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Lung Cancer Risks of Underground Miners: Cohort and Case-Control Studies

VICTOR E. ARCHER, M.D.

Rocky Mountain Center for Occupational and Environmental Health, Department of Family and Preventive Medicine, University of Utah, School of Medicine, Salt Lake City, Utah

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All underground mines have higher radon levels than are found in surface air. Ventilation is the primary method of controlling radon levels. Fourteen cohort and seven case-control studies done on underground miners are reviewed; they include many types of ore. Only five of the studies deal with more than 100 lung cancer deaths. Variations in the attributable risk are given. Some generalizations can be drawn from these studies: the longer the follow-up, the greater is the attributable risk, even though the relative risk is reasonably constant. The induction-latent period is quite variable but is shortened by high exposure rates, by cigarette smoking, and by increasing age at start of mining. The predominant histological type of lung cancer among miners changed from small-cell undifferentiated for short follow-up time to epidermoid after long follow-up times. With short follow-up time, a multiplicative interaction between smoking and radiation was indicated, but, with long follow-up time, the two factors appear to be simply additive. This difference is probably due to the shortened latent period among cigarette smokers, not to synergism.

Any hole in the ground has higher radon levels than are found in the atmosphere. This classification includes caves and all types of mines. Coal mines are subject to a high risk from explosive mixtures; therefore, their ventilation has usually been adequate to keep radon levels low. Other mines have had much less ventilation; therefore, many of these have radon problems. Radon decays into radioactive isotopes of lead, bismuth, and polonium, which are inhaled as free ions or as attachments to dust particles; it is this radon progeny which delivers most of the radiation dose to lungs. The unit of measurement is the working level (WL): any combination of radon progeny in one liter of air which results in the ultimate release of 1.3×10^5 MeV of potential alpha energy. The unit may be converted to a cumulative term (working level months, or WLM) by multiplying the WL by the number of months exposed at that WL. One WLM is equivalent to a radiation dose somewhere between 0.5 and 2 rad (cGy). "Miner" in this paper refers to all underground workers.

Excess lung cancer (LC) has been noted among the Schneeburg and Jachymov miners of central Europe for hundred of years [1]. Selected information on recent studies of mining groups is given in Tables 1 and 2. The first cohort study was of U.S. underground uranium miners [1,2]; it was formed from all uranium miners examined periodically between 1950 and 1960. This group included 3,362 white miners; they had worked in many small, short-lived mines—about 2,500. About 43,000 measurements

Abbreviations: I-L: induction-latent LC: lung cancer WL: working level WLM: working level months

TABLE 1
Cohort Radon-Mining Studies and Lung Cancer

Senior Author	Year	Nation	Ore Type	Mean Follow-up Time (years)	Lung Cancer Deaths	Attributable LC/WLM/10^6 Person-Years	Lowest Group Mean with Significant Excess (WLM)
Lundin [1]	1971	U.S.A.	Uranium	19	185	3–8	180
Archer [2]	1976						
Ševc [3]	1976	Czechoslovakia	Uranium	26	212	10–20	72
Ševc [4]	1987	Czechoslovakia	Uranium	30	484	16–62	65
	1987	Czechoslovakia	Iron	25	9	12	40
	1987	Czechoslovakia	Shale-clay	25	22	38	25
Muller [5]	1985	Canada	Uranium	18	119	2–12	65
Morrison [6]	1985	Canada	Fluorspar	30	89	6	700
Howe [7]	1986	Canada	Uranium	14	54	21	120
Radford [8]	1984	Sweden	Iron	44	50	19	81
Jorgensen [9]	1984	Sweden	Iron	31	28	13	96
Solli [10]	1985	Norway	Niobium	24	12	50	69
Wang [11]	1984	China	Tin	24+	499	7	140

TABLE 2
Radon-Mining Studies and Lung Cancer

Senior Author	Year	Nation	Ore Type	Mean Follow-up Time (years)	Lung Cancer Deaths	Attributable LC/WLM/10^6 Person-Years	Lowest Group Mean with Significant Excess (WLM)
Wagoner [12]	1963	U.S.A.	Copper	30+	47		<600
Boyd [13]	1970	U.K.	Iron		36		<200
Tirmarche [14]	1985	France	Uranium		36		<100
Pham [15]	1983	France	Iron	25	13		<150
Fox [16]	1981	U.K.	Tin		28		<400
Leira [17]	1986	Norway	Iron	33+	3	0	N.A.
			Case-Control Studies				
Damber [18]	1982	Sweden	Iron		20		<100
Edling [19]	1983	Sweden	Iron	40+	33	30–40	<160
Hewitt [20]	1976	Canada	Uranium	15	81	3.1	75
Axelson [21]	1978	Sweden	Lead-zinc	55+	21	28–35	240
Samet [22]	1984	U.S.A. Navajo	Uranium	25+	23		
Archer [23]	1985	U.S.A.	Uranium	30+	105		
Saccomanno [24]	1986	U.S.A.	Uranium	10+	489[a]		

[a]These were not deaths, but cases with sputum classification of moderate atypia or greater.

of radon progeny werre made in the mines before 1970. Not all mines or all years were included in the measurements; therefore, many estimates were necessary in order to calculate cumulative WLM exposure for each miner.

