

Health effects of radioactivity

Jan Willem Storm van Leeuwen
Independent consultant

member of the Nuclear Consulting Group

July 2019
storm@ceedata.nl

Note

In this document the references are coded by Q-numbers (e.g. Q6). Each reference has a unique number in this coding system, which is consistently used throughout all publications by the author. In the list at the back of the document the references are sorted by Q-number. The resulting sequence is not necessarily the same order in which the references appear in the text.

Contents

Summary

- 1 Some basics on radioactivity
 - Interaction of radioactivity with living matter
- 2 Biological properties of radionuclides
 - Tritium and carbon-14
 - Bioaccumulation in the food chain
- 3 Health effects
 - Deterministic effects
 - Stochastic effects
- 4 Attribution of health effects to radioactive exposure
 - Deterministic health effects
 - Stochastic health effects
 - View of UNSCEAR
 - Falsification
- 5 Biochemical aspects of radioactivity
 - Accumulation in specific organs
- 6 Non-targeted and delayed effects
- 7 KiKK study
- 8 COMARE
- 9 Geocap study
- 10 Chernobyl, the IPPNW study
 - Liquidators
 - Population
 - Radiation-induced diseases
- 11 Reliance on models
- 12 Radiation dose measurement
- 13 Radiation hormesis

References

FIGURES

- Figure 1 Radioactive decay of tritium
- Figure 2 Half-life of radioactive decay
- Figure 3 Pathways of tritium and carbon-14 into the human body
- Figure 4 Concentrations of radionuclides in environmental indicator materials
- Figure 5 Relationship between cancer incidence and distance to an NPP
- Figure 6 Incidence of thyroid cancer in Belarus 1985-2004

Summary

Health effects due to nuclear radiation and/or contamination by radionuclides are divided into deterministic effects (e.g. acute radiation syndrome) and stochastic effects. Attribution of deterministic effects to exposure of a particular individual to radioactivity is relatively easy due to the generally specific symptoms and the short latency period (minutes to months). Attribution of stochastic health effects of a particular individual to exposure to radioactivity is much more difficult, chiefly for reason of the long latency period (months, years, decades) and the often non-specific symptoms. It's not possible to predict which individual will develop which health effect due to exposure to radioactivity.

UNSCEAR and IAEA (and with the IAEA also the WHO) do not recognize non-cancer diseases as possible stochastic health effects caused by radioactivity.

The biochemical behaviour of radionuclides in the human body after ingestion by air, food and/or water, and the consequences of the radioactive decay in living cells are poorly understood or even not investigated. Some radionuclides are rapidly incorporated in biomolecules (e.g. DNA). The consequences of this phenomenon are hardly known. Synergistic effects of a number of different radionuclides in the body are hardly known either.

During the past decade new radiation-induced effects have been observed in living cells, the so-called non-targeted and delayed effects, which can cause damage to cells which were not exposed to radiation. There is still no scientific explanation of these effects.

The list of possible health effects of exposure to radioactivity is long. Elaborate epidemiological studies can demonstrate the causal relationship between health effects and radioactivity.

German and French epidemiological studies proved the relationship between childhood cancer (investigated until age 5) and the distance the children lived in relation to a nominally operating nuclear power plant. These empirical observations cannot be explained by the classical radiological models on which the nuclear world bases its assessments.

Based on large numbers of publications in Russian, which are usually not read in countries outside the former Soviet Union, several studies assessed the consequences of the Chernobyl disaster. The conclusions of these studies, which are based on empirical observations, are strikingly different from the findings of the IAEA/WHO. A high incidence of a number of diseases, such as premature senescence, hereditary disorders, and congenital malformations, are observed in addition to different kinds of cancer. Estimates of the number of deaths attributed to the Chernobyl disaster vary from roughly 100000 to nearly one million.

Downplay and denial of radioactivity-induced health effects by the IAEA and WHO are discussed in detail in the report **mo5** *Downplaying and denial of health effects*.

1 Some basics on radioactivity

Radioactivity, or radioactive decay, is the phenomenon where the unstable nucleus of a radioactive atom spontaneously decays into another kind of atom, coupled with the emission of nuclear radiation: alpha, beta and/or gamma radiation.

Atoms are composed of a nucleus surrounded by electrons. The nucleus consists of protons and neutrons. The number of protons determines to which chemical element the atom belongs, for the chemical properties of an atom are determined by the number of protons. The number of neutrons may vary; atoms with an equal number of protons but a different number of neutrons in the nucleus are called isotopes. The chemical properties of isotopes are identical. Some isotopes have an unstable nucleus and are called radioisotopes for they are radioactive.

Because in nuclear science generally only the nucleus of a given atom matters, usually the term nuclide is used: a nuclide is an atom with a specific kind of nucleus. If the nuclide is unstable it is called a radionuclide.

In the radioactive decay process a nuclide usually transforms into a nuclide of another chemical element. When a radioactive hydrogen-3 atom (H-3) decays, a helium-3 atom (He-3) comes into being. The nuclide (atom) which results from a decay reaction is called a decay product or decay daughter. Often the decay daughter is radioactive itself.

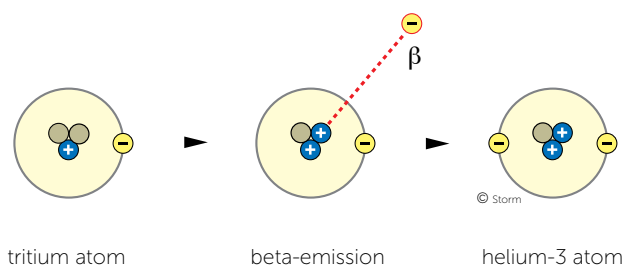


Figure 1

Radioactive decay of radioactive hydrogen, tritium. Tritium, symbols T, ^3H or H-3, is a heavy isotope of hydrogen, with one proton and two neutrons in the nucleus. When a tritium atom decays, it emits a beta particle (an electron) at high speed. After decay the nucleus contains two protons and one neutron, the nucleus of a helium-3 atom, which captures a second electron and becomes a neutral helium-3 atom. The sums of electric charges remain constant and a minute fraction of the mass is converted into energy.

