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ATOMIC ENERGY OF CANADA LIMITED

ÉNERGIE ATOMIQUE DU CANADA LIMITÉE

RADIATION HAZARDS IN URANIUM MINING: EPIDEMIOLOGICAL AND DOSIMETRIC APPROACHES

RISQUES DÛS AU RAYONNEMENT LORS DE L'EXPLOITATION MINIÈRE DE L'URANIUM: MÉTHODES ÉPIDÉMIOLOGIQUES ET DOSIMÉTRIQUES

D.K. MYERS and J.R. JOHNSON

Text for a paper presented at the
International Workshop on
Radiological Protection in Uranium Mining,
Darwin, Australia
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Chalk River Nuclear Laboratories

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Résumé

On examine les risques possibles pour la santé provenant de l'exposition à diverses sources de rayonnement associées à l'exploitation minière de l'uranium. (a) On interprète les observations épidémiologiques de groupes de mineurs exposés dans le passé à de fortes concentrations de descendants du radon comme la suggestion d'un risque pendant la vie d'environ 3×10^{-4} cancers du poumon par niveau opérationnel-mois. (b) On peut considérer le risque total d'effets graves sur la santé provenant de l'exposition au rayonnement de l'organisme entier des travailleurs comme étant d'environ 2×10^{-2} par Sv. (c) Les effets possibles sur la santé de l'inhalation des descendants du thoron ou des poussières de minerai radioactif ne peuvent être évalués qu'à partir de méthodes dosimétriques. L'examen des incertitudes qui existent dans ces calculs laisse supposer que les valeurs CIPR de toxicité possible des descendants inhalés du thoron sont aussi bonnes que celles des descendants inhalés du radon. Toutefois, il est probable qu'on ait surestimé les risques possibles pour la santé provenant des poussières inhalées de minerai d'uranium et de thorium, d'un facteur 2 à 10.

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Abstract

Potential health hazards resulting from exposure to various sources of radiation associated with uranium mining have been reviewed. (a) Epidemiological observations on groups of miners exposed in the past to high concentrations of radon progeny have been interpreted to suggest a lifetime risk of about 3×10^{-4} lung cancers per WLM. (b) The total risk of serious health effects resulting from exposure of workers to whole body gamma-radiation might be taken to be about 2×10^{-2} per Sv. (c) The potential health effects of inhalation of thoron progeny or of radioactive ore dusts can only be estimated from dosimetric calculations. A review of the uncertainties involved in these calculations suggests that ICRP estimates of the potential toxicity of inhaled thoron progeny are as good as those for inhaled radon progeny. However, the potential health hazards from inhaled uranium and thorium ore dusts have probably been overestimated by a factor of 2 to 10-fold.

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1. INTRODUCTION

Uranium miners may be exposed to a variety of radiation sources, notably, inhalation of short-lived daughters (progeny) of radon (^{222}Rn) and of thoron (^{220}Rn), inhalation of radioactive ore dusts, and whole body exposure to external gamma-rays. Recommendations on maximum permissible limits for exposure to these sources have been published by the ICRP (1,2) and the IAEA (3). These annual limits are intended to restrict the combined effects of all occupational exposures so that the probability of serious health consequences will not exceed that which might be expected to result from whole body exposure to 50 mSv per year of X- or gamma-radiation.

The purpose of the present paper is to review current information on the biological hazards associated with the various radiation sources to which uranium miners might be exposed. There are good epidemiological data from which the biological effects of inhaled radon progeny and of gamma-radiation on humans can be estimated. The potential effects of the other sources of radiation noted above must, however, be estimated by extrapolation from calculated doses to various tissues of the body.

2. EPIDEMIOLOGICAL DATA ON RADON PROGENY

The hazards of exposure of uranium miners to high concentrations of radon and radon progeny have been recognized for several decades. Since the radiation doses received after inhalation of radon alone are very small compared to doses from short-lived radon progeny, the health hazards are now attributed almost exclusively to inhalation of the progeny. Quantitative estimates of risk based on epidemiological followup of exposed miners are becoming increasingly available in recent years.

2.2.1 Risk coefficients from various epidemiological studies

Risk coefficients are usually expressed in terms of "absolute risk", i.e. the excess number of lung cancers per 10^6 person years at risk per WLM of exposure, or in terms of "relative risk", i.e. the percent increase in normal incidence of lung cancer per WLM of exposure. Table 1 summarizes some quantitative estimates of risk of induced fatal lung cancers per WLM of exposure to short-lived radon progeny. Lung cancers appearing less than 10 years after first exposure are usually excluded in these calculations in order to ensure that the risk coefficients are not too low because of the inclusion of years of followup during which little or no effect of exposure is anticipated (5). These data thus represent the average risk coefficients observed between 5 or 10 years after first exposure and the end of the followup, at which time most of the miners were still alive.

