Although a larger number of radionuclides are considered in the source term description and in the assumed relative isotopic composition of deposit, the dominant contributors to the exposure from the Fukushima Daiichi NPP accident were iodine-131 (^{131}I), in the early period after the accident, and caesium-134 (^{134}Cs) and caesium-137 (^{137}Cs) later on (see Box 2).

From the relative isotopic composition in Table 1 it can be seen that about the same amount of ^{134}Cs and ^{137}Cs radionuclides was released in the Fukushima Daiichi NPP accident (*9*). In contrast, there was twice as much ^{137}Cs compared with ^{134}Cs in the Chernobyl accident. As these two radioisotopes of caesium, ^{134}Cs and ^{137}Cs, have different physical half-lives (i.e. 2 years and 30 years, respectively), the fraction of lifetime dose to be delivered beyond the first year after the accident in Fukushima will be lower than in the Chernobyl accident.

Most releases of noble gases from the Fukushima site to the environment would have occurred early. Xenon (^{133}Xe), a noble gas that is released during nuclear accidents, contributes to external exposure from cloudshine, while its contribution to inhalation doses is negligible. Since it is a gas, it does not deposit on the ground; hence it is not listed in Table 1.

2.2 Identification of health hazards due to ionizing radiation

The potential hazard from radionuclides can be determined based on previous experimental and epidemiological studies. Radiation damage to tissue or organs has been shown to depend on the type of radiation, the sensitivity of different tissues and organs, the dose and the dose rate. This section introduces the adverse health effects of ionizing radiation, while Chapter 3 describes their dose-response relationship.

Adverse health effects of ionizing radiation result from two distinct mechanisms (*12*):

- cell killing, which may cause functional impairment of the exposed tissue or organ only if a sufficient number of cells are affected;
- non-lethal changes in molecules of a single cell, most commonly in the DNA molecule, which may result in an increased risk of disease long after exposure.

Box 2. Properties of the main radionuclides released

^{131}I emits beta and gamma radiation and has a half-life of 8 days. Due to its short half-life, ^{131}I is most relevant during the first weeks after a nuclear accident. ^{131}I has the potential to cause exposure by external radiation from the radioactive cloud (cloudshine) and deposits on the ground (groundshine). It is volatile and can be inhaled. It can also be ingested because it readily enters the food chain. Similar to stable iodine, ^{131}I is actively taken up by the thyroid gland. The fetal thyroid gland concentrates iodine by 11–12 weeks' gestation so if radioactive iodine enters the mother's blood stream after that period it can be taken up also by the fetal thyroid gland.

Beta and gamma radiation are emitted in the radioactive decay chain of ^{134}Cs and ^{137}Cs. ^{134}Cs has a half-life of 2.1 years and ^{137}Cs has a half-life of 30 years. They become the most relevant radioactive hazard after the first weeks of a nuclear accident. Once caesium enters the bloodstream, it distributes relatively homogenously throughout human visceral and muscle tissues and hence causes radiation exposure to the entire body (*11*).

The first type of effect has long been considered to be entirely determined by the initial interaction of radiation with tissues and organs, classically called "deterministic effects". It is now recognized that some of these effects are not determined solely at the time of irradiation, but can be modified later. It is therefore more appropriate to refer to them as tissue reactions. However, because the term "deterministic" is still often found in the literature, it is used in this report. Deterministic effects are mostly observed after exposure to moderate or high radiation doses.

The second type of effect occurs through a random process that is not entirely determined at the time of irradiation. These are called "stochastic effects" to reflect their probabilistic nature. Stochastic effects include cancer and heritable effects. At low doses, radiation risks are primarily related to stochastic effects, in particular, cancer, rather than the deterministic effects characteristic of higher-dose exposure.

2.2.1 Carcinogenic effects

About one fifth of people worldwide and one third of people in many industrialized countries are diagnosed with cancer during their lifetime (*13,14*). Radiation can induce cancers that are indistinguishable from cancers resulting from other causes. The International Agency for Research on Cancer (IARC) has categorized all types of ionizing radiation as carcinogenic to humans (*15*), on the basis of experimental studies on cells, tissues and animals, as well as through epidemiological research on people exposed to radiation. Most population-based cancer risk estimates come primarily from the Japanese atomic bomb survivor Life Span Study (LSS) cohort data (see Box 3). In addition to the LSS, there are several other sources of radiation exposure from which useful epidemiological data are available, including past accidents (e.g. the 1986 Chernobyl nuclear accident), medical exposures (diagnostic and therapeutic applications) and environmental exposures.

