Chapter for ***Nupecc***

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Invisible Threat
– The Risk of Ionizing Radiation –

Introduction

The destruction of Hiroshima and Nagasaki by atomic bombs, the nuclear arms race during the Cold War, and the reactor accident of Chernobyl have created deep concern with regard to radioactivity and ionising radiation. In the public perception, radiation has thus become one of the major risk factors, but this perception is often abstract and frequently not backed up by an informed judgement of the actual nature and magnitude of the risk of radiation and, in particular, the risk of low dose radiation exposures. The following synopsis is intended to outline basic facts and numbers that can put radiation and radiation risks into a more meaningful perspective and can thus facilitate a realistic judgement of the merits and the dangers of nuclear technology and of the various applications of radioactivity and ionising radiation.

The great reversal

When in 1895 W. C. Roentgen announced a mysterious new radiation that could penetrate all kinds of matter, the world took immediate and enthusiastic notice. In the spirit of the 19th century, the finding was taken to be the promise of unlimited technical progress and the apotheosis of classical physics. Both beliefs were universal, and both were wrong, as it should turn out later. In the same vane and long before its nature was understood, radiation and, subsequently radioactivity were considered not only a miracle of science, but also a medical panacea, a universally beneficial agent.

For half a century radioactivity continued to be seen as beneficial. Mineral water had to contain radioactivity, even toothpaste contained it. Radium-226 pillows were sold without prescription as a good-for-all agent to those who could afford it.

Health effects of ionizing radiation, specifically skin damage, had been observed early, when x-ray tubes were widely used without precaution. But such effects were explained as the results of gross negligence and continued high doses. For decades, radiation protection was not an issue. There was one notable exception: In New York a few days after Roentgen´s an-

nouncement Thomas Alva Edison pushed his mechanics to produce x-ray tubes, keeping them awake through day and night shifts by a hand organ set up in the workshop. Yet he was the one person to react and abandon at once all work with radiation when one of his assistants developed serious burns after exposure to the x-rays (1).

Most of the health effects that were observed in this first period were, indeed, the result of high exposures, but even before the nature of x-rays was recognized in the diffraction studies of Friedrich and Laue in 1912, a cluster of leukemia was noted among Berlin radiologists, perhaps the first observation of the effect of low dose rate continued exposures (2). But this did not cause particular alarm.

Larger tragedies were bound to happen. The first one resulted from the industrial use of radium-226 in the production of luminescent dials for watches and air plane control panels. Hundreds of young women in the US, in the UK, and in other countries applied the radium containing paint with fine brushes. Being paid by the piece, they tipped the brushes in the fastest way, i.e. with their lips. Thus they incorporated large bone-seeking α-ray and γ-ray activities. Many of these young women subsequently died from cancer, especially bone cancer (3). The tragedy was noted, but was not taken as a warning and certainly not as a warning with regard to possible late effects of small doses. Indeed, the doses to the dial painters had been large.

Other tragedies followed and they were mostly due to the use or misuse of radioactivity in medicine. One of the major misapplications was the production and use of *thorotrast* a contrast medium to be injected into blood vessels for x-ray imaging. As the name indicated, thorotrast contained the α-emitter thorium-232. Being a superb contrast medium it doubtlessly saved a great number of lives, especially during the last World War. But the price was paid by the long term survivors who experienced grave health effects. Some 50 years after the applications almost none of the surviving patients escape the late effects, the most serious threat being liver cancers with very high lethality (4).

The naive belief in the healing power of radioactivity persisted in spite of the negative experiences. In Germany after the 2^{nd} World War, when no treatment against tuberculosis was available, patients – many of them children – were given injections of high activities of Ra-224 in the erroneous expectation that this could inactivate the TB-bacilli. The same treatment was administered to patients who suffered from ankylosing spondylitis, a chronic inflammatory process of the skeleton. The Ra-224 did, indeed, reach the bone surfaces, but it damaged them rather than healing the illness. Many of the children suffered growth disturbances, and

patients in the entire group incurred other damage, such as opacification of the lens of the eye (5). The most severe consequence were more than 50 deaths from bone cancer among 900 patients. But other cancers were also increased, and even today the women who were treated as children show an increased rate of breast cancer.

While the treatment was totally inappropriate for bone tuberculosis, it would have made sense for ankylosing spondylitis, had it not been for the very high doses that were used. A later form of the therapy with much smaller doses is still found to be effective for a symptomatic, anti-inflammatory treatment.

These and other experiences demonstrated health effects of high radiation doses, but they did not indicate, nor were taken as indication, that small doses of radiation or small dose rates could be detrimental. It was, instead, believed that there was a threshold of dose below which there could be no undue health effects. In fact, small doses of radiation were still seen to be generally beneficial and stimulating to health.

The great reversal happened half a century after the discovery of x-rays and of radioactivity when the atomic bombs destroyed Hiroshima and Nagasaki. About 200 000 persons were killed by the heat flash and the blast of the bomb in the two cities, and thousands among those who survived the immediate effects suffered from severe radiation sickness. The horror of the nuclear explosions reversed the perception of radiation and radioactivity and turned the symbol of progress and life into the image of hell.

While research on radiation induced delayed health effects was interdicted by the US military administration in the first years after the atomic bombings, Japanese physicians nevertheless noted an increased incidence of leukemia among the inhabitants of Hiroshima and Nagasaki, a few cases per year, and this was the first tangible evidence of late radiation effects. The observation, still poorly quantified, changed the perception of the risks of ionizing radiation. It was then realized that radiation may not just cause hereditary damage through mutations of germ cells, but can equally cause leukemias through mutation of somatic cells. The critical point was that – at least conceptually – the mutation of a single cell could lead to a leukemia, which suggested that there is no threshold. Even very small doses could – with correspondingly low probability – cause a leukemia.

This recognition changed the philosophy of radiation protection. Earlier it was assumed that detrimental effects could be completely avoided by limiting radiation exposures to a safe level. Now it was realized that all radiation exposures are likely to cause a certain risk and

that consequently the aim and ambition of radiation protection could merely be to reduce exposures to a level *A*s *L*ow *A*s *R*easonably *A*chievable (ALARA principle) (6).

