Evidence Presented by DSIR to the ROYAL COMMISSION on NUCLEAR POWER GENERATION

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E. BIOLOGICAL EFFECTS OF RADIATION ON MAN

This report forms one of sixteen related reports presented to the Royal Commission on Nuclear Power on 1.12.76.

Titles of the series are -

A. The Nuclear Power Industry in U.S.A.

B. The Nuclear Power Industry in Canada

C. The Nuclear Power Industry in Europe

D. The Nuclear Power Industry in Japan and Russia

* E. Biological Effects of Radiation on Man

F. Biological Hazards of Plutonium

G. Nuclear Power Plant Siting

H. Radioactive Releases from Nuclear Power Plants

I. Reactor Safety (including comments on criticisms of WASH 1400)

J. Safeguards

K. Radioactive Waste Management

L. Transportation of Fuel and Wastes

M. Nuclear Insurance

N. Decommissioning of Nuclear Power Facilities

O. Nuclear Energy in a Food Exporting Country

P. Electricity Demand, Substitution and Resources

* This report also exists as one of the Nuclear Input series, and is available from the Institute of Nuclear Sciences (DSIR) as NIP 4.

C O N T E N T S

C O N T E N T S (Ctd)

A. Effects of an Accidental Release of Radioactivity (single exposure)

A single dose of radiation over the whole body is almost certain to cause death within a year if this dose exceeds approximately 500 rem. Fetuses are more sensitive and can be killed or suffer malformations after doses as low as 50 rem if received at very early stages of development.

Irradiation of particular organs of the body may occur by inhalation of radioactive material, or by its deposition on particular areas. Severe respiratory illness may follow irradiation of the lung but is unlikely to be lethal. Other local effects include loss of hair, skin burns, and cataracts in the eye.

Single exposures to radiation also cause delayed effects which increase the risk of cancer in later life and may cause genetic defects in later generations. Whilst the number of prompt deaths caused by a major release of radioactivity depends on the number of people within a small "lethal" range of the release point, a considerably greater number of delayed cancers could result from small doses to large populations downwind of the release. The WASH-1400 report estimates the maximum number of delayed total cancers which could result from the very improbable "worst conceivable" accident to a 1000 MW(e) light water reactor as approximately 1500 per year for 30 years (45,000), representing a 10% increase of the normal cancer incidence in an exposed population of 10 million. The same fractional increase could be envisaged in New Zealand, but the total number could scarcely exceed a tenth of the 45,000.

B. Effects of Continued Exposure to Low Levels of Radiation (Received by plant operators and members of the public exposed to routine effluents)

The most important damaging effects of small annual doses of radiation such as might result from the routine operation of nuclear power reactors are (a) increased risk of cancer in later life, and (b) genetic effects caused in the reproductive cells of irradiated individuals, leading to defects appearing in later generations.

There is no such thing as a dose of radiation or quantity of absorbed radioactive material which is toxic or cancer inducing above a certain level, and harmless below it. Instead, radiation increases the risk or incidence of cancer in later life in much the same way as smoking cigarettes. Just as the risk of lung cancer increases with the number of cigarettes smoked, so does the incidence of leukaemia and other cancers in a large population increase with the average dose of radiation received by this population. Observations on the survivors of the nuclear bomb attacks on Japan show that the increased incidence of leukaemia 10 to 30 years after the attack was very approximately proportional to the dose received by those groups of people in whom it was observed. These people received large doses up to 400 rem. Information on the cancer inducing effects of the small doses of concern in the nuclear power industry is difficult if not impossible to obtain, for two reasons. Firstly, the cancers induced by radiation are no different from those which occur naturally, and the natural incidence greatly exceeds that expected of these small doses. Secondly, the incidence which might arise from these small doses is so small that statistically useful information would require millions of observations. For example, it is estimated that 0.1 mrem per year (10 times the average dose rate due to nuclear power in the USA in 1971) might cause one additional cancer death per year in a population of 60 million. This compares with the normal incidence of 90,000 fatal cancers per year. It may be that small doses below a threshold value have no effect at all. However, the opinion of national and international committees considering this question is that one should take a conservative view in estimating the effects of small doses and in setting radiation protection standards. For this reason it is assumed that the increased risk or incidence of cancer due to radiation is directly proportional to dose at all doses, without a threshold; and is independent of the dose rate at which it is received. On this basis, the observations from Japan and elsewhere lead to the conclusion that 1 rem (1000 mrem) of whole body radiation to a population of one million causes approximately 165 lethal cancers. Thus, an increased dose rate of 1 rem per year increses the incidence

of cancer by approximately 165 cases per year. At low doses or dose rates the risk is likely to be over-estimated on this basis.

Genetic effects of radiation have been observed and studied in detail in insects and mice, and must be assumed to occur in humans. Radiation increases the mutation rate in genes, and hence increases the incidence of the same type of inherited damaging diseases as those which occur naturally for various reasons. Mutant or changed genes are not permanently accumulated and passed on for ever to later generations, even if they are dominant. Instead they are slowly eliminated in successive generations. If this were not so, the fraction of children born with inherited diseases would increase in each generation, and this is not the case. In fact, about 6% of all live births have some form of inherited damaging disease. It is estimated that the incidence of about half of these might be affected by radiation in a manner which is directly proportional to the mutation rate. It is also estimated that the natural mutation rate might be doubled by a radiation dose of about 100 rem per generation. This is unlikely to under-estimate the risk because there was no significant increase in inherited diseases in the offspring of the Japanese bomb survivors who received doses around 100 rem. These two conclusions form the basis of the estimated numbers of genetic "defects" or inherited diseases shown in the following table, which might be expected as a result of an increased radiation dose rate to the whole population. This increased dose rate would have to be maintained for many generations before all these defects appear; about a seventh of the estimated increase might show up in the first generation. These estimates refer to obvious deformities such as hydroencephaly (abnormal head), polydactylism (extra fingers or toes), cleft palate, congenital dislocations, etc. For a given increase in the radiation rate to a large population, the total number of resulting obvious genetic defects per year is estimated to be about the same as the increased annual number of fatal cancers.

The table summarises estimated effects of background radiation and of the doses anticipated from nuclear power operation on the population of New Zealand.

Table - Estimated incidence in New Zealand with 3-million population of cancer and genetic defects resulting from world-wide routine operation of nuclear power facilities, in comparison with their normal incidence.

The radiation induced cancer incidence could be twice that estimated or two or more times lower. Radiation-induced genetic risks are probably over-estimated but are subject to wide uncertainty. They could be five times higher or 20 times lower than estimated.

Incidence of cancer -	
Present average death rate from cancer	4,500 per year
Estimated incidence due to natural background radiation at 100 mrem per year	50 " "
Estimated incidence in the general public (but excluding radiation workers) due to world-wide operation of nuclear power at 4300 GW in the year 2000 resulting in 0.3 mrem per year	0.15 " " (1 in 7 years)
Incidence of inherited damaging diseases (defects) -	
Present birth rate	60,000 per year
Estimated natural incidence of all forms of inherited damaging disease based on 6% of birth rate	3,600 " "
Estimated natural incidence of those types of defect which increase in proportion to the mutation rate	1,800 " "
Estimated incidence of defects due to natural background radiation at 100 mrem per year	54 " "
Estimated additional incidence of defects due to world-wide operation of nuclear power at 4300 GW, adding 0.3 mrem per year to the general public, but 1.5 mrem per year averaged over the whole N.Z. population including radiation workers. (Their dose will add to the total genetic load):	
(a) if continued indefinitely	0.8 per year
(b) in first generation after the increase in dose rate	4

C. Radiation Protection Standards

The International Commission on Radiological Protection (ICRP) recommends that people who are occupationally exposed to radiation in the course of their work should limit their radiation dose rate to less than 5000 mrem per year over the whole body, and to prescribed higher doses for individual organs. The estimates given above imply that if a radiation worker were to receive this maximum permissible dose rate all his life, his risk of death from cancer would be increased from its normal incidence of approximately 1 in 670 to very approximately 1 in 430. This estimate is subject to the uncertainties mentioned in the preceding table. This estimated risk is similar to that incurred in smoking about 10 cigarettes per day which is less than the risk of fatal injury encountered in several other trades or professions, and in fact the health record in the nuclear industry shows no sign of increased cancer incidence or other harmful effects. The same Commission recommends a tenth of this dose rate as the limit for any individual in the public. This is estimated to increase the normal cancer risk from 1 in 670 to 1 in 632 (subject to the uncertainties mentioned above) if this dose rate is received continuously.

Further recommendations of the ICRP relate to maximum permissible average dose rates (which determine genetic effects) and to maximum permissible intakes of radionuclides which may be inhaled or ingested. It has been suggested that the latter are too high for plutonium which may be inhaled and deposited in the lung in small "hot particles" of allegedly extreme toxicity, but these suggestions have not received support from national committees or from recent experiments designed to test them.

1. INTRODUCTION

This report is complementary to NIP report 3 on Radioactive Releases from Nuclear Power Plants,[1] and has two major objectives. Firstly, it summarises information on the biological hazards of radiation and their relation to radiation dose, and hence estimates the biological risks associated with nuclear power production from information given in NIP report 3. Secondly, it describes the basis and present status of radiation protection standards in the nuclear power industry.

There is extensive literature on the biological effects of radiation, but this cannot be reviewed in a summary report of this type. For more detailed reviews, the reader is referred to the following reports;

(a) Report of the United Nations Scientific Committee on the Effects of Atomic Radiation, 1972, which will be referred to as UNSCEAR.[2]

(b) Report of the Advisory Committee of the National Academy of Sciences on the Biological Effects of Ionising Radiations, 1972, to be referred to as the report of the BEIR committee.[3]

(c) Appendix VI of the U.S. Nuclear Regulatory Commission's Reactor Safety Study, 1975, to be referred to as WASH-1400, Appendix VI.[4]

2. UNITS OF RADIATION DOSE TO EXPRESS BIOLOGICAL EFFECTS (REM and RAD)

The biological effects discussed below are caused by all kinds of ionising radiation including penetrating gamma radiation which irradiates the whole body, beta rays (fast moving electrons) with typical ranges of 4 mm in tissue for

1 MeV electrons and 0.005 mm for the beta rays emitted by tritium, ... alpha rays, and protons (produced by absorption of neutrons), some of which are stopped by paper-thin materials. The effects caused by all these ionising radiations are similar in kind but differ in degree for the same absorbed dose, when this dose is expressed in rads. A rad is a measure of energy absorption resulting from ionising irradiation. One rad equals 100 ergs per gram, and is independent of the type of ionising radiation which dissipates this energy. Largely because alpha rays and protons dissipate their energy in a very small volume or length of track and so cause extensive damage in vital biological entities such as chromosomes, their biological effect per rad generally exceeds that of less densely ionising radiations such as gamma rays or high energy electrons. This relative biological effectiveness per rad (RBE) is expressed with X-radiation as a basis, so that the RBE for X-radiation of 250 kV energy is one, whilst that for alpha rays may exceed 10.

The rem (originally called roentgen equivalent man) is a unit of abosrbed dose in rads multiplied by RBE, which is designed to take account of this varying effectiveness. One rem has the same biological effect as one rad of X-rays of energy 250 kV. For alpha radiation, a rem is typically 0.1 rad, but the factor 0.1 varies both with the biological effect considered and type and energy of radiation. For most penetrating gamma rays which irradiate the whole body, 1 rem = 1 rad.