Increased radiation-related risks have been observed in the LSS for leukaemia (*17*), and for a large number of solid cancer sites, including oral cavity, bone, oesophagus, stomach, colon, liver, lung, non-melanoma skin cancer, female breast, ovary, urinary bladder, brain/central nervous system and thyroid (*16*).

Current knowledge allows for the estimation of the magnitude of the risks and their variation by cancer site, sex, age-at-exposure, attained age and time since exposure. A summary of the current knowledge on radiation carcinogenesis is provided below for the individual cancer sites that have been evaluated in the present HRA (i.e. leukaemia, thyroid cancer and breast cancer). The HRA Expert Group chose to consider these separately in this assessment because the influence of early age-at-exposure is particularly relevant for the three cancer sites that are also most radiosensitive. The individual consideration of thyroid cancer risks was also related to the release of radioactive iodine and its influence on thyroid cancer risk. The dose-response relationship for each of these sites is further discussed in Chapter 3. Based on existing evidence, it was considered in this assessment that the increase in lifetime cancer risk following in utero exposure is similar to that from exposure in early childhood (see below and section 6.3.3).

Leukaemia

Leukaemia represents a number of proliferative diseases arising in white blood cells, and can be classified into four main types: acute lymphoblastic leukaemia (ALL), chronic

lymphocytic leukaemia (CLL), acute myeloid leukaemia (AML) and chronic myeloid leukaemia (CML). Leukaemia has often been the first malignancy to show an increase after radiation exposure, with excess incidence appearing 2–5 years after exposure. Among atomic bomb survivors, there was an indication of increased risk by the late 1940s, and the excess was confirmed when the follow-up of the LSS cohort began in 1950 (*18*). Clear evidence of the association between radiation exposure and ALL, AML and CML was found, especially in people exposed at young ages, but without evidence for increased risk of CLL. The risk of radiation-induced leukaemia was greatest 5–10 years after exposure[1] and declined gradually thereafter over the next 50 years (*5*). Findings from recent risk analyses, however, suggest the possible persistence of increased AML risk even after 1990 (*19*).

Thyroid cancer

The incidence of thyroid cancer has been increasing over the last few decades, partly on account of improved detection. A pooled analysis of several studies of thyroid irradiation confirmed the association between radiation exposure and increased thyroid cancer

1. The temporal pattern of risk 5–10 years after exposure is seen primarily in children.

Box 3. Study cohorts of the atomic bomb survivors of Hiroshima and Nagasaki

Scientists from the Atomic Bomb Casualty Commission (ABCC) set up in 1947 and its successor, the Radiation Effects Research Foundation (RERF), have assessed the long-term health effects of radiation exposure in the survivors of the atomic bombings of Hiroshima and Nagasaki, as well as in their offspring. The Life Span Study (LSS) is an epidemiological study of cancer mortality and incidence in a cohort of about 120 000 individuals, including a defined subset that underwent additional health surveillance (Adult Health Study, AHS). Other study cohorts were added later, including individuals exposed *in utero* and children conceived after the bombings. In total, approximately 200 000 individuals, 40% of whom are still alive today, were identified and followed up in these different study cohorts (*16*).

The systematic follow-up of the LSS cohort began in 1950, including survivors who were within 2.5 km of the hypocenters at the time of the bombings and a similar-sized sample of survivors who were between 3 and 10 km from the hypocenters whose radiation doses were negligible. In the context of radiation epidemiology, the cohort of the atomic bomb survivors from Hiroshima and Nagasaki is unique, owing to:

- the large number of members not exposed for any medical reason;
- the long follow-up period of more than 50 years;
- a composition that includes males and females, children and adults;
- whole-body exposures (which are more typical for radiation protection situations than the partial-body exposures associated with many medically exposed cohorts);
- substantial effort expended on reconstructing tissue-specific doses (DS02);
- a large dose range, from levels comparable to the natural background to lethal levels;
- an internal control group with negligible doses, i.e. those who survived at considerable distance (>3 km) from the hypocenter;
- mortality data that are virtually complete up to now[1], high-quality tumour registries, and less potential bias from confounding than other exposed cohorts.

