CORRESPONDENCE

Re: Oral Contraceptives and Breast Cancer

With reference to the commentary by Thomas (1), we would like to make the following comments.

We were surprised to note that the experimental model of progesterone induction of mammary cancer in mice was not mentioned or quoted in the commentary by Thomas. We developed this model in 1986; since then, our results have been accepted for publication in international cancer journals, including the Journal of the National Cancer Institute. We have, therefore, written an editorial, which has just appeared in Spanish in a journal in Argentina (2). The editorial contains a description of our model, its origin, and a chronological enumeration of our results and the publications in which they appear. We will provide copies of an English translation of this editorial upon request.

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References

Note
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Re: Oral Cavity Cancer in Non-Users of Tobacco

The findings of Ng et al. (1) that are related to beer drinking among men as a possible risk factor in squamous cancer of the oral cavity tend to support our findings (2) among a population of veterans who are heavy drinkers and smokers. In our preliminary study with 181 cases and 497 controls (2), 82% of the case patients drank 6 or more whiskey equivalents (WE) of alcohol per day. (The quantity of alcohol in 1 WE is approximately equivalent to that in 1 oz of whiskey, 12 oz of beer, or 4 oz of 10%-12% dry wine.) Because of this heavy consumption, we extended the categories of alcohol consumption beyond the “3 or more” category used in many other studies. In adjusting for smoking, we found a relative risk of 7.3 for those who consumed 6-9 WE of alcohol as whiskey only or as whiskey predominantly, compared with a relative risk of 24.7 for those who consumed equivalent WE of beer and/or wine.

There were few wine drinkers in this group.

The increased risk of beer drinking has been further supported in a continuation of our preliminary study (2); our recent analysis of data on 359 cases and 2280 controls will be published soon (3).

Nitrosamines have been suggested as possible etiologic agents. If memory serves me correctly, soon after publication of our findings in 1981, I received a communication from representatives of the beer industry indicating that the producers had decreased the nitrosamine content of beer considerably. If this reduction was implemented, studies accruing patients whose major beer consumption occurred in the last 10-15 years should begin to show a diminishing risk if, in fact, nitrosamines are the culprit.

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References