III. BASIS FOR THE RECOMMENDED STANDARD

A. Assessment of Effects

1. Human Studies

a. Association of Radon Exposure with Lung Cancer Mortality

Appendix I contains the report prepared by NIOSH and submitted to MSHA on May 31, 1985, entitled Evaluation of Epidemiologic Studies Examining the Lung Cancer Mortality of Underground Miners. Several of the epidemiologic studies evaluated in that report demonstrate an association between exposure to radon progeny and lung cancer mortality in underground uranium miners [Lundin et al. 1971; Sevc et al. 1976; Kunz et al. 1978; Waxweiler et al. 1981; Placek et al. 1983; Samet et al. 1984; Muller et al. 1985; Tirmarche et al. 1985]. The relationship between exposure to radon progeny and lung cancer mortality has also been observed in workers in underground metal mines [Wagoner et al. 1963], iron ore mines [Boyd et al. 1970; Jorgensen 1973 (updated in 1984); Damber and Larsson 1982; Edling and Axelson 1983; Radford and Renard 1984], tin mines [Fox et al. 1981; Jingyuan et al. 1981; Wang et al. 1984], fluorspar mines [Morrison et al. 1985], gold mines [Muller et al. 1985], and zinc/lead mines [Axelson and Sundell 1978]. In addition, some of these studies demonstrate a direct exposure-response relationship between lifetime cumulative exposure to radon progeny and lung cancer mortality [Lundin et al. 1971; Sevc et al. 1976; Kunz et al. 1978; Morrison et al. 1985; Muller et al. 1985].

Statistically significant standardized mortality ratios (SMRs)* above 400% were observed in three studies in which workers accumulated mean lifetime exposures above 100 WLM [Sevc et al. 1976; Kunz et al. 1978; Waxweiler et al. 1981; Morrison et al. 1985]. Statistically significant SMRs between 140% and 390% were observed in two other studies in which workers accumulated mean lifetime exposures below 100 WLM [Radford and Renard 1984; Muller et al. 1985] and in preliminary findings of a third study in which workers accumulated estimated mean lifetime exposures below 100 WLM [Tirmarche et al. 1985].

b. Synergistic Effects of Other Substances

Although the literature consistently demonstrates an association between lung cancer incidence and exposure to radon progeny, it is possible that some of the miners studied were exposed to other substances as well. These substances may have acted synergistically

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*The standardized mortality ratio (SMR) is the ratio of the mortality rates of two groups being compared. This ratio is expressed as a percentage and is usually adjusted for age or time differences between the two groups.

with radon progeny to potentiate the effects of the radon exposure. [Doull et al. 1980]. Substances with synergistic potential include arsenic [NIOSH 1975a; Wang et al. 1984; Sevc et al. 1984], hexavalent chromium, nickel, cobalt [NIOSH 1975b; NIOSH 1977; Sevc et al. 1984]; serpentine [Radford and Renard 1984]; iron ore dust [Boyd et al. 1970; Jorgensen 1973, 1984; Damber and Larsson 1982; Edling and Axelson 1983; Pham et al. 1983; Radford and Renard 1984], and diesel exhaust [Wagoner et al. 1963; Boyd et al. 1970; Waxweiler et al. 1981; Fox et al. 1981; Damber and Larsson 1982; Edling and Axelson 1983; Jorgensen 1984; Sevc et al. 1984; Muller et al. 1985; Morrison et al. 1985; Tirmarche et al. 1985].

Risk analyses were performed on data from epidemiologic studies of U.S. uranium miners [Whittemore and McMillan 1983; Appendix II] and Swedish iron ore mine workers [Radford and Renard 1984]. These analyses indicate that the risk of mortality from lung cancer among miners who are exposed to radon progeny is greater among those who smoke cigarettes than those who do not smoke.

c. Relation of Lung Cancer Risk to Cumulative Radon Exposure

Since completion of the NIOSH report (Appendix I), Howe et al. [1986] published a study of surface and underground mine workers who had worked in Canada in the Eldorado Lodge Uranium Mine between 1948 and 1980. That cohort included 8,487 workers. Table III-1 describes their characteristics with respect to age, lifetime cumulative exposure, duration of employment, and type of mine work (surface or underground).

Table III-1.--Characteristics of mine workers employed at the Eldorado Lodge Uranium Mine during the period 1948-80

Type of mine worker	Mean age at 1st employment	Mean lifetime cumulative exposure (WLM)	Mean duration of employment (months)	Miners in cohort	
				No.	% total
Surface only*	27.7	2.8	22.2	4,077	48
Surface and underground	---	28.9	43.9	572	7
Underground	28.8	16.6	15.0	3,838	45

*Never employed underground.

