Specialist Medical Review Council

Reasons for Decisions

Section 196W
Veterans' Entitlements Act 1986

Re: Statements of Principles Nos. 67 and 68 of 2001
In Respect of Chronic Lymphoid Leukaemia
Matter No. 2002/3
Requests for Review Declarations Nos. 9 and 10

SUMMATION

1. In relation to the Repatriation Medical Authority (the RMA) Statement of Principles No. 67 of 2001 in respect of chronic lymphoid leukaemia and death from chronic lymphoid leukaemia, made under subsection 196B (2) of the *Veterans' Entitlements Act 1986* (the VEA), the Specialist Medical Review Council (the Council) declares under subsection 196W (4) of the VEA, that it is of the view that there is sound medical-scientific evidence on which the RMA could have relied to amend Statement of Principles No. 67 of 2001. The Council accordingly directs the RMA to:

(a) amend Statement of Principles No. 67 of 2001 by including as a factor exposure to extremely low frequency electro-magnetic radiation at an exposure level, duration of exposure and period of time from exposure to onset of disease to be ascertained by the RMA and whether that exposure should be specified as whole or part body exposure;

(b) carry out an investigation to find out if there is new information available about:

(i) how chronic lymphoid leukaemia may be contracted, or death from chronic lymphoid leukaemia may occur; or

(ii) the extent to which chronic lymphoid leukaemia or death from chronic lymphoid leukaemia may be war caused;

and in particular, to find out whether there is sound medical-scientific evidence to justify the amendment of Statement of Principles No. 67 of 2001 to include as a factor or factors exposure to:

(A) radio frequency radiation;

(B) smoking tobacco cigarettes or pipe-smoking or exposure to environmental tobacco smoke (passive smoking); and

(C) benzene; and

(c) take into account in that investigation the sound medical-scientific evidence previously considered by the RMA; the articles identified in Appendices E - H as being of potential relevance (but not before the RMA at the relevant times); and any new body of sound medical-scientific evidence disclosed by the investigation.

2. In relation to the RMA Statement of Principles No. 68 of 2001 in respect of chronic lymphoid leukaemia and death from chronic lymphoid leukaemia, made under subsection 196B (3) of the VEA, the Council declares under subsection 196W (5) of the VEA, that it is of the view that the sound medical-scientific evidence available to the RMA at the time it determined, amended or last amended the Statement of Principles was insufficient to justify an amendment of Statement of Principles No. 68 of 2001 by including as a factor or factors exposure to:

(a) Radiation;

(i) Extremely low frequency radiation;

(ii) Radio frequency radiation;

(iii) Ionizing radiation; and

(iv) DC Batteries;

(b) Smoking tobacco cigarettes or pipe-smoking or passive smoking;

(c) Asbestos;

(d) Benzene;

(e) Petroleum Additives;

(f) Chromosomal Aberration;

(g) Viral and Bacterial Infection;

(h) Chemicals (particularly cleaning fluids and solvents); and

(i) Acetone.

3. The Council is of the view that there is a considerable body of relevant sound medical-scientific evidence which was not before the RMA at the relevant times. Accordingly, the Council recommends that the RMA carry out an investigation to find out if there is new information available about:

(a) how chronic lymphoid leukaemia may be contracted, or death from chronic lymphoid leukaemia occur; or

(b) the extent to which chronic lymphoid leukaemia or death from chronic lymphoid leukaemia may be defence-caused;

and in particular, to find out whether there is sound medical-scientific evidence to justify the amendment of Statement of Principles No. 68 of 2001 to include as a factor or factors exposure to:

(i) extremely low frequency electromagnetic radiation;

(ii) radio frequency radiation;

(iii) smoking tobacco cigarettes or pipe-smoking or passive smoking; and

(iv) benzene.

4. Any investigation should take into account the sound medical-scientific evidence previously considered by the RMA; the articles identified in Appendices E - H as being of potential relevance (but not before the RMA at the relevant times); and any new body of sound medical-scientific evidence disclosed by that investigation.

THE SPECIALIST MEDICAL REVIEW COUNCIL

5. The Council is a body corporate established under section 196V of the VEA, and consists of such number of members as the Minister for Veterans' Affairs determines from time to time to be necessary for the proper exercise of the functions of the Council as set out in the VEA. The Minister must appoint one of the Councillors to be the Convener.

6. When a review is undertaken of a Statement of Principles made by the RMA, the Council is constituted by 3 to 5 Councillors selected by the Convener. When appointing Councillors, the Minister is required to have regard to the branches of medical science expertise which would be necessary for deciding matters referred to the Council for review.

7. Dr Jonathan Phillips FRANZCP is the Convener of the Council. The other members of the Council were:

(i) Associate Professor Timothy Hughes, Deputy Head and Director of Research, Division of Haematology at the Institute of Medical and Veterinary Science (South Australia); and a clinical Associate Professor, of the Department of Medicine at the University of Adelaide;

(ii) Dr Charles Guest, Medical Director of the Health Protection Service of the ACT Department of Health and Community Care; and Visiting Fellow at the Australian National University, National Centre for Epidemiology and Population Health;

(iii) Dr David Joske, Head of Department of Haematology and Director of Bone Marrow Transplantation, Sir Charles Gairdner Hospital; Consultant Haematologist, Western Australia Centre for Pathology and Medical Research 'Pathcentre'; and Clinical Senior Lecturer, Department of Medicine, the University of Western Australia; and

(iv) Dr Michael Izard, a partner with Radiation Oncology Associates at the Mater Misericordiae Hospital; a Clinical Lecturer in the Department of Medicine with the Northern Clinical School, University of Sydney; and a member of the Radiation Advisory Committee of the NSW Environment Protection Authority, as the representative of the Royal Australian & New Zealand College of Radiologists, Faculty of Radiation Oncology.

THE LEGISLATION

8. The legislative scheme for the making of Statements of Principles is set out in Parts X1A and X1B of the VEA.

9. The functions and powers of the Council must be seen in light of the function and purpose of Statements of Principles in the scheme of the VEA. The significance of Statements of Principles to claims under the VEA for pensions in relation to eligible service is apparent from sections 120A and 120B of the VEA. Section 120 is also of importance.

10. Fundamental to Statements of Principles is the concept of 'sound medical-scientific evidence', which has been defined in section 5AB(2) of the VEA. Information about a particular kind of injury, disease or death is taken to be sound medical-scientific evidence if:

(a) the information:

(i) is consistent with material relating to medical science that has been published in a medical or scientific publication and has been, in the opinion of the Repatriation Medical Authority, subjected to a peer review process; or

(ii) in accordance with generally accepted medical practice, would serve as the basis for the diagnosis and management of a medical condition; and

(b) in the case of information about how that injury, disease or death may be caused - meets the applicable criteria for assessing causation currently applied in the field of epidemiology.

11. The functions of the Council are set out in section 196W of the VEA. In this case, the Council was asked (under section 196Y of the VEA) by a person eligible to make a claim for a pension, to review the contents of:

(a) Statement of Principles No. 67 of 2001 in respect of chronic lymphoid leukaemia and death from chronic lymphoid leukaemia, being a Statement of

Principles determined by the RMA under section 196B(2)[1] of the VEA ('the reasonable hypothesis standard'); and

(b) Statement of Principles No. 68 of 2001 in respect of chronic lymphoid leukaemia and death from chronic lymphoid leukaemia, being a Statement of Principles determined by the RMA under section 196B(3)[2] of the VEA ('the balance of probabilities standard').

12. In conducting its review, the Council must review all the information that was available to the RMA at the time it determined, amended, or last amended the Statements of Principles and is constrained to conduct its review by reference to that information only[3].

13. Under section 196W of the VEA, the Council can only reach the view that a Statement of Principles should be amended on the basis of sound medical-scientific evidence.

BACKGROUND

14. On 12 September 2001, the RMA under subsections 196B(2) and (3) of the VEA determined Statements of Principles Nos. 67 and 68 of 2001 in respect of chronic lymphoid leukaemia and death from chronic lymphoid leukaemia.

15. On 17 September 2001 and 18 September 2001, in accordance with section 196D of the VEA and sections 46A and 48 of the *Acts Interpretation Act 1901* the Statements of Principles were tabled in both the House of Representatives and in the Senate.

16. On 19 September 2001 the making of those instruments was notified in the Gazette (No. 37, p. 2734).

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[1] If the Authority is of the view that there is sound medical-scientific evidence that indicates that a particular kind of injury, disease or death can be related to:

(a) operational service rendered by veterans: or
(b) peacekeeping services rendered by members of Peacekeeping Forces; or
(c) hazardous service rendered by members of the Forces;

the Authority must determine a Statement of Principles in respect of that kind of injury, disease or death setting out:

(d) the factors that must as a minimum exist; and
(e) which of those factors must be related to service rendered by a person;

before it can be said that a reasonable hypothesis has been raised connecting an injury, disease or death of that kind with the circumstances of that service.

[2] If the Authority is of the view that on the sound medical-scientific evidence available it is more probable than not that a particular kind of injury, disease or death can be related to:

(a) eligible war service (other than operational service) rendered by veterans; or
(b) defence service (other than hazardous service) rendered by members of the Forces;

the Authority must determine a Statement of Principles in respect of that kind of injury, disease or death setting out:

(c) the factors that must exist; and
(d) which of those factors must be related to service rendered by a person;

before it can be said that, on the balance of probabilities, an injury, disease or death of that kind is connected with the circumstances of that service.

[3] *Vietnam Veterans' Association (NSW Branch) v Specialist Medical Review Council and Anor* (Full Federal Court decision) (2002) 72 ALD 378 at paragraph 35 per Branson J

17. An application dated 24 October 2001 for review of Statements of Principles Nos. 67 and 68 of 2001 was received by the Council on 29 October 2001. Specifically the application was concerned with the decision of the RMA of 12 September 2001 not to add to Statements of Principles Nos. 67 and 68 of 2001 in respect of chronic lymphoid leukaemia an additional factor or factors being exposure to radiation, electricity and magnetism, petroleum and gasoline products and additives, cleaners and solvents, other various chemicals, chromosomal aberration, and not to amend the factor in relation to HTLV-1 virus to include other viral and bacterial infections.

18. The Council held a meeting for the purposes of this review, and heard oral submissions complementing written submissions on 25 November 2002.

19. The applicant submitted a written submission and made an oral submission to the Council complementing the written submission at the Council's meeting on 25 November 2002.

20. A person entitled to make a submission submitted a written submission and made an oral submission to the Council complementing the written submission at the Council's meeting on 25 November 2002.

21. An expert in the field submitted a written submission and made an oral submission to the Council complementing the written submission at the Council's meeting on 25 November 2002.

22. The Repatriation Commission made a written submission. Dr Jon Kelley, representing the Repatriation Commission, made an oral submission to the Council complementing the written submission at the Council's meeting on 25 November 2002.

Applicant's submission

23. The applicant made an extensive written submission to the Council. The submission had a broad compass, some of which was not able to be considered by the Council. For example, the Council is precluded by section 196ZA of the VEA from considering submissions on a legal matter.

24. The applicant also sought to argue that the Council was at liberty to consider information other than that which had been actually before the RMA. This is not the case, the submission being contrary to the decisions of Spigelman CJ in the New South Wales Court of Appeal in *Repatriation Commission v Vietnam Veterans' Association of Australia NSW Branch Inc & Ors* (2000) 48 NSWLR 548 and of the Full Court of the Federal Court in *Vietnam Veterans' Association (NSW Branch) v Specialist Medical Review Council and Anor* (2002) 72 ALD 378 (see paragraph 12 and footnote 3 above).

25. The applicant was critical of the sorts of studies relied upon by the RMA when determining Statements of Principles generally, submitting that studies of civilian populations did not appropriately replicate or translate to Defence Force personnel. He was particularly critical in this case that the RMA had not awaited the outcome of the

investigation by the Board of Inquiry into the F-111 (Fuel Tank) Deseal/Reseal and Spray Seal Programs (F-111 Deseal/Reseal Board of Inquiry Report).

26. As mentioned above, the Council is constrained to review only that information which was actually before the RMA. Since the F-111 Deseal/Reseal Board of Inquiry Report was not part of the information before the RMA when it determined Statements of Principles Nos. 67 and 68 of 2001 (notwithstanding it pre-dates the determination of the Statements of Principles by the RMA), it was not part of the information reviewed by the Council in this review.

27. The submissions as to the particular factors contended for by the applicant are summarised below.

Petroleum Chemical Additives

28. The applicant submitted that some chemical additives used in petroleum fuels were causative of chronic lymphoid leukaemia. He further submitted that some of those petroleum products were exclusively used in a military context. Jet fuels and kerosene were specifically nominated, as well as additives used by the military in such fuels, for example, distillate oils, petroleums, kerosene and hydraulic oils and their additives. Anti-icing chemicals such as FSII were identified, together with chemicals like toluol, toluene, benzene, xylene, ethyls, ethyl-leads, ethyl-lead glycols, and ethyl glycol products.

