IN THE FIRST-TIER TRIBUNAL **WPAFCC Refs: as below**
WAR PENSIONS AND ARMED FORCES COMPENSATION CHAMBER

Sitting at Royal Courts of Justice, Strand, London, WC2A 2LL

Date: 16th December 2016

TRIBUNALS COURTS AND ENFORCEMENT ACT 2007

TRIBUNAL PROCEDURE (FIRST-TIER TRIBUNAL) (WAR PENSIONS AND ARMED FORCES COMPENSATION CHAMBER) RULES 2008

BEFORE:

THE HON MR JUSTICE BLAKE

MRS I MCCORD

DR J RAYNER

BETWEEN

1. LEONARD ABDALE (Deceased)	**ENT/00203/2015**
2. DARRYL BEETON	**ENT/00202/2015**
3. TREVOR BUTLER (Deceased)	**ENT/00258/2015**
4. DEREK HATTON (Deceased)	**ENT/00200/2015**
5. ERNEST HUGHES	**ENT/00254/2015**
6. BRIAN LOVATT	**ENT/00201/2015**
7. DAWN PRITCHARD (Deceased)	**ENT/00258/2015**
8. LAURA SELBY	**ENT/00199/2015**
9. DENIS SHAW (Deceased)	**ENT/00253/2015**
10. JEAN SINFIELD	**ENT/00204/2015**
11. DONALD BATTERSBY (Deceased)	**ENT/00250/2015**
12. ANNA SMITH	**ENT/00251/2015**
	Appellants

- and -

SECRETARY OF STATE FOR DEFENCE

Respondent

Hearing Dates: 13 to 30 June 2016

Representation:

Roger Ter Haar QC and Richard Sage (instructed *pro bono* by **HOGAN LOVELLS**) for Appellants 1 to 10.

Christopher Busby, Hugo Charlton and Cecilia Busby acting as *pro bono* lay representatives for Appellants 11-12.

Adam Heppinstall and Abigail Cohen instructed by the **Government Legal Department** for the Respondent.

TRIBUNAL'S DECISION AND REASONS

The unanimous DECISION of the Tribunal is: the appeal of each appellant is dismissed save for the appeal of Leonard Abdale deceased in respect of his claim for cataracts. On this issue his appeal is allowed.

INDEX TO DETERMINATION

PART ONE: INTRODUCTION

Outline of the issues in these appeals:

1. Each of these appeals is brought by or on behalf of former members of the armed forces (the veterans) who took some part in the atmospheric testing of nuclear weapons conducted by the United Kingdom between 1952 and 1958. Of the twelve veterans all but three have died and the appeals of the deceased veterans are brought or pursued by their widows or other family members. Each appeal concerns a claim to a war pension by reason of a specific medical condition that is claimed to have arisen as a consequence of service in this test programme.

2. The UK test programme started in October 1952 off the coast of Western Australia (Operations Hurricane, Totem and Mosaic)[1].

3. In September 1956 the Buffalo series of tests took place at the Maralinga Range, a desert area in South Australia. Of the twelve veterans with which these appeals are concerned only Donald Battersby served at Maralinga. He did so as a member of the RAF between 26 July and 19 November 1956 when he was aged 20. In 2005 he was diagnosed with chronic lymphatic leukaemia (CLL). He was also diagnosed with pancreatic cancer but this appeal is not concerned with a claim for that condition.

4. All the other veterans served at Christmas Island (Kiritimati), a coral island about 60 kilometres long and between 6 and 30 kilometres wide that is located a few degrees north of the Equator, in what is now the Republic of Kiribati. At the time it was selected for the tests Kiritimati was an uninhabited island, although a few hundred people from the indigenous population of the surrounding islands (the Gilbertese) came to Port London in the north west of the island to work under labour contracts and were subject to the supervision of the British authorities.

5. Between May and June 1957 there were three tests in the Grapple Series over the ocean off Malden Island in the Pacific Ocean, some 700 kilometres from Christmas Island (CI).

6. On 8 November 1957 the next detonation in the Grapple series took place; Grapple X was a high altitude air burst over the ocean just off CI.

[1] An informative history of the test programme was supplied to us in the form of extracts from Laura Arnold 'Britain and the Bomb' (2001) Palgrave (SB 17/5, 6, and 7). The full text was available in the FTT Bundle B7/34

7. On 28 April 1958, the most powerful detonation in the whole series took place; Grapple Y, another air drop at high altitude (estimated at 2500 metres or 8100 feet) with an explosive yield of 3 megatons. The detonation point was over the ocean 1.5 kilometres off the south eastern tip of CI, some 35 kilometres from the main camp and the neighbouring populated area of the island in the north west of the island.

8. Between August and September 1958, four smaller detonations completed the Grapple series, collectively known as Grapple Z. Two of these (Pennant and Burgee) were airbursts over land on Christmas Island detonated at 450 metres from a balloon with yields of 24 and 25 kilotons respectively. The other two (Flagpole and Halliard) were air dropped over the ocean off CI at heights of 2800 metres and with yields of 1 and 0.8 megatons respectively.

9. Thereafter, British testing of nuclear weapons was halted pending the making of an international treaty where signatory states agreed to stop such atmospheric testing.

10. Following the last British test on 23 September 1958, CI was used by the US Government for a series of high yield nuclear tests known as Operation Dominic between 1962 and 1964. Thereafter the military base was abandoned. There were various clean-up operations following the conclusion of British and US testing.

11. The veterans who were stationed on CI are as follows[2]. We set out here brief details of their service and medical conditions. Greater examination will be given to their medical conditions in Part Seven.

    (i) Leonard Abdale was at CI from 15 January 1958 to 29 November 1958 aged 22 to 23. He was thus on the island at the time of Grapple Y and the Grapple Z detonations. He served as a wireless operator at the Joint Operation Centre at the Main Camp. In 2000 and 2001 he had operations for cataracts and in 2006 was diagnosed with bladder cancer. He made a claim for both conditions in 2009.

    (ii) Daryl Beeton served at CI from 27 August 1957 to 10 August 1958. He was aged 19 to 20 and would have been there for the Grapple X and Y detonations only. He served as a Leading Aircraftman and had duties as a cook at the kitchens at the Port and Main camps. In 2001 and 2003 he had myocardial infarctions and had a coronary artery

[2] The following data has been abstracted from the medical and service history documentation before us

bypass graft in 2008. The underlying disease process was assessed as atherosclerosis.

(iii) Trevor Butler served at CI from 30 December 1957 to 12 December 1958 when he was aged 19 to 20. He was also there for the Grapple Y and Z detonations. He was a field engineer with the Royal Engineers. In 2006 he was diagnosed with a range of non-carcinogenic conditions including immune system dysfunction.

(iv) Derek Hatton served at CI from 21 August 1958 to 15 August 1959 aged 19 to 20. He served with the Royal Army Ordnance Corps. In 2007 he was diagnosed with polycythaemia rubra vera (PRV).

(v) Ernest Hughes served at CI as a ground wireless fitter with the RAF from 29 October 1956 to 10 September 1957 when aged 22 to 23. He was thus only present during the early Grapple series 700 kilometres away and had left the area before Grapple X and Y. In 1991 he was diagnosed with myocardial infarction and in 2006 with bladder cancer.

(vi) Brian Lovatt served at CI in the RAF from 9 September 1957 to 9 June 1958 aged 20. He was therefore present at the time of Grapple X and Y. In 2005 he was diagnosed with atherosclerosis and myocardial infarction.

(vii) William Pritchard served at CI as an RAF telegraphist between 24 January and 28 August 1958 at the time of Grapple Y and the first of the Grapple Z tests. He was then posted 330 kilometres away before returning to CI between 3 October and 28 November 1958. He died in 2005 of bronchopneumonia, cardiorespiratory failure and end stage renal failure. Other conditions including Berger's Nephropathy, hypertensive heart disease, type II diabetes and arterial atheroma were found.

(viii) Charles Selby served at CI with the Royal Engineers between 10 August 1957 and 20 July 1958 aged 22 to 23 at the time of Grapple X and Y. In 2000 he was diagnosed with type II diabetes and in 2003 with idiopathic fibrosing alveolitis.

(ix) Dennis Shaw served with the Royal Engineers at CI between 16 December 1958 and 27 November 1959 aged 21 to 22. He was thus not present at any of the tests. He was found to have bi-lateral nuclear sclerosis in 2005 and subsequently diagnosed with a sub-capsular cataract in the left eye.

(x) Herbert Sinfield served as a driver with the Royal Army Service Corps at CI between 10 June 1958 and 12 June 1959, and was there for the Grapple Z tests. In 2005 he was diagnosed with non-Hodgkin large cell lymphoma (NHL).

(xi) Barry Smith served as a catering assistant with the RAF at CI from 30 October 1959 and 27 July 1960 and also from 3 August and 11 November 1960 aged 20 to 21. Whilst there he also worked as camp barber. He developed pancreatic cancer that was diagnosed in 2007.

12. Each appellant says that these medical conditions arose as a result of the military service of the relevant veteran in either Australia or CI. They appeal against decisions made by medical advisers on behalf of the Secretary of State for Defence. In the opinion of the medical advisers there was no causal link between the military service and the medical condition claimed.

13. It is common ground that under Article 41 of the Naval Military and Air Forces (Disablement and Death) Service Pensions Order 2006 (SPO), made under s12(1) Social Security (Miscellaneous Provisions) Act 1977, a disablement or death shall be certified as attributable to military service if the qualifying conditions are met. Under Article 41(5):

> 'Where, upon reliable evidence, a reasonable doubt exists whether the conditions ... are fulfilled, the benefit of that reasonable doubt shall be given to the claimant.'

14. Each appellant contends that a reasonable doubt has been shown that, during the periods of service noted above, the veteran was exposed to ionising radiation resulting from detonations in the test programme. Although there is no evidence at the time that any of these veterans was exposed to radiation from the detonations, it is submitted that prolonged exposure to radiation by inhalation or ingestion of radioactive particles deposited on the land or in the sea off CI is a real possibility. Equally, it is contended that reasonable doubt exists as to whether any such exposure was a cause of the medical conditions claimed.

15. In the appeals relating to Messrs Battersby and Smith, Dr Busby, on their behalf, advances a more radical submission that that the guidance issued by the International Commission on Radiological Protection (ICRP) that forms the basis of radiological health protection in the UK and the EU is flawed and underestimates risks to health from internal exposure to radiation and in particular radiation from uranium.

16. In outline, the Secretary of State's position in these appeals is that, even if the possibility of some exposure to ionising radiation above background levels cannot be excluded, a conservative and cautious approach to the estimation of dose from all potential radiation pathways arising from military service produces such a low effective dose that the Tribunal, in evaluating these matters for itself, should conclude that no reasonable doubt exists for any of the appellants. In particular it is submitted:

    (i) None of the veterans (with the possible exception of Mr Battersby at Maralinga) were involved in duties that took them close to areas where exposure to ionising radiation was both expected and detected. This contrasts with the position of others including the MOD scientists observing the tests at ground zero; RAF pilots who were directed to fly through the mushroom cloud of Grapple X and Y to collect samples of radioactive residue for subsequent analysis or RAF personnel involved in washing and decontaminating aircraft that had contact with the radioactive cloud.

    (ii) The exception in respect of Mr Battersby derives from the fact that, although he is not recorded at the time as having been assigned to decontaminating aircraft at Maralinga, his witness statement made for these appeals indicates that he did undertake this function on occasions, and as the surviving records cannot be considered comprehensive this may have happened.

    (iii) Whilst bladder cancer, cataracts, atherosclerosis, immune system failure and myocardial infarction may all be caused by radiation at a significant dose, the other conditions detected in these appeals including pancreatic cancer, CLL and NHL are not radiogenic in nature.

(iv) Such large scale epidemiological studies as there are of groups of people exposed to ionising radiation, (the life time study of the Japanese survivors of the bombs dropped on Hiroshima and Nagasaki in 1945, and a number of assessments made of the British nuclear test veterans and workers in nuclear power and reprocessing plants), suggest that there is no statistically significant increase in cancers at a low dose.

(v) The length of time between the claimed exposures and the medical conditions being diagnosed detracts from the likelihood that military service at CI or Australia was a causative factor.

Litigation History

17. These appeals have a significant litigation history. In 1983 the British Nuclear Test Veterans Association (BNTVA) was formed to campaign for compensation for those service personnel (estimated to be in the order of 22,000) who were engaged in some way with nuclear testing between 1952 and 1958. It was only following the passage of the Crown Proceedings (Armed Forces) Act 1987 that service men and women were able to take civil proceedings against the Ministry of Defence for negligence. A little later it was clarified by the House of Lords that such a claim could be made for service injuries that preceded the lifting of crown immunity.

18. In 2004 notices of claims in negligence were issued against the Ministry of Defence. In 2006 a group litigation order was made and subsequent proceedings brought by a representative sample of some 1011 claimants. A Limitation Act defence was pleaded and the issues of knowledge, reasonable basis of belief and judicial discretion to extend the limitation period were examined at a hearing before Mr Justice Foskett in January and February 2009. He gave judgment for the claimants on 5 June 2009.[3] His extensive decision of some 897 paragraphs is a valuable source of information about the history of nuclear testing and the health effects of ionising radiation.

19. By the time of the High Court hearing the claim of the veterans was said to have scientific support from a study of 50 New Zealand sailors who served on ships in the New Zealand Navy near CI. The study by M Wahab and R Rowland and others was

[3] [2009] EWHC 1225

published in 2008.[4] It used a biological technique called M-Fish to examine blood cells and detected an abnormally high number of translocations in chromosomes in the cohort of veterans by comparison with a control group of service personnel that excluded sailors. The mutations were indicative of a radiological origin. Having obtained these results, human blood from volunteer donors was exposed to an acute single dose of radiation to obtain an estimate by retrospective dosimetry of the dose needed to produce such results.

20. Foskett J did not hear evidence from experts in radiology and related disciplines. However, reports were obtained on behalf of the Secretary of State for the purpose of the civil proceedings from Dr Lindahl (radiobiology), Dr Darroudi (cytogenetics), Dr Lilley (nuclear physics) and Professor Kaldor (epidemiology). These reports commented on current standards in radiological protection, the Wahab and Rowland report, as well as a report by Dr Busby 'Health consequences of exposures of British personnel to radioactivity'[5] that had been provided to the BNTVA .

21. In due course, the Court of Appeal reversed the decision of Foskett J on the Limitation Act issues[6]. The Supreme Court upheld this decision by a majority[7]. Whilst this decision concluded the civil litigation, the attention of the veterans then turned to claims for war pensions under the 2006 SPO. Indeed the possibility of making such a claim was a factor referred to in the evidence lodged on behalf of the defendant in the civil proceedings. The claim for a war pension, in contrast to a personal injury claim, did not require a claimant to establish injury resulting from a breach of a duty of care on the civil balance of probabilities.

22. The claims for a war pension in respect of the conditions listed at [3] and [11] above were refused and the decisions were appealed to this Tribunal.

23. In preparation of these war pension appeals, the appellants, who by now were all represented by Hogan Lovells acting *pro bono*, obtained reports from Dr Brenner on radiation biophysics, Professor Regan on nuclear physics and dosimetry, Professor Parker on epidemiology relating to radiation and Dr Mothersill on developments in radiobiology. It was also originally intended to call Dr Busby as an expert witness;

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[4] 'Elevated chromosome translocation frequencies in New Zealand nuclear test veterans' Cytogenetic and Genome Research 2008
[5] August 2012 FTT D1/10
[6] [2010] EWCA Civ 1317
[7] [2012] UKSC 9

he had given scientific advice to the BNTVA. In addition a number of the statements provided by or on behalf of the veterans for these appeals or the earlier litigation had drawn attention to reports of rainfall on CI during or shortly after the Grapple Y test. This required an extension of the inquiry to include meteorological evidence.

24. The nuclear test series had been devised by scientists at the Atomic Weapons Research Establishment (AWRE) at Aldermaston. The late Ken Johnston had been employed at AWRE at or shortly after the time of the test programme. Although he was not present on CI, he was present at tests in the US in 1962; he subsequently became Chief Scientist at AWRE. He made a number of witness statements for the war pension appeals in which he reviewed the surviving documentation relating to the tests and radiological protection, notably a 1993 report by Clare and others 'Environmental Monitoring at Christmas Island 1957-8' (the Clare Report)[8], a 1999 review by Harrison and others 'Radiological Safety Assurance at UK Atmospheric Weapon Trials' (the Harrison Report)[9] and a detailed 2006 examination of radiological health issues arising from the Australian tests: Carter and others 'Australian Participants in British nuclear tests in Australia' (the Carter Report)[10].

25. Significant parts of the information relating to the test programme remained classified information, and there was an international obligation under the treaties preventing nuclear proliferation not to publish details of the precise chemical ingredients of the weapons. Mr Johnston and Professor Regan were able to examine this material and provide an agreed gist of relevant information where, amongst other things, a maximum level of the plutonium ingredient of the Grapple Y detonation was assumed.

26. In 2012 the late Judge Stubbs, President of the Chamber, issued directions dealing with inspection, handling and further disclosure of documents that were marked classified, confidential, secret, top secret, and/or carried other relevant markings. These directions remain in force and restrict the use to which any document supplied for the purpose of this hearing may be put[11].

[8] SB 17/8
[9] SB 17/9
[10] SB 17/1 contains Vol 1 Dosimetry Chapters 2,3 and 6. The full report was available in the original tribunal materials
[11] The direction is found at SB 1/9 and relates to access to and use of documents classified as restricted, confidential, secret, top secret, atomic, principal, for UK eyes (or similar)

27. The appeals were heard in February 2013 by a panel with Judge Stubbs presiding. The panel heard expert evidence from Professor Regan and Ken Johnston on nuclear physics; Professor Mothersill on radiobiology; Dr Braidwood on the general medical conditions claimed by the veterans and Professor Parker on radiological epidemiology; Mr Nicholson and Mr Stretch on meteorological data at CI at the time of Grapple Y and the mechanics of deposition. A decision was taken by Hogan Lovells (HL) not to call Dr Busby as a witness.

28. The Tribunal dismissed the appeals of the present appellants while allowing some others. The unsuccessful appellants appealed to the Upper Tribunal (Administrative Appeals Chamber).

29. In a decision dated 22 October 2014[12] Mr Justice Charles found there had been a material error of law in the reasoning of the panel. We set out the material parts of his ruling in the following section of this determination.

30. He set aside the decisions and remitted them to this Chamber for rehearing and remaking without reference to the original decision. He invited the appointment of a High Court judge to chair the fresh proceedings. He issued directions for fresh statements of case from the parties in the light of his legal ruling.

31. Charles J dismissed a separate ground of appeal on behalf of Messrs Battersby and Smith (hereafter the BS appeals), namely that the decision not to call Dr Busby as a witness was an error of law. To resolve this ground of appeal he heard evidence from Dr Busby. Having done so, he directed that Dr Busby should not give evidence at the rehearing, although there was nothing in the Tribunal Procedure Rules to prevent him acting as advocate for these appellants. The material parts of this section of the decision are set out in a ruling we gave at the outset of this hearing as to whether we should receive meteorological evidence prepared by Mr Williams, a colleague of Dr Busby's. We append our ruling at Annex A to this determination.

The applicable law

32. Mr Justice Charles reviewed a considerable number of authorities in the field of war pensions and family law, where judges have had to make assessment of risks or possibilities. Having done so he reached his conclusions on the proper approach to Article 41 of SPO. We set out in full his conclusions as they provide the legal

[12] [2014] UKUT 477 (AAC) reported at [2015] AACR 20

directions that we must apply in the re-determination of these appeals. He said as follows:

> 'The approach to be taken to the application of the article 41(5) test
>
> 98. As was accepted in argument before me, the analysis in [72] to [92] above founds the view that it would be wrong in law for a decision-maker applying the Article 41(5) test to take an approach that:
>
> i) determined factors, ingredients or stepping stones and thus the matters that could give rise to possibilities relied on to found a reasonable doubt by reference to the normal civil standard, or any standard other than "reasonable doubt" and so, for example, an approach that rejected evidence that was not fanciful or worthless by preferring other evidence, and
>
> ii) carried forward those findings or conclusions (on a binary approach or otherwise) to the assessment of whether the claimant has established the existence of a reasonable doubt on reliable evidence that the conditions set by article 41(1) are satisfied
>
> 99. Rather:
>
> i) the factors, ingredients or stepping stones to found the possibilities relied on, and so the existence of those possibilities, have to be determined by reference to the standard of raising a reasonable doubt set by the article 41(5) test and thus on evidence that cannot be left out of account because it is fanciful or worthless, and
>
> ii) those conclusions or findings on the existence of possibilities then have to be carried forward to the determination of whether in the light of all the evidence the article 41(5) test has been satisfied.
>
> 100. In my judgment, unless a factor, ingredient or stepping stone can be established or ruled out on the basis that the decision-maker on the evidence has no reasonable doubt about whether (and so is sure that) it is right or wrong (or did or did not happen or exist) the doubt relating to it, and so the possibility that doubt creates, has to be carried forward in the decision-making process**.** So, evidence that is found to be fanciful or worthless can be ruled out. Also, some evidence relating to credibility (eg whether a claimant was in a particular place or did a particular thing) could found a conclusion that it does not raise a possibility that needs to be carried forward. Equally, and subject to the F-tT, wearing its inquisitorial hat, being satisfied that it does not need further investigation, evidence that is accepted or undisputed can be carried forward as an effective certainty and thus as something about which the decision-maker is sure.
>
> 101. The decision-making process on whether a reasonable doubt has been established on reliable evidence will also have to take account:
>
> i) of the relative importance of the relevant factors, ingredients and stepping stones and, on an evidence-based approach,
>
> ii) of the nature and extent of the doubts and possibilities relating to them.
>
> For example, as accepted by counsel for the HL appellants, an overview or cumulative consideration of all the evidence, of the combined effect of doubts, and so the

possibilities they give rise to, may or may not establish a reasonable doubt on reliable evidence that the conditions set by article 41(1) are met. Such an overview of the relevant ingredients and their combined effect is commonly carried out by reference to the normal civil standard (see, for example, Lord Nicholls in *Re H*) and it involves a judgmental or balancing exercise and thus a reasoning process.

102. It is in that exercise or process that the decision-maker has to revisit the impacts of the possibilities (and effective certainties) carried forward and weigh them against each other to determine what possibilities remain and whether they establish a reasonable doubt. This exercise is very dependant on the circumstances of each case and difficult to explain in the abstract. It is however of critical importance and one that decision-makers applying statutory tests and all of us in our daily lives take when considering whether we regard something as a possibility and whether we have a reasonable doubt about something. Tribunals and courts have to explain how they have done it.

103. In carrying out this process, it is highly likely, if not inevitable, that the decision-maker:

i) will have to identify the claimant's case and so the evidence and argument relied on to support it,

ii) do the same with the respondent's case,

iii) identify any additional matters he considers need to be addressed, and then

iv) in light of that identification of the issues evaluate the competing parts of the evidence to determine what possibilities should be carried forward in the decision-making process, and then

v) in light of all the evidence and argument and so, on an overview or an assessment in the round, evaluate the claimant's case to determine whether he has or has not satisfied the article 41(5) test.

It is at stage (v) that the decision-maker will form views that can be expressed by reference to the circumstances of the given case on whether the possibilities (and effective certainties) relied on by the claimant found a reasonable doubt. I repeat that, as was accepted by the HL appellants, at that stage it may be that the decision will be that the combined effects of the possibilities carried forward do not found a reasonable doubt because for example the combination of those possibilities is too far-fetched.

104. …

105. …

106. Counsel for the Secretary of State reminded me, by reference to authority, that cases may have to be decided by an application of the burden of proof. Naturally, I accept that decision-makers on the approach I have described have to be satisfied that there are possibilities that establish a reasonable doubt and an assertion that "anything is possible" is not enough. But I suggest that as in cases to which the normal civil standard applies it will only be rarely that cases under the article 41(5) test will be decided on the burden of proof. Further, and in any event, if a decision-maker is to so found his decision he would have to say so and explain why he was driven to that last resort.

107. The Secretary of State placed emphasis on a submission that *Dickinson* establishes a two-stage test. First, it is for the claimant to produce reliable evidence to establish his claim and second, if such reliable evidence exists, it must be sufficient to raise reasonable doubt because, at the second stage, it must be compared with such evidence as is called on behalf of the Secretary of State. The purpose of that argument was to found a submission that the approach of the F-tT, of making findings on the basis that there was insufficient reliable evidence, was correct. In this context the Secretary of State also submitted that the concept of something needing to be "sufficiently" supported so as to raise a reasonable doubt was expressly recognised in *Edwards* (see the quote in [65] above).

108. The Secretary of State recognised that there was an interrelationship between the two-stages but relied on their existence to make good his argument on the F-tT's use of the word "sufficiently".

109. I do not accept that it is appropriate to approach article 41(5) on the basis that it imposes a two-stage test. This is too analytical and unjustifiably seeks to differentiate the position of a claimant under article 41 from that of any other claimant in civil litigation who has to discharge a burden of proof. In all such cases, there can be said to be a two-stage test because the respondent can produce no evidence and argue that the claimant has not proved his case. This simply reflects the existence of the burden of proof and, to my mind, when a respondent does not choose to give any evidence it is artificial and unwarranted to classify the consideration of all the evidence by a court or tribunal as the second stage of a two-stage test or approach.

110. Further, in my judgment the Secretary of State seeks to read far too much into the passage he relies on in *Edwards* to show an express recognition of the concept of something needing to be sufficiently supported. First, that passage is directed to the degree of evidential support needed for a hypothesis to raise a reasonable doubt and not to a general proposition. Secondly, and more importantly, I have no quarrel with the point that there has to be sufficient evidence to found an evidence-based conclusion to the relevant standard but the concept of "sufficiency" is <u>not</u> a free standing concept, rather it has to be assessed in the context of the standard to which it relates. Thirdly, and also importantly, the way in which the concept is used by the F-tT (ie "there is insufficient evidence to raise a reasonable doubt") indicates that in their view although there is some reliable evidence it is not enough to raise a reasonable doubt and the F-tT fail to identify:

i) what that reliable evidence is, or

ii) why, although it is reliable, it was not enough or sufficient to raise a possibility or possibilities that found a reasonable doubt.

This failure demonstrates that, of itself, the concept of there being sufficient or insufficient evidence to satisfy a standard of proof does not identify or indicate the meaning of that standard, how it is to be applied or how it has been applied. At best, it is a neutral part of a bare assertion of a conclusion by reference to that standard.

111. So I agree with the appellants that if the F-tT made findings or reached conclusions on the ingredients to be carried forward and taken into account in applying the article 41(5) test (for example on internal or external exposure) by reference to the normal civil standard, or to any standard other than that of reasonable doubt on reliable evidence (ie evidence that is not fanciful or worthless), they erred in law. Indeed, as mentioned above, it was accepted before me that this would be the case.

112. In my view, the passage cited and relied on by the F-tT from *Edwards*, and so references to "was a mere hypothesis on a limited study" and "there are … three stages: no reasonable doubt, reasonable doubt and consensus" do not indicate where in a particular case the divisions lie. In the phrase "a mere hypothesis on a limited study" both the words "mere" and "limited" are words of degree and their impact is important not least because when something is not known (eg exposure to radiation and/or radiogenicity) the possibilities (including the consensus if there is one) are likely to be based on theory or hypothesis founded on facts, research and reasoning, all of which will have a part to play in determining whether that theory or hypothesis is fanciful or worthless and so (or for other reasons) should be left out of account, or whether it should be carried forward to the final judgmental exercise.

113. So I agree with the HL appellants that if the F-tT proceeded simply on the basis that an hypothesis or a mere hypothesis should be left out of account they erred in law.

114. ...

115. By the end of the oral argument, apart from the points:

i) on there being a two-stage test, and

ii) the utility of the decision in *Edwards* to developing hypotheses,

there was effective common ground between the parties on the meaning and correct application of the article 41(5) test that accorded with the conclusions I have set out above, albeit that none of the parties expressed them in precisely the same way as me or each other.

116. But, so far as I am aware in common with earlier cases in this jurisdiction, there was no consideration before the F-tT of the principled evidence-based approach to be taken in the application of the article 41(5) test and thus in particular of (a) how the stepping stones or ingredients relied on and advanced by the appellants to establish the relevant possibilities were to be approached, and (b) the carrying forward of the conclusions on the existence of such possibilities into the decision-making process on an evaluation of all the evidence in the round.'

33. These directions are binding on us and we apply them in our consideration of the evidence before us. Without in any way altering the substance of them, for short hand, we have used the following summary self-direction when examining and evaluating all the evidence before us:

(i) Is there plausible evidence, scientific or otherwise, that might found a possibility or certainty on which the overall evaluation is to be based?

(ii) Taking into account all plausible evidence, has the appellant satisfied us there is a reasonable possibility of a causal link between the military service and the medical condition claimed in his case? If so a reasonable doubt will exist.

Later in this determination, we address the issue of how and when an hypothesis advanced by a scientist may be said to be plausible and return to the theme in the opening paragraphs of Part Eight.

The Further Evidence

34. Judge McKenna as then Chamber President and Judge Wikeley as Acting President issued further directions on 13 May and 8 and 22 June 2015. By this time Mr Justice Blake had been appointed to chair the panel of the Tribunal that was to re-determine this appeal. On 28 July 2015, there was a contested directions hearing where the Secretary of State sought leave to adduce fresh expert evidence to re-examine dosimetry, health effects of any radiation exposure that dosimetry assessed may have occurred and the evidence of a bio-statistician as to the degree of likelihood of causation. There had been an earlier offer of joint instruction but this was not agreed. The Tribunal concluded that there was good sense in having the assistance of expert evidence looking at all plausible pathways to exposure in the light of the UT's ruling, and so acceded to the proposals for a revised timetable accommodating such a course.

35. The HL group of appellants, did not propose to adduce any live evidence of their own but relied on the possibilities revealed in the reports and the transcripts of the oral evidence of the experts that had been previously instructed

36. The BS appellants, now represented by Mr Busby, proposed to call their own witnesses.

37. In the end scientific reports were obtained on behalf of the defendant from Mr Hallard, an expert in dosimetry and radiological protection, Professor Thomas on radiobiology and Dr Haylock, a bio-statistician and head of Analytical Epidemiology at Public Health England's Centre for Radiation, Chemical and Environmental Hazards (CRCE). For the BS appellants, reports were obtained from Professor Sawada, principally concerned with uncertainties in the risk model based on the Japanese survivors of the 1945 bombs; Professor Schmitz-Feuerhake on aspects of radiobiology and reports of health outcomes from the Chernobyl disaster and other groups affected by radiation from nuclear power stations; Professor Hooper on the health effects of internal exposure to radiation; and Professor Howard on the congenital effects of the offspring of the BNTVA. In addition permission was

subsequently given to lodge a report from Dr Ash on fallout factors and related radiation risks at Maralinga and CI.

38. Further directions were made that the reports and response to questions should stand as evidence in chief, subject to correction and updating. Other directions were made on third party disclosure and other topics on 17 and 21 December 2015, and 14 and 15 January 2016, 24 February, 4 and 21 March and 11 and 14 April 2016.

39. We heard the appeals between 13 and 30 June when we heard from all the expert witnesses listed in the previous paragraph and in addition from Richard Bramhall, secretary of the Low Level Radiation Campaign, and a member with Dr Busby of the Committee Examining Radiation Risks from Internal Emitters (CERRIE) set up in 2001 by the then Minister of State for the Environment.

40. At the outset of the appeals we were provided with 22 supplementary bundles (SB), containing the material generated in the litigation history noted above, learned papers cited by experts on both sides, and such extracts from the previous library of materials before the First-tier Tribunal (FTT) as was considered relevant. In addition a complete set of the 50 or so volumes of material before the previous appeal was made available to us[13].

41. We have been assisted by the respective parties' statements of case, skeleton arguments, schedules, chronologies and cross references and written as well as oral submissions. This material was extensive and cross-referred to lengthy written submissions made by the HL appellants at the previous proceedings. We will not be addressing every point made to us orally and in writing although we have read this material; rather in Parts Four to Six we address the principal submissions advanced to us by the appellants in the light of the current state of the evidence.

42. This determination will proceed as follows:

    (i) In Part Two, we set out the scientific background to ionising radiation and protection of human health and in particular the role of International Commission for Radiological Protection (ICRP) in disseminating advice and setting protection standards.

[13] Referenced as FTT and thereafter by volume and Tab

(ii) In Part Three, we describe what is established by the available evidence as to the nature of the tests at Maralinga and Christmas Island (particularly Grapple Y) and the contemporary methods of protecting personnel from radiation and measuring radiation.

(iii) In Part Four, we consider the challenge to the ICRP model of radiological protection advanced by the BS appellants and their witnesses.

(iv) In Part Five, we turn to the evidence of dose retrospectively assessed by Mr Hallard for this appeal and the criticisms made of it.

(v) In Part Six, we examine the case of the HL appellants and the reliance they place on the evidence (in particular) of Professor Regan, Professor Parker, Professor Mothersill in the previous proceedings and the Rowland and Wahab study.

(vi) In Part Seven, we examine the nature of the medical conditions of the veterans in this appeal and the extent to which they are recognised as being caused by radiation. In doing so we note the conclusions of Professor Thomas and Dr Haylock based on Mr Hallard's assessment of dose.

(vii) In Part Eight, having identified what evidence we consider is reliable, we examine its overall plausibility to determine whether it gives rise to a reasonable doubt in any of the individual appeals according to the law expounded by Mr Justice Charles.

PART TWO: SCIENTIFIC BACKGROUND

Nuclear Physics

43. Some understanding of the mechanisms by which radiation is caused is necessary in order to understand the scientific evidence presented to us and evaluate the possibility of a health risk caused by an exposure pathway.

44. There are helpful discussions in many of the witness statements and expert reports that have been placed before us. A good starting point is the first report of Professor

Regan prepared for the 2013 appeal hearing[14]. It gives the Tribunal a basic grounding in nuclear physics.

Atoms

45. At the heart of the technology behind the devices tested is the atom. We summarise the following points:

    (i) Normal matter is composed of atoms and molecules. Atoms are constructed from a very small and highly dense atomic nucleus that contains nearly all of its mass. They also have a cloud of negatively charged atomic electrons.

    (ii) The nucleus of an atom consists of two sub atomic particles, positively charged protons and neutrons which carry no electrical charge. Neutrons outside the atomic nucleus (free neutrons) are unstable and radioactively decay to form a proton and an electron.

    (iii) Elements arise in different types known as isotopes. These are atoms which have the same number of protons in their nuclei but have different masses because they have different numbers of neutrons. The nuclei of different elements are described by their atomic mass number.

    (iv) Of the 7000 potential combinations of proton and neutron numbers that are theoretically possible, only 300 are stable and the rest undergo radioactive decay.

Radioactive decay

46. Radioactive decay is a process where nuclei can exchange some of their mass-energy by altering the internal structure of a nucleus. The energy released in this process is transferred to the emission of specific decay products which include alpha (α) and beta (β) particles and gamma (γ) rays.

47. Radioactive decays are described by decay half-lives. This is the time taken for half the nuclei present to undergo radioactive decay. The same period is then taken for half of the remaining nuclei to decay. Decay is thus not a straight line curve but is inversely proportional. Different isotopes have different half-lives that may be as short as a matter of seconds or as long as many thousands of years.

[14] SB 11/2 and 3

48. The activity of a sample of radioactivity is the number of radioactive decays per second from that sample. Activity is now measured in Becquerels (Bq) where 1 Bq is equal to one radioactive decay per second. Historically (and during the 1950's) it was measured as a Curie (Ci). One Ci is equal to 3.7 x 10^{10} disintegrations per second or 37,000 million Bq.

49. The energy released in α decay can be calculated using the famous Einstein relation $E=Mc^2$. The energy of an α particle is completely defined by the masses of the initial and final nuclei involved and is thus characteristic to the decay. This means that a direct measurement of the specific α particle energy can be used to determine which nucleus was present to give rise to the α decay. For example, uranium-234 decays into thorium-230 (main characteristic α particle energy of $E\alpha$=4.774 MeV) and plutonium-239 decays into uranium-235 (main characteristic α particle energy $E\alpha$=5.157 MeV). Plutonium-239 is artificially created by neutron activation of uranium-238 and is one of the two major fissile materials used in nuclear weapons.

Nuclear fission and fusion

50. Nuclear fission is a powerful way for a very heavy nucleus to change into more stable forms. In fission a heavy nucleus splits into two approximately equal parts. Energy is also gained when light nuclei are combined together to form a heavier nucleus. The process is called nuclear fusion.

51. As an energy source, fusion has several advantages over fission because the light nuclei are plentiful and easy to obtain and the end products are usually light, stable nuclei rather than heavy, radioactive ones. The main drawback is that nuclei can only be made to fuse together when they have enough energy to overcome the electrical repulsion when they collide with each other. This can only happen when the temperature of the light atoms is raised to a sufficiently high level – in excess of 10 million degrees centigrade. The process is called thermonuclear fusion because of the thermal energy needed to create the conditions for it to occur and these temperatures are reached during a nuclear fission explosion.

52. Nuclear weapons may be of the fission type (atom bomb) or thermonuclear (hydrogen bomb). Of the two uranium isotopes, uranium-235 is much more likely to undergo fission than uranium-238 and it is for this reason that a fission device needs enriched uranium to ensure that a chain reaction can be established. There must be

enough fissile material present to ensure that after the material has been compressed enough neutrons remain to interact.

53. The most commonly used fission bomb is an implosion type which consists of a set of concentric spherical shells. At the centre is a sub-critical shell of fissile material made up of uranium-235, plutonium-239 or a combination. This is surrounded by a tamper shell usually constructed of a very dense material which can be depleted or natural uranium. The outer shell consists of a chemical high explosive. The blast following detonation of the chemical explosive drives the tamper material inwards, compressing the fissile material into a supercritical mass and triggering the nuclear explosion. In addition to compressing the shell of fissile material the tamper also acts as a neutron reflector returning escaping neutrons back into the core and thus enhancing the explosive yield. A thermonuclear bomb includes a fission explosion which is responsible for heating and compressing the thermonuclear fuel to the point at which the reactions begin to proceed. They continue to sustain the explosion until eventually the expansion of the fuel disperses the material and the reactions stop.

54. Fission products originate from fission of the nuclear materials in the core of the weapon. When an atom of uranium (U) or plutonium (PU) in the core absorbs a neutron, the nuclide resulting is highly unstable and rapidly splits (fissions) into two smaller nuclides roughly half the mass of the original fuel.

55. A thermo-nuclear device (as was the case with Grapple Y) creates energy by fusion. However there is still a great deal of fission in such a device as there is a fission-fusion-fission cycle. There is the primary explosion that is a fission process and a secondary fusion stage resulting from the use as a heavy metal jacket of uranium-238 as a tamper device to sustain the chain reaction for as long as possible and undergo further fission from the neutrons produced in the core.

Ionising radiation

56. A helpful analysis of the nature of ionising radiation, and the means whereby humans may be exposed to it, is to be found in the Carter Report Vol 1 Dosimetry[15].

57. Ionising radiation is radiation that has enough energy to ionise matter through which it passes. Ionising is the process of stripping off one or more electrons within an

[15] Published May 2006

atom that carry a negative charge, leaving the nucleus that is positively charged (an ion). The health effects that arise from exposure to ionising radiation are understood to derive from ionisation taking place in living cells because ionising radiation can alter and damage the structure of cellular DNA.

58. Most of the tissue genetic damage caused by X rays is due to the conversion of water molecules to highly reactive hydroxyl radicals in the vicinity of DNA. In contrast, charged particles ionise numerous target molecules directly. Densely ionising radiation such as that from alpha rays is also referred to as high-LET (linear energy transfer) radiation whereas sparsely ionising radiation from gamma rays and X rays is called low-LET radiation.

59. DNA damage similar to that caused by ionising radiation is also generated by reactive oxygen species occurring as side products of normal metabolism. This is likely to be of significance because of the existence of several different DNA repair mechanisms to correct such metabolic damage.

60. Ionising radiation is of two types: through subatomic particles and electromagnetic radiation. The subatomic particles of relevance to these appeals are alpha particles, beta particles and neutrons. Electromagnetic radiation includes X rays and gamma rays.

61. Alpha particles are relatively heavy and slow moving and have short ranges – around three centimetres of air. They cannot penetrate a sheet of paper or the outer dead layers of human skin. Alpha particles and their decay products tend to have very long radioactive half-lives. Their relevance to these appeals is through internal exposure.

62. Beta particles are high energy electrons that are moderately penetrating (up to one metre of air, a few millimetres of aluminium and a short distance into animal tissue).

63. High energy neutrons can penetrate several centimetres of concrete and, like gamma and X rays, they can pass right through the body. Neutrons, by contrast with alpha and beta particles, can make objects that they irradiate radioactive. gamma rays are physically identical to X rays but are more energetic and penetrating.

64. There may be immediate or delayed exposure to radiation. Sources of delayed radiation exposure are:

(i) activation products;

(ii) fission products and

(iii) unconsumed nuclear fuel including in some devices uranium-238 as the tamper.

65. *Activation products* result from neutrons produced in the explosion being absorbed by stable non-radioactive atoms in the ground, the bomb casing, any supporting tower and other test components which then become radioactive.

66. *Fission products* are the radionuclides produced when atoms of the nuclear explosive (plutonium or uranium) split into two in the fission reaction. There are hundreds of different radionuclides produced in the fission process with most of them being beta and gamma emitters. Most will rise with the mushroom cloud from where they are dispersed as fallout but some may be distributed around the detonation point. As with activation products there is a wide range of half-lives ranging from less than a few seconds to millions of years.

67. *Unconsumed nuclear fuel* occurs because nuclear explosions are never completely efficient and sometimes a significant percentage of fuel (80% or more) may remain. These fuels have very long half-lives.

Radioactive exposure

68. Applying these general principles, Mr Hallard's report explains that exposure to radiation from a nuclear denotation arises in a number of ways. First, there is immediate exposure of people within a relevant proximity to the device to the radioactivity released at the moment of detonation, notably gamma rays and beta particles. The explosion creates very high levels of neutrons and gamma rays as a direct result of the nuclear fission or fusion reactions which create the explosion. These sources reduce rapidly with distance from the initial detonation known as the inverse square law, thus the dose rate at one metre from a source would reduce to one quarter of that level at two metres. There is insignificant effect beyond six to eight kilometres.

69. The explosion can create a source of exposure to radiation in other ways. For example neutrons may interact with non-radioactive atoms to create radioactivity, thus the non-radioactive Na23 in the sodium chloride (salt) in seawater may be activated to become the radioactive Na24. Radioactive dust (e.g. of contaminated

soil), aerosols (fine droplets of radioactive solution in the air) or gas can be suspended in the air and a person passing through such air would be exposed to such material.

70. Another important source of exposure is radioactive fallout. Fallout is very fine particulate radioactive material generated in the explosion which is then brought to earth by gravity rainfall or wind. The radioactive material comprises of two main products, fission products and the remnants (i.e. the unreacted bomb residues) of the uranium and plutonium nuclear fuel.

71. Hence the fallout from an explosion such as Grapple Y would comprise a mixture of beta emitting fission products such as uranium-239 and alpha emitting plutonium-239. Uranium-238 is also an alpha emitter but the activity is much lower and thus not significant by comparison with plutonium-239. We shall examine the nature of uranium and the argument made by the BS appellants in respect of it at Part Four [261] and following.

72. Exposure to radiation may be external or internal. External exposure comes from radiation sources outside the body such as exposure to X or gamma rays or beta particles from standing on ground contaminated by radioactive material. It can only arise from radiation that has sufficient range and energy to penetrate any gap or any shielding between the radiation source and the person and then pass through clothing and the outer layers of skin. External exposure ceases as soon as the source is no longer in contact with the person, although if clothes or equipment are contaminated they may continue to be a source of exposure.

73. External radiation is relatively easy to assess. One instrument for direct measurement of external radiation that was in use at the time is a film badge attached to the individual that can be processed after the event giving rise to the radioactive exposure. Instruments such as a Geiger-Muller counter can measure the radiation level (dose rate) in an area. A calculation can then be made by multiplying the dose rate by the number of hours of exposure.

74. Internal exposure may arise through inhalation via the lungs, ingestion by swallowing into the stomach, through absorption through the skin or penetration through wounds. All forms of radiation can produce internal exposures. Such exposure will continue until the radioactive material in the body has either decayed

away or has been excreted from the body. Thus internal exposure could continue for many years after an initial intake.

75. It is considerably more difficult to assess the amount of internal radiation. An estimation of the intake of internal radiation can be made from measurement of the external dose, for example, the radioactive content of the air breathed or items ingested, the breathing rate (in the case of inhalation) and the time spent in the radioactive area. With this information, if sufficient is known about the materials inhaled or ingested, particle size, chemical form, retention in bodily organs, radioactive half-life, and the excretion rate of the radio nuclides, it is possible to calculate effective dose.

Measurement of dose

76. The modern system of dose measurement has been devised by the International Commission on Radiological Protection (ICRP). It proceeds by way of absorbed dose, equivalent dose and effective dose. We here use the definitions of these terms provided by the UK Health Protection Agency (HPA).[16]

77. *Absorbed dose:* is the amount of energy deposited and is equal to 1 Joule of energy deposited per kg of matter (usually air or tissue). It takes no account of the biological effects that the radiation might provide in living matter. It is measured in Grays (Gy).

78. Effective, equivalent and committed doses are measured in Sieverts (Sv). This is used to measure the health effect of radiation on the human body in smaller doses. It is the basic tool of dosimetry. At lower levels of exposure outcome is expressed in milli-sieverts (mSv) that are one thousandth of a Sv. An exposure of 500 mSv or more is considered to be a high exposure with known health impact on some parts of the body. A low level exposure is considered to be 100 mSv or less. Exposures below 50 mSv can generally be regarded as very low for health purposes. Extremely low levels of exposure are measured in micro-sieverts (μSv) that are one millionth of a Sv. An even smaller measurement is the nano-sievert.

