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105-5

Independent Journal for
**Nuclear
Engineering
Energy Systems
and Radiation**

unabhängige Zeitschrift für
**Energiesysteme
und
Strahlentechnik**

KERNTECHNIK

Carl Hanser Verlag

Vol. 55 No. 4

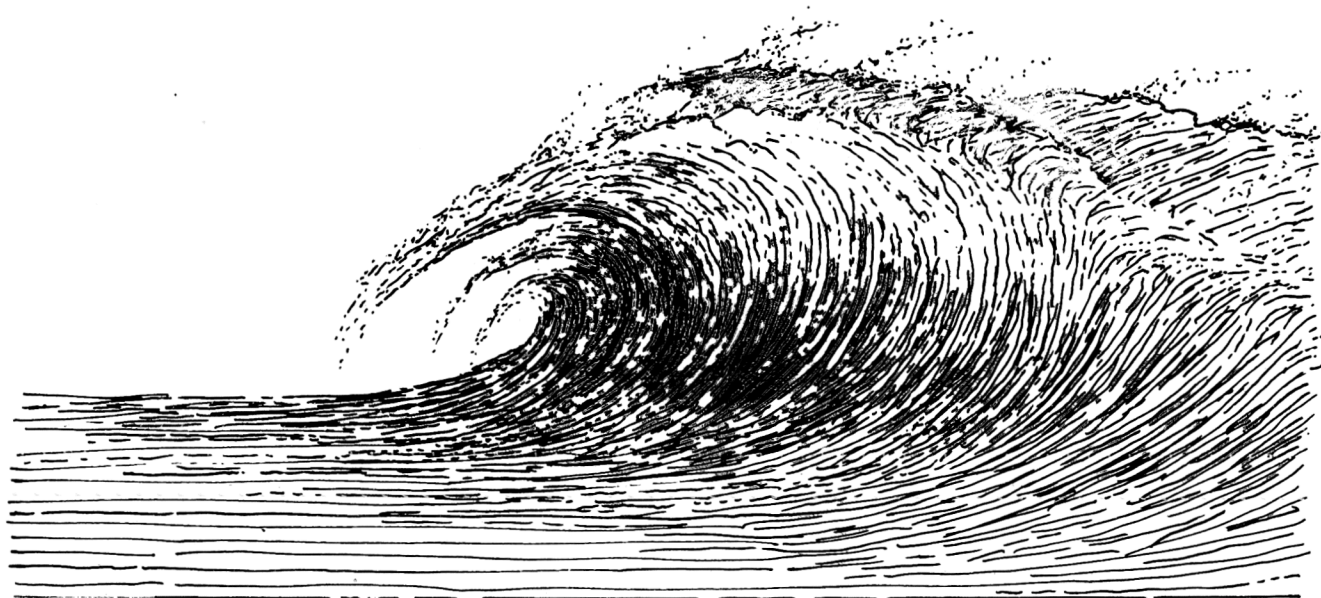
August 1990

Main subject:
Assessment of biological
radiation effects

Schwerpunktthema:
Biologische Strahlen-
wirkungen und ihre
Bewertung



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A. M. Kellerer

The new estimates of radiation risks

Hereditary damage and cancer are stochastic effects that can be due to individual lesions of DNA in individual cells, and that can therefore be caused even by small doses of ionizing radiation or of any other genotoxic factor. Where risks cannot be fully eliminated, they must be controlled and reduced to acceptable levels. This requires numerical estimates, which need to be derived from observations at doses that are high enough to let the induced rates of damage emerge from the statistical background of normal rates. The observations on the atomic bomb survivors are the most important basis for risk estimates. Earlier estimates were based on a tentative dosimetry system and on epidemiological observations that extended up to 1975. The dosimetry system has now been revised, and new analyses have been performed on the basis of the continued follow-up. The new estimates of the risk coefficients are substantially larger than earlier estimates. The magnitude of the changes, the underlying reasons, and some implications for radiation protection regulations are discussed.

