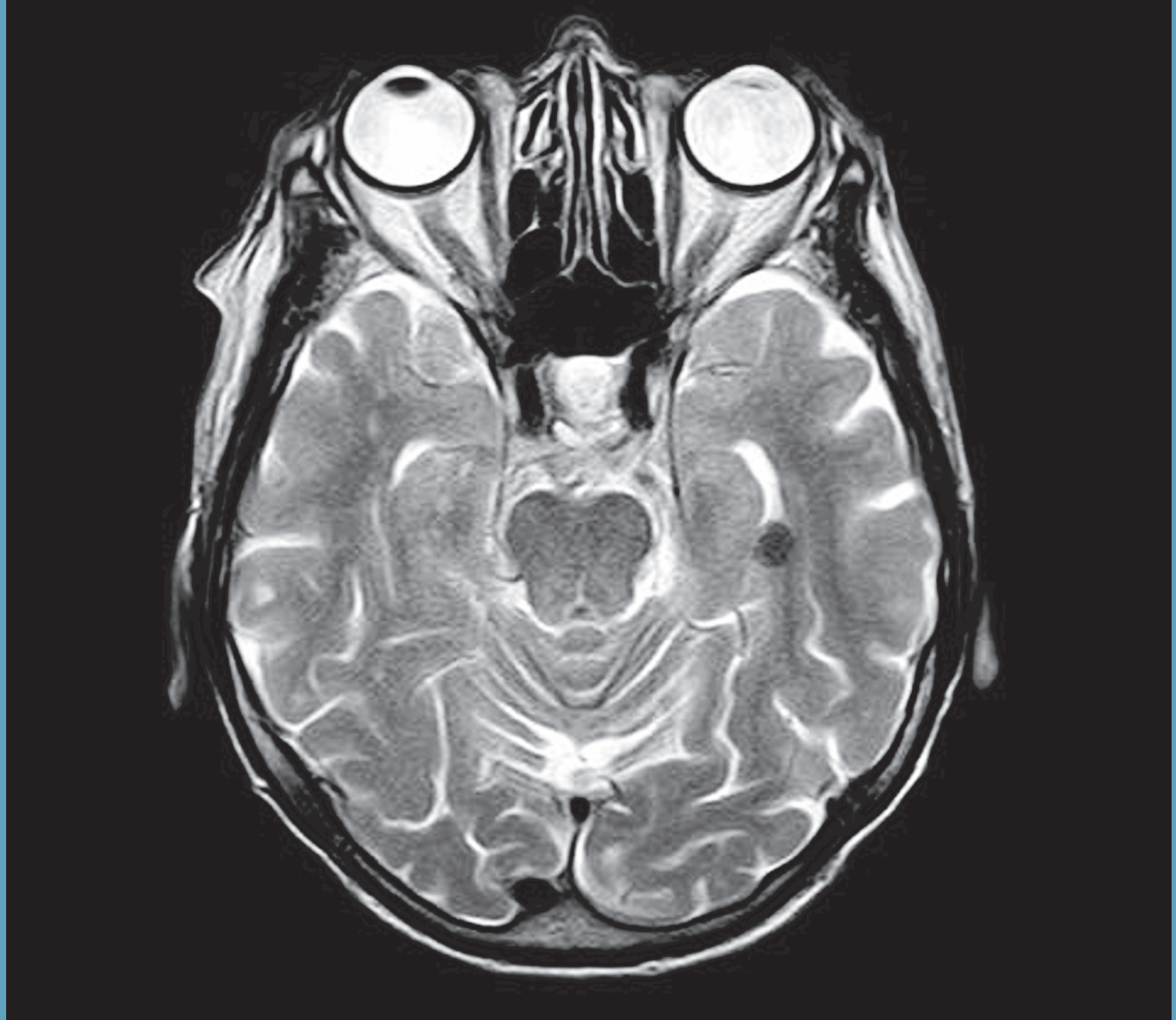
Health effects of ionising radiation and their consideration in RADIATION PROTECTION



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Health effects of ionising radiation and their consideration in radiation protection

Imprint

Authors

Gabriele Mraz, Austrian Institute of Ecology

Oda Becker, Independent Expert for the Risks of Nuclear Facilities, Hannover/Germany

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Foreword

Radiation and radioactive substances surround us and are part of our body as well. But to believe that they have no influence on our health or that we are already used to a certain level of radiation is utterly wrong in the same respect as it would be wrong to believe that smoking in a long range makes you less susceptible to harmful effects of smoking. Even though smoking one cigarette does not immediately kill, in a sufficiently large collective a very small rise in lung cancer could be observed even for this case. Radioactive substances emit radiation which is harmful for the human body, no matter how low the dose may be. Even though a lower limit for health effects does not exist, the effect might become invisible in comparison with other adverse effects. Nevertheless, some cases of illness and death have to be attributed to radiation effects statistically, no matter how low the collective dose is. Consequently, the dose received should be as low as reasonably feasible.

The average natural – and by that practically irreducible – background could represent a reasonable lower limit. Concerning the additional dose caused by human activities, the present paper shall provide a basis for the discussion. Nevertheless, the debate cannot be based on statistical reasoning only. Which dose is accepted by society as a whole or for its individuals in certain situations needs a broad discussion on a sound and evidence based ground. Even though it is correct that the outcome will be a dose limit which results in an acceptable elevated probability for some diseases, it is also a dose limit which results in the early death of a certain number of individuals of the society. For these the question cannot be taken too serious and the latest scientific results should be implemented to revise those limits regularly.

I do hope that the results provided in this paper will help to determine limits which are significantly lower than the limits in use today and which are at the same time not preventive to the many useful applications of radioactivity in medicine and research.

Andrea Schnattinger, Ph.D.

Head of the Ombuds-Office for Environmental Protection

Content

Extended Summary

What happened after the nuclear accident of Chernobyl in 1986 seems to happen all over again after the accident of Fukushima in 2011. After Chernobyl it took about a decade until organisations like the International Atomic Energy Agency (IAEA) and the World Health Organisation (WHO) admitted that thyroid cancer caused by radioactive contamination increased in children and adolescents, even though the increase was quite obvious from 1990 onwards. Now that we are in the sixth year after Fukushima, the same authorities together with Japanese authorities downplay the already visible increases in thyroid cancer in the contaminated regions. And it is not only thyroid cancer that shows an increase after these two accidents. Also the incidence of other types of cancer and a lot of other diseases increase in populations affected from the Chernobyl accident, including diseases in the descendants of contaminated people.

While it has already been proven that radiation can cause negative health impacts like thyroid cancer and leukaemia, it is disputed if radiation can also be responsible for other health effects like heart diseases. And it is disputed if low or even very low doses of ionising radiation can cause measurable effects at all.

The effects of high radiation doses on humans (like acute radiation sickness) are documented quite well. But **the effects of low doses are still one of the most disputed topics in radiation protection.** Low doses result from nuclear installations during normal operation, from accident situations in nuclear facilities for workers and the public, from the nuclear bombs on Hiroshima and Nagasaki, but also from medical exposure and natural background.

The health effects of low dose radiation are discussed highly controversially as they are not easy to detect due to lack of detailed data, unreliable medical systems and the very large number of people affected. Furthermore, diseases like cancer cannot be attributed to a single cause.

Looking into recent European legal texts, several questions arise: What are dose limits and levels based upon? What models and epidemiological results have been used to determine these dose limits? Which experts are allowed to give input to the underlying scientific discussions, and whose work is neglected and why?

New insights in health effects of ionising radiation

Radiation protection has long been based mainly on the research of the survivors of the atomic bombs on Japan. The new INWORKS study on a big collective of nuclear workers (Richardson et al. 2015a) confirmed that **low, protracting doses result in risks that are comparable to risks of higher doses**.

Especially the **chronic lymphoblastic leukaemia (CLL)** was long believed to not be radiation induced, but now the results of a new study on Ukrainian Chernobyl liquidators prove that there is evidence for the contrary. (Zablotska et al. 2013)

In August 2016 it became known that two **Fukushima workers** who had developed **leukaemia** after receiving low dose of 16 mSv and 54.4. mSv, respectively, were entitled to workers compensation.

Thyroid cancer incidence after Chernobyl showed no decrease or is even still increasing in several groups of Ukrainian people. (Prysyazhnyuk et al. 2014, Brenner et al. 2011) In his update of the TORCH report, Ian Fairlie (2016) also showed a long latency period for thyroid cancer. A first study about **thyroid cancer after Fukushima** supported the results from Chernobyl studies. (Tsuda et al. 2016) In 2016, the first worker of the Japanese nuclear enterprise TEPCO with thyroid cancer has been

acknowledged to have gotten the disease due to his work in NPP Fukushima. The man will receive compensation.

New studies show that **breast cancer** is not only caused by radioactive contamination but can even occur at low doses such as doses caused by effects of normal operation or well below 100 mSv like in the study of Pukkala et al. (2006). Breast cancer could also be caused by normal operation of NPPs. (Busby 2009)

Non-cancer diseases comprise a big group of diseases, among them cardiovascular diseases, diseases of the respiratory and the gastrointestinal tract, diabetes, cataracts etc. While the International Commission on Radiological Protection (ICRP) does not assume effects under a dose of 500 mSv, studies show that even at low dose an excess risk can be found (Buzunov et al. 1996, Ivanov 1996, Little et al. 2012) – which is of special interest, because f.e. cardiovascular diseases have a high prevalence and therefore many people can be concerned. **Cataracts** were long seen as deterministic radiation effect (occurring only over a certain threshold), but a new study suggest that they are also stochastic effects without a threshold. (Mämpel et al. 2015)

In several studies an increase in **leukaemia risk for children** who have been exposed in utero or in young years was found (Davies at al. 2006, Noshechenko et al. 2010, Busby 2009)

Normal operation of NPPs can also lead to health effects like childhood **leukaemia**, especially in children living in the vicinity. This is shown by studies from Germany, UK, France and Switzerland (Kaatsch et al. 2007, Bithell et al. 2008, COMARE 2011, Spycher et al. 2011). A recent published study reveals a highly statistically significant 37% increase in childhood leukaemia within 5 km of almost all NPPs in the UK, Germany, France and Switzerland. (Körblein and Fairlie 2012)

Furthermore, recent studies concerning **childhood cancer from natural background radiation** (Spycher et al. 2015, Kendall et al. 2013) and medical exposure indicate the high radio-sensitivity of children.

The ICRP assumes that the life-time cancer-risk following in utero-exposure is about three times higher than the risk of the overall population – but in the light of the depicted studies this assumption seems to be insufficient.

After exposure from ionising radiation (e.g. subsequent to nuclear accidents) **teratogenic effects** have been observed, even in those who were only exposed to low or very low levels of radiation. (Busby et al. 2009; Körblein and Küchenhoff 1997; Körblein 2003, 2004b) Exposure in-utero cannot only cause leukaemia and cancer, but also perinatal mortality, congenital effects etc.

The ICRP judges that, following **prenatal** (in-utero) exposure, a) cancer risk will be similar to that following irradiation in early childhood and b) a threshold dose (100 mSv) exists for the induction of malformations. In the light of recent scientific research this position has to be revised. (Körblein 2011)

Exposure of the germ cells (gonads) can cause mutations in the genetic material which may result in **heritable diseases** in the offspring of the exposed persons. According to ICRP, radiation-induced heritable disease has not been demonstrated in human populations but there is substantial evidence from animal studies of heritable damage to germ cells (ova and spermatozoa) as well as their precursor cells. However, the ICRP decreased its risk estimate for heritable damage between its recommendations of 1991 and the recent ones of 2007 (ICRP 1991, 2007)

Effects in populations exposed to Chernobyl fallout are excluded by the official committees (in particular ICRP), which claim that doses are too low to generate statistically observable increases. This, however, is certainly wrong, because it is known from many studies of chromosome aberrations (e.g. Busby 2015b), either that the doses calculated by the United Nations Scientific Committee on the

Effects of Atomic Radiation (UNSCEAR) are much too low or that there is an enhanced radiobiological effectiveness in the type of internal exposures or chronic delivery received by the Chernobyl groups.

Scientific uncertainty exists about the differences in tissue effects and therefore the risks from external versus internal radiation sources (NAS 2014).

When examining the risk of genetic damage by radiation it is very important to make a distinction between acute exposure to radiation and chronic exposition. Chronic radiation exposure results in permanent radiation of all stages of spermatogenesis. This explains the relatively high number of malformations and other congenital defects of the descendants of occupationally exposed men.

Schmitz-Feuerhake, Busby and Pflugbeil have published very recently a paper in which they bring up arguments for a new assessment. (Schmitz-Feuerhake et al. 2016) The authors criticize UNSCEAR and ICRP for their very low risk factors for hereditary diseases in humans based on reportedly absent genetic effects in the acute exposed Japanese atomic bomb survivors. Nearly all types of hereditary defects were found in cases affected by very low doses. The authors suggest that the results show that current radiation risk models fail to explain or even predict the many observations and should be abandoned.

All the congenital malformations effects are caused by mutation of DNA whether in the parental germ cells and precursors or from implantation to birth. Genetic effects in contaminated areas cannot be clearly distinguished from those resulting from in-utero exposure of embryos and foetuses.

In that light, the behaviour of the international associations (IRCP, WHO) is irresponsible, because at present it is already clear that the radiation risk for future generations will be much higher than assumed according to the existing risk factors, even though the full extent cannot yet be predicted.

Although there are numerous studies in the area of assessment of impacts of nuclear power plants on human health, it is still necessary to make follow-ups, especially to investigate radiation effects of normal operation of nuclear facilities in depth. Particularly in countries with many NPPs in operation and with NPPs situated in densely inhabited areas, **it is necessary to try to arrange for independent studies or independent reviews of existing studies.**

It is of uttermost importance that new insights in radiation effects will be considered in radiation protection law and measures.

European radiation protection legislation – the BSS-Directive

Council Directive 2013/59/Euratom of 5 December 2013 laying down basic safety standards for protection against the dangers arising from exposure to ionising radiation, the so-called BSS-Directive, establishes uniform basic safety standards in the EU. It applies to any planned, existing or emergency exposure situation with ionising radiation, caused by artificial or natural sources of radiation.

Based on new insights in health effects it can be concluded that **the dose limits in the BSS-Directive are too high**, they do not provide enough protection, especially for the embryo/foetus, children, pregnant women and young adults.

For the underlying dose calculations, it is important to shift the scientific focus from only studying the atomic bomb survivors to all other studies of consequences of Chernobyl, effects of natural background and of very low and low doses especially from normal operation of nuclear facilities. Recent studies show that using a dose and dose-rate effectiveness factor (DDREF) of two by ICRP is highly underestimating the measured effects. The **DDREF has to be reduced from 2 to 1**, which is now

recommended by the WHO and the German Commission on Radiological Protection (WHO 2013, p.32, SSK 2014).

Genetic and teratogenic effects are seriously underestimated, even though there is scientific evidence of effects like genetically induced malformations, cancers, and numerous other health effects in the children of father and/or mothers who were exposed to low doses of ionising radiation. The protection measures for **pregnant workers** have to be strengthened.

The assumptions of ICRP about the relative biological effectiveness of **neutrons** is also in question. A new approach from Walsh (2012) shows that a weighting of 10 according to ICRP 103 may not be optimal, and this practice should be reviewed.

Dose limits for single organs should be introduced, especially for the gonads and the thyroid.

In case of an emergency, countries have defined their dose levels for start of **emergency protection measures** like iodine tablets or evacuation. These intervention levels are based on the BSS-standards and therefore on recommendations of the ICRP. In Austria, a country without NPPs, some of the intervention levels are lower than in other countries, f.e. staying indoor for children and pregnant women is recommended if an effective dose of 1 mSv/7days is expected. The administration of iodine tablet for children should start if a thyroid dose of 10 mSv is expected. (IntV 2007) This can be considered as better practice. **Protecting people's health has to be the priority under any circumstances, in particular of the descendants.**

Because it has been proven that also very low doses can cause measurable health effects, it is recommended that besides the effective individual dose and single organ doses also the **collective dose** should be used in the BSS-Directive, levels for the collective dose should be determined especially in planned radiation situations.

It may not be possible to make amendments of the BSS-Directive itself (or even the underlying approach of ICPR), but the members states still have time until Feb 2018 to **implement the BSS-Directive into national law**. By doing so, member states could introduce dose limits that are below the maximum dose limits. Many countries have not implemented the BSS-Directive yet, so there is still time left for the interested public to enter the debate.

Medical diagnostics are valuable tools for human health, but can also cause measurable negative effects due to radiation. It contributes in Europe with approximately 1 mSv to the annual average dose, the largest part of it is received by X-ray diagnostics and computer tomography. Therefore, a reasonable reduction of the use of these diagnostic tools can be recommended.

ICRP and the Article-31-Group of Experts are the only expert groups who can at the time-being influence radiation protection legislation. The ICRP has no democratic legitimation. The Article-31-Group is staffed by the member states, but its consulting has often not been made public. **It would be preferable to have independently staffed expert groups with public participation, and whose work is made transparent.**

Permitted food contamination in case of another Super-GAU: the Food Level Regulation

After the accident of Chernobyl in 1986 large amounts of food and feed were contaminated by radioactive material. Not only Belarus, Ukraine and Russia were affected, but also many countries in Europe inside and outside the EC (European Communities at that time). The EC wanted to make sure that only such agricultural products were put on the EC-market that did not exceed a defined level of contamination. Therefore, three regulations for maximum levels in food and feed were established:

These regulations allowed the European Commission to quickly adopt an implementing regulation in case of a radioactive contamination – for the first time such an implementing regulation was applied in 2011 after the nuclear accident in Fukushima. After long years of amending these regulations, in February 2016 a new regulation has entered into force: Council Regulation Euratom 2016/52 for "laying down maximum permitted levels of radioactive contamination of food and feed following a nuclear accident or any other case of radiological emergency" (food level regulation).

But when analysing the underlying assumptions that have led to the food levels, errors and neglected facts become obvious. The maximum permitted food levels in Council Regulation Euratom 2016/52 are too high and should be reduced due to the following arguments:

For dose calculations in the food level regulation an assumption is used that only 10% of all food is contaminated up to the maximum and 1% of liquid food, respectively. This will not be true in a worst case of severe nuclear accident in one of the EU member states and under unfavourable meteorological conditions.

It is assumed that an effective ingestion dose of 1 mSv will not be exceeded it the food levels are not exceeded. But when the assessment of the Art.-31-Group of Experts in Publication 105 (EC 1998) is recalculated, an effective ingestion dose level of 1 mSv will be exceeded for infants and adults using the assumption that in one year only food is consumed of which 10% (1% for liquids) is contaminated up to the maximum permitted level. This recalculation results in 3.1-7.8 mSv instead of 1 mSv.

The underlying data on dietary habits and food consumption are outdated by more than 25 years. Moreover, for only 10 EU member states out of 28, food data have been researched and used in calculations. Dietary habits have changed in the meantime, this can lead to much higher ingestion dose than assumed in the food level regulation.

The Art.-31-Group recommends in its Publication 105 that member states should establish regularly the typical dietary habits for different regions so that in the case of an accident no underestimations of actual consumptions rate occur. This recommendation is very important. The interested public should ensure that member states have their updated dietary data prepared so that on the occasion of implementing a food level regulation they can derogate from the food levels and introduce food levels that are best for ensuring their people's health.

