

PART VI: RADIATION AND PARTICULATE MATTER

RESPIRATORY DISEASE MORTALITY AMONG URANIUM MINERS

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INTRODUCTION

Uranium, as found in nature, is normally at or near equilibrium with its decay products, which include the inert gas radon. Because radon diffuses from the rock into open areas of mines, most underground miners are exposed to radon concentrations in excess of those found above ground. The range of atmospheric radon concentrations in mines varies widely from 1×10^{-3} picocuries per ml (pCi/ml) of air in potash mines to 30 pCi/ml in some poorly ventilated mines.

When pure radon is breathed, it diffuses throughout the body and gives what is essentially whole-body radiation. Its retention, however, is limited, since most inhaled radon is also exhaled within its half-life of 3.8 days. For the immediate daughters of radon, however, the story is different. These radionuclides (^{218}Po , ^{214}Bi , ^{214}Pb , ^{214}Po) collectively have an average half-life of about 30 minutes. When formed in the air of a mine, they quickly become attached to solid surfaces, most notably dust particles. When these dust particles with attached radionuclides are inhaled, the radiation from them (largely alpha particles) is delivered to those sites in the nose, pharynx, and tracheo-bronchial tree where the dust particles are deposited. The radiation dose delivered to the lungs of uranium miners by these elements is about 20 times greater than that from inhaled radon. Measurements of radiation exposure and of the biologic effects of this exposure on miners have been attempted repeatedly since about the year 1880.

Previous studies of United States uranium miners have shown that their risk of respiratory cancer is related to the magnitude of exposure to radon daughters.¹⁻³ A similar excess, however, was not clearly demonstrated among either non-cigarette-smoking white uranium miners or among non-white uranium miners (mostly American Indians).¹ Some publications reported a loss of pulmonary function among uranium miners,⁴ but little attention was paid to their mortality from pulmonary insufficiency. An update of the earlier mortality studies now has been made that provides data for filling in these three deficiencies.

MATERIALS AND METHODS

An intensive follow-up was done on the 3,366 white and 780 non-white workers who had been examined by our traveling medical teams between 1950 and 1960 and who had one or more months of underground uranium employment prior to January 1, 1964. Detailed occupational and smoking histories had been obtained by personal interview at the time of examination, in many cases

repeatedly. These data were supplemented by an annual census of uranium miners from 1954 through 1969 and by main questionnaires.

The follow-up cut-off date was January 1, 1974, for Indians, and September 30, 1974, for whites. The followup was accomplished with the help of the Internal Revenue Service, credit agencies, state motor vehicle registrations, and telephone calls to relatives and trading posts (Indians), and by searching death certificate files in three states.

Death certificates were obtained for the known dead, and cause of death was interpreted according to the Revision of the International Lists of Diseases and Causes of Death in effect at the time of death by a qualified nosologist. Those individuals whose vital status was not ascertained were considered alive, thus making conservative any finding of increased cause-specific mortality among the study group. In addition, those individuals who had been reported dead but for whom no death certificates were obtained were considered as deceased on September 30, 1974, cause of death undetermined.

Cumulative radon-daughter exposure values were calculated for each miner from the date of his first mining to each sequential month of observation through September, 1969. These values were expressed as Working Level Months, that is, the cumulative product of length of underground exposure in working months (170 hours) and the concentration of radon daughters in "Working Levels" (WL) specific for mine and calendar year. One Working Level is equal to 1.3×10^5 MeV of potential alpha energy per liter of air. It was impractical to carry these calculations beyond 1969. Furthermore, little additional exposure was received by most of the workers after this date. Radiation levels in uranium mines dropped sharply after 1967, and relatively few of the study-group members continued mining uranium after 1967. With a dropout rate of 10–50%/year after 1960, it was determined in 1965 that only 12.3% of the study-group members were still mining uranium. The dropout has continued to date, but we have not determined the number still mining in 1974. It must be small.

A modified life-table technique was used to obtain person-months at risk of dying between August, 1960 and January 1, 1974 for Indians and between July, 1950 and September 30, 1974 for whites. The person-months were obtained for each calendar year for each five-year age group, for each time interval after the start of underground uranium mining, and by estimated cumulative radon-daughter exposure with estimated WLM from both uranium mines and other metal mines for whites, but just uranium mines for Indians, since few of them had ever worked in nonuranium mines.