Doses are quoted in the literature either in rads or rems, and it is not always clear which is meant. For this reason, the units quoted in this report will be those actually

used in the article cited. In most cases the two units are approximately the same, but this is not the case for alpha rays and neutrons.

3. HARMFUL EFFECTS OF RADIATION

3.1 Somatic effects:

Somatic effects occur in the irradiated individual only and are to be distinguished from genetic effects, which are passed on to future generations. They may be prompt, i.e., occurring soon after irradiation, or delayed.

3.1.1 - Prompt effects -

The following are the most important effects which occur quickly or within a year after single doses of irradiation exceeding (generally)100 rads. They would not occur in routine operation of nuclear power plants but could arise in accidental situations.

Whole body irradiation causes death within a year for doses in the range 320 rads (probability of death 1%) to 750 rads (probability 99.99%).[5] The major cause is damage to the bone marrow which suppresses the formation of new blood cells. Larger doses cause death more rapidly, particularly if the cells in the gastro-intestinal tract are killed by doses exceeding three thousand rads.[6]

The principal early illness resulting from a single dose of radiation involves respiratory impairment caused by irradiation of the lung. The probability of this effect is estimated to range from 5% at 3000 rads to the lung, to 100% at 6000 rads.[5] Larger lung doses are likely to be fatal.

Other non-lethal effects include anorexia, nausea, and vomiting, resulting from whole body doses in the range 20 to

500 rads;[7] loss of hair two to three weeks after doses exceeding 300 rads to the hair follicles[8] and effects on the skin ranging from erythema (redness) followed by dry desquamation (scaling) equivalent to a first degree burn resulting from skin doses of several hundred rads, to blistering after doses of 1000 to 2000 rads, and ultimately to localised death of skin cells equivalent to third degree burns after doses exceeding 2000 rads.[8]

Cataracts in the eye leading to impairment of vision occur with high probability after eye doses of 1000 rads or more, and with lower probability after doses above 500 rads. Cataract formation involves a latent period ranging from six months to 35 years, depending on dose and dose rate.[9]

Decreased resistance to disease-producing micro-organisms has been observed in animals and probably occurs in man after doses of 200 to 600 rads. This is attributed to radiation-induced immunological impairment.[9]

Temporary sterility in human males occurs with low probability after doses to the gonads exceeding 10 rads and with near certainty after doses in the approximate range 100 to 600 rads.[10] Higher doses may lead to permanent sterility. Radiation induced sterility in women would be permanent if it occurred, and it has been estimated that approximately 2000 rads to the ovary may cause this effect in young women.[10]

Irradiation of the human fetus may lead to prenatal death, neonatal death, congenital malformations and growth retardation both before and after birth.[11] Fetuses are more sensitive to radiation than adults, especially in their early stages of growth. Occasional embryonic cells in experimental

mammals are killed by a few rads at certain stages in early life. It is estimated that the median lethal dose for the human fetus is 67 to 95 rads on the first day after conception, increasing to 285 to 380 rads after the 50th day. The BEIR committee conclude from records of the Japanese bomb survivors that the minimum dose to the fetus which subsequently leads to morphological alteration in the growing child is in excess of 25 to 50 rads.[21] In the WASH-1400 report, it is estimated that the smallest dose which might be lethal to the fetus is 9.5 rads on the first day after conception and 95 rads after the 50th day.[11]

3.1.2 - Delayed effects -

Sub-lethal doses of radiation are known to cause cancer in humans some years after irradiation; e.g., the incidence of leukaemia (blood cancer) in Japan rose in the 11 years after the nuclear bomb attack on Hiroshima from its normal level of approximately 20 cases per million of population per year, to more than 500 per million per year amongst those survivors whose whole body dose exceeded 200 rads.[12] A latent period elapses after irradiation before this increased incidence is observed. For leukaemia (which is the most readily detectable of radiation induced cancers), this latent period is typically two years, and is followed by a "plateau" of 25 years during which increased incidence occurs.[13] For cancers in the lung, breast, bone, gastro-intestinal tract, and most other regions of the body the latent period is estimated to be 15 years, followed by a "plateau" of 25 or 30 years.[14]

Cancers induced by radiation are indistinguishable from those which occur naturally in unirradiated populations but

their incidence increases with radiation dose as discussed in section 4. Cancer induction is the most important somatic effect of low levels of radiation, and in the opinion of the BEIR committee, it is the only somatic risk which needs to be taken into account in setting radiation protection standards for the general population.[20)]

3.2 Genetic effects:

Genetic effects are inherited (i.e., passed on to the offspring of irradiated individuals), and arise from mutations in the genes or damaging effects on the chromosomes in reproductive cells before fertilisation. Radiation is known to induce such effects in insects and experimental mammals such as mice, and must be presumed to do so in man although the evidence for this is not conclusive.[22)] The effects of mutations range from the obvious, such as albinism and hydro-encaphaly (abnormal head), to those which can be detected only by laboratory tests. Some effects are severe and produce life shortening or limitations in ability, whilst others are neither incapacitating nor disfiguring. These effects occur naturally from various causes. The effect of radiation is to increase the natural mutation rate and hence produce a greater incidence of defects which are no different in kind from those which are normally observed.[23)] Estimates of radiation-induced genetic risks in man are somewhat uncertain (see section 4.2).

4. RELATION OF RISK TO RADIATION DOSE FOR CANCER INDUCTION AND GENETIC EFFECTS

These two risks are the most important ones for occupational radiation workers in nuclear power facilities, who may be exposed in routine work either to single doses of radiation less than one or two rems over the whole body, or

to low continuing dose rates up to 5 rem per year.[20] The same applies to members of the public subjected to natural background radiation averaging 0.1 rem per year, and to increases above this caused by routine operation of nuclear facilities possibly amounting to 0.0003 rem per year by the year 2000.[1]

4.1 Cancer induction:

4.1.1 - Effects of high doses -

The cancer inducing effects of high sub-lethal doses of radiation are fairly well established from information both on the bomb survivors in Japan, and on people exposed to massive doses of radiation for medical purposes, mainly for treatment of ankylosing spondylitis.[2,3] For single whole-body doses which are mostly in the range 50 to 500 rads, and for medical X-ray doses to the spine and neighbouring organs given in repeated doses totalling typically 300 to 1500 rads, the subsequent increased incidence of cancer increases with radiation dose, and may be regarded in some cases as very approximately proportional to dose.[12] This increased incidence may be expressed as -

(a) the increase in annual incidence rate per rad, i.e., the increased average number of cancers per million people per year over the "plateau" period of elevated risk, per rad; or

(b) the total increased number per rad, i.e., the total increased incidence per million people per rad over the whole period of risk.

Both the United Nations Committee (UNSCEAR)[2] and the BEIR committee[3] reviewed the available information in 1972 with conclusions summarised in the following table of risk estimates for lethal cancers.

Table 1: Increased incidence rate of lethal cancer per million people aged 10 years or more per year, over "plateau" period, per rad.

	BEIR*[24]	UNSCEAR[25]
Leukaemia	1.0	0.7 to 2
Breast cancer	1.5	0.3 to 1
Lung cancer	1.3	0.6 to 3
Cancer of gastro-intestinal tract and stomach	1.0	
Bone cancer	0.2	2.5
All other cancers	1.0	
Total -	6.0	4.1 to 8.5

*These are "best estimate" values per rem. UNSCEAR quotes a range per rad. There is some difference between the two dose units in the case of neutron irradiation with RBE greater than 1, which contributed significantly to the Hiroshima exposures, but not those at Nagasaki.[26]

The data for leukaemia are the most reliable because of the short latent period before appearance of this cancer, and because of its low natural incidence which makes the radiation effect relatively more apparent.[24] Estimates for "all other cancers" shown in Table 1 are somewhat speculative. The cancer risk for fetuses irradiated in utero is much higher; it is estimated to be 25 per million per year per rem for all cancers including leukaemia, but the plateau period of elevated risk is thought to be only 10 years in this case as compared with 25 or more for adults.[24] Children aged up to nine years are also thought to be more susceptible to radiation-induced cancer, particularly if the radiation risk is estimated in relation to the natural incidence of cancer rather than in absolute terms. The BEIR committee estimated that 1 rem might increase the normal incidence of leukaemia

and of all other cancers by 5% and 2%, respectively, for children, and by 2% and 0.2%, respectively, for people aged over nine.[24] These conclusions lead to greatly increased estimates of excess cancer deaths as a result of irradiating children rather than adults, but no such increase is predicted by the "absolute" risk estimates discussed above. The BEIR committee pointed out that no data are available to establish which estimate is correct.[24]

It should be noted that Table 1 refers to lethal cancers only; for this reason, thyroid cancer has been omitted even though its risk is appreciable, because only 10% of such cancers are estimated to be lethal (see section 4.1.1).

Total risk estimates may be obtained from these numbers if the duration of the "plateau" period of elevated risk is known. The information available in 1970, and reviewed in 1972, relates only to the first 25 years since irradiation, and this includes a latent period of two years for leukaemia and about 15 years for other cancers.[24] For a 25-year plateau as estimated by the BEIR committee for leukaemia, the total risk of all forms of lethal cancer becomes 6 x 25 or 150 cases per million per rem from the BEIR risk estimates,[24] and this committee noted that the observed total excess mortality was -

(a) roughly 50 to 78 deaths per million per rem amongst the Japanese from 5 to 25 years after irradiation; and

(b) 92 to 165 deaths per million per rem amongst spondylitic patients in the first 27 years since irradiation.[27] The UNSCEAR committee concluded[28] that the total risk of lethal cancer in the first 25 years after exposure is 10 to 40 cases per million per rad for leukaemia, 6 to 20 for

breast cancer, 10 to 40 for lung cancer, and approximately 40 for other cancers, totalling 66 to 140 cases per million per rem in reasonable agreement with the BEIR estimate of 150 quoted above.

4.1.2 - Effect of low doses and low dose rates -

Risks at the low levels of dose and dose rate encountered occupationally (1 or 2 rem at any one time and less than 5 rem per year), and the much lower levels which the public might receive from nuclear power (less than 0.3% of the average natural background dose rate of 0.1 rem per year) may be estimated from these numbers only with considerable reservations. It is possible that doses below a minimum or "threshold" value may have no carcinogenic effect. Possible reasons for this include recovery from radiation given at low dose rates, and the suggestion that the latent period required for appearance of cancer after irradiation might increase at low doses beyond the life span of the irradiated individual. There are indications of this (though not conclusively proved) in the records of cancers induced in humans by absorption of radium,[29,30] and in experimental data on the induction of cancers in some tissues in animals.[29] The UNSCEAR committee noted that "It is a matter of speculation whether doses of the order of those received continuously from natural sources may have similar effects. Animal experiments suggest that the yield of tumours per unit dose should be lower at very low doses, except when the target tissue has a susceptibility to radiation induction of malignancies much higher than has been observed in man. Animal experiments also indicate that radiation given continuously or in several fractions is usually less carcinogenic than if

administered in a single dose within a short period of time. The numbers given in the preceding paragraph are therefore likely to be over-estimates of the risk of doses and dose rates such as are received from environmental sources".[28] The BEIR committee had similar reservations,[27] but nevertheless made a "most likely" estimate of the increased risk of cancer at low doses and dose rates by assuming that the risk estimates quoted above apply directly to these small doses, i.e., by assuming that the radiation induced incidence of cancer is directly proportional to dose without a threshold, and is independent of the dose rate or duration of exposure.[27,31] The National Council on Radiation Protection and Measurements (NCRP) in a report published in 1975[31] noted that "all national and international groups which have studied the problems of quantitative carcinogenic risk estimates have regarded the practice of linear extrapolation [i.e., the assumption of direct proportionality] as over-estimating the risk", but added the important proviso that this applies only when the risk at large doses is estimated from a rising and fairly linear portion of the dose-effect relation below those high doses which reduce the effectiveness owing to cell killing. The NCRP considered that the estimates given by the BEIR committee for low doses and dose rates should therefore be regarded as most likely estimates of the risk derived from linear extrapolation, rather than of <u>actual</u> risk at such doses and dose rates.[31]

These estimates are -

(a) 50 to 165 cancer deaths per million persons per rem in the first 25 to 27 years after irradiation, and

(b) 3000 to 15000 deaths per year amongst the 2×10^8

population of the USA receiving 0.17 rem per year continuously.[27] This range covers both the absolute and relative risk estimates mentioned above.

