Committee on Medical Aspects of Radiation in the Environment (COMARE)

SEVENTEENTH REPORT

Further consideration of the incidence of cancers around the nuclear installations at Sellafield and Dounreay

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FOREWORD

i The Committee on Medical Aspects of Radiation in the Environment (COMARE) was established in November 1985 in response to the final recommendation of the report of the Independent Advisory Group chaired by Sir Douglas Black (Black, 1984). The terms of reference for COMARE are:

> 'to assess and advise government and the devolved administrations on the health effects of natural and man-made radiation and to assess the adequacy of the available data and the need for further research'

ii In 30 years of providing advice to government and the devolved administrations, COMARE has published 16 major reports (see Appendix C), in addition to numerous other statements and documents. These are mainly related to exposure to naturally occurring radionuclides, such as radon and its progeny, or to man-made radiation, usually a result of operations at major nuclear installations, although topics such as risks from sunbeds and health implications from CT scans have also been considered.

iii The report of the Independent Advisory Group chaired by Sir Douglas Black (also known as the 'Black Advisory Group') (Black, 1984) concluded that there was a raised incidence of leukaemia in young people living in the village of Seascale, adjacent to the Sellafield nuclear site in northwest England. Since publication of this report, numerous studies and reports on the possible risks of childhood leukaemia (and other cancers) in the vicinity of nuclear sites have been published. Some studies have observed positive associations between the risk of childhood leukaemia and proximity to a nuclear site, but only a few of these have been statistically significant and no conclusive evidence has been obtained to determine whether living near a nuclear installation might increase the risk of childhood leukaemia. Previous detailed critical reviews by COMARE and others have concluded that the radiation doses arising from the operation of nuclear installations are not nearly high enough to cause increases in childhood leukaemia (COMARE, 1988, 1989, 1996). There is growing epidemiological evidence that childhood leukaemia is linked to infections; two major hypotheses are that childhood leukaemia is either a rare response to a specific common infection (or infections) (Kinlen, 2011) or a rare response to general exposure to infectious agents that is enhanced by delayed exposure (Greaves, 2006). However, the biological mechanisms underlying these hypotheses remain the subject of considerable scientific debate.

iv The first COMARE report examined the implications of further information on radioactive discharges from Sellafield, including the revision of releases of irradiated uranium oxide particles in the 1950s, which had not been available to the Black Advisory Group (COMARE, 1986). COMARE concluded that this additional information did not change the essential conclusions of the report of the Black Advisory Group.

v The second COMARE report investigated the incidence of leukaemia in young people living near to the Dounreay nuclear establishment in Caithness, Scotland (COMARE, 1988). Evidence was found of an increased incidence of leukaemia in young people in the area compared with expected levels nationally. Although the conventional dose and risk estimates suggested that radioactive discharges could not be responsible, the raised incidence of leukaemia at both Sellafield and Dounreay tended to support the hypothesis that some feature of these two plants led to an increased risk of leukaemia in young people living in the surrounding area. The report also considered other possible explanations and recommended further investigations.

vi The third COMARE report considered suggestions of an increased incidence of childhood cancer near the Atomic Weapons Research Establishment at Aldermaston and the nearby Royal Ordnance Factory at Burghfield, in Berkshire, England (COMARE, 1989). A small, but statistically significant, increase in registration rates of childhood leukaemia and other childhood cancers was found in the vicinity of the two sites. COMARE judged that the doses from the radioactive discharges were far too low to account for the observed increase in the incidence of childhood cancer. A number of possible explanations for the findings were considered, including other mechanisms by which radiation could be involved. However, there was insufficient evidence to point to any one explanation, although the possibility remained that a combination of factors might be involved. Further investigations were recommended. The third report concluded by saying that the distribution of cases of childhood leukaemia or other childhood cancers around nuclear installations could not be seen in proper context in the absence of comparable information about the pattern of incidence rates throughout the UK. The report recommended that further work be carried out to determine the national geographical pattern of the distribution of childhood cancer.

vii The fourth COMARE report (COMARE, 1996) was a review of the dosimetric, epidemiological and other scientific data relating to the Sellafield site and the village of Seascale, together with other relevant advances in scientific knowledge, that had become available since publication of the report of the Black Advisory Group in 1984. The review was undertaken in response to a request by government in September 1989. The report concluded that there was good evidence for a continuing, significantly elevated level of all malignancies in young people (0–24 years of age) in Seascale throughout the period considered by the Black Advisory Group (1963–1983) and by COMARE's subsequent analysis (1984–1992), covering a total of three decades. The excess of all cancers was primarily due to an excess of lymphoid leukaemia and the related malignant disease, non-Hodgkin lymphoma (NHL). On the basis of current knowledge at the time, it was considered that the estimate of the radiation doses to the Seascale population, from both routine and accidental discharges from Sellafield, was too small to account for the observed excess of cases of leukaemia and NHL. A number of other hypotheses involving radiation exposure, in particular preconceptional exposure of parents, and also those involving exposures to chemicals and infectious agents, either singly or in combination, were considered in the report. COMARE concluded that no single factor could account for the excess of leukaemia and NHL, but that a mechanism involving infection may be a significant factor affecting the risk of leukaemia and NHL in young people in Seascale.

viii The sixth COMARE report summarised the work undertaken since 1995 and up until October 1998, to locate the source of the radioactive particles found in the general environment around the Dounreay nuclear establishment and reconsider the possible health implications of encountering these particles

(COMARE, 1999). COMARE considered whether ingestion of these particles could be associated with the previously reported excess of leukaemia and NHL in young people living in Thurso, Caithness. If individuals were to ingest particles with activities at the top of the range of those particles already found on the Dounreay foreshore, very serious acute radiation effects would occur. However, at that time very few particles had been found on the publicly accessible beach at Sandside Bay and all of them were of low activity. The report concluded that an implausibly large number of these particles would have needed to be ingested to have given rise to the known level of childhood leukaemia in the area around Dounreay. A recommendation was made for increased, regular and improved beach monitoring in the area to ensure any particles coming ashore could be found and removed.

ix The seventh COMARE report was written as part of the ongoing investigation into the Seascale cluster of cases of childhood leukaemia and other cancers between 1950 and 1991, particularly in the context of the suggestion that radiation exposure of fathers while working at Sellafield before the conception of their children increased the risk of childhood leukaemia (COMARE, 2002). The report included an extensive review of the most recent epidemiological studies of the offspring of radiation workers and also the latest laboratory and genetic research relating to the possible biological mechanisms that might explain any observed carcinogenic effects in the offspring of irradiated parents.

x The tenth COMARE report reviewed the evidence relating to childhood cancers in the vicinity of licensed nuclear sites (power stations and other nuclear installations) in Great Britain (COMARE, 2005). There was no evidence of statistically significant excess numbers of cases in any local 25 km area around any of the nuclear power stations. Around other nuclear installations the analysis showed increasing trends of leukaemia and NHL incidence in children living at decreasing distances from Dounreay and Sellafield; the results were consistent with previously published studies.

xi The eleventh COMARE report provided an overview of the geographical patterns of variation in the incidence of different types of childhood cancer, and the relation of these to socio-demographic factors (COMARE, 2006). Childhood leukaemia and other types of childhood cancers occurred unevenly within the population of Great Britain. There are a variety of incidence rates in different geographical and social circumstances and these differ more than would be expected from chance variations. The analyses in this report were carried out on the largest dataset of childhood cancer cases ever compiled anywhere in the world at that time, giving considerable confidence in the robustness of the results. The report recommended that the incidence of childhood leukaemia and other cancers in the vicinities of Sellafield and Dounreay should be kept under surveillance and periodic review.

xii The fourteenth COMARE report considered the incidence of childhood leukaemia around nuclear power plants, with a review of publications on installations around the world (COMARE, 2011). For the British study in this report, the analysis showed no significant evidence of an association between leukaemia risk and proximity to nuclear power plants in Great Britain in children less than 5 years of age. It was, therefore, possible to conclude that, in spite of its limitations, the geographical analysis of British data was suggestive of a risk estimate for childhood leukaemia associated with proximity to an nuclear power plant that is extremely small, if not actually zero. That study did not include data from nuclear installations such as Sellafield and Dounreay as these were to be addressed in the current report.

xiii The primary aim of this, the seventeenth COMARE report, is to provide the Department of Health and the Scottish Government with a review of the incidence of childhood leukaemia and other cancers around the Sellafield and Dounreay nuclear installations, bringing the analyses up to date. In addition to the incidence of childhood cancers in the 0–14 years age group, this report includes the incidence of cancers in the 15–24 years age group and adult cancers for the two sites.

CHAPTER 1

INTRODUCTION

1.1 Interest in possible health effects among the general public living around nuclear installations due to low level radiation exposure developed in the early 1980s, although there had been previous studies which were reviewed by Tokuhata and Smith (1981). The issue was brought into the public spotlight in the UK by a television report in 1983 of an excess of childhood leukaemia cases in the coastal village of Seascale, adjacent to the Sellafield nuclear installation in Cumbria, England, which prompted the investigation by the Independent Advisory Group, chaired by Sir Douglas Black (the 'Black Advisory Group') (Black, 1984) and the first COMARE report (COMARE, 1986). Further reports of excess cases of childhood leukaemia in the vicinity of nuclear installations followed, notably the raised incidence around Dounreay in northern Scotland and near Aldermaston and Burghfield in Berkshire, England, which were the subject of the second and third COMARE reports, respectively (COMARE, 1988, 1989). The fifth recommendation from the eleventh COMARE report requested that surveillance and review processes be updated specifically for Sellafield and Dounreay, forming the basis of this report (COMARE, 2006). A similar update was not requested for Aldermaston and Burghfield as these sites were included in the analyses in the tenth COMARE report, and the excesses were not as noteworthy as those associated with Sellafield and Dounreay (COMARE, 2005).

1.2 In its second report, COMARE concluded that the finding of a raised incidence of leukaemia in young people living around Dounreay, in combination with the excess of cases of leukaemia in young people living in Seascale, made it less likely that chance was the explanation for the excesses, and that an intensified programme of investigation was warranted. In its fourth report, COMARE found that the increased incidence of leukaemia and non-Hodgkin lymphoma (NHL) among young people in Seascale had continued beyond the period considered by the Black Advisory Group, and recommended that the incidence of childhood leukaemia and other cancers in the vicinity of Sellafield should be kept under surveillance and periodic review (COMARE, 1996). In its tenth report, COMARE reiterated its view that the Sellafield and Dounreay excesses were unlikely to be due to chance alone (COMARE, 2005).

Figure 1.1 Locations of Sellafield and Dounreay in Great Britain (contains Ordnance Survey data © Crown copyright and database right 2015)

Sellafield

1.3 Sellafield was the first site where it had been suggested that radioactive discharges were associated with local levels of childhood cancer. This hypothesis was examined by the Black Advisory Group in 1984 and by COMARE in 1986 and 1996 with no conclusive evidence of an association being found between radiation doses received from discharges and childhood cancer incidence (Black, 1984; COMARE, 1986, 1996). Historically, Sellafield is the nuclear site in the UK with the largest of all radioactive discharge levels, which peaked in the 1970s and have since declined to the much lower current levels.

Seascale cluster

1.4 While the tenth and fourteenth COMARE reports found that there is, in general, no evidence for an excess incidence of cancer in young people living in areas around nuclear installations in Great Britain, there is evidence that there is a highly unusual excess of leukaemia and NHL cases among children and young adults in Seascale, a coastal village about 3 km to the south of the Sellafield nuclear installation in west Cumbria, England.

Figure 1.2 Sellafield locality (contains Ordnance Survey data © Crown copyright and database right 2015)

1.5 In the seventh COMARE report, 21 cases of cancer were identified in young people who were diagnosed with, or died of, cancer during 1954–2001 while 0–24 years of age and who were either born and/or diagnosed while resident in Seascale (COMARE, 2002). Seven of these were of acute lymphoblastic leukaemia (ALL), which in the eleventh report showed general 'spatial' and 'space–time' clustering throughout Great Britain to an extent that was greater than could be expected by chance alone. Of the remaining cases, two were of myeloid leukaemia and five of non-Hodgkin lymphoma (NHL), which the second report noted was similar in some forms to ALL, so 14 (two-thirds) of the 21 cases of cancer were cases of leukaemia and NHL. The findings of the eleventh report are consistent with the proposition that at least part of the Seascale excess can be attributed to risk factors widespread in Great Britain and which tend to show some degree of clustering. Of the other cancers associated with Seascale, some (such as Wilms' tumour) have also exhibited a tendency to spatially cluster at the national level (COMARE, 2006).

1.6 As discussed in the fourth and seventh reports, there is, at present, no generally accepted explanation for the increased incidence of leukaemia and NHL among young people in Seascale. It seems most unlikely to be simply attributable to exposure to radiation from radioactive discharges because the doses are so low, and it has been suggested that the excess risk may be largely or wholly due to the effects of an infectious agent (or agents) introduced by exceptional rural population mixing.

1.7 COMARE re-examined the radiation-induced excess risk from radioactive discharges, in considerable depth, in its fourth report. All the known pathways of exposure to man from both external and internal radiation sources, including sea-to-land transfer, were examined. The risks to different stages of human development from the embryo and foetus to the adult, and also the risks to different tissues, were considered. The analyses incorporated as many of the variables that could introduce uncertainty in the dose calculations for which data were available. The possibility of radioactive objects and particles discovered on the beaches of, and around, Seascale contributing to the exposure of, and risks to, the public was also considered, and is covered in more detail in Chapter 5 (paragraph 5.7).

1.8 In its fourth report, COMARE also looked at other possible hypotheses that could explain the excess: these ranged from an investigation of the non-radioactive chemicals used and discharged from the Sellafield site, to the presence of untreated sewage on beaches. It was concluded that the excess of childhood leukaemia and NHL in the Sellafield area (largely located in the local village of Seascale), when examined in the context of the national distribution of these diseases, was highly unusual, that it persisted for some tens of years and that it was unlikely to be due solely to chance. However, COMARE found that, given the present state of knowledge, no one factor could account for the observed increase in the level of disease. It could not be ruled out that infection, at least in part, had some causal association with the excess and some interaction between different factors could also not be excluded.

Dounreay

1.9 During its enquiry concerning the area around Sellafield, the Black Advisory Group had requested information about the incidence of childhood leukaemia around Dounreay, the only other nuclear installation in the UK where large-scale nuclear fuel reprocessing was carried out.

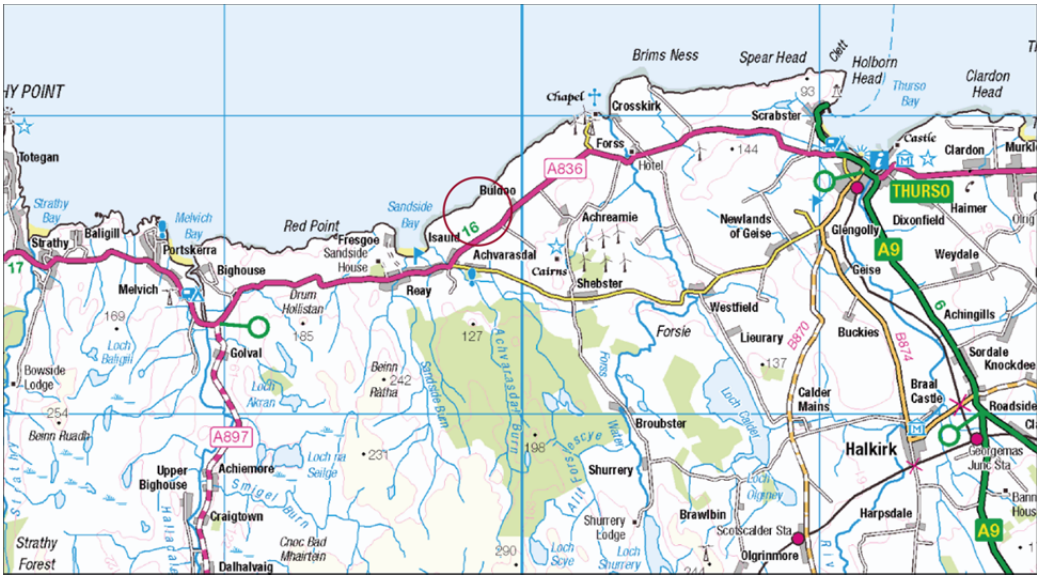

Figure 1.3 Dounreay locality (contains Ordnance Survey data © Crown copyright and database right 2015)

1.10 At that time, the available data did not suggest any evidence of an increase in leukaemia around this site. However, a further analysis (Heasman et al, 1986), prompted by the public enquiry into a proposed new reprocessing plant, suggested an elevated incidence of leukaemia in young people in the nearby town of Thurso.

1.11 COMARE was asked to investigate and report, which resulted in its second report. Six cases of leukaemia among people aged up to 25 years living within 25 km of Dounreay during the period 1968–1984 were identified, which was about double the number expected from Scottish national rates, but the excess was not statistically significant. However, the cases were concentrated within 12.5 km of Dounreay during 1979–1984. The radioactive discharge levels from the site were considerably lower than those from Sellafield and there was no excess of other types of childhood cancer in the area.

1.12 Some of the conclusions on the possible health effects from radioactive materials released from the Dounreay site were re-examined when radioactive particles were found on the Dounreay foreshore (COMARE/RWMAC, 1995) and on a local beach, Sandside Bay (COMARE, 1999). No causal link between the levels of radioactivity in the general environment and that of childhood cancer in the local area could be found. A further study (Black et al, 1994) showed that there was an increased level of leukaemia in this area, of borderline significance, in the years 1985–1991.

Cancer incidence

1.13 Health statistics make use of incidence and mortality data associated with disease. Broadly, the crude incidence rate is obtained by dividing the number of people newly diagnosed with the disease, in a particular period, by the total population at risk over the same period, while the crude mortality rate is obtained by dividing the number of people dying of that disease in a particular period by the total population at risk over the same period. These crude incidence and mortality rates would usually be standardised for age, sex and, where appropriate, ethnicity and socioeconomic status when carrying out comparisons between different populations, to account for different background rates in these groups.

1.14 The incidence rate can be affected in the short term by earlier diagnosis, brought about by improvements in diagnostic testing or the introduction of screening programmes. An increase in incidence may also be due, in part, to a heightened awareness by clinicians of a condition, or the enhanced detection of a disease that lies dormant for many years. Incidence data rely upon complete and accurate registration of cases through, for example, cancer registries.

1.15 The mortality rate can be affected by a number of factors: a reduction in the mortality rate could be due to a decrease in incidence, an improvement in the cure rate or an increase in survival time. The last could be due to earlier diagnosis or to new (non-curative) treatment options and will yield only a short-term improvement in the mortality rate. These factors render longer-term comparisons difficult, given the substantial improvement in cancer treatment and survival in recent times.

1.16 The cure rate for childhood leukaemia has improved markedly over the period under study; data from one large US children's hospital (Inaba et al, 2013) show a 5-year survival of 21±4% in 1962–1966 improving to 74±2% in 1979–1983 and to 91±1% in 2000–2007, and so the mortality data will not include a large proportion of more recent incident cases. In addition, since leukaemia is a rare disease, the numbers of cases entered into the analysis using mortality data would be even smaller, leading to lower statistical power.

1.17 The survival rate for papillary thyroid cancer that is most commonly associated with radiation exposure is higher than that for other types (Nikiforov and Gnepp, 1994); survival rates are also better for younger ages. Due to improved diagnostic techniques and earlier detection, survival rates for thyroid cancer have improved over the last 50 years (La Vecchia et al, 2015).

1.18 For these reasons, incidence data have been used in this report, rather than mortality as in the fourth COMARE report.

Seventeenth report

1.19 This seventeenth COMARE report is the outcome of the review based on the recommendations from the fourth and eleventh COMARE reports. The fourth report also recommended that it would be of interest to see if any cancer excess now occurred in age groups older than 24 years of age. This would require consideration being given as to whether or not the cancer experience of cohorts of people who have lived or continue to live in Seascale could be brought up to date to answer such questions. The remit of this report has been expanded to include adult cancers and also to consider thyroid cancers specifically, particularly in relation to discharges of iodine-131.

CHAPTER 2

REVIEW OF PREVIOUS COMARE REPORTS ON SELLAFIELD AND DOUNREAY AND THEIR RECOMMENDATIONS

Report of the Independent Advisory Group (Chairman: Sir Douglas Black)

Investigation of the possible increased incidence of cancer in West Cumbria

2.1 Following the Yorkshire Television programme *Windscale – the Nuclear Laundry*, shown nationally on ITV on 1 November 1983, Sir Douglas Black was asked by the Minister of Health to lead an independent expert inquiry into the possible increased incidence of cancer in the area adjacent to the Sellafield site. The terms of reference for the Independent Advisory Group (the Black Advisory Group) were:

> To look into the recently published claims of an increased incidence of cancer in the vicinity of the Sellafield site:
>
> (i) examine the evidence concerning the alleged cluster of cancer cases in the village of Seascale;
>
> (ii) consider the need for further research;
>
> (iii) make recommendations.

2.2 The Black Advisory Group divided its remit into three tasks:

> (i) establishing the incidence of cancer in the area adjacent to Sellafield and comparing it with the incidence of cancer in other areas in the UK as a whole and in Cumbria;
>
> (ii) considering the available data on radiation exposure in the area adjacent to Sellafield and the evidence relating radiation exposure to cancer, thus assessing the likelihood that any radiation exposure could have caused any increased incidence of cancer detected in the area;
>
> (iii) assessing other possible significant risk factors.

2.3 The Group met 16 times in all and, in addition, there were several meetings of subgroups to consider specific points in greater detail. The Group visited Cumbria in January 1984, viewing the Sellafield site and speaking to local medical staff and to Seascale's general practitioners. Subsequently, there were several visits to the area by individual members and by the Secretariat for consultation on particular questions raised as work progressed.

2.4 The then National Radiological Protection Board (NRPB)* prepared three reports following discussions with the Group and these provided the scientific basis of much of the data on which the conclusions on radiation exposure were based.

2.5 The report of the Black Advisory Group (the 'Black report') was published in July 1984 (Black, 1984).

* The NRPB was subsequently incorporated into the Health Protection Agency (HPA). On 1 April 2013 the HPA was abolished and its functions transferred to Public Health England.

2.6 The number of children who had developed leukaemia in a 30-year period in Seascale was less than 10 – a relatively small number. Due to uncertainty about the size of the population from which they were drawn, the true incidence of leukaemia could not be determined precisely. Since the final estimate of health risk had to be based on data which included those used to raise the hypothesis, assessment of the significance of the observed incidence of leukaemia proved difficult. However, taking west Cumbria as a whole, mortality from childhood cancer was near to the national average, particularly for cancers other than leukaemia, but this finding did not exclude local pockets of high incidence.

2.7 In the area covered by the then Northern Children's Tumour Registry, which contained 675 census wards in northern England, Seascale had the third highest lymphoid malignancy (acute lymphoblastic leukaemia, ALL, and the lymphomas) rate during 1968–1982 for children less than 15 years of age (this excess being entirely due to an increased incidence of ALL, based on four cases); Seascale ward was ranked first by cumulative Poisson probability. Millom Rural District (south of Sellafield and which includes Seascale) had the second highest leukaemia mortality rate for young people less than 25 years of age among 152 comparably-sized rural districts in England and Wales during 1968–1978. The equivalent leukaemia mortality rate in Ennerdale Rural District (to the north of Seascale and containing Sellafield) was not unusual. Mortality rates for other diseases in the local population, either for children or for adults, were not unusual. In particular, the overall mortality rate for young people less than 25 years of age in Millom Rural District was within normal limits.

2.8 The Black Advisory Group noted that it was impossible to establish for certain the situation with regard to environmental levels of radiation around Sellafield 20 or 30 years before, and that it was impossible to know the actual doses received by those children subsequently developing leukaemia. In addition, the possibility of unplanned discharges which were not detected by the monitoring programmes and yet delivered a significant dose to humans by an unsuspected route could not be excluded completely.

2.9 Subject to these uncertainties, the NRPB provided the Group with a 'best estimate' of the average radiation dose to the red bone marrow (RBM), considered to be the relevant target tissue for leukaemia, received by a model population of young people in Seascale. Using the then latest available radiation-induced leukaemia risk models, the NRPB concluded that the dose to the RBM of children in Seascale was less than 1% of the dose required to account for the observed excess mortality from childhood leukaemia in the village. The Group also made a 'worst-case' assumption that leukaemia in under 20 year olds in England and Wales was entirely due to the dose of background radiation to the RBM, and on this basis estimated the risk from low dose rate radiation exposure. Using this highly conservative risk estimate and a simple relationship between dose and effect, the Group calculated the number of additional deaths from leukaemia in under 20 year olds in Seascale that might be attributable to the additional dose their RBM received from discharges from the Sellafield site up to 1980. The number of deaths from leukaemia thus calculated was insufficient, by a factor of 40, to account for the deaths actually observed in Seascale, being around 20% of the number expected from background radiation.

2.10 The Group reached the conclusion that the calculations did not support the view that the radiation released from Sellafield was responsible for the observed incidence of leukaemia in Seascale and its neighbourhood. However, it stressed the unavoidable uncertainties on dose in this situation, noting that the

model used did not exclude other possibilities.

2.11 A number of recommendations were made, including:

(i) a study should be carried out on the records of those cases of leukaemia and lymphoma which had been diagnosed among young people up to the age of 25 years, resident in west Cumbria. These cases should be compared with suitable controls in respect of factors that could be relevant to the development of leukaemia and lymphoma;

(ii) a study should be carried out of the records on all children born since 1950 to mothers resident in Seascale at the time of birth, the main purpose being to examine cancer incidence and mortality among those children, including cases which might have occurred after moving from Seascale;

(iii) a study should be considered of the records of children who had attended schools in the area;

(iv) more attention should be concentrated on measuring doses of radiation actually received by members of the public in west Cumbria and in other relevant areas, including control areas;

(v) reviews of the site discharge authorisations should take place more frequently;

(vi) greater emphasis should be placed on the collection and consideration of relevant epidemiological data and any other human data relevant to the possible health consequences of discharges;

(vii) there should be formal consultation by the authorising department with the health departments and the NRPB on the possible health consequences of discharges;

(viii) the responsibility for monitoring, and for interpretation of the results of monitoring, this potentially serious environmental pollutant should be more clearly defined by government; these results of monitoring should be considered in their entirety on a regular basis by a designated body with significant health representation, thus enabling decisions on action with regard to the control of permitted discharges to take account of all relevant factors.

First COMARE report

The implications of the new data on the releases from Sellafield in the 1950s for the conclusions of the Report on the Investigation of the Possible Increased Incidence of Cancer in West Cumbria

2.12 As a result of the final recommendation of the Black report (Black, 1984), the Committee on Medical Aspects of Radiation in the Environment (COMARE) was established in 1985.

2.13 The estimation of doses and the associated risks carried out by the NRPB and detailed in the Black report were based on monitoring and discharge information supplied by British Nuclear Fuels Limited (BNFL). Subsequently, Dr Jakeman, a former United Kingdom Atomic Energy Authority (UKAEA) employee, came forward with information to suggest that discharges of irradiated uranium oxide particles from the chimneys of the Windscale piles in 1954/55 had been underestimated. As a consequence, BNFL undertook a major review of discharge records.

2.14 Details concerning about 20 items of new or reassessed discharge data were identified and the first COMARE report (COMARE, 1986) considered the implications of this information.

2.15 The original data provided to the Black Advisory Group suggested that some 440 g of irradiated uranium oxide had been released from the stacks of the Windscale piles during 1954 and 1955. Following discussion between BNFL, UKAEA, NRPB and Dr Jakeman, Dr Jakeman's estimate of 20 kg was accepted by BNFL as more likely to be correct. He stressed that a range of 10–50 kg was possible.

2.16 The NRPB was asked to review the effect of the new discharge data on its calculations (Stather et al, 1986). While most of the information resulted in only minor modifications of the doses, the above release of irradiated fuel particles, changes to the argon-41 discharges for the Windscale piles and a plutonium release in 1952 did have more significant effects on the estimates of doses to the RBM. For those born in Seascale between 1950 and 1954 and resident until 1970, the calculated RBM dose doubled; smaller increases were calculated for other cohorts. The discharges review also revealed an increased discharge of iodine-131 during the first irradiated fuel reprocessing campaign in 1952 due to the dissolution of 'short-cooled' fuel still containing substantial quantities of this radioisotope of iodine, although this had little impact on the RBM dose.

2.17 COMARE concluded that the increased doses were still well below (around 1%) those that would have readily explained the observed cases of leukaemia and so confirmed that the substance and essential conclusions of the Black Advisory Group's report remained unchanged.

2.18 Concerns over the manner in which the additional data had come to light were noted and it was stressed that the adequacy of the monitoring programme should be kept under review.

Second COMARE report

Investigation of the possible increased incidence of leukaemia in young people near the Dounreay nuclear establishment, Caithness, Scotland

Leukaemia incidence

2.19 In the second COMARE report, the incidence of leukaemia among young people aged 0–24 years living in the vicinity of the Dounreay nuclear establishment was examined using various areas and time periods (COMARE, 1988).

2.20 Over the full time period (1968–1984) for which incidence data were available, and within 25 km of Dounreay, six cases of leukaemia were registered among young people aged 0–24 years. This was twice the number expected on the basis of Scottish national rates, but the difference did not achieve conventional statistical significance ($p = 0.079$). Four of these cases were resident at diagnosis in the western part of Thurso, about 12.5 km from Dounreay and the only sizeable centre of population near Dounreay. Five of the six cases were diagnosed in the 0–14 year age group usually used in the study of childhood leukaemia – around twice the expected number of cases ($p = 0.073$).

2.21 Other comparisons were made varying the geographical area. The results were treated with caution because the boundaries fell close to the homes of the cases, transecting the town of Thurso. Nevertheless, some of the results showed a nominally statistically significant excess of leukaemia.

2.22 All six cases were registered in the final six years (1979–1984) of the study period and this number of cases was predicted to occur only very infrequently by chance. These results were unexpected and were regarded with caution, but they were a notable observation.

2.23 During the full period of study (1968–1984) there were two additional cases of acute lymphoblastic leukaemia (ALL) that were inappropriately registered as non-Hodgkin lymphoma (NHL). These two cases were not included in the analysis of leukaemia incidence because comparable data were not available for the rest of Scotland. However, when leukaemia and NHL were considered together, there was a significant excess incidence ($p = 0.039$) in the area within 25 km of Dounreay during the full 17-year period, and for the 0–14 year age group, the 7 observed cases compared with 2.65 cases expected ($p = 0.019$). COMARE was also aware of another case of NHL diagnosed outside the study period that should also have been more appropriately classified as ALL. Given this uncertainty about the classification of particular forms of childhood lymphoid leukaemia and NHL, particularly when historical data are being considered, COMARE recommended that studies of the incidence of childhood leukaemia should also include the incidence of NHL, if possible.

2.24 Taking the pattern of results as a whole, COMARE concluded that there was clear evidence of an excess incidence of leukaemia among young people living in the vicinity of Dounreay which was not an artefact of the selection of geographical or temporal boundaries. Given that a raised incidence of this particular disease at these ages had already been reported near Sellafield, COMARE considered that an excess incidence of leukaemia in young people in the areas around both sites made it less likely that these were chance occurrences.

Environmental radiation

2.25 To assess the possible relationship between the raised leukaemia incidence and the Dounreay nuclear establishment, doses arising from radioactive material released from Dounreay were considered. These took into account information on discharges, accidental releases and environmental measurements.

Authorised discharges

2.26 Authorised discharges of radioactivity from Dounreay were low in comparison with Sellafield. The airborne discharges were below those from most nuclear power generating installations and between one and two orders of magnitude below those from Sellafield. However, some of the radionuclides discharged to both air and sea were actinides emitting high linear energy transfer (LET) alpha radiation and these were discharged from both Sellafield and Dounreay in a higher proportion than from nuclear power generating plants.

2.27 Recorded discharges of total radioactivity were primarily to the sea, with lower quantities discharged to the air.

2.28 The estimates of activity in the discharges used when measurements were not available were sufficiently cautious to suggest that the totals reported for the authorised discharges were probably overestimated.

Accidental releases

2.29 The UKAEA provided details of accidental releases containing low levels of radioactivity. A few were reflected in transient slight increases in environmental levels. The total activity from recorded accidental releases added only minimally to total emissions.

Environmental monitoring

2.30 Marine monitoring, including radioactivity measurements in seafood, around Dounreay has been carried out by the Ministry of Agriculture, Fisheries and Food (MAFF)[*] since 1958. The levels were below those close to Sellafield, although individual radionuclides varied. The levels measured were below those estimated by the NRPB at the time, on the basis of recorded discharges, confirming that the dose was overestimated.

2.31 Air monitoring was introduced in 1980 and analyses confirmed the reported levels of discharges to air from Dounreay. These data supported the NRPB modelling assumptions. Monitoring was not in place for earlier years to confirm discharge levels, and none was carried out in the local centre of population, Thurso. Passive deposition collectors were, however, in place before 1970, but these did not provide quantitative data despite being sensitive to changes in environmental radioactivity levels.

2.32 Even with the lack of land-based environmental monitoring data to confirm discharges to air throughout the period, there is no real likelihood that discharges to air would have been sufficiently higher than those reported, to materially alter the assessment of the final dose and risk.

Estimates of radiation dose based on known radiation discharges and estimates of risk to the general population

2.33 Estimates of radiation dose to young people resident in Thurso were made by the NRPB using conventional dose modelling. Reported discharges for Dounreay contributed only a small proportion of the total estimated dose from all radiation sources.

2.34 Estimates of dose to human tissues were derived from animal models and, therefore, there were uncertainties concerning the estimates of dose to the foetus and infant.

2.35 Estimates made by the NRPB of the risk of leukaemia in Thurso attributable to the total radiation dose (from all sources) and employing the NRPB risk coefficients, suggested that the total risk of radiation-induced leukaemia in an estimated 4550 young people resident in Thurso between 1950 and 1984 will have been 0.34 case and the risk specifically attributable to discharges very small indeed (0.005 case).

2.36 There were also uncertainties associated with estimating the risk of radiation-induced leukaemia in young people. The human evidence available at that time indicated that exposure *in utero* was important for the development of childhood leukaemia. The estimates given in the second COMARE report demonstrated that the risk arising from radiation exposure of the foetus and infant from Dounreay discharges would not account for the leukaemia excess observed. However, the data concerning the risk of leukaemia following *in utero* and infant exposures were limited. In addition, these estimates were confined to low LET radiation exposures, whereas high LET radiation contributed about 20% of the estimated dose from Dounreay discharges. Although there were differences in the radionuclides present, high LET radiation also contributed to the dose from natural sources.

2.37 A comparison of the patterns of leukaemia incidence and doses from nuclear weapons testing fallout or background radiation suggested that errors in general risk factors for both low LET and high LET radiations were unlikely to be so high that doses from discharges could account for the leukaemia excess. However, there was limited knowledge about the physical and chemical forms

[*] MAFF was was dissolved in 2002, at which point its responsibilities were transferred into the Department for Environment, Food and Rural Affairs (Defra).

of the radionuclides concerned, their distribution in the body and the target tissues for leukaemogenesis, particularly for children. Thus, the possibility could not be excluded that some aspect of discharges could generate higher doses than the estimates published in the second COMARE report.

Other factors related to Dounreay

2.38 Hazardous chemicals were used at the plant only on a small scale under normal industrial controls, and there was no evidence that these could be responsible for the cases of childhood leukaemia in the neighbourhood.

Chance

2.39 The raised incidence of leukaemia in young people close to Dounreay could have been a 'chance' occurrence in the sense of the individual cases having no increased risk. However, it is less likely to be by chance that such an occurrence would take place around both Dounreay and Sellafield, the two installations in the UK involved in nuclear fuel reprocessing.

Final conclusion

2.40 There was evidence of a raised incidence of leukaemia among young people living in the vicinity of Dounreay. There were differences between Dounreay and Sellafield, principally a difference of one or more orders of magnitude in discharge levels. However, the evidence of a raised incidence of leukaemia near Dounreay, taken in conjunction with that relating to the area around Sellafield, tended to support the hypothesis that some feature of the nuclear plants leads to an increased risk of leukaemia in young people living in the vicinity of those plants. Conventional dose and risk estimates at that time suggested that neither authorised nor accidental discharges could have been responsible. There were, however, uncertainties about dose and risk calculations, especially with respect to exposure of the foetus and infant, high LET emissions and prolonged low level exposure.

2.41 COMARE considered a number of alternative explanations, including other mechanisms by which the authorised discharges could have been implicated, the possibility that parental occupational exposure could have been relevant and the possibility that factors other than radiation could have been important. However, in COMARE's view, the evidence did not point to any particular explanation and, therefore, it was suggested that all possible explanations needed to be investigated further. Although chance could not be entirely dismissed as an explanation of the raised incidence of childhood leukaemia in the vicinity of Dounreay, it was considered that it was less likely than when Sellafield was considered in isolation.

Third COMARE report

Report on the incidence of childhood cancer in the West Berkshire and North Hampshire area, in which are situated the Atomic Weapons Research Establishment, Aldermaston, and the Royal Ordnance Factory, Burghfield

2.42 Although this COMARE report did not address the risk of cancer near Sellafield or Dounreay specifically, it was the third in a series of COMARE investigations of reports of an excess incidence of childhood leukaemia in the vicinity of individual (or a few neighbouring) nuclear installations in Great Britain (COMARE, 1989). Aldermaston and Burghfield are nuclear weapons installations in west Berkshire, England, situated about 8 km apart, neither of which carried out any reprocessing activities and the discharges from which were considerably lower than those of Sellafield and Dounreay. COMARE inferred that a statistically significant excess of childhood leukaemia incidence had occurred within 10 km of either Aldermaston or Burghfield, but that the radioactive discharges from the installations were "far too low to account for the observed increase in childhood cancer incidence in the area".

2.43 The third report reached the important conclusion "that the distribution of cases of childhood leukaemia, or other childhood cancer, around individual nuclear installations cannot be seen in a proper context in the absence of comparable information about the general pattern throughout the rest of the UK". Consequently, COMARE recommended that "high priority" be given to the completion of an accurate, detailed and complete national registry of childhood cancer, and that when this was available studies of the geographical distribution of childhood cancer incidence on a nationwide basis be conducted to "provide essential information" on the background spatial pattern of cases. Only then should a systematic study of childhood cancer incidence in the vicinity of nuclear installations be carried out.

Fourth COMARE report

The incidence of cancer and leukaemia in young people in the vicinity of the Sellafield site, West Cumbria: further studies and an update of the situation since the publication of the report of the Black Advisory Group in 1984

2.44 The fourth COMARE report reviewed the epidemiological, dosimetric and other scientific data relating to Seascale and the Sellafield nuclear site that became available after the publication of the Black report and the first COMARE report. The fourth report's main aim was to draw conclusions about the main advances in scientific knowledge since the Black report and to clarify progress made and where uncertainties remained (COMARE, 1996).

Epidemiology

2.45 Cancer incidence was examined according to diagnostic groups among children aged 0–14 years living in the vicinity of Sellafield during 1963–1992, and among young adults aged 15–24 years during 1969–1992. These periods were selected because it was understood that the cancer incidence data for Cumbria were effectively complete for these age groups for these two periods. The whole time frame (1963–1992) was subdivided into the period examined by the Black Advisory Group (1963–1983) and the subsequent period (1984–1992) to determine whether the raised incidence of malignant disease identified by the Black Advisory Group had persisted.

2.46 For the period 1963–1983, the conclusions of the Black report were confirmed: an increased incidence of cancer among 0–24 year olds resident in Seascale, which was due to the excess of cases of leukaemia and NHL.

2.47 The analysis for the 'post-Black' period 1984–1992 examined whether this excess incidence of cancers among young people in Seascale had continued into later years. There was a significant excess of cancer incidence in this period, which was due primarily to a significant excess of cases of leukaemia and NHL, so strengthening the original Black report conclusion.

2.48 The overall conclusion was that there was good evidence for a continued elevated level of all malignancies in young people (0–24 years of age) throughout 1963–1983 and subsequently during 1984–1992. This was due to an excess of lymphoid leukaemia and NHL throughout the entire period 1963–1992. Although the excess in 1984–1992 was greater than that in 1963–1983, this was based on only three cases and the difference between the relative risks was not statistically significant.

2.49 The excess found among young people did not extend to the age group 25–74 years, nor did it extend to the two districts nearest to Sellafield, or to Cumbria as a whole.

Radiation risks

2.50 Both the Black report and the first COMARE report examined the risk of childhood leukaemia and other cancers in the population local to Sellafield arising from exposure to environmental radiation.

2.51 Since the mid-1980s, further data on routine and accidental discharges of radioactive materials from Sellafield had been obtained, which included a reassessment of the aerial discharges during the early years of plant operation.

2.52 At COMARE's request, and in collaboration with the committee, the NRPB carried out a complete reassessment of the doses and risks to the Seascale population in the light of further discharge data and advances in scientific knowledge.

2.53 Changes from the previous assessment for the Black investigation and the first report included:

(i) inclusion of the doses from the Chernobyl accident fallout and the discharges from the Marchon Works chemical plant in Whitehaven;

(ii) a review of BNFL discharges which revealed increases in atmospheric discharges in the early years of Sellafield site operations;

(iii) use of the most recent biokinetic and dosimetric models;

(iv) use of new models to calculate the risk of radiation-induced cancer and computing a best estimate rather than making 'worst case' assumptions.

2.54 The principal conclusions of the report are listed below:

(i) for Seascale, natural sources of radiation contributed by far the largest equivalent dose, whereas the Sellafield discharges contributed less than 9%;

(ii) in 1348 children born in Seascale between 1945 and 1992, the number of radiation-induced cases of leukaemia and NHL from all sources would be expected to be less than 1 (0.46 case). The number of other cancers expected from radiation exposure from all sources was also less than 1 (0.22 case). These estimates have to be compared with the 12 cases of leukaemia and NHL and 4 cases of other cancers observed in Seascale between 1955 and 1992;

(iii) the expected number of cases of leukaemia and NHL during 1945–1992 calculated to be attributable to Sellafield discharges was less than 0.05. Considering Sellafield discharges alone, the doses would have had to be about 200 times greater to account for the excess cases on the basis of the then current scientific knowledge. The probability of such a discrepancy was highly unlikely;

(iv) having reviewed discharge and environmental monitoring data, and having noted previous underestimations of some airborne discharges, COMARE considered that it was very improbable that the epidemiological findings could be attributed to undetected discharges, particularly of long-lived radionuclides;

(v) the habit data review suggested that radionuclide intakes were not substantially underestimated, nor had environmental pathways delivered substantially greater doses than previously estimated.;

(vi) COMARE considered the possibility that the biological effectiveness of particular types of radioactive material in particular tissues may be greater than attributed by the ICRP, but discounted the idea that relevant weighting factors had been substantially underestimated;

(vii) it was possible that uncertainties in biokinetic modelling of radionuclides could lead to higher doses than estimated, or that microdosimetric features of localisation could lead to higher doses to some cell types. However, it seemed improbable that such factors would apply uniquely to the Seascale population.

2.55 There was no evidence that other radiation exposures (such as radon or medical exposures) to Seascale residents differed substantially from those to residents elsewhere in the UK.

2.56 Despite these remaining uncertainties, the estimates of the radiation doses to the Seascale population were determined as too small to account for the observed excess of cases of leukaemia and NHL. Consequently, it seemed unlikely that Sellafield discharges were the sole cause of the excess.

2.57 Doses to the tracheobronchial lymph nodes had been considered by both the Black Advisory Group and COMARE previously, as it was known that these lymph nodes could be the sites of origin for leukaemia and lymphoma in laboratory animals and are sites which could be subject to exposure from inhaled radionuclides. For its fourth report, COMARE asked the NRPB to estimate radiation doses to the thoracic lymph tissue for the Sellafield discharges and for natural background radiation sources (Simmonds et al, 1995; COMARE, 1996).

2.58 The NRPB studies (NRPB-R276) showed that, during the early years of plant operation, the estimated doses to the thoracic lymph nodes from discharges of plutonium could have exceeded those estimated for the high LET component of natural background radiation. However, even in the most pessimistic case in 1955, the total equivalent dose to the thoracic lymph nodes from natural background radiation was greater than that estimated from plutonium. It was concluded in the fourth report that the doses involved were far too low to be a contributory factor to the Seascale cases. Based on the conclusions in the fourth report and NRPB-R276, thoracic lymph nodes doses have not been considered further in the current report.

Parental occupational factors

2.59 The fourth COMARE report considered alternative factors that could be relevant to the development of cancer in children. The report investigated the possibility that the excesses of childhood leukaemia at Sellafield and Dounreay could be related to parental occupation at the sites.

2.60 There are two mechanisms by which parental occupation could increase the risk of cancer in their offspring:

(i) preconceptional effect through irradiation of the gonads;

(ii) unrecognised pathways of exposure via workers.

Paternal preconceptional irradiation (PPI)

2.61 Gardner et al (1990b) suggested that paternal germ cell mutations from occupational exposure of the gonads to ionising radiation could increase the cancer rate in offspring sufficiently to explain the Seascale excess. Although there was a plausible genetic and mechanistic basis for a PPI effect, COMARE concluded that this risk was inconsistent with the doses received and estimates

of genetic risk.

2.62 While the principle that PPI could cause cancer in offspring is sustained by animal experimental data and human genetic determinants of cancer, consideration of mutation rates in models suggested the size of any effect was too small to explain fully the Seascale phenomenon. To explain the Seascale cluster of leukaemia and NHL cases, the PPI effect would have to be large, which was not consistent with the available evidence.

2.63 Moreover, the epidemiological data did not support the idea that PPI could explain the Seascale excess of leukaemia and NHL. The excess of cases among those diagnosed while resident in Seascale, but born elsewhere, could not be accounted for by PPI, so PPI was not sufficient to explain the Seascale cluster (Kinlen, 1993). The association between PPI and the risk of leukaemia and NHL was effectively confined to Seascale, whereas the doses of PPI for children born in Seascale formed a small proportion (<10%) of the PPI doses received by offspring of Sellafield workers, so that the effect should have been stronger in west Cumbria outside Seascale and there should have been more cases born to Sellafield workers. Further, in localities where many children were associated with PPI doses (more than in Seascale), those children affected with leukaemia and NHL were not those with PPI doses who, according to the PPI hypothesis, should have been most at risk.

Non-radiation hypotheses

Role of chemicals and infection in causing childhood leukaemia

2.64 From the evidence available at the time of the fourth COMARE report, it was concluded that population mixing was a factor in the increase in childhood leukaemia incidence described in some populations that had experienced unusual mixing of rural and urban populations. Therefore, it follows that the excess childhood leukaemia incidence in Seascale was likely to be causally associated with related demographic factors such as geographical isolation and mixing between residents who had migrated from different areas or had different patterns of exposure to infections. The evidence available at the time was not convincing that a large relative risk over three decades could be wholly attributed to population mixing.

Other possible factors existing in the locality before the establishment of the Sellafield nuclear site

2.65 Other factors unique to Seascale and its neighbourhood which might be associated with the epidemiological findings were investigated. In particular, COMARE investigated the activities in the Sellafield area prior to the start of nuclear operations at the site in 1950, and whether there was any elevated childhood cancer or leukaemia rate in Seascale at that time.

2.66 Between 1941 and 1945 two large chemical explosives factories (Royal Ordnance factories) were present near Seascale (at Sellafield and Drigg). Death certificates were analysed and no evidence of an excess of childhood cancer or leukaemia were found for those years.

2.67 It is unlikely that there was any unusual genetic predisposition to childhood cancer in the population of Seascale around 1950. It is possible to speculate that some carcinogenic factor entered the environment of Seascale about that time. It is also possible to conjecture that it is associated with the existence of the Sellafield site, perhaps with its construction, its operation or its effect on the make-up of the local population, or on their lifestyle, or with some combination of these factors. It was not possible to demonstrate such associations, or why they should be confined to the village of Seascale.

Main conclusion

2.68 There was a continuing excess of leukaemia and other cancers in 0–24 year olds in Seascale in the post-Black period 1984–1992, primarily due to an excess of acute lymphoblastic leukaemia and non-Hodgkin lymphoma (NHL).

Taken together with the results for the earlier periods 1955–1962 and 1963–1983, the data showed that there had been a continuing excess of leukaemia and NHL among young people in Seascale for four decades. Such evidence as available did not indicate any excess between 1900 and 1945. COMARE investigated possible causes of the excess in later decades and concluded that:

(i) environmental radiation exposure from releases could not account for the excess. Much work had been done to reduce the uncertainties, although some still remained;

(ii) parental occupational radiation exposure was also unlikely to account for the excess. Although there were uncertainties regarding internal radiation exposures, it was unclear how these could affect only the Seascale population;

(iii) other hypotheses regarding chemicals and infectious aetiology were considered and it was concluded that environmental chemical exposures were unlikely. A mechanism involving infection might be a factor affecting the risk of leukaemia and NHL in young people in Seascale due to high levels of population mixing.

2.69 It was concluded that the excess of leukaemia and NHL in young people in Seascale was unlikely to be due to chance alone. Various factors could affect the incidence of leukaemia and NHL, but no one factor alone could account for the increase.

Errors within the fourth report

2.70 Several errors have been noted within the fourth report since its publication. A number of these concerned the consideration by COMARE of hypotheses for the cause of the excess of childhood leukaemia and NHL in Seascale.

2.71 For the rural population mixing hypothesis, COMARE's particular focus on the parish of Seascale, the parish with the highest incidence of childhood leukaemia and NHL in the vicinity of Sellafield, for the period 1953–1983, has been criticised (Kinlen et al, 1997). It was argued by Kinlen et al (1997) that attention should have been primarily concentrated on an area around Sellafield, and that focusing upon Seascale within this area was a decision taken after knowledge of the Seascale 'cluster' of cases before 1984 had emerged. Part of this problem may have been due to the incorrect statement in the fourth report that Sellafield lies within Seascale parish, which could have encouraged attention to be concentrated upon the parish of Seascale in the erroneous belief that this also focused upon Sellafield. Under these circumstances, comparison between the excess in Seascale with other areas affected by rural population mixing was not a fair one – the area around Sellafield had an excess of childhood leukaemia and NHL that was comparable to that in other rural areas that had experienced notable population mixing and, conversely, there were parishes within these areas that had experienced similarly sized excesses to that which had been observed in Seascale.

2.72 Kinlen et al (1997) also noted that the 'post-Black' excess of leukaemia and NHL among young people in Seascale after 1983 occurred at a time when large numbers of workers had been brought into the area to meet the employment demands of the construction of the large THORP plant at Sellafield. The potential relevance of this for the population mixing hypothesis had been ignored in the fourth report.

2.73 The fourth report suggested that, if the population mixing hypothesis was correct, there should have been an excess of childhood leukaemia in Seascale during the Second World War, since many houses were built in Seascale during the war years to accommodate workers involved in the construction and operation of the ordnance factories at nearby Sellafield and Drigg. However, no case of childhood leukaemia had occurred in Seascale during the war. Kinlen et al (1997) argued that the report overlooked the small size of the village in those years, when the annual school roll showed only 41 children aged less than 15 years, so that there was very low statistical power to detect any excess risk.

2.74 Moreover, the period of construction of houses in Seascale was incorrectly stated in the fourth report as being during the Second World War. In fact, few houses were built in Seascale during the war years, with the marked expansion of the village occurring with the construction of 180 houses during 1949 for the nuclear operations at Sellafield (Kinlen, 2006). This mistake had arisen because a report written in 1949 referring to these 180 houses under construction in Seascale was erroneously believed to have been written in 1946 and referring to the war years, whereas it was actually referring to the extensive building programme in Seascale to meet the demands of housing nuclear workers (Kinlen, 2006).

Sixth COMARE report

A reconsideration of the possible health implications of the radioactive particles found in the general environment around the Dounreay nuclear establishment in the light of the work undertaken since 1995 to locate their source

2.75 A COMARE working group visited the Dounreay site in 1994 and was informed of further potential sources of contamination in addition to those used as the basis for the second report. As a result, the sixth report considered all information available up to 1998 (COMARE, 1999).

2.76 The most significant change was the detection of numbers of metallic particles on the foreshore of the plant itself and on the nearby Sandside beach. These contained primarily strontium-90 and caesium-137. Although the route of discharge could not be identified, the origin of the particles could be traced to swarf arising from on-site underwater milling of spent fuel elements prior to reprocessing.

2.77 COMARE took the view that health effects were most likely to arise from skin contact or ingestion and requested the NRPB to estimate the radiation dose that a member of the public might receive in the unlikely event of encountering such a particle, assuming a log-normal distribution with a mean activity of 1 MBq.

2.78 Subsequent computer simulations suggested that some 25,000 particle ingestions would have been required to produce the observed excess of leukaemia. On this basis, COMARE concluded that the particles could not be a plausible cause of the increased incidence of childhood leukaemia.

2.79 In the light of a then recent publication (Kinlen et al, 1995), COMARE noted that, in the absence of any reasonable alternative, the rural population mixing hypothesis could give a plausible explanation of the excess of cases of leukaemia in Thurso, due to the mixing brought about by the North Sea oil industry.

Joint report by COMARE and the Radioactive Waste Management Advisory Committee (RWMAC)

Report on radioactive contamination at a property in Seascale, Cumbria

2.80 In October 1998, radioactively contaminated pigeons were reported at a private property in the village of Seascale, Cumbria, where large numbers of pigeons had been fed for a number of years. Investigations showed that limited areas of the property, mainly the garden, were also significantly contaminated. Contamination of adjacent properties was much less.

2.81 COMARE was requested by the Department of Health (DH) to investigate the public health implications. COMARE requested assistance from the Radioactive Waste Management Advisory Committee (RWMAC) on the possible sources of radioactive contamination of the pigeons. The joint report by RWMAC and COMARE published in June 1999 contained the findings of the two committees and accompanied a report prepared jointly by the local authority, government departments and agencies which provided more details of the contamination at Seascale and of the subsequent steps in decontaminating the property (COMARE/RWMAC; 1995; Copeland Borough Council, 1999).

Possible sources of radioactive contamination of pigeons

2.82 The most likely explanation for the radioactive contamination at the property was that it had been brought about by pigeons contaminated by material from the Sellafield nuclear site and was not due to any pre-existing activity. Some older buildings on the Sellafield site that could be accessed by the pigeons were thought to be the most likely origin.

2.83 The contamination at the property at Seascale appeared to have arisen from an unusual combination of circumstances: pigeons flocking in large numbers to a particular location and carrying with them radioactive contamination. Apart from this property and a disused property, the pigeons were not thought to congregate elsewhere in Seascale.

Health implications

2.84 The additional risk to the health of the occupants of the Seascale property, and of the neighbouring properties, from this radioactive contamination was not significant. In particular, this incident was not thought to be associated with the observed elevated incidence of leukaemia in young people in Seascale.

Conclusions

2.85 COMARE and RWMAC stated that it was unacceptable that the Sellafield site had been managed in a way that allowed pigeons to become contaminated to the extent that property away from the site also became significantly contaminated. Furthermore, the two committees also stated that continued vigilance and preparedness was necessary in order to prevent similar incidents involving wildlife from occurring in the future, either at Sellafield or elsewhere.

Seventh COMARE report

Parents occupationally exposed to radiation prior to the conception of their children: a review of the evidence concerning the incidence of cancer in their children

2.86 The seventh report "reviewed in depth the genetic, biological and epidemiological data that has become available since the time of publication of our fourth report". The report also took the "opportunity of reviewing the current status of the Seascale cluster" (COMARE, 2002).

2.87 Overall, 21 cases* were identified of diagnoses of, or deaths from, cancer during 1950–2001 among young people of 0–24 years of age who were

* Excluding a case of osteosarcoma in a child who died while resident in Seascale, but was born and diagnosed while resident outside the area defined by the current county of Cumbria.

either resident in Seascale at diagnosis or death, or were born in the village. Of these, 14 were of leukaemia or NHL; ten cases were in the 0–14 year age group, six of whom were in the 1–4 year age group. There was a clear excess of cases of cancer among young people who had been resident for some period in Seascale, and this excess was most marked for cases of leukaemia and NHL in young children. The seventh report noted that the "currently available evidence indicates that population mixing is responsible for a substantial part of the excess of LNHL [leukaemia and NHL] among young people in Seascale", but that "while an infectious agent is thought by many experts to be involved in childhood leukaemia, it has not yet been identified and the biological mechanism for the population mixing hypothesis remains speculative".

2.88 It had been suggested by Gardner et al (1990b) that occupational exposure of fathers to radiation while working at Sellafield before the conception of their children increased the risk of leukaemia and NHL in these children, and that the association between the dose of paternal preconceptional radiation exposure and childhood leukaemia and NHL could explain the excess of childhood cases in Seascale. However, it was subsequently shown by Kinlen (1993) that paternal occupational exposure could not, by itself, explain the Seascale excess of childhood leukaemia and NHL. A cohort study of Cumbrian births during 1950–1991 (Dickinson and Parker, 2002) found a marginally significant dose response for paternal preconceptional dose and leukaemia and NHL among young people born in Seascale (which included the cases studied by Gardner et al (1990a,b), but a weaker and non-significant dose response for the much larger number of young people with, on average, higher paternal preconceptional doses born in Cumbria outside Seascale.

2.89 Following an extensive review, the seventh report found "no convincing evidence to suggest that ionising radiation alone at the doses to which male nuclear industry radiation workers have been exposed, results in an increased incidence of childhood cancer".

Ninth COMARE report

Advice to government on the review of the radiation risks from radioactive internal emitters carried out and published by the Committee Examining Radiation Risks of Internal Emitters

2.90 The Committee Examining the Radiation Risks of Internal Emitters (CERRIE) existed during 2001–2004 with the objective of examining the risks to health posed by intakes of radioactive materials, and the uncertainties associated with these risk estimates. The CERRIE final report was published in 2004 (CERRIE, 2004).

2.91 One of the topics addressed by CERRIE was whether there were substantially different effects for internal emitters and external sources of penetrating ionising radiation. CERRIE was divided on the adequacy of methods used to take account of the heterogeneity of energy deposition associated with internal emitters.

2.92 As part of its ninth report (COMARE, 2004b), COMARE sought to identify groups exposed to higher levels of internal emitters and commissioned a study of three such groups – Sellafield workers, workers at the Mayak plant in the former USSR and the residents of the Techa river regions exposed to discharges from the Mayak plant. Risk estimates for these three groups were compared with those obtained from studies of the Japanese atomic bomb survivors.

2.93 The results of this study demonstrated that the estimates of risk for leukaemia and solid cancers in these groups were close to those estimated from the atomic bomb survivors, indicating that the conventional approach to assessing risks from internal emitters is not grossly in error.

Tenth COMARE report

The incidence of childhood cancer around nuclear installations in Great Britain

2.94 The tenth COMARE report described the results of a systematic examination of the incidence of childhood leukaemia and NHL, and of other childhood cancers, around all nuclear installations in Great Britain, and of groupings of installations such as all nuclear power stations (COMARE, 2005). This study was a result of recommendations in the third COMARE report (see above) that a comprehensive national registry of childhood cancers should be generated and that when these data became available studies should be carried out to investigate the patterns of childhood cancer incidence on a nationwide basis (which was the subject of the eleventh COMARE report – see below). A comprehensive study of the incidence of childhood leukaemia and NHL (LNHL) and of other cancers in areas surrounding nuclear installations could then be conducted, the results of which could be placed into the context of the nationwide findings.

2.95 The tenth report examined, *inter alia*, the incidence of childhood LNHL and other childhood cancers during 1969–1993 in the groups of census wards lying within 25 km of Sellafield and Dounreay. The report also considered the variation of incidence rate with distance of a ward from an installation. Leukaemia was combined with NHL in the analyses because of a recommendation in the second report (see above) based on the similarity of certain forms of these childhood cancers and the difficulties in consistently distinguishing between them, especially over a 25-year period.

2.96 For Sellafield, the observed number of cases of childhood LNHL within 25 km of the site was 25, which compared with an expected number of 21.95, a statistically non-significant excess. However, the preferred test of the variation of the incidence rate with distance from the site showed a statistically significant trend with distance ($p = 0.018$), reflecting the excess of cases of LNHL in Seascale ward. For other childhood cancers, the observed number of 40 compared with an expected number of 35.96, a statistically non-significant excess, and there was no significant trend of incidence rate with distance.

2.97 For Dounreay, the observed number of cases of childhood LNHL within 25 km of the site of nine compared with 3.87 expected, a statistically significant excess ($0.01 < p < 0.05$). The distance trend test of choice for Dounreay showed a statistically significant variation of incidence rate by distance ($p = 0.014$). For other childhood cancers, the observed number of three compared with an expected number of 6.29, a statistically non-significant deficit, and there was no trend of incidence rate with distance from the site.

2.98 COMARE concluded that the results of the study were consistent with the previous findings for Sellafield and Dounreay, in that they reflected the known localised excesses of cases of LNHL in Seascale near Sellafield and in Thurso in the vicinity of Dounreay, and that these excesses “are unlikely to be due to chance, although there is not at present a convincing explanation for them”. The incidence of other childhood cancers around these sites was not unusual.

Eleventh COMARE report

The distribution of childhood leukaemia and other childhood cancers in Great Britain 1969–1993

2.99 The nationwide database of childhood cancer incidence used in this study (a recommendation of the third report – see above) was constructed from the National Registry of Childhood Tumours by staff of the then Childhood Cancer Research Group in Oxford, and contained 12,415 cases of childhood LNHL and 19,908 cases of other childhood cancers, diagnosed while less than 15 years of age in Great Britain during 1969–1993. The aim of the eleventh report was "to provide an overview of the geographical patterns of variation in the incidence of different types of childhood cancer, and the relation of these to certain socio-demographic factors, and to examine whether spatial or space-time clustering are a part of the general pattern of occurrence of childhood leukaemia and other cancers" (COMARE, 2006).

2.100 At each geographical level examined – country, region, county, county district and census ward – childhood cancers of many types had a non-random distribution, although the reasons for this were unknown. Owing to the large number of comparisons carried out in the study, some of these deviations from randomness could have been due to chance fluctuations. However, the overall pattern of results suggested that the main aetiological factors for childhood cancers are distributed heterogeneously, not only for childhood leukaemia but for a number of other cancer types, such as central nervous system (CNS) tumours.

2.101 For childhood leukaemia, socioeconomic status was strongly related to the levels of incidence at the county, district and ward levels, with the incidence being positively associated with socioeconomic status, an effect that was strongest for lymphoid leukaemia in the 1–4 and 5–9 years age groups. To a lesser extent, the rate of incidence increased as population density decreased, and increased slightly in more rural areas. Consequently, it was unsurprising that cases of childhood leukaemia were found to 'cluster' to a greater extent than expected by chance alone, particularly in the 1–4 years age group.

2.102 The analyses showed the extent to which variations in rates could reasonably be attributed to geographical variation or to certain explanatory variables. For more extreme values of the relevant factors, and particular combinations of them, greater variations in risk will occur, although whether this by itself was sufficient to account for the rarer 'clusters' such as that at Seascale could not be determined. Nonetheless, Seascale was notably extreme in terms of its remote rural nature, and the high socioeconomic status of its population, particularly in the earlier years of nuclear operations at Sellafield.

CHAPTER 3

INCIDENCE OF CANCER AND LEUKAEMIA IN INDIVIDUALS RESIDENT AT THE TIME OF BIRTH OR DIAGNOSIS IN THE VICINITIES OF SELLAFIELD AND DOUNREAY

Background

Sellafield

3.1 The report of the Independent Advisory Group (Black, 1984) concluded that the rate of incidence of childhood leukaemia in the village of Seascale over a 30-year period was unusually high, but that this was based on less than ten children who had developed the malignant disease. As a consequence, the Black Advisory Group was cautious in its conclusions, in particular because the Seascale 'cluster' was defined after the distribution of the data was known, which permitted a *post hoc* concentration upon a specific village, age group, disease and period. Among the recommendations of the report were four related to epidemiological studies that would help to clarify the situation.

3.2 The fourth COMARE report presented the findings of the epidemiological studies recommended by the Independent Advisory Group (Black, 1984), and of other relevant epidemiological studies (COMARE, 1996). The Seascale birth and schools cohorts studies (the second and third recommendations of the Group) confirmed an approximately ten-fold excess of leukaemia deaths among children born in the village during 1950–1983 (based on five deaths to June 1986, one of a child unknown to the Group who was born in 1950 and died soon after leaving the village), but found no leukaemia deaths among children attending schools in Seascale up to November 1984 who were not born to mothers resident there at the time of birth (Gardner et al, 1987a,b), suggesting the operation of some factor before birth or early in life. However, the absence of risk of leukaemia and NHL (LNHL) among those moving into Seascale after birth was later challenged by Kinlen (1993), who found a comparable excess of LNHL among young people born in Seascale and those born elsewhere.

3.3 Recommendation 4 of the Black report requested a study of the incidence of cancer among young people living in small areas (census wards) in the north of England. Craft et al (1993) reported the findings of this study, covering cancers diagnosed among those aged 0–24 years during 1968–1985 while resident in the 1276 wards of north and northwest England. Seascale was the most extreme ward (as measured by cumulative Poisson probability) for the rate of incidence of acute lymphoblastic leukaemia and NHL combined, emphasising the highly unusual number of cases that have occurred in the village. Wakeford and Parker (1996) examined incidence rates of LNHL in wards in west Cumbria in more detail, and found that unusual incidence rates in wards in the vicinity of Sellafield were effectively confined to Seascale, although a raised rate was noted in the ward of Egrement North (an inland ward, approximately 10 km to the north of Sellafield), but this was not nearly as extreme as that for Seascale.