Mortality follow-up of U.S. miners has been intermittent and is continuing every few years. An innovative life-table analysis demonstrated that the LC risk rose with increasing exposure, giving an exposure-response curve that extended from about 120 WLM to over 4,000 WLM. An attributable risk of 3 to 8 LC per million person-years was found. Analysis of smoking data demonstrated an interaction of radiation and smoking which was multiplicative in nature up to 1977 [25], but which was less than multiplicative (but more than additive) when data through 1982 were used [26]. The latest report on this cohort indicates that the LC risk per WLM is now greater than in earlier reports [26].

Of all the studies reported, this study is the only one done in a prospective manner. All others were retrospective, with cohorts being obtained from old employment or other records. Application of models to these data is discussed by Dr. Lubin in an accompanying article.

Another important study is from Czechoslovakia [3,4], which supplied much of the uranium for the Russian nuclear arsenal. The Czech exposure data all came from measurement of radon rather than radon progeny and had to be converted to a WLM equivalent. It is, however, probably better than the U.S. exposure data, since it came from a small number of mines that had undergone frequent radon measurements. This study has yielded a higher risk per WLM than did the U.S study and shows excess lung cancers at levels as low as 65 WLM. In the latest report [4], the risk per WLM is higher than it was in an earlier report that included less follow-up time [3].

There have been a series of reports from Canada [5–7], the most important one being that of Muller et al. [5], which included about 16,000 Ontario miners. This large study uses better exposure estimates and has longer follow-up than earlier studies; the follow-up time is still relatively short, however. The risk value is similar to the risk found in U.S. uranium miners. The exposure data is better than that for the U.S. study and is perhaps comparable to the Czech data.

The report by Morrison et al. on fluorspar miners from Newfoundland emphasized that uranium mines are not the only type of mines with a radon problem [6]. Its exposure estimates are more tentative than for most of the others in Table 1 because some of the mines had been closed before measurements were made. The results, however, are quite similar to those from other studies.

There are two cohort and several case-control studies from Sweden [8,9,18,19,21]. Sweden has no uranium mines, but they had a peculiar situation. When silicosis was recognized as a major problem in the 1920–30 period, most mining areas solved the problem by increasing ventilation. In Sweden, the extra ventilation caused severe problems from freezing, so they developed a method for warming the air: they circulated it through broken rock and old underground workings. This practice heated the air and solved the silicosis problem, but it gave them a radon and lung cancer problem which was not recognized for many years. The best of the Swedish studies is the one by Radford and Renard [8], which is notable for its long follow-up, the low average exposure, and the high LC risk per WLM. Extensive radon progeny measurements were first made in these mines in the 1968–72 period. Earlier exposures were estimated by considering changes in ventilation over time. They estimated the error in WLM estimates as ±30 percent. This precision, however, has been challenged

by others. Their analysis by smoking indicated that the attributable LC risks for smokers and nonsmokers were very similar—suggesting a simple additive effect between the two cancer-inducing agents.

Jorgensen's report from a different Swedish iron-mining area supports the results of Radford and Renard, with the exception that most of their LC occurred among cigarette smokers [9].

The largest study of all is the one from China, where boys were sold into bondage for work in the tin mines [11]. Their exposure data were scanty and recent, but their results tend to agree with other data. This study confirms other analyses, which have found that the younger a person is when he starts mining, the longer will be the induction-latent (I-L) period; that is, the time from start of mining to LC diagnosis will be lengthened when exposure occurs early in life. This lengthened I-L period, however, does not seem to diminish the lifetime risk.

The six cohort studies in Table 2 add little to our understanding of the radon problem, except to emphasize that the problem is found in many types of mines around the world, and that the risk appears at exposure levels of 100 WLM or less.

