The latency period (L) is dependent on the cancer site. Based on the evidence highlighted in section 2.2.1, the minimum latency periods adopted in the present assessment are 2 years for leukaemia, 3 years for thyroid cancer and 5 years for female breast cancer and all solid cancers.

3.3.3 Lifetime fractional risk

A related quantity, the lifetime fractional risk (LFR), is often used to reflect the relative increase in the lifetime cumulative probability of cancer, attributable to a given dose. This probability, when not linked to the probability of baseline cancer incidence, can be misleading. LFR is a relative number obtained when the LARis scaled, as suggested by Kellerer et al. (*80*), to LBR in the reference (non-exposed) population. LFR is defined as the fractional increase over the LBR, and is expressed as a percentage:

$$LFR = \frac{LAR}{LBR} \quad (6)$$

3.3.4 Cumulative risk for a segment of life

The LAR is a very useful concept in radiation protection as it integrates the expression of the radiation induced risk over the whole lifespan. This indicator, however, is associated with very large uncertainties since it is very difficult to extrapolate the cancer rates so far into the future. In this assessment, for a 1-year-old infant at the time of the Fukushima accident, the LAR corresponds to the risk predicted up to the year 2100.

The uncertainties associated with LBR, LAR and LFR can be decreased by calculating the cumulative risks for segments of lifetime. For the purpose of this report, these risk quantities are presented for the 15-year period of life after radiation exposure using the

Box 4. Mathematical definition of the lifetime attributable risk

The lifetime attributable risk, LAR, can be calculated using either an excess absolute risk (EAR) model or an excess relative risk (ERR) model or a mixture of the two. For a person of sex g exposed to dose D at age-at-exposure e, the LAR for a specific cancer site at attained-age a, is:

$$LAR(D,e,g) = \int_{e+L}^{a_{\max}} M(D,e,a,g)\frac{S_{aj}(a,g)}{S_{aj}(e,g)}da \quad (2)$$

where

- $M(D,e,a,g)$ is the risk model;
- $S_{aj}(a,g)$ is the probability of surviving cancer-free to age a,for the unexposed population;
- L is the minimum latency period;
- the ratio $S_{aj}(a,g)/S_{aj}(e,g)$ is the conditional probability of a person alive and cancer-free at age-at-exposure e to reach at least an attained-age a.

The risk model $M(D,e,a,g)$ can be defined in three ways:

Additive transfer:

$$M(D,e,a,g)=EAR(D,e,a,g) \quad (3)$$

Multiplicative transfer:

$$M(D,e,a,g)=ERR(D,e,a,g)\, m\,(a,g) \quad (4)$$

or a weighted arithmetic sum of both:

$$M(D,e,a,g)=w\ EAR(D,e,a,g)+(1\text{-}w)\ ERR(D,e,a,g)\ m(a,g) \quad (5)$$

where $m(a,g)$ is the baseline cancer incidence rate in the population or sub-population at risk, and w is a weighting factor, the risk-transfer weight.

abbreviations AR_{15}, BR_{15} and FR_{15} expressing, respectively, the attributable risk, the baseline risk and the fractional risk at age-at-exposure e (i.e. $a_{max} = e + 15$).

3.3.5 Other measures of lifetime risk

Other measures of lifetime risk have been used to express radiation risks (*78,79,80,82*). One of these quantities, the risk of exposure-induced death (REID), was used in a recent UNSCEAR report (*83*) as a measure of lifetime risk that estimates the probability that an individual will die from cancer associated with the exposure.The REID differs from the LAR in that the survival function used in calculating the REID accounts for persons dying of non-cancer radiation-induced disease. This difference may be important for estimating risks at high doses (> 1 Sv) where such deterministic effects are relevant, but not at the low doses of interest in this report. At doses below 0.5 Sv, REID and LAR values are very similar (*80*).

3.4 Cancer risk models

The cancer risk models describe the variation of the radiation-induced excess risk of a specific type of cancer with the magnitude of the relevant tissue-specific absorbed dose of radiation that has been received – the dose-response relationship for the site-specific cancer.

In this report, cancer incidence is assessed rather than cancer mortality because many cancers now have a high probability of cure; therefore, incidence is more relevant for public health.

