Specialist Medical Review Council

Reasons for Decisions

Section 196W
Veterans' Entitlements Act 1986

Re: Statements of Principles Nos. 9 and 10 of 2005
as not amended by the RMA on 14 April 2008
In Respect of Chronic Lymphoid Leukaemia
Matter Nos. 2011/1 & 2
Requests for Review Declaration No. 22

SUMMATION

1. In relation to the Repatriation Medical Authority (the RMA) Statement of Principles No. 9 of 2005, as not amended on 14 April 2008 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) under subsection 196W of the VEA:

> DECLARES that there is sound medical-scientific evidence on which the RMA could have relied to amend the Statement of Principles to include small lymphocytic lymphoma as the same disease as chronic lymphocytic leukaemia;

and

> DECLARES that there is sound medical-scientific evidence on which the RMA could have relied upon to amend the Statement of Principles to include the factors applying to chronic lymphocytic leukaemia as set out below;
>
> Non-Ionising Radiation;

Benzene;

Herbicides, Pesticides and Dioxin (separately or in combination);

Asbestos;

and

DECLARES that the sound medical-scientific evidence available to the RMA is insufficient to justify any amendment to the Statement of Principles to include as factors exposure to tobacco smoking or to any other factor;

and

2. In relation to the RMA Statement of Principles No. 10 of 2005 in respect of chronic lymphoid leukaemia and death from chronic lymphoid leukaemia, made under subsection 196B(3) of the VEA the Council under section 196W of the VEA:

DECLARES that there is sound medical-scientific evidence on which the RMA could have relied to amend the Statement of Principles to include small lymphocytic lymphoma as the same disease as chronic lymphocytic leukaemia;

and

DECLARES that the sound medical-scientific evidence available to the RMA is insufficient to justify an amendment of the Statement of Principles to include as a factor applying to chronic lymphocytic leukaemia exposure to:

Non-ionising Radiation;

Chemicals - Benzene, Toluene, Xylene, Carbon Tetrachloride;

Aircraft fuel (AVGas) and associated chemicals (MEK, sealants) and exposure due to F111 fuel tank repairs;

Herbicides, Pesticides and Dioxin;

Asbestos; and

Tobacco (by which the Council understands the Applicant to mean tobacco smoking); or

any other factor;

and

3. In relation to the RMA Statement of Principles No. 9 of 2005 in respect of chronic lymphoid leukaemia and death from chronic lymphoid leukaemia, made under subsection 196B(2) of the VEA:

> DIRECTS the RMA to amend the Statement of Principles concerning chronic lymphoid leukaemia No 9 of 2005 by including factors applying to chronic lymphocytic leukaemia for;
>
> Non-Ionising Radiation,
>
> Benzene,
>
> Herbicides, Pesticides and Dioxin (separately or in combination),
>
> Asbestos,
>
> at an exposure level, duration of exposure and period of time from exposure to onset of disease to be ascertained by the RMA;

and

4. In relation to the RMA Statement of Principles Nos. 9 and 10of 2005 in respect of chronic lymphoid leukaemia and death from chronic lymphoid leukaemia, made under subsection 196B(2) and (3) of the VEA the Council under section 196W of the VEA:

> REMITS the matter for RECONSIDERATION by the RMA in accordance with the following DIRECTIONS and RECOMMENDATIONS:
>
> A DIRECTION to the RMA to conduct an investigation taking account of the following recommendations:
>
> a. to determine new Statements of Principles concerning chronic lymphocytic leukaemia and small lymphocytic lymphoma as the same (that is, a single) disease,
>
> b. to excise small lymphocytic lymphoma from the non-Hodgkin's lymphoma Statements of Principles in force;
>
> AND for that purpose, include in its investigation;
>
> i. information available to the RMA when it last determined, amended, or last amended, or decided, or last decided, not to determine, or not to amend a Statement of Principles concerning chronic lymphoid leukaemia and

chronic lymphocytic leukaemia in particular, and

ii. information available to the RMA when it last determined, amended, or last amended, or decided, or last decided, not to determine, or not to amend a Statement of Principles concerning non-Hodgkin's lymphoma so far as that information relates to chronic lymphocytic leukaemia or small lymphocytic lymphoma, and

iii. information available to the RMA when it last determined, amended, or last amended, or decided, or last decided, not to determine, or not to amend any Statements of Principles concerning haematopoietic cancers so far as that information relates to chronic lymphocytic leukaemia or small lymphocytic lymphoma, and

iv. any new information concerning chronic lymphocytic leukaemia or small lymphocytic lymphoma and particularly any new information concerning the factors as directed in paragraph 3 above as well as;

Other Aromatic Hydrocarbons; and

AV Gas and associated chemicals in relation to aircraft fuel tank de-seal / reseal activities,

to determine new Statements of Principles for chronic lymphocytic leukaemia and small lymphocytic lymphoma as the same (that is, a single) disease.

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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 function of the Council as set out in the VEA. The Minister must appoint one of the Councillors to be 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.

6. When a review is undertaken the Council is constituted by 3 to 5 Councillors selected by the Convener. If the Council does not include the Convener, the Convener must appoint one of the Councillors selected for the review to preside at all meetings as Presiding Councillor.

7. Clinical Professor David Joske was the Presiding Councillor for this review. Professor Joske is a haematologist and pathologist, former head of department of Haematology at Sir Charles Gairdner Hospital, Perth and Clinical Professor of Medicine at University of West Australia, and Chairman of SolarisCare Foundation;

8. The other members of the Council were:

    (i) Professor Andrew Wirth: Senior Fellow, Department of Pathology, University of Melbourne and Consultant Radiation Oncologist, Peter MacCallum Cancer Institute, Melbourne; and

    (ii) Professor Stephen Mulligan: Senior Staff Specialist, Department of Haematology, Royal North Shore Hospital and Adjunct Professor, School of Molecular and Microbial Biosciences, University of Sydney; and

    (iii) Professor Andrew Grulich: Professor and Head, HIV Epidemiology and Prevention Program, University of NSW, Sydney; and

    (iv) Dr Constantine Tam: Senior Fellow, University of Melbourne and Consultant Haematologist, Peter MacCallum Cancer Centre.

THE LEGISLATION

9. The legislative scheme for the making of Statements of Principles is set out in Parts XIA and XIB of the VEA. Statements of Principles operate as templates which are ultimately applied by decision-makers in determining individual claims for benefits under the VEA and the Military Rehabilitation and Compensation Act 2004 (the MRCA)[1].

10. Fundamental to Statements of Principles is the concept of 'sound medical-scientific evidence', which is 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[2].

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:

> – Statement of Principles No. 9 of 2005, 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)[3] of the VEA ('the reasonable hypothesis test'); and

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1 See sections 120, 120A and 120B of the VEA and sections 335, 338 and 339 of the MRCA.

2 This has been held to mean 'information which epidemiologists would consider appropriate to take into account' see *Repatriation Commission v Vietnam Veterans' Association of Australia NSW Branch Inc* (2000) 48 NSWLR 548 (the New South Wales Court of Appeal decision) per Spigelman CJ at paragraph 117.

3 196B(2) provides;

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 service rendered by members of Peacekeeping Forces; or

(c) hazardous service rendered by members of the Forces; or

(caa) British nuclear test defence service rendered by members of the Forces; or

(ca) warlike or non-warlike service rendered by members;

– Statement of Principles No. 10 of 2005, 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)[4] of the VEA ('the balance of probabilities test').

12. Specifically, it was contended that there was medical-scientific evidence upon which the RMA could have relied to include:

– Non-ionising radiation;

– Chemicals - including all aromatic hydrocarbons – Benzene, Toluene, Xylene, Carbon Tetrachloride and others; and

– Asbestos; and

– Tobacco (understood by the Council to mean tobacco smoking); and

– Exposure to herbicides, pesticides and or dioxins,

as factors in Statements of Principles 9 and 10 of 2005.

13. In conducting its review, the Council must review all the information that was available to (before) the RMA at the time it determined, amended, or last amended the Statement of Principles and is constrained to conduct its review by reference to that information only[5].

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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.

[4] 196B(3) provides;

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 and British nuclear test defence service) rendered by members of the Forces; or

(ba) peacetime service rendered by members;

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.

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

14. 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

15. On 24 February 2005, the RMA under subsections 196B(2) and (3) of the VEA determined the Statements of Principles being instruments respectively numbered 9 and 10 of 2005 in respect of chronic lymphoid leukaemia.

16. On 3 March 2005 in accordance with section 42 of the Legislative Instruments Act 2003 the Statements of Principles were registered; and on 7 March 2012, they were tabled in the House of Representatives and in the Senate.

The first request for review

17. An application dated 13 April 2005 for review of Statements of Principles numbers 9 and 10 of 2005 was received by the Council on 19 April 2005. Specifically the application was concerned with the decision of the RMA of 24 February 2005 not to include:

    – Radiation - ionising or other; including electromagnetic radiation (EMR), radio, microwave VHF & UHF;

    – Chemicals - including all aromatic hydrocarbons – Benzene, Toluene, Xylene, Carbon Tetrachloride and others;

    – Asbestos;

    – Tobacco (by which the Council understood the Applicant to mean tobacco smoking); and

    – Herbicides and pesticides.

    as factors in those Statements of Principles.

18. A second application dated 27 May 2005 for review of Statements of Principles numbers 9 and 10 of 2005 was also received by the Council. Specifically the application was concerned with the decision of the RMA of 24 February 2005 not to include exposure to dioxins as a factor in those Statements of Principles.

19. Pursuant to section 196ZB of the VEA the Council published in the Gazette a notice of its intention to carry out a review of all the information available to the RMA about Chronic Lymphoid Leukaemia, and inviting persons or organisations authorised so to

do to make submissions to the Council[6]. The Council gazetted one subsequent notice as to the date by which written submissions must be received by the Council[7].

The second request for review

20. On 18 April 2008 the RMA advised the Council that it had completed an investigation[8], limited to dioxin exposure and dioxin contaminated herbicide exposure, with respect to Statements of Principles numbered 9 and 10 of 2005 concerning chronic lymphoid leukaemia. A copy of the RMA's Declaration of 14 April 2008 was enclosed.

21. On 14 April 2008 in accordance with subsection 196b(9) of the VEA the RMA determined not to make an amendment of the Statements of Principles numbered 9 and 10 of 2005 already determined in respect of chronic lymphoid leukaemia and provided its declaration and reasons for decision:

    – in relation to dioxin exposure and dioxin contaminated herbicide exposure; and

    – [that the] new evidence does not identify a statistically significant increase in the relative risk of chronic lymphoid leukaemia in individuals with documented dioxin exposure and does not report a dose-response relationship.

