Chapter 2. Hazards of Radiation Exposure

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Abstract. Radiation induced carcinogenesis and mutagenesis form the main risk to health from exposure to low levels of radiation. There is scant data on somatic and genetic risks at environmental and occupational levels of radiation exposure. The available data on radiation induced carcinogenesis and mutagenesis are for high doses and high dose rates of radiation. Risk assessments for low level radiation are obtained using these data, assuming a linear dose-response relationship - the so-called linear hypothesis. During uranium mining the chief source of radiation hazard is due to the inhalation of radon daughters. The correlation between radon daughter exposure and the increased incidence of lung cancer has been well documented. For radiation exposures at and below the occupational limits, the associated risk of radiation induced cancers and genetic abnormalities is small and should not lead to a detectable increase over naturally occurring rates.

INTRODUCTION

The acute effects of high levels of ionising radiations are well documented (for example the A-bomb victims at Hiroshima and Nagasaki). These effects range from cataracts of the lens of the eye, radiation burns and impairment of fertility, to extreme forms of radiation sickness. The radiation doses and dose rates required to induce these effects are at least a factor of 1000 greater than those normally encountered in the course of radiation protection.

Radiation protection for the general public and for occupationally exposed workers must deal with the effects of low level radiation. These effects can be categorised into two types:

(i) Somatic effects. These are radiation induced effects that are manifested in the person receiving the radiation. These often take the form of some type of carcinogenesis or leukaemia.
(ii) Genetic effects. These are effects to later generations through mutagenesis.

The effects can also be categorised into stochastic and non-stochastic processes.

> Stochastic effects are those for which the probability of the effect occurring has no dose threshold. Carcinogenesis and mutagenesis are considered to be stochastic effects.
> Non-stochastic effects are those for which the severity of the effect varies with dose, and for which a threshold may therefore occur. Cataracts of the lens are an example of a non-stochastic somatic effect.

The somatic and genetic effects of low level radiation exposure have a very small incidence rate and occur naturally in the population. In addition these effects often have latent periods of many years and can be only studied using statistical methods. The data on the effects of low level radiation exposure are very sparse. It is more practicable to estimate the risks of low dose levels and dose rates by the extrapolation of data from much higher dose levels, based on assumptions about the nature of the dose- response relationship, the mechanisms involved and the susceptibility of the population group at risk. In the next sections the somatic and genetic hazards of radiation exposure in general are considered, before discussing the specific hazards of radiation exposure due to uranium mining.

SOMATIC EFFECTS OF RADIATION EXPOSURE

(a) Cancer Induction at High Doses and Dose Rates

The major sources of data on radiation induced carcinogenesis in man are;

(i) Studies of A-bomb survivors at Hiroshima and Nagasaki
(ii) Studies of medical patients treated for ankylosing spondylitis using X-rays.
(iii) Pacific islanders irradiated following Bikini Atoll H-bomb tests.
(iv) Studies of lung cancer amongst uranium miners.
(v) Studies of patients following radiotherapy.

The UNSCEAR (1977) and BEIR (1972) reports provide extensive surveys of the available data on radiation induced carcinogenesis.

The derived risk estimates for mortality from radiation induced cancer are given in Table 1 (UNSCEAR 1977). Several qualifications concerning these values must be made;

(a) The observed dose-incidence data pertain to doses > 0.5 Sv and to dose rates > 10 mSv/min
(b) Some of the data were derived from patients undergoing medical treatment. These data may not be representative of the whole population.
(c) The radiation doses received by persons in some of these studies carry large uncertainties.
(d) For each cancer type, the total number of persons studied was relatively small, leading to large statistical uncertainties in the risk estimates.

Although the exact dose-response relationships for each cancer type are difficult to ascertain, the tumor induction data for high doses and dose rates are consistent with a linear, non-threshold dose effect relationship.