Other groups of miners demonstrating excess lung cancers as a result of exposure to high concentrations of radon progeny include the Joachimsthal-Schneeberg silver and pitchblende miners, French uranium miners, Cornish tin miners, Chinese tin miners, Swedish iron miners from Kiruna and Grangesberg, Swedish lead-zinc miners, Norwegian niobium miners (37) and Czechoslovakian iron miners and clay shale miners (36). In general, reliable estimates of risk coefficients cannot as yet be derived from these other studies.

Table 1. Estimates of risk of induced lung cancer in various groups of miners

Group (and approximate number) of miners	Average estimated exposure WLM	Excess lung cancers appearing more than 5-10 years after first exposure		Reference
		% increase in normal incidence per WLM	Excess cancers per 10^6 person-years per WLM	
Eldorado Port Radium uranium (2,103)	273[a]	0.27	3.1	(4)
U.S. uranium (3,366)	1180	0.3-0.45 0.8-1.4[b]	3.5 6[b]	(5,6) (5,7)
Nfld. fluorspar (1,772)	548	0.9	6.4	(8)
Ontario uranium (15,984)	33	1.0 1.3[c]	9.7 7.2[c]	(9)
Czech uranium, group A started 1948-52 (2,194)	303	1.8	19.0	(5)
Czech uranium, group S (includes group A), started 1948-57 (4,043)	226	about 1.6	21.0	(36)
Malmberget iron (1,415)	94	1.4-3.6	20.0	(37,39)
Eldorado Beaverlodge uranium (8,487)	36[d]	2.6-3.3	21.0	(10,37)

[a] 44% of the person-years at risk in the category with <5 WLM and with no excess lung cancers were excluded for this calculation.

[b] Calculated for groups receiving lower exposures (<360 WLM) only.

[c] Risk coefficients for the group of miners with no prior experience in gold mining. Data based on "special WLM" estimates (9,11) are not included.

[d] 74% of the person-years at risk in the category with <5 WLM and with no excess lung cancers were excluded for this calculation.]

A major problem in all the studies listed in Table 1 is the reliability of the exposure estimates. In general, exposure rates were highest during the earliest years of mining and were decreased, primarily by improved ventilation practices, in later years. However, monitoring of radon daughter concentrations was infrequent or absent during the earliest years of mining; the exposure histories of the early miners with the highest exposures and longest follow-up have necessarily been reconstructed from minimal data. In the study of 15 000 Ontario uranium miners, estimates of radon daughter concentrations were based on results of area monitoring in the mines in the early years or in cases where no reliable measurements were available, on estimates provided by three mining engineers who were familiar with the Ontario uranium mines over the early years of operation (11).

The risk coefficients derived from these studies (Table 1) vary over a range of about 10-fold. This is true both for estimates based on the relative risk model (% increase in normal incidence of lung cancer per WLM) and on the absolute or attributable risk model (excess number of lung cancers per 10^6 person-years at risk per WLM). Part of this variation is probably due to uncertainties in estimated exposures.

There are good reasons to believe that the exposure estimates for the Swedish iron miners at Malmberget (37,40) and for the Beaverlodge uranium miners (37,41) may be too low and that the corresponding risk coefficients are therefore too high. Most of the effort in assessment and re-assessment of exposure estimates has been devoted to the studies of the U.S., Ontario and Czech uranium miners and, more recently (42), of the Newfoundland fluorspar miners. These four studies could thus be considered to provide the most reliable risk coefficients at present. The risk coefficients derived from these four studies vary over a range of about 3-fold (Table 1).

2.2.2 Minimum latent period

Earlier analyses had in general not shown an appreciable excess of lung cancers within the first 10 years of followup after first exposure to radon progeny in mines (5). However, the studies of Beaverlodge and Port Radium miners both showed zero excess of lung cancers 0-5 years after first exposure but a statistically significant increase 5-10 years after first exposure (4,10). Recent data on Czech uranium miners also showed a statistically significant excess starting at the sixth year after first exposure (36). Analyses of epidemiological data on Ontario uranium miners have also shown excess lung cancers in the period 5-10 years after exposure (14, 43). The minimum latent period between exposure of miners to high concentrations of radon progeny and the first appearance of radiation-induced lung cancers is thus probably about 5 years.

2.2.3 Effect of time since exposure

A number of authors have attempted to analyse the epidemiological data on miners in terms of age at first exposure or of age at death (or diagnosis) from lung cancer. The effect of age at first exposure has been examined in 3 of the 4 Canadian studies. No consistent trends in the risk coefficients were observed

(4,8,10). Three of the Canadian studies showed a trend towards a decrease in relative risk coefficient with increase in age at the time of appearance of lung cancers (4,8,9). A similar trend is probably valid for the followup of U.S. (13) uranium miners. This trend is very definite for the data on group S of the Czech uranium miners (Table 2).