3.4 Recommendation 1 of the Black report was for a case–control study of leukaemia and lymphoma among young people in west Cumbria to be conducted. The findings of this study were reported in 1990 by Gardner (Gardner et al, 1990a,b). Although the results did not indicate raised risks associated with environmental exposure to radiation (such as the consumption

of seafood or playing on local beaches), the study found an association between the risk of LNHL and the recorded occupational dose of external radiation received at Sellafield by fathers before the conception of their children. The interpretation of this association was considered in detail in the fourth and seventh COMARE reports and will be considered further in Chapter 6 of this report.

3.5 The fourth COMARE report reviewed the findings of epidemiological studies published since the report of the Black Advisory Group, including those studies recommended by the Group and summarised above. Also available to COMARE were the results of other geographical studies of cancer incidence in Cumbria (Draper et al, 1993; Bithell et al, 1994; Kinlen et al, 1995), which confirmed that the highly unusual incidence rate of cancer near Sellafield was essentially confined to LNHL among young people in Seascale.

3.6 In the tenth COMARE report the incidence of childhood LNHL and of other cancers during 1969–1993 was examined for wards lying within 25 km of a nuclear installation in Great Britain, the observed (O) and expected (E) numbers of cases in these areas being compared and a test conducted for a trend of ward incidence rate with distance from the installation. For Sellafield and childhood LNHL, the O/E ratio of 25/21.95 was raised to a statistically non-significant extent, and a statistically significant decreasing trend of incidence rate with increasing distance from the site was found, both of these results being due to the high incidence rate in Seascale ward. This study did not suggest that unusual rates of incidence of childhood LNHL were to be found generally in the vicinity of Sellafield, apart from Seascale ward.

Dounreay

3.7 A proposed expansion of the nuclear reprocessing plant at Dounreay, Caithness, led to a public local development inquiry in 1986 (Bell, 1989). In response to a request to provide information on the risk of cancer among the population living near the plant, a higher than expected incidence of leukaemia in children and young adults in the period 1968–1984 was reported by Heasman and colleagues (Heasman et al, 1986). This finding was referred to COMARE.

3.8 The second COMARE report concerned a detailed review of the epidemiological data and an assessment of the leukaemogenic potential of radioactive discharges from the site (COMARE, 1988). Six cases of leukaemia in the age group 0–24 years were observed among the population living within 25 km of Dounreay in the period 1968–1984 compared with 3.0 expected (an O/E ratio of 2.0, $p = 0.08$). COMARE noted that, while the overall incidence during this period was greater than expected, the concentration of (all six) cases in the period 1979–1984 was of particular concern, as was the finding that two cases of childhood NHL diagnosed during 1968–1984 should be more appropriately classified as ALL. COMARE concluded that, given current knowledge of the potential for radiation exposure to induce leukaemia, the discharge levels from the plant were insufficient to account for the observed excess of cases among children and young adults living near Dounreay. Further epidemiological work was recommended.

3.9 Black et al (1992) examined the incidence of leukaemia in children born in the Dounreay area during 1969–1988 (birth cohort) and in children born elsewhere who attended local schools during the same period (schools cohort). Excesses of cases of leukaemia were found: O/E = 2.3 (95% CI 0.7, 5.4) in the birth cohort and 6.7 (95% CI 1.4, 19.5) in the schools cohort. In a case–control study of children and young adults diagnosed with leukaemia or NHL during 1970–1986 compared with disease-free subjects resident in Caithness, the only finding of potential concern was that parents of subjects with leukaemia or NHL

were more likely to report recreational use of local beaches than parents of controls (Urquhart et al, 1991). However, Urquhart et al (1991) warned against overinterpretation of this apparent association because of multiple statistical testing and the real possibility of recall bias. In contrast to the findings of the west Cumbria case–control study (Gardner et al, 1990b), the Caithness case–control study did not find an association between LNHL and paternal preconceptional irradiation (PPI), and paternal occupational exposure to radiation before conception could not account for the excess of cases around Dounreay. Consequently, PPI could not be the common 'feature' of the Sellafield and Dounreay areas that the COMARE Second Report (1988) proposed might explain the case excesses. These two Caithness studies included cases of leukaemia and NHL registered after 1984, the last year considered in the second COMARE report.

3.10 Kinlen and colleagues (Kinlen, 1993; Kinlen et al, 1993) noted a non-significant excess of childhood (0–14 years of age) leukaemia cases in Thurso during an earlier period, 1951–1967, in which two cases had been observed against 0.41 case expected.

3.11 A formal update of the incidence of childhood and young adult leukaemia and NHL in 1985–1991 was reported by Black et al (1994). Four new cases were observed in the zone within 25 km of Dounreay during this period, compared with 1.4 expected, a marginally significant excess, bringing the total for the period 1968–1991 to 12 cases observed compared with 5.2 expected, a significant excess.

3.12 A further COMARE recommendation was that the incidence of LNHL in young people living near any of the nuclear sites in Scotland should be examined. Sharp et al (1996) examined the incidence of LNHL in children less than 15 years of age diagnosed while resident within 25 km of seven nuclear sites in Scotland during 1968–1993, and tested for a trend of incidence with distance from a site. They found no evidence of a generally increased risk of childhood LNHL near such sites. For the Dounreay area, the observed number of cases of nine compared with an expected number of 4.53 ($p = 0.08$). However, in an analysis using 'boundary-free' statistical methods, the observed excess incidence of LNHL in the 0–14 age group living within 25 km of Dounreay was statistically significant ($p = 0.03$). The trend of risk with distance from Dounreay was not significant.

Update of cancer incidence analyses

Introduction

3.13 This chapter describes analyses commissioned by the Department of Health's Radiation Protection Research Programme at the behest of COMARE and undertaken by the then Childhood Cancer Research Group within the University of Oxford. These analyses examine cancer incidence among children, teenagers and young adults resident close to either Sellafield or Dounreay at the time of cancer diagnosis, and also cancer incidence at any age among those resident at birth near to either installation (Bunch et al, 2014).

3.14 Cancer excesses in the vicinities of Sellafield and Dounreay were investigated separately, but using methods as similar as the available data allowed. Briefly, standardised incidence ratios (SIRs) were calculated for cross-sectional populations and also for birth cohorts, applying national cancer registration rates to the calculated populations at risk to derive expected numbers for comparison with the numbers of events observed.

3.15 The analyses in this report consider defined areas around each site. For comparison, data from the analyses presented in the tenth COMARE report for wards within 25 km of Sellafield and Dounreay for the period 1969–1993 are given in Appendix B.

Materials and methods

Analyses relating to Sellafield

3.16 Three designated areas within Cumbria (shown in Figure 3.1) were defined:

(i) Seascale ward (as defined at the time of the 1981 census);

(ii) county districts of Allerdale and Copeland, excluding Seascale ward;

(iii) remainder of Cumbria, ie the county districts of Carlisle, Eden, South Lakeland and Barrow.

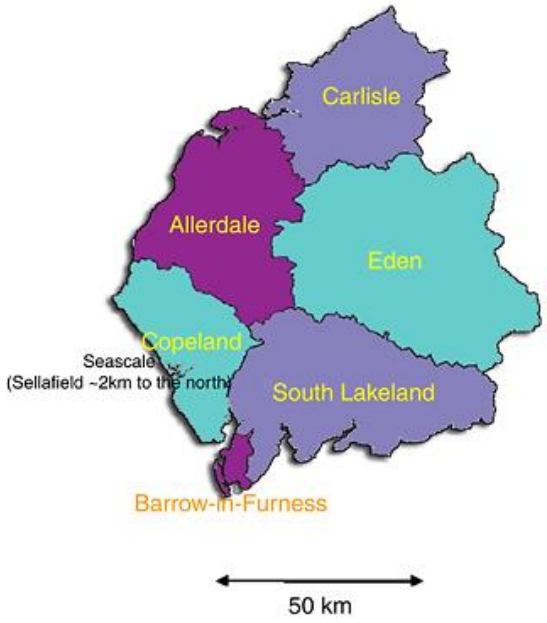

Figure 3.1 County districts of Cumbria

Cross-sectional analysis

3.17 Published data from the Office for National Statistics (ONS) from the decennial censuses (1991–2001) together with county district annual estimates from 1972 to 2006 were used to derive age-specific population estimates for the three areas around Sellafield described above (Table 3.1) and National Registry of Childhood Tumours (NRCT) data were used to determine the number of childhood cancers occurring among residents of each area in any given time period. These resulting rates were compared with NRCT national rates. Similarly, cancers in young adults were identified from the Northern Region Young Persons Malignant Disease Registry (NRYPMDR) and corresponding rates compared to NRYPMDR rates.

Table 3.1 Summary of average annual population estimates for areas within Cumbria

	Seascale ward		Copeland and Allerdale (excluding Seascale)		Rest of Cumbria	
Period	**0–14 years**	**15–24 years**	**0–14 years**	**15–24 years**	**0–14 years**	**15–24 years[b]**
1963–1983[a]	533	294	38,571	24,398	67,649	33,225
1984–1990	343	267	30,880	24,805	56,529	36,890
1991–2006	299	160	29,763	18,463	56,702	27,856
1963–2006	418	232	34,144	21,974	61,899	31,639

a 1969–1983 for 15–24 age group as cancer incidence data only available from 1969

b Rest of Cumbria excludes Barrow-in-Furness county district for 15–24 age group

3.18 For childhood cancers, standardised incidence ratios (SIRs) are based on national rates derived from NRCT registrations for England, Wales and Scotland, whereas for the young adult cancers (for 15–24 year olds) comparative rates were based on registrations held by the NRYPMDR, excluding those for individuals resident in Cumbria at the time of diagnosis. For the cross-sectional analyses for 15–24 year olds, the county district of Barrow was excluded, as the NRYPMDR does not register Barrow cases (Table 3.5).

Birth cohort analysis

3.19 A cohort was assembled to include all individuals born in Cumbria between 1950 and 2006 to mothers resident in Cumbria at the time of the child's birth. Cohort members were then assigned to one of the three designated areas on the basis of the postcode of their mother's usual address at the time of their birth.

3.20 An existing cohort of children born in Cumbria between 1950 and 1993 (held both at the ONS and by collaborators at Newcastle University) formed the starting point of the extended cohort and the ONS identified those children born between 1994 and 2006 to be added to the cohort.

3.21 The birth cohort was flagged at the ONS and researchers were notified of all deaths, cancer diagnoses (after 1971) or emigrations (and thus losses to follow up) among cohort members provided the individual was still alive in 1991 when records were transferred to the Central Health Register Enquiry System (CHRIS). Similarly, the General Register Offices (GROs)/the Scottish Information Services Division (ISD) provided notifications for members of the cohort who had migrated to Scotland. These returns were used to calculate incidence rates, for both childhood and adult cancers for the period 1971–2006, which have been compared with corresponding population rates. Careful cross-checking between the ONS and Newcastle University versions of the pre-1994 cohort meant that cohort members who died or emigrated before 1991 could be included in the study and any cancers recorded for these individuals between 1971 and 1993 were included in the analyses that follow.

Analyses relating to Dounreay

3.22 Two geographical areas surrounding the Dounreay nuclear installation have been defined for which population estimates from decennial censuses (1961–2001) for the relevant age groups were provided by the GROs and within which cancer cases can be identified. Childhood cancers (ages 0–14 years) were identified from the NRCT and cancers in young adults (ages 15–24 years) were identified from records held by the ISD in Scotland.

Cross-sectional analysis

3.23 The area closest to Dounreay consists of the civil parishes of Thurso and Reay (Figure 3.2). The second area consists of the remaining civil parishes of Caithness, geographically a much larger area with a low population density.

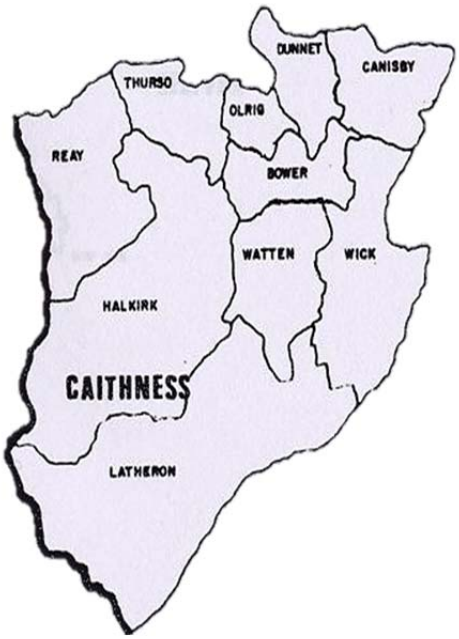

Figure 3.2 Civil parishes of Caithness

Table 3.2 Summary of average annual population estimates for civil parishes around Dounreay

	Thurso and Reay civil parishes		Remaining civil parishes of Caithness	
Period	**0–14 years**	**15–24 years**	**0–14 years**	**15–24 years**
1963–1983	2,817	1,586	4,297	2,478
1984–1990	2,067	1,590	3,606	2,482
1991–2006	1,747	1,193	3,188	1,969
1963–2006	2,309	1,444	3,784	2,293

Birth cohort analysis

3.24 GROs/ISD staff identified all children born between 1950 and 2006 in two specified areas around Dounreay (Figure 3.3):

(i) postcode sector KW 14-7;

(ii) postcode sectors KW12-6, KW13-6 and KW14-8.

3.25 The cohort was linked to the Scottish morbidity records at GROs to obtain information on cancer cases among cohort members during 1971–2006.

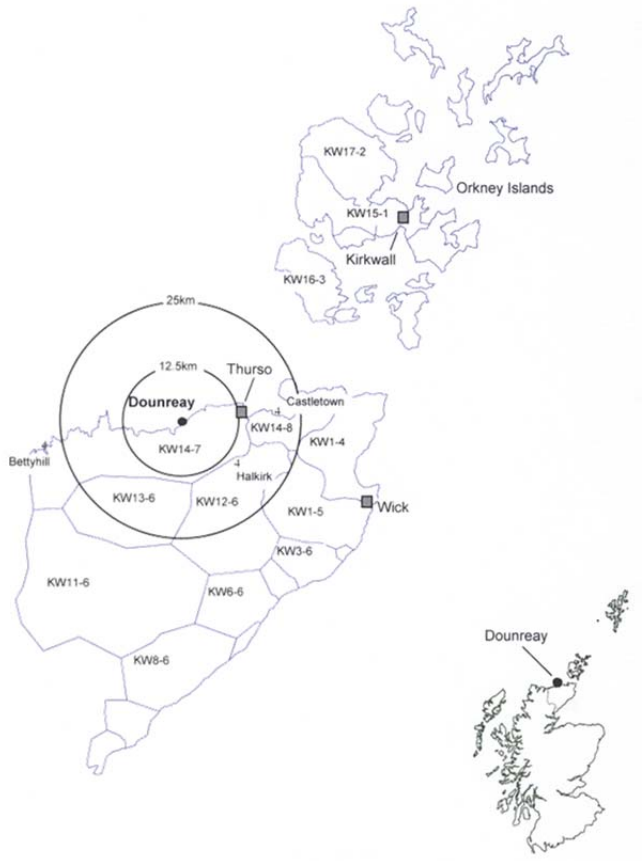

Figure 3.3 North east Scotland: Kirkwall (KW) postcode area showing approximate sector boundaries and concentric zones centred on Dounreay

Outcome measures

3.26 Throughout the analyses described, cancer incidence is the outcome measure.

3.27 The cross-sectional analyses concentrated on cancers among children (aged 0–14 years) and young people (aged 15–24 years). Total cancer was defined to include all malignant neoplasms together with all central nervous system (CNS) or brain tumours of uncertain or benign nature. Standardised incidence rates were also calculated separately for lymphoid leukaemia, other leukaemia, total leukaemia (see the glossary, Appendix A, for an explanation of the types of leukaemia), non-Hodgkin lymphoma (NHL), Hodgkin's lymphoma, total leukaemia and NHL (LNHL), brain and CNS tumours (including non-malignant tumours), and other solid tumours.

3.28 The cohort analyses considered cancer incidence at all ages during 1971–2006, with the earliest born cohort members approaching age 57 years at the close of the study in 2006. Total cancer was defined to include all malignant tumours (except for non-melanoma skin cancers) together with non-malignant brain tumours. Diagnostic subgroups considered were leukaemia, NHL, Hodgkin's lymphoma, CNS and brain tumours, melanoma of the skin, neuroblastoma and ganglioneuroblastoma, tumours of the upper respiratory tract, tumours of the gastrointestinal tract, tumours of the lower respiratory tract, breast cancer, tumours of the genito-urinary tract, and thyroid cancer.

3.29 The cohort analyses were carried out using STATA, version 11 software (StataCorp, 2009).

Results

Results relating to Sellafield

Cross-sectional analysis

3.30 The numbers of cancer cases identified among residents in Seascale ward (as defined for the 1981 census), Copeland (excluding Seascale) and Allerdale, and the rest of Cumbria, are shown in Tables 3.3, 3.4 and 3.5, respectively, together with standardised incidence ratios (SIRs) and 95% confidence intervals (CIs).

3.31 Table 3.3 shows a single case of childhood cancer – neuroblastoma – in the period 1991–2006 among children aged 0–14 years and diagnosed while resident in Seascale ward, which gives rise to a statistically non-significant excess of cases of 'other malignancies' during this period. This case was mentioned in the fourth COMARE report (COMARE, 1996) – case Q in Table 2.4 – but was not included in any of the analyses of the report because it was diagnosed too late (in 1995). The significant excesses of leukaemia and NHL observed in this age group in earlier periods (five cases during 1963–1983 and a further two cases during 1984–1990) are not evident in the more recent years, 1991–2006 (Table 3.3). The case of 'other leukaemia' recorded in the 0–14 year age group during 1984–1990 has not been reported in earlier studies: it arises now as a result of the reclassification of myelodysplastic syndrome as a malignancy within ICDO-3. Table 3.3 also shows a single case of lymphoid leukaemia among 15–24 year olds resident in Seascale ward during 1991–2006 (SIR 36.98, 95% CI 0.92, 205.98). This case was included in the fourth report (1996) – case P in Table 2.4 – and, since the year of diagnosis was 1991, it was included in the analyses reported there for the period 1984–1992. There was an absence of incident cases of cancer in the 15–24 year age group during the period 1969–1983 followed by a raised SIR (9.25, 95% CI 1.91, 27.01) for the period 1984–1990, based on three cases of cancer (Hodgkin's lymphoma, NHL and brain tumour), and then one case during 1991–2006 (the case of lymphoid leukaemia discussed above).

Table 3.3 Cases and SIRs for individuals aged 0–14 and 15–24 years resident in Seascale ward

	Ages 0–14 years				Ages 15–24 years			
	Observed	**Expected**	**SIR[a]**	**(95% CI)**	**Observed**	**Expected**	**SIR[b]**	**(95% CI)**
Lymphoid leukaemia								
1963–1983[c]	4	0.29	**13.71**	(3.73, 35.10)	0	0.04	0.00	(0.00, 71.70)
1984–1990	0	0.08	0.00	(0.00, 39.39)	0	0.02	0.00	(0.00, 133.26)
1991–2006	0	0.17	0.00	(0.00, 17.94)	1	0.03	36.98	(0.92, 205.98)
1963–2006	4	0.55	**7.28**	(1.98, 18.64)	1	0.09	10.91	(0.27, 60.77)
Other leukaemia								
1963–1983[c]	0	0.11	0.00	(0.00, 26.23)	0	0.05	0.00	(0.00, 66.29)
1984–1990	1	0.02	**47.04**	(1.18, 262.01)	0	0.02	0.00	(0.00, 156.52)
1991–2006	0	0.05	0.00	(0.00, 65.34)	0	0.03	0.00	(0.00, 111.66)
1963–2006	1	0.18	5.56	(0.14, 30.94)	0	0.09	0.00	(0.00, 32.86)
Total leukaemia								
1963–1983[c]	4	0.41	**9.85**	(2.68, 25.22)	0	0.09	0.00	(0.00, 34.45)
1984–1990	1	0.10	10.28	(0.26, 57.26)	0	0.04	0.00	(0.00, 72.00)
1991–2006	0	0.21	0.00	(0.00, 14.07)	1	0.05	18.56	(0.46, 103.38)
1963–2006	5	0.73	**6.85**	(2.22, 15.96)	1	0.18	5.47	(0.14, 30.47)
Non-Hodgkin lymphoma								
1963–1983[c]	1	0.08	12.33	(0.31, 68.68)	0	0.04	0.00	(0.00, 74.61)
1984–1990	1	0.02	**57.02**	(1.42, 317.60)	1	0.03	34.12	(0.85, 190.05)
1991–2006	0	0.04	0.00	(0.00, 79.87)	0	0.02	0.00	(0.00, 133.62)
1963–2006	2	0.14	**14.59**	(1.77, 52.67)	1	0.09	11.05	(0.28, 61.55)
NHL + total leukaemia								
1963–1983[c]	5	0.49	**10.26**	(3.32, 23.91)	0	0.13	0.00	(0.00, 23.57)
1984–1990	2	0.11	**17.41**	(2.11, 62.85)	1	0.07	14.10	(0.35, 78.54)
1991–2006	0	0.25	0.00	(0.00, 11.96)	1	0.08	13.11	(0.33, 73.03)
1963–2006	7	0.87	**8.08**	(3.24, 16.64)	2	0.27	7.32	(0.89, 26.43)
Hodgkin's lymphoma								
1963–1983[c]	0	0.05	0.00	(0.00, 60.05)	0	0.14	0.00	(0.00, 21.90)
1984–1990	0	0.01	0.00	(0.00, 245.351)	1	0.06	16.79	(0.42, 93.52)
1991–2006	0	0.03	0.00	(0.00, 104.09)	0	0.06	0.00	(0.00, 50.13)
1963–2006	0	0.09	0.00	(0.00, 32.07)	1	0.25	4.00	(0.10, 22.28)
Brain/CNS (including benign)								
1963–1983[c]	0	0.28	0.00	(0.00, 10.72)	0	0.10	0.00	(0.00, 29.17)
1984–1990	0	0.07	0.00	(0.00, 43.86)	1	0.04	25.81	(0.65, 143.76)
1991–2006	0	0.17	0.00	(0.00, 17.96)	0	0.06	0.00	(0.00, 48.54)
1963–2006	0	0.53	0.00	(0.00, 5.65)	1	0.20	4.90	(0.12, 27.29)

	Ages 0–14 years				Ages 15–24 years			
	Observed	**Expected**	**SIR[a]**	**(95% CI)**	**Observed**	**Expected**	**SIR[b]**	**(95% CI)**
Other malignancies								
1963–1983[c]	0	0.40	0.00	(0.00, 7.56)	0	0.29	0.00	(0.00, 10.22)
1984–1990	0	0.10	0.00	(0.00, 28.83)	0	0.16	0.00	(0.00, 19.32)
1991–2006	1	0.22	4.48	(0.11, 24.95)	0	0.24	0.00	(0.00, 12.591)
1963–2006	1	0.74	1.35	(0.03, 7.52)	0	0.70	0.00	(0.00, 4.26)
All malignancies								
1963–1983[c]	5	1.21	**4.12**	(1.33, 9.60)	0	0.66	0.00	(0.00, 4.54)
1984–1990	2	0.30	6.68	(0.81, 24.11)	3	0.32	**9.25**	(1.91, 27.01)
1991–2006	1	0.67	1.49	(0.04, 8.30)	1	0.44	2.30	(0.06, 12.81)
1963–2006	8	2.23	**3.58**	(1.54, 7.05)	4	1.43	2.80	(0.76, 7.17)

a Based on NRCT England, Wales and Scotland registrations excluding 86 cases with no postcode

b Based on registrations from the Northern Region Young Persons Malignant Disease Registry excluding those for Cumbria

c 1969–1983 for 15–24 age group as NRYPMDR data only available from 1969

SIRs which differ significantly from 1 are shown in bold ($p < 0.05$), bold and underlined ($p < 0.01$)

3.32 Fairlie and Korblein (2015) criticised this analysis for not simply considering the period 1963–2006 as a single entity. In reply, McNally et al (2015) opined that it would be misleading not to subdivide the period, since this would imply that any putative agent would have to be invariant over this prolonged timespan. Further, COMARE (1996) has previously subdivided the total period to determine whether the raised incidence of malignant disease identified by the Black Advisory Group had persisted (see paragraph 2.45 above), and the approach adopted by Bunch et al (2014) is consistent with this.

3.33 Table 3.4 shows the numbers of cancers and SIRs for Allerdale and Copeland county districts (excluding Seascale ward), the area surrounding Seascale ward. Corresponding information for the remainder of Cumbria is shown in Table 3.5. There are no suggestions of any significantly raised incidence of cancer in children or young adults in Allerdale and Copeland county districts during any of the periods covered by this study. In the remainder of Cumbria, when leukaemia and NHL were considered together, there was a significant excess of cases among children aged 0–14 years during the period 1984–1990 (SIR 1.64, 95% CI 1.11, 2.33 based on 31 cases), but this excess was not evident in either the earlier or later periods studied. Young adults (aged 15–24 years) appeared to be at a non-significantly reduced risk for leukaemia in each of the time periods studied and, when the time periods were combined, this reduced risk became significant albeit based on only 14 cases (SIR 0.58, 95% CI 0.32, 0.97). Driven by this reduced leukaemia risk, the risk for cancer overall in young adults was again non-significantly reduced in each of the three time periods considered and significantly reduced when the time periods were combined (SIR 0.85, 95% CI 0.73, 0.99).

Table 3.4 Cases and SIRs for individuals aged 0–14 and 15–24 years resident in Copeland and Allerdale county districts (not including Seascale ward)

	Ages 0–14 years				Ages 15–24 years			
	Observed	Expected	SIR[a]	(95% CI)	Observed	Expected	SIR[b]	(95% CI)
Lymphoid leukaemia								
1963–1983[c]	15	21.11	0.71	(0.40, 1.17)	5	3.23	1.55	(0.50, 3.60)
1984–1990	7	6.84	1.02	(0.41, 2.11)	1	1.85	0.54	(0.01, 3.00)
1991–2006	12	16.61	0.72	(0.37, 1.27)	5	3.06	1.63	(0.53, 3.81)
1963–2006	34	44.91	0.76	(0.52, 1.06)	11	8.19	1.34	(0.67, 2.40)
Other leukaemia								
1963–1983[c]	7	8.26	0.85	(0.34, 1.75)	4	3.75	1.07	(0.29, 2.73)
1984–1990	3	1.91	1.57	(0.32, 4.58)	1	1.61	0.62	(0.02, 3.47)
1991–2006	7	4.56	1.54	(0.62, 3.16)	1	3.13	0.32	(0.01, 1.78)
1963–2006	17	14.71	1.16	(0.67, 1.85)	6	8.53	0.70	(0.26, 1.53)
Total leukaemia								
1963–1983[c]	22	29.37	0.75	(0.47, 1.13)	9	6.98	1.29	(0.59, 2.45)
1984–1990	10	8.76	1.14	(0.55, 2.10)	2	3.46	0.58	(0.07, 2.08)
1991–2006	19	21.17	0.90	(0.69, 1.40)	6	6.19	0.97	(0.36, 2.11)
1963–2006	51	59.61	0.86	(0.64, 1.13)	17	16.72	1.02	(0.59, 1.63)
Non-Hodgkin lymphoma								
1963–1983[c]	4	5.87	0.68	(0.19, 1.75)	7	3.22	2.17	(0.87, 4.48)
1984–1990	0	1.58	0.00	(0.00, 1.90)	3	2.72	1.10	(0.23, 3.22)
1991–2006	2	3.73	0.54	(0.06, 1.93)	3	2.67	1.12	(0.23, 3.28)
1963–2006	6	11.20	0.54	(0.20, 1.17)	13	8.58	1.52	(0.81, 2.59)
NHL + total leukaemia								
1963–1983[c]	26	35.23	0.74	(0.48, 1.08)	16	10.20	1.57	(0.90, 2.54)
1984–1990	10	10.33	0.97	(0.46, 1.78)	5	6.18	0.81	(0.26, 1.88)
1991–2006	21	24.90	0.84	(0.52, 1.29)	9	8.86	1.02	(0.47, 1.93)
1963–2006	57	70.81	0.80	(0.61, 1.05)	30	25.30	1.19	(0.80, 1.70)
Hodgkin's lymphoma								
1963–1983[c]	0	3.61	0.00	(0.00, 0.83)	16	11.70	1.37	(0.78, 2.22)
1984–1990	1	1.10	0.91	(0.02, 5.07)	11	5.75	1.91	(0.95, 3.42)
1991–2006	3	2.86	1.05	(0.22, 3.06)	7	7.16	0.98	(0.39, 2.01)
1963–2006	4	7.63	0.52	(0.14, 1.34)	34	24.47	1.39	(0.96, 1.94)
Brain/CNS (including benign)								
1963–1983[c]	21	20.22	1.04	(0.64, 1.59)	8	8.80	0.91	(0.39, 1.80)
1984–1990	4	6.15	0.65	(0.18, 1.67)	7	3.59	1.95	(0.78, 4.02)
1991–2006	20	16.59	1.21	(0.74, 1.86)	5	7.16	0.70	(0.23, 1.63)
1963–2006	45	43.34	1.04	(0.76, 1.39)	20	19.58	1.02	(0.62, 1.57)

	Ages 0–14 years				Ages 15–24 years			
	Observed	**Expected**	**SIR[a]**	**(95% CI)**	**Observed**	**Expected**	**SIR[b]**	**(95% CI)**
Other malignancies								
1963–1983[c]	31	28.67	1.08	(0.73, 1.54)	24	26.23	0.91	(0.59, 1.35)
1984–1990	13	9.35	1.39	(0.74, 2.38)	9	15.46	0.58	(0.27, 1.11)
1991–2006	21	22.21	0.95	(0.58, 1.45)	33	28.46	1.16	(0.80, 1.63)
1963–2006	65	60.67	1.07	(0.83, 1.38)	66	70.47	0.94	(0.73, 1.20)
All malignancies								
1963–1983[c]	78	87.73	0.89	(0.71, 1.11)	64	56.94	1.12	(0.87, 1.45)
1984–1990	28	26.93	1.04	(0.69, 1.51)	32	30.97	1.03	(0.71, 1.46)
1991–2006	65	66.56	0.98	(0.76, 1.26)	54	51.63	1.05	(0.79, 1.37)
1963–2006	171	182.45	0.94	(0.80, 1.09)	150	139.82	1.07	(0.91, 1.26)

a Based on NRCT England, Wales and Scotland registrations excluding 86 cases with no postcode

b Based on registrations from the Northern Region Young Persons Malignant Disease Registry excluding those for Cumbria

c 1969–1983 for 15–24 age group as NRYPMDR data only available from 1969

SIRs which differ significantly from 1 are shown in bold ($p<0.05$), bold and underlined ($p<0.01$)

Table 3.5 Cases and SIRs for individuals aged 0–14 and 15–24 years resident in the remainder of Cumbria[d] from 1963 to 2006

	Ages 0–14 years				Ages 15–24 years			
	Observed	**Expected**	**SIR[a]**	**(95% CI)**	**Observed**	**Expected**	**SIR[b]**	**(95% CI)**
Lymphoid leukaemia								
1963–1983[c]	41	37.02	1.11	(0.79, 1.50)	2	4.46	0.45	(0.05, 1.62)
1984–1990	20	12.53	1.60	(0.98, 2.46)	1	2.83	0.35	(0.01, 1.97)
1991–2006	32	31.64	1.01	(0.69, 1.43)	4	4.62	0.87	(0.24, 2.22)
1963–2006	93	81.41	1.14	(0.93, 1.41)	7	11.89	0.59	(0.24, 1.21)
Other leukaemia								
1963–1983[c]	11	14.49	0.76	(0.38, 1.36)	4	5.11	0.78	(0.21, 2.00)
1984–1990	5	3.50	1.43	(0.46, 3.33)	2	2.44	0.82	(0.10, 2.96)
1991–2006	5	8.69	0.58	(0.19, 1.34)	1	4.72	0.21	(0.01, 1.18)
1963–2006	21	26.66	0.79	(0.49, 1.21)	7	12.30	0.57	(0.23, 1.17)
Total leukaemia								
1963–1983[c]	52	51.50	1.01	(0.76, 1.33)	6	9.57	0.63	(0.23, 1.37)
1984–1990	25	16.03	**1.56**	(1.01, 2.31)	3	5.27	0.57	(0.12, 1.66)
1991–2006	37	40.33	0.92	(0.65, 1.27)	5	9.34	0.54	(0.17, 1.25)
1963–2006	114	108.07	1.05	(0.87, 1.27)	14	24.19	**0.58**	(0.32, 0.97)
Non-Hodgkin lymphoma								
1963–1983[c]	11	10.29	1.07	(0.53, 1.91)	4	4.41	0.91	(0.25, 2.32)
1984–1990	6	2.89	2.08	(0.76, 4.53)	1	4.05	0.25	(0.01, 1.38)
1991–2006	5	7.11	0.70	(0.23, 1.64)	2	4.02	0.50	(0.06, 1.80)
1963–2006	22	20.30	1.08	(0.68, 1.64)	7	12.34	0.57	(0.23, 1.17)
NHL + total leukaemia								
1963–1983[c]	63	61.80	1.02	(0.79, 1.32)	10	13.99	0.71	(0.34, 1.32)
1984–1990	31	18.92	**1.64**	(1.11, 2.33)	4	9.31	0.43	(0.17, 1.10)
1991–2006	42	47.43	0.89	(0.64, 1.20)	7	13.36	0.52	(0.21, 1.08)
1963–2006	136	128.37	1.06	(0.89, 1.26)	21	36.53	**0.57**	(0.36, 0.88)
Hodgkin's lymphoma								
1963–1983[c]	4	6.33	0.63	(0.17, 1.62)	15	15.85	0.95	(0.50, 1.56)
1984–1990	4	2.01	1.99	(0.54, 5.09)	11	8.49	1.30	(0.65, 2.32)
1991–2006	3	5.45	0.55	(0.11, 1.61)	11	10.80	1.02	(0.51, 1.82)
1963–2006	11	13.83	0.80	(0.40, 1.42)	37	35.07	1.06	(0.74, 1.45)
Brain/CNS (including benign)								
1963–1983[c]	46	35.46	1.30	(0.95, 1.73)	10	11.92	0.84	(0.40, 1.54)
1984–1990	6	11.25	0.53	(0.20, 1.16)	2	5.34	0.37	(0.05, 1.35)
1991–2006	26	31.61	0.82	(0.54, 1.21)	8	10.80	0.74	(0.32, 1.46)
1963–2006	78	78.57	0.99	(0.79, 1.25)	20	28.13	0.71	(0.43, 1.09)

	Ages 0–14 years				Ages 15–24 years			
	Observed	**Expected**	**SIR[a]**	**(95% CI)**	**Observed**	**Expected**	**SIR[b]**	**(95% CI)**
Other malignancies								
1963–1983[c]	41	50.28	0.82	(0.58, 1.11)	34	35.25	0.96	(0.67, 1.35)
1984–1990	14	17.12	0.82	(0.45, 1.37)	21	22.67	0.93	(0.57, 1.42)
1991–2006	39	42.31	0.92	(0.66, 1.26)	38	42.90	0.89	(0.63, 1.22)
1963–2006	94	109.99	0.85	(0.69, 1.05)	93	100.68	0.92	(0.75, 1.14)
All malignancies								
1963–1983[c]	154	153.86	1.00	(0.85, 1.18)	69	77.01	0.90	(0.70, 1.14)
1984–1990	55	49.30	1.12	(0.84, 1.46)	38	45.81	0.83	(0.59, 1.14)
1991–2006	110	126.81	0.87	(0.72, 1.05)	64	77.86	0.82	(0.64, 1.06)
1963–2006	319	330.76	0.96	(0.86, 1.08)	171	200.42	**0.85**	(0.73, 0.99)

a Based on NRCT England, Wales and Scotland registrations excluding 86 cases with no postcode

b Based on registrations from the Northern Region Young Persons Malignant Disease Registry excluding those for Cumbria

c 1969–1983 for 15–24 age group as NRYPMDR data only available from 1969

d NRYPMDR registrations do not cover the Barrow county district

SIRs which differ significantly from 1 are shown in bold ($p<0.05$), bold and underlined ($p<0.01$)

Birth cohort analysis

3.34 Data relating to the Seascale birth cohort, both the earlier part of the cohort based on the pre-existing Cumbrian cohort of children born during 1950–1993 (n = 278,665) and the extension to the cohort – those born during 1994–2006 (n = 64,430) – were supplied by the National Health Service Information Centre (NHSIC).

3.35 The NHSIC was unable to supply birth address information for the 1950–1993 part of the cohort so, to enable identification of individuals born in the geographical areas of interest, it was necessary to match the NHSIC version of the cohort with that held at Newcastle University which included the necessary address information. No completely consistent identifiers linked the two versions of the cohort, making this a complex matching exercise, but 98.5% of the records in the NHSIC version of the cohort, taken as the definitive version for this study, were matched to a record from the Newcastle version.

3.36 On close examination of the data it became apparent that the Cumbria cohort as supplied by the NHSIC was missing those individuals born in Cumbria from 1950 to 1991 who had died before the introduction of the Central Health Register Enquiry System (CHRIS) in 1991. Similarly, there were no cancers recorded before 1991 unless the individual survived beyond 1991. The NHSIC subsequently accepted that the version of the cohort it had been using was not the complete Cumbrian cohort, but included only those members who survived beyond or were born after 1991. Despite extensive searches at the NHSIC the complete file could not be located and the NHSIC concluded that it had been discarded during re-organisation.

3.37 To trace all those who died before 1991 in the manual ledgers would have been prohibitively expensive and time consuming. Instead, the University of Newcastle agreed to allow access to the data held on deaths and cancers before 1991. The cancer information held may not be complete and, in many cases, the coding and diagnosis date recorded were not consistent with those

supplied by the NHSIC. To complete the cohort, 7788 individuals from the Newcastle version of the cohort identified as having died before 1991 and a further 3123 individuals identified as having been lost to follow up before 1991 were added. Newcastle had identified 356 cancer diagnoses among the 7788 individuals who had died prior to 1991. To obtain consistent cancer information for cohort members, the NHSIC was asked to check those individuals against the ONS Model 204 cancer records and this information was incorporated into the analyses. Scrutiny of the characteristics of additional cases included in the cohort suggested that their inclusion does not bias the findings of the study.

3.38 Table 3.6 gives SIRs (together with 95% CIs and p-value for the point estimate) for cancer overall and each of the 12 diagnostic subgroups listed earlier for each of the three designated study areas. One case of NHL, a female born in Seascale during 1970–1979 and diagnosed outside Cumbria after 1991 while 15–24 years of age, was mentioned in the seventh COMARE report (COMARE, 2002), but not included in any of the analyses reported there; the case is included in Table 3.6. There is no indication of any significant excesses either of cancer overall or of any individual diagnostic subgroup of cancers diagnosed during 1971–2006 among those born during 1950–2006 in Seascale ward or Cumbria more generally. Further analyses were undertaken stratifying by sex, age group at diagnosis, birth decade and diagnosis decade (the results for cancer overall are presented in Table 3.7). For cancer overall, the reduced cancer risk in both areas of Cumbria outside Seascale was greater in males than females, but otherwise no important variations across the strata were evident.

3.39 Table 3.6, taken from Bunch et al (2014), shows two cases of leukaemia diagnosed in the Seascale birth cohort between 1971 and 2006, which compares with an expected number of 1.2; for LNHL the observed number of cases of four compares with an expected number of 2.48. These excesses are not statistically significant. However, these expected numbers are based upon diagnoses at all ages. If attention is confined to cases of LNHL diagnosed while less than 25 years of age, the observed number of four LNHL cases compares with an expected number of 0.904 (K Bunch, personal communication), a statistically significant excess ($p = 0.027$).

3.40 Further, the observed number of two cases of leukaemia diagnosed during 1971–2006 shown in Table 3.6 is at variance with the data contained in other studies (Gardner et al, 1987b; Draper et al, 1993; COMARE, 2002; Kinlen, 2015b), which report three cases during this period. It is likely that the difficulties encountered and described by Bunch et al (2014) in assembling and monitoring the birth cohorts provide the explanation for this discrepancy, and they discuss a ‘missing case’ born in Seascale that may be case J in Table 2.4 of the fourth report (COMARE, 1996).

Table 3.6 SIRs for total cancer and defined diagnostic subgroups 1971 to 2006 for individuals born in Cumbria 1950 to 2006

	Seascale ward as at 1981				Allerdale and Copeland (excluding Seascale)				Remainder of Cumbria			
	Cancers observed	**Cancers expected**	**SIR**	**(95% CI[a])**	**Cancers observed**	**Cancers expected**	**SIR**	**95% CI[a]**	**Cancers observed**	**Cancers expected**	**SIR**	**95% CI[a]**
Total cancer	21	22.35	0.94	(0.58, 1.44)	1,573	1,796.63	**0.88**	(0.83, 0.92)	2,637	2,901.73	**0.91**	(0.87, 0.94)
Leukaemia	2	1.2	1.67	(0.2, 6.03)	81	102.96	**0.79**	(0.63, 0.98)	151	174.57	0.86	(0.73, 1.01)
Non-Hodgkin lymphoma (NHL)	2	1.28	1.57	(0.19, 5.66)	80	101.42	**0.79**	(0.63, 0.98)	133	164.07	**0.81**	(0.68, 0.96)
Leukaemia + NHL	4	2.48	1.61	(0.44, 4.13)	161	204.38	**0.79**	(0.67, 0.92)	284	338.64	**0.84**	(0.74, 0.94)
Hodgkin's lymphoma	2	0.91	2.19	(0.27, 7.91)	71	73.05	0.97	(0.76, 1.23)	107	120.51	0.89	(0.73, 1.07)
CNS/brain tumours	0	1.71	0.00	(0.00, 1.75)	122	140.65	0.87	(0.72, 1.04)	237	232.99	1.02	(0.89, 1.16)
Melanoma of the skin	1	1.58	0.63	(0.02, 3.53)	109	124.29	0.88	(0.72, 1.06)	200	201.32	0.99	(0.86, 1.14)
Neuroblastoma and ganglioneuroblastoma	0	0.08	0.00	(0.00, 35.41)	9	8.36	1.08	(0.49, 0.05)	15	15.07	1.00	(0.56, 1.64)
Mouth, oropharynx, larynx and URT tumours	1	0.73	1.37	(0.04, 7.61)	52	57.79	0.90	(0.67, 1.18)	95	91.90	1.03	(0.84, 1.26)
GI tract tumours	1	1.35	0.74	(0.02, 4.11)	83	106.37	**0.78**	(0.62, 0.97)	146	167.67	0.87	(0.74, 1.02)
Lung, trachea and LRT tumours	0	0.92	0.00	(0.00, 3.25)	76	73.61	1.03	(0.81, 1.29)	103	115.90	0.89	(0.73, 1.08)
Breast tumours	7	5.18	1.35	(0.54, 2.78)	382	413.00	0.92	(0.84, 1.02)	576	656.99	**0.88**	(0.81, 0.95)
GU tumours	3	4.81	0.62	(0.13, 1.82)	321	381.96	0.84	(**0.75**, 0.94)	562	615.97	**0.91**	(0.84, 0.99)
Thyroid cancer	0	0.44	0.00	(0.00, 6.88)	30	34.74	0.86	(0.58, 1.23)	59	56.85	1.04	(0.79, 1.34)

a CIs based on exact CIs for Poisson counts

SIRs which differ significantly from 1 are shown in bold ($p<0.05$), bold and underlined ($p<0.01$), bold and double underlined ($p<0.001$)

Table 3.7 SIRs for overall cancer from 1971 to 2006 for individuals born in Cumbria from 1950 to 2006

	Seascale ward as at 1981					Allerdale and Copeland (excluding Seascale)					Remainder of Cumbria				
	Person-years	Cancers observed	Cancers expected	SIR	(95% CI[a])	Person-years	Cancers observed	Cancers expected	SIR	(95% CI[a])	Person-years	Cancers observed	Cancers expected	SIR	(95% CI[a])
Overall	37,989.4	21	22.35	0.94	(0.58, 1.44)	3,192,368.7	1,573	1,796.63	**0.88**	(0.83, 0.92)	5,369,045.7	2,637	2,901.73	**0.91**	(0.874, 0.944)
By sex															
Males	20,086.9	6	9.16	0.66	(0.24, 1.43	1,641,891.9	581	723.85	**0.80**	(0.74, 0.87	2,751,666.0	1,023	1,170.41	**0.87**	(0.821, 0.929)
Females	17,902.5	15	13.19	1.14	(0.64, 1.88	1,550,476.8	992	1,072.78	**0.92**	(0.87, 0.98	2,617,379.7	1,614	1731	**0.93**	(0.887, 0.979)
By age category															
0–4	3,429.2	2	0.59	3.39	(0.41, 12.25)	347,272.1	62	60.35	1.03	(0.79, 1.32)	632,624.6	119	110.46	1.08	(0.892, 1.289)
5–9	3,893.1	1	0.39	2.54	(0.06, 14.12)	374,349.2	31	38.43	0.81	(0.55, 1.15)	663,703.4	54	68.36	0.79	(0.593, 1.031)
10–14	4,496.3	0	0.45	0.00	(0.00, 6.67)	398,166.0	32	40.5	0.79	(0.54, 1.12)	686,518.4	69	70.13	0.98	(0.766, 1.245)
15–19	4,982.1	2	0.77	2.60	(0.31, 9.38)	412,852.7	74	64.67	1.14	(0.90, 1.44)	692,213.4	108	108.88	0.99	(0.814, 1.198)
20–24	5,027.3	1	1.23	0.81	(0.02, 4.52)	403,241.2	106	100.17	1.06	(0.87, 1.28)	667,527.8	162	166.82	0.97	(0.827, 1.133)
25–29	4,454.7	3	1.85	1.62	(0.34, 4.75)	352,863.6	110	147.2	**0.75**	(0.61, 0.90)	582,380.8	209	244.5	**0.85**	(0.743, 0.979)
30–34	3,940.7	0	2.67	0.00	(0.00, 1.12)	304,125.9	172	207.51	**0.83**	(0.71, 0.96)	497,200.2	316	340.15	0.93	(0.829, 1.037)
35–39	3,243.1	4	3.38	1.18	(0.32, 3.03)	247,390.1	235	262.03	0.90	(0.79, 1.02)	395,959.3	379	420.27	**0.90**	(0.813, 0.997)
40–44	2,390.0	3	3.96	0.76	(0.16, 2.21)	182,096.4	258	305.46	**0.84**	(0.75, 0.95)	285,825.4	394	480.66	**0.82**	(0.741, 0.905)
45–49	1,464.7	3	3.99	0.75	(0.16, 2.20)	113,935.5	255	309.77	**0.82**	(0.73, 0.93)	177,574.8	458	484.64	0.95	(0.86, 1.036)
50–54	632.8	2	2.81	0.71	(0.09, 2.57)	51,526.4	218	229.43	0.95	(0.83, 1.09)	80,299.5	324	357.49	0.91	(0.81, 1.011)
55–60	35.5	0	0.24	0.00	(0.00, 12.34)	4,549.8	20	31.1	**0.64**	(0.39, 0.99)	7,218.1	45	49.36	0.91	(0.665, 1.22)

	Seascale ward as at 1981					Allerdale and Copeland (excluding Seascale)					Remainder of Cumbria				
	Person-years	**Cancers observed**	**Cancers expected**	**SIR**	**(95% CI[a])**	**Person-years**	**Cancers observed**	**Cancers expected**	**SIR**	**(95% CI[a])**	**Person-years**	**Cancers observed**	**Cancers expected**	**SIR**	**(95% CI[a])**
By birth decade															
1950–1959	11,468.7	11	13.29	0.83	(0.41, 1.48)	892,026.2	898	1,053.54	**0.85**	(0.80, 0.91)	1,389,907.6	1,462	1,645.13	**0.89**	(0.844, 0.935)
1960–1969	12,798.0	6	6.12	0.98	(0.36, 2.13)	950,696.9	398	465.22	**0.86**	(0.77, 0.94)	1,568,925.6	698	761.17	**0.92**	(0.85, 0.988)
1970–1979	7,178.1	2	1.86	1.07	(0.13, 3.88)	647,392.7	181	164.98	1.10	(0.94, 1.27)	1,148,765.5	275	293.2	0.94	(0.83, 1.056)
1980–1989	4,462.6	2	0.74	2.70	(0.33, 9.75)	442,082.7	57	72.31	0.79	(0.60, 1.02)	757,222.0	125	123.47	1.01	(0.843, 1.206)
1990–1999	1,698.0	0	0.26	0.00	(0.00, 11.63)	222,688.6	31	33.23	0.93	(0.63, 1.32)	428,702.0	60	63.95	0.94	(0.716, 1.208)
2000–2006	383.9	0	0.08	0.00	(0.00, 39.26)	37,481.6	8	7.35	1.09	(0.47, 2.15)	75,523.1	17	14.8	1.15	(0.669, 1.839)
By diagnosis decade															
1971–1980	8,402.0	2	1.23	1.63	(0.20, 5.88)	648,852.1	86	96.04	0.90	(0.72, 1.11)	1,066,198.6	149	156.42	0.95	(0.806, 1.118)
1981–1990	10,266.1	6	3.13	1.92	(0.70, 4.17)	838,986.6	236	249.59	0.95	(0.83, 1.07)	1,391,398.1	372	401.54	0.93	(0.835, 1.026)
1991–2000	11,680.1	7	8.09	0.86	(0.35, 1.78)	1,021,234.6	518	653.28	**0.79**	(0.73, 0.86)	1,732,068.7	933	1,056.26	**0.88**	(0.828, 0.942)
2001–2006	7,641.2	6	9.89	0.61	(0.22, 1.32)	683,295.5	733	797.71	**0.92**	(0.85, 0.99)	1,179,380.3	1,183	1,287.5	**0.92**	(0.867, 0.973)

a CIs based on exact CIs for Poisson counts

SIRs which differ significantly from 1 are shown in bold ($p<0.05$), bold and underlined ($p<0.01$), bold and double underlined ($p<0.001$)

3.41 Table 3.8 summarises the findings for LNHL in the Seascale birth cohort for the 0–24 year age group, on the basis of two or three cases of leukaemia and two cases of NHL diagnosed between 1971 and 2006. When three rather than two observed cases of leukaemia are adopted, the observed number of five cases of LNHL diagnosed among those born in Seascale during 1950–2006 and diagnosed while 0–24 years of age during 1971–2006 compares with an expected number of 0.904, which is a highly significant excess ($p = 0.005$).

Table 3.8 SIRs for LNHL in the 0–24 age group during 1971 to 2006 for individuals born in Seascale during 1950 to 2006

Cases observed	Cases expected	SIR	(95% CI)	p-value (2-sided)
4	0.904	4.43	(1.21, 11.33)	0.027
5	0.904	5.53	(1.80, 12.91)	0.005

3.42 Table 3.8 shows statistically significant excesses of LNHL for both four and five observed cases. The first study of the Seascale birth cohort found five deaths from, and one non-fatal case of, leukaemia from 1950 to June 1986 among those born during 1950–1983; two of these deaths and the non-fatal case occurred after 1970 (Gardner et al, 1987b). In addition to a death from NHL occurring before 1971, one case of (non-fatal) NHL diagnosed after 1970 was also identified in the Seascale birth cohort by Gardner et al (1987b), while the latest case of NHL was diagnosed in 1996 and has not been included in previous analyses (see paragraph 3.38 above). These numbers are also consistent with the eight cases of LNHL identified by Dickinson and Parker (2002) as born in Seascale from 1950 onwards and diagnosed during 1950–1991while under 25 years of age.

3.43 In contrast to the results for LNHL incidence among the 0–24 year age group in the Seascale birth cohort, there is an absence of cases diagnosed beyond 24 years of age, which compares with 1.58 cases expected, a non-significant deficit.

3.44 COMARE had intended to review the pathology of cases for this report, but this was not possible due to the dissolution of the Childhood Cancer Research Group and delays in transferring its data into the national database. It should be noted, however, that the review undertaken for the fourteenth report did include a characterisation of the cases of acute leukaemias in a set of young children (0–4 years of age) with residence at birth within 10 km of a nuclear power station (including Calder Hall on the Sellafield site), born between 1962 and 1999 (and therefore diagnosed between 1962 and 2004). This exercise would have included some of the Seascale LNHL cases.

Results relating to Dounreay

Cross-sectional analysis

3.45 The numbers of cancer cases at ages 0–14 and 15–24 years identified in the civil parishes of Thurso and Reay during 1963–2006 are shown in Table 3.9 and cancer cases in the same age groups identified in the remaining civil parishes of Caithness are presented in Table 3.10. In addition to showing the numbers of cases for cancer overall and for each of the predefined diagnostic outcome groups, the tables show SIRs with 95% CIs. For childhood cancers the national comparative rates were derived from NRCT registrations for England, Wales and Scotland, whereas for the young adult cancers (ages 15–24 years) comparative rates were based on the GROs registrations for Scotland.

3.46 Table 3.9 shows no excess of childhood cancer among 0–14 year olds or 15–24 year olds resident in Thurso and Reay civil parishes during the period 1991–2006. During this period there were two cases of cancer among 0–14 year olds and five cases of cancer among 15–24 year olds; none of these cases was leukaemia or NHL. For cancer overall, the SIR for the period 1991–2006 was 0.51 (95% CI 0.06, 1.84) for the 0–14 year age group and 0.95 (95% CI 0.31, 2.21) for the 15–24 year age group.

Table 3.9 Cases and SIRs for individuals aged 0–14 and 15–24 years resident in Thurso and Reay civil parishes

	Ages 0–14 years				Ages 15–24 years			
	Observed	**Expected**	**SIR[a]**	**95% CI**	**Observed**	**Expected**	**SIR[b]**	**95% CI**
Lymphoid leukaemia								
1963–1983	4	1.54	2.59	(0.70, 6.63)	0	0.25	0.00	
1984–1990	1	0.46	2.18	(0.05, 12.14)	2	0.11	18.76	(2.27, 67.72)
1991–2006	0	0.97	0.00		0	0.22	0.00	
1963–2006	5	3.04	1.65	(0.53, 3.84)	2	0.57	3.48	(0.42, 12.56)
Other leukaemia								
1963–1983	1	0.60	1.66	(0.04, 9.25)	0	0.40	0.00	
1984–1990	0	0.13	0.00		0	0.11	0.00	
1991–2006	0	0.27	0.00		0	0.33	0.00	
1963–2006	1	0.99	1.01	(0.03, 5.63)	0	0.85	0.00	
Total leukaemia								
1963–1983	5	2.14	2.33	(0.75, 5.43)	0	0.65	0.00	
1984–1990	1	0.59	1.71	(0.04, 9.52)	2	0.22	9.22	(1.12, 33.28)
1991–2006	0	1.24	0.00		0	0.55	0.00	
1963–2006	6	4.03	1.49	(0.55, 3.25)	2	1.43	1.40	(0.17, 5.05)
Non-Hodgkin lymphoma								
1963–1983	0	0.43	0.00		0	0.43	0.00	
1984–1990	0	0.11	0.00		0	0.17	0.00	
1991–2006	0	0.22	0.00		0	0.32	0.00	
1963–2006	0	0.76	0.00		0	0.92	0.00	
NHL + total leukaemia								
1963–1983	5	1.97	1.94	(0.63, 4.52)	0	1.08	0.00	
1984–1990	1	0.56	1.45	(0.04, 8.08)	2	0.38	5.20	(0.63, 18.77)
1991–2006	0	1.19	0.00		0	0.87	0.00	
1963–2006	6	3.79	1.25	(0.49, 2.73)	2	2.35	0.85	(0.10, 3.07)
Hodgkin's lymphoma								
1963–1983	0	0.26	0.00		1	1.07	0.93	(0.02, 5.18)
1984–1990	0	0.07	0.00		1	0.36	2.75	(0.07, 15.32)
1991–2006	0	0.17	0.00		0	0.74	0.00	
1963–2006	0	0.52	0.00		2	2.18	0.92	(0.11, 3.32)

	Ages 0–14 years				Ages 15–24 years			
	Observed	**Expected**	**SIR[a]**	**95% CI**	**Observed**	**Expected**	**SIR[b]**	**95% CI**
Brain/CNS (including benign)								
1963–1983	2	1.48	1.35	(0.16, 4.87)	1	0.54	1.84	(0.05, 12.25)
1984–1990	0	0.41	0.00		0	0.24	0.00	
1991–2006	1	0.97	1.03	(0.03, 5.74)	2	0.51	3.90	(0.47, 14.08)
1963–2006	3	2.93	1.02	(0.21, 2.98)	3	1.31	2.29	(0.47, 6.69)
Other malignancies								
1963–1983	0	2.09	0.00		2	3.22	0.62	(0.08, 2.24)
1984–1990	0	0.63	0.00		2	1.41	1.42	(0.17, 5.11)
1991–2006	1	1.30	0.77	(0.02, 4.29)	3	3.14	0.96	(0.20, 2.79)
1963–2006	1	4.10	0.24	(0.01, 1.34)	7	7.85	0.89	(0.36, 1.84)
All malignancies								
1963–1983	7	6.41	1.09	(0.44, 2.25)	4	5.91	0.68	(0.18, 1.74)
1984–1990	1	1.80	0.55	(0.01, 3.06)	5	2.40	2.08	(0.67, 4.85)
1991–2006	2	3.91	0.51	(0.06, 1.84)	5	5.26	0.95	(0.31, 2.21)
1963–2006	10	12.34	0.81	(0.39, 1.49)	14	13.69	1.02	(0.56, 1.71)

a Expectations and SIRs based on NRCT England, Wales and Scotland registrations excluding 86 cases with no postcode
b Expectations and SIRs based on GROs registrations for Scotland

3.47 For the remaining civil parishes of Caithness, there were six cases of childhood cancer during the period 1991–2006 and four cases of cancer among 15–24 year olds; there was one case of childhood leukaemia (Table 3.10). None of the individual SIRs for this period was significantly raised (or reduced) and for cancer overall the SIR was 0.84 (95% CI 0.31, 1.83) for 0–14 year olds and 0.46 (95% CI 0.13, 1.18) for 15–24 year olds.

Table 3.10 Cases and SIRs for individuals aged 0–14 and 15–24 years resident in Caithness outside Thurso and Reay civil parishes

	Ages 0–14 years				Ages 15–24 years			
	Observed	**Expected**	**SIR[a]**	**95% CI**	**Observed**	**Expected**	**SIR[b]**	**95% CI**
Lymphoid leukaemia								
1963–1983	3	2.35	1.28	(0.26, 3.74)	0	0.39	0.00	(0.00, 7.77)
1984–1990	3	0.80	3.75	(0.77, 10.95)	0	0.17	0.00	(0.00, 18.01)
1991–2006	0	1.78	0.00	(0.00, 1.68)	0	0.36	0.00	(0.00, 8.38)
1963–2006	6	4.98	1.21	(0.44, 2.64)	0	0.91	0.00	(0.00, 3.28)
Other leukaemia								
1963–1983	2	0.92	2.17	(0.26, 7.83)	0	0.63	0.00	(0.00, 4.75)
1984–1990	0	0.22	0.00	(0.00, 13.41)	0	0.17	0.00	(0.00, 17.388)
1991–2006	0	0.49	0.00	(0.00, 6.13)	0	0.55	0.00	(0.00, 5.46)
1963–2006	2	1.63	1.23	(0.15, 4.44)	0	1.35	0.00	(0.00, 2.21)
Total leukaemia								
1963–1983	5	3.27	1.53	(0.50, 3.56)	0	1.02	0.00	(0.00, 2.946)
1984–1990	3	1.02	2.93	(0.60, 8.56)	0	0.34	0.00	(0.00, 8.847)
1991–2006	0	2.27	0.00	(0.00, 1.32)	0	0.91	0.00	(0.00, 3.31)
1963–2006	8	6.61	1.21	(0.52, 2.38)	0	2.27	0.00	(0.00, 1.32)
Non-Hodgkin lymphoma								
1963–1983	2	0.65	3.06	(0.37, 11.05)	0	0.67	0.00	(0.00, 4.48)
1984–1990	0	0.18	0.00	(0.00, 16.25)	1	0.26	3.83	(0.10, 21.33)
1991–2006	1	0.40	2.50	(0.06, 13.93)	0	0.53	0.00	(0.00, 5.62)
1963–2006	3	1.24	2.42	(0.50, 7.07)	1	1.47	0.68	(0.02, 3.79)
NHL + total leukaemia								
1963–1983	7	3.93	1.78	(0.71, 3.67)	0	1.69	0.00	(0.00, 1.78)
1984–1990	3	1.21	2.49	(0.51, 7.27)	1	0.60	1.67	(0.04, 9.30)
1991–2006	1	2.67	0.37	(0.01, 2.06)	0	1.44	0.00	(0.00, 2.08)
1963–2006	11	7.85	1.40	(0.70, 2.51)	1	3.73	0.27	(0.01, 1.50)
Hodgkin's lymphoma								
1963–1983	0	0.40	0.00	(0.00, 7.45)	2	1.67	1.20	(0.15, 4.33)
1984–1990	0	0.13	0.00	(0.00, 23.35)	0	0.57	0.00	(0.00, 5.28)
1991–2006	0	0.31	0.00	(0.00, 9.77)	1	1.22	0.82	(0.02, 4.57)
1963–2006	0	0.85	0.00	(0.00, 3.54)	3	3.47	0.87	(0.18, 2.54)
Brain/CNS (including benign)								
1963–1983	1	2.25	0.44	(0.01, 2.45)	0	0.85	0.00	(0.00, 3.523)
1984–1990	0	0.72	0.00	(0.00, 4.17)	0	0.37	0.00	(0.00, 8.00)
1991–2006	4	1.78	2.25	(0.61, 5.76)	0	0.85	0.00	(0.00, 3.54)
1963–2006	5	4.80	1.04	(0.34, 2.42)	0	2.08	0.00	(0.00, 1.44)

	Ages 0–14 years				Ages 15–24 years			
	Observed	**Expected**	**SIR[a]**	**95% CI**	**Observed**	**Expected**	**SIR[b]**	**95% CI**
Other malignancies								
1963–1983	2	3.19	0.63	(0.08, 2.26)	3	5.03	0.60	(0.12, 1.74)
1984–1990	1	1.09	0.92	(0.02, 5.10)	2	2.20	0.91	(0.11, 3.27)
1991–2006	1	2.38	0.42	(0.01, 2.34)	3	5.17	0.58	(0.12, 1.69)
1963–2006	4	6.72	0.60	(0.16, 1.52)	8	12.46	0.64	(0.28, 1.26)
All malignancies								
1963–1983	10	9.77	1.02	(0.49, 1.88)	5	9.24	0.54	(0.17, 1.26)
1984–1990	4	3.15	1.27	(0.35, 3.25)	3	3.75	0.80	(0.16, 2.34)
1991–2006	6	7.13	0.84	(0.31, 1.83)	4	8.68	0.46	(0.13, 1.18)
1963–2006	20	20.22	0.99	(0.60, 1.52)	12	21.74	0.55	(0.28, 0.96)

a Expectations and SIRs based on NRCT England, Wales and Scotland registrations excluding 86 cases with no postcode
b Expectations and SIRs based on GROs registrations for Scotland

Birth cohort analysis

3.48 The issue arising from non-retention of records relating to individuals dead at the time of the introduction of CHRIS for the Cumbrian cohort was mirrored in the Dounreay cohort by issues surrounding the introduction of Community Health Index (CHI) numbers in Scotland. The information on cancer diagnoses among Dounreay cohort members was restricted to cancers in those still alive at the introduction of consolidated CHI numbers in 1996. As it was not possible to follow up individuals who had died before this time, an accurate assessment of the true person-years at risk could not be made. This meant that comparisons of cancer incidence in the study cohorts and any national estimate of cancer incidence, time from study entry (birth) to study exit (cancer diagnosis or 31 December 2006), was compared for those born in the two defined study areas instead.

3.49 The cohort identified by the ISD included 8091 individuals; 3932 of these had been born in the area closest to Dounreay (postcode sector KW14-7) and 4159 in the three surrounding postcode sectors.

3.50 Cancer notifications during 1971–2006 were supplied by the ISD on 164 individuals within the Dounreay cohort. However, only 93 of these notifications related to a malignant neoplasm (excluding non-melanoma skin cancers) or a non-malignant brain tumour; 42 of these cancers arose in individuals born in the area closest to Dounreay (postcode sector KW14-7) and the remaining 51 in individuals born in the area further from Dounreay.