1 Introduction

Ionising radiation affects human health. But while effects of high-level radiation are well documented, health effects of low-level radiation are one of the most disputed topics in medical science. Low-level radiation results from nuclear installations during normal operation and accident situations for workers and the public. Also the contamination from nuclear bombs (Hiroshima and Nagasaki) and atmospheric nuclear weapons testing[1] can still be measured – large parts of the northern hemisphere are contaminated with radionuclides like Caesium-137, Strontium-90 and Plutonium.

Why are health effects of low-level radiation so highly controversial and why is critical epidemiological research often not acknowledged properly in the nuclear community? Among other things, this is due to methods. Especially after Chernobyl when very large population groups were contaminated it was difficult to get valid and complete data, partly due to lack of monitoring systems, unreliable medical systems and political unwillingness. Besides lacking data, health effects of low-level radiation are not easy to detect. Diseases are normally caused by a variety of agents (like environmental toxins, smoking or bad lifestyle), and it is not easy to prove the cause of illness – especially when the investigated population group is small or data are incomplete.

In the last years, radiation effects on workers in nuclear facilities have been studied extensively. Here the data base is better. A big study (INWORKS) has been conducted recently proving adverse health effects of low-level radiation that can no longer be ignored. Also studies on the effects of natural radiation help to bring clarity into the debate.

Normal operation of NPPs can also lead to health effects, especially in children living in the vicinity. This is shown not only by the so-called KiKK-study (Germany) but also by studies from other countries (France, Great Britain and Switzerland).

The **basic safety standard** for radiation protection in the EU is the new **Directive 2013/59/Euratom (BSS-Directive)**. In this Directive, dose limits for workers, members of the public, patients and the environment are given for different exposure situations.

Also **Council Regulation (Euratom) 2016/52** on maximum permitted levels of radioactive contamination of food and feed following a nuclear accident is based on radiation protection dose limits.

Looking into these regulations, several questions arise: What are these dose limits and levels based upon? What models and epidemiological results have been used to determine these dose limits? Which experts are allowed to give input to the underlying scientific discussions, and whose work is neglected and why?

This study addresses the links between knowledge of health effects and their reflection in Directive 2013/59/Euratom and Council Regulation (Euratom) 2016/52 which are of high relevance for radiation protection.

Chapter 2 gives a short overview of concepts and methods for determination of dose and risk. For this study we researched new insights in radiation health effects in scientific journals, publications from relevant radiation protection organizations, and conference proceedings. An overview of the results is presented in chapter 3. In chapter 4 it will be determined what health consequences are (not) taken

[1] Over 2000 nuclear tests were carried out between 1945 and 1996 (https://www.ctbto.org/)

into account in the above mentioned EU legislation, and what consequences could arise. Conclusions and recommendations are given.

2 Dose and risk – overview of concepts and methods

Ionising radiation has many negative effects on human health. Nevertheless, humans cannot avoid radiation altogether, as it results not only from artificial sources like nuclear facilities but also from natural sources. To reduce these impacts, radiation protection as a scientific discipline is important – and like every science it has policy impacts, f.e. on radiation protection legislation.

Two especially important concepts in radiation protection are the **concept of dose** and the **concept of risk.**

If radiation hits a human body, energy is absorbed. To quantify this effect, the **absorbed dose** is used with its unit Gray (Gy). 1 Gy equals the absorption of 1 Joule per kg. The type of radiation that is absorbed influences the effect in the human body. Therefore the absorbed dose is multiplied with a radiation weighting factor and results in the **equivalent dose**. Its unit is Sievert (Sv). This equivalent dose is assessed for a tissue or organ, for example for the thyroid.

The **effective dose** results from adding the organ doses which were weighted with another factor, the tissue weighting factor. Its unit is also Sievert (Sv). The tissue weighing factors represent the contribution of the tissues/organs to the total effects of the body. For example: the tissue weighting factor for the thyroid is 0.04. So an equivalent dose to the thyroid of, for example, 10 Millisievert (mSv), is multiplied by 0.04 to determine its contribution to the effective dose (10*0.04 = 0.4 mSv)

Radiation exposure does not only have an effect in the moment when the human body is contaminated, but also in the time afterwards. Radioactive particles can be inhaled or ingested and remain for a certain period of time in the human metabolism until they are excreted or decayed. In case of radioactive decay, radioactive daughter products can result that also have an impact on the human body. In radiation protection, such effects are taken into account by the so-called dose-commitment. For a **committed equivalent or committed effective dose** every yearly dose is summed up from the start of contamination until the age of 70 (for children), or for 50 years (for adults).

All these doses are used for assessing individual radiation effects. In radiation protection, also the **collective equivalent or collective effective dose** is of relevance. Summing up of all individual doses of a defined population results in the collective dose. Therefore, the collective dose could consist of many individual doses that are very small or a few higher individual doses. It is disputed if and how the collective dose should be used for assessing radiation risk of large groups of population – see chapter 4.1.1.4.

How can the received radiation be measured? Only some radiation workers are wearing dosimeter to measure external radiation. For all other people, radiation doses can only be assessed, meaning doses have to be calculated based on models and assumptions. For assessment of dose, the human body is represented by several types of models. Amongst others it has to be known what happens to incorporated radioactive particles during metabolism, the human cell repair mechanism are of importance, and different radiation sensitivity of tissues. For modelling the effects of external radiation, phantoms (nowadays computational or voxel phantoms) are radiated that are simulating human bodies. For incorporated radioactive material models of the respiratory tract and the intestinal tract are used. The effects of radiation on cells and tissues are analysed in vivo and in vitro[2].

Phantoms and models can provide us with an approach to reality, but they can only result in average dose assessments with some uncertainties. Especially people who are not corresponding to the

[2] In vitro: living tissue is taken from the body and analysed; in vivo: living tissue is analysed in the body (f.e. animal studies)

idealized so-called reference person because they react differently to radiation could receive higher doses than assessed. Moreover, there do not exist voxel phantoms for pregnant women and the foetus, nor for children of different age. (ICRP 103 2007, p.69)

Radiation protection measures are based on assumed relationships between radioactive contamination and health effects. These relationships are seldom easily to determine. Only for higher radiation doses there is an increased likelihood for **deterministic effects** (ICRP 103 2007, p. 96) Deterministic effects occur if a dose of about 500 mSv or more is received. The higher the absorbed dose, the higher the damage. Severe deterministic health effects are called acute radiation sickness. This life-threatening disease will develop at absorbed doses of 1 Gray or more. Symptoms are damage of the blood production system, severe skin damage, damages of the intestinal tract and the immune system. After the nuclear accident of Chernobyl according to the International Atomic Energy Agency IAEA and the World Health Organization WHO (WHO 1996) between 134 and 143 persons have died due to acute radiation sickness.

For **stochastic radiation effects** the risk of damage increases with the received dose. Especially important radiation health consequences are several types of cancer. No threshold for the risk of a health effect is known, therefore the **LNT-model (linear-no-threshold)** is the scientific model in use.

Although much is known about the health effects after exposure to radiation at the 100 mGy–1 Gy dose range and high dose rates, the effects of **low-dose radiation** still leave many open questions. Debate continues about how to extrapolate radiation risks at low doses, the biological effectiveness of low-dose radiation, and the effects of dose rate and external versus internal exposure.

Low-dose radiation research involves both experimental studies of radiation effects on molecules, cells, tissues, animal testing and observational studies on populations (epidemiological studies). Experimental studies help to understand the mechanisms by which **low**-dose ionising radiation causes damage and how the cells and tissues respond to that damage. Epidemiological studies are important for assessing health effects and risk factors. But especially in the low and very low dose range it is often difficult to prove a relation between dose and effect (f.e. cancer). This is due to the facts that not all cancer types are radiation induced, and there are mostly not many cancer cases in absolute numbers in a given population which can make statistical plausibility very difficult. There are also a lot of other known triggers for cancer (like smoking, socioeconomic factors, genetic factors, environmental toxins….). Their effect on cancer incidence is often not precisely separable from radiation effects. Another problem is the data base which is often not appropriate. For types of epidemiological studies see the glossary in chapter 5.

On the other hand it should be mentioned that while statistical significance is important to assess the results of epidemiological studies, also non-significant results can be of relevance, especially for low number of cases.

Risk factors are used to assess incidence and mortality in a given population due to a received defined dose. These risk factors are based on experimental research and on results of epidemiological and clinical studies. The amounts of such risk factors are disputed – see chapters 3 and 4.

3 New insights in health effects of ionising radiation

Health consequences are a key issue for nuclear energy. Health effects, especially of low-level radiation, are often difficult to prove scientifically, which leads to disagreements of many scientist and NGOs with international organizations like the International Commission on Radiological Protection (ICRP) that are still ongoing more than 30 years after the Chernobyl accident and more than 5 years after Fukushima.

The ICRP is especially in the centre of critique because its recommendations are used in EU radiation protection legislation – even though ICRP is no governmental body. According to the BSS-Directive, for internal exposure the dose model of publication ICRP 103 should be used. (Directive 2013/59/Euratom, recitals 7, 9) And this ICRP publication does not consider results from epidemiological studies after Chernobyl: "In general, the parameters in these risk models were estimated using incidence data from the studies of the Japanese atomic bomb survivors with follow-up from 1958 through to 1998 for solid cancers […]" (ICRP 2007, p. 178) Since 2007, no new overall recommendations have been published by ICRP.

In this chapter, results from up-to-date epidemiological studies on health effects of low level radiation after Chernobyl and Fukushima are discussed to complement the ICRP data and to evaluate the conclusions ICRP has drown. Also up-to-date studies of nuclear workers, effects of medical exposures and of natural background radiation are discussed.

3.1 Cancer

Cancer is one of the most important stochastic health effects of ionising radiation. Many studies were made to analyse if ionising radiation is an agent for special types of cancer, and on the dose-effect-relationship. However, the ICRP states in its publication 103 on which the BSS-Directive is based: "The overall estimates of cancer risk attributable to radiation exposure have not changed appreciably in the past 16 years." (ICRP 103 2007, p. 9) But since the past sixteen years a lot of new evidence has been found that should be taken into account.

3.1.1 Solid cancer mortality and incidence

Cancer mortality from higher doses of ionising radiation are researched quite well, especially in the LSS cohort of the Japanese atomic bomb survivors. But what was missing until recently are studies about effects of low or very low protracting doses of ionising radiation. To fill this gap, a big international study of **nuclear workers** has been conducted. The INWORKs study investigated cancer mortality among a cohort of 308,297 nuclear workers. (Richardson et al. 2015a) The workers were mostly men (87%), and the men received 97% of the total dose. They received an average cumulative colon dose of 20.9 mGy. The estimated excess relative rate (ERR, see glossary) of mortality from all cancers was calculated as 0.51 per Gy[3], for solid cancers 0.47 per Gy[4]. Smoking can be a confounder for lung cancer, therefore the study authors estimated also ERR for solid cancers deaths without lung cancer deaths, the ERR is 0.46 per Gy[5], which was similar to the ERR for all solid cancer deaths.

[3] (90% CI: 0.23, 0.82), lagged by 10 years

[4] (90% CI: 0.18, 0.79)

[5] (90% CI: 0.11, 0.85)

Results show a linear increase in the rate of cancer with increasing radiation exposure. The estimated association of dose and risk over the dose range of 0-100 mGy was similar in magnitude to that obtained over the entire dose range but less statistically precise. The study provides a direct estimate of the association between protracted low dose exposure to ionising radiation and solid cancer mortality. The study authors state that results are compatible with the extrapolation from acute high dose to low chronic dose, which is a main underlying hypothesis of the current radiation protection. (Richardson et al. 2015b) The study authors therefore argue that the so-called dose and dose-rate effectiveness factor DDREF (see glossary) is not justified in the light of their findings. (Richardson et al. 2015b).

In summary, the INWORKS study is important for confirmation that low, protracting doses also have negative effects on health and **that the DDREF of 2 that is used by ICRP is not justified any longer**.

Also for the **cohort of the Japanese atomic bomb survivors (Lifespan Study LSS)** new research results were published recently for cancer mortality. (Ozasa et al. 2012) For solid cancers the additive radiation risk (i.e., excess cancer cases per 10,000 person-years per Gy) continues to increase throughout life with a linear dose-response relationship. The sex-averaged excess relative risk per Gy was 0.42[6] for all solid cancer at age 70 years after exposure at age 30 based on a linear model. The risk increased by about 29%[7] per decade decrease in age at exposure. Important in this study is that the estimated lowest dose range with a significant ERR for all solid cancer was 0 to 0.20 Gy, and a formal dose-threshold analysis indicated no threshold. This is an affirmation of the LNT-model and means that every, even very small, radiation dose can cause a negative effect, there is no safe dose.

In table 1, ERR per Gy for solid cancer deaths are compared for ICRP, the recent results from LSS and the INWORKS study.

Table 1: Comparison of ERR per Gy for solid cancer deaths

	Men		Women	
	ERR per Gy	CI	ERR per Gy	CI
ICRP 103 (2007, table A.4.8)	0.35		0.58	
Ozasa et al. 2012 (LSS)	0.31	95% (0.21-0.42)	0.66	95% (0.52-0.80)
Richardson et al. 2015a (INWORKS)	0.47	90% (0.18-0.79)	-	

The ERR of the INWORKS study is higher than the one used in ICRP 103, and also higher than the ERR from Ozasa et al. (2012). Even though the confidence intervals overlap, this effect should be taken seriously and result in the annulment of the DDREF as recommended by the authors of the INWORKS study. This is especially important because the BSS-Directive uses the ICRP risk factors and therefore the DDREF.

[6] (95% CI: 0.32, 0.53)

[7] (95% CI: 17%, 41%)

Previously reported increase in the incidence of solid cancers and leukaemia due to radiation from the **Chernobyl** accident in the exposed populations continues to be investigated. In particular, a Chernobyl cohort of 530,000 registered recovery and clean-up operation workers (liquidators), who received doses ranging from 20 to 500 mSv in 1986-1990, is being closely followed up for potential risk of cancer and other diseases. (WHO 2016)

3.1.2 Leukaemia and lymphomas

Leukaemia is the umbrella term for different types of cancers of the blood cells, which are forming in the bone marrow. Different types of leukaemia depend on the type of blood cell that develop cancer. There are chronic and acute types. The four common types are Chronic Lymphoblastic Leukaemia (CLL), Chronic Myeloid Leukaemia (CML), Acute Lymphoblastic Leukaemia (ALL) and Acute Myeloid Leukaemia (AML). Children's leukaemia is mostly of the acute type. *Lymphomas* are blood cell tumors developed from lymphocytes. They can be cancerous. Types are Hodgkin and non-Hodgkin Lymphomas.[8]

It is well known that leukaemia can be caused by high radiation doses (based on analysing the Japanese bomb survivors), but the effects of low doses are still disputed. Also it was believed that CLL is not radiation induced. New studies, which will be introduced below, show that also low, protracted doses increase the risk of leukaemia, and that CLL can also be radiation induced.

A nested case-control study was conducted within cohorts of **Chernobyl** liquidators from Belarus, the Russian Federation and the Baltic countries who had worked in 1986-87 around the Chernobyl plant. (Kesminiene et al. 2008) Most cases received very low doses to the bone marrow (median 13 mGy). For all diagnoses combined, a significantly elevated odds ratio (OR, see glossary) was seen at doses of 200 mGy and above. The ERR per 100 mGy was 0.60[9]. The corresponding estimate for leukaemia excluding CLL was 0.50[10]. The authors state that this ERR is slightly higher than, but statistically compatible with, those estimated from a-bomb survivors and recent low dose-rate studies.

Risks of most types of leukaemia from exposure to acute high doses of ionising radiation are well known, but risks associated with protracted exposures, as well as associations between radiation and chronic lymphocytic leukaemia (CLL), are not clear. Therefore, another nested case-control study of leukaemia was conducted in a cohort of Ukrainian liquidators. (Romanenko et al. 2008) The cases received a mean bone marrow dose of 76.4 mGy. The ERR of total leukaemia was 3.44 per Gy[11]. The increase in leukaemia risk was significant and similar in magnitude to the estimate from the Japanese atomic bomb survivors. The data indicated elevated risks for both CLL and other types of leukaemia. Therefore the study was extended through 2006, with a near doubling of the number of leukaemia cases. Results of the extension are reported from Zablotska et al. (2013). Analysed were all cases of leukaemia that have been diagnosed between 1986 and 2006. The controls were matched by age and place of birth. Individual radiation doses were estimated for the bone marrow. For CLL, the ERR/Gy was 2.58[12], and for non-CLL, ERR/Gy was 2.21[13]. Altogether, 16% of leukaemia cases (18% of CLL, 15%

[8] For more explanations see f.e. http://www.leukaemiacare.org.uk/leukaemia - but be aware that on this website there is still the assumption that only high doses of radiation can cause leukaemia.

[9] (90% CI: −0.02, 2.35)

[10] (90%CI: −0.38, 5.7)

[11] (95% CI: 0.47, 9.78) p<0.01

[12] (95% CI: 0.02, 8.43)

[13] (95% CI: 0.05, 7.61)

of non-CLL) were attributed to radiation exposure. Based on this primary analysis, the study concluded that **both CLL and non-CLL are radiosensitive**. Using the age-specific incidence rate of CLL among men in Ukraine for 2003, it was estimated that the number of CLL cases diagnosed in the analysed cohort over the period of 20 years after the accident was 60% higher than what would be expected for the general male population of Ukraine.

A Japanese online media platform published on Aug 20th, 2016 that already two workers who developed leukaemia after clean-up in **Fukushima** were entitled to workers compensations[14]. The first man received a dose of about 16 mSv, the second man of about 54.4 mSv. Additional applications for compensations are expected.

The above mentioned **International Nuclear WORKers** Study (INWORKS) analysed effects of low, protracted or intermittent doses on cancer mortality. One of the publications of the INWORKS team shows new insights into mortality by leukaemia and lymphoma. (Leuraud et al. 2015) In this study, 308,297 nuclear workers from three different countries (France, USA and UK) were included in an international cohort study. The workers have been monitored for external exposure to radiation with personal dosimeters and followed up for up to 60 years after exposure. The association between their bone marrow doses and mortality due to leukaemia and lymphoma was studied. The ERR of leukaemia mortality (without CLL) was 2.96 per Gy[15] mostly tributed by chronic myeloid leukaemia. As the authors state, this study provides strong evidence of positive associations between protracted low-dose radiation exposure and leukaemia.

The German Bundesamt für Strahlenschutz (BfS) commented on the results of the INWORKS study[16]. The BfS argued that only for doses from 50-100 mSv significant ERR was found, and in case of leukaemia only for the CLL subtype. The BfS recommended further research.

Nevertheless, also at doses lower than 50 mSv there is evidence for leukaemia risk. The studies of the Chernobyl liquidators cohort show that CLL is also radiation induced. This should not be neglected by waiting for further studies, but be reflected upon in the BSS-Directive by using lower dose limit in all protection situations.

Discussion about childhood leukaemia see chapter 3.1.5

3.1.3 Thyroid cancer

Radioactive iodine is one of the first radionuclides released by a nuclear accident. If inhaled or ingested it will accumulate in the thyroid gland and increase the risk of thyroid cancer.

An increase in thyroid cancer became evident a few years after **Chernobyl** in parts of Ukraine and Belarus. The physicist and radiation biologist Lengfelder and radioecologist Frenzel reported a more than 30-fold increase in thyroid cancer in children in Belarus already at the end of 1990 compared to the long-term mean value before 1986. (Lengfelder and Frenzel 2006) But in the report of the International Chernobyl Project in 1991, the International Atomic Energy Agency (IAEA) still tried to downplay this health effect[17].

