False Alarm or Public Health Hazard?: Chronic Low-Dose External Radiation Exposure

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Proponents of a practical threshold for radiogenic risk have ignored epidemiological findings of excess cancers among workers and have generalized about the effects of high doses from in vitro studies of DNA repair mechanisms. Aggregate studies of occupational exposures and of children x-rayed in utero show that the proposition of a safe dose range or dose rate is false. The repair system of the mammalian cell is never perfect. Epidemiological studies of exposed persons that have been accepted in the scientific literature show a statistically significant increase in cancer incidence in the exposed population, supporting the claim that very low doses cannot be regarded as safe with respect to cancer induction. [*M&GS* 1998;5:14-21]

Ionizing radiation from external sources, and the biological effects of such radiation, have been studied for more than a century. While acute health effects (e.g., burns, nausea, hair loss, bleeding, etc.) occur only at high-dose exposures, various official radiation commissions [1,2] now generally accept that delayed detriment due to mutations in the cellular DNA has been established at low doses, down to about 20 cSv (rem) for adults and less than 1 cSv for fetuses.

These mutational (or stochastic) biological effects, in which the level of dose determines the level of probability or likelihood of occurrence of the effect, can lead to initiation or promotion of malignancies (somatic effects such as cancers and leukemia) or to genetic defects in subsequent generations.

Some recent reviewers of radiation health effects have asserted the existence of an "effective" or "practical" threshold for radiogenic risk below about 10-20 cSv [3,4,5]. In doing so, however, they have:

- ignored epidemiological findings of excess cancers among workers occupationally exposed to doses comparable to natural background, and
- generalized from in vitro (laboratory) studies of DNA repair mechanisms in animal and human cells mostly at rather high doses.

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The Presumed Safe Dose Threshold: Downward to Zero?

These experts argue that current radiation standards cost too much while providing no benefit to public health. For all practical purposes, such an assertion implies perfect repair of radiogenic DNA damage at low doses of low-LET (linear energy transfer) radiation (beta, gamma, or X-rays).

Historically, the presumed safe dose threshold has had to be adjusted downward several times, following updates in epidemiological findings of cancer mortality at low doses, primarily among A-bomb survivors. These adjustments have led to revisions of the "allowable" occupational exposures from 30 cGy/year at the start of the Manhattan Project in 1942 to 2 cGy/year in 1990 (Figure 1) [6,7]. Thus, the notion of a safe dose threshold for stochastic radiation effects is a historical legacy passed on from scientists who were active during that era to those they trained.

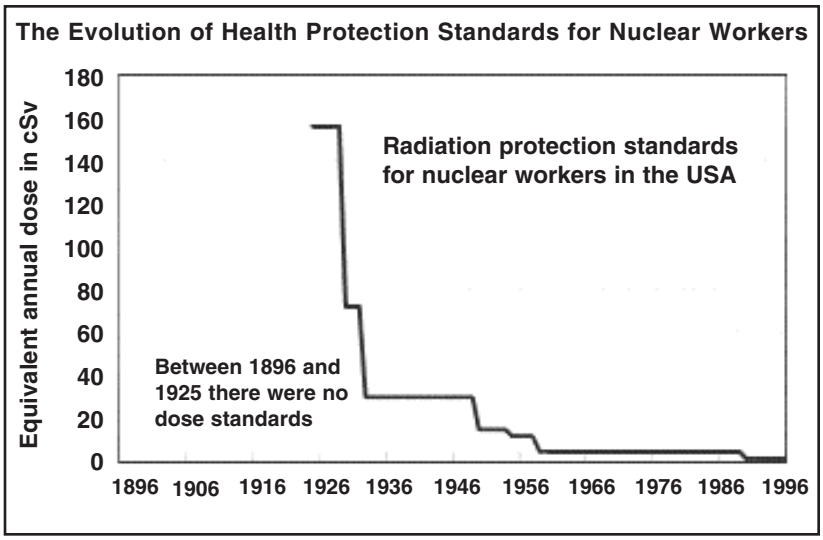

Figure 1: The evolution of health protection standards for nuclear workers is shown. It can be seen that international and national radiation protection commissions have had to revise their recommendations repeatedly in the past.Source: U.S. Department of Energy, Office of Environmental Management [7].

Defining "Safe Dose"

Such a tradition has led some scientists to suggest the existence of a practical dose threshold for low-LET radiation [3,4,5,8,9]. Others have expressed the opinion that the data have been inconclusive on the question of whether a safe dose threshold exists or not [2,10].