Sixty-five deaths from lung cancer were observed for the total cohort, but only 34.24 lung cancer deaths were expected on the basis of age-specific and calendar-year-specific Canadian national mortality rates (SMR = 190; $p<0.05$). Workers with lifetime cumulative exposures greater than 5 WLM experienced 46 deaths from lung cancer as opposed to 15.88 expected deaths (SMR = 290; $p<0.0001$).

In this study, Howe et al. [1986] estimated the annual average concentrations of radon progeny (WL) for the period 1954 through 1980 from measurements of radon gas and radon progeny. Samples of radon gas were collected from all areas of the mine from 1954 through 1967 to assess the effectiveness of dilution ventilation. Samples of radon progeny were collected several times per month per work site since 1967. These estimated values were used to assign annual exposure values (WLM) to the workers according to the number of hours worked underground. Howe et al. then subdivided the cohort into seven categories of WLM exposure: 0 to 4, 5 to 24, 25 to 49, 50 to 99, 100 to 149, 150 to 249, and greater than or equal to 250. Based on these stratified categories the risk of death from lung cancer increased linearly with increasing exposure. For the exposure categories of 5 to 24, 25 to 49, and 50 to 99 WLM, the relative risk was elevated, but the difference from the expected risk for an unexposed population was not statistically significant. For all exposure categories above 100 WLM, the relative risk was significantly elevated ($p<0.05$); note, however, that the first 10 years of followup were excluded from this calculation. For the total cohort, the relative risk coefficient was 3.28% per WLM, and the absolute risk coefficient was 20.8 per 10^6 person-years per WLM (any excess mortality within the 10 years following initial exposure was excluded from this calculation).

Two additional epidemiologic studies that were not included in the 1985 NIOSH report (Appendix I) have also been reported. Pham et al. [1983] studied 1,173 iron mine workers in France who were aged 35 to 55 and who had normal chest X-rays at the beginning of the study period in 1975. Thirteen mine workers who had worked underground for a mean of 25.2 years died of lung cancer between 1975 and 1980; only 3.7 deaths were expected from an age-standardized comparison with French males for this same period (SMR = 351; $p<0.05$). Although exposure records were not available, the authors estimated that some workers may have received lifetime cumulative exposures to radon progeny in the range of 100 to 150 WLM.

Solli et al. [1985] observed 318 niobium mine workers in Norway from 1953 through 1981; 77 of these miners were underground workers. This cohort experienced a total of 12 lung cancer deaths, though only 2.96 deaths were expected on the basis of age-specific rates for Norwegian males (SMR = 405; $p<0.001$). The underground workers experienced 9 lung cancer deaths whereas only 0.81 were expected on the basis of age-specific rates for Norwegian males (SMR = 1,111; $p<0.001$). From estimates of total exposure to alpha radiation (based on limited measurements of radon and thoron progeny taken in

1959), the authors determined that the risk of lung cancer increased significantly ($p<0.05$) with increasing alpha radiation for the exposure categories of 1 to 19, 20 to 79, 80 to 119, and greater than or equal to 120 WLM. The excess absolute risk for those exposed workers was reported to be 50 per 10^6 person-years per WLM.

The epidemiologic studies of lung cancer mortality in mine workers exposed to radon progeny (including those studies discussed in Appendix I) are summarized in Tables III-2 and III-3.

2. Animal Studies

a. Effects of Exposure to Radon Progeny

Chameaud et al. [1984a] studied the effects of exposure to radon progeny in specific pathogen-free (SPF) Sprague-Dawley rats. A total of 1,800 rats were exposed to radon progeny for 1 to 3 hr per day for 14 to 82 days, yielding an accumulated exposure of 20 to 50 WLM. An additional 600 rats were unexposed. The lung cancer incidence in rats was reported to be directly proportional to their lifetime cumulative exposure to radon progeny. The authors concluded that the amount of radiation needed to double the natural incidence of lung cancer in these rats was 20 WLM. Reduced life spans were not observed for rats in any of the exposure groups.

b. Relation of Lung Cancer Incidence to Radon Progeny Exposure

Chameaud et al. [1981; 1984a] determined that the lifetime risk coefficient (uncorrected for life span shortening) for the induction of lung cancers in rats was approximately 140 to 850 x 10^{-6} per WLM for exposures ranging from 20 to 4,500 WLM (Table III-4). This is consistent with the lifetime risk coefficient for lung cancer in humans (150 to 450 x 10^{-6} per WLM) estimated by the International Commission on Radiological Protection (ICRP) [ICRP 1981]. As shown in Table III-4, lung cancer incidence in rats increased as cumulative exposure to radon progeny increased. In contrast, the lifetime risk of lung cancer per unit of exposure (WLM) decreased with increasing exposure. These findings agree with those of the NIOSH risk assessment (Appendix II), in which the lifetime cumulative risk of lung cancer per unit of exposure decreased as cumulative exposure increased in underground uranium mine workers.

