Ministry of Defence

Policy Statement on Claims for Ionising Radiation Related Conditions

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Overview

1. This statement sets out the Department's policy on deciding claims for war pensions where service- related ionising radiation exposure is alleged to have caused disablement or death. It also provides the reasoning and evidence on which the policy is based. Situations where claims covered by the policy would be expected to arise include: participation in the UK atmospheric nuclear tests in the Pacific and Experimental Weapons Programme in South Australia, or the subsequent clean-up operations, prisoners of war held near Nagasaki or Hiroshima during the Second World War, accidents on board nuclear submarines, employment as an industrial or medical radiographer.

2. War pension may be claimed for any disablement by anyone who has served in the British Armed Forces before 6 April 2005. Claims may be made at any time from service release. Decisions are medically certified and evidence-based and each case determined on its individual merits. Taking into account the burden and standard of proof applicable to the claim, entitlement may be certified where the service and medical facts and the contemporary medical understanding of the condition claimed show a causal link between service and the claimed condition. For ionising radiation cases, entitlement will be considered where there is reliable evidence of service exposure to ionising radiation and there is a recognised causal link between the claimed condition and such exposure.

3. **Service-related ionising radiation exposure** will be accepted where, as a result of service, there is exposure to a measurable level of ionising radiation as determined by a radiological dosimetry specialist report and derived from direct measurement or estimate.

4. The statement considers general aspects of ionising radiation and the evidence on its adverse health effects including cancer, haematological malignancies, circulatory disorders and cataract. There is a section on military studies from the US, Australia and New Zealand, and because most war pension claims for disorders attributable to ionising radiation relate to the UK atmospheric military tests and Australian Weapons Experiment Programme (Minor Trials), there is detailed discussion of the three NRPB reports. This independent epidemiological study, begun in the mid-1980s, compared rates of mortality and incidence of cancers in a cohort of test participants and a carefully-matched service control group serving around the same time. The mortality rates in test participants were also compared with those of men of the

same age born in the same period from the general UK population.

5. It is not accepted as a matter of course that those present at UK atmospheric nuclear test detonations, or the Australian Weapons Experimental Programme, and clean-up operations, were exposed to harmful levels of ionising radiation as a result of service in these locations in the Armed Forces.

6. The present review has led to:

a) an investigation of the contemporary evidence on the link between ionising radiation and cancer. This has led to a list of malignancies recognised as radiogenic by the MOD.

b) an investigation of the link between radiation exposure and haematological disorders, circulatory disorders and cataract.

c) a review of the radiological protection and dosimetry arrangements at the Nuclear Test and Experimental Weapons Programme and identification of circumstances where participating personnel are accepted as being at particular risk of significant radiation exposure.

d) a statement of the Departmental policy on war pensions claims for solid cancers, haematological malignancies, circulatory disorders and cataract due to alleged service-related ionising radiation.

e) A number of background annexes:

- Annex A on radiation dose, internal radiation, radiological protection, tissue and probabilistic effects of ionising radiation exposure and the concept and calculation of probability of causation.
- Annex B on UK atmospheric nuclear tests.
- Annex C on ionising radiation and circulatory disorders and cataract.
- Glossary.

The Law – How the scheme works

7. For claims made not later than seven years after leaving the Armed Forces, Article 40 of the Service Pensions Order (2006) provides that the onus is on the Secretary of State to show beyond a reasonable doubt that the claimed disablement is not attributable to, or aggravated by, service, or that death was not due to, or hastened by, any such condition. If he cannot show this, entitlement to war disablement pension or war widow/er's pension, as appropriate, may be made.

8. For claims made more than seven years after the end of service, Article 41 of the Service Pensions Order (2006) puts the onus on the claimant to raise, by way of reliable evidence, a reasonable doubt that the claimed condition is attributable to, or aggravated by, a service injury or that death was due to, or substantially hastened by, an attributable injury or the aggravation by service of an injury. If he does so, entitlement to war pension will be certified.

9. About 21,000 UK servicemen participated in the UK nuclear tests and Minor Trials and the largest number of claims relate to presence at these operations. Because the adverse health effects of ionising radiation can take a long time to become apparent, most claims are made more than seven years after service termination, and Article 41 of the Service Pensions Order (2006) applies. This means that the onus is on the claimant to raise a reasonable doubt by reliable evidence that the claimed disablement is attributable to service.

Case Law

10. The High Court has held that the word "reliable", in the context of Article 5 (Article 41 in the 2006 Order), cannot have been intended to mean "convincing", but means "more than fanciful". A High Court Judge held that, with particular reference to "changes of medical opinion" that "there are... in my judgement, three stages: no reasonable doubt, reasonable doubt, and consensus." A war pensions claim under Article 5 would pass the test at the point where the (reliable) evidence raised a reasonable doubt, but: "a mere hypothesis based on a limited study.... would not have created a 'reasonable doubt' within the terms of Article 5(4) (Article 41(5) in the 2006 Order)."The real question, however, the judgement held, "is whether the evidence raises a reasonable doubt in the mind of the Secretary of State (SofS). If he finds the evidence unreliable, it obviously will not raise a reasonable doubt in his mind." (case of Edwards 1992 HCJ no. CO/2281/90).

11. In October 2014, the President of the Upper Tribunal Administrative Chamber clarified the "reasonable doubt" test under Article 41(5). It is for the claimant to establish by reliable evidence, "the possibilities that he asserts found the existence of that doubt". The decision-maker must then identify the claimant's evidence and arguments, go on to do the same for the respondent and consider any additional matters which need to be addressed. He must then carry forward these possibilities and matters upon which he has no reasonable doubt, i.e. effective certainties, and assess them in the round to determine whether or not, combined, they have met the Article 41(5) test. If the combination is too far-fetched the test will not be met.

12. The Courts have also held that a conflict of medical opinion does not, of itself, mean that a reasonable doubt has been established, and that a claim must therefore succeed. This applies irrespective of the eminence or authority of those expressing the opinions. In the case of Tigg v The Minister of Pensions, the presiding Judge stated: "Merely because a doctor of eminence, and I have no doubt the doctor in this case was of very great eminence, is expressing a view contrary to the view expressed by the medical witnesses called on behalf of the Ministry, does not mean there is a doubt and the Appellant must therefore be entitled to a pension. It is a question of fact for the Tribunal." (cases of Tigg ROSWPA vol.5 p.141 and Howard ROSWPA vol.5 p.515).

Evidence-based policy and individual decisions

13. Successive governments have held that in matters of public compensation regard must be paid to contemporary medical and scientific understanding

of causation and progress of disorders. In assessing any new approach in science, the evidence must always be considered and weighed relative to the existing body of evidence on a subject, with account taken of the robustness and authority of new studies. Attention must be paid to the design and methods, sample size, case and control selection, statistical validity, repeatability of findings, approach to bias and possible confounding factors. Other important factors include whether the findings have been replicated by other independent researchers and the overall plausibility/consistency relative to contemporary understanding.

Concepts in Ionising Radiation – Background

14. Exposure to ionising radiation in all its forms is part of being alive. "Ionising radiation" is radiation of sufficient energy to displace electrons from atoms and includes cosmic rays, gamma rays, X-rays, alpha and beta particle emissions. Levels of "natural" background radiation vary throughout the world depending mainly on the geology of the underlying earth. In the UK there is a range of values with average natural background radiation of about 2.2 mSv, half due to radon with contributions from cosmic rays, gamma radiation and internal radiation. For the UK, the addition of man-made radiation, predominantly through medical investigation, adds another 0.5 mSv average exposure per annum. Fallout from nuclear weapons testing, use and accidents accounts for 0.3% annual individual radiation dose. By the age of 70 the average UK citizen will have absorbed about 150 mSv of radiation from the natural background (1).

15. Human organs and tissues vary in their sensitivity to ionising radiation and the different types of ionising radiation have different capacities to cause cellular damage and adverse health effects. Other factors include age at exposure, with children and young people having a higher risk of cancer than those exposed at older ages. Direct evidence of damage and adverse health effects, including cancer at doses less than 100 mSv annually, is lacking and it is not known if effects are different when delivered in a single dose or over time. The International Commission on Radiological Protection (ICRP) in its most recent publication maintains that for radiological protection a Linear No Threshold (LNT) model is appropriate to estimate risk at acute or chronic annual dose below 100 mSv. The LNT model assumes no safe level of radiation, risk is proportional to dose and risk for multiple small exposures is equivalent to or less than that for a single acute exposure of the same energy (2). The ICRP confirms uncertainties in the processes involved in radiation tumorigenesis, particularly at low dose and relies mainly on the Japanese atomic bomb survivor high dose studies in calculating risk at low dose. Risk estimates inevitably represent a range. When expressed as a single value, the risk estimate is the most likely value derived from the distribution curve. The 2005 BEIR report predicts that if 100 Americans were exposed to 100 mSv either acutely or over time, one person would develop cancer due to radiation and 42 others would develop a cancer due to other factors (3).

16. The 'atomic' bombs dropped on Hiroshima and Nagasaki in 1945 produced a large and rapid energy release through a chain reaction ('fission') of the heavy nuclei of uranium 235 (Hiroshima) and plutonium 239 (Nagasaki). More

powerful ‘thermonuclear’ devices, detonated in the UK atmospheric nuclear tests in the 1950’s, were two-stage weapons. These relied on the fusion of isotopes of hydrogen, occurring at the very high temperatures created within an initial “atomic” nuclear fission reaction.

17. There are three sources of radiation exposure associated with atmospheric nuclear tests and weapons trials.

- the initial burst at the time of detonation – “prompt radiation“
- the activation products which result when neutrons from the nuclear reaction are mixed with soil in the area around detonation
- fallout i.e. radioactive material, including fission and activation products and unused fuel, falling to earth from the fireball

18. External exposure is produced by all three sources while internal exposure derives from inhalation, and to a lesser extent ingestion from hands to mouth. Absorption of radioactive material through broken skin can also arise and result in internal exposure.

19. External radiation is relatively easy to detect, monitor and quantitatively assess and most epidemiological studies on adverse health effects focus on it and report the effects of low Linear Energy Transfer external radiation (LET). All radioactive types and particles can be sources of internal radiation, but for the UK nuclear test and Minor Trials, alpha radiation from unspent uranium and plutonium is particularly important. Alpha particles are heavy and slow-moving, losing energy quickly with a short range (known as high Linear Energy Transfer (LET) radiation) and in contrast to external radiation, are unable to penetrate the outer layers of skin. At Annex A is a note on **radiation dose, internal radiation and alpha radiation, radiological protection and the tissue and probabilistic effects of ionising radiation and the concept and calculation of probability of causation**.

The adverse health effects of ionising radiation

20. Evidence that ionising radiation can cause human cancer and, more recently, other disorders has come from several sources. These include, most importantly, the Japanese atomic bomb survivor studies (4) and other high-dose external radiation studies on patients therapeutically irradiated for malignant conditions, such as cancer of the cervix (5) and non-malignancies like ankylosing spondylitis (6). More recently large cohort studies, notably of radiation workers with protracted low-dose external radiation exposures have been published, including pooled studies with data from several countries combined (7) (8) (9). Other evidence comes from internal exposure studies including low LET exposures to radioactive iodine (10) and high LET studies involving radon (11) and radium (12), and from follow-up of radiation workers with high levels of internal exposure to plutonium at the Mayak PA facility in the Russian Federation (13) and from studies of emergency and clean-up workers following the Chernobyl accident (14).