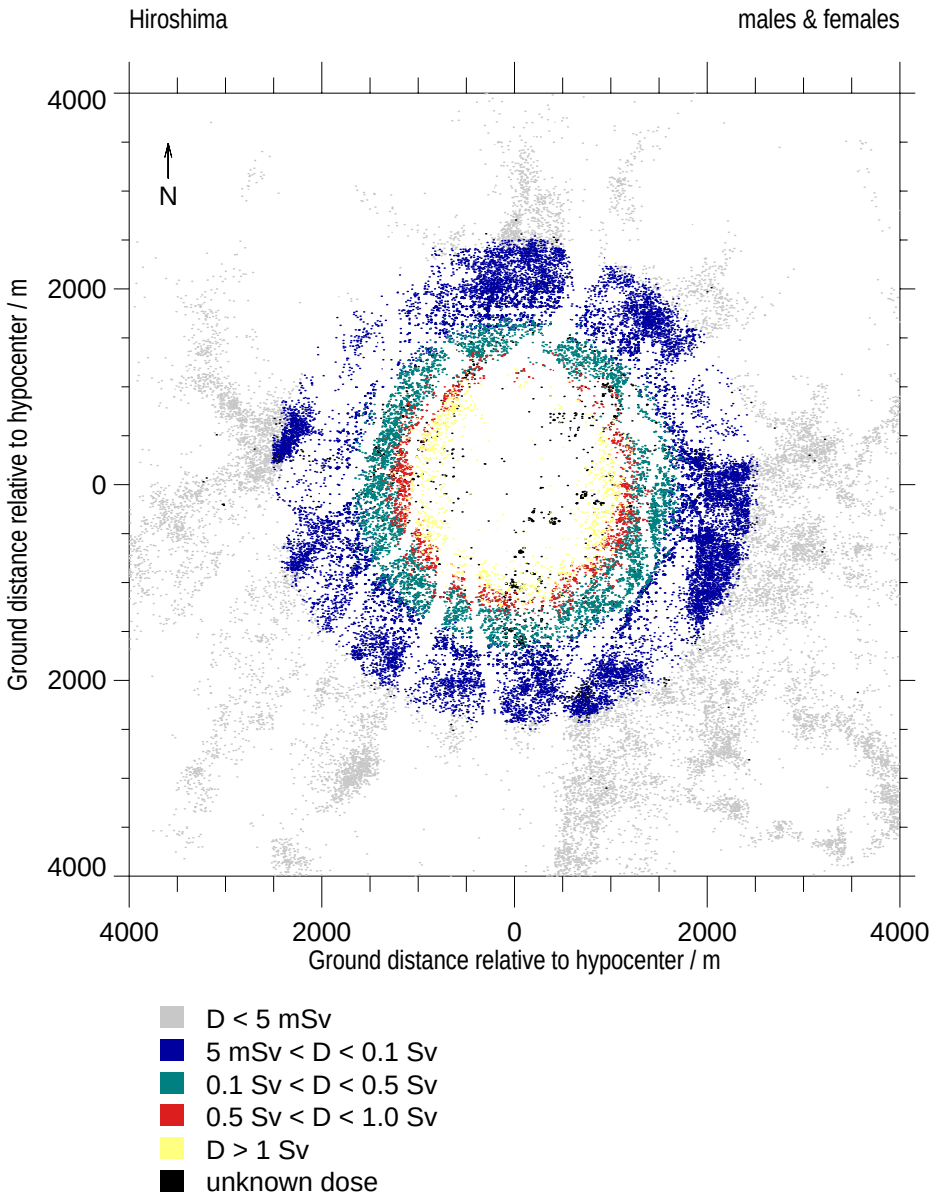

Fig.1: Point diagram of the A-bomb survivors in Hiroshima, color coded for dose. Each point represents the location at the time of the explosion of a member of the LSS-cohort. The pattern of points reflects the topography of the city with the river arms that lead to the harbour in the south. Very few survived within one kilometer from the hypocenter.

While this is a prudent and by now widely accepted position, it has nevertheless created controversy and has invited misinterpretations that produced – in association with the experience of the atomic bombs and the ensuing nuclear arms race – fear and apprehension. The highly biased perception of radiation risks is perhaps most poignantly expressed in the true but greatly misunderstood statement that even a single charged particle might create a cancer. The

force of this statement, but also its fallacy, lies in the fact that it evokes an image without quantifying its probability. A consideration of essential findings in decades of follow-up of the health status and the cancer mortality and incidence of the A-bomb survivors can provide the required numbers.

The cancer rates among the A-bomb survivors

When the increased frequency of leukemia among the A-bomb survivors was noted, it confirmed the need for extensive health studies, and even then the question began to be asked whether there might be a similar increase of solid cancers. In 1950 a large study was, therefore, initiated that included 120 321 survivors from Hiroshima and Nagasaki, the *L*ife *S*pan *S*tudy (LSS) cohort, whose causes of death were to be followed. In addition tumor registries were established in Hiroshima and Nagasaki, so that the cancer mortality data are supplemented by incidence data. While the detailed results of the study have been described in a number of highly informative reports (7,8), essential results will here be outlined.

For leukemia the increased incidence was seen clearly from the beginning of the study. Figure 2 gives the numbers of cases per year in the LSS cohort. No molecular markers or clinical distinctions are known today that could identify a leukemia or a solid cancer as being radiation induced, rather than being "spontaneous". Even conceptually there is little basis for this distinction: cancer is a complex multi-factorial process and radiation appears to be just one co-factor that tends to increase the incidence rate. Any excess incidence that is due to the irradiation must, therefore, be determined by statistical comparisons of the subgroups of survivors exposed to different doses. Sophisticated analyses of this type have been performed and the resulting attribution is depicted in Fig.2 by the red area which represents the excess rate, i.e. the cases attributed to the radiation exposure, and by the gray area which represents the expected leukemia cases, i.e. the incidence rate that would have happened even in the unexposed population.

The largest excess leukemia rate is seen in the first years of the study, subsequently the excess rate declined, and in the most recent observation periods it has largely disappeared. The rise in the expected annual numbers reflects the increase of the spontaneous leukemia rate as the average age in the LSS cohort increases. Ultimately the annual numbers will, of course, decline as fewer members of the cohort survive, but with about half of the cohort still alive in

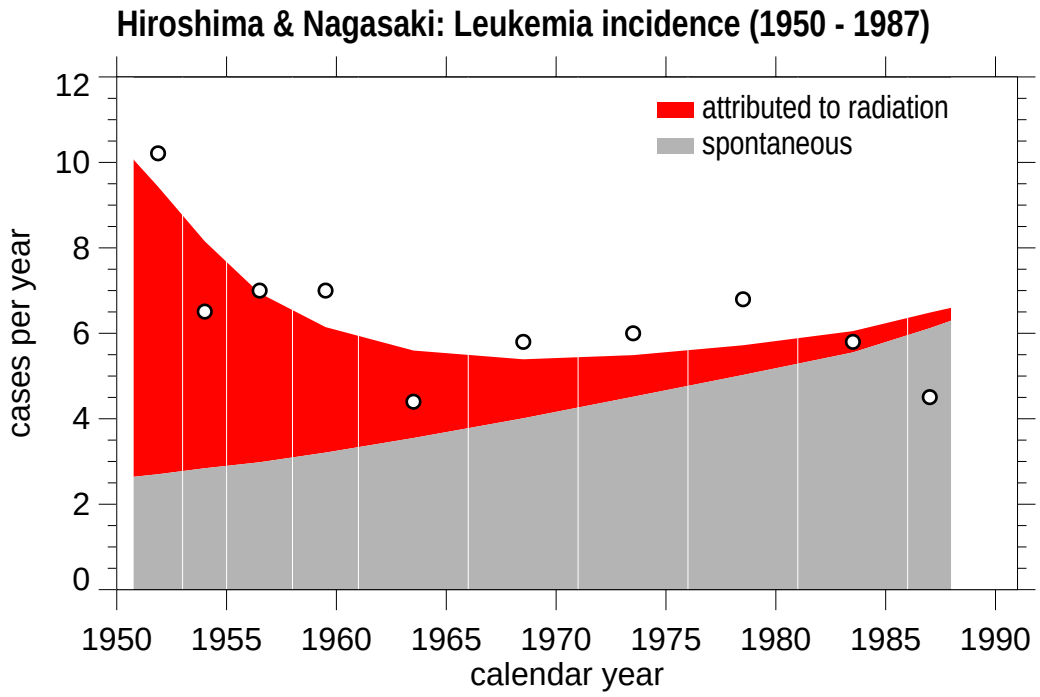

Fig.2: Annual number of leukemia cases (circles) among the A-bomb survivors in Hiroshima and Nagasaki in the different time periods. The gray area represents the annual numbers that would have occurred – according to the detailed statistical analysis – without the radiation exposure. The red area represents the excess incidence rate that has been caused, according to the computations, by the radiation exposure.

