Annex X of Technical Volume 4
RADIATION AND HEALTH EFFECTS AND INFERRING RADIATION RISKS FROM THE FUKUSHIMA DAIICHI ACCIDENT

Anxieties about the risk of harm from radiation are often out of proportion to the actual likelihood of harm. Therefore, in order to deal sensibly with situations involving exposure to radiation, it is important to clarify what is known and what is not known about radiation and health effects.

This annex provides a general qualitative overview of what is known about radiation-induced health effects, followed by a more detailed exploration of the quantitative inferences that may be drawn from past analyses of the relationships between radiation exposure and risk.

X–1. OVERVIEW: HEALTH EFFECTS OF EXPOSURE TO RADIATION

Humans have always been exposed to ionizing radiation. It is present everywhere in the environment, and indeed all living things have evolved in the presence of radiation. Among other sources, it comes in the form of cosmic rays from outer space and as gamma rays from naturally occurring radioactive elements in rocks and soils. This natural background radiation leads to very low doses, except in a few regions of the world where doses are markedly higher due to the local geology. At these levels of exposure to external radiation, epidemiological studies have not demonstrated a causal relationship between radiation exposure and health effects, but there is evidence of a relationship between lung cancer and lifetime exposure to radon at high naturally occurring concentrations.

There is, however, evidence of an increase in the incidence of some health effects arising from radiation exposure at doses that greatly exceed background levels. In some cases, for high doses, physical effects or symptoms may be observed in individuals soon after exposure. In other situations, the effect may be evident only as an increased incidence of disease (notably cancer) in a population, and it may not be possible to tell whether an individual case of the disease is attributable to radiation. The confidence with which relationships between dose and health effects can be postulated depends upon the many factors, including the level of dose to which the population is exposed. To assist in a better understanding, it is useful to consider four categories of dose which each have broad characteristic properties. Table X–1 is based on a classification suggested by the ICRP [X–1]. Radiation doses are given in gray (Gy), which is a measure of the energy deposited in tissue when radiation is absorbed. The terms ‘high dose’, ‘moderate dose’, ‘low dose’ and ‘very low dose’ are used in this annex in the sense given in the table. There is no sharp division between these categories, but they serve as a general guide to relating effects to doses.

Exposure to **high doses** of radiation (greater than 1 Gy) is extremely rare, except in radiotherapy where radiation is deliberately used to destroy cancer cells and is directed at the malignant tissue. If non-medical high doses should occur — either accidentally or as a result of a malicious act — they will likely persist for a relatively short time as their existence will become evident and avoiding action will be taken. At these levels of exposure, ionizing radiation can cause sufficient physical damage to tissues that the effects are clearly seen in the person exposed — for example: as erythema (reddening of the skin), tissue burns or organ malfunction. Some of these effects become evident within days or a few weeks; others may not be clinically observed until months or even years later, although there may be earlier pathological evidence of damage, for example from microscopic examination of tissue samples. Such effects are known as ‘tissue reactions’ or ‘deterministic effects’ and they are not observed at low doses or very low doses.

TABLE X–1. CATEGORIES OF RADIATION EXPOSURE BASED ON ICRP PUBLICATION 96 [X–2]

Exposure category	Dose range[a]	Examples[b]	Effects on individuals	Effects for an exposed population
High dose	Greater than 1 Gy	Severe accidents involving intense radiation sources; Radiotherapy	Nausea and vomiting; tissue reactions; additional lifetime risk of cancer (of about 10% or more); above ~5 Gy: organ failure; death	Observable increase in the incidence of cancer
Moderate dose	100 mGy to 1 Gy	Accidents involving commonly used industrial sources; lifetime occupational dose for a few radiation workers	Nausea and vomiting possible; mild bone marrow depression; additional lifetime risk of cancer (of a few %)	Probable observable increase in the incidence of cancer if the exposed group is large enough (~1000 people or more)
Low dose	10 to 100 mGy	Lifetime exposure to cosmic rays on the Earth's surface; whole body CT scan.	No prompt effects; possible additional lifetime risk of cancer (less than 1%)	Possible observable increase in the incidence of cancer if the exposed group is very large (~100 000 people or more)
Very low dose	Less than 10 mGy	Most diagnostic radiology procedures; annual dose from natural background radiation	No prompt effects; extremely small hypothetical additional lifetime risk of cancer (less than 0.1%)	No observable increase in the incidence of cancer

[a] Absorbed dose for 'low LET' radiation: for example, for gamma rays and beta radiation (see text).
[b] Some of these are examples of acute exposures (delivered over a short time), others are protracted. The same total dose delivered over different periods may have different effects.

For **moderate doses** (100 mGy to 1 Gy), there is a possibility that some kinds of tissue reactions may occur, especially if doses near the upper end of the dose range are repeated leading to a high cumulative dose. One example is the formation of cataracts in the eye as a result of exposure to radiation. However, generally the harm to health is different and is expressed as a likelihood of developing a deleterious effect — primarily cancer — in the future. If a population of individuals is exposed — all to the same moderate dose — there is no way of knowing which of them, if any, will be affected. Because of this random, statistical nature of the effect it is called a 'stochastic effect'. For moderate doses received over a short period, the delay (the latency period) between exposure and effect can be several years. Different types of effect have been observed to have different minimum latency periods [X–3].

There is strong epidemiological evidence of a relationship between high and moderate radiation doses and the frequency of observed effects (such as cancer) [X–4]. For some types of cancer, the relationship in the study population is roughly linear: the risk (likelihood) being proportional to dose. It is difficult, however, to infer the relationship between dose and effect for an individual because other factors also have a bearing. Such factors include: age at exposure, time since exposure, gender, physiological characteristics (e.g. size and weight), individual behaviour (e.g. diet and smoking history), and possible genetic predisposition. Mathematical modelling of the influence of such factors on the dose to risk relationship may be possible from epidemiological information, within bounds of uncertainty determined by the degree of information available.

The **low dose** range (10 to 100 mGy) is the most problematic with regard to understanding the consequences of exposure to radiation. Exposure at these levels is below the thresholds for tissue reactions, and the epidemiological evidence of the relationship between dose and harm from stochastic effects is generally weak. There is a plausibility argument, based on physics and biology, that quantitatively links radiation dose with the number of ensuing cellular initiation events for carcinogenesis. However, the progression of such events to a clinically significant outcome depends on many other factors. Biological defence mechanisms prevent the progression of most initiating events, and it is not certain that low doses are harmful to health.

At **very low doses** (less than 10 mGy), there is no definitive evidence of harm for exposures of short duration, either of tissue reactions or of an increased frequency of cancer in study populations. With the possible exception of exposure to radon in the home, epidemiological studies have not demonstrated a causal relationship between radiation exposure and health effects. For continuing exposures of up to 10 mGy per year, the epidemiological evidence of harm is inconclusive, even though cumulative exposures may reach a few hundreds of mGy over the long term. The exposure to natural background radiation generally falls within this range. While there is an absence of evidence of harm at very low doses, there is conversely no conclusive evidence of a threshold dose below which stochastic effects do not occur.

Because the science is unclear for low doses and very low doses, there is a need to establish a system of protection in the absence of full knowledge in order to take into account the possibility of harm. For the purposes of establishing a system of protection that would provide an appropriate level of safety in the event that there is a real risk of harm, a presumed linear relationship between dose and the probability of a stochastic effect has been adopted — called the linear, no-threshold (LNT) model.

X–2. ATTRIBUTION OF HEALTH EFFECTS TO RADIATION AND INFERENCE OF FUTURE RISK

This annex makes a necessary distinction between observed health effects that may or may not be attributed to radiation and predictions (or inference) about the likelihood of effects occurring in the future.

The term ‘attributed’ relates to the act of assigning an observed effect to a cause. If an event uniquely causes an effect, then the effect can be attributed unequivocally to that event. Death by poisoning can often be attributed to a particular poison through pathological analysis of body fluids and tissues. However, if there is more than one possible cause of an effect — with no identifying pathology — then the effect cannot be unequivocally assigned to one cause. In the context of cancer and exposure to radiation, there is no pathological identifier — no ‘biomarker’ — that connects the cancer exclusively with radiation. Attribution is then a matter of judgment about the degree of belief from the evidence available that a particular cancer is caused by radiation, or that an increase in the incidence of cancer in a population is caused by radiation rather than by some other agent. However, attribution of some non-fatal effects is, in principle, verifiable through pathology in the case of an individual, including through the observation of biomarkers such as chromosome aberration.

Knowledge of the degree to which an effect can be attributed to radiation may allow inferences to be drawn about the likelihood of such an effect occurring in the future as a consequence of a received radiation dose. Such inferences involve the concept of risk. In the context of possible harm from exposure to radiation, risk is taken here to mean the probability of a deleterious effect occurring. When applied to past events, it relates to the observed frequency of occurrence of an effect; when applied to the future, it relates to the inferred likelihood of the effect occurring. Other definitions exist, but for the purposes of this annex, risk is interpreted as a probability (mathematical likelihood). Risk therefore is a dimensionless number in the range of 0 to 1 (or a percentage between 0% and 100%), but in the context of risk arising from a particular causative agent it may be expressed as the

probability that a harmful event will occur for a given level of insult or dose delivered by that agent. It then takes on the inverse dimension of the parameters that measure the dose and is called a risk coefficient. For example, cancer risk coefficients may be expressed in terms of probability per unit of absorbed dose. Risk coefficients may be expressed in terms of a mathematical model that is dependent on relevant factors, which could include the age and gender of the persons exposed, for example.

For moderate and high doses of radiation, there is sufficient evidence from epidemiological studies to predict with a fair degree of confidence the future consequential increase in incidence of cancer in an exposed population. Statistical methods allow a band of uncertainty to be estimated within which the numerical value of the incidence of disease is expected to lie. Such predictions are often population averages — where incidence data are averaged over individual variations such as age, lifestyle, and gender.