The latter estimate is equivalent to a total risk over all time of 88 to 440 cancer deaths per million persons per rem, with the most likely value in the range 147 to 205 with a mean of 165 deaths per million per rem. Because these estimates are based on direct proportionality to dose without regard to dose rate, they may be expressed in several ways as follows:

(a) 165 cancer deaths per million people, each receiving 1 rem in a single dose;

(b) 165 deaths per million man-rem;

(c) 16.5 deaths per year amongst a population of one million people receiving 0.1 rem per year continuously;

(d) for approximate purposes one could consider the estimate as equivalent to an increased incidence rate of six per million per year for 27 years (6 x 27 = 162).

Population doses in man-rems mean the summed products of dose in rems and number of people receiving this dose. One hundred man-rems may mean one rem to each of 100 people, 10 rems to each of 10 people, or 20 rems to two people plus 2 rems to 30.

Marley of the British National Radiological Protection Board reviewed the findings of the UNSCEAR committee in 1973,[32] and after making liberal (two-fold) allowances for the increased total risk to be expected over all time rather than 25 years after irradiation, he concluded that the risk from exposure to protracted doses of the order of occupational exposure or natural background might be reduced from those

estimated above on the basis of strict proportionality to dose to about 100 fatal malignancies per million man-rad. The Medical Research Council in the UK adopts the same risk estimate of 100 per million man-rem for protracted irradiation at the levels of exposure encountered by radiation workers.[74] The WASH-1400 report bases its "central" (most likely) estimate on the assumption of a reduction in effectiveness by up to five-fold for doses less than 10 rem, or for dose rates less than 1 rem per day.[33] These assumptions have been questioned,[34] and although the balance of evidence available at present appears to indicate reduced effectiveness at low doses and dose rates there is some evidence to the contrary in certain cases.[34,35] It has even been suggested that effectiveness might increase at small doses because of the existence of small fractions of the population of exceptionally high radiation sensitivity,[37] though the BEIR committee regarded the evidence put forward for this view in 1971 as insufficient to justify it.[100]

4.1.3 - Thyroid cancer -

Thyroid cancer is particularly important as a radiation hazard because inhaled radioactive iodine accumulates in the thyroid gland, and the dose to this gland is likely to exceed that in any other organ of the body after an accidental release of the radioactivity from a nuclear reactor. Irradiation of the thyroid gland causes delayed formation of thyroid nodules, about a third of which are malignant and the remainder benign.[15] Both can be treated medically, but it is estimated that about 10% of the malignant nodules might be fatal.[15,16]

It is a curious but widely quoted observation that internal thyroid irradiation resulting from iodine-131 is less effective per rad in inducing malignant nodules than external irradiation with X or γ rays,[16,17,18] even though the beta rays emitted by I-131 would not be expected to differ in biological effectiveness from external X-rays. The magnitude of this relative effectiveness factor has been estimated at 1/10 from data available in 1963,[18] and to range from 1/10 to 1/60 on the basis of data available in 1975[19] including animal experiments in which the factor ranged from 1/10 to 1/20.[19]

Risk estimates at high doses for malignant thyroid cancer induced by external penetrating radiation have been quoted by the UNSCEAR committee[25] at 1-2 and 2-4 cases per million per year per rad for adults and children, at 1.6 to 9.3 for children by the BEIR committee which concluded that children are more susceptible than adults to this effect,[17] at 8.1 for doses of 6.5 rads to children under 15, as calculated in a report of the U.S. Environmental Protection Agency[107] from data reported by Modan and others in 1974,[108] and at 4.3 both for children and adults by the WASH-1400 study which reviewed the data available up to 1975.[17] The WASH-1400 report estimates that this risk continues for a plateau period of 30 years after a latent period of 10 years, that the same risk factor may be used for radiation by I-131 absorbed in the thyroid if the dose as normally calculated in rads is divided by 10 to allow for the unexplained lower effectiveness of this radiation as noted above, and that the total risk of radiation induced thyroid cancer is 134 malignant

cancers per million thyroid rad for doses up to 15000 rads. An earlier estimate of Dolphin and Marley[16] in 1969 is 30 per million man-rad for X-irradiated adults and 100 per million for children.

4.1.4 - Lung cancer induced by inhalation of radon in uranium mines

In past years uranium miners were exposed to dangerous concentrations of the radioactive gas radon, which results from the decay of radium and, in turn, produces radioactive daughter products which are suspended in the air in uranium mines and may be inhaled. This led to an increased incidence of lung cancer, which was greatest amongst white miners who smoke tobacco.[38] This carcinogenic effect is mainly due to the short range alpha rays emitted by radon and its daughters. The sensitive sites for cancer production are the epithelial layers of the airways in the lung, and the dose these receive by alpha irradiation depends critically on their depth below the site of deposition of such radioactive substances.[39] For this and other technical reasons, it is difficult to express the lung cancer risk in terms of radiation dose to the lungs, and instead a direct relation has been established between the lung cancer risk in humans and the concentration of radon and its daughters in the air which they breathe.[40] If a man breathes air containing radon and its daughter products in equilibrium with it at a concentration of 100 pCi/l for 40 hours per week, and does this for a whole year, then he is said to have an exposure of 12 WLM or "working level months". By methods similar to those given above the lung cancer risk is estimated to be approximately 12.6 cancers per year per million smoking miners exposed to

4 WLM. If this increased incidence rate continues for 30 years, then the total risk is estimated to be 380 cancers per million smoking miners exposed to 4 WLM.[40]

A more technical but very readable account of the WLM concept and associated topics is given by Fry.[40] The maximum permissible concentration of radon and its daughters recommended by the International Commission on Radiological Protection for radiation workers is 30 pCi/l in unfiltered air, which is equivalent to 3.6 WLM per year if equilibrium is assumed between radon and its daughter products.[40,41] In USA, Australia, and some parts of Canada, the recommended maximum annual exposure for uranium miners is 4 WLM, whilst in France it is equivalent to 15 WLM, and in Euratom countries it is equivalent to 36 WLM.[40]

4.1.5 - Lung cancer risk from uranium tailings -

The separation of uranium from uranium ores leaves thorium-230 and radium-226 (the common form of radium) which results from decay of this thorium in the residues which remain after chemical processing. These residues have a bulk almost equal to that of the original ore and it has been common practice to dump this material into tailings piles near the uranium mill.[101] The radioactive gas radon, with a half-life of 3.8 days, results from the decay of radium in these piles and is partially evolved to the surrounding atmosphere. In the early years of uranium mining these tailings were used as land fill in housing construction sites, and the resulting concentration of radon in such houses exceeded the permissible levels in some cases.[101] This practice has now stopped, but unless the tailings are covered or buried

underground, they continue to evolve radon in local concentrations which are detectable above normal background levels within a kilometre of the pile.[101)]

Although it is very low, this increased concentration of radon adds to the dose received in the lungs of neighbouring populations, and very slightly increases their risk of lung cancer. If this risk is assumed to be directly proportional to dose without a threshold (as discussed above), then it has been calculated for the population densities in the USA around such piles that the resulting death rate is initially 0.0035 deaths per year for every uncovered pile of the size resulting from the uranium requirements of a 1000 megawatt (electrical) light water reactor for one year.[102,103)] However, this radon would be evolved for the hundreds of thousands of years for which thorium-230 decays with its half-life of 80,000 years. Consequently, if the world's population remains unchanged over this period, the final death toll due to a single year's tailings pile is calculated to be 394 deaths.[104)] The magnitude of this estimate has been questioned[102)] but it is important to understand the reasoning for such conclusions. Firstly, the assumption of direct proportionality between risk and dose is carried to its ultimate extreme. In consequence, for every 0.25 cancers calculated to arise within 80 kilometres of a large pile within 30 years, 200 more are calculated to occur beyond 80 km within 100 years, because the population density and man-rad population dose increases in this region, even although the radon concentration diminishes to near zero levels.[105)] Secondly, this small but finite increased risk

is summed over all succeeding generations until the thorium has decayed.

If all the readily available uranium in the Northern Hemisphere were mined in this way, the average individual in any one generation would have his risk of lung cancer increased by much less than 0.15% on the basis of this calculation, because the resulting increase in average atmospheric concentration of radon would not exceed this percentage.* However, if this small added risk is maintained over many thousands of generations the total becomes appreciable, so for this reason it may be considered necessary to cover or bury the tailings piles in order to reduce their emission of radon.

This is an extreme example of the implications of the linear risk assumption when taken to its limit, and it has been included for this reason as an illustration.

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* It has been estimated that a tailings pile resulting from the mining of 1,140 tonnes per year of U_3O_8 for 20 years (i.e. 22,800 tonnes) could evolve up to 20000 Ci per year of radon, if left dry and uncovered.[105] Readily available world resources of uranium were estimated at 866,000 tonnes in 1973,[106] so the maximum possible increased evolution of radon is 7.5×10^5 Ci per year. The normal evolution from the 50×10^{12} m^2 land area in the Northern Hemisphere, at an estimated average rate of 0.3×10^{-12} Ci per m^2 per sec,[105] is 0.5×10^9 Ci per year. 7.5×10^5 Ci per year is 0.15% of this.

4.2 Genetic effects:

Approximately 6% of all live births have some form of hereditary disease.[42,46] The two key steps in assessing radiation effects on these are -

(a) estimation of that fraction of the birth rate which results in diseases which follow simple rules of inheritance and therefore increase in proportion to the mutation rate, and

(b) estimation of how the existing or natural mutation rate is influenced by radiation. This is commonly expressed as a doubling dose per generation, i.e., the dose which doubles the natural mutuation rate, and which if continued for many generations will ultimately double the fraction of abnormalities mentioned in (a).

Both the BEIR[42] and UNSCEAR[43] committees sub-divide the 6% approximately as follows:

4.2.1 - Dominant, single gene diseases, all of which follow simple rules of inheritance. These are thought to include polydactylism (extra fingers or toes), Huntingdon's chorea (involuntary movement and mental deterioration), retinoblastoma (eye cancer), hydroencephaly (abnormal head), some forms of anaemia, muscular dystrophy, and haemophilia which is dominant in males.[44,47] BEIR[42] and UNSCEAR[43] estimate their total incidence as 1% of the birth rate, largely on the basis of a study in Northern Ireland,[44] but a later study in British Columbia[45] indicates 0.1%.

