



On the Sensitivity of Children to Radiation

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Without a doubt, industrialization has made our lives richer, more pleasant and comfortable, and in the short term, healthier as well. But the price for this is high. Our living space was and is being invaded by both chemical and physical toxic agents. Through this, quality of life is being lost. The time will come when the loss outweighs the gain, when disease and early death from a contaminated environment increase faster than progress in medicine, nutrition, and hygiene can compensate - if that point has not been reached already.

In terms of environmentally provoked illnesses, cancer and leukemia come first to mind. This may be because the pathomechanism of carcinogenic development is basically known. Damage to the information in the genome of a single cell (in the area of the gene that controls cell division, growth, and differentiation) can finally lead to cancer following years or decades. This knowledge facilitates the acceptance of causality, to the extent that a high-intensity source is known (for example, the atomic explosion in Hiroshima). Further, cancer and leukemia are clear cut diagnoses that allow substantiation, and can therefore supply the necessary data

to the field of epidemiology to allow for causal connections.

However, it is much more difficult -- almost impossible -- to discern a connection when chemical and physical toxic substances have an influence in minimal activities and concentrations over a period of many years, and when the organism's response is not limited to cancer.

Indications of disturbances in the immune system are just as threatening as the increase in the rate of cancer and the younger ages of cancer cases [1a,1b]. Infectious diseases believed to be overcome long ago are appearing once again. (Health departments are registering an increase in new tuberculosis infections.) The increase in allergic reactions cannot be overlooked. (For example, less than 1% of the Swiss population suffered from hay fever in the 1920s; six decades later, that figure had risen to 15%, mostly children and young adults [2]. Allergic bronchial asthma and atopic eczema are also increasing; a prevalence of 3% to 10% for asthma and 10% to 15% for eczema during childhood is considered realistic.) This phenomenon can hardly be explained exclusively by an increase in outdoor and indoor allergens or by a change in their antigen characteristics.

Fundamental disturbances in the immune system may contribute to the phenomenon. The origin is unknown. With diagnoses that are difficult to substantiate, data are difficult to obtain. We will probably wait in vain for the discovery of causal connections that can be justified in secure, natural science terms. Nevertheless, it seems plausi-

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ble to assume the cause to be -- as it is in the case of cancer -- the increasing pollution of the environment with harmful substances.

Do Children Have a Higher Radiation Sensitivity Than Adults?

In general, cancer is the consequence of a primary injury, within the genome of a cell at some point by way of physical or chemical toxic agents (generally coming from the environment). Often, the primary injury is decades in the past. As such, for example, cancer developed in an adult might have its initiation in an unrepaired radiation injury stemming from the early childhood years.

A primary injury caused by minimal radiation exposure can remain without consequences in an adult: because of the latency of effects, the older person is less likely to experience the outbreak of cancer that might have resulted from this exposure. This is not the case for children: they are more likely to experience the long latent phase from primary injury to disease; as such, radiation exposure carries more consequences for them.

But this is surely not the only reason for an increased sensitivity to radiation. Many observations, of which some will be referred to below, support the assumption that a radiation exposure in utero and during childhood leads to cancer and leukemia not only more frequently, but earlier as well. Apparently, the cells in a growing organism are particularly radiation-sensitive. It seems that in rapidly dividing cells, the repair of mutations is less complete than it is in resting cells. Primary injuries to a cell's genome, no matter what their etiology, therefore remain unrepaired more often in children than in adults.

Therefore, in terms of the long-term consequences of the increasing contamination of soil, air, drinking water, food, and living spaces, we are less concerned with adults than with children and their fate in the coming decades. This concern extends to the chemical toxins from traffic, industry, agriculture, business, and waste incineration plants. This concern especially includes exposure to radiation -- globally from the explosion of nuclear bombs and nuclear power plant accidents, locally from the daily operation of nuclear plants, and individually from medical uses of radiation for diagnostic and therapeutic purposes, if the benefit and the probability of long-term diseases are not properly balanced.

Hiroshima and Nagasaki

In 1981, John Gofman published a rough estimate of the age-dependent radiation risk in his book *Radiation and Human Health* [3].

