Health risk assessment

from the nuclear accident after the 2011 Great East Japan Earthquake and Tsunami

based on a preliminary dose estimation

World Health Organization

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Contents

Executive summary

Introduction

The earthquake and tsunami in Japan on 11 March 2011 led to releases of radioactive material into the environment from the Tokyo Electric Power Company's Fukushima Daiichi nuclear power plant.

A major release of radioactivity to the environment is always of concern, owing to potential acute and long-term health effects. Evidence from historic events confirms that any major uncontrolled release of radiation should be cause for immediate response and scientific assessment of potential health effects.

When such an event occurs, the World Health Organization's mandate, as described in the Joint Radiation Emergency Management Plan of the International Organizations, is to assess and respond to public health risks.

The primary purpose of this health risk assessment of the Fukushima Daiichi nuclear accident is to estimate its potential public health impact so that future health needs can be anticipated and public health actions can be taken. This assessment is based on a preliminary estimate of radiation doses, as described in a WHO report published in May 2012.

Methods

This health risk assessment was conducted by independent international experts who were selected by WHO for their expertise and experience in radiation risk modelling, epidemiology, dosimetry, radiation effects and public health. All experts completed a declaration of interests form. The group met in December 2011 and March 2012. At both meetings, observers were in attendance from the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), the International Labour Organization, and the Government of Japan. The observers participated in discussions and sharing of data but were not involved in the decision-making process.

The risk assessment was made using four steps:

- The specific radiation sources, such as different radionuclides and pathways of exposure, were identified (hazard identification).
- The types of harmful effects that could result were identified based on scientific knowledge about the relationships between radiation dose and biological effects (dose-response relationships).
- Based on the preliminary dose assessment, lifetime organ doses were estimated for the general population within geographical locations ranging from the most affected areas of Fukushima prefecture to the rest of the world. Based on available data on

occupational exposure assessed by the operator of the nuclear power plant, first-year organ doses were also estimated for emergency workers (exposure assessment).

- The lifetime risks of cancer was estimated for all solid cancers combined, and also for individual cancer sites most closely associated with radiation exposure and with a known dependence of the magnitude of risk on age-at-exposure (leukaemia, thyroid cancer and female breast cancer). The lifetime risks were estimated for both sexes and three different ages at exposure (1 year [infant], 10 years [child], and 20 years [adult]). Calculations of the cumulative risks for the 15 years following the accident were also performed. Health risks for male emergency workers were estimated for three different ages (20 years, 40 years, and 60 years) (risk characterization).

Findings

In view of the estimated exposure levels, an increased risk of cancer is the potential health effect of greatest relevance. The relationship between radiation exposure and lifetime risk of cancer is complex and varies depending on several factors, mainly radiation dose, age at time of exposure, sex and cancer site. These factors can influence the uncertainty in projecting radiation risks, in particular when assessing risks at low doses.

Outside the geographical areas most affected by radiation, even in locations within Fukushima prefecture, the predicted risks remain low and no observable increases in cancer above natural variation in baseline rates are anticipated.

Some health effects of radiation, termed deterministic effects, are known to occur only after certain radiation dose levels are exceeded. The radiation doses in Fukushima prefecture were well below such levels and therefore such effects are not expected to occur in the general population.

The estimated dose levels in Fukushima prefecture were also too low to affect fetal development or outcome of pregnancy and no increases, as a result of antenatal radiation exposure, in spontaneous abortion, miscarriage, perinatal mortality, congenital defects or cognitive impairment are anticipated.

In the two most affected locations of Fukushima prefecture, the preliminary estimated radiation effective doses for the first year ranged from 12 to 25 mSv. In the highest dose location, the estimated additional lifetime risks for the development of leukaemia, breast cancer, thyroid cancer and all solid cancers over baseline rates are likely to represent an upper bound of the risk as methodological options were consciously chosen to avoid underestimation of risks. For leukaemia, the lifetime risks are predicted to increase by up to around 7% over baseline cancer rates in males exposed as infants; for breast cancer, the estimated lifetime risks increase by up to around 6% over baseline rates in females exposed as infants; for all solid cancers, the estimated lifetime risks increase by up to around 4% over baseline rates in females exposed as infants; and for thyroid cancer, the estimated lifetime risk increases by up to around 70% over baseline rates in females exposed as infants. These percentages represent estimated relative increases over the baseline rates and are not estimated absolute risks for developing such cancers. It is important to note that due to the low baseline rates of thyroid cancer, even a large relative increase represents a small absolute increase in risks. For example, the baseline lifetime risk of thyroid cancer for females is just three-quarters of one percent and the

additional lifetime risk estimated in this assessment for a female infant exposed in the most affected location is one-half of one percent. These estimated increases presented above apply only to the most affected location of Fukushima prefecture. For the people in the second most affected location, the estimated additional lifetime cancer risks over baseline rates are approximately one-half of those in the highest dose location. The estimated risks are lower for people exposed as children and adults compared to infants.

In the next most exposed group of locations in Fukushima prefecture, where preliminary estimated radiation effective doses were 3–5 mSv, the increased lifetime estimates for cancer risks over baseline rates were approximately one-quarter to one-third of those for the people in the most affected geographical location.

Among Fukushima Daiichi nuclear power plant emergency workers, the lifetime risks for leukaemia, thyroid cancer and all solid cancers are estimated to be increased over baseline rates, based upon plausible radiation exposure scenarios. These scenarios and their corresponding estimated risks are detailed in the body of this report. A few emergency workers who inhaled significant quantities of radioactive iodine may develop non-cancer thyroid disorders.

Conclusions

This health risk assessment is based on the current state of scientific knowledge. The assessment models used were derived from previous radiation events and experience, which do not exactly match the pattern of exposure seen in Fukushima; thus, adjustments were required. The dose estimates and assumptions used in this assessment were deliberately chosen to minimize the possibility of underestimating eventual health risks. The values presented in the report should be regarded as inferences of the magnitude of the health risks, rather than as precise predictions. Moreover, it is also important to note that the exposure data upon which this report is based are preliminary and include only data that were available as of September 2011. Because scientific understanding of radiation effects, particularly at low doses, may increase in the future, it is possible that further investigation may change our understanding of the risks of this radiation accident.

This health risk assessment concludes that no discernible increase in health risks from the Fukushima event is expected outside Japan. With respect to Japan, this assessment estimates that the lifetime risk for some cancers may be somewhat elevated above baseline rates in certain age and sex groups that were in the areas most affected.

These estimates provide valuable information for setting priorities in the coming years for population health monitoring, as has already begun with the Fukushima Health Management Survey.

On the basis of these findings, the continued monitoring of food and the environment remains important. When additional dose estimations become available from studies undertaken by UNSCEAR and others, such data can be used to further refine these risk estimates.

Preface

The World Health Organization (WHO) conducts a programme on radiation and health that aims to promote safe and appropriate use of radiation to protect patients, workers and the general public in planned, existing and emergency exposure situations. WHO's involvement in radiation and health began within a decade of its founding, and the International Commission on Radiological Protection has been in official relations with WHO since 1956. In 1972 the World Health Assembly requested the WHO Director-General to cooperate with the International Atomic Energy Agency (IAEA), the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) and other international organizations in evaluating the world situation regarding the medical use of ionizing radiation and the effects of radiation on populations.

Global public health security is one of the key priorities of WHO's agenda. The World Health Assembly requested the Director-General in 2005 to enhance WHO's capacity to implement health-related emergency preparedness plans and to prepare for disasters and crises through timely and reliable assessments. The nature of WHO's work on emergencies – whether resulting from natural, intentional or accidental events – requires a high level of coordination with a variety of partners within the United Nations system, as well as with external partners. One of the lessons from the 1986 Chernobyl nuclear accident was the need to strengthen international cooperation in radiation emergencies. The Joint Radiation Emergency Management Plan of the International Organizations, last published in 2010, establishes the mechanisms for implementing a coordinated response and describes the roles of each party. Within this joint plan, WHO is responsible for the coordination of public health risk assessment and response.

The decentralized structure of WHO – with its headquarters in Geneva, Switzerland, six regional offices and 147 country offices – provides optimal conditions for interacting with the Organization's 194 Member States. After the 11 March 2011 Great East Japan Earthquake and Tsunami, Tokyo Electric Power Company's (TEPCO) Fukushima Daiichi nuclear power plant was severely damaged and a substantial amount of radioactive material was released into the environment. The potential risks of human exposure to radiation resulting from this accident received priority attention around the world. As the United Nations directing and coordinating authority on international public health issues, WHO was directly engaged in assessing and communicating public health risks.

Assessment of the health risks arising from this accident requires knowledge of the radiation doses delivered to populations within Japan and beyond. WHO undertook an initial assessment of radiation doses received by populations inside and outside Japan as a consequence of the Fukushima Daiichi accident, which was published in May 2012.

This report summarizes the results of a health risk assessment conducted by a group of independent experts convened by WHO. UNSCEAR, the International Labour Orga-

nization and the Government of Japan participated as observers. It represents the first international effort to estimate radiation risks from this accident at the global level.

The health risk assessment, which is based upon currently available preliminary data, gives an indication of the health implications of this accident. Such information can support the identification of needs and priorities for public health actions. This report is primarily intended for use by policy makers and health professionals in WHO Member States, as well as by international organizations.

1. Introduction

On 11 March 2011 Japan suffered a magnitude 9 earthquake, the largest ever recorded in the country. The 2011 Great East Japan Earthquake created a series of large tsunami waves that struck the east coast of Japan, causing widespread damage to infrastructure, including to several nuclear power plants (NPPs). In most cases these power plants were successfully shut down. However, at the Fukushima Daiichi NPP the earthquake and tsunamis knocked out the power supply to the facility, and consequently the means to control and cool the reactors. In the days that followed, reactor meltdown, venting and hydrogen gas explosions released radionuclides into the environment.

Public health actions to manage and reduce the negative consequences of this event were taken by authorities in Japan and by other national authorities around the world. In Japan, a 3-km evacuation zone was put in place around the site, which was then quickly increased to a 20-km evacuation zone, with a sheltering zone between 20 and 30 km. As the availability of environmental monitoring data increased, other protective actions were implemented to reduce doses in the longer term, including the relocation of people in some areas (designated as "deliberate evacuation areas"). Stable iodine for thyroid blocking was distributed but it is thought that only a small number of persons in specific locations actually consumed stable iodine, because consumption (as opposed to distribution) was not officially recommended in most places (*1*). Provisional regulatory limits for the radioactive content of food were promptly established, and food monitoring was conducted at the local level on the basis of testing guidelines prepared by the Government of Japan. This monitoring meant that food samples were tested before being supplied to the market in the early harvest season and those samples found to contain higher concentrations of radionuclides than the provisional regulatory limits were subjected to appropriate measures. Furthermore, in case the contamination was spread over an area, distribution restrictions were implemented for foods from that area. Similarly, monitoring of tap water was conducted by both the central and local governments and by the water supply utilities, with special emphasis on Fukushima and neighbouring prefectures.

Around the world, the primary concern of governments was to protect their citizens residing in or visiting the most affected regions of Japan in the days and weeks after the nuclear accident, but there was also consideration of whether any steps were needed within their own countries, such as restrictions on food imports from Japan.

1.1 Motivation

Since the onset of the nuclear accident, the health risk of human exposure to radiation has received priority attention around the world. In its role as the United Nations directing and coordinating authority on international public health issues, WHO was directly engaged in assessing and communicating public health risks from all three components of the disaster, i.e. the earthquake, tsunami and nuclear accident. In line with its defined

role in radiation emergency response among international organizations, WHO is responsible for public health risk assessment and response (*2*).

Soon after the accident, WHO developed a formal health risk assessment (HRA) to estimate the risks to human health from radiation exposure due to the Fukushima Daiichi NPP accident. The HRA requires dose estimates; therefore, WHO conducted a preliminary global dose estimation for the general public, which was published in May 2012 (*3*). Based on conservative assumptions, first–year effective doses were estimated to be below 10 mSv in most of Japan with a few exceptions, and well below 0.01 mSv in the rest of the world.

1.2 Purpose and audience

This HRA is based on preliminary dose estimates and is intended to give an indication of the radiation-related health implications of the Fukushima Daiichi nuclear accident. Such information can support the identification of needs and priorities for public health actions. The target audience includes policy makers and health professionals as well as relevant international organizations.

It should be noted that this report discusses health risks rather than health effects (see Box 1). It is not intended to provide estimates of the disease burden in the population or to calculate possible excess disease cases due to the radiation exposure resulting from this accident.

1.3 Scope

The scope of the HRA includes the general population in Fukushima prefecture, the rest of Japan and around the world, and the Fukushima Daiichi NPP emergency workers, i.e. employees of the Tokyo Electric Power Company (TEPCO) and contractors exposed during the emergency phase. It does not include first responders (e.g. police, fire fighters, and Japan self-defence forces) because the information about their radiation doses was not available to the HRA Expert Group within the timeframe of its work.

The general population groups are defined by geographic location, age and sex. Four distinct geographical areas are identified based on preliminary estimated doses. The geographical coverage includes the whole world, with greater spatial detail in the estimated risks presented for Japan, and in particular for the Fukushima prefecture. Age groups considered are 1-year-old infants, 10-year-old children and 20-year-old adults.

Box 1. Health effects versus health risks

Health effects are changes in the health status of an individual or population, identifiable either by diagnostic or epidemiological methods.

Health risks express the likelihood or probability of a health effect to occur under defined circumstances and exposure to a certain hazard, e.g. radiation. Risks are estimated using available data and mathematical models.

Health risks from exposure *in utero* are considered in the risk characterization but are not quantitatively assessed.

For the emergency workers, the assessment considers male workers in age groups of 20-, 40- and 60-year–olds. The exposure assessment is based on dosimetric reports from the Japanese government and TEPCO.

This report examines a number of cancer and non-cancer health endpoints, on the basis of the estimated doses. The assessment considers separately specific cancer sites regarded as being more radiosensitive and with potentially higher risks at younger ages-at-exposure (*4*) – i.e. leukaemia (*5*), thyroid cancer (*6*) and breast cancer (*7*) – plus all solid cancers combined. It also covers non-cancer health effects, such as thyroid diseases, cardiovascular diseases and lens opacities.

This assessment focuses on radiation-related health risks. The psychological impact, recognized as the largest public health issue after the 1986 Chernobyl nuclear accident, is considered and discussed. However, it is not quantitatively assessed as the evaluation of social and psychosocial hazards and their risks to health requires different approaches, such as a Health Impact Assessment[1].

1.4 Overview of the process

This report is focused on the first of the three components of a risk analysis process, which are risk assessment, risk management and risk communication (see Figure 1) (*8*). Risk assessment predicts the likelihood of occurrence of adverse events based on scientific evidence. It does not attempt to indicate the level of risk that can be considered acceptable, or the appropriate level of public health protection. These considerations are within the scope of risk management. The third component, risk communication, is also an essential part of this process.

The HRA process is typically described as consisting of four basic steps: hazard identification, dose-response assessment (or more general hazard characterization), exposure assessment and risk characterization. These steps are defined below.

Hazard identification: This first step in a risk assessment is the identification of the type and nature of adverse effects that an agent can cause in a population, based on studies in humans and laboratory animals. In the context of this report, hazard identification is the process used to identify the specific radiation sources (i.e. radionuclides) and the type of harm they could cause.

Dose-response relationship: This second step examines the relationship between exposure to a particular agent and any adverse health effects in humans as a result of this exposure. The relationship is usually based on existing evidence from epidemiological studies that describe the endpoints for adverse human health effects at relevant exposures and the dose-response relationships for the different endpoints. In the context of this report, the endpoints considered include cancer as well as non-cancer risks.

Exposure assessment: This step gathers information about how much of a particular substance different groups have been exposed to, how the exposure took place (i.e. through

1. A Health Impact Assessment (HIA) is a means of assessing the health impacts in diverse economic sectors using quantitative, qualitative and participatory techniques (for more information, see http://www.who.int/hia/en/).

Figure 1. Risk analysis

Adapted from "Principles and methods for the risk assessment of chemicals in food". Environmental Health Criteria, No. 240. Geneva, World Health Organization, 2009.

which exposure pathways) and for how long the exposure occurred. In the context of this report, doses for the general population (*3*) as well as emergency workers are considered.

Risk characterization: This last step of the risk assessment process integrates the information collected in the previous steps to estimate qualitatively or quantitatively the risk of adverse health effects (i.e. cancer and non-cancer risks) under defined exposure conditions. In the context of this report, the risk characterization includes the quantitative estimation of specific cancer risks. Risk characterization takes into consideration the influence of several parameters, such as sex, age at the time of exposure, and attained age. Non-cancer risks are qualitatively assessed.

The report is organized in sections as shown in Figure 2.

Figure 2. Organization of this report

Introduction	**Chapter 1**	What is the motivation, purpose, scope and process for this health risk assessment?
Hazard identification	**Chapter 2**	What is the agent and what health problems are potentially caused by the agent?
Dose-response relationship	**Chapter 3**	What are the health problems at different exposure levels?
Exposure assessment	**Chapter 4**	What exposures are likely to occur, and what is the resulting dose to humans?
Risk characterization	**Chapter 5/6**	What is the estimated human health risk from the exposure? What are the uncertainties?
Public health considerations	**Chapter 7**	What are the public health implications and policy options?
Summary and conclusions	**Chapter 8**	What are the key findings?

1.5 Procedures

A group of experts (the HRA Expert Group) was convened in December 2011 to carry out an assessment of the possible range of health risks expected as a result of the human exposure to radiation due to the Fukushima Daiichi NPP accident. The HRA Expert Group consisted of independent experts, selected by WHO on the basis of their scientific competence and experience, and representatives from WHO, including the International Agency for Research on Cancer (IARC). The HRA Expert Group included experts on radiation risk modelling, epidemiology, dosimetry, radiation effects and public health. The experts, whose profiles are given in Annex A, were required to disclose any potential conflicts of interest (see Annex B).

UNSCEAR is conducting, over 2 years, an assessment of the exposure levels and effects of the Fukushima accident on humans and the environment. Its main scientific report will be submitted to the United Nations General Assembly in 2013. UNSCEAR participated in the HRA Expert Group as an observer to ensure compatible approaches and data sources for the two United Nations assessments. Close cooperation was maintained while the two assessments were in progress. Because the report includes an assessment of health risks for workers, the International Labour Organization (ILO) participated as an observer.

Collaboration with the Government of Japan and relevant Japanese institutions was deemed to be important for the successful completion of the work as they provided data for HRA. These representatives were observers at the meetings.

The HRA Expert Group met on two occasions in Geneva (December 2011 and March 2012) and communicated electronically. The HRA Expert Group agreed on the most appropriate dose-response models for estimating the health risks, considering different age groups and adverse health effects, and taking into account possible effect modifiers and other population characteristics.

The HRA Expert Group used, where possible, the existing evidence and the most widely accepted knowledge on the nature and probability of the effects that was available. Evidence-based decision making and expert consensus by unanimous agreement was achieved whenever possible. In a few cases decision making was based on majority agreement, with consideration of dissenting opinions and their rationale. For the general population, the HRA Expert Group considered the dose estimates provided by an international expert panel (the Dose Expert Panel), established by WHO in June 2011 (*3*). For the Fukushima Daiichi NPP workers, dose estimates were provided by the Government of Japan and by TEPCO.

2. Hazard identification

This chapter discusses the main radionuclides released during the Fukushima Daiichi NPP accident. It then describes current knowledge on health hazards from ionizing radiation as identified by key research findings, including those on cancer and non-cancer diseases. The relationship between these diseases and the dose is discussed in Chapter 3.

2.1 Identification of the source term

The amount and type of radionuclides released during a nuclear accident is called the source term. An early estimate of the source term was used in the preliminary dose estimation of the Fukushima accident published by WHO (*3*). The amount of radionuclides released during the accident and deposited around Japan was evaluated using both environmental monitoring data and computer simulation based on atmospheric dispersion modelling of radioactive materials (*9,10*). The basis for the estimation of the source term includes operational records, observed parameters and the chronology of events at the site.

Actual environmental measurements showed variability in the radionuclide composition for different locations in Japan. In the WHO preliminary dose assessment, the relative isotopic composition of the radioactive deposits on the ground was assessed in Japan on the basis of soil contamination. Two assumed radionuclide compositions were used (see Table 1).

Table 1. Assumed relative isotopic composition of the radioactive deposits on the ground (on 15 March 2011) from (*3*). The two approaches are based on publicly available data.

Radionuclide	Approach A[a]	Approach B[b,c]
^{131}I	7.8	11.7
^{132}I	7.6	–
^{132}Te[d]	7.6	8.0
^{134}Cs	0.92	0.94
^{136}Cs	0.16	0.2
^{137}Cs	1	1
^{140}Ba	–	0.1
^{110m}Ag	–	0.01
^{129m}Te	–	1.5

a. *Readings of soil monitoring around Fukushima NPP*. Ministry of Education, Culture, Sports, Science and Technology (http://radioactivity.mext.go.jp/en/ accessed 13 May 2012).

b. *Synthèse des informations disponibles sur la contamination radioactive de l'environnement terrestre japonais provoquée par l'accident de Fukushima Daiichi*. Paris, Institut de Radioprotection et de Sûreté Nucléaire, 13 juillet 2011.

c. *Interim report on radiation survey in Iitate village area conducted on March 28th and 29th*. 4 April 2011 (http://www.rri.kyoto-u.ac.jp/NSRG/seminar/No110/Iitate-interim-report110404.pdf, accessed 13 February 2013).

d. ^{132}Te (Tellurium-132) is important the first few days after a nuclear accident. It has a half-life of 3.2 days and decays to ^{132}I (iodine-132), which has a half-life of 2.3 hours.

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:

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).

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:

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

Multiplicative transfer:

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

or a weighted arithmetic sum of both:

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

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.

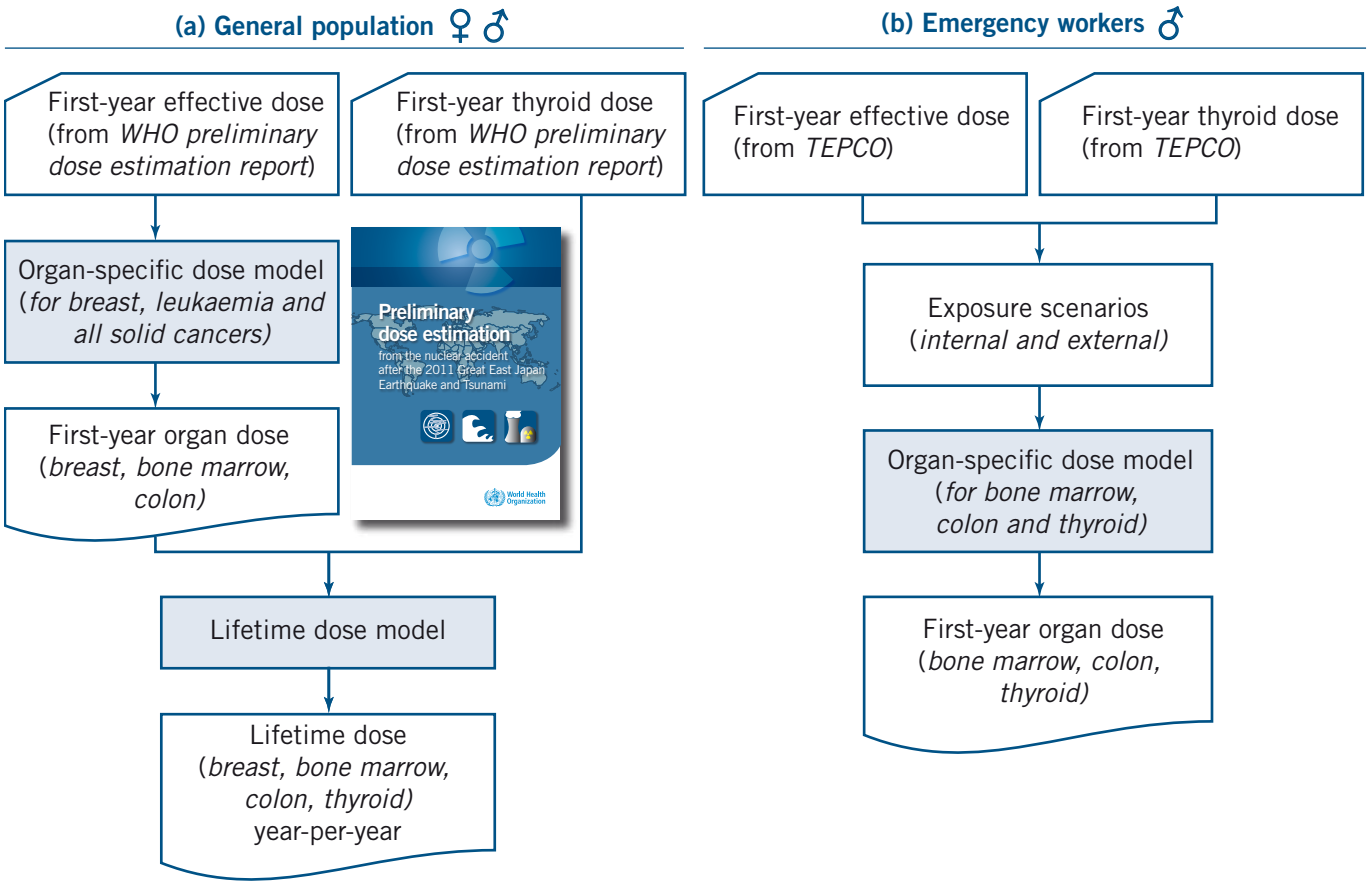
4. Exposure assessment

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



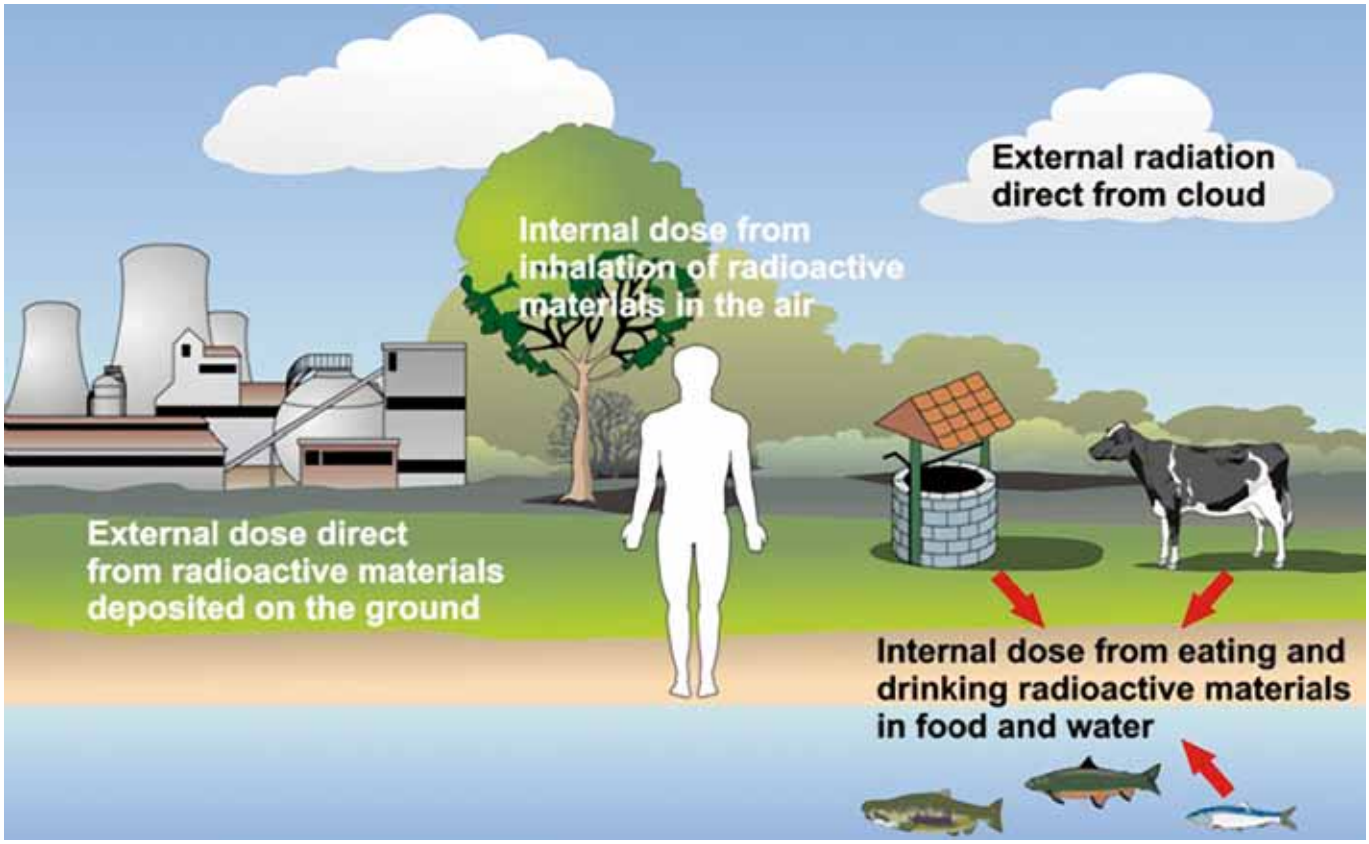
4.1.1 Pathways of exposure for the general population

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.

The dosimetric endpoints assessed by the Dose Expert Panel were effective doses and equivalent doses to the thyroid, resulting from the exposure during the first year after the accident. An explanation of dosimetric quantities is shown in Box 6. Three age-at-exposure groups were included in the dose assessment: adults aged 20 years, children aged 10 years, and infants aged 1 year. Doses to 6-month-old infants were considered for the consumption of infant formula made up with water. Doses to the fetus and breastfed infant were considered by the Dose Expert Panel but were not evaluated separately.

4.1.2 WHO preliminary dose estimation for the first year following the accident

The WHO preliminary dose estimation relies on measurements available as of mid-September 2011 and extrapolated to exposure in the first year. Additional data published later could not be incorporated because of the Dose Expert Panel's timeframe. This is further discussed in Chapter 6.

As far as possible, the Dose Expert Panel based its assessment directly on measurements of levels of radioactive material in the environment, such as levels of different radionuclides deposited on the ground or in soil, or found in foodstuffs. Inside Japan, the primary sources were official measurement data published by the Government of Japan. Such measurement data were not generally available for the rest of the world and consequently the Dose Expert Panel used environmental modelling predictions based on an estimated source term in combination with atmospheric dispersion modelling and environmental measurements to estimate doses outside Japan.

The assessment contained a number of assumptions that are described in detail in the dose assessment report. Although the assessment was intended to be realistic, given the limited information available to the Dose Expert Panel during its period of work, some conservative assumptions were adopted to avoid any underestimation of doses. For example, it was assumed that people consumed only food produced in the area where monitoring was implemented (e.g. those living in Fukushima ate only food produced in Fukushima). Moreover, some assumptions regarding the implementation of protective measures were conservative. For instance, it was assumed that relocation in the "deliberate evacuation area" took place at 4 months although the inhabitants of this area were subjected to relocation at different times earlier than this. It was also assumed that all the food monitored was on the market although the data set included the results of food samples that were collected for monitoring purposes and were not allowed on the market. In fact, food restrictions were introduced in Japan with the aim of banning from the market those food commodities produced in highly contaminated areas or exceeding regulatory limits. As a consequence of these conservative assumptions, some dose overestimation may have occurred.

In the preliminary dose estimation report, the Dose Expert Panel presented the estimated doses in order-of-magnitude dose bands of "characteristic" individual doses for each region considered. The main sources of uncertainty in the dose estimates and the implications of using conservative assumptions, possibly leading to dose overestimation, are extensively discussed in the dose assessment report. Some information published later, including in vivo measurements conducted in Fukushima prefecture, reported doses lower than the dose bands presented in the preliminary dose estimation report (*3*) although these are not directly comparable.

The key results of the estimated effective doses in the first year are as follows:

- In the most affected areas of Fukushima prefecture the estimated effective doses are within a dose band of 10–50 mSv.
- In the rest of Fukushima prefecture the estimated effective doses are within a dose band of 1–10 mSv.
- In prefectures neighbouring Fukushima, the estimated effective doses are within a dose band of 0.1–10 mSv.
- In all other Japanese prefectures, the effective doses are estimated to be within a dose band of 0.1–1 mSv.
- In the rest of the world, estimated effective doses are less than 0.01 mSv and are usually far below this level.
- The exposure pathways that contribute most to effective dose vary with location and distance from the site. In the more affected regions the external dose from groundshine is important, but with increasing distance from the site the ingestion of food becomes the main contributor.

The key results of the estimated thyroid doses in the first year are as follows:

- In the most affected area of Fukushima prefecture, the estimated thyroid doses are within the dose band of 10–100 mSv, with the exception of one example location where estimated thyroid doses to adults are within a dose band of 1–10 mSv and another example location where the upper bound of the estimated thyroid doses to infants is 200 mSv.
- In the rest of Fukushima prefecture, the estimated thyroid doses are within a dose band of 1–10 mSv for adults and 10–100 mSv for children and infants.
- In other Japanese prefectures, the estimated thyroid doses are within a dose band of 1–10 mSv for all age groups considered.
- In the rest of the world, estimated thyroid doses are less than 0.01 mSv, and are usually far below this level.
- The exposure pathways that contribute most to thyroid dose vary with location and distance from the site. In the more affected regions, inhalation from the cloud and the external dose from groundshine are important, but with increasing distance from the site (i.e. when overall exposure is very low) the ingestion of food becomes the main contributor.

For the purposes of this HRA the HRA Expert Group was provided with the detailed results of the first-year exposure assessment, which included the actual calculations and the point estimates used to create the dose bands. Four distinct geographical areas were identified based on estimated doses, as described below.

- Group 1: the two locations within Fukushima prefecture with effective doses of 12–25 mSv;
- Group 2: locations in Fukushima prefecture where effective dosesare between 3 and 5 mSv;
- Group 3: the less-affected locations of Fukushima prefecture and the rest of Japan, where effective dose values are around 1 mSv;

- Group 4 – the neighbouring countries and the rest of the world, where effective doses are well below 1 mSv.

4.1.3 Calculation of first-year organ doses

An important contribution to the effective dose in the Fukushima Daiichi NPP accident is the internal exposure to isotopes of caesium. Since the bio-distribution of caesium in the body is quite homogeneous, all organs are almost equally irradiated, and therefore the effective dose is a good indicator of organ doses. In addition to the effective dose, the Dose Expert Panel assessed organ doses to the thyroid because the intake of ^{131}I is also likely to be an important contributor to overall exposure and in this case the distribution in the body is far from uniform, with the thyroid being the most exposed organ.

Although the HRA Expert Group agreed with the above, it was considered appropriate to use organ doses rather than effective doses to estimate health risks. The decision was made based on the fact that effective dose is primarily intended for use when nominal risk coefficients and tissue-weighting factors for age- and sex-averaged worldwide populations are applied (e.g. radiation protection), while the organ-absorbed dose is more appropriate for risk assessments on specific populations when age and sex-specific models are used to transfer risks (*12,94*).

It was decided to calculate organ doses for red bone marrow, thyroid, breast, and colon, as input data for the cancer risk models for leukaemia, thyroid cancer, breast cancer and all solid cancers, respectively. The colon dose had already been used as a surrogate for whole-body dose within the LSS cohort. The methodology used to calculate organ doses for the first year after the accident is described in Annex G.

The effective dose is the summation of all the organ equivalent doses, each multiplied by its appropriate tissue weighting factor. Values expressed as the ratio between the absorbed dose and the effective dose in each of the organs mentioned above were calculated. The estimations of organ doses for red bone marrow, breast and colon were performed by applying those ratios to the effective dose values for adults, children and infants. This approach was validated by comparison with the thyroid doses estimated by the Dose Expert Panel.

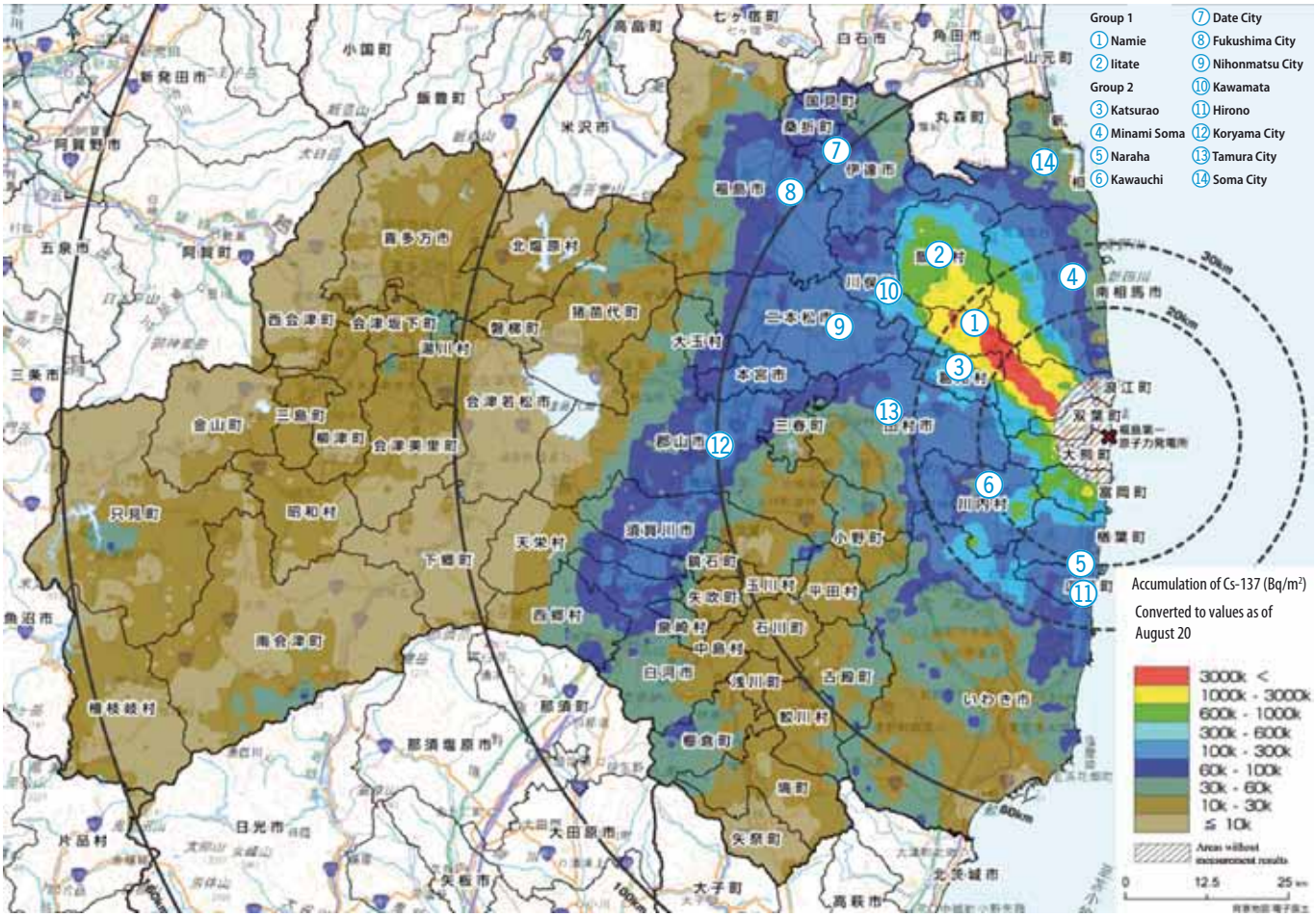
The first-year organ doses are reported in Tables 5 and 6 for the general population in the four distinct geographical areas representing the world. The example locations considered in the Fukushima prefecture are indicated in Figure 5. Table 5 shows the first-year organ doses for colon, breast and bone marrow estimated by the HRA Expert Group. Table 6 shows the first-year thyroid organ doses[3] assessed by the Dose Expert Panel and used as input data for the thyroid cancer risk model for the purposes of the HRA.

4.1.4 Calculation of lifetime doses

The WHO Preliminary dose estimation report presents doses from the first year after the Fukushima accident, based on data available up to mid-September 2011. As such, it includes extrapolations to estimate 1-year doses. The Dose Expert Panel considered that an estimation of doses beyond the first year would have resulted in a high degree of uncertainty.

3. For practical reasons, the first-year thyroid organ doses are presented in a separate table, because there are differences in the dose ranges and grouping of some locations compared to the other organ doses.

Figure 5. Locations in Fukushima prefecture considered in the assessment (Groups 1 and 2). Note that the rest of Fukushima (less affected) is part of Group 3.



Source: http://radioactivity.mext.go.jp/en/contents/4000/3168/24/1270_0912_2.pdf (Attachment 4 - Accumulation of Cs137 on the ground surface in Fukushima prefecture).

Lifetime doses in nuclear accidents

The radiation doses received in the second and subsequent years after a nuclear accident are expected to be considerably less than in the first year, even without application of remedial actions (*95*).

Experience from the Chernobyl accident showed that the radiation exposure decreased within the first year after the accident mainly due to radioactive decay of short-lived radionuclides (e.g. ^{131}I). Beyond the first year, the decrease was mainly due to radioactive decay of caesium and its migration into the soil (*93,96,97*). The shielding effect of this radionuclide migration in the soil was an important factor in reducing lifetime doses (*97*).

Besides the natural mechanisms mentioned above, the temporal distribution of the lifetime dose will also be influenced by a number of other factors, including additional protective measures (e.g. more stringent regulatory standards, such as those implemented for food) as well as long-term remedial actions (e.g. clean-up of buildings, remediation of soils and vegetation, treatment of agricultural fields, waste management), which would further reduce radiation exposure andconsequently lifetime doses.

Table 5. First year organ doses in the general population considered for the HRA (colon, breast and bone marrow).

Location Group	Locations	Organ dose for adults 20y (mSv)			Organ dose for children 10y (mSv)			Organ dose for infants 1y (mSv)		
		Colon	Breast	Bone marrow	Colon	Breast	Bone marrow	Colon	Breast	Bone marrow
Group 1	① Namie Town[a]	22	23	21	25	25	25	26	27	26
	② Iitate Village[a]	12	13	12	14	14	14	15	15	15
Group 2	③ Katsurao Village[a]	5	5	4	5	5	5	5	5	5
	④ Minami Soma City	5	5	5	5	5	5	5	5	5
	⑤ Naraha Town ⑥ Kawauchi Village ⑦ Date City ⑧ Fukushima City ⑨ Nihonmatsu City	4	4	4	4	4	4	5	5	4
	⑩ Kawamata Town ⑪ Hirono Town ⑫ Koriyama City ⑬ Tamura City ⑭ Soma City	3	3	3	3	3	3	3	3	3
Group 3	Rest of Fukushima prefecture (less affected)	1	1	1	1	1	1	1	1	1
	Neighbouring prefectures	1	1	1	1	1	1	1	1	1
	Rest of Japan	1	1	1	1	1	1	1	1	1
Group 4	Neighbouring countries	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
	Rest of the world	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01

a. Organ dose from the first 4 months after the accident only

Note: The first year organ doses shown here as rounded values were calculated by the HRA Expert Group on the basis of the point estimates of effective dose used to create the dose bands presented in the WHO Preliminary dose estimation report (*3*).

Ratio of lifetime dose to 1-year dose

For the internal dose, the dose calculated from inhalation for the first year is treated as non-recurring[4]. Although there might indeed be some additional dose owing to re-suspension, this is not considered an important pathway for the radionuclides released by the accident at the Fukushima Daiichi NPP. For the dose from ingestion of food, there will be an additional long-term dose owing to contamination of food crops and fodder by the soil-root and other pathways.