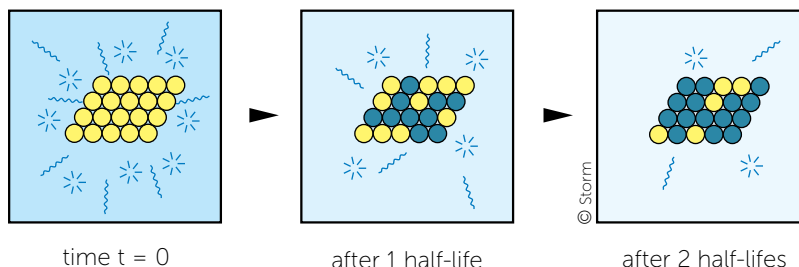


Figure 2

Decay of a radionuclide. One half-life period after creation of a given amount of a certain radionuclide at time $t = 0$, half of the radionuclides has decayed into another kind of nuclide, called the daughter nuclide. In this example the decay daughter is a non-radioactive, stable nuclide. During the next half-life period half of the remaining radionuclides decay, and so on. The total mass of matter remains almost constant during the decay process.

The half-life of a radionuclide is defined as the time period in which half of a given amount of radionuclides has decayed; this quantity is specific to each kind of radionuclide. The decay rate cannot be decelerated or accelerated by any means. Radionuclides occurring in nature, such as uranium and thorium, have very long half-lives measured in billions of years. Human-made radionuclides have much shorter half-lives, ranging from seconds to millions of years. The specific radioactivity of a radionuclide is measured in becquerel per gram, Bq/g (number of disintegrations per second per gram) and is higher as the half-life is shorter.

Radioactivity cannot be destroyed nor made harmless to man and other living organisms.

Interaction of radioactivity with living matter

Nuclear radiation is often called *ionising radiation*, because it strongly interacts with matter forming ions. Ionising radiation is harmful to living organisms, for it destroys or modifies biomolecules, such as DNA. Alpha radiation can be blocked by thick paper and beta radiation by aluminum foil, so these rays may seem not very harmful to man. However radionuclides radiating alpha or beta rays inside the human body are extremely dangerous, because the living cells are not protected by the skin or clothes. The energy of the alpha or beta rays are given off within a short range and causes a large number of damaged biomolecules inside living cells. For example a dose of only a few nanograms of the alpha-emitter polonium-210 in the human body is lethal.

A complicating factor is that alpha and beta radiation are hardly or not detectable by most hand-held counters; radionuclides that emit weak or no gamma rays are invisible to these detectors. A number of biologically very active radionuclides fall within this category, such as tritium (radioactive hydrogen), carbon-14 (radioactive carbon), radioiodine and a number of actinides. These radionuclides can be detected only by special equipment

2 Biological properties of radionuclides

Tritium and carbon-14

Of special importance are the radionuclides tritium (symbol ^3H , H-3 or T) and carbon-14 (symbol ^{14}C or C-14). As pointed out above these radionuclides are biochemically indistinguishable from their non-radioactive isotopes, normal hydrogen H, respectively normal carbon (mainly ^{12}C). Carbon and hydrogen are two of the six primary building blocks (C, H, O, N, S, P) of proteins and DNA. An aggravating factor is that both radionuclides are always discharged simultaneously.

Some fission and activation products, especially tritium and carbon-14, generated in a nuclear reactor are completely released into the environment by nominally operating nuclear power plants and the interim storage facilities of spent fuel. Tritium reaches the environment as tritiated water HTO, carbon-14 is mainly discharged as radioactive CO_2 . It dissolves in rain water as hydrogen carbonate ions and so enters groundwater and the food chain.

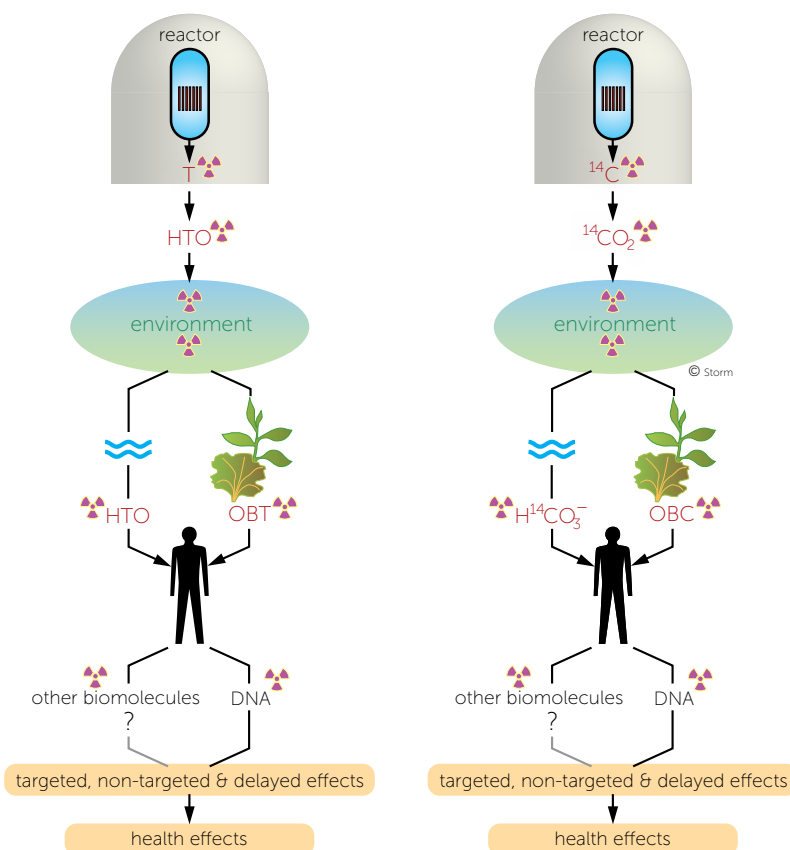


Figure 3

Pathways of radioactive hydrogen (tritium) and carbon-14 into the human metabolism. Both radionuclides are routinely released into the environment by operating nuclear power plants. The pathways are similar, OBT = organic bound tritium, OBC = organic bound carbon-14. It is generally assumed that damage to DNA molecules cause detrimental health effects. Cell damage is not limited to the cells directly hit by radiation, due to the bystander effect. It is not known if radiation damage to other biomolecules could cause detrimental health effects.

According to the classical dose-risk paradigm these discharges would have negligible public health effects, for these radionuclides do not emit gamma radiation, only weak beta rays, and for that reason unrestricted discharge of both radionuclides was (and still is) permitted. This assumption turns out to be untenable in view of the evidence of epidemiological studies and of non-targeted and delayed health effects, discussed below, and ignores the biochemical behaviour of these two nuclides.

Bio-accumulation in the food chain

The amounts of radioactive substances routinely discharged in a given year into the environment may perhaps seem relatively insignificant, however, year after year the radionuclides released can regionally build up to significant concentrations in groundwater and soil. Moreover a number of long-lived radionuclides bioaccumulate in the food chain to high concentrations, even in a medium with very low concentrations of radionuclides (e.g. seawater). An example is the bioaccumulation of technetium-99 (⁹⁹Tc) in seaweed, as proved by the graphs of Figure 4. Another example is the accumulation of cesium-137 (¹³⁷Cs) in mushrooms and wild boar. In Southern Germany the radioactive content of these foodstuffs still poses a health threat, even 25 years after the Chernobyl disaster [Rosen 2013].