Based on effects observed in epidemiological studies involving moderate to high doses, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) [X–3, X–5] has estimated excess lifetime mortality from all solid cancers arising from exposure to radiation, averaged over gender and all ages, to be between about 0.4% and 0.8% from an acute dose of 0.1 sievert (Sv). At this level of exposure and above, and for study populations of sufficient size, there is statistically significant evidence of harm that can be quantifiably attributed to radiation, and there is a justification for assuming a linear (LNT) relationship between dose and effect. Expressed as a risk coefficient, this leads to a value of between about 4×10^{-2} Sv^{-1} and 8×10^{-2} Sv^{-1}. This is a notional risk coefficient, and for low doses and very low doses it is unverified.

For low doses and very low doses, there is insufficient epidemiological evidence to demonstrate an increase in incidence of cancer in exposed populations. Observed effects cannot be unequivocally attributed to radiation, partly because other causes of the effect cannot be ruled out, without the existence of an associated biomarker, and partly because the studies have insufficient statistical power to draw conclusions with confidence due to the high natural incidence of cancer and associated uncertainties. Predictions based on the application of risk coefficients at such levels of dose are intrinsically scientifically untestable with current knowledge As a result, such coefficients should not be used to estimate the absolute number of radiation-induced cancers in a population. However, such estimates may serve a role in decision-making, for example, in comparative analyses (e.g. selecting which is the preferred option from a range of possible preventative or remediation measures), and for resource allocation for health care purposes. It is necessary, in such cases, to apply methods consistently, taking account of uncertainties, and recognizing that such estimates are notional.

One common misuse of the risk coefficients estimated by UNSCEAR and ICRP is to base predictions of future casualties on calculations of ‘collective dose’. The argument, which is usually not explicit, is that if the risk is real, then it would be expected that a number of real cases would be seen in a sufficiently large exposed population. For example, if one million people were each exposed to 1 mSv, the collective dose to the group would be 1000 manSv. If the risk coefficient for cancer is 4×10^{-2} per sievert (see above), this appears to imply 40 cases. However, the risk associated with an individual exposure of 1 mSv is unknown and such an approach should not be used for the purpose of estimating numbers of casualties.

Calculating collective doses disguises the underlying lack of knowledge of the true risk. Predictions of cancer fatalities using collective dose where individual exposures are in the low dose and very low dose range are based on an unsubstantiated premise and both UNSCEAR [X–6] and ICRP [X–7] have advised against this practice. This advice remains valid even if the process used for risk estimation does not formally calculate collective dose, but simply applies a risk coefficient to large numbers of people exposed to low doses.

X–3. INFERRING RADIATION RISKS ARISING FROM THE FUKUSHIMA DAIICHI ACCIDENT

No health effects among workers or members of the public that could be diagnosed by a physician and confirmed by pathology can be attributed to exposure to radiation arising from the Fukushima Daiichi accident (Sections 4.4.3 and 4.4.4). In short, there are no discernible early health effects of radiation arising from the radioactive material released during the accident. However, it is important to consider whether there is a potential for an increased incidence of stochastic health effects, such as cancer, in the future.

As noted above, stochastic health effects are known to be observable in populations exposed to higher doses than those received following the Fukushima Daiichi accident. Such effects appear after a delay (latency period), which is different for different types of cancer. For thyroid cancer, the latency period is estimated to be four years or more; for most solid cancers it is typically longer [X–3]. So even if exposures were sufficient to infer that an increase in the incidence of cancer may occur in principle, it was too early to see any evidence at the time of preparation of this technical volume and annex (a little over four years after the accident).

Since the accident occurred, several hypothetical estimates of future cancer risks have been reported in the media, sometimes based on calculations of collective dose or its computational equivalent. Such predictions are inappropriate, as explained above. The results may have contributed to the anxiety and emotional distress experienced by the Japanese population. Despite the caution that needs to be exercised when inferring future risks, it is desirable to provide some information on the likelihood of possible effects that might occur from exposure to the generally low levels of radiation prevailing following the Fukushima Daiichi accident, in order to place the hypothetical risk of harm from radiation in context with other risks. This annex therefore provides theoretical predictions of a statistical indicator — the lifetime attributable risk fraction (LARF) — of the nominal risks and the inferred notional numbers of health effects that could be predicted from the levels of radiation doses received by emergency workers and the public, using risk models that reflect the most widely held consensus of opinion in the scientific literature. These estimates are not intended to predict any medical outcome but to provide perspective and for the purposes of decision making and resource allocation.

X–4. RADIATION RISK MODELS

In an unexposed population, the basic risk factor is the background cancer incidence rate denoted as λ_0 (the annual number of cancer cases per 100 000 population). Due to exposure to radiation, λ_0 is assumed to increase by $\delta\lambda$. The overall cancer incidence rate λ is a sum of background and radiation associated incidence rates:

$$\lambda = \lambda_0 + \delta\lambda$$

Background incidence rates for cancer at site *l* depend on the age *a*, gender *s*, and calendar time *t*; that is $\lambda_0 = \lambda_0(a,l,s,t)$ and the radiation associated increment depend on radiation dose *D*, attained age *a*, tumour site *l*, gender *s*, and the age at exposure *g*:

$$\delta\lambda = \delta\lambda(g,a,l,s,D)$$

$$\lambda(g,a,l,s,D,t) = \lambda_0(a,l,s,t) + \delta\lambda(g,a,l,s,D).$$

The age specific incidence rate λ_0 for solid cancers, all types of leukaemia and thyroid cancer in Japanese males registered in 2004 is shown in Figs (X–1, X–2, X–3).

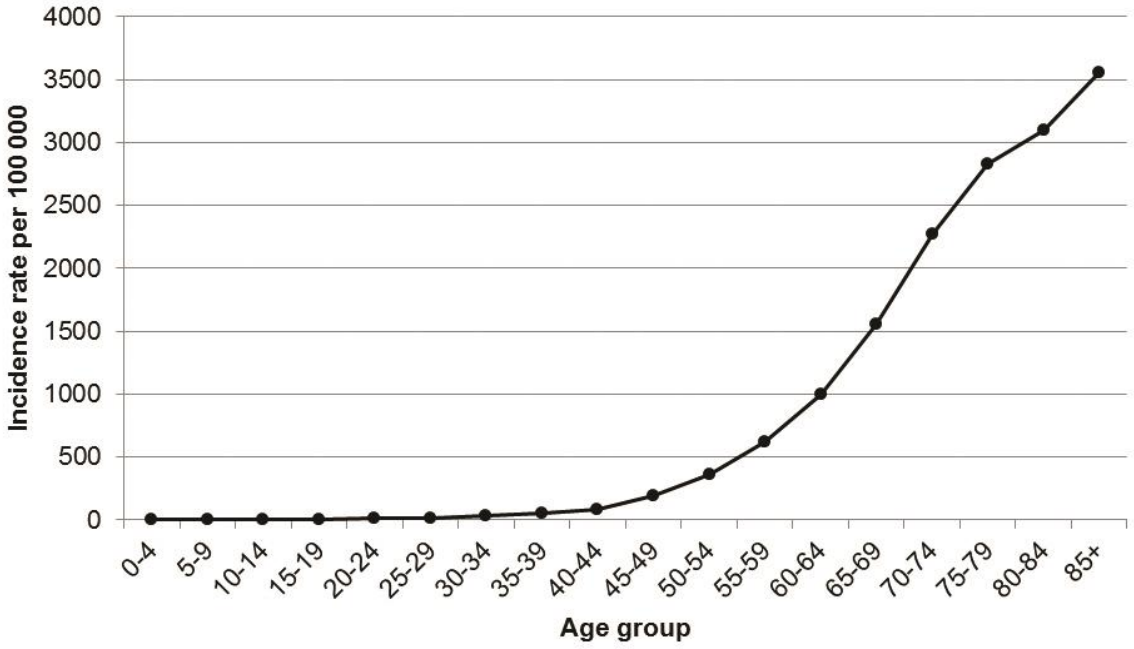

FIG. X–1. Rate of incidence of age specific solid cancers per 100 000 in Japanese males in 2004 [X–8].

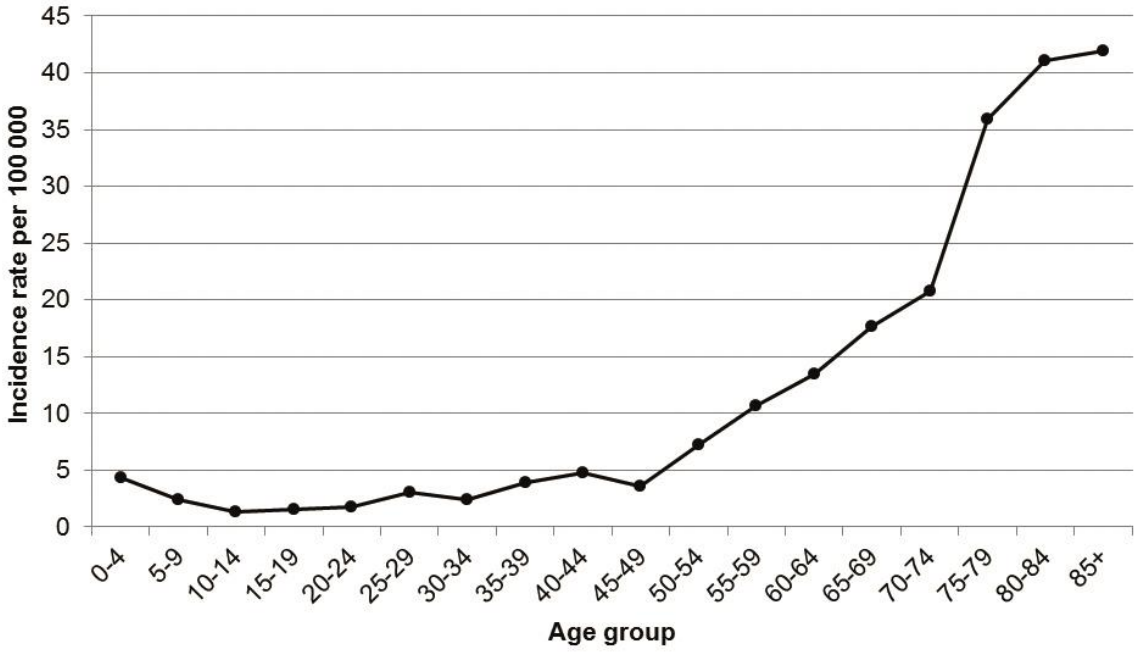

FIG. X–2. Rate of incidence of age specific leukaemia per 100 000 in Japanese males in 2004 [X–8].

