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REPORT NO. 8

REVISED

guidance for the control of radiation hazards in uranium mining

SEPTEMBER 1967

Staff Report of the

FEDERAL RADIATION COUNCIL

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SECTION I

INTRODUCTION

1.1 This report supersedes the preliminary FRC staff Report No. 8 which was released on May 7, 1967, for discussion in the Joint Committee on Atomic Energy hearings on radiation exposure of uranium miners. It contains background material used in the development of guidance for Federal agencies in regulatory programs and in programs of cooperation with States concerning radiation protection in the mining of uranium ore, and seeks to provide guidance for long-term radiation protection in uranium mining. Periodic review will be necessary to incorporate new information and new surveillance or control techniques as they are developed. The report also includes recommendations for additional research and recordkeeping needed to provide a firmer basis for the evaluation of radiation risks in this industry.

1.2 The use of uranium, as a source of nuclear energy for the electric power industry, is developing during a period when Government procurement for military purposes is declining. These two needs are complementary with respect to ore production, and operate to maintain the uranium mining industry as an activity of substantial importance to the national economy. The uranium mining industry is located in 10 Western States, 5 of which produce over 90 percent of the total domestic uranium ore. The value of recoverable uranium in ore produced in four of these States as a whole is about half the combined values of copper, lead, and zinc ores produced from the same States.

1.3 The natural radioactive decay of uranium leads to the formation of various radioactive nuclides in ore bodies; one of which, radon, is gaseous. Radon gas formed by the radioactive decay of radium 226 escapes from exposed rock surfaces into the air of uranium mines, where it continues to decay, generating a series of other radioactive products commonly termed radon daughters. Radon gas is also present in aboveground air in concentrations that may vary with location, time of day, and weather conditions. Some of the radon daughters contained in the air breathed by miners are known to be deposited, retained, and to irradiate tissues in the miner's respiratory system. Studies by the U.S. Public Health Service in cooperation with the Atomic Energy Commission and State agencies disclose that underground uranium miners are subject to lung cancer to a degree substantially greater than the general population, or of that in miners in other kinds of underground mines. The excess incidence apparently is related to the uranium miner's occupational environment, and is believed to be induced by the radioactive decay of radon daughters in the respiratory system.

1.4 Emphasis is focused on the radiation hazards associated with underground uranium mining, since control is more difficult than that which can be achieved in open pit mining or in subsequent milling operations. Open pit mining, being an aboveground operation, presents no special problems in radiation protection. Primary attention is given

in this report to the evaluation of radiation hazards resulting from the inhalation of radon and radon daughters in the confines of underground mines and methods by which these hazards can be controlled. In making this review, it is recognized that the benefit derived by the application of progressively more stringent control requirements must be evaluated in light of the resulting reduction in radiation risk and the total impact of the more stringent controls.

1.5 In addition to the recognized authority of the States to establish health and safety standards for mining operations conducted within their respective jurisdictions, responsibilities have been designated to certain Federal agencies. They include the Department of the Interior in the administration of the Federal Metal and Nonmetallic Mine Safety Act; the Department of Labor in the administration of the Walsh-Healey Act; the Department of Health, Education, and Welfare in providing technical advice in the matter of health standards and control of health hazards; and the Atomic Energy Commission in the regulation of source material (*i.e.*, uranium and thorium), after removal from the place of deposit in nature. States, Federal agencies, and the mining industry all have a direct interest in the development of uniform standards applicable to the practical problems of uranium mining, including a standard for radiation protection.

1.6 In the preparation of this staff report technical experts from various Federal and State agencies, industry, and individual nongovernment scientists assisted in developing information concerning mining practices, economic aspects of uranium mining, epidemiological evidence for associating adverse health effects with radon daughter concentrations in mine atmospheres, and considerations involving basic radiobiological mechanisms and the absorbed dose to tissue resulting from breathing mine atmospheres. Coordination of information between the staff of the Federal Radiation Council, the National Council on Radiation Protection and Measurements, the USA Standards Institute (formerly called the American Standards Association), and the Atomic Industrial Forum was achieved by including individuals associated with these organizations in the FRC task groups. In addition, the staff has had the benefit of consulting with representatives of organized labor, members of the International Commission on Radiological Protection, and with individuals in other countries where uranium is mined.

1.7 The Federal Radiation Council Working Group, which consists of senior technical personnel from the agencies comprising the Council, has supplied advice and information from their respective agencies for inclusion in the report. Personnel from the U.S. Bureau of Mines, Department of Interior, also have actively participated in the preparation of this report.

Scope of Uranium Mining Industry

Production

1.8 The domestic uranium mines in 1966 produced approximately 50 percent as much uranium ore as was produced in 1961, the year when the industry was at its peak. This curtailed rate is due to a stretch-out of Government procurement contracts designed to balance procurement with requirements and to tide the industry over an interim period between diminishing Government needs and the developing needs for the electric power industry. Procurement under the remaining Government contracts during the period

July 1967 through 1970 is estimated at 28,300 tons of U_3O_8. Procurement for electric power plants in this period will be of the order of 18,000 tons of U_3O_8.

1.9 The U.S. Atomic Energy Commission estimates that "by 1980 the United States will have between 120- and 170-million kilowatts of electricity generated by nuclear power, with a midrange of this projection about 150-million kilowatts—the best single estimate." [1] This estimate takes into account the growing demand for electric power, engineering and economic factors, and the current rapid acceptance of nuclear power. An increase of this magnitude in nuclear power plant facilities will probably require on the order of 250,000 tons of U_3O_8 over the 15-year period from 1966 through 1980 inclusive,[2] taking into account the pertinent factors of probable reactor types, sizes, and characteristics, as well as enrichment factors and fuel economy. About 100-million tons of mined ore would be needed to provide this quantity of uranium, assuming no major change in the U_3O_8 content of ore processed (currently about 0.23 percent U_3O_8). Although this is in excess of presently known low cost domestic ore reserves (economic at a price of $10 per pound of U_3O_8), it is considered reasonable to expect that additional ore will be discovered to keep pace with demand. The 1966 mining rate was 4.2-million tons. To meet the estimated total 15-year requirement, this rate is expected to rise to 16- to 18-million tons annually by 1980. The accuracy of these future estimates, however, is subject to the possible effect of foreign trade in uranium.

1.10 A substantial fraction of the known uranium deposits can be mined by the open pit method. Currently, about one-third of the domestic ore production is derived from open pit mines, some of which have been converted from shallow underground operations. Current technological developments tend to increase the advantages of the open pit method and therefore to extend the depth of economically recoverable deposits. On the other hand, deposits discovered in the future are expected to be at progressively increasing depths, so that the proportion of ore mined from open pits during the next 15 years may be expected to decline somewhat. Thus, underground ore production may, by 1980, rise to somewhat over 12-million tons per year, or several times the current underground mining rate.

1.11 Some perspective on the relative significance of uranium mining to the States involved is provided by comparison with copper, lead, and zinc mining. The latest comparative statistics available appear in the 1965 issue of the U.S. Bureau of Mines Minerals Yearbook. The total amount of copper, lead, and zinc ore produced from mines located in the principal uranium-producing States (New Mexico, Wyoming, Colorado, and Utah) was about 43-million tons, with a recoverable metal value of about $295 million. In the same year these States produced 4 million tons of uranium ore containing about $150 million worth of uranium.

Employment in U.S. Uranium Mines

1.12 The first recorded production of uranium-vanadium carnotite-type ore in the United States was in 1898 from the Uravan mining district, Montrose County, Colo.[3] Limited production continued until about 1935 when demand for vanadium for use in alloy steels increased the market for carnotite ores. Despite this production uptrend, there was no sustained large-scale employment until after the Atomic Energy Act of 1946. The first price schedule of the Atomic Energy Commission that became effective April 9, 1948,

launched a new major mining industry. Thereafter employment in uranium mines increased rapidly until 1961, when it declined as a consequence of curtailed Government purchases. Open pit mining of uranium did not become significant until 1955. The number of uranium mines providing ore during the years 1954 through 1966 and the corresponding employment figures are shown in tables 1 and 2, respectively.

TABLE 1.—*Estimates of the number of mines producing uranium ore during the calendar year as reported by the industry to the U.S. Bureau of Mines (1954–64) and AEC (1965–66)*

Year	Underground mines	Open pit mines	Year	Underground mines	Open pit mines
1954	450	50	1961	497	122
1955	600	75	1962	545	139
1956	700	100	1963	573	162
1957	850	125	1964	471	106
1958	850	200	1965	562	74
1959	801	165	1966	533	88
1960	703	166			

TABLE 2.—*Number of men employed in uranium mines*

Year	Underground mines[a]	Open pit mines	Year	Underground mines[a]	Open pit mines
1954	916	53	1961	4,182	1,047
1955	1,376	293	1962	4,174	1,074
1956	1,770	584	1963	3,510	886
1957	2,430	574	1964	3,249	726
1958	2,796	1,175	1965	2,900	700
1959	3,996	1,259	1966	2,545	359
1960	4,908	1,499			

[a]Excludes aboveground employees who may occasionally go underground.

1.13 Some further perspective is provided by considering the number of underground miners related to the size of mine. The categories selected for this purpose are: (1) mines employing 15 or fewer men (2) those employing from 16 to 50 and (3) those with more than 50. The data assembled in table 1-52 of U.S. Bureau of Mines report, "Health and Safety Study of Metal and Nonmetal Mines" [4] for 1963; are as follows:

Number of men per mine	Percent of mines	Percent of men
15 or less	60	16
16 to 50	27	32
More than 50	13	52

1.14 During 1966 a total of 621 mines produced ore, but the number in operation at any one time probably did not average more than 400. Underground mines account for the variability; the number of producing open pit mines was fairly constant at about 80. The number of underground miners involved was probably less variable than the number of operating mines because men customarily move from one mine to another when mine operations are intermittent. Many uranium miners have worked previously in nonuranium mines; conversely, uranium miners often quit uranium mines to work in nonuranium mines. The turnover rate in the work force is therefore difficult to evaluate. However, in a group of 1,888 uranium miners identified in a 1954 survey made by the U.S. Public Health Service, only 26 percent were found still working in uranium mines 6 years later. It is also estimated that less than 1 percent work longer than 15 years in uranium mines.

Health and Safety Hazards Other Than Radiation in Uranium Mining

Injury Experience

1.15 The hazards of traumatic injury associated with mining are commonly recognized. Although there are wide variations in the frequency and severity of accidents within the industry, miners as a group are exposed to serious risks. Experience in the metal mining industry shows that injury rates have been reduced to less than half over the last 30 years but, nevertheless, current injury rates remain about four times the average of all manufacturing industries.[4]

1.16 Uranium mining became prominent at a time after the injury rate in metal mines had been substantially reduced, but the rapid growth of uranium mining, and the fact that much of it was concentrated in areas remote from other metal mines, necessitated the employment of some inexperienced miners. The injury rate in uranium mines during the late 1950's was as high as that experienced in other metal mines 15 to 20 years earlier. Since 1960, however, experience in uranium mines has improved significantly.

Physical Hazards

1.17 U.S. Bureau of Mines records show that most disabling work injuries occurring in underground uranium mines arise from machinery, haulage, handling of materials, falls of persons, and falls of ground. Fatal injuries have resulted primarily from explosives, haulage, and falls of ground. This has also been the experience in other underground metal mines. The fatal accident rate in underground uranium mines during 1965 averaged about 1.93 per million man hours.[5]

Environmental Heath Hazards in Underground Mines

1.18 The quality of the atmosphere in underground mines is of primary importance to the health and safety of underground workers. The mine atmosphere is subject to contamination by harmful dusts and gases. Such contamination might result from drilling (dust), blasting (gases and dust), materials handling (dust), use of diesel engines (exhaust gases), emission of strata gases, welding and cutting, and from mine fires. In uranium mines and some other types of mines the radiation environment must also be considered (see sec. II). In general, appropriate measures used to control the hazards owing to common dust and gases will serve also to reduce concentrations of radioactive materials in the mine air.

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1.19 Most uranium ore is mined from sandstone that contains free silica, the causative agent of silicosis.[6] Surveys in uranium mines having highly siliceous ores indicate that effective dust control measures are necessary to restrict the concentration of airborne dust to less than recommended limits. Wet drilling and wetting of blasted material before and during loading and transportation are normally supplemented by ventilation to the extent required for effective control of dusts and gases. Toxic gases may constitute a hazard to the health of mine workers if ventilation is not adequate, or if men return to work areas before the gases have been removed or sufficiently diluted by ventilation. Ventilating fans can provide positive means for controlling the volume and flow of air underground. With a properly arranged duct system fresh air can be delivered directly to work areas or, alternatively, contaminated air can be withdrawn from such areas and delivered above ground.

1.20 The use of diesel engines in underground mines necessitates ventilation to dilute and remove toxic exhaust gases and to replace oxygen consumed by combustion in engines.[7] Required volumes of ventilating air for individual units are stipulated on approval plates of diesel-powered equipment approved by the U.S. Bureau of Mines for use in underground mines.

1.21 Guides for limiting concentrations of gases, mineral dusts, and airborne contaminants– other than radioactive substances that are likely to be encountered in underground atmospheres– are published annually by the American Conference of Governmental Industrial Hygienists. These threshold limits are widely used by engineers, inspectors, and regulatory agencies as guides for controlling underground atmospheric environments. These values represent conditions under which it is believed that nearly all workers may be repeatedly exposed without adverse effect. The concept of threshold limits is not used in radiation protection.

1.22 Control of temperature is an important factor in the operation of ventilation systems in underground uranium mines, particularly in cold climates. Incoming fresh air is often heated to prevent freezing of pipes and to prevent icing problems in shafts and haulageways.

REFERENCES

[1] Seaborg, G. T. Fast Breeder Power Reactors—A World Outlook. Annual Meeting of the Canadian Nuclear Association: Montreal, Quebec, Canada, May 31, 1967.

[2] Nininger, R. D. World Production and Reserves of Uranium. Twelfth Annual Minerals Symposium: American Institute of Mining, Metallurgical and Petroleum Engineers: Moab, Utah, June 23, 1967.

[3] Coffin, R. C. Radium, Uranium, and Vanadium Deposits of Southwestern Colorado. Colo. Geol. Survey Bull. 16, 1921.

[4] Report to Congress by the Secretary of the Interior. Health and Safety of Metal and Nonmetal Mines (Submitted in response to PL 87–300, 75 Stat. 649), 1963.

[5] Injury Experience, Employment, and Worktime in the Mineral Industries 1964–65. U.S. Bureau of Mines Mineral Industry Survey, August 1966.

[6] USPHS Pub. 1076. Silicosis in the Metal Mining Industry—Reevaluation 1958–61 (USPHS and Bureau Mines), 1963.

[7] Holtz, John C. Safety with Mobile Diesel-Powered Equipment Underground. U.S. Bureau of Mines Report of Investigations, No. 5616, 1960.

SECTION II

THE RADIATION ENVIRONMENT ASSOCIATED WITH URANIUM MINING

2.1 The atmosphere in underground uranium mines will normally contain a number of contaminants capable of producing various deleterious effects in the respiratory system. These entities include:

1. Airborne dust particles: Silica, alkaline earth and other metal carbonates, silicates, vanadates and aluminates with smaller amounts of iron, molybdenum, and uranium minerals including the radioactive uranium decay products, notably radium 226. Thorium and its radioactive decay products might also be present in minor amounts. Atoms of the radon decay products rapidly become attached to these particles (see par. 2.5).

2. The radioactive gas radon (and thoron if thorium is present).

3. Free ions: Single atoms of the radioactive elements resulting from the decay of radon, *e.g.*, polonium 218 (RaA), the first decay product of radon 222. Other free ions may be formed by the radioactive decay of a free polonium 218 atom, or may be ejected by recoil of a decay product from the surface of a larger particle and other mine surfaces.

4. Nuclei: Aggregations of a few molecules (*e.g.*, water molecules) around a polonium 218 atom or other decay product, or solid particles so small that diffusion is the dominant transport mechanism.

2.2 The naturally occurring radionuclide uranium 238 is the parent of the radioactive decay chain in which radon 222 is found. Table 3 presents the principal components of the uranium series. The parallel branches in the chain from polonium 218 to astatine 218 and from bismuth 214 to thallium 210 have been omitted; only the energies of the alpha particles of interest are shown. Natural thorium sometimes occurs as a constituent of uranium ore, but in domestic ores it is generally less than 1 percent by weight of the uranium content.

2.3 External gamma radiation intensities in domestic uranium mines seldom exceed 2.5 mR per hour,[1] and the average intensities are only a fraction of this. It is accordingly unlikely that uranium miners will be exposed to external whole-body radiation doses as large as the Radiation Protection Guide (RPG) recommended by the FRC for occupational radiation exposure (5 rems per year). However, in mining occasional high grade ore pockets (5 percent or greater U_3O_8) external radiation levels may necessitate limitation of personnel exposure. Beta radiation intensities near broken ores may be higher than gamma intensities by a factor of 10,[2] but are of relatively minor importance as an external radiation hazard under mining conditions.