22. On 7 May 2008 the making of the RMA Declaration was notified in the Gazette (No. 18, p. 1007).

23. On 26 May 2008 the Council wrote to the Applicant (Mr AR) who contended for the inclusion of exposure to dioxins as a factor in Statements of Principles 9 and 10 of 2005 advising him:

    a. of the completed investigation by the RMA in respect of chronic lymphoid leukaemia;

    b. of the RMA declaration of 14 April 2008 not to amend Statements of Principles numbers 9 and 10 of 2005 in relation to dioxin exposure and dioxin contaminated herbicide exposure;

    c. that the 14 April 2008 declaration by the RMA refers to 26 new pieces of information that had come to light since Statements of Principles numbers 9 and 10 of 2005 were determined by the RMA on 24 February 2005; and

    d. that if the SMRC receives a fresh application from him, he would be provided with the new information, and would be given an opportunity to make a written

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6 Gazette number 25 of 29 June 2005, p. 1444.

7 Gazette number 45 of 16 November 2005, p. 2764; advising that the closing date for written submissions was 31 March 2006.

8 RMA investigation, as notified in Gazette number 36 of 12 September 2007, pp.2501.

submission and in turn a complementary oral submission referable to the new information.

24. On 26 May 2008 the Council wrote to the other Applicant (Mr FR) and to Mrs F, Mr G, Mr T, the Repatriation Commission, and the Military Rehabilitation and Compensation Commission (the Commissions) (the interested parties) advising them[9]:

    e. that the review by the Council of Statements of Principles numbers 9 and 10 of 2005 was ongoing; and

    f. of the RMA declaration of 14 April 2008 not to amend Statements of Principles numbers 9 and 10 of 2005 in relation to dioxin exposure and dioxin contaminated herbicide exposure;

    g. that the 14 April 2008 declaration by the RMA refers to 26 new pieces of information that had come to light since Statements of Principles numbers 9 and 10 of 2005 were determined by the RMA on 24 February 2005; and

    h. that the Council had written to applicant (Mr AR) in similar terms and that if Mr AR were to submit a new application to the Council, this would enable the Council to take into account the new information.

25. A third application, dated 12 June 2008 for review of Statements of Principles numbers 9 and 10 of 2005 was received by the Council on 18 June 2008. The application was in identical terms to the second application (above) and was from that same applicant. Specifically the application was concerned with the decision of the RMA of 14 April 2008 not to include exposure to dioxins as a factor in those Statements of Principles.

26. Pursuant to section 196ZB of the VEA the Council published in the Gazette an amending notice of its intention to carry out a review of all the information available to the RMA about chronic lymphoid leukaemia, and inviting persons or organisations authorised so to do to make submissions to the Council[10]. The Council gazetted three subsequent notices as to the dates by which written submissions must be received by the Council[11].

The information sent by the RMA to the Council

27. The RMA is obliged under section 196K of the VEA to send to the Council all the information that was available to it (the RMA) when it determined, amended, or last amended the Statements of Principles. That comprises all the information that was

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9 Mrs F, Mr G, Mr T and the Commissions made written submissions to the Council in respect to the Gazette Notices numbered 25 of 29 June 2005 and 45 of 16 November 2005 respectively.

10 Gazette number 40 of 14 October 2009, p. 2618.

11 Gazette number 2 of 20 January 2010, p. 94; Special Gazette number S231 of 30 December 2010; Special Gazette number S17 of 28 January 2011, advising that the closing date for written submissions was 7 February 2011.

available to the RMA when it determined the original Statements of Principles in 1995, and all the information subsequently available at all times when the Statements of Principles have been amended, or revoked and replaced, up to and including that information which was available in February 2005 when the RMA determined the Statements of Principles under review, and that information available up to April 2008 when the RMA determined not to amend those Statements of Principles (under review). In other words, within 28 days after being notified that the Council has been asked to conduct a review, the RMA must send to the Council all the information in respect of Chronic Lymphoid Leukaemia which was in the possession of the RMA at the time it (the RMA) made the decisions that triggered the Council's review.

28. In 2005, within the 28 day period specified in section 196K of the VEA, the RMA sent to the Council the information the RMA advised was available to (before) it at the relevant times and provided to the Council lists of that information.

29. In November 2005 the RMA sent to the Council further information the RMA advised was available to (before) it at the relevant times.

30. In April 2009, the RMA resent to the Council the information the RMA advised was available to (before) it at the relevant times and provided to the Council new lists of that information. The RMA sent the information, which included the information it had sent to the Council in 2005 and the information which was available to the RMA when it decided on 14 April 2008 not to amend the Statements of Principles 9 and 10 of 2005, to the Council via electronic access to that information through the service provider – FileForce (FileForce).

31. In August 2005 and November 2005 copies of the 2005 information was sent to the applicants (Mr FR and Mr AR respectively) and to the representative of the Commissions.[12] Ultimately, by correspondence dated 3 September 2010, the Council provided to the applicants (Mr FR and Mr AR), and to Mrs F, Mr G, Mr T, and the representative of the Commissions, electronic access to the information which the RMA advised was available to (before) it at the relevant times. [13]

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12 Mr T (an eligible person under section 196ZA of the VEA), who contacted the Council in April 2005, requested that he be kept informed of the progress of the review, but advised he was unable to make a written submission.

13 An [other] potentially eligible person Mr M contacted the Council in December 2010, in respect to a Vetaffairs 2010 (vol. 26, no. 4 December, p. 3) article on the SMRC, which provided details of Reviews being undertaken by the SMRC requesting the Council to review a 8 March 2003 decision in respect to a claim for compensation concerning exposure to benzene and chronic lymphoid leukaemia. The Council referred MR M to the appropriate officer in DVA and advised Mr M that it did not have jurisdiction in respect to claims for compensation. Further, the Council advised that if Mr M confirmed he was an eligible person he may make a submission to the Council in respect of the information which was available to (before) the RMA at the relevant times, concerning chronic lymphoid leukaemia. Mr M did not make a written submission to the Council by the date on which written submissions must be received.

PROPOSED SCOPE OF REVIEW AND PROPOSED POOL OF INFORMATION

32. On 11 March 2011 the Council wrote to the Applicants (Mr FR and Mr AR), Mrs F, Mr G, Mr T, Mr M [14] and the Commissions advising them of the Council's preliminary decisions of the scope of the review and pool of information. Each was invited to make written comments as to these preliminary views by close of business on 25 March 2011, and to make any oral comments at the hearing of oral submissions complementing the written submissions.

Scope of review

33. The Council's preliminary decision, as advised to the Applicants (Mr FR and Mr AR), Mrs F, Mr G, Mr T, Mr M and the Repatriation Commission on 11 March 2011 was as follows:

> Without limiting the scope of the Council's review of (some or the whole of) the contents of the Statements of Principles, the Council presently proposes to have particular regard to whether there was sound medical-scientific evidence upon which the Repatriation Medical Authority (the RMA) could have relied to amend either or both of the Statements of Principles for either or both of the clinical:
>
> i) onset; and/or
>
> ii) worsening
>
> of chronic lymphoid leukaemia in the following way:
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> a) the possible inclusion rather than the existing exclusion in the kind of injury, disease or death set out in paragraph 2. (b) of both of the Statements of Principles of 'small lymphocytic lymphoma'; and
>
> of small lymphocytic lymphoma and B-cell chronic lymphoid leukaemia in the following ways:
>
> b) the possible inclusion of factors for which the Applicant and Eligible Persons who made submissions contend for:
>
> - Radiation - ionising or other; includes radio, microwave VHF & UHF and Aircraft radar including early radar systems in 1940-50 British Lancaster Bombers;
>
> - Chemicals - includes all aromatic hydrocarbons – Benzene, Toluene, Xylene, Carbon Tetrachloride and others as contended; and

[14] See footnote 13 above.

- Aircraft fuel (AVGas) and associated chemicals (MEK, sealants) and exposure due to F111 fuel tank repairs;

- Herbicides and Pesticides;

- Asbestos; and

- Tobacco (by which the Council understands the Applicant to mean tobacco smoking).

Pool of information

34. The Council's preliminary decision, as advised to the Applicants (Mr FR and Mr AR), Mrs F, Mr G, Mr T, Mr M and the Commissions on 11 March 2011 was that the pool of information should be identified from the information that was available to (before) the RMA when it determined, amended, or last amended the Statements of Principles, sent to the Council by the RMA under section 196K (in 2005 and subsequently in 2009) and should comprise sound medical-scientific evidence as defined in section 5AB(2) of the VEA being information which:

   – epidemiologists would consider appropriate to take into account; and

   – in the Council's view, 'touches on' (is relevant to) those contended factors advised in the preliminary decision on the proposed scope of review and has been evaluated by the Council according to epidemiological criteria, including the Bradford Hill criteria.[15]

35. A copy of the preliminary list of the proposed pool of information was forwarded to the Applicants (Mr FR and Mr AR), Mrs F, Mr G, Mr T, Mr M and the Commissions, and is attached at **Appendix A**.

36. No comment was received by the Council from the Applicants (Mr FR and Mr AR), Mrs F, Mr G, Mr T and the Commissions regarding the Council's preliminary decision on the scope of review or the Council's preliminary decision on the pool of information.

37. At the hearing of oral submissions held on 31 March 2011, the Council advised the Applicants (Mr FR and Mr AR) and the Commissions representative that a article by Guenel, P et al. 1993 [16], which the Council had included in its preliminary list of the

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15 see Bradford Hill, A (1965) 'The Environment and Disease: Association or Causation?' *Proceedings of the Royal Society of Medicine* Section of Occupational Medicine, Meeting January 14, pages 295 to 300.

16 Guenel, P et al. 1993, 'Incidence of cancer in persons with occupational exposure to electromagnetic fields in Denmark', *British Journal of Industrial Medicine*, vol. 50, pp. 758-763.

Council provided copies of this paper to Mr RF, Mr AR and the Commissions representative on 31 March 2011. The Council did not receive comment on Guenel, P et al. 1993 or any other article in its preliminary list of the proposed pool of information.

proposed pool of information was not on Fileforce and copies of the article were provided.

ORAL SUBMISSIONS COMPLEMENTING THE WRITTEN SUBMISSIONS

38. The Council held a meeting to hear oral submissions complementing the written submissions on Thursday 31 March 2011. The Applicants (Mr FR and Mr AR) and a Medical Officer with the Department of Veterans' Affairs representing the Commissions made oral submissions complementing their respective written submissions.

39. Mrs F and Mr G did not make an oral submission, notwithstanding they were afforded the opportunity to do so. Mr T and Mr M did not make a written submission to the Council and therefore could not make a complementing oral submission.