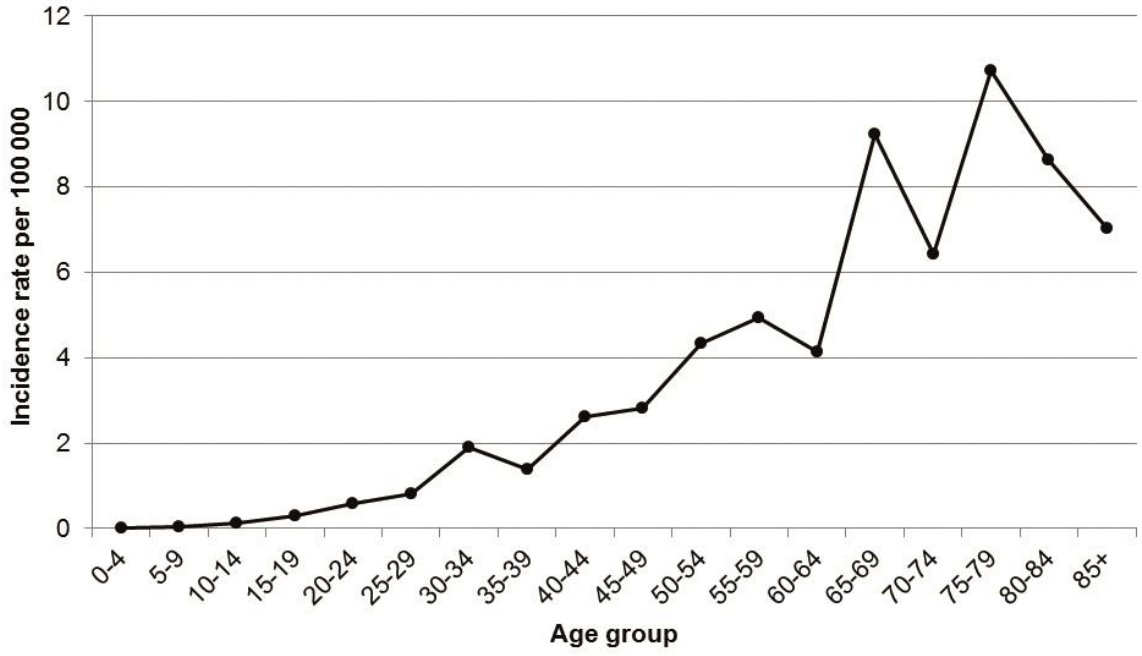

FIG. X–3. Rate of incidence of age specific thyroid cancer per 100 000 in Japanese males in 2004 [X–8].

The radiation increment $\delta\lambda$ is an excess absolute risk (EAR) for the attained age *a*, exposure age *g*, and radiation dose *D*. If the value of EAR is known, it is possible to estimate the lifetime attributable risk (LAR) (*g*, *l*, *s*, *D*) of developing cancer at the site *l* after a single exposure to dose *D* at the age *g*. LAR is the sum of values of EAR for the attained age. However, it is necessary to take into account the cancer-free survivor function, the probability that an unexposed individual will be alive and free of cancer of the site *l* from the age *g* to the age *a*. The Japanese male cancer free survival function (in 2004) for solid cancers and all types of leukaemia is given in Fig. X–4. The curve was built up from published data on cancer incidence and cancer mortality [X–8].

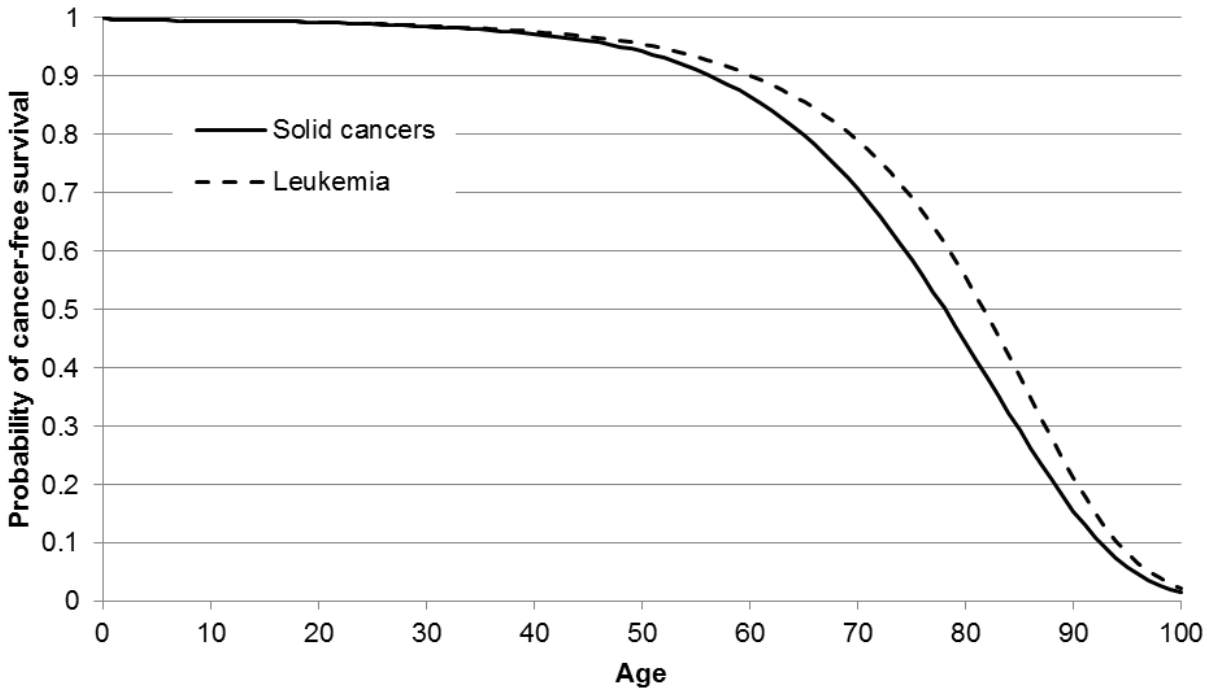

FIG. X–4. Japanese (2004 year) male disease-free survival function for solid cancers and leukaemia [X–8].

For computation of the LAR for cancer incidence rate, the following expression was used:

$$LAR(g,l,s,D) = \frac{1}{DDREF}\sum_{a=g}^{a_{\max}} S(s,l,g,a)\cdot EAR(g,a,l,s,D)$$

where *S(s,l,g,a)* is a cancer-free survivor function, and DDREF is the dose and dose-rate effectiveness factor.

In addition to the lifetime attributable risk of cancer incidence, one can use lifetime attributable risk fraction, LARF, for incidence of cancer at site *l* in males or females exposed to dose *D* at the age *g*. Mathematically, LARF is expressed as the ratio of lifetime attributable risk to the overall lifetime risk of cancer incidence:

$$LARF(g,l,s,t,D) = \frac{LAR(g,l,s,D)}{LAR(g,l,s,D) + LBR(g,l,s,t)}\cdot 100\%$$

where *LBR(g,l,s,t)* is a lifetime background risk of site-specific cancer incidence. It is calculated from the exposure age *g*. The background risk of cancer incidence rate is estimated by summing up background incidence rates with allowance for the cancer-free lifetime from the age of exposure *g*.

$$LBR(g,l,s,t) = \sum_{a=g}^{a_{\max}} \lambda_0(a,l,s,t)\cdot S(s,l,g,a)$$

where *S(s,l,g,a)* is a cancer-free survivor function.

For the purpose of assessing risks, and for comparison with the likelihood of other events, calculating the LARF is adequate and is understood to be a statistical quantity. However, several reports in the literature have expressed the likelihood of harm in terms of the number of hypothetical cancer cases. Despite the cautionary advice above, and for the sole purpose of comparing the numerical results of the modelling performed here with previously published studies, LARF has been converted to

radiation associated cancer incidence in some of the tables below. It should be remembered that for the low and very low doses associated with the Fukushima Daiichi accident, these incidence values are hypothetical.

To evaluate the statistically expected number of radiation associated cancer cases in a group, the number of people in the group *N* and LAR value should be multiplied and similarly, the number of expected baseline, non-radiation, cancer cases is formed by multiplying the *N* value and LBR.