TABLE 3.—*The uranium series*

Isotope	Symbol	Historical name	Half-life	Radiation	Alpha energy (MeV)
Uranium 238	^{238}U	Uranium I	4.5×10^9 yrs	α	
Thorium 234	^{234}Th	Uranium X_1	24.1 days	β, γ	
Protactinium 234	^{234}Pa	Uranium X_2	1.18 mins	β, γ	
Uranium 234	^{234}U	Uranium II	2.50×10^5 yrs	α, γ	
Thorium 230	^{230}Th	Ionium	7.6×10^4 yrs	α	
Radium 226	^{226}Ra	Radium	1620 yrs	α, γ	
Radon 222	^{222}Rn	Radon	3.82 days	α	5.49
Polonium 218	^{218}Po	Radium A	3.05 mins	α	6.00
Lead 214	^{214}Pb	Radium B	26.8 mins	β, γ	
Bismuth 214	^{214}Bi	Radium C	19.7 mins	β, γ	
Polonium 214	^{214}Po	Radium C′	164×10^{-6} sec	α	7.69
Lead 210	^{210}Pb	Radium D	22.0 yrs	β, γ	
Bismuth 210	^{210}Bi	Radium E	5.0 days	β	
Polonium 210	^{210}Po	Radium F	138.4 days	α	5.30
Lead 206	^{206}Pb	Radium G	Stable		

Radon and Radon Daughters

2.4 Radon 222 results from the radioactive decay of radium 226. Being an inert gas it diffuses readily through the interstices of rock to the rock face and from there into the air of the mine spaces. It has been observed that the rate of radon diffusion into the mine air varies inversely with changes in the barometric pressure. As noted in table 3, the half-lives of the first four successive daughters of radon are short. Under static conditions (quiet air) radioactive equilibrium will develop in about three hours. However, an equilibrium state is seldom found in an actively worked uranium mine area where fresh air is being continually brought into the mine. The amount of fresh air that is brought into a mine affects the concentration of the radon daughter products more than it affects the concentration of radon. The radon daughter concentration in the air is reduced by dilution and by adherence to dust particles and by preferential deposition on mine walls, whereas additional radon is diffusing into the air from the rock surfaces and from mine water.

2.5 Laboratory experiments have demonstrated that the unattached daughter products of radon 222 exhibit a high rate of diffusion in air;[3] that they quickly become attached to moisture or dust particles suspended in the air (the mean lifetime existence as free unattached ions is of the order of 10 to 50 seconds),[3] and that the human respiratory system retains a substantial portion of radon daughters attached to moisture or dust particles and virtually all of the unattached portion.[4]

2.6 The principal dose to lungs of uranium miners is generally attributed to the alpha particles emitted by the decay of the radon daughter products. In most United States uranium mines the concentration of radon daughters is obtained by using a specific field method of measurement, and the result is compared to the "Working Level" (WL), which is defined as any combination of radon daughters in 1 liter of air that will result in the ultimate emission of 1.3×10^5 MeV of potential alpha energy.[5] The numerical value of the WL is derived from the alpha energy released by the total decay of the short-lived radon daughter products at radioactive equilibrium with 100 picocuries (pCi) radon 222 per liter of air. The 100 pCi of polonium 218 give 1.3×10^4 MeV from the total decay of the polonium 218 and the same number of terminal polonium 214 atoms. The 100 pCi of lead 214 give 6.6×10^4 MeV from the decay of the resultant polonium 214. The 100 pCi of bismuth 214 give 4.8×10^4 MeV from the resultant polonium 214. The resultant total is 1.27×10^5 MeV which is rounded to 1.3×10^5 MeV.

2.7 A significant advantage in the concept of the WL is its practical application to field measurements of the radon daughter concentrations in mine air. The method of measuring the concentration of decay products in terms of total alpha particle emission is widely used for control and regulatory purposes. Exposure of an individual to radon daughters in air can be estimated from the length of time the individual breathes an atmosphere containing a stated burden of radon daughters. The Public Health Service publications usually express exposures as "Working Level Months" (WLM), although other time periods are sometimes used. Inhalation of air with a concentration of 1 WL of radon daughters for 170 working hours results in an exposure of 1 WLM.

2.8 Historically, radon daughter measurements have been made in the United States uranium mines since about 1950, and the records are preserved in large part by the U.S. Public Health Service Occupational Health Field Station in Salt Lake City, Utah. Since about 1960 similar records also have been kept by State regulatory agencies. The records maintained by the USPHS Occupational Health Field Station comprise such items as mine identity and location, identification of personnel working underground at the time of the survey, location of sampled mine areas, and concentrations of radon daughters in mine air expressed in terms of the WL. From time to time this agency has made summaries of these records available for public purposes. The summaries generally display WL data averaged on a calendar year basis prior to 1962. For subsequent years the reported averages use data for the third calendar quarter. The data are further detailed according to the State in which the mine is located; the States of main concern being New Mexico, Wyoming, Colorado, Utah, and Arizona. Uranium mines located in these States produced more than 90 percent of the total domestic uranium ore in 1966.

2.9 Table 4 presents a summary derived from records of radon daughter measurements in underground uranium mines prior to 1960. The table shows the number of mines measured and the percentage of mines with radon daughter concentrations falling in various ranges of WL values. These percentages are estimated to be about the same as the percentage of the work force whose annual average exposures fall within the WL ranges shown in the table, and are considered to reflect the status of the whole industry during that period of time.

TABLE 4.—*Estimated distribution of mines by Working Level ranges from 1956 through 1959*

Year	Number mines measured	<1.0 WL %	1.0–2.9 WL %	3.0–10.0 WL %	>10.0 WL %	Total %
1956	108	19	25	33	23	100
1957	158	20	26	28	26	100
1958	53	28	21	36	15	100
1959	237	18	26	28	28	100

2.10 This breakdown suggests that about one-fourth of the work force was probably exposed to atmospheres leading to annual exposures larger than 10 WL, and about one-fifth was exposed at average levels lower than 1 WL. It is also noteworthy that the number of mines surveyed between 1956 and 1959 was but a small fraction of the total uranium mines. This low coverage was due to the common event that many small mines were not in operation at the time of the survey. Many such mines were located in remote areas and operated only a few weeks or months in each year because of such factors as available ore, operating funds, labor supply, weather, and so forth.

2.11 In December 1960 a Governors' conference on health hazards in uranium mines [6] was held in Denver, Colo. This was an outgrowth of interagency studies on the occupational health problems of uranium miners carried out by the Public Health Service, the Atomic Energy Commission, the U.S. Bureau of Mines, and the Department of Labor. The objectives of the conference were to present to the Governors of States engaged in uranium mining, data on the prevailing radon daughter levels, such as those indicated in table 4; to present information on experience in controlling radiation hazards; and to assist in developing cooperative programs to reduce radiation hazards in uranium mines. As a result of this conference, many of the States placed more emphasis on their mine inspection programs.

2.12 As a measure of the prevailing levels of radon daughters in the mines, table 5 indicates the results of samples taken in the third calendar quarter of 1965 and 1966. These data indicate considerable success in reducing concentrations of radon daughters. The mine operating companies are cooperating with State regulatory agencies to improve control of radon daughter concentrations in working areas of the mines. The possibility of further reduction in the mines reporting average WL values between 1 and 10 involves considerations discussed in section IV.

2.13 The significance of these recorded data is limited by several considerations: (1) individual WL measurements usually represent a 10-minute sample at a selected location; (2) the number of samples taken per survey is restricted by the number of survey personnel that can be accommodated in an operating mine and by the man hours available to collect and analyze the individual samples; and (3) the frequency of surveys in individual mines varied widely; a single survey per year was common practice in some states, while in others surveys were even less frequent.

2.14 The U.S. Bureau of Mines carried out a study in 1962 on a modification of the usual practice in selecting locations to be sampled in a mine. The report on this project [7] indicates that an estimate of the time-weighted assessment of miner occupancy in spot-

TABLE 5.—*Summary of radon daughter concentrations by Working Level ranges during the third quarter of 1965 and 1966*

1965

State	Number mines	<1.0	Percent of mines in each WL range			
			1.0–2.9	3.0–4.9	5.0–10.0	>10.0
Arizona	16	50	19	12	19	
Colorado	124	39	42	12	6	1
New Mexico	26	15	38	42	5	
Utah	47	49	43	4	4	
Wyoming	16	44	56	.....	.....	
Total	229	39	41	13	6	1

1966

State	Number mines	<1.0	Percent of mines in each WL range			
			1.0–2.9	3.0–4.9	5.0–10.0	>10.0
Arizona	14	57	29	14		
Colorado	148	45	42	7	5	1
New Mexico	23	17	47	30	6	
Utah	33	55	39	.....	6	
Wyoming	13	38	54	8	.....	
Total	231	44	42	9	4	1

sampled areas permits an approximate evaluation of the exposure of individuals for the day of sampling. The report indicates that as few as four area samples may be sufficient to evaluate the exposure of an individual with a probable accuracy of ±25 percent. The examples cited in the Bureau report illustrate the method: (1) a miner working in various areas having concentrations ranging from 3.7 to 8.0 WL had an estimated weighted exposure of 6.1 WL, (2) another miner working in concentrations ranging from 0.3 to 6.4 WL had a weighted exposure of 1.0 WL, and (3) a third one working in concentrations of 0.2 to 1.4 WL had a weighted exposure of 0.5 WL. The results of this study suggest that the arithmetic average of concentrations found in the mine air does not give a reliable estimate of exposure. Similarly, the maximum concentration found in any representative mine sampling bears no direct relation to the exposure of individual miners.

2.15 Time-weighted assessment for evaluating exposures in uranium mines has not been generally adopted for regulatory purposes. Rather, pertinent State regulations and the recommendations of the USA Standards Institute stipulate a maximum concentration that, when exceeded, is used as a basis for closing the mine area concerned. Two of the five States (Colorado and New Mexico), for which data are reported in tables 4 and 5, maintain

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records both of concentrations found at each inspection and of the estimated time-weighted average exposure of individual workers who work in different areas in the mines.

2.16 Radon and its daughter products have also been measured in other than uranium mines. One study [8] showed that the air in certain nonuranium mines contained radon daughters in concentrations of about 0.1 to 0.2 WL. Jacoe [9] reported radon daughter concentrations in 24 nonuranium metal mines in Colorado ranging up to 2.0 WL, and a similar range in five clay mines. Rabson, *et al.*,[10] reported on radon daughter concentrations in the gold mines of South Africa. In five of these mines, which also contain uranium, radon daughter concentrations averaged between 0.1 and 0.9 WL with maximum concentrations up to 4 WL. The existence of relatively high concentrations of radon in water and air in Canadian fluorspar mines has been reported.[11] The authors found individual samples in unused spaces higher than 10 WL, and estimated that average mine air concentrations ranged from 2.5 to 10 WL.

REFERENCES

[1] Report to Congress by Secretary of the Interior. Health and Safety of Metal and Nonmetal Mines (submitted in response to PL 87-300, 75 Stat. 649), 1963.

[2] Harris, W. B., *et al.* Environmental Hazards Associated with Milling Uranium Ore. AMA Archives Ind. Health, Vol. 20, pp. 365–382, Nov. 1959.

[3] Chamberlain, A. C., and Dyson, E. C. The Dose to Trachea and Bronchi From Decay Products. British Journal of Radiology, Vol. 29, p. 317, June 1956.

[4] Shapiro, J. An Evaluation of the Pulmonary Radiation Dosage From Radon and Its Daughter Products. University of Rochester Atomic Energy Project, UR-298, April 1954.

[5] USPHS Pub. 494. Control of Radon and Daughters in Uranium Mines and Calculations on Biologic Effects, 1957.

[6] USPHS Pub. 843. Governors' Conference on Health Hazards in Uranium Mines—A Summary Report, 1961.

[7] U.S. Bureau Mines Report RI 6106. Estimating Daily Exposures of Underground Uranium Miners to Airborne Radon-Radon Daughter Products, 1962.

[8] Wagoner, J. K., *et al.* Unusual Cancer Mortality Among Group Underground Metal Miners. N. Eng. J. Med., Vol. 269, No. 6, pp. 284–289, Aug. 1963.

[9] Jacoe, P. W. Occurrence of Radon in Nonuranium Mines in Colo. AMA Archives Industrial Hygiene and Occup. Med., Vol. 8, p. 118, 1953.

[10] Rabson, S. R., *et al.* Experience in Measurement of Radon and Radon Daughter Concentration S. African Uranium Mines. Proc. IAEA Symposium Rad. Health and Safety Mining and Milling Nuclear Materials, Vol. II, pp. 335-347, 1964.

[11] deVilliers, A. J., and Windish, J. P. Lung Cancer in Fluorspar Mining Community. Brit. J. Ind. Med., Vol. 21, p. 94, 1964.

SECTION III

BIOLOGICAL EFFECTS ASSOCIATED WITH EXPOSURE TO RADON AND RADON DAUGHTERS WITH SPECIAL REFERENCE TO LUNG CANCER

3.1 When the mining of uranium-bearing ores began in the United States in 1898 [1] it was known that radioactivity was associated with the ore, but the potential health hazards from this agent were not suspected until about 1921 when Uhlig [2] suggested that the high numbers of lung cancers found among Schneeberg miners might be due to ionizing radiation. From a knowledge of radiobiology and of exposure conditions in uranium mine environments, one might expect the manifestation of radiation injury in the respiratory tract to be of three basic types: tumors, atrophy of functional tissue, and increased susceptibility to other disease agents. Possible effects from external radiation and effects related to the deposition of dust particles containing radionuclides on the skin and in the eyes cannot be excluded. However, these radiation doses are so minor in comparison to the doses to the respiratory tract that they are not treated in this report.

Animal Experiments

3.2 Animal experiments have demonstrated that doses of ionizing radiation delivered to the lungs may reduce pulmonary dust clearance,[3] produce emphysema,[4] cause loss of pulmonary function,[5,6] and cause pulmonary neoplasia, fibrosis, or changes in bronchial epithelium.[7-25] These experiments have explored many parameters: (1) species—rats, mice, rabbits, dogs; (2) radiation type—α, β, γ, x-rays; (3) internal and external radiation; (4) relationships between response and dose rate or total dose; and (5) tumor types. Such experiments are useful to the extent that they lead to sufficient insight into the mechanisms of radiation-induced carcinogenesis to aid in the interpretation of human experience. However, most of the pulmonary radiation dose received by uranium miners is from alpha particles. Since these particles have a very short range in tissue and are densely ionizing (high linear energy transfer), the different spatial distribution of energy absorption in tissue limits the utility of dose-effect relationships derived from animal experiments using beta, gamma, or x-rays.

3.3 In general, it has not been possible to produce pulmonary carcinomas in animals in a systematic way from controlled exposure to radon or radon daughters, although several attempts have been made.[26-28] Some animal experiments using radon alone, in combination with varying amounts of its decay products, or in combination with ore dusts, produced metaplasia of the bronchial epithelium and a few pulmonary tumors.[13,29-32] The small number of tumors and inadequate controls in some of the experiments precludes drawing definite conclusions from them. The administration of plutonium 239 (also an alpha emitter), in relatively large doses, has induced pulmonary malignancy in addition to severe lung damage in dogs.[12,33,34]

Mortality and Disease Patterns among Uranium Miners and Others

3.4 It has been known for many years that underground uranium miners are subject to elevated mortality rates from accidents and from lung disease. Accidental death rates among uranium miners decreased over a period of years to a low of 1 per million man hours in 1964, comparable to those of other underground miners.[35] However data presented in paragraph 1.17 indicate an accidental death rate of 1.93 per million man hours in 1965. Elevated lung cancer rates have been reported among fluorspar miners,[36] iron miners,[37 38] United States base metal miners,[39] and the gold miners of Gwanda.[40] Other authors report that no increase in lung cancer incidence was observed among South African gold miners,[41 42] nor among British coal miners.[43] The pneumoconioses (including silicosis with accompanying emphysema and cor pulmonale) found among most groups of miners may have predisposed them to pulmonary infections such as pneumonia and tuberculosis. The incidence of disability from silicosis and related chest diseases appears to have been markedly reduced by the use of modern industrial hygiene techniques, the most important of which is ventilation.[44]

3.5 Several methods have been used to express epidemiological findings in published papers. The two given below will be the principal ones used in this report:

1. "Incidence" is the number of cases of a disease appearing in a stated population per unit of time. Vital statistics are normally reported in terms of annual incidence. Since lung cancer has a high case fatality rate, the incidence of mortality is practically the same as the incidence of the disease.