A safe dose and safe dose rate of ionizing radiation (i.e., zero radiogenic risk) means that all exposed persons remain unharmed during and after the exposure. In other words, no one will suffer from a radiation induced cancer or die prematurely from other radiogenic disease. A non-zero risk at any dose or dose rate, on the other hand, means that no one is safe during exposure and afterward; a certain fraction of exposed persons will suffer from radiation induced cancer and die prematurely, whereas the rest will remain unharmed.

Much is at stake in considering the existence of a harmless dose threshold. If this notion is a fallacy, as is asserted here, then raising the present radiation safety standards—as advocated by the Health Physics Society [11]—would lead to an even larger increase of cancers and genetic defects worldwide over those already initiated by past radioactive releases. Such an action would be indefensible.

In contrast, the present authors conclude that the proposition of a safe dose range, a safe dose rate, or a reduced biological effectiveness at protracted low-dose exposures has been shown to be false. This firm conclusion is based on an aggregate of independent and diverse findings, such as studies of excess cancers (including leukemias and thyroid cancers) in nuclear workers exposed to accumulated occupational doses comparable to natural background or in children who had been x-rayed in utero at acute doses of a few tenths of cGy (rem) [12,13].

From a worldwide perspective, genetic effects are of even greater potential consequence for public health than the induction of somatic malignancies discussed above. Ample evidence is accumulating of chromosome aberrations induced by low doses without threshold that carry a high probability for transmitting detriment to future generations [12,13,14,15]. Recent studies found evidence for generationally delayed detriment as a consequence of radiogenically induced genomic instability [16]. Radiobiological studies on human cell models in vitro, at low doses and at varying dose rates, have also been consistent with epidemiological studies in contradicting both a conjectured reduced biological effectiveness at low doses and low dose rates and a safe dose threshold [17,18].

The conclusion reached here is that any increase in radiation exposure above unavoidable background leads to significant added risks for somatic and/or genetic health detriment whether for populations at

large (e.g., from venting or fallout from weapons tests and from releases from nuclear production sites or waste repositories) or for individuals (e.g., from occupational or medical exposures). Comprehensive and independent assessment of risks versus expected benefits will pose enormous ethical, economic, and political challenges to present national and international institutions, both public and private, which constitutionally or by international agreements have been entrusted with serving the people's well being.

A Microdosimetric Argument

By combining microdosimetric considerations for the induction of mutations in the cell nucleus with low-dose epidemiological findings, an even more compelling argument against the existence of a safe dose threshold can be made as follows [19].

1. The dose from low-LET ionizing radiation is delivered by high speed electrons (Compton-electrons and photo-electrons) traveling through human cells and creating primary ionization tracks. One such track is the least possible disturbance that can occur at the cellular level. A "high dose" means many tracks per cell; "low dose" means few tracks per cell; "low dose rate" means few tracks per cell per unit time. At any dose, including background, some cells and cell nuclei are being transversed by ionizing tracks.

2. Radiation-induced carcinogenic alterations are alterations in the genetic material of the cell, the DNA. Cancer initiation or promotion is a unicellular process, following the rules of chance. Every track, independently of every other track, has a chance of inducing cancer by creating such alterations. The energy deposited by a primary interaction is a multiple of characteristic chemical binding energies in organic molecules.

3. This implies that there is a non-zero likelihood for cancer initiation or promotion at any dose or dose rate. If every potentially carcinogenic alteration induced by tracks at low doses or low rates were successfully and invariably "undone" by repair processes, however, then there would be an inherently safe dose and dose rate. The key question is: Does repair of carcinogenic injuries operate flawlessly when the dose is sufficiently low and slow?

4. If a radiation dose is received at a low enough rate, i.e. in the time frame required for repair, and if repair were to operate flawlessly and were to leave no carcinogenic or genetic damage, then the net effect of that radiation dose relative to a detrimental effect would obviously be zero by definition and many such small doses, allowing for repair in between, could be absorbed without increasing radiogenic risk.

5. Epidemiological evidence shows, however, that repair fails to prevent radiation induced cancers, even at doses where the repair system has to deal with only one or a few tracks per cell at a time, and even at dose rates that allow ample time for repair before the arrival of additional tracks. By any reasonable standard such evidence is proof that there exists no perfectly safe dose or dose rate.