4. For this reason, for the calculation of the lifetime dose no contribution from the inhalation pathway was considered beyond the first year

Table 6. First year thyroid doses for the general population

Location Group	Locations	Thyroid dose for adults 20y (mSv)	Thyroid dose for children 10y (mSv)	Thyroid dose for infants 1y (mSv)
Group 1	① Namie Town[a]	63	95	122
	② Iitate Village[b]	34	52	73
Group 2	③ Katsurao Village[c]	17	28	48
	④ Minami Soma City	16	25	43
	⑤ Naraha Town ⑥ Kawauchi Village ⑦ Date City ⑧ Fukushima City ⑨ Nihonmatsu City ⑩ Kawamata Town	14	22	39
	⑪ Hirono Town ⑫ Koriyama City ⑬ Tamura City ⑭ Soma City	11	18	35
Group 3	Rest of Fukushima prefecture (less affected)[2]	8	15	31
	Neighbouring prefectures (Chiba, Gunma, Ibaraki, Miyagi, Tochigi)[3]	≤4	≤5	≤9
	Rest of Japan	~ 1	~ 1	~ 1
Group 4	Neighbouring countries	<0.01	<0.01	<0.01
	Rest of the world	<0.01	<0.01	<0.01

a. Organ dose from the first four months after the accident only

b. Although the preliminary estimated thyroid doses in this area (i.e. rest of Fukushima prefecture, less affected) are higher than the thyroid organ doses in the other locations in Group 3, the HRA Expert Group agreed to keep them within this group because the preliminary dose estimation was performed under very conservative assumptions and it is considered that in practice doses are much lower.

c. These are the rounded values for southern tip of Miyagi prefecture, and the other zones of the neighbouring prefectures are below those values

Note: These first year thyroid doses, calculated by the Dose Expert Panel, are the point estimates used to create the dose bands presented in the WHO Preliminary dose estimation report (*3*).

For the external dose, the Dose Expert Panelconsidered the long-term movement of caesium into the soil with the accompanying reduction in the external gamma-dose rate. This reduction factor, $r(t)$ is given in the equation below:

$$r(t) = 0.34 \times e^{-0.693 \times \frac{t}{1.5y}} + 0.66 \times e^{-0.693 \times \frac{t}{50y}}$$

where 34% of the exposure rate (groundshine) is projected to disappear with a half-time of 1.5 years and the remaining 66% is projected to disappear with a half-time of 50 years. These two half-times resulting from migration of caesium in the soil are in addition to the rate of disappearance of caesium due to its natural radioactive decay.

This equation was evaluated for the time periods of 1 year and 50 years and four radionuclides – ^{132}Te, ^{131}I, ^{134}Cs and ^{137}Cs (^{137m}Ba)[5]. The theoretically calculated ratio of long-term dose to 1-year dose was 7. An estimation was made using a similar approach during the initial UNSCEAR assessment of the Chernobyl accident (*98*), and a ratio of 8 was calculated. However, 20 years after the Chernobyl accident, the ratios predicted by this equation proved to be overestimated and the ratio of projected long-term dose to 1-year dose was identified as 3 (*99*).

This reduction of the theoretically calculated ratio from 7 to 8 to the ratio of 3 based on the Chernobyl experience is quite reasonable if the effects of countermeasures are considered. The reduction factor noted in the equation above comes from observations after Chernobyl of external dose rate measured over an undisturbed open field, i.e. not considering any remedial action. In urban environments the longer-term dose rates can be reduced by a factor varying from 1.5 to 15 (*93*) through efforts ranging from rather simple to strenuous.

The radiation doses received in the second and subsequent years after a nuclear accident are expected to be considerably less than in the first year, even without the application of remedial actions (*95*). A number of remedial actions were taken by the Government of Japan, municipal authorities and residents quite soon after the accident to lower radiation exposure (*100*), which will further lower lifetime radiation exposure resulting from this accident.

The Chernobyl experience showed that the ratio of long-term to 1-year dose was projected to be 3, with inclusion of data up to 20 years after the accident (99). On the basis of this experience, which appeared to be the most relevant, and taking into consideration the differences between the Chernobyl and Fukushima Daiichi NPP accidents, the HRA Expert Group considered it reasonable to assume that the ratio of long-term dose to 1-year dose would be equal to 2 and that the result should be treated as a lifetime dose commitment. Therefore, for purposes of calculating lifetime risk, it was agreed that the dose over the lifetime should be approximated to be twice[6] as much as the first-year dose. The results were provided on a year-per-year basis to the risk modellers for the health risk calculations. Figure 6 illustrates the distribution of the annual doses delivered to an organ over a lifetime using colon dose as an example.

4.2 Doses for the NPP emergency workers

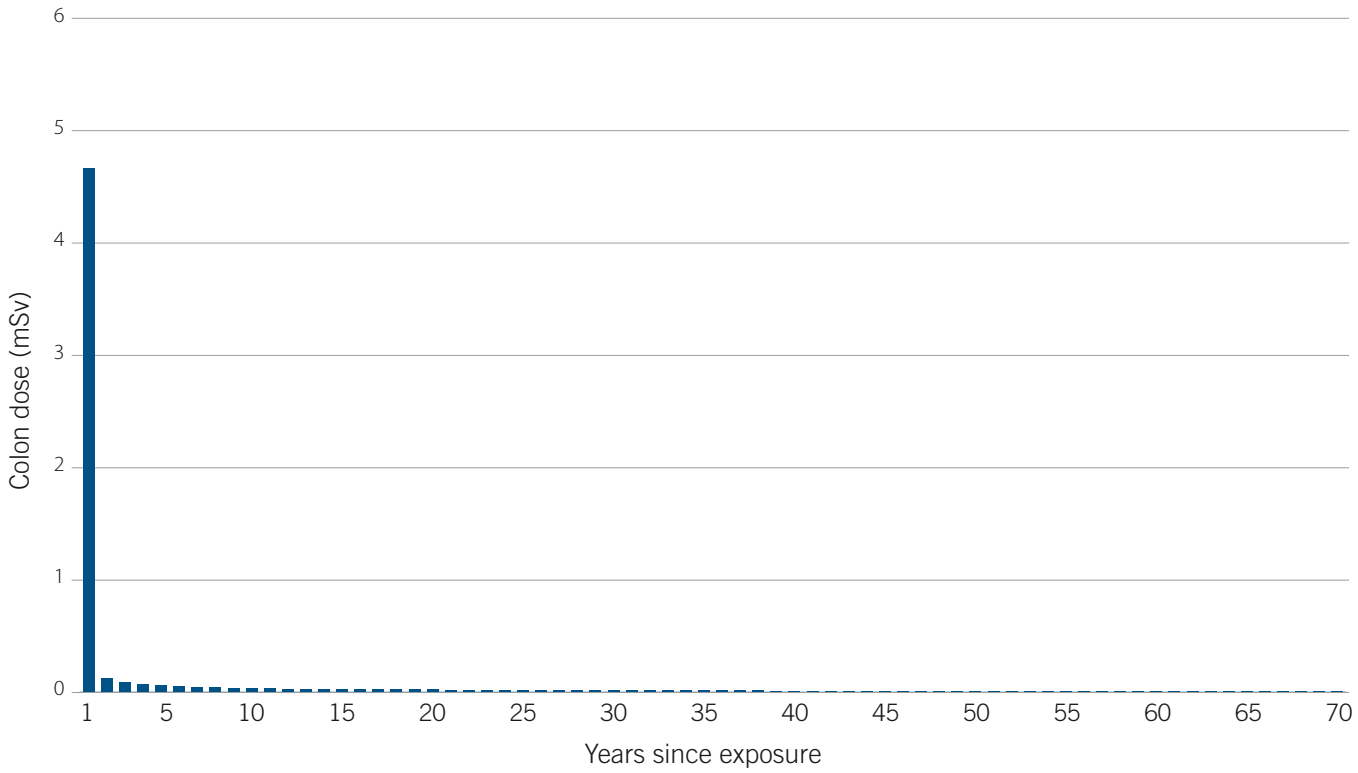
Recognizing that occupational health is closely linked to public health, WHO is addressing all determinants of workers' health[7], including risks for disease and injury in the occupational environment. Taking into account that disease and injury in the workplace comprise both normal and accidental exposure situations, the health risk assessment of

5. ^{137m}Ba is metastable barium

6. With an exception for the locations where people were relocated (i.e. Namie town in Futaba county, Iitate village in Soma county and Katsurao village in Futaba county). In those locations it was assumed that relocation took place at 4 months after the accident. Therefore the dose over the lifetime was calculated as the sum of the doses received during the first 4 months after the accident plus the lifetime dose calculated for the locations within Fukushima prefecture zone 1 (western least contaminated).

7. WHO is implementing a Global Plan of Action on Workers' Health 2008-2014 endorsed by the World Health Assembly in 2007 http://www.who.int/occupational_health/en/

Figure 6. Calculated distribution of the annual doses delivered to an organ over lifetime (70 years).
This example shows the lifetime distribution of the colon dose in a location where the first year organ dose is around 5 mSv



the emergency workers exposed during the emergency phase of the Fukushima Daiichi NPP accident was included in the scope of the present HRA.

The preliminary dose assessment conducted by the Dose Expert Panel (*3*) provided doses delivered to the general population and did not include occupational doses. Evaluation of occupational radiation exposure requires a different dosimetric approach.

Occupational radiation exposure is generally assessed retrospectively:

- for internal exposures, through individual bioassay monitoring either by in vivo direct measurements (e.g. whole-body counting, thyroid monitoring) and/or by in vitro assays (analysis of material excreted or removed from the human body);
- for external exposures, through personal dosimetry using monitoring devices ("personal dosimeters", e.g. thermoluminescent dosimeters [TLDs]).

In contrast, public exposure is commonly assessed prospectively through the application of dosimetric models, using environmental monitoring data as the input. While for the general population, both inhalation and ingestion are important routes of internal exposure, in the case of workers occupationally exposed to radiation, inhalation is the major route of internal exposure.

To evaluate health risks related to occupational exposure, the HRA Expert Group agreed to base its assessment on the occupational doses estimated by the operator TEPCO because this was the only exposure data available at the time of this assessment (Figure 3b). This HRA is focused on emergency workers employed by TEPCO or contractors.

Other categories of workers who may have been exposed to radiation during the response to the accident (e.g. rescue workers, firemen, policemen, self-defence forces, volunteers, government and municipal employees) were not included in the HRA because the information about their radiation doses was not available to the HRA Expert Group within the timeframe of its work.

Included in the exposure assessment, as reported by TEPCO in April 2012, are 23 172 emergency and mitigation workers, including 5 639 TEPCO employees (24%) and 17 533 contractors (76%). The data were provided to the HRA Expert Group in an anonymized way that ensured protection of the identity and privacy of the individuals concerned.

Owing to the extremely complex situation following the earthquake, tsunami and nuclear accident, the collection and reconstruction of data regarding workers' dosimetry are ongoing processes. Therefore, the estimates of workers' doses presented in this chapter should be considered as preliminary in nature.

4.2.1 Pathways for workers' exposure

Occupational exposure to radiation of Fukushima Daiichi NPP workers included internal and external exposure through four major pathways:

- internal exposure from inhalation of radioactive material in the workplace
- external exposure from radioactive material deposited in the workplace
- external exposure from radioactive material suspended in the workplace air
- external exposure from proximity to radiation sources within the damaged reactors.

Some workers were also exposed to radiation from radioactive material deposited on the skin or clothes (external contamination).

4.2.2 Radiation protection of female workers

Female workers are not considered in this HRA. Although most of the workers involved in the emergency response work at Fukushima Daiichi NPP were male, a few female workers were involved at an early stage after the earthquake. In May 2011 it was reported that two female workers had exceeded a cumulative effective dose of 5 mSv in 3 months, which is a regulatory limit established in Japan for female workers. TEPCO took measures so that working conditions for females would ensure that those limits were not exceeded again (e.g. working environment, personal protective equipment and alarms in the personal dosimeters pre-set at 4 mSv cumulative dose) (*101*).

4.2.3 Workers' exposure assessment reported by TEPCO

The assessment of exposure for emergency workers at the Fukushima Daiichi NPP was undertaken by TEPCO. The results were given by TEPCO to the Japanese authority to be considered by the HRA Expert Group in the preparation of this report. These results include information about effective dose ranges, mean effective doses and maximum effective doses for more than 23 000 workers from different age groups.

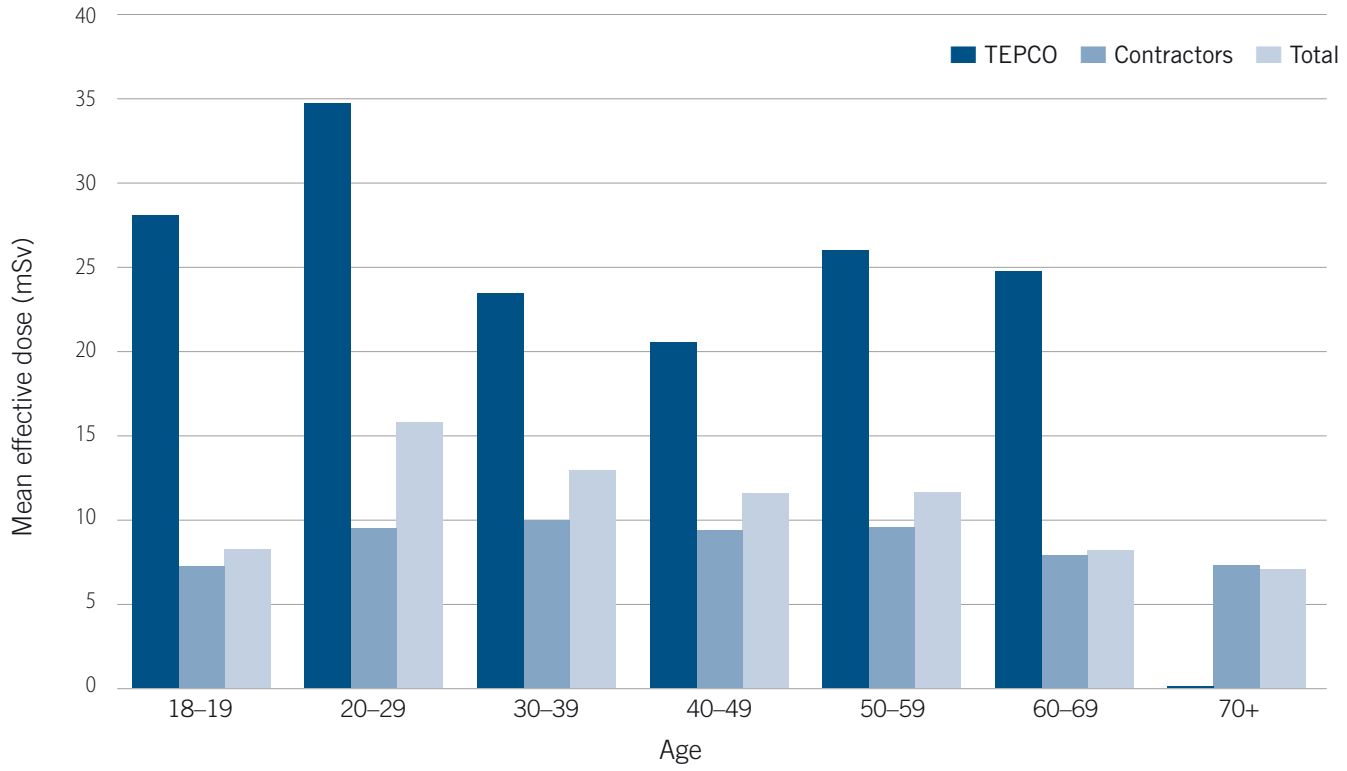
Table 7 shows the age distribution of the emergency workers considered in this HRA. Data from monitoring workers are available on TEPCO's website and are summarized in Annex H. In particular, information was provided on the contribution of internal and

external exposure to the total effective dose (Table 8), as well as some information about the radionuclides involved (see Table 25 in Annex H). Ranges of thyroid doses based on individual measurements taken on 522 of the most highly exposed workers were given separately (see Table 24 in Annex H).

Table 7. Age distribution of workers as of 31 January 2012

Age distribution	TEPCO	Contractors	Total
80	0	1	1
70-79	1	24	25
60-69	27	1831	1858
50-59	693	4716	5409
40-49	1173	4720	5893
30-39	925	3254	4179
20-29	511	1546	2057
18-19	3	61	64
Unknown	6	611	617
Total	**3339**	**16764**	**20103**
Oldest age	73	84	84
Youngest age	19	18	18

Figure 7. Dose distribution of workers by age group (data provided by TEPCO)



After the earthquake and tsunami there was a shortage of monitoring equipment. At the early stage of the emergency response, groups of workers were provided with a single personal dosimeter and the resulting measurements were taken to be representative of the external doses received by all members of the group. Once monitoring equipment was available for all workers, external dose assessment was based on the measurements of the individual personal dosimeters.

Based on the results of the internal dose estimation, TEPCO concluded that workers with the highest internal doses were those working in a central control room. For these workers, ^{131}I was the major contributor to internal dose (e.g. 98% for the worker with the highest internal dose). Stable iodine tablets were distributed to emergency workers beginning 13 March 2011. So far, no health effects have been observed for workers exceeding the dose limits.

A summary of the percentages of workers for different effective dose ranges as reported by TEPCO is presented in Table 8. Figure 7 shows the mean effective dose distribution as a function of age for TEPCO workers and contractors. Further data provided by TEPCO are presented in Annex H.

Table 8. Summary of the percentages of workers having received different effective dose ranges. For more detailed information on workers doses, see tables provided in Annex H

Effective dose range (mSv)	Internal exposure (% of workers)	External exposure (% of workers)	Total effective dose (% of workers)
< 10 mSv	> 95%	68.69%	66%
10 – 50 mSv	4.5%	28.23%	30%
50 – 100 mSv	0.3%	2.71%	< 4%
100 – 200 mSv	< 0.05%	0.37%	< 1%
> 200 mSv	< 0.05%	0%	< 0.05%

4.2.4 Exposure scenarios for workers at the Fukushima Daiichi NPP

The HRA Expert Group considered that the preliminary HRA for workers should not be based on individual doses and that it would be more appropriate to assess health risks in a few plausible exposure scenarios. As a result, four exposure scenarios were developed as shown in Table 9 below. Health risks were determined for each of these four sets of doses.

- Scenario 1 represents a group of around two thirds of the emergency workers with a total effective dose of 5 mSv as a "reasonably conservative" value.
- Scenario 2 represents about one third of the emergency workers with a total effective dose of 30 mSv.
- Scenario 3 represents less than 1% of emergency workerswith a total effective dose of 200 mSv.
- Scenario 4 represents a few emergency workers with a total effective dose of 700 mSv who received high doses to the thyroid gland from ^{131}I intake and low to moderate doses to other tissues. This scenario can be taken to be representative of the maximum exposure of emergency workers.

Table 9. Exposure scenarios assumed for the workers' health risk assessment

Scenario	Total effective dose (mSv)	External exposure (mSv)	Internal exposure (mSv)	Comments
1	5	5	–	Around 70% of the workers have < 10 mSv total effective dose and many workers in this group may have much lower doses (close to zero or even zero), so 5 mSv effective dose was considered a reasonably conservative assumption for this scenario. These workers probably appeared on the scene later and were not exposed to high levels of ^{131}I. Therefore: the assumption is that any internal dose is due to inhalation of ^{134}Cs and/or ^{137}Cs. Irrespective of the relative contribution of internal and external exposure, it is assumed that organ doses are equal to effective doses.
2	30	24	6	A total effective dose of 30 mSv is assumed with external exposure as the major contributor (80%) and internal exposure (20%) being all due to ^{131}I.
3	200	200	–	There are 75 workers with external effective doses > 100 mSv (the highest reported external dose is 199 mSv). It is assumed that there is no internal exposure to iodine and that organ doses are equal to effective doses.
4	700	100	600	There are 12 workers with internal effective dose > 100 mSv. The maximum reported total effective dose is 678.8 mSv and the maximum reported internal dose is 590 mSv (highest dose scenario). It is assumed that internal dose is entirely due to ^{131}I.

An objective of the HRA Expert Group was to provide estimates of health risk for emergency workers at the Fukushima Daiichi NPP from doses received during the emergency phase. Because there is no precise date when the emergency phase ended and an existing exposure situation was reached, the experts considered a reasonable approach to assess worker exposure for the first year only.

Thus, the question to be solved was how to convert the effective doses provided into doses to specific organs. The organs being considered were colon, red bone marrow, and thyroid. This HRA considered only male workers. Two different approaches were used to calculate organ doses for each of the exposure scenarios and the results were very similar (see Annex I). Approach A included the contribution to total dose from external exposure from immersion in a cloud but it did not consider the external exposure from radioactive material deposited in the workplace or radiation sources within the damaged reactors. Approach B addressed only the estimation of absorbed doses from intakes of radionuclides. Results for the estimated organ doses are presented in Table 10 for the four scenarios. Note that scenarios 1 and 2 cover more than 99% of workers and are

therefore more representative for this HRA. Scenarios 3 and 4 represent an upper bound in terms of internal and external exposure and cover less than 1% of workers. Note that the organ doses are very similar to the effective doses for Scenarios 1, 2 and 3. This is not the case for Scenario 4 where the dose to the thyroid is very high (around 12 Sv) while the doses to red bone marrow and colon are lower than the effective dose. The reason is that Scenario 4 assumes that most of the dose is due to ^{131}I. This dose level is an upper bound consistent with data provided by TEPCO about high thyroid doses in two workers (Annex H, Table 23).

Table 10. Estimated organ doses for the four scenarios assumed for the NPP workers (rounded values)

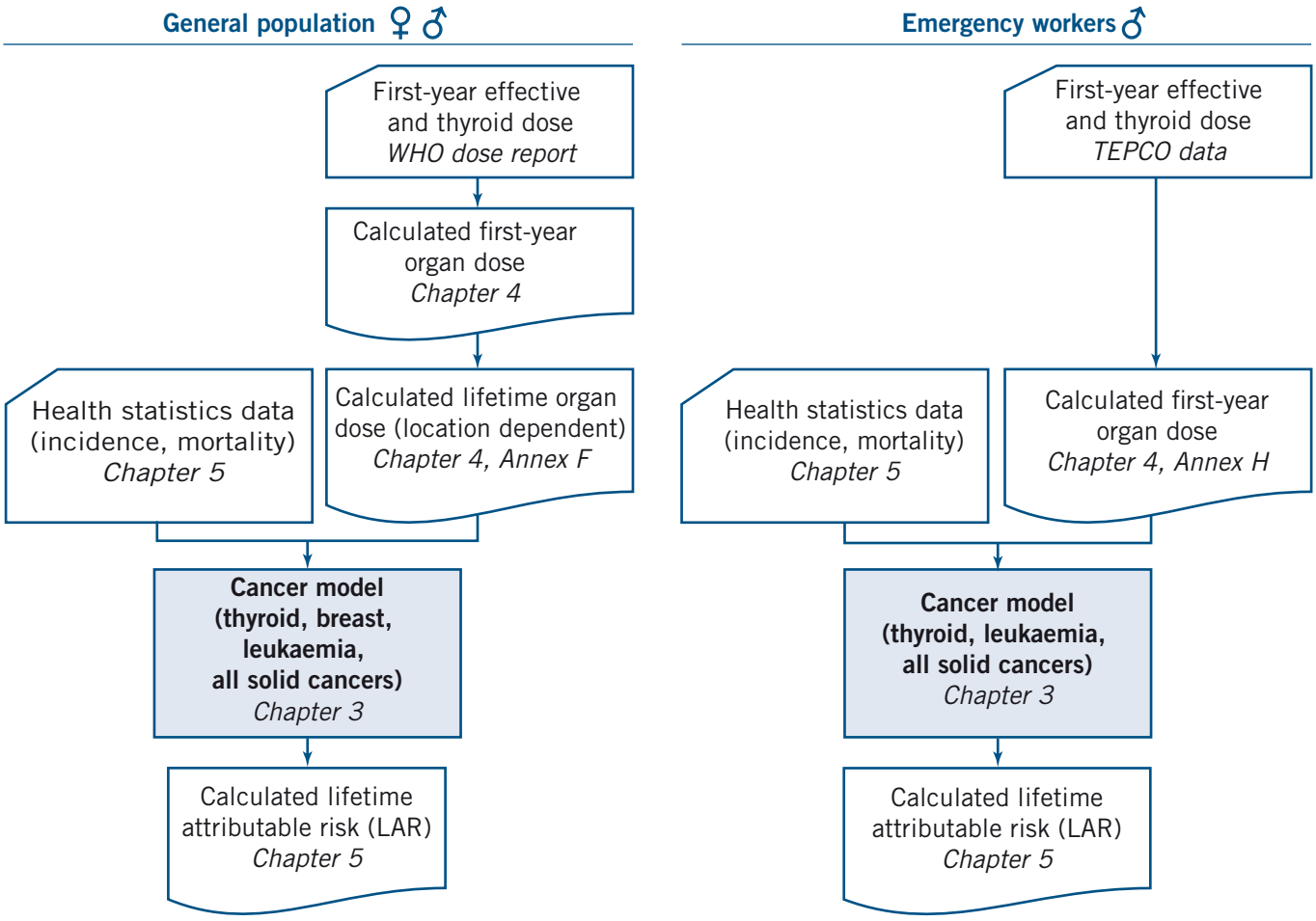
Scenario	Bone marrow (mSv)	Colon (mSv)	Thyroid (mSv)	Comments
1	5	5	5	This scenario covers around 69% of workers (~ 16 000 workers).
2	24	24	140	This scenario covers around 30% of workers (~ 7 000 workers).
3	200	200	200	This scenario represents less than 1% of workers (~ 200 workers).
4	100	100	11 800	This scenario represents an upper bound (a few workers).

5. Risk characterization

Risk characterization is the essential part of an HRA, where quantitative risk estimates are derived through the integration of the existing knowledge on the hazard (Chapter 2), the risk models (Chapter 3) and the dose estimates (Chapter 4). This last step in the risk assessment process is typically a quantitative statement about the magnitude and nature of risks derived by calculating the excess lifetime cancer risk associated with the estimated exposure (*102*). The general approach for the general population and for the workers is shown in Figure 8.

For this radiation risk characterization in particular, the aim is to provide estimates of radiation-related health risks derived from doses received by characteristic members of the general population and by workers occupationally exposed to radiation at the Fukushima Daiichi NPP, from the releases of radioactive material from 11 March 2011

Figure 8. General approach for characterizing the cancer risks for the general public and the workers



onwards, after the earthquake and tsunami in Japan. Cancer risks were quantitatively assessed and non-cancer risks were qualitatively discussed. The cancer sites considered for the risk characterization are leukaemia, thyroid cancer, female breast cancer, and all solid cancers. Risks are provided in terms of the probability of a premature incidence of a primary cancer from radiation exposure in a representative member of the public, a measure known as the lifetime attributable risk (LAR).

The general approach was to keep the risk assessment as simple as possible, given the uncertainty in dose estimates and the generally low doses involved. The next few sections give the full details of the input data and the results.

5.1 Input data

5.1.1 Dose for the general population

The exposure data used in this report are based on the levels of effective doses and thyroid doses calculated for the first year by the Dose Expert Panel that prepared the WHO Preliminary dose estimation report (*3*). The HRA Expert Group was provided with the detailed results of the exposure assessment including the point estimates, from which lifetime organ doses for thyroid, colon, breast and bone marrow were calculated (see chapter 4 for details). Lifetime organ doses D were used as input data to the cancer risk models for the calculation of LAR (see chapter 3). Distribution of lifetime organ doses were calculated on a year-by-year basis up to 70 years after exposure using the approach described in Chapter 4, section 4.1.4. Although for adults this 70-year period after exposure was enough to achieve the attained age of 89 years of age used in LAR, in the case of 1-year-old infants and 10-year-old children the 70-year period used for the calculation of their lifetime dose did not cover the entire period up to 89 years of attained age. However, the dose received beyond 70 years after exposure is very small (nearly zero) and will not influence the LAR calculations. An example of the temporal distribution of lifetime organ doses is presented in Figure 6, section 4.1.4.

As specified in section 4.1, the HRA Expert Group classified the geographical locations into four groups. It was agreed that health risks in terms of LAR would be calculated only for Groups 1 and 2, as the levels of dose estimated for all other locations were below the annual natural background level found in Japan, and the local variations in this level. It must be noted that the worldwide average annual effective dose from natural background radiation is about 2.4 mSv, with a typical range of 1–10 mSv in various regions of the world (*103*).

5.1.2 Dose for the emergency workers

The exposure input data for the HRA in workers were based on information provided by TEPCO, as described in section 4.2. Exposure data provided in terms of effective dose were used to calculate workers' organ doses for bone marrow and colon[1] in each of four assumed exposure scenarios. Only first-year organ doses were used as input data for the workers' HRA because the assessment is based on radiation doses related to the emergency exposure situation (i.e. emergency workers). Further occupational exposure

1. Organ doses for thyroid were already available as they had been included in the preliminary dose estimation.

to radiation beyond the first year will be considered under either an existing or a planned exposure situation, and will therefore be beyond the scope of this HRA.

5.1.3 Health statistics data

The following data are required as references for accurate calculation of LAR and LBR:

- age- and sex-specific all-cause mortality
- age- and sex-specific all cancer mortality (ICD 10 codes C00–C96)
- age- and sex-specific all cancer incidence (C00–C96)
- age- and sex-specific incidence for breast cancer (C50)
- age- and sex-specific incidence all solid cancers(C00–C89)
- age- and sex-specific incidence leukaemia (C91–C95)
- age- and sex-specific incidence thyroid (C73).

Mortality data for the general Japanese population (all-cause and cancer-specific) used to calculate overall survival $S(a)$ and cancer-free survival $S_{aj}(a)$ as a function of attained age a, were obtained from an official Japanese statistics website (http://www.e-stat.go.jp/SG1/estat/ListE.do?lid=000001082327). Cancer incidence data were taken from the 2004 Japan Cancer Surveillance Research Group compilation of 31 population-based cancer registries in Japan. Relying on data from 14 of those registries (excluding under-registered sites to avoid under-estimation), Matsuda *et al*. (*104*) published age-specific cancer incidence rates according to sex and primary site.

For the Fukushima prefecture and its cities and villages, no local cancer incidence rates were available at the time of this HRA, as the Fukushima cancer registry began data collection only in 2011. On the basis of the similarity of cancer incidence in two neighbouring prefectures for which cancer registries are available (Miyagi and Yamagata) and the other Japanese cancer registries, the HRA Expert Group agreed that cancer data from Fukushima were likely to be comparable to those from other parts of Japan (see section 6.2.2).

5.2 Cancer risk characterization in the general population

5.2.1 Overview of results

The HRA Expert Group assessed cancer incidence risk as (i) LAR assessed for the entire life (up to 89 years attained age) and (ii) cumulated attributable risks assessed over 15 years after exposure (AR_{15}). Tables 11–14 summarize the cancer risk estimates for the general population composed of both males and females exposed at 1 year of age, 10 years and 20 years.

As mentioned in section 4.1.2, the geographical locations were classified into four groups. Based on the estimated doses it was concluded that the risks in Groups 3 and 4 locations would be much lower than the temporal and spatial fluctuations of the baseline cancer incidence risks. It was therefore decided to calculate the LAR only in Group 1 and Group 2 locations.

The results are presented for leukaemia, female breast cancer and all solid cancer incidence in Table 11 and Table 13. For practical reasons the results for thyroid cancer incidence are shown separately in Table 12 and Table 14 because a slightly different grouping of locations was used. The complete set of results tables has been included in Annex J.

In this section several risk quantities are presented. The LBR, described in section 3.3.1, represents the accumulated baseline probability to have a specific cancer up to age 89 years. Some data about LBRs for infants, children and young adults of both sexes are provided in Annex L. The LAR expresses the probability of premature incidence of a radiation-related cancer. The concept of LAR has an implicit "cumulative" nature derived from the way LAR values are calculated: as an integration of the risk that could be attributed to radiation exposure, arising on a year-per-year basis (excluding the latency period). In this context, LAR is an "extra" lifetime risk that is added to an already existing baseline lifetime risk (the LBR). The LFR, defined as the ratio between LAR and LBR, reflects the relative increase in cancer risk that could be attributed to radiation exposure. Both LBR and LAR are represented by a number between 0 and 1 while LFR is provided here as a percentage (%).

5.2.2 Results of lifetime risk calculations

Figure 9a shows the LAR values for leukaemia incidence in two locations of Group 1 (highest estimated doses) and in one representative location of Group 2 for females of different ages at exposure. Leukaemia has the particularity that both LAR and LBR are higher for males compared with females. The LAR is greatest in male infants (4 in 10 000) in the most affected Group 1 location (Group 1a). The LAR for infant girls is estimated to be about two thirds of that for infant boys (female: male LAR ratio around 0.7) (Figure 10). It can be seen that LAR is higher for 1-year-old infants and 10-year-old children compared with adults (LAR ratios 2.7 and 1.3, respectively). In general, the LAR in Group 2 locations is about a quarter of that for the most affected Group 1 location. The LFR is greatest (6.6%) in the most affected Group 1 location, while it is less than 1.7% in Group 2 locations for all ages and both sexes.

Figure 9b shows the LAR values for all solid cancers[2] incidence in two locations of Group 1 and in one representative location of Group 2 for females of different ages at exposure. The LAR in the Group 1a location with the highest estimated doses is greatest in female infants at around 110 in 10 000, and is lower at around 60 in 10 000 for 20-year-old female adults. The LAR for Group 2 locations is less than 32 in 10 000. Unlike leukaemia, the LAR is higher for females while the LBR is smaller (female:male LAR ratio 1.5) as shown in Figure 11. In general, risks are higher for 1-year-old infants and 10-year-old children than for 20-year-old adults (LAR ratios 1.9 and 1.5, respectively). The LFR is greatest for infant boys in Group 1a (3.8%) while it is below 1% in Group 2 locations.

Figure 9c shows the LAR values for thyroid cancer in two locations of Group 1 and in one representative location of Group 2 for females of different ages at exposure. The LAR for thyroid cancer incidence is greatest in female infants in the most affected Group 1

2. The assessment of the risk of all solid cancers combined is intended to provide, together with the assessment of the risk of leukaemia, an overall indication of the lifetime risk of cancer. In circumstances where the tissue doses are highly heterogeneous such as the dose to the thyroid following an intake of radioactive iodine, the risk of all solid cancers combined will not fully account for the risk of thyroid cancer (See section 6.3.4).

location at 52 in 10 000 and it is around 9 in 10,000 for 20-year-old females. In Group 2 locations LAR is around 2 in 10,000 for 20-year-old females Both LAR and LBR are much higher for females than for males (female: male LAR ratio 4.6) as shown in Figure 12. Risks are much higher for 1-year-old infants and 10-year-old children than for adults (LAR ratios of around 6 and 3, respectively). The LFR in the most affected location of Group 1 is 68% and 11% for 1-year-old and 20-year-old females, respectively. In Group 2 LFR is 23% and 3% for females in the same age-at-exposure ranges.

Figure 9d shows the LAR values for female breast cancer in two locations of Group 1 and in one representative location of Group 2 for different ages at exposure. The LAR is greatest in female infants in Group 1 locations at 36 in 10 000, which represents a 6.4% increase over the LBR (Figure 13). In general the LAR for Group 2 locations is estimated to be about one third of that in the Group 1a location. For young women (20-year-olds), the LAR is one third of that in infant girls.

Comparison of the assessed risks for a given subgroup (i.e. sex, age-at-exposure, location) using the same scale results in a clearer identification of the relative contribution of the different cancer sites to the overall risks. For example, the LAR for all solid cancers, breast, thyroid and leukaemia for 1-year-old females in Group 1 and Group 2 locations shows a major contribution from all solid cancers, and dominance of breast and thyroid cancer risks compared with leukaemia (Figure 14). All solid cancers represent a pooling of a variety of cancers, including breast and thyroid cancer. The risk model for all solid

Figure 9. Lifetime attributable risk (LAR) in females of 1 year, 10 years and 20 years in different locations of Group 1 and Group 2 for (a) leukaemia, (b) all solid cancers, (c) thyroid cancer, (d) breast cancer.

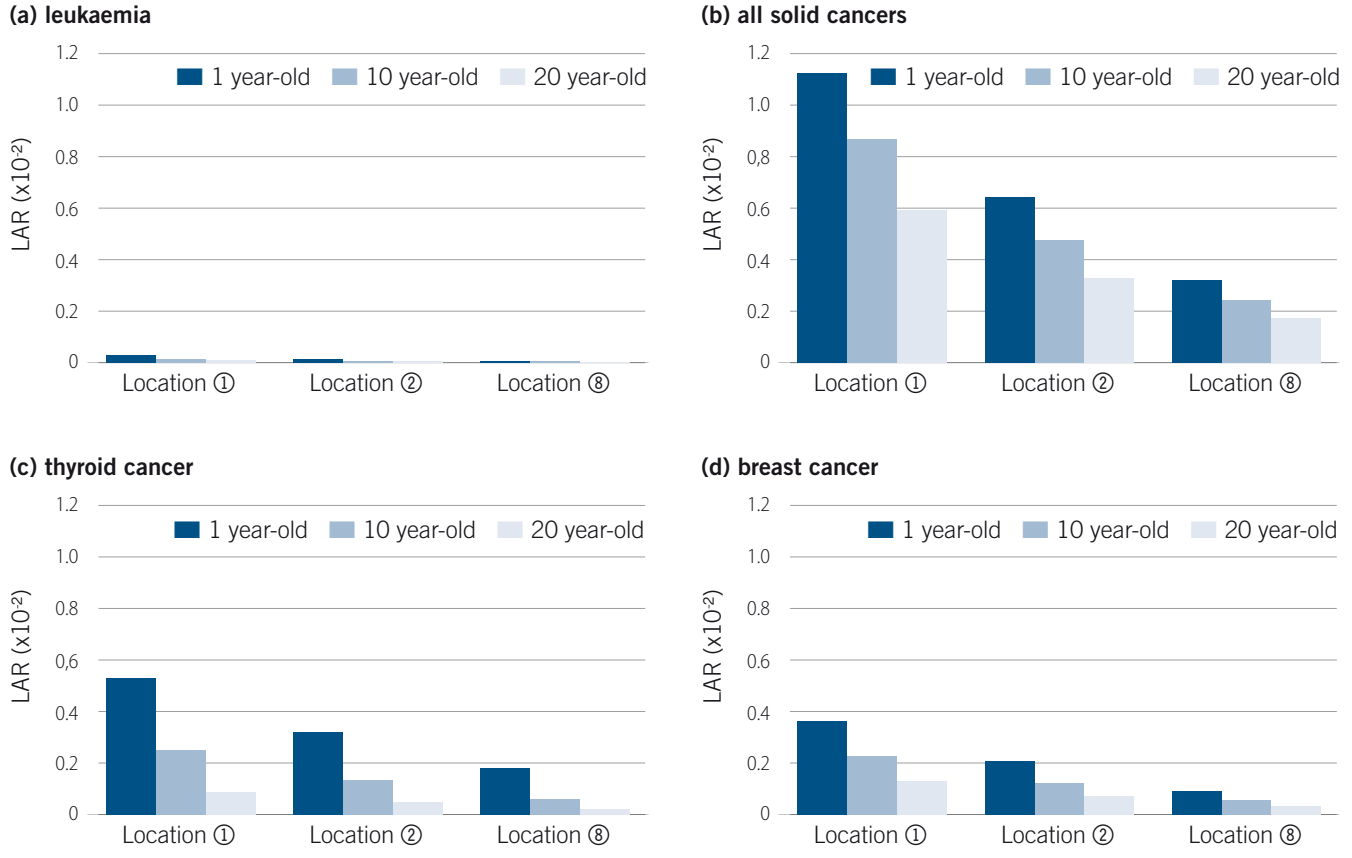


Table 11. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) up to attained age 89 for the general population (both sexes and three different ages at exposure) for all solid cancers, breast cancer and leukaemia incidence.

Location groups	Locations	Lifetime attributable risk (LAR x 10^{-2})					
		Males					
		Adults 20y		Children 10y		Infants 1y	
		All solid	Leukaemia	All solid	Leukaemia	All solid	Leukaemia
Group 1	①	0.394	0.015	0.568	0.020	0.730	0.040
	②	0.225	0.008	0.317	0.011	0.425	0.023
Group 2	③	0.093	0.003	0.124	0.004	0.160	0.008
	④	0.136	0.005	0.189	0.007	0.249	0.012
	⑤ to ⑨	0.115	0.004	0.159	0.006	0.208	0.010
	⑩ to ⑭**	0.115	0.004	0.159	0.006	0.208	0.010
Group 3	Rest of Fukushima prefecture (less affected)	*	*	*	*	*	*
	Neighbouring prefectures	*	*	*	*	*	*
	Rest of Japan	*	*	*	*	*	*
Group 4	Neighbouring countries	*	*	*	*	*	*
	Rest of the world	*	*	*	*	*	*
LBR (X 10^{-2}) for cancer incidence in Japan* **		**40.74**	**0.57**	**40.71**	**0.58**	**40.60**	**0.60**

* The HRA expert group agreed that mathematical calculations of health risks in terms of LAR would be not be performed for Group 3 and Group 4 locations, where the risks would be much lower than the normal temporal and spatial fluctuation of the baseline cancer incidence risks.

** For locations ⑩ to ⑭ no separate calculations were performed and LAR was assumed to be the same as locations ⑤ to ⑨.

*** Based on Japan 2004 cancer incidence rates from Matsuda et *al.* (*104*).

cancers, fits such a combination of diseases. In contrast, when breast and thyroid cancer risks are assessed by applying specific risk models for each of those cancer sites, their higher age-dependence becomes more evident. This explains why the LARs for breast and thyroid cancer do not necessarily sum exactly when compared with all solid cancer.

A different perspective is provided when considering the LFR, which expresses the relationship between the LAR and baseline (LBR). Figure 15 illustrates the LFR for the cancer sites mentioned above. While the LFR for all solid cancers is quite small, the LFR for thyroid cancer reaches a high value (around 70% for 1-year-old females). This dominant relative increase in thyroid cancer risk does not mean that the absolute risk is equally high. Even with a low number of "extra" cases of thyroid cancer (absolute risk), the very low baseline incidence of the disease results in a large relative increase as represented by the LFR. However, when the level of baseline incidence is that small, the actual number of "extra" cases is likely to be small also; therefore, the impact in terms of public health would be limited.

Lifetime attributable risk (LAR x 10^{-2})								
Females								
Adults 20y			Children 10y			Infants 1y		
All solid	Breast cancer	Leukemia	All solid	Breast cancer	Leukemia	All solid	Breast cancer	Leukemia
0.591	0.129	0.009	0.859	0.222	0.014	1.113	0.357	0.027
0.336	0.072	0.005	0.479	0.122	0.007	0.647	0.205	0.016
0.139	0.029	0.002	0.187	0.045	0.003	0.244	0.071	0.005
0.202	0.040	0.003	0.284	0.067	0.005	0.377	0.108	0.008
0.171	0.034	0.003	0.238	0.056	0.004	0.316	0.090	0.006
0.171	0.034	0.003	0.238	0.056	0.004	0.316	0.090	0.006
*	*	*	*	*	*	*	*	*
*	*	*	*	*	*	*	*	*
*	*	*	*	*	*	*	*	*
*	*	*	*	*	*	*	*	*
*	*	*	*	*	*	*	*	*
29.07	**5.55**	**0.40**	**29.09**	**5.54**	**0.41**	**29.04**	**5.53**	**0.43**

5.2.3 Temporal patterns of the risks

The results shown in the preceding graphs (i.e. LAR, LBR and LFR) provide a vision of the radiation risks integrated over the lifespan. In the context of this assessment, an infant who was 1 year old at the time of the Fukushima Daiichi NPP accident would typically reach the end of the lifespan at the turn of century (i.e. year 2100). Estimations over such a long duration carry a number of uncertainties associated with LAR and LBR that cannot be easily predicted or quantified (e.g. trends in cancer incidence rates, changes in demographic patterns, remedial actions, and increased early detection of diseases).