After assessing numerous reports on the consequences of radiation, including the studies of the survivors of Hiroshima and Nagasaki, he determined that the risk of developing cancer is relatively very high in utero and among newborns; it decreases in the course of life. It is three to four times higher among infants than among 20 year-olds, whose risk in turn is greater than that of 40 year-olds by a factor of three, and more than 30 times greater than that of 60-year-olds. After the 50th year of life, the risk of cancer resulting from a radiation exposure in the low-exposure range becomes negligible.

The cancer registries from those exposed to the atomic bomb in Hiroshima and Nagasaki have yielded, under several different analyses, several sets of risk coefficients. They refer not to illness, however, but to fatalities (the difference here is significant depending on how one views the relative efficacy of modern cancer therapy for different types of cancer). These risk coefficients also do not differentiate among various age groups. Until recently, the coefficient used by the International Commission on Radiological Protection (ICRP) from the year 1977 was quoted for official risk assessment (ICRP-26) as 125×10^{-6} man rem [2]. However, further analysis of the Hiroshima and Nagasaki data and revisions in dosimetry resulted in the cancer risk among the survivors of the atomic blasts being determined as at least 10 times, and perhaps as high as 20 times greater (RERF-1987). After considerable hesitation, the ICRP also considered itself obligated to increase the risk coefficient; however, only by a factor of 4 (ICRP-61) to 500×10^{-6} man-rem, arguing that radiation consequences are lesser in the low-exposure range. (There is little evidence for this; in reality, the opposite seems to be the case [4]).

In his analyses of the 1987 cancer statistics in Hiroshima and Nagasaki, Radford [5] found that the risk of an early cancer death among those who were exposed to the atomic explosion as children in 1945 is up to eight times greater than among survivors as a whole. Eight times the maximum figure given in 1987 by the Hiroshima Radiation Effects Research Foundation as the risk range for all age groups (RERF-1987) [6] does, in fact, correspond to the childhood risk coefficients reached by the Gofman analysis

Prenatal X-ray Exposure

Thirty years ago it was still common in the field of obstetrics to use X-rays to "measure the pelvis" of pregnant women (pelvimetry). As I know from my own clinical work back then, nobody thought twice about it. The understanding that this mea-

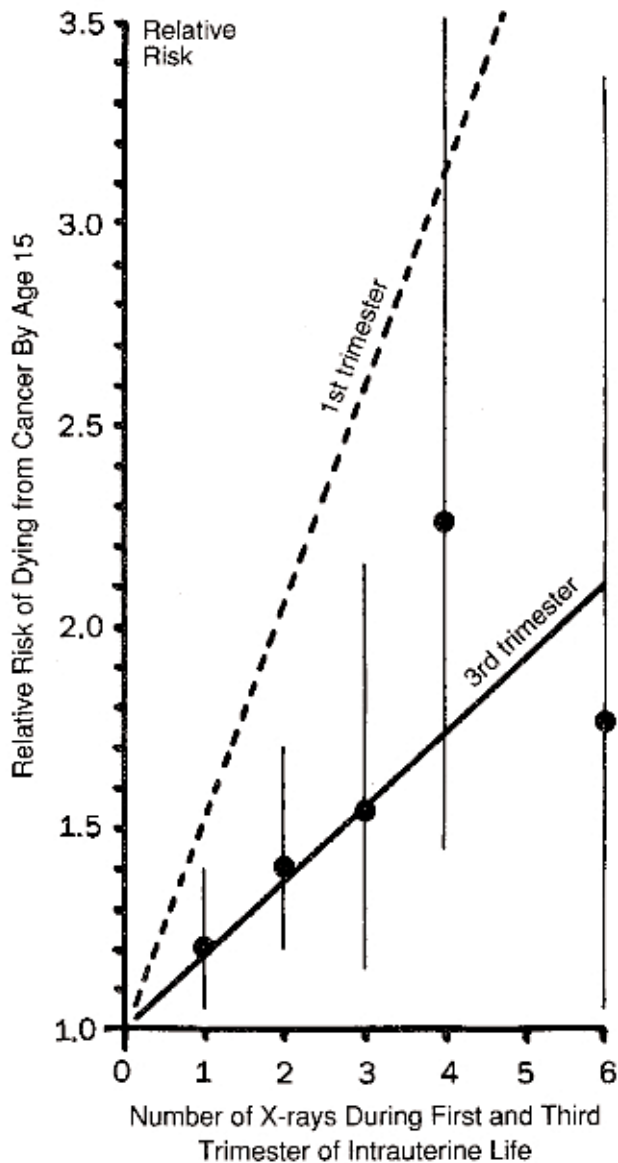


Figure 1. Children's relative risk of dying of cancer (including leukemia) by the age of 15 when their mother had been exposed to X-ray irradiation during the third trimester of pregnancy. Mean values with a 95% confidence level, according to Bithell [12]. The adapted straight line inclines by 0.2 units of additional risk per X-ray (0.13-0.28). The relative risk of X-rays in the first trimester is also indicated, calculated according to the relationship of the risks in the first and third trimester of 3.8:1.3 [12].