In the UNSCEAR Scientific Annex for Chernobyl effects (UNSCEAR 2011, p. 148) an overview of epidemiological studies on thyroid cancer is given. Cohort studies and case-control-studies show

[14] The Asahi Shimbun, 20. Aug. 2016

[15] (90% CI: 1.17, 5.21), lagged 2 years

[16] http://www.bfs.de/SharedDocs/Stellungnahmen/BfS/EN/2015/08-03-inworks-study.html, seen 24 Feb 2017

[17] "The data did not show a marked increase in leukaemia or thyroid tumors since the accident". (IAEA 1991, p.12)

excess relative risk (ERR, 95% CI) of 1.65 to 48.7 per Gy, ecological studies of 4.4 to 67.8. Women are more at risk than men. Increases have not stopped by now.

Also Cardis and Hatch (2011) made an overview of studies on thyroid cancer that have been published until 2011. In their findings they stated that from the available data, it appears that in the case of external radiation thyroid cancer risk following I-131 exposure from Chernobyl decreases with increasing age at exposure. The excess post-Chernobyl thyroid cancers that are now occurring arise primarily in young adults who were exposed at young ages. This, together with the more modest ERRs/Gy reported for exposed adults, suggests the age-at-exposure effect is likely to be real.

Both Belarus and Ukraine have reported continuing increases in thyroid cancer incidence for all ages and both genders. Although dose was not directly taken into account in either case, in Belarus greater increases were found in areas with higher exposure to Chernobyl fallout. Thyroid cancer occurs more often among females than males.

A prospective cohort study, involving individual dose estimates and serial screening examinations of children and adolescents in Ukraine through 2007, found no variation in radiation risk by time since exposure. (Brenner et al. 2011) I-131–related thyroid cancer risks persisted for two decades after exposure, with no evidence of decrease during the observation period. The radiation risks, although smaller, were found to be compatible with those of retrospective and ecological post-Chornobyl studies.

Also in a study of cancer rates in liquidators, evacuees and inhabitants of a highly contaminated Ukrainian region significant excess of thyroid cancer was found. (Prysyazhnyuk et al. 2014) The authors explain that the increase of thyroid cancer incidence was registered not only in children, but also in adolescents and adults. Appearance of excess thyroid cancer cases as an effect of radiation exposure tends to increase during the time.

In his update of the TORCH report, Ian Fairlie cited two more studies that showed a long latency period for thyroid cancer (Fairlie 2016): Before the Chernobyl accident, the principal source of information about radiation-induced thyroid cancer in children were studies in which children had been exposed to external X-rays for medical reasons. A survey of these (Ron et al. 1995) showed that the thyroid cancer risk was still increased more than 40 years after the initial exposure. A study of thyroid cancer incidence in the survivors of the Japanese atomic bombs (Imaizumi et al. 2006) found a significant dose-response relationship still existing nearly 60 years after exposure. The authors also observed that the effects were much greater in those exposed at younger ages.

In 2016, the first study about thyroid cancer after **Fukushima** was published (Tsuda et al. 2016). After the accident, the Fukushima Prefecture performed ultrasound thyroid screening on all residents aged ≤18 years in March 2011. The first round of screening included 298,577 examinees. A second round for residents who were born between Apr 2011 and Apr 2012 began in April 2014 and was completed in March 2016. Tsuda et al. analysed the Prefecture results from the first and second round up to December 31, 2014, in comparison with the Japanese annual incidence and the incidence within a reference area in Fukushima Prefecture. As a result they observed the highest incidence rate ratio, using a latency period of four years, in the central middle district of the prefecture compared with the Japanese annual incidence (incidence rate ratio = 50[18]). The prevalence odds ratio compared with the reference district in Fukushima Prefecture was 2.6[19]. In the second screening round, even under the assumption that the rest of examinees were disease free, an incidence rate ratio of 12 has already

[18] (95% CI: 25, 90)

[19] (95% CI: 0.99, 7.0)

been observed[20]. As conclusion it can be said that an excess of thyroid cancer has been detected among children and adolescents in Fukushima Prefecture within four years of the release, and is unlikely to be explained by a screening surge.

In 2017, IPPNW analysed results from the completed second round. They found an incidence rate of 8.1 thyroid cancer cases per 100,000 children per year. Before Fukushima, this incidence rate was 0.3 cases per 100,000 and per year. (IPPNW 2017) In 2016, the first worker of TEPCO with thyroid cancer has been acknowledged to have gotten the disease due to his work in NPP Fukushima[21]. The man will receive compensation.

3.1.4 Breast cancer

Besides thyroid cancer and leukaemia which are discussed above, breast cancer as a type of solid cancer is a main cause of death for women.

Cardis et al. (2006) published a review of knowledge about breast cancer after **Chernobyl**. The authors concluded that several studies showed increases but lacked information about dose levels. This gap was closed by an ecological study investigating doses and increases in breast cancer in Belarus and Ukraine in age-cohorts in differently contaminated regions. (Pukkala et al. 2006) A significant two- to three-fold increase in risk was observed during the period 1997–2001 in the most contaminated districts (average cumulative dose of 40.0 mSv or more) compared with the least contaminated districts. The relative risk (RR) in Belarus was 2.24[22] and in Ukraine 1.78[23]. The ERRs seen in those areas were much higher than expected in comparison to the LSS results: 120%[24] in Belarus and 80%[25] in Ukraine; these are, however, very uncertain as they are based on small numbers of cases (34 in Belarus and 22 in Ukraine). The increase, though based on a relatively small number of cases, appeared approximately ten years after the accident, and it was highest among women who were younger at the time of the accident. The authors also state that it is unlikely that this excess could be entirely due to the increased diagnostic activity in these areas.

These results are supported by a case-control study among young Ukrainian women exposed by Chernobyl. (Khyrunenko et al. 2011) Women investigated were up to minus 9 months (in utero) to 18 years at the time of accident. For each case and control living in contaminated territories the individual accumulated dose of irradiation was estimated. The odds ratios calculated from the case-control comparisons are indicative of a relationship between radiation dose and the development of breast cancer.

Another descriptive epidemiological study of clean-up workers from Ukraine, evacuees from the 30km zone and residents of the most contaminated areas of the Ukraine was carried out. (Prysyazhnyuk et al. 2014) Significant excess for breast cancer was revealed among female clean-up workers.

Because of a longer latency period there are no data for breast cancer caused by **Fukushima** available by now.

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[20] (95% CI: 5.1, 23)

[21] http://www.spreadnews.de/fukushima-aktuell-erster-akw-arbeiter-mit-schilddruesenkrebs-anerkannt/1151520/, seen 24. Feb 2017

[22] (95% CI: 1.51, 3.32)

[23] (95% CI: 1.08, 2.93)

[24] (95% CI: 50%, 230%)

[25] (95% CI: 10%, 190%)

The question if breast cancer can also result from **normal operation of NPPs** was investigated by C. Busby. (Busby 2015a) He studied breast cancer because in contrary to childhood leukaemia an excess risk caused by ionising radiation is better detectable because of higher background rates (150 breast cancer cases per 100,000 in UK) and of a bigger study population at risk (age 45-75). Busby examined the risk of breast cancer mortality between 1995 and 2001 in wards adjoining the estuary of the River Blackwater in Essex, UK, where radionuclide contamination can be measured in muddy sediment and other material, derived from discharges from the Bradwell Nuclear Power station. Estuary wards were compared to inland wards using social class adjusted expected numbers based on national mortality rates for the period. Results showed a significant effect with relative risk for the River Blackwater Estuary vs. the other wards with RR = 1.7[26]. In addition, Busby compared the contaminated Blackwater wards to the wards of the River Crouch which he defined as non-contaminated. Comparison with this ward showed also RR = 2.1[27].

These results show that breast cancer is not only caused by radioactive contamination but can even occur at low doses such as doses caused by effects of normal operation or well below 100 mSv like in the study of Pukkala et al. (2006.). Breast cancer could also be caused by normal operation of NPPs. To provide for better radiation protection, such results should lead to lower dose limits and levels (for all three protection levels: planned situations, emergency exposure and existing exposure) in the BSS-Directive.

3.1.5 Childhood cancer including leukaemia

Recent studies about childhood cancer[28], especially about leukaemia, provide new evidence by linking even very low doses to the embryo/foetus[29] or children in their first years of life to increases in cancer.

3.1.5.1 Childhood cancer resulting from Chernobyl

In 1996, a Greek study was published that showed an increase in childhood leukaemia in children (Petridou et al. 1996). This study was important because the authors found a significant increase of the incidence rate of 2.6%[30] in children who were prenatally irradiated (born between 7-1-1986 and-12-31-1987) compared to the prenatal non-irradiated children. This control group included children who were born between 1-1-1980 and 12-31-1985, and 1-1-1988 until 12-31-1990. Especially children from regions that were contaminated with Cs-137 above 1 kBq/m^2 showed an increase. For comparison: In Austria, the mean surface contamination after Chernobyl was 37 kBq Cs-137/m^2. A contamination of 1 kBq/m^2 has occurred all over Europe – see figure 1.

[26] (CI: 1.22, 2.34), p = 0.0015

[27] (CI: 1.12, 3.98), p = 0.018

[28] Thyroid cancer in children see chapter 3.1.3

[29] A foetus is an embryo after the development of internal organs (9th week of pregnancy)

[30] (95% CI; 1.4 – 5.1), p= 0.003

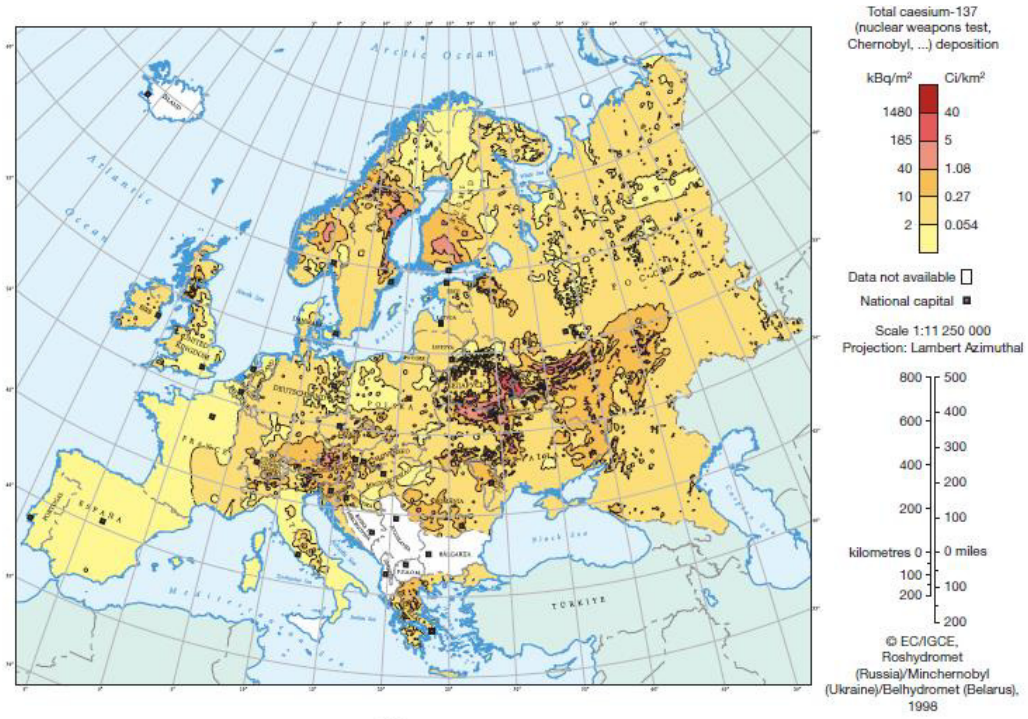

FIG. 3.5. Surface ground deposition of ^{137}Cs throughout Europe as a result of the Chernobyl accident [3.13].

Figure 1: Surface contamination with Cs-137 in Europe (IAEA 2006)

Newer epidemiological studies also found increases in risk for children who were in utero or younger than six years when the contamination from Chernobyl occurred. (Davies et al. 2006) The mean bone marrow dose was estimated below 10 mGy. In the Ukraine, a significant leukaemia increase was found, in Belarus a non-significant increase and no increase in the contaminated parts of Russia. The excess relative risk at 1 Gy (ERR/Gy) was estimated for each republic and all republics combined. For all republics combined the estimated ERR/Gy was 32.4. The ERR/Gy was much larger in Ukraine (78.8) compared with Belarus (4.1) and Russia (4.94). CIs were very wide and overlapped.

A case-control study was conducted among children aged 0-5 years in the Ukraine's mostly contaminated regions. (Noshchenko et al. 2010) The aim of the study was to analyse the children's risk of acute leukaemia. The children were diagnosed with leukaemia between 1987 and 1997 and were resident in the contaminated region. Four dose-range groups were selected for statistical analysis (0–2.9 mGy, 3–9.9 mGy, 10–99.9 mGy and 100–313.3 mGy). The risk of leukaemia was significantly increased, ERR was 2.4[31] among those with radiation exposure doses higher than 10 mGy. The risk was increased particularly for acute myeloid leukaemia.

In 2009, C. Busby published his work on childhood leukaemia after foetal exposure with very low dose after Chernobyl (Busby 2009). He studied children who were born between 1980 and 1990 in Greece, Germany and UK, assuming a similar model like Petridou et al. (1996). The exposed cohort included children born between 1 July 1986 and 31 Dec. 1987. This period was chosen because internal exposures of the mothers could be detected via whole-body counters until spring 1987. All other children were included in the non-exposed cohort. The ERR was 1.43[32] and significant. The mean foetal

[31] (95%CI: 1.4, .0), p=0.01

[32] (95% CI: 1.13, 1.80)

dose was calculated 0.067 mSv. Because the ICRP did not expect significant increases for such very low doses, the author questions the ICRP dose model.

3.1.5.2 Childhood cancer from normal operation of NPPs

Even the event-free routine operation of nuclear power plants leads to discernible health effects in the surrounding population. Childhood leukaemia and other forms of childhood cancers show higher incidence rates in populations living in the vicinity of nuclear power plants, with a clear correlation between cancer risk and the distance to the plant. The strongest evidence comes from the German KIKK[33] study (Kaatsch et al. 2007), with consistent results in studies from Switzerland (Spycher et al. 2011), France (Körblein and Fairlie 2012) and the UK (Bithell et al. 2008; COMARE 2011).

A pattern of epidemiological evidence world-wide now clearly indicates increased leukaemia risks near nuclear power plants (NPP). Laurier and Bard (1999) and Laurier et al. (2008) examined the literature on childhood leukaemia near NPPs world-wide. Result: over 60 epidemiological studies world-wide have examined cancer incidences in children near NPPs. An independent review of these studies showed that most of them (>70%) indicate leukaemia increases (Fairlie 2013; Fairlie 2014).

The above-mentioned 2008 KiKK study commissioned by the German Government found relative risks (RR) of 1.6 in total cancers and 2.2 in leukaemia among children under the age of 5 years living within 5 km of all German NPPs. In this study, the environments of all German NPP were examined between 1980 and 2003; equivalent cases outside this area were studied as controls (Spix et al. 2008).

The KiKK study has retriggered the debate as to the cause(s) of these increased cancers. Yet many Governments of nuclear countries and the nuclear industry refute these findings and continue to resist their implications.

Increased childhood leukaemia near NPPs has been a contentious issue for several decades. As a result of these findings, governments in France (Sermage-Faure et al. 2012), Switzerland (Spycher et al. 2011) and the UK (COMARE 2011) hurriedly set up studies near their own NPPs. All of them found leukaemia increases but because their numbers were small the increases are not of statistical significance.

Körblein and Fairlie (2012) combined datasets in a meta-study to get larger numbers and, thus, reach higher levels of statistical significance. They pooled the data of acute leukaemia in children under 5 years within 5 km of NPPS from four studies (see table 2).

[33] KIKK=Kinderkrebs in der Umgebung von Kernkraftwerken; engl. Childhood Cancer in the Vicinity of Nuclear Power Plants.

Table 2: Studies of observed (O) and expected (E) leukaemia cases within 5 km of NPPs (Körblein and Fairlie 2012)

	Observed (O)	Expected (E)	SIR=O/E	90% CI	p-value
Germany	34	24.1	1.41	1.04-1.88	0.0328
Great Britain	20	15.4	1.30	0.86-1.89	0.1464
Switzerland[a]	11	7.9[a]	1.40	0.78-2.31	0.1711
France[b]	14	10.2	1.37	0.83-2.15	0.1506
Pooled data	**79**	**57.5**	**1.37**	**1.13–1.66**	**0.0042**

[a] derived from data in Spycher et al. (2011).

[b] acute leukaemia cases

This table reveals a highly statistically significant 37% increase in childhood leukaemia within 5 km of almost all NPPs in the UK, Germany, France and Switzerland. Thus, there is a very clear association between increased childhood leukaemia and proximity to NPPs. The question remains open what is/are the reason(s) for this.

The authors of the KIKK study stated that a dose of 2 Sv was necessary for the observed leukaemia rate, i.e. more than a thousand times the actually received dose. Fairlie shows that the discrepancy can be explained. A suggested hypothesis is that the increased cancer incidence results from radiation exposures of pregnant women near NPPs. An explanation may be that doses from spikes in NPP radionuclide emissions are significantly larger than those estimated by official models which are diluted through the use of annual averages. In addition, risks to embryos/foetuses are greater than those to adults and haematopoietic tissues appear more radiosensitive in embryos/foetuses than in newborn babies.[34] The product of possible increased doses and possible increased risk per dose may provide an explanation. (Fairlie 2014)

After several recent European studies found disturbing links between childhood cancer and kids living close to nuclear plants, the US Nuclear Regulatory Commission NRC contracted with the National Academies of Sciences (NAS), a separate agency, to design a modern scientific assessment in 2010. The NRC spent five years and $1.5 million on the project before abandoning it two years ago. Expecting that nothing would be found, NRC officials decided they couldn't justify the costs.[35] (Sforza 2017)

[34] Fairlie got his explanation from the observation of the KIKK study: the increased solid cancers were mostly "embryonal", i.e. babies were born either with solid cancers or with pre-cancerous tissues which, after birth, developed into full-blown tumours: this actually happens with leukaemia as well. (Fairlie 2014)

[35] This study design is much more rigorous than what was done in Europe, and the NAS was the first to admit it was a complicated endeavour that would take an enormous amount of work. The NAS would track not just geography and cancer incidence, but also radiological releases from the plants themselves, and see if there was any cancer correlation. According to the NAS, a pilot study of seven of sites would take 39 months and cost $8 million, and those results would not necessarily extrapolate out to all nuclear sites. Studying them all would take many more years, and many more millions, officials said. The head of the aborted study at the NAS criticized this decision and explained "You do not know whether the study will find something unless you do the study."

3.1.5.3 Childhood cancer from natural background radiation

Two recent studies show the high radio-sensitivity of children[36]:

A Swiss study investigated childhood leukaemia and lymphoma caused by natural background radiation from terrestrial gamma and cosmic rays. (Spycher et al. 2015) A nationwide census-based cohort study was conducted for children < 16 years in 1990 and 2000, with follow-up until 2008.[37] The study found evidence of an increased risk of cancer among children exposed to external dose rates of background ionising radiation of ≥200 nSv/h (1.75 mSv/a) compared to those exposed to <100 nSv/h (0.88 mSv/a). The increased risk among children exposed to dose rates ≥200 nSv/h compared to those exposed to <100 nSv/h for leukaemia was hazard ratio (HR) = 2.04[38].