Accumulation of radionuclides into the food chain greatly amplifies the health risks posed by routine or accidental discharges of radionuclides. Bioaccumulation is not addressed in detail in this study, to limit the scope.

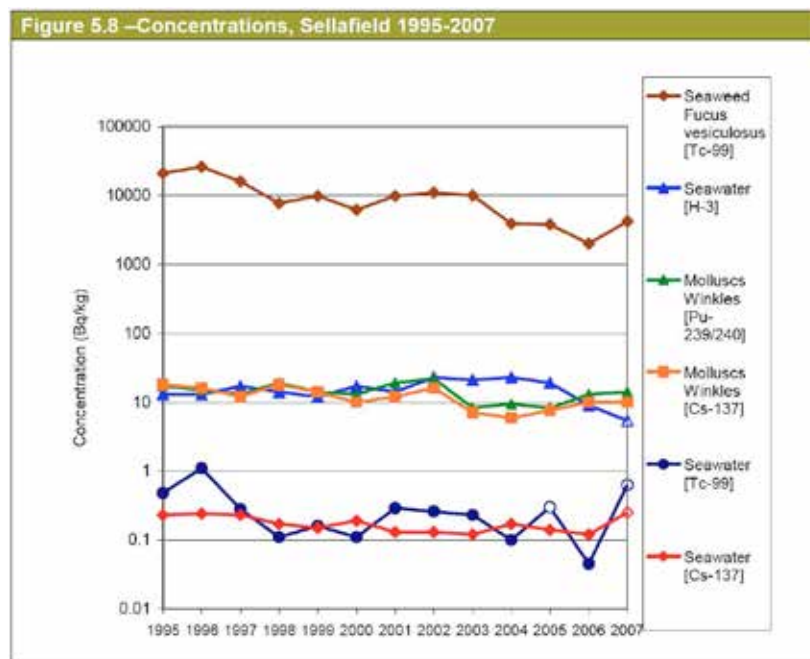


Figure 4 Concentrations of some radionuclides (H-3, Tc-99, Cs-137 and Pu-239 + Pu-240) in environmental indicator materials. Source: [DECC 2009, Crown copyright].

This graph shows two examples of the bioaccumulation effect: Technetium-99 accumulates in the examined seaweed with a factor of about 10 000 or more (seawater concentration 0.1-1 Bq/kg, seaweed roughly 5000-20 000 Bq/kg), cesium-137 accumulates in moluscs with a factor of about 100.

Deterministic effects

The effects of radiation exposure fall into two main classes: deterministic (also called non-stochastic) and stochastic effects. Deterministic effects occur at very high doses within a short period and are due to cell killing on a massive scale. The effects, often called Acute Radiation Syndrome (ARS), become evident within a time period of minutes to weeks, depending on the contracted dose. A clear relationship exists between the effects and the received dose. Deterministic effects may occur in case of exposure to nuclear explosions and to unshielded spent nuclear fuel or other highly radioactive materials, for example in case of large nuclear accidents.

The International Atomic Energy Agency [IAEA/NEA 2013] describes deterministic effects as follows:

“deterministic effect. A health effect of radiation for which generally a threshold level of dose exists above which the severity of the effect is greater for a higher dose.

Note: The level of the threshold dose is characteristic of the particular health effect but may also depend, to a limited extent, on the exposed individual. Examples of deterministic effects include erythema and acute radiation syndrome (radiation sickness).”

According to [UNSCEAR 2008]:

“... deterministic effects, where the effect is certain to occur under given conditions (e.g. individuals exposed to several grays over a short period of time will definitely suffer ARS);”

Stochastic effects

Stochastic effects (also called probabilistic effects) occur at random and involve mainly cancer and genetic effects. A common wisdom is that a larger received dose means an increased chance of cancer or other effects. The classical radiobiology assumes a linear relationship between dose and effects. However it is not certain if an individual will develop a cancer or other health effect. If a large number of individuals receive the same dose, one can predict the number of individuals who will develop a health effect, but which effect and which individual is not predictable. With regard to stochastic effects there is no threshold of the received dose below which effects could not occur, apart from zero dose.

According to [UNSCEAR 2008]:

“... stochastic effects, where the effect may or may not occur (e.g. an increase in radiation exposure may or may not induce a cancer in a particular individual but if a sufficiently large population receive a radiation exposure above a certain level, an increase in the incidence of cancer may become detectable in that population).”

Above UNSCEAR definition seems to imply that only cancers are recognized as possible health effects of radiation exposure. This confirmed by the statement:

“Cancer is the major stochastic effect of radiation exposure that has been demonstrated in human populations (inherited effects have only been observed in animal populations exposed to relatively high doses of radiation, although they are also presumed to occur in humans).”

Other publications, e.g. [IPPNW 2011], mention a number of diseases which could be induced by exposure to radiation, such as: chronic diseases (e.g. leukemia), many forms of cancers, non-cancer diseases (e.g. diabetes), but also premature senescence, hereditary disorders, congenital malformations, premature births, low birth-weight and infant mortality.

4 Attribution of health effects to radioactivity exposure

'Exposure to radiation' does not necessarily equal 'exposure to radioactivity'. Exposure to radioactivity is a much broader notion, including not only the interaction of radiation with matter, but also the biological behaviour of radionuclides in the body and the consequences of radioactive decay of all kinds of radionuclides in living cells. When attributing health effects to radioactivity the official nuclear institutes (e.g IAEA, UNSCEAR) make use of models originally based on radiation (X-ray and gamma) sources outside the body. This issue is discussed in more detail in report **mo5** *Downplaying and denial of health effects*.

Deterministic health effects

Attribution of deterministic effects to radiation exposure requires, according to [UNSCEAR 2008]:

- at least a suspicion of an exposure above a threshold, usually of a gray or more,
- observation of a specific set of clinical or laboratory findings in a particular time sequence.

Stochastic health effects

Attribution of stochastic health effects to radioactivity is not easy. Usually it is not possible to prove unambiguously the relationship between a once contracted dose of radiation and carcinogenic, mutagenic and teratogenic effects occurring many years later, because a number of factors and uncertainties are involved, such as:

- long latency periods of the observable health effects
- stochastic character of the biological effects
- many effects are not specific to exposure to radiation or radioactive substances and can be induced also by other, non-radioactive causes
- age, gender of the individual
- uncertainties of the actually received dose
- has a particular individual been exposed to low doses during a long period or higher doses during a shorter period
- which nuclides are involved
- basic biological unknowns.

