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Miller Replies

The comments by Bowers, Allen, and Hickey referring to our study (*1*) on the effects of cigarette smoking on the male-female longevity difference is a fine example of misuse of biostatistics. The writers reintroduce the "constitutional hypothesis" proposed several decades ago by the distinguished statistician Fisher (*2*). His argument with the Doll-Hill report (*3*) on the damaging effects of smoking was that inherited factors—not cigarette smoking—might be the real cause of the higher mortality rates of smokers over nonsmokers. He postulated that since cigarette smoking is a self-selection process, those with weaker constitutions might be more likely to smoke, thus creating higher mortality rates for smokers. Fisher advocated more research on this topic to determine the truth of his hypothesis. The bulk of modern epidemiological and other biomedical research on smoking has shown his hypothesis to be invalid (*4,5*).

Bowers et al. then cite a number of reports showing significant differences in male-female longevity. They overlook research showing little difference in male-female longevity in societies where there is an absence of smoking (*6,7*) and in the latest reports on third world countries—such as China, India, Indonesia, Iran, and Pakistan—where the men live as long or longer than the women (*8,9*).

These writers proceed through the whole maze of theoretical verbiage of highly questionable validity. They suggest that through the behavioral regulation of physiological homeostasis there is a therapeutic effect of smoking on ulcerative colitus which results in normalizing the levels of biogenetic monoamine neurotransmitter hormones. They also imply that, through biobehavioral processes and psychoendocrinology, personal gratification brought about by smoking may be "highly advantageous to the survival of the species."

Let us now consider the scientific evidence for two of our critics' most cogent arguments: (*a*) that smoking counteracts the symptoms of ulcerative colitus and (*b*) that the "expected benefits of reduced smoking" from the Multiple Risk Factor Intervention Trial (MRFIT) study were few, if any.

Ulcerative colitus is a fairly rare disease and it is hardly fatal. Analysis of the articles cited shows that nicotine alone without smoking will eliminate the spasms of ulcerative colitus.

With regard to the MRFIT report (*10*), Bowers et al. apparently overlooked the final results as well as the main body of the report, where the principal investigators concluded " . . . men who stopped smoking cigarettes had lower CHD [coronary heart disease] and total mortality than those who continue to smoke." The analysis of the MRFIT data showed a two-fold decrease in cardiovascular disease (CVD) for those who had stopped smoking in both groups.

The writers' suggestion that it is good for the species to ingest an addictive substance through smoking in order to counteract a rather rare ailment, while markedly increasing the risk of contracting diseases which are the leading causes of death, is totally unrealistic (*11–14*).

We therefore see no reason why the arguments of Bowers et al. based on such speculations should shake confidence in our findings or lessen anyone's confidence in the vast amount of research demonstrating that cigarette smoking is dangerous to one's health. We, of course, welcome further research on this important topic and agree with our critics that it should be free from bias and preconceived opinion. Unfortunately we must conclude that their efforts do not meet their own criteria.

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