Table 1
Comparison of sources of risk coefficients for human carcinogenesis (UNSCEAR 1977) at high doses and dose rates

Cancer Type	Data Source	Risk Coefficient (Mortality/person-Sv)
Leukaemia	Hiroshima and Nagasaki	30×10^{-4}
	Ankylosing spondylitis	$11 \text{ to } 25 \times 10^{-4}$
	Pelvic irradiation (not in utero examinations)	17×10^{-4}
Thyroid cancer	Hiroshima and Nagasaki	$0.5 \text{ to } 2 \times 10^{-4}$
	Marshall Islanders	6×10^{-4}
	Radiotherapy of children	$1 \text{ to } 5 \times 10^{-4}$
Lung cancer	Hiroshima and Nagasaki	$10 \text{ to } 25 \times 10^{-4}$
	Uranium mining	$40 \text{ to } 180 \times 10^{-4}$
Breast cancer	Hiroshima and Nagasaki	13×10^{-4}
	Multiple fluoroscopy	110×10^{-4}
	Radiotherapy	210×10^{-4}
Bone cancer	Radiotherapy	$3 \text{ to } 5 \times 10^{-4}$
	Treatment with ^{224}Ra	$20 \text{ to } 25 \times 10^{-4}$

(b) Cancer Induction at Low Doses and Dose Rates

The extrapolation of the dose-response data to the low doses and dose rates of interest in radiation protection requires the assumption that the functional relationship is unchanged. However data from unicellular and animal studies indicate that repair mechanisms for radiation induced injury exist at low doses and dose rates, and in particular for low LET (Linear Energy Transfer) radiations such as X-rays and γ-rays.

Counter to this effect, there is evidence for an increase in the carcinogenic effect of radiation at low dose rates, due possibly to the reduced killing of cancer susceptible cells. (BEIR 1972).

The dose-response functions are also dependent on the radiation type. The effects of γ-rays and X-rays on biological material are attributable to the production of fast electrons in the interaction with the material. The γ-rays and X-rays produce sparsely distributed energy releases, with perhaps one or two interactions within the volume of a cell (of size ~ 1 μm). High LET radiation, such as protons, neutrons and α-particles produce very dense ionisation within materials (the neutrons, although uncharged, quickly produce knock-on protons). Therefore, there are many more interactions within the volume of a cell.

The differences in the rate of energy release for the different radiation types lead to a variation in the relative biological effectiveness (RBE) of the radiation. That is, high and low LET radiations might be expected to have different dose response relationships. The available experimental data indicate that for simple organisms, the dose-response relationships are linear. This can be interpreted as indicating that a hit to a single radiation sensitive target within the organism is sufficient to cause the

effect. For more complex cells, a linear dose-response function is obtained only for high LET radiation. The data on human radiation induced carcinogensis, although limited, indicate that α-particles and neutrons at low dose rates are at least a factor of 5 more effective per rad to average tissue than γ-rays or X-rays at high dose rates (BEIR 1972).

(c) The Linear Hypothesis for Risk Assessment at Low Doses and Dose Rates.

There is insufficient data available to fit functional forms to the dose-response relationships for the different types of human carcinogenesis and mutagenesis. Within a population group, these functions are likely to be dependent upon a number of modifying effects such as age, sex and heredity, making their application on a general basis of dubious validity.

The currently accepted method of risk assessment at low doses and dose rates requires the extrapolation of the data assuming that there is no threshold and that the response varies linearly with dose. This assumption, termed the "linear hypothesis" is consistent with much of the epidemiological data and provides the only workable method of risk estimation at occupational and environmental radiation levels. Estimates made using the linear hypothesis are usually interpreted as being an upper limit to the actual risk. There is evidence to suggest that a linear extrapolation from dose-response data at doses less than 1Gy provides an accurate estimate of the low level risk (Brown 1976). However such data are scarce.

The risk coefficients for human carcinogenesis, derived from the high dose data and subject to the restrictions of the linear hypothesis, are given in Table 2. (Watson 1977).

Table 2
Risk Coefficients for Carcinogenesis (Watson 1977)

Whole-body exposure	Risk (per person-Sv)
Leukaemia	20×10^{-4}
All cancer including leukaemia	100×10^{-4}
Specific organs	
Cancer of bone	5×10^{-4}
Cancer of lung	15×10^{-4}
Cancer of breast	15×10^{-4}
Cancer of thyroid	5×10^{-4}
Cancer of other organs (excluding leukaemia)	40×10^{-4}

GENETIC EFFECTS OF RADIATION EXPOSURE

The genetic effects of radiation arise from the production of gene mutations and chromosome abberations. The incidence rate of all types of non-radiation induced genetic effects in the general population is ~ 10%/o (BEIR 1979). There is very little human data on radiation induced mutanogenesis. The studies of the A-bomb survivors showed an apparent absence of radiation induced genetic effects, and allow only upper limits to be placed on possible effects.