Comparisons with the male non-white population of Arizona and New Mexico were made for Indians, and the male white population of the United States was used for white miners. Excesses and deficits were identified by noting the observed number of deaths among miners in relation to the expected number. This expected number was calculated by applying male rates specific for age, race, calendar year, and cause of death to the appropriate number of person-years at risk. No further analysis was done with the Indian mortality, since there were few respiratory cancers.

An exposure-response analysis by cigaret-smoking groups was done for both respiratory cancer and for all respiratory disease deaths other than tuberculosis and cancer among white miners. These diseases were largely pulmonary insufficiency and were recorded as pneumoconiosis, pulmonary fibrosis, emphysema, and others, but there were also a few pneumonias, asthmas, and so on.

The chest films made between 1950 and 1960 and the readings of them made by a panel of radiologists were available. The 98 persons found to have pneumoconiosis when examined were matched against the men who had died of respiratory cancer or "other respiratory disease," or who had some type of chronic obstructive pulmonary disease mentioned on their death certificates as "other significant condition," in an attempt to learn what contribution x-ray pneumoconiosis made to the later respiratory disease deaths.

RESULTS

Follow-up was 100% among the Indians, with death certificates obtained on all deceased members. Only 60 white (1.8%) were lost to follow-up, and death certificates were obtained on all of the known dead. The seven unknown causes of death among Indians and the nine among whites had such notes as "natural causes" and "dead on arrival," with no further notations on the death certificates.

TABLE 1 shows that 107 deaths occurred among Indians during the follow-up periods. Among Indians, the overall expected and observed rates were about the same, but there were marked differences in distribution. Respiratory cancer was the only death cause which was significantly higher than expected. Three additional deaths were probably due to lung cancer, but were not studied sufficiently to establish the diagnosis. There was a trend toward excess deaths

TABLE 1
OBSERVED AND EXPECTED DEATHS BY CAUSE AMONG AMERICAN INDIAN
UNDERGROUND URANIUM MINERS: JANUARY 1960–JANUARY 1, 1974

Cause of Death	List Number	Observed	Expected
Tuberculosis, all forms	001–019	8	3.7
All malignant neoplasms	140–205	17 †	9.9
Digestive system *	150–159	4	3.8
Respiratory	160–164	11 ‡	2.6
All other malignant disease		2	3.5
Vascular disease of central nervous system	330–334	1 †	6.2
All heart diseases	401–443	2 ‡	19.2
All respiratory disease (nonmalignant)	470–527	7	5.4
Cirrhosis of liver	581	1 †	6.8
All accidental deaths	800–962	39	31.7
Motor vehicle	810–825, 830–835	21	16.2
All other accidental deaths		18	15.5
Suicide and homicide	963, 964, 970–979, 980–985	5	9.7
All other known causes		20 †	31.3
Unknown causes		7	0.0
TOTAL		107	123.9

* Indented causes of death are not added into totals.

† Significant at <5%.

‡ Significant at <1%.

TABLE 2
CIGARET-SMOKING HABITS OF THE AMERICAN INDIAN MINERS

Packs per day	Smoking at Time of Examination	Amount Smoked by Former Smokers	Unknown	Never Smoked
0.0	—	—		487
0.1	117	18		
0.2	50	3		
0.3	23	2		
0.4	3	3		
0.5	37	5		
0.6	1			
0.7	2			
0.8	1			
0.9	1			
1.0	20			
1.5	1			
2.0		1		
Unknown			4	
TOTALS	256	32	4	487

for tuberculosis, other nonmalignant respiratory disease, and accidental deaths. There were significantly fewer deaths from heart diseases, from vascular disease of the central nervous system, and from liver cirrhosis than had been expected. Even if all the unknown causes of death were sudden death from heart attacks or strokes, there would still be a deficit of these deaths.

The distribution of cigaret smoking among the Indians is given in TABLE 2, indicating that as a group they used little tobacco.