79. *Equivalent dose*: (sometimes known as organ/tissue dose) is the unit devised by the ICRP to apply the risk factors from the A-bomb (external gamma radiation) to

[16] HPA RCE 12 Application of the 2007 Recommendations of the ICRP to the UK (SB5/45). We were provided with the ICRP Paper 103 the 2007 Recommendations on Radiological Protection (SB/3/ Tab 2) and the update Paper 119 Compendium of Dose Coefficients (SB/3/Tab 1)

radiation of all types both external and internal. The equivalent dose is calculated by multiplying the absorbed dose by a radiation weighting factor (W_R) to take account of the relative effectiveness of different radiation types per unit absorbed dose in causing stochastic effects (see [91] below for discussion of this term) at low doses. For example, alpha particle and neutron radiation is much more effective at producing biological damage than gamma or beta radiation.

80. The weighting factors used by the ICRP to calculate effective dose are as follows:

    (i) The radiation weighting factors for alpha = 20; beta and gamma =1; and therefore for alpha radiation, an absorbed dose of 1 mGy = equivalent dose of 20 mSv.

    (ii) For beta or gamma radiation, an absorbed dose of 1 mGy = equivalent dose of 1 mSv.

81. *Effective dose*: (sometimes called the individual/whole body dose; measured in Sv) is the sum of the equivalent doses to each organ or tissue using defined tissue weighting factors to provide a measure of the stochastic (random) risk of cancer or hereditary effects[17].

    (i) Effective dose can be calculated from equivalent dose by the use of weighting factors for the different types of organ or tissue.

    (ii) The current levels in the UK are based on ICRP 60 rather than 103, but this may change with the revision of the UK legislation currently expected in 2018[18].

    (iii) The tissue weighting factor allows for different radio sensitivities eg skin is 0.01. The sum of the weighting factors over all organs is 1.

    (iv) For whole body gamma exposure both weighting factors are 1 so the effective dose (in Sv) = absorbed dose (in Gy).

82. The concept of effective dose is valuable for use in radiological protection and allows doses from partial and whole body exposure as well as doses from external

[17] HPA ibid p13
[18] Hallard p275

radiation and from intakes of different radionuclides to be summed and compared with dose limits[19].

83. *Committed dose*: Equivalent and effective doses from intakes of radionuclides are commonly integrated over a 50 year period for adults and to age 70 years for children and the resulting values are referred to as committed effective dose.

84. *Collective effective dose*: is the sum of all the effective doses of a population or group of people exposed to radiation and is expressed as man-Sieverts.

85. As we have noted the unit of radioactivity is the Becquerel (Bq). This is 1 disintegration of a radioactive nuclide per second. There is no simple relationship between the activity (Bq) of a radioactive source and the resulting dose (Sv).

86. *Historical units*: As we have also noted above at [48], Curie (Ci) was the unit of activity based on the activity of 1g of radium. 1 Ci is equivalent to 3.7 x 10^{10} Bq. Roentgen (R) is a measure of the radiation emitted from a source and is defined as the ionisation created in air from gamma or X ray radiation so is a unit of exposure rather than absorption. It has no modern equivalent. Prior to the use of Grays, dose was defined by units of rads ('Rad' = 'radiation absorbed dose').1 Rad = 0.01 Gy or 100 rad = 1 Gy[20]. Rem (Roentgen equivalent man): this was the unit of biological effectiveness of the radiation and allows for the differing radio sensitivities of different body organs. 1 Rem approximately equals 10 mSv.[21]

87. Acute radiation takes the form of a single exposure over a short period of time. Protracted radiation is exposure over a lengthy period of time. Protracted or delayed exposure to radiation is most likely to arise from internal exposure.

Deterministic and stochastic effects

88. We acknowledge the assistance of the Carter Report [22] in this part of the summary of the science. Acute external exposure radiation of 1 Sv and above results in a deterministic effect. This means that sufficient cells are killed to cause radiation sickness in the form of skin burn, vomiting, diarrhoea and hair loss (epilation) that will be manifest within a few hours of exposure.

[19] Report of the independent Advisory Group on Ionising Radiation (August 2011) SB 5/42. Risk of solid Cancers following Radiation Exposure.
[20] Prof Regan report SB11/2
[21] Hallard p276
[22] Vol 1 dosimetry at 6.4.3 and following

89. For the average individual, no immediate deterministic effects are observed at doses of less than 1 Sv. An external exposure of 3 Sv or more has a 50% likelihood of causing death within 60 days as enough cells are killed to cause breakdown in tissue structure or function. An acute dose of 15 Sv will result in unconsciousness within a few minutes and death within a few days.

90. At the other end of the scale, the authors of the Carter Report state that in 2006 there was no biological evidence that doses of less than 50 mSv can cause deterministic effects. Temporary sterility for a month or so can result from a dose of 150 mSv. Permanent sterility or cataracts may arise from higher doses in the range of 500 to 5000 mSv (i.e. 0.5 to 5 Sv). An issue in these appeals is whether this account still reflects contemporary understanding, particularly with respect to cataracts.

91. Whilst deterministic effects result from cells being killed, ionising radiation can also damage cells by causing changes in the DNA of the cell nucleus. If the damage is not repaired and the cell remains viable and continues to reproduce this event may initiate the development of a cancer. Damage to the cells connected with reproduction may result in genetic disease in offspring. The effect of radiation that initiates a cancer or genetic damage is called stochastic, meaning that the effect is governed by probability. An increase in the magnitude of the dose will increase the probability but not the severity of the effect.

92. The Carter Report explains:

> 'stochastic effects do not generally became apparent for many years after exposure, and there is no way of distinguishing a particular cancer or genetic effect that might have been caused by radiation from one arising from other origins. There are some forms of cancer that do not seem to be caused by radiation exposure….stochastic effects, in particular cancer, have only been clearly been demonstrated in humans following moderate or high exposures of the order of 0.1 Sv and above, and there is no direct evidence that these effects can arise at the significantly lower doses characteristic of present day occupational exposures.'

93. We will examine in greater deal in Part Four the evidence relating to harm from internal exposure to radionuclides and in particular, an internal dose of uranium. A large dose of internal radiation, for example by ingestion of an alpha emitting particle may cause death. As we explain later, however, the consensus of scientific opinion is that in the case of large doses of such a particle this is usually from its toxicity rather than its radioactive effect, as, for example, uranium-238 and plutonium-239 isotopes have slower decay rates and are therefore considered less

active. A smaller ingestion dose may only kill a few cells; a large particle that is not easily dissolved or absorbed into the blood stream is likely to be excreted, rather than continuing to damage further cells and the body is thus able to repair itself. However, at the lowest end of the scale, a smaller radioactive exposure, for example, by very fine particles that may be more easily absorbed into body tissues may continue to emit radiation. We will examine in Parts Four and Six whether such exposures may have biological impact.

Background radiation

94. Everyone is exposed to naturally occurring radiation. This may be from sunlight or from gases such as radon that occur in local geological formations. The authors of the US BEIR Report VII[23] state that:

> 'average exposures to natural radiation sources (both high and low linear energy transfers) would generally be expected to be in the range of 1-10mSv with 2.4mSv being the present estimate of the central value. Of this amount about one half (1.2mSv per year) comes from radon and its decay products.'

95. In addition there is atmospheric radiation that has accrued as a result of past releases of nuclear energy whether by way of weapons testing or emissions from power stations or other man made sources of radiation (man-made radiation).

96. The BEIR Report observes:

> 'A 1987 study of ionizing radiation exposure of the population of the United States estimated that natural background radiation comprised 82% of the annual US population exposure, while man-made sources contributed 18%....Medical X rays and nuclear medicine account for about 79% of the man-made exposure in the US. Other sources may be elements in consumer products such as tobacco, domestic water supply, building materials and, to a lesser extent, smoke detectors, televisions and computer screens account for another 16%. Occupational exposures, fallout and the nuclear fuel cycle comprise less than 5% of the man-made component and less than 1% of the combined background and man-made component. All small amounts of exposure from background and man-made radiation comes from travelling by jet aircraft, (add 0.001 mSv for each 1,000 mile travelled) living near a coal fired power plant emissions (add 0.0003 mSv), being near X ray luggage scanners or living within 50 miles of a nuclear power plan (add 0.000009 mSv).'

97. Professor Thomas in her evidence to us makes a similar point. She estimates that 85% of radiation exposure is from natural sources and 15% from man-made ones. The average global exposure to these combined sources of background radiation is

[23] Beir VII Phase 2 Health Risks From Exposure to Low Levels of Ionizing Radiation National Academies Press SB 17/2 p3

2.4 mSv a year giving an exposure of 160 mSv to someone who lived to be 80[24]. The range can be from 1 mSv to 10 mSv a year and those living where there is an unusually high amount of radon may have exposure of up to 50 mSv per year.

98. These appeals are concerned with any additional radiation to which the veterans may have been exposed over background radiation by reason of their military service.

99. In the next section of this determination we review what was known about radiological risk at the time of the relevant tests 1956-8, what was done to prevent or reduce exposure and measure the amount of exposure to which veterans may have been exposed.

Protection of health from radiation

Origins

100. The potential hazards of radiation were known about by the start of the twentieth century. The first International Congress of Radiology (ICR) met in 1925.

101. 1928 saw the development of the International X-ray and Radium Protection Committee (IXRPC) which was reconstituted in 1950 after the Second World War. By this time there was interest in the military use of the energy to be released from an atomic or nuclear explosion.

102. In 1945 such military use was deployed against the cities of Hiroshima and Nagasaki. The consequent loss of life arose first from the shock waves released that destroyed buildings and structures. Second, people were also killed by the deterministic impact of external radiation. Third, people present within the vicinity of the explosions were exposed to radioactive fallout, some of it in the form of rain cloud deposition, from which longer term effects have arisen.

103. The impact of radiation on the surviving population has been a matter of study and concern ever since. The Radiation Effects Research Commission (RERC) and its predecessor the Atomic Bomb Casualty Commission (ABCC) has conducted a study called the Life Span Study (LSS) since 1950 of a fixed population of 120,000 subjects who were a combination of survivors and residents who were not in the cities at the time of the bombing. The purpose is to determine the late health effects of ionising radiation from the bombs. This is the largest and longest study of such

[24] Report paragraph 1.8 SB 2/2.18

health effects, and the LSS has been one of the primary sources of information on the health effects of atomic and nuclear bombs. It has been an influential source of information for all scientists studying the problem and making estimations of risk.

The LNT model

104. The ICR reconvened in 1950 and changed its name to the International Commission on Radiological Protection (ICRP) that it retains to the present time. The 1950 recommendations were based on recommendations of a maximum exposure with a minimum threshold below which no harm was believed to have occurred. By 1953 ICRP recognised the principle that no radiation level above natural background level can be regarded as absolutely safe and the task for science was to identify a practical level that involves negligible risk in the light of present knowledge.

105. It did so by developing the dosimetry tools previously discussed: absorbed dose, equivalent dose, effective dose and controlled dose. By these means it considered it was able to assess the health effects of both internal and external exposure to radiation. It recognised that acute exposure to radiation and the deterministic effects resulting from large doses were easier to evaluate in terms of cause and effect. It nevertheless concluded that although there was no safe threshold of radiation it could assess the stochastic effects of low level radiation (i.e. less than 100 mSv) from applying the linear module used in higher doses. This is to say that the ICRP concluded that even at low levels the higher the dose the greater the risk. This is known as the linear no threshold model (LNT). Over the years the model has been modified with respect to dose estimates and human tissue weighting factors as information that is assessed to be reliable becomes available. There has been revised guidance issued in the 1991 and 2007 review of risks, and responses are made to other issues as and when they arise. In adopting and maintaining this model, ICRP does not accept the theory that has been advanced by some that low level internal exposures have greater impact on health than higher level exposure, particularly external exposure. We shall review the evidence relating to this debate later in Part Four of this determination.

106. By 1955 other international bodies were advising on radiological protection: the International Atomic Energy Authority (IAEA) and the World Health Organisation (WHO). In the same year the UN General Assembly set up United Nations Scientific

Committee on the Effects of Atomic Radiation (UNSCEAR). We have found the UNSCEAR 2006 Report to be a helpful summary of current knowledge.

107. National agencies consider and apply the advice of these international bodies; in the USA, the BEIR Committee, whose report has already been quoted, is the national body providing scientific guidance to those making assessments of radiological risk.

108. In the United Kingdom, an Advisory Committee on Radiation Protection was established in 1949. In 1956 the Medical Research Council produced a comprehensive report 'The Hazards to Man of Nuclear and Allied Radiations'. The Radiological Protection Act 1970 (RPA) established the National Radiation Protection Board (NRPB) whose reports we will consider later. In 2004 the Health Protection Agency Act repealed the RPA and established the Health Protection Agency (HPA). The NRPB became the Radiological Protection Division of the HPA. In 2012 the HPA was itself abolished and its functions assumed by Public Health England. The radiological protection division is the Centre for Radiation Chemical and Environmental Hazards (CRCE).

109. All these bodies, and the relevant institutions of the European Union, apply the LNT model. They also consider and review epidemiological studies of those who have been exposed to radiation when outcomes are assessed by comparison with a control group. Although epidemiology does not determine medical causation in any particular case, conclusions on studies that are reliably conducted, can contribute to the assessment of risk of the health effects of low level radiation. We will review sound epidemiological principles and the studies and scientific opinions based on them, in Part Six of this determination.

110. We will also examine further work in the United Kingdom performed by the HPA and other bodies specially convened in response to concerns about the effects of low level environmental radiation and what is currently known as to the biological effects of radiation. In doing so we take into account not only the reports already cited but also a number of other papers to which we have been referred during the hearing, for example those by Brenner, Muirhead, Puncher and colleagues[25].

[25] Advisory Group on Atomic Radiation (AGIR) (SB5/4 and 5; Puncher 'An assessment of the reliability of dose co-efficients' Journal of radiological protection July 2014 (SB/3/11), Muirhead et al Mortality and cancer incidence following occupational exposure to radiation , British Journal of Cancer (2009) (SB4 /24)

PART THREE: THE UK TESTS AT MARALINGA AND CHRISTMAS ISLAND

The Australia tests

111. Between October 1952 and October 1958 there was a series of nuclear weapons tests in Australia. Prior to the Buffalo series at Maralinga, in the dusty and relatively treeless desert region of South Australia, there had been: Hurricane, an ocean surface burst with a 25kt yield off the Monte Bello Islands in Western Australia on 3 October 1952; Totem 1 (10 kilo-tonnes (kt)) and 2 (8kt) that were both tower mounted tests at Emu Field in October 1953 and Mosaic 1 (15kt) and 2 (60kt) in the Monte Bello Islands in May and June 1956. After Buffalo there was the Antler series of 3 tests at Maralinga in September and October 1957. These were at the sites Tadje, Biak and Taranaki.

112. There was also a minor test series between 1953 and 1963 mainly at Maralinga with one at Emu Field some 190km north of Maralinga. These were used to investigate the effects of fire and non-nuclear explosions on atomic weapons. They were not fission explosions and therefore there was no fission fragmentation or fallout. They did present a radiological hazard and residual contamination was found at the sites after completion of the tests.

Radiological protection employed during the Australian nuclear test programme

113. At the time of the tests in Australia it was known that exposure to high levels of radiation was harmful and radiation safety regulations including designated radiation protection staff were in place at all the tests. Radiological safety on the site was the specific responsibility of AWRE[26].

114. The scientific personnel who designed the UK tests had the relevant contemporary information from the bodies noted earlier. In addition, particular experience in radiological effect and measurement was derived from the test programme itself where, it seems, significant expertise was shared between the UK and the US.

Brenner and others (with others) 'Cancer Risks attributable to low levels of radiation: assessing what we really know' (2003) (SB17/4) and expert report 2010 (SB 11/1).

[26] The Atomic Weapons Research Establishment (AWRE) is subsequently renamed AWE

115. In 1999, scientists at the AWE reviewed the literature and safety measures adopted at the time in the Harrison Report[27] from which is drawn much of the historical information in this section of the judgment.

116. In 1951, Lt Colonel Walking, an officer at AWRE with protection responsibilities, assessed that the risks from the proposed detonation could be managed. In particular:

(i) Initial radiation from the burst would be innocuous at two miles.

(ii) A potentially hazardous fallout zone about two miles wide and ten miles downwind should be used.

(iii) Aircraft should not approach within four miles of the radioactive cloud until at least 24 hours after detonation.

(iv) Ships could anchor five miles from surface zero for an unlimited time if condensers and evaporators were shut down and without this restriction at ten miles.

(v) The safety distance within which no person should be at a detonation unless absolutely essential was 10 miles.

117. It was recognised that distance limits were dependent on accurate meteorological conditions that needed careful monitoring. Advice was then taken from the Medical Research Council and dose levels were established for those closely connected with the detonation, and requirements for protective clothing and monitoring by personal ionisation chambers and film badges for all personnel entering the radiation area. These radiation safety requirements were generally in line with the standards of the day.

118. In 1952 Operation Hurricane Trial Orders identified seven general measures to be taken:

(i) The minimum safety distances laid down in orders were to be observed.

(ii) Firing will not take place until meteorological and tidal conditions are such that there will be no hazard to participants.

(iii) All persons likely to be exposed to radiation are to undergo a special pre exposure medical examination.

(iv) Detailed surveys as necessary of the degree and extent of contamination of air land and water to be carried out.

[27] Harrison and Johnson SB 23/1

(v) Strict physical control of re-entry to the contaminated area is to be exercised and full monitoring and decontamination facilities provided.

(vi) All exposed personnel were to wear protective clothing, carry personal monitoring devices and be accompanied by an escorting radiological surveyor.

(vii) Complete records of radiological exposure are to be kept for all personnel.

119. Authorised dosage limits were set for this operation. A seven person radiological division was set up to monitor distinct aspects of safety plans and radiological measurement and assessment including: implementation of detailed safety plans and control on re-entry, gamma ray measurements from 0-15 seconds and 0-10 hours after detonation, radiation instrumentation, personnel monitoring, contamination survey, decontamination of personnel on return from the contaminated area and collection and measurement of air samples.

Operation Hurricane

120. Detonation took place at Monte Bello on 3 October 1952. Analysis of 1000 films from 273 individuals revealed that personal exposure was less than predicted and the highest recorded individual dose was 42.5 mSv with the mean of 1.7 mSv per day over 28 days. These tests produced a vast amount of data and generated a high degree of confidence that radiological hazards could be both predicted and controlled.

Operation Totem

121. Similar measures were applied for Operation Totem in 1953 although fewer hazards were expected as the devices were of significantly lower yield. Further, this operation involved sampling of the radioactive cloud to be undertaken by aircraft. There were seven objectives for data capture including assessing dose rates, contamination, efficacy of monitoring equipment and decontamination of the aircraft. Of the 186 participants whose doses were recorded, 59 had doses at or below 200 micro Svs (μSv) (that is to say 0.2 mSv). Only 19 had doses exceeding 30 mSv and two of the Canberra aircrew had doses exceeding 100 mSv.

Operation Mosaic

122. In 1956 the Mosaic tests also required HMS Diana to be stationed in the vicinity of the predicted fallout to obtain 'scientific data and to provide operational experience of conditions that were thought likely to arise in the event of nuclear warfare'. Of the 728 personnel issued with film badges 545 had total doses at or below 200 μSvs. 139 badges were recorded for the crew of HMS Diana of which 117 could be processed. These showed no doses higher than 200 μSv. It appears that Lt Colonel Walking's original observations about excluding aircraft from the radiation zone were being modified in the light of the importance attached to the objectives quoted.

123. Turner, a member of the Australian Health Physics Team, in his evidence to the Royal Commission[28] stated:

> 'Elaborate safety procedures were adopted during each major and minor test. After each explosion re-entry to the test area could only be through a suitably equipped and staffed health control. Entry was always restricted to a small number of authorised personnel'.

124. Public concern about the effect of radiation was heightened by an incident in 1954 when fallout from the US Bravo programme contaminated Marshall Islands and caused harm to fishermen.

Operation Buffalo: Maralinga

125. The Maralinga test site, some 190km south of Emu Field used for Totem, was selected in 1954 partly because its remote and treeless desert location made entry easier to control. There was an awareness of the potential impact on the transitory aboriginal population who were vulnerable to exposure because they wore no (let alone protective) clothing. This required a larger exclusion zone and monitoring trays in the area where aborigines may be present to measure any environmental radiation from fallout. In 1956 the Maralinga site was ready for use for the Operation Buffalo series of tests.

126. The Buffalo series consisted of four tests at relatively low levels at the following sites:

    (i) One Tree on 27 September was 15kt yield tower mounted at a height of 31 metres.
    (ii) Marcoo on 4 October was a ground surface burst with 1.5kt yield

[28] Chapter Eight FTT B14 252

(iii) Kite on 11 October was a 3kt yield air dropped explosion over land from a height of 150 metres.

(iv) Breakaway on 22 October was another tower mounted with 10kt yield at a height of 31 metres.

Radiological Safety

127. The radiological safety procedures for Buffalo were contained in the Radiological Safety Regulations Maralinga issued by the Director AWRE on 29 March 1956[29]. The regulations covered maximum permissible levels of exposure based on the then ICRP recommendations (November 1955) including external and internal radiations.

128. Weekly and integrated dose levels were again deployed, based on the latest scientific understanding and according to the need for exposure of the personnel concerned. The relevant limits were 30 mSv for the lower integrated dose and 100 mSv for the higher integrated dose.

129. As with the earlier tests, film badges were to be worn by personnel at all times to measure gamma radiation. These were processed in the Health Physics Centre and cumulative dose records based on the results were maintained. For participants employed on tasks outside the forward area not all film badges were collected and developed. Personal monitoring was to be primarily the responsibility of the individual. Personal ionisation chambers and portable rate meters were available to all personnel working in active areas or buildings. Air sampling was to be done in areas with a potential for inhalation risk and neutron and gamma levels measured by fixed instruments where necessary. In addition, periodic surveys were to be carried out by Health Physics representatives.

130. The Regulations classified areas into Active and Non-active, with the former being further divided into Blue, Red and Yellow areas. In these areas there might be a risk of radiation and protective measures were required in Red and Yellow areas. Signs were to be displayed at all entrances to an active area. No-one was allowed to enter the active areas without permission. Protective clothing was to be worn in Red and Yellow areas.

[29] FTT B14 248

131. In the view of the Australian Royal Commission the radiological and physical safety arrangements for participants were well-planned and sound. Security was strictly policed during the major tests but relaxed afterwards[30]. Carter found evidence that the controls were not always completely implemented at least in respect of delineation of the active areas[31].

132. As previously noted, accurate meteorology was key to predicting fallout. The AWRE report No T25/58[32] describes fallout at Maralinga Village following Round 3 (Kite). This was due to change in wind direction from south-west to north-west resulting in the cloud blowing to the south. A record from a background counter in one of the laboratories in the village showed that fallout started to arrive shortly before eight hours after detonation (H+8) and there was no further fallout after about H+9. Sticky paper samplers exposed in the village showed a fallout deposition of 0.09 c/m corrected to H+1[33]. This was considered marginal but requiring extra precautions for further detonations.

Radiological Monitoring

133. There was radiological monitoring of people, equipment and the environment using a variety of instruments to survey dose rates and contamination including the following:

    (i) Personnel were issued with film badges. For those outside the Forward Area these were changed monthly though not all were collected and developed.

    (ii) Dosimeters (quartz fibre electroscope) were carried by the Health Escort or Group Leader in areas where there was a risk of radiation and in the case of the Indoctrinee Force a radiation monitor, usually a Survey Meter type 1324.

    (iii) Detailed testing for ground contamination was done using a 1390A type monitor for gamma radiation and the 1391A for gamma/beta immediately after Rounds 1 and 2 as reported in the AWRE Report No.

[30] FTT B14 252
[31] SB10 161 Page 50
[32] Operation Buffalo Theoretical Predictions of Cloud Height and Fallout August 1958 (SB8 132)
[33] Para 7 p. 20

T49/57[34]. Canberra and Varsity aircraft were used for cloud sampling and the Varsity and Whirlwind helicopters for radiological survey. Contamination measuring on aircraft was done using the 1320, 1324, 1324A and NIS 44 instruments. They did not cover the full range of exposure encountered.

(iv) Testing of the environment was done by air samplers, cascade impactors which measure the particle size down to a millipore, and sticky paper collectors. More than 30 sites were established within a 6 mile radius of the test site with one type of sensor at each site.

(v) An Australian-wide sampling network involving 86 stations was set up. Each of these had a sticky paper collector while 76 of them had air pumps with filters to determine the level of radioactive contamination inhaled by people. Rain was collected at 13 meteorological stations and water tested at 14 reservoirs.

134. Despite the variety of instruments used, as the Australian Royal Commission acknowledged, the existing records of personal radiation doses may be incomplete and inaccurate.

135. The Carter Report[35] states that the evidence both written and anecdotal indicates that the radiological controls implemented for the Buffalo series were the most thorough for any part of the total test programme. Health physics management was carried initially out by AWRE personnel with the support of the Australian Health Physics team. By round three, control of access to the test area was managed directly by Australians. Access to Forward Areas was through semi-permanent control points. There was a large caravan at each of these points that provided a dressing area for putting on protective clothing and showering for personnel leaving the contaminated area. Despite these controls it was noted that there was no evidence that RED and BLUE areas were ever clearly marked and control points do not appear to have been set up for RED areas.

Aircraft Contamination

[34] Operation Buffalo: The Radiation Survey of Ground Deposited Radioactivity' by J. J. Rae received on 22 July 1957. (SB 8 126).

[35] (SB10 161 Page 48)

136. In his Witness Statement[36] Mr Battersby states that his duties at Maralinga included the decontamination and servicing of the 'Sniffer' aircraft. The AWRE report No. T22/57[37] dated July 1957 show that Canberra and Varsity aircraft were used for cloud sampling. Those used for Buffalo had already carried out this role for the Mosaic tests and rather than being decontaminated before being used on Buffalo they were resprayed to seal in the contamination. In addition to the aircraft the main body of the Active Handling Flight (formerly the Decontamination Team) was common to both operations. It was based at the airfield at least 10 miles from the range. The AWE Report Aircraft[38], written in 1998 does not list Mr Battersby as part of this flight, although it seems that Appendix J was largely compiled from dose records.

137. The 1957 report records the decontamination process and the four measuring instruments used (types 1320, 1324, 1349A and NIS 44 monitors). It noted that accurate measurements in the course of the decontamination work were not possible owing to the absence of a suitable instrument covering the full range of fields encountered. There was first Inter-Round action to render the aircraft clean to touch for servicing. This was a wash down with Detergent GS. The final decontamination activity was to bring the aircraft within the fixed and loose contamination tolerance levels laid down in the Maralinga Radiological Safety Regulations. This included stripping off the barrier paint. Due to the short time interval between tests the treatment of aircraft between rounds was at most perfunctory but all were washed down in some way after each sortie. The levels of contamination found constituted a relatively severe handling problem in the first few days after firing.

138. The state of the finish on the aircraft was far from satisfactory. Many bare patches, moving rivets and peeling of top coats were apparent. The roundels in particular showed severe crazing. Decontamination on defective finishes was far less efficient than on a good smooth surface. The report recognised that this was an exposure risk and recommended the use of a light gantry platform capable of spanning the wings of the aircraft to enable ground crews to avoid walking over a highly contaminated surface when servicing, refuelling or decontaminating.

[36] SB 16 A2 dated 12 December 2012)
[37] Decontamination Group Report at Part 3a Aircraft decontamination at the UK Atmospheric Nuclear Tests SB 8 129 and B9 88)
[38] Decontamination at the UK Atmospheric Nuclear Tests SB 8 129

139. Further the report noted that the situation is complicated by the extensive and regular servicing required by the aircraft. The beta field from the aircraft was reduced to a satisfactory level after decontamination but gamma levels remained high, particularly near the engines since no internal treatment was possible. The report commented that carelessness on the part of the ground personnel leading to excessive skin and clothing contamination must be overcome by stricter supervision and more thorough indoctrination and training.

140. In conclusion, it recommended that amongst other improvements in future trials a barrier paint be used as a matter of routine. All aircraft should be coated prior to the operation and further coats should be applied after each round to facilitate handling and servicing.

Indoctrinee Force

141. A feature of these tests was that the War Office wanted a group of officer spectators (Indoctrinees) to be present during the detonation to obtain operational experience of what a nuclear detonation was like.

142. This group comprised of 283 service personnel (mainly officers) and a few civilians from UK, Australia and New Zealand. They visited the blast area to view at first hand the impact on the ground and on service equipment and structures of the blast. The whole force took part in Round 1 (One Tree) located at North Base for the explosion some 8.5 km from ground zero while a smaller group of 100 were selected to witness Round 2 (Marcoo) with four UK officers in a Centurion tank, 24 officers in covered shelters with 500 millimetres of overhead cover at a range of 1.7 km from ground zero and the remaining being a mix of officers, scientists, health physicists and others civilians at a witness stand 2.7 km from ground zero. They did not re-enter the target response area after Round 2.

143. The review of information conducted by the Australian Royal Commission showed the total average exposure of this group was less than 5 mSv with a maximum of 20 mSv. This was further confirmed in the Harrison Report. There is no evidence to suggest that Mr Battersby was part of this force.

144. The Harrison Report then notes[39]:

[39] 10.17 p31/57

> 'The accusation has frequently been levelled at HMG that the service Indoctrinees were "guinea-pigs": i.e. that they were deliberately and for experimental purposes, exposed to weapon effects that were known to be injurious. These allegations emerged during the planning for Operation BUFFALO and were immediately rejected by both UK and Australian authorities. The facts are that, as has been set out above, the Indoctrinees were observers of the target responses trials and not part of them. They were at all times subject to the Radiation Safety Regulations Maralinga, and their doses were towards the lower end of the total distribution among the Operation BUFFALO participants.'

145. We have no reason to doubt the accuracy of the information provided in this report, including the reason for the participation of the Indoctrinee Force as observers. However, in the light of what is known of the hazards of stochastic effect of radiation exposure, particularly from ingestion of alpha emitting particles, we have considerable doubt as to the sufficiency of the justification for placing people within 1700 metres of the detonation site to observe it. We might add that there would be equal concerns as to the potential health impact of flying through the radioactive cloud within a few hours of detonation to collect samples.

146. The Harrison Report noted doses for participants[40]:

> 'Again doses were generally low. Only 18 individuals, mostly Canberra aircrew, had total doses exceeding the Lower Integrated Dose limit of 30 mSv. No person exceeded the Higher Integrated Dose Limit of 100 mSv. For Indoctrinees, the maximum dose was 20 mSv and the mean dose less than 5 mSv. An investigation into the radioactivity in the cockpits of air sampling Canberra aircraft concluded that internal doses to aircrew from inhaled radionuclides was a very small fraction of the external dose.'

147. In our view, in addition to providing information relevant to aircraft decontamination at Maralinga, these measurements provide relevant background evidence to the assessment of dosimetry when we come to examine the tests that were conducted on Christmas Island.

Contamination Post Maralinga Tests

148. An AWRE Report from 1967[41] reported that during the period October to December 1966 an AWRE party assisted by Royal Engineers conducted a radiological survey of firing sites at Maralinga and Emu. The surveys were made with monitoring instruments of the external gamma and beta dose rates and soil samples taken to determine the extent of neutron induced activity. In areas where plutonium had been

[40] At 10.15-16
[41] Interim Results of Op RADSUR a radiological survey of Maralinga Range and Emu site 6 Feb 67) (FTT B8 51)

deposited surveys were made with x-ray monitors and surface and depth soil samples were taken.

149. Three sites at Maralinga known as Taranaki, TM100/101 and Wewak were shown to be contaminated with plutonium. These were the sites used for the minor trials.

The Grapple Tests

Malden Island

150. The first detonation in the Grapple series took place at Malden Island some 700 kilometres from Christmas Island (CI). None of the veterans in these appeals were located in the vicinity of these tests and a description of them is not necessary. The aircraft that dropped the devices were based at CI and although none of the veterans based there was concerned with de-contamination of these aircraft, we note that Mr Hallard has taken into account as a possible exposure pathway leakage of water used in the decontamination process into the fresh water pools from which drinking water was drawn.

Christmas Island Grapple X Y and Z

151. The data obtained from the Australian tests gave the supervising scientists responsible for the CI tests substantial experience of the radioactive effects of a detonation and the range of radioactive deposition when the Grapple X Y and Z series were devised. On the basis of this data it was decided that the next detonations could take place on the same island as service personnel and facilities were based. It was concluded that there were greater risks in sending planes on a 700 mile return journey to an uninhabited island[42].

152. Equally, it was decided that there was no good purpose in issuing photosensitive badges to all personnel on the island, as had been done at Maralinga, as there had been little or no evidence of radioactive measurement in most of these badges[43]. The predictions based on scientific modelling and the assessments of the information derived from the previous tests had indicated that it was only those who were directly in contact with radioactive material who were at risk of exposure to either acute radiation or fallout by reason of proximity to the detonation site.

[42] Harrison Report at 13.1 see also Personnel Safety Plan Grapple X, Y, and X FTT B1 5/6/7/.
[43] Harrison Report 1999 SB17/9 11.17

153. For the Grapple series, badges would only be issued to those who handled radioactive materials, the Canberra air sampling crews and those on board ships in the target area. Although this decision has subsequently been the subject of adverse comment from the appellants' experts, it was not an arbitrary one based on either cost cutting or indifference to or ignorance of the health effects but a piece of reasoning based on observation and analysis. Nevertheless, one consequence has been to introduce an area of uncertainty into the primary narratives of these appeals. With the exception of Mr Butler, none of the veterans with whom we are concerned were issued film badges. This means that there is no direct evidence of any gamma and beta radiation to which they may have been exposed.

Risk Assessments

154. The Grapple X and Y detonations were intended to be high yield (a greater explosive impact) achieved by size of the device and the planned reactions. They were delivered by aircraft rather than a fixed balloon mechanism or other low altitude platform. The detonations were thus planned for a considerably greater height than those at Maralinga.

155. From the Harrison Report, the evidence of Ken Johnston and the other AWRE papers before us, we can summarise the risk assessments as being informed by the following considerations:

(i) Exploding the device at an altitude of around 8,000 feet (2500 metres) would mean that there was no fireball contact with the earth and sea. If it was not a ground burst, the mechanical consequences of such an event (bringing into the explosion particles of earth or water that would be contaminated by radiation and would have to be deposited in the environment in due course) would not arise.

(ii) Having a high yield detonation would mean that the large quantity of plutonium needed to generate the high yield and the relevant quantity of uranium deployed to trigger the detonation would be substantially used up in the intensity of the explosion with the residue reduced to tiny particles of a size and weight that would be taken into the atmosphere.

(iii) The location of CI was chosen on account of both the prevailing weather systems and the presence of the Pacific Ocean to receive and dilute any

radioactive fallout. The planning envisaged that after detonation at 8,000 feet the radioactive particles that formed the body of the cloud would rise through the troposphere and reach the stratosphere at around 55,000 feet. Once at this height it would be distributed by the stratospheric winds all over the atmosphere and would be unlikely to come down in the equatorial zone. Previous observations had indicated that such material became part of the background radiation and came down most regularly in temperate zones of both hemispheres.

(iv) It was recognised that attention was needed to be given to wind direction at the various levels of the atmosphere. These were ground wind; low to medium level tropospheric winds up to 10,000 feet and higher level tropospheric winds from 10,000 to the tropopause at 55,000 feet. The tropopause marks the boundary between troposphere and stratosphere and the stratospheric winds when the highest part of the atmosphere is reached.

(v) It was also recognised that the possibility of rainfall during or in the immediate aftermath of the detonation was a potential confounding factor that could bring wet deposition of radioactive particles to the ground despite the previous calculations. A number of contemporary documents indicate that there was full awareness of the potential for contamination by rain.

156. We accept that the fact that these risk factors were known about and considered does not mean that measures taken to address them eliminated all risk of radioactive deposition on CI. There were potential problems in predicting the place and intensity of fallout. These included fractionation (where the particles in the radioactive cloud bind together as a result of the reaction and thus become heavier); hot particles (deposition of large intensely radioactive particles), unpredictable and changeable wind patterns at different heights of the trajectory, localised rainfall (inhomogeneous rain) and other matters that might have led to hot spots (higher than predicted areas of radioactive depositions).

157. All these topics were the subject of analysis in the previous evidence (written and oral) that has been given by Professors Regan and Dr Nicholson on behalf of the appellants. They formed a significant part of the HL group of appellants' statement

of case of possibilities lodged in response to the case management directions of Charles J.

158. However, in our view, the contemporary safety assessments and predictions reflected in such documents as the Interim Reports on radiological measurements for Grapple Y[44] and Z[45] and the regulatory regime put in place to address risks provide a coherent starting point for the assessment that has to be made. The assessments were made on the basis of substantial previous experience and lessons learned. They were also supported by subsequent measurements taken.

159. In 1993 the Clare Report reviewed the evidence of Environmental Monitoring at Christmas Island 1957-1958. This report is important information as to the source of the risks that were known about and the results obtained after all the detonations on CI. The authors note at [12]:

> 'It was well known that radioactive materials such as plutonium, emitting alpha radiation, were present in fallout from a nuclear detonation as well as beta and gamma emitters. In view of the relative ease of detection of beta and gamma radiation compared with alpha radiation it was therefore decided that the former would be measured to monitor the environment. Additionally it was known that the hazard from beta- and gamma emitting radionuclides overwhelmingly dominated in the short term, that posed by alpha emitters.'

160. At the 2013 appeal there was consensus between Professor Regan and Mr Johnson that co-deposition of particles in fallout meant that it was reasonable to measure alpha radiation by calculation from the amount of beta and gamma radiation, as long as the measurements of the latter were taken at a time when there was still such radiation to measure. This is because the amount of the alpha radiation can be calculated back to an hour after deposition by using the information known about the half-lives of the nuclides.

161. Dr Busby in his submissions is more sceptical about whether monitoring for gamma and beta radiation will enable a calculation to be made about all alpha emitters and he particularly emphasises the risk from uranium. We will examine the risk from uranium at Part Four.

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[44] J.R. Jones (1959) CB 17/13)
[45] Trial Panning Branch AWRE November 1958 SB 17/14.subsequent

162. The Clare Report noted that a remote risk of a low altitude detonation was contemplated in the planning of these events. It was foreseen that this risk may result from an accident in loading weapons or a crash on take-off.

Grapple X

163. According to Mr Hallard, no specific safety regulations have been discovered in the documentation now available for Grapple X. Our reading of the AWRE reports supports the assessment subsequently made by him that the same regime was deployed as at Maralinga.

164. Grapple X was detonated on 8 November 1957 at a height of 2,200 metres (approximately 7,000 feet). It was dropped by an aircraft over the sea off the south east of the island. It had a yield of 1.8 megatons.

165. The Harrison Report (1999)[46] summarised the outcome as follows:

> 'As planned, there was no fallout on Kiritimati; the prevailing north easterly trade winds took the cloud away from the inhabited areas. There was some minor blast damage to buildings but there were no injuries to personnel. Recorded doses to participants (were retained). Doses were generally low. Only 12 individuals, mostly Canberra aircrew, exceeded the Lower Integrated Dose Limit of 30 mSv. Three individuals, all Canberra aircrew, exceeded the Higher Integrated Dose Limit of 100 mSv.'

Grapple Y

166. The Regulations for Grapple Y are available: 'Radiological Safety Regulations Christmas Island' were issued in March 1958 and again were based on the fifth edition of the Maralinga Regulations.[47] The Introduction states:

> 'Radiation which may be encountered during a trial may be α particles, β particles, γ rays or neutrons. Under properly controlled conditions, work involving exposure to these radiations can be carried on in perfect safety. Excessive exposure, however, results in damage to the human body. The danger is insidious because the effects are not immediately felt and damage may become apparent only after a period of years. Damage may arise not only from external exposure but from irradiation of internal organs as result of ingestion, inhalation, injection into the bloodstream through cuts abrasions, or even by absorption through an intact skin. The maximum permissible levels of the various radiations and radioactive substances are based on the recommendations of the International Commission on Radiological Protection, and have been approved by UKAEA and other Authorities concerned. These are the levels to be used throughout a trial. The object of the regulations is to ensure complete protection both of staff and of the general public, whilst imposing the

[46] SB 17/9 at 13.5
[47] FTT B1/8

minimum interference with work. To this end, the regulations will, at all times, apply to all who are concerned with the Christmas Island Trials, both servicemen and civilians wherever stationed.'

167. Effect was given to these aims in the body of the Regulations by:

(i) adopting the same maximum integrated dose limits as were employed at Maralinga, based on ICRP standards for occupational workers for the various sources of radiation;

(ii) dividing the island into controlled and uncontrolled areas determined by contamination levels;

(iii) film badge monitoring for those who worked in controlled areas;

(iv) requiring protective clothing to be worn by those in controlled areas;

(v) laundering and disposal of contaminated clothing after use;

(vi) controlling the movement of radioactive substances in preparing the devices and the means of delivering them;

(vii) establishing a monitoring regime to ensure both the protection of personnel and prevent contamination interfering with the scientific analysis of the tests.

168. The monitoring regime included: the provision of personal monitors (whether film badges or other more specialist equipment depending on assessment of risk); air monitoring of laboratories, workshops or special operations where there was an inhalation risk and monitoring of vehicles and equipment. The available equipment on the island included two types of Geiger Mueller counters able to detect gamma and beta radiation. More sophisticated tests had to be conducted on more specialist equipment back at Aldermaston.

169. There was also some environmental monitoring that will be considered below. All relevant data was to be obtained by the Health Physics Adviser to maintain effective control over the hazards of fallout.

170. Regulatory precautions included a shipping exclusion zone whose size depended on wind speed. No firing was to be permitted if the mean wind from surface to maximum cloud height exceeded 25 knots.

171. A safety plan and regulatory regime based on the previous model was devised. Personnel on the island were divided into three classes and mustered before the explosion to facilitate evacuation in the event of an accident.

172. Controlled areas were divided into three types: Blue (lowest level) where there was a risk of external radiation penetrating but not of internal radiation through inhalation or ingestion and no special clothing was required to be worn; Red area (medium) where there was a risk of external penetrating radiation and of slight inhalation or ingestion and clothing as directed by the Health Physics Controller was to be worn; and Yellow (highest) where there was a risk of serious inhalation or ingestion and full protective clothing was to be worn.

173. Those not immediately concerned with the operation or its support (which group comprised all the veterans we are concerned with) were to be formed up in transport at Main Camp or at Port London.

174. Regulations required that the Officer in Charge was to ensure that regular contamination checks were made to ensure that radiation was below the maximum values laid down and in addition periodic surveys were to be made by the Health Physics organisation.

175. The Personnel Safety Plan for what was intended as the largest of any UK trial included division of personnel according to function and those in the open were to wear protective suits, sitting with backs to the detonation and in the event of a contaminating incident all those not essential to the recovery were to be evacuated on ships to take them away from the fallout plume.

176. Following detonation, badges were reviewed and once again doses were generally low with nine individuals exceeding the Lower Integrated Dose limit of 30 mSv and six the High Integrated Dose Limit of 100 mSv, all of them were members of the Canberra crew.

177. A summary of the meteorological data recorded at the time and retained on the records was made in 1985[48]. It said:

> 'All radio-sonde and radar-winds ascents for the time of the test and up to 24 hours later were examined. The 1, 1.5, and 2 km winds meaned to obtain the low flow for

[48] See Annex A to the expert report of Richard Stretch SB13/45 p 42/51

> detonation at 2.4km and the 1km wind was used for those at 0.45km (the only lower wind in the official record is the surface wind at 12 m above ground level which may not be representative of the higher flow). A graph was plotted of those winds for 0,6,12 and 18 hours after each detonation. Directions varied from 082° to 109°, and speeds from 9 to 28 knots, except for the period up to about 9 hours after the test at 1905 GMT (10.05 local) on 28 April 1958 when winds were light and near to being south easterly (135°). Since the airfield and camp were almost due north west from the explosion, the winds on this occasion were examined in more detail……
>
> Assuming particles were released at 2.4 km, 22.3 miles to the south east of the camp (direction 142°) it appears that those with fall speeds of about 1/3 m/s could have reached the camp at .(1200 local time). Heavier particles released from the thermonuclear cloud at greater altitudes could have arrived at the camp later.
>
> Lighter particles could only have been deposited at the camp by being washed out in the precipitation, and there is no evidence of that, although precipitation reaching the surface in a shower possibly caused by the bomb more than 5 kms away (at 11.57) presumably but not definitely to the south east.'

178. The Harrison Report concluded in respect of the whole test series[49]:

> 'In no case did hazard arise from unexpectedly high yield at a UK atmospheric nuclear trial. In no case did hazard from fallout arise due to a failure to accurately predict the weather conditions at the time of firing. In all cases where devices were air dropped or balloon supported, the detonation took place at, or very close to, the altitude intended. In no case did any hazard arise from a detonation taking place at other than the altitude assumed in the planning process.'

Issues arising from the Grapple Y detonation

179. The radiation exposure pathways generated by Grapple Y (GY) were at the heart of the debate at the previous hearing and inform most of the contested issues at the present one. There are a number of possibilities and uncertainties that will have to be taken forward into the overall evaluation, but equally, in our view, there are a number of issues that we are sure are clearly established by the evidence.

The video record of the detonation

180. Mr Johnson's evidence before the previous FTT included a narrative of the GY explosion presented by way of commentary on the video of the detonation retained in the Imperial War Museum that he took the Tribunal through at the previous hearing. Unfortunately there are no timings attached to the video so only a sequence of actions can be described with timings derived from extraneous data.

181. We summarise this evidence from Mr Johnson as follows[50]:

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[49] Para 19.3 p. 41/57.
[50] SB 14/5.2 p.112-125

(i) At 10.05 the detonation took place at 8,000 feet over the ocean at a point 1.3 miles from the southeast tip of the island (ground zero).

(ii) The fireball grew in one second to 8,000 feet in diameter (4,000 feet radius) and its lower edge was well clear of sea.

(iii) There was a sharp pulse of radiation at one second and when the shot broke away from the fireball a few seconds later, then a second burst of radiation after 10 seconds.

(iv) After 20-30 seconds the shock wave induced ring went outwards. This was a pulse of pressure but not a wind. We understand that this is the force that can cause damage to buildings or knock people over some distance away.

(v) The shock wave went outwards to airfield diminishing with distance. It also went over the sea where the vast majority of the energy is reflected. The reflected shock joined up with the outward shock to produce what is known as the Mach stem causing compression and some spray upwards.

(vi) The Mach stem formed from the pressure and moved outwards and produced dust and other debris that rose immediately and eventually degraded into the blast wave.

