Letters

Greenhouse Gas Tax

The Briefing "Greenhouse gas tax" (News & Comment, 10 Jan., p. 154) contains the remark that "[the gas tax] would end up soaking consumers of about $95 billion a year." This is so misleading that it is untrue. The estimate of $95 billion is in line with other estimates I have seen of the same possibility. But that would not be the end of it—$95 billion is big money. It is more money than the corporate income tax raises and amounts to more than 40% of the fiscal 1990 deficit. That kind of money, once collected, doesn't just go away. Something has to be done with it and, in fact, it has to come back to the consumers who were "soaked" in one form or another. I suspect it would come back partly in the form of reduced personal income taxes, partly as increased government services (which we need), and partly as a reduction in the overhanging national debt. Such a collection is what we economists call a "transfer payment," which must benefit the recipient to the same extent that it hurts the payer. In short, a greenhouse gas tax would not "end up soaking consumers."

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Radon Risk in the Home

We disagree with Philip H. Abelson's portrayal (Editorial, 8 Nov., p. 777) of both the current scientific understanding of radon-induced lung cancer risk and the basis for the Environmental Protection Agency's (EPA's) radon policy. The radon policy of EPA is not based on data from one cohort study of uranium miners (the Colorado Plateau cohort) exposed to "huge amounts" of radon. Rather, it reflects scientific consensus developed through review of the extensive epidemiologic data on thousands of underground miners exposed to a broad range of radon concentrations. Studies of miners have been conducted in the United States, Canada, Australia, China, and Europe in metal, fluorspar, shale, and uranium mines. The National Academy of Sciences, the International Commission on Radiological Protection, the National Council on Radiation Protection and Measurements, and other national and international organizations have reviewed the data and have concluded that there is strong evidence that radon causes lung cancer in humans.

Abelson questions the assumptions used by EPA for extrapolating from "high doses of radon in mines to low doses in homes." He suggests that a threshold for cancer induction exists because humans have remediation mechanisms for α particle damage. However, research has established that even low doses of α radiation produce genetic damage that cannot always be repaired. Damage from α particles is added to a background of genetic damage from multiple sources. The net effect of this accumulated damage is an increased risk of cancer. Linear models are widely held to be adequate for extrapolating from high to low doses of high linear energy transfer radiation, including that from α particle doses from radon daughters (*1*). However, large extrapolations to the residential environment are not needed for radon risk assessment. Significant increases in lung cancer mortality have been observed in miners at a wide range of cumulative radon exposures, including low levels comparable to a lifetime residential exposure at 4 pCi/liter (*2*).

Abelson also suggests that silica dust may have been an important factor in the increased lung cancer mortality observed in the miners. The potential confounding of the radon–lung cancer relationship by the presence of silica dust in mines has been investigated by epidemiologists since the 1930s. Studies have shown that lung cancer rates correlate with cumulative radon exposure regardless of silica dust levels (*3*). The International Agency for Research on Cancer (IARC) has concluded (*4*) that, for crystalline silica and amorphous silica, respectively, the evidence of carcinogenicity in humans is limited and inadequate. On the other hand, the IARC has concluded that there is *sufficient* evidence that "radon and its decay products are carcinogenic to humans (Group 1)" (*5*).

Finally, Abelson questions the public health threat posed by residential exposure to radon on the basis of ecologic studies, stating that in some states with high radon levels, "inhabitants have less lung cancer than those in states with low levels." The limitations of ecologic studies for testing etiologic hypotheses have been well established. The average radon level for a state does not necessarily reflect the levels to which the individuals dying of lung cancer were exposed. Additionally, other important factors, such as individual smoking habits and mobility, cannot be assessed in this type of study. Because of these limitations, the Study Design Group of the International Workshop on Residential Radon Epidemiology has recommended against the further use of ecologic studies for the study of residential radon risk (*6*).

The EPA recognizes the uncertainties associated with the estimation of radon risks, as well as the uncertainties of risk assessment in general, and has supported studies to reduce uncertainty (*7*). However, given the extensive epidemiologic evidence that radon causes cancer in humans, the magnitude of the estimated risk, and the potential for elevated radon levels in homes, EPA's recommendation that American homes should be tested for radon and that elevated levels should be reduced represents prudent and responsible public health policy.

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5. *Ibid.*, vol. 43, *Man-made Mineral Fibers and Radon* (International Agency for Research on Cancer, Lyon, France, 1988).
6. U.S. Department of Energy and Commission of European Communities, *International Workshop on Residential Radon Epidemiology—Workshop Proceedings, July 1989* (CONF-8907178, National Technical Information Service, Springfield, VA, 1989), pp. 7–12.
7. National Academy of Sciences, *Comparative Dosimetry of Radon in Mines and Homes* (National Academy Press, Washington, DC, 1991).

