

The safety of nuclear radiation;
a careful re-examination for a world facing climate change

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Education and understanding are slow incremental processes. The bombing of Hiroshima and Nagasaki in 1945 generated a shockwave in the public understanding of science. This shock damaged understanding, not only among the public, but also within the scientific community and created the spontaneously polarised view that nuclear radiation is intrinsically different from other hazards. This view was cultivated during the Cold War and today a quite exceptional attitude is still taken to its safety despite widespread use of high doses in medicine. With current knowledge such an attitude is not justifiable and furthermore with the onset of climate change difficult choices have to be made.

In simple terms the effect of ionising radiation on life may be described by a plot of damage against dose. Such characteristic curves may be made for single cells examined *in vitro*, for individual organs and for complete creatures. In the absence of perfect data some assumptions have to be made. The mathematically simplest solution is that the characteristic is linear, a straight line through the origin with slope α ,

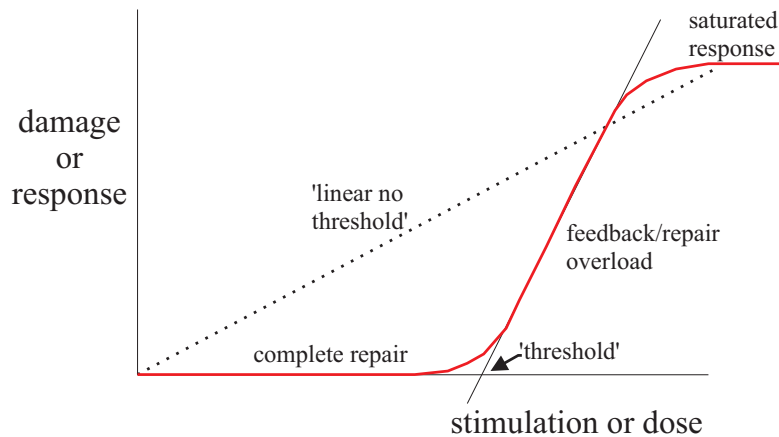
$$\text{damage} = \alpha \times \text{dose} .$$

This is called the Linear No Threshold (LNT) assumption and the consequence of adopting it is made clear in this quotation[1] from Sir Richard Doll,

Whether there is a threshold dose below which no effect is produced is still open to doubt, but on present knowledge it seems unlikely that any such threshold exists. It must, therefore, be assumed that even very small doses produce some small risk.

However, mathematical simplicity is unusual in the natural world and such an assumption cannot be justified unless uniquely required by observational data.

The usual characteristic response of living organisms to a source of danger is quite different from LNT and includes the effect of any repair and recovery mechanisms that have evolved for its protection. An elementary example is the response to laceration. The body has evolved a response to minor cuts and grazes such that a week or two later complete recovery is effected, but above a certain threshold the repair mechanism is over-stressed and a permanent response in the form of scar tissue remains. Although full functionality may appear to be restored, the distorted scar tissue may cause trouble in later life. Severe laceration may cause permanent loss of function, and even death, beyond which the response saturates. The result is an S-shaped dose-response characteristic, sketched in the figure below. Such characteristics are not unique to living systems. For example, a well designed bridge may sway elastically when blown by wind and weather, and only if stressed beyond a certain threshold is damage sustained. The safety of a bridge is not described by an LNT characteristic because recovery is assured for the effects of the elements below the threshold.



So the important question is whether life responds to nuclear radiation in the same way. For this S-response to be applicable we need to be assured that

- life has evolved in a radiation environment, thereby giving it the opportunity to develop repair and protection mechanisms;
- the repair mechanisms are known and understood, at least in outline;
- available data are consistent with this S-shaped behaviour.

Such data may then be used to determine values for threshold and repair time, and also a value for the maximum dose consistent with tolerable damage levels.

