UK Health Security Agency

Impact of sex and age on prospective off-site health risk assessments of radiological accidents at nuclear sites

Prepared for:
Office for Nuclear Regulation (ONR)
Redgrave Court
Merton Road
Bootle
L20 7HS
UK
Report Reference: RCEHD-RAD-2023-04
Version Number: Issue 1

Impact of sex and age on prospective off-site health risk assessments of radiological accidents at nuclear sites

[redacted]

Executive Summary

This study evaluates the impact of age and sex on the assessments of off-site health risks to the public from accidents at nuclear facilities. These assessments are undertaken to demonstrate that such risks are tolerable and as low as reasonably practicable. Typically, the risks to public health from accidents presented in safety case submissions for UK licensed nuclear sites are based on risk factors averaged over age and sex as given in International Commission on Radiological Protection (ICRP) Publication 103 (ICRP, 2007). However, the recent ICRP Publication 147 (ICRP, 2021) provides, for the first time, age- and sex-specific factors and there have been several studies demonstrating higher radio-sensitivities in females and younger age groups.

The evidence of differences in radio-sensitivity to deterministic or acute health effects with age and sex is not sufficiently robust to allow quantification and therefore this document focuses on differences in risk of stochastic health effects or cancer.

Four atmospheric release accident scenarios were used in conjunction with wet and dry deposition conditions to explore the impact of age at the time of the accident and sex, on the calculation of doses and risks of stochastic health effects at near and far locations. Annual doses to selected organs were calculated using a modified version of the UKHSA's commercially available PACE (Probabilistic Accident Consequence Evaluation) Level-3 Probabilistic Safety Analysis tool (Charnock et al, 2020). The tool was adapted to enhance its approach to managing age and aging in the dose calculations. The risk calculations were based on Lifetime Attributed Risk (LAR) methodology (Thomas et al, 1992). Risks were calculated using tissue-specific risk models, parameterised by ICRP and US National Research Council Biologic Effects of Ionizing Radiation (BEIR) to be age- and sex-specific. The application of such models to accident scenarios, with multiple exposure pathways, extended durations of exposure and distinct dose profiles for different age groups is novel. The most similar study to date has been for the risks of medical exposures, which generally consider a fixed level of dose, delivered by single exposure pathway, for a short duration.

For the source terms and populations considered, a higher risk of cancer fatality, cancer detriment and cancer incidence can generally be seen in females and for those exposed as children compared to the population weighted risk averaged over age and sex (based on ICRP Publication 103 organ specific risk factors). For example:

- The *risk of fatality* for the 1-year-old female at near and far locations from the four accidents considered in the study was in the range of approximately 1 to 20 times higher. The ratio of 1 was obtained for a position adjacent to the most severe accident considered in the study, ST2, and resulted from high dose where calculated risks are

100% for all ages and sexes. The upper number was obtained for both near and far locations for the Design Basis accident (ST1) where doses are lower.

- The *detriment* for the 1-year-old female at near and far locations from the four accidents considered in the study was approximately in the range of 1 to 50 times higher. The ratio of 1 was obtained for a position adjacent to the most severe accident considered in the study, ST2, and resulted from high dose where calculated risks are 100% for all ages and sexes. The upper number was obtained for the far location for the Design Basis accident (ST1), where doses are lower. The broader range results from contributions made from non-fatal cancers to the detriment risk quantity.
- Similar comparisons for *incidence* of thyroid cancer and leukaemia yielded approximate ranges of 1 to 100 times and 1 to 10 times higher respectively. For both ranges the ratio of 1 was obtained for a position adjacent to the most severe accident considered in the study, ST2, and resulted from high dose where calculated risks are 100% for all ages and sexes. The upper number for incidence of thyroid cancer arises from the far location for the Design Basis lower dose source term (ST1). The upper number for the incidence of leukaemia arises at the near location for the large severe accident ST2.

The risks of cancer fatality, detriment and incidence calculated for locations near to the large severe accident ST2 were not representative of the rest of study. This is because the risks are due to very high doses where the predicted risk of occurrence is 100% for all ages and sexes. For the majority of other source terms and receptor locations, a degree of enhanced risk is evident for the younger ages and females compared to the general population. For the Design Basis Accident ST1, where lower doses are estimated to be received by the public off-site but with a higher likelihood of occurring, the health impacts are low and no increased rates in cancer over the background level is likely to be detectable in the population.

An important observation is that given the smaller datasets for people exposed when young, there are larger uncertainties in the risk models for those groups, though experts expect younger age groups to be more radio-sensitive. For the 10-year-old group, where there is more information available compared to 1-year-olds and therefore less uncertainty, the risk of fatality for the 10-year-old female was in the range 1 to 7 times higher compared to the population weighted risk, while the detriment was in the range of 1 to 8 times higher. This demonstrates that the use of both age and sex-averaged risk estimates can lead to an underestimation of risk for groups such as younger females when using organ-specific risk factors.

The next international periodic mortality and cancer incidence analyses of the Life Span Study cohort data will provide improved information on the risk models for those exposed at younger age groups as follow-up will encompass nearly their whole lifespan. ICRP are working to update their risk estimates as new population health statistics data, dosimetry information and radiation risk models are available. The differences in risk due to age-at-exposure and sex are already acknowledged by ICRP in its 2021 Publication 147. ICRP is currently carrying out a wider review of its system of radiological protection. This includes the setting up of a Task Group on Update of Detriment Calculation for Cancer which will also consider potential improvements to the calculational methodology. As part of this review ICRP will be considering whether sex and age-at-exposure calculations of effective dose and detriment can be used through a newly defined quantity, rather than the current use of simplified age- and

sex-averaged tissue weighting factors. However, this review is likely to take up to a decade to conclude.

In the meantime, safety cases for UK nuclear licensed sites should estimate age-specific doses to identify the most exposed individuals. ONR should also consider whether, given the markedly greater radio-sensitivity of the thyroid by age-at-exposure and sex, a separate specific dose or risk target for the thyroid should be developed where the accident scenarios involve the release of radioiodine. For cancers which vary significantly in risk according to age or sex, using age- and sex-specific risk factors should be considered.

This work was undertaken under the Radiation Assessments Department's Quality Management System, which has been approved by Lloyd's Register Quality Assurance to the Quality Management Standard ISO 9001:2015, Approval No: ISO 9001 - 00002655.

Contents

1 Introduction

This study evaluates the impact of age and sex on the assessments of health risks to the public from accidents at nuclear facilities. Typically the risks to the public presented in safety case submissions for UK licensed nuclear sites use risk factors averaged over age and sex as given in ICRP Publication 103 (ICRP, 2007). However, the recent ICRP publication 147 (ICRP, 2021) provides age-specific factors and there have been several studies demonstrating higher radio-sensitivities in females and younger age groups (Harrison et al, 2023; Puncher et al, 2017; Zhang et al, 2020).

This work uses four atmospheric release accident scenarios to explore the impact of age at the time of the accident and sex on calculations of doses and risks of stochastic health effects. The doses and risks were evaluated with respect to the ONR Safety Assessment Principles (SAPs) (ONR, 2014) to generate insights on the significance of any variation of risk with age and sex.

Four example source terms were compiled, described in detail below. For each source term, two sets of weather conditions were considered, one representing mostly dry deposition conditions and the other mostly wet. For each set of weather conditions, two receptor locations are considered, one close to the release and one further out. Therefore, there are four ‘examples’ for each source term: dry-near, dry-far, wet-near and wet-far giving sixteen sets of results.

PACE (Probabilistic Accident Consequence Evaluation) is a tool for performing Level-3 Probabilistic Safety Analysis (Charnock et al, 2020). It performs atmospheric dispersion modelling using the Met Office NAME model and was used to calculates doses to the selected organs. Some adaptation was required, to incorporate new ICRP data and to enhance its approach to managing age and aging in the dose calculations.

Risk calculations were performed externally to PACE based on Lifetime Attributed Risk (LAR) methodology (Thomas et al, 1992). The software for the calculation was developed by NRPB (now part of UKHSA) and was extensively tested and used in previous applications by the ICRP Task Group on Detriment Calculation Methodology (TG102) (ICRP, 2022) and by the WHO International Expert Working Group on the health risk assessment from the nuclear accident after the 2011 Great East Japan Earthquake and Tsunami (WHO, 2013).

Given the importance of the ingestion of contaminated food as a pathway, the impact of varying the percentage of milk consumed being locally produced from 50% to 10% is considered for ST1-DBA, providing an additional four sets of results.

A coastal location in the southwest part of England was chosen for the study. Prevailing winds in the UK are south westerlies so most plumes from this location will be over UK land mass. It is the site of an existing facility so it is evidently representative in terms of topography, land-usage and population distribution for a facility that would be built. However, the choice of this location for the study, and the position of the release point and the source terms used should not be taken as indicative of any current or future operations or facilities on the site.

The source terms are independent and do not form a coherent set of release categories that might be used as part of a safety case analysis for this or any site. They were chosen by ONR to cover the spread of nuclear accidents that could be envisaged in the UK but are generic and not specific to any specific licensee or reactor plant being built or operated in the UK. The

first source term (ST1-DBA) was chosen to represent a gigawatt light water reactor (LWR) design basis accident. The second source term (ST2-acute) represents low frequency, but very severe accidents in light water reactors, with widespread consequences that include deterministic health effects. Information provided by ONR states that ST2 is specific to new reactor designs where potential caesium releases are significantly reduced by design measures compared to ST4, thus enhancing the relative magnitude of incidence of iodine related thyroid cancer compared that of other caesium related cancers. The third and fourth source terms (ST3-fuel and ST4-Chornobyl) represent real world severe accidents, and their inclusion allows some limited benchmarking against observed consequences. The third source term (ST3-fuel) contains a radionuclide inventory defined to be representative of a generic severe accident at a fuel processing facility. It is based on the accident at Mayak, a large nuclear facility in USSR (now Russian Federation), which happened in 1957. The fourth source term (ST4-Chornobyl) is based on the accident at the Chornobyl nuclear power station in USSR (now Ukraine) in 1986.

Figure 1 gives an overview of the calculations and where the age- and sex- dependent factors are considered.

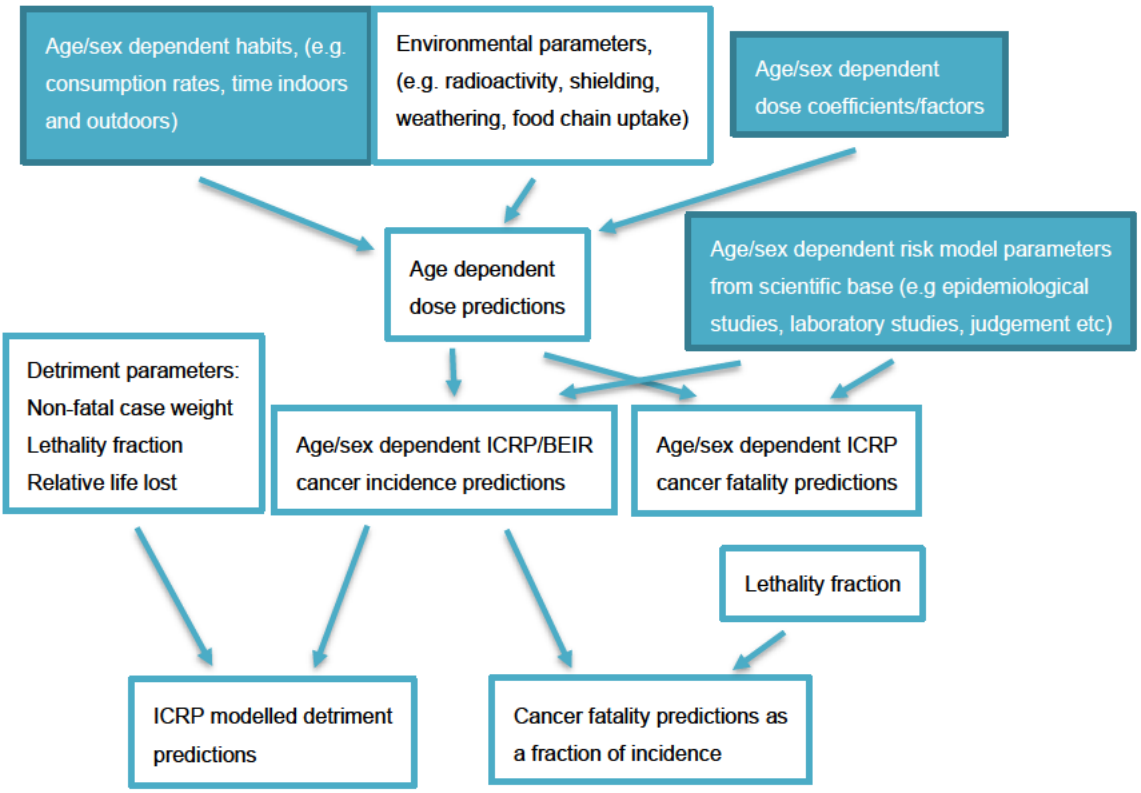


Figure 1 Connections between inputs, models, and endpoints in stochastic health risk

2 Dose calculation methodology

Table 1 lists the risks that will be calculated as part of this project and the organs or tissues for which equivalent doses are therefore required. In addition, effective dose was calculated.

Table 1 Health effect risks calculated

Health effect	Organ equivalent dose required	Grouping	Endpoint calculated: Risk of Incidence (I), Fatality (F) and detriment (D)
Oesophagus Solid Cancer (SC)	oesophagus	1y, 10y, 35y, 60y Male (M) and Female (F)	I, F, D
Stomach (SC)	stomach	1y, 10y, 35y, M F	I, F, D
Colon (SC)	colon	1y, 10y, 35y, M F	I, F, D
Liver (SC)	liver	1y, 10y, 35y, M F	I, F, D
Lung (SC)	lung	1y, 10y, 35y, M F	I, F, D
Breast (SC)	breast	1y, 10y, 35y, 60y, F	I, F, D
Ovary (SC)	ovary	1y, 10y, 35y, F	I, F, D
Bladder (SC)	bladder	1y, 10y, 35y, M F	I, F, D
Thyroid (SC)	thyroid	1y, 10y, 35y, 60y, M F	I, D
Other solid cancers[a]	remainder	1y, 10y, 35y, M F	I, F, D
All solid cancer	colon	1y, 10y, 35y, M F	I
Leukaemia	bone marrow	1y, 10y, 35y, 60y, M F	I, F, D
Total age/sex averaged risk [b]	All organ doses above and effective dose	ICRP103 Age/sex averaged	I, F, D

[a] not including bone cancer or skin cancer because suitable models are not available. Remainer tissues: Adrenals, extrathoracic region, gall bladder, heart, kidneys, lymphatic nodes, muscle, oral mucosa, pancreas, prostate (♂), small intestine, spleen, thymus, uterus/cervix (♀).
[b] for comparison against current approaches, age-averaged and sex-averaged, incidence and detriment risk estimates using the age average (0 – 89 years) factors from ICRP Publication 103 (ICRP, 2007) will also be calculated.

The dose calculations account for exposure via the following pathways:

a) external exposure to radioactivity in the plume,
b) inhalation of radioactivity in the plume
c) external exposure to radioactivity deposited on the ground,
d) ingestion of radioactivity in contaminated food.

The resuspension pathway and other minor pathways are omitted because they seldom make a significant contribution to dose.

The nominal risk factors presented in, for example, ICRP Publication 103 and ICRP Publication 147 are based largely on epidemiological studies of short-term external exposures to gamma radiation, mainly the atomic bomb survivors. However, ICRP Publication 147 concluded that it was reasonable, for protection purposes, to assume that internal exposures give an equivalent risk and that it was acceptable to sum external dose and lifetime committed internal doses to give a total dose from which risk could be calculated, although, as ICRP noted, there would be some conservatism for long-lived radionuclides which remain in the body for some time and do not deliver all their dose immediately. However, ICRP give little guidance for a situation of ongoing exposures by multiple pathways some of which may persist for years or even a lifetime.

In this study, it is important to account for the aging of each age group because dose coefficients change, as do other important parameters such as consumption rates as people get older. In addition, calculating a lifetime of exposure using, for example, dose coefficients appropriate for a one-year-old, is too conservative. Therefore, instead of calculating lifetime dose for external exposure and lifetime committed dose for internal exposure, yearly doses will be calculated for external exposure and yearly committed dose for internal exposure, from age at the time of the accident to 89 years old. This approach will avoid the conservatism in assuming lifetime committed dose is equivalent to external dose.

In addition, lifetime doses with be calculated by summing annual doses for the purposes of estimating the age/sex averaged risks based on Publication 103 for comparison purposes.

To accomplish this, the PACE software dose calculations have been adapted as described below.

2.1 Age groups and aging

PACE was modified to give doses for any arbitrary age-group from 1-year-old upwards. Since there will inevitably be some smearing of the delivery of the dose across adjacent years, particularly for the ingestion pathway (see below), the age-group for 1-year-olds for example can be best thought of as applying to 1 to 2 years-old and age group for 30-year-olds represents people from 30 to 31 years old etc.

The details of how aging is accounted for, for individual pathways, are given in the sections below, but the following general approaches to aging are taken in all calculations:

Calculations are performed on a yearly basis with all internal intakes treated as if they occurred at the start of each year. Dose calculations proceed until an assumed age of death of 89 years, i.e. the 1-year-old is assumed to live a further 88 years and the 10-year-old will live a further 79 years.

Generally, ICRP dose coefficients (DC), both internal and external, are provided for six age-groups, new-born (sometimes labelled 3-month-old), 1-year-old, 5-year-old, 10-year-old, 15-year-old and adult. For this project the new-born coefficients are not required.

When allowing for aging, DC for other ‘non-standard’ ages are approximated by selecting the nearest available standard DC as indicated in Table 2.

Table 2 Mapping of years of life to closest suitable dataset

Year of life	Dataset used	Year of life	Dataset used
1 -2	1-year-old	11 - 12	10-year-old
2 -3	1-year-old	12 - 13	10-year-old
3 -4	5-year-old	13 - 14	15-year-old
4 -5	5-year-old	14 -15	15-year-old
5 -6	5-year-old	15 - 16	15-year-old
6 - 7	5-year-old	16 - 17	15-year-old
7 - 8	5-year-old	17 - 18	15-year-old
8 – 9	10-year-old	18 - 19	Adult
9 - 10	10-year-old	19 -20	Adult
10 - 11	10-year-old	20 onwards	Adult

For consistency and because of the availability of data, intake rates (breathing and ingestion) are compiled for these standard ICRP age groups and again the nearest appropriate age specific rate are used as given in the Table 2. Other factors of secondary importance may also change with age, such as time spent indoors. However, these are included.

2.2 Dose coefficients

This project uses the latest available published dose coefficients (DC) from ICRP.

ICRP are in the process of updating their internal dose coefficients for the public, but they are not currently published. Therefore, the age specific DC have been extracted from the current version 3 of the ICRP database. These were calculated using a hermaphrodite phantom; a male but with ovaries, uterus and female breasts added. So, the internal DCs for breasts and ovaries are for females, but the dose coefficients of the other organs in Table 1 can be used for either sex.

The recent ICRP Publication 144 (ICRP, 2020) provides external dose coefficients, but these could not be incorporated directly into PACE on the project timescales, since they are instantaneous and do not account for decay, ingrowth, and weathering or soil migration in the environment. Therefore, they were used to scale the existing PACE external dose factors that do account for these processes, to provide the required age differentiation as described below. The ICRP publication 144 coefficients were calculated using sex specific phantoms for the standard ages. However, a preliminary investigation indicates that differences in dose coefficients between sexes are not large for most radionuclides, exclusive of breast and ovaries. They are typically within less than 10% difference often much less. Therefore, the doses provided by PACE for the risk calculation, are female for breast and ovary tissues, but sex averaged for the other organs in Table 1.

PACE uses the Met Office NAME atmospheric dispersion model to calculate external dose from the cloud since it has a good representation of the position of the plume in relation to the receptor. Unfortunately, NAME does not currently provide age-dependent doses and cannot be updated in the time scales of this project. Therefore, ICRP Publication 144 dose coefficients were used to scale the doses provided by NAME to provide the required age and sex dependent differentiation.

The approach to scaling the current PACE external dose factors and the external dose calculated by NAME is as follows.

The scaling factor for the effective dose for radionuclide *n* and age group *a* is given by the formula

$$SF_{eff,n,a} = DC_{eff,n,a}/DC_{eff,n,adult}$$

Where DC is the value taken from the appropriate ICRP Publication 144 effective DC dataset. NB the scaling factor for adults will be 1.0.

The scaling factor for dose to organ *o,* from radionuclide *n* and age group *a* is given by the formula

$$SF_{o,n,a}=DC_{o,n,a}/DC_{o,n,adult}$$

Where DC is the averaged value derived from the ICRP Publication 144, sex-specific organ dose datasets (or female for breast and ovary tissue). The scaling factor for adults will be 1.0.

2.3 Internal exposure from contaminated food

The dose delivered each year after the accident will be the sum of doses from contaminated food eaten and delivered in that year, together with those doses delivered from radionuclides in the body from radioactivity ingested in previous years.

PACE represents ingestion as two components, local produce contaminated by local deposition, and national produce contaminated at the national average deposition levels.

Local produce component

Dose from a given radionuclide in year 1 after the accident is simply the dose delivered in year 1 from ingestion in year 1:

$$Dose1 = Ing1 * DC(a)1$$

Where:

Ingz is the radioactivity consumed in the zth year after the accident (Bq)

DC(a)x is the ingestion dose coefficient for age-group a at time of ingestion, delivered during year x after ingestion (Sv/Bq)

For simplicity it is assumed that all the radioactivity is consumed at the start of the year. In reality, of course, ingestion will occur over the whole year, though this assumption will tend to be true in the early stages following the accident, and for shorter-lived radionuclides. However, due to cropping patterns, animal husbandry practices, restrictions, and seasonality the ingestion of contaminated foods will never be uniform across the year and additionally cannot be assumed to be continually decreasing. Consequently, some of the dose calculated to one year may be delivered in the subsequent year, this slight smearing of dose is deemed acceptable.

The dose in the second year is:

$$Dose2 = Ing1 * DC(a)2 + Ing2 * DC(a+1)1$$

And the dose in the third year is:

$$Dose3 = Ing1 * DC(a)3 + Ing2 * DC(a+1)2 + Ing3 * DC(a+3)1$$

And so on.

It should be noted that because of aging, the dose coefficients for a given period relative to consumption will change. As will the ingestion rate that is used to calculate the amount ingested. The DC and ingestion rate are selected according to Table 2.

Unfortunately, ICRP do not provide dose coefficients for annual periods following ingestion. Instead, they are provided for periods from time zero to 1-day, 7-days, 30-days, 1-year, 5-years, 10-years, 20-years 30-years,40-years, 45-years, and to age 70 years (e.g. 69 years for a 1-year-old, 50 years for an adult). However, dose coefficients for the required yearly periods can be approximated from the available data with sufficient accuracy.

For the 1st year following consumption the standard 0 to 1-year-period as provided by ICRP can be used. For the 2nd, 3rd, 4th and 5th year following consumption the yearly DC are approximated by

(5-year DC – 1-year DC)/4

For the 6th to 10th year following consumption the DC are approximated by

(10-year DC – 5-year DC)/5

For the 11th to 20th year following consumption the DC are approximated by

(20-year DC – 10 year DC)/10

The 21-30th, 31-40th, and 41-45th dose coefficients are approximated in a similar way, and the 46th to final year following consumption, the DC are approximated by

(lifetime DC – 45 year DC)/(lifetime-45)

Doses from radionuclides ingested more than 'lifetime' years ago are zero, though there will still be dose delivered from radionuclides ingested more recently.

National produce component

For the calculation of contamination levels of the national average produce the calculation is similar to the local component, but PACE uses crop production data to calculate a total collective committed dose from all production in the grid divided by the total population of the UK to give a per caput dose. This approach needs to account for different age-groups that have different annual consumption rates. Therefore, the total production of a food in a grid square P, is first apportioned to an age-group to give Pa, using the factor PFa

PFa = (IRa * Fa) / (sum over all age-groups of (IRa * Fa))

Where IRa is the ingestion rate of a food of age group a, and Fa is the fraction of the total population in age group a (from the national census). The collective dose to the age group is then calculated by using Pa to calculate the total integrated activity ingested by an age group in each year (IXa) in the usual way.

2.4 Internal exposure from inhaled radioactivity in the plume

Internal exposure from inhaled radioactivity in the plume shares similar complications to ingestion because it gives a committed dose. However, it is simpler as inhalation occurs only during the passage of the plume. Thus, dose from a given radionuclide in the first year is

Dose1 = Inh * DC(a)1

And dose in the second year and so on is:

Dose2 =Inh * DC(a)2

Where:

Inh is the radioactivity inhaled during the passage of the plume (Bq)

DC(a)x is the inhalation dose coefficient for age group (a) at time of inhalation, delivered during year x following inhalation (Sv/Bq)

The required inhalation dose coefficients are derived from the standard ICRP data sets in a similar fashion to the ingestion dose coefficients. Since inhalation occurs once at a specific age for each age group there is no need to adjust either the DC or the inhalation rate to account for aging.

2.5 External exposure from deposited radioactivity

The pathway of external exposure from deposited radioactivity is protracted but is simpler than the ingestion pathway because it does not give a committed dose. Thus, dose from a given radionuclide in the first year is:

Dose1 = Dep * DC(a)1 * Lf

And dose in the second year and so on is:

Dose2 =Dep * DC(a+1)2 * Lf

Where:

Dep is the deposition of a given radionuclide (Bq m^{-2})

DC(a)x is the external deposition dose coefficient for age-group a, for dose delivered during year x following deposition (Sv y^{-1} per Bq m^{-2})

Lf is a location factor, that accounts for shielding and time spent indoors and outdoors

As with ingestion the dose coefficient will be selected to account for aging according to Table 2. Lf will be treated as constant although it is acknowledged that it could be affected by age.

2.6 External exposure from radioactivity in the plume

External exposure from radioactivity in the plume is the simplest pathway as it is not protracted and does not give a committed dose. The dose only arises in the first year during the passage of the plume. Doses are calculated by the NAME model for an adult and scaled using the appropriate age dependent factor as described in Section 2.2.

2.7 Habit Data

TAG-45 (ONR, 2019) states that the representative person concept as presented by ICRP in Publication 101, should be used. The representative person is defined such that 'the probability is less than about 5% that a person drawn at random from the population will receive a greater dose' (ICRP, 2006). ICRP Publication 101 recommends that the 95th percentile provides suitable caution in a risk assessment as higher percentiles are likely to be composed of ever more unlikely extremes. This is the whole exposed population, not a subset defined by age or sex which TAG-45 makes clear:

The most appropriate person should be identified as the receptor for the analysis. ICRP has provided advice on how such a person should be identified (Ref 29).*

> *Usually this is an individual whose physical attributes (e.g. age) and habits give rise to a relatively high exposure (95th percentile) among the local population whilst remaining credible. As the exposure is prospective over the lifetime of a facility, this individual may be hypothetical in nature, and not an actual person living near the facility. Age specific dosimetry data has been provided by ICRP for foetus, newborn, adult and intermediate ages …*
>
> * ICRP-Publication 101a "Assessing Dose of the Representative Person…" (ICRP, 2006)

As the purpose of this project was not to make a safety case, it was inappropriate to perform an extensive habit survey and collection of data and the bulk of parameters are taken for those developed to conform to the REPPIR Consequence Assessment methodology (Bexon et al, 2019). This were supplemented where necessary with information as in those documents referenced by TAG-45.

The most difficult parameters to define are those related to consumption including age dependent consumption rates, fraction of food that is consumed locally and delay times for the fresh and the processed components of the diet and which are not extensively discussed in the REPPIR guidance. It is likely that the most sensitive assumption which affects dose is the proportion of local produce consumed.

Consumption rates

NRPB Report W-41 (Smith and Jones, 2003) collates information from national dietary surveys to provide data on annual consumption rates for the foods that PACE considers for the UK population, including mean, median and 95th percentile consumption rates. ICRP Publication 101 (ICRP, 2006) states that it considers that using the 95th percentile of behaviour is a cautious assumption for defining an intake rate in the absence of site-specific data. However as noted in the National Dose Assessment Working Group (NDAWG) Principles (Environment Agency et al, 2012) there is evidence from national and regional habit surveys that people rarely consume more than two foods at high rates. The NDAWG Principles conclude that 'In assessments where consumption rates for each food type are used, then two foods are assumed to be consumed at high rates while other foods are assumed to be consumed at average rates'. The two foods chosen are those which give rise to the highest dose. For this project the two foods chosen are milk and leafy green vegetables since these are often the most significant foods in accident situations.

NRPB W-41 provides values for offal consumption, but the data is aggregated i.e. no information is provided about the type of animal or internal organ. Therefore, offal was assumed to be 50% cow liver and 50% sheep liver. Values are provided for fruit, but the data does not distinguish between soft or orchard fruit. A split of 25% soft and 75% orchard fruit was assumed. Both these assumptions are the defaults used in PACE.

The consumption rates for 1-year-olds, 10-year-olds, 15-year-olds, and adults were extracted directly from NRPB Report W-41, with rates for 5-years olds being assumed an average between 1-year-olds and 10-year-olds.

Delays between harvesting and consumption

The PACE default delays were used for all foods except the fresh fraction of milk which was reduced to 12 hours to account for the representative person likely being a dairy farmer or farmer's family member consuming their own produce. The PACE default delays are consistent with Jones and Sherwood (2008), though simplified for the limited food groups in PACE.

Fraction of locally produced food consumption

This parameter, which is provided separately for each food, has been identified as particularly sensitive for the ingestion dose calculation. However, there is limited information about this for any food, including milk. As part of an assessment of risks of leukaemia and other cancers around Sellafield, farming practices in West Cumbria in the 1990s were reviewed and it was concluded that 40% of milk consumed in the area is produced locally, with the remaining proportion being assumed to come from other parts of West Cumbria. 25% of green vegetables, meat and offal and 50% of root vegetables were assumed to be of local origin with no grain produced locally being consumed by the local population (Simmonds et al, 1995). The defaults in PACE are currently 25% for all foods but are likely to be conservative except for local farmers consuming their own produce.

NRPB (1994) discusses the consumers of locally contaminated food in the context of calculating exposures when CFILs (Council Food Intervention Levels, now called maximum permitted levels MPLs) are applied after an accident.

> *… Therefore, in assessing the doses received as the result of adopting the CFILS after an accident, it is unreasonable to simply assume that all an individual's food is contaminated to that level. It is judged that an assumption of 10% of an individual's diet of a particular food type being contaminated at the CFILs will provide a cautious estimate of the upper levels of dose that would be received from that food with the exception of those (probably very few) individuals consuming home-produced vegetables or milk which had become highly contaminated.* [Appendix C p26]

The 10% value given is a judgement and not derived from any specific survey. The authors make it clear that while it includes the proportion of food sourced locally, it also includes a judgement about the impact of both the spatial and temporally extent of the accident (i.e. even locally sourced food is not all going to reach the MPL, and those that do will not remain at the MPL for the long term). It should be noted that the authors explicitly acknowledge that there will be some individuals for whom 10% is not conservative, because they source a large proportion of some of the foods in their diet locally. This is likely to be the case for the locality chosen for this scenario, which is a milk producing area and it is likely that some farmers do consume milk from their own holdings.

After discussions with ONR, to get a greater understanding of the impact of this parameter, two different assumptions of 10% and 50% of the amount of locally produced milk consumed were chosen. Calculations were done for ST1-DBA as for this scenario no food restrictions were predicted and therefore the different assumptions in this parameter could be seen.

1) Assume 50% of milk consumed is locally produced, with 25% of all other foods being local
2) Assume 10% of milk consumed is locally produced, with 25% of all other foods being local

3 Lifetime Risk calculation methodology

Lifetime risk of cancer incidence can be estimated for a population exposed to ionising radiation based on the lifetime doses which were calculated up to age 89 years, starting with the doses received during the first year after the accident and ending eighty-nine years after the accident.

The lifetime attributable risk $LAR_c(e,D)$: the lifetime risk of a cancer *c* that has been caused by exposure *D* at age *e,* can be calculated as following:

$$LAR_c\,(e,D,s) = \int_{a=e+L}^{a=89}[\mu_c\,(a|e,D,s) - \mu_c(a,s)]S(a|e,s)da \quad (1)$$

Where

μ_c(a, s): the incidence rate from cancer *c* at age *a* for sex *s*

$\mu_c(a|e,D,s)$: the incidence rate at age a from cancer c on exposure to a dose *D* at age *e* for sex *s*

S(a|e,s): the survival function – gives the probability of surviving to age *a* on exposure to a dose D at age e for sex s.

For each age at exposure, the risk was cumulated up to the attained age of 94 years. *L* was assumed to be 5 years for all cancers including leukaemia.

The survival curve S(a|e, s) can be calculated as:

$$S(a|e,s) = \exp(-\int_e^a \mu(x,e,s)dx)$$
$$\approx \prod_{n-e}^{a}[1-\mu(n,e,s)] \quad (2)$$

Where $\mu(x)$ is the rate of dying from causes other than cancer or contracting any cancer at age x. It is described as

$$\mu(x) = \mu_m(x) - \mu_{mc}(x) + \mu_{ic}(x) \quad (3)$$

Where $\mu_m(x), \mu_{mc}(x), \mu_{ic}(x)$ are all-cause mortality rate, all-cancer mortality rate, and all-cancer incidence rate.

The incidence rate $\mu_c(a|e,D,s)$ can be calculated as:

$$\mu_c(a|e,D,s) = [1+ERR(a|e,D,s)] \times \mu_c(a,s) \text{ or } \mu_c(a|e,D,s) = \mu_c(a,s) + EAR(a|e,D,s) \quad (4)$$

Where ERR(a|e,D,s) and EAR(a|e, D,s) are the excess relative risk and excess absolute risks. They are modelled from Japanese A-bomb survivors studies (Preston et al, 2007)

The lifetime attributable risk based on lifetime organ dose, *D*, *LAR* (*D*, *e*, *s*) is a summation of integral terms from Eq. 1, i.e.,

$$LAR_c(D,e,s) = \sum_{i=1}^{50} LAR_c\,(d_i,e,s) \qquad (5)$$

To put radiation-related cancer risks into perspective *LAR* was also considered as a percentage of the lifetime baseline risk (*LBR*) of cancer (i.e., the risk in the absence of radiation exposure from the accident). Applying the same notation for definition of *LAR,* the *LBR* is calculated as:

$$LBR_c(a_{min},s) = \int_{a_{min}}^{a_{max}} m(a,s)S_{aj}\,(a|a_{min},s)ds \qquad (6)$$

3.1 Demographic Data input

Demographic data of Euro-American population, such as age-sex specific population size, all causes mortality rate, all cancer mortality rate, all cancer incidence rate and specific cancer incidence rate from the International Commission on Radiological Protection, Publication 103 (ICRP, 2007) are used for the calculations. To define representative populations for lifetime risk calculations, populations with long-running cancer registries were selected as sources of the baseline health statistics. Population datasets from Sweden, UK, and the Surveillance, Epidemiology, and End Results (SEER) Program of the US National Cancer Institute were used (ICRP Publication 103). An unweighted average among these selected populations was calculated to form the data of a composite population (ICRP Publication 103). The data are tabulated in Annex A of ICRP Publication 152 (ICRP, 2022) as well as in Table A.4.10 to A.4.17 of ICRP Publication 103.

3.2 Risk model

For estimating cancer risk from radiation exposure, risk models were developed for oesophagus, stomach, colon, liver, lung, female breast, ovary, bladder, thyroid and bone marrow (leukaemia) (ICRP Publication 103, ICRP Publication 152). Cancers of other tissues were consigned to a remainder category called ‘other solid’. Excess relative risk (ERR) and excess absolute risk (EAR) were modelled to calculate the ERR and EAR lifetime risk estimates separately.

Risk models for solid cancers mostly derive from the report on solid cancer incidence among the Life Span Study (LSS) cohort of the Japanese atomic bomb survivors, which were based on the data of first primary cancers diagnosed from 1958 through 1998 (Preston et al, 2007). The ERR model and the EAR model are both expressed as

$$Excess\ Risk = \beta dexp(\frac{\alpha_1(e-30)}{10} + \alpha_2 \ln\left(\frac{a}{70}\right)) \qquad (7)$$

where d = dose (Gy), e = age at exposure (years), a = attained age (years), and α_1, α_2 and β are parameters. Parameter values are presented in ICRP Publication152 for the ERR model and the EAR model.

Leukaemia risk was modelled based on an analysis of unpublished incidence data among the LSS cohort for the period from 1950 through 2000. Mathematical expression of the EAR model for leukaemia is

$$\text{Leukaemia EAR} = (\beta\, d + 1.53\, d^2)(t/25)^{\alpha} \quad (8)$$

Where t represents time since exposure (i.e. attained age minus age at exposure, in years), and α and β are parameters. The values of the parameters differed according to sex and category of age at exposure as shown in ICRP Publication 152.

Instead of developing an ERR model for leukaemia, ERRs for certain attained ages and ages at exposure were computed by taking the ratio of EAR to the baseline rate. For this purpose, baseline incidence of leukaemia in the LSS cohort was modelled as

$$\text{Leukaemia baseline rate} = exp\,(\alpha_1 + \alpha_2\,(e-30) + \alpha_3\,\ln(\tfrac{a}{50}) + \alpha_4\,(\ln(\tfrac{a}{50})^2 + \alpha_5\,(\ln(\tfrac{a}{70})^2) \quad (9)$$

where α_1, ..., α_5 are parameters provided in ICRP publication 152. Similar models were also developed by the US National Research Council (BEIR VII) (National Research Council, 2006). Both the ICRP risk models and BEIR VII risk models were used in lifetime attributed risk calculations for comparison.

3.3 Calculational procedure

Lifetime risk was calculated for each combination of model (ERR and EAR) as a weighted mean of LAR. The lifetime risk was averaged across models, with ERR:EAR weights of 0:100% for breast, 100:0% for thyroid, 30:70% for lung, and 50:50% for all other cancer sites.

In order to derive risks from low dose or low dose-rate exposures from risk estimates based on moderate-to-high dose and high dose-rate data, ICRP introduced a dose- and dose-rate-effectiveness factor (DDREF). The choice of DDREF has been a topic of discussion in recent years. ICRP Publication 103 uses a DDREF of 2 in their lifetime risk calculation, the National Academy of Sciences/National Research Council proposed a DDREF value of 1.5 in BEIR VII (National Research Council, 2006), and some consider that there is no need to apply a DDREF value (SSK, 2014) at all. However, since the DDREF value does not make a difference to the risk ratio of either age-at-exposure or sex as it would be applied to all ages and sexes, a value of 1 was assumed for both risks of solid cancer and leukaemia.

3.4 Adjustment for severity

The adjustment of the nominal risks for severity was done by applying three adjustment factors that reflect lethality, quality of life (QOL), and years of life lost (YLL), respectively. These factors are independent of radiation dose.

Since the nominal risk coefficient was calculated based on the excess incidence, the lethality fraction (*k*) was applied to take account of cancer severity. Lethality fractions were derived as judgement-based values reflecting the impact of medical treatment (ICRP Publication 103).

A QOL factor (*q*) was applied to the non-lethal fractions of cancers, to adjust not only for lethality but also for pain, suffering, and any adverse effects of cancer treatment that cancer survivors generally experience. It is expressed in the formula

$$q = k + q\_min\ (1 - k) \quad (10)$$

Where *k* is the lethality fraction and q_{min} is a factor representing the minimum weight for non-lethal cancers.

Relative years of life lost (*l*) is another adjustment factor for the detriment calculation. YLL for a certain cancer was computed for each sex in the Euro-American population. For each cancer site, *l* was YLL for that cancer divided by the average YLL for all cancers. The Years of life lost (YLL) for a particular cancer can be calculated as:

$$YLL_c = LLE_c(D, e, s)/LAR_c \quad (11)$$

Where $LLE_c\ (D, e, s)\ \int_e^{94} S(a|e)ds - \int_e^{94} S(a|e, D, s)da$ is loss of life expectancy, and S(a|e, D, s) is the survival function taking into account of radiation exposure D at age e (Thomas et al, 1992).

3.5 Calculation of radiation detriment

The radiation detriment (D_c) for each organ or tissue was calculated by applying the adjustment factors to the nominal risk coefficient (LA*R*). It is given by the following formula.

$$D_c = LAR_c(k + q(1 - k))\ \ l \quad (12)$$

It represents a theoretical estimate of the weighted number of excess cases per unit dose of radiation, as described in ICRP publication 103 (ICRP, 2007).

3.6 Calculation of risk of fatalities

ICRP Publication 103 provides a set of mortality models and mortality rates for various cancer types, so that the risk of fatality can be directly evaluated. However, there is no mortality model for thyroid cancer in ICRP Publication 103 because such a model was difficult to be derive due to the scarce information on thyroid cancer mortality data in the Japanese A-bomb survivors' cohort. An alternative method to evaluating the risk of fatality is provided in the

ICRP Publication 103 which involves the multiplication of the cancer incidence risk with an averaged lethality fraction. It provides a rough estimate of the fatality risk for the cancer concerned. However, it needs to be borne in mind that it might introduce uncertainties as an age and sex averaged lethality fraction is used when trying to estimate an age and sex dependent fatality risk. Caution is required when the direct comparison is made between the fatality risks from the two mentioned methods above.

4 Presentation of results

This project generated thousands of intermediate and final endpoints, including dose by radionuclide, pathway, for 4 age groups, annually for up to 88 years for twenty different examples and variants. In addition, results have been produced on risks of incidence, fatality, and detriments in several tissues, and again for four age groups and for several examples and modelling approaches. It is not possible to show all the results or to evaluate all the relationships between them in detail. The following sections include example graphs and tables to illustrate the most important findings. The following points should be kept in mind when interpreting the graphs and results.

1. All risks are presented as percentages.
2. Unless explicitly stated all results are based on the assumption requested by ONR that 50% of milk consumption is local.
3. Some of the results have been truncated or derived from truncated values and these are marked with an asterisk ('*') in the graphs:
    a. No risk can be higher than 100%. However, some of the doses predicted are very large, and the risk models can appear to give greater than 100% risk. These results are spurious and have been truncated to 100% in tables and graphs.
    b. For some tissues, two risk models are combined to give a weighted average. It is possible that one model predicts a value greater than 100% whilst the other is lower. In this case the model is truncated before the two results are combined. Thus, some results that are apparently less than 100% may still be marked as truncated because one of the underlying models was greater than, but truncated to, 100% while the other was less.
    c. Some risks of fatality are calculated by applying a lethality fraction to the risk of incidence. Where a risk of incidence has been truncated, this is done before estimating the subsequent risk of fatality from it. The risk of fatality will be less than 100% but still marked with an asterisk to indicate that it was derived from a truncated incidence value.
    d. The detriment is evaluated according to Section 3.5 and is 'lifetime risk of incidence' given as a percentage and modified by three dimensionless factors to account for lethality, quality of life, and relative years of life lost. These 3 factors mean that detriment and lifetime risk of incidence, have slightly different values. Detriment values can be higher than the lifetime risk because the relative years of life lost can be greater than 1 for some cancers,

but they are only meaningful in the context when their values are at or below 100%. Where detriment has been truncated to 100%, or is derived from an incidence that has been truncated to 100% it is marked with an asterisk.

4. Male and female risks are calculated for all organs except ovaries and breasts using ICRP models. The same age-dependent organ dose was assumed for males and females so differences in risk between sexes arise from the risk modelling and not the dose modelling. For ovaries and breast only risks to females are calculated and presented as the doses are specifically female.
5. Risks of incidence of cancer are calculated using both ICRP and BEIR models. Where necessary ICRP model results are labelled 'ICRP' to distinguish from 'BEIR' model results in the graphs, but by default have no label. BEIR VII model results are always labelled 'BEIR'. Risks of fatalities and detriment are calculated using ICRP models only and are not labelled.
6. Risk of fatalities are calculated in two ways, firstly directly using ICRP mortality risk models (labelled 'mort' in the graphs), and secondly by applying an age and sex averaged lethality fraction to the modelled age-dependent incident risks (labelled 'inc' in the graphs). ICRP have no model for thyroid cancer fatality so there are only 'inc' estimates of thyroid cancer fatality risk.
7. Age averaged risk has been calculated for comparative purposes using ICRP Publication 103 nominal risk factors, combined with the organ doses of the 35-year-old. For all organs except breast and ovaries, the risk is sex-averaged as well.
8. Risks of incidence of cancer in each tissue have been estimated using ICRP models and recommendations. In addition, the risk incidence and fatality of all-solid-cancers has been calculated by using the ICRP model combined with the colon organ dose. It is noted that in some cases the predicted risk of solid cancer in an individual organ appears greater than the predicted risk of all-solid-cancers. The risk of all-solid-cancers is a combined risk, and therefore one would expect it to be higher than the risk of solid cancer in any individual tissue. The predicted risk of all-solid-cancer would be higher if a similar value organ dose was used for all the tissues and for the all-solid-cancer risk. However, in these examples' organ doses can be very different, and so in some cases, risks can appear inconsistent.
9. Leukaemia risk uses a linear-quadratic model meaning that the risk can be very high at high doses. Solid cancer risk models in contrast are all linear.

5 Source term 1 – ST1-DBA

The first source term was obtained from a previous ONR funded project: "Provision of Guidance on the Radiological Impact of Rainfall on Nuclear Plant Design Basis Accidents" and in that project was designated the 'LBL' source term. It is a light water reactor (LWR) design basis accident and is a small release, but notably with a very long duration of 30 days, for this project is termed ST1-DBA.

Because of the long duration it is not possible to find a set of meteorological data in which the release is completely directed towards land and does not contain a mixture of wet and dry deposition.

5.1 Dry Deposition

The weather set selected as being predominantly dry begins at 00:00hrs 22/6/19. The pattern of total deposition of ^{137}Cs is shown in Figure 2. The 'near' location was placed in the grid square with the highest total deposition outside of the boundary fence and is towards the southeast. At this location the predicted wet deposition of ^{137}Cs is less than 16% of total predicted ^{137}Cs deposition. The 'far' location was placed in the grid-square with the maximum deposition at 10km and is towards the east, with the wet ^{137}Cs deposition at this location accounting for approximately 28% of the total ^{137}Cs deposition. Based on the lower ERLs, no emergency protective actions are required. Food restrictions would not be required beyond the site boundary.

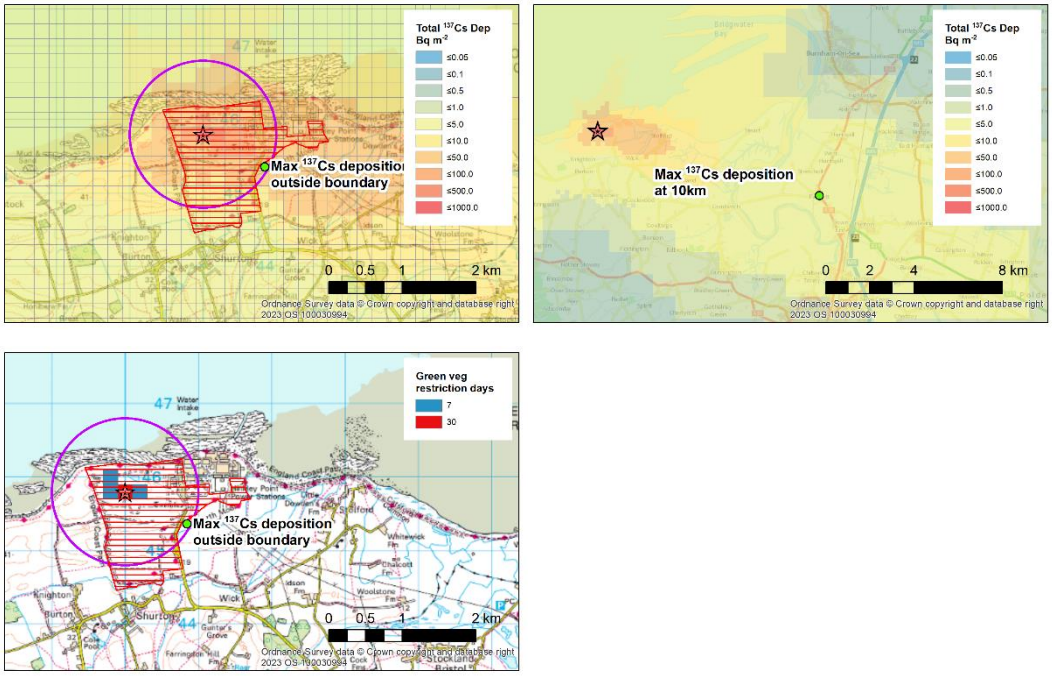


Figure 2 Predicted consequences of dispersal of ST1-DBA. The model run beginning 00:00 22/6/19, giving predominantly dry conditions.

5.2 Wet deposition

The weather set selected as predominantly wet begins at 00:00hr 1/5/21, Figure 3. The near location was placed in the grid-square with the highest total deposition outside of the boundary and is at the same position as for the dry deposition example, here approximately 70% of the predicted ^{137}Cs deposition is wet. The far location was placed in the grid-square with the maximum total predicted ^{137}Cs deposition at 10km is towards the south, with approximately 97% of the ^{137}Cs deposition occurring due to wet deposition. Based on the lower ERLs, no emergency protective actions are required. Food restrictions are not required beyond the site boundary.

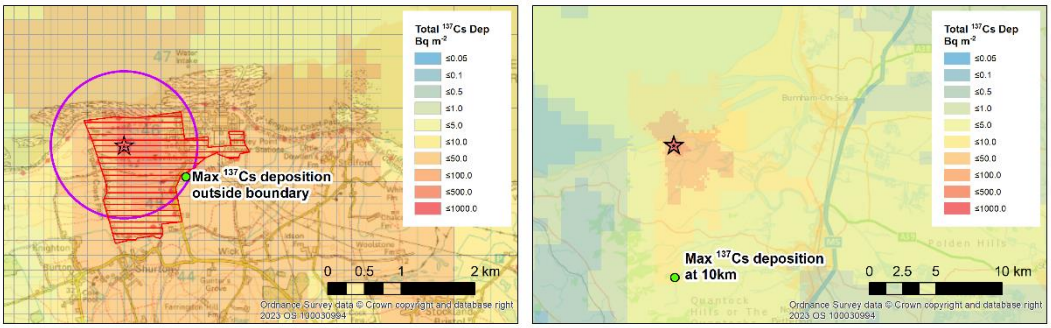


Figure 3 Predicted consequences of dispersal of ST1-DBA. The model run beginning 00:00 1/5/21, giving predominantly dry conditions.

5.3 Dose profiles

The effective doses for the ST1-DBA scenario are below 0.1 mSv with the highest predicted dose being 0.074mSv (see Table A1). The thyroid has the highest organ doses, with the highest predicted for the examples being 1.8mSv. Doses to other organs are substantially smaller. All doses are far below the levels at which deterministic risks would be expected.

Predicted thyroid doses based on an assumption of 10% locally produced milk consumption are reduced by approximately 50% to 75% compared with the 50% locally produced milk assumption.

The thyroid dose shows a strong age dependency which is not present for the other organs. The predicted 1-year-old thyroid dose is 5 to 9 times higher than the 35-year-old with the 50% locally produced milk assumption, dropping to 3 to 6 times higher with the 10% locally produced milk assumption.

A more detailed description of the dose profiles can be found in Appendix A1.

5.4 Risk profiles

5.4.1 Incidence

Figure 4 and Figure 5 compare all the predicted risks of incidence of cancer, but the thyroid risk is so dominant that it is difficult to discern the risks for other tissues. To obviate this, Figure 6 and Figure 7 show the same results but with the thyroid risk omitted. Relative risks are very similar between examples (i.e. disregarding absolute values, the graphs appear very similar).

The following features can be seen:

- The risk of incidence of thyroid cancer is the highest of all individual tissues, followed by breast, remainder organs (‘Other solid’), and lung.
- For thyroid, colon, breast, bladder, ovary, stomach, liver, remainder (‘other solid’) and all-solid-cancer (‘Solid’) there is a clear age dependency, generally with 1-year-old having the most risk, followed by 10-year-old, 35-year-old, and 60-year-old. For the other tissues the relationship is either not strong, or not consistent, or risks are so small that the relationship is not discernible.

- The risk for females is notably higher than for males for thyroid and lung and for all-solid-cancers (‘Solid’). For the other tissues, the male risk is larger or there is no clear pattern.
- There are only small differences between the corresponding risks of incidence of calculated by the ICRP and BEIR models.
- The risks of thyroid cancer incidence are much larger than the corresponding risks of all-solid-cancers (‘Solid’). Excluding thyroid, the risk of all-solid-cancers is larger than the risk of any single other tissue and looks to be a reasonable approximation of the combined risks.
- The age/sex-average risk estimated with ICRP Publication 103 nominal factors is similar to the 35-year-old for most tissues including thyroid but does not generally conservatively represent the risk to 1-year-old or 10-year-old.