In choosing the radiation excess risk models to apply, the HRA Expert Group considered several existing models (Box 5). The sex-specific radiation risk models used in this assessment for both the general population and emergency workers are based on the LSS cohort of Japanese atomic bomb survivors. For all solid cancer and site-specific cancers, Preston et al. (*70*) provided incidence models with details of the model fit parameters available with the required number of decimal places from the RERF website (*84*), making these cancer incidence models a good choice for the HRA. However, no recent leukaemia incidence models were available at the time of this assessment[4]. Therefore, it was decided to apply a leukaemia mortality model with a linear quadratic dose-response from the UNSCEAR 2006 report (*83*). A recent analysis of leukaemia mortality in Japanese atomic bomb survivors showed that this model has a better fit to the atomic bomb data than other models considered (*85*). Although this HRA focuses on incidence risks, it was considered that the radiation risks for mortality and incidence of leukaemia, as derived from the atomic bomb data on children (i.e. pertaining to the 1950–1960 time period), were probably very similar owing to the generally poor survival rates of children with leukaemia in the middle of the last century.

Annex E gives full details of the radiation excess risk models and fit parameters that were applied in this assessment – i.e. the UNSCEAR 2006 report (*83*) on leukaemia mortality models (EAR and ERR), with the linear quadratic dose-response as developed by Little *et*

4. At the time of the publication of this report, new data on incidence of leukemia, lymphoma and multiple myeloma among Atomic Bomb Survivors between 1950–2001 were published by Hsu W-L et al. (published online in the *Journal of Radiation Research*, February 11, 2013 http://www.rrjournal.org/doi/abs/10.1667/RR2892.1).

al.(*86*) and the Preston *et al.* (*70*) incidence models for all solid cancers (EAR and ERR), thyroid cancer (EAR and ERR) and female breast cancer (EAR).

3.5 Transfer of excess risk between populations

A risk model, $M(D,e,a,g)$, allows the transfer of risk estimates from one context to another, for example, from the population providing the data from which the risk model was derived to another population with a different baseline cancer risk.

As seen in section 3.2, the extra cancer risk resulting from a particular exposure to radiation can be expressed either as a multiplicative model (ERR) or an additive model (EAR). Different combinations of these two models of interaction are possible. Although the selection of either of these two approaches may make little difference to the predicted radiation-related excess risk for the population from which the epidemiological data were derived, it can make a substantial difference when a risk model is transferred to another population. This is particularly critical for cancer sites for which the baseline incidence or mortality rates differ markedly between the two populations. Table 2 summarizes the current views of international expert groups on approaches to risk transfer for the cancer sites relevant to this report.

Box 5. Recent cancer risk models

Cancer incidence risk models describe how the probability of radiation-inducing cancer varies with the dose absorbed in different tissues or organs. These models take into account parameters such as sex, age-at-exposure, attained age and time since exposure. They can be regarded as tools for quantitatively assessing the impact of radiation in populations with similar characteristics (e.g. sex, age-at-exposure). Several expert groups and international committees have used the knowledge of health effects of radiation from experimental and epidemiological studies to construct risk models.

- The 2006 **UNSCEAR** report (*83*) derived specific risk models for leukaemia, thyroid, stomach, colon, liver, lung, female breast, oesophagus, bladder, bone, brain and central nervous system, non-melanoma skin, and all other solid cancers combined. UNSCEAR applied these models to the current baseline rates in China, Japan, Puerto Rico, the United Kingdom and the United States of America.
- The 2006 **BEIR VII** report (*87*) derived site-specific cancer risk models for leukaemia, 10 solid cancer sites (thyroid, stomach, colon, liver, lung, female breast, prostate, uterus, ovary, bladder), and all other solid cancers combined. These estimates are based on the USA cancer incidence rates for 1995–1999.
- The **ICRP** 2007 recommendations (*12*) derived specific risk models for leukaemia, thyroid, stomach, colon, liver, lung, female breast, ovary, oesophagus, bladder and all other solid cancers combined, and applied those models to cancer incidence data from six different Asian and Euro-American populations. These risk models assumed sex-averaged and age-at-exposure averaged populations to generate nominal cancer incidence risk coefficients in the context of the system of radiological protection.
- The **United States Environmental Protection Agency** (EPA) modified and extended BEIR VII risk models in 2011, including other solid cancer sites (*88*).
- The **United States National Cancer Institute** published in 2012 an online radiation risk assessment tool (RadRAT) to calculate lifetime cancer risks from single or multiple exposures, including uncertainty distributions (*89*). It is based on BEIR VII methods, with a number of small modifications, and includes risk models for seven additional cancer sites.

