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.