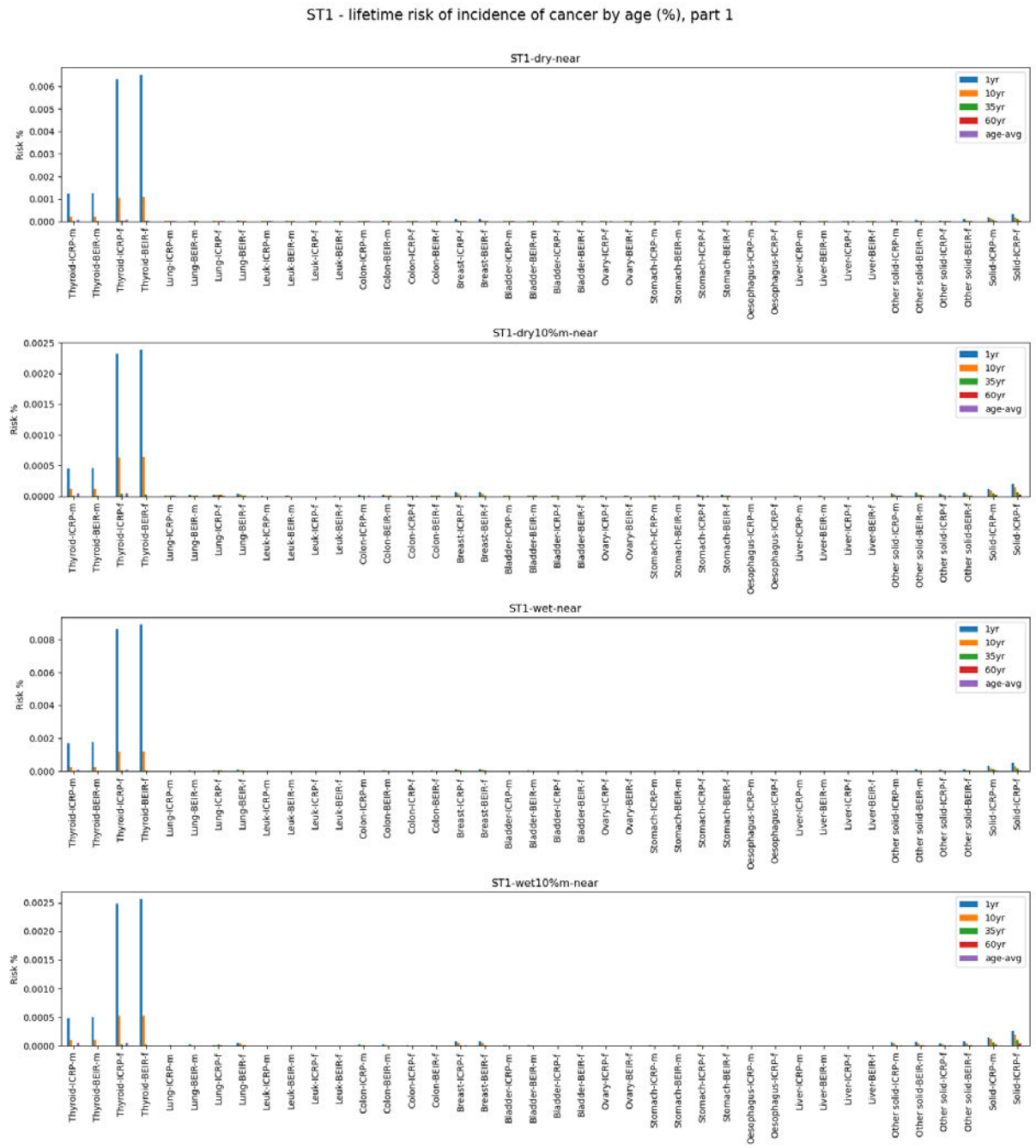


Figure 4 ST1-near – all predicted risks of incidence of cancers using both ICRP and BEIR models

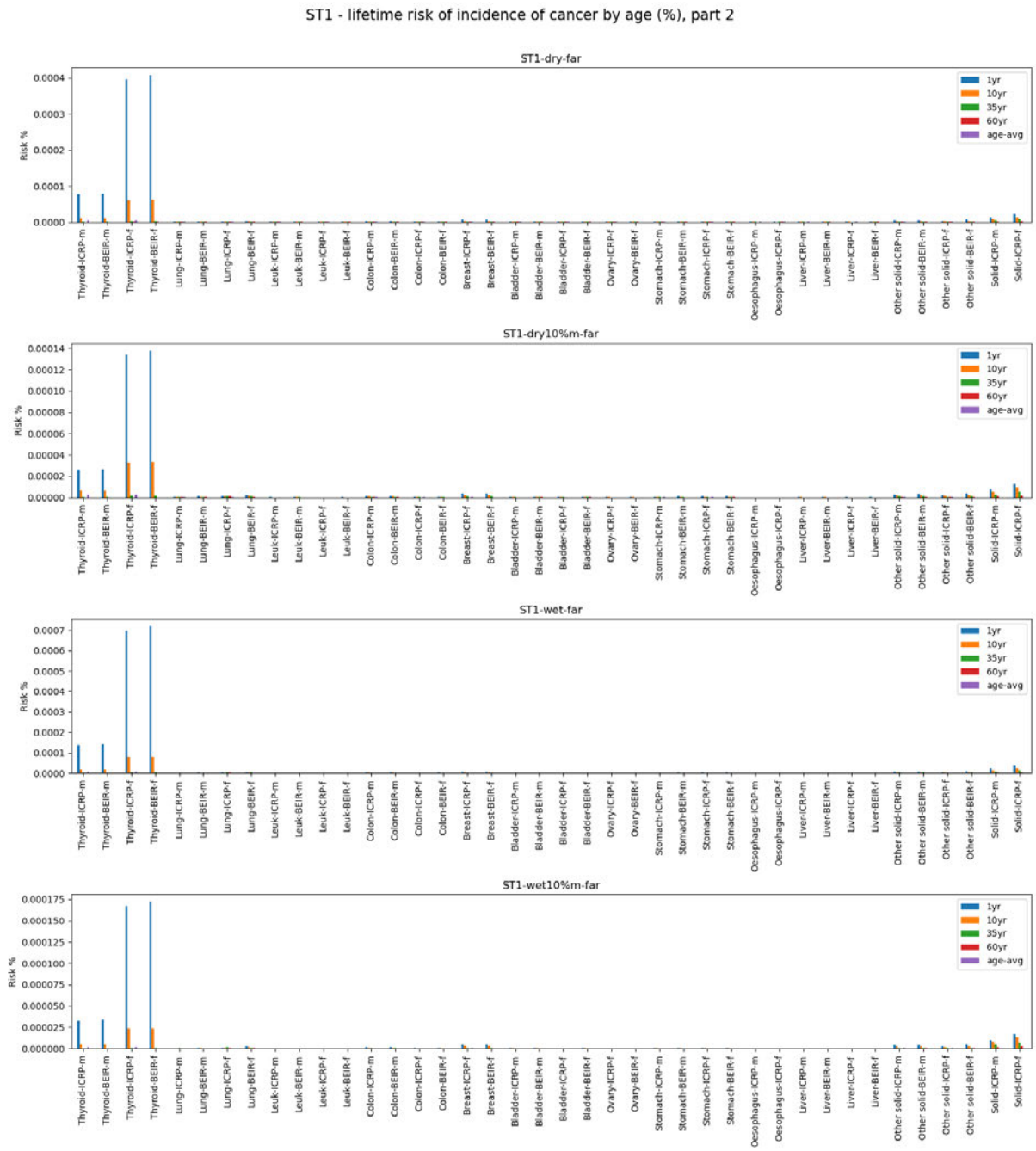


Figure 5 ST1-DBA-far – all predicted risks of incidence of cancers using both ICRP and BEIR models

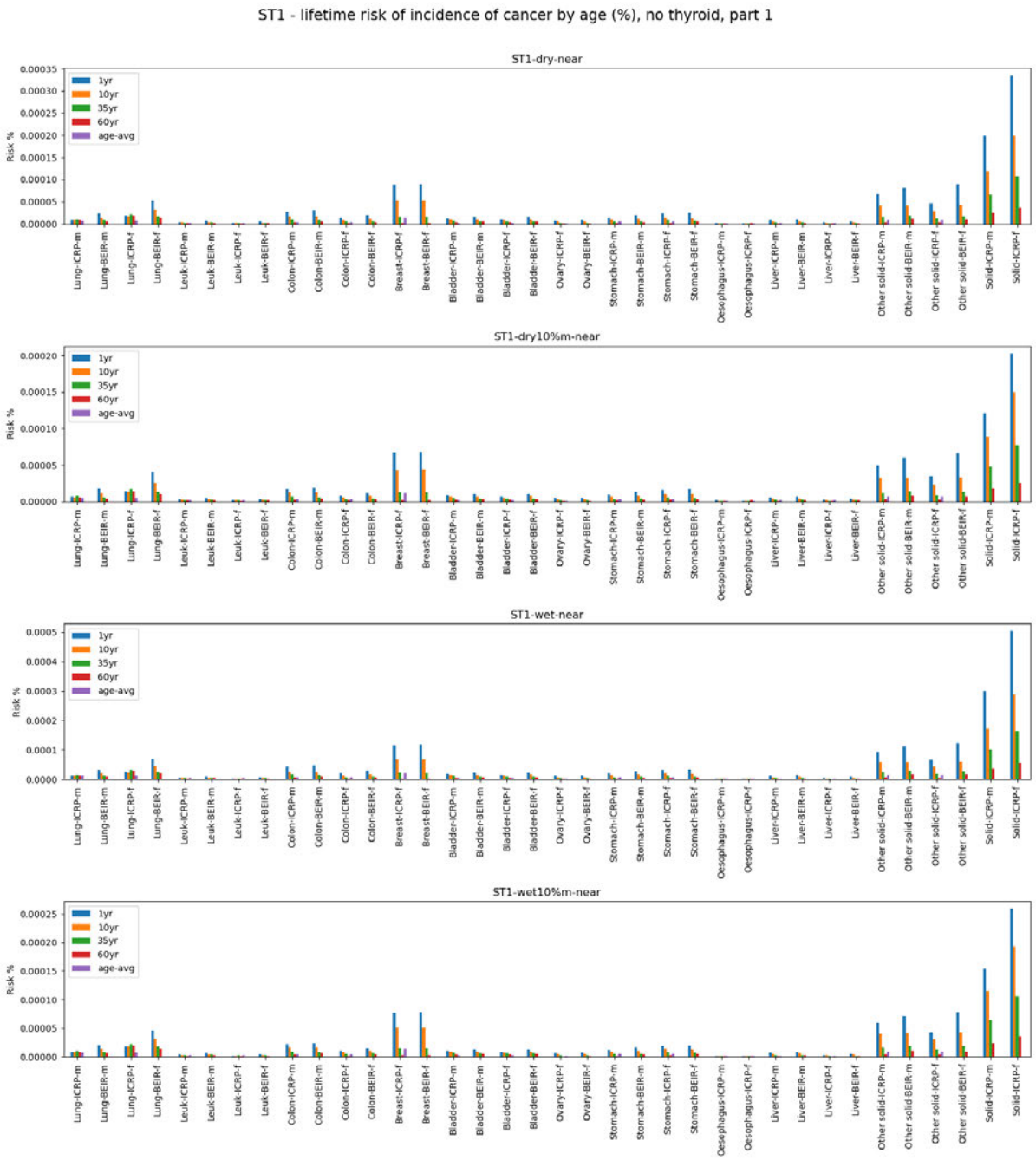


Figure 6 ST1-DBA-near – predicted risks of incidence of cancers using both ICRP and BEIR models, omitting the thyroid

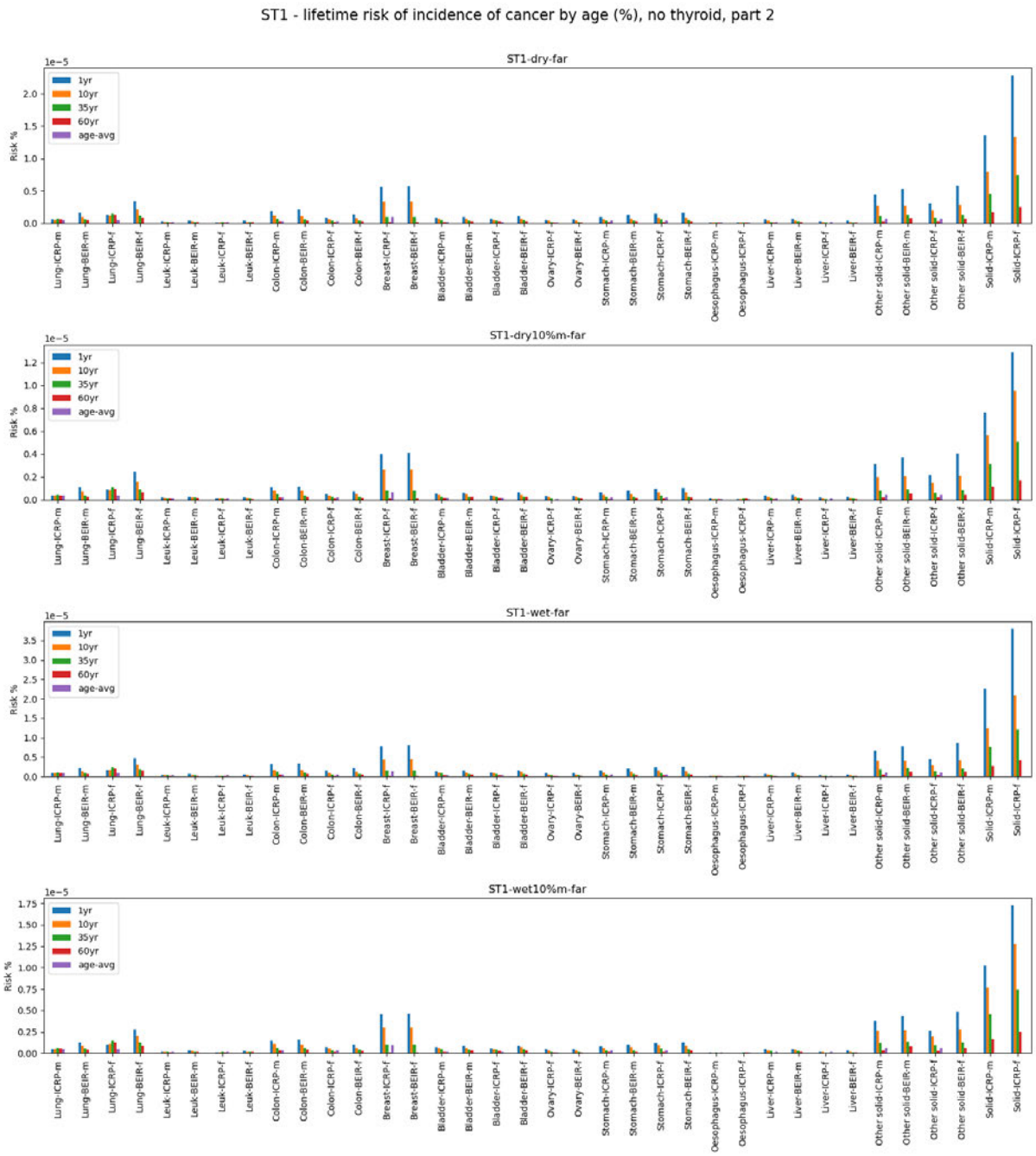


Figure 7 ST1-DBA-far – predicted risks of incidence of cancers using both ICRP and BEIR models, omitting the thyroid

5.4.2 Fatalities

The predicted risks of cancer fatalities are shown in Figure 8 and Figure 9. As with the incidence risks, the thyroid dominates. To obviate this, Figure 10 and Figure 11 show the same results without thyroid. As noted in Section 3.6, there is no mortality model for thyroid cancer in ICRP Publication 103 so only results based on multiplying the cancer incidence risk with an averaged lethality fraction ('inc' results) are presented for the thyroid.

Relative risks of fatality are consistent between examples (i.e. ignoring the absolute values, all the graphs appear very similar in pattern). The following features can be observed:

- As with incidence, the risk of fatalities is dominated by thyroid. However, the lethality fraction assumed is low (7%), so it is less dominant.
- The use of a lethality fraction ('inc' results) gives predictions that are broadly consistent with the modelled risks ('mort') for most tissues. For lung and oesophagus, the 'inc' predictions lack the age dependency that is present in the 'mort' prediction, while for the bladder the opposite is found.
- For thyroid, lung ('mort'), colon, breast, bladder ('inc'), ovary, oesophagus ('mort'), stomach, liver and remainder (other solid), there is a clear age dependency, with generally 1-year-old having the most risk, followed by 10-year-old, 35-year-old, and 60-year-old. For the other tissues including lung ('inc') the relationship is either not strong, or not consistent, or risks are so small that the relationship is not discernible.
- For thyroid, lung, stomach, and other solid ('mort'), the female risk is higher than the male. For bone marrow ('Leuk'), colon, bladder, oesophagus, and liver, the male risks are larger or the same, or the risks are so small that differences are not discernible.
- Assuming 10% local milk consumption reduces the thyroid cancer fatality risk of a 1-year-old to 25-35% of the risk calculated using 50% local milk consumption, with smaller reductions to older age groups where discernible. Sex makes little difference. For other cancer sites the reduction is to 60-80%, and age and sex generally do not make much difference.

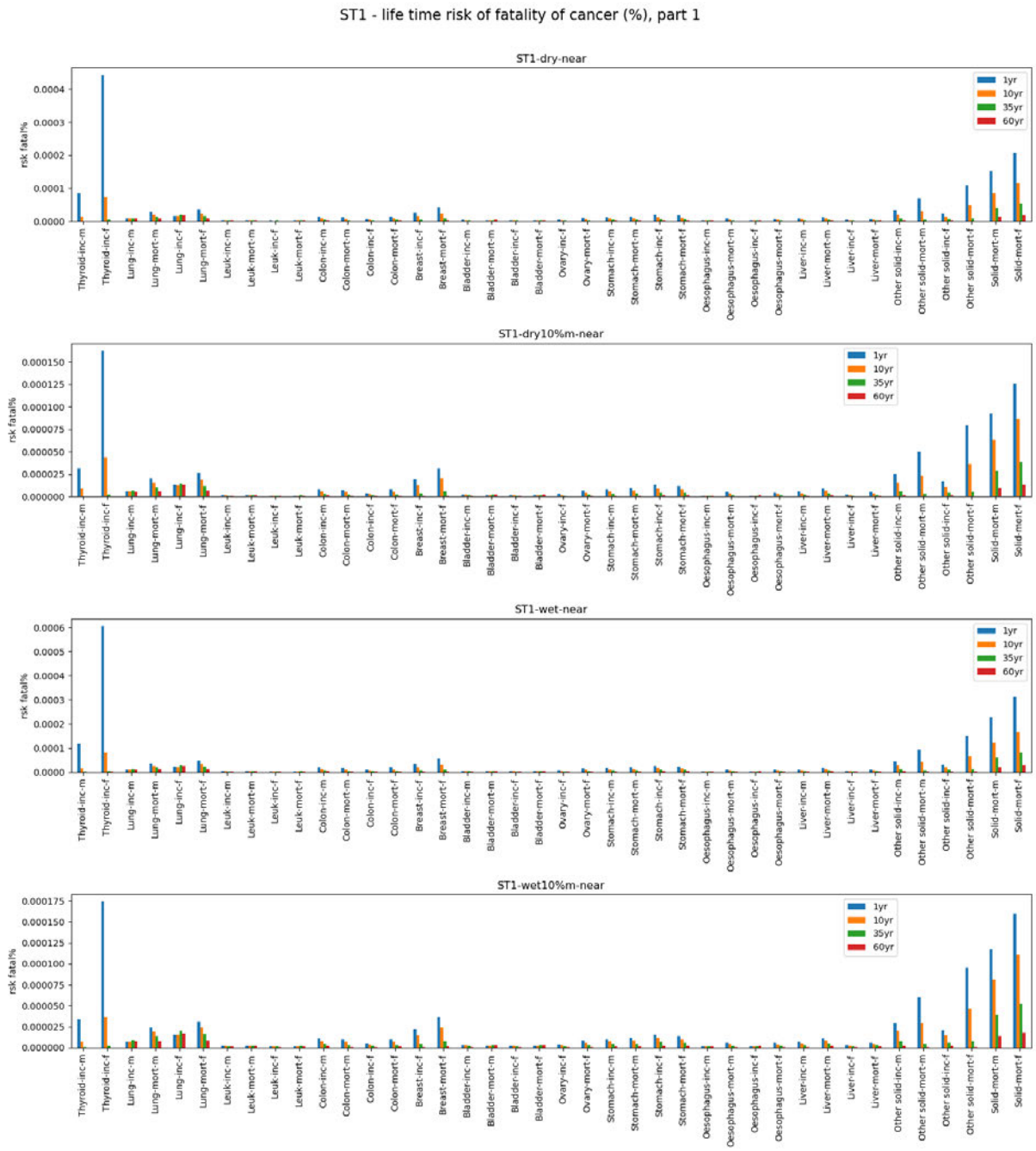


Figure 8 ST1-DBA-near – all predicted risks of fatality of cancers. Risks are calculated either by applying an age/sex independent lethality fraction to the corresponding risk of incidence (labelled 'inc'), or by applying ICRP mortality risk models (labelled 'mort').

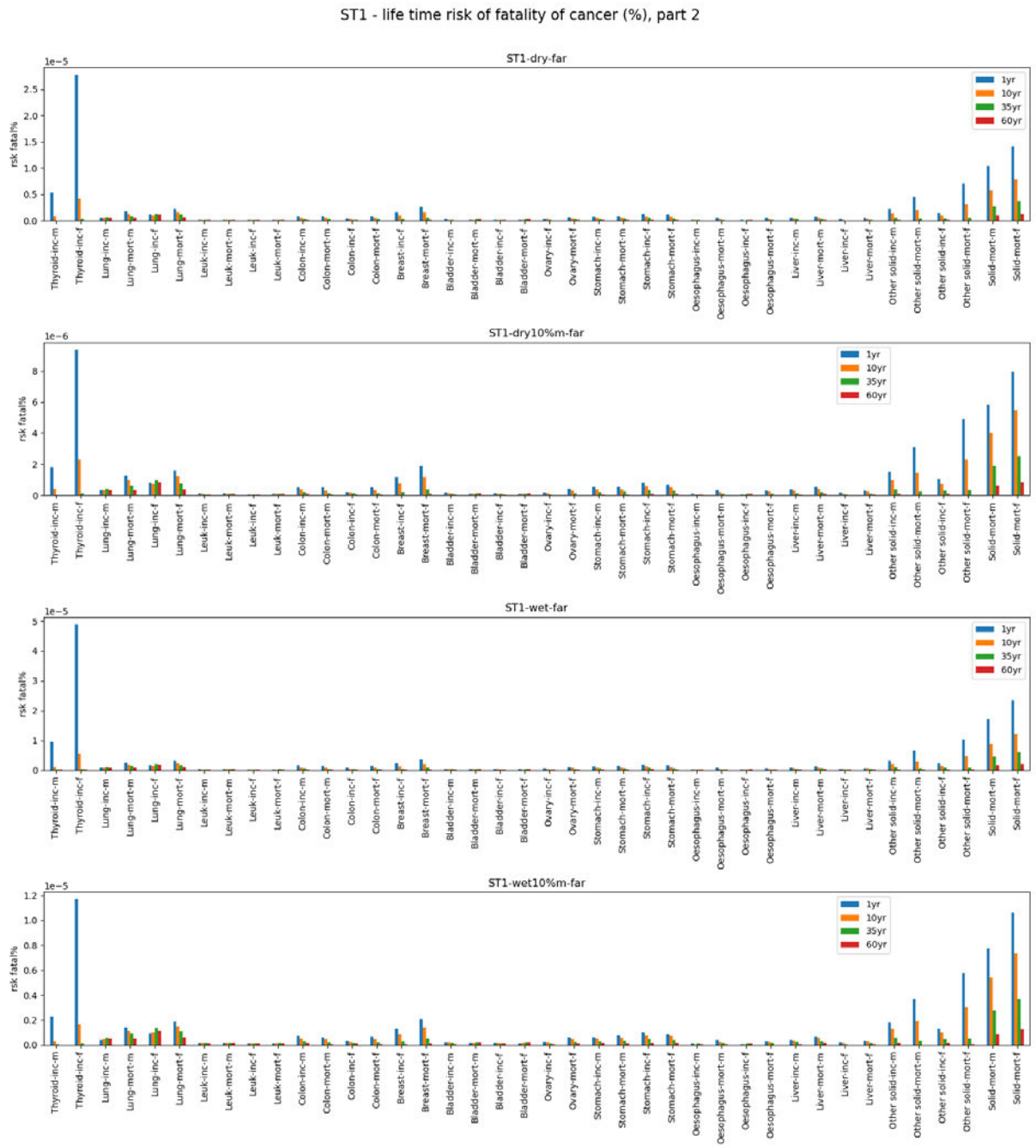


Figure 9 ST1-DBA-far – all predicted risks of fatality of cancers. Risks are calculated either by applying an age/sex independent lethality fraction to the corresponding risk of incidence (labelled 'inc'), or by applying ICRP mortality risk models (labelled 'mort').

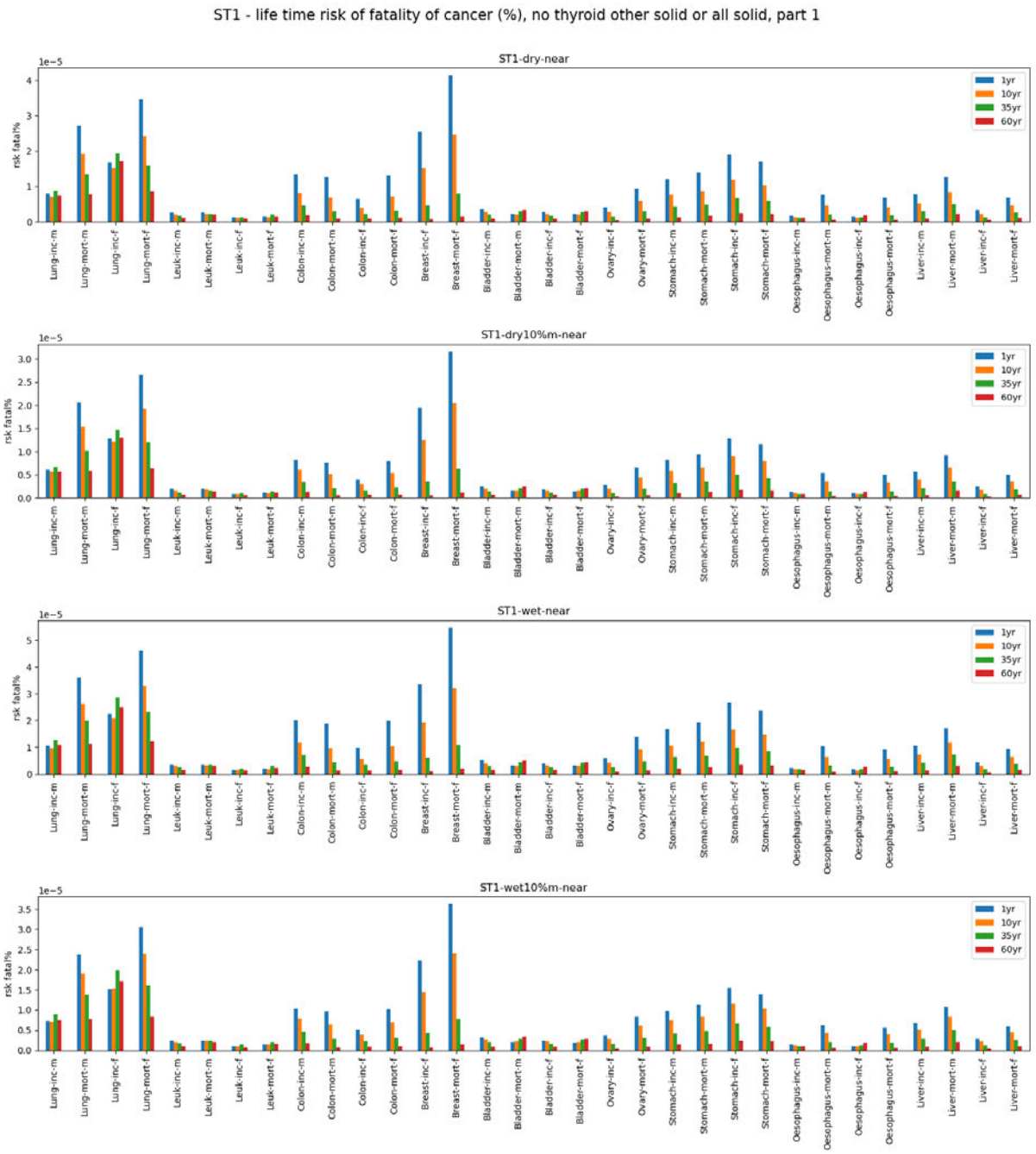


Figure 10 ST1-DBA-near – predicted risks of fatality of cancers. Risks are calculated either by applying an age/sex independent lethality fraction to the corresponding risk of incidence ('inc') or by applying ICRP mortality risk models ('mort'). Omitting the thyroid, remainder ('Other solid') and all-solid-cancer ('Solid') risks.

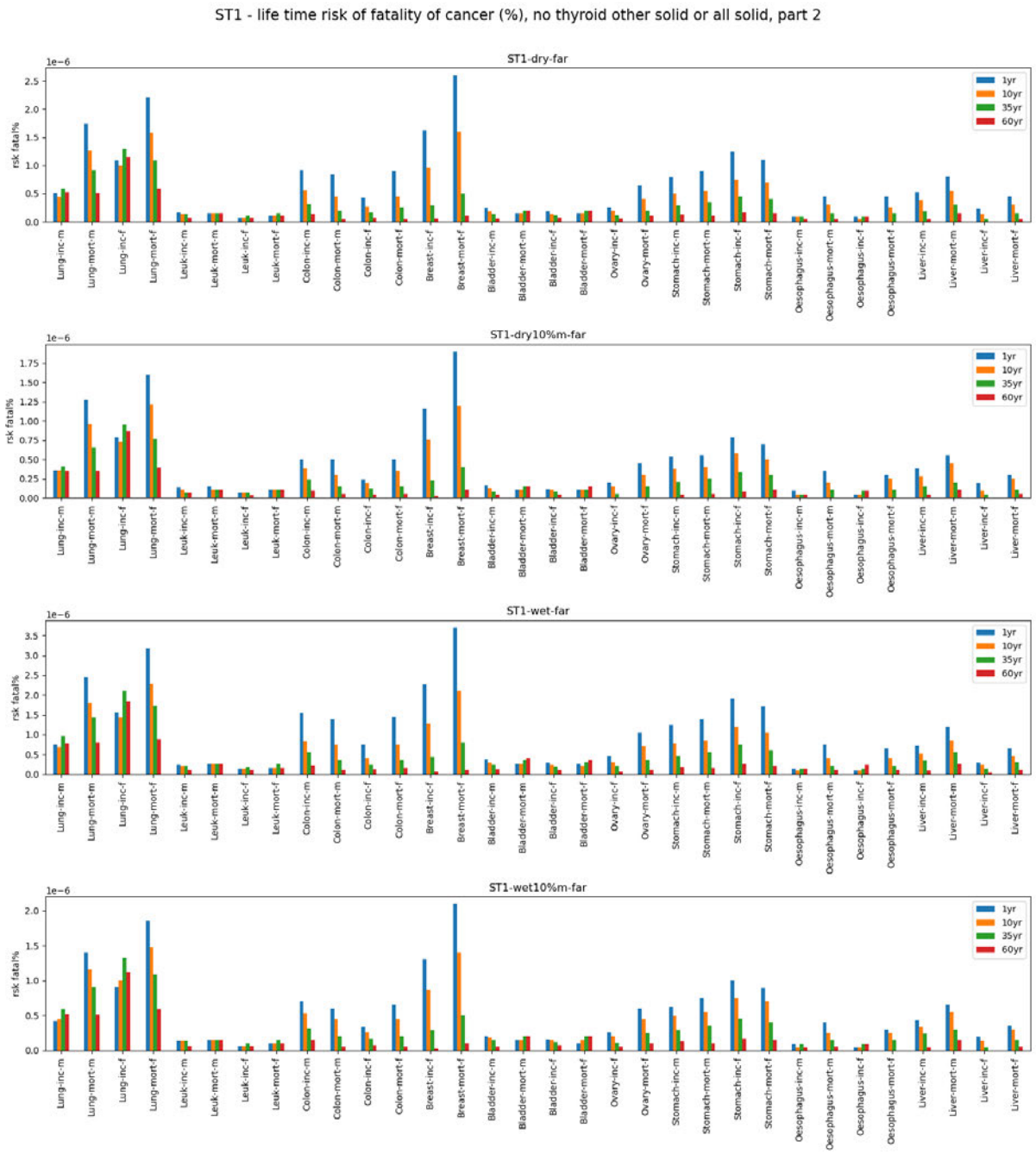


Figure 11 ST1-DBA-far – predicted risks of fatality of cancers. Risks are calculated either by applying an age/sex independent lethality fraction to the corresponding risk of incidence ('inc') or by applying ICRP mortality risk models ('mort'). Omitting the thyroid, remainder ('Other solid') and all-solid-cancer ('Solid') risks.

5.4.3 Detriment

As with predicted incidence and fatality risks, the predicted risk of detriment is dominated by thyroid as shown in Figure 12 and Figure 13, and Figure 14 and Figure 15 show the same results without the thyroid.

The relative risks of detriment are consistent between examples (i.e. ignoring the absolute values, all the graphs appear very similar). The following features can be observed:

- As with incidence and fatality, the predicted risk of detriment is dominated by thyroid.
- For thyroid, bone marrow ('Leuk'), colon, breast, bladder, ovary, stomach, liver and remainder ('Other solid'), there is a clear age dependency, with 1-year-old having the most risk, followed by 10-year-old, 35-year-old, and 60-year-old. For the other tissues the relationship is either not strong, or not consistent, or risks are so small that the relationship is not discernible.
- For thyroid, lung and stomach, the female risks are larger, whereas for all other tissues the male risks are larger or the same, or the risks are so small that differences are not discernible.
- Assuming 10% local milk consumption reduces the thyroid cancer detriment risk of a 1-year-old to 25-35% of the risk calculated using 50% local milk consumption, with apparently smaller reductions for older age groups where discernible. Sex makes little difference. For other cancer sites the reduction is to 60-80% age and sex generally do not make much difference.

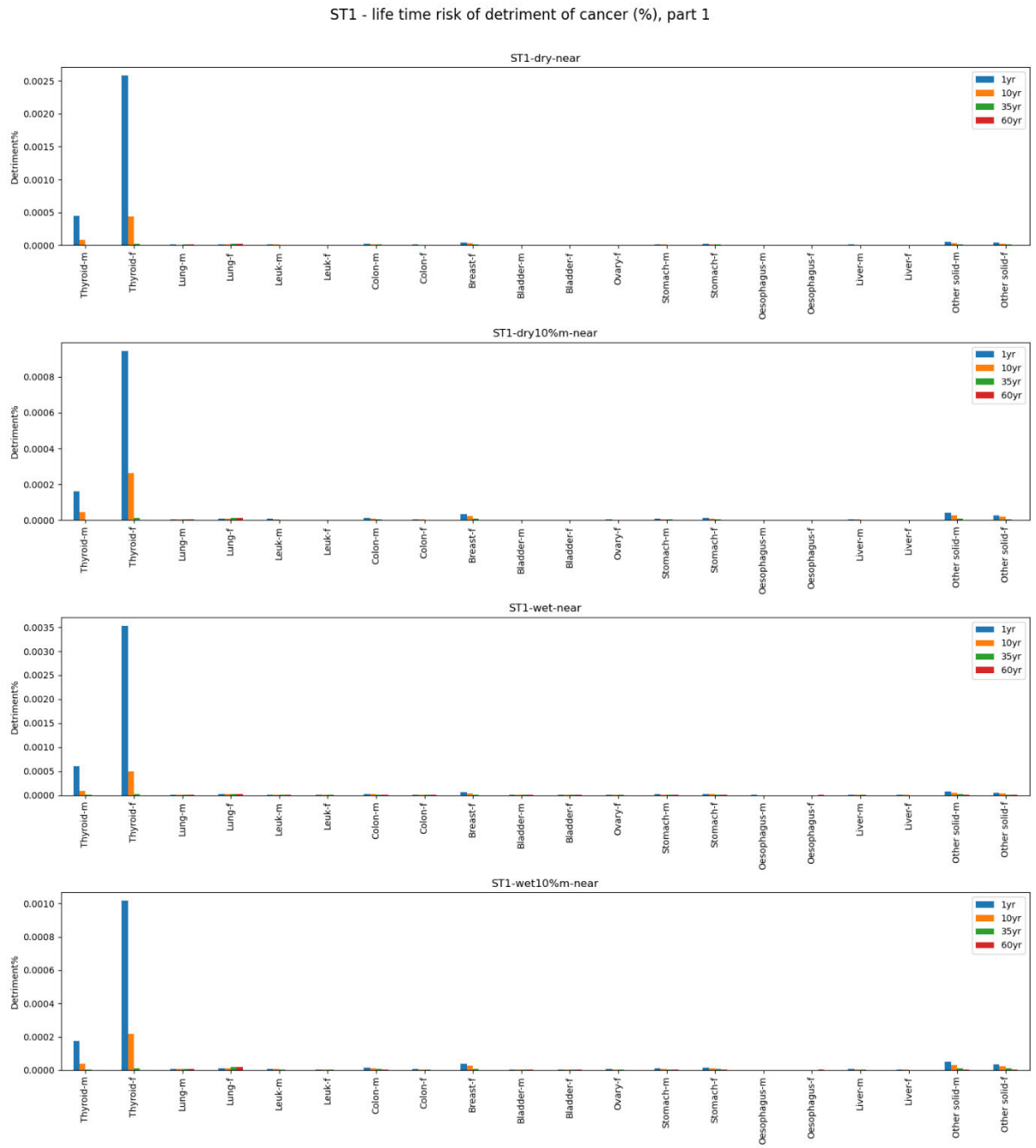


Figure 12 ST1-DBA-near – all predicted risks of detriment of cancers

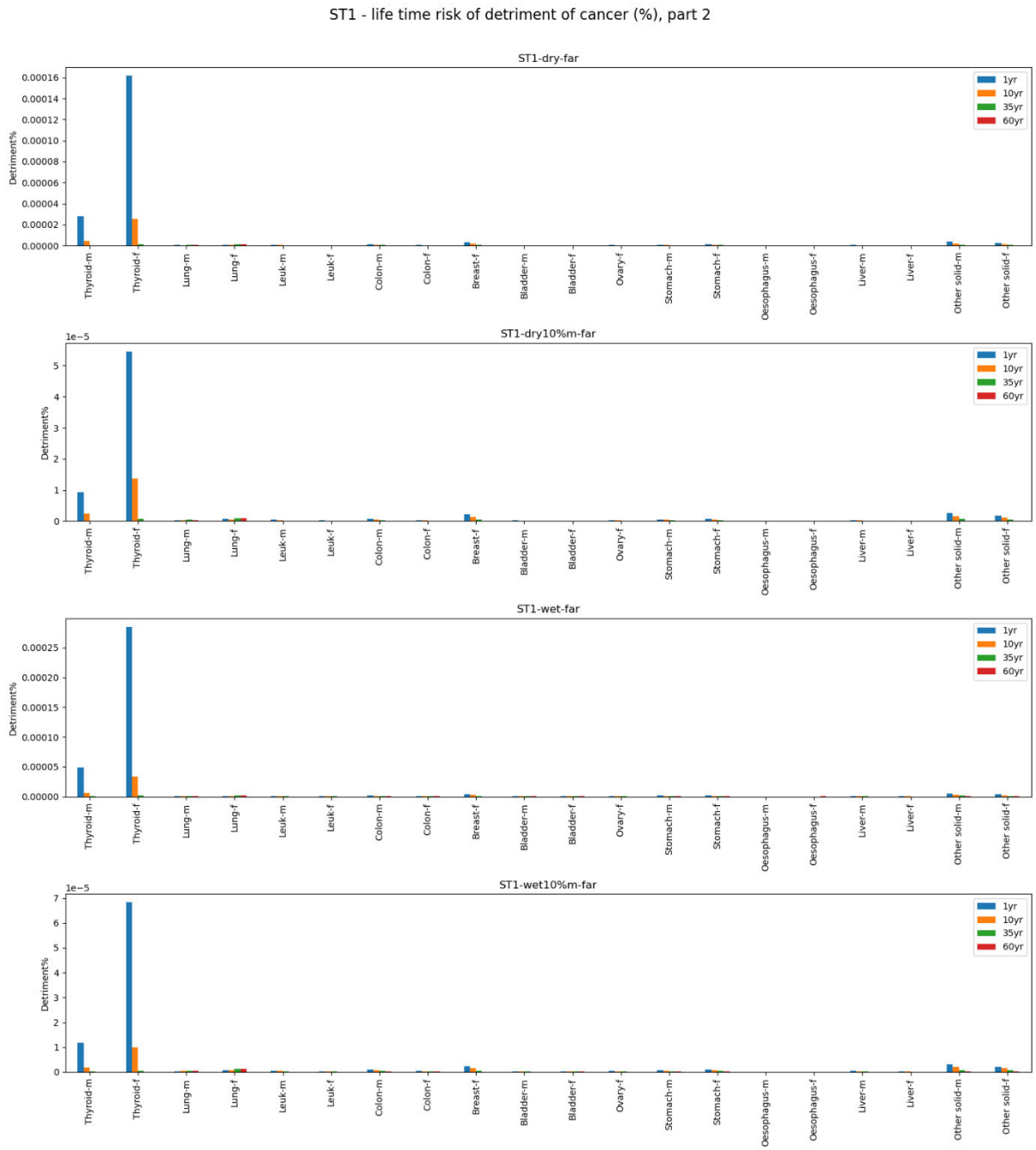


Figure 13 ST1-DBA-far – all predicted risks of detriment of cancers

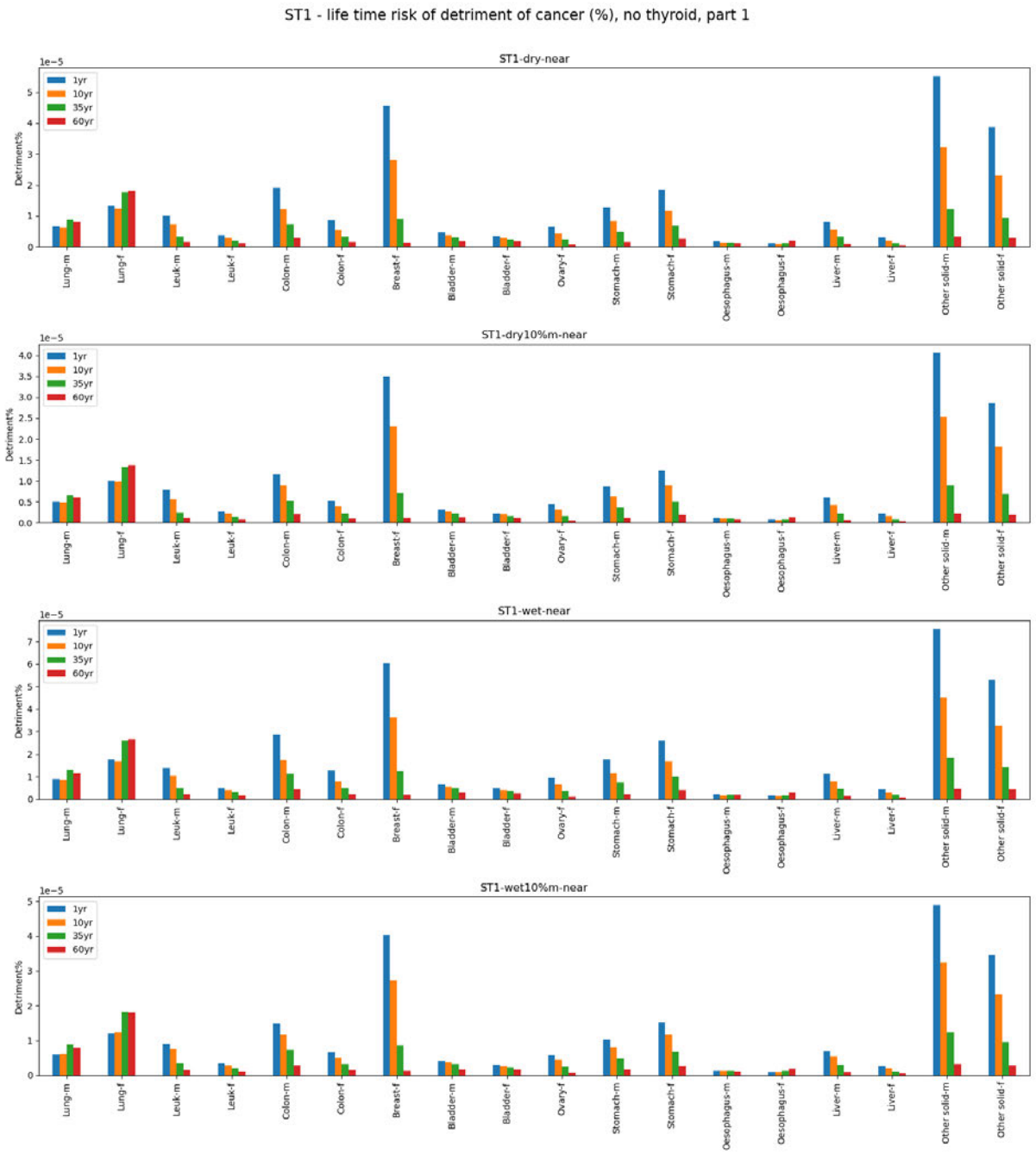


Figure 14 ST1-DBA-near – predicted risks of detriment of cancers, omitting thyroid risks

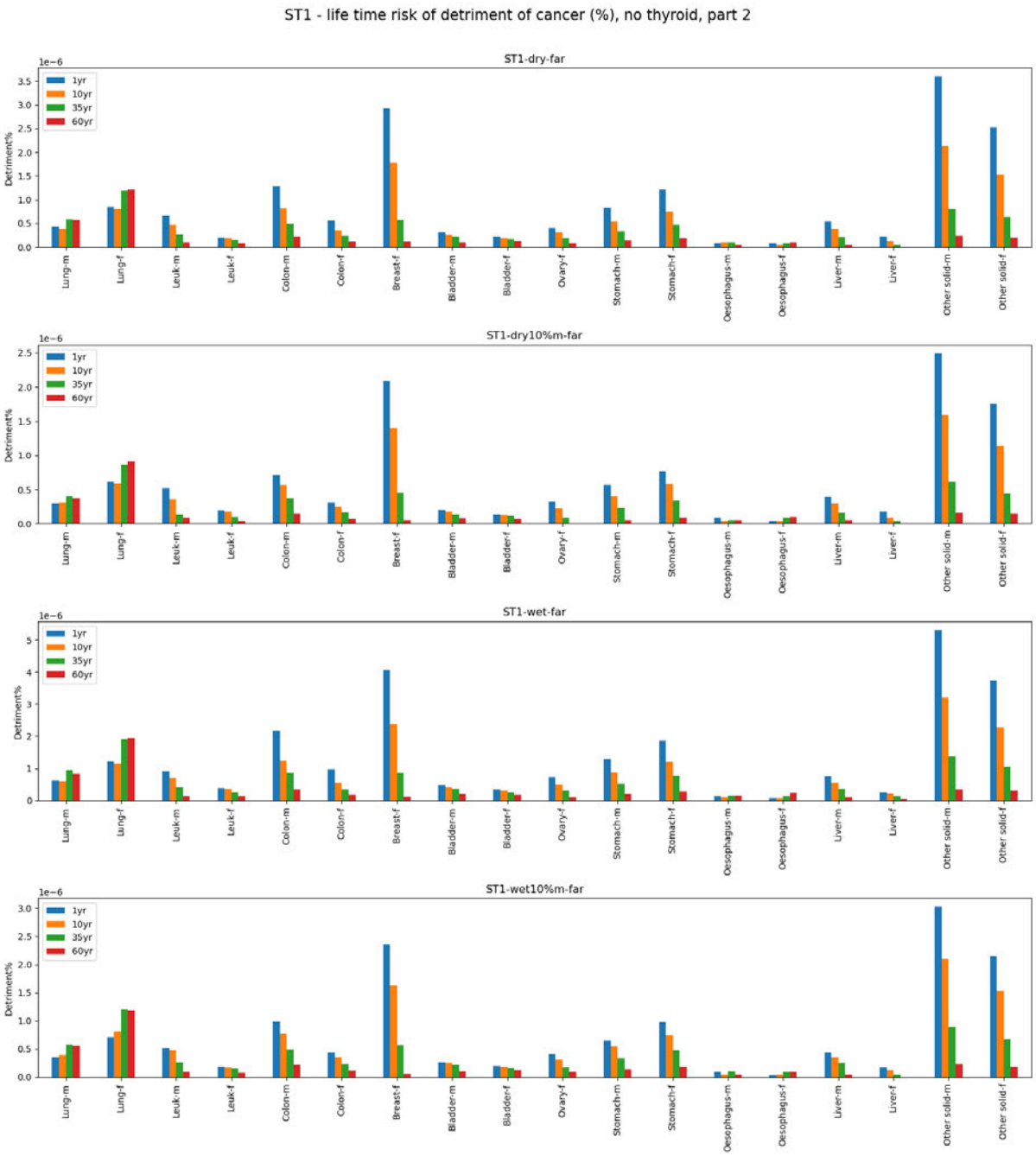


Figure 15 ST1-DBA-far – predicted risks of detriment of cancers, omitting thyroid risks

5.5 Summary

The overall doses in all the ST1 examples to all age groups are within a range in which protective actions are not required and the corresponding increased risks of incidence (Figure 4 to Figure 7) would be unlikely to be detectable in an epidemiological study. The most significantly exposed organ by far is the thyroid, and the dose is dominated by ^{131}I.

Thyroid cancer incidence risks show a large age dependency. The risk to 1-year-olds is 5 to 10 times the risk to a 10-year-old of the same sex, 50 to 150 times the risk to a 35-year-old of the same sex, and 10 to 130 times the risks estimated using an age/sex-averaged approach

(applied to an adult dose). The thyroid is the only organ where the doses showed a strong age dependency with the 1-year-dose up to 9 times larger than the 35-year-old, depending on the example and the assumption about whether 10 or 50% of milk consumed is locally produced.

There is also a strong sex dependency on the incidence of thyroid cancer, with female risks appearing about 4-6 times the corresponding male risks. Since the same dose was used in the calculation, this difference derives entirely from the risk models.

Other tissues show age and sex dependency in incidence risk, but not nearly as extreme as thyroid.

Employing a BEIR VII model instead of ICRP makes very little difference overall to risk of incidence in any tissue. The most obvious differences are for the lung, where there is an age-dependency in the BEIR VII prediction which is not apparent in the ICRP results, meaning while adult risks are similar, BEIR VII predicts higher risks to 1-year-olds and 10-year-olds by a factor of 2-3 but note the comments in Section 9.6.

Thyroid cancer is not a particularly lethal form of cancer and ICRP have no model of thyroid cancer fatality risk, therefore fatality risk is estimated by applying an age and sex independent lethality fraction of 7% and consequently the relative age and sex dependency of thyroid cancer incidence risk carries through unchanged to fatality risk.

Generally, fatality risk calculated with a lethality fraction applied to incidence risk (‘inc’), compared to modelled fatality risk (‘mort’) are in reasonable agreement, mostly within a factor of about 3 with a tendency for risks based on the ICRP model (‘mort’) to be higher.

Thyroid cancer fatality risk is predicted in all examples to be the most significant. However, other more lethal forms of cancer are also significant. Some tissues such as breast and lung also exhibit age and sex dependency, but it is hard to find examples where this is as extreme as thyroid.

In terms of detriment risk, thyroid is by far the most dominant form of cancer. Since all the factors for calculating detriment are age and sex independent there is the same strong age and sex dependency seen for the incidence and fatality risk.

Figure 16 and Figure 17 show the predicted age and sex-specific risks of incidence and fatality and detriment summed across all organs considered in the study compared to the age and sex- averaged detriment calculated by multiplying the age-specific effective dose with the ICRP nominal risk coefficient for cancer detriment of 5.5% Sv^{-1} (ICRP, 2007a). The nominal risk coefficient is a sex-averaged and age-at-exposure-averaged lifetime risk estimate per unit dose for a representative population. As seen in the Figures, females are calculated to have higher risks of incidence and detriment compared to males of the same age. Overall, the predicted risk of incidence of cancer, particular for the younger females, are much higher than those calculated using the nominal detriment value.

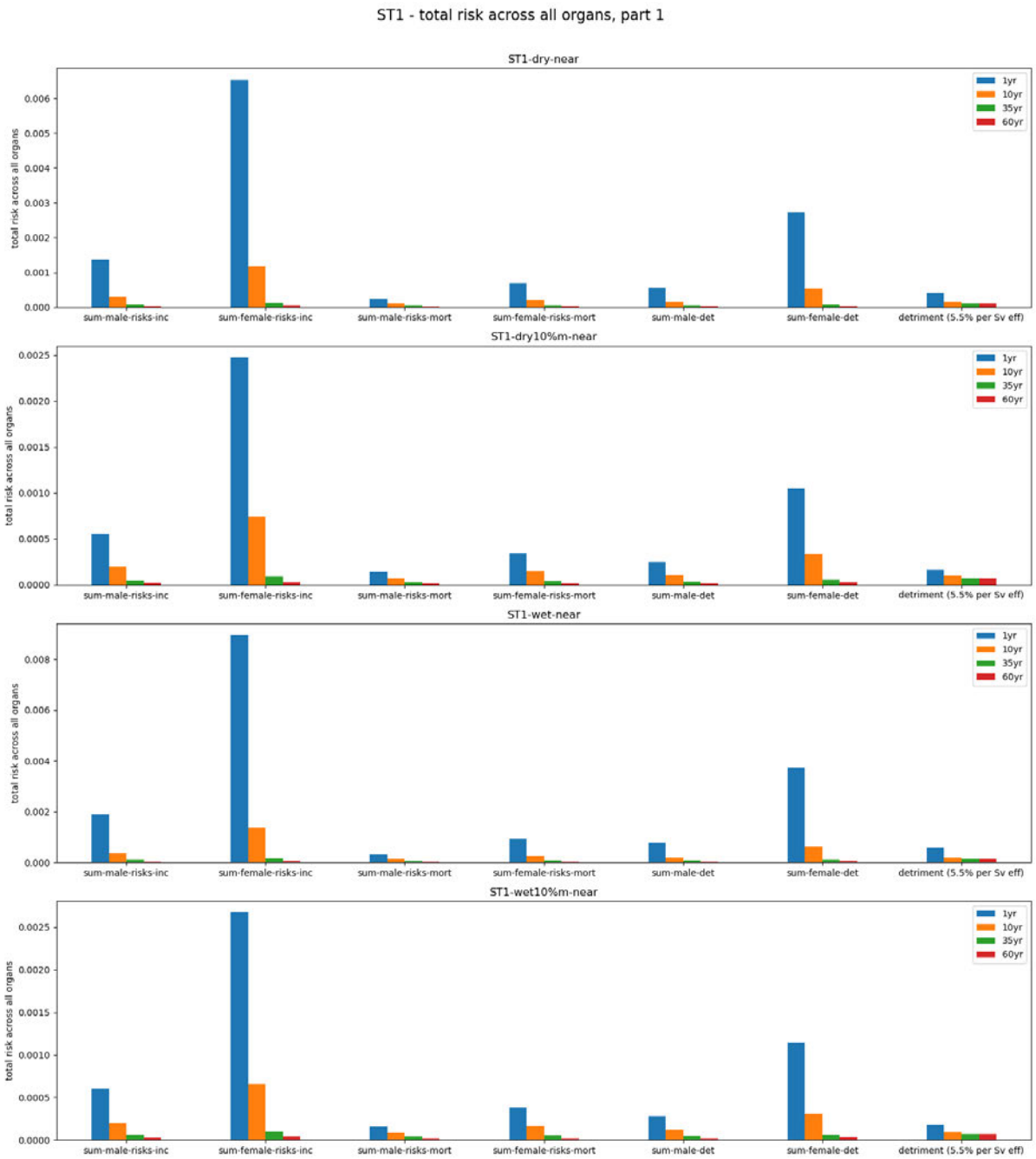


Figure 16 ST1-DBA-near – Age and sex-specific risks of cancer and detriment summed across tissues in the study compared with detriment calculated by applying ICRP nominal risk coefficient of 5.5% Sv^{-1} to the age-specific effective dose

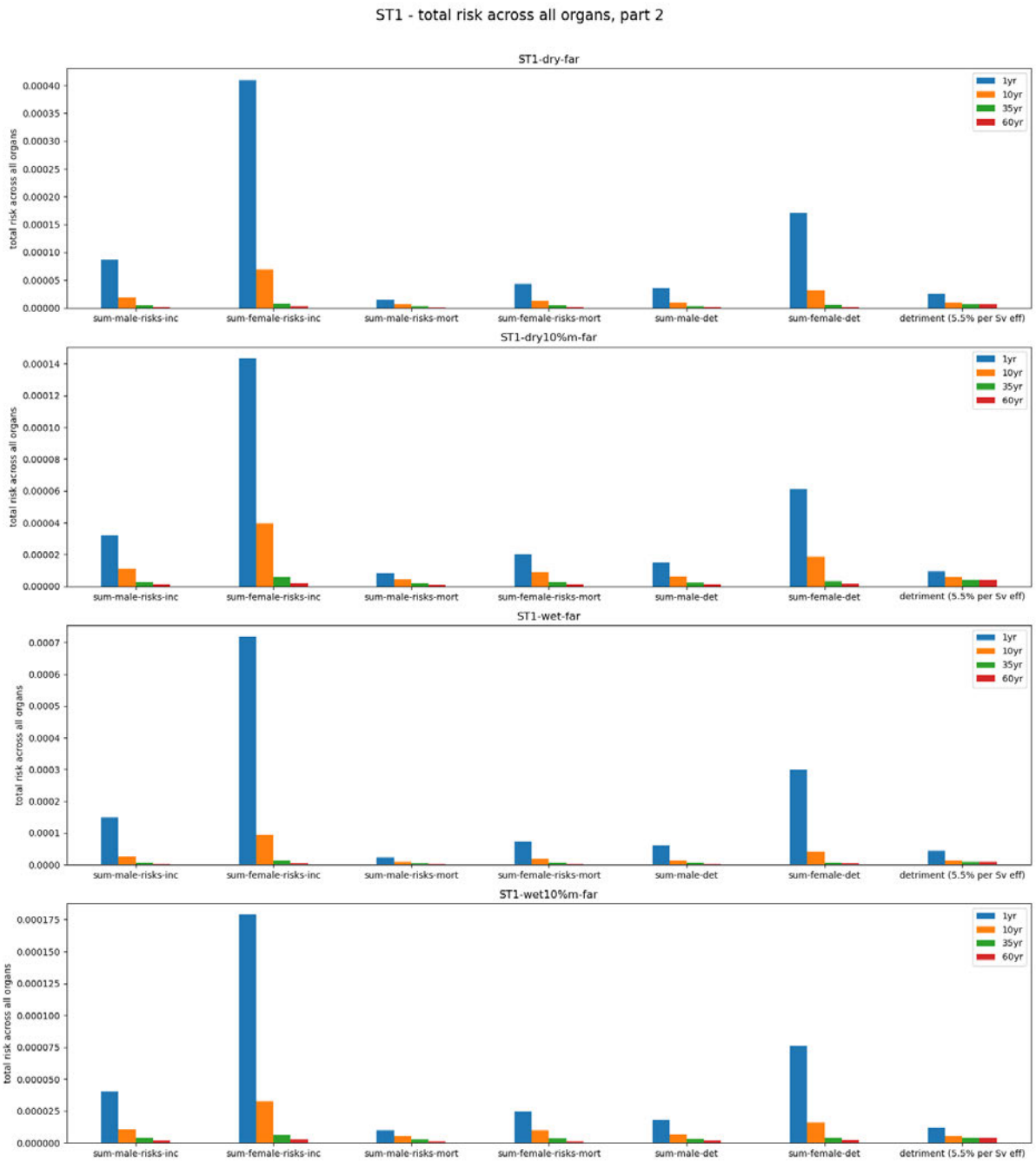


Figure 17 ST1-DBA-far – Age and sex-specific risks of cancer and detriment summed across tissues in the study compared with detriment calculated by applying ICRP nominal risk coefficient of 5.5% Sv^{-1} to the age-specific effective dose

6 Source term 2 – ST2-acute

The second source term was provided by ONR to represent a light water reactor, severe accident, and is a much more severe and shorter accident than ST1-DBA. It is a generic

severe accident at a large PWR and it should not be taken as representative of or specific to any particular facility being designed, built or operated.