Ergebnisse der Neubewertung von Strahlenrisiken. *Erbschäden und Kanzerogenese sind stochastische Wirkungen, die auf einzelne DNA-Schäden in einzelnen Zellen zurückgehen und daher selbst durch kleine Dosen ionisierender Strahlen oder anderer genotoxischer Faktoren hervorgerufen werden können. Wo Risiken nicht völlig auszuschalten sind, müssen sie kontrolliert und hinreichend verringert werden. Dazu benötigt man numerische Schätzungen, die man jedoch nur durch Extrapolation von Beobachtungen bei hohen Dosen erhalten kann. Die epidemiologische Untersuchung der Atombombenüberlebenden wurde zur Grundlage der Risikoschätzungen für ionisierende Strahlen. Frühere Schätzungen beruhten auf vorläufiger Dosimetrie und auf Beobachtungen bis 1975. Das Dosimetriesystem wurde nunmehr revidiert, und neue Berechnungen stützen sich auf die weitergeführten Beobachtungen. Die neuen, wesentlich erhöhten Risikoschätzungen, die Gründe für die Erhöhungen und Folgerungen für den Strahlenschutz werden erörtert.*

1 Introduction

The dose makes the poison. This statement of *Paracelsus* applies to substances that play a useful role in the metabolism of cells but can cause cell depletion and tissue damage when they are present in excessive levels. The threshold-like action is so familiar that the concepts of risk and threshold seem inevitably associated.

The familiar association fails for ionizing radiations. These can indeed cause cell death, and any resulting tissue damage occurs at high doses only. But such *non-stochastic* effects can be readily avoided, and the main issue of radiation protection is therefore the *stochastic* effects that result from cytogenetic damage. DNA lesions in individual germ cells can lead to hereditary damage in the descendants of an exposed individual. DNA lesions in somatic cells can cause cancer that arises years or even decades after exposure. A single charged particle can produce a DNA lesion, and stochastic effects can thus – although with correspondingly small probability – be induced even by small radiation doses; hence there need not be a threshold.

Due to exposure to natural radiation alone, millions of cells of the human body are traversed each second by energetic electrons. This does not make ionizing radiation a major cause of cancer – such as smoking or the UV light of the sun – but it is likely to contribute somewhat to the spontaneous cancer frequency, and any added exposures will contribute accordingly. Analogous considerations apply to hereditary damage; in this case there is strong evidence that any contribution of exposure to natural radiation is far below that of other factors, but it is also clear that one deals with a unicellular process that cannot have a threshold.

The absence of a threshold for cellular effects is not the characteristic of ionizing radiations only; it must be seen as a general feature of genotoxic agents. For chemical carcinogens, too, one cannot give critical doses or concentrations that would separate the safe from the unsafe. The principle of protection is then the same as with ionizing radiations; the

risks must be reduced to acceptable levels. To be acceptable, a risk must usually be unobservable, and epidemiological data can therefore not be obtained at doses which are relevant in radiation protection. Instead, risk coefficients are obtained by extrapolations from high to low doses. Accordingly they are hypothetical, and where this tends to be overlooked, one ought to add the qualification *nominal* whenever one talks of risk coefficients.

Extrapolations lack scientific rigour, but they are required in the attempt to assess possible risks. The critical attitude towards the effects even of small doses of ionizing radiations requires risk quantifications, and analysis of epidemiological data is therefore essential in spite of all its limitations. It will be considered here with the main emphasis on the new results from the follow-up of the atomic bomb survivors.

2 The results from Hiroshima and Nagasaki

Nearly a century ago, when *W. C. Röntgen* found his new kind of rays, he provoked world-wide fascination and high expectations that were kept alive for half a century and were not diminished when severe damage was caused by careless use of X-rays and, later, of radionuclides. Skin damage and then skin cancer, and the first cluster of leukaemias among radiologists, were seen merely as the result of continued and readily avoidable overexposure. Even the first great tragedy resulting from unprotected handling of radionuclides did not lead to a changed attitude; hundreds of young women, mostly in the United States, marked dials with luminescent paint containing the long-lived radium-226. Being paid by the piece, they sharpened their brushes in the quickest way they could by using their lips; in this way they incorporated excessive activities that killed many of them, years and tens of years later, mainly by bone tumors [1, 2]. Even after these dreadful experiences the belief continued that the health effects were merely the result of excessive doses.

Those who used the atomic bombs against Hiroshima and Nagasaki expected few acute radiation effects in addition to

the damage done by blast and heat. There was no expectation at all of late effects.

In the first few years after the war inquiries into the medical effects of the atomic bomb radiation were prohibited. When the first excess of leukaemia cases was recognized in Hiroshima and Nagasaki at the beginning of the fifties, the realization set in that leukaemias could be the result of somatic mutations of individual bone-marrow stem cells and that they might therefore be caused, although with small probability, by small doses [3]. This was the beginning of the recognition that, with ionizing radiations, one cannot separate safe doses from dangerous doses. There are some risks even at small doses, and protection must not be aimed at total avoidance of radiation effects but at a reasonable reduction of risks.

