CHAPTER 3. BIOLOGICAL EFFECTS AND HAZARDS OF RADIATION EXPOSURE

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ABSTRACT

Radiation induced carcinogenesis and mutagensis form the main risk to health from exposure to low levels of radiation. This risk effects can be at least qualitatively understood by considering the effects of radiation on cell DNA. Whilst exposure to high levels of radiation results in a number of identifiable effects, exposure to low levels of radiation may result in effects which only manifest themselves after many years. Risk estimates for low levels of radiation have been derived on the basis of a number of assumptions. In the case of uranium mine workers a major hazard arises from the inhalation of radon daughters. Whilst the correlation between radon daughter exposure and lung cancer incidence is well established, the numerical value of the risk factor is the subject of controversy. ICRP 50 gives a value of 10 cases per 10^6 person-years at risk per WLM (range 5-15 x 10^{-6} PYR^{-1} WLM^{-1}). The effect of smoking on lung cancer incidence rates amongst miners is also controversial. Nevertheless, smoking by miners should be discouraged.

INTRODUCTION

The biological effects of ionizing radiation arise from the changes induced by radiation in the cells of the body. These changes result in either cell death, which occurs when the cell is unable to produce viable daughters on cell division, or in cell damage. In the case of cell damage, the cell can survive and divide, but may transmit an induced abnormality to future generations. On a molecular level, the energy lost by radiation in passing through a cell causes ionization of water molecules along the track of the particle or photon. These ions are then able to interact with the DNA molecules of the nuclear chromosomes of the cell, and cause breaks in the strands of the DNA double helix, which carries the genetic code. If only one

strand is broken, the cell is able to repair the break correctly within a few minutes, using the unbroken strand as a template. If both strands are broken in approximately the same position and at the same time (i.e. before repair of one can take place), there is no template and the strands may either not rejoin or may be rejoined with an incorrect sequence of base pairs. This incorrect sequencing will affect the structure (and hence the function) of the proteins of the cell, which are formed using the information carried by the DNA molecule.

The molecular model of the effects of radiation allows us to explain a number of important biological observations, at least on a qualitative basis. These include

(a) The acute effects of high doses of radiation, which result from substantial numbers of cell deaths, probably due to double strand breaks which are not repaired prior to cell division.

(b) Cancer induction, which probably results from incorrect repair of the DNA molecule and the subsequent breakdown of the enzyme mechanisms controlling cell function and division.

(c) Genetic effects, which result from the transmission of incorrectly repaired DNA molecules to future generations.

(d) The differences between the dose-response relationships postulated for high LET radiation (α-particles, protons and neutrons) and low LET radiation (photons and electrons) and the greater effectiveness of high LET radiation in inducing radiation effects.

(e) The variation in the radiosensitivity of different organs or tissues is related to the rate at which the cells divide and the rate at which DNA repair can take place within a cell.

Some of these observations will be discussed in more detail in subsequent sections. However, it should be noted at this point that few areas of biology generate more controversy than those associated with the questions of dose-response curves and thresholds at low doses. Since even persons who are exposed as a result of their occupation are unlikely to receive lifetime occupational dose equivalents in excess of around 100 mSv (Thorne 1987), most radiation exposures, to both radiation workers and to members of the public are in the low dose category.

CLASSIFICATION AND NATURE OF RADIATION EFFECTS

The effects of radiation are classified as somatic or genetic and stochastic or non-stochastic.

- Somatic effects are those which appear in the exposed person. They include acute short-term effects, which appear as a result of a single large exposure (e.g. nausea, infection) or late effects (e.g. cancer, cataract formation).

- Genetic effects are those which appear in future generations. They may be inconsequential to the individual of a later generation or may result in a serious handicap.

- Stochastic effects are those which occur in a statistical manner, i.e. the probability of the effect occurring is a function of dose and has no threshold (i.e. there is no dose below which the effect does not occur). Cancer induction and the induction of genetic defects are normally considered to be stochastic effects. Given a population exposed to a known amount of ionizing radiation, it is possible to estimate how many cancers will be induced but not to identify which particular individuals will contract cancer as a result of that exposure. The severity of a stochastic effect is independent of the dose received.