Kendall et al. (2013) conducted a large record-based case-control study testing associations between childhood cancer and natural background radiation. Cases (27,447) born and diagnosed in Great Britain between 1980 and 2006 and matched cancer-free controls (36,793) were from the National Registry of Childhood Tumours. The mean cumulative red bone marrow (RBM) equivalent dose from gamma-rays and radon[39] combined over the period from birth to diagnosis for the first controls is 4.0 mSv with a range from zero (for those diagnosed at birth) up to about 31 mSv.[40] There was 12% excess relative risk (ERR)[41] of childhood leukaemia per mSv of cumulative RBM dose from gamma radiation. The authors concluded: The results of the study contradict the idea that there are no adverse radiation effects, or might even be beneficial effects, at these very low doses and dose-rates.

3.1.5.4 Childhood cancer from medical exposure

The same logic as above—focusing on subgroups of the population in which the excessive relative risk of cancer after radiation exposure is supposed to be largest—applies to two more recently published epidemiological studies of cancer risks associated with pediatric exposure to computed tomography (CT) scans, both of which had a relatively short mean follow-up of about 10 years. (Mathews et al. 2013; Pearce et al. 2012) The relatively short follow-up after pediatric exposure permits detection of radiation-induced cancers with short latency while excluding investigation of those cancers that may appear at later ages. Both of these studies had large cohorts, and both showed a statistically significant association between the number of CT scans and increased cancer risk. Although CT scans are very useful clinically, potential cancer risks exist from associated ionising radiation, in particular for children who are more radiosensitive than adults.

Mathews et al. (2013) reported a statistically significant dose-response relationship over the range of zero to more than three CT scans, and the cancer incidence rate ratio increased by 0.16[42] for each additional scan. Mathews et al. (2013) derived direct estimates of the increased cancer risk after CT scan exposure by comparing cancer incidence in over 680,000 people exposed to CT scans at ages 0-

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[36] Note: none of the two investigations takes into account the genetic risks resulting from the parents' radiation or the exposition in utero, although this would be required according to Schmitz-Feuerhake et al. (2016). (See chapter 3.2).

[37] On average, natural terrestrial radiation contributed 54 nSv/h, cosmic radiation 45 nSv/h and artificial terrestrial radiation 8 nSv/h.

[38] (95% CI: 1.11, 3.74)

[39] On average, radon contributed about 10% of the RBM equivalent dose, although contributions were very variable with a range 1% to 80%.

[40] To compare the risk estimates from this study with published estimates, it was necessary to calculate doses to the target tissue in question, and if the risks from gamma-rays and radon are to be examined together doses from both sources must be calculated on the same basis. This could be done only for leukaemia, for which the relevant quantity is the (RBM) equivalent dose.

[41] (95% CI 3, 22), two-sided P=0.01

[42] (95% CI: 0.13, 0.19)

19 years with cancer incidence in a comparison cohort of over 10 million unexposed persons of similar age. The mean duration of follow-up after exposure was only 9.5 years.

60,674 cancer cases were recorded, including 3,150 in 680,211 people exposed to a CT scan at least one year before any cancer diagnosis. Overall cancer incidence was 24% greater for exposed than for unexposed people, after accounting for age, sex, and year of birth (incidence rate ratio (IRR) 1.24[43]. The IRR increased significantly for many types of solid cancer (digestive organs, melanoma, soft tissue, female genital, urinary tract, brain, and thyroid); leukaemia, myelodysplasia, and some other lymphoid cancers. The average effective radiation dose per scan was estimated as 4.5 mSv.

Pearce et al. (2012) aimed to assess the excess risk of leukaemia and brain tumours after CT scans in a cohort of children and young adults. In a retrospective cohort study, the authors included patients without previous cancer diagnoses who were first examined with CT in National Health Service (NHS) centres in England, Wales, or Scotland (Great Britain) between 1985 and 2002 when they were younger than 22 years.

They noted a positive association between the radiation dose from CT scans and leukaemia with an excess relative risk (ERR) per mGy of 0.036[44], and brain tumours 0.023[45]. Compared with patients who received a dose of less than 5 mGy, the relative risk of leukaemia for patients who received a cumulative dose of at least 30 mGy was 3.18[46] and the relative risk of brain cancer for patients who received a cumulative dose of 50-74 mGy was 2.82[47]. Pearce et al. (2012) concluded that in children the use of CT scans that deliver cumulative doses of about 50 mGy might triple the risk of leukaemia, and doses of about 60 mGy might triple the risk of brain cancer.[48]

3.1.5.5 Childhood cancer resulting from in-utero medical exposure and preconceptional exposure

A study of childhood cancers after in-utero medical diagnostic imaging shows an excess relative risk among the exposed subjects, because they were exposed at a critical point in their development. Indeed, Doll and Wakeford (1997) detected a significant increase in childhood cancer risk for a mean dose of about 10 mGy.[49]

The results from this large study and others showing an association between **in-utero** exposure and **cancer risk in childhood** (IARC 2012) are widely accepted and have changed medical practice related to exposure of pregnant women to ionising radiation.

But also cancer in children after preconceptional low-dose exposure of parents (occupational or medical exposure) has already been found in several studies. They are listed in Schmitz-Feuerhake et al. (2016), among them:

McKinney et al. (1991) found a 3.2-fold increase in leukaemia and lymphomas in children of occupationally exposed men in three British regions in a case-control study.

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[43] (95% CI: 1.20, 1.29), P<0.001

[44] (95% CI: 0.005. 0.120;), p=0.0097

[45] (95% CI: 0.010, 0.049), p<0·0001

[46] (95% CI: 1.46, 6.94)

[47] (95% CI: 1.33–6.03)

[48] Because these cancers are relatively rare, the cumulative absolute risks are small: in the 10 years after the first scan for patients younger than 10 years, one excess case of leukaemia and one excess case of brain tumour per 10 000 head CT scans is estimated to occur.

[49] Note that the absolute risk of childhood cancer is low, even among those exposed in utero.

In 1984, an exceptionally high level of leukaemia cases in children and juveniles was reported in Seascale, near the nuclear reprocessing plant in Sellafield (UK). The authors explained this as a hereditary effect, because the fathers of the patients had worked in the plant (Gardner et al. 1990). The authorities argued that the doses were too low to be possibly responsible for such effects. The effect, however, had been described in principle already in experimental studies (Nomura 1982), and also after X-ray diagnostic exposures. (Genetic and teratogenic effects are further discussed in chapter 3.2)

3.1.5.6 Conclusions for childhood cancer including leukaemia

A pattern of epidemiological evidence world-wide now clearly indicates an increased leukaemia risk near nuclear power plants (NPP). Furthermore, recent studies concerning childhood cancer from natural background radiation and medical exposure indicate the high radio-sensitivity of children. Studies about leukaemia risk for unborn and very young children show significant increases in leukaemia risks for foetal exposure to the Chernobyl contamination.

The ICRP emphasised that the limited data from the atomic bomb survivors suggest that the lifetime cancer risk from in-utero exposure may be similar to that from exposure in early childhood. However, the ICRP also stated: given the limitations of the available data, the ICRP has not attempted to derive a specific value for the nominal coefficient for life-time cancer risk after prenatal exposure.[50] The ICRP emphasizes that there are uncertainties in the risk of radiation-induced solid cancers following in-utero exposure. Nevertheless, the ICRP considers that it is prudent to assume that life-time cancer risk following in-utero exposure will be similar to that following irradiation in early childhood, i.e., at most, about three times that of the population as a whole (=16.5% per Sv). (ICRP 2007)

In the light of the depicted studies there is considerable doubt whether the risk for the embryo/foetus and very young children is not more than three times higher than the risk for the overall population. Especially for embryo/foetus this ICRP assumption seems to be insufficient.

3.2 Genetic and teratogenic effects

Subsequent to nuclear accidents, **teratogenic effects** have been observed, even in those who were only exposed to low or very low levels of radiation. (Busby et al. 2009; Körblein and Küchenhoff 1997; Körblein 2003, 2004b)

The ICRP judges that, following **prenatal** (in-utero) exposure, a) cancer risk will be similar to that following irradiation in early childhood and b) a threshold dose exists for the induction of malformations and for the expression of severe mental retardation. It is explained that the risks of tissue reactions and malformation in the irradiated embryo and foetus have been reviewed in Publication 90 (ICRP 2003). In respect of the induction of malformations, the new data strengthen the view that there are gestational age-dependent patterns of **in-utero radio-sensitivity** with maximum sensitivity being expressed during the period of major organogenesis. On the basis of animal data, it is

[50] The largest case-control study of cancer after in-utero irradiation, the Oxford Study of Childhood Cancers (OSCC), found that radiation increased all types of childhood cancer by approximately the same degree. The second largest study showed a larger relative risk of leukaemia than for solid tumours, while several cohort studies of in-utero radiation found no clear evidence of radiation-induced childhood cancer. The OSCC data suggest that cancer induction is at least as likely following exposure in the first trimester as in later trimesters. From the data published to date, it is not possible to determine tissue-weighting factors in order to define cancer risk in different tissues and organs. Adequate human in-utero exposure data are not available to define the dose and dose-rate effectiveness factor (DDREF) for low-LET radiation or the RBE values for neutron or other high-LET radiations. (ICRP 2007)

judged by the ICRP that there is a true dose threshold of around 100 mGy for the induction of malformations.

According to ICRP, the new data also confirm embryonic susceptibility to the lethal effects of irradiation in the pre-implantation period of embryonic developments. But according to the ICRP at doses under 100 mGy such lethal effects will be very infrequent.

However, Körblein (2011) states that the ICRP threshold dose of 100 mSv of teratogenic effects has to be given up. He argues that the mortality rate of new-born (perinatal mortality) was increased in Germany in 1987, the year after Chernobyl, compared with the trend of the years 1980-1993. Also in Poland and Eastern European countries, significant peaks of perinatal mortality and stillbirths were found in 1987.

Medical exposure in utero cannot only cause leukaemia and cancer (see chapter 3.1.5), but also perinatal mortality, congenital effects etc. Studies have also shown that in-utero exposure of the brain to ionising radiation leads to impaired cognitive development. (Hall et al. 2004; Heiervang et al. 2010).

Recently, Körblein noticed a 15% drop, which is statistically highly significant, in the numbers of births in **Fukushima** Prefecture in December 2011, i.e. nine months after the accident. This might point to higher rates of early spontaneous abortions. Also, a (statistically significant) 20% increase in the infant mortality rate in 2012 was observed, relative to the long-term trend in Fukushima Prefecture plus six surrounding prefectures. These are indicative rather than definitive findings and need to be verified by further studies. Unfortunately, such studies are notable by their absence. (Fairlie 2015)

All the congenital malformations (CM) effects are caused by mutation of DNA whether in the parental germ cells and precursors or from implantation to birth. The question of germ cell damage in parents or in utero damage to development, though important, is not to answer yet.

Exposure of the germ cells (gonads) can cause mutations in the genetic material which may result in **heritable disease** in the offspring of the exposed persons. Heritable diseases are expressed in children and further generations as malformations, metabolic malfunctions, immune deficiencies etc.

Most serious effects of ionising radiation – hereditary defects in the descendants of exposed creatures – had been already detected in the 1920s by Herman Joseph Muller. He exposed fruit flies (drosophila) to X-rays and found malformations and other disorders in the following generations. He concluded from his investigations that low dose exposure, and therefore even natural background radiation, is mutagenic and there is no harmless dose range for heritable effects or for cancer induction. His work was honoured by the Nobel Prize for medicine in 1946. (Schmitz-Feuerhake et al. 2016)

According to ICRP 103 (2007), radiation-induced heritable disease has not been demonstrated in human populations but there is substantial evidence from animal studies[51] of heritable damage to germ cells (ova and spermatozoa) as well as their precursor cells. Therefore, the ICRP prudently continues to include the risk of heritable effects in its system of radiological protection.

But the estimate of genetic (heritable) risk from radiation has been substantially revised as a result of both new information that has become available and the work of ICRP during the interim. It has to be noted: the new ICRP 103 is only based on studies published not later as 2001.

The risk of heritable effects in the whole population associated with gonadal dose is now estimated to be around 20 cases per 10,000 people per Sv, rather than about 100 cases per 10,000 per Sv.[52]

[51] Mouse studies continue to be used to estimate genetic risks because of the lack of clear evidence in humans that germline mutations caused by radiation result in demonstrable genetic effects in offspring.

[52] For heritable effects, the detriment- adjusted nominal risk in the whole population is estimated as $0.2\ 10^{-2}$ per Sv and in adult workers as $0.1\ 10^{-2}$ per Sv^{1}.

Nevertheless, the ICRP emphasises that this reduction in the gonadal tissue weighting factor provides no justification for allowing controllable gonadal exposures to increase in magnitude.

However, the ICRP decreased its risk estimate for heritable damage between its recommendations of 1991 and the recent ones of 2007 (ICRP 1991, 2007). Its Detriment Adjusted Nominal Risk Coefficient for radiation heritable effects in an exposed population was reduced from the previous 1991 value of 1.3% per Sv to 0.2% per Sv. The ICRP approach is based on a linear relation between dose and end-point, measured as heritable disease at or before birth. The belief that heritable consequences of radiation were negligible followed from studies of the Japanese survivors of the atomic bomb explosions in Hiroshima and Nagasaki in 1945. The American-Japanese Institute in Hiroshima, Atomic Bomb Casualty Commission (ABCC), apparently did not find mutations in the descendants of the survivors. Therefore, the ICRP derives its current risk figure from experiments in mice. The result corresponds to the evaluation by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR committee) (UNSCEAR 2001).

Schmitz-Feuerhake, Busby and Pflugbeil have published very recently a paper in which they bring up arguments for a new assessment. They criticize UNSCEAR and ICRP for their very low risk factors for hereditary diseases in humans based on reportedly absent genetic effects in the acute exposed Japanese A-bomb survivors. Schmitz-Feuerhake et al. (2016) made a compilation of findings about early deaths, congenital malformations, Down´s syndrome, cancer and other genetic effects observed in humans after the exposure of parents who were contaminated by Chernobyl fallout, parents who were clean-up workers and nuclear test veterans. Nearly all types of hereditary defects were found in cases affected by very low doses. The authors suggest that the results show that current radiation risk models fail to explain or even predict the many observations and should be abandoned.

3.2.1 Classes of hereditary defects

Different classes of hereditary defects are known from animal experiments and research in the fields of molecular genetics. The following table lists the defects that have been observed after exposure of humans to radiation (Schmitz-Feuerhake 2014):

(a) **Mendelian**

- Autosomal dominant: congenital abnormalities as syndactyly (fusion of fingers), brachydactyly (short fingers), polydactyly (more than 5 fingers or toes in each limb),
- Sex-linked: loss of females

(b) Chromosomal

- Aneuploidy (numerical chromosomal anomaly): Down's syndrome (trisomy 21),
- Structural anomalies: preimplantation loss, embryonal death, foetal abortions

(c) **Polygenic** cluster in families:

- Congenital abnormalities as neural tube defects, heart defects, pyloric stenosis, cleft lip with or without cleft palate, undescended testes
- Common disorders of adult life of varying severity, schizophrenia, multiple sclerosis, epilepsy, acute myocardial infarction, psychoses, diabetes mellitus, essential hypertension, asthma, peptic ulcer, rheumatoid arthritis.
- Cancer

The first group comprises defects that are caused by a single mutated gene and are dominantly or recessively inherited according to Mendel's Laws of Heredity.

The second group (b) lists defects accompanied by morphological mutation of chromosomes or a changed number of chromosomes. The most famous example for a changed number of chromosomes is Down's syndrome. Another concomitant of chromosomal mutation is foetal death.

The third group (c) includes diseases resulting from defects in several genes. In humans, effects of this type are often due to polygenic clusters in families. They may cause developmental disorder of new-born babies or disorders of adult life.

3.2.2 Congenital effects in regions affected by the Chernobyl accident

Many studies show the increase of foetal deaths, perinatal mortality and congenital malformations (CM) after the Chernobyl accident. However, the official view is summarised in a 2006 Joint News Release by WHO/IAEA/UNDP (World Health Organization, International Atomic Energy Agency and the United Nations Development Programs) which asserts "… Because of the relatively low doses to residents of contaminated territories, no evidence or likelihood of … effect on the number of stillbirths, adverse pregnancy outcomes, delivery complications or overall health of children … A modest but steady increase in reported congenital malformations … appears related to better reporting, not radiation" (Hoffman and Fleming 2005).

3.2.2.1 Re-evaluation of the EUROCAT Europe-wide Study

The study of Dolk and Nichols (1999) is widely cited as evidence for the lack of effect after the Chernobyl accident. The authors examined Down's syndrome, neural tube defects (NTD), microcephaly, hydrocephaly, anophthalmos and congenital cataract in 16 EUROCAT registers. There were 231,401 births in the areas in 1986. The 16 registries were divided into three groups of high (200 to 800 µSv), medium (97 to 190 µSv) and low (29 to 55 µSv) dose. Three comparison cohort periods were defined as E (conception May 1986), T (conception May 1986 to April 1987 contains E), and C (control: conception May 1987 to April 1989). The authors concluded "no evidence of a generalised detectable increase in the prevalence of congenital anomalies in the first month or first year following Chernobyl." Schmitz-Feuerhake et al. (2016) criticise the choice of these cohort periods for this study, because genetic damage to the germ cells from internal exposures will have continued well into the control period C and damage will have been cumulative. Schmitz-Feuerhake et al. (2016) have re-analysed the data of the EUROCAT-Study. A comparison of T vs. C cohorts showed a significant effect with odds ratio (OR) of 1.20[53]. But there was no increasing monotonic relation between assumed "dose" category and effect. However, for genetic damage, increasing dose will not linearly increase effects since at high doses there will be sterility or foetal loss. (Doll 1973)

3.2.2.2 Studies about congenital malformations in contaminated regions

Down´s syndrome increased in several contaminated European countries after the Chernobyl accident (Busby et al. 2009; Sperling et al. 2012). The geneticist Sperling, for example, registered in West Berlin, which was a kind of closed island at that time, a sharp and significant increase in cases exactly nine months after the accident.

Busby et al. (2009) published findings about foetal deaths, perinatal mortality and congenital malformations (CM) after the Chernobyl accident. These appeared not only in the area of the exploded reactor, but also in Turkey, Bulgaria, Croatia, and Germany. The results are not in line with the current

[53] (95% CI: 1.02, 1.4), p=0.014

ICRP/UNSCEAR assumptions. Evidence of increased CM rates after Chernobyl in Germany, Turkey, Croatia and Bulgaria was already described in several older studies. (Akar et. al. 1989; Akar 1994; Caglayan et al. 1989; Güvenc et al. 1993; Hoffmann 2001; Kruslin et al. 1998; Mocan et al. 1990; Moumdijev et al. 1992)

Belarus received most contamination from Chernobyl. A central registry for congenital malformations (CM) existed from 1979 and rates of CM before and after the Chernobyl accident could thus be compared. Comparison of legal abortuses in 1982 to 1985 and 1987 to 1994 showed combined CM increases of 81%, 49%, and 43% in regions of high (>555 kBq/m^2), medium (>37 kBq/m^2), and low (<37 kBq/m^2) contamination, the effect being significant at the 0.05 level in all three regions (Lazjuk et al. 1997). (The genetic origin is confirmed in those anomalies which are combined with a recognized mutation that is not present in either of the parents (Busby et al. 2009).)