The enhanced leukaemia rates suggested that solid tumors, too, might be induced by the atomic bomb radiation. However, it took many years until this suspicion was confirmed and answered in quantitative terms. The reason for the delay was the entirely different temporal distribution of the excess cases of leukaemia on the one hand, and of solid tumors on the other hand.

3 The model of absolute and relative risk

The epidemiological follow-up of the atomic bomb survivors comprises about 75000 persons, the so-called *life-span study sample* (LSS). Initially rough dose estimates were used; from 1965 a tentative dosimetry system (TD 65) was employed that had been developed at the Oak Ridge National Laboratory [4]. According to this dosimetry, fast neutrons played an important role in Hiroshima next to the γ -rays, while the contribution of neutrons was insignificant in Nagasaki. The difference of radiation quality resulted from the fact that the plutonium bomb of Nagasaki was embedded in tons of conventional explosive, i.e., in low atomic-number material that shielded the fission neutrons effectively. The uranium bomb of Hiroshima, however, was covered merely by a steel tube that produced little neutron shielding.

In the initial years of the follow-up mortality rates due to leukaemia and other cancers were determined. Subdividing the LSS into a number of dose classes, the correlation of cancer mortality with dose was determined. A positive correlation was seen immediately for leukaemias. It was confirmed much later for solid tumors. The results were first expressed in excess mortality rates per person per year per gray.

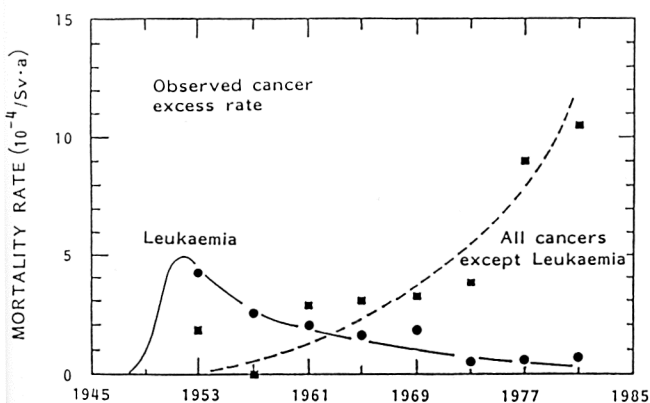


Fig. 1. Excess cancer mortality of the atomic bomb survivors. Each data point represents an observation period of 4 years. The interpolated curves are given merely for easier visualization. The data contain a temporal shift of the age distribution of the collective and can therefore not provide risk estimates directly. Diagram adapted from [5]

The result of this comparatively crude analysis is represented in Fig. 1. In this form it cannot provide numerical risk coefficients, because the observations are not stratified for age, sex or dose. One recognizes, however, the strictly different temporal distribution of radiation induced leukaemias and of the solid tumors occurring later. The essential observation is that the excess leukaemia cases began to arise only a few years after radiation exposure – and probably even before the epidemiological follow-up had been initiated. Statistical analysis demonstrated that in the early fifties the excess rate of leukaemias in the LSS was larger than the spontaneous rate. In the subsequent years the leukaemia rates decreased, and while an excess is still present it is now much less significant.

The pattern of occurrence was entirely different for the solid tumors. Some ten years after the bombing the first uncertain excess rates were recognized, several years later they became significant. Today a far more quantitative picture has emerged. Excess rates can be seen that grow in parallel to the age-specific spontaneous cancer rates. Although the excess is by now well established, it contributes – in contrast to the situation with leukaemias – only a minor part of about 5% to the total cancer mortality in the LSS.

More quantitative results were obtained by a detailed analysis that accounts not only for the dose and the time after the bombing but also for age and sex. An essential conclusion is brought out in Fig. 2. The lower panel shows the age-specific spontaneous rate of leukaemia mortality. The waves that are super-imposed on this curve represent the enhanced leukaemia rates that result, according to the new risk estimates, from an exposure with the specified doses at an age of about 45 years.

The upper panel gives the markedly different dependences for overall cancer mortality without leukaemias. The relative enhancements of the spontaneous rates are smaller, and the enhancements begin later. However – and this is an essential result from 40 years of follow-up in Hiroshima and Nagasaki – they appear to persist into old age. Since the relative increases remain constant when the exposed person ages, one speaks of a *relative risk model*.

