Review of the Controversy on Risks from Low Levels of Radiation

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SUMMARY: The need for regulation of low levels of radiation exposure, and the estimation of risks from such exposures, are based on the assumption that risk is proportional to dose without a threshold, the “linear no-threshold (LNT) hypothesis”. This assumption is not supported by scientific data. There is no clear evidence of harm from low levels of exposure, up to at least 20 mSv (acute dose) or total dose rates of at least 50 mSv per year. Even allowing for reasonable extrapolation from radiation levels at which harmful effects have been observed, the LNT assumption should not be used to estimate risks from doses less than 100 mSv. Laboratory and epidemiological evidence, and evolutionary expectations of biological effects from low level radiation, suggest that beneficial health effects (sometimes called “radiation hormesis”) are at least as likely as harmful effects from such exposures. Controversy on this matter strikes at the basis of radiation protection practice.

1. INTRODUCTION

It has been well established by the work of the Radiation Effects Research Foundation (RERF) in Hiroshima, Japan, that acute doses of ionising radiation greater than about 200 mSv cause an increase in the incidence of cancer in human populations. An “acute dose”, in this context, is a dose that is incurred at a very high rate in a short space of time, as in an atomic bomb explosion or in some medical procedures. The controversy to be addressed in this paper is whether there are risks from lower levels of exposure and from low dose rates.

Unfortunately, this has become a contentious issue even among scientists, with opinions often tending to be polarised. The author is simply an observer of this controversy and he is not presenting any new evidence in this paper. However, he is attempting to consider the evidence on both sides without prejudice.

2. LABORATORY AND CLINICAL EVIDENCE

Damage caused by ionising radiation to DNA in living cells can lead to risks of cancer in exposed persons and risks of hereditary effects in their descendants. The UK National Radiological Protection Board (NRPB) [1] and the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) [2] argue that even the lowest possible dose and dose rate (a single radiation track traversing a single living cell) might result in incorrectly repaired DNA damage and hence lead to cancer.

There is considerable evidence that low levels of radiation exposure may induce or activate cellular DNA repair capacity, additional to that which exists normally [3]. This “adaptive response” to radiation may reduce the effects of damage from subsequent doses of radiation or from other causes. The effect of single doses appears to be transitory. However, it might be expected that any such effect from chronic exposures would be on-going. Moreover, there are other effects of low level radiation at the cellular and molecular level, particularly apoptosis (see below).

There is also considerable evidence that a dose incurred at a low dose rate, or from intermittent exposures, is less likely to cause cancer than an acute exposure to the same total dose. One reason for this is that, the lower the rate of damage, the greater the chance for cellular DNA repair to be effective.

Experiments reported last year at the IRPA-10 Congress by Mitchel and Boreham [4] showed that human and rodent cells, exposed to low doses and/or low dose rates, responded by increasing their ability to correctly repair broken chromosomes. Cells unable to adequately repair their chromosomes were sensitised to die by apoptosis. These responses of cells reduced their risk of being transformed into cancer cells by exposure to radiation, and also protected them against their own inherent, spontaneous risk of transforming into cancer cells.

Most human cancers arise from DNA damage due to agents other than radiation. Hence, DNA repair systems exist primarily for damage other than radiation damage. If low doses of radiation enhance repair of any such damage, the net effect could be a reduction in overall risk, in spite of any risk increment due to the radiation itself. Such an effect, sometimes called “radiation hormesis”, has been observed in cells from virtually all types of organisms, in whole plants and animal species other than humans, and in human cells.

One of the more striking illustrations of an adaptive response to radiation has been reported by Sakamoto and his co-workers in Japan [5]. Beneficial effects in the treatment of cancer have been reported from repeated therapeutic whole-body and half-body doses of the order of 100-150 mGy. It has been suggested that this effect may be due to stimulation of the immune system and,
hence, that stimulation of the immune system by radiation might help in the treatment of diseases other than cancer. The use of radiation to achieve such effects is, of course, a matter for the medical profession to pursue but it has implications for the balance between benefit and harm in radiation protection.