2. "Mortality ratio" relates the number of observed deaths to the number that would be expected in the same population if the mortality rates derived from the vital statistics records in large populations were applicable. Calculation of the "expected" value must take into account such variables as age, sex, race, and years at risk. Of all the malignant diseases, lung cancer is the most common cause of death among males in the United States. The incidence of lung cancer in the general population increases rapidly after age 40 and reaches a peak around age 60.[45]

U.S. Uranium Miners

3.6 In 1950 the USPHS, in cooperation with other Federal and State agencies, initiated a program to evaluate the health problems inherent in the uranium mining industry. Miners were enrolled in the study if they volunteered for at least one physical examination and provided social and occupational data in sufficient detail to allow followup of their health status. Small numbers of men were examined in 1950, 1951, and 1953. During 1954, and later, attempts were made to examine as many men as could be located and would cooperate. It has been estimated that in 1957 and 1960, 90 percent of the men working in the industry in the areas visited were examined.[46]

3.7 Estimates were made of each man's exposure to radon daughters, expressed in WLM (see para. 2.7), on the basis of his occupational history and measurements of radon daughter levels in mine air. Where such measurements were not available, the probable value was estimated from measurements made in mines of similar location, depth, ore type and grade, and ventilation arrangements. The occupational history, including identifi-

cation of particular mines and when the individual worked in each, depends on individual recall rather than payroll records.

3.8 For various reasons the PHS study group does not represent the entire mining population or a random sample thereof. This complicates the evaluation of the number of lung cancers that would be expected in the group from causes unrelated to exposure to radon daughters in the mine atmosphere, since a bias resulting from the voluntary method of selection is possible. This possibility was examined by the PHS in a previous analysis, and it was concluded that this factor, although present, did not affect the general conclusions of that analysis.[46]

3.9 The total study group consists of approximately 5,000 underground miners, uranium mill workers, and other types of aboveground workers in the industry (both white and nonwhite) who have had at least one physical examination under the program. After examining the composition of the group enrolled in the PHS study from different viewpoints, the analysis made for the Federal Radiation Council focused on a subgroup of 1,981 white miners who started underground uranium mining before July 1, 1955.

3.10 The number of lung cancers observed in the subgroup selected for analysis was compared with the number that might be expected based on examination of vital statistics records of the male population of the States in which the miners worked (see par. 3.5). For purposes of the analysis, person-years were divided into categories according to increasing exposure to radon daughters expressed as cumulative WLM (see table 6).

3.11 As an example of the procedure, consider a subject who was born in July 1900, and started underground uranium mining in July 1950 in a mine where the atmosphere was estimated to contain 10 WL of radon daughters in 1950 and each year thereafter. He mined full time until December 1957 and has not mined since. In July 1954 this miner was first examined by a PHS team and thereby entered the study group. For August and each succeeding month in 1954 he was assigned one person-month at risk in the age group 50–54, WLM category C (360–839 WLM) and the category for less than 5 years after he started underground uranium mining. This individual was in comparable categories during the first 6 months of 1955. However, in July 1955 he was removed from the age group 50–54 and changed to age group 55–59 years. Also, this individual was removed from the category of less than 5 years since he started mining to the category of 5–9 years since he started mining. The 12 months at risk in 1956 and the first 6 months of 1957 did not alter the category designations for this particular individual. In July 1957 the calculated WLM value reached 840 and thereafter the person-months were assigned to category D (840–1,799 WLM) where he remained until the cutoff date for the analysis (June 1965). If the individual had died during this period, the month of death would have been used to determine the last person-month at risk.

3.12 After this accounting procedure had been completed for each member of the study group, the person-months were converted into person-years at risk, classified by calendar year, age group, WLM category, and time after the individual started underground uranium mining. The expected number of lung cancer deaths was then calculated for the person-years in each of these categories. For example, in 1958 there were 16.59 person-years in age group 45–49 and WLM category D (840–1,799) with 5–9 years after

the individuals started underground uranium mining. The annual lung cancer rate for white males in this age group in Colorado, Utah, New Mexico, and Arizona combined was 2.827 per 10,000 in that year. The term "lung cancer" is used in this report to designate cancer of the lung or of other tissues of the respiratory tract. The expected number of lung cancer deaths in this particular group is therefore $16.59 \times \frac{2.827}{10,000}$ or 0.0047. Expected numbers obtained in this way for all age groups and calendar years in each exposure category were then added to obtain the expectations shown in the analysis (see table 6).

3.13 A basic requirement for a comparison of the number of lung cancers observed in the uranium miner group with the rates in the general population is that the same criteria be used for including a lung cancer death in the study group that were used in deriving the basis for calculating the expected deaths, *i.e.*, lung cancer was listed on the death certificate as the underlying cause of death. The analysis, shown in table 6, is the same as that appearing in table 8 of the preliminary draft issued May 1967, with three additional columns – (1) the number of person-years at risk, (2) the expected annual lung cancer mortality per 10,000 miners, and (3) the calculated annual mortality per 10,000 miners. The 95 percent confidence limits for the observed mortality rates are also shown. This information, expressed in terms of expected and observed rates per 10,000 miners, is also shown graphically in figure 1.

3.14 The analysis indicates the presence of a clear association between exposure to radon daughters in mine air expressed as cumulative WLM and the number of lung cancer deaths in the study group. The 24 deaths observed versus 1.82 expected in exposure categories D, E, and F, may be compared to 10 deaths observed versus 3.31 expected in exposure categories A, B, and C. As can be seen from figure 1, all of the lung cancer rates for categories D, E, and F are significantly above the expected rate. Even though the observed incidence for categories A, B, and C is above that expected, only category B is significantly high and the lack of a progressively increasing incidence fails to support a causal role for radon daughter exposures at these levels. However, the absence of a steadily increasing incidence could easily be the result of chance alone, so that the data do not suggest or exclude the existence of a threshold. Since the quantitative relationship shown in table 6, and figure 1 may be, in part, a consequence of the way exposure categories were selected, alternative breakdowns of 1,000 WLM categories in the upper range and of 250 WLM under 1,000 WLM were considered. These alternative cumulative WLM exposure category designations for the 1,981 miners in table 6 were estimated from a random stratified sample of 299 white underground uranium miners without lung cancer and from the 19 cases of lung cancer. Examination of the ratios of cases to estimated number of miners by exposure categories at the end of 1963 suggests that the first clearly demonstrable excess of lung cancer with progressively increasing risks at each higher WLM level may be in a category somewhat above 1,000 WLM. This is hardly surprising in view of the sampling variation which might commonly occur with the small number of cases in most categories. In any event the data are not sufficient to indicate an association between exposure to radon daughters and the subsequent development of lung cancer when the cumulative exposures are less than about 1,000 WLM.

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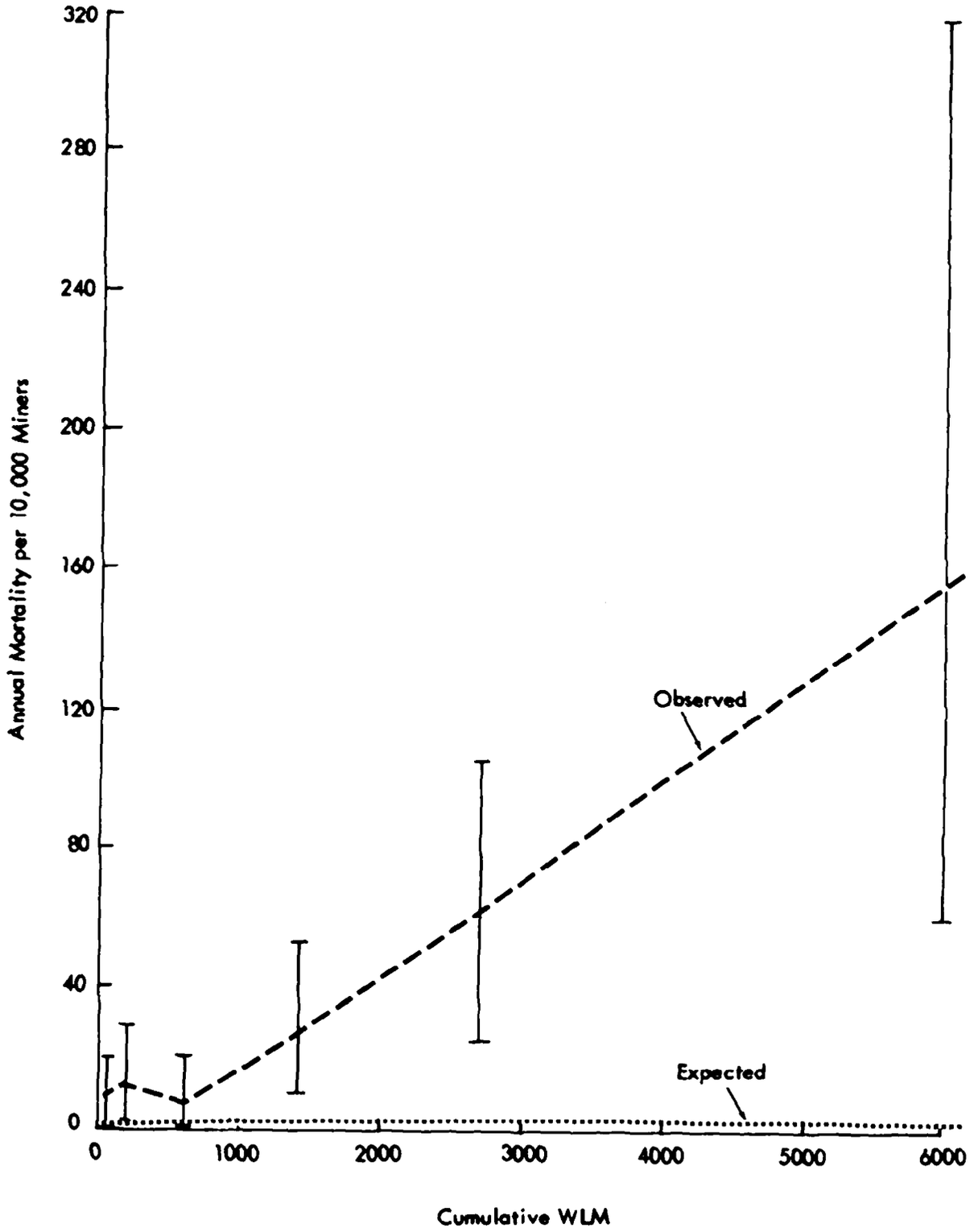
TABLE 6.—*Lung cancer mortality between July 1955 and June 1965 inclusive—white miners who began underground uranium mining before July 1955*

Exposure category	Estimated cumulative WLM	No. of miners[a]	No. of person-years at risk	Years between beginning of underground uranium mining and death from lung cancer								Calculated annual mortality per 10,000 miners			
				<5		5–9		≥10		Total		Expected	Observed (95% confidence limits)		
				Exp. no.	Obs. no.	Exp. no.	Obs. no.	Exp. no.	Obs. no.	Exp. no.	Obs. no.[b]				
A	<120	383	3,788	0.20	1	0.48	0	0.37	1	1.05	2	2.8	0.6–	5.3–	19.1
B	120–359	421	3,914	.11	0	.43	0	.50	5	1.04	5	2.7	4.1–	12.8–	29.8
C	360–839	496	4,036	.07	0	.36	0	.79	3	1.22	3	3.0	1.5–	7.4–	21.7
D	840–1,799	400	3,148	.02	0	.22	2	.77	6	1.01	8	3.2	11.0–	25.4–	50.1
E	1,800–3,719	218	1,623	.00	0	.08	0	.58	9	.66	9	4.1	25.4–	55.5–	105.2
F	≥3,720	63	455	.00	0	.02	3	.13	4	.15	7	3.3	61.8–	153.8–	316.9
Total		1,981	16,964	.40	1	1.59	5	3.14	28	5.13	34	[c]3.0	13.9–	20.0–	[c]28.0

[a] By cumulative WLM in underground uranium mines through 1963 (par. 3.32).
[b] Par. 3.16.
[c] Average rates.

Figure 1. Observed and Expected Annual Lung Cancer Mortality per 10,000 Miners and 95-percent-Confidence Limits in Relation to Exposure [a]



*See par. 3.13 and table 6.

3.15 Lung cancer is not an immediate effect of exposure to radon daughters. The largest number of lung cancers observed in all exposure categories appeared 10 or more years after the individuals first started mining, even though several of the miners worked underground only a few years in the uranium industry. For this reason, another subgroup of 1,434 miners who started mining after July 1, 1955 have not been included in the present analysis. The estimated exposures of 114 miners in this subgroup at the end of 1963 were 840 WLM or more. No lung cancers have been reported from this higher exposure group versus an expected 0.09, although 3 have been reported in the lower exposure group of 1,320 miners versus an expected number of 1.7.

3.16 The 34 observed deaths from lung cancer shown in table 6 are those that meet the requirements for group comparisons mentioned in paragraph 3.13 (*i.e.*, the underlying cause of death was recorded as lung cancer on the death certificate). Fifteen additional deaths from lung cancer make a total of 49[a] observed in the subgroup of 1,981 miners who began mining before July 1955. These were not included in table 6 for one of two reasons: (1) the "cause of death" was not listed as respiratory cancer on the death certificate, or (2) the date of death was after June 1965, the cutoff date for the mortality analysis. Five of these additional 15 cases occurred in the group of 63 miners whose recorded exposures were greater than 3,720 WLM making a total of 12 in this small group. The remaining 10 were about evenly distributed among the other exposure groups. As related to the years since the beginning of underground uranium mining, but without regard to the exposure level, 3 appeared among miners with 5 to 9 years elapsed time after the start of mining, and 12 after 10 or more years had elapsed. Further evidence of a time factor is indicated by the death of 26 individuals with lung cancer by the end of 1963, and of 23 thereafter. The 49 cases may be considered as more indicative of the real incidence of lung cancer in the study group, although there is no generally accepted way to determine the expected value. Detailed information on the 49 cases will be found in the transcript of the hearings conducted by the Joint Committee on Atomic Energy between May 9 and August 10, 1967.

Cell Type of Uranium Miners' Lung Cancer

3.17 Although the malignant nature of the growths found at autopsy in miners was recognized as early as 1879, it was not until 1926 that they were established as carcinoma.[47] As pointed out by Gates and Warren,[9] the reason that they were initially thought to be a form of lymphosarcoma is probably that most of the cancers seen were of the small cell undifferentiated variety which bears some resemblance to lymphosarcoma. Saccomanno *et al.*,[48] found that 57 percent of the lung cancers among United States uranium miners were of the small cell undifferentiated variety. The majority of these were considered by the authors to be of the "oat cell" type. The authors also showed that the proportion of small cell undifferentiated cancers among uranium miners increased with the estimated exposure to radon daughters. Small cell undifferentiated bronchial carcinoma rarely exceeds 20 percent of lung cancers found among nonminers.[48 49] Although small cell undifferentiated carcinoma or "oat cell" carcinoma of the lung is described as the morphological type representative of the lung cancers identified in uranium miners, it may be well to call

[a] For the purposes of this discussion, the 34 deaths shown in table 6 will be referred to as "lung cancer deaths" and the 15 additional deaths will be referred to as "cases."

attention to the fact that bronchogenic (lung) cancers may vary appreciably in cell type in different parts of the tumor and the diagnosis of a specific cell type related to a specific causal agent is subject to some caution.

Base Metal Miners

3.18 An elevated incidence of lung cancer has been reported among miners of a sulfide ore that contained iron, copper, zinc, lead, manganese, arsenic, antimony, calcium, fluorine, and silver.[39] The uranium-thorium content is not known but it was probably about 1 percent of that in normal uranium ore. Wagoner, *et al.*, suggested several possible etiological factors, among which was radiation from radon decay products. While being followed for 25,033 person-years, this group experienced 47 lung cancer deaths, approximately three times the expected number. The radon daughter concentrations in those mines in 1958 were estimated to have been in the range of 0.1 to 0.2 WL. The concentrations during earlier years before the advent of improved ventilation are not known but were estimated to have been possibly 5 to 10 times higher. The threefold increase in the observed numbers of lung cancers relative to those expected was attributed by the authors not to be an effect of age, smoking, nativity, urbanization, socioeconomic status, heredity, diagnostic accuracy, or silicosis. The authors concluded that it appeared likely that the mine air contained materials that singly or in combination could be carcinogenic. Wagoner *et al.*, also concluded that chronic exposure to radon or radon daughters alone is unlikely to account for the observed increase, although there appeared to be a higher percentage of undifferentiated cancers than in the comparison group.[50]

Fluorspar Mines

3.19 deVilliers, Windish,[36] and Parsons, *et al.*,[51] have made detailed studies on Newfoundland fluorspar miners. Although uranium minerals could not be identified in the surrounding rock, radon entered the mine in ground water. The average concentration of radon daughters in the mine air was estimated to range between 2.5 and 10 WL. The group studied by deVilliers and Windish contained 630 men who had worked 12 months or more in the mines over a 29-year period (1933–61). Of 69 deaths reported in the group, 26 were attributed to lung cancer. This number is 25 to 40 times the number that might be expected if the lung cancer rates in the study group were compared to those of the adult male population of Newfoundland.[36] Lung cancer deaths were not noted during the first 19 years of operation of the mines.

