

The Importance of Radiation Risk Assessment

by E.E. Pochin

INTRODUCTION

Radiation protection is about safety, and the prevention of any undue risk from radiation exposure at work or in the general environment. Obviously therefore, any quantitative recommendations on exposure limits and procedures must be made in relation to quantitative estimates of the risks that would be involved in the use of these limits.

SOMATIC EFFECTS

In its Publication 26, Ref.[1] the International Commission on Radiological Protection (ICRP) reviewed the types of harm that might result from radiation exposure at low dose, and gave estimates of the frequency with which the major such effects might occur. For the induction of cancer or of leukaemia, the reviews made by the Commission's Committee on Radiation Effects, and the extensive survey by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) Ref.[2] had examined the substantial amount of new epidemiological evidence on the frequency with which malignancies were induced in human tissues, by absorbed doses of up to a few gray*. A number of estimates were therefore available, not only of the total number of malignancies that might result from whole-body exposure, but also of the number of cancers to be expected when individual body organs or tissues were selectively irradiated, for example as a result of the intake of radionuclides.

Thus in the case of several such tissues, and particularly those of the bone marrow, thyroid, lung and breast, there are now several separate sources of risk estimation giving reasonably consistent estimates, and in some instances giving evidence on the variation of the induction rate with the sex or age of those exposed Ref.[3]. For a number of other body organs or tissues, estimates have been obtained from one or more sources of evidence which indicate the approximate induction rate and show this rate is low relative to that for the more "sensitive" tissues. Much work still needs to be done, particularly in identifying the cell types within certain organs which are responsible for cancer development following radiation, so that the absorbed dose from incorporated radionuclides can, when necessary, be estimated in relation to these cell types rather than as averaged over the organ as a whole. Enough human epidemiological evidence, of reasonable reliability, has accumulated,

* Units 1 gray (Gy) = 100 rad, 1 sievert (Sv) = 100 rem

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however, to indicate the contribution that cancer induction in different body organs is likely to make to the total carcinogenic effect of whole-body irradiation. This has the important consequence that a quantitative comparison can be made between the effects, for example in the induction of fatal malignancies, of uniform whole-body irradiation — as from external sources — and selective organ irradiation — as from internally retained radionuclides. In this way a more valid and coherent basis can be recommended for internal limits than previously Ref.[4].

GENETIC EFFECTS

For genetic effects also, the radiation risk to man can now be estimated with somewhat increased confidence. It remains true that the estimated frequency with which genetic effects are induced *in vivo* still depends very largely upon observations in the mouse or other species rather than in man. Studies of human and other cells in culture, however, indicate that the induction of chromosome aberrations by radiation follows a similar type of dose-effect relationship in different species Ref.[5]. There is reasonable agreement between the estimates of genetic risks of radiation, as derived by two separate methods which have been very fully and ably reviewed by a task group of the Commission's Committee on Radiation Effects, and in the 1977 UNSCEAR report. Firstly, the hazard in man has been assessed in relation to the estimated size of the genome, the amount of genetic material, in man relative to the mouse, and the sensitivity of the mouse genome to radiation. And secondly, an estimate has been based on the radiation dose which is likely to double the natural incidence of genetic abnormalities, since the "doubling dose" in animals is found to be remarkably similar, at about 1 gray, for a variety of types of genetic change Refs [6,7].

The risk of causing an inherited abnormality depends naturally upon the likely number of children that will be conceived subsequently, and therefore upon the age at exposure; and this risk of inducing any substantial genetic effect is estimated as a rate falling from about 2 per 100 sieverts during adolescence to zero by the age of about 50 Ref.[8]. The average risk of inducing such a defect in the children or grandchildren of an occupationally exposed worker is a little less than 0.4 per 100 sieverts. This risk is about one-third that of inducing a fatal cancer in the worker himself.