c. Synergistic Effects of Cigarette Smoke

Chameaud et al. [1980, 1982] studied the ability of radon progeny to initiate lung cancer in groups of 50 SPF Sprague-Dawley rats that were subsequently exposed to cigarette smoke. The chamber concentrations of alpha radiation were 0, 300, and 3,000 WL; these concentrations yielded cumulative dose levels of 0, 100, 500, and 4,000 WLM, respectively, over a 2-month period for those groups of animals. Treatment groups were exposed to a total of 352 hr of cigarette smoke (9 cigarettes/500 L of air for 10 to 15 min per day, 4 days per week for 1 year. Exposure to radon progeny alone

Table III-2.--Summary of principal studies of lung cancer mortality in underground mine workers exposed to radon progeny*

Type of mine (location)	Reference	Mean lifetime cumulative exposure (WLM)	Person-years	Lung cancer deaths		
				Observed	Expected	SMR†
Uranium (U.S.)	Waxweiler et al. [1981] Lundin et al. [1971]	821§ (median = 430)	62,556	185	38.4	482
Uranium (Czechoslovakia)#	Placek et al. [1983]# Kunz et al. [1978]#	289**	56,955	211	42.7	496
Uranium (Ontario, Canada)	Muller et al. [1985]	40-90††	202,795††	82††	56.9††	144††
Iron (Sweden)	Radford & Renard [1984]	81.4§§	24,083§§	50	12.8§§	390§§
Fluorspar (Newfoundland)	Morrison et al. [1985]	---##	37,730##	104	24.38##	427##
Uranium (Saskatchewan, Canada)	Howe et al. [1986]	16.6***	118,341†††	65†††	34.24†††	190†††

*Comparisons between these studies, especially for purposes of risk assessment, should be made with caution because of differences in the calculations of person-years, expected deaths, and SMR values in the various studies.
†$p<0.05$ except in Muller et al. [1985], Radford & Renard [1984], and Morrison et al. [1985]; because p-values were not provided in these three studies, they were estimated from the observed lung cancer deaths and the Poisson frequency distribution.
§Lifetime cumulative exposures ranged from less than 60 to greater than 3,720 WLM.
#Studies are of uranium mine workers who started work underground between 1948 and 1952.
**Lifetime cumulative exposures ranged from less than 50 to approximately 1,000 WLM.
††Values are for uranium mine workers with no previous gold mining experience; exposures were lagged up to 10 years; lifetime cumulative exposures ranged from 0.1 to greater than 340 WLM.
§§Person-years for the first 10 years of mining experience were excluded; expected deaths were adjusted for smoking status; exposures were lagged 5 years; lifetime cumulative exposures ranged from 0 to greater than 200 WLM.
##Person-years for surface and underground mine workers were included; person-years for the first 10 years of mining experience were excluded; radon progeny exposure levels were recently reestimated [Corkill and Dory 1984]; lifetime cumulative exposures ranged from 0 to greater than 2,040 WLM.
***Value was based on underground workers (surface workers received a mean exposure of 2.8 WLM); lifetime cumulative exposures ranged from 0 to greater than 250 WLM.
†††Values were based on surface and underground workers.

Table III-3.--Summary of additional studies of lung cancer mortality in underground mine workers exposed to radon progeny*

Type of mine (location)	Reference	Estimated concentration or exposure	Comparison groups	Lung cancer deaths Observed	Lung cancer deaths Expected	Rate ratio† for lung cancer deaths
Iron (Grangesberg, Sweden)	Edling and Axelson [1983]	0.3 to 1.0 WL	Underground miners aged 50 and above vs. nonexposed individuals in the parish aged 50 and above	33	2.87§	11.50
Zinc-lead (Sweden)	Axelson and Sundell [1978]	1 WL	Underground miners vs. non-exposed individuals in the parish	21	1.28§	16.4
Iron (Kiruna, Sweden)	Jorgensen [1973, 1984]	0.5 WL	Underground miners vs. Swedish males	28	9	3.11
			Underground miners vs. Kiruna males	28	6.79	4.12
Iron (Kiruna and Gallivare, Sweden)	Damber and Larsson [1982]	0.095 to 2.025 WL	Underground miners vs. non-exposed individuals in the Kiruna and Gallivare parishes	20	2.74§	7.3
Metal (U.S.)	Wagoner et al. [1963]	0.05 to 0.40 WL	White male underground miners vs. white males from the same States	47	16.1	2.92
Uranium (U.S.)	Samet et al. [1984]	Lifetime exposure: 30 to 2,698 WLM; median exposure: 1,207 WLM (values are for 14 of 23 uranium miners)	Navajo males with uranium mining experience vs. Navajo males listed in the New Mexico tumor registry who died of cancer other than lung cancer	23	0	NA#
Tin (Cornwall, United Kingdom)	Fox et al. [1981]	1.2 to 3.4 WL	Underground miners vs. English and Welsh males	28	13.27	2.11