Joachimsthal and Schneeberg Miners

3.20 Although mines in the Joachimsthal and Schneeberg areas have been operated since about 1500 A.D., the excess mortality was not generally attributed to lung cancer until the 1920 decade.[2 47 52] The radon 222 concentrations in the mine air were estimated to have averaged between 3,000 and 15,000 pCi per liter of air [53 54] (*i.e.*, radon daughter concentrations of 30–150 WL at radioactive equilibrium). The percent of deaths attributed to lung cancer has been reported to vary between 30 and 70.[55 56] Although knowledge about the population size and age distribution of the miners is incomplete, it was estimated in the course of this review that the incidence was possibly more than 20 times what might have been expected.

Relation to Other Agents and Disabilities

3.21 Analysis of medical histories of U.S. uranium miners examined in 1957 and in 1960 showed a greater prevalence of the following complaints which are also observed in other miners: (1) shortness of breath, (2) persistent cough, (3) history of wheezing or whistling sounds in the chest, and (4) history of chest pain or pressure in the chest.[51 57] Ventilatory function studies made during these examinations showed that cumulative exposure to the atmosphere in uranium mines contributes to a loss in ventilatory function in a manner similar to that observed in aging and from cigarette smoking.[51 58]

3.22 Since a relatively high percentage of United States uranium miners smoke cigarettes, and since cigarette smoking has been associated with an increased incidence of lung cancer, it is necessary to consider this agent in the etiology of the lung cancers of uranium miners.[60 61] Although United States uranium miners smoke more than the general population, they smoke no more than some other occupational groups whose lung cancer rate is not comparable to that of uranium miners.[62] Since smoking habits appear not to differ systematically among individuals assigned to the different cumulative exposure categories in table 6, this variable is not considered important to the comparisons drawn between groups in that table. The cigarette consumption of the most highly exposed group was about the same as that of the less exposed groups. Although cigarette smoking may affect the total occurrence of lung cancer among uranium miners (*i.e.*, cancers observed compared to expected), smoking alone does not explain the trend with cumulative WLM. An analysis made by the PHS led to the conclusion that the smoking history of the uranium miners might increase the expected number of lung cancers from 5.13 as shown in table 6 to about 7. The data neither prove nor disprove the existence of a relationship between cigarette smoking and radon daughter exposure in the etiology of lung cancer among uranium miners.

3.23 There is evidence that cigarette smoking contributes materially to nonmalignant pulmonary impairments to which uranium miners are subject.[57 58] Cigarette smoking can influence lung cancer development among uranium miners in other ways—by changing the radiation dose distribution (through alterations in ciliary motion or thickness of the mucus blanket). It might act as a mitotic stimulant to bronchial epithelium which could reduce latent periods or influence the cell type of carcinoma produced. Although cigarette smoking seems to increase the incidence of several cell types of carcinoma, the one most prominently increased is the epidermoid type. This is in contrast to the small cell undifferentiated type which has been reported to be most prominently increased among uranium miners.[48] The possibility that a synergistic relationship may exist between cigarette smoking and exposure to radon daughters needs further study.

3.24 Increased incidence of lung cancer among miners has at times been attributed to silicosis. However, a number of studies suggest that silicosis by itself does not necessarily predispose to lung cancer.[56 63 64] The prevalence of silicosis among United States uranium miners has been reported to be comparable to that among other United States metal miners,[44] but lung cancer rates of the two groups are quite different.

3.25 The observations described in the previous paragraphs are consistent with the older observations from Schneeberg and Joachimsthal that many miners had silicosis and tuberculosis [47 56 59] although Pirchan and Sikl reported that no notable degree of anthracosis

or silicosis was found in the lungs of Joachimsthal miners submitted to autopsy.[52] Miners have undoubtedly experienced parenchymal pulmonary damage from exposure to the atmosphere in uranium mines as well as in other types of mines, quite aside from induced lung cancer. What portion of this parenchymal damage is due to radiation and what portion is due to nonradioactive dust (such as free silica) cannot be determined at this time, but it seems possible that sufficient exposure to radon daughters may enhance and alter the fibrogenic response to silica, and may possibly exert a fibrogenic effect of its own.

3.26 Among the many items considered in assessing the etiology of the Schneeberg and Joachimsthal lung cancers was the possible role of heredity.[56 59 63] Since both mining areas were relatively isolated, it was felt that centuries of inbreeding might have resulted in a high lung cancer strain of people as has occurred among certain strains of mice.[65-67] Although this was a possibility, the development of such high rates of lung cancer has never been observed among isolated nonmining populations. The fluorspar miners mentioned in paragraph 3.19 also constituted a relatively isolated population, but it was noted that a nearby similarly isolated community at Grand Banks, which did no mining, had a low lung cancer rate.[36] It has also been noted that high lung cancer rates have been observed primarily in males of the population. Among U.S. uranium miners, where marked differences in lung cancer rates have been shown between different cohorts,[46] and different exposure groups,[60] there is no reason to believe that hereditary factors were significantly different among the groups. There is no basis on which to implicate a common genetic factor as an important contributor to lung cancer in United States uranium miners.

3.27 The presence of long-lived radioactive materials in the dust of uranium mines [68] has given rise to speculation that these may contribute to the lung cancer rate of uranium miners. The various isotopes of uranium, thorium, and radium have been the principal radionuclides considered. In uranium ores, the relative abundance of these nuclides is generally low. Their concentration in mine dust is therefore a function of the richness of the ore. Uranium determinations on miners' urine have indicated that uranium is not absorbed in sufficient amounts to present a toxic hazard.[69] Thorium isotopes might be expected to concentrate in lymph nodes, spleen, liver, lung, and bone.[73] Radium isotopes would be expected to concentrate in bone. However, no radium could be detected by whole-body counting in several uranium mill workers who had been heavily exposed to ore dust.[70] All the radionuclides in the uranium decay series undoubtedly contribute something to the radiation dose of uranium miners, and there is some indication that thorium 230 may have a longer pulmonary retention than its parent uranium.[71 73] However, the contribution by the immediate daughters of radon is so great that the contribution by other radionuclides to the lung dose during the working years is probably negligible by comparison.

3.28 Since carcinogenic hydrocarbons have been observed to be present in the exhaust from diesel engines,[72] there is the possibility that exposure to the exhaust gases might increase the incidence of lung cancers in uranium miners. There were no diesel engines in the Schneeberg and Joachimsthal mines during the periods when high lung cancer rates were noted. Beginning about 1952 a substantial part of United States production of uranium ore has involved underground diesel equipment. Many of the United States uranium miners in the present study who developed lung cancer had relatively little exposure to diesel fumes in their early work experience. In many nonuranium mines diesel

equipment has been used underground for as long as, or longer than in uranium mines. Gasoline engines are rarely, if ever, used underground.

3.29 Circumstantial evidence appears to rule out diesel exhaust as an important agent responsible for the observed increase of lung cancer rates among uranium miners. However, since diesel exhaust contains the same type of carcinogens as cigarette smoke,[72] it is entirely possible that diesel smoke might contribute in the manner discussed above with respect to cigarettes (pars. 3.22 and 3.23).

Discussion and Conclusions

3.30 Respiratory impairment of several varieties has, for many years, been a recognized hazard of underground mining. Analysis of available evidence permits the conclusion that sufficient exposure to radon 222 and its short-lived radioactive daughters in the mine atmospheres is associated with an increased incidence of lung cancer. The highest incidence of lung cancer is occurring now in the group of miners (1) who worked in mines in which the average concentration of radon daughters was usually higher than 10 WL; (2) whose total cumulative exposures ranged upward from about 1,000 WLM; (3) who started mining uranium ore more than 10 years ago; and (4) who were moderate to heavy cigarette smokers. These observations suggest that additional cases of lung cancer can be expected to develop in the study group.

3.31 The USPHS epidemiological study was designed to identify possible etiologic agents that might be implicated as a reason for the high incidence of lung cancer observed among the uranium miners. Observations on the United States uranium miners who started underground mining before July 1955 clearly demonstrate that an association exists between exposure to radon daughters and a higher than expected incidence of lung cancer when cumulative exposures are more than about 1,000 WLM. The degree of risk at lower levels of cumulative exposure cannot be determined from currently available epidemiological data. It is prudent to assume, however, that some degree of risk exists at any level of exposure, even though effects may not now be evident at the lower levels of cumulative exposure.

3.32 A review of the epidemiological data conducted by the USPHS after the preliminary report 8 was issued in May 1967, and testimony provided to the Joint Committee on Atomic Energy, amplify the previously described uncertainties in the exposure data and their interpretation.[74] These include:

1. The early measurements were very infrequent (sometimes less than once a year) and sampling sites were selected for purposes of control.

2. Information about the mines in which each individual had worked, including the year and the number of months in each, was obtained from an interview with each miner several years later.

3. The information in table 6 is based on estimates of the months worked in underground uranium mines only. Many of the miners also had extensive experience in other underground mines where radon daughters were present, but at lower concentrations. Present evidence suggests that underground uranium mining may account for less than one-half of the total exposure to radon daughters that might be associated with the complete mining experience of the individual when the WLM assigned to underground uranium

mining is in exposure categories A or B (*i.e.*, less than 360 WLM).[74] The addition of exposure experience in other underground mines could result in some redistribution of the 34 lung cancer cases in table 6.

4. The miners in the study group started underground uranium mining before 1955, when radon daughter concentrations up to several hundred WL were commonly found in individual samples. Other factors taken alone (*i.e.*, ore dust, blasting gases, and other noxious agents) do not seem to be implicated as etiological agents for the lung cancers found in underground uranium miners. However, these factors, together with smoking, can influence any quantitative relationship between exposure to radon daughters and the probability of developing lung cancer by their individual or collective effects on lung tissue.

3.33 Several dosimetry models have been developed in an attempt to derive a numerical estimate of the effective radiation dose to the lung that might result from exposure to a given concentration of radon and radon daughters. Details of some of these models are presented in the appendix of this report. The differences among the various models and the uncertainties in the basic radiological parameters that must be used are too large for the models to be acceptable at the present time as a basis for estimating the risk of radiation-induced carcinogenesis. Inference of risk drawn from epidemiological studies of lung cancers associated with exposure to various concentrations of radon and its daughter products appears to be the most satisfactory basis for evaluating the associated lung cancer risk at the present time, although no reliable quantitative statement can be made if cumulative exposures are less than about 1,000 WLM.

REFERENCES

[1] Coffin, R. C. Radium, Uranium, and Vanadium Deposits of Southwestern Colorado. Colo. Geol. Survey, Bull. 16, 1921.

[2] Uhlig, M. Virchows Arch. Path. Anat., Vol. 230, p. 76, 1921.

[3] Ferin, J., Urbankova, G., and Vlekova, A. Pracovni Lekar, Vol. 17, p. 105, 1965.

[4] deVilliers, A. J. Dissertation submitted to Graduate School of Public Health, U. Pittsburgh (partial fulfillment requirement for degree of Doctor of Science), 1964.

[5] Michaelson, S. M., *et al.* Cardiopulmonary Changes in Leg Following Exposure to X-rays. Univ. Rochester Atomic Energy Project, UR-596, 1961.

[6] West, J. E., and Bair, W. J. Plutonium Inhalation Studies V. Radiation Syndrome in Beagles after Inhalation PuO_2. Radiation Research, Vol. 22, p. 489, 1964.

[7] Clarke, W. J., and Bair, W. J. Plutonium Inhalation Studies VI. Pathologic Effects of Inhaled Plutonium in Dogs. Health Physics, Vol. 10, p. 391, 1964.

[8] Altmann, H. W., Lick, R., and Stutz, E. The Histogenesis of Radiation-Induced Squamous Epithelial Carcinoma in Rat Lung Changes in Bronchial Epithelium. Beitrage zur Pathologischen Anatomie und zur Allgemeinen Pathologie, Vol. 125, p. 403, 1961.

[9] Gates, O., and Warren, S. Histogenesis of Lung Carcinoma in Mice Induced by Gamma Radiation. AMA Arch. Path., Vol. 71, p. 693, 1961.

[10] Lisco H. Autoradiographic and Histopathological Studies in Radiation Carcinogenesis of Lung. Lab. Invest., Vol. 8, p. 162, 1959.

[11] Cember, H., and Watson, J. A. Bronchogenic Carcinoma From Radioactive Barium Sulfate. AMA Arch. Industr. Health, Vol. 17, p. 230, 1958.

[12] Clarke, W. J., *et al.* Bronchioloalveolar Tumors of the Canine Lung following Inhalation of Plutonium Particles. Amer. Rev. Resp. Diseases, Vol. 90, p. 963, 1964.

[13] Scott, J. K. The Histopathology of Mice Exposed to Radon. Univ. Rochester Atomic Energy Project, UR-411, 1955.

[14] Altmann, H. W., Hunstein, W., and Stutz, E. On Lung Changes and Lung Tumors in Rats After Irradiation With Radioactive Strontium. Beitrage zur Pathologischen Anatomie und zur Allgemeinen Pathologie, Vol. 124, p. 145, 1961.

[15] Scott, J. K., and Thomas, R. Polonium Induction of Pulmonary Tumors Following Intratrachial Injection. Univ. Rochester Atomic Energy Project Quarterly Report, July September 1957.

[16] Lisco, H., and Finkel, M. P. Observations on Lung Pathology Following Inhalation of Radioactive Cerium. Federation Proc., Vol. 8, p. 360, 1949.

[17] Kuschner, M., *et al.* Radiation Induced Bronchogenic Carcinoma in Rats. Amer. J. Path., Vol. 34, p. 554, 1958.

[18] Laskin, S., *et al.* Carcinoma of the Lung in Rats Exposed to Radiation of Intrabronchial Ruthenium^{106} Pellets: I. Dose Response Relationships. J. Nat. Cancer Inst., Vol. 31, p. 219, 1963.

[19] Laskin, S., *et al.* Tissue Reactions and Dose Relationships in Rats Following Intrapulmonary Beta Radiation. Health Physics, Vol. 10, p. 1229, 1964.

[20] Kotschketkowa, T. A., and Awrunina, G. Arch. Gewerbepath., Vol. 16, p. 24, 1957.

[21] Wager, R. W., *et al.* Toxicity of Radioactive Particles. Biology Research Annual Report for 1955, HW-41500, Hanford Atomic Products Operation, p. 61, 1956.

[22] Lorentze, E., *et al.* Pulmonary Tumors in Mice Following Chronic Gamma Radiation. J. Nat. Cancer Inst., Vol. 6, p. 349, 1946.

[23] Furth, J., and Furth, O. B., Neoplastic Diseases Produced in Mice by General Irradiation With X-rays: I. Incidence and Types of Neoplasms. Amer. J. Cancer, Vol. 28, p. 54, 1936.

[24] Cember, H. Radiogenic Lung Cancer. Prog. Exp. Tumor Research, Vol. 4, p. 251, 1964.

[25] Bullock, F. D., and Curtis, M. R. Spontaneous Tumors of Rat. J. Cancer Research, Vol. 14, p. 1, 1930.

[26] Morken, D. A. Survey of Literature on Biological Effects of Radon and Determination of Its Acute Toxicity. Univ. Rochester Atomic Energy Project, UR-379, 1955.

[27] Jackson, M. L. Biological Effects of Inhaled Radon (thesis for work toward M.S., MIT, 1940) and R. Evans private commun.

[28] Reed, J., and Mottram, J. C. Tolerance Concentration Radon in Atmosphere. Brit. J. Radiol., Vol. 12, p. 54, 1939.

[29] Kahlau, G. Experimentelle Erzeugnung von Lungen Tumoren Durch Radiumemanation. Verhandl. d. Deutsch. Gesellschaft f. Path., Vol. 32, p. 379, 1950.

[30] Unnewehr, F. Durch Radioinhalation Hervorgerufene Tumorbildung und Gewebsveranderung. Strahlentherapie, Vol. 108, p. 421, 1959.

[31] Kushneva, V. S. On the Problem of the Long-Term Effects of Combined Injury to Animals of Silicon Dioxide and Radon. AEC Tr-4473, p. 22, 1959.

[32] Kushneva, V. S. Peculiarities of the Course of Experimental Silicosis with Inhalation of Radon. Proc. IAEA Symposium on Radiological Health and Safety in Mining and Milling of Nuclear Materials. Vienna, p. 317, 1964.

[33] Clarke, W. J., Palotay, J. L., and Bair, W. J. Plutonium Particle Induced Neoplasia of the Canine Lung: II. Histopathology and Conclusions (presented Second International Conf. Lung Tumors in Animals, Perugia, Italy). BNWL-SA-147, April 1965.

[34] Park, J. F., Clarke, W. J., and Bair, W. J. Chronic Effects Inhaled Pu-239 Oxide in Dogs. BNWL-122, 1965.

[35] Report to Congress by the Secretary of the Interior. Health and Safety of Metal and Nonmetal Mines (submitted in response to PL 87-300, 75 Stat. 649). 1963.

[36] deVilliers, A. J., and Windish, J. P. Lung Cancer in a Fluorspar Mining Community: I. Radiation, Dust, and Mortality Experience. Brit. J. Industr. Med., Vol. 21, p. 94, 1964.