Another study confirmed the CM excess finding 86% increase in 1987 to 1996 vs. 1982 to 1985 (high contamination) and 59% (control regions) ($p<0.05$). The study found significant excess chromosome aberrations of dicentric and centric rings rates in Gomel and Mogilev (>555 kBq/m^2) compared with a control region (<37 kBq/m^2) (Feshchenko et al. 2002).

Another study compared Gomel (high exposure) with Vitebsk (presumed low exposure) for mortality in children zero to four finding absolute CM rates of 4.1% vs. 3%, respectively (Bogdanovich 1997). Savchenko (1995) reported a frequency of CM in regions of Gomel between 1982 to 1985 and 1987 to 1989 ranging from 170% in Dobrush to 680% in Chechersk.

Petrova et al. (1997) compared two high and two low contaminated regions of Belarus for a number of indicators of pregnancy outcome and child health. For CM, before and after Chernobyl increases for all CM were: Gomel 150%, Mogilev 130%, Brest 120% and Vitebsk 110%, the rank of their contamination levels.

Kulakov et al. (1993) examined 688 pregnancies and 7,000 births in Chechersky (Gomel, Belarus) and Polissia (Kiev, Ukraine). Sharp reductions in birth rates in both regions after Chernobyl were ascribed partly to abortions. High perinatal mortality was ascribed partly to congenital malformations (CM). Incidence increased by a factor of two following the accident for congenital heart disease, esophageal atresia, anencephaly, hydrocephaly and multiple malformations. The total number of neonatal disorders increased in Polissia from 1983-1985 to 1986-1990 from 6.81 to 21.32 (313%) and in Chechersky from 5.15 to 10.49.

It is worth to add that congenital effects were also found near the former Soviet **nuclear test site in Kazakhstan**. Sviatova et al. (2001) studied congenital malformations (CM) in three generations of inhabitants, investigating births between 1967 and 1997. The authors found significantly increased rates of CM combined, including Down's syndrome, microcephaly and multiple malformations in the same individual.

3.2.2.3 Findings in Polissia

One of the populations most exposed to chronic low-dose radiation from Chernobyl lives in Polissia, the region representing the northern half of the Rivne Province in Ukraine. Malformations, as defined by international standards, noted among all 96,438 births in Rivne between 2000 and 2006, were analysed statistically. Contrasts of rates in Polissia compared with the rest of Rivne also were investigated. The overall rate of neural tube defects in Rivne is among the highest in Europe (22.2 per 10 000 live births). In Polissia, the overall rates of neural tube defects are even higher (27.0 vs 18.3, respectively. The malformation patterns observed suggest early disruptions of blastogenesis. (Wertelecki 2010)

Wertelecki et al. (2014) confirm and expand their previous studies (2000-2009) in Rivne that demonstrated elevated population-based rates of CM. Among 145,437 live births in Rivne between 2000 and 2009 are included 2,348 (1.61%) infants with anomalies noted before one year of age.

The native people of Polissia (Polishchuks) represent a population isolatedly surviving mostly by consumption of locally grown products, foods and fuels inherently contaminated by nuclides. Polishchuks continue to inhale and ingest nuclides and a growing proportion of pregnant Polishchuk women have themselves incorporated nuclides to which all of their conceived children are exposed prenatally. The large size and well defined nature of the Polishchuk population facilitates long term studies of the health and teratogenic impacts of protracted exposures to low levels of ionisation radiation.

According to Wertelicki et al. (2014), the results of this descriptive epidemiological study provide a starting point for prospective investigations of cause-effect associations. Elevated incorporated ionisation radiation levels in pregnant women are detected solely among those living in Polissia.[54] In the opinion of the authors, the concurrence of elevated rates of CM with elevated ionisation radiation levels in Polissia lends coherence to a hypothetical cause-effect association. Internal contamination was quantified for two groups, a high and lower dose group by whole body monitoring for caesium-137 (Cs-137). In addition, local produce was analysed for both Cs-137 and the DNA seeking Sr-90. The Sr-90/Cs-137 ratio was between 0.5 and two, so Sr-90 (with its DNA affinity and anomalous RBE) represented a significant internal exposure. (Wertelecki et al. 2014)

3.2.3 Congenital anomalies in the descendants of occupationally and medically exposed women

A German investigation of occupationally exposed females showed a 3.2-fold significant increase in congenital abnormalities, including malformations, in offspring. (Wiesel et al. 2011) The authors interpret the effect as generated *in utero* but do not prove such a connection. In the opinion of Schmitz-Feuerhake et al. (2016), this appears to be improbable given the short sensitive phase in pregnancy and the ban on pregnant females working in high risk environments.

The findings confirm early results in the Department of Medical Genetics of Montreal Children's Hospital where the genetic effects of diagnostic X-rays were investigated. (Cox 1964) The author observed the offspring of married mothers who had been treated in childhood for congenital hip dysplasia since 1925 and were X-rayed for several times in the pelvic region. The ovarian dose was estimated to be between 75 mSv and 200 mSv. In 201 living births of these females there were 15 individuals with severe malformations and other congenital distortions or Down's syndrome and 11 cases with other abnormalities (all congenital abnormalities 12.9%) while the control group (402) showed less than half of this rate. The latter was composed of married siblings of the probands who were not X-rayed.

3.2.4 Congenital anomalies in the descendants of occupationally exposed men

Studies in children of exposed men where the mothers were not exposed will show definite hereditary effects. Three studies of nuclear test veterans, for example, have shown large increases in congenital effects in children.

[54] Concerning plausibility, three teratogenic risks are of concern in Rivne: alcohol, genomic mutations, and IR. Alcohol teratogenesis is not prevalent in Polissia and genomic mutations are unlikely to cause the blastopathies observed.

Recently Busby and de Messieres (2014) examined descendants (**children and grandchildren**) of members of the British Nuclear Test Veteran Association (BNTVA). Based on 605 veteran children and 749 grandchildren compared with 311 control children and 408 control grandchildren there were significant excess levels of miscarriages, stillbirths, infant mortality and congenital illnesses in the veterans' children relative both to control children and expected numbers. There were 105 miscarriages in veteran's wives compared with 18 in controls (OR 2.75[55]). There were 16 stillbirths; three in controls (OR 2.70[56]). Perinatal mortality OR was 4.3[57] on 25 deaths in veteran children. 75 veteran children had congenital conditions vs. three control children (OR 9.77[58]) – these rates being also about eight times those expected on the basis of the UK EUROCAT data for 1980 to 2000. For grand-children similar levels of congenital illness were reported with 46 veteran grandchildren compared with three controls (OR 8.35[59]).

Roff (1999) carried out a questionnaire study of members of the British Nuclear Test Veteran Association (BNTVA) and reported excess rates of cardiovascular disorders, spina bifida, hydrocephalus and hip deformities.

Urquhart (1992) analysed data in children from 1,147 veteran families[60]. 233 out of them had illnesses or defects (cancer, malformations, mental retardation) that could have a genetic origin. The authors registered a 7:1 rate of abnormal children conceived before the tests vs. those conceived after the tests.

A compilation of studies concerning congenital anomalies in the descendants of occupationally exposed men is given in Schmitz-Feuerhake et al. (2016). The findings are listed in the following table.

Table 3: Congenital anomalies, especially malformations, in descendants (1st generation[a]) of occupationally exposed men

Cohort of fathers	Kind of defect	Dose	References
Radiologists USA 1951	Congenital malformations Increase 20%		Macht and Lawrence 1955
Workers of the Hanford Nuclear facility, USA	Neural tube defects significantly increased by 100%	In general < 100 mSv	Sever et al. 1988
Radiation workers at Sellafield nuclear reprocessing plant, UK	Stillbirths with neural tube defects significantly increased by 69% per 100 mSv	Mean 30 mSv	Parker et al. 1999
Radiographers in Jordan	Congenital anomalies significantly increased 10-fold	Mainly 10-250 mSv	Shakhatreh et al. 2001
Liquidators from Obninsk (Russia), 300 children	Congenital anomalies increased 1994-2002	10-250 mSv	Tsyb et al. 2004
Liquidators from Russia, Bryansk region	Congenital anomalies increased about 4-fold		Matveenko et al. 2006

[55] (95% CI: 1.56, 4.91), p<0.001

[56] (95% CI: 0.73, 11.72), p=0.13

[57] (95% CI: 1.22, 17.9), p=0.01

[58] (95% CI: 2.92, 39.3), p<0.001

[59] 95% CI,:2.48, 33.8), p<0.001

[60] The British carried out nuclear weapon tests and activities in Australia (Maralinga) and Christmas Island in the Pacific between 1952 and 1967. More than 20,000 young national servicemen and other military personnel were stationed at the test sites. The sites were contaminated with fission fallout and nanoparticles of uranium and plutonium from the weapons, tritium and carbon-14.

Cohort of fathers	Kind of defect	Dose	References
Liquidators from Russia, 2379 new-borns	Significant increase for: anencephaly 310%, spina bifida 316%, cleft lip/palate 170%, limb reduction 155%, multiple malformations 19%, all malformations 120%	5-250 mSv	Liaginskaia et al. 2009
British nuclear test veterans	All malformations Down´s syndrome OR 1.6 for early vs. later births	Less than 10 mSv but internal	Urquhart 1992
British nuclear test veterans	All congenital conditions increased	Less than 10 mSv but internal	Roff 1999
British nuclear test veterans case control/EUROCAT study	Miscarriages odds 2.7 Congenital conditions: children OR 9.8; grandchildren OR 8.3[a]	Less than 10 mSv but internal	Busby et al. 2014

[a] Mean first year committed effective doses are given by the authors or are calculated by Schmitz-Feuerhake et al. (2016)

3.2.5 Sex-ratio and X-linked lethal factors

Normally, it is not possible to study how many inseminated oocytes (zygotes) will be aborted after irradiation of the gonadal cells in humans. But it is observed that males who were exposed have fewer daughters than sons i.e., the male/female sex-ratio increases with dose. Thus, the sex-ratio is a very relevant parameter. It shows that genetic alterations are induced in the germ cells of males by very low doses, and it proves to be a sensitive indicator for exposures of the population.

Gene mutations may be responsible for the death of the zygote and will also occur in the sex chromosomes where they will predominantly affect the greater X-chromosome which can only be transmitted to a daughter. A dominant lethal factor will then lead to the death of the female zygote. Recessive lethal factors in the X-chromosome are much more frequent than dominant ones (Vogel et al. 1969). They affect only female births.

Scherb and Voigt studied different groups of inhabitants in a variety of countries after the Chernobyl accident for hereditary effects. They found radiation-induced foetal deaths and early mortality and Down's syndrome but also alterations of the birth sex-ratio. They also examined nuclear tests above ground which affected US inhabitants and those living near German and Swiss nuclear plants. Results showed significant reduction in the female birth rate in all sites. (Scherb and Voigt 2007, 2011)

A similar effect was detected in cardiologists, who undertook interventional angiographic procedures involving X-ray exposures. (Choi et al. 2007)

3.2.6 Discussion and conclusion of genetic and teratogenic effects

Effects in populations exposed to Chernobyl fallout are excluded by the official committees (in particular ICRP), which claim that doses are too low to generate statistically observable increases. This, however, is certainly wrong, because it is known from many studies of chromosome aberrations (e.g. Busby 2015b; Domracheva et al. 2000; Feshchenko et al. 2002; Schmitz-Feuerhake 2011), either that the doses calculated by UNSCEAR are much too low or that there is an enhanced radiobiological

effectiveness (RBE) in the type of internal exposures or chronic delivery received by the Chernobyl groups.

Malformations, cancers, and numerous other health effects in the children of populations who were exposed to low doses of ionising radiation have been unequivocally demonstrated in scientific investigations (Schmitz-Feuerhake et al. 2016).

3.2.6.1 External versus internal sources of radiation

In particular, the study of Wertelecki et al. (2014) indicates that internal exposure is an important factor for these observations.

According to the NAS (2014), scientific uncertainty exists about the differences in tissue effects and therefore the risks from external versus internal radiation sources. Although currently, for radiation-protection purposes, an assumption is made that the effect is the same, independent of the source location, it is understood that internal deposition of radionuclides is not as uniform as external irradiation is. Even at the level of whole tissues or major tissue components, estimation of average doses (or dose coefficients) from intakes of radionuclides requires highly complex biokinetic and dosimetric model calculations. Comparisons of risks derived from the ICRP dosimetric approach with those obtained from direct epidemiological observations in the few available situations, indicate that the discrepancies can vary from about a factor 2 in some cases to 10 or more in others. The need for improved biokinetic and dosimetric models is crucial for making progress with this scientific question (EC 2009).

However, despite this knowledge and the known uncertainties of internal doses, the doses upon which the ICRP risks are based, either from humans or mice, are external doses. There are significant issues regarding the equivalence for causing genetic damage of internal and external dose calculations (Busby 2013). Internal exposure to uranium by inhalation, for example, has been associated with significantly high genotoxicity resulting in anomalously high excess levels of chromosome damage and birth defects in a number of different groups (Busby 2015b). Uranium binds to DNA, this fact that has been known since the 1960s. (Huxley et al. 1961; Constantinescu and Hatieganu 1974; Nielsen et al. 1992)

In other words, the biological or genetic damage from unit internal dose e.g., from a radio-nuclide bound to DNA is far greater than for the same dose delivered externally. (Schmitz-Feuerhake et al. 2016)

3.2.6.2 Difference between acute and chronic exposition

When examining the risk of genetic damage by radiation it is very important to make a distinction between acute exposure to radiation in the event of environmental contamination and chronic exposition of occupationally exposed persons.

The assumption of the ICRP and other official bodies, who estimate the radiation risk to be very low, is based on the observation of Japanese survivors of the atomic bomb explosions and on studies investigating the descendants of parents who had to undergo a radiation therapy at a younger age (ICRP 2007). In both cases, however, there is only a short-time exposure to radiation. The point in time at which the preconceptional exposure to radiation occurs is of major importance for the mutation of parental germ cells. This is especially valid for spermatogenesis with a normal duration of approximately 86 days. The stages of spermatogenesis are many times more radiation-sensitive than stem cells and developed sperm cells. (Fritz-Niggli 1997)

The chronic radiation exposure of men results in permanent radiation of all stages of spermatogenesis. This explains the relatively high number of malformations and other congenital defects of the descendants of occupationally exposed men (see table 2). Also, the congenital effect in the contaminated regions affected by the Chernobyl accident are probably caused due to this reason. (z.B. Lazjuk et al. 1997, 2003; Scherb and Weigelt 2003, 2004; Schmitz-Feuerhake et al. 2016)

3.2.6.3 In-utero or genetic exposure effects

The question of germ cell damage in parents or in utero damage to development, though important, is not to answer yet. All the congenital malformations (CM) effects are caused by mutation of DNA whether in the parental germ cells and precursors or from implantation to birth. However, from the sex-ratio results it would seem that parental exposure is a dominant cause of radiation induced CM, concluded Schmitz-Feuerhake et al. (2016).

As men and women in contaminated areas have persistently been exposed to radiation, the genetic effects cannot be clearly distinguished from those resulting from in-utero exposure of embryos and foetuses. All in all, both in-utero and genetic effects are relevant for the underestimation of the radiation effects for descendants.

3.2.6.4 Discussion of the risk factor for heritable effects

For both radiation-induced cancer and heritable disease it is the probability of the occurrence of the effect, not its severity, that depends upon the dose. The general assumption for radiological protection is that the risk of these stochastic effects increases in the low dose range (doses below about 100 mSv) linearly with dose, with no threshold (LNT model). This dose-response model is generally known as 'linear-non-threshold' or LNT.

The ICRP risk model is based on two main ideas: absorbed-dose, which is average energy per unit mass of tissue, and the linear no threshold (LNT) response. As explained above, for internal exposure to substances like Sr-90 and uranium, which both have high affinity for DNA, the concept of dose is meaningless. For CM as an outcome, it is also clear that the LNT model is unsustainable (Doll 1973). Biological plausibility would predict an increase in damage and thus CM at very low dose, followed by a drop in CM due to failure to implant, early miscarriage, abortion. This would result in a saturation dose response in the lowest dose region. Only the survivors would make it to be registered as CM.

It is out of the scope of this report to develop appropriate risk factors. However, it should be mentioned that in Schmitz-Feuerhake et al. (2016) a new model for a risk factor is suggested:

The Chernobyl studies may be used to obtain an approximate risk factor for all CM. The excess relative risk (ERR) for all CM follows a specific shaped response and is about 0.5 per mSv at 1 mSv, falling to 0.1 per mSv at 10 mSv exposure and thereafter remaining roughly constant was found. This means that the background rate will double or triple up to 10 mSv exposure and thereafter fall. But it also results in a 50% excess risk at doses as low as 1 mSv. This ERR and dose response model accommodates all the observational data from Chernobyl. This model is for mixed internal and external exposure to fission product contamination doses as employed by UN agencies and may not necessarily apply to pure external exposures. (Schmidt-Feuerhake et al. 2016)

Genetically induced malformations, cancers, and numerous other health effects in the children of populations who were exposed to low doses of ionising radiation have been unequivocally demonstrated in scientific investigations. All in all, results show that current radiation risk models fail to predict or explain the many observations and should be abandoned. The experts suggest further research and analysis of previous data. (Schmitz-Feuerhake et al. 2016)

The behaviour of the international associations (IRCP, WHO) is irresponsible, because at present it is already clear that the radiation risk for future generations will be much higher than stated and the full extent cannot yet be predicted.

3.3 Non-cancer diseases

The ICRP states: “Whilst recognising the potential importance of […] observations on non-cancer diseases, the Commission judges that the data available do not allow for their inclusion in the estimation of detriment following radiation doses in the range up to around 100 mSv. This agrees with the conclusion of UNSCEAR (2008), which found little evidence of any excess risk below 0.5 Sv.” (ICRP 103 2007, p.56f.)

But some studies provide evidence of non-cancer diseases occurring also below 100 mSv.

Non-cancer diseases comprise a big group of diseases, such as meningioma and other benign tumour entities, cardiovascular, cerebrovascular, respiratory, gastrointestinal and endocrine disease, psychiatric conditions, as well as cataracts.