- Non-stochastic effects are those where the severity is a function of dose and where there is a clear causal relationship between the exposure and the effect in a particular individual. There is usually a threshold below which no effect is observed. An example of a non-stochastic effect is skin reddening e.g. as in a sunburn.

Effects of High Radiation Doses - The Acute Radiation Syndrome

The acute effects of radiation exposure have been documented and are summarized in Table 1 for the case of a single, large, short-term, whole-body dose of gamma radiation (see e.g. Turner 1986).

Table 1

Acute Radiation Syndrome for Gamma Radiation

Dose (Sievert)	Symptoms	Remarks
0 - 0.25	None	No detectable effects.
0.25 - 1	Mostly none. A few persons may exhibit mild prodromal symptoms, such as nausea and anorexia	Bone marrow damaged; decrease in red and white blood-cell counts and platelet count. Lymph nodes and spleen injured; lymphocyte count decreases.
1 - 3	Mild to severe nausea, malaise, anorexia, infection.	Haematologic damage more severe. Recovery probable, though not assured.
3 - 6	Severe effects as above, plus haemorrhaging, infection, diarrhea, epilation, temporary sterility.	Fatalities will occur - about 50°/o in the range 4.5-5 Sv.
More than 6	Above symptoms plus impairment of central nervous system; incapacitation at doses above 10 Sv.	Death expected.

The acute radiation syndrome exhibits four sequential stages, where the individual exhibits symptoms which depend on the magnitude of the dose.

- up to 48 hours after exposure (the prodromal period), tiredness, nausea, sweating, anorexia.

- 48 hours to 2 or 3 weeks after exposure (latent period), general well-being.

- 2 or 3 weeks to 6 to 8 weeks after exposure (manifest illness stage), damage to the haematological system as shown by haemorrhaging and infection, fever, loss of hair (epilation), lethargy, perception disturbances, diarrhoea. Death may also occur during this period.

- several weeks or months later, a recovery stage occurs.

An acute, whole body gamma ray dose of around 5 Sv is regarded as being fatal to 50°/₀ of the population within 30 days. This is designated as the LD50/30 dose.

Delayed Somatic Effects

These are effects which are only manifested many years after exposure of the individual concerned. The most important of these is the production of cancer. However, there a number of other effects which may occur, including degenerative changes of specific organs and organ systems, cataracts, impairment of fertility and growth and developmental defects in fetuses and young children. The ICRP has set special limits on exposures to the lens of the eye and on the exposures to women during the gestation period (see below).

Genetic Effects

Studies on various species (e.g. the mouse or the or the fruit-fly Drosophila) have given evidence for the dose dependence of the induction of genetic abnormalities. However, there appears to be no reliable data for similar occurrences in man. Part of the difficulty in arriving at reliable estimates of the genetic risk arises because the normal incidence of genetic abnormalities is approximately 10°/₀ of all live births. (Not all of the defects are necessarily harmful or fatal). In the case of the A-bomb survivors, extensive studies have failed to provide clear evidence of inherited abnormalities in children born since 1945. Even though there does appear to be evidence of a slight increase in the incidence of several types of genetic defect, the overall statistical evidence is assessed as being not reliable enough (Mettler and Moseley 1985, p60). However, there is evidence for an increased frequency of mental retardation in children exposed in utero to the atomic bomb radiation at Hiroshima and Nagasaki (Otake and Schull, 1984).

Both UNSCEAR (1982) and BEIR (1980) have compiled extensive reports on the genetic effects of ionizing radiation. These reports are broadly consistent with the estimates of risk given by ICRP (1977) of 4×10^{-3} serious hereditary effects in the first two generations per parental Sv. The risks to future generations are regarded as being twice this figure. The figure given above, which corresponds to 20 cases per generation per million persons exposed to 10 mSv (1 rem), may be compared with the incidence of serious genetic defects not attributable to radiation of ca 10,000 per million live births and of another 90,000 per million live births of irregularly inherited genetic disorders (Mettler and Moseley 1985 p56, BEIR 1980 p85).

CANCER INDUCTION BY IONISING RADIATION

Data on the number of cancers induced following radiation exposure have been obtained from

(a) studies of the A-bomb survivors at Hiroshima and Nagasaki.

(b) accidental irradiation from fallout after nuclear weapons tests (e.g. at Bikini Atoll).

(c) studies of patients after radiotherapy and diagnostic radiation exposures.

