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

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.

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 **s**moking, 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.

---

[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.