Molecular Medicine
Karolinska Institute
S-171 77 Stockholm
Sweden

ERRATUM

RE: “DIETARY FLAVONOIDS INTAKE AND RISK OF CARDIOVASCULAR DISEASE IN POSTMENOPAUSAL WOMEN”

Because of an error in the person-years calculation in the paper by Yochum et al. (1), incorrect results were presented. The information reflected person-years for 1994, with cardiovascular disease deaths only partially calculated for 1995. Correct analyses have been performed, and revised tables 2 and 3 will be published on the Journal website (http://www.jhsph.edu/Publications/JEPI/erratum.htm).

The impact of this error on the overall conclusions was minimal; the new analyses showed slightly wider confidence intervals, and previously nonsignificant p for trend values moved closer to 1 in several instances. However, the association between overall flavonoid intake and reduced numbers of deaths from cardiovascular disease remained, as did the strong dose-response relation with intake of broccoli.

The authors and the Journal regret the error.

REFERENCE