(vii) Some of the pressure towards the sea is not reflected and 1% of it caused a small amount of spray that identifies the location of the shock wave.

(viii) Two Wilson cloud rings formed: one over the sea because of humidity and one at 10,000 feet.

(ix) The stem of the mushroom cloud then emerged ascending. It is composed of moisture drawn in. It was assessed that most of the radioactive material is in the main cloud but some (10% no more) in the stem with finely dilute particulate.

(x) The video captures the main cloud and quite a few other clouds at a lower level.

(xi) The anvil of the cloud formed on top of the stem and rises very quickly.

(xii) There is an image of the upper part of the stem being displaced by winds that were assessed to run from approximately 10,000 to 40,000 feet.

(xiii) The main cloud soon became fully extended at 52,000 feet when it could be seen against a clear blue sky.

(xiv) It then pushed into the tropopause and thence into the stratosphere at 55,000 feet (over 16 kilometres).

182. Overall, Mr Johnson is thus describing the upwards progression of the main cloud from 8,000 to 55,000 feet when the stratospheric winds will carry it far away from the island. The stem reaches down to earth and is therefore liable to be blown by the middle and lower level winds. He pointed out that his rough diagram of lower level tropospheric winds (somewhat simplifying the picture obtained from Mr Stretch's diagram) might explain the radioactive contamination subsequently recorded in sticky paper sampling at the narrow strip of Vaskess Bay an uninhabited area south of the lagoon and towards the south west tip part of the island (see [211](i) below).

183. As to movement of the main cloud, Dr Nicholson indicated that the video alone could not be used to determine where the clouds went. He also thought the speed of the tropospheric winds was less important than the expansion of the cloud at 50,000 feet that is clearly seen.

184. From the available material, Mr Johnson has made his own rough calculation of the diameter of the cloud at its height. He suggests that it measures approximately 4.5 x 16 kilometres giving a cloud diameter of 72 kilometres, He was aware that his estimate is somewhat smaller than that made by Flt Lt Pasquini, who had flown a Canberra aircraft after the detonation to 'sniff' the cloud and take measurements. Pasquini had recorded certain matters in his flight log at the time. A number of matters not mentioned in the flight log were addressed in a witness statement made for the purpose of this litigation where he gave an estimate of a cloud diameter 111

kilometres (60 nautical miles)[51]. The relevance of cloud size is that the smaller the cloud's diameter the less there is to overhang the inhabited parts of the island and be a potential source of deposition there.

185. We are cautious about attaching any weight to assertions made as to the mechanics or size of the detonation cloud many years after the incident without independent documentary or other cogent evidence. These are primarily matters of technical expertise by those qualified to record and interpret data and who have the requisite scientific knowledge rather than lay observation, even if some military personnel will have some technical understanding of the issues. Further, the memories of veterans and their comrades will be both fading and capable of distortion, in the knowledge that a campaign for compensation on their behalf was underway.

The height of the detonation

186. The actual height of the GY detonation was of significance for the reasons already explained. If it had been detonated at a height of 4,000 feet or less there would be a real possibility that contact by the fireball with the surface of the land or sea would have been significant and with it the entrainment of additional material into the forming cloud. There were suggestions in the evidence presented by the appellants in 2013 that the detonation height was considerably less than 8,000 feet.

187. We have no doubt that GY was detonated at the intended and recorded height of around 8,000 feet. In our view, it is inconceivable that an error was made about this either by the pilots flying the plane or those responsible for this part of the operation and the scientists measuring it. We are unimpressed by the attempts of Mr Large, in a report prepared for the previous hearing, to make a retrospective calculation of height from trigonometric calculation based the use of photographs of the cloud. Equally the estimates given by some of the veterans in statements first made in the context of these appeals seem to us to be purely subjective recollections unsupported by contemporary documentation. We note the assistance that Dr Ash has endeavoured to provide us from the information provided to him on behalf of the BS appellants, but he was in an impossible position of coming late into this appeal and being provided only with inadequate and limited material and consequently being

[51] September 2011 paragraph 41 to 42 (SB 8/130) and further statement 2013 SB 8/131. A transcription of the flight log is exhibited to his first statement

unable to identify precisely what the photographs were that he was asked to give an opinion on. We do not propose to review his report and oral evidence before us. It is sufficient to state that nothing that he told us causes us any doubt as to the height of GY.

188. Although, Dr Nicholson pointed to one document in the archives that asserts that atmospheric pressure was recorded at the time of the exposition that would suggest that this height was more in the region of 5,000 feet, we are satisfied that this stray reference is an inaccurate pointer to height. It does not raise any doubt as to the evidence of those who planned, executed and recorded the detonation and its aftermath or the emphatic evidence of Mr Johnston on the topic.

189. Accordingly, we are sure that this explosion was not in any sense a ground burst with the entrainment consequences that might otherwise have followed. Further the size of the yield, and the upward direction of travel of the main cloud means that the radioactive particles within it would be predictably small with low terminal velocities for returning to earth.

Wind direction

190. Further, we are also sure that Mr Stretch's meteorological evidence given at the previous hearing derived from contemporary weather data recorded at the weather station gives an accurate picture of wind strength and direction on 28 April 1958 and the rain fall actually measured at the weather station. All the planning documentation emphasised the importance of wind factors to radioactive fallout and health hazards. We have no doubt that great care was taken to monitor and record these matters. His report confirmed the accuracy of the assessment made in November 1985 noted at [177] above.

191. Mr Stretch provided the previous Tribunal with a diagram of wind vectors from the retained data as of 12.00 hours (some two hours after the detonation). His evidence was not disputed. Taking the point of detonation as the centre of a clock face, there was one surface wind (marked 1) recorded at 11.35 blowing just west of north between the 11 o'clock and 12 o'clock positions. There were three (2, 3, 4) low level tropospheric flows blowing north north-west in between the 10 and 11 o'clock positions. There were three medium and high level tropospheric flows (5, 6, 7) blowing away from the island in the 7 to 8.30 o'clock positions. There were two high

level winds sub tropopause and stratospheric blowing east or north-east between the 2.30 and 3.30 o'clock positions (8 and 9).

192. Of these only the surface wind (1) was heading directly to the inhabited north part of the island. However if surface is taken to mean between 12 and 450 metres as per the 1985 report, then a surface wind at this time and place would have little relevance for a detonation at 2,500 metres where the product was by and large rapidly ascending. The low level tropospheric winds (2, 3 and 4) are by mainly skirting the southern edge of the island although the more northerly of the three appears to be heading to the lagoon area. The other winds are all blowing away from the island.

193. Dr Nicholson sounded a note of caution about the application of the meteorological data obtained at regular intervals to what was happening at the site of the explosion. He suggested that the shock wave itself would make it impossible to predict the behaviour of local winds with regard to the dispersion of radionuclides as there would be significant and sudden change. He concluded:

    (i) Dry deposition of material not entrained in the mushroom cloud (i.e. from the stem) could have occurred. Such deposition would have been sporadic and dominated by large particles, although impact cannot be assessed on the available information.

    (ii) It is improbable that there was dry deposition from the top of the mushroom cloud.

    (iii) It is quite possible that rain formed in the mushroom cloud given the relative humidity of the latitude of the island.

Rainfall

194. The topic of rainfall is more debatable. The weather station was located in the inhabited north coast of the island approximately 0.8 kilometres (kms) from the Main Camp and roughly 3 kms from the airport. Base camp (where some reports of rain were made) was between these locations (Stretch 2 10/48 3.1). At 5.27 am on the morning of the explosion light rain was collected there. No more was reported that day as having fallen at the weather station.

195. Mr Stretch explained that:

(i) The logs that have been retained record rain hourly, but rain that fell at the station in the intervening periods would have been noted in the remarks column of the log.

(ii) Adjacent rain was recorded 5 kms away at 11.57 am that day. Distance was a human estimate dependent on the known distances or visibility points in an essentially flat island. Adjacent rain would have been recorded, if observed on the hour when the record was made but not in the intervening periods.

(iii) Given the thermodynamic evidence, the wind shear and the reporting of cumulo-nimbus (CB) clouds, it is reasonable to infer that a shower or showers could have developed at any time not related to the explosion.

(iv) Subsequent observations at 10.56 and 11.57 indicate that the second sighting of CB cloud was a direct result of the bomb. As with any CB storm, rainfall would have been localised in heavy downpours in association with storm downdraughts but other areas remaining dry.

(v) Given the corroborative accounts by ground personnel of heavy rain within the hour after the detonation in the north of the island, there seems no reason to refute them although there was no official record of rain at or after the explosion at the weather station or anywhere else on the island.

(vi) From the photographic evidence of the anvil/canopy of the detonation umbrella cloud in its initial stages, it is thought to have contained super cooled water or a mix of water with ice crystals even with ambient temperatures below -40° C.

(vii) Therefore, the report by Flt Lt Pasquini of rain at 40,000 feet seems plausible, although this was not a report recorded in his flight log at the time, but first mentioned in his witness statement for the purpose of this litigation.

196. In addition to the Pasquini evidence, there are a number of witness statements made by veterans located at Port London and elsewhere on the main coast of the island shortly after GY was detonated. Both Mr Stretch and Dr Nicholson were agreed that the weather records at the meteorological station were not inconsistent with adjacent

precipitation and it has not been suggested that we should reject these accounts, although they were first made some 50 years after the events, for the purpose of compensation, and point to no extrinsic record confirming the account.

197. Dr Nicholson thought that a probable explanation of the contamination recorded at Vaskess Bay was fallout deposited by spots of rain once low level winds changed to blow westerly. He thought it would be a miracle if the contaminated rain was simply confined to Vaskess Bay.

198. If there was post-detonation rain, as the witnesses reported, it was possible that it was from low level cloud rather than the high level radioactive cloud. Equally, it was possible that it originated from the radioactive cloud, although the ability of rain drops to reach the ground without evaporating would depend on how quickly it would freeze.

199. Although we find that it is somewhat surprising that the lay evidence of rainfall over the Island at the time of GY has come into existence as late as it has and does not appear to be supported by contemporary records, there can be no certainty that there was not rainfall other than as recorded at the weather station and we must take the possibility of some wet deposition from rain into account in reaching our overall evaluation.

200. Mr Hallard reviewed this material along with the reports of rainfall in the witness statements prepared for these appeals. He concluded that rain deposition was the most likely source of fallout deposition, although the possibility of some dry deposition cannot be excluded. He also noted evidence of rainfall in contaminated cloud seven hours after detonation when a Shackleton aircraft was conducting a survey of airspace about 100 miles west of CI.

201. From the meteorological and other information already noted, we would tend to agree with the proposition put to Mr Hallard in cross-examination by Mr Ter Haar that such contamination was more likely to have been from the residue of the stem blown westward than the main cloud, by now in the stratosphere and beyond the flying height of a Shackleton. If so, this has two consequences: first, the diameter of the stem would be considerably smaller than that of the main cloud; second, the scientists estimate that the stem only contained 10% of the radioactive residues from the detonation.

Grapple Z

202. We have given a summary of these tests in the introduction to this determination. Mr Hallard was able to review the personal safety plan, the measurement reports following each detonation, and in addition there is in existence an Imperial War Museum Video of the Grapple Z (GZ) tests. In his view as a health physicist, this showed excellent facilities for control and decontamination even by modern standards. The Grapple Z Interim Report (ref 18 to Hallard) refers to initial surveys of dose rates at 1 metre for all four GZ tests with summaries of results for both fallout and activation products. The same report provides some evidence of area monitoring and control surveys.

203. The badge results once again showed that only 37 members of the Canberra crew exceeded the lower integrated dose, of whom 8 exceeded the higher integrated dose.

204. The GZ tests took place during the dry season. There was only one recorded instance of rainfall on 22 August 1958, the day of GZ1, when radioactivity to a maximum level of 0.15 Bq per millilitre was found.

205. Three clams sampled off the south east coast of the island after GZ1 and GZ4 were reported to have radiation at the level of a few pico-curies. Using a conversion table from the Carter report Mr Hallard has converted this reading to 2 nano-sieverts (one thousandth of a micro Sievert). There was no record of rain during the remaining GZ tests. They took place during the dry season.

206. Given the height of the detonations and the yield, there may well have been ground contamination and dry deposition of radioactive material.

Radiological Measurements from the Grapple Tests:

Film Badge monitoring

207. The overall film badge results have been set out in a table prepared by Mr Hallard.

Table 1 Film Badge Data from Grapple tests

Test	Participants	Badges Issued	Zero dose	Some dose	Dose over 1 mSv	Highest individual dose in mSv
Malvern	3515	83	4	79	62	92
Grapple X	2340	179	53	126	18	117

Grapple Y	3723	114	18	96	38	130
Grapple Z	4374	618	395	223	97	300

Environmental Monitoring

208. An over-view of environmental monitoring is provided in the Clare Report (1993) and its references. It indicates that there was both Pacific-wide and local monitoring. Pacific-wide monitoring was to confirm that no unseen deposition had occurred on inhabited islands within 2,500 kilometres of CI using pumped air, sticky paper rainwater collectors and fish sampling.

209. As part of this Pacific monitoring there was an air sampling point and a monitoring station for ground deposition at the Joint Operation Centre (JOC) on CI. There was rainfall monitoring at the Meteorological Station on CI and fish were caught around CI for a month after each detonation

210. Local monitoring confirmed that levels of radioactivity on land and sea were negligible and not a danger. This consisted of:

    (i) Sticky papers at several locations at and following GY and GZ

    (ii) Studies of radioactivity in the sea and marine life

    (iii) Radiological surveys using portable monitors all over the island.

211. The sticky paper samples following GY and GZ had revealed only three high readings of contamination. These were:

    (i) At the Decca Master Site, Vaskess Bay (South West corner, uninhabited) following GY. This was a reading of 150 micro-curies per square metre (μCi/m²). The Clare Report states that a subsequent survey using hand-held instruments did not confirm this high figure, although other reports are ambiguous on this.

    (ii) Following GZ1 a reading of 100 μCi/m² and following GZ4 a reading of 300 μCi/m² after extrapolation back to detonation plus one hour. Both these sites were on the uninhabited southern coast of the island at least eight kilometres from the nearest inhabited area.

(iii) The Clare report calculated that a person living for a year with exposure to the highest of these readings (300) would have an effective dose equivalent of 120 micro Sieverts (i.e. 0.120 mSv)[52].

(iv) The highest level of detection in an inhabited area was 2.8 μCi/m2 at the Main Camp following GZ1.This was above the recommended limit of 1 μCi/m2 but it was assessed to be unlikely to have been the product of that test within an hour of detonation given wind speeds and distance from ground zero. No other contamination was found at nearby sites. Otherwise results from other inhabited sites were well below this figure and within recommended limits. These included the Port, JOC, Main Camp, Airfield, Sites C and D where the veterans resided or were deployed at the time of detonations.

(v) The overall conclusion[53] was:

> 'in summary apart from occasional, very localised and just measureable, but radiologically insignificant, fall-out activity, and zero barely detectable but insignificant levels of neutron-induced activity in seawater under the megaton detonations, and on the land under the kiloton detonations, all of which rapidly decayed to negligible levels, there was no detectable increase in radioactivity on land, in the sea or in the air from fall-out directly attributable to the UK atmospheric test.'

212. At the 2013 hearings, there was critical comment made by the appellants' experts as to the adequacy of the sticky tray samples. There were too few of them; they were not systematically placed every few metres; they were vulnerable to the effect of rainfall in the rainy season or evaporation in a hot climate. Deposition mechanics mean that there would be no even capture of radioactive fallout even if it was in the close proximity of a tray. If there was some particle deposition, there was a realistic possibility of hot spots (more intense deposition) and hot particles (more radioactively intense larger particles).The point was made that it was highly improbable that any one tray would capture the highest level of deposition on the island.

213. We accept that the trays cannot be expected to capture everything that might have been deposited. Many of the other criticisms made previously have weight. It is probable that operators endeavoured to ensure that sticky papers were only exposed

[52] Para 28
[53] Para 46

when it is dry and bottles were used during rain[54]. Rain contamination was only a problem for GY, conducted during the rainy season, when there is a real possibility of some local rain having fallen in parts of the island in the hours following detonation. Random deposition of a few hot particles could not be measured by these trays.

214. Nevertheless, we also conclude that there is substance in Mr Hallard's assessment that, although imperfect and incomplete, the data from the sticky paper trays does tend to negate the proposition that there was *general* radioactive contamination from any of the Grapple tests or GY in particular. If such general fallout had existed, even if deposition mechanics may have meant that not every tray would have captured it, it is highly improbable that none would.

215. There is a persistent absence of readings of meaningful quantities (i.e. more than 1 μCi/m²) at any of the inhabited sites in the north. The three occasions when readings of 100 μCi/m² and above were recorded were in the uninhabited south, close to ground zero. In the record of contaminated deposition at Vaskess Bay following GY, the wind readings plausibly suggest that this is where contamination from the explosion would have been deposited before the stem moved west and dissipated.

216. There is some support for this conclusion from the rain and air readings taken over the periods of the test. Again we recognise that samples taken from one sampling station of air and water in the north of the island (Joint Operation Centre and Meteorological Station) cannot be said to exclude the possibility of higher local deposition in an area that was not sampled. However, the sample record is another pointer against overall contamination.

217. Mr Hallard has abstracted a summary of the available data of the highest readings recorded over the period.[55] Some do not coincide with test dates. The readings are in Bq per cubic metre (air) and millilitre (water) and show both maximum and mean concentrations.

Table 2 Environmental Records

[54] See the MacDougall Report of Pacific Wide Sampling (SB 23/5/ p.4)
[55] We have combined the tables at pages 32 and 33 of his first report SB 2/2/14

Test	Sample Date	Air (Main Camp) Bq per m3	Water Bq per ml
Grapple X 8.11.57	8.11.57	0.04 max 0.04 mean	No record
Grapple Y 28.4.58	30.5.58	0.6 max 0.06 mean	
	7.7.58		0.03 max 0.01 mean
GZ1 22.8.58	22.8		0.15 max 0.09 mean
	24.8	0.16 max 0.07 mean	
GZ2 2.9.58	10.9.58	0.13 max 0.05 mean	No rain
GZ3 11.9.58	13.9.58	0.06 max 0.04 mean	No rain
GZ4 23.9.58	27.09.58	0.06 max 0.04 mean	No rain

218. Further support for the absence of generalised radioactive deposition can also be obtained from the fact that no significant contamination was reported in the numerous surveys and clean-up operations conducted after all the tests were completed (including the US activity after British use of CI had ceased). In this context we remind ourselves that alpha emitting particles have long half-lives. If they had been deposited on land and had not all been washed away, there would be traces of them some years later.

219. The absence of evidence of such deposition is the consistent theme of a number of reports. First, there is a further report to this effect from Dr Harrison and others on Radiological Decontamination at Christmas Island[56]. There is a report from Mr Oldbury of the UK Atomic Energy Authority [57] (1964) which reveals that there was

[56] SB 17/10 pp 5-15
[57] SB/3/9

known contamination to buildings and equipment in the aircraft decontamination area near the airfield in the north of the island. Apart from operations to remove this contaminated equipment, the Report reveals at 4.5 that numerous surveys and spot checks were conducted over the island generally, including all operational areas, but no residual contamination was found at any site, with the exception of some contamination that was just detectable at the touch down area of the aircraft runway.

220. In 1977 the University of Washington published a Radiological Survey of Plants, Animals and Soils at Christmas Island and Scene Atolls in the Marshal Islands[58]. This was preceded by a preliminary report in September 1975 from Mr Seymour (the Director of the Laboratory of Radiation Ecology at the University of Washington)[59] devoted to CI. Trace quantities of fallout radionuclides were present, and the amounts were less significant than naturally occurring radionuclides (K40 and U238). Of the eleven fallout radionuclides detected only caesium-137 was found in a concentration greater than 37 Bq per kilogram.

221. The island of Kiritimati (Christmas Island) became part of the independent Republic of Kiribati in 1978. The Republic wanted specific assurance on environmental radiation and in 1981 a report of the National Radiological Laboratory of New Zealand, 'An Environmental Radiation Survey of Christmas Island Kiribati' by McEwan and others[60] was commissioned. The report found that there was low natural radiation, and while traces of residual contamination from the nuclear weapons era were detectable in a few localised areas, notably where aircraft had been washed down for decontamination[61], in all cases:

> 'concentrations in soil and vegetation remained several orders of magnitude below derived reference level concentrations. No radioactive contamination was detected which would present a hazard to resident islanders[62]'.

222. Caesium-137 was detected in groundwater and lagoons at rates of between .002 and 0.05 Bq per litre. Traces of plutonium-239 were found at rates of less than 0.07 to 1.13 Bq per kilogram.

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[58] SB 6/79
[59] SB 6/77
[60] NRL 1981/9 SB8/128
[61] P. 10
[62] At paragraph 7 p.11

223. Other surveys and cleaning up operations were commissioned in 1998, (the Aspinwall Report)[63]and 2004 (the Enviros Report)[64]. These reports are consistent in not disclosing significant radioactive contamination on the island generally and its inhabited areas in particular. In assessing them account needs to be taken of the subsequent use of CI by US forces for test activity.

224. The Aspinwall Report found traces of plutonium-239 and -240 in parts of the islands connected with military operations including the Joint Operation Centre Laboratory, Bomb Loading Ditch Area, Laundry soakaway, Washdown Pad, Aircraft Soakaway area and soil from Ground Zero Balloon shots. The effluent soakaway at the airport laundry. This is consistent with the hypothesis that apart from the site of the low altitude GZ detonations, it was the washing of clothes or aircraft that was likely to result in contamination.

225. This was the broad evidential picture of the Grapple tests and the measurements of their consequences that was before Mr Hallard. In Part Five of this determination we will consider his evidence in some detail.

PART FOUR

THE BS APPELLANTS' CHALLENGE TO THE ICRP MODEL

Introduction

226. The written submissions of the BS appellants address a number of issues, both scientific and philosophical and can be summarised as follows:

    (i) The current ICRP radiation risk model is unsafe to apply to the veterans generally because:

    (a) the veterans were exposed to internal radioactivity and the ICRP model is inadequate for assessing the health effects of internal radiation;

    (b) they were exposed to uranium which exhibits anomalous genotoxicity and the ICRP model is not appropriate to assess the health risks from internal exposure to uranium;

    (c) there is new biological evidence of non-targeted effects at low doses that has resulted in a paradigm shift and the ICRP model in its most recent iteration has been superseded as a result of this shift.

[63] Aspinwall and Co 1998 SB 8/137
[64] SB8/125

(ii) CLL (Battersby) and pancreatic cancers (Smith) are both radiogenic. Four veterans out of the original 13 appellants had pancreatic cancer recorded. This is such a very high degree of improbability that by itself it indicates that this group shared an exposure to some genotoxic agent and that this can only have been some agent at the test sites.

(iii) The continued use of the ICRP model and the rejection of criticisms made of it by the European Committee on Radiation Risks (ECRR) group is evidence of bias (conscious or unconscious) by those who are in some way connected with the nuclear industry (civil or military).

(iv) Overall, the criticisms made of the ICRP LNT model made by ECRR and its supporting scientists advanced in this appeal, mean there is at least reasonable doubt, as defined by Mr Justice Charles, with respect to the causation issues in these appeals.

227. In this section of our determination we will review the evidence presented in support of the attack on the ICRP model and explain our conclusions on it. We will address submission (i)(c) when we examine the evidence of Professor Mothersill in Part Six of this determination. The BS appellants' submissions on bias will be considered when we examine the objections taken to Mr Hallard's evidence at Part Five. We will address the issue of medical conditions of these two veterans and what might have caused such conditions in Part Seven.

The BS witnesses

228. The direction of Charles J that Dr Busby was precluded from giving evidence in the re-hearing of these appeals was clearly a source of some frustration for him. Sometimes we were required to remind him that he could not give evidence indirectly by making an unsupported submission or by merely citing the abstract of a learned paper in argument but he had to call experts with the relevant expertise to establish the evidential propositions that he relied on.

229. On behalf of the BS appellants, Dr Busby called the following witnesses:

Mr Richard Bramhall

(i) He describes himself as the company secretary of the Low Level Radiation Campaign (LLRC) which since 1995 has been closely involved in the arguments and scientific evidence for an error in the risk estimates of the

ICRP. As a result of his concerns he was appointed to be a member of the CERRIE Committee and his evidence was directed to the workings of that Committee and how the majority conclusions came to be published. He is a musician and has never been trained as a scientist, although is cited as co-author in some papers with Dr Busby as he told us that he checked the grammar in the papers.

(ii) At the hearing Mr Bramhall told us that he had never been a member of ECRR but stated that the LLRC has given the ECRR money. He claims that his skills lie in explaining scientific concepts to lay people, which is one of the reasons he was asked to sit on the CERRIE committee. He stated that the CERRIE process was skewed by bias and manipulation. The reason for writing a minority report was that the drafts prepared by the secretariat on behalf of the other members did not represent his and Dr Busby's views and they found it difficult to propose amendments and so they were reduced to writing dissenting texts which were referred to as "offensive and possibly libellous material".

Professor Malcolm Hooper

(iii) Professor Hooper is an Emeritus Professor of Medicinal Chemistry at Sunderland University. He has been involved with veterans of the first Gulf War since 1997 and serves as their scientific advisor. He was a member of the Depleted Uranium Oversight Board (DUOB) 2001-2006 and was appointed President of the National Gulf War Veterans and Families Association in 2000. In his evidence he comments on the health effects of uranium. He claims that that uranium played a greater role in adverse health effects to the survivors of Hiroshima than has been recognised and that there is evidence that it binds to DNA. Consequently health physics has got the indicators for uranium exposure very wrong. The conventional dosimetry error seems to be upwards of 1000 fold.

(iv) He points to the establishment of CURE (Concerted Uranium Research in Europe) as evidence of a changing view of the toxicity of uranium. In his witness statement he identified Dr Busby as the leading expert nationally and internationally on the effects of radioactivity on the environment and

stated that his qualifications and experience are second to none. He quotes a number of Dr Busby's papers in his report. He was unaware of the contributions of experts of international repute such as Little and Brenner. He cited the Rabbit Roff findings[65] and the Busby and De Messiers observations[66] on them without qualification, although he has no expertise in epidemiology. He has not published original research on the question of uranium toxicity.

(v) He considered that there was little difference between the fact recognised in a long standing paper by Huxley[67] that uranium when combined with acetic acid to make uranyl acetate binds with DNA and the proposition that uranium remaining after a detonation does the same thing. He thought that such uranium will be subject to chemical processing in the atmosphere. He recognised that neither his statement nor the supporting papers he relied on explained this mechanism.

Professor Charles Howard

(vi) Professor Howard is a toxico-pathologist and until recently was Professor of Bio-imaging. He is now Emeritus Professor at the University of Ulster and has published over 130 peer-reviewed papers. He has a long standing association with Dr Busby and was responsible for Dr Busby being granted a visiting professorship at the University of Ulster. He is particularly interested in the issue of photoelectron amplification of external photon radiation (gamma and X-ray) by particles of elements of high atomic number and supervised a PhD student who investigated this effect through experimental work with gold nanoparticles and by computer modelling.

(vii) He stated in his expert report that 'There is unequivocal evidence that uranium has strong affinity for DNA' – citing the Huxley paper. He was challenged on this statement during cross-examination where he was rather less dogmatic instead stating that:

[65] SB10/154 see [255] of this determination where criticisms of this study are noted.
[66] SB6/84
[67] H.E. Huxley and others 'Preferential staining of nucleic acid containing structures for electron microscopy' 1961 SB 7/97. At pp 276-281 this paper reports that uranyl acetate is appropriate mechanism for preparing slides as it binds with DNA

> 'Nobody has ever done any research to know what happens to internalised uranium particles. We can assume that some form of biotransformation will occur.'

(viii) In his report he quoted the 1998 Rabbit Roff paper and the 2007 paper by Busby and de Messieres[68] as persuasive evidence that the test veterans as a group shared some prior exposure to genotoxic stress which caused transmissible genetic or genomic damage. However on cross-examination he accepted that the methodology used (survey questionnaire) was less than ideal as there is a potential source of bias but felt that the papers were still a source of useful information.

Professor Shoji Sawada

(ix) Professor Sawada is an Emeritus Professor at Nagoya University, Japan. He is a physicist whose main area is particle physics and has published more than 100 papers in the area of particle physics. As a 13 year old he himself experienced the effects of the Hiroshima bomb and his mother was one of the civilian casualties of this bomb. He has understandably developed an interest in the effects of ionising radiation on health. He has, however, only published two papers on these effects: 'Cover up of the effects of internal exposure by residual radiation from the atomic bombing of Hiroshima and Nagasaki' in Medicine, Conflict and Survival (2007), and 'Estimation of residual nuclear radiation effects on survivors of Hiroshima atomic bombing from incidence of the acute radiation disease' in the Bulletin of Social Medicine (2011). As the title of the first of these papers indicates, his assertion is that there has been a long term cover up of the effects of internal exposure and that although many Japanese people are suffering from the long term effects of radiation, they cannot be compensated as they cannot prove that their medical conditions are due to the effects of radiation. Neither of the journals in which these papers were published are recognised as the appropriate scientific fora to publish a critique of the work of the ICRP. There was no evidence of rigour or robustness in the way that these papers were compiled, peer-reviewed and published.

[68] See footnote 55

(x) His report explains that the criteria for certifying atomic bomb disease were based on the Atomic Bomb dosimetry system 1986 (DS86) and the results of the epidemiological research at the Radiation Effects Research Foundation (RERF), the successor to the Atomic Bomb Casualty Commission (ABCC). The RERF study emphasised only the primary radiation (gamma rays and neutrons) emitted within one minute of the explosion. He claims that survivors were exposed to both external radiation and internal radiation from the fallout – the so called 'black rain' – but it has been difficult to assess the actual dose because some of the fallout was washed away by the heavy rains accompanying the typhoons. Evidence of internal exposure comes from studies that show small but increased incidence of acute radiation diseases (eg epilation and purpura) among survivors in the region where the primary radiation hardly reached. He states that the results of the epidemiological studies based on the LSS are wrong because the term 'unexposed group' is misleading as they could have been exposed to low doses of radiation. There is also an issue as to whether the number of cancers detected in the exposed group is artificially raised because of the intensive screening programme.

(xi) Professor Sawada was called to give evidence without any request being made for an interpreter. It soon became apparent that there was real difficulty in comprehending his answers, and cross-examination was suspended whilst attempts were made to find a Japanese interpreter. There was insufficient time available to find an interpreter with scientific expertise, but we were assisted by an, understandably anxious, Japanese interpreter who assisted the process of communication. It transpired that the Secretary of State had had equal difficulty in understanding Professor Sawada's original written report prepared for this hearing and had asked for a translated copy to be made available. The report was written in English and so the request for a translation was not understood by the Tribunal. Overall, trying to understand why Professor Sawada had reached the conclusions he did was a difficult experience.

(xii) The high point of his contribution was his contention, based on diagrams figures 4, 5 and 6 in his report, that there was a mismatch between the

RERF data with respect rate of hair loss (epilation) of the survivors at Hiroshima and Nagasaki and the distances they were from the epicentre and the ABCC model of risk of harm. This was unpublished work based on a mathematical calculation of data originating from RERF. It was not a new piece of health research into the topic. The contention has not been peer-reviewed. We have not had the benefit of any comment on this work from a competent expert from RERF or ICRP or any similar body. Despite the assistance of an interpreter, there remained considerable uncertainty as to how he reached his calculations and what relevant factors he took into account in reaching them.

(xiii) It was entirely unclear how the hypothesis that some of the survivors had received health damaging radiation doses at greater distances from the epicentre than might have been previously believed married up with the detailed statistics on health outcomes for survivors. On one view, if there was greater exposure to radiation but the same recorded health outcomes, it would suggest that the dosimetry based on this data was too conservative.

(xiv) Professor Sawada concluded his report with a statement that because of the lack of safety of the ICRP model:

> 'Therefore in the case of the test veterans, the argument that their dose was low is not a valid argument because their internal contamination from fallout and rainout will have caused the health effects just as it did at Hiroshima and Nagasaki'.

In cross-examination it was explored on what basis he was possibly able to give evidence of causation of health effects to these veterans. Making every allowance for difficulties in translation we are satisfied that he had no scientific basis for that statement.

(xv) We recognise that Professor Sawada is an undoubted expert in particle physics. We are not satisfied this he has similar expertise with respect to the effects of radiation, although he undoubtedly has a strong personal interest in the effects of the bombs on Japanese survivors.

(xvi) The issue in this case is a possible causal link between health effects and service of the veterans and not with the workings of the Japanese

compensation scheme. His criticisms of the ICRP model are not within his area of scientific expertise. He is a signatory to the Lesvos Declaration stating that the ICRP risk model is unreliable and is a party to the ECRR campaign on that issue.

Professor Schmitz-Feuerhake

(xvii) Professor Schmitz-Feuerhake is an Emeritus Professor of Physics at the University of Bremen, Germany. She is now retired. One of her research interests is the health effects of ionising radiation. She is the current chair of ECRR. She, like Professor Sawada, provides the same arguments about the present ICRP risk model being wrong as it is based on external radiation exposures and uses the LSS data which is unreliable. She asserts that the problems with the LSS data are that the study only started in 1950, five years after the bombing and therefore excludes all those who had died in the interim as a result of their injuries. Therefore, the study groups were healthy survivors. In addition, the fallout contaminated areas of the city were quite far from the epicentre and this contamination remained for a long time.

(xviii) She rehearsed the ECRR objections to the ICRP model including (amongst others) the presence of clusters of childhood leukaemia near nuclear installations, the incidence of cancer in Sweden following Chernobyl, cancer and genotoxic effects following depleted uranium exposure and genetic and chromosome damage in the test veterans. She states that in some cases the differences between the outcomes predicted by ICRP and the observed effects are greater than 1000 fold and that a significant number of expert scientists and researchers in the area of radiation risk agree on this.

(xix) Many of her observations are based on papers by others and reviews of those papers, but on examination of the full article we find that important qualifications were lost. What had been, in some cases, issues for further inquiry became established propositions in ECRR-connected literature. She had no specialist expertise in epidemiology but was dismissive of criticisms of the statistical base and methodology of epidemiological studies she cited. She was also willing to comment on a medical issue as to whether CLL is radiogenic in nature although this again is outside the area of her expertise.

We are perplexed as to how a Professor of Physics was able to express strong opinions on epidemiology and radiobiology without any specialist expertise on these topics. She told us that her speciality was CLL and that she co-authored a paper challenging the previously accepted wisdom that CLL was not radiogenic that was influential in changing the view of the US government[69] but this was published in a journal of environmental health.

(xx) She also insisted that uranium has an affinity for DNA but could not comment on the US report put to her that uranium is readily secreted and acknowledged that this was not something which she had personally investigated and accepted that it was some years since she worked with uranium[70].

(xxi) We were surprised by her response 'yes but so what' to the proposition that the ECRR critique of the ICRP had been examined by ICRP, CERRIE and NRPB and found to have no scientific basis.[71] Her expanded answer suggests that all these bodies ignored the findings of leukaemia from Chernobyl and those living near nuclear installations.

(xxii) She did not initially accept that the ECRR studies she cites were not a proper basis to carry on such a study, and thought self-completion of questionnaires appropriate. She subsequently told us it was not ideal but better than nothing. She disagreed that it was better not to publish a flawed study because it will raise a debate[72].

Conclusions on the BS witnesses

230. We have explained in the ruling in Annex A to this determination, and the further discussion at Part Five, why we conclude that only experts understanding the requirements of the CPR protocol and meeting the common law tests for objective evidence should be received in this Chamber in determining appeals of the present class.

231. Mr Bramhall was not an expert of any sort, and we shall consider what he has to say about the CERRIE report below. The other witnesses were all scientists with expert

[69] Ionizing Radiation and CLL Environmental Health Perspectives vol 113 January 2005 SB 7/103.
[70] See transcript of evidence 15 June 2016 pp93 to 97
[71] 15 June 2016 p120.
[72] Ibid 126-148

qualifications, but all made it only too plain that they subscribed to the ECRR campaign; many were giving opinions outside their area of expertise and/or without reference to the views of recognised experts that were wholly inconsistent with the propositions they were advancing. We will consider the nature of the ECRR campaign further below.

232. In summary, for the reasons that we have given in this part of the determination, we found the contentious evidence of Professors Howard, Hooper, Sawada and Schmitz-Feuerhake to be lacking in scientific robustness and objectivity. We do not consider any of these witnesses in their reports and oral evidence on the issues in these appeals met the standards expected of experts giving evidence before a British court. Indeed, if a tribunal had received any of the reports tendered by these witnesses in isolation, unaware of the vast literature on the topic including the response of reputable experts to the criticisms made, it would have been utterly misled if any reliance had been placed on them.

233. If objection had been taken to their admissibility, in the case management stage, we consider it to be likely that we would have excluded some or all of them, essentially for the same reasons that Mr Justice Charles gave in respect to Dr Busby himself. Professor Schmitz-Feuerhake's view that a flawed piece of research was better than nothing because it provokes a debate is symptomatic of the ECRR approach to scientific issues (see [256] to [257] below). This is not the standard we expect of an expert assisting a tribunal on a matter of supreme importance to the individual veterans and the public at large. The determination of this appeal is not an exercise in polemics. We are not now concerned with whether such evidence should have been received at all, but whether the contentious opinions expressed amount to plausible evidence to be carried into the balance in the determination of the issues in this appeal. For the reasons given, we have no doubt that they do not.

234. We nevertheless have spent some time reviewing the considerable documentation cited in the reports and at the hearing to see whether there is a plausible hypothesis along the lines of the BS contention that we would need to take into account in the later stages of this determination.

The BS challenge to ICRP

235. As we have noted in Part Two, general radiation protection recommendations were proposed in the UK in the early 1920s and the First International Congress of Radiology was held in 1925. The International Commission on Radiological Protection (ICRP) was formed in 1950. ICRP recommendations have provided a system of radiological protection which is intended to cover all situations involving exposure to ionising radiation and which provides the range of dose that may be received. In particular:

(i) These include a component for hereditary effects.

(ii) Risk estimates for radiation-induced cancers are largely derived from studies of the effects of external radiation, the principal source being the long term survivors of the atomic explosions at Hiroshima and Nagasaki who were exposed to external photon and neutron radiation.

(iii) The cancer incidence and mortality data for A-bomb survivors show a statistically significant increase in solid cancers at doses from around 100 mSv up to around 3 Sv.

(iv) In order to calculate doses from radionuclides incorporated into the body, ICRP uses bio-kinetic and dosimetric models.

(v) The ICRP quantity, effective dose, takes account of the effectiveness of different radiations in causing cancer using radiation weighting factors. For example a weighting factor of twenty is used for alpha particles, compared with one for beta and gamma. Account is also taken of differences between organ/tissues in their contribution to total risk using tissue weighting factors. For example a weighting factor of 12 is used for the colon on the basis that colonic cancer contributes 12% of the total detriment from cancer and hereditary effects.

(vi) The major portion of the effective dose delivered to the general population results from radiation emitted by radionuclides located inside the body after internal contamination.

(vii) Many large populations of radiation workers are also exposed internally to alpha radiation emitted by radionuclides such as uranium or plutonium.

236. In the UK there have been challenges to the relevance of the ICRP radiation risk model since the 1980s following the identification of a cluster of childhood leukaemia cases in the village of Seascale near Sellafield. In response to these challenges, the Committee on Medical Aspects of Radiation in the Environment (COMARE) was established in November 1985. In reports from 1986 and 1996 COMARE concluded that the 'clusters' of leukaemia and non Hodgkins lymphoma in Seascale could not be related to Sellafield because the recorded doses were too low and therefore must be due to some other factor.

237. Many environmental groups and some scientists have not accepted the view that the occurrence of a leukaemia cluster is due to coincidence or some other factor (such as rural-urban mixing or infections) and in their view, the more straightforward explanation was that the ICRP risk models were incorrect and that a re-evaluation of these models was indicated. Another concern was the extent of the effects caused throughout Europe by radionuclides released in the reactor accident at Chernobyl in April 1986. The incidence of infant leukaemia and the presence of mini satellite mutations in the children of individuals irradiated as a result of the accident were cited as providing strong evidence of large underestimates of risks from internal emitters. Therefore, in July 2001, the Government requested COMARE to provide up to date advice on the risk estimates applied to radiation from internal radionuclides and the Committee Examining Radiation Risks of Internal Emitters (CERRIE) was born out of this.

238. The Minister of State for the Environment at the time, the late Michael Meacher MP, chose the membership of CERRIE which was designed to be widely representative and included members with a wide range of views including scientists associated with anti-nuclear groups, the nuclear industry and the NRPB. The chairman was Professor Dudley Goodhead who was the Director of the Medical Research Council Radiation and Genome Stability Unit. Dr Christopher Busby and Mr Richard Bramhall were both appointed to be members of CERRIE. Other members were Dr Roger Cox, Professor Sarah Darby, Dr Philip Day, Dr John Harrison, Dr Colin Muirhead, Mr Peter Roche, Professor Jack Simmons, Dr Richard Wakeford and Professor Eric Wright. There was a two-person secretariat: Dr Ian Fairlie and Paul Dorfman.

239. The Committee held 16 meetings during which it examined evidence from radiobiology and epidemiology and in June 2003 prepared a preliminary report that was considered at a workshop of invited delegates before its final report was sent to COMARE. Much of the Committee's work consisted of evaluating the available biological and epidemiological evidence of effects of exposure to radiation. In addition the Committee devoted considerable attention to the significant uncertainties in current models for radiation doses and risks as it was recognised that there were a range of views between the extremes. The Committee however devoted most effort to examining the evidence to ascertain whether there had been any underestimation of risks.

240. CERRIE published their final report in 2004. The Introduction contains a section 'Dissenting Views' that explains that two members of the group (Busby and Bramhall) argued that the dissonance between the Committee's views and their own was so great that attempting to express all views within a unified narrative would misrepresent their views. An attempt had been made to include the dissenting views in the main report but this was unsuccessful as the majority of members felt that the dissenting statement did not adequately explain the points of dissent, made factually inaccurate assertions and statements of a personal nature about third parties that were considered libellous. No dissenting statement was therefore included in the report. The dissenting members said they would not endorse the Committee's report and instead they went on to publish a separate document described as their own minority report (see [246] below).

241. CERRIE concluded that insufficient attention has been paid in the past to uncertainties in dose and risk estimates for internal emitters and that further work was required to quantify these uncertainties. On induced genomic instability, bystander effects, mini-satellite mutation induction and specific issues of micro-dosimetry there was general agreement that many of the phenomena were real and some may well be an integral part of cellular and tissue response but there was substantial disagreement as to whether the available data were sufficient to draw firm conclusions on the implications for radiation-induced health effects. CERRIE concluded that new findings on the biological effects of radiation should continue to be included in the consideration of health risks at low doses and their quantitative uncertainty. CERRIE also recommended further investigation of 'warm particles'

and the possibility of enhanced effects from radionuclide binding to DNA particularly in relation to strontium-90.

242. Mr Bramhall complains that the other members of CERRIE did not engage (seriously or at all) with the views of the ECRR. However, in our view, it is clear that the argument as to risk arising from evidence of clusters and such like was considered by CERRIE but the majority of the members were not persuaded of the scientific merit or validity of the ECRR approach to the issues and their 2003 formulation of an alternative risk calculation methodology .

243. With regards to epidemiological studies, CERRIE commented on the fact that the Committee had become aware of a few instances where errors had been made in epidemiological analyses carried out by governmental and non-governmental organisations and that these errors had not been discovered until after the findings were made public. The Committee therefore recommended that all epidemiological studies should employ rigorous scientific methods. It recognised that the peer-review process may tend to reject evidence that does not conform to existing paradigms but that where epidemiological studies are self-published authors had a responsibility to check their work.

244. COMARE was asked to comment on the CERRIE report and their findings are summarised as follows:

(i) The ICRP models were never intended and should not be used for any purposes other than the system of radiological protection of which they are a part.

(ii) The uncertainties involved are greater for internal emitters than for external radiation and must be tested. However, it should not be forgotten that uncertainties apply in two directions and that they can equally result in both an underestimation and an overestimation of risk. COMARE accepted that current uncertainties for internal emitters may be of the order of a factor of ten and they hoped that the ICRP would reassess this.

(iii) It acknowledged that CERRIE spent considerable time examining possible uncertainties concerning the dosimetric aspect of certain radionuclides (such as tritium and strontium) or those uncertainties that could be introduced by

the new paradigms such as the bystander effect and genomic instability. COMARE stated that they accepted the conclusions drawn in the CERRIE report on these matters and also accepted that considerable further work is needed to understand and adequately to quantify the risk of these uncertainties. However, COMARE noted that although these paradigms are new to the scientific community they have always been in biological operation and thus to some extent are considered in the currently available epidemiological data. Thus, the levels of these uncertainties in current risk estimates were to some extent incorporated in current models. They also noted that the biological variability of response between individuals to internal emitters is largely unknown, but could be comparable to the uncertainties in dosimetric calculations themselves.

(iv) On the subject of epidemiology, COMARE pointed out that considering individual or small epidemiological studies is not the best way to examine uncertainties. Wherever possible, large studies are required, particularly when examining effects at low levels of exposure. Although large and well-designed studies take a long time to carry out, they present the best way of estimating future risks.

245. The NRPB[73] was also asked to comment on the CERRIE report and their findings are summarised as follows:

(i) NRPB agreed with most of the CERRIE committee that low level radionuclides will lead to some increased risk of adverse health effects as a result of the internal irradiation of organs and tissues.

(ii) However, NRPB stated that the epidemiological evidence taken as a whole does not suggest that the predictions of current risk models are materially in error[74]. There was general agreement within CERRIE that new findings on the biological effects of radiation should continue to be included in considerations of health risks at low doses and their quantitative uncertainty but NRPB agreed with the majority committee view that there are no

[73] The Response of the NRPB to the report of CERRIE. Cox, Muirhead, Harrison 2005. (SB5 TAB 56)

[74] The response of the NRPB to the CERRIE report 2005 p 8

immediate implications for current estimates of doses and risks from internal emitters.