Clearly, there are bio-positive effects of low level radiation, as well as any bio-negative effects that occur. The net effect, if there is one, may be too small to be of any practical significance. This is not simply a matter of thresholds to carcinogenesis or mutagenesis, although thresholds may exist. Any significant net effect on health should be expected to vary from person to person and it is unlikely that it would be known in advance for any specific individual. It is even conceivable that a specific exposure to radiation could be harmful to one person and beneficial to another. The net effect on an exposed population might be measurable only by epidemiology. One day, it may be possible to assess the probability of such an effect individually. Currently, epidemiology appears to be important in the evaluation of this probability, viz: as an average for members of the exposed population.

3. EPIDEMIOLOGICAL EVIDENCE

3.1 Exposures caused or enhanced by “man”

As mentioned earlier, it is well established that “man-made” doses of ionising radiation greater than about 200 mSv cause an increase in the incidence of cancer. Studies of the atomic bomb survivors and the consequences of some medical exposures provide evidence that risks of increased cancer may extend to doses less than 200 mSv [6,7], but with lower levels of statistical significance. Wakeford [8] believes there is evidence that risks exist down to about 10 mSv but this (like many other matters relating to the effects of low doses) is controversial.

Acute radiation sickness and early fatalities of workers who were at the Chernobyl reactor at the time of the accident in 1986, and thyroid cancers among children in the district around Chernobyl, were associated with high doses of radiation. Risks of thyroid cancer are still being evaluated. UNSCEAR [2] has reported no evidence of any other health effects attributable to radiation exposures from Chernobyl. In particular, there has been no increase in the incidence of cancer, apart from the child thyroid cancers. The incidence of leukaemia, one of the main concerns due to its short latency period (5-10 years after radiation exposure in adults), is not elevated in any of the exposed groups, including the emergency response workers.

This is despite the fact that about 100,000 workers involved in the emergency responses and cleanup of Chernobyl incurred doses in excess of 100 mSv, the average being about 170 for those employed in 1986. Several thousand of them probably received doses greater than 500 mSv. The average effective doses (excluding the thyroid) to more than 100,000 people evacuated from the most contaminated areas of Ukraine and Belarus were about 30 and 40 mSv respectively. The maximum estimated total dose commitment (due to the accident) for persons who were not relocated was reported to be 250 mSv, with the average for the 1,500 most highly exposed members of this group being 160.

A study [9] of cancer mortality among workers with protracted occupational exposures in the nuclear industry in other parts of the world (US, UK and Canada) has found that there is “no evidence of an association between radiation dose and mortality from all causes or from all cancers” for individual doses up to 100 mSv. Significant dose-related increases were found in mortality from multiple myeloma and most types of leukaemia, but these increases appeared to be balanced by dose-related decreases for some other types of cancer. Tests of statistical significance were applied to the increases in mortality which were observed but apparently not to the decreases.

Some other studies of this type, for example a recent study of health records of the Lucas Heights workforce [10], have shown that radiation workers have lower mortality rates from all causes and from all cancers than the general population (and sometimes lower than other workers). This is usually written-off as the “healthy worker” effect, but prompts the question: “are radiation workers more healthy because they are exposed to radiation?”

A clear risk of lung cancer, due to high levels of exposure to radon gas in poorly ventilated underground mines, has been observed from occupational exposures of uranium miners which occurred before the potential for such a hazard was recognised. Many studies have been conducted of the possible risk of lung cancer due to environmental radon in homes, which is usually at much lower levels than in underground uranium mines. The results range from positive to negative correlations. For example, Cohen [11] observed that the higher exposures to domestic radon correlated with the lower incidences of cancer (a negative correlation), whereas Lubin and Boice [12] have reported a positive correlation.