After Chernobyl, studies in Belarus showed increased morbidity in the cohort of Belarussian liquidators (Okeanov et al. 1996) for diseases of the endocrine system, the gastrointestinal tract, diseases in metabolism and immune system, diabetes mellitus, psychic disorders, cardiovascular diseases and cataracts. Also for Ukraine significant results have been found for the same diseases in the liquidators cohort with people having received more than 250 mGy. (Buzunov et al. 1996) And in Russia also significant higher non-cancer disease rates for liquidators with doses >50 mGy compared to Russian population have been found. (Ivanov 1996)

Despite of these results, the ICRP decided that “Risks of non-cancer disease at low doses remain most uncertain and no specific judgement is possible.” (ICRP 103 2007, p. 144). Even though at that time there also have been results from the LSS cohort for evidence of non-cancer disease mortality increases, the ICRP stated: “However, the Commission notes current uncertainties on the shape of the dose-response at low doses and that the LSS data are consistent both with there being no dose threshold for risks of disease mortality and with there being a dose threshold of around 0.5 Sv.” (ICRP 103 2007, p. 56)

Also in a new LSS study increases in non-cancer diseases have been found. Ozasa et al. (2012) observed an increased risk of diseases of the circulatory, respiratory and digestive systems. The overall ERR for non-cancer diseases was 0.13 per Gy[61], for circulatory disease 0.11[62], for respiratory disease 0.23[63] and for digestive disease 0.20[64]. But the authors did not assess them as causal relationships, further investigation were recommended. Nevertheless, for the German BUND this is critical because these non-cancer diseases have a far higher prevalence than cancer, therefore more people can be affected. (BUND 2016)

Although high doses of ionising radiation have long been linked to circulatory disease, evidence for an association at lower exposures remains controversial. Little at al. (2012) conducted a systematic review and meta-analysis on circulatory disease risks associated with moderate and low-level whole-body ionising radiation exposures. Radiation exposures had to be whole-body, with a cumulative mean dose of < 500 mSv, or at a low dose rate (< 10 mSv/day). Estimated excess population risks for all circulatory diseases combined ranged from 2.5%/Sv[65] for France to 8.5%/Sv[66] for Russia. The authors found an association between circulatory disease mortality and low and moderate doses of ionising radiation.

[61] (95% CI: 0.08, 0.18) p<0.001

[62] (95% CI: 0.05, 0.18). p<0.001

[63] (95% CI: 0.11, 0.36) p<0.001

[64] (95% CI: 0.05, 0.38) p=0.009

[65] (95% CI: 0.8, 4.2)

[66] (95% CI: 4.0, 13.0)

The estimates of population-based excess mortality risks for circulatory disease are similar to those for radiation-induced cancer. Moreover, if associations between low-level exposure to radiation and circulatory diseases reflect an underlying causal relationship that is linear at low doses, then the overall excess risk of mortality after exposure to low doses or low dose rates of radiation may be about twice that currently assumed based on estimated risks of mortality due to radiation-induced cancers alone. (Little et al. 2012)

A new study from Mämpel et al. (2015) analysed among other non-cancer effects studies on eye cataracts. Cataracts have been seen as deterministic effect, meaning that a threshold was assumed But the authors found a mean doubling dose for the eye lens of 209 mSv (without LSS cohort) – obviously under the threshold of 500 mSv used by ICRP. Cataracts should therefore be seen as stochastic effects with no threshold.

In conclusion it can be stated that non-cancer effects are wrongly neglected by ICRP and should be included in the radiation protection model.

3.4 Conclusions

In earlier studies the effects of low dose radiation have already been investigated, but newer studies of natural background, occupational exposure, Chernobyl consequences, and first effects from Fukushima can provide better information on effects of ionising radiation. The excess risks of low, protected doses have been found to be similar to those of higher doses.

Radiation protection has long been based mainly on the research of the survivors of the Japanese atomic bombs. From the so-called Lifespan Study (LSS) results health effect of ionising radiation for individuals who received doses mainly from 100 mSv upwards can be derived, this is more than what is typically defined as low doses. The survivors were exposed over a short time to penetrating high-energy gamma radiation. In the Ulm Meeting 2014 of independent experts (IPPNW 2014) the critique was formulated that radiobiological research has shown that such exposure is less damaging to tissue than chronic alpha or beta irradiation following the incorporation of nuclides, and chronic exposure to x-rays or gamma-rays from natural background or artificial sources at dose levels comparable to normal background. On the contrary, the ICRP argues that the mutagenicity of the Japanese bomb radiation has a two-fold higher risk than that from other sources. Therefore, in ICRP dose models a “dose and dose-rate effectiveness factor”, the so-called DDREF of 2, is used to reduce the calculated risk to 50%. This DDREF therefore is also included in every dose limit provided by EU legislation, and it is strongly disputed.

This is one question that is discussed in radiation research – how are the health effects of radiation patterns different to those which were received by the atomic bombs in Japan? In new studies this question can be better answered because there is strong evidence of excess relative risk (ERR) caused by low and very low doses of radiation that are protracting, meaning being received chronically. The ERR describes the risk of getting a disease that is excess to the risk of a comparable group of people and therefore can be assumed to be attributable to radiation. Some of the study results show an ERR that is statistically significant, others do not, but it has to be kept in mind that some of the diseases that are caused by radiation are rare diseases like leukaemia and a statistically significant increase is difficult to detect.

Due to the new LLS-study, the LNT model has been confirmed, meaning that the risk is proportional to the dose even in the very low dose range – only a dose of zero is a totally safe dose.

Also another open question in radiation research has been answered better by new studies, the question what types of diseases are caused by low-level radiation. Especially the CLL type of leukaemia was long believed to not be radiation induced, but now there is evidence for the contrary.

Genetic and teratogenic effects have been studied intensely. There is evidence for genetically induced malformations, cancers, and numerous other health effects in the children of father and/or mothers who were exposed to low doses of ionising radiation. Current radiation risk models fail to predict or explain the many observations and should be revised.

A pattern of epidemiological evidence world-wide now clearly indicates increased leukaemia risk near nuclear power plants (NPP). A recent published study reveals a highly statistically significant 37% increase in childhood leukaemia within 5 km of almost all NPPs in the UK, Germany, France and Switzerland. Thus, there is a very clear association between increased child leukaemia and proximity to NPPs. (Körblein et al. 2012)

Furthermore, recent studies concerning childhood cancer from natural background radiation and medical exposure indicate the high radio-sensitivity of children. Studies about leukaemia risk for unborn and very young children show significant increases in leukaemia risk for foetal exposure to the Chernobyl contamination.

In the light of the depicted studies there is considerable doubt whether the risk for unborn and very young children is not more than three times higher than the risk for the overall population. Especially for embryo/foetus this ICRP assumption seems to be insufficient.

Non-cancer diseases comprise a big group of diseases, among them cardiovascular diseases, diseases of the respiratory and the gastrointestinal tract, diabetes, cataracts etc. While the ICRP does not assume effects under a dose of 500 mSv, studies show that even at low dose ERR can be found – which is of special interest, because f.e. cardiovascular diseases have a high prevalence and therefore many people can be concerned. Cataracts were long seen as deterministic radiation effects, but new studies suggest that they are also stochastic effects – without a threshold.

Although there are numerous studies in the area of assessment of impacts of nuclear power plants on human health, it is still necessary to make follow-ups, especially to investigate radiation effects of normal operation of nuclear facilities in depth. Particularly in countries with many NPPs on operation and where the NPPs are situated in densely inhabited areas, it is necessary to try to arrange for independent studies or independent reviews of existing studies.

It is of uttermost importance that new insights in radiation effects will be considered in radiation protection law and measures.

4 Radiation protection in EU legislation: are new insights in radiation health effects considered?

Protection of humans and environment from adverse effects of ionising radiation is one key task of the Euratom Treaty. According to Art. 2 (b) the Community shall "establish uniform safety standards to protect the health of workers and of the general public and ensure that they are applied". (Euratom Treaty 2012) Such a basic safety standard has to include maximum permissible doses compatible with adequate safety and maximum permissible levels of exposure and contamination. (Art. 30) The Commission has to establish such basic standards after consulting a group of experts according to Art. 31.

The basic safety standard in force since 2014 is **Council Directive 2013/59/Euratom (BSS-Directive)**. A directive has to be implemented into national law by the member states, this has to be done until 06. Feb. 2018.

On this basic safety standard other EU legislation is based, among them **Council Regulation (Euratom) 2016/52** of 15 January 2016 on "Maximum permitted levels of radioactive contamination of food and feed following a nuclear accident".

In this chapter, these two legal documents will be analysed regarding their consideration of up-to-date insights in radiation health effects as discussed in the previous chapter.

4.1 Basic safety standard: Council Directive 2013/59/Euratom

Council Directive 2013/59/Euratom of 5 December 2013 laying down basic safety standards for protection against the dangers arising from exposure to ionising radiation, the so-called BSS-Directive, establishes uniform basic safety standards in the EU. According to Art. 2 it applies to any planned, existing or emergency exposure situation with ionising radiation, caused by artificial or natural sources of radiation (f.e. the operation of aircraft in relation to the exposure of crew). This includes also medical exposure and exposure from indoor radon. Out of scope are exposures to the natural level of radiation.

In Art. 5, the general principles of radiation protection are defined – justification, optimisation and dose limitation. Radiation protection should do more good than harm, individual exposure shall be kept as low as reasonably achievable, and in planned exposure situations (except medical exposure) the dose limits shall not be exceeded. Beside dose limits, reference levels are established in Art. 7.

In Art. 12, a **dose limit for members of the public** of 1 mSv/year (effective dose) is defined. This dose limit shall apply to the sum of the annual exposures of a member of the public from all authorized practices. Moreover, the limit of the equivalent dose for the lens of the eye shall be 15 mSv/a, and for the skin 50 mSv/a.

Beside dose limits, **reference levels** are established in Art. 7. In Annex I, these reference levels for effective dose are defined, they should be between 1 and 20 mSv/a for existing exposure situations and 20-100 mSv acute or per year for emergency exposure situation.

Dose limits for workers are 20 mSv in a single year, but up to 50 mSv can be allowed by national law, if the average dose over five consecutive years does not exceed 20 mSv. The equivalent dose for the lens of the eye shall not exceed 20 mSv in a single year or 100 mSv in any five consecutive years with a maximum of 50 mSv in a single year. For the equivalent skin dose and dose to extremities the limit is 500 mSv /a, each. **Pregnant workers** shall not get an equivalent dose to the unborn child exceeding 1 mSv during the remainder of the pregnancy. **Breastfeeding workers** shall not work in areas with

exposure. The dose limits for **apprentices and students up to age 18** is 6 mSv/a effective dose, 15 mSv/a equivalent does of the lens of the eye, and 150 mSv/a equivalent dose for the skin and extremities, each.

In several recitals of the BSS-Directive, the use of the ICRP approach (publications ICRP 103, 116 and 119) is declared as bases for the approach of the BSS-Directive.

The BSS-Directive has to be implemented into national law of the member states until 6 Feb 2018. The member states are allowed to introduce more severe protection measures if not stated else.

4.1.1 Discussion of specific problems in the BSS-Directive

Based on up-to-date scientific results discussed in chapter 3, among others, the following problems become apparent in the underlying assumptions of the ICRP and therefore also in the BSS-Directive.

The German environmental organisation BUND published in 2016 an extensive comment to the planned revision of the German radiation protection law implementing the BSS-Directive. (BUND 2016) In 2014, a summary of an expert meeting at Ulm, Germany, has been published by IPPNW. (IPPNW 2014) These two publications provide us with critiques of the current radiation protection approach. In the following chapter, discussion of problems take these two important publications into account.

4.1.1.1 Basing risk factors for low dose radiation only on the Japanese bomb survivors is outdated

As discussed at length in chapter 3, the ICRP relies mostly on study data from the LSS (Lifespan Study) of the survivors of Hiroshima and Nagasaki, even though the survivors were exposed to high radiation delivered in short time. This radiation pattern is not the same as f.e. after the accidents of Chernobyl and Fukushima, where people receive low protracting doses. But ICRP believes the atomic bomb radiation to be twofold stronger in effect than chronical long-term exposure. Therefore a factor DDREF (dose and dose-rate effectiveness factor) of 2 is used by ICRP. This decision is based upon dose-response features of experimental data, the results of the Lifespan Study, and the results of probabilistic uncertainty analysis conducted by others which are discussed in Annex A of ICRP publication 103. (ICRP 103 2007, p. 53). Using the DDREF of 2 results in two-times lower risk coefficients for description of health risks. This can no longer be considered as up-to-date, amongst others due to the INWORKS study (Richardson et al. 2015a) which has been able to proof that there is no reduction in excess relative risk for chronic low dose exposure compared to risk resulting from radiation of atomic bombs (see also table 1).

That low and very low doses of ionising radiation have measurable health effects was also shown in studies on the effects of natural background, studies on occupational exposure, studies on cohorts or cases with low doses in epidemiological studies after Chernobyl, and studies of effects of normal operation of NPPs, and studies of effects of medical use of ionising radiation.

Therefore it is no longer adequate to use proportionate relationships for the induction of low dose health effects from high dose which has been done up to now with the use of a DDREF of 2.

The DDREF has to be reduced to 1 due to this new scientific evidence. The WHO and the German Commission on Radiological Protection (SSK) already both recommend a DDREF of 1. (WHO 2013, p.32, SSK 2014)

4.1.1.2 Underestimation of genetic and teratogenic effects

The ICRP provides risk factors for heritable diseases. In publication 103 they are 0.002 per Sv for the whole population and 0.0041 for adult workers. (ICRP 103 2007, p. 143) In the former ICRP 60 recommendations these risk factors were 0.013 and 0.08, respectively.

As discussed in chapter 3, there is scientific evidence of genetic and teratogenic effects like genetically induced malformations, cancers, and numerous other health effects in the children of father and/or mothers who were exposed to low doses of ionising radiation. The current radiation risk model of ICRP fails to predict or explain the many observations and should be revised.

Moreover, in the BSS-Directive and accordingly in the national implementations a concretion of the aim of the BSS should be made by decidedly including the foetus, children and young adults in the scope of the radiation protection focus.

The working conditions of a pregnant worker, after declaration of pregnancy, should be such as to ensure that the additional dose to the embryo/foetus would not exceed about 1 mSv during the remainder of the pregnancy. (ICRP 103 2007, p. 85) This dose seems to be too high, esp. according to results of the effects of natural background on childhood cancer where external dose rates of ≥200 nSv/h (1.75 mSv/a) compared to those exposed to <100 nSv/h (0.88 mSv/a) already show effects. (Spycher et al. 2015)

4.1.1.3 Radiation effects from neutrons and protons are not considered sufficiently

It has generally been assumed that the neutron and gamma-ray absorbed doses in the data from the life span study (LSS) of the Japanese A-bomb survivors are too highly correlated for an independent separation of all solid cancer risks due to neutrons and due to gamma rays.

However, with the release of the most recent data for all solid cancer incidences and the increased statistical power over previous datasets, it is instructive to consider alternatives to the usual approaches. Walsh (2012) presented a simple evaluation of the degree of independent effects from gamma-ray and neutron absorbed doses on the all solid cancer risk with the hierarchical partitioning (HP) technique.

The average relative biological effectiveness of neutrons relative to gamma-rays, calculated directly from fit parameters to the all solid cancer ERR model with both colon absorbed dose co-variables, is 65[67]. Therefore, the determination of all solid cancer risks based on reference to the colon absorbed doses with a neutron weighting of 10 according to ICRP 103[68] may not be optimal, and this practice should be reviewed. Any future improvements in neutron relative biological effectiveness precision could have important public-health consequences, for example, for the types of proton therapy [69]that produce unwanted by-product neutron doses, but also for the transport/storage of high radioactive material and spent fuel.

[67] (95% CI: 11, 170)

[68] The biological effectiveness of neutrons incident on the human body is strongly dependent on the neutron energy< because of the variation of the secondary radiation with energy. In ICRP 103, the radiation weighting factor for neutrons is defined by a continuous function.

[69] Proton therapy is a type of radiation treatment that uses protons.

4.1.1.4 Equivalent doses to single organs

According to BUND (2016) dose limits for single organs additional should be included in the BSS-directive, especially for gonads. But also thyroid doses would be of relevance – so reference levels for food limits could be derived (see chapter 4.2.1.8)

The dose limit for skin in the BSS-Directive is set to 500 mSv for occupational exposure and 50 mSv for members of the public. New studies show skin cancer in workers and after X-ray diagnostics, mostly with doubling doses below 100 mSv (Mathews et al. 2013; Schmitz-Feuerhake 2014; Mämpel et al. 2015) Therefore in BUND (2016) a dose limit for skin dose for workers of 10 mSv/a and for members of the public of 1 mSv/a is recommended.

Also the equivalent dose for the eye lens is regarded as too high. Cataracts are now seen as stochastic effects. Children are more sensitive (Worgul et al. 1996a). Dose limit recommendations of BUND (2016) for the eye lens are 10 mSv/a for workers and 1 mSv for members of the public.

4.1.1.5 Dose limits and levels are too high

Not only the above mentioned equivalent doses for skin and eye lens are too high, also the dose limit and levels for the public.

The radiation protection model of ICRP has three phases (ICRP 103 2007, p 103ff.):

- Planned exposure situations: normal operation in nuclear facilities, decommissioning, waste management, medical exposure, exposure in emergency situations once the emergency has been brought under control
- Existing exposure situations (indoor radon, NORM)
- Emergency exposure situations

These situations are related to different dose limits and levels:

Table 4: Dose limits and reference levels of ICRP (ICRP 103 2007, table 5, 6)

	Dose limit/reference levels by ICRP 103
Planned exposure situations:	Members of the public: 1 mSv/a Workers: 20 mSv/a
Existing exposure situations:	>1-20 mSv/a
Emergency exposure situations (acute or annual doses):	>20-100 mSv/a or acute

If people are allowed to receive doses up to 20 or even 100 mSv, ICRP argues that there will be no significant increase in health effects or at least the benefit outweighs the negative effects. As discussed in chapter 3, some studies have already proven that also at doses lower that 100 mSv health excess risk has been found.

Therefore, these dose levels need to be reduced for all radiation protection situations. In case of an emergency, countries have defined their dose levels for start of emergency protection measures like iodine tablets or evacuation. These intervention levels are based on the BSS-standards and therefore on recommendations of ICRP and IAEA.

In Austria, a country without NPPs, some of the intervention levels are lower than in other countries, f.e. staying indoor for children and pregnant women is recommended if an effective dose of 1 mSv/7days is expected. The administration of iodine tablet for children should start if a thyroid dose of 10 mSv is expected. (IntV 2007) Protecting people's health has to be the priority under any circumstances, in particular of the descendants

4.1.1.6 Collective versus individual dose

The ICRP has the opinion that collective effective dose is not intended as a tool for epidemiological risk assessment, and it is inappropriate to use it in risk projections. The aggregation of very low individual doses over extended time periods is inappropriate, and in particular, the calculation of the number of cancer deaths based on collective effective doses from trivial individual doses should be avoided. (ICRP 103 2007, p. 11)

The experts of the Ulm Meeting (IPPNW 2014) have a completely different opinion. They argue that the concept of collective dose is the current evidence-bases school of scientific thought for quantitatively predicting stochastic radiation risk. Due to the LNT model also very low doses can have health effects (as was already proved by several studies, see chapter 3). Also in BUND (2016) it is recommended that besides the effective individual dose and single organ doses also the collective dose should be used. Levels for the collective dose should be determined esp. in planned radiation situations.

4.1.2 Recommendations for the interested public

The underlying assumptions of ICRP dose and risk calculation are outdated. It may not be possible to make amendments of the BSS-Directive itself (or even the underlying approach of ICPR), but the members states still have time until Feb 2018 to implement the BSS-Directive into national law. When a member state implements the BSS-Directive in its national law, it could introduce dose limits that are below the maximum dose limits. Many countries have not implemented the BSS-Directive yet, so there is still time left for the interested public to enter the debate.

Of uttermost importance is the reduction of dose limits and levels, and of inclusion of single organ doses for gonads and thyroid. The biological effectiveness of neutrons has to be considered.