Table 1: Tissue Dose in Centi-Gray when the Average Track-Rate per Cell Nucleus is One

Origin of Electron Packets	Average Nuclear Traversals per per Nucleus in Delivery of 1 cGy	Dose in cGy when Average Track-Rate per Nucleus is one
30 keV X-rays	1.34	0.75 cGy
250 keV X-rays	2.30	0.43 cGy
596 keV gammas Radium-226	2.94	0.34 cGy
662 keV gammas Cesium-137	3.16	0.32 cGy
1608 keV gammas A-Bomb	5.41	0.19 cGy

Tracking Imperfections of Cell Repair

In order to consider the meaning of dose at the cellular level we must relate the number of primary ionization tracks traversing a cell nucleus to a given dose. The smallest possible dose is not a fraction of a Gray but a single traversal of an ionizing track through the cell nucleus.

As we know, the energy of x-rays and gamma-rays is deposited in biological material via Compton-electrons and photo-electrons. One can, therefore, use the calculations of Paretzke and a recursion method [20] to convert the energy of an x-ray or gamma-ray into a number of electrons and their energy distribution. Thus, it is possible to convert the original photon energy to electrons and to calculate their summarized range [21,22].

With help of the relation 1cGy = 6.24 x 1010 keV/g one can now determine how many photons of a given energy are required to deposit a dose of 1 cGy. With this information the corresponding number of electron tracks is obtained.

Since the average dimensions of a mammalian cell and its nucleus are known [23], we can calculate the number of nuclear traversals per dose unit for X- and gamma-radiation of different origin [Table 1].

Gaps in the Repair Process

We know from numerous experiments with model systems that enzymatic repair processes are seen to work without impairment even at doses of a few Grays [24,25,26,27,28]. Furthermore, it has been confirmed repeatedly in studies with human cells in vitro that repair is achieved within six hours or less even after doses of several Grays [29,30,31,32]. There is also confirmed information on the number and type of DNA lesions.

There are, however, numerous references in the literature supporting the assertion that certain DNA lesions are not repaired or are misrepaired[1]. For example:

- The UNSCEAR-Report 1986 [33] states the following about repaired, unrepaired, and misrepaired carcinogenic lesions induced by radiation: "The error-free repair of the DNA, the most likely target involved, leaves some fraction of the damage unrepaired and the error-prone repair may produce misrepaired sequences in the DNA-structure."
- Kellerer describes a type of radiation-induced DNA damage that would be difficult to repair [25]: "A simple example would be neighboring single-strand breaks in complementary strands of DNA, which interfere with excision repair."
- This is confirmed by Feinendegen et al [30] who, reporting on irradiated cells, say "not all double-strand breaks are fully repaired."

With the information discussed so far, we can examine whether there is or is not any safe dose.

1. There are many repair pathways in a normal and transformed mammalian cell. The enzymes responsible for detecting and eliminating lesions like strand breaks, base loss and so on depend on the correct information in the complementary DNA strand. If, however, radiation damage affects both strands at the same place and at the same time, enzymes no longer can repair such lesions although the enzymes molecules themselves are completely intact. These so-called sites of multiple damage are frequently induced by ionizing radiation. Repair of

Challenging the Notion of a Safe Threshold

Imagine the following scenario, in which the repair processes are presumed to work flawlessly up to a certain dose of a few cSv (100 cSv = 1 Sv).

1. A number of individuals are exposed to a small dose no greater than this limit on Monday. All induced lesions in the DNA are flawlessly repaired within a few hours. No increase of cancer risk results from this exposure.

2. On Tuesday there is another exposure with the same small dose. Since we assume that the repair systems are working free of error, there is no increase in cancer risk after the first two doses. On the following days additional dose fractions are given, and so on, until a certain accumulated dose is reached.

In this scenario the individuals could accumulate rather high doses in many small dose fractions. No increased cancer risk should be detectable, however, in a long term follow up. Since it is acknowledged that the accumulated high dose, given all at once, will increase the cancer risk, we would have to conclude that each of the small dose fractions is harmless and that a dose threshold and a safe dose rate would indeed be real.

If, however, the long term followup studies were to reveal increased cancer incidence in the population exposed only to small dose fractions over a long time period, then the presumption of an error-free repair system, even at low doses, would be untenable. Also, the idea of a safe dose threshold would be wrong.

Dose fractions or doses, respectively, and the derived number of tracks per cell nucleus per exposure, drawn from a number of epidemiological studies of exposed persons that have been accepted in the scientific literature, are compiled in Table 2. In all nine studies a statistically significant increase in cancer incidence was observed in the exposed population.

