Letters

Virus Hunting and the Scientific Method

The suggestion in the News briefing "Virology dead, says Duesberg" (14 Dec., p. 1514) that I should be "exempt from teaching any course that relies on the scientific method" because I favor the "innocent until proven guilty" approach is perhaps the most honorable discharge I have earned so far. Unfortunately, the view that according to "the usual scientific method . . . a hypothesis remains a candidate until it is disproven" can have serious consequences, in particular, if it is a candidate for a way to confront disease.

Take the currently popular virus-AIDS hypothesis. The virus remains to be proven guilty (*1*). But the hypothesis is currently the only candidate for a way to confront AIDS (*2*). This confrontation has produced no cure, no vaccine, a highly toxic antiviral medicine, and an infectious syndrome that does not spread from behavioral or clinical risk groups [the United States has now lifted its ban on visitors who test positive for the human immunodeficiency virus (HIV)], and all that for about $3 billion a year. Lately it looks as if there is even disagreement about how to prove HIV guilty, in particular, about what kind of microbial allies are needed to cause AIDS (*3*). In view of the emerging HIV schism, I wonder which candidate hypothesis professors should be "exempt" from teaching.

Other examples demonstrate that the ever-popular germ theory has at times "remained a candidate" far too long, until finally disproved at great cost to the affected people. In the United States tens of thousands died unnecessarily in the 1920s because pellagra was considered infectious by the U.S. Public Health Service, until Joseph Goldberger proved it to be a noninfectious vitamin B deficiency (*4*). Indeed, the disease was said to be transmitted by "poor hygiene" among corn farmers in the South—the primary risk group for pellagra (*4*). In Japan, at least 10,000 suffered in the 1960s and 1970s from a drug-induced neuropathy, including blindness, that had been misdiagnosed as a viral diease for more than 10 years (*5*). The pursuit of oncogenic viruses as the causes of cancer by me and by many of my learned retrovirology colleagues provides another example. Although it has generated such academic triumphs as viral oncogenes and reverse transcriptase, it has been a total failure in terms of clinical relevance to cancer, primarily because, with a very few exceptions, cancers are not infectious. Perhaps professors should not be exempt from questioning clinically unproductive hypotheses from a generation of virus hunters who have never seen a frontier outside the laboratory.

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Radon Risk and EPA

I would like to respond to Philip H. Abelson's editorial "Uncertainties about health effects of radon" (19 Oct., p.353). The information provided here will help to clarify some of the misconceptions about the potential health risks from radon and about the Environmental Protection Agency's (EPA's) Radon Action Program.

The Indoor Radon Abatement Act of 1988 sets a national, long-term goal of achieving indoor radon levels similar to ambient outdoor levels. This is not yet technologically achievable. Consequently, EPA is not planning to lower the current action level to ambient levels at this time. The cost of reducing elevated radon levels to EPA's current action level of 4 picocuries (pCi) will range from $500 to $1500 in most cases, not $10,000 per house.

The National Academy of Science's (BEIR IV) committee extrapolated lung cancer risks derived from epidemiologic studies of miners to project lung cancer risks for U.S. males and females (*1*). EPA's central estimate of approximately 20,000 annual lung cancer deaths is derived by using the relative risk models, in conjunction with lifetable analyses, presented in the reports of the BEIR IV committee and the International Commission on Radiological Protection (ICRP 50 report)(*2*). This estimate is not based on screening data from EPA measurement studies. Rather, the estimate was calculated by assuming that the average residential exposure is about 0.25 working level month (WLM) per year (about 1.3 pCi per liter) (*3*). This estimate of exposure is consistent with the estimates by Anthony Nero (*4*) of the theoretical frequency distribution of annual radon levels in homes. EPA is currently conducting a National Radon Residential Survey that will determine the national frequency distribution of annual radon levels in residences.

EPA believes priority should be given to identifying those areas where high radon levels are highly prevalent. Over the past 5 years, EPA and the U.S. Geological Survey have been working to develop a radon potential map using residential survey data and geological indicators. The map will identify those areas of the United States that have the highest potential for elevated indoor radon levels. It will not identify individual homes with elevated radon levels.

Testing is the only method currently available that will identify individual structures with high radon levels. Different levels can be found in adjacent homes, and homes with elevated levels can be found in areas of low radon potential. Consequently, EPA recommends that most homes be tested (typical cost is $10 to $30) to determine whether they have elevated radon levels. It should also be noted that homes with high levels are not "rare." On the basis of Nero's frequency distribution, we estimate that there may be several million homes with annual radon levels above 4 pCi per liter, and more than 100,000 homes with levels above 20 pCi per liter.

Abelson seems to be unconvinced that the BEIR IV committee findings (which are based on studies of several cohorts of individuals) demonstrate that there is a risk from radon. He urges a cautious epidemiological approach. However, he relies on an unpublished ecological study by B. L. Cohen as the basis for questioning whether residential radon is a significant problem.

Although EPA supports the need for further epidemiologic studies of indoor radon, these studies should be designed so that they are of value for risk assessment purposes. The strongest scientific evidence of a causal relationship between exposure and risk comes from analytical epidemiologic studies. Ecologic studies are not recommended for the study of residential radon risk(*5*).

Unlike case-control or cohort studies, Cohen's work is based on descriptive ecological studies that examine groups of people and data on average radon exposures. In these studies, there is no way to relate the level of radon exposure for an individual to that individual's health status. Nor do these studies provide a way to assess other lung cancer risks, such as smoking experience, that could significantly affect cancer rates in the study region.

A number of case-control studies of lung cancer risk and residential radon show some correlation between indoor radon and lung cancer mortality (*6*).