The 745 deaths among white miners (TABLE 3) are substantially greater than the expected number. Substantial excess deaths occur in three categories: respiratory cancer, nonmalignant respiratory disease, and accidents. Accidents while at work (most of the "other accidents") account for most of the excess deaths from this cause. There was a tendency toward elevated deaths from "all other malignant disease," and from motor vehicle accidents. The excess of both auto and other accidental deaths is greatest among inexperienced workers (less than 10 years on the job) and falls essentially to expected levels at 25 years after start of mining. Respiratory disease (both malignant and nonmalignant) is not elevated among workers with less than five years experience, begins to rise during the five- to ten-year period and rises rapidly with longer time intervals after the start of mining. There is a tendency to a deficit of deaths due to liver cirrhosis and to heart and other vascular diseases.

The exposure-response curves for both bronchogenic cancer and for pulmonary insufficiency show a clear relationship to cumulative exposure as expressed in Working Level Months (WLM) (TABLES 4 and 5, FIGURES 1 & 2). The separation into cigaret-smoking groups clearly indicates that cigaret smoking has played a part in both cancer and pulmonary insufficiency deaths, but that both occur in the absence of cigaret smoke. The linearity of several of the curves, especially for nonsmokers, is remarkable.

TABLE 3
OBSERVED AND EXPECTED DEATHS BY CAUSE AND BY TIME AFTER START OF URANIUM MINING
AMONG WHITE UNDERGROUND MINERS: JULY 1950-SEPTEMBER 1974

Cause of Death List	Years after Start of Underground Uranium Mining											
	0-4.9		5-9.9		10-24.9		25 and over		Totals *			
	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.		
Tuberculosis 001-019	0	0.6	1	0.8	4	2.0	0	0.5	5	3.9		
All malignant 140-205 neoplasms	6	6.9	22	15.1	142 †	56.2	36 †	15.8	206 †	93.9		
Brain and nervous system 193 †	0	0.4	0	0.7	3	2.0	0	0.4	3	3.5		
Digestive system 150-159	2	2.0	5	4.3	13	15.8	5	4.8	25	26.9		
Respiratory system 160-164	2	1.9	12 †	4.6	103 †	18.3	27 †	4.9	144 †	29.8		
Lymphatic & hematopoietic system												
200-205	1	1.0	2	1.9	4	5.9	1	1.4	8	10.2		
All other malignant diseases	1	1.6	3	3.6	19	14.1	3	4.2	26	23.5		
Vascular disease of central nervous system 300-334	0	2.0	2	4.5	15	18.7	2	7.0	19 §	32.3		
All heart disease 401-443	0 §	15.6	27	34.1	102	129.4	28	39.0	166 §	218.2		
All respiratory disease (nonmalignant) 470-527	1	1.5	7	3.5	54 †	15.0	18 †	4.8	80 †	24.9		
Cirrhosis of liver 581	0	1.2	0	2.6	7	8.7	0	1.8	7	14.2		
All accidental deaths 800-962	42 †	7.2	41 †	10.1	51 †	22.5	6	3.6	140 †	43.3		
Motor Veh. 810-825, 830-835	12	4.0	9	5.3	15	10.8	2	1.5	38 †	21.5		
All other accidental deaths	30 †	3.2	32 †	4.8	36 †	11.7	4	2.1	102 †	21.8		
Suicide and homicide 963, 964, 970, 979, 980-985	7 §	2.3	2	4.0	13	9.8	0	1.5	22	17.7		
All other known causes	16 †	5.7	23 †	10.8	43	37.1	9	10.6	91	64.2		
Unknown causes	1	0	0	0	7	0	1	0	9	0		
Totals *	73	43.1	125	85.4	438 §	299.3	100	84.7	745 §	512.5		

* Marginal totals may not check exactly because of rounding.

† Difference is significant at 1% level.

‡ Indented causes of death are not used in obtaining totals.

§ Difference is significant at 5% level.