The enhancement factors depend on dose. They depend also on age at exposure, and there is a general trend of larger enhancement factors for exposure at young age. Those who were exposed as children have not, as yet, reached the age of substantial spontaneous cancer rates. Accordingly, the statistical results are still uncertain for them. It is equally uncertain whether the initially observed enhancement factors will persist with increasing age.

4 Estimation of risk coefficients

If only those atomic bomb survivors were investigated who received a dose of less than 0.5 Gy – and most of the members of the LSS received doses which are much smaller – one would recognize dose related increases of the tumor rates that are only marginally significant. The results of the epidemiological follow-up are therefore essentially derived from the few thousand persons who received higher doses. There are, accordingly, no reliable statements about the effects of small doses. However, the principles of radiation protection have long been based on the cautious assumption that there is no threshold for stochastic effects, and it was therefore evident that attempts should be made to estimate risk coefficients applicable to small doses. The International Commission on Radiological Protection (ICRP) and other international bodies made such attempts even in the seventies [6–8].

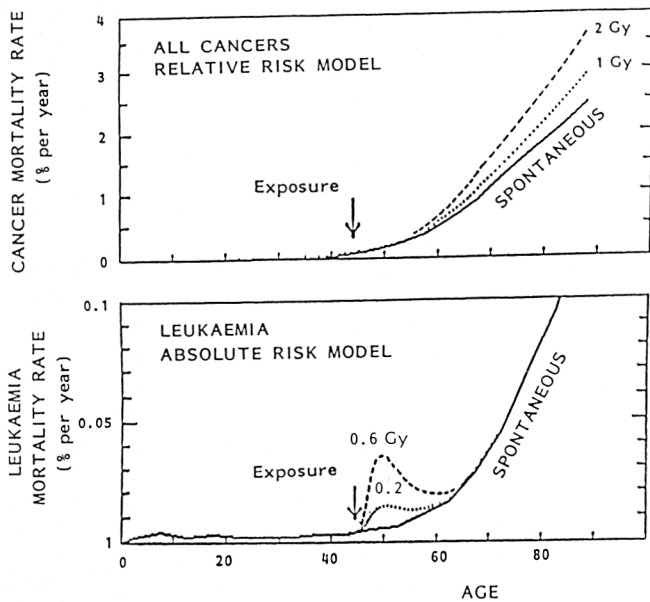


Fig. 2. Diagram of cancer mortality according to the relative and the absolute risk model. The solid curves give the average rates for the male population of the USA. The dotted and the dashed lines indicate the excess rates due to radiation exposure and correspond to the new risk estimates [16, 20], with an assumed reduction factor of 2 for the extrapolation to low doses

The earlier analyses utilized the dosimetry system TD 65. One obtained non-linear, almost threshold-like dose dependences for Nagasaki while the dose relations for Hiroshima were almost linear. This was taken as expression of the fact that γ -rays dominated in Nagasaki, and that this sparsely ionizing radiation is comparatively inefficient at small doses, while the effects in Hiroshima were largely determined by the densely ionizing neutron radiation which is equally effective per unit dose at all dose levels [9]. This characteristic difference, well known from radiobiological studies, was seen as justification for applying a substantial reduction factor of 2.5 when risk coefficients for high doses were extrapolated to small doses of γ -rays [6-8].

The risk estimate for the overall cancer mortality in an adult, i.e., working-age, population was given as 0.013 Sv^{-1} by ICRP [8]. Although the unit sievert is used here, it must be noted that the estimate referred to smaller doses or to high doses delivered with low dose rate over a period of months or years. Without entering into the complex problem of comparability of somatic and genetic effects, one may note that the risk estimate for hereditary damage was, and will continue to be, substantially smaller than that for cancer mortality.

Soon after publication of the earlier risk estimates by ICRP [8] the first doubts arose concerning the validity of the TD 65 dosimetry system. New radiobiological findings indicated unexpectedly high values of the relative biological effectiveness of neutrons at small doses [10, 11]. This prompted suggestions that the quality factors for densely ionizing radiations needed to be increased [12], and led to a re-evaluation of the influence of neutrons in Hiroshima. New transport calculations demonstrated that the calculations for the TD 65 had employed a neutron spectrum that was too hard, had disregarded neutron attenuation by atmospheric humidity and had, as a whole, overestimated the neutron doses in Hiroshima.

The revision of atomic bomb dosimetry was performed in a cooperation of the U.S. and Japan. It took nearly 10 years and was completed in 1986 [13].

5 New results after the dose revision

The new dosimetry system (DS 86) confirmed the assumption that the neutron doses in Hiroshima had been overestimated. The computations give by now a very small contribution of neutrons, even in Hiroshima.