21. Since 2003 there have also been major reviews of the evidence of the adverse

health risks of ionising radiation exposure in the UNSCEAR (2006) (15a) (15b), BEIR (2006) and ICRP (2007) reports, as well as a series of reports by the Advisory Group on Ionising Radiation of the NRPB, subsequently Health Protection Agency (HPA) and now Public Health England (PHE), reviewing the published peer- reviewed evidence on leukaemias and related haematological malignancies (16), solid cancers (17) and circulatory disease (18). UK radiation protection legislation is based on the ICRP recommendations, and in 2009 the HPA published a response to the 2007 ICRP recommendations (19). This acknowledged the potential impact of emerging concepts like genomic instability, bystander signalling and adaptive response, but concluded that understanding of these and their effect on cancer induction and development was not yet well enough advanced to alter existing cancer risk data. Rather, the risk estimates should continue to be based on human epidemiological studies using the LNT model.

22. From 2001-2004 a committee including independent scientific experts, members of NRPB and the nuclear industry as well as anti-nuclear activists was set up to examine the radiation risk of internal emitters (CERRIE) and to consider risk models for health effects. Interpretation of the epidemiological studies on internal emitters is difficult because information is limited, with few studies having individual dose estimates, and findings are inconsistent. However, based on studies of lung cancer in radon-exposed miners, bone cancer in radium-exposed workers, and liver cancer in patients injected with thorotrast, the majority of the committee concluded that risk estimates were consistent with external dosimetry studies and that there was no evidence that risks from internal emitters were significantly underestimated. CERRIE considered that more work was required on internal emitters, but that dose and dose risk estimates from internal and external sources should be combined "using ICRP 2007 methodology for equivalent and effective dose and risk estimates" (Annex A). In response to the report ICRP 103 agreed. The NRPB (now PHE) also endorsed this position, provided risk estimates include an appreciation and explicit statement of the uncertainties involved (20) (21).

23. The Japanese atomic bomb survivor studies are an especially valuable source of information on adverse health effects of ionising radiation. Open studies began in 1947 with the establishment of the Atomic Bomb Casualty Commission (ABCC), and the cohort has now been followed for 65 years. The group received almost exclusively whole-body external radiation. Of the 120,000 original subjects, 54,000 were within 2.5 km of the epicentre of the detonations and 45,000 were located 2.5-10 km away where levels of ionising radiation were low. 26,000 controls were residents of Hiroshima or Nagasaki between 1951 and 1953 but had not been present at the detonations. Individual dose estimates are available for 92% of the population. The study population is of varied ages and exposures, and was not selected by diagnosis or occupation. 40% are still alive, including 80% of those exposed when aged less than 20 years. A sub-population, oversampled for those with high dose exposure, forms the Adult Health Study. This was established in 1958 with biennial health examinations and an on-going high participation rate. The 2004 ICRP publication 99 on low-dose extrapolation of radiation-related cancer risk includes a table of distribution of "estimated radiation dose among the atomic bomb

survivor cohort." Of the over 79,000 total, almost 24,000 were more than 3 km from the epicentres and were estimated to have received no radiation exposure; 10,000 had less than 5 mGy; 30,000, 5-100 mGy and fewer than 500, more than 2 Gy. The mean dose was 200 mGy. These features allow reliable estimates of excess relative risk for cancers and other health effects (22).

24. The main focus of study in the atomic bomb survivors is mortality and cancer incidence, although more recent papers cover non-cancer outcomes, e.g. lens opacities, thyroid and circulatory disease. A detailed overview to 2011 shows that of 17,448 new solid cancers in more than 100,000 subjects (1958-98), 853 are estimated to be due to radiation. About 75% of the cohort were exposed to doses between 5 and 200 mGy. The proportion of total solid cancers, or attributable fraction, in those exposed to more than 5mGy is 11%. This increases with increasing dose and where the dose is above 2 Gy, the attributable fraction is 61% (23). The Japanese koseki system of family registration allows accurate follow-up of mortality data and, as early as 1959, cancer registries were established in Nagasaki and Hiroshima. Results of epidemiological studies and their wider applicability is affected by the underlying general community risk of disorders, e.g. compared with North American or European populations, Japanese populations have since 1950 had low risk of haematological malignancy, breast cancer and circulatory diseases over the period, but generally higher rates of stomach cancer. There are also generational effects. With better nutrition, public health measures and technical advances, incidence and mortality of many cancers has declined although at the same time lifestyle changes, e.g. obesity and alcohol consumption, have reversed the pattern. PHE data show that age-standardised five-year survival in England and Wales over the period 1971-75 to 2004-2008 in males for stomach cancer has gone from 4% to 16.5%, colon cancer 22% to 52.4%, prostate cancer 31% to 80.6% and all leukaemias 12% to 41.7%.

Dosimetry and Probability of Causation (PoC)

25. The Japanese atomic bombs were kiloton devices. For both bombs initial extreme heat and pressure blast was accompanied by gamma radiation and a more limited burst of neutrons. The heat blast set the mainly wooden buildings in the cities on fire and most people within 1.5 km of the epicentre were killed. Although radiation doses were not directly measured, various methods were used to estimate retrospective doses. These included information about location, distance from the epicentre and shielding, both from the person's body and from buildings. Beyond 1.5 km the numbers of survivors much increased, giving a skewed population with many more exposed to low than high dose. The free in-air dose of radiation suitably weighted for the neutron component at 1 km from the epicentre was 7 Gy at Hiroshima and 10 Gy at Nagasaki, while at 2.5km the values were 13 mGy at Hiroshima and 23 mGy at Nagasaki (24).

26. Cancer due to ionising radiation is indistinguishable clinically from cancer due to other causes. Although it is not possible to say with absolute certainty whether a cancer in an individual is due to ionising radiation, in some circumstances, epidemiological data, information about the person and the population to which they belong, as well as radiation dose and exposure circumstances, and

recognised risk models can be used to estimate the probability that the cancer was caused by radiation. The Probability of Causation (PoC) is expressed as a percentage and is the risk the disease is due to radiation exposure divided by the overall disease risk in the parent population, i.e. the radiation risk/the base line risk and radiation risk multiplied by 100. For further discussion see Annex A.

The Results of the Japanese atomic bomb studies

27. For cancers:

- dose responses are significant for cancer of the salivary gland, oesophagus, stomach, colon, primary liver, lung, female breast, urinary bladder, gall bladder, central nervous system and thyroid.

- Rectal cancer, prostate cancer and kidney parenchyma cancer have not been associated with radiation in the Japanese studies.

- The evidence on pancreas, testis and kidney, pelvis and ureter is unclear.

28. Similarly while leukaemias other than CLL were the first group to be identified as having increased risk, there is no evidence in the latest mortality or incidence analysis of haematological malignancies of raised rates of Hodgkin's, Non-Hodgkin's Lymphoma (NHL) or multiple myeloma (25) (26).

Other sources of evidence on the links between ionising radiation and malignancy

29. There is some inconsistency between the atomic bomb survivor findings, usually considered acute high-dose studies, and conclusions from other study populations, e.g. occupational protracted low- dose exposed groups which themselves show heterogeneity. In considering the epidemiological literature on cancer and other adverse health effects of ionising radiation, attention should be paid to evidence quality, including the study design and power, i.e. case numbers, suitability of controls, age at exposure, age at diagnosis, duration of follow-up, whether the study is high or low-dose, and how the dose was delivered, acute or protracted or episodic and whether looking at disease mortality or incidence and the presence of bias or confounding, as well as case ascertainment. The concept of lag time is also important. Leukaemias have a short lag time, first appearing about two years after whole-body irradiation and peaking at six or seven years after exposure, while radiation-induced solid cancers, i.e. breast, bone, stomach etc. can occur any time from 10 years onwards. PHE adopts the 2007 ICRP publication, 103 assumptions on cancer risk coefficients, tissue and radiation weighting factors and the use of a dose and dose rate effectiveness factor (DDREF). The 2007 ICRP risk estimates took into account new cancer incidence data from the atomic bomb survivors cohort that was not available at the time of the previous 1990 recommendations (see Annex A).

Military Ionising Radiation Studies

30. About 210,000 military personnel took part in the US nuclear tests between 1945 and 1963 where 99% of those with film badges had a total dose of less

than 50 mSv, with the average about 6 mSv. In 1978 the US established a register of service personnel participants. There was some risk of selection bias in assembly of this database, which used a number of methods including self-report. Dates of birth were not always recorded. On it, three studies were based, all using matched service control groups. The first study looked at mortality among 70,000 participants at the Five Test series and 65,000 controls. Standardised Mortality Ratio (SMR) was 71 for all causes of death and 74 for malignancies. Leukaemia mortality in the test veterans was slightly greater than in the controls, but the SMR was 74, indicating that the veterans were at lower risk than the US general population (27).

31. The second study was of 38,000 naval personnel present at Operation Crossroads at Bikini Atoll in 1946 and included a matched group of 35,000 controls (28). In this study the relative risk (RR) for all- cause mortality was slightly raised among participants relative to the controls and the mortality for malignancies including leukaemias was also above 1, although not statistically significantly raised. This means that either the study did not have the power to detect a raised risk or that the raised relative risk was due to chance.

32. Similar findings were recorded in the third study (29) involving 8,500 naval veterans present at Operation Hardtack in 1958 and 14,000 controls, and followed up until 1 September 1991. This group was chosen because of its high proportion of veterans with film badge dosimetry data and median dose 3.88 mSv. Among those who received doses of more than 10 mSv there was an increased mortality from all causes and all cancers. However, for all gastro-intestinal cancers, although overall risk was high, there was no dose/response relationship and the risk of many cancers considered radiogenic was not significantly elevated or occurring in those with highest exposures. These mortality studies were based on death certificate data and did not control for factors such as tobacco and alcohol use, etc. It was concluded from the US atomic veteran evidence in its entirety that the risk of death from certain cancers could not be ruled out.

33. About 500 personnel from the Royal New Zealand Navy (RNZN) took part in the UK tests, and they have been the subject of two follow-up studies. The first covered the period 1957-87 and the second extended follow-up to 1992. Controls were again service personnel of similar age and serving around the same period as the veterans, but who did not participate in the tests (30) (31). The low power of these studies, especially when considering rare disorders, is noted but again relative risk of all causes of mortality and from all cancers was not significantly different from 1. However there were four deaths from leukaemia from a total of eight haematological malignancies by the end of the 1992 study. This was on the border of statistical significance.