Table 2. Risk transfer approaches adopted by international expert groups

Cancer site	UNSCEAR		BEIR VII	ICRP 103
Leukaemia	100% ERR	100% EAR	70% ERR and 30% EAR	100% EAR
Thyroid cancer	100% ERR	100% EAR	100% ERR	100% ERR
Breast cancer	100% ERR	100% EAR	100% EAR	100% EAR
All solid cancers	100% ERR	100% EAR	70% ERR and 30% EAR	50% ERR and 50% EAR

Table 3. Risk transfer weights adopted in the current assessment

Cancer site	Transfer weights adopted to calculate LAR	Evidence for the transfer weight choice	ICRP 103
Leukaemia	50% ERR, 50% EAR (w = 0.5)	UNSCEAR 2006 (83) BEIR VII 2006 (87) EPA 2011 (88) ICRP 2007 (12)	100% ERR (w = 0) and 100% EAR (w = 1)
Thyroid cancer	50% ERR, 50% EAR (w = 0.5)	Jacob et al 2006 (*107*) Walsh et al 2009 (*178*)	100% ERR (w = 0) and 100% EAR (w = 1)
Breast cancer	100% EAR (w = 1)	Preston et *al.* 2002 (*30*)	–
All solid cancers	50% ERR, 50% EAR (w = 0.5)	ICRP 2007 (*12*)	100% ERR (w = 0) and 100% EAR (w = 1)

Note that UNSCEAR results are presented for ERR and EAR separately.

For this assessment, a hybrid model has been adopted combining relative and absolute risk approaches for all cancer sites except for breast cancer, for which a pure absolute risk model was used (see Table 3). The risk transfer weights w (defined in Equation 5, Box 4) used in this assessment are shown in Table 3. The percentages are an alternative representation, where, for example, an assigned value of $w=1.0$ for breast cancer is equivalent to a 100% EAR. The risk transfer weights were chosen on the basis of expert judgement supported by evidence. For the Fukushima accident, the transfer is from the Japanese population exposed in 1945 (the LSS cohort) to the Japanese population exposed in 2011 (and following years). While it is clear that changes have occurred over the past 60 years in terms of cancer incidence baselines and in terms of possible interactions between radiation and other cancer risk factors (*90*), the choice of the risk transfer weights is expected to have low impact, as discussed in section 6.2.3.

The HRA Expert Group also tested the option of transferring 100% of the risks as ERR or EAR, with the exception of breast cancer. Based on the reviewed evidence described in section 2.2.1, the HRA Expert Group agreed that the minimum age for breast cancer risk expression considered for the present HRA would be attained at age 20 years. This is consistent with the Japanese baseline cancer rates used in the present assessment, indicating no female breast cancer incidence before the age 20 years.

3.6 Dose and dose rate effectiveness factor

At high doses, a modest upward curvature is observed in the overall dose response for some solid cancers (*91*). This finding, as well as evidence from experimental studies, have suggested the need to apply a factor when extrapolating from cancer risks assessed at a high dose and a high-dose rate to estimate risks at a low-dose and a low-dose rate. This factor, called the "dose and dose rate effectiveness factor" (DDREF), represents the ratio between risks at high-dose/high-dose rates and low-dose/low-dose rates. The ICRP currently proposes the application of a DDREF of 2 for radiation protection purposes (*12*) while the BEIR VII report (*87*) proposes a DDREF of 1.5.