1. LSS cancer burden from early childhood exposure will continue to be expressed for another decade or two.

risk (*6*). The risk was shown to be greater among women and decreased significantly with increasing age-at-exposure, with little risk apparent after age 20 years. Radiation-induced thyroid cancer can be detected in approximately 4–5 years after exposure. The International Commission on Radiological Protection (ICRP) has proposed a 4-year latency period for radiation protection purposes (*12*). However, a report of Chernobyl data (*20*) showed a minimum latency period of 3 years, followed by a linear increase with time after exposure. A recent review of thyroid cancer incidence in the LSS cohort for the 1958–2005 period confirmed the earlier findings and showed that the excess thyroid cancer risk associated with childhood exposure persisted for more than 50 years after exposure (*21*). The data collected from external radiation exposures are consistent with the findings from internal exposures to radioactive iodine from the Chernobyl accident, indicating that thyroid cancer risk is higher in children (*22,23*). Significant increases in thyroid cancer incidence risks have also been seen after radiotherapy in childhood (*24*).

Breast cancer

Breast cancer is the most common cancer in women worldwide. Its incidence has grown rapidly during recent decades in many countries. Radiation effects on female breast cancer rates have been extensively studied in the LSS cohort (*7*) as well as in many other populations (*25,26,27,28,29,30*). Epidemiological data indicate that the risk of breast cancer is higher after radiation exposure at younger ages. The LSS cohort showed a minimum latency period of about 12 years. The epidemiological findings reviewed regarding excess female breast cancer risks after radiation exposure in early life indicate that no breast cancer cases have been seen before 20 years of age, regardless of the time since exposure (*31,32,33*). Only one study of secondary cancer reported a single breast cancer case before age 20, and it must be taken into account that those individuals developing secondary cancers after childhood or adolescent primary cancers may have heightened genetic susceptibility.

All solid cancers

The concept of "all solid cancers[2]" comprises a variety of clinical entities. Pooling the data provides a picture of the overall cancer risk from radiation and reflects the fact that radiation causes cancer in most body organs. It also enhances statistical power, which is particularly relevant when assessing risks at low doses (*16*). For all solid cancers, the minimum latency is thought to be approximately 5 years. In general, risks are higher among women and for younger ages-at-exposure. The LSS cohort showed a gradual increase in solid cancers beginning several years after the bombings (5–10 years). Excess risk is still seen among atomic bomb survivors more than 50 years after exposure (*19,21*).

Cancer risk following *in utero* exposure

On balance, the evidence points to an increased risk of leukaemia and other cancers in childhood after exposure *in utero* to radiation. An association between in utero exposure and childhood leukaemia and other childhood cancers has been observed in a number of case-control studies of prenatal X-ray examinations (*34*). While cohort studies of *in utero* exposure have not confirmed this association between prenatal radiation exposure and childhood cancer, it should be noted that their findings are limited by low statistical

2. All cancers other than leukaemia, lymphoma and multiple myeloma

power (*35*). However, when statistical, dosimetric, modelling and other uncertainties are taken into account, the risk estimates for childhood cancer obtained from X-ray case-control studies are comparable with the risk of childhood leukaemia among the Japanese survivors exposed as children. The cancers typical of childhood differ from those of adult life, and the risk models developed from adult cancer mortality and incidence are not necessarily applicable to childhood cancers. Preston et *al.* (*36*) reported an increased risk of adulthood solid cancers in atomic bomb survivors exposed *in utero*, which approached the level of risk among the survivors exposed in early childhood. The ICRP has reviewed epidemiological data on cancer risks after irradiation *in utero* and concluded that, overall, the risks are not greater than those predicted for early post-natal exposures (*35*). Consequently, in this assessment the lifetime cancer risk of *in utero* exposure is considered to be similar to that from exposure in early childhood.

2.2.2 Health effects other than cancer

This section describes non-cancer radiation effects that are expected after moderate or high dose exposures (see chapter 3). On the basis of the WHO preliminary dose estimation, these effects are not relevant for the general population but may contribute to further discussions on potential health risks for the NPP emergency workers.

Thyroid diseases (nodules, dysfunction)

Two types of thyroid diseases are related to radiation exposure, i.e. dysfunction and benign nodules. A number of studies have been published on the development of hypothyroidism as a deterministic effect after external radiotherapy of benign and malignant diseases of the neck, as well as after nuclear medicine procedures using radioactive isotopes of iodine (*37,38,39,40,41*). Exposure to high levels of radioactive fallout has also been linked to hypothyroidism (*42*).