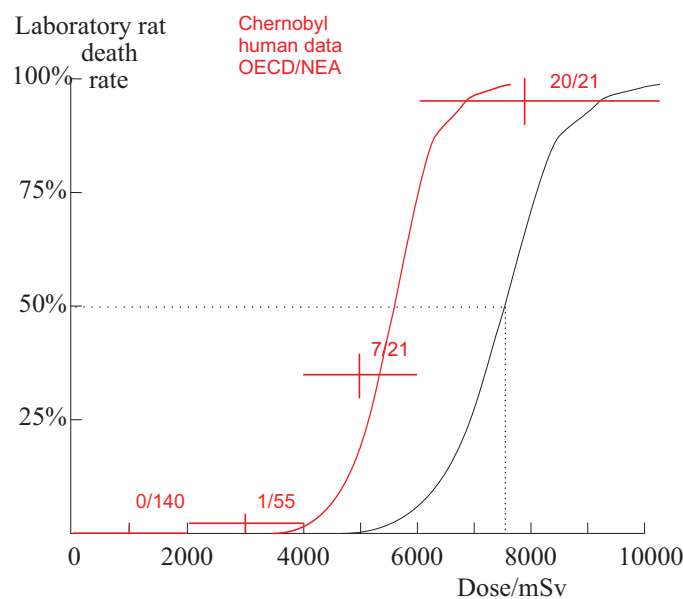
The human environment is bathed in low levels of ionising radiation[2]. On average this level is about 2.5 mSv per year.¹ Of this, 15% is the mean medical dose and the rest comes from space in the form of cosmic rays and from radioactive decays in natural rock and materials within and around us. These decays generate γ -radiation and radon gas which itself is radioactive. Radon may be inhaled and absorbed in the lungs where further decay can cause damage. These sources of natural radiation have stimulated the evolution of mechanisms that prevent long term damage by continual repair.

What do we know of such repair mechanisms? There follows a heavily simplified description for the non-biologist. Nuclear radiation damages the DNA structure that records the form in which life propagates. The first level of protection at the cell level is to maintain many copies of these DNA records. In laboratory experiments it is found that cells that have recently divided and have fewer copies are more radiation sensitive. The second level is to repair single breaks in strands of DNA. This work is carried out by enzymes which complete their job on a timescale of hours. This still leaves DNA that have multiple breaks; by their nature these are non-linear in the local radiation deposition. Often dysfunctional they are usually swept away by the process of cell replacement which takes place over a period of days, weeks or months, depending on age and the tissue concerned. Residual damaged or mis-repaired chromosomes may persist – such scar material is often used to determine the history of radiation dose. However, the relation between radiation dose and damaged chromosome count is found to be non-linear as would be expected. Independent of their detail the existence of these repair mechanisms is at variance with the LNT characteristic.

¹ The unit, the milli-sievert (mSv), represents the radiation that inflicts the same biological damage as the absorption of 1 mJ per kg of X-rays.

Non-linearity of response to radiation dose would lead us to expect that damage was reduced when the same deposited energy is dispersed in either space or time. Both of these effects are well established.

- The effect of dispersal in space has the effect that a few heavily ionising charged particles do much more damage than X-rays or electrons depositing the same energy per kg. The ratio, the relative biological effectiveness, can be as high as 20.
- Doses spread in time, termed fractions, are used in radiotherapy and allow healthy peripheral tissue some time, typically a day, to recover while the tumour tissue with its higher dose has less opportunity to do so. In simplest terms the ratio of multiple to single strand breaks is much larger in the heavily irradiated tumour. But for this non-linear mechanism, the success rate of deep cancer radiotherapy with γ -rays would be very poor indeed.