4.2.2 - Chromosomal and recessive diseases, of which only a very small fraction follow simple rules of inheritance. These are thought to include Down's syndrome (mongolism), Klinefelter's syndrome, Turner's syndrome, phenyl ketonuria (mental deficiency), Tay Sach's disease (blindness and early

death), albinism, cystic fibrosis and sickle cell anaemia.[44,47] Their total incidence is estimated to be 1% of the birth rate.[42]

4.2.3 - Congenital anomalies and constitutional and degenerative diseases with uncertain modes of transmission. These include diabetes mellitus, cleft palate and cleft lip, various forms of epilepsy and metal retardation, and congenital dislocations.[44,47] Their total incidence is estimated at 4% of the birth rate,[42] (UNSCEAR quotes 2% for a somewhat ill-defined group)[43] and the BEIR committee estimated that 5% to 50% of this 4% (i.e., 0.2% to 2%) might increase in proportion to the mutation rate.[42] A more recent estimate by Newcombe was 0%.[46]

Summarising, the overall fraction of live births which have heredity damaging diseases behaving as though they were dominant and occurring in proportion to the mutation rate was estimated by the BEIR committee[42] to range from -

(a) all of the 1% in 4.2.1 plus none of the 1% in 4.2.2, plus 0.2% in 4.2.3 (i.e., 1.2% total), to

(b) 1% in 4.2.1, plus 2% in 4.2.3 (i.e., 3% total).

If the results of the British Columbia study are accepted for 4.2.1. and combined with the BEIR committee's lowest estimate for 4.2.3, then the overall fraction could be as low as -

(c) 0.1% in 4.2.1, plus 0.2% in 4.2.3 (0.3% total).

UNSCEAR assumed 3% for computational purposes[43] and this estimate will be assumed in what follows. It could be regarded as an upper limit.

Radiation affects the natural mutation rate by a fraction which appears to be directly proportional to dose without a threshold for single gene mutations in the fruit fly,

Drosophila,[48] but decreases with decreasing dose rate in the mouse.[48] The doubling dose is estimated to be approximately 100 rads for male mice irradiated at occupational or lower dose rates[43] (as compared with approximately 30 rads for high acute X-ray doses[43]), and the UNSCEAR committee considered this may apply to man although no data on man are available.[43] For various reasons, including the lack of observable genetic effects on the bomb survivors in Japan after doses averaging 100 rad, the BEIR committee concluded that the doubling dose for man is in the range 20 to 200 rem to the male gonads.[49]

If one adopts 100 rem as the most realistic value for the doubling dose (also assumed in the WASH-1400 report[47]), this means that a dose of 1 rem per generation (approximately 30 years of reproductive life) will increase the natural mutation rate by 1%. Radiation induced genetic effects may then be estimated as follows:

4.2.4 - Estimation of radiation induced genetic hazards -

4.2.4.1 - Continued irradiation, leading to a new equilibrium - The birth rate in USA is approximately 15,000 per million of population per year. 3% of these births have inherited disease of a type whose incidence is affected by radiation. If the whole population receives additional radiation at the rate of 1 rem in 30 years (0.033 rem per year), then the resultant increased incidence of inherited disease will be 1% of this 3%, or 4.5 per year, when a new equilibrium is reached after many generations. For an increased radiation rate of 0.1 rem per year (equivalent to doubling the average natural background dose rate) the corresponding estimated number is 13.5 per year per million of population of which approximately a seventh (i.e. a fifth

of the dominant diseases in 4.2.1, plus a tenth of those in 4.2.2) might appear in the first generation after increasing the dose rate.[42]

For New Zealand with a population of three million, and birth rate of 20,000 per million, the corresponding numbers for an increase of 0.1 rem per year are (3 x 20,000 x 3% x 1% x 30 x 0.1), or 54 additional defects per year at equilibrium, and 54/7 per year for 30 years (i.e., 230) in the first generation. These estimates would be increased five-fold if the doubling dose is taken to be 20 rem per generation (the lower extreme of the BEIR estimate), and reduced 20-fold if the mutation controlled component of natural incidence is taken as 0.3% instead of 3%, together with a doubling dose of 200 rem per generation (the other extreme of the BEIR estimates).

4.2.4.2 - Effect of a single dose of radiation to one generation - In this case, the dominant fraction of inherited defects would be expected to increase in the first generation and subsequently decline to its original level. A single dose to a population of one million would affect those born in the next generation of 30 years (approximately 30 x 15,000 or 450,000) and their descendants, but not for ever, because even the dominant mutants in these 450,000 births are eliminated at a rate which is thought to be approximately 20% per generation.[47] In this case, the total number of such additional defects which would appear in the following generations because of a single dose to the initial population is the same as the additional number occurring in every generation in the equilibrium case considered above, when the

same dose per generation is given continuously.* From the estimates given above for defects which behave as though they were dominant and normally constitute 3% of the birth rate, the effect of 1 rem per generation would be to cause 3% x 1% of defects in the 450,000 births every generation, if this dose rate of 1 rem per generation were continued to equilibrium. The same number, namely, 135, is expected over all time for a single dose of 1 rem to the million population we are considering. Of these, about a fifth (if the elimination rate is 20%) is expected in the first generation after irradiation, and the remainder in the next 10 or more generations. If the birth rate is 20,000 per million of population per year as in New Zealand, then the above estimate becomes 180 per million man-rem instead of 135. Cohen[51] suggests 160 defects per million man-rem, and the WASH-1400 report estimates 54 to 124 per million man-rem made up of 40 dominant defects, 8 to 78 multifactorial disorders and six disorders due to chromosomal abberations.[47]

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*If x is the number of dominant mutant sites in a population which result from a mutation rate of m sites per generation, and a fraction s of these is eliminated per generation, then at equilibrium $m = xs$ and $x = m/s$. If continuous radiation doubles m, then x rises at equilibrium to $2 m/s$, i.e. x increases by m/s. Now suppose that enough radiation is given at one time to double m for one generation only. The resulting additional number of mutants produced over all successive generations is then $\Delta x = m + m(1-s) + m(1-s)^2 + \ldots = m/s$, which is the same as the increase occurring in every generation in the equilibrium case.

4.2.5 - Spontaneous abortions -

Approximately 45% of all conceptions end as spontaneous abortions, although only about a third of these are recognised as such.[107] The BEIR committee estimate that 55,000 recognised abortions per million live births are due to types of chromosome damage which could be increased by radiation, and they also estimate that 5 rem per generation might increase this 55,000 by 520.[42] Thus, if 1 rem per year is given continuously to a population of one million with a birth rate of 20,000 per year, then the added dose per generation is 30 rem and the estimated additional number of recognised spontaneous abortions so caused per year is 62 ($520 \times 30/5 \times 20{,}000/10^6$).

4.3 Summary of risk estimates:

The following estimates are based on the assumption, discussed above, that risk is directly proportional to dose without a threshold, and is independent of dose rate for carcinogenic effects. For the reasons given above they are likely to be over-estimates of carcinogenic risk at low doses and dose rates.

Table 2 - Risk Estimates

Cancer	Best estimate	Maximum range
All forms of lethal cancer deaths per million man rem	165	88 to 440
Thyroid cancers per million thyroid-rem in children and adults (10% fatal)		
(a) external radiation	134	uncertain
(b) I-131 radiation	13.4	"
Genetic		
Increased number per year of inherited damaging diseases resulting from 1 rem per year to a population of 1 million, with a birth rate of 20,000 per year	180	9 to 900
Number expected in first generation of births after an increase of 1 rem per year (1/7 of 180 per year for 30 years)	770	40 to 4000
Total number of inherited damaging diseases appearing in subsequent generations after a single dose of 1 million man rem or 1 rem to a million people	180	9 to 900
Increased number per year of recognised spontaneous abortions resulting from 1 rem per year to a population of one million, with a birth rate of 20,000 per year	60	uncertain

4.4 Effects of an accidental release of radioactivity:

These risk estimates together with those quoted above for acute effects may be used to estimate the effects of an accidental release of radioactivity if the weather conditions which determine dispersion are known, together with the population density downwind. An example of the likely range of various effects is given in NIP report 3.[1] The effects of a release depend so much on local conditions that generalisations are of little value, but it is worth noting that the number of early or prompt deaths depends on the number of persons within a small range of the reactor who receive more than a fairly well defined whole body dose

and could therefore be the same for a New Zealand site as for an American site as considered in the WASH-1400 report, depending on the size of the exclusion area. In contrast, the number of delayed cancers depends mainly on the population dose at considerable distances from the reactor and is limited in New Zealand by its size and population. WASH-1400 estimates the maximum number of delayed fatal cancers resulting from the worst conceivable accident to a 1000 MW(e) light water reactor as 1500 per year for 30 years, i.e., 45,000, representing a 10% increase in the normal cancer incidence amongst an exposed population of 10 million. The same fractional increase could be envisaged in New Zealand, but the numbers could scarcely exceed a tenth of those quoted.

4.5 Effects of routine operation of nuclear power facilities in New Zealand:

The following table summarises the situation, as estimated from the risk estimates given in Table 2.

Table 3 - Estimated incidence in New Zealand of cancer and genetic defects resulting from world-wide routine operation of nuclear power facilities, in comparison with their normal incidence.

These are based on the best estimates given in Table 2 and are therefore likely to be over-estimates of radiation-induced cancer incidence. The uncertainties associated with the estimates are also quoted in Table 2. A population of three million is assumed.

Incidence of cancer -	
Present average death rate from cancer[50]	4,500 per year
Estimated incidence due to natural background radiation at 0.1 rem per year	50 " "
Estimated incidence in the general public (but excluding radiation workers), due to world-wide operation of nuclear power at 4300 GW in the year 2000 resulting in 0.3 mrem per year[1]	0.15 " " (1 in 7 years)
Incidence of inherited damaging diseases (defects) -	
Present birth rate[50]	60,000 per year
Estimated natural incidence of all forms of inherited damaging disease based on 6% of birth rate	3,600 " "
Estimated natural incidence of those types of defect which increase in proportion to the mutation rate	1,800 " "
Estimated incidence of defects due to natural background radiation (100 mrem per year)	54 " "
Estimated additional incidence of defects due to world-wide operation of nuclear power at 4300 GW, adding 0.3 mrem per year to the general public, but 1.5 mrem per year averaged over the whole N.Z. population including radiation workers.[1] (Their dose will add to the total genetic load):	
(a) if continued indefinitely	0.8 " "
(b) in first generation after the increase in dose rate	4

5. RADIATION PROTECTION STANDARDS

The International Commission of Radiological Protection (ICRP) was established in 1928 as an independent international body to make recommendations on radiation protection standards and dose limits. In its present form it consists of 12 internationally recognised experts in many fields related to radiation.[52,109] Its function is advisory. Many countries have similar national committees, such as the National Research Council on Radiation Protection and Measurements (NCRP) in the USA, and a committee of the Medical Research Council in the UK. Recommendations made by these various committees are

in fairly close agreement. Regulations (including dose limits) are set in each country by appropriate governments and are generally based on these recommendations. In New Zealand these are embodied in the Radiation Protection Act 1965, and the Radiation Protection Regulations 1973, administered by the Department of Health. Dose limits which are more restrictive than national requirements have been set in some cases by regulatory authorities attached to nuclear power facilities.