In the general population, deaths from lung cancer are normally rare before age 40 and death rates tend to increase exponentially as the age at death increases from 40 to 75. If the relative risk model were more appropriate than the attributable risk model, one would expect to see a constant relative risk coefficient for younger and older age groups with a marked increase in absolute risk coefficient in going from the younger to the older age groups. The data from different studies are not consistent and thus do not provide good reason to reject one risk model in favour of the other.

Table 2. Risk coefficients[a] for Czech uranium miners (group S) of different ages (36).

Age at start of exposure (years)	Age at death (years)	Total number of lung cancers	Relative risk coefficient	Absolute risk coefficient
up to 30	40-49	49	4.3	6.0
30-39	50-59	59	2.0	13.1
40 and above	60-69	79	1.3	25.2
all ages	all ages	341	1.5	20.0

[a] These risk coefficients are not corrected for a 5-year minimum latency period.

The above models are relatively simple in assuming that the relative risk or absolute risk would remain constant throughout life after the minimum latent period. This assumption may be true for excess lung cancer incidence in the Hiroshima-Nagasaki survivors who were exposed to high doses of gamma-radiation in 1945 (37,44). However, a number of publications now suggest that these simple models are not the most appropriate for uranium miners exposed to alpha-particles from radon progeny. A more sophisticated risk model was also tested in a recent reanalysis of the Ontario data by Muller and co-workers (14,43). In this model, the increase in relative risk was assumed to be 0% during the first 5 years after each increment in radon daughter exposure and best values for relative risk were calculated for subsequent times after each exposure. The available data on Ontario uranium miners suggested an increase in relative risk per WLM of 1.6% at 5-10 years, 3.4% at 10-15 years and 0.3% at 15 or more years after each annual increment in exposure (14,43). For the purpose of calculating lifetime risk

estimates, Muller et al. (14) assumed that the increase in relative risk would be zero at times greater than 20 years after each exposure to radon progeny.

Other recent data have similarly suggested that risk varies appreciably with time since exposure. An analysis of U.S. uranium miner data indicated that the excess relative risk of lung cancer "fell dramatically in the years following cessation of exposure" (7). Data are also available for 4043 Czech uranium miners in study group S who started work in 1948-57 and terminated after an average of 10 years, i.e. about 1962 (36). Most of the excess lung cancers appeared in the period 10-27 years after first exposure (36) and apparently decreased to approach zero by about 33 years after first exposure, i.e. 23 years after the average termination of exposure to radon progeny in the uranium mines (45). More detailed analyses of the raw data have not yet been carried out, but the available data for Czech uranium miners (36,45) would appear to fit closely with the type of model suggested by Muller et al (14,43).

Related analyses of epidemiological data on four cohorts, the Beaverlodge, Ontario and Colorado plateau uranium miners plus the Malmberget iron miners, were carried out by the BEIR-IV committee (37). As noted above, the exposure data for the Beaverlodge and Malmberget miners are not very reliable. Moreover, the BEIR-IV analysis was based on time since last exposure in the mines, rather than on preferable parameter of time after each annual exposure. However, the basic data are most valuable and are summarised in Table 3. This summary gives observed/expected (O/E) numbers of lung cancer deaths and % excess relative risk (E.R.R.) for all miners in each cohort excluding those listed with zero exposure. The excess relative risk decreases with time after last exposure for all 4 groups of miners, although not as drastically in the other groups as in the Ontario miners group. It is, however, not obvious that an appreciable excess risk would continue throughout life after each exposure, as was assumed in the BEIR-IV report (37).

The BEIR-IV model (37) includes corrections not only for time since exposure but also an appreciable downward adjustment in relative risk coefficient for persons over age 65 at the time of appearance of lung cancer (37). The interpretation of this empirical adjustment is not clear.

2.2.4 Effect of cigarette smoking

Approximately 70% of the miners in all groups listed in Table 1 were cigarette smokers. The Ontario miners' study included large groups of gold miners and of copper-nickel miners as well as uranium miners. An excess of lung cancers was observed in the group of gold miners as well as the uranium miners, but not in the group of copper-nickel miners, who are believed to have a similar smoking history. The major cause of lung cancer among the gold miners' group appeared to be correlated with exposure to high ore dust levels in the earlier years of mining before 1945. Smoking also increases the risk of silicosis (15).

Interactions between cigarette smoking and inhalation of radon daughters were examined in the studies of Newfoundland fluorspar miners and of Ontario uranium miners. The data did not permit a clear choice between the relative risk model, indicative of a multiplicative interaction, and the absolute risk model, indicative of an additive interaction (8,14). Other data have suggested

Table 3. BEIR-IV data on excess lung cancers in four groups of miners at various times after last exposure in the mines.