In order to provide some perspective on the level of risk that may be inferred from the radiation exposures arising from the Fukushima Daiichi accident, two mathematical models have been used: the ICRP model [X–7] and the model described in the WHO report on health effects from the Fukushima Daiichi NPP accident [X–9].The basic features of the models are described below:

1. Both models adopt a linear dose-risk relationship for solid cancers and quadratic dose-risk relationship for all types of leukaemia.
2. Both models were based on data for Life Span Study cohort of Hiroshima and Nagasaki atomic bomb survivors.
3. The dose and dose rate effectiveness factor (DDREF)[1] is 2 in the ICRP model and 1 in the WHO model.
4. In the ICRP model, the latency period for solid cancers is 10 years, and for all types of leukaemia it is 2 years. In the WHO model for solid cancers except for thyroid and breast cancers the latency period is five years; for thyroid and breast cancers it is three and five years, respectively; and for all types of leukaemia the latency period is two years.
5. Both models use a weighted average of multiplicative and additive models. The multiplicative model derives excess relative risk is the background cancer incidence rate; the additive model derives EAR directly.
    - In the WHO model the weights of both multiplicative and additive models are 50% for all types of solid cancers excluding breast cancer. For this cancer additive model only is used.
    - In the ICRP model, weights of both multiplicative and additive models are 50% for solid cancers; for breast cancer the additive model only is used; for thyroid cancer the multiplicative model only is used; and for all types of leukaemia the additive model only is used.
6. In the WHO report the leukaemia morbidity risk is estimated using the UNSCEAR 2006 model for estimating mortality risk from leukaemia.
    - The ICRP and WHO risk models have been applied to the data available on radiation doses received by members of the public and Fukushima Daiichi NPP workers (Section 4.2). The worker group includes both employees of TEPCO and contractors exposed during the emergency and recovery phase.

X–5. INFERRING RADIATION RISKS TO WORKERS

Tables X–2 and X–3 expand on the data presented in Section 4.4.5 and include the results of calculations of LARF using the models of the ICRP and WHO. These calculations were performed using the reported data for workers doses available in May 2014. These data were updated in December 2014 to provide more recent information and the results of a reanalysis of some of the dose information for the first year. The updated information is presented in Section 4.2.1. The magnitude of the changes between the two datasets did not warrant re-running the risk models; these changes would not significantly affect the inferred risks and general conclusions presented below.

[1] The ratio between the risk or radiation detriment per unit effective dose for high doses and/or dose rates and that for low doses and dose rates.

The numbers of radiation induced cancer cases presented are not predictions of actual medical outcomes but expressions of risk as hypothetical fractions of the number of people exposed. Given the background prevalence of cancer the additional LARF from these calculations is small.

The data for each year from the application of each risk model are provided in subsequent sections.

TABLE X–2. LARF AND INFERRED NUMBER OF CANCER CASES FOR FUKUSHIMA DAIICHI NPP EMERGENCY WORKERS, CALCULATED WITH THE ICRP MODEL. SUMMARY FOLLOW-UP PERIOD FROM MARCH 2011 TO AUGUST 2013

	LARF (%)			Expected number of spontaneous cases			Hypothetical number of radiation induced cases		
	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers
TEPCO	0.13	1.37	0.15	2 916	44	2 959	3.8	0.6	4.4
Contractors	0.06	0.76	0.07	17 371	259	17 630	11	2.0	13
Total	0.07	0.85	0.08	20 286	303	20 589	15	2.6	17

TABLE X–3. LARF AND INFERRED NUMBER OF CANCER CASES FOR FUKUSHIMA DAIICHI NPP EMERGENCY WORKERS, CALCULATED WITH THE WHO MODEL. SUMMARY FOLLOW-UP PERIOD FROM MARCH 2011 TO AUGUST 2013

	LARF (%)			Expected number of spontaneous cases			Hypothetical number of radiation induced cases		
	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers
TEPCO	0.26	0.95	0.27	2 916	44	2 959	7.7	0.4	8.1
Contractors	0.13	0.49	0.14	17 371	259	17 630	23	1.3	24
Total	0.15	0.56	0.16	20 286	303	20 589	30	1.7	32

For calculations using both risk models, the LARF for all cancers is less than 1%. As a consequence, while there may be a small increase in the overall lifetime risk of cancer, it would not be discernible in a population study. For comparison, in the case of the Chernobyl accident, a discernible increase on cancer incidence occurred only with a LARF greater than 10%, about ten times the LARF calculated for the Fukushima Daiichi accident [X–10].

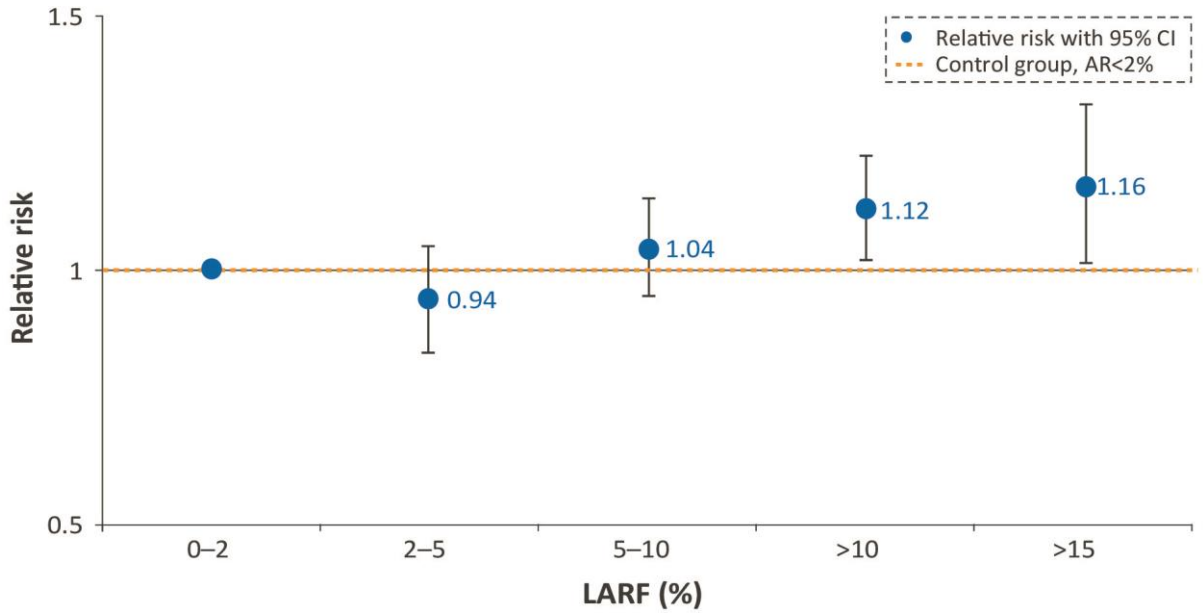

FIG. X–5. Relative risk of solid cancers among Chernobyl emergency workers as a function of LARF [X–8].

As the calculated LARF values for Fukushima Daiichi NPP workers are less than 1%, no solid cancers due to radiation exposure are expected to be observed.

X–5.1. Inferring radiation risks to workers using the ICRP Model

Tables X–4 to X–6 show the calculated lifetime attributable risk fraction (LARF), as well as the expected number of spontaneous cases and the hypothetical number of radiation-induced cases for solid cancers and all types of leukaemia for each of 3 years of follow-up. Summarized data for the whole follow-up period are given in Table X–2.

TABLE X–4. LARF AND INFERRED NUMBER OF CANCER CASES FOR FUKUSHIMA DAIICHI NPP EMERGENCY WORKERS, CALCULATED WITH THE ICRP MODEL. FOLLOW-UP PERIOD FROM MARCH 2011 TO DECEMBER 2011

	LARF (%)			Expected number of spontaneous cases			Hypothetical number of radiation induced cases		
	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers
TEPCO	0.21	2.24	0.24	1 477	22	1 499	3.1	0.5	3.6
Contractors	0.08	1.00	0.10	7 317	109	7 426	6.1	1.1	7.2
Total	0.11	1.21	0.12	8 794	131	8 925	9.3	1.6	11.0

TABLE X–5. LARF AND INFERRED NUMBER OF CANCER CASES FOR FUKUSHIMA DAIICHI NPP EMERGENCY WORKERS, CALCULATED WITH THE ICRP MODEL. FOLLOW-UP PERIOD FROM JANUARY 2012 TO DECEMBER 2012

	LARF (%)			Expected number of spontaneous cases			Hypothetical number of radiation induced cases		
	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers
TEPCO	0.06	0.59	0.06	799	12	811	0.5	0.07	0.5
Contractors	0.05	0.66	0.06	5 573	83	5 656	3.1	0.60	3.6
Total	0.06	0.65	0.06	6 373	95	6 468	3.5	0.60	4.1

TABLE X–6. LARF AND INFERRED NUMBER OF CANCER CASES FOR FUKUSHIMA DAIICHI NPP EMERGENCY WORKERS, CALCULATED WITH THE ICRP MODEL. FOLLOW-UP PERIOD FROM JANUARY 2013 TO AUGUST 2013

	LARF (%)			Expected number of spontaneous cases			Hypothetical number of radiation induced cases		
	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers
TEPCO	0.03	0.31	0.03	640	10	649	0.2	0.03	0.2
Contractors	0.04	0.49	0.05	4 480	67	4 547	1.8	0.30	2.2
Total	0.04	0.47	0.05	5 120	76	5 196	2.0	0.40	2.4

The average age-specific lifetime attributable risk fraction for solid cancers, all types of leukaemia, all cancers and thyroid cancer in emergency workers, estimated using the ICRP model, are plotted against radiation dose is given in Section 4.4.5.2, Fig. 4.4–1.

For calculation of LARF for thyroid cancer in the emergency workers dose distribution available in May 2004 was used. The inferred number of radiation-associated and background thyroid cancer cases in a specific dose group and LARF, in %, were calculated with the ICRP model. Calculated parameters are dose-response data averaged by size of an age group. Calculation results are given in Section 4.4.5.2, Table 4.4–2. The numbers of radiation induced cases presented are not predictions of actual medical outcomes but expressions of risk as hypothetical fractions of the number of people exposed.