It is divided into three phases. During Phase 1 there is no release of radioactivity. Phase 2 contains the bulk of the release with a 1-hour duration. Phase 3 represents the tail of the release of 10 hours duration. The effective height of the release is 10 m, and iodine isotopes are released in the fractions of 30% elemental, 45% organic and 25% as components of aerosol, which are defaults recommended by FRMAC (2015). For this project it is termed ST2-acute.

Table 3 Phases and releases of ST2-acute (Bq)

Radionuclide	Phase 1 0-1 hour	Phase 2 1-2 hours	Phase 3 2-12 hours
Kr-88	-	3E+17	1E-01
Zr-95	-	2E+15	1E+10
Ru-103	-	3E+16	1E+11
Ru-106	-	9E+15	5E+10
Ag-110m	-	2E+14	5E+08
Te-132	-	6E+16	4E+10
I-131	-	2E+18	4E+16
I-132	-	3E+18	4E+16
I-133	-	3E+18	5E+14
I-134	-	2E+17	2E-26
I-135	-	9E+17	3E+09
Xe-135	-	3E+18	2E+13
Cs-134	-	8E+15	3E+10
Cs-136	-	2E+15	5E+09
Cs-137	-	5E+15	2E+10
Ba-140	-	8E+15	0E+00
Ce-144	-	2E+15	6E+09
Pu-238	-	6E+12	2E+07
Pu-241	-	2E+14	7E+08
Cm-242	-	8E+13	5E+08
Cm-244	-	6E+12	4E+07

6.1 Dry deposition

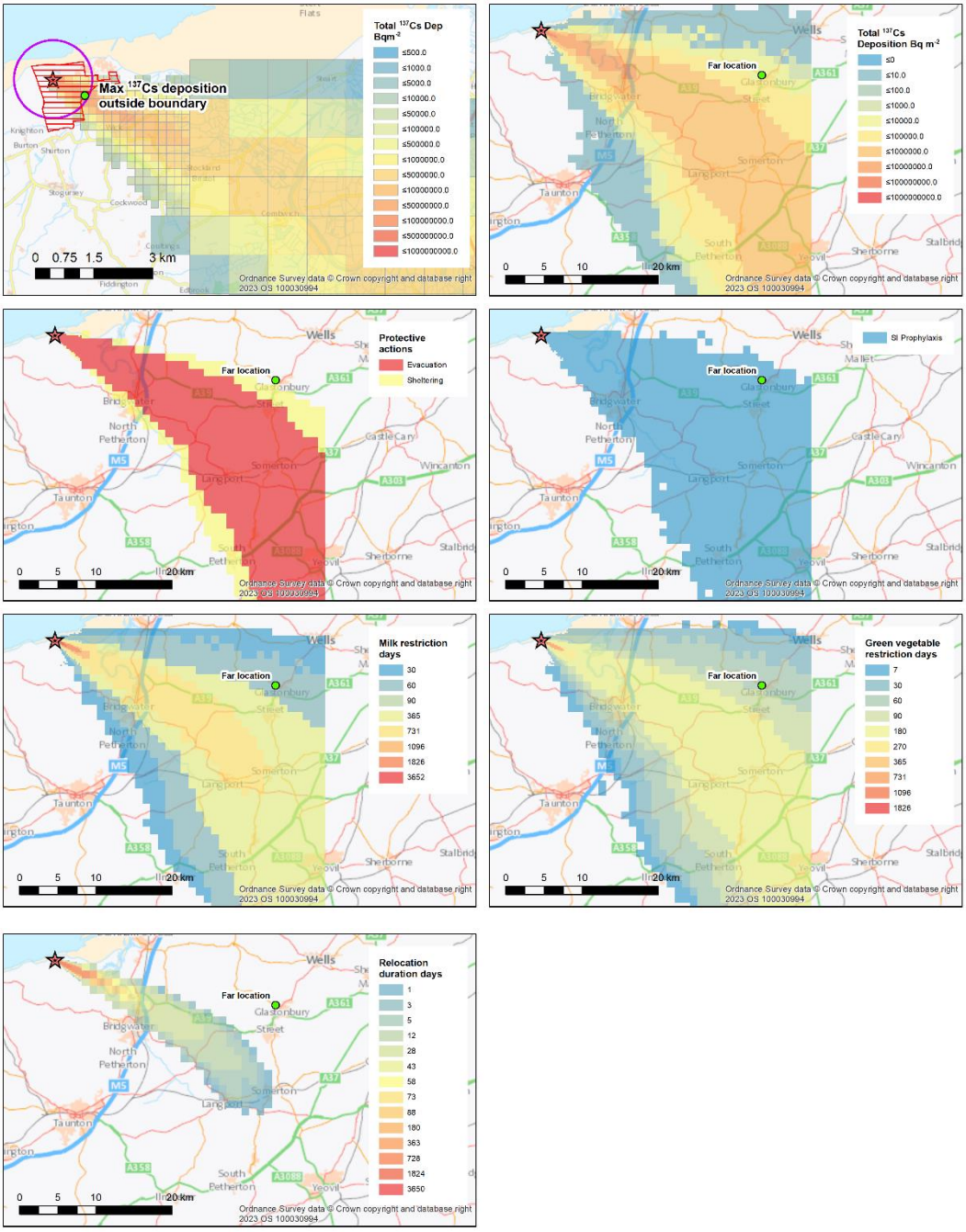


Figure 18 Predicted consequences of the dispersal of ST2-acute for a model run beginning 16:00 11/6/21 giving dry conditions

The weather sequence selected as dry begins at 16:00hr 11/6/21. The pattern of total deposition of ^{137}Cs, and the extent and duration of protective actions are shown in Figure 18.

The near location was placed in the grid-square with the highest total deposition outside of the boundary fence and is towards the southeast where no wet deposition is predicted. Based on the upper ERL criteria, evacuation and the administration of stable iodine would be recommended, relocation being required for five years (based on a criterion 20mSv y^{-1} and

assuming no clean-up is performed). In the highest deposition grid-square, milk restrictions are predicted to be required for 10 years (because of long-lived radionuclides, and assuming that no other agricultural protective actions are applied), green vegetables restriction for 3 years, and the restrictions on all foods ranging between 6 months and 50 years.

The far location was placed off the plume centre line towards the east where there is not wet deposition, in the grid square with the maximum total deposition at 30 km that is outside the predicted zones where sheltering and evacuation would be required (based on upper ERL criteria) and outside the zone where relocation would be required without clean-up (based on a criterion of 20mSv y^{-1}). Here the proportion of wet deposition is zero. Stable iodine administration would be advised based on the upper ERL. Milk and green vegetable restrictions are predicted to be required for 60 days, and the predicted restrictions on all foods do not exceed 60 days.

6.2 Wet deposition

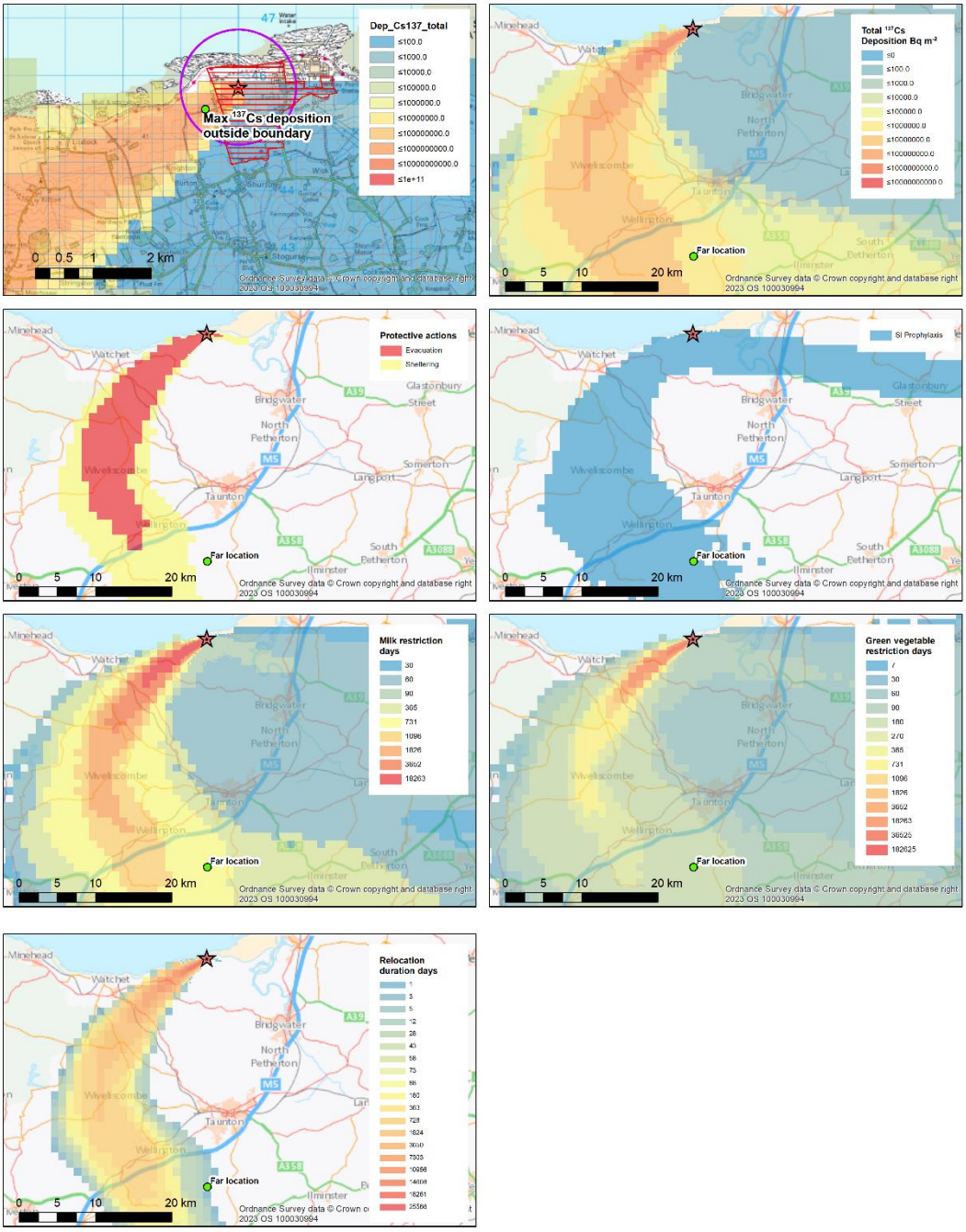


Figure 19 Predicted consequences of dispersal of ST2-acute. The model run beginning 08:00 30/7/19, giving predominantly wet conditions.

The weather sequence selected as predominantly wet begins 08:00 30/7/19. Figure 19 shows the deposition patterns and the extent of duration and extent of protective actions.

The near location was placed in the grid-square with the maximum deposition outside of the boundary and is towards the west, where approximately 98% of the deposition is due to rainfall. Based on the upper ERL criteria, evacuation and stable iodine prophylaxis would be advised, and relocation being required for 70 years (based on a criterion of 20mSv y^{-1} and

assuming no clean-up is performed). Similarly, in this highest deposition grid-square milk restrictions are predicted to be required for 50 years (because of long-lived radionuclides, and assuming that no other agricultural protective actions are applied), green vegetable restrictions for 100 years, and predicted restrictions for all foods ranging between 50 years and 500 years.

The far location was placed off the plume centre line, in the grid square with the maximum deposition at 30 km, that is outside the zones where sheltering and evacuation would be required (based on upper ERL criteria) and outside the zone where relocation would be required without clean-up (based on a criterion of 20 mSv y^{-1}). It is towards the south where more than 99% of the deposition is due to rainfall. Based on the upper ERL criteria, stable iodine prophylaxis would be advised. Milk restrictions are predicted to be required for 2 years, green vegetable restrictions for 6 months and the predicted restriction of all foods range between none and 5 years.

6.3 Dose profiles

Predicted total effective doses at the near locations range from 84 to 250 Sv (see Table A2). These are doses at which deterministic or acute effects would be expected and this is discussed further in Section 6.4. At the far location effective doses range from 8.5 to 55 mSv well below the range in which deterministic effects are expected.

In all examples the thyroid dose is the largest. In the dry examples, other organ doses are relatively very small compared to the thyroid, whereas in the wet examples, the other organ doses are still relatively smaller but are about 30-50% of the thyroid dose.

There is a clear age-dependency in the thyroid dose in all examples, with the 1-year-old dose being between about twice the corresponding adult thyroid dose. The other tissues show some age dependency in the wet examples with 1-year-old dose being between about 20-100% larger than the 35-year-old one. The dry examples do not show the same level of dependency in the other organs.

The requirement for food restrictions means that the assumptions concerning the proportion of locally produced milk consumed make very little difference to doses.

A more detailed description of the dose profiles can be found in Appendix A2.

6.4 Deterministic effects

The doses at the near location are very high and within the range at which deterministic or acute health effects are expected.

In the ST2-acute-dry example, PACE predicts a non-zero risk of haematopoietic syndrome, pre-natal and neo-natal death, and skin burn mortality. In addition, skin burn, mental retardation, cataract, and hypothyroidism morbidity risks were predicted. Figure 20 shows the predicted extents, some of which encompass the near location but does not reach the far.

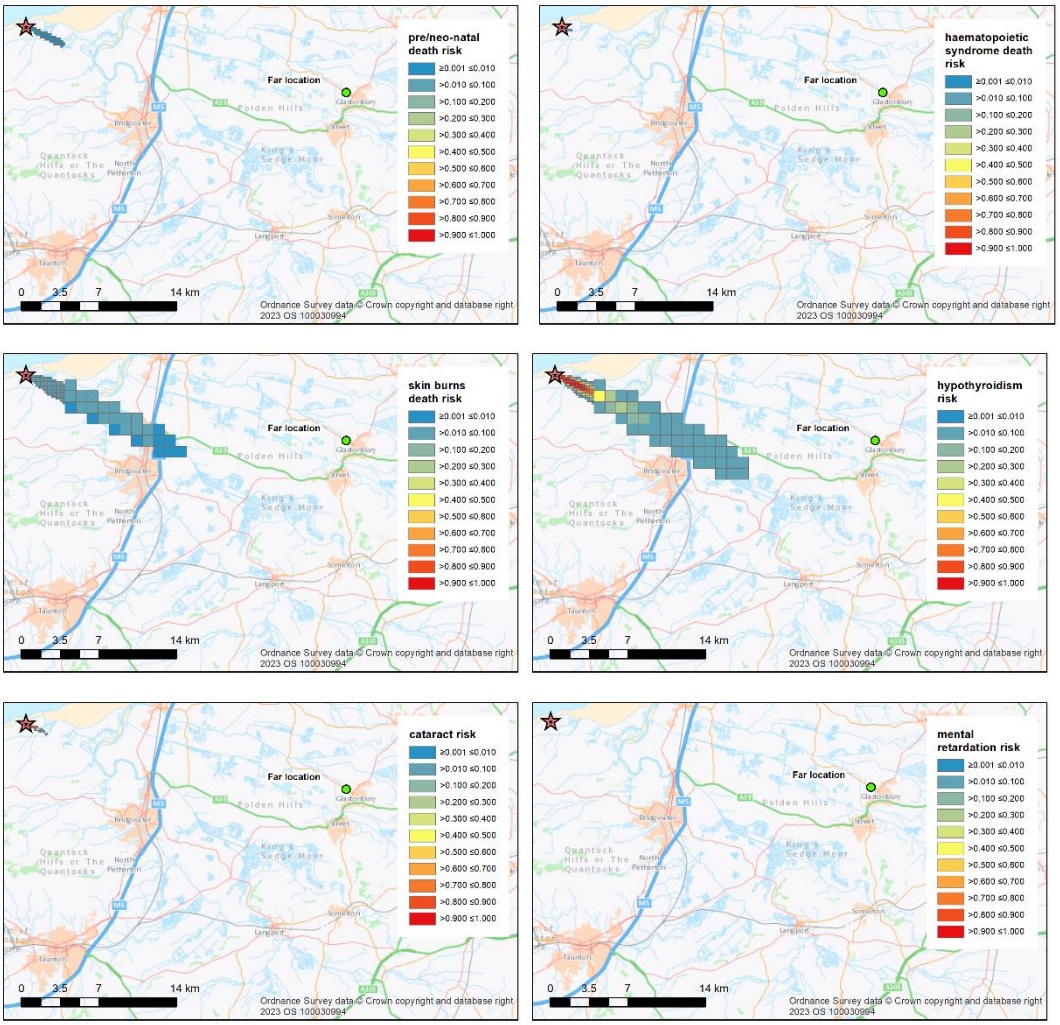


Figure 20 ST2-acute-dry – predicted extent of deterministic effects, risk of mental retardation predicted at less the 0.001 at all locations

In the ST2-acute-wet example, PACE predicts haematopoietic syndrome, gastrointestinal syndrome, pre- and neo-natal and skin burn deaths, and at the point of release but inside the boundary also predicts pulmonary syndrome. In addition, PACE predicts lung impairment hypothyroidism, cataract, mental retardation, and skin burn morbidity risks. Figure 21 shows the predicted extents, which encompass the near location but not the far.

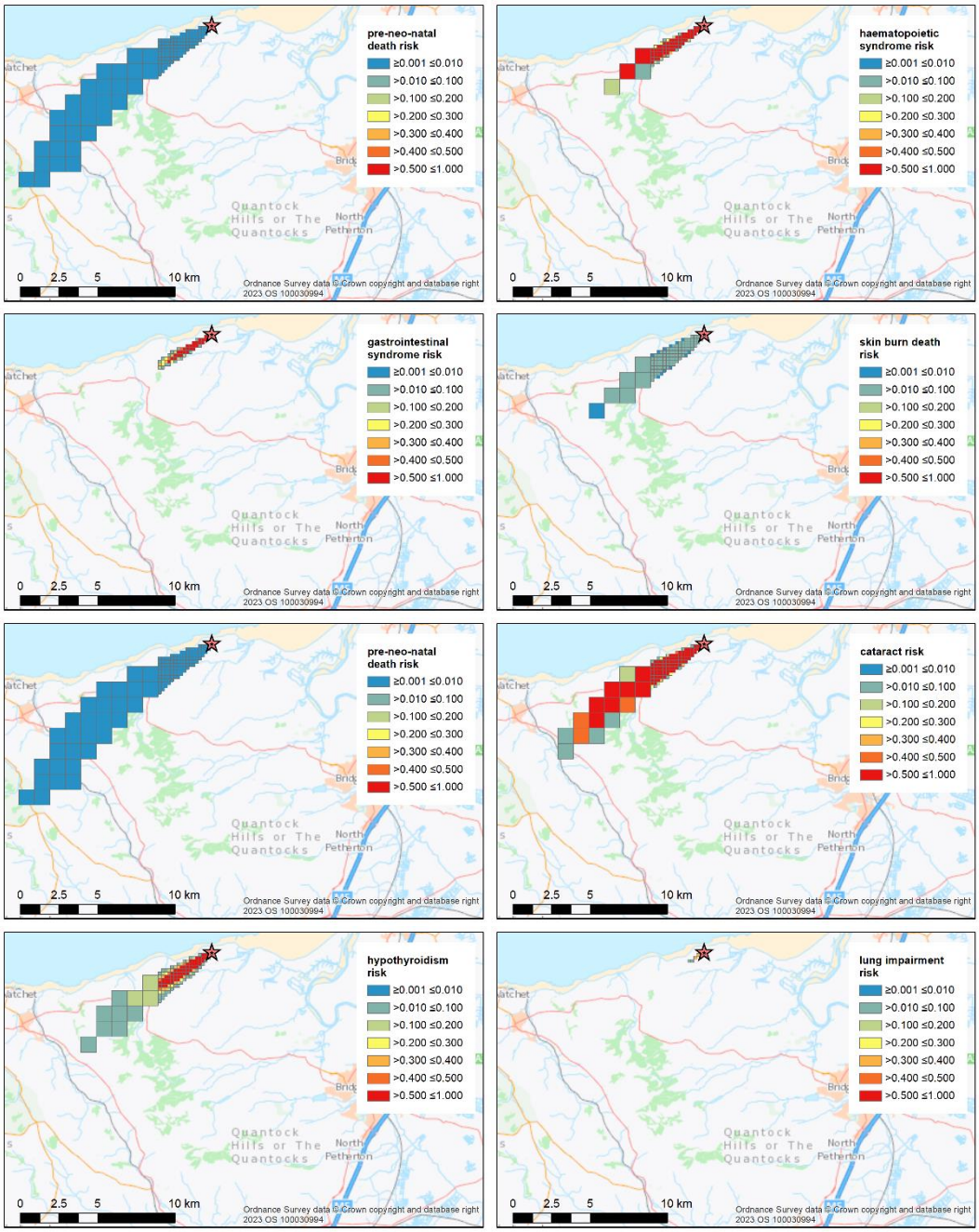


Figure 21 ST2-acute-wet – predicted extent of deterministic effects, risk of mental retardation predicted at less than 0.001 at all locations, pulmonary syndrome predicted only at the point of release

6.5 Stochastic risk profiles

6.5.1 Incidence

Figure 22 show all the predicted risks of incidence of cancer in all tissues.

Relative risks of incidence are not consistent between all locations and deposition conditions (i.e. ignoring the absolute values, the graphs appear somewhat different). The near locations, particularly under the wet conditions, have such high exposures that many of the risks are

truncated at 100%. Because of the high exposures, and because deterministic effects are expected (6.4), the near examples are not discussed further, but are included in the graphs.

The dry-far example is dominated by the thyroid cancer incidence risks, whereas in the wet-far example, the thyroid cancer incidence risks are the largest individual tissue risks for the 1-year-old, but the breast and the remainder ('Other solid') cancer incidence risks for the other age-groups are similar or larger. The all-solid-cancer ('Solid') risk is highest. To highlight the risks in other tissues, Figure 23 shows the risk of incidence in the dry-far example without the thyroid cancer risk.

For the dry-far example the following features can be seen:

- The predicted thyroid cancer incidence risks dominate.
- For most tissues including thyroid, there is a clear age-dependency with the risk to 1-year-old generally highest, followed by 10-year-old, 35-year-old and 60-year-old. However, for the lungs, oesophagus and bone marrow ('Leuk') tissues, the relationship is either not strong, or not consistent, or risks are so small that the relationship is not discernible.
- The risks for females are notably higher than for males for thyroid, lung, stomach and for 'all solid' tissues. For other tissues, the male risks are larger or the same, or the risks are so small that differences are not discernible.
- There are only small differences between the corresponding risks of incidence calculated using the ICRP and BEIR models. For example, for lungs the BEIR results show more age dependency than the ICRP.
- The predicted risk of thyroid cancer is not conservatively represented by the corresponding risk of all-solid-cancers ('Solid'). Excluding thyroid, the risks of all-solid-cancers are larger than the risks of individual tissues and looks to be a reasonable approximation of the combined risks.
- The age/sex-average risk estimated using ICRP Publication 103 nominal factors is similar to the 35-year-old for most tissues including thyroid, but does not always conservatively represent the risk to 1-year-old or 10-year-old.

For the wet-far example the following features can be seen:

- The predicted thyroid cancer incidence risks are highest for 1-year-old, but the breast and remainder ('Other solid') tissues risks appear higher for some of the other age groups.
- For most tissues, the risk to 1-year-old is highest, followed by 10-year-old, 35-year-old and 60-year-old. However, for the lungs, oesophagus and bone marrow ('Leuk') tissues, the relationship is either not strong, or not consistent, or cancer incidence risks are so small that the relationship is not discernible.
- The predicted risk to females is higher than for males for thyroid, lung, stomach and for all-solid ('Solid') tissues. For other tissues, the male risks are larger or the same, or the risks are so small that differences are not discernible.
- There are only small differences between the corresponding risks of incidence calculated using the ICRP and BEIR models. For example, for lungs, the BEIR results show more age dependency than ICRP.

- The predicted risk of all-solid-cancers (‘Solid’), is larger than the risk in any single tissue and looks to be a reasonable approximation of the combined risk.
- The age/sex-average cancer incidence risk estimated using ICRP Publication 103 nominal factors is similar to the 35-year-old for most sites including thyroid, but does not often conservatively represent the risk to 1-year-old or 10-year-old.

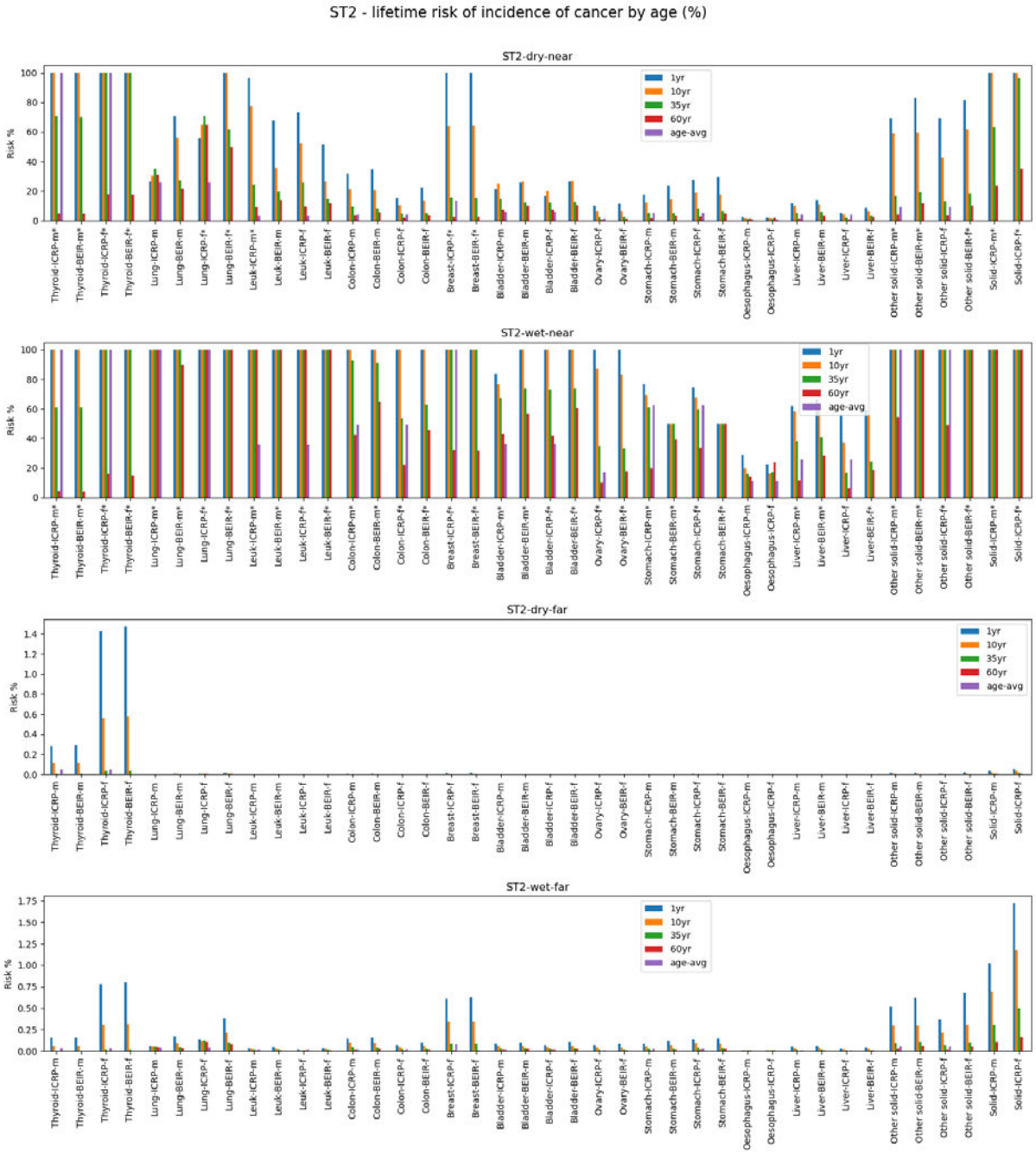


Figure 22 ST2-acute – all predicted risks of incidence of cancers using both ICRP and BEIR models

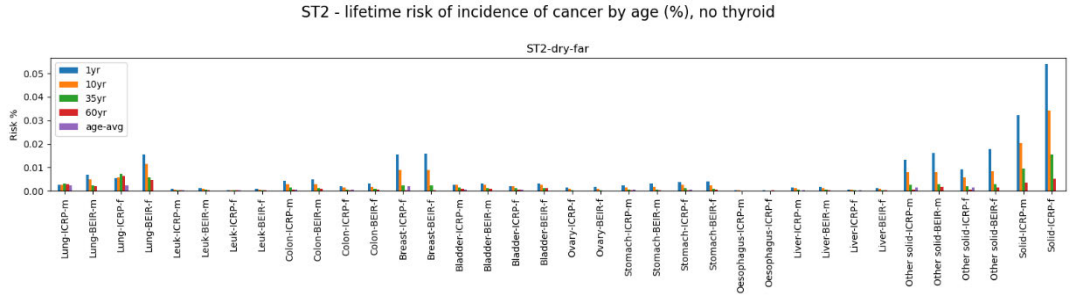


Figure 23 ST2-acute-dry-far – predicted risks of incidence of cancers using both ICRP and BEIR models, omitting thyroid risks

6.5.2 Fatalities

Figure 24 shows the predicted risks of fatality. As noted in Section 3.6, there is no mortality model for thyroid cancer in ICRP Publication 103 so only results based on multiplying the cancer incidence risk with an averaged lethality fraction ('inc' results) are presented for the thyroid. Relative risks of fatalities are not consistent between all examples (i.e. even ignoring the absolute values, the graphs appear somewhat different). For the near locations the risks are very high, particularly for the wet example, where many of the risks are truncated at 100%. Because of the high exposures, the near examples are not discussed further.

Between the two far locations there are inconsistencies. The dry-far example is dominated by the risk of fatality from thyroid. Whereas, in the wet-far example other tissue risks are larger, notably the remainder organs ('Other solid'), breast and lung. To highlight the risks in other tissues, Figure 25 shows the risks in the dry-far example without the thyroid risks.

For the dry-far example the following features can be seen:

- The predicted thyroid risks dominate.
- For most tissues including thyroid, there is a clear age dependency, with the risk to 1-year-old generally the highest, followed by 10-year-old, 35-year-old and 60-year-old. Whereas, for the lungs, bone marrow ('Leuk'), bladder and oesophagus, the relationship is either not strong, or not consistent, or risks are so small that the relationship is not discernible.
- The risk of fatality for females is notably higher than for males for thyroid, lung, stomach and all solid ('solid'). For other tissues, the male risks are larger or the same, or the risks are so small that differences are not discernible
- The predicted fatality risks of all-solid-cancers ('solid') are much smaller than the thyroid risk.

For the wet-far example the following features can be seen:

- The remainder ('other solid') tissue risks tend to be largest, followed by breast and lung.
- For most sites, the risk to 1-year-old is highest, followed by 10-year-old, 35-year-old and 60-year-old. However, for the lungs and oesophagus tissues, the relationship is either not strong, or not consistent, or risks are so small that the relationship is not discernible.

- The risks for females are higher than for males for thyroid, lung, and stomach tissues. For other tissues, the male risks are larger or the same, or the risks are so small that differences are not discernible.
- The predicted risks of fatality from all-solid-cancers ('Solid') are larger than any individual risk and look to be a reasonable approximation of the combined risk

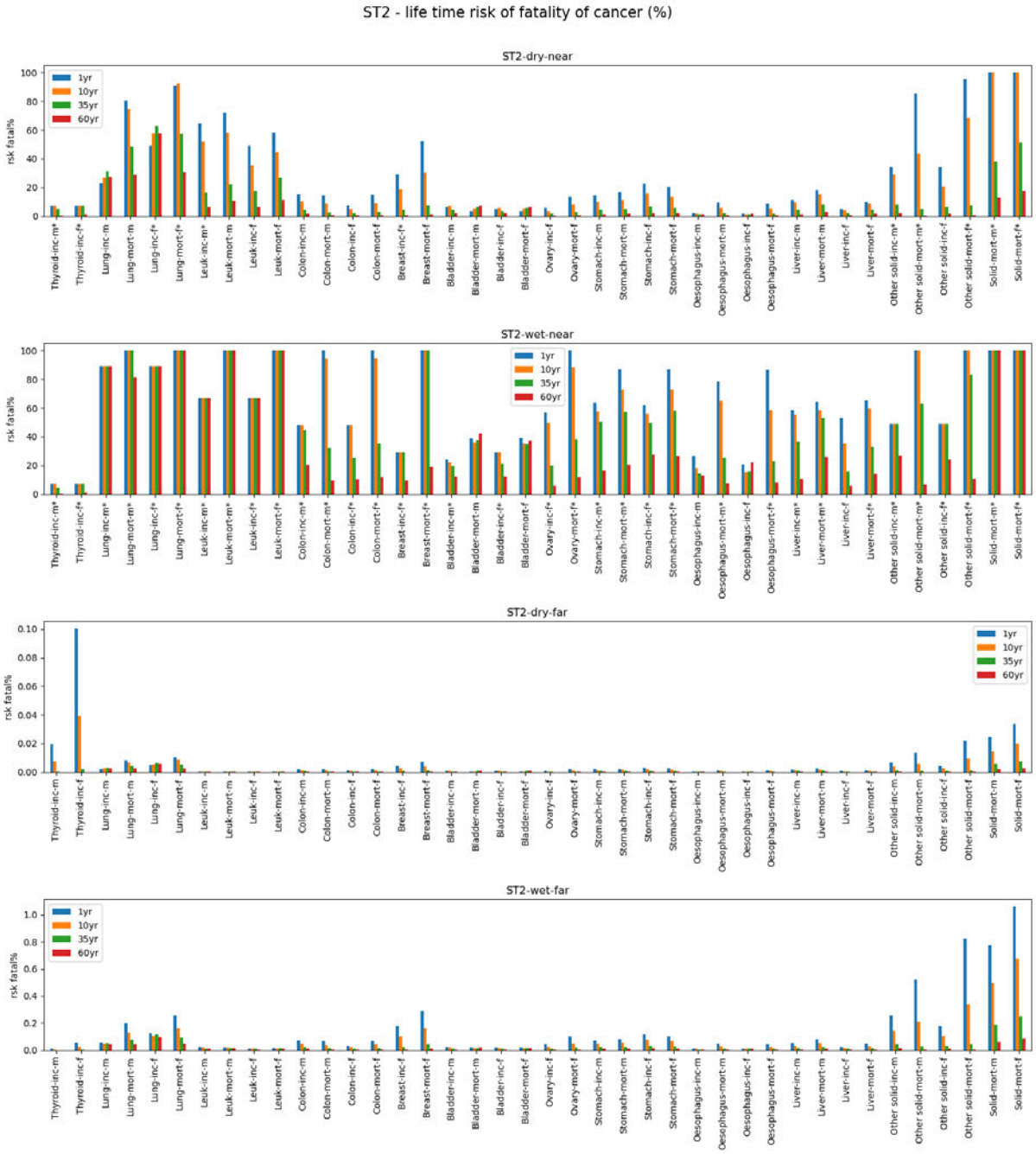


Figure 24 ST2-acute – predicted risks of fatality of cancers. Risks are calculated either by applying an age/sex independent lethality fraction to the corresponding risk of incidence ('inc') or by applying ICRP mortality risk models ('mort')

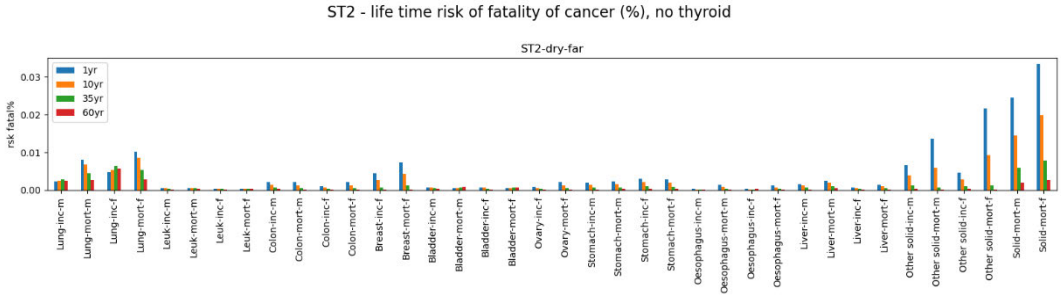


Figure 25 ST2-acute-dry-far – predicted risks of fatality of cancers. Risks are calculated either by applying an age/sex independent lethality fraction to the corresponding risk of incidence ('inc') or by applying ICRP mortality risk models ('mort')

6.5.3 Detriment

The predicted risks of detriment are shown in Figure 26. Relative risks of detriment are not consistent between examples (i.e. even ignoring the absolute values, the graphs appear different). For the near locations the risks are very high, particularly the wet example, where many of the risks are truncated at 100%. Because of the high exposures, the near examples are not discussed further.

Between the far locations with lower doses and risks there are inconsistencies. The dry-far example is dominated by the risk from thyroid. Whereas in the wet-far example, risks to other tissues are larger, notably the remainder organs ('Other solid'), breast and lung for older age groups. To highlight the risks in other tissues, Figure 27 shows the predicted risk of detriment in the dry-far example without the thyroid risks.

For the dry-far example the following features can be seen:

- The risk of detriment is dominated by thyroid cancer.
- For thyroid, bone marrow ('Leuk'), colon, breast, ovary, stomach, liver and remainder ('Other solid'), there is a clear age dependency, with 1-year-old having the most risk, followed by 10-year-old, 35-year-old, and 60-year-old. For the other tissues the relationship is either not strong, or not consistent, or risks are so small that the relationship is not discernible.
- For thyroid, lung and stomach, the female detriment risks are generally higher than the male. Whereas for all other tissues the male risks are generally larger or the same, or the risks are so small that differences are not discernible.

For the wet-far example the following features can be seen:

- The predicted risk of detriment is highest in the remainder ('Other solid') tissue, followed by breast and thyroid.
- For thyroid, bone marrow ('Leuk'), colon, breast, bladder, ovary, stomach, liver and remainder ('Other solid'), there is a clear age dependency, with 1-year-old having the most risk, followed by 10-year-old, 35-year-old, and 60-year-old. For the other tissues the relationship is either not strong, or not consistent, or risks are so small that the relationship is not discernible.

- For thyroid, lung and stomach, the female detriment risk is higher than the male. Whereas for all other tissues the male risks are generally larger or the same, or the risks are so small that differences are not discernible.

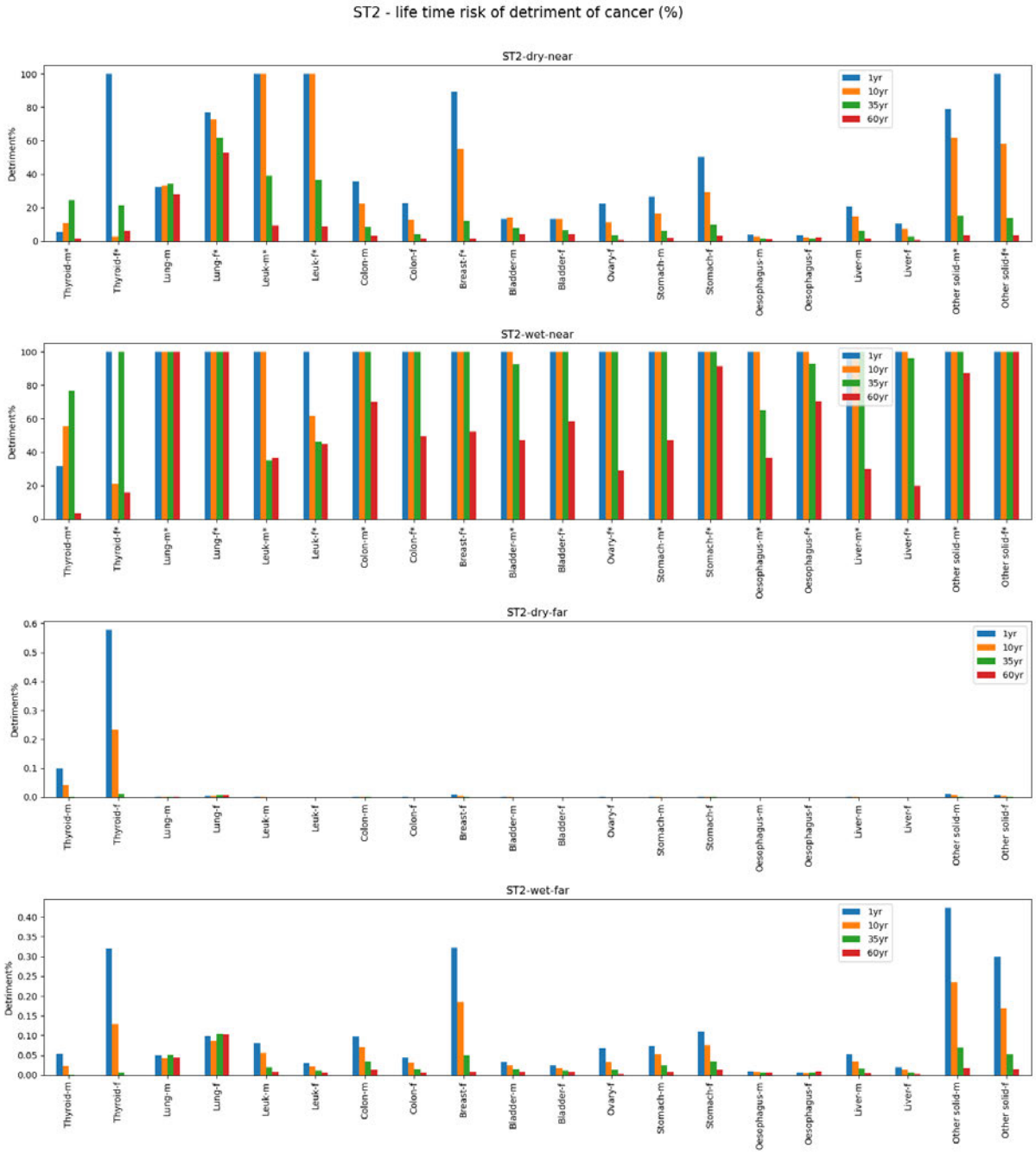


Figure 26 ST2-acute – predicted risks of detriment of cancers

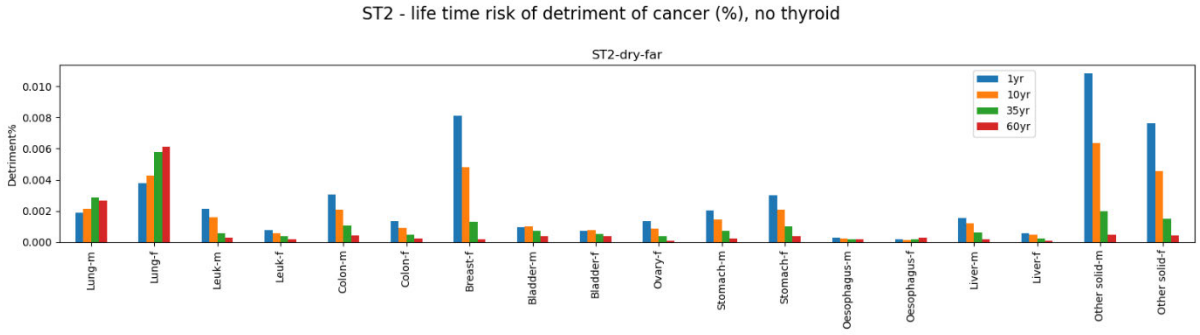


Figure 27 ST2-acute-dry-far – predicted risks of detriment of cancers for all tissues, omitting thyroid risks

6.6 Summary

In the ST2 case, the two near examples have dose predictions that are very high and well into the range where acute or deterministic effects may be expected (Figure A13 and Figure A14). With such doses, many of the values of risk of incidence, fatality and detriment are truncated at 100% or derived from truncated values, and therefore there must be a question-mark over the validity of these predictions even for the limited purpose of investigating age and sex dependency. Therefore, the discussions focus on the far examples which are in the range of doses where acute effects would not be predicted. In the dry-far example, the thyroid doses are dominant similar to the ST1 examples (Figure A2). In addition, there is an age-dependency apparent though less obvious than ST1 with the 1-year-old thyroid dose about 2 times the dose to the 35-year-old. Similarly, like ST1, doses of other organs do not show age-dependency (compare dry-far examples in Figure A4 and Figure A5).

In the wet-far example the thyroid is also the most significantly exposed organ, but the other organs have significant doses. Age-dependency is present in all organs, but it is not strong, the ovaries showing the strongest, with 1-year-old being 2 times the corresponding 35-year-old. The thyroid 1-year-old dose is less than twice the 35-year-old.

The age-dependency that was seen in thyroid dose in the far examples, is increased further when the thyroid incidence risks are calculated. The predicted risk of incidence of thyroid cancer in 1-year-olds is about 2-3 times the 10-year-old, 5-20 times the age-averaged estimate, and the 35-year-old and 60-year-old are not discernible on the graph. In the dry-far example, the age-dependency seen in the organ doses is also exaggerated in the cancer incidence risk predictions of other tissues (Figure 23). In the wet-far example, there was no age-dependency in the organ doses, but some of the incidence risk predictions do show such dependency, notably the breast, remainder (‘Other-solid’) and all-solid-cancers (‘Solid’). In other tissues it is not present, notably the lungs.

Sex-dependency can also be seen in the incidence risk predictions. In the far example, the female 1-year-old thyroid risk is about 4-5 times the corresponding male risk. Other tissues show some sex-dependency, in some cases reversed, but in no case does it appear as marked as for the thyroid.

The BEIR VII prediction of incidence are similar to the corresponding ICRP predictions being generally well within a factor of 2-3. The most obvious differences are for the lung, where there is an age-dependency in the BEIR VII prediction which is not apparent in the ICRP results, meaning while adult risks are similar, BEIR VII predicts higher risks to 1-year-old and

10-year-olds by a factor of 2-3. There is no basis to judge which predictions are ‘better’, but they are useful as being indicative of the uncertainty associated with risk modelling in general.

The predicted risks of fatality in the two far examples differ. In the dry-far example thyroid cancer fatalities dominate, despite not being a particularly lethal form of cancer. ICRP have no model of thyroid cancer fatality, so fatality risk is estimated by applying an age and sex independent lethality fraction of 7%. Consequently, the relative dependencies of thyroid cancer incidence risk prediction carry through unchanged to fatality risk. In the wet-far example, thyroid fatality is a small risk compared to other tissues, with remainder (‘Other solid’) being the most significant.

The predicted all-solid-cancer fatality risks in the dry-far example do not satisfactorily encompass the corresponding thyroid risks, particularly the female risks and lower ages. Whereas in the wet-far example, they are much more satisfactory, and by inspection of the graph, looks to be a reasonable estimate of the combined risks of all solid cancers. As discussed in Section 4, the all-solid-cancer risk is calculated from the colon doses. In the dry-far example the colon doses were much smaller than the corresponding thyroid doses whereas in the wet-far example they were about 25-30% of the corresponding thyroid doses. This, combined with the low lethality of thyroid, is why the all-solid-cancer risk prediction is much more consistent with the individual tissue cancer risk predictions in the wet-far example.

In the dry-far example, risks of thyroid detriment are dominant and show the same pattern of age and risk dependency seen in incidence and fatality. In the wet-far example, thyroid detriment risk is still relatively large, but some other tissues, notably breast and remainder (‘Other solid’) also have a significant risk.

Figure 28 gives the predicted age and sex-specific risks of incidence and fatality and detriment summed across all organs in the study compared to the age and sex-averaged detriment calculated by multiplying the age-specific effective dose with the ICRP nominal coefficient for cancer detriment of 5.5% Sv^{-1}. Ignoring the near locations where doses are very higher, females show higher risks and detriments compared to males of the same age. It is the younger ages and female groups that show the largest differences to the values calculated using nominal detriment.

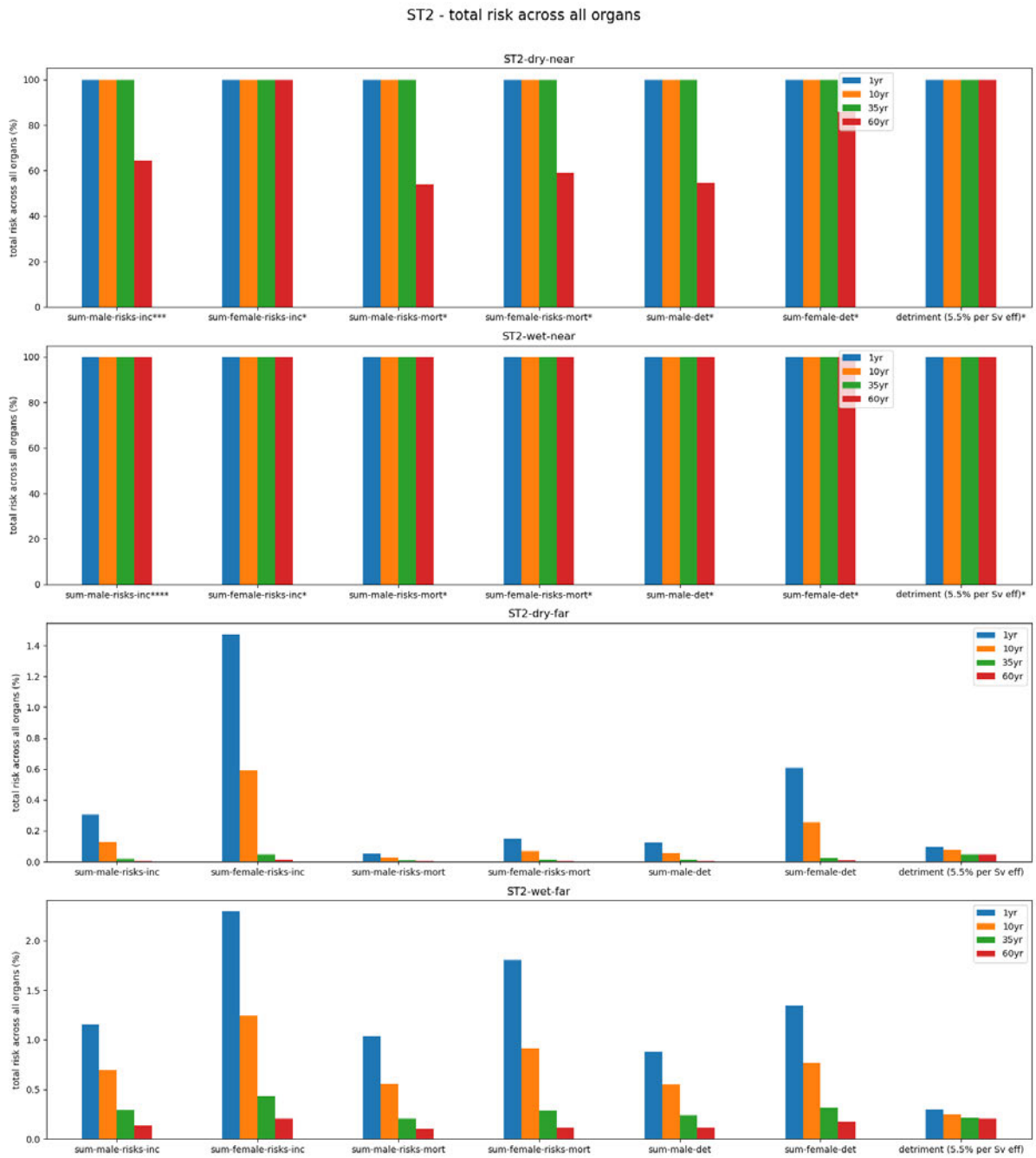


Figure 28 ST2-Acute – Age and sex-specific risks of cancer and detriment summed across tissues in the study compared with detriment calculated by applying ICRP nominal risk coefficient of 5.5% Sv^{-1} to the age-specific effective dose

7 Source term 3 – ST3-fuel

The third source term is based on an estimate of the Mayak accident source term but has adjusted amounts of plutonium and caesium to give a generic fuel-cycle source term. The release is assumed to be a single 1-hour phase with an effective height of 10m. All the plutonium is assumed to be ^{239}Pu. For this project it is termed ST3-fuel.

Table 4 The single phase and releases of the ST3-fuel source term (Bq)

Radionuclide	Phase 1 0 – 1 hours
Sr-90	4.00E+15
Zr-95	1.84E+16
Ru-106	2.74E+15
Ce-144	4.87E+16
Cs-137	2.59E+15
Pu-239	2.96E+13

7.1 Dry deposition

The weather sequence selected as dry begins at 18:00hrs 30/8/20. The pattern of total deposition of ^{239}Pu and the predicted extent and duration of protective actions are shown in Figure 29.

The near location was placed in the grid-square with maximum total deposition outside of the boundary fence and occurs towards the southeast. Based on the upper ERL criteria, evacuation would be advised, but no stable iodine prophylaxis would be required. Relocation is calculated to be required for two years (based on a criterion of 20mSv y^{-1} and assuming no clean-up is performed). Milk restrictions are predicted to be required for 50 years, green vegetable restrictions for 100 years, and the restrictions on all foods ranging between 10 and 100 years.

The far location was placed in the grid-square with the maximum total deposition at 30km outside of the sheltering and evacuation zones and outside the relocation zone. It is towards the southeast. No emergency protective actions are required at the upper ERL and no relocation (criterion of 20mSv y^{-1}). Milk restrictions are predicted to be required for 2 years (because of long-lived radionuclides, and assuming that no other agricultural protective actions are applied), green vegetable restrictions for 6 months, and the predicted restrictions on all foods ranging between 3 months and 3 years.