PREVIOUS COUNCIL REVIEW OF STATEMENTS OF PRINCIPLES IN RESPECT OF CHRONIC LYMPHOID LEUKAEMIA

40. The Council has previously considered similar contended factors in a previous review of now revoked Statements of Principles in respect of chronic lymphoid leukaemia. The Minister has appointed, and the Convener has selected, a newly constituted Council, to conduct this review, to ensure that there is no apprehension of bias or prejudgement[17].

41. On 25 September 2003 the previously constituted Council published its Declarations and Reasons for Decision in respect of the now revoked Statements of Principles Nos. 67 and 68 of 2001[18].

42. The Council holds a copy of the previously constituted Council's Declarations and Reasons for Decision and a copy was included in the information which was forwarded to this Council by the RMA[19]. However, the Council took the view that it should not rely on the (previously constituted) Council's Reasons, but that it should consider for itself all the information that was before the RMA at the relevant times.

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17 Noting, however, that the Presiding Councillor (only) was a member of the previously constituted Council as a Councillor. The Presiding Councillor has only a deliberative vote (subsection 196K(5)).

18 The previous Council's 25 September 2003 Declarations were published by Gazette Notice 39 of 1 October 2003, pp. 2878-2882. The Declarations and Reasons for Decision are on the SMRC web site at www.smrc.gov.au.

19 Previous Council's 25 September 2003 Declarations at FileForce Item ID 15842 & 15843 and Reasons for Decisions at Item ID 15841.

43. The Council was cognisant that not all the information before it had been before the (previously constituted) Council when it had considered the now revoked Statements of Principles. Accordingly, the Council approached its task without any preconceptions, applying the two-stage process discussed below to the entirety of the information which was before the RMA at the relevant times.

APPLICANT'S SUBMISSIONS

Mr FR

44. In his application of April 2005, Mr FR stated that his grounds for review were as follows:

> My 02/04 submission [to the RMA] was technically sound, used peer reviewed scientific evidence to support my contention that several factors exist which positively identify the causative links of CLL with exposure to both EMR and several toxic and carcinogenic chemicals and materials.
>
> T-Cell CLL is relatively rare and only accounts for about 2% of CLL patients. The most common form is T-Cell CLL which accounts for about 98% of all CLL cases. It appears that the RMA has ignored B-Cell CLL side of the argument by again disregarding valid evidence that other factors exist which are associated with it.

45. Mr FR made a number of comprehensive written submissions, all of which were taken into account by the Council. These comprised an undated written submission sent to the Council by email on 17 June 2005 as an updated addendum to a written submission to the RMA dated 16 February 2004, and written submission prepared on or about 10 November 2005 sent to the Council by email on 14 December 2005, a written submission prepared on or about 23 February 2006, and a written submission prepared on or about 14 December 2010 with attached further information[20] respectively. Further, the Council considered the written submissions Mr FR made to the RMA in 2000, and 2004, which were in the information the RMA

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[20] Rossie, JP 2009, [LETTER] Department of Veterans Affairs, Washington DC, Blue Water Navy Vietnam Veterans Association, pp. 1-3 (Annex 3, December 2010 Mr FR Submission to SMRC).

Institute of Medicine 2009, *Veterans and Agent Orange: update 2008*, Committee to Review the Health Effects in Vietnam Veterans of Exposure to Herbicides (Seventh Biennial Update), The National Academic Press, Washington DC, Accessed by Applicant n.d. via http://www.nap.edu/openbook.php?record_id+12662&page+652. (Annex 4, December 2010 Mr FR Submission to SMRC).

Wilson, EJ et al. 2005, *The Third Australian Vietnam Veterans Mortality Study*, Department of Veterans' Affairs, Canberra. RMA ID 41296 and FileForce ID 19090 (Annex 5, December 2010 Mr FR Submission to SMRC).

Hugo, V 2008, *Men become accustomed to poison by degrees*, Executive summary of an address to Vietnam Veterans Federation, ACT Branch, ACT, pp. 1-9. (Annex 6, December 2010 Mr FR Submission to SMRC).

These 3 articles above (excluding Wilson, EJ et al. 2005) were not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

sent to the Council in 2005 and 2009. On request from the Council Mr FR also submitted by email and mail respectively:

a. dated 24 and 26 March 2011, a summary of the references of articles upon which he relied upon;

b. dated July 2012 the list of 31 of 35 articles [21] to which he referred as missing from the RMA reference list.

As mentioned above, the Applicant made a comprehensive oral submission complementing his written submissions[22].

46. At hearing of oral submissions on 31 March 2011 Mr FR provided copies of two articles[23] which he stated he had not submitted to the RMA and wanted the Council to consider. The Council advised the Applicant that he could send the articles to the RMA asking the RMA to undertake an investigation on the basis of them and any other new information that the RMA may identify.

47. In summary, Mr FR's primary submission was that there was sound medical-scientific evidence upon which the RMA could have relied to have included multiple contended factors in the Statements of Principles.

> I will show that small lymphoid leukaemia [sic] must be included in the SOP for CLL and argue that exposure to EMR can cause DNA strand breaks, chromosomal damage and this leading to cell transformation, mutations, cancers and that benzene, agent orange and other known carcinogenic chemicals and toxic substances such as perchloroethylene,

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21 Mr FR provided a copy of the index to his 16 February 2004 submission to the RMA and SMRC claiming that it was 31 of these refereed journal articles that did not appear on the RMA reference list. The Council noted the submission itself (FF No. 15808) and 7 separate documents from the index were in the information sent by the RMA to the SMRC. In any case the Council considered all the submissions and attached documents that Mr FR submitted to the RMA and to the SMRC. The summary of Mr FR's submissions are at [45] to [51.8] of these Reasons.

22 The discussion of the Applicants submissions set out in these Reasons is derived from the written submissions and the complementary oral submission here identified. The Council has not identified in which of the Applicant's submissions, individual citations and quotes appear as the Applicant submitted multiple copies of the information he relied upon in the various submissions he made to the RMA and to Council (see above). Further, it has not been possible to identify with any certainty whether an asserted quote was that of an author of an article or of the Applicant himself; hence page numbers for quotes have only been included in these Reasons when an actual article page number was obvious.

23 Goldsmith, JR 1997, 'Epidemiologic evidence relevant to radar (microwave) effects', *Environmental Health Perspectives*, vol. 105, suppl. 6, pp. 1579-1587. RMA ID 14259.

The Council notes that this article was information available to (before) the RMA at the relevant times.

Pirogova, E Vojisavljevic, V Cosic, L 2008, *Non-thermal effects of 500MHz – 900Mhz microwave radiation on enzyme kinetics*, Engineering in Medicine and Biology Society, 30th Annual International Conference of the IEEE.

This article was not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

tetrachloroethylene, trichloroethylene, polychlorinated biphenyls and others [24] identified in my 2004/5 submission should be included as factors in the SOP for CLL. [25]

48. Mr FR contended that the definition of CLL in Statements of Principles 9 and 10 of 2005, as not amended in 14 April 2008, was not in keeping with the latest medical science [26], in that the definition specifically excluded small lymphocytic lymphoma (SLL).

> This RMA definition of CLL contradicts the views of many researchers who conclude that B-Cell Small Lymphocytic Lymphoma (B-SLL) and mature B-Cell Chronic Lymphocytic Leukemia (B-CLL) are not separate entities but one and the same disease at different stages. This view, supported by the Revised European American Lymphoma (REAL) classification scheme (Harris et al. 1994), which is widely accepted and was adopted by the WHO, considers B-cell CLL and SLL (a subtype of non-Hodgkin's lymphoma) to be a single disease entity, in recognition of the biologic and clinical similarities between these B-lymphocyte malignancies (Harris et al. 1999).

a. Harris NL et al 1994[27]

> …small lymphocytic, consistent with CLL…(at p. 1365)

b. Harris NL et al in Jaffe, ES Harris, NL et al. 2001[28]

> …B-cell chronic lymphocytic leukaemia and B-cell small lymphocytic lymphoma are simply different manifestations of the same neoplasm…(at p. 13)

c. Lin, Y n.d.[29]

> The new WHO 'Classification for tumours of hematopoietical and lymphoid tissue; does not recognise these two [SLL & CLL] as separate entities'.

d. Rai, KR & Keating, MJ, n.d. [30]

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24 Toluene etc 2004/4 submission at page 6.

25 Oral hearing of 31 March 2011, transcript at p. 9.

26 Mr FR cited 39 medical science articles – Submission of 23 February 2006, at pp. 15-17.

27 Harris, NE et al. 1994, 'A revised European-American classification of lymphoid neoplasms: a proposal from the International Lymphoma Study Group', *Blood*, vol 84, pp. 1361-1392. RMA 20964

> Harris, NE et al. 1994 – In Richardson, DB et al 2005, 'Ionizing radiation and chronic lymphocytic leukemia', *Environ Health Perspectives*, vol. 113, no. 1,pp. (not cited) accessed by the Applicant n.d. via http://ehp.niehs.nih.gov/members/2004/7433/7433.html

28 Harris, NL et al. 2001, *WHO classification of tumours of haematopoietic and lymphoid tissues: Introduction*, pp. 12-13, 109-117, 119-187, 189-203. In Jaffe, ES Harris, NL Stein, H and Vardiman, JW eds. 2001, *Pathology and genetics of tumours of haematopoietic and lymphoid tissue,* International Agency for Research on Cancer (IARC), IARC Press, Lyon. RMA ID 28596.

29 Lin, Y M.D n.d. accessed by Applicant n.d via http://www.clin-path.com/html/newsletters/jan2002.html, cited in February 2006 Submission to the Council at p.11.

30 Rai, KR MD Keating, MJ MD n.d. accessed by Applicant n.d via http://patients.uptodate.com/topic.asp?file=leukemia/12356;

and in similar terms:

> Freedman, AS & Harris, NL n.d. *Clinical and pathologic features of small lymphocytic, prolymphocytic, and lymphoplasmacytic lymphomas*, accessed by Applicant n.d via

> According to the current WHO classification, B-cell CLL is considered identical (ie, one disease at different stages) to the mature (peripheral) B-cell neoplasm small lymphocytic lymphoma…

49. Mr FR further contended that the contents of Statements of Principles 9 and 10 of 2005, as not amended in 14 April 2008, focussed on T-cell CLL. He questioned why factors relating to the 98% of patients with B-cell CLL were ignored.

a. School of Medicine, University of Virginia n.d.[31]

> Immunophenotype:
>
> In CLL the malignant lymphocytes are almost always B cells (98%). Only 2% are of T cells. The CLL-B lymphocytes weakly express surface immunoglobulin IgM or IgM & IgD…

b. Kantarjian, H ed 1996[32]

> While the vast majority of patients with chronic lymphocytic leukaemia (CLL) have classical B-cell CLL, a number of patients have the misfortune not to be in this category. A small number of patients will have a T-cell CLL (in our experience < 2% of cases)…

Mr FR submitted that his written submissions to the RMA and to the Council concerned evidence which he contended supported the factors he was contending should be included in Statements of Principles 9 and 10 of 2005 in respect to B-cell CLL.