A way to lower the uncertainties is to use risk quantities over a shorter period of life. In the present assessment, the risks were also calculated over a 15-year period of life following the accident i.e. up to the year 2026. For the purposes of this report, such risk indicators are denoted as AR_{15}, BR_{15} and FR_{15}. In addition to reducing the associated uncertainties, these risk quantities appear more pertinent for priority setting when they

Table 12. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) up to attained age 89 for the general population (both sexes and three different ages at exposure) for thyroid cancer incidence

Location groups	Locations	Lifetime attributable risk (LAR x 10^{-2})			Lifetime attributable risk (LAR x 10^{-2})		
		Males			Females		
		Adults 20y	Children 10y	Infants 1y	Adults 20y	Children 10y	Infants 1y
Group 1	①	0.019	0.054	0.118	0.088	0.245	0.524
	②	0.010	0.029	0.071	0.048	0.133	0.317
Group 2	③	0.005	0.016	0.046	0.025	0.072	0.207
	④	0.005	0.015	0.044	0.025	0.070	0.194
	⑤ to ⑩	0.005	0.013	0.040	0.021	0.061	0.177
	⑪ to ⑭	0.003	0.011	0.035	0.016	0.049	0.154
Group 3	Rest of Fukushima prefecture (less affected)**	0.003	0.009	0.030	0.012	0.039	0.135
	Neighbouring prefectures	*	*	*	*	*	*
	Rest of Japan	*	*	*	*	*	*
Group 4	Neighbouring countries	*	*	*	*	*	*
	Rest of the world	*	*	*	*	*	*
LBR (X 10^{-2}) for cancer incidence in Japan* **		**0.21**	**0.21**	**0.21**	**0.76**	**0.77**	**0.77**

* Mathematical calculations of LAR were not performed for Group 3 and Group 4 locations, where the risks would be much lower than the normal temporal and spatial fluctuation of the baseline cancer incidence risks.

** Exceptionally the HRA Expert Group performed mathematical calculations of LAR for the rest Fukushima prefecture less affected area, even though it was included within Group 3. Thyroid doses in this area were calculated with very conservative assumptions. In practice, doses are considered to be much lower in this area and therefore, the thyroid cancer risks would be also lower than those presented in this table.

*** Based on Japan cancer incidence rates in 2004 from Matsuda et al (*104*).

Figure 10. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) for leukaemia in Group 1 Location ① for males and females exposed at 1, 10, 20 year-old.

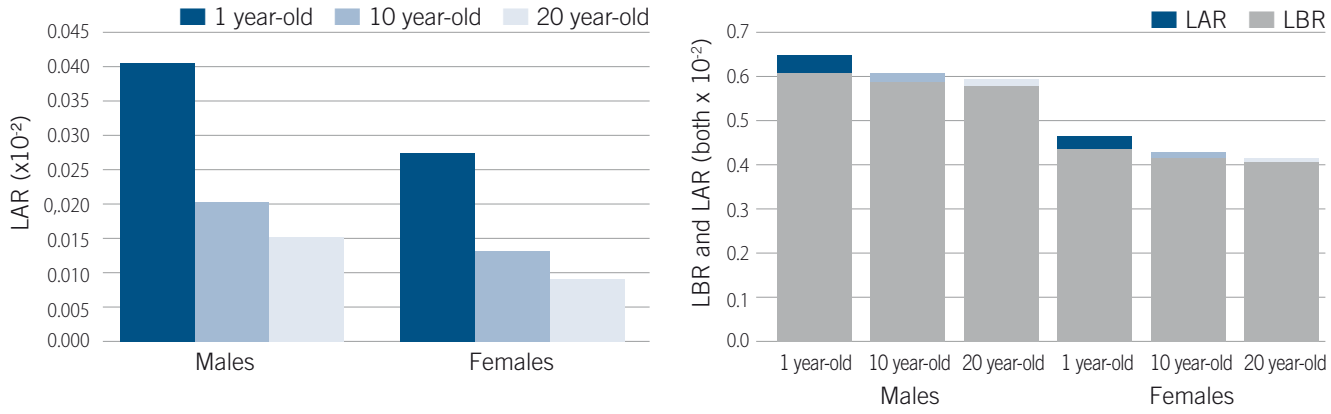


Figure 11. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) for all solid cancer in Group 1 Location ① for males and females exposed at 1, 10, 20 year-old.

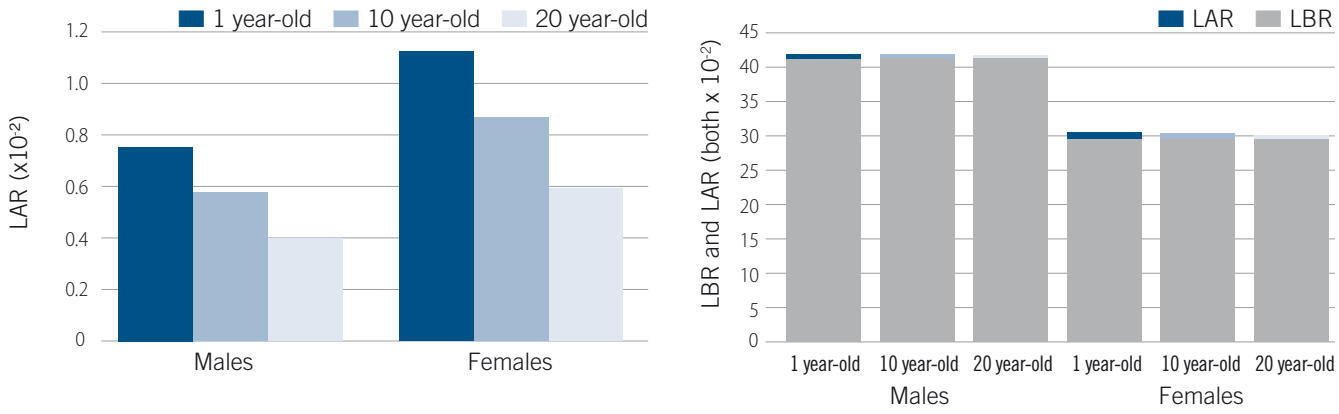


Figure 12. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) for thyroid cancer in Group 1 Location ① for males and females exposed at 1, 10, 20 year-old.

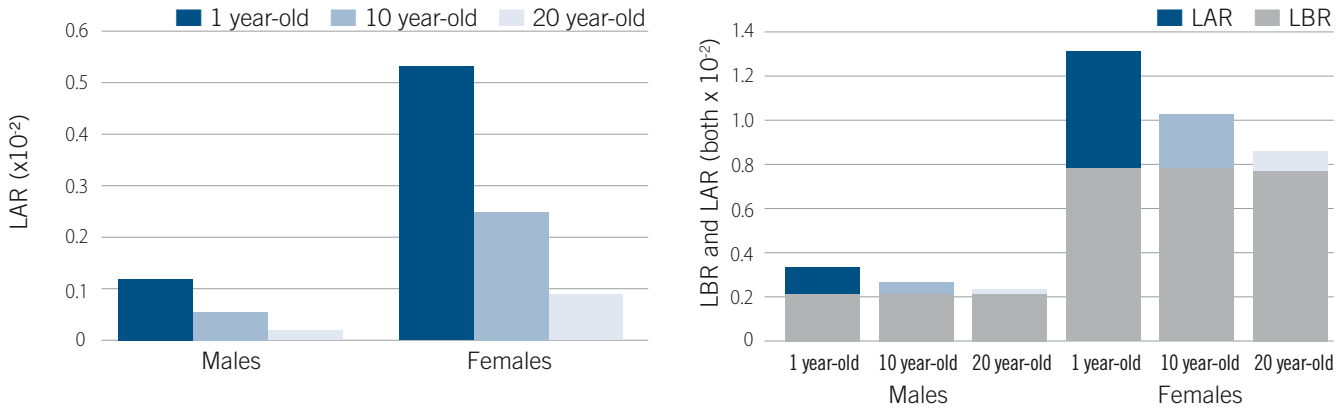


Figure 13. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) for breast cancer in Group 1 Location ① for females exposed at 1, 10, 20 year-old.

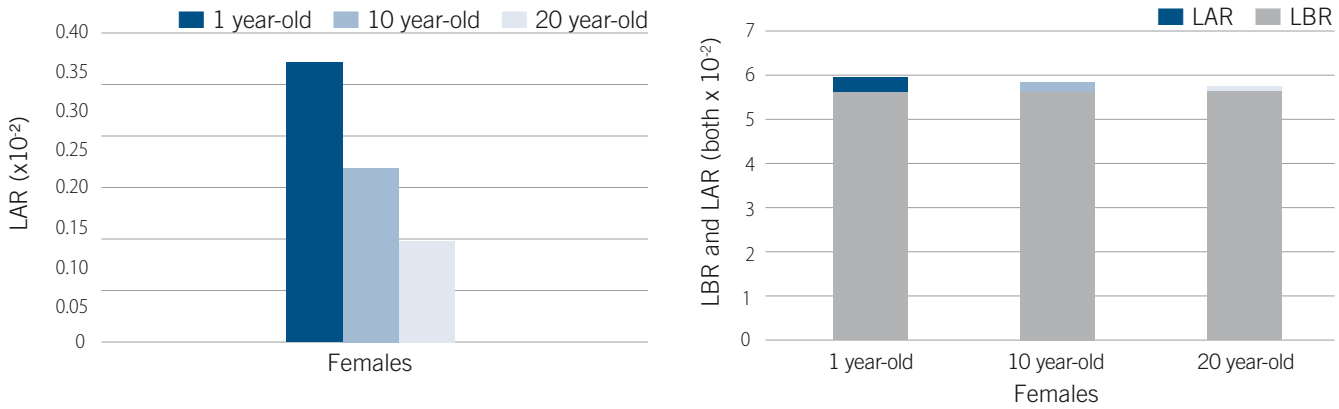


Figure 14. Lifetime Attributable Risk (LAR) for the studied cancer sites in a female, 1-year old at exposure in Group 1 and Group 2 locations

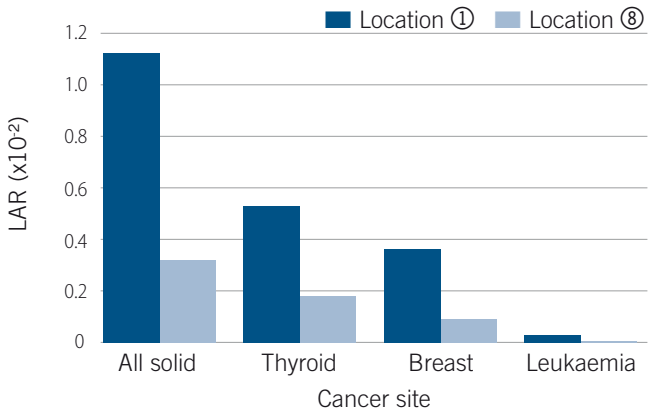


Figure 15. Lifetime Fractional Risk (LFR) for the studied cancer sites in a female, 1-year old at exposure in Group 1a and Group 2 locations

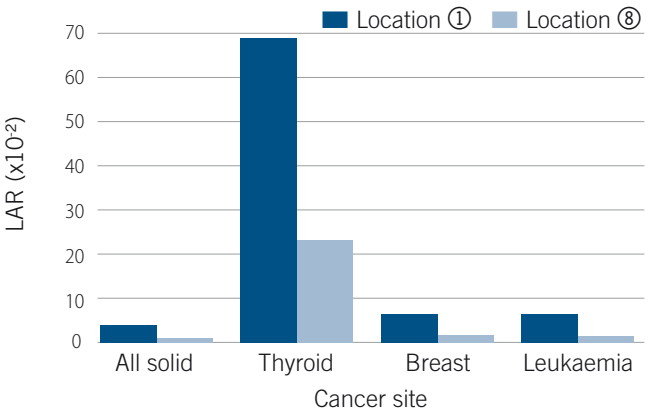


Figure 16. Cumulative attributable risk (AR_{15}) and lifetime attributable risk (LAR) for leukaemia as a function of attained age for a female, one year age-at-exposure, in Location ①.

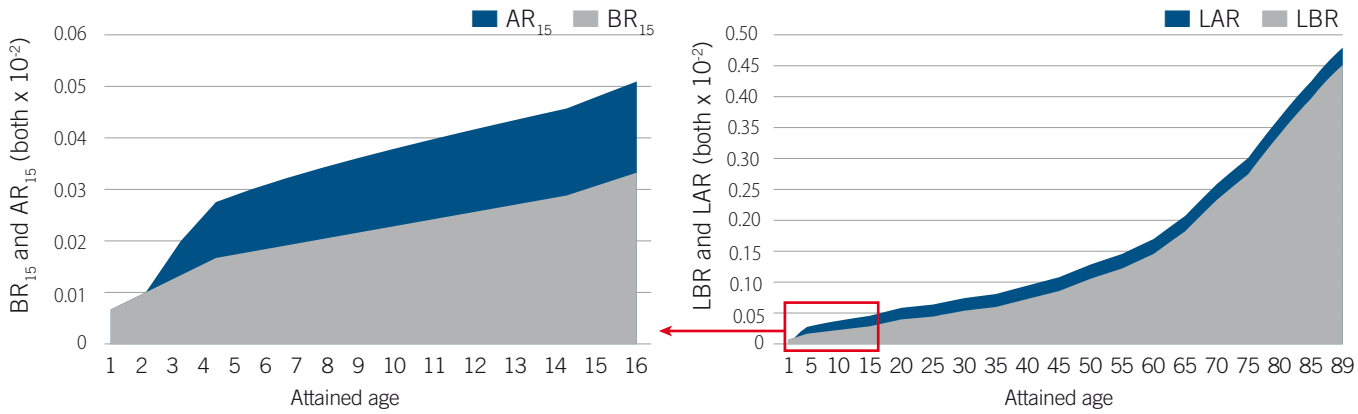


Figure 17. Leukaemia: cumulative attributable cancer risk over 15 years after exposure (AR_{15}) (a) for both genders and 3 age groups (infants, children and adults) in locations ① and ⑧; and (b) with cumulative baseline risk (BR_{15}) in location ①

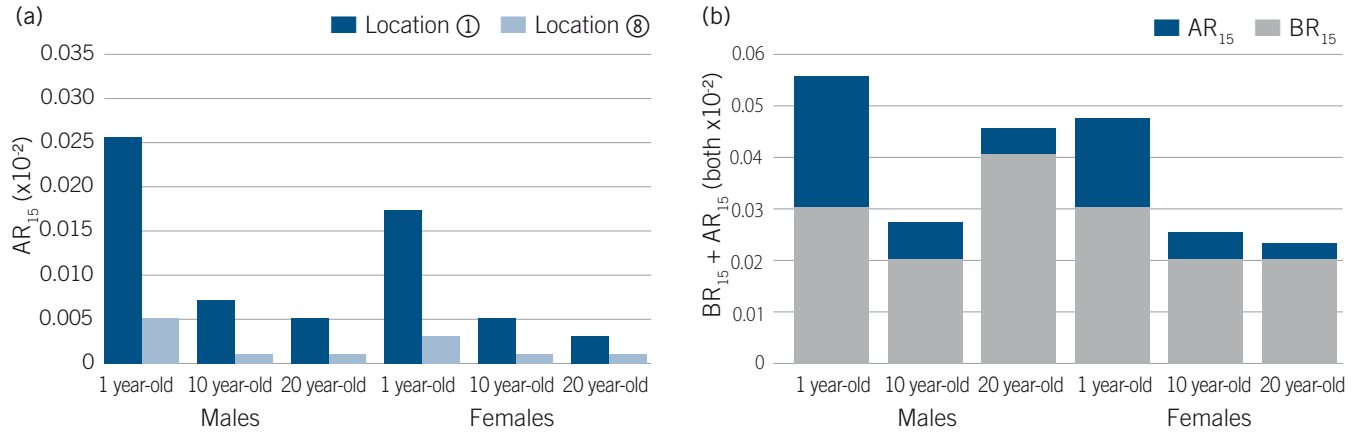


Figure 18. All solid cancer: cumulative attributable cancer risk over 15 years after exposure (AR_{15}) (a) for both genders and 3 age groups (infants, children and adults) in locations ① and ⑧; and (b) with cumulative baseline risk (BR_{15}) in location ①

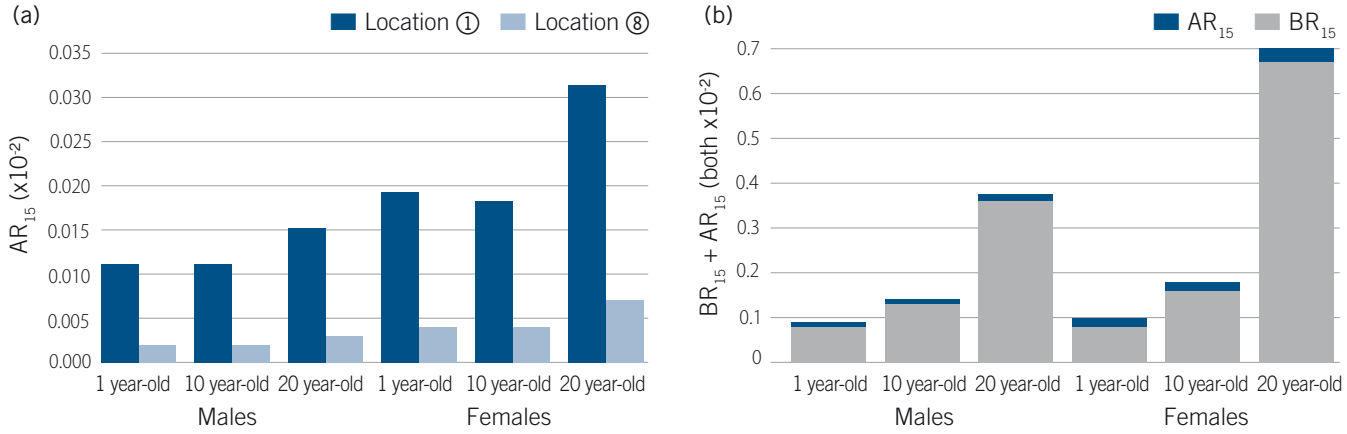


Figure 19. Thyroid cancer: cumulative attributable cancer risk over 15 years after exposure (AR_{15}) (a) for both genders and 3 age groups (infants, children and adults) in locations ① and ⑧; and (b) with cumulative baseline risk (BR_{15}) in location ①

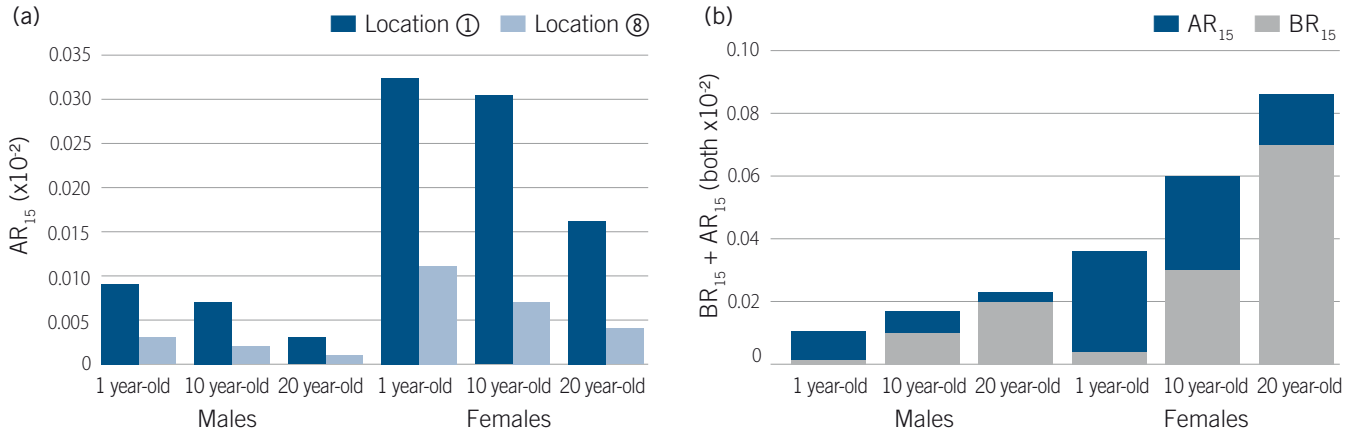
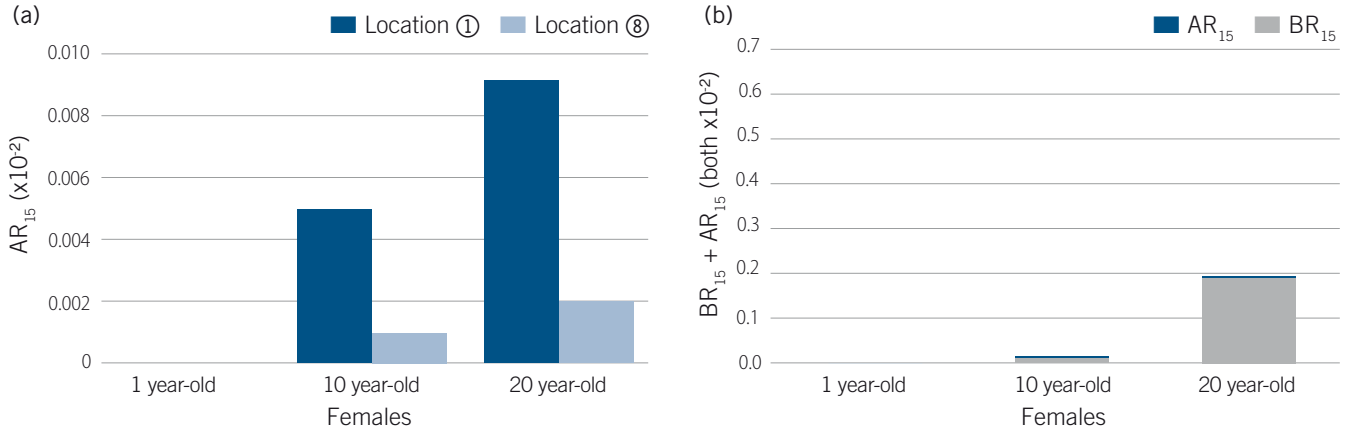


Figure 20. Breast cancer: cumulative attributable cancer risk over 15 years after exposure (AR_{15}) (a) and 3 age groups (infants, children and adults) in locations ① and ⑧; and (b) with cumulative baseline risk (BR_{15}) in location ①



Note that AR_{15} is not calculated for a 1 year-old (age-at-exposure) because existing evidence shows no breast cancer before an attained age of 20 years (section 2.2.1).

Table 13. Cumulative attributable risk over 15 years after exposure (AR_{15}) and cumulative baseline risk over the same segment of life (BR_{15}) for the general population (both sexes and three different ages at exposure) for all solid cancers, breast cancer and leukaemia incidence

Location groups	Locations	Cumulative attributable risk over 15 years (AR_{15} x 10^{-2})					
		Males					
		Adults 20y		Children 10y		Infants 1y	
		All solid	Leukaemia	All solid	Leukaemia	All solid	Leukaemia
Group 1	①	0.015	0.005	0.011	0.007	0.011	0.025
	②	0.008	0.003	0.006	0.004	0.006	0.014
Group 2	③	0.003	0.001	0.002	0.001	0.002	0.005
	④	0.004	0.001	0.003	0.002	0.003	0.006
	⑤ to ⑨	0.003	0.001	0.002	0.001	0.002	0.005
	⑩ to ⑭***	0.003	0.001	0.002	0.001	0.002	0.005
Group 3	Rest of Fukushima prefecture (less affected)	*	*	*	*	*	*
	Neighbouring prefectures	*	*	*	*	*	*
	Rest of Japan	*	*	*	*	*	*
Group 4	Neighbouring countries	*	*	*	*	*	*
	Rest of the world	*	*	*	*	*	*
15-y cumulative baseline cancer incidence risk (BR_{15} X 10^{-2}) ****		**0.36**	**0.04**	**0.13**	**0.02**	**0.08**	**0.03**

* The HRA Expert Group agreed not to calculate the AR15 for Group 3 and Group 4 locations, where the risks would be much lower than the normal temporal and spatial fluctuation of the baseline cancer incidence risks.

** The HRA Expert Group considered that the minimum attained age for breast cancer risk expression is 20 years. Note that the baseline female cancer rates in Japan used in the present assessment indicate no baseline incidence before age 20 (i.e. rate = zero).

are compared with the risk that could be observed in the next decades, mainly during the second half of the 21st century.

Figure 16 illustrates the impact of the use of risk quantities over a different period of life (i.e. 15 years vs. lifetime). It presents the LFR (LAR/LBR) vs. AR_{15}/BR_{15} for leukaemia, as a function of the attained age for one location of Group 1 for females exposed at 1 year of age. Note that the latency period is particularly visible in the inset of Figure 16.

Some data about LBRs for infants, children and young adults of both sexes are provided in Annex L. Note that these LBR data are based on cancer incidence data for Japan in 2004. They are representative of the lifetime baseline risks expected for the forthcoming decades but might substantially differ from them.

It is interesting to note in Figures 10–13 that LBR does not differ much at different ages for any of the cancer sites, while it does differ between sexes: it is generally lower in females than in males except for the LBR for thyroid cancer incidence, which is about 3.5 times higher in females. Figures 17–20 illustrate the contribution of AR_{15} and BR_{15} for

Cumulative attributable risk over 15 years (AR_{15} x 10^{-2})								
Females								
Adults 20y			Children 10y			Infants 1y		
All solid	Breast cancer	Leukemia	All solid	Breast cancer	Leukemia	All solid	Breast cancer	Leukemia
0.031	0.009	0.003	0.018	0.005	0.005	0.019	**	0.017
0.018	0.005	0.002	0.010	0.003	0.003	0.011		0.010
0.007	0.002	0.001	0.004	0.001	0.001	0.004	**	0.003
0.009	0.002	0.001	0.005	0.001	0.001	0.005		0.004
0.007	0.002	0.001	0.004	0.001	0.001	0.004		0.003
0.007	0.002	0.001	0.004	0.001	0.001	0.004		0.003
*	*	*	*	*	*	*	**	*
*	*	*	*	*	*	*		*
*	*	*	*	*	*	*		*
*	*	*	*	*	*	*		*
*	*	*	*	*	*	*		*
0.67	**0.19**	**0.02**	**0.16**	**0.01**	**0.02**	**0.08**	**0.00**	**0.03**

*** For locations ⑩ to ⑭ no separate calculations were performed and AR_{15} was assumed to be the same as locations ⑤ to ⑨.
**** Based on Japan 2004 cancer incidence rates from Matsuda et *al.* (*104*).

different ages and cancer sites and for both sexes. The BR_{15} differs among cancer sites; the values are much lower at younger ages for solid cancers, female breast and thyroid. The BR_{15} for leukaemia shows a different trend with age (baseline risks in 1-year-old infants are comparatively higher than in 10-year-old children). The BR_{15} does not show important sex-related differences for leukaemia. It is interesting to note that for all solid cancer and female breast cancer, the AR_{15} is higher for 20-year-old adults. These are mostly adulthood cancers sites, with longer latency. In contrast, thyroid cancer and leukaemia have a shorter latency and are considered more relevant in childhood.

5.3 Cancer risk characterization for the emergency workers

The HRA Expert Group assessed cancer incidence risks in workers as (i) LAR assessed for the entire life (up to 89 years attained age) and (ii) cumulative attributable risks assessed over 15 years after exposure. Risks of leukaemia, thyroid cancer and all solid cancers were assessed as a function of the first-year radiation dose to the relevant organs

Table 14. Cumulative attributable risk over 15 years after exposure (AR_{15}) and cumulative baseline risk over the same segment of life (BR_{15}) for the general population (both sexes and three different ages at exposure) for thyroid cancer incidence

Location groups	Locations	Cumulative attributable risk over 15 years (AR_{15} x 10^{-2})					
		Males			Females		
		Adults 20y	Children 10y	Infants 1y	Adults 20y	Children 10y	Infants 1y
Group 1	①	0.003	0.007	0.009	0.016	0.030	0.032
	②	0.002	0.004	0.005	0.009	0.016	0.020
Group 2	③	0.001	0.002	0.003	0.004	0.009	0.013
	④	0.001	0.002	0.003	0.004	0.008	0.012
	⑤ to ⑩	0.001	0.002	0.003	0.004	0.007	0.011
	⑪ to ⑭	0.001	0.001	0.003	0.003	0.006	0.009
Group 3	Rest of Fukushima prefecture (less affected)**	***	0.001	0.002	0.002	0.005	0.008
	Neighbouring prefectures	*	*	*	*	*	*
	Rest of Japan	*	*	*	*	*	*
Group 4	Neighbouring countries	*	*	*	*	*	*
	Rest of the world	*	*	*	*	*	*
15-y cumulative baseline thyroid cancer incidence risk (BR_{15} X 10^{-2})**		**0.02**	**0.01**	**0.0014**	**0.07**	**0.03**	**0.0040**

* Mathematical calculations of AR_{15} were not performed for Group 3 and Group 4 locations, where the risks would be much lower than the normal temporal and spatial fluctuation of the baseline cancer incidence risks.

** Exceptionally the HRA Expert Group performed mathematical calculations of AR_{15} for the rest Fukushima prefecture less affected area, even though it was included within Group 3. Thyroid doses in this area were calculated with very conservative assumptions. In practice, doses are considered to be much lower in this area and therefore, the thyroid cancer risks would be also lower than those presented in this table.

*** AR_{15}=0.0005

**** Based on Japan cancer incidence rates in 2004 from Matsuda et al (*104*).

using the same risk models derived from epidemiological studies of radiation-induced cancer as for the general population. Background information on recent cancer incidence and mortality data from Japan were used to derive the baseline cumulative risk. The cancer risk was assessed for workers exposed at 20 years, 40 years and 60 years of age. These ages are representative of the workers' population distribution, according to the data provided by TEPCO (see Table 7 and Annex H).

The LAR calculated for male workers under the four assumed exposure scenarios described in section 4.2) are presented in Tables 15 and 16. The complete set of results tables for workers has been included in Annex K. The results are analysed below, with particular emphasis on scenarios 1 and 2, which together represent more than 99% of the total workforce (i.e. 69% and 30% of the workers, respectively). Some particular

Table 15. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) up to attained age 89 for male workers (four scenarios of exposure and three different ages at exposure) for all solid cancers, thyroid cancer and leukaemia incidence

Scenario		Lifetime attributable risk (LAR X 10^{-2})								
		Age 20y			Age 40y			Age 60y		
		All solid	Thyroid	Leukae-mia	All solid	Thyroid	Leukae-mia	All solid	Thyroid	Leukae-mia
> 99% of the workers	1	0.086	0.001	0.003	0.050	<0.001*	0.002	0.023	<0.001*	0.002
	2	0.413	0.242	0.016	0,242	0.011	0.012	0.111	0.002	0.008
< 1% of the workers (upper bound)	3	3.437	0.060	0.157	2.018	0.016	0.119	0.922	0.003	0.080
	4	1.774	3.558	0.075	1.042	0.918	0.057	0.476	0.191	0.038
LBR (x 10^{-2}) for cancer incidence in Japan		**40.74**	**0.21**	**0.57**	**40.90**	**0.19**	**0.52**	**38.10**	**0.14**	**0.44**

* The calculated LAR values are between 0.0001 and 0.0004

considerations are made about scenarios 3 and 4, which represent less than 1% of the workers.

The LAR for leukaemia is on the order of 0.3 in 10 000 in scenario 1 and on the order of 1 to 2 in 10 000 in Scenario 2. No remarkable age-related differences in the magnitude of LAR were observed. This represents a LFR of around 0.5% and 2.8%in scenarios 1 and 2, respectively. The highest LAR levels are observed in Scenario 3 (LAR around 16 in 10 000, representing a relative increase (LFR) of 27% in the baseline lifetime risk of leukaemia).

Thyroid cancer risks are low in Scenario 1, where LAR is on the order of 0.1 in 10 000 for workers exposed at 20 years of age and one order of magnitude below for workers exposed at 40 and 60 years of age. The LAR for Scenario 2 is on the order of 4 in 10 000, while for scenario 3 is on the order of 6 in 10 000. In general, risks show a strong dependence on age-at-exposure, with much lower risks for workers exposed at 60 years of age compared with 20-year-old workers. For Scenario 1 the LFR was estimated to be below 0.5% for all ages, while under Scenario 2 the LFR was estimated to be markedly age-dependent: around 20%, 5.8% and 1.4% for workers exposed at 20, 40 and 60 years old, respectively). Thyroid cancer is the dominant risk in Scenario 4, which assumes the highest thyroid organ dose and effective dose, resulting primarily from internal exposure to radioactive iodine. The risk is much higher for workers exposed at 20 years old compared with workers exposed at 40 years of age and 60 years of age (LAR values about 356 in 10 000, 92 in 10 000 and 19 in 10 000, respectively. This strong dependence with age-at-exposure is further discussed in section 6.1.1.

The LAR values for all solid cancers in Scenario 1 vary from 2 to 9 in 10 000 (60 years of age and 20 years of age at exposure, respectively), which represents a LFR of around 0.1%. The LAR is on the order of 40 in 10 000 in workers 20 years of age exposed under

Table 16. Cumulative attributable risk over 15 years after exposure (AR_{15}) and cumulative baseline risk over the same segment of life (BR_{15}) for male workers (four scenarios of exposure and three different ages at exposure) for all solid cancer, thyroid cancer and leukaemia incidence

Scenario		Cumulative attributable risk over 15 years after exposure (AR_{15} x 10^{-2}) *								
		Age 20y			Age 40y			Age 60y		
		All solid	Thyroid	Leukae-mia	All solid	Thyroid	Leukae-mia	All solid	Thyroid	Leukae-mia
> 99% of the workers	1	0.003	<0.001*	0.001	0.008	<0.001*	0.001	0.013	<0.001*	0.001
	2	0.016	0.008	0.006	0.038	0.004	0.005	0.061	0.001	0.006
< 1% of the workers (upper bound)	3	0.133	0.011	0.055	0.319	0.005	0.052	0.505	0.002	0.056
	4	0.069	0.650	0.026	0.165	0.309	0.025	0.261	0.124	0.026
15-y cumulative baseline cancer incidence risk (BR_{15} x 10^{-2})*		**0.36**	**0.02**	**0.04**	**3.71**	**0.05**	**0.08**	**21.03**	**0.09**	**0.23**

* The calculated AR_{15} values are between 0.0001 and 0.0003.

Scenario 2 and somewhat lower at older ages (11–24 in 10 000). This represents a LFR of less than 1% (all ages).

Some data about LBR for male adults of 20, 40 and 60 years of age are provided in Annex L. They are presented in two ways: (i) as a cumulative baseline cancer incidence over 15 years (BR_{15}) and (ii) as the lifetime baseline cancer incidence up to 89 years of attained age (LBR). It is interesting to note that:

- LBR does not differ much between young and middle-aged adults, and is slightly lower at 60 years of age (the remaining lifespan for a person exposed at 60 years of age is lower than for a person exposed at 20 years of age, and therefore the prospective lifetime risk of developing a cancer will be lower).
- If the lifetime baseline risk is "truncated" at 15 years after exposure (BR15) the age-dependent differences become more evident and younger adults show lower cumulative baselines. This is more evident for leukaemia and all solid cancer, but less for thyroid cancer.
- It is noted that these LBR data are based on cancer incidence data for Japan in 2004. They are therefore representative of the lifetime baseline risks expected for the forthcoming decades. However, it is clear that cancer incidence trends may change over time, and that future true baseline risks will differ from those used in this assessment.

5.4 Non-cancer risk characterization

5.4.1 General population

No acute effects of radiation exposure such as acute radiation syndrome or skin injuries have been observed among the general population. Such acute effects are observed after

exposure to high doses and are not therefore expected among the general public as a result of the Fukushima Daiichi NPP accident. Based on the preliminary dose estimation in the general population both inside and outside Japan, no increase in the frequency of tissue reactions attributable to radiation exposure is expected in the general population; no respective clinical reports were received in Japan or elsewhere. This is supported by current knowledge on radiation biology and existing evidence about threshold doses for deterministic effects (see Annex F).

As a result of this assessment, it was concluded that no increase in the frequency of cataracts, circulatory diseases or any other tissue reaction is expected for the general population, given the range of doses under consideration.

The HRA Expert Group did not perform a quantitative assessment of radiation risks of non-cancer thyroid nodules through mathematical risk models. Instead, a qualitative assessment was supported by the evidence summarized in sections 2.2.2 and Annex F, indicating that even low doses of radiation may increase the risk of non-cancer thyroid nodules in individuals exposed.

The HRA Expert Group gave particular consideration to the assessment of non-cancer risks of radiation exposure of the unborn child. For that purpose it took into consideration the preliminary dose estimation conducted by the Dose Expert Panel and the results of the calculation of first-year organ doses conducted for the present HRA. The HRA Expert Group concluded that, even under the conservative assumptions adopted, the radiation doses in the general population are below the thresholds for the deterministic effects after prenatal radiation exposure, described in chapters 2 and 3. Therefore, no increase is expected in the incidence of congenital or developmental abnormalities, including cognitive impairment attributable to *in utero* radiation exposures during the Fukushima Daiichi NPP accident.

As described in section 2.2, the risk of radiation-induced hereditary effects has not been definitively demonstrated in human populations. Based on animal data, international scientific bodies consider that any risk effect of hereditary effects for the offspring of those who were exposed at reproductive age would be much lower than the additional lifetime risk of cancer for the exposed individual him- or herself (about one order of magnitude lower).

5.4.2 Emergency workers

The HRA Expert Group reviewed the level of doses reported among emergency workers, taking into account current knowledge and new scientific evidence about non-cancer effects provided in sections 2.2.2 and Annex F. Taking into account that 99% of workers were exposed to low doses (< 100 mSv), non-cancer risks are less relevant than cancer risks in terms of health impact. However, the HRA Expert Group considered all the possible health outcomes relevant to the four exposure scenarios assumed in the present assessment.

To date, no radiation injuries have been observed among Fukushima Daiichi NPP emergency workers as a result of the accident (i.e. no cases of acute radiation syndrome or skin injuries). None of the seven reported deaths among emergency workers is attributable to radiation exposure. In the early phase of the emergency three workers were exter-

nally contaminated. Local decontamination procedures took place, and no deterministic effects were reported in those workers.

Thyroid organ doses exceeding 10 Sv were estimated in two workers. This is a dose level that may result in deterministic effects such as thyroid dysfunction (i.e. hypothyroidism).

Approximately 2 000 workers were given stable iodine (potassium iodide [KI]) during the emergency response phase. Although most of these workers took fewer than 10 tablets, some took up to 87 tablets (self-administration). No allergic reactions were seen in this group. Three workers presented increased levels of thyroid-stimulating hormone (TSH) and decreased levels of thyroxin (T4), one of the two hormones produced by the thyroid. This thyroid dysfunction was transient and parameters returned to normal values once the KI administrations were stopped (see Box 7). The considerations given to the radiation-related risks of thyroid nodules in the general population (see section 5.4.1) are also applicable to workers.

Scenario 4 refers to an effective dose exceeding 500 mSv, but it is assumed to be as primarily due to internal exposure to ^{131}I, implying a high dose to the thyroid (>10 Sv) rather than to any other organ. Scenario 3 refers to an effective dose of 200 mSv and, in contrast to Scenario 4, no internal exposure due to ^{131}I is assumed here. If caesium intake was involved, the value of 200 mSv would be representative of organ doses, as caesium is distributed homogeneously in the body.

New evidence about the dose-response relationship for radiation-induced cataract indicates that the threshold doses can be around 500 mSv. In light of the most recent scientific evidence presented in Chapter 2, the HRA Expert Group concluded that there should be no expectation of cataract. It appears unlikely that the relatively small number

Box 7. Possible adverse effects of iodine thyroid blocking

During nuclear emergencies, public health protective actions may be implemented to prevent radiation exposure and associated health risks. The administration of stable iodine can prevent the uptake of radioactive iodine by the thyroid gland. When potassium iodide is taken before or shortly after exposure it can saturate the thyroid gland, thus reducing the dose and risk of thyroid cancer. Potassium iodide should be taken only when instructed by competent authorities, following dosage recommendations, especially for children.

Side effects may result from the administration of stable iodine for thyroid blocking, especially in iodine-deficient regions and in specific age groups or sub-populations. These side effects are not related to radiation and are therefore beyond the scope of this HRA. However, they are noted here as background information to support the interpretation of possible signs and symptoms that might otherwise be wrongly attributed to effects of radiation.

Reported side effects of iodine thyroid blocking include extra-thyroidal effects (e.g. digestive and skin reactions) and thyroid dysfunctions in connection with pre-existing thyroid disorders, such as auto-immune thyroiditis, Graves disease and nodular goiter. Hypothyroidism has been observed when stable iodine has been taken for longer than 1 week or for 10 days continuously. A large-scale survey in Poland after the Chernobyl accident (*131*) provided solid evidence based on over 10 million doses of stable iodine (potassium iodide [KI]) to children and around 7 million doses to adults. This topic was recently reviewed (*132*) and available studies did not reveal severe adverse reactions to KI in the general public. Persons with known iodine sensitivity as well as newborns and elderly people might be at higher risk.

of workers with doses exceeding 100 mSv[3] will show a statistically significant increase in the incidence of vision-impairing lens opacities at lower doses, although dosimetry data for the lens of the eye are not available.

As discussed in Annex F, section F.3, a threshold of 500 mSv would be compatible with epidemiological data on circulatory disease after radiation exposure, although work is in progress to determine whether such effects may be probabilistic rather than deterministic. It is concluded that there may be an increased risk of long-term circulatory disease, particularly among the workers whose doses exceeded 500 mSv[4]. It must be noted that the risks of circulatory disease among the LSS atomic bomb survivors were about three times lower than the risk for cancer (*56*) so if the cancer risk is small the circulatory disease risk is expected to be even smaller.

The considerations made above for heritable risks in the general population (section 5.4.1) are also applicable to workers.

3. This refers to effective doses or to the organ doses calculated by the HRA Expert Group. Doses to the lens of the eye were not available within the timeframe of the HRA Expert Group work.

4. Note that these are effective doses and are mainly due to thyroid doses.

6. Discussion

6.1 Factors influencing the radiation-related health risks

6.1.1 Age at exposure

Age at the time of irradiation is one of the most important biological variables influencing both short- and long-term effects of ionizing radiation. The influence of age-at-exposure on radiation-induced excess absolute risks (EAR) and excess relative risk (ERR) varies by cancer site (*105,5*). In the context of this HRA, the age-at-exposure becomes particularly relevant to the consideration of possible long-term health effects. Radiation exposure at a young age generally tends to result in higher risks than those resulting from exposure at older ages. Cancer risks in the unborn child are considered to be similar to those in 1-year-old infants (*12*) (see section 6.3.3).

The age pattern in radiation-related leukaemia incidence excess risk is a function of both age-at-exposure and time since exposure (see below). A very strong dependence on age-at-exposure has been found in the excess risk of leukaemia mortality, with exposure at 10 years of age associated with approximately 20-fold ERR/Gy in the period 5–20 years after exposure (*106*). The results of this HRA are consistent with this trend. Indeed, risks are higher in children and infants (LAR ratios 10 years: 20 years = 1.4, and 1 year: 20 years = 2.8).

Of the various cancer types, perhaps the most illustrative example is thyroid cancer, with a robust body of evidence of higher risks at younger ages and with weak evidence for an excess (or substantially lower risks) following radiation exposure in adulthood. The risk of thyroid cancer due to radioiodine exposure from the Chernobyl accident has shown strong age dependence. An ecological study based on a very large number of measurements of individual thyroid doses showed an ERR of 9.1/Gy for 0–4 years at exposure versus 3.4/Gy at 10–18 years (*107*). The HRA Expert Group took into consideration all the existing evidence to support their choice of the risk models to be applied to assess thyroid cancer risk in this HRA. The results reflected the presumptions that higher risks would be estimated in the youngest groups. Indeed, the LAR is much higher for 1-year-old infants and 10-year-old children compared with young adults. It is important to note that, on an absolute scale, the calculated excess risks (LAR) are small. However, in relative terms, the comparison with the low baseline thyroid cancer incidence rates in children translates into a large relative increase (LFR).