Group of miners		Time since last exposure		
		<5 years	5-15 years	>15 years
Beaverlodge	O/E	21/2.84	18/7.37	15/9.08
	(E.R.R.)	(6.4%)	(1.4%)	(0.65%)
Ontario	O/E	14/5.12	35/18.39	28/27.94
	(E.R.R.)	(1.7%)	(0.9%)	(0.0%)
Malmberget	O/E	25/4.28	15/4.94	11/5.54
	(E.R.R.)	(4.8%)	(2.0%)	(1.0%)
U.S.	O/E	70/11.32	132/24.37	54/23.43
	(E.R.R.)	(5.2%)	(4.4%)	(1.3%)

that cigarette smoking accelerates the appearance of lung cancers in exposed miners, with the result that a multiplicative interaction between smoking and radon daughter exposure is most evident at the earlier stages of followup but may be absent or less evident many years after exposure (7,16).

The epidemiological data on the Malmberget miners indicate that the risk of radiation-induced lung cancers in smokers was 1.4 times that in non-smokers (39). The ratio of risks in smokers compared to non-smokers was 1.6 for the Czech shale-clay miners (36). The expected ratios would be 1.0 for an additive model and 7 to 10 for a multiplicative risk model. Other data reviewed by the ICRP (12) suggested the use of a relative risk model. However, as indicated in the 1980 BEIR-III report (5), it appears probable that neither risk model is correct and that the true risk is intermediate between those suggested by these two models.

2.2.5 Dose-response relationship

The data from all 4 Canadian studies are compatible with a linear, non-threshold relationship between accumulated exposure and incidence of induced lung cancer. The fluorspar miners' study did not provide any evidence of a decreased response per unit dose at high accumulated exposures in the region of 2500 WLM (8), such as has been observed in the study of U.S. uranium miners (5,7,16). The overall Czech data for group S are also compatible with a linear dose-response relationship, although there is some suggestion of decreased response per unit dose at cumulative exposures above 300 WLM (36).

In theory, a curvature in the dose-response relationship would not be expected until there was an appreciable probability that the target cell in the respiratory tract would be traversed by more than one alpha-particle from the radioactive decay of radon progeny. At low exposures, the chances of any given target cell being exposed to an alpha particle would be either one or zero. This would appear to be true up to cumulative doses in the region of 50 WLM during the normal lifespan of these target cells (18). In general, the epidemiological data are consistent with a linear dose-response function up to cumulative exposures of about 300 WLM over a few years.

2.2.6 Lifetime risk estimates for fatal lung cancers

In most cases, estimates of the lifetime risk of radiation-induced lung cancer are necessarily based on extrapolation from epidemiological observations over a limited period of time to projections over the total lifetime of the irradiated cohort, most of whom are fortunately still alive at the time of the observation. The particular risk projection models chosen are important for this reason. Three previous estimates of lifetime risk as derived from epidemiological studies on adult male miners are given below:

ICRP Publication 32 (1981)	$1.5\text{-}4.5 \times 10^{-4}$ WLM^{-1}
ACRP-1 (1982)	$1\text{-}6 \times 10^{-4}$ WLM^{-1}
BEIR-IV (1988)	5×10^{-4} WLM^{-1} [males exposed from birth]

The first two estimates suggest an average risk of about 3×10^{-4} WLM^{-1} for uranium miners. The BEIR-IV estimate (37) is for males exposed from birth and would be somewhat lower for adult males exposed from age 20 to 60. The BEIR-III report (5), despite an ill-advised statement to the contrary in BEIR-IV (37), did not provide an estimate of lifetime risks.

The epidemiological data on uranium miners were also reviewed in ICRP Publication 50 (12). The most appropriate average risk coefficients were considered to be about 1% increase per WLM in normal lung cancer incidence (relative risk model) or about 10 excess lung cancers per 10^6 person-years at risk per WLM (absolute risk model). These values are compatible with the data on Ontario uranium miners but are rather low for the data on Czech uranium miners (Table 1). In two recent Canadian studies, the excess risk of death from lung cancers before age 70 has been calculated for male uranium miners, the expected values for non-exposed males being derived from Canadian vital statistics (Table 4). Using the average risk coefficients suggested in ICRP Publication 50 (12), the values are equivalent to a risk of $2.5\text{-}2.9 \times 10^{-4}$ per WLM. A total lifetime risk of about 3×10^{-4} per WLM would appear to be a reasonable choice, assuming that exposure of adult male miners to radon daughters at any age resulted in an increased risk of lung cancer which continued throughout life. If the relative risk model were correct, the risk to non-smoking miners would of course be appreciably smaller (12, 17, 37); as noted above, this assumption is rather dubious.

Assuming that increased risk ceased 20 years after each exposure, the lifetime risk would be closer to 2 x 10^{-4} per WLM for male miners exposed to 1 WLM per year from age 20 to age 60, based on the time-dependent relative risk coefficients which were derived from the study of Ontario miners (14). Further study of other groups of miners and of miners exposed only at younger ages would be required to substantiate the risk coefficients for this latter model. Exposure of miners at a constant rate for 40 years is of course unlikely to occur in uranium mining, but some assumption of this kind is required in order to derive average lifetime risk estimates.