These studies show that the following doses cannot be regarded as safe with respect to cancer induction:

9 cSv, 7.5 cSv, 4.6 cSv, 1.6 cSv, 1 cSv, 0.9 cSv, 0.5 and 0.1 cSv

We can conclude, therefore, that whenever an ionizing track traverses a nucleus of

a mammalian cell there is always a non-zero chance that it will cause a carcinogenic lesion and that the lesion will remain unrepaired, will be inherently unrepairable, or will be misrepaired. In short, there is an intrinsic failure rate in the repair system even at the lowest conceivable doses and rates.

The Flawed Case for a Safe Dose

In summary, for the essential stochastic end points of radiation damage (cancer induction and mutation) the idea of a safe dose threshold and of a safe dose range must be given up. According to present epidemiological data, only the linear (curve 3) or the supralinear (curve 4) dose-effect relationships shown in Figure 2 are consistent with scientific evidence from human data. Recently, radiobiologists have also come to the conclusion that cancer can be initiated as a result of a single radiation track through a single cell nucleus [44].

The proponents of dose thresholds and even of hormetic effects will argue that there are many studies in which no statistically significant radiation effect was found by the authors. These studies are, however, unsuited for deciding whether there is a dose threshold or not. Inability to find a significant effect can never be an argument for a safe dose. In many of these studies the followup periods were too short, the size of the cohorts was too small, or important confounding factors were not properly taken into account.

A group of independent scientists (physicians and epidemiologists), assembled and sponsored by Physicians for Social Responsibility, have critically reviewed 124 epidemiological studies supported or financed by the U.S. Department of Energy and/or by the British Government and have found that they are decisively flawed and "tend to produce false negative results"[45].

It is no surprise, therefore, that a large number of government-sponsored epidemiological mortality studies show no significant association between cancer induction and low dose radiation exposure.

Conclusions

Combining the known mechanism of low-LET interactions in human cells with findings from several independent epidemiological studies clearly shows that the repair

Table 2: Average Tracks per Cell Nucleus from each Exposure in Nine Human studies

Nr.	Study	Organdose [cSv]/Exposure	Tracks per Cell Nucleus at 1 cSv	Average Number of Tracks per Cell Nucleus from each Exposure
1	Nova Scotia Fluoroscopy [31]	7.5	1.34	10.03 Rounded : 10
2	Israeli Scalp-Irradiation [32] (Authors' revised estimate)	7.5 9.0	1.34	10.03 Rounded : 10 12.04 Rounded : 12
3	Massachusetts Fluoroscopy [33]	4.6	1.34	6.15 Rounded : 6
4	Canadian Fluoroscopy [34]	4.6	1.34	6.15 Rounded : 6
5	Stewart In-Utero Series [35-37]	0.5	1.34	0.67 <one ; 51% with one track
6	MacMahon In-Utero Series [38]	0.9	1.34	1.20 ~ one
7	British Luminizers [39]	0.1	2.94	0.29 < one; 75% with no track
8	Harvey Twins In-Utero Series [40]	1.0	1.34	1.34 ~ one
9	Israeli Breast-Cancer in Scalp-Irradiation Study [32]	1.6	1.34	2.1 ~ 2

system of the mammalian cell is imperfect and that there is no harmless dose threshold.

This conclusion, drawn from the aggregate of scientific evidence, has complex ethical, economic, and political implications for continuing radioactive contamination of our soil, water, and atmosphere. The following two facts have to be faced:

1. There exists no confirmed or scientifically reliable method of ascertaining permanent isolation of radioactive wastes from the biosphere (measured in geological time spans).

2. Future statistically predictable reactor accidents, such as Chernobyl, will add worldwide somatic and genetic health detriment.

From this perspective, the authors deem continued application of nuclear technology for energy production, whether in the U.S. or as an export to developing nations, a violation of the fundamental spirit of the Universal Declaration of Human Rights.

How should the application of nuclear technology be managed and are there viable alternatives?

As radiologists and physicians in general become better informed about the lack of any risk-free threshold and about the magnitude of radiogenic risks, they will and should opt to minimize the use of radiation in diagnostic and therapeutic procedures. The saga of the early embrace of x-raying pregnant women and the ultimate warnings against such a procedure can serve as an object lesson [12,13]. It is essential, however, that physicians include better informed patients in meaningful risk-benefit assessments.