X–5.2. Inferring radiation risks to workers using the WHO model

Calculations similar to those described above were also made using the WHO model, as shown in Tables X–7 to X–9. Summarized data for the whole follow-up period are given in Table XI-3.

TABLE X–7. LARF AND INFERRED NUMBER OF CANCER CASES FOR FUKUSHIMA DAIICHI NPP EMERGENCY WORKERS, CALCULATED WITH THE WHO MODEL. FOLLOW-UP PERIOD FROM MARCH 2011 TO DECEMBER 2011

	LARF (%)			Expected number of spontaneous cases			Hypothetical number of radiation induced cases		
	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers
TEPCO	0.43	1.55	0.44	1 477	22	1 499	6.3	0.4	6.7
Contractors	0.17	0.65	0.18	7 317	109	7 426	13.0	0.7	13.0
Total	0.22	0.81	0.22	8 794	131	8 925	19.0	1.1	20.0

TABLE X–8. LARF AND INFERRED NUMBER OF CANCER CASES FOR FUKUSHIMA DAIICHI NPP EMERGENCY WORKERS, CALCULATED WITH THE WHO MODEL. FOLLOW-UP PERIOD FROM JANUARY 2012 TO DECEMBER 2012

	LARF (%)			Expected number of spontaneous cases			Hypothetical number of radiation induced cases		
	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers
TEPCO	0.11	0.40	0.12	799	12	811	0.9	0.05	1.0
Contractors	0.11	0.43	0.12	5 573	83	5 657	6.3	0.40	6.7
Total	0.11	0.42	0.12	6 373	95	6 468	7.2	0.40	7.6

TABLE X–9. LARF AND INFERRED NUMBER OF CANCER CASES FOR FUKUSHIMA DAIICHI NPP EMERGENCY WORKERS, CALCULATED WITH WHO MODEL. FOLLOW-UP PERIOD FROM JANUARY 2013 TO AUGUST 2013

	LARF (%)			Expected number of spontaneous cases			Hypothetical number of radiation induced cases		
	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers	Solid cancers	Leukaemia	All cancers
TEPCO	0.06	0.21	0.06	640	10	649	0.4	0.02	0.4
Contractors	0.08	0.32	0.09	4 480	67	4 547	3.8	0.20	4.0
Total	0.08	0.30	0.08	5 120	76	5 196	4.2	0.20	4.4

Average age-specific lifetime attributable risk fraction for solid cancers, all types of leukaemia, all cancers and thyroid cancer in emergency workers plotted against radiation dose is given in Section 4.4.5.2, Fig. 4.4–2.

Estimating risk of thyroid cancer in each dose group was based on the distribution of equivalent thyroid dose received during 2011 by TEPCO emergency workers presented in Section 4.4.5.2, Table 4.4–3. The WHO model, as well as the ICRP model in the above section, was used for inferring

the number of radiation induced thyroid cancer, spontaneous thyroid cancer cases and LARF (%) in each dose group on the basis of this dose distribution. The numbers of radiation induced cases presented are not predictions of actual medical outcomes but expressions of risk as hypothetical fractions of the number of people exposed.

Application of the WHO model resulted in higher risk of hypothetical radiation induced cancer in Fukushima Daiichi NPP emergency workers. In particular, for TEPCO workers who participated in the clean-up work in 2011, the LARF for all cancers estimated with the WHO model was 0.22% (Table X–7) compared with 0.12% (Table X–4) with the ICRP model. The LARF values for thyroid cancers among the emergency workers on the site for which thyroid measurement data are available was estimated to be 3.39 and 1.00% using the WHO and ICRP models, respectively (see Section 4.4.5.2, Tables 4.4–2 and 4.4–3).

X–6. APPLICATION OF RISK MODELS TO THE GENERAL POPULATION

To infer risks of future stochastic radiation effects in the affected population, both the ICRP and WHO models were used, adjusting for medical and demographic data for Japan. LARF values as a function of dose for children and adolescents are shown in the following figures: males Fig. X–6 (ICRP) and Fig. X–7 (WHO); females Fig. X–8 (ICRP) and Fig. X–9 (WHO). It is seen that calculated values are different between the two models.

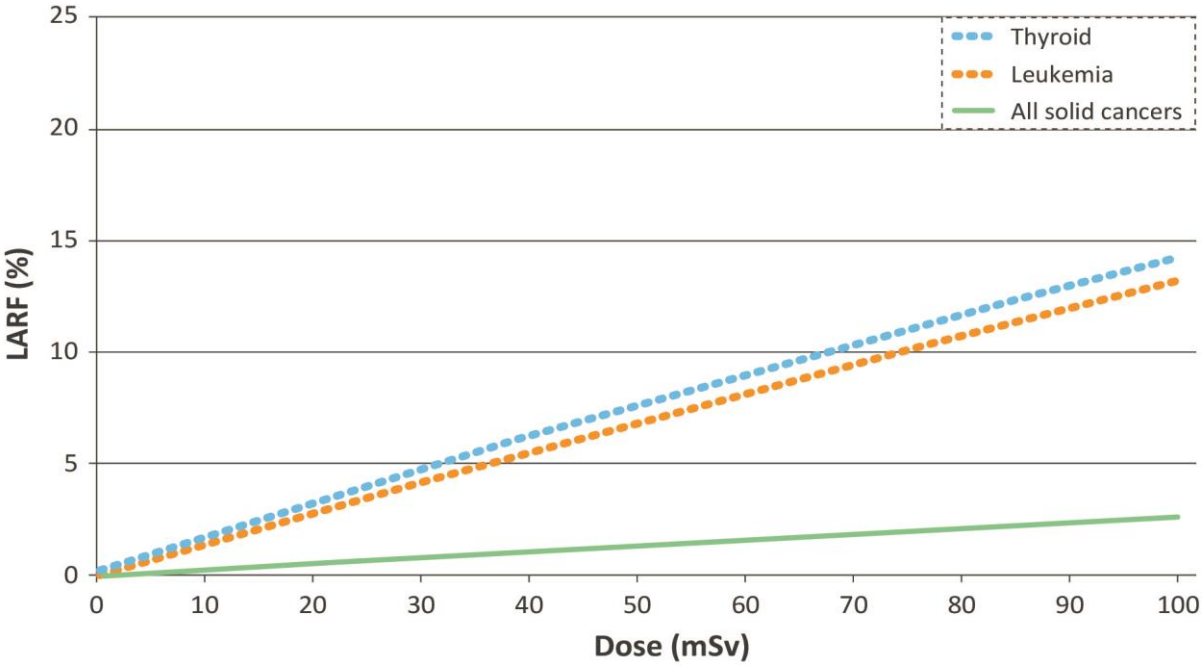

FIG. X–6. Average age specific LARF as a function of effective dose, estimated with the ICRP model (children, male) [X–8].

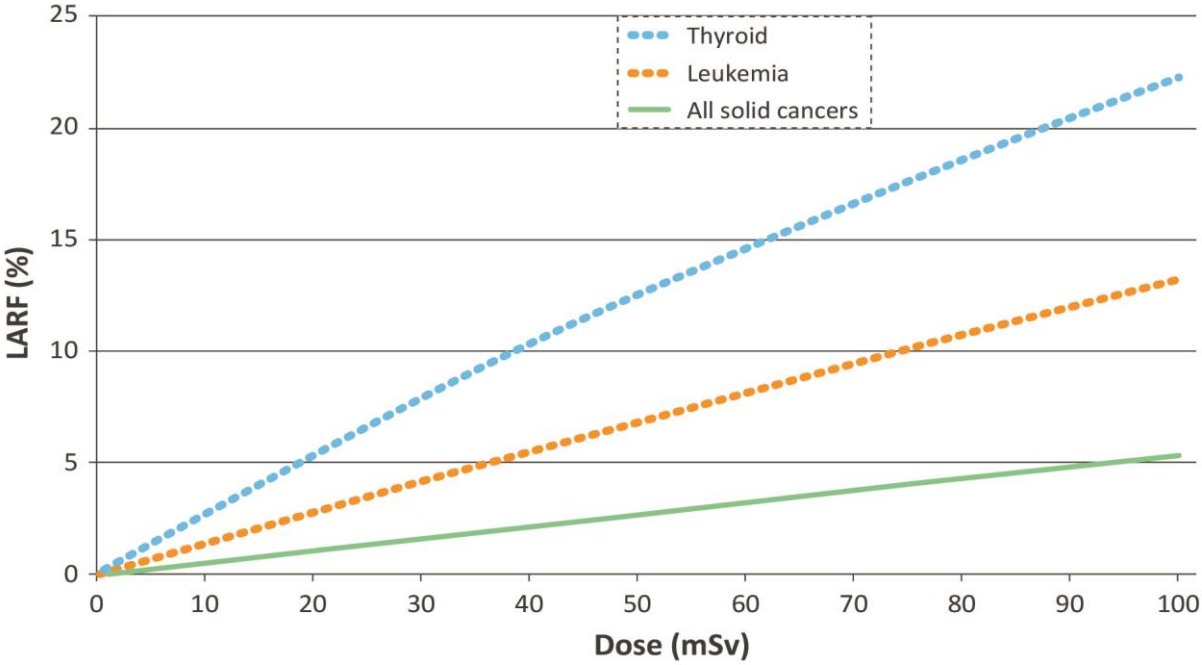

FIG. X–7. Average age specific LARF as a function of effective dose, estimated with the WHO model (children, male) [X–8].