As mentioned above, epidemiological studies have indicated that the risk of thyroid cancer decreases with increasing age-at-exposure, with little or negligible risk apparent after the age of 20 years (*21,23*). This age dependence of thyroid cancer risks was also observed in the results of the HRA for workers. Indeed, much lower risks were found for 40-year-old and 60-year-old workers compared with 20-year-old workers.

Figure 21. Influence of the age-at-exposure on the lifetime attributable risk (LAR) of solid cancer

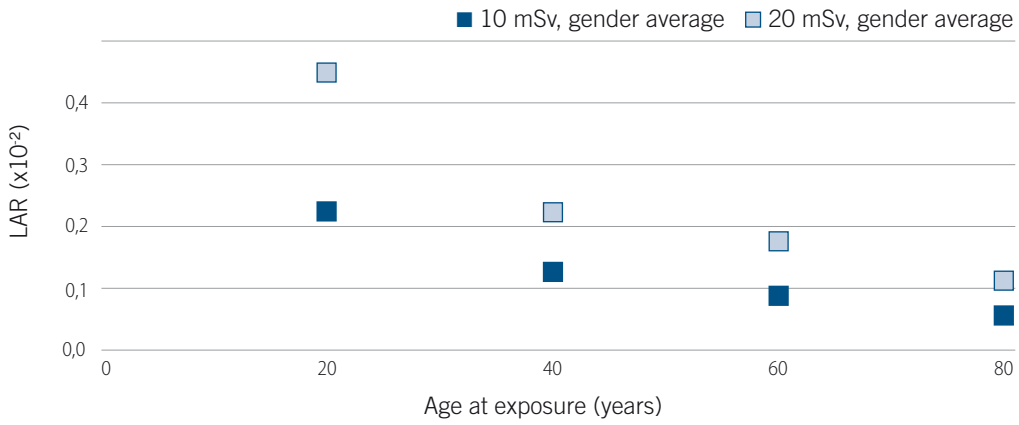
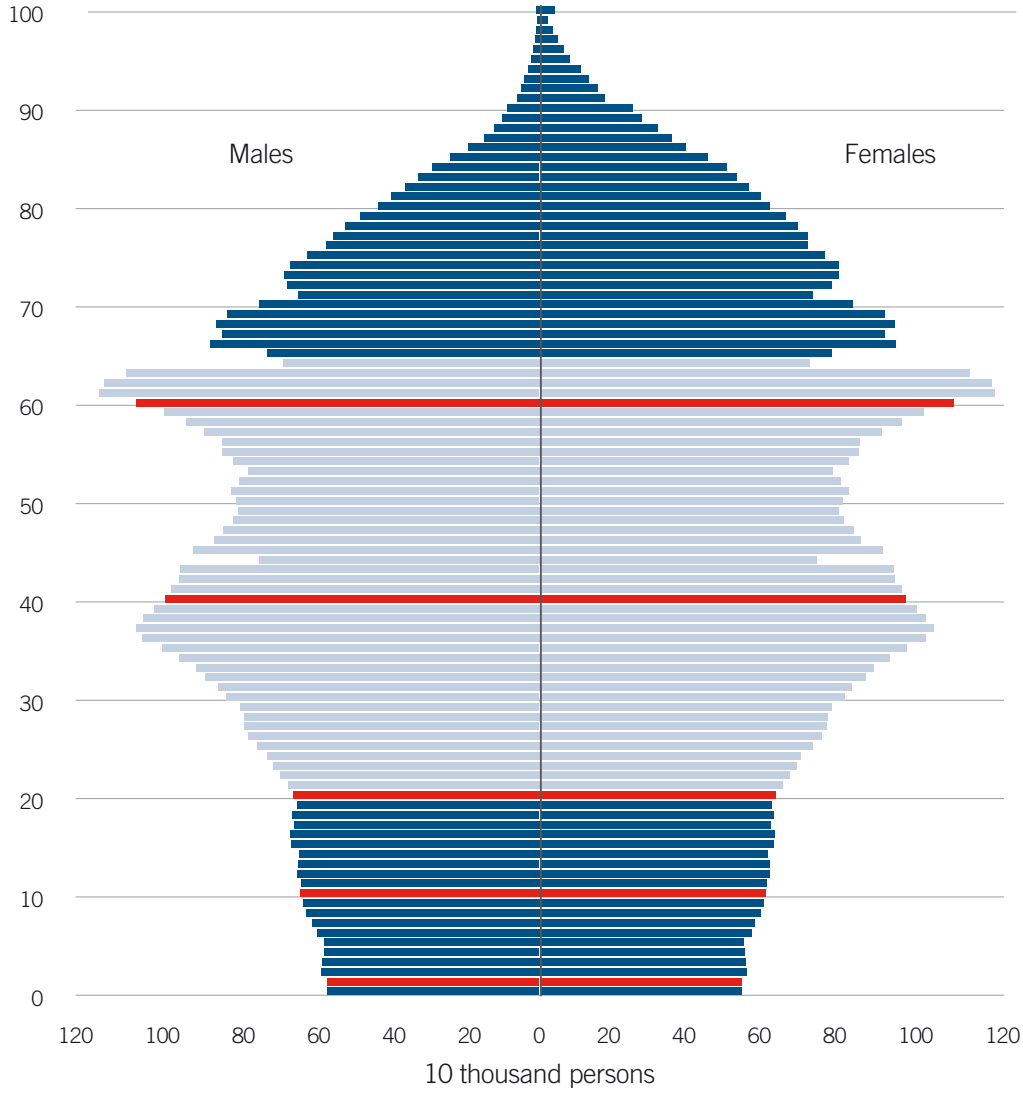


Figure 22. Japan population pyramid for 2010



Source: Ministry of Internal Affairs and Communication, Statistics Bureau, Director-General for Policy Planning (Statistical Standards) and Statistical Research and Training Institute (http://www.stat.go.jp/english/data/nenkan/1431-02.htm)
The red lines indicate the ages-at-exposure that were selected to be representative of the general population (infant, child, adult) and of the emergency workers (20, 40 and 60 years old).

For female breast cancer, a large impact of age-at-exposure on the excess absolute risk (EAR) was shown in epidemiological studies. This was one of the presumptions of the HRA Expert Group, which supported the decision to consider this cancer site separately from the other solid cancers in this assessment. The results of the HRA reflected this trend, though the risks were small in all age groups.

Less variability in risk by age-at-exposure has been shown for all solid cancers among atomic bomb survivors. However, the general trend of higher risks at younger ages was also found for solid cancers in this HRA. To illustrate this, Figure 21 shows an example of the influence of age-at-exposure on the LAR for solid cancers following radiation exposure to 10 and 20 mSv (sex-averaged values). It should be noted that the oldest population group considered for the HRA in the general population is 20-year-old adults. This is a conservative approach because, as seen in Figure 21, this is the most sensitive age group of adults in terms of cancer risk. This is particularly relevant for Japan's population, which has long been among the oldest in the world (Figure 22).

As most of the LSS survivors aged 40 years or older at the time of bombings have already died, information on cancer risk in this population is essentially complete today. In contrast, many survivors who were exposed in childhood are still alive and going through the period of life when the baseline risk of developing cancer rises. Thus, further evidence about the influence of age-at-exposure on the risk of cancer in adulthood can be expected in the near future.

6.1.2 Time since exposure and attained age

In general, baseline cancer incidence and mortality rates increase with age. As the baseline risk is low in childhood: even a modest absolute excess translates into a large relative risk in children (expressed as a multiple of the baseline), as reflected in the LAR and LFR results presented in this report. In the LSS a model that includes both age-at-exposure and attained age (age at observation) provides the best fit for all solid cancers. For an alternative representation of age-time patterns, "time since exposure" can be derived from attained age minus age-at-exposure. The cancer risk for someone exposed at age 20 years and observed at an attained age of 35 years (i.e. 15 years later) would be different from the risk among persons exposed at the same age but observed later– e.g. attained age 89 years (i.e. 69 years after exposure). However, the effect of attained age varies between risk models, being generally negative (decreasing relative risk with older age) in the relative risk models, but positive (higher absolute risk at older ages at observation) in the absolute risk models.

The tables in Annex L show cumulative baseline incidence up to 15 years (BR_{15}) and lifetime baseline incidence up to 89 years of age (LBR). The small absolute increase in risk that translates into a large relative increase in the15 years after exposure may be less evident in a lifetime follow-up (e.g. up to 89 years of attained age). Taking this into account, the HRA Expert Group assessed the cancer risks not only over the whole lifetime (i.e. up to 89 years of attained age) but also over a segment of life of 15 years after exposure.

6.1.3 Sex

Females tend to be at a greater risk of cancer from a given unit dose of radiation than males. The sex difference is largely independent of age-at-exposure. In the LSS study

of atomic bomb survivors, both the ERR and the EAR estimates for solid cancers are about 50% higher for women than for men (the female-male sex excess risk ratio is 1.6 for ERR and 1.4 for EAR). This is one of the key features that can be generalized about the association between radiation exposure and solid cancers observed in the atomic bomb survivors (16). No strong difference in leukaemia risk has been found between sexes (5,106). Nevertheless, no systematic differences between men and women have been consistently observed in studies with low exposures, such as in environmental and occupational settings.

The thyroid cancer risk following the Chernobyl accident has not shown a consistent sex difference as reflected in various findings reported in the literature (*107,108*). In the studies of fallout from the Nevada test site, no obvious sex differences were reported (*109*). In studies of the fallout in the Marshall Islands (*42*), and also in Mayak workers (*110*), there was some evidence for higher risk among women.

In this HRA, the female-to-male ratio was 1.46 for solid cancer LAR, and 4.5 for thyroid cancer LAR. In contrast, leukaemia LAR values are higher for males compared to females, with a female-to-male sex ratio of around 0.67.

6.2 Main sources of uncertainty

6.2.1 Exposure estimates for the general population

First-year dose estimation

The dose estimates used for the risk calculations are conservative. Owing to the preliminary nature of the data available as of September 2011, some assumptions about the implementation of protective measures and food consumption were deliberately based on options that are more likely to overestimate than to underestimate the radiation exposure. For example, assumptions were made that people in the most affected areas outside the 20-km radius continued to live there for 4 months after the accident, whereas a proportion of the population was relocated earlier, the assumption that all monitoring data on food were obtained from food available on the market, and the assumption that all people in Fukushima prefecture consumed only food produced in Fukushima prefecture. Therefore, some possible dose overestimation may have occurred.

This report is based on dose estimates calculated by dosimetric modelling using environmental and food monitoring data, rather than actual human measurements. The experience from the Chernobyl accident indicates that, when human monitoring data (e.g. whole-body counting) were used to determine more precise estimates of human exposure, actual doses were much lower than the hypothetical doses calculated through modelling (93). Data concerning internal and external exposure following the Fukushima Daiichi NPP accident were published during the last year. In some cases the doses reported were substantially lower than those reported in the WHO preliminary dose estimation (*111,112,113,114,115,116,117*). The Fukushima prefectural government and the Fukushima Medical University are now carrying out the estimation of external doses of all residents in Fukushima prefecture, in collaboration with the National Institute of Radiological Sciences (NIRS) (*116*). At the time of the publication of this report, interim data from the Fukushima Health Management Survey indicate external exposure for 99%

of residents at less than 10 mSv, with a highest estimate of 25.1 mSv. Internal exposure from $^{134}Cs/^{137}Cs$ is below 1 mSv in 99.9% of persons surveyed, based on whole-body counting (WBC) performed between June 2011 and July 2012 (*117,118*). It should be noted that short-lived radionuclides such as ^{131}I were no longer detectable when the reported WBC was performed. The methodologies and exposure pathways considered differ from those in the WHO Preliminary dose estimation (*3*). These, together with the main sources of uncertainty in the dose assessment for the first year, are discussed in more detail in section 4.6.

Organ doses

The Dose Expert Panel provided effective doses and equivalent thyroid doses resulting from the exposures during the first year after the Fukushima Daiichi NPP accident. The HRA Expert Group considered it more appropriate to refer to individual organ doses in red bone marrow, thyroid, breast and colon as input data for the cancer risk models for leukaemia, thyroid cancer, breast cancer and all solid cancers, respectively. As explained in Chapter 4, the ratios between the absorbed dose and the effective dose in each of the above organs were applied to the effective dose to obtain organ doses. There is a relationship between organ doses and effective dose, as the effective dose is a weighted sum of organ doses, but this relationship depends on the radionuclide involved. The calculation of the appropriate ratios has therefore an intrinsic uncertainty due to the radionuclide composition of the releases and consequent deposits.

Lifetime doses

For the purpose of calculating the lifetime risk, the HRA Expert Group assumed that the ratio of long-term dose to 1-year dose would be equal to 2[1] on the basis of the Chernobyl experience, and taking into consideration the differences between the Chernobyl and Fukushima Daiichi NPP accidents. The distribution of the lifetime dose on a yearly basis was provided to the risk modellers for the health risk calculations. Implicit in this assumption on the temporal distribution of the lifetime dose are a number of uncertainties associated with the influence of the natural mechanisms mentioned in section 4.1.4, as well as a number of interventions that can reduce radiation exposure – including more stringent standards and remedial actions (e.g. clean-up of buildings, remediation of soils and vegetation, treatment of agricultural fields, waste management). The HRA Expert Group acknowledged these uncertainties and considered it important to perform additional LAR calculations, taking into account first-year exposures. These additional calculations included LAR both up to 15 years after exposure and up to 89 years of age. By comparing the results, the HRA expert group concluded that LAR values do not differ substantially (see one example in Table 17). This is predictable for the locations in Group 1 where it was assumed that relocation of the population took place at 4 months. The factor 2 applied to the first-year dose to get the lifetime dose does not translate into a doubling of the risk. This is because lifetime doses are delivered over many decades, and cancer risks decrease with attained age.

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1. With an exception for the locations where people were relocated (i.e. Namie town in Futaba county, Iitate village in Soma county and Katsurao village in Futaba county). In those locations it was assumed that relocation took place at 4 months after the accident. Therefore the dose over the lifetime was calculated as the sum of the doses received during the first 4 months after the accident plus the lifetime dose calculated for the locations within Fukushima prefecture zone 1 (western least contaminated).

Figure 23. Lifetime attributable risk (LAR) for leukaemia in one-year old males (first-year dose 10 mGy) using different transfer weights (100% ERR, 100% EAR and 50% ERR-50% EAR)

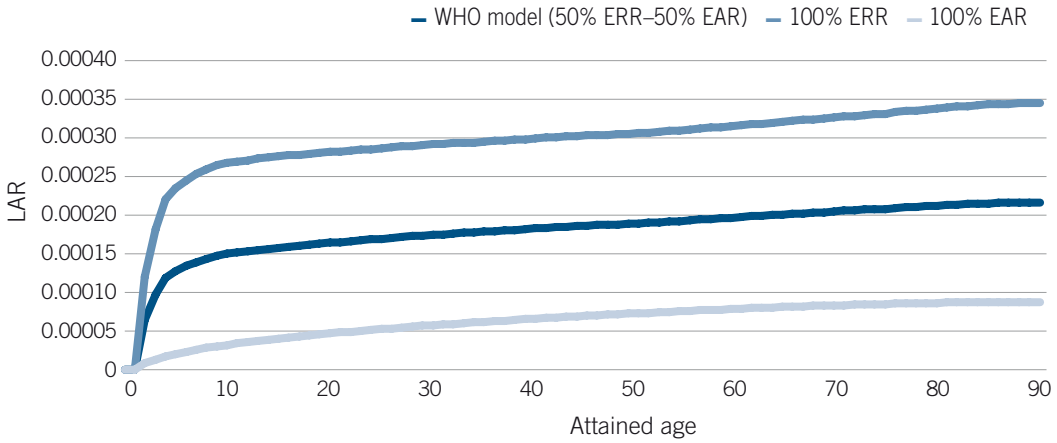


Figure 24. Lifetime attributable risk (LAR) for thyroid cancer in one-year old males (first-year dose 10 mGy) using different transfer weights (100% ERR, 100% EAR and 50% ERR-50% EAR)

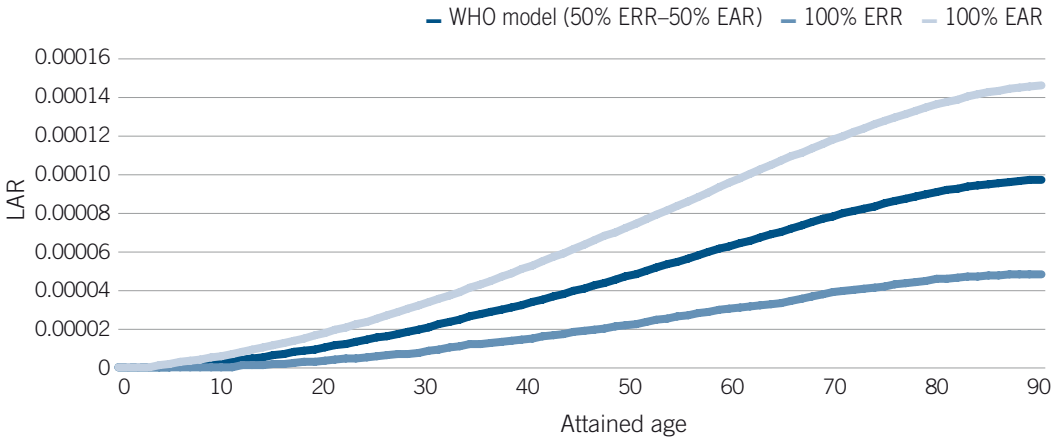


Figure 25. Lifetime attributable risk (LAR) for solid cancer in one-year old males (first-year dose 10 mGy) using different transfer weights (100% ERR, 100% EAR and 50% ERR-50% EAR)

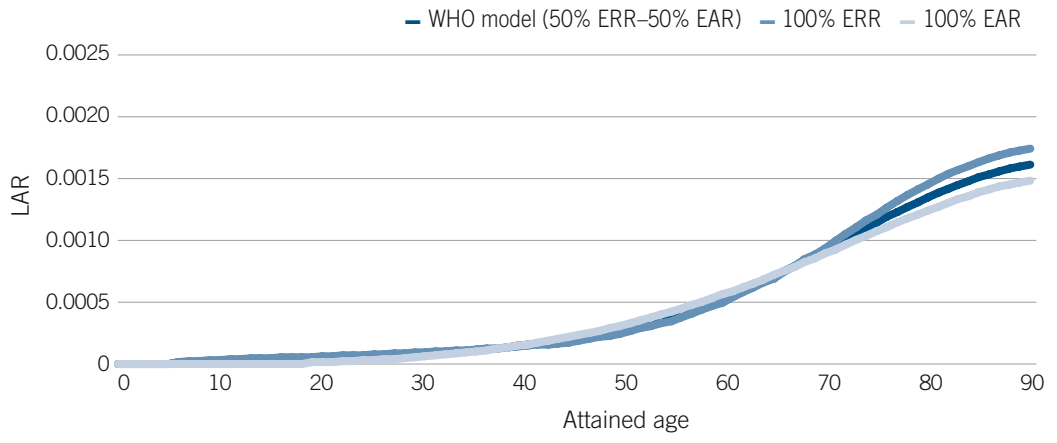


Table 17. Comparison of LAR for all solid cancer calculated using first year doses vs. lifetime doses (20 year old female)

Locations	First year exposure	Lifetime exposure
①	0.567	0.591
②	0.309	0.336
⑤ – ⑭	0.103	0.171

6.2.2 Health statistics

The HRA Expert Group agreed that cancer data from Fukushima were likely to be comparable to those from other parts of Japan. This determination was made on the basis of the similarity of cancer incidence in two neighbouring prefectures for which cancer registries are available (Miyagi and Yamagata) and the other Japanese cancer registries. Also, similarities were found between cancer mortality data in those two neighbouring prefectures compared with cancer mortality data in Fukushima and data from the rest of Japan (although it is noted that thyroid cancer data were insufficient to make a comparison for this organ).

As cancer registries were an important data source for this study, the HRA Expert Group looked for evidence of the robustness of these data sets. First, it was noted that the increasing trend in cancer incidence and declining mortality among both men and women in Japan for the 1984–2005 period was in agreement with international trends. An examination of skin cancer data from registries provided a specific opportunity to probe for registry reliability, as reported disease rates are very low in Japan (on the order of 5% of Caucasian rates). A comparison with the Hawaiian registry (which allows comparison of Japanese and Caucasian populations with similar environmental exposure and the same systems for ascertainment) shows that reported rates of skin cancer in Japan appear to be consistent with the incidence of disease in the Japanese population and confirm that low reporting from Japanese registries is not a result of poor case ascertainment.

Ideally, the ICD codes for the input data (mortality and incidence reference rates) should match those of the data used to fit the incidence risk models for all solid cancers, thyroid cancer and breast cancer (*70*) and the mortality risk models for leukaemia (*83*). In practice, incomplete concordance between some ICD codes was found, but discrepancies were not large, and hence the HRA Expert Group considered that this would not constitute an impediment to using the proposed risk models. The cancer incidence used for this assessment (*104*) corresponds to statistics from 2004, and mortality data used to build the survival curves are more recent (2010). This mismatch was acknowledged by the HRA Expert Group, which considered that it would not substantially affect the results.

6.2.3 Risk models applied

As explained in section 3.3, the HRA Expert Group adopted a hybrid model combining relative and absolute risk approaches for transferring risks estimated from the Japanese atomic bomb survivors (i.e. LSS cohort) to the Japanese population exposed to radiation from the Fukushima Daiichi NPP accident, except for breast cancer for which a pure absolute risk model was used (see Table 3). In addition, calculations were performed for the

pure absolute and relative risk models for leukaemia, thyroid cancer and solid cancers that are also included in the results tables provided in Annexes J and K.

When choosing an approach to transfer radiation-related cancer risks from one population to another, considerations should include comparison of radiation risks from epidemiological studies in populations with different baseline cancer rates. The knowledge about the interactions between radiation and other cancer risk factors (e.g. environmental and genetic cancer risk factors) and their influence on baseline cancer rates is still limited, and this represents one of the sources of uncertainties related to the risk-transfer models applied.

The risk-transfer approach adopted by the HRA Expert Group (i.e. the hybrid model) is considered to be a reasonable compromise that provides an intermediate estimate of the risks, as seen in Figures 23–25.

The differences between the relative and absolute risk models were largest for young age-at-exposure. When risks were predicted over a 15-year observation period, excess risk estimates for all solid cancers and leukaemia obtained using the relative risk model were substantially higher than those predicted by the absolute risk model for the children exposed at 1 year of age, particularly for girls (ERR:EAR ratio of around 5 for 1-year-old males and 6 for 1-year-old females). For thyroid cancer, the relation was reversed. For exposure at 10 years of age, the risk estimates for all solid cancers in boys were comparable and the relative risk model gave slightly higher estimates than the absolute risk model among girls (ERR:EAR ratio of around 1.4). When risks were predicted over a lifetime, differences between the two models were not evident for all solid cancers but persisted for leukaemia and thyroid cancer.

In addition to the uncertainties associated with exposure estimates and other input data, the proposed model structure and parameters are also based on common assumptions derived from uncertain values. Uncertainty attached to the model definition includes estimates of uncertainty on the parameter values of EAR and ERR models (drawn from the literature), latency, weighting of EAR versus ERR, and other modelling assumptions, such as survival curve parameters.

Overall, the model assumptions were either conservative (i.e. they are intended to overestimate rather than underestimate the radiation-related risk) or consistent with the published literature. Preliminary sensitivity analysis of LAR estimates with respect to these model assumptions showed that uncertainty in model parameters was likely to be lower than uncertainty in exposure, so that if other risk models were applied the uncertainties would still be dominated by those related to exposure. It is noted that the chosen model structure becomes highly hypothetical for very low-dose exposures so that the proposed risk estimates are reasonably robust only for those proposed exposure scenarios for which risk quantification was judged to be feasible.

Radiogenic risks in children are generally associated with more uncertainty than risks in the entire population. One reason for this is the fact that the LSS atomic bomb cohort has many survivors who were children at the time of the bombing and who still have years of life to express the risk.

Further research is still needed before achieving definitive conclusions about the optimal choice for risk-transfer between populations (*119*). The risk-transfer approach adopted by

the HRA Expert Group to assess thyroid cancer risk (i.e. the hybrid model mixing 50% ERR and 50% EAR) results in higher risk estimates than the pure relative risk model approach (i.e. 100% ERR). This can be illustrated by comparing the cumulative attributable risks over the first 15 years after the accident (AR_{15}) presented in Annex J. As seen in Table 43, the AR_{15} is estimated to be 3 in 10 000 for female infants in the most affected Group 1 location using the hybrid risk-transfer approach, while using the 100% ERR approach the AR_{15} value is 1 in 10 000. In choosing a hybrid model for thyroid cancer risk-transfer instead of the 100% ERR model adopted by other international bodies such as ICRP (*12*), BEIR VII (*87*) and EPA (88), the HRA Expert Group took into account the extrapolation of LSS results to shorter times after exposure based on information from Chernobyl (see Annex E section E.3). It was also considered that the pure application of the ERR to calculate the LAR for thyroid cancer in this HRA report might have involved more uncertainties, particularly because of the very low baseline incidence rates at young ages[2].

Thyroid cancer risks in workers deserve particular consideration in the context of the models applied. Scenario 4 assumes the highest effective dose (700 mSv) with the radioiodine inhalation pathway being the major contributor, resulting in a very high thyroid dose (>10 Sv). This scenario, which could be considered as an upper bound, represents a few (< 0.01%) of the emergency workers. The assessment of cancer risks at such high thyroid doses is associated with high uncertainties because, as discussed in section 3.4, a flattening of the dose-response for thyroid cancer has been observed at high doses (*6,24,107,120*). Therefore, the actual risks are probably not higher than, and may well be lower than those indicated by the calculated LAR values.

6.2.4 Extrapolation of data from moderate doses to low doses

To date, neither radiobiological nor epidemiological research has provided a definitive answer to the question of whether or not the use of a factor to extrapolate epidemiological data from moderate doses to low doses is warranted. The HRA Expert Group based the risk calculations on models derived from the atomic bomb survivors' cohort without applying any factor for low dose or low dose rate. Correspondingly, a linear dose-response model is used for solid cancer, and a linear-quadratic dose-response model is used for leukaemia. As leukaemia models are linear-quadratic there is no need to apply any DDREF. With respect to solid cancers, the experts chose not to apply a factor, based on the evidence cited in section 3.6. LAR values are proportional to ERR and the concept of LAR is based on linear dose-responses: therefore, any number could be taken into account for DDREF if warranted in the future and could be applied directly to the LAR values presented in this report.

6.3 Specific considerations

6.3.1 Occupational radiation safety

The annual limit of effective dose for occupational exposures in normal situations established in the International Radiation Basic Safety Standards (BSS) (*121*) is 20 mSv

2. The thyroid cancer incidence data for Japan show zero incidence rates for some of the youngest age groups, suggesting that a risk-transfer approach not purely based on a 100% ERR transfer would be more appropriate for this population.

per year averaged over five consecutive years (i.e. 100 mSv in 5 years), and 50 mSv in any single year. As described in section 4.2 less than 5% of the workers exceeded the radiation dose limit for a single year under the normal working conditions classified in the BSS as "planned exposure situations".

Dose limits do not apply to emergency exposure situations. However, it has been internationally agreed that no emergency worker should exceed the annual limit of effective dose for occupational exposures referred to above other than in exceptional circumstances, for which guidance values are provided. All reasonable efforts shall be made to keep doses for emergency workers below 500 mSv for lifesaving interventions, actions to prevent severe deterministic effects or catastrophic conditions that could affect people and the environment.

Before the Fukushima Daiichi NPP accident, Japan had established 100 mSv as a guidance value for emergency workers. This value was revised to 250 mSv on 14 March 2011 (i.e. 50% of the BSS guidance value referred to above). This new regulation was applicable only to emergency workers responding to this particular accident. Data provided by TEPCO indicate that, by the end of January 2012, around 1% of the emergency workers had received more than 100 mSv and 0.03% of the workers exceeded the guidance value of 250 mSv.

6.3.2 Health burden

It is important to note that the present HRA estimates relate to cancer incidence rather than cancer mortality. For consideration of the health burden, an approach to account for disease severity can be considered. For instance, the ICRP 103 model-weighting approach (*12*) quantifies the harmful effect of radiation exposure, taking into account the radiation detriment related to the disease severity (lethality of the disease and years of life lost). For example, non-melanoma skin cancer (NMSC) is rarely lethal and surgical treatment is highly effective. Therefore NMSC does not have the same implications for health burden compared with other solid cancers such as pancreatic cancer, where curative treatment is rarely possible. In contrast to pancreatic cancer, treatment strategies for thyroid cancer are very effective and therefore mortality is very low. This consideration about the improvement of cancer cure rates is also applicable to leukaemia and breast cancer. When assessing and analyzing risk of all solid cancers it should be kept in mind that all solid cancers include various diseases that may have very different health burdens.

6.3.3 Prenatal exposure and carcinogenic risks

Since the first reports that *in utero* radiation was associated with an increased risk of leukaemia and solid cancers during childhood were published in the late 1950s, this issue has been debated. The estimated excess absolute risk (EAR) for childhood cancer due to prenatal exposure derived from case-control studies is around 0.06 per Gy for all cancers and about 0.025 per Gy for leukaemia (*122*), although the evidence from cohort studies is equivocal (*123*). Few studies have addressed the potential risk of adult cancer after *in utero* exposure. An increased risk of adult-onset solid cancers was observed in atomic bomb survivors exposed *in utero*, and risks were lower than risk among those exposed early in life. Many survivors of the LSS atomic bomb survivors cohort who were exposed *in utero* are now in the period of life when the baseline incidence of adulthood cancer markedly rises, so more data can be expected in the future.

There is evidence-based international consensus that the lifetime risk of cancer following exposure *in utero* does not differ greatly from that following exposure in infancy, nor does the risk of childhood leukaemia following exposure *in utero* differ greatly from that following exposure in infancy. On these bases, it can be considered that the lifetime attributable risks for leukaemia and solid cancers after *in utero* exposure do not differ significantly from the LAR values calculated for 1-year-old infants. As mentioned in section 2.2.1, this criterion was adopted by the HRA expert group for the present assessment.

Although consensus effectively exists on increases in the lifetime risk of cancer and the risk of childhood leukaemia following in utero exposure to radiation, there remains some controversy about the risk of child onset cancers other than leukaemia. Following postnatal radiation exposures at early ages, there is no clear evidence of an increased risk of solid cancers during childhood or adolescence from exposures in the low or moderate dose range, although some types of cancer are seen at high doses. Thyroid cancer, which is not a typical childhood cancer, is an exception, because firm evidence exists for increased risk in individuals exposed in childhood during the Chernobyl accident. In contrast, neither the Chernobyl study (*124*) nor the atomic bomb study (*125*) showed a statistically significant increase of thyroid cancer risk after *in utero* exposure, probably due to low statistical power. Whatever the explanation for this apparent difference between *in utero* and postnatal exposures, childhood cancers make up a relatively small component of cancer risk over a lifetime.

6.3.4 Assessment of all solid cancer risks

This HRA considers the risk of all solid cancers combined, which includes the risk of thyroid and breast cancer. No model to calculate the risk for all other solid cancer excluding breast and thyroid cancer risks is available from the LSS data.

The assessment of the risk of all solid cancers combined is intended to provide, together with the assessment of the risk of leukaemia, an overall indication of the lifetime risk of cancer and this was the reason why it was included in the present HRA. In the LSS, the risk of all solid cancers combined uses a dose to tissues based on the colon organ dose as a surrogate for the averaged whole-body dose.

In circumstances where the tissue doses are highly heterogeneous, such as with thyroid exposure to radioactive iodine, this approach can lead to underestimation of cancer risks in specific tissues. When an intake of radioactive iodine occurs, the resulting dose is predominantly to the thyroid, leading to a thyroid dose that is greater than the colon dose, so that the risk of all solid cancers combined will not, under these circumstances, fully account for the risk of thyroid cancer. In contrast, the risk of thyroid cancer assessed as a specific cancer site is based on the thyroid dose and therefore does take account of the tissue dose distribution resulting from the intake of radioactive iodine.

6.3.5 Iodine status and thyroid disease

It is well-known that iodine status influences the avidity of the thyroid gland to concentrate radioactive iodine. Whether, in addition, iodine status can modify the radiation effect in terms of cancer risk is not clear. In view of pre-existing iodine deficiency in the areas affected by the Chernobyl fallout, several studies addressed this issue. A case control study used environmental indicators to assess the stable iodine intake at the time of

exposure in the contaminated regions of Belarus and Russia (*177*). Thyroid cancer risk was modified by both soil iodine content and iodine consumption in the years after the accident for prevention of iodine deficiency. Consumption of stable iodine was associated with a reduction in the radiation-induced thyroid cancer risk to approximately one third. Correspondingly, the risk related to radiation was three fold in the areas with the lowest relative to the highest amount of soil iodine content. Some indication of a higher thyroid cancer risk for areas with lower iodine intake (assessed as excretion, though with a narrow range of variation) was also reported from the Bryansk region in Russia (*126*), though a cohort study in Belarus did not find an association of the background level of stable iodine intake with thyroid cancer risk (*127*).

In contrast to the region of Chernobyl, Japan has a diet with one of the highest iodine contents (e.g. sea fish, shellfish and seaweed), although not all diets in Japan are rich in iodine (*128,129*). A positive association between seaweed consumption and the risk of thyroid cancer (especially for papillary carcinoma) in postmenopausal women in Japan was recently reported (*130*). Existing scientific evidence does not support any quantitative estimation of this potential source of uncertainty in the assessment of thyroid cancer risks. However, this factor has been minimized in the present assessment because it is based on risk models derived from a Japanese population (the LSS cohort).

6.4 Summary of key choices

Table 18 summarizes the key choices made in this HRA and indicates areas where conservative approaches have been adopted.

Table 18. Summary of key choices in the health risk assessment

Selection of input data	
Exposure data	Dose estimates for the general population were taken from the WHO Preliminary Dose Estimation Report, where efforts were made to prevent underestimation of doses (*3*). Some of the conservative assumptions that may have resulted in dose overestimation are: ■ that relocation in the "deliberate evacuation area" took place at four months (though some inhabitants of this area were subjected to relocation earlier than this); ■ that consumers only ate food produced in the area where monitoring was implemented (i.e., that those living in Fukushima ate only food produced in Fukushima, though this was not always the case); ■ that all the food monitored was on the market, although the data set included the results of food samples collected for monitoring purposes, which were not allowed on the market.
Lifetime dose	A ratio of lifetime dose to first-year dose (of two) was selected based on i) experience from Chernobyl (ratio of three), (ii) consideration of differences such as a lower proportion of the long-lived 137Cs in Fukushima than in Chernobyl, and iii) information about ongoing and planned protective measures and remedial actions in Japan. Additional remedial actions can further reduce the calculated ratio of lifetime to first-year dose (section 4.1.4).
Health statistics data	The 6-year difference between available cancer incidence data (2004) and cancer mortality data (2010) was assumed not to introduce significant bias. No alternative data sets were available when starting the collection of input data for the risk models*. (section 5.1)
Incidence vs. mortality data for cancer	The major health risk indicator used in this HRA was cancer incidence rather than cancer mortality. Many cancers have a high chance to cure, a chance that is increasing with time. From a public health perspective, incidence maximizes relevance to populations with different health systems, while mortality is affected by the strength of health systems, screening programs, and access to early treatment (section 3.4).
Adjusted survival curves vs. survival curves	This HRA selected "cancer-free" survival (adjusted survival curve) rather than overall survival as more suitable for the calculation of LAR and LBR of cancer related to this radiation event (Annex D).
International classification of diseases (ICD)	There was incomplete concordance of ICD codes between LSS data and Japan health statistics data for breast and all solid cancers, but this did not substantially affect the results. (section 5.1.3; Annex E, sections E1 to E4).
Healthy worker effect (HWE)	The health statistics and demographic data used in the present HRA for the emergency workers were the same as in the general population. In general the mortality rates for workers tend to be lower than for the general population (HWE), and this choice might overestimate all-cause mortality risk in workers (Annex D). The health statistics and demographic data used in this HRA for the emergency workers were the same as in the general population. In general, the mortality rates for workers tend to be lower than for the general population (HWE) and this choice might overestimate all-cause mortality risk in workers (Annex D). No alternative data were available.
Assumed exposure scenarios for workers	The assumed exposure scenarios 1 and 2 are considered to be representative of about 99% of the emergency workforce, while scenarios 3 and 4 represent upper bounds of external and internal exposure respectively, representing less than 1 % of the workers. (section 4.2.4). In particular, scenario 4 would be only applicable to <0.01% of the workforce (i.e. a few workers) who received the highest effective dose, with a very high thyroid dose due to inhalation of radioactive iodine.

* Cancer incidence data from 2006 became available later, showing a trend to higher incidence rates for female breast cancer and thyroid cancer compared to the 2004 rates (for age ranges of 45-80 years and 25-55 years respectively). For all solid cancers and leukaemia the rates just show random variation. This may result in higher LAR estimates for thyroid cancer in adults if they are calculated based on the 2006 rates. However this would not affect the lifetime fractional risk (LFR) estimates.

Selection of models and approaches	
Non-threshold models	For the low-dose radiation exposures estimated from the Fukushima-Daiichi accident, the best approach was considered to be the use of a linear non-threshold (LNT) model for solid cancers, and a linear-quadratic non-threshold model for leukaemia (Annex E).
Dose and dose rate effectiveness factor (DDREF)	Epidemiology does not provide support for the use of a DDREF for extrapolating risks from high or moderate to low doses.
Selection of cancer sites	Leukaemia, thyroid cancer and female breast cancer were modelled separately from other cancers because of the known radiosensitivity of these tissues and the demonstrated dependence of their risk on the age-at-exposure. Moreover, thyroid cancer is especially relevant to this HRA because of the release of radioactive iodine from the Fukushima Daiichi NPP. In addition, to provide an overall assessment of cancer this HRA used the "all solid cancer" risk model (not subtracting for breast and thyroid, as no such model is available from the LSS data) (section 2.2.1).
Latency periods	Minimum latency period was applied based on the ones reported in the literature (i.e. 2 years for leukaemia, 3 for thyroid cancer; 5 for breast cancer and all solid cancer). For breast cancer, the youngest age of disease onset of age 20 years is supported by epidemiological evidence, including populations with childhood radiation exposure (section 3.3.2).
Selected age at exposure	The three age groups considered for this HRA (i.e. infants aged 1 year, children aged 10 years, and adults aged 20 years) were selected to ensure representation of the youngest, most radiosensitive members of the population. The Japanese population has long been among the oldest in the world. Therefore, the choice of a 20 year age-at-exposure to represent an adult population, as well as the selection of a 1-year-old to represent all children under age 10, represents the most conservative scenarios.
Adopted risk quantity	The lifetime attributable risk (LAR) was selected as a simple quantity to quantify risk. At the low doses estimated from this accident, it is equivalent to the more complex risk of exposure-induced death (REID).
Models based on atomic bomb survivors vs. a nuclear accident	Despite the differences between types of exposure from the atomic bomb (largely external exposure) and nuclear accidents (internal and external exposure), LSS models were used in this study because they provide the largest body of epidemiological data on cancer and non-cancer radiation risks. Calculations of risk for thyroid cancer took data from the Chernobyl accident into consideration (Annex E, section E.3).
Transfer weights for each cancer sites	Overall, the choices of the transfer weights were either consistent with the published literature or were more conservative. The influence of the choice of the transfer weights on the LAR results can be seen in figures 23-25 (section 6.2.2). In general this choice did not substantially affect the LAR results.
Thyroid cancer risk at high doses	Epidemiological data suggest a flattening of the dose-response for thyroid cancer risk at very high doses. Therefore, thyroid cancer risks estimated for workers in scenario 4 in the present HRA may be overestimated.
Workers' sex and age-at-exposure	As only a few female workers were involved in the early response and their doses were very low, using males to represent all workers is a realistic choice. The selected ages-at-exposure (20, 40, 60) are judged sufficient to represent the workforce. Only 10% of the workforce was younger than 29 years at the time of the accident, while almost 40% were over 50 years old (Table 7 section 4.2.3). Taking into account that cancer risks are lower at older age-at-exposure, the workers' HRA provides conservative risk estimates (section 6.1.1). This is particularly relevant for thyroid cancer because epidemiological data indicate that the risk decreases significantly with increasing age-at-exposure, with little risk apparent after age 20 years (section 2.2.1).

7. Public health considerations

The Fukushima Daiichi NPP accident took place in the context of a natural disaster that caused catastrophic loss of life and massive loss of property. Owing to this unique association of an earthquake followed by a tsunami that caused a nuclear accident, this event is referred to as a "combined disaster" (*133*). An overview of the public health issues related to the disaster has been published by the WHO Western Pacific Regional Office (*134*). The health risks assessed in the present report focus on those effects potentially related to the radiation exposure resulting from the Fukushima Daiichi NPP, mainly cancer and some non-cancer outcomes. However, it should be kept in mind that WHO defines "health" as "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" (*135*). Other non-radiation-related health impacts of this combined disaster are discussed in this chapter, including mental health and psychosocial consequences.

7.1 Public health response during the emergency phase of the Fukushima Daiichi NPP accident

As the lead health agency within the United Nations, WHO is responsible for the international public health response in emergencies. The early response of WHO to this combined disaster included the assessment of and response to evolving health concerns related to mental health and the psychosocial impact of the disaster; prevalence of communicable diseases (e.g. acute diarrhoea, respiratory diseases, measles and other vaccine-preventable diseases); non-communicable diseases (NCDs) and priority-setting for chronically and critically ill patients who had to be evacuated (e.g. patients on dialysis, insulin-dependent patients, those on post-transplant critical care, or with lung, cardiovascular, and other NCDs).

For public health response in radiation emergencies, WHO relies on specialized technical networks, such as the Radiation Emergency Medical Preparedness and Assistance Network (REMPAN), which includes more than 40 medical and research institutions worldwide that support WHO's work on preparedness and response to radiation emergencies. The importance of global collaboration and coordination of emergency preparedness for timely and efficient emergency response was one of the lessons learned from the Fukushima accident that complements the knowledge gained after the Chernobyl accident (*136*).

The existing arrangements for the coordinated actions of international organizations (*2*) proved efficient during the response to the Fukushima Daiichi NPP accident. International organizations demonstrated a strong commitment to addressing issues related to health, food, drinking water, environment, trade, travel and nuclear safety. As discussed in section 1.1, short-term emergency public health actions were taken in Japan

and around the world to manage and reduce the consequences of the accident. These actions, which in Japan included evacuation, sheltering, and food and drinking water monitoring, were consistent with the internationally recommended criteria for emergency interventions (*137*). In many countries around the world, governments considered measures to protect their citizens with a primary concern for those residing in or visiting the most affected regions of Japan in the days and weeks after the earthquake.

The safety of the food supply was of concern early on and extensive monitoring was put in place in Japan as well as in other countries. The FAO/WHO International Food Safety Authorities Network (INFOSAN) was instrumental in providing technical briefing notes related to food safety aspects and regular updates on food monitoring results to Member States.