Originally, the basis for these standards was the protection of radiation workers in medical and other occupations. A maximum permissible dose rate of 0.3 roentgens per week (approximately equivalent to a whole body dose rate of 0.3 rads per week) was recommended for this purpose in 1949, because lower dose rates did not appear to have observable damaging effects.[53] In 1958, the ICRP recognised -

(a) the possibility that small doses and dose rates might entail some risk (i.e., that a "safe" or threshold dose may not exist);

(b) the need to protect the public because of the growing nuclear industry with consequent small dose increments to the public; and

(c) the need to limit the population averaged dose to the gonads because of genetic effects which ultimately affect the whole population.[54] Maximum permissible doses for radiation workers were reduced to 5 rem per year to the whole body or its most sensitive organs (blood forming organs); the dose limit recommended for any individual in the general public was set at 10% of these values, and it was proposed that the

average genetic dose per generation be limited to 5 rem. In 1965, the ICRP ratified these recommendations and extended them somewhat.[55] The committee stressed the need to avoid unnecessary exposure and to keep all doses "as low as is readily achievable, economic and social considerations being taken into account". The major aim of the 1965 proposals was to attempt to balance risks against benefits. Clearly it is justifiable for a radiation worker to accept a higher dose than a member of the public because of the benefits of his work. The aim is to limit the associated risk so that it is no higher than that which is regarded as acceptable in other trades or professions. Also, it is desirable to reduce the risk to the public from nuclear power facilities, but not to the point where the cost outweighs the benefits of an electricity supply. For example, Cohen[56] estimated that the cost of reducing the emission of routine effluents from American nuclear power stations to 1% of ICRP recommendations is equivalent to very approximately $15 million per life saved. One would certainly question any further expenditure. In 1973, the ICRP published some guide lines to assist in interpreting the "as low as practical" recommendations.[57] These are based on the assumption, discussed above, that radiation-induced damage or risk is directly proportional to dose without a threshold. Whilst this relation, together with risk estimates such as those given in Table 2, may be used to estimate data such as the maximum numbers of cancers avoided by a given reduction in population dose (in man-rems), the committee is careful to point out that such numbers may be over-estimates because of the assumption made.[57]

The following table summarises the recommendations of the ICRP. For convenience in presentation, some related recommendations and requirements are included and will be discussed below.

Table 4 - Summary of radiation protection standards

A. Recommendations of ICRP, made in 1965[55)]

Organ or tissue	Maximum permissible dose for adults exposed in the course of their work (rems per year)*	Dose limits for members of the public (rems per year)
Gonads, red bone-marrow	5	0.5
Skin, bone, thyroid	30	3†
Hands and forearms, feet and ankles	75	7.5
Other single organs	15	1.5

† 1.5 rem per year to the thyroid of children up to 16 years of age

* up to half of these yearly limits in any one quarter year.

B. Additional recommendations of ICRP, made or ratified in 1965[55)]

(a) Keep doses as low as is readily achievable, economic and social considerations being taken into account.

(b) Average genetically significant dose to be less than 5 rem per generation (approximately equivalent to 0.17 rem per year).

(c) Average dose to the "critical" group of the population near a radiation source to be less than 0.5 rem per year.

C. Additional requirements of national regulatory authorities

(a) USAEC in 1971 interpreted "as low as practical" for effluents from water reactors as "contributing less than 0.005 rem per year to any individual in the public, i.e. less than 1% of ICRP recommendations.[58)]"

(b) Similar requirementswere made for the Canadian power reactors at Pickering in 1972.[59)]

(c) The average dose to the whole population in the UK resulting from nuclear effluents is to be less than 1 rad in 30 years.[60)]

5.1 Risk to individuals resulting from permissible doses:

Estimates of individual risk may be obtained from the data in Table 2 as follows:- The death rate from cancer in New Zealand is 1500 cases per year per million of population.[50] The estimated additional incidence due to irradiation at 5 rem per year (the maximum permissible dose rate for radiation workers) is 5 x 165 or 825 cases per million per year, bringing the estimated total to 2325. Thus, an individual radiation worker's probability of death from cancer is normally one in 670 ($10^6/1500$) and this is increased to one in 430 ($10^6/2325$) by irradiation at 5 rem per year if he receives this added dose rate all his life. The corresponding change in risk for a member of the public receiving 0.5 rem per year continuously is from one in 670 to one in 632 ($10^6/1582$).

These estimated risks are appreciable; they are not zero as would result from a toxic agent at doses below its threshold. To put them in perspective, the following remarks may be helpful.

Firstly, they are based on an assumed linear relation between risk and radiation dose, and are therefore likely to be over-estimates.

Secondly, a very approximate comparison might be made with the cancer risk due to smoking cigarettes. From information published by the Royal College of Physicians in

1971[61]) it may be estimated that the increased incidence of lung-cancer amongst males aged 35 to 64 due to smoking 20 cigarettes per day is approximately 11,200 per year in a population of 7.5 million.* This is equivalent to 1500 per year per million. If the lung cancer risk is approximately proportional to the number of cigarettes smoked, as is indicated by the available evidence,[62]) then the cancer risk estimated for 0.5 rem per year (82 per million per year) is approximately equal to that due to smoking one cigarette per day (20 x 82/1500). Fry estimated that the risk associated with smoking one to two cigarettes per day is 400 lung cancer cases per million per year.[40]) Thus, the radiation worker receiving 5 rem per year takes a risk which might be regarded as comparable with that due to smoking 5 to 10 cigarettes per day, whilst the member of the public recieving 0.5 rem per year takes a ten-fold lower risk.

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*The following numbers are taken from reference (61). The estimated death rate in the UK in 1968 amongst males aged 35-64 was 99,082. Of these, 12,494 were due to lung cancer, 6,492 were due to chronic bronchitis, and 31,013 to coronary heart disease. The report attributes to cigarette smoking 90% of lung cancer deaths (11,200), 75% of those due to chronic bronchitis (4,800) and 25% of those resulting from coronary heart disease (7,800). In New Zealand, about 30% of males are aged 35-64.[50]) The population of males in the UK in this age group may therefore be estimated as 0.5 x 0.3 x 50 million, or 7.5 million. On p.145 of ref. (61) it is shown that the sample of doctors on which this estimate was based contained 43% non-smokers, 16% smoking 1-14 cigarettes per day, 24% smoking 15-25 per day, and 17% smoking 25+ per day. As an average for smokers, take 20 per day. We conclude: the risk from smoking 20 cigarettes per day among males aged 35-64 is increased lung cancer incidence of 11,200 in 7.5 million, i.e., 1,500 per million.

Thirdly, the recommended dose limits are not based on such risk estimates, for they were not available when the limits were suggested. Instead they are based partly on experience indicating a lack of adverse effects amongst radiation workers, and partly on comparisons with existing natural background radiation rates which average 0.1 rem per year but vary considerably.[54,55] The following two figures illustrate these points. The first is extracted from a USA survey reported by the USAEC in 1971[63] and shows a comparison of death rates from all causes in the nuclear industry with those published by the U.S. National Safety Council (NSC) for USA industry as a whole in the same period. The second is taken from a report of the Argonne National Laboratory published in 1973,[64] and shows how the malignant cancer rate in the 50 States of USA varies with the average natural background level of radiation in each State.

However, neither of these figures indicates any adverse effect of radiation at dose rates which in the former case are those encountered by radiation workers, and in the latter case covered a range of dose rates up to half the maximum level permitted for members of the public.

The major conclusion from these studies is that if radiation has any effect at these dose rates, it is small in comparison with other natural risks and with those normally arising in industry. These other natural risks have not been identified in the American cancer study, though they were sought with some care.[64]

Similarly, a search for genetic effects of radiation has been reported in areas of India where the natural background

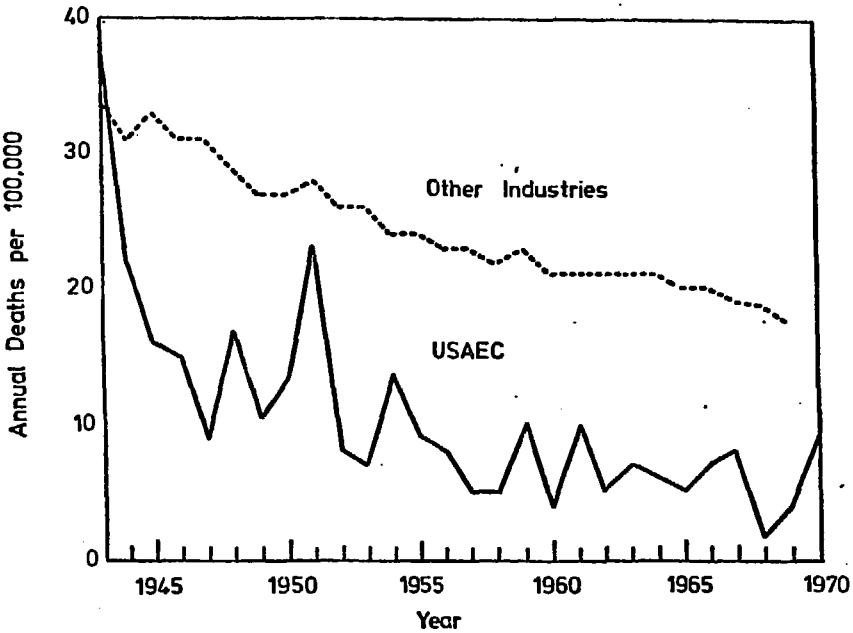

Fig. 1 - Death rates in USAEC and in those industries in the USA reviewed by the U.S. National Safety Council (NSC) from ref. (63).

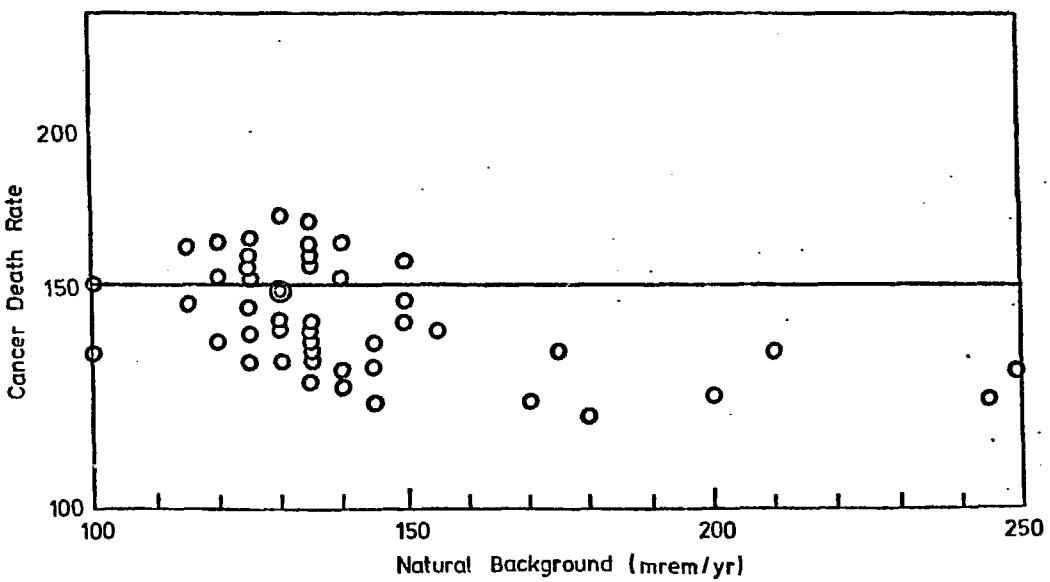

Fig. 2 - The relationship between cancer mortality rates (deaths per 100,000 population) for the US white population, and the average natural background for each state. The horizontal line and open circle indicate the rate and background for the US as a whole. The data are for the period 1950-67. From ref. (64).

dose rate varies from 0.1 to over 2 rem per year.[65] Surveys of fertility index, sex ratio among offspring, infant mortality rates, pregnancy terminations, multiple births, and gross abnormalities were conducted. No statistically significant differences in the average mean values of the data for differently exposed groups were reported. However, the authors suggested that the total loss of offspring (low fertility index and high infant mortality rates) in one group of nine couples receiving over 2 rem per year was somewhat higher than in other groups.[65] These data also indicate that risks due to radiation at these dose rates are less than those due to other natural causes.