Excess risk of thyroid nodules has been documented following brief exposures to external radiation associated with medical procedures or the atomic bomb (*43,44*) and from protracted exposure to radioactive iodine (*45,46,47,48*). However, studies in areas with high background radiation have not shown significant elevations in thyroid nodule risk (*49,50*). Excess risks are higher at younger ages-at-exposure and are somewhat higher in females than in males. The prevalence of thyroid nodules varies with the population studied and the methods of detection. Studies using ultrasound show a prevalence of 19–35% (*51*). Detected prevalence has increased in recent years, likely owing to improved resolution from advanced imaging technology. For most thyroid nodules detected, there are no long-term adverse consequences, and intervention is not usually indicated since surgery carries risks that may more than offset any possible benefits (*43*). Non-cancer thyroid nodules are not lethal and seldom cause any medical problems (*52*). Although the risk of subsequent cancer development is small, regular monitoring of those with thyroid nodules may be warranted (*53*).

Visual impairment (lens opacities, cataracts)

The lens of the eye is one of the most radiosensitive tissues in the body. Epidemiological studies have demonstrated that radiation exposure of the eyes can induce lens opacities (*54*). Although the initial stages of these kinds of radiation-induced changes do not imply visual disability, they can progress to more severe changes, including vision-impairing

cataracts. The severity of radiation-induced lens opacities increases with the dose, and the latency is inversely related to the dose (*55*).

Circulatory diseases

For circulatory diseases – i.e. cardiovascular and cerebrovascular diseases – the LSS on atomic bomb survivors (*19,56*), as well as other epidemiological studies on occupational, medical and accidental exposures, show a statistically significant radiation effect (*57,58,59*). Also, significant associations between radiation exposure and both cerebrovascular and cardiac disease mortality were observed among Mayak nuclear facility workers (*60,61,62*). However, there is still substantial heterogeneity in the observed associations (*63*).

Reproductive dysfunctions

The effects of radiation on reproductive function have been studied in people occupationally exposed (e.g. radiologists, nuclear workers), in patients treated with radiotherapy and in individuals accidentally exposed to radiation (*64*). Transitory or permanent infertility after exposure to moderate or high radiation doses, respectively, is the major long-term consequence reported, with temporal patterns and threshold doses that differ widely between male testes and female ovaries (see section 3.7).

Teratogenic effects

The sensitivity of mammalian embryo and fetus to radiation exposure has been well documented by experimental research, mainly in rodents. The risk of radiation-induced tissue damage and developmental changes in embryo and fetus has been reviewed by the ICRP (*65,35*), who concluded that teratogenic effects are not expected in humans after prenatal exposure to low doses.

Animal data indicate that death is the dominant teratogenic effect after exposure to high doses of radiation in the pre-implantation period of embryonic development (*35*). However, no human data of radiation effects in this period are available.

During organogenesis (2–7 weeks post-conception in humans), the type of effects depends on the natural sequence of developmental steps. No radiation effects in human embryos exposed in this period have been observed. Data from experimental research in rodents suggest that radiation might induce lethality or interfere with normal embryonic development.

The fetal period in humans starts around the eighth week post-conception and extends up until the end of pregnancy. Data from the LSS indicate that the period of 8–15 weeks post-conception constitutes the "window" of maximum radiosensitivity of the developing brain. The sensitivity is lower at weeks 16–25 post-conception. There is no evidence for mental retardation associated with radiation exposure before week 8 or after week 26 post-conception. Dose-response relationships and threshold values for deterministic effects after in utero exposure are discussed in Annex F, section F.1.

Heritable effects

Heritable effects of radiation have not been definitively demonstrated in human populations, but their existence is suggested by experimental studies that have shown radiation-

induced hereditary effects in laboratory animals (*66*). No significant increase in heritable effects has been found in studies of the children of the survivors of the atomic bombings of Hiroshima and Nagasaki (*67*) or in the offspring of cancer survivors treated with radiotherapy (*68*), which indicates that moderate acute radiation exposures have little impact on the overall risk of heritable effects in humans. The ICRP has based its heritable disease risk estimates for the purpose of radiological protection upon the findings of large studies involving experimental animals and knowledge of human genetics. The ICRP concluded that, as a stochastic effect, the risk of radiation-induced heritable effects has no threshold dose, and that the risk per unit dose in the offspring of those exposed at reproductive age is much less than that of cancer in the exposed individual (about one order of magnitude lower) (*12*).