Table 3.11 Total cancer incidence during 1971–2006 within the Dounreay birth cohort (births during 1950–2006)

By age (years)	Number of people at risk	Cumulative number of cancers	% number of cancers	(95% CI)
Individuals resident at birth in postcode sector KW14-7				
5	3,656	2	99.95	(99.79, 99.99)
10	3,370	2	99.95	(99.79, 99.99)
15	3,063	3	99.92	(99.74, 99.97)
25	2,238	12	99.57	(99.24, 99.76)
35	1,496	23	98.95	(98.40, 99.31)
45	513	34	97.86	(96.89, 98.54)
55	45	42	92.71	(87.03, 95.96)
Individuals resident at birth in postcode sectors KW12-6, KW13-6 and KW14-8				
5	3,844	3	99.92	(99.77, 99.98)
10	3,535	5	99.87	(99.69, 99.95)
15	3,196	7	99.81	(99.60, 99.91)
25	2,444	14	99.57	(99.27, 99.75)
35	1,693	26	99.00	(98.51, 99.32)
45	688	37	98.01	(97.15, 98.61)
55	85	51	93.26	(89.39, 95.75)
Log-rank test for equality of survivor functions gives a chi squared statistic of 0 on 1 degree of freedom (p = 0.99)				

3.51 Table 3.11 shows the overall cancer incidence for those born in each of the two study areas. Log-rank tests (p = 0.99) confirm that there is no statistical difference between the overall cancer incidence of the two subgroups within the cohort. Where there were sufficient numbers, incidence for the diagnostic groups (see paragraph 3.28) within the two subgroups of the cohort were compared (data not shown) and no differences were found.

Conclusions from the latest cancer incidence analyses

3.52 The Cumbrian cross-sectional analyses show that the significant excess incidence of childhood (0–14 years of age) cancer in Seascale observed previously (based on seven cases of leukaemia and non-Hodgkin's lymphoma, LNHL, incident between 1963 and 1990) is not evident in more recent data covering the period 1991–2006, when no case of childhood LNHL occurred.

3.53 There has been a single new case of cancer among 15–24 year olds living in Seascale ward during the period 1991–2006, a lymphoid leukaemia diagnosed in 1991.

3.54 There is no excess incidence of LNHL or other cancers in young people (0–24 years of age) living in the remainder of the two districts surrounding Sellafield, or in the rest of Cumbria.

3.55 In comparison to the (non-significant) two-fold excess incidence of LNHL among the 0–24 years age group in Thurso and Reay parishes during 1963–1990 (based on six cases of leukaemia and two of NHL), there has been no case of LNHL incident in these parishes during 1991–2006. The incidence of other cancers in young people living in these two parishes is not unusual.

3.56 The Cumbrian birth cohort analyses show a significant excess incidence of LNHL among 0–24 year olds born in Seascale in 1950–2006 and diagnosed during 1971–2006, consistent with previous analyses. However, for all ages at diagnosis (ie including those diagnosed at 25 years of age and older), there is no significant excess of LNHL or other cancers in the Seascale birth cohort. For those born during 1950–2006 in the remainder of the two districts surrounding Sellafield, and in the rest of Cumbria, there are highly significant deficits of cases of cancer during 1971–2006.

3.57 There is no excess cancer incidence evident in those members of the Dounreay birth cohort born closest to Dounreay as compared to those cohort members born further away from the nuclear installation.

Seascale and Dounreay schools cohorts

3.58 Following a recommendation in the Black report, a cohort study of children attending schools in Seascale, but born elsewhere, was established and the findings reported by Gardner et al (1987a). COMARE wished to update this study, but practical difficulties have been encountered in complying with current research governance regulations, which are much more stringent than those in place when the original study was designed. COMARE is continuing to explore how the analysis might be conducted and would intend to publish an addendum to this report if a way to proceed can be found.

3.59 A study with similar methods and objectives was carried out in relation to populations living near Dounreay by Black et al (1992). The birth cohort component of this exercise was effectively reconstructed for the purposes of the current study. It appears, however, that reconstruction of the schools cohort is infeasible as the source data are no longer available.

Summary and overall conclusions on cancer incidence near Sellafield and Dounreay

3.60 In 1984, the report of the Black Advisory Group noted an unusually high incidence of childhood leukaemia in the west Cumbrian village of Seascale over the previous three decades, although this was based upon just seven recorded cases. The Group recommended further epidemiological studies to clarify the situation.

Sellafield

3.61 Two of the recommended studies were the Seascale birth and schools cohorts studies (Gardner et al, 1987a,b), which found a significant, approximately ten-fold, excess of childhood leukaemia mortality among those born in Seascale since 1950, but no apparent excess in those born since 1950 and attending schools in Seascale but born elsewhere. In a later study, however, Kinlen (1993) found broadly similar significant excesses of cases of childhood leukaemia among those born in Seascale, and among those born elsewhere but diagnosed while resident in the village.

3.62 A further recommended study was the west Cumbria leukaemia and lymphoma case–control study, which suggested that paternal exposure to radiation while working at Sellafield before the conception of a child could explain the excess cases in Seascale (Gardner et al, 1990a,b). This explanation for the raised rate of incidence of childhood leukaemia in Seascale has now been discounted, and will be examined further in Chapter 6.

3.63 The fourth recommended epidemiological study was an updated geographical study of cancer incidence among young people living in wards of north and northwest England (Craft et al, 1993). This study confirmed the unusually high rate of incidence of leukaemia and NHL in Seascale ward during 1968–1985, but apart from one other ward (Egremont North), the incidence rates in other wards near Sellafield were not exceptional.

3.64 In 1996, the fourth COMARE report reviewed the evidence that had been produced since publication of the report of the Black Advisory Group. In addition to those studies summarised above, a study of cancer incidence among young people in Cumbria during 1963–1983 and 1984–1990 (Draper et al, 1993) was considered. The high rate of leukaemia and NHL in Seascale during 1963–1983 was confirmed, and this was found to extend into 1984–1990. However, the high rate did not extend to the remainder of the two districts around Sellafield, or to other cancers among young people, or to older people. Further, a study by Bithell et al (1994), which had investigated childhood leukaemia and NHL incidence in small areas (wards) within 25 km of nuclear installations in England and Wales during 1966–1987, found a non-significant excess of cases within 25 km of Sellafield, but a highly significant trend of increasing risk with decreasing distance from Sellafield; these findings were entirely due to the excess of cases in Seascale ward.

3.65 Kinlen et al (1995) examined the incidence of childhood (0–14 years of age) leukaemia and NHL in parishes largely within 10 km of Sellafield during 1947–1993, and reported an observed number of cases of 18, which compared with an expected number of 11.3, producing a non-significantly elevated O/E ratio of 1.6 (95% CI 0.94, 2.5). Seascale accounted for eight observed cases against 0.9 expected, a highly significant O/E ratio of 8.9 (95% CI 3.8, 17.5), but for the 10 km area outside Seascale the O/E of 10/10.4 is unremarkable.

3.66 In 2005, the tenth COMARE report presented the numbers of observed and expected cases of leukaemia and NHL among children (less than 15 years of age) living within 25 km of nuclear installations in Great Britain, including Sellafield, during 1969–1993; this was an update of the study of Bithell et al (1994) considered in the fourth report. A non-significant excess of cases was found around Sellafield and a significant trend of risk with distance (due to the Seascale cases), although the degree of significance had reduced with the extended timescale.

3.67 The geographical study of cancer incidence among those 0–24 years of age living around Sellafield presented in this report now covers the period 1963–2006, and the birth cohort study now includes cases of cancer diagnosed during 1971–2006 among births throughout Cumbria during 1950–2006. The highly unusual rate of incidence of leukaemia and NHL among young people in Seascale during 1963–1990 was confirmed, but of note is the absence of a significantly raised risk of leukaemia and NHL in Seascale during 1991–2006, when only one case was recorded (in 1991). A significant excess of leukaemia and NHL in the 0–24 year age group during 1971–2006 among those born in Seascale during 1950–2006 was confirmed, with only one further case (of NHL diagnosed in 1996 outside Cumbria) being added to previously identified cases. No case at older ages was recorded during this period.

3.68 In summary, there have been highly unusual incidence rates of leukaemia and NHL among young people either living or born in Seascale over the four decades from 1950, but during 1991–2006 these high incidence rates have reduced to unexceptional levels. There is little evidence that the earlier raised incidence of leukaemia and NHL in young people living or born in Seascale was reflected in other cancers, other age groups, or other areas in the vicinity of Sellafield.

Dounreay

3.69 In 1988, the second COMARE report found that leukaemia incidence during 1968–1984 among those less than 25 years of age living in an area within 25 km of Dounreay was unusual. This was particularly so for those living within 12.5 km of Dounreay during 1979–1984 and when leukaemia was

combined with NHL. Further epidemiological studies, including a case–control study and birth and schools cohort studies, were recommended.

3.70 The two cohort studies found excess rates of leukaemia incidence, which were statistically significant only in the schools cohort. The case–control study did not reveal reliable evidence for any particular factor influencing the risk of leukaemia and NHL in Caithness, and found that paternal occupation at Dounreay before a child's conception could not provide the explanation for the observed excess of cases.

3.71 A study updating the incidence of leukaemia and NHL among young people within 25 km of Dounreay during 1985–1991 found an excess of cases that was of borderline statistical significance, leading to an overall O/E ratio of case numbers during 1968–1991 of 12/5.2, which was statistically significant.

3.72 In 2005, the tenth COMARE report presented the number of observed cases of childhood (less than 15 years of age) leukaemia and NHL within 25 km of Dounreay during 1969–1993 in relation to the expected number: O/E = 9/3.87 = 2.33, a significant excess. During the same period, the O/E ratio of cases of childhood solid cancers was 3/6.29 = 0.48, a non-significant deficit.

3.73 In the present report, the geographical study of cancer incidence among those 0–24 years of age living around Dounreay now covers the period 1963–2006. During 1963–1990 in the Thurso and Reay parishes, eight cases of leukaemia and NHL were observed against 3.99 cases expected, a marginally significant excess consistent with previous reports. However, during 1991–2006 no case of leukaemia or NHL was observed, whereas 2.06 cases were expected, a non-significant deficit. In the remainder of Caithness the rates of incidence were not unusual.

3.74 In summary, there has been an unusual incidence rate of leukaemia and NHL among people living around Dounreay during the period 1963–1990, but in 1991–2006 this high incidence rate has reduced to an unexceptional level. There is no evidence that the earlier raised incidence of leukaemia and NHL in young people living around Dounreay was reflected in other cancers or in the rest of Caithness.

CHAPTER 4

INCIDENCE OF THYROID CANCER IN NORTHWEST ENGLAND

Background

4.1 The fourth COMARE report recommended that it would be of interest to determine whether any cancer excess now occurred in age groups older than 25 years (COMARE, 1996). The remit of the present report was expanded to include a study of thyroid cancers specifically in the counties of Cumbria and Lancashire in northwest England (McNally et al, 2016). This is of importance also because of the recent upsurge of interest in thyroid cancer rates around the Chernobyl and Fukushima plants following major accidents at these sites.

4.2 Those who were children at the time of the Windscale nuclear reactor fire in 1957 are now of an age where the background risk of thyroid cancer incidence is rising and so it is necessary to establish whether a proportion of any increase could be linked to the 1957 accident. In addition, following discussions with the Department of Health, the Nuclear Decommissioning Authority (NDA) and Sellafield Ltd, COMARE obtained more accurate data on Sellafield historical discharges (see paragraph 5.13). These data indicate radioiodine discharges in excess of those hitherto known at times other than that of the fire.

4.3 The Windscale fire at Sellafield in 1957 released radioactive materials to the atmosphere, including 1800 TBq of iodine-131 (radioactive half-life of 8 days), which led to a local milk ban being established (Arnold, 2007; Wakeford, 2007b). The plume of radioactive material from the reactor fire initially travelled to the northeast before then being taken by a northwesterly wind to the southeast, over southern Cumbria and Lancashire to the rest of England and mainland Europe (Garland and Wakeford, 2007; Johnson et al, 2007).

4.4 Iodine-131 localises in the thyroid gland. Radiation doses to the thyroids of local children from iodine-131 released during the Windscale fire were measured just after the accident up to a maximum of 160 mGy, although the majority of measured thyroid doses were much lower, in the range 10–20 mGy within 20 km of Sellafield and <10 mGy beyond beyond (Crick and Linsley, 1984; Dunster et al, 2007). During the Chernobyl nuclear reactor accident in Ukraine in 1986, 1800 PBq of iodine-131 was released to the atmosphere (1000 times greater than that from the Windscale accident) and tens of thousands of children in the heavily contaminated regions of the former USSR received thyroid doses >1 Gy, mainly as a result of drinking contaminated milk, due to the lack of prompt countermeasures. This high level of exposure to a large number of children caused several thousand additional cases of thyroid cancer, with future cases predicted.

4.5 It is not anticipated that the scale of exposure to iodine-131 that occurred following the Chernobyl accident was experienced after the Windscale accident. Nonetheless, during the late-1980s, the then director of the NRPB estimated from information available at that time that around 60 thyroid cancers would result from exposure to iodine-131 released during the Windscale accident (Clarke, 1989, 1990). These predicted excess thyroid cancers will be distributed geographically according to the areal distribution of the collective

thyroid dose (under the conventional assumption of a linear no-threshold dose response), so that although the highest individual thyroid doses will have occurred in Cumbria, followed by Lancashire, only about one-sixth of the collective thyroid dose was received by the population of Cumbria, the remainder being received principally by the population of the rest of England (Crick and Linsley, 1984).

4.6 Standard radiation-induced thyroid cancer risk models, based on the Japanese atomic bomb survivors and those with medical exposures, demonstrate that the excess risk of thyroid cancer is greatest for those exposed as infants and young children, and that the risk falls with increasing age at exposure. For an age at exposure of 20 years and older, it is difficult to reliably detect any radiation-induced excess risk of thyroid cancer.

4.7 Further, the standard thyroid cancer risk models predict that the temporal evolution of this excess thyroid cancer risk is best expressed by the excess relative risk (ERR, the proportional increase in risk over the background risk in the absence of the exposure). The ERR, after a minimum latent period of around 5 years, remains constant over the remaining lifetime of the exposed individual, possibly with some weak attenuation with increasing time since exposure or attained age. Consequently, the risk models predict that most of the excess thyroid cancer cases will occur when the background risk is highest, which means that for those exposed at young ages the risk will be expressed mainly in later life, particularly for females who have a higher background risk of thyroid cancer than males.

4.8 Therefore, the predicted excess risk of thyroid cancer resulting from exposure to iodine-131 released during the Windscale accident will be expressed mainly through excess cases spread over England and over several decades – around ten additional cases would be predicted to occur among those exposed as young children in Cumbria, but most of these cases would occur later in the lives of these individuals. Until now, no epidemiological study specifically investigating whether any excess risk of thyroid cancer in those areas most affected by contamination of iodine-131 from the Windscale accident has been conducted. Further, since the Windscale fire iodine-131 release estimate has been revised upwards (although this is unlikely to have significantly affected thyroid dose estimates as these are largely based upon environmental monitoring data), and risk models for radiation-induced thyroid cancer have become more sophisticated (especially for those exposed as children), estimates of thyroid cancers caused by iodine-131 released during the Windscale accident may have increased.

Methods

4.9 Data on all cases of thyroid cancer registered as diagnosed in patients less than 85 years of age resident in England during 1974–2012 were provided by the National Cancer Registry. Registration of cancer incidence in England at the National Cancer Registry started in 1971, but the reorganisation of local government in England in 1974 led to county boundary changes that affected both Cumbria and Lancashire and so the latter year was chosen as the start date for this study. The end date of 2012 was the last year for which registration was considered complete at the time of the analysis. Case data were provided for three areas of residence at diagnosis: the (post-1974) county of Cumbria, the (post-1974) county of Lancashire and the ‘rest of England’.

4.10 Since individuals were potentially identifiable from the cancer registration data, a data sharing agreement was negotiated with the Public Health England (PHE) Office for Data Release; this stipulated the destruction of all copies of the original data in the investigators’ possession on completion of

the analysis and that the published results would not permit identification of individuals.

4.11 Mid-year population data for these three areas were obtained from the ONS from 1974–2012. Population estimates were obtained separately for males and females for 17 five-year age groups: 0–4, 5–9, 10–14, …, 80–84. Birth data for the three areas for the years 1929–1973 were obtained from the ONS also.

4.12 The case data were grouped into seventeen 5-year calendar periods of birth: 1929–1933, 1934–1938, …, 1969–1973 for males and females and for each of the three areas of interest (Cumbria, Lancashire and the rest of England). These were chosen so that one period comprised those born in 1954–1958, thus including those exposed *in utero* and at a young age at the time of the Windscale fire in 1957.

4.13 Since the areas of birth of the cases and the corresponding populations were not available, an approximate method was used to obtain incidence rates by period of birth. First, annual population estimates for the seventeen 5-year attained age groups were used to construct age- and sex-specific populations for each of the three areas for seven 5-year calendar periods of diagnosis, 1974–1978, 1979–1983, …, 2004–2008, and one 4-year period, 2009–2012; age-period tables of population data were then constructed.

4.14 Age group, period of diagnosis and period of birth are intrinsically related (McNally et al, 1997), so labelling age groups (i) as 1, 2, …, 17, corresponding to 0–4, 5–9, …, 80–84 years, and diagnosis periods (j) as 1, 2, …, 8, corresponding to 1974–1978, 1979–1983, …, 2009–2012, approximate ten-year overlapping birth periods (1929–1938, 1934–1943, …, 1964–1973) are defined as $k = 17 – i + j$. For example, a diagnosis at age 15–19 years during the period 1974–1978 corresponds to the 10-year birth period 1954–1963.

4.15 Rates were calculated also for 5-year birth periods, estimating populations by dividing the 10-year overlapping birth period populations by two. This approach has been criticised because it assumes no change in birth rates or early mortality rates (Clayton and Schifflers, 1987a,b; McNally et al, 1997). Nevertheless it provides an approximate estimation.

4.16 Using this approach, combined male and female, age-group-specific incidence rates were calculated for each combination of diagnosis period and birth period. An additional analysis was carried out after generating 5-year birth period populations proportionally according to annual birth rates obtained from the ONS, but the results were little changed and so are not reported.

4.17 The primary focus of this study was the incidence of thyroid cancer among those aged 0–19 years in 1958 (that is, those young at the time of the accident, and allowing for those who were potentially exposed to iodine-131 *in utero*). Consequently, approximate incidence rates were calculated for those aged 0–19 years in 1958 (ie those born in 1954–1958, 1949–1953, 1944–1948, and 1939–1943) and the subgroups of those aged 0–9 and 0–14 years of age in 1958 for the areas Cumbria, Lancashire, Cumbria and Lancashire combined, and the rest of England. Incidence rate ratios (IRRs), together with corresponding 95% confidence intervals (CIs) calculated assuming that thyroid cancer incidence rates follow a Poisson distribution, were used to compare Cumbria, Lancashire, and Cumbria plus Lancashire, with the rest of England providing reference rates.

4.18 For comparison purposes, equivalent crude incidence rates and IRRs were also calculated for those aged 0–9 and 0–14 years in 1943 (born in 1934–1938 and 1929–1933) and also for those born in 1959–1963, 1964–1968 and 1969–1973 (ie after the Windscale accident). The assumption of Poisson distributed rates was examined by testing for overdispersion. Statistical analyses were performed using Stata and statistical significance was taken to be a two-sided $p < 0.05$.

Results

4.19 Of those born in England between 1929 and 1973, 24,118 people [6,679 (27.7%) males and 17,469 females] were diagnosed with thyroid cancer during 1974–2012. The numbers resident in Cumbria and Lancashire at diagnosis were 304 [83 (27.3%) males and 221 females] and 499 [134 (26.9%) males and 365 females], respectively.

Table 4.1 Thyroid cancer incidence registration and population data, and derived thyroid cancer incidence rates and incidence rate ratios, for people aged 0–19 years in 1958 (born during 1939–1958) and age subgroups 0–9 and 0–14 years in 1958

	Age in 1958 (years)		
Area of England	**0–9**	**0–14**	**0–19**
Number of cases			
Cumbria	69	113	147
Lancashire	130	188	231
Cumbria and Lancashire	199	301	378
Rest of England	5,316	8,259	10,509
Population at risk (thousand person-years)			
Cumbria	2,620	3,922	5,073
Lancashire	5,841	8,728	11,245
Cumbria and Lancashire	8,461	12,650	16,318
Rest of England	247,409	365,152	467,084
Incidence rate (cases per million person-years)			
Cumbria	26.34	28.81	28.98
Lancashire	22.26	21.54	20.54
Cumbria and Lancashire	23.52	21.54	23.16
Rest of England	21.49	22.62	22.50
Incidence rate ratio with respect to the rest of England (95% CI)			
Cumbria	1.23 (0.97,1.55)	1.27 (1.06,1.53)	1.29 (1.09,1.52)
Lancashire	1.04 (0.87,1.23)	0.95 (0.82,1.10)	0.91 (0.80,1.04)
Cumbria and Lancashire	1.09 (0.95,1.26)	1.05 (0.94,1.18)	1.03 (0.93,1.14)

4.20 Table 4.1 presents data for thyroid cancer cases and populations, and resulting incidence rates and IRRs, for the study areas for those aged 0–19 years in 1958, ie for those born during 1939–1958 and diagnosed during 1974–2012. For Cumbria there were raised IRRs (relative to the rest of England) for those aged 0–9, 0–14 and 0–19 years in 1958, which were statistically significant for those aged 0–14 and 0–19 years. For 0–19 year olds, the IRR was 1.29 (95% CI 1.09, 1.52), the significantly raised IRR being due primarily to the IRR in females [male IRR, 1.07 (95% CI 0.76, 1.49); female IRR, 1.38 (95% CI 1.15,

1.67)], although the difference in sex-specific IRRs was not significant. In contrast, for Lancashire, incidence rates did not differ significantly from those for the rest of England, although for the 0–19 year age group the IRR was lowered to a marginally statistically non-significant extent: 0.91 (95% CI 0.80, 1.04). For the combined counties of Cumbria plus Lancashire, the IRRs did not differ significantly from 1.0, the IRR for the 0–19 year age group being 1.03 (95% CI 0.93, 1.14).

4.21 Table 4.2 presents data for cases and populations, and incidence rates and IRRs, for the study areas for the three successive 15-year periods of birth, 1929–1943, 1944–1958 and 1959–1973. These three birth periods represent people who were beyond childhood, children or *in utero*, and not conceived, respectively, at the time of the Windscale accident in 1957. For Cumbria, the IRRs were significantly elevated for those born in 1929–1943 and 1944–1958, while for Lancashire, the IRR was significantly low in 1929–1943 and non-significantly low in 1944–1958. The IRRs for Cumbria and Lancashire for those born in 1959–1973 did not differ significantly from 1.0.

Table 4.2 Thyroid cancer incidence registration and population data, and derived thyroid cancer incidence rates and incidence rate ratios, for areas of England, for those born during successive 15-year periods and diagnosed during 1974–2012. People born during 1944–1958 were children or *in utero* at the time of the Windscale fire, those born during 1929–1943 were beyond their childhood years at the time of the accident, and those born during 1959–1973 were not exposed to iodine-131 released during the accident

	Period of birth		
Area of England	**1929–1943**	**1944–1958**	**1959–1973**
Number of registered incident cases of thyroid cancer			
Cumbria	102	113	89
Lancashire	122	188	189
Cumbria and Lancashire	224	301	278
Rest of England	6,807	8,259	8,279
Population at risk (thousand person-years)			
Cumbria	3,162	3,922	4,079
Lancashire	6,818	8,728	9,444
Cumbria and Lancashire	9,980	12,650	13,523
Rest of England	276,766	365,152	413,831
Thyroid cancer incidence rate (per million person-years)			
Cumbria	32.26	28.81	21.82
Lancashire	17.89	21.54	20.01
Cumbria and Lancashire	22.45	23.79	20.56
Rest of England	24.59	22.62	20.01
Incidence rate ratio with respect to the rest of England (95% CI)			
Cumbria	1.31 (1.08, 1.59)	1.27 (1.06, 1.53)	1.09 (0.88, 1.34)
Lancashire	0.73 (0.61, 0.87)	0.95 (0.82, 1.10)	1.00 (0.87, 1.16)
Cumbria and Lancashire	0.91 (0.80, 1.04)	1.05 (0.94, 1.18)	1.03 (0.91, 1.16)

4.22 Table 4.3 and Figure 4.1 present a summary of IRRs by successive 5-year periods of birth. For Cumbria, there were raised IRRs for all seven consecutive birth periods from 1929–1933 to 1959–1963 (the elevated IRRs for 1929–1933 and 1959–1963 being statistically significant), but not for those born during 1964–1968 and 1969–1973. The largest and most statistically significant IRR was for those born in 1959–1963, with the sex-specific IRRs being raised to a similar extent. For Lancashire, there were decreased IRRs for those born in the four consecutive periods 1929–1933 to 1944–1948 (the low IRR for 1929–1933 being statistically significant), but the IRRs for the following periods were unremarkable. For Cumbria and Lancashire combined, the IRRs did not differ significantly from 1.0 for any period of birth.

Table 4.3 Thyroid cancer incidence rate ratios, for cases diagnosed during 1974–2012 while resident in Cumbria, Lancashire and Cumbria combined with Lancashire, for successive 5-year calendar periods of birth during 1929–1973. Reference thyroid cancer incidence rates are those for the rest of England

	Incidence rate ratios (95% CI)		
Period of birth	**Cumbria**	**Lancashire**	**Cumbria and Lancashire**
1929–1933	1.44 (1.05,1.99)	0.61 (0.43,0.85)	0.87 (0.69,1.10)
1934–1938	1.15 (0.80,1.65)	0.80 (0.60,1.08)	0.91 (0.73,1.15)
1939–1943	1.34 (0.95,1.88)	0.77 (0.57,1.05)	0.95 (0.76,1.19)
1944–1948	1.35 (1.00,1.82)	0.80 (0.62,1.04)	0.97 (0.80,1.19)
1949–1953	1.32 (0.96,1.81)	1.02 (0.80,1.30)	1.11 (0.92,1.35)
1954–1958	1.12 (0.78,1.61)	1.05 (0.82,1.35)	1.07 (0.87,1.32)
1959–1963	1.49 (1.10,2.02)	0.91 (0.70,1.17)	1.09 (0.89,1.32)
1964–1968	0.93 (0.64,1.36)	0.97 (0.76,1.24)	0.96 (0.78,1.18)
1969–1973	0.80 (0.51,1.25)	1.15 (0.90,1.47)	1.04 (0.84,1.30)

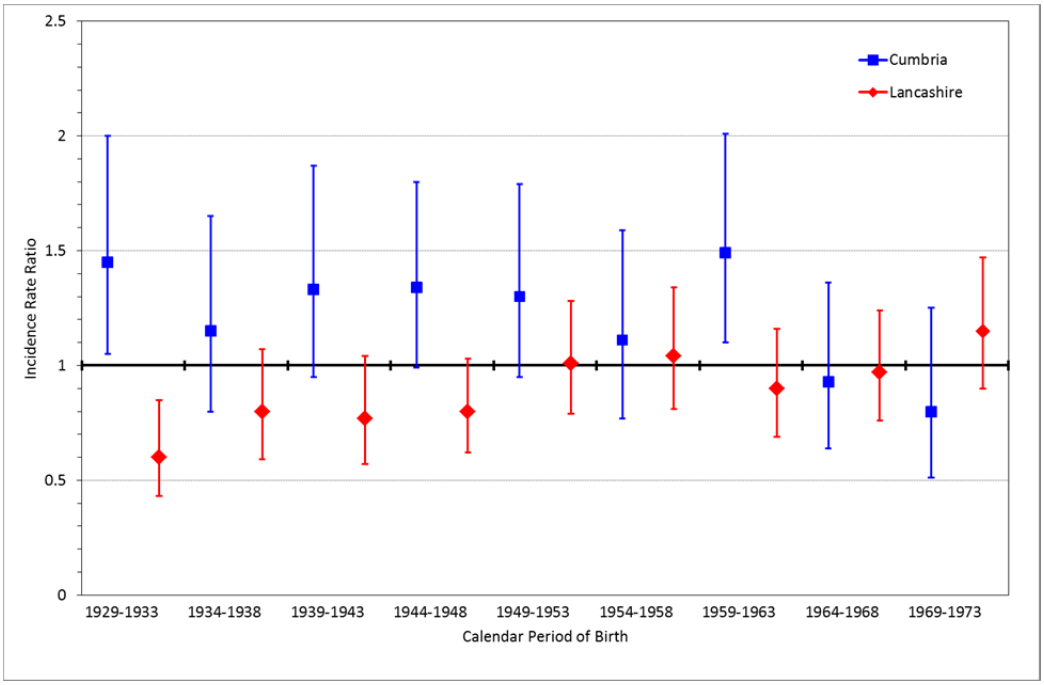

Figure 4.1 Incidence rate ratios (IRRs) for successive 5-year periods of birth for thyroid cancer cases diagnosed in Cumbria and Lancashire during 1974–2012. Reference rates are those for the rest of England. Error bars are 95% Poisson confidence intervals

4.23 Figure 4.1 demonstrates a notable decline in the IRR for Cumbria between the 1959–1963 and 1964–1968 birth periods. Conversely, for Lancashire there is a notable increase in the IRR between the 1944–1948 and 1949–1953 birth periods. One possibility to account for the pattern of thyroid cancer IRRs found is incompleteness of thyroid cancer registration that has varied geographically and over time. In their study of the geographical distribution and temporal trends of thyroid cancer incidence in England and Wales during 1968–1985, dos Santos Silva and Swerdlow (1993) note that the completeness of cancer registration is known to vary across England and Wales (Swerdlow et al, 1993), and the degree of completeness will have varied geographically over time (Swerdlow, 1986). To address this problem, dos Santos Silva and Swerdlow (1993) obtained for each county the ratio of the age- and sex-adjusted thyroid cancer registration rate to that for a weighted sample of other cancers, and then compared this county ratio with the equivalent ratio for the rest of England and Wales. This approach was used to attempt a correction for any differential cancer registration completeness at the county level, but the method does rely implicitly upon the untested assumption that the efficiency of registration of largely non-fatal thyroid cancer is the same as that for cancers with a greater lethality.

4.24 Enquiries were made of the National Cancer Registration Service as to whether there were any registration problems in the areas of interest in the 1960s. Both the North West and Northern and Yorkshire teams replied that they were unaware of any such problems (S Vernon, personal communication). To check this possibility further, data on thyroid cancer and total cancer registrations for Cumbria, Lancashire and the rest of England were obtained for the years 1975–1989, these being the earliest complete datasets available.

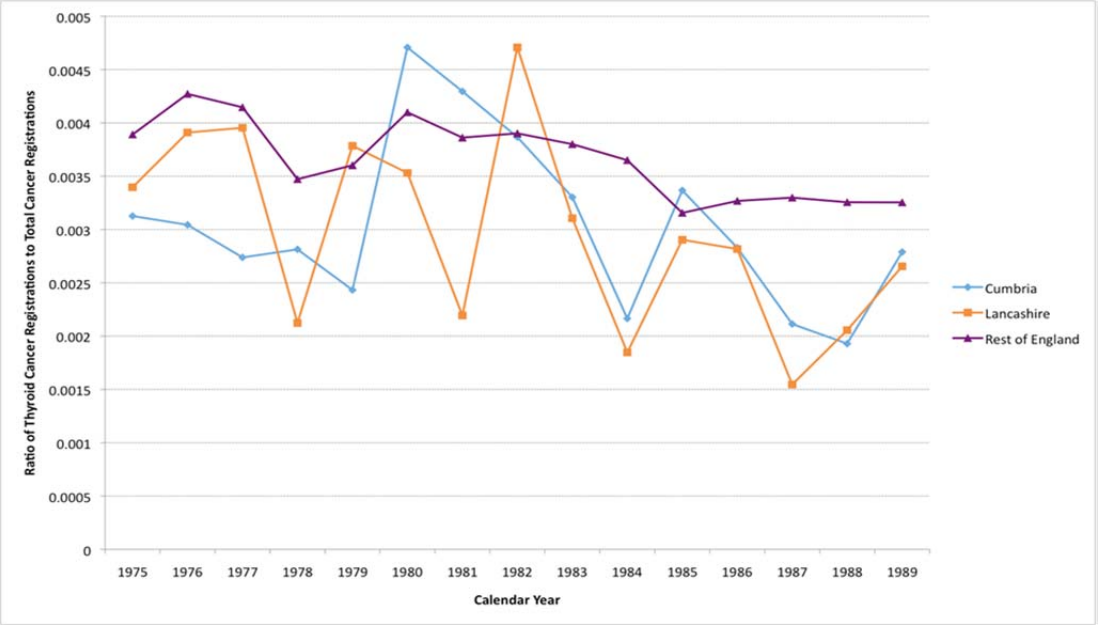

Figure 4.2 Ratios of annual thyroid to total cancer registrations

4.25 Figure 4.2 shows the ratios of thyroid to total cancer registrations. No marked trend or discontinuities are seen in the ratios for the two counties (bearing in mind the limited numbers of annual cases at the county level, leading to statistical instabilities), nor are any material differences from the national ratio, suggesting that registration rates are reasonably consistent.

4.26 Testing for overdispersion demonstrated highly significant extra-Poisson variation in the temporal distributions of the IRRs for Cumbria and Lancashire. As a consequence, additional analyses were undertaken in which adjustments for extra-Poisson variation were made using the method proposed by Breslow (1984). None of the adjusted CIs excluded unity.

Discussion

4.27 The thyroid cancer IRR for those aged 0–19 years in 1958 was significantly increased for those diagnosed during 1974–2012 while resident in Cumbria, an IRR of 1.29 (95% CI 1.09, 1.52). However, the equivalent IRR for Lancashire was decreased to a marginally non-significant extent, an IRR of 0.91 (95% CI 0.80, 1.04). Hence, although the raised thyroid cancer incidence rate among those born during 1939–1958 and diagnosed while living in Cumbria might be thought to suggest an effect of radiation exposure from the release of radioiodine during the 1957 Windscale fire, the decreased rate in Lancashire does not provide support for this inference.

4.28 This caution in interpretation is reinforced by the patterns of thyroid cancer IRRs found in Cumbria and Lancashire among those born in successive 5-year periods during 1929–1973 (Figure 4.1). For those born during 1954–1958, who were youngest at the time of the fire and therefore at greatest potential risk of radiation-induced thyroid cancer, for neither Cumbria nor Lancashire did the IRRs differ significantly from 1.0. Moreover, Figure 4.1 shows a general tendency for increased IRRs in Cumbria that extends from those born in 1929–1933 to those born in 1959–1963, a tendency that is not repeated for Lancashire where those born in the earliest four 5-year periods exhibit decreased IRRs (significantly so for those born during 1929–1933). Indeed, the Cumbrian IRR for those born in 1959–1963, 1.49 (95% CI 1.10, 2.02), is the highest and most statistically significant of all the IRRs examined, and relates to individuals who were not exposed to iodine-131 released during the 1957 Windscale fire.

4.29 Cotterill et al (2001) studied the incidence of thyroid cancer among young people (less than 25 years of age) living in the north of England during 1968–1997, so encompassing any cases in Cumbria that may have occurred during the period 1968–1973, which preceded the study period for this report and would have included those who were children at the time of the 1957 accident. They reported one case in Cumbria during 1968–1986, resident in east Cumbria, implying that the omission of cases in the current study incident in Cumbria before 1974 does not have a serious impact on its findings. Cotterill et al (2001) suggested that an increased incidence rate in Cumbria during 1987–1997 might be attributable to contamination from the Chernobyl accident in April 1986 and Magnanti et al (2009) noted that this increased incidence rate for Cumbria persisted into 1998–2005; the raised incidence during 1986–2005 was entirely due to cases in females. However, exposure to iodine-131 from the Chernobyl accident will have been effectively confined to May to July 1986, so any children conceived after this period will not have been exposed, but any such cases have not been excluded from the analyses nor has account taken of the minimum latent period of about 5 years, so the findings are not readily interpretable in terms of the potential role of iodine-131 contamination from Chernobyl. In any event, thyroid doses in Cumbria from iodine-131 originating from Chernobyl were very low (see Chapter 5, paragraph 5.28).

4.30 There is a clear and large excess of thyroid cancer cases among those heavily exposed at a young age to radioiodine in the former USSR following the Chernobyl accident. The risk of thyroid cancer experienced by these highly exposed individuals broadly conforms to what would be predicted from studies of those exposed as children to external gamma radiation (Little et al, 2014). The thyroid doses received by the most exposed children in the worst contaminated areas of the former USSR were high, at >1 Gy, but evidence of an excess risk of thyroid cancer in areas less affected by Chernobyl contamination is much less clear. In Finland, which was one of the countries most affected by Chernobyl contamination outside the former USSR, no increased risk of thyroid cancer among those exposed while less than 21 years of age in 1986 was detected (But et al, 2006), although the thyroid doses of a few milligray were far less than those received around Chernobyl.

4.31 Substantial quantities of radioiodine were released during the Fukushima accident in Japan in 2011, although the largest consequent thyroid doses are assessed to have been considerably less than those received after the Chernobyl accident (UNSCEAR, 2014). A thyroid screening programme of all residents aged 18 years and younger was instituted in Fukishima Prefecture in 2011 by the Prefectural Government and the data up to the end of 2014 have been analysed by Tsuda et al (2015). Some 300,000 subjects, just over 80% of the eligible population, were examined and approximately 2250 (0.75%) were identified as screen-positive. Of these, 2000 underwent a second round of investigations and 110 were defined as thyroid cancer following fine needle biopsy. This is approximately 30 times the Japanese mean annual incidence rate (largely based on an unscreened population).

4.32 It is not known from the study what thyroid doses might have been received by the cancer cases detected by the screening programme. Of the nine districts into which the study area was divided, the prevalence rate was highest in the ‘central middle district’ (not the area most affected by the accident), while the neighbouring ‘north middle district’ has a rate essentially equal to the reference ‘southeastern least contaminated district’.

4.33 The Ministry of the Environment of the Government of Japan instituted a smaller programme of thyroid screening of children aged 3–18 years in three

areas distant from Fukushima (Aomori, 420 km distant; Yamanashi, 380 km distant; Nagasaki, 1480 km distant). Although only some 4300 children were screened, both the proportions identified as having nodules/cysts (56%) and as requiring further investigation (1%) were remarkably similar to those found in Fukushima (MoE, 2013).

4.34 The study of Tsuda et al (2015) should be interpreted also in the light of the experience in South Korea following the introduction there of a thyroid screening programme (Ahn et al, 2014). Between 1993 (prior to the programme) and 2011, there was an apparent 15-fold rise in the rate of incidence of thyroid cancer, almost exclusively papillary, and thyroid cancer is now the most commonly diagnosed cancer in South Korea; the mortality rate, however, remained the same. There is no suggestion of any link to ionising radiation. Jacob et al (2014) concluded that screening could have a 'drastic' effect on thyroid cancer incidence in Fukushima.

4.35 Given that the expected minimum latency for thyroid cancer is 4–5 years, it would be expected that further cases will be diagnosed. To understand the data properly, the Fukushima Prefecture study will have to be continued and a proper and substantial control study of an unaffected area implemented.

4.36 From 1944 to 1957, the Hanford nuclear site in the northwestern USA reprocessed irradiated nuclear fuel that had been stored for a comparatively short time, so that substantial quantities of iodine-131 (27 PBq) were released to atmosphere during this period. Davis et al (2004) conducted a historical cohort study of thyroid disease among almost 3500 people who had potentially been exposed as children to iodine-131 discharged from Hanford. Considerable efforts were made to reconstruct thyroid doses, and medical examinations of study subjects were carried out by thyroid specialists during 1992–1997. The maximum estimated individual thyroid dose was 2823 mGy, with a median of 97 mGy and a mean of 174 mGy, doses that are around an order of magnitude greater than those received locally from the Windscale fire, but much less than those received after Chernobyl in heavily contaminated areas of the former USSR. Davis et al (2004) found no evidence of a relationship between the dose received from Hanford releases and the cumulative incidence of thyroid cancer (19 cases) or of any other thyroid disease. The authors noted that the study had sufficient statistical power to detect the magnitude of effects that had been reported elsewhere following exposure to iodine-131.

4.37 The Mayak nuclear complex in the Southern Urals region of the former USSR commenced reactor operations in 1948 and started reprocessing irradiated nuclear fuel in 1949. During 1948–1972 about 38 PBq of iodine-131 was discharged to atmosphere from Mayak (Eslinger et al, 2014). A child born in 1947 in the nearby closed city of Ozyorsk and living there until 1972 is estimated to have received a cumulative thyroid dose during this period of 2280 mGy. For young children of 5 years of age living in Ozyorsk, the maximum annual thyroid dose was approaching 1000 mGy in 1949, with annual doses decreasing to around 10 mGy by the late 1950s. Koshurnikova et al (2012) studied thyroid cancer incidence during 1948–2009 in Ozyorsk and the neighbouring city of Kyshtym and compared rates based on registries in these cities with those derived from incidence data for the regional centre of Chelyabinsk during 1993–2006. They reported that thyroid cancer incidence rates in Ozyorsk and Kyshtym were 50% higher than the rate in Chelyabinsk, although details of the Chelyabinsk data were not given.

4.38 There have been reports of raised levels of thyroid cancer incidence around some nuclear installations that have discharged much lower levels of iodine-131. In Belgium, thyroid cancer incidence was reported to be raised to a marginally significant extent around the Mol-Dessel and Fleurus sites where a number of nuclear research and industrial facilities are located (Bollaerts et al, 2014). In 2008, 48 GBq of iodine-131 was accidentally released from the radioiodine production plant at Fleurus, but a conservative assessment of the maximum thyroid dose received by an infant as a consequence of this discharge was just 0.6 mGy (Vandecasteele et al, 2011), and a causal link is unlikely (Mabuchi and Schneider, 2014). Similarly, a report of an elevated incidence rate of thyroid cancer in the vicinity of the Three Mile Island nuclear power station in Pennsylvania (Levin et al, 2013), where 550 GBq of iodine-131 was released to atmosphere during a reactor accident in 1979 – leading to an estimated maximum individual thyroid dose of <0.2 mGy (Clarke, 1989) – is difficult to interpret given the background of high thyroid cancer incidence throughout Pennsylvania (Bann et al, 2014). Kim et al (2015) have reviewed, and conducted a meta-analysis of, studies of the risk of thyroid cancer and residence near nuclear power stations. Overall, they concluded that the evidence does not support an association, but noted various shortcomings in the studies available to their investigation.

4.39 Above-ground nuclear weapons test explosions led to locally raised deposition of radioiodine near the test sites, which frequently involved exposure to short-lived iodine-133 (half-life of 21 hours) in addition to iodine-131. Studies of thyroid cancer incidence have been undertaken near the Nevada test site in the USA, the Semipalatinsk nuclear test site in Kazakhstan (formerly part of the USSR) and in the Marshall Islands in the Pacific Ocean. Lyon et al (2006) studied thyroid disease in a cohort of nearly 2500 people who were children in 1965 and living near the Nevada test site, who were medically examined in 1985–1986. The maximum assessed individual thyroid dose was 1.4 Gy and the mean 0.12 Gy (Simon et al, 2006). Eight cases of thyroid cancer were found, but the dose response was unremarkable, although the positive dose response for benign thyroid disease was highly significant. Land et al (2015) investigated thyroid disease among almost 2400 people who were resident downwind of the Semipalatinsk nuclear test site during 1949–1962 while less than 21 years of age. The prevalence of thyroid nodules was assessed by ultrasound screening in 1998, and 35 cases of thyroid cancer were detected. Estimated thyroid doses ranged up to several gray, with a mean of 100–200 mGy, although uncertainties were substantial. The dose response for thyroid cancer was positive, but not significantly so. Residents of the Marshall Islands were exposed to radioiodine as a result of testing of nuclear weapons by the USA, and particularly so by inadvertent exposure from the Castle Bravo thermonuclear test explosion in 1954 that led to assessed thyroid doses of around 20 Gy for young children living on Rongelap Island (Simon et al, 2010). Examination of nearly 6000 Marshallese during 1993–1997 found a high prevalence of thyroid cancer, especially among those alive at the time of the Castle Bravo test (Takahashi et al, 2003).

4.40 Three studies including more than 6000 children administered known amounts of iodine-131 for diagnostic purposes (giving a mean thyroid dose of 1 Gy) did not find an excess of thyroid cancer (Boice, 2005). However, the numbers of children exposed while less than 10 years of age, who would be at most risk, were small.

4.41 Table 3.6, taken from Bunch et al (2014), shows the incidence of thyroid cancer among those born during 1950–2006 in the village of Seascale, the rest of Copeland and Allerdale county districts, and the remainder of

Cumbria, and diagnosed while resident in the UK during 1971–2006. Overall, 89 cases of thyroid cancer were observed: 0 in Seascale births (0.44 case expected), 30 in the rest of Copeland and Allerdale (34.7 expected), and 59 in the remainder of Cumbria (56.9 expected). It is not possible from the results as reported to infer much of substance about the incidence of thyroid cancer in relation to the 1957 Windscale accident, except that thyroid cancer incidence rates among those alive at the time of the accident have not made a notable impact upon the overall level of incidence of thyroid cancer among births in Cumbria from 1950. However, the Cumbrian birth cohort potentially permits a study of thyroid cancer risk among those born during 1950–1958 in relation to the thyroid doses assessed to have been received as a result of the radioiodine release during the Windscale fire.

Conclusions

4.42 There is no conclusive evidence linking the Windscale nuclear reactor fire with an excess risk of thyroid cancer in those exposed to iodine-131at a young age. Those born in 1954–1958 and diagnosed in Cumbria during 1974–2012 have a significantly high excess rate of thyroid cancer incidence, but not as high as those born in 1959–1963 (ie after the 1957 Windscale fire).

4.43 This was a geographical study and so could not account for inward and outward migration in Cumbria and Lancashire. It also dealt with the whole of Cumbria and Lancashire, whereas the iodine-131 contamination was not geographically uniform. A study using the Cumbrian birth cohort would shed more light on these findings.

4.44 Since exposure in childhood is predicted to lead to an increased risk of thyroid cancer throughout lifetime, excess cases among those exposed at a young age during 1957 may yet occur.

4.45 Further research should include a review of possible geographical variation in ascertainment rates and investigation of other areas where excesses of thyroid cancer have been reported.

CHAPTER 5

RADIATION EXPOSURE AND THE RISK OF RADIATION-INDUCED LEUKAEMIA AND OTHER CANCERS IN YOUNG PEOPLE AND ADULTS EXPOSED AS CHILDREN, IN THE VICINITIES OF SEASCALE AND DOUNREAY

5.1 The aim of this part of the study was to consider the radiation exposures of young people living in Seascale and around Dounreay as a function of time from the start of operations until 2010. Any changes to estimates of exposures from those reported in previous COMARE reports for the two locations (the second report for Dounreay and the fourth report for Seascale: COMARE, 1988, 1996) were reviewed. Finally, the implications of the exposures for the inferred risks of radiation-induced leukaemia and other cancers were considered. Major changes to relevant scientific data were taken into account, such as changes in dosimetric data or radiation risk modelling. However, in contrast to previous studies on this topic a full quantitative exposure and risk assessment was not undertaken. Rather, a semi-quantitative approach was adopted to investigate possible changes since publication of the earlier COMARE reports, because these changes were judged not to have a sufficiently large impact on the previous results to warrant a full quantitative assessment. This study also specifically considered radiation doses to the thyroid and the possible risks of radiation-induced thyroid cancers.

5.2 The same basic approach was used as in the previous studies in that all significant sources of radiation exposures were considered and the emphasis was on the exposures of young people, although possible exposures to adults were addressed also. However, the aim was to estimate average doses as a function of time for different age groups rather than following cohorts of children born in different years until the age of 24 years, as was the case for Seascale in the fourth COMARE report. The emphasis was on estimating the exposures in the years following publication of the previous reports and so for consistency the same parameter values were used where possible. However, the implications of using more recent data were considered, particularly any revisions to discharge data or to dosimetric data. The significance of changes to the models for radiation-induced leukaemia was investigated also.

5.3 The previous reports from COMARE found that estimated doses to the red bone marrow (RBM) and the associated risks of radiation-induced leukaemia and other cancers were mainly due to exposure to natural background radiation with, in general, only a small contribution from operations at the Dounreay or Sellafield sites. In the fourth report it was noted that, for Seascale, natural background was the main contributor to RBM dose for all birth years, with medical exposure generally being the main component for other sources for most years of birth. The exceptions were for children born in 1954 when uranium oxide (UO_2) particle releases from Sellafield peaked and those born in the late 1950s and early 1960s when fallout from atmospheric testing of nuclear weapons was relatively important. For Dounreay, the contribution from natural background dominated the exposures and risks at all times.

Radiation exposure of young people in Seascale

5.4 The sources of radiation exposure considered were:

(i) natural background radiation

(ii) medical exposures

(iii) fallout from the atmospheric testing of nuclear weapons

(iv) discharges from the Sellafield site (planned and accidental releases)

(v) releases from the 1986 accident at the Chernobyl nuclear power plant

(vi) discharges from the Marchon Works phosphate plant in Whitehaven (operated by Marchon Products, Albright and Wilson and then Rhodia)

5.5 For the first three of these routes of exposure, estimates were based on average exposures in the UK, with some limited account taken of local information for Seascale. The exposures from Sellafield discharges were specifically for Seascale throughout the period considered. For Chernobyl and Marchon the exposures were specific to Seascale where possible, but in later years when the levels of exposure from these routes were reduced to very low levels, it was not generally possible to specifically estimate exposures to the Seascale population.

5.6 For the fourth COMARE report, the exposures were based on measured or estimated levels of radiation in the Seascale environment for each year from 1951–1992 (COMARE, 1996). COMARE had asked the then NRPB to assess the exposures and associated risks and full details of the assessments were presented in the report NRPB-R276 (Simmonds et al, 1995). For the current study, the data given in NRPB-R276 were expanded to cover the period up to 2010 using similar approaches to those used originally. In doing this, it was particularly important to review the discharges from the Sellafield site, as well as the available environmental monitoring data. The levels of radiation in the environment were then used to estimate radiation doses to average 1 and 10 year old children living in Seascale as a function of time, using standard PHE models as discussed below.

5.7 At the time of the original work on Seascale for the Black report (1984), there was concern over possible exposures due to radioactive objects and particles that were being found on the beaches near Seascale. This was considered in the 1984 NRPB assessment, but was found not to be a significant contributor to assessed exposures or risks (Stather et al, 1984). The beaches have continued to be monitored since 1984 and, more recently, enhanced monitoring has detected objects and particles, the vast majority of which contain low levels of radioactivity*. The likelihood of any individual coming into contact with a radioactive object is very low and, if exposure did occur, none of the finds has contained levels of activity that would give rise to exposures significantly higher than those found in the 1980s (Brown and Etherington, 2011). It was, therefore, not deemed necessary to consider specific exposure from radioactive objects on beaches in this assessment, although exposures due to general levels of radioactivity on beaches (including from particulate materials) were taken into account.

* COMARE receives and evaluates reports on the Sellafield beach monitoring programme at each of its meetings, while the Contaminations Working Group takes a more in-depth approach and has made recommendations on the monitoring system and schedule.

Sellafield site, discharges and environmental monitoring data

5.8 The Sellafield site is located in west Cumbria near to the coast and is about 3 km north of the village of Seascale (see Figure 1.2). The nuclear site has a complicated history having originally been used for the production of plutonium and other materials for defence purposes and there have been various changes in uses, responsibilities and operators over the years. Table 5.1 summarises some of the key stages in the development of the site up until 1984, while Table 5.2 summarises the stages from 1984 until 1992, covered in the fourth report. Table 5.3 summarises the major new plant brought online on the site since 1992 or under construction, while the principal functions of the site are given in Table 5.4. Currently, large sections of the Sellafield site are being decommissioned, including the Calder Hall reactors, the original Windscale piles, the original reprocessing plant and various fuel storage and waste treatment facilities.

Table 5.1 Stages in the development of the Sellafield nuclear site up to 1984

	Date operational	Date shut down
Site available July 1947	Work commenced September 1947	
First and second pipeline to sea	Laid June 1950	Isolated from sea 1992 Sealine 1 removed in 2006 Sealine 2 seaward section was replaced in 1991 and is still in operation
Temporary emergency sea line was installed in 1987	1987	Removed 2006
No. 1 pile	Critical October 1950	October 1957
No. 2 pile	Critical June 1951	October 1957
1st reprocessing plant and associated facilities	January 1952	Reprocessing plant converted to head end plant for oxide fuel – used 1969–1973
1st Calder Hall reactor	August 1956	2003
Other three Calder Hall reactors	1958	2003
Prototype advanced gas cooled reactor	1963	April 1981
2nd reprocessing plant (Magnox fuel)	1964	Still in operation – shut down due 2019
First generation oxide storage pond	1968	Still in operation – shut down due 2040
Fast reactor fuel fabrication plant	1970	1992
3rd pipeline to sea	Laid 1976	Still in operation

Table 5.2 Stages in the development of the Sellafield site from 1984 to 1992

	Date operational
Site ion exchange plant (SIXEP)	1985
Salt evaporator	1985
New spent fuel storage and decanning	1986
THORP receipt and storage	1988
Vitrification plant for high activity (HA) liquid waste	1990
Magnox encapsulation plant	1990

Table 5.3 Major new plant brought on line since 1992 or under construction at the Sellafield site

	Date operational (actual/expected)
National Nuclear Laboratories	2004
Fellside CHP	July 1994
FGMSP sludge buffer	March 2014
PFSP Drum Filling Plant	September 2015
SPP1 Sludge packaging plant buffer storage (for FGMSP)	2015
Overbuilding for Floc Storage Tanks	April 2001
Additional tanks to support emptying Floc Storage Tanks	April 2001
(EP2)(WEP) Waste Encapsulation Plant	1994
(EPS2) Encapsulated product store #2	March 1997
(EPS3) Encapsulated product store #3	April 2014
(SPRS) Sellafield Product and Residue Store	2011
THORP Head End and Chemical Separation	1994
(SMP) Sellafield Mox Plant	2000
(SMP) export facility	February 1999
(STP) Solvent Treatment Plant	January 2000
(EARP) Enhanced Actinide Removal Plant	March 1994
(EPMF) Effluent Plants Maintenance Facility	November 1993
Effluent plants service building	September 1991
(WPEP) Waste Packaging and Encapsulation Plant	March 1994
(SDP) Silos Direct Encapsulation Plant	2022
Vitrification Plant (Vit Line 3)	2000
(BEP) Process Building Box Encapsulation Plant	2020
(BEPPS) Box Encapsulation Plant Product Store	2018
(WTC) Waste Treatment Complex Phase 1 b)	January 1996
(EDS2) Engineered Drum Store #2	May 2000
(EDS3) Engineered Drum Store #3	2006
(WAMAC) Waste Monitoring and Compaction Plant	October 1994
(REF) Revised Export Facility	October 2005
(SAV) Separation Area Ventilation Improvements (Plant and Stack)	2016
SNM Store 9 Extension	May 1997
THORP UO3 drum store	July 2002

Table 5.4 Sellafield site – principal functions

Receipt, storage and reprocessing of spent Magnox fuel
Treatment and storage of products of reprocessing
Receipt and storage of spent oxide fuel
Receipt and storage of nuclear materials from other nuclear sites
Treatment, storage or disposal of waste products

5.9 As discussed in the fourth COMARE report, the liquid and atmospheric discharges of radionuclides from the Sellafield site have changed significantly over the years of operation. This reflects the different uses of the site, but also differences in what was deemed acceptable from regulatory and radiological protection viewpoints. At the time of the fourth report, the Sellafield site was operated by BNFL and, at the request of COMARE, BNFL carried out a detailed review of the discharges from the site. Since that time, additional work has been carried out to review the historical discharges. The current owners of the site (the NDA) provided COMARE with information on discharges up to and including 2010 for use in the current study. The discharges were reviewed for consistency between sources, including published information, and a final set of discharges was compiled for use in the project. Full details of the annual liquid discharges from the Sellafield site used in the current study from 1952–1998 are given by Jackson et al (2000). From 1999–2010, discharge data have been provided to COMARE by the Environment Agency; these data are consistent with those published by Sellafield Limited in its annual monitoring reports. Liquid discharges of iodine-129 have not been considered previously, but have been taken into account in the current study from 1952–2010. Gray et al (1995) is the primary source of aerial discharge data from 1951–1992, discharges being listed for two effective stack heights consistent with the approach adopted previously. Between 1993 and 2010, data from Sellafield annual monitoring reports have been used; for 2005–2010 where no information has been given on the height at which the effluent is released, a single effective stack height of 80 m has been assumed. Aerial discharges of iodine-129 have been included since 1994 from buildings with an effective stack height of 10 m in addition to discharges from high stacks. Aerial discharges of cobalt-60 from 1951–2010 have been included; these were not considered in NRPB-R276.

5.10 Liquid discharges started from the Sellafield site in 1952; Figure 5.1 shows the annual discharges to sea of three key alpha emitting radionuclides (plutonium-238, plutonium-239/240 and americium-241) as a function of time from 1952–2010. The annual discharges of four key beta emitting radionuclides (strontium-90, ruthemium-106, caesium-137 and plutonium-241) are given in Figure 5.2 for the same period. In both cases there are distinct peaks in the discharges in the mid-1970s with reductions following the installation of various different actinide or caesium and other effluent treatment plants. Discharges from the late 1980s onwards have been a small fraction of the peak discharges, but have continued at a similar rate throughout the remaining period to 2010.

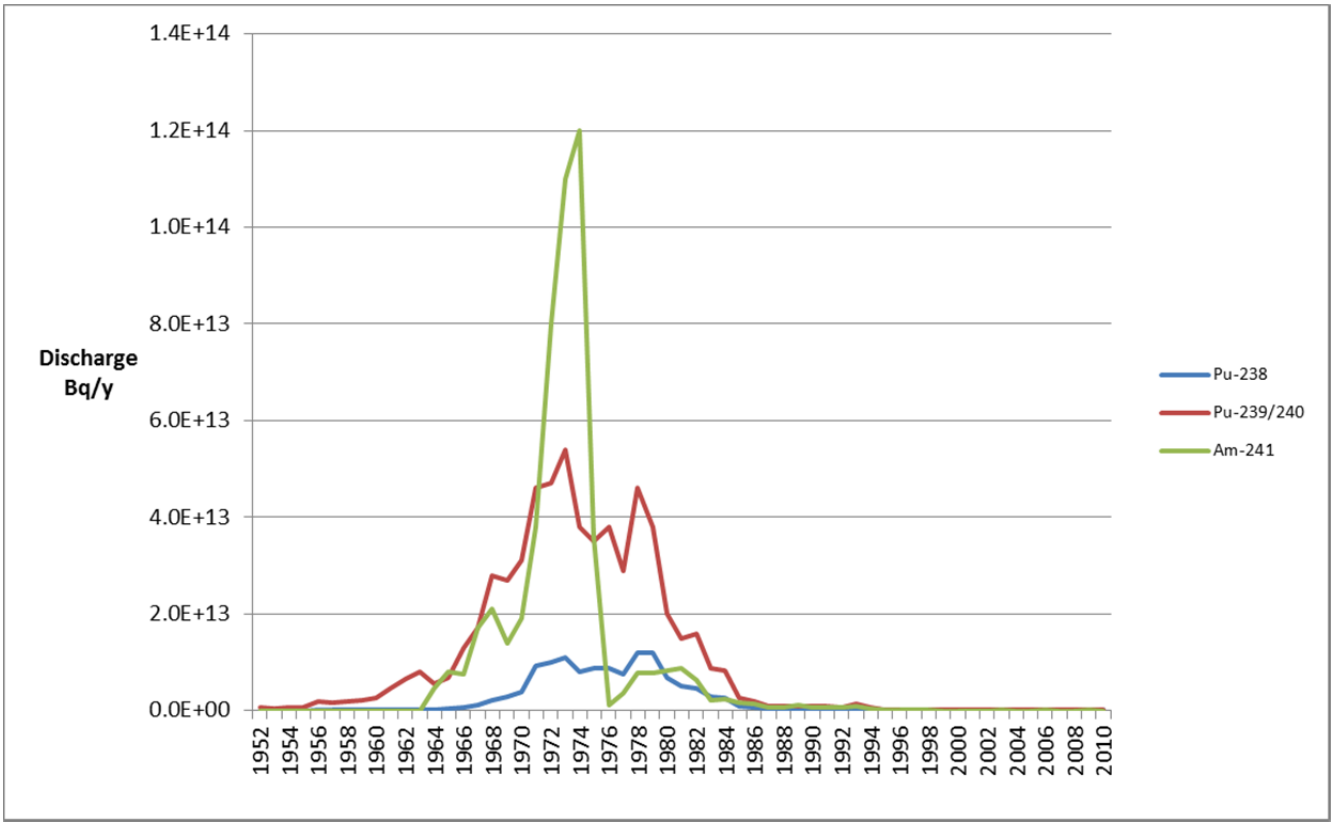

Figure 5.1 Annual liquid discharges from the Sellafield site – key alpha emitters

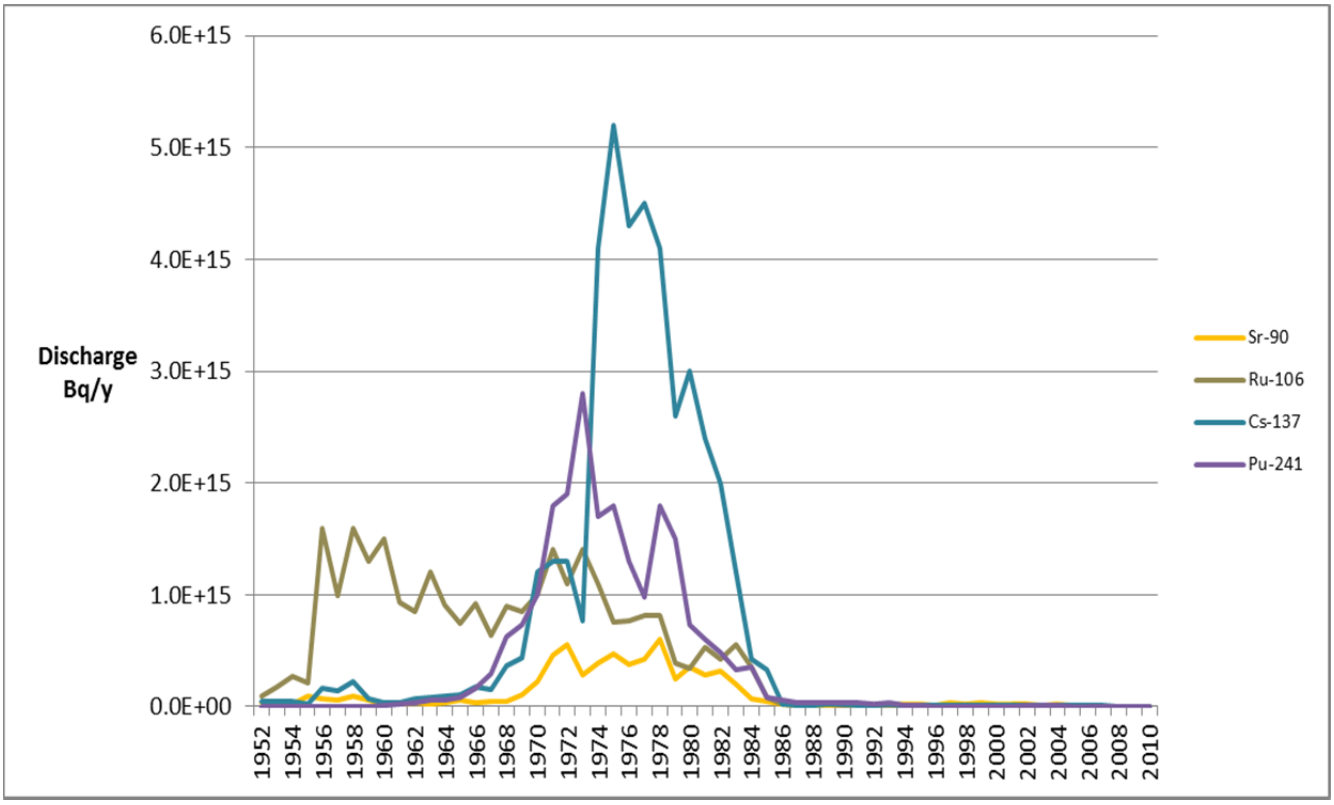

Figure 5.2 Annual liquid discharges from the Sellafield site – key beta emitters

5.11 As discussed in detail in the earlier reports (Simmonds et al, 1995; COMARE, 1996), data on discharges to atmosphere are associated with greater uncertainties than are discharges to sea, due to difficulties in monitoring all sources of discharge, particularly in the early years of operation of the site. Based on the information provided by the NDA, the annual discharges to atmosphere of plutonium-239/240 from high stacks from 1952–2010 are shown in Figure 5.3. There were peaks of discharge in the early years of operation of the site, some smaller peaks of discharge in the early 1970s, but then a significant reduction with ongoing low levels of discharge until 2010. Figure 5.4 shows the annual discharges to atmosphere of the main beta emitters, strontium-90 and caesium-137. These discharges are again very variable, with distinct peaks in the 1950s, early 1960s and early 1970s and, notably for caesium, a significant peak in the early 1980s. As for all other discharges from the Sellafield site, discharges were reduced significantly from the mid to late 1980s and remained at about the same low rate until the end of the study period (to 2010).