(d) occupational exposures, such as those of radium dial painters (for luminous watch dials), doctors and others engaged in radiation diagnosis and therapy and workers in uranium mines. We will consider the latter studies in more detail below.

Several qualifications need to be made when discussing this data.

(a) The doses and dose-rates are generally high e.g. doses greater than 500 mSv (50 rem) and dose rates greater than 10 mSv min^{-1} (1 rem min^{-1}).

(b) Data derived from patients undergoing medical treatment may not be representative of the population as a whole.

(c) The actual radiation doses received in some of these studies are subject to large uncertainties. In the case of the A-bomb data, the doses received have recently been re-interpreted and this may lead to a revision of the risk factors (RERF, 1987).

(d) Since the total number of persons with each type of cancer was relatively small, there are large uncertainties in the rates of induction of cancer of each type.

From these types of study mortality rates have been derived and are expressed in terms of the probability of induction of a fatal cancer in a person exposed to 1 Sv of radiation. Some of these mortality rates are given in Table 2.

Table 2

Comparison of mortality rates 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 to 25×10^{-4}
	Pelvic irradiation (not in utero examinations)	17×10^{-4}
Thyroid cancer	Hiroshima and Nagasaki	0.5 to 2×10^{-4}
	Marshall Islanders	6×10^{-4}
	Radiotherapy of children	1 to 5×10^{-4}
Lung cancer	Hiroshima and Nagasaki	10 to 25×10^{-4}
	Uranium mining	40 to 180×10^{-4}
Breast cancer	Hiroshima and Nagasaki	13×10^{-4}
	Multiple fluoroscopy	110×10^{-4}
	Radiotherapy	210×10^{-4}
Bone cancer	Radiotherapy	3 to 5×10^{-4}
	Treatment with ^{224}Ra	20 to 25×10^{-4}

CANCER INDUCTION AT LOW DOSES AND DOSE RATES

Whilst estimates can be readily made of the increased cancer risks at high doses, most occupational and public exposures are quite low. Few occupational exposures are greater than 3 mSv $year^{-1}$ (Pochin, 1987), and it must be remembered that natural background exposures are around 2 mSv per year. Thus valid estimates of carcinogenic risks from populations exposed at dose rates of a few mSv per year or less are very difficult to obtain. Three types of study may be mentioned in this context.

(a) attempts to detect excess cancers in populations living in high background areas (e.g. at high altitude or in areas with high natural backgrounds such as Kerala (India)).

(b) detection of excess cancers following occupational exposures.

(c) detection of excess (or apparent excess) cancers in small communities.

These three types of study have been discussed by Pochin (1987). However, it still remains true that at the present time there are no reliable estimates of the risks of radiation exposure at low dose rates. Thus the risk factors for low doses and low dose rates are based on the results at high doses - making the assumptions that

(a) there is no threshold

and (b) that there can be a linear extrapolation of the risk factors at high doses to zero at zero dose. This is the linear hypothesis.

There is very considerable debate about the validity of both these assumptions. The ICRP (1977) and the majority of the BEIR III Committee (BEIR 1980) consider that up to a few gray of absorbed dose, the dose-response curve has the form

$$E = aD + bD^2$$

where E represents the effect concerned, D the dose and a and b are constants. This means that a linear term predominates at low doses and a quadratic term at high doses. The assumption is then made that this expression can be simplified to

$$E = aD$$

at low doses and dose rates (ICRP 1977). This is justified on the basis that the doses of interest in radiation protection are generally relatively small and/or are delivered at low dose-rates. The linear expression is then used to extrapolate the risk factors derived at high doses and dose rates (Table 2) to low doses and dose rates, with the risk being zero at zero dose. Despite these difficulties, the ICRP has derived risk factors which "are intended to be realistic estimates of the effects of irradiation at low annual dose equivalents (up to the Commission's recommended dose-equivalent limits)" (ICRP 1977). The risk factors are given in Table 3.