(iii) With regards to induced genomic instability and bystander signalling, NRPB found that the experimental data on these cellular responses was at an early stage of development and their relationships with cancer risk remained to be established. NRPB therefore agreed with the view expressed by CERRIE that knowledge on genomic instability and bystander signalling was at that stage insufficient to incorporate into a judgement relevant to radiological protection. CERRIE and NRPB shared the view that more research was needed into this and similarly, for the induction of mutations in mini-satellite DNA of germ cells, NRPB concluded that whilst these are of considerable scientific interest, the data considered by CERRIE did not imply that genetic risks to humans from internal or external radiation have been greatly underestimated.

(iv) With regards to the Second Event Theory (SET) of Dr.Busby, NRPB considered the theory in detail and was in full agreement with the majority view of CERRIE that 'the available studies to date offered little or no support to the SET as propounded by Dr Busby. Instead the available evidence substantially contradicted it'. Overall, in the view of the NRPB, SET had very little biological credibility and failed to provide coherent support to the thesis that cancer risks from certain internalised radionuclides and particulate forms of plutonium oxide had been greatly underestimated by ICRP and others.

(v) NRPB agreed with the general scientific consensus view that LNT is the most appropriate treatment of current data[75] and was not persuaded by the minority view that dose responses can be biphasic which in principle could lead to greater risks at low doses than those predicted by linear extrapolation procedures.

(vi) NRPB agreed with the CERRIE conclusion that ionising radiations from internal and external sources generate similar physical and chemical

[75] NRPB Response to CERRIE report 2005

interactions in living matter but did not agree with the minority view that man made radionuclides would tend to be more harmful than those occurring naturally.

(vii) With regards to epidemiology, NRPB noted that there are only a few epidemiological studies with individual estimates of exposures that provide relatively reliable and precise estimates of risks from internal emitters. Consequently risks from internal exposures have tended to be estimated, using estimates of doses to various organs together with estimates of the risk per unit dose from epidemiological studies of groups exposed to external radiation. The only studies that have been used are for lung cancer in miners exposed to radon gas and its short lived progeny; bone cancer in patients and workers exposed to radium and liver cancer in patients injected with Thorotrast. Risk estimates based on these groups, whilst subject to some uncertainty, are consistent with estimates from studies of external exposure or might even indicate smaller risks.

(viii) NRPB agreed with CERRIE that no firm conclusions can be drawn from the studies of infant leukaemia following the Chernobyl accident and that further results on this topic would be desirable in particular from an on-going pan-European study that is being co-ordinated by the International Agency for Research on Cancer.

(ix) NRPB did not agree with claims made by two members that the data in the Nordic study was flawed but did agree with CERRIE that overall the studies of childhood leukaemia and fallout from atmospheric nuclear weapons testing suggest an increased risk due to the exposure but provide no consistent or sufficiently persuasive evidence that this risk has been seriously underestimated by standard radiation risk models.

(x) CERRIE had noted the various studies conducted around nuclear installations in the UK to look at cancer near nuclear sites and near coastal and estuarine areas. These studies, some of which had been conducted and reported by Green Audit had previously been considered by COMARE. NRPB together with the majority of CERRIE agreed that the studies did not

support the hypothesis of a general increase in cancer rates in these areas. NRPB did not agree with the minority view that uncertainties in estimates of doses and risks from internal emitters are sufficient to allow the possibility that radionuclides discharged from the Sellafield nuclear fuel reprocessing plant are responsible for childhood leukaemias occurring in adjacent Seascale. This issue has been investigated exhaustively by COMARE. In all cases the relevant doses are dominated by contributions from natural sources including natural alpha emitting radionuclides.

(xi) NRPB agreed with CERRIE that further research needs to be done on workers at the Mayak plant in the Southern Urals in Russia as well as other groups in the former Soviet Union such as people who were living near the Techa River. NRPB agreed with the majority view that data on trends in infant mortality do not support an association with fallout from weapons testing.

(xii) With regards to ICRP methodology and uncertainties, there was agreement within CERRIE that uncertainties in bio-kinetic and dosimetric models used to calculate organ and tissue doses from internal emitters are inherently greater for short range emissions from low energy beta and alpha particle emitters than for penetrating photon emissions because their effect will depend on their location in tissues relative to target cells. NRPB agreed with the conclusions that doses and risks from internal emitters should be calculated on the basis of best current information.

The CERRIE Minority Report 2004

246. Although this is described as the Minority Report of CERRIE, it transpired at the hearing that it was not a minority report approved and published by CERRIE[76] but instead was a report written by the minority of members (Bramhall, Busby and Dorfman). It was published by Sosiumi press which is controlled by Richard Bramhall[77] and the extracts the Tribunal were given contain an eclectic collection of documents. These include a letter of resignation from CERRIE and a letter to her MP from Ms Marion Hill; a statement from Paul Dorfman voicing concerns over the way

[76] TS day 1 p132
[77] TS day 1 p134

in which CERRIE may not have adhered to its remit; an article published in the Sunday Times in 2004 titled 'Government gags experts over nuclear plant risks' and a series of reports which have been translated from Russian on the effects of Chernobyl (but containing just a headline summary) and an appendix written by Chris Busby entitled 'Atomic lies'. The contents have every appearance of a political campaign rather than sober scientific analysis.

The European Committee on Radiation Risks (ECRR)

247. The ECRR itself is a campaigning group, the precise membership of which is difficult to ascertain but many of its supporters (Sawada, Schmitz-Feuerhake, Mothersill, Busby) have played a role in these appeals. We were told at the hearing by Richard Bramhall that ECRR has 'no members, not in a formal sense'. The ECRR is described in its 2010 Recommendations report as:

> 'a spontaneous creation of Civil Society which was faced with clear and alarming evidence of the failure of its democratic institutions to protect it from the effects of radioactive pollution. Predictably, the engine which generated this development was the Green movement, the result of another and earlier Civil Society reassessment of the aims and ideologies behind the systematic exploitation of the planet.'

248. The ECRR published a new risk model in 2003 and this was updated in 2010 to include the effects of uranium. In May 2009, ECRR held an international conference in Lesvos, Greece, at which there was discussion of the 2003 risk model, along with incorporation of the phenomenon of photoelectron enhancement by elements of high atomic number and the effects of uranium exposure. This led to a concluding statement called the Lesvos Declaration which was signed by 16 attendees, calling for the urgent abandonment of the ICRP risk model by governments.

249. The ECRR arguments are as follows:

    (i) The ICRP risk model is inadequate for the estimation of risks from internal radiation exposure to radionuclides and may lead to a significant underestimate for some radionuclides. This proposition is predicated on observations of clusters of leukaemia around nuclear processing plant, the incidence of infant leukaemia and the presence of mini satellite mutations in the children of individual irradiated as a result of the 1986 Chernobyl accident.

(ii) The ECRR claim that the ICRP risk model is wrong because it is derived from studies of the effects of high doses of external radiation and may not be directly applicable to situations where there has been exposure at low doses of internal emitters.

(iii) The ECRR has developed its own risk co-efficients which differ from the ICRP ones by up to 1000. These have not been accepted by any of the advisory bodies including the ICRP which the ECRR argue is because there is a heavy bias in favour of those with a vested interest in the continuation of the nuclear industry and the scientific evidence that ECRR bring to the table is not considered with the due regard that it deserves.

(iv) The LSS results are flawed because of the way the study is conducted, i.e. the 'not in city' group were removed in 1973, and the previous dosages calculated may be in error, as Professor Sawada has demonstrated that there was evidence of epilation and diarrhoea (i.e. deterministic effects) in the groups who were as far as 6km away from the hypocentre.

(v) The ICRP model is not applicable to uranium.

250. ECRR heavily criticise Richard Wakeford (Chief Scientist for British Nuclear Fuels) and Editor of the Journal of Radiological Protection and suggest that by reason of this position he is biased towards those who believe in the ICRP system. They apparently fail to see that support for the aims of a campaign critical of civilian and military use of radioactivity might be said to predispose its own members to a partial view when giving evidence on the topic. Scientists may have personal views, passionately held, on controversial issues and are perfectly entitled to become part of a campaigning organisation to raise public awareness of controversial issues. We accept that activities that generate radiation are certainly controversial. In our view, however, when giving evidence to a court or tribunal, scientific objectivity requires scientists to put aside both personal predilections and occupational connections, and address all the available data critically, dispassionately and reasonably. As we have explained elsewhere in this determination, the standards demanded of an expert giving evidence before a court or tribunal are designed to be demanding and to ensure that the court is given a fair and balanced assessment of the issues beyond its

own expertise. Appropriate disclosure as to any connection that may affect the evaluation of the evidence must be made.

251. During the hearing we were taken to an editorial which was written by Richard Wakeford about the way in which CERRIE operated[78]. Wakeford was a member of CERRIE and apparently attended 16 meetings, 4 sub-committee meetings, a 3 day workshop, and a press conference. Despite having some reservations about being part of this group he thought that the chair (Dudley Goodhead) was a well-respected scientist with strongly independent views. However he goes on to say that:

> 'I felt that the first meeting of the Working Group had confirmed my suspicions that we had been brought together largely to consider (and presumably endorse) the views of Chris Busby….'

252. Wakeford is deeply critical of Dr Busby's work, writing that it is self-published and difficult to access and that he seems mainly to avoid publication in the recognised scientific literature which presents difficulties for a proper review of the evidence underlying his conclusions. He felt that it was somewhat ironic that the CERRIE process permitted the Committee members to conduct a detailed and critical review of the work of Dr Busby much to its detriment and that the process revealed the deeply flawed nature of many of his studies that rendered their results effectively meaningless.

> 'Chris Busby is apparently quite prepared to self-publish reports containing glaring errors in data and/or analyses. The Committee should not be criticised for exposing the serious failings of studies supposedly supporting one particular extreme position.'

Mr Wakeford is not alone in his concerns. Dr Lindahl, a Swedish scientist who became head of Cancer Research UK, made an equally critical comment on the work of Dr Busby when asked to comment on this work in about 2008[79], describing him as a self-taught individual without formal training in radiobiology, who is outspoken and opinionated and has a talent for court proceedings, noting:

> 'In my opinion he does not have the professional knowledge in epidemiology that would be required to take issue with the conclusions of the best and most famous epidemiology department in Britain, that is the one at the University of Oxford.

[78] Reflections on CERRIE- Editorial in the Journal of Radiological Protection by Richard Wakeford (handed out at the hearing also B8/38

[79] SB12/17

> Moreover, I am not convinced by his polemical style describing professional experts with different opinions to his own e.g…. "it is only the biased scientists of the nuclear military project and the economic and military vested interests that continue to support the conventional model". This is nonsense. I was invited to serve on the international committee to issue BEIR VII and I know that the members represented a wide range of academic backgrounds in radiology, genetics and biochemistry, with each member having a much better academic background in radiation research than Dr Busby and nevertheless largely supporting current conventional models.'

253. The language of the ECRR report is that of campaigning zeal rather than scientific objectivity. For example, we note one statement that:

> 'Polanyi's comparisons with Azande witch doctors are familiar territory to those who have registered the sequences of denials and implausible explanations which have followed discovery of the Sellafield (Seascale) child leukaemia cluster and many other examples of the failure of the ICRP risk models.'

254. Our own analysis of the ECRR material that forms the background to the papers cited by the BS expert witnesses, confirms that there is a lack of scientific rigour about the way conclusions are reached and propounded. We give the following examples:

(i) ECRR cite a number of examples in the 2010 report which they state show between a 600-1000 fold error in the ICRP model. However, none of these observations are evidenced in the report, so it is very difficult to see where the figures have come from.

(ii) ECRR state that they consider the results of studies published in the peer-review literature but also reports, books and articles which have not been submitted for peer review. More alarming from a scientific point of view they happily include reports which started life as television documentaries and ended as court cases.

(iii) There are no scientific references in the ECRR report *per se* but if we look at the assertions of the same group of scientists from whom we heard evidence from and who are signatories to the Lesvos declaration there are some common themes. Much of the epidemiological work quoted by this group is not regarded as 'evidence-based' by the majority of scientists.

255. The criticisms that are made of epidemiology that the ECRR quotes and relies on can be summarised as follows:

a) Sample size: Many of the studies quoted by this group used a very small sample size which increases the risk that it will be difficult to determine whether the findings are due to chance or a real effect. For example, Dr Haylock was asked about the Hristova paper at the hearing and[80] commented that it was a poor comparison because the conclusions are from a group that only consists of six people. Professor Thomas was asked about a number of papers at the hearing provided by Dr Busby and we heard the same response: that the sample sizes were too small to be of any use.[81]

b) Confidence intervals (CI): Confidence intervals provide information about the range of possible effect sizes. Very large confidence intervals are to be regarded with some suspicion, particularly if the confidence interval contains the value where there is no difference between the two groups. Several studies fell foul of this principle. For example in the Cardis study 2005[82] Dr Haylock pointed out that the confidence interval has a lower boundary which is negative, implying that the data does not support the hypothesis that the excess risk is statistically different from no risk[83].

c) Statistical bias: This was introduced by poor selection criteria and inappropriate use of questionnaires. This was evidenced in the much quoted 1998 study by Dr Rabbit Roff about congenital abnormalities in the children and grand-children of BNTVA members based on the responses to a questionnaire survey. Having regard to the exacting standards to be required of epidemiology, we accept the criticism that this type of survey questionnaire used to provide data often leads to poor quality and biased results especially when using a highly self-selected subset. We, therefore, find that the data from this survey are highly unlikely to provide unbiased measures of risk. In addition the sample size is small: the survey was sent to 2,087 members of BNTVA of which only 1,041 responded. Considering that the total number of service personnel involved was over 20,000 this is a tiny proportion and is much more likely to be completed by those who have an interest in the issue being enquired about.

[80] TS day 9 p18
[81] TS day 5 p38-43
[82] SB 6/68
[83] TS day 9 p29

d) Ecological studies: Studies of populations living around nuclear facilities and of other environmentally exposed populations do not contain individual estimates of radiation dose or provide a direct quantitative estimate of risk in relation to dose. This limits the interpretation of such data. Several cohort studies have reported health outcomes among persons exposed to environmental radiation. No consistent or generalisable information is contained in these studies and some results are inconsistent. The most informative findings are from studies of individuals exposed to radiation after the Chernobyl accident. Recent evidence indicates that exposure to radiation from Chernobyl is associated with an increased risk of thyroid cancer and that the relationship is dose dependent. The quantitative estimate of excess thyroid cancer risk is generally consistent with estimates from other radiation-exposed populations and is observed in both males and females. Iodine deficiency appears to be an important modifier of risk, enhancing the risk of thyroid cancer following radiation exposure[84].

e) Statistical significance: Even if the p-value is low, this does not necessarily mean that the effect is real. By chance alone 1: 20 significant findings will be spurious[85].

> 'P values unless you know the sample size can be very misleading…when you look at the number of people you study you realise that it is such a small population that it is highly suspect as a genuine p value that represents the population'[86].

In addition, something that is statistically significant may not be clinically important.

256. There is an interesting narrative in the ECRR report on the difference between types 1 and 2 errors which explains much of their *modus operandi*. A Type 1 error is defined as when the effect seems statistically significant but the difference has arisen by chance; a Type 2 error when the results are ignored on the grounds that the effect is non-significant when in fact the effect may well be real.

257. The authors acknowledge that the Committee made two decisions:

[84] BEIR VII Executive Summary
[85] TS day 9 Dr Haylock p29
[86] Prof Thomas day 5 p41

> 'The first was to take a precautionary approach and avoid making a type 2 error in such an area of low-probability high-impact risk, for if the evidence showing excess risk from the exposure were in fact a chance finding the mistaken inclusion of it as evidence of radiation-induced effects would not harm the human race. If on the other hand, the Committee were to take the opposite view and exclude it as evidence when in fact it was in fact a true measure of a real effect but merely formally non-significant then much harm would follow its dismissal. Consequently the second decision to use a Bayesian approach to the refinement of belief in the area of risk assessment and allow each non-significant observation (including unpublished results) to weight and modify the overall probability of belief in the area of radiation risk according to their degree of significance.'[87]

258. We agree with the criticism that ECRR makes highly selective use and interpretation of studies that support their position. There was a lack of scientific robustness in the papers we were taken to. The examples of this are too numerous to recite all of them. A number of times during the hearing we were taken to abstracts of papers which in fact were citing findings that were not substantiated in the body of the paper. One such example arose when we were taken to the paper by Araneta et al (Prevalence of birth defects among infants of gulf war veterans in Arkansas, Arizona, California, Georgia, Hawaii and Iowa 1989-1993 (2002)) in an attempt to show us the genotoxic effects of uranium, because infants conceived post war to male Gulf War Veterans had significantly higher prevalence of congenital heart or renal defects. However, on closer reading the authors of the paper conclude that evaluating the relationship between war time environmental exposures and teratogenesis is complex because of the methodological challenges including limited statistical power for rare events and finally state that:

> 'we did not have the ability to determine if the excess was caused by inherited, environmental or synergistic factors or was due to chance.'

259. When Dr Haylock was asked about the Feuerhake/Busby/Pflugbeil paper on genetic radiation risks he told us that:

> 'it appears to be a review but it's not a review in the sense that I would have done it where you look at the studies and give a dare I say try to give a balanced view as to the plus points and the negative points…Dr Busby seems to have picked out points from the studies which support his argument but don't seem to take into account any of the issues with these studies as to whether those points are valid or not.'[88]

[87] ECRR 2010 p77
[88] Dr Haylock TS day 9 p41

We agree and conclude that it is indicative of how ECRR operate generally to reach and disseminate many of its conclusions. This is supported by the evidence of Professor Schmitz-Feuerhake to which we have already referred.

260. We next turn to the particular issue of the impact of uranium that the BS appellants advance.

The BS submissions relating to uranium

261. The submissions from the BS appellants are that:

(i) Uranium was the main component by mass of all the bombs used in the UK tests. They assert that uranium was the main component by mass of the Hiroshima and Nagasaki bombs and that uranium contamination resulted from rainout from what has been termed 'black rain'.

(ii) All uranium isotopes have a strong affinity for DNA and uranium is an effective absorber for natural background radiation. The results of cell-culture and animal studies show that uranium carries an unusually high degree of hazard out of all proportion to its radioactivity as modelled by the ICRP/BEIR model approach.

(iii) Uranyl ions bind to DNA, therefore, this is evidence that uranium binds to DNA. This is evidenced by the 1961 Huxley and Zubay paper which states that uranyl acetate can combine with nucleic acids.

(iv) The hazard from contamination of the test sites with uranium was raised in 1953 by the US expert Dr Karl Morgan in a meeting at Harwell. Morgan pointed out that U-234 was a radiological rather than a toxic hazard.

(v) The presence of uranium isotopes at Maralinga or CI could not have been measured by the monitoring equipment used at the time.

(vi) The secondary photoelectron effect is the ability of high atomic number elements in the body (gold, platinum, uranium) to absorb radiation more effectively than living tissue and then release this energy into tissue as local photoelectrons which means that the radiation dose near such particles would be very high. This effect has not been incorporated into the ICRP risk modelling. The BS submission states that the only two computer studies that

have been carried out by 'the radiation establishment' have conceded that there is a finite but modest enhancement of dose near such particles but it is contended that the methodology used is flawed.

(vii) There is evidence from environmental and other studies that uranium exposed populations have an increased incidence of chromosomal damage and birth defects.

General science about uranium

262. A helpful review of the nature of uranium and the risks to health it may pose is found in a document prepared by an agency of the US Department of Health, the Agency for Toxic Substances and Disease Registry. It is entitled Uranium Toxicity and was published in May 2009[89]. It is common ground that uranium is a naturally occurring radioactive element and is commonly found in plants, rocks and soil. Natural uranium is a mixture of three isotopes: by mass U-238 is the most abundant, while U-235 and U-234 represent tiny amounts[90]. All three isotopes behave the same chemically but are very different radioactive materials with different radioactive properties. U-234 has the shortest half-life (2.5×10^5 years) and is, therefore, the most radioactive followed by U-235 and U-238 (4.5×10^9 years)[91].

263. Enriched uranium has more U-235 (and also U-234, the most radioactive of the three uranium isotopes); depleted uranium has less U-235 and is less radioactive than natural uranium. Enriched uranium is used in reactors and weapons. Depleted uranium can be used as shielding materials and is used by the armed forces. It can be a source of exposure to alpha radiation when embedded in soft tissues.

264. Uranium is a heavy metal that forms compounds of different varieties and solubility.

265. Uranyl acetate and uranyl nitrate are water soluble compounds which are used commonly as stains in electron microscopy.

266. Food and drinking water are the main sources of exposure to natural uranium isotopes among the general population[92]. The occupations most heavily exposed to

[89] SB 4/12
[90] Uranium Toxicity p7/51.
[91] Professor Thomas acknowledged by reference to this document that her initial answers in cross-examination that natural or stable uranium was not radioactive at all were wrong and a silly mistake.
[92] The most common source of exposure for members of the public is through eating or drinking material that contains uranium: UT p14/51

uranium are those employed in mining or milling operations or in uranium enrichment and processing activities. Many large populations of radiation workers are also exposed to alpha radiation emitted by radionuclides such as uranium or plutonium.

267. Inhaled uranium becomes deposited in the various portions of the respiratory tract and the lungs. The place of deposition is based on particle size (i.e. the larger particles deposited higher in the respiratory tract). Most of the deposited uranium clears rapidly via mucociliary transport to the throat. Once there the uranium is cleared via sputum or swallowing and primarily faecal excretion. Soluble uranium dissolves and is absorbed into the circulatory system more rapidly than insoluble forms.[93]

268. Once absorbed, uranium is deposited throughout the body and the highest levels are found in the bones and kidneys. Overall, most ingested uranium is excreted in faeces (95%) with the remainder eliminated in urine. Many animal studies have reported damage caused by acute exposure to uranium to the kidney, which is considered to be the main organ targeted for uranium toxic effects under such exposure conditions. Experimental studies on the effects of chronic exposure to uranium have reported mixed findings with some reporting nephrotoxicity and others not.

269. Current estimates of risk associated with internal exposure to alpha (α) radiation are derived from the risk models based on the LSS data by applying a radiation weighting factor of 20 to the absorbed dose. However it has been acknowledged that the health effects of chronic exposure to uranium as it occurs in populations of workers involved in the nuclear fuel industry and in the general population are not well known and concerns have been raised about the reliability and accuracy of the conversion of risk.

CURE

270. In 2009 the EURATOM-convened HLEG (High Level Expert Group) considered the limited knowledge about the effects of internal contamination by radionuclides to be a key scientific issue for the purpose of radiation protection policy. Subsequently the effects of internal contamination have been considered as a cross-cutting issue by European groups in the field of radiation research: MELODI, EURADOS, NERIS

[93] UT p15/51 What is the biologic fate of uranium in the body?

and ALLIANCE. The DoReMi network of excellence supported by the European Commission also identified gaps in the knowledge of the effects of internal contamination as key issues for radiation protection research.

271. The CURE (Concerted Uranium Research in Europe) project was an 18-month concerted action, funded and coordinated by the Institut de Radioprotection et de Sûreté Nucléaire (IRSN) and was integrated as task 5.8 of DoReMi. Its aim was to elaborate a collaborative research project on the biological and health effects of uranium contamination, integrating epidemiology, biology/toxicology and dosimetry. In total nine partners from six countries (France, UK, Germany, Belgium, Czech Republic, Spain) participated in the CURE project. It reported in March 2015[94] recommending a model for further research into occupational exposures.

272. It reviewed in the Background section of the report what is already known and reported: that uranium has been found to be genotoxic in both *in vivo* and *in vitro* studies even in its depleted form which generates little alpha radiation. Experimental studies on natural or depleted uranium have reported other significant biological effects such as renal toxicity, lung damage and neuro-physiological perturbations. This suggests that uranium, as a heavy metal, may cause harm by chemical toxicity aside from any radiation effects. It concluded however that there are considerable uncertainties in the extrapolation of the biological effects at low doses observed in animals to possible disease risks in humans[95].

273. In order to look at uranium effects from epidemiological studies, cohorts of uranium miners and other workers were identified as populations of priority interest. Studies conducted in non-occupational setting (i.e. populations living around uranium processing sites) were excluded as they mostly employed ecological designs and were therefore considered to be minimally informative. They concluded that doses from alpha radiation to the lung from uranium or other radionuclides present in uranium ore are negligible when compared to that of radon exposure and that individual epidemiological studies concluded so far do not provide reliable evidence on the potential health effects associated with uranium exposure.

[94] SB7/112
[95] P6-7

274. CURE looked at several studies which investigated the biological effects of exposure to uranium in human populations which included exposure to depleted uranium in Gulf war veterans, those populations exposed to drinking water naturally contaminated with uranium, uranium miners and uranium millers. The conclusions were that molecular epidemiology studies conducted in uranium workers and published to date have focused only on a limited number of biomarkers and generally lacked proper organ dosimetry. Most of these studies have been cross sectional and have not allowed for a detailed assessment of the long term effects of protracted exposure to uranium. Based on the results of this concerted action, a large scale integrated collaborative project was proposed to improve the characterisation of the biological and health effects associated with uranium internal contamination in Europe.

Genetic effect of uranium

275. The BS contention is that uranium radiation has a particularly harmful effect on human DNA and causes genetic damage.

276. In evaluating the BS contention with respect to uranium we found that the BEIR VII Public Summary[96] to be helpful in explaining the current state of knowledge. The topic is also addressed in Part Six where we consider Professor Mothersill's evidence.

277. Naturally occurring genetic (*i.e.*, hereditary) diseases contribute substantially to illness and death in human populations. These diseases arise as a result of alterations (mutations) occurring in the genetic material (DNA) contained in the germ cells (sperm and ova) and are heritable (*i.e.*, can be transmitted to offspring and subsequent generations). Among the diseases are those that show simple predictable patterns of inheritance (which are rare), such as cystic fibrosis, and those with complex patterns (which are common), such as diabetes mellitus. Diseases in the latter group originate from interactions among multiple genetic and environmental factors.

278. Early in the twentieth century, it was demonstrated that ionising radiation could induce mutations in the germ cells of fruit flies. These findings were subsequently

[96] SB 17/2

extended to a number of other organisms including mice, establishing the fact that radiation is a mutagen (an agent that can cause mutations in body cells); human beings are unlikely to be exceptions. Thus began the concern that exposure of human populations to ionising radiation would cause an increase in the frequency of genetic diseases. This concern moved to centre stage in the aftermath of the detonation of atomic weapons over Hiroshima and Nagasaki in World War II. Extensive research programmes to examine the adverse genetic effects of radiation in the children of A-bomb survivors were soon launched. Other studies focusing on mammals that could be bred in the laboratory—primarily mice—were also initiated in different research centres around the world.

279. The aim of the early human genetic studies carried out in Japan was to obtain a *direct* measure of adverse effects in the children of A-bomb survivors. The indicators that were used included adverse pregnancy outcomes (*i.e.*, stillbirths, early neonatal deaths, congenital abnormalities); deaths among live-born infants over a follow-up period of about 26 years; growth and development of the children; chromosomal abnormalities; and specific types of mutations. Specific genetic diseases were *not* used as indicators of risk, because not enough was known about them when the studies began.

280. As in previous BEIR reports, a method termed the 'doubling dose method', is used to predict the risk of inducible genetic diseases in the children of people exposed to radiation using naturally occurring genetic diseases as a framework. The doubling dose (DD) is defined as the amount of radiation that is required to produce as many mutations as those occurring spontaneously in *one generation*. The DD is expressed as a ratio of *mutation rates*. It is calculated as a ratio of the average spontaneous and induced mutation rates in a set of genes. A large DD indicates small relative mutation risk, and a small DD indicates a large relative mutation risk. The DD used in the present report is 1 Sv (1 Gy) and derives from human data on spontaneous mutation rates of disease-causing genes and mouse data on induced mutation rates. Therefore, if three mutations occur spontaneously in 1 million people in one generation, six mutations will occur per generation if 1 million people are each exposed to 1 Sv of ionising radiation, and three of these six mutations would be attributed to the radiation exposure.

281. More than four decades have elapsed since the genetic studies in Japan were initiated. In 1990, the final results of those studies were published. They show (as earlier reports published from time to time over the intervening years showed) that there are no statistically significant adverse effects detectable in the children of exposed survivors, indicating that at the relatively low doses sustained by survivors (of the order of about 400 mSv or less), the genetic risks, as measured by the indicators mentioned earlier, are very low. Other, mostly small-scale studies of the children of those exposed to high doses of radiation for radiotherapy of cancers have also shown no detectable increases in the frequencies of genetic diseases.

282. During the past 10 years, major advances have occurred in our understanding of the molecular nature and mechanisms underlying naturally occurring genetic diseases and radiation-induced mutations in experimental organisms including the mouse. These advances have shed light on the relationships between spontaneous mutations and naturally occurring genetic diseases and have provided a firmer scientific basis for inferences on the relationships between induced mutations and diseases. The risk estimates presented in this report have incorporated all of these advances. They show that at low or chronic doses of low-LET irradiation, the genetic risks are very small compared to the baseline frequencies of genetic diseases in the population. Additionally, they are consistent with the lack of significant adverse effects in the Japanese studies based on about 30,000 children of exposed survivors. In other words, given the BEIR VII estimates, one would not expect to see an excess of adverse hereditary effects in a sample of about 30,000 children (the number of children evaluated in Hiroshima and Nagasaki). One reason that genetic risks are low is that only those genetic changes compatible with embryonic development and viability will be recovered in live births[97].

283. We also find the 2010 HPA[98] document to be a helpful review and its conclusions compelling in assessing this issue. In our view the fact the HPA is required to advise government and other stakeholders on the application of ICRP recommendations in the UK does not make it biased, but merely notes the pre-eminence of the ICRP methodology in the legal framework of the UK and the EU. We have no doubt that if

[97] BEIR VII Public Summary
[98] Mobbs, Muirhead and Harrison 'Risks from Ionising Radiation' HPA 2010 SB/22/1

there were good scientific reasons to conclude that applying the ICRP model might expose a section of the British public to risk, the HPA would have said so.

284. We have noted that follow up studies of the A-bomb survivors have consistently provided the best source of information on radiation induced cancers and other health effects after exposures to around 100 mSv and greater. The third analysis of the UK National Registry for radiation workers which examines cancer risks in a very large cohort of workers exposed to low doses of radiation over many years show a clear dose-response relationship consistent with the extrapolation of A-bomb risk factors to low doses. Confidence is enhanced when two large scale and long term surveys reach similar conclusions.

285. The best direct evidence of risk from internal emitters comes from studies of lung cancer following exposure to radon in mines and homes, bone cancer in radium exposed patients and workers and liver cancer and leukaemia in patients given injections of thorotrast (thorium oxide particles). The risk estimates from these studies are consistent with those from the A-bomb survivor studies when account is taken of the greater effectiveness of alpha particles in causing cancer (by factors of up to 20).

286. Considerable efforts are being devoted to the studies of the health effects from internal and external exposures at the Russian Mayak plant and the associated discharges to the Techa river. Several hundred thousand people were involved in recovery work and there are indications of an increase in leukaemia and cataracts among those most highly exposed. There has been a clear and substantial increase in thyroid cancer in persons exposed as children or adolescents.

287. The HPA response to the ECRR states:

> 'a critical examination of the ECRR report has been undertaken by the NRPB staff. The cited epidemiological studies have been investigated in detail by the NRPB staff and previously by other experts; their conclusions are generally different from those reached by ECRR. The methodology proposed by ECRR for estimating radiation risks from internal emitters is arbitrary and does not have a sound scientific basis. Furthermore there are many misrepresentations of ICRP, misunderstandings, inconsistencies and unsubstantiated claims in the ECRR report. The ECRR report therefore provides no scientific basis for changing protection standards. Overall NRPB believes that the recommendations of ICRP provide a sound basis for radiological protection standards. In particular risks from internal emitters are acceptably well understood and may, in some cases be overestimated by ICRP.'

288. The HPA in agreement with the French IRSN was fully supportive of the need for further research to understand radiation risks at low doses, including risks from internal emitters and cited the emerging findings on non-targeted effects of radiation, including genomic instability and bystander effects.

289. Internationally respected organisations such as UNSCEAR and BEIR have reviewed all the materials and arguments put forward by ECRR including arguments that low doses of radiation are more harmful than a LNT model of effects would suggest, but have consistently concluded that radiation health effects research, taken as a whole, does not support this view and the data currently available does not require changes in radiation risk co-efficients for cancer and hereditary effects of radiation in humans.

290. Professor Thomas was questioned at some length by Dr Busby on these issues. She is a Professor of Molecular Pathology at Imperial College London and Director of the West London Genomic Medicine Centre. She has a specialist interest in the molecular biology of radiation induced thyroid cancer, and has studied the pathology of thyroid cancers following the Chernobyl accident and has reviewed the issue with respect to Fukushima accident in Japan. Her evidence was commissioned and adduced by the Secretary of State to assess whether the medical conditions claimed by the veterans were or could be radiogenic and her evidence on that issue is summarised in the next part of this determination.

291. Her expertise, however, meant that she was able to assist in responding to the BS criticisms of ICRP that were put to her. We note the following[99]:

    (i) Dose is absolutely critical. You cannot assign a cause and effect unless you know the dose.

    (ii) Assessing the health impact of a dose depends on the nature of the ionising radiation, the organ, its half-life, and the organ affected. These are reflected in the weighting factors used by the ICRP.

    (iii) She told Dr Busby that natural uranium was not radioactive but in re-examination she recognised that this was an error and a silly mistake

[99] Transcript 16 and 17 June

(iv) If uranium is ingested into the body, most will come out in the mucociliary pathway or be excreted, but a small amount may enter the gut and be absorbed by the blood if it is soluble. Most of this will in turn be excreted by the kidney. Uranium oxide is not soluble. According to the data obtained from uranium miners the small amount that may enter the gut may be from 5% to 0.5% and of this 75% goes out in urine in the next 24 hours.

(v) Data on effects of uranium in animals cannot be transferred to humans as the metabolism is very different. Animal models have been given up in cancer research as they do not replicate the human situation.

(vi) There was an increase in thyroid cancer after Chernobyl resulting from radioactive iodine. This was particularly the case with those who were children or *in utero* at the time of exposure. The dose range varied from several gray down to 50 milligrays, with a mean dose of evacuees of 500 milligrays but the range of dose was enormous.

(vii) She was not an expert on dosimetry but the population exposed and the assessment of dose was accurately examined by UNSCEAR 2008.

(viii) In the responses to both the Chernobyl and Fukushima accidents, screening programmes were put in place that could distort the estimation of cause and effect. The sophisticated screening regime may pick up pre-existing health effects that would otherwise not have been detected.

(ix) One published paper reporting increased thyroid cancer following Fukushima where the mean dose (4.2 milligrays) was much lower than Chernobyl was flawed in part because it failed to account for this effect. Its methodology has been criticised by experts in the field for this and similar reasons.

(x) She considered that the prospect of natural uranium in a bomb detonation resulting in genetic damage through heavy metal effects as postulated by the BS experts to be vanishingly small and unlikely. The difference between an assay *in vivo* and *in utero* was critical. The amount of dose that would reach the cells is vanishingly small. Everybody is exposed to natural uranium. In the US it is assessed that the daily human intake is 1.5 micrograms of uranium per day.

(xi) A paper by Canu and others entitled 'Cancer Risks in Nuclear Workers', where the abstract was relied on by Dr Busby to show evidence of increased risk of cancer in workers occupationally exposed to uranium[100], in fact showed the opposite. The authors referred to cohorts where 'risk increased non-significantly' and if the effect was not significant it was irrelevant. In cohorts where significant excess existed the details of the paper showed the workers had been exposed to high levels of dust. Further, the effect of other chemicals and tobacco could not be assessed.

(xii) Although not an expert in epidemiology she made criticisms of studies based either on small numbers and flawed methodology or bias in self-selected response to questionnaires. You have to know that the method you are choosing is appropriate for the question you seek to answer and a badly designed study will skew the information provided to others.

(xiii) Amongst the critical observations she made on the Wahab/Rowland paper were sample size, and the proposition that any conclusion could be drawn about the source of any chromosome aberration after 50 years, when most circulating cells in the body will have been lost and many other sources of exposure could have made a contribution.

(xiv) While uranium is recognised to be genotoxic there is no evidence that it causes cancers in man.

Conclusions on the BS challenge to ICRP

292. Despite the concerns raised by environmental groups and ECRR for more than 30 years, the general conclusion of ICRP, UNSCEAR, BEIR, IRSN and HPA is, and remains, that the higher the dose, the greater is the risk; conversely the lower the dose, the lower is the likelihood of harm to human health and this is for a number of reasons:

(i) Any single track of ionising radiation has the potential to cause cellular damage. However, if only one ionising particle passes through a cell's DNA, the chances of damage to the cell's DNA are proportionately lower than if there are 10, 100, or 1000 such ionising particles passing through it. There is

[100] SB22/9

no reason to expect a greater effect at lower doses from the physical interaction of the radiation with the cell's DNA.

(ii) The so called 'new' evidence from biology suggests that cells do not necessarily have to be hit directly by a radiation track for the cell to be affected. Some speculate that hit cells communicate with non-hit cells by chemical signals or other means. To some, this suggests that at very low radiation doses, where all of the cells in the body are not hit, 'bystander' cells may be adversely affected, resulting in a greater health effect at low doses than would be predicted by extrapolating the observed response at high doses. Others believe that increased cell death caused by so-called bystander effects might lower the risk of cancer by eliminating cells at risk for cancer from the irradiated cell population. Although additional research on this subject is needed, it is unclear at this time whether the bystander effect would have a net positive or net negative effect on the health of an irradiated person.

293. The BS submission suggests that the toxicity of uranium may have been substantially underestimated because as a high Z element it may convert natural background gamma rays into short range photoelectrons.

294. This secondary photoelectric effect is a well-known phenomenon where photons passing through material lose energy by exciting atomic electrons, leading to the emission of a photoelectron followed by a cascade of Auger and Coster-Kronig electrons and fluorescence. The HPA considered this and noted that peer-reviewed studies looking at this have concluded that the enhancement is of negligible biological significance compared to the intrinsic activity of the uranium.

295. Similar considerations apply to the suggestion that soluble forms of uranium might concentrate within the cells, bind to DNA and enhance the effect of natural background photon radiation. The HPA conclude that the extent of direct association with DNA will be important only for consideration of energy deposition from very short range emissions. However, calculations demonstrate that the effect will be of less biological significance for uranium because the higher Z element produces relatively longer range secondary radiation.

296. There is no evidence from animal experiments of unusually high toxicity of uranium. For both bone cancer and myeloid leukaemia induction, uranium-233 was considerably less effective than plutonium-239 and americium-241. Concerns over the toxicity of depleted uranium have led to a number of reviews; the Royal Society in 2001 and 2002 discounted any association between depleted uranium and reported medical problems. Uncertainties are larger in relative terms at low and very low doses and are generally larger for internal than for external exposures. However claims that these uncertainties correspond to underestimates by factors of two or three orders of magnitude or more are unsubstantiated. Current estimates are as likely to overestimate as to underestimate the very low risks at very low doses.

297. Although she made an error about the radioactive nature of natural uranium, Professor Thomas was well within her area of professional expertise when reporting that there was no evidence of enhanced cancer risk from uranium although it was genotoxic in high doses.

298. Each one of the ECRR theories relied on by the BS appellants to challenge the basis of ICRP recommendations has been examined in the past twelve years by the reputable bodies noted above. Whilst uncertainties always remain and further research is recommended, nothing has emerged to justify the proposition that ICRP model is either flawed generally or defective with respect to internal ingestion of uranium in particular.

299. We find that the ECRR hypothesis has been promoted without scientific rigour while, by contrast, the wide-ranging response to it has properly analysed the data with rigour and given coherent reasons for the conclusions reached rejecting it.

300. Nothing has emerged from the evidence of the BS expert witnesses and the materials they cite to throw any doubt on the ICRP model. Indeed our evaluation of this evidence merely confirms the reasons given by others for rejecting it. The positive case that risk assessment using ICRP is flawed is rejected. The rejection is not a matter of preferring one body of scientific opinion to another, but an acceptance of the consensus of scientific opinion against the unscientific assertions of another body of campaigners. The fact that these campaigners include scientists, many of whom who are eminent in their specialised fields, does not elevate, doubtless passionately held, concerns into scientific evidence. Nothing has emerged

from the BS reliance on the ECRR hypothesis that would qualify as a reasonable doubt to be taken into consideration in the later stages of this analysis.

301. We, therefore, consider that the application of LNT when assessing risk to health is appropriate. This means that any potential risk to the health of the veterans from their service at Maralinga or CI, requires some estimation of exposure in terms of dose. We accordingly turn to Mr Hallard's evidence concerned with that issue.

PART FIVE:

THE EVIDENCE OF MR HALLARD

Expertise

302. As we have indicated in Part One, following the decision of the Upper Tribunal, the Secretary of State decided to commission fresh expert reports to address the possibilities arising from the evidence. Mr Hallard is a health physicist with a background in radiological protection at British Nuclear Fuels Limited Sellafield. He was tasked to review all the evidence before the previous Tribunal and relevant available documentation and make an estimate of radiation dose to which these veterans could have been exposed. He did so in exhaustive detail.

303. His first report consists of 285 pages and his second, when he re-calibrated his results taking into account additional nuclides, amounts to 94 pages. He then gave 151 pages of answers to questions posed by the BS group of appellants. It would be difficult to attempt to summarise all of his reasoning and the various imponderable factors he took into account when making his calculations.

304. Although he has no specialist expertise in theoretical nuclear physics, radiological environmental monitoring, deposition dynamics, meteorology, or the biological effects of radiation, we are satisfied that he has appropriate expertise in dosimetry and radiological safety. At the case management stage of these appeals, the Tribunal accepted the submission that it would be helpful and relevant to our task, if it were possible to do so, to have an assessment of the maximum absorbed dose to which these veterans could have been exposed, and from that proceed to the effective and equivalent dose calculations applying all known possible pathways.

305. We are also satisfied that he has at all times sought to act as an independent expert, applying the principles of the common law as reflected in CPR 35 and the observations in GMC v Meadows [2006] EWCA Civ 1390 (see Annex A). In particular, Mr Hallard has provided us with a list of materials that he took into account; he has explained his reasoning process by reference to the evidence and relevant literature; he has readily acknowledged the limits of his expertise and has not sought to base his conclusions on an opinion of his own that he is not qualified to make. Of necessity, the task he was asked to conduct relies heavily on the expertise and opinions of others. Such a method of giving an opinion is a perfectly legitimate one for an expert to take. His report recites that he has taken his guidance from the direction of Mr Justice Charles that all possibilities (and not merely facts established on balance of probabilities) are to be taken into account when applying the test under the SPO.

306. In identifying his assumptions for the purpose of the calculations he has been asked to make, he has explained that he had to make assessments on matters on which he has no specialist expertise. He has at all times, in his report and oral evidence, been at pains to explain the limits of his expertise and the reasons why he has made the choices he has done. The basis for some of those assumptions and choices was explored in cross-examination, to which we will turn later.

307. Dr Busby has questioned his independence and his qualification as an expert by pointing out that he has spent much of his life (34 years) as an employee of BNFL Sellafield and is therefore a member of the nuclear industry and would be, consciously or otherwise, biased in his opinions on the topics on which he has given evidence.

308. In our view, there is no substance in this criticism:

    (i) Any experienced health physicist who is asked to give practical calculations of dose will have had some experience of working closely with nuclear activities.

    (ii) This fact has been fully disclosed in his CV, as it should be, along with any other connection to the parties in the litigation that might result in a conflict of interest.

(iii) He is not and has never been an employee of the MOD and has had no previous engagement with the military use of nuclear energy.

(iv) Since 2011, he has been engaged as an independent radiological consultant, and has served as both President and Treasurer of the UK Society for Radiological Protection, and has been a UK representative to the EU expert group for the EU Joint Research Centres.

(v) There is no conflict of interest that would throw doubt on his ability to give relevant evidence and he has declared his understanding of CPR 35 duties.

309. A separate criticism that was advanced in respect, not just of Mr Hallard but all of the Secretary of State's other witnesses, was that they had not referred to the evidence and contentions of the ECRR group challenging the ICRP model. It is a requirement of CPR 35PD 3.2(6) that 'Where there is a range of opinion on the matters dealt with in the report' the expert should 'summarise the range of opinions and give reasons for the expert's own opinion'.

310. For reasons we have given already, it is important that any court or tribunal considering a topic on which expert evidence has been admitted, should be informed of differing expert views on the same topic. However, the Secretary of State's experts were instructed to give an opinion on estimate of dose and medical causation resulting from the dose. Neither Mr Hallard nor Professor Thomas and Mr Haylock were being asked to give an opinion on whether ICRP risk models are and remain sound. These appeals are not a public inquiry into radiological protection or a re-opening of the debates that took place in the CERRIE Committee in 2004. What Mr Hallard was being asked to do was to calculate maximum dose exposure on the various hypotheses and possibilities discussed in 2013. Any competent radiological health physicist will do so using the professionally accepted tools. The ICRP has been accepted internationally and nationally; as we have explained in the previous section the criticisms that have been made of it by ECRR have been considered and rejected by mainstream radiological opinion.

311. In those circumstances, an expert technician such as Mr Hallard is not required to alert the court to a debate on the validity of the professionally accepted tools. A range of opinion is a situation where there is plausible scientific evidence of a view on an issue, even though the expert concerned gives another view that is preferred. In

our view, there is 'no range of opinion' on dosimetry, but a consensus of main stream opinion, and a minority view that is not accepted as plausible by the mainstream, despite a debate that has continued over a number of decades.

312. In evidence before us Mr Hallard said that he was not even aware of the ECRR reports when he was writing his own. There is no reason why he should have been as they do not provide the accepted model for risk assessment. If expert evidence was needed to provide an opinion responding to the concerns of ECRR as the reliability of the ICRP model it would not be Mr Hallard who was appropriate to provide it. In all these circumstances, there was no obligation on him to mention it when reaching the conclusions that he has.

313. His evidence qualifies to be received as expert evidence according to common law criteria. What we make of it after it has been tested in cross-examination is another matter.

The function of the respondent's evidence

314. Neither do we accept a submission made by the HL appellants, supported by the BS group, that, by commissioning Mr Hallard's report at all, the Secretary of State was failing to explain his response to the appellants' case as lodged, pursuant to the directions of Charles J. In our view, once the Tribunal had accepted the relevance of revised estimate of dose and varied the directions of Charles J for it to be obtained, it was apparent that the response to the appellants' cases would depend on the outcome of the calculations that Mr Hallard was being asked to perform and that in turn formed the basis for the medical opinion of Professor Thomas and the bio-statistical opinion of Mr Haylock.