The major part of the radiation effects that had previously been attributed to neutrons was now seen to be caused by γ -rays. This suggested that the risk estimates for γ -rays had to be substantially raised. The situation was, however, more complicated. DS 86 showed that there were smaller neutron doses, but it also showed that there were larger γ -doses than previously assumed. According to the revised dosimetry, more effects are now attributed to γ -rays, but there are higher γ -doses.

Even in the dosimetry of γ -rays there were complicated and partly compensating changes. In particular, one found substantially more shielding by houses, but the human body turned out to be more transparent, so that deeper-lying organs were less shielded by body tissue than had previously been assumed.

The dose revision led to a *de novo* analysis of the entire cancer mortality follow-up from Hiroshima and Nagasaki. The analysis was performed with improved statistical methods, its results are documented in a series of reports of the Radiation Effects Research Foundation (RERF) in Hiroshima [14-16]. Omitting the complexities of dosimetry and of numerical procedures, the essential results are given in the following.

For cancers other than leukaemia a relative risk model was used (see Fig. 2). The computations were based on the postulate that the tumor incidences and, therefore, also the mortality rates remain unaffected for a latent period of about 10 years after exposure and that they are then enhanced over the spontaneous age dependent rates by a constant factor that remains unchanged throughout further life. The factor of proportional enhancement depends on dose and on age at exposure. That the enhancement factors will continue to be constant, is still a hypothesis. It is, however, well borne out for the 40 years of observation that are already covered by the follow-up.

The essential result is indicated by the upper curve in Fig. 3. This curve shows the enhancement factors, i.e., the relative risks for cancer mortality, for six dose groups. The data represent all age groups and both sexes and are therefore accompanied by a table of scaling factors. It can be noted that the values for females exceed those for males by more than a factor of two. This does not imply that, at equal doses, women are subject to more excess cancer cases per year than men. For women the age specific cancer rates are, at least at higher age, only about half the rates for men. Double the relative excess risk leads therefore to about the same incidence rate of excess cases. In total, however, a given exposure at specified age will still induce a larger number of cancers in women, but this is merely a matter of their greater longevity that exposes them longer than men to the risk of cancer.

At doses of more than 4 Gy the computed relative risks are potentially misleading. At these doses few survived, and a bias must have arisen because the survivors were predominantly those whose doses were in fact smaller than the estimated values. Some uncertainty of this type may still persist in the range of 2 to 3 Gy. Therefore the initial part of the computed curve is most meaningful for risk estimation and it indicates an increase of cancer mortality by about 50% after a dose of 1 Gy of γ -rays.

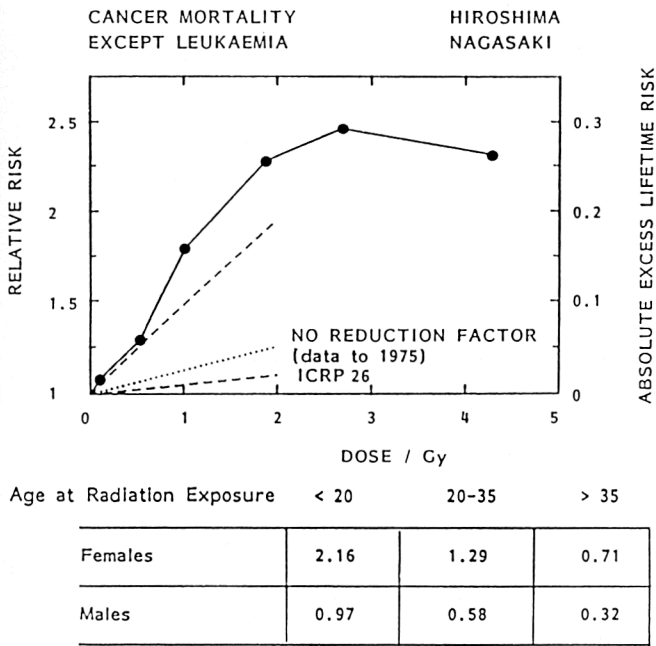


Fig. 3. Dose dependence of the relative and the absolute risk of cancer mortality for a population of all ages according to the new results of RERF [14]. The upper dashed line represents the excess lifetime risk. For comparison, the previous risk estimates of ICRP [8] with and without reduction factor are indicated. The ICRP estimates were based on data up to 1975 and relate to an adult working-age population. The results of the new analyses refer to a population of all ages. The table contains the adjustment factors for different age groups

Recent results from RERF employ somewhat more sophisticated computations of organ doses and they suggest somewhat less excess risk. The differences, however, are small compared to the statistical uncertainties of the results, which may be in the range of $\pm 30\%$ [17]. It is interesting to translate the relative risk into absolute risk. Based on population statistics in Japan, Europe, and the United States, one may estimate that cancer causes at present about 20% of all deaths; the doubling dose of 2 Gy for cancer mortality corresponds then to a risk coefficient for cancer mortality of 0.1 Gy^{-1} .