The black curve in the figure shows the survival rate of laboratory rats exposed to a single radiation dose. The experimentally determined S-curve indicates that 50% die from an exposure of 7000 mSv. The equivalent value for humans is about 5500 mSv and a correspondingly scaled curve is drawn in red. The red data points are for the fatalities of the 238 acute cases at Chernobyl.[3] Of those receiving doses above 6000 mSv, 20 out of 21 died in a few

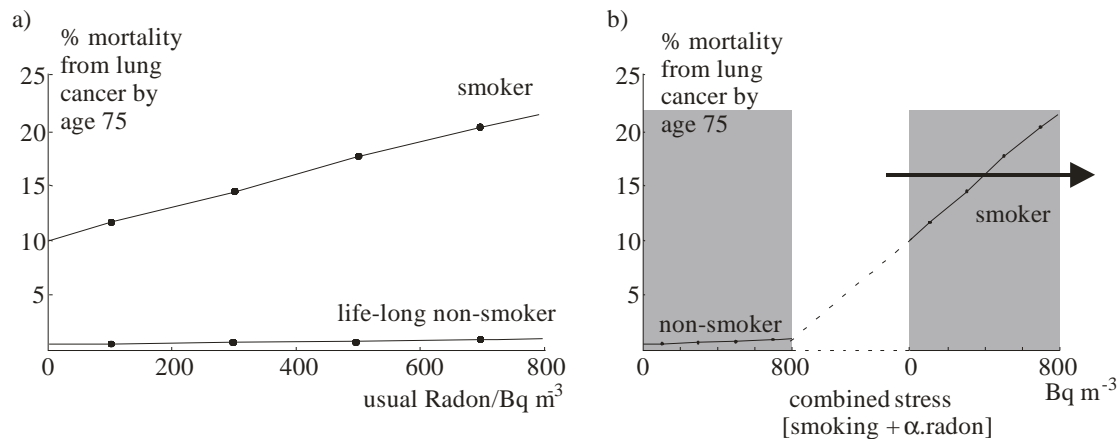
weeks. Of those receiving less than 4000 mSv, only 1 died in the immediate aftermath. (A further 11 died in the following 15 years from a variety of conditions.)

Dose/mSv	Number	Leukaemia deaths	Risk per 10,000	Number	Solid cancer deaths	Risk per 10,000
0-5	35458	73(64)	3±3	39507	4270(4268)	0±20
5-100	32915	59(62)	-1±3	29960	3387(3343)	15±20
100-200	5613	11(11)	0±10	5949	732(691)	70±45
200-500	6342	27(12)	24±10	6380	815(716)	155±45
500-1000	3425	23(7)	46±16	3426	378(262)	340±60
1000-2000	1914	26(4)	120±30	1764	326(213)	640±100
>2000	905	20(2)	310±60	625	114(58)	900±170

The health of a large number of the survivors of Hiroshima & Nagasaki has been followed for 50 years.[5,6] These data cover many conditions; here we consider just the incidence of leukaemia and solid cancers. The table above shows the number of people in each dose range and the number of those who contracted disease over the period 1950-1990 compared to the number expected from similar populations who were not irradiated, given in brackets. The difference per 10,000 population is given

with statistical random error. Assuming that an outcome that afflicts 10 or less people in 10,000 over a lifetime is acceptable, only survivors with doses greater than 200 mSv (leukaemia) and 100 mSv (solid cancers) are significantly affected by the irradiation. We note that this is at the limit of statistical validity of these data, largely due to the incidence of solid cancer from other causes.

The result of a single dose of radiation at Hiroshima and Nagasaki, primarily neutrons, gammas and betas, could be quite different to the steady lifelong exposure to radon experienced by the inhabitants of certain areas, for instance, Devon and Cornwall, the Massif Centrale and various parts of Czechoslovakia. They receive three or more times the average normal radiation levels through the alpha-decay of radon in their lungs. An important question then is whether the incidence of lung cancer is higher in Devon and Cornwall, for instance, than elsewhere. The answer is negative.² In a recent paper Darby et al [4] combined the data from 13 studies across Europe in search of a significant effect based on 7148 cases of lung cancer and 14,208 controls. The plot a) below summarises their analysis which shows the large effect of smoking on lung cancer.



The most general LNT analysis based on the two causes is

$$\text{prob of cancer by 75} = A + B \times \text{radon} + C \times \text{smoking}, \text{ where}$$

A , the chance of lung cancer without radon or smoking, is 0.4%;

C , the chance of cancer due to smoking alone, is 10%;

B , the gradient describes the dependence on radon.