Table 3

Risk Factors

Organ or tissue	Risk (Sv^{-1})	Comments
Gonads	4×10^{-3}	Serious hereditary effects in the first two generations of offspring
Red bone marrow	2×10^{-3}	Fatal leukaemia
Endosteal cells on bone surfaces	5×10^{-4}	Fatal osteosarcoma
Lung	2×10^{-3}	Fatal lung cancer
Thyroid	5×10^{-4}	Fatal thyroid cancer
Breast	2.5×10^{-3}	Fatal breast cancer
Skin	1×10^{-4}	Fatal skin cancer
Other tissues	$<5 \times 10^{-3}$	Fatal cancer

If the linear-quadratic dose-response curve is correct, the use of a linear extrapolation to estimate the risks at low doses from those at high doses will result in an over-estimate of these risks. A section of the radiation protection community believes that there is a gross over-estimate of the risks. However, a number of authors consider that the linear hypothesis results in an under-estimate of the risks (e.g. Gofman 1981), particularly for neutron irradiation (i.e. high LET) (Rossi 1980). Conversely, other authors claim that there are hormesis, i.e. beneficial, effects at low doses. (Sagan 1987).

RADIATION HAZARDS DURING MINING AND MILLING OF RADIOACTIVE ORES

The three main sources of radiation hazard during the mining and milling of radioactive ores arise from

(a) the external γ-radiation

(b) inhalation of the decay products of radon

(c) inhalation or ingestion of long-lived radioactive dust

The existence of radiation hazards during the mining and milling of thorium-containing ores has only recently been recognized as a matter of concern. Similarly, the hazards arising from inhalation or ingestion of radioactive dust have only recently been recognized as the major hazard in some Australian uranium mines. There is therefore no body of epidemiological data on which to base estimates of risk from these sources of exposure. Whilst there has been a greater recognition of the hazard due to γ-radiation in a uranium mine (e.g. McCurdy et al 1969, Miller 1977, Frank and Benton 1981) the epidemiological data again does not appear to have been collected.

The procedure followed to estimate the risk of fatal or genetic effects is therefore to use the radiation dose as measured or estimated in the field, dosimetric models (see e.g. Chapter 8) and the risk factors of Table 3. This procedure may be illustrated by the following example.

Uranium ore grades for deposits in Australia are of the order of 0.2°/$_{\circ}$ (with the exception of deposits similar to the Narbalek deposit, where the average ore grades were as high as 2°/$_{\circ}$. Such ore grades are atypical). The γ-ray field above a 0.2°/$_{\circ}$ ore body is ~ 10 μGy h^{-1}. 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°/$_{\circ}$ or less than a 0.1°/$_{\circ}$ increase to the natural mutation rate.

Historically the largest single cause of deaths from radiation exposure in the uranium mining industry has been regarded as arising 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 kBq m^{-3} (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 radon daughters not attached to aerosols were deposited in the upper respiratory tract where they subsequently decayed. Atoms attached to aerosols 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.

Epidemiological Studies of Lung Cancer Risks for Mining Populations

Epidemiological studies of the lung cancer risks for mining populations have been appearing since the early 1970's. The field is currently of considerable interest and controversy, as the risk factors derived from these studies are the only reliable estimates of the risks applicable to elevated levels of radon in homes. The various epidemiological studies and the approaches to models for lung cancer risks have been summarized in ICRP 50 (1987). A number of detailed accounts of the problems due to the hazards of indoor radon have also appeared recently (see e.g. Bodansky et al. (1987) and Nazaroff and Nero (1988).

It is useful to bear in mind that the risks can be expressed either in terms of a relative risk model or an absolute risk model. The relative risk model is based on the assumptions that a single exposure to radon daughters at a particular age leads, after a constant latent period to an excess of the age-specific lung cancer rate which is (a) proportional to this exposure and (b) is also proportional to the normal rate in the particular population group without exposure to radon daughters. The risk coefficient is expressed in terms of the relative increment of the expected lung cancer frequency per unit of exposure (i.e. Observed - expected/expected, expressed as a percentage, per WLM).

The absolute (or excess) risk model assumes that there is no correlation between the radiation induced excess rate and the normal, strongly age-dependent, appearance of lung cancer, i.e. that a single exposure to radon daughters at a particular age, leads, after a certain time lag, to a potential excess rate of lung cancer which remains constant in the subsequent lifetime but which can depend on the age at exposure. The absolute risk coefficient is expressed as the number of excess cases per 10^6 person-years at risk (PYR) per unit of exposure (usually WLM).