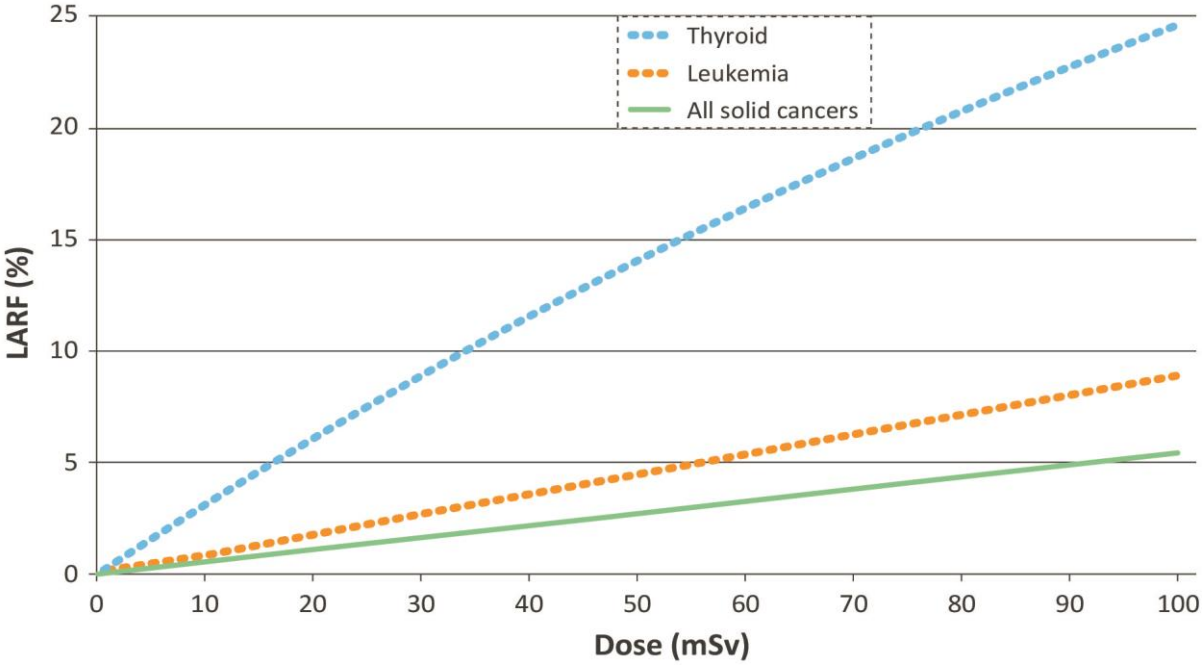

FIG. X–8. Average age specific LARF as a function of effective dose, estimated with the ICRP model (children, female) [X–8].

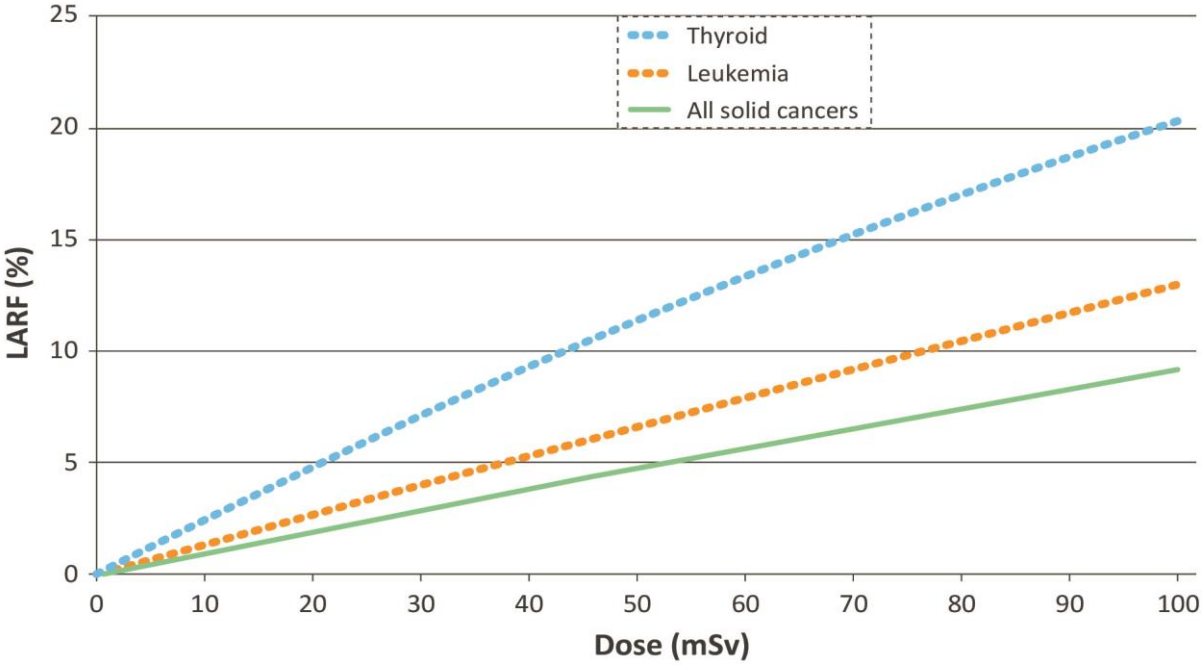

FIG. X–9. Average age specific LARF as a function of effective dose, estimated with the WHO model (children, female) [X–8].

The same calculations for adults are shown in the following four figures: males Fig. X–10 (ICRP) and Fig. X–11 (WHO); females Fig. X–12 (ICRP) and Fig. X–13 (WHO). It is seen that LARF for thyroid cancer does not exceed 5% even at a thyroid dose of 100 mGy. Consequently, it is very unlikely that an increase in thyroid cancer due to radiation exposure will be discernible among this population.

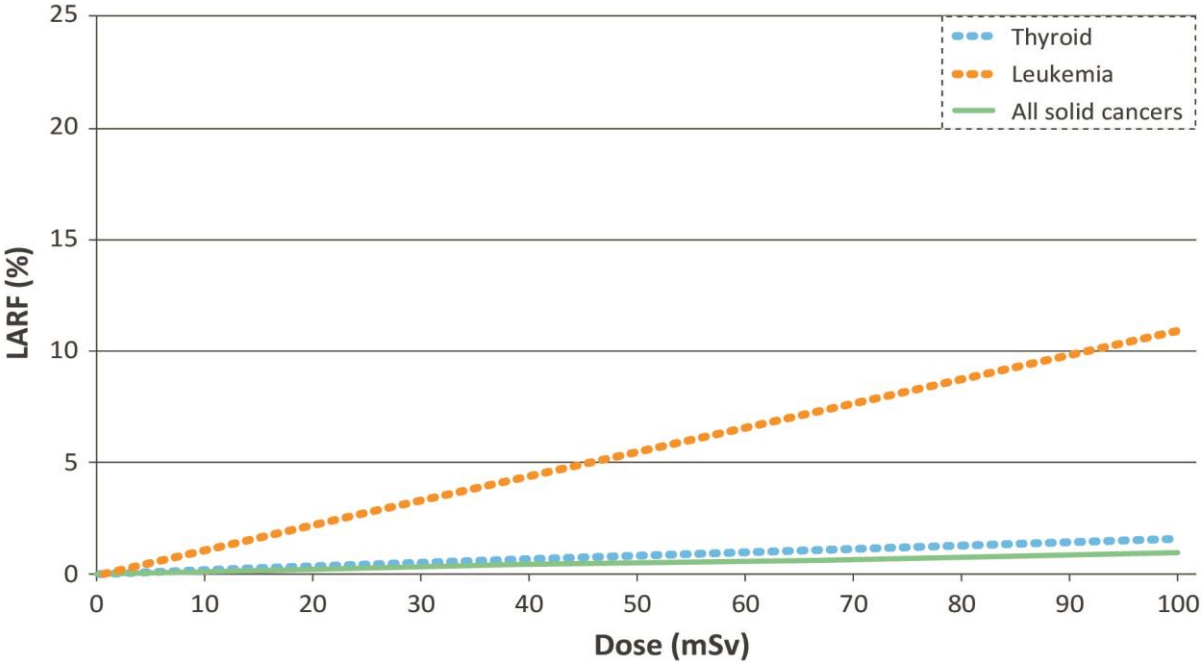

FIG. X–10. Average age specific LARF as a function of effective dose, estimated with the ICRP model (adults, male) [X–8].

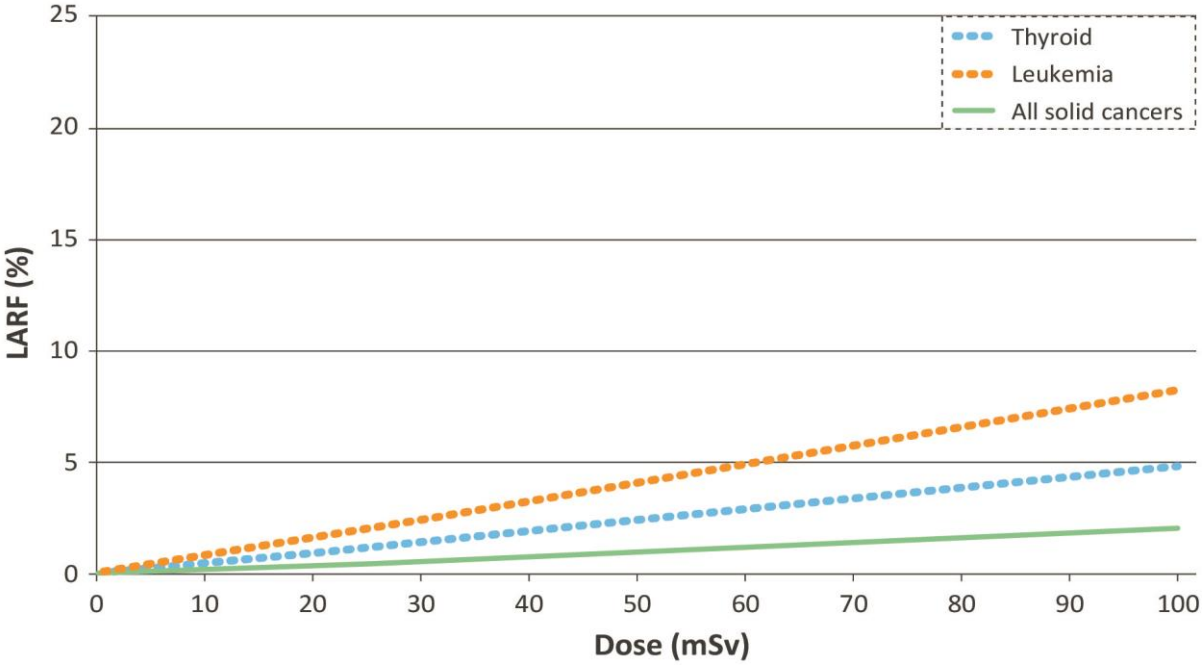

FIG. X–11. Average age specific LARF as a function of radiation dose, estimated with the WHO model (adults, male) [X–8].