315. In our view, there has been a very significant shift in the Secretary of State's position from the 2013 hearing. At that time, the respondent disputed the proposition that possibilities of exposure as identified by Professor Regan and Dr Nicholson were plausible when they did not accord with the assessments of the AWE scientists. Accordingly, it was contended that they were not evidence that should enter the overall factual balance, rather they were mere hypotheses not supported or established by reliable evidence. The AWE assessments are no longer relied on as conclusive as to the possibilities. It is now accepted that the results obtained from the film badges and/or the sticky paper trays used in the environmental monitoring,

cannot themselves be sufficient to determine all plausible possibilities as to risk to health from military service. In those circumstances, the debate has moved on. The issue now is much more the estimation of the impact of such possibilities on dosimetry and health assessment.

The Hallard approach to dose assessment

316. Mr Hallard has thus taken a different starting point from that adopted in 2013. As his detailed reports and oral evidence makes clear, he has not based his calculations of absorbed dose on the highest sticky paper sampler readings. Rather he has looked at the whole scheme of radiological protection at Christmas Island (CI) and calculated what the maximum amount of radioactive material to which the veterans could conceivably have been exposed.

317. We summarise this part of his evidence as follows:

(i) There was an appropriate scheme of radiological protection at both the Maralinga and CI tests.

(ii) Reliance on sticky papers as the primary way to detect fallout out from the Grapple (G) X and Y detonations was a weakness with the system of environmental measurement as it would not provide a system for confidently detecting localised fallout. However, when this data was combined with the ground surveys using portable instruments and other available information, then it can confidently be concluded that there was no widespread fallout as it would have been detected if it had existed.

(iii) The background radiation is very low on CI. Any significant increase in radioactive fallout would have resulted in a notable rise in gamma and beta radiation that would have been apparent as soon as the sensitive equipment used for monitoring was switched on.

(iv) Although there is no detail of systematic environmental surveys using the Geiger counter detectors, it is apparent from a number of references in the documentation that these detectors were used when assessing contamination of in a number of ways and places. Thus the Jones Report[101] states at 2.3 that methods of detection included 'a ground survey

[101] 17/16/2 Operation Grapple Y Residual Radiation Measurements

with sensitive radiation detecting instruments to supplement data contained from the sticky paper containers'. The instruments included the 1250 and 1320 Geiger contamination detectors. The Clare Report identifies that where sticky sample positive results were found confirmation was sought by hand held instruments. The Oldbury and Jones Reports both indicate that equipment was used at tests sites, and decontamination centres that measured background radiation and observed increase of radiation over background rates. Much of this activity took place in the populated north of the island.

(v) Under the Radiological Safety Regulations for Christmas Island paragraph 3.3.2, the Health Physics controller was required to determine the boundaries of the Controlled Area by reference to the maximum permissible level (MPL). The MPL for products of any age was 4.3 $\mu Ci/cm^2$ x 10^{-2} (i.e. a figure of 4.3 micro curies per square centimetre). This amounts to 430μ Ci/m^2 (square metre). Using the information provided in the Carter Report (vol 1; p. 76) Mr Hallard converted this to Becquerel as 1.6 x 10^7 Bq or 16 million Bqs per square metre (16M Bqm^2) This amount of deposition would give a dose rate of 32 μSv per hour at one metre above the ground an hour after detonation. This would be more than 1,000 times the background gamma radiation level and widespread fallout at this rate would have been obvious to operators of these detectors, particularly if the level increased significantly and remained high on more than one detector.

(vi) By contrast to this assumed level of general contamination, the highest recorded contamination in any of the 18 sticky paper samplers distributed over the island, was at Vaskess Bay, in the uninhabited south of the island following GY, where a single reading of 150 $\mu Ci/m^2$ was recorded. As the Jones Report indicates, this was the only positive result yet surveys using portable 1320 monitors were carried out over the whole island after this test.

(vii) For the GZ series of tests there was also positive evidence of low dose rates recorded by means other than sticky paper samples. Vehicles leaving the control area for the balloon mounted tests (GZ1 and 4) were monitored

and low levels found. For the air burst tests (GZ2 and 3) there was no measurable dose rate in the forward area. The sticky paper samples were also low with a maximum of 3 x 10^3Bq per square metre (i.e. 3000 Bqm^2).

318. Nevertheless, Mr Hallard goes on to assume that given the imponderable issues about local deposition, that the whole island was contaminated to the maximum permitted level for fission product contamination of 16M Bq for each detonation (i.e. after each of GX, GY and GZ detonations). He concluded that the likely mechanism for such a high level of deposition would be rainfall shortly after each detonation.

Overestimation of deposition

319. Whilst we understand the purpose and the reasoning behind this assumption, and we will follow it through to see what calculations he has reached from the maximum possible exposure in Bqs to the effective dose in Svs for each individual veteran, we have no doubt there was nothing like this level of deposition all over the island for any of the tests, let alone all of them.

320. Amongst other things, we note:

(i) Rainfall was assessed to be the most likely means of this assumed contamination, but there was no rainfall for three of the four GZ tests, and there was positive evidence of low contamination for all of these tests, supported by, but not reliant on, the sticky paper sampling. While we assume that there was some local rainfall after GY, it was clearly not island wide rainfall. It is improbable that the only place it did not rain was the meteorological collecting station.

(ii) Whilst rainfall is a potentially significant mechanism of wet deposition in GY, and nothing is known about GX, rain not only brings radioactive fallout to earth, it also washes it away into the adjacent sea where it is rapidly diluted to an insignificant level.

(iii) It is impossible that no radioactive deposition in the environment would be recorded, other than at Vaskess Bay, if there had been a significant deposition at the assumed levels.

(iv) The records of measurement of sticky papers and Geiger counter monitoring are reinforced by water and air sampling and other equipment monitoring contamination.

Calculation of dose

321. We are, therefore, sure that Mr Hallard was indeed using figures for the maximum conceptually possible degree of contamination on the ground that was very considerably greater to anything that could actually have been there.

322. Direct radiation from any burst was not an issue for any of these veterans. They were located more than eight kilometres from any blast that took place while they were on the island. Although the orders to service personnel to turn their backs to the detonation for a few seconds, hardly seems a sophisticated precaution, it is not disputed that, given the distance from ground zero, this measure was sufficient to prevent these veterans from suffering harm from an acute exposure.

323. Mr Hallard made his assessment of dose from deferred exposure based on the possibility of wet and dry deposition and radioactive material in the air, resulting from all previous tests. He listed the exposure pathways as follows.

324. *External dose*: Submersion in a cloud of radioactive materials suspended in the air or deposition of fallout on skin over a 12 month period would lead to an effective whole body dose of 320 μSv and effective dose for skin exposure of up to 18 mSv.

325. *Internal dose*: This was through inhalation of material in the ground through re-suspension, ingestion of contaminated material in local food and drinking water, contamination through exposure of wounds or cuts to radioactive air or water and working with significantly contaminated objects. This might result in the following doses: 145 μSv for inhalation; 200 μSv plus 10 μSv after one year from deposition for drinking water, and up to 70 μSv for exposure for nuclides other than Pu23. No value was given for exposure to carbon-14.

326. Detailed mathematical calculations were given for each potential exposure pathway. We make a number of observations:

(i) The calculation of inhalation of Pu239 takes account of the possibility of fractionation increasing the presence of this isotope in the range of

fission products applying the evidence derived from the discussion below between Mr Johnson and Professor Regan.

(ii) The inhalation dose is used using a re-suspension factor of 10^{-4}/m. He explains he made this choice of factor on a conservative assessment having regard from the Carter Report on inhalation of dust in Maralinga where a lower factor of 10^{-5}/m was employed. Carter in turn referred to experiments on resuspension of dust into the air conducted by Turner that ranged from normal wind to a dragging experiment producing dust of an intensity that a normal person would not tolerate living in. Mr Hallard thought it improbable that re-suspension of radioactive dust into the humid air at CI in or shortly after April 1958 was comparable to dust in the desert at a high right rate of intolerance.

(iii) Some further support is derived from the cloud sampling data of the Shackleton aircraft who completed a tracking programme approximately 150 miles west of the island some 7 hours after detonation. From the measurements recorded in their instruments 600 μR/h (6 μSv/h) calculated back to 1 hour after the detonation. Mr Hallard has worked out that this amounts to 20,000 Bq/m^3 (per cubic metre). When we were considering this evidence we were interested in identifying what such a density in the air might amount to on the ground and we asked for Mr Hallard's assistance on this and other questions put to him. His response was characteristically detailed and cautious as he pointed out the imponderable issues in calculating deposition rate but if one assumed a deposition velocity of 10^{-2} per second one reaches an approximate value of 200 Bqm^2 per second multiplied by the duration of the deposition. We are not in the field of precise calculations, but the tenor of his answer confirmed our provisional view that this would be a tiny fraction of the assumed level of 16 million Bqm^2 even if the deposition from the assumed cloud stem were multiplied by 10 to account for the intensity of any particle deposition that may have reached the land on CI from the main cloud.

(iv) The ingestion dose was partly based on drinking water contaminated with radioactive products. The principal mechanism for this was a

hypothesis explored by the appellants in evidence in 2013, namely that water used to decontaminate aircraft may have soaked back through the coral until, after 12 months, it re-entered an underground pool of water that was used as part of the water supply. This was in fact a layer of fresh water on top of the heavier seawater in an area that was not far from the aircraft decontamination zone where subsequent environmental records had found trace evidence of fission products. There seems to have been a variety of sources of drinking water for veterans on the island, but Mr Hallard's conservative estimate proceeds on the assumption that *all* drinking water came from this potentially contaminated source. This seems highly improbable in reality.

327. The ingestion figure also takes account of the possibility of eating seafood contaminated by radioactive seawater. The only fruit or vegetables grown on the island were coconuts whose husk and thick shell protected the flesh from radioactive exposure. Some radiation was detected in clams on the eastern side of the island but only to a few pico-curie (pCi), that is to say one million millionth of a curie. Mr Hallard assessed that a few meant 25 pCi or 1 Bq. Mr Hallard assumed that the clams were not the only source of radiation in seafood and arbitrarily multiplied the level by a value of 1000 to achieve a dose from eating local produce of 2 μSv. Given the other available data from Pacific-wide fish and seafood monitoring this would also appear to be a significant overestimate.

328. The next stage in Mr Hallard's report was to apply these dose calculations to the individual veterans, having regard to their length of residence on the island, what they say as to their occupational and recreational activities whilst there, and any other individual factors that might create an exposure pathway. It seems that swimming in the lagoon, eating seafood, and playing sports in the northern part of the island near the main and base camps was a common theme. Some of the veterans disclosed they had received cuts from the sharp coral when swimming or playing football.

329. The conclusions can be summarised in the following table. Column 3 is the External Effective Dose based on the exposure during the length of service; Column Four is the Equivalent Dose of such an exposure to the skin. Column 5 is the Internal

Equivalent Exposure. All Doses are given in mSv. The figures in bold are the revised rates calculated after some observations from the BS group of appellants caused Mr Hallard to add some additional nuclides to the products giving rise to radioactive exposure. The comments column includes information about location at time of detonation. The various areas where these veterans were mustered: Joint Operation Centre, Main Camp and Port London are all 40 km or over from the Ground Zero detonation site. The column also records if a film badge was issued to those who worked in a controlled area on or shortly after any of the tests or whether no badge was issued because it was assessed that there was no occupational exposure

Table 3 Estimates of Effective dose for each veteran.

1. Name	**2. Duration**	**3. External Effective**	**4. External Equivalent**	**5. Internal Equivalent**	**6. Comments**
Abdale	Jan-Nov 58	2 **3**	90 **120**	2 **2**	41 km from Ground Zero at detonation. No special occupational exposure.
Beeton	Aug 57-Aug 58	1 **1**	36 **44**	1 **1**	50 km plus from Ground Zero at detonation. No special occupational exposure.
Butler	Dec57-Dec58	13 **14**	90 **110**	2 **2**	3 film badges GZI 22-23 8 58 recorded 2 x nil and 1 x 0.3mSv when 11 km from blast site for all GZ. 40 kms for GY
Hatton	Aug 58-Aug 59	2 **2**	72 **88**	2 **2**	Main camp 40kms from detonation. No film badge; duties including working in laundry
Hughes	Oct 56-Sep 57	0 **0**	0 **0**	0.002 **0.010**	Not present for any of the CI tests. Only source of exposure decontamination of planes from early Grapple series.

Lovatt	Sep 57- July 58	1 **1**	36 **44**	1 **1**	No controlled area activities. Main camp during detonations.
Pritchard	Jan 58- Aug 58 Oct-Nov 58	1 **1**	36 **44**	1 **1**	No witness statement but assumed 30 kms from detonation. Of GY and Z1
Selby	Aug 57- July 58	1 **1**	36 **44**	1 **1**	? 25 kms from detonation. No controlled area activities
Shaw	Dec 58- Nov 59	0.010 **0.100**	2 **2**	1 **1**	No controlled sites but had been asked to empty an oil filter from and clean a generator before dispatch back to UK
Sinfield	Jun 58- June 59	2 **2**	72 **88**	2 **2**	No controlled area work
Smith	Oct 59 to July 60; Aug to Nov 60	0 **0**	0 **0**	1 **1**	No presence during tests. No controlled area activity Camp Barber
Battersby	Maralinga July to Nov 59	A 2 **2** B 10 **10**	A 40 **440** B 400 **680**	A 32 1 to eyes **40 and 1 to eyes** B 35 and 10 to eyes **43 and 11 to eyes**	Assisted in decontamination of 6 sniffer Canberra aircraft Supervised visit to forward area when grazed leg disembarking

330. It will be seen that of these veterans only Mr Butler entered a controlled zone for any of the tests and was accordingly assigned two badges on the day of GZ1 and one badge the following day when he went to repair a damaged mast. The assumed

external dose made by Mr Hallard is six times the amount that was actually recorded on the film badge for the second day.

331. Mr Hughes was not present for any of the tests conducted on CI. His witness statement refers to his presence on an unannounced test. There was no such detonation recorded in the scientific records and his recollection is possibly confused with a practice run that did not result in a detonation. We are sure that Mr Hallard was right not to assign a dose on the hypothesis of presence at a detonation that never took place.

332. The highest doses estimated are all skin doses occasioned by lengthy exposure to the atmosphere in which the residue of all or most of the tests were conducted with the assumed rate of radioactive deposition. Whilst estimates of external exposure can be relevant to assessing internal exposure, it is of limited relevance to causation of most of the medical conditions with which these appeals are concerned. Thus, although therapeutic radiation of the human body is an external source of radiation that may contribute to cancers, the nature of the present medical conditions meant that Professor Thomas was principally concerned with internal dose when assessing cancer risks. In a case where internal dose led to possible causation of medical condition, Professor Thomas deferred to Dr Haylock's calculation as to the probability of any causal link. External dose is relevant to other medical conditions such as cataracts and heart disease. We examine the evidence relating to the health conditions of each of the veterans at Part Seven of this determination.

333. Mr Hallard's original (unrevised) figures were the basis on which Professor Thomas made her assessments of the possibility of causation. As the internal dose figures did not change at all or materially, the further calculations done in May 2016 generally had no impact on the work of Professor Thomas and Dr Haylock.

334. Internal exposures were all assessed to be at the very low levels of 1 to 2 mSv with the exception of Mr Battersby. His exposure was assessed to be significantly higher because his witness statement indicates that he assisted in the process of decontamination of six Canberra sniffer planes which flew through radiation clouds after the Buffalo tests. There is no record of his being engaged in such work, but we cannot exclude the possibility that he might have done so. Mr Hallard devotes a number of pages of calculations as to the mechanics of exposure by this pathway,

given the evidence that the monitoring of Canberra pilots revealed that as a group they were the most at risk of the upper range and above of the tolerated exposure levels. In the end he came up with two calculations. Option A involved Mr Battersby being involved in the decontamination of the six least contaminated aircraft and Option B involves assessment on the basis of involvement with the six most contaminated such aircraft. The high external dose for such activity led to a high internal dose as a result of possible mechanisms of ingestion and inhalation of the contamination.

The appellants' response to Hallard

Cross-examination by the HL appellants

335. From Mr Ter Haar's cross-examination of Mr Hallard the following topics of potential relevance to our determination emerged:

(i) Safety standards are evolving. There is a gap between advice first being given and legislation to enact it: for example the ICRP reduced recommended exposure limits for medical radiation of the eyes have not yet been adopted in legislation. As a mere health physicist Mr Hallard may not be aware of all current scientific hypotheses about risk.

(ii) Contemporary safety standards for those exposed to handling plutonium in a nuclear power plant means that they are regularly tested for the presence of plutonium in an urine sample and dose details are given by the employer for a lifetime record of exposures. A personal air monitor was used effectively previously.

(iii) There was no evidence of urine monitoring during the Grapple tests. That is not necessarily an astonishing omission by modern standards as one would still need to assess whether there was a case for monitoring by reason of exposure. The judgment at the time was this was not necessary for the service personnel engaged in tasks that were not considered a source of risk. Further the equipment for measuring urine by spectrometry would not have been available at CI at the time, only back at Aldermaston.

(iv) The film badges used in the Grapple tests did not give readings for alpha emissions. It was only possible to tell the broad range of the types

of gamma energies and a fairly crude discrimination between gamma and beta energy.

(v) There was no evidence of monitoring for internal alpha radiation during the Grapple tests. By modern standards those who tested positive for some radiation exposure by film badges or otherwise might well be expected to have been tested for alpha exposures.

(vi) The absence of contemporary monitoring for alpha emissions and more thorough use of film badges makes the dose of dosimetry assessment 56 years later more difficult. Alpha emission specific monitoring equipment could not have been done in the field at the time, but the basic gamma ray detecting Geiger counter can tell you something about the presence of alpha emitters, once you have agreed information on the composition of the radionuclides and you can calculate the proportion of alpha emitters from that of gamma emitters.

(vii) Sticky paper samplers are not the best way of recording radioactivity in a wet deposition. However, even with the loss of information caused by rain on a sticky paper sampler some information would be retained if there was a high level of radioactivity. There was also the wet deposition sampling from the one rain water sampling site on the Island.

(viii) The Clare Report does not deal with particle size. Mr Johnson calculated the larger particle sizes likely to be present. The Carter Report gives more details on particle size that can to some extent be read across, although the Maralinga weapons were atomic and not thermonuclear. Information about particle size and distribution was available from the cloud sampling missions and records of analysis.

(ix) Contrary to Dr Harrison's comment, it would not be unusual for radioactivity to be detected in the uninhabited south of the island and not be found in the inhabited north given that the Joint Operation Command, Main Camp, Base Camp, Port London were 40 plus kilometres away from the detonation site.

(x) The mechanics of particle deposition could lead to an underestimate in any sticky sampling tray of up to five fold, but Mr Hallard's dose estimates were not based on the sticky sampling trays. On the evidence that there was electronic sampling of the island after detonations and, with the exception of some specialist buildings in the JOC, nothing was found requiring an additional controlled area to be declared.

(xi) If the Tribunal found that the chromosome aberrations noted in the Wahab and Rowland report were caused by radiation at CI in the doses that they retrospectively assessed, that would have an impact on the dose calculation assumptions made in that report.

(xii) Although Dr. Nicholson has estimated a range for re-suspension factors from 10^{-3} to 10^{-6}, Mr Hallard considered that, given the dust dragging trials in Australia referenced by the Carter report suggested that the level of dust would be intolerable to most people at 10^{-5}, a re-suspension factor of 10^{-4} was appropriately cautious for CI where by comparison with the Australian desert there was less dust and a more humid climate particularly at the time of GY.

(xiii) Pacific sampling of fish after detonation used a 1257 Geiger counter to test for activity, and if a positive result was achieved more detailed examination would follow. Mr Hallard calculated that using a Geiger counter on a fish caught within 200 days after detonation could detect radioactivity that gave rise to an internal dose of few (10 or below) mSv.

336. He was pressed by Mr Ter Haar on his calculations of rain deposition using the 16 million Bq per m^2. It was put that to arrive at a figure of density of concentration in a cubic metre of air he needed to multiply his assumed figure by 10. This would give a concentration of 160 million Bq m^3. Whilst acknowledging his lack of expertise in deposition rates and density of such particles, we note the following answers given in response:

> 'A. My interpretation of what they were saying was that wet deposition, if any deposition had occurred, was much more likely. And also we clearly have reports, well documented in Mr Stretch's report, of heavy rain at Port London, Main Camp and other areas. What I have done is to -- and the reports of rain, if I

read them correctly, and some of them were a little bit ambiguous, but I believe the reports of the rain were between 30 minutes and an hour. So what I'd assumed is that the rainfall had carried on for an hour, at an unspecified activity, had deposited activity on the ground at the levels that I calculated -- and again I can explain why I've chosen that figure if need be -- and that those levels were then deposited on the ground and that activity in the form of fission products, plus plutonium, plus uranium but the fission products in particular would have then decayed at the standard rate if I can use that term, which is a very rapid decay rate. But I've then made my calculations based on that and integrated some of those estimates in order to get the total activity over a period of time….

I felt that there was so much uncertainty about whether any rainfall had been contaminated that I would never be able to answer that question with any certainty whatsoever. I felt that there was more reliability in the monitoring on the ground and that is what I've taken as the starting point.

Q. I understand and you have made that very clear. The difficulty for this Tribunal is that what this Tribunal is looking at are possibilities --

A. Yes, I understand that.

Q. -- rather than probabilities or the balance of probabilities. The reason I took you to that part of Dr Nicholson's report at some length is that he certainly regarded it is as being, in his own language, if you like, a serious possibility that there was radioactive rain falling on the Island very soon after the explosion. And this I think is within your expertise, that radioactive material suspended in rain may not yet be wet deposition but is actually a very effective way of taking in material that is suspended in the air?

A. Can I just test I understand what you mean by that question? Do you mean that if it was raining and if that rain was contaminated that that's a very effective way of inhaling the activity in that rain?

Q. Yes.

A. I'm not sure I would agree with that, simply on the basis -- and there is some intuitive judgment here --that if you are standing or walking in rainfall you don't tend to inhale the raindrops. It tends to be because the particle size is too big, apart from anything else, that we don't walk across the street and inhale raindrops. The things that we would inhale would tend to be much smaller particle size, much more akin to a dusty environment where the activity has already been deposited on the ground and then re-suspended in the way that I've indicated in the report, particularly the smaller particles of the order of 1 micrometre or smaller.

…

Q. What is suggested is that if that is the atmosphere that is much more -- when you breathe in you are much more likely to take in the radioactive materials than if there is dry deposition on a sunny day.

A. I think that's more in Dr Nicholson's area of expertise than mine. Just to make a comment. Water is a very good suppressant for dust. Indeed, I've noticed a comment about HMS Diana, I think, in one of the reports, where the ship which had been involved in testing at Australia, the instruction was given to damp down the deck because the deck had found to be contaminated and then there was some

> debate as to how effective that was. If you put water on to a contaminated surface it's a very good way of damping down dust. I mean we might do that in our homes. Therefore, I'm a little surprised -- surprised from an intuitive point of view. I think I would just ask a question and I don't think I have the expertise to be able to answer that adequately.'

337. On the basis of the data supplied by the Shackleton aircraft survey, conducted seven hours after the detonation of Grapple Y, Mr Hallard calculated the size of the radioactive cloud through which the planes were flying at a low level of a few hundred feet. From the size of the cloud he obtained from these calculations he concluded that this was the anvil of the main cloud rather than the stem, and accordingly assumed that the cloud with this level of activity had passed before over the whole island. This was a worse case assumption on his behalf, because the stem was much narrower than the main cloud and would not have passed over the inhabited part of the island. It was not intended to be an assumption of meteorological movement. If what was being surveyed was the diffuse remnants of the stem 150 miles west of the island, then it was very unlikely to have been a source of exposure by means of contaminated rainfall to veterans in the north of the island as it would not have extended that far when it passed over them.

Cross-examination by the BS appellants

338. Dr Busby's cross-examination of Mr Hallard was principally devoted to the contention that he was wrong to use the ICRP model to calculate internal dose, particularly if such a dose included small particles of alpha emitters such as uranium. Mr Hallard was willing to assist as far as he could but stressed that the overall critique of ICRP was beyond his level of expertise as a practising health physicist.

339. With this general description of the exchange, we note his answers on the following topics:

    (i) He used ICRP data because it is the internationally accepted standard and is relied on by the IAEA and the EU Directive in setting basic safety standards. He did not think that using such a standard was evidence of cultural bias on his part. He was unaware of any public authority using the ECRR model modifying the ICRP one.

(ii) He considered that ICRP had looked at opposing theories and considered them in an informed way when responding to them and maintaining their models rather than ignoring the evidence.

(iii) The ICRP model averaged the effect of an ingested radionuclide over the whole organ in question to achieve a dose. This was considered to be appropriate because although there may be more localised exposure of tissue to such a radionuclide, the most likely outcome would be that the cell would receive so much radiation that it would die and not be around to mutate. The risk would therefore be zero. Averaging the impact over the organ was thus a more conservative estimate of health risk rather than a zero outcome.

(iv) The majority report of CERRIE recognised that there could be uncertainties in internal dose using ICRP model and they could range from a factor of 3, if there was high confidence in the data, to a factor of 10 if there was not. Mr Hallard had answered a written question in response to his report that drew attention to the CERRIE Report, the COMARE report given in response to it and two papers from the Health Protection Agency in 2012 and 2013. He explained that having used the ICRP model to assess internal dose he did not apply any uncertainty factor to it. He gave two explanations for not doing so: he did not have sufficient expertise to understand what uncertainty factor he should be applying and why and as he understood the literature it was suggested that ICRP dose efficient should be used without applying any uncertainty factors. For reasons that we give below at [340] having reviewed the material for ourselves we conclude that is second answer is correct. Whether ICRP itself provides a reliable model for internal assessment is a matter that we have already considered.

(v) Taking the gist document that gives a conservative upper limit of the total mass of uranium-238 in all the devices on CI (multiplying the true amount by up to 10 times for security reasons) gave a calculation of 8,000 kilograms before detonation. The amount for GY alone was 3.3

tonnes. It would be unrealistic to make a calculation of radioactive deposit on the assumption that all this fell on CI. Much of it would be destroyed in the detonation, and of the remainder it cannot be assumed that it all fell on the territory of the island as opposed to the sea or being carried away in the cloud.

(vi) Monitoring using Geiger Mueller counters had taken place following GY at Vaskess Bay, Port London and Main Camp. Although these machines did not detect alpha radiation, as agreed between Professor Regan and Mr Johnston in 2013 (on the topic of co-deposition), once you knew the range of nuclides in the fissile material, the distribution will be proportionate and by measuring the gamma and beta radiation you will be able to estimate the alpha radiation. This ability would only be impaired if the beta radiation had all disappeared at the time of the monitoring. If there was some radiation left to be measured the quantity at the time of detonation could be assessed by calculating backwards reversing the rate of decay using the known properties of the relevant half-life. These Geiger counters would be able to detect beta radiation in the rain, although not beyond a depth of around two centimetres in a pool of water.

(vii) Allyn Seymour of the University of Washington conducted a preliminary radiological survey of CI in 1975 and found naturally occurring potassium (K-40) and uranium-238 there. In fish samples taken at Port London area, uranium-238 was detected in pCi per gram 0.56 to 1.67. The quantities were very small and consistent with background radiation elsewhere in the world. The quantities in the fish were higher than for the soil but there was natural uranium to be found in the sea and uranium-238 is the principal constituent of naturally occurring uranium.

(viii) Mr Hallard was asked to (and in his revised report did) undertake further calculations adding uranium-240, plutonium and neptunium-240 into the equation as his original calculations were derived from the Carter Report that was concerned with atomic rather than

thermonuclear weapons. He derived the information on these nuclides from the gist document on the British weapons rather than a study by S. Simon on nuclear detonations in the Marshall Islands using weapons with which he was not familiar. He believed that his recalculations of Carter were appropriate and had a short conversation with Aldermaston to confirm this. Any difference between the rates used by Carter and Simon would have been limited to assessment of external dose. It was better to work with the ingredients of the specific weapon as all weapons may be somewhat different.

(ix) He was asked to comment on the record of a 1953 conversation in which Dr Karl Morgan had stated that hazards from enriched uranium were radioactive rather than toxic[102]. He agreed that this could be the case depending on the proportion of enriched uranium (U-234) used. He assumed that in 1953 the conversation referred to a device with a significant proportion of uranium-234 which is 3000 times more radioactive than uranium-235. Uranium-238 is the least radioactive of the three. The gist document disclosed the maximum values of uranium in the weapons used in the British tests were: U-238 8,000 kilograms, U-235 400 kgs, U-234 10 kgs.

(x) It was suggested that Mr Hallard had missed out another relevant exposure pathway: sea to land transfer, as had been measured in the Irish Sea in relation to Sellafield. He was aware of such a potential pathway but did not consider it to be significant in the present context. The dilution of radionuclides into the Pacific Ocean around CI would be considerable and would rapidly dilute any radiation. To be a significant pathway it would have to be greater than the assumed deposition rate for the island as a whole 16M Bq metre^2 and he did not think that this could be the case. He could not comment on local currents and their ability to deposit such material.

(xi) He agreed that the photoelectron effect is one of several means by which gamma radiation is absorbed by atoms. One would expect to see

[102] Recorded in a document found at SB 22/11

more photoelectrons from uranium than for example from oxygen in water. There is a significantly higher absorption of gamma in big atoms like uranium than small atoms like oxygen. These features do not affect his dose calculations. They have been examined in a paper by Public Health England.

(xii) He accepted that his inability to calculate the effect of carbon-14 in his report weakened it somewhat. He was at the limits of his expertise but noted that an UNSCEAR report recorded that the dose from carbon-14 was perhaps 2 to 3 times greater than for tritium. The agreed gist recorded that there was 1500 moles of carbon-14 plus/minus 500 moles. This amounts to 28 kilograms for all the Grapple tests.

Uncertainty factors:

340. We have noted at [339](iv) that Mr Hallard was a little unsure about whether he should have applied uncertainty factors to his estimates of internal dose applying the ICRP model. Potentially this was an admission that might have significantly undermined his calculations of dose. Having re-examined the literature to which he referred in his written and oral evidence, we conclude that there is nothing in this point. It is recognised that estimation of internal dose is more difficult than external dose, and estimating dose at a very low level is more difficult than high levels. The context for the CERRIE Committee comments on uncertainty in dose calculation was the further problem of general environmental exposures to members of the public at nano or micro levels of exposure.

341. In commenting on this problem, the COMARE report noted that uncertainties can operate in both directions over and under estimation of risk and:

> 'where statistical uncertainties are concerned, while confidence limits (a measure of uncertainty) may be wide, in practice the real value is much more likely to be close to the central value than the other extremes'.

342. The most recent report to which we referred on this issue is Puncher and Harrison 'Assessing the reliability of Dose Coefficients for Ingestion and Inhalation of

Radionuclides by Members of the Public'[103]. Mr Hallard was clearly aware of this document. Its summary and conclusions is as follows:

> 'ICRP is clear on the intended use of equivalent and effective dose as reference quantities, without uncertainty, for use in internal radiation protection. However, ICRP and others also recognise that there are uncertainties in the process of estimating dose and risk that affect the derivation and application of these quantities. This report addresses this issue as follows:
>
> 1. The issue of relevance for regulators and other stakeholders is not the magnitude of the 'uncertainty' on dose estimates, but how 'reliable' dose co-efficients are for protection purposes, as a protection device. It is argued that a dose coefficient, as applied to a defined exposure pathway, is considered reliable if it ensures exposures comply with dose limits and constraints.
>
> 2. The best estimate of risk and its uncertainty for a given internal exposure pathway is a pre-requisite to making an informed judgment of a particular dose co-efficient in the context of a specified exposure pathway …...
>
> 3. A general assessment of the reliability of dose coefficients can be made by assessing the reliability of dose coefficients that are applied to the more significant exposure pathways.
> 4. …
> 5. …
> 6. Although a general assessment of reliability of the protection quantities is beyond the scope of this report, the derived (uncertainty factor) values for the radionuclides considered here seem acceptable when considered alongside the likely levels of exposure that is expected from them (the sub-micro sievert to the micro sievert) and the dose limit for planned exposures for the planned exposures for members of the public: 1000 micro sieverts; and viewed in the context of all radiological hazards to the general public: an estimated annual dose of 2700 micro-sieverts….'

343. We draw the following conclusions from all this material:

(i) The ICRP model asks the health statistician to apply its dose efficient for internal exposures of radionuclides and not then go on to multiply the risk by an uncertainty factor (UF) of between 3 and 10.

(ii) The issue is whether the ICRP model is reliable and there is not an additional calculation to be made outside of it. The relevant possibility for our consideration is whether the ICRP model is wrong, rather than whether the dosimetry calculated in accordance was wrong for failing to apply an UF.

(iii) It would not be possible for the health statistician to calculate risk by adding a multiplier for an uncertainty factor as the uncertainty may be to exaggerate or underestimate risk.

[103] HPA April 2013) (SB3 /10)

(iv) The discussion of UF from CERRIE onwards is in the context of tiny environmental exposures to the general public when there are many unknowns.

(v) Here the dosimetry was on the basis of a very precise assumed dose and then applied the approved mathematical mode; with tissue weighting and the like.

344. Accordingly we conclude that there was no error in his calculations applying the ICRP model by failing to make a further adjustment for uncertainty.

Carbon-14

345. Mr. Hallard accepted that his calculations did not take into account the contribution that Carbon-14 might make to dose as he had no expertise in the calculation of environmental dose. On reviewing his evidence we were unsure whether he would be able to give us any further assistance on this topic. We therefore posed a written question whether he was able to assist as to the potential contribution of carbon-14 to dose. We are grateful for the additional work he undertook which was voluminous and his response was accompanied by a number of documents evidencing his reasoning. Not for the first time during these appeals, we were struck by what a cautious health physician he was.

346. In the substance of his answer he indicates that he found relevant information in the UNSCEAR 2000 report that permitted him to make the calculation. On the basis of these figures he has made calculations that he has disclosed of a conservative indication of dose from carbon-14 released during the tests at CI of less than 160 μSv. This estimate used an uncertainty factor of 1000. Carbon-14 is relevant to internal dose. Adding these estimates to his revised dose figures (the bold figures in Table 3 above) will not increase the overall internal dose estimate made for each veteran. The tests at Malden Island would have had no impact on the veterans at CI. He considered also the impact of carbon-14 on Mr Battersby in Maralinga but as the data for the Buffalo tests showed a total of 30 kilotons this would have been an insignificant amount of carbon-14. Overall, therefore, the addition of carbon-14 to the estimate of dose makes no material impact on dose and the health conclusions resulting from those estimates.

Conclusions as to the issues that we take forward into the overall assessment

347. The HL appellants took no issue with Mr Hallard's calculations at the second stage of the exercise, namely converting the assessment of dose in Bq to equivalent dose in Sv (see HL concluding submissions paragraph 29). The contention is that it is his assessment of the overall amount of radiation exposure that is in error. This was subject to the submission made about whether uncertainty factors had to be added to the calculation. We have already concluded that they should not be.

348. However, the HL submissions at paragraphs 32 to 44, (on sticky paper monitoring) and 45 to 54 (on ground monitoring) go on to repeat the criticisms made previously of the sticky paper sampling, and in our view erroneously state that this was a key element in Mr Hallard's reasoning. As we have endeavoured to show from the preceding analysis of his evidence this was simply not the case. Whilst he made reference to the sticky paper sampling, this was by way of comparison with assumptions of the much higher deposition made by him.

349. Equally, we found his account of why there could not have been widespread contamination at a rate higher than the assumed level to be convincing. If such widespread contamination had existed it would have been bound to be detected by the Geiger counters when they were switched on. We are quite satisfied that such surveys were conducted all over the island after GY and also that Geiger counters were used to monitor particular places or people such as the laundry, seafood, contaminated pilots and the like. The fact that there are no records of the figures obtained from such activity does not mean that the machines were not used and switched on, as the documentation as a whole suggests that they were. If *anything* significant had been found, we have no doubt it would have been mentioned in the reports issued after each event. If there had been contamination at or above the assumed figure, then the matter would have been bound to have resulted in a serious investigation as it should have led to the declaration of a controlled zone under the Regulations.

350. We are sure that there is no reasonable possibility of such widespread contamination at over 430μ Ci/m^2 (or 16M Bqm^2). The assumption of a generalised level of contamination was not a topic on which Dr Nicholson or Professor Regan gave evidence previously. No witness has been supplied by the appellants in these appeals to suggest whether deposition rates could be even higher than these extreme rates

assumed. For reasons we have already given we are sure that they are indeed a considerable overestimate.

351. We are also satisfied that Mr Hallard's assumptions of radioactive deposition also addresses the HL submission on internal monitoring (at paragraphs 59 to 64). We accept that there was no regime of urine testing and analysis by spectrometry back in the UK of biological material from these veterans. There is, therefore, no contemporary scientific record of the veterans to confirm or undermine the AWRE modelling that there was no radioactive exposure at all. However, Mr Hallard has assumed that there was internal exposure by the ingestion and inhalation pathways he has identified. He has equally assumed that a proposition of the fallout from fission products would be alpha emitters and has used ICRP models to convert this rate of exposure to equivalent dose in mSv. The submissions made thus miss the point of what has happened at this appeal.

352. Nevertheless, whilst we are sure that there was no possibility of general or widespread radioactive exposure above the 430μ Ci/m^2 level, we do not find anything in Mr Hallard's evidence that eliminates the possibility of one or more localised areas of hot particle deposition on the island. In the absence of positive evidence of a comprehensive survey by Geiger counter of every metre of the island we cannot be sure that a particle of a single micron or so might not have landed.

353. It seems to us, therefore, that we must take into account, when evaluating the overall possibilities and certainties, the possibility that:

(i) There was rainfall in the northern part of the island shortly after GY.

(ii) The rainfall included 'rainout' from the radioactive cloud that might have been over parts of the island on its progress to the stratosphere.

(iii) The rainout included hot particles of fissile material from the detonation.

(iv) One or more of the veterans might have come into contact with such a hot particle and received an internal dose by inhalation or ingestion.

(v) There may have been localised deposition from GX where there is no recorded information about rainfall.

(vi) Although dry deposition from the high altitude tests at GZ, where there was no evidence of rain, does not seem a hypothesis favoured in 2012 or now, we must take it in account as a further possibility.

(vii) Equally we recognise that having regard to Dr Busby's cross-examination of Mr Hallard we take into account the possibility that there were other contributors to the absorbed dose in Bqs, namely a sea to land exposure pathway.

(viii) For reasons explained we are satisfied that we can exclude carbon-14 from the overall assessment.

354. In reaching our conclusions we will also take into account the inherent uncertainties involved in estimating equivalent dose applying the ICRP model which are factors that we need to take into account when assessing the possibilities and certainties.

355. Ultimately, we will have to determine whether there is a real possibility that:

(i) any of the veterans was exposed to dose that was significantly higher than the final calculations that Mr Hallard gave us and

(ii) any of their medical conditions resulted from any such exposure.

PART SIX

THE CASE OF THE HL APPELLANTS

356. The written submissions of the HL appellants address the possibility of radiation exposure causing health hazards to the veterans under four headings:

(i) Mr Hallard has underestimated the level of radioactive deposition on Christmas Island (CI), and if the true dose may have been higher so would the risk of illness.

(ii) The Rowland and Wahab study of chromosome aberrations in New Zealand (NZ) Veterans raises a reasonable doubt about the level of radioactive exposures of the veterans.

(iii) Epidemiological studies of the test veterans show an enhanced health risk that cannot be dismissed as fanciful.

(iv) Professor Mothersill's evidence given in 2013, as to health outcomes at low doses of radiation, is plausible evidence raising a doubt.

357. We have already considered the first of these issues in Part Five of this determination and do not repeat our conclusions here.

Rowland and Wahab study of New Zealand veterans

358. All the appellants rely on the study of selected NZ test veterans conducted by Rowland, Wahab and others in 2006. The report of this study formed an important part of the debate in the Limitation Act issue before Foskett J.

359. There are in fact two papers on the topic. The first is an unpublished report to the New Zealand Nuclear Test Veterans Association 2007 (hereafter Rowland et al[104]). The second is a paper that has been peer-reviewed and published in the journal Cytogenetic and Genome Research 2008[105] (hereafter Wahab et al).

360. The Wahab et al paper reveals the following method:

(i) Blood samples from 49 of the estimated 551 naval personnel who took part in the Operation Grapple series of tests at Malden and Christmas Island were examined for excess chromosomal damage in their blood lymphocytes.

(ii) The 49 were selected first by response to questionnaire and then by application of exclusion criteria, removing those who had subsequently been exposed to a theatre of war or nuclear radiation, had had radiation or chemotherapy treatment, or had had occupational exposure for a year to toxic substances (defined to include asbestos, oil fumes, microwave radiation and other radiography work).

(iii) The lymphocytes were cultured for 72 hours and then sent off for analysis using the Multicolour-Fish (M-Fish) technique to detect translocations in stable cells using a scoring system.

(iv) A 72 hour culture period was used to accommodate a number of different assays although a shorter period of 46-50 hours is normally used to ascertain the frequency of stable aberrations many years after exposure.

(v) The results were compared with a control group of 50 male age-matched veterans who had undergone military or police training when younger. All naval ex-servicemen were excluded from the control

[104] SB 7/123 after the published paper
[105] Wahab and others 'Elevated chromosome translocation frequencies in New Zealand nuclear test veterans'

group on the basis that they might have served in ships used in the Grapple tests that had been contaminated in some way.

(vi) From a total of 9360 cells scored in the veterans 226 cells were observed with one or more translocations.

361. The outcome showed that the Grapple veterans had a higher level of translocations per 1000 cells than the control group. A 95% confidence interval was used for the mean results with an upper and lower value.

Table 5 Rowland/Wahab results

Group	No in Group	Mean no of translocations per 1000 cells	Standard Deviation	Standard Error	CI Lower	CI Upper
Veterans	49	29.38	17.52	2.50	24.08	34.15
Control	50	10.05	8.86	1.25	7.29	12.32

362. There was also a comparatively higher frequency of variation in the dicentric (12) and acentric (77) cells in the veterans compared with controls: dicentrics (1) accentrics (48).

363. The Wahab paper concludes:

> 'The significantly higher translocation frequencies in the group of veterans compared to the control suggests that this may be a consequence of their participation in Operation Grapple… However, since statistical association is not necessarily proof of a causal relation, possible confounders need to be considered.[106]'

364. The authors excluded the higher rate of smokers among the veterans by comparing results amongst the two groups of smokers. Some confounding factor due to naval service might be possible although was not thought to have had such a lasting effect.

> 'Whether radiation exposure during Operation Grapple or contaminations incurred by the naval personnel can have been the causative factor is not easily answered. It will require careful reconsideration of the type and magnitude of potential exposures.'

365. An estimate of retrospective dosimetry was made by exposure of blood samples of donors *in vitro* to X ray radiation with cobalt-60 at various doses between 1 and 3.5

[106] P.84

Gy to obtain an extrapolated dose response curve. This was done in order to indicate the magnitude of exposure that might have been responsible for the observed translocations.

366. Retrospective biological dosimetry was then attempted from the M-Fish data relating to the veterans. Dose estimates were made and they ranged from 0 to 0.431 Gy in the veterans with a mean dose of 0.170 Gy and with 0 to 0.22 Gy in the controls with an estimated dose of 0.037. These can be converted to Sv at the rate 1 Gy = 1 Sv for gamma radiation.

367. The Rowland paper (although it is to be noted not the peer-reviewed and published Wahab paper) set out in table form the significant variation in estimated dose range amongst the samples from the veterans ranging from 1.4 Gy (i.e. 1431 mSv) at the highest extreme to 0.12 (120 mSv) at the lowest measurable end of the scale. 14 of the 49 were assessed as having 0 dose. This paper recognised that there were many uncertainties surrounding the estimate of dose that could only be used as a guide.

368. In conclusion the authors of the study were of the view that the highly elevated frequency is most likely attributable to radiation exposure.

369. These results have been the subject of expert comment in the written evidence before us of Dr Brenner and Professor Parker for the appellants, and Professor Kaldor and Dr Darroudi. The work has also been the subject of a review by the HPA in 2007. Professor Thomas and Dr Haylock also commented on this work when giving their evidence before us.

370. As we made plain in the course of the hearing, despite a criticism made by Professor Thomas, we accept that the M-Fish is a recognised technique for detecting chromosome variation in cells and there is no reason to doubt that this study was performed with scientific rigour and has produced a discrepant comparison of cell translocations by comparison with a group of controls.

371. We further accept that the result of this work means that there is a plausible possibility that the discrepancy between the control group and the veterans may have been a consequence of radiation exposure.

The rival submissions

372. The HL appellants invite us to go further and conclude that it also raises a plausible possibility that:

(i) the source of the radiation was exposure during the Grapple tests and

(ii) that the doses of exposure received during those tests might have been as high as 1430 mSv in one case and on average 170 mSv.

If both these submissions succeed it follows that there is a real possibility it undermines Mr Hallard's assessments of dose for the CI veterans, as he himself accepted.

373. Mr Heppinstall, by contrast, contends that these results can lead to no possibility of such conclusions. He points out that:

(i) The history of the deployment of the NZ naval veterans shows a different proximity and activity in connection with the tests than the present veterans.

(ii) The estimate of dosimetry was uncertain and what the authors produced was a calibration curve rather than an individual estimate of dose to each veteran.

(iii) The Rowland table of dosimetry was never published and peer-reviewed. The high rate of disparity between the dose estimates for the veterans makes it improbable that they have shared a common source of exposure. This has a significant impact on the conclusions to be drawn from the published paper.

(iv) The high rate of radioactive exposure and dose resulting from this assessment is, in reality, ludicrous. There was sophisticated radioactive monitoring on the ships and no excess was recorded, let alone an astonishingly high dose of 1400 mSv. Some comparison can be made from the dose consistently recorded from the Canberra pilots who flew through the radioactive cloud after Grapple X and Y where the highest recorded dose was 130 mSv.

(v) The papers needed to be assessed alongside the epidemiological research into the NZ veterans[107] where naval personnel were used as controls and where no excess health risk was found save for haematological cancers (leukaemia).