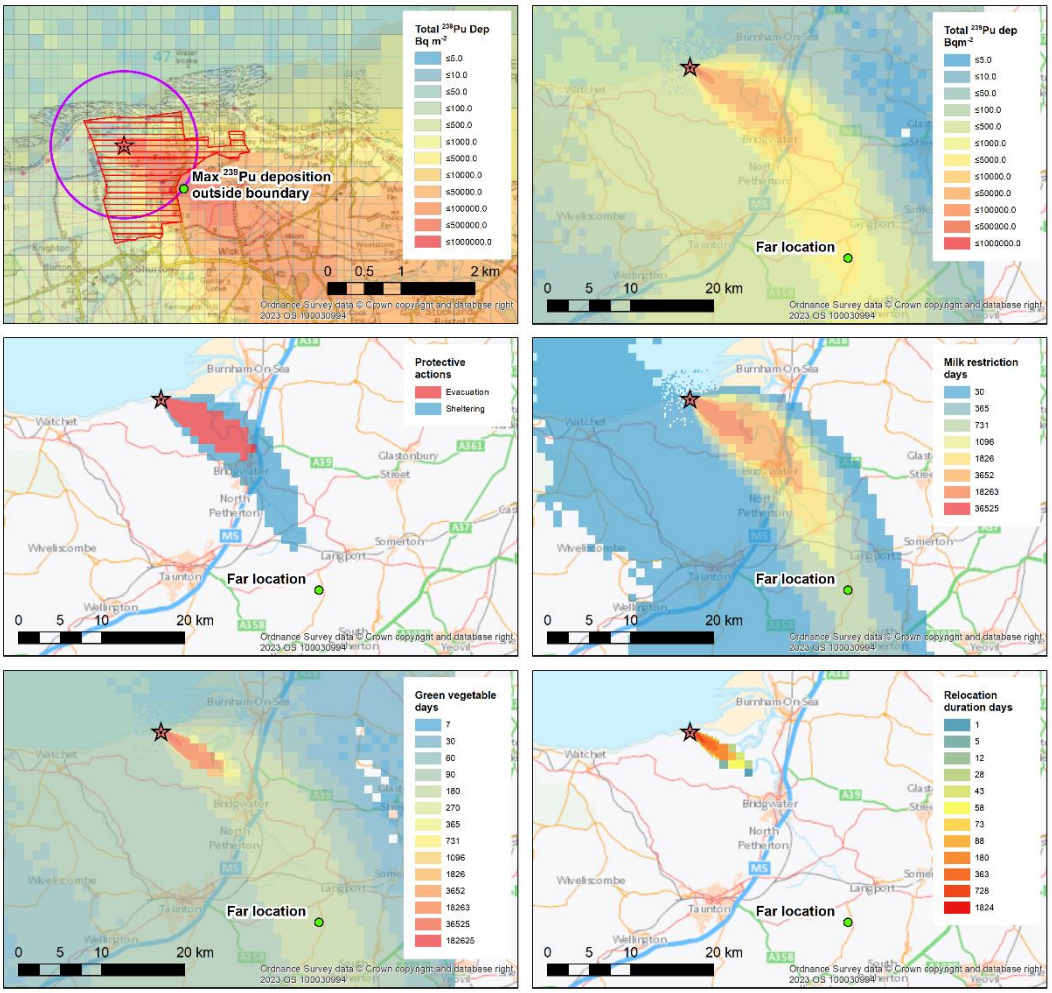


Figure 29 Predicted consequences of dispersal of ST3-fuel. The model run beginning 18:00 30/8/20 giving predominantly dry conditions.

7.2 Wet deposition

The weather sequence selected as predominantly wet begins 12:00hr 25/7/20 (met #349), Figure 30.

The near location was placed in the grid-square with the maximum deposition outside of the boundary, with approximately 91% of the deposition being due to rainfall. Based on the upper ERL, evacuation would be advised, and relocation would be required for 10 years (based on a criterion of 20mSv y^{-1} and assuming no clean-up is performed). Milk restrictions are predicted to be required for 100 years (because of long-lived radionuclides, and assuming that no other agricultural protective actions are applied), green vegetable restrictions for 500 years and predicted restrictions for all foods ranging between 50 years and 500 years.

The far location was placed in the grid-square with the maximum deposition at 30km and is towards the east, where more than 99% of the deposition is due to rainfall. No emergency protective actions are required, but relocation is predicted to be required for 6 months based on a dose criterion of 20mSv y^{-1} and assuming no clean-up performed. Milk and green vegetable restrictions are predicted to be required for 50 years, and predicted restrictions of other foods ranging between 6 months and 50 years.

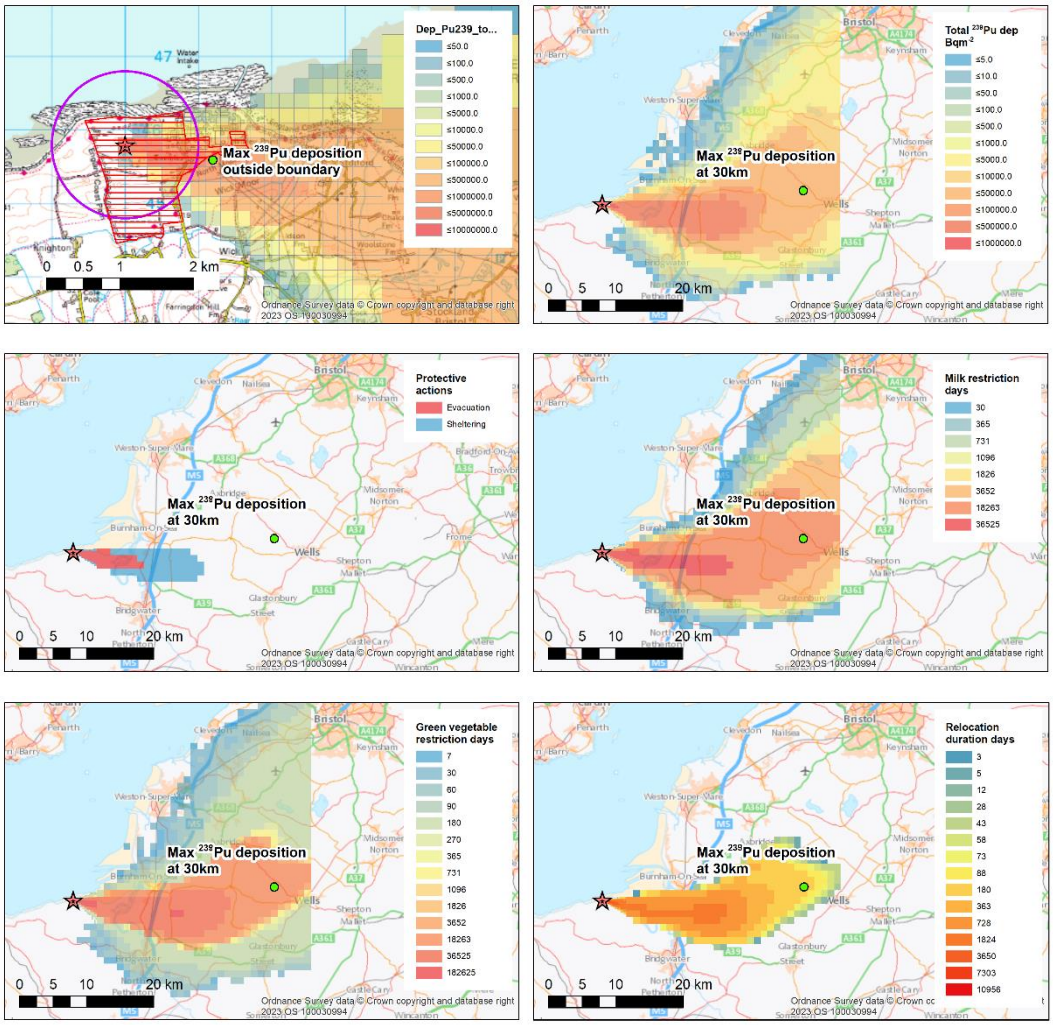


Figure 30 Predicted consequences of dispersal of ST3-fuel. The model run beginning 12:00 25/7/20, giving predominantly wet conditions.

7.3 Dose profiles

The predicted effective doses in the near examples range from 5.4 to 7.6 Sv, and in the far examples from 34 to 140 mSv.

The largest organ doses in the near examples and the far-dry example are predicted to the liver, lungs, and bone marrow. In the far-wet example, the bone marrow is predicted to be highest, with corresponding doses to other organs approximately 50% as high.

Age-dependency is visible but not strong with the most severe being the wet-far Bone Marrow and Ovary doses where the 1-year-old is just more than twice the 35-year-old. Also notable are the liver doses in the near examples and dry-far example, where the 35-year-old is approximately twice the 1-year-old.

A more detailed description of the dose profiles can be found in Appendix A3.

7.4 Deterministic profiles

In the ST3-fuel-dry example, PACE predicts very little in the way of deterministic effects limited to mental retardation at very low levels of risk (<0.0002) out to about 3km and skin burns at low levels of risk (<0.02) that do not extend beyond the site boundary.

In the ST3-fuel-wet example, PACE also predicts low deterministic effects limited to mental retardation at very low levels of risk (<0.0005) to 22km, and low level risks of skin burns (<0.005) and low level risks of neo/pre-natal deaths (<0.0005), both do not extend off-site.

7.5 Stochastic Risk profiles

7.5.1 Incidents

Figure 31 shows all the predicted risks of incidence of cancer in all tissues.

Relative risks of incidence are not completely consistent between examples (i.e. the graphs appear somewhat different). The near locations have high exposures and a few of the risks for lungs and all solid ('Solid') tissues have been truncated.

In the dry-near example, the largest risks are for lung, bone marrow ('Leuk'), and all-solid ('Solid') tissues.

The wet-near example is similar to the dry-near example, with the largest risks also being for lung and all-solid ('solid'), with the bone marrow ('Leuk') slightly reduced relatively and the breast and remainder ('Other solid') relatively increased.

The pattern of risks seen in the different organs is also similar in the dry-far example, with the largest risks being for lung and all-solid ('solid'), but with no other tissues standing out.

The wet-far example is the most different of all the 4 examples with the all-solid ('solid') risk still being the largest, but the lung is very reduced compared to the dry-far example.

The following features can be seen:

- Generally, there is good consistency between ICRP and BEIR results. However, there are differences for the lung where there is a distinct age-dependency in the BEIR VII results which predict higher risks to a 1-year-old with the ICRP model showing a slighter higher risk to a 35-year-old adult. The calculated risks of lung cancer incidence adult risks between the two models are similar, but BEIR VII predicts higher risks to 1-year-olds and 10-year-olds by a factor of about 2. There is no basis to judge which predictions are 'better', but it gives a useful indication of the uncertainty on risk modelling in general.
- For thyroid, bone marrow ('Leuk'), colon, breast, ovary, stomach, remainder ('Other solid') and all-solid ('Solid') tissues, there is a clear age dependency, with 1-year-old having the most risk, followed by 10-year-old, 35-year-old, and 60-year-old. For lung, bladder, liver and oesophagus there is no strong or consistent age dependency.
- For lungs and thyroid tissues, the female risks are larger, whereas for all other organs the male risks are larger or the same.

- The age/sex-averaged risk estimated using ICRP Publication 103 nominal factors track the adult risk, but are generally not conservative for most tissues or ages.

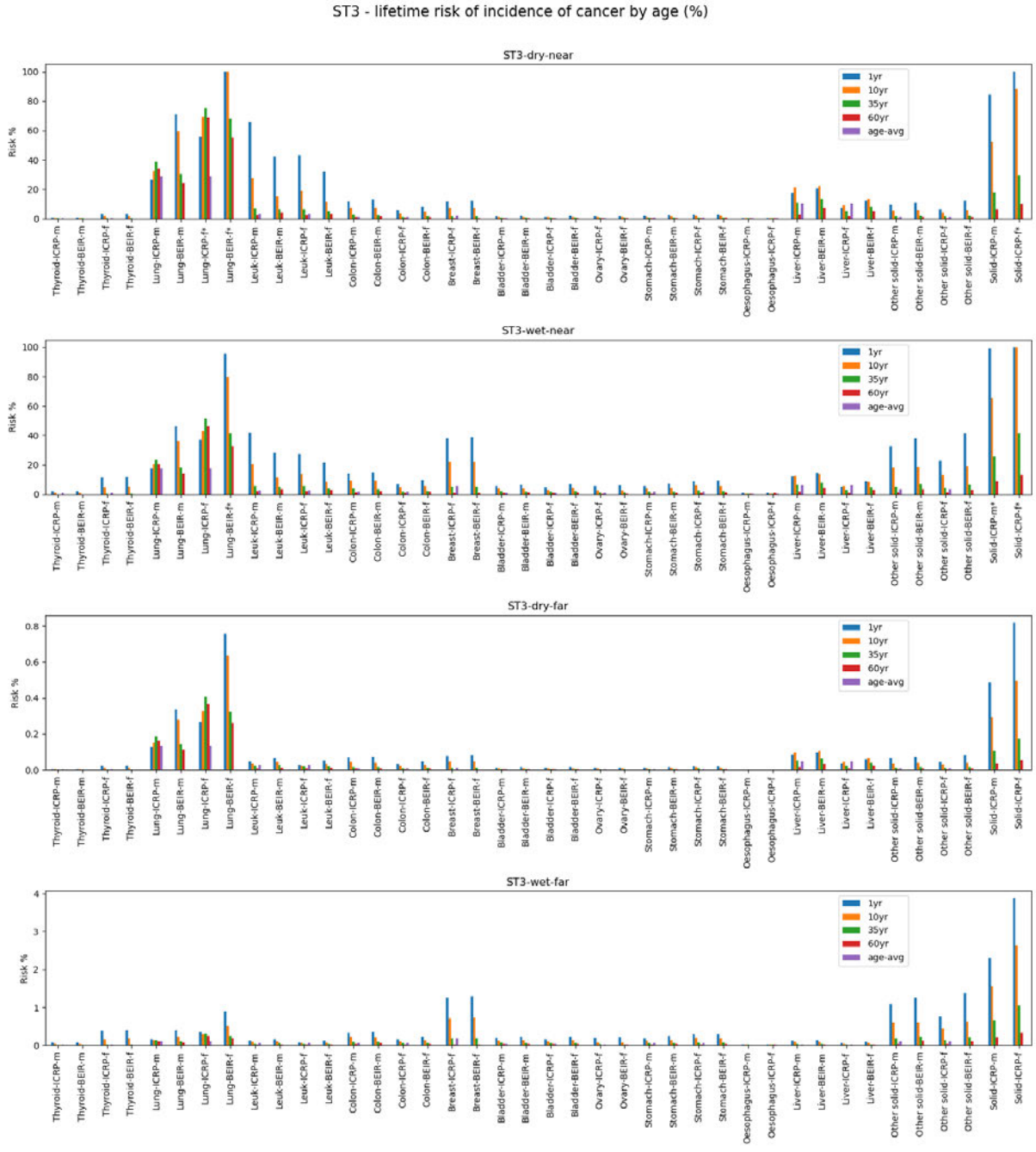


Figure 31 ST3-fuel – all predicted risks of incidence of cancers using both ICRP and BEIR models

7.5.2 Fatalities

Figure 32 shows all the predicted risks of fatalities of cancer in all tissues.

Relative risks are not completely consistent between examples (i.e. the graphs appear somewhat different). The near locations have high exposures and a few of the risks for lungs and all solid ('Solid') tissues have been truncated.

In the dry-near example, the largest risks are to lung, bone marrow (‘Leuk’), liver, and all-solid (‘Solid’) tissues.

The wet-near example is similar to the dry-near example, with the largest risks also being to lung, all-solid-cancer (‘Solid’) and bone marrow (‘Leuk’) with liver slightly reduced, and remainder (‘Other solid’) increased compared with the dry-near example.

The dry-far example is also similar, with the largest risks also being lung and all-solid-cancer (‘Solid’), with liver also standing out.

The wet-far example is the most different of all the 4 examples with the all-solid-cancer (‘Solid’) risk is still the largest and the breast and remainder (‘Other solid’) are the next largest. However, the risk of fatalities due to lung and liver cancers are reduced compared to the dry-far example.

The following features can be seen:

- For lungs, the female risks are larger, whereas for all other organs the male risks are larger or the same, or risks are so small that differences are not discernible.
- For thyroid, bone marrow (‘Leuk’), colon, breast, ovary, stomach, remainder (‘Other solid’) and all-solid (‘Solid’) tissues, there is a clear age dependency, with 1-year-old having the most risk, followed by 10-year-old, 35-year-old, and 60-year-old. For lung, bladder, liver and oesophagus there is no strong or consistent age dependency or risks are so small that differences are not discernible.

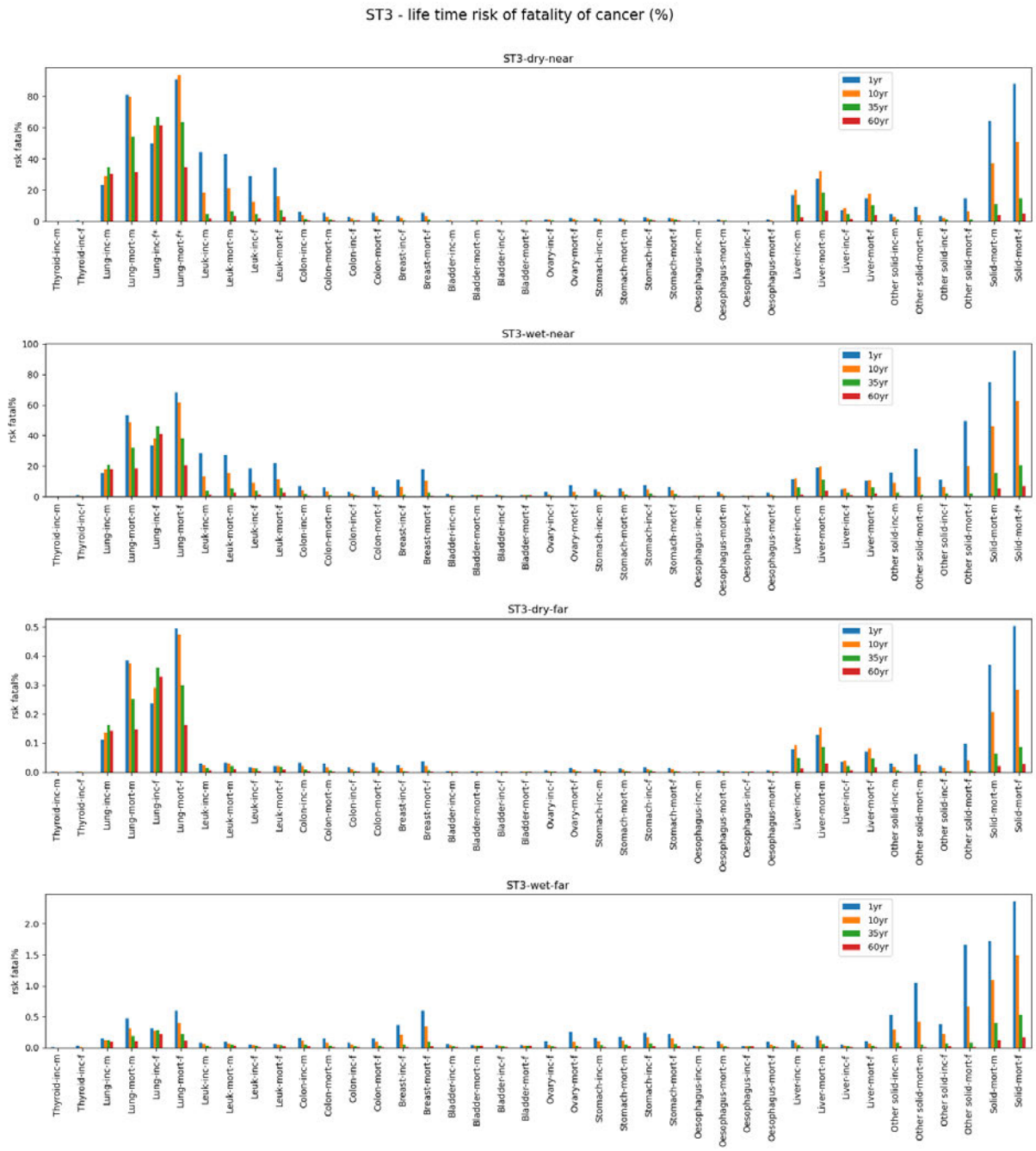


Figure 32 ST3-fuel – all predicted risks of fatality of cancers. Risks are calculated either by applying an age/sex independent lethality fraction to the corresponding risk of incidence ('inc'), or by applying ICRP mortality risk models ('mort').

7.5.3 Detriment

Figure 33 show all the predicted risks of detriments in all tissues.

Relative risks are not completely consistent between examples (i.e. the graphs appear somewhat different). The near locations have high exposures and the risks for some of the organs such as lungs and bone marrow ('Leuk') have been truncated.

In the dry-near and wet-near examples, the largest risks are for bone marrow ('Leuk'), followed by lung.

In the dry-far example, the largest risks are to the lung, followed by bone marrow ('Leuk') and liver.

The wet-far example the largest risk is to remainder tissues ('Other solid') followed by breast.

The following features can be seen:

- For thyroid, lungs and stomach, the female risks are larger, whereas for all other organs the male risks are larger or the same, or risks are so small that differences are not discernible.
- For thyroid, bone marrow ('Leuk'), colon, breast, ovary, stomach, liver and remainder ('Other solid') tissues, there is a clear age dependency, with 1-year-old having the most risk, followed by 10-year-old, 35-year-old, and 60-year-old. For lung, bladder and oesophagus there is no strong or consistent age dependency or risks are so small that differences are not discernible.

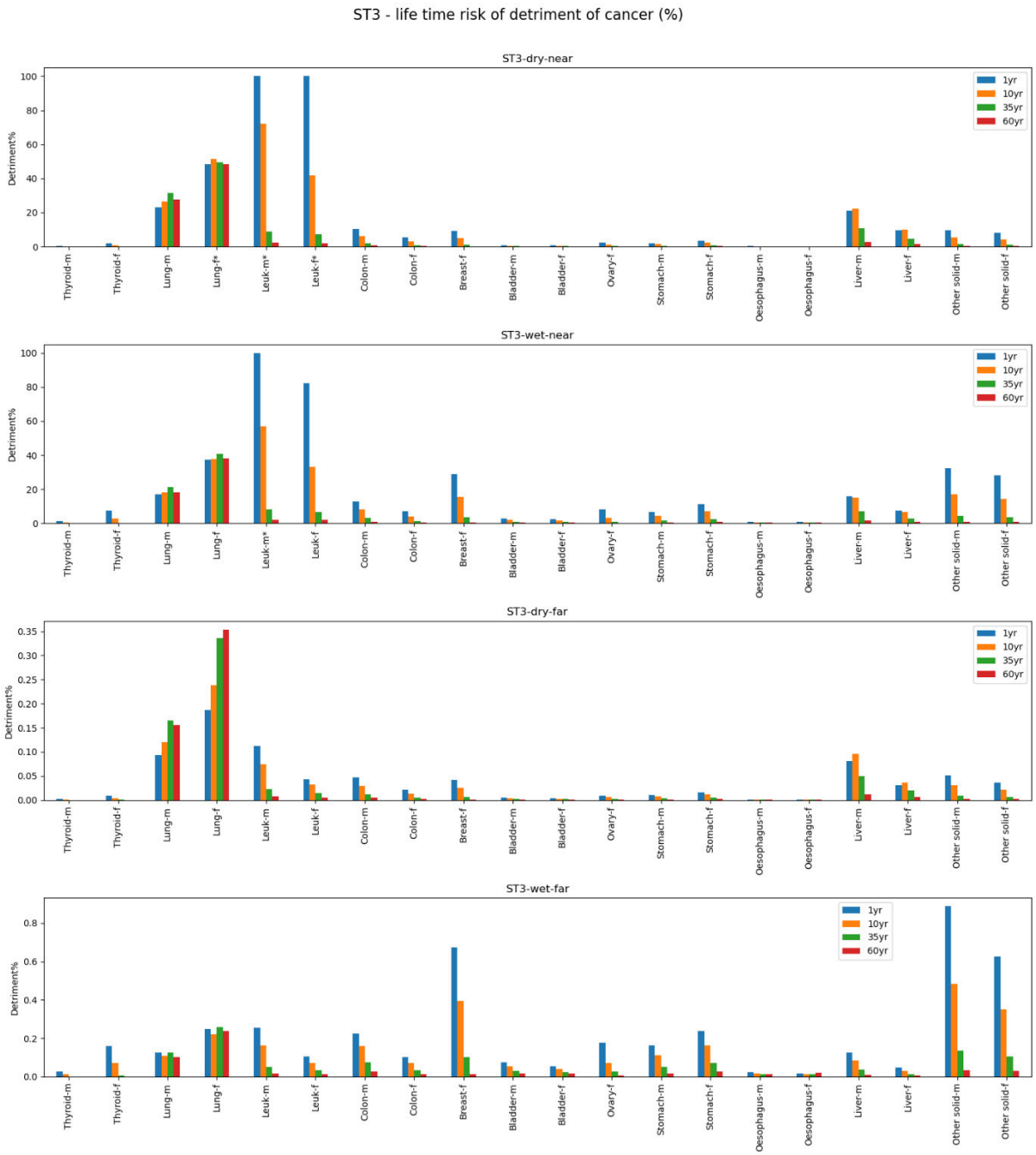


Figure 33 ST3-fuel – all predicted risks of detriment of cancers

7.6 Summary

The ST3-fuel example can be divided into two groups. The first group includes the two near examples and the dry-far example. The second group is the wet-far example. In all the examples except the wet-far example the dose is unevenly received by the organs, with liver and lungs receiving the most, followed by bone marrow. In the second group, the wet-far example, the dose is more evenly distributed between the organs, with bone marrow about a factor of two larger than the other organ doses which are all very similar.

Age dependency is small with not much more than a factor of two between the highest and the lowest in any organ or example. In all the examples except the wet-far example, it is the 35-year-old who received the highest predicted dose for lung and liver (the two highest exposed organs), though the age-dependency seen in the lung is slight.

The two near examples have organ dose predictions that are very high and extend into the range where acute or deterministic effects may be expected. Consequently, the predicted risks are very high and some of the predicted lung and all solid cancers risk of cancer incidence are truncated at 100% or derived from truncated values. Therefore, there must be a question-mark over the validity of all the near predictions even for the limited purpose of investigating age and sex dependency. The remainder of the discussion concerns the far examples.

In the dry-far example the predicted risk of incidence in the lung is the highest, with BEIR VII and ICRP models both showing some age dependency in the risks. However, they are not consistent, with the ICRP model predicting the highest risk to the 35-year-old and BEIR VII to the 1-year-old. For most other tissues ICRP and BEIR VII are closer.

In the wet-far example, breast and remainder ('Other solid') tissues have the highest incidence risks. The age dependency seen in the corresponding organ doses is exaggerated in the predictions of risk with a factor of about 5-6 between the 1-year-old and the 35-year-old.

For both examples age/sex-average risk estimates made using ICRP103 nominal risk factors are a reasonable approximation of the corresponding 35-year-old risk. For the lung dose it is about 70% of the 35-year-old male ICRP model prediction and within about a factor of three of the 35-year-old female.

In the wet-far example, the all-solid-cancer incidence risk is larger than the risk for any individual tissue cancer incidence. This is also mostly true for the dry-far example except for the lung risk which in the 35-year-old and 60-year-old exceeds it by a factor of 2-4. All-solid-cancer risk shows a distinct age dependency with the 1-year-old being about a factor 3-5 of the 35-year-old risk. It also shows a sex-dependency with the female risks being about a factor of 1.5 higher than the corresponding male risks.

The dry-far fatality risk predictions exhibit similar patterns to the corresponding incidence risk predictions. Lung cancer is the highest risk in the far-dry example with the risk about a factor of 2 higher for females. The prediction made by applying a lethality fraction to the incidence risk ('inc') gives the highest risk to the 35-year-old, whereas the modelled risk ('mort') predicts the highest risk to the 1-year-old followed closely by the 10-year-old. In dry-far example, the all-solid-cancer fatality prediction mostly encompasses the risks from individual dose except for some of the predicted lung cancer fatality risks,

The wet-far fatality risk predictions also exhibit similar pattern to the incidence risks

Overall, for all tissues, the predictions of fatality risk made by applying a lethality fraction to the predictions of incidence risk ('inc'), are reasonably close to the directly modelled predictions of fatality ('mort'), and usually with a factor of 2-3.

Figure 34 gives the predicted age and sex-specific risks of incidence and fatality and detriment summed across all organs in the study, compared to the age and sex-averaged detriment calculated by multiplying the age-specific effective dose with the ICRP nominal coefficient for cancer detriment of 5.5% Sv-1. Ignoring the near locations where doses are very high, younger females show the higher risks and detriments compared to males. For adults the

differences between sexes are much less. Younger ages show a bigger difference to the nominal detriments than the adults which general show a good consistency.

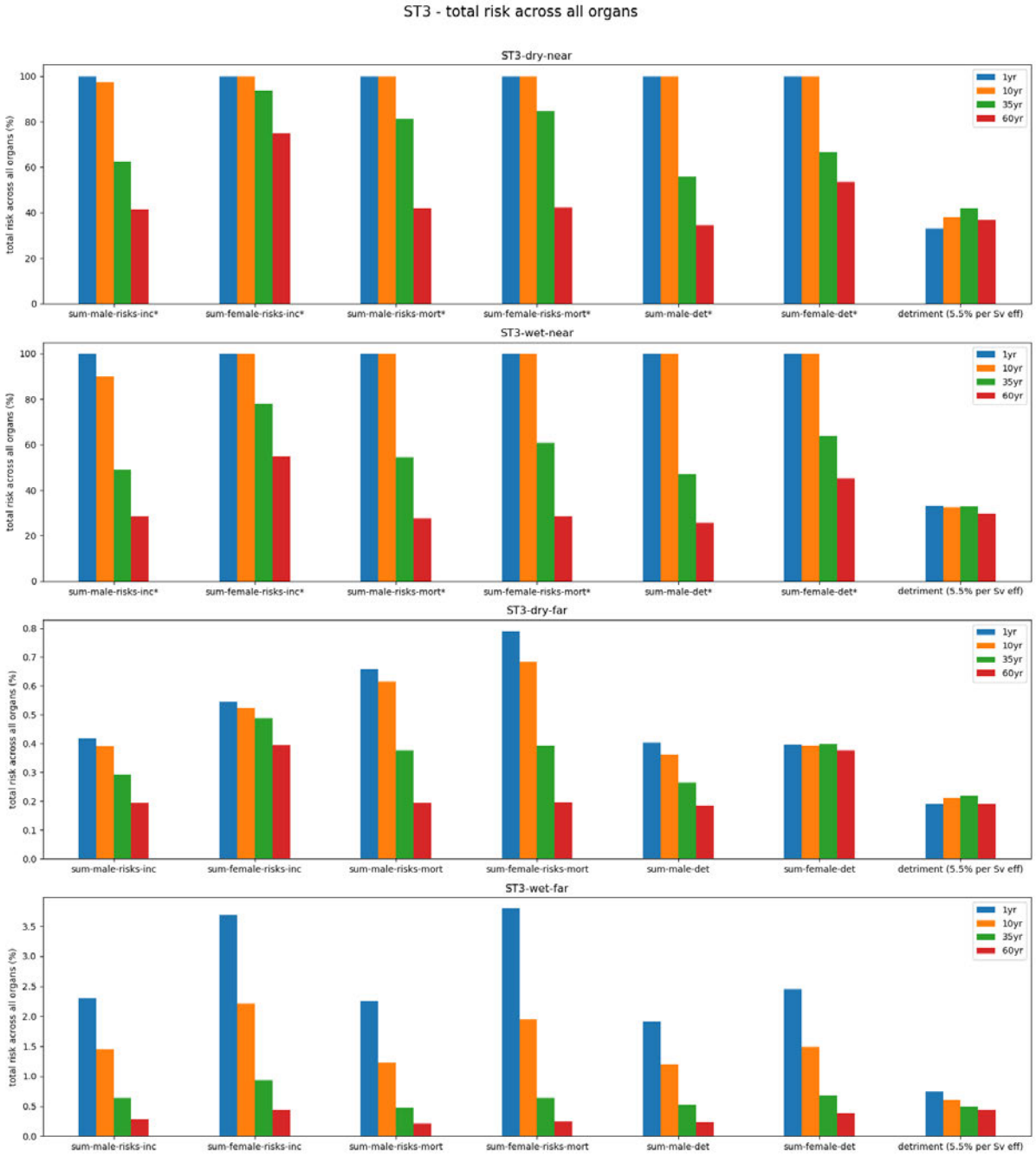


Figure 34 ST3-fuel – Age and sex-specific risks of cancer and detriment summed across tissues in the study compared with detriment calculated by applying ICRP nominal risk coefficient of 5.5% Sv^{-1} to the age-specific effective dose[1]

[1] An inconsistency is apparent in the ST3-dry-far example, with the predicted risk of mortality being apparently higher than risk of incidence particular in the younger groups. This is attributed to uncertainty of the model and model parameters.

8 Source term 4 – ST4-Chornobyl

The final source term is based on the Chornobyl accident and is termed ST4-Chornobyl. There is a lot of uncertainty about the accident and activities released. The ST4-Chornobyl source term was developed from an IAEA report (IAEA, 2006), which gives estimates of the total activities released summed over the 11-day duration (in some cases just a maximum possible activity), as well as a graph indicating the daily total radioactivity released but with very large error bars and with little indication as to the pattern of release from individual radionuclides which will depend on both half-life and volatility. The temporal information was augmented with a table from a separate report (NEA, 2002) which refers to the IAEA report but gives the estimated daily release of ^{131}I up to the 10th day, and a graph from a third report (UNSCEAR, 2000) giving a consistent daily release of ^{131}I, and additionally ^{132}Te, ^{137}Cs and ^{133}I. For the remaining radionuclides, it was assumed that all the noble gases were released on the first day, as were radionuclides with half-lives shorter than 15 days (^{140}Ba and ^{239}Np) and radionuclides noted as being volatile (^{129m}Te). The remaining radionuclides were scaled to the general daily release estimated by IAEA (2006).

There is no information about iodine fractions, so the recommended default fractions from FRMAC (2015) (30% elemental, 45% organic and 25% as components of aerosol) were used.

Effective release height is also very uncertain and likely variable depending on both conditions on site and the atmosphere. There are indications that some material reached very high levels, potentially as high as 1500m at certain points during the accident progression, but this is unlikely to form the bulk of the release. For this investigation an effective release height of 150m was used for all phases.

Table 5 Phases and releases of ST4-Chornobyl (Bq)

Radio-nuclide	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10	Day 11	Total
Kr-85	3.3E+16	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	3.3E+16
Sr-89	2.7E+16	9.0E+15	8.4E+15	6.0E+15	4.8E+15	4.8E+15	9.6E+15	1.1E+16	1.6E+16	1.8E+16	3.6E+13	1.2E+17
Sr-90	2.4E+17	7.9E+16	7.3E+16	5.2E+16	4.2E+16	4.2E+16	8.4E+16	9.9E+16	1.4E+17	1.6E+17	3.1E+14	1.0E+18
Zr-95	2.0E+16	6.6E+15	6.2E+15	4.4E+15	3.5E+15	3.5E+15	7.0E+15	8.4E+15	1.1E+16	1.3E+16	2.6E+13	8.4E+16
Mo-99	1.7E+16	5.7E+15	5.3E+15	3.8E+15	3.0E+15	3.0E+15	6.0E+15	7.2E+15	9.8E+15	1.1E+16	2.3E+13	7.2E+16
Ru-103	4.0E+16	1.3E+16	1.2E+16	8.8E+15	7.0E+15	7.0E+15	1.4E+16	1.7E+16	2.3E+16	2.6E+16	5.3E+13	1.7E+17
Ru-106	1.7E+16	5.7E+15	5.3E+15	3.8E+15	3.1E+15	3.1E+15	6.1E+15	7.3E+15	9.9E+15	1.1E+16	2.3E+13	7.3E+16
Te-129m	2.4E+17	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	2.4E+17
I-131	7.0E+17	2.0E+17	1.5E+17	1.0E+17	6.9E+16	6.2E+16	1.0E+17	1.1E+17	1.3E+17	1.3E+17	0.0E+00	1.8E+18
I-133	6.9E+17	1.4E+17	4.8E+16	1.7E+16	6.2E+15	4.1E+15	2.8E+15	6.9E+14	5.5E+14	3.5E+14	0.0E+00	9.1E+17
Te-132	3.3E+17	7.4E+16	6.6E+16	5.8E+16	4.9E+16	4.9E+16	7.4E+16	8.2E+16	1.6E+17	2.1E+17	0.0E+00	1.2E+18
Xe-133	6.5E+18	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	6.5E+18
Cs-134	1.3E+16	3.0E+15	2.7E+15	2.4E+15	2.0E+15	2.0E+15	3.0E+15	3.4E+15	6.7E+15	8.4E+15	0.0E+00	4.7E+16
Cs-136	1.0E+16	2.3E+15	2.1E+15	1.8E+15	1.5E+15	1.5E+15	2.3E+15	2.6E+15	5.1E+15	6.4E+15	0.0E+00	3.6E+16
Cs-137	2.4E+16	5.5E+15	4.9E+15	4.3E+15	3.6E+15	3.6E+15	5.5E+15	6.1E+15	1.2E+16	1.5E+16	0.0E+00	8.5E+16
Ba-140	2.4E+17	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	2.4E+17
Ce-141	2.0E+16	6.6E+15	6.2E+15	4.4E+15	3.5E+15	3.5E+15	7.0E+15	8.4E+15	1.1E+16	1.3E+16	2.6E+13	8.4E+16
Ce-144	1.2E+16	3.9E+15	3.7E+15	2.6E+15	2.1E+15	2.1E+15	4.2E+15	5.0E+15	6.8E+15	7.9E+15	1.6E+13	5.0E+16
Np-239	4.0E+17	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	4.0E+17
Pu-238	3.5E+12	1.2E+12	1.1E+12	7.9E+11	6.3E+11	6.3E+11	1.3E+12	1.5E+12	2.0E+12	2.4E+12	4.7E+09	1.5E+13
Pu-239	3.1E+12	1.0E+12	9.5E+11	6.8E+11	5.4E+11	5.4E+11	1.1E+12	1.3E+12	1.8E+12	2.0E+12	4.1E+09	1.3E+13
Pu-240	4.2E+12	1.4E+12	1.3E+12	9.4E+11	7.5E+11	7.5E+11	1.5E+12	1.8E+12	2.4E+12	2.8E+12	5.7E+09	1.8E+13
Pu-241	6.1E+14	2.0E+14	1.9E+14	1.4E+14	1.1E+14	1.1E+14	2.2E+14	2.6E+14	3.5E+14	4.1E+14	8.2E+11	2.6E+15
Pu-242	9.4E+09	3.1E+09	2.9E+09	2.1E+09	1.7E+09	1.7E+09	3.3E+09	4.0E+09	5.4E+09	6.3E+09	1.3E+07	4.0E+10
Cm-242	9.4E+13	3.1E+13	2.9E+13	2.1E+13	1.7E+13	1.7E+13	3.3E+13	4.0E+13	5.4E+13	6.3E+13	1.3E+11	4.0E+14

8.1 Dry deposition

The weather sequence selected as dry begins at 23:00hrs 09/05/20. The pattern of total deposition of ^{137}Cs is shown in Figure 35.

The near location was placed in the grid-square with the highest total deposition outside of the boundary fence and occurs towards the southwest, the proportion of wet deposition is 0%. Based on the upper ERL criteria. evacuation and stable iodine prophylaxis would be advised with relocation being required for 2 years (based on a criterion of 20mSv y^{-1} and assuming no clean-up is performed). Milk restrictions are predicted to be required for 100 years (because of long-lived radionuclides, and assuming that no other agricultural protective actions are applied), green vegetable restrictions for 500 years with predicted restrictions for all foods ranging between 50 years and 500 years.

The far location was placed off the plume centre line in the grid-square with the maximum total deposition at 30km that is outside the zones where sheltering and evacuation would be required (based on upper ERL criteria) and outside the zone where relocation would be

required without clean-up (based on a criterion of 20mSv y^{-1}). It is towards the east. The proportion of wet deposition at this location being 0%. Based on the upper ERL criteria, stable iodine prophylaxis would be advised. Milk and green vegetable restrictions are predicted to be required for 50 years and predicted restrictions of all foods ranging between 6 months and 50 years.

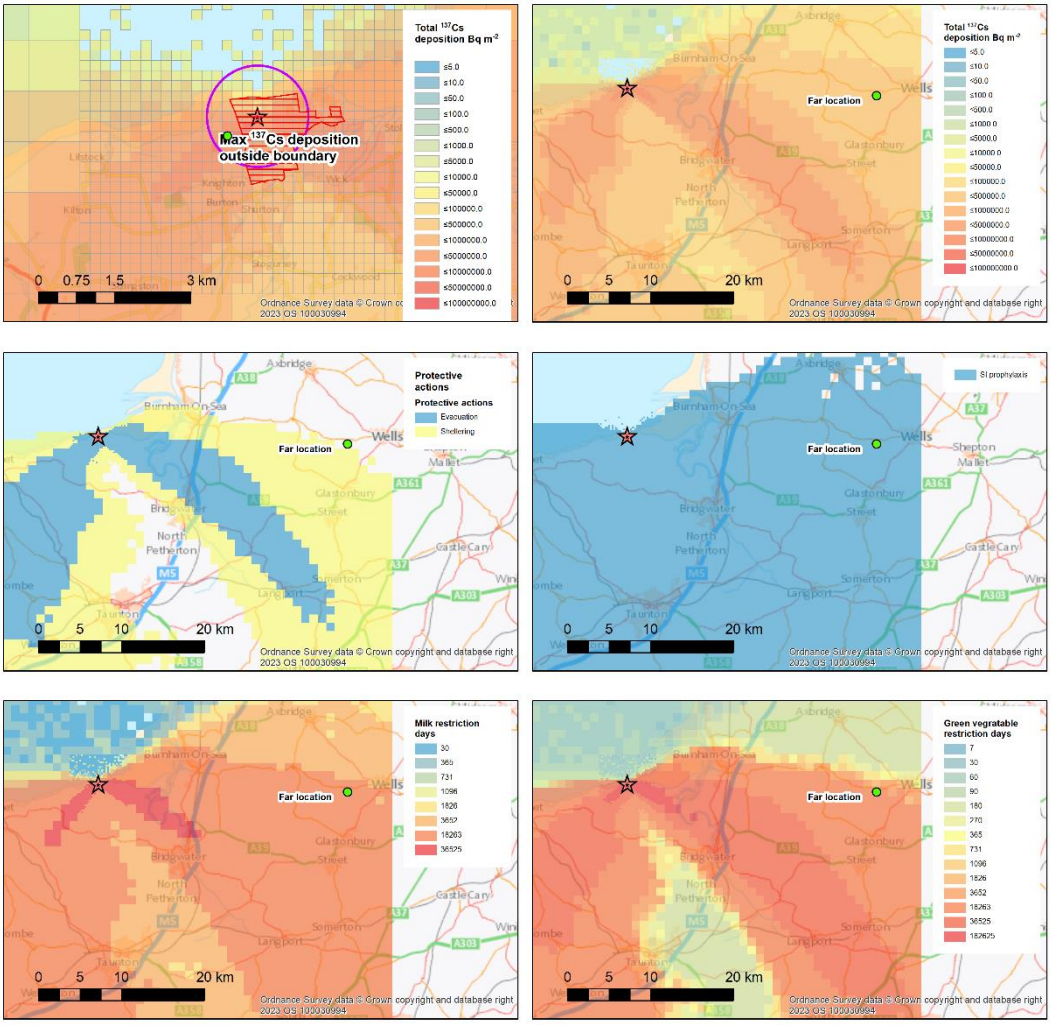


Figure 35 Predicted consequences of dispersal of ST4-Chornobyl. Model run begins 23:00hrs 09/05/20, giving predominantly dry conditions.

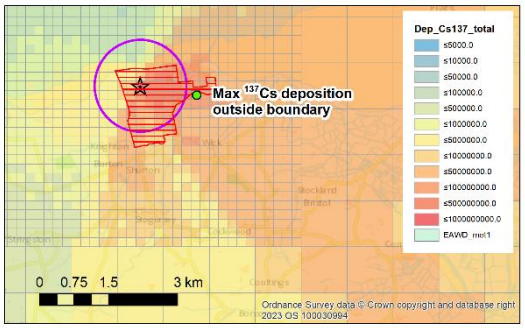
8.2 Wet deposition

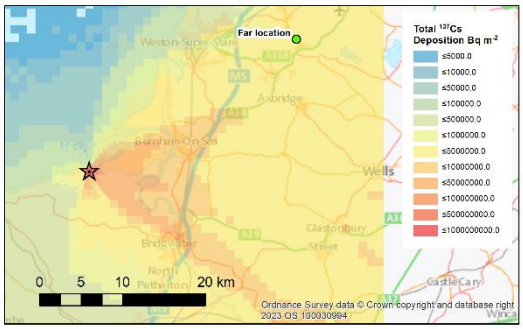
The weather sequence selected as wet begins at 20:00hrs 05/07/20. The pattern of total deposition of ^{137}Cs is shown in Figure 36.

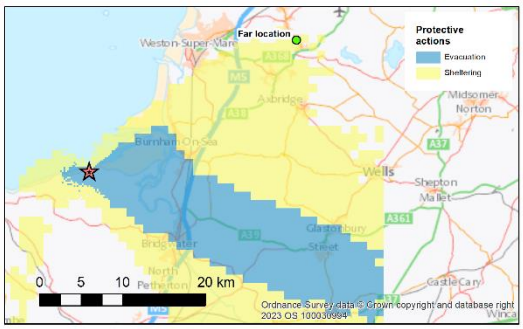
The near location was placed in the grid-square selected as having the maximum total deposition outside of the boundary fence. The proportion of wet deposition is greater than 84%. Based on the upper ERL criteria. evacuation and stable iodine prophylaxis would be advised with relocation being required for 20 years (based on a criterion of 20mSv y^{-1} and assuming no clean-up is performed). Milk and green vegetable restrictions are predicted to be required for 500 years with predicted restrictions for all foods ranging between 50 years and

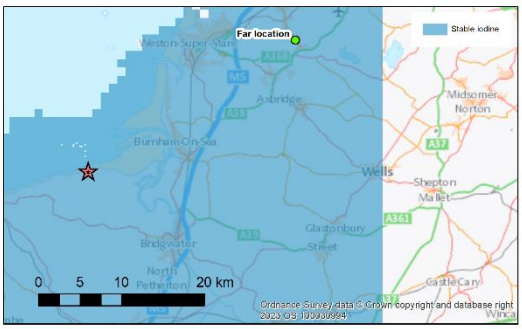
500 years (because of long-lived radionuclides, and assuming that no other agricultural protective actions are applied).

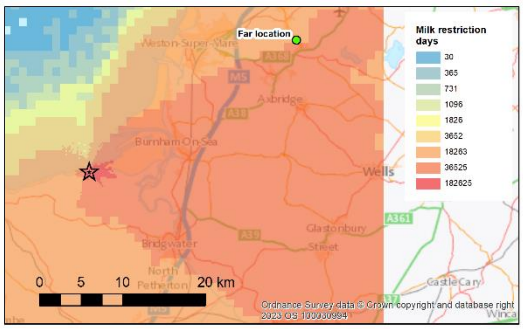
The far location was placed off the plume centre line, in the grid-square having the maximum total deposition at 30km outside the zones where sheltering and evacuation would be required (based on upper ERL criteria) and outside the zone where relocation would be required without clean-up (based on a criterion of 20mSv y^{-1}), the proportion of wet deposition at this location is about than 90%. Stable iodine prophylaxis is required at the upper ERL, Milk restriction lasts 50 years and green vegetable restriction lasts 100 years. with predicted restrictions for all foods ranging between 10 years and 100 years.

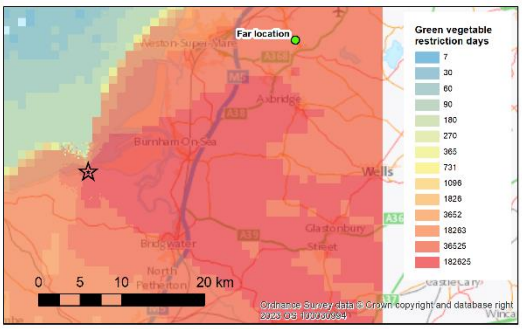












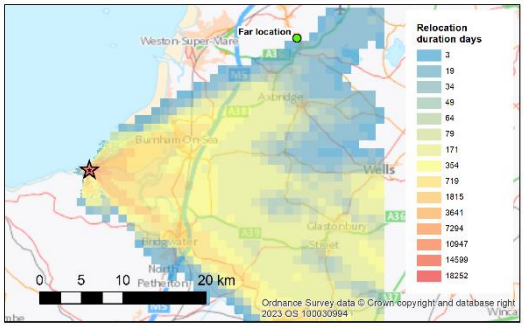


Figure 36 Predicted consequences of dispersal of ST4-Chornobyl. Model run begins 20:00hrs 05/07/20, giving predominantly wet conditions.

8.3 Dose profiles

The predicted lifetime effective doses at the near location range from 3.3 to 6.3 Sv, and at the far locations from 54 to 130 mSv. In all examples the thyroid dose is largest followed by the lung dose. The lifetime thyroid doses at the near locations range from 17 to 52 Sv, and at the far locations from 200 to 660 mSv.

There is some age-dependency in thyroid dose, with the 1-year-old dose being about twice that of the 35-year-old. The lung dose and most of the other organs exhibit either no or very weak age dependency.

A more detailed description of the dose profiles can be found in Appendix A4.

8.4 Deterministic risks

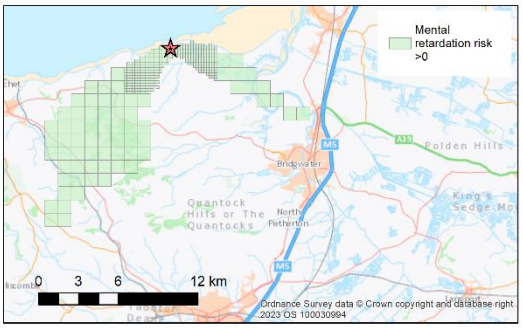


Figure 37 ST4-Chornobyl-dry – predicted extent of deterministic effects

In the ST4-Chornobyl-dry example (shown in Figure 33) the predicted risk of mental retardation is less than 0.0002 at all locations and extends to 16km.

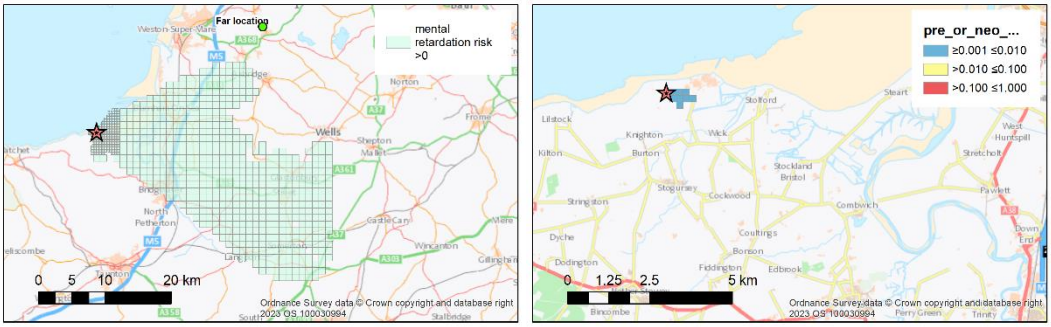


Figure 38 ST4-Chornobyl-dry – predicted extent of deterministic effects

In the ST4-Chornobyl-wet example (shown in Figure 34), the predicted risk of cataracts is <0.1 at all locations and does not extend off-site, the predicted risk of mental retardation is <0.0001 at all locations and extends to the edge of the grid, and the predicted risk of pre-natal and neo-natal death is <0.01 at all locations and extends about 1km.

8.5 Risk profiles

8.5.1 Incidence

Figure 39 show all the predicted risks of incidence of cancer in all tissues.

Relative risks of incidence are mostly consistent between examples (i.e. the graphs appear mostly the same but with a few differences). The near locations have high exposures and a few of the risks for thyroid and all solid ('Solid') tissues have been truncated.

The following features can be seen in all graphs:

- Thyroid and all-solid ('Solid') has the largest risk of incidence, followed by lungs. Notably in the wet examples, breast and remainder ('Other solid') tissues stand out more than in the dry examples.
- Generally, there is good consistency between the ICRP and BEIR results. However, with the lung dose being one of the most significant individual tissues, it is notable that BEIR risks are larger and that the age-risk relationship seen in the ICRP and BEIR predictions are different.
- For thyroid, bone marrow ('Leuk'), colon, breast, bladder, ovary, stomach, liver, remainder ('other solid') and all-solid-cancer ('Solid') risks, there is a clear age dependency, with 1-year-old having the most risk, followed by 10-year-old, 35-year-old, and 60-year-old. For lung, and oesophagus there is no strong or consistent age dependency.
- For thyroid, lungs and all- tissues, the female risks are larger, whereas for all other organs the male risks are larger or the same.
- The age/sex-average risks estimated using ICRP Publication 103 nominal factors track the adult risk but are generally not conservative for most tissues or ages.

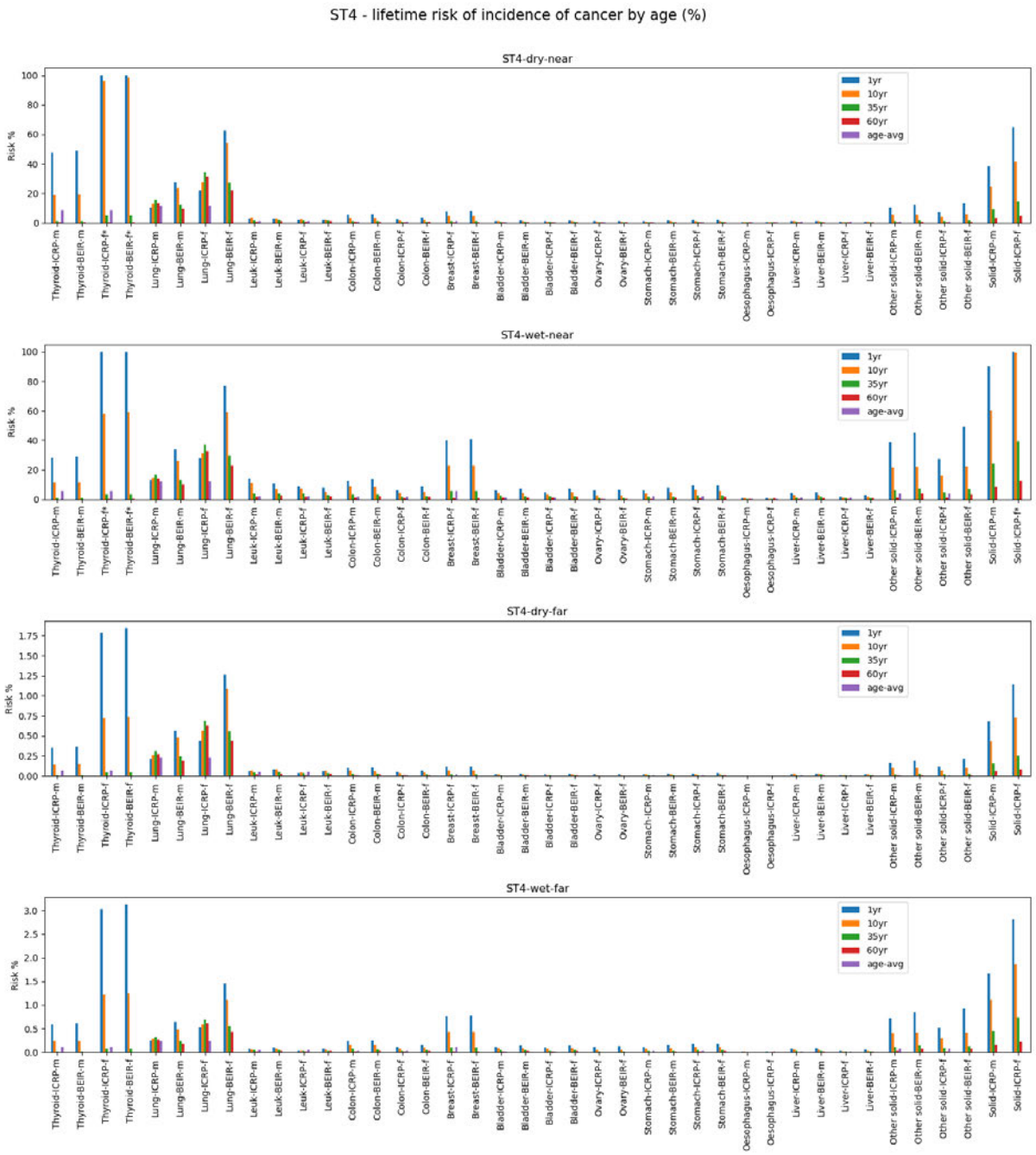


Figure 39 ST4-Chornobyl – all predicted risks of incidence of cancers using both ICRP and BEIR models

8.5.2 Fatalities

Figure 40 show all the predicted risks of fatalities of cancer in all tissues.