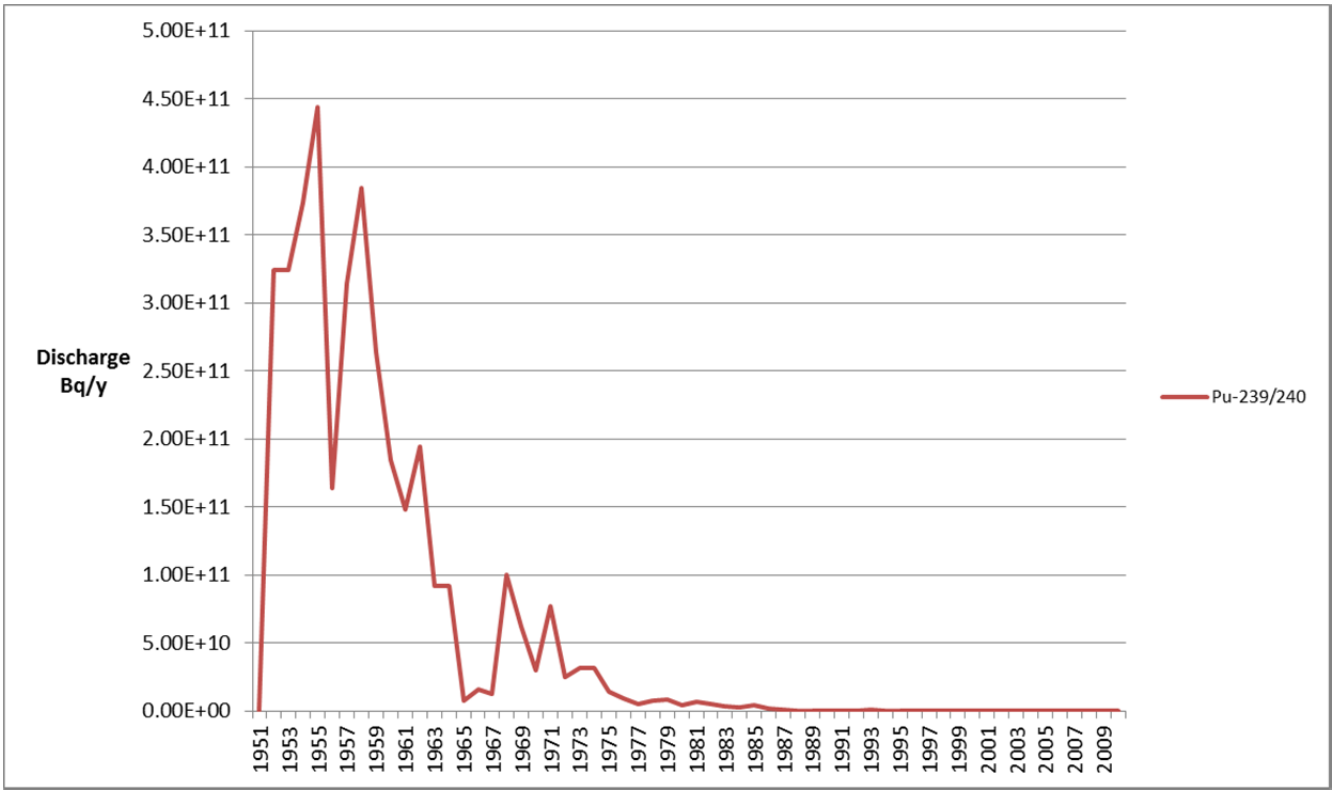

Figure 5.3 Annual aerial discharges from the Sellafield site – plutonium-239/240 (high stack)

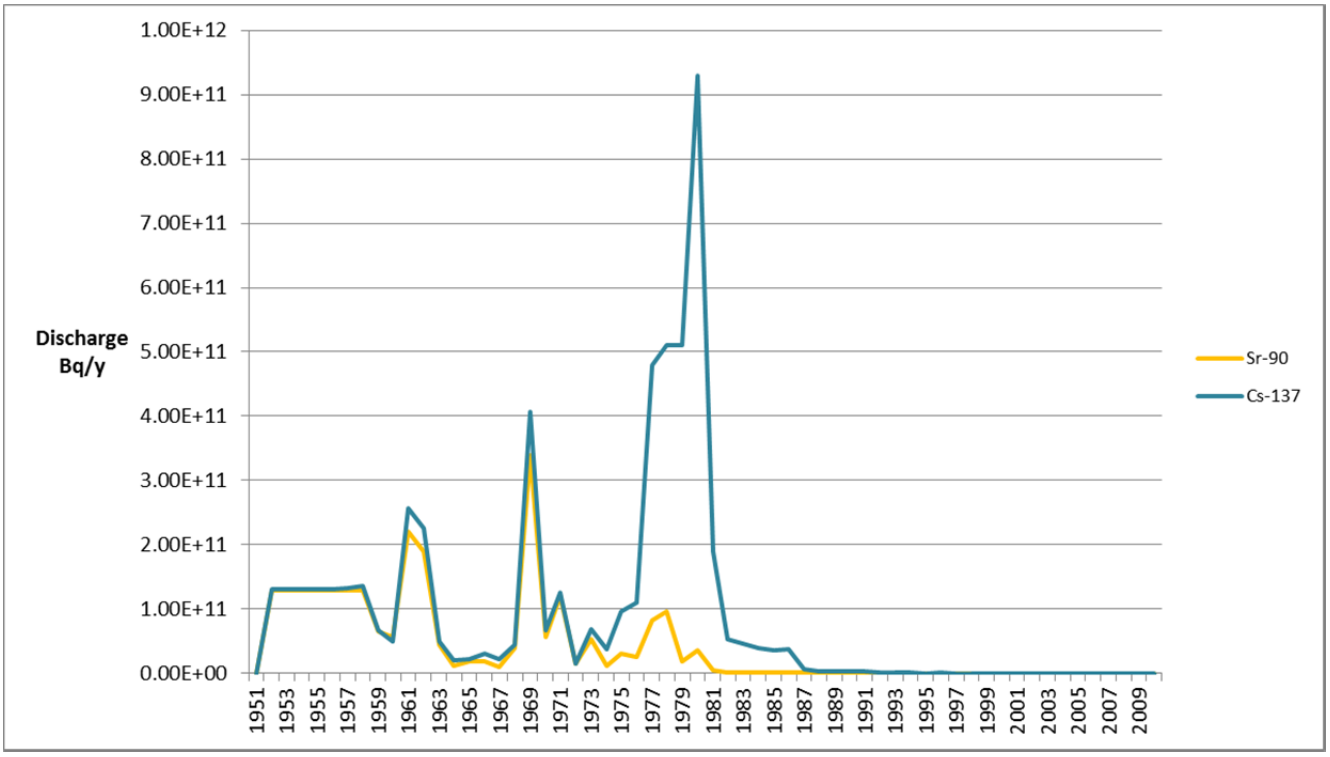

Figure 5.4 Annual aerial discharges from the Sellafield site – key beta emitters

5.12 The radionuclide krypton-85 was not considered in NRPB-R276 (Simmonds et al, 1995) because it made no significant contribution to dose estimates. The fission product is released, almost exclusively, from reprocessing operations involving the chemical dissolution of irradiated nuclear fuel. Discharges of this radionuclide increased significantly from 1994, however, due to the operation of the Thermal Oxide Fuel Reprocessing Plant (THORP). Jackson et al (1998) calculated the annual associated committed effective dose to individuals in a small critical group to peak at around 1.5 µSv.

5.13 The discharges considered in this study also included unplanned, short duration discharges, such as of iodine-131 due to the inadvertent reprocessing of 'short-cooled' fuel and also of the planned release of iodine-131 during the first reprocessing campaign in 1952 (Simmonds et al, 1995). The discharges of iodine-131, mainly due to the routine reprocessing of short-cooled fuel after 1952, have been revised, based on additional unpublished data provided by the NDA, which affects the timing of the discharges between 1953 and 1966, but leads to the overall discharges over the period 1952–1966 being higher. The NDA (2014) advised COMARE that it remains appropriate to assume (as for 1952 in Simmonds et al, 1995) that 10% of the dissolver inventory of iodine-131 was released during 1952–1966 and this forms the main basis for the discharges and doses given here. However, there is evidence from an analysis of available information that the percentage may have been significantly lower than this (Wakeford, 2007a). Therefore, a second analysis was carried out assuming the same pattern of iodine-131 entering the reprocessing plant, but with only 1% of the dissolver inventory discharged. Figures 5.5a and 5.5b show the estimated discharges of iodine-131 to atmosphere from the Sellafield site assuming that 10% and 1% of the dissolver inventory is discharged, respectively.

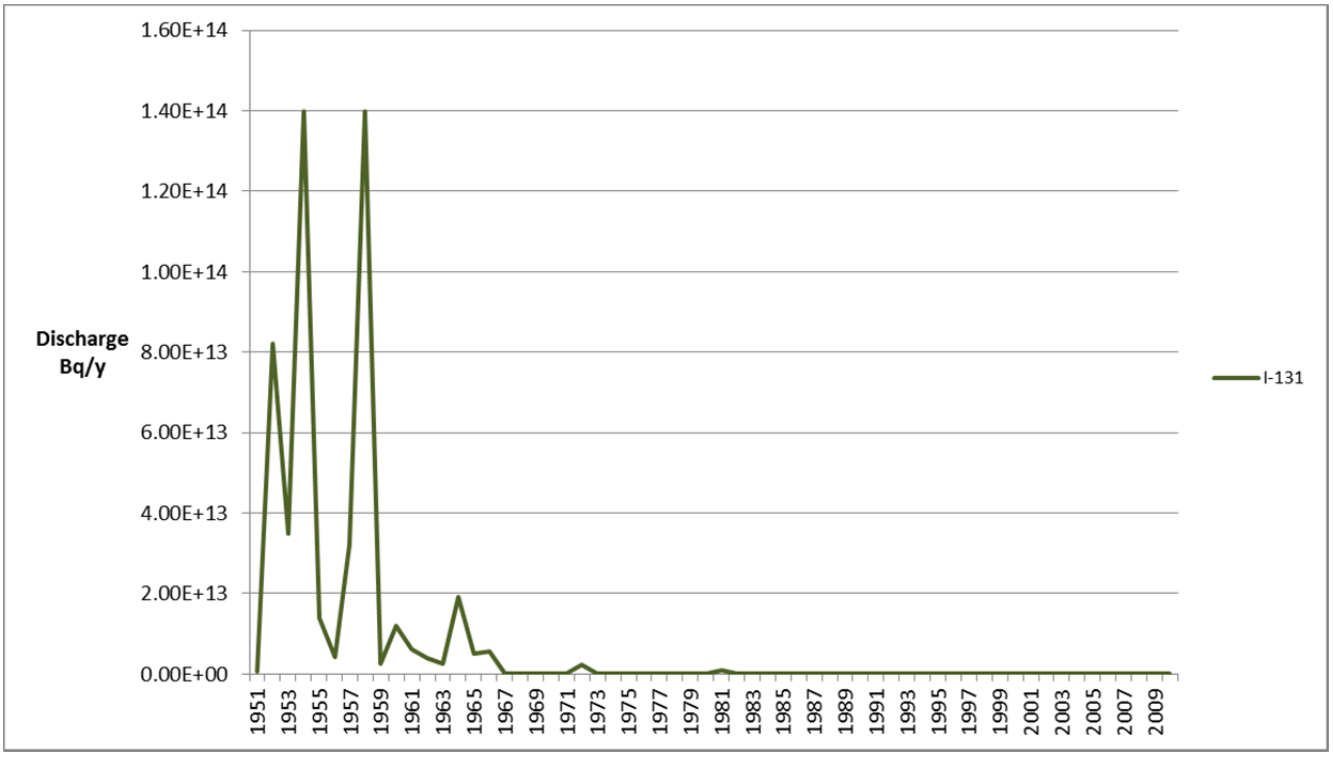

Figure 5.5a Annual routine aerial discharges of iodine-131 from the Sellafield site assuming that 10% of the inventory of the dissolver is discharged during 1952–1966

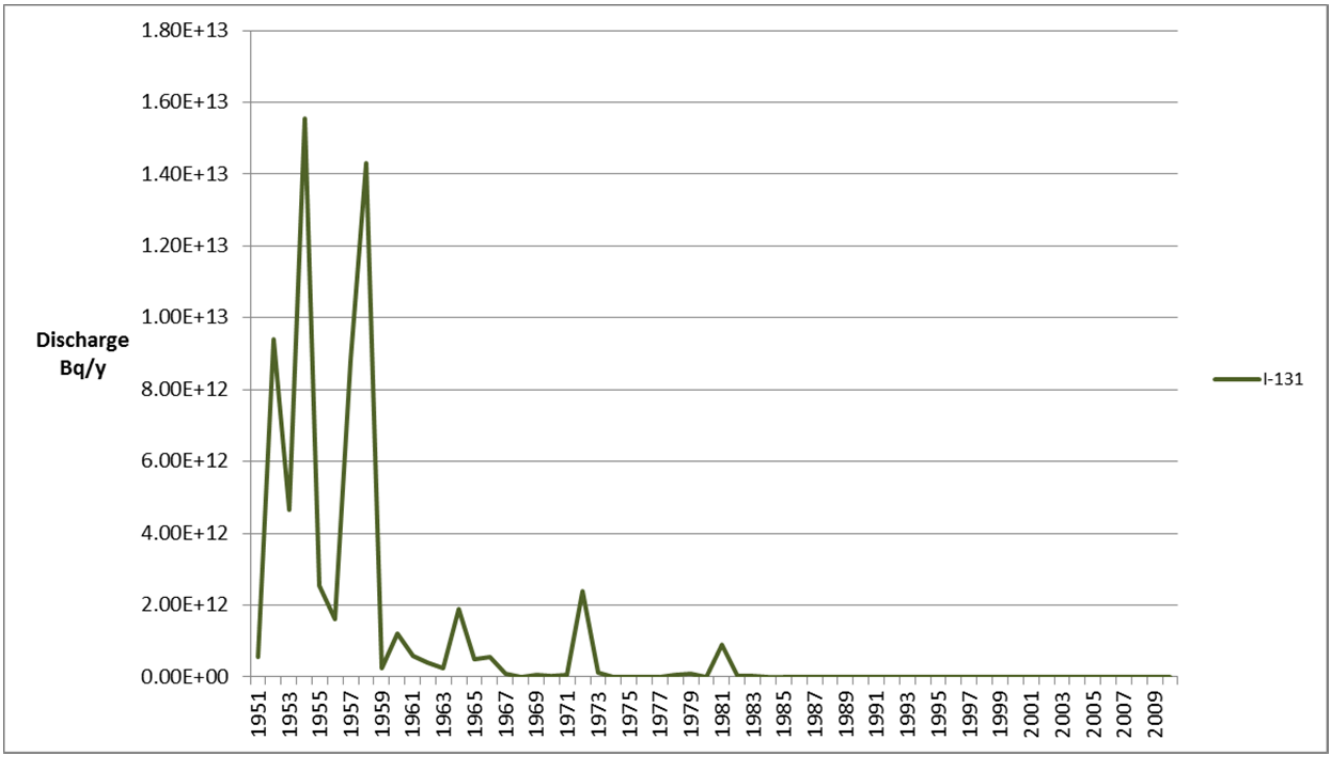

Figure 5.5b Annual routine aerial discharges of iodine-131 from the Sellafield site assuming that 1% of the inventory of the dissolver is discharged during 1952–1966

5.14 The accidental release from the fire in Windscale no. 1 pile in 1957 was also considered as an input, together with the inadvertent releases of irradiated uranium oxide particles from the two Windscale piles from 1950/51 to 1957. Detailed information on these sources of exposure is available elsewhere

(Simmonds et al, 1995; COMARE, 1996; Garland and Wakeford, 2007; Johnson et al, 2007; Smith et al, 2007).

5.15 The releases to atmosphere of irradiated uranium oxide particles from the Windscale pile chimneys before mid-October 1957 have been extensively re-examined by Smith et al (2007). They concluded that the mass of uranium oxide particles released was probably less than the 20 kg assumed by the NRPB (Simmonds et al, 1995) and COMARE (COMARE, 1996) for the purposes of risk assessment, but that the figure of 20 kg represented a 'realistically conservative' estimate of the quantity of particles released.

5.16 The Windscale accident in October 1957 involved a fire in the nuclear reactor core of Windscale no. 1 pile (Arnold, 2007) that released significant amounts of radionuclides to atmosphere, in particular iodine-131, which therefore led to an increase in doses to the thyroid gland. However, immediate measures were taken to dispose of milk from the local area contaminated with iodine-131 and so the ingestion doses (mainly thyroid doses) to children in Seascale were generally low, with the main contribution to the thyroid dose being from inhalation of iodine-131. Measurements of doses to the thyroids of local people made just after the accident indicated maximum thyroid doses of 95 and 160 mGy to an adult and a child, respectively, although most thyroid doses were in the ranges 5–20 and 10–60 mGy, respectively (Crick and Linsley, 1984; Dunster et al, 2007). Crick and Linsley (1984) have suggested that, in the absence of the milk ban, typical thyroid doses could have reached levels of 70 and 360 mGy in adults and children, respectively, and that the highest measured thyroid doses could have been a reflection of the milk ban not being completely effective.

5.17 The aerial discharges of radionuclides during the Windscale fire were comprehensively re-examined at the time of the 50th anniversary of the accident in 2007 (Nelson et al, 2006; Garland and Wakeford, 2007; Johnson et al, 2007; Wakeford, 2007b). Garland and Wakeford (2007) found that, on the basis of detailed modelling by the Met Office of the atmospheric dispersion of radioactive materials (Nelson et al, 2006; Johnson et al, 2007), the estimates of the quantities of the major radionuclides released during the fire should be increased by about a factor of two. However, this increase resulted from an assessment that the plume travelled further east over the North Sea than had been assumed previously and so this does not significantly affect the dose estimates for the areas under consideration, which were based on actual measurements.

5.18 Webb et al (2006) assessed unplanned operational events at Sellafield that had, or could have had, radiological implications for members of the public. They used records to identify events from 1950 onwards that gave rise to off-site releases of radioactive material from Sellafield that rated as level 3 or above using the criteria of the International Nuclear and Radiological Event Scale (INES). They identified 21 such events, with 15 of these rated as INES level 3 and one (the 1957 fire at Windscale no. 1 pile) as INES level 5, with the majority of these occurring during the 1960s and 1970s. The authors suggested that these 21 events represented a comprehensive list of unplanned events at Sellafield that had, or could have had, an off-site impact.

5.19 Atmospheric and marine discharges of carbon-14 from Sellafield have been reassessed by Isogai et al (2002) and Cook et al (2004), respectively. Aquatic discharges for 1967–1999 were determined using archived seaweed samples, and showed that carbon-14 levels in seaweed samples were broadly consistent with measured discharges of carbon-14 when such measurements

were made at Sellafield from the mid-1980s, but that the annual discharges of carbon-14 as indicated by seaweed samples before this period, when discharges were not measured but had to be estimated, were greater than the estimates by a cumulative activity of approximately 36 TBq. The measurements of carbon-14 in seaweed were used to reconstruct annual carbon-14 marine discharges for the period 1967–1984, before marine discharge measurements were made at Sellafield (Cook et al, 2004). Annual atmospheric discharges of carbon-14 during 1950–1999 were determined from tree-ring measurements (Isogai et al, 2002). The tree-ring measurements indicate that after 1977, when carbon-14 aerial discharges were measured at Sellafield, the two sets of estimates produced very similar total releases. However, between 1951 and 1977 the total discharge as estimated by Gray et al (1995) is substantially overestimated when compared with the tree-ring measurements: 215 and 101 TBq, respectively. The tree-ring measurements have been used to revise the annual atmospheric carbon-14 releases from Sellafield for 1951–1977 (Isogai et al, 2002).

5.20 The original studies to determine the risk of leukaemia and other cancers in Seascale made as much use as possible of measurements of radionuclides in environmental materials as an input to the dose and risk assessment. Measurements were used directly to estimate intakes of radionuclides by inhalation and ingestion or external doses in a number of cases. In addition, as part of the work carried out as an input to the fourth COMARE report (COMARE, 1996), measurements were compared with model predictions to determine the validity of the approach adopted where models were used (Simmonds et al, 1995). Extensive measurements of radionuclides continue to be made around the Sellafield site, both by the operators of the site and by the relevant government agencies. The results from the latter measurements are published annually in the Radioactivity in Food and the Environment (RIFE) reports. This source of information was used where possible to estimate intakes and doses due to discharges from Sellafield from 1992 until 2010. Measurements in milk were used as an indicator of activity concentrations in terrestrial foods. The measurements include contributions from routine discharges from Sellafield, along with residual levels from the Chernobyl accident, fallout from nuclear weapons testing and the uranium oxide discharges from the Windscale piles. To avoid assigning artificial variations in activity concentrations from the different sources due to the low activity concentrations being measured, the measured activity concentrations have been assigned to routine Sellafield discharges in the presentation of doses from 1992 to the present day. Contributions of caesium-137 from Chernobyl and strontium-90 from the uranium oxide discharges, in addition to fallout from nuclear weapons, can account for about 50% of the ingestion doses over this period. As liquid discharges and activity concentrations in the marine environment have decreased, the measurements made in marine foods have also decreased and many measurements are now below the limits of detection of the sensitive equipment used. Model predictions have been used to estimate these low activity concentrations in the marine environment.

Radiation exposures due to Sellafield discharges

5.21 Average equivalent doses to the RBM for 1 and 10 year old children living in Seascale were estimated as a function of time to illustrate how radiation exposures would have changed over the period 1951–2010 covered by this study. The doses estimated for each year are the sum of the external dose in the year and the doses from intakes by inhalation and ingestion in the year resulting from the discharges from Sellafield. The doses from intakes were integrated to (and including) age 24 years (eg for 24 years for 1 year olds and for 15 years for 10 year olds) rather than being the full dose commitment to age 70 years that is normally used for radiological protection purposes. This approach was used in the previous study (Simmonds et al, 1995) as it was

consistent with the main risk assessment where cohorts of children born each year were followed to age 24 years or to 1992. An RBE of 20 was used for alpha radiation, as previously. Figure 5.6 shows the estimated doses for 1 year old children from 1951–2010 for routine discharges, while Figure 5.7 shows the same results for 10 year old children. In both cases, the total doses are given together with the doses from external irradiation, ingestion and inhalation. For both 1 and 10 year old children, the highest estimated RBM doses were received in the 1950s and early 1960s. Until 1957, the Windscale piles were operating, leading to high levels of external irradiation from routine discharges of argon-41 and doses from radionuclides discharged inadvertently in the releases of uranium oxide particles and during the 1957 Windscale accident. Up to 1963, the site routinely discharged polonium-210, leading to high ingestion doses (see paragraph 5.31 below). The estimated doses decreased significantly once the Windscale piles were closed down in 1957 and the polonium-210 discharges ceased in 1963. The inhalation dose is mainly due to discharges of plutonium-239/240 and reflects the discharges shown in Figure 5.4. Doses from ingestion and the total dose increased from the late 1960s and into the 1980s, reflecting the increased marine discharges over that period (see Figures 5.1 and 5.2). The impact on the estimated doses is more pronounced for 10 year old children than for 1 year old infants due to the much greater consumption of seafood by the older age groups and hence a higher ingestion dose. As the liquid discharges declined due to the installation of treatment plant, the estimated doses also declined and, although there are minor fluctuations, they have remained at about the same low level from the late 1980s until 2010. The RBM doses from discharges over the period 1993–2010 are mainly due to ingestion of both terrestrial and aquatic foods and are at similar levels to those for the late 1980s and early 1990s. It should be noted that the assumptions regarding the discharges of iodine-131 to atmosphere do not have a significant effect on the estimated RBM doses.

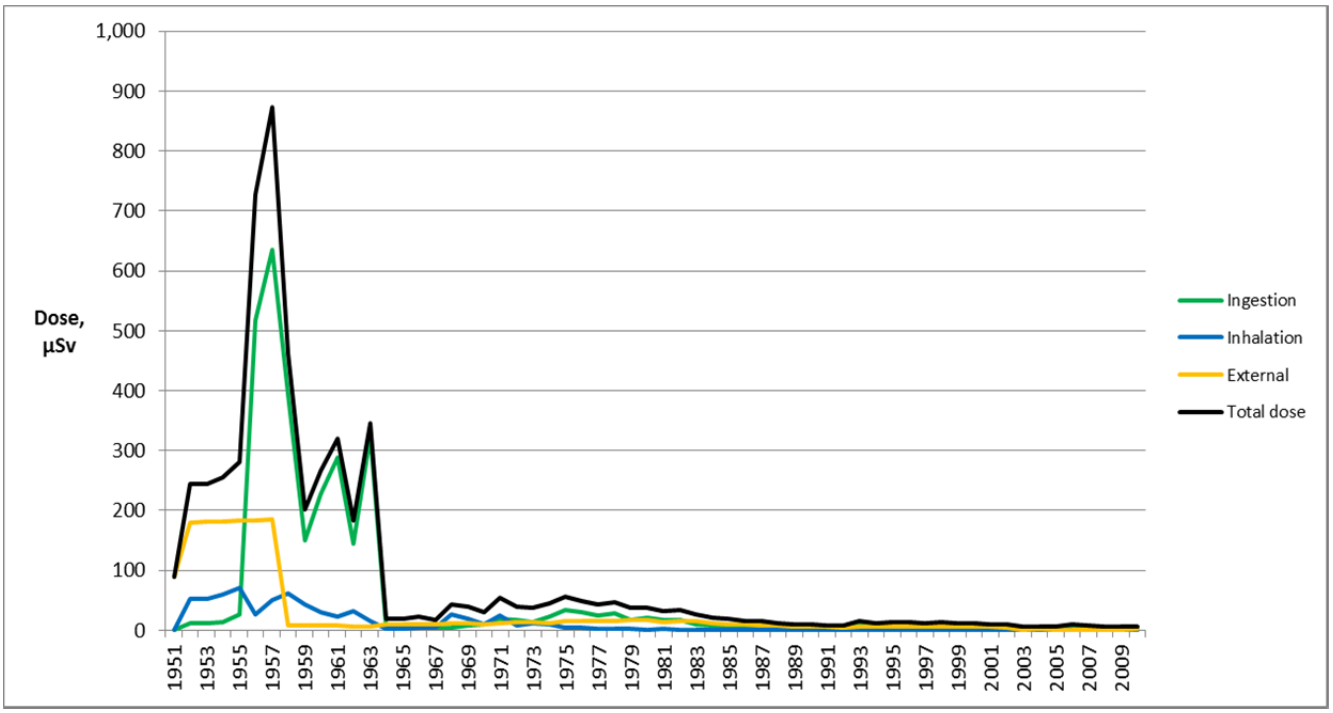

Figure 5.6 Cumulative red bone marrow dose to a 1 year old in Seascale from routine Sellafield discharges, with the dose from intakes of radionuclides integrated to (and including) 24 years of age

Note: the ingestion doses from terrestrial foods from 1993 onwards include contributions from residual contamination in the environment from the Chernobyl accident, fallout from nuclear weapons testing and UO_2 discharges from the Windscale piles. This is about half of the dose from 1993–2010

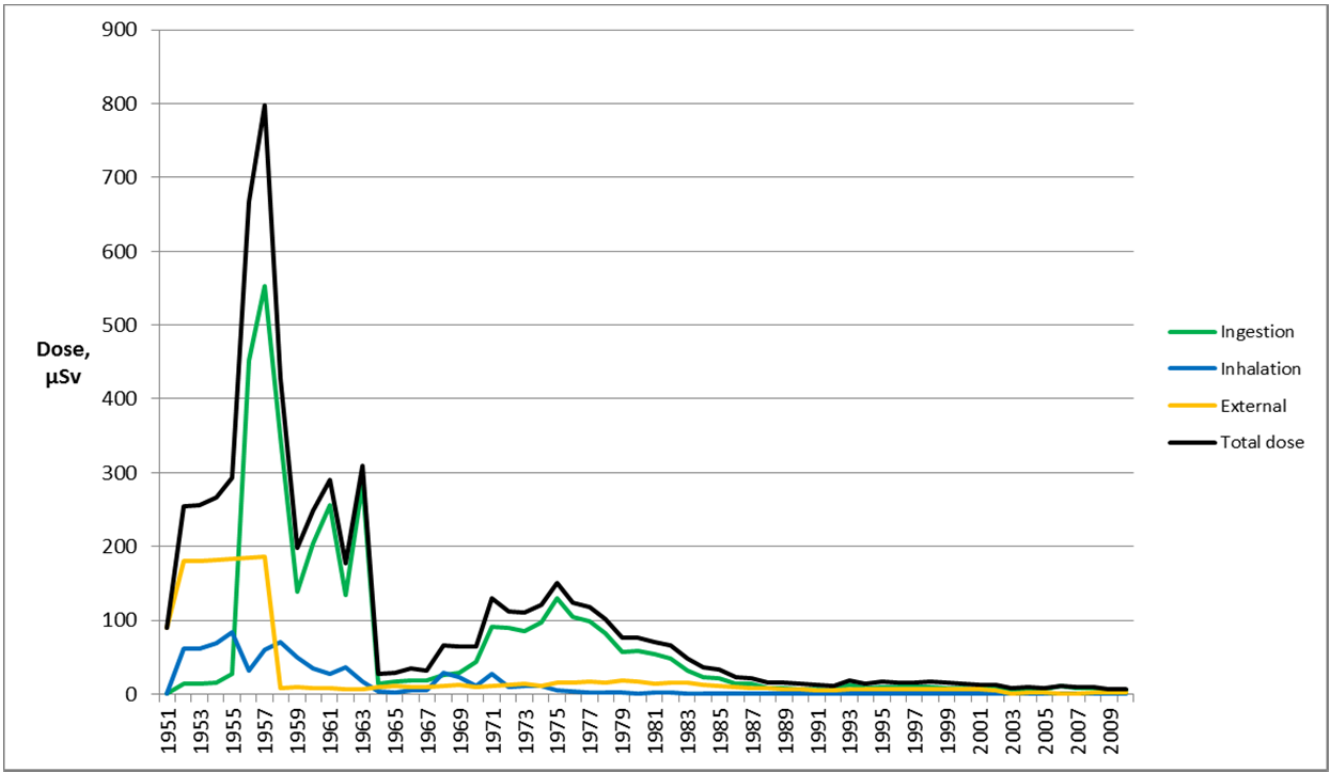

Figure 5.7 Cumulative red bone marrow dose to a 10 year old in Seascale from routine Sellafield discharges, with dose from intakes of radionuclides integrated to (and including) 24 years of age
Note: the ingestion doses from terrestrial foods from 1993 onwards include contributions from residual contamination in the environment from the Chernobyl accident, fallout from nuclear weapons testing and UO_2 discharges from the Windscale piles. This is about half of the dose from 1993–2010

5.22 Doses to the thyroid from Sellafield discharges (excluding those due to the Windscale fire) were also estimated over the same period for 1 and 10 year old children and the results are shown in Figures 5.8 and 5.9, respectively. Figures 5.8a and 5.9a show iodine-131 discharges to atmosphere assuming 10% of the dissolver inventory was released during 1952–1966, while Figures 5.8b and 5.9b assume 1% of the dissolver inventory was released. The doses reflect the pattern of discharges of iodine-131 shown in Figures 5.5a and b, with peaks in doses in the 1950s. As expected, the early thyroid doses are some five to six times higher when 10% of the iodine-131 in the dissolver inventory is assumed to be discharged rather than when only 1% is discharged. These early doses are mainly due to ingestion of iodine-131 in terrestrial foods, particularly milk. For the 10% discharge case the contribution from other routes of exposure is relatively very small. If a 1% discharge is assumed then there is a significant proportionately greater contribution from other surces, such as external irradiation due to the argon-41 discharges from the Windscale piles, particularly for 10 year old children. The general increase in marine discharges in the late 1970s and early 1980s has only a minor overall impact on the thyroid doses for 1 and 10 year old children if 10% of the iodine-131 is discharged to atmosphere, but makes a higher proportional contribution, particularly for 10 year old children, if only 1% is discharged.

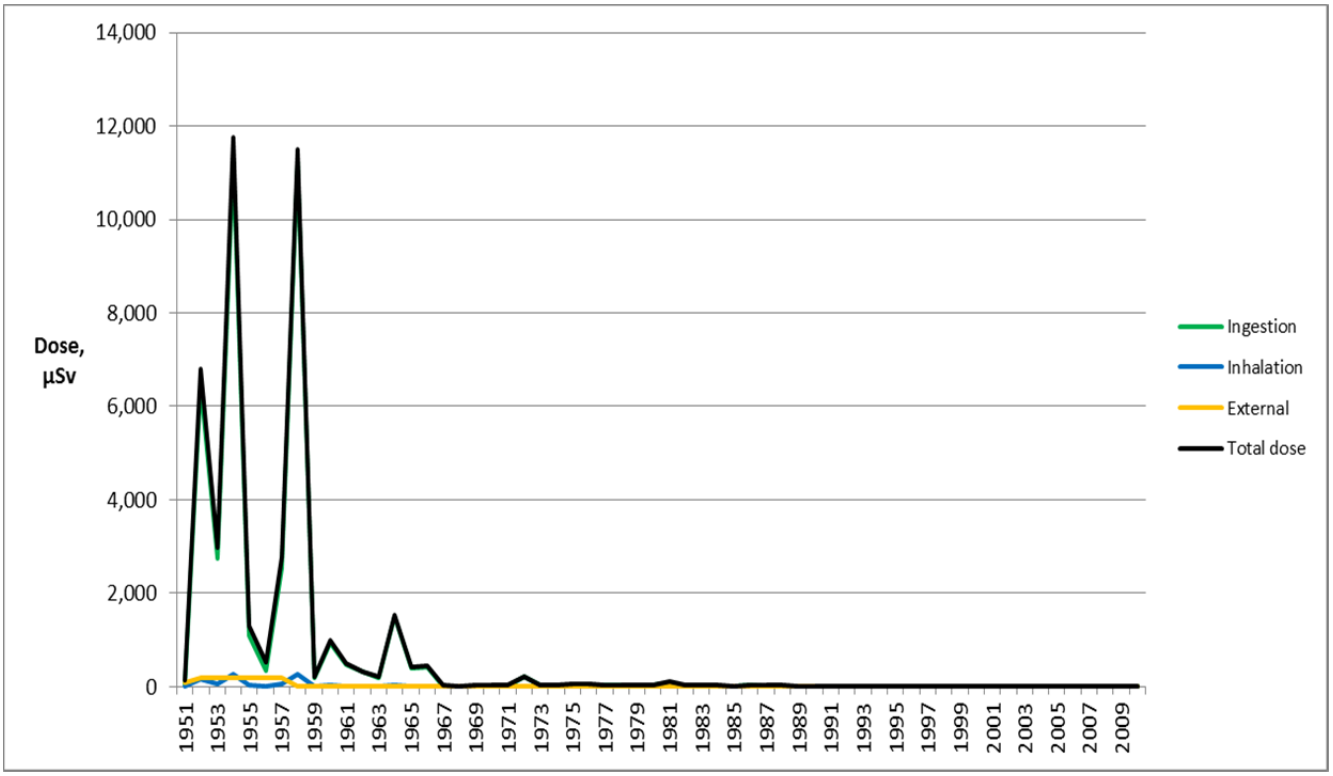

Figure 5.8a Thyroid dose to a 1 year old in Seascale from routine Sellafield discharges based on 10% of the iodine-131 inventory in the dissolver being discharged during 1952–1966

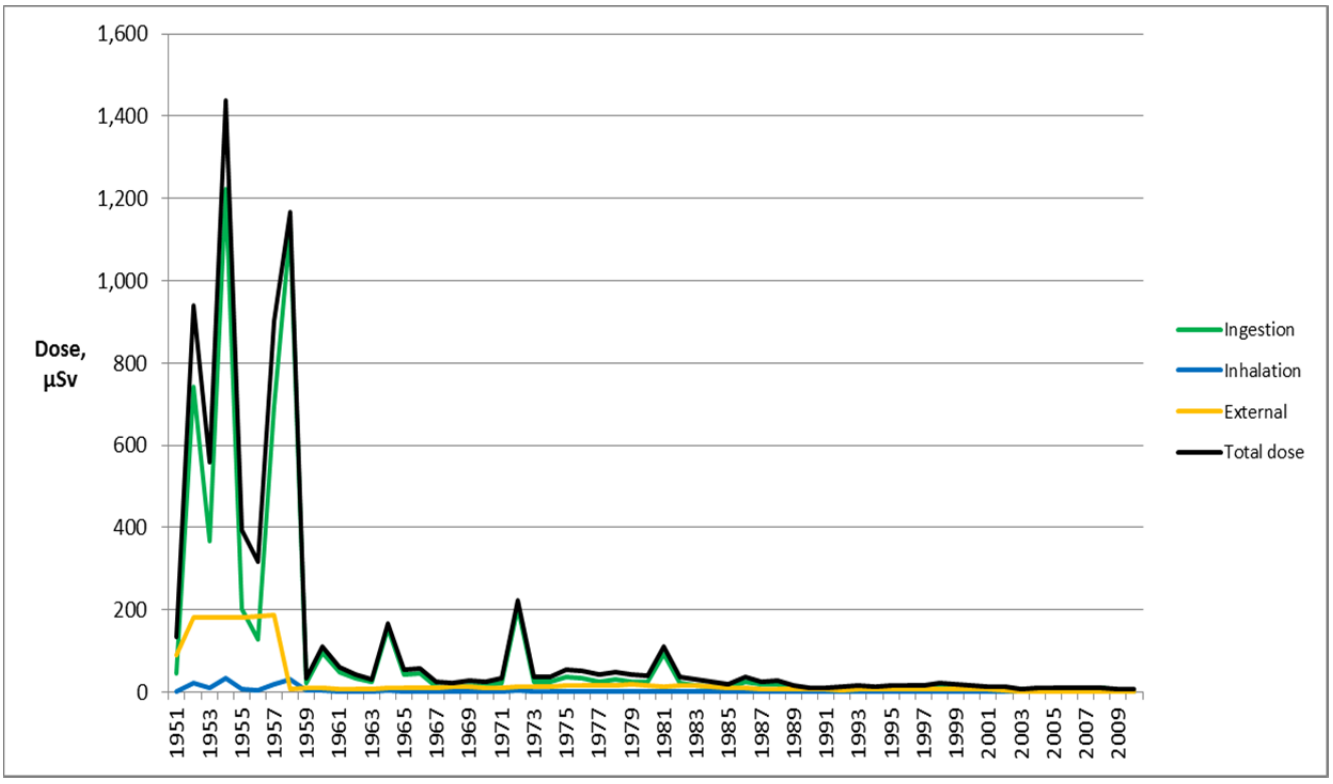

Figure 5.8b Thyroid dose to a 1 year old in Seascale from routine Sellafield discharges based on 1% of the iodine-131 inventory in the dissolver being discharged during 1952–1966

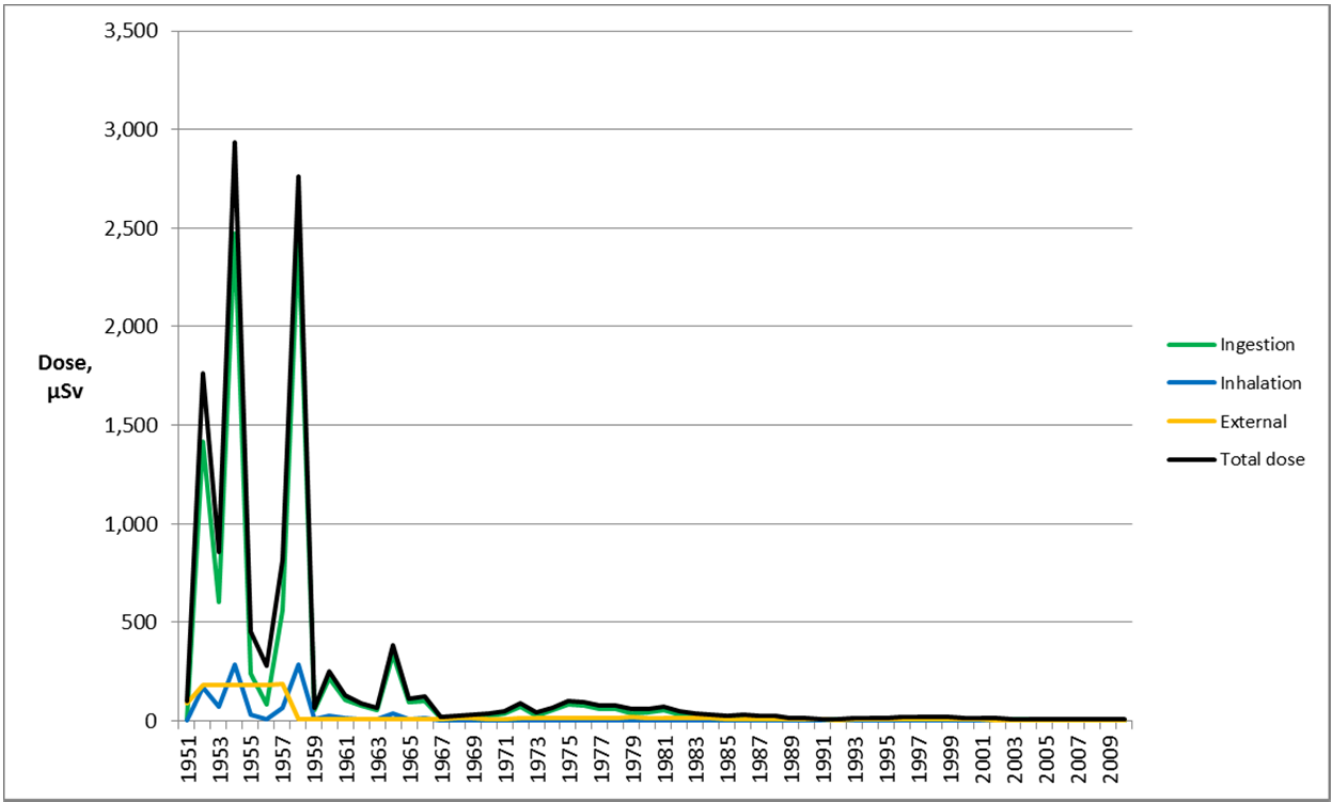

Figure 5.9a Thyroid dose to a 10 year old in Seascale from routine Sellafield discharges based on 10% of the iodine-131 inventory in the dissolver being discharged during 1952–1966

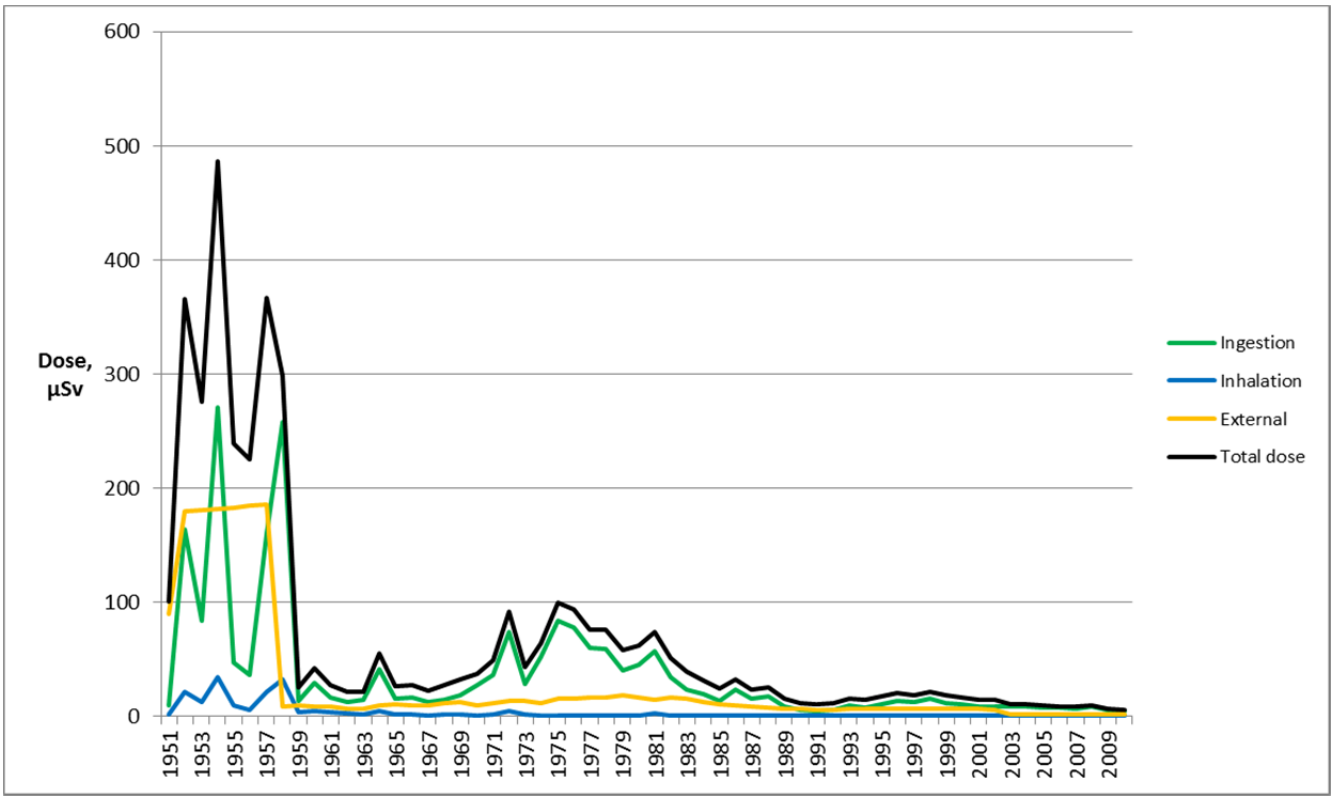

Figure 5.9b Thyroid dose to a 10 year old in Seascale from routine Sellafield discharges based on 1% of the iodine-131 inventory in the dissolver being discharged during 1952–1966

Radiation doses from all routes of exposure

5.23 The main source of radiation exposure throughout 1950–2010 was that due to natural background radiation, which remained constant throughout the period (Jones et al, 2007) and gave an estimated annual dose* to the RBM of 1.4 mSv for 1 year olds and 1.2 mSv for 10 year olds. Another important source of exposure is medical examinations, particularly diagnostic X-rays including dental X-rays; while these are delivered on an individual basis (ie not all people are exposed), average population doses are calculated and are used here. There have been changes in the type and frequency of medical examinations during the period considered, but the effect on doses to the RBM is not large. Recent trends are taken from Hart and Wall (2005), Jones et al (2007) and Hart et al (2010). Average estimated annual doses for children increased slightly since the early 1990s, from about 0.05 to 0.09 mSv by 2010 (Jones et al, 2007). The doses to the thyroid from natural background radiation are approximately the same as those to the RBM, while average thyroid doses from medical examinations are much lower than those to the RBM.

5.24 Doses from fallout from the atmospheric testing of nuclear weapons were greatest in the late 1950s and in early 1960s prior to the limited test ban treaty. Although fallout contributes to exposures to the present day, the levels of dose from this source have continued to decline. For a 10 year old in 1962, when the estimated doses were highest, the annual RBM doses were about 0.3 mSv, decreasing to about 0.01 mSv by the early 1990s (Simmonds et al, 1995) and to about 0.005 mSv by 2010. Account has been taken of the decline of activity concentrations in the environment since the early 1990s. The approach taken to estimate fallout levels in the environment is consistent with more recent calculations by Wright et al (1999). Thyroid doses from weapons fallout for a 10 year old are similar to the RBM doses; however, the annual thyroid doses for a 1 year old are higher than the RBM doses and are estimated to be almost 1 mSv at their highest in the early 1960s. This is due to the higher consumption of milk containing iodine-131 by 1 year olds. The accident at the Chernobyl nuclear power station in Ukraine in 1986 led to a slight increase in radiation doses in the UK and was taken into account in the analyses carried out for the fourth COMARE report. These doses have decreased with time and are currently low. In 1986, when the accident occurred, estimated RBM doses to 1 and 10 year old children were about 0.2 mSv (Simmonds et al, 1995). The doses have steadily decreased and are estimated to be about 0.01 mSv in 2010, taking into account the decline in activity concentrations in the environment. Thyroid doses in 1986 were an order of magnitude higher than RBM doses for a 1 year old and about a factor of four higher for a 10 year old due to ingestion of iodine-131 in milk, based on the measurements reported in NRPB-R276. Thyroid doses subsequently decreased rapidly, becoming similar to those for the RBM.

5.25 There are also contributions to the estimated overall exposures from the accidental release from the Windscale fire in 1957 and from the inadvertent releases of uranium oxide particles from 1952–1957. The annual RBM doses from the Windscale fire were about 1 mSv in 1957 falling rapidly to about 0.005 mSv in subsequent years. The estimated average thyroid doses due to the Windscale fire reached 25 mSv in 1957, mainly due to inhalation of iodine-131; the introduction of restrictions on milk supplies significantly reduced any intakes of iodine-131 by ingestion. The annual RBM doses due to the uranium oxide discharges varied from 0.03 to 0.8 mSv over the period from 1952–1957, while the annual thyroid doses from iodine-131 discharges over this period varied between 0.01 and 1 mSv.

* All doses are the sum of external doses in the year plus the internal doses from intakes in the year integrated to age 24 years.

5.26 Another source of radiation exposure for the population of Seascale is the routine marine discharges from the Marchon Works, a phosphate plant at Whitehaven, to the north of Seascale and formerly operated, among others, by Albright and Wilson. The discharges of enhanced amounts of naturally occurring radionuclides led to elevated levels of polonium-210 and related radionuclides in seafood, and hence intakes by ingestion. The plant stopped discharges in 2002, but elevated levels of polonium-210 are still reported occasionally in the marine environment in Cumbria above typical natural background levels (EA et al, 2012), presumably due to ingrowth from previous discharges of radium-226. In this assessment, which uses the polonium-210 discharge data from NRPB-R276, the annual estimated doses to the RBM from ingestion of seafood were about 0.3–0.4 mSv at their highest in the late 1980s and early 1990s during operation of the plant, decreasing to around 0.02 mSv in 2010, with most activity concentrations in marine foods now being within the range of that expected from natural background. Annual thyroid doses at their highest during operation of the plant were estimated to be about 0.1 mSv and in 2010 were about an order of magnitude lower than doses estimated to the RBM. This reflects the relatively high contribution of polonium-210 and related radionuclides in seafood to doses to the RBM (see paragraph 5.31 for a discussion of changes from previously assessed RBM doses).

5.27 The relative contributions of the different sources of RBM dose for 1 and 10 year olds are illustrated in Figures 5.10a and 5.11a, which show the percentage contribution to the total dose from different routes of exposure for a range of years. At all times, natural background is the major source of exposure and, from the mid-1990s until the present day, it contributes around 90% of the total dose. The contribution from operations at Sellafield varies with time as expected, with the highest estimated for 1953–1955 at about 15%, up to 40% in 1954, including the uranium oxide particle discharges. Since the mid-1950s this has reduced to 5% or lower until 2010, except for the mid-1970s, when the contribution approached 10% for 10 year old children due to ingestion of seafood during the time when there was an increase in liquid discharges. This increase is not seen for 1 year old infants, as their intake of seafood is much lower than that for 10 year olds. There is no effect on the RBM doses of assuming different discharges of iodine-131 to atmosphere. Figures 5.10b and 5.11b show the absolute dose values for particular years.

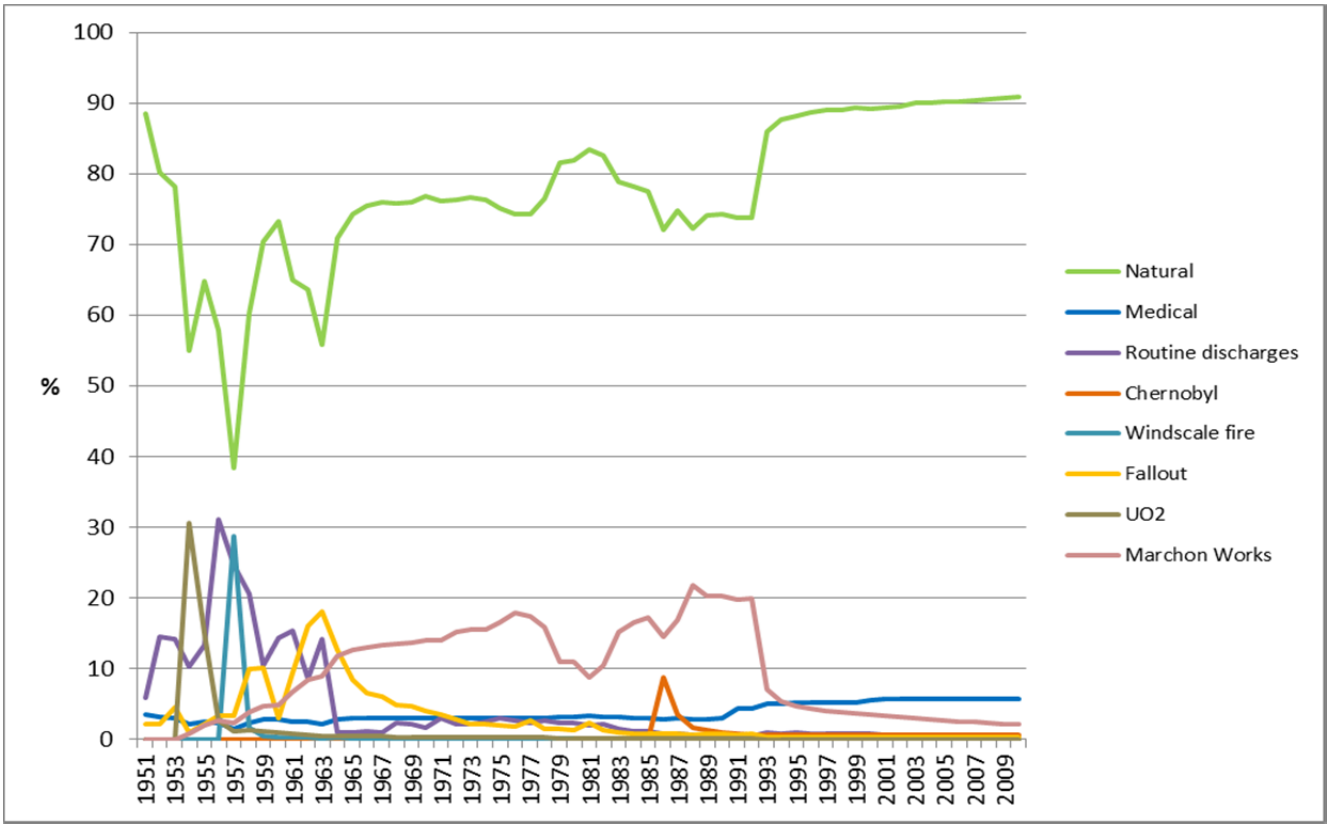

Figure 5.10a Percentage contributions from different sources to the annual red bone marrow doses to 1 year old children in Seascale from 1950– 2010 resulting from exposure to radionuclides at age 1 year in the given years, integrated to (and including) 24 years of age

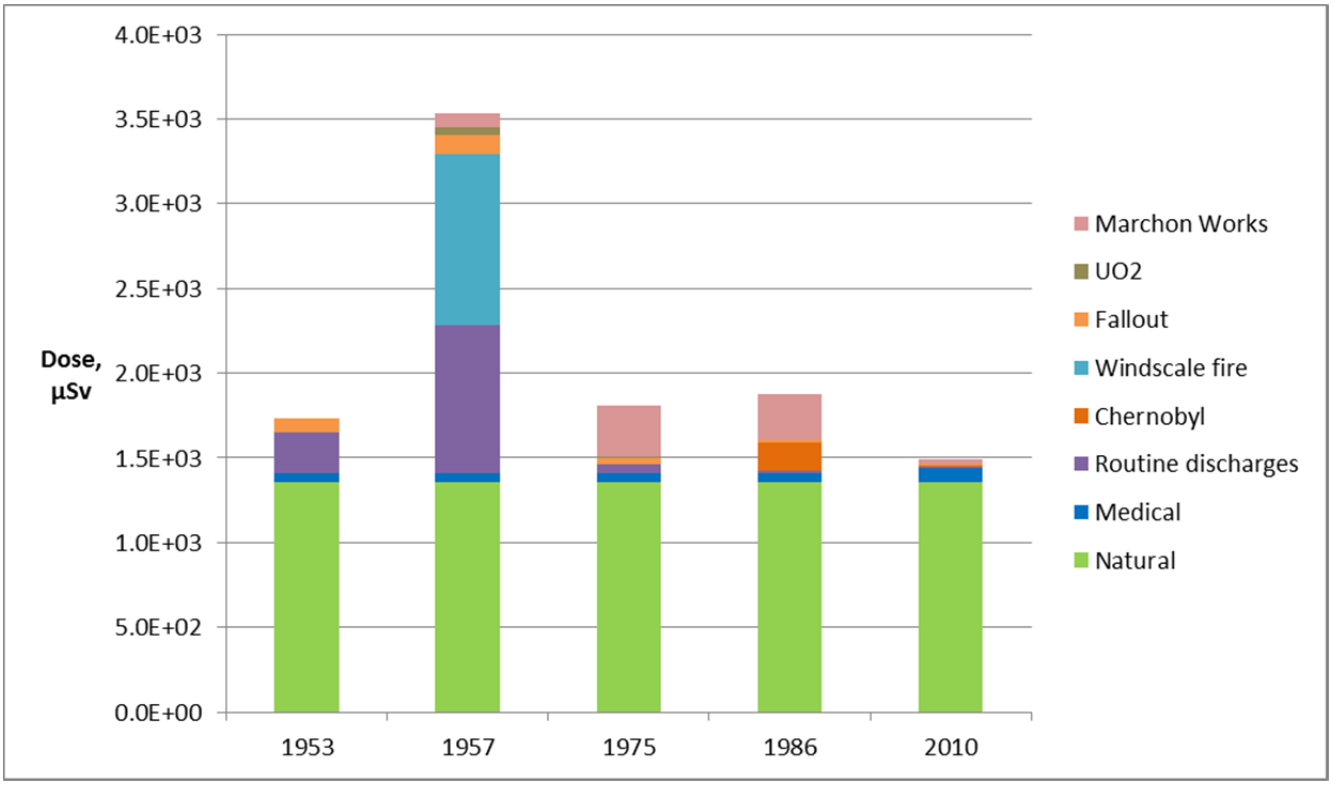

Figure 5.10b Absolute values of red bone marrow dose resulting from exposure in Seascale to radionuclides at age 1 year in the given years, integrated to (and including) 24 years of age

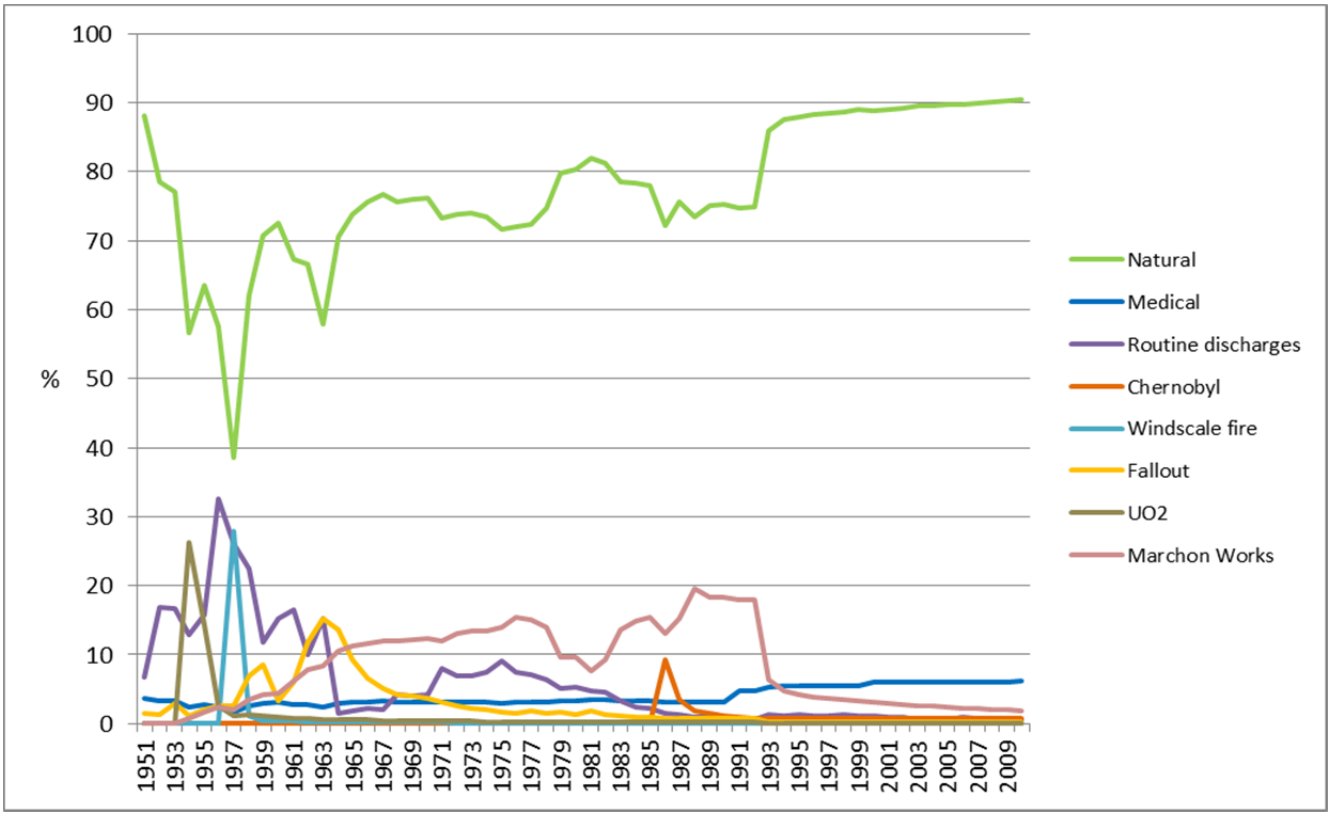

Figure 5.11a Percentage contributions from different sources to the annual red bone marrow doses to 10 year old children in Seascale from 1950–2010 resulting from exposure to radionuclides at age 10 years in the given years, integrated to (and including) 24 years of age

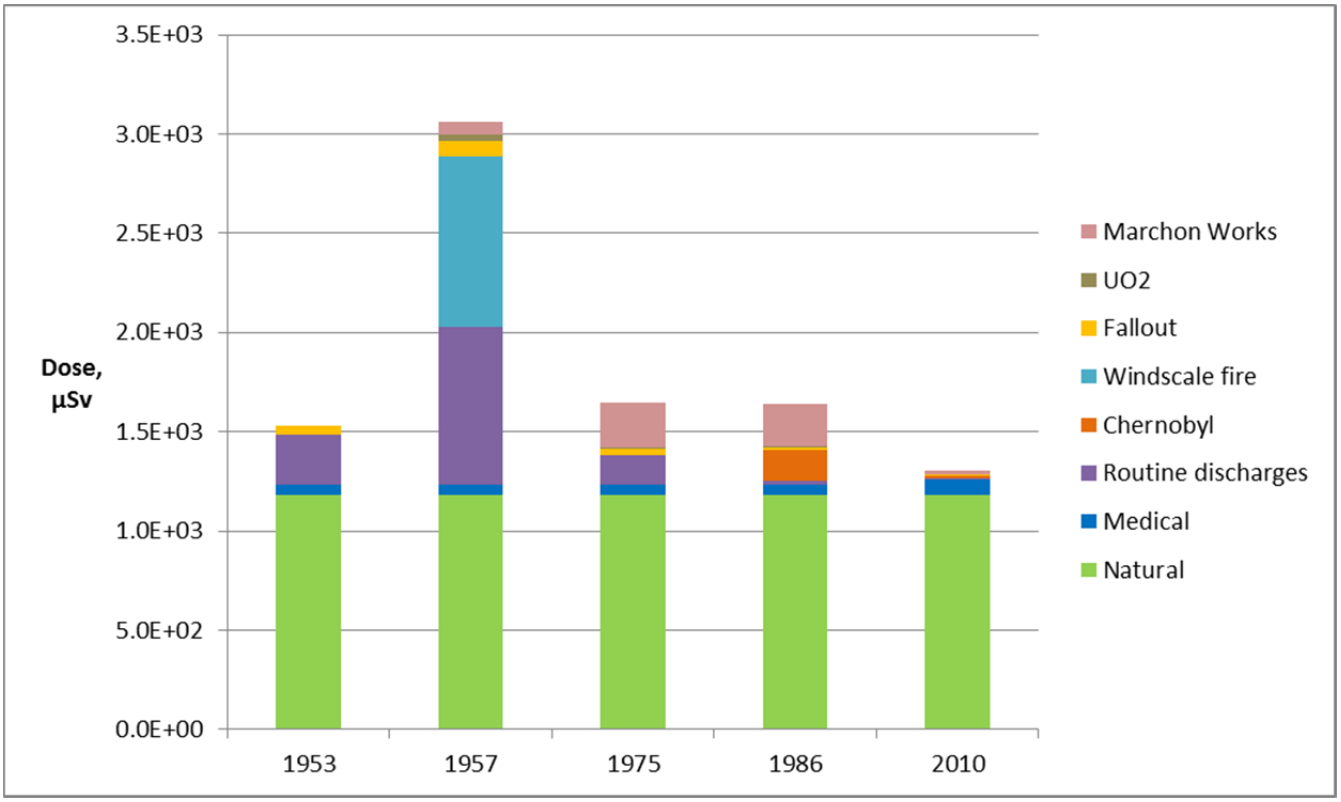

Figure 5.11b Absolute values of red bone marrow dose resulting from exposure in Seascale to radionuclides at age 10 years in the given years, integrated to (and including) 24 years of age

5.28 The situation is slightly different for estimated thyroid doses for 1 and 10 year old children. The main contribution to the thyroid dose is natural background radiation for most of the period, as shown in Figures 5.12a and 5.13a for 1 and 10 year olds, respectively (based on a 10% iodine-131 release from the dissolver during 1952–1966). In 1957 the average thyroid dose to a 10 year old child increased to 25 mSv, with 95% of this dose due to the iodine-131 releases from the Windscale fire. In 1952, 1954 and 1958 the contribution to the thyroid dose from routine site discharges was greater than that from natural background radiation, with up to 70% due to discharges, and the total doses were correspondingly greater. In most other years, natural background radiation contributed more than 80% of the thyroid dose to 10 year old children. In 1986, releases from Chernobyl were calculated to contribute about 40% of the thyroid dose, with 55% of the thyroid dose coming from natural background radiation, as shown in Figure 5.13a. Similar results are seen for the 1 year old infant, with natural background contributing over 85% of the thyroid dose in many years except for the period 1952–1966. In 1952, discharges from the site contributed 80% of the thyroid doses, while in 1957 the Windscale fire led to about 85% of the thyroid dose to a 1 year old infant. In 1986 the Chernobyl accident contributed about 65% of the thyroid dose to 1 year olds, with 30% from natural background and only 1% from discharges from Sellafield, as shown in Figure 5.12a. In the early 1960s, the thyroid doses from the atmospheric testing of nuclear weapons contributed between 15% and 30%, in addition to a maximum of about 15% from discharges from the Sellafield site. Figures 5.12b and 5.13b show the absolute dose values for particular years.