Chemicals (Particularly Cleaning Fluids and Solvents)

29. The applicant contended there was evidence that exposure to some chemicals, for example methyl ethyl ketones, could cause alterations to human chromosomes, which in turn could be relevant to the onset of chronic lymphoid leukaemia.

Electrical and Electro-magnetic radiation

30. The applicant submitted that the exposure of Defence personnel to electrical and electromagnetic radiation was qualitatively and quantitatively different from the exposure of people in a civilian environment.

31. With respect to radiation, it was argued that the full spectrum of ultraviolet and infrared radiation was not taken into account, particularly in the context of Defence personnel exposed to radar units and other navigational equipment which does not exist in the civilian environment. It was submitted that the effects of standing close to the output of radiation had been underestimated by the RMA.

32. It was submitted that in contrast, studies concerning civilian exposure to radio-frequency transmissions were limited to exposure to therapeutic x-rays and radiation therapy, low-powered fully enclosed microwave transmissions in cookers and sterilisers, and varied transmissions of lower radio-frequencies from telephone use and two-way radio transmissions.

33. In contrast, it was submitted that Defence personnel are subjected to more intensive usage, including high-powered radio transmissions, radar signals and navigational equipment, and high output of unprotected microwave transmissions. It was further submitted that the inherent requirements of Defence service adversely impacted upon the capacity of Defence personnel to manage the risk of such exposure, for example, by not being able to wear protective clothing.

Chromosomal Aberration

34. It was submitted there were significant materials to justify chromosomal aberration as associated with the onset of chronic lymphoid leukaemia. Reference was again made to the singularity of Defence conditions, and the impact of those conditions upon exposure to the power outputs of equipment peculiar to Defence personnel.

Person entitled to make a submission

35. A written and complementary oral submission were made, in accordance with section 196ZA(1) by a person eligible to make a claim for a pension under parts II or IV of the VEA. The submissions were sourced to both the medical literature and the person's own history of prolonged and high level exposure to radiation and chemicals, without protective clothing, during his Defence service.

36. The person indicated that Dr Nicholls would be making a submission on his behalf as to the posited association between these exposures and the onset of chronic lymphoid leukaemia.

37. He also submitted that the exposure should be considered as a 'toxic cocktail' - an aggregated exposure, as opposed to a series of independent and unrelated exposures.

Dr Nicholls' Submission

38. A helpful submission was made by Dr E. M. Nicholls, a geneticist, in support of the submissions made by the person entitled to make a submission. Dr Nicholls contended that that person's exposure to microwaves, radar systems and other non-ionising radiation equipment during most of his Defence service, with insufficient safety precautions, was 'the primary likely cause of his leukaemia'. Dr Nicholls submitted that:

> there is now a vast literature on the mutagenic and carcinogenic potential of ultraviolet light, ionising radiation and carcinogenic chemicals, and in all these cases the fundamental activity relates to damage to the DNA in individual cells …

39. In Dr Nicholls' submission, there is a clear dosage effect and consequent severe damage to the DNA of cells irradiated with ionising radiation. In his submission, chromosomal breaks resulted. Similarly, he contended that severe damage resulted from irradiation with microwaves.

> The process of transforming cells is to change them in such a way that they will continue to divide even in the absence of the normal *in vivo* stimulus which initiates cell division …
>
> No physical process (e.g. microwaves) should cause transformation of lymphocytes unless it is acting through the production of mutations unrelated to the normal production of antigen receptors. Such a genetic change will be permanent (unless the mutant cell dies) and a clone of cells will arise with the variant DNA. Such a change leading to independence from the normal antigenic stimulus which is normally self-limiting, may provide the basis of cancer initiation because the presence of a large clone of abnormal cells, running into millions, will provide the basis for further mutations if there is a repetition of the initial stimulus.

40. Dr Nicholls submitted it was well understood that microwaves are mutagenic, but not in the same way as x-ray or gamma rays impacting throughout the body, nor as ultraviolet light on the outer millimetre of the skin.

41. Dr Nicholls identified certain methodological difficulties in assessing the epidemiology of mutagenic exposure. These included delay in onset, chance, and the difficulties inherent in the study of a condition usually affecting people at an advanced stage, some or all of whom may die of something other than that disease. These issues apply to the study of chronic lymphoid leukaemia.

42. Nevertheless, Dr Nicholls submitted there was 'an index of suspicion' and referred to a number of studies which he considered on point. For example, one study reported a statistically significant increase in deaths from cancer of related lymphatic tissues among amateur radio operators (Milham, S 'Increased Mortality in Amateur Radio Operators due to Lymphatic and Haematopoietic Malignancies', *American Journal of Epidemiology* 127: 50-54 (1988)). In another:

> amongst 438,000 deaths in Washington State, 1950 - 1979, in 11 of 12 categories of workers occupationally exposed to electrical and magnetic fields, proportionate mortality ratios for death from leukaemia exceeded 100, in many cases more than double expectation (Milham, S 'Mortality from Leukaemia and Workers Exposed to Electrical and Magnetic Fields' *NEJM* 307:249 (1982)).

43. Dr Nicholls cited two other papers. In the first, a declining risk of leukaemia was observed by increasing the distance from a radio and television transmitter from 2 to 10 km. The second paper showed a decline in the rise of leukaemia with increasing distance from such transmitters, although the level of statistical significance was low, see Dolk et al 'Cancer Incidence near Radio and Television Transmitters in Great Britain I Sutton Coldfield Transmitter' *American Journal of Epidemiology* 145: 1-9 (1997) (SMRC folder 4, article 49), and Dolk et al 'Cancer Incidence near Radio and Television Transmitters in Great Britain II All High Power Transmitters' *American Journal of Epidemiology* ibid at pages 10 – 17 (SMRC folder 4, article 48).

44. Dr Nicholls submitted that asbestos, beryllium, carbon tetrachloride, methyl ethyl ketone and polychlorinated biphenyls all had carcinogenic potential, with some specificity of effect and that:

> any one of those substances could be an adjunct to another carcinogen helping to push mutant cells past a threshold towards malignancy.

45. Dr Nicholls supported the proposition that:

> the damage would not have all been done on one occasion. Small dosages capable of mutating the DNA of a small number (relatively) of cells could initiate appropriately mutated cells to produce clones running to a few million cells. The next step is a further irradiation of mutated cells and eventually, by a sequential process, leading to enough mutations in a particular cell or cells to initiate a malignant clone.

Repatriation Commission's submission

46. The submission for the Repatriation Commission was made by Dr Jon Kelley. The submission focused upon the role of radiation (particularly non-ionising electromagnetic radiation), and chemicals (particularly benzene), in the aetiology of chronic lymphoid leukaemia. Dr Kelley indicated that:

> electromagnetic radiation [EMR] covers a wide spectrum of radiation types, including radio waves, infrared radiation, visible light, ultraviolet radiation, and forms of ionising radiation. It is the radio frequency and sub-radio-frequency parts of the spectrum that have been of interest to investigators in exploring adverse health effects from EMR.

Radiation

47. The Repatriation Commission submitted that 'electromagnetic radiation is ubiquitous and exposure is unavoidable'. Concerns about adverse health impacts have:

> focused upon elevated exposures from man-made sources, particularly in occupational settings and from living in proximity to radiation sources.

48. Dr Kelley noted the limited material before the RMA. What was available was primarily concerned with extremely low-frequency radiation. There was little radio-frequency material, and in particular, no original occupational studies. In the Commission's submission, it was evident that the RMA did not have available to it many original papers addressing the respective relationships, if any, between extremely low-frequency radiation, radio-frequency radiation and chronic lymphoid leukaemia.

49. The Commission submitted that evidence concerning extremely low-frequency radiation from electricity should be evaluated separately from the evidence concerning radio-frequency radiation. The Commission considered it may be necessary to go further, and consider different frequency bands within the radio-frequency spectrum.

50. The Commission contended that there were methodological problems with much of the information before the RMA. It identified the primary difficulty as exposure assessment, describing this as 'the most critical element in studies of electric and magnetic fields'. The Commission considered some early studies assessing occupational exposure by job title was deficient, and that many studies did not take account of other exposures such as to electrical appliances within the home.

51. Further, the Commission criticised many studies for having:

(a) insufficient case numbers;

(b) no or only a limited control of potential confounders such as age, gender and race; and

(c) potential sources of bias.

52. The Commission identified three original studies on residential exposure to radio-frequency radiation available to the RMA. It noted no occupation studies were available. The Commission described it as 'evident that there is a wider body of literature on r[adio] f[requency] exposure that was not obtained by the [RMA].' The three studies considered exposure to television and radio transmitters. There was no information on exposure to radar or microwave frequency communications equipment such as that to which personnel would be exposed in the Defence context. Of those studies that were available, the Commission submitted that they were methodologically flawed.

53. The Commission concluded that there was a body of evidence before the RMA suggesting a weak association between extremely low-frequency radiation exposure and the occurrence of chronic lymphoid leukaemia. On that basis, and given it had identified a body of relevant material which was not before the RMA, the Commission submitted that it would be open to the Council to conclude that the evidence for high-level occupational exposure to extremely low-frequency magnetic fields did point to the requisite association for the purpose of the reasonable hypothesis (but not the balance of probabilities) test.

54. With respect to radio-frequency radiation, the Commission's submission was that the evidence before the RMA was insufficient to justify amendment to the relevant Statements of Principles, but that a new investigation should be undertaken by the RMA to consider the wider body of evidence on radio-frequency exposure and leukaemia that is available in the published literature.

55. In the Commission's submission, ionizing radiation had not been linked to chronic lymphoid leukaemia.

Benzene

56. The Commission submitted that exposure to benzene is 'universal'. It described benzene as:

> the simplest aromatic hydrocarbon … It is a constituent of crude oil, gasoline, and cigarette smoke … It is produced in large quantities in the refining of petroleum. It is widely used in industry in the production of eg plastics, rubber, lubricants, dyes, detergents, drugs, and pesticides. Another major use is as a petrol additive.

57. The Commission submitted that the best data available to the RMA on benzene exposed cohorts were from the pliofilm cohort, a study of a group of over 1800 workers between 1936 and 1975, as:

(a) there was no exposure to other potential carcinogens other than benzene;

(b) detailed records of specific jobs were kept; and

(c) industrial hygiene data were gathered

making the cohort suitable for quantitative risk assessment. The exposure was high, with those most highly exposed having hundreds of parts per million years of exposure. The Commission submitted that of 14 deaths identified in the cohort against 3.89 expected, none was attributable to chronic lymphoid leukaemia (see Wong O, 1995 (SMRC folder 3, article 17), Paxton et al, 1994 (SMRC folder 3, article 13), Crump KS, 1993 (SMRC folder 3, article 6), and Rinsky et al, 1987 (SMRC folder 3, article 14).

58. The Commission considered a study by Aksoy et al in 1974 'Leukemia in Shoe-Workers Exposed Chronically to Benzene' *Blood* vol 44, pages 837-841 was important, notwithstanding it was not before the RMA. In the Commission's assessment of the study, based on a review by Askoy M (1989) in 'Hematotoxcity and Carcinogenicity of Benzene' *Environmental Health Perspectives* vol 82, pages 193-197 (SMRC folder 3, article 16), a significant excess rate of leukaemia had been found in shoe, slipper and handbag workers chronically exposed to benzene. Incidents of leukaemia declined following discontinuation of the use of benzene in adhesives and thinners. However, in the Commission's submission, the leukaemia was acute and not chronic.

59. Whilst the Commission concluded that it was not possible to state with confidence that there was no association between benzene exposure and chronic lymphoid leukaemia, it submitted that the available evidence was not supportive of such an association. The Commission submitted its conclusion was supported by methodological limitations with the information available to the RMA at the relevant times, particularly with regard to exposure assessment, small case numbers, and lack of statistical power.

Other Chemicals

60. The Commission submitted that the material available to the RMA as to any putative association between chronic lymphoid leukaemia and exposure to chemicals other than benzene was limited. Of the one study that did, in the Commission's view, address the matter, no statistically significant association was found (see Linet MS, et al in 'Comparison of Methods for Determining Occupational Exposure in a Case-control Interview Study of Chronic Lymphatic Leukaemia' *Journal of Occupational Medicine* 1987: 29 (2): 136 – 141 (SMRC folder 3, article 1)).

61. The Commission concluded by submitting there was no sound medical-scientific evidence before the RMA linking chemical exposure other than benzene to an increased risk of chronic lymphoid leukaemia.