[37] Roussel, J., *et al.* Considerations Statisques sur le Cancer Bronchique du Mineur de Fer du Bassin de Lorraine. J. de Radiologie et d'Electrologie, Vol. 45, p. 541, 1964.

[38] Bonser, G. M., Faulds, J. S., and Stewart, M. J. Occupational Cancer of the Urinary Bladder in Dyestuffs Operations and of the Lung in Asbestos Textile Worker and Iron Ore Miners. Amer. J. Clin. Path., Vol. 25, p. 126, 1955.

[39] Wagoner, J. K., *et al.* Unusual Cancer Mortality among a Group of Underground Metal Miners. N. Eng. J. Med., Vol. 269, p. 284, 1963.

[40] Osborn, H. S. Cancer of the Lung in Gwanda. Central African J. Med., Vol. 3, p. 215, 1957.

[41] Oosthuizen, S. F., *et al.* Experience in Radiological Protection in South Africa. Proc. Second UN Inter. Conf. Peaceful Uses Atomic Energy, Vol. 21, p. 25, 1958.

[42] Chatgidakis, C. B. The Incidence of Malignant Disease in 1,620 African Bantu Gold Miners. AMA Arch. Envir. Health, Vol. 1, p. 17, 1960.

[43] Goldman, K. P. Mortality of Coal Miners from Carcinoma of Lung. Brit. J. Ind. Med., Vol. 22, p. 72, 1965.

[44] USPHS Pub. 1076. Silicosis in Metal Mining Industry—Reevaluation 1958-1961 (USPHS and Bureau Mines), 1963.

[45] Rubin, P. Bronchogenic Carcinoma. JAMA, Vol. 195, p. 653, Feb. 1966.

[46] Wagoner, J. K., *et al.* Cancer Mortality Patterns among U.S. Uranium Miners and Millers—1950 through 1962. J. Nat. Cancer Inst., Vol. 32, p. 787, 1960.

[47] Rostoski, D., Saupe, E., and Schmorl, G. Die Bergkrankheit der Erzbergleute in Schneeburg in Sachsen (Schneeberger Lungenkrebs). Ztsch. f. Krebsforsch. Vol. 23, p. 360, 1926.

[48] Saccomanno, G., *et al.* Lung Cancer of Uranium Miners on Colorado Plateau. Health Physics, Vol. 10, p. 1195, 1964.

[49] Yesner, R., Auerback, O., and Gerstl, B. Application of WHO Classification of Lung Carcinoma to Biopsy Material. Annal. Phoracic Surgery, Vol. I, p. 33, Jan. 1965.

[50] Saccomanno, G., *et al.* Concentration of Carcinoma or Atypical Cells in Sputum. Acta Cytologica, Vol. 7, Sept.-Oct. 1963, p. 305.

[51] Parsons, W. D., *et al.* Lung Cancer in a Fluorspar Mining Community: II. Prevalence of Respiratory Symptoms and Disability. Brit. J. Industr. Med., Vol. 21, p. 110, 1964.

[52] Pirchan, A., and Sikl, H. Cancer of the Lung in Miners of Joachimsthal. Amer. J. Cancer, Vol. 16, p. 1681, 1932.

[53] Evans, R. D., and Goodman, C. Determination of Thoron Content of Air and its Bearing on Lung Cancer Hazards in Industry. J. Industr. Hyg. and Toxicology, Vol. 22, p. 89, 1940.

[54] Mitchell, J. S. Memorandum on Some Aspects of the Biological Action of Radiations with Special Reference to Tolerance Problems. Montreal Lab. Report, HI-17, Nov. 20, 1945.

[55] Peller, S. Lung Cancer among Mine Workers in Joachimsthal. Human Biology, Vol. 11, p. 130, 1939.

[56] Hueper, W. C. Occupational Tumors and Allied Diseases. C. C. Thomas, Springfield, Ohio, 1942 (pp. 435–468).

[57] Archer, V. E., *et al.* Epidemiological Studies of Some Nonfatal Effects of Uranium Mining. IAEA Symposium on Radiological Health and Safety in Mining and Milling of Nuclear Materials, Vienna, Vol. 1, p. 21, 1964.

[58] Archer, V. E., Brinton, H. P. and Wagoner, J. K. Pulmonary Function of Uranium Miners. Health Physics, Vol. 10, p. 1183, 1964.

[59] Lorenz, E. Radioactivity and Lung Cancer—A Critical Review. J. Nat. Cancer Inst., Vol. 5, p. 1, 1944.

[60] Wagoner, J. K., *et al.* Radiation as the Cause of Lung Cancer among Uranium Miners. New Eng. J. Med., Vol. 273, p. 181, 1965.

[61] Seltzer, R. Lung Cancer and Uranium Mining, A Critical Review. AMA Arch. Env. Health, Vol. 10, p. 923, 1965.

[62] Dunn, J. E., Linden, G., Breslow, L. Lung Cancer Mortality Experience of Men in Certain Occupations in California. Amer. J. PHA, Vol. 50, p. 1475, 1960.

[63] USPHS Pub. 1103, Report of the Advisory Committee to the Surgeon General of the Public Health Service; Smoking and Health, 1964.

[64] Gardner, L. U. Fourth Saranac Symposium on Silicosis, 1939.

[65] Macklin, C. C., and Macklin, M. A. A Critical Review of the Usual Methods of Investigation of the Role of Extrinsic Causes in Producing Human Cancers, Especially of the Lung. Acta Union Internat. Contr. Cancrum, Vol. 6, p. 1334, 1950.

[66] Little, C. C. Hybridization and Tumor Formation in Mice. Proc. Nat. Acad. Sci., Vol. 25, p. 452, 1939.

[67] Stewart, H. L. Pulmonary Tumors in Mice—The Physiopathology of Cancer published by P. B. Hoeber and Co.—Harper and Row, New York—1959 (pp. 18–37).

[68] Couix, A., and Pradel, J. Methodes de Mesure des Poussieres Radioactive a Vie Longue. IAEA Symposium on Radiological Health and Safety in Mining and Milling of Nuclear Materials, Vol. 1, p. 425, 1964.

[69] Smith, C. F. The Control of Radiation Hazards in Canadian Uranium Mines and Mills With Specific Reference to the Beaverlodge Operation of Eldorado Mining and Refining Ltd. IAEA Symposium on Radiological Health and Safety in Mining and Milling of Nuclear Materials, Vol. I, p. 67, 1964.

[70] Archer, *et al.* Hazards to Health in Uranium Mining and Milling. J. Occup. Med., Vol. 4, p. 55, 1962.

[71] Hill, C. R. Identification of Alpha Emitters in Normal Biological Materials, Health Physics, Vol. 8, p. 17, 1962.

[72] Lyons, J. J. Comparison of Aromatic Polycyclic Hydrocarbons From Gasoline Engine and Diesel Engine Exhausts, General Atmospheric Dust, and Cigarette Smoke Condensate. Symposium Analysis Carcinogenic Air Pollutants, NCI Monograph No. 9, 1962.

[73] Stewart, B. O., and Beasley, T. M. Nonequilibrium Tissue Distributions of Uranium and Thorium Following Inhalation of Uranium Ore by Rats. Proc. Second International Symposium on Inhaled Particles and Vapours, Cambridge, England, 1965.

[74] USPHS Report on a Review and Evaluation of the Public Health Service Epideminological Study of Lung Cancer among Uranium Miners. Submitted to the Joint Committee on Atomic Energy, 1967.

SECTION IV

CONTROL CAPABILITIES IN URANIUM MINES

4.1 Several procedures are available to control exposure of miners to radon and its radioactive daughters in mine atmospheres. These include: (1) removing radon and its radioactive daughters from mine areas by air replacement, (2) inhibiting the diffusion of radon into the air of the work areas from the surrounding rock or from abandoned workings, (3) limiting the time individuals can work in areas with an excessive air concentration of radon-radon daughters (work force management), and (4) reducing the concentration of radon daughters in the inspired air from that prevalent in the mine atmosphere.

4.2 Ventilation of underground work areas is the most common procedure. Natural draft ventilation is rarely adequate to provide the needed rate of air supply. Therefore, mechanical movement of the large volumes of air needed into or out of uranium mines is now the general practice. Portable air ducts and auxiliary fans are commonly used to conduct fresh air from its supply source through passageways to deliver the air to work areas in all types of underground mining. The incoming air dilutes and removes air fouled by dust, drill exhaust, blasting fumes and gases, and exhaust gases and heat from internal combustion engines. Ventilation is also necessary for comfort and health in underground mines of all kinds. In uranium mines these same considerations hold, but in addition ventilating air serves to remove radon and radon daughters as well as the dust and noxious gases.

4.3 Currently the most practical means for removing radon and its daughters from mines is to remove the contaminated air to the outside and replace it with fresh air. Mine ventilation procedures must take into account the size, number, and complexity of mine workings and the level of radon and its daughter products present within these workings. The concentration of radon and its daughters in mine atmospheres, if not diluted and carried away, can build up to very high levels (*i.e.*, hundreds of WL). Continuous planned ventilation is much more effective than an intermittent supply or exhaust of air, or solely relying on ventilation by natural means.

4.4 The general considerations involved in ventilation control include:

1. Fresh air supply should be channeled through the mine passageways in such a manner that it would avoid mixing with contaminated air. The displaced air should be promptly exhausted aboveground at a distance from the air intake.

2. Old workings not needed for other purposes should be sealed off to inhibit the release of radon into the work areas. This will also decrease the concentration of radon daughters that can build up in the work areas.

3. Ventilation systems must be promptly altered or modified as development proceeds.

4. Air velocities through mine areas where men work must be kept within practical comfort limits.

4.5 The volume of fresh air that can be supplied to underground mine workings is subject to physical limitations on the air velocity and pressure within the portable air ducts, and the velocity of the displaced air through exhausting passageways. In regard to the former, the power requirements to move a unit volume of air through a duct of a given cross-sectional area are reported to increase with the third power of the volume. The air velocity in passageways directly affects the miners.[1] There are valid objections to working in a high velocity air flow, especially when the temperature is low. Underground work crews, particularly those working continuously in passageways, object to being subjected to uncomfortably high air flow rates, even though the air temperature is maintained at a reasonable level.

4.6 Mines that are developed with foreknowledge of the ore body extent and with engineering design for mining and ventilation generally provide mechanical equipment to move air into and out of the mine through multiple openings with the least possible dilution or recirculation. A Bureau of Mines survey [2] has related radon daughter levels in underground uranium mines to the various ventilation practices; that is, with fans blowing or exhausting, with fresh air intake via the main mine entrance, or with auxiliary ventilation holes and the total volume of fresh air supplied. The results do not indicate any systematic differences among these procedures. Regardless of the system, however, multiple exits are advantageous in shortening air retention time.

4.7 Prior to 1960 mine surveys were infrequent and in many cases the results were not representative of the average concentration of radon daughters in the mine air. Since 1960 State agencies and the major mining companies have conducted systematic monitoring in underground mines. Tables 4 and 5 (sec. II) show that a significant reduction in the higher concentrations of radon daughters in mine air has been achieved since 1960.[3] Although the number of mines reporting average concentrations of 1 WL or less is unchanged, average concentrations of 10 WL or more in mines have been virtually eliminated. The percentage of mines reporting average radon daughter concentrations in the range of 1 to 3 WL has increased. Public statements by mine association and company officials in States having most of the underground uranium mines suggest that the improvement is continuing.[4-6]

4.8 A meaningful comparison of mine ventilation cost from mine to mine is difficult because the evolution of ventilation systems is intimately associated with and conditioned by individual mine development. While it might be supposed that the cost of ventilation in uranium mines would be greater than the cost for ventilation in nonuranium mines, or that the cost of ventilation in mines with a lower grade ore would be less than that for those with a higher grade ore, this is not necessarily the case. Many other cost-affecting features may be of greater significance than air supply alone. These include such factors as depth below surface, lateral extension of workings, arrangement of passageways, number and size of mine openings, and so forth. Because of the general lack of specific information in this area, special analyses were necessary to evaluate the cost-effect of ventilation rate. A number of uranium mining companies have carried out and reported on mine ventilation cost studies at the request of FRC staff (see tables 7 and 8). These estimates are intended to illustrate the general magnitude of cost in a few selected mines and are not applicable to the industry as a whole.

4.9 One study concerns 11 of the larger underground mines that produced more than 20 percent of the national total of uranium ore. These mines have all been in operation for 6 to 7 years, and it seems likely that they could continue in operation for several more years if, after the end of the Government procurement program in 1970, there is a continuing favorable market. These mines are individually profitable enterprises and, in general, they exemplify predevelopment planning for advantageous operation, including the extra ventilation necessary for radon daughter control. They are similar in operation and depth from the surface. The reported estimates are for the group as a whole.

4.10 To obtain a general estimate of an "exposure index" in these 11 mines the following procedures were used. The concentrations of radon daughters in the mine air were determined by taking 10-minute air samples at about monthly intervals at underground work locations. The results of these determinations were then correlated with the time of occupancy by various categories of mine workers to give approximate time-weighted average exposures. In this way, an "exposure index" related to the WL was derived for each of the various work categories. These average exposures were then weighted by the number of persons involved in the respective categories to reach a "mine index" value representing the average exposure in the mine. The average mine index value for the 11 mines was reported as about 2 WL in 1965.

4.11 Total costs are reported for the 11 mines collectively in terms of "investment costs" and "annual operating costs." Based on this cost experience covering 6 or 7 years, the mining companies have also derived estimates of what these costs might be for two hypothetical cases: (1) the investment cost and operation expense necessary to provide the nominal ventilation that would have been needed for these mines without considering radon control (the prevailing mine index value would have been about 10 WL), and (2) the investment and operating costs that would be entailed in the further reduction of the prevailing mine index to 1.0 WL.

4.12 With these estimated costs, the extrapolated "10-year" cost of mining for operation of mine indexes of 10 WL and 1 WL were estimated. This extrapolation is intended to provide some appreciation of the way total cost might vary under progressively more stringent control requirements. These figures will differ from the actual costs as follows:

1. Ore bodies that do not last for 10 years will have less than the extrapolated 10-year operating cost and more than the stated investment cost, since the investment would have to be repeated when a new ore body is opened.

2. Ore bodies that do allow operation for 10 years will require additional investment and operating costs as the present operations are extended. These costs cannot be estimated at the present time because the rate of new development in particular locations cannot be predicted that far in advance. The relationships are shown in table 7.

4.13 Another study concerns estimates for each of 3 mines where conditions differ from the group of 11 mines as to geology, depth, extent of workings, productive capacity, arrangement of passageways, number of openings, and so forth. Furthermore, the economy of operations is so different that the data reported for these 3 mines are not comparable to the group of 11 mines discussed above, and perhaps not even comparable among themselves. Collectively, the annual production of uranium ore of the three mines has been at a rate of about 2 percent of the national total. This study presents ventilation investment costs

and ventilation operating costs over a 6-year period of continual improvement effort together with current concentration values of radon daughters in all mine areas of concern. It should be noted that air concentration values are reported rather than a mine index. The WL values reported in table 8 are averages for 1965 and represent the present state of control development. The costs are then compared with corresponding costs that would have been incurred if the ventilation had been limited to that which would have been needed in these mines without considering radon control. It has been estimated that the radon daughter concentrations would then have been in the range of 5 to 20 WL with this limited ventilation.

TABLE 7.—*Ventilation cost estimates—11 mine study*

	Investment cost	Oper. cost (10-yr. est.)	Total cost (10 yrs.)	Estimated mine index WL
	Millions of dollars			
Past experience	3.9	7.9	11.8	[a] 2
Estimated ventilation costs without radon control	2.0	2.8	4.8	[b] 10
Additional cost radon control from 10 WL to 2 WL	1.9	5.1	7.0	
Estimated additional cost to reduce from 2 WL to 1 WL	1.5	6.0	7.5	1
Total cost to control at 1 WL—10 years			19.3	

[a] Composite mine index for 1965.

[b] Estimate of what the composite mine index would be with normal ventilation practices.

TABLE 8.—*Ventilation cost estimates—3 mine study*

	Investment cost	Oper. cost (6-yr. est.)	Total cost (6 yrs.)	Average concentration WL
	Thousands of dollars			
Past experience				
Mine A	361	120	481	[a] 1.4
Mine B	321	85	406	[a] 1.5
Mine C	75	50	125	[a] 1.5
Total	757	255	1,012	
Estimated for case of minimum ventilation				
Mine A	63	21	84	[b] 5 to 20
Mine B	66	18	84	
Mine C	06	04	10	
Total	135	43	178	

[a] Average WL concentrations in 1965.

[b] Estimate of what the average WL concentrations would be with normal ventilation practices.

4.14 This study indicates that radon daughter control at 1.5 WL in these mines is being achieved at a total cost of about $834,000 over a 6-year period. The relation of ventilation cost to tons of ore produced for the group of three mines is about 1.5 times the corresponding relation in the 11 mine group because of differences in size and other factors.

