LETTERS TO THE EDITOR

Longevity of Nonsmoking Men and Women

Many attempts have been made to determine factors which can explain the difference in male-female longevity. Possible explanatory factors include genetic differences, differences in health care use, behavioral differences in drinking and smoking habits, and behavioral differences in aggression and coping with stress.

The study by Miller and Gerstein (*1*) purports to show that all of the male-female differences in longevity can be attributed to higher rates of smoking among males. This contradicts a large body of evidence which indicates that differential smoking accounts for between one-third and one-half of the sex difference in longevity, for example, Waldron (*2*). Miller and Gerstein's work raises many issues which require more careful consideration.

Estimates of death rates which are needed for computing life expectancies are subject to sampling and nonsampling errors in estimating the populations at risk and the number of deaths. Miller and Gerstein used a 2 percent simple random sample to estimate the proportion of nonsmokers in their population. The possible effects of noncoverage error and of variance due to sampling were ignored. The sample frame for the population data covered only about 75 percent of the target population, but the estimates were applied to the entire population. Because some of the age-sex groups are fairly small the sampling errors may be relatively large. For example, the estimated percent of male nonsmokers was 36.8 percent for ages 85 years and over. Using the estimated population published in the tables in their article it can be calculated that an approximate 95 percent confidence limit is 15–60 percent nonsmokers for men 85 years and over. Similarly, when these highly variable population estimates are used to compute mortality rates, some ambiguous results are apparent, particularly at the older ages which are crucial for the estimation of life expectancy. Thus, given the sampling variation for the population estimates, the estimated life expectancy for men at age 85 could range from 5.4 years to 21.4 years.

There can be no question that sampling error in the population estimates has a substantial effect on the life table estimates. But another error, not due to sampling, may be even more important, that is, the determination of deaths of nonsmokers. Information on smoking status was obtained from relatives for only 63 percent of the deaths. There was no adjustment for nonresponse; decedents for which no information could be obtained (37 percent of all deaths) were essentially classified as "smokers." Thus, the number of deaths to nonsmokers was considerably underestimated, their death rates were similarly underestimated, and their life expectancies were therefore overestimated. Of crucial importance is the fact that Miller and Gerstein did not consider whether the biases in their estimates were the same for both males and females. For females, Miller and Gerstein classified about 65 percent of the population and 55 percent of the deaths as nonsmokers. For males, they classified about 30 percent of the population but only about 10 percent of the deaths as nonsmokers. Because of differential nonresponse rates, it is likely that deaths for male nonsmokers may have been underestimated to a significantly greater extent than deaths for female nonsmokers.

Thus, we feel that the conclusion that all the male-female difference in longevity was due to differences in smoking is not warranted since the estimated differences may have resulted from the inherent variability present in their small data sets and from them ignoring the potential for differential biases in their methodological approach. Within these limitations their results are actually quite consistent with those previously presented in the literature that differential smoking accounts for between one-third and one-half of the sex difference in longevity.

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2. Waldron, I.: Why do women live longer than men? Soc Sci Med 10: 349–362 (1978).

Others Would Get Similar Longevity Results If They Took Greater Care

Dr. Feinleib and Dr. Luoto present two methodological criticisms of the Miller-Gerstein report (*1*). They also state that our results both "contradict a large body of evidence" on smoking and the male-female longevity difference and are "actually quite consistent" with this same evidence. We will first discuss the methodological questions, then comment on the overall relation of our conclusions to the previous literature.

The first issue is sampling variability. Feinleib and Luoto illustrate the width of confidence intervals that result from data from small subsamples such as our smallest cohort, males age 85 and over. Vitality and other data on those aged 85 and older are notoriously unreliable in demographic sample analyses, and the observed results for this age category are often omitted or replaced by figures derived from large standard data bases. Rather than use this highly variable data in our life expectancy calculations, we obtained life expectancy figures for the 85+ cohort from the U.S. Bureau of Vital Statistics, as noted in the table in our report. The criticism based on this illustration is, therefore, inappropriate.