sure was anything but harmless is thanks to British physician and epidemiologist Alice Stewart. In 1956, she first expressed the suspicion that children were more likely to contract cancer if their mother had an X-ray examination during pregnancy [7]. She was often attacked for her views; criticism of perceived progress in medicine, especially of a technical nature, can on occasion be seen as tantamount to sacrilege. Thanks to the Oxford Childhood Cancer Registry, which

was established back then for Great Britain, numerous studies over the years have confirmed Stewart's initial suspicion [8a,8b,8c,9]. Other similar studies worldwide, for example, in the U.S. [10] have produced results that support this concern. In the mean time, it can no longer be disputed that pelvimetry, which is mentioned in at least some radiology text books [11] with "1,200 mrem fetal whole-body radiation," can double a child's cancer risk.

Figure 1 shows the current state of the analysis of the Oxford Childhood Cancer Registry [12]. The more X-ray exposures that were done, the greater the risk. The risk increases with earlier irradiation (the average relative risk following a radiation exposure of 1 rem is estimated at 3.8 for the first trimester and at 1.3 for the third).

According to the Oxford studies, depending on the amount of radiation exposure a fetus supposedly will receive through one film, and whether the calculation is based on only the third trimester or on the entire pregnancy, one arrives at a risk coefficient of between 300 [12] or 2,000 [9] deaths per 1 million children exposed to 1 rem in utero. These figures are not significantly higher than the risk coefficients calculated for the entire survivor cohort of Hiroshima and Nagasaki (ICRP-1977:125, RERF-1987:600-1,300, ICRP-1990:500). However, it would be a mistake to conclude from this that the unborn do not have a significantly greater sensitivity to radiation than adults. First, we can assume that a not insignificant number of the exposed fetuses in the Oxford study were injured so acutely that they did not survive the pre- or perinatal phase, and therefore could not appear in the Oxford Childhood Cancer Registry as "having died of cancer or leukemia." Second, only cancer cases that led to death by the age of 15 were counted. It is possible that other cancers caused by this in utero imaging would appear after the period of observation.

X-ray Exposure of Children

In the 1950s, on their immigration to Israel, 11,000 children (half of them between the ages of 6 and 9) were treated with X-rays for a fungus infection of the roots of the hair (tinea capitis). Shortly thereafter, a prospective study was initiated; the exposed children were compared with a control group composed of 5,400 siblings and 11,000 children from the general population. Based on phantom trials, it was estimated that among exposed children the following organ doses had been received: 9 rem to the thyroid gland, 5 to 7 rem to the pituitary gland, and 1 to 2 rem to the chest [13]. Since then, the two

Table. Relative Risk of Contracting Cancer as an Adult Among Persons Exposed to X-ray Irradiation During Childhood*

	Years following exposure	
	15 -25	25-35
All types of cancer	1.8 (1.4-2.2)	1.7 (1.2-2.3)
Head and neck	3.1 (2.3-4.4)	3.4 (1.7-6.9)
Thyroid	4.2 (2.6-6.7)	3.7 (1.04-14)
Leukemia	2.6 (1.3-5.3)	1.5 (0.2-15)
Breast	1.0 (0.5-1.8)	2.1 (1.1-4.2)

*Therapeutic X-ray treatment of the scalp with a dose of 9 rem for the thyroid and 1 to 2 rem for the chest

Risk of the control group = 1; 95% confidence level

Modan B, Chetrit A, Alfandary E, Katz L. Increased risk of breast cancer after low-dose irradiation. *Lancet* 1989;March 25;629-631

groups have been under careful observation.