The main sources of data for assessing these risks come from extensive studies of Drosophilia (fruit flies) and of mice. For irradiation of Drosophilia spermatozoa the induction of genetic effects appears consistent with a linear dose-response relationship down to 0.25 Gy. However, the data on irradiation of Drosophilia eggs and of mice are consistent with a dose response function having a quadratic component. This suggests that in some circumstances estimates derived using the linear hypothesis are likely to over estimate the risks. The over-estimate using the linear hypothesis occurs because the points from the high dose data are assumed to lie on a straight line which passes through the origin, i.e. the zero dose-zero effect point. This line lies above any quadratic dose response function.

A conservative estimate of risk for radiation induced abnormalities is 5 per million parental mSv, expressed mostly in the first two generations (Watson, 1977).

RADIATION HAZARDS DURING URANIUM MINING

During the mining of uranium the two main sources of radiation hazard arise from the external γ-ray field due to the uranium ore and from the inhalation of the decay products of the radon present in the ore. Uranium ore grades for deposits in Australia are of the order of 0.2^{0}/o (with the exception of deposits similar to the Narbalek deposit, where the average ore grades are as high as 2^{0}/o. Such ore grades are atypical). The γ-ray field above a 0.2^{0}/o ore body is ~ 10 μGy / hour. For a 170 hour working month this amounts to ~ 20 mSv/year, a factor of 2.5 below the present occupational limit for continuous exposure. Using the risk factors from Table 2, and subject to the assumption of the linear hypothesis, an upper limit of 2 radiation induced cancers per 10,000 worker-years can be inferred from external irradiation for somatic effects. The associated rate of radiation mutagenesis for those miners capable of producing children, would be 0.01^{0}/o or less than a 0.1^{0}/o increase to the natural mutation rate.

In the uranium mining industry the greatest cause of deaths from radiation exposure has been from lung cancer caused by the inhalation of radon daughters. Studies going back to the 15th century had shown an increase in the proportion of respiratory deaths amongst miners working underground in silver mines in Germany and Czechoslovakia. These mines were later shown to contain significant amount of uranium and estimates of the radon concentration range as high as 400 Bq/l (Holaday 1969). Initially termed "mountain sickness", the disease was identified during the 19th century as being a form of primary lung cancer. In the 1920's the radon gas associated with the uranium ore was suspected as a possible cause for the increase in lung cancer deaths.

From calculations of the radiation dose to the lungs and trachea following the inhalation of radon, it was found that the main contribution to the lung dose arose from the alpha-particle radiation produced by the 4 short lived radon daughters; ^{218}Po, ^{214}Pb, ^{214}Bi and ^{214}Po. The calculations indicated that free atoms were deposited in the upper respiratory tract where they subsequently decayed. Atoms attached to dust were deposited deep in the lungs. The heaviest radiation doses arose from α-particles incident on the cells lining the bronchial tubes. This is the region where most lung cancers occur in uranium miners. The conclusion drawn from this modelling was that the concentration of the radon daughters are of greater importance to radiation protection than the radon concentration.

The epidemiological studies to date show a definite correlation between the cumulative exposure to radon daughter products, as measured in WLM (Working Level Months) and the increased incidence of lung cancer amongst miners. The two most comprehensive studies have been carried out in the U.S. and in Czechoslovakia. Both studies demonstrate that the excess in lung cancer amongst uranium miners was not attributable to age, smoking, nativity, heredity, organisation, self-selection, diagnostic accuracy or prior hard rock mining.

The American study was carried out by the U.S. National Institute for Occupational Safety and Health (NIOSH) and covered miners who had worked one or more months in the Colorado uranium mines prior to 1964 (Archer et al 1973). The incidence of lung cancer over the period 1950 to 1968 was studied in a sample of 3366 white and 780 non-white uranium miners. The results showed an excess of 58 deaths due to lung cancer above the match controls. The miners in the sample group were classified into six cumulative exposure categories. The resultant exposure-response data were consistent with a linear response with dose, as shown in Figure 1, with a slope of ~ 3.2 cases per year per 10^6 miners-WLM. Over a 30 year period this amounts to a total risk of 100×10^{-6} cases per WLM. (Lundin et al 1971).