Relative risks are mostly consistent between examples (i.e. the graphs appear mostly the same but with a few small differences). The near locations have high exposures and a few of the risks for thyroid and all solid ('Solid') tissues have been truncated.

The following features can be seen:

- The lung tissue has the largest risks of fatality, followed by the remainder organ (‘Other solid’)
- For thyroid, lungs, stomach and all-solid-cancers (‘Solid’), the female risks are generally larger, whereas for all other tissues the male risks are larger or the same, or the risks are so small that differences are not discernible.
- For thyroid, lung (‘mort’), bone marrow (‘Leuk’), colon, breast, ovary, stomach, liver, remainder (‘Other solid’) and all-solid-cancer (‘Solid’) risks, there is a clear age dependency, with generally 1-year-old having the most risk, followed by 10-year-old, 35-year-old, and 60-year-old. For the other tissues including lung (‘inc’) the relationship is either not strong, or not consistent, or risks are so small that the relationship is not discernible.
- The prediction using a lethality fraction are generally a good approximation for the modelled predictions (‘mort’). The lung (‘inc’) predictions are between about 50% and 100% of the lung (‘mort’) but do not exhibit the same age dependency.

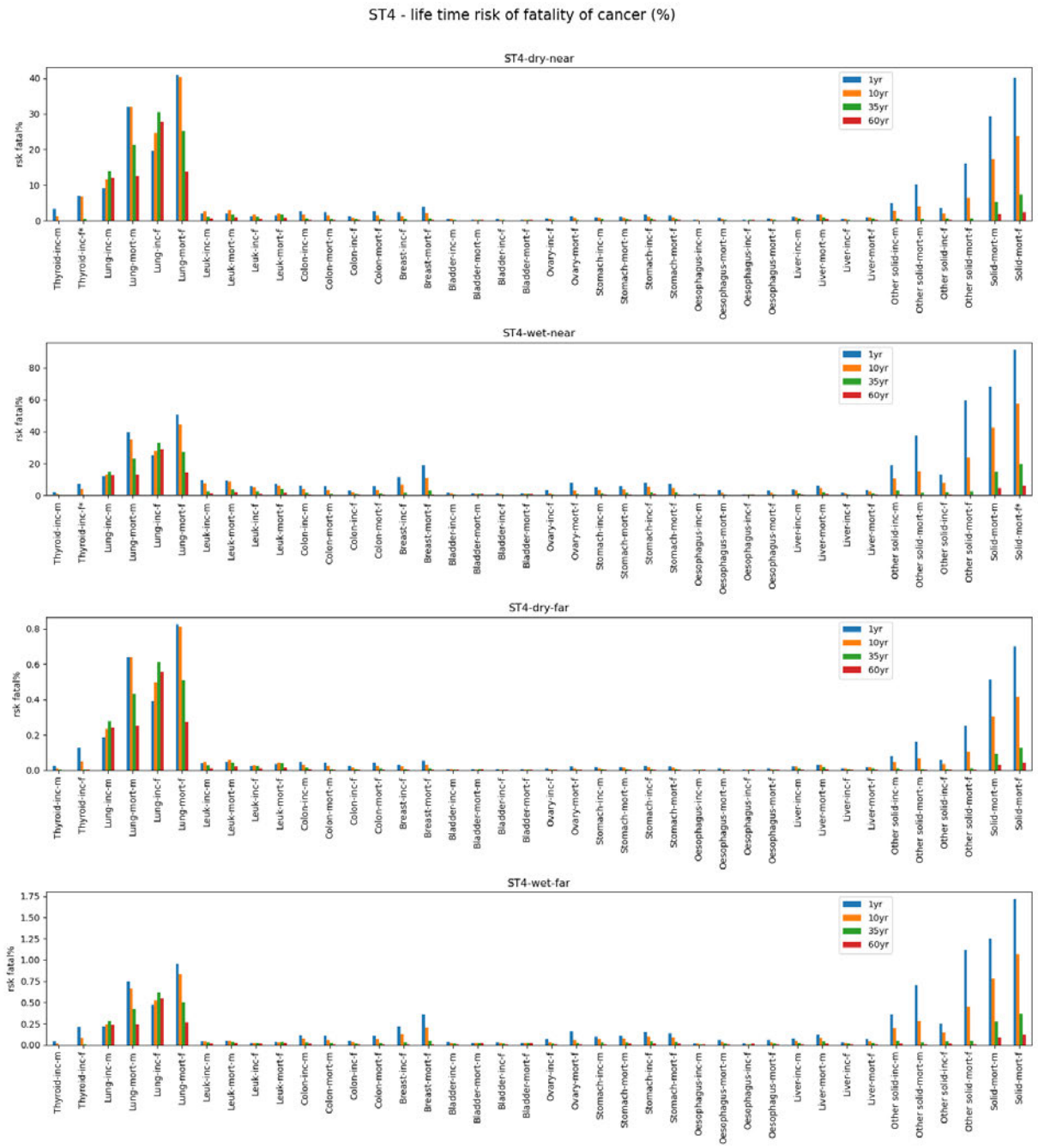


Figure 40 ST4-Chornobyl – all predicted risks of fatality of cancers. Risks are calculated by either applying an age/sex independent lethality fraction to the corresponding risk of incidence ('inc'), or the ICRP mortality risk models ('mort').

8.5.3 Detriments

Figure 41 shows all the predicted risks of detriments in all tissues.

Relative risks are similar but there are some inconsistencies between examples (i.e. the graphs appear mostly the same but with a few differences with the near-wet example being the most different). The near locations have high exposures and a few of the risks for thyroid have been truncated.

The following features can be seen:

- Generally thyroid has the largest risk of detriment, followed by lungs and remainder (‘Other solid’). In the near-wet example, bone marrow (‘Leuk’) has the largest detriment, followed by thyroid, remainder (‘Other solid’), lung and breast.
- For thyroid, bone marrow (‘Leuk’), colon, breast, bladder, ovary, stomach, liver and remainder (‘other solid’), there is a clear age dependency, with 1-year-old having the highest risk, followed by 10-year-old, 35-year-old, and 60-year-old. For the other tissues the relationship is either not strong, or not consistent, or risks are so small that the relationship is not discernible.
- For thyroid, lungs and stomach, the female risks are generally larger, whereas for all other tissues the male risks are larger or the same, or the risks are so small that differences are not discernible.

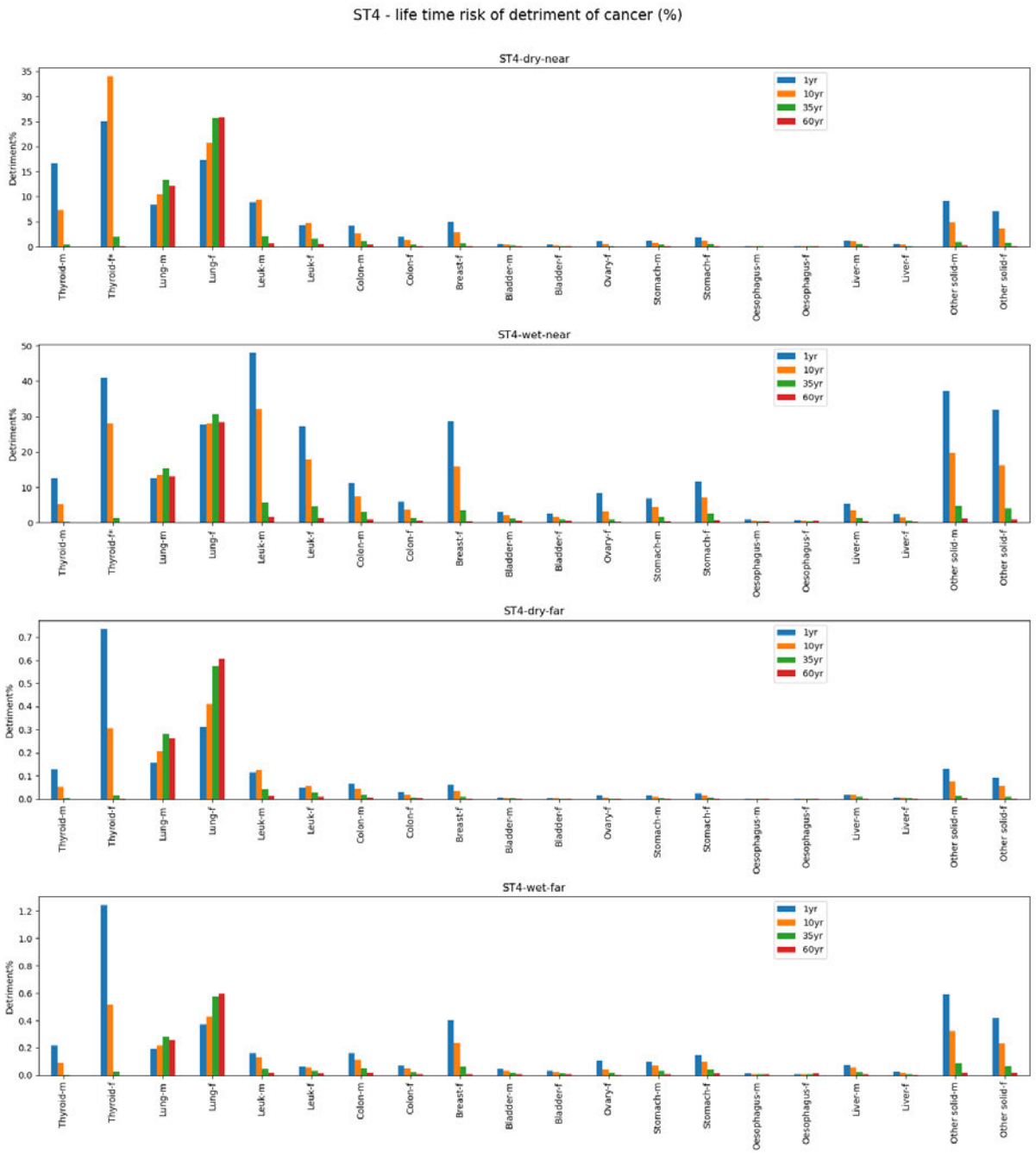


Figure 41 ST4-Chornobyl – all predicted risks of detriment of cancers

8.6 Summary

There is some age dependency in the predicted doses, but for most organs it is slight. The most distinct is the thyroid where the 1-year-old dose is a factor of about 2 higher than the 35-year-old.

The near examples have organ dose predictions that are very high and extend into the range where acute or deterministic effects may be expected. Consequently, the predicted risks are very high and some of the predictions of thyroid cancer risk of incidence, fatality and detriment are truncated at 100% or derived from truncated values. Therefore, there must be a question-

mark over the validity of all the near predictions even for the limited purpose of investigating age and sex dependency. The remainder of the discussion concerns the far examples.

On both the far examples, the risk of incidence of thyroid cancer is highest, followed by that for lung cancer. The thyroid risks show a strong age-dependency with the 1-year-old about a factor of 2-3 higher than the 10-year-old, which is a factor of 2-10 (depending on sex) higher than the age/sex-average risks estimated using ICRP Publication 103 nominal risk factors. In contrast, there is not a strong age dependency in the lung cancer incidence risk (though the BEIR VII prediction shows more dependency than the ICRP one).

Both lung and thyroid cancer incidence risk show sex dependency in both the ICRP and BEIR VII results. With the female thyroid cancer incidence risk about a factor of 5-6 higher than the male and the female lung cancer incidence risk about a factor of 2-3 higher than the male.

Thyroid cancer is relatively a less lethal form of cancer, so it is not the tissue with the highest risk of fatality, but the female risk is still predicted to be higher than most tissues. ICRP have no direct model for thyroid cancer fatality, and when estimating using an age and sex independent lethality fraction from the incidence risk ('inc'), the age and sex dependencies seen in the incidence risk are carried through to the fatality risk.

In the far-dry example, the highest risk of fatality is from lung cancer, and it is also one of the highest in the far-wet example. ICRP do have a model of lung cancer fatality risk, and when this is applied to the lung doses, an age dependency is apparent with the 1-year-old having a risk that is about a factor of 2 higher than the 35-year-old. This contrasts with the fatality risk estimated using a lethality fraction, where the age dependencies are not strong, but the highest risk is to the 35-year-old. Lung cancer fatality risks have a slight sex dependency but within a factor of 2 for most ages. The remainder organs ('Other solid') are another important tissue for cancer fatality in both far examples, this tissue shows a distinct age dependency and a slight sex dependency as do many of the other tissues.

Figure 42 gives the predicted age and sex-specific risks of incidence and fatality, and detriment summed across all organs in the study, compared to the age and sex-averaged detriment calculated by multiplying the age-specific effective dose with the ICRP nominal coefficient for cancer detriment of 5.5% Sv^{-1}. Females show the higher risks and detriments compared to males. Younger age groups show the largest differences to the nominal detriments.

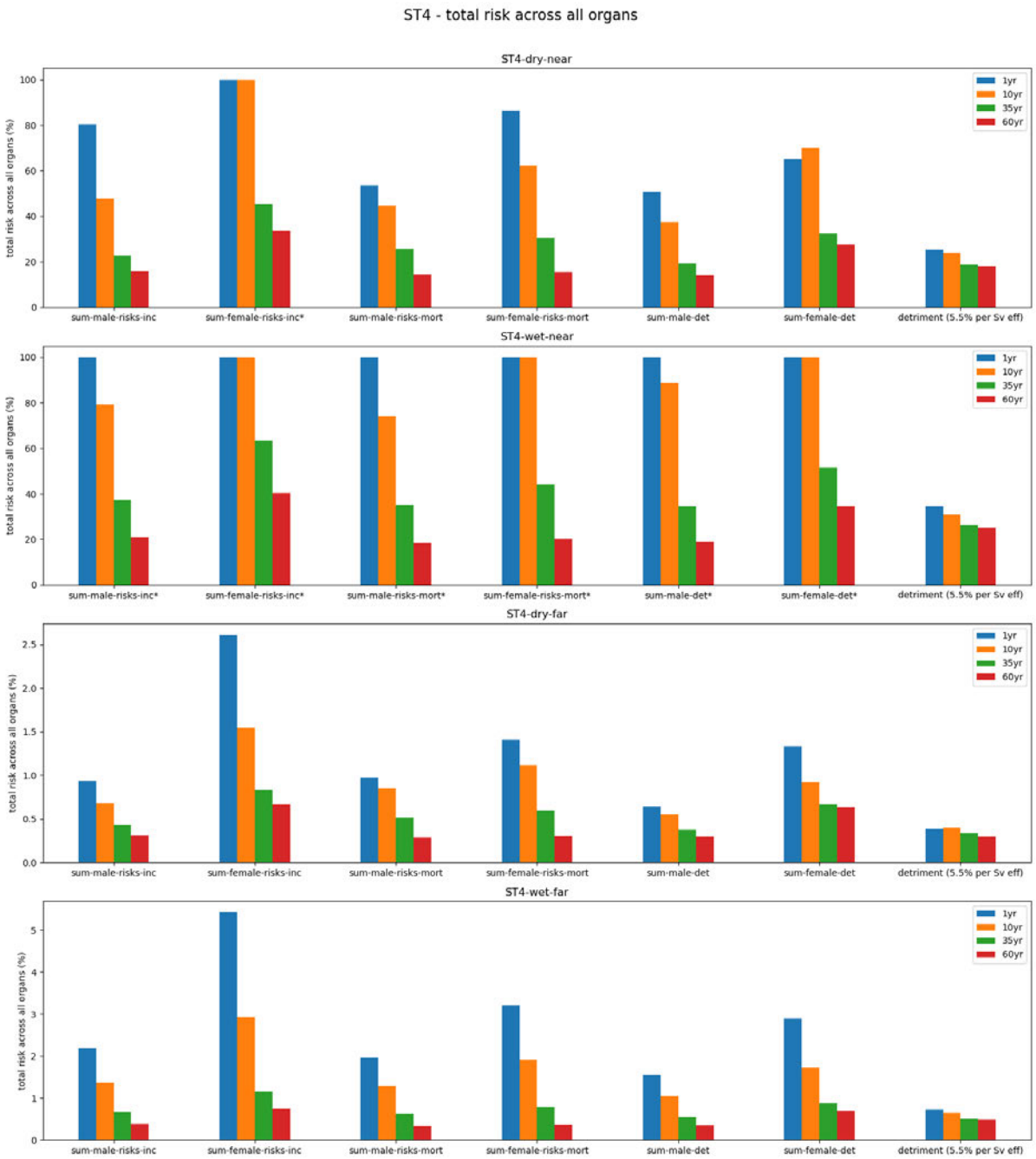


Figure 42 ST4-Chornobyl – Age and sex-specific risks of cancer and detriment summed across tissues in the study compared with detriment calculated by applying ICRP nominal risk coefficient of 5.5% Sv^{-1} to the age-specific effective dose

8.7 Comparison with Chornobyl Accident

Any comparison between the results presented here and the real consequences of the Chornobyl accident must necessarily be very tentative and must bear in mind the considerable differences between the scenarios, and the uncertainties in both the predicted consequences and the real consequences. In particular:

- The Chornobyl source term used in this report was constructed from several international reports, but there is a lot of uncertainty about what was released, the timing of the release, particle size ranges, and the height of the release that will never be fully resolved.
- The weather used in this document was chosen to give a release in generally dry and in generally wet conditions and neither necessarily resembles the weather around the Chornobyl plant in 1986, of which there is much uncertainty.
- Consumption habits, health care capacity, general state of health (e.g. iodine deficiency) and farming practices were very different in 1980's Soviet Union compared to modern UK.
- The scenarios here assume the imposition of protective actions according to UK criteria and it should be noted that the UNSCEAR committee report 2008 (UNSCEAR, 2010) found elevated doses to the thyroid, almost entirely because of the consumption of fresh milk containing ^{131}I in the first few weeks after accident. In the ST4-Chornobyl scenario investigated here, the ingestion pathway is effectively eliminated for the thyroid by food restrictions, and the principal pathway for thyroid dose in all examples was inhalation, see Figure A32. In this respect the Chornobyl accident more closely resembles ST1 which also has no food restriction, albeit with a vastly different magnitude of release.
- The risks predicted in this document are for expressed risks, whereas, though the Chornobyl accident was more than 30 years ago and the vast majority of dose has been delivered, not all the cancers that will arise from that dose have been expressed.

Notwithstanding the above, UNSCEAR, found a difference between adult and child dose, with pre-school children receiving 2-4 times the population average. In ST4-Chornobyl the 1-year-olds received a thyroid dose which is a factor of two greater than adults and in ST1 (depending on assumptions about locally produced milk consumption) between a factor 3 to 9 times greater.

Average dose to the thyroid for all evacuees was found to be 500 mGy with a maximum of more than 5000 mGy, and the average dose to those who were not evacuated was 100 mGy with a maximum of more than 1000mGy. In ST4-Chornobyl the predicted thyroid dose for the near locations was between 17000 to 52000 mSv which is more than an order of magnitude higher than the evacuees. For the far location, thyroid doses between 200 to 660 mSv were predicted which is similar to the average dose to thyroid for the evacuees

UNSCEAR observed both the age dependency and sex dependency in risk seen in both the ST1-DBA and ST4-Chornobyl predictions. However, they acknowledge considerable uncertainty, for example an estimated 0.25 (25%) of all incidences of thyroid cancer in non-evacuated residents of Belarus, Ukraine and the most contaminated oblasts of Russia Federation who were children or adolescents at the time is due to radiation exposure, but this is given an uncertainty range extending at least from 0.07 to 0.5 (7 to 50%).

9 Discussion

This project aims to address whether higher radio-sensitivities in younger age groups and females could result in insufficient conservatism in the off-site risks to the public presented in safety case submissions for new nuclear sites as these risks are typically calculated using age and sex averaged risk factors. This was done by performing dose and risk calculations for a necessarily limited number of source-terms, deposition conditions, and distances, and, for one source term (ST1), additional assumptions about local milk consumption.

9.1 Main findings from the results

For all the source terms considered, variations in the estimated risk of the incidence of cancer, detriment and fatality with age and sex were found and particularly so for the most radio-sensitive organs such as the thyroid, breast, lung, bone marrow and remainder organs. Most of the results discussed in this section relate to ST1 as this the most representative source term of those considered for accident safety cases for new nuclear power stations and as it also showed the most variation in risk by age and sex due to the importance of ^{131}I in the ingestion pathway and its uptake by the thyroid. ST1 is of a magnitude that might be expected for a design basis accident. The other source terms considered are all severe beyond-design-basis accidents, and given that it is realistic to assume that food restrictions would be implemented in such circumstances, this has been assumed in the calculations. For ST1, food restrictions would not be required, and this means that in this case, the ingestion pathway is significant. As can be seen in Figure A5, which shows the contribution to lifetime thyroid dose by pathway for ST1, ingestion is the major contributor to the dose. Figure A11 gives the contribution to lifetime thyroid dose by radionuclide and shows that ^{131}I is the main radionuclide contributing to the thyroid dose.

Age dependency has been seen clearly in many of the risk endpoints (incidence, fatality, and detriment) of the example calculations. In most cases, where it is seen in a tissue that is a significant contributor to total risk, it has been the 1-year-old who has the highest risk, followed by the 10-year-old and then the adults (Table 6 below). The exception to this pattern has been the lung and liver cancer incidence and detriment risks where often it is the adults who are most at risk. This ties in with the sensitivity analysis by Zhang et al (2020) who calculated that for most cancers, the detriment for the young age-at-exposure population (0-14) is higher than that for a whole population averaged (0-84 years) with the detriment being two or three times higher for the thyroid, breast and other solid cancers.

The variation of risk of cancer incidence with sex is also seen, with some tissues notably the thyroid having risks higher for females. For ST1, risks to females were found to be higher than those to males, particularly for females young at the age of exposure (Figure 4 and Figure 5). For example, the risk of incidence of thyroid cancer is four to five times higher for a 1-year-old female than 1-year-old males. However, as shown in Table 9, there are only limited differences in the risk between the 35-year-old female to the risk calculated using 35-year-old and the age-averaged nominal risk factors. Zhang et al (2020) compared the sex-averaged detriment with those for males and females separately and found that for thyroid and lung cancers, detriment for females appears to be higher, whereas the inverse is observed for liver,

colon and other solid cancers (ovary cancer and female breast cancer are obviously sex-specific).

This study also demonstrates that the thyroid does show the most extreme age- and sex-variation of risk, with differences of up to three orders of magnitude between the 1-year-old-female and both the age/sex average risk (Table 8) and the 35-year-old-male risk (Table 7). Other organs show age and sex variation in risk of cancer incidence, detriment and fatalities but not as great as for the thyroid. For example, the fatality risk of other solid cancers (remainder organs) for the 1-year-old female is estimated to be 30 times higher than for the 35-year-old male (Table 7).

The risk of cancer incidence in the thyroid also illustrates the multiplicative effects of combining age-specific dose and age/sex-specific risk. The variations in thyroid dose are largest for ST1 reflecting the importance of ^{131}I in the milk pathway for this organ. The risk calculation step magnifies the differences in the dose with age-at-exposure and introduces differences in the risks according to sex. Since the same dose was used for males and females in the calculations, any differences in risk between the sexes derives entirely from the risk models.

For ST1, the following differences in the dose and risk of incidence of cancer were calculated for the thyroid:

- Dose to a 1-year-old was calculated to be three to nine times higher than that of 35-year-old (Table 7).
- Risk of the incidence of cancer in a 1-year-old-female at age-of-exposure based on the LAR risk model and the age-averaged risk calculated using a 1-year-old dose and the ICRP nominal factor from ICRP 103 was estimated to be a factor of around 14 times higher (Table 8).
- Risk of the incidence of cancer in a 1-year-old-female at age-of-exposure based on the LAR risk model and the age-averaged risk calculated using a 35-year-old dose and the ICRP nominal factor from ICRP 103 was estimated to be a factor of around 50 to 120 times higher (Table 8)
- Risk of the incidence of cancer in a 1-year-old-female at age-of-exposure based on the LAR risk model and the 35-year-old risk calculated using 35-year-old dose and the LAR risk model is estimated to be a factor of around 300 to 900 times higher (Table 7)

It should be noted that an estimate of the incidence risk of cancer to a 1-year-old-female made using the 1-year-old lifetime dose and the female 0-9 years old at age of exposure risk factor from ICRP Publication 147 (Table 2.4, given as 1.9 cases per 100 per Gy for thyroid) would be within a factor of 2 to 3 of the risk based on the LAR risk model. Thus, both methodologies agree quite well.

There is approximately an order of magnitude difference in the predicted risk between a 1-year-old-female and the age/sex average thyroid cancer incidence risk when calculated with an age-specific dose. This is consistent with Harrison et al (2023) which looked at medical diagnostic x-ray examinations and found a factor of two to three increase in risk per Sv (effective dose) for the lower 0-9 years old group compared to 30-39 years old group across all organs but identified that the age-dependency was more pronounced for thyroid cancers.

There is no thyroid cancer fatality risk model available. Therefore, the thyroid fatality risk was calculated by using the thyroid cancer incidence risk model to predict incidence risk and

multiplying that by a lethality fraction. The study also took the opportunity to compare the prediction of fatality of the ICRP mortality risk model compared with the prediction of the ICRP incidence risk model for organs where both models are available. In general, the approaches were in fair agreement, with differences identified in the individual sections above and no systematic overestimation was identified for one or other of the approaches. A shortcoming of the lethality fraction approach was that the lethality fractions were not age or sex or even population-specific with the values used being global ones taken from the ICRP detriment methodology (ICRP, 2022). For example, the 0.07 value taken for thyroid cancer lethality is certainly an overestimation when considering the UK population.

Table 6 Age of most exposed and the age/sex of most at risk[a] for each example and most significant organ. Not all organs show the same age/sex dependency across examples, where this occurs the modal age/sex group is given along with the label 'inconsistent'.

Source term	Organ	Most exposed	Most at risk of incidence	Most at risk of fatality[g]	Highest risk of detriment
ST1-DBA	Thyroid	1-year-old	1-year-old-female	1-year-old-female	1-year-old-female
ST2-acute[c]	Thyroid	1-year-old	1-year-old-female	1-year-old-female	1-year-old-female
	Breast[d]	1-year-old	1-year-old-female	1-year-old-female	1-year-old-female
	Remainder	1-year-old	1-year-old-male	1-year-old-female	1-year-old-male
ST3-fuel	Bone marrow[e] (far)	1-year-old	1-year-old-male	1-year-old-male	1-year-old-male
	Bone marrow[e] (near)	1-year-old	1-year-old-male	1-year-old-male	1-year-old-male
	Lung	35-year-old/ inconsistent	35-year-old-female/inconsistent	1-year-old-female	female/inconsistent
	Liver	35-year-old/ inconsistent	10-year-old-male/inconsistent	10-year-old-male/inconsistent	1/10-year-old-male/inconsistent
	Breast[d]	1-year-old	1-year-old-female	1-year-old-female	1-year-old-female
	Remainder	1-year-old	1-year-old-male	1-year-old-female	1-year-old-male
	Thyroid[f]	1-year-old	1-year-old-female	1-year-old-female	1-year-old-female
ST4-Chornobyl[c]	Thyroid	1-year-old	1-year-old-female	1-year-old-female	1-year-old-female
	Lung	35-year-old	35-year-old-female	1-year-old-female	60-year-old-female
	Breast	1-year-old	1-year-old-female	1-year-old-female	1-year-old-female
	Remainder	1-year-old	1-year-old-male	1-year-old-female	1-year-old-male

a Only results from the ICRP risk models are included in this table. BEIR VII model of lung cancer incidence showed an age dependency that was not apparent in the ICRP model.
b Other organs, not included in the table, may also exhibit sex and age dependency but were not found to be very significant contributors to risk in these examples.
c Only far locations included for ST2-acute and ST4-Chornobyl as near locations have very high doses predicted that may lead to deterministic effects.
d Male doses and risks were not assessed for the breast organ, but known to be significantly lower than female.
e Leukaemia risk model is linear-quadratic and therefore there are distinct differences between near and far locations due to large differences in doses.
f Thyroid is not predicted to be significant organ for risk in the ST3-fuel examples, but it has been included here for comparison with other examples.
g Considered only the modelled fatality risk and not those risks calculated by applying a lethality fraction to the modelled incidence risk, except for thyroid where no fatality risk model is available.

Table 7 Ratios of organ dose between 1-year-old and 35-year-old. Ratios of risk[a] between most at risk age/sex and 35-year-old-male. Ratio of detriment between most at risk group and 35-year-old-male.

Source term	Organ[b]	Ratio of dose (1-y-old to 35-y-old)	Ratio of risk of incidence (most at risk age/sex to 35-year-old-male)	Ratio of risk of fatality[g] (most at risk age/sex to 35-year-old-male)	Ratio of detriment (highest detriment age/sex to 35-year-old-male)
ST1-DBA	Thyroid	3 to 9	300 to 900 (1f)	300 to 900 (1f)	400 to 1000 (1f)
ST2-acute[c]	Thyroid	2	200 (1f)	200 (1f)	200 to 240 (1f)
	Breast[d]	1.1 to 1.2	7 (1f)	6 to 7 (1f)	6 to 7 (1f)
	Remainder	1.2 to 1.4	6 (1m)	30 (1f)	5.5 to 6 (1m)
ST3-fuel	Bone marrow(far)[e]	1 to 2	2 to 3 (1m)	1.5 to 2 (1m)	5 (1m)
	Bone marrow(near)[e]	1.3 to 1.4	8 to 10 (1m)	5 to 7 (1m)	11 to 12 (1m)
	Lung	0.8 to 1.3	2 to 3 (35/1f)	2 to 3 (1f)	1.5 to 2 (35/10/60f)
	Liver	0.5 to 1.2	2 to 3 (10/1m)	2 to 3 (10m/1m)	1.5 to 3 (1m)
	Breast[d]	1.1 to 1.2	7 (1f)	6 to 7 (1f)	6 to 9 (1f)
	Remainder	1.2 to 1.4	6 (1m)	30 (1f)	6 to 8 (1m)
	Thyroid[f]	1.1 to 1.2	120 (1f)	120 (1f)	140 to 200 (1f)
ST4-Chornobyl[c]	Thyroid	2	200 (1f)	190 to 200 (1f)	220 (1f)
	Lung	0.8 to 0.9	2 (35f)	2 (1f)	2 (60f)
	Breast[d]	1.1 to 1.2	7 (1f)	6 to 7 (1f)	6 to 7 (1f)
	Remainder	1.5 to 2	6 to 9 (1f)	35 to 49 (1f)	7 to 9 (1m)

a Only results from the ICRP risk models are included in this table. The BEIR VII model of lung cancer incidence showed an age dependency that was not apparent in the ICRP model.
b Other organs, not included in the table, may also exhibit sex and age dependency but were not found to be very significant contributors to risk in these examples.
c Only far locations included for ST2-acute and ST4-Chornobyl as near locations predict very high doses that may lead to deterministic effects.
d Male doses and risks were not assessed for the breast organ, but are known to be significantly lower than female. Therefore ratios for breast are most at risk age female to 35-year-old female.
e Leukaemia risk model is linear-quadratic and therefore there are distinct differences between near and far locations due to large difference in doses.
f Thyroid is not predicted to be significant organ for risk in the ST3-fuel examples, but it has been included here for comparison with other examples.
g Considering only the modelled fatality risk and not those risks calculated by applying a lethality fraction to the modelled incidence risk, except for thyroid where no fatality risk model is available.

Table 8 Ratios of risk[a] of incidence between most at risk age/sex to the risk estimated from 35-year-old dose combined with age-averaged nominal risk factors, and corresponding age dose and age-averaged nominal risk factors

Source term	Organ[b]	Ratio of risk of incidence (most at risk age/sex to 35-year-old dose with age/sex averaged nominal risk factor)	Ratio of risk of incidence (most at risk age/sex to corresponding age dose with age/sex averaged nominal risk factor)
ST1-DBA	Thyroid	50 to 120 (1f)	14 (1f)
ST2-acute[c]	Thyroid	24 to 30 (1f)	14 (1f)
	Breast[d]	8 (1f)	6 to 7 (1f)
	Remainder	10 (1m)	7 to 8 (1m)
ST3-fuel	Bone marrow(far)[e]	2 (1m)	1.2 to 1.4 (1m)
	Bone marrow(near)[e]	16 to 19 (1m)	12 to 15 (1m)
	Lung	3 (35/1f)	2 to 3 (35/1f)
	Liver	2 to 5 (10/1m)	3 to 4 (10/1m)
	Breast[d]	6 to 7 (1f)	6 (1f)
	Remainder	8 to 9 (1m)	7 (1m)
	Thyroid[f]	12 to 13 (1f)	11 (1f)
ST4-Chornobyl[c]	Thyroid	26 to 28 (1f)	14 (1f)
	Lung	3 (35f)	3 (35f)
	Breast[d]	6 to 7 (1f)	6 (1f)
	Remainder	10 to 13 (1m)	7 (1m)

a Only results from the ICRP risk models are included in this table. The BEIR VII model of lung cancer incidence showed an age dependency that was not apparent in the ICRP model.
b Other organs, not included in the table, may also exhibit sex and age dependency but were not found to be very significant contributors to risk in these examples.
c Only far locations included for ST2-acute and ST4-Chornobyl as near locations have very high doses predicted which may lead to deterministic effects.
d For most organs the nominal risk factor is sex-averaged but for the breast it is female specific.
e Leukaemia risk model is linear-quadratic and therefore there are distinct differences between near and far locations due to large differences in doses.
f Thyroid is not predicted to be significant organ for risk in the ST3-fuel examples, but it has been included here for comparison with other examples.

Table 9 Ratios of risk[a] of incidence between risk calculated for 35-year-old-female to risk calculated for 35-year-old-male, and risk estimated from 35-year-old dose and age-averaged nominal risk factors. For each example and most significant organs.

Source term	Organ[b]	Ratio of risk of incidence (35-year-old-female to 35-year-old-male)	Ratio of risk of incidence (35-year-old-female to 35 y-old dose with age/sex averaged nominal risk factor)
ST1-DBA	Thyroid	4 to 5	0.6
ST2-acute[c]	Thyroid	4	0.6
	Breast[d]	-	1.1
	Remainder	0.8	1.4
ST3-fuel	Bone marrow(far)[e]	0.8 to 0.9	0.6 to 0.7
	Bone marrow(near)[e]	1	1.9 to 2.1
	Lung	2	2.6 to 3
	Liver	0.4 to 0.5	0.5
	Breast[d]	-	0.9 to 1
	Remainder	0.8	1.1 to 1.2
	Thyroid[f]	4	0.4 to 0.5
ST4-Chornobyl[c]	Thyroid	4	0.6
	Lung	2	3
	Breast[d]	-	0.9
	Remainder	0.8	1.2

a Only results from the ICRP risk models are included in this table. The BEIR VII model of lung cancer incidence showed an age dependency that was not apparent in the ICRP model.
b Other organs, not included in the table, may also exhibit sex and age dependency but were not found to be very significant contributors to risk in these examples.
c Only far locations included for ST2-acute and ST4-Chornobyl as near locations have very high doses predicted which may lead to deterministic effects.
d Male doses and risks were not assessed for the breast organ but are known to be significantly lower than female. Therefore, there is no specific male risk to be compared. For most organs the nominal risk factor is sex-averaged but for the breast it is female specific.
e Leukaemia risk model is linear-quadratic and therefore there are distinct differences between near and far locations due to large differences in doses.
f Thyroid is not predicted to be significant organ for risk in the ST3-fuel examples, but it has been included here for comparison with other examples.

Figure 16, Figure 17, Figure 28, Figure 34 and Figure 42 compare the sex and age-specific risks and detriments summed across all organs, with age and sex-averaged detriment calculated by applying the ICRP Publication 103 nominal risk coefficient for cancer detriment 5.5 10^{-2} Sv^{-1} to the age specific effective dose. This value is also referenced in ONR's Technical Assessment Guide-45 (ONR, 2019) as a risk conversion factor for late health effects for the whole population. There are clearly differences between the estimates of risk based on organ doses and those based on effective doses. This is particularly pronounced for younger age groups and for females, with the nominal risk being applied to effective dose approach generally giving the smaller estimate by around an order of magnitude in the most severe cases when compared to total incidence risk. The results arecloser for fatality risk and detriment. For example, the risk of fatality to the most vulnerable group (1-year-old female) calculated using organ-specific doses and risks was between 2 to 6 times higher for ST2, ST3 and ST4 than ICRP sex-age and population averaged values. This is ignoring locations near to the accident where the very high doses mean that the predicted risk of fatality was at or approaching 100%. It should be noted that this study used a DDREF of 1 as opposed to a value of 2 used by ICRP. Therefore if a consistent DDREF is used across both approaches then the largest difference in risk of fatality calculated using organ-specific risks compared to the nominal risk of ICRP is reduced to a factor of 3.

For this document the nominal risk coefficient for detriment from ICRP Publication 103 was used, but a different coefficient such as the often used 5.0 10^{-2} Sv^{-1} risk of fatality could have been employed and would have given similar conclusions.

Since the nominal risk values are derived by averaging across age, sex and organ, their use when calculating the fatality risk where the effective dose is dominated by the thyroid dose will lead to large overestimate of risk. This is because thyroid cancer fortunately has a very low lethality. It should also be noted that the lethality fraction of 0.07 for thyroid is a global number. The lethality of thyroid cancer in a well-resourced health care country such as the UK will be very much lower than 0.07. Therefore, estimates of risk of fatality from incidence risk multiplied by the lethality fraction are also likely to be large overestimates.

Table 10 gives the ratio of effective doses, risk of incidence of cancer for specific organs, fatality and detriment for 1 and 10-year females to corresponding age and sex averaged endpoints. This table differs from Table 8 in that it compares the 1 and 10-year-old-females to the value averaged over age and sex, whereas Table 8 compares the age and sex most at risk (this is often but not always the 1-year-old-female). The ratio for the lifetime effective dose is based on the ratio of the 1-year-old to 35-year-old dose. The ratio of lifetime risk of cancer incidence of specific tissues was estimated using the ratio of ICRP tissue-specific risk model for a 1-year-old female to an age and sex averaged risk calculated by multiplying the 35-year-old organ dose by a tissue-specific age and sex averaged cancer incidence risk factor taken from ICRP Publication 103 (Table A4.2 Comparison of sex-averaged nominal risks and detriment in whole population based on different methods of calculation. The incidence factors derived by the 'Current Incidence' method were used). The lifetime risk of fatality was calculated by summing over the age/sex/tissue-specific ICRP models (except for the thyroid where a lethality fraction was applied). The summed fatality is then compared to the summed fatalities calculated by multiplying the 35-year-old organ dose by the age/sex averaged cancer fatality risk model from ICRP Publication 103 (Table A4.2, 'Current Incidence' method of calculation). The detriment over all the tissues is calculated by summing over the age/sex/tissue-specific ICRP models of cancer incidence for a 1-year-old female. This detriment is then compared to the detriment calculated by multiplying the 35-year-old organ dose by tissue-specific age/sex averaged cancer detriment factor taken from ICRP Publication 103 (Table A4.2, 'Current Incidence' method of calculation).

Table 10 shows that the risk of fatality for the 1-year-old female was in the range 1 to 20 times higher compared to the population weighted risk, while the detriment was in the range of 1 to 50 times higher. Similar comparisons for incidence of thyroid cancer and leukaemia yielded ranges of 1 to 100 times and 1 to 10 times higher respectively compared to population weighted incidence. However, it should be noted that given the smaller datasets for people exposed when young, there are larger uncertainties in the risk models for those groups, though experts expect younger age groups to be more radio-sensitive. For the 10-year-old group, where there is more information available and therefore greater confidence in the risk estimates, the risk of fatality for the 10-year-old female was in the range 1 to 7 times higher compared to the population weighted risk, while the detriment risk was in the range of 1 to 8 times higher.

Table 10 Ratio of effective doses, risk of incidence of cancer for specific organs, cancer fatality and cancer detriment for 1 and 10-year females to corresponding age and sex averaged endpoints for the considered scenarios

		ST1		ST2		ST3		ST4	
		near	far	near[a]	far	near	far	near[a]	far
1-year-old female									
Effective dose		4	5	2	2	1	1	1	1
Incidence[b]	Thyroid	89	106	1	27	13	13	15	27
	Lung	2	2	1	3	2	3	2	2
	Breast[b]	12	12	8	15	13	13	14	13
	Bone marrow (Leukaemia)[c]	1	1	12	1	11	1	3	1
	Remainder	5	5	4	7	6	6	8	8
Fatality		22	22	1	15	3	8	6	7
Detriment		43	47	1	15	3	3	4	5
10-year-old female									
Effective dose		2	2	1	1	1	1	1	1
Incidence	Thyroid	13	14	1	11	6	6	11	11
	Lung	2	2	1	3	2	3	3	3
	Breast[b]	7	7	5	9	8	8	8	8
	Bone marrow (Leukaemia)[c]	1	1	9	1	5	1	3	1
	Remainder	3	3	3	4	4	4	5	5
Fatality		6	6	1	7	3	5	5	5
Detriment		8	7	1	7	3	2	4	3

a Table 8 omits near locations in scenario ST2 and ST4 where doses are approaching deterministic levels and the risk models give results greater than 100% and are therefore truncated in the graphs. This table gives values for all locations in all scenarios, but it should be noted that the calculation of risk may be hard to defend at these locations

b Unlike Table 8 which uses an age-averaged sex-specific risk factor for breast (from ICRP Publication 103 Table A4.18), this table uses the age- and sex-averaged breast risk factor (from ICRP Publication 103 Table A4.2). Hence a doubling in ratio is seen in this Table.

c Leukaemia is the only cancer where distance is consistently important, with larger differences estimated close to the site except for ST1 where doses are too low for this effect to be seen. This is likely to be due to the non-linearity of the leukaemia model discussed in Section 3.2.

9.2 Comparisons between ICRP and BEIR VII models

For this work, both the ICRP and BEIR VII models were used to calculate the risks of incidence of cancer. Generally, there is good consistency between the results calculated using both approaches. However, for the lung dose it is notable that BEIR VII estimated higher risks than those of ICRP and that the age-risk relationship seen in the ICRP and BEIR VII predictions is different. As with all calculations in the report, uncertainty of risk estimates is manifest in the comparison of the ICRP and BEIR VII predictions of incidence risk. It should be noted that both ICRP models and BEIR VII models have an age-at-exposure component: exp((e-30/10)), where e is the age-at-exposure but that there are differences in implementation. This term is set to 1 when age-at-exposure is greater than 30 years old in the BEIR VII models so this allows the risk to vary when age-at-exposure is less than 30 years old but then the risk is not dependent on age-at-exposure for ages greater than 30 years old. Given that the BEIR VII model deliberately does not allow the risk to vary for age-at-exposure greater than 30 years old, the risk for the younger age-at-exposure groups has to increase to make up the difference in overall risk based on data. The ICRP model does not have this condition attached, so that the predicted risk is less sensitive to the younger age group. Therefore, the differences with age-at-exposure seen particularly in the lung cancer incidence between the two models is to some extent artificial and largely a result of different approaches taken. Therefore wider conclusions cannot be made.

9.3 Impact of amount of locally produced milk consumed

TAG-45 (ONR, 2019) states that "the approach taken to assess the dose to a person off the site should be flexible and reflect the type of analysis that is being performed and its regulatory purpose. The location and habit data should be chosen to be conservative for analysis in support of DBA, and best estimate for a Probabilistic Safety Assessment or Severe Accident Assessment." The often-used assumption of 10% locally produced food being consumed can be traced back to NRPB (1994) where it was a judgement value chosen to eliminate extreme conservatism that would have been introduced in a specific dose calculation had it been assumed all food consumed was at the highest intake rate. However, it was not based on any specific site survey. Given the importance of the ingestion pathway and in particular milk in the UK diet, the impact on dose of the fraction of locally produced food consumed was carried out. As detailed in Section 2.7, two sets of calculations were undertaken for ST1 at the request of ONR assuming that either 10% or 50% of milk consumed was locally produced, with 25% of all other foods being locally produced. Due to the magnitude of the assumed release and the presence of ^{131}I in this source term, food restrictions were not calculated to be required with the ingestion of ^{131}I being the most important pathway for doses and risk to the thyroid. Given that younger age groups consume a higher proportion of milk in their diet than adults, the assumption about the fraction of milk which is locally produced has therefore a larger impact on their dose. Changing the assumption of local consumption of milk from 50% (the default used in this study) to 10% reduces the prediction of 1-year-old effective and thyroid doses by more than half and has a significant but smaller effect on predictions for the other age groups. Given that there are no assumed differences in intakes between the sexes, there is no difference in dose between the sexes and only limited differences in risks.

In terms of variation with age for the risk of thyroid cancer incidence, assuming 10% local milk consumption reduces the thyroid risk of the 1-year-old to 20-40%, the 10-year-old risk to 30-60%, the 35-year-old risk to 40-70% and the 60-year-old risk to 50-70% of the 50% locally produced milk results. Assuming 10% local milk consumption compared to 50% reduces the risk of incidence for other cancers by 0 to 60%. For the risk of fatality from thyroid cancer, assuming 10% local milk consumption compared to 50% reduces the risk of the 1-year-old to 25-35%, with smaller reductions to older age groups. For other cancer sites, the reduction is by 60-80% with little difference across the ages. For the risk of detriment, assuming 10% local milk consumption compared to 50% reduces the thyroid cancer detriment risk of the 1-year-old to 25-35%, with smaller reductions for older age groups. For other cancer sites, the reduction is by 20 to 40% with the age making little difference.

In summary, the fraction of consumption of milk which is locally produced does affect the doses and risks calculated to the different age groups. However, any differences are dwarfed by the variances in the risk models for the different ages so the impact is not considered further.

9.4 Deterministic Health Effects

The model for deterministic effect risk implemented in PACE is based on that described in NRPB (1996) which does not review the underlying evidence but instead relies on previous

large reviews by Mettler and Upton (1995) and UNSCEAR (1988). Regarding differences by age, UNSCEAR (2013) states:

> *For direct effects that occur after high (either acute or fractionated) doses (so-called deterministic health effects), the differences in outcome between exposure in childhood and in adulthood are complex and can be explained by the interaction of different tissues and mechanisms. These effects may be seen after radiation therapy or following high exposures in accidents. The difference between the radiation sensitivity of children and that of adults for deterministic effects in a specific organ is often not the same as the difference for cancer induction. There are some instances in which childhood exposure poses more risk than adulthood exposure (e.g. risk of cognitive defects, cataracts and thyroid nodules). There are other instances where the risk appears to be about the same (e.g. risk of neuroendocrine abnormalities), and there are a few instances where children's tissues are more resistant (e.g. lungs and ovaries);*

ICRP Publication 118 (ICRP, 2012) is the latest statement from ICRP about deterministic effects, or tissue reactions (their preferred terminology). It reviews many studies, some of which identified either sex or age dependency of risk. However, the dependencies were not considered to be consistent and the authors did not suggest any changes to the ICRP Publication 103 estimates of thresholds of tissue effects on the basis of sex or age.

Therefore, it is not possible at the present time to make any allowance for age or sex sensitivity in the calculation of risk of deterministic effects. The age-and sex-sensitivity seen in stochastic effects is not a reliable guide to deterministic effects. Furthermore, even if such sensitivity could be defined, deterministic effects are a threshold effect and for the most part the predicted risk is either, at or close to zero, or, at or close to one.

9.5 ICRP advice and developments

ICRP Publication 101 provides guidance on assessing the dose of the Representative Person for the Purpose of Radiation Protection of the Public. It recommends "the use of three age categories for estimating annual dose to the representative person for prospective assessments. For practical implementation of this recommendation, dose coefficients and habit data for a 1-year-old infant, a 10-year-old child, and an adult should be used to represent the three age categories."

Effective dose is used to represent the overall dose to an individual received from different types of radiation by the various organs/tissues of the body. ICRP Publication 103 defines effective dose in relation to a nominal value of stochastic detriment following low-level exposure of 5.5 10^{-2} Sv^{-1}, as an average over both sexes and all ages. Despite the acknowledged averaging, ICRP in Publication 147 recommends the use of nominal risk values in setting risk constraints for potential exposures (paragraph 86, ICRP Publication 147). However, in the conclusions (paragraph 117), ICRP recommends that situations which require best estimates of risk should be evaluated using best scientific data including organ doses and age, sex-, and population-specific risk, with consideration of uncertainties. ICRP is in the early stages of preparing for new recommendations and is planning to consult on considerations on the use of effective dose with Clement et al (2021) suggesting that an approach could be to specify detriment and relative detriment for males and females of different age groups with

effective doses and associated detriment being calculated separately for each group. It suggests that simplifications could be made at the end of the process by setting appropriately averaged dose criteria. However, it acknowledges that such an evolution would obviously have implications for the management of radiation risk and those implications would need to be identified and assessed.

ICRP Task Group 122 on Update of Detriment Calculation for Cancer will also consider potential improvements to the calculational methodology with regard to the development of the future ICRP General Recommendations. One of their objectives is to reconsider the populations and age groups for which the nominal risk coefficients are calculated in order to allow and facilitate a more detailed practical application of the coefficients (e.g. for both sexes separately and for children below 18 years of age).

Effective dose represents the overall (whole-body) dose received by a person for the purposes of radiological protection but it does not provide a measure that is specific to the characteristics of the exposed individual. There has been discussion about whether effective dose is well-suited to assess doses to medical patients given that it does not take account of sex and age-at-exposure of individual patients (Andersson et al, 2017; Brenner, 2008). Harrison et al (2023) studied the effective doses and risks from medical diagnostic x-ray examinations for male and female patients from childhood to old age. The paper estimates that for x-ray examinations, average lifetime risks of cancer incidence per Sv may be two or three times higher for exposures at age 0-9 years than at age 30-39 years. However, the paper concludes that taking into account these differences in the risk per Sv, and noting the substantial uncertainties associated with risk estimates, effective dose as currently formulated provides a reasonable basis for assessing the potential risks from medical diagnostic examinations. Harrison et al (2023) also states that there are practical reasons, including continuity, for keeping the current formulation of effective dose.

To conclude, ICRP has started its cycle of reviewing the System of Radiological Protection. As part of this review, they will be considering whether sex and age-at-exposure calculations of effective dose and detriment could and should be used, rather than the current use of simplified age- and sex-averaged tissue weighting factors. However, this review is likely to take up to a decade to conclude.

9.6 Uncertainties

There have been numerous papers examining the uncertainties in both dose and risk assessments of exposure to radiation to the public. Puncher et al (2017) looked at those relating to exposure to radioiodine. The paper stated that the uncertainties on risk estimates are largely determined by uncertainties on risk estimates rather than on biokinetic model parameters. It was noted that although direct proportionality of the excess thyroid cancer risk and dose observed from low to moderate acute dose to very small doses received at very low dose rates was assumed, the uncertainty in this assumption was considerable, but also largely unquantifiable.

Many parameters and steps are involved in the calculation of cancer lifetime risk and cancer detriment. The variation in the values adopted for these have effects on the calculation results in this study but also generally.

Calculation of lifetime risk also requires the use of demographic data for sex and age specific cancer baseline rates. The baseline rates used in this study are the same as those in ICRP Publication 103 and Publication 152, and are composite rates from Sweden, UK, and the SEER program of the US National Cancer Institute. The cancer baseline rates can change significantly over the years due to changes in lifestyle, advances in diagnostic methods and improvements in treatment. The variation in cancer baseline rates can therefore also cause uncertainty in the results of lifetime risk calculation.

In addition, the lifetime risk calculation uses radiation models which describe the relationship between the organ/tissue dose and cancer risk for specific cancer sites. Radiation-associated cancer risk models were mainly derived statistically from the Life Span Study (LSS) of Japanese Atomic Bomb Survivors, based on follow-up from 1958 to 1998. (Preston et al, 2007). These models work well for the moderate dose range but there will be greater uncertainty when they are applied to extremely low and also very high dose and doserate levels. The risk models are presented without the uncertainties associated with them. The LSS dataset used to define the ICRP103 and BEIR VII models contains less information about the risks from radiation to people exposed when young compared to that for people exposed while older (although this is now rapidly changing since even those members of the LSS cohort who were infants at the time of the explosions are now into their seventies and their cancer incidence and mortality rates are increasing rapidly). Therefore, the models currently do not fully reflect the health outcomes of those exposed when young at the time of the bombing. Therefore, the uncertainties are very difficult to quantify. Although no measures of uncertainty are presented with the models, it is highly likely that the uncertainly in the model predictions will be less accurate for those exposed young than for those exposed when older.

Lifetime risk of fatality for cancers can be calculated by using the mortality models and mortality rates provided in ICRP Publication 103 (ICRP, 2007). However, a mortality model for thyroid cancer is not available at the time of writing and so the lifetime risk of thyroid cancer fatality was calculated using the lifetime risk of thyroid cancer incidence multiplied by a lethality fraction for thyroid cancer which is provided in ICRP Publication 103. However, this lethality fraction is age-averaged and it may introduce uncertainty in the calculation of the lifetime risk of thyroid cancer fatality for a specific age.

9.7 Risks to the thyroid

Throughout this study, the thyroid is significant because of the large age and sex-dependency in cancer incidence, fatality and detriment exhibited. As noted, due to lack of thyroid cancer mortality data, ICRP do not have a specific model for thyroid fatality risk so a significant amount of uncertainty in the detriment and fatality calculations is introduced by applying an age and sex independent lethality fraction.

The risk of thyroid cancer has long been recognised as a specific hazard given the observed child thyroid cancers seen following the Chornobyl accident (UNSCEAR, 2008 – Annex D) (UNSCEAR, 2008). Most atmospheric accident scenarios for operating Light Water Reactors estimate that a large amount of radioiodine will be released in a volatile vapour form. Fortunately if stable iodine tablets are taken by individuals before or at the start of an incident, then this will almost completely block uptake of the radioactive iodine (Kovari, 1994; WHO, 2017)(WHO, 1999).

Because of this, thyroid equivalent dose is usually considered explicitly alongside effective dose in emergency guidance, advice and regulations, e.g.