5.29 The results presented in Figures 5.12 and 5.13 assume that 10% of the iodine inventory of the dissolvers was discharged between 1952 and 1966 (see paragraph 5.13). If it was assumed that only 1% was discharged, the thyroid doses due to discharges in the 1950s and early 1960s would be correspondingly lower. Assuming a 1% release, the estimated annual thyroid doses from routine discharges are less than those from natural background, except in 1954 for 1 year olds, when doses from routine discharges and natural background radiation were about the same, in 1957 (from the Windscale fire) and to some extent in 1986 (from Chernobyl), when doses due to natural background were not the major contribution to the total thyroid dose.

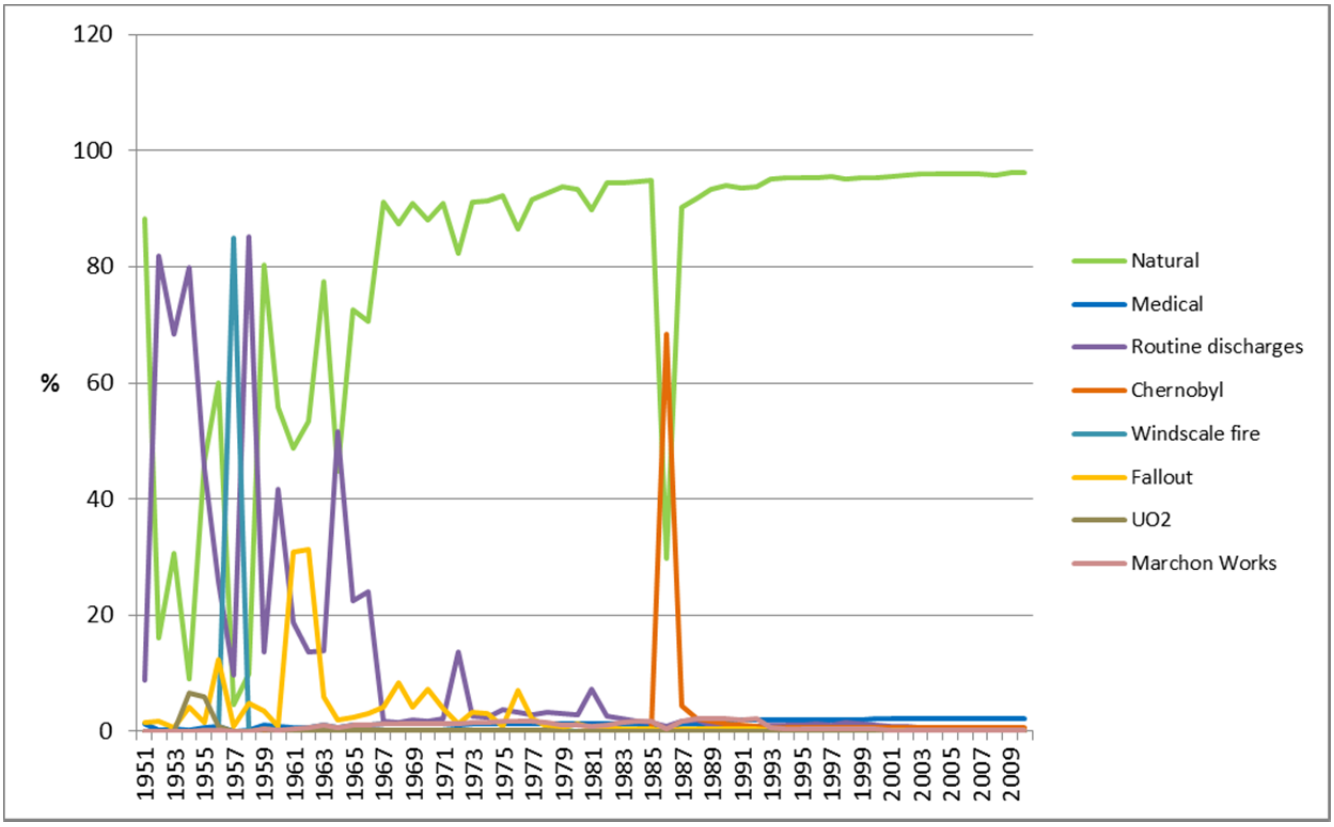

Figure 5.12a Percentage contributions from different sources to the annual thyroid doses to 1 year old children in Seascale from 1950–2010 resulting from exposure to radionuclides at age 1 year in the given years, integrated to (and including) 24 years of age

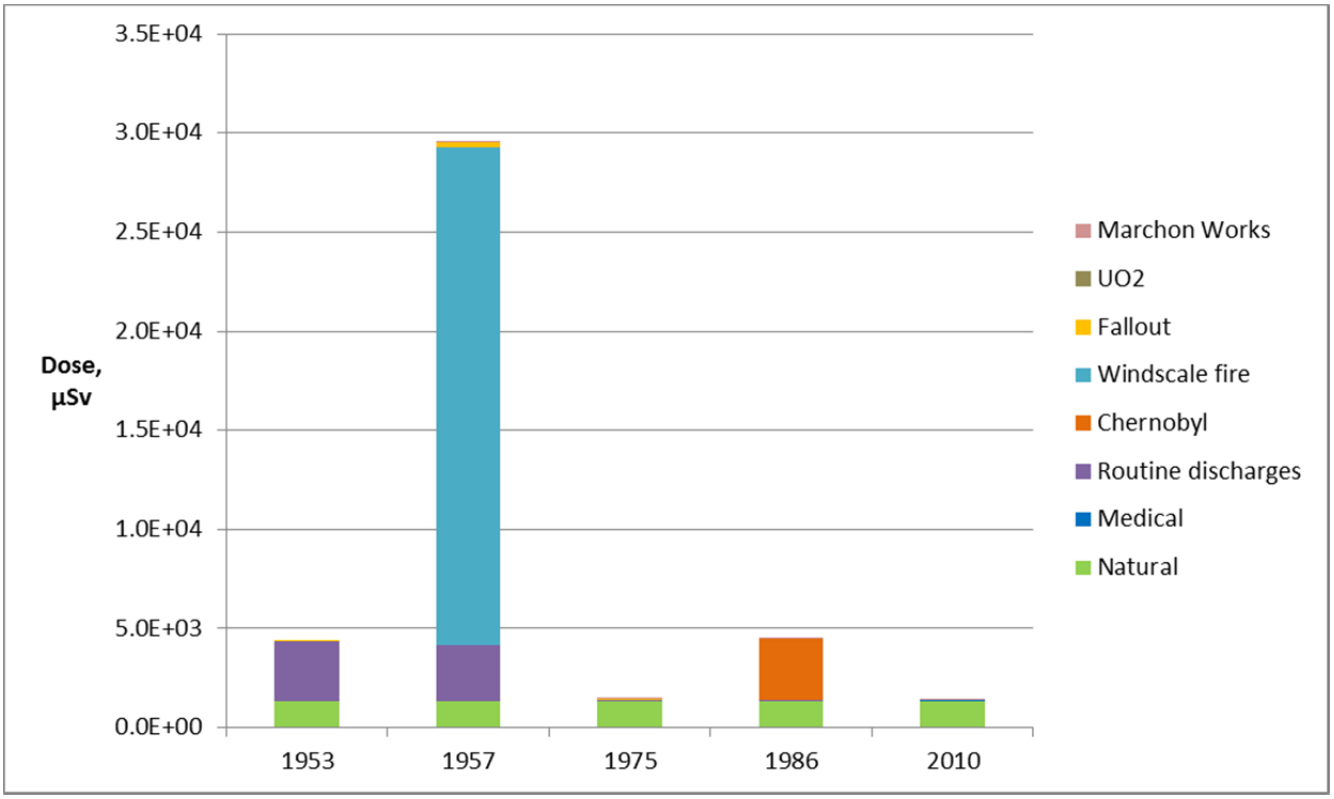

Figure 5.12b Absolute values of thyroid dose resulting from exposure in Seascale to radionuclides at age 1 year in the given years, integrated to (and including) 24 years of age

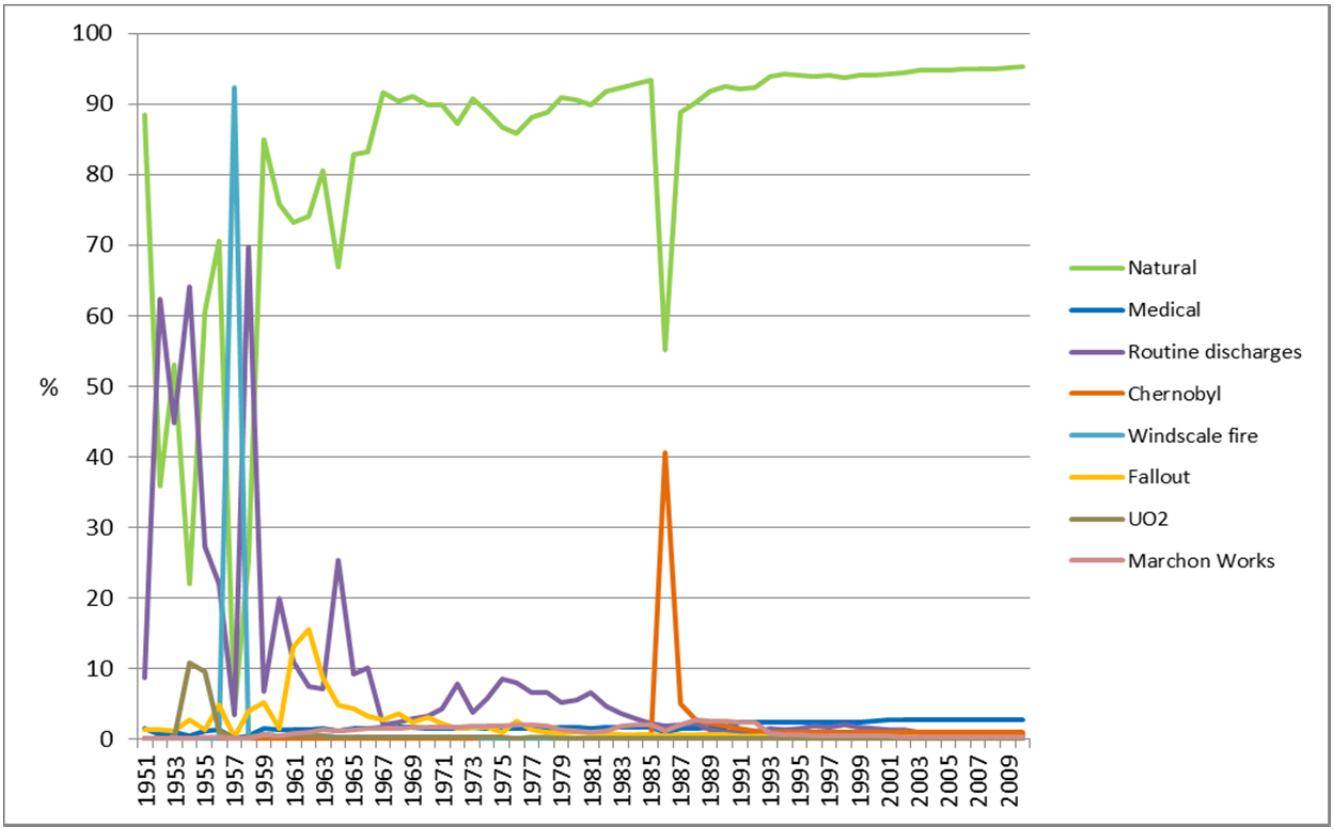

Figure 5.13a Percentage contributions from different sources to the annual thyroid doses to 10 year old children in Seascale from 1950– 2010 resulting from exposure to radionuclides at age 10 years in the given years, integrated to (and including) 24 years of age

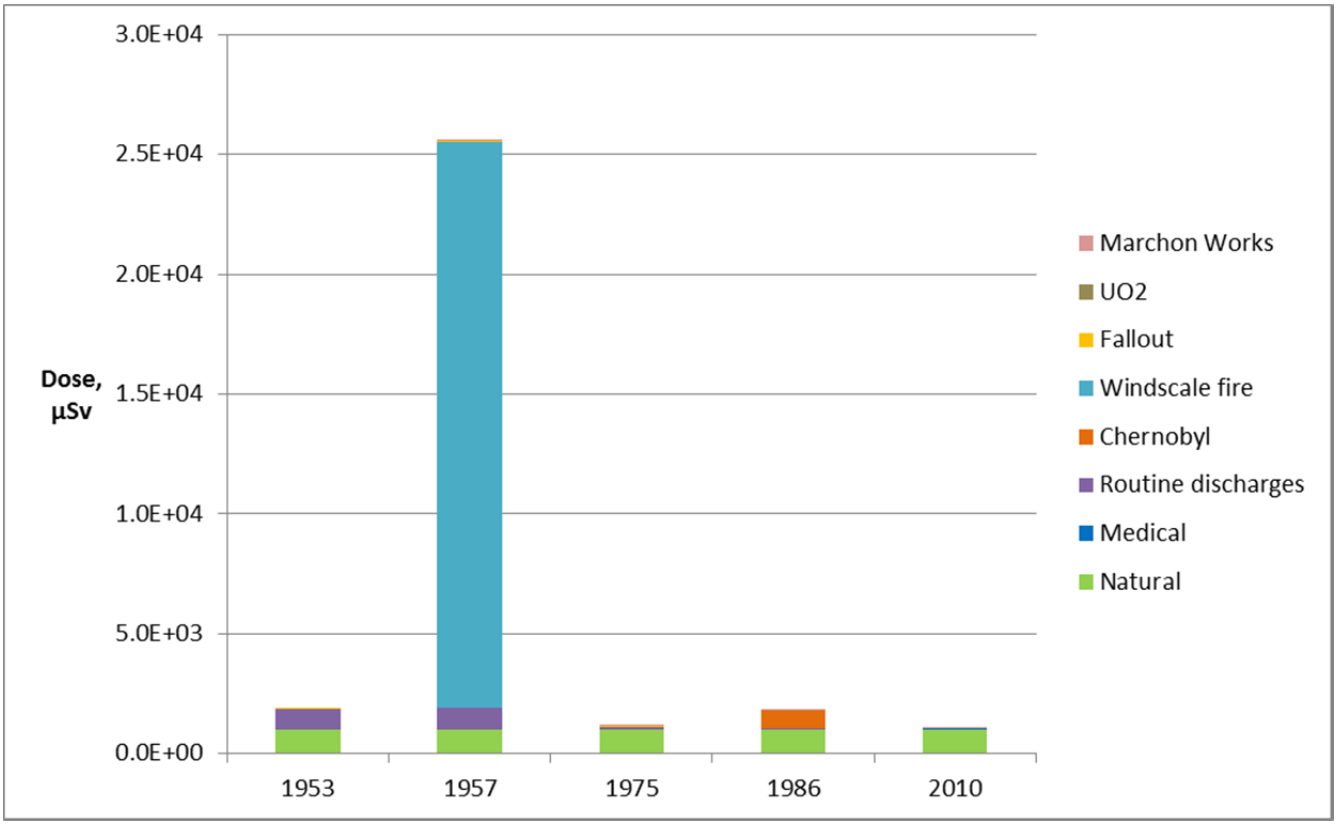

Figure 5.13b Absolute values of thyroid dose resulting from exposure in Seascale to radionuclides at age 10 years in the given years, integrated to (and including) 24 years of age

Effects of changes in key parameter values since publication of the fourth COMARE report

5.30 As discussed, the approach adopted was generally to use the same parameter values as were used in the assessment carried out for the fourth COMARE report as described in NRPB-R276 (Simmonds et al, 1995). However, significant changes in key parameter values were considered and their implications for the assessed doses, and hence associated risks, were addressed.

5.31 With respect to RBM dose, there are some relatively minor changes to the discharge data for the Sellafield site since publication of the fourth report, as noted above. These do not have any significant impact on the estimated RBM dose arising from liquid or aerial discharges from the Sellafield site. The discharges of iodine-131 to atmosphere due to the reprocessing of short-cooled fuel have been revised based on additional data from the NDA. This affects the timing and size of the discharges between 1952 and 1966. As discussed earlier, the NDA advised using a discharge of 10% iodine-131 from the total inventory of the dissolver, but there are indications that this may be too cautious, ie discharges will be overestimated (Wakeford, 2007a). Therefore, the possibility that only 1% of the iodine-131 was discharged to the atmosphere has also been considered here. Iodine-129 discharges to atmosphere between 1952 and 1966 from reprocessing of fuel have also been revised and are between 10 and 100 times lower than those used previously in NRPB-R276. The estimated contributions of marine discharges from both the Sellafield site and the Marchon Works to RBM doses have been increased substantially in this assessment, using an updated methodology that includes an order of magnitude increase in the transfer of polonium-210 to fish. It should be noted, however, that the limited available data that allow comparisons between modelled (Smith and Simmonds, 2009) and measured (MAFF, 1993, MAFF & SEPA 1997; EA et al, 2005) levels of polonium-210 in seafood suggest that doses from this source are likely to be overestimates. The use of a radiation weighting factor of 20 for alpha particles in the calculation of the RBM equivalent doses may also contribute to an overestimation of the risks attributable to the Sellafield and Marchon discharges, since it has been suggested that a value of two for relative biological effectiveness is more appropriate for incorporated alpha emitters (USEPA, 2011).

5.32 The values of dose per unit intake by inhalation and ingestion used to assess the RBM doses were generally those used previously. However, since publication of the fourth COMARE report and NRPB-R276 there have been some changes to the dose coefficients (dose per unit intake values) published by the International Commission on Radiological Protection (ICRP, 2012). Most changes are small, but there are larger differences for a few radionuclides (in addition to polonium-210) whose doses contribute to the overall doses from discharges from the Sellafield site. Of note are the increases in the RBM dose coefficients for inhalation and ingestion for plutonium-241, where the values have increased by factors of between about 8 (inhalation) and 30 (ingestion) for children. Also of note is the increase in the ingestion dose coefficient for strontium-90 for children by between 30% and 60%. If the new dose coefficients were used, the increase in doses from inhalation would be less than 15%. The increase in RBM dose coefficients for ingestion of strontium-90 would lead to ingestion doses in marine foods increasing by up to about 25% for 1 year olds, with increases about a factor of two lower for 10 year olds. With the updated dose coefficients, ingestion doses from terrestrial foods would increase by 30–60% for 1 year olds and 15–30% for 10 year olds. This increase would also be seen for doses from weapons testing fallout and the uranium oxide particle discharges in the 1950s. The overall impact of the changes in the dose coefficients is small as the total estimated RBM doses would increase by a maximum of 5%.

5.33 The previous study considered the exposure of the embryo, foetus and breastfed infant due to intakes of radionuclides by the mother using information that was available at the time. Since then, the ICRP has published comprehensive compilations of dose coefficients for these cases (ICRP, 2012). There are differences between the values used in the previous study and the more recent compilations. However, given the relatively small contribution of the exposure of the embryo, foetus and breastfed infant to the overall exposure of a child and their risk of a radiation-induced leukaemia or other cancers, these differences are not of significance.

5.34 There have also been advances in knowledge of the models for the induction of radiation-induced cancer and how they are applied. To see the possible implications of these changes, the risk of exposure-induced cancer (REIC) was used as a measure of attributable risk from radiation exposure (Thomas et al, 1992). For a given dose, REIC is the cumulative probability of the radiation-induced incidence of a specific cancer up to a particular attained age. The REIC calculations rely upon the use of a risk model derived from the epidemiological literature.

5.35 Age- and sex-specific leukaemia and NHL and thyroid cancer incidence rates for England and Wales were used for the risk calculations. Incidence rates for the age groups under consideration in this report have remained relatively stable over recent decades, so incidence data for the 1990s, and for the 1990s and 2000s, have been used for the risk calculations for leukaemia and NHL and for thyroid cancer, respectively. The risk models chosen for the analysis of leukaemia are the excess relative risk (ERR) and the excess absolute risk (EAR) models from the UNSCEAR 2006 report, using the linear-quadratic dose-response models with the parameters given in Table 46 of that report (UNSCEAR, 2008). For the present calculations, leukaemia risks were derived by applying both the EAR and ERR models in equal proportion (50% EAR and 50% ERR). A 2-year minimum latency period for leukaemia was used for the calculation, with the cumulative risk calculated to the 25^{th} birthday. For thyroid cancer, the risk is derived from the (linear) risk models presented in Table 57 of the UNSCEAR 2006 report, taking a 100% excess relative risk (ERR) model (ICRP, 2007). A 5-year minimum latency period for thyroid cancer was used in the calculation and the period of follow up was to age 60 years; no dose and dose rate reduction factor (DDREF) was applied to the risks resulting from the models for thyroid cancer.

5.36 In the previous study, Appendix E of NRPB-R276 gave tables of annual absorbed doses to the RBM and consequent risks of fatal leukaemia to 25 years of age for individuals born in Seascale in 1950 and in 1970, for high and low LET radiation for various exposure routes (Simmonds et al, 1995). The absorbed dose values to the RBM from NRPB-R276 were used with the more recent risk models to estimate leukaemia and NHL incidence risks to males and females, which can be compared with the previous estimates of the risks of leukaemia mortality. Tables 5.5 and 5.6 summarise the results by source of exposure and show the cumulative equivalent doses to RBM to the 25^{th} birthday, the consequent cumulative risks of leukaemia mortality from NRPB-R276 and the leukaemia and NHL incidence estimated using the approach outlined in the previous paragraph, specific to individuals born in Seascale in 1950 and 1970. The baseline risks of leukaemia and NHL incidence based on national statistics are also given in Tables 5.5 and 5.6. The results show that using the recent UNSCEAR risk models tends to reduce the estimated risks of radiation-induced leukaemia from those presented in NRPB-R276, with females having slightly lower risks than males (reflecting the lower baseline risks). Natural background radiation gives the highest cumulative dose to the RBM,

and consequent risk of radiation-induced leukaemia, of any of the sources of radiation exposure considered.

5.37 Although the UNSCEAR 2006 report models were used for these calculations, radiation-induced leukaemia risk models from other sources (the BEIR VII report (National Research Council, 2006) and an analysis by Hsu et al (2013) of leukaemia incidence in the Japanese atomic bomb survivors) were also used as a sensitivity check. Using these risk models increased the estimates of radiation-induced leukaemia and NHL incidence risks by less than a factor of two. In addition, owing to the variation of ERR/EAR model mixtures used by various bodies for calculating leukaemia risk (NAS, 2006; ICRP, 2007) a sensitivity check using a 100% ERR model and a 100% EAR model has been applied. Use of a 100% ERR model rather than an ERR/EAR 50/50 model mixture would increase the risk estimates by less than a factor of two, while use of a 100% EAR model would decrease the risk estimates by a factor of around two to three. These sensitivity checks demonstrate that the leukaemia risk estimates shown in Tables 5.5 and 5.6 are reasonably robust.

Table 5.5 Estimated average cumulative 25-year doses to the red bone marrow, mortality risk (from NRPB-R276) and modelled average risks of radiation-induced leukaemia and NHL incidence to 25 years of age for an individual born in Seascale in 1950

	Total individual RBM dose (mSv)	**Individual risk (leukaemia mortality, NRPB-R276)**	**Individual male risk (leukaemia and NHL incidence)**	**Individual female risk (leukaemia and NHL incidence)**
Baseline risk	–	–	$1.3\ 10^{-3}$	$9.2\ 10^{-4}$
Routine discharges	6.2	$2.4\ 10^{-5}$	$3.3\ 10^{-5}$	$2.1\ 10^{-5}$
UO_2 releases	1.3	$1.3\ 10^{-5}$	$9.8\ 10^{-6}$	$6.2\ 10^{-6}$
Windscale fire	$9.4\ 10^{-1}$	$1.8\ 10^{-5}$	$5.3\ 10^{-6}$	$3.3\ 10^{-6}$
Marchon Works*	2.8	$2.0\ 10^{-7}$	$6.2\ 10^{-6}$	$3.8\ 10^{-6}$
Weapons fallout	2.3	$1.6\ 10^{-5}$	$9.7\ 10^{-6}$	$6.3\ 10^{-6}$
Medical exposures	1.7	$2.2\ 10^{-5}$	$7.9\ 10^{-5}$	$5.4\ 10^{-5}$
Total man-made radiation	$1.5\ 10^{1}$	$9.3\ 10^{-5}$	$7.2\ 10^{-5}$	$4.6\ 10^{-5}$
Natural radiation	$3.0\ 10^{1}$	$3.0\ 10^{-4}$	$1.9\ 10^{-4}$	$1.3\ 10^{-4}$
Total radiation	$4.5\ 10^{1}$	$3.9\ 10^{-4}$	$2.6\ 10^{-4}$	$1.8\ 10^{-4}$

* Doses from Marchon Works and Sellafield discharges have been increased from those presented in NRPB-R276 for reasons discussed in paragraph 5.31

Table 5.6 Estimated average cumulative 25-year doses to the red bone marrow, mortality risk (from NRPB-R276) and modelled average risks of radiation-induced leukaemia and NHL incidence to 25 years of age for an individual born in Seascale in 1970

	Total individual RBM dose(mSv)	Individual risk (leukaemia mortality, NRPB-R276)	Individual male risk (leukaemia and NHL incidence)	Individual female risk (leukaemia and NHL incidence)
Baseline risk	–	–	$1.3\ 10^{-3}$	$9.2\ 10^{-4}$
Routine discharges	1.4	$9.0\ 10^{-6}$	$8.3\ 10^{-6}$	$5.5\ 10^{-6}$
UO_2 releases	$9.0\ 10^{-2}$	$1.2\ 10^{-6}$	$6.9\ 10^{-7}$	$4.8\ 10^{-7}$
Windscale fire	$3.3\ 10^{-2}$	$2.8\ 10^{-7}$	$2.7\ 10^{-7}$	$1.9\ 10^{-7}$
Marchon Works*	5.3	$5.7\ 10^{-7}$	$3.4\ 10^{-5}$	$2.3\ 10^{-5}$
Chernobyl	$3.5\ 10^{-1}$	$3.1\ 10^{-7}$	$6.3\ 10^{-7}$	$3.9\ 10^{-7}$
Weapons fallout	$6.1\ 10^{-1}$	$7.8\ 10^{-6}$	$6.1\ 10^{-6}$	$4.4\ 10^{-6}$
Medical exposures	1.9	$1.3\ 10^{-5}$	$8.0\ 10^{-6}$	$5.5\ 10^{-6}$
Total man-made radiation	9.7	$3.2\ 10^{-5}$	$5.8\ 10^{-5}$	$3.9\ 10^{-5}$
Natural radiation	$3.0\ 10^{1}$	$2.3\ 10^{-4}$	$1.9\ 10^{-4}$	$1.3\ 10^{-4}$
Total radiation	$4.0\ 10^{1}$	$2.6\ 10^{-4}$	$2.5\ 10^{-4}$	$1.7\ 10^{-4}$

* Doses from Marchon Works and Sellafield discharges have been increased from those presented in NRPB-R276 for reasons discussed in paragraph 5.31

Estimated risks of radiation-induced thyroid cancers

5.38 Estimates were also made of the incidence of radiation-induced thyroid cancers for groups of individuals born in Seascale at particular times. Three groups were considered: those born in 1951 (who would have been 1 year old at a time of high routine iodine-131 discharges from the site), 1956 (who would have been 1 year old in 1957 at the time of the Windscale fire) and 1970. The aim was to estimate the risks to the most exposed young children and then to those born in 1970 as representative of other times when exposures were mainly due to natural background radiation. In all cases the risks presented are those of developing thyroid cancer up to age 60 years resulting from doses of radiation to the thyroid during the first 25 years after birth.

5.39 The results are presented in Tables 5.7–5.9 for an individual born in Seascale in 1951, 1956 or 1970, respectively. It has been assumed that routine discharges of iodine-131 between 1952 and 1966 from reprocessing operations are based on 10% of the dissolver inventory being released (NDA, 2014). If the risks are calculated with 1% of the dissolver inventory released, the risks from routine discharges are lower by a factor of five to six for individuals born in 1951 and 1956; the risks from discharges after 1966 are unchanged. The baseline risks of thyroid cancer incidence based on national statistics are also given in Tables 5.7–5.9. The highest doses and risks are for those born in 1956, with females having a higher risk of radiation-induced thyroid cancer (reflecting the higher baseline risk of thyroid cancer in females). Thyroid doses and consequent risks of thyroid cancer for those born in 1951 and 1956 are greater for Sellafield discharges than for natural background radiation by up to a factor of four, largely due to iodine-131 released from the Windscale fire and early routine operations. Nonetheless, the absolute risks of thyroid cancer from all sources of radiation exposure remain low.

5.40 The ERR model from the UNSCEAR 2006 report was used for the calculations reported here. An alternative ERR model was given in the

BEIR VII report (National Research Council, 2006), and this model was used to produce thyroid cancer incidence risk estimates as a sensitivity check. When this was done, the risks decreased by a factor of around two, showing that risk estimates presented in Tables 5.7–5.9 are reasonably robust. There is a consensus that a 100% ERR model should be used in calculating thyroid cancer risks (National Research Council, 2006; ICRP, 2007), so the UNSCEAR 2006 report EAR model has not been used (and the BEIR VII report does not give an EAR model for thyroid cancer).

Table 5.7 Estimated average cumulative 25-year doses to thyroid and modelled average risks of radiation-induced thyroid cancer incidence to 60 years of age for an individual born in Seascale in 1951

	Total individual thyroid dose (mSv)	**Individual male risk**	**Individual female risk**
Baseline risk	–	$5.2\ 10^{-4}$	$1.4\ 10^{-3}$
Routine discharges	$2.9\ 10^{1}$	$1.4\ 10^{-4}$	$4.0\ 10^{-4}$
UO_2 releases	1.3	$4.9\ 10^{-6}$	$1.5\ 10^{-5}$
Windscale fire	$2.4\ 10^{1}$	$6.9\ 10^{-5}$	$2.1\ 10^{-4}$
Marchon Works	$3.0\ 10^{-1}$	$4.7\ 10^{-7}$	$1.4\ 10^{-6}$
Weapons fallout	2.1	$6.8\ 10^{-6}$	$2.0\ 10^{-5}$
Medical exposures	$5.9\ 10^{-1}$	$1.4\ 10^{-6}$	$4.1\ 10^{-6}$
Total man-made radiation	$5.7\ 10^{1}$	$2.2\ 10^{-4}$	$6.5\ 10^{-4}$
Natural radiation	$2.6\ 10^{1}$	$7.5\ 10^{-5}$	$2.2\ 10^{-4}$
Total radiation	$8.3\ 10^{1}$	$3.0\ 10^{-4}$	$8.7\ 10^{-4}$

Table 5.8 Estimated average cumulative 25-year doses to thyroid and modelled average risks of radiation-induced thyroid cancer incidence to 60 years of age for an individual born in Seascale in 1956

	Total individual thyroid dose (mSv)	**Individual male risk**	**Individual female risk**
Baseline risk	–	$5.5\ 10^{-4}$	$1.6\ 10^{-3}$
Routine discharges	$1.9\ 10^{1}$	$1.0\ 10^{-4}$	$3.2\ 10^{-4}$
UO_2 releases	$1.1\ 10^{-1}$	$5.0\ 10^{-7}$	$1.6\ 10^{-6}$
Windscale fire	$2.5\ 10^{1}$	$1.9\ 10^{-4}$	$5.9\ 10^{-4}$
Marchon Works	$4.0\ 10^{-1}$	$8.3\ 10^{-7}$	$2.7\ 10^{-6}$
Weapons fallout	2.2	$1.1\ 10^{-5}$	$3.5\ 10^{-5}$
Medical exposures	$5.9\ 10^{-1}$	$1.4\ 10^{-6}$	$4.6\ 10^{-6}$
Total man-made radiation	$4.7\ 10^{1}$	$3.0\ 10^{-4}$	$9.5\ 10^{-4}$
Natural radiation	$2.6\ 10^{1}$	$7.7\ 10^{-5}$	$2.5\ 10^{-4}$
Total radiation	$7.3\ 10^{1}$	$3.8\ 10^{-4}$	$1.2\ 10^{-3}$

Table 5.9 Estimated average cumulative 25-year doses to thyroid and modelled average risks of radiation-induced thyroid cancer incidence to 60 years of age for an individual born in Seascale in 1970

	Total individual thyroid dose (mSv)	Individual male risk	Individual female risk
Baseline risk	–	$7.1\ 10^{-4}$	$2.3\ 10^{-3}$
Routine discharges	1.2	$5.0\ 10^{-6}$	$1.8\ 10^{-5}$
UO_2 releases	$3.4\ 10^{-2}$	$1.6\ 10^{-7}$	$5.5\ 10^{-7}$
Windscale fire	$3.3\ 10^{-2}$	$1.5\ 10^{-7}$	$5.3\ 10^{-7}$
Chernobyl	$6.1\ 10^{-1}$	$1.2\ 10^{-6}$	$4.4\ 10^{-6}$
Marchon Works	$5.1\ 10^{-1}$	$1.8\ 10^{-6}$	$6.4\ 10^{-6}$
Weapons fallout	$4.6\ 10^{-1}$	$3.1\ 10^{-6}$	$1.1\ 10^{-5}$
Medical exposures	$6.6\ 10^{-1}$	$2.0\ 10^{-6}$	$7.0\ 10^{-6}$
Total man-made radiation	3.5	$1.3\ 10^{-5}$	$4.8\ 10^{-5}$
Natural radiation	$2.6\ 10^{1}$	$9.9\ 10^{-5}$	$3.5\ 10^{-4}$
Total radiation	$3.0\ 10^{1}$	$1.1\ 10^{-4}$	$4.0\ 10^{-4}$

Radiation exposures to young people in Thurso

5.41 The sources of radiation exposure considered were:

(i) natural background radiation

(ii) medical exposures

(iii) fallout from the atmospheric testing of nuclear weapons

(iv) discharges from the Dounreay site

(v) liquid discharges from the Sellafield site

(vi) releases from the 1986 accident at the Chernobyl nuclear power plant

5.42 For the first three of these sources, the exposures were based on the averages for the UK (see paragraphs 5.23 and 5.24 above), with some limited account taken of local information for Thurso. The exposures from Dounreay discharges were specifically for Thurso throughout the period considered. For Chernobyl the exposures were specific to Thurso where possible, but this was not feasible for later years when the levels of exposure from this route were reduced to very low levels.

5.43 For the second COMARE report (COMARE, 1988) the exposures were based on estimated levels of radiation in the Thurso environment for each year from 1958–1984. The report made use of work carried out by the NRPB to assess the exposures and risks to the local population for the 1986 Dounreay Inquiry (Dionian et al, 1986; Hill and Cooper, 1986). Full details of the assessment of the levels in the environment were discussed in the report NRPB-R195 (Hill and Cooper, 1986). For the current study the values given in that report were reviewed and the study repeated to cover the period from 1958–2010 using similar approaches to those used originally. In doing this, it was particularly important to review the discharges from the Dounreay site as well as the available environmental monitoring data. The levels of radiation in the environment were then used to estimate radiation doses to average 1 and 10 year old children living in Thurso as a function of time, as discussed below.

5.44 The sixth COMARE report (COMARE, 1999) considered whether the ingestion of radioactive particles found in the general vicinity of Dounreay could be associated with the reported excess of childhood leukaemia and NHL; however, an implausibly large number of particles was determined to be required. The publicly accessible beaches around Dounreay continue to be monitored for radioactive particles, together with the Dounreay foreshore*. In their case–control study of childhood leukaemia and NHL in Caithness, Urquhart et al (1991) found an apparent association between the use of beaches around Dounreay and the development of leukaemia and NHL, but were cautious in their interpretation because of multiple statistical testing and, in particular, the possibility of recall bias. Sandside is the only publicly accessible beach on which numbers of radioactive particles have been found, but it was not one of the beaches instanced. As with Sellafield, it was not deemed necessary to specifically include exposure from radioactive particles on beaches in this assessment, although exposures due to general levels of radioactivity on beaches (including particulate material) were considered.

Dounreay site, discharges and environmental monitoring data

5.45 The Dounreay site is located in Caithness near to the coast and is about 12.5 km west of the town of Thurso (see Figure 1.3). Construction of nuclear facilities commenced in 1955. Three nuclear reactors and various nuclear fuel fabrication and reprocessing plants have been in operation at varying times over the period of interest. Table 5.10 summarises some of the key stages in the development of the site up until 2010. Currently, large sections of the site are being decommissioned.

Table 5.10 Dounreay reactor operations and associated processes

Dounreay materials testing reactor (DMTR)	Achieved criticality Shut down	1958 1969
DMTR reprocessing plant	First fuel reprocessed Shut down	1958 1968
Dounreay fast reactor (DFR)	Achieved criticality Achieved full power Shut down	1959 1963 1977
DFR reprocessing plant	Commissioned First reprocessing Last run	1960 1961 1979
Prototype fast reactor (PFR)	Achieved criticality Shut down	1974 1994
PFR reprocessing plant	Commissioned First reprocessing Last run	1979 1980 1996
Fuel cycle area	Fuel fabrication ceased	2004

5.46 Next to the Dounreay site is HMS Vulcan, the Naval Reactor Test Establishment, which also uses radioactive materials that give rise to low levels of discharges. The first reactor at HMS Vulcan operated between 1965 and 1987, while the second reactor operated between 1987 and 1996 and between 2002 and 2015. The liquid effluent from HMS Vulcan is discharged through the Dounreay site and these releases are included in the Dounreay discharges

* The Contaminations Working Group receives updates from the Dounreay monitoring programme and has made recommendations on the monitoring system and schedule. COMARE receives and evaluates a summary on the programme at each meeting.

considered in this study. The aerial discharges were reported separately; for the year 2014, these amounted to only 650 MBq of noble gases and 1 MBq of beta emitting particulates (EA et al, 2015).

5.47 The discharges from the Dounreay site included in NRPB-R195 have been reviewed for consistency between data sources, including published information, and a final set of discharges was compiled for use in the project. The primary sources were Tyler (1998), NRPB-R195 and data provided to COMARE by SEPA (Hill and Cooper, 1986; Tyler, 1998). Additional liquid discharges of cobalt-60, silver-110m and curium-242 from 1989 onwards have been included, which were not considered in the original study. A number of aerial discharges reported from the early 1980s of radionuclides that do not significantly contribute to doses have been included in the study for completeness.

5.48 Liquid discharges started from the Dounreay site in 1958; Figure 5.14 shows the annual discharges to sea of the key alpha emitting radionuclides (plutonium-239/240 and americium-241) as a function of time from 1958–2010. The annual marine discharges of four key beta emitting radionuclides (strontium-90, ruthenium-106, caesium-137 and plutonium-241) are given in Figure 5.15 for the same period. Discharges of iodine-131 to sea are reported until 1979, with only sporadic releases identified from the early 1970s. The annual discharges to atmosphere of plutonium-239/240 and americium-241 from 1958–2010 are given in Figure 5.16. Figure 5.17 shows the annual discharges to atmosphere of the main beta emitters: strontium-90, zirconium-95/niobium-95, ruthenium-106 and caesium-137. The discharges are very variable, with distinct peaks at various times for both liquid and aerial discharges reflecting the various operations at the site. Gaseous discharges of iodine-131 started in 1964 and dropped to low levels in the early 1980s. Low gaseous discharges of iodine-129 have been reported since 1986 and these have been included in the assessment of doses.

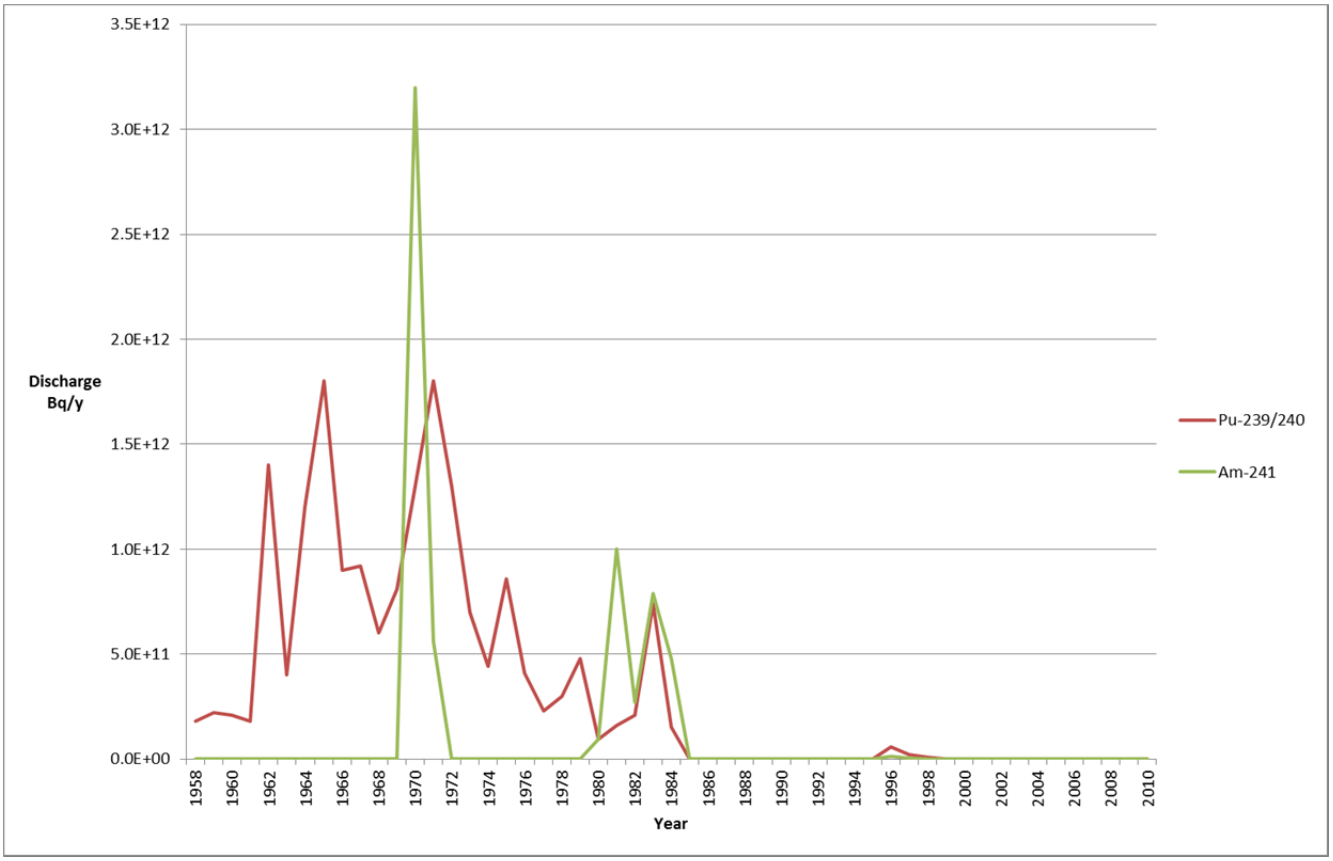

Figure 5.14 Annual liquid discharges from the Dounreay site – key alpha emitters

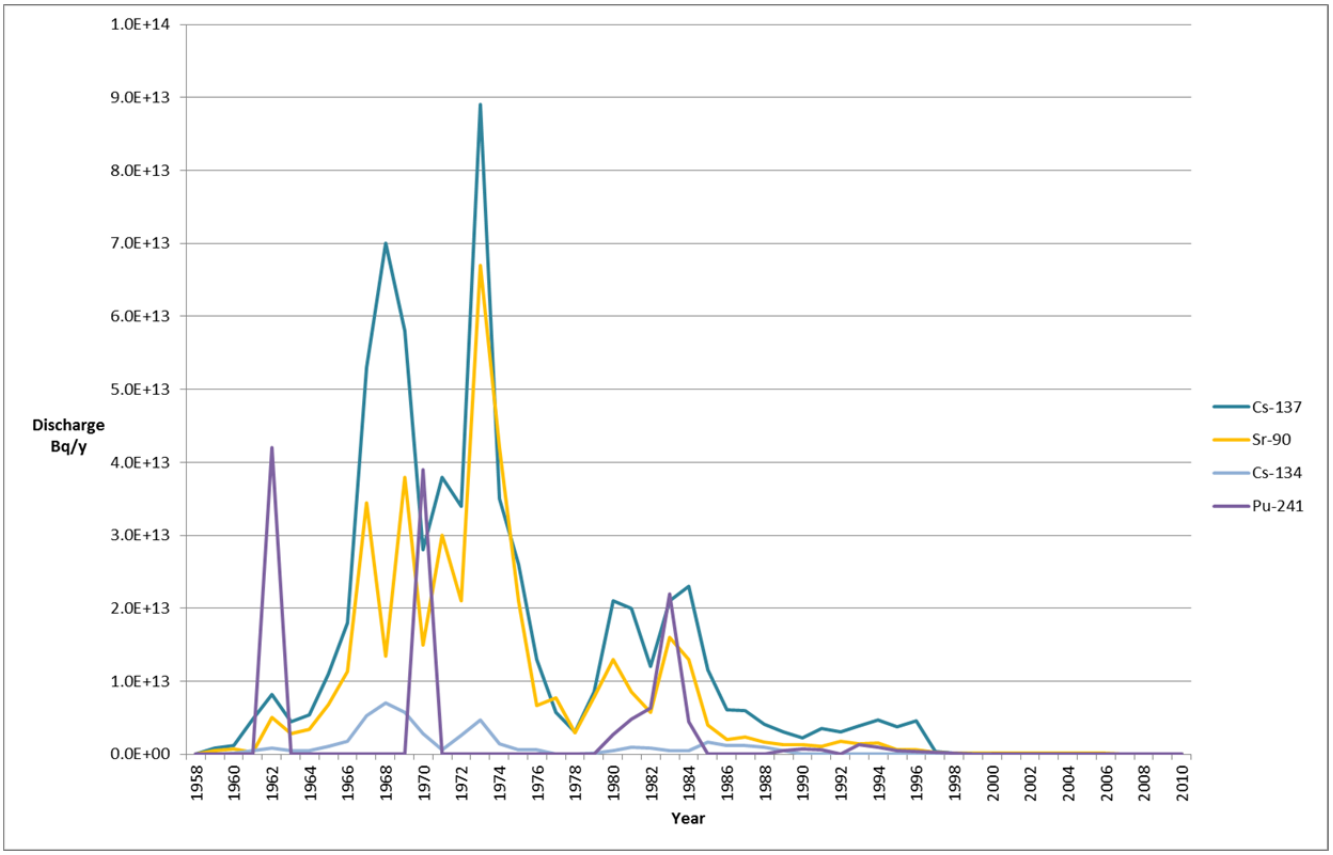

Figure 5.15 Annual liquid discharges from the Dounreay site – key beta emitters

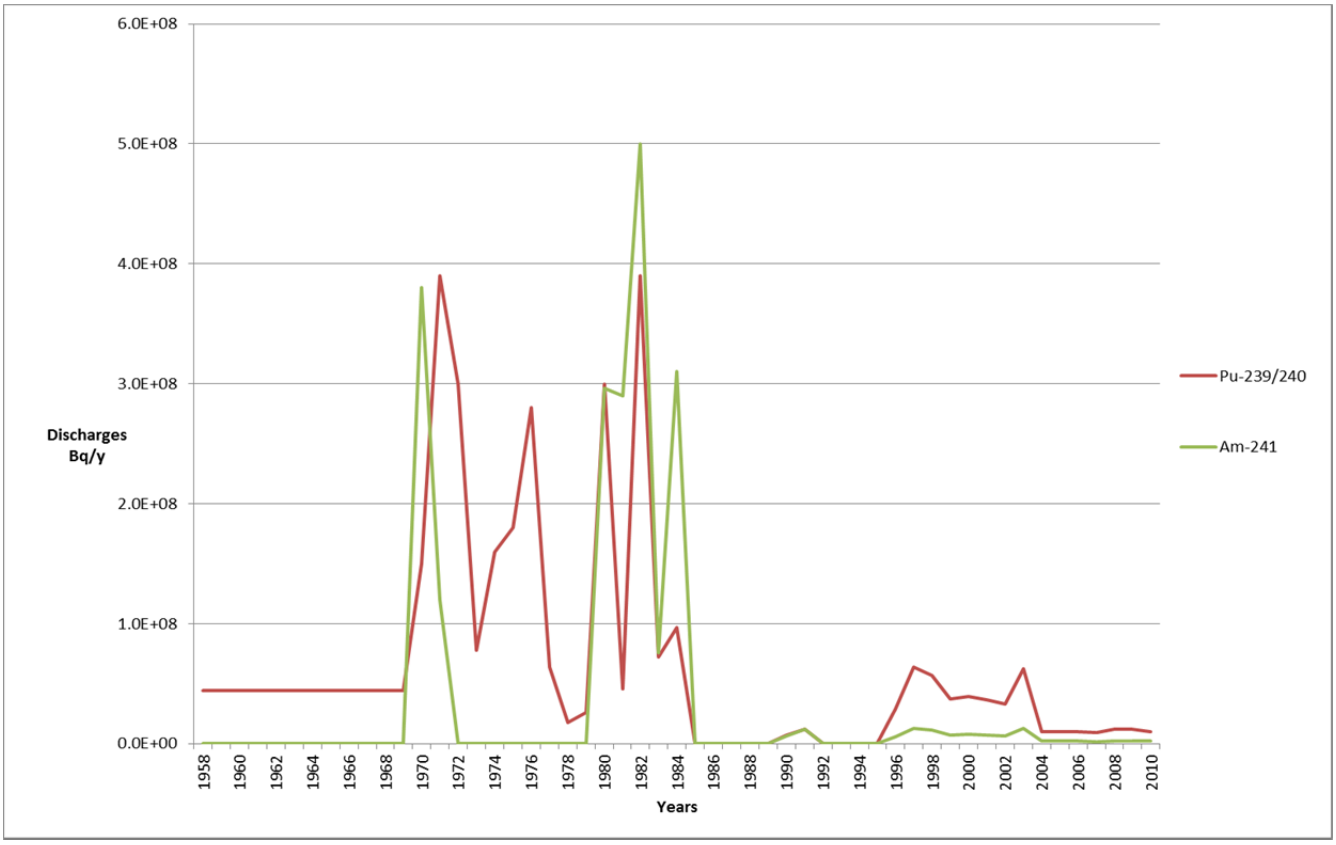

Figure 5.16 Annual aerial discharges from the Dounreay site – key alpha emitters

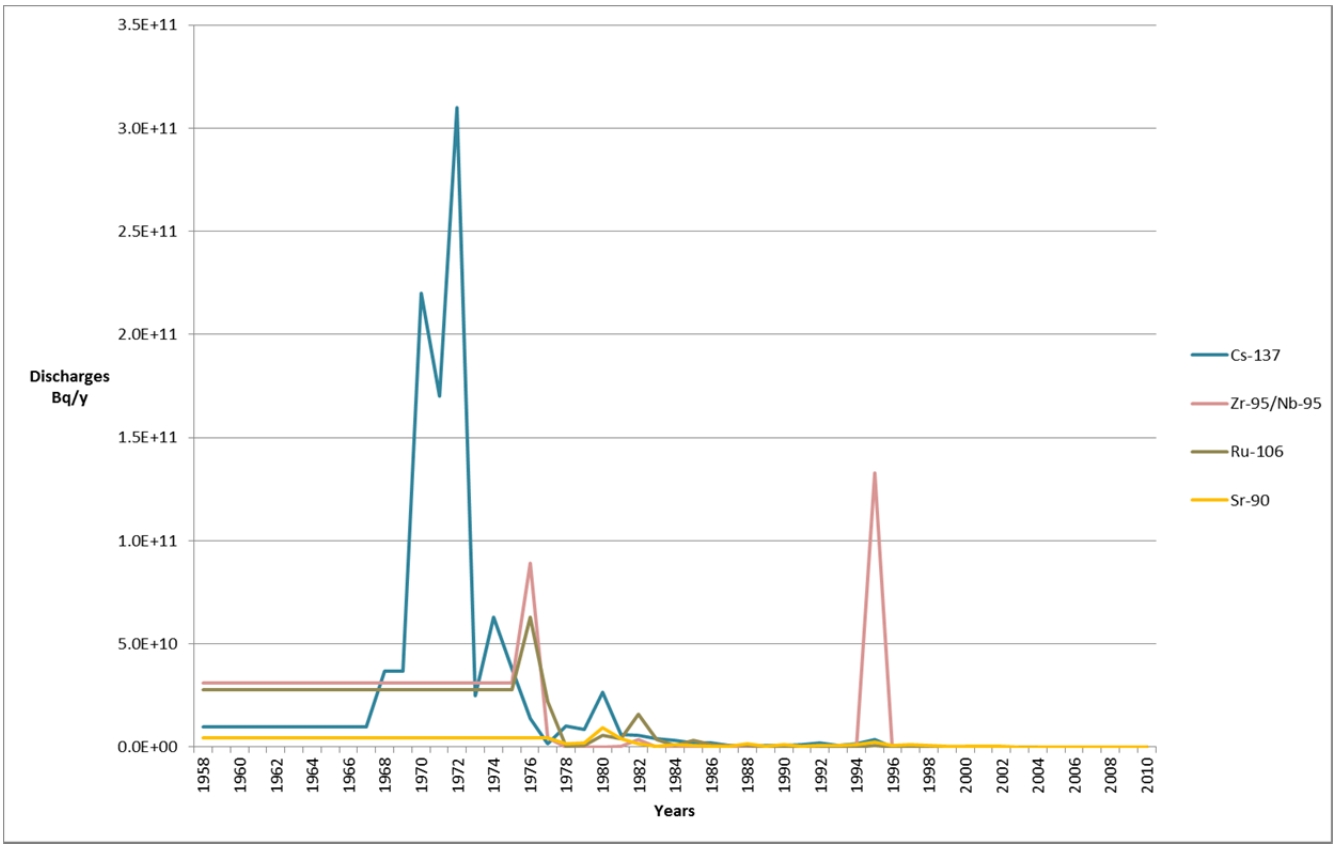

Figure 5.17 Annual aerial discharges from the Dounreay site – key beta emitters

5.49 Liquid discharges from the Sellafield site are transported round the coast of Great Britain from the Irish Sea to the North Sea and beyond. A proportion of the radionuclides in the sea and marine biota around the Dounreay coast therefore come from the liquid waste discharged from Sellafield. Doses from these discharges have been considered separately in the Thurso dose assessment.

5.50 The original study of the risk of leukaemia and other cancers in Thurso made use of discharge data and mathematical models to calculate intakes of radionuclides by inhalation, ingestion and external doses as the limited environmental monitoring data did not provide an adequate basis on which to assess the doses. This remains the case and the same approach has been used here. Current PHE models have been used (PC CREAM-08) in the assessment, while the marine models, in particular, have changed since publication of NRPB-R195. In addition, the doses arising from liquid discharges from Sellafield have been calculated using models. Where possible, measurements have been compared with model predictions to determine the validity of the approaches adopted.

Radiation exposures due to Dounreay discharges

5.51 Average RBM doses to 1 and 10 year old children living in Thurso were estimated as a function of time to illustrate how radiation exposures would have changed over the period 1958–2010 covered by this study. The doses estimated for each year are the sum of the external dose in the year and the doses from intakes by inhalation and ingestion in the year resulting from the discharges from Dounreay. The doses from intakes were the RBM dose commitment to the 25th birthday. Current ICRP dose coefficients have been used (ICRP, 2012). Figure 5.18 shows the estimated RBM doses for 1 year old infants from 1958–2010, while Figure 5.19 shows the same results for 10 year old children. In both cases the total doses are given together with the doses from external irradiation, ingestion and inhalation.

5.52 For both 1 and 10 year old children the highest estimated RBM doses were received in the mid-1960s to the early 1970s, with the greatest proportion of the estimated dose due to external irradiation arising from liquid discharges to sea. The estimated doses decreased significantly after the early 1970s and, although there are minor fluctuations, they have remained at about the same level from the late 1980s until 2010.

5.53 The inhalation doses are very low and are mainly due to aerial discharges of plutonium-239/240. For 1 year old infants, ingestion doses are dominated by ingestion of terrestrial foods, whereas for 10 year old children, who eat more seafood, doses are a little higher from the mid-1960s until the mid-1970s, reflecting the increase in liquid discharges over this period.

5.54 Doses to the thyroid are higher than those for the RBM in the 1960s and early 1970s by about a factor of 30 for 1 year olds and a factor of 3 for 10 year olds and then rapidly decrease to very low levels, as is the case for the RBM doses. For 1 year olds, the thyroid doses are dominated by ingestion, as shown in Figure 5.20; this is in contrast to what is observed for RBM doses. For 10 year olds, both external doses and ingestion contribute to the overall thyroid doses, with external doses dominating in most years, as shown in Figure 5.21.