A very recent study[108] indicates slightly increased incidence of Down's syndrome and related abnormalities in the inhabitants of Kerala in South India where the background exposure rate is 1,500 to 3,000 mr per year (1 mr approximately equals 1 mrad). This is attributed to increased radiation-induced genetic damage. The observation is difficult to understand as the incidence of the chromosome anomaly leading to Down's syndrome is not considered likely to be affected by radiation (section 4.2.2).

5.2 Average risk over the whole population:

The average dose or dose rate to any group of people near a nuclear facility will generally be very much less than the maximum to any individual; e.g., the average dose to the 100,000 residents within 80 km of a power reactor at Humboldt Bay due to its gaseous emissions was estimated to be 0.0011 rad per year in 1969, as compared with the maximum to any individual remaining at the site boundary of 0.15 rad per year.[66] For this reason, the ICRP emphasises

individual rather than average limits. However, there may be local critical groups which receive unusually high doses, and the ICRP recommends that when such groups can be identified and have reasonably homogeneous exposures then the mean dose to such groups should be limited to 0.5 rem per year instead of limiting the maximum dose to any individual within them.[55] The reason for this is the difficulty of determining individual maximum doses. An example concerns those residents of Wales who eat laverbread. This is made from seaweed which, in the vicinity of Windscale, is contaminated by the radionuclide ruthenium-106 discharged as a routine effluent from a nuclear reprocessing plant.[67] In 1967, a critical group of about 100 such residents was identified. These consumed unusually large amounts of laverbread, and thus incurred annual doses to their gastro-intestinal tracts which averaged somewhat less than the permissible limit of 1.5 rem. The average dose to the 30,000 or more inhabitants of the area in which laverbread is sold was very much less than this.[67]

Genetic effects of radiation are determined by the average 'genetically significant" dose to the gonads, multiplied by the mean age of child bearing, which is taken to be 30 years.[55] This is somewhat less than the average of individual gonad doses because the genetically significant dose is weighted according to the expected number of children conceived after exposure,[55] but more important is the fact that this average should include the whole population including radiation workers. The ICRP recommends that this average dose should be kept to the minimum consistent with necessity, and should certainly not exceed 5 rem from all sources additional

to the dose from natural background radiation and medical exposures.[55] This is approximately equivalent to 5 rem in 30 years or 0.17 rem per year which is also the whole body exposure rate recommended by the BEIR committee as a permissible average over the whole population, being one-third of the dose limit recommended for any individual.[68] The corresponding risks estimated from the data in Table 2 are 0.17 x 180 or 30 additional radiation-induced inherited damaging diseases per million of population per year, and approximately the same increased members per year of lethal cancer. Gofman and Tamplin objected to these recommendations, regarding them as too high for a permissible <u>average</u>, and claimed that if nuclear power were permitted to increase the natural dose rate by 0.17 rem per year to the two hundred million inhabitants of the USA, this might cause additional cancer death rates in the USA which they estimated initially at 16,000 per year,[69] later at 32,000 per year,[70] and finally at 104,000 per year.[71,72] These estimates correspond to 480,960, and 3,000 cancer deaths per million man-rem which considerably exceed the BEIR estimates summarised in Table 2, and the BEIR committee has given reasons for rejecting Gofman and Tamplin's estimates.[73] However, their objections to a "high" permissible average dose rate still remain in principle. In fact, the effective limit to average dose rates is likely to be set by the individual limits considered above, and in the case of nuclear power facilities it is further reduced by the recent requirements of most regulatory authorities which limit individual doses to less than 1% of ICRP recommendations.[58,59] It is estimated that the additional world-wide average dose rate incurred by all

aspects of nuclear operation up to the year 2000 will not exceed approximately 0.0003 rem per year to the general public (excluding radiation workers), and approximately 0.003 rem per year (0.0015 rem/y in New Zealand) if radiation workers are included in the average.[1)]

5.3 Inhalation and ingestion of radionuclides:

Protection standards for internally deposited radioactive substances are set as follows:

(a) The ICRP[41,55)] recommends a maximum permissible annual dose (MPAD) for each of the more critical organs of the body. This dose is intended to carry the same risk as that resulting from a dose of 5 rem to the whole body or blood forming organs. These doses are listed in Table 4; for most organs the MPAD is 15 rem. The distinction between rem and rad is important here because many internally absorbed radionuclides emit short range alpha or beta rays with a relative biological effectiveness which differs considerably from that of penetrating X- or γ-rays.

(b) A maximum permissible burden is set for each radionuclide. If maintained continuously in any organ such as the lung, this will give an annual dose to that organ corresponding to (a) (e.g. 15 rem to the lung).

(c) Taking account of the chemical form of each nuclide, its route of entry into the body and thence to the organ and its rate of decay or elimination when abosrbed, derived limits are set for the maximum permissible annual intake (MPAI) in air or in water.

(d) Corresponding derived limits may be set for the maximum permissible concentrations in air or water which will lead to (c) and thence to (a).

(e) These limits are then adjusted for the general public or critical groups within it, usually by lowering them ten-fold or by 20-fold for the thyroid gland in children under 16 years of age.

This procedure enables one to apply information on human risk relating to one or a few radionuclides to others for which such information is lacking. It involves several uncertainties, particularly relating to (a) above, because information on risk in relation to dose in individual organs is limited, and to (c) above, because of the many factors involved. If the risk of inducing cancer in any one cell is directly proportional to the dose it receives (as implied by the linear assumption discussed above), then one would expect the total risk of inducing cancer in a given organ to be proportional to the number of cells within it multiplied by the average dose for the organ. However, cells vary in radiation sensitivity, and the available risk estimates in terms of cancer incidence per million organ-rem (analogous to the risk per million man-rem discussed in section 4.1) can only be regarded as very approximate. The Medical Research Council in the UK reviewed the information up to 1975[74] and suggested the following risk estimates for comparison with MPAD values recommended by the ICRP:

Table 5: Radiation-induced cancer risks in various organs

Organ	Risk estimate (cancers per million organ-rem)	MPAD set by ICRP (rem)	Relative risk for one MPAD (cancers per million organs)
Bone	5	30	150
Gut	20	15	300
Liver	20	15	300
Lung	25	15	375
Whole body	100	5	500

The MRC regards its risk estimates for individual organs as conservative, which implies that the permissible doses recommended by the ICRP for individual organs are also conservative relative to that recommended for the whole body.

In 1959, the ICRP published a list of recommended maximum permissible concentrations in air and water for numerous radionuclides, together with other information on their biological properties.[41] This forms the basis of health physics regulations designed to ensure that permissible doses are not exceeded. However, considerable supplementary information is required to identify critical groups within the population and the pathways of exposure for some radionuclides released in the nuclear power industry.

In some cases direct relation between risk and exposure has been obtained without the estimation of radiation dose. One such is the cancer risk due to inhalation of radon discussed in section 4.1.2. In this case the recommended permissible exposure of 4 WLM per year for uranium miners confers an estimated cancer risk of 380 per million miners per year, which corresponds approximately with that estimated from Table 2 for a 5 rem whole-body dose per year (825 per million per year).

6. OBJECTIONS TO THE ICRP PROTECTION STANDARDS

Gofman and Tamplin's objections to the recommended permissible average dose rate of 0.17 rem per year have been noted above (section 6.2). Objections on other grounds have been raised by Sternglass, and by Gofman and Tamplin, as discussed below.

6.1 Infant mortality attributed to genetic effects of radiation (Sternglass):

In 1969, Dr Sternglass claimed to have found a causal relationship between the deposition of strontium-90 from fallout in the USA from nuclear weapon testing, and increased infant mortality of children in their first year of life.[75,76,77] He suggested that this was an extremely sensitive genetic effect of radiation from strontium-90 causing the death of 400,000 children in USA as a result of nuclear weapon testing, and later alleged that similar effects occur in the vicinity of nuclear power plants.[78]

The BEIR committee in the USA commented on this claim as follows:[79]

"The evidence assembled by Sternglass has been critically reviewed by Lindop and Rotblat[80] and by Tompkins and Brown.[81] It is clear that the correlations presented in support of the hypothesis depend on arbitrary selection of data supporting the hypothesis and ignoring of those that do not. In several regards, the data used by Sternglass appear to be in error. One of the most vital assumptions in the model - that without the atomic tests the infant mortality rate would have continued to fall in a geometrically linear fashion - is without basis either in theory or in observation of trends in other countries and other times. The doses of strontium-90 used in the experiments referred to by Sternglass as supporting evidence were of the order of 100,000 times greater than those received by humans from all the atomic tests and were associated with extremely small differences in infant mortality (8.7% in the irradiated vs. 7.5% in the control mice)."

"In short, there is at the present time no convincing evidence that the low levels of radiation in question are

associated with increased risk of mortality in infancy. Hence, for the purposes of this report, no estimate of risks are considered to be applicable."

6.2 Hot particles of alpha emitting radionuclides (Gofman and Tamplin):

This objection relates particularly to finely dispersed particles of plutonium lodged in the lung, but it applies to all alpha emitting radionuclides and will therefore be considered in this report. All other aspects of plutonium are considered in a separate report.[83]

The inhalation of aerosols of finely dispersed particles containing radioactive substances causes these to lodge initially in the linings of the airways, but in the course of time these migrate to the deep lung.[84] Because plutonium and other alpha emitting radionuclides are long-lived, the ICRP estimates their risk in terms of an average dose to the whole mass of the lung, obtained by dividing the mass of the lung by the total energy deposited within it by alpha rays, and multiplying by an appropriate biological effectiveness factor to obtain the dose in rems.[74] The cancer risk is then evaluated by assuming this to be proportional to dose, as mentioned in sections 4.1 and 6.3. This procedure is likely to over-estimate the risk due to small average doses for reasons given above.