REASONS FOR THE COUNCIL'S DECISION

Pool of information and scope of the review

62. Having reviewed all the information available to (before) the RMA at the time it determined, amended or last amended the Statements of Principles, the Council was of the view that all the articles should be included in the pool of information. Accordingly, the Council had regard to all the information that was available to the RMA at the time it determined, amended or last amended the Statements of Principles.

63. After reviewing all the information, the Council decided to confine its attention to the factors for which the applicant and the person entitled to make a submission contended. So far as the reasonable hypothesis Statement of Principles was concerned (Statement of Principles No. 67 of 2001), the Council's task was therefore to ascertain whether the sound medical-scientific evidence available to the RMA at the relevant times pointed to whether exposure to:

(a) Radiation;

  (i) Extremely low frequency radiation;

  (ii) Radio frequency radiation;

  (iii) Ionizing radiation; and

  (iv) DC Batteries;

(b) Smoking tobacco cigarettes or pipe-smoking or passive smoking;

(c) Asbestos;

(d) Benzene;

(e) Petroleum Additives;

(f) Chromosomal Aberration;

(g) Viral and Bacterial Infection;

(h) Chemicals (particularly cleaning fluids and solvents)[4]; and

(i) Acetone

[4] being the petroleum additives and chemicals listed in paragraphs 28 and 29 above and carbon tetrachloride, methyl chloride, ethanol, methanol, methyl tertiary butal ether, beryllium, freon, isopropyl alcohol, polychlorinated biphenols, transformer and capacitor oils, trichlorethylene, Turcosolve and Comprox and paints.

(if found to exist in a particular case) could provide a link or element in a reasonable hypothesis connecting operational service to chronic lymphoid leukaemia[5].

64. Specifically, the Council's task was to determine whether the sound medical-scientific evidence in the pool of information pointed to, as opposed to (merely) leaving open, the relevant possibility[6] (ie whether exposure to any of the various factors contended for (as set out in paragraph 63 above) could provide a link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia to operational service). The Council must find that the hypothesis contended for was reasonable, and not one which was too tenuous or remote.

65. So far as the balance of probabilities Statement of Principles was concerned (Statement of Principles No. 68 of 2001) the Council needed to consider whether any or all of the contended factors (as set out in paragraph 63 above) if found to exist in a particular case, could provide a relevant connection between the kind of injury, disease or death (chronic lymphoid leukaemia) and eligible service according to a standard of satisfaction on the balance of probabilities, or as being more probable than not[7].

66. If the Council did not consider the contended exposure could provide a link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia to operational service, it did not go on to consider whether it could provide the relevant connection on the balance of probabilities. The reasonable hypothesis test is a test of possibility and an unusually light burden[8], and if that (very low) threshold was not reached for a particular contended factor, the higher and more onerous standard (the balance of probabilities) could not possibly be satisfied.

67. It was with these quite separate tests firmly at the forefront of its collective mind that the Council considered the pool of information and the submissions made by the applicant, the person entitled to make a submission, Dr Nicholls and the Repatriation Commission referable to each of the contended factors. The Council's analysis is set out below under each of the nominated factors.

The Council's Analysis of the Information

68. When considering the factors for which those making submissions had contended, the Council considered it of primary importance to recognise that chronic lymphoid leukaemia is a distinct disease, and likely to have an aetiology different from the other leukaemias. Leukaemias as a whole constitute less than 5% of all cancers[9]. To find that

[5] See *Vietnam Veterans' Association of Australia (NSW Branch) Inc v Specialist Medical Review Council and Anor* (2002) 69 ALD 553 (Moore J decision) per Moore J at paragraph 29.
[6] See the Full Federal Court decision at paragraph 49 per Branson J.
[7] Moore J decision at paragraph 29.
[8] op cit at paragraphs 49 and 55 per Branson J
[9] see Verkasalo (1996) 'Magnetic fields and leukaemia – risk for adults living close to power lines' *Scandinavian Journal of Work and Environmental Health* 22 supplement 2, pages 4-56 at page 12 (SMRC folder 5, article 26 & 27).

exposure to a substance is carcinogenic, or even leukemogenic, does not necessarily imply that there will be an association with chronic lymphoid leukaemia.

69. As mentioned above, the Council in its review was confined to the information which was actually before the RMA at the relevant times. However, the Council was conscious that there are some deficiencies in the research into the aetiology of chronic lymphoid leukaemia generally. For example, a genetic predisposition seems to be a very important element, yet studies into the incidence of the disease in different racial groups are scarce. Differences have also been identified for geographic (urban/rural), socio-economic and age variation (Verkasalo supra at page 13). Similarly, there is no research known to the Council which considers in any serious way any potential synergistic relationship between multiple factors and chronic lymphoid leukaemia.

70. The Council noted the applicant's submissions as to the different circumstances under which exposures may occur in a military context as opposed to a civilian context. However, the Council's task was to review the information available to the RMA at the relevant times. It was limited to its analysis of the studies before it, and it had no discretion to speculate as to whether the situation may have been different had different studies been done.

71. While the Council notes that the applicant, the person entitled to make a submission and Dr Nicholls referred to many articles which were not before the RMA (see Appendices E, F and G), it was impossible for the Council to form a view as to their relevance or merits as they were submitted as study titles and/or study abstracts only. However, the Council considers this additional material may advance the RMA's new investigation/s.

72. The Council when reviewing the pool of information noted several references which touched on the likelihood of an association between each of the contended factors (set out in paragraph 63) and chronic lymphoid leukaemia. The references thus identified contained material both in favour of, and against, the possibility of an association. Set out for each contended factor are relevant extracts identified by the Council in its analysis of the articles which touched on the possibility of an association.

73. The Council considered the information touching on each contended factor holistically, in determining whether the sound medical-scientific evidence pointed to, as opposed to merely leaving open, the possibility that a particular contended factor had the requisite association with operational (and if relevant, eligible) service. As mentioned above, the Council had to find more than a mere possibility. It had to be satisfied the hypothesis contended for was reasonable, and not too tenuous or remote.

Radiation

74. The Council recognised the fundamental importance of breaking up its consideration of exposure to radiation into a consideration of the different bands on the

spectrum. The Council noted the use of the following bands as submitted by the Commission in its written and oral submissions:

- Extremely Low Frequency Radiation (ELF): 30 Hz to 300 Hz
- Radio Frequency Radiation (RF): 3 KHz to 300 GHz
- Ionizing Radiation: 3×10^{16} Hz

75. The Council was of the view that this was not a completely accurate representation, and from its own expertise, considered use of the table attached as Table 1 to be more accurate. Table 1 includes the approximate wavelength, frequency, and energy limits of the various regions of the electromagnetic spectrum. Extremely low frequency radiation is included as part of radiowave frequency, and lasers are in the range of infra-red to optical wavelengths.

76. Radiation is not a single entity, and different frequencies may or may not cause different effects.

Extremely low frequency radiation

77. The Council noted there were articles among the information before the RMA at the relevant times which touched on[10] a putative association between exposure to extremely low frequency radiation and chronic lymphoid leukaemia. Such exposures include electrical power lines, wiring, appliances and equipment.

78. The sentinel study which the Council found convincing and methodologically very sound, was that of Floderus et al (1993) 'Occupational Exposure to Electromagnetic Fields in Relation to Leukaemia and Brain Tumours: a Case Control Study in Sweden' *Cancers Causes and Control* 1993 vol 4 pages 465 – 476 (SMRC folder 3 article 36).

79. The study involved 250 leukaemia patients, 261 brain tumour patients, and 1121 men in a randomly selected control group. The exposure assessment was based on measurements from different workplaces, on the basis of the job held longest during the ten-year period prior to diagnosis. The participants in the study were not limited to any particular industry, hence the yielded results were population rather than occupation based. Potentially confounding factors such as exposure to benzene, solvents, ionizing radiation, pesticides and smoking were taken into account.[11]

[10] see the Full Federal Court decision at paragraph 46 per Branson J

[11] The Council noted that the Floderus and Minder studies (Sweden and Switzerland) were based on a 'supply' of 16 Hz power as distinct from the rest of Europe and Australia (at 50 Hz) and the United States (at 60Hz). It can be seen from Table 1 that the energy carried by electromagnetic radiation increases as the frequency increases. Assuming the relevant association between the induction of chronic lymphoid leukaemia and electromagnetic frequency, then the Floderus and Minder studies may underestimate the risk in Australia, whilst the Savitz and Loomis study may overestimate such risks.

80. The study reports that the risk ratio for chronic lymphoid leukaemia increased significantly with increasing levels of exposure[12], particularly if the first exposure occurred after the age of 30. Duration of exposure did not have a decisive effect.

81. While the authors noted that their methodology was an improvement from earlier studies in which exposure assessment was estimated from aggregations of job titles, they acknowledged that their own exposure assessment remained an estimate. They acknowledged that residual confounding may exist.

> While it seems unlikely from the analyses performed that benzene, ionising radiation, pesticides, solvents, smoking habits, or residential area could have a decisive influence on the EMF findings, there may be other yet unknown confounding factors (at page 474).

82. They also sounded a cautionary note in their finding of an association:

> [c]ompared with childhood cancer studies, occupational EMF studies are very complex. Lifetime exposure is longer, and the level of exposure may have shifted many times, because of changes in occupation or employer, changes in tasks performed, technical developments for the same task in the same workplace, etc. Such circumstances reduce our possibilities to find a true association and probably have worked as a damper on the risk estimates from the present study. Furthermore, occupational exposure is just a fraction of the total EMF exposure. EMF outside the workplace was taken into consideration by rough questions on, for example, residential heating, proximity to a powerline, and the presence of electrical appliances in the home (at page 475)…

83. Other papers were identified by the Council as being suggestive of the relevant association between exposure to extremely low frequency radiation as a link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia with operational service. They were generally supportive of the Floderus paper, but were not as convincing due to methodological shortcomings. They contained different approaches to the analysis of information, and assessment of the relevant exposures. Their findings did not, as a general rule, achieve statistical significance.

84. Of those papers, the data discussed by Feychting, M, Forssen, U and Floderus, B (1997) in 'Occupational and Residential Magnetic Field Exposure and Leukaemia and Central Nervous System Tumours' *Epidemiology* vol 8 (4) pages 384-9 (SMRC folder 4, article 19) provides further support for the posited association between occupational

[12] Floderus et al at page 470, Table 3. Age-adjusted relative risk of leukaemia by EMF exposure: job held longest during the 10 years before diagnosis. Swedish occupational EMF study, 1983 – 87

Estimate of average daily:	Q1[a]		Q2		Q3		Q4		P90	
	Reference OR[b]	No.	OR[b] (CI)[c]	No.	OR[b] (CI)[c]	No.	OR[b] (CI)[c]	No.	OR[b](CI)[c]	No.
Mean	≤15 *T		0.16-0.19 *T		0.20-0.28 *T		≥0.29*T		≥0.14 *T	
CLL	1.0	13	1.1(0.5-2.3)	17	2.2 (1.1-4.3)	33	3.0 (1.6-5.8)	41	3.7 (1.8-7.7)	22

[a]Q1 + Q2 for median reference
[b]OR =odds ratio.
[c]CI - 95% confidence interval
*T = microTesla

magnetic field exposure as a link or element in a reasonable hypothesis linking chronic lymphocytic leukaemia with operational service[13]. However, the study dealt with a small number of subjects, and was subject to some methodological limitations. The most notable of these was potential confounding, particularly from smoking.

85. Prior to setting out the results of her own research, the paper by Verkasalo PK (1996) 'Magnetic Fields and Leukaemia - Risk for Adults Living Close to Powerlines' *Scandinavian Journal of Environment and Health* vol 22 supplement 2 pages 1 – 54 and 5 – 56 (SMRC folder 5, articles 26 & 27) analysed the studies to that date. She noted that:

> in summary, several studies **have suggested** that work in electrical occupations is associated with an increased risk of leukaemia; **some of the most recent studies with extensive magnetic field measurements have supported the earlier observations of risk increase, whereas others have not.** The greatest limitation of these occupational studies is perhaps that power-frequency magnetic field exposures vary substantially between persons, occupations, and industries. Other limitations include failure to consider potential confounders or biases (at page 18, emphases added).

86. Verkasalo's own research was weakly positive, identifying an increased incidence of chronic lymphoid leukaemia in persons living near powerlines. This was noted after a long-term exposure, but in only six cases, and the risk of misclassification and confounding was acknowledged. Whilst the author concluded that most risk estimates for chronic lymphoid leukaemia were somewhat elevated, she concluded that with the exception of exposures of > 10 years before diagnosis, and > 12 years of duration, risk increases were not statistically significant (see at page 39).

87. Minder CE and Pfluger DS in their 2001 paper 'Leukaemia, Brain Tumours, and Exposure to Extremely Low Electromagnetic Fields in Swiss Railway Employees' *American Journal of Epidemiology* vol 153(9) pages 825 – 835 (SMRC folder 4, article 1) found a dose response relation between leukaemia and occupational exposure to electromagnetic fields, among persons in highly exposed occupational groups, and in persons with high exposures (see page 831).