Consideration of uncertainty led to the development of probability distributions of DDREF for use in risk assessment (*89*). Still there is a lack of a full understanding of the processes leading to cancer after low-dose radiation exposure. The solid cancer risk in 12 epidemiological studies of radiation-exposed workers and of the population residing at the contaminated Techa River in the Southern Urals, Russia, was compared to cancer risks among the Japanese atomic bomb survivors (*74*). Overall, risk estimates were similar to those among the atomic bomb survivors, suggesting that a DDREF of 1 would be reasonable. A meta-analysis has considered recent epidemiological evidence on leukaemia mortality and incidence risks from protracted low-dose and low-dose-rate exposures to γ-rays. It included an extensive literature review of studies on groups of people who were either occupationally or environmentally exposed (*92*). The main risk measure value reported in this meta-analysis (ERR) indicated that the baseline leukaemia risk (i.e. risk for a group of unexposed persons) increases by 19% after exposure to a dose of 100 mGy. This increase was reported to agree closely with the risk from acute exposure of the Japanese atomic bomb survivors and is therefore an indication that leukaemia risks are similar for protracted and acute exposures[5].

Exposures of the population to ionizing radiation from radionuclides released in the course of the Fukushima accident are expected to occur over periods of days, weeks, months and even years. These exposures are thus not acute, in contrast to the exposures of the survivors of the atomic bombings of Hiroshima and Nagasaki, which provided most of the evidence for estimates of cancer risks after exposure to ionizing radiation.

The question therefore arises as to whether the risk estimates for the atomic bomb survivors are applicable to populations that have accumulated radiation doses on the order of 100 mGy or below over a long time. Thus far, radiobiological research has provided ambiguous answers to this question. Based on the findings of the two meta-analyses discussed above (*74,92*), which showed similar risks for protracted and acute exposures, the HRA Expert Group considered it prudent to base risk calculations on models derived from the atomic bomb survivors cohort without applying any modification factor for low dose or low dose rate. This decision, which represents a departure from standard practice in radiation risk assessment, was not unanimous as two members expressed a dissenting opinion[6].

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5. The leukaemia dose-response relationship is linear in the low-dose and low-dose-rate region. The quadratic component is relevant at a higher doses received at high dose-rate.
6. Dr O. Niwa and Dr M. Akashi supported the use of a DDREF of 2.

3.7 Threshold dose-response models for deterministic effects

The dose-response relationship for deterministic effects, characterized by the presence of a threshold dose below which the effect is not observed, has been extensively studied. The ICRP has recently reviewed early and late reactions in normal tissues and organs, including the response of the skin and eye, as well as haematopoietic, immune, reproductive, circulatory and endocrine systems, among others (*64*). For practical purposes, the updated estimates of threshold doses for tissue injury were defined in most cases as the dose level that would result in 1% incidence of an effect, including morbidity and mortality endpoints in the reviewed organ systems[7] after acute, fractionated and chronic exposure. Taking into account the level of these threshold doses, tissue reactions are generally not relevant health outcomes for environmental exposures to low radiation doses.

The dose thresholds for deterministic effects are summarized in Table 4 (adapted from ICRP 103 (*12*) and ICRP 118 (*64*), and further details on the dose-response relationship of specific endpoints are provided in Annex F. It was recently suggested that dose thresholds for some late tissue reactions such as eye lens opacities and circulatory diseases might be lower than earlier thought. The dose-response relationship for these effects is currently a matter of discussion – i.e. whether these non-cancer effects are deterministic or stochastic in nature.

7. The organ systems comprise haematopoietic, immune, reproductive, circulatory, respiratory, musculoskeletal, endocrine and nervous systems, digestive and urinary tract, skin and eye.

Table 4. Projected threshold estimates of the absorbed doses for 1% incidence morbidity for acute exposure to gamma radiation (adapted from ICRP 103 (*12*) and ICRP 118 (*64*)).

Effect	Organ/tissue	Threshold (Gy)*	Time to develop the effect	Observations
Temporary sterility	Testes	0.1	3–9 weeks	
Permanent sterility	Testes	6	3 weeks	
	Ovaries	3	< 1 week	
Depression of haematopoiesis (blood-forming process)	Bone marrow	0.5	3–7 days	In case of chronic exposure the threshold is 0.4 Gy/year
Cardiovascular disease	Heart	0.5	Long-term effect	Recently estimated by ICRP based on epidemiological findings
Stroke	Circulatory system	0.5	Long-term effect	
Pneumonitis	Lung	6.5	3–6 months	In case of highly fractionated exposures (e.g. radiotherapy) the threshold is 18 Gy
Renal failure	Kidneys	7		In case of highly fractionated exposures (e.g. radiotherapy) the threshold is 18 Gy
Skin reddening (erythema)	Skin	3–6	1–4 weeks	
Skin burns	Skin	5–10	2–3 weeks	
Temporary hair loss	Skin	4	2–3 weeks	
Visual impairment (cataract)	Lens of the eye	0.5	Long-term effect	A previous threshold of 1.5 Gy was later lowered to 0.5 Gy.