7.2 Public health challenges in the recovery phase of the radiation emergency

The recovery phase after a radiological emergency is considered a different type of exposure situation under the current international system of radiological protection, with some specific radiation safety requirements that differ from those applied under emergency situations (*121*) (see Box 8). As a general principle, the introduction of countermeasures should bring the maximum net benefit to the population. All the risks and benefits resulting from a particular measure should therefore be considered in reaching a decision, and this includes both radiological and non-radiological risks (*138*). This is particularly challenging during the emergency response phase, characterized by strategies driven mainly by urgency and predominantly centralized decisions. The transition period between the emergency and the recovery phases is characterized by a change towards more decentralized strategies aimed at improving living conditions, protecting people with the highest exposures and reducing radiation exposure to "as low as reasonably achievable" (the ALARA principle) (*12,96,139*).

The Government of Japan, municipal authorities and residents implemented a number of remedial actions to lower radiation exposure (*100*). Environmental monitoring, including monitoring of food and drinking water, is continuing. In April 2012, the limits for radioactivity in food and drinking-water were reduced in Japan to levels that are consistent

Box 8. Categories of exposure and exposure situations

People may be exposed to radiation as members of the general population (public exposures), as a result of their work (occupational exposures) or for medical purposes (medical exposures). These are the three categories of exposure considered in the international system of radiological protection (*12*). Exposure to radiation can occur under any of the following three types of exposure situations:

- planned exposure situations arising from any planned activity that results in an exposure to radiation (e.g. a patient undergoing a radiological medical procedure);
- emergency exposure situations arising from an accident, a malicious act, or any other unexpected event (e.g. public or occupational exposures during a nuclear emergency);
- existing exposure situations which already exist when a decision on the need for control has to be taken (e.g. radon exposure in dwellings, prolonged exposures after an emergency).

with a maximum annual dose of 1 mSv (*140*). Decontamination and remedial actions are ongoing. The most efficient remediation options need to be considered, to ensure that benefits outweigh the hazards to the environment and people's well-being, recognizing that radioactive contamination is just one component in a complex mix of (often interacting) factors that demand careful attention. With this in mind, priority-setting for remedial actions will help identify target locations where people are expected to stay for prolonged periods (e.g. playgrounds, schools). The results presented in this report support the importance of prioritization with respect to the most sensitive populations (e.g. infants, children, pregnant women), for whom such remedial actions would have the highest impact in terms of reducing long-term risks.

The results of this HRA indicate that the health effects of radiation exposure resulting from the Fukushima Daiichi NPP accident inside and outside Japan are likely to be less ominous than the socioeconomic impact. This particularly applies to residents of Fukushima prefecture. A key issue during the current recovery phase is effective policy- and decision-making for the restoration of a normalcy.

During and after emergencies, public health risk management is supported by scientific evidence, taking into account ethical and social values, socioeconomic factors, and public perceptions and expectations of society. In the case of the Fukushima Daiichi NPP accident, public perceptions and the expectations of stakeholders – e.g. residents, consumers, producers, farmers, manufacturers – are particularly related to radiation safety, levels of risk that are tolerable and degrees of protection that are necessary. It is important to take into account perceptions and expectations when stakeholders are informed about available options and as they are encouraged to undertake self-help protective actions (*96*).

Social interventions aimed at building community strengths, capabilities and self-reliance can help large numbers of people to preserve a sense of social solidarity, to improve the quality of community life. The engagement of the affected population in developing and implementing protective and remedial actions (such as "self-help actions") can reduce people's feelings of vulnerability. Making use of existing mechanisms to promote personal and societal cohesion increases the effectiveness of the radiation protection interventions and may also contribute to recovering and improving mental health (*96,141*). Community-centered interventions that facilitate the involvement of stakeholders from Fukushima and neighbouring prefectures can help individuals express their own needs and to participate in the selection of suitable protective and remedial actions by making informed choices.

As of November 2012, many residents are still unable to return to their homes, and for some there is uncertainty about when – or whether – they will ever be able to go back to their homes and communities. Their engagement in the implementation of remedial actions and recovery plans will help them rebuild their lives.

Risk communication is a key component of the risk analysis process, and is linked closely to risk assessment and risk management. Proactive risk communication, coupled with public involvement in the remedial process, is critical to the success of any remedial activity. Addressing public health concerns is a major communication challenge. The building blocks of an effective risk communication strategy are trust, transparency, ethics, technical accuracy, values, credibility and expression of caring. Different types

of messages may be more – or less – suitable for different audiences (e.g. the general public, policy-makers, decision-makers, the mass media). Fears and perceptions need to be addressed – even if they are not commensurate with the actual risks. It is of utmost importance to prevent reactions that themselves carry risk (such as self-administration of potassium iodide), to allay unnecessary fears (such as avoidance of breastfeeding because of health fears), and to promote healthy coping mechanisms (such as social solidarity).

7.3 Long-term follow-up of populations following radiation emergencies

Programmes for medical monitoring of populations after radiation emergencies are intended to address two different target populations:

- persons who have developed clinical conditions requiring medical assistance during the emergency (e.g. acute radiation syndrome, local radiation injuries);
- asymptomatic persons known (or presumed) to have been exposed to low doses of radiation.

No clinical conditions have been identified after the Fukushima NPP accident for the general population, nor for workers. The current HRA, in particular related to specific cancer risks, identifies the aspects of most concern and helps target follow-up actions with respect to cancer types, age groups and geographic location.

Medical follow-up of asymptomatic persons involved in a radiation emergency poses major concerns regarding the identification of populations at higher risk and whether screening for disease in the "at-risk" population produces more benefits than potential harm. The goal is to detect disease as early as possible, with the assumption that earlier diagnosis will result in reduced morbidity and mortality. In addition, health monitoring and surveillance can provide reassurance in response to the population's concerns about health risks (*96,141*).

Several factors can help ensure that such screening is beneficial:

- Disease risk should be identified in the most vulnerable population or population subgroups (e.g. children, pregnant women).
- An accurate practical screening tool should be available.
- Early detection of the disease must lead to improved survival.
- Effective treatment of the disease should be available.
- The benefits of the screening must be greater than any potential harm (individual and public health dimensions).

A thyroid ultrasound screening programme is currently being conducted in Fukushima prefecture (see Box 9). It is important to ensure that this programme includes a representative sampling of residents, selected from geographical areas with different levels of radionuclide ground deposition over the whole prefecture. The screening should be conducted without knowledge of the specific exposure situation of the person (if at all possible). This ultrasound screening for thyroid disease is likely to lead to an increase

in the incidence of thyroid diseases due to earlier detection of non-symptomatic cases (screening effect).

Information on possible effects of screening programmes on the reported thyroid cancer incidence can be obtained from the Adult Health Study (AHS) of the Japanese survivors of the atomic bombings of Hiroshima and Nagasaki, and from post-Chernobyl studies. The AHS sample is a subset of the full cohort of the atomic bomb survivors Life Span Study (LSS) who volunteered to undergo medical examinations every second year. The reported thyroid cancer incidence in the AHS was higher than in the full LSS cohort due to a screening effect (*70*). A screening effect was also observed among populations who had high thyroid exposures due to the Chernobyl accident and who were subjected to intensified surveillance of thyroid diseases (*142,143*).

The epidemiological follow-up of persons involved in a radiation emergency has the general goal of detecting radiation effects or diseases that are potentially related to radiation exposure. The specific purposes of the follow-up are:

- to identify radiation effects in a group of people known (or presumed) to have been exposed to ionizing radiation;

Box 9. The Fukushima Health Management Survey

The Fukushima prefectural government launched the Fukushima Health Management Survey at the end of June 2011 to assist in the long-term health management of Fukushima residents (*147*). The study, conducted by Fukushima Medical University, has the primary purpose of monitoring the long-term health of residents, promoting their future well-being and determining whether long-term low-dose radiation exposure has health effects. The Fukushima Health Management Survey consists of a basic survey covering the population of Fukushima (2 million people) and four detailed surveys: a thyroid ultrasound examination (residents between 0 and 18 years), a comprehensive health check (residents of all ages living in the evacuation zones), a mental health and lifestyle survey (residents of all ages living in the evacuation zones) and a pregnancy and birth survey (of around 16 000 women who received maternal and child health care in Fukushima prefecture).

The aim of the basic survey is to determine the whereabouts of every prefectural resident from the time of the 11 March nuclear accident onwards (a "record of movement") and will provide a basis for the estimation of individual radiation doses (external exposure) that will then be linked to data on internal exposure as measured by whole-body counters, with detailed data from the other four detailed surveys, cancer registries and vital statistics.

By the end of August 2012 responses from more than 470 000 questionnaires had been collected. The thyroid ultrasound examination collected data from more than 80 000 children and adolescents. Thus far, about 74 000 (April 2011 to March 2012) and 16 000 (March 2012 to April 2012) of 210 000 people have been included in the comprehensive health check, and around 92 000 of 200 000 people have answered the mental health survey. About 9 000 women replied to the questionnaires on pregnancy and birth. This large-scale long-term cohort study is expected to provide data on radiation health effects and disaster-related stress.

At the time of this publication, interim data from the Fukushima Health Management Survey indicate that external exposure for 93.8% of the respondents was less than 5 mSv and that for 99.2% of residents' external exposure was less than 10 mSv, with a highest estimate of 25.1 mSv (*118,148*). Internal exposure was estimated in 81 000 residents by WBC between June 2011 and September 2012; the reported committed effective doses were below 1 mSv in 99.9% of the persons surveyed, and the maximum dose was 3 mSv (*117,118*).

- to determine whether the risk of such effects is statistically significantly greater in this group than in a comparable (e.g. by age and sex) unexposed group of individuals;
- to determine whether any identified increased risk is statistically associated with the exposure;
- to determine whether there is a relationship between the increased risk and other factors (e.g. tobacco smoking, exposure to chemicals);
- to derive and refine risk estimates;
- to plan health interventions as necessary.

In addition to the doses, health monitoring and surveillance of people exposed to low radiation doses necessitates consideration of legal, social, economic and psychological factors, which go beyond the scope of the present report. In some cases, even if there is insufficient evidence to recommend long-term follow-up for stochastic effects (i.e. cancer) from a medical management perspective, it is prudent to develop a registry and conduct epidemiological research.

Several factors influence the possibility of detecting a statistically significant increase in cancer morbidity or mortality through a radiation epidemiology study. The level of radiation dose is a major factor in determining the size of the population to be studied in order to give a sufficient statistical power to the study. Hundreds of thousands or even millions of individuals may be needed to detect an increase after a low-dose radiation exposure, because the differences compared with background incidence or mortality (i.e. baseline rates) are small (*141,144*). Epidemiological detectability in terms of excess absolute risk increases in population subgroups for which baseline rates are lower than those of the general population (e.g. thyroid cancer incidence in children). All these factors must be taken into account when planning and implementing long-term follow-up programmes for the Fukushima Daiichi NPP accident. This topic has been discussed in previous WHO publications (*22,141*).

Population screening and health surveillance programmes often include medical imaging that can help identify an increase in cancers and other conditions. However, medical imaging procedures can result in effective doses that exceed the exposure levels estimated to have occurred in residents of Fukushima prefecture as a result of this accident. The radiation risks of medical imaging, particularly in children, have been extensively addressed and evidence from epidemiological studies is available (*145,146*). The benefits of the medical applications of radiation outweigh the risks when radiation is used but the opposite may be true if procedures are performed without a clear clinical indication. The need for justification of medical exposures is particularly relevant in long-term follow-up of large populations. Mass screening using radiological imaging procedures may result in substantially more radiation exposure and risks than those assessed in the present report (in the general population and in the workers).

In addition to the Fukushima Health Management Survey, as part of occupational health programmes, a special medical follow-up protocol for emergency workers is being followed (see Box 10).

Besides medical follow-up of the most affected parts of the population, continued environmental monitoring and monitoring of the food and water supply, with strict enforce-

ment of existing regulation, is a further important component of the long-term goal of decreasing radiation exposure to be as low as possible.

7.4 Psychological consequences of the accident

Although the assessment of mental health risk is beyond the scope of this HRA, this topic is highlighted as a challenge to the medical community and health authorities that may have an impact at all levels of society. The psychosocial impact is one of the major consequences of nuclear emergencies; this was one of the lessons learned from the Chernobyl accident (*141,149*). As with the Chernobyl accident, the psychological impact of the Fukushima accident may outweigh other health consequences (*150*).

Psychological reactions following disasters may include multiple symptoms such as fear, grief, anxiety, anger, depression and distrust (*151*). These reactions may be exacerbated in radiation emergencies because radiation cannot be perceived by the senses and most people either do not know or do not fully understand the terminology used to express the size of exposures and their potential effects. As a result, community-wide feelings of helplessness and vulnerability may arise. Those disasters with a high degree of uncertainty about potential future health effects are more psychologically traumatic than situations with more visible, immediate and predictable outcomes.

A high incidence of psychosomatic symptoms, psychological distress and psychiatric disorders has been observed among victims of radiation accidents. Parents with young children (*152*), pregnant women, children, elderly persons, emergency workers, people with pre-existing mental disorders, clean-up workers (*153*), evacuees (*154*) and the population as a whole in some instances (*155*) may all be at increased risk. Acute stress reactions typically include physical, emotional, cognitive and interpersonal effects. A persistent state of anxiety may result in chronic stress reactions that have behavioural, emotional and physiological consequences. Some people may develop mental disorders such as post-traumatic stress disorder (PTSD), anxiety disorders, depression and alcohol use disorder.

Box 10. Medical follow-up of emergency workers at TEPCO

All emergency workers responding to the accident at the NPP were enrolled in a medical follow-up programme. Workers who have since left their jobs have an opportunity to continue enrolment. The items included in the medical examination for the follow-up vary according to the doses:

- all workers: general medical examination required by law and psychological evaluation;
- workers > 50 mSv: general medical examination required by law; psychological evaluation; and examination of lens of the eyes (cataract);
- workers > 100 mSv: general medical examination required by law; psychological evaluation, examination of the lens of the eyes (cataract); thyroid test, cancer screening (lung, stomach, and colon).

The medical examination includes clinical evaluation, blood and urine tests, visual and auditory acuity, electrocardiogram, thyroid hormones, thyroid ultrasound, and imaging procedures.

More information is available at:

http://www.mhlw.go.jp/stf/houdou/2r9852000001plbx-att/2r9852000001plen.pdf

The psychological impact of the 2011 Great East Japan Earthquake and Tsunami was compounded by the subsequent nuclear accident at the Fukushima Daiichi NPP. For many people, the Fukushima Daiichi NPP accident resulted in many stressors that constitute a potentially traumatic situation. In addition to the significant impact of the loss of lives and missing loved ones because of the earthquake and tsunami (*134*), other conditions such as evacuation, relocation, material and financial loss – as well as fear and uncertainty related to radiation exposure and its potential consequences – increased the mental health impact of the combined disaster. As of September 2012, 329 777 people remain relocated or evacuated (*156*).

Early management of mental health issues is important (*141,157,158*). Most people with common psychiatric symptoms such as anxiety and depression do not seek professional care, and those who do seek care often present to general practitioners or paediatricians with physical symptoms. Consequently, medical practitioners need to understand the full scope of health effects of radiation exposure, to recognize and manage psychosomatic, anxiety or depression symptoms and to treat mental and physical health with equal respect (*159*).

The psychosocial effects of radiation accidents may extend far beyond the geographical area of impact because of people's worries about future risks. The size of the population exhibiting chronic stress may be quite large and social stigma attached to residents of affected areas may exacerbate the problems.

8. Summary and conclusions

This report presents the results of a HRA of the Fukushima Daiichi NPP accident for the general population and for workers dealing with the emergency. The assessed health risks include cancer and non-cancer effects of exposure to radiation, encompassing both stochastic and deterministic health effects.

8.1 Health risk assessment in the general population

Health risks in the general population were assessed in different geographical locations inside and outside Japan, for both sexes and three age-at-exposure groups: 1-year-olds (infants), 10-year-olds (children) and 20-year-olds (young adults). These age groups were considered suitable to permit the characterization of risks for younger and more sensitive populations.

Based on the doses estimated to be substantially below threshold levels, deterministic effects (i.e. tissue reactions) are not expected. For this reason, no increase in the incidence of spontaneous abortions, miscarriages, perinatal mortality, congenital malformations, developmental abnormalities or cognitive impairment is expected as a result of *in utero* radiation exposure.

A risk of radiation-induced hereditary effects has not been definitively demonstrated in human populations. Based on animal data, international scientific bodies consider that any risk of hereditary effects for the offspring of those who were exposed before they have conceived children would be much lower than the additional lifetime risk of cancer for the exposed individual him- or herself (about one order of magnitude lower).

The present results suggest that the increases in the incidence of human disease attributable to the additional radiation exposure from the Fukushima Daiichi NPP accident are likely to remain below detectable levels.

The predicted magnitude of cancer risks was assessed for leukaemia, thyroid cancer, female breast cancer and all solid cancers combined. The risks were calculated over a lifetime and over the 15 years following the accident. The lifetime attributable risks (LAR) were quantitatively estimated only in the most affected parts of Fukushima prefecture. For all other locations in Japan and around the world, the radiation-related cancer risks were estimated to be much lower than the usual fluctuation in the baseline cancer risks.

The results show the largest additional cancer risks among those exposed in infancy (leukaemia in males and solid cancers in females). Given the exposure to radioactive iodine, during the early phase of the emergency, the lifetime attributable risk of thyroid cancer was specifically assessed. The results show the greatest risk among girls exposed as infants in the most affected area in Fukushima prefecture, although the excess absolute risk is small, because of the low baseline risk of thyroid cancer, it represents a comparatively high relative increase in the lifetime risk of up to around 70% (as an upper

bound). The high relative risk of childhood thyroid cancer becomes more evident when risks are calculated over the first 15 years after the accident for those exposed as infants, because the baseline thyroid cancer risk in early life is very low. Monitoring children's health is therefore warranted.

The risk of leukaemia as a result of radiation exposure from the accident was assessed to be greatest in males exposed as infants in geographical locations with the highest exposure, slightly above 5% over baseline risk as an upper bound. A similar result is found for breast cancer in girls exposed as infants. For all solid cancers, a maximum relative increase of about 4% was estimated.

8.2 Health risk assessment in emergency workers

To date, the Fukushima Daiichi NPP accident has not resulted in acute radiation effects among workers. None of the seven reported deaths among workers is attributable to radiation exposure[1]. Thyroid dysfunction was reported in three workers as a result of repeated self-administration of stable potassium iodide for thyroid blocking against radioactive iodine. This effect was transient and thyroid function returned to normal once the administrations were stopped.

The potential health consequences of exposure to radiation of the emergency workers have been assessed assuming four scenarios that describe different exposure patterns. Scenario 1 represents around two thirds of the emergency workers with quite low doses to all tissues. Scenario 2 contains about one third of the emergency workforce who received moderate thyroid doses and lower doses to other tissues. Scenarios 3 and 4 represent upper bounds for external and internal exposure respectively. Scenario 3 concerns less than 1% of workers who received higher, more homogeneous tissue doses (including thyroid doses). Scenario 4 relates to those few workers who received high thyroid doses as a result of inhalation of radioactive iodine and lower doses to other tissues.

Because tissue doses received were below threshold doses, no deterministic effects of radiation are expected in the workers, apart from possible thyroid disorders in those few workers who inhaled significant quantities of radioactive iodine.

Cancer risks were calculated for workers aged 20 years, 40 years and 60 years. The estimated risks were consistently lower for workers exposed at an older age. Relevant findings are summarized below. For around two thirds of the emergency workers (Scenario 1), all calculated risks are of similar magnitude as the normal fluctuations in the baseline cancer risks. For about one third of the workers (Scenario 2), the relative increase over background for thyroid cancer is estimated to be up to 20% for the youngest workers. For less than 1% of workers (Scenario 3), the relative increase over background for leukaemia and thyroid cancer is as high as 28% in the youngest workers. For those few emergency workers who received very high doses to the thyroid (Scenario 4), a notable risk of thyroid cancer is estimated, especially for young workers.

1. The causes of these deaths have been reported as disaster-related (two cases), heart attack (three cases), sepsis (one case) and leukaemia (one case for which the time of the onset was shorter than the minimum latency period for radiation-induced leukaemia).

There may be an increased risk of long-term circulatory disease among workers with the highest doses (Scenarios 3 and 4), which is likely to be substantially smaller than any additional cancer risk.

The considerations made above for heritable risks in the general population (section 8.1) are also applicable to workers.

8.3 Final considerations

This HRA was drawn up to give an indication of the health implications of the Fukushima NPP accident for the identification of needs and priorities for public health actions. The estimates presented in the report must be regarded as indicative of the magnitude of the health risks based on best judgment rather than as precise predictions.

The estimation of radiation risks, at doses below which increases in cancer incidence have been readily observed in epidemiological studies, involves a number of uncertainties. The main sources of uncertainty in this HRA were discussed in chapter 6. Owing to the preliminary nature of the dose estimation and the time frame for the HRA Expert Group to complete its work, a fully quantitative assessment of the uncertainties associated with the LAR central estimates was not performed.

Although substantial information on radiation risks is available, further evidence would be highly desirable. This accident highlights the need for continuing and improving low-dose and low-dose-rate radiation research.

To avoid any underestimation of risks, the HRA Expert Group adopted the LNT model as the most reasonable approximation of the relation between low-dose radiation exposure and cancer risks and made the prudent choice of not applying a dose and dose rate effectiveness factor (DDREF). Because this HRA is based on a LNT model and the risk quantity adopted (LAR) is proportional to the dose, more refined risk estimations can be provided in the future if a more detailed dose assessment becomes available.

The HRA Expert Group considers the risk estimates robust on the basis of existing knowledge and information at the time of this assessment. The input data and risk models used are considered to be the most appropriate at present. An effort was made to avoid any underestimation of risks when adopting assumptions; hence, any possible bias is likely directed toward overestimation of health risks.

This HRA is not intended to provide estimates of disease burden in the population or cases of excess disease resulting from radiation exposure. This report uses preliminary dose estimates based on environmental and food monitoring data for the calculation of lifetime attributable risks. This makes no assumption of how many persons were exposed according to the different exposure scenarios, for which distributions of individual dose estimates are needed. In addition to the environmental and food monitoring data, an evaluation of the population dose distribution requires the knowledge of the behaviour of persons, e.g. how much time they spent in the differently exposed areas. Moreover, population figures by sex, age and area were not considered for this report, especially in light of the expected substantial migration and movement in the months following the accident. Reliable numbers would be needed for the estimation of population doses and associated risks. Provided these become available, such assessment could be performed

in future studies, such as the upcoming 2-year UNSCEAR study and the on-going survey conducted in Fukushima prefecture to determine the whereabouts of the residents.

It is important to note that this is a report on radiation health risks and that it does not refer to radiation-induced health effects. While radiation risks can be estimated prospectively, radiation-induced health effects are assessed retrospectively and this requires a long-term follow-up of the exposed population. Surveillance of health and monitoring of disease occurrence is required for empirical assessment of the health consequences of the accident and quantification of health outcomes resulting from it.

The Fukushima Health Management Survey is expected to contribute to future health effect assessments. Population health surveillance will permit the identification of additional needs for the delivery of health care. In addition, as part of the occupational health programmes, a special protocol for medical follow-up of emergency workers is being adopted. These initiatives are also relevant for the mitigation of the psychosocial impact of this accident and the prevention of adverse mental health consequences, which are considered to be of major significance.

References

1. *Final report of Investigation Committee on the Accident at Fukushima Nuclear Power plants of Tokyo Electric Power Company*. Tokyo, Tokyo Electric Power Company, 23 July 2012 (http://icanps.go.jp/eng/final-report.html, accessed 23 November 2012).
2. *Joint radiation emergency management plan of the international organizations. Emergency preparedness and response*. Vienna, International Atomic Energy Agency, 2010 (http://www–pub.iaea.org/books/IAEABooks/8453/Joint–Radiation–Emergency–Management–Plan–of–the–International–Organizations–EPR–JPLAN–2010–Emergency–Preparedness–and–Response, accessed 16 November 2012).
3. *Preliminary dose estimation from the nuclear accident after the 2011 Great East Japan earthquake and tsunami*. Geneva, World Health Organization, 2012 (http://www.who.int/ionizing_radiation/pub_meet/fukushima_dose_assessment/en/index.html. accessed 28 December 2012).
4. *Low-dose extrapolation of radiation-related cancer risk*. Ottawa, International Commission on Radiological Protection, 2005 (Annals of the ICRP, 99(35):4).
5. Preston DL et al. Cancer incidence in atomic bomb survivors. Part III: leukaemia, lymphoma and multiple myeloma, 1950–1987, *Radiation Research*, 1994:S68–S97.
6. Ron E et al. Thyroid cancer after exposure to external radiation: a pooled analysis of seven studies. *Radiation Research*, 1995, 141:259–277.
7. Land CE et al. Incidence of female breast cancer among atomic bomb survivors, Hiroshima and Nagasaki, 1950–1990. *Radiation Research*, 2003, 160:707–717.
8. *Principles & methods for the risk assessment of chemicals in food*. A joint publication of the Food and Agriculture Organization of the United Nations & the World Health Organization, 2009 (IPCS Environmental health criteria 240).
9. Chino M et al. Preliminary estimation of release amounts of ^{131}I and ^{137}Cs accidentally discharged from the Fukushima Daiichi nuclear power plant into the atmosphere. *Journal of Nuclear Science and Technology*, 2011, 48(7):1129–1134.
10. *Regarding the evaluation of the conditions on reactor cores of Units 1, 2 and 3 related to the accident at Fukushima Daiichi nuclear power plant*. Tokyo, Nuclear and Industrial Safety Agency, 2011 (www.nisa.meti.go.jp/english/press/2011/06/en20110615–5.pdf, accessed 21 November 2012).
11. Leggett RW et al. A physiologically based biokinetic model for caesium in the human body. *Science of the Total Environment*, 2003, 317(1–3):235–255.
12. *The 2007 Recommendations of the International Commission on Radiological Protection*. Ottawa, International Commission on Radiological Protection, 2007 (Annals of the ICRP, 103(37):2–4).
13. *World cancer report*. Lyon, International Agency for Research on Cancer, 2008 (http://www.iarc.fr/en/publications/pdfs–online/wcr/2008/wcr_2008.pdf).
14. *Estimated cancer incidence, mortality, prevalence and disability–adjusted life years (DALYs) worldwide in 2008*. GLOBOCAN 2008 database, version 3.0. Lyon, International Agency for Research on Cancer (http://globocan.iarc.fr/, accessed 16 November 2012).

15. *Radiation: a review of human carcinogens*. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol.100D. Lyon, International Agency for Research on Cancer, 2012.
16. Douple EB et al. Long-term radiation-related health effects in a unique human population: lessons learned from the atomic bomb survivors of Hiroshima and Nagasaki. *Disaster Medicine and Public Health Preparedness*, 2011, 5:S122–S133.
17. Tsushima H et al. Late effect of atomic bomb radiation on myeloid disorders: leukemia and myelodysplastic syndromes. *International Journal of Hematology*, 2012, 95(3): 232–238.
18. Toranosuke I et al. Leukemia in Atomic Bomb Survivors, Hiroshima and Nagasaki, 1950–1966. *Radiation Research*, 1971, 45(1):216–233.
19. Ozasa K et al. Studies of the mortality of atomic bomb survivors, Report 14, 1950–2003: an overview of cancer and non-cancer diseases. *Radiation Research*, 2012, 177(3):229–243.
20. Heidenreich WF et al. Time trends of thyroid cancer incidence in Belarus after the Chernobyl accident. *Radiation Research*, 1999, 151(5):617–625.
21. Furukawa K et al. Long-term trend of thyroid cancer risk among Japanese atomic-bomb survivors: 60 years after exposure. *International Journal of Cancer*, 201 3, 132(5):1222–1226
22. *Health effects of the Chernobyl accident and special health care programmes*. Report of the United Nations Chernobyl Forum Expert Group "Health". Geneva, World Health Organization, 2006 (http://www.who.int/ionizing_radiation/chernobyl/assessment_mitigation/en/index.html, accessed 16 November 2012).
23. Ivanov VK et al. Radiation-epidemiological studies of thyroid cancer incidence in Russia after the Chernobyl accident (estimation of radiation risks 1991–2008 follow-up period). *Radiation Protection Dosimetry*, 2012, 151(3):489–499.
24. Veiga LH et al. A pooled analysis of thyroid cancer incidence following radiotherapy for childhood cancer. *Radiation Research*, 2012, 178(4):365–376.
25. Shore RE et al. Breast cancer among women given X-ray therapy for acute postpartum mastitis. *Journal of the National Cancer Institute*, 1986, 77(3):689–696.
26. Boice JD Jr et al. Frequent chest X-ray fluoroscopy and breast cancer incidence among tuberculosis patients in Massachusetts. *Radiation Research*, 1991, 125(2):214–222.
27. Howe GR et al. Breast cancer mortality between 1950–1987 after exposure to fractionated moderate-dose-rate ionizing radiation in the Canadian fluoroscopy cohort study and a comparison with breast cancer mortality in the atomic bomb survivors study. *Radiation Research*, 1996, 145(6):694–707.
28. Mattson A et al. Radiation-induced breast cancer: long-term follow-up of radiation therapy for benign breast disease. *Journal of the National Cancer Institute*, 1993, 85(20): 1679–1685.
29. Lundell M et al. Breast cancer risk after radiotherapy in infancy: a pooled analysis of two Swedish cohorts of 17,202 infants. *Radiation Research*, 1999, 151(5):626–632.
30. Preston DL et al. Radiation effects on breast cancer risk: a pooled analysis of eight cohorts. *Radiation Research*, 2002, 158(2):220–235.
31. Hildreth NG et al. The risk of breast cancer after irradiation of the thymus in infancy. *The New England Journal of Medicine*, 1989, 321(19):1281–1284.
32. Holmberg E et al. Excess breast cancer risk and the role of parity, age at first childbirth and exposure to radiation in infancy. *British Journal of Cancer*, 2001, 85(3):362–366.
33. Haddy N et al. Breast cancer following radiotherapy for a hemangioma during childhood. *Cancer Causes Control*, 2010, 21(11):1807–1816.

34. Wakeford R, Little MP. Risk coefficients for childhood cancer after intrauterine irradiation: a review. *International Journal of Radiation Biology*, 2003, 79(5):293–309.

35. *Biological effects after prenatal irradiation (embryo and fetus).* Ottawa, International Commission on Radiological Protection, 2003 (Annals of the ICRP, 90(33):1–2).

36. Preston DL et al. Solid cancer incidence in atomic bomb survivors exposed in utero or as young children. *Journal of the National Cancer Institute*, 2008, 100(6):428–436.

37. Hancock SL et al. Thyroid diseases after treatment of Hodgkin's disease. *New England Journal of Medicine*, 1991, 325(9):599–605.

38. Huysmans DA et al. Long-term results of two schedules of radioiodine treatment for toxic multinodular goitre. *European Journal of Nuclear Medicine*, 1993, 20(11):1056–1062.

39. Peters H et al. Radioiodine therapy of Graves' hyperthyroidism: standard vs. calculated 131iodine activity. Results from a prospective, randomized, multicentre study. *European Journal of Clinical Investigation*, 1995, 25(3):186–193.

40. Metso S et al. Long-term follow-up study of radioiodine treatment of hyperthyroidism. *Clinical Endocrinology*, 2004, 61(5):641–648.

41. Ostroumova E et al. Subclinical hypothyroidism after radioiodine exposure: Ukrainian-American cohort study of thyroid cancer and other thyroid diseases after the Chornobyl accident (1998–2000). *Environmental Health Perspectives*, 2009, 117(5):745–750.

42. Larsen PR et al. Thyroid hypofunction after exposure to fallout from a hydrogen bomb explosion. *The Journal of the American Medical Association*, 1982, 247(11):1571–1575.

43. *Risk to the thyroid from ionizing radiation*. National Council on Radiation Protection and Measurements, 2008.

44. Ron E, Brenner A. Non-malignant thyroid diseases after a wide range of radiation exposures. *Radiation Research*, 2010, 174(6):877–888.

45. Hall P et al. Thyroid nodularity after diagnostic administration of iodine-131. *Radiation Research*, 1996, 146(6):673–682.

46. Land CE et al. Ultrasound-detected thyroid nodule prevalence and radiation dose from fallout. *Radiation Research*, 2008, 169(4):373–383.

47. Lyon JL et al. Thyroid disease associated with exposure to the Nevada nuclear weapons test site radiation: a reevaluation based on corrected dosimetry and examination data. *Epidemiology*, 2006, 17(6):604–614.

48. Zablotska LB et al. A cohort study of thyroid cancer and other thyroid diseases after the Chornobyl accident: dose-response analysis of thyroid follicular adenomas detected during first screening in Ukraine (1998–2000). *American Journal of Epidemiology*, 2008, 167(3):305–312.

49. Pillai NK et al. Nodular lesions of the thyroid in an area of high background radiation in Coastal Kerala, India. *Indian Journal of Medical Research*, 1976, 64(4):537–544.

50. Wang ZY et al. Thyroid nodularity and chromosome aberrations among women in areas of high background radiation in China. *Journal of the National Cancer Institute*, 1990, 82(6): 478–485.

51. Deans DS, Gharib H. Epidemiology of thyroid nodules. *Best Practice & Research: Clinical Endocrinology & Metabolism*, 2008, 22(6):901–911.

52. Imaizumi M et al. Long-term prognosis of thyroid nodule cases compared with nodule-free controls in atomic bomb survivors. *The Journal of Clinical Endocrinology and Metabolism*, 2005, 90(9):5009–5014.

53. Ahmed S et al. Prevalence of unsuspected thyroid nodules in adults on contrast enhanced 16- and 64-MDCT of the chest. *World Journal of Radiology*, 2012, 4(7):311–317.

54. Ainsbury EA et al. Radiation cataractogenesis: a review of recent studies. *Radiation Research*, 2009, 172(1):1–9.

55. Neriishi K et al. Radiation dose and cataract surgery incidence in atomic bomb survivors, 1986–2005. *Radiology*, 2012, 265(1):167–174.

56. Shimizu Y et al. Radiation exposure and circulatory disease risk: Hiroshima and Nagasaki atomic bomb survivor data, 1950–2003. *British Medical Journal*, 2010.

57. Hauptmann M et al. Mortality from diseases of the circulatory system in radiologic technologists in the United States. *American Journal of Epidemiology*, 2003, 157(3):239–248.

58. Little M et al. Analysis of dose response for circulatory disease after radiotherapy for benign disease. *International Journal of Radiation Oncology Biology Physics*, 2012, 84(5):1101-119.

59. Ivanov VK et al. The risk of radiation-induced cerebrovascular disease in Chernobyl emergency workers. *Health Physics*, 2006, 90(3):199–207.

60. Azizova TV et al. Ischemic heart disease in nuclear workers first employed at the Mayak PA in 1948–1972. *Health Physics*, 2012, 103(1):3–14.

61. Little MP et al. Systematic review and meta-analysis of circulatory disease from exposure to low-level ionizing radiation and estimates of potential population mortality risks. *Environmental Health Perspectives*, 2012, 120(11):1503–1511.

62. Azizova TV et al. Cerebrovascular diseases in nuclear workers first employed at the Mayak PA in 1948–1972. *Radiation and Environmental Biophysics*, 2011, 50(4):539–552.

63. Little MP et al. Review and meta-analysis of epidemiological associations between low/moderate doses of ionizing radiation and circulatory disease risks, and their possible mechanisms. *Radiation and Environmental Biophysics*, 2010, 49(2):139–153.

64. *ICRP Statement on Tissue Reactions/early and late effects of radiation in normal tissues and organs – threshold doses for tissue reactions in a radiation protection context*. Ottawa, International Commission on Radiological Protection, 2012 (Annals of the ICRP, 118(41):1–2).

65. *Pregnancy and medical radiation*. Ottawa, International Commission on Radiological Protection, 2000 (Annals of the ICRP, 84(30):1).

66. Hereditary Effects of Radiation. Annex in: *Hereditary Effects of Radiation*. UNSCEAR 2001 report to the General Assembly. Vienna, United Nations Scientific Committee on the effect of Atomic Radiation, 2001 (http://www.unscear.org/unscear/en/publications/2001.html, accessed 21 November 2012).

67. Nakamura N et al. Genetic effects of radiation in atomic-bomb survivors and their children: past, present and future. *Journal of Radiation Research*, 2006, 47(Suppl B):B67–B73.

68. Winther JF. Genetic disease in the children of Danish survivors of childhood and adolescent cancer. *Journal of Clinical Oncology*, 2012, 30(1):27–33.

69. Waselenko JK et al. Medical management of the acute radiation syndrome: recommendations of the Strategic National Stockpile Radiation Working Group. *Annals of Internal Medicine*, 2004, 140(12):1037–1051.

70. Preston DL et al. Solid cancer incidence in atomic bomb survivors:1958–1998. *Radiation Research*, 2007, 168(1):1–64.

71. Non-targeted and delayed effects of exposure to ionizing radiation. Annex C in: *Effects of ionizing radiation*. UNSCEAR 2006 report to the General Assembly, Vol II. Vienna, United Nations Scientific Committee on the Effects of Atomic Radiation, 2008 (http://www.unscear.org/docs/reports/2006/09-81160_Report_Annex_C_2006_Web.pdf, accessed 16 November 2012).

72. Little MP et al. Risk associated with low doses and low dose rates of ionizing radiation: why linearity may be (almost) the best we can do. *Radiology*, 2009, 251(1):6–12.

73. Little MP et al. Do non-targeted effects increase or decrease low dose risk in relation to the linear-non-threshold (LNT) model?. *Mutation Research*, 2010, 687(1–2):17–27.

74. Jacob P et al. Is cancer risk of radiation workers larger than expected? *Occupational and Environmental Medicine*, 2009, 66(12):789–796.

75. Krestinina LY et al. Solid cancer incidence and low-dose-rate radiation exposures in the Techa River cohort: 1956–2002. *International Journal of Epidemiology*, 2007, 36(5):1038–1046.

76. Brenner AV et al. I-131 dose response for incident thyroid cancers in Ukraine related to the Chornobyl accident. *Environmental Health Perspective*, 2011, 119(7):933–939.

77. Wakeford R. Cancer risk modelling and radiological protection. *Journal of Radiological Protection*, 2012, 32(1):N89–N93.

78. Thomas D et al. Definition and estimation of lifetime detriment from radiation exposures: principles and methods. *Health Physics*, 1992, 63(3):259–272.

79. Vaeth M, Pierce D. Calculating excess lifetime risk in relative risk models. *Environmental Health Perspectives*, 1990, 87:83–94.

80. Kellerer AM et al. On the conversion of solid cancer excess relative risk into lifetime attributable risk. *Radiation and Environmental Biophysics*, 2001, 40(4):249–257.

81. Kellerer AM et al. Risk coefficient for gamma-rays with regard to solid cancer. *Radiation and Environmental Biophysics*, 2002, 41(2):113–123.

82. Health effects due to radiation from the Chernobyl accident. Annex D in: *Sources and effects of ionizing radiation*. UNSCEAR 2008 report to the General Assembly, Vol II. Vienna, United Nations Scientific Committee on the Effects of Atomic Radiation, 2011 (http://www.unscear.org/docs/reports/2008/11–80076_Report_2008_Annex_D.pdf, accessed 16 November 2012).

83. Epidemiological studies of radiation and cancer. Annex A in: *Effects of ionizing radiation*. UNSCEAR 2006 report to the General Assembly, Vol I. Vienna, United Nations Scientific Committee on the Effects of Atomic Radiation, 2008 (http://www.unscear.org/docs/reports/2006/07–82087_Report_Annex_A_2006_Web_corr.pdf, accessed 16 November 2012).

84. Radiation Effects Research Foundation. [Online]. [cited 2012 October](http://www.rerf.jp/index_e.html, accessed 25 November 2012).

85. Walsh L, Kaiser JC. Multi-model inference of adult and childhood leukaemia excess relative risks based on the Japanese A-bomb survivors mortality data (1950–2000). *Radiation and Environmental Biophysics*, 2011, 50(1):21–35.

86. Little MP et al. New models for evaluation of radiation-induced lifetime cancer risk and its uncertainty employed in the UNSCEAR 2006 report. *Radiation Research*, 2008, 169(6): 660–676.

87. *Health risks from exposure to low levels of ionizing radiation: BEIR VII Phase 2*. Washington, DC, National Academy of Science – National Research Council Committee to Assess

Health Risks from Exposure to Low Levels of Ionizing Radiation, 2006 (http://www.nap.edu/openbook.php?isbn=030909156X, accessed 25 November 2012).

88. *EPA radiogenic cancer risk models and projections for the US Population*. EPA 402–R–11–001.Washington, DC, Environmental Protection Agency, 2011 (http://www.epa.gov/radiation/docs/bluebook/bbfinalversion.pdf, accessed 21 November 2012).

89. Berrington de Gonzalez A et al. RadRAT: a radiation risk assessment tool for lifetime cancer risk projection. *Journal of Radiological Protection*, 2012, 32(3):205–222.

90. Wakeford R. Radiation effects: modulating factors and risk assessment – an overview. In: *Proceedings of the First Symposium on the system of radiological protection*. Washington, DC, 2012:98–107.

91. Preston DL et al. Effect of recent changes in atomic bomb survivor dosimetry on cancer mortality risk estimates. *Radiation Research*, 2004, 162(4):377–389.

92. Daniels RD, Schubauer-Berigan MK. A meta-analysis of leukaemia risk from protracted exposure to low-dose gamma radiation. *Occupational and Environmental Medicine*, 2011, 68(6):457–464.

93. *Environmental consequences of the Chernobyl accident and their remediation: twenty years of experience*. Report of the Chernobyl Forum Expert Group "Environment-2. Vienna, International Atomic Energy Agency, 2006 (http://www–pub.iaea.org/mtcd/publications/pdf/pub1239_web.pdf, accessed 16 November 2012).

94. Menzel HG, Harrison J. Dosimetric quantities in radiological protection and risk assessment. *Journal of Radiological Protection*, 2012, 32(1):N41–N46.

95. *Derived intervention levels for radionuclides in food: guidelines for application after widespread radioactive contamination resulting from a major radiation accident*. Geneva, World Health Organization, 1988 (http://apps.who.int/iris/bitstream/10665/40421/1/9241542330_eng.pdf, accessed 16 November 2012).

96. *Application of the commission's recommendations to the protection of people living in long–term contaminated areas after a nuclear accident or a radiation emergency*. Ottawa, International Commission on Radiological Protection, 2009 (Annals of the ICRP, 111(39):3).

97. Golikov VY et al. External exposure of the population living in areas of Russia contaminated due to the Chernobyl accident. *Radiation and Environmental Biophysics*, 2002, 41(3):183–193.

98. Exposures from the Chernobyl accident. Annex D in: *Sources, effects and risks of ionizing radiation*. UNSCEAR 1988 report to the General Assembly, Vol II. Vienna, United Nations Scientific Committee on the Effects of Atomic Radiation, 1988 (http://www.unscear.org/docs/reports/1988/1988i_unscear.pdf, accessed 16 November 2012).

99. UNSCEAR 2008 report to the General Assembly, with scientific annexes. Health effects due to radiation from the Chernobyl accident, 2011.

100. *Final report of the international mission to support the remediation of large contaminated areas off-site of the Fukushima Daiichi Nuclear Power Plant (NPP)*. Document NE/NEFW/2011. Vienna, International Atomic Energy Agency, 2011 (http://www.iaea.org/newscenter/focus/fukushima/final_report151111.pdf, accessed 16 November 2012).

101. Website from the Tokyo Electric Power Company (in Japanese). [Online]. [cited November 2012 (http://www.tepco.co.jp/cc/press/betu12_j/images/120309d.pdf, accessed 02 December 2012).

102. *WHO human health risk assessment toolkit: chemical hazards*. World Health Organization, 2010 (IPCS Harmonization project). (http://www.who.int/ipcs/publications/methods/harmonization/en/index.html, accessed 28 December 2012).