The case-control studies of Table 2 give about the same range of lung cancer risk as the cohort studies, but the populations of most were quite small. The Swedish studies listed here used general population controls matched on age and residence. The cigarette-radon interaction was considered by Damber and Larsson to be multiplicative [18], by Edling to be additive [19], and less than additive (smoking gave protection) by Axelson and Sundell [21]. The Axelson and Sundell study used miners who started mining at the turn of the century, but LC were collected only during a recent 20-year period. This delay gave them a very long follow-up period after start of mining, and most of the LC cases were old. Their analysis not only indicated a protective effect of smoking but yielded a high LC risk per WLM. The first Canadian study by Hewitt used a National Register to locate lung cancers [20]. A random 1 percent selection from this register was used for controls and was matched on age at start of mining. This study was heavily criticized when first published, but later cohort studies have given remarkably similar results, except that exposure estimates have been revised upward somewhat.

The study of Samet et al. was limited to Navajo Indians, who smoke very little [22]. This study was important because it demonstrated a high lung cancer rate among a group of miners who smoked very little, which showed that cigarette smoking is not essential for the high LC rates found among uranium miners. This feature was first exhibited by central European miners before cigarettes were invented [1].

The case-control study by Archer [23] was designed to answer criticism of earlier analyses, which had reported a shortened I-L period among cigarette-smoking miners. It controlled for date of birth, start of mining, follow-up period, WLM, and exposure rate; it again demonstrated that the I-L period is significantly shorter among smokers than among nonsmokers [23]. In all of the studies in Tables 1 and 2 which compared smokers and nonsmokers, the smokers had shorter I-L periods.

The study of Saccomanno et al. [24] was unique in that it used changes in exfoliated bronchial cells as the end-point, rather than cancer. The end-point was moderate atypia or more severe changes. Controls were miners who never had sputum with such severe changes. The analysis indicated that both smoking and radiation exposure contributed to the sputum changes and that the combined effect of the two agents was probably additive.

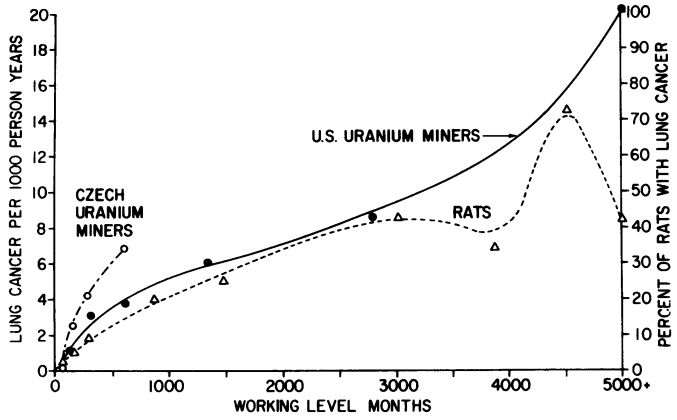

FIG. 1. Exposure-response curves for two cohort studies and for one animal study; exposures to radon daughters.

Statistically significant excesses of attributable LC were found by some of the studies in the 25–100 WLM range. Some of the studies found elevated LC rates below levels indicated in Tables 1 and 2, but numbers were too small for statistical significance. Some instances were reported of LC rates lower than expected at the lowest exposure level. This finding was attributed to the healthy worker effect, to short follow-up, or to small populations. The lifetime mean radon progeny exposure in residential houses is in the range of 10 to 20 WLM, with some homes giving exposures above 100 WLM. Extrapolation of LC data from miners to home situations is therefore reasonable, if allowance is made for differences in age and temporal factors.

The differences in LC risk per WLM indicated in Tables 1 and 2 are probably due to several factors: *(1)* errors in estimation of average worker exposures: none of the studies measured individual exposures, some had better and earlier measurements than others, and all had to estimate average mine WL levels over the years; *(2)* time of follow-up: in both the U.S. and Czech studies, risk per WLM has risen with longer follow-up, and those studies with the longest follow-up time in Tables 1 and 2 usually have yielded the highest risk per WLM; *(3)* variations in cigarette smoking among the different cohorts: in most of the studies the amount of smoking was unknown, but it is clear that smoking was much less prevalent among Swedish miners than among U.S. miners—yet the lung cancer risk per WLM found among Swedish miners has been consistently higher than among U.S. miners; *(4)* ethnic differences: there are some ethnic differences, but with the exception of the Chinese and Navajo reports, all the miners were of white Euorpean ancestry. Ethnic differences are probably not important in these comparisons.

Those reports in Tables 1 and 2 which yield the lowest LC risk per WLM tend to have the shortest follow-up time and/or tend to have overestimated exposures. Those which have the highest LC risk per WLM tend to have the longest follow-up time and/or tend to have underestimated exposures.