[107] Pearce Report SB 22/4

(vi) There is no finding that the Wahab and Rowland veterans had adverse health effects.

The military history of the New Zealand naval veterans

374. In 1989 J Crawford, a Research Officer of the New Zealand Defence Force, wrote an account of the involvement of the New Zealand Navy in the British nuclear testing programmes of 1957 and 1958. This reveals that two ships, the HMNZS Pukaki and HMNZS Rotoiti, each with a complement of 150 officers and crew were tasked with assisting with weather monitoring of the Grapple series of tests at Malden Island and later at CI.[108]

375. The preparations for participation in the mission included taking on board a substantial amount of radiological protective equipment including battery operated contamination meters, four survey meters of two different types, 80 quartz dosimeters of three different types and 300 film badges, as well as radiological protective clothing. There was an officer on board in charge of radioactive monitoring. The film badges for the NZ crew were not processed, however, due to a problem about the storage of chemicals.

376. Both ships were in the vicinity of Malden Island on the occasion of the first Grapple detonation on 15 May 1957. The Pukaki was 50 nautical miles away upwind of the detonation and the Rotoiti was making measurements 150 nautical miles away.

377. The crew of the Pukaki observed the detonation on the deck facing away and after fifteen seconds were ordered to remove their hands and goggles from their eyes and face the fireball. The Pukaki later passed within six nautical miles of surface zero after the detonation, when making rendezvous with the British ship, HMS Warrior. The crew remained in their protective clothing until the message was received that the risk had reduced for the whole Grapple task force. Full protective clothing was also worn on the Rotoiti which was 100 miles further away

378. However, 'no notable radiation readings were received either from the atmosphere or the water by the Geiger counter'. The monitoring results for neighbouring islands revealed no appreciable change above background levels.

[108] SB 22/25

379. Similar positions were adopted for the second test on 31 May 1957. Radiological measurements were passed to the New Zealand authorities. The highest readings from local monitoring stations were found at Canton Island where an air sample in micro curies per square metre was assessed to be one thousandth part of a year's dose that would be breathed in from natural background radiation.

380. The Pukaki took part in Grapple Y (GY) but Rotoiti did not and its place was taken by HMNZS Wellington. Pukaki was 80 nautical miles from ground zero at GY. The crew wore no protective clothing in the light of previous experience. The engines were stopped so as many as possible could see the detonation, with eyes closed and turned away until 15 seconds after burst. The record notes:

> 'The cloud from the test "spread and drifted across the whole sky and was still clearly distinguishable at sunset". This situation led Lt Commander Elliott to comment that "such was our faith in the scientists ashore that no-one was heard to say 'I hope it doesn't rain'". Clearly Elliott was aware of the dangers posed by rainout.'

381. The following day Pukaki passed through Ground Zero. Seawater in the boiler room detected low levels of radiation, the only occasion when the monitoring officer on the ship detected any radiation from the nuclear tests.

382. Pukaki returned to Port London CI and took part in a local regatta before returning to NZ three days later. The ship was also involved in weather monitoring for all the Grapple Z tests.

The HPA analysis

383. The Wahab/Rowland results were reviewed by the HPA in 2007. In summary it noted the following points that needed to be born in mind when interpreting the results:

(i) The cell culture times were atypically long, and it was disappointing that scoring was not confined to the first *in vitro* division.

(ii) The statistical power of the groups compared was at the limit of possibility. A larger group would have been desirable.

(iii) The exclusion of naval personnel from the control group leaves open the possibility that some facet of military service in the navy other than the nuclear tests might explain the distinctions found.

(iv) The *in vitro* dose response curves which were used to derive tentative dose estimates were inadequate.

(v) The dicentric curve done with 96 hour cultures is quite suspect.

(vi) Chromosome aberration is not the same as adverse health effect.

(vii) The authors of the study noted that there was no report of adverse health to the veterans studied.

(viii) Follow up studies were needed to explore the results.

384. Although the results showed a possible radiation exposure explaining the discrepancy in results, however overall it was concluded that:

> 'the unstable aberrations data leave open the question of whether all the dose was delivered around the time of the nuclear weapons tests.'

The Pearce Report into the New Zealand veterans

385. Pearce and others (1990) 'Follow Up of New Zealand participants in British atmospheric nuclear weapons tests in the Pacific[109]' followed up the health of 94% of 536 naval veterans who participated in the Grapple tests on the Rotati and Pukaki until December 1987.

386. The results were compared with 91% of 1,516 controls who were drawn from NZ naval veterans who did not participate in these tests.

387. The authors concluded:

> 'In summary, the findings presented here indicate that New Zealand participants in the British nuclear weapons test programme have not experienced any detectable increase in risk of death for causes other than cancer, and there is little evidence of an increased risk for non-haematological cancers. These findings are reassuring and indicate there has not been a detectable effect on overall life expectancy from participation in the nuclear weapons test programme. These findings should be interpreted with caution as they are based on small numbers. If these findings are not merely due to chance than they are most likely to be causal rather than due to bias or confounding.'

388. Support for a causal link for haematological cancers was provided by the findings of a similar excess risk in the 1988 NRPB study of British participants in the same programme.

[109] SB 22/4

Conclusions on the findings relating to New Zealand veterans

389. We are not persuaded that the study by Wahab et al provides significant evidence for the proposition that the disparity in chromosomes was as a result of a common experience of radiation exposure during the Grapple tests. We have noted the cautious conclusion of the authors at [361]. In our view greater uncertainty is created when this paper is set against the unpublished Rowland paper.

390. Apart from the uncertainties expressed in the papers themselves we note the reasonable observations about method made by the HPA. The study was also performed on small numbers which makes it more likely that findings are due to chance and the Rowland report also admits that there were no dose estimates for 15 veterans which means that the statistical power of this attempt at retrospective dosimetry is even more limited.

391. We find that there is substance in Professor Thomas's observation (noted in Part Four at [291](xiii)) that there is a real difficulty assigning a causal link to an event nearly fifty years before the tests were conducted. Cells will have died off and been replaced. There are many different potential sources of radiation exposure that may have occurred since1957-8. We have noted at Part Two [96] some of the potential sources of radiation recorded in the BEIR report. The longer the passage of time between the putative exposure and the biological analysis, the greater the possibility of other factors occurring.

392. We recognise that this conclusion does not eliminate, as a matter of certainty, all possibility of such an exposure being causative of the results found. Nevertheless, we are sure that the studies do not result in a possibility of dose to the present veterans as a result of their service on CI being as high as 1400 mSv or a mean dose of 170 mSv and the second part of Mr Ter Haar's submission, noted at [372], accordingly fails.

393. First, we are sure that the retrospective dosimetry results for the NZ veterans recorded in the Rowland paper are highly suspect in themselves. There are a number of reasons for this conclusion. The rates are impossibly high. Such a rate of effective or equivalent dose would have required a very large exposure to radiation. None was reported although there was Geiger counter monitoring on board the vessels and alertness to the risk of radioactive exposure. Such a high level of dose would also be

expected to have produced a significant disparity in expected health outcomes by comparison with epidemiological studies of those exposed to radiation. None were reported by the authors or found in the Pearce study of a significantly larger cohort of veterans with more appropriate controls. The variation in dose between veterans is strongly suggestive that there was no common experience of exposure, even making some allowance for different locations on a ship and possible shielding effects. There are reasonable methodological queries as to the dosimetry raised by NRPB and other commentators.

394. If we had concluded that this study played a significant role in the issues before us, we would have welcomed more assistance on how exposure of blood *in vitro* to acute radiation at a high dose can be compared with the effects of delayed radiation from low doses.

395. Second, whilst the results themselves are of interest as a starting point to further inquiries into what may have caused the changes observed to the cells of the NZ veterans, we do not consider that they provide assistance to us in the present task of assessing the possibility of adverse health effects to the British participants whose cells have not been examined for chromosome aberrations.

396. Assuming that the crew of the Pukaki remained the same throughout the Grapple series of tests, the NZ veterans seem to have had a longer period of potential exposure than the majority of the present veterans. They also moved closer to or over ground zero for the first Malden island test and GY. They were performing different duties in different locations. They appeared to have had no experience of rain, following GY, yet rainout shortly after this detonation is the main proponent for contamination in the present appeals.

397. We are sure that any common experience of environmental exposure on the island during the three days the ship was at Port London following GY cannot have been the source of significant radiation exposure, for the reasons given by Mr Hallard when calculating the doses in mSv when he assumed a purposefully exaggerated high overall level of island wide contamination yet found no significant exposure to radiation.

Epidemiology

The appellants' submission

398. The third limb of the HL closing submissions to us was that support for the possibility of radiation-induced damaging effects of these veterans from their service at CI was to be found in the epidemiological studies that had been conducted on veterans participating in these tests.

    (i) There are three reports from the NRPB in 1988, 1993 and 2003 on all participants in the tests in Australia and CI between 1952 and 1967. Thus these studies included those who participated in the clean-up operations.

    (ii) There is the 1990 Pearce Report on the NZ veterans whose conclusions have already been summarised.

    (iii) Reference is also made to the Carter Report into the Australian veterans at Maralinga that found an increased rate of cancers although did not attribute this to radiation.

NRPB studies

399. Of these three sources of epidemiological data, the NRPB studies are the most significant as they have reviewed outcomes for longer and involved the greatest numbers of subjects and controls. A total of 21,358 participants in the tests were identified from MOD archives. 85% of these participants were the subject of study. Results were obtained from a control group of 22,333.

400. The first study was made in 1988 and concluded that test participation may have caused small hazards of leukaemia (other than chronic lymphatic leukaemia) and multiple myeloma, based on the observation of a significantly greater relative risk in test participants than controls. However, this conclusion was based on the fact that there was an exceptionally low number of men in the control group who had developed these diseases rather than an abnormally high number of test veterans who had. As there was no reason to think that military personnel were less susceptible to such diseases there was a possibility that the differential was due to chance.

401. The second study in 1993 tended to support the hypothesis that the outcomes in the first study were chance. In the following six years the number of deaths from these diseases for the control groups was close to national mortality rates, while those for

the veterans groups fell considerably below the numbers to be expected nationally for the extended period, although slightly greater in the earlier period.

402. This data for the extended period was also supported by further studies into the health effects on the LSS group and a group of radiation workers in the USA. Previously different results in the earlier period had influenced the conclusions reached in the first report.

403. The conclusion of the second report was that the previous findings on multiple myeloma were by chance. The position with respect to leukaemia other than CLL was less straightforward:

    (i) There was conclusive evidence that the proportionate increases in risk from whole body exposures to X and gamma rays is substantially greater for leukaemia than other cancers and the proportionate increase is greater in the first 10 years and continues at a lower rate for over 30 years.

    (ii) It was expected that an increase would most likely be revealed by an increased risk of leukaemia in the period of 2 to 25 years after exposure. A slight increase over national levels was found; a greater increase over the controls was observed, and the results of the Pearce report showing a statistically significant increase in risk from leukaemia were noted.

    (iii) However the authors of the second report concluded that this evidence is weaker than might appear for various reasons that they give, and on balance concluded that although the excess was likely to be chance, the possibility that participants did experience some small risk of developing leukaemia after 25 years cannot be ruled out.

404. The third report in 2003, reached similar conclusions to the second report. These conclusions were reviewed in an article published by the nine authors in the Journal of Radiological Protection 2004[110]. This article also summarised the findings from Australia, New Zealand and studies in the USA on a significant number of the estimated 219,000 participants in US nuclear tests.

405. Dr Haylock was one of the authors of the second and third NRPB reports but not the first. He was cross-examined by Mr Ter Haar on the criticisms made of the NRPB

[110] (SB 22/11)

studies by Professor Parker called on behalf of the appellants at the 2013 hearing. In her evidence she had thought that although a smaller study, the Pearce Report was more persuasive about leukaemia outcomes, as New Zealand had a cancer register (from which, data about cancers that had not led to deaths could be analysed) going back to the late 1950s, whilst the UK only maintained from 1971. She also thought that the missing 15% of participants in the UK studies might have skewed the results as they may well have had more serious health issues than the 85% who were studied. She made other criticisms of NRPB methodology and changes in the study group between the first and second study. Her own view was that the case for causation of leukaemia and possibly other cancers was greater than NRPB assessed.

406. Dr Haylock's responses when these matters were put to him seemed to us to be well-informed, well-reasoned and appropriate. He stressed that the essence of an epidemiological study is to eliminate factors that may cause bias. The first and second studies were set up by very distinguished experts in the field. He did not agree with Professor Parker's criticisms because they were only relevant if they applied differently to one group and not the other which was not the case. The results concur with the long term LSS studies and other studies of those exposed to radiation in power stations.

407. However, we are well aware that our task is not simply to express a preference as between competent experts giving suitably cautious opinions on matters within their expertise on which a range of reasonable views can differ. In those circumstances we do not propose to review his evidence on this issue in detail.

Our conclusions on epidemiology

408. In our view, taking what is now known about the outcome of epidemiological studies in the UK and the other national studies noted above, there is a considerable degree of concordance that there is no reliable epidemiological evidence of increased risks of adverse health effects to nuclear test veterans other than for leukaemia (excluding CLL). There is some evidence of increased occurrence for leukaemia (other than CLL): the preponderance of studies consider such a causal nexus unlikely but cannot exclude it as a possibility altogether.

409. The 2004 article states:

> ‘Epidemiological studies are observational rather than experimental in nature and cannot usually give a complete answer to the question whether exposure to a particular agent has caused a disease. They rather show whether disease levels are or are not raised in the exposed population. When increases are found then further considerations are needed to decide whether chance was responsible for the findings or whether some other factor may be correlated with exposures to the agent under study.
> Clearly epidemiology will have a better chance of detecting an increase in levels of disease if the increase is large and if the natural background level is low … there is the potential for chance to play a part in elevating or depressing relative risks where random variation in the number of cases may operate in different directions in participants and controls.
> Except for a few rare diseases, epidemiology cannot usually say anything about the cause of a disease in an individual person. It can only detect statistical effects in groups.’

410. This reflects the consensus of medical opinion as expressed in such bodies as UNSCEAR 2006.

411. We agree with the HL submission that we should be cautious about drawing inferences in support of epidemiological findings based on the recorded evidence of dose at CI. One of the functions of the Tribunal in this appeal is to examine whether the recorded dose can have been reliable having regard to any statistically significant discrepancy in health outcomes for nuclear veterans.

412. However, the present state of the epidemiological evidence taken as a whole, does not suggest that dose at CI might have been considerably greater than that recorded by the AWRE scientists. Still less does it suggest that there is any basis at all for concluding that the dose might have been greater than that assessed by Mr Hallard, who made highly precautionary assumptions about overall deposition of radiation.

413. The one area where epidemiological evidence might have assisted the HL appellants in a War Pensions claim based on reasonable possibility, is if the health condition in question has been a non CLL leukaemia. None of the appeals make a claim for such a condition. Accordingly we are sure that the submissions based on epidemiology do not assist the appellants either on the issue of dose or the possibility of medical causation of their condition. Indeed, on the latter question they support the negative opinions of Professor Thomas and Dr Haylock to be considered in Part Seven.

Professor Mothersill

414. All the appellants rely on the expert report and evidence of Professor Mothersill presented to the previous Tribunal in 2013. Professor Mothersill is a radiobiologist

who is currently a Professor and Tier 1 Canada Research Council Chair at McMaster University having qualified in zoology in 1972 and gained a PhD in 1976. She was a lecturer in medical physics and radiation biology until 1995 and was the scientific director of the radiation and environmental science centre at the Dublin Institute of Technology until 2003. She has been an invited speaker at many conferences including the ECRR conference in Lesvos in 2009 and was one of the signatories to the Lesvos Declaration. She cites her specific interests in basic mechanisms of low dose exposure to radiation on humans and the environment but the majority of her experiments involve animals or fish.

415. She was instructed to prepare a report for the pensions appeal in 2010. That report distinguished between the 'old paradigm' of linear relationship between radiation doses and biological effect and the 'new paradigm'. The proposition she presented to the previous Tribunal is:

> 'our understanding of the biological effects of low dose radiation exposure has undergone a major paradigm shift … this means that previously held views about safe doses or lack of harmful effects cannot be sustained.'

416. Amongst the landmarks that she identified in the process of the establishment of the new paradigm was a paper by Little and others published in 1992[111] on chromosome damage in cells. She cited a number of subsequent papers from 1996 to 2007 (some on the basis of experiments on animals) suggesting a bystander effect: i.e. that cells could receive signals from irradiated cells that resulted in chromosome damage and other effects. She also considered significant Professor Sawada's paper 'Cover up of the effects of internal exposure by residual radiation from the atomic bombing of Hiroshima and Nagasaki' criticising the epidemiology of the LSS studies.

417. Amongst the papers she cites was her own study, Mothersill and Seymour 'Communication of ionising radiation signals – a tale of two fish' June 2009[112]. This paper looked at four different types of fish. The finding was that all types produce signals when irradiated which can be transmitted through water to other fish which cause those fish to produce bystander signals using a reporter cell line. The fish also induced protective proteins in the gills and these proteins are different to the proteins induced in the gills of the directly irradiated fish.

[111] SB 20/19
[112] SB 20/18

418. The conclusion is that:

> 'This mechanism is a universal stress response … which is designed to enable rapid adaptation to changed environmental conditions.
> … Further investigations of these phenomena may reveal the answer to the million dollar question – are these effects intrinsically "good" or "bad"? .do humans emit these signals?'

419. In her report she reached the following conclusions:

> 'Given all the new uncertainties, the LNT model cannot be called an LNT hypothesis anymore. It is clearly not correct to say that a linear extrapolation describes low dose radiation effects. The new paradigm contains complexity and unpredictability. There are arguments and data to support any relationship between dose and effect at low doses but the reality is that any outcome can happen to an individual and there are ample data showing effects at low doses. The possibility that there are no effects at low doses is very remote. The purpose of the LNT model now is to provide a tool for regulation in an environment of uncertainty and on scientific analysis the LNT dose effect relationship has been rejected by various radiological bodies asked to consider the evidence such as the CERRIE minority and majority reports of 2003/4 and the French Academy of Sciences.'

420. Accordingly, having commented on the contrary evidence of Dr Lindahl, she concluded as follows:

> 'my conclusion, given the new developments in radiobiology and the availability of new and sophisticated cytogenetic techniques, is that (the assumed) exposure to ionising radiation during the post war nuclear tests played a causal role in the development (sic) the illnesses which are the subject of these appeals.'

The new paradigm

421. Dr Lindahl was scathing about whether such a new paradigm existed in his response to an original report from Dr Mothersill[113]. We note the following five extracts from his report (where he refers to her as both Dr and Professor):

> 'There are a small number of radiobiologists who think that low dose radiation exposure could be potentially harmful, but again in this case there are no clear facts to support such views. Prof Mothersill writes about "new knowledge" since about 2005 and the "new paradigm". This appears to be based to a significant degree on a publication by herself from 1986 which I have to report had little impact in the radiation biology field although Dr Mothersill herself has published re-reviews of her ideas in 1997 and more extensively in 2004. Bystander effects and related matters have been the subject of discussion and debate amongst experts in the field for may years and new theories emerge all the time. But it is not fair or accurate to claim that there is some new paradigm in the sense that Dr Mothersill has done in her report.'

> 'Dr Mothersill also draws the remarkable conclusion about radiation exposure that "the actual dose received is probably irrelevant". This is based on her own highly unconventional idea that a very low dose might have a disproportionately large

[113] 8 December 2008 SB 12/16

damaging effect …. If this kind of reasoning were accepted or even seriously considered it would make dosimetry and other methods to quantify radiation exposure obsolete. It is not surprising that Dr M's views have not been seriously considered in large reviews in the field such as the recent BEIR VII document. Her idea that "the same doses of radiation could be good for one person and bad for another" is in contrast to the standard radiobiological knowledge and understanding.'

[she] 'discusses the "bystander" effect in an unconventional way. The phenomenon discovered by Prof J Little of Harvard University over ten years ago shows that cells immediately adjacent to a cell irradiated with a microbeam are also somewhat susceptible to radiation damage. Prof. Mothersill has a different unconventional view of the bystander effect and her current work attempts to show that an irradiated cell produces some undefined diffusible substance that can cause genetic damage in other organs. This will only become a credible hypothesis if the "soluble factor" can be found, which has not happened to date.'

'In 2006 she published the first report of "communicated" effects between irradiated and un-irradiated fish. This is again a highly unusual and bizarre view. In connection with conventional radiotherapy of cancer patients there is no cancer centre in the world that believes it necessary to isolate previously treated patients from medical staff and visitors yet the requirement to do so would be the logical conclusion of this hypothesis."

[she concludes that the risk of most of the illnesses identified was materially increased in all 13 veterans cases but this is way out of her area of expertise and such views are] 'inappropriate, misleading and unrealistic'.

422. The BEIR Committee (Health Risks from Exposure to Low Levels of Ionizing Radiation Phase 2)[114] considered the evidence available at that date and rejected the proposition that Professor Mothersill asserts, stating at p. 9:

'Some of the materials the committee reviewed included arguments that low doses of radiation are more harmful than a LNT model of effects would suggest. The BEIR VII committee has concluded that radiation health effects research, taken as a whole, does not support this view. In essence, the committee concludes that the higher the dose, the greater is the risk; the lower the dose, the lower is the likelihood of harm to human health. There are several intuitive ways to think about the reasons for this conclusion. First, any single track of ionizing radiation has the potential to cause cellular damage. However, if only one ionizing particle passes through a cell's DNA, the chances of damage to the cell's DNA are proportionately lower than if there are 10, 100, or 1000 such ionizing particles passing through it. There is no reason to expect a greater effect at lower doses from the physical interaction of the radiation with the cell's DNA.
New evidence from biology suggests that cells do not necessarily have to be hit directly by a radiation track for the cell to be affected. Some speculate that hit cells communicate with non hit cells by chemical signals or other means. To some, this suggests that at very low radiation doses, where all of the cells in the body are not hit, "bystander" cells may be adversely affected, resulting in a greater health effect at low doses than would be predicted by extrapolating the observed response at high doses. Others believe that increased cell death caused by so-called bystander effects might lower the risk of cancer by eliminating cells at risk for cancer from the irradiated cell population. Although additional research on this subject is needed, it is unclear at this

[114] SB 17/2 (undated but by inference 2005)

time whether the bystander effect would have a net positive or net negative effect on the health of an irradiated person.

In sum, the total body of relevant research for the assessment of radiation health effects provides compelling reasons to believe that the risks associated with low doses of low-LET radiation are no greater than expected on the basis of the LNT model.'

423. UNSCEAR reported in 2006 on bystander effects:

'There has been a resurgence of interest in radiation induced bystander effects largely because of the development of single-cell charged-particle irradiators.
The term bystander effect was adopted from the gene therapy literature where it usually refers to the killing of several subpopulations of tumour cells by targeting only 1 type of cell within a heterogeneous population. Bystander effect describes the ability of cells affected by an agent to convey manifestations of damage to other cells not directly targeted by the agent or not necessarily susceptible to it per se. Thus radiation-induced bystander effects are effects manifesting in cells that were non-irradiated neighbours of irradiated cells or that received factors shed or secreted by irradiated cells' (Annex C p.23).

'Mothersill and co-workers showed that repair deficient human cell lines produced a moderate to severe amount of bystander induced cell death … interpreted as supporting the hypothesis that bystander effects play a protective role in biological systems by terminating divisions in cells containing DNA damage' Annex C p33.
"Clearly bystander effects can modify cellular response to radiation and it remains to be determined whether these effects characterised in non- irradiated cells in vitro have a major role in the response of irradiated cells in vitro or in irradiated and non-irradiated cells in vivo).'

'Although it is generally assumed that protraction of radiation dose results in a reduction of effect (DDREF>1) largely as a result of the extra time that protraction allows for cellular repair processes to operate, there are biological mechanisms that could increase the effect when dose is protracted (i.e. DDREF <1). Bystander effect implies that the linear extrapolation from high dose exposures would lead to substantial underestimates of effects at low doses but the work from Little and Wakeford implies that low dose rate lung cancer risks associated with alpha particle exposure are not seriously underestimated by extrapolation' (Annex A p 127).

'Significantly these bystander effects appear to be limited to the organ irradiated, ie are organ specific. Thus at the present state of our knowledge it is reasonable to assume that any bystander effect in vivo is accounted for in models of organ risk evaluation. As a result it is unlikely that the resurgence of interest in these non-targeted radiation effects will substantially alter risk estimates as discussed in detail in the BEIR VII report. Nevertheless it cannot be excluded that increasing the knowledge basis for in vivo bystander effects at low doses and low dose rates in specific organs may affect current organ estimates.' (our emphasis)

424. UNSCEAR concludes (at paragraphs 160-164 of Annex C).

'In spite of the large body of new information, considerable disagreement remains concerning any definitive relationship between these non-targeted effects and the observed health effects attributable to radiation. The Committee stresses that direct epidemiological observations and associated quantification of the health effects of

radiation incorporate all mechanistic elements including the targeted (direct) effects of irradiation as well as the non- targeted and delayed effects.

A specific role for non-targeted effects in the observed health effects associated with radiation exposure cannot be determined directly. Such effects can provide mechanistic information at doses of below about 200mGy that could be pertinent to evaluating health effects at these low doses. However in ascribing a mechanism to a particular biological effect the data in question should be independently replicated and show a strong coherence with the particular end point considered.

In light of these considerations, the overall view of the Committee is that the data currently available do not require changes in radiation risk co-efficients for cancer and hereditary effects of radiation in humans'. (our emphasis)

425. In addition, we were supplied with a 2012 paper by Mancuso et al[115] which summarises the current position:

> 'In truth, without appropriate in vivo models, the significance of these indirect effects on human health remains limited'

426. It is alarming that there was no reference to the opinions of BEIR VII and UNSCEAR, in Professor Mothersill's 2010 report that was before the Tribunal. Whatever the strength of her own views, for reasons already stated, an expert who complies with the common law requirements for receipt of such materials reflected in CPR 35 would be bound to draw attention to a responsible expression of contrary opinion on the very topic to which her report was directed. We are of the view that if Mr Ter Haar had specifically sought a direction for the 2010 report to be admitted as expert evidence into this appeal, he would have encountered considerable difficulty in persuading us to do so, applying the approach we have directed is appropriate for these appeals. In brief:

(i) She has not referred to or discussed a range of contrary views.

(ii) She seems to step well beyond the range of her expertise as a radiobiologist with experience in experiments on fish to give an opinion on health effects in humans. An example of this is her comments on Mr Sinfield who has a diagnosis of ALCL and his health records refer to 'many years of a chronic anaemia' which Professor Mothersill attributes incorrectly to being caused by bone marrow damage due to irradiation but in fact was a classic hypochromic anaemia due to iron deficiency caused by a hiatus hernia and acid reflux, as was demonstrated on endoscopy.

[115] The Radiation Bystander Effect and its potential implications for human health' SB 20/17

(iii) She has every appearance of having a partisan view point as exemplified by her signature of the Lesvos Declaration, her selective citation and her misreading of the AWE reports on Maralinga to comment:

> 'the military were interested not only in the harmful effects of radiation but how long the soldiers could remain operational once exposed to radiation that would ultimately lead to harmful effects.'

427. Despite these failings we have not excluded this evidence from consideration but gone on to see if it may carry any weight on the issues before us.

428. We have reviewed the transcript of her cross-examination on 7 February 2013 with some interest. When the BEIR VII conclusions were put to her, her observation was that they were 'dinosaurs' and out of date but predicted they would change their views. She similarly thought that UNSCEAR 2006 was highly conservative, and a paper by Brenner and Doll amongst others in 2003 was out of date. However, her report indicated that the new paradigm had been established as the accepted wisdom by 2007, and looking at the range of opinion as of that date, we are quite clear that it had not.

429. It was put to her that she had no expertise in human health, and she agreed that her expertise was in radiobiology. She could not comment on what the outcome of the mechanism that she was certain was in play would be. This led to an adjournment while matters were discussed between counsel and, in the light of a subsequent statement modifying her ability to comment on specific health outcomes, no further questions were asked of her by the Secretary of State.

430. The Tribunal had their own questions of her, and the medical member, Dr Anscombe, asked a series of questions on her evidence and the information to be derived from the references cited, and the meaning of high and low dose. It concluded that her references were misleading in citing doses of 250 mSv and above (up to 5 Sv) as 'low doses'.

431. We have been provided with a copy of the Secretary of State's closing submissions in 2013 on Professor Mothersill's evidence and note they included the submissions:

> 'She provides no evidence let alone reliable evidence of causation. It does not go towards raising a reasonable doubt of causation, her evidence says nothing about causation of the disablements suffered by these Appellants at all.'

After noting the misleading use of the references cited the submission concluded:

> 'This reinforces the fact that Professor Mothersill's evidence has very little to do with low doses which are at the heart of these appeals and further renders her evidence irrelevant.'

432. We asked for a copy of these submissions in order to evaluate a contention by the HL appellants that the defendant had not made clear what his response was to Professor Mothersill's evidence. It seems to us that the approach was made very clear in 2013 and nothing that has emerged in the subsequent history of the case or the more recent literature reviews has served to change this picture all.

433. We note that Professor Little and others produced a paper for the National Institute of Health on 'Evidence relevant to untargeted and transgenerational effects in the offspring of irradiated parents', on a different but related topic[116]. We see no evidence in that paper of any new paradigm yet we understand that Little's 1992 paper was an important document relied on by Professor Mothersill in 2010 for the recognition of the new paradigm. We have already noted the paper from Mobbs and others on behalf of the HPA 'Risks from Ionising Radiation' reviewing the literature on risks to workers and civilians exposed to radiation from power plant emissions, that again does not suggest that there is any new paradigm.

434. It was always open to Mr Ter Haar to seek to call Professor Mothersill before us, and address the criticisms and concerns expressed following her evidence in 2013. It is quite understandable why he did not. He did not canvas Professor Mothersill's views with Professor Thomas, as might have been appropriate, but put some of them to Dr Haylock. When matters were within his expertise he disagreed with what she had said.

435. In the light of all of the above, it is in the very least 'ambitious' for the HL appellants to invite this Tribunal to receive Professor Mothersill's report as reliable evidence that creates a doubt as to causation. This is particularly so as her connections to ECRR were not a feature of the previous criticisms of her, but are so today in the light of Dr Busby's participation as advocate in this case and the witnesses he has called. We have made our conclusions on the views of the ECRR group clear in Part Four of this determination.

[116] Other papers of Professor Little were cited by Professor Thomas in her anthology of supporting data SB4/22 and 23

436. It is sufficient to address the HL submission to say that we have spent some time examining Professor Mothersill's evidence and the surrounding science she cites. We conclude that what she has done in her report is to describe a whole series of reported observations without any evidence as to the health outcomes of these observations.

437. We have no doubt that her 2013 evidence is at odds with main stream opinion, is materially overstated, unsupported and makes misleading reliance on the work of others. She sought to address issues on which she has no expertise and for which she could provide no reliable supporting science. She subsequently, albeit belatedly, recognised this fact. Dr Lindahl's critical observations in 2008 have been supported by the subsequent papers discussed above. We acknowledge that she has expertise in her specialist field, but on the issues before us, we are sure that she has not provided plausible evidence capable of raising any doubt at all, let alone a real one. This is not a question of preferring one expert view to another but dismissing from further consideration as irrelevant a view that we conclude is not an expression of relevant expertise at all.

SECTION SEVEN

THE MEDICAL CONDITIONS OF THE VETERANS

Introduction

438. In this section of our determination, we will consider whether any of the health outcomes which these veterans have encountered is considered to be radiogenic.

439. In considering this issue we remind ourselves of some of the relevant science, briefly summarised by way of introduction in section two of this determination.

440. Ionising radiations differ in the way they react with biological materials and cause damage so that equal absorbed doses (i.e. equal amounts of energy deposited) do not necessarily have equal biological effects. Radiations may have a low rate of loss of energy per unit track length and be termed low linear energy transfer (LET) eg X rays, gamma rays or beta particles or they may have a high rate of loss of energy and be termed high linear energy transfer as for alpha particles or neutrons. The biological effects of high LET radiations are in general much greater than those of low LET radiations with the same energy. This is because high LET radiation can deposit most of its energy within the volume of one cell of the body and the chance

of damage to the cell DNA is therefore larger[117]. Hence the use of the radiation weighting factor to give an 'equivalent dose' measured in Sv.

441. The atomic bombings in Japan led to a mean effective dose of 200 mSv (Brenner et al 2003). The Chernobyl nuclear plant incident led to mean cumulative whole body doses of 100 mSv for emergency workers (liquidators), between 10 and 50 mSv for evacuees and residents of strict control zones and 7 mSv for other people living in contaminated areas[118].

442. Although radiation exposure can increase the incidence of cancer in exposed populations there is at present no way of distinguishing the cases caused by radiation exposure from those resulting from other causes. The identification of radiation-induced cancers in a population can therefore only be determined by comparing populations with different exposures. Brenner et al (2003) considered radiation exposures at which a statistically significant increase in cancer risk could be observed. The HPA review of epidemiological data suggested that the lowest dose at which good evidence of an increased risk could be obtained was around 10–50 mSv for a single acute exposure and around 50–100 mSv for a protracted (chronic) exposure[119]. It also provides a good summary of the main epidemiological studies (based on UNSCEAR) (p24–90).

443. Professor Thomas[120] cites a number of large cohort studies involving both acute and protracted radiation exposures that confirm the data from the lower range of the LSS study. These include the National Registry of Radiation Workers (NRRW), a study of UK nuclear workers; the Techa River residents who were exposed to discharges of radioactive waste into the river near which they lived; the cohorts of workers who cleaned up after the Chernobyl accident and also data from Yangjiang, an area of high natural background radiation in China and from the workers at British Nuclear Fuels Limited. She states that it is to be noted that the majority of the estimates of excess relative risk lie close to 0, particularly in the dose range between 0 and 0.1 Sv, and in the majority of cases the 95% confidence intervals (where given) span 0.

[117] Risk of solid cancers following radiation exposure: estimates for the UK population. Report of the independent advisory group on ionising radiation 2011 p5
[118] Circulatory Disease Risk Report of the Independent Advisory Group on Ionising radiation HPA 2010 p13
[119] Ibid p9
[120] Prof Thomas report 17.12.15

This indicates that there is no statistical evidence that an effect of radiation at these levels is proven scientifically but rather could be a chance finding.

444. We now examine the claim of each veteran, their relevant medical history and the nature of the condition claimed. As this part of the decision is a review of medical conditions and what may cause them, we have varied the order of the appellants from that set out at [11] in turn reflecting the order in which they appear in the proceedings.

Leonard Coulson Abdale
History

445. The late Leonard Coulson Abdale was born on 3rd February 1935. He submitted a claim form dated 16 May 2009 for transurethral resection of bladder cancer. On 26 June 2009 he wrote to the Service Personnel and Veterans Agency (SPVA) advising that he wished to claim for cataracts in both eyes. He attributed both these conditions to his service on Christmas Island (CI). A certificate was signed by a SPVA Medical Adviser on 21 October 2009 (Certificate Refused) dismissing the claims for the conditions: Cataracts (both eyes) and Transitional Cell Carcinoma of the Bladder.

446. He served in the RAF from 13 May 1953 until 1 February 1976. He served on CI from 15.1.58 to 29.11.58 and was therefore present for Grapple Y and Grapple Z 1, 2, 3 and 4 tests. During this time he was a Senior Aircraftman (SAC) in the trade of a Wireless Operator. He was based at Main Camp. The AWE report of 2009 records that no dosimetry record is held in respect of him.

447. His past medical history included various fractures between 1957 and 1978 and there is reference to his developing a basal cell carcinoma in 2004 below his left ear when he was described as a 'sun worshipper'. He has had gout and a rotator cuff tear.

448. He had an eye examination on 4 April 1960 when he was referred to the ophthalmologist for refraction when his visual acuity was 6/9 in the right eye and 6/18 in the left eye both corrected to 6/6 with glasses. At his release medical on 8 October 1975 a slight deterioration in his eyesight was noted with a visual acuity reading of right eye 6/6 and left one 6/24. There was no diagnosis of cataract in either eye.

449. According to the GP print out of his medical conditions, a lens opacity is noted in his right eye in 1999. At the war pensions medical examination (WPME) on 24 August 2009 his visual acuity was 6/9 using both eyes without correction[121]. He stated he was a non smoker. There is a record of an examination in 2004 following a six month history of reduced vision in the left eye. The examination documents bilateral pseudophakia (the presence of an artificial intraocular lens) due to previous cataract surgery in 2000 and 2001 and early Fuch's (corneal endothelial dystrophy) which had been noted in 2000.

450. He had haematuria thought to be mild haemorrhagic cystitis in 1955 and had urethritis in 1955.

451. His GP reports his bladder cancer was diagnosed on 27 June 2006 when he was aged seventy one years old. He had a cystoscopy to remove the tumours followed by chemotherapy. A recurrence occurred in 2009 when more tumours were removed.

Cataracts

452. The lens is an optically clear, avascular tissue that receives nourishment from its surrounding aqueous and vitreous fluids. The main pathology of the lens is its opacification, termed cataracts in its advanced stages. Cataracts can be classified according to the part of the lens in which they form[122]. The predominant forms of cataract depending on their anatomical locations in the lens are capsular and sub-capsular cataract (can be anterior or posterior), nuclear and cortical.

453. The position of the cataract can provide an indication of the aetiology, for example nuclear sclerosis is usually associated with age or diabetes and there is little evidence that such cataracts are radiogenic. The general consensus is that cortical cataracts are age-related or congenital.

454. The evidence before the Tribunal is that cataracts have been shown to be radiogenic, as the lens is a relatively radiosensitive tissue in adults[123]. Radiation doses of 1 Gy or more are associated with an increased risk of posterior sub-capsular cataracts and accumulating evidence from the Japanese atomic bomb survivors, Chernobyl

[121] FTT Part A Abdale Doc 106
[122] Synopsis of Causation: Cataracts April 2010
[123] Mark P Little. A Review of non-cancer effects, especially circulatory and ocular diseases Radiat Environ Biophys 2013

liquidators, US astronauts and various other groups suggest that cortical cataracts may also be associated with ionising radiation.

455. The latency period between irradiation and the appearance of lens opacities is uncertain. Early studies among atomic bomb survivors exposed to 1 Gy or more showed an approximate average latency period for the development of lens opacities of 2–3 years depending on the dose to the eye. According to the Carter report:

> 'opacities have not been seen at doses below approximately 0.5 Sv and are only severe enough to affect vision at doses above approximately 5 Sv.'

456. In recent years there has been a downward revision of the safe dose limit of exposure of the eye by the ICRP. The 2007 ICRP report noted that new data on the radio sensitivity of the eye with regard to visual impairment are expected and concluded:

> "because of the uncertainty concerning this risk, there should be particular emphasis on optimisation in situation of exposure to the eyes"

457. The values were changed in 2013 as a result of a number of reports indicating prevalence of opacities in the eyes of staff exposed to radiation levels below the thresholds as established by the ICRP[124] and the threshold in absorbed dose for the lens of the eye is now considered to be 0.5 Gy.

458. Further, for occupational exposure in planned exposure situations, the Commission now recommends an equivalent dose limit for the lens of the eye of 20 mSv in a year, averaged over defined periods of five years, with no single year exceeding 50 mSv. For public exposure, the values are unchanged from those recommended in ICRP 103 in 2007; i.e. 15 mSv in one year. The HPA response to the ICRP recommendations also mentions that it is possible that cataract induction is a stochastic process and that a threshold does not apply. Accordingly the limit is now 20 mSv with a maximum annual dose of 50 mSv.

459. In the case of cataracts, the equivalent dose to the skin is taken as representative of a dose to the lens of the eye, and the dose is therefore an external rather than an internal one. Mr Hallard's revised estimate for the external dose to Mr Abdale during

[124] ICRP Publication 60 of 1990 and ICRP Publication 103 of 2007

the 10 months of his service on CI was <120 mSv, substantially in excess of the revised annual limit.

460. We note that the development of cataracts is a condition common in elderly people and Mr Abdale was 65 years old when he was diagnosed with them. We were not shown any evidence as to the specific type of cataracts that Mr Abdale developed.

461. Neither Dr Haylock nor Professor Thomas specifically addressed the issue of causation of Mr Abdale's cataracts. Dr Thomas seems to have confined her consideration to the claim for cancer based on internal dose.

462. Dr Haylock did perform a probability of causation for Mr Shaw, who also has a claim for cataracts. Dr Haylock states that ICRP specifies a value of 0.5 Gy as the absorbed dose threshold for the induction of radiation induced cataracts. He uses the upper limit for the equivalent skin dose as a more appropriate dose estimate to use for eye exposure than the effective dose, but also assumes that cataract induction is a stochastic effect and so uses the assumption of a linear no threshold excess relative risk model with a doubling dose of 2 Gy, as suggested in the HPA response to ICRP 118, to perform the calculation.

463. In his written submissions, Mr. Heppinstall recognised that Mr Abdale had a claim for cataracts but referred us to the calculations for Mr Shaw in inviting us to dismiss it. However, Mr Hallard's estimates for external dose for Mr Shaw were very different to those for Mr Abdale.

Bladder cancer

464. Bladder cancer is a common urologic cancer: in the UK there are 12,700 new cases per year. It is the fourth most common malignancy in Caucasian men and most commonly comes to medical attention in the 6th and 7th decades of life[125]. Almost all bladder cancers are transitional cell carcinomas. It is one of the best examples of a human cancer linked to environmental carcinogens such as aniline dyes and petroleum products. Cigarette smoking is a leading cause of bladder cancer. In western countries approximately 50% of the cancers in men and 30% in women would be attributable to smoking.

[125] Synopsis of causation: Cancer of the Bladder September 2008

465. Bladder cancer can be caused by ionising radiation and is known to occur in patients who have had radiotherapy for cancer of the cervix or testicular cancer.

466. The results from the LSS demonstrate that there is an increased risk of developing bladder cancer in the survivors of the atomic bombs. The most recent LSS report stated that 150 bladder cancers occurred between 1950 and 1997. Of these 99 occurred among survivors exposed to 5 Sv or more of which about 16% would be attributable to radiation exposure.

467. UNSCEAR in 2006 state:

> 'updated mortality information from studies of the survivors of the atomic bombings continue to demonstrate a positive radiation response for bladder cancer. In the aggregate, studies of cancer patients treated with high dose radiotherapy also demonstrate an association between radiation exposure and risk of bladder cancer. Studies of nuclear workers do not provide evidence of a radiation–related bladder cancer risk but, because the radiation exposure of these workers was low, the statistical power of the studies is quite limited.'

468. The HPA report states that there is convincing evidence of a relation between low LET radiation exposure and bladder cancer risk based on the LSS incidence and mortality data as well as on studies of several populations medically exposed to radiation for benign diseases. The risk of bladder cancer associated with exposure to high LET radiation is unclear. In general no risk was seen among patients exposed to thorotrast as a contrast medium or in a Finnish study of patients exposed to dissolved radioactive material.

469. Professor Thomas states that the most likely exposure of the bladder epithelium to radiation is from exposure to radioisotopes dissolved in the urine, therefore the internal dose would be critical in assessing the likelihood of a radiogenic aetiology for his cancer.

470. Mr Hallard's revised calculations estimated that Mr Abdale's internal dose due to service on CI was <2 mSv and he would have been exposed to 2 mSv per year on average from background radiation. By the time of his diagnosis at the age of 71 this would be a total exposure of about 142 mSv from background radiation.

471. Dr Haylock addresses probability of causation for Mr Abdale. He states in his report that bladder cancer has been identified by ICRP in its most recent recommendations (2007) as a cancer for which there is sufficient evidence from the LSS study to justify the publishing of specific risk models. The UK Advisory Group on Ionising

Radiation (AGIR) also identifies bladder cancer as a radiation inducible cancer and endorses the models proposed by ICRP as the best available for estimating risk to a UK population. Dr Haylock therefore uses the ICRP models in the risk calculations.

472. For Mr Abdale he assumed that the internal and external dose was received by the bladder at the same time, as this assumption results in the highest probability of causation compared to an assumption that the internal dose was delivered over a long time period and a committed effective dose of 4mSv is applied. The resulting probability of causation is 0.08% chance of his bladder cancer being caused by his exposure to ionising radiation whilst serving on CI. The epidemiological evidence therefore predicts that there is a greater than 99.9% chance that Mr Abdale's bladder cancer was not caused by the radiation exposure from the nuclear tests.

Conclusions

473. The Tribunal find that both cataracts and bladder cancer can be radiogenic but whether exposure to ionising radiation causes such conditions is dose dependent.

474. Mr Hallard has calculated the external equivalent skin dose for Mr Abdale to be up to 120 mSv and, although this is a conservative calculation and there was a long period after service before the development of the cataracts, taking this data together we are satisfied that, if he might plausibly have been exposed to such a dose, we cannot exclude the reasonable possibility that some of his cataracts were caused by it. Where current guidance identifies risks from radiation at doses in excess of 50 mSv in one year or chronic exposure in excess of the current ICRP limit, we accept that this raises a reasonable doubt as to causation of this condition.

475. By contrast, as regards the causation of bladder cancer however, we are satisfied that Mr Hallard's conservative estimate of dose generates such a statistically marginal possibility of causation as to raise no reasonable doubt because of the extremely low internal dose.

Darryl Beeton

476. Darryl Beeton was born on 9 August 1937. He submitted a claim form dated 21 July 2009 for heart disease and a triple heart bypass, difficulty breathing and walking. He attributed his heart problems to his service on Christmas Island (CI). A Certificate Refused for the conditions Atherosclerosis and Myocardial Infarction (2001) was

given by the SPVA and this decision was notified to him by letter on 25 November 2009.

477. Mr Beeton served in the RAF between 1956 and 1959. He served on CI from 27 August 1957 to 10 August 1958 and was therefore present for Grapple X and Y. During this time he was a Leading Aircraftman (LAC) in the RAF employed as a cook. He worked in the kitchens at Port Camp and later transferred to Main Camp.