*These studies contain limitations in study design, radon progeny exposure records, smoking history information, followup, etc. Comparisons between these studies, especially for the purposes of risk assessment.
†$p<0.05$ (some p-values were estimated from the observed lung cancer deaths and the Poisson frequency distribution); rate ratios depend on lung cancer mortality in the comparison population and are sensitive to error in rates that are based on a small number of expected deaths.
§The expected number of deaths was estimated from the rate ratios provided by the authors.
#Not applicable. The 95% confidence limits of the rate ratios range from 14.4 to infinity.

Table III-4.--Radon progeny exposure and risk of lung cancer in specific, pathogen-free Sprague-Dawley rats*

Lifetime cumulative exposure (WLM)	Number of rats exposed	Number of rats with lung cancer	Percentage of rats with lung cancer	Lifetime lung cancer risk coefficient (10^{-6}/WLM)†
2§	600§	5§	0.83§	---
20-25	1,000	23	2.3	850
50	794	30	3.8	580
290	21	2	9.5	330
860	20	4	20.0	280
1,470	20	5	25.0	170
1,800	50	17	34.0	180
1,900	20	7	35.0	180
2,100	54	23	42.6	200
2,800	180	76	42.2	150
3,000	40	17	42.5	140
4,500	40	29	72.5	160

*Adapted from Chameaud et al. 1981, 1984a.
†Values were not corrected for life span shortening.
§Values are for rats in control group.

demonstrated a directly proportional dose-effect relationship for induction of cancer (500 and 4,000 WLM). However, when a similar period of radon exposure was followed by exposure to cigarette smoke, a dose-related, twofold to fourfold increase occurred in lung cancer incidence. The authors stated that the groups receiving high and medium doses of radon and cigarettes had cancers that were not only larger but were more invasive and metastatic compared with the groups exposed to radon alone. Conversely, neither the cigarette smoke alone nor the low-dose radon progeny exposure (100 WLM) alone induced lung cancer.

In a parallel lifetime study, Chameaud et al. [1981] related the sequence of exposure to radon progeny and cigarette smoke to the

incidence of lung tumors (cancer incidence was not specified) in groups of 50 SPF Sprague-Dawley rats. One group of rats was exposed to radon progeny only (a cumulative exposure of 4,000 WLM); a second group was exposed first to cigarette smoke and then to radon progeny (4,000 WLM); a third group was exposed to radon progeny (4,000 WLM) and then to cigarette smoke; and a fourth group was exposed to cigarette smoke only. Similar incidences of tumors were observed among the rats exposed to radon progeny only (10 tumors) and those exposed first to cigarette smoke and then to radon progeny (8 tumors). In contrast, when the exposure to radon progeny preceded the exposure to cigarette smoke, the effect was potentiated--that is, 32 rats developed tumors. As stated previously, none of the rats exposed to cigarette smoke only developed lung cancer. No statistical analyses were performed on the results of this study.

d. Significance of Animal Studies

Life span experiments in animals exposed to radon progeny alone have demonstrated that increasing exposures produce increasing incidences of lung cancer. This finding is similar to those of the epidemiologic studies cited in the preceding section (III, A, 1). Because epidemiologic data are available, these animal data contribute relatively little to the final assessment of risk in humans or to the determination of an REL for exposure to radon progeny. Thus this document discusses only selected animal studies of the carcinogenic potential of radon progeny. Other studies have examined the sequential or concomitant exposures of rats, dogs, and hamsters to substances other than radon progeny (e.g., uranium ore dust, thorium, and tobacco smoke). Several additional studies (critiqued but not described in this document) confirm the adverse health effects of radon progeny on exposed animals [Chameaud et al. 1974, 1984b; Filipy et al. 1977a, 1977b; Gaven et al. 1977; PNL 1978; Cross et al. 1981, 1982a, 1982b, 1983, 1984; Cross 1984]. These animal studies generally confirm the risk of lung cancer reported among workers exposed to radon progeny.

