Re: Nutritional and Socioeconomic Factors in Relation to Prostate Cancer Mortality: a Cross-national Study

In the discussion section of the recent ecologic mortality study of prostate cancer by Hebert et al. (1), several misleading statements are made concerning the cohort study of dietary and other lifestyle factors and prostate cancer among Seventh-day Adventist men that was published nearly 10 years ago (2). Specifically, Hebert et al. state that, in regard to the dietary findings presented in that study, “These results need to be interpreted cautiously because the authors do not provide details regarding their dietary assessment methodology.” However, in a companion paper published in the same issue as the prostate cancer report, details were provided for the methodology of the entire cohort study, including the details of the dietary assessment methodology (3). In addition, the development of the dietary assessment technique used in the study was described years earlier, at the inception of the cohort study (4). Hebert et al. add that “their results for intake of other foods (e.g., beef, poultry, and milk) do not agree with the majority of the prostate cancer literature.” This statement is untrue. Not only did our results suggest a role for moderately increased prostate cancer risk in association with increasing animal fat intake (multivariate adjusted relative risk for daily meat intake = 1.41; 95% confidence interval = 0.79–2.51), it was one of the first studies to identify a protective relationship between intake of several types of vegetables, notably beans, lentils, peas, raisins, and tomatoes and prostate cancer risk. The findings regarding tomato intake have been supported in several studies, particularly in other well-designed and conducted prospective cohort studies of prostate cancer incidence (5). Finally, Hebert et al. also erroneously state that “The results of that study may be unique because their sample comprised a homogeneous group of predominantly white middle-class vegetarian men.” Indeed, the Adventist cohort was characterized by a wide degree of variation in socioeconomic status [see Table 7 in (3) for the distribution of education and occupation variables in male members of the cohort]. Additionally, dietary habits ranged from the pure vegetarian (i.e., the vegans) to the “omnivorous” diet in which several kinds of animal products are consumed to one degree or another. The Seventh-day Adventist church recommends, but does not require, church members to be vegetarian. An inspection of the person-years distributions of the meat-intake variables in the tables of our paper supports this wide range of dietary habits.

I hope this clarifies the interpretation of the findings from the Adventist Health Study with regard to diet and prostate cancer risk. The results from any international ecologic mortality study of a disease, such as prostate cancer that relies on food disappearance data as the “exposure variable,” suffers from a multitude of methodologic limitations, which the authors have correctly noted. In addition, prostate cancer mortality as an outcome is influenced by several predictive factors, including screening practices, survival, and lifestyle as much as by biologic factors that
may be etiologically relevant. However, a prospective, incidence-based cohort study, such as the Adventist Health Study, in which dietary data are collected at the individual level before the development of disease is more desirable in that it is rigorous and methodologically sound and offers much in the way of etiologic insight.

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REFERENCES


NOTE

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RESPONSE

We appreciate Dr. Mills’ response to our recent article (1) and would like to clarify the passages to which he refers and to respond to specific comments about methodologic issues pertaining both to his study (2) and to ours (1).

First, Dr. Mills mentions that details of the methods for the Adventist Health Study (AHS) are contained in another paper (3). However, the description of the diet assessment method is limited, referring to “a more detailed discussion...” [reference (15) in (3)]. Apparently, that paper was never published. In his letter, Dr. Mills refers to an abstract (4), which also is lacking in detail sufficient to assess agreement in data derived from the Food Frequency Questionnaire and the comparison method. In the absence of a published record of these methods, we believe that our statement is accurate.

Second, the matter of interpreting the effect of animal products on prostate cancer resulted from differences in how data were combined in the paper by Mills et al. (2) versus ours (1). The passage in our article to which Dr. Mills referred discussed the effect of fish. In our data, energy from fish was inversely associated with prostate cancer mortality. Therefore, it has an effect opposite to that of other foods of animal origin. The statistically nonsignificant odds ratio of 1.41 that Dr. Mills quoted from his paper (2) combined fish with meat and poultry. Mills et al. (2) reported that “various types of beef... were not associated with prostate cancer risk” and found only “a suggestive relationship between reported consumption of meat, poultry or fish as a composite variable,” acknowledging the absence of statistically significant results for these foods in their data.

Third, slightly before the study by Mills et al. (2) appeared, we had published several papers drawing attention to methodologic issues that could distort diet–disease relationships. These issues concerned dietary assessment and selection of groups for study that are homogeneous for dietary factors of putative etiologic importance (5–7). Relevant to this point, Table 2 in (3), which presents data on household response rates to mailings of the 1974 census questionnaire by ethnic group, does not support Dr. Mills’ statement regarding the distribution of education and occupation. Although the AHS population may be different in some ways from the general U.S. population, it is a relatively homogeneous group of predominantly well-educated, non-Hispanic Whites [see Table 7 in (3)]. In terms of dietary exposures, the AHS cohort represents a small portion of the range available in our study (1). Furthermore, while we acknowledge the limitations of cross-national studies, the use of food inventory data does avoid the “multiple problems potentially affecting estimated reliability and validity” based on dietary self-report data, as noted by the AHS investigators (3).

To understand the relationship between diet and cancer, we need results from all types of studies, with their vari-