- The UK's Emergency Reference Levels (Nisbet, 2019) give a specific averted dose level for planning when stable iodine tablets should be administered. The dose is given in units of equivalent dose to thyroid.
- REPPIR 2019 requires that the operator, for the purpose of evaluating potential off-site radiation doses to members of the public, evaluates both effective dose and, where relevant, the equivalent dose to the thyroid.

There is no similar requirement in the SAPs. Despite the larger uncertainties in the risk model for younger age groups, the significantly increased risk estimates for the thyroid for them would indicate that a separate dose or risk to the thyroid should be considered to ensure adequate protection across all age groups. It should be noted that risk of fatality from thyroid cancer is relatively low so it would be more appropriate to consider the risk of incidence or detriment of thyroid cancer.

9.8 Implementation of findings for Risk Assessments Tools

This study has demonstrated that both age and sex should be accounted for in the process of risk assessment process. This section discusses how this could practically be achieved in existing risk assessment tools such as PACE. The process of risk assessment has broadly two steps: the calculation of organ doses and the calculation of risks from those doses.

To calculate risks this study used a modified version of the UKHSA PACE code for the first step of calculating organ doses, and then linked with a sophisticated external code for the second step of calculating risks. This was necessary as doses can occur over many years, and therefore a sequence of annual organ doses was needed that could be used by the risk calculation software to determine the overall lifetime attributable risk (LAR). Even though the modified approach was automated, it was a lengthy process and only feasible because of the limited number of locations and scenarios. Such an approach is unlikely to be a practical proposition for risk assessment codes that need to make predictions for several different candidate representative persons to find the most appropriate one and make assessments for multiple locations around a site and for varying weather conditions. The standard operational version of PACE and, to the best of the author's knowledge, all comparable codes, proceed by calculating a set of lifetime organ doses which are converted to risk by a set of nominal risk factors. Going forward this is likely to remain being the approach. Nevertheless, it is recommended that the process is expanded. For the step of calculating organ doses, and to correspond to the definition of the representative person by ICRP (ICRP Publication 101) (see Section 9.5), assessors should incorporate age by setting appropriate behaviours and utilising appropriate age-specific dose coefficients. However, assessors should go further than ICRP Publication 101 requirements, and consider sex-specific organs. In most cases the differences in dose coefficients for organs between males and females are slight. However, for some organs, notably the breast which was identified as a significant tissue in three of the four scenarios in this study, differences are significant. The use of these sex-specific dose coefficients provides a greater level of information and is relatively easy to incorporate into existing tools.

Regarding the second step, for some release categories such as one dominated by iodine (for example ST4, see Figure A34) most of the dose can be expected to be delivered soon after the release. Therefore accounting for aging may be an unnecessary refinement. In this case the calculation of risk as a lifetime dose multiplied by a nominal risk factor would be acceptable provided the risk factor was appropriate for the age-range when the majority of the dose is received. For scenarios where dose is delivered more evenly over a lifetime (for example ST3, see Figure A25) the use of a risk factor averaged over a broader age range would be appropriate.

Therefore, the most suitable data to use will be the risk factors provided in ICRP Publication 147. These are defined by sex, and given in broad age ranges (0-9 years, 10-19 years etc), with the current large uncertainties recognised in the lowest ages. It is unlikely that any more narrowly defined bands will be forthcoming from ICRP or BEIR until after the next iteration of the Life Span Study of the Atomic Bomb Survivors (see Section 9.6).

Table 11 compares the use of ICRP Publication 147 0-9-year female risk categories, with the modelled age- and sex-specific risks generated for this report. Almost all results are within a factor of 4. Pragmatically these results are likely to be the best that can be achieved in an operational rather than research assessment tool and a considerable improvement on Table 10 ie the ratios are much lower.

Table 11 Ratios of 1-year-old female lifetime attributable risk endpoints calculated by applying ICRP Publication 147 risk factors for a female 0-9 years at time of exposure to those based on tissue-specific risk factors and lifetime 1-year-old organ doses for the considered scenarios

		ST1 near	ST1 far	ST2 near	ST2 far	ST3 near	ST3 far	ST4 near	ST4 far
Incidence	Thyroid	2	2	1	2	2	2	1	2
	Lung	2	2	1	2	2	2	2	2
	Breast	2	2	2	2	2	2	2	2
	Bone marrow (Leukaemia)	1	1	8[b]	1	7[b]	1	3[b]	1
	Remainder	2	2	2	2	2	2	2	2
Fatality[a]		3	3	1	4	2	4	3	4

a ICRP Publication 147 gives only cancer incidence risk factors. Therefore, cancer fatality is calculated by applying lethality fractions. The fatality value also includes cancers in tissues not listed in the table.

b At high doses, the non-linear nature of the leukaemia risk model is apparent. A single risk factor for leukaemia which is applicable to all levels of dose will be hard to emulate, regardless of whether they are averaged over broad or narrow age ranges.

10 Conclusions

This study found that assessments of off-site risks to the public for accidents at nuclear facilities based on age and sex averaged doses and risk factors cannot implicitly be assumed to be conservative as the dose varies for the different age groups and the difference in risk per unit dose depending on age and sex varies.

For the source terms and populations considered, a higher risk of cancer fatality, cancer detriment and cancer incidence can generally be seen in females and for those exposed as children compared to the population weighted risk averaged over age and sex (based on ICRP Publication 103 organ specific risk factors). For example:

- The *risk of fatality* for the 1-year-old female at near and far locations from the four accidents considered in the study was in the range of approximately 1 to 20 times higher. The ratio of 1 was obtained for a position adjacent to the most severe accident considered in the study, ST2, and resulted from high doses where calculated risks are 100% for all ages and sexes. The upper number was obtained for both near and far locations for the Design Basis accident (ST1) where doses are lower.
- The *detriment* for the 1-year-old female at near and far locations from the four accidents considered in the study was approximately in the range of 1 to 50 times higher. The ratio of 1 was obtained for a position adjacent to the most severe accident considered in the study, ST2, and resulted from high doses where calculated risks are 100% for all ages and sexes. The upper number was obtained for the far location for the Design Basis accident (ST1), where doses are lower. The broader range results from contributions made from non-fatal cancers to the detriment risk quantity.
- Similar comparisons for *incidence* of thyroid cancer and leukaemia yielded approximate ranges of 1 to 100 times and 1 to 10 times higher respectively. For both ranges, the ratio of 1 was obtained for a position adjacent to the most severe accident considered in the study, ST2, and resulted from high doses where calculated risks are 100% for all ages and sexes. The upper number for incidence of thyroid cancer arises from the far location for the Design Basis accident (ST1) where doses are lower. The upper number for the incidence of leukaemia arises at the near location for the large severe accident ST2.

It is noted that the risks of cancer fatality, detriment and incidence calculated for locations near to the large severe accident ST2 were not representative of the rest of study. This is because the risks are due to very high doses where the predicted risk of occurrence is 100% for all ages and sexes. For the majority of other source terms and receptor locations, a degree of enhanced risk is evident for the younger ages and females compared to the general population. For the Design Basis Accident ST1, where lower doses are estimated to be received of the public off-site but with a higher likelihood of occurring, the health impacts are low and no increased rates in cancer over the background level is likely to be detectable in the population.

An important observation is that given the smaller datasets for people exposed when young, there are larger uncertainties in the risk models for those groups, though experts expect younger age groups to be more radio-sensitive. For the 10-year-old group, where there is more information available compared to 1-year-olds and therefore less uncertainty, the risk of fatality for the 10-year-old female was in the range 1 to 7 times higher compared to the population weighted risk, while the detriment was in the range of 1 to 8 times higher. This demonstrates that the use of both age and sex-averaged risk estimates can lead to an underestimation of risk for groups such as younger females when using organ-specific risk factors.

The importance of the age of the individual at time of exposure and their sex will depend on the radionuclides released during the accident, the subsequent pathways of exposure and the protective actions implemented. In this study, three of the source terms related to reactor accidents and one relates to a waste accident based on the Mayak accident source term but adjusted to give a generic fuel-cycle source term. The impact of the age-at-exposure and sex can be seen for the thyroid in all source terms. However, it is most clearly demonstrated in

the example of an operating reactor Design-Basis Accident source term, ST1-DBA, where no food restrictions are calculated to be required and therefore the ingestion of ^{131}I pathway dominates the dose to the public. The dependence of sex and age at exposure of the thyroid can also been seen to a lesser extent in ST2-acute and ST4-Chornobyl source terms which are both much more severe accident source terms so protective actions such as administration of stable iodine and food restrictions would be implemented. For ST3-fuel, the different radionuclide mix means that the lung becomes the organ of most interest and although there is some age and sex dependence, it is not as marked as for the thyroid.

In the context of risk assessments carried out for nuclear safety cases, this means that the use of both age and sex-averaged risk estimates for source terms similar to those considered in this study would likely lead to an underestimation of risk to the young and females. However, it should be noted that given the smaller datasets for people exposed when young, there are larger uncertainties in the risk models for the younger age groups than across the whole population. These uncertainties are very substantial and will add to the uncertainties in other aspects of the radiological modelling for off-site consequences.

However, the next international periodic mortality and cancer incidence analyses of the Life Span Study cohort data will provide much improved information on the risk models for the younger age groups. ICRP are working to update their risk estimates as new population health statistics data, dosimetry information and radiation risk models are available. The differences in risk due to age-at-exposure and sex are already acknowledged by ICRP in Publication 147 (2021). ICRP are currently reviewing their system of radiological protection. This includes the setting up of a Task Group on Update of Detriment Calculation for Cancer which will also consider potential improvements to the calculational methodology. As part of this review ICRP will be considering whether sex and age-at-exposure calculations of effective dose and detriment can be used through a newly defined quantity, rather than the current use of simplified age- and sex-averaged tissue weighting factors. However, this review is likely to take up to a decade to conclude.

In the meantime, safety cases for UK nuclear licensed sites should estimate age-specific doses to identify the most exposed individuals. ONR should also consider whether, given the markedly greater radio-sensitivity of the thyroid by age-at-exposure and sex, a separate specific dose or risk target for the thyroid should be developed where the accident scenarios involve the release of radioiodine. For cancers which vary significantly in risk according to age or sex, using age- and sex-specific risk factors should be considered.

11 References

Andersson M, Eckerman K and Mattsson S (2017). Lifetime attributable risk as an alternative to effective dose to describe the risk of cancer for patients in diagnostic and therapeutic nuclear medicine. *Physics in Medicine and Biology* **62**, 9177-9188.

Bexon AP, Haywood SM, Mortimer K and Bedwell P (2019). *REPPIR 2019 consequence assessment methodology.* Chilton (UK), PHE-CRCE-050.

Brenner DJ (2008). Effective dose: a flawed concept that could and should be replaced. *British Journal of Radiology* **81**, 521-523.

Charnock TW, Bexon AP, Sherwood J, Higgins NA, Field S, Smith J and Brown IK (2020). *The Probabilistic Accident Consequence Evaluation (PACE) Software Methodology for version 3.3.2.* Public Health England, CRCE-RAD-006-2020.

Clement C, Rühm W, Harrison J, Applegate K, Cool D, Larsson CM, Cousins C, Lochard J, Bouffler S, Cho K, Kai M, Laurier D, Liu S and Romanov S (2021). Keeping the ICRP recommendations fit for purpose. *Journal of Radiological Protection* **41**(4), 1390-1409.

Environment Agency, Scottish Environment Protection Agency, Northern Ireland Environment Agency, Health Protection Agency and Food Standards Agency (2012). *Principles for the Assessment of Prospective Public Doses arising from Authorised Discharges of Radioactive Waste to the Environment.* Penrith (UK).

FRMAC (2015). *The Federal Manual for Assessing Environmental Data During a Radiological Emergency.* Sandia National Laboratories.

Harrison JD, Haylock RGE, Jansen JTM and Zhang W (2023). Effective doses and risks from medical diagnostic x-ray examinations for male and female patients from childhood to old age. *Journal of Radiological Protection* **43**.

IAEA (2006). *Environmental Consequences of the Chernobyl Accident and their Remediation: Twenty Years of Experience. Report of the Chernobyl Forum Expert Group 'Environment'.* International Atomic Energy Agency, Vienna (Austria), STI/PUB/1239.

ICRP (2006). Assessing dose of the representative person for the purpose of radiation protection of the public. ICRP Publication 101a. *Annals of the ICRP* **36**(3), 1-62.

ICRP (2007). The 2007 Recommendations of the International Commission on Radiological Protection. ICRP Publication 103. *Annals of the ICRP* **37**(2-4), 9-34.

ICRP (2012). ICRP Statement on Tissue Reactions and Early and Late Effects of Radiation in Normal Tissues and Organs — Threshold Doses for Tissue Reactions in a Radiation Protection Context. ICRP Publication 118. *Annals of the ICRP* **41**(1-2), 1-322.

ICRP (2020). Dose Coefficients for External Exposures to Environmental Sources. ICRP Publication 144. *Annals of the ICRP* **49**(2), 11-145.

ICRP (2021). Use of Dose Quantities in Radiological Protection. ICRP Publication 147. *Annals of the ICRP* **50**(1), 9-82.

ICRP (2022). Radiation Detriment Calculation Methodology. ICRP Publication 152. *Annals of the ICRP* **51**(3), 9-103.

Jones AL and Sherwood JC (2008). *Delay Times between Harvesting or Collection of Food Products and Consumption for Use in Radiological Assessments.* Health Protection Agency, Chilton (UK), HPA-RPD-043.

Kovari M (1994). Effect of delay time on effectiveness of stable iodine prophylaxis after intake of radioiodine. *Journal of Radiological Protection* **14**(2), 131-136.

Mettler J, F. A. and Upton AC (1995). *Medical effects of ionizing radiation, 2nd Edition*. United States.

National Research Council (2006). *Health Risks from Exposure to Low Levels of Ionizing Radiation, BEIR VII Phase 2.* T. N. A. Press, Washington, DC.

NEA (2002). *Chernobyl: Assessment and Radiological and Health Impacts, 2002 Update of Chernobyl Ten Years On.* OECD.

Nisbet AF (2019). *Public Health Protection in Radiation Emergencies.* Public Health England, Chilton (UK), PHE-CRCE-049.

NRPB (1994). Guidance on Restrictions on Food and Water Following a Radiological Accident. *Documents of the NRPB* **1**.

NRPB (1996). Risk of Deterministic Effects of Ionising Radiation. *Documents of the NRPB* **7**(3).

ONR (2014). *Safety Assessment Principles for Nuclear Facilities.* Office for Nuclear Regulation, Bootle, Merseyside.

ONR (2019). *Radiological Analysis for Fault Conditions.* Office for Nuclear Regulation, NS-TAST-GD-045 Revision 6.

Preston DL, Ron E, Tokuoka S, Funamoto S, Nishi N, Soda M, Mabuchi K and Kodama K (2007). Solid Cancer Incidence in Atomic Bomb Survivors: 1958–1998. *Radiation Research* **168**(1), 1-64.

Puncher M, Zhang W, Harrison JD and Wakeford R (2017). Assessing the reliability of dose coefficients for exposure to radioiodine by members of the public, accounting for dosimetric and risk model uncertainties. *Journal of Radiological Protection* **37**, 506-526.

Simmonds JR, Robinson CA, Phipps AW, Muirhead CR and Fry FA (1995). *Risks of Leukaemia and Other Cancers in Seascale from All Sources of Ionising Radiation Exposure.* NRPB, Chilton (UK), NRPB-R276.

Smith KR and Jones AL (2003). *Generalised Habit Data for Radiological Assessments.* National Radiological Protection Board, Chilton (UK), NRPB-W41.

SSK (2014). *Dose- and dose-rate-effectiveness factor (DDREF). Recommendation by the German Commission on Radiological Protection with Scientific Grounds.* Strahlenschutzkommission.

Thomas D, Darby S, Fagnani F, Hubert P, Vaeth M and Weiss K (1992). Definition and Estimation of Lifetime Detriment From Radiation Exposures: Principles and Methods. *Health Physics* **63**(3), 259-272.

UNSCEAR (1988). *Sources and Effects of Ionizing Radiation. United Nations Scientific Committee on the Effects of Atomic Radiation. 1988 Report to the General Assembly, with annexes.* United Nations, New York.

UNSCEAR (2000). Annex J. Exposures and effects of the Chernobyl accident. *Sources and Effects of Ionizing Radiation. United Nations Scientific Committee on the Effects of Atomic Radiation UNSCEAR 2000 Report of the General Assembly*. New York, United Nations.

UNSCEAR (2008). *Effects of Ionizing Radiation. UNSCEAR 2006 Report to the General Assembly, with Scientific Annexes.* U. Nations, New York.

UNSCEAR (2010). *Sources and Effects of Ionizing Radiation. UNSCEAR 2008 Report to the General Assembly, with scientific annexes.* New York.

UNSCEAR (2013). *Sources, Effects and Risks of ionizing radiation - UNSCEAR 2013 Report - Volume II - Scientific Annex B: Effects of radiation exposure of children.*

Walsh L, Zhang W, Shore RE, Auvinen A, Laurier D, Wakeford R, Jacob P, Gent N, Anspaugh LR, Schüz J, Kesminiene A, van Deventer E, Tritscher A and del Rosario Pérez M (2014). A Framework for Estimating Radiation-Related Cancer Risks in Japan from the 2011 Fukushima Nuclear Accident. *Radiation Research* **182**, 556-572.

WHO (1999). *Guidelines for Iodine Prophylaxis following Nuclear Accidents Update 1999.* World Health Organization, Geneva.

WHO (2013). *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, Geneva (Switzerland).

WHO (2017). Iodine Thyroid Blocking: Guidelines for Use in Planning for and Responding to Radiological and Nuclear Emergencies. Geneva, World Health Organization.

Zhang W, Laurier D, Clèro E, Hamada N, Preston D, Vaillant L and Ban N (2020). Sensitivity analysis of parameters and methodological choices used in calculation of radiation detriment for solid cancer. *International Journal of Radiation Biology* **96**, 596-605.

Appendix A Detailed dose profiles

Calculations of effective and organ doses is the first step in the calculation of risks of incidence and fatalities. This appendix gives a more detailed description of the dose profiles for different age groups in each of the examples. When interpreting the graphs and the results the following points should be noted:

1. Scales on the line graphs are not linear. The y axis is usually logarithmic, whereas the x axis starts yearly and then continues at intervals of several years.
2. Apart from breast and ovary organs, doses were calculated and are given by age only. While there maybe systematic differences between males and female doses due to example body size, organ size, lung capacity and consumption habits, these are expected to be small and have not been incorporated, so male and female doses are the same for corresponding ages. The exception are doses to breast and ovary which are for females only.
3. Unless explicitly stated graphs assume that 50% of milk consumption is local.
4. On the line graphs, the delivery of the inhalation dose often appears to cease after 1 or a few years, whereas one would expect that, for long lived radionuclides, the dose delivery to continue for as long as the radionuclides are in the body. This is an artefact caused by the limited precision of the available dose coefficients which are all given from time zero. In some cases, the dose delivery in subsequent years is so small that it does not change the given value of subsequent dose coefficients. In extreme cases dose delivery may appear to cease for a period and then recommence. While this looks strange in the graphs it will not affect the risk calculations significantly as it is caused by relatively very small amounts of dose. It should be noted that sometimes the rate of dose delivery can increase slightly in later years because of ingrowth and changes in organ size and position. These artefacts can sometimes be seen with the ingestion dose but is less noticeable because of the ongoing intake of radionuclides from contaminated food.

A1 ST1 - DBA

Table A1 ST1-DBA – total lifetime effective and thyroid doses by age group

	1yr	10yr	35yr	60yr
Effective mSv				
dry-near	7.4E-02	2.9E-02	1.9E-02	1.8E-02
dry-10%milk-near	3.0E-02	1.9E-02	1.3E-02	1.3E-02
wet-near-	1.0E-01	3.5E-02	2.3E-02	2.2E-02
wet-10%milk-near	3.3E-02	1.7E-02	1.3E-02	1.3E-02
dry-far	4.7E-03	1.7E-03	1.1E-03	1.1E-03
dry-10%milk-far	1.7E-03	1.0E-03	7.3E-04	7.3E-04
Wet-far	8.1E-03	2.4E-03	1.6E-03	1.5E-03
wet-10%milk-far	2.2E-03	9.2E-04	7.5E-04	7.4E-04
Thyroid mSv				
dry-near	1.3E+00	4.7E-01	2.5E-01	2.5E-01
dry-10%milk-near	5.0E-01	2.8E-01	1.7E-01	1.7E-01
wet-near	1.8E+00	5.2E-01	2.7E-01	2.7E-01
wet-10%milk-near	5.3E-01	2.3E-01	1.4E-01	1.4E-01
dry-far	8.5E-02	2.7E-02	1.4E-02	1.4E-02
dry-10%milk-far	2.9E-02	1.5E-02	8.8E-03	8.8E-03
wet-far	1.5E-01	3.6E-02	1.7E-02	1.7E-02
wet-10%milk-far	3.6E-02	1.1E-02	6.3E-03	6.3E-03

Table A1 gives the lifetime effective and thyroid doses, and Figure A1 and Figure A2 illustrate total doses of all organs. The largest total effective dose is 0.074 mSv and the largest total thyroid dose is 1.8 mSv. The doses to a 1-year-old are the largest in all examples. The figures demonstrate that changing the assumption of local consumption of milk from 50% (default) to 10% reduces the prediction of 1-year-old effective and thyroid doses by more than half and Table A1 has a significant but smaller effect on predictions for the other age groups. Figure A1 and Figure A2 shows that in all examples the thyroid dose is by far the largest.

Figure A1 and Figure A2 show a strong age dependency in the thyroid dose with the 1-year-old lifetime dose between 4-7 times higher than the 35-year-old. With an assumption of local consumption of milk of 10% this drops slightly to between 2-4 times higher.

Because of the dominance of thyroid, it is difficult to discern the effective dose or doses to other organs in Figure A1 and Figure A2. Therefore Figure A3 and Figure A4 show the same doses but omitting the thyroid. The corresponding doses to different organs are very similar and there appears to be no age dependency. The assumption of local consumption of milk of 10% gives only a slight reduction in predicted doses, except for the effective dose which, of course, includes a component of thyroid dose.

Figure A5 gives the breakdown of thyroid dose by pathway and shows that, for the 1-year-old, ingestion is the dominant pathway. It is also a dominant pathway for the other age groups but less so. In three out of the four examples, inhalation is the second most significant pathway. However, in the far-wet example, the contribution to dose from inhalation is small and comparable with the dose from external deposition.

For comparison, Figure A6 give the breakdown of lung dose by pathway. Ingestion is still an important pathway, but in three of the four example external pathways dominate and the dose

is split roughly equally between external cloud and external deposition. In the wet-far example ingestion is dominant but about a third of the dose is from external deposition and very little from external cloud. In all examples inhalation is a minor contributor.

Figure A7 shows the predicted delivery over time of thyroid dose for all age groups. The patterns of dose delivery in each example are very similar, with most of the dose delivered in the first year for all age groups and locations and falling 2 to 3 orders of magnitude by the second year. The 1-year-old receives more dose than other age groups in the first year, while the adults receive slightly more than other age groups in years 2 to 5, with the annual doses converging for all age groups in subsequent years.

For comparison, Figure A8 shows the predicted delivery over time of the lung dose. The patterns of dose delivery in each example are very similar, with the most dose delivered in the first year and falling by about an order of magnitude in the second year. The delivery of lung dose appears more protracted than the thyroid dose.

Figure A9 and Figure A10 show the delivery of thyroid dose from different pathways to the 1-year-old and 35-year-old as a function of time. The profiles are similar for both two ages and the four examples. Ingestion of the radionuclides via food is an important pathway in the first year and external exposure from deposition on the ground is the least important except for the wet-far example. However, in subsequent years, external exposure from deposition on the ground is the most significant contributor to dose in all examples. Inhalation is much more important for the 35-year-old, but for both the 35-year-old and 1-year-old its contribution is insignificant after the first year.

Figure A11 gives the total lifetime thyroid dose contribution by radionuclide. It shows that ^{131}I makes by far the most dominant contribution to thyroid dose for all age groups particularly the 1-year-old. For comparison, Figure A12 show the total lifetime lung dose by radionuclide. ^{131}I is a minor contributor with ^{134}Cs and ^{137}Cs particularly significant and in three of the examples ^{133}Xe also being significant.

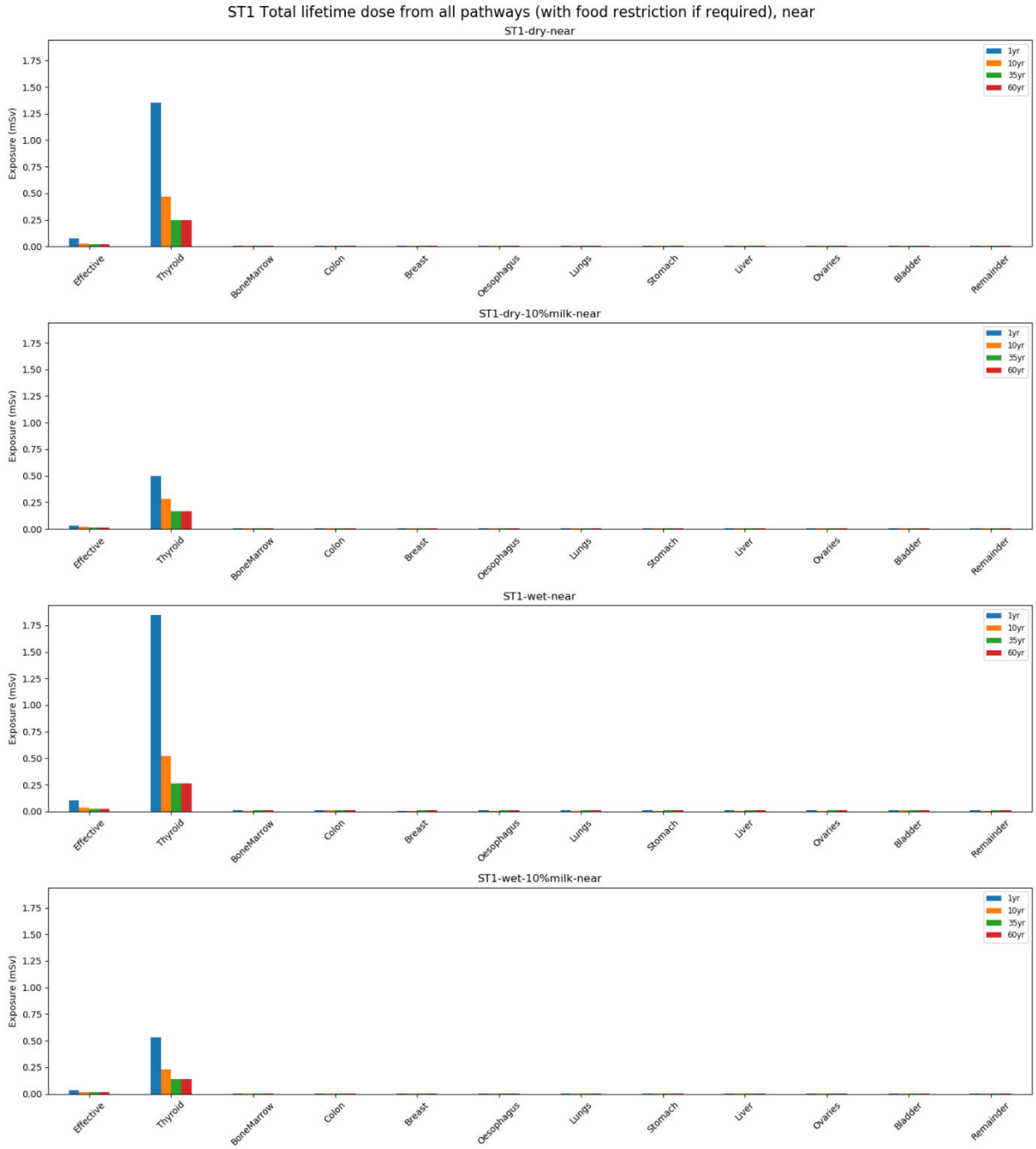


Figure A1 ST1-DBA-near – total lifetime effective doses

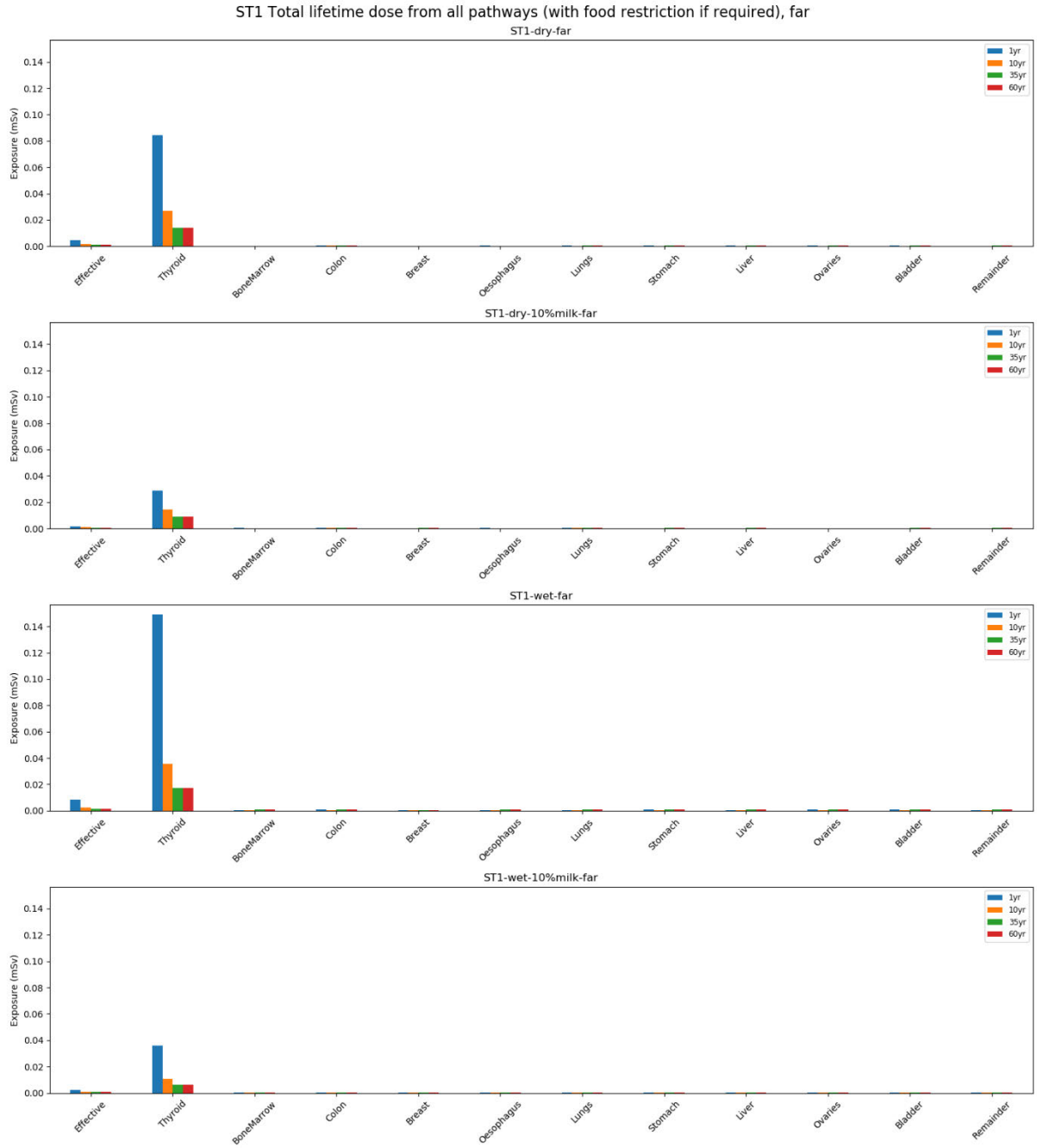


Figure A2 ST1-DBA-far – total lifetime effective doses

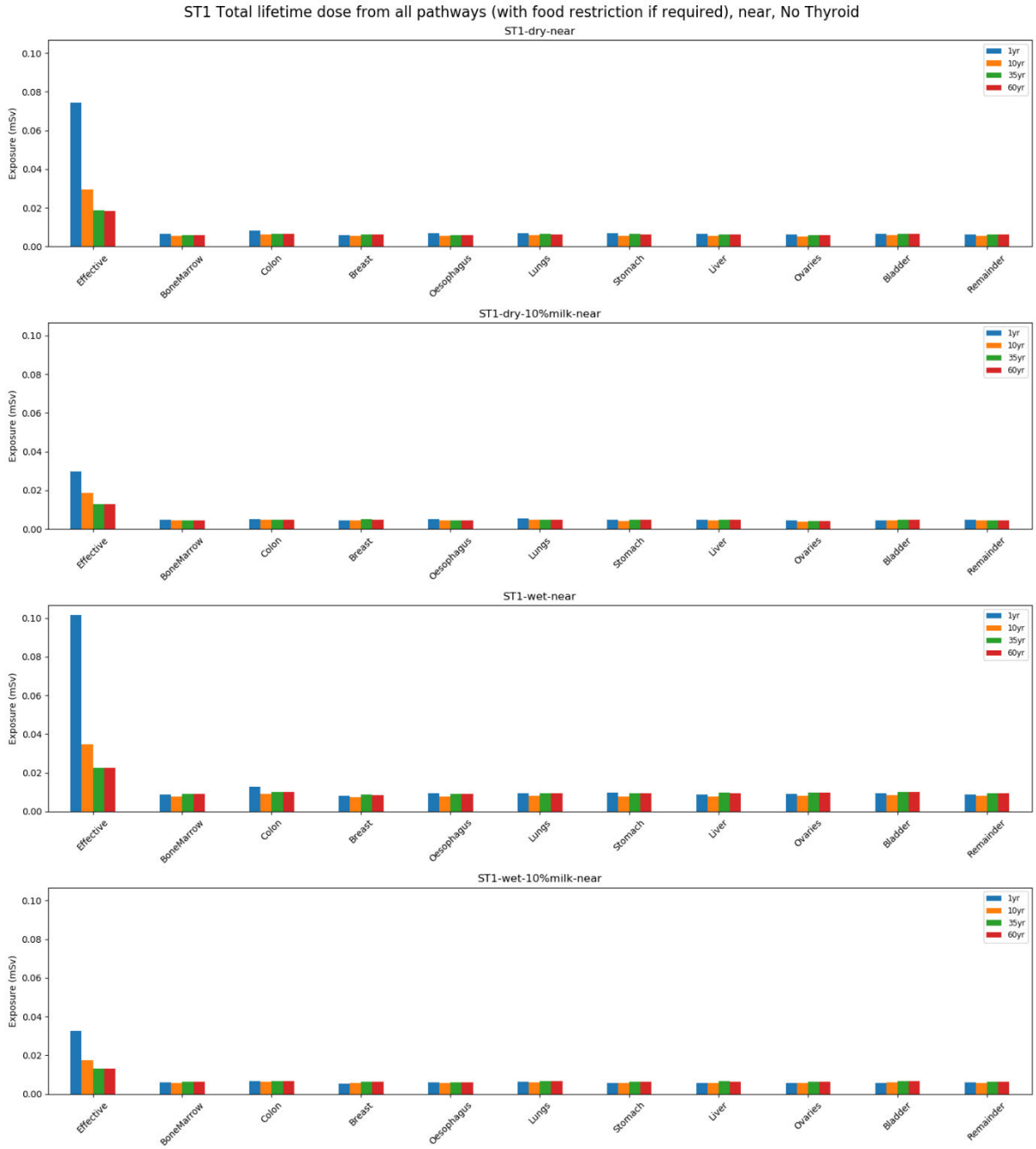


Figure A3 ST1-DBA-near – total lifetime effective doses without thyroid

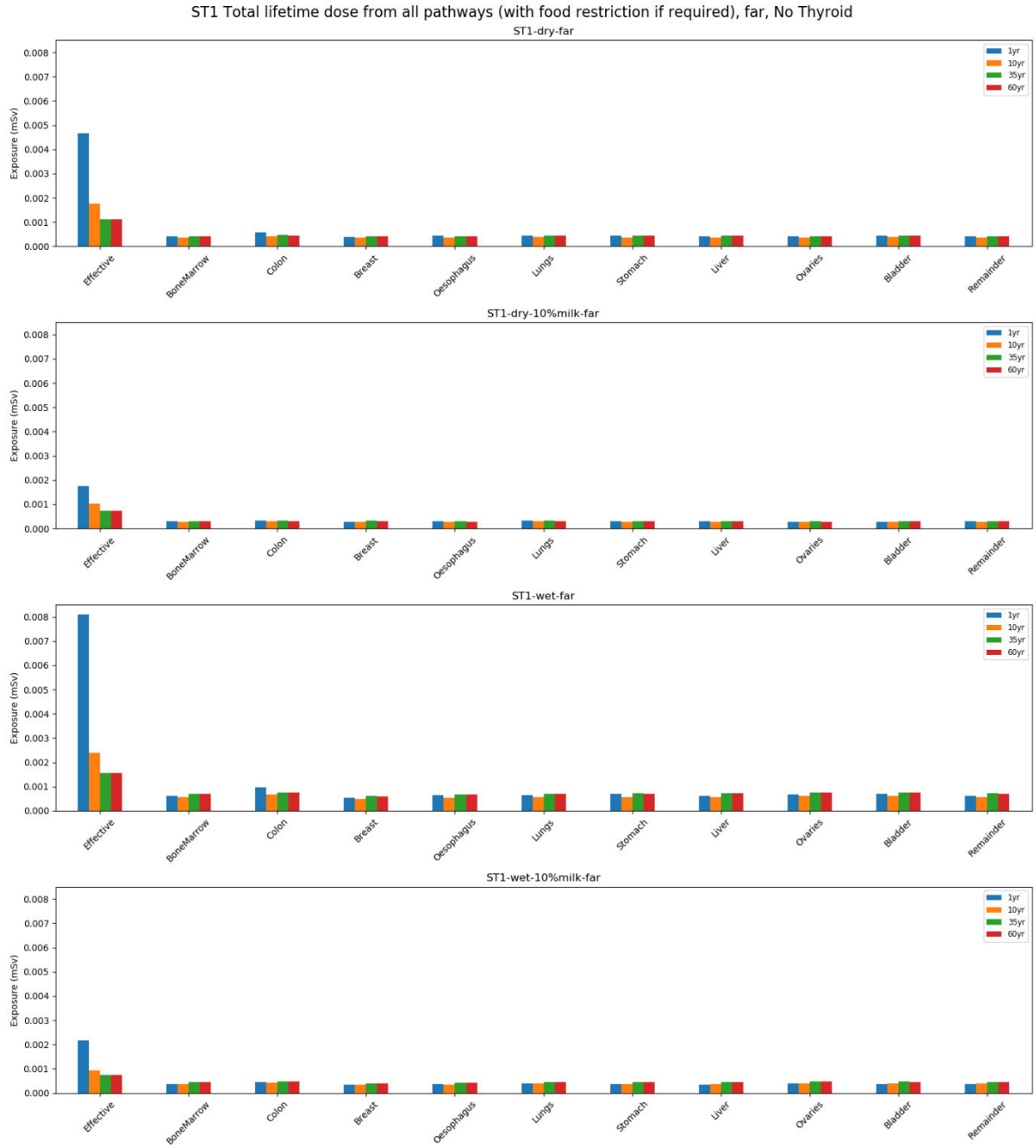


Figure A4 ST1-DBA-far – total lifetime effective doses without thyroid

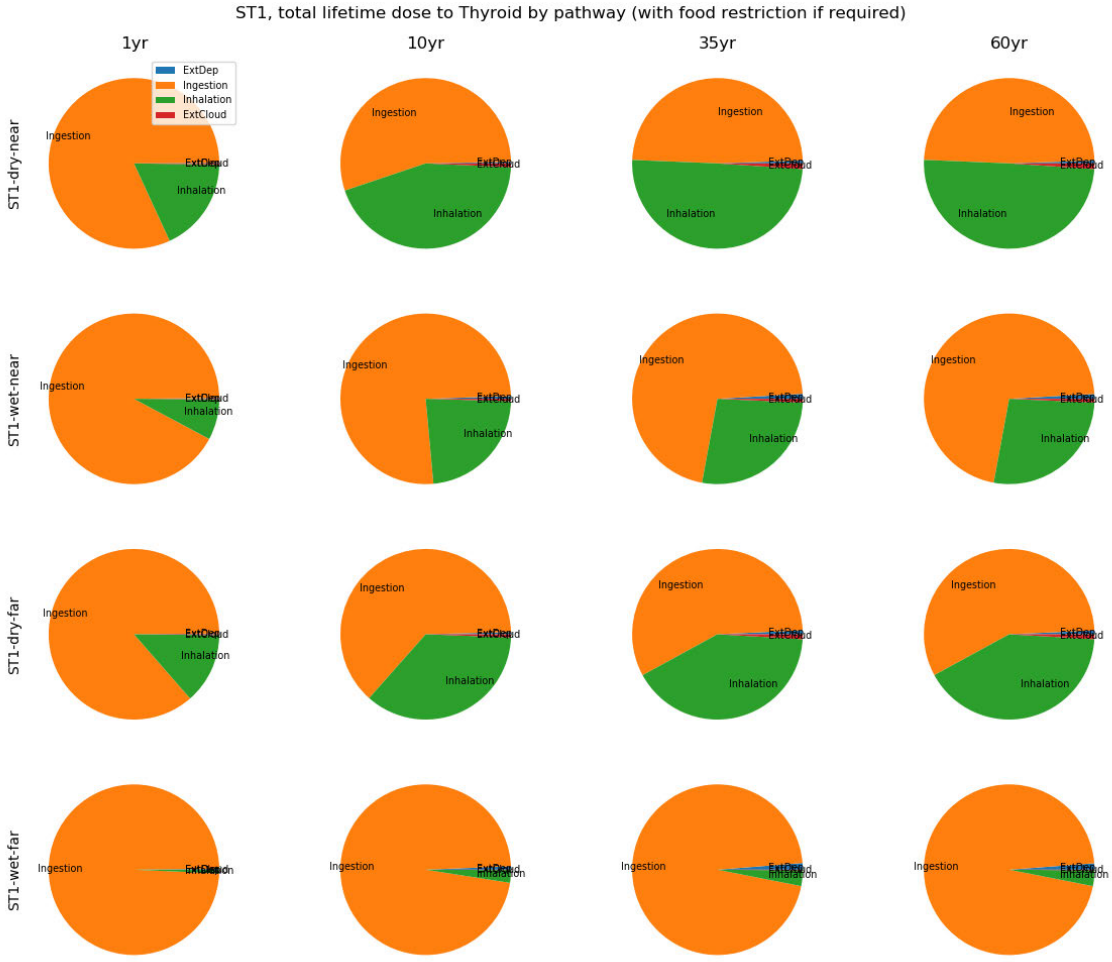


Figure A5 ST1-DBA – proportion of total lifetime thyroid dose by pathway

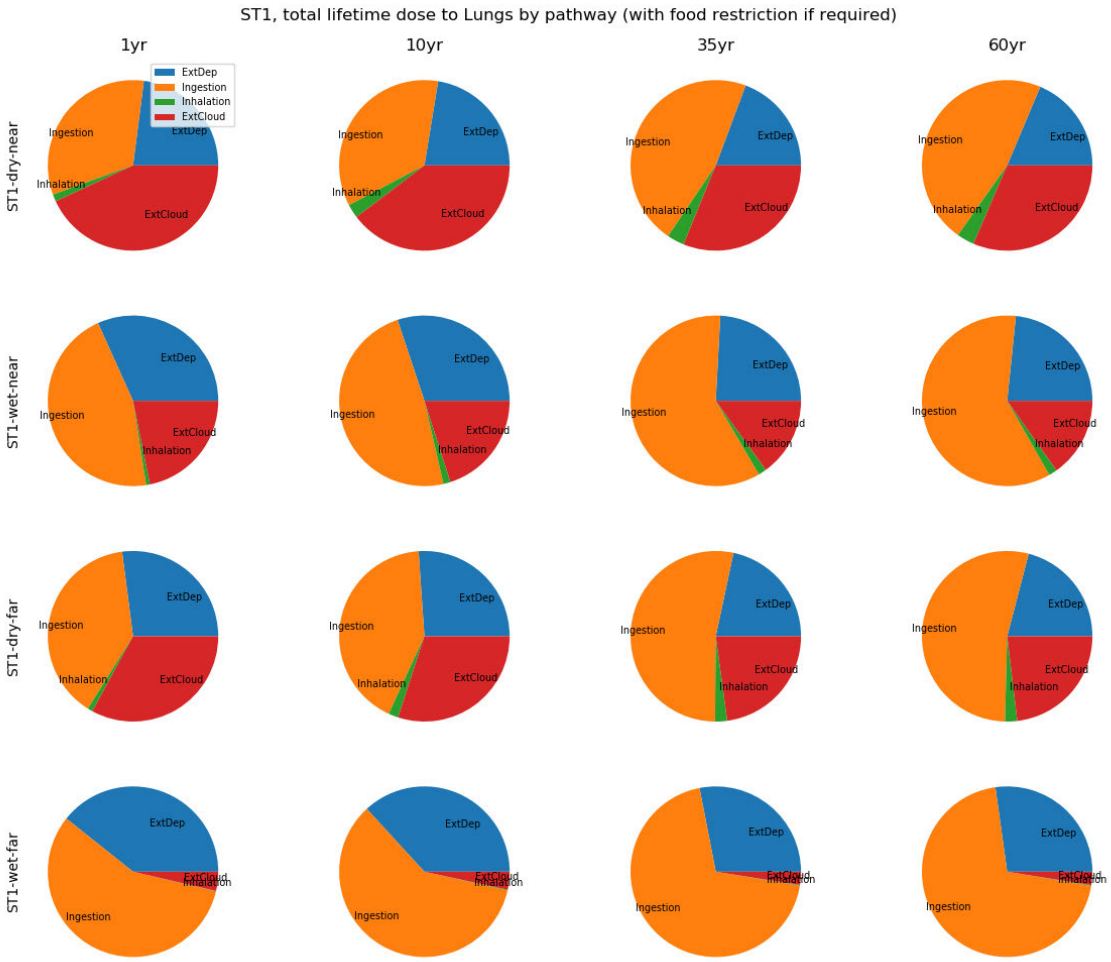


Figure A6 ST1-DBA – proportion of total lifetime lung dose by pathway

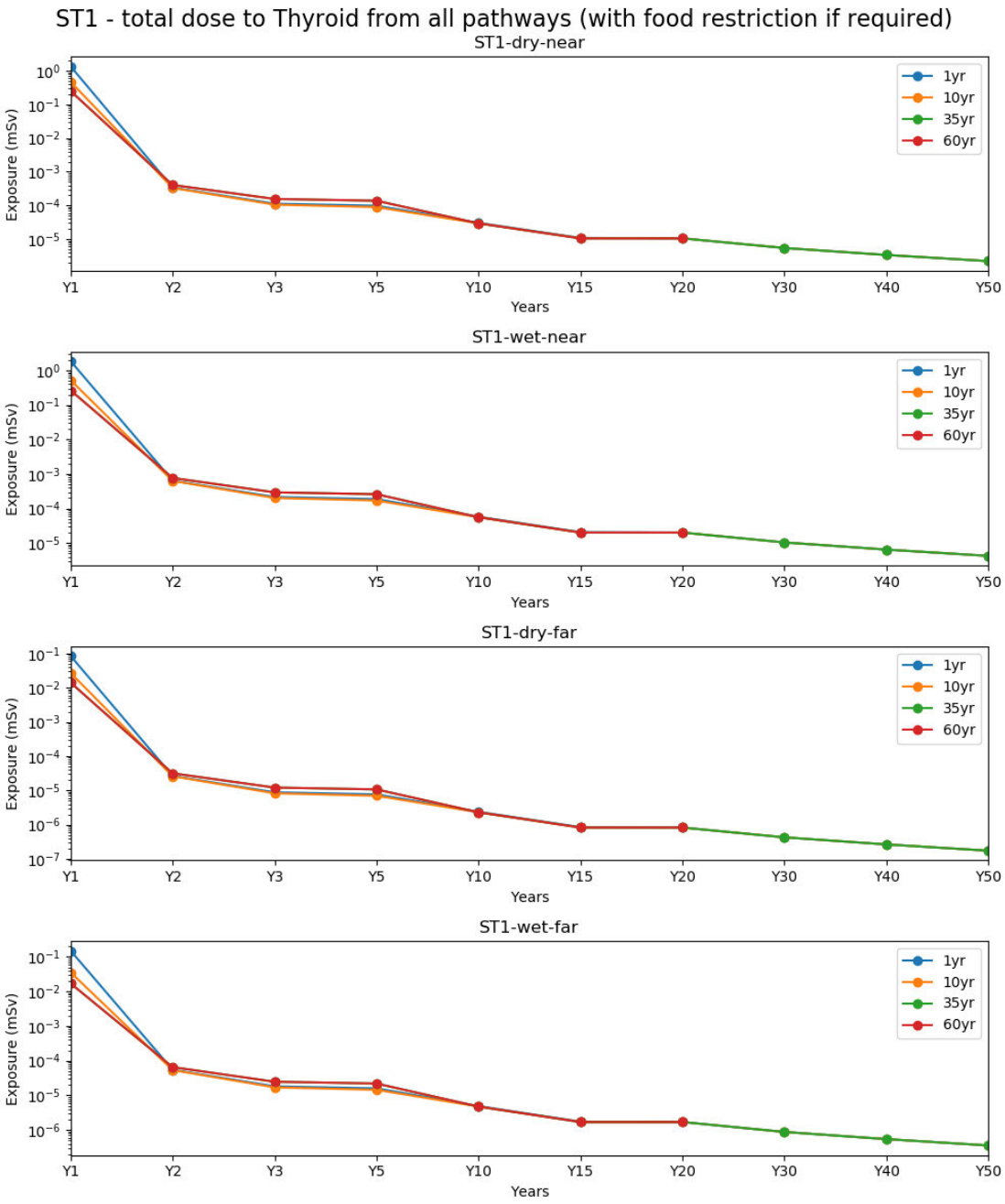


Figure A7 ST1-DBA – total thyroid dose by year for each age group

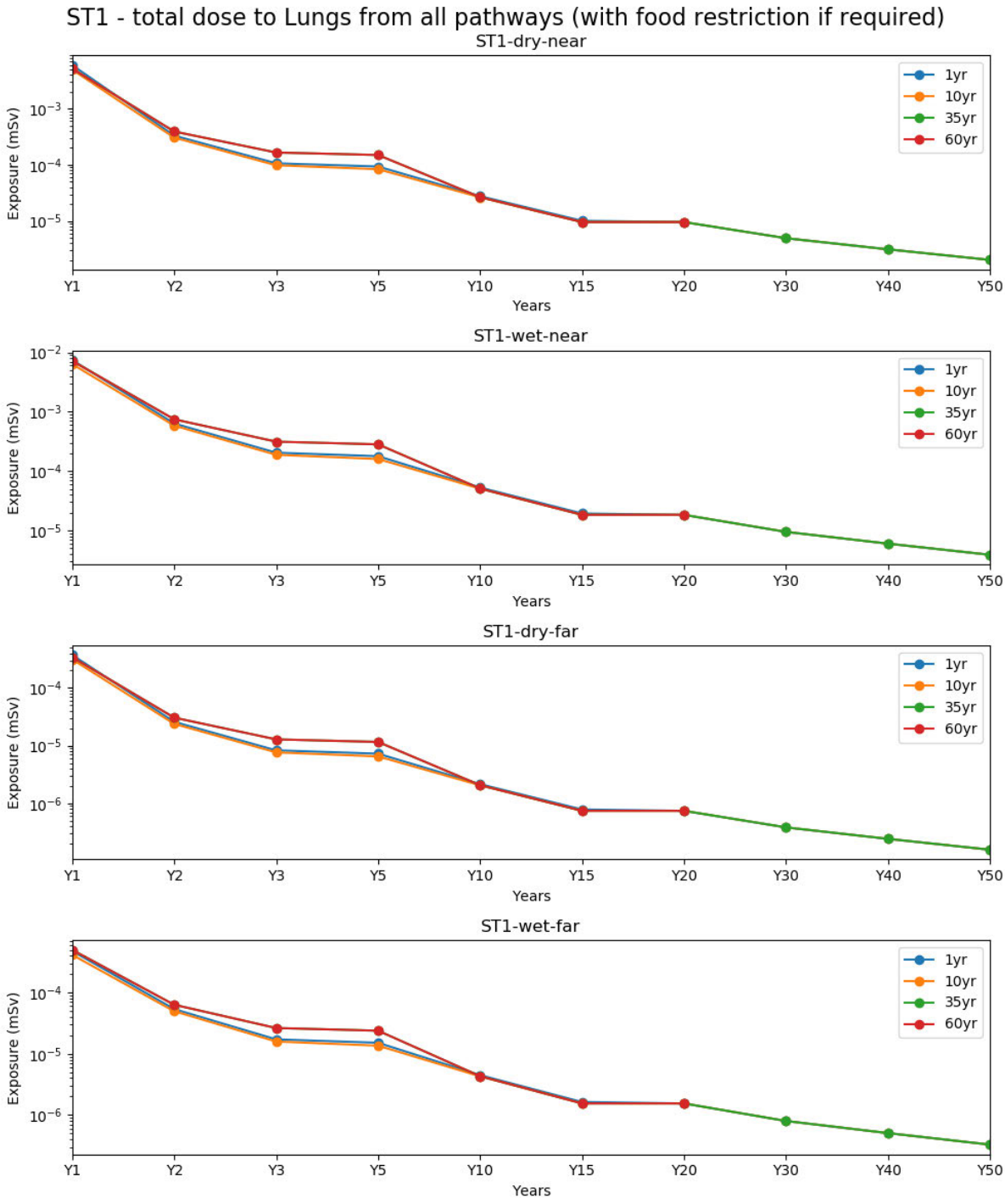


Figure A8 ST1-DBA – total lung dose by year for each age group

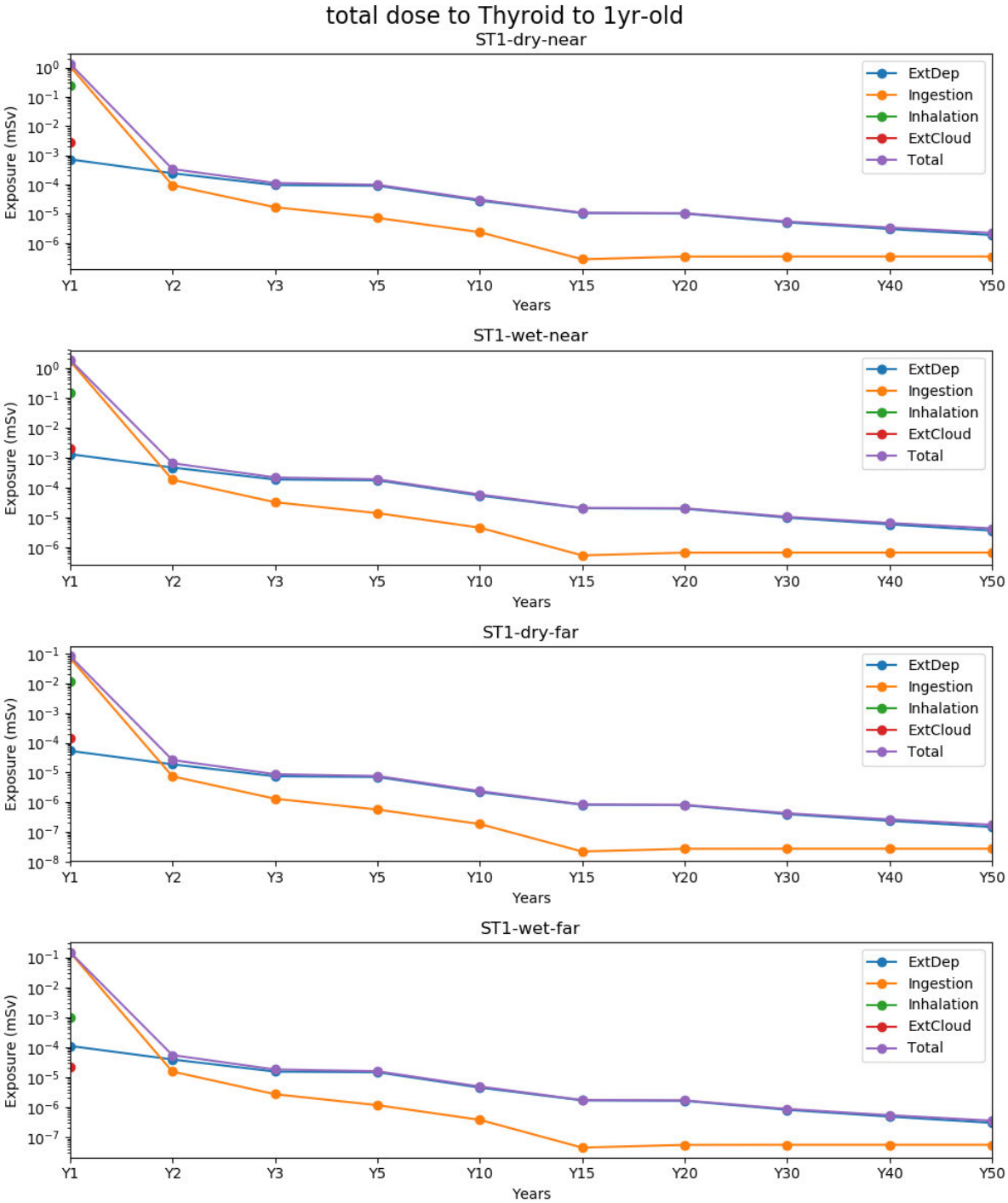


Figure A9 ST1-DBA – total lifetime thyroid dose to 1-year-old by pathway, by year of delivery