The protection of the embryo/foetus and the genetic integrity of future generations have to be given highest priority. Radiation protection must therefore supplement adult based models and take into consideration the increased vulnerability of the embryo and the young child.

Radiation protection for female workers who are pregnant have to be strengthened.

The use of radiation for medical diagnostics is valuable for peoples' health, but nevertheless can also cause measurable health effects due to radiation. Reducing the use of diagnostic X-rays and nuclear medicine to the absolute necessary minimum is urgently recommended. Strict indication guidelines should be adhered to and only low-dose CT equipment used. Wherever possible, ultrasound or magnetic resonance imaging (MRI) should be preferred.

Transparency and public participation in radiation protection has to be promoted. We have to live with radiation risks from which we do not even benefit in some cases. The nuclear waste will accompany human society for many generations. Therefore the public should get the possibility to participate in the national BSS-implementing process.

Authorities should inform the public and radiation workers about uncertainties and gaps in existing radiation protection knowledge, and NGOs should call for this kind of information.

ICRP and the Article-31-Group of Experts are the only expert groups who can at the time-being influence radiation protection legislation, though the ICRP has no democratic legitimation. And the Article-31-Group which is staffed by the member states does also not consult with the public. It would be preferable to have also independently staffed expert groups with public participation who work more transparently.

4.2 Maximum permitted levels of radioactive contamination of food and feed following a nuclear accident

After the accident of Chernobyl in 1986 large amount of food and feed were contaminated by radioactive material. Not only Belarus, Ukraine and Russia were affected, but also many countries in Europe inside and outside the EC (European Communities at that time). The EC wanted to make sure that only such agricultural products were put on the EC-market that did not exceed a defined level of contamination. Therefore, three regulations for maximum levels in food and feed were established: Council Regulation (Euratom) No 3954/87, amended by Council Regulation 944/89 Euratom and Council Regulation 770/90/Euratom. These regulations allowed the European Commission to quickly adopt an implementing regulation in case of a radioactive contamination – for the first time such an implementing regulation was applied in 2011 after the nuclear accident in Fukushima.

After long years of revisioning these regulations, in February 2016 a new regulation has entered into force: Council Regulation Euratom 2016/52 for "laying down maximum permitted levels of radioactive contamination of food and feed following a nuclear accident or any other case of radiological emergency" (food level regulation). The food and feed levels in this new regulation are basically the same as in the old regulations from 1987. In the following tables, the food and feed levels are presented:

Table 5: Maximum permitted levels of food contamination (Council Regulation Euratom 2016/52, Annex I)

	Infant food** in Bq/kg	Dairy produce*** in Bq/kg	Other food except minor food in Bq/kg	Liquid food**** in Bq/kg	Minor food***** in Bq/kg
Sum of isotopes of strontium, notably Sr-90	75	125	750	125	7,500
Sum of isotopes of iodine, notably I-131	150	500	2,000	500	20,000
Sum of alpha-emitting isotopes of plutonium and transplutonium elements, notably Pu-239 and Am-241	1	20	80	20	800
Sum of all other nuclides of half-life greater than 10 days, notably Cs-134 and Cs-137*	400	1,000	1,250	1,000	12,500

* Carbon-14, tritium and potassium-40 are not included in this group.

**Infant food: food that is clearly identified and labelled as infant food, for consumption by infants in the first year of life

*** Dairy produce is defined as products falling within the following CN codes (Combined Nomenclature) including, where appropriate, any adjustments which might subsequently be made to them: 0401 and 0402 (except 0402 29 11).

These CN codes are (EC 2015):

- 0401: Milk and cream, not concentrated nor containing added sugar or other sweetening matter
- 0402: Milk and cream, concentrated or containing added sugar or other sweetening matter.
- 0402 29 11: Special milk, for infants, in hermetically sealed containers of a net content not exceeding 500 g, of a fat content, by weight, exceeding 10 % (EC 2016)

**** Liquid food is defined as products falling within heading 2009 and Chapter 22 of the Combined Nomenclature. Values are calculated taking into account consumption of tap-water and the same values could be applied to drinking water supplies at the discretion of competent authorities in Member States.

These CN codes are:

- Heading 2009: Fruit juices (including grape must) and vegetable juices, unfermented and not containing added spirit, whether or not containing added sugar or other sweetening matter (EC 2016)
- Chapter 22: Beverages, Spirits and Vinegar

*****Minor food is defined in Annex II in the food level regulation. This category includes several CN codes, among them spices, some vegetables like garlic or sweet potatoes, Jerusalem artichokes, caviar, cocoa, etc.

Table 6: Maximum permitted levels of radioactive contamination of feed (Council Regulation Euratom 2016/52, Annex III)

Feed for	Sum of Cs-137 and Cs-134 in Bq/kg
Pigs	1,250
Poultry, lambs, calves	2,500
Other	5,000

There are no feed levels defined for other nuclides that Cs-137 and Cs-134.

The food levels are based on a reference level of 1 mSv per year for the increment in individual effective dose by ingestion. (Council Regulation Euratom 2016/52, recital 3) The feed levels shall contribute to the observance of the maximum food levels. (ibid., Annex III)

An important assumption in the food level regulation is that **only 10% of food consumed annually will be contaminated**. (Council Regulation Euratom 2016/52, recital 3) The maximum levels should apply to food and feed originating in the European Union or imported from third countries. (ibid., recital 7)

Drinking water is regulated by Council Directive 2013/51/Euratom, but member states are free to use the maximum food levels for liquid food for water intended for human consumption. (ibid., recital 6)

In this Directive, derived activity concentrations up to 11 Bq/l are defined for several nuclides, which is only a fraction of the maximum food levels.

Member states can **derogate temporarily from the maximum permitted levels** for specified food or feed consumed on their territory. (Council Regulation Euratom 2016/52, recital 19)

4.2.1 Discussion of specific problems of the food level regulation

In radiation protection the overall aim is the protection of human health and the environment. Consumption of radioactive contaminated food results in an ingestion dose to the human body. In the first years after the accident of Chernobyl, ingestion contributed extensively to the total dose. For example: In Austria, 75% of total dose for adults were due to ingestion in the first year after Chernobyl (May 1986-May 1987). (BKA 1988, p. 232) The most important nuclides were Iodine-131, Caesium-134 and 137, Strontium-90 and plutonium isotopes, but also other nuclides contributed to contamination and doses.

Therefore it is very important for minimizing radiation health effects to consume as less contaminated food as possible. With these food level regulations of the EC it is in question if this aim can be reached at all. In this chapter, the problems are discussed that could undermine protecting people's health.

4.2.1.1 How much food can be contaminated after a nuclear accident?

In Council Regulation Euratom 2016/52 it is assumed that only 10% of food consumed annually will be contaminated after a nuclear accident. (Council Regulation Euratom 2016/52, recital 3) In the same recital, a publication of the European Commission (EC 1998) is mentioned as the scientific basis for the maximum food levels. This so-called Publication 105 has been written in 1998 by the Article-31-Group of Experts to review the rationale underlying Council Regulation Euratom 3954/87. The expert group introduced a factor f which reflects a judgement that the average annual concentration in food actually consumed is only a fraction of the food limit (level); the Art.-31-experts suggest f=0.1 (EC 1998, p. 6f). This means that after an accident, only 10% of all food consumed can be assumed as contaminated up to the maximum food levels. In the contrary, the other 90% have to be assumed as not contaminated.

This may be the case if a nuclear accident happens far from the borders of the EU, and if the EU does not receive much fallout. In the case of a nuclear accident in the EU or unfavourable meteorological conditions after an accident near the EU borders this is highly unlikely.

Even the Art.-31-Group recommends that this 10% may be valid for countries where food is mainly obtained from shops, but not for regions where food is less widely distributed – meaning regions with high degree of self-supply and local food consumption. (EC 1998, p. 7) In such cases, a higher value of f could be specified. As mentioned before, member states can derogate temporarily from the maximum permitted levels for specified food or feed consumed on their territory. (Council Regulation Euratom 2016/52, recital 19)

In their opinion from 2012, the Art.-31-Group invited the Commission "to seek their opinion as a matter of urgency in the event of an emergency exposure situation causing widespread contamination of food consumed in the EU so that the assumptions underlying the maximum permitted levels are no longer valid and exposures to members of the public may exceed a reference level on 1 mSv per year." (Article 31 Group of Experts 2012) This shows that the Art.-31-experts do not eliminate the possibility that a more widespread contamination can occur than they assumed. But it is questionable if in case of such a situation there will be enough time for adaption of the food level regulation. After the accident in Fukushima (11 March 2011) it took only two weeks before the first implementing regulation was in

force on 26 March 2011 (Commission Implementing Regulation EU 297/2011) From a radiation health perspective it would be much better if the maximum food levels would be prepared for the worst imaginable situation and be derestricted afterwards if need be, for example if the food supply cannot be guaranteed otherwise.

4.2.1.2 Outdated nutrition and food data

Nutrition and food data are not up-to-date. Regulation Euratom 2016/52 is based on food data from Publication 105 (EC 1998). These dietary data of Publication 105 again are based on an EC study of 1991. (CEC 1991) So **the underlying food and nutrition data are outdated for about a quarter of a century.**

In CEC (1991), dietary habits and consumption patterns have been analysed for the countries that have been EC member states by that time (Belgium, Luxembourg, Denmark, Germany, France, Greece, Ireland, Italy, Netherlands, Portugal, Spain, UK). Missing are food consumption data from 18 EU countries: Austria, Finland, Sweden, Bulgaria, Czech Republic, Estonia, Latvia, Lithuania, Hungary, Poland, Romania, Slovenia, Slovakia, Cyrus, Malta, Croatia. **Therefore out of 28 member states, only (old) food data of 10 are included** in the underlying data for the food level regulation.

An example for a change in dietary habits with possible large consequences on ingestion doses is the use of sweet potatoes, a vegetable that is getting increasingly popular in several European countries[70]. Even in countries like Austria, where there has been no agricultural production of sweet potatoes, since a few years this vegetable is produced in rising amounts. Nevertheless, in Council Regulation Euratom 2016/52 a "superfood" like the sweet potato is still listed as **minor food** and has therefore much higher food levels as other vegetables. When consuming only 2.4 kg of maximal contaminated sweet potatoes per year, an ingestion dose of 2.5 mSv results for adults. 2.4 kg responds to 200g/month. For children the resulting dose is even higher: consuming of one serving (120g[71]) of sweet potatoes per month that are contaminated to the maximum level results in an ingestion dose of 4 mSv. The same is true for Jerusalem artichokes and other vegetables on the minor food list that have become fashionable in the modern European kitchen.

New dietary data would be available for example at the European Food Safety Authority EFSA[72].

Another problem is the **category dairy produce**. In Council Regulation Euratom 2016/52 it is clearly defined that only milk and cream are included in this category. All other dairy produce (yoghurt, butter, cheese, buttermilk etc.) belongs to the category "other food". In the last years, there have been shifts of consumption patterns in the dairy produce section, f.e. in Austria less milk and more cheese are consumed[73] – and this results in changes in assessed ingestion doses because other maximum food levels are allowed for these two product groups.

An example: According to nutrition recommendations in Austria, an adult woman should eat 172 kg dairy produce per year. If it is assumed that all 172 kg are milk or cream, an effective ingestion dose of

[70] See f.e. https://www.theguardian.com/business/2015/oct/18/british-farmers-crack-the-sweet-potato, seen 23. Feb. 2017, http://www.freshplaza.de/artikel/6570/%C3%9Cbersicht-Weltmarkt-S%C3%BC%C3%9Fkartoffeln (Spain), seen 20. Feb. 2017, http://www.nachrichten.at/freizeit/essen_trinken/Rosaorange-Batata-aus-dem-Seewinkel;art115,1053103, seen 23. Feb. 2017

[71] Recommended vegetable consumption for 1 year old children 120g/d (Alexy et al. 2008)

[72] https://www.efsa.europa.eu/en/food-consumption/comprehensive-database

[73] Milk consumption per capita is slowly decreasing, cheese consumption has changed by 2.6% up to 19.4 kg/a (BMLFUW 2014)

5.6 mSv would result if every product is contaminated to the maximum. But if it is assumed that all 172 kg are butter, yoghurt, cheese etc., an effective ingestion dose of 17.4 mSv would result.

In the Publication 105 there is no such distinction for the dairy produce into two separate categories foreseen. The experts especially highlight the necessity of more restrictive food levels for dairy produce due to their large consumption quantities. (EC 1998, p. 6)

Most of the **food consumption data for 1 year old children** have been taken from Kaul (1988). Nevertheless, the value of 10 kg potatoes/year can neither be found in Kaul (1988) nor in CEC (1991). The Art.-31-Group describes the food consumption rate for 1 year old infants as about 50% of an adult's diet (CEC 1991, p. 318) – therefore, at least 17.5 kg potatoes per year should have been used if compared to the EU adult (lower level[74]). Even this small adaption of 7.5 kg could lead to an ingestion dose of 2.1 mSv in one year (under the assumption that all 17.5 kg of potatoes are contaminated).

The Art.-31-Group recommends in its Publication 105 that member states should establish regularly the typical dietary habits for different regions so that in the case of an accident no underestimations of actual consumption rates occur. (EC 1998, p. 7) This is really necessary and should be done by member states on a regular basis.

4.2.1.3 Ingestion doses could exceed 1 mSv/a

The food levels in Council Regulation Euratom 2016/52 are based on a reference level of 1 mSv/year for individual effective dose by ingestion according to recital 3. But will this dose level really not be exceeded when using the food levels in case of contamination?

In Publication 105 (EC 1998), the Art.-31-experts calculated doses for 1-year old children and for two types of adults with different dietary habits (so-called lower and higher level) for five different nuclides (C-14, Sr-90, I-131, Cs-137, Pu-239). When calculating the ingestion doses, factor f (see chapter 4.2.1.1) was set to 0.1 for all food except baby food. For liquid food the factor f of 0.01 was used.

In the following table, these calculated ingestion doses were summed up. The dose received by C-14 was not included in the sum because in Regulation Euratom 2016/52 it is also not included. In EC (1998) a wrong dose coefficient for Pu-239 was used, this was not corrected in the following table.

Table 7: Sum of effective ingestion dose for nuclides Sr-90, I-131, Cs-137 and Pu-239 (based on EC 1998, table 5)

Foodstuff	1 year old	EU adult lower level	EU adult higher level
Total (without C-14)	6.33 mSv	3.11 mSv	7.57 mSv (7.77 mSv)*

*If 600l liquid food was included with f=0.01, this would result in an additional dose of 0.2 mSv (total 7.77 mSv)

The resulting effective ingestion doses are by far higher than 1 mSv per year. And this is even the case if it is assumed that only 10% of food and 1% of liquid food are contaminated up to the maximum!

[74] In Publication 105 (European Commission 1998), the Art.-31-experts calculated doses for 1-year old children and for two types of adults with different dietary habits (so-called lower and higher level).

For comparison ingestion doses are calculated based on dietary recommendations data. In the following table it is calculated how much ingestion dose would result from the consumption of food that is contaminated up to the allowed level. This calculation is based on the example of Austria's nutrition recommendations. For minor food, an assumption is included additionally to dietary recommendations.

Table 8: Example Austria: Dose calculation based on nutrition recommendations for an adult woman and children (2-3 years); calculation based on assumption that the food is contaminated up to the allowed level the whole year long (own calculation based on data from IfEW and AGES 2012)

	Adult woman (18-64 years), recommended consumption in kg/a	Effective ingestion dose for adult woman in mSv/a	Child (2-3 years), recommended consumption in kg/a	Effective ingestion dose for child in mSv/a
Milk	73	2.4	46	3.3
Other food	605	61.2	280	76.7
Liquid food	548	17.8	297	21.4
Minor food*	2.4	2.5	1.5	4.0
Total		**83.9**		**105.4**
Total under the assumption that only 10% of food is contaminated (and 1% of liquid food)		**6.79**		**8.61**

*Sweet potato: assumed consumption per month 200g, for the child 120g; not part of recommendation but realistic assumption

Iodine-131 has a half-life of 8 days, therefore it could be assumed that food would not be contaminated up to maximum food level for the whole year. But in case of high contamination of milk, powdered milk could be produced and used later for regeneration into dairy produce resulting in a longer exposure phase – therefore for iodine also the maximum food levels for the whole year were used for calculation.

These calculated doses are in some aspects comparable to the dose calculations by EC (1998), but for children they exceed the doses from the Art.-31-Group. The nutrition recommendations are not conservative, therefore people could even get higher doses, especially if higher amounts of "minor food" like sweet potatoes are consumed, and bigger amounts of "other food". For example, in Austria meat consumption is recommended up to 19 kg per year for women, but actual consumption is 33 kg/a. (IfEW and AGES 2012) Consumption data for men are even higher.

4.2.1.4 Drinking water

Radioactive contamination of drinking water is regulated by Council Directive 2013/51/Euratom[75], but in case of a nuclear emergency member states are also free to use the maximum food levels for liquid food for water intended for human consumption. (Council Regulation Euratom 2016/52, recital 6)

In Council Directive 2013/51/Euratom no maximum food levels as such for drinking water are defined, but parametric values which have indicative function. This means if such a value is exceeded, radiological investigations are needed. 0.1 mSv indicative dose is defined as parametric value for radionuclides (resulting from artificial and natural radionuclides, but without radon and tritium). From this indicative dose, activity concentrations for radionuclides can be derived. Basic assumptions are that an adult is assumed to consume 730l drinking water/a; and the ingestion dose coefficients of ICRP are used.

In the following table, the maximum food levels and the derived concentration levels are compared.

Table 9: Drinking water: Comparison of maximum food levels according to Council Regulation Euratom 2016/52, and derived concentrations according to Directive 2013/51/Euratom, in Bq/l based on consumption of 730l/a (adult)

	Council Regulation Euratom 2016/52 Food level	Directive 2013/51/Euratom Derived concentration
Sr-90	125 Bq/l	4.9 Bq/l
I-131	500 Bq/l	6.2 Bq/l
Pu-239	20 Bq/l	0.7 Bq/l
Cs-137	1,000 Bq/l	11 Bq/l
Total effective ingestion dose for an adult per year after consuming 2l/d	17.8 mSv	0.43 mSv

As mentioned before, in Publication 105 the contamination rate of drinking water is assumed to be only 1%. (EC 1998, table 6, footnote e) If the calculated ingestion dose for adults of 17.8 mSv is divided by 100, it results in 0.18 mSv. But if a country uses (mainly) surface water as source for drinking water, in case of radioactive contamination it can be expected that a large part of the surface water is contaminated severely. According to a water quality report, six EU countries mostly rely on surface water, many other use surface water in combination with ground water for drinking water. (KWR 2011) Therefore, an assumption of a 1%-contamination rate could lead to massive underestimations of ingestion dose.

Moreover it is confusing that two regulations are valid at the same time which are based on two different doses (1 mSv versus 0.1 mSv). The 1 mSv according to Council Regulation Euratom 2016/52 can only be reached if a contamination rate of 1% is assumed (see above), and the 0.1 mSv of Council Directive 2013/51/Euratom can only be reached if just one of the nuclides is present at maximum activity concentration. To ensure the lowest possible contamination of humans with radionuclides of

[75] This directive should have been brought into force by member states until 28 Nov 2015; as of 20 Feb 2017, still three countries (Portugal, Spain, Belgium) have open infringement cases for failure to adopt and/or notify transposition of by Council Directive 2013/51/Euratom.

artificial origin[76], a more conservative approach should be preferred. Needed are maximum food levels for drinking water that do not lead to an ingestion dose of more than about 0.025 mSv for a sum of relevant nuclides (if 0.1 mSv for the maximum total ingestion dose is assumed (see chapter 4.2.1.6), about one quarter could be due to drinking water – see table 8).