Tamplin and Cochran[85,86] suggested that this approach may under-estimate the risk of large local doses, which

certainly occur within the alpha ray range (typically 0.04 mm) of each particle. In 1967, Albert and co-workers showed that localised irradiation of hair follicles in the rat caused cancer with a frequency of about one in 2,000 follicles irradiated to 1,000 rem or more.[87] Tamplin and Cochran suggested the same may occur in the much smaller volumes of lung which receive this dose adjacent to alpha emitting particles, and thus concluded that any "hot particle" containing 0.07 pCi or more of plutonium (or any other alpha emitter), might have a probability of one in 2,000 of causing cancer at this site. Such particles cause local irradiation at doses exceeding 1,000 rem per year. This reasoning led them to suggest that presently accepted ICRP standards for the maximum permissible concentration of plutonium in air and corresponding maximum permissible burdens in the lung (16,000 pCi) are too high by factors of approximately 100,000 for such hot particles.[85,86] It was claimed that this suggestion draws support both from observations on a single plutonium-239 particle (5,000 pCi) embedded in the skin of a plutonium worker, which after four years caused tissue damage described as pre-cancerous,[88] and from an alleged relationship between entry of plutonium from a leaking carboy into a wound and subsequent development of cancer in soft tissue.[85,89]

The reasoning and evidence for this suggestion have been carefully examined by the British Medical Research Council in 1975,[74] by Dolphin of the British Radiological Protection Board in 1975,[90] and by the American National Council on Radiation Protection and Measurements (NCRP) in 1975.[89] All these authorities conclude that the evidence available does

not support the suggestion. The NCRP report concludes "The substantial body of experimental animal data available at the present time indicates that particulate plutonium in the lung is no greater hazard than the same amount of plutonium more uniformly distributed throughout the lung."[89] The British MRC report cited above concludes: "There is at present no evidence to suggest that irradiation of the lung by particles of plutonium is likely to be markedly more carcinogenic than the same activity if uniformly distributed. Accordingly, in the calculations which follow, dose is averaged throughout the lung in accordance with current ICRP concepts." In considering existing derived safety standards for plutonium-239 their broad conclusion is that "... these standards, including that for inhaled insoluble particles of plutonium, fulfil the intentions of the ICRP, although adjustment of the standards by small factors may be needed."[74]

It is not appropriate in this report to consider all the evidence for these conclusions and that which has been published subsequently, but the following points deserve particular mention: Firstly, the possibility of greater cancer risk per dose at larger doses (which could result from a non-linear relation) is limited to those cells which survive the sterilising or killing effect of very large doses. Considerations of this kind have led Mayneord and Clarke to conclude that the maximum possible increase of risk from alpha emitting particles in excess of that calculated by the normal ICRP methods is unlikely to exceed five-fold, even for those exceptional distributions of activity which lead to maximum expectations of risk.[91]

Secondly, Albert rejects the use of his rat skin experiments as a proper basis for the suggestion and points out that alpha irradiation of localised regions of the hair follicle, in a pattern similar to that from a plutonium particle, is not tumorigenic.[92)]

Thirdly, detailed experimental studies to test the suggestion are in progress at Los Alamos.[93,94)] These relate to hamsters bearing microspheres permanently lodged in their lungs as "hot particles" containing 0.22 to 59 pCi per sphere, and giving lung burdens of 440 to 354,000 pCi.[94)] Preliminary results do not support the Tamplin/Cochran suggestion but suggest the reverse conclusion, namely that highly localised radiation produces less damage than the same dose distributed more uniformly. 756 hamsters given 30 x 10^6 microspheres have developed four neoplasms so far, as compared with 15,000 expected from the Tamplin/Cochran model.[94)]

Fourthly, experience with plutonium in humans does not support the suggestion. Despite the hundreds of workers that have been exposed to plutonium, mostly in particulate form, no tumours have been reported that are clearly attributable to plutonium exposure.[89)] In particular, long-term studies are available on 25 workers who absorbed plutonium internally in the war years, mainly because of inhalation of plutonium-bearing aerosols.[88)] Three of these have plutonium burdens exceeding the ICRP maximum permissible body burdens of 40,000 pCi by more than five-fold, five others by more than three-fold, 10 others have more than the permissible burden, and seven others have burdens exceeding one-tenth of this recommended limit. One additional worker died, aged 38, of coronary heart disease. The 25 have shown no abnormalities

except for ailments that one would expect in a group of men mostly in their early fifties. A malignant melanoma has been removed from the chest wall of one subject, a second had a partial gastrectomy for a bleeding ulcer, and a third had a "coin lesion" in his lung which proved to be benign.[88] If all the 16,000 pCi of plutonium regarded as permissible in a man's lung were contained in 10^5 or fewer hot particles, each containing 0.07 pCi or more and having a probability of inducing cancer of one in 2,000, then the hot particle theory would predict that his overall probability of contracting cancer would exceed 0.99 (99%). In comparison, the expectation from the conventional ICRP approach is equivalent to the risk from 15 rem per year to the lung for about 30 years, which is $30 \times 15 \times 25 \times 10^{-6}$ or 0.01, based on the risk estimates given in Table 5. It appears reasonable to conclude either (a) that plutonium aerosols as normally encountered do not contain a significant porportion of hot particles of 0.07 pCi or more, or (b) that these hot particles do not have the exceptional cancer-inducing properties ascribed to them.

7. COMPLIANCE WITH RADIATION PROTECTION STANDARDS

7.1 Health physics at nuclear power facilities:

The major task of health physics staff attached to regulatory authorities for nuclear power facilities is to ensure that the dose limits which may be prescribed for radiation workers and members of the public are not exceeded. To do this, they must monitor external radiation levels (which is relatively simple), identify the pathways to man of potentially dangerous radionuclides which may be emitted from

nuclear power facilities and make appropriate routine measurements of their concentrations in samples of air, water, milk, fish, soil, and any other relevant substance in the vicinity of nuclear facilities. By now the most important pathways of entry to the human food chain of such radionuclides have been identified in various countries.[95] One such is ruthenium in laverbread, as mentioned above.[67] Several involve a process commonly described as "concentration" of radionuclides in animals, fish, and plants, following their release either as dilute suspensions or solutions in water or as fallout onto the ground from nuclear weapon testing.[96]

Such processes cannot concentrate a radionuclide such as strontium-90 relative to the total amount of strontium present, but they may concentrate one element relative to another. Thus, the ratio of strontium-90 to calcium in plants is comparable with that in the soil in which they grow; in the milk from cattle it is typically one-tenth of that in the grass they consume, and in the bones of humans drinking this milk it is typically one-quarter of that in the cow's milk.[96] These are examples of concentration of calcium relative to strontium. Nevertheless, some 0.3 to 4% of strontium-90 and about 5% of iodine-131 consumed by cows from contaminated pasture are transferred to their milk, and much of this accumulated radioactivity is ultimately localised as in bone (strontium-90) or in the thyroid gland (iodine-131) of humans who consume this milk.[96]

Again, owing to the need for specific elements such as chromium or cobalt, considerable accumulation of these elements occurs in fish, algae, seaweed, etc., which grow in the water containing their salts. This is particularly the case if the

water is normally lacking in such elements. This accumulation may be expressed as a concentration factor (ratio of radioactivity per gram of fish to radioactivity per gram of water). In the case of some shell fish such as oysters, the process is more akin to a "carpet sweeper" action. Typical values of such concentration factors relative to sea water are 100,000 for silver and zinc in oysters,[67] 1,000 for ruthenium in porphyra seaweed,[67] 30,000 for chromium in fish muscle,[97] 200 for cobalt in fish muscle,[97] 20 to 600 for strontium in fish bone,[98] 1 to 10 for plutonium in fish muscle,[98] 300 for plutonium in mussels and clams,[98] 20,000 for plutonium in seaweed,[98] 1,000 for polonium in fish muscle,[98] and 100 for caesium in fish muscle.[97]

Most of the elements cited above for sea water would apply only to releases from fuel reprocessing plants, and thus would have no relevance in New Zealand.

7.2 Record of the nuclear industry:

The following table has been extracted from an Australian Atomic Energy Commission publication to illustrate the radiation exposures received by workers in the whole nuclear industry, of which nuclear power generation forms only a part.[99] There have been several instances where the recommended maximum permissible dose has been exceeded, but the overall record appears satisfactory.

Table 6 - Radiation exposure of nuclear workers (from ref. (99))

Organisation or country	No. of years	Man-years' experience	No. of times 5 rem/y exceeded
USAEC	28 (1943-1970)	1,663,852	1971
UKAEA	3 (1965-1967)	53,815	249
CEGB	9 (1962-1970)	19,500	0
AAEC	9 (1966-1974)	9,016	1
Canada	1 (1969)	28,643	17
France	1 (1969)	19,595	0
Germany (Federal Republic)	1 (1969)	22,550	18
India	1 (1969)	26,391	between 100 & 250*

*Not directly reported, inferred from other reported data.

USAEC - United States Atomic Energy Commission and its contractors (now part of ERDA - the Energy Research & Development Administration)

UKAEA - United Kingdom Atomic Energy Authority

CEGB - Central Electricity Generating Board (England & Wales)

AAEC - Australian Atomic Energy Commission

KEY TO REFERENCES

Many reports have been listed as letter codes. The following list gives the publication source for these reports.

AAEC - Australian Atomic Energy Commission (Sutherland, NSW)

ACRL=ARCRL - Agricultural Research Council Radiological Laboratory (Risley, UK)

AEC - U.S. Energy Research and Developement Agency (Washington, DC)

AECL - Atomic Energy of Canada Ltd (Ottawa, Ontario)

AERE - Atomic Energy Research Establishment (Harwell, UK)

ANL -
BNWL -
BRH/DER -
CONF -
DBE=BRH/DBE -
EPA -
ERDA - } U.S. Energy Research and Development Agency (Washington, DC)

DSIR Report NIP- See NIP

EUR - European Atomic Energy Community (Brussels)

IAEA/STI -
IAEA/INFCIRC -
IAEA/WHO - } International Atomic Energy Agency (Vienna)

ICRP - International Commission for Radiological Protection (UK)

IEEE - Institute of Electrical and Electronics Engineers (New York)

INFCIRC - See IAEA

INSL - held by the library, Institute of Nuclear Sciences (Lower Hutt)

LA -
LA-UR - } U.S. Energy Research and Development Agency (Washington, DC)

NCRP - National Council on Radiation Protection and Measurement (USA)

NIP - "Nuclear InPut" - file held at the Institute of Nuclear Sciences, DSIR (Lower Hutt). The six-digit number following some references is the abstract number in the NIP file (e.g., 000398)

NRC - U.S. Energy Research and Development Agency (Washington, DC)

NRL - National Radiation Laboratory (Christchurch, NZ)

NRPB - National Radiation Protection Board (UK)

OECD/NEA - Organisation for Economic Co-operation and Development, Nuclear Energy Agency (Paris)

ORNL -
ORO -
RD -
USAEC -
WASH - } U.S. Energy Research and Development Agency (Washington, DC)

REFERENCES

(1) NIP report no. 3. Radioactive releases from nuclear power plants. (000488)

(2) U.N. Scientific Committee on the Effects of Atomic Radiation. Ionizing radiation: levels and effects. Vol. 2. Effects. New York, 1972.

(3) National Academy of Sciences. National Research Council Committee on the Biological Effects of Ionizing Radiations. The effects on populations of exposure to low levels of ionizing radiation: report of the advisory committee. Washington, 1972. (000233)

(4) U.S. Nuclear Regulatory Commission. Reactor safety study. Appendix VI. Calculation of reactor accident consequences. (WASH-1400) 1975.

(5) U.S. Nuclear Regulatory Commission. Reactor safety study. Main report. (WASH-1400) 1975, p. 51.

(6) Ref. (4) p 9-8.

(7) Ref. (4) p F-44.

(8) Ref. (4) p F-13.

(9) Ref. (4) p F-14.

(10) Ref. (4) p F-15 and F-16.

