

# ARE THERE RISKS FROM EXPOSURE TO LOW LEVEL RADIATION?

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## ABSTRACT

For doses up to at least 50 mSv received at a high rate in a single event by adults (or 10 mSv for exposure of the embryo and foetus) and up to at least 15 mSv per year received routinely, there is no proof that radiation increases risks of cancer. One reason for this may be that there are beneficial or protective effects of radiation which tend to offset and perhaps dominate over the harmful effects at low levels of exposure. Another reason is that the effects are too small to be measured. It is not necessarily in the best interests of individuals or of society to assume that the linear hypothesis should be used to estimate risks throughout this range.

## INTRODUCTION

With the resumption of nuclear weapons testing by France, there has been much in the news and "on the streets" in Australia about the dangers of exposure to radiation. We continually hear and read statements to the effect that there is no safe level of exposure and, regrettably, our profession has to bear some of the responsibility for this misconception.

Elsewhere in the world, a great deal of dissent and concern is being expressed regarding radiation protection regulations and practices which are based on the view that no level of radiation dose is free of risk. The first sixteen pages of the Health Physics Society's Newsletter of June 1995 provides examples. Particular concerns have been expressed that such regulations impose high costs on industry without achieving demonstrable benefits. It has even been suggested that some providers of radiation protection services have a vested interest in this situation.

Central to both issues is the ICRP's recommendation that risks of radiation induced cancers should be assumed to be proportional to dose without thresholds (1), the "linear hypothesis". It needs to be stressed that the linear hypothesis is an assumption, not a proven fact or a law of nature. Its application to low levels of radiation needs to be placed in perspective. This should not be taken as support for nuclear weapons testing or as opposition to the assessment and regulation of radiation protection practices. It simply reflects the need to apply a scientific approach to the question of risk at low levels of radiation exposure.

## RISK ESTIMATION IN PERSPECTIVE

The linear hypothesis was formulated for the purpose of assessing radiation protection practices, not for estimating risks to individuals from low levels of radiation. The risk coefficients recommended by the ICRP (1) are based on the observed health effects of high doses received at high dose rates, e.g. by atomic bomb survivors at Hiroshima and Nagasaki. Radiation is more likely to be harmful when delivered at these high rates rather than spread over a protracted period. However, even at the highest rates which have been experienced, statistically significant increases in the incidence of cancers have not been observed at dose levels less than about 50-200 mSv for adults (1-2). For exposure of the embryo and foetus, detectable health effects have been observed at dose levels down to about 10-20 mSv.

The extent to which the linear hypothesis and the recommended risk coefficients apply

at lower doses and dose rates is questionable. As explained below, this is not so much a matter of thresholds as of the relative importance of different effects. However, the ICRP does not rule out the possibility that thresholds may exist (1).

It appears to be well established that radiation has beneficial or protective effects on living cells and organisms, as well as harmful or potentially harmful effects (2,3). Determination of the net effects on humans (such as changes to life expectancies or risks of cancer) requires properly designed epidemiological studies. At low levels of exposure, net effects may be too small to discern, i.e. because of statistical difficulties of measuring them against variations not caused by radiation. Hence, although biologically positive effects (sometimes called "hormesis") have been demonstrated in the form of adaptive responses of cells and organisms to damage from radiation, these effects cannot be reliably expressed as reductions in the incidence of cancer (2). On the other hand, there has been no detectible excess of cancers attributable to radiation for the same conditions of exposure.

## PROTRACTED EXPOSURE TO RADIATION

A recent study (4) of protracted occupational exposures in the nuclear industry has shown that there is "no evidence of an association between radiation dose and mortality from all causes or from all cancers". At 100 mSv, there are dose-related increases in mortality from some cancers but these appear to be balanced by dose-related decreases for some other types of cancer.

The average rate of protracted public exposure to background radiation is about 2 mSv per year in Australia. In other parts of the world, it varies from less than 1 mSv per year to more than 15 mSv per year (2). Local populations living above some mineral deposits incur lifetime doses well in excess of 1,000 mSv from natural background radiation. The ICRP risk coefficient of  $5 \times 10^{-5}$  per mSv applied to a dose rate of 2 mSv per year gives a risk rate of 10 fatalities per hundred thousand per year, which is about 5% of the total rate of fatal cancers from all causes in Australia.

There are many variable factors other than radiation which affect the incidences of cancers. Nevertheless, on the basis of a linear extrapolation from figures in the foregoing paragraph, it would be expected that differences of dose rate over its range of natural variation would cause substantial differences in the total rates of cancers in the exposed populations. In fact, no correlation has been established between background radiation and rates of cancer (or genetic effects) in humans, suggesting that the ICRP recommendations do not apply to routine exposures within the range of background radiation rates. Effects, if they exist, are so small that they cannot be measured.

Even if radiation in the natural environment is having effects, it might be inappropriate to describe these effects as harmful. Human evolution has taken place in the presence of naturally occurring radiation and it is a fundamental tenet of evolutionary biology that organisms adapt to their environment (5). This means that, within the range of natural background, levels of survival and fitness should be expected which are optimum with respect to radiation.

## APPLICATIONS OF RISK ESTIMATES IN RADIATION PROTECTION

The practice of radiological protection is concerned mainly with two of its three general principles - compliance with dose limits and the optimisation of radiation protection practices. Compliance with these principles can reasonably be equated with "safety", because the total absence of risk is fundamentally not achievable in the practice of any potentially hazardous activity and therefore cannot be a legitimate objective of regulation.

The linear hypothesis is the basis for the establishment of individual dose limits and is

intentionally conservative for this purpose. Dose limits recommended by the ICRP (1) are actually at levels where the risk can only be estimated hypothetically. These dose limits provide a high level of protection for individuals whose exposures are reliably known.

However, the postulation of the linear hypothesis has given rise to the belief that any dose of radiation, no matter how small, increases the risks of cancer and other adverse health effects. In fact, there is no evidence to support this belief except for the linear hypothesis itself, which is an assumption, and radiation appears to have biologically positive (beneficial) effects which may predominate at low dose levels. When dealing with the effects of low doses, estimated in accordance with the linear hypothesis, optimisation may thus involve balancing real costs against hypothetical benefits which are too small to be demonstrated and might well be non-existent. This application of the linear hypothesis should be viewed with caution.

Applications of the linear hypothesis to justification, the third general principle of radiological protection, should also be viewed with caution. The justification of a practice requires that it produce sufficient benefit to offset the radiation detriment it causes (1). This goes far beyond the scope of radiological protection. Clearly, however, many of society's concerns about the justification for uses of nuclear energy relate to risks, and hence to detriments, which have been overestimated - perhaps greatly overestimated. Decisions made on this basis may not be in society's best interests.

## CONCLUSIONS

For doses up to at least 50 mSv received at a high rate in a single event by adults (or 10 mSv for exposure of the embryo and foetus) and up to at least 15 mSv per year received routinely, there is no proof that exposure to radiation increases the risk of cancer. Biologically positive and negative effects of radiation have been demonstrated in this range. However, because of statistical difficulties of measurement, they cannot be evaluated quantitatively in terms of changes to rates of cancer incidences. It is not likely that there is a discontinuity or cut-off to any of these effects at either high or low levels of exposure. The assumption that effects observed only at high doses and dose rates can be extrapolated to low doses and dose rates, to the exclusion of other effects, is questionable. It is not necessarily in the best interests of individuals or of society to make this assumption.

## REFERENCES

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