In addition the kind of exposure is important: did the individual get radiation from nuclides external to the body, or internally from nuclides within the body? In which chemical form did the nuclides enter the body: by inhalation of dust and gas, or by ingestion via food and drinking water? In which chemical state did the radionuclide enter the body: as a free element, as an inorganic species or as an organically bound species? This issue is further complicated in case of chronic exposure to low doses of a number of radionuclides simultaneously.

View of UNSCEAR

In a circumstantial and poorly accessible text [UNSCEAR 2008] explains how difficult it is to attribute stochastic health effects to radiation exposure. Chronic exposure to radionuclides in food and water as a result of a large nuclear accident (i.c. Chernobyl) are not mentioned.

The report does explicitly exclude diseases other than cancer as being stochastic effects due to radiation, and does not refer to investigations of non-cancer diseases. Only thyroid cancer is mentioned as a radiation induced stochastic effect, because this type of cancer is normally very rare among children.

[UNSCEAR 2008] states:

“... the Chernobyl accident is known to have had major effects that are not related to the radiation dose. They include effects brought on by anxiety about the future and distress, and any resulting changes in diet, smoking habits, alcohol consumption and other lifestyle factors, and are essentially unrelated to any actual radiation exposure.”

The International Atomic Energy Agency (IAEA) and the World Health Organization (WHO, which cannot operate independently from the IAEA on nuclear matters, see report **m05** *Downplaying and denial of health effects*, endorse the viewpoint of UNSCEAR and seem not to recognize exposure to radioactive contamination as a possible cause of non-cancer diseases.

Falsification

Generally it not possible to attribute a detrimental health effect with a particular individual to a once contracted dose of nuclear radiation or to contamination and ingestion of radionuclides, because many observable effects can have also other, non-radioactive causes. Reversely it might be also impossible to prove that radiation or radioactivity is *not* the cause of the observed effect.

For example, suppose a given effect can have three causes A, B or C. It cannot be proved directly that A is the cause of the observed effect, as little as it can be proved that B or C is the cause. In that case it is scientifically wrong to say A cannot be the cause, so the observed effect must be caused by cause B and/ or C. Such an assertion has to be backed by an unambiguous proof that A really cannot be the cause of the observed effect. This scientific procedure is called falsification. We return to this important issue in report **m05** *Downplaying and denial of health effects*.

Due to the complexity of the dose-effect relationship the only way to obtain reliable empirical data on the health effects of radioactivity are extensive epidemiological investigations, involving large populations. Such studies should be performed by independent scientific institutions without direct or indirect financial ties with the nuclear industry.

5 Biochemical aspects of radioactivity

The relationship between irradiation of living cells and health risks is exceedingly complex. Available knowledge is based on experiments with bacteria, mice and other animals and often comprises little more than mathematical models based on theoretical assumptions. The standards for the public exposure to nuclear radiation were (and probably still are) based on the experience with diagnostic X-rays and gamma rays from external sources and originate from the early 1950s. Not included in the early models are the fact that the adverse effect of radiation is 10s to 100s of times more serious for the developing infant in the mother's womb and young children than for adults studied following medical X-ray exposures [Sternglass 2009].

Not until the early 1970s was it discovered that protracted radiation exposures from long-lived radionuclides accumulating in the body is much greater than from the same total dose received in a short X-ray exposure. A number of radionuclides have been investigated to some extent, other nuclides (among them carbon-14) have gone practically uninvestigated. The empirical database on effects in the human body seems to be very small. Synergistic effects remain basically unknown. What are the effects of several radionuclides together in a biological system?

Accumulation in specific organs

Several radionuclides have a specific biological behaviour and tend to accumulate in a specific organ or tissue. For example: technetium-99 (Tc-99) and radio-iodine (I-129 and I-131) tend to accumulate in the thyroid gland. Strontium-90 (Sr-90), ruthenium-106 (Ru-106) and plutonium isotopes tend to accumulate in bones. In such cases, the radioactivity is not evenly distributed in the body and doubling of the radioactivity of the body as a whole, means a sharp local, or organ/site specific increase in radiation. The chemical properties of an element are not affected by the radioactivity of its atoms. For example, the biochemical machinery of the human body cannot distinguish between a normal water molecule H_2O or a water molecule with one or two tritium atoms (HTO respectively T_2O). As a consequence the chemical behaviour of radionuclides in the human body is identical to that of their non-radioactive isotopes, but their biomedical properties are not.

High concentrations of a specific radionuclide in a specific organ are possible as a consequence of its biochemical properties. Radioactive iodine atoms (^{129}I and ^{131}I) for example, seek out the thyroid gland, together with its non-radioactive sister atoms, and damage the production of key growth hormones and cause thyroid cancer. Strontium-90 and plutonium tend to accumulate in the bones, where they irradiate the bone marrow, causing leukemia in newly forming red blood cells as well as damage to crucial white cells of the immune system that fight cancer cells and bacteria. Cesium-137 collects in soft tissue organs, such as the breasts and reproductive organs of females and males, leading to various types of cancer in the individuals and their children as well as in later generations [Sternglass 2009].

When a radioactive atom decays in a human body, or elsewhere, an atom of another element comes into being. This change of identity will cause a chemical reaction. The nuclear radiation from the decay will generate large numbers of secondary ions, each of which will cause also chemical reactions. Chemical bonds will be broken and new ones will be formed. Existing molecules can be destroyed and new molecules can be formed. Several factors are important in judging the biological hazards of radioactive substances in the human body, such as:

- biochemical behaviour of the radioisotope itself and of its decay products
- biochemical reactions initiated by the ionizing radiation of the radioactive decay, via primary and secondary ions
- biochemical reactions initiated by the energy transfer of the decay (recoil) and of the secondary electrons.

6 Non-targeted and delayed effects

Relatively recent studies proved the existence of 'non-targeted' and 'delayed' radiation effects. These effects had probably been observed in earlier studies but had gone unrecognised as they fell outside the then accepted paradigm of radiation effects. Non-targeted effects, which arise as a result of damage/changes to unknown areas in the cell, are termed 'non-targeted' because they mainly do not cause damage/changes to DNA or chromosomes, heretofore believed to be the main site for radiation's lesions. Non-targeted effects include, according to [Fairlie 2010a]:

- genomic instability (effects occurring up to 20-30 generations later in the progeny of an irradiated cell),
- bystander effects (effects in unirradiated cells situated close to irradiated cells),
- clastogenic effects (causing chromosome disruption or breakages in blood plasma that result in chromosome damage in non-irradiated cells), and
- heritable effects of parental irradiation that occur in succeeding generations.