Approximate lifetime risk estimates for group S of the Czech uranium miners can also be derived directly from the data without the use of any risk projection models. Followup data to the end of 1980, when excess lung cancers were apparently declining but were still appreciably above zero, indicate 386.5 excess lung cancers in 4043 miners exposed on average to 226 WLM (36), suggesting a risk of 4.2 x 10^{-4} WLM^{-1} at 18 years after the average last exposure. The data available to date for another 5 years of followup (45) suggest that the extrapolated lifetime risk will probably be in the region of 5 x 10^{-4} WLM^{-1} for group S of the Czech uranium miners. This value is similar to the projected lifetime risk of 4.5 x 10^{-4} WLM^{-1} suggested for the Czech uranium miners in the UNSCEAR report of 1977 (23) and in ICRP Publication 32(1).

Table 4. Lifetime risk estimates for Canadian miners, adjusted to the risk coefficients given in ICRP Publication 50[a]

Age at exposure	Excess risk of death from lung cancer by age 70 per 10^4 WLM: Relative risk model	Absolute risk model	Reference
Age 30-35	2.9 [9.6]	2.5 [5.1]	(10)
Age 20-50	2.8 [2.4]	2.6 [1.7]	(8)

[[a] The original lifetime risk estimates are given in brackets in the table; these values were adjusted to the average risk coefficients of 1.0% per WLM (relative risk model) or 10 per 10^6 person-years per WLM (absolute risk model) suggested in ICRP Publication 50 (12)].

The analyses of epidemiological data available to 1988 do not suggest any reason for a major change in the range of 1-6 x 10^{-4} WLM^{-1}, or in the average value of about 3 x 10^{-4} WLM^{-1}, for the lifetime risk of fatal lung cancer following exposure of adult male miners to high concentrations of radon progeny in the air in uranium mines. This average value, based on epidemiological data only, is of course nearly twice that suggested earlier by the ICRP (1) and the NCRP (19) after consideration of both epidemiological and dosimetric approaches.

2.2.7 Other hazards of exposure to radon progeny

There is no reproducible epidemiological evidence of excess fatal cancers other than lung cancers among miners who were previously exposed to high concentrations of radon progeny. Calculations of doses to the gonads and other tissues after inhalation of radon progeny also indicate that these doses should be very small by comparison with doses to the respiratory track (1,18,20,21). The risk of induction of lung cancers is therefore considered to represent essentially all of the risk of serious health hazards attributable to inhalation of short-lived radon daughters.

An increased incidence of curable skin cancers (basal cell carcinomas) has been reported for the Czech uranium miners (36,46). No comparable increase was observed for surface workers at the uranium mines or for underground coal miners or surface workers at the coal mines in Czechoslovakia. The increased incidence among the uranium miners was attributed to contamination of the skin surface by radon progeny. The approximate risk estimate given (36) is compatible with recent estimates suggested in ICRP Publication 45 (28). Data on incidence of curable skin cancer among uranium miners in other countries are not available for comparison; the Czech data are unique in this respect.

2.2.8 Other radiation sources

Uranium miners are exposed to a variety of radiation sources in addition to inhaled radon progeny, notably: external gamma-rays from the radioactive ore, inhaled thoron progeny and inhaled uranium and thorium ore dusts. It is unlikely that these other sources made a major contribution to the excess lung cancers produced in uranium miners at a time when the concentration of radon progeny in the mine air was very high. The external gamma-ray exposures depend on the concentration of radioactive material in the ore body and would remain unchanged by the increased ventilation which has typically reduced the concentration of radon progeny in mine air by factors of 10 to 100-fold since the 1950's. Thoron progeny represent an appreciable radiological problem in some underground mines, notably at Elliot Lake in Ontario; however, their concentration is affected less than the concentration of radon progeny by increased ventilation (47). There is also some evidence suggesting that the increased ventilation rates and other practices which have been introduced since the 1950's have had less effect upon ore dust concentrations than upon the concentration of radon progeny in the mine air (48). The ICRP Task Group (12) concluded, for similar reasons, that "the observed excess lung cancer frequency among the study groups of uranium miners is mainly attributable to the inhalation of short-lived ^{222}Rn daughters. The risk contribution from inhaled long-lived radionuclides and gamma-radiation might be, on the average, about 10-20% of the total" (12). This conclusion appears reasonable. However, it should be noted that the radon progeny in the air in the Newfoundland fluorspar mines and in the Malmberget iron mines arose not from the ore itself but from dissolved radon carried into the mines by seepage water; thus the excess lung cancers observed in these two groups of miners (Table 1) cannot be attributed to radiation sources other than radon progeny.

No epidemiological data exist for direct estimation of the risk of exposure to thoron progeny or to radioactive ore dusts.

