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:

$t = 0.5699 \pm 0.1649$,

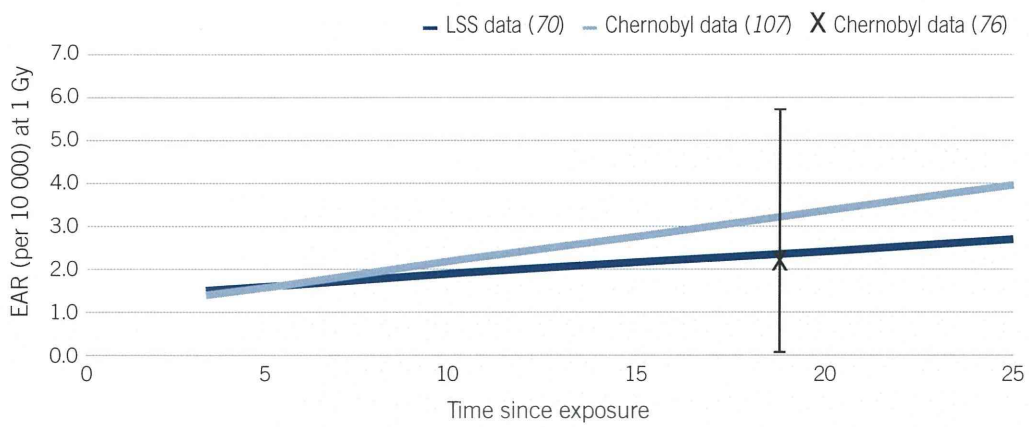
$k_d = 1.232 \pm 0.5061$,

$g_e = 0.05903 \pm 0.02385$,

$g_a = 0.5921 \pm 0.6191$

(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.