TABLE 4
RESPIRATORY CANCER MORTALITY AMONG WHITE MINERS BY WLM AND BY CIGARET SMOKING,
FIVE OR MORE YEARS AFTER START OF MINING: JULY 1950-SEPTEMBER 1974

Smoking Group	Working-Level Months									
	1-359			360-1799			1800 and above			
	Cancers	Person-Years	Cases per 1,000	Cancers	Person-Years	Cases per 1,000	Cancers	Person-Years	Cases per 1,000	
Nonsmokers	1	4917.73	0.20	3	3487.94	0.86	2	1436.54	1.39	
1-19 Cigaretts/day	5	3169.42	1.58	3	2617.77	1.14	6	716.78	8.3	
20 Cigaretts/day	9	6907.38	1.30	29	8236.88	3.52	30	3175.58	9.4	
Over 20 Cigaretts/day	8	2944.12	2.72	17	3634.63	4.68	15	1126.53	13.3	
Former Smokers	3	1009.6	2.97	6	1842.18	3.26	9	888.03	10.13	

TABLE 5
 MORTALITY FROM RESPIRATORY DISEASES OTHER THAN CANCER AND TUBERCULOSIS
 AMONG WHITE MINERS BY WLM AND BY CIGARET SMOKING FIVE OR MORE YEARS
 AFTER START OF MINING: JULY 1950-SEPTEMBER 1974

Smoking Group	Working-Level Months										
	1-359			360-1799			1800 and above				
	Resp. Deaths	Years Person	Cases per 1,000	Resp. Deaths	Person Years	Cases per 1,000	Deaths Resp.	Person Years	Deaths Resp.	Person Years	Cases per 1,000
Nonsmokers	3	4917.73	0.61	4	3487.94	1.15	2	1436.54	2	1436.54	1.39
1-19 Cigaretts/day	1	3169.42	0.315	8	2617.77	3.05	5	716.78	5	716.78	6.9
20 Cigaretts/day	11	6907.38	1.59	14	8236.88	1.70	10	3175.58	10	3175.58	3.1
Over 20 Cigaretts/day	1	2944.12	0.34	6	3634.63	1.65	4	1126.53	4	1126.53	3.54
Former Smokers	3	1009.63	2.97	6	1842.18	3.26	2	888.03	2	888.03	2.25

In the matching of those 98 individuals whose films had been interpreted as showing pneumoconiosis between 1950 and 1960 (TABLE 6), an apparent difference in frequency was found. However, since those men who had x-ray pneumoconiosis were generally older and had more mining experience than those without it, it is not appropriate to compare the two groups directly. The important parameter is the ratio of the three subsequent conditions. These ratios are nearly the same for both groups. In fact, the ratio of lung cancer to

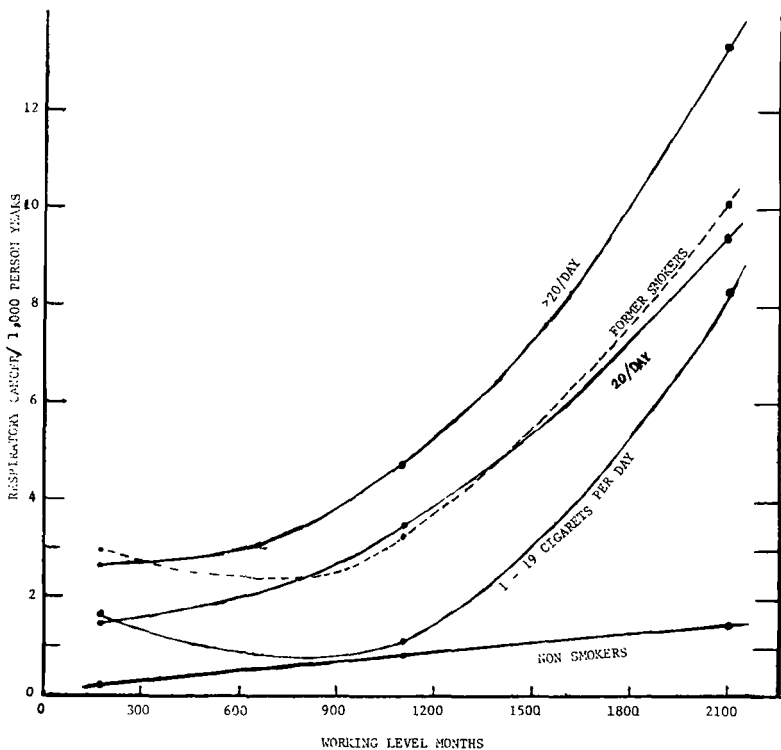


FIGURE 1. Respiratory cancer mortality as related to radon daughters, by cigarette-smoking groups.

those who died of pulmonary insufficiency tends to be slightly higher among those who did not have x-ray pneumoconiosis.