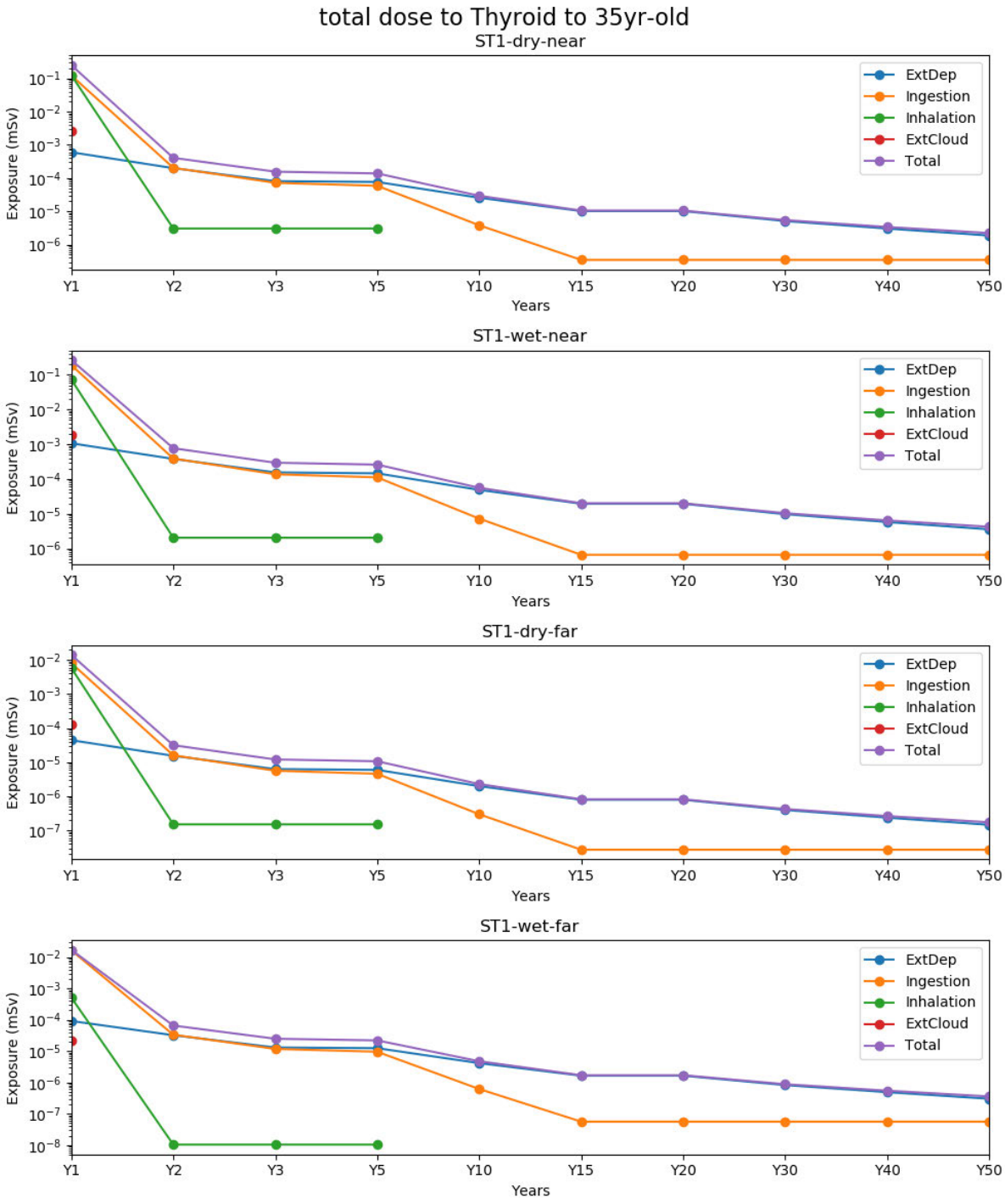


Figure A10 ST1-DBA – total lifetime thyroid dose to 35-year-old by pathway, by year of delivery

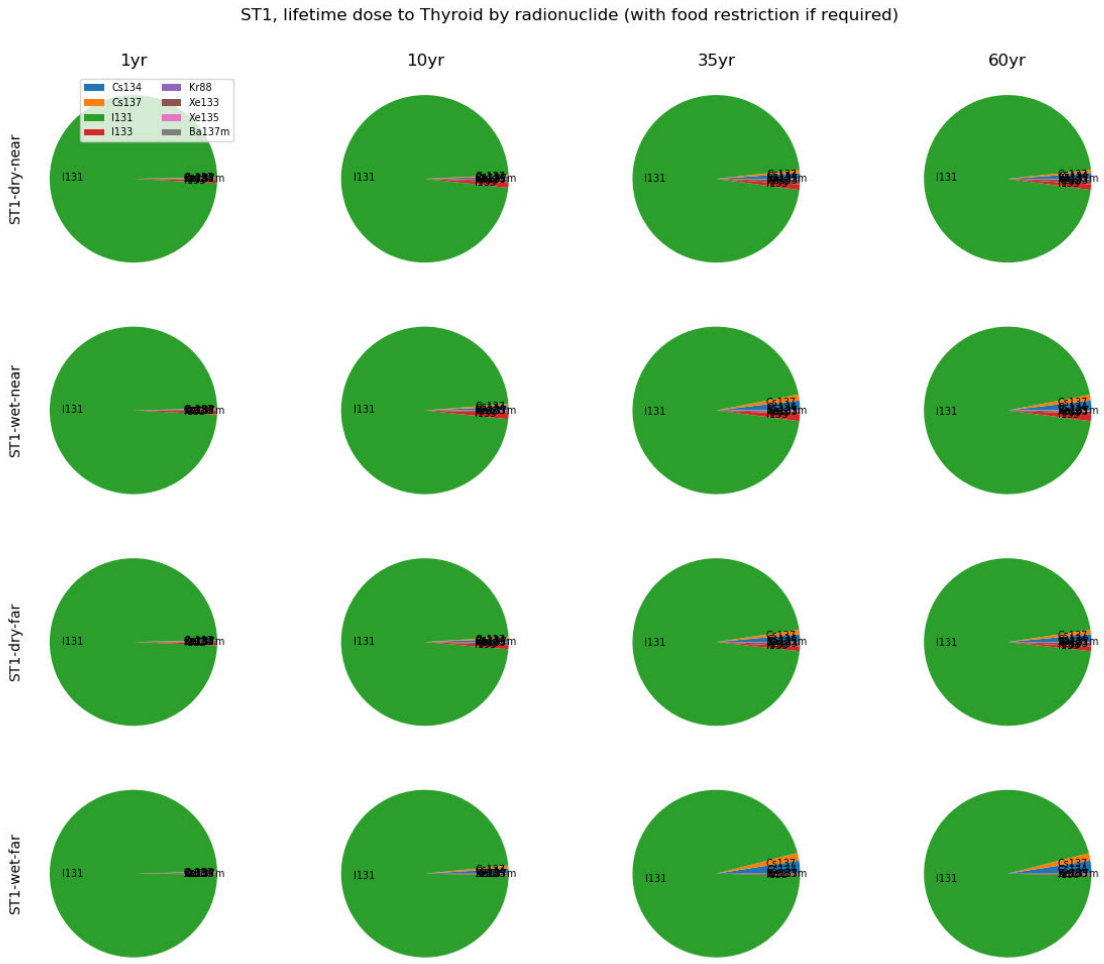


Figure A11 ST1-DBA – proportion of total lifetime thyroid dose by radionuclide

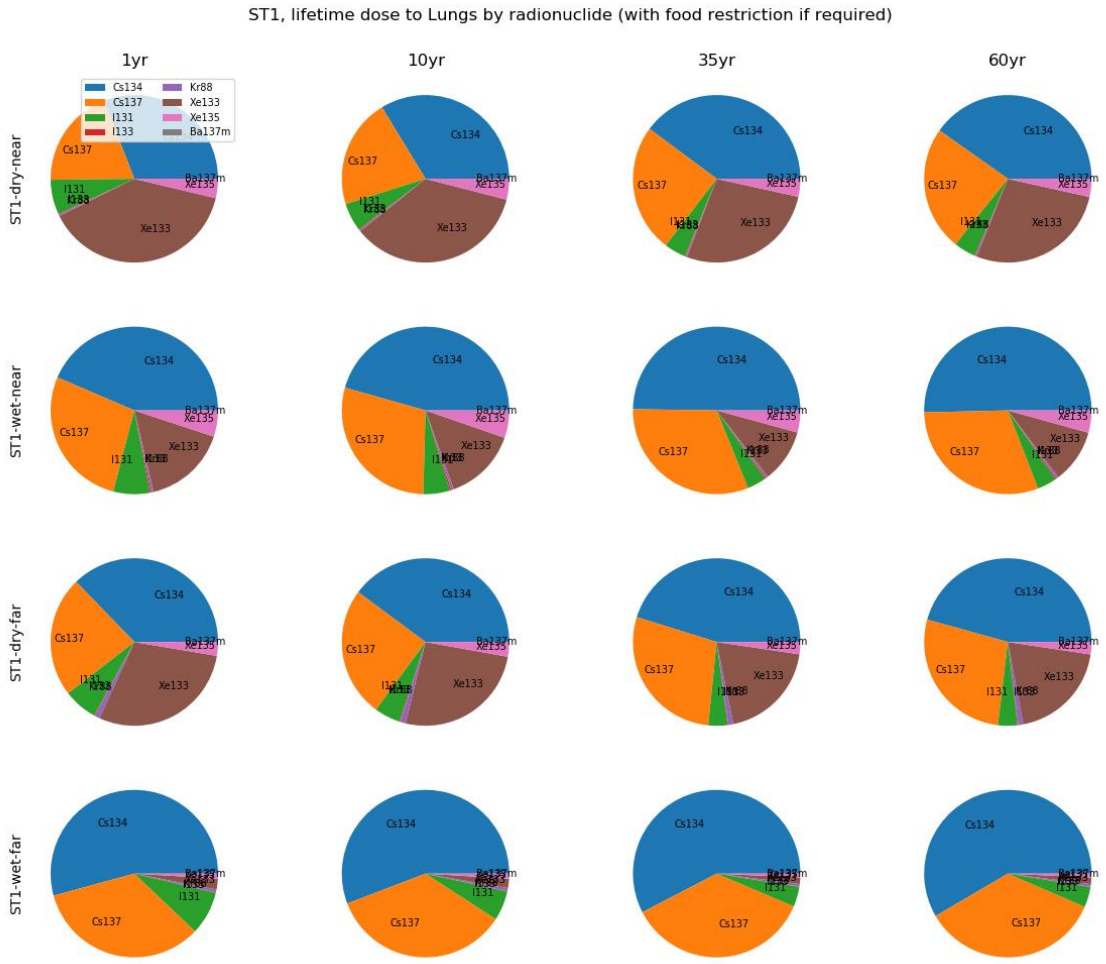


Figure A12 ST1-DBA – proportion of total lifetime lung dose by radionuclide

A2 ST2 - acute

Table A2 ST2-acute – total lifetime effective and thyroid doses by age group

	1yr	10yr	35yr	60yr
Effective mSv				
near-dry	1.7E+05	1.4E+05	8.4E+04	8.4E+04
near-wet	2.5E+05	2.0E+05	1.5E+05	1.5E+05
far-dry	1.7E+01	1.4E+01	8.5E+00	8.5E+00
far-wet	5.5E+01	4.5E+01	3.9E+01	3.8E+01
Thyroid mSv				
near-dry	3.2E+06	2.5E+06	1.5E+06	1.5E+06
near-wet	2.7E+06	2.2E+06	1.3E+06	1.3E+06
far-dry	3.1E+02	2.5E+02	1.5E+02	1.5E+02
far-wet	1.7E+02	1.4E+02	9.9E+01	9.9E+01

Table A2 gives the effective and thyroid lifetime doses at near and far locations. Total effective doses at the near locations range from 75 to 250 Sv. These are doses at which deterministic or acute effects might be expected and this is discussed further in Section 6.4.

Figure A13 shows all lifetime doses. In all examples, the thyroid dose is the largest. In the dry examples, other organ doses are relatively very small compared to the thyroid. In wet examples other organs doses are still relatively small but somewhat larger than the dry examples, particularly the wet-far example where other organs doses are between 30-50% of the thyroid dose. Figure A14 shows all lifetime dose omitting thyroid.

There is a clear age-dependency in the thyroid dose in all examples, with the 1-year-old being between about twice the corresponding adult thyroid dose. The other tissues show some age dependency in the wet examples with 1-year-old dose being between about 20-100% larger than the 35-year-old. The dry examples do not show this dependency in the other organs.

Figure A15 shows that for thyroid dose, inhalation is the dominant pathway in all examples, with external deposition being significant in the wet examples. Whereas Figure A16 shows that for ovary dose the dominant pathway is external deposition in all examples, with the dry example also getting significant contribution from the external pathways and in the dry-far example, also from ingestion.

Figure A17 shows the predicted delivery over time of thyroid dose. The patterns of dose delivery are very similar in each example, with most of the dose delivered in the first year for all age groups and locations and falling by over 2-4 orders of magnitude by the second year. The 1-year-old received more dose than other age groups in the first year. In subsequent years the dose profiles converge.

Figure A18 shows the predicted delivery of ovary dose over time. The falloff of dose between first and subsequent years is gentler than seen for thyroid dose, and there are differences in patterns between dry and wet examples, with the dry example tending to converge and the wet examples diverging in later years.

Figure A19 shows the delivery of thyroid dose from different pathways to the 1-year-old. The impact on ingestion of food restriction and its subsequent removal, can be seen.

Figure A20 shows the total lifetime thyroid dose contribution by radionuclide. In all examples isotopes of iodine are dominant. Figure A21 shows the lifetime ovary dose contribution by radionuclide where iodine isotopes are still significant but so are a range of other radionuclides notably isotopes of caesium.

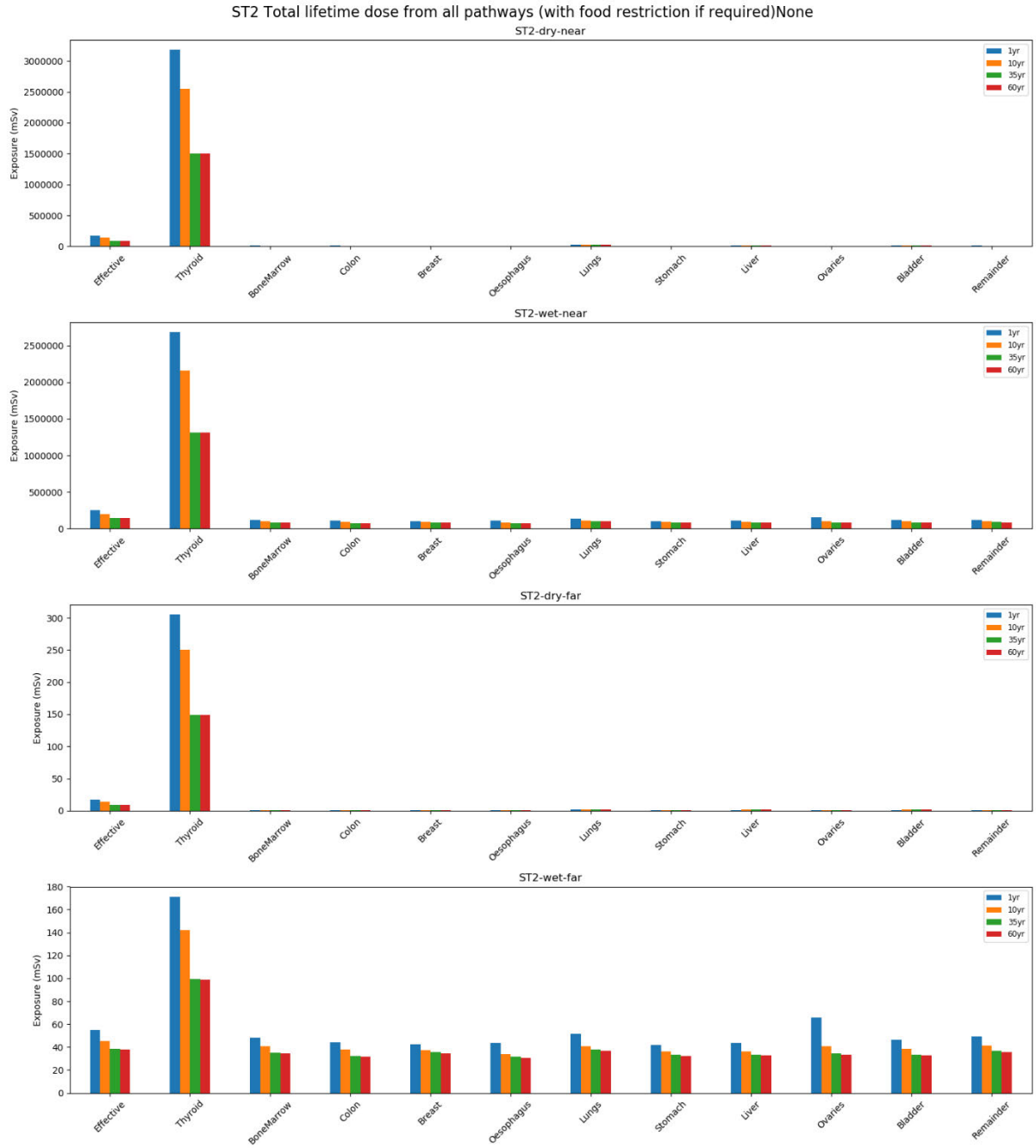


Figure A13 ST2-acute – total lifetime doses

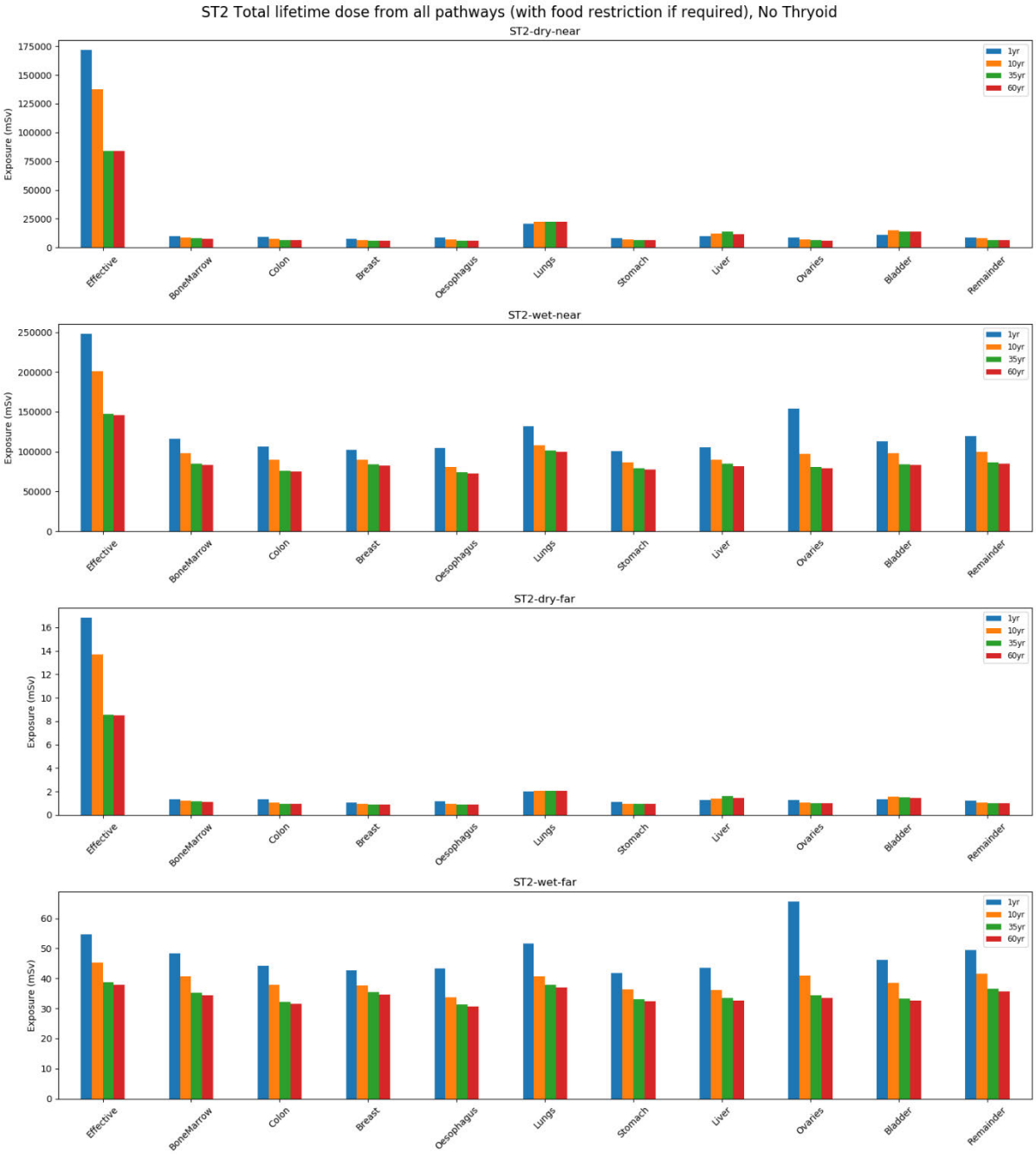


Figure A14 ST2-acute – total lifetime doses without thyroid

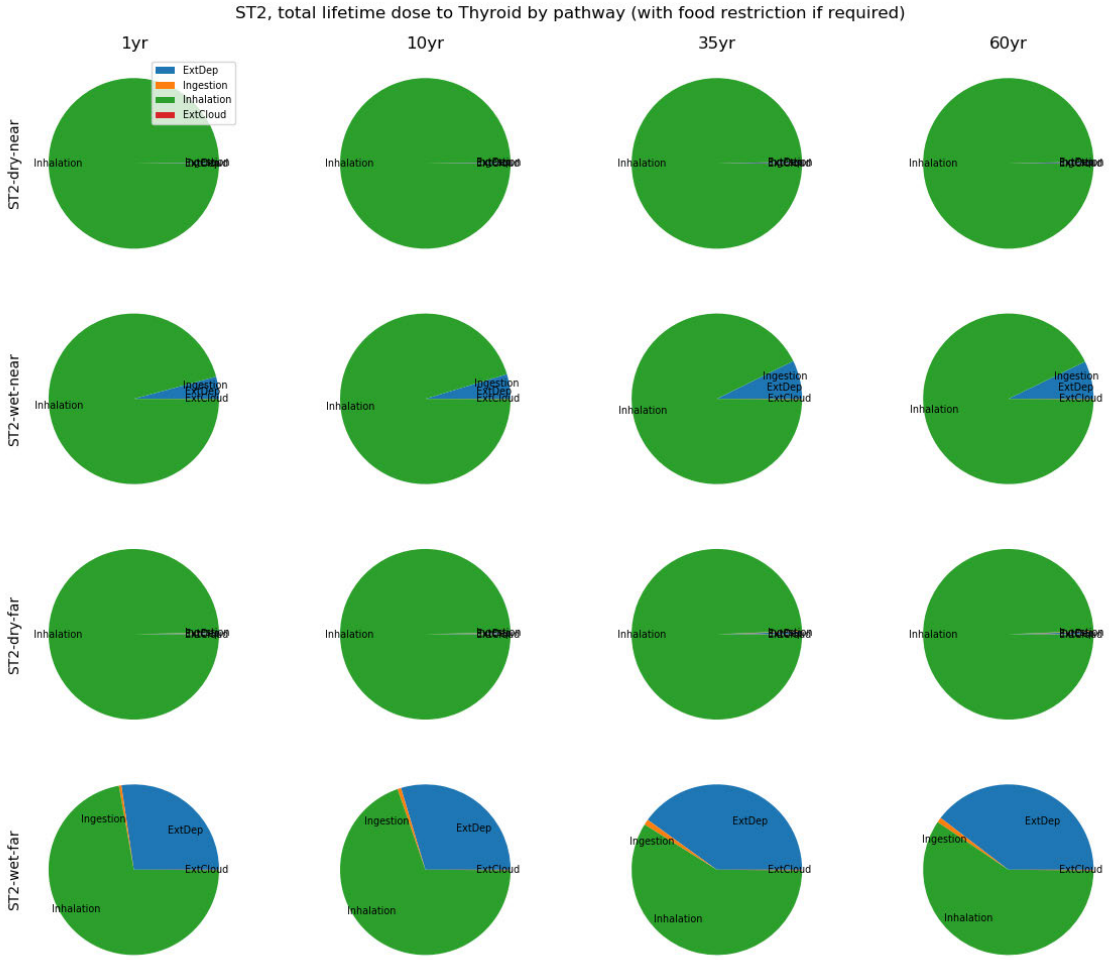


Figure A15 ST2-acute – proportion of total lifetime thyroid dose by pathway

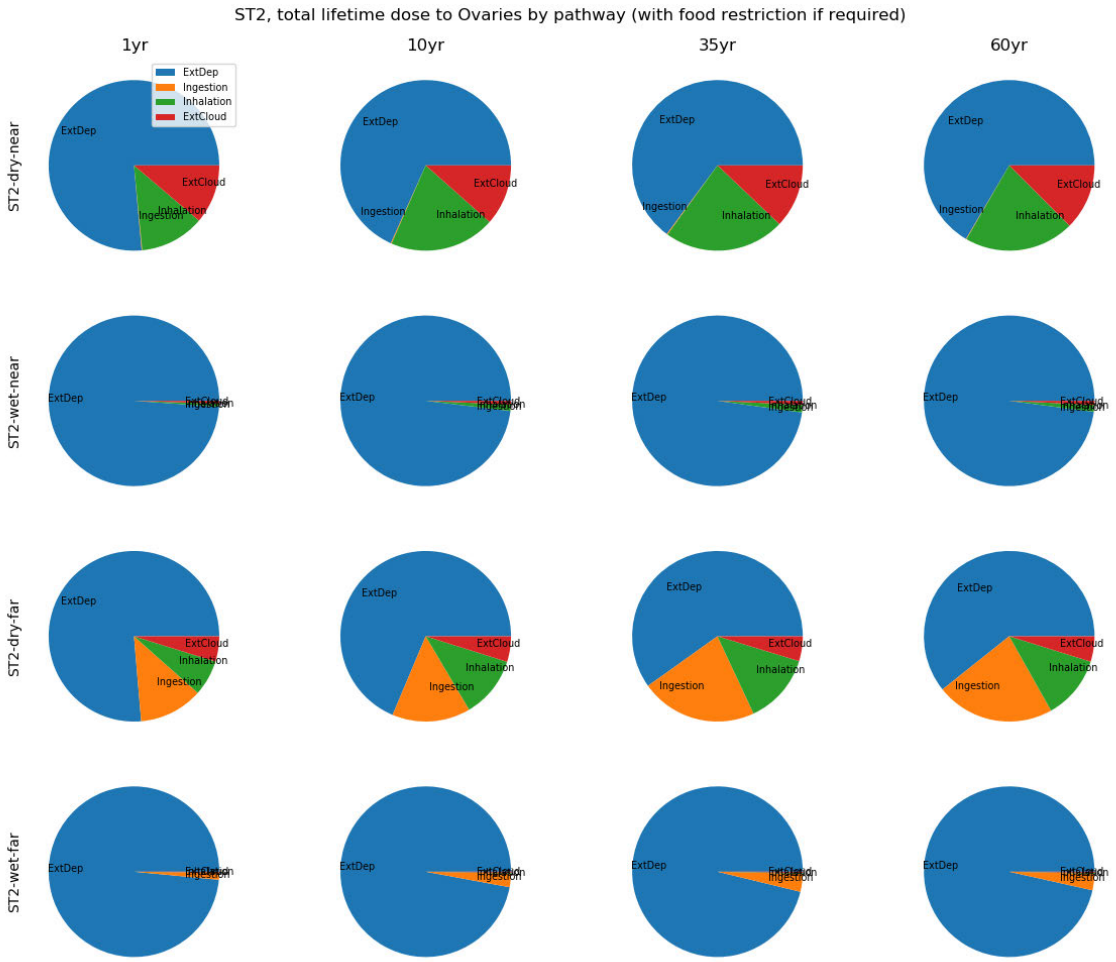


Figure A16 ST2-acute – proportion of total lifetime ovary dose by pathway

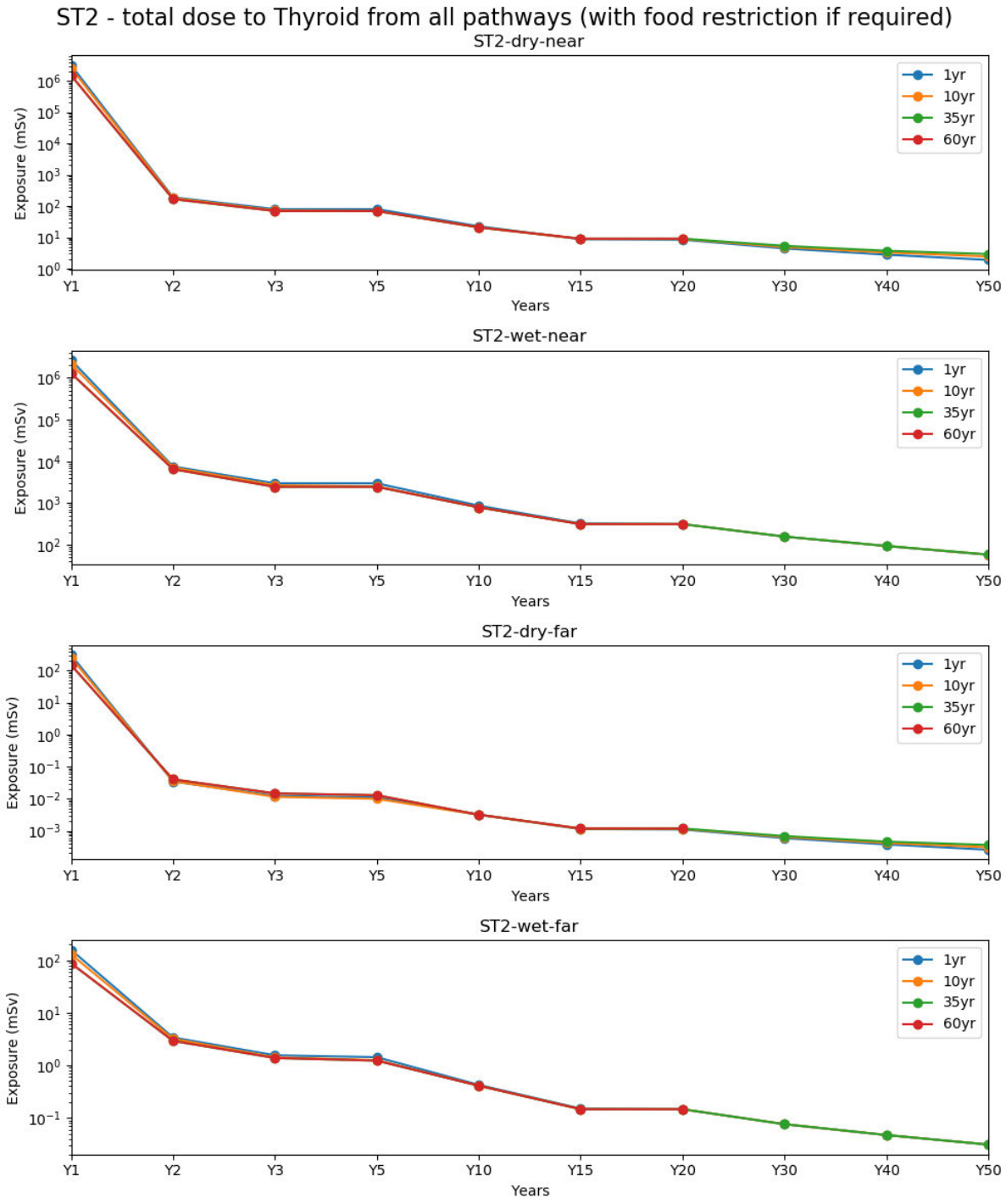


Figure A17 ST2-acute – total thyroid dose by year for each age group

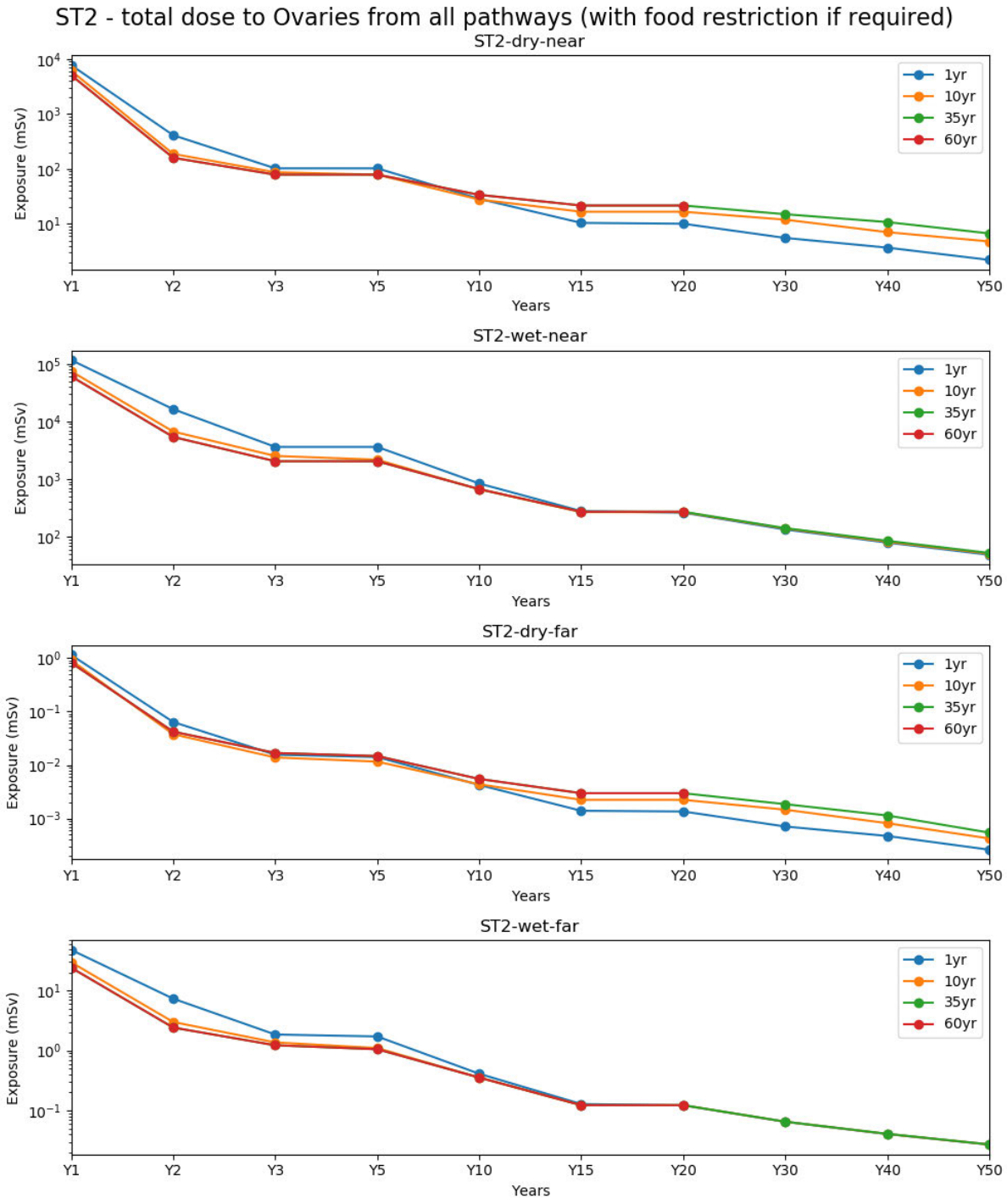


Figure A18 ST2-acute – total ovary dose by year for each age group

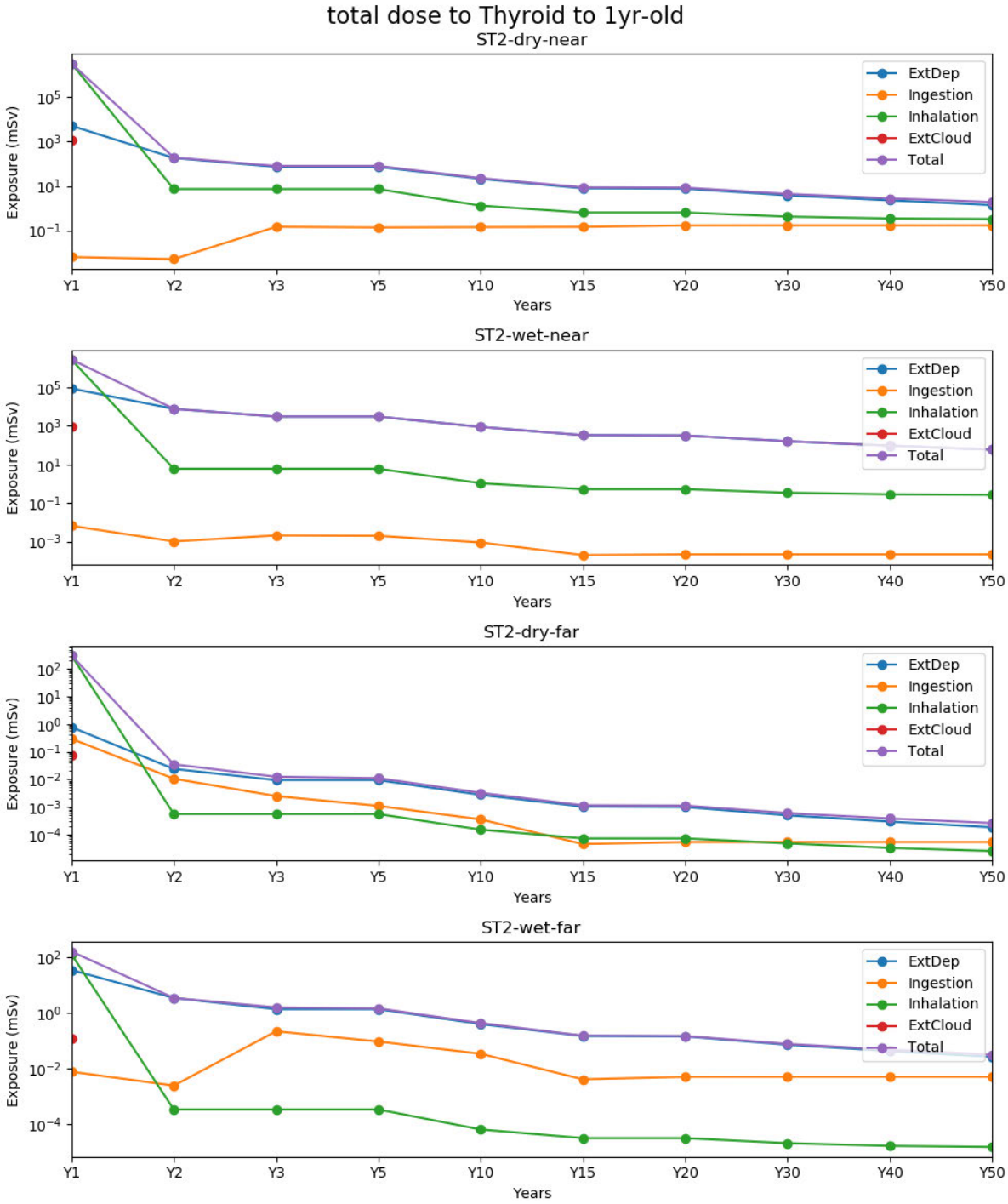


Figure A19 ST2-acute – total lifetime thyroid dose to 1-year-old by pathway, by year of delivery

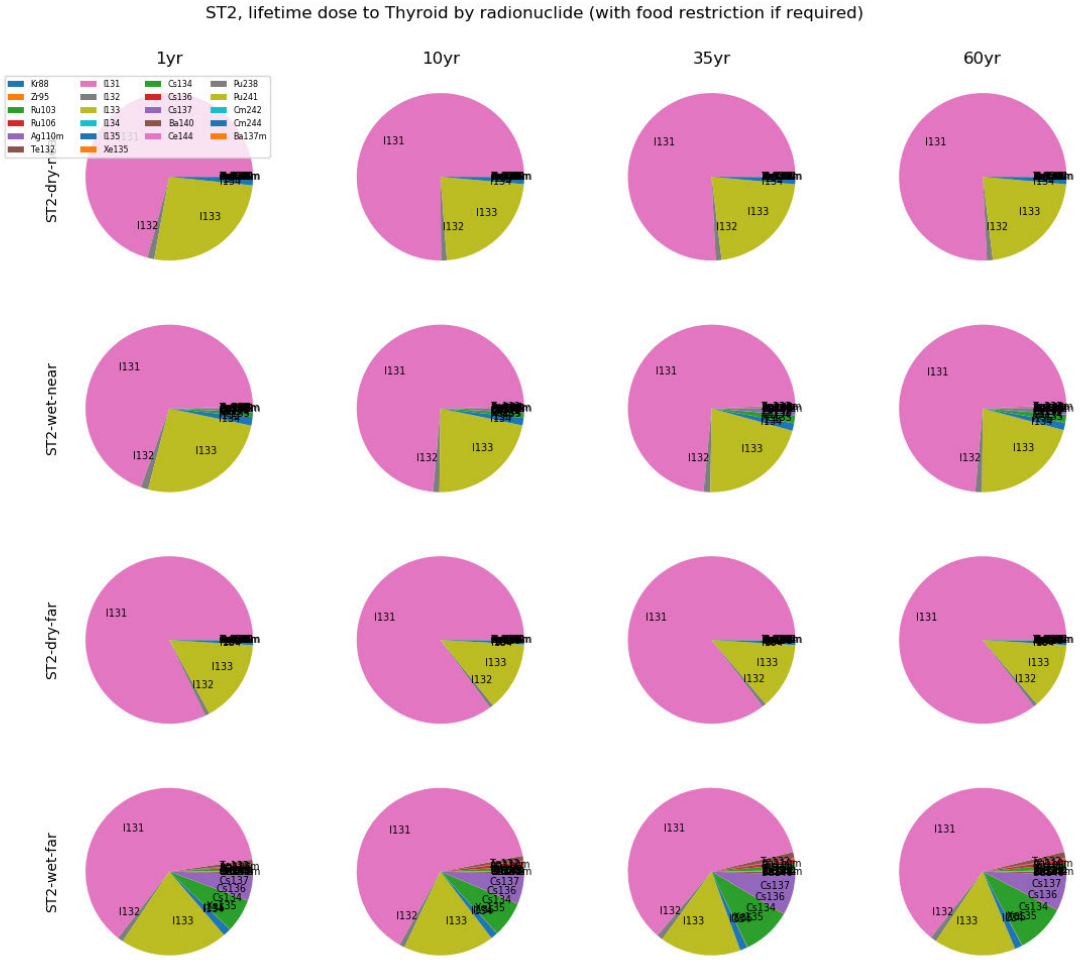


Figure A20 ST2-acute – proportion of total lifetime thyroid dose by radionuclide

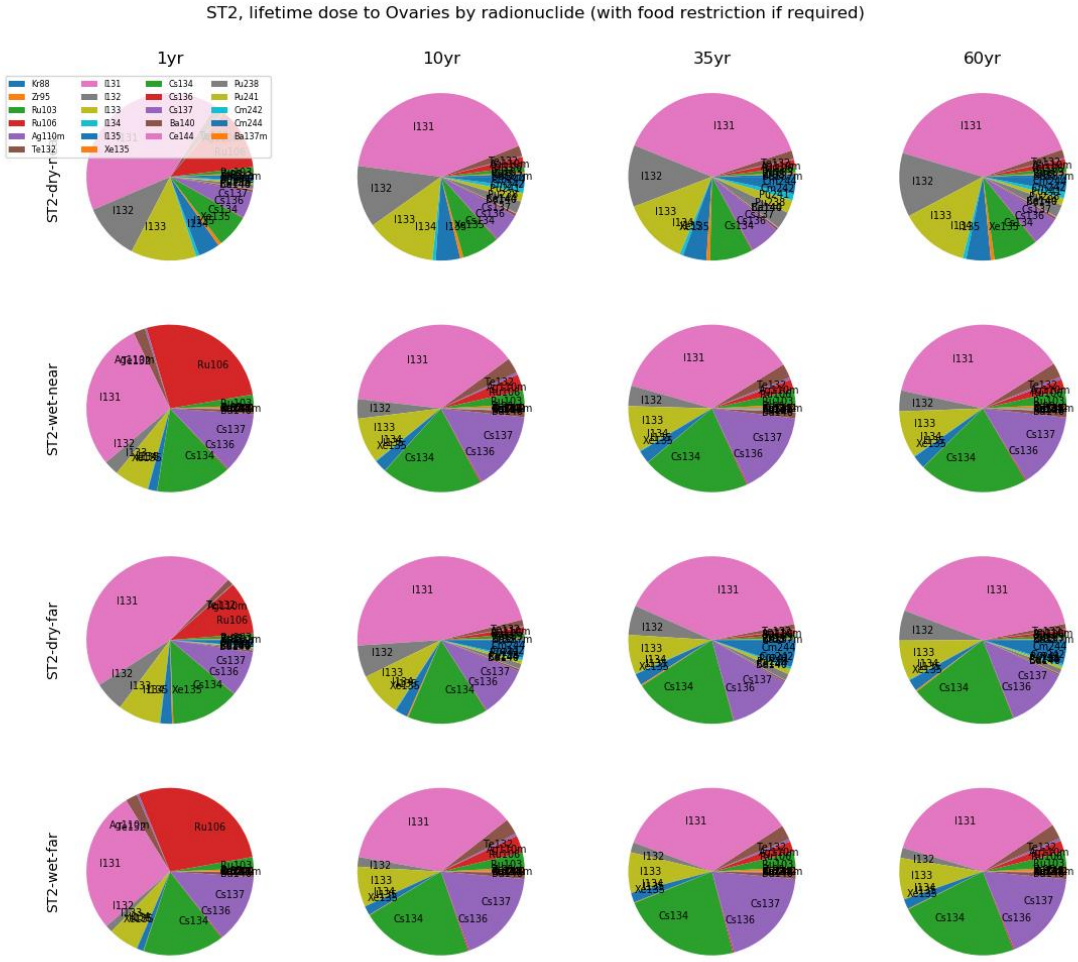


Figure A21 ST2-acute – proportion of total lifetime ovary dose by radionuclide

A3 ST3 - fuel

Table A3 ST3-fuel – total lifetime effective and bone marrow doses by age group

	1yr	10yr	35yr	60yr
Effective mSv				
dry-near	6.0E+03	6.9E+03	7.6E+03	6.7E+03
wet-near	6.0E+03	5.9E+03	6.0E+03	5.4E+03
dry-far	3.4E+01	3.9E+01	4.0E+01	3.4E+01
wet-far	1.4E+02	1.1E+02	9.1E+01	8.1E+01
Bone marrow mSv				
dry-near	1.1E+04	9.0E+03	8.3E+03	7.1E+03
wet-near	8.5E+03	6.9E+03	6.3E+03	5.6E+03
dry-far	7.9E+01	7.4E+01	5.9E+01	4.7E+01
wet-far	2.4E+02	2.0E+02	1.4E+02	1.0E+02

Table A3 gives the predicted lifetime effective and bone marrow doses and Figure A22 ST3-fuel – total lifetime dose by organ shows all lifetime organ and effective doses. In the near examples and the far-dry example, the doses to liver, lungs and bone marrow are the largest. In the wet-far example, the bone marrow is the organ that receives the highest dose, with corresponding doses to other organs that are very roughly 50%. Age-dependency is visible but not strong with the most severe being the wet-far Bone Marrow and Ovary doses where the 1-year-old is just more than twice the 35-year-old. Also notable are the liver doses in the near and dry-far examples, where the 35-year-old is roughly 2 times the 1-year-old.

Figure A23 ST3-fuel – proportion of total lifetime bone marrow dose by pathway shows predicted total bone marrow dose contribution by pathway. For the near examples and the dry-far example, inhalation is the dominant pathway. The wet-near example has a sizable external deposition component, whereas the dry-far has a sizable ingestion component. The wet-far contributions are roughly equally split between external deposition and ingestion with the other pathways making a minor contribution, except for the 60-year-old where the split is roughly 70% external deposition and 25% ingestion.

Figure A24 ST3-fuel – proportion of total lifetime liver dose by pathway shows predicted liver dose contribution by pathway which shows a similar pattern as bone marrow. For the near examples and the dry-far example, as for bone marrow, the inhalation pathway dominates and for the wet-far example external deposition dominates.

Figure A25 and Figure A26 show how bone marrow and liver dose are delivered as a function of period after the accident. A large amount of dose is delivered in the first year but a significant portion depending on age and organ is delivered after twenty years.

Figure A27and Figure A28 show the delivery of bone marrow and liver dose to a 1-year-old by pathway For this source term, the resulting activity concentrations in the local food mean that food restrictions would need to be implemented and all graphs clearly show the impact of food restriction on the ingestion dose.

Figure A29 and Figure A30 shows the contribution to lifetime bone marrow and liver dose by radionuclide.