Among the late evacuees from the immediate vicinity of Chernobyl there were about 30000 persons who may have received an estimated whole-body dose of 0.3 Gy [18]. If the above risk coefficient was applied to this group of persons and used conservatively, i. e., without any reduction factor for lower doses, one would infer an increase of about 15% beyond the roughly 6000 cancer deaths that would normally be expected in this population. This figure may, however, be a substantial overestimate. International scientific bodies tend to postulate a reduction factor that would reduce the estimate at least to half its value [19, 20]. Since any extrapolation is tentative, one can merely state that future cancer rates may exceed normal rates by 5 to 15% in this important collective of persons who were most highly exposed in the vicinity of the reactor. Even changes of 15% in cancer rates are quite difficult to ascertain in an epidemiological study, and it is therefore of evident importance that a very careful investigation be carried out in the Soviet Union. Intentions for such a study have been stated, but as yet there has been little evidence of progress. In the absence of serious studies it is not surprising that reports of a multitude of radiation effects abound that are unsupported by facts.

A heated discussion was provoked when the recent reports of RERF demonstrated substantial increases of the risk

coefficients. The lower straight line in Fig. 3 represents the risk coefficient that had previously been proposed for small doses and that corresponded to an absolute risk of 0.013 Gy^{-1} . One notes the striking difference of a factor of nearly 10 compared to the new results.

The difference is, however, reduced to a factor of 4 when one compares the results to the earlier estimates without the reduction factor of 2.5 that was used for the extrapolation of the earlier observations to small doses. Before the dose revision the reduction factor was suggested by the epidemiological results themselves; it accounted for the apparent difference between the dose dependences in Nagasaki and Hiroshima. In the new analysis, the significant difference of dose dependences in the two cities and the significant deviations from linearity at small and moderate doses have been lost. A reduction factor for the extrapolation to small doses is thus less readily justified. It is still postulated [19, 20], because it is indicated in most animal studies.

How can the remaining factor of 4 between the old and the new estimates be explained? Surprisingly it does not result from the dose revision; it reflects merely the continuation of the epidemiological follow-up from 1975 up to now, and from now into the future. A factor of about 2 accounts for the excess cancer deaths that have occurred between 1975 and 1985. This surprisingly large number can be understood if one realizes that many atomic bomb survivors have now reached an age where age specific cancer rates increase steeply and where, according to the model of relative risk, one expects also the majority of the radiation induced cases.

The remaining factor of 2 is largely hypothetical. It represents an extrapolation into the future lifetime of all surviving members of the LSS. The extrapolation utilizes the postulate of persisting enhancement ratios; but this postulate is entirely uncertain for those who were exposed at young age. For this group one begins to see increasing cancer rates that are still subject to considerable statistical uncertainties but that indicate large enhancement factors. If the large enhancement factors were confirmed statistically, and if they persisted into old age, they would contribute an added number of excess cancer deaths sufficient to double the total number that has been observed up to now. The fate of those who have survived the atomic bombs in adolescence or in childhood will therefore be the essential topic of the epidemiological study which will reach into the next century.

For the leukaemias, too, new results have been obtained. The situation is simpler because few added excess cases have been seen since 1975. Nevertheless, increased risk estimates are obtained even for leukaemias (cf. Fig. 4). Comparing the new with the earlier results, a factor of about 2 can be noted. This factor of increase is due to the dose revision. The bone marrow is a relatively superficial organ, and the higher transparency of the body that has resulted from the new dosimetry system is therefore less relevant for bone marrow than for deeper-lying organs. There is accordingly no compensation for the dose reduction that results in the DS 86 from the increased shielding by houses. The bone-marrow doses are smaller than they appeared to be in the previous dosimetry system; the risk estimates are correspondingly larger. If the enhancement factors for solid cancers persist into the future, leukaemia will contribute only about 10% of all radiation induced mortality. But one must note that leukaemias occur sooner after exposure than solid cancers and that they consequently cause more loss of life expectancy.