Twenty years later, the incidence of thyroid cancer had increased by a factor of four among those who were exposed; other cancers in the head and neck area had increased by a factor of three, and the rate of leukemia has more than doubled (Table). In 1989, a twofold increase in the rate of breast cancer was reported among the women in the exposed group, who were at that time 35- to 45-years-old. Especially affected are those women who were of grade-school age at the time of their exposure; in this group, the relative risk is greater by a factor of 10 [13]. The radiation dose to the chest area in this group was less than the then allowable annual dose for radiation workers (5 rem). (One to 2 rems correspond to the radiation exposure of the breast from two X-rays of the thoracic spinal column or from five or 50 chest radiographs, using older or the most modern technology, respectively [13].)

Thyroid Cancer Following the Chernobyl Accident

The radioactive cloud that rose above the damaged reactor in Chernobyl on April 26, 1986, initially drifted northward over Belarus. Four days later, it reached southern Germany. In Munich, on May 1, the gamma dose output from the cloud was about 0.1 mrem per hour, mostly from the radiation of the short-lived radioisotopes iodine-131 and tellurium-132. In large areas of Belarus, it was 50 to 200 times higher for several days than the relatively short maximum reached in Germany [14]. Radioactive iodine was ingested with milk and vegetables and was inhaled. In southern Bavaria, children's thyroid glands may have accumulated up to a level of 3 rem; in Belarus, in contrast, up to 1,000 rem or more.

The May 1991 report of the International Atomic Energy Agency (IAEA) [15] states that while the health situation of the popula-

tions in the highly exposed regions of the former Soviet Union is generally not good, no increase in the rate of illnesses could be determined that could be directly attributed to radiation exposure. Page 116 of the chapter "Thyroid Cancer" states:

There is no clear pathological documented evidence of an increase in thyroid cancer...most of the reports of thyroid cancer were anecdotal [meaning the survivors from Hiroshima and Nagasaki] and thyroid cancers in other exposed populations have not occurred within 10 years of exposure.

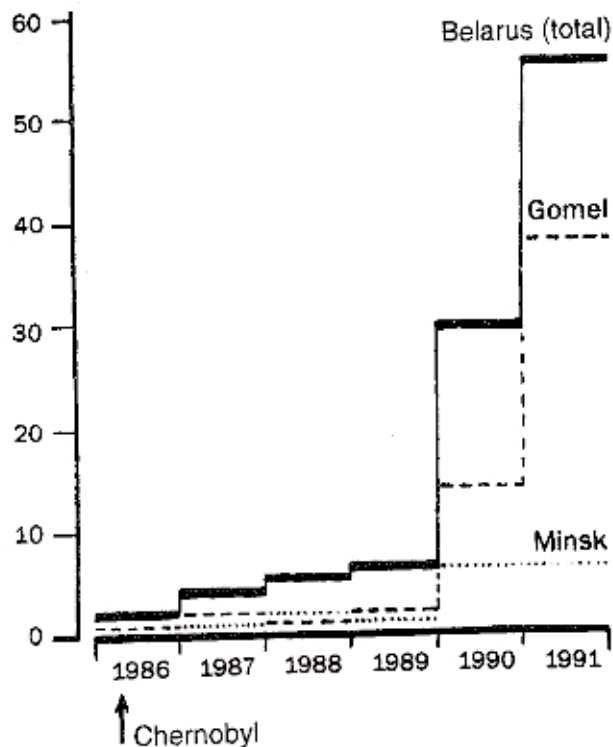


Figure 2. Number of diagnosed cases of thyroid cancer in children in Belarus. See reference 17.

By 1991, it was well known that there was an increasing number of thyroid tumors being diagnosed among children in the district hospital of Gomel and many of these were classified as malignant. The latter are extremely rare in children. From 1977-1986, in Belarus (with approximately 14 million inhabitants), only seven cases of childhood thyroid cancer were registered: zero to a maximum of two per year. By 1989, the number of cases had increased to six; in 1990 to 29, and in 1991 to 55 [16]. From the Chernobyl accident until mid-1992, 131 children were diagnosed with thyroid cancer; half of the children are from the district of Gomel, the most seriously exposed of Belarus's seven districts (Figure 2).