In many European countries, people do not use tap water as drinking water but buy mineral water/water in bottles. In mineral water, radionuclides of natural origin can lead to a higher ingestion dose, which should be regulated by the BSS-Directive and national legislation to avoid unnecessary ingestion doses.

4.2.1.5 Missing: food levels for pregnant women, breastfeeding women, children above 1 year and young people

Besides children up to 1 year there are more risk groups that should be especially considered in radiation protection. These are pregnant women and their foetus, breastfeeding women, children above 1 year and young adults (see also chapter 4.1). But for these groups there are no food levels given.

4.2.1.6 Does a reference level of 1 mSv effective dose from ingestion provide enough protection?

The ICRP and the BSS-Directive defined a limit of effective dose of 1 mSv/a for members of the public. As discussed in chapter 4.1, this dose limit is too high and should be lowered. The current recommendation of the German BUND is to reduce the dose limit from 1 mSv by a factor of ten to 0.1 mSv[77] based on new insights in radiation health effects (see chapters 3 and 4.1). Also in 2010, the European Committee on Radiation Risk (ECRR) recommended "that the total maximum permissible annual dose limit to members of the public involving releases of anthropogenic isotopes or natural isotopes delivered in a novel fashion should be kept below 0.1 mSv as calculated using the ECRR model." (ECRR 2010, p. 181)

In the food level regulation, 1 mSv only from ingestion dose is used as reference level for the maximum food levels. In the first year after an accident the contribution of the ingestion dose can be very high. The share of caesium ingestion in the effective dose after Chernobyl was in average 54% for Europe, and the share of iodine ingestion in the thyroid dose was 50-100%. (Drozdovitch et al. 2007) In Austria, for example, 75% of the total dose for adults in the first year after Chernobyl resulted from ingestion. (BKA 1988) Therefore it is of uttermost importance to keep the ingestion dose as low as possible. A substantial reduction of the maximum food levels is necessary

If a 1 mSv dose limit from all pathways is set in the BSS Directive for members of the public, than the reference level for the ingestion pathway should be even lower than this limit, because after a nuclear accident there will also be contributions from other pathways, especially external radiation or inhalation, besides the exposures from medicine and radon. Following the argumentation of ECRR (2010) and the German BUND[78] for lowering the dose limit to 0.1 mSv/a, the reference level for the food levels should also be lowered to 0.1 mSv effective dose from ingestion.

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[76] In drinking water also naturally occurring radionuclides are of relevance, this is not discussed here.

[77] https://www.bund.net/themen/aktuelles/detail-aktuelles/news/neues-strahlenschutzgesetz-muss-gesundheit-klaren-vorrang-einraeumen/, seen 23 Feb. 2017

To keep under an ingestion dose of 0.1 mSv for all three groups of people (infants, adults lower level and adults higher level), the current food levels have to be reduced by a factor of 80 (multiplied with f = 0.0125).

Table 10: Comparison of effective ingestion doses for nuclides Sr-90, I-131, Cs-137 and Pu-239 (based on EC 1998, table 5), reduced by a factor of 80

Foodstuff	1 year old	EU adult lower level	EU adult higher level
Total (without C-14)	6,33 mSv	3,11 mSv	7.77 mSv
Reduced by a factor of 80	0.08 mSv	0.04 mSv	0.10 mSv

4.2.1.7 Confusing variety of legislation

Interesting in this context are food levels in use (or in former use) by other countries.

The first example is Austria – a country without NPPs in operation. The levels in the following table were valid before Austria's accession to the EU in 1995 (BKA 1991).

The European Communities also defined food levels after Chernobyl. In parallel to Council Regulation Euratom 2016/52, maximum food levels for imports of food from third countries after the Chernobyl accident were in force. They were first established by Council Regulation (EEC) No 1707/86 of 30 May 1986. Until March 2020, Council Regulation 1048/2009 is in force, still using the same food levels as the first regulation from 1986.

After the accident of Fukushima, the EU made an implementing regulation for food imports from Japan, based on the precursor Council Regulation (Euratom) No 3954/87. (Commission Implementing Regulation EU 297/2011) In this first implementing regulation the food levels of Council Regulation (Euratom) No 3954/87 were put into force for products imported from Japan. But this implementing regulation was adapted shortly after that to become better comparable to Japanese food levels. Food levels from implementing regulation 351/2011 are also given in the following table for comparison. (Commission Implementing Regulation EU No 351/2011)

Table 11: Comparison of food levels for selected food groups in Austria after Chernobyl before accession to the EU, in EC/EU after Chernobyl and Japan after Fukushima

	Nuclides	Council Regulation Euratom 2016/52; implementing regulation 297/2001 (first after Fukushima)	EU: Second Implementing regulation 351/2011 after Fukushima	Japan 2011 after Fukushima	Austria until accession to EU (BKA 1991)	EC/EU for food imports affected by Chernobyl Council Regulation 1048/2009
Infant food	Caesium	400	200		11.1	370
	Iodine	150	100			
Milk	Caesium	1,000	200	200	185	370
	Iodine	500	300	300	185	
Vegetables	Caesium	1,250	500	500	111	600
	Iodine	2,000	2,000	2,000	74	
Drinking water	Caesium	1,000	200	200	1.85	
	Iodine	500	300	300 (100 for infants, pregnant and breastfeeding women)	3.7	

The maximum food levels in these legislation differ very much and cause confusion for consumers. In the case of Fukushima it is especially not understandable why in the very first phase the high food levels of the EU were used even though Japan – also a country with a big nuclear industry – has lower levels. Only after protests these levels were lowered. Also the Art.-31-Group recognizes that food levels have to be lowered if the "public and political understanding is that any level is a borderline between safe and unsafe". (Article 31 Group of Experts 2011) Only after the experts recognized that the food levels in Japan were lower, they agreed to lower the food levels in case of Fukushima imports to match with Japanese levels.

4.2.1.8 Missing: thyroid dose

Especially after Chernobyl it became obvious that the risk for health effects to the thyroid was higher than expected. Therefore not only the effective dose but also the thyroid dose (equivalent dose) should be used as a basis for the maximum food levels of iodine isotopes.

When using the assumptions of the food level regulation, the following thyroid dose could result from the consumption of 35 kg baby food in half a year (EC 1998, table 6 footnote d), under the assumption that 50% of it is contaminated up to the maximum food level: An infant would get a thyroid dose from ingestion of 9.8 mSv (mainly from iodine).

For comparison: In the German Radiation Protection VO (StrlSchV 2001, § 47), a threshold for thyroid dose resulting from nuclear installations during normal operation is defined with 0.9 mSv/year.

Especially for all risk groups (infants, pregnant and breastfeeding women, foetus, children and adolescents) it would be necessary to re-define the maximum food levels to include a reference dose for the thyroid.

4.2.1.9 Missing nuclides

During nuclear accidents many nuclides can be emitted, among them C-14 and H-3 (tritium). These nuclides have not been taken into consideration in the food level regulation, even though the Art.-31-experts have recommended to include at least C-14 and have made calculations for it in Publication 105 (EC 1998). Both nuclides of natural origin are in the upper atmosphere, but they can also be produced by normal operation in nuclear facilities, nuclear bomb fallout and accidents. For the health effects of tritium see Bertell (2005), for C-14 see the website of IRSN[79].

Also the maximum permitted levels for feed are not complete, because only the contamination with caesium is regulated. But especially iodine is important in the first phase after an accident. Green fodder can be severely contaminated from iodine fallout. Iodine levels for feed should be added.

4.2.2 Excursus: Food levels and agricultural countermeasures in case of nuclear emergencies

In Austria and Germany a catalogue of agricultural countermeasures in case of a severe nuclear accident defines decision bases for the start of certain agricultural countermeasures in the pre-release phase of a nuclear accident. **If the following values are exceeded, food products could be**

[79] http://www.irsn.fr/EN/Research/publications-documentation/radionuclides-sheets/environment/Pages/carbon14-environment.aspx

contaminated above the maximum food levels of Regulation Euratom 2016/52. (BMLFUW 2014b, p. 29)

Soil contamination:

- Iodine: 700 Bq/m^2
- Caesium: 650 Bq/m^2

Example: Environmental Impact Assessment (EIA) Bohunice III (WUA et al 2015): Data from the Slovak authorities showed that in case of a severe accident a small part of Austrian territory could be contaminated with about 2,460 Bq Cs-137/m^2 and 10,000 Bq I-131/m^2.

According to the Austrian catalogue, the following agricultural measurements should start immediately if soil contaminations above the above mentioned values are expected:

- Immediate harvesting of marketable products
- Putting livestock into stables

Both measures will lead to a variety of consequences for farmers and consumers, most of them can be argued to cause negative impacts.

In future EIA procedures neighbouring countries should control contamination data in case of nuclear accidents to review if its food production could be impacted with food reaching the maximum food levels according to Regulation Euratom 2016/52.

4.2.3 Food contamination: conclusions and recommendations

The maximum permitted food levels in Council Regulation Euratom 2016/52 are too high due to the following facts:

For dose calculations in the food level regulation an assumption is used that only 10% of all food is contaminated up to the maximum, and 1% of liquid food, respectively. This will not be true in a worst case of a severe nuclear accident in one of the EU member states and under unfavourable meteorological conditions. Therefore, the maximum food levels should be conservatively calculated without using these two factors.

When the assessment of the Art.-31-Group of Experts in Publication 105 (EC 1998) is recalculated, an effective ingestion dose level of 1 mSv will be exceeded for infants and adults using the assumption that in one year only food is consumed of which 10% (1% for liquids) is contaminated up to the maximum permitted level. This recalculation results in 3.1-7.8 mSv instead of 1 mSv (see chapter 4.2.1.3)

The underlying data on dietary habits and food consumption are outdated by more than 25 years. Moreover, for only 10 EU member states out of 28, food data have been researched and used in calculations. Dietary habits have changed in the meantime, this can lead to much higher ingestion dose than assumed in the food level regulation. For example, if 200g (adult) or 120g (child) of the new “Superfood” sweet potatoes are consumed per month, an ingestion dose of 2.5 mSv (adult) and 4 mSv (child) would result, because sweet potatoes are classified as minor food and therefore have a very high food level.

Moreover, 1 mSv as reference dose for the effective ingestion dose is as such too high. Following recommendations by the ECRR (ECRR 2010) and the German BUND[80] for the reduction of the dose limit

[80] https://www.bund.net/themen/aktuelles/detail-aktuelles/news/neues-strahlenschutzgesetz-muss-gesundheit-klaren-vorrang-einraeumen/, seen 23 Feb. 2017

in the BSS-Directive from 1 mSv to 0.1 mSv/year for members of the public, such a reduction can also be demanded for the reference ingestion dose underlying the maximum food levels. Taking into account that the ingestion dose contributes to a large extent to the total dose, the reference dose in the food level regulation should also be reduced substantially to 0.1 mSv.

To reach an ingestion dose of 0.1 mSv for all three groups of people (infants, adults lower level and adults higher level), the food levels have to be reduced by a factor of 80.

Such lower food levels would provide better protection than the food levels from the recent Euratom regulation.

Additional necessary changes in the food level regulation:

1. The food category ”dairy produce” should be defined broader by including all dairy produce and not only milk and cream. Milk products can be as highly contaminated as milk itself.
2. The food levels for drinking water should be regulated more precisely – are they obligatory for all member states or not? Otherwise this could lead to different food levels for drinking water in different EU countries.
3. The missing nuclides C-14 and H-3 should be included in the food levels.
4. For feed, levels for other nuclides, especially for the iodine group, should be introduced.
5. The food levels should be adapted by underlying also a reference dose for the thyroid dose.

The Art.-31-Group recommends in its Publication 105 that member states should establish regularly the typical dietary habits for different regions so that in the case of an accident no underestimations of actual consumptions rate occur (European Commission 1998, p. 7) This recommendation is very important. The interested public should ensure that member states have their updated dietary data prepared so that on the occasion of implementing a food level regulation they can derogate from the food levels and introduce food levels that are best for ensuring their people’s health.

In case of a nuclear accident and no changes in the existing food level regulation, people should be advised to abstain from certain food products (like fresh milk and dairy produce and fresh vegetables) for a certain period of time. Independent experts and NGOs should be prepared to inform people if need be. Even if it can be assumed that authorities conduct food and feed measurements properly, independent laboratories for food control are very valuable as we have seen after Chernobyl, where people can measure their food products very cheaply and get independent information on food levels.

As a preventive measure, in EIA procedures assessment of severe accidents should also include soil contamination data and not only assessment of doses. With these contamination data, the need for agricultural countermeasures in a possibly concerned region can be evaluated so that the maximum food levels will not be reached.

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5.2 Glossary

Absorbed dose (D)	The absorbed dose is the energy absorbed per unit mass. The unit of the absorbed dose is Gray (Gy), 1 Gy = 1 Joule/kg.
Absolute risk (AR)	Absolute risk = incidence or prevalence Incidence = number of new cases of a disease occurring in a specified time period divided by the number of individuals at risk of developing the disease during the same time Prevalence = total number of affected individuals in a population at a specified time period divided by the number of individuals in the population at the time
Case-control study	The second best type of epidemiological study, of the type analytical study. Each case (a person with the disease) is compared to a comparable person without the same disease (control-person). It is analysed if the exposure has been different. Results are given as odds ratios (OR).
CI	Confidence interval: In statistics, a confidence interval is a range of values so defined that there is a specified probability (e.g. 90%) that the value of a parameter lies within this interval
Cohort study	Cohort studies are the most reliable epidemiological studies, of the type analytical study. A cohort is a defined group of people who have been exposed, and their developing a disease over time is compared to a so-called control-group, which is another cohort who has not been exposed. A cohort study starts before the analysed disease occurs for the first time. the cohort and the control-group should be comparable except for the exposure. Results are presented as relative risk (RR or ERR).
Committed effective dose, committed equivalent dose	Dose commitment means that the effect of radiation is integrated over a time interval. For children, committed doses are calculated up to the age of 70, for adults a period of 50 years following the contamination is used. For each year in this time interval, equivalent or effective doses are calculated and summed up.
Confounder	A confounder is another variable which can distort the effect or association between an exposure and outcome, f.e. smoking is a confounder for determination of lung cancer caused by radiation.
DDREF	Dose and dose-rate effectiveness factor; a factor introduced by ICRP that generalises the suspected lower biological effectiveness of low dose radiation exposures as compared with exposures at high doses and high dose rates. ICRP uses a DDREF of 2.
Dose coefficient	Sv/Bq, based on ICRP 119 (2012)
EAR, excess absolute risk	The attributable, additive risk
Ecological study	The third-best type of epidemiological study, of the type descriptive study. They are not based on individual but on collective dose-

	response-relationships. Results are given in number of persons having the disease per 100,000 persons in a given area.
Effective dose	The effective dose (E) is the sum of weighted equivalent doses in all tissues or organs of the body from internal and external exposure. For this purpose, the equivalent doses are multiplied with tissue weighing factors (w_T). The unit of the effective dose is the Sievert (Sv). $E = \sum_T w_T \sum_R w_R D_{T,R}$ or $E = \sum_T w_T H_T$
Equivalent dose	The equivalent dose (H_T) is used to assess how much biological damage is expected from the absorbed dose in a tissue or an organ (T). For calculation, the absorbed dose is multiplied with the radiation weighing factor (R). For different types of radiation different factors R are used. R is highest for alpha radiation and lowest for gamma radiation, depending on their possible biological damage. The equivalent dose can be calculated for single tissues or organs; if these are summarized, the total equivalent dose is resulting. The unit of equivalent dose is the Sievert (Sv). $H_T = \sum_R w_R D_{T,R}$
Epidemiology	Epidemiology is the study of health effects in specified populations. There are several types of epidemiological studies, among them case-control-studies, cohort studies and ecological studies.
ERR = excess relative risk, also named attributable risk	ERR is an epidemiological risk measure that quantifies how much the level of risk among persons with a given level of exposure exceeds the risk of non-exposed persons; difference of incidence rates between exposed and non-exposed individuals
Gray, Gy	Unit of absorbed radiation, defined as the absorption of one joule of radiation energy per kilogram of matter 1 Gy = 1 J kg^{-1}
Incidence ratio	Also called absolute risk, cumulative incidence; number of new cases of the disease per year and 100,000 people
LSS, Lifespan Study	Long-term study on health effects on survivors of the atomic bombs in Hiroshima and Nagasaki
Nested case-control study	This is a variation of a case-control study in which only a subset of controls from the cohort are compared to the incident cases. The nested case control model is generally more efficient than a case-cohort design.
Nominal risk coefficient	Sex-averaged and age-at-exposure-averaged lifetime risk estimates for a representative population.
OR	Odds ratio; OR is the ratio of the chance (not the probability) for the ex-posed person (case) to develop a disease in comparison to the unexposed person (control-case). Only for rare diseases (such as leukaemia), OR is about the same as RR. If OR =1, both persons have

	the same chance to develop the disease, OR =2 means that the exposed person has a two-fold chance to get the disease compared to the non-exposed person.
p	The p-value determines the significance of the results. It is a number between 0 and 1. A result is statistically significant if $p < 0.05$.
Prevalence	Proportion of surveyed population having developed the disease
Radiation detriment	A concept used to quantify the harmful health effects of radiation exposure in different parts of the body. It is defined by the Commission as a function of several factors, including incidence of radiation-related cancer or heritable effects, lethality of these conditions, quality of life, and years of life lost owing to these conditions.
Reference level	A reference level is not a dose limit, but represents a dose above which it is strongly recommended to reduce contamination
RR	Relative risk or risk ratio; RR is the ratio of the probability of occurrence of a disease among the exposed group to that among the unexposed group. RR is the result of a cohort study. Example: RR = 1 means that the risk for the disease does not depend on exposure. RR = 2 means that the exposed cohort will twice as likely develop the disease than the non-exposed group. RR = 0.1 means that the surveyed exposure is beneficial for people's health.
Standardization	Real life groups of people (f.e. inhabitants of different districts) are not comparable as such due to different age structures. For such groups it can be calculated how many cases of a disease would occur if the groups would be similar in age-structure, this is called age standardization or age adjustment.
Sievert, Sv	Unit of radiation dose

5.3 Abbreviations

For more explanations see the glossary.

AR	Absolute risk
Bq	Becquerel
BSS	Basic safety standard
CI	Confidence interval
CLL	Chronic lymphoblastic leukaemia
CM	Congenital malformations
CT	Computer tomography
DDREF	Dose and dose-rate effectiveness factor
EAR	Excess absolute risk
EC	European Commission
ERR	Excess relative risk
EU	European Union
Gy	Gray
IAEA	International Atomic Energy Agency
ICRP	International Commission on Radiological Protection
IPPNW	International Physicists Against Nuclear War
LNT	Linear-no-threshold
LSS	Lifespan Study
m	milli
NGO	Non-governmental organization
OR	Odds ratio
RBM	Red bone marrow dose
RR	Relative risk
Sv	Sievert
TORCH	The other report on Chernobyl
UNSCEAR	United Nations Scientific Committee on the Effects of Atomic Radiation
WHO	World Health Organization

5.4 List of Tables and Figures