34. In the 1980s there was a self-report Australian study (32). Response rates were low and bias an issue so interpretation of results was difficult and a further study was commissioned. Published in 2008, this compared the mortality and cancer incidence in 10,983 UK atmospheric test Australian veterans with the general Australian population, and between groups of veterans with different radiation dose assessments or estimates (33). All-cause mortality was not raised but

mortality and incidence was raised for cancers of the head and neck, lung, colon and rectum and prostate and for all cancers combined. Incidence was raised for oesophageal cancer, melanoma and all leukaemias but mortality was not raised. There was no association between estimated radiation exposure and overall cancer mortality or incidence, nor with any specific cancer or cancer deaths. The estimated average radiation dose was comparable with natural background levels and fewer than 5% received more than 20 mSv. The comparator group from the Australian general population were very different from the veterans with a lower number born in Australia, as well as marked differences in ethnicity. The pattern of cancer in the nuclear test veterans was, however, very similar to that found in Australian Korean war veterans serving around the same time as the UK Minor Trials (34). Here ionising radiation exposure was not an issue and 15% of the veterans had served in both campaigns. The authors related some of the findings to smoking and, in naval personnel, where there was an excess of mesothelioma and lung cancer, to asbestos exposure. They were unable to explain the excess leukaemias, but concluded that the excess cancers and cancer deaths were not attributable to radiation exposure.

The UK Atmospheric nuclear test and weapons experiments

35. The UK atmospheric nuclear tests carried out from 1952-58 involved a series of 21 explosions (12 in Australia (Monte Bello Islands, Maralinga) and 9 in the South Pacific (Malden and Christmas Islands in 9 operations) where natural background radiation is low, e.g. in the Christmas Islands, 0.58 mSv per annum. The Maralinga Experimental Weapons Programme (MEP), also known as the Minor Trials, examined weapons design and safety and did not involve significant nuclear fission, although some of the experiments, notably at Vixens A and B, did generate radioactive contamination with uranium and plutonium dispersal. The Minor Trials lasted until 1963 with clean-up operations until 1967.

36. About 16,000 Australian personnel, military and civilian, also took part in the atmospheric tests and MEP. As with UK personnel, the majority had support functions including transport, construction and catering, with only a minority directly involved with weapons trials or detonations and entering potentially contaminated areas. They have been the subject of a separate epidemiological study looking at mortality and cancer incidence.

37. The atmospheric tests and Minor Trials were carried out to the highest contemporary radiological standards, including the use of high altitude air-bursts and tower-mounted detonations to minimise the production of radioactive fallout. All participants were monitored for radiation exposure in the early tests, but measurable exposures only occurred to those participants who were at high risk because of their duties and locations. For later tests a targeted approach was adopted with only “at risk“ personnel being monitored.

38. In Australia (all kiloton-range devices), tests were generally tower-mounted detonations. At Christmas and Malden Islands, all megaton tests were high altitude air-bursts, and the kiloton-range tests were suspended at high elevation from balloons. These measures ensured that participants’ exposure to fallout was minimised. Test planning took account of weather conditions so that

radioactive debris from the explosion went into the highest levels of the atmosphere and remained there for a significant time, with decay and dilution before descent to the Earth's surface and minimal immediate dispersion of contaminated materials downwind. During the course of the tests and Minor Trials, and afterwards, environmental monitoring programmes (radiation surveys (air, water etc.) flora and fauna analysis, etc.) were performed.

39. In 1967 after the MEP was completed, the UK conducted a clean-up of all the sites to reduce contamination to safe and acceptable levels on the understanding that access to the experimental area would thereafter be restricted. In 1968 the Australian government confirmed that they were content with the decontamination and debris clearance. By the early 1980s, the Australian government was being lobbied by antinuclear, environmentalist and pro-land rights activists. In 1984 a Royal Commission was set up and recommended that the affected experiment areas should be returned to a state which allowed unrestricted habitation. The resultant Technical Assessment Group (TAG) reported on the residual contamination with options and costings for decontamination. A partial clearance option leaving about 100 square kilometres to which access was prohibited was agreed, and in December 1993 the UK government offered £20 million in a full and final settlement to fund a further clean-up operation. In April 2000 the Australian government announced completion of the clean-up and that it was safe for the indigenous population (35).

The NRPB nuclear test follow-up studies

40. As a result of concern amongst some test participants about the effects that participation could have had on their health, in 1983 the Ministry of Defence commissioned an independent study by the NRPB to investigate whether the health of participants at the UK atmospheric nuclear tests and weapons experimental programme showed any correlation with radiation exposure.

41. This comprehensive cohort study compared the mortality and cancer incidence in over 20,000 test and Minor Trials participants with that of a similar-sized control group of ex-servicemen who were age- matched, had served around the same time and had deployed overseas but had not participated in the test programme.

42. The term ‘test participant” has a particular definition and includes servicemen present at the due dates, at any of the following test sites and experimental programmes:

Operation	Site	Date
Hurricane	Monte Bello W Australia	April 1952-June 1956
Mosaic	Monte Bello W Australia	May-June 1956
Totem	Emu Field S Australia	Aug 1953-Aug 1957
Buffalo	Maralinga S Australia	April 1955-Aug 1967
Antler	Maralinga S Australia	Sept/Oct 1957
Grapple X Y Z	Christmas Island S Pacific	1957-58
Op Brigadoon	Christmas Island	1962
RAAF Pearce RAAF Edinburgh	W Australia S Australia	May-August 1956 Aug 1956-Nov 1960
Minor Trials:		
Kittens Tims Rats Vixen A Vixen B	Emu Field Maralinga Maralinga Maralinga Maralinga	1953-61 1955-63 1956-60 1959-61 1960-63
Clean-up ops:		
Ayres Hercules Brumby	Maralinga Maralinga Maralinga	1960-63 1964 1967

To be identified as a test "participant" there is no requirement to be present at actual detonations.

Op Brigadoon was a series of US tests, part of Op Dominic, which took place off Christmas Island between April and July 1962. At the RAAF sites, the work included cloud sampling and handling contaminated aircraft. RN ships were associated with tests at Monte Bello, Malden and Christmas Island. The Minor Trials did not involve nuclear detonations. They took place at Maralinga (Tims, Rats and Vixen A and B) while Kittens was at Emu Field. Major clean-up operations took place at Christmas Island in 1964 and Maralinga in 1964 and 1967.

43. The main conclusions of the first NRPB Report (36) were that the test participants showed increased risk of multiple myeloma and leukaemia (other than chronic lymphatic leukaemia) compared with service controls. However, the conclusion that this was the result of the participants attending the tests being exposed to ionising radiation from the explosions was not considered appropriate. This was because there was a particularly low rate of the conditions in the controls, meaning that the raised risks were not due to increased disease among the participants but lower rates of disease among the controls. In addition, among the sub-groups, those considered most highly radiation-exposed did not show high rates of the conditions.

44. Otherwise, presence at the sites;

- did not have a detectable effect on the participants' expectation of life,
- did not have a detectable effect on participants' risk of developing any other malignancy.

45. The study was extended and the second NRPB Report (37)) produced an additional seven years' data, and:

- confirmed the overall conclusion of the 1988 Report, that participation in the tests had no detectable effect on the participants' expectation of life nor on their risk of developing most cancers.
- concluded that the small hazard of multiple myeloma suggested by the 1988 Report was not supported by the additional data, although the possibility of some small risk of developing leukaemia (other than chronic lymphatic leukaemia) in the first 25 years after participation could not be ruled out.

With regard to other cancers the report concluded that:

- overall the number of deaths and cancer incidence amongst participants is lower than amongst the control group.
- as expected, because a large number of diseases were considered, any excesses in participants are due to chance.

46. Following pressure for a further investigation into the alleged effects of exposure, a third analysis of the NRPB study was commissioned. The report of the study which extended the follow-up period to 1998 was published in February 2003 (38).

Key findings:

- Re-affirmed the overall findings of the 1988 and 1993 reports, that participation in the Tests had no detectable effect on the participants' expectation of life, nor on their risk of developing most cancers.
- Confirmed the conclusion of the 1993 report on the alleged association between participation in the UK test programme and multiple myeloma, that there is no evidence to support a link.
- Suggested, particularly in the 2–25 years after first test participation, a small increase in risk of leukaemia (excluding chronic lymphatic leukaemia) among test participants relative to controls, although the difference in rates between the two groups was narrowing with longer follow-up.

Impact of the NRPB reports on the Department's normal policy on claims for cancers due to service- related ionising radiation

47. Applying the test set out at para 7-9 of this statement, the Secretary of State considered the National Radiological Protection Board Reports, of which a principal author was Sir Richard Doll, to be **reliable evidence**. In particular the following points were noted:

- The study identified the test participants, and followed them up to monitor the occurrence of disease and death in the participant population. It then compared this, over the same time period, with the rates in both a service and civilian control population.
- The study involved 20,000 subjects and an equal number of controls.
- The reports describe in detail the efforts made to ensure sample completeness and to control bias.
- The study limitations are discussed by the authors and conclusions are reasoned and restrained.

The Secretary of State's opinion as to the reliability of the evidence in the reports is in accord with the general opinion of the scientific community, including the US Presidential Advisory Committee on Human Radiation Experiments (32).

48. Based on the first report, the Secretary of State's normal policy became to award war pension for claims for leukaemia (other than chronic lymphatic leukaemia) and multiple myeloma in those present at test sites. The policy also included awards for primary polycythaemia rubra vera, the red blood cell equivalent of

leukaemia. In light of the 1993 report, the Secretary of State's normal policy was revised. Since then, on the basis of presence at atmospheric nuclear test sites, new claims for multiple myeloma are rejected but awards continue to be made for leukaemia (other than chronic lymphatic leukaemia) and primary polycythaemia rubra vera having clinical onset within 25 years of first presence at the test sites. On the basis of the findings of the 2003 report, the Secretary of State's current normal policy remained unchanged from that in 1993.

49. The reports did not causally link development of those conditions to ionising radiation exposure and the policy is not an acknowledgement that those present at the tests were exposed to harmful levels of ionising radiation. The accepted Service link is purely presence at the test sites (see Annex B).

50. Having carefully considered the reports, the Secretary of State was and remains of the opinion that they do not provide reliable evidence to raise a reasonable doubt that generally other cancers (e.g. primary liver and urinary bladder) might be attributable to service in the Armed Forces simply because of presence at the nuclear test sites. Consequently it is presently his normal policy that entitlement for solid cancers, causing disablement or death, may not be presumed, i.e. accepted on the basis of presence at atmospheric nuclear test detonations, weapons tests or clean-up operations alone. However, it is also his normal policy that an entitlement to war pension may be certified for cancer or other radiogenic disorders in any case where, on the case-specific facts, there is reliable evidence of service exposure to a sufficient level of ionising radiation and there is a recognised causal link between the claimed condition or cause of death and such accepted exposure.

51. At Annex B details of the design, radiological protection and dosimetry arrangements at the UK tests are set out as well as discussion of those groups of Service personnel considered to be at high risk of exposure to significant doses of ionising radiation.

Children of test participants and MOD civilian employees

52. The sample on which the 1988, 1993 and 2003 NRPB Reports were based did not include the children of test participants, and was solely concerned with a study of the test participants themselves and not with any possible effect their participation might have had on their progeny. Any claim for compensation for a child in respect of disablement or death said to be due to the parent's participation in the UK Tests does not fall within the scope of the Service Pensions Order. Similarly, compensation for civilian MOD employees or their widows who participated in the tests is not covered by the War Pension Civilians Scheme (3 September 1939 and 19 March 1946).