OTHER EFFECTS

The third main group of radiation effects for which risk estimates are needed is that of the so-called non-stochastic effects. These include such consequences as cataract, sterility, tissue fibrosis and impairment of organ function. These are ordinarily thought to occur only if a relatively high threshold dose has been exceeded, so that the risk estimate at lower doses is believed to be zero. In many cases, the dose limits set to restrict the induction of fatal cancers and genetic effects should therefore prevent the occurrence of non-stochastic effects. In certain tissues however, and particularly for bone, skin and thyroid, for which the fatal cancer induction rate is low, this might not be so. An overriding limit of 0.5 sieverts per year is therefore imposed for any such tissues to prevent the induction of non-stochastic effects even after 50 years of constant occupational exposure at the dose limit. This seems to represent a policy of extreme caution since rather few workers are likely to accumulate even half of a lifetime occupational dose of this size. Nor is it yet clear what forms of non-stochastic harm would result from this dose of 25 sieverts delivered over 50 years say

to bone, or whether any such harm induced at the very end of working life would be comparable in detriment to a fatal cancer or a major genetic defect. And, with a ten-times lower non-stochastic limit for the general public, it seems even less likely that any significant non-stochastic harm would result from exposures below these limits.

I believe that much further work is needed on the possible induction of non-stochastic effects by prolonged exposure of certain tissues or organs, as well as on such questions as the age dependency of the induction rate of various forms of cancer and on the genetic risk as affecting man or human cells. More information is needed also on the factors influencing the neoplastic transformation of cells and their survival, and so the likely form of the dose-effect relationship at low doses and the way in which the carcinogenic effect of radiation at a level of milligrays should be inferred from that observed at a level of grays. This radiobiological approach is particularly important, since any epidemiological evidence is likely to be so unreliable statistically at these low dose levels.

COMPARISONS OF RISK

Enough clear and quantitative information is now available, however, for estimating the general level of risk involved in radiation exposure. Indeed, the numerical bases for radiation risk estimation are very much better and more comprehensive than those for establishing the risks of exposure to many important chemical and other potentially harmful agents in the working or the general environment. It is in fact an unusual situation, that the probable hazard of an environmental contaminant should — as it clearly desirable — be estimated before its effects at environmental levels have been detected. Anomalously, but predictably, some of the problems in public acceptance of radiation hazards appear to arise from this essentially responsible attempt to estimate the degree of safety or level of risk involved in practices which entail radiation exposure, and to present these estimates for consideration. It must be important, however, that any recommendations should be accompanied by a statement, and as clear a statement as possible, on their implications, and on the degree of safety or of hazard that may result from their adoption.

Such a statement can I think only be seen in a true perspective however if it is made, not merely in absolute numbers of possible fatalities or other defects, but in comparison with the corresponding levels of safety or hazard of other and more familiar situations or procedures. This type of comparison is the more important since many people tend to think of procedures as being either safe or unsafe, in an absolute sense, and, understandably, do not think in a quantitative way about different levels of risk or have familiarity with the different levels of risk involved in familiar situations.

It is important to emphasize that a comparison of numerical levels of risk from different procedures cannot and should not in itself determine the acceptability of any one procedure. It should however, surely be an important factor in influencing acceptance or rejection of alternatives, or in determining the way in which they should operate. Biological safety is only one component in any decision between available alternatives, for example as between different occupations or different sources of electricity production. It must however be regarded as a very important component, and one which must be based on an evaluation, not only of the types of harm, but also of the numerical frequency of these types in the different alternatives.

OCCUPATIONAL RISK

In Publication 26 the Commission gives an estimate of the detriment that might result in occupations in which the radiation exposure is controlled on the basis of the recommended dose limits, when exposures above these dose limits are avoided, and when doses are reduced as much below these limits as is reasonably achievable, as the Commission advises. When this is done, it is ordinarily found that the average dose throughout the occupation is in the region of 5 millisieverts (mSv) per year – and this is shown in most of the occupations reported by UNSCEAR. In these circumstances, and with a risk of fatal induced cancer of just over 1% per sievert, the annual fatality rate from this cause would be about 6 per 100 000 workers. Adding the risk of hereditary defects induced in the workers' families would bring this figure to 8; and to a total of 9 or 10 per 100 000 when one adds also the fatal accident rate, which is ordinarily low in nuclear establishments. Under these conditions therefore it can be estimated that the total annual risk of fatalities and of major genetic defects is unlikely to exceed 10^{-4} , or the equivalent of 100 deaths per million employed per year.