The classical explanation for radiation's effects was that they were mostly caused by structural DNA damage (i.e. single and double-strand DNA breaks) which resulted in mutations in the cell's genetic information that, without repair or elimination, would end eventually in cancers. This is the target theory of radiation effects, the target being specific sequences in DNA and chromosomes.

The doses causing non-targeted effects are too low to cause structural DNA damage. The dose-response curve of these effects is often not linear, with substantial increases at very low doses followed by a levelling off at higher doses. Presently there is no mechanical explanation for how the non-targeted effects actually occur [Fairlie 2010a]. The target for radiation damage is greater than the initial tissue volume irradiated [Morgan & Sowa 2005]. A historical overview is given, among others, by [Mothersill & Seymour 2006].

The observed phenomena pose many fundamental questions to be answered and result in a paradigm shift in the understanding of radiation biology.

One of the few independent epidemiological inquiries of the relationship between nuclear power and health risks is the German Epidemiological Study on Childhood Cancer in the Vicinity of Nuclear Power Plants (*Epidemiologische Studie zu Kinderkrebs in der Umgebung von Kernkraftwerken* [KiKK 2007]. The report was published on the web in 2008. The study was commissioned by the German government and carried out by the Deutsches Kinderkrebsregister DKKR (German Childhood Cancer Register) during the years 2003-2007. The validity of the study has been accepted by the German government.

The KiKK study includes all the cases of children reported to the German Childhood Cancer Register diagnosed with cancer during 1980-2003, who were under 5 years of age at the time, and living in preassigned regions - in the vicinity of the 16 German nuclear power plants (1592 cases). Controls of equal sex and age in the year of the onset of the disease were chosen randomly for each case (4735 controls).

With regard to the incidence of cancers with children before their 5th birthday, living within a distance of 5 km from a nuclear power plant, the KiKK study concluded:

- 1.2x increase in child leukemias
- 0.6x increase in child solid cancers
- strong association with proximity to a nuclear reactor.

The KiKK study was unable to pronounce which biological risk factors could explain the results of the study. Existing models of the relationship between the incidence of cancers and low radiation doses are generally based on adults and solid cancers, not on children and blood cancers. These models cannot explain the results of the KiKK study.

Abbildung 3.6: Vergleich der geschätzten Regressionskurven im konfirmatorischen Modell (6) und Box-Tidwell-Modell
Diagnose 1980-2003, alle Erkrankungen
Auswertedatensatz, 1592 Fälle und 4735 Kontrollen

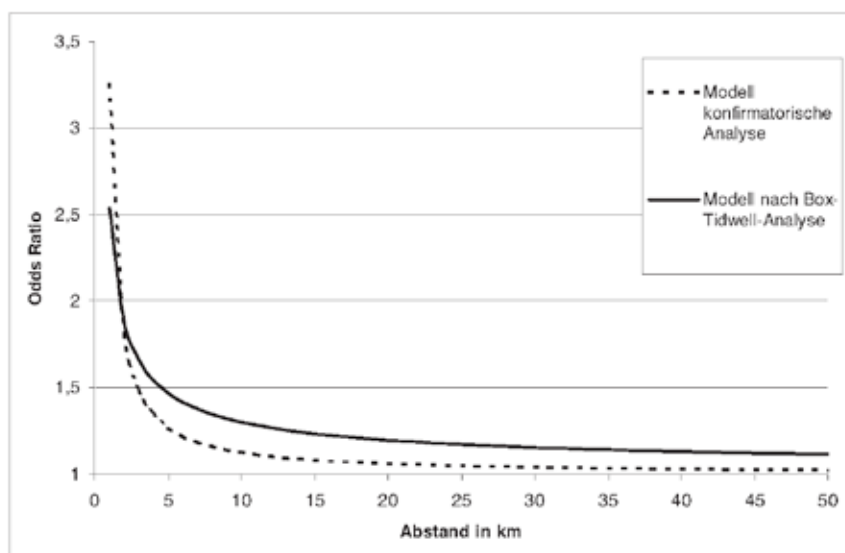


Figure 5

KiKK's regression analysis showing the statistical relationship between the incidence of cancer with children under 5 years of age and distance to the nearest nuclear power plant. The closer to a reactor, the greater the risk of childhood leukemia and solid cancers. Source: [KiKK 2007].

The increased risks as observed by the KiKK study seem to be not explainable by the radiation from nuclear power plants, as officially estimated doses from NPPs appear to be too low for such consequences, according

to the official radiological models. The radioactive releases of nuclear power plants, mainly tritium and carbon-14, exhibit spikes when the reactor is shut down and opened for removing spent fuel and exchanging it for fresh fuel during the refueling process. This may point to the difference between 'exposure to radiation' and 'exposure to radioactivity', which involves more than exposure to radiation.

To explain the results of the KiKK study the following hypothesis has been suggested [Fairlie 2010b]. The spikes in the radioactive releases may result in the labeling of the embryos and foetuses of pregnant women living nearby at high concentrations. Such high radionuclide concentrations could occur in long-lived cells and could result in large exposures to radiosensitive tissues and subsequent cancers. Some foetal tissues *in utero* may be exceedingly radiosensitive. [Fairlie 2009] observes the absence of essential knowledge with regard to radiation risks for embryos and fetuses.

This hypothesis concurs with the observation of [Rosen 2013] that the unborn child is the most sensitive form of human life: the higher tissue-metabolism and cell-division rates in a fetus increase the chance that mutations would cause malignancies before they could be stopped by the body's self-regulatory mechanisms. Additionally, as the immune system and cell-repair mechanisms of a fetus are not yet fully developed, these defensive mechanisms cannot adequately prevent the development of cancer.

According to Fairlie, vulnerable people, in particular pregnant women and women of child-bearing age, should be advised to move away from nuclear facilities as a precautionary step. Also local residents should be advised not to eat products from their gardens or wild foods, as the food pathway is the largest contributor to local doses.

The British Committee on Medical Aspects of Radiation in the Environment [COMARE¹⁴ 2011] does not endorse the findings of the KiKK study and has critical comments. In its foreword COMARE shows that it understands well the possible impact of its report:

“... and to determine whether there is any evidence to support a revision of the previous COMARE advice. However, the interest in this issue extends beyond the remit of the Department of Health and the recommendations made in this report will be pertinent to other government departments and agencies, particularly with the consideration of a new nuclear build programme.”

This sentence seems to suggest that interests other than public health are playing an important role in the conclusions and wording of the report.

In the introduction of the report COMARE states that the radiation doses arising from the operation of nuclear installations are not high enough to cause increases of childhood leukaemia and that there is growing evidence that childhood leukaemia is linked to infections.