103. Exposures of the public and workers from various sources of radiation. Annex B in: *Sources and effects of ionizing radiation*. UNSCEAR 2008 report to the General Assembly, Vol I. Vienna, United Nations Scientific Committee on the Effects of Atomic Radiation, 2008 (http://www.unscear.org/docs/reports/2008/09-86753_Report_2008_Annex_B.pdf, accessed 27 November 2011)

104. Matsuda T et al. Cancer incidence and incidence rates in Japan in 2004: based on data from 14 population-based cancer registries in the monitoring of cancer incidence in Japan (MCIJ) Project. *Japanese Journal of Clinical Oncology*, 2010, 40(12):1192–1200.

105. Preston DL et al. Studies of mortality of atomic bomb survivors, Report 13: solid cancer and non-cancer mortality: 1950–1997. *Radiation Research*, 2003, 160(4):381–407.

106. Richardson D et al. Ionising radiation and leukaemia mortality among Japanese atomic bomb survivors. *Radiation Research*, 2009, 172(3):368–382.

107. Jacob P et al. Thyroid Cancer Risk in Areas of Ukraine and Belarus affected by the Chernobyl Accident. *Radiation Research*, 2006, 165(1):1–8.

108. Kopecky KJ et al. Childhood thyroid cancer, radiation dose from Chernobyl, and dose uncertainties in Bryansk Oblast, Russia: a population-based case-control study. *Radiation Research*, 2006, 166(2):367–374.

109. Gilbert ES et al. Thyroid cancer rates and ^{131}I doses from Nevada atmospheric nuclear bomb tests: an update. *Radiation Research*, 2010, 173(5):659–664.

110. Gilbert ES et al. Lung cancer in Mayak workers. *Radiation Research*, 2004,162(5): 505–516.

111. Koizumi A et al. Preliminary assessment of ecological exposure of adult residents in Fukushima Prefecture to radioactive cesium through ingestion and inhalation. *Environmental Health and Preventive Medicine*, 2012, 17(4):292–298.

112. Monzen S et al. Individual radiation exposure dose due to support activities at safe shelters in Fukushima prefecture. *Public Library of Science*, 2011, 6(11):1–4.

113. Takahara et al. Assessment of doses from external exposure in contaminated areas resulting from the Fukushima Daiichi Nuclear Power Plant accident. *Progress in Nuclear Science and Technology,* 2012,3:25-29.

114. Kamada N et al. Radiation doses among residents living 37 km northwest of the Fukushima Daiichi Nuclear Power Plant. *Journal of Environmental Radioactivity*. 2012, 110:84–89.

115. Tsubokura M et al. Internal radiation exposure after the Fukushima nuclear power plant disaster. *Journal of the American Medical Association*, 2012, 308(7):669–670.

116. Anzai K et al. The Fukushima Nuclear Power Plant accident and exposures in the environment. *The Environmentalist*, 2012, 32(2):136–143.

117. Website of the Fukushima Prefecture (http://wwwcms.pref.fukushima.jp/pcp_portal/PortalServlet?DISPLAY_ID=U000004&CONTENTS_ID–24809, accessed 21 November 2012).

118. Nagataki S.Thyroid Consequences of the Fukushima Nuclear Reactor Accident. *Eur Thyroid J*, 2012,1:148–158.

119. Walsh L and Schneider U. A method for determining weights for excess relative risk and excess absolute risk when applied in the calculation of lifetime risk of cancer from radiation exposure. *Radiation and Environmental Biophysics*, 2012.

120. Bhatti P et al. Risk of second primary thyroid cancer after radiotherapy for a childhood cancer in a large cohort study: an update from the childhood cancer survivor study. *Radiation Research*, 2010,174(6):741–752.

121. *Radiation protection and safety of radiation sources: international basic safety standards (BSS).* IAEA safety standards series, Interim edition. Vienna, International Atomic Energy Agency, 2011 (http://www–pub.iaea.org/MTCD/publications/PDF/p1531interim_web.pdf, accessed 16 November 2012).

122. Doll R, Wakeford R. Risk of childhood cancer from fetal irradiation. *British Journal of Radiology*, 1997, 70:130–139.

123. Boice JD Jr, Miller RW. Childhood and adult cancer after intrauterine exposure to ionizing radiation. *Teratology*, 1999, 59(4):227–233.

124. Hatch M et al. A screening study of thyroid cancer and other thyroid diseases among individuals exposed *in utero* to iodine–131 from Chernobyl fallout. *The Journal of Clinical Endocrinology and Metabolism*, 2009, 94(3):889–906.

125. Imaizumi M et al. Thyroid diseases in atomic bomb survivors exposed *in utero*. *The Journal of Clinical Endocrinology and Metabolism*, 2008, 93 (5):1641–1648.

126. Shakhtarin VV et al. Iodine deficiency, radiation dose, and the risk of thyroid cancer among children and adolescents in the Bryansk region of Russia following the Chernobyl power plant accident. *International Journal of Epidemiology*, 2003, 32(4):584–591.

127. Zablotska LB et al. Thyroid cancer risk in Belarus among children and adolescents exposed to radioiodine after the Chornobyl accident. *British Journal of Cancer*, 2011,104(1):181–187.

128. Zava TT and Zava DT. Assessment of Japanese iodine intake based on seaweed consumption in Japan: A literature-based analysis. *Thyroid Research*, 2011, 4:14 (http://www.thyroidresearchjournal.com/content/4/1/14, accessed 21 November 2012).

129. Suzuki M, Tamura T. Iodine intake of Japanese male university students: urinary iodine excretion of sedentary and physically active students and sweat iodine excretion during exercise. *Journal of Nutritional Science and Vitaminology*, 1985, 31(4):409–415.

130. Miyakawa T et al. Seaweed consumption and the risk of thyroid cancer in women: the Japan Public Health Center-based prospective study. *European Journal of Cancer Prevention*, 2012, 21(3):254–260.

131. Nauman J et al. Iodide prophylaxis in Poland after the Chernobyl reactor accident: benefits and risks. *The American Journal of Medicine*,1993, 94(5):524–532.

132. Spallek L et al. Adverse effects of iodine thyroid blocking: a systematic review. *Radiation Protection Dosimetry*, 2011, 150(3):267–277.

133. Coleman CN et al. Commentary on the combined disaster in Japan. *Radiation Research*, 2012, 177(1):15–17.

134. *The Great East Japan Earthquake. A story of a devastating natural disaster, a tale of human compassion*. Manila, World Health Organization Regional Office for the Western Pacific, 2012 (http://www.wpro.who.int/publications/9789290615682/en/index.html, accessed 16 November 2012).

135. *Constitution of the World Health Organization*. Geneva, World Health Organization, (http://www.who.int/governance/eb/constitution/en/, accessed 21 November 2012).

136. Carr Z, Li C. Proceedings of the 13th coordination and planning meeting of the WHO-REMPAN: radiation emergency medical preparedness and assistance network, Nagazaki, Japan, 16–18 February 2011. *Radiation Protection Dosimetry*, 2012, 151(4):605–606.

137. *Requirements GS–R2 Preparedness and Response for a Nuclear or Radiological Emergency*. Jointly sponsored by FAO, IAEA, ILO, OECD/NEA, PAHO, OCHA, WHO. Vienna, International Atomic Energy Agency, 2002.

138. Aumonier S and Morrey M. Non-radiological risks of evacuation. *Journal of Radiological Protection*, 1990,10(4):287–290.

139. *Application of the commission's recommendations for the protection of people in emergency exposure situations*. Ottawa, International Commission on Radiological Protection, 2009 (Annals of the ICRP, 109(39):1).

140. *Notice No. 0315 Articles 1 of the Department of Food Safety*. Japan, Website of the Ministry of Health, Labour and Welfare, 2012 (http://www.mhlw.go.jp/english/topics/2011eq/index.html, accessed 21 November 2012).

141. Rojas-Palma C et al. Triage, monitoring and treatment of people exposed to ionizing radiation following a malevolent act. *TMT Handbook*, 2009 (http://www.tmthandbook.org, accessed 21 November 2012).

142. Jacob P et al. Thyroid cancer among Ukrainians and Belarusians who were children or adolescents at the time of the Chernobyl accident. *Journal of Radiological Protection*, 2006, 26(1):51–67.

143. Likhtarov I et al. Post–Chernobyl thyroid cancers in Ukraine, Report 2: Risk analysis. *Radiation Research*, 2006, 166(2):375–386.

144. *Sources and effects of ionizing radiation*. UNSCEAR 1993 report to the General Assembly. Vienna, United Nations Scientific Committee on the effect of Atomic Radiation, 1993 (http://www.unscear.org/unscear/en/publications/1993.html, accessed 21 November 2012)

145. Pérez M, Lau L, WHO workshop on radiation risk assessment in paediatric health care: summary and recommendations. *Journal of Radiological Protection*, 2010, 30(1):105.

146. Pearce et al. Radiation exposure from CT scans in childhood and subsequent risk of leukaemia and brain tumours: a retrospective cohort study. *Lancet*, 2012, 380(9840):499–505.

147. Yasumura S et al. Study protocol for the Fukushima health management survey. *Journal of Epidemiology*, 2012, 22(5):375–383.

148. Survey Fukushima Medical University 2012(http://frh.fukushima-mimamori.jp/results/20120911.html, accessed 21 November 2012).

149. *Chernobyl's legacy: health, environmental and socio-economic impacts: Chernobyl Forum*. Vienna, International Atomic Energy Agency, 2006.

150. Van Deventer E et al. WHO's public health agenda in response to the Fukushima Daiichi nuclear accident. *The Journal of Radiological Protection*, 2012, 32(1):N119–N22 (http://iopscience.iop.org/0952–4746/32/1/N119/, accessed 20 November 2012).

151. Berger ME, Sadoff RL. Psychological support of radiation accidents: patients, families and staff. The medical basis for radiation accident preparedness: the clinical care of victims. New York, *Parthenon Publishers*, 2002.

152. Bromet EJ et al. Long-term mental health consequences of the accident at Three Mile Island. *International Journal of Mental Health*, 1990, 19(2):48–60.

153. Loganovsky K et al. The mental health of clean-up workers 18 years after the Chernobyl accident. *Psychological Medicine*, 2008, 38(4):481–488.

154. Bromet EJ, Havenaar JM. Psychological and perceived health effects of the Chernobyl disaster: a 20-year review. *Health Physics*, 2007, 93(5):516–521.

155. Helou S, Costa Neto SB, Césio-137, consequências psicossociais do acidente de Goiânia [*Psychosocial consequences of the Goiania accident*]. Brazil, Federal University of Goiás,1995.

156. *Number of evacuees in the country*. Japan, Reconstruction Agency, Japanese Government, 2012. (http://www.reconstruction.go.jp/topics/120912_hinansya.pdf).

157. *Generic procedures for medical response during a nuclear or radiological emergency*. EPR–MEDICAL 2005. Vienna, International Atomic Energy Agency, 2005 (http://www–pub.

iaea.org/MTCD/publications/PDF/EPR–MEDICAL–2005_web.pdf, accessed 16 November 2012).

158. *Mental health in emergencies. Mental and social aspects of health of populations exposed to extreme stressors*. Document WHO/MSD/MER/03.01. Geneva, World Health Organization, 2003 (http://www.who.int/mental_health/media/en/640.pdf, accessed 16 November 2012).

159. Bromet E. Lessons learned from radiation disasters. *World Psychiatry*, 2011, 10(2):83–84.

160. Mole RH. Childhood cancer after prenatal exposure to diagnostic x-ray examinations in Britain. *British Journal of Cancer*, 1990, 62(1):152–168.

161. Harrison JD, Muirhead CR. Quantitative comparisons of cancer induction in humans by internally deposited radionuclides and external radiation. *International Journal of Radiation Biology*, 2003, 79(1):1–13.

162. Ron E. Cancer risks from medical radiation. *Health Physics*, 2003, 85(1):47–59.

163. Berrington A et al. 100 years of observations on British radiologists. *British Journal of Radiology*, 2001, 74(882):507–519.

164. Shilnikova NS et al. Cancer mortality risk among workers at the Mayak nuclear complex. *Radiation Research*, 2003, 159(6):787–798.

165. Cardis E et al. Risk of cancer after low doses of ionizing radiation: retrospective cohort study in 15 countries. *BMJ*, 2005, 331(7508):77–80.

166. Akiba S, Mizuno S. The third analysis of cancer mortality among Japanese nuclear workers, 1991–2002: estimation of excess relative risk per radiation dose. *Journal of Radiological Protection*, 2012, 32(1):73–83.

167. Bauer S et al. Radiation exposure due to local fallout from Soviet atmospheric nuclear weapons testing in Kazakhstan: solid cancer mortality in the Semipalatinsk historical cohort, 1960–1999. *Radiation Research*, 2005, 164(4):409–419.

168. Nair RR et al. Background radiation and cancer incidence in Kerala, India-Karanagappally cohort study. *Health Physics*, 2009, 96(1):55–66.

169. Tao Z et al. Cancer mortality in the high background radiation areas of Yangjiang, China during the period between 1979 and 1995. *Radiation Research*, 2000, (Suppl. 41):31–41.

170. Akiba S et al. Child cancer risk in high-background radiation areas. *International Congress Series*, 2002, 1225:283–287.

171. Ghiassi-Nejad M et al. Very high background radiation areas of Ramsar, Iran: preliminary biological studies. *Health Physics*, 2002, 82(1):87–93.

172. Hendry JH et al. Human exposure to high natural background radiation: what can it teach us about radiation risks? *Journal of Radiological Protection*, 2009, 29(2A):A29–A42.

173. Wakeford R et al. The proportion of childhood leukaemia incidence in Great Britain that may be caused by natural background ionizing radiation. *Leukemia*, 2009, 23(4):770–776.

174. Kendall GM. A record-based case-control study of natural background radiation and the incidence of childhood leukaemia and other cancers in Great Britain during 1980–2006. *Leukemia*, 2013, 27(1):3–9.

175. *Portal site of official statistics of Japan*. Japan, 2012 (http://www.e-stat.go.jp/SG1/estat/ListE.do?lid=000001082327, accessed 25 November 2012).

176. Iwasaki T et al. Second analysis of mortality of nuclear industry workers in Japan, 1986–1997. *Radiation Research*, 2003, 159(2):228–238.

177. Cardis E et al. Risk of thyroid cancer after exposure to ^{131}I in childhood. *Journal of the National Cancer Institute*, 2005, 97:724–732.

178. Walsh L et al. Radiation risk modeling of the thyroid cancer with special emphasis on the Chernobyl epidemiological data. *Radiation Research*, 2009, 172(4):509–518.

179. Heiervang KS et al. Effect of low dose ionizing radiation exposure in utero on cognitive function in adolescence. *Scandinavian Journal of Psychology*, 2010, 51(3):210–215.

180. Igumnov S, Drozdovitch V. The intellectual development, mental and behavioural disorders in children from Belarus exposed in utero following the Chernobyl accident. *European Psychiatry*, 2000, 15(4):244–253.

181. Igumnov SA et al. Intellectual development of the persons exposed in utero: 10-years follow-up investigation. *Meditsinskaya Radiologiva*, 2004, 49(4):29–35.

182. Kolominsky YS et al. The psychological development of children from Belarus exposed in the prenatal period to radiation from the Chernobyl atomic power plant. *Journal of Child Pyschology and Psychiatry and Allied Disciplines*, 1999, 40(2):299–305.

183. Kozlova IA et al. Psychological and psychiatric study of children living in Kaluga and Bryansk regions of Russia (aftermaths of the Chernobyl catastrophe). *Zhurnal Nevropatologii i Psikhiatrii Imeni S.S.Korsakova*, 1999, 95(1):70–74.

184. Loganovsky KN et al. Disrupted development of the dominant hemisphere following prenatal irradiation. *Journal of Neuropsychiatry and Clinical Neurosciences*. 2008, 20(3):274–291.

185. Nyagu AI et al. Psychophysiological after effects of prenatal irradiation. *International Journal of Psychophysiology*, 1998, 30(3):303–311.

186. Hall P et al. Effect of low doses of ionising radiation in infancy on cognitive function in adulthood: Swedish population based cohort study. *BMJ*, 2004, 328(7430):19.

187. Ron E et al. Mental function following scalp irradiation during childhood. *American Journal of Epidemiology*. 1982, 116(1):149–160.

188. Cousens Pet al. Cognitive effects of cranial irradiation in leukaemia: A survey and meta-analysis. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 1998, 29(6):839–852.

189. Fuss M et al. Full Scale IQ (FSIQ) changes in children treated with whole brain and partial brain irradiation: A review and analysis. *Strahlenther Onkol*, 2000, 176(12):573–581.

190. Grill J et al. Long-term intellectual outcome in children with posterior fossa tumors according to radiation doses and volumes. *International Journal of Radiation Oncology Biology Physics*, 1999,45(1):137–145.

191. Kramer JH et al. A prospective study of cognitive functioning following low-dose cranial radiation for bone marrow transplantation. *Pediatrics*, 1992, 90(31):447–450.

192. Meadows AT et al. Declines in IQ scores and cognitive dysfunctions in children with acute lymphocytic leukaemia treated with cranial irradiation. *Lancet*, 1981, 2(8254):1015–1018.

193. Vaño E et al. Radiation cataract risk in interventional cardiology personnel. *Radiation Research*, 2010, 174(4):490–495.

194. Ciraj-Bjelac O et al. Risk for radiation–induced cataract for staff in interventional cardiology: is there reason for concern?. *Catheter Cardiovasc Interventions*, 2010,76(6):826–834.

195. Mrena S et al. Lens opacities among physicians occupationally exposed to ionizing radiation: a pilot study in Finland. *Scandinavian Journal of Work, Environment & Health*, 2011,37(3):237–243.

196. Jacob S et al. Interventional cardiologists and risk of radiation-induced cataract: results of a French multicenter observational study. *International Journal of Cardiology*, 2012.

197. Worgul BV et al. Cataracts among Chernobyl clean-up workers: implications regarding permissible eye exposures. *Radiation Research*, 2007,167(2):233–243.

198. Hsieh WA et al. Lens opacities in young individuals long after exposure to protracted low-dose rate gamma radiation in 60 co-contaminated buildings in Taiwan. *Radiation Research*, 2010, 173(2):197–204.

199. Darby SC et al. Radiation-related heart disease: current knowledge and future prospects. *International Journal of Radiation Oncology*, *Biology, Physics*, 2010, 76(3):656–665.

200. McGale P, Darby SC. Low doses of ionizing radiation and circulatory diseases: a systematic review of the published epidemiological evidence. *Radiation Research*, 2005, 163(3):247–257.

201. Azizova TV et al. Ischemic heart diseases in nuclear workers first employed at the Mayak PA in 1948–1972. *Health Physics*, 2012, 103(1):3–14.

202. McGeoghegan D et al. The non-cancer mortality experience of male workers at British Nuclear Fuels plc, 1946–2005. *International Journal of Epidemiology*, 2008, 37(3):506–518.

203. Vrijheid M et al. Mortality from diseases other than cancer following low doses of ionizing radiation: results from the 15-Country Study of nuclear industry workers. *International Journal of Epidemiology*, 2007, 36(5):1126–1135.

204. Muirhead CR et al. Mortality and cancer incidence following occupational radiation exposure: third analysis of the national registry for radiation workers. *British Journal of Cancer*, 2009; 100(1):206–212.

205. Kreuzer VK et al. Mortality from cardiovascular diseases in the German uranium miners cohort study, 1946–1998. *Radiation and Environmental Biophysics*, 2006, 45(3):159–166.

206. Jacob P et al. Calculation of organ doses from environmental gamma rays using human phantoms and Monte-Carlo methods: part 2: radionuclides distributed in air or deposited on the ground. *Munchen–Neuherberg: GSF–Forschungszentrum*; 1990.

207. *The ICRP database of dose coefficients: workers and members of the public*. Ottawa, International Commission on Radiological Protection, 2001 (http://www.icrp.org/page.asp?id=145, accessed 21 January 2013).

208. UNSCEAR 2000 report to the General Assembly. Sources and Effects of Ionizing Radiation., Vol II Scientific Annex J: Exposures and effects of the Chernobyl accident, 2000.

Glossary

Absorbed dose
Mean energy imparted by ionizing radiation to an irradiated medium per unit mass, expressed in grays (Gy). 1 Gy = 1 J/kg

Activity
See radioactivity.

Acute effects
Adverse effects that occur within a short period of time (minutes to a few days) after an exposure.

Acute exposure
An exposure occurring within a short time relative to the life of a person or organism, usually consisting of a single exposure or dose administered for a period of 24 hours or less in humans.

Acute radiation syndrome
A set of characteristic signs and symptoms observed after whole-body or large-volume partial-body high-dose radiation exposure.

Age-at-exposure
Age of an individual when the radiation exposure takes place. Cancer risk models based on human epidemiological data predict higher lifetime risks for exposure at younger ages than at older ages.

Atmospheric dispersion
The spreading of radionuclides in air, resulting mainly from physical processes affecting the velocity of different molecules.

Attained age
Age of a person calculated by adding the period elapsed since the radiation exposure took place (i.e. the "time since exposure") and the age of that person when the radiation exposure took place (i.e. the "age-at-exposure").

Becquerel
In the International System, a unit of activity equal to one disintegration per second.

Cancer
A group of related diseases characterized by the uncontrolled growth of abnormal cells.

Cancer risk estimate
The probability of developing cancer from exposure to radiation over a period of time.

Carcinogen
A physical, chemical or biological agent capable of inducing cancer.

Chronic effects
Adverse effects that occur within a long period of time after an exposure (years to lifetime).

Cloudshine
Gamma radiation from radionuclides in an airborne radioactive plume (i.e. radioactive cloud).

Cohort
A defined population group followed prospectively in an epidemiological study. Cohorts can also be used for retrospective epidemiological studies, also called historical cohort studies.

Committed dose
The lifetime dose expected to result from a radionuclide intake.

Conservative
An approach that deliberately chooses an option (e.g. an assumption) that is more likely to overestimate than to underestimate the risk.

Cumulative risk
Cumulative incidence/mortality risk is the probability of individuals getting/dying from the disease during a specified period.

Deterministic effects
Health effects, the severity of which varies with dose; typically, there is a threshold below which they will not occur (e.g. acute radiation syndrome). Deterministic effects are also referred to as “tissue reactions” or non-stochastic effects.

Dose
A general term denoting the quantity of radiation or energy absorbed in a target. Related terms:absorbed dose, effective dose, committed dose.

Dose assessment
Assessment of the dose(s) to an individual or group of people.

Dose coefficients
Factors used to convert the amount of incorporated radioactive substances (radionuclide intake) to the dose in tissues or organs, or the whole-body dose. These factors (also called "dose conversion factors") may depend on the radionuclide, the incorporation route (e.g. inhalation, ingestion), the chemical compound and the age of the person. Usually expressed as dose per unit intake, e.g. sieverts per becquerel (Sv/Bq).

Dose limit
In planned exposure situations, the value of the individual effective dose or equivalent dose that is not to be exceeded. Dose limits do not apply to existing exposure situations or emergency exposure situations.

Dose rate
Dose delivered per unit time.

Dose-response assessment:
Assessment of the relationship between exposure to a particular agent and any adverse health effects in humans as a result of this exposure.

Dose-response relationship
Relationship between the magnitude of a dose and the biological response in an organism, system or (sub)population. Related term: dose-effect relationship.

Effective dose
Sum of the products of absorbed dose to each organ multiplied by a radiation-weighting factor and a tissue-weighting factor that takes into account the radiosensitivity of tissues and organs. Related term: absorbed dose.

Effective half-life (see also half-life)
The time taken for the activity of a radionuclide in the body to halve as a result of all relevant processes (e.g. radioactive decay, biological half-life). The physical half-life is the time required for the activity of a specified radionuclide to decrease, through a radioactive decay process, by half. The biological half-life is the time taken for the quantity of a radioactive material in a specified tissue, organ or region of the body to halve as a result of biological processes.

Emergency worker
A person having specified duties as a worker in response to an emergency.

End-points
In the context of this report, end-points refer to the occurrence of a disease or adverse effect (cancer and non-cancer effects).

Environmental monitoring
The measurement of external dose rates due to sources in the environment or of radionuclide concentrations in environmental media.

Equivalent dose
Absorbed dose averaged over a tissue or organ, further applying a radiation-weighting factor that varies by radiation type and is related to the density of ionization created.

Excess absolute risk (EAR)
Difference in the rate of occurrence of disease between an exposed population and a comparable non-exposed population. It represents the additional risk beyond the baseline risk in the absence of exposure.

Excess relative risk (ERR)
Ratio of the rate of occurrence of disease in an exposed population to that in a comparable non-exposed population. It represents the proportional increase in risk in comparison with the baseline risk in the absence of exposure.

Exposure
The state or condition of being subjected to irradiation from a source outside the body (i.e. external exposure) or within the body (i.e. internal exposure).

Exposure assessment
Evaluation of the exposure of an organism, system or (sub)population to an agent. In the context of this report it refers to radiation exposure. Exposure assessment is one of the steps in the process of risk assessment.

Exposure pathway
A route by which radiation or radionuclides can reach humans and cause exposure. Related term: exposure route.

External exposure (see exposure)

Groundshine
Gamma radiation from radionuclides deposited on the ground.

Half-life (see also effective half-life)
The time taken for the quantity of a specified material (e.g. a radionuclide) in a specified place to decrease by half as a result of any process or processes that follow similar exponential patterns as those of radioactive decay (see also effective half-life).

Hazard identification
Hazard identification is the identification of the type and nature of adverse effects that an agent has, an inherent capacity to cause harm in an organism, system or (sub)-population. Hazard identification is the first step in the process of risk assessment.

Healthy worker effect
The healthy worker effect (HWE) is a bias found in occupational studies when rates of disease among employed people are compared with disease rates for the general population.

Intake
The activity of a radionuclide taken into the body (by inhalation or ingestion or through the skin) in a given time period or as a result of a given event.

Internal exposure (see exposure**)**

Ionizing radiation
For the purposes of radiation protection, radiation capable of producing ion pairs in biological material(s).

Latency
The time between exposure to a potential hazard (e.g. radiation exposure) and the appearance of a related health effect.

Life Span Study (LSS)
A research program investigating life-long health effects based on epidemiologic studies on atomic bomb survivors. Its major objective is to investigate the long-term effects of atomic bomb radiation on causes of death and incidence of cancer. About 120 000 subjects selected from residents of Hiroshima and Nagasaki identified through the national census in 1950 have been followed since that time, including 94 000 atomic bomb survivors and 27 000 unexposed individuals.

Linear no-threshold (LNT) model
Risk model that assumes that health effects are directly proportional to the dose at all dose levels (i.e. linear dose-response), without any threshold below which such effects are not expected.

Lifetime attributable risk (LAR)
Probability of a premature incidence of a cancer attributable to radiation exposure in a representative member of the population.

Lifetime baseline risk (LBR)
The probability of having a specific disease over the lifetime, in the absence of radiation exposure.

Lifetime fractional risk (LFR)
Fractional increase over the lifetime baseline risk attributable to radiation exposure.

Lifetime dose
Radiation dose resulting from exposure over the entire life.

Modelling (risk modelling)
Quantitative relationships established by using mathematical functions to calculate the magnitude of risks associated with an estimated exposure.

Natural background radiation
Amount of radiation to which a population is exposed from natural sources, such as terrestrial radiation resulting from naturally occurring radionuclides in the soil, cosmic radiation originating in outer space, and naturally occurring radionuclides deposited in the human body.

Noble gas
An inert radioactive gas that does not readily enter into chemical combination with other elements. Examples are helium, argon, krypton, xenon and radon.

Organ dose
The mean absorbed dose in a specified tissue or organ of the human body. Sometimes called tissue dose.

Radioactivity (also called "activity")
The property of the nucleus of unstable atoms that causes them to spontaneously release energy in the form of photons (e.g. gamma rays) or subatomic particles (e.g. alpha or beta particles). The amount of radioactivity is defined as the mean number of decays per unit time. The unit of activity in the International System (SI) is the reciprocal second (s^{-1}), termed the becquerel (Bq).

Radionuclide
Radioactive species of an atom characterized by the constitution of its nucleus.

Remedial action (see remediation)

Remediation
Any measures carried out to reduce radiation exposure, from existing contamination of land areas through actions applied to the contamination itself (the source) or to the exposure pathways to humans.

Risk
Hazard, danger or chance of harmful consequences associated with exposures or potential exposures.

Risk assessment
The cumulative combination and results from the scientific method of evaluating the toxic properties of a given agent and how humans and the ecosystem are exposed. A risk assessment generally determines the likelihood, to what extent, and/or characterizes how humans and/or the ecosystem are adversely affected.

Risk characterization
The last phase of risk assessment, in which all information from toxicity and exposure are combined to calculate risk estimates. This will include all the assumptions and

scientific information used to estimate risk, the uncertainty associated with the assessment, and any other information that may be useful to decision makers.

Risk model
Mathematical function that allows calculation of the magnitude of risks associated with a given exposure.

Sievert
The SI unit of equivalent dose and effective dose, equal to 1 J/kg.

Solid cancers
Cancers originating in solid organs, as opposed to blood cancers such as leukaemia.

Source
Anything that may cause radiation exposure through emission of ionizing radiation or release of radioactive substances or material, and that can be treated as a single entity for protection and safety purposes.

Source term
The amount and isotopic composition of material released (or postulated to be released) from a facility.

Stochastic effect
Adverse effects of ionizing radiation due to transformation of a single cell, that may result in an increased risk of disease a long time after exposure. These effects are probabilistic and include cancer and heritable effects. At low doses, radiation risks are primarily stochastic effects, in particular, cancer.

Survival curve
Mathematical functions representing the probability of being alive at a given age (also called “survival functions”).

Teratogenic (see teratogens)

Teratogens
Agents that can disrupt prenatal development when the mother is exposed during pregnancy.

Threshold (or "threshold dose")
Minimal absorbed radiation dose that will produce a detectable degree of any given effect.

Tissue reactions (see deterministic effects).

Abbreviations

ABCC	Atomic Bomb Casualty Commission
AHS	Adult Health Study
ALL	acute lymphoblastic leukaemia
AML	acute myeloid leukaemia
AR	absolute risk
BEIR	Biological Effects of Ionizing Radiation
BfS	Bundesamt für Strahlenschutz (Federal Office of Radiation Protection), Germany
BSS	Basic Safety Standards
CLL	chronic lymphocytic leukaemia
CML	chronic myeloid leukaemia
Cs	caesium
DDREF	Dose and dose rate effectiveness factor
DNA	deoxyribonucleic acid
DS02	2002 dosimetry system (DS02) for the cohort of the atomic bomb survivors of Hiroshima and Nagasaki
EAR	Excess absolute risk
ERR	Excess relative risk
Gy	gray
HPA	Health Protection Agency, United Kingdom
HRA	Health risk assessment
I	iodine
IAEA	International Atomic Energy Agency
IARC	International Agency for Research on Cancer
ICD	International Classification of Diseases
ICRP	International Commission on Radiological Protection
ILO	International Labour Organization
INFOSAN	International Food Safety Authorities Network
IRSN	Institut de Radioprotection et Sureté Nucléaire
JAEA	Japan Atomic Energy Agency
LAR	Lifetime attributable risk
LBR	Lifetime baseline risk
LFR	Lifetime fractional risk
LNT	Linear no threshold
LSS	Life Span Study
^{m}Ba	metastable barium
mSv	millisievert
NIRS	National Institute of Radiological Sciences, Japan
NPP	nuclear power plant
REID	risk of exposure-induced death
RERF	Radiation Effects Research Foundation
RR	relative risk
Sv	sievert
Te	tellurium
TEPCO	Tokyo Electric Power Company
UNSCEAR	United Nations Scientific Committee on the Effects of Atomic Radiation
WHO	World Health Organization
Xe	xenon

Annex A. Profiles of the HRA Expert Group members

Dr Makoto Akashi, Chiba, Japan

Dr Makoto Akashi is the Executive Director of the National Institute of Radiological Sciences (NIRS). He was awarded his M.D. degree from Yamagata University School of Medicine and started his medical career as a junior resident of internal medicine in 1981. In 1988 he received a Ph.D. from the Graduate School of Medicine, Jichi Medical School, where he also did his residencies in Internal Medicine and Hematology. He has been a research fellow at the Division of Hematology/Oncology at the University of California at Los Angeles (UCLA) School of Medicine. He has been working at the National Institute of Radiological Sciences (NIRS) in Chiba Japan since 1990. His major interests are: 1) Research on radiation injuries, including molecular and cellular mechanisms, 2) Development of methods for mitigation of radiation injuries, and 3) Biochemistry. His group performed the dose estimation of patients of the Tokaimura criticality accident and their medical treatment. He led the efforts from the NIRS for the establishment of the Radiation Emergency Medical Assistance Team (REMAT) program, aiming to support primary medical care after accidental radiation exposures either inside or outside Japan. He is now playing a leading role in providing advice and support as a radiation emergency medicine expert for the Fukushima Daiichi Nuclear Power Plant accident caused by the Great East Japan Earthquake in 2011.

Dr Billy Amzal, Paris, France

Dr Billy Amzal holds a Maths Engineering Degree from Ecole Polytechnique ("X-Ponts"), a Masters of Public Administration from AgroParisTech and a Ph.D. in Decision Mathematics from Paris-Dauphine University which was awarded by the International Society of Bayesian Analysis and by the International Biometrics Society. Over the last 13 years, he has developed quantitative methodologies to inform and support strategic decision making in healthcare as a national civil servant. He led the model-based drug development function at Novartis Pharma. He then joined the European Food Safety Authority (EFSA) developing the quantitative assessment methodologies for all EFSA Panels. He was also Director of the Data Center at the NIH-sponsored Program for HIV Prevention and Treatment in Thailand. He is now Senior Scientific Vice President at Analytica LASER, an independent scientific consulting and analytical group, and acts as a modelling expert for various Public Health Authorities such as ANSES in France.

Professor Lynn Anspaugh, Salt Lake City, United States of America

Dr Lynn Richard Anspaugh is a Research Professor in the Radiobiology Division of the Department of Radiology at the University of Utah. Before assuming his current position in January 1997 Dr Anspaugh had worked 33 years at Lawrence Livermore National Laboratory (LLNL) in a number of positions, including 10 years as Leader of the Environmental Sciences Division. Dr Anspaugh has been involved in dose-reconstruction studies for persons exposed to fallout from nuclear weapons tests, workers and the general public exposed as a result of the Chernobyl accident, and members of the public exposed

from releases from the Mayak Production Association in Russia. Dr Anspaugh was an elected member of the U.S. National Council on Radiation Protection and Measurements (NCRP), and he is now a NCRP Distinguished Emeritus Member. He is a Fellow of the Health Physics Society, and a 25-year member of the U.S. Delegation to the UNSCEAR. He is the author or co-author of 350 papers and reports, most of which are related to radiation-dose reconstruction activities.

Professor Anssi Auvinen, Tampere, Finland

Dr Anssi Auvinen has a professional background in medicine, with a Ph.D. in epidemiology. He is a professor of epidemiology at the University of Tampere and a part-time research professor at the Finnish Radiation and Nuclear Safety Authority (STUK). He has previously been employed at the Radiation Epidemiology Branch at U.S. National Cancer Institute (NCI) the Finnish Cancer Institute and the Section on Environment and Radiation at the International Agency for Research on Cancer (IARC). He has worked in radiation epidemiology since 1989 and published extensively on the health effects of both ionizing and non-ionizing radiation including cancer risk and other end-points (roughly 90 journal articles on radiation effects). His research contributions have focused on health effects of indoor radon, Chernobyl fallout, occupational radiation exposure and medical uses of radiation. He has participated in the international collaborative studies of nuclear workers, airline personnel, indoor radon and Chernobyl cleanup workers. He has previously worked as an invited expert for the WHO on Chernobyl and radon, as well as for the European Commission (EC DG SANCO) on body scanners.

Dr Nick Gent, London, United Kingdom

Dr Nick Gent was initially trained in medicine at Liverpool University, UK, before specializing in public health and health protection. Dr Gent obtained an M.Sc. in Public Health from the University of Newcastle upon Tyne, UK, and a LLM degree in Environmental Law from the University of Central Lancashire, UK. He is a fellow of the Faculty of Public Health of the Royal College of Physicians of the United Kingdom.

He is a senior medical specialist at the UK Health Protection Agency (HPA) at Porton Down, where he is Deputy Head of the Emergency Response Department, specializing in the scientific and clinical response to the release of chemical, biological, radiological or nuclear materials. His work involves close liaison and collaboration with scientific staff at the HPA Centre for Radiation, Chemical and Environmental Hazards.

In 2009 he was appointed to the WHO roster of experts under Article 47 of the International Health Regulations in the area of public health response to radiation emergencies, and has served as a consultant or expert on a number of WHO and IAEA working groups including the WHO consultations on management of acute radiation syndrome and multi-organ failure (ARS/MOF), the development of the Triage Monitoring and Treatment (TMT) handbook on radiological injuries and the IAEA/WHO EPR-MEDICAL 2005 emergency preparedness and response manual.

Dr Peter Jacob, Munich, Germany

Dr Peter Jacob completed his Ph.D. in mathematical physics at the Technical University of Munich. He is acting Director of the Institute of Radiation Protection at the Helmholtz Zentrum München. His main research interests include the modelling of late health effects after exposure to ionizing radiation with a focus on cancer and cardiovascular disease. Dr Jacob coordinates the European project combining epidemiology and radio-

biology to assess cancer risk in the breast, lung, thyroid and digestive tract after exposure to ionizing radiation with total doses on the order of 100 mSv or below (EpiRadBio). He is the head of the collaborative project 'Personalised assessment of late health effects of radiation exposure and decision support for radiation application in medicine (PASSOS)'. Dr Jacob is member of the German Commission on Radiological Protection and Head of the Radiation Risk Committee. He is member of the German delegation at the UNSCEAR.

Dr Dominique Laurier, Fontenay-aux-Roses, France

Dr Dominique Laurier is the Head of the Laboratory of Epidemiology at the Institute for Radiological Protection and Nuclear Safety (IRSN, France). He holds a Ph.D. in Biomathematics, and received the Accreditation to Supervise Research in Epidemiology from the University Denis Diderot Paris VII (France). He joined the IRSN in 1995. His research focuses on the quantification of risks associated with ionizing radiation at low doses and low dose rates. Dr Laurier is the author or co-author of more than 80 articles in peer-reviewed scientific journals. He has been involved in several European collaborative research projects, and he has contributed to different expert groups or scientific committees at the national or international level, in the fields of public health and radiation protection.

Dr Charles Miller, Atlanta, United States of America

Dr Charles Miller joined the Centers for Disease Control and Prevention (CDC) in January 1992. He is currently chief of the Radiation Studies Branch, Division of Environmental Hazards and Health Effects, National Center for Environmental Health. In this position he provides leadership for the agency's radiological emergency response and consequence management efforts. Previously, Dr Miller worked with the Illinois Department of Nuclear Safety, Oak Ridge National Laboratory, and Anderson (Indiana) University. His primary area of expertise is the transport and dose assessment of radionuclides released to the atmosphere, and other facets of environmental radiological dose assessment. He has authored or coauthored over 100 journal articles, laboratory reports, and meeting papers. Dr Miller is a member of the NCRP and a Fellow of the Health Physics Society. Dr Miller holds a B.S. in Physics/Math from Ball State University, a M.S. in Meteorology from the University of Michigan, and a Ph.D. in Bionucleonics (Health Physics) from Purdue University.

Professor Ohtsura Niwa, Fukushima, Japan

Dr Ohtsura Niwa was trained in radiation biology for his early graduate study at Kyoto University. After obtaining his Ph.D. at Stanford University in 1975, he mainly studied the molecular mechanisms of untargeted mutagenesis and its implications in somatic and heritable effects of radiation. In early 1980s, he discovered that radiation demethylates the endogenous leukaemia virus genome in mice and the activated virus then integrates into new sites in the genome to induce leukaemia. He also discovered mutations of maternally inherited minisatellite sequences in F1 mice born to irradiated male parents in 1990–2010. These were well received as pioneering findings and he was awarded Roentgen Medal in 2005 for the work of radiation induced genomic instability.

He served in a number of academic positions at Hiroshima University, Kyoto University and the National Institute of Radiological Sciences (NIRS), and currently holds a position at Fukushima Medical University. He contributed to the promotion of radiation biol-

ogy by serving as the president of International Association of Radiation Research from 2007 to 2011. His contribution extends to the radiation protection field, serving the ICRP since 2001 and as a member of its Committee 1 on radiation effects as well as a Main Commission member.

Professor Roy Shore, Hiroshima, Japan

Dr Roy Shore received a Ph.D. in psychology from Syracuse University (1967) and a DrPH in epidemiology from Columbia University in 1982. He served as Professor and Chief of the Epidemiology Division at the New York University School of Medicine before going to the Radiation Effects Research Foundation (RERF) in Hiroshima-Nagasaki as Vice Chairman and Chief of Research in 2006. He is an author on numerous radiation-related publications and currently supervises RERF investigators on studies of radiation risks for a variety of diseases. Dr Shore has served on many governmental and scholarly committees, including as a long-time member of the ICRP and NCRP, and has served on various committees or task groups for UNSCEAR, WHO, the US National Academy of Sciences, the US National Cancer Institute and the US Environmental Protection Agency, among others. His interests include the effects of radiation on both cancer and non-cancer disease incidence, and understanding the epidemiologic and biological modification of radiation effects by various environmental, genetic and host-susceptibility factors.

Professor Richard Wakeford, Manchester, United Kingdom

Dr Richard Wakeford worked for British Nuclear Fuels plc (BNFL) for almost 30 years before taking early retirement in 2006. For much of this time he specialized in the risks to health posed by exposure to ionizing radiation, particularly low-level exposure. He is now Visiting Professor in Epidemiology at the Dalton Nuclear Institute of The University of Manchester, and is a member of a number of national and international expert groups such as the UK Committee on Medical Aspects of Radiation in the Environment (COMARE) and Committee 1 of the ICRP. In 2011, he was a member of the UK Government's Scientific Advice Group for Emergencies (SAGE) for the Japan Nuclear Incident, and his statement to the Japanese people on the risks from the Fukushima accident is available at the website of the Japanese Government's Cabinet Secretariat. He has been Editor-in-Chief of the *Journal of Radiological Protection* since 1997. Dr Wakeford has extensively studied the risks of radiation exposure in infants and children, as well as the risks associated with prenatal exposure.

Dr Linda Walsh, Munich, Germany

Dr Linda Walsh obtained her Ph.D. in Physics from the University of Manchester, U.K. in 1985. She has worked in British, Australian, Dutch, German and European Universities and research institutions in the fields of data analysis, applied statistics, numerical analysis and radiation epidemiology. Dr Walsh is currently working as a senior scientist at the German Federal Office for Radiation Protection (BfS). Dr Walsh's extensive research in the field of radiation epidemiology has included papers on the Life Span Study of Japanese Atomic bomb survivors covering a range of topics from cancer risks related to neutron and X-ray doses, organ-specific doses and carcinogenesis. She has also worked on the analysis of the cohort of German "Wismut" uranium miners exposed to radon and other potential carcinogens, and developed epidemiological models for lung and extra-pulmonary cancers.

Other studies that Dr Walsh has been involved with have considered the incidence of malignant diseases in humans injected with radium-224; the development of epidemiological models for thyroid cancer risk in areas affected by the 1986 Chernobyl accident; and analyses of data pertaining to cellular radiation damage relevant to the evaluation of both diagnostic radiation characteristics and the effects on cancer patients. Dr Walsh submitted her Doctor of Science thesis, based on 50 publications, entitled "Quantifications of the detrimental health effects of ionising radiation" to the Medical Faculty of Manchester University in December 2012. She has also been involved in various international research projects, most recently as a partner, task leader and project board member of an international research project started under the seventh framework programme of the European Union, FP-7-EU-ANDANTE (Multidisciplinary evaluation of the cancer risk from neutrons relative to photons using stem cells and the analysis of second malignant neoplasms following paediatric radiation therapy). As a WHO health risk assessment panel member, Dr Walsh made substantial contributions to the selection of risk assessment methodology, performed actual risk calculations and contributed to the writing of some major sections of the final report.