Inhibiting the Diffusion of Radon

4.15 Sealing off old workings not needed for other purposes is common practice and is effective, but the use of various types of coatings sprayed on the mine walls has proved to be ineffective in reducing the diffusion of radon from the walls into the work areas. Variations in barometric pressure have been shown to have an inverse effect on the rate of radon emanation into mines and, therefore, on the concentration of radon and radon daughters in mine air.[7] This suggests that an applied overpressure might be used to advantage in some mines for reducing the release rate of radon into mine air. Experiments designed to augment the normal atmospheric pressure in mines with an overpressure approximately equal to the usual range of atmospheric pressure variation have been performed. A pressure of 0.6 cm of mercury above normal reduced the rate of radon release to mine air by a factor of about 5. It has also been reported that if the pressure can be lowered in a nearby underground area—removed perhaps a hundred feet from the mine workings—the resulting flow of air into the rock can induce a flow of radon away from the work spaces. The practical value of these suggestions for mine application remains to be demonstrated on an engineering scale. However, an electrical analog system that permits analysis of the air flow in various mine configurations at different simulated air pressures has been developed.[8] Many of the primary factors can be investigated cheaply without the necessity of large scale engineering experimentation.

Work Force Management

4.16 Although limiting a miner's occupancy time in relatively high concentrations of radon or its daughter products has not been a normal practice in uranium mining, this procedure has been used in various activities of the nuclear industry when men routinely work in radiation fields of varying intensities. Since the concentrations of radon daughters vary widely with location and with time at a single location in a mine (from 1 WL in well ventilated areas to several hundred WL in stagnant areas), it has been difficult to establish an accurate record of the time-weighted average exposure for each worker. However, reasonable estimates of the average values are possible, and improvement in the evaluations can be expected in the future. Controlling a miner's exposure to radon daughter concentrations by controlling the time he is allowed to work in different atmospheres should generally be feasible and not too restrictive, provided the radon daughter concentrations are known and are not too high.

4.17 It is normal practice to control mine operations primarily on the basis of estimated exposures to radon daughter concentrations found in a working area at the time of sampling. The Colorado Bureau of Mines, for example, assumes that in a small mine with less than 15 men an individual miner will not spend more than 50 percent of his time in areas with significant concentrations of radon daughters. In larger mines, where most of the men stay underground for a full working shift, the men are assigned an exposure value of 75 percent of the average value for the mine.

4.18 The basic philosophy of control in Colorado is that if the maximum radon daughter concentration in working areas is kept within acceptable limits, the individual worker will be adequately protected without reference to mine averages or time-weighted exposure calculations. New Mexico also limits maximum concentrations in mines, but places more emphasis on estimates of time-weighted exposures in its control program. Both regulatory programs are derived from recommendations made in 1960 by Committee N-7 of the USA Standards Institute. Acceptable limits, as recommended in the Institute's standard are:

(a) "If the 13-week weighted average exposure of the workers to radon daughters, is less than the MPC, the conditions may be considered to be controlled and no action is necessary.

(b) "If samples in any working area show a concentration of radon daughters exceeding the MPC, but less than three times this level, sufficient additional measurements shall be taken to determine the worker's weighted average exposure for 13 weeks.

(c) "If samples show a concentration of radon daughters more than 3 times the MPC, but less than 10 times this value, corrective action shall be initiated.

(d) "If samples show a concentration of radon daughters greater than 10 times the MPC, immediate action shall be taken to reduce the worker's exposure and correct the condition."

The MPC referred to in the Institute's recommendations is equivalent to 1 WL of radon daughters in the mine atmosphere.

4.19 Integrating personnel exposures to a varying radiation field by providing a personal dosimeter, such as a film badge, is standard practice for controlling exposure to external sources in the nuclear industry. A film badge capable of recording the alpha particles emitted from nuclides in mine air has been developed by the mining industry and its reliability is being tested. With proper calibration, such a device may have utility as an integrating dosimeter. The availability and use of a reliable integrating dosimeter may permit more effective estimates of exposure than can be achieved by calculation of time-weighted average exposures. In addition, a detection system permitting continuous measurement and remote readout of radon daughter concentrations in various mine areas are under development.

4.20 The possibility that the quantities of the longer-lived lead 210 (RaD) or polonium 210 (RaF) in various body tissues could serve as an index of the total quantities of radon and radon daughters taken in has been investigated. Concentrations of these radionuclides in blood, excretion in urine, and deposition in hair, teeth, and bone have been examined; however, present information does not permit correlation of these quantities directly to lung dose.

Respirators

4.21 Reducing the concentrations of radon daughters in inspired air, as compared to the concentrations in the mine air by the use of respirators, is an obvious exposure control technique. Available estimates suggest that an ordinary surgeon's mask could give a 2- to 5-fold reduction. More efficient filters with low impedance might achieve a 5- to 100-fold reduction. Respirators would be primarily effective for the partial removal of radon daughters. Radon concentrations would not be affected. Therefore, respirators can be

useful as ancillary devices to reduce exposure when the radon daughter concentrations in the mine atmosphere are relatively high. The primary problem with respirators now in use is that it is very difficult to assure that they are used and that they are worn properly, even if the design of the respirator is appropriate to mining operation.

4.22 The use of respirators or self-contained breathing devices in United States uranium mines has been generally reserved for emergencies or control of silica dust. It has been reported that respirators that reduce the radon daughter products in inhaled air are worn by uranium miners in some other countries.[9] Since it is accepted that there should be more stringent control over the internal radiation exposure received by uranium miners, the Council staff strongly recommends that there be a concerted effort to improve respiratory devices. It is imperative that they be reliable and that they be acceptable to the miners. In most cases, underground uranium miners wear additional equipment, such as the hard hat, miners' lamp and battery (which may weigh up to 6 pounds), safety boots, and in some cases safety lines. In addition to his regular equipment a miner may also have to carry many different pieces of equipment and tools. His work sometimes requires overhead work, barring of loose rock, crawling in cramped spaces, and climbing ladders. If a respiratory device is to be imposed as an additional requirement it must not interfere with the miner's vision or with his freedom of motion to such an extent that it results in a net increase in his probability of incurring a serious accident. Furthermore, since oral communication is of major importance in mines, any respirator design should take this into account.

4.23 Use of respirators by miners should not deter mine management from providing adequate ventilation in their mines. Ventilation remains the primary means by which radon daughter concentrations can be controlled. If the basic principles of ventilation engineering and design are closely followed, radon daughter concentrations in mines can be significantly reduced.

REFERENCES

[1] Coleman, R. D., *et al.* Radon and Radon Daughters Hazards in Mine Atmospheres Investigations on Supplemental Control. AIHI Quarterly, *Dec.* 1956.

[2] Report to Congress by Secretary of the Interior. Health and Safety of Metal and Nonmetal Mines (submitted in response to PL 87 300, 75 Stat. 649), 1963.

[3] Colorado Bureau of Mines Annual Report, 1965.

[4] Romney, Miles P. Utah Mining Assoc. Testimony, p. 373, U.S. Congress, Senate, Committee on Labor and Public Welfare. To establish a Federal Metal and Nonmetallic Mine Safety Act: Hearings before Subcommittee on Labor, 89th Congress, 2d session.

[5] Swent, L. W. Homestake Mining Co., N. Mex. Testimony, p. 408, U.S. Congress, Senate, Committee on Labor and Public Welfare. To establish a Federal Metal and Nonmetallic Mine Safety Act: Hearings before Subcommittee on Labor, 89th Congress, 2d session.

[6] Beamer, R. W. Wyoming Mining Assoc. Testimony, p. 474, U.S. Congress, Senate, Committee on Labor and Public Welfare. To establish a Federal Metal and Nonmetallic Mine Safety Act: Hearings before Subcommittee on Labor, 89th Congress, 2d session.

[7] Schroeder, G. L., Evans, R. D., and Kramer, H. W. Effect of Applied Pressure on Radon Characteristics of an Underground Mine Environment Trans. Society of Mining Engineers, March 1966.

[8] MIT Annual Progress Report (1966), MIT 952 3, Radium and Mesothorium Poisoning and Dosimetry and Instrumentation Techniques in Applied Radioactivity.

[9] Proc. IAEA Symposium on Radiological Health and Safety in Mining and Milling of Nuclear Materials, Vienna, p. 32, 1964.

SECTION V

SUMMARY AND RECOMMENDATIONS

5.1 To provide a Federal policy on human radiation exposure, the Federal Radiation Council was formed in 1959 (Public Law 86–373) to ". . . advise the President with respect to radiation matters, directly or indirectly affecting health, including guidance for all Federal agencies in the formulation of radiation standards and in the establishment and execution of programs of cooperation with States." The present report fulfills this responsibility with regard to a Federal policy relating to the protection of underground uranium miners against deleterious health effects resulting from the inhalation of the radioactive daughters of radon 222. Although primary emphasis has been placed on underground uranium mining, the guidance contained in this report may be applied, as necessary, to any type of mine where similar concentrations of radon daughter products may be found.

5.2 This report emphasizes the radiation hazard associated with the inhalation of radon daughters as they occur in the air of underground uranium mines. Although other sources of radiation exposure occur in connection with underground mining, and potential radiation effects are not necessarily confined to the respiratory system, they are considered of secondary importance as compared to the possible effects resulting from the inhalation of radon daughters.

5.3 In prior reports the Federal Radiation Council has expressed the philosophy that guidance for radiation protection involves achieving a balance between the risk of radiation-induced injury and the benefits derived from the practice causing the exposure to radiation. An implicit part of such a balance is a necessity for considering the relation between the difficulties involved in reducing the radiation exposure by a given amount and the risk that might be associated with that amount of exposure.

Evidence of Radiation Hazards in Underground Uranium Mines

5.4 Available information on the radiological factors involved in the underground mining of uranium ore has been carefully examined. The findings of immediate interest for establishing radiation protection guidance are:

1. It has been observed that underground uranium miners have a higher incidence of lung cancer than is found in the male population in the same geographic area. Continued exposure to the radioactive decay products of the naturally occurring gas radon 222 has been implicated as an important cause of this increased incidence. The decay products of radon 222 (radon daughters) of interest are: polonium 218, lead 214, bismuth 214, and polonium 214. For purposes of this report, the radiation dose from radon itself is not considered.

2. The principal radiation hazard is associated with the inhalation of mine air containing radionuclides that irradiate lung tissue nonuniformly. The most serious result is

the development of lung cancer, which generally does not appear for 10 to 20 years after the individual started uranium mining.

3. In most domestic underground uranium mines the measurement of radon daughter concentration is obtained by using a specific field method. The results are then compared to the "Working Level" (WL), a unit defined as any combination of radon daughters in 1 liter of air that will result in the ultimate emission of 1.3×10^5 MeV of potential alpha energy. The concentration of radon daughters in air of unventilated underground uranium mines ranges from a fraction of a WL to several hundred WL. Exposure to radon daughters over a period of time may be expressed in terms of cumulative "Working Level Months" (WLM). Inhalation of air containing a radon daughter concentration of 1 WL for 170 working hours results in an exposure of 1 WLM.

4. Observations on United States uranium miners who started underground mining before July 1955 clearly demonstrate that an association exists between exposure to radon daughters and a higher than expected incidence of lung cancer when the cumulative exposures are more than about 1,000 WLM. The degree of risk at lower levels of cumulative exposure cannot be determined from currently available epidemiological data. As discussed in paragraph 3.14, the data do not suggest or exclude the existence of a threshold. It is prudent, however, to assume that some degree of risk exists at any level of exposure, even though possible effects may not now be evident at the lower levels of cumulative exposure.

5. The highest incidence of lung cancer is now occurring in the group of miners: (1) who worked in mines in which the average concentration of radon daughters was usually higher than 10 WL; (2) whose total cumulative exposures were estimated to range upward from about 1,000 WLM; (3) who started mining uranium ore more than 10 years ago; and (4) who were moderate to heavy cigarette smokers. These observations suggest that additional cases of lung cancer can be expected to develop in the study group.

Factors Related to the Evaluation of Benefit and Control Capabilities

5.5 Available information on benefits to be derived from the mining of uranium ores, difficulties encountered in reducing radon daughter concentrations from previous levels to current levels, and the additional difficulties that can be anticipated if further reductions in radon daughter concentrations are required has also been reviewed. The findings of immediate interest to the derivation of guidance for radiation protection in uranium mining are as follows:

1. Uranium is currently the basic fuel needed for the development of nuclear energy, and all projections point to an increasingly important role for nuclear energy in meeting national electric power requirements.

2. Uranium mining is an important economic asset to the States in which the ore is mined. In addition to the value of the ore, mining provides important opportunities for employment. It is estimated that the work force will vary between 2,000 and 5,000 men in the next decade.

3. A significant reduction in the concentration of radon daughters in the air of underground mines has been achieved by the industry since 1960. Estimates of probable exposures of 2,177 miners in a five-State area during the third quarter of 1966 indicate that about 31 percent of miners were exposed to levels of 1 WL or less; 55 percent were exposed in the range of 1–3 WL; and about 14 percent were exposed to levels higher than 3 WL.

4. Ventilation with fresh air is presently considered the most feasible technique for controlling the concentrations of radon daughters in mine air. The highest concentrations are usually found in the stope areas where some work must be done before ventilation can be brought directly to the miner's working area. This suggests that there are practical limits to the degree by which radon daughter concentrations in mine air can be controlled by ventilation alone. Available information suggests that it may be physically impossible to maintain radon daughter concentration in all parts of the mine to as low as 1 WL at all times.

5. Research studies have been made on possible procedures that might be used to block the diffusion of radon from rock into mine air. Positive pressure ventilation can be useful if the rock is porous enough to permit air to flow into it. Coating the rock surface might be useful if it is capable of blocking the diffusion of radon.

6. The effectiveness, feasibility, and safety of various types of auxiliary respiratory protective equipment, as they might be used in underground uranium mining, deserve the most thorough study.

7. It is common practice to limit the highest radon daughter concentrations in which normal mining operations are allowed without determining the radiation exposure at lower concentrations. This procedure makes it possible to estimate the maximum exposure rate, but does not provide the information necessary for estimating a true average exposure, and hence the radiation risk for any one individual or group.

Guidance for Radiation Protection in Underground Mines

5.6 On the basis of the preceding findings, the Federal Radiation Council has concluded that radon and its radioactive daughter products occur in sufficient concentrations in underground uranium mines to require actions to control the potential radiation exposure associated with working in such environments. Although primary emphasis has been placed on underground uranium mining, the guidance contained in this report may be applied, as necessary, to any type of mine where similar concentrations of radon daughter products may be found.

5.7 The Council has considered: (1) the apparent relationship between cumulative exposure to radon daughters and the risk of subsequent development of lung cancer as shown by presently available epidemiological data; (2) the range of annual exposures received by various categories of miners now engaged in uranium mining; (3) the technological problems involved in achieving control to various levels of annual exposure, and the ability of present technology as practiced by the industry to reduce radon concentrations to different levels; (4) improvements that might be introduced by application of a more advanced technology, and the length of time such improvements might take; and (5) the magnitude of the radiation risk in the light of the other risks that are faced in underground uranium mining, and the impact of the Council's recommendations might have on efforts to reduce these risks simultaneously.

5.8 The selection of the proposed standard for the control of radon daughters in underground mine air must necessarily involve a judgment based on all relevant information. In selecting the standards the Council has also considered: (1) the possible magnitude of the cumulative radiation exposure that individuals might receive under the practical application of the proposed standard; (2) the range of individual risk that might result;

(3) the practical difficulties and feasibility of reducing exposures; (4) the change in individual risk that could be associated with such reduced exposures; and (5) various control standards used by different countries for control of radon and radon daughter concentrations in underground uranium mines.

5.9 On the basis of the information presented in the preceding sections, the following guidance is recommended:

1. Occupational exposure to radon daughters in underground uranium mines be controlled so that no individual miner will receive an exposure of more than 6 WLM in any consecutive 3-month period and no more than 12 WLM in any consecutive 12-month period. Actual exposures should be kept as far below these values as practicable.

2. Areas in underground uranium mines, whether normally or occasionally occupied, be monitored for the concentration of radon daughters in the mine air. The location and frequency of taking samples should be determined in relation to compliance with recommendation 1.

3. Appropriate records of the exposure from radon daughters in the mine air received by individuals working in uranium mines be established and maintained.

5.10 The Federal Radiation Council recognizes that current mining conditions are much better than those prevailing 10 years ago. However, it also considers that more improvement is needed to provide proper control of exposure to radon daughters. Steps to make improvements should be initiated immediately and made operational as soon as possible.

Research and Development Needs

5.11 The Federal Radiation Council recognizes that present regulatory procedures and the presently used technology are not adequate to insure compliance with the foregoing recommendations. The development of the appropriate technology and the modification of existing regulatory procedures needs to be supported by an applied research and development program. The general areas deserving attention include: (1) the development of the technology directed to the control and more reliable estimate of individual exposures to radon daughters; (2) registration and compilation of individual exposure records; (3) causal relationships between varying exposures to radon daughters and subsequent development of disability; and (4) improvement of mining practices. In addition, continued attention needs to be given to the development of adequate compensation procedures and the provision of educational opportunity and training programs wherever needed.