Response: Studies bearing on the carcinogenicity of radon and its products have been affected by confounders. The data have been collected on miners, many of whom have been exposed to mineral dusts, and most of whom were smokers. Some were exposed to polycyclic hydrocarbons in diesel fumes. The combination of breathing some mineral dusts and smoking is known to be synergistic in causing lung cancer in the absence of radon. This is true of quartz (SiO_2) (*1*), amphiboles (asbestiform minerals), and a zeolite. On the Colorado Plateau, heavy exposures to mineral dusts were the rule during the 1950s. Deaths from silicosis and other nonmalignant pathology characteristic of exposure to silica have repeatedly been noted in Plateau miners (*2*).

Oge and Farland state that significant numbers of lung cancers have occurred in miners exposed to levels of radon comparable to a lifetime residential exposure at 4

pCi/liter. In a major study, it was found that the relatively few miners who died of lung cancer after low exposures to radon were cigarette smokers (*3*). Most of the nonsmoking miners of the Colorado Plateau who died of lung cancer experienced levels of radon orders of magnitude greater than 4 pCi/liter. The miners' exposure was poorly controlled and measured during the crucial early 1950s and has probably been understated.

In view of the difference in exposure levels between mines and homes and the pathologic effects of heavy exposures to mineral dusts and diesel fumes, extrapolation from mines to homes is questionable. Nevertheless, EPA has been emphasizing for the past 5 years that radon in homes is the second leading cause of lung cancer. The total annual number from all causes, including smoking, is about 140,000. Various numbers quoted by EPA of the deaths resulting from radon have ranged as high as 43,200. This was a statistical limit, but how many members of the public are versed in statistics? If radon is such a potent cause of lung cancer in the general public, the pathology should be highly obvious. In recent times, levels of radon in millions of homes have been measured. High levels of radon have not correlated with high rates of lung cancer (*4*). In the three states with the highest mean radon levels in home living areas (Colorado, North Dakota, and Iowa: 3.9, 3.5, and 3.3 pCi/liter, respectively), the death rate from lung cancer averages 41 per 100,000. In the three states with the lowest radon levels (Delaware, Louisiana, and California: 0.75, 0.96, and 0.97 pCi/liter, respectively), the rate averages 66 per 100,000.

Before 1930, lung cancer was a rare disease. At that time radon exposures were comparable to those of today. Lung cancer became important only after the advent of smoking on a large scale (*4*).

In spite of the flimsiness of the evidence to support its radon program, EPA has engaged in a campaign designed to frighten all of us, and especially mothers. This has gone on for years and has been fostered by the media. The most egregious tactic has been a 30-second spot TV film that has been repeatedly shown. I have a VCR copy of it.

In the TV spot a family is seen in front of their television set. A voice says that high radon in one's home is like having hundreds of chest x-rays a year. Flashes occur that appear to cause the entire skeleton of a child seated on his mother's lap to be revealed. It isn't only the child's chest that is exposed to x-rays; it's his entire skeleton—conveying an impression of death.

Prolonged exposure to high levels of radon in miners undoubtedly is a cause of cancer in both smokers and nonsmokers. The failure of EPA to produce rigorous data on effects of low levels of radon on nonsmokers in homes detracts from its credibility.—PHILIP H. ABELSON

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Structure of RNA Polymerase II–Associated Protein: Correction

A recent paper by M. Horikoshi *et al.* (*1*) pointing out an error in the complementary DNA sequence encoding the RNA polymerase II–associated protein, RAP30, has prompted us to write a correction of our recent report "Related RNA polymerase–binding regions in human RAP30/74 and *Escherichia coli* σ70" (23 Aug., p. 900) (*2*). Because the inferred amino acid sequence of RAP30 is changed by the correction of Horikoshi *et al.*, the COOH-terminal fragment is now predicted to be 7.5 kD rather than 5.5 kD, and it might be expected to have a lower electrophoretic mobility than the internal 7.3-kD fragment, which contains the σ homology region. The schematic representation of RAP30 [(*2*), figure 1C] should be altered to reflect this change. We have performed amino acid sequence analysis of the 7.3- and the 7.5-kD cyanogen bromide fragments, obtained from bacterially expressed RAP30, that were purified by urea SDS–gel electrophoresis and have confirmed that our earlier identification of the 7.3-kD fragment was correct. Therefore, the conclusions stated in our report are not affected by this change.

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Erratum: In the report "Cloning and expression of a cocaine-sensitive dopamine transporter complementary DNA" by S. Shimada *et al.* (25 Oct., p. 576), three amino acids were inadvertently omitted in figure 1. A tyrosine (Y) should have been at position 156 so that phenylalanine (F) and asparagine (N) were at 155 and 157, respectively; a cysteine (C) should have been at position 242 so that alanine (A) and leucine (L) were at 241 and 243, respectively; and a proline (P) should have been at position 272 so that methionine (M) and Y were at 271 and 273, respectively.