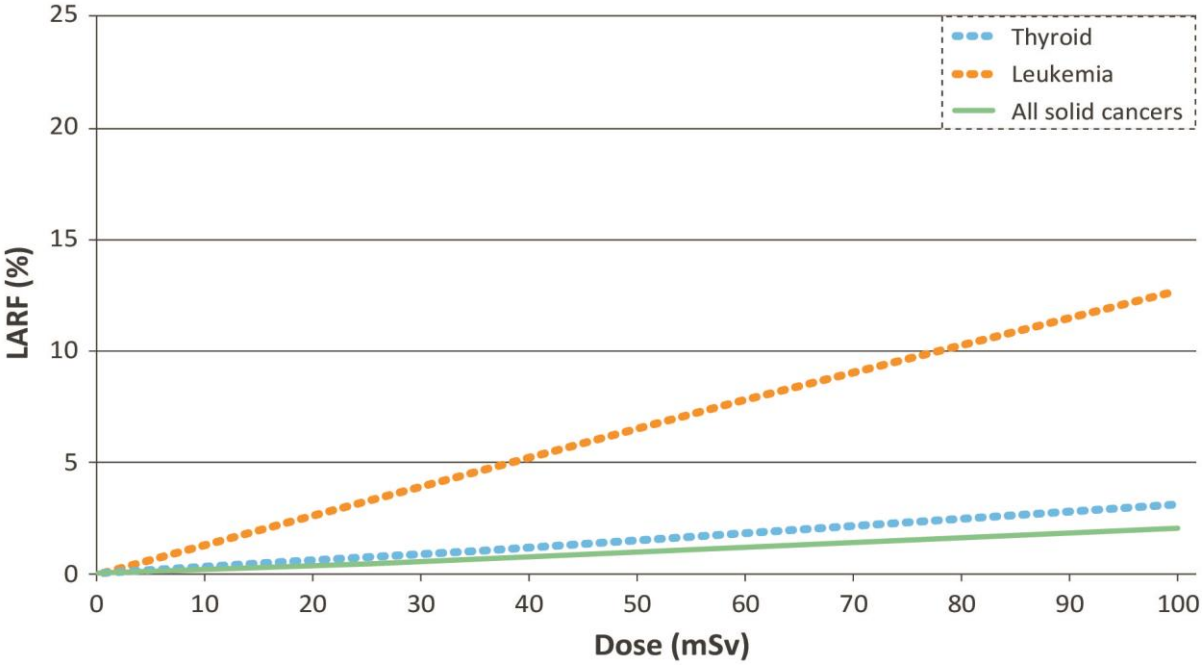

FIG. X–12. Average age specific LARF as a function of radiation dose, estimated with the ICRP model (adults, female) [X–8].

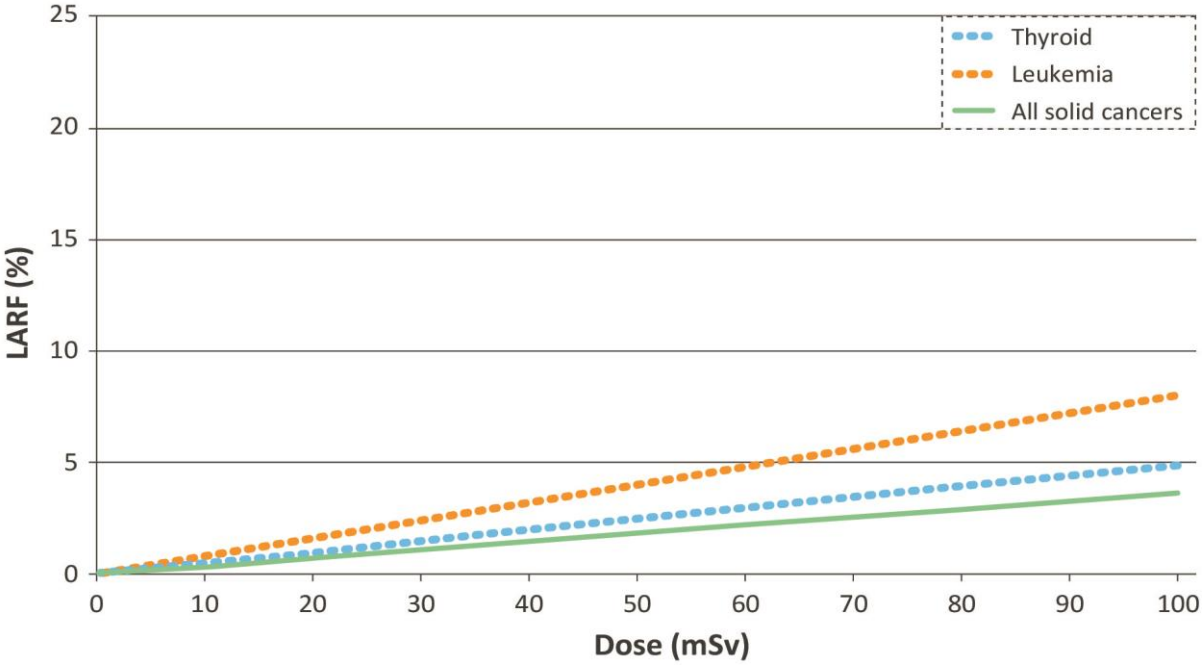

FIG. X–13. Average age-specific lifetime attributable risk fraction as a function of radiation dose, estimated with the WHO model (adults, female) [X–8].

It is significant that the inferred risk of thyroid cancer per unit dose for children of 0–4 years of age is higher than for older age groups. LARF values for boys and girls at this age group calculated with the ICRP and WHO models are shown in Figs X–14 and X–15. The calculated LARF value using the WHO model reaches 10% at effective dose of about 30 mGy. These data show the need for accurate estimation of radiation dose (and confidence intervals) for the general population, especially to the thyroid of children.

The LARF values were determined on the basis of estimated doses received by adults and children from a range of municipalities in the first four months following the accident (11 March–11 July 2011). The municipalities were selected to include those close to the Fukushima Daiichi NPP where higher dose rates were observed and from which people were evacuated.

These estimated doses from external exposure were based on the results of the Fukushima Health Management Survey, as they were available in February 2014 [X–11]. As described in Technical Volume 4, Section 4.2.2.2, these doses were estimated on the basis of maps of gamma dose rates, the results of a dose projection model and the results of questionnaires on individual behaviour during the period in question. The tabulated values are 95th percentiles of the estimated doses for people in the given municipalities. It should be noted that the Fukushima Medical University has since updated the estimated doses to take account of, among other things, additional responses to the questionnaire. The data presented and analysed in Section 4.2.2.2 are based on the most recent information available at the time of writing [X–12]. However, the estimated doses are generally very similar and the differences are unlikely to affect the assessed risks or the overall conclusions presented below. The greatest difference in the 95th percentiles presented here and in Section 4.2.2.2. is for Namie Town; a value of 2.3 mSv is presented below, while the corresponding value in Section 4.2.2.2. is 3.9 mSv.

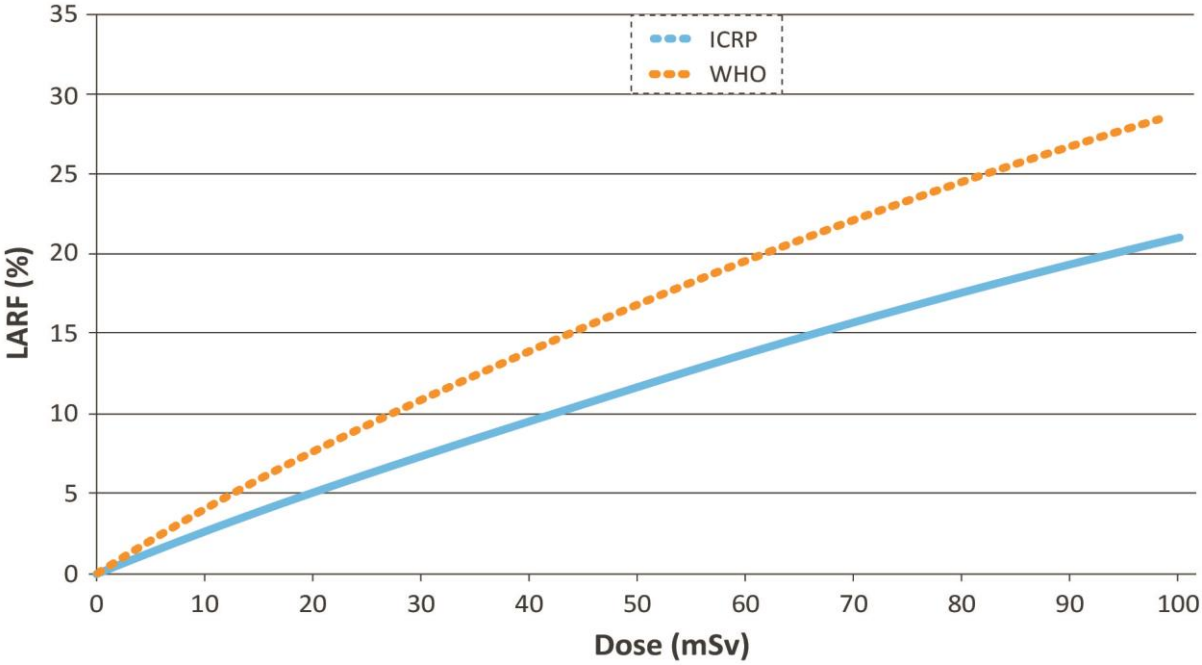

FIG. X–14. Average age specific LARF for thyroid cancer as a function of thyroid equivalent dose, estimated with the ICRP and WHO models (small children, male) [X–8].