“It is plausible that unusual infectious processes of relevance to the risk of childhood leukaemia have occurred in the vicinities of some nuclear installations, increasing the risk there. However, the biological mechanism needs to be established before a definitive conclusion on the role of infection in the aetiology of childhood leukaemia can be drawn.”

In contrast to the KiKK study COMARE found no evidence of excess leukaemia incidence at ages 0-14 years within a 25 km area around any of the NPPs in Great Britain, nor statistically significantly raised risk within 5 km. The findings of the KiKK study are consistent with results from studies in France and Finland, according to the report. In the report different kinds of leukaemias are mentioned.

COMARE has established a subgroup to specifically review the incidence of childhood leukaemia and other cancers in the vicinity of Sellafield and of Dounreay up to the present time. Sellafield and Dounray are nuclear complexes including reprocessing plants.

Based on the evidence presented in his review, COMARE sees no reason to change its previous advice to Government that there is no evidence to support the view that there is an increased risk of childhood leukaemia and other cancers in the vicinity of NPPs in Great Britain.

Evidence presented to date does not support the suggestion that discharges of tritium and carbon-14 may be responsible, in part, through in utero exposure of embryos and fetuses, according to the report. In its recommendations COMARE states:

In the course of our investigations, it became clear that carbon-14, a radioactive isotope of carbon, is a significant contributor to the radiation doses which the public receive from discharges from NPPs. This radionuclide has not been specifically implicated in health risks to date.

9 Geocap study

The results of the KiKK 2007 study are confirmed by a French epidemiological investigation: the [Geocap 2012] study, which has been published online in 2012. The Geocap study has been conducted by a team of authors from different French institutes, such as: Center for Research in Epidemiology and Population Health, Environmental Epidemiology of Cancer Team (INSERM U1018, CESP), French National Registry of Childhood Hematological Malignancies (NRCH), and Institut de Radioprotection et de Sûreté Nucléaire (IRSN). The contribution of the IRSN to this study may point to a surprising and encouraging change at IRSN, which had endeavored to discredit earlier French epidemiological studies that had shown an impact of nuclear facilities on health (www.beyondnuclear.org 12-01-2012).

The way the Geocap study reports information is somewhat complicated and not readily accessible to non-experts, graphic representations are lacking.

The outline and procedure of the Geocap study are different from the KiKK study, but the results are similar: a doubling of occurrence of childhood leukemia, between the years 2003-2007, among children under 5 years living within 5 km of nuclear power plants. The results of the Geocap study cannot be explained by estimates of doses resulting from exposure to gaseous discharges from nuclear power plants, as far as known. Overall, the findings call for investigation for potential risk factors related to the vicinity of nuclear power plants, and collaborative analyses of multisite studies conducted in various countries.

Strikingly, the nuclear world seems to ignore the KiKK and GeoCap studies. On the website of the World Nuclear Association (www.world-nuclear.org/), representing the international nuclear industry, none of these studies are mentioned.

10 Chernobyl, the IPPNW study

By coincidence one month after the nuclear disaster at the Fukushima Daiichi nuclear power plants in Japan, the report *Health effects of Chernobyl, 25 years after the reactor catastrophe* [IPPNW 2011] was published. The report is written by a team of authors from the German Affiliate of International Physicians for the Prevention of Nuclear War (IPPNW) and of the Gesellschaft für Strahlenschutz.

The study is based on large numbers of analyses, which were found comprehensive and methodically sound, not only papers that have been published in peer-reviewed journals. There are a lot of serious analyses from scientists in Russia, Belarus and Ukraine which have been published in Russian and discussed at congresses in Russian. They are almost completely ignored in the Western world.

One of the findings of the IPPNW study is:

“Essential data on the course of events of the Chernobyl catastrophe and the subsequent effects on health are not publicly available. They are classified in both East and West. This makes independent scientific analysis of the effects of Chernobyl extremely difficult. The United Nations pro-nuclear organs such as the IAEA are attempting – with the use of questionable scientific methods – to minimise the effects of the catastrophe by inaccurate use of Chernobyl data. From a scientific point of view, this is unacceptable.”

Liquidators

The number of liquidators, the people who helped to clean up the site of Chernobyl and to construct the sarcophagus to cover the exploded reactor, is estimated at 600000 - 1000000 people. It is not clear if these people worked voluntarily, under pressure, knowingly or in ignorance; they were exposed to high, but unknown levels of radioactivity. As early as 1992 some 13000 liquidators had died and 70000 had become invalids, according to a source in Minsk. On the basis of a number of studies the death toll amongst the liquidators in 2005 is estimated at 112000 - 125000 people. Other sources estimate that 50000 - 100000 liquidators have died. In 2005 94% of the surviving liquidators were ill and had become an invalid, according to the Ukrainian embassy in Paris.

Population

Available studies estimated the number of fatalities among infants to be about 5000. According to the IPPNW study between 12000 and 83000 children were born with congenital deformations in the region of Chernobyl, and around 30000-207000 genetically damaged children worldwide. Only 10% of the expected damage can be seen in the first generation.

Hundreds of thousands of people, particularly the evacuees from the 30 km zone, having lost their home and job, ended up in a situation of serious societal disruption. In some areas in Belarus and Ukraine nearly all the inhabitants are suffering from one or more radiation-induced diseases.

The IPPNW study cites many other observed consequences of the Chernobyl disaster. One of the conclusions of the study reads:

“By 2050 thousands more cases of illnesses will be diagnosed that will have been caused by the Chernobyl nuclear catastrophe. The delay between cause and noticeable physical reaction is insidious. Chernobyl is far from over.”

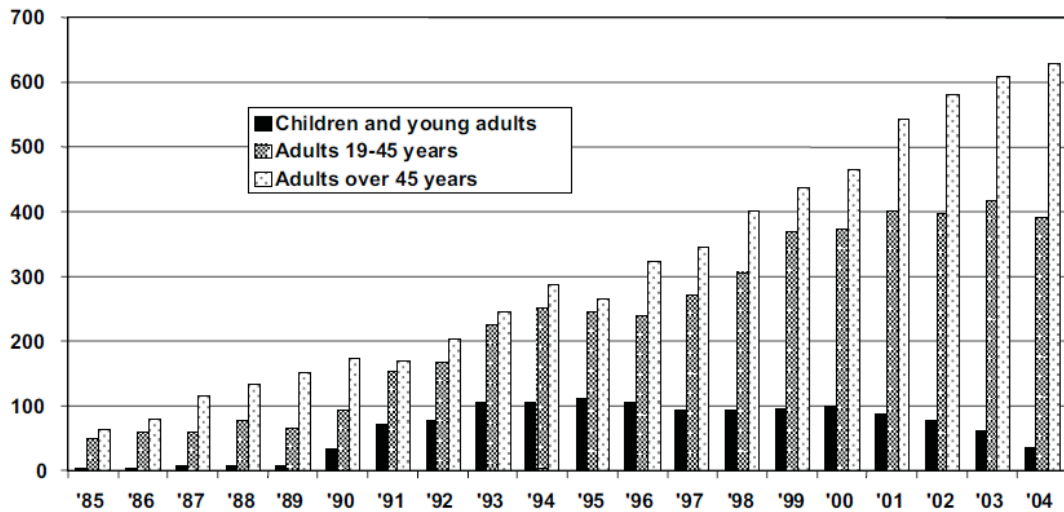


Figure 6
Incidence of thyroid cancer in Belarus 1985-2004. Source: [IPPNW 2011].