The Czechoslovak study used a sample group somewhat larger than the American study and examined the incidence of lung cancer amongst Czechoslovakian uranium miners over the period 1948-1973 and 1948-1975 (Kunz et al 1979). The data from this study suggest that a statistically significant excess of lung cancers may result from cumulative exposure levels as low as 150 WLM. Figure 2 shows the results for miners who commenced work during the years 1948-1952. The miners were followed up for a period of 26 years. The resultant exposure-response data are again consistent with linearity, and some of the data are shown in Figure 2. The data yield an excess lung cancer rate of 230×10^{-6} cases per WLM. The study also found that this rate was dependent upon the age of the miners at the start of mining, varying from 140×10^{-6} cases/WLM for those under 30 years to 370×10^{-6} cases/WLM for those older than 40 years.

Further studies on excess lung cancers due to radon daughter exposure include;

(i) A study of miners in Sweden (non-uranium) yielded an excess mortality rate of 3.4×10^{-6} cases per year/WLM (Snihs 1973).
(ii) A study of fluorospar miners in Newfoundland yielded an excess mortality rate of 2.2×10^{-6} cases per year/WLM (de Villiers et al 1979).
(iii) A study of miners in British iron ore mines found an excess mortality rate of 6.0×10^{-6} cases per year / WLM (Boyd 1970).

In each of these three studies, there was occupational exposure to increased concentrations of radon, present in the mines. Studies of miners in Canadian uranium mines showed an increase in the death rate from lung cancer, from 0.3% for the unexposed control group, to 3.7% in the group with a cumulative exposure of 180 WLM. (Ham et al 1976). These data were consistent with a linear, non-threshold dose-response function down to exposure levels of 10 WLM.

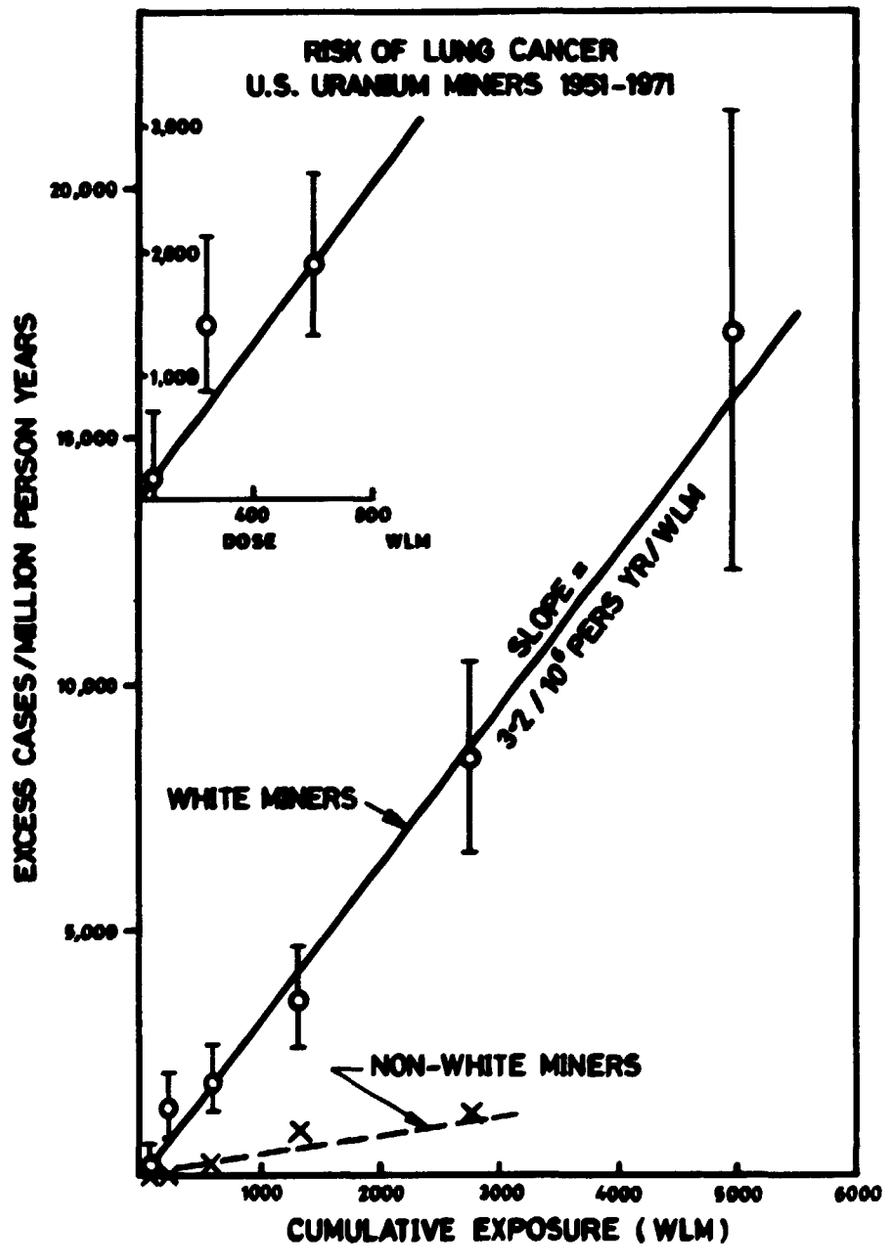
RISK OF LUNG CANCER
U.S. URANIUM MINERS 1951-1971
EXCESS CASES/MILLION PERSON YEARS
20,000
15,000
10,000
5,000
3,000
2,000
1,000
400
800
DOSE
WLM
SLOPE =
3·2 / 10⁶ PERS YR/WLM
WHITE MINERS
NON-WHITE MINERS
1000
2000
3000
4000
5000
6000
CUMULATIVE EXPOSURE (WLM)