Impact of overall evidence on the Department's normal policy on the radiogenicity of malignant conditions

53. Having carefully considered the overall contemporary medical and scientific published peer-reviewed literature in the context of the war pensions onus and standard of proof, the normal policy in war pensions (at date of statement publication) is that there is reliable evidence to raise a reasonable doubt that

there may be a causal link between ionising radiation exposure and the following cancers:-

leukaemia (other than chronic lymphatic leukaemia)
female breast
lung
oesophagus
stomach
colon
primary cancer of the liver
gall bladder
thyroid
urinary
renal pelvis and ureter
central nervous system
salivary gland
bone
rectum

54. In war pension claims for disablement or death due to these conditions **and** where the Secretary of State has accepted **service-related ionising radiation exposure, either from i) expert dosimetry measurement or estimate or ii) where there has been service at the locations listed at Annex B**, war pension entitlement will be considered. Although the Japanese studies do not find a significant dose response for rectal cancer, and because several studies do not differentiate rectal and colon cancer, rectal cancer is also included in the list. The Secretary of State does not accept evidence of participation in nuclear tests as itself equating to proof of service-related ionising radiation exposure. Based on the NRPB studies, entitlement will continue to be presumed for leukaemias other than CLL in those present at the UK atmospheric nuclear tests and weapons experimental programmes. Since the nuclear test studies, more evidence has been published on haematological malignancies and that new evidence and its impact on the Departmental policy is considered below.

Haematological malignancies including Polycythaemia Rubra Vera, Chronic Lymphatic leukaemia, Hodgkin's and Non-Hodgkin's Lymphoma and multiple myeloma

55. The earlier war pensions policy decision to accept Polycythaemia Rubra Vera (PRV) on a presumptive basis as for the analogous leukaemia (other than CLL) diagnosed within 25 years of presence at the tests was based on a single claim and a small US case study suggesting an excess of cases in a population who had taken part in a nuclear test (40). This finding and its interpretation was challenged at the time (41) and has not been replicated in any other population. There are also issues as to the soundness of the histological diagnosis in the cases in the study. As a result, from the date of this policy statement, PRV will not be accepted on the basis of presumption amongst nuclear test and weapons

programme participants.

56. Evidence on the radiogenicity of the leukaemias and related haematological disorders is not consistent across studies. An important issue is accuracy of diagnosis: the pathology of haematological malignancy is complex and the disorders relatively uncommon, with small case numbers in many studies. In recent years, new haematological classification systems have been developed, often based on clinical features, genetics and treatment response. This makes pooling of study results or comparison of findings over time very difficult. It is also true that the incidence of some haematological malignancies has increased in recent years. This may relate to higher awareness and more assiduous case ascertainment as well as factors such as HIV infection in communities. Background incidence of haematological malignancies also varies in different populations so that extrapolation of results to other populations may not be valid.

57. The 2003 NRPB Advisory Group on Ionising Radiation (AGIR) review of the literature on the risk of leukaemia and related malignancies concluded that apart from chronic lymphatic leukaemia (CLL), for which there was no evidence of radiogenicity, there was good evidence of a causal link between radiation, and the acute leukaemias and Chronic Myeloid Leukaemia (CML). For Non-Hodgkin's Lymphoma (NHL), considered a group of disorders, not a single diagnosis, they found little evidence of a link to radiation; there was no evidence of a causal link between Hodgkin's disease (HD) and radiation and similarly only weak evidence of a causal link to multiple myeloma (MM) (16).

CLL as a radiogenic disorder

58. CLL is by nature different from other types of leukaemia. It mainly affects older people and is a disorder with a long latent period. It is often asymptomatic with diagnosis made fortuitously. Similarly, there tends to be prolonged morbidity rather than rapid death. These features mean that many of the published studies with short lag and follow-up time as relevant to acute leukaemias may report no link with ionising radiation simply because they have failed to detect cases. Similarly CLL may not be recorded on death certificates as a cause or factor in mortality. Publication bias is also an issue. As in the atomic bomb studies, numbers of cases of CLL may simply be too small for analysis. Finally, as with other haematological malignancies, there may be misclassification and misdiagnosis.

59. In 2011, following advice from the National Institute for Occupational Safety and Health (NIOSH), the US, having previously regarded CLL in the context of its federal occupational and military disability compensation schemes as a disorder with a zero link to ionising radiation, accepted that CLL was radiogenic.

60. A 2005 review article had proposed that, while epidemiological evidence of an association between CLL and radiation was weak, within its limits there was nothing to suggest that CLL was an exception to the general principles of radiation carcinogenesis (42). Meanwhile, follow-up case-control studies of Chernobyl clean-up workers (43) (44) suggesting increased rates of CLL began

to appear. There are issues regarding the basis of diagnosis, case numbers are small and history for dose reconstruction often reliant on patient recall or proxy interview. It is also true that the Ukrainian Chernobyl follow- up studies with regular clinical surveillance, including subject review and blood tests, provide higher opportunity for detection of disorders not seen in the equivalent comparator (Ukrainian general male) population. This might go some way to explain the approximately 60% higher rates of CLL in the Chernobyl workers compared with the general population. In the 2013 paper looking at cases of leukaemia diagnosed amongst the 111,645 workers over the period 1986-2006, worker controls were matched to cases by age and residence. Dose reconstruction provided estimated average case radiation exposure as 132.3 +/- 342.6 mGy while for controls it was 81.8+/- 193.7 mGy. Exposure dose was reconstructed from interviews of subjects or with next of kin regarding work location, the clean-up tasks and time spent. There were 137 leukaemia cases in total including 79 CLL with dose estimation. The study found similar radiation-related risks for CLL and non-CLL except for a sub-set of cases interviewed less than two years from the start of chemotherapy. In that group, radiation risk of CLL was much lower as was their mean bone marrow dose. This group includes personnel within the 30 km zone of the explosion. On the other hand, no such difference was found with worker controls. No explanation was available for this finding. The authors also recognised that their finding of a link between radiation and CLL was not replicated in other high-quality studies, e.g. the third analysis of UK radiation workers (45) or the Techa river contaminated population follow-up (46). They conclude that further study on the relation between the two is required. The 2015 INWORKS chronic low dose study followed up over 300,000 radiation-monitored workers in France, the US and the UK for 8.22 million person years and showed accrual of a mean dose of 1.1 mGy per year. There was strong association between leukaemia mortality and radiation dose (RR 2.96 per Gy (1.17-5.21), mainly due to chronic myeloid leukaemia with an ERR per Gy of 10.45 (Annex A). In this study a negative association was found between CLL and radiation exposure (9).

61. The US decision to accept CLL as radiogenic in its occupational injury schemes was also influenced by their previous decision, despite the lack of direct evidence, to accept Non-Hodgkin's lymphoma (NHL) as radiogenic. Like NHL, CLL is a B cell lymphoma. This makes it biologically plausible that both malignancies should share similar radiogenicity and the US concluded that to continue to assert zero risk for CLL, having accepted NHL as radiogenic, was illogical.

Impact on the Department's normal policy on claims for haematological malignancies due to service- related ionising radiation exposure

62. At this date, based on overall evidence, policy remains to accept entitlement for leukaemias other than CLL simply on the basis of participation at the tests or experimental programmes without case-specific dose determination, when they present clinically within twenty-five years of presence at the tests or weapons experiments. CLL, PRV, HD, NHL and MM are not accepted as radiogenic disorders. The literature will continue to be monitored.

Evidence of radiation induction of non-cancer conditions

63. While reports of increased rates of leukaemia in atomic bomb survivors began to emerge in the 1960s, longer follow-up suggested that ionising radiation exposure may also be associated with non-cancer diseases (47) (48). Associations have been described with uterine fibroids, certain non-cancerous thyroid and para-thyroid tumours and, importantly, with circulatory disorders. Cataract is known to be caused by high doses of ionising radiation. A review of the current evidence on ionising radiation and circulatory diseases and cataract is at Annex C.

64. On present overall evidence, mainly from high-dose radiotherapy studies, it is generally accepted that there is a raised risk of circulatory disorders (including stroke, atherosclerotic coronary artery disease and heart failure) at about 5 Gy acute exposure, and evidence is accumulating for an association at doses between 0.5 Gy and 5 Gy. However, results from the atomic bomb survivor studies and nuclear industry protracted dose studies are heterogeneous and inconsistent, and few studies adequately control for the major established cardiovascular lifestyle risk factors. At present the ICRP does not recommend that calculation of the Probability of Causation (PoC) is appropriate for circulatory disease risk. It states that evidence of excess risk of mortality from circulatory disease is good only at doses of several Gy or more. There are uncertainties about the shape of the dose response curve at low doses, and Japanese data are consistent both with a no-dose threshold or a threshold of 0.5 Sv). ICRP recommends adoption of a linear dose response with threshold at 0.5Sv.

65. Lens opacification can also be caused by ionising radiation. The mechanism of radiation-induced cataract is not understood, nor whether the effect is deterministic or stochastic. Cataracts can be induced by 2 Gy of acute low LET radiation and 5 Gy of chronic low LET. For visual disablement higher doses estimated to be about 10 Gy exposure are required (49).

Impact on Departmental normal policy on the relation between ionising radiation and circulatory disorders and cataract

66. For circulatory disorders – stroke, coronary artery disease and cardiac failure and lens opacity/cataracts, where the Secretary of State accepts service-related ionising radiation exposure, claims will be considered on their individual merits including measured or estimated dose exposure. The literature will continue to be monitored.

References:

(1) Watson, S.J. et al (2005) Ionising radiation exposure of the UK population Chilton HPA-RPD-001

(2) ICRP (2007) The 2007 recommendations of the ICRP, ICRP. Pub. 103 Ann. ICRP 37(2-4)

(3) BEIR VII (2006) Committee to assess health risks for low levels of ionising radiation. US Nat. Acad. of Science, Nat. Research Council, Washington DC. Nat. Acad. Press

(4) Preston, D.L. et al (2007) Solid cancer incidence in atomic bomb survivors.

Radiat. Res 168:1-64

(5) Boice, J.D. et al (1985) Second cancers following radiation treatment for cervical cancer: an international collaboration using cancer registries. J. Natl. Cancer Inst. 14:955-75

(6) Weiss, H.A. et al (1994) Cancer mortality following X-Ray treatment for ankylosing spondylitis. Int. J. cancer 59: 327-38

(7)) Vrijheid, M. et al (2007) Mortality from diseases other than cancer following low doses of ionizing radiation: results for the 15-country study of nuclear industry workers. Int. J. Epid. 36:1126-1135

(8) Richardson, D.B. et al (2015) Risk of cancer from occupational exposure to ionizing radiation: retrospective cohort study of workers in France UK and US. (INWORKS) BMJ 351:h5359

(9) Leuraud, K. et al (2015) Ionising radiation and risk of death from leukaemia and lymphoma in radiation-monitored workers (INWORKS): an international cohort study. Lancet Haematol. 2:e276-281

(10) Dickman, P.W. et al (2003) Thyroid cancer risk after thyroid examination with I 131: a population- based cohort study in Sweden. Int. J. Cancer 106:580-587

(11) Darby, S.C. et al (2005) Radon in homes and risk of lung cancer: collaborative analysis of individual data from 13 European case control studies. Br. Med. J. 330: 223-6

(12) Wick, R.R. et al (1999) Late effects in ankylosing patients treated with 224 Radium. Radiat. Res. 152(6 supp). S8-S11

(13) Shilnikova, N.S. et al (2003) Cancer mortality risk among workers at the Mayak nuclear complex. Radiat. Res. 159: 787-98

(14) Kascheer, V.V. et al (2015) Incidence and mortality of solid cancer among emergency workers of the Chernobyl accident: assessment of radiation risk for the follow-up period 1992-2009. Radiat. Environ. Biophys. 54: 13-23

(15a) UNSCEAR 2006 Scientific Epidemiological studies of radiation and the effects of ionising radiation. UNSCEAR report 2006 NY

(15b) UNSCEAR 2006 Scientific Annex B Epidemiological evaluation of cardiovascular disease and other non-cancer diseases following radiation exposure. Vol 1 UNSCEAR report 2006 NY

(16) AGIR (2003) Risk of leukaemia and related malignancies following radiation exposure: estimates for the UK population. Doc NRPB,14(1) 1-119

(17) AGIR (2011) Risk of solid cancers following radiation exposure: estimates for the UK population. Doc HPA RCE-19, 1-245

(18) AGIR (2010) Circulatory disease risk. Doc HPA RCE-16, 1-116

(19) HPA (2009) Application of the 2007 recommendations of the ICRP to the UK Advice from the HPA. RCE -12

(20) CERRIE (2004) Report of the committee examining risks of internal

emitters.