One animal study [31] and two cohort studies [2,3] suggest that the exposure-response curve has a supralinear component at its lower end (Fig. 1). Analysis using a Cox proportional hazards model supports supralinearity [26]. This supralinearity might be an artifact resulting from misclassification of the exposure of some miners, but this explanation cannot apply to the rat study. It may simply be an artifact of small number of tumors at the lower end of the curve; it may also be an expression of the lower dose-rate received by the men with low WLM. In these studies, exposure rate

and cumulative exposure are generally proportional to each other. We are not at all sure of the existence of supralinearity, but it could have considerable significance for the problem of radon exposure in homes.

A number of the mines used in these studies contained small amounts of fibrous minerals, arsenic, or chromium in their ores, so etiology is not entirely clear. Only the Chinese tin mines had enough arsenic to be considered a problem in itself, yet their risk per WLM was similar to that found in other studies. Some of the mines, however, contained negligible amounts of these elements. The only known environmental exposures of a toxic nature that all of these mines had in common were silica and radon. There is little evidence that silica is carcinogenic in man.

Data collected early in the epidemic of lung cancer among uranium miners has been misleading in at least two ways. For example, when enough lung cancers among uranium miners had been collected to examine, a predominance of small-cell undifferentiated, mostly oat cell cancers were found [27]. It was announced that small-cell cancer was characteristic of radiation-induced LC. For several years, uranium miners with LC did not receive workers' compensation benefits unless they had a small-cell type.

Histologic data on LC continued to be collected. The frequency of small-cell LC among uranium miners declined sharply from about 60 percent in 1960 to 20 percent in 1980. Epidermoid types had shown a corresponding increase from about 20 percent in 1960 to 70 percent in 1980. This shift was readily apparent when the data were analyzed by age or by time after start of mining [28]. Apparently radiation induced several types of cancer, and the small-cell type was simply the one that appeared first.

Another example of misleading early data has occurred with the interaction of smoking and radiation. Nearly 80 LC had been collected from U.S. miners before the first one among a nonsmoker was found. This fact was interpreted by some to mean that smoking was much more important than radiation in causing LC among miners. One scientist even declared that if uranium miners would not smoke, they would not get lung cancer. In reviewing the studies listed in Tables 1 and 2, we noted that some found a multiplicative effect (interpreted as synergism), some found a simple additive effect, and one even found a protective effect from smoking. Each investigator felt that his results were right, and the others were wrong. The problem has recently been approached from another viewpoint [29]. This approach assumes that all the findings are correct but differ because each applies to different stages in the epidemiologic development of LC among miners.

In order for all the findings to be correct, we would need to have time-response curves for radiation-induced LC something like the ones in Fig. 2 [29]. These are hypothetical curves, which assume that both smokers and nonsmokers received the same radon exposure during the early period. It then projects their resultant lung cancer distribution by increasing age. Since tobacco smoke contains powerful cancer-promoting agents [30], we can hypothesize that radiation-induced LC among smokers appears fairly early, in short latent periods, and then drops off at advanced ages as the induced cancers are exhausted. The LC among nonsmokers would continue appearing to near the end of their life spans in proportion to LC rates expected from their ages.

If an epidemiologist collects his data while the men are mostly 35–65 years of age (as was the case with the early reports on U.S. miners), then a high proportion of the LC will be among smokers, and the radon-smoking relationship will appear to be multiplicative. Data collected while the miners are from 60–70 years of age will find

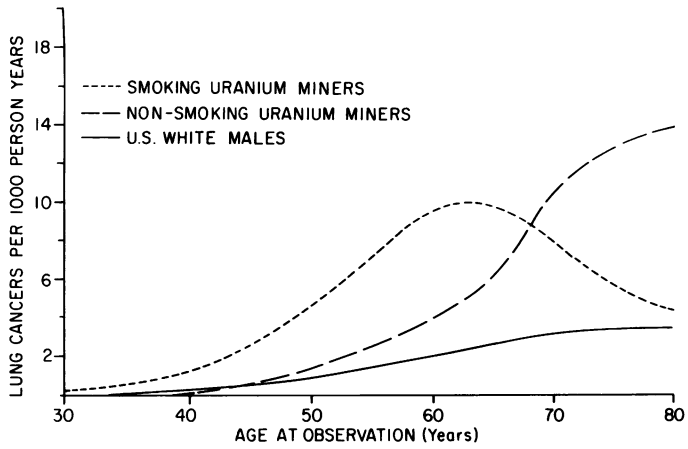

FIG. 2. Hypothetical curves for time distribution of lung cancers induced in smokers and nonsmokers by an equal radon daughter exposure between 30 and 40 years of age.

roughly equal LC rates among smokers and nonsmokers, whereas if LC are collected mainly from men 65–85 years of age, LC will appear predominantly among the nonsmokers, and one might conclude that smoking is protective. This method of data collection is essentially what Axelson did—most of his LC were among retirees [21]. If one collects data over the whole life span, then the smoking and radiation effects would appear to be little more than additive. This result is approximately what happened in several of the studies [4,8,19].