Radiation-induced diseases

In the regions contaminated with radioactive materials after the Chernobyl disaster a greatly increased incidence of a many different malignant and non-malignant diseases and disorders are observed, such as:

- multimorbidity classified as radiation-induced premature senescence
- cancers and leukaemia
- thyroid cancer and other thyroid diseases
- damage to nervous system, mental disorders
- heart and circulatory diseases
- infant mortality
- congenital malformations
- endocrinal and metabolic illnesses
- diabetes
- miscarriages and pregnancy terminations
- genetic damage, hereditary disorders and diseases
- teratogenic damage, such as:
anencephaly, open spine, cleft lip/palette, polydactylia, muscular atrophy of limbs, Down's syndrome.

11 Reliance on models

The chapters above addressed some reports on the health effects of radioactivity. Widely different views prove to exist, not only regarding the health hazards of normal operation of nuclear installations, and limited nuclear accidents, but even more so regarding the effects of large-scale exposure to radioactivity caused by nuclear disasters, such as Chernobyl and Fukushima.

The reasons for the controversies turn out to be based on principally divergent approaches to this complex matter: the use of models versus empirical evidence. Besides this, the economic and financial interests of the nuclear industry are playing an important role.

The dominant role of radiological models in the nuclear world with regard to health effects of contamination with radioactivity is the form of reporting the consequences of the Chernobyl disaster used by the World Health Organization WHO and the International Atomic Energy Agency IAEA in, for example, [WHO 2005] and [IAEA 2008]. This matter is addressed in report **m05** *Downplaying and denial of health effects*.

12 Radiation dose measurement

The chance of developing a cancer as a result of exposure to nuclear radiation is assumed to increase linearly with the contracted dose of radiation, according to the Linear Non Threshold (LNT) model. The dose is defined as the amount of energy from the nuclear radiation absorbed per kilogram body mass. Because one type of radiation inflicts more biological damage than another the biologically effective equivalent dose has been defined as absorbed dose multiplied by the *radiation weighting factor*, also called the *quality factor*. This factor gives the Relative Biological Effectiveness (RBE) of the various kinds of ionizing radiation. The unit of the biological effective radiation dose is the *sievert*, symbol Sv; usually the unit millisievert (mSv) is used because the sievert is a large unit.

Because of the simple relationship between externally measured gamma-activity and dose or dose rate, the sievert continues to be the model generally used by regulatory agencies as base for human radiation exposure.

The sievert is not a measurable unit in itself but is composed of the activity of a given amount of matter measured in becquerel per second (Bq/s) multiplied by the weighting factor, which depends on the kind of radiation, as pointed out above. In principle the activity (in Bq/kg) a measurable quantity, although a number of important radionuclides are not detectable by common radiation counters. The value of the weighting factor is based on models and arbitrary assumptions and therefore is not unambiguous. Consequently the sievert is an ambiguous unit, and its use may lead to wrong conclusions with regard to health hazards.

Lack of clarity remains:

- To what extent are doses cumulative, for example does a once-only dose of 1 Sv during 1 hour equal 1000 hours of exposure to a dose rate of 1 mSv/h?
- What is known about chronic exposure to 'low' doses?
- How is a 'low' dose defined? Is it an invariable quantity?
- Are the different biochemical properties of the dozens of types of radionuclides released into the environment by the nuclear power system accounted for in the models?
- How do the models handle exposure to a number of different radionuclides simultaneously, for example after a nuclear disaster like Chernobyl and Fukushima?
- Which radiation-induced diseases are included in the models used to define the weighting factors and the safety standards? Are only solid cancers accounted for, or also other, non-cancerous diseases?
- What is known about bioaccumulation of radionuclides in the food chain? How is this phenomenon incorporated into the models?
- How can aerial surveys of easily detectable radionuclides as Cs-137 over contaminated areas, presented in average dose rates (mSv/h), be translated into health hazards for individuals living in that area?
- The models seem to be based only on the physical interaction of radiation with matter. Are biochemical mechanisms, involving biologically active and hardly detectable radionuclides like tritium and carbon-14, included in the models?
- On what assumptions are the models based? Are these assumptions continually verified and adjusted on the basis of empirical evidence coming available year by year?
- What was the original purpose of the models? To estimate the acute radiological risks for military personnel in (nuclear) wartime, or to estimate the health risks for the public posed by chronic exposure to radionuclides produced and released by civilian nuclear power?
- For what reason does the nuclear industry exclude extensive epidemiological studies of the health effects of exposure to radiation and to radionuclides inside the body?
- Why not start from empirical evidence?

13 Radiation hormesis

Radiation hormesis, also called radiation homeostasis, is the hypothesis that low doses of radiation – within the region and just above natural background levels – would be beneficial, stimulating the activation of repair mechanisms that protect against disease, that are not activated in absence of radiation. The reserve repair mechanisms are hypothesized to be sufficiently effective when stimulated as to not only cancel the detrimental effects of ionizing radiation but also inhibit disease not related to radiation exposure (wikipedia 2012a).

The radiation hormesis hypothesis seems to be based on limited model studies and on analogy with chemical hormesis. Chemical hormesis is the phenomenon that a chemical species is assumed to be non-toxic in very low doses, or even beneficial, and toxic in higher doses. In fact this view seems to be based on the ideas of Paracelsus (1493-1541), summarized in his statement: *dosis facit venenum* ('the dose makes the venom'). The analogy with the supposed chemical hormesis is highly questionable, because of the completely different biological mechanisms involved in the effects of chemical species and of radioactivity in the human body.

Perhaps more important is the evidence that a number of substances exhibit the reverse effect: at very low doses they have significant and often unexpected detrimental effects, much higher than expectation based on a linear dose-effect relationship [Fagin 2012].

The studies of radiation hormesis seem to focus on the incidence of solid cancers, other radiation-induced (non-cancerous) diseases are not included. Apparently only external exposure to radiation is included in the models. Epidemiological evidence of the hormetic effect of low radiation doses is absent [Wikipedia 2012a]. On the contrary, the Kikk and Geocap studies proved that very low doses of radioactivity have significant detrimental health effects and that these effects cannot be explained by the usual radiological models.