5.12 The technology concerned with the control and estimate of individual exposure to radon daughters include: (1) the development of more sensitive and more rugged equipment for the measurement of radon daughter concentrations in mine air; (2) the development of continous air monitoring equipment; (3) the development of devices permitting measurement of integrated exposures (personnel dosimetry); (4) the development and testing of low impedance respiratory protective devices; and (5) a more precise definition of the composition of the mine atmospheres, including (a) a measurement of radon 222 and each of its first three daughters separately in representative mine air, (b) the distribution of the three daughters between ions, nuclei, and various larger particle sizes, and (c) the conditions under which these may be altered by varying modes of ventilation, or by various designs of respirators.

5.13 The practical determination of what is needed to implement the recommendations should take cognizance of the following: (1) data needed to indicate compliance with recommendation 1; (2) an evidentiary record needed to support or deny claims for occupational disability; (3) studies to evaluate possible causal relationships between exposure to radon daughters and the incidence of lung cancer at the lower cumulative exposure levels; and (4) development of a basis for estimating cumulative occupational exposure that the average miner might receive in the future under this guidance.

5.14 In regard to causal relationships, epidemiological studies should be continued on uranium miners and expanded to include other miners who could serve as appropriate comparison groups. Such studies should include, with proper categorization, low, intermediate, and high radon -radon daughter exposure groups as well as a health history followup.

5.15 The development of improved mining practices should place the emphasis on mine-planning as it relates to ventilation, removal of radon daughters from underground mine air, and reduction of radon emission from the rock into the mine. This program is primarily directed to the control of the mine air environment.

APPENDIX

DOSIMETRIC AND RADIOBIOLOGICAL CONSIDERATIONS RELATED TO THE ANALYSIS OF RADIATION HAZARDS IN URANIUM MINING

1. Since the observed lung cancers appear to arise primarily in the bronchi near the hilus of the lung, most authors concerned with the dosimetric and radiobiological aspects of the problem assume the relevant biological target to be the basal cells in the bronchial epithelium. It is further presumed that the principal radiation dose arises from the alpha particles (see table 3, sec. II) released by the decay of the short-lived radioactive decay products of radon on or in the mucous membrane of the bronchi. The small contribution of alpha particles released from radon gas in the air passages or from radon gas dissolved in the mucus covering the walls of the bronchial passages and beta or gamma radiation originating from any of the radon decay products is disregarded in the present analysis. It is further assumed that, except for the extent that they carry the short-lived daughter products of radon, the airborne dust particles (par. 2.1) contribute only indirectly to the risk of lung cancer, but the point is not absolutely established.

2. The immediately relevant nuclides start with polonium 218 and terminate with polonium 214; the intervening short-lived nuclides come into radioactive equilibrium with the parent radon 222 in about 3 hours. The formation of lead 210, with its 22-year half-life, then effectively blocks the series; its formation in the respiratory system is inconsequential. Bismuth 214 has an alternative mode of disintegration by alpha emission to thallium 210, which then decays by beta emission to lead 210; however, the fraction of the disintegrations following this mode is too small to be of consequence in the present analysis.

Range of Alpha Particles in Tissue

3. One of the most important radiobiological parameters is the range of alpha particles in tissue. Alpha particle ranges as a function of energy are accurately known in air, many simple gases, and a few solids, notably mica, which can be manipulated in thin sheets. The range in liquids is estimated by calculations based on the summation of stopping powers in an aqueous material of unit density. However, the actual ranges in real tissue depend primarily on the tissue density and the energy of the alpha particle.

4. Large differences in the range for the 6.0 MeV alpha particle from polonium 218 are reported by different authors; Lea [1] calculates a range of 47μ, while Jacobi [2] gives 55μ. Since most radiological physicists accept the values given by Lea, his values (table 1) have been used for the calculations in this appendix. It is recognized, however, that what would appear to be a simple physical parameter, is actually subject to an important degree of uncertainty.

Linear Energy Transfer Along Alpha Particle Tracks

5. The primary ion density of an alpha particle is relatively high throughout the track and increases sharply as the energy of the particle falls below 2 MeV. Typical values as

given by Lea [1] are shown in table 2. If it is assumed that virtually all alpha particles penetrating to sensitive tissue in the bronchial epithelium are already depleted to an energy of about 2 MeV, the effective ionization would all take place near the peak of the Bragg Curve. More importantly, the possible role of the delta rays (secondary electrons) as contributors to the total ionization has apparently not been pointed out. At 2 MeV the delta rays produce about 80 percent as much ionization as the primary alpha particle ionizations. Because of their lower linear energy transfer and low mass, the delta rays produce a "fuzz" of ionization around the alpha track whose total track length is two to three times that of the primary track. The ionization produced by delta rays is considered primarily in relation to the detailed mechanisms by which ionizing radiation may initiate changes that ultimately lead to injury. It is conceivable that the delta rays could play a significant role in the physical dose actually delivered to the critical tissue. However, the empirical observations of biological effects associated with exposure to stated quantities of radon daughters must inevitably include whatever effects of delta ray ionization are present.

TABLE 1. - *Energies and ranges of alpha particles in tissue*

Nuclide	Energy (MeV)	Range in microns
^{222}Rn	5.49	41.1
^{218}Po	6.00	47.0
^{214}Po	7.69	70.8
^{210}Po	5.30	38.9

TABLE 2.—*Ion density in soft tissue*

Alpha particle energy (MeV)	Primary ionizations per micron of tissue
1	5,207
2	2,883
3	2,031
4	1,581
5	1,301
6	1,109

The Basic Biological Model and Radiation Protection Standards

6. The first development of an appropriate maximum permissible concentration of radon in air is ascribed to Evans and Goodman.[3] This did not involve a specific biological model but rather went to the heart of the matter by relating the incidence of lung carcinoma in the Schneeberg and Joachimsthal miners to the observed radon concentration and applying a reduction factor by analogy with their observations on radium poisoning. Evans and Goodman recommended a permissible concentration of 10 pCi radon 222 per liter. Stewart and Simpson,[4] who have prepared an excellent review of the early considerations, believe that this value was adopted in 1941 by the then U.S. X-ray and Radium Protection Committee (now the NCRP) without further analysis. The British X-ray and Radium Protection Committee, in 1943, recommended the higher value of 100 pCi radon 222 per liter, which is not explained in the literature.

7. Failla,[5] in a report prepared in 1942, proposed a simple model of the bronchus 1.5 centimeters in diameter and 3 centimeters long containing the inhaled radioactive gas. Corrections were proposed for solubility of the gas in the fluid of the bronchial tube. The entire radiation dose was assumed to be absorbed in a cylindrical shell of thickness equal to the "effective range" in tissue. In line with a familiar device of the time, the effective range was taken to be one-half of the nominal full range in tissue. Failla's argument was generalized for all radioactive gases. Some discussions were held at the time on the role of the "active deposit" in inhaled radon gas. Mitchell [6] in 1945, using a model very similar to Failla's, included a contribution from radon decay products formed by the radioactive decay of radon in the bronchus.

8. The first clear identification of the short-lived decay products of radon as the principal source of radiation dose to the bronchus is attributable to Bale.[7] Work at the University of Rochester by Bale, Shapiro, and others has notably clarified understanding of the important parameters.[8-11] Chamberlain and Dyson [12] recognized that polonium 218, formed by the radioactive decay of radon 222 gas, initially exists as a highly mobile free ion or atom. In relatively dust-free air it may persist in that form for about 50 seconds. Free atoms impinge on the walls of the trachea and are trapped with virtually 100 percent efficiency. The authors demonstrated this deposition in glass tubes using controlled flow rates, and later in a simulated trachea and main bronchus. The observations were successfully related to diffusion theory by the method of Gormley and Kennedy.[13]

9. With the dust loading in actual mine atmospheres the mean life of the free atoms may be as short as 10 seconds and rarely as long as 50 seconds. A mean life of 30 seconds is often assumed for calculation purposes. Chamberlain and Dyson calculated representative average doses in a cylindrical shell of tissue 45-microns thick at different breathing rates, and for an atmosphere containing 1,000 pCi radon 222 (1.72×10^7 atoms), 1,000 unattached polonium 218 atoms, and 100 unattached lead 214 atoms per liter of air. Typical results are shown in table 3. These calculations assume that only the alpha particle from polonium 218 is effective, other decay products being removed from the site of initial deposition along the mucus escalator. Also, no correction was made for deposition of polonium 218 free atoms in the nasal passages, although this was estimated at about 30 percent.

TABLE 3.—*Average dose rates to the epithelium of the trachea and main bronchi*

Minute volume (liters)	Lower trachea (mrads/hr)	Main bronchi (mrads/hr)
10	17	23
20	30	21
40	67	46

10. The authors noted that if the later decay products were not removed by ciliary action the dose to the epithelium would be doubled. Also, the decay products removed from the initial site of deposition by ciliary action may be replaced by decay products brought up from material initially deposited in more peripheral portions of the bronchial tree. Later models (see par. 17 to 23) have concentrated on these factors.

11. As a result of the conclusions reached in 1953 at a tripartite conference on radiation protection,[14] Morgan [15] revised some earlier calculations to include an RBE of 10 for alpha irradiation and to convert from "rep" to "rad," the new term for expressing absorbed dose in tissue. Using the concepts of Bale, he also assumed that:

1. Polonium 218 in the relevant atmosphere is in radioactive equilibrium with the radon 222, while the longer-lived lead 214 and bismuth 214 are at 50 percent of equilibrium. All the radon daughter products are inferentially attached to dust particles.

2. Twelve percent of the dust particles are fixed in the bronchial passages of the lung and remain there through the decay of polonium 214.

3. The volume of air in the bronchial passages was taken as 100 cc, and the mass of "uniformly" irradiated tissue was taken as 20 grams.

12. From these assumptions, Morgan concluded that either an atmosphere of 8.8 pCi of radon 222 per liter of air with the stated burden of associated radon daughters, or 3,500 pCi of radon per liter of air with no burden of daughters, would give a dose of 0.3 rem per 168-hour week to bronchial tissue.

13. According to Stewart and Simpson, the maximum permissible concentration of radon 222 presented in the International Commission on Radiological Protection (ICRP) report of Committee II on Permissible Dose for Internal Irradiation [16] is a compromise interpolating between the calculations of Morgan and those of Chamberlain and Dyson. The ICRP in its latest publication on this subject [17] has not changed its recommendations; however, a subcommittee is now reviewing the recommendations. The ICRP formula is given as:

$$MPC_a = \frac{3{,}000}{(1+1{,}000f)} \text{ pCi } ^{222}\text{Rn per liter of air}$$

where f is the fraction of the equilibrium amount of polonium 218 ions which are unattached to nuclei. Morgan's case for radon 222 alone is then equated with f = zero, leading to a value of 3,000 pCi radon 222 per liter of air instead of the 3,500 pCi radon 222 per liter of air originally calculated by Morgan. Chamberlain and Dyson's case corresponds to $f = 0.1$ leading to a value of 30 pCi radon 222 per liter of air.

14. A more recent report by a task group on lung dynamics for ICRP Committee II contains a valuable compilation of data appropriate for lung deposition calculation.[18] The latter report presents arguments both for and against the importance of free atoms (or ions), and concludes that "neither the concentrations occurring nor their deposition tendencies has been established."

15. The two models described next attempt to improve on the Chamberlain and Dyson model and attribute the principal hazard to the inhalation of radon daughters attached to respirable dust.

16. Altshuler, Nelson, and Kuschner [19] chose a reference atmosphere of 100 pCi radon 222 per liter having 200 pCi of total daughter products per liter of air ($^2/_3$ the equilibrium value). Sixty percent of the daughter products were distributed between free ions (about 150 per liter of air) and those attached to particles of less than 0.1-micron (μ) diameter. Forty percent of the daughter products were considered to be attached to airborne particles greater than 0.1μ in diameter. These larger particles were further subdivided into five groups of aerodynamic size 0.2, 0.6, 2, 6, and 20μ.

17. Although these subdivisions are arbitrary, they appear to be reasonable. The total daughter activity of 200 pCi per liter is compatible with the measurements of Tsivoglou, Ayer, and Holaday.[20] The 10-percent attachment to particles greater than 0.1μ appears to be compatible with measurements in mines of the Colorado plateau made by the AEC Health and Safety Laboratory,[21] if the interpretation of the cascade impactor measurements is accepted. The number (150) of free ions per liter of air is said to have come from

the work of Chamberlain and Dyson.[12] The number of particles with diameters less than 0.1μ was apparently obtained from the difference.

18. The deposition of free ions and nuclei in the lungs was calculated by the general methods of Shapiro and of Chamberlain and Dyson. Particle deposition was deduced by the method of Landahl.[22] Separate calculations were made for mouth breathing and nose breathing. These derivations of the regional deposition of radon daughters in the lung appear to provide a better estimate of the initial deposition than those used in previous models.

19. A further improvement is the attempt to calculate regional distribution of the radon daughter product disintegrations. This is based on calculations of mucus flow throughout the bronchial tree. The end product is an estimate of the number of disintegrations of polonium 218 and polonium 214 separately occurring in the respiratory regions: (1) trachea, (2) main bronchi, (3) lobar or secondary, (4) segmental or tertiary, (5) subsegmental or quaternary, and (6) terminal bronchi or bronchioles. The average regional dose is computed separately over the depth of penetration of each alpha particle. It is further assumed that the tissue dose from polonium 218 alpha particles falls linearly to zero at 47μ, while that from polonium 214 alpha particles falls linearly to zero at 71μ, each being normalized to its own average dose at midrange. Combined depth dose curves are thus obtained from which the effective dose at any prescribed depth may be derived.

20. Altshuler *et al.*,[19] visualized the mucus layer as being 7μ thick, with all the active particles resting on the free surface. This layer is swept upward by the cilia which are bathed in a serous layer of the same thickness. Below this lies the bronchial epithelium containing goblet cells, ciliated cells, and basal cells supported on the basement membrane. The relevant biological target was considered to be the nuclei of the basal cells, some 7μ above the basement membrane. The prescribed depth is obtained by measuring the bronchial epithelium thickness and adding 7μ (*i.e.*, 7μ mucus+7μ serous layer−7μ above the basement membrane). See figure 1.

21. The thickness of the bronchial epithelium is highly variable. Typical values quoted by Altshuler *et al.* (table 4), came from one subject and included a correction factor of 1.30 for tissue shrinkage. Engel [23] shows sections that are generally compatible with the figures in table 4, although they appear to show even wider variations. The calculated dose is very sensitive to the estimated thickness of the mucus layer and epithelium thickness; small changes in the estimated thickness cause large changes in the calculated dose.

TABLE 4.—*Thickness of bronchial epithelium in different parts of the lungs* [19]

Category	Main (μ)	Lobar (μ)	Segmental (μ)
Exceptionally thin	52	42	29
Median thickness	89	63	56

For example, the prescribed depth for the exceptionally thin portions of the segmental bronchi is taken as 29μ+7μ=36μ, which would give a calculated dose of 24 rads per year for the assumed burden of radon daughters. At the other extreme, the prescribed depth would be 89μ+7μ leading to a "zero" dose in the region of median thickness in the main bronchus since the maximum range for the polonium 214 alpha particle is only 71μ (see table 1).

22. A model which is conceptually similar to that proposed by Altshuler *et al.* was proposed at the same symposium by Jacobi.[2] However, there are important differences in detail between the two. These are:

1. Jacobi used a continuous distribution of particle size in aerosols of ordinary air rather than the multicompartment model of Altshuler *et al.*, leading to different values for the distribution of radon daughters among different particle sizes. For example, Jacobi reports 25 percent of polonium 218 as free ions instead of the 9 percent used by Altshuler. Similarly, Jacobi considers 52 percent of the radon daughters are attached to nuclei of 0.1μ diameter or less, versus Altshuler's value of 60 percent.

2. Jacobi assumes that all polonium 218 atoms remain on the surface of the mucus layer, but that the terminal polonium 214 atoms are distributed in a linear gradient from a maximum at the mucus surface to zero at the boundary of the bronchial epithelium.

3. Jacobi uses values for the thickness of the mucus layer which are higher than those used by Altshuler: (1) in the trachea and main bronchi, 80 microns; (2) in the lower bronchi, 20 microns; and (3) in the bronchioli, 5 microns. If Jacobi's values for the thickness of the mucus layer are correct, the alpha particles from polonium 218 cannot reach the underlying tissues in the main bronchi. The Chamberlain and Dyson model, by contrast, attributes practically all of the dose in this region of the lung to the deposition of polonium 218 atoms in the form of free ions.

4. Jacobi accepts the effective biological target as the basal cell layer in apparent agreement with Altshuler *et al.* He appears to describe the histological location of this layer as 10μ below the interface of the goblet-ciliated cells and the mucus layer (depth c—fig. 1).