88. While 'there [was] no statistical disagreement between the findings of [this] study and Floderus' (at page 832), the Council noted that Minder et al's conclusions related

[13] Feychting et al, Table 2 at page 387. Occupational Exposure: Leukaemia and Central Nervous System (CNS) Tumor Relative Risk (RR) Estimates and 95% Confidence Intervals (CI), According to Measurements of Occupational Magnetic Field Exposure, Adjusted for Age and Sex.

Diagnosis	≤0.12 #T*	0.13-0.19 #T			≥0.20 #T		
	No.	No.	RR	95% CI	No.	RR	95% CI.
Leukemia	74	97	1.2	0.9-1.7	62	1.5	1.0-2.2
CLL	32	37	1.2	0.7-1.9	28	1.7	1.0-2.9

*Referent category for relevant risk estimates.
CLL - chronic lymphocytic leukemia
#T = microTesla

only to leukaemia generally, and not specifically to chronic lymphoid leukaemia. The findings were discounted by the Council for this reason. The Council also noted the authors' comments as to moderate exposure misclassification leading to insignificant results.

89. Miller et al in their 1996 paper, 'Leukaemia Following Occupational Exposure to 60 Hz Electric and Magnetic Fields among Ontario Electric Utility Workers' *American Journal of Epidemiology* vol 144(2) pages 150 – 160 (SMRC folder 5, article 21) noted an increase in the risk of leukaemia for increasing exposure to electric fields, save for medium level exposure to both electric and magnetic fields (see page 154). They acknowledged that 'the paucity of numbers of cases and controls did not permit a similar analysis for the leukaemia subtypes' (at page 155) (of which chronic lymphoid leukaemia is one).

90. The authors expressed the view that their findings for leukaemia were largely compatible with those in the published literature. However, they described Floderus' study as:

> **anomalous in showing elevated risk only for chronic lymphoid leukaemia**. Although odds ratios were elevated for chronic lymphoid leukaemia in the present study, the effect was mainly for electric fields and was less than for risks of acute non-lymphoid leukaemia and its component acute myeloid leukaemia (see page 158 emphasis added).

91. Miller et al (1997) in 'Brain Cancer and Leukaemia and Exposure to Power Frequency (50 to 60 Hz) Electric and Magenetic Fields' *Epidemiologic Reviews* vol 19(2) pages 273 – 293 (SMRC folder 5, article 23) frankly concede the limitations on, and disparities between, the various studies to date, noting the following:

- **the literature is far from unanimous** in concluding that electric and magnetic fields in general or the magnetic field in particular is related to cancer, whether as an initiator or as a promoter. Laboratory evidence of increased cancer rates or other deleterious effects stemming from electric and magnetic fields in animals or cell cultures is weak at best (page 273);

- almost all occupational studies included cases of white men only, although environmental studies included women and children and, in many cases, all races (page 274);

- almost all occupational studies determined a subject's exposure from his job title, usually from a death certificate or a cancer registry, but occasionally from an employer's records or interview (page 274);

- greater emphasis has been placed on those studies taking into account changing exposure over time, particularly before and after cancer diagnosis; residence mobility; and multiple field sources (appliance use as well as power line fields) (page 275).

92. The authors concluded that 18 of 21 studies:

> showed a significant or borderline-significant increase in risk in at least one subgroup, and three showed a non-significant increase in risk. Although consistently elevated risk ratios are suggestive, the increases in risk are small (many with a risk < 2.0), **and no consistency is apparent** in the job title most at risk or **in the specific leukaemia subtype(s) most commonly elevated**. Further, a dose-response trend is not evident when odds ratios from each study are ranked with the job title correlated to presumed exposure (at page 275 emphasis added).

93. Overall, the authors concluded that no single subtype of leukaemia 'dominated the[] results'. Whilst they again said they noted an inconsistency with the Floderus findings of an association only with chronic lymphoid leukaemia, they concluded that:

> there is **reasonable evidence for a weak association** (odds ratio between 1.0 and 2.0) between adult leukaemia and occupational exposure to electric and magnetic fields but that electric and magnetic fields cannot account for the majority of leukaemia cases among working men … Although most odds ratios are greater than one, very few are statistically significant (at page 280 emphasis added).
>
> **[O]ccupational studies with the best exposure assessment methods found … a significant elevated risk of chronic lymphocytic leukaemia (but not of all leukaemia) related to occupational exposure to electric and magnetic fields** (at page 282 emphases added).

94. In contrast, London S. et al in their 1994 paper 'Exposure to Magnetic Fields Among Electrical Workers in Relation to Leukaemia Risk in Los Angeles County' *American Journal of Industrial Medicine* vol 26 pages 47-60 (SMRC folder 4, article 17) did not observe a clear association between magnetic field exposure and chronic lymphocytic leukaemia (at page 53).

95. This was so, notwithstanding there was:

> a weak positive trend[14] of only borderline statistical significance … observed between occupation-specific magnetic field measurements and risk of **all** leukaemias (sic) (emphasis added) (at pages 55 and 56).

[14] London S et al at page 54, Table V (as modified). OR [(Odds Ratio)] for Leukaemia According to Estimates of Average Magnetic Field and Percent of Work Day Above 2.5 and 25 mG by Occupation.

Variable and category	Cases	Controls	OR (95% CI) categorical[a]	OR (95% CI) per 10 unit increase continuous
Average magnetic field (mG)				
All	2,355	67,212		1.2 (1.0-1.5)

[a]ORs (odds ratio) are age adjusted. CI, confidence interval.

The contrast with Floderus was explained because the authors had studied all employed workers, rather than focusing on workers in electrical occupations.

96. The authors identified that their study was potentially flawed by it having as their controls subjects with cancers (other than leukaemia) which cancers themselves may have been related to magnetic field exposure. In this case, the data would be biased towards the null. Further, smoking was a potential confounding factor. Their ultimate conclusion was that the weak positive trend for risk of all leukaemia combined may be due to chance.

97. The 1990 study by Loomis DP, and Savitz DA 'Mortality from Brain Cancer and Leukaemia Among Electrical Workers' *British Journal of Industrial Medicine* vol 47 pages 633-8 (SMRC folder 5, article 28) found a tendency toward an association between exposure to electromagnetic radiation with (only) acute leukaemia.

98. The same authors in their 1995 article, 'Magnetic Field Exposure in Relation to Leukaemia and Brain Cancer Mortality Among Electric Utility Workers', *American Journal of Epidemiology* vol 141(2) pages 123-34 (SMRC folder 5, article 20), concluded that:

> [f]irm conclusions regarding whether magnetic fields cause cancer, based on our study alone or on the entire literature, are not yet possible. Nonetheless, the methodological strengths of our study give our results sizeable influence on the overall pattern. The **general hypothesis that prolonged exposure to power frequency magnetic fields increases the risk of leukaemia is not supported by our study**. **The same hypothesis applied to chronic lymphocytic leukaemia is somewhat less clearly disputed**[15] (at page 133 emphasis added).

99. The Council was cognisant of the weaknesses inherent in the various studies, and the inconsistencies in results between and within the various studies. These are usefully summarised in the meta-analysis by Kheifets L I et al (1997) in 'Occupational Electric

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ibid at page 56 Table VII (as modified). ORs for Chronic Lymphocytic Leukaemia and Chronic Myeloid Leukaemia According to Estimates of Average Magnetic Field Exposure and Percent of Workday Above 2.5 and 25 mG by Occupation

Variable and category	Cases	OR (95% CI) categorical[a]	OR (95% CI) per 10 unit increase continuous
Chronic lymphocytic leukaemia			
Average magnetic field (mG)			
All	534		1.0 (0.6-1.5)

[a]ORs (odds ratio) are age adjusted. CI, confidence interval.

[15]Savitz and Loomis: Results for leukaemia subtypes … were imprecise but yielded a similar pattern of somewhat reduced risks for work as a lineman, no association for power plant workers, and increased risk for work as an electrician (acute myeloid leukaemia rate ratio (RR) = 2.0, 95 percent confidence interval (CI) 0.7-5.9; chronic lymphocytic leukaemia RR = 1.9, 95 percent CI 0.5-6.5). Work for 5 or more years in any exposed occupation also yielded an increased risk of chronic lymphocytic leukaemia (RR = 2.1, 95 percent CI 0.9-5.1) (at page 128).

and Magnetic Field Exposure and Leukemia' *Journal of Occupational & Environmental Medicine*, vol 39(11) pages 1074-91 (SMRC folder 5, article 8):

- whether observed risks are associated with exposures to EMF in the work environment, other non-EMF work-related exposures, or exposures outside the workplace;
- wide variation of exposure information;
- publication bias;
- allowance for confounders (when incorporating into studies, a higher average risk resulted);
- potentially large selection and information biases;
- use of non-representative populations;
- case control analyses of PMR data and potential recall bias; and
- misclassification.

100. Further, studies of mortality may not be suitable for looking at subgroups of leukaemia such as chronic lymphocytic leukaemia, and different assumptions about latency could also give different results (see Feychting, M (1996) 'Occupational Exposure to Electromagnetic Fields and Adult Leukaemia: A Review of the Epidemiological Evidence' *Radiation and Environmental Biophysics* 35 (4) pages 237-242 at pages 240 and 241) (SMRC folder 6, article 3).

101. The Council noted that laser was itself an exposure to electromagnetic radiation. In the Council's view, it could not be separated from its thermal effect. However, this relates to the laser light pointing at the exposed individual. The Council noted that the machinery that produced such laser light might also emit electromagnetic radiation at a low level and frequency that might be of relevance to the operator of such equipment. The Council was unaware of any studies measuring such incidental emission, or its potential for risk either generally or for chronic lymphoid leukaemia in particular.

102. Clearly the information before the RMA at the relevant times was contradictory as to whether exposure to extremely low frequency electromagnetic radiation could be a possible link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia and operational service.

103. Despite these limitations, the Council was of the view (bearing firmly in mind that it was applying an unusually light test in determining whether the sound medical-scientific evidence in the pool of information pointed to, as opposed to merely leaving open the relevant possibility) on the basis of the Floderus paper and those which supported it, that the evidence before the RMA at the relevant times did point to (as

opposed to merely leaving open) that possibility. This was so notwithstanding that none of the papers indicated the likely pathogenic frequency band of radiation, nor the likely duration of exposure. In the Council's view, the 'unusually light' threshold of a reasonable hypothesis had been met.

104. Given the Council's view that the information available to the RMA at the relevant times pointed to, as opposed to merely leaving open, the possibility that exposure to extremely low frequency radiation could provide a link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia with operational service, it was of the view that Statement of Principles No. 67 of 2001 should be amended to include such exposure as a factor.

105. Having decided that the information before the RMA at the relevant times discharged the unusually light burden of pointing to an association (and so satisfied the reasonable hypothesis test) the Council went on to consider whether the information satisfied the higher test of the balance of probabilities.

106. The Council was not satisfied that the information available to the RMA at the relevant times could provide a relevant connection between the kind of injury, disease or death (chronic lymphoid leukaemia) and eligible service according to a standard of satisfaction on the balance of probabilities, or as being more probable than not. That being said, the Council considered it quite possible that there may be other studies which could further advance the issue. The Council recommends the RMA undertake a new investigation to ascertain whether this is the case.

Radio frequency radiation

107. The Council noted there were only three articles[16] among the information before the RMA at the relevant times which touched on a putative association between exposure to radio frequency radiation and chronic lymphoid leukaemia. Such exposures include radio and television, broadcast frequencies, microwave**,** mobile phones, satellite communications, various medical devices and radar.

108. Of the three studies identified by the Council as pertaining to this issue the first, by Dolk et al (1997) 'Cancer Incidence Near Radio and Television Transmitters in Great Britain I Sutton Coldfield Transmitter' (SMRC folder 4, article 49) concluded that there was a reported excess of leukaemia near the Sutton Coldfield radio and television transmitter which declined in risk with distance from the site.

109. This finding was confirmed in the authors' subsequent 1997 article, 'Cancer Incidence Near Radio and Television Transmitters in Great Britain II All High Power Transmitters' (SMRC folder 4, article 48). That study found a significant decline in risk of leukaemia with distance from transmitters, although no clear interpretation of differences between leukaemia subtypes was possible (page 15).

110. The authors considered the apparent decline in risk statistically significant, although potentially due to chance. It was also considered possible that a decline in risk associated with distance may not imply any association with radio frequency transmission.

111. After analysing those articles, the Council's view was that they did not discharge the unusually light burden of pointing to the relevant association. Whilst the information before the RMA at the relevant times was vaguely suggestive of a putative association, it was too tenuous and remote, with very limited research available. Of that which was available, the respective authors themselves indicated that their findings were very weak. Further, as mentioned above, there was no clear distinction between different leukaemia subtypes.