“Other” accidental deaths among whites (mostly mine accidents, TABLE 6) show a drop from 9.4 times expected for the less than five-year group, with a steady drop to 2 × expected for the 25-year-and-over group.

Items of general interest on non-cigarette smokers are listed in TABLE 7. It should be noted that of the 17 men listed, only one ever smoked as much as 26 cigarettes per day, and that of 15 whose histologic tumor type is known, all but three were small-cell undifferentiated.

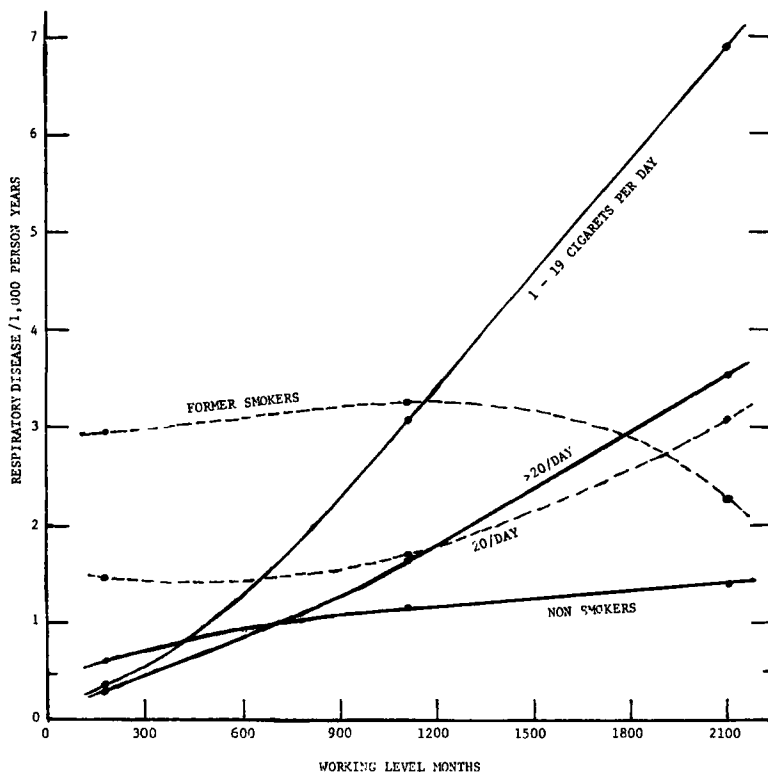


FIGURE 2. Respiratory disease mortality (other than cancer and TB) as related to radon daughters, by cigaret-smoking groups.

TABLE 6
OTHER ACCIDENTAL DEATHS AMONG WHITE UNDERGROUND URANIUM MINERS
BY LENGTH OF MINING EXPERIENCE

Years After Start of Underground Mining	Observed	Expected	Ratio of Obs./Exp.
0-4.9	30	3.199	9.4
5-9.9	32	4.812	6.6
10-14.9	17	5.466	3.1
15-24.9	19	6.246	3.0
25 or more	4	2.078	1.9

DISCUSSION

The excess respiratory cancer among Indians not only shows that Indians are not immune to radiation-induced cancer, but also indicates that radio-induced bronchogenic cancer occurs when cigaret usage in the exposed group is minimal or absent (TABLES 7, 8). Of the 14 respiratory cancers among Indians (including three not definitely diagnosed), four stated, at time of examination, that they