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http://patients.uptodate.com/topic.asp?file=lymphoma/9728&title=SLL+%28Small+lymphocytic+lymphoma%29; and

Harris, NL et al. 1997 *World Health Organization Classification of Neoplastic Diseases of the Hematopoietic and Lymphoid Tissues*, Report of the Clinical Advisory Committee Meeting—Airlie House, Virginia, accessed by the Applicant n.d. via http://www.biocolleges.org/colhemato/Hemato/OMS.htm.

Alkan, S & Karcher, DS 1996, 'Indolent lymphomas, classic subtypes and newer entities, Pathology Update', *Cancer Control Journal*, vol. 3, no. 2, accessed by Applicant n.d. via http://www.moffitt.org/moffittapps/ccj/v3n2/dept3.html.

The Applicant Mr FR included in his submission the 40 citations of medical science studies referenced in the Alkan, S & Karcher, DS 1996 article.

National Cancer Institute, National Institutes of Health, n.d. *B-cell neoplasms***,** accessed by the Applicant n.d. via (http://www.cancer.gov/cancertopics/pdq/treatment/adult-non-hodgkins/HealthProfessional/page2)

National Cancer Institute n.d. *Cellular classification of Adult Non-Hodgkin Lymphoma*, accessed by the Applicant n.d. via http://www.cancer.gov/cancertopics/pdq/treatment/adult-non-hodgkins/healthprofessional/page2

All six documents are cited in June 2005 (at pp. 15-22) and February 2006 Submission to the Council (at pp.11-18).

31 School of Medicine, University of Virginia, *Pathology Thread, chronic lymphoid proliferations*, accessed by Applicant n.d. via http://www.med-ed.virginia.edu/courses/path/innes/wcd/lympleuk.cfm, cited in February 2006 Submission to the Council at p. 10.

32 Kantarjian, H MD ed. 1996, *Leukemia Insights Newsletter*, vol.1, no. 4 Spring, MD Anderson Cancer Centre, University of Texas, accessed by Applicant n.d via http://www.mdanderson.org/leukemia/insight/letter14.html, cited in February 2006 Submission to the Council at p.10.

50. Mr FR's comprehensive submissions to the Council were on the basis of his interpretation of a large number of articles in support of each of his contended factors. Included in his written submissions were the Applicants commentary on articles, what appear to be verbatim copies of abstracts and / or complete reports and articles, including the reference lists from some of those reports / articles, or internet website links within his submission to access reports / articles directly. Some of these articles / reports were information that was available to (before) the RMA, but some were not information that was available to (before) the RMA, at the relevant times[33], They were all contended by the Applicant to be sound medical-scientific evidence.

51. Mr FR contended that in his 12 and a half years of service between 1958 and 1970, his exposure to EMR exceeded safety standards and that he was exposed to various chemicals. In support of his claim of unique circumstances, Mr FR provided a comprehensive work history, including examples of the equipment he worked on, his general exposure to EMR, which he estimated totalled at least 6590 hours[34], details of two significant radar events[35] and made oral submissions on the basis of these contentions and his interpretation of articles in support of the factors the Applicant contends should be included in the Statements of Principles under review.

> …my situation was unique…I had an old man's disease when relatively young [diagnosed in 1986 at age 46[36]]…active and healthy…no family history of cancers including CLL.

51.1. Radiation - ionising or other; includes EMR, radio, microwave VHF & UHF – Mr FR submits;

> My RAN employment was the most likely source of my CLL as it had exposed me to many of the known hazards associated with CLL…exposure to levels of radar EMR that far

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33 The Council has identified in the footnote citations of articles those that were information available to (before) the RMA at the relevant times and those that the Council could only consider as new information. Council notes that the Applicant has made multiple comments on many of the articles he cites as those articles are cited in numerous reviews or articles. Council has not attempted to cross-reference the Applicants comments or the article / review citations. Also see [119].

34 Mr FR provided his work history in written submissions to the RMA in 2000 & 2004 and to the SMRC in addition to those submissions, further submissions in 2005 (June and December) and in his oral submission (2011 electronic copy) presented at Councils hearing of oral submissions complimenting writing submissions held on 31 March 2011.

35 The meaning (US Dept of Commence wiki web site) of abbreviations used by the Applicant, in cited articles, and in paragraph [50] are:

EMR – electromagnetic radiation

RF – radio frequency of 3 KHz to 300 GHz

VSWR test probe – voltage standing wave ratio test probe

HF – high frequency of 3 MHz to 30 MHz (short wave)

LF – low frequency of 30 KHz to 300 KHz (loran)

VHF – very high frequency of 30 MHz to 300 MHz

UHF – ultra high frequency of 300 MHz to 3 GHz (analogue TV and analogue cell phones.

36 Oral hearing of 31 March 2011, transcript at p. 9.

exceeded maximum levels of safe radiation limits. Exposure to ELF, LF, HF and micro FR known as EMR during my enlistment period, passive exposure to toxic and carcinogenic vapours in the workplace and the unprotected use of toxic and carcinogenic chemicals and materials including asbestos and beryllium…[37]

In the 1960's…accidentally sprayed with insecticide from a helicopter…the spray was not identified[38].

…I maintained VHF transceivers, AI-17 and Mk19B aircraft radars, and repaired the Ground Controlled Approach (GCA) and Decca Airfield radars. I received a 16000 volt shock and RF burns to my fingers when the radar I was working on was accidentally re-powered. I also worked for two hours on two consecutive days on the high powered ground controlled approach radar that leaked vast amounts of EMR [1962-1964][39].

…277 radar…[1964] exposed to excessive EMR radiation for 15 minutes as a VSWR test probe had been incorrectly fitted in the wave pipe…the radar was transmitting during this period and I received burns to my fingers…[40]

This period [1964-65]…I was responsible for operating, operation of high power HF and LF transmitters and VHF and UHF transceivers[41].

I maintained squadron [1965] avionic systems and serviced Gannet and Venom aircraft radars and HF and VHF transceivers in the base repair section for 6 weeks…on the flight deck [1964-65] supervising and testing squadron avionic systems prior to launch and recovery[42].

Items requiring repair included APS-88 type – APS88 and Doppler radar from 725 squadrons, Sky Hawk air intercept radars, Bell Iroquois 'Hudat' Station keeping radars and electronic counter measure systems from 725 and 816 squadrons….To attend to the repair backlog after my deployment the APS-88 radar ran all day and actively transmitted four to five hours daily…the distance separating me from the radar antenna or dummy load was two metres....[43]

I therefore contend that I have suffered from two separate [in 1960-61 and in 1962] and deliberate close range exposures from a 200 kilowatt active radar, a succession of significantly high EMR exposures that for three years and a half years exceeded maximum safe working standards, unprotected use and exposure to toxic and carcinogenic chemicals

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37 Oral hearing of 31 March 2011, transcript at p. 10.

38 Oral hearing of 31 March 2011, transcript at p. 11.

39 Oral hearing of 31 March 2011, transcript at p. 13.

40 Ibid.

41 Ibid.

42 Ibid.

43 Oral hearing of 31 March 2011, transcript at p. 15.

and lastly the cumulative effect of 12 years of EMR exposure, increasingly compromising already stressed genetic systems.[44]

The first [200 kilowatt active radar] exposure was not calculated and the second exposure at 25 metres was calculated…to be 80.52 milliwatts per centremetre squared or 80.5 times greater than the maximum allowable safe exposure level[45].

My last three and a half years of employment, my work conservatively equalled or exceeded 4087 hours in an environment that was 42 and a half times greater than the maximum allowable safe radiation levels.

a. Feychting, M Forssen, U and Floderus, B 1997[46]

Subjects in the highest category of occupational exposure to magnetic fields (0.20 micro T, or 2 mG) had nearly double the risk of developing acute myeloid leukaemia, a 40 % increase in risk of developing chronic myeloid leukemia and a 70% increase in risk for chronic lymphoid leukemia when compared with unexposed subjects.

Among subjects who had high exposures to magnetic fields at home and at work, the risk…doubled for chronic lymphocytic leukemia.

b. Farrell, J n.d.[47]

Scientists working in the field of "bioelectromagnetics" are now convinced that man-made EMFs disturb biological processes, and that there is a substantial body of scientific evidence pointing to a relationship between EMFs and cancer.

The Applicant Mr FR included in his submission the 62 citations of medical science studies referenced in this article.

c. Litovitz, T et al. 1993[48]

[authors]…proposed that living cells discriminate against thermal noise fields by recognising them as spatially incoherent, i.e. uncorrelated at different receptor locations on the cell membrane.

The team suggested that biological cooperativity…(bacterial chemotaxis)…is an essential feature of cellular response to EMF.

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44 Oral hearing of 31 March 2011, transcript at p. 18.

45 Oral hearing of 31 March 2011, transcript at p. 17. Mr FR states that the calculation was undertaken by Dr Roy (the then Director of Non-Ionising Radiation Branch ARPANZA at the CSIRO) in 2000 and is based on Australian standard AS-2772, which Mr FR states is…the safe radiation exposure level is 1 / one milliwatt per centimetre squared (transcript at p. 12).

46 Feychting, M Forssen, U and Floderus, B 1997, 'Occupational and residential magnetic field exposure and leukemia and central nervous system tumors', *Epidemiology*, vol 8, no. 4, pp. 384-389. RMA ID 20987.

47 Farrell, J n.d. *The scientific controversy*, accessed by the applicant n.d. via www.icswebsite.com/emf/emfissues/emfissuesreferences.html.