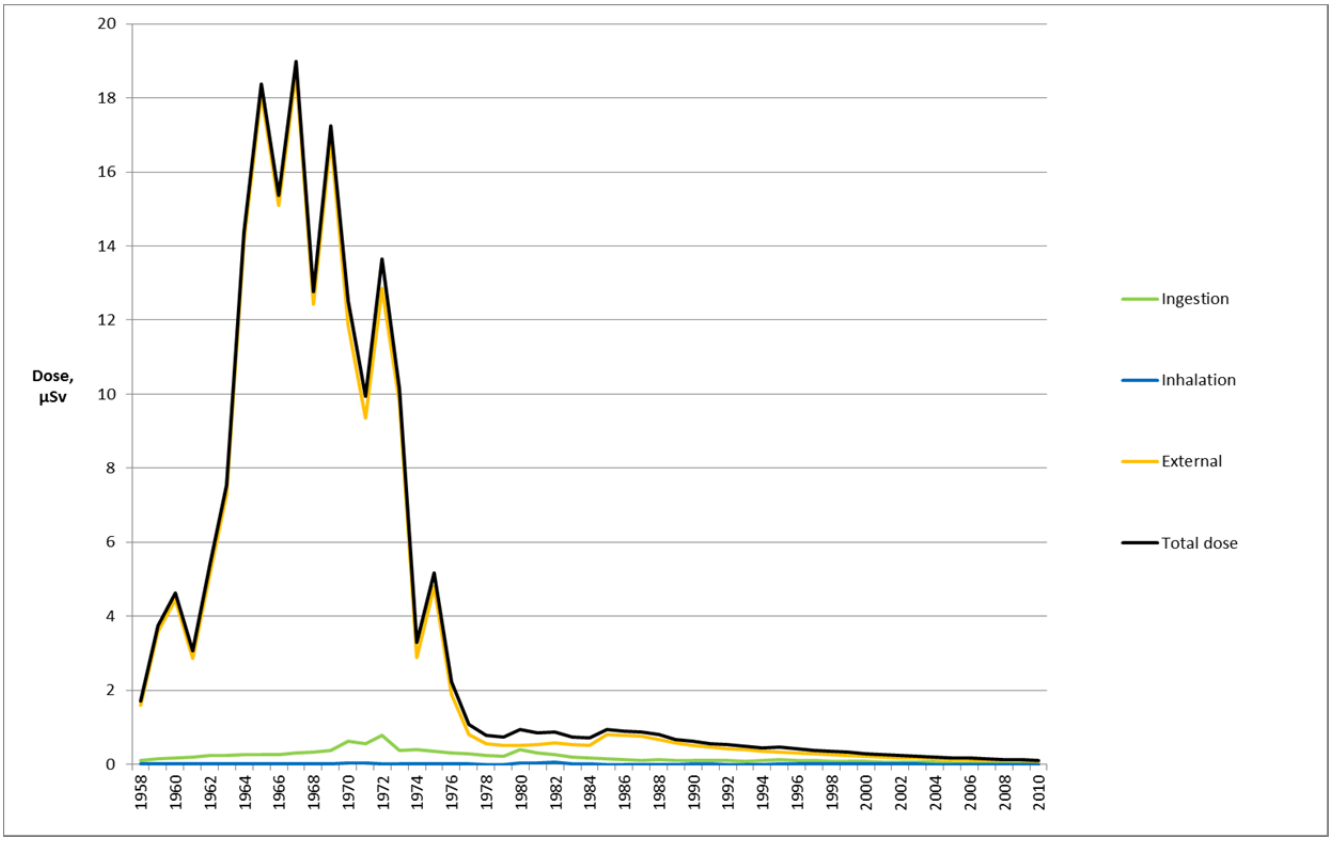

Figure 5.18 Cumulative red bone marrow dose to a 1 year old in Thurso from Dounreay discharges, with the dose from intakes of radionuclides integrated to (and including) 24 years of age

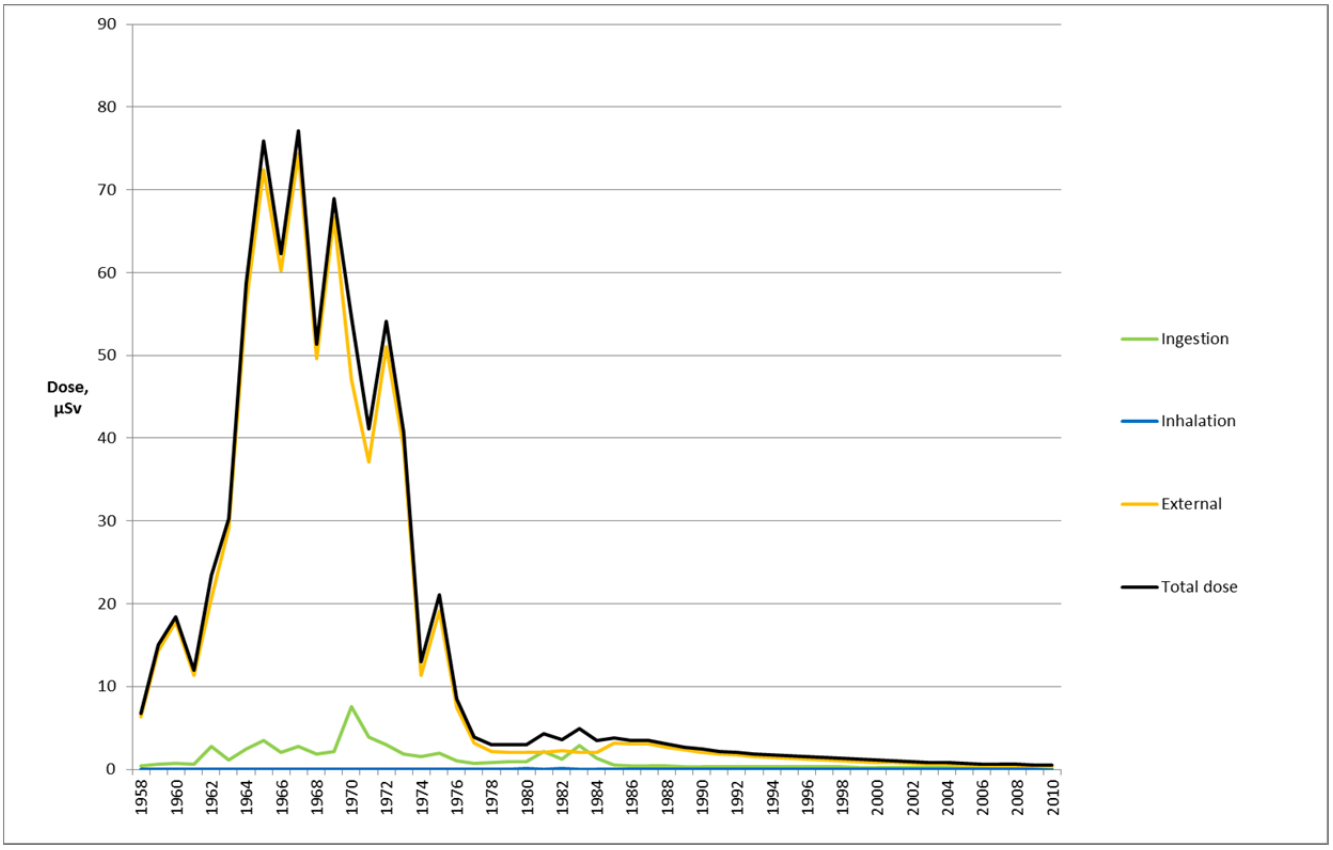

Figure 5.19 Cumulative red bone marrow dose to a 10 year old in Thurso from Dounreay discharges, with the dose from intakes of radionuclides integrated to (and including) 24 years of age

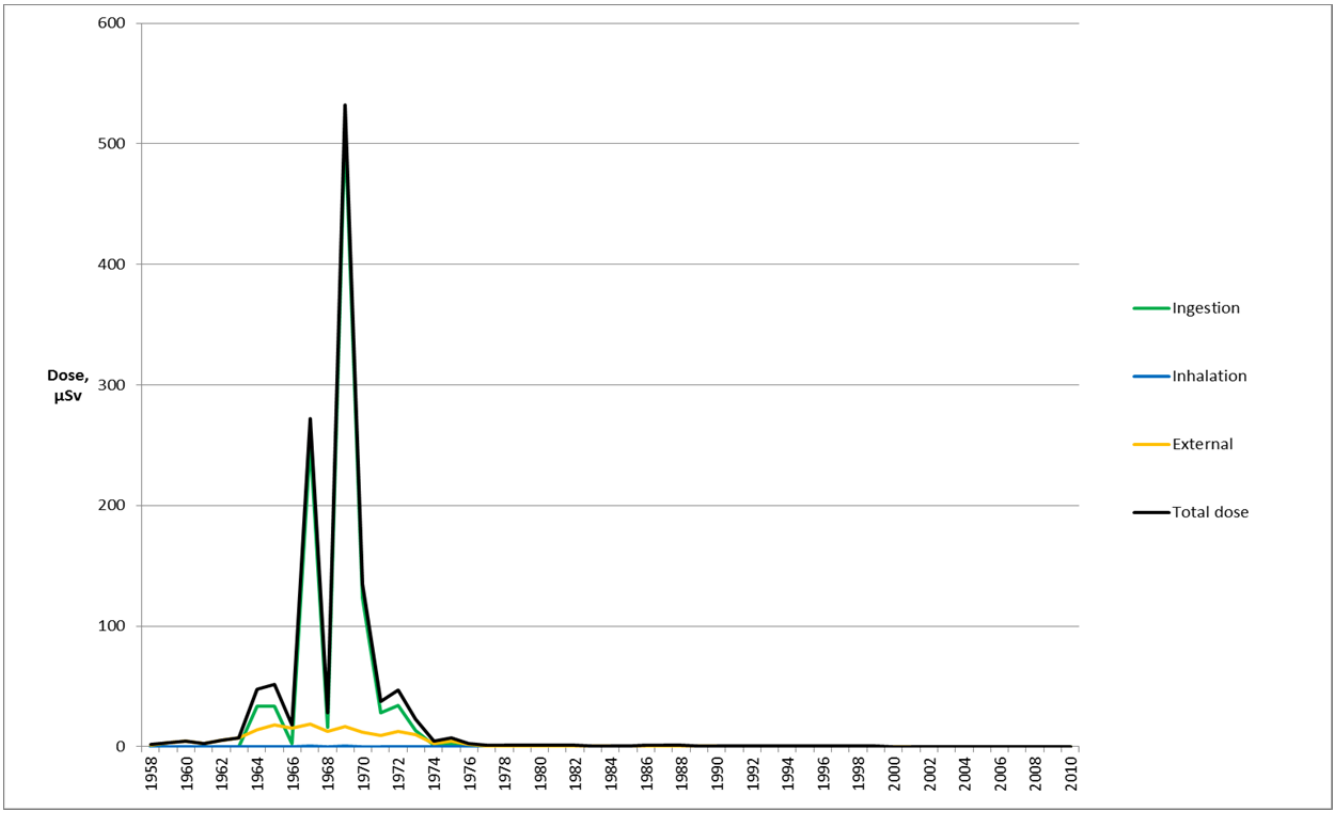

Figure 5.20 Thyroid dose to a 1 year old child in Thurso from routine Dounreay discharges

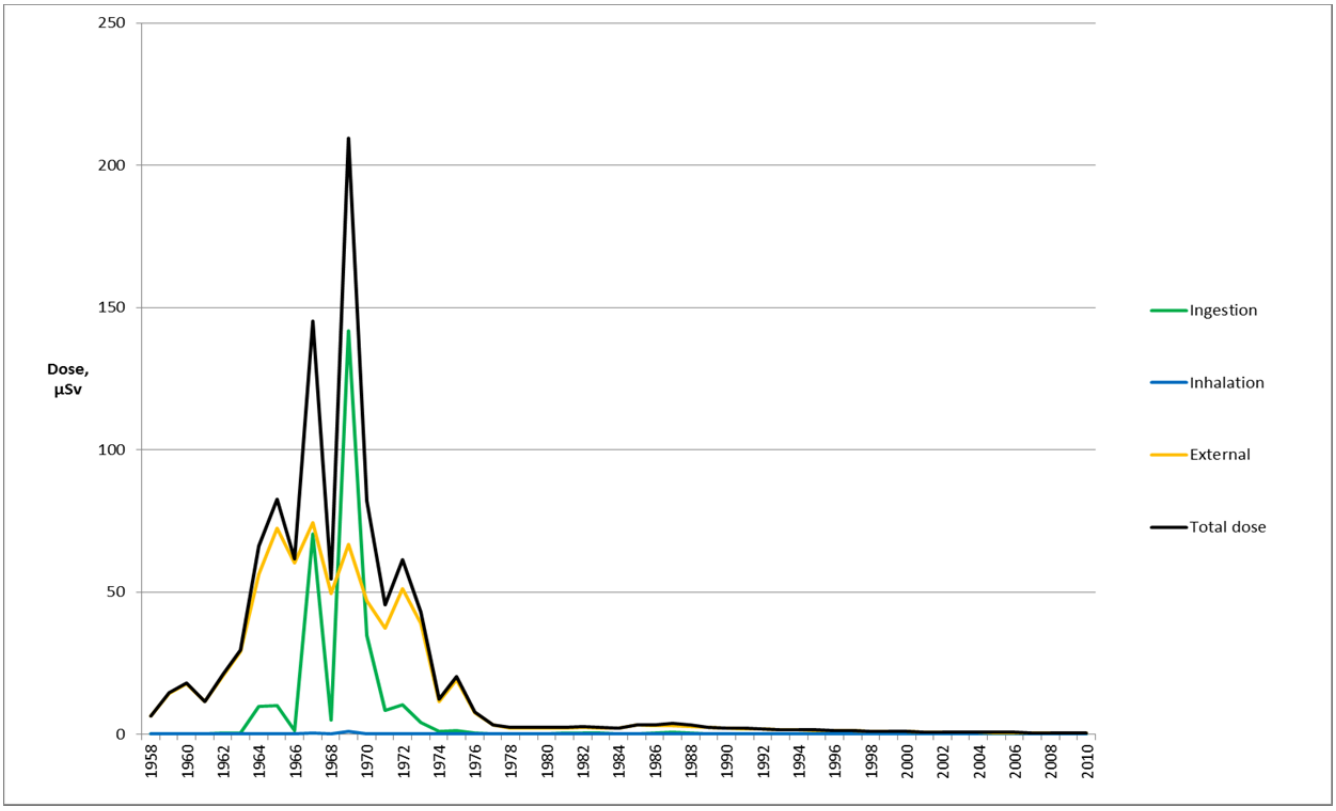

Figure 5.21 Thyroid dose to a 10 year old child in Thurso from routine Dounreay discharges

Radiation doses at Thurso due to Sellafield liquid discharges

5.55 Figure 5.22 shows the estimated annual RBM doses to 1 year old infants from 1958–2010 arising from liquid discharges from Dounreay and Sellafield, while Figure 5.23 shows the corresponding results for 10 year old children. In both cases doses are given for external irradiation and ingestion of seafood. From the mid-1970s until the mid-1980s the RBM doses to children living at Thurso were mainly due to liquid discharges to sea from Sellafield with similar contributions subsequently from the two sites until the end of the 1990s. Doses from external irradiation were due mainly to beach occupancy. Over the last 10 years as discharges from Dounreay have continued to decrease, the contribution from Sellafield has continued at a fairly constant level, with external irradiation from beach occupancy being the major contributor to the doses from liquid discharges from both sites. Doses from ingestion of seafood for 1 year olds in Thurso are higher from Sellafield discharges than those from Dounreay discharges. For 10 year olds who consume shellfish, the contribution from Dounreay discharges is more important up to the mid-1970s, with similar contributions from the two sources over subsequent years. The thyroid doses from marine pathways are very similar to those for the RBM, with contributions dominated by external doses from Dounreay discharges over the period when the doses were highest between the start of operation and the mid-1970s.

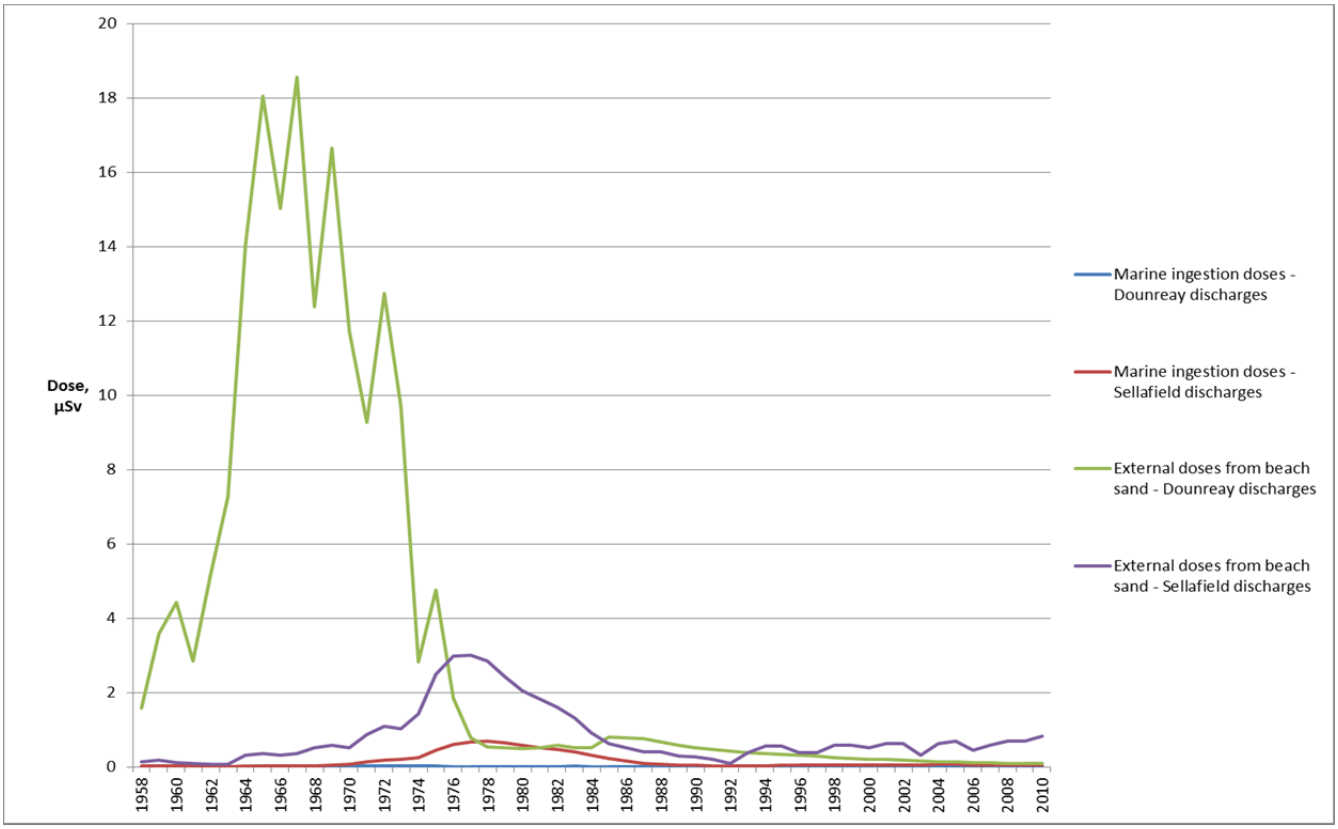

Figure 5.22 Cumulative red bone marrow dose to a 1 year old in Thurso from Dounreay and Sellafield liquid discharges, with the dose from intakes of radionuclides integrated to (and including) 24 years of age

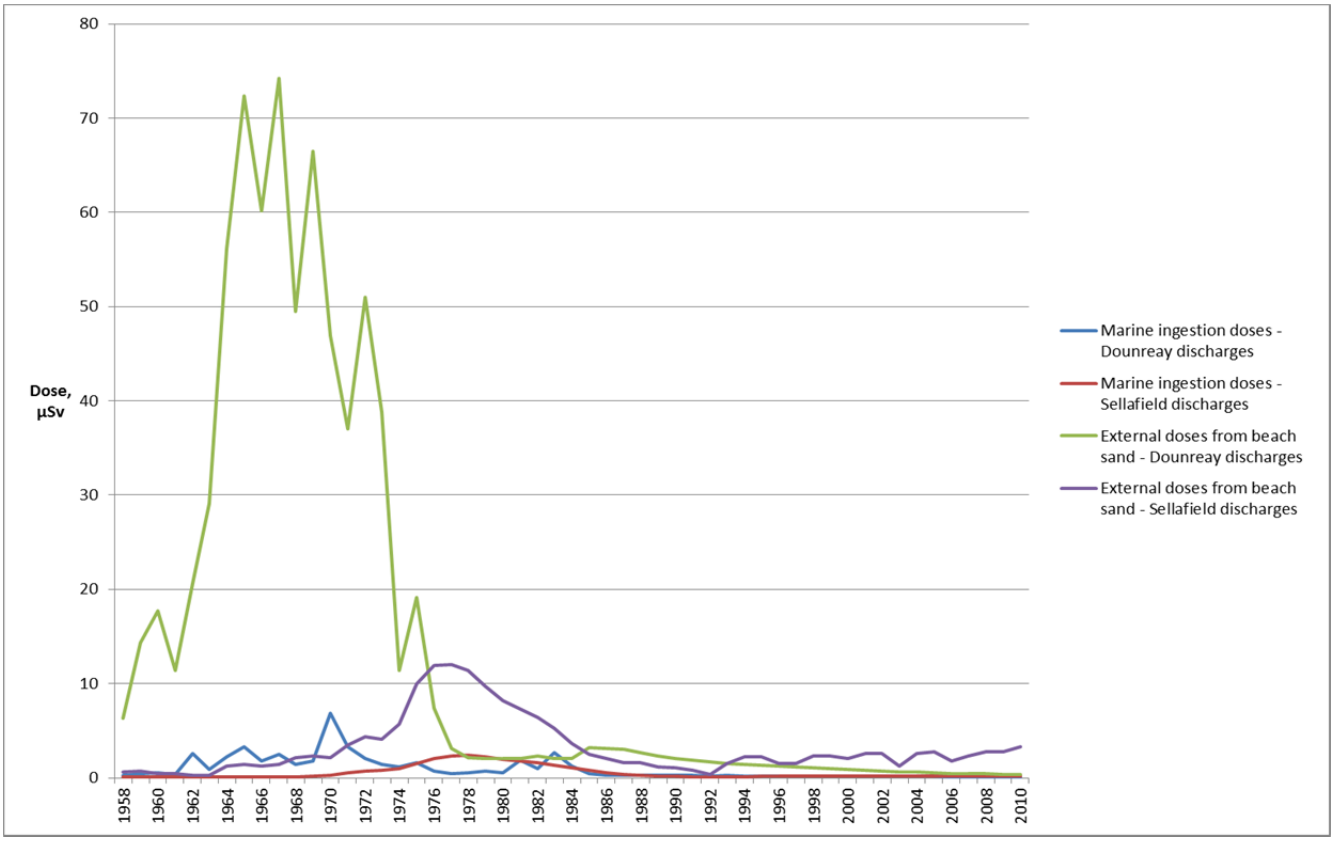

Figure 5.23 Cumulative red bone marrow dose to a 10 year old in Thurso from Dounreay and Sellafield liquid discharges, with the dose from intakes of radionuclides integrated to (and including) 24 years of age

5.56 The total RBM doses for 1 and 10 year old children from discharges at Dounreay and Sellafield over the period 1958–2010 are given in Figures 5.24 and 5.25, while Figures 5.26 and 5.27 show the same results for thyroid doses.

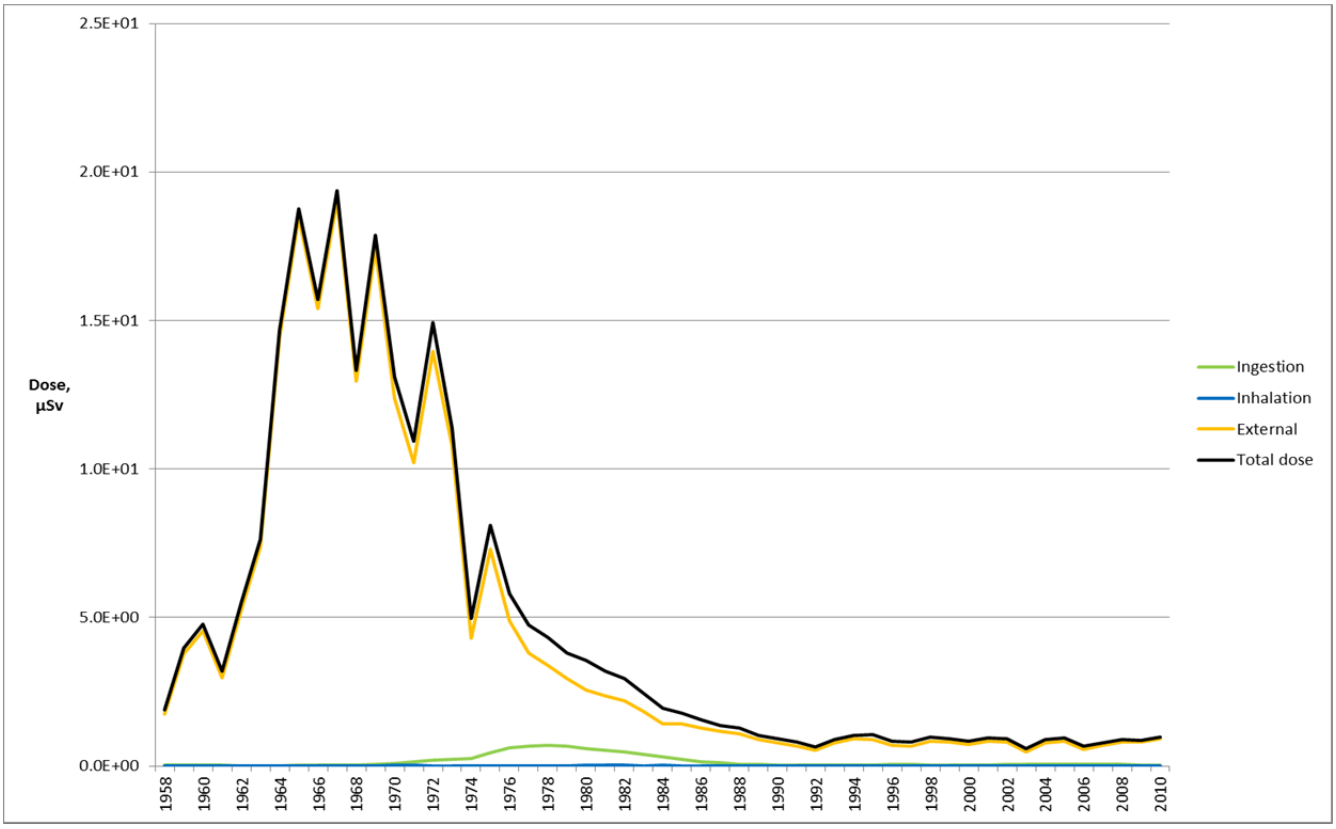

Figure 5.24 Cumulative red bone marrow dose to a 1 year old in Thurso from both Dounreay and Sellafield discharges, with the dose from intakes of radionuclides integrated to (and including) 24 years of age

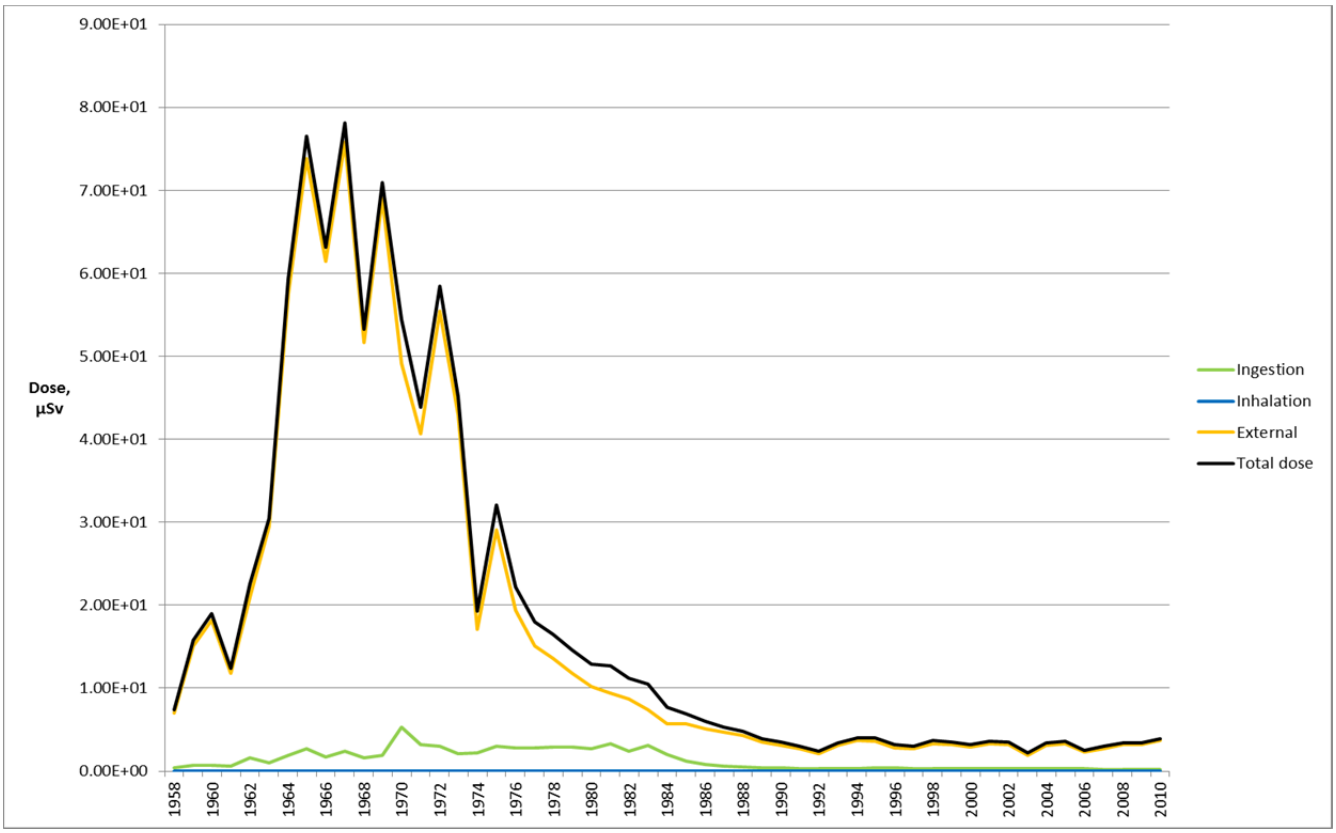

Figure 5.25 Cumulative red bone marrow dose to a 10 year old in Thurso from both Dounreay and Sellafield discharges, with the dose from intakes of radionuclides integrated to (and including) 24 years of age

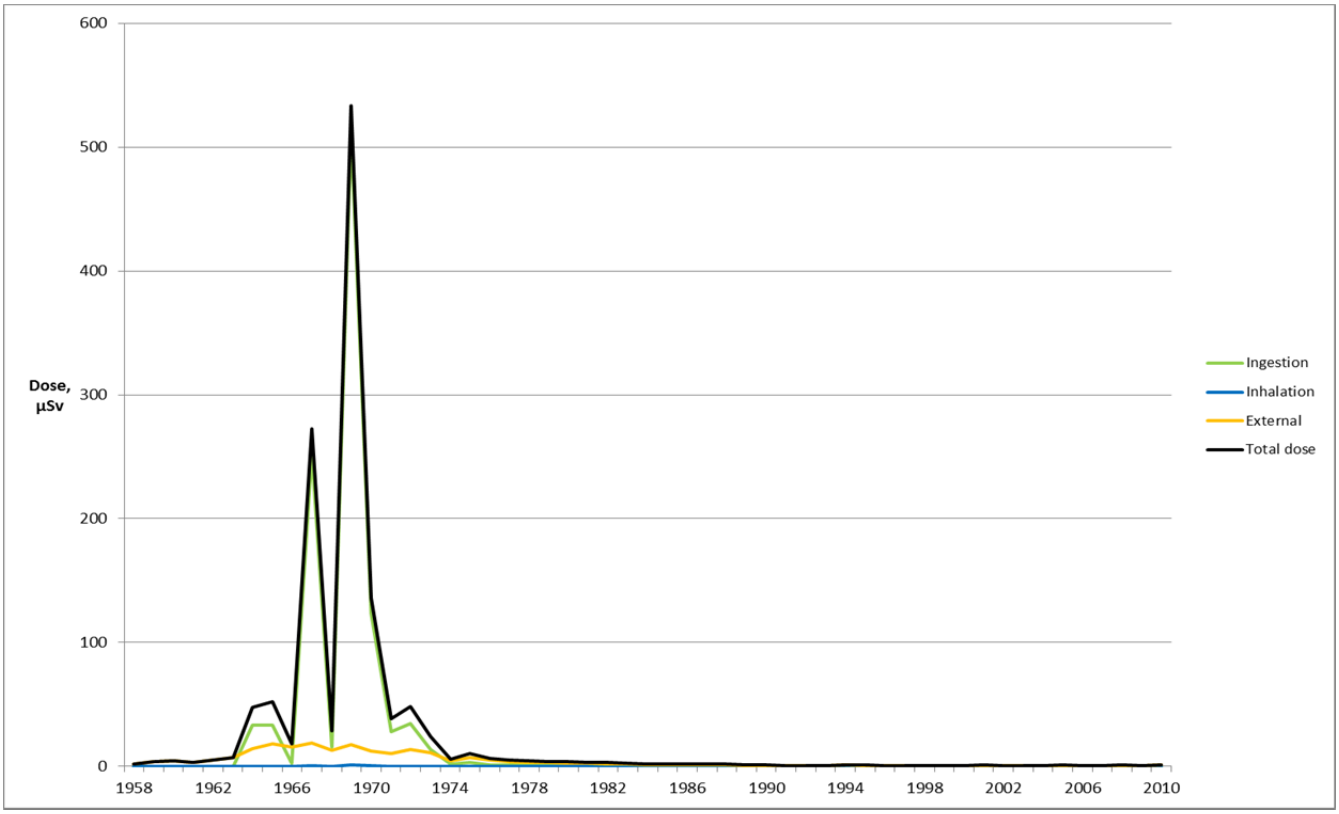

Figure 5.26 Thyroid dose to a 1 year old child in Thurso from Dounreay and Sellafield discharges

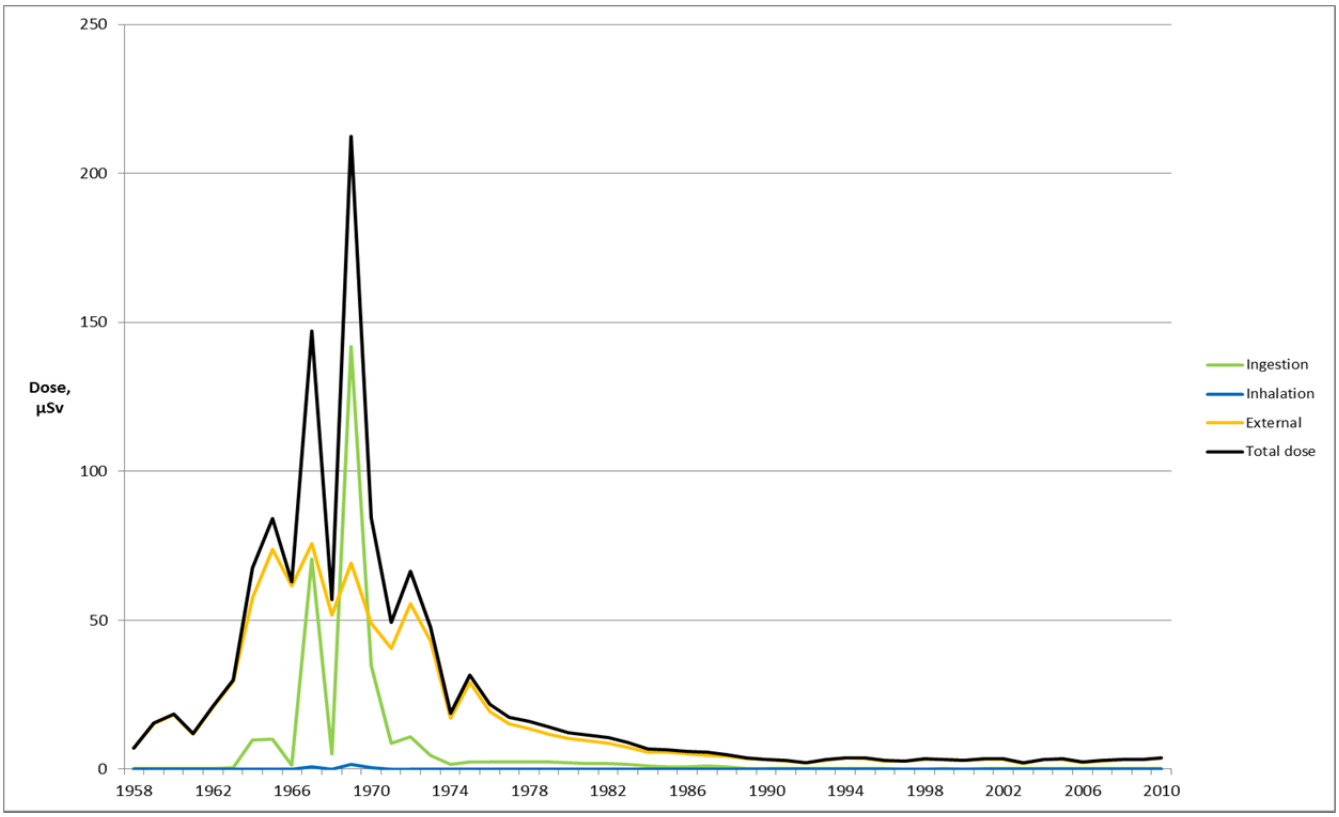

Figure 5.27 Thyroid dose to a 10 year old child in Thurso from Dounreay and Sellafield discharges

Radiation doses from all routes of exposure

5.57 As for Seascale, the main source of radiation exposure in Thurso throughout the period 1958–2010 was that due to natural background radiation. Doses from fallout from the atmospheric testing of nuclear weapons were similar to those for children living in Seascale. The accident at the Chernobyl nuclear power plant in 1986 led to a slight increase in radiation doses in the UK and, due to the timing of the work, this source was not taken into account in the analyses carried out for the second COMARE report. These doses have decreased with time and are currently very low. In 1986, when the accident occurred, estimated RBM doses to 1 and 10 year old children in Thurso were about 0.1 mSv (Morrey et al, 1987). The doses have steadily decreased and are estimated to be less than 0.01 mSv in 2010. The temporal pattern for thyroid doses is similar to that observed for RBM doses, with higher doses in 1986 due to the ingestion of iodine-131 in milk.

5.58 The relative contributions of the different sources of exposure are illustrated in Figures 5.28a and 5.29a, which show the percentage contribution to the total RBM dose from different routes of exposure for a range of years, while Figures 5.28b and 5.29b show the absolute doses in particular years. At all times, natural background is the major source of exposure and from the late 1980s until 2010 it contributes around 90% of the total dose. The contribution from operations at Dounreay and Sellafield is always <1% and is much lower than that estimated from planned and unplanned discharges from Sellafield for children living in Seascale, particularly in the mid-1950s when the relative contribution from Sellafield discharges to doses to children living in Seascale were at their highest. The pattern is similar for thyroid doses in Thurso (Figures 5.30 and 5.31), except in 1986 when there was a relatively large contribution from doses arising from the Chernobyl accident. In 1986, as seen for Seascale, the contributions from Chernobyl were about 50% and 25% of the total doses for 1 and 10 year olds, respectively. Figures 5.30b and 5.31b show the absolute thyroid doses in particular years.

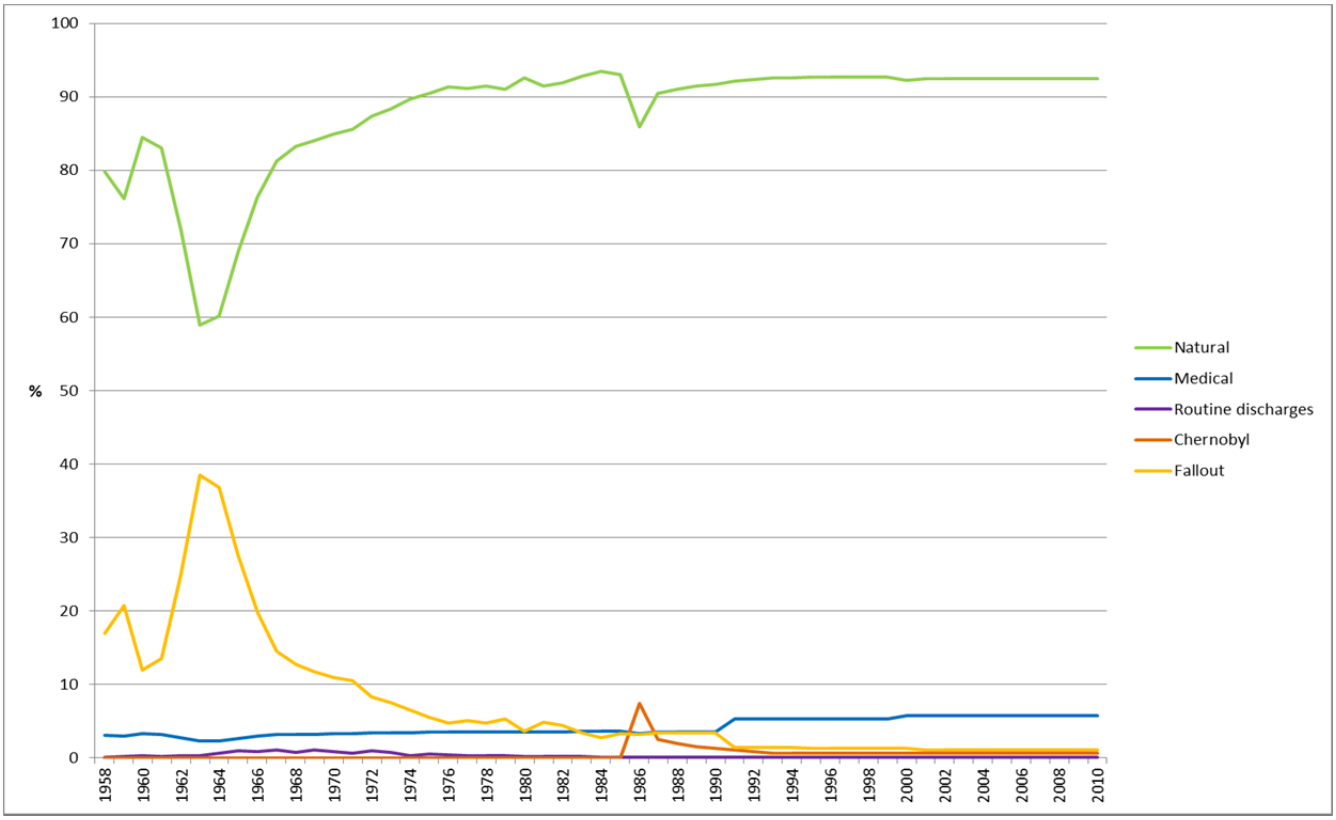

Figure 5.28a Percentage contributions from different sources to the annual red bone marrow doses to 1 year old children in Thurso from 1958 to 2010 resulting from exposure to radionuclides at age 1 year in the given years, integrated to (and including) 24 years of age

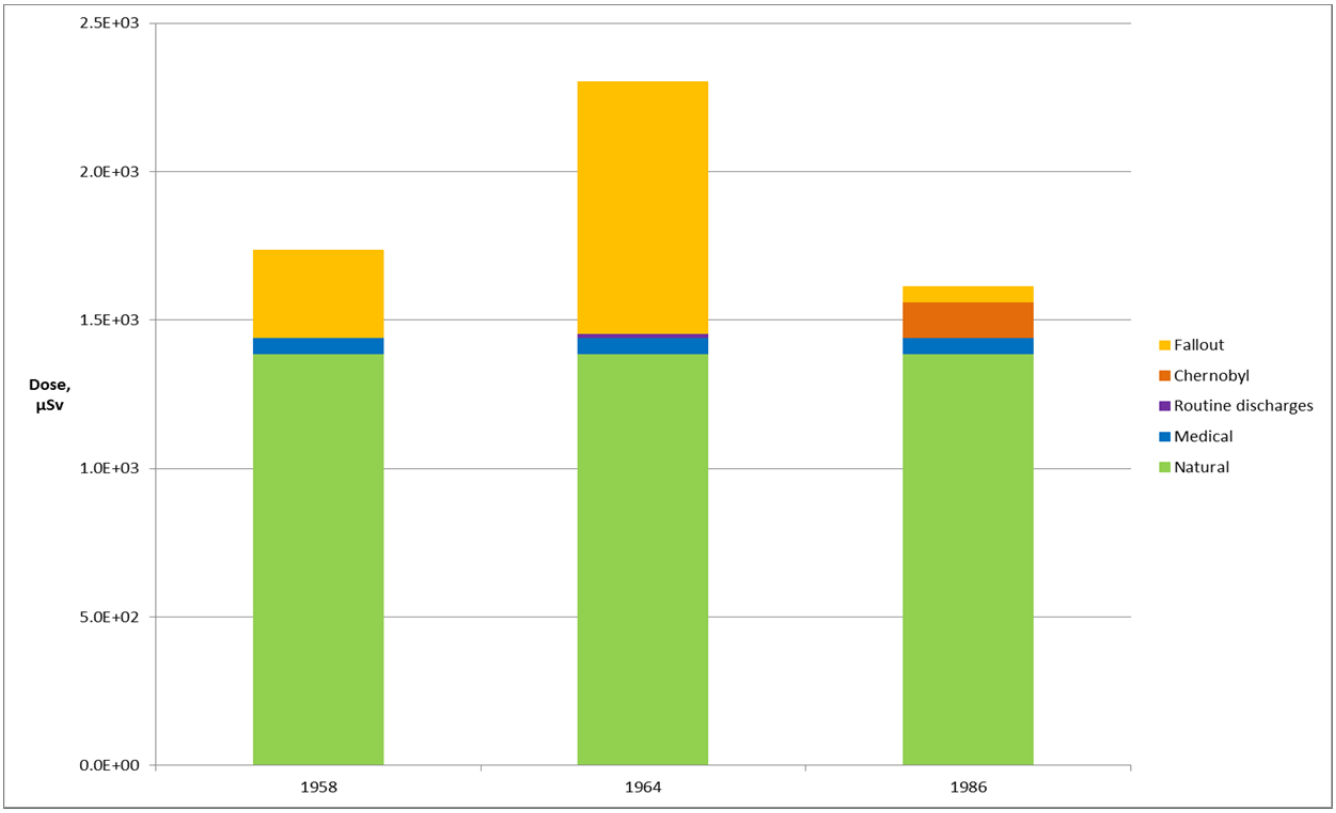

Figure 5.28b Absolute values of red bone marrow dose resulting from exposure to radionuclides in Thurso at age 1 year in the given years, integrated to (and including) 24 years of age

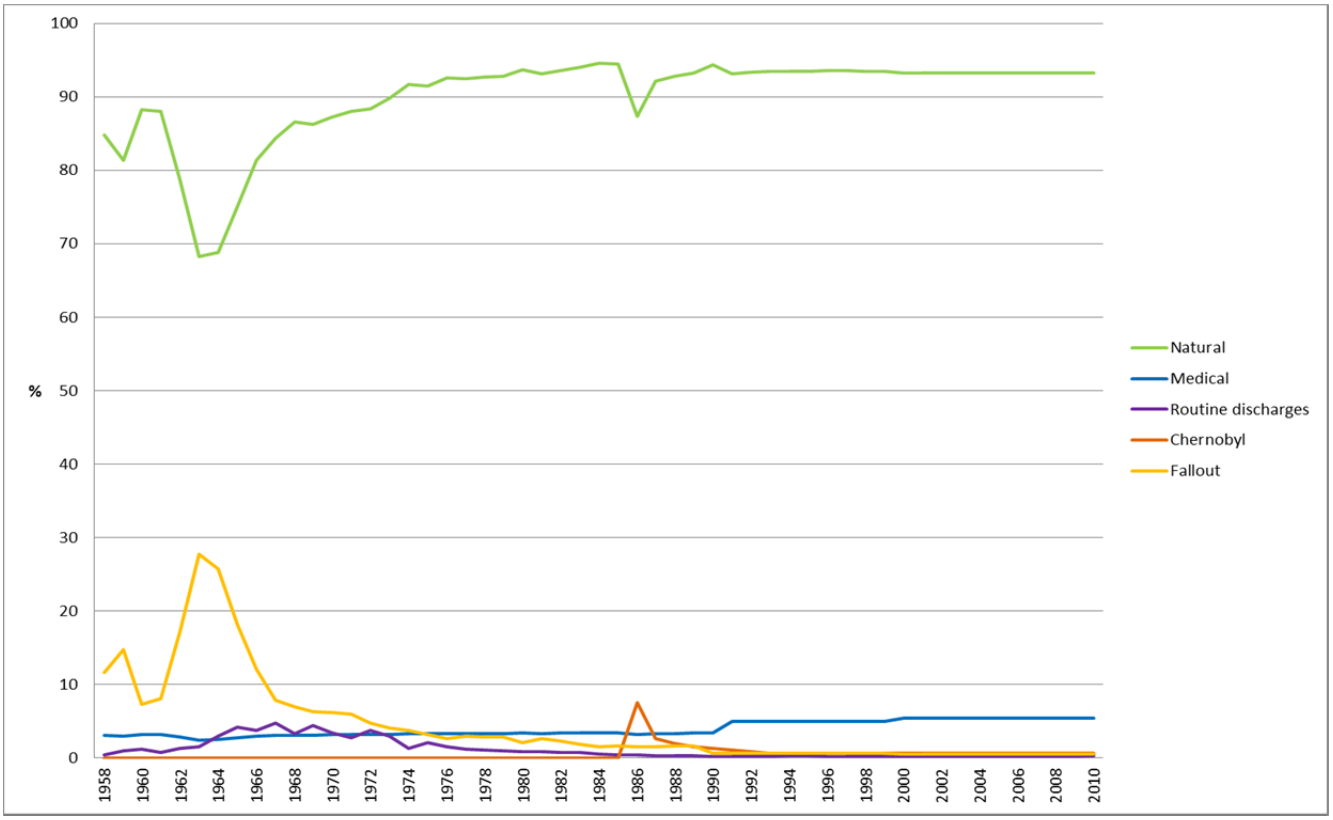

Figure 5.29a Percentage contributions from different sources to the annual red bone marrow doses to 10 year old children in Thurso from 1958 to 2010 resulting from exposure to radionuclides at age 10 years in the given years, integrated to (and including) 24 years of age

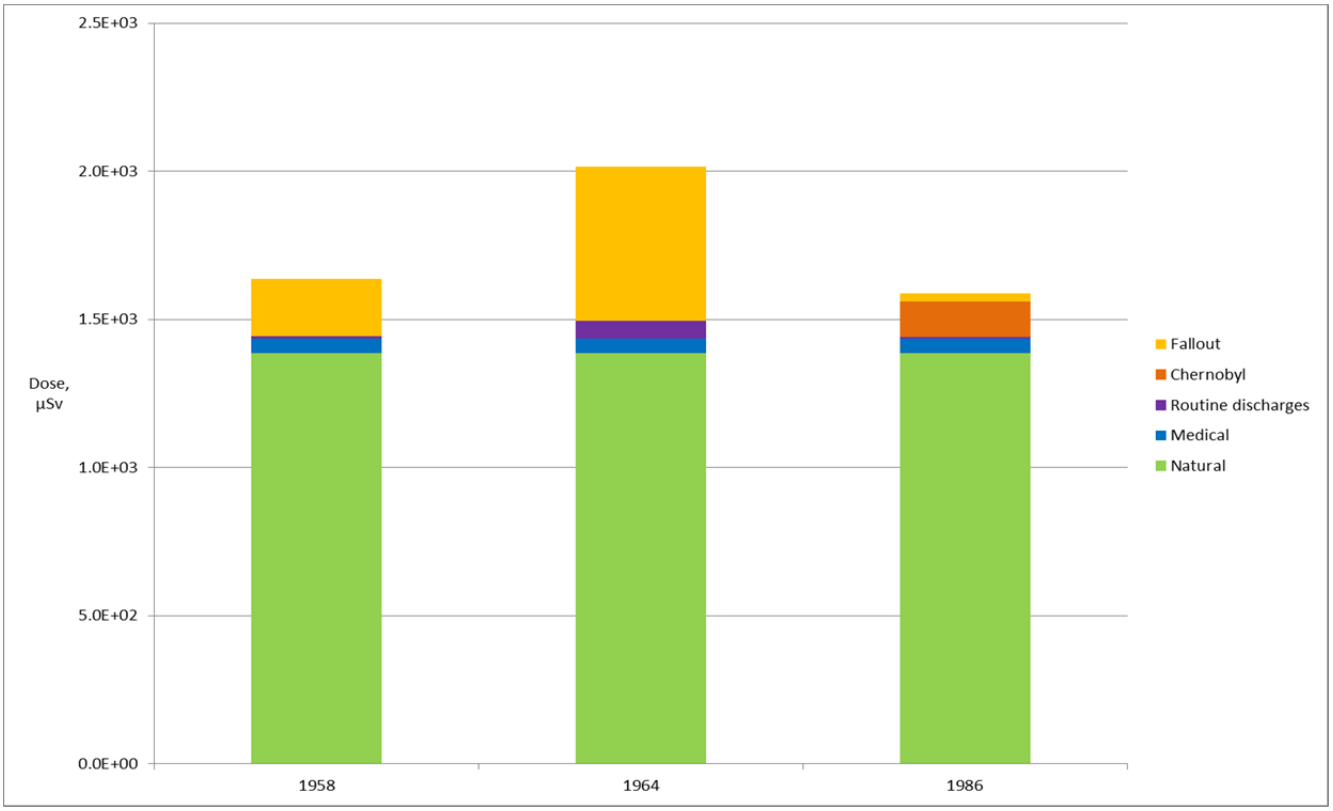

Figure 5.29b Absolute values of red bone marrow dose resulting from exposure to radionuclides in Thurso at age 10 years in the given years, integrated to (and including) 24 years of age

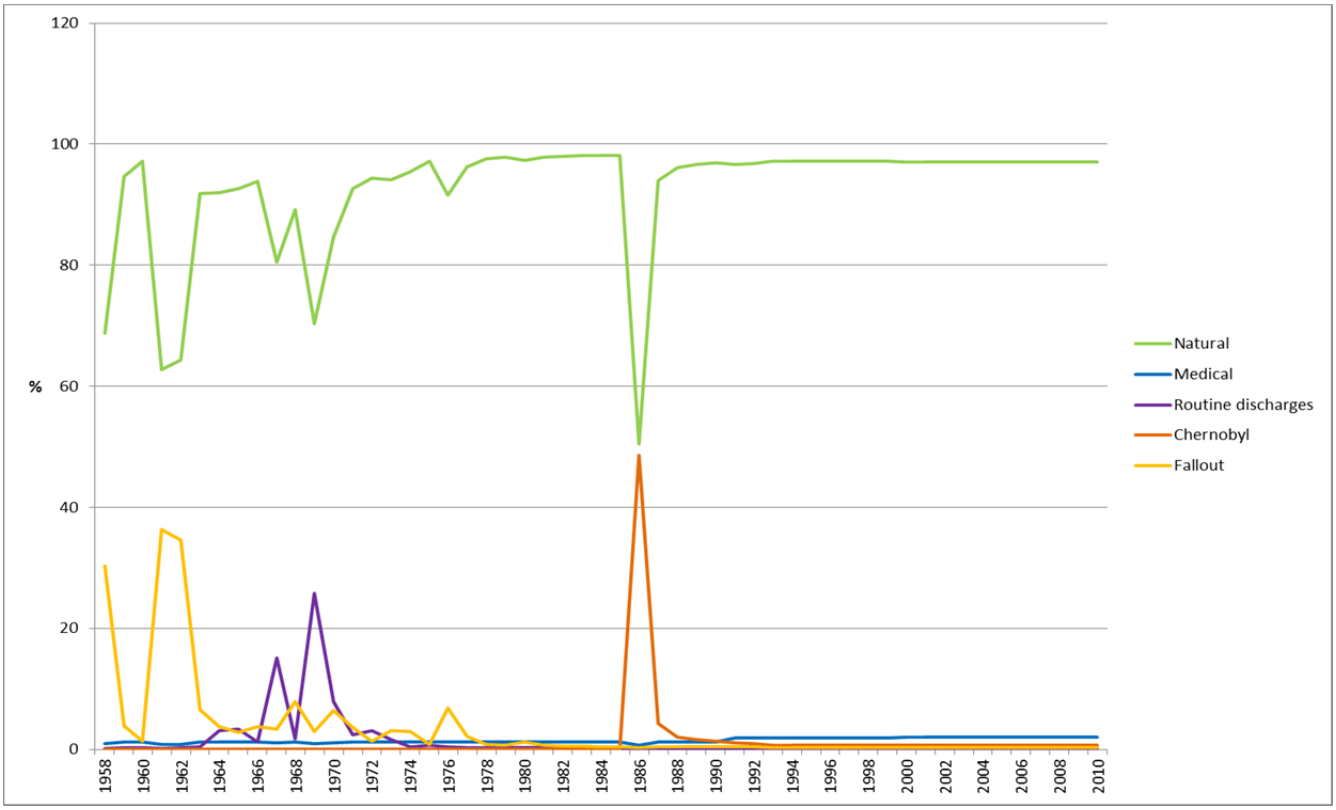

Figure 5.30a Percentage contributions from different sources to the annual thyroid doses to 1 year old children in Thurso from 1958 to 2010 resulting from exposure to radionuclides at age 1 year in the given years, integrated to (and including) 24 years of age

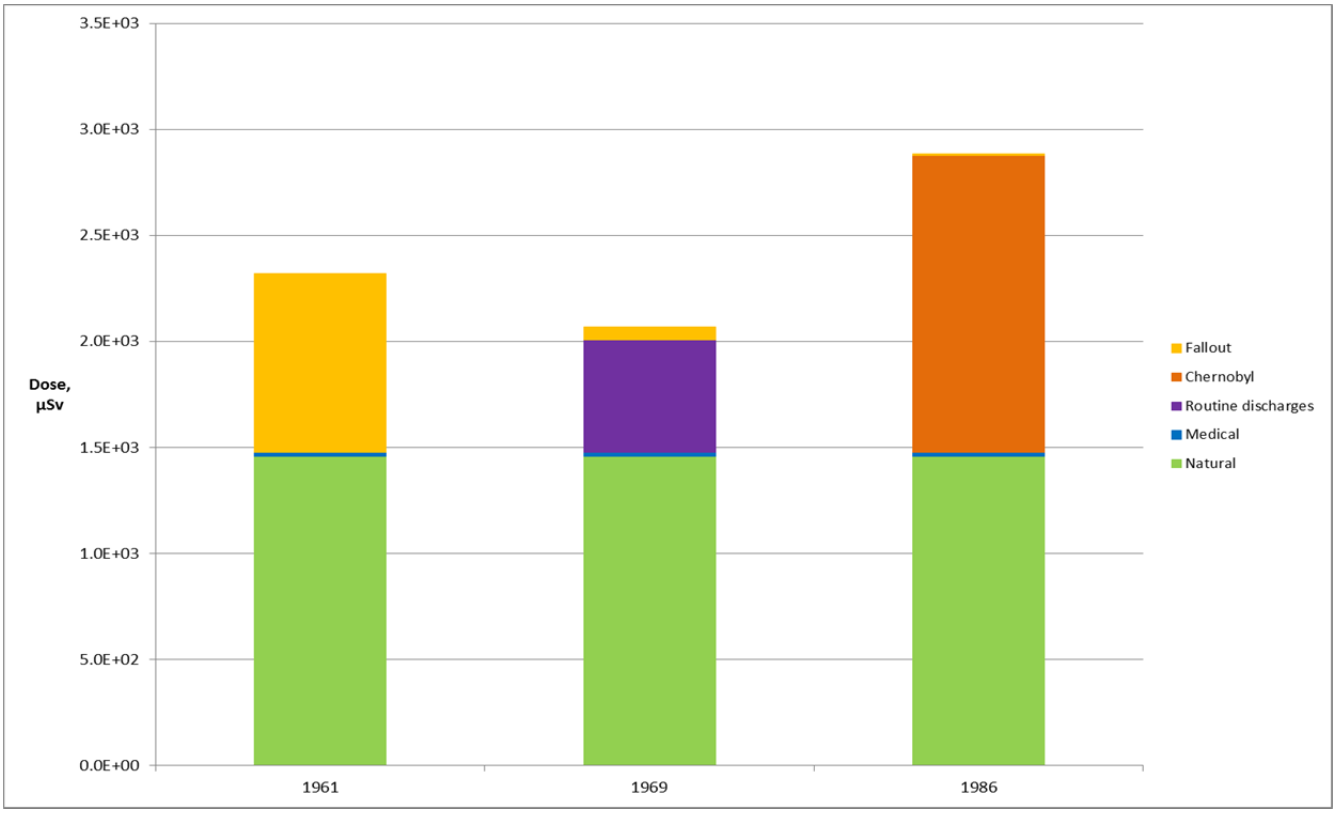

Figure 5.30b Absolute values of thyroid dose resulting from exposure to radionuclides in Thurso at age 1 year in the given years, integrated to (and including) 24 years of age

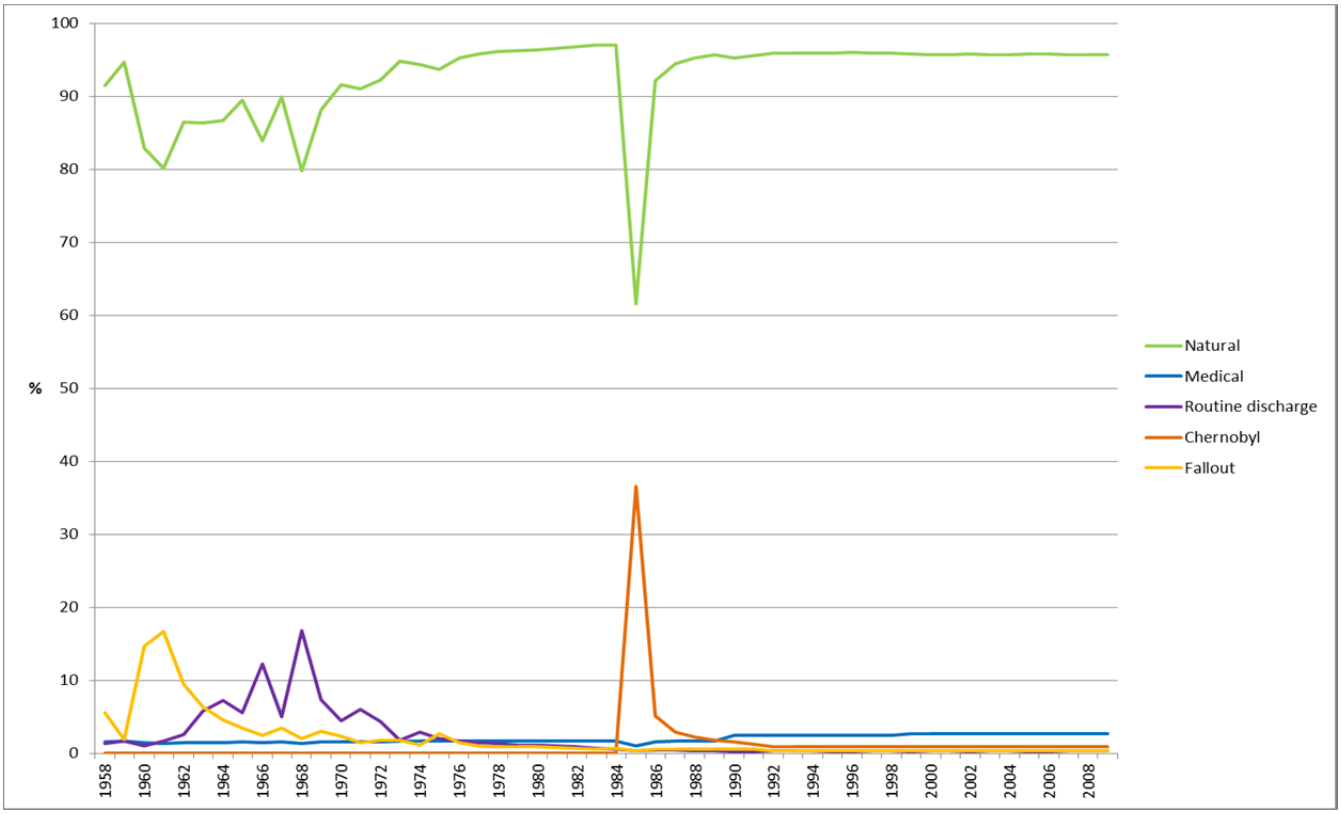

Figure 5.31a Percentage contributions from different sources to the annual thyroid doses to 10 year old children in Thurso from 1958 to 2010 resulting from exposure to radionuclides at age 10 years in the given years, integrated to (and including) 24 years of age

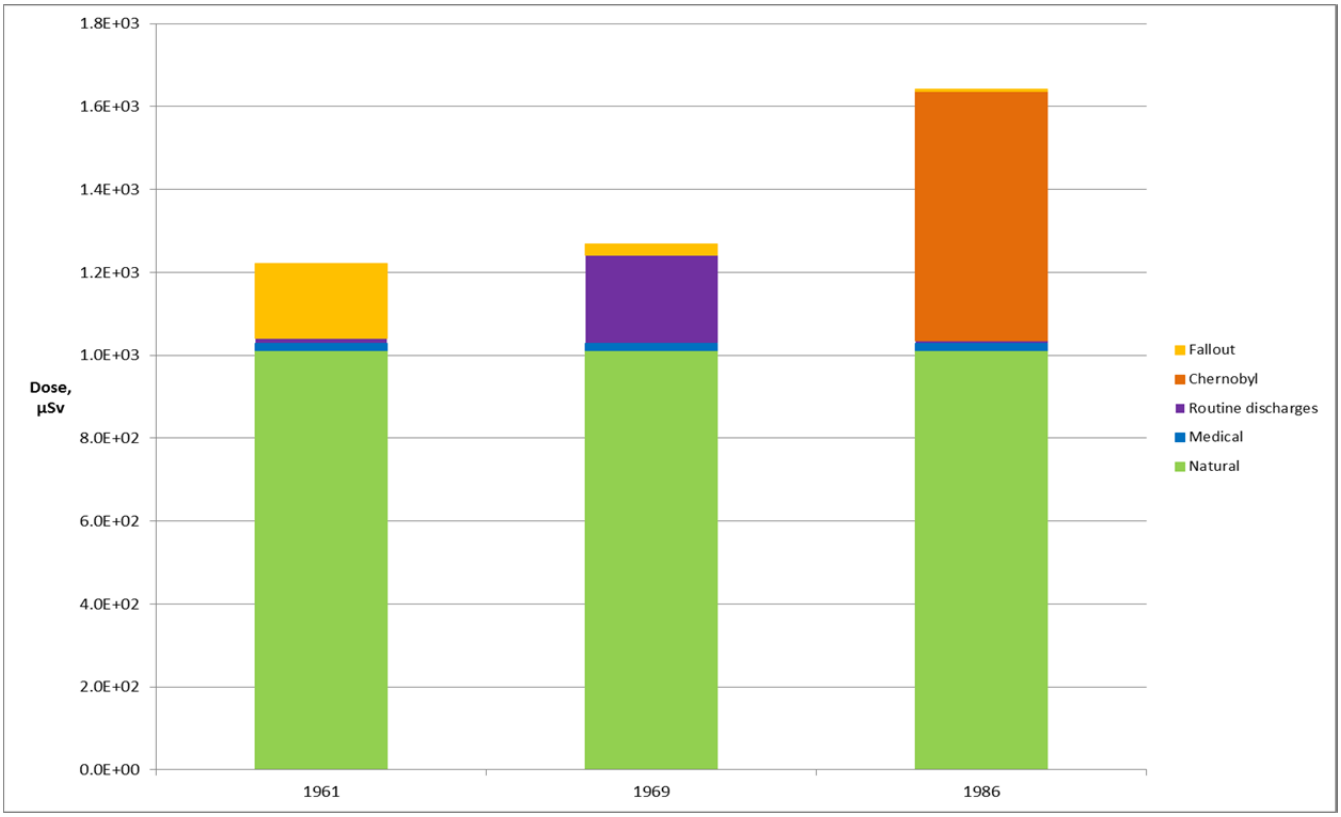

Figure 5.31b Absolute values of thyroid dose resulting from exposure to radionuclides in Thurso at age 10 years in the given years, integrated to (and including) 24 years of age

5.59 The second COMARE report (COMARE, 1988) noted that land-based environmental monitoring around the Dounreay facility had not been as comprehensive as that at Sellafield and recommendation 7 of that report stated that "priority should be given to measuring the levels of radioactivity actually received by the public where possible".

5.60 Watson and Sumner (1996) made measurements of radionuclides in seven groups – Caithness LNHL cases, case siblings, case parents, local controls, local control parents, leukaemia cases from west-central Scotland and controls from west-central Scotland. They argued that if radiation doses from radioactive discharges played a role in the cases of LNHL near Dounreay, then it was most likely that the exposures responsible would be dominated by radionuclides emitting alpha particles, principally plutonium-239, which has been discharged from Dounreay since 1958 with a peak output in the early 1970s (see Figures 5.14 and 5.17 above). Consequently, they assessed plutonium-239 contamination directly by urine analysis and also indirectly by the external counting of americium-241 activity in the skull. Low level whole-body contamination was assayed by whole-body counting – this was capable of detecting the caesium-137 found in seafood and beach particles. In addition, they measured urinary strontium-90 (a radionuclide also present in discharges and in particles) and carried out chromosomal aberration analysis on a subset of the Caithness subjects. Should, for example, the use of local beaches by children be an important source of risk, this study would be expected to reveal excess levels of discharged radionuclides in Caithness cases.

5.61 No significant differences between the groups were observed in measurements of contamination for groups of leukaemia cases, siblings, parents, matched local controls and controls living remote from the plant. Although the detection limits of the equipment limited the measurements in a few instances, the detection limits were well below the levels of radionuclide contamination which could give rise to radiation doses which would explain the LNHL excess. The measurement of plutonium in urine was sufficiently sensitive to be able to detect the presence of weapons testing fallout, but plutonium levels in the urine of case children from Caithness were unexceptional when compared with levels in other children.

Effects of changes in key parameter values since publication of the second COMARE report

5.62 As discussed, the approach adopted was generally to use the same parameter values as were used in the assessment carried out for the second COMARE report, as described in NRPB-R195 (Hill and Cooper, 1986). However, significant changes in key parameter values were considered and their implications for the assessed doses and inferred associated risks were addressed.

5.63 There are some relatively minor changes to the discharge data for the Dounreay site since publication of the second report, as noted above. These do not have any significant impact on the estimated dose arising from liquid or aerial discharges from the Dounreay site.

5.64 The dose coefficients (dose per unit intake values by inhalation and ingestion) used to assess the RBM doses were the values published by the ICRP (2012). There are differences in a number of the dose coefficients of relevance to the calculation of doses from Dounreay and Sellafield discharges compared to the values used in NRPB-R195, which were taken from ICRP Publication 30 (ICRP, 1979). Of note are the decreases in the RBM dose coefficients for inhalation for plutonium-239, americium-241 and strontium-90 by factors of between three and five. There is also an increase of about a factor of two in the

dose per unit intake by ingestion for plutonium-239 between ICRP Publication 30 and current values, and a reduction of about three for americium-241.

5.65 There have also been changes made to the marine model since the NRPB-R195 study, which have changed the estimated activity concentrations in sand from Dounreay discharges and those in seafood from both Sellafield and Dounreay discharges. In turn, these have led to changes in the estimated doses from external irradiation from beach occupancy and ingestion doses from seafood.

5.66 The decrease in doses from inhalation due to changes to dose coefficients is up to a factor of four in years where the aerial discharges from plutonium-239 and americium-241 were highest during periods of the 1970s into the early 1980s when compared with the doses estimated in NRPB-R195. This has no impact on the overall doses due to the very small contribution of inhalation to the total dose. The changes in ingestion dose coefficients and the marine model used have not led to any significant changes to the overall estimated total doses or the trend in the doses observed.