Dr Wei Zhang, Chilton, United Kingdom

Dr Wei Zhang received his Ph.D. in Plasma Physics and Controlled Nuclear Fusion from the University of Saskatchewan, Canada in 1993. He subsequently worked as a research scientist at the Centre Canadien de Fusion Magnétique in Quebec, Canada, and the Joint European Torus (JET) in the UK. In 1997, Dr Zhang started his career in Medical Statistics and Epidemiology at the University of Oxford. He joined the National Radiological Protection Board (now part of the Health Protection Agency) in 2002 and currently is a principal scientist at the Health Protection Agency. His interests cover health risk assessment, epidemiological studies of radiation workers and radiotherapy patients.

Annex B. Declaration of interests statement

All experts who participated in the meetings of the Health Risk Assessment Expert Group on the initial evaluation of radiation exposure from the nuclear accident after the 2011 Great East Japan Earthquake and Tsunami were asked to complete a WHO Declaration of Interest form. In some cases, the experts were asked to provide additional information on the form submitted by them.

At the start of the meetings of this Group, all participants were asked to confirm their interests and to provide any additional information relevant to the subject matter of the work.

Certain experts declared interests of a non-commercial nature, having worked for organizations such as UNSCEAR, the US National Cancer Institute (NCI), NCRP, and governmental radiation protection agencies. These interests were not deemed to give rise to a conflict. In addition, two of the experts declared having a potential conflict of interest of a commercial nature, as follows:

Dr Richard Wakeford: He is currently and has over the last 2 years performed consultancies (on the health effects arising from exposure to ionizing radiation) for EDF Energy plc, Augean plc, British Nuclear Fuels plc and Sellafield Ltd.

Dr Dominique Laurier: His unit at IRSN has received research support from Areva and EDF (for research projects on workers).

Dr Ohtsura Niwa later disclosed that, as a member of the International Commission on Radiological Protection (ICRP), he has (over the last 11 years) received travel support from the Radiation Effect Association using funds provided by several sources, one of which is the Federation of Electric Power Companies (an umbrella association of electricity companies). WHO brought this information to the attention of the other experts.

As noted in the section describing the background of each of the experts, Dr Wakeford has a unique expertise on radiation effects in infants and children, particularly on childhood leukaemia. Dr Laurier belongs to a WHO Collaborating Centre which includes in its terms of reference the provision of technical support on radiation risk assessment. He has particular expertise in the field of radiation epidemiology. Dr Niwa has particular expertise in molecular biology and radiation biology.

Considering these experts' unique knowledge and expertise in the fields described above, and bearing in mind that the remaining experts did not disclose any interests of a commercial nature, it was decided that their declared interests did not merit their exclusion from this Group, provided that these interests be publicly disclosed.

Annex C. Overview of radiation epidemiology

Available scientific data on the biological effects of ionizing radiation are based on experimental and epidemiological studies. The most informative source of epidemiological data about human exposure to radiation is the Life Span Study (LSS) of the Japanese survivors of the atomic bombings of Hiroshima and Nagasaki. In addition, several other sources have provided useful epidemiological data, including past accidents (e.g. Chernobyl), medical exposures, occupational exposures and natural radiation exposures, as described below.

C.1 Atomic bombings of Hiroshima and Nagasaki

The strongest evidence for cancer risk from ionizing radiation in humans has been obtained from the Life Span Study (LSS) of individuals exposed from the atomic bombs of Hiroshima and Nagasaki. Although cancer is the main late effect demonstrated in the LSS, it has also provided information about other health outcomes such as benign tumours and non-cancer diseases (*5,16,19,21,55,56,70,105*). There are reports showing excess cancer risk associated with in *utero* radiation exposures on the order of a few tens of mGy among children exposed to radiation *in utero* due to maternal X-ray pelvimetry as well as in various other populations prenatally exposed to radiation, including the LSS (*34,36,122,160*).

C.2 The Chernobyl accident

The follow-up of people exposed as a result of the nuclear power plant accident that occurred in 1986 at Chernobyl (Ukraine) provided information about radiation risks, particularly thyroid cancer, from internal exposure to radioactive iodine. A significant increase in the incidence of thyroid cancer was observed in residents of affected areas of Ukraine, Belarus and the Russian Federation, with higher risks among those exposed at a young age (i.e. infants, children and adolescents) compared with adults. Some increase in the risk of leukaemia among emergency workers was also reported (*22,82*).

C.3 Medical exposures

The carcinogenic effects (leukaemia and liver cancer) of long-term internal exposure were first reported beginning in the 1930s, among patients who had received thorium-containing Thorotrast as a contrast medium for radiological imaging procedures (*161*). Since the 1960s, long-term adverse consequences of radiotherapy from benign disease were documented in studies showing an increased risk of cancer after X-ray treatment for ankylosing spondylitis (*162*). These findings were confirmed during the 1970s, when analyses of cancer among children treated for enlarged thymus, enlarged tonsils and scalp ringworm showed an increased risk of thyroid cancer. Increased breast cancer risk

has been observed among young women who were repeatedly examined with fluoroscopy for tuberculosis (*162*). Recently, increased risks for leukaemia and brain tumours were reported in children undergoing repeated computed tomography scans that had cumulative doses in the range of around 50 mSv (*146*).

C.4 Occupational exposures

There have been a number of studies on workers exposed to radiation. The earliest cases of radiation-induced neoplasms were skin cancers, reported by pioneering researchers working with X-rays in the first years of the 20th century. Large doses of highly localized exposures resulting in excess risks of specific cancer types were demonstrated when bone cancers were reported among radium dial painters in the 1920s. Further evidence was reported in the 1950s, showing excess deaths from leukaemia among radiologists who began working in the early period with scanty radiation protection procedures (*163*). Excess cancer risks were observed among workers at the Mayak Plant in the Southern Urals after high-dose, low-dose-rate radiation exposure (*164*). A comprehensive review of 12 recent epidemiological studies on occupational exposures, including the 15-country study compiled by the International Agency for Research on Cancer (IARC) (*165*), concluded that there is evidence for an excess cancer risk among the populations occupationally exposed to moderate radiation doses at a low dose rate, and that there is no indication that such excess is smaller than for the atomic bomb survivors (*74,166*).

C.5 Environmental exposures

Excess cancer risks were reported in local residents after high-dose, low-dose-rate exposure to radiation from the Semipalatinsk nuclear test site (*167*). Epidemiological studies on cancer incidence or mortality conducted in residents of regions with some of the highest levels of background radiation in the world did not show excess cancer risk (*168,169,170,171*). Those studies have major methodological limitations and it is uncertain whether the studies conducted up to now were able to detect small excess risks (*172*). By applying recent risk models it was estimated that around one fifth of childhood leukaemia in Great Britain may be caused by exposure to natural background radiation in childhood. Authors acknowledged the uncertainties associated with such predictions, particularly concerning the nature of the risk transfer between populations (*173*). Indeed, a significant association between dose and red bone marrow owing to background radiation and childhood leukaemia risk was recently observed in a national case–control study (*174*).

Annex D. Survival curves

The probability of developing a cancer induced by radiation depends on the probability of being alive at that time. To calculate lifetime incidence cancer risks it is therefore necessary to know, for each age interval, the probability of the person being alive. This information is presented in "survival curves" or "survival functions". $S(a)$, the survival function, represents the probability of surviving to age a. $S(a)$ can be calculated from the age-specific all-cause mortality rates from $S(a) = exp\{-Mc(a)\}$, where $Mc(a)$ is the cumulative death rate up to attained age a. A summary of basic survival analysis concepts can be found in the appendix of a paper published by Thomas et al. in 1992 (*78*).

Deriving the survival function from all-cause mortality rates is appropriate where the lifetime risks of radiation-induced cancer death are concerned. However, it would be inadequate if the lifetime risks of radiation-induced cancer incidence are calculated, as many cancers have a high rate of cure and therefore the cancer incidence rate is higher than the cancer mortality rate. Thus, for calculating the lifetime attributable risk (LAR) pertaining to cancer incidence, an "adjusted" survival curve, $S_{aj}(a)$, which represents the probability of cancer-free survival, is preferable to the unadjusted curve $S(a)$. To calculate $S_{aj}(a)$, suitable data that relate to the same population are required. For the present assessment, "adjusted" survival curves $S_{aj}(a)$ were derived on the basis of all-cause mortality plus the difference between all-cancer incidence and all-cancer mortality (see Figures 26 and 27).

For this HRA, the HRA Expert Group agreed to calculate the LAR using adjusted survival curves $S_{aj}(a,g)$ as a function of age-attained a, describing the probability of surviving cancer-free of a person of sex g. The ratio used in the LAR equation $(S_{aj}(a,g))/(S_{aj}(e,g))$ is the conditional probability of a person of sex g, alive at age e, reaching at least age a.

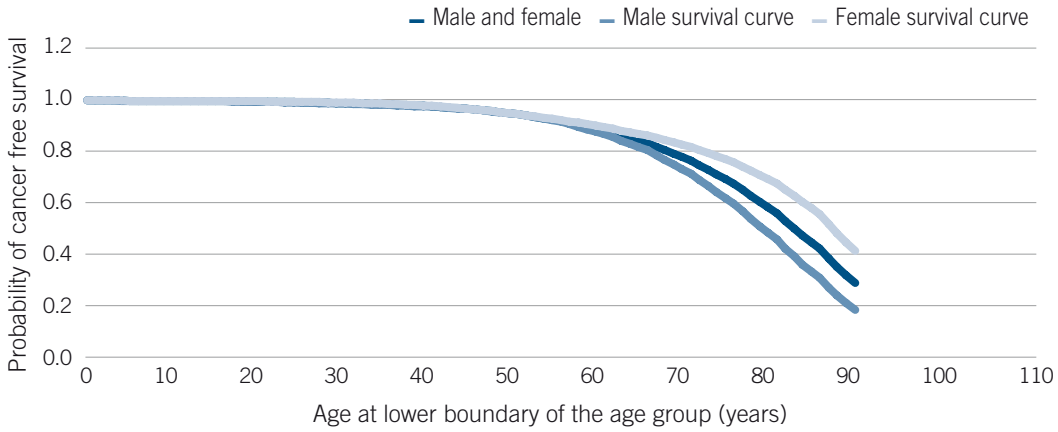
For the general population, the adjusted survival curves $S_{aj}(a,g)$ shown in Figure 26 were applied in the LAR calculations, and calculated from the all-cause mortality rates and all cancer mortality rates for 2010 obtained from the Portal Site of Official Statistics of Japan (*175*) and the all-cancer incidence rates for 2004 obtained from Table 3 of Matsuda et al. (*104*).

The healthy worker effect (HWE) is a bias found in occupational studies when rates of disease among employed people are compared with disease rates for the general population[1]. The so-called standardized mortality ratio (SMR) quantifies the difference in the mortality of workers with respect to the general population. If the SMR does not deviate much from 1, it indicates that the HWE is not very strong. The HRA Expert Group noted that this potential HWE might require care in the selection of a comparative population

1. The general population includes both employed and unemployed people and may therefore have a greater incidence, prevalence or mortality of disease than those who are employed. The strength of the HWE may vary from one occupational cohort to another and it is modified by a number of factors such as gender, age, length of employment and health monitoring status, among others.

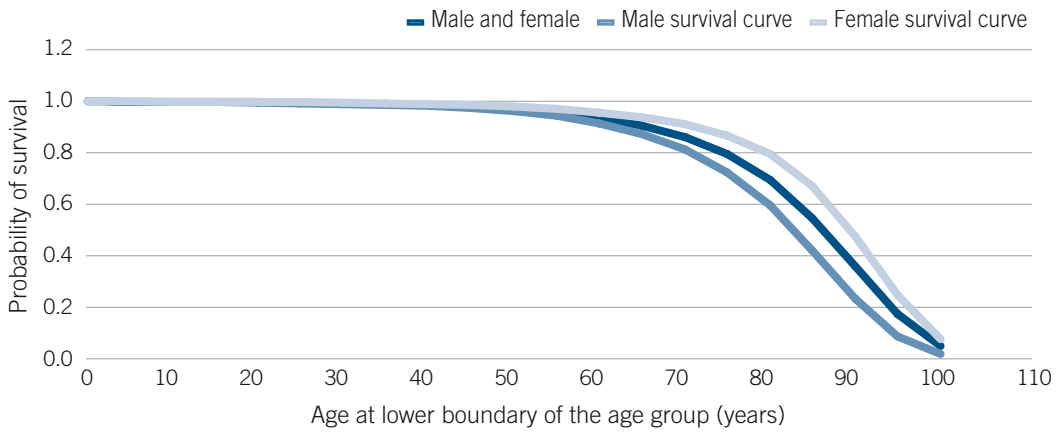
to calculate the LAR values for emergency workers in the present HRA. Two studies conducted in Japan were analysed. A study conducted in 2003 (*176*) showed a small deviation from one of the standardized mortality ratios (SMR). A recent study of Japanese nuclear workers does not give SMR values (*166*). The HRA Expert Group agreed to use for emergency workers the same adjusted survival curve (males) and the same baseline mortality and incidence rates as for the Japanese general population.

Figure 26. The adjusted survival curves, $S_{aj}(a)$, applied in the LAR calculations.



Note: adjusted survival curves cannot be plotted to age 100, as for S(a) – because the population cancer incidence and mortality data are not available for the 5-year age intervals beyond 90 years of age.

Figure 27. The unadjusted survival curves $S(a)$ are presented for comparison purposes.



Note: they were not applied for LAR calculation in this HRA.

Annex E. Risk models for assessing cancer risks

This annex summarizes the risk models used to calculate the lifetime attributable risks for the difference cancer sites considered in the present assessment.

In the latest LSS solid cancer incidence analysis, a statistically significant dose-response was seen when analysis were limited to cohort members with doses of 0.15 Gy or less (*70*). The threshold model did not fit better than a linear non-threshold model (LNT). Within the latest LSS cancer mortality analysis, the estimated lowest dose range with a significant ERR for all solid cancers was 0–0.2 Gy, and the analysis indicated no threshold (*19*).This is consistent with the concept that there is weak evidence for a threshold at any dose meaningful to radiological protection.

For leukaemia, the dose-response is better described by a linear-quadratic than by a simple linear pattern (i.e. curving upwards with increasing dose). The dose response is often approximated by a purely linear model at doses less than 100 mGy (*22*).

Studies of thyroid cancer following radiation exposure in childhood have provided evidence of excess risk at dose levels down to 0.1-0.5 Gy (100-500 mGy). The risk decreased significantly with increasing age-at-exposure, with little risk apparent after age 20 years (*6*). Studies of thyroid cancer following exposure to radioiodine from the Chernobyl accident have shown substantially elevated risks at organ doses around 0.5. The largest study covered more than 1000 cases and showed an excess relative risk of 19 per Gy, with a negative quadratic term (indicating a flattening of the dose-response at high doses) (*107*). This latter observation is consistent with findings reported at very high doses (>10 Gy), associated with cancer therapy, where it seems to be a decrease or leveling of thyroid cancer risk (*6*). A pool analysis of 4 high-dose studies also shows a flattening effect above 10 Gy (*24*). A large study of thyroid cancer risk among 12 500 survivors of childhood cancer treated with radiotherapy showed a downward bend of the dose response curve at approximately 20 Gy (*120*). In a case-control study of 276 thyroid cancer cases, the odds ratio was approximately 5 at 1 Gy but there was little further increase at doses exceeding 2 Gy (*177*). Subsequent studies with longer follow-up times (and higher attained ages and time since exposure) have shown lower risk estimates (around 2 per Gy) (*76,127*). One of these studies reported a flattening of the risk above 5 Gy (*127*). A study conducted in Bryansk, Russia, reported a very high risk estimate, though with a wide confidence interval (ERR 49 per GY, 95% CI 5-1150) (*108*). Also, an increased incidence of childhood thyroid cancer has been reported by the US from atmospheric atomic bomb testing with thyroid doses below 0.3 Gy and a relative risk of approximately 1.2 at dose 0.1 mGy, but only for exposure before the age of 1 year (*109*). The latest LSS thyroid cancer incidence analysis (*21*) indicated excess risks persisting for more than 50 years for those exposed as children and “little” evidence of increased thyroid cancer rates for those exposed after 20 years old. This latter finding is consistent

with the conclusion of Ivanov et al., based on populations affected by the Chernobyl accident (*23*).

E.1 Risk models for leukaemia mortality

The risk models chosen for the analysis for leukaemia mortality (ICD10 code: C91–C95) used a linear-quadratic dose response, from Table 46 of the UNSCEAR (*83*) report (see below E.1.1 and E.1.2) and Little et al. (*86*). In its 2006 report UNSCEAR (*83*) used excess absolute risk (EAR) and excess relative risk (ERR) models for leukaemia mortality for two conditions: quadratic response and linear-quadratic response. For the present assessment the EAR and ERR models with the linear-quadratic dose response were used. The coefficients of leukaemia mortality linear-quadratic models, fitted to current data for the survivors of the atomic bombings in Japan, are shown below. All models are fitted by Poisson maximum-likelihood, using adjustments for dosimetric error and assuming 35% geometric standard deviation errors.

E.1.1 Generalized ERR model

$$ERR = \lambda(a,e,D,g)[1+(\alpha D+\beta D^2)\exp[\kappa_1 \ln(a)]$$

where D is the radiation dose (Sv), a the attained age, e the age-at-exposure, g the sex, and the fit parameters are

$\alpha = 864.552\ Sv^{-1}$,

$\beta/\alpha = 1.180\ 92\ Sv^{-1}$,

$\kappa_1 = -1.647$

E.1.2 Generalized EAR model

$$EAR = \lambda(a,e,D,g)+(\alpha D+\beta D^2)\exp[\kappa_1 1_{female}+\kappa_2 \ln(a-e)]$$

where D is the radiation dose (Sv), a the attained age, e the age-at-exposure, g the sex, and the fit parameters are

$\alpha = 7.516\ 50\ X\ 10^{-4}\ Sv^{-1}\ a^{-1}$,

$\beta/\alpha = 1.034\ 55\ Sv^{-1}$,

$\kappa_1 = -525.26$,

$\kappa_2 = -614.1$

E.2 Risk models for all solid cancer incidence

Risk models and fit parameters for solid cancers (ICD-10 code: C00–C89) used in this assessment for calculating LAR were taken from Preston et al. (*70*) for all solid cancer incidence (1958–1998). The characteristics of the fit parameters are given in Preston et al. (*70*), Table 10. However the actual values of the fit parameters have been taken from the original EPICURE output on the RERF website and are given below.

E.2.1 ERR model

When use is made of a general rate (hazard) model of the form

$$\lambda(D,a,e,s) = \lambda_o(a,e,s)[1 + ERR(D,a,e,s)]$$

for the excess relative risk (ERR) where $\lambda_o(a,e,s)$ is the LSS baseline cancer death rate, a is age-attained and e is age-at-exposure, s is an indicator variable for sex ($s=-1$ for males, $s=+1$ for females) and D is the weighted colon dose (gamma colon dose plus ten times the neutron colon dose). The ERR is factorized into a linear function of dose and a modifying function that includes both age variables, $ERR(D, a, e)$. The functional form is exponential for age-at-exposure or a power function for age-attained where the modifying factors have been modelled as

$$ERR(D,a,e) = (1 + t.s).k_d D \exp[-g_e(e-30) + g_a \ln(a/70)]$$

where the fit parameters with standard errors are:

$t = 0.2465 \pm 0.06762$,

$k_d = 0.4666 \pm 0.04413$,

$g_e = 0.01849 \pm 0.00636$,

$g_a = -1.621 \pm 0.3058$

(fit parameters and deviance=14735.954, degrees of freedom, df=25551 values all from www.rerf:filename:-lss07sitemod.log).

E.2.2 EAR model

Similarly for EAR:

$$EAR(D,a,e) = (1 + t.s).k_d D \exp[-g_e(e-30) + g_a \ln(a/70)]$$

where the fit parameters with standard errors are:

$t = 0.1622 \pm 0.06988$,

$k_d = 51.63 \pm 4.982$,

$g_e = 0.02805 \pm 0.006215$,

$g_a = 2.406 \pm 0.2731$

(fit parameters and deviance =14739.933, df=25551, from www.rerf:filename:-lss-07sitemod.log).

E.3 Risk models for thyroid cancer incidence

The risk models for thyroid cancer incidence (ICD10 code: C73) are provided in this section. The ERR model is generally used in transfers of thyroid cancer risk from one population to another. However, the LSS cohort only provided data beginning 13 years after exposure. Extrapolation of the LSS results to shorter times after exposure was done on the basis of information from Chernobyl (*107,179*). For the period of 4–15 years after exposure, it can be seen from Figure 28 that an extrapolation of the EAR function for the LSS to periods shorter than 13 years shows good agreement with the Chernobyl experience. Therefore it was decided to present initial LAR results based on 50% EAR transfer and 50% ERR transfer (see section 3.5).

E.3.1 ERR model

$$ERR(D,a,e) = (1 + t.s).k_d D \exp(-g_e(e-30) + g_a \ln(a/70)]$$

where the fit parameters with standard errors are:

$t = 0.1433 \pm 0.2871$,

$k_d = 0.5767 \pm 0.2636$,

$g_e = 0.03739 \pm 0.02258$,

$g_a = -1.445 \pm 0.8157$

(fit parameters and deviance =3037.968, df=42020 from www.rerf:filename:lss07site-ahs.log).

E.3.2 EAR model

$$EAR(D,a,e) = (1 + t.s).k_d D \exp(-g_e(e-30) + g_a \ln(a/70)]$$

where the fit parameters with standard errors are:

$t = 0.5699 \pm 0.1649$,

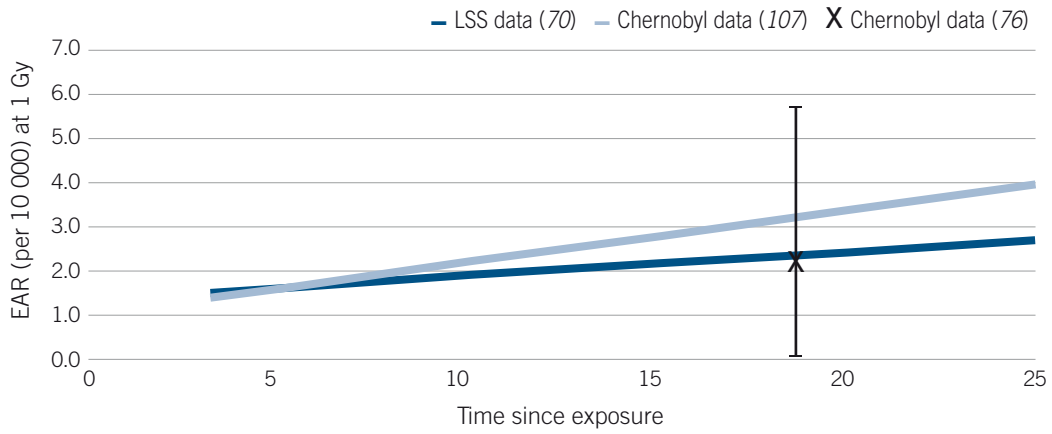
$k_d = 1.232 \pm 0.5061$,

$g_e = 0.05903 \pm 0.02385$,

$g_a = 0.5921 \pm 0.6191$

(fit parameters and deviance =3041.987, df=42020 from www.rerf:filename:-lss07site-ahs.log).

Figure 28. EAR estimates for thyroid cancer incidence in the LSS (Preston et al. (*70*), extrapolated to times after exposure shorter than 13 years) and in a cohort of Ukrainians having been exposed to radioactive iodine after the Chernobyl accident (Brenner et al (*76*)), and in Ukrainian settlements in which thyroid dose measurements have been performed during the first three months after the Chernobyl accident (Jacob et al. (*107*)), extrapolated to times after exposure longer than 15 years



E.4 Risk model for female breast cancer incidence

The risk model for breast cancer incidence (ICD10 code: C50) is provided in this section. Excess absolute risk models have been used for calculating LAR for female breast cancer.

The EAR function is:

$$EAR(D,a,e) = k_d D \exp[-g_e(e-30) + g_a \ln(a/70)]$$

where the fit parameters with standard errors are:

$k_d = 9.257 \pm 1.578$,

$g_e = 0.04543 \pm 0.01209$,

$g_a = 1.725 \pm 0.4526$

(fit parameters and deviance=3307.119, df=13199, from www.rerf:filename:-lss07site-mod.log).

Annex F. Dose response for deterministic effects

F.1 Dose response for cognitive impairment

The threshold doses above which deterministic effects may occur from prenatal exposure to ionizing radiation are set out in ICRP 84 (*65*) and ICRP 90 (*35*), which based their estimations on data from the LSS in atomic bomb survivors. The LSS did not find evidence of cognitive effects from radiation exposure during pre-implantation and organogenesis periods (i.e. weeks 0–7 after conception). Based on extrapolations from experimental models in rodents the 50% lethal dose (LD_{50}) in the pre-implantation period is considered to be around 1 Gy. Effects during the organogenesis period were observed in rodents when the radiation dose exceeded thresholds of around 0.5–1 Gy (i.e. moderate radiation doses). Threshold values for radiation effects during the organogenesis period are considered to be in general above 100–200 mGy. The LSS provided data on the frequency of severe mental retardation that support a lowest dose threshold of around 0.3 Gy (*35*). The threshold for a decrease in the intelligence quotient (IQ) is considered to be around 100 mGy. It is known that the brain has differing sensitivities to ionizing radiation at different stages of development. During the period of greatest radiation sensitivity of the developing brain (i.e. between the 8th and 15th week) a dose of 1 Gy is associated with a reduction of 25 points in IQ. Several studies have sought to identify evidence of cognitive impairment in children exposed to radiation *in utero* as a consequence of the Chernobyl accident. The outcomes of these studies have been conflicting and could not establish statistically significant dose-response relationships (*179, 180, 181,182,183,184,185*). Direct evidence that effects may exist at moderate doses comes from a cohort study of therapeutic irradiation for cutaneous haemangioma given before 18 months of age (*186*). This study demonstrated a dose-effect on cognitive impairment as judged by school attendance, occupation and achievement on military aptitude scores with effects seen only above 100 mGy and becoming more pronounced in patients with a dose over 250 mGy to the head. Another study showed deficits in IQ and scholastic aptitude in the irradiation group who received a mean brain dose of about 1.3 Gy at an average age of 7 years (*187*). After birth the brain appears to be very resistant to radiation damage, with effects on cognitive function being reported only in studies of children who had very high doses of radiation to the central nervous system (CNS) for the treatment of tumours, usually of at least 18 Gy, with significant decreases in IQ usually being noted above 24 Gy. The detrimental effect of very high dose irradiation of the CNS has been consistently noted to be greatest among the youngest children in these studies; although the effect was related both to the effect of the primary disease and the CNS irradiation (*188,189,190,191,192*). In summary, moderate to high doses of ionizing radiation received *in utero*, especially during the 8-25th weeks of pregnancy, may be associated with cognitive impairment. This is unlikely to occur at doses of less than 100 mGy. Statistical evidence of reduced IQ in children born in areas where radioactive contamination from the Chernobyl accident exists is controversial, and is likely, if

present, to result from many factors, including social disruption due to evacuation, and is potentially only a transient observation in early childhood. Cognitive impairment associated with exposure to ionizing radiation after birth has been demonstrated only where very high doses have been given to children to cure intra-cranial malignancies.

F.2 Dose response for cataract induction

The lens of the eye is recognized as one of the most radiosensitive tissues in the human body, although the mechanisms involved in it are still not fully understood. Cataracts had been considered as deterministic effects with a threshold of around 1.5 Gy (ICRP 103 (*12*)), and higher threshold values (around 5 Gy) for protracted exposures and for vision-impairing cataracts. During the last few years a number of studies including occupationally exposed medical staff (*193,194,195,196*), Chernobyl clean-up workers (*197*) and environmental exposures (*198*) indicated that radiation-associated lens opacities can occur at lower doses. The most recent data from the Japanese A-bomb survivors cohort provided evidence that the risk for vision-impairing cataracts is seen at doses lower than 1 Gy. The observed dose response was nearly linear, with a best-estimate of a threshold dose of around 0.5 Gy. The risk was highest for those who were young at exposure (*55*).

F.3 Dose response for circulatory diseases

There is considerable epidemiological evidence that doses of a few sieverts to the heart, such as those from older radiotherapy regimens for Hodgkin disease or breast cancer, are associated with an increased risk of heart and cardiovascular disease (*199*). There is, however, less evidence for heart disease risk at doses on the order of a few hundred millisieverts. The LSS of atomic bomb survivors found a dose-response association over the full dose range of 0 to about 3 Sv (*56*). However, the dose-response association over the range 0–0.5 Sv was not statistically significant. A test for a dose threshold showed no evidence for a threshold; however, a threshold of up to 0.5 Sv was compatible with the data. Several studies of medical low-dose radiation and a number of occupational radiation studies have examined cardiovascular risk (*59,63, 200, 201,202,203,204,205*). Most of those studies have reported positive radiation associations without statistical significance, suggesting that there is no clear evidence for risk after radiation exposures with doses of several hundred millisieverts. Many of those studies have been based on exposures in adulthood, but even those that include exposures at young ages have generally not demonstrated significant associations of radiation and heart disease. Furthermore, the atomic bomb study did not find a statistically significant age-at-exposure effect for heart disease (*56*). The radiation response in the studies on cardiovascular disease is related to doses larger than 100 mSv. A recent meta-analysis of 10 epidemiological studies with cumulative doses <500 mSv, or low dose rates of <10 mSv per day derived a significant dose-risk relationship with circulatory disease mortality (*61*). From the available evidence, there is no basis for considering cardiovascular disease risk in relation to expected exposures of <100 mSv, so it will not be considered in estimating the population health risks from Fukushima. Studies of radiation exposure and stroke or cerebrovascular disease risk have also shown evidence of an association at doses of several sieverts from radiotherapy of the head and neck. In this case, the atomic bomb study showed an association only at doses above 0.5–1 Sv and no evidence of an effect

below that level (*56*). The studies of cerebrovascular disease after low-dose or protracted radiation exposures have likewise mostly shown no risk, and in the few that have suggested risk, there are questions of confounding lifestyle or methodological factors. Again, most of those studies have reported positive radiation associations without statistical significance, suggesting that there is no clear evidence for risk after radiation exposures with doses of a few hundred millisieverts. There is no evidence to support a risk of stroke or cerebrovascular disease in relation to exposures of less than 100 mSv.

F.4 Dose response for thyroid nodules

The excess relative risk per gray for thyroid adenomas (benign neoplasms) and external irradiation appears to be somewhat less than for thyroid cancer (*44*). For nodules without histopathological diagnosis – representing a generally unknown mixture of adenomas, colloid nodules and hyperplastic nodules – the excess relative risks vary greatly by study, probably as a result of screening and methodological variations, so it is difficult to develop a risk estimate, though most studies indicate that an elevated nodule incidence is associated with radiation exposure (*43,44*). The limited data available suggest that thyroid nodule risk from internal exposure to radioactive iodine is roughly the same as that from acute external exposure. For thyroid adenomas there is a weak suggestion that adenoma risk is higher for early ages at exposure than at later ages, but most studies have not been able to examine age effects (*44*). Gender differences in the magnitude of excess relative risk are also unclear, though most studies indicate that excess absolute risks are greater for females than males.

Annex G. Methodology to calculate organ doses for the general public (different pathways)

The Dose Expert Panel provided their results in terms of effective doses and thyroid organ doses from the first-year exposure. The HRA Expert Group converted the effective doses into doses to specific organs: colon, red bone marrow and breast. Thyroid organ doses were also calculated and compared with the thyroid dose estimates from the Dose Expert Panel in order to test and validate the approach. This Annex describes the methodology applied to calculate organ doses for the general population.

G.1 Organ dose resulting from external exposure from ground deposition

The organ doses from external exposure from ground deposition were calculated by the HRA Expert Group based on data on effective doses provided by the Dose Expert Panel. Those effective doses had been estimated by using two different approaches, but there were only minor differences in the assumed relationships among the radionuclides deposited on the ground (see Table 1). In fact, according to the methods and data used, substantial contributions to external exposure were made by only four radionuclides: ^{132}Te, ^{131}I, ^{134}Cs and ^{137}Cs (^{137m}Ba). Based on that, a weighted ratio of organ dose-to-effective dose was calculated according to the following equation:

$$\frac{\sum_r Q_r \times DC_{o,r} \int_0^{365} e^{-0.693 \times \frac{t}{T_r}} dt}{\sum_r Q_r \times DC_{e,r} \int_0^{365} e^{-0.693 \times \frac{t}{T_r}} dt}$$

where

Q_r = relative source term for radionuclide r

$DC_{o,r}$ = dose coefficient for organ o for radionuclide r

$DC_{e,r}$ = effective dose coefficient for radionuclide r

T_r = half-life of radionuclide r

t = time.

The values of Q_r are taken from the Dose Assessment report (*3*); values of DCs are taken from Jacob et al. (1990) (*206*). The use of this equation thus weights the calculated ratio by the relative contribution of each radionuclide to the dose. Weighted values of the ratio of organ dose-to-effective dose for external exposure to ground-deposited sources are given in Table 19.

Table 19. Weighted values of the ratio of organ dose-to-effective dose for external exposure to ground-deposited sources from Fukushima release

Age group	Breast	Colon (LLI**)	Red bone marrow	Thyroid
Adult 20y	0.99	0.91	0.89	1.0
Child 10y	1.0 *	0.96	1.0	1.0
Infant 1y	1.0 *	0.91	0.94	1.0

* Jacob et al. (1990) (206) do not give values of dose coefficients for child and infant breast tissue, so the value is assumed to be equal to that of effective dose

** LLI: lower large intestine.

G.2 Organ dose resulting from external exposure from the plume

No additional radionuclides (such as ^{133}Xe)[1] were assumed to contribute substantially to external dose from the plume, so the ratios of organ dose-to-effective dose are assumed to be the same as for external dose from ground-deposited radionuclides. Values are as given in Table 19.

G.3 Organ dose resulting from internal exposure from inhalation of radionuclides in the plume

The internal dose from inhalation of radionuclides contained in the plume was calculated by the members of the Dose Expert Panel by "suspending" the ground deposits into the air by dividing by a deposition velocity. For the calculations given here the same radionuclide mix as before was used, and nine radionuclides were considered. Dose coefficients for inhalation of radionuclides were taken from (*207*). The weighted ratios were calculated as before with use of the equation given above. Results are given in Table 20. In this case the ratios differ substantially from 1.0.

Table 20. Weighted values of the ratio of organ dose-to-effective dose for inhalation of radionuclides contained in the Fukushima cloud

Age group	Breast	Colon (LLI**)	Red bone marrow	Thyroid
Adult 20y	0.45	0.69	0.56	7.3
Child 10y	0.23	0.49	0.31	12.0
Infant 1y	0.12	0.42	0.16	15.0

* LLI: lower large intestine.

1. Although ^{133}Xe is a major contributor to the external dose from the plume, its contribution to the organ doses from external exposure would be similar to that for the other radionuclides considered. Therefore, not considering it explicitly was not important for the purpose of the organ dose calculation.

G.4 Organ dose resulting from internal exposure from ingestion of radionuclides in food

The report of the Dose Expert Panel gives values of dose calculated for three radionuclides: ^{131}I, ^{134}Cs, and ^{137}Cs. Dose coefficients for ingestion of radionuclides were taken from (*207*). The ratios of organ dose-to-effective dose were examined for each of these three radionuclides, and no weighting was assigned for the composite mix. The calculated values are given in Table 21.

Table 21. Values of the ratio of organ dose-to-effective dose for ingestion of radionuclides contained in food

Age group	Breast	Colon (LLI**)	Red bone marrow	Thyroid
		^{131}I		
Adult 20y	0.0027	0.0055	0.0045	20
Child 10y	0.0029	0.0054	0.0031	19
Infant 1y	0.0023	0.0083	0.0021	20
		^{134}Cs		
Adult 20y	0.74	1.1	0.95	0.95
Child 10y	0.70	1.2	0.93	1.00
Infant 1y	0.69	1.5	0.81	1.00
		^{137}Cs		
Adult 20y	0.85	1.2	1.00	1.00
Child 10y	0.80	1.3	0.93	0.97
Infant 1y	0.76	1.9	0.82	0.91

* LLI: lower large intestine.

Annex H. Data Provided by the Tokyo Electric Power Company (TEPCO)

H.1 Workers exposure assessment

According to information provided by TEPCO, the internal dose assessment of Fukushima Daiichi NPP workers was based on in vivo measurements performed with whole-body counters (WBCs) used for internal dosimetry[1]. Three kinds of WBCs were used for internal dosimetry:

1. WBCs with plastic scintillator without capability for radionuclide identification were used for initial screening,
2. WBCs with sodium iodide (NaI) scintillator were used as a second step for identification and quantification of radionuclide body burden (Bq) in workers whose internal dose assessment exceeded 20 mSv. Overestimation of the amount of ^{131}I deposited in the thyroid gland was observed with these detectors, owing to geometrical characteristics.
3. WBCs with germanium (Ge) semiconductor detector for more precise radioactivity measurement and radionuclide identification if internal doses were 250 mSv. or higher.

Before June 2011 substantial levels of internal contamination were detected in many workers. Measurements were therefore taken with WBC NaI scintillators and more precise measurements were taken as described above. After end of June 2011 WBC plastic scintillators were used for screening because no more ^{131}I was detected 3 months after the accident. Consequently, the number of measurements with WBC plastic scintillator was increased. If measurements were below the established screening level (20 000 cpm) the process was stopped as "below recording level". If the screening level was exceeded, a precise measurement using WBC NaI was taken.

TEPCO reported that internal doses were calculated by multiplying the intake of radionuclides, as estimations based on the measurements through WBC (Bq), by the appropriate effective dose coefficient for inhalation (mSv/Bq) (see Table 22). The internal dose was defined as an effective dose over 50 years after the intake of radionuclides, which was calculated by dividing the amount of radionuclides measured by WBC by a retention rate (ratio between the radioactivity measured by WBC and the intake).

The retention rate is the ratio between the activity measured by WBC and the intake. It is a function of time after intake and it depends on the radionuclides and their physicochemical forms. The target nuclides considered were ^{131}I, ^{132}Te/^{132}I, ^{137}Cs and ^{134}Cs.

1. The internal dose assessment included iodine, tellurium and caesium (^{131}I; ^{132}Te/^{132}I; ^{137}Cs; ^{134}Cs).

Table 22. Effective dose coefficients used to estimate internal dose in workers

Radionuclide	Effective dose coefficients (mSv/Bq)
^{131}I (vapour)	2.0×10^{-5}
^{132}Te / ^{132}I (vapour)	5.1×10^{-6} / 3.2×10^{-7}
^{137}Cs (all compounds : type F)	6.7×10^{-6}
^{134}Cs (all compounds : type F)	9.6×10^{-6}

TEPCO used retention data from MONDAL 3 software developed by NIRS[2], assuming that the route of intake for workers was inhalation. The following assumptions were adopted:

- The chemical form of the iodine was gaseous (vapour).
- The absorption type for caesium was Type F (fast).
- The intake scenario was an acute intake from inhalation on the first day of work for workers who started in March or April 2011 (conservative approach). For workers who started working in May 2011 or later, it was assumed that the acute inhalation intake occurred in the middle of the working period.
- Correction was made when ^{131}I was not identified by WBC (see below).
- For workers whose internal doses exceeded 20 mSv, an individual assessment based on behaviour information was attempted.

When ^{131}I was not detected by WBC measurements, a correction was made by using the minimum detectable activity (MDA), assuming an intake corresponding to the MDA of ^{131}I and by multiplying the amount of ^{137}Cs by the ratio ^{131}I to ^{137}Cs in the working environment. The actual intake must be smaller than the intake calculated from the MDA value. If the amount calculated from the ratio of ^{131}I to ^{137}Cs was larger than the one calculated from the MDA value, the latter was used for the internal dose assessment.

The method described above could be applied only for single intakes. In case of multiple intakes, the method considered the residual activity of the radionuclide identified in a previous measurement as follows:

1. The intake I1 was calculated from the measured value S1.
2. A residual amount was estimated from I1 at the time of a second measurement BG_{S2}.
3. A net measured value was calculated by subtracting BG_{S2} from S2.
4. An additional intake I2 was estimated from S2 minus BG_{S2}.

Data concerning workers' monitoring are available on TEPCO's website at:

http://www.tepco.co.jp/en/press/corpcom/release/betu11_e/images/111130e20.pdf

http://www.tepco.co.jp/en/press/corp-com/release/betu11_e/images/111227e3.pdf

2. MONDAL software for internal dosimetry is based on the methodology and parameters proposed in ICRP Publications 54 and 78, and includes additional radionuclides.

H.2 Exposure data provided by TEPCO in March 2012

Table 23. Distribution of workers by age ranges and by effective dose E (including external and internal exposure)

18-19 years old

Effective dose, E (mSv)	TEPCO	Contractors	Total
250< E	0	0	0
200 < E ≤ 250	0	0	0
150< E ≤ 200	0	0	0
100< E ≤ 150	0	0	0
50< E ≤ 100	1	0	1
20< E ≤ 50	0	7	7
10< E ≤ 20	2	7	9
E ≤ 10	0	47	47
Total	3	61	64
Max(mSv)	56.89	44.34	56.89
Mean (mSv)	28.28	7.27	8.26

20-29 years old

Effective dose, E (mSv)	TEPCO	Contractors	Total
250< E	3	0	3
200 < E ≤ 250	0	1	1
150< E ≤ 200	2	0	2
100< E ≤ 150	19	0	19
50< E ≤ 100	116	33	149
20< E ≤ 50	108	183	291
10< E ≤ 20	92	288	380
E ≤ 10	171	1041	1212
Total	511	1546	2057
Max(mSv)	477.01	230.90	477.01
Mean (mSv)	34.98	9.55	15.86

30-39 years old

Effective dose, E (mSv)	TEPCO	Contractors	Total
250< E	1	0	1
200 < E ≤ 250	1	1	2
150< E ≤ 200	2	1	3
100< E ≤ 150	32	2	34
50< E ≤ 100	106	76	182
20< E ≤ 50	175	437	612
10< E ≤ 20	157	563	720
E ≤ 10	451	2174	2625
Total	925	3254	4179
Max(mSv)	678.80	238.42	678.80
Mean (mSv)	23.61	10.03	13.03

40-49 years old

Effective dose, E (mSv)	TEPCO	Contractors	Total
250< E	1	0	1
200 < E ≤ 250	0	0	0
150< E ≤ 200	11	0	11
100< E ≤ 150	32	5	37
50< E ≤ 100	103	102	205
20< E ≤ 50	208	598	806
10< E ≤ 20	163	727	890
E ≤ 10	655	3288	3943
Total	1173	4720	5893
Max(mSv)	645.54	139.60	645.54
Mean (mSv)	20.66	9.40	11.64

50-59 years old

Effective dose, E (mSv)	TEPCO	Contractors	Total
250< E	1	0	1
200 < E ≤ 250	0	0	0
150< E ≤ 200	7	0	7
100< E ≤ 150	32	6	38
50< E ≤ 100	86	104	190
20< E ≤ 50	145	613	758
10< E ≤ 20	76	739	815
E ≤ 10	346	3254	3600
Total	693	4716	5409
Max(mSv)	352.08	137.00	352.08
Mean (mSv)	26.19	9.57	11.70

60-69 years old

Effective dose, E (mSv)	TEPCO	Contractors	Total
250< E	0	0	0
200 < E ≤ 250	0	0	0
150< E ≤ 200	0	1	1
100< E ≤ 150	2	2	4
50< E ≤ 100	3	20	23
20< E ≤ 50	5	195	200
10< E ≤ 20	2	264	266
E ≤ 10	15	1349	1364
Total	27	1831	1858
Max(mSv)	124.63	169.60	169.60
Mean (mSv)	24.91	7.95	8.20

70 to 80 years old

Effective dose, E (mSv)	TEPCO	Contractors	Total
250< E	0	0	0
200 < E ≤ 250	0	0	0
150< E ≤ 200	0	0	0
100< E ≤ 150	0	0	0
50< E ≤ 100	0	1	1
20< E ≤ 50	0	0	0
10< E ≤ 20	0	6	6
E ≤ 10	1	18	19
Total	1	25	26
Max(mSv)	0.11	59.67	59.67
Mean (mSv)	0.11	7.33	7.06

Table 24. Distribution of thyroid doses D for workers

Thyroid dose D (mSv)	Persons
D ≥ 10 000	2
2 000 < D ≤ 10 000	10
1 000 < D ≤ 2 000	32
500 < D ≤ 1 000	50
200 < D ≤ 500	69
100 < D ≤ 200	15
100	344
Total	522

Note: Measurement of thyroid gland was performed until 5 February 2012 at NIRS or JAEA. These results are based on activity of ^{131}I; the contribution of Cs is not considered. Doses were estimated based on activity of ^{131}I.

