EDITORIAL

Lung Cancer and Smoking Cessation: Patterns of Risk

Jay H. Lubin, William J. Blot*

Studies carried out over more than four decades show conclusively that cigarette smoking is the principal cause of lung cancer, accounting for over 100000 deaths from this cancer among U.S. men and women every year (1). Risk increases with intensity and duration of smoking, regardless of the type of tobacco (light or dark) or method of consumption (cigarettes, cigars, pipes, or water pipes) (2).

Just as convincingly, the recent 1990 Surgeon General's Report documents that those who stop smoking reduce their lung cancer risk (1). While it is clear that smoking cessation lowers risk, the precise patterns of this reduction with years since smoking cessation have not been fully defined. About 25% of adults in the United States continue to smoke (3), increasing the risk of lung cancer to themselves and, by providing a source of environmental tobacco smoke, to others. An additional 40%-50% of adults are former smokers (3). Accordingly, for public health reasons, it is important to precisely describe the excess risk with smoking cessation.

In this issue of the Journal, the analysis by Halpern et al. (4) of data from the American Cancer Society's Cancer Prevention Study II (CPS-II) helps to more fully describe patterns of excess risk after cessation of smoking. The results are important and provocative and can be viewed as a base for further analyses. They show that the relative reduction in risk increases the earlier in life that smoking is stopped. This result is consistent with prior reports demonstrating that relative risk (RR) of lung cancer declines with increasing years since smoking cessation (5). It has also been shown that the magnitude of the decline depends on the interaction between the number of years since smoking cessation and other factors. For example, the rate of decline in the RR with years since smoking was stopped is lower among long-duration smokers. The rate of decline may be lower among those who smoked a greater number of cigarettes per day or among those who inhaled with a greater frequency and inhaled more deeply, but further study of these factors, as well as the interactions of cessation and historical profiles of cigarette use, is needed (5).

Most epidemiologic analyses have focused on the pattern of RRs in relation to time since smoking cessation, generally finding a decline with years since individuals stopped smoking. Because the rate of lung cancer increases with age, a decline in the RR is compatible with a variety of patterns in the excess risk, compared with risk for those who have never smoked (never smokers): (a) The excess risk may decline. (b) The excess risk may remain fixed at the level occurring at the time of cessation. (c) The excess risk may continue to increase but at a reduced rate. (The excess risk patterns may, of course, manifest a combination of patterns. For example, the excess risk may remain fixed initially, the increase some time later.) Each of these three possibilities would generate a different estimate of the incidence of lung cancer among the general population. The data presented by Halpern et al. (4) suggest that the second and third patterns or variants of them, are most likely. The authors, however, were unable to adjust for the differences in age-specific lung cancer rates by birth cohort because there was a maximum of 6 years of follow-up in the CPS-II. In addition, since smoking data were collected only at study entry, they were unable to evaluate any changes in the subjects' smoking patterns during follow-up.

Using Poisson regression methods (6) and the data of person-years of smoking provided by Halpern et al. in Table 5 of their article, we estimate that lung cancer incidence increases in proportion to duration of smoking to the 4.5 power, assuming that all smokers begin to smoke at age 20. This finding agrees closely with an increase proportional to the fourth power of age in the British Physician's Study (7). However, in contrast to that study, in which lung cancer incidence in never smokers also increases with the fourth power of age, we estimate that lung cancer incidence in never smokers increases approximately proportional to age to the 5.5 power. This difference could be the result of limited data, although other factors such as exposure to environmental tobacco smoke could also play a role. A graphical presentation of the rise in risk among never smokers and current smokers using a log-log scale, in contrast to the arithmetic scale used by Halpern et al., is shown in Fig. 1. Rates are plotted by age for never smokers and by time since smoking was started for smokers.

The precise modeling of risk patterns with smoking cessation has implications in other areas of public health. Indeed, smoking has been shown to act synergistically with several other agents in increasing risk of lung cancer. In the case of radioactive radon gas, which is found in some underground mines and, at lower levels, in homes, the joint association of this exposure and smoking is consistent with a submultiplicative RR relationship (7). However, the joint pattern of risk after smoking cessation is largely unknown and has yet to be explored. Similarly, it is not clear how rapidly smoking cessation reduces the risk of lung cancer among individuals exposed to asbestos, another potent lung carcinogen (8).

In summary, because of the public health consequences of smoking cessation programs and the interrelationship of smoking and other lung carcinogens, there is a need for further research to characterize excess lung cancer incidence with smoking cessation. A particularly useful project would

*See "Notes" section following "References."
be the joint analysis of the major cohort data sets, which would offer an opportunity to statistically evaluate the consistency of risk patterns across study populations. Nevertheless, the information available today regarding the benefit of smoking cessation is unequivocal. Smokers of any age should be encouraged to quit. The resulting reduction in risk of lung cancer, not to mention other smoking-related diseases, becomes rapidly apparent and is long-lasting.

References


Notes

Affiliation of authors: J. H. Lubin, W. J. Blot, Biostatistics Branch, Division of Cancer Etiology, National Cancer Institute, Rockville, Md. Correspondence to: Jay H. Lubin, Ph.D., National Institutes of Health, 6130 Executive Blvd., Rm. 403, Rockville, MD 20852. Manuscript received February 12, 1993; accepted February 16, 1993.