The increase of the spontaneous cases reflects the rise of the incidence rate with age of the A-bomb survivors. The excess rate was largest in the initial years; no leukemia registries existed before 1950, but it is known from other radioepidemiological studies that a certain excess rate may have been present already three to four years after the radiation exposure.

1990 this is not yet the dominant factor in the diagram. The essential point is that a considerable fraction (about one third for the period 1950 –1987) of the total leukemia incidence in the LSS cohort is attributed to the radiation exposures. This relatively high contribution corresponds to 75 out of a total of 231 leukemia cases and it is especially notable in view of the fact that only a minor part of the cohort received high doses, the average marrow dose in the entire LSS-cohort being only 0.14Sv (0.26Sv in the subgroup of survivors with more than 0.01Sv).

For quite a number of years after the beginning of the observations, no similar increase was seen in the solid cancer mortality of the A-bomb survivors. When finally ascertained it turned out to be percentage wise much smaller than the increase in the leukemia incidence. Figure 3 gives the results in analogy to Fig.2 and shows that the attribution to the irradiation is only about 4% over the entire period of observation. This corresponds to about 315 deaths out of a

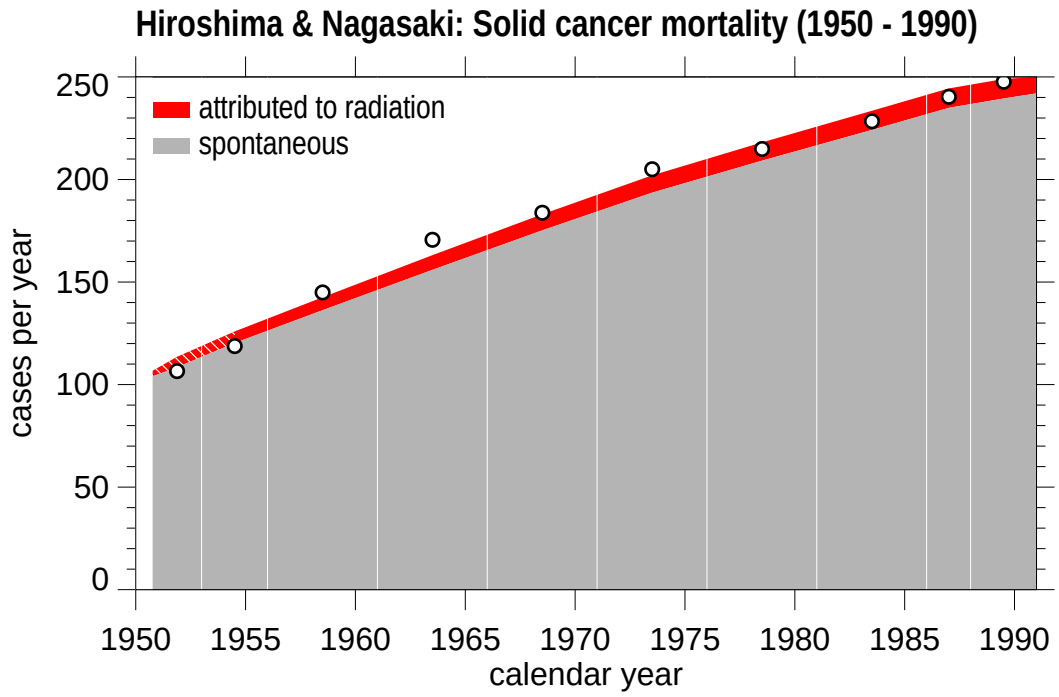

Fig. 3: The annual number of deaths from solid cancers (circles) in the LSS-cohort of the A-bomb survivors. The gray area represents the annual number of cases that would have occurred – according to the detailed statistical analysis – without the radiation exposure. The red band represents the excess that has been caused, according to the computations, by the radiation exposure.

The increase of the spontaneous cases reflects the rise of the cancer rate with age of the A-bomb survivors. Only a small fraction of the observed cases is attributed to the A-bomb radiation. This is so because the average dose in the LSS-cohort was only 0.14Sv, and the relative increase of the frequency of solid tumors is much smaller than the relative increase of the leukemia incidence after a radiation exposure.

total of 7 558 cancer deaths during the observation period. While the relative increase is much smaller than for leukemia, the absolute number of 315 excess deaths is more than four times larger than the number of excess leukemia cases (75). Since the excess rates persist for solid cancers, but not for leukemia, the ratio will increase further.

Attribution of "only" 75 leukemia cases and 315 deaths from solid cancer to radiation among the A-bomb survivors is at odds with the common perception – a perception shared also by the people of Hiroshima and Nagasaki – that most of the many thousands of cancer cases among the A-bomb survivors are due to radiation. While the result is an important qualification, it must not be taken as indication that the observations on the A-bomb survivors are uncertain. In the subgroup of 5 489 A-bomb survivors who were exposed to doses in excess of 0.5Sv the association with radiation exposure is, in fact, firmly established for the majority of cancer types. Figure 4 gives the excess relative risk, i.e. the ratio of the excess incidence to the spontaneous incidence, at 1Sv for various tumor sites.