48 Litovitz T et al. 1993, *Spatial and temporal coherence affects the response of biological systems to electromagnetic fields*, Electricity and magnetism in Biology and Medicine, Martin Blank ED. In Farrell, J n.d. *The scientific controversy*, accessed by the applicant n.d. via www.icswebsite.com/emf/emfissues/emfissuesreferences.html.

d. Kundi, M et al. 1999[49]

> …the "Vienna Resolution"…participants agreed that biological effects from low-intensity exposures are scientifically established. However, the current state of scientific consensus is inadequate to derive reliable exposure standards.

e. NIEHS n.d.[50]

> More than 100 epidemiological studies have shown an association between residential and occupational EMF exposure and many types of cancer.
>
> This scientific evidence led…the NIEHS to conclude…1998, that extremely low frequency (ELF) electromagnetic fields should be regarded as possible carcinogens.

f. Cherry, N 2000[51]

> …Dr Cherry concludes:"Many scientific studies have found chromosome aberrations and DNA damage with RF/MW exposure, the first being published in 1959.
>
> Two primary biological mechanisms are linked to these effects, calcium ion efflux and melatonin reduction….Hence, there is strong evidence that ELF and RF/MW is associated with accelerated aging (enhanced cell death and cancer) and moods, depression, suicide, anger, rage and violence, primarily through alteration of cellular calcium ions and melatonin/serotonin balance."

g. Slessin, N 2003[52]

> The IAC agrees with the NIEHS conclusion about health risk. Namely, "ELF EMR exposure cannot be recognised as entirely safe because of weak scientific evidence that exposure may pose a leukemia hazard" and "…the associations reported for childhood leukemia and adult chronic lymphocytic leukemia can not be dismissed easily as random or negative findings."

h. Cherry, N n.d.[53]

Included in this conference presentation about EMR and breast cancer, Dr Cherry provides data on RF and leukemia[54]:

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49 Kundi, M et al. 1999, *Vienna Resolution, Workshop on possible biological and health effects from RF electromagnetic fields*, October 25-28, 1998, University of Vienna Institute of Environmental Hygiene. In Farrell, J n.d. The scientific controversy, accessed by the applicant n.d. via www.icswebsite.com/emf/emfissues/emfissuesreferences.html.

50 National Institute of Environmental Health Sciences NIEHS n.d. *Epidemiological studies associating EMF exposure to disease*, accessed by the applicant n.d. via www.icswebsite.com/emf/emfissues/emfissuesreferences.html.

51 Cherry, N 1998, *Actual or potential effects of ELF and RF/MW radiation on accelerating ageing of human, animal or plant cells*, Lincoln University, Auckland New Zealand. In Farrell, J n.d. *The scientific controversy*, accessed by the applicant n.d. via www.icswebsite.com/emf/emfissues/emfissuesreferences.html.

52 Slessin, N 2003, *A report on Non-Ionizing radiation*, March/April Microwave news.

53 Cherry, N no date, *Electromagnetic radiation and cancer - Implications for reducing breast cancer risk*, pp. 1-10. From a presentation taken from Proceedings of World breast Cancer Conference, accessed via www.worldbreastcancerconf.ca?Archives/C28DrNeil Cherry.doc, pp. 1-10. RMA ID 1.18 F8 (68) FF ItemID 15913.

Table 1: A summary of epidemiological studies involving adult leukaemia mortality or incidence, ranked by probable RF/MW exposure category.

Study Reference Exposure Leukaemia Risk 95% Confidence

Category Type Ratio Interval

Polish Military Szmigielski (1996) High ALL 5.75 1.22 - 18.16,

Mortality CML 13.90 6.72 - 22.12,

AML 8.62 3.54 - 13.67,

All Leuk. 6.31 3.12 - 14.32

Korean War Robinette et al. (1980) High Leuk/Lymp 2.22 1.02 - 4.81

Radar Exposure (Mortality) AT/ET

Radio and TV Milham (1985) Moderate Acute Leuk. 3.44

Repairman Leuk. 1.76

Amateur Radio Milham (1988) Moderate AML 1.79 1.03 - 2.85

(Mortality)

UK Sutton Dolk et al. (1997a) Moderate Leuk. 1.83 1.22 - 2.74

Coldfield < = 2km

North Sydney Hocking et al. (1996) Low All Leuk. 1.17 0.96 - 1.43

TV/FM towers ALL+CML 1.39 1.00 - 1.92

(Mortality) AML+CML 1.01 0.82 - 1.24

Other Leuk. 1.57 1.01 - 2.46

UK TV/FM Dolk et al. (1997b) Low Adult Leuk. 1.03 1.00 - 1.07

(incidence)

Note: ALL: Acute Lymphatic Leukemia; CLL: Chronic Lymphatic Leukemia; AML Acute Myeloid Leukemia; CML: Chronic Myeloid Leukemia; and All Leuk.: All Adult Leukemia.

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54 Szmigielski, S 1996, 'Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation', *The Science of the Total Environment*, vol. 180, pp. 9-17. RMA ID 10413.

Robinette, CD Silverman, C and Jablon, S 1980, 'Effects upon health of occupational exposure to microwave radiation (radar)', *Am J of Epidemiology*, vol. 112, no. 1, pp. 39-53.

Milham, S 1985,' Mortality in workers exposed to electromagnetic fields', *Environ Health Perspectives*, vol. 62, pp. 297-300.

Milham, S 1988, 'Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies', *American Journal of Epidemiology*, vol. 127, no. 1, pp. 50-54. RMA ID 29753.

Dolk, H et al. 1997a, 'Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield Transmitter', *American Journal of Epidemiology*, vol. 145, no. 1, pp. 1-9. RMA ID 9620.

Dolk, H Elliot, P Shaddick, G Walls, P Thakrar, B 1997b, 'Cancer incidence near radio and television transmitters in Great Britain. II. All High Power Transmitters', *American Journal of Epidemiology*, vol. 145, no. 1, pp .10-17. RMA ID 9621.

Hocking, B Gordon, IR Grain, HL and Hatfield, GE 1996, 'Cancer incidence and mortality and proximity to TV towers', Medical Journal of Australia, vol. 165, no. 2, pp 601-605. RMA ID 14035.

i. Trower, B n.d.[55]

In this paper the author summarises 4 papers[56] that in his view illustrate the published peer reviewed and unpublished medical scientific evidence on EMR in support of his recommendation of a delay in the use of TETRA handsets by police in the United Kingdom.

The Applicant Mr FR included in his submission the 21 citations of medical science studies referenced in this report.

j. Mobile Phone Inquiry reports 2001[57]

> The inquiry found that research from around the world shows exposure to radiofrequency and microwave radiation can cause biological changes including DNA breaks, changes in the blood brain barrier, melatonin reduction and altering of calcium ion signalling.

k. Fist, S 1997[58]

> …Australian research study into the biological effects of radiation from digital cellular phones…
>
> The research showed a very significant increase in a form of B-cell lymphoma…B-cell (rather than the normal T-cell)…that B-cell effects are implicated in roughly 85% of all cancers

l. Fist, S 1997[59]

This paper reports the comments from a number of medical scientists commenting on the Fist, S 1997 study:

> Dr Neils Kuster – "It is incomprehensible to me that industry did not replicate this study 18 months ago, when the preliminary results became known."
>
> Dr John Goldsmith – "present startling new evidence that must be carefully evaluated".
>
> Dr John Stather – "this needs to be investigated thoroughly".
>
> Dr Stan Barnett – "The effect reported in this paper appears to be substantial."

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55 Trower, B n.d. *Confidential report on TETRA: Strictly for the Police Federation of England and Wales,* accessed via http://www.planningsanity.co.uk/reports/trower.htm.

56 Cherry, N 2000, *Evidence that Electromagnetic Radiation is Genotoxic: The implications for the epidemiology of cancer and cardiac, neurological and reproductive effects*, accessed at http://www.whale.to/b/cherry6.html, pp. 1-44.

Hyland 2000, *Potential adverse health impacts of mobile telephony memorandum* (no further citation provided).

Coghill, R 1998, *Are mobile telephony base stations a potential health hazard? A review of the present scientific literature* (no further citation provided).

Eklund, DA n.d. *New medical evidence on electromagnetic fields and health is alarming: Do not expose local people to mobile phone base stations* (no further citation provided).

57 Mobile phone Inquiry reports 2001, Accessed by the Applicant n.d. via http://www.democrats.org/lynallison/newsletter/Senate_update_june_2001.pdf.

58 Fist, S 1997, *The Adelaide mice study*, (no further citation provided by Applicant).

59 Fist, S 1997, *What they are saying: The Adelaide study*, supplied to the Applicant courtesy S. Fist.

Dr Gregory Lotz – "The findings are very significant,"…"They used a sizable number of animals, and it appears to be a clear effect."

Dr Ross Adey – "We now appear to have two, non-thermal effects, both linked to pulsed fields, and once again we must investigate the possibility that it is the low-frequency modulation that is the essential element"…

Dr Henry Lai – "The main point is that RF radiation promotes cancer"…"It is irresponsible and unwise to keep the data secret for two years, knowing their implications. The secrecy only reinforces the public's suspicion that the industry is trying to cover up."

m. Muntane, M n.d.[60]

…the CSIRO report lists many reproducible laboratory studies showing that radiofrequency power levels many hundreds of thousands of times smaller than even the existing maximum radiation exposure level of 1 mW/sq.cm., can have an adverse effect on the human immune system.

n. Unknown Author 1995[61]

…in 1994 the Spectrum Management Agency…asked CSIRO's Division of Radiophysics to undertake a comprehensive review of the world wide research results on the effects of radiofrequency radiation on the human body.

At radiofrequency power levels well below the threshold for thermal effects, the report lists many epidemiological studies that show an increased risk to disease such as leukaemia, eye cataracts and a suspected link with breast cancer.

o. Cherry, N 2000[62]

This literature review was prepared by the author for presentations to NZ Parliament, in Italy, Austria, Ireland and at the European Parliament in Brussels. The author presents the case for non-thermal effects from exposure to EMR (includes RF/MW and ELF fields) based on the findings of many studies referenced in this paper.

Hence RF/MW radiation has been confirmed to enhance DNA damage under RF/MW exposure from radar-like and cell phone exposures, including an exposure level which is 0.22% of the ICNIRP guideline.

There is more than sufficient evidence of chromosome aberrations, DNA strand breakage altered oncogenic activity and neoplastic transformations if cells to conclude that EMR across the spectrum from ELF to RF/MW is genotoxic. This is independently confirmed by the established biological mechanisms of calcium ion efflux and melatonin reduction.

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60 Muntane, M n.d. *Limit of 1mW/sq.cm is likely to be harmful to many people*, commenting on Barnett, S 1994, *Status of Research on Biological Effects and safety of Electromagnetic Radiation: Telecommunications Frequencies*, CSIRO, Division of Radiophysics.

61 Unknown author 1995, [LETTER] *Health effects of radiofrequency radiation*, sent to Senator Robert Bell.

62 Cherry, N 2000, *Evidence that Electromagnetic Radiation is Genotoxic: The implications for the epidemiology of cancer and cardiac, neurological and reproductive effects*, accessed at http://www.whale.to/b/cherry6.html, pp. 1-44. RMA ID 1.18 F8 (68) and FF ItemID 15898.