Chernobyl Syndrome

The term "radiation injury" initially brings to mind acute damage following radiation above a certain threshold: at 50 rem, radiation sickness with vomiting, hair loss, mucous membrane ulcers; above 250 rem, acute bone marrow depression with anemia, bleeding, immunodeficiency; above 500 rem, death from radiation [17]. Then, one thinks of cancer and leukemia that manifest years or decades after the radiation exposure. Revised dosimetry from Hiroshima taught us that gamma radiation can cause cancer even in the relatively low-dose range. Just as Hiroshima then has proved instructive with respect to external radiation by energy-rich photons, Chernobyl is instructive today with respect to incorporated radioactivity.

The radiation deaths of 31 workers and firemen in May 1986 (officially the only victims of radiation exposure), and thyroid cancer beginning in the fifth year, fit into the textbook knowledge of deterministic early injury and stochastic late injury, respectively. However, new and completely unexpected (according to the textbooks) are symptoms among those exposed at Chernobyl that have been observed since the third year with increasing frequency and intensity: anemia, hemorrhagic diathesis, immunodeficiencies. Children are especially affected. The clinical symptoms are not uniform and therefore difficult to assess.

Those symptoms, which have been described by Jurij Stscherbak, former Kiev medical officer, as "Chernobyl-AIDS" [18], are typical of a chronic bone marrow depression. The following pathomechanism is possible: strontium-90, traces of which are in food, becomes incorporated into growing bones and accumulates there. Prolonged exposure of the bone marrow to this incorporated radiation results in the loss of stem cells (precursor cells for erythrocytes, thrombo-

cytes, and all cells of the immune system) as a deterministic early injury. This loss can be compensated for by the multiplication of unaffected stem cells, but continuing accumulation of bone-seeking radionuclides will eventually exhaust this capacity.

Experimental data documenting this subacute early radiation injury have been available for quite some time. In 1968, experiments with animals showed that extremely low doses of strontium-90 are sufficient to affect the capacity of bone marrow to compensate for stem cell loss; a dose of 0.01 rem results in a reduction of the number of bone marrow cells [19].

To explain these symptoms of the children exposed after the Chernobyl accident with reference to a possible strontium bone-marrow mechanism remains a hypothesis, but much speaks in favor of it: the high strontium contamination in the highly exposed regions, the appearance of symptoms only after a three-year latency period and mostly among children, the characteristics of strontium, and the biology of bones.

It is not out of the question that this heretofore unnoticed pathomechanism is also at work in the area of nuclear installations. A nuclear power plant constantly emits traces of radionuclides; traces enter the food chain through the soil; a portion of this radiation could be incorporated into the growing bones. That which might seem minimal and completely harmless in terms of emissions at any given moment could accumulate over the course of several years into a significant radiation exposure of the bone marrow and could be the cause of various diffuse health disturbances in children and young people.

Chernobyl has opened our eyes: in addition to acute radiation death and cancer, there are a multitude of possible injuries to health that have not been considered before.

Radiation Sensitivity and the Molecular Biology of Repair Mechanisms

All organs whose cells have a high frequency of division are highly radiation-sensitive, in children as well as in adults. Among the survivors of Hiroshima and Nagasaki and in the fallout area following nuclear tests, the most commonly seen forms of cancer were leukemia and cancer of the breast, prostate, and thyroid. Leukemia (a cancer of the most radiation-sensitive organ, the bone marrow) is particularly prevalent in childhood (the most radiation-sensitive phase of human life). That is the reason why childhood leukemias are looked for in the areas around nuclear plants as a "biological radiation indicator." (Of the 11 malignant blood

diseases close to the Krummel nuclear plant near Hamburg, which occurred within three years, beginning with the sixth year of plant operation -- six are in children under the age of 10.)

The phenomenon of high-radiation sensitivity can be explained in terms of the efficiency by which mutations of DNA caused by radiation are repaired [20]. The process of recognizing a mutation, excising the mutated DNA section, the accompanying resynthesis and strand linking is complex and lengthy; up to 10 different enzymes must work together in concerted action [21,22]. Some researchers have claimed that "cells have highly active enzymes at their disposal, which within seconds completely repair radiation damage up to a certain point" [23]. This mechanism is less straightforward in mammalian cells. The repair processes of mammalian cells depend upon the phase of the cell cycle [24]. The younger the organism, the faster the tissue grows, the faster the cells therein divide, and the shorter the cell cycles. In the case of short cell cycles, there may not be enough time for the repair of an injury caused by radiation. If the mutation is not repaired in time, the injury is usually permanent after the subsequent cell division.