(21) Cox, R. et al (2005) The response of the NRPB to the Report of the Committee examining radiation risks of internal emitters (CERRIE).

(22) ICRP (2005) Low dose extrapolation of radiation-related cancer risk. ICRP pub. 99 Ann. ICRP 35(4)

(23) Doupe, E.B. et al (2011) Long term radiation-related health effects in a unique human population: lessons learned from the atomic bomb survivors of Hiroshima and Nagasaki. 5(01): S122-S133

(24) Cullings, H.M. et al (2006) Dose estimation for atomic bomb survivor studies: its evolution and present status. Radiat. Res. 166: 219-254

(25) Hsu, W.L. et al (2013) The incidence of leukaemia, lymphoma and multiple myeloma among atomic bomb survivors 1950-2001. Radiat. Res. 179(3): DOI; 10.1667

(26) Ozasa, K. et al (2012) Studies of the mortality of atomic bomb survivors, Report 14 1950-2003 – an overview of cancer and non-cancer diseases. Radiat. Res. 177: 229-243

(27) Thaul, S. (2000) The Five Series study: mortality of military participants in US nuclear weapons tests. Medical Follow-up Agency. Ins. of Med. Nat. Acad. of Sciences, Washington D C. National Ac. Press

(28) Johnson, J.C. et al (1997) Mortality of veteran participants in the Crossroads nuclear test. Health Physics 73(1): 187-197

(29) Watanabe, K.K. et al (1995) Cancer mortality risk among military participants of a 1958 atmospheric nuclear weapons test. Am. J. Pub. Health 85: 523-7

(30) Pearce, N. et al (1990) Follow-up of NZ participants in British atmospheric nuclear weapons tests in the Pacific. BMJ 300:1161-6

(31) Pearce, N. et al (1997) Further follow-up of NZ participants in British atmospheric nuclear weapons tests in the Pacific. Cancer Causes and Control 8: 139-145

(32) Donovan, J. et al (1983) Health of atomic personnel. Canberra, Commonwealth Dept. of Health

(33) Gun, R.J. et al (2008) Mortality and cancer incidence of Australian participants in the British nuclear tests in Australia. Occup. Env. Med. 65: 843-8

(34) Aust. Ins. of Health and Welfare (AIHW) (2003) Cancer incidence study: Australian veterans of Korean War.

(35) Rehabilitation of former nuclear test sites at Emu and Maralinga (Australia) 2003 – Report by the Maralinga Rehabilitation Technical Advisory Committee MARTAC Report

(36) Darby, S. et al (1988) Report on Mortality and Cancer Incidence in UK Participants in UK Atmospheric Nuclear Weapon Tests and Experimental Programmes. NRPB-R214

(37) Darby, S. et al (1993) Report on Mortality and Cancer Incidence 1952-1990 in UK Participants in the UK Atmospheric Nuclear Weapon Tests

and Experimental Programmes. NRPB-R266

(38) Muirhead, C.R. et al (2003) Report on Mortality and Cancer Incidence 1952-1998 in UK Participants in the UK Atmospheric Nuclear Weapons Tests and Experimental Programmes. NRPB-W27 – ISBN 0-85951-499-4

(39) Thomas, J. (1999) Letter to the editor. J. Radiol. Prot. 18(3):209-10

(40) Caldwell, G.G. et al (1984) Polycythaemia vera among participants of a nuclear weapons test. JAMA 252(5):662-4

(41) Sobell, J.L. et al (1987) Polycythaemia vera among participants of a nuclear weapons test. JAMA 259(9):1178-79

(42) Richardson, D.B. et al (2005) Ionising radiation and chronic lymphocytic leukaemia Env. Health Perspect. 113:1-5

(43) Romanenko, A. et al (2008) The Ukrainian-American study of leukaemia and related disorders among Chernobyl clean-up workers from Ukraine 111. Radiation Risks Radiat. Res. 170: 711-720

(44) Zablotska, L. et al (2013) Radiation and the risk of lymphocytic and other leukaemias among Chernobyl clean-up workers. Env. health Perspec. 121:59-65

(45) Muirhead, C.R. et al (2009) Mortality and cancer incidence following occupational radiation exposure. Third Analysis of the National Register of Radiation Workers. Brit. J. Cancer 100(1): 206-12

(46) Krestinina, L.Y. et al (2010) Leukaemia incidence among people exposed to chronic radiation from the contaminated Techa river 1953-2005. Radiat. Env. Biophys. 49 (3):195-201

(47) Yamada, M. et al (2004) Non-cancer disease incidence in atomic bomb survivors 1958-98. Radiat. Res. 161: 622-32

(48) Pham, T-M. et al (2013) Radiation exposure and the risk of mortality from non-cancer respiratory diseases in the Life Span Study. Radiat. Res. 179:46-52

(49) Ainsbury, E.A. et al (2009) Radiation cataractogenesis: a review of recent studies. Radiat. Res. 172:1-9

Annex A Radiation dose

1. The first definition of a unit of radiation dose was made in 1928 by the International Congress of Radiology. The rontgen (R) was defined as that quantity of radiation which produces in 1 cm of air one unit of charge of either sign, thus defining a unit of exposure.

2. Units of **absorbed dose**, rads, the actual energy absorbed in the tissue being irradiated, were later introduced and are now cited in SI (Systeme Internationale) units – joules per kg of absorbing material. The fundamental unit – 1 joule/kg – is 1 gray (1Gy), equivalent to 100 rads (R).

   Different types of radiation differ in the way they interact with living tissues and equal absorbed doses cause different degrees of damage. X-rays, Gamma rays and beta particles transfer a low rate of energy as they pass through tissues, and are referred to as **low Linear Energy Transfer (LET)**, while alpha particles and neutrons are examples of **high Linear Energy Transfer (LET)** radiation. The biological effects of high LET radiation are greater than those of low LET particles. This is taken account of by a **Radiation Weighting Factor** defined by ICRP (2007) as 1 for X-rays, gamma and beta radiation, and 20 **for alpha particles.**

3. The **absorbed dose** multiplied by the **radiation weighting factor** provides the **equivalent dose** i.e. all doses regardless of radiation type are expressed relative to the effects of X-rays.

4. Not all tissues are equally radiosensitive and this is reflected in **Tissue weighting factors**, so that lung and bone marrow, which are radiosensitive, have a higher value, 0.1, than skin or bone surface at 0.01.

   The current SI unit of equivalent dose is the **Sievert (Sv)**. This weighs radiation according to type and the sensitivity of the exposed tissue so that different types of radiation can be added together.

   The **effective dose** is derived from **the equivalent dose** multiplied by the **tissue weighting factor** and summed across the body organ and tissues. This can be used for whole-body and local irradiation, and external and internal radiation can be summed together.

   For X-rays and gamma rays the equivalent dose in Sieverts and the absorbed radiation dose in Grays are the same. The relationship between the different dose units is:

   1 gray (Gy) = 1 joule/kg = 100 rads (R) = 100 rems (r) = 1 sievert (Sv) = 1,000 millisieverts (mSv) = 1,000,000 microsieverts (microSv).

Typical effective doses, i.e. whole body of radiation

Chest X-ray (PA) – 0.014 mSv
Head CT scan – 1.4 mSv
Bone scan – 4 mSv
Chest CT scan – 6.6 mSv
Coronary angiography – 3.9 mSv
Ba swallow – 1.5 mSv

Radiotherapy treatment (radical)

Non-small cell cancer of lung	60 Gy in 30 fractions
Lymphoma	30-40 – Gy in 20 fractions

Average annual UK dose from cosmic rays – 0.26 mSv
Average annual UK dose from gamma rays – 0.35 mSv
Average annual UK dose natural background radiation – 2.2 mSv

Most information on cancer risk in populations comes from high-dose studies. It is generally accepted that at low doses and dose rates the risks are lower and a reduction factor, the dose and dose rate effectiveness factor (DDREF) is applied to the risks calculated from high-dose studies and for radiological protection. ICRP 2007 maintained a DDREF of 2. HPA agreed that while the value cannot presently be precisely calculated, the ICRP recommended value of 2 is compatible with other recent estimates.

Internal radiation

5. Radiation from outside the body is relatively easy to detect, monitor and quantitatively assess, and most epidemiological studies on adverse health effects focus on it. Internal radiation results from inhalation, ingestion or absorption through broken skin. All types of radiation can produce internal radiation but for the nuclear test and Experimental Programmes alpha radiation from unspent uranium and plutonium is particularly important. Made up of two protons and two neutrons, alpha particles are heavy and slow-moving, losing energy quickly, with a short range.

6. Internal radiation dose cannot be measured directly but is calculated from estimated radionuclide intake using air, food and water measurements. There are, however, few such measurements in relation to the UK nuclear atmospheric tests. A three-stage model can be used to estimate the ground deposit of fallout, the airborne concentration of radionuclides due to the ground deposit and then using dose conversion factors, dose due to intake of radionuclides. Dose rates over external and internal radiation decline over time as the material decays. After three months or so the internal dose inhaled from the unburnt nuclear fuel becomes dominant while radiation exposure of reducing levels continues while any material remains in the body.

7. When alpha particles enter the body by inhalation, some particles are lodged in the lung while some travel to the thoraco-bronchial lymph nodes and systemic circulation. Dependent on dose and tissue or organ sensitivity there is varying risk of cancer development. Tissues most at risk from particulate radiation include lung, liver and bone. UNSCEAR (2006) and ICRP find that taking into account the higher relative biological effectiveness of alpha particles compared with external radiation, radiation risks from internal and external emitters can be combined. They also conclude that there are no data suggesting that risks from alpha radiation have been substantially underestimated. The evidence of cancers due to alpha radiation at other sites and for the leukaemias is very limited, of low statistical power and quality, and inconsistent.

8. The 2006 study of mortality and cancer in Australian nuclear test veterans reconstructed estimated doses for personnel, concluding that 79% received less than 1 mSv. The mean Australian dose at Maralinga was 15 mSv, while for UK participants it was about 7 mSv (1). The Australian study, like the NRPB studies, found no evidence that Minor Trials participants were different in terms of mortality or cancer incidence from the nuclear test participants overall, nor was there any relation between measured or estimated radiation dose and incidence or mortality of leukaemia or a range of malignancies.