Epidemiological data on the health effects of X- and gamma-rays have been reviewed many times by scientific committees (cf. 5,22-25). For purposes of radiation protection, it has generally been assumed that exposure of radiation workers to 10 mSv of whole body radiation is associated with an average lifetime risk of about 1.3×10^{-4} fatal cancers in the workers plus a risk of 0.4×10^{-4} serious genetic diseases in the children and grandchildren of the exposed workers (22). These values are near the middle of the ranges of the risk estimates suggested by the 1980 U.S. BEIR committee (5). The suggested values are currently being reviewed by various committees in view of recent changes in estimates of absorbed doses received by atomic bomb survivors at Hiroshima and Nagasaki in 1945. Final reports on this topic are not anticipated for some time, but recent statements from the ICRP suggest that any resultant increase in the risk estimates for induction of fatal cancers in radiation workers is likely to be fairly small (27).

Whole body radiation is also expected to cause an appreciable number of non-fatal or curable cancers, primarily cancers of the skin, thyroid gland and female breast (5,22,23). These curable cancers were not considered in the derivation of weighting factors for various tissues by the ICRP in 1977 (22). If curable cancers were on average assigned roughly one-eighth the weight of fatal cancers (28), the total risk of serious health effects resulting from exposure of radiation workers might be taken to be about 2×10^{-2} per Sv (or possibly 3×10^{-2} per Sv after allowing for any future increases in risk estimates as a result of the Hiroshima-Nagasaki data). The potential health hazards associated with whole body exposure of radiation workers to 10 mSv gamma-radiation are thus expected to be of the same order of magnitude as those associated with exposure to 1 WLM radon daughters. These estimates apply to adult populations only. Complications and uncertainties associated with exposures of populations that include young children will not be considered here.

3. THORON PROGENY AND RADIOACTIVE ORE DUSTS

Potential health hazards from thoron progeny and radioactive ore dusts can only be estimated by using the results of dosimetric calculations to relate risks from exposure to these substances to risks from exposure to radon progeny or from exposure to X- or gamma-rays. For intakes of radionuclides, exposures are related to a common quantity, the committed effective dose equivalent (CEDE) or the annual limit on intake (ALI) (22). The limits recommended by the ICRP are summarized in Table 5. These limits were intended to result in a CEDE of 50 mSv to Reference Man. A lower ALI for thorium ore dust, based on the concept of limiting their commited dose to bone surfaces to 500 mSv, is recommended by ICRP (2). This limit is meant to restrict doses to values below those that might result in non-stochastic effects. Since this paper is primarily concerned with the induction of cancer and genetic effects (i.e. stochastic effects), the ALI based on this limit is given in Table 5.

Table 5. Annual limit on intake recommended by the ICRP (1,2) for various inhaled radionuclides

Radiation source	Annual limit on intake
Radon progeny	0.02 J of potential alpha energy (4.8 WLM)
Thoron progeny	0.06 J of potential alpha energy (14. WLM)
Uranium ore dust	1700 Bq of long-lived alpha activity
Thorium ore dust	380 Bq of long-lived alpha activity

Some important differences might be noted in the calculations of effective dose equivalents from which the annual limits on intake are derived. Dosimetric calculations for inhaled radon progeny which are based on a weighting factor of 0.12 for the whole lung (22) suggest a CEDE of about 4 mSv per WLM (1), i.e. a lifetime risk of about 0.7×10^{-4} lung cancers per WLM. This is appreciably lower than the lifetime risk as estimated from epidemiological studies (1). Calculations of CEDE's for radon and thoron progeny were therefore usually based on the regional lung concept (1,20,21), in which the tracheo-bronchial (TB) and pulmonary (P) regions of the lung were each assigned a weighting factor of 0.06 compared to the weighting factor of 1.0 for whole body irradiation. The average CEDE for inhaled radon progeny in the mine environment would then become about 8 mSv per WLM (21). Using these weighting factors, most (nearly 90%) of the health hazards of inhalation of radon progeny appears to be associated with irradiation of the TB region (21). Most of the potential hazard of lung cancer due to inhalation of thoron progeny would also appear to be due to irradiation of the TB region; however, the relative long half-life (10.6 hours) of thorium B (^{212}Pb) allows appreciable time for translocation of this daughter to other tissues in the body. Thus 30% of the total hazard (or, more precisely, total CEDE) associated with inhalation of thoron progeny is thought to be due to irradiation of tissues other than the lung, notably bone surfaces and kidneys (21).

The ALI's for ore dust, on the other hand, were based on the mean lung concept, in which the whole lung is assigned a weighting factor of 0.12 (22). Suggested refinements (29) of the CEDE calculations including application of the regional lung concept would lead to ALI's which are appreciably higher than those given in Table 5.

Some of the uncertainties associated with the dosimetric calculations involved in the derivation of ALI's have been considered in the NEA Experts

Report on dosimetry aspects of exposure to radon and thoron progeny (21) while other problems are currently being considered by the ICRP Task Group on respiratory tract models (30). The major uncertainties are reviewed below.