3.2 Natural exposures

Natural background radiation is the main source of exposure for most people, and it is therefore a major potential source of information on the effects of radiation exposure. As would be expected, chronic exposure appears to cause less risk than acute exposure to the same total dose. The dose rate from background radiation ranges around the world from...
less than 1 to more than 100 mSv per year locally, with lifetime doses up to several thousand mSv, but there is no conclusive evidence of increased incidences of cancer or other adverse health effects at the higher levels of radiation. Populations exposed to very high environmental dose rates for long periods, and for many generations, may well be too small for reliable statistically based conclusions to be drawn. However, it can reasonably be said that exposures to chronic dose rates up to (say) 50 mSv per year are not doing any demonstrable harm (“harm” as defined in paragraph 42 of reference [6]).

In fact, the reverse has been reported [11,13,14], viz: comparatively low rates of cancer have been observed where levels of radiation are comparatively high. For example, Jagger [13] has compared radiation levels and cancer mortality rates in three Gulf Coast states and three Rocky Mountain states of the US, and estimated that—

- the average natural background level of radiation is 3.2 times higher in the Rocky Mountain states, but the total cancer rate is 21% lower, than in the Gulf Coast states; and

- the average radon level in air is 3.9 to 5.2 times higher in the Rocky Mountain states, but the lung cancer rate is 31% lower, than in the Gulf Coast states.

A problem with epidemiological studies conducted in the USA is said to be that the population is far too mobile. This contrasts with the situation in much of China, where populations have been stationary for thousands of years and have moved little even recently because of restrictions on movement between regions. Of 150,000 Han peasants, living near each other for six generations, about half receive around three times the background radiation exposure of the other half. Various studies of doses and health effects have shown either that there are no discernible differences in the health of these populations or that mortality rates for some cancers and other adverse health effects are lower in the more highly exposed population.

In 1999, there was a preliminary report of a 9-year government funded study of 100,000 inhabitants of a small coastal area in Kerala, southern India, where background radiation (mainly due to thorium in beach sands) is up to 30 times the global average. Further information on this work has been filtering through, to the effect that no excess incidence of cancer had been found in the region and that cancer rates may be lower than in other parts of India where radiation levels are lower. The final report has not yet come to hand and it will be interesting to see whether biologically positive effects of radiation were found (or sought).

3.3 Discussion of epidemiology

The lack of clear evidence of risks from low doses of “man-made” or “man-enhanced” radiation may be because there are no harmful effects of radiation at such low levels or because the health effects, whatever they may be, are too few to be statistically significant. The “absence of evidence” of such effects should not be confused with “evidence of absence”.

On the other hand, there are many observations of apparently bio-positive effects of radiation in human populations, which are consistent with the laboratory observations of an adaptive response (discussed earlier) and the evolutionary expectation (discussed below). Hence, it is reasonable to postulate that radiation hormesis may occur in humans, although the reasons for it and its overall significance are not fully understood. Many scientists who have studied this matter consider that evidence of radiation hormesis in humans is conclusive. Luckey [14] suggests that the dose rate for optimal health is about 10 mSv per year. Others reject the possibility of hormesis out of hand, or ignore it.

It should be recognised that epidemiology tends to be a rather “blunt instrument” at the best of times and that some studies, which are said to be epidemiological, tend to be very fuzzy. Other studies are carefully case-controlled, with allowances for confounding factors built in from the beginning. It can be argued that none of the claimed observations of radiation hormesis in humans is the finding of a properly conducted epidemiological study. Jagger [13] himself (see findings above) states: “It is possible that confounding factors, such as smoking, poverty, or environmental pollution contribute to the differences... (but) the factor of disproportion is so great ... that it strains credulity to assume that such confounding factors could reverse ... (the) ... correlation.” Unfortunately, it appears that funds and effort for major epidemiological studies tend to be forthcoming only when harmful effects are to be investigated, not for beneficial effects. It has also been suggested that studies have sometimes been commissioned with the specific aim of demonstrating that harmful effects occur.

4. HEREDITARY EFFECTS

Where there is a risk of cancer, there is a risk of genetic damage, which might cause hereditary effects. At high doses and high dose rates, radiation certainly has discernible effects at the molecular and cellular level that change genetic material in living organisms, and there is substantial evidence of hereditary effects in non-human species. There is no direct evidence that radiation causes hereditary effects in humans, although it must be assumed that it may do so.
At low doses and low dose rates, the position is similar to that for cancers, as follows: Hereditary effects have many causes not attributable to ionising radiation, and no specific case could be identified with certainty as being caused by exposure to radiation. If there is a change in the total incidence of hereditary effects in humans, due to low levels of radiation exposure, it is too small a change to be determined.