Reference:
Crouch, P. et al (2009) Assessment of radiation doses to Australian participants in British nuclear tests. Rad. Prot. Dos. 136:158-67.

Radiological protection

9. Radiation dose limits were first recommended for ionising radiation exposure in 1928. The statutory limit on the amount of radiation to which the general public may be exposed in excess of natural background radiation and excluding medical exposure is set from 1 January 2000 at 1 mSv per annum.

10. The most important source of man-made exposure is medical investigation which accounts for 90 per cent of man-made exposure. Average natural background radiation is raised to 2.6 mSv by all man- made exposure. UK estimated experience excluding medical investigation is 0.04 mSv. Other statutory limits include occupational dose limits. From 1 January 2000 these are 20 mSv per annum for classified workers and 6 mSv per annum for unclassified workers. Recent average effective occupational dose is 0.4 mSv with only 1% of recorded doses exceeding 5 mSv and none more than 10 mSv.

Reference:
Watson, S.J. et al (2005) Ionising radiation exposure of the UK population. Chilton HPA-RPD-001

Health effects of ionising radiation

11. Adverse health effects of ionising radiation are independent of the source of radiation and are of 2 types, largely related to exposure dose and occurring early or late.

Deterministic/tissue effects

- These effects arise shortly after exposure, usually within hours or weeks.
- There is a threshold dose, beneath which no effects are seen.
- This threshold is relatively high, exceeding natural background radiation levels in all parts of the planet by several hundred-fold.
- The severity of the effect varies directly with the dose.
- Duration of exposure is also important and for a given total dose, acute exposure is more harmful than a protracted dose.
- The tissues affected are those whose cells have a high turnover rate, i.e. bone marrow/skin/ gastro-intestinal tract.

Stochastic/probabilistic effects

- These effects arise years (2-40 or more) after exposure and the probability depends on the level of the dose.
- There appears to be no threshold and the severity of the effects is not

dose-dependent.

- This means that there is a finite risk even from low-level natural background radiation. At the same time persons exposed to a high dose may suffer no ill effects.
- The two main late effects are induction of cancer and hereditary disease in subsequent generations.
- All diseases which can be radiation-induced can also occur naturally or in relation to other exposures – cigarette smoke, alcohol, diet (both excesses and deficiencies), occupational exposures – and are not distinguishable on the basis of cause.
- Current best evidence is that radiation of all types gives rise to less than 2% of all cancers worldwide. The most important carcinogenic radiation type is in fact ultraviolet light (UVB), not ionising radiation.
- Not all types of cancer have been shown by evidence to be caused by ionising radiation.
- Hereditary effects have not been demonstrated in humans but there is such evidence in some types of animals.

Effects of total body irradiation

Equivalent dose (Sv)	Effect
Sublethal to man 0.0001 (0.1 mSv)	Around 2 weeks' natural background radiation, no detectable effect.
0.001 (1 mSv)	Around 6 months' natural background radiation, no detectable effect.
0.01 (10 mSv)	No detectable effect.
0.1 (100 mSv)	Minimal decrease in peripheral lymphocyte count, no clinical effect.
1 (1000 mSv)	Mild acute radiation sickness in some individuals (nausea, possible vomiting), no acute deaths, early decrease in peripheral lymphocyte count, decrease in all WBC and platelets at 2-3 weeks, increase in late risk of leukaemia, solid tumours.
Lethal to man 10 (10,000 mSv)	Severe acute radiation sickness, severe vomiting, diarrhoea, death within 30 days of all exposed individuals. Severe depression of blood cell and platelet production, damage to gastrointestinal mucosa.

100 (100,000 mSv)	Immediate severe vomiting, disorientation, coma, death within hours.
1000 (1,000,000 mSv)	Death of some micro-organisms, some insects within hours.
10,000 (10,000,000 mSv)	Death of most bacteria, some viruses.
100,000 (100,000,000 mSv)	Death of all living organisms, denaturation of proteins.

The concept and calculation of probability of causation

1. Cancer due to ionising radiation is indistinguishable clinically from cancer due to other causes. Although it is not possible to say with absolute certainty whether a cancer in an individual is due to ionising radiation, in some circumstances, epidemiological data, information about the person and the population to which he belongs, as well as exposure circumstances and recognised risk models, can be used to estimate the probability that the cancer was caused by radiation. The Probability of Causation (PoC) is expressed as a percentage. It is the risk the disease is due to radiation exposure divided by the overall disease risk in the parent population, i.e. the radiation risk/the base line risk, i.e. the risk in an unexposed population plus the radiation risk, multiplied by 100.

2. The baseline risk of cancers in a society is influenced by many factors but most importantly by age at diagnosis and sex. Taking into account the improved survival experience of cancers and other disorders over time it is important to use baseline information pertinent to the relevant dates. For UK calculations, ONS age standardised baseline risks at different dates for men and women are available.

3. The epidemiological evidence that radiation can cause cancer derives from many sources as discussed above, and where there is evidence that a cancer can be caused by radiation (i.e. it is radiogenic) International organisations, e.g. International Atomic Energy Authority (IAEA) and ICRP have developed risk models for all solid cancers as a group and for various individual cancer sites where the evidence of radiogenicity is strong (1) (2). In the UK, ICRP recommendations inform the worker and public radiological protection regulations, and the 2007 risk models apply to cancer risk estimates. Because calculated risk estimates are only available for radiation doses typically much larger than of interest in the context of occupational injury and compensation, and rarely from the population of interest, in 2004 an ICRP Task group considered the Low-dose extrapolation of radiation- related cancer risk and how one might fairly and reasonably, in terms of scientific certainty, calculate risk at low dose. They looked at the epidemiological evidence including dependence on radiation dose and the existence of a dose response. Based on acute doses in the moderate to high dosage range, the review covered modification of dose response by age and sex, lifestyle factors, population and radiation quality (3).

4. The atomic bomb high-dose survivor data show a radiation dose low LET response relationship that is linear for solid cancer with doses from 2 Gy to 200 mGy, while the evidence below 100 mGy is equivocal, neither confirming nor refuting linearity. For leukaemia, the data support a linear quadratic response relationship, i.e. risk reduces at low dose. ICRP 1991 and UNSCEAR 1993 recommended that, for low and very low doses, dose-specific risk estimates should be divided by a DDREF of 2 with no DDREF applied to leukaemia modelling. ICRP 2007 report maintained that approach, taking the shape of the response models for the 12 site-specific cancers and the general cancer model as LNT. For each site, there are two risk models based on absolute and relative risk. This is because although the risk per unit dose is assumed to be the same at all doses, there is little evidence of excess cancer risk in populations exposed to very low doses, e.g. 10mGy or lower.

5. **Absolute risk** is the probability a given radiogenic cancer will occur at a given radiation dose while the **Relative risk** considers the risk, i.e. numbers of cases in the exposed population relative to the baseline risk. The reason for the different risk models based mainly on the atomic bomb studies is that the baseline risk of cancers is different in different populations and it is not known how to apply such information between the different populations. While **Absolute risk** is not altered by baseline risk, that is not so for **Relative risk**. The convention, in calculating PoC, is to use an average of the two. The **Excess Absolute Risk** is the different rates of occurrence between an exposed and unexposed otherwise comparable population, while the **Relative Risk** (RR) is the occurrence rate in the exposed population compared with that in the non-exposed population. The **(ERR)** is RR -1, i.e. the Excess Absolute Risk. A RR of 1 for a disorder means that radiation is unlikely to be a causal factor. On the other hand, the absolute risk model provides a value between 0 and 1. This is the probability that a given cancer is due to the exposure of interest. If 1, the causal relationship is certain, while as the figure approaches 0 it is increasingly likely that the exposure played no part.

References:

(1) IAEA – Tech – Doc 870 (1996) Methods for estimating the probability of cancer from occupational radiation exposure.

(2) ICRP (2007) The 2007 recommendations of the ICRP. ICRP pub.103 Ann. ICRP 37(2-4)

(3) ICRP (2005) Low dose extrapolation of radiation-related cancer risk. ICRP pub. 99 Ann. ICRP 35(4)

Annex B

UK atmospheric nuclear tests

1. A nuclear explosion first produces a rapid initial burst of intense light/heat and subsequent air blast. The flash of light can cause 'flash-blindness' at considerable distances and permanent eye injury at shorter ranges. The heat from a nuclear detonation can cause first-degree burns to exposed human skin at ranges up to a few kilometres from a 10 kiloton detonation or approximately 20 kilometres from a 1 megaton detonation). The air blast is unlikely to cause injury to a person more than 3 kilometres from a 10 kiloton burst or 6 kilometres from a 1 megaton burst. At the UK trials, protection to personnel included careful mustering of personnel at distances considered safe, as well as eye protection and anti-flash clothing where indicated. Items were secured, moved or partly dismantled (e.g. tentage) and windows in buildings left open to avoid glass breakage and subsequent injury due to flying shards.

2. The ionising radiation exposure associated with nuclear detonations is of three types:

    a) Firstly, radiation emitted by the device as it explodes (known as 'prompt' radiation). This is absorbed by the air over distances of a few kilometres, i.e. the general area devastated by the nuclear explosion. To be sufficiently close to receive a significant dose of 'prompt' ionising radiation, a person would also be within the lethal range of the air-blast and heat. This, therefore, does not need to be considered as contributing to a participant's radiation dose.

    b) However, neutrons from prompt radiation irradiate the surrounding ground producing short-lived 'neutron-activated' activation products, radioactive isotopes in the soil. These are highly radioactive with half-lives measured in hours. They generally emit beta and gamma radiations. At the UK tests, following a detonation, both aerial- and ground- based radiation surveys were undertaken by specialist teams. Controlled areas were then established with checkpoints where required personnel could only enter wearing personal dosimetry and suitable protective clothing. Such teams then worked in the area for specified periods to recover instruments and records. Careful monitoring ensured adherence to the radiological safety instructions issued for participants.

    c) Radiation is also emitted by the remains of the exploded device and fallout (where ground materials are entrained by the explosion, made radioactive and thus dispersed by the explosion and ensuing winds).

3. UK trial detonations were carried out at altitude to minimise drawing ground materials into the explosion. Planning also took account of weather to disperse debris into the higher atmosphere and carry it away from the detonation site. All UK atmospheric nuclear trials devices produced yields at, or very close to design figures and took place at appropriate altitudes. There is documented evidence that individual trials were postponed to ensure they took place in the correct meteorological conditions. Subsequent monitoring confirmed that

detonations were as ‘clean’ as planned in respect of fallout.

4. Specialist instrumentation was used to measure ionising radiation. Personal dosimeters, in the form of film badges, estimate the dose to an individual from gamma radiation and beta particles. In general usage, these were typically carried for a month. During post-detonation operations, film badges were issued for an individual day/task. The film badge consisted of a piece of photographic film, sealed in a light-tight package bearing a unique number, the whole contained in a cassette adapted for securing to the clothing. Exposure to ionising radiation causes a chemical change within the film. After conventional photographic development, the film is compared with a ‘standard’ (where the degree of darkening to the film can be related to the amount of incident radiation required to produce such darkening) and a measure of dose to the individual obtained. It is primarily sensitive to photons (gamma rays and X-rays), less so for beta particles and low-energy neutrons and is not sensitive to alpha particles.