3.1 Deposition in the lungs

The main factors affecting deposition of inhaled radon and thoron progeny in lungs are breathing rate, unattached fraction of daughters, and size (plus geometric standard deviation of size) of the aerosol particles for the attached fraction of radon and thoron daughters. The effective dose equivalent will vary about a factor of two over the range of these parameters that can reasonably be expected to represent the long-term average in underground mines (21).

In the case of inhaled ore dusts, ALI's calculated with the current lung model vary over a 7-fold range as the average particle size changes from 0.5 μm to 10 μm (29). The ICRP recommendations (2) are most appropriate for 1 μm particle sizes and probably overestimate the presumed toxicity of ore dusts with larger particle sizes, since these tend to deposit in the naso-pharyngeal region (29,31). Average particle sizes for ore dusts in the Elliot Lake mines in Canada appear to be about 5 μm, which would increase the ALI's in Table 5 by a factor of about 2.5 (29). Revisions to the current lung model by the ICRP Task Group (30) suggest that even at 1 μm diameter, deposition is 2-fold lower than previously recommended (31), i.e., the ALI is too low by a factor of 2.

3.2 Retention of deposited material

The main factor governing retention of radon progeny, and to a lesser extent thoron progeny, is radioactive decay. Since the total transit time of mucus through the TB region of the lung is between 0.25 and 2 days, most of the radon progeny will decay in the TB region, whereas a considerable fraction of the thoron progeny could leave the TB region before decay. This range in retention could result in a variation in CEDE of less than two-fold for radon progeny, and of four-fold or greater for thoron progeny (21). There is also a possibility that some of the activity may be transported into the epithelium of the TB region, which would increase the dose. Although the dosimetry used by ICRP (1) has not considered this possible increase in dose, the additional contribution to the average dose to target cells in the respiratory tract is probably not very large.

Because of the long half-lives of the alpha-emitters in uranium and thorium ore dusts, the major fraction of the accumulated radiation dose to the lungs from inhaled dusts is delivered to the P region rather than the TB region. Other tissues which receive appreciable radiation doses after inhalation of ore dusts include liver, kidney, bone surfaces and red bone marrow (29). Previous calculations of doses received after inhalation of ore dusts have assumed that the alpha-emitters in the ore dusts were highly insoluble class Y materials (29), while those in Table 5 are based on the assumption that the solubility of the least soluble classification for each radionuclide is appropriate (2). Recent studies indicate that uranium ore dust in Canadian mines may be more soluble than Class Y (31). Measurements of the solubility of various ore dusts in simulated lung fluid indicated that about 40% of the uranium and a lesser amount of the other radionuclides were removed by dissolution of the ore dust

within two weeks. Measurements of the translocation of the radionuclides in ore dust from the lung to other organs in a study of instilled ore dust in rat lungs (49) have confirmed the in vitro results. If ore dusts are more soluble than has been assumed, the dose to lungs will be lower, and those to other organs will be higher, than previously calculated. However, this will have only a minor impact on the ALI as the ALI is based on the weighted sum of doses to all organs, and the decrease in lung dose is offset by a corresponding increase in doses to other organs.

3.3 Calculation of absorbed dose

The initial calculation of absorbed dose from the integrated activity of deposited radon and thoron progeny in the TB region was done by ICRP by assuming that cells at risk were distributed in the epithelium at a depth below the cilia ranging from 30 to 60 μm at the upper air-ways to 10 to 20 μm in the last bronchiole generation (1). There is considerable uncertainty in these values, and hence in the dose, particularly in the upper airways. It is noted that the absorbed dose to the TB cells calculated by ICRP is the average over all generations of the TB tree; this average is less variable than is the calculated dose in any given airway generation. This is a major difference between the ICRP (1) and the U.S. NCRP (19) dosimetry as the NCRP only uses the dose to the cells at a depth of 22 μm in the fourth airway generation. This makes the ICRP calculations less sensitive to variations in unattached fraction, to the particle size distribution of the attached fraction, and to uncertainties in cell depth than the NCRP calculations. Revisions of the lung model currently being considered by the ICRP Task Group (30) include averaging the dose over all of the bronchial epithelium over all generations in the TB region. This revision would not cause any major change from that currently calculated (1) in the mean absorbed dose to the TB region.

In the case of ore dusts, the absorbed dose to the lung was based on the average dose from activity in the pulmonary region and the associated lymph nodes. However, activity tends to concentrate and be retained in the lymph nodes, which are less radiosensitive than many other tissues in the body; the net effect of corrections for this factor would be a decrease in potential risk to the whole body (29). Calculations of absorbed doses from inhaled activity which is translocated to the bone also requires further consideration of the distribution of this activity in the bone. The assumed distribution of uranium and radium in bone is likely realistic but thorium is assumed to be deposited on bone surfaces, and to remain there indefinitely. There has been no detailed studies of the thorium distribution in bone but if it acts similar to other actinides, it will become buried in mineral bone during bone remodeling, and the dose to bone surfaces and red bone marrow will be significantly reduced (50). Priest and Birchall (51) have calculated that this remodeling results in a decrease in the bone surface dose by a factor of 6 and in the red bone marrow dose by a factor of 3 for plutonium, and similar reductions could be expected for thorium (29). Decreases in the doses of this size will result in an increase in the ALI's given in Table 5 by about a factor of 2 for thorium ore dusts, and a smaller increase for uranium ore dust.