The paper provides the same data as the paper cited at paragraph 51.1 [f] and [h], in respect of RF/MW exposure findings and Leukaemia from military, occupational and residential studies. The author states that these studies:

> …show a global dose response relationship for increased adult leukaemia and RF/MW exposure with a dose-response threshold close to zero.
>
> …show a casual effect of adult and childhood leukaemia are levels of residential exposure involving exposure levels produced by cell sites out to over 500m.

The Applicant Mr FR included in his submission the 142 citations of medical science studies referenced in this report.

p. Cherry, N 2000[63]

In this literature review the author suggests that radiofrequency fields:

> With an odds ratio 1.15 95% CI 1.1-1.2,

are as dangerous as toxic chemicals;

> Carbon Tetrachloride – Odds ratio 1.13, 95% CI 1.1-1.2; source Cantor et al. 1995,

and contrasts the authors' interpretations and conclusions of the medical science literature with those of Dr Michael Repacholi from WHO, ICNIRP (International Commission on Non-Ionizing Radiation Protection) and others.

> Dr Repacholi relied heavily on the WHO 1993 review, that he referred to as "The Consensus of Science". He chaired the task group and was the technical editor of the report. The review report refers to a study that concludes that "In the RF range, the black body radiation" is "0.3mW/cm^2, when integrated up to 300 GHz, Repacholi (1983)."

The author contends that the Dr Michael Repacholi and WHO, ICNIRP view is based on only thermal effects. In contrast the author was a signatory to the Vienna and Salzburg Resolutions. They state

> …there are established biological effects of low level exposure to RF/MW radiation (Vienna), and that cell site exposures should be limited to 0.1mW/ cm^2 (Salzburg).

Further, in respect of the WHO 1993 review of six cancer related studies and the ICNIRP citing of 13 cancer related studies the author focuses on two studies[64] that the author claims the WHO and ICNIRP use to show RF/MW does not cause cancer in humans. The author's interpretation of these articles proving some support for a contrary view:

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63 Cherry, N 2000, *Health effects of Electromagnetic radiation Evidence for the Australian Senate Committee*, 8 September, pp. 1-63. Attached to Eligible Person 2001-2003, Email to SMRC. RMA ID 30574.

64 Lilienfeld, AM et al. 1978, *Foreign service health status study – evaluation of health status of foreign service and other employees from selected eastern European posts*, a final report (contract number 6025-619073) to the U.S. Dept of State, July 31.

Robinette, CD Silverman, C and Jablon, S 1980, 'Effects upon health of occupational exposure to microwave radiation (radar)', *Am J of Epidemiology*, vol. 112, no. 1, pp. 39-53.

> Blood samples from the Moscow [Lilienfeld et al. 1978] study show significant chromosome aberrations.
>
> This project…[Robinette et al. 1980]…whether radar exposure to service personnel during the Korean War produced health hazards….This data shows that radar exposure is (through extremely significant increases and significant dose-response relationships) casually related [to] increased mortality and illness, including cancer and diseases in many body organs.
>
> Hence the WHO, ICNIRP and authors conclusions are wrong.

Specifically, in respect of leukaemia the author provides the same data as the paper cited at paragraph 51.1 [o], in respect of RF/MW exposure findings and Leukaemia from military, occupational and residential studies.

> Leukaemia and Lymphoma are very RF-sensitive cancers, Szmigielski (1996), Milham (1985, 1988) Hocking et al. (1996). …Dolk et al 1997a. …shows elevated childhood leukaemia and brain tumor, and a set of dose-response relationships, which are likely to be highly significant, if related to realistic radial RF patterns, for cancer…including Leukaemia, Non-Hodgkin's Lymphoma…this is also consistent with Robinette et al. (1980), Szmigielski (1996), Milham (1985, 1988).

The author concludes with the recommendation of:

> …a maximum exposure level at the boundary of a property is set at 0.1mW/cm^2, then the indoor exposure will be less than 0.01mW/ cm^2, or 10nW/ cm^2…outdoor public exposure limit at the boundary of properties of 0.1mW/ cm^2, the Salzburg cell site limit.

The Applicant Mr FR included in his submission the 380 citations of medical science studies referenced in this report.

q. Penn State University 2001[65]

This paper examines historical and recent research on whether microwaves are capable of breaking the covalent bonds of DNA. Two possible processes are postulated:

> …what we have here is a two stage process of DNA covalent bond breakage resulting from oxygen radicals generated by microwave irradiation. This is one theory, and awaits experimental verification.
>
> An alternative theory…certain ions can stimulate DNA breakage and OH radical production (Kashige et al 1990, Kashige et al 1994). They also determined that amino sugars and derivatives could induce DNA strand breakage (Kashige et al 1991). It is possible that microwaves may be causing generation of cupric ions and hydroxyl radicals, and that auto-oxidation of aminosugars in solution are involved in DNA strand breakage (Watanabe et al 1990, Watanabe et al 1986). The link between microwaves and these secondary products remains to be established.[66]

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65 Penn State University, 2001, *Report on DNA and the microwave effect*. (Applicant has provided no further citation).

66 Kashige et al. 1990,'Function of cupric ion in the breakage of pBR322 ccc-DNA by D-Glucosamine', *Agric Biol Chem*, vol. 54, pp. 677-684.

The Applicant Mr FR included in his submission the 43 citations of medical science studies referenced in this report.

r. Balode, Z 1996[67]

> Blood samples were obtained from female Latvian Brown cows from a farm close to and in front of the Skrunda Radar and from cows in a control area.
>
> The counting of micronuclei in peripheral erythrocytes gave low average incidences, 0.6 per 1000 in the exposed group and 0.1 per 1000 in the control, but statistically significant ($P < 0.01$) differences were found in the frequency distribution between the control and the exposed groups.

s. Bortkiewicz, A et al. 1996[68]

> …study of the EMF effect on the circulatory system…The study indicated that exposure to EMF in parameters found in AM broadcast station increased risk for electrographic disturbances (detected by means of resting ECG and a 24-hour Holter recording) by six times in comparison with that in radio link station workers not exposed to medium wave EMF. In radio service workers this risk was twice as high as in link station workers. It seems that in AM broadcast station workers, resting ECG should be complemented by 24-hour Holter measurements, particularly if workers complain of the circulatory system disturbances.

t. Bortkiewicz, A et al. 1997[69]

> …study…to evaluate the function of the circulatory system in workers occupationally exposed to medium frequency electromagnetic fields. …at four AM broadcast stations

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Kashige, N et al. 1990, 'Correlation between DNA-breaking activity of aminosugars and the amount of oxygen molecules generated in their aqueous solutions', Agric Biol Chem vol. 55, pp. 1497-1505.

Kashige, N et al. 1994, 'Structure-activity relationships inn the induction of single-strand breakage in plasmid pBR322 DNA by amino sugars and derivatives', *Carbohydrate Research*, vol. 257, pp. 285-291.

Watanabe, K et al. 1985, 'DNA strand scission by d-glucosamine and its phosphates in plasmid pBR322' Agric Biol Chem vol. 50, pp. 1459-1465.

Watanabe, K et al. 1989, 'Specificity of nucleotide sequence in DNA cleavage induced by d-glucosamine and d-glucosamine-6-phosphate in the presence of Cu2+', Agric Biol Chem, vol. 54, pp. 519-525.

These 4 articles were not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

67 Balode, Z 1996, 'Assessment of radio-frequency electromagnetic radiation by the micronucleus test in bovine peripheral erythrocytes', *Sci Total Environ*, vol. 180, no. 1, pp. 81-86. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

68 Bortkiewicz, A et al. 1996, 'Evaluation of selected parameters of circulatory system function in various occupational groups exposed to high frequency electromagnetic fields, II, Electrocardiographic changes, *Med Pr*, vol. 47, no. 3, pp. 241-252. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

69 Bortkiewicz, A et al. 1997, 'Ambulatory ECG monitoring in workers exposed to electromagnetic fields, *J Med Eng Technol*, vol. 21, no 2, pp. 41-46. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

(0.738 - 1.503)… The electrocardiographic abnormalities detected in the resting and/or 24 h ECG were significantly more frequent (p=0.006) in workers exposed to electromagnetic fields than in non exposed subjects (75% versus 25%). A clear tendency for a higher number of rhythm disturbances (mostly ExV) was observed in AM broadcast station workers.

u. French, PW Donnellan, M & McKenzie, DR 1997[70]

A human astrocytoma cell line, U - 87 MG, was exposed to 835 MHz electromagnetic radiation for 20 min, 3 times per day for 7 days, at a power density of either 40+15 nWcm -2 or 8.1 + 3 mWcm-2. At the lower it was observed that the rate of DNA synthesis decreased, and that the cells flattened and spread out in comparison to the unexposed culture. At 40 mWcm-2, there were no effects seen in cell proliferation, but alteration in cell morphology included increased cell spreading and also the appearance of actin-containing blebs at localized sites on the membrane. It is hypothesised that 835 MHz radiation at low power density may be affecting a signal transduction pathway involved in cell proliferation.

v. Frey, AH 1998[71]

…headaches as a consequence of exposure to low intensity microwaves were reported in the literature 30 years ago…the blood-brain barrier appears to be involved in headaches, and low intensity microwave energy exposure effects the barrier…the dopamine-opiate systems of the brain appear to be involved in headaches, and low intensity electromagnetic energy exposure affects those systems.

w. Gadzicka, E et al. 1997[72]

The study covered male workers of middle wave broadcast stations (71) [exposed to EFM at the frequency of 1 Mhz], radio service (40) [exposed to EFM at about 150 Mhz] and radio line stations (42) [not exposed - control]…The study revealed that the mean arterial blood pressure and the day/night blood pressure variability indicator showed no significant differences between the groups, whereas the daily heart rate was significantly lower in the workers of middle wave broadcast stations in comparison with the controls despite similar type of work as far as physical effort and psychic burden are concerned, and similar non-occupational activities. The day/night heart rate variability indicator was significantly lower in the groups exposed. …In persons employed at radio service stations a higher incidence

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70 French, PW Donnellan, M and McKenzie, DR 1997, 'Electromagnetic radiation at 835 MHz changes the morphology and inhibits proliferation of a human astrocytoma cell line', *Bioelectrochem Bioenerg*, vol. 43, pp. 13-18. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

71 Frey, AH 1998, headaches from cellular telephones, are they real and what are the implications?,' *Environ Health Perspect*, vol. 106, no. 3, pp. 101-103. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

72 Gadzicka, E et al. 1997, 'Evaluation of selected functional circulation parameters of workers from various occupational groups exposed to electromagnetic fields of high frequency, III, 24-h monitoring of arterial blood pressure', *Med Pr*, vol. 48, no. 1, pp. 15-24. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

of the increased arterial blood pressure, in comparison with the control group, was observed.

x. Garaj-Vrhovac, V 1999[73]

…12 subjects occupationally exposed to microwave radiation. Results showed an increase in frequency of micronuclei (MN) as well as disturbances in the distribution of cells over the first, second and third mitotic division in exposed subjects compared to controls.

y. Hardell, L et al. 1999[74]

…case-control study on brain tumours and use of cellular telephones….Exposure was assessed by questionnaires supplemented over the phone.…Use of cellular telephones gave odds ratio (OR) = 0.98 [95%CI] =0.69-1.41. For the digital GSM system OR = 0.97, CI = 0.61-1.56 and for the analogue NMT system OR = 0.94, CI = 0.62-1.44 were calculated. Dose-response analysis and using different tumour induction periods gave similar results. Non significant increased risk was found for tumour in the temporal or occipital lobe on the same side as a cellular phone…used, right side OR = 2.45, CI = 0.78-7.76, left side OR = 2.40, CI 0.52-10.9. Increased risk was found for use of the NMT system.

z. Hardell, L et al. 2000[75]

…case control study…Work as a physician yielded an…OR…of 6.00…[CI of 0.62-57.7]…Radiotherapy of the head and neck region yielded an OR of 3.61 (95% CI, 0.65-19.9)…Medical diagnostic x-ray examination of the same area yielded an OR of 2.10 (95% CI, 1.25-3.53), with a tumour induction period of 5 years or more.