Table 1. Major causes of higher mortality in men

Ratio of male to female death rates	Cause of death	Male death rate (deaths 100,000 population)	Female death rate[1] (deaths 100,000 population)
5.9	Malignant neoplasm of respiratory system, not specified as secondary	50.1	8.5
4.9	Other bronchopulmonic disease (71 percent emphysema)	24.4	5.0
2.8	Motor vehicle accidents	39.4	14.2
2.7	Suicide	15.7	5.8
2.4	Other accidents	41.1	17.4
2.0	Cirrhosis of liver	18.5	9.1
2.0	Arteriosclerotic heart disease, including coronary disease	357.0	175.6
1.6	All causes	1081.7	657.0

This table lists all causes of death which had a sex mortality ratio of 2.0 or more and were responsible for at least 1 percent of all deaths in the United States in 1967. These causes of death are responsible for three-quarters of the sex differential in mortality.

[1] Female death rates have been age-adjusted using the age-specific death rates for females and the age distribution for males to calculate the death rate which would be expected for a population of females that had the same age distribution as the male population. Thus, the male and female death rates are directly comparable and are not affected by the higher proportion of females at older ages.

Reprinted with permission from Social Science and Medicine (*2*).

The second criticism, concerning differential nonresponse, is based in part on the assertion that we "essentially considered" all nonrespondents to be smokers. This assertion is erroneous. We assumed that the percentage of nonsmokers among nonrespondents was the same as among respondents. In order to test the effect of this assumption, we performed a sensitivity analysis; we recalculated the male life table, assuming that nonsmoking among nonrespondent males was actually double that of respondent males. The life table results were not substantially different, indicating that the possibility of differential nonresponse does not lessen confidence in our conclusion.

More broadly, Feinleib and Luoto cite as exemplary Waldron's 1976 review of the literature (*2*), which concluded that "very roughly" one-third of the difference between male and female death rates may be due to men's cigarette smoking, one-sixth to a greater prevalence of coronary-prone behavior pattern, one-twelfth to higher alcohol consumption (increased accidents and cirrhosis), and one-twelfth to physical hazards related to employment (increased accidents and lung cancer). We reproduce Waldron's table 1 listing causes of death accounting for three-fourths of all male-female death rate differences.

With external causes removed, the male-female longevity difference is dominated by smoking-related diseases: respiratory cancer, pulmonary obstructive disease, and heart disease. The issue is what proportion of the male deficit in life expectancy is owed to cigarette smoking. We have pointed out the relative inaccuracy of the classification procedures which many studies, including those based on National Center for Health Statistics data (*3,4*), have used to assign smoking categories. These procedures place some smokers in the nonsmoking category and vice versa, which reduces the estimated mortality difference due to smoking. It is no surprise that the strongest positive finding in the massive MRFIT study (*5*) was the reduction in mortality risk incident to proven sustained abstinence from smoking.

We encourage other researchers interested in longevity differences and related epidemiological matters to take greater care to assure accurate data and use appropriate smoking categories. When this is done, we expect that their results will be very similar to those we obtained.

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Knowledge of Health Does Not Always Begin With Disease, Dr. Krause

Bravo to Dr. Edward Brandt for his spirited response to Dr. Richard Krause's broad-based attack on health promotion and disease prevention (Public Health Reports, vol. 98, pp. 529–530 and 531–535). Dr. Brandt stated the case for prevention clearly, concisely, and well. Of course it is too bad that we are in an era of runaway military spending such that prevention and basic research must quarrel with each other over the leftovers of Federal monies. The best solution, of course, would be to do as much of both as is necessary. That might require reducing military spending by up to 2 percent. Lacking that solution, however, there are several additional points that should be made about Dr. Krause's paper.

• Health, as Hippocrates and many successors have correctly noted, is a positive state, not merely the absence of disease. Therefore, although the "beginning of health" can be "to know the disease," knowledge of health does not always begin with disease and it never ends simply with knowledge of disease.