3.4 Conversion of absorbed dose to EDE

The conversion of absorbed dose to dose equivalent and thence to EDE requires the selection of a quality factor (Q) and weighting factor (W_T). The quality factor based on the strict definition of radiation quality used by the International Commission on Radiation Units and Measurements (ICRU) is greater than 20; however, ICRP uses 20 as the limit on Q (22) because very high quality (high LET) radiation is more likely to result in cell death than a viable but damaged cell. Using similar arguments, Hoffman and Katz (32) derived a quality factor of less than 10 for alphas, and work at the U.K. National Radiological Protection Board on chromosome aberrations would tend to support this value (33). However, a Joint Task Group of the ICRP and ICRU has recently recommended a Q of 25 for alpha-particles (34).

The weighting factor of 0.12 assigned to the whole lung is derived from epidemiological data suggesting that the risk of lung cancer after exposure to radiation is in the region of 2×10^{-3} per Sv (20). As with all epidemiological data (see for example Table 1), there are uncertainties associated with this number; these uncertainties can only be minimized by further followup of exposed populations. The regional lung concept, in which half the weighting factor for the whole lung was assigned to the TB region and half to the P region, was introduced in order to arrive at some reasonable dosimetric estimates of the potential toxicity of inhaled radon and thoron progeny (1). However, "human cancers induced by cigarette smoking or exposure to environmental agents nearly always arise from epithelium in proximal regions of the bronchial tree" (5). It has been suggested therefore that about 80% of the weighting factor for the whole lung should be assigned to the TB region and only 20% to the P region (35). This change would result in an increase of up to 70% in calculated CEDE's for radon and thoron progeny; the average CEDE for inhaled radon daughters would then be closer to 14 mSv per WLM than to the previously suggested value (21) of 8 mSv per WLM. The potential hazard from inhaled radon progeny as derived from dosimetric calculations would, under these circumstances, approach more closely to that derived more directly from epidemiological studies. The suggested change in weighting factors would, on the other hand, result in a major decrease in calculated CEDE's for inhaled ore dusts that are retained in the lung. This could increase the ALI's for very insoluble uranium and thorium ore dusts by about 5-fold. The increase would be less for more soluble ore dusts because of contributions to the CEDE from radionuclides transferred from the lung to other tissues in the body.

It might be noted the dosimetric calculations provide a strong link between the potential toxicities of radon and thoron progeny, which is independent of any uncertainties in the best values for Q or for weighting factors. The ratio of the risks of lung cancer induced by radon progeny and by thoron progeny does not depend upon the particular values of Q and W_T that are selected.

4. SUMMARY

Potential health hazards resulting from exposure to various sources of radiation associated with uranium mining have been reviewed. (a) Epidemiological observations on groups of miners exposed in the past to high concentrations of radon progeny have been interpreted to suggest a lifetime risk of about 3 x

10^{-4} lung cancers per WLM. (b) The total risk of serious health effects resulting from exposure of workers to whole body gamma-radiation might be taken to be about 2×10^{-2} per Sv. (c) The potential health effects of inhalation of thoron progeny or of radioactive ore dusts can only be estimated from dosimetric calculations. A review of the uncertainties involved in these calculations suggests that ICRP estimates of the potential toxicity of inhaled thoron progeny are as good as those for inhaled radon daughters. However, the potential health hazards from inhaled uranium and thorium ore dusts have probably been overestimated by a factor of 2 to 10.

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DEFINITION OF UNITS

1 Bq (becquerel) equals 1 radioactive disintegration per second.

1 mSv (millisievert) equals one-tenth of a rem or one-thousandth of a sievert.

The Sv (sievert) is the unit of dose equivalent or of effective dose equivalent. The dose equivalent in sieverts equals the absorbed dose in joules per kilogram multiplied by the appropriate quality factor, which is conventionally taken to be one for X-, beta- or gamma-rays and to be twenty for alpha particles. Prior to 1977, dose equivalents were expressed in rem, where 100 rem equals one sievert.

The WLM (working level month) was originally defined as the exposure received by miners while working for 168-170 hours in an atmosphere containing 100 picocuries (3.7 becquerel) of radon-222 per litre in equilibrium with its short-lived progeny. If the average breathing rate is taken to be 1.2 m^3 per hour, one WLM is equivalent to inhalation of short-lived radon progeny with a total potential alpha energy of 4.2 millijoules.

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