* Thresholds are expressed as organ-absorbed doses and are therefore expressed as Gy units. For comparison purposes, and taking into account that the radiation weighting factor for gamma rays is 1, these threshold values are numerically equal to the organ-equivalent dose expressed in Sv.

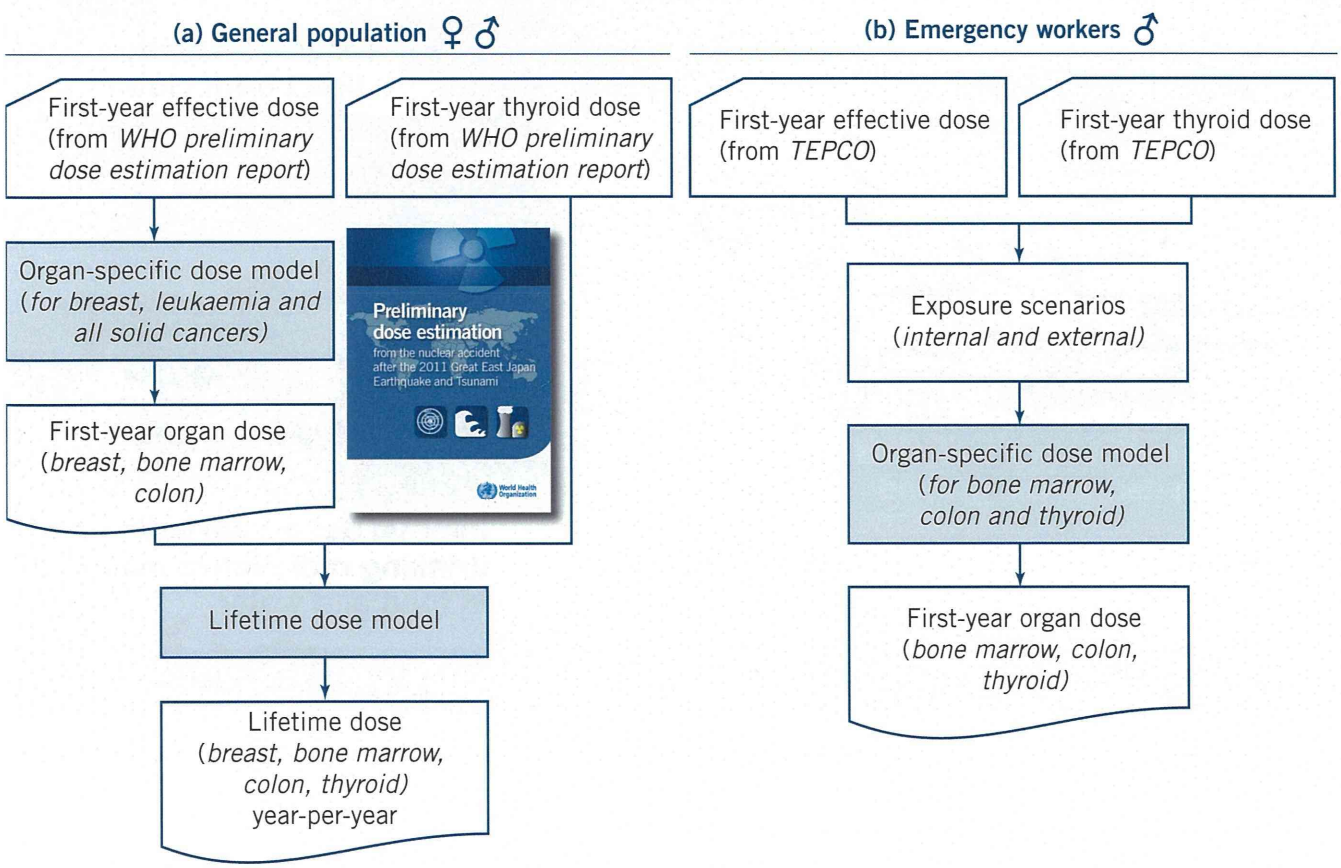
4. Exposure assessment

This chapter provides dose estimates as a result of the Fukushima Daiichi NPP accident or the general population and for the NPP emergency workers. The pathways of exposure and the methodology used are described for each population group (Figure 3).

