Letter to the Editor

Salt Intake and Stomach Cancer: Some Contrary Evidence

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Considerable evidence has accumulated linking high salt intake to the risk of developing stomach cancer (1, 2). In addition to a geographic correlation of areas showing high salt intake with those having high stomach cancer mortality (3) and evidence incriminating preserved, often salty foods (4, 5), case-control studies in which individuals have been asked about the routine addition of salt to food (4, 6), preference for salty food (7, 8), or salt consumption (9, 10) have often shown the association of higher salt intake with stomach cancer, in study groups in the United States and other countries.

We recently completed a case-control study of stomach cancer based on persons who had had serum placed in frozen storage in 1964–1969 at multiphasic health checkups. This showed that prior infection with Helicobacter pylori was associated with considerably increased risk (11). In the self-administered checkup questionnaire there was one question about salt intake: "Do you usually salt food before tasting it?" This permitted an attempt to replicate findings of other studies implicating the self-reported routine addition of salt. Partially allaying our skepticism about assessing salt intake by this single crude question, subjects of another study who answered yes showed an increased risk of developing hypertension (12).

The study group consisted of 200 exameses randomly selected from 246 who subsequently developed stomach cancer by the end of 1988, according to computer-stored records obtained from the Kaiser Permanente hospitalization files and the San Francisco Bay Area Surveillance, Epidemiology, and End Results Registry (13) and 200 comparison subjects who did not develop cancer and who were individually matched to the cases for age (born in same 5-year interval, e. g., 1900–1904), date of examination (same month and year), sex, race, and location of examination (Oakland or San Francisco). Review of available pathology slides led to subclassification by histological type and location of the tumor (11).

There was no evidence that routine salting led to increased risk. In fact, for all subjects with available data and for intestinal-type adenocarcinoma, risk was lower in the routine salters (Table 1). This finding persisted when salt use was entered into a logistic regression analysis along with Helicobacter pylori infection as independent variables, using all subjects with serum (relative risk estimates: routine salting, 0.57, 95% confidence interval, 0.34–0.94; Helicobacter pylori infection, 2.62, 95% confidence interval, 1.36–4.41). To make sure that these results were not due to errors in computer programming or data entry, relative risk was confirmed by hand tally, and the written answers in 40 original questionnaires (10 cases and 10 controls each, from Oakland and San Francisco) were compared with the data in computer storage, with complete agreement.

Given the contrary published evidence, it is tempting to attribute this unexpected finding to chance and ignore it. However, to do so would contribute to publication bias (14). There is no a priori reason to believe that the answers provided by the subjects of this study are less accurate than those of previous studies. In fact, to our knowledge, this is the only case-control study of salt intake and stomach cancer in which the information was obtained from the cases before stomach cancer was diagnosed, thus eliminating the possibility of recall bias. We must conclude that in this U.S. study population heavy salt intake, as evidenced by self-reported usual salting of food before tasting it, was not associated with increased risk of developing stomach cancer.

References

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