The argument is now being made by the nuclear energy industry and its government and corporate supporters that nuclear energy generation is the only solution to forestall global warming. Studies by respected scientists, however, have shown that energy conservation, using state-of-the-art improvements in the efficiency of energy-driven devices, combined with the development of community-based alternative energy technologies including solar power, wind, biomass, and fuel cells, can meet the energy needs of both developed and developing nations [46]. In addition, these technologies can also provide employment in both small and large enterprises, including jobs for those with advanced technical skills who presently work in the nuclear industries.

What is needed is a well-integrated policy that must include a reordering of national priorities. Such redirection of the enormous public and private resources presently

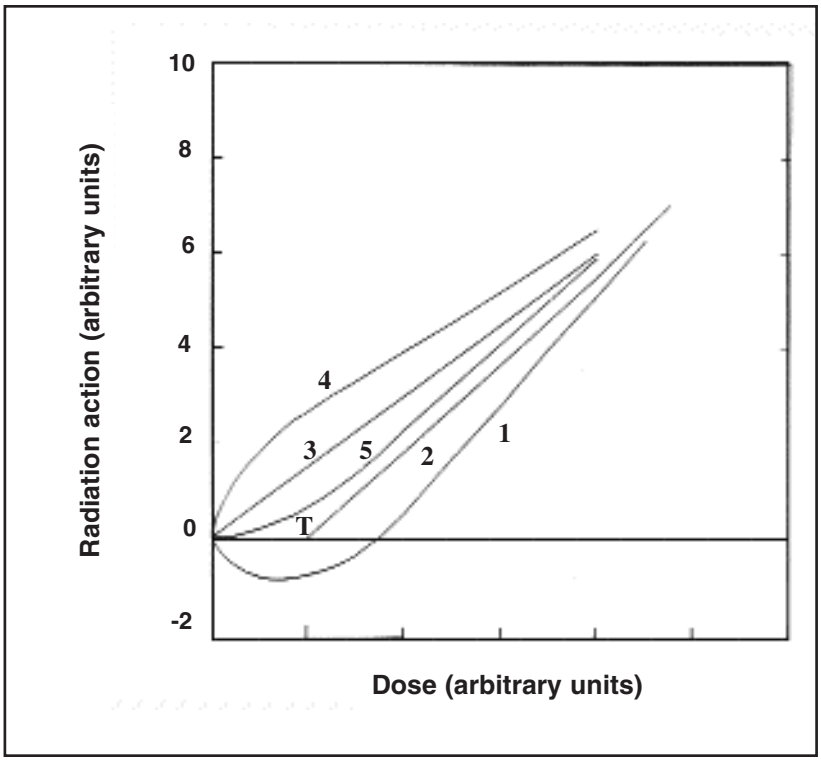

Figure 2: Various models for the shape of dose effect curves have been proposed, mainly to allow extrapolation from effects found at higher doses to effects in the low dose range relevant to occupational exposure and radiation protection. The model represented by the J-shaped curve (1) presumes that at low doses detrimental effects are even lower as compared to those occuring in the unexposed population. This so-called "hormetic" effect has, however, no scientifically credible foundation for stochastic effects such as radiogenic mutation and cancer induction. Model 2 (curve 2) assumes no detrimental effect up to a dose threshold T, followed by a linear increase at higher doses. No supporting data can be found for this model as long as stochastic effects of radiation are considered. Model 3 (curve 3), the most widely accepted, assumes a linear relationship between absorbed dose and detrimental effect without threshold. Numerous supporting data can be found in the relevant literature. In the linear-quadratic relation (curve 5) the assumption is made that a linear extrapolation from high to low doses would overestimate the detrimental effects and that dose rate effectiveness factors (DREF) between 2 and 5 have to be employed to describe the detrimental effects at the low dose region. In the BEIR V Report [2] the committee states: "There are scant human data that allow an estimate of the dose rate effectiveness factor" and "for most other cancers in the life span study (LSS) the quadratic contribution is nearly zero, and the estimated DREF's are near unity." The supralinear model (curve 4) describes the observation made by several investigations in the low dose range in model systems as well as epidemiological research. There is evidence that the supralinear curve correctly describes the excess cancer risk of the A-bomb survivors exposed to doses below 20 cGy [48].

invested in industries responsible for polluting the earth with chemicals and radiation can only be brought about by the effective commitment of informed citizens. ❧

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