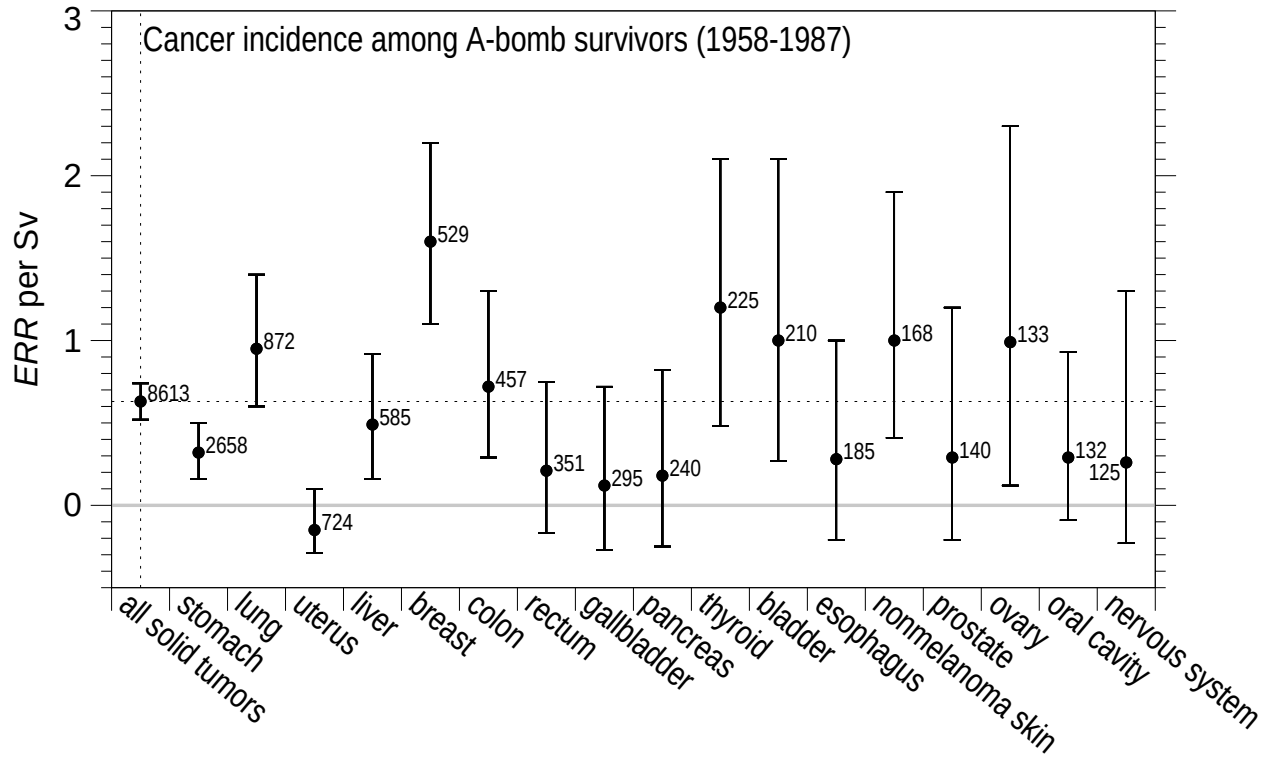

Fig.4: The excess relative risk, *ERR*, per Sv derived for different types of tumors from the observations on the A-bomb survivors. *ERR*=1 per Sv implies that the incidence rate of the tumor is doubled after an exposure to 1 Sv. In all but one tumor type, the tumor rate has been found to increase with dose. The estimates are given together with their 95 % confidence bands. The total numbers of cancer cases from 1958 to 1987 are noted next to the points.

Similarly, there are reliable conclusions on the age and time dependencies of the excess tumor rates. The diagrams in Fig.5 demonstrate some essentials. The upper panel refers to leukemia. The solid curve depicts the spontaneous incidence rate and its steep increase at older age. The broken curves represent the rates predicted – on the basis of the A-bomb data – for an exposure to 0.2Sv at age 5 or age 40 (see arrows). The excess is wavelike with only a few years latent period and with similar absolute excess for the two ages at exposure. But due to the much smaller spontaneous rates at young ages the excess is much more visible for young ages at exposure. This is why childhood leukemia is the first indicator of late radiation effects in an exposed population, a point that will be taken up in subsequent considerations of the expected and observed health effects due to the Chernobyl reactor accident.

The lower panel in Fig.5 relates to all solid cancer mortality combined. Reference is here made to a higher dose, since the relative excess at specified dose is less for solid cancers than for leukemia. The latent periods are longer than for leukemia, and the excess rates persist – unlike those for leukemia – into old age. The overall excess is somewhat larger for younger

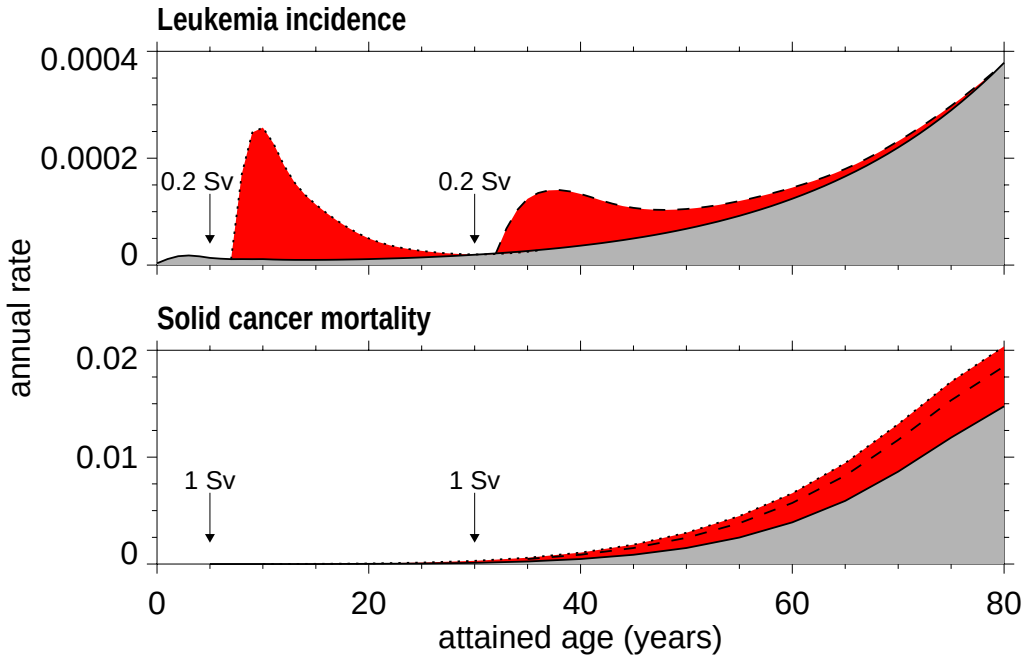

Fig.5: Age dependence of the leukemia and the solid cancer rate as derived from the A-bomb data. The gray areas represent the age dependent normal rates in an unexposed population (UK). The red areas represent the excess rates due to the specified radiation exposures at age 5 or age 30. For leukemia, *incidence* rates are given, because improved methods of therapy have reduced and continue to reduce substantially the leukemia mortality. The lower panel shows that the excess in the solid cancer mortality rate begins to be recognizable many years after the radiation exposure; the relative increase is somewhat larger for the earlier age at exposure.

ages at exposure, and the relative risks are generally larger at younger ages. The numbers are, of course, subject to some uncertainty, but the essential features and the general magnitude of the excess is reliably represented in the diagram.

Dependence on dose

Observed dose relations:

Most members of the LSS-cohort have received doses that were substantially below 0.2Sv. Their excess risk can not be determined with much precision, since the number of excess cancer or leukemia cases is very small in comparison to the spontaneous cases. Low dose risk estimates must, therefore, be derived by extrapolation, and this has become a matter of heated debate and of continued controversy.

The diagrams in Fig.6 represent the excess relative risk (*ERR*) for solid cancer mortality and leukemia mortality of the A-bomb survivors in its dependence on dose (9). The values are "gliding averages" computed from the observed data for intervals (+/- 33%) around the specified doses. The shaded bands represent the standard error of the estimated *ERR*.