As regards fatalities, therefore, this risk is comparable with the risk of fatalities in many conventional occupations in many countries (Table 1*) Ref.[9] for example in the United States where the occupational fatality rate varies widely in different industries, and is falling by a few per cent per year, but exceeds this figure of 10^{-4} , or of 100 per million per year, in most occupational groups. And comparable rates are found in other countries in similar types of work (Tables 2, 3) Refs [10, 11, 12].

* Tables 1 and 2 are reproduced from the Journal of the Royal College of Physicians of London, and Table 3 from Community Health, by permission of the Editors

Table 1. US fatal occupational accident rates ($10^{-6}/\text{yr.}$)

Industry	1955	1958	1961	1964	1968	1971	1975	Mean
Trade	120	90	90	80	70	70	60	83
Manufacturing	120	120	110	100	90	100	80	103
Service and government	150	140	130	130	125	125	115	131
Transport and public utilities	340	330	430	440	380	360	330	373
Agriculture	550	570	600	670	650	670	580	613
Construction	750	740	740	730	740	710	610	717
Mining and quarrying	1 040	960	1 080	1 080	1 170	1 000	630	994
All (these) industries	240	220	210	210	190	180	150	200

Table 2. France, National Statistics of Occupational Accidents (Statistiques Nationales d'Accidents du Travail, 1968-70) ($10^{-6}/\text{yr.}$)

Clothing industries	17
Textile industries	42
Metal workers	118
Chemical industries	169
Quarrying, etc.	365
Dockers (marine)	1,020
Trawling, téléphériques, pleasure vessels, etc.	1,636

Table 3. United Kingdom fatal accident rates (Annual Reports of the Chief Inspector of Factories, 1959-70) ($10^{-6}/\text{yr.}$)

Occupation or manufacture	Mean \pm S.E.
Clothing and footwear	3 \pm 1
Engineering and electrical goods	23 \pm 1
Textiles	23 \pm 2
Vehicles	26 \pm 2
Paper, printing, and publishing	28 \pm 2
Metal goods not elsewhere specified	29 \pm 2
Food, drink and tobacco	34 \pm 2
Leather, leather goods and fur	37 \pm 8
Timber, furniture, etc.	64 \pm 5
Bricks, pottery, glass, cement, etc.	75 \pm 5
Chemicals and allied industries	87 \pm 5
Metal manufacture	136 \pm 5
Shipbuilding and marine engineering	162 \pm 8

This comparison is based on fatalities, and on major genetic defects only. But if account is taken of a wider range of occupational harm, including non-fatal accidents and diseases as is attempted in ICRP Publication 27 on an Index of Harm, a similar comparison emerges Ref.[8]; with the average risk in many occupations involving radiation exposure being equivalent to that in other occupations with a fatal accidents rate of about 50 per million per year — again, a risk which should be reduced by any reasonably achievable means but comparable with that in existing occupations which are ordinarily regarded as being essentially safe ones.

In certain occupations, however, or sections of an occupation, the average radiation exposure, and therefore the average radiation risk is substantially higher. If in an extreme case, radiation exposure were to be planned or conducted so that all workers received the dose limit of 50 millisievert every year, the harm would on the same criteria be equivalent to that of an occupation with a fatal accident rate of about 350 per million per year. This would correspond to the risks recorded for transport and public utility workers in the United States, for quarry workers in France, or as the average for coal miners in the United Kingdom from 1967 to 1976 Ref.[13].

In uranium mining, the average radiation exposures have been high in the recent past, both from inhalation of radon and to a lesser extent from external radiation. To these must be added a substantial accidental death rate which will have had an impact in occupational harm several times that from radiation, having an annual fatality rate often exceeding 10^{-3} Ref.[3].