112. Again, however, the Council considered it quite possible that there may be other studies which could further advance the issue (potentially including those relied upon by the applicant, person entitled to make a submission and Dr Nicholls, as listed in Appendices E - H).

113. So far as Statement of Principles No. 67 of 2001 is concerned, the Council directs that in the course of the new investigation to be undertaken by the RMA, the RMA

[16] Dolk et al 'Cancer Incidence Near Radio and Television Transmitters in Great Britain I: Sutton Coldfield Transmitter' *American Journal of Epidemiology* 1997 vol 145 no. 1 pages 1 – 9; Dolk et al, 'Cancer Incidence Near Radio and Television Transmitters in Great Britain II All High Power Transmitters' *American Journal of Epidemiology supra* pages 11 – 17; Cooper et al 2001 Re: 'Cancer Incidence Near Radio and Television Transmitters in Great Britain No I Sutton Coldfield Transmitter; II All high Power Transmitters *American Journal of Epidemiology* vol 153(20) pages 202 – 204.

ascertain whether there is sound medical-scientific evidence that exposure to radio frequency radiation, if found to exist in a particular case, could provide a link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia with operational service.

114. So far as Statement of Principles No. 68 of 2001 is concerned, the Council recommends that the RMA undertake a new investigation to ascertain whether there is sound medical-scientific evidence that exposure to radio frequency radiation, if found to exist in a particular case, could provide a relevant connection between chronic lymphoid leukaemia and eligible war service (other than operational service) or defence service (other than hazardous service) according to a standard of satisfaction on the balance of probabilities, or as being more probable than not.

Ionizing radiation

115. The Council identified only one article which was before the RMA at the relevant times which touched upon a putative association between ionizing radiation and chronic lymphoid leukaemia, being Inskip PD et al (1990) 'Leukemia Following Radiotherapy for Uterine Bleeding' *Radiation Research* vol 122 pp 107-119 (SMRC folder 3, article 31). The Council did not consider that article transformed the theoretical possibility of a putative link to a reasonable hypothesis.

116. The study found that the observed number of deaths for chronic lymphoid leukaemia and lymphoid leukaemia was nine (mortality rates did not enable separation of the two leukaemias) (see page 111). The authors considered that the unanticipated excess of deaths from chronic lymphoid leukaemia and lymphoid leukaemia, together with the small numbers of leukaemia deaths in the various subgroups examined, gave reason for caution in the interpretation of their results (page 114). They concluded that:

(a) their findings for deaths from chronic lymphoid leukaemia and lymphoid leukaemia may have occurred by chance;

(b) chronic lymphoid leukaemia and lymphoid leukaemia may have been over-diagnosed, and some deaths mistakenly attributed;

(c) since chronic lymphoid leukaemia has a low case fatality rate relative to other leukaemias, and is predominantly a disease of old age, differential classification error may have occurred; and

(d) they could not determine the extent to which confounding associated with presenting gynecologic disorders or noncomparability in the ascertainment of outcome may have biased the results. The slight excess of deaths raised concern about possible upward bias in the risk estimates (see at page 117).

117. Since the authors of the one relevant study themselves expressed significant reservations as to the reliability of their findings, and given the small number of adverse events, the Council was not able to say that it pointed to, as opposed to merely leaving open, the possibility of an association. Accordingly, the Council did not consider the

information before the RMA at the relevant times provided any basis for amendment to either Statement of Principles No. 67 of 2001, or Statement of Principles No. 68 of 2001.

Direct Current Radiation - DC Batteries

118. It was contended that there was a putative link between exposure to direct current batteries and chronic lymphoid leukaemia. The Council notes that direct current batteries do not produce an electromagnetic sinusoidal waveform. Rather, they create a field of energy from a fixed magnetic field which is constant, and not fluctuating. There was nothing in the information before the RMA which in any way supported a putative link, and in the Council's view, there is no merit in this submission.

Smoking Tobacco Cigarettes or Pipe-smoking or Passive Smoking

119. From the information before the RMA, the Council identified a number of papers touching on this potential factor. Brown et al (1992) 'Smoking and Risk of Leukaemia' *American Journal of Epidemiology* vol 135 (7) pages 763 – 768 (SMRC folder 6, article 5) was a case control study of 578 white men with leukaemia, and 820 controls. It found significantly elevated risks for all leukaemias, including chronic lymphoid leukaemia, the increased incidence for the latter being 1.1 – 2.3. Notwithstanding the increased risk, only weak trends with duration of smoking, and no trends with the amount smoked were discerned.

120. Despite the apparent clarity of the findings, the authors speculated on the general reasons for inconsistencies (chance, race, sex, patterns of tobacco use and demographic variables) between (tobacco and chronic lymphoid leukaemia) studies and the mechanism by which tobacco may influence leukaemia risk. The Council noted that all smokers, including passive smokers, experience a raised benzene intake. The authors' other suggested mechanisms include radioactive components, and other leukemogens in tobacco and tobacco smoke, and that tobacco may act through the production of chromosomal defects.

121. The Council identified that there were a number of negative studies among the information before the RMA at the relevant times. The first of these, Friedman GD (1993) 'Cigarette Smoking, Leukemia, and Multiple Myeloma *Annals of Epidemiology* vol 3 pages 425-428 (SMRC folder 4, article 34) asserts that cigarette smoking is not associated with chronic lymphocytic leukaemia.

122. Another negative paper was Adami J, et al (1998) 'Smoking and the Risk of Leukemia, Lymphoma, and Multiple Myeloma' (Sweden) *Cancer Causes & Control* vol 9 (1) pages 49-56 (SMRC folder 4, article 43). This article was less adamant in its conclusions, suggesting that the role of tobacco in the aetiology of leukaemia and other neoplasms remained unclear. It was acknowledged that tobacco smoke contains radioactive compounds and benzene, which are known to be leukemogenic. One further suggested possibility was that tobacco may have an effect on the immune system which predisposes to the development of lymphoid neoplasms generally.

123. The report found a non-significant positive relationship between smoking and the risk of developing myeloid or acute leukaemia. Chronic (and acute) lymphoid leukaemias were not found to be related to smoking.

124. Notwithstanding its (negative) findings, the authors considered there was some ambiguity with respect to a putative link between chronic lymphoid leukaemia and smoking. Some suggested reasons for the disparities with the Brown article were:

- the use of death as an outcome measure as opposed to incidence;
- some of the diseases are rare, which means that the power to detect small risks differences may be weak;
- potential ascertainment bias - since smoking causes a number of adverse health effects, smokers may have closer medical supervision than non-smokers, leading to both over-diagnosis and earlier (sub-clinical) diagnosis;
- lack of data about other lifestyle factors which could confound findings; and
- restriction of the study to men (see at pages 53-4).

125. The authors argue the strengths of their study contrast with their review of many other studies, and conclude that their study:

> with elaborate collection of smoking data, a long follow-up, and almost complete ascertainment of incident cancers, provides no evidence that smoking is related to any major extent to the leukaemias (at page 54).

126. The Council closely analysed the articles, and noted the overall paucity of relevant articles in the information which was before the RMA at the relevant times. The Council considered that the information available to it (and the RMA) was a limited sample of the very significant body of academic writing pertaining to the issue.

127. Given the findings in the Brown study, the Council considered it seemed theoretically possible that there may be a low-risk relationship between environmental exposure to tobacco smoke and chronic lymphoid leukaemia. However, the Council was not in a position to say that the Brown article of itself pointed to, as opposed to merely leaving open the relevant association. In this regard, the Council considered persuasive the analysis in the Adami study, and particularly its discussion of the disparities with the Brown study.

128. The Council could not say on the basis of the information available to the RMA at the relevant times that it pointed to the relevant association as opposed to merely leaving it open as a theoretical (not reasonable) hypothesis. However, the Council was aware from its own expertise that there was research into passive smoking from the National Health and Medical Research Council which did not appear to have been before the RMA at the relevant times.

129. So far as Statement of Principles No. 67 of 2001 is concerned, the Council directs that in the course of the new investigation to be undertaken by the RMA, the RMA ascertain whether there is sound medical-scientific evidence that exposure to smoking tobacco cigarettes or pipe-smoking or passive smoking, if found to exist in a particular case, could provide a link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia with operational service.[17]

130. So far as Statement of Principles No. 68 of 2001 is concerned, the Council recommends that the RMA undertake a new investigation to ascertain whether there is sound medical-scientific evidence that exposure to smoking tobacco cigarettes or pipe-smoking or passive smoking, if found to exist in a particular case, could provide a relevant connection between chronic lymphoid leukaemia and eligible war service (other than operational service) or defence service (other than hazardous service) according to a standard of satisfaction on the balance of probabilities, or as being more probable than not[18].

Asbestos

131. The Council identified three primary papers among the information before the RMA which touched on this putative association as follows:

(i) Schwartz et al (1988) 'B Cell Neoplasms and Occupational Asbestos Exposure' *American Journal of Occupational Medicine* vol 14 pages 661 – 671 (SMRC folder 4, articles 9 & 42);
(ii) Kagan E and Jacobson R J (1983) 'Lymphoid and Plasma Cell Malignancies: Asbestos Related Disorders of Long Latency' *American Society of Clinical Pathologists* vol 80 (1) pages 14 -20 (SMRC folder 4, article 44); and
(iii) Linet et al (1987) 'Comparison of Methods for Determining Occupational Exposure in a Case-control Interview Study of Chronic Lymphocytic Leukaemia' *Journal of Occupational Medicine* vol 29 (2) pages 136- 141 (SMRC folder 3, article 1).

132. The Kagan article concerned a very small sample of 13 asbestos workers, 6 with chronic lymphoid leukaemia. Notwithstanding some conceded limitations with the study (particularly that smoking could be a confounding factor) the authors considered their findings were such **as to suggest** the need for further investigation as to whether asbestos exposure can be linked to chronic lymphoid leukaemia.

133. The Schwartz article showed a modest increased risk for chronic lymphoid leukaemia, however overall, the Council did not consider it a positive study. The study involved predominately white males, and the modest increased incidence shown was not statistically significant.

[17] See section 196B(2) of the VEA
[18] See section 196B(3) of the VEA

134. Further, notwithstanding the apparent findings of the Schwartz study, the authors themselves conceded that there could in fact have been no true association, and their findings could have been the result of chance (at page 668). A further limitation was the reliance upon the subjects' occupational history as the sole measure of exposure, which the authors acknowledged could lead to misclassification.

135. The authors concluded that:

> although an increasing risk of C[hronic] L[ymphoid] L[eukaemia] [(CLL)]was noted with increasing levels of asbestos exposure, the magnitude of the excess risk was small and **may be due to other concomitant occupational exposures, undetected confounding factors, or simply chance** … [however] our findings are at least **suggestive** that an association may exist between asbestos exposure and developing CLL [hence] additional studies in different populations seem warranted (at page 669 emphases added).

136. The Council's analysis of the Linet article is discussed at paragraphs 172 - 173 below. The authors' conclusion in summary was that there was no statistically significant positive association between chronic lymphoid leukaemia and exposure to chemicals, including asbestos.

137. On the basis of the information available to the RMA at the relevant times, the Council was not able to say that it pointed to, as opposed to merely leaving open, the possibility of an association. As acknowledged by the authors, the studies at best appeared to be suggestive of (only) the mere possibility of an association, which in the Council's view remained remote and untenable. Accordingly, the Council did not consider the information before the RMA at the relevant times provided any basis for amendment to either Statement of Principles No. 67 of 2001 or Statement of Principles No. 68 of 2001.

Benzene

138. The Council was cognisant of the very significant number of studies among the information before the RMA which considered benzene as a potential carcinogen. However, those studies overwhelmingly considered leukaemia other than chronic lymphoid leukaemia, and as mentioned above, chronic lymphoid leukaemia is likely to have a distinct aetiology.

139. Nevertheless, the Council identified some papers which touched on the specific issue. Wong O and Raabe G, in their 1989 meta-analysis, 'Critical Review of Cancer Epidemiology in Petroleum Industry Employees, with a Quantitative Meta-Analysis by Cancer Site' *American Journal of Industrial Medicine* vol 15 pages 283 – 310 (SMRC folder 3, article 20) reviewed epidemiologic reports of petroleum industry employees in a number of countries. Their findings indicated that the industry experienced significantly lower cancer mortality than the general population for all cancer sites combined.