B. Risk Assessment

NIOSH studied the lung cancer risk of uranium miners by using data from a U.S. Public Health Service (USPHS) study [Lundin et al. 1971] of white male uranium mine workers from the Colorado Plateau area (Colorado, Arizona, New Mexico, and Utah). That NIOSH risk assessment is described in a report entitled Quantitative Risk Assessment of Lung Cancer in U.S. Uranium Miners, which is reproduced in Appendix II. Appendix I contains a detailed discussion of the USPHS data.

In the NIOSH risk assessment, data were analyzed for 3,346 workers who had been followed from 1950 through 1982. By 1982, 1,215 workers had died; 256 of these deaths (21.1%) were due to lung cancer. A generalized version of the Cox proportional hazards model was used to estimate the relative risk of death resulting from lung cancer over a 30-year working lifetime at several cumulative exposure values. (The 30-year working lifetime was

selected to maintain consistency with the working lifetime commonly described by MSHA.) Relative risk is defined as the ratio of lung cancer mortality in a selected exposed group to lung cancer mortality in a comparison group. The quantitative risk assessment model presented in Appendix II did not include the length of time since the end of the mining exposure, which is a significant predictor of relative risk. This term was subsequently added to the generalized Cox model, and new parameter estimates were computed. The estimates in Tables III-5 and III-6 are based on this model. The major difference between the two quantitative risk assessment models is that under the new model, the relative risk estimates increase more rapidly during exposure and decrease more rapidly after exposure.

The risk of death resulting from lung cancer increased with increasing lifetime cumulative exposure to radon progeny (Table III-5); this finding is consistent with Appendix II. This direct relationship has been observed in previous epidemiologic studies [Lundin et al. 1971; Sevc et al. 1976; Kunz et al. 1978; Morrison et al. 1985; Muller et al. 1985]. As shown in Table III-5, the relative risk of 1.57 at 30 WLM corresponds to an average exposure of 1 WLM per year for a working lifetime of 30 years.

Table III-5.--Relative risk estimates of lung cancer at age 60 by annual and lifetime cumulative exposures to radon progeny

Annual mining exposure above background (WLM/year)	Cumulative exposure* (WLM over a 30-year working lifetime)	Relative risk†	95% confidence limits
0.5	15	1.31	1.23 - 1.39
1.0	30	1.57	1.42 - 1.74
2.0	60	2.04	1.74 - 2.40
3.0	90	2.45	2.00 - 2.99
4.0	120	2.81	2.23 - 3.56

*Values are exclusive of background exposure.
†Estimates are based on a log-relative risk model fitted to age at initial exposure, time since cessation of exposure, and the natural logarithms of the following variables: cumulative mining and background exposure to radon progeny, cumulative cigarette smoking and background smoking, and rate of exposure to radon progeny.

Table III-6.--Estimated excess lung cancer deaths per 1,000 miners* resulting from 30 years of occupational exposure to radon progeny

Annual mining exposure above background (WLM/year)	Total mining exposure (WLM)	Estimated excess lung cancer deaths per 1,000 miners		
		Point estimate	Approximate 95% confidence limits†	
			Lower	Upper
4.0	120.0	42.0	25.0	71.0
3.0	90.0	32.0	19.0	54.0
2.0	60.0	22.0	13.0	36.0
1.0	30.0	10.0	7.0	17.0
0.5	15.0	4.9	3.4	7.6

*Estimates are based on a log-relative risk model fitted to age at initial exposure, time since cessation of exposure, and the natural logarithms of the following variables: cumulative mining and background exposure to radon progeny; cumulative cigarette smoking and background smoking; and rate of exposure to radon progeny.

†The approximate 95% confidence limits were calculated by applying the parameters from the quantitative risk assessment model together with their variances and covariances to the lung cancer mortality rates in the Colorado Plateau using an actuarial approach.

In addition to receiving workplace exposures to radon progeny, workers were assumed to have received average environmental exposures of 0.4 WLM/year. This is the value derived in the NIOSH risk assessment that led to the best fit of the model to the data. This assumed value is consistent with estimates of exposure to radon progeny for persons living near ore-bearing lands in the United States [NCRP 1975; Brookins 1986]. The average non-occupational exposure to radon progeny from natural geologic sources for persons in the United States is approximately 0.2 WLM/year [NCRP 1984b].

Relative risk modeling is common in the epidemiologic literature (especially in studies with lengthy followup) because the dramatic changes that occur in mortality rates with age and calendar year make absolute risk models extremely complicated. Most relative risk models assume that mortality rates in exposed populations are roughly proportional to the rates in unexposed populations at all ages and calendar periods. Because this assumption often approximates reality over a broad range of ages and calendar periods, the relative risk model can be expressed in a less complex form than absolute risk models (i.e., the relative risk model can be expressed without terms involving age and calendar year).