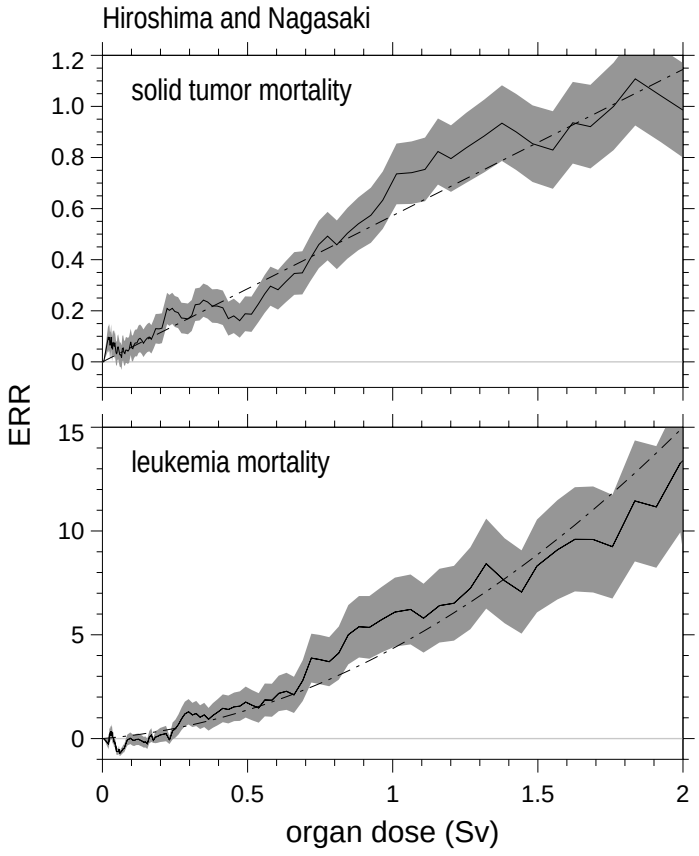

Fig.6: Non-parametric analysis of the excess relative risk, *ERR*, for solid tumors and for leukemia as a function of dose. The curves and the gray shaded bands represent a gliding average for intervals (+/- 33%) around the specified dose. *ERR* = 1 implies that the rate is doubled due to the radiation exposure. The broken lines represent a linear fit to the solid tumor data and a linear-quadratic fit to the leukemia data.

The dose dependence for all solid cancers follows a trend that seems to be linear. For leukemia it appears to be somewhat curved, the data suggesting little or no risk at low doses. In both instances the statistical uncertainty is too large at low doses, say below 0.2Sv, to permit meaningful direct estimates. Any such estimates must therefore be based on an extrapolation from the observations at high doses.

The ICRP nominal risk coefficient:

A conservative approach would base the risk estimates on a simple linear correlation in dose. The International Commission for Radiological Protection (ICRP) has taken a somewhat different point of view (6). Arguing that the leukemia data indicate reduced effectiveness at low doses and that animal experiments suggest likewise a reduction for solid cancers it recommended risk estimates at low doses that are only half as large as those obtained on the basis of overall linearity in dose. On this basis *a nominal risk coefficient* of 0.05/Sv has been derived for lifetime attributable cancer mortality. This is meant to imply that, for example, the exposure of 2 000 persons (distributed over age and gender) to 0.1Sv would be expected to cause 10 excess cancer deaths, in addition to the 400 to 500 cancer deaths that would normally occur (in a developed country) in such a group of 2 000 persons. It is readily seen that the order of magnitude of this risk estimate agrees well with the data given in Fig.6 for the solid cancer mortality which makes up the major part of total cancer mortality among the A-bomb survivors.

The ICRP assumption of a reduction factor for the derivation of the low dose risk estimates has been disputed. But other assumptions – for example the postulate that the excess relative risk for solid cancers remains constant throughout life – have been conservative. It is also likely that part of the observed effects that are currently ascribed to the γ-radiation have, in fact, been due to neutrons (10). When all aspects are taken into consideration, the nominal risk coefficient 0.05/Sv appears realistic, regardless of the controversial reduction factor.

While the study of the A-bomb survivors has become the major source of information on the risk of low radiation doses, studies of groups of patients exposed for medical reasons have largely confirmed the results, although they are usually less informative and tend, on average, to suggest somewhat lower risk numbers. Among the "low dose studies" the combined follow-up of several large groups of nuclear workers in Western countries has found particular attention. No dose related statistical excess has been seen for solid cancer mortality in this analysis, but the data are not statistically inconsistent with the current risk estimates, and this is true also for leukemia.

There has been considerable controversy about the *L*inear *N*o *T*hreshold (LNT) postulate that underlies the risk estimates, and there is, in fact, no definitive proof for this assumption. Epidemiology can not resolve the issue, since the few postulated excess cancers at low doses exhibit no "molecular markers" that would make them recognizable within the statistical noise of the spontaneous cancer incidence. Certain mechanistic considerations have been invoked to

support the idea of linearity in dose. They made use of the traditional ideas of target theory (11) by linking the assumption of linearity at low doses to the interpretation that even individual ionizing particles cause DNA damage, that some DNA damage will be misrepaired, and that certain rare mutations could thus be caused that enable the affected stem cell to initiate – with small but finite probability – a tumor. At very low doses where only few cells are affected by a particle, their number would be proportional to dose and so would be the excess cancers.

However, there are radiobiological observations (12-14) of complexities – such as *adaptive response, genomic instability,* or the *bystander effec*t – that could modify the cellular or tissue response at low doses. While such observations cast doubt on the, perhaps, too simplistic arguments that have been invoked in favor of linearity in dose, they are still inadequately understood, and it is unclear whether they might be relevant to late radiation effects and, if so, whether they would tend to decrease or enhance the response

Since there is no direct evidence for the low dose effects, the risk estimates and the ICRP nominal risk coefficient need to be seen as a pragmatic guideline (15). They are part of a prudent approach to radiation protection that accounts for putative – although statistically unrecognizable – risks by keeping them sufficiently low to be acceptable in comparison to other tolerated risk factors.

Radiation risks in perspective

Radiation protection is concerned with the small doses of up to 20mSv per year that may occur in occupational settings, and it is predominantly in this context that the ICRP nominal risk coefficient 0.05/Sv is used as guidance.

The annual limit of occupational exposure is currently 20mSv. This annual dose is rarely reached, and in one European country (Germany) a lifetime limit for occupational exposure has been set at 0.4Sv. The average exposure among the more highly exposed group of nuclear workers or the average dose for members of aircrews that regularly fly on certain long distance routes is close to 5mSv per year; if continued over a working life of 40 years this would add up to 0.2Sv. Space travel is a new condition where even higher doses can occur.

The most straightforward and the most common quantification of the risks of such relatively high doses or of the more common smaller exposures is given in terms of the number of expected excess cancer deaths. Thus, if 100 workers were actually exposed to a lifetime occu-

pational dose of 0.2Sv – under present conditions in Western countries a rather unlikely assumption –, the *collective dose* would be 20*person*Sv, and the ICRP nominal risk coefficient would then predict one excess cancer death in addition to the 20 to 25 cancer deaths normally expected among 100 persons. It is clear that this is a rather substantial risk – a putative 4% to 5% increase against normal cancer mortality – in a small group of persons. It is also clear that this magnitude of risk exceeds the job related fatality rate in most professions. If smaller exposures, but larger groups of persons are involved, the putative numbers of excess deaths are more difficult to judge, and they can, in fact, be highly misleading if very large populations and small – or even trivial – doses are involved.

To keep the magnitude of radiation risk in perspective, the putative number of excess cancer cases or cancer deaths needs to be quoted in relation to the spontaneous number expected in the exposed population, i.e. it makes far more sense to give the *relative increase* of cancer frequencies than the *absolute number* of excess cases.