478. His GP records state that he had myocardial infarctions in 2001 and 2003 and a coronary heart bypass on 7 July 2008. He had been referred in 2002 for worsening angina and had undergone PCI (percutaneous coronary intervention) in September 2002 but this was unsuccessful. He was found to have severe three-vessel disease comprising severe left anterior descending stenosis, subtotal Right coronary occlusion and significant marginal circumflex artery disease[126]. He was referred for coronary bypass surgery but Mr Beeton decided to delay surgery because he was then asymptomatic. A consultant cardiac surgeon gives a diagnosis of ischaemic heart disease on 16 July 2008[127].

479. The SPVA Medical Adviser notes the underlying disease process is atherosclerosis. He was 64 years old at the time of presentation. He has a history of raised cholesterol treated with statins.

Atherosclerosis and cardiovascular disease

480. The evidence before us showed that, in addition to cancer, radiation exposure has been demonstrated to increase the risk of other diseases, particularly cardiovascular disease, in persons exposed to high therapeutic doses and also in A-bomb survivors exposed to more modest doses. However, there is no direct evidence of increased risk of non-cancer diseases at low doses, and data are inadequate to quantify this risk if it exists.

481. There is a well-established association between high doses (>5 Gy) of ionising radiation exposure and damage to the heart, coronary, carotid and other large arteries (for example in the radiotherapy treatment of Hodgkin's disease where doses to the heart can exceed 40 Gy). The association between lower dose exposures and late

[126]FTT Part A Beeton Doc 65 dated 23 September 2002
[127] FTT Part A Beeton Doc 73

occurring circulatory disease has only recently begun to emerge in the Japanese atomic bomb survivors and in various occupationally exposed cohorts and is still controversial. Excess relative risks per unit dose in moderate and low dose epidemiological studies are somewhat variable, possibly a result of confounding and effect modification by well known (but unobserved) risk factors.

482. The current ICRP Publication 118 (2012)[128] states:

> 'from current evidence a judgement can be made of a threshold acute dose of approx 0.5Gy or 500mSv for both cardiovascular and cerebrovascular disease. On that basis 0.5Gy may lead to approximately 1% of exposed individuals developing the disease in question >10 years after exposure. This is in addition to the high natural incidence rate (circulatory diseases account for 30-50% of all deaths in most developed countries). The value of 0.5Gy could be reached during some complex interventional procedures.'

483. We conclude that such a condition can be the product of radiation exposure at doses of 500 mSv or more.

484. Mr Hallard calculates that Mr Beeton received an external (effective) dose of <1 mSv, an external (equivalent) skin dose of <44 mSv and an internal dose of <1 mSv. These doses are so small that Dr Haylock was unable to perform a probability of causation calculation.

485. Professor Thomas states that there is evidence of cardiac disease as a result of high dose radiation but the effects are seen more commonly in those irradiated as children and the doses of radiation are in the order of 14 Gy. The most recent data from the LSS study indicates no risk for cardiovascular disease can be determined in those that received doses of <500 mGy. She also notes that the cardiologist (Dr Hayward) explains that Mr Beeton had significant risk factors for his heart disease (i.e. hyperlipidaemia and hypertension) and Mr Beeton confirmed at the FTT hearing that he was taking statins.

486. At the time of his diagnosis he was 64 years old and his lifetime dose from background radiation would have been approximately 128 mSv.

Conclusion

487. In the light of the above we are certain that ionising radiation was not the cause of Mr Beeton's cardiovascular disease.

[128] SB 20 tab 12 p21

Barry John Smith

488. Mr. Smith was born on 4 May 1939 and died on 20 February 2009. He submitted a claim form dated 5 February 2008 for pancreatic cancer which he attributed to his service on Christmas Island (CI). A Certificate Refused for this condition was signed by a SPVA Medical Adviser on 7 May 2008. Mr Smith appealed this decision. He died before the appeal was heard by a tribunal and his widow, Mrs Anna Smith, confirmed she wished to continue with her late husband's appeal. She also submitted a claim for a war widows pension and funeral expenses on 19 June 2009 attributing her husband's pancreatic cancer to CI service. The Certificate Refused (Widow) was signed by a SPVA Medical Adviser on 30 July 2009.

489. Mr Smith served in the RAF from July 1958 to July 1961. He was stationed on CI from 30 October 1959 to 1 November 1960 in the role of catering assistant. From 28 July to 2 August 1960 he was at the US base at Hickcam. Therefore he was not present for any of the tests. From Mrs Smith's witness statement it is noted that amongst his other duties he was the camp barber.

490. Mr Smith died on 20 February 2009 aged 69. The cause of his death was recorded as I(a) Adenocarcinoma of Head of Pancreas. This condition was considered by Secretary of State in the rejection of both claims.

491. Mr Smith had applied for a War Disablement Pension in 2002. He had keratoses on his legs and forearms and stated that a dermatologist had told him that his skin was 'cooked' and 'irradiated'. He also claimed for Ménières disease. He smoked for one year aged 17 but had then given up. He also stated that:

> 'on Christmas Island we were sprayed twice a day for 13 months by a low flying crop sprayer with aviation fuel, DDT, tri-orthocresol in mixture to kill flies and mosquitos.'

However, no submission has been advanced or is evidentially supported before us as to a possible link between this spraying, if it occurred as claimed, and the health conditions in the present claim.

492. Mr Smith's post-service medical records show that he was first seen in the rapid access jaundice clinic on 17 October 2007, reporting that he had noticed his jaundice about a week ago and it was associated with upper abdomen cramp-like pain. It was noted he was a non smoker and did not drink alcohol. An ultrasound scan revealed a distended gall bladder, no obvious gall stones and no obvious pancreatic lesion. He

had a CT scan in October 2007 which showed mild intrahepatic biliary dilatation along with a dilated common bile duct which tapered at the lower end and absence of any stones in the common bile duct or gall bladder. It also confirmed that his pancreas was very atrophic with a cyst in the tail and some changes in the head of the pancreas suggestive of chronic pancreatitis. He was advised to stop taking bendrofluazide which can sometimes cause idiopathic pancreatitis. After further tests he was diagnosed with carcinoma of the head of the pancreas which was locally advanced (T3 N0) and underwent a pancreaticoduodenectomy in December 2007 followed by chemotherapy. He was 68 at the date of diagnosis.

Pancreatic cancer

493. Around 8,800 people in the UK get pancreatic cancer each year. It is the 10th most common cancer, excluding non melanoma skin cancer. It is more common in older people. Almost half of all new cases are diagnosed in people aged 75 and over. Pancreatic cancer is uncommon in people under 40 years old[129]. The incidence of this type of cancer rises with age and as we have noted Mr Smith was 68 when diagnosed.

494. In the US pancreatic cancer is the fourth leading cause of cancer deaths, being responsible for 7% of all cancer-related deaths in both men and women.

495. Risk factors include smoking, chronic pancreatitis and a prior history of diabetes mellitus. There may also be a genetic component in up to 1 in 10 cases of pancreatic cancer (10%).

496. The synopsis of causation for cancer of the pancreas states that the only study linking ionising radiation and cancer of the pancreas is a 1965 study from Tayside looking at a cohort of patients who had received irradiation of the spine for ankylosing spondylitis. Although the treatment dose was not detailed in the paper, the Tayside region at the time was using doses of between 150 and 180 centigrays given in 10 fractions[130].

497. The most informative epidemiological studies are the LSS of the Japanese atomic bomb survivors and the study of females treated with radiotherapy for cervical

[129] Cancer research UK
[130] Synopsis of causation. Cancer of the pancreas September 2008.

cancer. These and the combined study of nuclear workers are the only studies with more than 100 cases or deaths; most of the remaining studies have low precision[131].

498. Based on these studies, it is unclear whether there is an association between radiation exposure and pancreatic cancer. Most of the studies that suggest a possible association have low precision. Several of the larger studies including the study of patients with cervical cancer and the mortality data from the LSS suggest that there may be no association with radiation exposure.

499. Pancreatic cancer is difficult to diagnose accurately and the histological verification of cases in the LSS was amongst the lowest of any cancer site. Misclassification of outcome could be an explanation for the lack of dose response.

500. Two studies of radiotherapy patients in which the pancreas received a very high radiation dose have found significantly elevated risks of pancreatic cancer although the studies lacked individual dose assessments[132].

501. The conclusion of the 2011 AGIR paper was:

> 'It is unclear whether cancers of the pancreas, connective tissue, melanoma of skin … can be induced by radiation. In general, the data for these cancers are too sparse to assess consistency across studies and the possibility of a small raised risk cannot be ruled out.[133]'

502. When asked about this at the hearing, Dr Haylock told us 'I don't believe it is radiogenic'[134] and as pancreatic cancer is not radiogenic he was unable to perform a probability of causation calculation.

503. Prof Alastair Forbes (Professor of Gastroenterology) in a medico legal report of 26 February 2011[135] concerned with the death of Barry Smith notes that an association of ionising radiation with pancreatic cancer is generally thought to be absent or very weak and that this cancer is not included in many lists of radiation-induced tumours. He refers to the LSS which recorded three excess pancreatic cancers in the 93,000 survivors which is not statistically significant.

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[131] AGIR 2011 (HPA) Risk of solid cancers following radiation exposure SB5/42
[132] ibid
[133] AGIR. 2011. (HPA) Risk of solid cancers following radiation exposure. SB 5 /42 p191
[134] TS day 9
[135] SB 13 TAB 26

504. Professor Thomas states that there is no scientific study that has been able to demonstrate a causal relationship between radiation and adenocarcinoma of the pancreas[136].

505. The risk of developing carcinoma of the pancreas increases with the development of diabetes mellitus and smoking but there was no evidence of this in the case of Mr Smith. There was however evidence of chronic pancreatitis which is a risk factor.

506. We are aware that in the case of Mr Battersby, he was also diagnosed with pancreatic cancer and that the Secretary of State acceded to a claim to war pension on the basis of another tribunal ruling that was not appealed. We do not take this previous finding into account one way or another. It stands in his case, although we understand that the respondent would submit that in the light of current information it would not follow if there was a re-assessment of this issue. It does not amount to evidence that the disease is radiogenic generally.

507. In our assessment, a fair summary of the data is that evidence of an association between radiation exposure and pancreatic cancer is weak or non-existent. The only reliable evidence before the Tribunal is that large doses of external radiation can cause diabetes and that is not the case here. We were not taken to any reliable evidence that internal exposure either by ingestion or inhalation can cause carcinoma of the pancreas.

508. Dr Busby placed particular emphasis on the fact that four of the original group of appellants had developed pancreatic cancer as evidence that their military service and exposure to radiation during it was a causative factor and asked Dr Haylock to perform a calculation on the probability of that occurring. Dr Haylock considered that to be a meaningless statement from the point of view of epidemiology as such a cohort was biased from the outset (as they were all known to Dr Busby and were all part of the same organisation) and therefore not representative of the group as a whole.

509. Dr Haylock had however done a calculation of the probability of developing pancreatic cancer in a population of people born in 1939, alive in 1959 and who were now 70 years old. He calculated that 0.5% will die from pancreatic cancer and so, in

[136] SB2 2.18

a group of 20,000 test veterans, one would expect 100 cases of pancreatic cancer irrespective of radiation exposures. In 1998 there were 77 such cases. The fact that four such people made war pensions claims tells us nothing about causative possibility.

Conclusions

510. Mr Hallard assessed that Mr Smith had no exposure to external radiation by release of his service dates on CI and assigned only 1 mSv for internal dose. Whatever room for scientific debate there may be about whether radiation can cause pancreatic cancer, we are sure that there is no possibility that this condition was caused by Mr Smith's military service.

Donald Battersby

511. Donald Battersby was born on 3 January 1936. He submitted a claim form dated 17 August 2009 for chronic lymphatic leukaemia (CLL) which he believed was due to his service at Maralinga at the time of the nuclear weapons tests. A Certificate Refused for this condition was signed by a SPVA Medical Adviser on 15 October 2009.

512. He served in the RAF from 1954 to 1959 and was stationed at Maralinga from 26 July and 19 November 1956 and therefore was present for the four tests in the Buffalo series. During this time he was a SAC employed as an airframe mechanic working at the airfield and accommodated in Maralinga Village.

513. His medical records show he was referred to a specialist in Haematology who noted in September 2009 that Mr Battersby had been referred to the same Department in 2005 following a diagnosis of CLL[137]. In an undated letter of around January 2013 (Doc 79) Mr Battersby says he had a stroke in January 2005 as result of which doctors found he had CLL. Also he states he had been diagnosed with skin cancer and that there was no history of CLL in his family. He was 69 at the time of diagnosis.

[137] FTT Part A Battersby Doc 47

Chronic Lymphocytic Lymphoma (CLL)

514. Chronic lymphocytic leukaemia/small lymphocytic lymphoma (CLL) is the most prevalent lymphoid neoplasm in Europe and North America. It represents 25% of all leukaemias and 40% of all leukaemias in adults aged up to 50 years. CLL is very rare in patients under the age of 30, and the median age at diagnosis is about 72 years with a 1.5 to 2:1 male to female predominance. The 'cell of origin' is a mature B lymphocyte that has a rearranged immunoglobulin gene.[138]

515. The aetiology of CLL remains essentially unknown. Although ionising radiation has been implicated in most leukaemias, it has not been established as a risk factor in CLL. Genetic predisposition plays a major role in the development of the disease. Epidemiological surveys show a seven fold increase of the disease in the relatives of patients with CLL. Research at the Institute of Cancer Research in London has now demonstrated 10 predisposition gene loci as the basis for this high familial predisposition[139]. Additional evidence for a genetic predisposition for CLL is the marked ethnic variation in the incidence of the disease, which remains relatively unchanged after large population migrations. The highest incidence rates of CLL are in patients of European descent, with a substantially lower risk in people of South East Asian ancestry. In most patients with access to modern medical care, CLL is an incidental diagnosis made during investigation of leucocytosis and lymphocytosis, and these patients usually have early-stage asymptomatic disease.

516. Most patients will die of the disease or its complications. CLL is likely to decrease the overall survival of all patients who have the disease, and this also applies even to older patients with early-stage disease. CLL has always been considered a malignancy of mature B cells and therefore grouped with the B cell lymphomas. There is no single mention in the description of the 35 diseases included in the mature B cell malignancies to radiation involved in causation. The only mention of radiation as causative agent is in the description of the myeloid leukaemias which are a quite different group of diseases.

517. Dr Busby relies on a change in practice in 2012 in the US by the authority responsible for listing the diseases for which compensation is payable for those

[138] Oxford Textbook of medicine 5th Ed
[139] Prof Daniel Catovsky 16/2/11 SB 13 TAB 25

exposed to radiation NIOSH-IREP. He suggests that this indicates that the US now accepts that CLL is radiogenic.

518. The reason for and the evidential basis of the US decision was explored before us in the cross examination and re-examination of Dr Haylock. The epidemiological studies on CLL are described as non-determinative in the US reports. Five experts were consulted, two of whom concluded that there was no evidence that CLL was radiogenic, but the others concluded that the evidence was insufficient to rule out that possibility and/or that exclusion of CLL might be considered arbitrary. In the event, a quantitative radiation risk model for CLL was developed, including a major modification to the original risk model of the shortening of the midpoint of the latency period for CLL from fifteen to ten years while maintaining the uncertainty in the midpoint at +/- five years. The risk model was then tested by calculating probability of causation results for males between twenty and forty years of age hypothetically exposed to 1Sv of high energy gamma radiation. The results of these evaluations indicated that the probability of causation exceeds fifty percent only at the ninety ninth percentile and then only for times since exposure greater than fifteen years for men initially exposed at age twenty. Doses higher than 1Sv would be required to produce ninety ninth percentile values of probability of causation that equal or exceed a value of fifty percent for older ages at the time of exposure or at the time of diagnosis. In addition, because NIOSH recognise that reconstructing doses due to internally deposited radionuclides is problematic, they employ a probabilistic approach to dose reconstruction where the radiation dose to the B lymphocytes is a weighted average based on the dose to a given site and the probability that a B cell precursor for CLL will occupy that site.

519. We recognise that the NIOSH-IREP scheme represents a change of US policy but the scientific evaluations behind it reveal no body of new evidence that CLL is radiogenic. Dr Haylock was very dismissive of the methods employed as he opined that the model employed is not specific for CLL and instead just puts all the cancers together. On any view, the existence of the scheme provides no assistance to Mr Battersby's claim of a causal link with CLL. The latency period in this case is 49 years following a putative exposure to radiation of four months in 1956. The CLL was not regarded as aggressive when it was diagnosed. We considered both his potential external effective dose of 680mSv and internal effective dose of 43mSv in

the context of the NIOSH scheme and are certain that this level of dose, the duration of exposure and the late onset of the condition bring him nowhere near the NIOSH-IREP model**.** This model does not apply the legal test we do. We have considered if this change in US policy is plausible evidence that raises a reasonable doubt as to causation in Mr Battersby's case. We do not think that it is to be regarded as plausible evidence of radiogenicity of CLL; further even if it were, it can provide no conceivable assistance to Mr Battersby in his claim for CLL for the reasons we have already noted.

520. Professor Catovsky who has spent his career studying CLL and who received the Rai Binnet medal in 2006 for his contribution to CLL does not consider it to be radiogenic and neither does Professor Hamblin (quoted by both Professor Parker and Catovsky). Both Professor Thomas and Dr Haylock comment on the 2013 Zablotska paper[140] which looked at the incidence of CLL in Chernobyl liquidator workers. This paper is widely quoted by some as stating that this shows there was an increase in CLL. There are a number of issues, however, with this study, particularly the small numbers of cases with CLL, that would indicate that the results should be interpreted with caution and that they could be a chance finding and so are not reliable. These findings are not replicated in the third analysis of UK radiation workers and the contaminated Techa river population follow up.

521. By contrast we note that when she previously gave evidence Professor Parker noted the NIOSH report and concluded that there were studies that concluded that CLL was radiogenic[141]. She acknowledged that she was not a clinician and could not comment on the clinical features of Mr Battersby's disease.

522. We do not accept that the papers authored by Professor Schmitz-Feurhake on the subject of CLL raise any plausible evidence that CLL can be caused by radiation, for reasons already noted in reviewing her evidence. We are surprised that she should publish scientific assessments on the topic or give evidence to us on it and we do not therefore think that her opinion is worthy of being carried forward into the overall evaluation as she is neither an epidemiologist, haematologist or clinician of any sort.

[140] Radiation and the Risk of CLL and other leukaemias among Chernobyl Clean up workers . Zablotska et al 2013. SB 4 /39
[141] 8 February 2013 SB 14 5.5 p 83 -86 ; pp 169-174

Conclusions

523. There is no evidence that exposure to radiation ever caused CLL. We consider expressions of opinion to the contrary not to be valid scientific assessments. Even if there is a hypothesis applicable in the US that in certain circumstances it might be radiogenic, the level of dose and the time for the disease to appear are simply inapplicable in this case. We are sure that Mr Battersby's disease was not caused by radiation.

Herbert George Sinfield

524. Mr Sinfield was born 9 November 1938 and died 30 March 2007 aged 69 years. He submitted a claim form dated 11 December 2006 in which he claimed the progressive effects of non-Hodgkins lymphoma (NHL) as a result of serving on Christmas Island. A Certificate Refused was signed by a SPVA Medical Adviser on 1 March 2007 for the condition large cell lymphoma explaining that this label answered the claim for NHL that is neither attributable to or worsened by service. Mr Sinfield died before he could appeal this decision and his widow continued with it.

525. His cause of his death was recorded as I (a) Ischaemic Heart Disease and (b) Anaplastic Non-Hodgkins Lymphoma.

526. He served in the Royal Army Service Corps on CI from 10 June 1958 to 12 June 1959 and therefore was present for all Grapple Z tests. His statement of 26 February 1984 shows he was a Private employed as a driver transporting stores from the Port to Main Camp and carrying asphalt for the engineers building the roads and airstrip.

527. Mr Sinfield was initially referred to a consultant urologist in September 2005 and then to a dermatologist in relation to a lesion on his penis. He was subsequently admitted to hospital in October and November 2005 with hypercalcaemia and histology of the glans biopsy confirms anaplastic large cell lymphoma.

528. The Consultant Haematologist records that he had chemotherapy which achieved a complete remission. However in November 2006 his lymphoma recurred. In a clinic letter dated November 2006 she states that exposure to excess radiation is known to cause an increased risk of haematological malignancies including lymphoma. She

noted this type of lymphoma was relatively rare and that the presentation was highly unusual[142].

529. It is noted that he had a previous history of anaemia since the 1970s which was treated with iron tablets and was checked by the GP. He was diagnosed with a hiatus hernia at the end of January 2005 and was taking omeprazole prior to his admission. Mr Sinfield was referred to the gastroenterology team in November 2006 because of diarrhoea and weight loss.

Non Hodgkins Lymphoma (NHL/ALCL)

530. Anaplastic large cell lymphoma (ALCL) is a special form of non-Hodgkins lymphoma which in the WHO classification corresponds to two variants according to the expression in the neoplastic cells of a protein called ALK. While ALCL in younger patients is ALK positive, in older patients (40-70yrs old) it is negative.

531. ALCL is a disease affecting T-lymphocytes and like most other types of non-Hodgkins lymphoma (NHL) there is no known causative relationship with radiation exposure. ALCL accounts for 3% of all adult NHLs.

532. There is no well documented increase in NHL in atomic bomb survivors or in other studies of workers exposed to X-rays. Most NHLs have multiple possible aetiologies or pathogenetic mechanisms except for ionising radiation. Professor Catovsky is not aware of any single report linking radiation exposure and ALCL[143]. He does not accept that Mr Sinfield's presentation was unusual for this type of lymphoma in adults.

533. A review of Mr Sinfield's medical file gives rise to no reason to believe that his is such a case. Although the consultant haematologist has opined that the condition may be due to exposure to ionising radiation, Professor Catovsky states that this is not a scientifically sound argument as Mr Sinfield's presentation was not unusual and there is no epidemiological evidence linking ALCL and radiation exposure. Dr Braidwood defers to the opinions of Professors Kaldor and Catovsky.

534. Professor Thomas notes Professor Catovsky's conclusion that there is no evidence that this type of lymphoma is increased in people with a known history of radiation

[142] FTT Part A Sinfield Doc 24 30 November 2006
[143] Prof Catovsky 2011

exposure. She concluded the disease was not radiogenic and Dr Haylock was not asked to calculate a probability of causation for Mr Sinfield.

535. Dr Parker, when giving evidence previously, noted that epidemiology could not assist in showing a link with rare diseases and that there was little evidence and no recent studies, but despite the conclusions of the UNSCEAR Report thought that 'the findings were mixed'[144].

Conclusions

536. We were not shown any reliable evidence to show that NHL/ALCL can be caused by ionising radiation. The clinical experts are clear that it cannot. Even if there is a hypothetical case that it might be, we are satisfied that such a possibility could only arise in the case of exposure to a very significant dose. Mr Hallard estimates that the dose to which Mr Sinfield was exposed on CI was <2 mSv. We are sure that there is no possibility that his NHL was caused by such an exposure.

Gwillym Avron Pritchard

537. Mr Pritchard was born on 11 September 1935. He died on 5 December 2005.

538. Mrs Pritchard submitted a widow's claim form dated 9 January 2006 in which she attributed her husband's health problems and particularly his diabetes to his service on Christmas Island. A Certificate Refused (Widow Case) signed by a SPVA Medical Adviser and dated 13 February 2006 was issued.

539. Mr Pritchard served in the RAF from 1951 until 1961. He was on CI from 24 January to 28 November 1958 and therefore was present for Grapple Y and Z1. He was on Fanning Island from 28 August to 2 October 1958. During this time he was a Corporal employed as a telegraphist. He stated he was at Witness Point for Grapple Z1.

540. The causes of Mr Pritchard's death were recorded as follows: I (a) Bronchopneumonia, Cardiorespiratory Failure, End Stage Renal Failure (b) Berger's Nephropathy, Hypertensive Heart Disease, Arterial Atheroma and II Diabetes Mellitus.

[144] 1 February 2013 SB 14 Tab 5.6 pp 50-53

541. These conditions were considered by the Secretary of State in the rejection of Mrs Pritchard's claim.

542. Mr Pritchard's post-service medical records show a diagnosis of what was termed 'mild' diabetes (controlled by diet) in 1984 and thereafter frequent follow up in a diabetes clinic. Despite taking Metformin, by 1987 his diabetic control is recorded as not well controlled and he is referred to the dietician. In August 1988 he is reviewed in the diabetic clinic when he is noted as not sticking to his diet and persisting in drinking alcohol. By October 1988 his control is described as poor and this pattern persists.

543. He is reported to have smoked heavily – 40 a day for 20 years[145]. His GP records hypertension on 3 December 1991 with a BP of 150/100.

544. A Consultant Physician's report in 1992 diagnoses diabetes mellitus (non insulin dependent), angina pectoris (diagnosed 6 years previously) and sero-negative arthritis and notes his usual weight being 11 stone and that his joint symptoms limit his physical activity.

545. A diagnosis of possible Ménières was made in 1994.

546. By 2003 Mr Pritchard has developed near end stage renal failure due to diabetes and it is recommended that he have dialysis. In July 2003 an Associate Specialist in Renal Medicine writes about his renal failure due to diabetes mellitus/IgA nephropathy and later that year on 11 August Mr Pritchard started dialysis twice weekly. In July 2004 however he has surgery for bilateral inguinal hernias and suffers a cardiac arrest on 27 July 2004. He subsequently has a cardiorespiratory arrest during dialysis in August 2004 and is admitted to hospital from August until October. He then has further admissions during 2004 and 2005 with various medical conditions including drug induced Parkinsons and is finally admitted in November 2005 with pneumonia, heart failure and Type 1 respiratory failure.

547. Mr Pritchard died on 5 December 2005 and a post mortem was conducted. The pathologist states that 'in my opinion death was due to natural causes. I have not found any evidence of radiation induced pathology either neoplastic or non-neoplastic'.

[145] FTT Part A Pritchard Doc 15 dated 20 January 1988

548. The medical records show that his renal failure and undoubtedly his ischaemic heart disease was a result of his diabetes mellitus which was poorly controlled leading to complications.

Bergers nephropathy (Immunoglobulin A nephropathy (IgAN))

549. Bergers nephropathy was first described by Berger in 1968 and at one time was known as Berger's disease. It is the commonest glomerulonephritis in countries where renal biopsy is widely used. In most cases the aetiology of IgAN remains unclear and although infections by cytomegalovirus and haemophilus parainfluenzae have been implicated, neither these nor any other viral or bacterial antigens have been consistently associated with development of the disease or identified in IgAN immune complexes or mesangial deposits. Alternatively, it has been suggested that IgAN results from hypersensitivity to food antigens, in view of its association with gluten-sensitive enteropathy IgAN.

550. The Tribunal was not shown any evidence as to ionising radiation being a possible causative factor in the development of this disease.

Hypertension

551. Hypertension (high blood pressure) induces changes in the heart and blood vessels and is often associated with diabetes either as part of the insulin resistance syndrome or as a manifestation of renal disease. High blood pressure is treated particularly aggressively in patients with diabetes as it accelerates the progression of both microvascular (such as eye and kidney disease) and macrovascular complications (such as ischaemic heart disease) in diabetes.

Diabetes

552. The evidence before the Tribunal showed that there are very few studies looking at the relations between the development of diabetes and exposure to radiation. Type 2 diabetes mellitus is a very common condition. In the UK 700 people a day are diagnosed with diabetes. Since 1999 the number of people diagnosed with diabetes has more than doubled from 1.4 to 3.5 million.

553. Professor Thomas notes that the risk factors include being aged over 45, being overweight, physically inactive and smoking. Mr Pritchard was 49, had been a smoker for 20 years, was overweight and inactive.

554. De Vathaire et al[146] in a paper published in the Lancet in 2012 concluded that children and young adults treated with total body or abdominal radiotherapy have an increased risk of insulin resistance and diabetes mellitus. The mean dose of radiation to the body of the pancreas was 12 Gy whereas the mean dose to the tail or head of the gland was 8.8 Gy. The risk of diabetes increased strongly with radiation doses to the tail of the pancreas where the islets of Langerhans are concentrated up to 20-29 Gy and then reached a plateau of higher radiation doses. The radiation dose to the other parts of the pancreas did not have a significant effect. The risk was highest in children under two.

555. As a side issue, we note that this latter study is also interesting with regards to the issue of bias as a result of using questionnaires to quantify self-reported conditions. 16% of respondents claimed to have developed diabetes but this was not substantiated when the researchers checked with their GPs.

556. Professor Parker expressed the opinion in 2013 that Mr Pritchard's diabetes and kidney disease are due to chronic inflammation caused by perturbation of the immune system. The Tribunal were not shown any evidence to raise this as a reasonable doubt and find that as an epidemiologist this is not within Professor Parker's area of expertise.

Conclusions

557. The evidence before the Tribunal already considered with respect to Mr Beeton (see paragraphs 480-483) showed that Mr Pritchard's heart condition could be caused by high dose radiotherapy of 40 Gy but that there was no evidence that radiation could be a contributory cause with doses of less than 500 mSv. Mr Hallard estimated that Mr Pritchard's dose was 1 mSv.

558. Dr Haylock was unable to perform a probability of causation calculation for these conditions at such low doses.

559. We are satisfied that with an effective dose of 1 mSv Mr Pritchard's diabetes and other claimed conditions were not caused by radiation exposure.

[146] Radiation dose to the pancreas and risk of diabetes mellitus in childhood cancer survivors: a retrospective cohort study SB 4 TAB 15

Charles Frederick Selby

560. Mr Charles Selby was born on 9 June 1935. He died on 25 August 2005 aged seventy. Mrs Selby submitted a widow's claim form dated 6 February 2006 in which she attributed her husband's death to his service on Christmas Island (CI). A Certificate Refused (Widow) was signed by a SPVA Medical Adviser on 8 March 2006.

561. Mr Selby enlisted in 1956 and served in the Royal Engineers on CI from 10 August 1957 to 20 July 1958 and therefore was present for Grapple X and Y. During this time he was a Sapper employed as an Engineering Plant Fitter which involved him servicing the plant used by the Royal Engineers.

562. The causes of his death were recorded as: I (a) Idiopathic Fibrosing Alveolitis (IFA) and II Type 2 Diabetes Mellitus. These conditions were considered by the respondent in the rejection of Mrs Selby's claim.

563. Mr Selby's post-service medical records show a pulmonary embolism in 1998 and a history of asbestos exposure due to his work as a miner for 10 years and then as a maintenance fitter in the Carbon Black factory. It reports him stopping smoking in the 1960s. In January 2003 he is referred to a chest physician because of breathlessness and a CT scan is arranged because of his history of exposure to asbestos. The CT scan did not show evidence of exposure to asbestos and early stage fibrotic lung disease is diagnosed. He is diagnosed with essential hypertension in June 2004.

564. His GP reports a diagnosis of type 2 diabetes in 2000 and in 2004 a specialist registrar in respiratory medicine includes in Mr Selby's diagnosis hypercholesterolaemia in addition to diet controlled diabetes. By the middle of July 2004 inhalers and oxygen were prescribed and then steroids by May 2005 but unfortunately his conditions deteriorated and he died in August 2005.

Radiation pneumonitis

565. The lungs can be injured by the high doses of radiation used in cancer treatment and the initial injury is followed by an inflammatory response and at a later stage by fibrosis. Acute radiation pneumonitis is characterised by interstitial inflammation occurring up to four months after radiotherapy and then resolving over a matter of

weeks or months. Radiation fibrosis, which can occur without preceding pneumonitis, develops about six months after radiotherapy and may progress over six to 24 months: it does not resolve, but usually stabilises by two years. Factors which influence the development of radiation pneumonitis and fibrosis include the volume of lung irradiated, the total radiation dose administered, and the dose rate and fractionation[147]. Lung fibrosis can be associated with radiation but it is at a high dose (5000mSv).

Idiopathic Fibrosing Alveolitis

566. The disorder previously known as fibrosing alveolitis, first described in 1907, was increasingly recognised following the description of a small group of patients with rapidly progressive fatal disease, grouped as the Hamman–Rich syndrome. Until late in the 20th century, a stereotypical clinical presentation of idiopathic interstitial lung disease was termed idiopathic pulmonary fibrosis (IPF) or cryptogenic fibrosing alveolitis (CFA), and a number of histological patterns were unified under this term.

567. Mr Selby's consultant has made the diagnosis of idiopathic or cryptogenic fibrosing alveolitis which means that there is no cause identified.

568. The evidence before the Tribunal showed that Mr Selby had been a smoker and had worked in the mining industry. These are risk factors for idiopathic fibrosing alveolitis (IFA).

569. With regards to the development of diabetes mellitus, the Tribunal considered the De Vathaire et al[148] paper published in the Lancet in 2012 which concluded that children and young adults treated with total body or abdominal radiotherapy have an increased risk of insulin resistance and diabetes mellitus. The mean dose of radiation to the body of the pancreas was 12 Gy whereas the mean dose to the tail or head of the gland was 8.8 Gy. The risk of diabetes increased strongly with radiation doses to the tail of the pancreas where the islets of Langerhans are concentrated up to 20-29 Gy and then reached a plateau of higher radiation doses. The radiation dose to the other parts of the pancreas did not have a significant effect. The risk was highest in children under two.

[147] Oxford textbook of Medicine 5th Ed
[148] Radiation dose to the pancreas and risk of diabetes mellitus in childhood cancer survivors: a retrospective cohort study SB 4 TAB 15

570. Type 2 diabetes mellitus is a very common condition and the risk factors for men include being aged over 45, overweight, physically inactive and smoking. Mr Selby was 65 and had been a smoker giving up in the 1960s.

Conclusions

571. The Tribunal is sure that with Mr Hallard's conservative estimate of effective dose of 1mSv that Mr Selby's type 2 diabetes and IFA were not caused by radiation exposure.

Derek Hatton

572. The late Mr. Hatton was born on 4 November 1938. He submitted a claim dated 18 December 2007 for polycythaemia rubra vera (PRV) which he attributed to his service on Christmas Island (CI). A Certificate Refused signed by a SPVA Medical Adviser dated 27 May 2008 for the claimed condition.

573. He served on CI from 21 August 1958 to 15 August 1959 and therefore was present for Grapple Z 1, 2, 3 and 4 tests. During this time he was a Private in the Royal Army Ordnance Corps. He was based at Main Camp.

574. His GP notes that his PRV was first recorded on the computer on 1 January 1997 but may go back much further[149]. On 12 October 2001 a Specialist Registrar to a Consultant Physician notes that perhaps he has PRV but is unable to make a firm diagnosis[150] and an Associate Specialist in Haematology diagnoses JAK-2 positive polycythaemia rubra vera on 30 November 2007. His diagnosis was made when he was 63 and some 40 years after service.

Polycythaemia rubra vera (PRV)

575. The evidence before the Tribunal shows that PRV is a rare chronic progressive haematological malignancy and therefore it is difficult to establish a link from epidemiology. It is now identified in more than 95% of patients by detection of an acquired mutation of the tyrosine kinase JAK2 gene.

576. No alternative evidence was found to establish a causal link between PRV and radiation exposure. However there are no known environmental factors responsible

[149] FTT Part A Hatton Doc 39
[150] FTT Part A Hatton Doc 127

for the development of the disease. There is no excess of cases of PRV as compared with controls in the report on the UK participants. No excesses of PRV were found following the atomic bomb explosions in Nagasaki but a few cases were noted in Hiroshima. There is no evidence in the literature that PRV could have developed after 40 years from exposure[151].

577. Dr Braidwood states that War Pensions policy based on the findings of the NRPB studies is to automatically accept claims for leukaemias (other than CLL) where they have a clinical onset within 25 years of presence at a site. NRPB did not analyse data on PRV but on a case report from the USA, the presumption was extended to include PRV with onset within 25 years of service termination. That finding was not subsequently replicated. We understand that such a presumption has not subsequently been applied to war pensions claims but in any event it would not apply here as the condition was not diagnosed within 25 years of service.

578. Professor Thomas did not consider that this condition could be caused by exposure to radiation at the dose estimated by Mr Hallard and in the circumstances noted above. Dr Haylock made no statistical calculation of probability.

Conclusion

579. The Tribunal is sure that there is no plausible evidence to indicate that radiation was a possible cause of Mr Hatton's condition.

Trevor Michael Butler

580. Mr Butler was born on 7 March 1938. He submitted a claim dated 15 February 1990 for a congenital cataract in his right eye which was noted at his entry medical examination, was surgically removed in 1965 and which a tribunal allowed as aggravated by service on 26 January 1995[152]. He made another claim on 18 March 1996 for back and skin problems. He appealed the refusal of these conditions under the labels of lumbar spondylosis and psoriasis on 21 November 1996.

581. He made a further claim on 30 August 2006 for reduced immune system, glomerulonephritis, nightmares, klebsiella, septicaemia, streptococcal viridans and

[151] Prof Catovsky 2011
[152] FTT Part A Butler Doc 87

high blood pressure. He attributed all these conditions to his service on Christmas Island (CI).

582. A Certificate Refused signed by a SPVA Medical Adviser on 20 April 2007 was issued for his last claim listing the conditions as: staphylococcal lumbar discitis, streptococcal viridans infection, glomerulonephritis, klebsiella urinary tract infection and hypertension with septicaemia and associated side effects of medication (nightmares) included as part and parcel.

583. He served on CI from 30 December 1957 to 12 December 1958 and therefore was present for Grapple Y and Grapple Z 1, 2 ,3 and 4 tests. During this time he was a Sapper in the trade of a Field Engineer. He was based at Main Camp and at Site B for some of the Grapple Z tests.

584. His medical records show that he was admitted in 1957 with influenza and that in 1958 there is a consultation for a skin rash which was diagnosed as tinea. A report from his GP dated 20 December 2006[153] records renal impairment and possible glomerulonephritis on biopsy on 20 February 2001, nightmares from 2002 due to medication (diltiazem) for hypertension, essential hypertension from 20 August 2004 and staphylococcal discitis L2/L3 in 2001 which had completely resolved by November 2001.

585. He was admitted in January 2001 with abdominal pains. He was septicaemic and blood cultures initially grew staphyloccus aureus and then streptococcus viridans. CT scans showed a small pleural effusion and multiple benign renal cysts[154]. There was also the suggestion of a hepatic abscess. He was treated with antibiotics but then developed renal impairment and a renal biopsy was conducted in March 2001 which confirmed pauci immune focal glomerulonephritis. An MRI scan of his spine in February 2001 showed L2/3 discitis. There is a tentative diagnosis of bacterial endocarditis at the same time but it is thought unlikely on the echocardiogram which shows moderate mitral regurgitation.

586. In March 2001 a urine sample grows Klebsiella/enterobacter.

[153] FTT Butler p131
[154] There is evidence that his sister has cysts to her kidneys

587. There is a clinic letter from 8 November 2001 from a consultant Physician/Nephrologist which confirms the diagnoses of staphylococcal lumbar discitis as being completely resolved, renal impairment with possible glomerulonephritis on biopsy, hypertension and duodenal ulcer induced by NSAIDS.

588. By March 2005 a letter from the cardiologist confirms that he has moderate mitral regurgitation but is asymptomatic and haemodynamically stable. He has mild chronic renal impairment with a creatinine of 202 in 2004 and also a diagnosis of ankylosing spondylitis.

589. From the hospital case notes the respondent's Medical Adviser labels one of his conditions, immune system dysfunction[155], although on p131 the consultant states that there is no evidence of reduced immune system. The Secretary of State however does not accept that this is related to his service on CI.

590. Both Dr Braidwood and Professor Thomas agree that immune system dysfunction is radiogenic as a high dose deterministic effect. A dose above 1000 mSv has direct effects on the cells of the haematopoetic system especially lymphocytes. Data from the LSS study suggests that there may be a minor effect on the proportion of CD4 positive T cells in some members of the study. However the dose response relationship of this finding is unclear and the change in proportion of cells of this particular type does not translate into illness or clinically detectable changes in immune function. Mr Butler did not receive a dose above 1 Sv.

591. Professor Parker previously states that radiation exposure can result in long term perturbation of the immune system which can result in chronic inflammation, increased autoimmune disease and increased rate of infection but we observe that such a hypothesis would not fit with an infective episode 43 years after exposure which resolved completely with antibiotics and where there was no recurrence or evidence of abnormal blood tests.

592. Acute glomerulonephritis refers to a specific set of renal diseases in which an immunological mechanism triggers inflammation and proliferation of glomerular tissue that can result in damage to the basement membrane, mesangium or capillary endothelium[156] It occurs most commonly after a streptococcal infection but can also

[155] FTT Part A Butler Doc 190
[156] emedicine Acute Glomerulonephritis (contained in Mr Butlers file (2))

be caused by vasculitis. Immunofluorescence usually reveals deposition of immunoglobulins other than in the pauci-immune types (hence the name).

Conclusions

593. The evidence before the Tribunal is that Mr Butler was 66 years old when he developed this infection and would have already received a life time dose from background radiation of 132 mSv. Mr Hallard calculates that his likely internal dose would have been <2 mSv. There is no plausible evidence before the Tribunal that his conditions could be caused by radiation at a low dose, many years after exposure.

Ernest Wynne Hughes

594. Mr Hughes was born on 8 March 1935.

595. Mr Hughes submitted a claim dated 1 November 2008 for skin problems, coronary disease/heart attack and bladder cancer which he attributed to his service on Christmas Island (CI). A Certificate Refused signed by a SPVA Medical Adviser dated 11 February 2009 was issued listing the conditions as Sebaceous Cysts (1991), Epidermal Cysts (1992), Pruritus, Atherosclerosis, Myocardial Infarction (1991) and Transitional cell carcinoma of the bladder (2006) with Coronary artery disease and angina as part and parcel.

596. He served on CI from 29 October 1956 to 10 September 1957 and therefore was present for Grapple 1, 2 and 3 which were conducted 700 kms away off Malden Island. He was a Leading Technician in the RAF employed as a radio engineer at the airfield and he was accommodated at Main Camp.

597. In his claim form he stated he was at CI for Grapple X. However his service record shows he left on 10 September 1957 and he confirmed this in his first witness statement dated 21 February 2012. In this document he recalled that in the build up to Grapple X he witnessed ‘some form of small bomb test or radioactive release’. As we have already observed there is no contemporaneous record of this additional test and there would have been if it had occurred. A practice test would not have involved any form of ionising radiation. Grapple X took place on 8 November that year.

598. The Secretary of State also draws our attention to the fact that Mr Hughes also asserts in his second witness statement that shortly after his return from CI in 1957 when he was stationed at RAF Edlesborough (near Dunstable in Bedfordshire) a fire erupted in the pile used to manufacture the nuclear fuel and contaminated the atmosphere with radioactive material. Mr Hughes confirmed in his previous oral evidence that the location of this alleged fire was in Cumbria and that the radioactive material was transported by wind vectors to Edlesborough.

599. His GP notes a skin rash on his trunk in 1995, acute myocardial infarction (MI) in December 1991 and bladder cancer confirmed on 22 May 2006 when he was aged 70. In his claim form he states that his skin rash was diagnosed as pruritus on 9 April 2001, that his medical notes show he had a sebaceous cyst on 4 April 1991 and on 24 November 1992 he had an epidermal cyst[157]. The diagnosis of papillary transitional cell carcinoma was confirmed by histology[158], grade 2, and showing infiltration into the lamina propria (G2 pT1) when he was 73. He had a transurethral resection of the bladder tumour on 5 June 2006 and had a course of Mitomycin C for some superficial recurrence in September 2006.

600. With regards to his ischaemic heart disease, there is a history of MI in December 1991 and angina which was medically treated. On 11 March 2005 when he was 70 years old an SHO in Cardiology reviewed his diagnosis of two vessel coronary artery disease. She noted that his blood pressure and cholesterol were controlled on treatment. The underlying disease process is atherosclerosis. Mr Hughes is a non smoker and is not diabetic. It was noted that a second angiogram done on 21 July 2004 revealed a large antero-apical scar with an apical aneurysm, the LAD remained occluded and there was moderate stenosis of the dominant R coronary artery plus minor disease in the circumflex. He had no cardiac symptoms other than angina and so it was decided that medical treatment only was appropriate and he was discharged.

Myocardial infarctions and atherosclerosis

601. The evidence before the Tribunal shows myocardial infarctions and atherosclerosis could be caused by high dose radiotherapy of 40 Gy but that there was no

[157] FTT Part A Hughes Doc 30
[158] FTT Part A Hughes Doc 54

contributory cause with doses of less than 500 mSv (see [480] to [483]). The UK AGIR 2010 reviewed the evidence and concluded that there was not sufficient evidence of excess risk for cumulative doses <500 mGy (equivalent to 500 mSv external dose).

602. The most recent data from the LSS suggests that no risk for CV disease can be determined in <500 mGy.

603. Mr Hughes's dose did not exceed this figure. High blood pressure and raised cholesterol are risk factors in coronary artery disease.

Bladder cancer

604. As we have already noted in discussing Mr Abdale's case (see [464] to [472]) bladder cancer can be caused by either external or internal radiation by radioisotopes dissolved in the urine and this depends on the internal dose.

605. Mr Hughes's only dose was an internal dose of 0.01 mSv and he would have been exposed to 2 mSv per year on average from background radiation. This would equate to 140 mSv at the time of diagnosis of his cancer. Dr Haylock calculated this gave a 0.0004% chance of his bladder cancer being caused by his exposure to ionising radiation whilst serving on CI.

Epidermoid and pilar (sebaceous) cysts

606. A cyst is a sac that is filled with a fluid or semi-fluid material. Cysts develop in various places in the body and arise from different tissues in the body. Two of the most common types of cyst that occur under the skin surface are epidermoid and pilar cysts. These cysts used to be called sebaceous cysts but this term is no longer correct, as the origin of these cysts is not from the sebaceous glands in the skin (as was once thought).

607. An epidermoid cyst is a cyst where the cyst sac forms from cells that normally occur on the top layer of the skin (the epidermis). A pilar cyst is a cyst where the cyst sac forms from cells similar to those that are in the bottom of hair follicles (where hairs grow from). There is no evidence to suggest that ionising radiation is a cause of these cysts.

Pruritus

608. As Dr Braidwood states this term is a symptom, not a diagnosis. It can be related to many health problems ranging from serious systemic diseases such as liver or kidney disease to eczema or anxiety. There is no evidence in this case that the pruritus is a symptom of a serious underlying pathology and there is nothing to show a link between pruritus and ionising radiation.