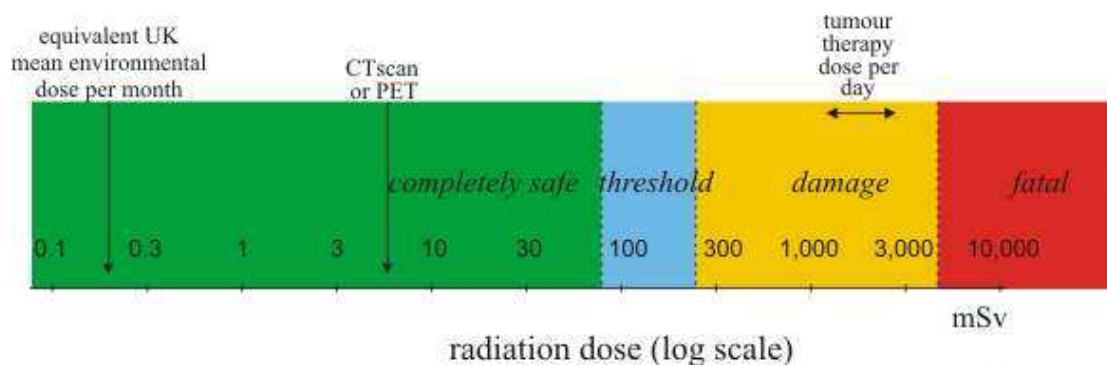
In any linear model the two lines must have the same slope, B . In spite of this Darby et al claimed that these data are consistent with LNT. The data have a simple interpretation in terms of a non-linear S-shaped response to the combined stress of the two causes. This is illustrated in diagram b) where the smoker data has been shifted to the right to show qualitatively the response to the combined stress. The response is consistent with a single S-shape. The conclusion is that for non-smokers radon is not a cause of lung cancer (less than ~1-2 per 1000 before age 75). Smoking however is a serious cause and exposure to radon simply exacerbates the stress which already exceeds threshold. This suggests that resources spent on radon protection would be more effectively spent persuading people not to smoke.

² In some studies it has been suggested that low levels of steady radiation have the effect of improving resistance to further doses. The possibility should not be lightly dismissed. The enhancement of resistance by stimulation is the basis of vaccination. It is reasonable that the repair of DNA strand breaks might be similarly stimulated and there are some interesting laboratory studies to support this.

Existing views die hard. The IAEA Safety Standards 115 [7] states that “Basic Principles: The total impact of the radiation exposure due to a given practice or source depends on the number of individuals exposed and on the doses they receive.” That is clear, but then it continues “The collective dose, defined as the summation of the products of the mean dose in the various groups of exposed people and the number of individuals in each group, may therefore be used to characterize the radiation impact of a practice or source. The unit of collective dose is the man-sievert (man-Sv).” (underline added)

It is not acceptable to argue that, because the damage is a function of dose and population, it is therefore a function of their product. Once this Collective Dose has been defined, people start to use it although nothing depends on it. Regulations and legislation are drawn up in terms of it, and it starts a life of its own, though removed from relation to any real risk.

We consider a simple illustration that shows how wrong such an argument can be. Blood is vital, and in an accident the more people loose blood and the more blood they loose, the worse is the accident. However, we should not introduce the collective blood loss measured in man-litres. Five man-litres loss to one person is fatal. Five man-litres spread amongst ten people is regular risk-free blood donation. The introduction of safety regulations based on collective blood loss would be unworkable and, given time for recovery, blood loss gets replaced. Radiation damage gets repaired too and the use of Collective Dose in radiation protection distorts risk estimation and generates fear of danger where none exists.