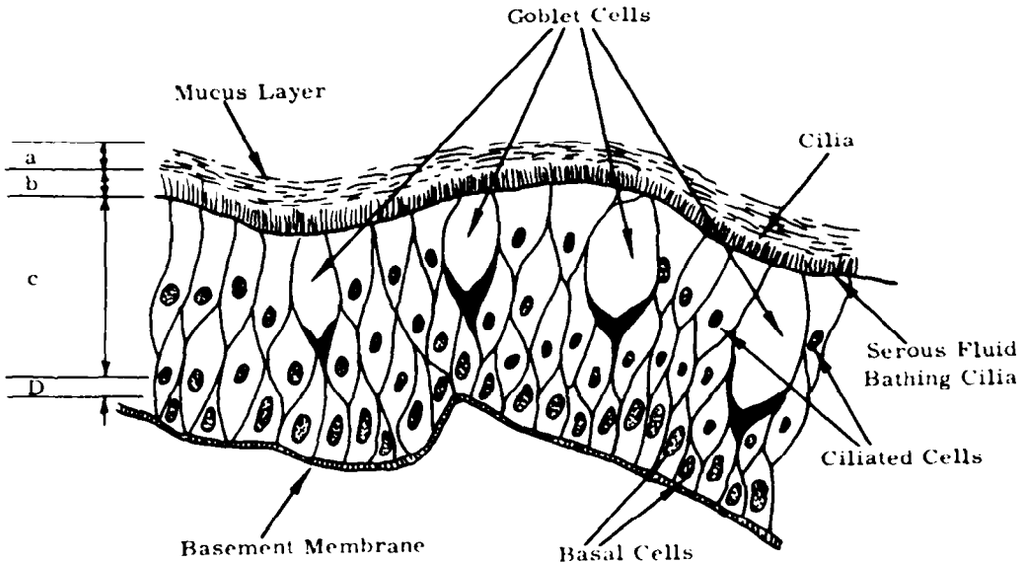
5. Jacobi used a range of 55μ for the polonium 218 alpha particles instead of the 47μ used by Altshuler.

23. In addition to the principal differences described in the preceding paragraph, the authors of the two models compute their results for different anatomical subdivisions of the lung so that a direct comparison of the original calculations cannot be made. However, if one makes some reasonable assumptions to normalize both models to the same anatomical subdivisions, at least an approximate value of the dose in rads can be estimated from each model. The values are shown in table 5 for mouth breathing with a minute volume of 15 liters per minute, a 40-hour week (2,000-hour work year) and a reference atmosphere of 100 pCi of radon 222 per liter plus the selected burden of daughter products (200 pCi of radon daughters per liter of air).

TABLE 5.—*Approximate annual dose (rads) in reference atmosphere*

Region	Altshuler *et al.*	Jacobi
Trachea and main bronchi	16	4
Secondary—quat. bronchi	20	22
Bronchioli	3 (or more)	0.8
Alveolar tissue	0.8	0.8

24. Results from these two models agree in the estimate of alveolar dose, because both involve a calculation which is independent of tissue structure in this region. They also appear to agree in the estimates of dose in the secondary and quaternary bronchi. This apparent agreement is fortuitous since it disappears if the same depth to the target tissue and the same values for the alpha particle ranges are used in both. They disagree substantially in estimates of the dose to bronchioli and the trachea and main bronchi.



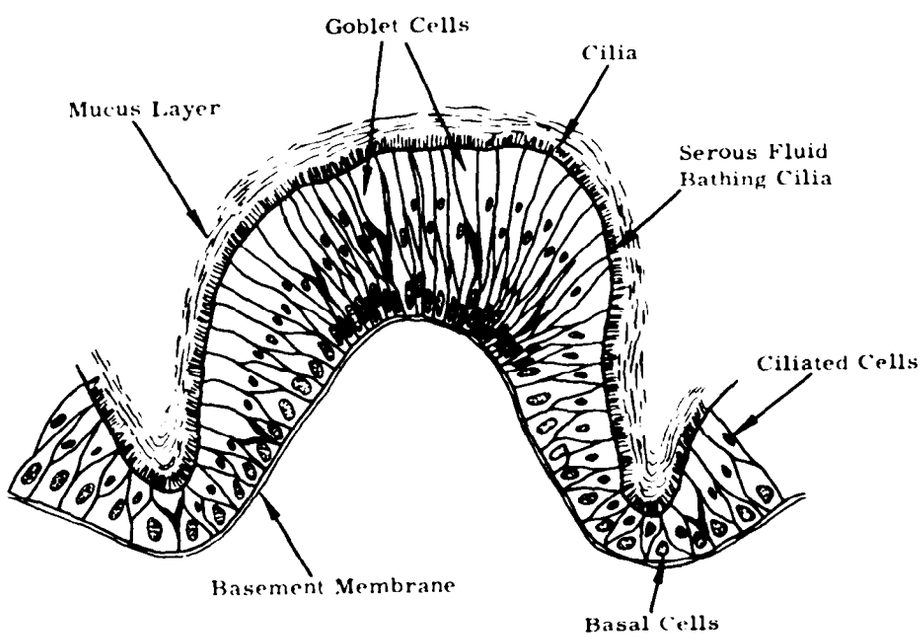


Figure 1. Location of the Biological Target

Upper: Stylized cross-section of bronchial epithelium: D is height of basal cell nuclei above basement membrane. Target depth = (a+b+c). Altshuler model: a, b, and D each 7 microns. Epithelium thickness (c+D) variable.

Lower: Typical folding: For segmental bronchi Altshuler quotes C = 22, 49, and ~77 microns for thin, median, and thick parts. Actual sections (*e.g.*, Engel) show greater variation.

25. Haque and Collinson have recently presented another model for calculating the dose to the respiratory system owing to radon and its daughter products.[24] Based on Weibel's lung model,[25] the dissipation of alpha particle energy from the daughter activity deposited on the walls of the respiratory system was calculated for anatomical locations above the alveoli and at various depths in tissue. The equilibrium daughter activity was calculated from the deposition rate and the mucus movement. Doses calculated by this method were found to be highest in the segmental bronchi and were generally higher than those calculated by Altshuler *et al.* The annual dose for an exposure equivalent to 0.1 WL, at a depth of 30 microns from the top of the mucus layer of the segmental bronchi, was calculated to be 13.8 rads.

26. These models, which attempt to correct for the defects assumed to exist in the Chamberlain and Dyson model, appear to represent the best that can be achieved at this time. The differences between them emphasize the significant uncertainties that exist in the needed radiological and physiological data and lead to the conclusion that a realistic relationship among the atmospheric burden of radon and radon daughters, the physical description of the relevant radiation dose to the lung, and radiation induced carcinogenesis cannot yet be defined.

Radiation Dose Estimates Associated With the "Working Level"

27. The chief merit of the WL (described in par. 2.6 of the report) approach is its attention to the predominant position of the alpha emitters in the decay product chain. A simple field method for estimating the concentration of decay products in terms of total alpha particle emission has been developed and serves as a distinct advantage in the use of the WL concept. The measurements are not unduly sensitive to the actual ratio of polonium 218, lead 214, and bismuth 214 in the atmosphere. A possible disadvantage in relating such measurements to radiation hazards is the necessary assumption that the hazard is adequately defined by the total alpha emission alone, and that the relevant dose is not sensitive to the distribution of the daughters between free ions, nuclei, and other various particle sizes.

28. The short-lived decay products through polonium 214 are not in radioactive equilibrium with the radon in actual mine atmospheres. Generally the first product, polonium 218, is close to radioactive equilibrium. The intervening nuclides are typically in the range of 20 to 80 percent of the equilibrium value, the lower values being associated with higher ventilation rates (see par. 2.4 of the report). Using the basic model of Morgan (par. 11) it was estimated that the average lung dose from inhalation of the short-lived decay products would be about 20 times greater than that from the radon alone.[26]

29. The reference atmosphere of Altshuler *et al.* contained 200 pCi of radon daughters per liter of air, or ostensibly a radon daughter concentration of $\frac{2}{3}$ (67 percent) of a WL. The potential alpha energy calculated from the stated composition of Altshuler's reference atmosphere (*i.e.*, 91 pCi ^{218}Po, 62 pCi ^{214}Pb, 44 pCi ^{214}Bi) is 7.4×10^4 MeV or 57 percent of a WL as defined.

30. Using Altshuler's model, calculations of the dose to different segments of the lung for nose breathing and mouth breathing, each at a rate of 15 liters per minute, give results that range between 55 percent and 65 percent of the dose that would be associated with a radon daughter concentration of 1 WL. It is considered accurate enough to conclude that Altshuler's reference atmosphere results in 60 percent of a "Working Level dose" to the bronchi, and has a value between 10 and 30 rads a year. In view of the ambiguities of conversion, Altshuler's reference atmosphere will be considered, with important reserva-

tions, to produce 20 rads in a normal working year (2,000 hours at a breathing rate of 15 liters per minute) to the bronchial epithelium. Thus the "WLM" corresponds to $\frac{20}{12} \times \frac{100}{60}$ or 2.8 rads, and exposure for 1 year at a radon daughter value of one WL would give 33 rads. The mean organ dose would be lower. In the absence of an appropriate factor for the RBE, the calculated dose cannot be converted to rem.

Discussion and Evaluation

31. Although the models discussed in this appendix appear to be more realistic than merely averaging over the alpha particle range, there are still serious uncertainties in the various parameters that must be considered. These include:

1. Distribution of radioactive material in or on the mucus layer. Some authors [27] consider the mucus sheet having as many as three layers in laminar flow with no mixing between them, each layer arising from different regions of the lung. However, the movement of the viscous layer by ciliar brushing implies some disturbance of laminar flow. In addition, the detection of cancerous or precancerous cells by sputum tests also implies quite sizable intrusions into the mucous.

2. Uncertainties remain concerning the actual range of alpha particles in tissue, the role of the delta rays, and the thickness of the mucus and bronchial epithelium.

3. Engel [23] states that the mucous membrane of the bronchi is normally arranged in folds, which in some instances almost close off separate channels. Although many models consider a uniform deposition on the surface of a smooth tube, it is reasonable to suppose that the true deposition is far from uniform. If so, it is reasonable to suppose that the most deposition will occur at the crests of the folds where the epithelium is thickest.

4. Observations on mucus flow rate are necessarily practical averages. If the flow rate differs between the crests and the troughs, the actual distribution of regional disintegrations could differ from the computed values in a systematic way related to the local epithelium geometry.

5. The thickness of the mucus sheet is probably more variable than provided in most models although this weakness is usually recognized. Mucus secretion is normally considered to increase as the result of irritation which is not necessarily confined to radiation and may increase with time in an occupation such as mining. If the mucus sheet is thick enough the alpha particles, particularly those from polonium 218, cannot penetrate to the depth of the assumed biological target. This implies that the physical dose to lung tissue following inhalation of a given quantity of radon and radon daughters may change progressively with time in a particular individual.

6. The choice of basal cells as the critical biological target is plausible, but injury to these cells has not been shown to be the source of cancer induction. Presumably, this choice would relate the radiation injury to a somatic mutation in the basal cell nucleus. At some later time, a breakdown of control occurs, leading to the development of malignancy. The long delay time, however, makes proof of such a relation very tenuous. If somatic mutation is a factor, the determination of the relative biological effectiveness of the alpha radiation is complicated by redundant ionization along any alpha track intersecting a chromosome and by the delta ray component.

7. If either mucus secreting cells or ciliated cells are injured to the point of impaired function, an initially small injury may be compounded into a progressively more disadvantageous cellular environment without injury of the chromosome. In this regard it

would be of great interest to learn what association there might be between the production of lung cancer and the ciliary "faults" (islands of squamous epithelium or metaplastic tissue devoid of cilia), which are reported to be found in the lungs of adults and smokers.[28]

8. Finally, there remains a question as to the effects of the doses from radioactive particles in the lymphatic vessels, lymphoid tissue, connective tissue, and alveolar tissue immediately adjacent to the broncheolar structures.

REFERENCES

[1] Lea, D. E. Action of Radiations on Living Cells (second edition). Cambridge Press, 1955.

[2] Jacobi, W. The Dose to the Human Respiratory Tract by Inhalation of Short-lived Radon 222 Decay Products. Proc. Hanford Symposium Inhaled Radioactive Particles and Gases. Health Physics, Vol. 10, p. 1163, 1964.

[3] Evans, R. D., and Goodman, C. Determination of the Thoron Content of Air and Its Bearing on Lung Cancer Hazards in Industry. J. Ind. Hyg. and Toxicol., Vol. 22, p. 89, 1940.

[4] Stewart, C. G., and Simpson, S. D. The Hazards of Inhaling Radon 222 and Its Short-lived Daughters: A Consideration of Proposed Maximum Concentrations in Air. IAEA Proc. Symposium Radiological Health and Safety in Mining and Milling Nuclear Materials, Vienna, Vol. I, p. 333, 1964.

[5] Failla, G. Tolerance Concentration of Radioactive Gases in Air. AECD-2362, Nov. 1942.

[6] Mitchell, J. S. Memorandum on Some Aspects of the Biological Action of Radiations, Special Reference to Tolerance Problems. Montreal Lab. Report HI-17, 1945.

[7] Bale, W. F. Hazards Associated With Radon and Thoron. Unpublished memo., 1951.

[8] Shapiro, J., and Bale, W. F. A Partial Evaluation of the Hazard from Radon and Its Degradation Products. Univ. Rochester Atomic Energy Project Report, UR-242, p. 6, 1953.

[9] Shapiro, J. An Evaluation of the Pulmonary Radiation Dosage from Radon and Its Daughter Products. Univ. Rochester Atomic Energy Project, UR-298, 1954.

[10] Bale, W. F., and Shapiro, J. V. Radiation Dosage to Lungs from Radon and Its Daughter Products. Proc. UN International Conf. on Peaceful Uses of Atomic Energy, Vol. 13, p. 233, 1955.

[11] Shapiro, J. Radiation Dosage From Breathing Radon and Its Daughter Products. AMA Arch. Ind. Health, Vol. 14, p. 169, 1956.

[12] Chamberlain, A. C., and Dyson, E. D. The Dose to the Trachea and Bronchi From the Decay Products of Radon and Thoron. Brit. J. Radiol., Vol. 29, p. 317, 1956.

[13] Gormley, P. G., and Kennedy, M. Diffusion From a Stream Flowing Through a Cylindrical Tube. Proc. Royal Irish Acad., Vol. 52, p. 163, 1948.

[14] Triparite Conf. on Permissible Doses, March-April 1953. Arden House, Harriman, N.Y.

[15] Morgan, K. Z. Maximum Permissible Concentration of Radon in the Air. Unpub. memo., October 27, 1954.

[16] ICRP Pub. 2: Report of Committee II on Permissible Dose for Internal Radiation. Pergamon Press, 1959.

[17] ICRP Pub. 6 (as amended 1959 and revised 1962). Pergamon Press, 1964.

[18] ICRP Task Group on Lung Dynamics: Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract. Health Physics Vol. 12, p. 173, 1966.

[19] Altshuler, B., Nelson, N., and Kuschner, M. Estimation of the Lung Tissue Dose From Inhalation of Radon and Daughters. Proc. Hanford Symposium Inhaled Radioactive Particles and Gases. Health Physics, Vol. 10, p. 1137, 1964.

[20] Tsivoglou, E. C., Ayer, H. E., and Holaday, D. A. Occurrence of Nonequilibrium Atmospheric Mixtures of Radon and Its Daughters. Nucleonics, Vol. 11, p. 40, 1953.

[21] HASL-91: Experimental Environmental Study of AEC Leased Uranium Mines. Health and Safety Laboratory, USAEC, New York, 1960.

[22] Landahl, H. D. Particle Removal by the Respiratory System—Note on the Removal of Airborne Particulates by the Human Respiratory Tract with Particular Reference to the Role of Diffusion. Bull. Math. Biophys., Vol. 25, p. 29, 1963.

[23] Engel, S. Lung Structure. C. C. Thomas, Springfield, Ill., 1962.

[24] Haque, A. K. M. M., and Collinson, A. J. L. Radiation Dose to the Respiratory System Due to Radon and Its Daughter Products. Health Physics, Vol. 13, p. 431, May 1967.

[25] Weibel, E. R., Morphometry of the Human Lung. Academic Press, Inc., New York, 1963.

[26] USPHS Pub. 494 (1957). Control of Radon and Daughters in Uranium Mines and Calculations on Biologic Effects.

[27] Gross, P. The Processes Involved in the Biologic Aspects of Pulmonary Deposition, Clearance, and Retention of Insoluble Aerosols. Proc. Hanford Symposium Inhaled Radioactive Particles and Gases. Health Physics, Vol. 10, p. 995, 1964.

[28] Hilding, A. C. On Cigarette Smoking, Bronchial Carcinoma and Ciliary Action. III. Accumulation of Cigarette Tar Upon Artificially Produced Deciliated Islands in Respiratory Epithelium. Ann. Otol., Vol. 65, p. 116, 1956.

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