Chemical industry work yielded an OR of 4.10 (95% CI, 1.25-13.4), and laboratory work yielded an OR of 3.21 (95% CI, 1.16-8.85). Ipsilateral use of cellular telephones increased the risk for tumours in the temporal, temporoparietal, and occipital lobes (OR, 2.42; (95% CI, 0.97-6.05)… The result was further strengthened (OR, 2.62; 95% CI, 1.02-6.71) in multivariant analysis that included laboratory work and medical diagnostic x-ray investigation of the head and neck.

We conclude that low-power microwave exposure may act on HMC-1 cells.

aa. Hocking, B et al. 1996[76]

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73 Garaj-Vrhovac, V 1999, 'Micronucleus assay and lymphocyte mitotic activity in risk assessment of occupational exposure to microwave radiation', *Chemosphere*, vol. 39, no. 13, pp. 2301-2312. RMA ID 30040.

74 Hardell, L et al. 1999, 'Use of cellular telephones and the risk for brain tumours, a case-control study, *Int J Oncol*, vol. 15, no. 1, pp. 113-116. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

75 Hardell, L et al. 2000, 'A case-control study on radiology work, medical x-ray investigations and use of cellular telephones as risk factors for brain tumours' *Medscape General Medicine*. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

76 Hocking, B et al. 1996, 'Cancer incidence and mortality and proximity to TV towers', *Medical Journal of Australia*, vol. 165, no. 2, pp. 601-605. RMA ID 14035.

An ecological study comparing cancer incidence and mortality, 1972-1990…The calculated power density of the radiofrequency radiation in the exposed area ranged from 8.0 microW/cm2 near the towers to 0.2 microW/cm2 at a radius of 4km and 0.02 microW/cm2 at 12 km…[Setting – Northern Sydney, where the three TV towers have been broadcasting since 1956]

[Results:]…for all ages, the rate ratio for total leukaemia incidence was 1.24 [95% CI] 1.09-1.40. Among children, the rate ratio for leukaemia was 1.58 CI, 1.07-2.34 and for mortality 2.32 CI, 1.35-4.01. The rate ratio for childhood lymphatic leukaemia…was 1.55 CI, 1.00-2.41 for incidence and 2.74 CI, 1.42-5.27 for mortality.

bb. Hocking, B & Gordon, I 2000[77]

In a previous Study [above Hocking, B et al. 1996]… The rate ratio for incidence, comparing the inner ring of municipalities to the outer ring, was 1.55 (CI, 1.00-2.41) and for mortality the rate ratio was 2.74 (CI, 1.42-5.27). …the current study was to analyse the survival experience of the cases…

[Results:] There were 123 diagnosed cases of acute lymphatic leukaemia…which 29 (16 deaths) were in the inner ring…and 94 (34 deaths) were in the outer ring. …The 5 year survival in the inner ring was 55% and in the outer ring 71% (inner 23% worse); at 10 years the survival was 33% and 62% respectively (inner 47% worse). After adjustment for the potential confounders…the mortality ration comparing inner ring with outer ring was found to be 2.1 (CI, 1.1-4.0). We were not able to control for cytogenetic abnormalities.

cc. Jauchem, JR 1996[78]

In summary, studies have not yielded any obvious cardiovascular-related hazards of acute to long-term exposures to ELF EMFs [extremely low frequency electromagnetic radiation] or RFR [radio frequency radiation] at levels below current exposure standards.

dd. Johnson Liakouris, AG 1998[79]

A review of statistically significant health effects noted in the Lilienfeld Study provided evidence…establishing a possible correlation between health effects and chronic exposure to low-intensity, modulated Microwave radiation.

This paper [Kolodynski, AA & Kolodynski, VV 1996, 'Motor and psychological functions of school children living in the area of the Skrunda Radio Location Station (RLS) in Latvia, *Sci Total Envir*, vol. 180, no. 1, pp. 87-93.]… Motor function, memory and attention significantly

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77 Hocking, B & Gordon, I 2000, *Decreased survival for childhood leukaemia in proximity to TV towers*, Annual Scientific Meeting of the Royal Australasian College of Physicians, Adelaide, SA.

The Hocking, B & Gordon, I 2000 article was not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

78 Jauchem, JR 1996, 'Exposure to extremely-low-frequency electromagnetic fields and radiofrequency radiation: cardiovascular effects in humans', *Int Arch Occup Environ Health*, vol. 70, no. 1, pp 9-21. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

79 Johnson Liakouris, AG 1998, 'radiofrequency (RF) sickness in the Lilienfeld study, an effect of modulated microwaves?', *Arch Environ Health*, vol. 53, no. 3, pp. 236-238. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

differed between the exposed and control groups. Children living in front of the RLS had less developed memory and attention, their reaction time was slower and their neuromuscular apparatus endurance was decreased.

ee. Lai, H & Singh, NP 1997[80]

Since both melatonin and PBN are efficient free radical scavengers it is hypothesized that free radicals are involved in RFR-induced DNA damage in the brain cells of rats… Since cumulated DNA strand breaks inn brain cells can lead to neurodegenerative diseases and cancer and an excess of free radicals in cells has been suggested to be the cause of various human diseases, data from this study could have important implications for the health effects of RFR exposure.

ff. Malypapa, RS et al. 1998[81]

…we did not confirm the observation that DNA damage is produced in cells of the rat cerebral cortex or the hippocampus after a 2-h exposure to 2450 MHz CW [continuous wave] microwaves or at 4 h after exposure.

gg. Malypapa, RS et al. 1998[82]

…we conclude that the version of the alkaline comet assay…is as sensitive as other modifications of the comet assay…for the detection of DNA damage in cells exposed to low doses of ionizing radiation.

hh. Michelozzi, P et al. 1998[83]

…a small area study to investigate a cluster of leukemia near a high power radio-transmitter… The leukemia mortality within 3.5 km (5,863 inhabitants) was higher than expected (SMR=2.5, 95% CI 1.07-4.83)…after adjusting for socio-economic confounding, showed a significant decline in risk from the transmitter only among men (p=0.005).

ii. Neshev, NN & Kikilova, EI 1996[84]

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80 Lai, H & Singh, NP 1997, 'Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells', *Bioelectromagnetics*, vol. 18, no. 6, pp. 446-454. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

81 Malypapa, RS et al. 1998, 'DNA damage in rat brain cells after in vivo exposure to 2450 MHz electromagnetic radiation and various methods of euthanasia, *Radiat Res*, vol. 149, no. 6, pp. 637-645. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

82 Malypapa, RS et al. 1998, 'Detection of DNA damage by the alkaline comet assay after exposure to low-dose gamma radiation, *Radiat Res*, vol. 149, no. 4, pp. 396-400. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

83 Michelozzi, P et al. 1998, 'Risk of leukemia and residence near a radio transmitter in Italy, *Epidemiology*, vol. 9, p. 354. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

84 Neshev, NN & Kirilova, EI 1996, 'Environmental-health aspects of pulse-modulated microwaves, *Rev Environ Health*, vol. 11, no. 1-2, pp. 85-88. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

…Thus, short periods of exposure to pulse-modulated microwaves could be beneficial to cellular function, whereas maintaining the amplitude of such oscillations at a maximum for long periods may have a stressful effect on biochemical processes. The model discloses the possible environmental-health risks of long-term exposure in ambient fields that are created by radar, navigation, and communication systems.

jj. Persson, BRR Salford, LG and Brun, A 1997[85]

Biological effects of radiofrequency electromagnetic fields (EMF) on the blood-brain barrier (BBB)…studied in Fischer 344 rats…We have in total investigated 630 exposed rats at various modulation frequencies and 372 controls. The frequency of pathological rats is significantly increased ($P < 0.0001$) from 62/372 (ratio 0.17+0.02) for control rats to 244/630 (ratio: 0.39+0.043) in all exposed rats.