TABLE 7
DATA ON INDIANS AND WHITE NON-CIGARET SMOKERS WHOSE DEATH
WAS CERTIFIED AS DUE TO RESPIRATORY CANCER

Case No.	Age at Death	Usual Cigarette Usage	Years of Smoking	Induction-Latent Period	Histologic Type of Tumor WHO Classification *	WLM of Radon-Daughter Exposure	Race
7554	42	0	0	17	2B	867	Indian
3036	60	3-6/day	50?	24	1A	2760	Indian
0469	55	2-3/week	30?	14	2A	1325	Indian
7583	40	3-4/day	~ 3	20	2A	2039	Indian
3076	44	2-3/day	15	19	2A	782	Indian
7555	35	3-6/day	~ 15	17	2B	3234	Indian
3046	43	0	0	21	not available	3202	Indian
7551	39	0	0	21	not available	3052	Indian
10538	45	10-20/day	29	15	2B	2038	Indian
7577	56	2-3/week	20	15	2B	1415	Indian
3095	60	0	0	23	2B	1336	Indian
5806 †	73	0	0	23	3A	16467	white
0222	65	0	28 ‡	13	1A	2573	white
10360	56	0	0	15	2A	465	white
7607	67	0	0	14	2B	783	white
10777	42	0	0	18	2A	520	white
7050	62	0	0	17	2A	2908	white

* In the WHO Classification, 1A=well differentiated epidermoid; 2A=oat cell; 2B=small round or polygonal cell; and 3A=well-differentiated adenocarcinoma.

† Clinical data not clear as to origin; may have originated in colon.

‡ He smoked 6-10 cigars daily for 28 years.

never smoked, two smoked two to three cigarets/week, six smoked two to six cigarets/day, one smoked one to ten/day, and one smoked ten to twenty/day. The time from start of mining to cancer (induction-latent period) for these fourteen men range from 11 to 24 years, with a mean of 17.1 years. The mean induction-latent period for the four nonsmokers was 20.5 years. These mean times may be contrasted with a mean of 13.7 years induction-latent period among 15 white uranium miners with lung cancer who smoked 20 cigarets or

more per day and died before 1963, when an excess was first clearly demonstrated among them. This shortened induction-latent period for heavy smokers has been previously demonstrated.⁶

The expected lung cancer rates calculated on the basis of non-white rates for Arizona and New Mexico is as close as we could get to appropriate rates, since there are substantial Indian populations in both states. There are, however, substantial numbers of blacks and orientals in both states who are included in the non-white mortality rates. The ratio of Indians to other non-whites is greater for these two states than for Utah or Colorado, where some of the Indian miners worked. Most of the Indian miners were Navajos from Arizona and New Mexico, although a few were born in Utah and Colorado. Male Indians (nonminers) have a total cancer and respiratory cancer rate that is

TABLE 8
RELATIONSHIP OF EARLY X-RAY PNEUMOCONIOSIS AMONG WHITE MINERS TO SUBSEQUENT BRONCHOGENIC CANCER AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE

	No.	Men who Later Developed Broncho-genic Cancer	Men who Died of Pulmo-nary Insuffi-ciency	Men who Had Chronic Obstructive Pulmonary Disease Noted on Death Certif-icate as "Other Sig-nificant Con-dition"	Ratio of Cancer:In-sufficiency: D.C. Note
X-ray pneumoconiosis					
1950-1960					
Number	98	17	15	10	1:0.88:0.59
Percentage	—	17.3	15.3	10.2	
No x-ray pneumoconiosis					
1950-1960					
Number	3268	126	120	65	1:0.95:0.52
Percentage	—	3.8	3.7	2.0	

approximately half that of United States rates for white or non-white.^{7, 8} For this reason, the "expected" respiratory cancer rates in TABLE 1 are undoubtedly overestimated. It should also be noted that most Indian uranium mine respiratory cancers are of the small-cell undifferentiated variety (8 out of 9 with good histology were small-cell undifferentiated), similar to the predominant type among white uranium miners.⁹ The radiation exposure among the 14 Indians ranged from 782 to 3234 WLM with a mean of 2004 WLM, which indicates that most of the cancers have come from the more heavily exposed groups, consistent with the findings among white miners.

The deficit of vascular diseases of the heart, kidneys, and central nervous system among Indians is thought to be due to a combination of medical selection of miners through preemployment examinations, to minimal tobacco use, to

their diet, which is generally low in saturated fats, to a way of life involving considerable exercise, and possibly to a racial factor. Low death rates from cirrhosis of the liver among most employed groups probably reflects lower use of alcohol by working groups than by the general non-white population. Miners who abuse alcohol do not usually hold jobs very long.