5. Although film badges provided an individual’s dose, they required processing and could not provide an ‘on the spot’ dose measurement. For this purpose, quartz fibre electroscopes (QFE) could be issued to measure incident gamma (only) radiation. Once a pre-determined level had been reached, personnel would leave the controlled area, and submit their film badges for assessment. From the original dose records, it can been seen where both film badge and QFE dose data are available for the same individual, then the resulting measured dose values are similar.

6. Doses of ionising radiation can also arise by internal contamination, through breathing air containing contaminated dusts. Although alpha-emitting materials (e.g. uranium and plutonium as part of exploded device components) would be the most hazardous in this respect, such would constitute a very small component (if any at all) of fallout compared with beta and photon-emitting materials generated by a nuclear detonation. The risk of internal dose was minimised at the UK trials by the planning as described above i.e. ensuring that only essential, fully-protected personnel entered areas where internal contamination was possible, and by minimising activation products and fallout.

7. Neither a film badge nor a QFE could measure internal contamination/dose directly. However, to receive a significant internal dose, an individual would have to enter an area where there were high levels of fallout emitting photon and beta radiation. It is highly unlikely that this could happen without at the same time there being a measurable **external** dose as would subsequently be indicated by his film badge dose measurement. The only exception to this might have been at some of the Minor Trials, particularly Vixens A and B.

8. The Atomic Weapons Establishment, Aldermaston, holds the film badge records of the test participants. Film badges were not issued to all personnel; the Ministry of Defence estimates that approximately 20% of total participants were issued with film badges. At the earlier trials, e.g. operation Hurricane (1953) almost all participants were issued with film badges. The majority detected nil dose and by operation Grapple in 1957, a more targeted approach was in place with badges issued only to those whose duties or location were likely to put

them at risk. About 20% overall had personal dosimeters.

"At risk" groups

9. Not all of those monitored showed a measurable dose above the detection threshold of the film badge. In fact, a majority were found to have a measured dose of 'nil'. The records show that less than 1000 of the doses recorded were 1 mSv or above: 81 received 50 mSv or more and 37 more than 100 mSv. From information held, on the location and operation of those with measured doses, certain groups are identified as being more liable to be exposed to significant doses of radiation. These are:-

i) RAF aircrews involved in sampling from airburst clouds (205 men). Mosaic. Totem. Buffalo. Antler. Grapple.

ii) RAF decontamination flight crews who sluiced the aircraft (129 men).

iii) RN personnel on HMS Diana when she sailed through the fallout at Operation Mosaic (282 men).

iv) The officers of the Buffalo Indoctrinee Force and Target response group. They assembled to observe at first hand the effects of the detonation (249 men).

v) Others – with recorded exposures greater than zero (1123 men).

10. The records also identified those men present at the Minor Trials who were at highest risk of radionuclide ingestion or inhalation. There were 847 in total. In the NRPB study, this group was considered separately. It did not show any increased risk of multiple myeloma, leukaemia or other malignancies relative to the rest of the participant group. When analysed as part of the main study, this group was indistinguishable from other participants. However, it is acknowledged that at some of the Minor Trials, notably Vixen A and B, there was some risk of dispersal of radiation into the environment because of explosions on the ground or on low towers. As a result, the Secretary of State has added to the "at risk" groups where service-related ionising radiation exposure is recognised, regardless of direct dose measure or estimate:

vi) Those present at the Minor Trials at Vixen A and B and the clean-up operations.

Impact on Secretary of State's policy for radiogenicdisorders, cancers, circulatory disorders and cataract where service-related ionising radiation exposure is contended

11. Where claims for radiogenic disorders are made by personnel who took part in any of the activities listed, or otherwise as above, the Department will accept that there is reliable evidence of service exposure to ionising radiation. Certifying entitlement for claimed disablements will depend on the case facts, including the measured or estimated dose exposure and, as required, calculated PoC.

Annex C

Ionising radiation and circulatory disease

1. Until the 1960s the heart and blood vessels were thought to be completely resistant to ionising radiation (1). Since then, many reports have appeared describing inflammation of the heart lining, and conduction disorders, from damage to the electrical system following high-dose (of the order of 40 + Gy) mediastinal irradiation of malignant tumours. Today these effects of high-dose ionising radiation exposure are generally accepted and reflected in treatment programmes.

2. There is a significant literature on the biological mechanisms of radiation-related circulatory disease. Much of this work is animal-based and there remain gaps in understanding. The AGIR report on circulatory diseases reviews the evidence. Some principles are emerging, including that radiation has an effect on the inflammatory response. At high dose it increases the inflammatory response while at low dose the inflammatory response is dampened down. The heart itself is relatively resistant to irradiation, and clinical changes, signs and symptoms can present, particularly vessel occlusion some time after irradiation (2).

3. In 1958 a human case study reported a myocardial infarction following deep X-ray therapy (3) and since then there have been many reports linking death due to coronary disease following radiotherapy for medical conditions including Hodgkin's disease and breast cancer (4) (5). In most of these studies, confounders were present, e.g. they did not control serum cholesterol, blood pressure or cigarette smoking, and the study subjects were already ill and in some cases had chemotherapy.

4. Further information from long-term follow-up studies of heavily irradiated populations (6) has shown excess mortality from circulatory disease, especially myocardial infarction in these populations. There are also case reports of cerebral infarction following radiotherapy to head and neck and of peripheral vascular disease of the lower limbs following pelvic irradiation (7). However, these effects have again only been reported with large dose of ionising radiation (20-60 Sv). Studies involving up to 20 years' follow-up of patients irradiated according to more recent radiotherapy procedures have shown no significant difference in myocardial infarction death rate between irradiated and control populations. A detailed discussion of the evidence was presented in a review paper by Kodama (8).

5. An American 50-year follow-up study of 90,000 radiologic technicians suggested that in those who started practice before 1940 there was increased risk of circulatory disease, mainly cerebrovascular disease, compared with those beginning after 1960 (9). However, a British 60-year follow-up study of 25,000 radiologists did not confirm this effect. For radiologists registered during 1897-1921, mortality from circulatory disease was lower than in other medical practitioners with no trend in date of registration (10). Similarly, follow-up studies of 14,500 patients treated with deep X-ray therapy for ankylosing

spondylitis over 30-50 years suggested no increase in coronary deaths (11).

6. Most follow-up studies have focussed on mortality rates, subject to many uncertainties and inaccuracies. A more accurate estimate of the association would come from incidence studies in large populations with lengthy follow-up and controlled classic risk factors.

7. The issue of the association between ionising radiation and stroke or coronary heart disease in non- medical settings has been addressed periodically in the atomic bomb studies. The findings have varied over time and it must be acknowledged that other factors such as baseline risk and generational effect as well as malnutrition, presence of other injuries and burns, may have played a part. Until the report summarising the results for the period 1950-70 (12), there was no suggestion of a relation between atomic bomb radiation exposure and mortality from stroke or coronary disease. That analysis reported an increased mortality from coronary disease in women exposed to 100 mSv or more. The increase was particularly marked where dose exceeded 500 mSv. The trend was not however confirmed in the subsequent report for the period 1950-1978 (13), although this did show increased mortality from "all diseases other than cancer" where exposure exceeded 2000 mSv. The report on the period 1950- 85 (14) used a new method of exposure dose estimate, and showed clearly increased mortality from circulatory disease, including stroke and cardiac disease but again only in heavily exposed survivors.

8. The issue of accuracy of death certificates for the Radiation Exposure Research Foundation studies has been examined (15) and it is apparent that death certification for circulatory disease is less accurate than for malignancies. In addition, in these mortality studies the classic known cardiac risk factors cannot be controlled.

9. A few studies have been published which look at the **incidence** of coronary heart disease and stroke in relation to ionising radiation exposure associated with the atomic bombs, again with varied results. For the period 1958-1964, in an early study, Johnson et al (16) found no association. The later report covering the period 1950-1970 suggested an increase of stroke and coronary disease in females heavily exposed (over 2 Sv) in Hiroshima. The effect was not seen in men or in Nagasaki survivors (12).

10. Kodama's 1994 study (16), now covering the period up to 1990, again confirmed an increase in myocardial infarction incidence in heavily-exposed survivors regardless of age, gender or location, although the excess of myocardial infarction was very small compared with the excess of cancers in the population. The relative risk of myocardial infarction at 1 Sv exposure was 1.17. The associated p value is 0.02 with a confidence interval (95%) of 1.01-1.36. Lifestyle risk factors for coronary disease were not adjusted for.

11. In 2004 generally statistically non-significant excess risks were found for incidence of myocardial infarction and hypertension in a follow-up of the Adult Health Study subgroup of atomic survivors (17). Outcomes of other studies of nuclear workers (18) and Mayak workers (19), while suggesting a positive

association, show considerable heterogeneity and in most of these study groups there was again no or only limited adjustment for the major cardiovascular risk factors. The most recent Japanese follow-up mortality study, which updates to 2003, does adjust for the major lifestyle and other factors, and reports significantly elevated circulatory disease risk at doses above 0.5 Sv (20), while a 2001 update confirms the causal link to high-dose radiotherapy with doses of the order of 40 Gy, and most commonly seen in those irradiated as children (21).

12. In conclusion, at this date, it is accepted that circulatory disorders, including stroke, coronary artery disease and heart failure may be caused by ionising radiation exposure in high doses, i.e. 500 mSv or more. Below that dose, while the evidence is suggestive, the studies are heterogeneous and not always statistically significant. Most do not adjust for the major known cardiovascular risk factors.

Impact on Departmental normal policy for claims for circulatory disorder due to service-related ionising radiation exposure

13. Claims for circulatory disorders, stroke, myocardial infarction, and cardiac failure linked to service- related ionising radiation exposure will be considered on their case-specific evidence including measured or estimated exposure dose. The literature will continue to be monitored.

References:

(1) Warren, S. (1942) Effects of radiation on the cardiovascular system. Archives of Pathol. 34: 1070-1079

(2) AGIR (2010) Circulatory disease risk. Doc HPA RCE-16, 1-116

(3) Pearson, H. (1958) Incidental changes of X-ray therapy. Lancet, I: 222

(4) McReynolds, R. A. et al (1976) Coronary heart disease after mediastinal irradiation for Hodgkin's disease. Am. J. Med. 60: 39-15

(5) Host. H. et al (1986) Post-operative radiotherapy in breast cancer: Long-term results from the Oslo Study. Int. J. Rad. Oncol, Biol. Phys. 12: 727-732

(6) Tracy G. P. et al (1974) Radiation-induced coronary artery disease. J. Am. Med. Ass. 228: 1660-1662

(7) Pettersson, F. et al (1990) Atherosclerotic occlusive disease after radiation for pelvic malignancies.
Act. Chir. Scan. 156:367-71

(8) Kodama, K. (1995) Circulatory diseases. Shigematsu, I. et al. (eds). Effects of A-bomb radiation on the human body. Tokyo, Japan. Harwood Academic Publishers. Pp 182-194.