Other non-cancer effects

Early tissue reactions can be observed during the days and weeks following exposure (e.g. acute radiation syndrome [ARS], skin burns), while late tissue reactions may develop months or years after exposure. Acute skin reactions, including erythema (reddening), dry desquamation, and moist epithelitis (blistering), as well as late cutaneous fibrosis, are tissue reactions observed only after exposure to high doses. ARS is observed after whole-body exposure to high doses. Clinical changes in ARS result from radiation-induced damage to early reacting organ systems (haematopoietic, gastrointestinal and neurovascular, depending on the dose range) and they are mainly manifested within a few weeks after exposure. The haematopoietic system is the primary target in ARS, showing characteristic changes in peripheral blood cells, whose kinetics and severity are closely related to the dose (*69*). These kinds of effects are not expected when assessing health risks resulting from exposure to low radiation doses.

3. Dose-response relationship

A fundamental component of hazard characterization is the dose-response relationship, which provides the quantitative means for translating radiation exposure into corresponding health risks. This relationship, also called risk model, is a necessary tool for risk assessment, albeit a simplified summary of observations.

Radiation effects are highly dependent on dose. In this document, exposures are generically referred to as moderate/high dose above 100 mSv and as low dose below 100 mSv. This terminology is broadly consistent with the categories defined in ICRP publication 99 (*4*).

This chapter presents the dose-response relationships for stochastic and deterministic effects and describes the main risk quantities used in this HRA. Risk models for four different cancer sites, or groups of cancer sites (i.e. all solid cancers), are used for the Fukushima Daiichi NPP accident.

3.1 Non-threshold dose-response models for stochastic effects

Much of the epidemiological information used to develop cancer risk models comes from exposures to moderate or high radiation doses. As mentioned in section 2.2.1, a major source of epidemiological data for these models is the LSS on Hiroshima and Nagasaki atomic bomb survivors (*70*). Additional supporting evidence to LSS comes from studies on medical, occupational and environmental exposures. Risk estimates resulting from analyses of these epidemiological data sets do not allow for definitive statements about the shape of the dose response when the dose is low and/or delivered over a long period of time (low dose rate), although they are consistent with excess cancer risks that are proportional to exposure as predicted by the Linear No Threshold (LNT) model (*71,72, 73,74,75,76,77*).

For the purposes of radiological protection, the assumption is made that the risk of inducing cancer by low doses of radiation is proportional to the dose. The underlying dose-response relationship is linear with no threshold. In other words, radiation exposure is always considered to pose some level of risk (albeit very small at low doses), and the sum of several very small exposures is assumed to have the same effect as one larger exposure of the same overall magnitude. The LNT basically rests on the assumption that biological damage, which, if repaired incorrectly, could lead to cancer and is directly proportional to dose throughout a relevant range of doses and dose rates. The predicted level of excess cancer risk related to low-dose radiation exposures is small and it is therefore difficult to detect reliably against the normal fluctuations in the baseline cancer incidence rate[1] (*4,77*).

Although some dissenting views on the LNT have been expressed, it is thought to be a prudent basis for risk assessment. Extrapolation of the dose-response relationship to low-dose exposure involves several assumptions that rely on expert opinion. Attempting not

1. The baseline cancer incidence rate refers to the number of cancers of a specific site or type naturally occurring in a specified population during a year.

to underestimate the risks, the HRA Expert Group judged that the LNT model provided the most reasonable description of the relation between low-dose exposure to ionizing radiation and the incidence of cancers[2].

3.2 Multiplicative and additive risk models

In epidemiological studies, two risk models are commonly used to describe the health effects of radiation. In the relative risk (multiplicative) model, the risk induced by radiation is seen as a multiple of the baseline disease risk[3] and is expressed in terms of relative risk (RR) or excess relative risk (ERR). The RR is the ratio of the rate of occurrence of disease in an exposed population to that in a comparable non-exposed population. Such a model inherently assumes that radiation increases the occurrence in direct proportion to the baseline rate in the population. This means that a larger absolute effect is expected for a population with a higher risk of baseline cancer. The excess relative risk is the relative risk minus 1 (ERR = RR –1) and is the proportional increase in the baseline risk.

Alternatively, an absolute risk (AR) model can be adopted, which presumes a constant absolute increase in risk per dose unit, regardless of the baseline risk. The excess absolute risk (EAR) refers to the difference in the rate of occurrence of disease between an exposed population and a comparable non-exposed population. If the radiation-related absolute risk is independent of other risk factors that may be influencing the baseline cancer rates, the EAR simply adds to any other absolute risk factor and the interaction between radiation and other risks is "additive". The EAR is a measure of the absolute size of the radiation effect, which may be of public health or clinical significance (*16*).