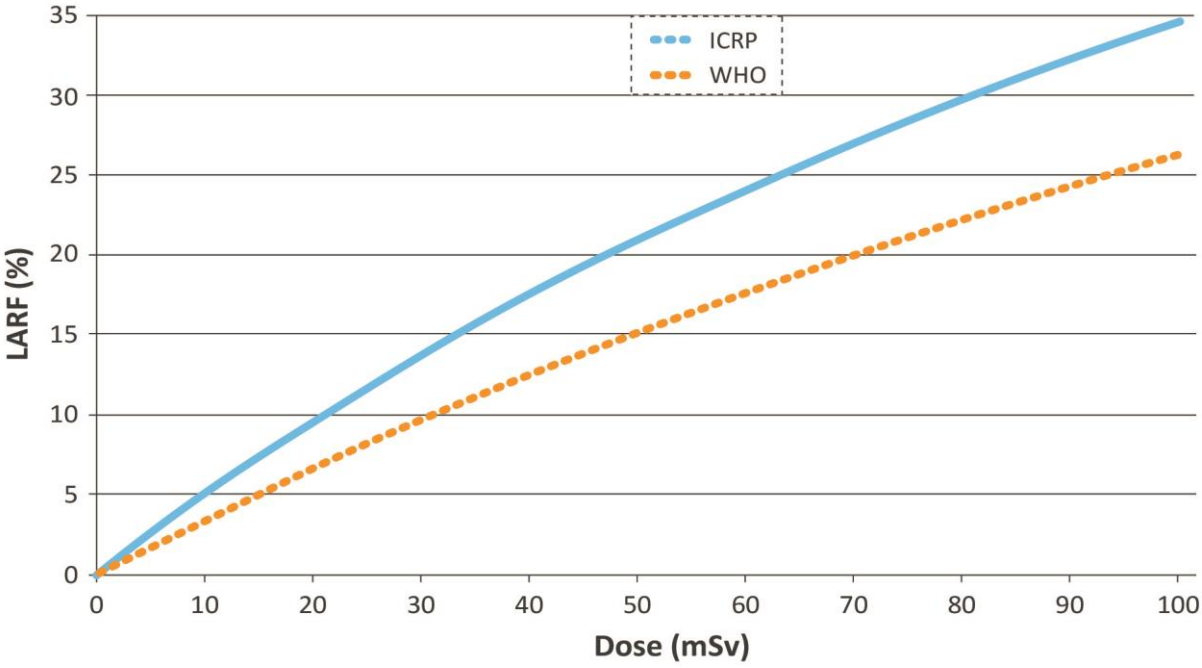

FIG. X–15. Average age specific LARF for thyroid cancer as a function of thyroid equivalent dose, estimated with the ICRP and WHO models (small children, female).

The equivalent doses to the thyroid presented below were derived from results of direct measurements of the thyroids of people in Namie Town and Minamisoma City between 12 and 16 March 2011 [X–13]. These results indicated that the median equivalent dose to the thyroid of people under 20 years of age was 4.2 mSv while the value for adults was 3.5 mSv.

The values of LARF calculated on the basis of estimated doses using the ICRP and WHO models are given in Tables X–10 to X–13.

TABLE X–10. LARF FOR ADULT MALES, INCLUDING EVACUEES, FROM A RANGE OF MUNICIPALITIES, OVER THE FIRST FOUR MONTHS FOLLOWING THE ACCIDENT, BASED ON TYPICAL COMMITTED EFFECTIVE DOSES OBTAINED FROM DIRECT MEASUREMENT

			LARF (%)					
			Solid cancers		Leukaemia		Thyroid cancer	
Location	External radiation (mSv)	Thyroid dose (mGy)	ICRP	WHO	ICRP	WHO	ICRP	WHO
Iitate Village	7.5	3.5	0.073	0.163	0.855	0.612	0.057	0.178
Minamisoma City	1.7	3.5	0.017	0.037	0.194	0.139	0.057	0.178
Namie Town	2.3	3.5	0.022	0.050	0.263	0.187	0.057	0.178
Fukushima City	2.3	3.5	0.022	0.050	0.263	0.187	0.057	0.178
Fukushima Prefecture	2.4	3.5	0.023	0.052	0.274	0.196	0.057	0.178

TABLE X–11. LARF FOR ADULT FEMALES, INCLUDING EVACUEES, FROM A RANGE OF MUNICIPALITIES, OVER THE FIRST FOUR MONTHS FOLLOWING THE ACCIDENT, BASED ON TYPICAL COMMITTED EFFECTIVE DOSES OBTAINED FROM DIRECT MEASUREMENT

			LARF (%)					
			Solid cancers		Leukaemia		Thyroid cancer	
Location	External radiation (mSv)	Thyroid dose (mGy)	ICRP	WHO	ICRP	WHO	ICRP	WHO
Iitate Village	7.5	3.5	0.152	0.286	1.003	0.597	0.115	0.181
Minamisoma City	1.7	3.5	0.035	0.065	0.228	0.135	0.115	0.181
Namie Town	2.3	3.5	0.047	0.088	0.309	0.183	0.115	0.181
Fukushima City	2.3	3.5	0.047	0.088	0.309	0.183	0.115	0.181
Fukushima Prefecture	2.4	3.5	0.049	0.092	0.322	0.191	0.115	0.181

TABLE X–12. LARF FOR MALE CHILDREN, INCLUDING EVACUEES, FROM A RANGE OF MUNICIPALITIES, OVER THE FIRST FOUR MONTHS FOLLOWING THE ACCIDENT, BASED ON TYPICAL COMMITTED EFFECTIVE DOSES OBTAINED FROM DIRECT MEASUREMENT

			LARF (%)					
			Solid cancers		Leukaemia		Thyroid cancer	
Location	External radiation (mSv)	Thyroid dose (mGy)	ICRP	WHO	ICRP	WHO	ICRP	WHO
Iitate Village	7.5	4.2	0.202	0.416	1.061	1.026	0.689	1.180
Minamisoma City	1.7	4.2	0.046	0.095	0.241	0.233	0.689	1.180
Namie Town	2.3	4.2	0.062	0.128	0.326	0.315	0.689	1.180
Fukushima City	2.3	4.2	0.062	0.128	0.326	0.315	0.689	1.180
Fukushima Prefecture	2.4	4.2	0.065	0.133	0.341	0.329	0.689	1.180

TABLE X–13. LARF FOR FEMALE CHILDREN, INCLUDING EVACUEES, FROM A RANGE OF MUNICIPALITIES, OVER THE FIRST FOUR MONTHS FOLLOWING THE ACCIDENT, BASED ON TYPICAL COMMITTED EFFECTIVE DOSES OBTAINED FROM DIRECT MEASUREMENT

			LARF (%)					
			Solid cancers		Leukaemia		Thyroid cancer	
Location	External radiation (mSv)	Thyroid dose (mGy)	ICRP	WHO	ICRP	WHO	ICRP	WHO
Iitate Village	7.5	4.2	0.431	0.748	0.683	1.011	1.353	1.062
Minamisoma City	1.7	4.2	0.098	0.171	0.155	0.229	1.353	1.062
Namie Town	2.3	4.2	0.133	0.231	0.210	0.310	1.353	1.062
Fukushima City	2.3	4.2	0.133	0.231	0.210	0.310	1.353	1.062
Fukushima Prefecture	2.4	4.2	0.138	0.241	0.219	0.324	1.353	1.062

X–7. SUMMARY OF INFERRED RISKS

As indicated above, the ‘lifetime attributable risk fraction’ (LARF) is one of the indicators used by epidemiologists in order to facilitate the theoretical inference of prospective radiation risks on a human population exposed to ionizing radiation. LARF is used to express the fraction of the total cancer incidence (both radiation-associated and non-radiation associated) that is radiation-induced for a population exposed to radiation. LARF is commonly expressed as a percentage. Risk models and approaches have been developed for calculation of LARF on the basis of experience and conclusions from epidemiological studies.

The figures and tables presented in this annex indicate that the LARF associated with the doses to emergency workers and members of the public arising from the Fukushima Daiichi accident were generally less than 1%. This result may be placed in context by considering that data obtained from large-scale epidemiological studies carried out following the Chernobyl accident have not been able to confirm inference of risk for LARF lower than 5–10% [X–10, X–14]. The prospective theoretical risk inferred from the range of doses that appear to have been delivered during and following the Fukushima Daiichi accident are small and it will not be possible to verify such a level of risk by the results of epidemiological studies.

In the low dose range, increases in cancer incidence are not discernible and hence increases in the incidence of cancer are not attributable to radiation at such doses.

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