4.1 Doses for the general population

The characterization of the lifetime attributable risk (LAR) for different cancer types requires knowledge of the dose to the affected organ over the lifetime of the individual. From the doses provided in the WHO preliminary dose estimation report (*3*), the first-year organ doses to each of four organs are calculated, providing the basis for a lifetime dose to each organ (Figure 3a).

Figure 3. Process to assess doses for the general public and the workers



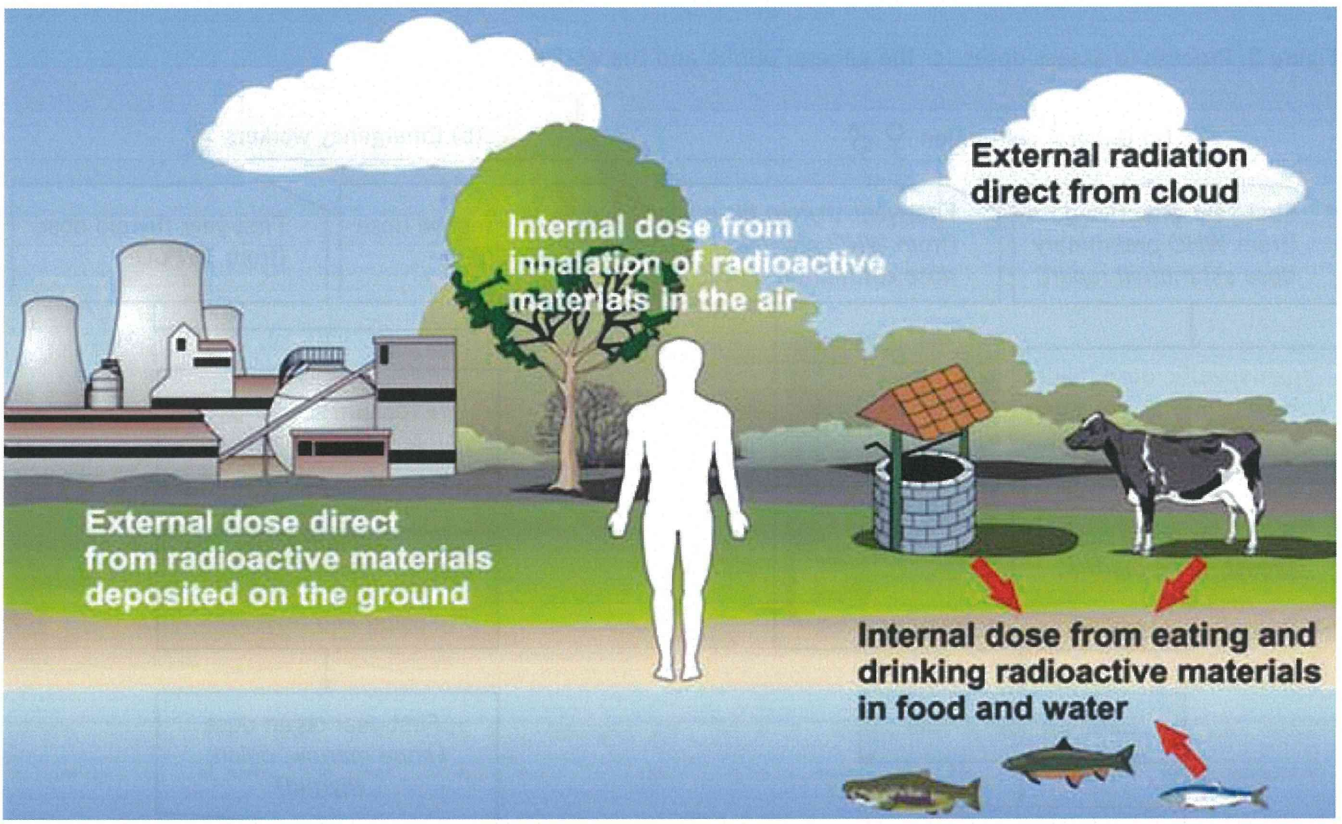
4.1.1 Pathways of exposure for the general population