There is a factor just over twenty between the threshold dose level of 100-200 mSv and the fatal level of 5500 mSv.³ The diagram above shows the regions of threshold, damage and fatality for radiation on a logarithmic scale. It marks the range of dose, just a factor of two, between the therapeutic dose to a tumour and to surrounding healthy tissue, underlining the difficulty of inflicting fatality on the one without generating too much scar tissue on the other. Single diagnostic radiation doses are very far below the threshold and therefore quite safe. To estimate a threshold for a continuous dose, we need the effective time during which dose accumulates. This is the recovery repair time. Such repair times vary with tissue and age. We note that in radiotherapy fractions are given each day in the course of which recovery occurs. However, it is a matter of general experience that the human body can take up to a month to recuperate after an accident. A conservative estimate would be to compare the radiation exposure in a month with the single dose threshold. Thus 2.5 mSv per

³ A similar factor applies both to blood supply and to changes in body temperature. Thus, efficient cooling mechanisms keep body temperature stabilised to within 1°C. The threshold of damage is known to be about 2°C; excursions of 20°C cause coagulative necrosis – death by melting.

year (or 0.2 mSv per month) is about a factor 1000 below threshold (200 mSv). So environmental regulations that attempt to keep variations in radiation exposure to a fraction of the natural level are over cautious by a factor⁴ of about 500-1000. This factor is unnecessary and unaffordable. In no other field is such a safety factor applied. Members of the public tolerate radiation exposure⁵ up to 1000 mSv per day for their own health, while they have been lead to understand that an extra 1 mSv per year is unacceptable in the environment. A far greater tolerance to radiation in the environment is needed if the health of the planet is to be treated with the same respect and judgment as personal health.

At Chernobyl there was a major accident.[3] As already discussed 28 died in the early months from large doses in excess of 4000 mSv. In addition there were 1036 cases of childhood thyroid cancer caused by ingestion of radioactive iodine. Incidence peaked in 1995 and has since declined. As of 1998 there had been 3 deaths. These cancers would have been avoided by the provision of iodine tablets in the immediate aftermath. From the statistics of those receiving doses above threshold (8484 in range 100-200 mSv and 1123 above 200mSv) and the data from the Hiroshima and Nagasaki survivors one may calculate the number of extra deaths over 50 years from leukaemia (3) and solid cancers (78). This estimate is obviously very crude but is unbiased. Most of the other major effects of the disaster were caused by the disintegration of the socio-economic structure, in the form of lack of information and advice (eg about iodine tablets), political paralysis, official over-reaction and then panic and distrust by the public.

With properly designed nuclear reactors the likelihood of further accidents like Chernobyl should be seriously reduced. With liberal provision of iodine tablets and proper public education and services the consequences of any such incident would be small on the scale of world accidents. Even as it was, the Chernobyl nuclear accident was less serious than the Bhopal chemical disaster in which over 3800 people lost their lives. Both accidents were small compared to the likely effect of climate change. The costs of nuclear reactor decommissioning and waste storage would be eased substantially with a more rational approach to nuclear safety. Further, the power of nuclear threats by terrorists and rogue states would be devalued if public opinion came to accept that risks due to nuclear radiation were much less significant at low levels. After all, in a medical context it already accepts a rather high level.

References

- [1] 'Epidemiological evidence of effects of small doses of ionising radiation with a note on the causation of clusters of childhood leukaemia', R Doll 1993 *J. Radiol. Prot.* **13** 233-241
- [2] Publications of the UK Health Protection Agency (HPA, formerly NRPB)
- [3] OECD/NEA report on Chernobyl, www.nea.fr/html/rp/chernobyl/chernobyl.htm
- [4] 'Radon in homes and risk of lung cancer; collaborative analysis of individual data from 13 European case-controlled studies' S Darby et al, *BMJ* (21 Dec 2004)
- [5] 'Radiation & Health', T Henriksen and HD Maillie, Taylor & Francis (2003)
- [6] 'Fundamental Physics for Probing and Imaging' Wade Allison, Oxford (2006)
- [7] IAEA website, http://www-pub.iaea.org/MTCD/publications/PDF/SS-115-Web/Pub996_web-1a.pdf

⁴ In this paper the numbers may be debated by factors of two but the powers of ten are robust.

⁵ The radiation levels used in radiotherapy create significant scar tissue. Current research and development using highly charged ion beams and high intensity focussed ultrasound can be expected to improve therapy for deep cancers over the next few years.