Comparison of 'low' doses with background radiation, as is done in numerous publications, involves a caveat. How is the 'background radiation' defined? Only gamma radiation from easily detectable radionuclides? What about the doses from background radiation in areas where radionuclides are constantly being released into the environment, for instance in the vicinity of nuclear power plants, or in areas being contaminated by large releases of radionuclides elsewhere on the world after a large-scale accident?

Consensus reports by the United States National Research Council and the National Council on Radiation Protection and Measurements and the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) argue that there is no evidence for hormesis in humans and in the case of the National Research Council, that hormesis is outright rejected as a possibility [Wikipedia 2012a].

References

- CERRIE 2004
Report of the Committee Examining the Radiation Risks of Internal Emitters,
Health Protection Agency, October 2004,
file: cerrie_report_e-book.pdf
www.cerrie.org
- COMARE14 2011
Committee on Medical Aspects of Radiation in the Environment (COMARE),
Chairman Prof A Elliott,
Fourteenth Report,
Produced by the Health Protection Agency, Crown Copyright 2011.
<COMARE14report.pdf>
www.comare.org.uk/press_releases/documents/
retrieved 22 November 2013
- DECC 2009
UK Strategy for Radioactive Discharges,
Department of Energy & Climate Change, July 2009,
Crown copyright 2009.
<0084424.pdf>
<http://www.scotland.gov.uk/Resource/Doc/280203/>
http://decc.gov.uk/en/content/cms/what_we_do/uk_supply/energy_mix/nuclear/issues/radioactivity/radioactivity.asp
- Fagin 2012
Fagin D,
The learning curve,
Nature, vol. 490, pp 462-465, 25 October 2012.
- Fairlie 2009
Fairlie I,
Childhood cancers near German nuclear power stations: hypothesis to explain the cancer increases,
Medicine, Conflict and Survival, 25:3, pp206-220,
online publication July 2009,
<http://dx.doi.org/10.1080/13623690902943396>
- Fairlie 2010a
Fairlie I,
Commentary on UNSCEAR 2006 Report: Annex C – New effects of radiation,
Letter,
Radiat Prot Dosimetry, 2010 Febr; 138 (2): 190-3
- Fairlie 2010b
Fairlie I.
Hypothesis to Explain Childhood Cancer near Nuclear Power Plants
Int J Occup Environ Health 16:341–350 (2010)
- Geocap study 2012
Sermage-Faure C, Laurier D, Goujon-Bellec S, Chartier M, Guyot-Goubin A, Rudant J, Hémon D & Clavel J,
Childhood leukemia around French nuclear power plants – The Geocap study, 2002-2007,
International Journal of Cancer, doi: 10.1002/ijc.27425, February 2012,
<http://onlinelibrary.wiley.com/doi/10.1002/ijc.27425/pdf>
download 5 March 2012.
- IAEA 2008
Chernobyl: looking back to go forward,
Proceedings of an international conference on Chernobyl:
Looking back to go forward,
Organized by the International Atomic Energy Agency on behalf of the Chernobyl Forum and held in Vienna, 6-7 September 2005,
- IAEA, Vienna, 2008,
<Pub1312_web.pdf>
<http://www-pub.iaea.org>
- IAEA/NEA 2013
INES, The International Nuclear and Radiological Event Scale, Users Manual,
2008 edition, emended edition March 2013,
IAEA-INES-2009,
IAEA, Vienna, March 2013.
<INES2009_web.pdf>
<http://gnssn.iaea.org/regnet/Pages/INES.aspx>
<http://www-pub.iaea.org>
- IPPNW 2011
Pflugbell S, Paulitz H, Claussen A & Schmitz-Feuerhake I,
Health effects of Chernobyl. 25 years after the reactor catastrophe,
German Affiliate of International Physicians for the Prevention of Nuclear War (IPPNW) and Gesellschaft für Strahlenschutz (GFS), report, April 2011
http://www.medact.org/content/nuclear/IPPNW%20Germany%20chernob_report_2011_en_web.pdf
- KiKK 2007
Kaatsch P, Spix C, Schmiedel S, Schulze-Rath R, Mergenthaler A & Blettner M,
Epidemiologische Studie zu Kinderkrebs in der Umgebung von Kernkraftwerken (KiKK-Studie),
Vorhaben StSch 4334 (in German),
Im Auftrag des Bundesministeriums für Umwelt, Naturschutz und Reaktorsicherheit und des Bundesamtes für Strahlenschutz, Germany, 2007,
4334_KiKK_Gesamt_T.pdf
www.bfs.de/de/bfs/druck/Ufoplan/
- Morgan & Sowa 2005
Morgan WF & Sowa MB,
Effects of ionizing radiation in nonirradiated cells,
PNAS, October 2005, vol.102 no 40, pp 14127-14128.
www.pnas.org/cgi/doi/10.1073/pnas.0507119102
- Mothersill & Seymour 2006
Mothersill C & Seymour CB,
Radiation-induced bystander effects and the DNA paradigm: An “out of field” perspective,
Mutation Research 597 (2006) 5-10.
www.sciencedirect.com
- Rosen 2013
Rosen A,
Critical Analysis of the WHO's health risk assessment of the Fukushima nuclear catastrophe,
German Section of the International Physicians for the Prevention of Nuclear War (IPPNW Germany), March 1, 2013.
<WHO_Fukushima_Report2013_Criticism_en.pdf>
www.fukushima-disaster.de/information-in-english/
retrieved 12 October 2013
- Sternglass 2009
Sternglass EJ,
letter to Dr Steven Chu, Secretary of Energy, Febr 7, 2009,
www.radiation.org/reading/090423_ejs_to_doe.html
- UNSCEAR 2008
UNSCEAR 2008 Report. Sources and effects of ionizing radiation.
ANNEX D. Health effects due to radiation from the Chernobyl accident,
United Nations, April 2011.

<11-80076_Report_2008_Annex_D.pdf>
www.unscear.org/docs/reports/11-80076_Report_2008_Annex_D.pdf
retrieved 11 Sept 2013

WHO 2005
*Chernobyl: the true scale of the accident,
20 Years Later a UN Report Provides Definitive Answers and
Ways to Repair Lives,*
<http://www.who.int/mediacentre/news/releases/2005/pr38/en/index.html>
and
<http://www.who.int/mediacentre/news/releases/2005/pr38/en/index1.html>

wikipedia 2012a
Radiation Hormesis,
http://en.wikipedia.org/wiki/Radiation_hormesis
August 29, 2012, retrieved October 2012.