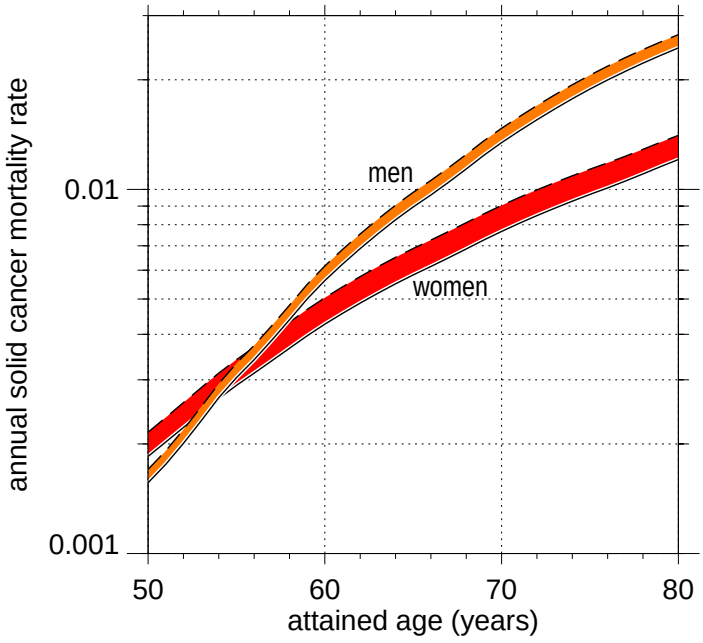

Fig.7: Solid cancer mortality rates and their increase due to an assumed occupational exposure to annual doses of 10mSv from age 25 to age 65. The lower solid lines represent the normal age specific rates for members of a West European population (UK). The red band represents the increase that is caused, according to the current risk model of ICRP (6), by the occupational radiation exposure. Average occupational lifetime exposures are substantially less than 0.4 Sv.

An added perspective for the magnitude of radiation induced increases of the cancer rates can be provided by seeing them not only in relation to the overall spontaneous rates, but also in their dependence on age or sex. Fig.7 provides such information. It gives as solid lines the age

dependent solid cancer mortality rates for men and women in a European population. Superimposed on the spontaneous rates is the increase that results according to the data of the A-bomb survivors from an assumed maximum occupational exposure to 0.4Sv during a working life from age 25 to 65. One notes the substantially larger cancer mortality rate for men, and realizes that the increase from the exposure could equally be expressed as an increase of the rates at specified ages or a shift towards increased age with regard to cancer. Either change is percentage wise larger for women than for men, but the lower overall cancer rate in women accounts for the fact that the absolute excess risk from a specified dose is not greatly dependent on gender.

Radiation protection used to be primarily directed at occupational exposures, but in the recent past the focus has shifted to the protection of the public and to questions that arise with regard to the costly management or restoration of contaminated areas, or to the regulation of radioactive emissions from nuclear industry or from nuclear medicine. This is not only a change in direction but also a shift to far lower doses than those that had earlier been of concern.

Within the European Community, the "dose limit" for the general population is presently set to 1mSv per year. Very little insight is gained by measuring such small exposures against the nominal risk coefficient 0.05/Sv that has been deduced from exposures hundred or thousand times larger. It is far more meaningful to relate them to the magnitude of the ubiquitous "natural" radiation exposure from terrestrial γ-rays, cosmic rays, radioactivity of the human body, and from radon in houses. The overall contribution from this natural radiation exposure is 1 or 2mSv per year. The regional fluctuations are substantial and the contribution from radon alone can, in radon prone regions, be far larger than the average total.

The annual limit of 1mSv for the general public has been based on the principle that any "controllable" exposure of the population – apart from the medical exposures that are justified by individual benefit – must not add appreciably to the natural radiation background. A limit to the public of 1mSv per year ensures that the average added population exposure is considerably smaller than this value and, in fact, the average population exposure from nuclear facilities or from commercial releases of radioactivity amounts to much less than 0.1mSv per year, which is clearly an insubstantial increase of the natural radiation exposure and lies well within the regional variations of the background level. This comparison to the natural background including its regional variations is a simpler and more assuring justification of the 1mSv annual limit to the public than any consideration in terms of numerical risk estimates.

Other late radiation effects

Hereditary effects:

Radiation induced increases of cancer rates are today seen as the major risk of low level radiation exposures. This used to be seen differently. When the potential risk of small radiation exposures was first recognized, the mutation of germ cells and the resulting hereditary damage were held to be more of a threat than the somatic mutations and the resulting increase of cancer rates.

Radiation induced heritable mutations have been studied extensively in mice, and experiments had earlier been conducted on plants and on *drosophila*. There can, thus, be no doubt that ionizing radiation produces mutations also in human germ cells. Yet, hereditary damage due to radiation has never been demonstrated in man, and this includes the extensive studies on the children of the A-bomb survivors. Even advanced methods of molecular biology, including sophisticated protein analyses and determinations of the mutation rates of mini-satellites, have failed to show in the children of the A-bomb survivors any association with the parental radiation exposures (16).

The absence of demonstrable hereditary radiation effects is, of course, no evidence against such effects in man. It merely means that any radiation induced increase of hereditary damage is difficult to detect in the presence of other factors that have much larger impact, such as the increasing age of the parents – in modern society – at the conception of their children.

The studies of the children of the A-bomb survivors can, on the other hand, be taken as proof that the doubling dose for hereditary damage is not much smaller than the dose of about 2Sv which doubles the total cancer rate in man. Even in the absence of precise numbers it is, therefore, a reliable conclusion, that substantial doses would be required to increase the rate of heritable diseases in an exposed population by a few percent. While a detailed discussion would be required of the long term impact of radiation induced genetic damage in an exposed population, it is sufficient in the present context to note that quantitative analyses have identified increased cancer rates – rather than hereditary effects – as the dominant detriment from low level radiation exposure.

Prenatal effects:

Prenatal radiation effects are sometimes confused with hereditary damage. In fact, they are quite distinct in not being due to a mutation in a single cell, being instead the result of extensive cell killing in the developing embryo or fetus. Prenatal malformations have resulted from improper medical application of x-rays to pregnant women in the early days of radiology, and they have also been observed among the children exposed *in utero* to the A-bomb radiation.

From animal studies it is known that the embryo is sensitive to ionizing radiation and that it dies after comparatively low doses. But it is unlikely to transmit damage if it survives, because its cells tend to be pluri-potent, so that they can replace any minor fraction of cells that is inactivated. The major risk of prenatal damage from radiation exposure occurs in the initial fetal period, and particularly in a critical phase between 8 to 13 weeks after conception when highly sensitive processes in the development of the central nervous system take place. Among about 1 600 children prenatally exposed to the A-bomb radiation, more than 20 were born with severe mental retardation which was due to radiation exposure in the critical period. The alarming conclusion is that in this period a dose of only 0.5Sv causes severe mental retardation in 2 out of 10 children. On the other hand, there is still reason to assume a dose threshold for such damage, because it is due to substantial cell killing which requires a certain dose level.

In the same context it needs to be noted that prenatal radiography, a common procedure in the first half of the last century, has been associated with increased leukemia and solid cancer rates in childhood (17,18). X-ray diagnostics of pregnant or potentially pregnant women is, by now, strictly avoided.

Increase of non-cancer mortality rates:

For a long time it had been assumed that the non-cancer mortality rates of the A-bomb survivors are entirely normal. In recent years this has been shown to be fallacious. There is now, in the group of the more highly exposed A-bomb survivors, a recognizable trend of increased general mortality rates. While these observations are not, as yet, of particular concern for radiation protection because the increases are seen only at substantial doses, the issue remains incompletely understood and will require continued study.

Expected and observed health effects from Chernobyl

The reactor accident in Chernobyl is recognised as the largest and most costly technical catastrophe ever - both in human and in economic terms. It is less clearly perceived, that the technical disaster has been amplified by a second disaster of comparable dimension, the continued failure to establish credible communication between administrations, scientific experts and a public that is deeply concerned, but fails to find the required guidance in a flood of contradictory information.

If the lasting confusion were confined to the countries of the former Soviet Union, it might be ascribed to years of secrecy and distorted information in a paralysed political system. In actuality the confusion has been equally deep and equally persistent in the much less affected societies of Western Europe.