One can again consider the collective of 30000 persons who may have been exposed near Chernobyl to 0.3 Gy. Using the risk estimate for leukaemias of 0.005 Gy^{-1} and ap-

plying it to the exposed group, one would infer an excess of about 45 leukaemia cases. In spite of the smaller absolute numbers, leukaemias will be the critical end point that is most likely to be detected in an epidemiological follow-up of the exposed people. Focussing on childhood leukaemias might offer an even better chance to obtain improved risk coefficients, even if only few cases will be observed. But, as stated, a very thorough study would be required.

6 Conclusions

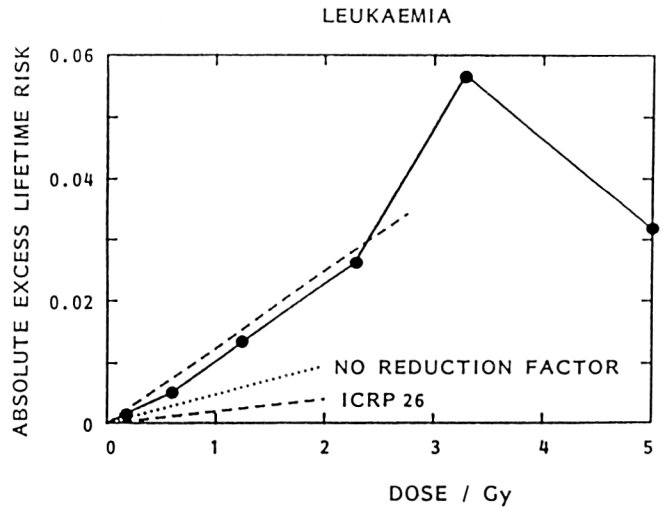
Different reasons have been given why the new analysis of cancer mortality in Hiroshima and Nagasaki leads to increased risk estimates. The main factor is that cancers are now included that occur more than 3 decades after exposure, and possibly much later. The revision of the dosimetry is important because it has led to nearly linear dependences for Hiroshima and Nagasaki. A reduction factor for the extrapolation from high doses to low doses can no longer be based on epidemiological data; it is merely supported by general radiobiological findings. In 1975 a reduction factor of 2.5 seemed conservative. Today a factor of 2 appears to be a realistic assumption, and a strictly conservative approach would employ no reduction factor.

There is special importance in the large relative enhancement factors for the tumor rates of those who were exposed as adolescents and children. If these enhancement factors should persist over the entire life – and this can only be seen in the continued follow-up – they would result in high absolute risk factors for radiation exposure in young age. The re-evaluation of radiation risk for radiation workers will not be affected by this unresolved question.

A recent report [20] puts the nominal risk coefficient for cancer mortality in an adult working population at 0.04 Sv^{-1} . To illustrate the magnitude of this risk estimate one may consider a dose of 0.2 Sv accumulated at 5 mSv per year, over a 40 year working life. This leads to a nominal cancer mortality probability of 0.008, i.e., to a 4% increase over the “normal” probability of 0.2. The numbers must be seen as rough illustrations: the risk estimate of 0.04 Sv^{-1} is uncertain, the dose 0.2 Sv is a crude estimate of an average that may be accumulated over a working life, and the figure for cancer mortality probability may by now be 0.25 rather than 0.2 in the developed countries. The total dose accumulated over life from natural radiation exposure, including radon in houses, is on average somewhat less than 0.2 Sv. But it needs to be given slightly more weight since it is partly delivered in childhood and adolescence where the risk factors appear to be highest.

For a perhaps more comprehensible illustration of the magnitude of possible risks, one may refer to the loss of years of life. If a cancer death causes on average a loss of 15 years of life – a value intermediate between those for leukaemia and solid cancers –, the dose equivalent of 0.2 Sv and the cancer death probability of 0.008 correspond to a mean loss of about 0.1 years of life. The number of about 8 years of life lost by a typical smoker is sometimes noted for comparison, but it must certainly not be used to play down possible risks of radiation. Smoking, as the most prominent cause of cancer, causes excessive risks that are far beyond anything that could today be accepted as an occupational hazard.

The numerical values considered here are subject to considerable uncertainties; in spite of the large group of people who have been followed, they are based on only limited numbers of cases. In total, the statistical analysis indicates about 70 excess leukaemia cases and about 350 other excess cancer deaths observed up to now in the LSS.