Whilst both models may be applicable to the data (see e.g. Muller et al. 1985, but cf. ICRP 50 (1987) p 30) there seems to be general agreement that the conclusions derived from the relative risk model are more readily extrapolated to a different population (e.g. to a study of the exposure of the general public to indoor radon) than the absolute risk model. However, for convenience and ease of comparison between the various studies we will express the risk coefficients below in terms of the absolute risk, i.e. number of excess cases per 10^6 person-years at risk (PYR) per WLM of exposure. It should be noted that it is often assumed that for exposure at young ages, the excess rate starts at age 40 years and for exposure at higher ages, a time lag of 10 years is often assumed (ICRP 50 (1987), p 36). However, detailed comparisons require a knowledge of these assumptions.

The most comprehensive of the earlier epidemiological studies of the effects of exposure to radon daughter products were carried out on miners from the Colorado Plateau in the U.S.A. and on uranium and other mine workers in Czechoslovakia. Both studies showed a correlation between the cumulative exposure, as measured in Working Level Months (WLM) and the increased incidence of lung cancer amongst the mine workers. This correlation remained valid even when other possible influences such as age, smoking habits, population selectivity, measurement accuracy and prior hard rock mining experience were considered.

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

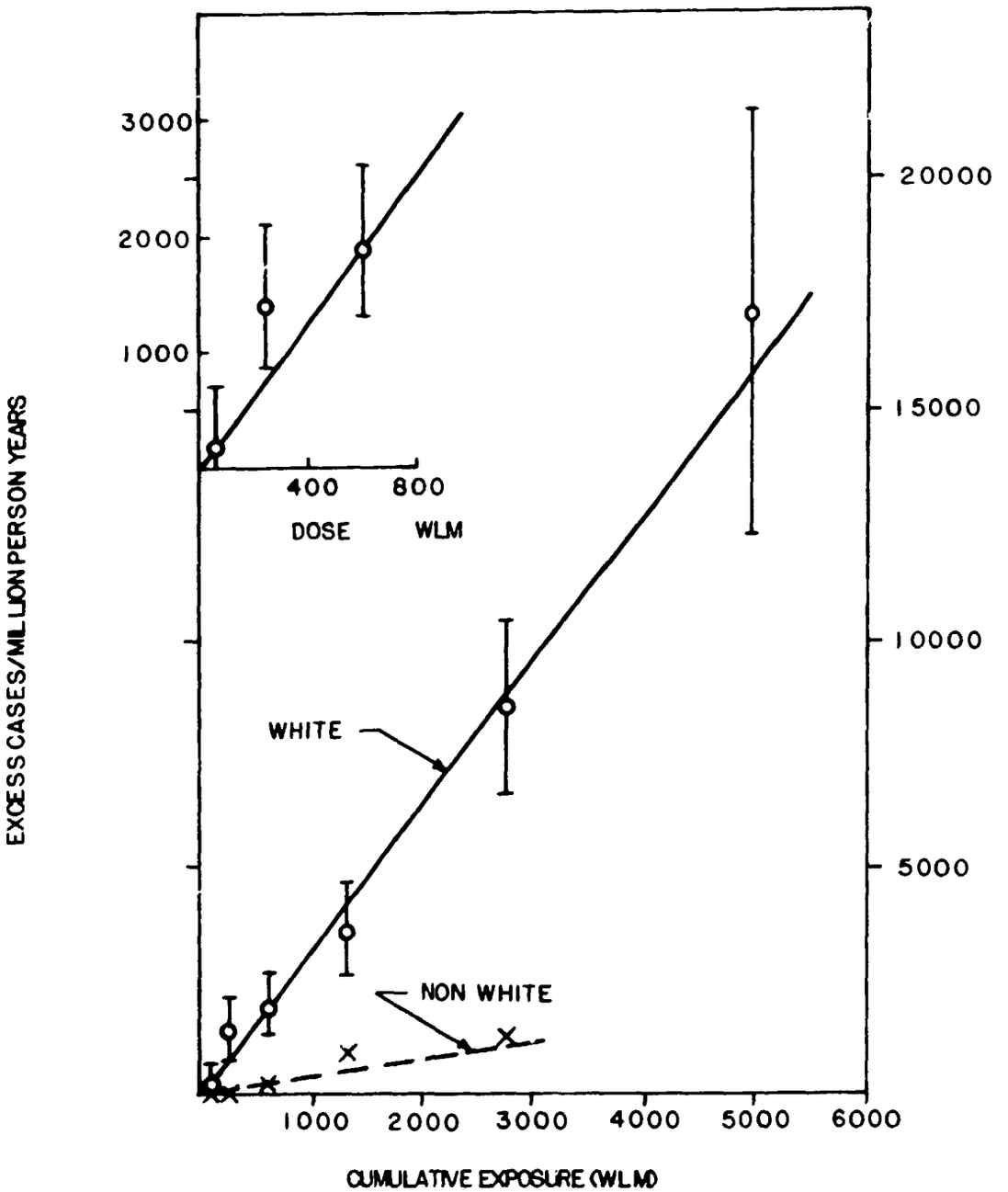

Figure 1. Risk of Lung Cancer for U.S. Uranium Miners, 1951-1971. (The inset shows the risk for low cumulative exposures).

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.

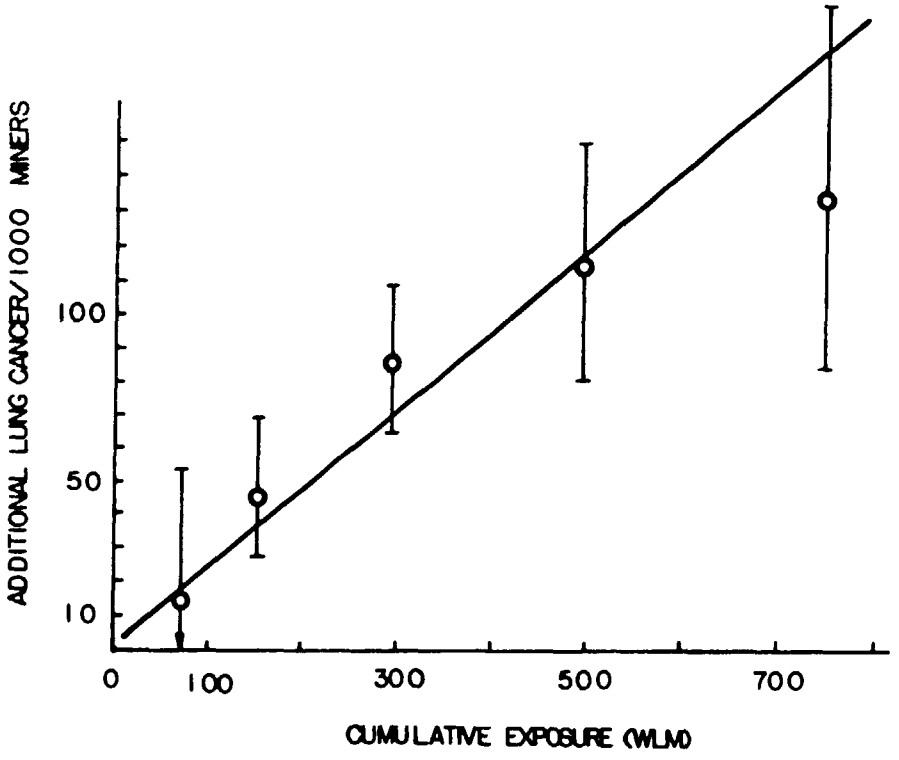

Figure 2. Risk of Lung Cancer for Czechoslovakian Uranium Miners, 1948-1978.

Other studies on excess lung cancers due to radon daughter exposure have included:-

(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. 1971).

(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).

(iv) Studies of miners in uranium mines in Ontario, Canada showed an increase in the death rate from lung cancer, from 0.3°/$_{o}$ for the unexposed control group, to 3.7°/$_{o}$ in the group with a cumulative exposure of 180 WLM. (Ham 1976). These data were consistent with a linear, non-threshold dose-response function down to exposure levels of 10 WLM. A separate study of miners at Port Radium, Northwest Territories, Canada (Grace et al 1980) confirmed the earlier trends but indicated that a more exhaustive follow-up on mine employees exposed to air-borne radiation, was required.