5.67 The REIC calculations for Thurso were carried out using the same models as detailed above for Sellafield. Tables 5.11 and 5.12 give the results for leukaemia and NHL for those born in 1964 and 1975, respectively. Tables 5.13, 5.14 and 5.15 show the data for thyroid cancer for those born in 1961, 1969 and 1975, respectively; the first two of these were chosen to include the periods of highest thyroid doses due to man-made radiation (fallout and site discharges, respectively, see Figures 5.20 and 5.30a).

Table 5.11 Estimated average cumulative 25-year doses to red bone marrow and modelled average risks of radiation-induced incidence of leukaemia or non-Hodgkin lymphoma for an individual born in Thurso in 1964

	Total individual dose (mSv)	**Individual male risk**	**Individual female risk**
Baseline risk	–	$1.3\ 10^{-3}$	$9.2\ 10^{-4}$
Routine discharges	$6.0\ 10^{-1}$	$3.6\ 10^{-6}$	$2.4\ 10^{-6}$
Chernobyl	$1.9\ 10^{-1}$	$9.2\ 10^{-8}$	$5.6\ 10^{-8}$
Weapons fallout	3.1	$4.9\ 10^{-5}$	$3.7\ 10^{-5}$
Medical exposures	1.7	$7.9\ 10^{-6}$	$5.4\ 10^{-6}$
Total man-made radiation	5.6	$6.1\ 10^{-5}$	$4.5\ 10^{-5}$
Natural radiation	$3.5\ 10^{1}$	$2.0\ 10^{-4}$	$1.4\ 10^{-4}$
Total radiation	$4.1\ 10^{1}$	$2.6\ 10^{-4}$	$1.8\ 10^{-4}$

Table 5.12 Estimated average cumulative 25-year doses to red bone marrow and modelled average risks of radiation-induced incidence of leukaemia or non-Hodgkin lymphoma for an individual born in Thurso in 1975

	Total individual dose (mSv)	Individual male risk	Individual female risk
Baseline risk		$1.3\ 10^{-3}$	$9.2\ 10^{-4}$
Routine discharges	$1.4\ 10^{-1}$	$9.8\ 10^{-7}$	$6.9\ 10^{-7}$
Chernobyl	$3.3\ 10^{-1}$	$9.5\ 10^{-7}$	$5.5\ 10^{-7}$
Weapons fallout	$7.3\ 10^{-1}$	$7.9\ 10^{-6}$	$5.7\ 10^{-6}$
Medical exposures	2.1	$8.4\ 10^{-6}$	$5.7\ 10^{-6}$
Total man-made radiation	3.3	$1.8\ 10^{-5}$	$1.3\ 10^{-5}$
Natural radiation	$3.5\ 10^{1}$	$2.0\ 10^{-4}$	$1.4\ 10^{-4}$
Total radiation	$3.8\ 10^{1}$	$2.2\ 10^{-4}$	$1.5\ 10^{-4}$

Table 5.13 Estimated average cumulative 25-year doses to thyroid and modelled average risks of radiation-induced thyroid cancer incidence for an individual born in Thurso in 1961

	Total individual dose (mSv)	Individual male risk	Individual female risk
Baseline risk	–	$6.3\ 10^{-4}$	$1.8\ 10^{-3}$
Routine discharges	1.1	3.110^{-6}	$1.1\ 10^{-5}$
Weapons fallout	2.2	$1.9\ 10^{-5}$	$5.8\ 10^{-5}$
Medical exposures	$6.0\ 10^{-1}$	$1.7\ 10^{-6}$	$5.2\ 10^{-6}$
Total man-made radiation	3.9	$2.4\ 10^{-5}$	$7.4\ 10^{-5}$
Natural radiation	$2.7\ 10^{1}$	$9.5\ 10^{-5}$	$3.0\ 10^{-4}$
Total radiation	$3.1\ 10^{1}$	$1.2\ 10^{-4}$	$3.7\ 10^{-4}$

Table 5.14 Estimated average cumulative 25-year doses to thyroid and modelled average risks of radiation-induced thyroid cancer incidence for an individual born in Thurso in 1969

	Total individual dose (mSv)	Individual male risk	Individual female risk
Baseline risk		$7.0\ 10^{-4}$	$2.3\ 10^{-3}$
Routine discharges	1.0	$9.5\ 10^{-6}$	$3.2\ 10^{-5}$
Chernobyl	$4.8\ 10^{-1}$	$8.7\ 10^{-7}$	$3.1\ 10^{-6}$
Weapons fallout	$4.9\ 10^{-1}$	$3.0\ 10^{-6}$	$1.0\ 10^{-5}$
Medical exposures	$6.4\ 10^{-1}$	$1.9\ 10^{-6}$	$6.6\ 10^{-6}$
Total man-made radiation	2.6	$1.5\ 10^{-5}$	$5.3\ 10^{-5}$
Natural radiation	$2.7\ 10^{1}$	$1.0\ 10^{-4}$	$3.6\ 10^{-4}$
Total radiation	$3.0\ 10^{1}$	$1.2\ 10^{-4}$	$4.1\ 10^{-4}$

Table 5.15 Estimated average cumulative 25-year doses to thyroid and modelled average risks of radiation-induced thyroid cancer incidence for an individual born in Thurso in 1975

	Total individual dose (mSv)	Individual male risk	Individual female risk
Baseline risk	–	$7.6\ 10^{-4}$	$2.5\ 10^{-3}$
Routine discharges	$1.4\ 10^{-1}$	$6.6\ 10^{-7}$	$2.4\ 10^{-6}$
Chernobyl	$8.3\ 10^{-1}$	$2.4\ 10^{-6}$	$9.1\ 10^{-6}$
Weapons fallout	$2.9\ 10^{-1}$	$2.0\ 10^{-6}$	$7.3\ 10^{-6}$
Medical exposures	$7.4\ 10^{-1}$	$2.4\ 10^{-6}$	$8.6\ 10^{-6}$
Total man-made radiation	2.0	$7.5\ 10^{-6}$	$2.8\ 10^{-5}$
Natural radiation	$2.7\ 10^{1}$	$1.2\ 10^{-4}$	$4.3\ 10^{-4}$
Total radiation	$2.9\ 10^{1}$	$1.3\ 10^{-4}$	$4.6\ 10^{-4}$

Other risk estimate considerations

5.68 Soon after publication of the Black report (Black, 1984) and the accompanying radiological assessment by the NRPB (Stather et al, 1984) it was argued that there were serious shortcomings in the estimates of the risk of radiation-induced childhood leukaemia in Seascale resulting from Sellafield discharges (Crouch, 1986). Criticisms included a possible large underestimation of the leukaemogenic risk from alpha particle emitters such as plutonium and from exposure *in utero*.

5.69 Concern about the role of radioactive discharges increased with the need for a revision of the Sellafield discharge chronology that was addressed by the first COMARE report (COMARE, 1986) and the observation of an increased incidence of leukaemia and NHL among young people living near Dounreay, which was the subject of the second COMARE report (COMARE, 1988). However, in its third report (COMARE, 1989), dealing with childhood leukaemia near the Aldermaston and Burghfield nuclear installations, COMARE found that doses from radioactive discharges were “much too low” to account for excess cases, and noted the very different levels of discharges from Sellafield, Dounreay and Aldermaston and Burghfield.

5.70 Substantial efforts were made following publication of the Black report (Black, 1984) to examine uncertainties in the radiological assessments to determine if any major errors were present. It was apparent by the end of the 1980s that no radical errors capable of accounting for the large gaps between the observed numbers of childhood leukaemia cases and the predicted numbers of radiation-induced cases were present (Stather et al, 1988; Wheldon, 1989).

5.71 In particular, measurements of radionuclides in people (Ham et al, 2003; Hodgson et al, 2004) did not find that assessed levels present in tissues as a result of discharges had been seriously underestimated. In many instances, the presence of radionuclides from discharges could not be detected above the background levels from atmospheric nuclear weapons testing (and other sources such as Chernobyl), for example, in the measurement of plutonium in urine samples from childhood leukaemia cases living near Dounreay (Watson and Sumner, 1996). In instances when radionuclides in tissues could be attributed to discharges, levels in excess of background did not differ substantially from predictions as, for example, in the measured levels of plutonium in tissues sampled post-mortem from long-term residents of the locality of Sellafield (Stather et al, 1988; Popplewell and Ham, 1989; Wheldon, 1989).

5.72 The Committee Examining Radiation Risks of Internal Emitters (CERRIE) was established in 2001 to consider the risks associated with radiation exposure from internally deposited radioactive materials. This followed suggestions that conventional radiation risk estimates (based largely, but not solely, on data from those exposed to external sources of penetrating low LET radiation) were inappropriate for internal emitters and that the risk from internally deposited radionuclides had been underestimated by a large factor (in excess of 100).

5.73 The CERRIE final report (CERRIE, 2004) noted that there were features of some internally deposited radionuclides, such as tritium, that required further research, but that there was no reliable evidence for a gross underestimation of the risks arising from internal emitters.

5.74 In its ninth report, COMARE responded to the CERRIE final report, which included the findings of a review commissioned by COMARE of the exposure and health data associated with three groups known to have been exposed to significant internal contamination – Sellafield workers, workers at the Russian Mayak plant and residents of the Techa river villages exposed to radioactive discharges from the Mayak plant (COMARE, 2004b). It was concluded that the risks of radiation-related cancers among these groups exposed to substantial quantities of internally deposited radionuclides were broadly compatible with those derived from the atomic bomb survivors exposed to external sources of radiation; risks from internal emitters that had been underestimated by a factor of several hundreds could be ruled out. An expanded version of the COMARE commissioned review was later published in a scientific journal (Little et al, 2007).

5.75 The doses estimated to have been received from discharges from nuclear power stations have been far too small to account for the excess cases of leukaemia in young children living near the plants that was reported in the German KiKK case–control study (Kaatsch et al, 2008). The KiKK study and similar studies were reviewed in the fourteenth COMARE report (COMARE, 2011).

5.76 Fairlie (2014) has recently returned to the proposition that the risk of radiation-induced childhood leukaemia has been radically underestimated, and suggested that various uncertainties in the radiological risk assessments could combine to produce an overall factor of a 10^4–10^5 underestimate of the radiation-induced risk of childhood cancer, postulating an underestimation of relevant doses by a factor of around 1000, together with an underestimation of risk estimates by a factor of around 100.

5.77 To yield this result of a factor of 10^4–10^5 underestimate, several individual elements are concatenated: ‘spikes’ in power plant aerial emissions, enhanced radiosensitivity of the embryo and early foetus to radiation-induced cancer, uncertain risks from internal emitters (in particular, tritium), no proper account taken of human growth, risks per unit dose greater for infants than for adults, and a highly radiosensitive foetal haematopoietic system. Estimated upper bounds for each of these individual elements are then multiplied together to obtain the overall factor of 10^4–10^5. However, this this factor would be obtained only if the uncertainties were all at their extremes in one direction (ie gross underestimation is assumed to have occurred in all of these contributing factors). Further, it would appear that some of the references from which these individual factors have been obtained have been used inappropriately, with a general tendency to inflate and overestimate individual factors.

5.78 To permit increased emissions from power plants during refuelling outages (that occur approximately annually and last only a few days) to be combined with other uncertainty factors, spikes of releases would have to lead to the exposure of pregnant women resident nearby within the first trimester of pregnancy. Clearly, the number of women downwind of an infrequent transient release while in the first trimester of pregnancy would be limited, but the erroneous assumption would appear to be made that all children in the vicinity of the plant would have been so affected.

5.79 Fairlie (2014) argues that an increase in childhood cancer risk following exposure during the first trimester by a factor of five, as compared to the risk from exposure during the third trimester, can be obtained from the study of Stewart et al (1958). However, Stewart et al (1958) observed that first trimester exposures in their study were few, and subsequently Stewart and Kneale (1970) emphasised that this is in any event likely to be "more a dose effect than a susceptibility effect" because of the higher doses used in the types of X-ray examinations occurring during the first trimester (ie examinations not performed for obstetric reasons).

5.80 It is correct to note that the Advisory Group on Ionising Radiation (AGIR, 2007) recommended the use in epidemiological studies of an RBE of two for tritium beta particles compared to gamma rays but this factor will apply only to the relevant component of the overall dose.

5.81 The factor of 10 used for radionuclide dose coefficients (Richardson, 2009) specifically was not intended to apply to the foetus/embryo but to infants and young children. Furthermore, it was proposed for ingestion and not inhalation as assumed by Fairlie.

5.82 A factor of five increase proposed in the relative risk from internally deposited emitters is based upon the work of Fucic et al (2008), who compared the rates of spontaneous abortion in women working with X-rays with that of women working with radionuclide sources. The rate for those working with radionuclides was 3.9 times that for the X-ray group, but there were no dose estimates available and the authors suggested that a possible explanation lay in the toxicity/mutagenicity/teratogenicity of the materials handled by the radionuclide group. Therefore, it would appear necessary to exercise caution in applying these data to the current discussion.

5.83 There is a misinterpretation of the findings reported by Ohtaki et al (2004) as showing that an enhanced radiosensitivity of the foetal haematopoietic system leads to an underestimation of the risk of childhood leukaemia by a factor of around 100. This study demonstrates that the Japanese atomic bomb survivors who were irradiated *in utero* and received a dose in excess of around 100 mGy did not exhibit raised rates of stable chromosomal translocations (in contrast to their mothers, who did), suggesting that haematopoietic cells that were damaged by moderate and high doses to an extent that would have produced translocations had the same dose been received by an adult were killed by this dose. The particular sensitivity of this population of foetal haematopoietic cells to cell-killing has been suggested to be the reason for the absence of childhood leukaemia among the survivors who were irradiated *in utero* – the cells that might have become leukaemic through their exposure to moderate and high doses were killed instead. As a consequence, it is difficult to see how this study justifies a claim of an underestimate of childhood leukaemia risk by a factor of around 100.

5.84 In summary, the suggestion by Fairlie (2014) of evidence for an underestimation of risk by a factor of 10^4–10^5 is not supported by the literature that is quoted to provide its basis.

5.85 If the above hypothesis were to be correct, however, and there are great underestimates in dose and risk estimates to the extent required to explain the observation of excess cases of childhood leukaemia near some nuclear installations, then much higher than expected rates of childhood leukaemia should be apparent under other circumstances of exposure to radionuclides. Particularly important in this respect is atmospheric nuclear weapons testing, which was at its peak in the late 1950s and early 1960s, leading to worldwide exposure to the radioactive debris of these explosions and to the intake of a range of radionuclides similar to that released from nuclear reactors and spent nuclear fuel reprocessing plants, including a large quantity of tritium released from thermonuclear weapons tests. However, large-scale accurate and complete registration of childhood leukaemia in the early 1960s and before was not commonplace, and the use of childhood leukaemia mortality data is not an acceptable alternative since treatment, and hence survival, was becoming increasingly successful after 1960. Nonetheless, a number of childhood cancer registries covering this period do exist.

5.86 Darby et al (1992) found that childhood leukaemia incidence in the Nordic countries during the period following the highest fallout from test explosions was slightly, and marginally significantly, higher than in adjacent periods. Stevens et al (1990) conducted a case–control study of leukaemia deaths in Utah and found a significantly raised relative risk of acute leukaemia mortality among young people who had received the highest assessed doses (>6 mGy) from radioactive fallout from nuclear weapons explosions at the Nevada test site in the neighbouring state. The results of both studies were compatible with conventional risk estimates and do not indicate a large underestimation of risk from fallout.

5.87 The childhood leukaemia incidence data from 11 large-scale registries from three continents has been collated and examined by Wakeford and colleagues (Wakeford et al, 2010; Wakeford, 2014), who found no evidence that the marked peak of intake of man-made radionuclides present in nuclear weapons testing fallout detectably influenced the subsequent risk of childhood leukaemia beyond the predictions of conventional leukaemia risk models. Further, the southern hemisphere received lower levels of fallout than the northern hemisphere (where most testing took place), but levels of childhood leukaemia incidence do not differ detectably between hemispheres. These findings provide strong evidence against the suggestion of materially higher risks arising from internally deposited radionuclides.

5.88 The question of risks from low doses and dose rates and the validity of the linear no-threshold (LNT) dose-response model have been addressed recently by a number of investigators. A study of some 65,000 employees of BNFL (Gillies and Haylock, 2014) found excess cancer risks for those exposed to low level gamma radiation over a number of years to be broadly in line with those calculated from the Japanese atomic bomb survivors. Approximately 20,000 employees were classed as external radiation workers, monitored only for exposure to external sources of radiation, and nearly 23,000 as internal radiation workers, monitored for exposure to both external radiation and potential internal deposition of radionuclides (principally uranium, plutonium and tritium), although no estimates of internal doses were available for the study; the ascertainment rate was over 99%. Within this cohort, internal radiation monitoring status modified the dose response for external dose, with

significant heterogeneity observed, suggesting lower cancer risks associated with external radiation exposure for workers who were additionally monitored for internal radiation. No explanation for this phenomenon was found and further investigations are required to address this issue.

5.89 Leuraud et al (2015) studied a cohort of over 300,000 workers employed in the nuclear industry in France, the UK and the USA (the International Nuclear Workers Study, INWORKS) in order to establish the excess risk of leukaemia (excluding chronic lymphoid leukaemia) associated with protracted low dose rate exposure to external sources of radiation; the BNFL workers formed a significant part of this cohort. The mean annual RBM dose was 1.1 mGy with a median of 2.1 mGy and the follow-up period was some 25 years. The results were consistent with the conventional risk estimates extrapolated from male atomic bomb survivors exposed to moderate/high acute doses as adults.

5.90 The leukaemia association in INWORKS did not depend upon the inclusion of any one country, but the overall result was influenced strongly by the UK and US cohorts. Even though the association is statistically significant only when workers with cumulative RBM doses greater than 200 mGy are included, these doses were received protractedly, generally over many years. Sensitivity analysis found little effect modification by monitoring status for exposure to internal emitters.

5.91 These recent studies of radiation workers lend support to risk estimates based upon the LNT dose-response model, but are relevant only to adult exposures. However, recent studies have also directly assessed risks arising from exposure in childhood. Large studies of children who have been examined using computed tomography (CT) have the potential of detecting the predicted radiation-induced risk of childhood leukaemia (and possibly other cancers). The first such study to be reported was of more than 175,000 young people examined with CT in Great Britain during 1985–2002 who were followed up for a diagnosis of leukaemia or a brain tumour during 1985–2008 (Pearce et al, 2012). Statistically significant associations between assessed radiation dose and leukaemia and brain tumours were found. There are difficulties of interpretation of the results of such studies because of the potential influence of reverse causation and confounding by indication – the disease, or a factor predisposing to the disease, caused the CT scan rather than vice versa (Walsh et al, 2014; Boice, 2015). However, this may be less of a problem for leukaemia, and the leukaemia ERR per mGy reported by Pearce et al (2012) is compatible with that obtained from models based upon the Japanese atomic bomb survivors.

5.92 Conventional radiation-induced leukaemia risk models predict that around 15–20% of cases of childhood leukaemia in Great Britain may be caused by natural background radiation (Wakeford et al, 2009). Very large case–control studies of 10,000 or more cases of childhood leukaemia are required to have a reasonable prospect of detecting this risk, but one such record-based study has now been conducted, and the initial findings show a significant association between natural background gamma radiation and childhood leukaemia risk (Kendall et al, 2013). The level of this association is consistent with predictions of conventional radiation-induced leukaemia risk models. This suggests that an excess risk of childhood leukaemia exists following the lowest levels of exposure to radiation, but that the magnitude of this excess risk is predicted by recent risk models with reasonable accuracy.

5.93 Although there is evidence for an excess risk of leukaemia following low dose or low dose rate exposure to radiation, this does not necessarily imply

that all cancers have a similar response to radiation exposure. Indeed, some types of cancer (eg Hodgkin's lymphoma and cancers of the uterus and rectum) exhibit a low sensitivity to induction by radiation, in contrast to other types (eg cancers of the bladder and female breast).

5.94 In contrast to the belief that radiation risks have been underestimated, there are suggestions that very low doses of radiation are at least harmless and may possibly be beneficial – see, for example, Feinendegen (2005), Thompson (2011) and Jargin (2012). This concept of 'radiation hormesis' generally depends on the proposition that very low doses of radiation, around or just above natural background, are beneficial by stimulating repair mechanisms that protect against disease and are not activated in the absence of ionising radiation. These mechanisms are hypothesised to be sufficiently effective when stimulated so as not only to cancel the detrimental effects of ionising radiation but also to provide future protection.

5.95 COMARE believes that there is no reason to revise its view stated in its ninth report (COMARE, 2004b) that such processes are not impossible, but that no substantial evidence has been presented to date to place any reliance upon the concept of radiation hormesis.

5.96 Finally, were the assessed radiation doses to be responsible for the observed leukaemia excesses, the incidence rates should show a correlation with dose, but this is not the case. Table 5.16 shows the calculated and reported enhancement factors for leukaemia incidence in three areas of Great Britain that have been the subject of COMARE reviews: Sellafield, Dounreay and Aldermaston and Burghfield (Wheldon, 1989). The calculated enhancement factor is the excess risk of leukaemia incidence predicted from the estimated population radiation dose, while the reported enhancement factor is the ratio of the observed incidence rate compared to that expected from the national incidence rate. The discrepancy factor is the ratio of the excess over unity of the reported enhancement factor to the excess over unity of the predicted enhancement factor. The discrepancy factor thus gives a crude indication of the extent to which the predicted excess falls short of explaining the reported excess.

5.97 The discrepancy factors vary by some orders of magnitude. Since any error in the risk factor for conversion of dose to excess risk would be common to all sites, this implies either that the calculated doses are in error by these varying amounts or that radiation cannot be the sole factor causing the increase. The former is highly unlikely given the amount of data available and so these data lend weight to the latter hypothesis.

Table 5.16 Predicted enhancement of childhood leukaemia risk compared with the reported enhancement of risk (Wheldon, 1989)

Nuclear site	Predicted enhancement ratio	Reported enhancement ratio	Discrepancy factor
Sellafield	1.02	~8	350
Dounreay	1.002	~2–6	500–2,500
Aldermaston	1.000002	~1.4	200,000

Conclusions

5.98 COMARE are content that the position remains that there is no reliable evidence of substantial inaccuracies in the conventional radiation risk estimates. The present evidence for radiation-induced leukaemia in young people suggests that there is some risk from exposure to low doses or low dose rates of radiation, but that this risk is broadly compatible with the predictions of models based on brief exposures to moderate and high doses.

5.99 Although radiation doses to the red bone marrow (RBM) of children living in Seascale arising from Sellafield radioactive discharges before 1960 are significantly higher than those received subsequently, when set in the context of doses from other sources, the doses from Sellafield discharges are a minor component of overall RBM doses, even during the early years of site operations. Natural background is the main contributor to the RBM dose at most times. There continues to be no evidence that RBM doses from discharges from Sellafield or Dounreay have been substantially underestimated.

5.100 COMARE concludes that the current best estimates of the radiation doses to both the Seascale and Dounreay populations remain far too small to account for the observed numbers of cases of leukaemia and NHL that have occurred in young people living in these areas during the time period studied.

5.101 The increased risk of thyroid cancer incidence among those born in Thurso due to Dounreay and Sellafield discharges is very small, less than a tenth of the risk due to natural background radiation. For those born in Seascale during the 1950s, the average risk of thyroid cancer incidence due to Sellafield discharges is three to four times greater than that due to natural background radiation in some birth years, although always less than the background risk in the absence of additional radiation exposure. No excess of thyroid cancer was found among those born during 1950–2006 in Seascale or the rest of the area surrounding Sellafield.

CHAPTER 6

POSSIBLE EFFECTS OF PATERNAL PRECONCEPTIONAL IRRADIATION ON CANCER IN OFFSPRING

Previous consideration of paternal preconceptional irradiation (PPI) by COMARE

6.1 The second COMARE report (COMARE, 1988) investigated a reported excess of leukaemia among young people living around the Dounreay nuclear establishment in northern Scotland, particularly in the town of Thurso, Caithness (Heasman et al, 1986, 1987). The second report stated that "some feature" of the nuclear installations at Sellafield and Dounreay was likely to be responsible for the raised incidence of leukaemia and non-Hodgkin lymphoma (LNHL) among young people living in their vicinities, but that "Conventional dose and risk estimates suggest that neither authorised nor accidental discharges could be responsible" for the raised incidence and alternative explanations were considered, including the possibility that parental occupation could be relevant. A recommendation from the report was that "epidemiological studies should be set up to consider any possible effects on the health of the offspring of parents occupationally exposed to radiation". This recommendation was reiterated in the third COMARE report (COMARE, 1989).

6.2 The first recommendation made by the Black Advisory Group was that a case–control study should be undertaken using records of cases of leukaemia and lymphoma diagnosed in young people (up to the age of 25 years), resident in west Cumbria (Black, 1984). The findings of this study, funded by the Department of Health (DH) and the Medical Research Council (MRC), were published by Professor Martin Gardner and his colleagues in 1990 (Gardner et al, 1990b) and is often referred to as the 'Gardner report'.

The Gardner report

6.3 The Gardner report (Gardner et al, 1990a,b) examined demographic, social and behavioural factors and medical histories for 52 leukaemia and 45 lymphoma patients aged 0–24 years in West Cumbria district between 1950 and 1985. These cases were compared with two control groups matched for sex and date of birth, and on birth either in the same community or in the district.

6.4 The study showed a statistical association between paternal employment at Sellafield and the risk of leukaemia (and leukaemia plus NHL) in children, although similar associations were found for paternal employment in the iron and steel and farming industries. However, the raised risk for fathers employed at Sellafield was particularly notable and statistically significant for those who had received relatively high recorded doses of external radiation before the conception of their children: >100 mSv in the total preconceptional period or >10 mSv in the six months immediately preceding conception. Nonetheless, these associations were based on just (the same) four cases of leukaemia and a similarly small number of controls.

6.5 Gardner et al (1990b) suggested that the association between leukaemia and NHL in young people and paternal preconceptional irradiation (PPI) could explain statistically the excess of cases in Seascale. This suggestion was made more attractive by the failure of environmental exposure to radiation to provide a plausible explanation for the excess of cases in Seascale.

6.6 COMARE was asked to provide advice to government following publication of the Gardner report. The interim statement agreed with the Gardner study methodology, and with the broad thrust of the main conclusions (COMARE, 1990), but COMARE was cautious in its interpretation because the findings "are unavoidably based on very small numbers and are novel observations which have not been recorded previously". It noted that the case–control study demonstrated a statistical association, but no causal relationship, and that the findings required confirmation by other studies. The statement reaffirmed the need for further major epidemiological studies.

Health and Safety Executive (HSE) study

6.7 Given the potential implications of the findings of the Gardner report, the Health and Safety Executive (HSE, 1993, 1994) conducted its own investigation into cancers among the offspring of workers at Sellafield. This consisted principally of a case–control study, with inevitably a considerable overlap of cases with those included by Gardner et al (1990a,b). The HSE study confirmed the result of Gardner et al (1990b) of a significant association between the cumulative PPI dose of external radiation and LNHL in young people but, using more detailed dose records, was not able to confirm the association with the external dose received in the period immediately preceding conception. The HSE study also had access to internal dose records, and found no association between LNHL and internal PPI dose to the testes. No association was found for PPI dose and other cancers.

6.8 One of the more notable findings of the HSE study was that the PPI association for LNHL was effectively confined to Seascale: a highly significant dose response in Seascale contrasted with an unexceptional trend with dose in the rest of west Cumbria. Moreover, the initial results of a Cumbrian birth cohort study had found that less than 10% of births to fathers with a PPI dose at Sellafield were Seascale births, and births outside Seascale tended to be associated with higher PPI doses, which made the effective confinement of the PPI effect to Seascale even more remarkable (Parker et al, 1993).

Fourth COMARE report

6.9 The fourth COMARE report considered in depth the possible effects of PPI on cancer risk and in particular whether PPI could explain the raised incidence of childhood leukaemia in Seascale (COMARE, 1996). It reviewed data published both before and after the Gardner report.

6.10 Studies of the effects of PPI in humans are primarily based upon the offspring of the Japanese atomic bomb survivors, of nuclear industry workers and of patients who have been exposed to radiation for medical reasons, and these studies were reviewed by COMARE.

6.11 Yoshimoto et al (1990) examined the risk of cancer among over 31,000 live-born Japanese children, with one or both parents having received a gonadal dose in excess of 10 mSv during the atomic bombings. No association between overall cancer or leukaemia incidence before the age of 20 years and preconceptional dose was found (Yoshimoto et al, 1990, 1991). Little (1993) demonstrated that the absence of detectable leukaemia risk in the offspring of irradiated Japanese atomic bomb survivors was incompatible with the findings of Gardner et al (1990b).

6.12 Case–control studies conducted in the vicinities of Dounreay and of Aldermaston and Burghfield, although of low statistical power, had nonetheless demonstrated that PPI could not explain the excesses of LNHL that had been found there, because only a limited number of cases were so exposed (Urquhart et al, 1991; Roman et al, 1993). Consequently, PPI could not be the 'common feature' of the neighbourhoods of Sellafield and Dounreay that could account

for the excess cases found there (COMARE, 1988).

6.13 Studies of the offspring of nuclear industry workers that used data independent of the Gardner report did not find support for the association between PPI and leukaemia: neither the case–control study of Ontario workers (McLaughlin et al, 1993) nor that of Scottish workers (Kinlen et al, 1993) found evidence for a risk from PPI.

6.14 No evidence was found from studies of the offspring of cancer patients treated with radiation and/or cytotoxic drugs for an effect of PPI upon the risk of leukaemia. Studies of external exposure for diagnostic reasons produced broadly negative findings, but were limited in their reliability and power. A study of offspring of Danish patients to whom the radioactive (thorium-based) contrast medium Thorotrast had been administered found no cases of leukaemia or NHL; the internal alpha particle dose received by the testes of these patients was sufficient to provide reasonable power (Andersson et al, 1994).

6.15 Little et al (1996) compared the PPI dose responses obtained from studies of the children of nuclear industry workers, Japanese atomic bomb survivors and Danish Thorotrast patients. With the notable exception of the highly significant dose response for offspring of Sellafield workers born in Seascale derived from the HSE study, the dose responses were unremarkable, including that for the offspring of Sellafield workers born outside Seascale.

6.16 COMARE noted that, particularly from large-scale mouse studies, the experimental evidence for non-carcinogenic transgenerational effects of PPI, such as congenital malformations, was well established. However, the fourth report expressed reservations over the findings of laboratory studies showing a raised incidence of leukaemia and other cancers in the offspring of irradiated animals, and noted that, even if these findings were real, they were specific to certain strains of laboratory animals and not generally applicable.

6.17 The fourth report also considered other potential transgenerational effects, such as those from chemical exposure. One candidate in this regard is benzene, which, for over a century, has been known to cause damage to the bone marrow of highly exposed individuals, resulting in aplastic anaemia; in 1938, Penati and Vigliana published the first study of occupational exposure leading to leukaemia (Penati and Vigliani, 1938). This chemical is of interest because benzene or derivative substances have been used in the nuclear, farming, and iron and steel industries, all of which were identified in the Gardner report as potentially conferring a higher risk of leukaemia in offspring (Gardner et al, 1990b).

6.18 While there has been substantial progress in understanding the causative mechanisms involved in leukaemia induction by benzene (Snyder, 2012), there is no evidence of any mechanism for the occurrence of leukaemia in the offspring of exposed workers. It has been found that myeloid leukaemia in exposed individuals is the much more likely result of high level exposure to benzene than lymphoid leukaemia, with a mean latent period of around 11 years (Infante et al, 1977; Yin et al, 1987). Both the Seascale and Dounreay excesses, however, comprise a majority of lymphoid cases. Although it is known that large quantities of benzene were used by and emitted from both sites, were there to be a link, it would be expected that the adult rate of leukaemia around the plants would be raised, which is not the case. There is no evidence of a link between low level environmental exposure to benzene and childhood leukaemia. It is thus highly unlikely that this is even a contributory cause.

6.19 The fourth COMARE report concluded "We have not found any epidemiological study elsewhere to support Gardner's findings in Seascale in relation to preconception radiation effects" and "We consider that PPI cannot account for the Seascale childhood leukaemia excess" (COMARE, 1996).

Co-ordinating Committee on Health Aspects of Radiation Research (CCHARR)

6.20 In 1990, the Co-ordinating Committee on Health Aspects of Radiation Research (CCHARR) was established by DH and HSE to manage government-sponsored research investigating the association found by Professor Gardner and his colleagues. On completion of the CCHARR studies, COMARE was asked by DH and HSE to review all of the evidence available for the existence of carcinogenic effects in the children of radiation workers in the UK. The seventh COMARE report was the outcome of that review.

Seventh COMARE report

6.21 Several epidemiological studies of relevance to childhood cancer and parental exposure to radiation had been published since the fourth COMARE report, including the two large studies carried out in the UK that had been recommended by CCHARR.

6.22 The Record Linkage Study (RLS) was a nationwide case–control study in which the parents of children who had developed cancer and of matched controls were linked to people in the National Registry for Radiation Workers (Draper et al, 1997). They excluded cases studied by Gardner et al (1990b) to achieve independent results, and found that fathers of children with leukaemia or NHL were more likely to have been radiation workers than fathers of controls, but that there was no evidence of a PPI dose response, children of fathers with the lowest doses having the highest risk. Draper et al (1997) concluded that the results of the RLS did not support the hypothesis that PPI is a cause of childhood leukaemia and NHL.

6.23 The Nuclear Industry Family Study (NIFS) (Roman et al, 1999) was a questionnaire-based study enquiring of nuclear industry workers whether their children had developed cancer. The authors concluded that the incidence of cancer and leukaemia among children of nuclear industry employees was similar to that in the general population. Only one case of leukaemia with a PPI dose >100 mSv was included in the NIFS that was not included in the hypothesis-generating study of Gardner et al (1990a,b).

6.24 In 2002, the findings of the extended Cumbrian birth cohort study of cancer in the offspring of Sellafield workers were published (Dickinson and Parker, 2002; Dickinson et al, 2002). The risk of leukaemia and NHL in children of male Sellafield radiation workers was significantly raised in comparison to children of non-Sellafield, non-Seascale fathers, particularly among young children in Seascale, although adjustment for demographic factors reduced the relative risk to a non-significant level (Dickinson and Parker, 2002). The PPI dose response was slightly and non-significantly raised for children born outside Seascale but, surprisingly, given the findings of the HSE study, the positive dose response for Seascale-born children was rather shallow and of marginal statistical significance (Dickinson and Parker, 2002). For cancers other than leukaemia and NHL the results were unexceptional (Dickinson et al, 2002).

6.25 A case–control study of childhood cancer around three nuclear sites in the USA, and linkage to fathers working at those sites and to PPI doses, was conducted by Sever et al (1997). There was no preferential tendency for fathers to be employed at the nuclear facilities and the slope of the PPI dose response for leukaemia and NHL was non-significantly negative.

6.26 A study of offspring of around 18,000 Swedish women treated in early childhood with radiation for skin haemangioma found a non-significant deficit of childhood cancers when compared with national rates (Kallen et al, 1998). Studies of offspring of cancer survivors did not provide evidence of a link between the risk of childhood cancer and PPI, although COMARE noted the problems of interpretation of these studies, including genetic predisposition to some forms of cancer.

6.27 Following the review of these and other epidemiological studies, and of evidence from experimental studies of the response of cells and of animals to radiation exposure, COMARE arrived at the conclusion in its seventh report: "We find no convincing evidence to suggest that ionising radiation alone at the doses to which male nuclear industry radiation workers have been exposed, results in an increased incidence of childhood cancer" (COMARE, 2002).

Studies of parental exposure since the seventh COMARE report

6.28 The eighth COMARE report examined the evidence for adverse pregnancy outcomes (eg congenital malformations and stillbirths) following preconceptional exposure to radiation of parents (COMARE, 2004a). Both epidemiological and experimental studies were reviewed. COMARE concluded that while there was no doubt from the results of large-scale laboratory animal studies that high preconceptional doses of radiation could induce congenital malformations in offspring, there was little evidence from epidemiological studies that the hereditary genetic risk estimates for humans that have been derived from these experimental studies have underestimated the transgenerational risks of radiation exposure.

6.29 The evidence relevant to PPI and childhood leukaemia that has become available since publication of the seventh COMARE report will now be considered.

Nuclear and other workers

6.30 Dickinson et al (2003) made a detailed comparison of the findings of the HSE case–control study and the extended Cumbrian birth cohort study. They found that the notable difference between the steep and highly significant dose response for Seascale found in the HSE case–control study and the shallow and marginally significant dose response for these births found in the Cumbrian birth cohort study (based on the same six cases of LNHL) was due to the unusual PPI dose distribution for Seascale births – a long upper 'tail' in this distribution had a strong downwards leverage effect in the cohort study, but no controls were sampled from this 'tail' in the HSE case–control study. The authors concluded that the cohort study provided a more reliable assessment of the dose response for births in Seascale.

6.31 In a further analysis of the RLS data, Sorahan et al (2003) examined the timing of paternal employment as a radiation worker. They found that children of fathers who had stopped radiation work prior to conception were not at an increased risk of LNHL, although they could not distinguish between the risks associated with such work at conception or diagnosis. There was no risk associated with PPI dose.

6.32 Also working with RLS data, Bunch et al (2009) analysed the risk of cancer in the offspring of female radiation workers. No evidence was found of an increased risk of childhood cancer and maternal preconceptional work with radiation.

6.33 In contrast to the epidemiological studies based on records of exposure to ionising radiation and the doses of PPI received, a number of studies have been conducted in which exposure to radiation has been inferred, for example,

from job title. The results of such studies are less reliable because of the assumptions about the likelihood of exposure to radiation that have to be made, but the studies of McKinney et al (2003) and Keegan et al (2012) did not find evidence to support an effect of PPI upon the risk of childhood leukaemia.

6.34 Johnson et al (2008) examined the risk of cancer in children born to US radiologic technologists (radiographers). Parental doses were estimated from work history data, badge dose data and literature doses. They did not demonstrate any convincing evidence of an increased risk of childhood cancer in the offspring of radiologic technologists in association with paternal occupational radiation.

Japanese atomic bomb survivors

6.35 Offspring of the Japanese atomic bomb survivors have been further studied in relation to cancer incidence (Izumi et al, 2003a) and mortality (Izumi et al, 2003b; Grant et al, 2015) and the PPI doses received during the bombings. The mortality studies found no increase in cancer mortality rate that was related to parental radiation dose, either for offspring of all ages or for deaths at less than 20 years of age. The cancer incidence study confirmed these negative findings.

Medical exposures

6.36 Studies of the health of offspring of survivors of cancer who were conceived after treatment with radiation continue to show little adverse effect. A nationwide study in Finland found that offspring were not at an increased risk of cancer except when a cancer-predisposing syndrome exists (Madanat-Harjuoja et al, 2010). Winther et al (2012), in a study of genetic disease in the offspring of Danish cancer survivors, noted that not a single case of leukaemia had occurred in these offspring; 722 fathers had a mean testicular PPI dose of 410 mGy.

6.37 Winther et al (2004) studied the offspring of children treated for cancer, using the offspring of untreated siblings as a comparator cohort. They found no difference in the rate of occurrence of abnormal karyotypes in the two groups.

6.38 These negative findings for cancer risk in the offspring of cancer survivors extend to other adverse outcomes, such as congenital malformations. Mulvihill (2012) has concluded that the evidence suggests that the human germ-cell genome is much more resistant to environmental mutagens than previously thought. Draper (2008) also concluded, from a review of the evidence, that while human germ-cell mutations must be assumed to exist, studies so far have failed to detect their effects.

6.39 As noted in the seventh COMARE report, the interpretation of the results of studies of the offspring of cancer survivors in terms of an effect of PPI is not straightforward because of factors such as the hereditary genetic predisposition to some forms of cancer, the effects of the potential administration of chemotherapeutic drugs, and the heterogeneity of the dose received during most treatments with radiation. However, considerable effort has been expended in addressing these problems, such as detailed gonadal dose reconstruction programmes (Boice et al, 2003), and the continued lack of evidence from more recent studies of offspring conceived after the diagnosis of cancer in a parent gives reassurance that these children are not at a materially increased risk of cancer or other adverse health outcome.

6.40 In a case–control study of childhood leukaemia and parental exposure to diagnostic X-ray examinations prior to the birth of the child, Bailey et al (2010) found little evidence of any effect of maternal exposures, although a weak and non-significant association with paternal exposures was found.

However, in contrast to studies of nuclear workers based upon dose records, X-ray exposures were based on parental reporting and were not checked against medical records.

Experimental studies

6.41 The seventh COMARE report noted that the limited findings for an increased risk of leukaemia in the offspring of male mice receiving high preconceptional radiation doses, if real, are clearly strain dependent, and cannot be explained in terms of conventional mutations directly induced by irradiation of the germ cells. COMARE noted the evidence for 'unconventional' mechanisms, such as epigenetic effects and genomic instability, which needed further investigation, but concluded that such mechanisms could not feasibly account for the excess cases of LNHL among the offspring born in Seascale to male Sellafield workers, particularly given the lack of evidence for a raised risk among other fathers exposed to radiation.

6.42 Despite this conclusion and the epidemiological evidence now firmly indicating that PPI cannot be the cause of the excess cases of leukaemia and NHL among young people in Seascale, it has been proposed that transgenerational genomic instability could imply that heritable disease risks in humans have been underestimated, that offspring of irradiated parents may be at a greater risk of developing cancer over their lifetime, and that this can "provide a plausible explanation" for the Seascale excess cases (Barber and Dubrova, 2006).

6.43 Bouffler et al (2006) summarised the information then available from molecular genetic and related studies of germline mutation, largely in males, which reflected the outcome of a review conducted by the authors for COMARE in the preparation of the seventh report. The review paid particular attention to one example of an 'unconventional' transgenerational mechanism, that of minisatellite mutations (changes in the non-coding sequences of DNA), raised rates of which had been reported in humans and mice. The authors concluded that the available findings did not warrant a dramatic revision of germline or cancer risk estimates for radiation exposure.

Minisatellite mutations

6.44 A high rate of minisatellite mutations has been reported from the studies of Dubrova and colleagues, and others. These studies include the offspring of those living in areas heavily contaminated by the Chernobyl accident (Dubrova et al, 1996, 1997, 2002b), of those living in Kazakhstan exposed to fallout from the Semipalatinsk nuclear weapons testing site (Dubrova et al, 2002a), and of those living along the Techa river in areas contaminated by radioactive discharges from the Mayak plant (Dubrova et al, 2006). The increase in minisatellite mutation frequency was also reported from some experimental studies with mice (Barber et al, 2002).

6.45 However, investigations of minisatellite mutations in the children of Japanese atomic bomb survivors have continued to produce negative findings (Kodaira et al, 2004, 2010). No increase in the germline minisatellite mutation rate was found in the offspring of cancer survivors (male or female) treated with radiation (Tawn et al, 2011). Further studies of the families of Chernobyl emergency and clean-up workers have continued to find little support for an association between radiation exposure and a raised rate of minisatellite mutations (Kiuru et al, 2003; Slebos et al, 2004) or of microsatellite mutations (Furitsu et al, 2005), which in any event have not been associated with PPI (Little et al, 2013). Tawn et al (2015) have investigated germline minisatellite mutations in the families of male radiation workers at Sellafield and found no increase of mutation rate with PPI dose. No significant increase in either minisatellite or microsatellite mutations was observed in residents of a high

natural background radiation area in Kerala, India, when compared to those living in a neighbouring area with 'normal' ambient radiation levels (Ahmad et al, 2013).

6.46 In a recent review of the evidence for radiation-induced minisatellite mutations, Little (2015) has noted the disparity between the findings of the studies conducted by Dubrova and colleagues and those of other investigators, and discussed possible reasons for this. Whatever the reason might be, it is clear that germline minisatellite mutations cannot provide an explanation for the excess of cases of leukaemia and NHL among a subset of young people in Seascale associated with relatively high doses of PPI, particularly given the absence of reliable evidence for such an association from other studies.

6.47 Mughal et al (2012) exposed mice to acute low-to-moderate and moderate-to-high doses and chronic low dose rates of gamma radiation, investigating the effect of PPI on transgenerational genomic instability. While paternal exposure to moderate-to-high doses of acute irradiation had a detectable effect upon genome stability of offspring, low-to-moderate acute doses or low dose rates did not have a discernible effect. The authors suggested that their results implied that transgenerational genomic instability may only be triggered by acute paternal irradiation above a threshold dose, which would be higher for chronic exposure than for acute exposure.

6.48 Little et al (2013) performed a comprehensive review of the literature relevant to health effects in the offspring of irradiated parents. They concluded that irradiation of male laboratory animals at high doses (mostly 1 Gy and above) can cause detectable genetic and epigenetic effects in the somatic cells of their offspring over several generations that are not attributable to the inheritance of a simple mutation through the parental germline. However, human studies have found no evidence of any effect of parental irradiation on their offspring. The authors suggested that one possible explanation for the difference between the laboratory animal and human findings may be that transgenerational effects are restricted to relatively short times post-exposure. They proposed that the striking differences between cellular/animal studies and studies of human health should be the subject of further research.

Conclusions

6.49 The epidemiological and experimental studies of relevance to PPI and childhood leukaemia that have been published since the seventh COMARE report have consolidated the previous conclusions of COMARE that irradiation of parents before the conception of their children does not detectably increase the risk of leukaemia in these children. The novel statistical association between PPI and childhood leukaemia reported by Gardner et al (1990b) cannot account for the excess of childhood leukaemia cases in Seascale and is most likely to have been a chance effect.

CHAPTER 7

EVIDENCE FOR AN INFECTIOUS AETIOLOGY FOR CHILDHOOD LEUKAEMIA IN RELATION TO THE EXCESS CASES AROUND SELLAFIELD AND DOUNREAY

7.1 This chapter focuses on the potential for an infectious aetiology for childhood leukaemia, first suggested nearly 100 years ago (Ward, 1917) and revisited in subsequent years – see, for example, Poynton et al (1922). Kellett (1937) and Cooke (1942). Early efforts were directed mainly at identifying a directly transmissible human leukaemic virus but, more recently, attention has been paid to investigating a rare leukaemogenic response to a common infection or infections as a causal factor. Particular consideration will be given to the relevance of an infectious aetiology to the Sellafield and Dounreay areas and the potential role of population mixing.

7.2 The Black Advisory Group (Black, 1984) considered briefly the possibility that the increased incidence of childhood leukaemia in West Cumbria was due to factors other than ionising radiation. The Group found no convincing evidence for the involvement of any unexpected environmental carcinogen or agent peculiar to the village of Seascale, although the rapid development of research on human leukaemia viruses was noted.

7.3 In its second (1988), third (1989) and fourth (1996) reports, COMARE suggested hypotheses for the observed excess leukaemia incidence which were not related to ionising radiation. These concerned chemicals, infectious agents and demographic factors. The fourth report (COMARE, 1996) devoted a chapter to the possible relationship of childhood leukaemia to infectious agents and the potential role of rural population mixing*.

7.4 The fourth report concluded that "it is probable that population mixing is a factor in the increase in childhood leukaemias described in some population groups. Therefore, it follows that the excess childhood leukaemia incidence in Seascale is likely to be causally associated, at least in part, with related demographic factors such as geographical isolation and mixing between residents who have migrated from different areas, or additional exposure to infections such as from sewage outflow". However, "the evidence, available at present, does not convince us that such a large relative risk persisting over more than three decades could be wholly attributed to population mixing". This chapter updates the evidence for such an association since publication of the fourth report.

7.5 Childhood leukaemia is the most common form of paediatric malignant disease in developed countries, accounting for around 30% of childhood cancer cases. Childhood leukaemia represents a spectrum of types, with acute lymphoblastic leukaemia (ALL) being around five times more frequent than

* The fourth report contained errors in relation to population mixing which have been detailed in Chapter 2 of this report.

acute myeloid leukaemia (AML), and of subtypes, with B-cell-precursor ALL (common ALL, cALL) being the most common subtype of childhood ALL. These types and subtypes of leukaemia may have different aetiologies.

7.6 Childhood leukaemia is a heterogeneous malignant disease and is likely to result from more than a single agent or pathway. A combination of genetic predisposition and environmental factors may be involved and it is known that certain medical conditions, such as Down's syndrome, increase the risk of childhood leukaemia. Onset is hypothesised to require the involvement of at least two events (Rossig and Juergens, 2008). For cALL, initiation is now understood to occur *in utero* (Marshall et al, 2014; Swaminathan et al, 2015), leading to a pre-leukaemic clone carrying a genetic lesion being present at birth. Postnatally, these clones may acquire further mutations, leading to leukaemic transformation. If infections are involved in the aetiology of leukaemia, they could act as either an initiating or a promoting agent or both.

7.7 It has been estimated that there were 1.9 million infection-attributable cancers worldwide in 2002, which accounted for 17.8% of the global cancer burden, with a higher proportion in developing countries (Parkin, 2006). A similar proportion (16.1%) of the number of new cancer cases in the world attributable to infectious agents was estimated by de Martel et al (2012). The discovery of more virus-related cancers and the role of cofactors may lead to an increase in this proportion (Kinlen, 2004). In the UK, around 3% of all cancers are presently thought to be attributable to infectious agents (Parkin, 2011). Most infections are due to viruses with seven classed as Group 1 human carcinogens by the International Agency for Research on Cancer (Chen et al, 2014): Epstein-Barr (EPV), hepatitis B (HBV), hepatitis C (HCV), Kaposi's sarcoma herpes (KSHV), human immunodeficiency virus type 1 (HIV-1), human T cell lymphotrophic virus type 1 (HTLV-1) and human papillomavirus (HPV). For example, oropharyngeal cancer was reported to have the greatest rate of increase of any cancer in Scotland, with the rise attributed to increasing HPV associated disease (Junor et al, 2010). However, most infected patients do not develop cancer. The estimated three-fold higher burden of infection-related cancers in developing compared with developed countries suggests that additional cofactors, including secondary infections, are important. A role for childhood infection and immune response in the aetiology of childhood leukaemia has been the subject of speculation for many decades.

7.8 The possibility that infections may influence the risk of leukaemia is given indirect support by a number of observations, some of which are summarised below before more detail is given on the rural population mixing hypothesis.

Childhood peak age

7.9 The early childhood peak in the incidence of leukaemia, with the highest incidence at around 2–6 years of age (Eden, 2010), is seen in most developed countries and is now emerging in middle income countries. In the UK and USA, the peak emerged during the first half of the twentieth century. Court Brown and Doll (1961) and Stewart and Kneale (1969) have noted the inverse correlation between this rise in the occurrence of leukaemia in young children and the decrease in mortality from pneumonia. This, they suggested, could be due to cases of leukaemia going unrecognised before the existence of effective treatments for infections (which are particularly common in the early stages of leukaemia). It has been argued that undetected cases of leukaemia could be responsible for the absence of the peak in some developing countries – for example, those in sub-Saharan Africa (IARC, 2003) – but it has also been hypothesised to be due to children from affluent populations living in conditions of good hygiene and with limited social contact and so in relative isolation from

infectious agents in early life (Greaves, 2006). The early childhood peak is due predominantly to cALL and this has given rise to proposed causes of cALL linked to infective processes. It has been hypothesised also that exposure to infections *in utero*, in particular the JC virus of the polyomavirus family, may lead to cALL in childhood (Smith, 1997; Smith et al, 1998).

Space-time clustering

7.10 Cases of infectious diseases which are transmitted from person to person often show evidence of clustering in space and time, as opposed to purely spatial clustering which is, generally, linked more closely to environmental factors, although it can also include infective causes. A large study based on data from all of Great Britain during 1969–1993 reported evidence that space-time clustering exists for ALL among young children (COMARE, 2006; McNally et al, 2006, 2009). Kreis et al (2016) found leukaemia-specific space-time clustering in Switzerland for cases diagnosed in children below 16 years of age between 1985 and 2010. These authors found that space-time clustering was stronger for place and time of birth rather than at diagnosis.

Social contact in early childhood

7.11 Given that the most common exposure to infections in infancy is through contact with other children, attendance at crèches or day-care has been investigated as a proxy marker for infections in early childhood. A UK study of 6305 children aged 2–14 years without cancer and 3140 children with cancer (1286 with ALL) demonstrated that social contact with other infants and children during the first few months of life reduced the risk of ALL (Gilham et al, 2005). A meta-analysis of studies assessing the association with day-care attendance showed a significantly reduced odds ratio of ALL for children who had attended day-care either before the age of 2 years, or anytime between birth to diagnosis (Urayama et al, 2010). Another, more recent, meta-analysis supported the hypothesis that stimulation of the early immune system, including attendance at day-care, protects against childhood ALL (Rudant et al, 2015). The evidence to date suggests that day-care attendance, with the associated exposure to common infections in early childhood, is protective against childhood ALL.

Infections during infancy

7.12 Studies continue to provide an unclear picture as to the possible role of infections in infancy in the development of childhood leukaemia. Chan et al (2002) showed a protective effect of infection in the first year, when accompanied by fever and rash, against ALL. A study in California found a significantly reduced odds ratio of childhood ALL in children who had an ear infection in infancy (Ma et al, 2005a), while a study in France found a protective effect of repeated common infections (Rudant et al, 2010). In contrast, children with leukaemia were found to have had more infections than controls when using GP-recorded data from the UK Childhood Cancer Study (Roman, 2007; Roman et al, 2009). A similar finding was made in the study of Cardwell et al (2008) using GP records of infection. A comparison between maternal reports of infections and GP records indicates under-reporting by mothers (Simpson et al, 2007; Roman et al, 2009). Greaves and Buffler (2009) have speculated that many infections in infancy are sub-clinical and would therefore not be apparent in either GP records or maternal recollections. Overall, there remains inconsistent evidence regarding the role of infections in infancy or early childhood on the risk of childhood leukaemia.

Infections during pregnancy

7.13 Smith's hypothesis (Smith, 1997) is that the childhood peak of ALL is due to *in utero* exposure to infections, particulary the JC virus. An increased risk of leukaemia has been reported among children whose mothers had a viral infection during pregnancy (Roman et al, 1997). Specific maternal infections during pregnancy linked to an increased risk of childhood leukaemia include Ebstein-Barr virus (EBV) (Lehtinen et al, 2003) and lower genital tract

infections (Naumburg et al, 2002). However, Tedeschi et al (2009) found no association with maternal EBVinfection. Overall, the evidence to date remains inconclusive.

Childhood vaccination

7.14 Several studies have shown a decreased risk of childhood leukaemia among children who have been vaccinated, both generally (McKinney et al, 1987; Schuz et al, 1999; Pagaoa et al, 2011) and against a range of infections, including the Haemophilus influenza type b-conjugate vaccine (Groves et al, 1999; Ma et al, 2005b), or BCG or measles vaccinations (Nishi and Miyake, 1989). However, there are also studies showing the opposite associations (Buckley et al, 1994; Dockerty et al, 1999) and a number of studies showing no significant effects (McNally and Eden, 2004; Mallol-Mesnard et al, 2007).

Breastfeeding

7.15 A number of studies have shown a protective effect of breastfeeding, particularly prolonged breastfeeding, on the risk of childhood leukaemia (Shu et al, 1999; Infante-Rivard et al, 2000; UK CCSI, 2001; Perrillat et al, 2002; Kwan et al, 2004; Bener et al, 2008; MacArthur et al, 2008). These studies may reflect the early exposure to infections and passive transfer of antibodies given through breastfeeding, although breastfeeding is also highly socioeconomically patterned, so may give rise to confounding. However, other studies have shown non-significant associations (in both protective and increased risk directions) (McNally and Eden, 2004).

Parental occupational contact

7.16 It has been suggested that parents having occupations with high levels of social contacts while at work (eg teachers) may expose their children to higher rates of infection than children of parents in occupations with fewer contacts. Some studies have shown increased rates of childhood leukaemia in the more highly exposed groups in rural areas (Kinlen, 1997; Kinlen et al, 2002; Pearce et al, 2004; Keegan et al, 2012), with evidence that the effect is for young children (0–4 years of age), while for the 5–14 year age group the association is in the opposite direction (Kinlen and Bramald, 2001; Kinlen et al, 2002). Broader studies including urban areas show no association at all (Fear et al, 1999, 2005).

7.17 A case–control study of occupational contact levels experienced by the parents of the 13 LNHL cases diagnosed before the age of 25 years and associated with Seascale has been reported by Kinlen (2015b). Up to 20 age- and sex-matched controls were selected for each case, although the actual numbers ranged from 8–19, the loss of controls mainly arising through the children moving from the village prior to the diagnosis of the matched case. In one case, diagnosis was at age 23 years and so the individual's own occupational contacts were deemed more important than those of the parents; the associated controls were treated similarly. In 11 cases, the father worked at Sellafield, as did the oldest case and most of the controls.

7.18 As in previous work (Kinlen and Bramald, 2001), each pre-diagnosis occupation of cases and controls was categorised into three groups based on estimated contact level – 'low, medium and unknown', 'high' and 'very high'. The higher contact level of either mother or father was used, except for the young adult and associated controls, whose own data were used. The analysis was carried out using conditional logistic regression, adjusting for social class.

7.19 Taking the 'low, medium and unknown' category as the reference, the odds ratio (OR) for the 'high' category was 8.18 (95% CI 0.95, 70.33) and for the 'very high' category was 14.90 (95% CI 1.20, 184.9); the p-value for trend was 0.024.

7.20 A supplementary analysis, based on more detailed information on the occupational contact levels, placed more cases into the 'very high' category. The ORs for the 'high' and 'very high' categories were then calculated as 2.96 (95% CI 0.24, 37.22) and 29.68 (95% CI 2.12, 415.79), respectively; the p-value for trend was 0.011.

7.21 Kinlen (2015a) concluded that high occupational contact levels are associated with LNHL risk in young people in Seascale and that the village is not an exception to the finding that contact levels are an important determinant in areas of marked rural population mixing.

Population mixing

7.22 The primary evidence for an infectious aetiology for childhood leukaemia has come from studies that were developed to test the hypothesis, proposed by Kinlen in 1988, of a role for population mixing in the aetiology of childhood leukaemia (Kinlen, 1988). This postulates that childhood leukaemia is a rare response to a common (but presently unidentified) infection (or possibly infections) and that when substantial mixing of infected and susceptible individuals occurs 'mini-epidemics' (often sub-clinical) of the pertinent disease(s) occur, leading to an excess of cases of the rare response, childhood leukaemia. Such mixing would occur when there is an influx of a large number of people from urban areas into a rural area, in particular into a remote area with a population that has not previously experienced much contact with urban populations.

7.23 Kinlen argued that this might provide the explanation for the excesses of childhood leukaemia near the two remote rural nuclear sites at Sellafield and Dounreay (Kinlen, 1988). The large initial influx of both professional and other workers, combined with an unusual level of migration and job turnover, has given these geographically isolated areas a highly unusual demographic pattern. In such situations, children may have been exposed to previously unencountered infections at a higher intensity than might otherwise have been the case.

7.24 Since the fourth COMARE report, Kinlen and Balkwill (2001) found a significant excess of childhood leukaemia mortality in a cohort of children resident in Orkney and Shetland during the Second World War, when large numbers of troops were stationed in these remote and isolated islands to protect against invasion. Kinlen (2006) has studied the impact of rural population mixing upon the whole of west Cumbria brought about by the construction and operation of three ordnance factories there during the Second World War. When the wider area containing the communities most affected by employment at the factories was considered, a significant excess of childhood leukaemia was found (although based on small numbers).

7.25 Boutou et al (2002) found evidence for population mixing increasing the risk of childhood leukaemia in rural communes near the La Hague and Flamanville nuclear installations in Normandy, France, brought about by workers coming into the area to work on construction projects at the sites.

7.26 In 2012, Kinlen published a meta-analysis of studies of childhood leukaemia, diagnosed at ages 0–14 years, in relation to rural population mixing (Kinlen, 2012). This included 17 studies, including the fourth COMARE report findings for Seascale during 1984–-1992, and the large overall sample size allowed also a detailed analysis by age group. Kinlen's is the most recent review, updating those of Little (1999) and McNally and Eden (2004), although the previous reviews considered a wider range of potential infectious aetiology routes rather than being limited to population mixing.

7.27 Kinlen (2012) split the studies into ‘marked rural population mixing’ and ‘marked urban population mixing’. This included seven studies that could be used in both categories as they had reported separately urban and rural results, as well as nine that were rural only and one that was urban only. Due to the different study designs used, Kinlen had to further sort the studies into those for which a comparison between the highest population mixing exposure category and national rates was made, and those for which the highest and lowest population mixing groups were compared.

7.28 For rural population mixing, all but one (the New Zealand forestry area study by Dockerty et al, 1996), of the ten studies showed an increased risk of childhood leukaemia in the highest population mixing groups when compared to national rates. The overall relative risk for this group of studies was 1.67 (95% CI 1.45, 1.93). All seven studies comparing low and high population mixing reported an increased risk of childhood leukaemia in the high population mixing group, with an overall relative risk of 1.57 (95% CI 1.44, 1.72).

7.29 For urban population mixing, neither of the two studies comparing with national rates showed an increased risk of childhood leukaemia in relation to high population mixing (overall relative risk 0.87 (95% CI 0.67, 1.14). Three of the six studies comparing highest and lowest population mixing showed an increased risk, but the highest individual relative risk was 1.30. The overall relative risk for this group was 0.99 (95% CI 0.93, 1.06).

7.30 Kinlen interpreted the difference in findings for urban and rural areas as being due to those living in urban areas having a higher level of herd immunity. The number of children in urban areas who, at any one time, had not encountered the relevant infectious agent(s) will be relatively small, so that the rare response (childhood leukaemia) to the infection will be at the level expected in the absence of epidemic conditions.

7.31 As Kinlen stated in his meta-analysis paper, the situations in which rural population mixing have occurred have varied considerably, including war-time evacuations from urban areas, military occupations, new towns and, as was the case for Sellafield and Dounreay, major construction projects that required large workforces and external expertise.

7.32 In terms of an infectious aetiology for childhood leukaemia, the primary alternative to Kinlen’s hypothesis is that proposed by Greaves (1988), which focuses on an abnormal immunological response to infection, rather than a specific infection, as a risk factor for childhood leukaemia.

7.33 Greaves has proposed that a ‘two hit’ model of leukaemogenesis could explain, *inter alia*, the emergence in developed countries of the cALL peak in young children where a ‘first hit’ occurs *in utero* leading to covert susceptibility to leukaemia that only becomes manifest when a ‘second hit’ occurs after birth. The ‘second hit’ is postulated to be an infection that triggers a pathological immune response leading to cALL, and a delay in exposure to the generality of infectious agents (due to factors such as increased hygiene or lack of contacts) increases the risk of this abnormal response because of the increased presence of ‘first hit’ cells in the immune system. Conversely, early exposure to infections will decrease the risk of cALL.

7.34 In his meta-analysis, Kinlen (2012) suggested that both his and Greaves' hypotheses supported the role of rural population mixing as a risk factor within the childhood peak, but differed mainly in the postulated mechanisms.

7.35 However, in his meta-analysis, Kinlen reported a similar association of childhood leukaemia with rural population mixing for under 2 year olds as for age groups later in childhood and pointed out that delayed exposure to infections is not likely to play a role for very young cases. Other evidence for a reduced risk of leukaemia among those children likely to have been exposed to infections early after birth (such as crèches and playgroups) is equivocal.

7.36 If it is supposed that Kinlen's hypothesis concerning the aetiology of childhood leukaemia is correct, then the features of the areas around Sellafield and Dounreay that may enhance the risk of childhood leukaemia need to be considered. These factors, and the relevant studies, were considered in the fourth COMARE report (COMARE, 1996), which concluded that the evidence then available "provides reasons to believe that some kinds of infective process may be associated with childhood leukaemia". Evidence accrued in the past 20 years is considered in more detail below.

7.37 Dickinson and Parker (1999) used the Cumbrian births database to develop a statistical model based on Poisson regression of the incidence of childhood leukaemia and non-Hodgkin lymphoma in relation to population mixing among children born during 1969–1989 to mothers living in Cumbrian wards, but excluding Seascale ward. Their measure of population mixing was based on residence at birth, rather than at diagnosis. This model was used to predict the number of cases that would have been expected in Seascale children born between 1950 and 1989, and diagnosed with cancer before 1993. The incidence of both ALL and NHL was significantly higher than expected among children born in areas with the highest levels of population mixing. They concluded that population mixing could account for the excess of childhood leukaemia in Seascale, but that additional risk factors could also be involved.

7.38 The same authors later studied leukaemia risk in the offspring of male radiation workers at Sellafield, incorporating the father's total preconceptional external radiation dose, but also the previously used measures of population mixing (Dickinson and Parker, 2002). They first compared the risk of leukaemia or NHL among children of male Sellafield radiation workers to that among children of fathers who had never worked at Sellafield. Children of radiation workers had a higher risk of leukaemia or NHL than other children (rate ratio, RR = 1.9, 95% CI 1.0, 3.1, p = 0.05]. Adjusting for population mixing greatly reduced the excess risk in Seascale, but had little effect on excess incidence rates elsewhere

7.39 In contrast to the evidence above, two studies from the UK found deficits in incidence associated with population mixing (Parslow et al, 2002; Law et al, 2003). Kinlen (2012) has pointed out, however, that these studies, and some similar studies, have not considered rural areas with striking population changes, but largely urban areas not known to be associated with recent large population changes. A recent census-based Swiss cohort study (Lupatsch et al, 2015) found no evidence to support the population mixing hypothesis, but Kinlen (2015a) has observed that no marked rural population mixing occurred in the areas studied and so the hypothesis could not be tested by this study. Lupatsch et al (2015) did find that, in rural areas, point estimates were suggestive of a positive association between leukaemia risk and population growth, particularly for leukaemia occurring before the age of 5 years; however,

statistical evidence for this association was weak.