Age at Radlation Exposure	< 20	20-35	> 35
Females	0.57	0.63	0.88
Males	1.01	1.46	1.45

Fig. 4. Dose dependence of the absolute risk of leukaemia mortality according to the results of RERF [14]. The dashed line represents the mean lifetime risk. For comparison, the previous risk estimates of ICRP [8] with and without reduction factor are indicated. The ICRP estimates were based on data up to 1975 and relate to an adult working-age population. The results of the new analyses refer to a population of all ages. The table contains the adjustment factors for different age groups

The results of the Japanese study are largely confirmed by other epidemiological investigations that are each by itself less informative. Most of these investigations relate to persons that were exposed for medical reasons [19, 20].

The risk estimates for hereditary effects of radiation exposures are even less certain than those for cancer mortality. It is not generally recognized that such effects have never been demonstrated in man. Not even the very large study on the descendants of the bomb survivors have, up to now, shown significant increases of hereditary damage [21]. This implies in no way that such damage does not occur or that it is less likely per unit dose than previously assumed. From animal studies one infers that the doubling dose for hereditary damage should be between 1 and 2 Gy. Since hereditary damage is due to individual DNA lesions in individual cells, one has less reason than for cancer to doubt the linearity of the dose-effect relation in the range of small doses.

Suggestions of large genetic risks have recently been raised by a reported association between 5 excess childhood leukaemia cases near the Sellafield reprocessing plant and the fathers' above-average occupational exposures with accumulated γ -ray doses of about 0.15 Gy and doses of about 15 mGy in the half year before the child's conception [22]. Similar, although somewhat weaker, associations were found in the same study between childhood leukaemia and suspected or unsuspected factors such as age of the mother or work in the iron and steel industry. All previously unexpected associations urge further studies, but it would be premature to interpret each association as a causal relation. A large study of the frequency of childhood tumors of the descendants of the atomic bomb survivors shows – even at far higher doses to the fathers – no evidence of any increases [23]. In view of this disparity, attention has been focussed on a shorter period, of assumed higher sensitivity, before conception; this

would imply a childhood leukaemia probability of about 0.0025 due to a dose of 15 mGy in the half year before conception. But even with this assumption there is clear disparity to the Japanese data. 250 children conceived within half a year after the atomic bomb explosions developed no childhood leukaemia although the average paternal dose was about 0.25 Gy; with the above estimate, about 10 leukaemia cases would have been expected. The lower dose rate, too, in the occupational exposure is an unlikely explanation; in animal experiments the hereditary effects are reduced when the γ -ray dose is fractionated or protracted. The statistical association found in the recent study is therefore of interest, but it appears not as a causal connection. The results from similar current studies may shed further light on the problem.

The ICRP has stated in past recommendations that radiation workers, either in nuclear industry or in medicine, are subject to risks similar to those in other "safe occupations". This statement resulted from a comparison between the *nominal* cancer mortality risk in radiation workers and the *observed* accident mortality in other occupations. Since accident rates have markedly decreased in recent years in most professions, and since the risk estimates for radiation exposure have now been increased, the numerical equality has ceased to apply. Whether this should force a reduction of dose limits for radiation workers is a socio-political rather than a scientific issue. But, irrespective of an impending decision that needs to be made jointly for the member states of the European Community, added caution is indicated. Even less than in the past, it would be acceptable to expose radiation workers routinely to doses that are close to the annual limits. Such exposures would be far above the average of current practice and they need to be avoided even for small groups of workers. The revised radiation protection regulations in the Federal Republic of Germany take these matters into account by adding to the earlier annual dose limit of 50 mSv the further constraint of a lifetime limit of 400 mSv.

In the United Kingdom and in Sweden largely equivalent regulations were adopted; one requires that radiation workers must – on average over their working life – not be subjected to an annual dose of more than 15 mSv. New recommendations of ICRP are to appear soon; they are likely to contain similar rules.

It is occasionally said that numerical risk estimates cannot be justified in view of the existing uncertainties, and that they cause fear even of the most minimal doses. Unreasoned fear, however, can only be countered by reason, and reason needs to be guided by observed facts and their cautious extrapolation into the uncertain. The great interest in the follow-up of the atomic bomb survivors will therefore continue.

Risk estimates and dose limits are important but not central to radiation protection. The overriding consideration is the principle to keep exposures *as low as reasonably achievable*, even in those cases where the limits are fully met. This principle of ALARA may appear as an overconservative approach, particular to radiation protection. In fact it may be the only adequate response to a situation where choices need to be made between risks that cannot be entirely eliminated. A more balanced perception of risks will be reached when the principle is extended to all genotoxic agents.

(Received on May 15, 1990; in English version on May 28, 1990)

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