140. While they concluded that the evidence was **suggestive** of a small increased risk of leukaemia with increased length of service, they identified a number of

methodological difficulties which undermined these findings. These may be summarised as follows:

- high-risk groups cannot be identified, and only a few studies provided job or area specific analyses;
- most of the leukaemia cases in the petroleum industry were not found among those who worked on benzene units directly;
- the issue of whether benzene or other petroleum hydrocarbons could affect only acute myelogenous leukaemia or other cell types remains unresolved;
- diagnostic specificity (or lack of) for lymphatic diseases on death certificates, particularly on older death certificates, may result in inaccuracy of underlying cause of death classification;
- this may confound site-specific analyses;
- latency was of the order of 20 - 30 years, different from the short latency reported for benzene related leukaemia in the early literature;
- it is not clear whether benzene was the only responsible agent or if other chemicals were also involved (see at page 304).

141. The findings of their own studies are set out in Wong and Raabe (1995) 'Cell-Type Specific Leukaemia Analysis in a Combined Cohort of more than 208,000 Petroleum Workers in the United States and the United Kingdom' 1937 - 1989 *Regulatory Toxicology and Pharmacology* vol 21 pages 307 – 321 (SMRC folder 3, article 7).

142. They stressed the importance of cell-type specific analysis when studying leukaemia. In particular, they noted that chronic lymphoid leukaemia does not stem from a common progenitor, but from separate stem cells. The authors subjected other studies to close scrutiny, and noted the disparate results. In some cases, there was a deficit of chronic lymphoid leukaemia relative to the normal population; in other cases, the findings were explicable by chance, and in other cases, there was an excess risk of chronic lymphoid leukaemia. Overall, the authors concluded that there was no increased risk for any cell-type-specific leukaemias among petroleum workers.

143. Infante PF (1993) in 'State of the Science on the Carcinogenicity of Gasoline with Particular Reference to Cohort Mortality Study Results' *Environmental Health Perspectives Supplements* vol 101 (supplement 6) pages 105 – 109 (SMRC folder 3, article 5) considered there was an elevated risk of leukaemia (not necessarily chronic lymphoid leukaemia) observed among workers having a high or medium exposure to benzene. Infante came to this conclusion having reviewed 9 petro-chemical industry studies, two of which were before the RMA.

144. The 1989 study by Malone K E et al 'Chronic Lymphocytic Leukaemia in Relation to Chemical Exposures' *American Journal of Epidemiology* vol 130 pages 1152 – 1158 (SMRC folder 4, article 53) focused primarily upon antigenic stimulation of the immune system as a possible risk factor for chronic lymphoid leukaemia. However, it also considered other possible risk factors including (other) chemical exposure. The study took into account age, sex, race, level of education and area of residence as potentially confounding factors.

145. The authors identified the potential for the following weaknesses:

(a) recall bias;

(b) misclassification;

(c) scarcity of information on the level of exposure; and

(d) size constraints of the studies.

146. Only a very weak (if any) association was noted between chronic lymphoid leukaemia and benzene, and aromatic hydrocarbons as a whole. The authors considered this unexpected 'since chemicals in this category have long been suspected of being related to the occurrence of leukaemias, including chronic lymphocytic leukaemia' (at page 1157).

147. Askoy M (1989) in 'Haemotoxicity and Carcinogenicity of Benzene' *Environmental Health Perspectives* vol 82 pages 193 – 197 (SMRC folder 3, article 16) drawing on the data and results from an earlier 1974 study (see paragraph 148 below) noted a lack of consistency in the distribution of leukaemia types caused by benzene exposure. In the cases of chronic lymphoid leukaemia, the benzene content of adhesive and thinners was very low, with a high percentage of toluene, and intermittent exposure (see at page 196).

148. The Council noted the original Turkish Shoemakers study by Aksoy et al (1974) 'Leukaemia in Shoe-workers Exposed Chronically to Benzene' *Blood* vol 44 pp 837-841 was not before the RMA, although from the references to it in the Askoy review referred to in paragraph 147 above, it seemed to be relevant. That the only way in which this material was before the RMA at the relevant times was in the form of a review was considered by the Council to be unsatisfactory.

149. Another important group of studies considering the putative association between benzene and chronic lymphoid leukaemia is those considering a cohort of workers engaged in the manufacture of pliofilm[19]. This cohort had a relatively high exposure to

[19] Crump (1994) 'Risk of Benzene-induced Leukaemia: a Sensitivity Analysis of the Pliofilm Cohort with Additional Follow-up and New Exposure Estimates' *Journal of Toxicology and Environmental Health* 42:219-242 (SMRC Folder 3, Article 6); Paxton et al (1994) 'Leukemia Risk Associated with Benzene Exposure in the Pliofilm Cohort: I. Mortality Update and Exposure Distribution' *Risk Analysis* volume 14 number 2, pages 147 - 154 (SMRC Folder 3 Article 13); Rinsky et al (1987) 'Benzene and Leukemia: An Epidemiologic Risk Assessment' *New England Journal of Medicine* vol. 316 no. 17 1044 – 1050 (SMRC

benzene alone, without the potential confounders of exposure to other chemicals. These studies did not record any deaths from chronic lymphoid leukaemia, although there was an elevated risk of other leukaemia cell types.

150. Given the negative findings of the pliofilm cohort studies (so far as chronic lymphoid leukaemia is concerned), serious methodological difficulties in the various studies, and the negative or weak evidence for a suggested connection, the Council considered that the information before the RMA at the relevant times did not point to, as opposed to merely leave open, the possibility of an association. However, it considered that there may well be subsequent or other studies which touched on the issue which were not available to the RMA at the relevant times.

151. In this regard, the Council particularly noted the additional references cited by the applicant (see Appendix E). Since those references were not before the RMA, the Council was precluded from considering them. In any event, only abstracts were provided, and whilst the Council was not in a position to form any view as to the merits of those materials, they did provide sufficient evidence that additional studies exist.

152. So far as Statement of Principles No. 67 of 2001 is concerned, the Council directs that in the course of the new investigation to be undertaken by the RMA, the RMA ascertain whether there is sound medical-scientific evidence that exposure to benzene, if found to exist in a particular case, could provide a link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia with operational service.

153. So far as Statement of Principles No. 68 of 2001 is concerned, the Council recommends that the RMA undertake a new investigation to ascertain whether there is sound medical-scientific evidence that exposure to benzene, if found to exist in a particular case, could provide a relevant connection between chronic lymphoid leukaemia and eligible war service (other than operational service) or defence service (other than hazardous service) according to a standard of satisfaction on the balance of probabilities, or as being more probable than not.

Petroleum Additives

154. The Council considered the article by Malone et al 'Chronic Lymphocytic Leukaemia in Relation to Chemical Exposures' supra touched on a putative link between exposure to petroleum additives and chronic lymphoid leukaemia. The authors, after referring on the one hand to studies which have reported elevated risk for leukaemia associated with exposure to petroleum or other aliphatic hydrocarbons, and on the other to studies which have found no such association, concluded that more study was needed.

155. In light of this, and the results of the Malone study generally (of very weak associations if any between nominated chemicals and chronic lymphoid leukaemia) (see

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Folder 3, Article 14); Wong, O (1995) 'Risk of Acute Myeloid Leukaemia and Multiple Myeloma in Workers Exposed to Benzene' *Occupation and Environmental Medicine* vol 52 380 - 384 (SMRC Folder 3, Article 17).

paragraphs 144 – 146 above), the Council concluded that the information before the RMA at the relevant times did not point to, as opposed to merely leave open, the possibility of an association. Accordingly, the Council did not consider the information before the RMA at the relevant times provided any basis for amendment to either Statement of Principles No. 67 of 2001, or Statement of Principles No. 68 of 2001.

Chromosomal Aberration

156. The Council noted the submissions which were made by the person entitled to make a submission and Dr Nicholls as to chromosomal aberration. In the Council's view, this was an interesting theory.

157. As noted in paragraph 120 above, Brown in his article,[20] whilst noting that the mechanism by which tobacco products might influence leukaemia risk is unknown, speculated that radioactive compounds and other leukemogens present in tobacco and tobacco smoke may act through the production of chromosomal defects which have been observed in the peripheral blood cells of smokers (see at page 767). However, Brown concluded:

> cigarette smoking has been associated with specific cytogenetic abnormalities in patients with acute nonlymphocytic leukaemia … and with immunologic perturbations, including elevated white blood counts, alterations in T-cell subsets, and lower percentages of natural killer cells … These findings are noteworthy since leukaemia occurs excessively among patients with certain conditions associated with chromosomal instability or immunodeficiency … **Thus, it is possible that exposure-response relations with smoking may be elusive not only because of the low-level of risk but also because of possible indirect mechanisms (e.g. immune defects)** (at page 767, emphasis added).

158. The Brown article, being the only article before the RMA at the relevant times which touched on this putative association, pointed to significant confounders, and described any putative association between chromosomal aberration and chronic lymphoid leukaemia as 'elusive'.

159. The Council considered it was a theory which was, on the basis of the information available to the RMA at the relevant times, too remote to satisfy the unusually light burden of a reasonable hypothesis. Accordingly, the Council considered there was no basis upon which to amend either Statement of Principles No. 67 of 2001 or Statement of Principles No. 68 of 2001.

Viral and Bacterial Infection

160. The Statements of Principles presently include as a factor exposure to the HTLV virus. The Council could identify nothing in the information before the RMA which suggested any basis for amending either Statement of Principles No. 67 of 2001 or Statement of Principles No. 68 of 2001 so far as this factor is concerned.

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[20] 'Smoking and Risk of Leukaemia' *American Journal of Epidemiology* vol 135(7) pages 763 - 768 (SMRC folder 6, article 5)

Chemicals (Particularly Cleaning Fluids and Solvents)[21]

161. The Council was unable to examine the full spectrum of chemicals, and focused upon those for which the applicant and person entitled to make a submission contended. The Council agreed that many chemicals may be carcinogenic. However, there was scant evidence in the information before the RMA that exposure to them could be a link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia with operational service. Again, the Council reminded itself that chronic lymphoid leukaemia is a distinct form of leukaemia and so likely to have a distinct aetiology.

162. The Council nevertheless identified some relevant articles within the RMA information which touched on the issue. Amadori D, et al (1995) 'Chronic Lymphocytic Leukemias and non-Hodgkin's Lymphomas by Histological Type in Farming-Animal Breeding Workers: A Population Case-control Study Based on Job Titles' *Occupational Environmental Medicine,* vol 52 pp 374-379 (SMRC folder 4, article 31) ascertained that a higher risk for chronic lymphoid leukaemia applied to farmer-breeders. The results were statistically significant.

163. The suggested reason for that finding was the use of chemicals in agriculture, or exposure to animal transmitted diseases or specific chemicals used in animal breeding. There was consistency between chronic lymphoid leukaemia cases and controls for socio-demographic and lifestyle characteristics such as marital status, education, migration from the south (of Italy), alcohol intake and cigarette smoking (see at pages 375 - 6).

164. Blair A, et al in (1992) 'Clues to Cancer Etiology from Studies of Farmers' *Scandinavian Journal of Work and Environmental Health* vol 18 pp 209-215 (SMRC folder 3, article 34) considered the studies concerning risks to farmers. They noted they were unable to adjust for confounding and bias, as they did not have access to the raw data. Some of the trends of elevated risk among farmers were ascribed to improvements in diagnosis and reporting. They also suggested any association may be due to considerable exposure to ultraviolet light from the sun and insecticides.

165. Further work was done in this area by Nanni et al (1996) in 'Chronic Lymphocytic Leukaemias and non-Hodgkin's Lymphomas by Histological Type in Farming-Animal Breeding Workers: A Population Case-control Study Based on a priori Exposure Matrices' *Occupational Environmental Medicine* vol 53 at pages 652 – 7 (SMRC folder 4, article 32). The association of chronic lymphoid leukaemia and those working in farming animal breeding (extending on the 1995 study reported by Amadori) was, in the views of the authors, partially explained by exposure to pesticides, particularly insecticides and stannates.

166. Shortcomings of the study primarily centred around the potential for errors of recall - subjects could not report the specific pesticides used, the amounts, nor the periods of use.

[21]The Council's focus when considering this potential factor was upon the cleaning fluids and solvents mentioned in paragraphs 28 and 29, and footnote 4 above.

167. The studies are focused upon an occupational group, hence it was not apparent what the actual exposures comprised. This in the Council's view significantly weakened any conclusions. In the absence of specific exposure measurement, the Council considered it impossible to form a view other than that any putative association was too tenuous and remote to comprise a reasonable hypothesis. It was not sufficiently specific as to the relevant (multiple) exposures.

168. Bethwaite et al (1990) in 'Cancer Risk in Painters: Study Based on the New Zealand Cancer Registry' *British Journal of Industrial Medicine* vol 47 pages 742 - 6 (SMRC folder 3, article 23) noted that painters are potentially exposed to a wide group of substances, including organic solvents and dyes. These include toluene and xylenes, trichlorethylene, n-hexane and methyl ethyl ketone. The authors concluded that there was little evidence of an increased risk for cancer other than multiple myeloma.