When complex technologies and their inherent risks become unintelligible to the majority of the people, they are bound to lose viability. Nuclear technology and the perception of its potential risk are the perhaps most visible example. The fear of radiation is merely one aspect of the general concern, but it has become a focus. The presentation of scientific facts – even if they are well established – will not resolve a problem that has deeper roots than lack of technical information. But it is still a necessary component of any attempt to arrive at a realistic judgement.

Evacuees and liquidators:

Some aspects of the Chernobyl accident are fairly well understood and are subject to little controversy. Within days or a few weeks of the accident, 28 reactor employees and firemen died from acute radiation sickness. Others survived but continue to suffer lasting health damage. The accident caused the evacuation of about 120 000 people from the near zone around the reactor in 1986, and the later relocation of more than 200 000 people. Large territories of what is now Belarus, the Russian Federation, and the Ukraine were contaminated, and some level of contamination occurred in all countries of the northern hemisphere. About 240 000 emergency workers (so-called *liquidators*) were sent to the reactors or the evacuated 30-km zone in 1986 and 1987. Many of them – especially among those who worked on the reactors – must have been highly exposed, close to or even in excess of the 0.3Sv limit that was officially adopted but was unlikely to be reliably enforced.

A systematic follow-up of the liquidators and of the persons who had been evacuated from the 30km zone with delays of up to a few days would be desirable, but there is little hope for more than limited studies, because the people in question have been dispersed and their dose records are poor. According to the report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) (19) none of the existing epidemiological reports can, as yet, be seen as firm evidence for radiation related cancer or leukemia increases or the increase of other radiation related morbidity or mortality rates among the liquidators or in the evacuated populations.

The thyroid cancers:

The one dramatic radiation effect among the population in Belarus, the Ukraine, and Russia is the large excess of thyroid tumors. When very large activities of the short-lived radio-iodine were released during the accident, no measures were taken to reduce the consumption of fresh milk and vegetables, and no stable iodine was provided to the population to suppress the accumulation of radio-iodine in the thyroid. Very high thyroid doses were thus caused in children in the affected regions. The exposures occurred during the first days and weeks of the accident; thereafter when the radio-iodine had decayed, it was too late for remedial action. In the intervening years about 1 800 childhood thyroid cancers – an extremely rare disease under normal conditions – occurred (19). In adults an excess is less readily quantified, but it is likely to be present. The excess, especially in those exposed at young ages, will continue, with thousands of further cases expected. Although thyroid cancer is rarely lethal, it has major impact on the victim who requires permanent thyroid hormone substitution after successful therapy. There will also be a severe and lasting impact on the health services in the three affected republics: since the prognosis for the thyroid cancer patients depends critically on early diagnosis, extensive and costly mass screening for thyroid tumors will be required for many decades.

The current issue, the continued radioactive contamination and its impact:

The dramatic increase of thyroid cancers has been due to the high thyroid doses that were caused by the short-lived radio-iodine in the first phase after the reactor accident. The lasting concerns of the people in contaminated regions, however, and the discussions on the need for restrictions and for remedial actions are directed at a different issue, namely the continued contamination and the resulting elevated radiation levels. The exposure is predominantly due to cesium-137. It results from external exposure and, to a somewhat smaller extent, from the uptake of contaminated food. The exposures are much lower than the doses to the thyroid

from the initial uptake of radio-iodine. However the elevated radiation persists and exposes all organs of the body. Distrustful of official information, the population perceives it as a deadly threat widely taken to be responsible for assumed increases not only of cancer rates, but also of a multitude of other illnesses. Large numbers of persons in the affected regions believe that they are bound to perish from the surrounding danger.

In such a situation, the assessment of radiation risks is not an academic exercise. One needs to ask for the doses the population receives and one needs to relate these doses to earlier experience, in particular to the findings from the follow-up of the A-bomb survivors. In addition to this assessment – and regardless of its conclusions – one needs to observe the actual health statistics of the population.

UNSCEAR (19) reports population doses – from external and internal exposure to the long lived activity – that have been accumulated in the first ten years after the Chernobyl accident in the contaminated regions. These total doses from Chernobyl during the first ten years are given in Table 1 – together with the number of inhabitants – for Belarus, for the Russian Federation and the Ukraine, and for some of the subregions.

Table 1: Mean cumulated doses (excluding thyroid dose) from the Chernobyl accident in the contaminated areas during the period 1986-1995. Contaminated areas are taken to be the regions with initial radio-cesium concentration in excess of 37 kBq $\cdot$ m^{-2}.

Region	Population (inthousands)	Meandose mSv
Belarus	**1,881**	**8**
Brest	167	6
Gomel	1,465	7
Gomel (>555 kBq m^{-2})	78	40
Grodno	28	5
Minsk	25	6
Mogilev	195	18
Mogilev (>555 kBq m^{-2})	20	72
Russia	**1,983**	**7**
Bryansk	451	17
Bryansk (>555kBq m^{-2})	95	36
Tula	724	4
Ukraine	**1,296**	**11**
Zhytomyr	313	14

The major conclusion from Table 1 is that the average doses from Chernobyl in the contaminated regions – while being highly undesirable from a protection point of view – are never-

theless smaller than or comparable to the natural radiation exposure during this period which amounts to about 15mSv. These doses are, therefore, not very likely – even for leukemia as the most conspicuous indicator of whole body exposures – to cause an observable excess.

Figure 6 shows that in Hiroshima there has been no leukemia excess up to doses of 200mSv. Even if one were to assume that this was a statistical fluke and that the underlying dose dependence is, in fact, linear with a doubling dose of 200mSv, a mean dose of 10mSv would cause only a 5% increase over the normal leukemia rate for the lifetime of the exposed population, which would be difficult to detect. The excess could be substantially larger in the initial years after the exposure (see Fig.5), but the exposure was spread out over time after the accident and any peak of the excess rate is, therefore, unlikely to be very pronounced.