The initial studies have been re-evaluated and additional data included. Summaries are given in the 1980 BEIR report (BEIR 1980), by Thomas et al. (1985), in the 1986 UNSCEAR report (UNSCEAR 1986) and in ICRP 50 (1987). Some of the uncertainties in these studies have been discussed by Steinhauser and Hofmann (1985). The most recent of these follow-up studies is of the Czechoslovakian mine workers (Sevc et al. 1988). Their conclusions may be summarized as follows:-

(i) A significant excess of lung cancers occurs in exposure categories below 50 WLM and furthermore, there appears to be an increased risk per WLM at low exposures when compared with that at high accumulated exposures. However, the effects of background radiation, e.g. from Rn daughters in dwellings, were not taken into account.

(ii) The mean attributable annual cancer risk after about 30 years of observation was <u>ca</u> 20 x 10^{-6} y^{-1} WLM^{-1} and for persons commencing exposure after 30 years of age, <u>ca</u> 30 x 10^{-6} y^{-1} WLM^{-1}.

(iii) The dose-effect relationship and the attributable risk of lung cancer per WLM were influenced by the total exposure accumulated, the rate of the accumulation of the exposure and the age at first exposure.

(iv) The effects of smoking and exposure to α-radiation from Rn daughters were nearly additive.

The substantially larger risk estimates resulting from the Czechoslovakian study when compared with those resulting from the study of the Colorado miners has been noted (see e.g. Sevc et al. 1988, Thomas et al. 1985 and BEIR 1980 p 325). The Czech estimates are consistent with the revised estimates from

the studies of Swedish and Canadian miners. There seem to be only two explanations for this difference namely that either the radon-daughter measurements in the U.S. mines have overestimated the exposures by a factor as large as 3 or that the higher dose rate in the U.S. mines has led to less risk per unit of cumulative exposure than the lower dose rates in other mines. (BEIR 1980 p 325). A recent re-evaluation of the early measurements of the lifetime exposures to radon and radon daughters of the U.S. uranium miners indicates that overestimation did occur (Schiager 1989).

As a result of these studies, the Task Group responsible for ICRP 50 gives an average value for the absolute risk coefficient which it regards as the best estimate for the radiogenic lung cancer risk among miners averaged over all age groups at the start of mining. The average is 10 cases per 10^6 PYR per WLM, with a probable range between 5 and 15 x 10^{-6} PYR^{-1} WLM^{-1}. The risk coefficient takes into account a minimum latency of 5 to 10 years and includes also the risk contribution from external γ-irradiation and inhaled long-lived radionuclides in mines.

For a working-year exposure at 4 WLM per year (approximately the occupational limit), the number of induced lung cancers may be estimated to be 1 to 2 cases per 1000 workers, over a period of 40 years after exposure.

However some authors (e.g. Cohen 1982, 1987) contend that the linear, no-threshold relationship over-estimates the risk from Rn-decay-products by a factor of at least 4.

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 were 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 were 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, 77% of the miners and 99% 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.

A more recent study (Saccomanno et al 1986) has re-analysed the earlier data. The conclusions were reached that the risk of lung cancer was significantly increased by radon exposure in cigarette smokers over that in non-smokers. However, whilst the contributions to the risk of age, smoking and mining were additive, there was no synergistic (i.e. multiplicative) interaction between the latter two. In addition, the authors concluded that radon decay product accumulations of less than 300 WLM were not carcinogenic in non-smokers. However, the issue of whether the risks of lung cancer due to exposure to radon decay products and to smoking are synergistic or merely additive is still debated, as Whittemore and McMillan (1983) found that a multiplicative model gave a better fit to the data than did the additive model.

It should be noted that a contrary view is expounded by Sterling (1983) who has reviewed data which suggests that smokers may develop less lung cancers than did non-smokers. A similar observation has been made from studies on beagle dogs and explained as arising from the increased mucus production (Gies et al. 1982). Notwithstanding these results, current radiation health practice accepts the possibility of a synergystic effect and aims at discouraging smoking amongst mine and mill workers.

SUMMARY

For most mining and milling operations in Australia, both planned and operational, the somatic and genetic risks associated with γ-radiation exposures are small, with exposures being well within the occupational limits.

The largest risks related to radiation exposure arise from the inhalation of radon daughter products and the inhalation or ingestion of radioactive dust. The risks are small at exposure levels below the occupational limits. In view of the possibility that cigarette smoking has a synergystic effect on lung cancer induction when combined with radon daughter inhalation, it should be particularly discouraged amongst workers involved in mining and milling of radioactive ores.

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