The difference between the two models can be further illustrated by the following example. A cohort study might report cancer incidence of 150 per 100 000 person-years in an unexposed group and 200 per 100 000 person-years among subjects exposed to radiation. The RR for the exposed cohort is then 1.33 (200/150), and the ERR is 0.33. The AR among the exposed group is 200/100 000 person-years and the EAR is 50/100 000 person-years (200/100 000–150/100 000). Adopting an ERR risk model would imply that the effect of a similar exposure in any other population would result in 1.3-fold increase of the baseline rate, whereas extrapolation using an EAR model would predict an increase by 50/100 000, independent of the baseline rate.

3.3 Lifetime risk concepts

3.3.1 Lifetime baseline risk

Based on cancer incidence rates from a general population, the lifetime baseline risk (LBR) is the cumulated baseline probability of having a specific cancer over the lifetime (calculated in this HRA up to the age of 89 years).

For the present HRA, the LBR is as follows:

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2. Note that this also applies to leukaemia at low doses and dose rates because of the linear part of the linear-quadratic model.
3. While the baseline cancer incidence rate is a measure of disease occurrence, the baseline cancer incidence risk (or, in general, the baseline disease risk) is a measure of the probability of developing the disease during a year.

$$LBR = \int_{a_{\min}}^{a_{\max}} m(a,g) S_{aj}(a,g)\, da \quad (1)$$

where a is the attained age, g is the sex, $m(a,g)$ is the baseline cancer incidence rate in the population or sub-population at risk and $S_{aj}(a,g)$ is the cancer-free survival function (adjusted survival function) of the unexposed population (see Annex D for further discussion on the survival function). In this HRA, the LBR is calculated from the age-at-exposure a_{min} (e.g. 1, 10 or 20 years, depending on the age group selected for the calculation) up to a_{max} (89 years old). It is assumed that a person must be alive and cancer-free at a_{min}; therefore $S_{aj}(a,g)$ equals 1 at that age and then decreases as the attained age increases. For example, to follow a one-year-old infant it is assumed that the person is alive and cancer-free at 1 year of age and therefore $S_{aj}(a,g)$=1 at 1 year of age. Similarly, to follow a 20 year-old person it is assumed that the person is alive and cancer-free at 20 years of age and $S_{aj}(a,g)$=1 at 20 years of age.

3.3.2 Lifetime attributable risk

The lifetime attributable risk (LAR) specifies the probability of a premature incidence of a cancer attributable to radiation exposure in a representative member of the population (*78,79,80,81*). For a given dose, LAR is the additional cumulated probability of having a specific cancer up to the age of 89 years. It relies on the use of a risk model derived from the epidemiological literature and is a classical risk indicator in the field of radiation protection. Its mathematical definition is provided in Box 4.

For the present HRA, the HRA Expert Group deemed LAR to be appropriate as a primary risk measure. As mentioned above, the assessment separately considered some cancer sites as being more radiosensitive and with higher dependence on age-at-exposure, namely leukaemia, breast cancer and thyroid cancer, plus all solid cancers combined (*4*).

The choice for the risk models $M(D,e,a,g)$ is described in section 3.4 and is provided for the four cancer sites (Annex E). The input data related to the dose (D) are given in Chapter 4. The survival curves $S_{aj}(a, g)$ are further discussed in Annex D, and the health statistics data, which form the basis of the derivation of the survival curves, are provided in section 5.1.3.

The LAR calculations were provided using sex (g)-specific models, thereby accounting for differences between males and females. For the general public, both sexes were analyzed. For workers, the HRA Expert Group decidedto perform the risk modelling calculations only for male workers on the basis of information indicating that the workforce engaged in the emergency response work was composed mainly of male workers.

The calculation of LAR requires, as one of the parameters, the age-at-exposure (e). For the general population, the risks were calculated for persons who were 1-year-old infants, 10-year-old children and 20-year-old adults at the time of radiation exposure. For workers, the risks were calculated for adults who were 20 years old, 40 years old and 60 years old at the time of the accident.

Calculations of the LAR were performed as a function of attained age (a) for the period of life after radiation exposure up to the end of the 90th year of life (i.e. $a_{max} = 89$ in equation 2).