Conclusion

609. In the light of Hallard's assessment of Mr Hughes's internal dose of 0.01 mSv (see [605]) we find that his service on CI did not cause his bladder cancer, or atherosclerosis. Furthermore we find that epidermoid and pilar cysts and pruritis are not radiogenic.

Brian Lovatt

610. Mr Lovatt was born on 14 April 1937 He submitted a claim form dated 3 November 2009 in which he claimed three heart attacks and pronounced difficulty breathing on exertion which he had had since 1969 and which had not been fully investigated. He attributed these conditions to his service on Christmas Island (CI). A Certificate Refused signed by a SPVA Medical Adviser dated 20 January 2010 was issued labelling the conditions as Atherosclerosis and myocardial infarction (2005) with Angina and shortness of breath as part and parcel.

611. He served on CI from 9 September 1957 to 9 June 1958 and therefore was present for Grapple X and Y. During this time he was a Leading Aircraftman (LAC) and his main role was paint spraying aircraft and jeeps. He was based at Main Camp but spent a few months based at the harbour to the west of the island spraying fuel pipes.

612. He was admitted in 1999 with hypertension and had a CT scan which showed a cerebral infarct (lacunar infarct in the head of the caudate nucleus on the left side and a mature infarct in the left genu of the corpus callosum). He had a normal renal ultrasound and all other investigations were normal.

613. He had an anteroseptal ST elevation myocardial infarction (STEMI) in September 2005 which was treated with thrombolysis. Risk factors were noted as being an ex-smoker, hypertension, untreated hypercholesterolaemia and two previous transient

ischaemic attacks (TIAs). His mother was reported to have 'heart problems'. He subsequently had angioplasty and stenting to the LAD with drug-eluting stents with no complications. He had a positive exercise test in 2007 and was due to have further angiography in May 2009 but his angina had improved with the increased dose of Nicorandil. Further angiography was undertaken in October 2009 but no further stents were introduced. He had a perfusion scan which precipitated an episode of atrial flutter because of the withdrawal of medication and an exercise test with a good haemodynamic response.

Atherosclerosis

614. Atherosclerosis is a disease of large and medium sized muscular arteries characterised by inflammation and dysfunction of the lining of the involved blood vessels and the build up of cholesterol, lipids and cellular debris. This results in the formation of a plaque, obstruction of blood flow and diminished oxygen supply to target organs[159].

615. Risk factors include age, sex, family history, hyperlipidaemia, hypertension, diabetes, smoking and the metabolic syndrome.

Conclusions

616. As we have discussed before, the evidence before the Tribunal showed the conditions claimed could be caused by high dose radiotherapy of 40 Gy but that there was no contributory cause with doses of less than 500mSv. Mr Lovatt's dose was 1 mSv. As noted in his medical records his other risk factors for developing his condition were hypertension, high cholesterol, TIA and family history. We are accordingly sure that his conditions were not caused by the service.

Dennis Shaw

617. Mr Shaw was born on 17 April 1937. The late Mr Shaw claimed for sub capsular cataract left eye as a result of radiation exposure on Christmas Island (CI). His claim is dated 18 March 2009. A Certificate Refused signed by a SPVA Medical Adviser dated 6 August 2009 was issued for the condition Left cataract.

[159] Synopsis of causation . Atherosclerosis 2008

618. Mr Shaw served in the Royal Engineers. He was posted to CI from 16 December 1958 to 27 November 1959. He was not present during any of the tests. He was a Sapper and undertook maintenance work. He spent time at Main Camp, Port Camp and the Airfield.

619. His GP records record bilateral cataracts in 2004 with the right one treated in 2005[160]. Ophthalmic diagnosis of bilateral nuclear sclerosis is made on 17 October 2005[161]. His left eye was treated in 2006. He underwent further treatment (3 snip procedure to inferior lacrimal puncti) on both eyes on 17 August 2007[162]. In his appeal Mr Shaw referred to his eye specialist diagnosing him with a sub capsular cataract on his left eye as well as the bilateral nuclear sclerosis ones. Mr Shaw was 68 at diagnosis.

620. The evidence before the Tribunal already discussed in the case of Mr Abdale is that cataracts have been shown to be radiogenic and in recent years there has been a downward revision of the safe dose limit of exposure of the eye. This limit is 20 mSv with a maximum annual dose of 50 mSv. In the case of cataracts the equivalent dose to the skin is taken as representative of a dose to lens of the eye. Mr Shaw's external skin dose was 2 mSv.

621. Further it is a condition common in elderly people and Mr Shaw was 68 when diagnosed with cataracts.

Conclusion

622. In the light of Mr Hallard's estimates of maximum dose and the discussion the Tribunal is satisfied that Mr Shaw's condition was not caused by his service on CI.

PART EIGHT:
CONCLUSIONS

Introduction

623. We now bring together the conclusions reached in previous parts of this determination to make an overall assessment as required by paragraphs [101] to [103] of the UT decision, already quoted in Part One of this determination.

[160] FTT Part A Shaw Doc 41
[161] FTT Part A Shaw Doc 43
[162] FTT Part A Shaw Doc 51

624. We recognise that nuclear energy and military use of nuclear energy has over the decades raised real concerns as to the safety of those living or working in close proximity and to members of the public who may be affected by environmental radiation.

625. It is perfectly legitimate for people campaigning on these issues to raise all the doubts and uncertainties in language that delivers a strong message and alerts decision makers and the uninformed alike to issues that need to be addressed. We equally recognise why veterans and their families would be indignant and react strongly to any suggestion that risks to health to those who have served their nation as members of the armed forces are not being rigorously or fairly assessed. The unique test of reasonable doubt based on reliable evidence, as expounded by Mr Justice Charles, seems to us designed to recognise the special position of the armed forces and the special risks to which they may be exposed as part of their service to the nation.

626. Nevertheless, there must be some evidential basis for the claim. Although a scientific hypothesis that is plausible in all the circumstances may amount to such evidence, the emotive and colourful language of political discourse is not. Science has a commitment to scientific method: constantly asking questions, exchanging and reviewing knowledge, objectively evaluating possible answers raised in the inquiry, distinguishing in varying degrees between the certain, the probable and the possible. Dr Busby drew to our attention the treatment of Galileo when he challenged conventional scientific assumptions, but in our view the point is that Galileo had concrete evidence and rigorous reasoning based on it to make the challenge that he did. There is a wide range of theories on matters of interest to science that are not plausible (with the meaning set out at [33]). The range may extend from the speculative to the eccentric, but all such theories share the feature that they are advanced without supporting evidence whether empirical, based on sound scientific principles or hypothetical, based on a coherent analysis of what is known.

627. Equally, the function of the law is the objective evaluation of the issues, in good faith and with the degree of anxious scrutiny appropriate to the subject matter. Here the degree of scrutiny has been authoritatively stated by the Upper Tribunal. It would, in our judgment, be wrong to give further colour to that test by reference to

the fact that the subject matter is the statutory scheme for compensation for those who have served the nation or that the issue of causation is the difficult one relating to the consequences of potential internal exposure to low level radiation.

628. We reach our decision on the evidence that has been placed before us and that we consider to be material to the issues. We have not been impressed with sweeping generalities from limited or debatable data. We have not let our focus become distracted into engaging with possibilities remotely connected to the present appeals although some have been touched on in the material we have received, such as the adequacy of the compensation scheme for Japanese survivors. We have endeavoured in the previous parts of the determination to examine what we consider to be the principal arguments of the parties on the issues. We have not endeavoured to deal with every point made in the voluminous submissions made to us.

629. The members of the Tribunal have brought their different skills and experiences to bear on the issues arising in this appeal, but always within the context of the evidence and arguments addressed to us, and not by means of an independent inquiry of our own. It will have been apparent that Parts Two, Four, Six and Seven have required very considerable analysis of scientific and medical issues of some complexity. We are fortunate that the skills and experiences available to us have included the necessary medical expertise to evaluate the evidence presented and submissions made.

630. What we have made of the evidence and argument applying the test set for us by the UT, is a collective view based on our knowledge, experience and understanding.

631. With these observations, by way of preamble, we now address the sequence of issues we need to determine when reaching our overall conclusions.

The LNT model of assessing health risk from radiation

632. For the reasons we have given in Part Four of this determination we are sure (and therefore have no reasonable doubt) that the appropriate means to examine the risk to health in each of these appeals is the internationally recognised LNT model approved by the ICRP and applied by UNSCEAR and national agencies such as the HPA. The linear no threshold model recognises that no level of radiation is absolutely safe but that risk of an adverse health consequence increases with dose.

633. Equivalent dose is a sophisticated concept based on rigorous inquiry into all that is known about the health effects of radiation exposure and produces a calculation taking into account the nature of the radioactive nuclides to which there has or may have been exposure, the duration of exposure, and the impact on the particular organ of the body. We are sure that this is the instrument by which assessments of internal exposure and possible consequences should be made.

634. We are equally sure that neither the particular nature of uranium nor the existence of potential pathways of internal exposure through inhalation or ingestion make either the LNT model or the concept of equivalent dose inappropriate in the determination of these appeals. We equally have no doubt that for the reasons we have explained in Part Six why no modification of the LNT is required by developments in the understanding of radiobiology such as bystander or photoelectron effects, or radiation related genetic mutation of cells.

635. Accordingly, the existence of reasonable doubt must depend on whether any of the veterans was exposed to a dose of radiation at all and if they might have been whether such exposure was at an intensity that science suggests might cause a risk to health of the kind of condition that forms the basis of the claim. We are, therefore, sure first that dosimetry is and remains an essential element of the process of assessment of risk to health, and second that it is the possibility of the particular health condition being caused by exposure to the dose assessed is the focus of the particular appeal.

636. We recognise that dosimetry itself may not always be capable of giving a precise measure of exposure, particularly as here, where events were close to 60 years ago and data on individual measurements may be limited, lost or never existed. We are also sure that assessment of risk based on low level exposure (i.e. an exposure that results in an effective dose of below 100 mSv) is more difficult than in cases of high exposure (for present purposes an effective dose of 500 mSv and above). The stochastic effect of internal exposure is also more difficult to assess than for external exposure.

637. Where internal dose is very low (for present purposes below an effective dose of 50 mSv) epidemiology may be able to provide data as to statistical probability of risk of particular health outcomes being caused by such a dose. Epidemiology can only play

a limited role in individual decisions relating to low dose exposure: it can draw together the information about specific health outcomes on sections of the population that have experienced radiation exposures. It can only inform as to predicted outcomes, and the ability to predict depends on the quality of the previous studies. It aims to eliminate bias, identify confounding factors and compare with a control group of broadly similar age and qualities without the experience of exposure and the general population. The strength of the prediction depends on the statistical power of the study and the extent to which one study is supported or challenged by the outcomes of similar studies.

638. When very low doses are in issue, it is relevant, when considering causation, to compare the background radiation that any individual may receive depending on place of residence and other environmental factors. Effective dose that is no higher than background radiation is less likely to have any causative effect, but we recognise that this is not a determinative test and that low doses when added to background radiation or other exposure experiences may make a causal contribution to an adverse health outcome.

Exposure to Radioactivity on Christmas Island

639. It is common ground that none of the veterans in these appeals could have had radiation exposure resulting from the early Grapple tests on Malden Island, save for the possible contamination pathway from May 1958 onwards of water used in the decontamination of aeroplanes involved in the Malden Island detonations, filtering through the coral to the lens of fresh water from which drinking water was or may have been drawn.

640. Further, none of the veterans was sufficiently close to any of the detonations of Grapple X Y or Z to have had any realistic possibility of acute direct exposure. None was required to be sufficiently close to the detonation or the cloud containing radioactive residues to be at risk from this means. By contrast, reasonable doubt has been found to exist for a number of veterans who may have had significant levels of direct exposures: such as the pilots of aircraft.

641. The low level Grapple Z tests presented significantly less risk of atmospheric deposition of fissile products, but greater risk of contamination of dust within the

vicinity of ground zero. The high altitude bursts of Grapple X Y and Z were all devised so as to:

(i) avoid any fireball contact with the surface of the sea or land and entrain material that would return to earth as contaminated;

(ii) ensure effective detonation of the fuel and tamper materials at high altitude;

(iii) ensure that the particles of fission products in the cloud at high altitude rose through the troposphere through the tropopause to the stratosphere where it would be carried far away from Christmas Island (CI) and any possible risk of health exposure to the human populations of the island or surrounding regions;

(iv) avoid detonations where wind or rain conditions would create a significant risk of rainout or wind born deposition of fission products in the cloud. To this end, considerable resources were deployed in meteorological observation on the north of the Island and at sea to gather relevant information;

(v) ensure that non-essential personnel were located at a significant distance from controlled zones and that access to the controlled zone was restricted.

642. There is no plausible evidence to suggest that there is any doubt that each of the tests were conducted in a manner intended to give effect to all these principles. We are satisfied that the detonations took place at the intended height and in circumstances where the meteorological assessments suggested it was appropriate to do so. We are further satisfied that there was a robust safety regime during the tests at CI that was designed to protect individuals from significant exposures and would have triggered a set of obligations if there was any reason to believe that radioactive contamination in excess of the regulated level had taken place. There were numerous instruments on the island used for a variety of monitoring and testing purposes that would have registered if there was significant background radiation when first switched on and calibrated. There was no indifference to the risks of service personnel or others on CI and human populations were not used as guinea pigs, despite the apparently unsophisticated instructions to those observing events 40 kilometres or 25 miles away to turn their backs at the moment of detonation and only turn to face the blast

15 seconds later. Further, each of the present veterans (with the possible exception of Mr Battersby) was located well away from the areas of the detonation and was not tasked to perform a function that would bring them within predicted proximity of radioactive material or the product of the explosion.

643. Grapple Y was the largest detonation in the series, and although each of the safety modelling features that we have noted above applied to it, we recognise that on the information before us, we cannot exclude the possibility that there were unintended and unmonitored radiation exposure pathways presented by this explosion.

644. These possible pathways were:

(i) Adventitious deposition from the stem by low level unpredictable winds at ground zero generally blowing west or north-north-west and probably reflected in the contamination found at Vaskess Bay.

(ii) Fissile materials including hot particles becoming deposited on parts of CI and in the adjacent ocean through rainout and less plausibly dry deposition.

645. We are sure that deposition in the ocean was not a source of internal exposure to any of these veterans:

(i) Direct radiation of the ocean by ionising the sodium in the sea was a short lived exposure that caused no risk to humans who would subsequently swim in or consume produce from the sea.

(ii) The scale of any deposition would be very limited having regard to the meteorological data and the planning considerations.

(iii) The Pacific Ocean is vast and an enormous and effective diluter of any fallout deposited therein.

(iv) Direct monitoring of seawater by ship or aircraft and monitoring of fish and seafood after detonation and during clear up operations either never revealed any measurable radiation save in a few instances where detectable radiation in pico-curies was recorded.

646. Accordingly we are sure that the possibility of sea to land transfer from nuclide deposition in the sea becoming a source of internal exposure by ingestion or inhalation of dust is so trivial a factor for CI that it should be ignored. The position

might be different for a closed area of sea into which dilute radioactive product is constantly fed, such as the by-product of cooling or reprocessing at BNFL Sellafield.

647. We are equally sure that there was no widespread deposition on land from any of the Grapple detonations. If there had been it would have been noted in Geiger counters and other instruments as a sources of radioactivity well above the low background radiation on the island.

648. We are also sure that the possibility of significant dry deposition on the part of the island where each of the veterans was located during the tests is remote. Once the cloud had ascended into the stratosphere after a few hours, the micron sized radioactive particles would not be able to fall on the island and will have been dispersed. The expert assessment of the possibility of dry deposition during the short period when the cloud remained in the troposphere from a starting point of 8,000 feet and rising was that the prospect was remote. Gravitational deposition seems equally unlikely, and if a particle was large enough to fall to earth, the expert evidence suggests that it would be too large to be inhaled. If such a large particle existed of a nuclide with a long half-life, the overwhelming likelihood is that it would have been detected either in the random environmental monitoring that did take place after Grapple Y and other detonations or in the clean-up monitoring of subsequent years.

649. Although the conclusion that such a possibility is remote does not as a matter of logic eliminate all risk of it occurring, the focus of an examination of a real possibility of an exposure pathway at CI following Grapple Y has therefore been wet deposition through rainout.

650. The available evidence suggests that such a pathway is highly improbable, in the light of the following:

    (i) We are sure that we can rely on the meteorological report as an accurate account of what it records. Rainfall was recognised to be an important issue. We have no doubt that a detonation of the largest and most powerful weapon of the whole series would have ensured that particular attention was paid to rainfall and the risk of rainout.

(ii) There was no rain falling at the weather station on the day of Grapple Y. It is implausible that this was an uniquely dry location that day, although the possibility of local rainfall elsewhere cannot be eliminated.

(iii) If there was rainfall in the northern coast of CI (where all the CI veterans were located at the time of detonation) within a few hours of detonation, it would have been capable of observation from the meteorological station as mostly within a radius of five kilometres on a flat coral island.

(iv) The fact that adjacent rainfall would have been recorded hourly after the detonation suggests that any such rainfall that may have occurred but was not recorded would have been for a short period i.e. less than an hour.

(v) The adjacent rainfall recorded at 11.00 GMT and any local rainfall that was observed by the veterans and others in the statements made in the context of the litigation from 2007 onwards was most likely low/medium level cumulo-nimbus rainfall as those were the clouds observed. Such rainfall would not have been above the height of the radioactive cloud and been the source of rainout.

(vi) High level rain at the height of or above the radioactive cloud or a rain event caused by the cloud itself is highly unlikely. It was not recorded on the video of the event. It was not observed by the meteorologists at the weather station, or the scientists and commanding officers at the Joint Command Centre or other nearby locations. Such rainfall is more likely to be spread over a wider area than a purely local shower. It could and would have been seen and reported as an issue of concern.

(vii) The absence of any record of such a form of rain in any of the records of the event, when rainout was known to be a potential source of hazard to the personnel on the island, speaks louder and more reliably than retrospective descriptions in the witness statements, unsupported by contemporary records and capable of being the subject of distortion of memory in the light of what was known or suspected as a source of risk in 2007.

(viii) This mechanism requires not merely rain to have fallen through the cloud, but the cloud itself to have been located over the northern part of the island.

The two factors pull in different directions. If the rain fell within an hour of detonation and the cloud was still on its journey from 8,000 to 50,000 feet, it would not have had the opportunity to spread out from 8000 feet diameter on detonation to its greatest extent before rain fell through it. It was thus less likely to be over the northern shore of the island, and the medium and high level winds were not blowing that direction. If the rain was later and the cloud was at or close to its maximum diameter at 55,000 feet, and thus a greater possibility that it or a part of it was over the island, then it is less likely that the rain would have started at this height.

651. Nevertheless, as we have indicated, remote as the possibility of exposure through contaminated rainfall is, it cannot be excluded altogether as a possibility. The question remains whether there is a reasonable possibility that it occurred to any significant degree so that it might (in combination with any remote possibility of dry deposition) have resulted in exposure to a dose that was injurious to health.

Maximum dose at Christmas Island

652. In the light of these conclusions we now return to the issue of dose at Christmas Island. We have already explained our reasons for accepting Mr Hallard's approach to dosimetry and rejecting the submissions to the effect that:

    (i) He was using the wrong model.

    (ii) He started from the false premise that the sticky papers gave an accurate assessment of dose.

    (iii) The calculation of dose was not possible or appropriate and the determination of the appeal should have been based on the calculation of the possible risks raised in the 2013 evidence.

653. We have concluded that the assumption of a generalised radioactive deposition throughout the CI Grapple tests of 16 Mbq/m^2 was, as intended, a massive overestimate of anything that could conceivably have been deposited by any or all pathways.

654. We reserved for further consideration and evaluation two topics arising from the cross-examination and final submissions of the appellants, inhalation/ingestion of

radioactive rain after Grapple Y before it hit the ground and become an exposure source by re-suspension and/or the uncertainties inherent in calculating internal dose at low levels of exposure. We here also consider Dr Busby's further written submissions on Carbon-14

Carbon-14

655. As we have noted at [345] to [346] at our request a calculation has now been performed by Mr Hallard for carbon-14, and demonstrates that it has no material impact on dose relevant to the issues in these appeals. We have noted the BS appellants' response to these calculations. We consider that for the reasons that Mr Hallard gave in his answers, UNSCEAR data is a legitimate source of assistance. Mr Hallard as ever applied a highly precautionary approach and the trivial contribution to dose indicates that the point raised in the hearing by Dr Busby and his subsequent written submissions provide no plausible evidence for undermining the maximum estimates of dose made.

Mechanics of rainfall ingestion

656. This leaves the question of direct or indirect inhalation/ingestion of radioactive rain. We have set out in full Mr Hallard's answers to the questions on this issue. We conclude that they reflect both common sense and such empirical experience as has been noted.

657. This is not an issue that causes the Tribunal to have any doubt as to the robustness of the calculation and the assumptions made. If there was radioactive rain falling on the north of the island shortly after the detonation then we think that the possibility of people drinking it in or breathing it would be marginal. The rain could not have fallen for any significant duration of time without being observed. If the New Zealand sailors were alert to the risk of rain fallout, as they appear to have been, we do not consider it a credible proposition that the military personnel based on Christmas Island itself, in connection with these tests, would have been any less aware. It is unlikely to the point of incredulity that any rain that might have fallen from the radioactive cloud would be a source of attraction and attempted ingestion by military personnel located to the north coast of the island for their own safety.

658. In the light of the mechanics of ingestion and the duration of any opportunity we are sure that it is not a plausible source of a significant amount of rain ingested. We are further sure an appropriate re-suspension rate was used to calculate the impact on dose of any radioactive deposition that had come to the ground through rainfall.

659. There can be no scientific precision on such matters. Mr Hallard took the view that the imponderable factors were such that the safest course was to take the highest conceivable degree of Bequerel deposition per square metre, assuming an hour's rainfall and apply to that deposition the conservative re-suspension rate of a factor of 10-^{4}, even though this was a humid tropical island in the rainy season and not a dusty dry Australian desert. We consider these assumptions and overestimations entirely reasonable in all the circumstances, and more than sufficient to accommodate the risk of a chance inhalation of radioactive particles suspended in rain. We are sure that it would have been ludicrous to apply a multiplier of 10 to obtain the concentration of 160M Bq/m^3.

660. As we have explained, some support for these conclusions can be obtained from both the measurements found from the (probable) rainfall deposition at Vaskess Bay and the information to be derived from the Shackleton data. We recognise that there are many imponderables about applying the Shackleton data of intensity of radioactive deposition in the air to deposition and re-suspension on the ground.

661. We are also conscious that if the Shackleton was sampling the radioactivity of the stem rather than the main cloud, it is estimated that this only represents one tenth of the materials following deposition. It does, however, give a real measurement of radioactivity in the air an hour after deposition, from the part of the cloud that did pass at low level over the south of the island. Manipulating the data to take account of these factors still indicates that the 16 million bequerel figure is a vast overestimate and ample to accommodate all other factors. This degree of overestimation gives ample room for all the uncertainties in the estimate of dose that we have identified above and indeed any other theoretical possibilities that we have not expressly considered.

662. Dr Busby in his written submissions responding to the further answers provided at our request took a wholly different point about the Shackleton data and suggested that the fact that it was assumed that the aircraft had been affected by contamination

from an earlier flight that day through the cloud had not been taken into account. We conclude, first that this observation was not explored at the hearing when it might have been; second, that contamination from an earlier flight through radioactive cloud is readily explained and addressed in the decontamination evidence and third misses the point that the relevance of the Shackleton data is to give a reading of the radioactivity in the stem cloud that had passed over the southern uninhabited part of the island from where deposition may well have occurred. We are sure that Dr Busbsy's comments in no way detract from the reliability of Mr Hallard's assumptions as to maximum dose.

663. There are other uncertainties that we have identified at [353] that we have to consider, but taking every one of them as a whole we are sure that Mr Hallard's deliberately exaggerated estimations of possible dose amply accommodate every potential uncertainty. There is no conceivable possibility on the evidence that we have seen of greater radioactive deposition in Gys having occurred than given in his calculations.

664. Equally, we consider that in the case of Mr Battersby at Maralinga, Mr Hallard has made robust assessments that take into account every conceivable possibility of exposure.

Overall conclusions

665. In the previous parts of this determination, we have identified facts and issues of which we are sure and by contrast such facts and hypotheses where there is or may be doubt. The ultimate focus of this decision is whether the health conditions that were diagnosed and made the subject of the claims for a war pension were caused by radiation exposure to the veterans from their military service. We now come to stage (v) as described by Mr Justice Charles at paragraph 103(v) of his decision:

> 'in the light of all the evidenced and argument and so, on an overview or assessment in the round, evaluate the claimant's case to determine whether he has or has not satisfied the article 41(5) test.
> It is at stage (v) that the decision maker will form views that can be expressed by reference to the circumstances of the given case on whether the possibilities (and effective certainties) relied on by the claimant found a reasonable doubt. ….At that stage it may be that the decision will be that the combined effects of the possibilities carried forward do nor found a reasonable doubt because for example the combination of those possibilities is too far-fetched.'

666. In Part Seven of this determination we examined each of the medical conditions and noted that a number of them were either not known to medical science as radiogenic at all or only where the levels of exposure exceeded an equivalent or effective dose of 500 mSv.

667. Some of these claims can only be described as fanciful: Mr Hughes was only on CI when the Malden Island tests were conducted 700 miles away. His belief that a real preliminary Grapple X test was conducted whilst he was there is mistaken and any practice activity that may have taken place while he was on CI did not result in radiation being released. The estimate of his external exposure is a tiny fraction of the background radiation to which he has been exposed.

668. Equally Mr Smith was not on CI at the time of any detonation, and his case rests upon radioactive residues in the dust transferred to him through retention in the hair of servicemen who had been present at or closer to these detonations whose hair he cut and then he inhaled the dust released by this activity. We are sure that this is not a plausible hypothesis.

669. Excluding, Mr Abdale's claim to a pension on account of cataracts, each of the other appellants submits that there is reasonable doubt as to causation because:

    (i) Mr Hallard's estimates of dose are not sufficiently conservative given the remaining uncertainties as to radioactive deposition at CI and the exposure possibilities for Mr Battersby at Maralinga and the findings of the Wahab study;

    (ii) Developments in radiobiology and/or unanswered questions resulting from environmental studies throw doubt on the accepted medical wisdom as to the level of dose needed to have a biological impact.

670. For the reasons we have already given at Part Five and in the preceding section of this Part of the determination we are sure that Mr Hallard's estimates were the maximum conceivable estimates of dose and in reality inflated the possibility of exposure to a considerable extent. They were therefore robust or conservative assessments. For the reasons given in Part Six, we are satisfied that neither the epidemiological studies nor the Wahab results throw any reasonable doubt on the upper limits of Mr Hallard's cautious assessments.

671. For the reasons given in Part Seven, we are sure that the vast majority of these conditions could not be caused by radioactive exposure at less than 500 mSv.

672. Taking all these different strands of our reasons together, we are sure, looking back at all the issues in these complex cases, that these estimates more than amply accommodate any of the inherent uncertainties about a conclusive assessment of dose from radiation pathways on CI and Maralinga. Put simply, even on the improbable hypothesis that there was some undetected radioactive deposition on CI through rainout or dry deposition, it could not conceivably have been at anything approaching the amount that could have caused the kind of medical conditions for which the veterans made their claims. We are sure (and therefore have no reasonable doubt) that the combination of possibilities is, in every case, save for Mr Abdale's cataracts, too far fetched.

673. We recognise that reasonable doubt is not measured in statistical chance but is an overall legal conclusion. 'Highly improbable' is not the same as 'sure of no possibility'. Depending on circumstances a chance of around 5% might be capable of creating a reasonable doubt. We do not accept Mr Ter Haar's fall-back submission in the case of Mr Abdale's bladder cancer that a risk of 0.08% itself was a reasonable doubt as the Tribunal can never be sure that his case was not the one where causation existed. Reasonable doubt is a legal and not a scientific term. It amounts to something more than *any* possibility of doubt; reasonable is an ordinary word that takes out of the equation fanciful and purely theoretical possibilities.

674. We now apply these general conclusions about dose exposure to the medical conclusions we have reached for each veteran.

675. Abdale: for reasons we have explained in Part Seven at [452] to [463] and [474] we cannot be sure that Mr Abdale's cataracts were not caused by exposure during his service history, despite the realistic arguments against this being probable. His appeal with respect to cataracts will accordingly be allowed. His appeal with respect to bladder cancer is dismissed for the reasons given at [464] to [472] and [475].

676. Beeton: for the reasons we give at [480] to [487] this appeal is dismissed.

677. Butler: for the reasons we give at [590] to [593] this appeal is dismissed.

678. Hatton: for the reasons given at [575] to [579] this appeal is dismissed.

679. Hughes: for the reasons given at [601] to [609] this appeal is dismissed.

680. Lovatt: for the reasons given at [614] to [616] this appeal is dismissed.

681. Pritchard: for the reasons given at [549] to [559] this appeal is dismissed.

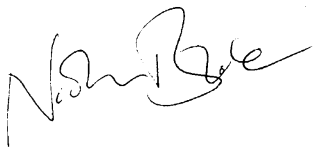
682. Selby: for the reasons given at [565] to [571] this appeal is dismissed.

683. Shaw: for the reasons given at [620] to [622] this appeal is dismissed.

684. Sinfield: for the reasons given at [530] to [536] this appeal is dismissed.

685. Battersby: for the reasons given at [514] to [523] this appeal is dismissed.

686. Smith: for the reasons given at [493] to [510] this appeal is dismissed.



Mr Justice Blake

Dated: 14 December 2016

Decision issued to the parties on December 2016

Annex A

The Tribunal's ruling of 15 June 2016 on admissibility of BS evidence as expert evidence

RULING

Introduction

1. These appeals are re-hearings of appeals against the assessment made on behalf of the respondent that no award should be paid in respect of medical conditions said to be have been caused in part by exposure to ionising radiation in the course of their military service at either the Maralinga testing site in Australia and Christmas Island (now part of Kiribati) between 1956 and 1960. The appeals were remitted following the determination of the Upper Tribunal Administrative Appeals Chamber (UTAAC) with Mr Justice Charles presiding.

2. The appeals have been the subject of extensive case management in June 2015, December 2015 and May 2016. Those directions included the preparation of expert evidence and bundles in good time for the hearing of these appeals starting on 13 June. Last week we became aware of an issue which arose with respect to a document produced by Mr Williams that was a plan compiled by him seeking to reconstruct wind direction and other matters at the time of material events. It is common ground that Mr Williams has no expertise in meteorology.

3. In further written directions (Appendix 1) the Tribunal directed that there be a preliminary hearing of the issue. Direction 4 stated the understanding of the meaning of a direction made by Charles J with respect to excluding evidence from Dr Busby. This resulted in an application being made to vary direction 4 to permit witnesses to rely on articles authored or co-authored by Dr Busby.

4. Having heard argument on 13 June we announced our decisions namely:

   i) The chart prepared by Mr Williams should be excluded from the bundle.

   ii) The direction in respect to Dr Busby's expression of opinions on issues arising in this appeal in published articles would remain, subject to

relaxation in the case of any specific article where another expert proposed to rely on it for objective reasons.

5. We now give reasons for our decision.

Expert evidence in the Tribunal

6. The common issue in each of these appeals is the possibility of causation of damage by exposure to ionising radiation on the basis of reliable evidence. Applying the guidance provided by the UTAAC, the ultimate issue when all relevant information has been received and in so far as is possibly evaluated, whether there is reasonable doubt on the question of causation in each appeal. Undoubtedly the issues that are ventilated in the parties' statements of case are complex and difficult. Their exploration has already resulted in an extensive hearing before the F-tT WPAFCC where Dr Busby made a witness statement although in the end was not called to give evidence. He is a campaigner against nuclear atmospheric testing and any other activity civil or military that releases radioactive material into the atmosphere.

7. A ground of appeal to the UTAAC was whether the F-tT of its own motion should have called Dr Busby to assess his evidence. That ground was rejected. There was also an issue raised before the UTAAC hearing about whether Dr Busby could combine the role of campaigner and objective expert witness to assist this Tribunal in the challenging task it faces. The UTAAC heard Dr Busby give evidence.

8. In the course of his judgment in Abdale and others v Secretary of State for Defence [2014] UK AAC 477; [2015] AACR 20 Charles J first dealt with the argument that the FtT had acted unfairly. Having dealt with that he then said:

> '234. Further and in any event, having regard to my conclusions under the next heading I consider that if Dr Busby had been called and so been cross-examined the F-tT would or should have found him to be a witness on whom they could place no reliance when his views differed from or were not supported by those of the other experts called and relied on by the appellants.

235. I acknowledge that this has not been the view of other F-tTs in the past but in those cases his expertise and impartiality were not as I understand it challenged at all or in the way that they would have been before this F-tT.'

9. He then considered whether Dr Busby was eligible to give expert evidence in the remitted appeals and the following passage explains his conclusions:

'**Dr Busby's suitability to give expert evidence in cases of this type (ie ones raising issues relating to the existence, impact and effect of ionising radiation)**

237. The BS appellants urged me not to deal with this because of its impact on future cases where they argued it should be left to the trial court or tribunal. In support of that they urged on me the difficulties facing veterans in advancing their cases against the Secretary of State. I fully accept the existence of those difficulties but in my view I must deal with this issue because it is relevant to the directions I will give concerning the re-hearing of these appeals and it would be wrong not to do so given the time, cost and effort expended on this issue in this case which would have to be repeated if it was left to other trial courts or tribunals.

238. Dr Busby was cross-examined thoroughly and with scrupulous fairness on his suitability to give expert evidence in cases of this type. His oral evidence spread over three days.

239. I am sorry to have to conclude that the upshot of that evidence was that to my mind it is clear that he is not a suitable person to give expert evidence in cases of this type. I am sorry because I do not dispute that:

(i) Dr Busby is passionate in his views and believes them to be true and based on valid science and reasoning,

(ii) on their face some of them appear to have some force,

(iii) he has been invited to sit in and has sat on relevant bodies dealing with exposure to radiation (eg the Committee Examining Radiation Risks and Internal Emitters CERRIE – see paragraphs 226 to 231 of the F-tT's Decision) and so it has been recognised by others that he has a contribution to make to the issues that arise in cases of this type,

(iv) there is some force in Dr Busby's view that the confirmation by the National Radiological Protection Board (NRPB) of the majority view in the CERRIE

report (Dr Busby and another wrote a minority report) is in effect confirmation by that majority and he is arguing against an establishment view in a developing area, and

(v) as supported by his participation in CERRIE and him giving evidence in other cases before the F-tT in this jurisdiction his lack of qualifications does not constitute a bar to him giving expert evidence based on his research and experience albeit that on some aspects he may have to defer to others with particular expertise, qualifications or experience.

240. The reason why I have concluded that he is not a suitable person to give evidence in cases of this type is that as he accepts and is obvious he is a campaigner and activist on issues relating to radiation and radiation risks (amongst other things) and in my view his oral evidence demonstrated clearly that:

(i) he cannot put aside his passion and beliefs as such and act in a dispassionate way as an expert witness,

(ii) this is demonstrated by his descriptions of his approach to his role as an expert witness, and

(iii) too frequently his campaigning or activist hat or mind-set leads him to make unwarranted assumptions or to jump to unwarranted conclusions because he has not checked underlying material with appropriate thoroughness, and wishes to support a conclusion.

241. This conclusion is founded on the duties and responsibilities of an expert witness set out set out in *National Justice Compania Naviera SS v Prudential Assurance Co Ltd (The Ikarian Reefer)* [1993] 2 Lloyds's LR 68 at 81–2. This is a well known summary that has been approved and applied on a number of occasions. Of particular relevance here are:

(i) the evidence should be, and be seen to be, the independent product of the expert uninfluenced as to form or content by the exigencies of litigation, and

(ii) it should provide assistance to the court or tribunal by way of an objective unbiased opinion.

242. The BS appellants argued that all of these problems could be addressed by him giving a declaration or statement of impartiality such as that suggested in *Toth v*

Jarman [2006] EWCA (Civ) 1028, [2006] 4 All ER 1276 and that when Dr Busby had done this the issues raised should go only to weight and not to admissibility. I acknowledge this important distinction but I do not agree because the evidence shows that the problems relating to Dr Busby's partisan approach to litigation of this type and his impartiality and objectivity are far too ingrained.

243. Dr Busby's evidence was transcribed and I imagine that the Secretary of State would make it available together with the two cross-examination bundles should the issue of Dr Busby's suitability to give expert evidence in cases of this type arises in another tribunal or court.

244. I shall confine myself to extracts from evidence that have founded my conclusion but record that in reaching it I have had regard to his evidence as a whole and have not forgotten that he was in the witness box a long time.'

Specific examples are then given and Charles J observes in the course of reciting them:

'All this shows that he is stuck in a groove as an activist and campaigner and is either not capable of acknowledging or refuses to acknowledge that parts of his evidence are demonstrably wrong or based on a false or inadequately researched premise (see the examples in [231] above).'

10. Following the judgment being handed down, on 4 December 2014 he directed at 1 (x) that 'Dr Busby may not give expert evidence (whether in writing, orally or otherwise) at the remitted hearings'. Dr Busby sought unsuccessfully to challenge this direction by judicial review.

11. Dr Busby then indicated that he would be acting as representative for two of the appellants in these remitted proceedings. This has been the case ever since the remitted hearing has been case managed in this chamber from June 2015. There are no rules restricting who may represent an appellant in this chamber.

12. The Tribunal has from time to time pointed out that the ground rules for the new hearing had been set by Charles J and the direction precluded introducing his expert opinion into the appeal whether directly by way of a witness statement or indirectly by way of expressing an opinion in the legal submissions or by citation of his own views relying on articles he has written on the issues that are live in the

appeal. This required the Tribunal to direct that in the case of the BS appellants that an amended statement ofcase be served to exclude references to such opinions.

13. The normal rule in the Tribunal is that there are no rules governing the admissibility of evidence, by contrast with the codes applied in the civil courts: see rule 15 (2)(a) of the Tribunal Procedure (First-tier Tribunal) (War Pensions and Armed Forces Compensation Chamber) Rules 2008 SI 2008/2686 (the Rules). This is reflected Hampshire CC v JP [2009] UKUT 239 (AAC) [2010] AACR 15 at [34].

> 'In tribunals, such strict rules of evidence do not usually apply but the weight to be attached to an opinion expressed on a matter beyond the professional expertise of a witness is likely to be limited and reliance on such an opinion is likely to require some explanation by a tribunal.'

14. However, the Tribunal has ample case management powers in rule 5 (1) and (2). Without prejudice to the generality of these powers under rule 15 (1) the tribunal may give directions as to the issues, and nature of the evidence it receives, including at (1) (c) 'whether the parties are permitted or required to provide expert evidence'. There are sanctions for non-compliance with directions.

15. It is plain that these powers are sufficient to refuse to permit expert evidence to be adduced unless it is accompanied by a declaration that is equivalent in effect to the provisions of CPR 35 (10) and the Practice Direction 35PD that supplements this rule.

16. We note that in the Immigration and Asylum Chambers, there is a Practice Direction issued by the Senior President of Tribunals, para 16.1 of which is to the effect that expert evidence in the chambers of the First-tier and Upper Tribunal shall meet these requirements.

17. Our attention has been drawn to a decision of the F-tT Tax Chamber Chandanmal v HMRC [2012] UKFTT 188 (TC) where such a requirement was directed and it was considered that there was every advantage in so doing and no disadvantage.

18. Clearly Charles J was exercising such a case management power in the direction that he made about Dr Busby. In his ruling he referred to the well-known principles applicable in the civil courts governing receipt of expert evidence where permission is needed to rely on such evidence. In his judgment in GMC v Meadow [2006] EWCA Civ 1390; [2007] 1 QB 462 Sir Anthony Clarke MR said as follows:

> '70 It is common ground between the parties that the relevant principles to be adopted by expert witnesses are summarised by Cresswell J in *The Ikarian Reefer* in the passage quoted in paragraph 21 above. I extract these principles as being of particular relevance:
>
> 1. Expert evidence presented to the court should be, and should be seen to be, the independent product of the expert uninfluenced as to form or content by the exigencies of litigation.
>
> 2. An expert witness should provide independent assistance to the court by way of objective unbiased opinion in relation to matters within his expertise. An expert witness in the High Court should never assume the role of an advocate.
>
> 3. An expert witness should state the facts or assumptions upon which his opinion is based. He should not omit to consider material facts which could detract from his concluded opinion.
>
> 4. An expert witness should make it clear when a particular question or issue falls outside his expertise.
>
> 5. If an expert's opinion is not properly researched because he considers that insufficient data is available, then this must be stated with an indication that the opinion is no more than a provisional one. In cases where an expert witness who has prepared a report could not assert that the report contained the truth, the whole truth and nothing but the truth without some qualification, that qualification should be stated in the report.
>
> 71. It is in my opinion of the utmost importance that an expert should only give evidence of opinion which is within his particular expertise and that, where a statement, whether made in writing or orally, is outside his expertise, he should expressly say so. If, for example, it depends upon work done or opinions expressed by others, that work or those opinions should be identified in the statement, so that their validity can be ascertained by the parties to the

> proceedings or by the court. All reasonable attempts should be made check the validity of an opinion which is not within the expert's expertise. These are simple precautions which should be taken by experts because of the risk that the opinion might be wrong, with what may be very serious consequences.'

19. We make four observations on the application of these principles to the present appeal:

    i) Although there is no general requirement that a witness of opinion must always meet CPR 35PD criteria, a Tribunal may case manage a case to ensure that it will only give permission for such evidence to be adduced if these criteria are met.

    ii) If the Tribunal exercises its powers to so direct then a person who has no relevant expertise cannot comply with the direction that he should confirm that s/he is acting within the area of expertise. It is only those who have relevant expertise gained by study or experience who have the authority to give opinions on the issue in question (see per Bingham LCJ in R v Robb (1991) 93 Cr App R at 165).

    iii) A person with relevant expertise but whose status as an independent expert is subjugated to a role of advocate and campaigner on the issue in question does not have sufficient authority to be received as an expert by the court or tribunal in question.

    iv) Experts need to state the basis for their opinions to ensure that they are founded on a correct understanding of the facts that may include generally accepted standards of assessment in the relevant expert discipline.

Application

20. We have no doubt that whatever the position may be by way of generality we should ensure that the only expert evidence we receive in this appeal is evidence that meets the common law standards and the provisions of CPR 35PD. As expert evidence is admissible as evidence of opinion, its admission is controlled at common law to ensure only those with sufficient expertise may give it and that the only opinion to be received by those with expertise is an objective and independent

one unprompted by the exigencies of the litigation and based on an objective and transparent basis. Opinions from those who have either no or sufficient expertise in the area on which an opinion is offered is thus irrelevant, incapable of being given any weight and therefore should not be admitted despite the general absence of an admissibility requirement. Second, opinions from those who are not truly independent as regards the issue in the litigation are equally irrelevant and should be excluded.

21. Although the impact of the admission of flawed or inadequate expert evidence may not be the same as in a criminal trial, quality control is of particular importance in this jurisdiction where the assessment of causation of harm caused by ionising radiation is made on the basis of reasonable possibility and the absence of reasonable doubt. For a number of reasons, the defects in expert evidence of debatable quality may not always be explored adequately or at all in the hearing to make a reliable assessment as to weight to be given to it. For these reasons we conclude that the Tribunal should ensure that the opinions it receives in the assessment process should be limited to ones that meet or appear to meet the requisite threshold derived from the experience of the common law.

The meteorological chart

22. Applying these criteria is obvious that with the best will in the world Mr Williams is not an expert in meteorology and his plan is an attempt at giving opinion evidence on contentious matters of fact outside of any special skill.

23. The only argument advanced by Mr Charlton for its admission was that it had been received by the F-tT at the previous hearing. Mr Heppinstall indicated that objection to the F-tT relying on it had in fact been made although not judicially determined.

24. In our view, if this issue had been raised before Charles J he would have applied the principles set out in the case law to this piece of evidence as well. If the evidence remains others will be asked to give assessments of probability of contamination based on it. It has already been shown to be inaccurate in some respects. Having regard to the over-riding objective and the general principles relating to expert evidence, it should be excluded.

Dr Busby's articles

25. Dr Busby is not a witness in the case and has not made a witness statement. He does intend to call five people with varying degrees of expert experience and qualification, on whom he relies to support the hypotheses he proposes to develop in the cases of Messrs Battersby and Smith. Some of these witnesses speak of their view of the expertise of Dr Busby and a number of them cite as the basis for their findings articles published by Dr Busby as sole or joint author.

26. Mr Charlton contended that the direction of Charles J should be narrowly construed to be limited to precluding merely formal witness statements or expressions of opinion in the skeleton arguments and statements of case. It is contended that the direction was limited to evidence that might be shaped by the contingencies of this litigation and not statements made outside it.

27. We do not consider that a narrow reading can or should be given to the direction and the term 'otherwise'. The purpose of the direction is that the Tribunal's assessments are not based on debatable opinions that do not meet the standards of objectivity and independence set out in the principles described above and applied by Charles J. Here all the contentious issues that the appellants wish to raise: reliability of ICRP criteria as to dosimetry, the adequacy of epidemiological studies of groups possibly exposed to low level radiation and the such like have been the subject of a campaign in which Dr Busby and a number of his witnesses have been party to since long before this litigation arose and many of the articles in question were written. If this material is directly or indirectly the foundation of an expert opinion relied on in these proceedings, it results in the same vice as that which the direction was intended to prevent but without even the possibility of cross-examination of the author.

28. We therefore affirm the written direction given that concludes that Charles J's direction precludes the indirect introduction of Dr Busby's opinions into the decision making process. At the same time, we recognise that there must be room for some flexibility in the case of jointly authored articles that cite other references than Dr Busby alone, and where the witness citing it may be able to express

agreement with Dr Busby's opinions on the basis of their own independent research and expert experience.

29. The objective basis for individual opinions can be explored in the evidence rather than by way of pre-evidence admissibility inquiry, but it appeared to us to be necessary at the outset of this hearing to spell out our approach to the direction and the indirect use of Dr Busby's opinions in advance of the evidence being called, so that thought can be given to the identification of the objective nature of such opinions untainted by what may be Dr Busby's personal and doubtless sincerely held views on the issues that he has been campaigning about for a number of years.

The Hon Mr Justice Blake

Dr Jane Rayner

Isabel McCord

15 June 2016