169. Spirtas et al (1991) in 'Retrospective Cohort Mortality Study of Workers at an Aircraft Maintenance Facility. I Epidemiological Results' *British Journal of Industrial Medicine* vol 48, pages 515-530 (SMRC folder 3, article 3) conducted a study of persons involved in the repair and maintenance of aircraft. Their exposures included chlorinated hydrocarbons (trichlorethylene, perchlethylene, chloroform, trichlorethane, methylene chloride, ortho-dichloro-benzene, and freon), aliphactics (isopropyl alcohol and other alcohols) and other compounds such as carbon tetrachloride, solder flux, zinc chromate, silica, and high octane fuels.

170. While the study was large, the authors conceded it was subject to several limitations. First was the potential for multiple exposures, with considerable overlap of chemical uses. It was accordingly difficult to relate excess risk to specific chemical exposures. Some solvents were used interchangeably, some were contaminated with other chemicals, and others had metabolised into other chemicals. No allowance was made for smoking as a compounding factor. Other lifestyle factors were similarly not accommodated.

171. The authors concluded that no significant persuasive association was found (see at page 528). In respect of those associations which were found, the authors counselled caution, as the results could have been due to chance.

172. Linet et al in their 1987 study, 'Comparison of Methods for Determining Occupational Exposure in a Case-control Interview Study of Chronic Lymphocytic Leukaemia' *Journal of Occupational Medicine* volume 29 (2) pages 136- 141 (SMRC folder 3, article 1) found:

> there were no statistically significant positive associations between c[hronic] l[ymphoid] l[eukaemia] [(CLL)] and employment in farming, the rubber industry, the dry cleaning business, construction, manufacturing, utilities and communications, other personal services or medical services industries, all of which had been reported to be associated with an increased risk of CLL (at page 137).

173. The authors posit the following as suggested reasons for the contrast of their findings with previous studies:

- variation in the precision in defining occupational exposures;
- errors in reporting occupation or exposure;
- incorrect assumption that occupations have a single and defined exposure; and
- that many of the occupations and industries the subject of previous research do not apply in the location of this study.

174. The Council was of the view that the information available to the RMA at the relevant times as to any putative association between exposure to chemicals (being those listed in paragraphs 29 and 29 and footnote 4) as a link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia to operational service was conflicting and tenuous. The authors of the various studies frankly conceded suggested associations were mere possibilities, due to chance or adversely affected by methodological difficulties.

175. The Council did not consider the information pointed to, as opposed to merely leaving open, the possibility of the requisite connection. Accordingly, the Council did not consider, on the basis of the information before the RMA at the relevant times, that any amendment to Statement of Principles No. 67 of 2001 or Statement of Principles No. 68 of 2001 should be made.

Acetone

176. Again, the Council noted that there may be a carcinogenic effect from exposure to acetone. However, there was nothing in the information before the RMA at the relevant times which suggested that such exposure could provide a link or element in a reasonable hypothesis connecting chronic lymphoid leukaemia to operational service.

177. The Council noted the dearth of information before the RMA at the relevant times. One potentially relevant study was that of Garland FC et al (1990) 'Incidence of Leukemia in Occupations with Potential Electromagnetic Field Exposure in United States Navy Personnel' *American Journal of Epidemiology* vol 132, pp 293-303 (SMRC folder 5, article 24)**,** which considered 102 cases of leukaemia in United States Navy personnel ('with possible exposure to organic solvents, strong electrical and magnetic fields, and varying exposures to sun light' (at page 295)).

178. In the Council's view, the results showed a marginally increased incidence of leukaemia. Again, however, those increases were of acute leukaemia, both myeloid and lymphoid, and unspecified myeloid leukaemia. There was no indication of an increased rate of chronic lymphoid leukaemia. The Council also noted the criticisms of the study made by the person entitled to make a submission. The Council did not consider this study pointed to the relevant association. Accordingly, the Council was of the view that no amendment to either Statement of Principles No. 67 of 2001 or Statement of Principles No. 68 of 2001 was justified.

THE COUNCIL'S CONCLUSIONS

179. Attached for ease of reference in tabular form are the Council's conclusions in respect of each contended factor (see Table 2).

DECISION

180. The Council made the directions and recommendations in paragraphs 1 – 4 above.

EVIDENCE BEFORE THE COUNCIL

Documents

181. The information considered by the Council (being the information that was available to the RMA and sent to the Council by the RMA in accordance with section 196K of the VEA) was as is listed in Appendix A.

182. The information upon which the applicant, the person entitled to make a submission and Dr Nicholls relied (being information which was available to the RMA and sent to the Council by the RMA in accordance with section 196K of the VEA) was as is listed in Appendices B, C & D respectively.

183. The information upon which the applicant, the person entitled to make a submission and Dr Nicholls further relied (being information which was not available to the RMA) is listed in Appendices E, F & G respectively.

184. The information to which the Repatriation Commission referred (being information which was not available to the RMA) is listed in Appendix H.

TABLE 1

(REFER PARAGRAPH 75)

Regions of the Electromagnetic

	Wavelength (m)	Frequency (Hz)	Energy (J)
Radio	$> 1 \times 10^{-1}$	$< 3 \times 10^{9}$	$< 2 \times 10^{-24}$
Microwave	1×10^{-3} - 1×10^{-1}	3×10^{9} - 3×10^{11}	2×10^{-24}- 2×10^{-22}
Infrared	7×10^{-7} - 1×10^{-3}	3×10^{11} - 4×10^{14}	2×10^{-22} - 3×10^{-19}
Optical	4×10^{-7} - 7×10^{-7}	4×10^{14} - 7.5×10^{14}	3×10^{-19} - 5×10^{-19}
UV	1×10^{-8} - 4×10^{-7}	7.5×10^{14} - 3×10^{16}	5×10^{-19} - 2×10^{-17}
X-ray	1×10^{-11} - 1×10^{-8}	3×10^{16} - 3×10^{19}	2×10^{-17} - 2×10^{-14}
Gamma-ray	$< 1 \times 10^{-11}$	$> 3 \times 10^{19}$	$> 2 \times 10^{-14}$

URL: http://imagine.gsfc.nasa.gov/docs/science/know_l1/spectrum_chart.html

TABLE 2

(REFER PARAGRAPH 179)

CONTENDED FACTORS	STATEMENT OF PRINCIPLES No. 67 of 2001	STATEMENT OF PRINCIPLES No. 68 of 2001
Radiation:		
Extremely Low Frequency (ELF)	Directs an amendment	Recommends a new investigation
Radio Frequency (RF)	Directs a new investigation	Recommends a new investigation
Ionizing (IR)	No basis for an amendment	No basis for an amendment
DC Batteries	No basis for an amendment	No basis for an amendment
Smoking Tobacco Cigarettes or Pipe-smoking or Passive smoking	Directs a new investigation	Recommends a new investigation
Asbestos	No basis for an amendment	No basis for an amendment
Benzene	Directs a new investigation	Recommends a new investigation
Petroleum Additives	No basis for an amendment	No basis for an amendment
Chromosomal Aberration	No basis for an amendment	No basis for an amendment
Viral and Bacterial Infection	No basis for an amendment	No basis for an amendment
Chemicals (Particularly Cleaning Fluids and Solvents)	No basis for an amendment	No basis for an amendment
Acetone	No basis for an amendment	No basis for an amendment

Appendix A

SMRC Folder No.	Title No	Title
1 and 6	1 and 7	McGrath MS & Ng VL. (1992). 'Human retroviruses and cancer'. In P Calabresi & PS Schein. *Medical Oncology* (2nd Ed.), McGraw Hill, New York, pp 79-82.
3	1	Linet MS, Stewart WF, Van Natta, McCaffrey LD and Szklo M (1987). 'Comparison of methods for determining occupational exposure in a case-control interview study of chronic lymphocytic leukemia'. *J of Occupational Med*, Vol 29(2), pp 136-141.
3	2	Ward JH. (1992). 'Hematologic effects of occupational hazards'. In Rom WN (Ed), *Environmental and Occupational Medicine* (2nd Ed.) Little, Brown & Co., Boston, pp 619-631.
3	3	Spirtas R, Stewart PA, Lee JS, et al. (1991). 'Retrospective cohort mortality study of workers at an aircraft maintenance facility. I Epidemiological results'. *British Journal of Industrial Medicine*, Vol. 48, pp 515-530.
3	4	Landrigan PJ & Nicholson WJ. (1992). 'Benzene'. In WN Rom (Ed), *Environmental and Occupational Medicine*. Little Brown & Co., Boston, pp 861-865.
3	5	Infante PF. (1993). 'State of the science on the carcinogenicity of gasoline with particular reference to cohort mortality study results'. *Environmental Health Perspectives* Suppl, Vol 101(Suppl. 6), pp 105-109.
3	6	Crump KS (1993). 'Risk of benzene-induced leukemia: A sensitivity analysis of the pliofilm cohort with additional follow- up and new exposure estimates'. *Journal of Toxicology and Environmental Health*, Vol 42, pp 219-242.
3	7	Wong O and Raabe GK. (1995). 'Cell-type-specific leukemia analyses in a combined cohort of more than 208,000 petroleum workers in the United States and the United Kingdom, 1937-1989'. *Regulatory Toxicology and Pharmacology*, Vol 21, pp 307-321.
3	8	Harrington JM, Rose FG, & Koh D. (1994). 'Paint-Health and Environmental Risk Management'. *Asia-Pacific Journal of Public Health*, Vol 7(2), pp 115-118.

3 9 McGregor DB, Heseltine E & Moller H. (1995). 'Dry cleaning, some solvents used in dry cleaning and other industrial chemicals'. IARC meeting, Lyon, 7-14 February 1996. *Scandinavian Journal of Work, Environ & Health*, Vol 21(4), pp 310-2.

3 10 Brandt L. (1992). 'Exposure to organic solvents and risk of haematological malignancies'. *Leukemia Research*, Vol 16(1), pp 67-70.

3 11 Brett SM, Rodricks JV and Chinchilli VM. (1989). 'Review and update of leukemia risk potentially associated with occupational exposure to benzene'. *Environmental Health Perspectives*, Vol 82, pp 267-281.

3 12 Snyder R and Kalf GF. (1994). 'A perspective on benzene leukemogenesis'. *Critical Reviews in Toxicology*, Vol 24(3), pp 177-209.

3 13 Paxton MB, Chinchilli VM, Brett SM and Rodricks JV. (1994). 'Leukemia risk associated with benzene exposure in the pliofilm cohort: 1. Mortality update and exposure distribution'. *Risk Analysis*, Vol 14(2), pp 147-154.

3 14 Rinsky RA, Smith AB, Hornung R et.al. (1987). 'Benzene and Leukemia. An epidemiologic risk assessment'. *N Engl J of Med*, Vol 316(17), pp 1044-1050.

3 15 Wong O, Harris F and Smith TJ. (1993). 'Health effects of gasoline exposure. II. Mortality patterns of distribution workers in the United States'. *Environmental Health Perspectives Supplements*, Vol 101(Suppl. 6), pp 63-76.

3 16 Aksoy M. (1989). 'Hematotoxicity and carcinogenicity of benzene (1989)'. *Environmental Health Perspectives*, Vol 82, pp 193-197.

3 17 Wong O. (1995). 'Risk of acute myeloid leukaemia and multiple myeloma in workers exposed to benzene'. *Occup Environ Med*, Vol 52, pp 380-384.

3 18 Hansen ES. (1993). 'A follow-up study on the mortality of truck drivers'. *Am Journal of Industrial Medicine*, Vol 23, pp 811-821.

3 19 Schnatter AR, Katz AM, Nicolich MJ & Theriault G. (1993). 'A Retrospective Mortality Study among Canadian Petroleum Marketing and Distribution Workers'. *Environmental Health Perspectives Supplements,* Vol 101(Suppl 6), pp 85-99.

3 20 Wong O and Raabe GK. (1989). 'Critical review of cancer epidemiology in petroleum industry employees, with a quantitative meta-analysis by cancer site.' *Am Journal of Industrial Med*, Vol 15, pp 283-310.

3 21 Doll R and Peto R. (1976). 'Mortality in relation to smoking: 20 years' observations on male British doctors'. *British Medical Journal*, Vol 2, pp 1525-1536.

3 22 Christie D, Robinson K, Gordon I and Bisby J. (1991). 'A prospective study in the Australian petroleum industry. II. Incidence of cancer'. *British Journal of Industrial Med*, Vol 48, pp 511-514.

3 23 Bethwaite PB, Pearce N and Fraser J. (1990). 'Cancer risk in painters: study based on the New Zealand Cancer Registry'. *British J of Industrial Medicine*, Vol 47, pp 742-6.

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3 25 Hjelle B, Mills R, Swenson S, Mertz G, Key C and Allen S. (1991). 'Incidence of hairy cell leukemia, mycosis fungoides, and chronic lymphocytic leukemia in first known HTLV-II-Endemic population'. *JID*, Vol 163, pp 435-440.