FIG. 1

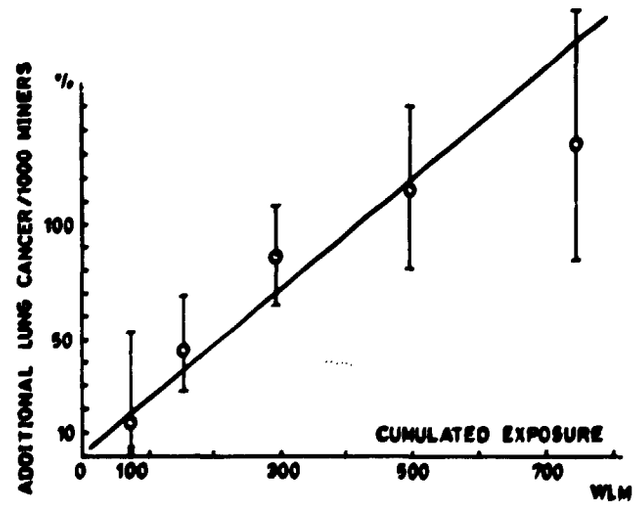
ADDITIONAL LUNG CANCER/1000 MINERS
%
100
50
10
CUMULATED EXPOSURE
0
100
200
500
700
WLM

FIG. 2

For a working-year exposure at the occupational limit of 4 WLM, the number of induced lung cancers may be expected to be .3 to 1 cases per 1000 workers, over a period of 40 years after exposure.

The Effect of Cigarette Smoking

The risk estimates given above mainly pertain to cigarette smoking miners. Although cigarette smoking is itself associated with increased lung cancer incidence, smoking alone is not able to account for all excess uranium miner lung cancers. The lung cancer rates for cigarette smoking uranium miners are at least a factor of 6 greater than from smoking non-miners. There is also evidence for a possible interaction between cigarette smoking and radon daughter exposure. It has been noted that cigarette smoke particles are the optimum size for attachment of radon daughter products. The excess mortality rates from lung cancer amongst non-smoking uranium miners are a factor of 8 lower than for miners who smoke at least 20 cigarettes per day (Archer 1976). The difference in the slopes of the curves in Figure 1, for white and non-white miners, may be due to differences in smoking habits. For the white sample group, 77o/o of the miners and 99o/o of the lung cancer cases were cigarette smokers. The non-white sample group were predominantly non-smoking amerindians. There are also data showing the latent period for lung cancer induction to be 6 to 7 years less for cigarette smoking miners than for non-smoking miners. Overall, there is a strong case for discouraging cigarette smoking amongst uranium miners.

SUMMARY

For most uranium mines in Australia, planned or operational, the somatic and genetic risks associated with irradiation by the γ-ray radiation from the uranium ore are small, the exposures being well within the occupational limits.

The largest risk related to radiation exposure is expected to arise from the inhalation of radon daughter products, leading to possible induction of lung cancer. This risk, although small at exposure levels below the occupational limits, can be increased by cigarette smoking.

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