Additionally, the effectiveness of the repair depends on the functional condition of the cell. Mutations can set in during any phase of a cell cycle; the nucleic acids are subjected constantly and without protection to ionizing radiation, no matter what degree of organization they have at that moment. However, a mutation can only be recognized and repaired if the appropriate enzymes have access to it. This is the case with an "active" gene whose information is being summoned. The functional condition of a cell, the hormonal signals that steer it, the timespan until the next doubling of the genetic make-up, the spatial relationships of a mutated region within a chromosome -- all of these determine whether and how quickly a mutation will be repaired.

From a molecular biology perspective, the varying effectiveness of repairs could also explain why tissues with a high rate of division (this includes all tissue of a growing organism, as well as bone marrow, glandular organs, and mucous membranes of the fully grown organism) are particularly radiation-sensitive. The latency period also depends on the rate of cell division between the time of the initial primary injury and the outbreak of cancer. Consistent with this notion is the fact that leukemia is the most common cancer among small children.

Due to the short latency period, childhood leukemias are considered indicators of

increased exposure to mutagenic toxic agents. If childhood leukemias appear in large numbers near a nuclear power plant, within a clearly defined area and time span, then in the search for cause, radiogenesis is a well-founded hypothesis. On the other hand, official experts may claim that the possible doses calculated from measured radioactive emissions are too small to be the cause for the cluster of leukemia.

Summary

As I have discussed, based on accepted data and reasoned speculation, there are three scientifically grounded explanations for the fact that the radiation sensitivity of the old and young can vary by one to two orders of magnitude:

- * First, because there generally is a long latency period between the primary injury (unrepaired mutations in a cell's DNA) and the outbreak of a cancer, children are more likely than older people to experience the long-term consequences;

- * Second, the bone-seeking radionuclides accumulate more rapidly in growing bones than in bones of the adult, with the possible consequence of an alteration of the immune system, resulting not only in impaired defense against infection, but also in impaired ability to recognize and kill cancer cells;

- * Third, in cells with a high frequency of division (such as those in a growing organism), there is a higher probability that mutations of DNA will not be repaired as rapidly or completely as mutations occurring in adults.

The science of medicine is reluctantly recognizing that X-ray technology has not only brought many benefits, but has caused suffering as well. In practice, much has already changed. (We remember with a shudder the X-ray machines in shoe stores and the mass X-ray screenings). Despite this, it cannot be emphasized enough that the medical use of radiation in children must be limited to that which is absolutely necessary. The benefit to the young patient must be clearly greater than the probability of disease in adulthood occurring as a consequence of childhood exposure [14,25].

In addition, since nuclear war is always possible as long as nuclear weapons are in a standby position and since a supercatastrophe at a nuclear power plant would contaminate vast land areas and pose a serious

health risk to all residents in these areas, especially children, we must ask ourselves two questions. Given the high radiation sensitivity of the unborn, infants, and children, can the constant increase of the radioactive inventory on this earth for military and civilian use be justified? Do the security and wealth that allegedly result from military and civilian technology warrant the danger to the health of our children and the risk of a catastrophe that would mean the end of human civilization? For all those who are concerned with the health and life of the coming generations, the answer can only be NO! ❦