I believe however that when the dose limits are treated truly as limits which are never to be exceeded by any worker in any year, and when average exposures can therefore be held considerably below this limit, the total harm of occupational origin will usually be within the range of that observed in industries of recognized safety, unless the occupation itself also involves substantial accidental hazards which in any case exclude it from this category of safety.

RISK TO THE PUBLIC

The maximum radiation risk to members of the general public is much less readily expressed in relation to comparable risks, for several reasons. Firstly, the limits recommended for exposure of a member of a critical group in any one year (of 5 mSv) are likely in most cases to be related, not to the dose actually received, but to that which might be received on the basis of environmental models designed to maximize the estimated exposure. And secondly, it is difficult to select other environmental sources from which the risk is imposed on the public in the same way and for which the risk is known quantitatively. The risks from atmospheric discharges from coal-fired power plants are similarly “imposed” but — remarkably — cannot yet be estimated with confidence. The risks from natural phenomena can be estimated, but are not imposed in the same way, by human action. The Commission reviewed various types of environmental risk, as have many authors Refs [15, 16, 17, 18], and expressed the opinion that annual risks of fatality from an environmental source of 10^{-6} to 10^{-5} (i.e. with a risk of “killing the individual” once in 100 000 years or longer) “would be likely to be acceptable to any individual member of the public”.

The subject of public risk is not addressed in Publication 27. The estimate of harm by length of life lost may however be applied to a member of the public who was exposed

continually to radiation at the limit suggested for lifelong exposure, of 1 mSv per year. (This limit applies to exposures actually thought to be received, rather than as estimated by the maximizing assumptions of an environmental model). Taking account of the likely latencies in the expression of radiation induced cancers, both in childhood and in adult life, the continuous lifelong exposure of such an individual at the limiting rate would appear on average to involve a life shortening of about six days. For perspective, it would be useful if estimates were made of the mean life shortening attributable to other environmental agents. The Commission refers in Publication 26 to the average risk of death from traffic accidents. In the UK population, the mean life shortening from this cause is about 1.5 days Ref.[19] The mean loss of life from man-made radiation exposures, if similarly averaged throughout the population, would be about 3.3 days, of which 3.1 days would be attributable to exposures from *medical radiology*, and 0.2 days from *all other sources* Ref [20].

ACCEPTABILITY OF RISKS

It must be repeated, that neither statements of the numerical level of risk, nor comparisons of these levels with those arising from other activities, should or will determine occupational or public acceptability of the risk or choice of the activity. Where radiation is involved, such decisions are likely to be affected also by many other considerations: by the type of risk, with carcinogenic risks coloured by the fear of cancer and by the nature of the disease, and genetic risks associated with the unfairness of affecting later generations by the activities of today, and the presence of carcinogens and mutagens also in fossil fuel and other chemical discharges is often not recognized. An accidental death is often felt by the worker, rightly or wrongly, to be due to a lack of skill on the part of the victim, whereas this cannot be true of a cancer resulting from a given radiation exposure sustained under normal working conditions. The very action of estimating the size of radiation risks may also suggest their severity, particularly to those to whom the levels, and even the occurrence, or risks from other sources is unknown. Moreover, the need to estimate the risks of low doses prospectively and partly on theoretical grounds will necessarily carry less conviction than when risks can be determined retrospectively from observed statistics. And above all, risks from unfamiliar sources are usually more feared, or are felt to be greater, than those from sources which have long been commonplace. This remains true for radiation, even despite the fact that the average annual exposure from all artificial sources (apart from medical ones) is probably less than 2% of that which has always been received from natural sources Ref.[20].

So the importance of radiation risk assessment may lie, not only in the necessity for knowing that the public health protection is adequate in circumstances where some exposure is unavoidable, nor yet in the need to accompany any recommended limits by a statement of their implications. It must lie also in part in attempting to ensure that an informed public and their representatives make correct and informed decisions which minimize human harm.

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