Human exposure to ionizing radiation may be internal or external. Internal exposure occurs when a radionuclide is inhaled or ingested, or after it has entered the bloodstream as a result of wound or skin absorption. Once the radionuclide enters the body, internal radiation exposure will continue until radioactivity disappears owing to radioactive decay or elimination of the radionuclide through excretion. External exposure to ionizing radiation occurs when a radiation source irradiates a person from outside the body. External exposure can result from radiation sources located at some distance from the body surface (e.g. deposited on the ground, suspended in the air). This kind of external irradiation can be reduced or even stopped by shielding or removing the radioactive source, or moving the person outside the radiation field.

After the Fukushima Daiichi NPP accident, the general public was exposed to radioactive material through four major exposure pathways[1] (see Figure 4):

- external exposure from radionuclides deposited on the ground (groundshine)

Figure 4. Exposure pathways to humans from environmental releases of radioactive material



Source: IAEA report on Environmental consequences of the Chernobyl accident and their remediation: twenty years of experience (2006) p. 100 (reproduced with permission).

1. The radioactive material (dust, liquid, aerosol) can also be deposited on clothes and/or the skin. In this situation often called "external contamination", radioactivity can be removed from the body by changing clothes and/or washing the skin. External radioactive contamination as a route of exposure was not a relevant contributor to the doses received by the general public after the Fukushima Daiichi NPP accident.

- external exposure from radionuclides in the radioactive cloud (cloudshine)
- internal exposure from inhalation of radionuclides in the radioactive cloud (inhalation)
- internal exposure from ingestion of radionuclides in food and water (ingestion).

In June 2011 WHO established the Dose Expert Panel to make an initial evaluation of radiation doses incurred in the general population for the first year after the Fukushima accident. The estimated doses were provided in a WHO report released in May 2012 (*3*).

Doses in the following areas were considered:

- locations within Fukushima prefecture (outside the 20 km evacuation zone[2]) where doses were likely to be among the highest of those received by the general population;
- the rest of Fukushima prefecture;
- the prefectures in Japan nearest Fukushima;
- the rest of Japan;
- countries neighbouring Japan;
- the rest of the world.

Doses within a 20-km radius around Fukushima Daiichi NPP were not assessed in the WHO preliminary dose estimation and therefore this geographical area is not included in this HRA. Although most people in that area were rapidly evacuated, a certain dose may have been received prior to evacuation. The assessment of such doses would have required more precise data than were available to the Dose Expert Panel.

2. Most people within 20 km of the nuclear power plant were rapidly evacuated and the Dose Expert Panel chose not to estimate doses in this area. Outside the 20-km radius, inhabitants of the most affected area, coined the "deliberate evacuation zone", were subject to relocation at different times after the accident. For the assessment of doses in this area, the Dose Expert Panel estimated only doses in the first four months of the first year, with the conservative assumption that relocation took place at 4 months (although in some places people were relocated earlier).

Box 6. Dosimetric quantities

Dosimetric quantities are needed to assess human radiation exposures in a quantitative way. The International Commission on Radiological Protection (ICRP) provides a system of protection against the risks from exposure to ionizing radiation, including recommended dosimetric quantities.

The fundamental measure of the radiation dose to an organ or tissue is the **absorbed dose**, which is the amount of energy absorbed by that organ or tissue divided by its weight. The international unit of absorbed dose is the gray (Gy), which is equal to one joule per kilogram.

The response of tissues and organs varies for different types of radiation. The **equivalent dose** in a tissue or organ is the organ dose averaged over that tissue or organ, including a *radiation weighting factor* that varies by radiation type and is related to the density of ionization created. The international unit of equivalent dose is the sievert (Sv).

Also, tissues and organs have different sensitivities to radiation. An additional and frequently used concept is the **effective dose**, which is the sum of the organ dose to each organ multiplied by the *radiation weighting factor* mentioned above and a *tissue weighting factor* that takes into account the radiosensitivity of tissues and organs. The international unit of effective dose is also the sievert.

Absorbed dose is the appropriate quantity to refer to threshold doses for deterministic effects (i.e. tissue reactions). The equivalent and effective doses are radiological protection quantities that are only applicable to stochastic effects.