Genetic effects of radiation may have been an important factor in the evolutionary process. The human race (as it now exists) represents only the small, successful part of all the trials and errors of evolution. Parsons argues that radiation hormesis must be expected because evolution has occurred in the presence of ionising radiation [15]. During the evolution of life on earth, natural background radiation has previously been substantially higher than it is today and it has ranged (in space and time) from less than 2 to significantly more than 20 mSv per year for the human race, during its evolution.

It is a fundamental tenet of evolutionary biology that organisms adapt to their environment. Fitness measures, including health, longevity and resistance to diseases, should be maximal for organisms in the habitats in which they normally occur. The evolutionary expectation leads to what Parsons calls “background radiation hormesis”, viz: because radiation is part of our normal environment on earth, it follows that fitness should be at its highest within the range of natural background radiation - somewhere between (say) 2 and 20 mSv per year. This effect has been well-documented in lower organisms.

Parsons also postulates, on the basis of evolutionary biology, that “stress-derived radiation hormesis” should be expected from short bursts at 100-1000 times background levels.

5. THE LNT HYPOTHESIS

For radiation protection purposes, the International Commission on Radiological Protection (ICRP) recommends the assumption that the risk of radiation induced cancer is proportional to dose without a threshold [6]. The use of this assumption, the "linear no-threshold (LNT) hypothesis", was reconfirmed in June 2000 by UNSCEAR [2]. However, the words “for radiation protection purposes” are essential. The LNT hypothesis is not intended to be used, for example, to estimate the number of casualties due to an accidental release of radioactive material (viz: multiplying very large numbers of people by the very small doses to which they might be exposed).

In June 2001, the American Nuclear Society (ANS) updated its own position statement on the health effects of low level radiation [16] to include the Position Statement "Radiation Risk in Perspective", issued by the Health Physics Society (HPS) in January 1996. It is now the position of the ANS that there is insufficient scientific evidence to support the use of the LNT hypothesis in the projection of the health effects of low level radiation. The ANS recommends that an independent, multidisciplinary group of reputable scientists should be established to conduct an open scientific review of all data and analyses on this subject and that new, interdisciplinary research should be initiated on the health effects of low-level radiation. In the meantime, the ANS agrees with the HPS that quantitative estimation of health risks should not be made for individual doses less than 50 mSv in one year, or less than 100 mSv in a lifetime, in addition to background radiation. The treatment of risks in this dose range should be strictly qualitative, accentuating a range of hypothetical health outcomes with an emphasis on the likely possibility of zero adverse health effects.

Mitchel and Boreham [4] have reported that no actual scientific data support the LNT approach at occupational and public exposure levels. They have tested the LNT hypothesis and found that it is not consistent with laboratory observations of the responses of plants and animals, and of human and animal cells, to low or chronic doses of low LET radiation. This does not, by itself, obviate the utility of the LNT hypothesis in the practice of radiation protection. The LNT hypothesis is simple to apply and, in so far as it defines the upper boundary to the range of uncertainty in radiological risk estimation, it is usually considered to be conservative. However, Mitchel and Boreham have also concluded: “data indicate that the use of the LNT hypothesis and ALARA* is not conservative, but may actually increase the overall risk of cancer.” This is a possibility that the radiation protection profession cannot afford to ignore.

Gonzalez [17] has pointed out that regulators can only regulate radiation exposure that is attributable to a particular source. Source-related exposures are incurred in addition to the prevalent background exposure, which is 2.4 mSv per year on average and typically up to 10. However, background radiation exceeds 100 mSv per year in some places. Given the ubiquitous nature of radiation and the practical fact that regulators cannot change standards from...
place to place, Gonzalez argues that the relevant exposure is the highest background level that exists. At those levels, the biological effects of radiation exposure can be observed under the microscope even though harm may not be evident. (Paragraph 42 of ICRP-60 [6] clearly distinguishes between change, damage and harm. Changes that can be observed are not necessarily harmful.)

