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What Is the Relative Risk of Disease in Smokers?

The article on differences in reported relative risk of disease in smokers[1] found in this issue is an overview of 83 English-language population studies of the risk of circulatory disease (cardiovascular disease, coronary heart disease, myocardial infarction, and stroke), lung cancer, or chronic obstructive lung disease (COLD) that is attributable to tobacco consumption. The authors ascribe the differences in reports of the relative risk of smoking to a variety of factors that have been identified in the literature, including artifactual factors (misclassification of former smokers or differences in the age distribution of the population) and methodological differences (definitions of amount smoked, strength of tobacco, smoking duration, intensity of inhaling).

As the authors note, the article is not a meta-analysis. A meta-analysis requires definition of key variables that are identically specified by the selected studies and that permit aggregation of findings from the studies. Both a meta-analysis and the present study are constrained by the limits of what gets published (or to be more precise, what doesn't get published). However, as the authors of this article decided to compare methodologically disparate studies, they can only address in general terms the differences in reported relative risks across the 83 studies. As a consequence, the readers of this journal should look directly at the research[2,3,4,5,6] to find a useful, valid estimate of the relative risk of disease associated with smoking.

The purpose for which this study was conducted was to judge whether relative risks of smoking based on studies in one population may be applied justifiably to another population. The underlying premise of the study appears to be an assumption that genetics plays a role in the consequences of smoking. Few doubt the role of genetics in determining individual susceptibility to disease, but the authors do not develop the theme further. They do not consider somatic genetic changes attributable to smoking that might modify disease risk. Instead, the authors explain that known and measurable factors (summarized above) rather than "true biological differences" are likely to be responsible for reported variations in relative risk of disease associated with smoking across populations. In all fairness, given the constraints of their data, there is little opportunity to derive other conclusions from these data. As they reviewed population studies, any attempts to interpret individual factors into the data would confront the ecological fallacy.

The results of their analysis are sufficiently constrained that the authors conclude only that researchers should adopt relative risks calculated from a population that is similar to their own. This is altogether a reasonable and obvious decision. It is comforting that their discussion manages to discount much of the variability that they encounter in risk estimates from published studies. On the other hand, it is disturbing that the underlying risk of disease associated with smoking remains an elusive target.

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