The mortality pattern of white miners (TABLE 3, FIGURE 1) has changed somewhat from earlier reports. The excess of "other respiratory" and the deficit of cardiovascular disease deaths is a definite shift. The two may be related; i.e., men dying of pulmonary insufficiency might have died a few months or years later of heart disease. The death certificate coding appears to underestimate the magnitude of the shift, inasmuch as many of those heart disease deaths carry such notes as "right heart failure," "cardiorespiratory failure," emphysema, pulmonary fibrosis, and so on, often as an "other significant" entry.

The linear curves for nonsmokers in both FIGURES 1 and 2 argue for a direct dose-response in both respiratory cancer and "other respiratory disease" deaths. Some of the curves from cigaret groups are nearly linear also, but others are markedly curvilinear. The heaviest smokers have the highest incidence of respiratory cancer, as expected, but the differences between them and a pack a day or former smokers is not large.

The curves for light smokers are anomalous. At high radiation levels their cancer risk is nearly as great as among heavier smokers, but is much less at lower radiation levels. With respect to deaths from "other respiratory disease," the light smokers appear to have all the worst of it, and exhibit a more linear exposure-response curve than heavier smokers. This pattern suggests that heavy smoking may give protection against the development of parenchymal injury—possibly by the production of extra mucus, which shields cells from the minimally penetrating alpha radiation of radon daughters—but that light smoking does not give this protection. This apparent protection does not extend to cancer induction, however, which probably arises in the walls of larger bronchi.

No specific condition can be pinpointed as the cause of the excess "other respiratory disease" deaths. It appears to be pulmonary insufficiency resulting from diffuse radiation injury to lung parenchyma, leading to later fibrosis and emphysema. Cigaret smoke clearly contributes to it. Siliceous dust and diesel exhaust in mines would be expected to contribute an additional increment of damage.

The ratio of "other accidental deaths" (mostly due to mine accidents, TABLE 7) to expected, drops with time after start of mining. This undoubtedly represents a learning response, since beginners are at highest risk. This drop with time is in strong contrast to respiratory cancer and "other respiratory" deaths that rise with time. This latter pattern is typical of chronic occupational diseases: low initially, but constantly rising with increasing time after start of exposure.

The ratios of x-ray pneumoconiosis:lung cancer:pulmonary insufficiency deaths:chronic obstructive lung disease noted as "other significant condition" were surprising (TABLE 8). We had supposed that persons with x-ray pneumoconiosis would be much more likely to die of pulmonary insufficiency than other men. However, the ratios suggest that the earlier x-ray pneumoconiosis had little or no relevance to subsequent respiratory disease deaths, and that both types of disease (cancer and insufficiency) were primarily related to radiation exposure or cigaret smoking rather than to siliceous dust. This is consistent

with the data in TABLE 4 and FIGURE 2. An alternative hypothesis, not ruled out by present data, is that early x-ray pneumoconiosis increases lung susceptibility to both cancer and pulmonary insufficiency equally.

Data in FIGURE 1 and TABLE 7 clearly indicate that underground uranium miners who do not smoke cigarettes or who smoke just a little, suffer an increased risk of lung cancer (most bronchogenic cancers among nonsmoking, nonminers are adenocarcinomas), although their risk is substantially less than that of heavy smokers.

In an earlier report,¹ with use of a computer model, it was calculated that for 30 years' exposure at 4 WLM/year, a group of 20-year-olds, by age 80 would have about a 45% increase of respiratory cancer over expected numbers. Since the bulk of expected cancers is due to cigarette smoking, this means that the respiratory cancer risk from 30 years' exposure at 4 WLM/year is about half that of smoking cigarettes at the rate our miners have used them in the past. However, because there was some overestimation of exposures in that analysis, and because the median ten-year latent period now appears to be a little short, that earlier estimate of risk from 4 WLM/year is probably a little low.

SUMMARY

A mortality analysis of a group of white and Indian uranium miners was done by a life-table method. A significant excess of respiratory cancer among both whites and Indians was found. Nonmalignant respiratory disease deaths among the whites are approaching cancer in importance as a cause of death, probably as a result of diffuse parenchymal radiation damage. Exposure-response curves for nonsmokers are linear for both respiratory cancer and "other respiratory disease." Cigarette smoking elevates and distorts that curve. Light cigarette smokers appear to be most vulnerable to lung parenchymal damage. The predominant histologic cancer among nonsmokers is small-cell undifferentiated, just as it is among cigarette smokers.

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