There may well be a better case for applying the LNT hypothesis to dose increments above 100 mSv per year than there is for dose increments above 2.4 mSv per year. However, the logical conclusion to Gonzalez’ argument is that Australian authorities, who are planning the establishment of a radioactive waste repository where the background radiation is around (say) 2 mSv per year, should be constrained to ensure that the increment of individual dose from its operation is less than 0.1 mSv per year because background dose rates in some parts of the world (e.g. at Ramsar in Iran) are higher than 100 mSv per year. It might also be claimed that Gonzalez’ argument justifies the expenditure of billions of dollars to meet an environmental protection standard of 0.15 mSv per year in the clean up of contaminated sites in the US, regardless of whether there are or are not risks from such exposures. Perhaps it would be controversial to suggest that national regulatory authorities could set standards to suit conditions within their national boundaries.

6. CONCLUDING REMARKS

It is generally agreed that the lowest dose at which a statistically significant increase in the incidence of cancer has been shown to occur is about 100 mSv. This risk has been observed from doses incurred at very high dose rates, as in an atomic bomb explosion or from some medical procedures. There is a wide range of disagreement about the biological effects of lower doses and low dose rates. In recent years, it has become clear that many scientists are concerned that the practices of radiation protection and regulation, applied to low levels of radiation, are not consistent with the scientific evidence.

The main reason for the belief that there are risks from low level radiation is that the ICRP recommends the use of the LNT hypothesis in the practice of radiation protection. Unfortunately, many people are now placing an entirely unwarranted level of confidence in the accuracy and validity of risks that are estimated using it. The LNT assumption is almost certainly not correct, but is claimed by its advocates to be a reasonable and conservative approximation. Others have criticised it as being seriously misleading in some applications.

Controversy about the health effects of low levels of ionising radiation, and the LNT hypothesis in particular, has become polarised to an extent which should be considered unacceptable in a rational community. On the one hand, ICRP-adherents promote the view that LNT can be extrapolated reliably, over more than four orders of magnitude in terms of dose and over an even greater range in terms of dose rate, to estimate cancer fatality risks from dose rates down to zero – or, at least, down to 0.02 mSv per year. Those who disagree are at risk of being ostracised.

At the other extreme, there are claims that public fears about low level radiation are being deliberately exploited for a variety of improper purposes. One critic of LNT in the US has stated that the head of a national radiation protection authority accused him, at an international conference, of trying “to kill the golden goose”.

The radiation protection profession is the meat in this sandwich. For its members, the LNT hypothesis is a convenient tool, without which their work would be difficult to perform. Most of them are not involved in research. Their job is to apply nationally approved standards, not to question those standards or evaluate the results of research or develop independent ideas about radiological risks.

It does not seem possible to know for sure whether there are risks from low level radiation, although some people are obviously convinced that they do. On the other hand, it may be possible to find out for certain whether there can be health benefits from exposure to low level radiation. There is a lot that we might be able to find out about radiation hormesis, if we chose to do so, for example:

- Does radiation hormesis really occur in humans?
- If so, what are the relevant dose-response relationships?

Mitchel and Boreham [4] appear to be making good progress in laboratory investigations of the biological mechanisms that might explain, and help to quantify, hormesis in human populations. However, most (if not all) significant epidemiological studies appear to be directed toward the investigation of harm, not hormesis. Funding and effort should be provided for more objectively based studies, and opportunities for such work do exist. Apart from those small areas of the world (like Kerala and Ramsar) where background radiation levels are extremely high, and the different regions of the USA (which are the subject of Jagger’s paper [13]) with many millions of population and good health records, there are significant areas in the UK (for example) and elsewhere in Europe where background radiation levels are high.

I have been told that Boulder, Colorado, where the background dose rate is around three times the national average for the US, is home to one of the principle centres of the Green movement in that country. This prompts me to leave you with the
question: “Would any of you let an environmental dose rate between 1 and 10 mSv per year determine your choice of where to live?”

References