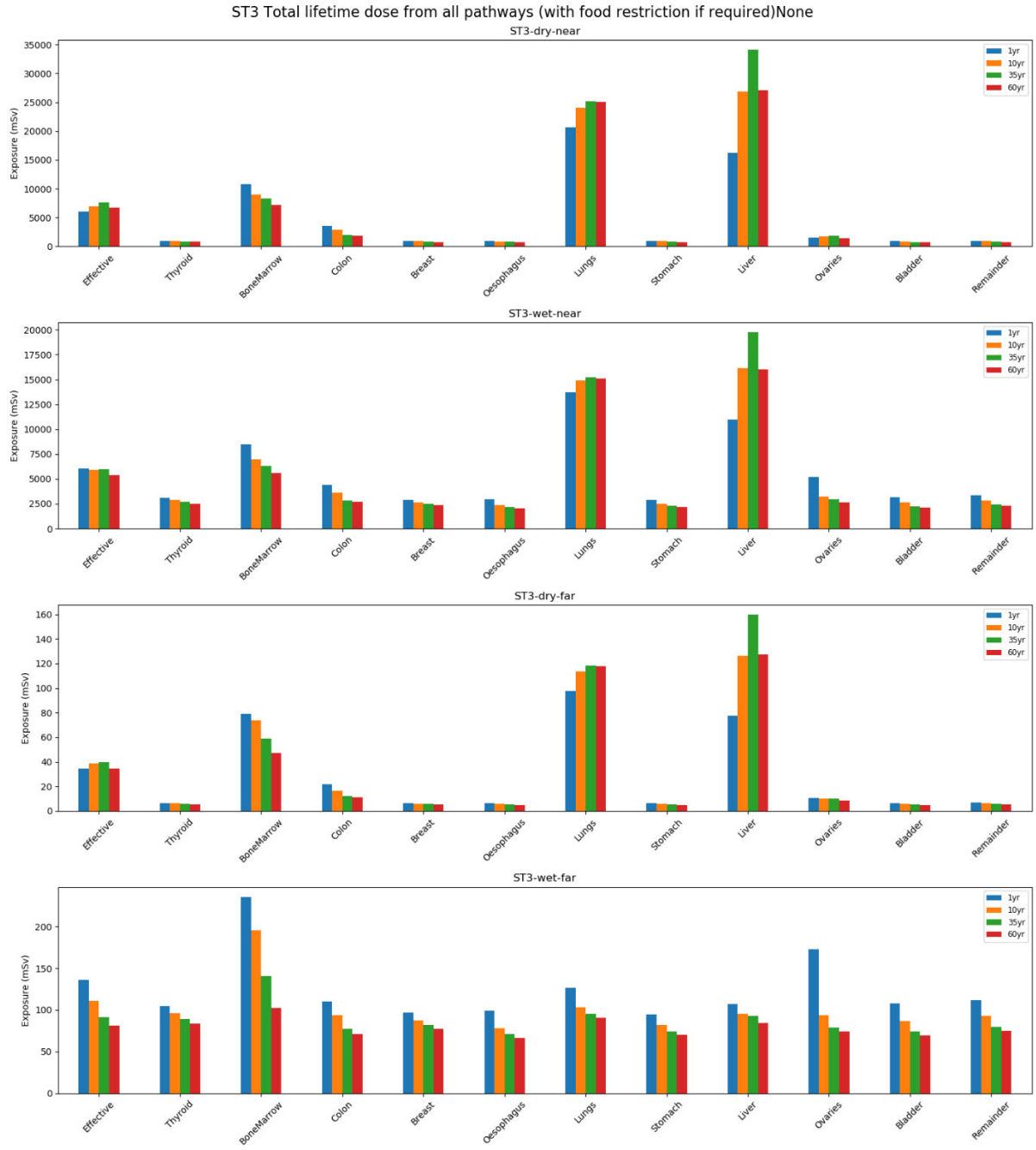


Figure A22 ST3-fuel – total lifetime dose by organ

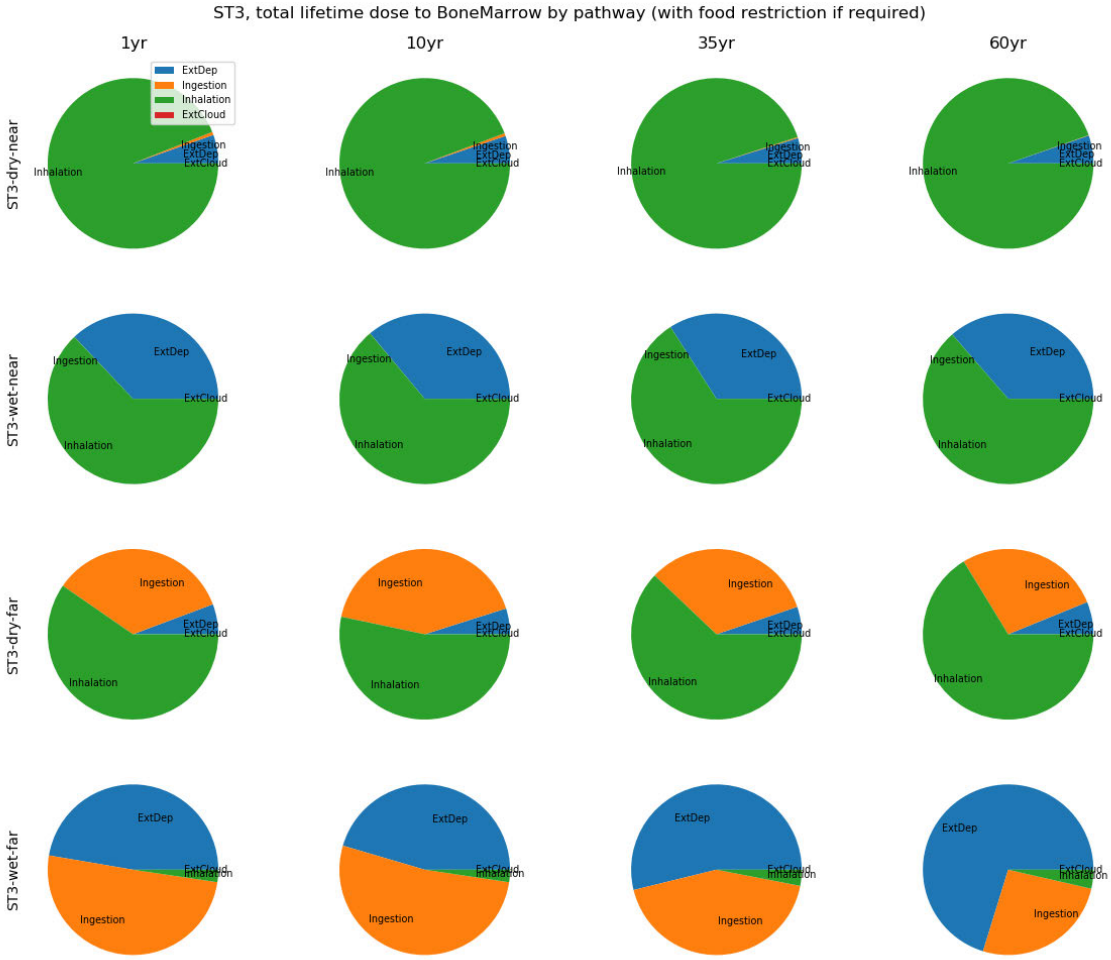


Figure A23 ST3-fuel – proportion of total lifetime bone marrow dose by pathway

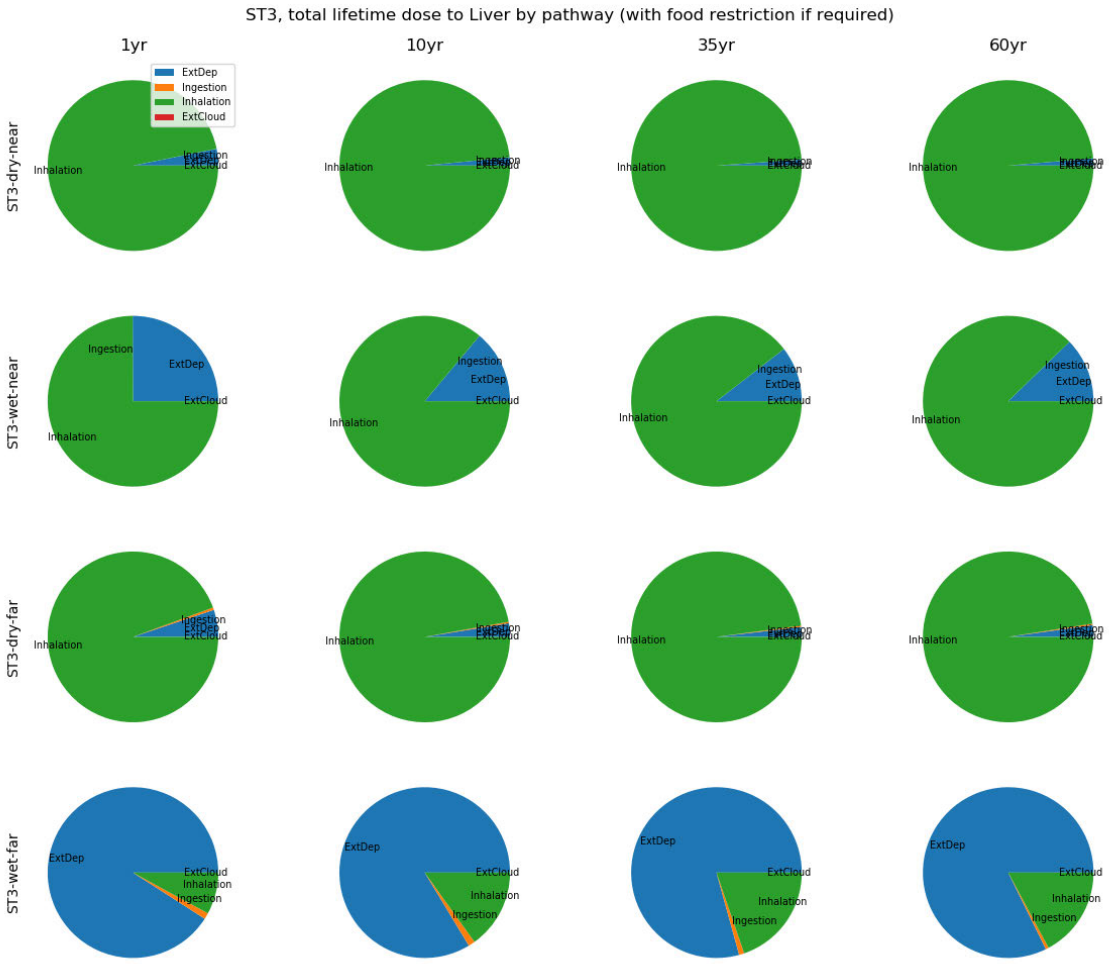


Figure A24 ST3-fuel – proportion of total lifetime liver dose by pathway

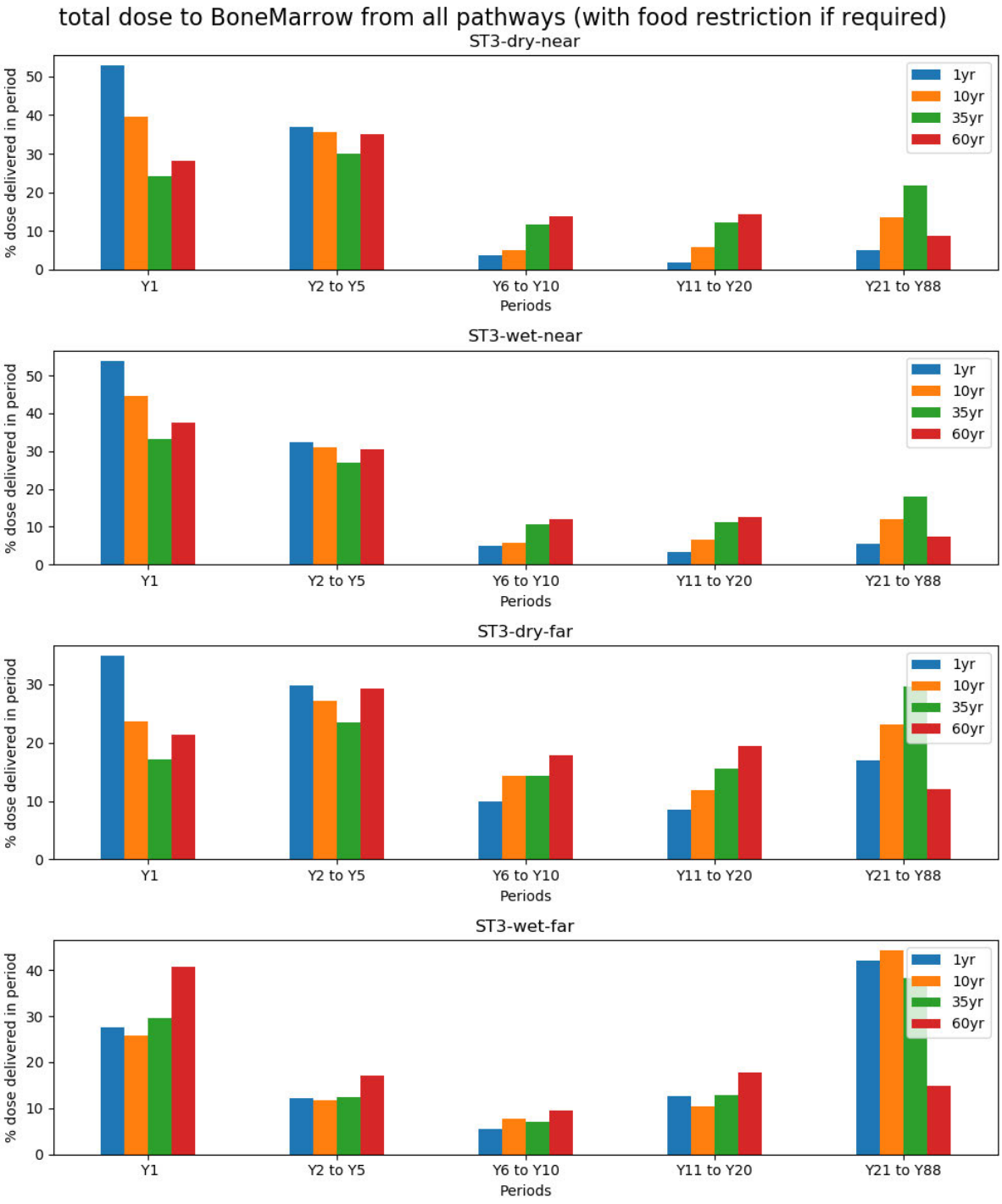


Figure A25 ST3-fuel – delivery of total **bone marrow dose by period**

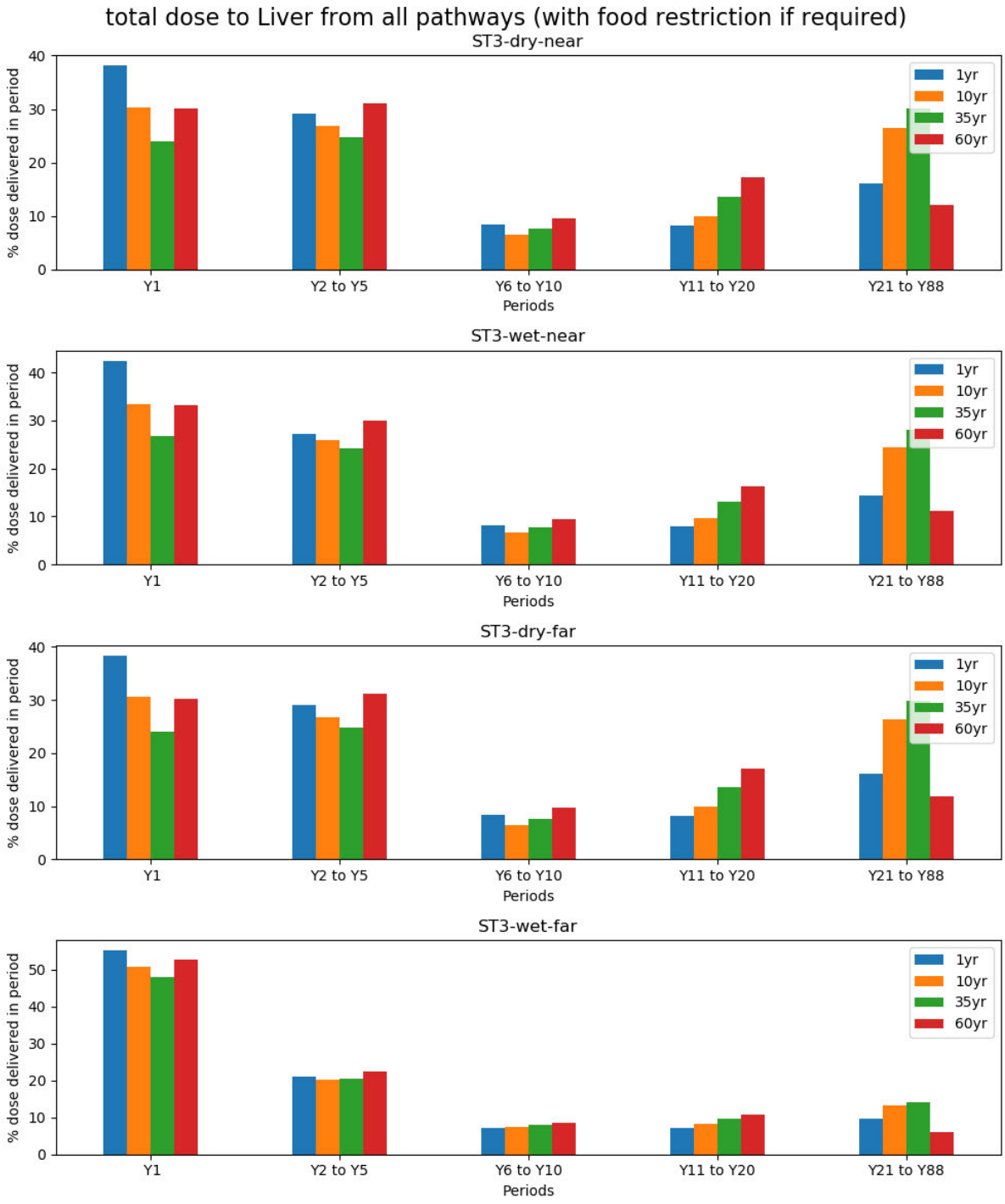


Figure A26 ST3-fuel – delivery of total liver dose by period

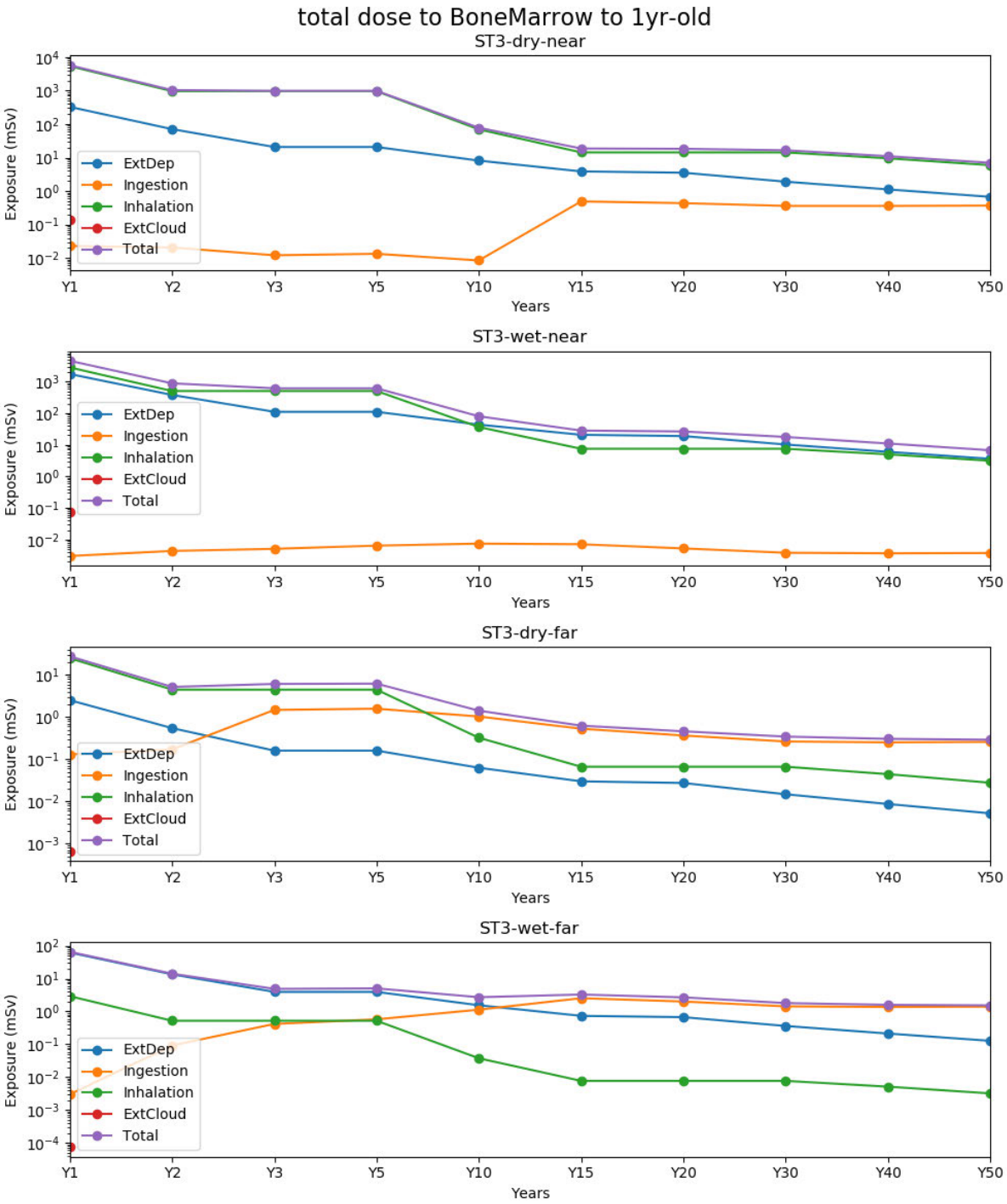


Figure A27 ST3-fuel – total lifetime bone marrow dose to 1-year-old by pathway, by year of delivery

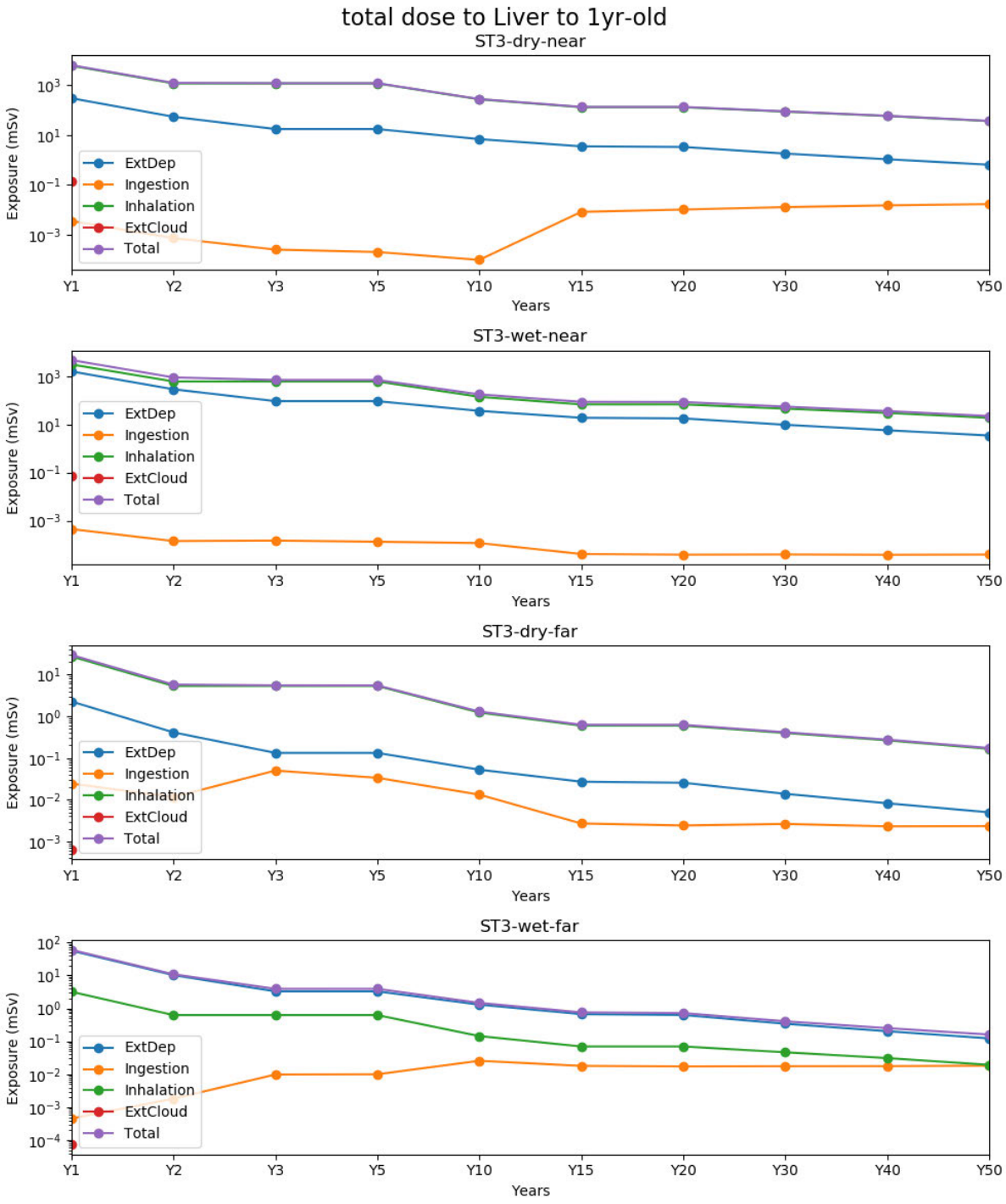


Figure A28 ST3-fuel – total lifetime liver dose to 1-year-old by pathway, by year of delivery

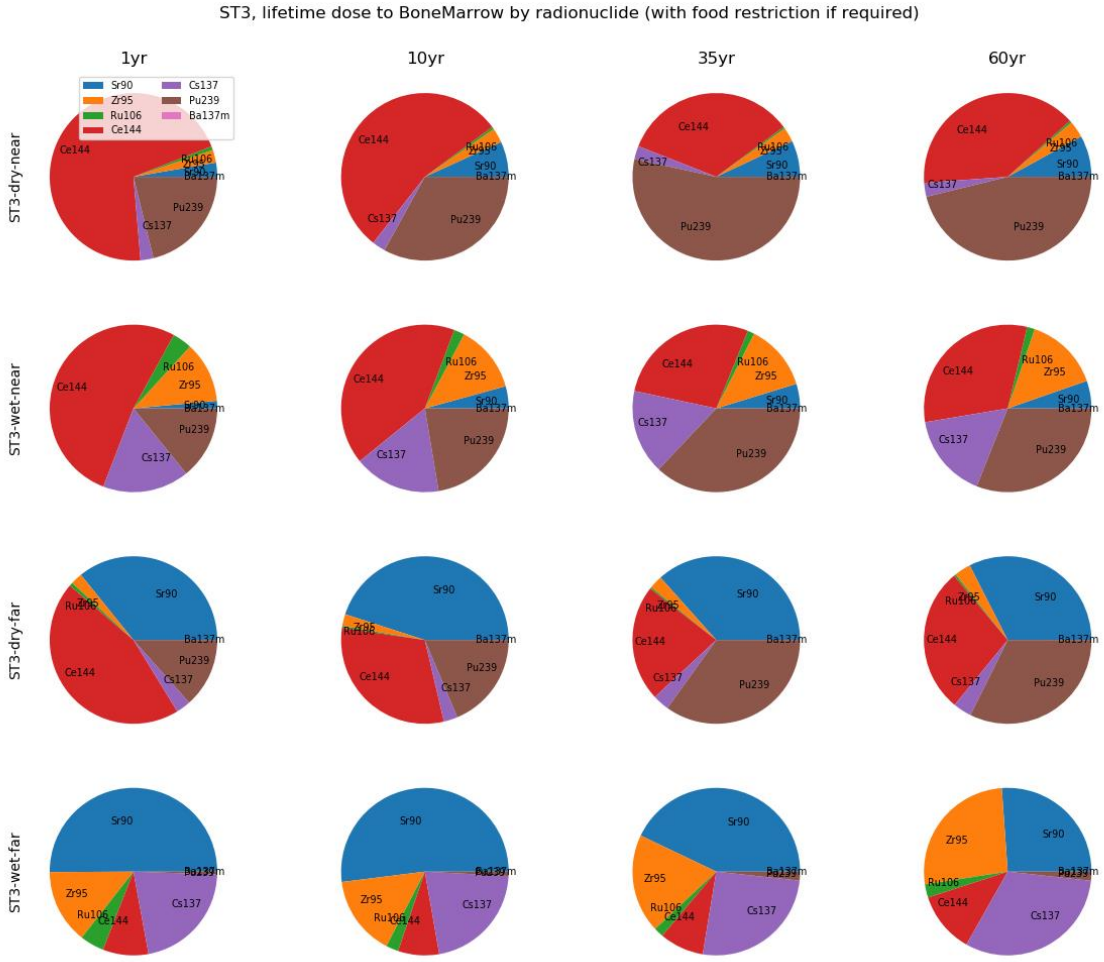


Figure A29 ST3-fuel – proportion of total lifetime bone marrow dose by radionuclide

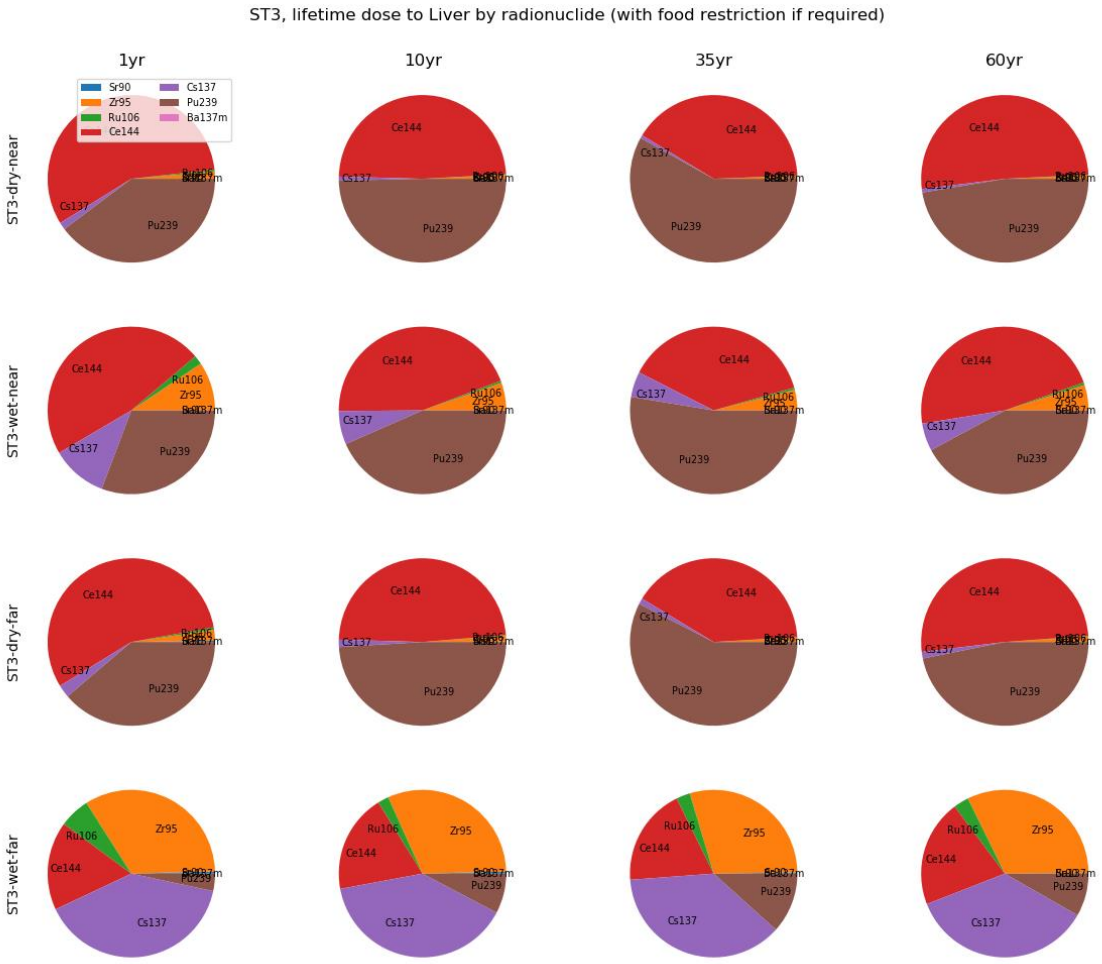


Figure A30 ST3-fuel – proportion of total lifetime liver dose by radionuclide

A4 ST4 – Chornobyl

Table A4 ST4-Chornobyl – total lifetime effective and thyroid doses by age group

	1yr	10yr	35yr	60yr
Effective mSv				
near-dry	4.6E+03	4.3E+03	3.4E+03	3.3E+03
near-wet	6.3E+03	5.6E+03	4.7E+03	4.5E+03
far-dry	7.0E+01	7.3E+01	6.0E+01	5.4E+01
far-wet	1.3E+02	1.2E+02	9.3E+01	8.8E+01
Thyroid mSv				
near-dry	5.2E+04	4.3E+04	2.6E+04	2.6E+04
near-wet	3.2E+04	2.7E+04	1.7E+04	1.7E+04
far-dry	3.8E+02	3.2E+02	2.0E+02	2.0E+02
far-wet	6.6E+02	5.6E+02	3.5E+02	3.5E+02

Table A4 gives the effective and thyroid lifetime doses, and Figure A31 shows all the lifetime organ and effective doses. In all examples the thyroid and lung doses are the highest. There is some age dependency in the thyroid doses with the 1-year-old dose being about a factor 2

higher than the 35-year-old. The lung dose and most of the other organ exhibit very little age dependency.

Figure A32 and Figure A33 show the lifetime thyroid and lung dose contributions by pathway. Both have similar patterns with for all examples most of the dose from inhalation, and in the wet example a significant component from external deposition.

Figure A34 and Figure A35 show how thyroid and lung dose are delivered. For both organs all most all the dose is delivered in the first year.

Figure A36 and Figure A37 shows the contribution to lifetime thyroid and lung dose by radionuclide. For thyroid in all examples, ^{131}I dominates, with the remainder coming from a range of radionuclides including ^{133}I. For the lung in all example, ^{90}Sr contributes more than 50% of the dose, with the remainder coming from a range of radionuclides.

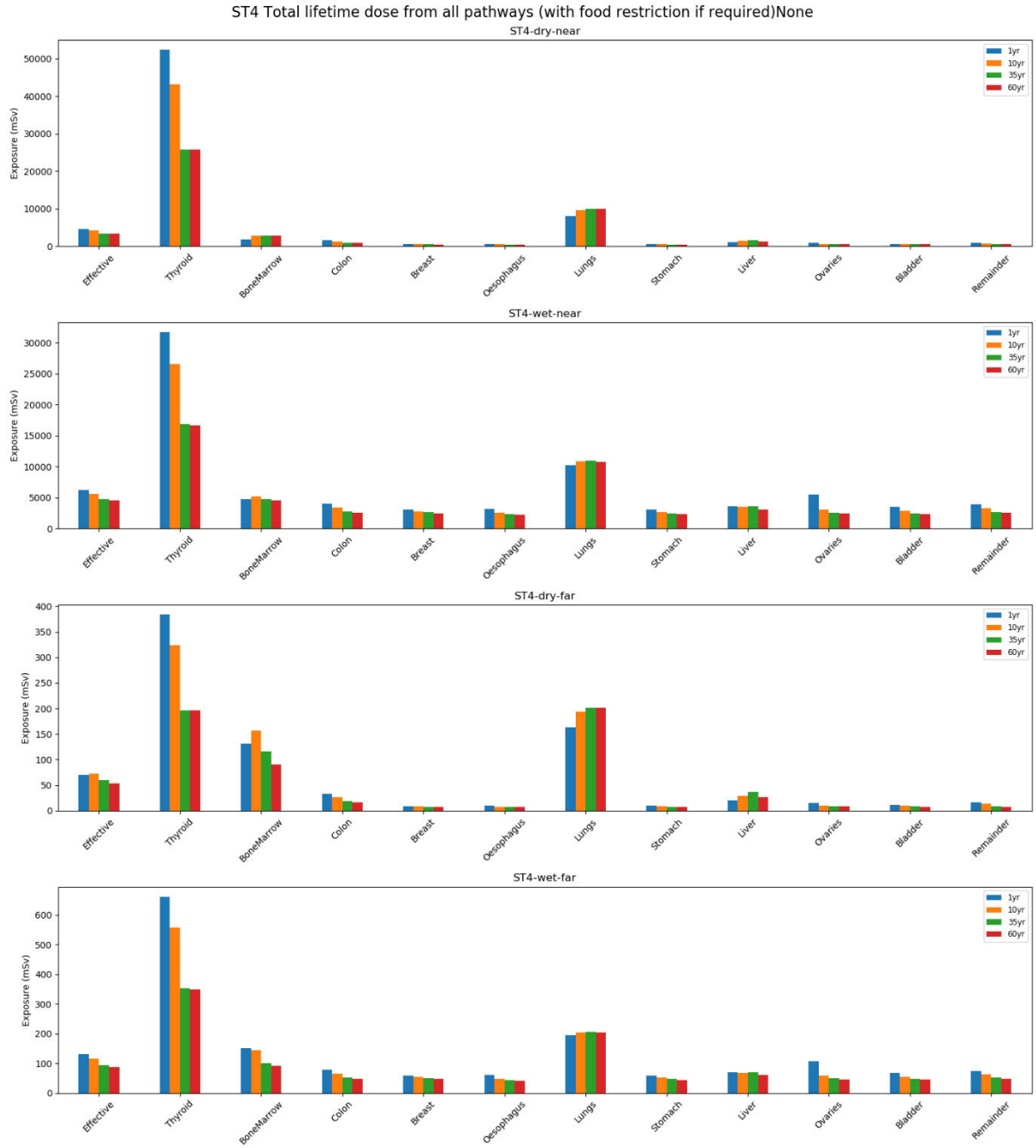


Figure A31 ST4-Chornobyl – total lifetime dose by organ

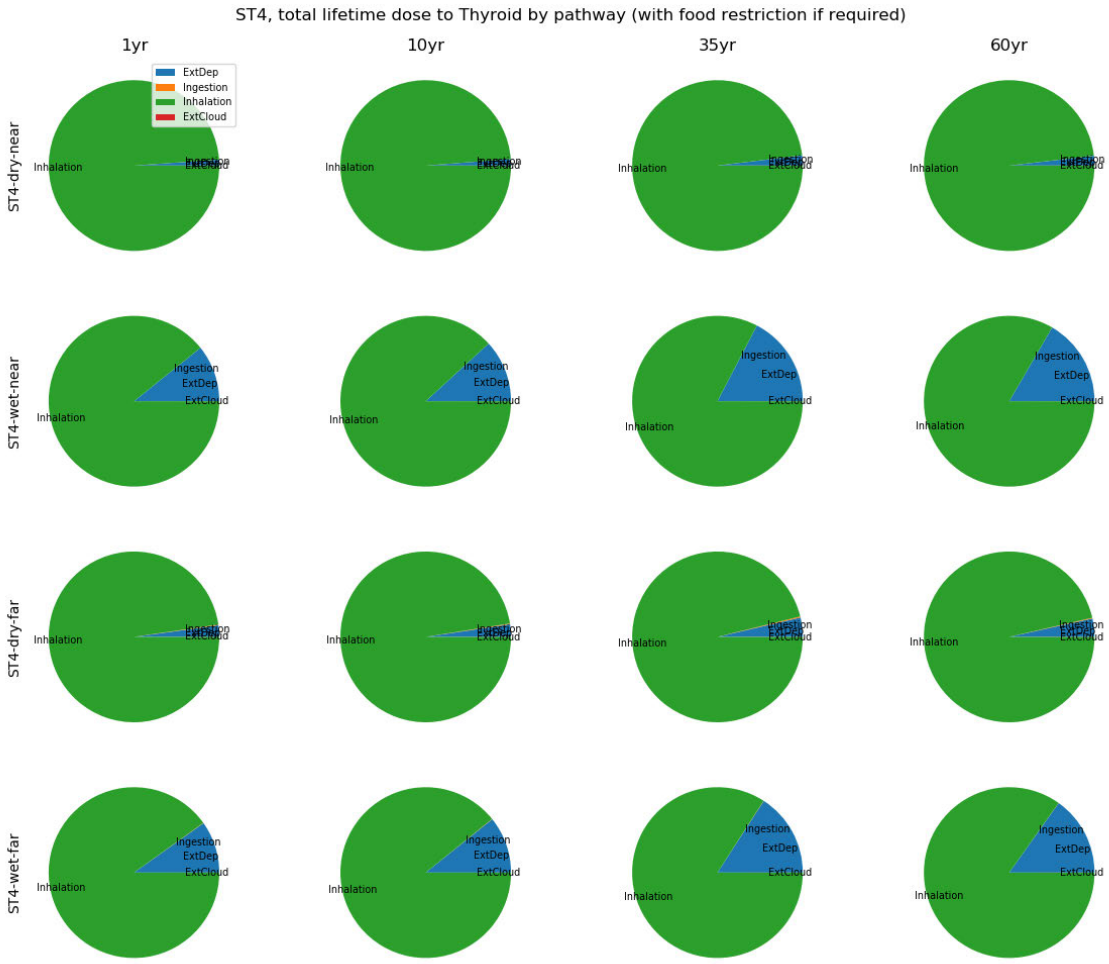


Figure A32 ST4-Chornobyl – proportion of total lifetime thyroid dose by pathway

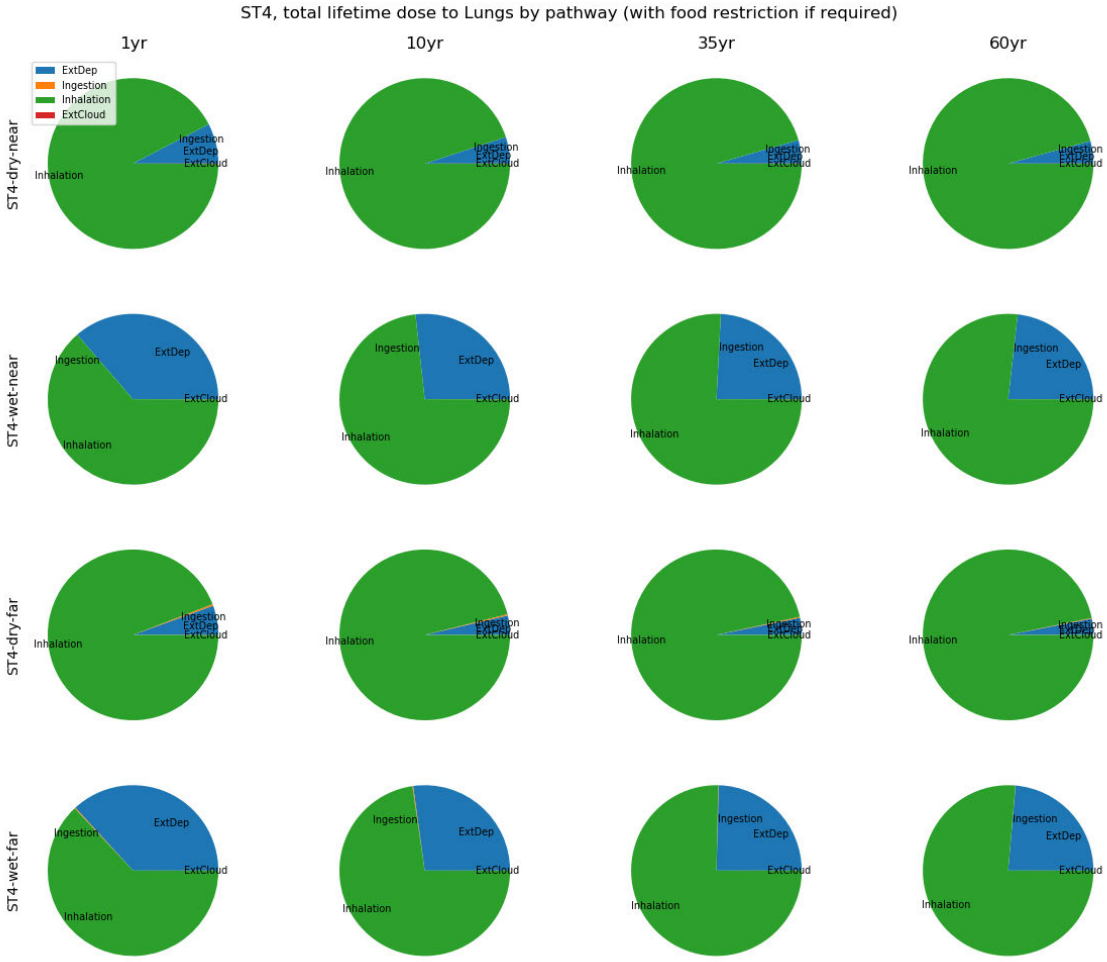


Figure A33 ST4-Chornobyl – proportion of total lifetime lung dose by pathway

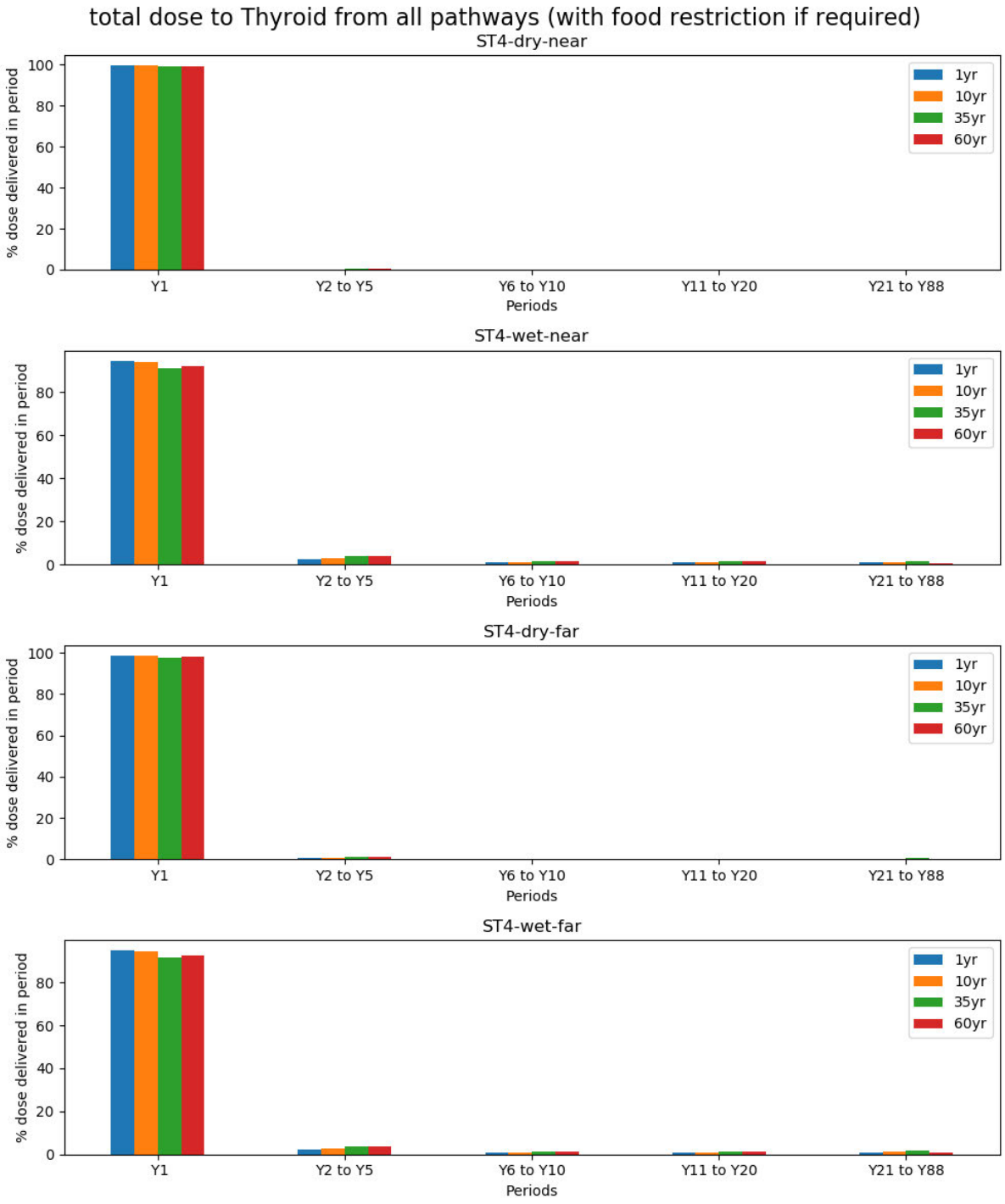


Figure A34 ST4-Chornobyl – delivery of total thyroid dose by period

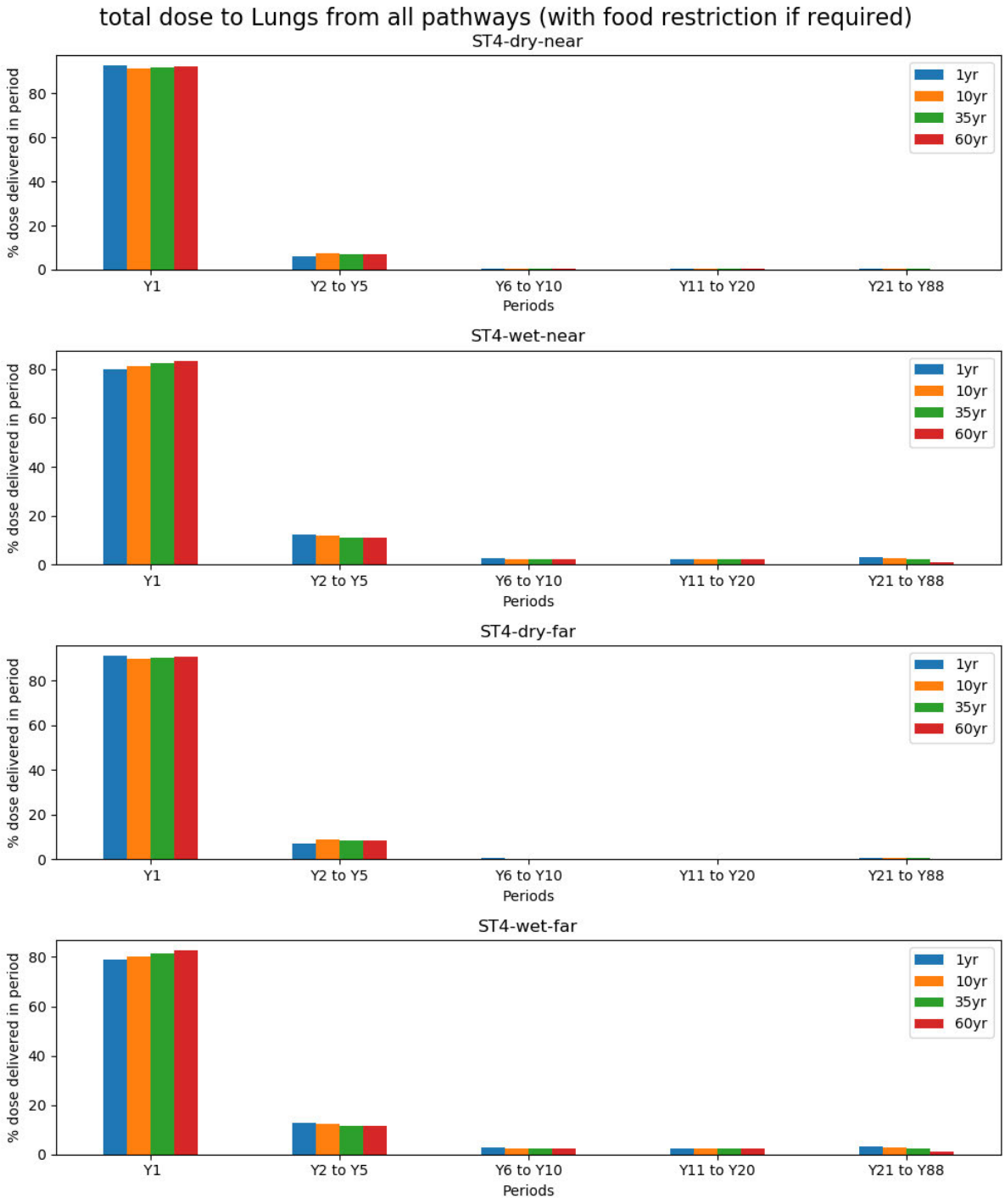


Figure A35 ST4-Chornobyl – delivery of total lung dose by period

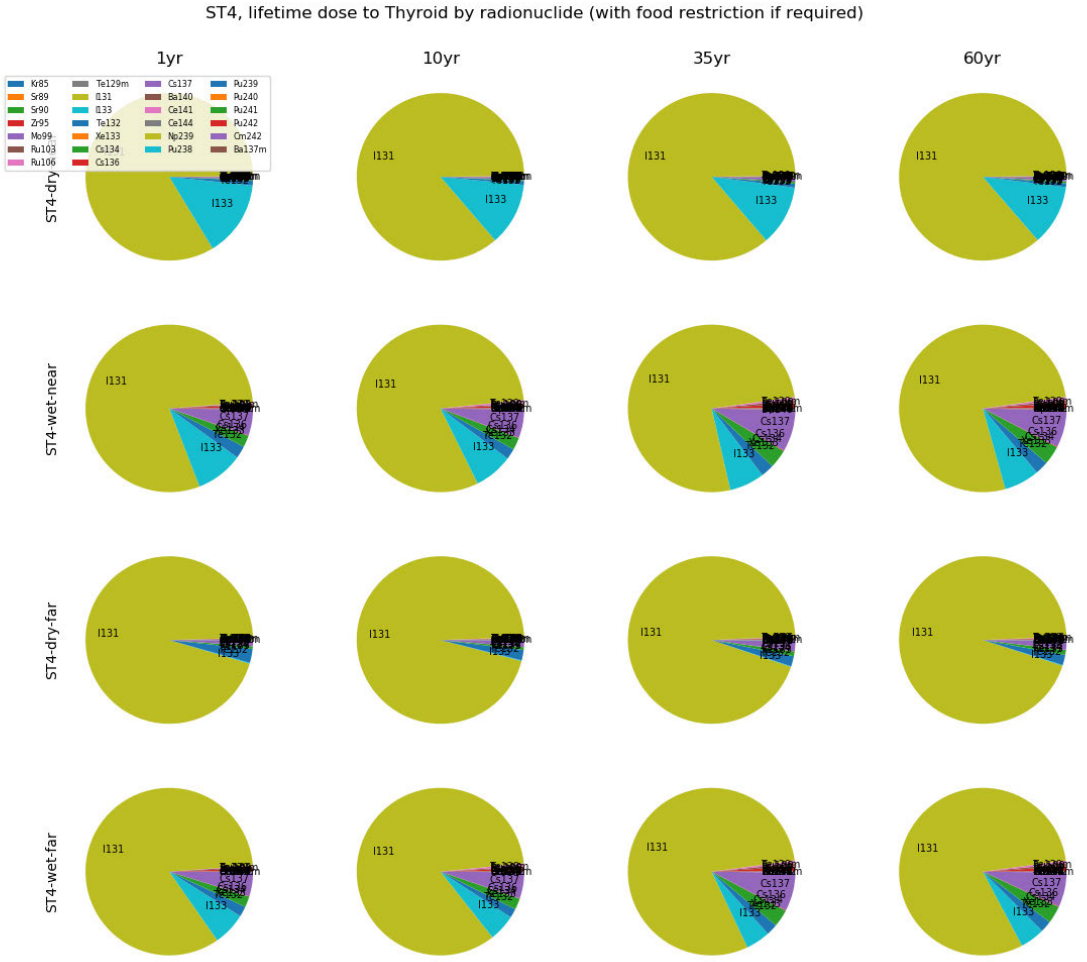


Figure A36 ST4-Chornobyl – proportion of total lifetime thyroid dose by radionuclide

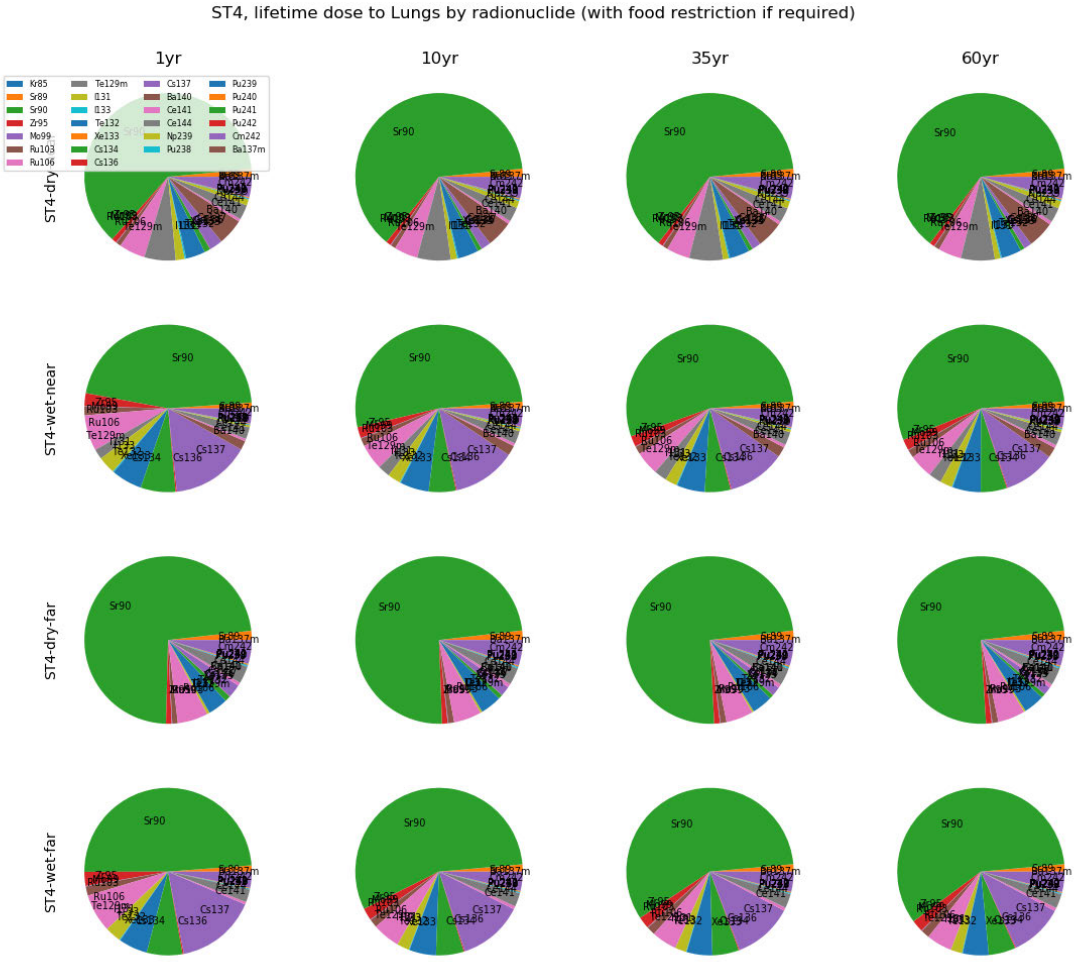


Figure A37 ST4-Chornobyl – proportion of total lifetime lung dose by radionuclide

About the UK Health Security Agency

UKHSA is responsible for protecting every member of every community from the impact of infectious diseases, chemical, biological, radiological and nuclear incidents and other health threats. We provide intellectual, scientific and operational leadership at national and local level, as well as on the global stage, to make the nation health secure.

UKHSA is an executive agency, sponsored by the Department of Health and Social Care.

www.gov.uk/government/organisations/uk-health-security-agency

Version: Issue 1

For queries relating to this document, please contact: [redacted]

Published: May 2024

UKHSA supports the Sustainable Development Goals