Excess lifetime risk estimates for lung cancer mortality have been generated (Table III-6) by applying the relative risk estimates in Table III-5 (see also Appendix II) to the lung cancer and all-causes mortality rates for

white males in the Colorado Plateau States. Excess risk is defined as the arithmetic difference between the risk of lung cancer mortality in a selected exposed group and the risk of lung cancer mortality in an unexposed comparison group.

The estimated excess lung cancer deaths (i.e., excess lifetime risk) in Table III-6 were computed by approximating the average of the exposure-determined relative risk function over 5-year age intervals spanning an entire lifetime. These average relative risks and the corresponding mortality rates for lung cancer and for all causes of death among white males in the Colorado Plateau were used to compute the probability of lung cancer mortality during a lifetime using the National Academy of Sciences actuarial method [NAS 1987].

It is important to understand the limitations of these risk estimates when examining the values in Table III-6. These limitations include the following:

- A relatively small portion of the cohort had the observed lower levels of cumulative occupational exposure, and the ability to generate precise point-risk estimates at the lower range of occupational exposure is not as strong. Only 7% of the workers in the cohort had lifetime cumulative exposures below 30 WLM, and only 7 of the 256 lung cancer deaths occurred among workers with lower cumulative exposures.

- The reliability of these excess lifetime risk estimates depends on (1) the accuracy of the original relative risk estimates and (2) the appropriateness of using lung cancer rates for the general white male population in the Colorado Plateau as an estimate of the background lung cancer rate (i.e., that which would occur in populations exposed only to background levels of radon progeny). This cohort contained no unexposed mine workers from which to estimate background lung cancer rates. Although certain limitations exist in using this type of rate [Monson 1980], background lung cancer mortality rates from the general U.S. white male population were used to estimate the background rates for the cohort.

- The background lung cancer rates were not corrected for cigarette smoking. However, the relative risk estimates used to calculate the lifetime risk estimates were adjusted for smoking. This implies that the lifetime risk estimates are only appropriate for a population with a pattern of smoking similar to that of the white male population of the Colorado Plateau.

In developing its recommendations, NIOSH has attempted to compare the risk of occupational exposure to radon progeny with the risk of background exposure in homes (i.e., exposure accruing outside the mining environment). The estimated average background exposure to radon progeny is approximately 0.2 WLM per year for the general U.S. population; this estimate is higher in the vicinity of radiation-emitting ore bodies [NCRP 1975]. The NIOSH risk assessment (Appendix II) indicated that 0.4 WLM per year was the background exposure value that led to the best fit of the model to the data. Data are

not available on actual background exposures (i.e., exposures accruing outside the mining environment) of underground miners in the Colorado Plateau, but background exposures probably vary substantially. No risk assessment has been completed on the lung cancer risk associated with background exposures for the general U.S. population or for the population living in the Colorado Plateau. Until studies in homes are completed, it will be impossible to directly contrast risk estimates for occupational exposures with those for background exposures in homes.

Currently it is not possible to compare the risk of occupational exposure with the risk of background exposure in homes. Nonetheless, it is important to consider occupational risk in the context of the background lung cancer risk. On the basis of State vital statistics records, NIOSH estimates that the lifetime risk of lung cancer in the Colorado Plateau, uncorrected for smoking, is approximately 45 lung cancers per 1,000 white males. Unfortunately, accurate lung cancer rates are not available for nonsmokers in the Colorado Plateau.

Given this value for background lung cancer risk, another question that must be considered is to what level it would be reasonable to control occupational risk. In its benzene decision, the U.S. Supreme Court gave the following example as the basis for evaluating the occupational risk of chemically induced leukemia: An exposure associated with 1 excess death per million exposed persons might pose an acceptable risk, whereas an exposure associated with 1 excess death per 1,000 exposed persons would pose a significant risk that should be reduced. This example is useful, but it cannot be strictly applied in all cases because it was offered as an illustration and not a fixed rule. In the specific case of lung cancer risk associated with radon progeny exposure, the example cannot be strictly applied because the background risk of lung cancer is much greater than the risk of leukemia at background exposure levels and because cigarette smoking is known to create a confounding effect that greatly increases risk. An excess of 1 lung cancer death per 1,000 would probably not be detectable in a general population if data were subject to considerable uncertainty, since it would necessitate differentiating between 46 and 45 deaths per 1,000.