References

- 1a. Patorok EM. Brustkrebs: Lebensalter und Tumor durchmesser. *Geburtshilfe und Frauenheilkunde* 1986;46:898-890.
- 1b. Patorok EM et al. Mammakarzinom: Trends von 1964-1990, Ergebnisse einer Langzeitstudie. *Roentgen-Praxis* 1992;45:325-329.
2. Wuthrich B. Epidemiologie atopischer Erkrankungen. *Der informierte Arzt - GazeHe Medica* 1992;6a-90:557-566.
3. Gofman JM. Radiation and human health. San Francisco: Sierra Club Books, 1981; Tables 21 and 22.
4. Kohnlein W, Nussbaum R. Reassessment of radiogenic cancer risk and mutagenesis at low doses of ionizing radiation. *Advances in Mutagenesis Research* 1991;3:53-80.
5. Radford EP. Recent evidence of radiation induced cancer in the Japanese atomic bomb survivors. In: *Radiation and health: the biological effects of low level exposure to ionizing radiation*, 1987;87-96.
6. Preston DL, Pierce DA. The effects of changes in dosimetry on cancer mortality risk estimates in the atomic bomb survivors. *Hiroshima: Radiation Effects Research Foundation, Hiroshima, 1987 RERF TR-9-87*.
7. Stewart AM, Webb J, Giles D, HewiH D. Malignant diseases in childhood and diagnostic irradiation in utero, *Lancet* 1956; September 1:447.
- 8a. Stewart AM. Leukemia and prenatal X-rays. *BMJ* 1960;11:1381.
- 8b. Stewart AM, Kneale GM. Radiation dose effects in relation to obstetric X-rays and childhood cancers. *Lancet* 1970; June 6:1185-1187.
- 8c. Bithnell JF, Stewart AM. Prenatal irradiation and childhood malignancy. *Br J Cancer* 1975; 31 :271-287.
9. Knox EG, Stewart AM, Kneale GW, Gilman EA. Prenatal irradiation and childhood cancer. *Journal for Radiation Protection* 1987;7:177-189.
10. Diamond EL. The relationship of intrauterine radiation to subsequent mortality and development of leukemia in children. *Am J Epidemiol* 1973,97:283-289.
11. Lissner J, Hug O. Gegenstand: Radiologie. Stuttgart: Ferdinand Enke Verlag, 1975:(Table 3-1)67.
12. Bithell JF. Epidemiological studies of children irradiated in utero. In: Baverstock K, Stather, eds. *Low dose radiation: biological bases of risk assessment*. 1989;77-87.
13. Adzersen KH. Medizinische Strahlenbelastung in der Bundesrepublik Deutschland: Möglichkeiten der Dosisreduktion" In: *Berichte des Otto-Hug-Strahleninstituts Bonn*, No. 3, 1990.
14. Israel J. Tschernobyl: Vergangenheit und Prognose für die Zukunft. *Prawda*, 29 March 1989 (translated into German by Sebastian Scholz, printed in IPPNW newsletter No. 29).
15. International Atomic Energy Agency (IAEA) Summary of thyroid neoplasms. In: *The radiological consequences in the USSR of the Chernobyl accident*, Vienna: IAEA, 1991; Vol. II, part F, 116.
16. Kazahov VS, Demidchik EP, Astakhova LN, et al. Thyroid cancer after Chernobyl. *Nature* 1992;359:21-22.
17. Lengfelder E. Strahlenwirkung, Strahlenrisiko: Ergebnisse, bewertung und Folgen nach einem kerntechnischen Unfall aus ärztlicher Sicht. 2nd and expanded edition. Landberg: Ecomed-Verlag, 1990.
18. Stscherbak J. Das Chernobyl-AIDS greift um sich. In: *Suddeutsche Zeitung*, 7 April 1990.
19. Stokke T, Oftedal P, Pappas A. Effects of small doses of radioactive strontium on the rat bone marrow. *Acta Radiol* 1968;7:321-329.
20. Scholz R. Strahlensensibilität und DNA-Reparatur-Zum Mechanismus und Polymorphismus von Enzymsystemen für die Reparatur von DNA-Schaden. In: Kohnlein W, Kuni H, Schmitz-Feuerhake, eds. *Niedrigdosisstrahlung und Gesundheit - Medizinische, rechtliche und technische Aspekte*. Heidelberg: Springer-Verlag, 1990.
21. Friedberg EC. DNA repair. New York: Freeman & Company, 1985.
22. Schweiger M, Auer B, Burtscher HJ, Hirsch-Kaufmann M, Klocker H, Schneider R. DNA repair in the human cell: biochemistry of the hereditary disease Fanconi's anaemia and Cockayne syndrome. In: *Biological chemistry*. Hoppe Seyler 1986;367:1185-1195.
23. Wachsmann F. Die Strahlengefahr - realistisch gesehen. *Naturwissenschaften* 1989;76:45-51.
24. Bohr VA, Wassermann K. DNA repair at the level of the gene. *Trends Biochem Sci* 1988;13:429-433.
25. Kuni H. Die gesundheitlichen Folgen des Umgangs mit radioaktiven Stoffen. In: Bastian T, Bonhoeffer K, eds. *Thema: Radioaktivität*. Edition Universitas. Stuttgart: Wissenschaftliche Verlagsgesellschaft, 1991.