Table 25. Number of workers exposed to different effective doses E from the main radionuclides

Effective doses (mSv)	^{131}I (no correction)	^{132}Te / ^{132}I	^{137}Cs	^{134}Cs
E ≥ 100	13	0	0	0
50 < E ≤ 100	32	0	0	0
20< E ≤ 50	86	0	0	1
10 < E ≤ 20	195	0	1	0
5 < E ≤ 10	145	1	2	6
2 < E ≤ 5	190	1	18	24
1 < E ≤ 2	134	2	41	91
E ≤ 1	22 877	23 668	23 610	23 550

Table 26. Relative contribution to the internal effective dose for the main radionuclides

Internal effective dose (mSv)	^{131}I	^{132}Te / ^{132}I	^{137}Cs	^{134}Cs
All workers	0.28	0.00	0.08	0.10
< 15	0.25	0.00	0.08	0.10
15 – 150	0.95	0.00	0.02	0.02
> 150	1.00	0.00	0.00	0.00

Note: the values in each of the first two rows do not add to 1 because workers with zero doses are included.

Table 27. Number of workers having doses in the specified ranges arising from from internal exposure by inhalation

Effective dose, E (mSv)	TEPCO	Contractors	Total
E ≥ 250	5	0	5
200 < E ≤ 250	1	0	1
150 < E ≤ 200	1	0	1
100 < E ≤ 150	5	0	5
50 < E ≤ 100	37	42	79
20 < E ≤ 50	194	94	288
10 < E ≤ 20	425	337	762
5 < E ≤ 10	316	424	740
E ≤ 5	4 655	16 636	21 291
Total	5 639	17 533	23 172
Max	590	96.84	590

Table 28. Workers' effective dose E resulting from external exposure (March 2011 to January 2012)

Effective dose, E (mSv)	TEPCO	Contractors	Total
E ≥ 250	0	0	0
200 < E ≤ 250	0	0	0
150 < E ≤ 200	7	3	10
100 < E ≤ 150	57	8	65
50 < E ≤ 100	307	237	544
20 < E ≤ 50	677	1889	2566
10 < E ≤ 20	550	2 559	3 109
E ≤ 10	1 741	12 068	13 809
Total	3 339	16 764	20 103
Max	188.14	199.42	199.42
Mean	18.83	8.38	10.11

Annex I. Methodology to calculate organ doses for workers (different pathways)

The workers' doses were provided by TEPCO in terms of effective doses, and only for a limited group of workers in terms of thyroid organ doses. The HRA Expert Group converted the effective doses into organ doses as of colon, red bone marrow, and thyroid. This annex describes the methodology applied to calculate workers' organ doses.

I.1 Approach A

This section describes the methodology (Approach A) used to calculate organ doses resulting from internal and external exposure of workers, from the effective dose reported by TEPCO. Another approach (Approach B) was used in addition to calculate organ doses resulting from internal exposure, as described in section I.2.

Mixture of radionuclides

One of the missing ingredients needed to make the calculation of organ doses was the mix of radionuclides to which the workers were exposed. The information provided initially by TEPCO was very limited, and some additional data were provided later upon request. Some important information provided by TEPCO is in Table 29, showing that almost all of the high doses were due to ^{131}I. Table 29 shows the number of workers who were assessed as having doses in the specified range arising from intakes of each radionuclide, rather than the relative contribution of each radionuclide to effective dose (which would vary among workers). Confounding factors could include, for instance, the possibility that workers monitored for iodine in the thyroid were not monitored for caesium in a whole-body counter (WBC), or vice versa. These reservations were the reason for requesting the data shown in the Table 26, Annex G. Relative contribution to the internal effective dose for the main radionuclides. TEPCO reported that 98% of the dose to the worker with the maximum internal dose was due to ^{131}I and provided a summary report to the HRA Expert Group during the process of development of this HRA.

Table 29. Contribution of different radionuclides to the effective dose *E*

Effective doses (mSv)	^{131}I	^{132}Te / ^{132}I	^{137}Cs	^{134}Cs
$E \geq 100$	13	0	0	0
$50 < E \leq 100$	32	0	0	0
$20 < E \leq 50$	86	0	0	1
$10 < E \leq 20$	195	0	1	0
$5 < E \leq 10$	145	1	2	6
$2 < E \leq 5$	190	1	18	24
$1 < E \leq 2$	134	2	41	91
$E \leq 1$	22 877	23 610	23 610	23 550

Note: The table indicates the number of workers having doses in the specified ranges arising from intakes of each of those radionuclides.

Thus, it seemed reasonable to the HRA Expert Group to make the assumption that virtually the entire effective internal dose came from inhalation of ^{131}I. This assumption could be improved if individual dosimetric or monitoring data could be provided. If virtually the entire internal dose was due to ^{131}I, this implies that the ^{131}I must have been in a volatile form, and if so, there must have been a substantial amount of ^{133}Xe accompanying the ^{131}I. Both ^{131}I and ^{133}Xe are short-lived radionuclides and would have come into equilibrium concentrations within the reactor well before the releases occurred. In order to calculate external doses from the two radionuclides it is important to know the ratio of ^{133}Xe to ^{131}I. Because of the equilibrium situation, it is not unreasonable to assume that the ratio is the same as the releases from the Chernobyl accident. According to UNSCEAR (*208*) the total release of ^{133}Xe was 6500 PBq and the release of ^{131}I was ~1760 PBq. This gives a ratio of 3.7.

Ratio of internal organ doses to effective dose via inhalation

It has generally been assumed that the larger internal doses to workers resulted from acute exposures. Moreover, given the assumption for these high exposures that the only radionuclide contributing substantially to internal dose was ^{131}I, the ratios of organ dose-to-effective dose are simply the ratios of the dose coefficients. These data are shown in Table 29 (ICRP (*207*)). So, for example, if doses to workers are provided in terms of effective dose from inhalation of ^{131}I, the equivalent (or absorbed) dose to each of the five organs listed in Table 30 is obtained by simply multiplying the given effective dose by the ratio. For example, the dose to the thyroid would be 19.5 times the effective dose.

Table 30. Dose coefficients for inhalation (Sv/Bq) from (*207*) for the organs of interest for an adult worker

Time after intake	1 day	30 days	1 year	5 years	Ratio: organ dose / effective dose
Colon	3.20E-11	6.10E-11	6.50E-11	6.50E-11	0.0033
Red bone marrow	2.60E-11	8.80E-11	9.30E-11	9.30E-11	0.0047
Thyroid	2.30E-08	3.70E-07	3.90E-07	3.90E-07	19.5
Effective dose	1.30E-09	1.90E-08	2.00E-08	2.00E-08	1.0

Note: The last column gives the ratio of organ dose-to-effective dose.
^{131}I, Inhalation of elemental vapour, f1 = 1.0, highest equivalent dose coefficient: Thyroid, 3.9E-07 Sv/Bq. Remainder formulation: default

Once the exposure scenarios were developed, it was decided to use the ratios presented in Table 30 (inhalation of elemental iodine vapour) only in Scenario 1 and to assume that iodine was in particulate form for Scenario 2. The rationale for such an assumption was that Scenario 1 corresponds to the high thyroid doses resulting from iodine in elemental form that was seeping into the control room (s) and therefore it had to be elemental in form. ^{133}Xe had to be seeping with the iodine, and although no data on ^{133}Xe were provided it was considered in the scenario. In contrast, Scenario 3 would be more applicable at later times. Because elemental iodine does not exist for a long time, it was assumed that iodine was attached to particles. Table 31 provides the ratios or organ dose to effective dose for particulate iodine.

Table 31. Dose coefficients from (*207*) for the organs of interest

Organ	Ratio: organ dose / effective dose
Colon	0.0036
Red bone marrow	0.005
Thyroid	19
Effective dose	1

Note: It is assumed that iodine was in particulate form

Calculation of external dose based upon the reported internal effective dose from inhalation

To calculate the external dose it is necessary to account for the external dose being submerged in the cloud of ^{131}I and it is prudent to consider the external dose from the ^{133}Xe that must have accompanied the ^{131}I in the ratio given above.

The first step is to calculate the ratio of integrated air concentration-to-effective dose for ^{131}I. This requires several assumptions be made. Again, it is assumed that an acute intake occurred. Another reasonable assumption is that the breathing rate of the workers was 1.5m^3 h^{-1}. The desired ratio is given by the equation below

$$R_a = \frac{1}{DC_e} \times \frac{1}{BR} \qquad (1)$$

where

R_a = ratio of integrated air concentration to effective dose, $Bq\ h\ m^{-3}\ Sv^{-1}$

DC_e = effective dose coefficient for ^{131}I, $Sv\ Bq^{-1}$ and

BR = breathing rate, $m^3\ h^{-1}$.

For example, the value of this ratio for ^{131}I is 3.3 x $107\ Bq\ h\ m^{-3}\ Sv^{-1}$. Keeping in mind that the bottom part of the ratio is the effective dose for ^{131}I, the value of this ratio for ^{133}Xe is 3.7 times larger, or 1.2 x$108\ Bq\ h\ m^{-3}\ Sv^{-1}$.

It is assumed that the external dose received by workers is due to submersion in a cloud containing these two radionuclides are required. In order to make the calculation the dose coefficients for immersion in a cloud for the two radionuclides are required. Values to be used are taken from (*208*), as was done for the assessment of the dose for members of the general population. The values are shown in Table 32; the last column is the sum of the dose coefficient for ^{131}I + 3.7 x the dose coefficient for ^{133}Xe. The factor of 3.7 is the ratio of assumed concentrations for the ^{133}Xe-to-^{131}I ratio discussed above.

Table 32. Dose coefficients for submersion in a semi-infinite cloud

Organ	Dose coefficient (nSv m^3 h^{-1} Bq^{-1})		
	^{131}I	^{133}Xe	^{131}I + 3.7 x ^{133}Xe
Colon	0.051	0.0031	0.062
Red marrow	0.059	0.0035	0.072
Thyroid	0.075	0.0055	0.094
Effective dose	0.060	0.0046	0.077

Note: Values in the second and third columns are taken from (*207*)

The ratio of external dose-to-effective dose from inhalation is given by the equation below:

$$R_{ext} = R_a \times DC_{ext} \times C = \frac{1}{DC_e} \times \frac{1}{BR} \times DC_{ext} \times C$$

where

R_{ext} = ratio of organ external dose-to-effective dose from the inhalation of ^{131}I, $Sv\ Sv^{-1}$

DC_{ext} = dose coefficient for external dose, as given in Table 31, $nSv\ h^{-1}\ m^3\ Bq^{-1}$ and

C = constant equal to $10^{-9}\ Sv\ nSv^{-1}$.

Values of the ratio, R_{ext}, for the organs of interest are shown in Table 33. Therefore for instance, if we receive data on effective dose due to inhalation exposure and we wish to know the external dose to the thyroid, we would simply multiply by $0.0032\ Sv\ Sv^{-1}$. Table 27 shows that the contributions to thyroid dose and effective dose from external irradiation due to submersion in the cloud are much less than those due to inhalation[1].

Irradiation from deposited activity and irradiation due to loss of containment, which could have been the dominant sources of external exposure for many workers, were not specifically considered in Approach A, although the possibility of higher external exposures was taken into account when specifying Scenario 1. Consideration of these other possible routes of exposure would have required more detailed individual-specific information that was not available when the HRA expert group did its work.

Table 33. Calculated values of the ratio of organ external dose-to-effective dose (R_{ext}) from the inhalation of ^{131}I

Organ	R_{ext}
Colon	0.0021
Red marrow	0.0024
Thyroid	0.0032
Effective dose	0.0026

1. Note that the dose coefficients in Table 32 are expressed in $nSv\ m^3\ h^{-1}\ Bq^{-1}$, while in Table 30 they are expressed in Sv/Bq ($1nSv = 10^{-9}\ Sv$)

I.2 Approach B

In addition to the methodology described above, organ doses resulting from internal exposure through inhalation were also calculated using a commercial code that implements the current ICRP biokinetic and dosimetric models and can calculate both absorbed dose to organs in each calendar year (Gy) and also the effective dose to the individual (Sv). Absorbed doses received by specified organs in each calendar year after exposure were calculated for an intake of a specified radionuclide that would give rise to an effective dose of 1 mSv. To calculate organ-absorbed doses for an effective dose of "x" mSv resulting from an intake of the specified radionuclide, these organ-absorbed doses would be multiplied by "x".

The procedure was thus to use the code to calculate (i) the effective dose E (mSv) from an intake of 1 Bq of the specified radionuclide and (ii) the organ doses following an intake that would give rise to an effective dose of 1 mSv, i.e. 1/E Bq; and repeat for each radionuclide. For the purposes of the workers' HRA the organs considered were red bone marrow, colon and thyroid.

The radionuclides considered were ^{131}I, ^{137}Cs, ^{134}Cs and ^{132}Te/^{132}I.

It was assumed that:

- All intakes were by acute inhalation of particulate material with an activity median aerodynamic diameter (AMAD) of 5 μm.
- All model parameter values were standard ICRP defaults for the adult worker.
- ^{132}I and ^{132}Te were in secular equilibrium.
- ^{131}I was in particulate form, not vapour[2].

With the exception of ^{137}Cs, organ doses received after the first calendar year are negligible, because of the short effective half-life of the radionuclides. For ^{137}Cs, about 15% of the total absorbed dose to an organ is received in the 2nd and subsequent years[3]. The four exposure scenarios assumed for this assessment were simplified to facilitate the calculation of organ doses based on the available information on radionuclide composition.

2. Approach A assumed vapour form for Scenario 1 and particulate form for Scenario 2.
3. Given the gross assumptions of the four scenarios presented below, this percentage can be considered as negligible.

Annex J. Lifetime attributable risk (LAR) and cumulative attributable risk (AR_{15}) in the general population based on lifetime doses

The total lifetime exposure presented in the tables included in this annex refers to the organ doses in colon (for all solid cancers), bone marrow (for leukaemia), thyroid (for thyroid cancer) and female breast (for breast cancer).

Table 34. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) for all solid cancers, leukaemia and breast cancer for a one-year old age-at-exposure

Region	Model	All solid cancers			Leukaemia			Breast cancer	
		Total lifetime exposure (mSv)	LAR (x 10^{-2}) (males)	LAR (x 10^{-2}) (females)	Total lifetime exposure (mSv)	LAR (x 10^{-2}) (males)	LAR (x 10^{-2}) (females)	Total lifetime exposure (mSv)	LAR (x 10^{-2}) (females)
①	**WHO weighted**	27.04	**0.7305**	**1.1131**	26.34	**0.0404**	**0.0274**	27.74	0.3567
	LSS ERR		0.7206	0.964		0.0596	0.0418		
	LSS EAR		0.7403	1.2624		0.0211	0.0129		
②	**WHO weighted**	15.84	**0.4246**	**0.6469**	15.30	**0.0229**	**0.0156**	16.10	0.2053
	LSS ERR		0.4193	0.5606		0.0338	0.0237		
	LSS EAR		0.4300	0.7333		0.0121	0.0074		
③	**WHO weighted**	6.17	**0.1604**	**0.2442**	5.56	**0.0079**	**0.0054**	5.78	0.0712
	LSS ERR		0.1590	0.2122		0.0115	0.0081		
	LSS EAR		0.1619	0.2762		0.0043	0.0026		
④	**WHO weighted**	10.75	**0.2487**	**0.3769**	10.35	**0.0117**	**0.0079**	10.89	0.1083
	LSS ERR		0.2501	0.3314		0.0158	0.0111		
	LSS EAR		0.2472	0.4223		0.0075	0.0046		
⑤ to ⑨	**WHO weighted**	9.01	**0.2084**	**0.3158**	8.58	**0.0049**	**0.0065**	9.01	0.0897
	LSS ERR		0.2096	0.2777		0.0077	0.0092		
	LSS EAR		0.2071	0.3539		0.002	0.0038		
LBR (x10^{-2})			**40.60**	**29.04**		**0.60**	**0.43**		**5.53**

Table 35. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) for all solid cancers, leukaemia and breast cancer for a 10-year old age-at-exposure

Region	Model	All solid cancers			Leukaemia			Breast cancer	
		Total lifetime exposure (mSv)	LAR (x 10^{-2}) (males)	LAR (x 10^{-2}) (females)	Total lifetime exposure (mSv)	LAR (x 10^{-2}) (males)	LAR (x 10^{-2}) (females)	Total lifetime exposure (mSv)	LAR (x 10^{-2}) (females)
①	**WHO weighted**	26.21	**0.5683**	**0.8594**	26.31	**0.0199**	**0.0136**	25.88	0.2218
	LSS ERR		0.5797	0.7692		0.0200	0.0150		
	LSS EAR		0.5569	0.9497		0.0198	0.0121		
②	**WHO weighted**	14.75	**0.3169**	**0.4791**	14.74	**0.0109**	**0.0075**	14.45	0.1225
	LSS ERR		0.3236	0.4292		0.0110	0.0082		
	LSS EAR		0.3102	0.5291		0.0109	0.0067		
③	**WHO weighted**	5.97	**0.1242**	**0.1875**	5.75	**0.0041**	**0.0028**	5.53	0.0450
	LSS ERR		0.1273	0.1684		0.0041	0.0031		
	LSS EAR		0.1210	0.2065		0.0041	0.0026		
④	**WHO weighted**	10.24	**0.1891**	**0.2840**	10.23	**0.0068**	**0.0046**	10.02	0.0669
	LSS ERR		0.1969	0.2580		0.0067	0.0049		
	LSS EAR		0.1812	0.3100		0.0069	0.0043		
⑤ to ⑨	**WHO weighted**	8.59	**0.1587**	**0.2384**	8.54	**0.0056**	**0.0038**	8.34	0.0556
	LSS ERR		0.1653	0.2166		0.0056	0.0041		
	LSS EAR		0.1521	0.2602		0.0057	0.0036		
LBR (x10^{-2})			**40.71**	**29.09**		**0.58**	**0.41**		**5.54**

Table 36. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) for all solid cancers, leukaemia and breast cancer for a 20-year old age-at-exposure

Region	Model	All solid cancers			Leukaemia			Breast cancer	
		Total lifetime exposure (mSv)	LAR (x 10^{-2}) (males)	LAR (x 10^{-2}) (females)	Total lifetime exposure (mSv)	LAR (x 10^{-2}) (males)	LAR (x 10^{-2}) (females)	Total lifetime exposure (mSv)	LAR (x 10^{-2}) (females)
①	**WHO weighted**	23.34	**0.3943**	**0.5909**	22.40	**0.0146**	**0.0094**	24.17	0.1288
	LSS ERR		0.4214	0.5540		0.0139	0.0093		
	LSS EAR		0.3672	0.6279		0.0153	0.0095		
②	**WHO weighted**	13.47	**0.2246**	**0.3364**	12.87	**0.0082**	**0.0053**	13.76	0.0723
	LSS ERR		0.2403	0.3155		0.0078	0.0052		
	LSS EAR		0.2088	0.3572		0.0086	0.0053		
③	**WHO weighted**	5.81	**0.0931**	**0.1391**	5.45	**0.0034**	**0.0022**	5.59	0.0290
	LSS ERR		0.1001	0.1308		0.0032	0.0021		
	LSS EAR		0.0861	0.1474		0.0035	0.0022		
④	**WHO weighted**	9.56	**0.1363**	**0.2024**	9.13	**0.0052**	**0.0034**	9.71	0.0402
	LSS ERR		0.1487	0.1916		0.0049	0.0033		
	LSS EAR		0.1240	0.2132		0.0055	0.0034		
⑤ to ⑨	**WHO weighted**	8.11	**0.1155**	**0.1715**	7.71	**0.0044**	**0.0028**	8.15	0.0337
	LSS ERR		0.1260	0.1624		0.0041	0.0028		
	LSS EAR		0.1051	0.1807		0.0046	0.0029		
LBR (x10^{-2})			**40.74**	**29.07**		**0.57**	**0.40**		**5.55**

Table 37. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) for thyroid cancer for a one-year old age-at-exposure

Region	Model	Thyroid		
		Total lifetime exposure (mSv)	LAR (x 10^{-2}) (males)	LAR (x 10^{-2}) (females)
①	**WHO weighted**	122.34	**0.1178**	**0.5241**
	LSS ERR		0.0588	0.3033
	LSS EAR		0.1783	0.7510
②	**WHO weighted**	74.12	**0.0712**	**0.3170**
	LSS ERR		0.0356	0.1835
	LSS EAR		0.1078	0.4542
③	**WHO weighted**	48.52	**0.0465**	**0.2070**
	LSS ERR		0.0233	0.1199
	LSS EAR		0.0704	0.2966
④	**WHO weighted**	47.93	**0.0436**	**0.1942**
	LSS ERR		0.0220	0.1134
	LSS EAR		0.0658	0.2774
⑤ to ⑩	**WHO weighted**	43.13	**0.0397**	**0.1769**
	LSS ERR		0.0200	0.1030
	LSS EAR		0.0600	0.2527
⑪ to ⑭	**WHO weighted**	37.18	**0.0347**	**0.1543**
	LSS ERR		0.0174	0.0897
	LSS EAR		0.0524	0.2206
Rest of Fukushima prefecture (less affected)	**WHO weighted**	31.92	**0.0304**	**0.1351**
	LSS ERR		0.0152	0.0783
	LSS EAR		0.0459	0.1935
LBR (x10^{-2})			**0.21**	**0.77**

Table 38. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) for thyroid cancer for a 10-year old age-at-exposure

Region	Model	Thyroid		
		Total lifetime exposure (mSv)	LAR ($\times 10^{-2}$) (males)	LAR ($\times 10^{-2}$) (females)
①	**WHO weighted**	96.06	**0.0540**	**0.2454**
	LSS ERR		0.0320	0.1693
	LSS EAR		0.0765	0.3237
②	**WHO weighted**	52.45	**0.0294**	**0.1335**
	LSS ERR		0.0174	0.0922
	LSS EAR		0.0416	0.1761
③	**WHO weighted**	28.32	**0.0158**	**0.0716**
	LSS ERR		0.0094	0.0495
	LSS EAR		0.0223	0.0944
④	**WHO weighted**	29.88	**0.0154**	**0.0701**
	LSS ERR		0.0093	0.0488
	LSS EAR		0.0217	0.0920
⑤ to ⑩	**WHO weighted**	25.65	**0.0134**	**0.0610**
	LSS ERR		0.0080	0.0424
	LSS EAR		0.0189	0.0802
⑪ to ⑭	**WHO weighted**	20.41	**0.0108**	**0.0492**
	LSS ERR		0.0065	0.0341
	LSS EAR		0.0153	0.0647
Rest of Fukushima prefecture (less affected)	**WHO weighted**	15.85	**0.0086**	**0.0393**
	LSS ERR		0.0051	0.0272
	LSS EAR		0.0122	0.0518
LBR ($\times 10^{-2}$)			**0.21**	**0.77**

Table 39. Lifetime attributable risk (LAR) and lifetime baseline risk (LBR) for thyroid cancer for a 20-year old age-at-exposure

Region	Model	Thyroid		
		Total lifetime exposure (mSv)	LAR (x 10^{-2}) (males)	LAR (x 10^{-2}) (females)
①	**WHO weighted**	64.04	**0.0192**	**0.0881**
	LSS ERR		0.0136	0.0703
	LSS EAR		0.0249	0.1065
②	**WHO weighted**	35.19	**0.0105**	**0.0481**
	LSS ERR		0.0074	0.0384
	LSS EAR		0.0136	0.0581
③	**WHO weighted**	18.22	**0.0053**	**0.0246**
	LSS ERR		0.0038	0.0196
	LSS EAR		0.0069	0.0297
④	**WHO weighted**	20.39	**0.0054**	**0.0247**
	LSS ERR		0.0039	0.0199
	LSS EAR		0.0069	0.0297
⑤ to ⑩	**WHO weighted**	17.09	**0.0046**	**0.0211**
	LSS ERR		0.0033	0.0170
	LSS EAR		0.0059	0.0253
⑪ to ⑭	**WHO weighted**	13.02	**0.0035**	**0.0163**
	LSS ERR		0.0025	0.0131
	LSS EAR		0.0046	0.0196
Rest of Fukushima prefecture (less affected)	**WHO weighted**	9.43	**0.0027**	**0.0122**
	LSS ERR		0.0019	0.0098
	LSS EAR		0.0034	0.0147
LBR (x10^{-2})			**0.21**	**0.76**

Table 40. Cumulative attributable risk (AR_{15}) and cumulative baseline risk (BR_{15}) for all solid cancers, leukaemia and breast for a one-year old age-at-exposure

Region	Model	All solid cancers			Leukaemia			Breast cancer	
		Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (males)	AR_{15} (x 10^{-2}) (females)	Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (males)	AR_{15} (x 10^{-2}) (females)	Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (females)
①	**WHO weighted**	26.65	**0.0110**	**0.0191**	26.04	**0.0252**	**0.0172**	27.47	*
	LSS ERR		0.0182	0.0330		0.0417	0.0291		
	LSS EAR		0.0037	0.0052		0.0088	0.0052		
②	**WHO weighted**	15.45	**0.0063**	**0.0110**	15.01	**0.0143**	**0.0097**	15.83	*
	LSS ERR		0.0105	0.0189		0.0235	0.0164		
	LSS EAR		0.0022	0.0030		0.0050	0.0030		
③	**WHO weighted**	5.77	**0.0023**	**0.0040**	5.27	**0.0048**	**0.0033**	5.51	*
	LSS ERR		0.0038	0.0068		0.0079	0.0055		
	LSS EAR		0.0008	0.0011		0.0017	0.0010		
④	**WHO weighted**	8.54	**0.0029**	**0.0049**	8.10	**0.0059**	**0.0040**	8.62	*
	LSS ERR		0.0047	0.0084		0.0093	0.0065		
	LSS EAR		0.0010	0.0014		0.0024	0.0014		
⑤ to ⑨	**WHO weighted**	7.15	**0.0024**	**0.0041**	6.71	**0.0049**	**0.0033**	7.13	*
	LSS ERR		0.0039	0.0071		0.0077	0.0054		
	LSS EAR		0.0009	0.0012		0.0020	0.0012		
BR_{15} (x10^{-2})			**0.08**	**0.08**		**0.03**	**0.03**		**0.00**

* The HRA Expert Group considered that the minimum attained age for breast cancer risk expression is 20 years. Note that the baseline female breast cancer rates in Japan used in the present assessment indicate no baseline incidence before age 20 (i.e. rate = zero)

Table 41. Cumulative attributable risk (AR_{15}) and cumulative baseline risk (BR_{15}) for all solid cancers, leukaemia and breast cancer for a 10-year old age-at-exposure

Region	Model	All solid cancers			Leukaemia			Breast cancer	
		Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (males)	AR_{15} (x 10^{-2}) (females)	Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (males)	AR_{15} (x 10^{-2}) (females)	Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (females)
①	**WHO weighted**	25.8	**0.0107**	**0.0183**	25.94	**0.0067**	**0.0049**	25.56	0.0049
	LSS ERR		0.0105	0.0215		0.0046	0.0046		
	LSS EAR		0.0108	0.0151		0.0088	0.0052		
②	**WHO weighted**	14.35	**0.0059**	**0.0101**	14.37	**0.0036**	**0.0027**	14.13	0.0027
	LSS ERR		0.0058	0.0118		0.0025	0.0025		
	LSS EAR		0.0060	0.0083		0.0048	0.0028		
③	**WHO weighted**	5.56	**0.0022**	**0.0038**	5.38	**0.0013**	**0.0010**	5.22	0.0010
	LSS ERR		0.0022	0.0044		0.0009	0.0009		
	LSS EAR		0.0022	0.0031		0.0017	0.0010		
④	**WHO weighted**	8.13	**0.0028**	**0.0048**	8.02	**0.0018**	**0.0013**	7.94	0.0012
	LSS ERR		0.0027	0.0056		0.0012	0.0012		
	LSS EAR		0.0029	0.0040		0.0024	0.0014		
⑤ to ⑨	**WHO weighted**	6.82	**0.0024**	**0.0040**	6.69	**0.0015**	**0.0011**	6.61	0.0010
	LSS ERR		0.0023	0.0047		0.0010	0.0010		
	LSS EAR		0.0024	0.0034		0.0020	0.0012		
BR_{15} (x10^{-2})			**0.13**	**0.16**		**0.02**	**0.02**		**0.01**

Table 42. Cumulative attributable risk (AR_{15}) and cumulative baseline risk (BR_{15}) for all solid cancers, leukaemia and breast cancer for a 20-year old age-at-exposure

Region	Model	All solid cancers			Leukaemia			Breast cancer	
		Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (males)	AR_{15} (x 10^{-2}) (females)	Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (males)	AR_{15} (x 10^{-2}) (females)	Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (females)
①	**WHO weighted**	22.86	**0.0150**	**0.0315**	21.95	**0.0051**	**0.0030**	23.78	0.0087
	LSS ERR		0.0114	0.0370		0.0028	0.0017		
	LSS EAR		0.0187	0.0260		0.0073	0.0044		
②	**WHO weighted**	12.99	**0.0085**	**0.0177**	12.42	**0.0028**	**0.0017**	13.37	0.0049
	LSS ERR		0.0064	0.0208		0.0015	0.0009		
	LSS EAR		0.0105	0.0146		0.0041	0.0024		
③	**WHO weighted**	5.33	**0.0033**	**0.0070**	5.00	**0.0011**	**0.0007**	5.2	0.0018
	LSS ERR		0.0025	0.0083		0.0006	0.0004		
	LSS EAR		0.0042	0.0058		0.0016	0.0009		
④	**WHO weighted**	7.59	**0.0042**	**0.0088**	7.16	**0.0014**	**0.0009**	7.7	0.0023
	LSS ERR		0.0032	0.0104		0.0008	0.0005		
	LSS EAR		0.0052	0.0073		0.0021	0.0013		
⑤ to ⑨	**WHO weighted**	6.44	**0.0035**	**0.0075**	6.05	**0.0012**	**0.0007**	6.46	0.0020
	LSS ERR		0.0027	0.0088		0.0006	0.0004		
	LSS EAR		0.0044	0.0061		0.0018	0.0011		
BR_{15} (x10^{-2})			**0.36**	**0.67**		**0.04**	**0.02**		**0.19**

Table 43. Cumulative attributable risk (AR_{15}) and cumulative baseline risk (BR_{15}) for thyroid cancer for a one-year old age-at-exposure

Region	Model	Thyroid		
		Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (males)	AR_{15} (x 10^{-2}) (females)
①	**WHO weighted**	122.07	**0.0090**	**0.0325**
	LSS ERR		0.0036	0.0104
	LSS EAR		0.0148	0.0564
②	**WHO weighted**	73.85	**0.0054**	**0.0197**
	LSS ERR		0.0022	0.0063
	LSS EAR		0.0090	0.0341
③	**WHO weighted**	48.25	**0.0035**	**0.0128**
	LSS ERR		0.0014	0.0041
	LSS EAR		0.0059	0.0222
④	**WHO weighted**	45.66	**0.0033**	**0.0118**
	LSS ERR		0.0013	0.0038
	LSS EAR		0.0054	0.0204
⑤ to ⑩	**WHO weighted**	41.49	**0.0030**	**0.0108**
	LSS ERR		0.0012	0.0035
	LSS EAR		0.0049	0.0187
⑪ to ⑭	**WHO weighted**	36.12	**0.0026**	**0.0095**
	LSS ERR		0.0010	0.0030
	LSS EAR		0.0043	0.0164
Rest of Fukushima prefecture (less affected)	**WHO weighted**	31.53	**0.0023**	**0.0084**
	LSS ERR		0.0009	0.0027
	LSS EAR		0.0038	0.0145
BR_{15} (x10^{-2})			**0.0014**	**0.0040**

Table 44. Cumulative attributable risk (AR_{15}) and cumulative baseline risk (BR_{15}) for thyroid cancer for a 10-year old age-at-exposure

Region	Model	Thyroid		
		Total lifetime exposure (mSv)	AR_{15} (x 10^{-2}) (males)	AR_{15} (x 10^{-2}) (females)
①	**WHO weighted**	95.74	**0.0066**	**0.0302**
	LSS ERR		0.0035	0.0236
	LSS EAR		0.0098	0.0373
②	**WHO weighted**	52.13	**0.0036**	**0.0164**
	LSS ERR		0.0019	0.0129
	LSS EAR		0.0053	0.0203
③	**WHO weighted**	28.00	**0.0019**	**0.0088**
	LSS ERR		0.0010	0.0069
	LSS EAR		0.0029	0.0109
④	**WHO weighted**	27.8	**0.0018**	**0.0084**
	LSS ERR		0.0010	0.0066
	LSS EAR		0.0027	0.0103
⑤ to ⑩	**WHO weighted**	24.13	**0.0016**	**0.0073**
	LSS ERR		0.0008	0.0058
	LSS EAR		0.0024	0.0090
⑪ to ⑭	**WHO weighted**	19.40	**0.0013**	**0.0059**
	LSS ERR		0.0007	0.0047
	LSS EAR		0.0019	0.0073
Rest of Fukushima prefecture (less affected)	**WHO weighted**	15.42	**0.0010**	**0.0048**
	LSS ERR		0.0006	0.0038
	LSS EAR		0.0016	0.0059
BR_{15} (x10^{-2})			**0.01**	**0.03**

Table 45. Cumulative attributable risk (AR_{15}) and cumulative baseline risk (BR_{15}) for thyroid cancer for a 20-year old age-at-exposure

Region	Model	Thyroid		
		Total lifetime exposure (mSv)	AR_{15} ($\times 10^{-2}$) (males)	AR_{15} ($\times 10^{-2}$) (females)
①	**WHO weighted**	63.65	**0.0035**	**0.0159**
	LSS ERR		0.0025	0.0148
	LSS EAR		0.0045	0.0171
②	**WHO weighted**	34.80	**0.0019**	**0.0087**
	LSS ERR		0.0014	0.0081
	LSS EAR		0.0024	0.0093
③	**WHO weighted**	17.83	**0.0010**	**0.0044**
	LSS ERR		0.0007	0.0041
	LSS EAR		0.0012	0.0047
④	**WHO weighted**	18.38	**0.0009**	**0.0043**
	LSS ERR		0.0007	0.0040
	LSS EAR		0.0012	0.0046
⑤ to ⑩	**WHO weighted**	15.59	**0.0008**	**0.0036**
	LSS ERR		0.0006	0.0034
	LSS EAR		0.0010	0.0039
⑪ to ⑭	**WHO weighted**	12.00	**0.0006**	**0.0028**
	LSS ERR		0.0005	0.0026
	LSS EAR		0.0008	0.0030
Rest of Fukushima prefecture (less affected)	**WHO weighted**	8.94	**0.0005**	**0.0022**
	LSS ERR		0.0003	0.0020
	LSS EAR		0.0006	0.0023
BR_{15} ($\times 10^{-2}$)			**0.02**	**0.07**

Annex K. Lifetime attributable risk (LAR) in workers based on first-year doses

The first year dose presented in this annex refers to the organ doses in colon (for all solid cancers), bone marrow (for leukaemia), and thyroid (for thyroid cancer).

Table 46. Lifetime attributable risk (LAR) due to radiation exposure and lifetime baseline risk (LBR) for all solid cancers, leukaemia and thyroid cancer based on first year exposure for male workers. The risk values are calculated for 20, 40 and 60 age-at-exposure

Scenario	Model	All solid cancers			
		First year dose (mSv)	LAR (x 10^{-2}), 20y	LAR (x 10^{-2}), 40y	LAR (x 10^{-2}), 60y
1	**WHO weighted**	5	**0.0859**	**0.0505**	**0.0231**
	LSS ERR		0.0917	0.0605	0.0309
	LSS EAR		0.0802	0.0404	0.0152
2	**WHO weighted**	24.022	**0.4128**	**0.2424**	**0.1108**
	LSS ERR		0.4403	0.2905	0.1483
	LSS EAR		0.3853	0.1943	0.0733
3	**WHO weighted**	200	**3.4370**	**2.0182**	**0.9221**
	LSS ERR		3.6661	2.4189	1.2343
	LSS EAR		3.2076	1.6173	0.6099
4	**WHO weighted**	103.24	**1.7742**	**1.0418**	**0.4760**
	LSS ERR		1.8925	1.2486	0.6372
	LSS EAR		1.6558	1.8349	0.3148
LBR (x10^{-2})			**40.74**	**40.90**	**38.10**

Leukaemia				Thyroid cancer			
First year dose (mSv)	LAR (x 10^{-2}), 20y	LAR (x 10^{-2}), 40y	LAR (x 10^{-2}), 60y	First year dose (mSv)	LAR (x 10^{-2}), 20y	LAR (x 10^{-2}), 40y	LAR (x 10^{-2}), 60y
	0.0032	**0.0024**	**0.0016**		**0.0015**	**0.0004**	**0.0001**
5	0.0031	0.0022	0.0015	5	0.0011	0.0004	0.0001
	0.0034	0.0027	0.0017		0.0020	0.0004	0.0001
	0.0159	**0.0120**	**0.0081**		**0.0416**	**0.0107**	**0.0022**
24.03	0.0151	0.0110	0.0076	138	0.0294	0.0102	0.0027
	0.0166	0.0130	0.0085		0.0541	0.0113	0.0018
	0.1570	**0.1189**	**0.0797**		**0.0603**	**0.0156**	**0.0032**
200	0.1512	0.1101	0.0756	200	0.0427	0.0148	0.0039
	0.1627	0.1277	0.0838		0.0784	0.0164	0.0026
	0.0747	**0.0566**	**0.0379**		**3.5584**	**0.9184**	**0.1907**
104.26	0.0716	0.0521	0.0358	11802	2.5176	0.8717	0.2298
	0.0779	0.0611	0.0401		4.6280	0.9700	0.1523
	0.57	**0.52**	**0.44**		**0.21**	**0.19**	**0.14**

Table 47. Cumulative attributable risk over 15 years after exposure (AR_{15}) due to radiation exposure and cumulative baseline risk over 15 years after exposure (BR_{15}) for all solid cancers, leukaemia and thyroid cancer based on first year exposure for male workers. The risk values are calculated for 20, 40 and 60 age-at-exposure

Scenario	Model	All solid cancers			
		First year dose (mSv)	LAR (x 10^{-2}), 20y	LAR (x 10^{-2}), 40y	LAR (x 10^{-2}), 60y
1	**WHO weighted**	5	**0.0033**	**0.0080**	**0.0126**
	LSS ERR		0.0025	0.0082	0.0173
	LSS EAR		0.0041	0.0077	0.0080
2	**WHO weighted**	24.022	**0.0160**	**0.0384**	**0.0607**
	LSS ERR		0.0122	0.0396	0.0831
	LSS EAR		0.0199	0.0371	0.0384
3	**WHO weighted**	200	**0.1335**	**0.3193**	**0.5055**
	LSS ERR		0.1015	0.3298	0.6915
	LSS EAR		0.1655	0.3089	0.3194
4	**WHO weighted**	103.24	**0.0689**	**0.1648**	**0.2609**
	LSS ERR		0.0524	0.1703	0.3570
	LSS EAR		0.0854	0.1594	0.1649
BR_{15} (x10^{-2})			**0.36**	**3.71**	**21.03**

Leukaemia				Thyroid cancer			
First year dose (mSv)	LAR (x 10^{-2}), 20y	LAR (x 10^{-2}), 40y	LAR (x 10^{-2}), 60y	First year dose (mSv)	LAR (x 10^{-2}), 20y	LAR (x 10^{-2}), 40y	LAR (x 10^{-2}), 60y
	0.0011	**0.0011**	**0.0011**		**0.0003**	**0.0001**	**0.0001**
5	0.0006	0.0005	0.0009	5	0.0002	0.0001	0.0001
	0.0017	0.0016	0.0014		0.0004	0.0001	0.0000
	0.0056	**0.0053**	**0.0056**		**0.0076**	**0.0036**	**0.0015**
24.03	0.0031	0.0026	0.0043	138	0.0055	0.0034	0.0018
	0.0081	0.0080	0.0070		0.0098	0.0038	0.0011
	0.0552	**0.0523**	**0.0556**		**0.0110**	**0.0052**	**0.0021**
200	0.0308	0.0265	0.0430	200	0.0080	0.0050	0.0026
	0.0797	0.0782	0.0683		0.0142	0.0055	0.0016
	0.0264	**0.0250**	**0.0265**		**0.6505**	**0.3086**	**0.1245**
104.26	0.0146	0.0125	0.0204	11802	0.4726	0.2946	0.1526
	0.0381	0.0374	0.0327		0.8371	0.3246	0.0968
	0.04	**0.08**	**0.23**		**0.02**	**0.05**	**0.09**

Annex L. Baseline cancer incidence data

Table 48. Comparison between the cumulative baseline incidence up-to 15 years after exposure and the lifetime baseline incidence (both expressed as %) for infants, children and young adults of both sexes

Cancer site	1 year infant				10 years child			
	up-to 15 years after exposure		up-to 89 years old		up-to 15 years after exposure		up-to 89 years old	
	male	female	male	female	male	female	male	female
Leukaemia	0.03	0.03	0.60	0.43	0.02	0.02	0.58	0.41
Thyroid	0.0014	0.0040	0.21	0.77	0.01	0.03	0.21	0.77
Breast	–	0.0003	–	5.53	–	0.01	–	5.54
All solid cancers	0.08	0.08	40.6	29.04	0.13	0.16	40.71	29.09

Note: These numbers are derived from 2004 cancer incidence data for Japan (*104*)

Table 49. Comparison between the cumulative baseline incidence up-to 15 years after the exposure and the lifetime baseline incidence (both expressed as %) for male adults of 20y, 40y and 60y

Cancer site	60 years old		40 years old	
	up-to 15 years after exposure	up-to 89 years old	up-to 15 years after exposure	up-to 89 years old
Leukaemia	0.23	0.44	0.08	0.52
Thyroid	0.09	0.14	0.05	0.19
All solid cancers	21.03	38.10	3.71	40.90

Note: These numbers are derived from 2004 cancer incidence data for Japan (*104*)

20 years adult			
up-to 15 years after exposure		up-to 89 years old	
male	female	male	female
0.04	0.02	0.57	0.40
0.02	0.07	0.21	0.76
–	0.19	–	5.55
0.36	0.67	40.74	29.0

20 years old	
up-to 15 years after exposure	up-to 89 years old
0.04	0.57
0.02	0.21
0.36	40.74

HEALTH RISK ASSESSMENT

The earthquake and tsunami in Japan on 11 March 2011 led to releases of radioactive material into the environment from the Fukushima Daiichi nuclear power plant. This health risk assessment is intended to give an indication of the potential public health implications of the accident to support the identification of needs and priorities for public health actions. This report, based on a preliminary dose estimation published by WHO in May 2012, represents the first international effort to assess the radiation health risks from this accident at the global level.

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