To see if the types of age-related curves hypothesized in Fig. 2 might be appearing among the U.S. cohort, a recent analysis produced the curves given in Fig. 3 [29]. These data indicate that the lung cancer rate is still rising among nonsmokers of all ages, and among the younger smoking miners, but is falling rather sharply among the oldest group of retired smoking miners. Although the data are still not firm enough to be sure, they are consistent with the types of curves postulated in Fig. 2. A similar decreasing lung cancer rate at older ages is suggested among Czech miners, but that data does not separate smokers from nonsmokers [4]. The curves in Fig. 3 suggest that the attributable LC rates among persons over 65 years of age are similar for smokers and nonsmokers, even though the rates among younger smokers are much higher than those among corresponding nonsmokers.

Earlier LC data from U.S. uranium miners support the argument for a multiplicative (synergistic) interaction between cigarettes and radiation [25]. Figure 3 and the temporal change noted by Hornung and Meinhardt [26], however, suggest that with the passage of additional time, the U.S. data may agree with the European data, which indicates an additive effect for the two agents [4,8,19].

The hypothetical curves in Fig. 2 require that the mean I-L period be shorter among smokers than among nonsmokers, and that the difference become larger as miners are followed longer. The mean of this difference was 5.3 years for U.S. uranium miners [23] and was nine years in the study with the longest follow-up after start of mining [21].

Animal studies have repeatedly demonstrated an interaction between cigarette smoke (or its components) and ionizing radiation [29]. With one exception, these studies have been interpreted as demonstrating a tumor-promoting effect by the cigarette smoke or components. Tumors in animals exposed to both agents have generally appeared earlier in time than in groups exposed to only one of the agents and

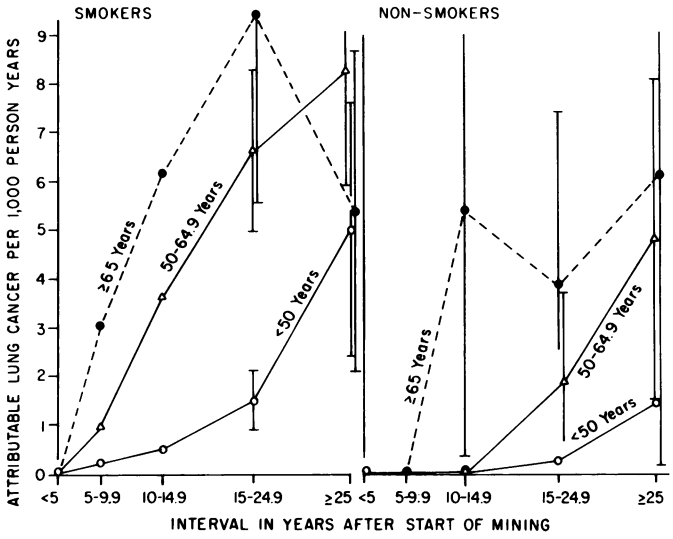

FIG. 3. Lung cancer rates among U.S. white uranium miners by age groups, by smoking, and by interval after start of mining uranium.

have been additive or somewhat more than additive [31–35]. Some were thought to be multiplicative when short-term analyses were done, but none were reported as showing a multiplicative interaction when based on life-span data. These experiments have used mice, rats, hamsters, and dogs and exposed them to either beta or alpha radiation. The one with exceptional results was a dog experiment, in which cigarette smoke reduced the number of LC produced by radon progeny [36]; this result has not been satistactorily explained.

The hypothetical curves in Fig. 2 are consistent with actual I-L data noted above, with observed changes in LC rates among smoking and nonsmoking miners, and with animal data. They explain very well why different epidemiologists have reported such divergent results with regard to the interaction of smoking and radiation among miners.

An understanding of the nature of the interaction between smoking and radon progeny is quite important for assessing the problem of radon in homes. If the miner data reflects true synergism, the problem in homes should largely disappear as people stop smoking. If the interaction is simply promotive and additive, then the problem will continue and will increase as people live longer and houses are tightened to conserve energy, unless countermeasures are employed.

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