Another important consideration is the technical feasibility of a given exposure limit. As stated earlier, NIOSH has determined that a cumulative exposure limit of 1 WLM per year is achievable (though some argue that it is not feasible on the basis of economics or current technology). NIOSH has found no evidence that any cumulative exposure lower than 1 WLM per year is feasible. As shown in Table III-6, the occupational exposure limit required to reduce expected lifetime risk to 1 excess lung cancer death per 1,000 miners is approximately 0.1 WLM per year. This cumulative exposure would require an occupational exposure concentration that is less than the concentration associated with a cumulative background exposure of 0.4 WLM per year, assuming background expoure is acquired outside the occupational environment. An occupational exposure limit of 0.1 WLM per year would therefore require average mine concentrations lower than the estimated background exposure concentrations.

In view of the preceding factors, the level of uncertainty in available data, and the apparent unfeasibility of limiting cumulative exposures to

less than 1.0 WLM per year, it does not seem reasonable to recommend exposure limits that would yield only 1 excess lung cancer death per 1,000 miners. NIOSH has determined that an exposure limit of 1 WLM is feasible in some mines and that such an exposure limit would substantially reduce risks from those associated with the current MSHA standard. An REL of 1 WLM per year is therefore recommended to substantially reduce risk and to stimulate the implementation and development of engineering and mining techniques to reduce exposure. The enforcement of this recommendation combined with additional health and developmental research may facilitate future exposure reductions and thereby reduce lung cancer risk.

C. Technical Feasibility

In a report to the U.S. Bureau of Mines, Bloomster et al. [1984a] analyzed the technical feasibility of reducing the airborne concentrations of radon progeny in underground uranium mines. The study included data from 14 underground uranium mines operating during the study period (September 1981 to May 1984). The authors concluded that some mines could operate at an annual exposure standard of 2 WLM by using dilution ventilation alone if it was introduced early in the development of the mine and if no contamination of inlet air was present. An extensive engineering analysis of 2 of the 14 mines indicated that it might be feasible to meet an operating standard of 1 WLM by using dilution ventilation in combination with other control methods such as bulkheading, air filtration, and use of sealants. The authors expressed doubt about the technical feasibility of operating these uranium mines at a standard of 0.5 WLM. Appendix III provides descriptions of engineering control methods that may be useful in underground mines.

D. Recommendations

Several schemes exist for identifying and classifying a substance as a carcinogen. For example, the National Toxicology Program (NTP) [NTP 1984], the International Agency for Research on Cancer (IARC) [WHO 1979], and the Occupational Safety and Health Administration (OSHA) [29 CFR 1990, Identification, Classification, and Regulation of Potential Occupational Carcinogens (also known as "The OSHA Cancer Policy")] have all considered this problem. NIOSH considers the OSHA classification the most appropriate for use in identifying potential occupational carcinogens and supports the following definition:

> A "potential occupational carcinogen" is any substance, or combination or mixture of substances, which causes an increased incidence of benign and/or malignant neoplasms, or a substantial decrease in the latency period between exposure and onset of neoplasms in humans or in one or more experimental mammalian species as the result of any oral, respiratory, or dermal exposure, or any other exposure which results in the induction of tumors at a site other than the site of administration [29 CFR 1990.103].

This definition also includes any substance that mammals may potentially metabolize into one or more occupational carcinogens.

The epidemiologic data examined by NIOSH demonstrates that occupational exposure to radon progeny in underground mines has the potential for causing lung cancer in miners (see Appendix I) [Pham et al. 1983; Solli et al. 1985; Howe et al. 1986]. These human data are supported by a number of studies in which various animal species exposed to radon progeny also developed lung cancer [Chameaud et al. 1974, 1980, 1981, 1982, 1984a, 1984b; Filipy et al. 1977a, 1977b; Gaven et al. 1977; PNL 1978; Cross et al. 1981, 1982a, 1982b, 1983, 1984; Cross 1984]. Furthermore, the NIOSH risk assessment presented in Appendix II, which was based on the human studies, clearly demonstrates that a relationship exists between cumulative radon progeny exposure and the risk of developing lung cancer. The risk assessment shows that as cumulative exposure decreases, the risk of developing cancer decreases.

In arriving at an REL, NIOSH attempts to identify that exposure at which no worker will suffer material impairment of health or functional capacity. In the case of radon progeny, this task is difficult because the NIOSH risk assessment shows that even with an exposure of 0.5 WLM per year (15 WLM for a 30-year cumulative exposure) or below, the risk of developing lung cancer increases (see Table III-4 and Appendix II).

These results indicate that NIOSH should recommend an annual cumulative exposure limit well below 0.5 WLM, but NIOSH must also consider the technical feasibility of the REL. In previous NIOSH recommendations for the control of carcinogens, technical feasibility has often been interpreted as the ability to quantitate exposure; however, those recommendations were intended for use by nonmining industries where product substitution and engineering and process controls are generally more feasible.