3 and 4 26 29 Hoar Zahm S, Weisenburger DD, Babbitt PA, Saal RC, Vaught JB and Blair A. (1992). 'Use of hair coloring products and the risk of lymphoma, multiple myeloma, and chronic lymphocytic leukemia'. *Am J Pub Health*, Vol 82(7), pp 990-998.

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3 31 Inskip PD, Monson RR, Wagoner JK, Stovall M, Davis FG, Kleinerman RA and Boice JD. (1990). 'Leukemia following radiotherapy for uterine bleeding'. *Radiation Research*, Vol 122, pp 107-119.

3 32 Viel JF and Richardson ST. (1993). 'Lymphoma, multiple myeloma and leukaemia among French farmers in relation to pesticide exposure'. *Soc Sci Med*, Vol 37(6), pp 771-777.

3 33 Wigle D T, Semenciw R M, Wilkins K, et al. (1990). 'Mortality study of Canadian male farm operators: non-Hodgkin's lymphoma and agricultural practices in Saskatchewan'. *J Natl Cancer Inst*, Vol 82(7), pp 575-582.

3 34 Blair A, Hoar Zahm S, Pearce N E, Heineman EF and Fraumeni JF. (1992). 'Clues to cancer etiology from studies of farmers'. *Scand J Work Environ Health*, Vol 18, pp 209-215.

3 35 Zheng W, Linet M S, Shu X, Pan R, Gao Y and Fraumeni J F. (1993). 'Prior medical conditions and the risk of adult leukemia in Shanghai, People's Republic of China'. *Cancer Causes and Control,* Vol 4, pp 361-368.

3 36 Floderus B, Persson T, Stenlund C, Wennberg A, Ost A and Knave B. (1993). 'Occupational exposure to electromagnetic fields in relation to leukemia and brain tumors: a case-control study in Sweden'. *Cancer Causes and Control*, Vol 4, pp 465-476.

3 37 Scheinberg DA and Golde DW. *Harrison's Principles of Internal Medicine* - 13th Edition - McGraw Hill (1994). 'The Leukemias'. Chapter 310, pp 1764-1774.

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Appendix B

SMRC Folder No.	Title No	Title
8	34	SMRC 404: Savitz DA et al. (2000). 'Case-cohort analysis of brain cancer and leukaemia in electric utility workers using a refined magnetic field job-exposure matrix'. *Am J of Ind Med,* Vol 38, Issue 4, pages 417-425.
8	35	SMRC 405: Villeneuve PJ et al. (2000). 'Leukaemia in electric utility workers: The evaluation of alternative indices of exposure to 60 Hz electric and magnetic fields'. *Am J Ind Med*, Vol 37, Issue 6, pages 607-617.
8	67	SMRC 227: Raabe GK, Wong O. (1996). 'Leukemia mortality by cell type in petroleum workers with potential exposure to benzene'. *Environ Health Perspect*, Dec. 104 Suppl 6, pp 1381-92.
8	71	SMRC 223: Huebner WW. (1997). 'Mortality experience of a young petrochemical industry cohort: 1992 follow-up study of US-based employees'. *J Occup Environ Med*, Oct. 39(10), pp 970-82.
8	85	SMRC 209: Huebner WW et al. (2000). 'Incidence of lymphohaematopoietic malignancies in a petroleum industry cohort: 1983-94 follow-up'. *Occup Environ Med*, Sep. 57(9), pp 605-14.

Appendix C

SMRC Folder No.	Title No	Title
2	20	Reference List used by Dr E.M. Nicholls for the 24/07/2000 Written submission to the RMA and SMRC.
2	21	Dr M Harvey. 02/08/2000. Report in support of [person entitled to make a submission to the SMRC].
2	22	[person entitled to make a submission to the SMRC] letter of 30/07/2000 to VVFed on RAN exposures to Electromagnetic Radiation (EMR) and Toxic Chemicals.
2	23	CDR GH McNally. 06/1983 Memo. Radiation Hazards on Upper Decks.
2	24	Request and response of 16/03/2000 on EMR by RF Radiation Branch, Air Force Research Laboratory, Brooks Air Force Base Texas, USA.
2	25	Request and response of 13/04/2000 by Dr Roy, ARPANZA.
2	27	Dr Marino. 1992 Review. Electromagnetic fields, cancer, and the theory of neuroendocrine-related promotion.
2	28	Dr Marino and Morris D. Undated paper. 'Chronic electromagnetic stressors in the environment: A risk factor in human cancer'.
2	29	The EMR Alliance: Press Release. June 13, 1995. American Physical Society.
2	30	Responses by Dr Marino and Allan Frey to APS Press Release at 29 above.
2	31	Microwave News July/Aug 1996. Cover Story by Louis Slessin.
2	32	Microwave News Jan/Feb 1998. Polish RF/MW Cancer data shows Dose Response Trend by Louis Slessin.
2	33	General Abstracts of Important Scientific Findings by de Pomeroi et al.
2	34	General Abstracts of Important Scientific Findings by Roti Roti et al.

2	35	B Blake Levitt. 1999. 'Electromagnetic Fields - A Consumer's Guide to the issues and how to protect ourselves'.
2	36	OSHA, US Dept. of Labor. Electromagnetic / Microwave Radiation.
2	37	Four (4) Statutory Declarations from ex RAN technicians.
2	38	Addendum – Employment statement by [person entitled to make a submission to the SMRC].
5	24	Garland FC, Shaw E, Gorham ED, Garland CF, White MR & Sinsheimer PJ. (1990). 'Incidence of leukemia in occupations with potential electromagnetic field exposure in United States Navy personnel'. *American Journal of Epidemiology*, Vol 132, pp. 293-303.
10	18	Dr EM Nicholls. 24/07/2000. Report on the likely cause(s) of the development of Chronic Lymphatic Leukaemia in [person entitled to make a submission to the SMRC].

Appendix D

SMRC Folder No.	Title No	Title
2	20	Reference List used by Dr E.M. Nicholls for the 24/07/2000 Written submission to the RMA and SMRC.
4	48	Dolk et al. (1997). 'Cancer incidence near radio and television transmitters in Great Britain. II. All high power transmitters'. *Am J Epidemiol*, Vol 145(1), pp 10-17.
4	49	Dolk et al. (1997). 'Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield transmitter'. *Am J Epidemiol*, Vol 145(1), pp 1-9.
10	18	Dr EM Nicholls. 24/07/2000. Report on the likely cause(s) of the development of Chronic Lymphatic Leukaemia in [person entitled to make a submission to the SMRC].

Appendix E

SMRC Folder No.	Title No	Title
Not submitted		The Board of Inquiry into the F-III (fuel tank) Deseal/Reseal and Spray Seal Programs (1977-1999) report of 29 June 2001.
Not submitted		1995 Federal Government - Exposure Standards For Atmospheric Contaminants in the Occupational Environment. [NOHSC: 3008 (1995)].
7	7	CLL23: Goldsmith JR. (1997). 'Epidemiologic evidence relevant to radar (microwave) effects'. *Environ Health Perspect*, Dec. Vol 105 (Suppl 6), pp 1579-87.
7	8	CLL22: Jauchem JR. (1998). 'Health effects of microwave exposures: a review of the recent (1995-1998) literature'. *J Microw Power Electromagn Energy*, Vol 33(4), pp 263-74.
7	9	CLL21: Velizarov S et al. (1999). 'The effects of radiofrequency fields on cell proliferation are non-thermal'. *Bioelectrochem Bionerg*, Feb. Vol 48(1), pp 177-80.
7	10	CLL19: D'Andrea JA. (1999). 'Behavioral evaluation of microwave irradiation'. *Bioectrectromagnetics*, Suppl 4, pp 64-74.
7	11	CLL18: Garaj-Vrhovac V. (1999). 'Micronucleus assay lymphaocycte mitotic activity in risk assessment of occupational exposure to microwave radiation'. *Chemosphere*, Dec. Vol 39(13), pp 2301-12.
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7	13	CLL16: Repko JD, Lasley SM. (1979). 'Behavioural, neurological, and toxic effects of methyl chloride: a review of the literature'. *CRC Crit Rev Toxicol*, Nov. Vol 6(4), pp 283-303.
7	14	CLL15: Maeda H, Akaike T. (1998). 'Nitric oxide and oxygen radicals in infection, inflammation, and cancer'. *Biochemistry* (Mosc), Vol 63(7), pp 854-65.
7	15	CLL014: Karakaya A et al. (1999). 'Proliferation response of peripheral blood lymphocytes and lymphocyte subpopulations in n-hexane, toluene, and methyl ethyl ketone co-exposed workers'. *Environmental Toxicology and Pharmacology*, Vol 8(1), pp 53-58.

7	16	CLL09: Porru S et al. (1997). 'The toxicology and prevention of the risks of occupational exposure to aromatic polycyclic hydrocarbons. III. The effects: epidemiological evidence, early effects. Individual hypersusceptibility. Health surveillance'. *G Ital Med Lav Ergon*, Oct-Dec. Vol 19(4), pp 152-63
7	16	CLL10: Hathway DE. (2000). 'Toxic action/toxicity'. *Biol Rev Camb Philos Soc*, Feb. Vol 75(1), pp 95-127.
7 and 52	16	CLL11: Golding BT, Watson WP. (1999). 'Possible mechanisms of carcinogenesis after exposure to benzene'. *IARC Sci Publ*, (150), pp 75-88.
7	16	CLL12: Twersok LE. (1999). 'Development of toxicity criteria for petroleum hydrocarbon fractions in the Petroleum Hydrocarbon Criteria Working Group approach for risk-based management of total petroleum hydrocarbons in soil'. *Drug Chem Toxicol*, Feb. Vol 22(1), pp 275-91.
7	17	CLL08: Air Care International, INC. (1999). 'Jet Fuel Health & The Environment, p 1'.
7	18	CLL07: Apostoli P. (1998). 'Update on the subject of lead toxicology'. *Ann 1st Super Sanita*, Vol 34(1), pp 5-15.
7	19	CLL06: Sanin LH et al. (1998). 'Accumulation of lead in bone and its effects on health'. *Salud Publica Mex*, Jul-Aug. Vol 40(4), pp 359-68.
7 and 8	20 65	CLL 05: A et al. (1996). 'Metabolism of Benez[alpha]anthracene by human bone marrow in vitro'. *Chem Biol* Interact, Vol 99, pp 29-40.
7 and 7	21 74	CLL04: Reese E, Kimbrough RD. (1993). 'Acute toxicity of gasoline and some additives'. *Environ Health Perspect*, Dec. Vol 101, Suppl 6, pp 115-31.
7 and 8	22 65	CLL03: Voelker R. (1999). 'Fuel Additive Metabolities Probed'. *JAMA*, Vol 282, No 18, November 10.
7	23	CLL02: White RD. (1999). 'Refining and blending of aviation turbine fuels'. *Drug Chem Toxicol*, Vol 22, pp 143-53.
7	25	*The Merck Manual of Diagnosis and Therapy Evaluation of Drug Toxicity*, Section 22, Chapter 302, pages 1-4.
7 and	26	MCT 01: Feron VJ et al. (1998). 'Toxicology of chemical mixtures: International perspective'. *Environmental Health Perspectives*, Vol

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7	48 and 58	HC 21: ‘Copy’ titled Diethylene Glycol Monomethyl Ether. A list of 6 Research Excerpts.
7	49 and 59	HC 22: ‘Copy’ titled Toluene. pp 135 & 136
7	50 and 60	HC 23: ‘Copy’ titled Toluene. A list of 37 Research Excerpts.
7	51 and 61	HC 24: ‘Copy’ titled Benzene. pp 20 & 21.
7	53	HC 29: ‘Copy’ titled Xylene. p 143.
7	54	HC 27: ‘Copy’ titled Naphthalene. A list of 34 Research Excerpts.
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Appendix F

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9	5	**SMRC 4**: Extract from Medical Oncology: Nazario LA et al. 'A Comprehensive Review – Epidemiology of Cancer and Prevention Strategies'. Dpt of Clinical Cancer Prevention, The University of Texas.

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10	5	Unknown Author and data. - Liverpool Hospital statistics.
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10 19 EMF Issues: Field strength, safety standards and sources of man made RF fields.

Appendix G

SMRC Folder No.	Title No	Title
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Not submitted		Goldsmith JR. (1995). 'Epidemiologic evidence of radiofrequency (microwave) effects on health in military, broadcasting and occupational studies'. *Intl J Occup Environ Health*, Vol 1, pp 47-57.
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Appendix H

SMRC Folder No.	Title No	Title
Not submitted		Askoy M et al. (1974). 'Leukaemia in shoe-workers exposed chronically to benzene'. *Blood*, Vol 44, pp 837-841.