(9) Hauptmann, M. et al (2003) Mortality from diseases of the circulatory system in radiologic technologists in the United States. Am. J. Epidemiol. 157: 239-48

(10) Berrington, A. et al (2001) 100 years of observation on British radiologists:

mortality from cancer and other causes 1897-97. Brit. J. Radiol. 74: 507-19

(11) Darby, S.C. (1987) Long term mortality after a single treatment with X-rays in patients treated for ankylosing spondylitis. Brit. J. Cancer 55:179-90

(12) Jablon, S. et al (1971) Life Span Study report 6 – Mortality among A-bomb survivors. 1950-70 ABCC TR10-71

(13) Kato, H. et al (1981) Life Span Study report 9 part 2 – Mortality from causes other than cancer among atomic bomb survivors 1950-78. RERF TR5-81

(14) Shimizu, Y. et al (1992) Studies of the mortality of atomic bomb survivors 9. Mortality 1950-85.
Non-cancer mortality based on the revised doses (DS86). Radiat. Res. 130(2) 249-66

(15) Carter, R.L. et al (1991) Combining diagnostic categories to improve agreement between death certificate and autopsy classification of cause of death for atomic bomb survivors, 1950-87. RERF technical report. 15-91. RERF

(16) Johnson, K.G. et al (1966) Coronary heart disease in Hiroshima – report of a six- year period of surveillance. 1958-64 ABCC TR24-66)

(17) Kodama, K. (1994) Cardiovascular disease in atomic bomb survivors. RERF update, 5(4): 3-4

(18) Yamada, M. et al (2004) Non-cancer disease incidence in atomic bomb survivors, 1958-98. Radiat.
Res. 161:622-32

(19) Muirhead, C.R. et al (2009) Mortality and cancer risk following occupational radiation exposure: third analysis of the National Registry of radiation workers. Brit. J. Cancer 100:206-12

(20) Azizova, T.V. et al (2010) Cardiovascular diseases in the cohort of workers first employed at Mayak PA in 1948-58. Radiat. Res. 174(2):155-68

(21) Shimizu, Y. et al (2010) Radiation exposure and circulatory disease risk: Hiroshima and Nagasaki atomic bomb survivor data 1950-2003. BMJ 340:b5349

(22) Yusuf, S.W. (2011) Radiation-induced heart disease – a clinical update. Cardiol. Res. Practice Feb 27:317659

Radiation and cataract

1. There are several causes of lens opacification and development of sight-limiting cataract. These include ageing, diabetes, treatment with oral corticosteroids, trauma to the eye, family history and radiation of various types, e.g. ultraviolet, infrared and ionising. Studies of lens changes and cataract formation are difficult to interpret because of lack of agreed definitions and end points used in the studies. Typically, they focus on lens opacification rather than disabling cataract. The mechanism and longitudinal course of lens opacification is not yet understood and, in particular, whether lens opacification inevitably produces disabling visual loss. In the context of compensation awards, cataract is a treatable disorder with high rates of return to normal visual acuity following operative treatment. It is also not established whether the radiation effect is deterministic with a threshold exposure dose below which lens opacification does not take place, or whether it is in fact stochastic with no level of ionising radiation exempt from some level of risk. In 2007, the ICRP, assuming the process to be deterministic, set the threshold radiation dose for detectable lens opacity at 5 Sv for chronic exposure and 0.2- 2 Sv for acute exposure, with higher doses estimated at 2-10 Sv single acute exposure required for disabling effects (1). More recently, on further review of the evidence, the ICRP has concluded that the lens is more radiosensitive than formerly assessed, and the threshold for chronic exposure has been revised downward to 0.5 Sv for chronic exposures (2) (3).

References:

(1) ICRP (2007) The 2007 recommendations of the International Commission on Radiological Protection. ICRP publication 103 Ann. ICRP 37

(2) ICRP (2012) ICRP Statement on tissue reactions and early and late effects of radiation in normal tissues and organs – threshold doses for tissue reactions in the radiation protection context. Ann. ICRP 41:1-322

(3) Bouffler. A. et al (2012) Radiation-induced cataracts – the Health Protection Agency's response to the ICRP statement on tissue reactions and recommendations on the dose limits for the eye lens. J. Radiol. Prot 34: 479-88

Impact on Departmental normal policy in claims for cataract due to service-related ionising radiation exposure

2. Claims for cataract linked to service-related ionising radiation exposure will be considered on their case-specific evidence including measured or estimated exposure dose and as required, calculated PoC. The literature will continue to be monitored.

Glossary

Absorbed dose see **dose**.

Acute radiation syndrome (ARS) The onset, within hours of high-**dose** whole-body **irradiation**, of nausea and vomiting followed by destruction and diminished (or absent) replacement of essential blood cells resulting in vulnerability to serious infection and bleeding; recovery is possible but with increasing **doses** these effects are more severe and death more likely.

Alpha particle A particle consisting of two protons plus two neutrons, emitted by a radionuclide. Alpha particles are produced following spontaneous decay of certain radioactive atoms, such as radium, plutonium, uranium, and radon. Because of its large mass and positive charge, an alpha particle can usually travel only a short distance – less than 1 mm – in water. A single piece of paper can stop an alpha particle effectively. Therefore, health effects of alpha exposures appear only when alpha- emitting materials are ingested (i.e. internal exposure).

Background radiation Ionising radiation from naturally occurring **radionuclides** both in the environment (from soil, rock and building materials and from space – cosmic radiation) and in the body.

Beta particle An electron emitted by the nucleus of a radionuclide. The electric charge may be positive, in which case the beta particle is called a positron. Beta particles are produced following spontaneous decay of certain radioactive materials, such as tritium (an isotope of hydrogen), carbon-14, phosphorus-32, and strontium-90. Depending on its energy (i.e. speed), a beta particle can traverse different distances in water – less than 1 mm for tritium to nearly 1 cm for phosphorus-32. As with alpha particles, the major concern for health effects is after their ingestion (i.e. internal exposure).

Contamination The suspension in air or deposition of **radionuclides** upon, or in, the ground, water and other surfaces, and personnel and equipment.

- **External contamination** Of a person – deposition, general or localised, of **radionuclides** upon all, or any, of clothing, hair, skin and/or equipment.

- **Internal contamination** Of a person – deposition within the body, usually by inspiration, by ingestion or sometimes through penetration of (usually broken) skin by **radionuclides** which will then **irradiate** the cells of surrounding body tissues.

Cosmic rays High-energy ionising radiation from outer space.

Decay The process of spontaneous transformation of a radionuclide. The decrease in the activity of a radioactive substance.

Dose The amount of **ionising radiation** received as deduced from the energy absorbed from an external radiation source.

- **Absorbed dose** Quantity of energy imparted by ionising radiation to unit mass of matter such as tissue. Unit gray, symbol Gy. 1Gy = 1 joule per kilogram.

- **Equivalent dose** The quantity obtained by multiplying the absorbed dose by a factor to allow for the different effectiveness of the various ionising radiations in causing harm to tissue. Unit sievert, symbol Sv.

- **Effective dose** The quantity obtained by multiplying the equivalent dose to various tissues and organs by a weighting factor appropriate to each and summing the products. Unit sievert, symbol Sv.

Dosimeter A small device worn on the person to measure absorbed energy and from which a record of **Absorbed Dose** may be obtained.

Dosimetry The estimating, recording and maintaining of records of **dose**.

Emitter A **radionuclide** decays by emission of certain radioactive particles and/or electromagnetic radiation. A particular **radionuclide** may be described as an **alpha** or **beta** or **beta/gamma** emitter.

Fallout The transfer of radionuclides produced by nuclear weapons from the atmosphere to earth.

Fission products The two, invariably radioactive, fragments remaining after an atom has been split (undergone fission).

Gamma ray A discrete quantity of electromagnetic energy without mass or charge, emitted by a radionuclide. Cf X-ray. A gamma ray is similar to ordinary visible light but differs in energy or wavelength. Sunlight consists of a mixture of electromagnetic rays of various wavelengths, from the longest, infrared, through red, orange, yellow, green, blue, indigo, and violet, to the shortest in wavelength, ultraviolet. A gamma ray's wavelength is far shorter than ultraviolet (i.e. it is far higher in energy). Gamma rays are produced following spontaneous decay of radioactive materials, such as cobalt-60 and caesium-137. A cobalt-60 gamma ray can penetrate deeply into the human body, so it has been widely used for cancer radiotherapy.

Ionising radiation Radiation that produces ionisation in matter. Examples are alpha particles, gamma rays, X-rays and neutrons. When these radiations pass through the tissues of the body, they have sufficient energy to damage DNA.

Ionisation The process by which a neutral atom or molecule acquires or loses an electric charge. The production of ions.

Lag time the period from first radiation exposure of a population or individual

to the time when a radiation relation effect could be observed, typically a minimum of two years for leukaemia and a minimum of five years for solid cancers.

Linear No Threshold model (LNT) is a model used in radiation protection to quantify radiation risk. It assumes that the long-term risk is directly proportional to the dose. It defines that radiation is always considered harmful with no safety threshold, and the sum of several very small exposures is considered to have the same effect as one larger exposure (response linearity).

Monitoring The process of searching for the presence of and then measuring, reporting and recording radiation **dose rates** found within a given area or on a person.

Neutron A nuclear particle (similar to a hydrogen atom but without electrical charge), emitted during fission and fusion by only a few **radionuclides**; long range (kilometres) in air and highly penetrating; an external hazard only at detonation; densely **ionising**.

Non-ionising radiation Radiation that does not produce ionisation in matter. Examples are ultraviolet radiation, light, infrared radiation and radiofrequency radiation. When these radiations pass through the tissues of the body they do not have sufficient energy to damage DNA directly.

Radiation weighting factor (RWF) A factor intended to take account of the relative biological effectiveness of different types of radiation according to both their energies and how densely ionising they are.

Radionuclide An unstable nuclide that emits ionising radiation.

Relative Risk the rate of disease (incidence or mortality) in an exposed group divided by the rate in an unexposed group. (Usually standardised to adjust for differences in factors such as age and sex between the two groups).

Excess Relative Risk Excess relative risk is expressed as relative risk (RR) minus one, or that portion of the RR accounted for by the particular risk factor under study – i.e. radiation exposure.

Attributable Risk Attributable risk refers to the fraction of diseases or deaths that is estimated to result from exposure to radiation. It increases with dose.

Standardised Mortality Ratio SMR – Useful for comparing deaths in population of interest with that in a standard population

$$SMR = \frac{\text{observed deaths}}{\text{expected deaths}} \times 100$$

SMR < 100 fewer deaths than expected
SMR > 100 more deaths than expected

X-ray A discrete quantity of electromagnetic energy without mass or charge. Emitted by an X-ray machine. Cf gamma ray. X-rays have the same characteristics as gamma rays, although they are produced differently. When high-speed electrons hit metals, electrons are stopped and release energy in the form of an electromagnetic wave. This was first observed by Wilhelm Roentgen in 1895, who considered it a mysterious ray, and thus called it an X-ray. X-rays consist of a mixture of different wavelengths, whereas gamma ray energy has a fixed value (or two) characteristic to the radioactive material.

Abbreviations

ICRP – International Commission on Radiological Protection

NIOSH - The National Institute for Occupational Safety and Health

(US Federal Agency) NRPB - National Radiological Protection Board