(11) Ref. (4) pp F-18, 19, and 35.

(12) Ref. (2) pp 405 and 408.

(13) Ref. (3) p 171.

(14) Ref. (4) p G-23.

(15) Ref. (5) p 74.

(16) G.W. Dolphin and W.G. Marley. Risk evaluation in relation to the protection of the public in the event of accidents at nuclear installations. (U.K. Atomic Energy Authority Health and Safety Branch Report AHSB (RP) R93). 1969.

(17) Ref. (4) pp 9-26 and 9-27.

(18) G.W. Dolphin and S.A. Beach. Health Physics, 9: 1385, 1963.

(19) Ref. (4) p H-13.

(20) Ref. (3) pp 85 and 91.

(21) Ref. (3) p 80.

(22) Ref. (3) p 62.

(23) Ref. (3) p 46.

(24) Ref. (3) p 171.

(25) Ref. (2) p 441.

(26) Ref. (2) p 404.

(27) Ref. (3) pp 89-91.

(28) U.N. Scientific Committee on the Effects of Atomic Radiation. Ionizing radiation: levels and effects. Vol. 1. Levels. New York. 1972. pp 9-10.

(29) Ref. (2) p 387.

(30) R.D. Evans, Health physics. 27: 497, 1974.

(31) National Council on Radiation Protection and Measurements. Review of the current state of radiation protection philosophy. (NCRP report no. 43) 1975. (INS Lib.)

(32) W.G. Marley. In Proceedings of the symposium on the "Management of radioactive wastes from fuel reprocessing", Paris, 27 Nov-1 Dec. 1972. Paris, OECD, 1972. p.37.

(33) Ref. (4) pp 9-25 and 9-36.

(34) U.S. Environmental Protection Agency Office of Radiation Programs. Reactor safety study (WASH-1400): a review of the final report. 1976. pp 2-2 to 2-6 and references cited therein. (000776)

(35) B.D. Modan, H. Baidatz, H. Mart, R. Steinitz and S.G. Lenin. Lancet. (7852):277, 1974.

(36) J.W. Baum (a) Health Physics. 25: 97, 1971. (b)Mutation theory of carcinogenesis and radiation protection standards. Sixth annual Health Physics Society symposium, Richland, Washington, 1971. p 11.

(37) Ref. (3) p 123.

(38) Ref. (3) pp 145-155.

(39) Ref. (28) pp 35 and 81.

(40) R.M. Fry. Radiation hazards of uranium mining and milling. (AAEC/IP9) 1975.

(41) International Commission on Radiological Protection. Report of Committee 11 on permissible dose for internal radiation. (ICRP publication 2) 1959.

(42) Ref. (3) pp 55-57.

(43) Ref. (2) pp 257-259.

(44) A.C. Stevenson, Radiation research Suppl. 1: 306, 1959.

(45) B.K. Trimble and J.H. Doughty, Annals of human genetics. 38: 199, 1974.

(46) H.B. Newcombe In Proceedings of the fifth international congress of radiation research, Seattle, 1974, p 937.

(47) Ref. (4), Appendix I, p I-1 to I-8, and table VI 9-11, p 9-40.

(48) Ref. (3) pp 64-65.

(49) Ref. (3) p 53.

(50) N.Z. Dept of Health. Health Statistics Report. Mortality

(51) B.L. Cohen. Health physics. 30: 351, 1976.

(52) International Atomic Energy Agency. World Health Organization. Nuclear Power and the Environment. (STI/PUB/321) Vienna, 1973. p 11. (000315)

(53) International Recommendations on Radiological Protection. Revised by the International Commission on Radiological Protection at the 6th International Congress of Radiology, London, 1950. Radiology. 56: 431, 1951.

(54) International Commission on Radiological Protection. Recommendations of the International Commission on Radiological Protection. (ICRP Publication 1) 1959.

(55) International Commission on Radiological Protection. Recommendations of the International Commission on Radiological Protection. (ICRP Publication 9) 1969.

(56) B.L. Cohen. Bulletin of the Atomic Scientists, 30(8): 35, 1974.

(57) International Commission on Radiological Protection. Implications of Commission recommendations that doses be kept as low as readily achievable. (ICRP Publication 22) 1973.

(58) Cited by T.H. Pigford. Annual Review of Nuclear Science. 24: 515, 1974. (000247)

(59) Design description of Pickering Generating Station. Ontario Hydro and AECL Power Projects, 1972, p 192. (000756)

(60) B. Windeyer, Atom (209):58, 1974.

(61) Royal College of Physicians. Smoking and Health Now. 1971, pp 144-147.

(62) H.S. Diehl. Tobacco and your health: the smoking controversy. New York, McGraw Hill, 1969, p 63.

(63) USAEC operational accidents and radiation exposure experience within the USAEC, 1943-1970. (WASH-1192) 1971.

(64) N.A. Frigerio, K.F. Eckerman and R.S. Stowe, Argonne National Laboratory Environmental Statement. The Argonne radiological impact program (ARIP?. Part I. Carcinogenic hazard from low-level, low-rate radiation. (ANL/ES-26) 1973.

(65) A.R. Gopel-Ayengar, K. Sundaram, K.B. Mistry, C.M. Sunta, K.S.V. Nambi, S.P. Kathuria, A.S. Baso and M. David, Evaluations of the long-term effects of high background radiation on selected population groups on the Kerala Coast. Proceedings of 4th international conference on the peaceful uses of atomic energy, Geneva, 6-16 September 1971. IAEA, Vol. II. 1972. Vienna, 1972.

(66) Ref. (28) p 105.

(67) A. Preston and N.T. Mitchell, In Radioactive contamination of the marine environment, Vienna, IAEA, 1973. (STI/PUB/313) p. 575. (000411)

(68) Ref. (3) p 9.

(69) Joint Committee on Atomic Energy. Hearings on environmental effects of producing electric power," 91st Congress Session, October 28-31 and November 4-7, 1969. Appendix 12. p 655, Washington, D.C. Govt. Print, 1969.

(70) A.R. Tamplin and J.W. Gofman, "'Population Control' through nuclear pollution," Chicago, Nelson-Hall, 1970.

(71) J.W. Gofman, J.D. Gofman, A.R. Tamplin and E. Kovich, Radiation as an environmental hazard. Paper presented at the 1971 Symposium on Fundamental Cancer Research, M.D. Anderson Hospital and Tumor Institute, Houston, Texas, 1971.

(72) J.W. Gofman and A.R. Tamplin. Epidemiologic studies of carcinogenesis by ionizing radiation, Proceedings of the Sixth Berkeley symposium on mathematical statistics and probability, April 9-12, June 16-21, and July 19-22, 1971. Volume VI, p 235. Berkeley.University of California Press, 1972.

(73) Ref. (3) pp 183-188.

(74) Medical Research Council. The toxicity of plutonium. London, HMSO, 1975. p 34.

(75) E.J. Sternglass. Infant mortality and nuclear tests. Bulletin of the Atomic Scientists. 25(4): 18, 1969.

(76) E.J. Sternglass. Death of all children. Esquire. September 1969.

(77) E.J. Sternglass, Evidence for low-level radiation effects on the human embryo and fetus: Radiation biology of the fetal and juvenile mammal. Proceedings of the ninth annual Hanford biology symposium, Richland, Washington, May 5-8, 1969, Ed. M.R. Sikov and D.D. Mahlum. (CONF-690501) 1969.

(78) E.J. Sternglass. Environmental radiation and human health. Proceedings of the sixth Berkeley symposium on mathematical statistics and probability, April 9-12, June 16-21, and July 19-22, 1971. Volume VI. p 145, Berkeley, University of California Press, 1972.

(79) Ref. (3) pp 178-179.

(80) P.J. Lindop and J. Rotblat. Nature. 224: 1257, 1969. (000408)

(81) E. Tompkins and M.L. Brown, Evaluation of a possible causal relationship between fallout deposition of strontium-90 and infant and fetal mortality trends. (DBE 69-2) 1969.

(83) NIP report no. 30. Biological hazards of plutonium.

(84) Ref. (74) p 23.

(85) A.R. Tamplin and T.B. Cochran. Radiation standards for hot particles: a report on the inadequacy of existing radiation protection standards related to internal exposure of man to insoluble particles of plutonium and other alpha-emitting hot particles. Washington, Natural Resources Defense Council, 1974.

(86) A.R. Tamplin and T.B. Cochran. New scientist. 66: (951): 497, 1975.

(87) R.E. Albert, F.J. Burns and R.D. Neimbach. Radiation research. 30: 515, 525, and 590, 1967.

(88) G.L. Voelz. Health physics. 29: 551, 1975. (000409)

(89) National Council on Radiation Protection and Measurements. Alpha-emitting particles in lungs. (NCRP Report no. 46) 1975. p 9-10. (INS Lib.)

(90) G.W. Dolphin. A Brief Critical Review of "A Report on the Inadequacy of Existing Radiation Protection Standards Related to Internal Exposure of Man to Insoluble Particles of Plutonium and Other Alpha-Emitting Hot Particles" by A.R. Tamplin and T.B. Cochran, 14th February 1974", NRPB, Harwell, Didcot, Oxfordshire, England, 1974.

(91) W.V. Mayneord and R.H. Clarke. Nature. 259: 535, 1976.

(92) R.E. Abert. Plutonium and other transuranium elements: sources, environmental distribution and biomedical effects. (WASH-1359) 1974. p 311.

(93) C.R. Richmond. Health physics. 29: 525, 1975. (000332)

(94) L.M. Holland, J.R. Prine, D.M. Smith and E.C. Anderson. (LA-UR-75-1870) (Conf. 751043-3) 1975. INS Lib.)

(95) IAEA, Environmental Behaviour of Radionuclides Released in the Nuclear Industry (STI/PUB/345) 1973. And numerous other IAEA publications.

(96) R. Scott Russell. Radioactivity and human diet. Oxford, Pergamon, 1966.

(97) D. Van As, H.O. Fourie and C.M. Vleggaar. In Radioactive Contamination of the Marine Environment. IAEA Vienna, 1973. (STI/PUB/313) p 615.

(98) V.E. Noshkin. Health physics. 22: 537, 1972.

(99) M.W. Carter, E. Carruthers and J.C.E. Button. (AAEC/IP8) 1975. p 4. (000297)

(100) Ref. (3) p 182.

(101) National Council on Radiation Protection and Measurements. Natural background radiation in the United States. (NCRP report no. 45) 1975, pp 88-89.

(102) B.L. Cohen Bulletin of the Atomic Scientists. 32 (2): 61, 1976. (000599)

(103) D.D. Comey, Bulletin of the Atomic Scientists 32 (2): 63, 1976. (000599)

(104) D.D. Comey, Bulletin of the Atomic Scientists 31: 43 1975. (000598)

(105) U.S. Environmental Protection Agency. Environmental analysis of the uranium fuel cycle. Part I - Fuel supply (EPA-520/9-73-003-B) 1973, pp 24, 61 and 73.

(106) OECD Nuclear Energy Agency and IAEA. Uranium. Resources production and demand. Paris, 1973. p 9.

(107) Ref. (2) p 256.

(108) N. Kochupillai, I.C. Verma, M.S. Grewal and V. Ramalingaswami. Nature. 262: 60, 1976.

(109) Great Britain Parliament. Command no. 6618. Royal Commission on environmental pollution. London, HMSO, 1976, p 87.