Data from 1984 indicate that 94.4% of the workers in U.S. underground uranium mines accumulated annual radon progeny exposures of less than 2 WLM [AIF 1986]. Information obtained from the Bureau of Mines [Bloomster et al. 1984a] indicates that it is now technically feasible to achieve an annual radon progeny concentration of 1.0 WLM. NIOSH therefore recommends that cumulative exposure to radon progeny be limited to 1.0 WLM per year. This recommendation is intended to protect the health of America's underground miners, but it is tempered by the fact that currently it is not technically feasible to achieve annual exposures lower than 1.0 WLM using work practices and engineering controls.

To meet the NIOSH recommendation for an annual cumulative exposure of 1 WLM and to assure that mining is a viable year-long occupation, NIOSH believes that the daily average work shift concentration of radon progeny should not exceed 1/12 WL in any work area. Although adherence to the NIOSH REL will significantly reduce the risk of lung cancer in underground mine workers, it will not eliminate it.

No effective medical procedure currently exists to treat lung cancer caused by exposure to radon progeny. Furthermore, it has been demonstrated that exposure to both radon progeny and tobacco smoke result in a combined lung cancer risk that is greater than the risk posed by radon progeny or smoke alone. Thus it should be noted that the interaction between radon progeny and smoking is at a minimum additive, and more likely multiplicative. Cigarette smoking should therefore be emphasized as an even greater

detriment to mine workers exposed to radon progeny than it is to the general public. The implementation of smoking cessation programs should reduce the incidence of lung cancer in underground mine workers.

Because radon progeny are ubiquitous, exposure cannot be totally eliminated. For U.S. residents, the average annual nonoccupational exposure to radon progeny from natural geologic sources is approximately 0.2 WLM, and annual occupational exposures may be considerably higher. The REL of 1 WLM per year is not designed for the population at large, and no extrapolation is warranted beyond occupational exposures in underground metal or nonmetal mines. The REL is designed only for the radon progeny exposures of underground metal and nonmetal mine workers as applicable under the Federal Mine Safety and Health Act of 1969 [Public Law 91-173], as amended by the Federal Mine Safety and Health Act of 1977 [Public Law 95-164].

IV. RESEARCH NEEDS

The following research is needed to further reduce the risk of lung cancer development from occupational exposure to radon progeny.

A. Epidemiologic Studies

Needs for epidemiological studies have been identified as follows:

- A need exists for a followup study of the U.S. miner cohort from the original Public Health Service study to explore the risk of lung cancer among nonsmoking miners exposed to low concentrations of radon progeny. NIOSH is currently resurveying this cohort to update exposure histories and gather additional information on smoking behavior, dietary practices, and tumor cell types.

- A study is needed to determine whether radon gas itself or other contaminants that might be found in uranium mines are associated with increased morbidity and mortality.

- An epidemiologic study of injuries is needed in the mining industry to identify safety-related problems, since this industry has one of the highest injury rates in the United States. Particular emphasis should be given to whether or not respirator use is associated with an increased injury and health risk. This investigation should examine slips, trips, falls, and heart attacks.

B. Engineering Controls and Work Practices

Research should be conducted to develop more effective control technology methods for reducing exposure to radon progeny to less than 1 WLM. A control technology assessment of the uranium mining industry will assist in this effort by examining existing state-of-the-art technologies and work practices and by recommending new methods for controlling exposure to radon progeny.

C. Respiratory Protection

The following two types of research are recommended for respiratory protection:

- Research should be conducted to determine the extent of gamma radiation emitted from particles trapped on high-efficiency particulate air (HEPA) filters used on air-purifying respirators.

- A study should also be conducted to evaluate the physiological stress placed on miners who must wear respiratory protection. This study should be conducted both in the laboratory and in the mines.

D. Environmental (Workplace) Monitoring

Studies needed for environmental (workplace) monitoring are as follows:

- Research should be conducted to characterize and evaluate the importance of particle size, unattached fraction, and condensation nuclei concentrations in estimating the bronchial dose of radon progeny. The dose of alpha radiation affecting the bronchial airways depends on the size of the particles to which it is attached. Recent studies have shown that as particle size decreases, the concentration of radon progeny increases.

- Continued research is also needed to determine which factors affect the equilibrium between radon progeny and radon gas in mines. These studies should examine how such information may be used to predict the extent of exposure to radon progeny. Additional development and field testing of personal sampling devices is also needed for more complete determination of a miner's daily exposure.