7.40 In spite of the many studies that have considered the putative link between rural population mixing and an increased risk of childhood cancer (especially leukaemia), there has been no general consistency in the literature of the definitions used for 'population mixing' (Law et al, 2008). This is an important issue for the interpretation of findings related to population mixing, For example, on the basis of the Kinlen hypothesis the rare response to the pertinent common infection will only be detectable above the background process under epidemic conditions, when there are sufficient susceptible individuals encountering sufficient infected individuals. These circumstances are rare outside unusual population mixing in rural areas and so studies of urban population mixing or of unexceptional mixing in rural areas are unlikely to produce a discernible signal of the rare response.

7.41 COMARE commissioned a study to compare a number of different measures of population mixing defined in a study by Taylor et al (2008) using UK census data. Taylor and colleagues found that hospital admissions data on infections could be used as a proxy for level of community infections, that commuting distance was a consistent measure of population mixing as related to infectious disease and that both deprivation and population density were good proxies for infectious exposure. The investigators also noted that areas with high levels of population mixing do not necessarily have high rates of hospital admissions for infectious disease.

7.42 Given the availability of small-area population density over a prolonged time span, the temporal changes in key measures of population mixing in relation to Sellafield ('Seascale') and the surrounding areas ('Allerdale plus Copeland without Seascale' and 'Cumbria without Seascale') have been analysed.

7.43 The following measures of population mixing were used (see Taylor et al, 2008):

Migration – defined as a person moving their residential address into a ward in the study areas within 1 year of the census. Three separate types of migration-based population mixing were calculated, defined as:

Volume – ln (proportion of in-migrants within each ward)

Diversity – Shannon index (Shannon and Weaver, 1948) – measures the diversity of origins of in-migrants into each ward

Distance – ln (median distance moved by in-migrants to the ward)

Each measure was calculated for 'all ages' and, separately, for children aged '0–15 years'

Commuting – defined as the daily and/or regular movement of an individual from home to a work place outside their residential ward. Three separate types of commuting-based population mixing were calculated, defined as:

Volume – ln (proportion of commuters from each ward)

Diversity – Shannon index (Shannon and Weaver, 1948) – measuring the diversity of destinations of commuters from each ward

Distance – ln (median distance commuted by commuters from the ward)

Each measure is calculated for those aged '16–59 years' only

For both migration and commuting the above measures were repeatedly calculated for minimum distances migrated or commuted (in km).

Deprivation – measured by the Townsend index (Townsend et al, 1988) – a composite socioeconomic index derived from the standardised census based area proportions of unemployment, household overcrowding, car non-ownership and housing non-owner occupiers. Four separate components of the Townsend index were also analysed.

Population density – population weighted persons per km^2 within each geographical area. It was calculated for 'all ages', for ages '0–14', '15–24' and '0–24' years.

All measures were available for the 1981, 1991 and 2001 censuses. In addition, deprivation data were available for the 1971 census and population density data for the 1951, 1961 and 1971 censuses.

7.44 The results for the volume and distance migration measures of population mixing showed no great differences between the three areas, but the diversity migration measure (the Shannon index) for 'Seascale' was more than an order of magnitude greater than that for the other two areas. Similar results were obtained for the three commuting measures.

7.45 The deprivation results are shown in Table 7.1. Using the Townsend score, there was a trend towards decreasing deprivation between 1971 and 2001 in all three areas, but 'Seascale' has consistently remained the most affluent area with car ownership consistently much higher. There were no trends in unemployment for any area. For all three areas, there was a trend towards decreased levels of household overcrowding between 1971 and 2001, the lowest levels being seen consistently in 'Seascale'. For all three areas there was a trend towards higher levels of home ownership; interestingly, Seascale only achieved the highest levels of home ownership in 1991 and 2001 compared with the other areas, which is due to much of the housing being owned by the UKAEA in earlier times and subsequently being sold to private owners.

Table 7.1 Deprivation[a] summary

	1971	1981	1991	2001
Townsend deprivation				
Seascale	–1.19	–1.64	–3.95	–4.25
Copeland and Allerdale (excluding Seascale)	2.89	2.38	–0.03	–2.05
Cumbria (excluding Seascale)	1.69	1.37	–0.83	–2.70
Unemployment (%)				
Seascale	2.36	3.96	3.53	3.10
Copeland and Allerdale (excluding Seascale)	4.78	10.42	8.98	4.41
Cumbria (excluding Seascale)	3.49	8.56	6.95	3.45
Overcrowding (%)				
Seascale	1.64	0.94	0.46	0.49
Copeland and Allerdale (excluding Seascale)	6.32	3.17	1.29	0.76
Cumbria (excluding Seascale)	5.46	2.73	1.25	0.78
No cars (%)				
Seascale	19.51	15.83	12.12	10.06
Copeland and Allerdale (excluding Seascale)	48.82	38.87	32.43	25.61
Cumbria (excluding Seascale)	45.92	36.75	30.58	24.06
Home not owned (%)				
Seascale	56.64	47.74	18.94	18.56
Copeland and Allerdale (excluding Seascale)	55.86	50.16	34.50	31.00
Cumbria (excluding Seascale)	49.27	43.05	30.18	27.40

a Deprivation for Copeland and Allerdale (excluding Seascale) and Cumbria (excluding Seascale) is calculated from Townsend ward data with all age ward population as weights

7.46 The population density results are shown in Table 7.2. It can be seen that, for 'all ages' Seascale had markedly lower levels of population density than the other areas for all censuses. The same is true for the other age groups. A notable increase occurs between 1951 and 1961 for Seascale which is not reflected in the other areas.

Table 7.2 Population density[a] summary

Area[b,c]	1951	1961	1971	1981	1991	2001
All ages						
Seascale	20.8	27.6	25.8	22.9	20.7	20.5
Copeland and Allerdale minus Seascale	86.5	89.0	88.1	90.9	90.5	87.9
Cumbria minus Seascale	64.3	65.0	66.0	67.9	68.6	68.8
Age 0–14 years						
Seascale	5.0	7.1	6.2	4.2	3.4	3.4
Copeland and Allerdale minus Seascale	20.7	22.6	21.6	18.7	17.1	15.7
Cumbria minus Seascale	14.5	15.4	15.6	13.5	12.4	12.1
Age 15–24 years						
Seascale	2.6	4.0	3.8	3.9	2.4	1.5
Copeland and Allerdale minus Seascale	11.9	11.7	12.8	14.0	11.9	9.3
Cumbria minus Seascale	8.5	8.3	9.2	10.2	9.0	7.2
Age 0–24 years						
Seascale	7.7	11.1	10.0	8.1	5.7	4.9
Copeland and Allerdale minus Seascale	32.6	34.2	34.3	32.7	29.0	25.0
Cumbria minus Seascale	23.1	23.7	24.7	23.7	21.5	19.3

a Density is the number of people per square km

b Seascale is excluded from Copeland and Allerdale and from Cumbria

c Population figures for Copeland and Allerdale and Seascale for the years 1951, 1961 and 1971 estimated as for 2001 census boundaries

7.47 Unfortunately, it has not been possible to calculate the migration measures for 1951, 1961 or 1971 as these may be the parameters which would be of most interest. The adoption of the Taylor et al (2008) definition of migration within 1 year of the census may have been too limiting also. Nevertheless, the data demonstrate that Seascale has exhibited marked demographic differences from its surrounding area throughout the time period under consideration; taken together with the social class differences noted above, these indicate that the community would be susceptible to any sequelae of population mixing.

7.48 The use of these measures is unlikely to account fully for the quantity and quality of population mixing experienced in any community. Kinlen has emphasised the importance for any epidemic of an infectious disease of the numbers of susceptible and infected individuals in a community, and that rural population mixing may be very different from urban population mixing in this respect because many people in urban areas are likely to have encountered the pertinent infection if this is common. Of further relevance to Sellafield is the number of non-local contractors working at the site with whom resident workers would mix.

7.49 The prolonged duration of the Seascale childhood leukaemia excess may be viewed as a problem for the population mixing hypothesis. In its fourth report COMARE noted, however, that "Seascale has received a constant reintroduction of 'susceptibles', as a result of the unusually high level of inward and outward migration of high social class scientists and executives with their families in the early years. Continued population flux may explain why the incidence of leukaemia in Seascale has been raised for a long time. In summary, Seascale is extreme in terms of the length of time in which a high level of population mixing has occurred" (COMARE, 1996). The almost continuous presence of construction workers on the Sellafield site would also promote infection (Kinlen, 2011).

7.50 In considering the possible attribution of the leukaemia excess at Seascale to population mixing, it is of note that the last major construction project carried out at the Sellafield site which required a workforce numbering in the thousands (mostly from outside the area) was the THORP facility. This was built between 1983 and 1992. More recent, but smaller, construction projects have used a high proportion of local construction workers as the NDA is keen to have a high proportion of spend being directed to a more locally based community (Fisher, personal communication). Also of relevance is the increasing mobility of populations in recent decades, including rural populations, decreasing the likelihood of substantial groups of children remaining susceptible to infection by the relevant agent(s) and so reducing the chance of 'mini-epidemics' and an increase in the rare response.

7.51 As for Seascale, the area around Dounreay has experienced large-scale population changes, with many workers moving into the area to work at the nuclear site, particularly during the original construction in the mid-1950s. The population of the nearby town of Thurso increased by some 150% between 1951 and 1961. The Prototype Fast Reactor was built between 1968 and 1974 and was the largest subsequent construction project, involving 500–600 workers. Other projects, such as the construction of the Marshall Laboratory in the mid-1980s and the cementation plant at the end of the 1980s, used much smaller workforces (under 100), generally comprising locally mobilised labour.

7.52 An intriguing difference between the cluster of LNHL among young people in Seascale and that around Dounreay (centred on west Thurso) is that the former developed in the early 1950s, soon after the marked expansion of the village to meet the employment needs of Sellafield, whereas the latter commenced in 1979, some years after the first influx of nuclear workers. However, Kinlen et al(1993) pointed out that there was a five-fold excess of childhood leukaemia (ages 0–14 years) in Thurso in the 5 years after operations commenced at Dounreay in 1958, but this was based on just two cases and did not achieve statistical significance.

7.53 With the Dounreay area's proximity to the North Sea oil industry, rural population mixing and childhood leukaemia in the area were studied in the context of the effect of employment demands of the oil industry upon the whole of rural Scotland (Kinlen et al, 1993). A significant excess of LNHL was found during 1979–1983 in those home areas providing the largest proportion of oil workers, including the area around Dounreay. Kinlen et al (1993) interpreted these results as indicating that the excess of LNHL in young people in the area around Dounreay was part of a wider excess in rural areas of Scotland during 1979–1983 due to large numbers of men from these areas mixing with large numbers of workers from urban areas as a consequence of their employment in the oil industry and bringing new infections back to their home communities.

7.54 Since the fourth COMARE report (COMARE, 1996) a marked cluster of cases of childhood leukaemia near the Krummel nuclear power station in northern Germany has been reported (Hoffmann et al, 2007), which was considered in the fourteenth COMARE report (COMARE, 2011). The cluster consisted of 14 cases during 1990–2005, whereas 4.0 would have been expected on the basis of national rates, and the proportional excess was greatest for young children less than 5 years of age at diagnosis (observed =10, expected =2.04). Despite intense investigations, no explanation for the cluster has been forthcoming, including radiation exposure and population mixing.

7.55 An even more extreme cluster of cases of childhood leukaemia has occurred at Fallon in Churchill County, Nevada, where fourteen cases of childhood ALL were incident during 1997–2003, eleven of these during 1999–2001 when less than one case would be expected (Steinmaus et al, 2004; Francis et al, 2012). Steinmaus et al (2004) calculated that such marked clustering would only occur in the USA by chance alone about once in every 22,000 years. Fallon is geographically isolated and not close to any nuclear facility, but there is a large military airbase nearby that specialises in pilot training so that large numbers of military personnel pass through the base. The Fallon cluster continues to be the subject of investigation, with population mixing and the introduction of infectious agents having been proposed to be involved by Francis et al (2012), who also noted a statistically significant peak in the incidence of childhood ALL in 1999–2000 in families within the US armed services, that is, coinciding with the peak incidence in Fallon.

7.56 It is clear that, despite the strong epidemiological evidence pointing to an infectious aetiology for childhood leukaemia, the underlying mechanism still needs to be resolved through appropriate biological research. If, as suggested by Kinlen, childhood leukaemia is a rare response to a common infection(s), then this infective agent(s) needs to be identified. Similarly, if as suggested by Greaves, childhood leukaemia (strictly, cALL) results from a ‘two hit’ process, then the nature of these ‘hits’ requires elucidation.

7.57 Recently, Swaminathan et al (2015) have suggested that concurrent activation of the enzymes RAG1-RAG2 and AID could be a mechanism by which infection gives rise to clonal evolution towards cALL in childhood.

7.58 Candidate viruses have been investigated, but none has been firmly identified to date. For example, MacKenzie et al (1999) analysed the leukaemic cells of 15 children with cALL for the presence of the JC and BK polyomaviruses and found no positive result, indicating that these viruses were not involved in the development of childhood cALL. More recently, neonatal dried blood spots from 50 children who later developed ALL and from 100 control children were analysed for the presence of three newly discovered polyomaviruses, but these were not detected suggesting that infection *in utero* by these viruses is not involved in childhood ALL (Gustafsson et al, 2012). Waugh et al (2011) looked for the presence of a novel retrovirus, xenotropic murine leukaemia virus-related virus (XMRV), in DNA samples from 58 children with ALL (mainly cALL) and did not detect the presence of XMRV in any sample.

7.59 In another study, using representational difference analysis (RDA), MacKenzie et al (2006) analysed the DNA of 11 children with cALL, searching for differences between the genomes of leukaemic cells and of either granulocytes from the patient in remission or pooled parental samples, differences that could be due to the presence of viral genomes. Sequences of, or related to, viral genomes were not detected and there was no evidence of non-

human DNA in the difference products, which provides evidence against a directly transforming virus. However, the authors did point to certain limitations in the methodology – for example, viral genomes present to a similar extent in the comparison samples would escape detection by RDA (MacKenzie et al, 2006)). Kinlen (2015a) has noted that any study searching for the presence of potentially leukaemogenic viruses within the human genome should not – if using an unaffected control group for the purposes of comparison – include family members or people from urban areas, since these individuals could be infected if the virus is common.

7.60 In conclusion, there is now convincing epidemiological evidence for the involvement of infections in the aetiology of childhood leukaemia. There are two main hypotheses as to the underlying leukaemogenic process, that of Kinlen and that of Greaves; at present, there is insufficient evidence to conclusively favour either. Both are consistent with rural population mixing being an important factor contributing to the observed increased risks in the populations living in Seascale and around Dounreay, while not completely excluding a role for other causes.

CHAPTER 8

CONCLUSIONS

8.1 In this report, COMARE has reviewed the incidence of leukaemia, non-Hodgkin lymphoma (NHL) and other cancers among young people less than 25 years of age around the Sellafield and Dounreay nuclear installations, updating its previous work at the request of the Department of Health to fulfil the fifth recommendation of the eleventh COMARE report (COMARE, 2006). In addition, and in the light of new information on radioactive discharges, a semi-quantitative assessment of the radiation exposures of young people living in Seascale and in Thurso as a function of time from the start of operations until 2010 was undertaken to determine possible changes since earlier COMARE reports. The implications of the exposures on the risk of radiation-induced leukaemia and other cancers were also considered. In addition to radiation doses to the red bone marrow, this analysis specifically considered doses to the thyroid and the possible risks of radiation-induced thyroid cancer. The current evidence for the influence of paternal preconceptional irradiation and of rural population mixing upon the risk of childhood leukaemia was reviewed also.

8.2 The previously reported increased incidence of leukaemia and NHL among children (0–14 years of age) and young adults (15–24 years) resident in Seascale and around Dounreay between 1963 and 1990 has been confirmed; however, no significantly increased cancer incidence was found in more recent years (1991–2006) among either age group in either area.

8.3 There has been no new case of leukaemia or NHL among children, and a single new case of cancer (a lymphoid leukaemia) among young adults, living in Seascale ward during the period 1991–2006.

8.4 Among those individuals born in Seascale, the previously reported increased incidence of leukaemia and NHL among young people has been confirmed, but no currently increased incidence of cancer has been found among those born near Sellafield.

8.5 There has been no new case of leukaemia or NHL among either children or young adults resident in the area around Dounreay during the period 1991–2006.

8.6 For Dounreay, there is no evidence of an excess cancer risk in those born closest to Dounreay as compared to those born further away from the nuclear installation.

8.7 Difficulties encountered with current research governance regulations have prevented the Seascale schools cohort study being updated. It is a matter of regret that the Dounreay schools cohort dataset is no longer available.

8.8 The analysis of thyroid cancer incidence between 1974 and 2012 reveals a consistent excess in Cumbria for those born between 1929 and 1963, with the highest point value noted for those born between 1959 and 1963;

however, no excess is observed for those born after 1963. This result is consistent with earlier research showing an excess thyroid cancer incidence in Westmorland*, and an association between this county (and others) and benign thyroid diseases that pre-dated the Sellafield plant.

8.9 There is no conclusive evidence of an excess of thyroid cancers in those potentially exposed at a young age to radioactive releases, particularly iodine-131, from the Windscale reactor fire in 1957. Although 1954–1958 births show a particularly high excess rate in Cumbria, this is not as high as, or statistically different from that for 1959–1963 births (ie those born after the fire who would not have been exposed to iodine-131). The high thyroid cancer incidence rate for Cumbria for 1954–1958 births is not repeated for Lancashire.

8.10 It is recognised that there are limitations with a geographical study of thyroid cancer incidence and that iodine-131 contamination within Cumbria and Lancashire was not uniform. An analysis using the Cumbrian birth cohort would shed more light on these findings.

8.11 COMARE has reviewed recent publications on radiation dosimetry and risk, finding no reason to amend the internationally accepted approach to dose and risk estimation.

8.12 The only material changes to the dose estimates arise from the increases due to the revised transfer coefficient for polonium-210 (paragraph 5.31). Although there have been some relatively minor changes to the discharge data and revisions to other dose coefficients used in the risk assessments, these have not led to any overall significant changes to the estimated total doses or the trend in doses observed. As noted in paragraph 4.12, the NDA advised COMARE that it is still appropriate to assume that 10% of the dissolver inventory of iodine-131 was released at Sellafield and doses have been calculated on this basis. This is despite evidence from an analysis of available information that the percentage may have been significantly lower than this (Wakeford, 2007a); it may be possible to use environmental measurements of the long lived radionuclide iodine-129 to further elucidate this matter, but current data are inadequate.

8.13 The revised assessment of risk from radiation exposure at both sites concludes that the current best estimates of the radiation doses to both the Seascale and Thurso populations is much too small to account for the observed numbers of cases of leukaemia and NHL that occurred in the young people resident in these areas during 1963–1990.

8.14 Suggestions that serious errors are present in conventional radiation risk assessments have been examined and found to be lacking in evidence.

8.15 The absence of correlation between the incidence rates of leukaemia and NHL predicted on the basis of assessed radiation doses and the observed incidence rates at three different nuclear sites further supports the conclusion that radiation cannot be a major causal factor in these areas.

8.16 Following the reviews in its fourth and seventh reports (COMARE, 1996, 2002), COMARE has examined the subsequent evidence for an effect of irradiation causing increased risks of cancer in the offspring of fathers exposed before conception of the child, in particular whether such paternal

* Westmorland was one of the 39 historic counties of England. From 1974 the whole county was administered by the new administrative county of Cumbria.

preconceptional irradiation could account for the excess incidence of LNHL among young people in Seascale. Although there is evidence from cellular and animal studies that high doses of PPI can produce observable effects in future generations, no health effects have been detected conclusively in humans. Specifically, the epidemiological evidence for an effect of PPI in man continues to provide no support for a detectable risk to health, and the initial association between PPI and the risks of leukaemia and NHL among young people in Seascale reported by Gardner et al (1990b) appears most likely to have been a chance finding.

8.17 For this report, COMARE commissioned a study to compare a number of different measures of population mixing in relation to Seascale and the surrounding areas, which were derived from UK census data. The Seascale area consistently showed substantially lower levels of population density and deprivation than its surroundings. COMARE has corrected errors made in its fourth report in relation to population mixing around Sellafield and has considered more recent publications. In addition, publications relating to population mixing around Dounreay have been reviewed.

8.18 Given the highly unusual conditions experienced in Seascale and around Dounreay, it is likely that infectious agents are responsible, at least in part, for the excesses of LNHL among young people there (and in those communities where other marked clusters of cases have been reported).

8.19 In light of these data, COMARE believes that the hypothesis that marked rural population mixing can increase the risk of childhood leukaemia, and that the disease is likely to have an infectious aetiology, should be given greater credence and be the subject of greater research effort.

8.20 In the report of the Independent Advisory Group (Black, 1984) a recommendation was made to coordinate centrally the monitoring of small area health statistics to provide an early warning of any untoward health effect. This resulted in the establishment in 1987 of the Small Area Health Statistics Unit (SAHSU), presently located at Imperial College, London. One of the functions of SAHSU is to identify localised clusters of cases of disease, enabling unusual aggregations of cases of childhood leukaemia to be investigated rapidly, including the timely collection of environmental and biological samples, which would be desirable in the investigation of the potential role of infectious (and other) agents in the clustering. The findings of the eleventh COMARE report suggest that clusters of cases of childhood leukaemia occur more frequently than would be expected by chance alone. Any notable cluster of childhood leukaemia that is detected in this way should be thoroughly investigated with due urgency to provide evidence of possible causes.

CHAPTER 9

RECOMMENDATIONS

In this report, COMARE has updated its previous work, reviewing the incidence of leukaemia, non-Hodgkin lymphoma and other cancers in young people around the Sellafield and Dounreay nuclear installations and the risk assessments for the radiation exposure of young people living in Seascale and in Thurso as a function of time from the start of operations until 2010. This review also specifically considered radiation doses to the thyroid and the possible risks of radiation-induced thyroid cancer, together with the current evidence for risks arising from paternal preconceptional irradiation and rural population mixing.

Recommendation 1

COMARE continues to accept that, in the past, there were demonstrable excesses of leukaemia and non-Hodgkin lymphoma among young people in Seascale and around Dounreay. Although these increased risks are no longer detectable in the most recent period studied, COMARE acknowledges that it is impossible to conclude that excess cases will not occur in the future. Therefore, we reiterate to the Department of Health and the Scottish Government the recommendation made in our eleventh report (COMARE, 2006: recommendation 5, page 131) that the incidence of leukaemia, non-Hodgkin lymphoma and other cancers in the vicinities of Sellafield and Dounreay be kept under surveillance and periodic review (at least every 10 years).

Recommendation 2

COMARE holds concerns regarding the current UK research governance regulations preventing the updating of epidemiological studies. These apply to the wider research community, but have already impeded COMARE's work. We recommend that appropriate UK authorities review this aspect of the regulations. In particular, we recommend that further investigations should be undertaken by DH to establish a route whereby a follow-up study of the Seascale schools cohort might be carried out to support the cancer analysis presented in this report.

Recommendation 3

In respect of thyroid cancer, a review of possible geographical variation in ascertainment rates should be undertaken. We also recommend that an epidemiological study in relation to thyroid doses from Sellafield discharges, in particular the 1957 Windscale accident, should be carried out using the Cumbrian birth cohort, along with investigation of other areas where excesses of thyroid cancer have been reported.

Recommendation 4

COMARE has noted in a number of reports that it is widely recognised that the initiation and progression of normal white cells into leukaemic cells is not a straightforward process and that a variety of factors have been proposed as causes of leukaemia, together with a number of hypotheses for potential mechanisms. We recommend the continuation of initiatives into leukaemia and cancer research to identify the causative mechanisms for childhood leukaemia. In this respect, COMARE notes the strong epidemiological evidence for an infective cause of childhood leukaemia, but that the mechanism underlying these epidemiological findings has yet to be identified. We recommend an intensified research effort to investigate the role of infection in childhood leukaemia, including virus studies with appropriate controls.

Recommendation 5 We recommend that the apparent discrepancies between animal and human studies of the transgenerational effects of exposure to ionising radiation should be investigated further.

Recommendation 6 We recommend that the NDA and Sellafield Ltd review the data on dissolver discharges. This should include further work as necessary on environmental measurements of iodine-129, particularly in relation to historical discharges. Further investigation of the modelling of doses due to polonium-210 discharges should be undertaken also.

Recommendation 7 We reiterate recommendation 3 of our fourteenth report (COMARE, 2011: page 103) regarding the continuation of effective and comprehensive environmental surveillance around these and other nuclear sites in the UK. This includes the use of modern sensitive measurement techniques to assess the residual presence in environmental media of radionuclides from historical, as well as current, discharges.

Recommendation 8 On the basis of the evidence detailed in Chapter 7 for the influence of rural population mixing upon the risk of childhood leukaemia, we recommend that prospective studies be made of the incidence of childhood leukaemia in rural areas in which any large-scale construction projects (both non-nuclear and nuclear) are to be carried out, which require an influx of large numbers of construction workers. Further, should any striking excesses of childhood leukaemia be reported from any part of the UK, these should be the subject of a detailed investigation.

Recommendation 9 The UK currently has one of the highest quality cancer registration systems in the world, which provides the capability to carry out comprehensive epidemiological analyses of cancer incidence data. As in previous reports, COMARE wishes to highlight serious concerns about possible changes to the way in which data relating to cancer incidence are collected. Changes already proposed could mean that patients would have the right to opt out of the cancer registry. Also, the use of new information sources to build the cancer registry dataset may remove data items, such as postcodes, which may be vital to the type of research described in this report.

COMARE is concerned particularly with the transfer of data held in the previous National Registry of Childhood Tumours (NRCT) database on to the national database now administered by PHE (especially the pre-1985 segment). The complete NRCT database has been fundamental to a number of COMARE reports and it is important that the entire database is transferred intact (including its validation data) to allow future analyses to be carried out. We therefore recommend that these and other UK-wide resources, which allow such studies to be carried out in both children and adults, should continue to be specifically maintained and kept up to date.

Recommendation 10 We wish to recommend that government and the devolved administrations collaborate to ensure that an adequate dataset and research governance (access/analysis) arrangements are shared throughout the UK in respect of cancer registries. Care should be exercised to prevent data loss when new procedures or IT systems are introduced.

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APPENDIX A

GLOSSARY AND ABBREVIATIONS

ABSORBED DOSE The quantity of energy imparted by ionising radiation to a unit mass of matter such as tissue. Absorbed dose has the units J kg^{-1} and the specific name gray (Gy), where 1 Gy = 1 joule per kilogram

ACTINIDES A series of 15 radioactive elements with increasing atomic numbers beginning with actinium (89) and ending with lawrencium (103). Many of them decay by the emission of alpha particles. Some can also decay by spontaneous fission or can be made to undergo fission by bombardment with neutrons and are therefore used as nuclear fuels. Only four of the actinides – actinium, thorium, protactinium and uranium – occur in nature in significant quantity; the remaining 11 are produced artificially by bombardment of other related elements with high energy particles

AETIOLOGY The causes of disease

ALL See LEUKAEMIA

ALPHA EMITTER A radionuclide which decays through emission of alpha particles

ALPHA PARTICLE A charged particle emitted during the radioactive decay of many heavy radionuclides. It is identical to the nucleus of a helium-4 atom, consisting of two protons and two neutrons. An alpha particle has low penetrating power, but high linear energy transfer (LET)

AML See LEUKAEMIA

BACKGROUND RADIATION Radiation that comes from naturally occurring radioactive material in the ground and from cosmic rays irradiating the Earth from the sun and outer space. The UK average effective dose from background radiation is 2.2 millisievert (mSv) per year: regional averages range from 1.5–7.5 mSv per year

BECQUEREL (Bq) A unit of radiation equal to one disintegration per second. Discharges are normally expressed in:

megabecquerels (MBq) – one million Bq

gigabecquerels (GBq) – one thousand million Bq

terabecquerels (TBq) – one million million Bq

BETA EMITTER A radionuclide which decays through emission of beta particles

BETA PARTICLE A particle emitted from a nucleus during the radioactive decay of certain types of radionuclides. It has a mass and charge similar to that of an electron. It has greater penetrative power than an alpha particle, but is low linear energy transfer (LET) radiation

CANCER REGISTRATION In England and Wales, a formally coordinated, but non-statutory, scheme whereby all cases of cancer should be notified to regional registries, in agreed detail, as soon as possible after diagnosis. Coordination is undertaken by the National Cancer Registration Scheme. The data are forwarded to the Office of Population, Censuses and Surveys (OPCS) for collation and publication

CARCINOMA A malignant tumour that may spread to surrounding tissue and distant areas of the body

CASE–CONTROL STUDY A retrospective study in which the potential risk factors for a group of individuals identified as having the disease, the cases, are compared to those for a group of individuals not having the disease, the controls

CENSUS The enumeration of an entire population, usually with details being recorded on residence, age, sex, occupation, ethnic group, marital status, birth history, and relationship to head of household

CHROMOSOMES Rod-shaped bodies visible in the nucleus of cells at the time of cell division. They contain the genes. Normal human cells possess 22 identical pairs plus two sex chromosomes in each cell

CLL See LEUKAEMIA

CLUSTERING The irregular grouping of cases of disease in time (where cases of a particular disease which might normally occur at a fairly constant rate in a community appear with unduly high frequency in a certain time period); space (where cases of a particular disease occurring within a certain time period tend to cluster in a well-defined location); or in both time and space (where cases that occurred close together in space would tend also to be close in time, eg in the aetiology of some rare diseases such as leukaemia)

COHORT STUDY Cohort studies are designed to answer the question, ‘What are the effects of a particular exposure?’ They compare a group of individuals with the exposure under consideration to a group without the exposure, or with a different level of exposure. The groups (cohorts) are followed over a period of time, and the disease occurrence is compared between the groups or between the cohort and rates expected from national statistics

COLLECTIVE DOSE Collective dose is a measure of the total amount of effective dose multiplied by the size of the exposed population. Collective dose is usually measured in units of person-sievert or man-sievert

COMMITTED EFFECTIVE DOSE The sum of the products of the committed organ or tissue-equivalent doses and the appropriate organ or tissue weighting factors integrated over 50 years for adults and 70 years for children

CONFIDENCE INTERVAL (CI) An interval calculated from the data to indicate the (im)precision of an estimate of some parameter, eg the risk of a disease. A CI conveys the effect of sampling variation on the precision of the estimate. Specifically, the true rate will lie inside a 95% CI on 95% of occasions. This ‘confidence coefficient’ is often chosen to be 95%, although this is entirely arbitrary

CONFIDENCE LIMIT (CL) A quantity calculated from the data to indicate a limit below (or above) which a parameter is unlikely to lie, in the sense that in a stated proportion of such calculations (say 97.5%), the calculated limit will be less (or greater) than the true value. Two such limits form a (95%) confidence interval

CONFOUNDING	Confounding is a problem in epidemiological studies which arises when there is a factor associated with both the exposure being investigated and the disease under study. This can give rise to an apparent relationship between the factor being investigated and the disease, even though the factor did not cause the disease. For example, suppose lung cancer was being studied in workers exposed to a particular chemical. If those exposed to higher levels of the chemical smoked more than other workers, then the chemical would be associated with lung cancer even if it did not actually cause the disease. The problem can be addressed in the design and analysis of studies, but requires that data on the confounder be collected and analysed appropriately
DECAY (RADIOACTIVE)	The process of spontaneous transformation of a radionuclide. The decrease in the activity of a radioactive substance
DECAY PRODUCT	A nuclide or radionuclide produced by decay. A product may be formed directly from a radionuclide or as a result of a series of successive decays through several radionuclides
DECOMMISSIONING	Removal of a facility (eg reactor) from service
DNA (DEOXYRIBONUCLEIC ACID)	The compound that controls the structure and function of cells and is the material of inheritance. The DNA molecule consists of two polynucleotide chains in the form of a double helix. The chains are linked by pairs of DNA bases, the order of which is the genetic code
EFFECTIVE DOSE	The effective dose is the sum of the equivalent doses received by organs or tissues throughout the body with each equivalent dose multiplied by the tissue weighting factor, w_T, appropriate for that organ or tissue. The effective dose is designed to produce the same risk of stochastic health effects as a uniform whole-body absorbed dose of reference low LET radiation. The unit of effective dose is the sievert (Sv). The effective dose is used in the context of radiological protection for exposure to low doses or low dose rates of radiation
EFFLUENT	A discharge of liquid waste, as from a factory or nuclear plant
EPIDEMIOLOGY	The study of factors affecting health and illness of populations, regarding the causes, distribution and control
EPIGENETIC	A heritable change in the properties of a cell that is not due to a mutation in DNA**.** It usually reflects an alteration in the degree of expression of a gene. Epigenetic changes are not permanent and may be unstable
EQUIVALENT DOSE	The equivalent dose is the absorbed dose to an organ or tissue with each component type of radiation contributing to the absorbed dose multiplied by the appropriate radiation weighting factor, w_R. For example, the w_R for alpha particles is 20, while the w_R for gamma rays is 1. The unit of the equivalent dose is the sievert (Sv), so that for alpha particles, 1 Gy = 20 Sv. The equivalent dose is used in the context of radiological protection for exposure to low doses or low dose rates of radiation
EXCESS ABSOLUTE RISK (EAR)	The excess absolute risk (EAR) is the increase in the risk of a disease due to a particular exposure above the background risk of that disease in the absence of that exposure

EXCESS RELATIVE RISK (ERR)	The excess relative risk (ERR) is the proportional increase in the risk of a disease due to a particular exposure relative to the background risk of that disease in the absence of that exposure
EXPECTED NUMBERS	The average number of events or cases that will occur in a specified location and time period if overall mortality or incidence rates apply to that location and time period
EXTERNAL AND INTERNAL EXPOSURES	External exposure arises from radioactive sources which remain outside the body. Internal exposure arises from radioactive materials which are taken inside the body, through inhalation or ingestion or penetration of the skin. An alpha particle has a very short range and hence very little penetrative power, so that if it were to come from an external source it would be unlikely to penetrate the surface of the skin, giving up most of its energy in the dead surface skin layers. If, however, an alpha particle were emitted from a source that had been inhaled into the lungs its closer proximity to living cells could result in damage to those cells. Internal exposures are generally received from sources that have been inhaled or ingested. Beta and gamma radiation sources can give rise to either internal of external exposures
FERTILE	(Of a nuclide) capable of absorbing a neutron to produce a fissile nuclide. An example of a fertile nuclide is uranium-238, which on absorption of a neutron produces uranium-239, beta decaying to neptunium-239, beta decaying to plutonium-239, a fissile nuclide
FISSILE	(Of a nuclide) capable of absorbing a thermal (low energy) neutron and undergoing nuclear fission
FISSION	The splitting of a heavy nucleus into two (or more) parts, accompanied by the release of a relatively large amount of energy and generally one or more neutrons. It may be spontaneous, but is usually due to a nucleus absorbing a neutron
FISSIONABLE	(Of a nuclide) capable of absorbing a neutron and undergoing nuclear fission
GAMMA RAYS	High energy photons, without mass or charge, emitted from the nucleus of a radionuclide following radioactive decay, as an electromagnetic wave. They are very penetrating, but have a low Linear Energy Transfer (LET)
GENE	Unit of hereditary material arranged into linear sequence to form chromosomes, each gene occurring at a specific point. Composed of DNA having unique base sequence. See DNA
GERM LINE	Cell responsible for genetic continuity from one generation to the next. Primary germ cells (gonia) divide ultimately to form sperm or ova (gametes) which fuse with one another at the moment of fertilisation (conception)
GEOGRAPHICAL (ECOLOGICAL) STUDY	An epidemiological study in which the frequency of disease (or death) is observed in different areas and the locations of these areas are then related to putative sources of risk of the disease. In effect, the location and other attributes of the area are imputed to the cases without any possibility of distinguishing between them at an individual level
GONAD	Organ (testis or ovary) in which germ cells reside

GRAY (Gy)	The international (SI) unit of absorbed dose: one gray is equivalent to one joule of energy absorbed per kilogram of matter such as body tissue
HALF-LIFE (t ½)	The time taken for the activity of a radionuclide to lose half its value by decay**.** During each subsequent half-life its activity is halved again so its activity decays exponentially
HAZARD	A property that in particular circumstances could lead to harm, eg exposure to radiation leading to damage to an individual's health
HYPOTHESIS	A suggested explanation for a group of facts or phenomena. See also null hypothesis
ICRP	International Commission on Radiological Protection. It consists of experts in radiology, genetics, physics, medicine and radiological protection from a number of countries. Established in 1928, it meets regularly to consider the results of research on the effects of radiation and publishes recommendations on all aspects of radiological protection, including dose limits for man
INCIDENCE	The number of instances of illness commencing, or of persons falling ill, during a given period in a specified population. More generally, the number of new events, eg new cases of disease in a defined population, within a specified period of time. The term incidence is sometimes used to denote 'incidence rate', ie the number of cases divided by the (average) number at risk in the relevant time period
INFECTIOUS AETIOLOGY	The process by which disease is brought about by a transmissible agent, eg a virus
IONISATION	The process by which a neutral atom or molecule acquires or loses an electron. The production of ions
IONISING RADIATION	Radiation which is sufficiently energetic to remove electrons from atoms in its path. In human or animal exposures, ionising radiation can result in the formation of highly reactive particles in the body (known as free radicals) which can cause damage to individual components of living cells and tissues. The term includes radiation at least as energetic as X-rays; gamma rays and charged particles such as alpha and beta particles are also forms of ionising radiation
ISOTOPE	Nuclides containing the same number of protons (ie same atomic number), but different numbers of neutrons

LEUKAEMIA

A group of malignant diseases of the blood-forming tissues in which normal control of cell production breaks down and the cells that are produced are abnormal. Leukaemia can be classified as lymphoid or myeloid and as either acute or chronic (eg ALL, AML, CLL and CML). Lymphoid and myeloid refer to the type of white cell affected. If this is a lymphocytic cell the condition is called lymphocytic or lymphoblastic leukaemia. Myeloid leukaemias affect any of the other types of white blood cells or the red cell or platelet producing cells. Acute leukaemias develop quickly and progress rapidly; chronic leukaemias are slower to develop and slower to progress. Acute lymphoblastic leukaemia (ALL) is subdivided into three types using the French- American-British classification of:

L1 small monotonous lymphocytes

L2 mixed L1- and L3-type lymphocytes

L3 large homogeneous blast cells

Each subtype can be further classified by immunophenotyping, with two main immunological types: pre-B-cell and pre-T-cell. The mature B-cell ALL (L3) is now classified as Burkitt's lymphoma/leukaemia. Subtyping helps determine the prognosis and most appropriate treatment for ALL

LINEAR ENERGY TRANSFER (LET)

A measure of the density of ionisation along the track of an ionising particle in biological tissue or other medium. Particles or rays of radiation are generally described as having a high or low LET, ie their tracks leave high or low density deposits of energy in the tissue they pass through. High LET radiation is more damaging to body tissue than low LET radiation

LINEAR RISK SCORE (LRS)

A test statistic designed to determine whether a group of cases are closer to a particular point (such as a nuclear power plant) than would be expected given the population distribution in the area. It simply scores each case with a suitable measure of proximity, such as the reciprocal of distance, and adds the scores over all cases, comparing this with the value that would be expected for a random sample from the population

LYMPHOMA

A malignant tumour of the lymphatic system (lymph nodes, reticuloendothelial system and lymphocytes)

MALIGNANT

Synonymous with cancerous. Malignant neoplasms or tumours can invade and destroy other tissues and spread to other parts of the body via the bloodstream or lymphatics (metastasis)

META-ANALYSIS

A statistical analysis used to combine the results of several studies addressing a set of related research hypotheses, usually conducted to pool findings and incorporate information from small studies with low power. It can test whether the study outcomes show more variation than expected owing to population differences and different study designs

MUTAGEN

An agent (eg radiation or chemical) that can induce mutations

MUTATION

The changing of the structure of a gene through the alteration of single base units in DNA, or the deletion, insertion, or rearrangement of larger sections of genes or chromosomes, resulting in a variant form which may be transmitted to subsequent generations. Certain specific mutations are involved in the process leading to malignant disease. Mutations occur spontaneously and may be induced by various agents (mutagens)

NEOPLASM	An abnormal growth of tissue in animals or plants, such as a tumour. Neoplasms can be benign or malignant
NON-HODGKIN LYMPHOMA (NHL)	A group of lymphomas that differ in important ways from Hodgkin's lymphoma and are classified according to the microscopic appearance of the cancer cells. In children, NHL and leukaemias are often combined due to historical difficulties in making diagnostic distinctions
NUCLEAR REACTOR	An engineering construction in which a nuclear fission chain reaction occurs under controlled conditions so that the heat yielded may be harnessed or the neutron beam used
NUCLEAR FUEL REPROCESSING	The chemical processing of spent irradiated fuel from a nuclear reactor, to remove radioactive fission products and to recover fissile and fertile material for further use. Chemical solvents play a major role in this process
NULL HYPOTHESIS	The statistical hypothesis that one variable has no association with another variable or set of variables, or that two or more population distributions do not differ from one another
P-VALUE	The probability that, under a given null hypothesis, a particular test statistic would have, purely by chance, a value at least as disparate with the hypothesis as that observed. A P-value provides an idea of the strength of the evidence against the null hypothesis. A low P-value points to rejection of the null hypothesis. For a significance test at the 5% level, any result giving a P-value less than 0.05 would be regarded as significant and lead to rejection of the null hypothesis in favour of an alternative hypothesis. Its interpretation depends on the plausibility of available alternative hypotheses or explanations
PATERNAL PRECONCEPTION IRRADIATION (PPI)	Exposure of the father to ionising radiation prior to conception of offspring. A hypothesis suggests that PPI may produce radiation-induced mutations in the germ line, resulting in a predisposition to leukaemia or NHL in the next generation
PLUTONIUM	A transuranic radioactive element: plutonium-239 is fissile, has a half-life of 24,100 years and is produced in a nuclear reactor by the capture of a neutron by uranium-238, forming uranium-239 which beta decays to neptunium-239, which then beta decays to plutonium-239
POISSON DISTRIBUTION	The Poisson distribution is a probability distribution describing the numbers of events happening independently of one another, eg the number of cancers within an area. The mean and variance of counts that follow the Poisson distribution are equal
POPULATION MIXING	The population-mixing hypothesis proposes that childhood leukaemia can be a rare response to a common, but unidentified infection (hence the absence of marked space–time clustering). Epidemics of this (mainly sub-clinical) infection are supposedly prompted by influxes of people into rural areas, where susceptible individuals are more prevalent than the average. Such influxes would increase population density and hence the level of contacts between susceptible and infected individuals, thereby increasing the risk of childhood leukaemia
PRECONCEPTIONAL EFFECT	An event such as a mutation occurring in a germ cell before the moment of conception (fertilisation), ie while still in the parental gonad

PROBABILITY A measure of how likely an unpredictable event is to occur on a given occasion. Mathematically it is measured on a scale of zero to one, which may be expressed as a percentage. Its usefulness in statistics stems from the fact that it can be estimated from the proportion of corresponding outcomes in repetitions of the same experimental or observational situation, and this estimation becomes more precise as the number of repetitions increases

RADIATION The emission and propagation of energy by means of rays or waves or sub-atomic particles

RADIONUCLIDE A type of atomic nucleus which is unstable and which may undergo spontaneous decay to another atom by emission of ionising radiation (usually alpha, beta or gamma)

REGRESSION COEFFICIENT The slope of the straight line that most closely relates two correlated variables

RELATIVE BIOLOGICAL EFFECTIVENESS (RBE) The RBE of one radiation compared with another is the inverse ratio of the absorbed doses producing the same degree of a defined biological effect

RELATIVE RISK (RR) Ratio of the risk of disease or death among those exposed to a potential hazard to the background risk of the disease or death in the absence of that potential hazard

RETINOBLASTOMA A common childhood malignancy of the eye that develops from retinal cells

RISK A combination of the probability, or frequency, of occurrence of a defined hazard and the magnitude of the consequences of the occurrence. See HAZARD and RELATIVE RISK.

Risk is sometimes taken to mean the probability that an event will occur, eg that an individual will become ill or die within a stated period of time or age. Risk is also used as a non-technical term encompassing a variety of measures of the probability of a (generally) unfavourable outcome

SIEVERT (Sv) The international (SI) unit of effective dose obtained by weighting the equivalent dose in each tissue in the body with ICRP-recommended tissue weighting factors and summing over all tissues. Because the sievert is a large unit, effective dose is commonly expressed in millisievert (mSv) – ie one-thousandth of one sievert. The average annual effective radiation dose received by members of the public in the UK is around 2.7 mSv

SIGNIFICANCE TEST A formal procedure for assessing the evidence against a null hypothesis, specified in advance. The formal version results either in rejection of the null hypothesis in favour of some alternative, or in its acceptance. A test is associated with a ‘significance level’, which is the probability that this rejection would occur by chance when the null hypothesis is true. Typically this significance level is chosen to be 5%, but the choice is entirely arbitrary. In a less formal version of the significance test a p-value is calculated. Data that result in the rejection of a hypothesis at a given significance level, or equivalently in a p-value less than such a level, are described as being ‘statistically significant’ at this level

SOCIO-DEMOGRAPHIC	A population variable relating either to intrinsic properties of an area, such as population density, or to the average of some personal characteristic of the inhabitants, such as age, socioeconomic status or degree of household overcrowding
SOCIOECONOMIC STATUS	A measure related to levels of living or social class. It may apply to individuals or groups. In this report it is applied to the populations of census wards or county districts, and is based on information from the 1981 census
SPECIFIC ACTIVITY MODEL	A straightforward method of calculating doses from atmospheric releases of and carbon-14. The basic assumption is that the radionuclide is in equilibrium with the stable form of its element. For example, with tritium the concentration of the radionuclide in water taken into the body by ingestion and inhalation is assumed to be the same as that in atmospheric water vapour
SPERMATOGENESIS	The formation and maturation of spermatazoa in the testis
STANDARDISED INCIDENCE RATIO (SIR)	The ratio of the actual number of cases in a study group or population to the expected number. The expected number is calculated using the age- and sex-specific incidence rates for a reference population. These 'reference rates' will often be those of the national population, but may also be taken from a smaller area
STANDARDISED MORTALITY RATIO (SMR)	The standardised incidence ratio for the deaths in a study group or population
STEM CELL	An undifferentiated cell that gives rise to specialised cells, such as blood cells
TRANS-GENERATIONAL EFFECT	An effect in the offspring resulting from exposure of a parent to some risk factor
TREND	The tendency for the values of a variable to increase or decrease as some other variable – most commonly time – changes
URANIUM	A naturally occurring radioactive element with two long-lived isotopes, uranium-238 (99.28% of uranium found on Earth) and uranium-235 (0.72%). Uranium-233 and uranium-235 are fissile isotopes. Uranium is the basic raw material of nuclear energy

APPENDIX B

TABLES REPRODUCED FROM THE TENTH COMARE REPORT

Table B1 Results for leukaemia and non-Hodgkin lymphoma in 1969–1993 in 25 km regions around nuclear installations in Great Britain (data taken from Table 2.2)

Site (start-up year)	Operator	Number of wards[a]	Observed (O)	Expected (E)	O/E (SIR)	Test choice[b]	p-value[c]
Dounreay (1959)	UKAEA	5	9	3.87	2.324	1	0.014
Sellafield (1950)	BNFL and UKAEA	32	25	21.95	1.139	2	0.018

a Excluding wards with zero population under 15 years

b The tests selected were as follows:

(1) LRS test using 1/(ward distance) as a score

(2) LRS test using the square root of 1/(ward rank) as a score

c p-value using chosen (unconditional) test, based on 10,000 simulations

Table B2 Results for solid tumours in 1969–1993 in 25 km regions around nuclear installations in Great Britain (data taken from Table 2.5)

Site (start-up year)	Operator	Number of wards[a]	Observed (O)	Expected (E)	O/E (SIR)	Test choice[b]	p-value[c]
Dounreay (1959)	UKAEA	5	3	6.29	0.477	3	0.868
Sellafield (1950)	BNFL and UKAEA	32	40	35.96	1.112	2	0.177

a Excluding wards with zero population under 15 years

b The tests selected were as follows:

(1) LRS test using 1/(ward distance) as a score

(2) LRS test using the square root of 1/(ward rank) as a score

(3) Poisson maximum test

c p-value using chosen (unconditional) test, based on 10,000 simulations

APPENDIX C

REPORTS OF THE COMMITTEE ON MEDICAL ASPECTS OF RADIATION IN THE ENVIRONMENT

Sixteenth report	Patient radiation dose issues resulting from the use of CT in the UK. PHE, Chilton, August 2014
Fifteenth report	Radium contamination in the area around Dalgety Bay. PHE, Chilton, May 2014
Fourteenth report	Further consideration of the incidence of childhood leukaemia around nuclear power plants in Great Britain. HPA, Chilton, May 2011
Thirteenth report	The health effects and risks arising from exposure to ultraviolet radiation from artificial tanning devices. HPA, Chilton, June 2009
Twelfth report	The impact of personally initiated X-ray computed tomography scanning for the health assessment of asymptomatic individuals. HPA, Chilton, December 2007
Eleventh report	The distribution of childhood leukaemia and other childhood cancer in Great Britain 1969–1993. HPA, Chilton, July 2006
Tenth report	The incidence of childhood cancer around nuclear installations in Great Britain. HPA, Chilton, June 2005
Ninth report	Advice to Government on the review of radiation risks from radioactive internal emitters carried out and published by the Committee Examining Radiation Risks of Internal Emitters (CERRIE). NRPB, Chilton, October 2004
Eighth report	A review of pregnancy outcomes following preconceptional exposure to radiation. NRPB, Chilton, February 2004
Seventh report	Parents occupationally exposed to radiation prior to the conception of their children. A review of the evidence concerning the incidence of cancer in their children. NRPB, Chilton, August 2002
COMARE and RWMAC* joint report	Radioactive contamination at a property in Seascale, Cumbria. NRPB, Chilton, June 1999
Sixth report	A reconsideration of the possible health implications of the radioactive particles found in the general environment around the Dounreay nuclear establishment in the light of the work undertaken since 1995 to locate their source. NRPB, Chilton, March 1999
Fifth report	The incidence of cancer and leukaemia in the area around the former Greenham Common Airbase. An investigation of a possible association with measured environmental radiation levels. NRPB, Chilton, March 1998

* Radioactive Waste Management Advisory Committee.

Fourth report	The incidence of cancer and leukaemia in young people in the vicinity of the Sellafield site, West Cumbria: further studies and an update of the situation since the publication of the report of the Black Advisory Group in 1984. Department of Health, London, March 1996
COMARE and RWMAC* joint report	Potential health effects and possible sources of radioactive particles found in the vicinity of the Dounreay nuclear establishment. HMSO, London, May 1995
Third report	Report on the incidence of childhood cancer in the West Berkshire and North Hampshire area, in which are situated the Atomic Weapons Research Establishment, Aldermaston and the Royal Ordnance Factory, Burghfield. HMSO, London, June 1989
Second report	Investigation of the possible increased incidence of leukaemia in young people near the Dounreay nuclear establishment, Caithness, Scotland. HMSO, London, June 1988
First report	The implications of the new data on the releases from Sellafield in the 1950s for the conclusions of the Report on the Investigation of the Possible Increased Incidence of Cancer in West Cumbria. HMSO, London, July 1986

* Radioactive Waste Management Advisory Committee.

APPENDIX D

COMMITTEE ON MEDICAL ASPECTS OF RADIATION IN THE ENVIRONMENT

Chairman

Professor A Elliott BA PhD DSc CPhys FInstP FIPEM ARCP (*Chair until April 2015*)
University of Glasgow

Dr C Gibson BA MSc PhD CSci FIPEM (*Chair from November 2015*)
National School of Healthcare Science

Present members

Dr J Barrett BSc MB ChB FRCP FRCPE FRCR OBE
Clinical oncologist

Dr P Darragh MD PhD MSc FRCP FFPHM
Public Health Agency for Northern Ireland, Belfast

Dr F de Vocht BSc Ir MSc PhD
School of Social and Community Medicine, University of Bristol

Professor J Harrison BSc PhD FSRP
Faculty of Health and Life Sciences, Oxford Brookes University

Professor B Howard MBE
Centre for Ecology and Hydrology, Lancaster Environment Centre

Professor P Marsden MSc PhD FSRP MIPEM MInstP CRadP
UCL Hospitals NHS Foundation Trust, London

Dr C Martin BSc PhD FinstP FIPEM FSRP CRadP
University of Glasgow

Professor S McKeown MA PhD FRSB CBiol
School of Biomedical Sciences, University of Ulster, Coleraine

Dr T Nunan MD FRCP FRCR
Nuclear medicine physician

Professor M Pearce BSc MSc PhD
Institute of Health and Society, Newcastle University, Newcastle upon Tyne

Professor K Prise BSc PhD
Centre for Cancer Research and Cell Biology, Queen's University Belfast

Mr I Robinson BSc CRadP FSRP FNucI
Consultant on nuclear and radiation safety (formerly HM Superintending Inspector of Nuclear Installation, Office for Nuclear Regulation)

Professor R Taylor MA FRCPE FRCP FRCR
School of Medicine, Swansea University

Dr M Toledano BA MSc PhD DLSHTM DIC FHEA
School of Public Health, Imperial College

Professor R Wakeford BSc PhD CSci CPhys FInstP CStat CEng MNucI CRadP HonFSRP
Institute of Population Health, University of Manchester

Professor P Warwick BA MSc PhD DSc CChem FRSC
Centre for Environmental Studies, Loughborough University

Professor Catharine West BA PhD
University of Manchester

Former members who served during preparation of this report

Dr J Bithell BA MA DPhil
Childhood Cancer Research Group, Oxford

Professor W Evans MA PhD FInstP FIPEM HonMRCR MBE
University Hospital of Wales, Cardiff

Professor T Helleday BSc MSc PhD
Gray Institute for Radiation Oncology and Biology, Oxford

Professor S Hodgson BM BCh DM FRCP
Department of Clinical Development Sciences, St George's University of London

Professor P Hoskin
Mount Vernon Cancer Centre, Northwood

Professor P Jeggo BSc PhD
Genome Damage and Stability Centre, University of Brighton

Professor M Kadhim PhD
Department of Biological and Medical Sciences, Oxford Brookes University

Dr G Maskell MA FRCP FRCR
Department of Radiology, Royal Cornwall Hospital, Truro

Secretariat

Dr S Mann BSc DPhil CEng MIET (Scientific)

Dr E Petty BSc PhD (Scientific)

Dr K Broom BSc DPhil CBiol FRSB (Scientific)

Ms K Stonell (Minutes)

Ms J Humphries (Administrative, until April 2015)

Mrs S Deacon (Administrative, from April 2015)

Assessors in attendance representing the following organisations	Department for Children, Schools and Families
	Department for Communities and Local Government
	Department of Energy and Climate Change
	Department for Environment, Food and Rural Affairs
	Department of Health
	Department of Health, Social Services and Public Safety (Northern Ireland)
	Department for Innovation, Universities and Skills
	Environment Agency
	Food Standards Agency
	Health and Safety Executive
	Medical Research Council
	Ministry of Defence
	Nuclear Decommissioning Authority
	Office for National Statistics
	Public Health England
	Public Health and Intelligence, NHS National Services Scotland
	Scottish Environment Protection Agency
	Scottish Government
	Welsh Government

SELLAFIELD AND DOUNREAY REVIEW SUBCOMMITEE

Chairman

Professor M Pearce BSc MSc PhD
Institute of Health and Society, Newcastle University, Newcastle upon Tyne

Members

Professor A Elliott BA PhD DSc CPhys FInstP FIPEM ARCP
University of Glasgow

Mr R Black MA
National Information and Intelligence, NHS National Services Scotland

Mrs K Bunch MA
Formerly of Childhood Cancer Research Group, Oxford

Professor S Hodgson BM BCh DM FRCP
Department of Clinical Development Sciences, St George's University of London

Mrs J Simmonds BSc
Formerly of Public Health England

Professor R Wakeford BSc PhD CSci CPhys FInstP CStat CEng MNucI CRadP HonFSRP
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Public Health England

APPENDIX E

DECLARATION OF MEMBERS' INTERESTS CODE OF PRACTICE

1 Introduction

This code of practice guides members of COMARE as to the circumstances in which they should declare an interest in the course of the Committee's work. To avoid any public concern that commercial interests of members might affect their advice to Government, Ministers have decided that information on significant and relevant interests of members of its advisory committees should be on the public record. The advice of the Committee frequently relates to matters which are connected with the radiation industry generally and, less frequently, to commercial interests involving radioactivity. It is therefore essential that members should comply with the code of practice which is set out below.

2 Scope and definitions

This code applies to members of COMARE and its subcommittees, subgroups, working groups and working parties which may be formed.

For the purposes of this code of practice, the 'radiation industry' means:

(a) companies, partnerships or individuals who are involved with the manufacture, sale or supply of products processes or services which are the subject of the Committee's business. This will include nuclear power generation, the nuclear fuel reprocessing industry and associated isotope producing industries, both military and civil and also medical service industries;

(b) trade associations representing companies involved with such products;

(c) companies, partnerships or individuals who are directly concerned with research or development in related areas;

(d) interest groups or environmental organisations with a known interest in radiation matters.

This excludes government departments, professional bodies, international organisations and agencies.

It is recognised that an interest in a particular company or group may, because of the course of the Committee's work, become relevant when the member had no prior expectation this would be the case. In such cases, the member should declare that interest to the Chairman of the meeting and thereafter to the Secretariat.

In this code, 'the Department' means the Department of Health, and 'the Secretariat' means the secretariat of COMARE.

3 Different types of interest – definitions

The following is intended as a guide to the kinds of interests which should be declared. Where a member is uncertain as to whether an interest should be declared they should seek guidance from the Secretariat or, where it may concern a particular subject which is to be considered at a meeting, from the Chairman at that meeting. Members of the Committee and the Secretariat are under no obligation to search out links between one company and another, for

example where a company with which a member is connected has a relevant interest of which the member is not aware and could not reasonably be expected to be aware.

If members have interests not specified in these notes but which they believe could be regarded as influencing their advice they should declare them to the Secretariat in writing and to the Chairman at the time the issue arises at a meeting.

3.1 Personal interests

A personal interest involves current payment to the member personally. The main examples are:

(a) *Consultancies and/or direct employment:* any consultancy, directorship, position in or work for the radiation industries which attracts regular or occasional payments in cash or kind.

(b) *Fee-paid work:* any work commissioned by those industries for which the member is paid in cash or kind.

(c) *Shareholdings:* any shareholding in or other beneficial interest in shares of those industries. This does not include shareholdings through unit trusts or similar arrangements where the member has no influence on financial management.

(d) *Membership or affiliation:* any membership role or affiliation that the member or close family member has to clubs or organisations with an interest or involvement in the work of the Department. This will not include professional bodies, organisations and societies.

3.2 Non-personal interests

A non-personal interest involves current payment which benefits a department to which a member is responsible, but is not received by the member personally. The main examples are:

(a) *Fellowships:* the holding of a fellowship endowed by the radiation industry.

(b) *Support by industry:* any payment, other support or sponsorship by the radiation industry which does not convey any pecuniary or material benefit to a member personally but which does benefit their position or department, eg:

(i) a grant from a company for the running of a unit or department for which a member is responsible;

(ii) a grant or fellowship or other payment to sponsor a post or a member of staff in a unit or department for which a member is responsible. This does not include financial assistance for students, but does include work carried out by postgraduate students and non-scientific staff, including administrative and general support staff;

(iii) the commissioning of research or work by, or advice from, staff who work in a unit or department for which a member is responsible.

(c) *Support by charities and charitable consortia:* any payment, other support or sponsorship from these sources towards which the radiation industry has made a specific and readily identifiable contribution. This does not include unqualified support from the radiation industry towards the generality of the charitable resource.

(d) *Trusteeships:* where a member is trustee of a fund with investments in the radiation industry, the member may wish to consult the Secretariat about the form of declaration which would be appropriate.

3.3 Specific interests

A specific interest relates explicitly to the material, product, substance or application under consideration by the Committee.

A member must declare a personal, specific interest if they currently receive a payment, in any form, for any significant fundamental development work undertaken previously or at this time, on a material, product or substance or its application under consideration. This will include the production of radioactive substances and devices designed to use ionising or non-ionising radiation for diagnostic, treatment or other purposes.

A member must declare a non-personal, specific interest if they are aware that the department to which they are responsible currently receives payment for significant fundamental development work undertaken previously or at this time, on a material, product or substance or its application under consideration but they have not personally received payment for that work in any form. This will include the production of radioactive substances and devices designed to use ionising or non-ionising radiation for diagnostic, treatment or other purposes.

3.4 Non-specific interests

A non-specific interest relates to a company or associated material, product, substance or application, but not to the specific material, product, substance or application under consideration by the Committee.

A member must declare a personal, non-specific interest if they have a current personal interest with a material, product, substance or application from a particular company, which does not relate specifically to the material, product, substance or application from that company under consideration.

A member must declare a non-personal, non-specific interest if they are aware that the department to which they are responsible is currently receiving payment from the company concerned which does not relate specifically to a material, product, substance or application under discussion.

If a member is aware that a material, product, substance or their application under consideration is or may become a competitor of a material, product or substance manufactured, sold or supplied by a company in which the member has a current personal interest, they should declare their interest in the company marketing the rival material, product or substance.

Members are under no obligation to seek out knowledge of such work done for or on behalf of the radiation industry within departments to which they are responsible if they would not reasonably expect to be informed. This applies to all non-personal, specific and non-specific interests.

4 Declaration of interests

4.1 Declaration of interests to the Secretariat

Members should inform the Secretariat in writing when they are appointed of their current personal and non-personal interests and annually in response to a Secretariat request. Only the name of the company (or other body) and the nature of the interest is required; the amount of any salary, fees, shareholding, grant, etc, need not be disclosed. An interest is *current* if the member has a continuing financial involvement with the industry, eg if they hold shares in a radiation company, have a consultancy contract, or if the member or the department to which they are responsible is in the process of carrying out work for the radiation industry. Members are asked to inform the Secretariat at the time of any change in their personal interests, and may be invited to complete a form of declaration when required. It would be sufficient if changes in non-personal interests are reported at the next annual declaration following the change. (Non-personal interests involving less than £5000 from a particular company in the previous year need not be declared.)

The register of interests should be kept up-to-date and be open to the public.

4.2 Declaration of interests at meetings and participation by members

Members are required to declare relevant interests at Committee meetings and to state whether they are personal or non-personal interests. The declaration should include an indication of the nature of the interest.

(a) If a member has a current (personal or non-personal) interest in the business under discussion, they will not automatically be debarred from contributing to the discussion subject to the Chairman's discretion. The Chairman will consider the nature of the business under discussion and of the interest declared (including whether it is personal or non-personal) in deciding whether it would be appropriate for the relevant member to participate in the item.

(b) If a member has an interest which is not current in the business under discussion, this need not be declared unless not to do so might be seen as concealing a relevant interest. The intention should always be that the Chairman and other members of the Committee are fully aware of relevant circumstances.

A member, who is in any doubt as to whether they have an interest which should be declared, or whether to take part in the proceedings, should ask the Chairman for guidance. The Chairman has the power to determine whether or not a member with an interest shall take part in the proceedings.

If a member is aware that a matter under consideration is or may become a competitor of a product, process or service in which the member has a current personal interest, they should declare the interest in the company marketing the rival product. The member should seek the Chairman's guidance on whether to take part in the proceedings.

If the Chairman should declare a current interest of any kind, they should stand down from the chair for that item and the meeting should be conducted by the Deputy Chairman or other nominee if the Deputy Chairman is not there.

Members' declarations of interests – 2015

Member	Company	Personal interest	Company	Non-personal interest
Dr J Barrett		None		None
Dr P Darragh		None		None
Dr F de Vocht	EPRI	Consultancy		None
Prof A Elliott		None		None
Prof W Evans		None		None
Dr C Gibson		None		None
Prof J Harrison		None		None
Dr B Howard		None		None
Prof P Marsden		None		None
Dr C Martin	1 Aberdeen Radiation Protection Services 2 Aurora Health Physics Services Ltd	Consultancy Consultancy		None
Prof S McKeown		None		None
Dr T Nunan		None		None
Prof M Pearce		None		None
Prof K Prise		None		None
Mr I Robinson	AMEC	Consultancy		None
Prof R Taylor		None		None
Dr M Toledano		None	1 DH MTHR programme 2 RIHMT	Principal investigator Principal investigator
Prof R Wakeford	Compensation Scheme for Radiation-linked Diseases	Consultancy		None
Prof P Warwick	1 Sellafield Ltd/Golder 2 NNL/LLWR Ltd	Contract Consultancy	NDA	Grants
Prof C West		None		None