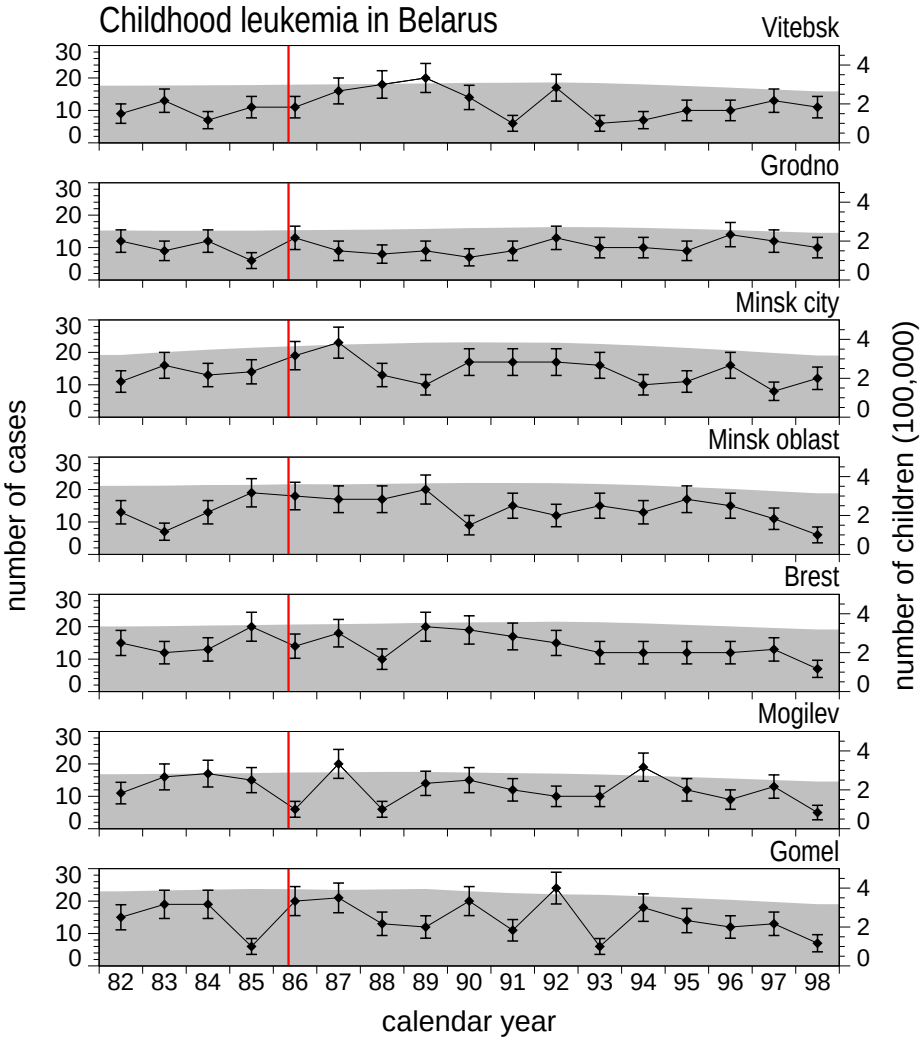

Fig.8: Annual childhood leukemia cases (age < 15 years) for the 7 major regions of Belarus. The bars on the points indicate the standard fluctuations that would occur according to Poisson statistics even if the rates were constant. The gray areas indicate the total number of children in the population (right ordinate). The time of the reactor accident is indicated by the vertical red line. No increase of the childhood leukemia rates after the reactor accident is seen in these data.

While it seems, thus, unlikely that an excess of leukemia will be seen in the overall rates, an increase might most readily be visible among children. Accordingly, special efforts have been made to monitor childhood leukemia rates, and Fig.8 gives the numbers for Belarus (20).

There is no indication of an increase either in the more highly contaminated regions Mogilev and Gomel against the less contaminated regions, nor in the period after the reactor accident against the period before.

If it is accepted that no increases of childhood leukemia have been seen even in the more highly contaminated parts of Belarus, it is concluded that the lasting radioactive contamination is unlikely to have caused recognizable increases in the rate of other cancers, and it is equally unlikely that it should have been responsible for generally increased morbidity rates from illnesses never before associated with low – or even high – radiation doses.

The contamination from Chernobyl has been much lower in the countries of Western Europe, with cumulated doses due to Chernobyl below 1mSv in all but a few smaller regions. However, there has been almost as much alarm and apprehension – at least initially – in some Western European countries as in the directly affected regions of the former Soviet Union. The alarm was justified where it was focussed on the fact that nuclear accidents can be an immense and far ranging threat, which demands greatly improved supranational safety conventions. A similar and equally essential alarm had been raised by the increasing number and magnitude of the atmospheric nuclear weapons tests in the 1950s. In both cases the alarm was also justified by the level of the radioactive releases that had already taken place. The nuclear weapons tests were in this respect even worse than the Chernobyl accident, since they caused an additional dose of about 1mSv not just in limited regions but throughout the Northern hemisphere. However, the individual health threat due to the comparatively small radiation doses was largely overrated, especially after the Chernobyl accident and especially in some Western countries. The present synopsis of expected and observed health effects from the Chernobyl accident may help to correct some of the misperceptions, but it is equally necessary to understand how such misinformation can arise.

125 000 radiation deaths in the Ukraine ?

A multitude of reports have kept alive the perception of increased cancer rates, increased rates of congenital malformations, and increased general morbidity and mortality as a result of the

elevated radiation levels in Belarus, the Ukraine, and the Russian Federation. The mere number of such reports, if seen with no possibility to assess their reliability or their degree of documentation, tends to discredit the statements by expert bodies and scientific committees. Even the United Nations Scientific Committee on the Effects of Atomic Radiation may encounter disbelief, when it acknowledges the increase of thyroid tumors due to the initial radioiodine exposures, but continues to say that otherwise "*there is no evidence of a major public health impact attributable to radiation exposure fourteen years after the accident. There is no scientific evidence of increases in overall cancer incidence or mortality or in non-malignant disorders that could be related to radiation exposure." (19)*

To disentangle facts and fallacies will remain a difficult task. But one example – a report that received considerable attention – may elucidate the mechanisms of negligent, if unintentional, misinformation.

A few years ago, at the 9th anniversary of the Chernobyl accident, the Minister of Health of the Ukraine was cited in almost all major international public media with the horrific news that, up to this point, 125 000 people had died, in addition to 6 000 "liquidators", from radiation effects. These numbers were taken to be the final confirmation that the worst expectations had been exceeded. In reality a statement of the ministry had been misunderstood and had been spread without further examination. The original statement read: "*The total number of deaths among the population in the most contaminated regions was more than 125 000 in the years 1988 to 1994*". It was then added that most of these deaths occurred among old people.

The report of the Ukrainian ministry was widely taken to refer to mortality caused by radiation – not a far fetched assumption for a deeply concerned public – in fact, however, the ministry had referred to *all deaths* in the contaminated regions. Inquiries at the Ministry of Health confirmed this all too evident fact. While no population number was quoted, it was known that the administration generally referred to about 2.2 million people in the most contaminated regions. In Western European countries the mortality rate is somewhat in excess of 1% per year. Assuming the smaller mortality rate of 0.9% per year for the probably somewhat younger population in the Ukraine, one would expect roughly 7·20 000 = 140 000 deaths in the period 1988 to 1994. The number that was seen as apocalyptic horror in the public media was, in fact, perfectly normal.

Conclusion

Half a century of naive confidence in the unlimited power of technical progress had been accompanied by unfounded beliefs in the positive health effects of x-rays and of radioactivity. The atomic bombs on Hiroshima and Nagasaki and the subsequent nuclear arms race have reversed this perception and during another half century the attitude against radiation and radioactivity has become increasingly critical. By now, fear and apprehension are directed against the peaceful uses of nuclear technology, and – focussed on radiation risk – they relate even to basic research and to the medical applications of radiation that help to save or restore countless lives.

It is a tragic aspect of radiation science that the most detailed insights on the late effects of ionizing radiation derive – as has here been outlined – from the use of the atomic bombs. The heritage of the nuclear arms race includes an equally dark source of knowledge which has only recently been uncovered. Along the river Techa in the southern Urals, high radiation exposures occurred a few years after the destruction of Hiroshima and Nagasaki. The exposures resulted from the release into the river of vast amounts of fission products that were generated in the secret plutonium plants of Mayak. Very high radiation exposures of the population along the river resulted, and thousands of workers at Mayak received similar radiation doses (21). These events were long kept hidden, but the exposures and their health effects are now the object of an international cooperation that is about to parallel and complement the studies on the atomic bomb survivors.

Much is known about the potential effects of low doses of radiation, but the issue will remain a major task for science and a challenge to those who have to convey scientific insights into general knowledge and into political decisions.

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