The frequency of pathological rats was 170/480 (0.35+0.03) among rats exposed to pulse modulated (PW) and 74/149 (0.50+0.07) among rats exposed to continuous wave exposure (CW). These results are both highly significantly different to their corresponding controls ($p < 0.0001$) and the frequency of pathological rats after exposure to pulsed radiation (PW) is significantly less ($p < 0.002$) than after exposure to continuous wave radiation (CW).

kk. Phillips, JL et al. 1998[86]

It was found that: 1) exposure of cells to the iDEN signal [pulsed signals at cellular telephone frequencies of 813.5625 MHz] at a SAR of 2.4 microwatt/g for 2 h or 21 h significantly decreased DNA damage; 2) exposure of cells to the TDMA signal [8.36.55 MHz] at an SAR of 2.6 microwatts/g for 2 h and 21 h significantly decreased DNA damage; 3) exposure of cells to the iDEN signal at an SAR of 24 microwatts/g for 2 h and 21 h significantly increased DNA damage; 4) exposure of cells to the TDMA signal at an SAR of 26 microwatts/g for 2 h significantly decreased DNA damage.

ll. Repacholi, MH et al. 1998[87]

Lymphoma risk was found to be significantly higher in the exposed mice than in controls (OR = 2.4. P = 0.006, 95%CI = 1.3-4.5).

mm. Sarkar, S 1999[88]

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85 Persson, BRR Salford, LG and Brun, A 1997, 'Blood-brain barrier permeability in rats exposed to electromagnetic fields used in wireless communication, *Wireless Network*, vol. 3, pp. 455-461. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

86 Phillips, JL et al. 1998, 'DNA damage in Molt-4 T-lymphoblastoid cells exposed to cellular telephone radiofrequency fields in vitro', *Bioelectrochem Bioenerg*, vol. 45, pp. 103-110. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

87 Repacholi, MH et al. 1997, 'Lymphomas in E mu-Pim1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields, Radiat Res, vol. 147, no. 5, pp. 631-640. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

88 Sarkar, S 1999, *Tandem repeat sequences as markers to study microwave-DNA interaction*, presented at the National seminar on Low-level electromagnetic field phenomena in biological systems, New Delhi, India. In Unknown Author, n.d. Recent studies (1995-2000) on the biological

DNA sequence was measured in cells of testis and brain of mice exposed to 2450-MHz microwaves (2 hrs daily for 10, 150, and 200 days at a SAR of 1.18 W/kg). DNA rearrangement was consistently observed in the DNA of exposed animals. The rearrangement can be attributed to some sort of non-specific stress created by low intensity microwave field.

nn. Schilling, CJ 1997[89]

Three men were accidentally exposed to high levels of ultrahigh frequency radiofrequency radiation (785 MHz mean frequency) while working on a television mast. They experienced an immediate sensation of intense heating of the arts of the body in the electromagnetic field followed by a variety of symptoms and signs which included pain, headache, numbness, and paresthesiae, malaise, diarrhoea, and skin erythema. The most notable problem was that of acute then chronic headache…

oo. Schilling, CJ 2000[90]

Six men…accidentally exposed to high levels of very high frequency (VHF) radiofrequency radiation (100 MHz)…transmission masts… They experienced symptoms and signs which included headache, paresthesiae, diarrhoea, malaise and lassitude… .

pp. Schirmacher, A et al. 1998[91]

An in vitro model of blood-brain barrier was used to study the effect of exposure to RFR (1.75 GHz, 217 Hz pulsed, 0.3 W/kg averaged over 1 g of tissue). Results indicate an increase of the radiation on permeability of the BBB-model. Highly significant differences between exposed and control cultures were obtained. Thermal effects could be excluded.

qq. Stagg, RB et al. 1997[92]

…hypothesis that modulated radiofrequency (RF) fields may act as a tumor-promoting agent b altering DNA synthesis, leading to increased cell proliferation… normal rat glial cells were exposed to 836.55 MHz, packet-modulated RF field at three power densities: 0.09, 0.9

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effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

89 Schilling, CJ 1997, ' Effects of acute exposure to ultrahigh radiofrequency radiation on three antenna engineers, *Occup Environ Med*, vol. 54, no. 4, pp. 281-284. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

90 Schilling, CJ 2000, 'Effects of exposure to very high frequency radiofrequency radiation on six antenna engineers in two separate incidents', *Occup Med*, vol. 60, pp. 49-56. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

91 Schirmacher, A et al. 1998, *Electomagnetic fields (1.75 GHz) influence the permeability of the blood-brain barrier in cell culture model*, presented at the Twentieth Annual Meeting of the Bioelectromagnetics Society, St Pete Beach, FL. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

92 Stagg, RB et al. 1997, 'DNA synthesis and cell proliferation in C6 glioma and pituitary glial cells exposed to a 836.55 MHz modulated radiofrequency field, *Bioelectromagnetics*, vol. 18, no. 1, pp. 230-236. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant).

and 9 mW/cm2, resulting in specific absorption rates (SARs) ranging from 0.15 to 59 muW/g.

…this modulated RF field did not increase cell proliferation of normal or transformed cultures of glial origin.

rr. Szmigielski, S 1996[93]

Subjects exposed occupationally to radiofrequencies (RF) and microwaves (MW) were selected…on the basis of their service records and documented exposures at service posts. …The cancer morbidity rate of the RF/MW-exposed personnel for all age groups (20-59 years) reached 119.1 per 100,000 annually (57.6 in non-exposed) with an OER [observed/expected ratio] of 2.07, significant at $P < 0.05$. The difference between observed and expected values results from higher morbidity rates due to neoplasm of…and malignancies of the haematopoietic system and lymphatic organs (OER = 6.31). Among malignancies of the haematopoietic/lymphatic systems, the largest differences in morbidity rates between exposed and non-exposed personnel were found for chronic myelocytic leukaemia (OER = 13.9), acute myeloblastic leukaemia (OER = 8.62) and non-Hodgkin lymphomas (OER = 5.82).

ss. Szmigielski, S et al. 1998[94]

The aim of this study was to determine the course of diurnal rhythms of blood pressure and heart rate in a group of workers exposed to various intensities of radiofrequency electromagnetic fields.

tt. Slesin, L ed. 2003[95]

Swicord, M & Cress, L 1993

"Of approximately eight chronic animal experiments known to us, five resulted in increased numbers of malignancies, accelerated progression of tumours, or both,"…

Salford, L & Persson, B n.d.

…report that they see nerve damage following a single two-hour exposure at a specific absorption rate (SAR) of 0.002 W/Kg. The effect becomes statistically significant at 0.02 W/Kg. These nonthermal levels are a hundred to a thousand times lower than the 2 W/Kg

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93 Szmigielski, S 1996, 'Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation', *The Science of the Total Environment*, vol. 180, pp. 9-17. RMAID 10413.

94 Szmigielski, S et al. 1998, 'Alteration of diurnal rhythms of blood pressure and heart rate to workers exposed to radiofrequency electromagnetic fields', *Blood Press Monit*, vol. 6, no. 6, pp. 323-330. In Unknown Author, n.d. Recent studies (1995-2000) on the biological effects of radiofrequency and cell phone radiation, (no further citation has been provided by Applicant and the results of the study were not provided by the Applicant's submission).

95 In Slesin, L ed. 2003, *Report on Non-Ionizing radiation*, Microwave News, accessed by the Applicant n.d. via www.microwavenews.com.

Swicord, M & Cress, L 1993, Memo, *Food and Drug Administration's Centre for Devices and Radiological Health*,

Salford, L & Persson, B n.d.

exposure standard recommended by the International Commission on Non-Ionizing Radiation Protection (ICNIRP).

…they again show that microwave radiation can impair the BBB, but they now add that the chemicals that leak through the BBB probably damage neurons in the cortex, the hippocampus and the basal ganglia of the brain.

uu. Cherry, N n.d.[96]

The author claims:

There is a large body of epidemiological scientific literature that is relevant to the assessment of RF/MW exposures risk of cancer. Almost all of these studies have not been referenced in the WHO/UNEP/IRPA review, WHO (1993), that is cited by the ICNIRP to be one of the" more detailed reviews".

The author states that:

Three of the studies[97] cited by WHO (1993) are omitted by ICNIRP. They are the study by Archimbaud et al (1989), and the Air Force base studies of Lester and Moore (1982) and Lester (1985) and Amateur Radio Study of Milham (1985).

WHO (1993) omits the[98] Wichita Kansas Study of Lester and Moore (1982a) and the Operator Electrical Workers Study of Miham (1985) and the Amateur Radio Operators study, Milham (1988).

The author comments on why in his view the WHO (1993) and ICNIRP (1998) should have considered these studies, and or provides reasons for these studies being persuasive.

Lester (1985) adjusted the analysis accordingly and concludes: "This strengthens the possibility of an association between some factor associated with AFBs [Air Force Bases] – our original hypothesis was microwave radiation…

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96 Cherry, N n.d. *Additional studies not cited by ICNIRP*, accessed by the Applicant n.d. via www.verdinrete.it/ondakiller.

Council notes that the articles by Cherry, N in particular, as cited by and commented upon by the Applicant and articles by other authors (commented upon by the Applicant) have cited a common pool of articles. The Council has not cross-referenced the articles or citations.

97 Archimbaud, E et al 1989, 'Acute myelogenous leukaemia following exposure to microwaves', British J of Haematology, vol. 73, no. 2, pp. 272-273.

Lester, JR & Moore, DF 1982b 'Cancer mortality and air force bases', J of Bioelectricity, vol, 1, no. 1, pp. 77-82.

Lester, JR 1985, 'Reply to Cancer mortality and air force bases, a revaluation', J of Bioelectricity, vol. 4, no. 1, pp. 129-131.

Milham, S 1985, Mortality in workers exposed to electromagnetic fields, Environ Health Perspectives, vol. 62, pp. 297-300. Also fn. 51, 95, 99, 229.

98 Lester, JR & Moore, DF 1982a, 'Cancer incidence and electromagnetic radiation, J of Bioelectricity, vol. 1, no. 1, pp. 59-76.

Miham, S 1985, op.cit. Also fn. 51, 94, 99, 229.

Miham, S 1988, 'Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies, Am J Epidemiology, vol. 127, no. 1, pp. 50-54. Also fn. 51, 94, 99, 229.

Lester and Moore (1982a)…They tested the hypothesis be separating populations which were exposed to no radar signals, living in valleys, one radar signal, on one or other hill, and two radar signals by living on the ridges. The cancer incidences are 303, 429, and 470 per 100,000 (1.00: 1.42-1.55). The dose-response association persisted through age, sex, race and socio-economic adjustments.

Miham (1985a)… and (1988)…showed elevated and significantly elevated cancer rates in many body organs.

Table 23: Summary of all site cancers from Robinette et al. (1980), using AT/ET except for Brain cancer (FT/ET), Milham (1985a), Szmigielski (1996) and for Dolk (1997a,b) using the maximum and/or significant result in the radial patterns.

Robinette [1980] Milham [1985a] Szmigielski [1996] Dolk (a) Dolk (b) [1997]

Exposure Regime RF/MW Mixed RF/MW

Relationship RR PMR RR O/E

Sample size (N) 202 2649 55,500 17409 13372

Symptoms

Leukaemia 2.22* 136* 6.31*** 1.74* 1.15

Non-Hodgkins Lymphoma 164** 5.82*** 1.30*

Acute Leukaemia (Lympho) 162** 5.75* 3.57 1.04

Acute Myeloblastic Leuk. 8.62*** 1.02 1.17

Chronic Myeloblastic Leuk. > 13.90*** 1.23

Chronic Lymphoblastic Leuk. 3.68** 2.56* 1.20 >

p-values * < 0.05; ** < 0.01; *** < 0.001

The author provides data from Milham (1988);

Elevated cancer rates were found for…Lymphoma + Leukaemia, SMR = 123 (99-152); Hodgkin's Disease, SMR = 123 (40-288); Leukaemia, SMR = 124 (87-172) and Other Lymphatic Tissue, SMR = 162 (117-218).

and concludes in respect of the four studies above and Milham:

…a great deal of consistency between several large studies which stand as proof, backed by many dose-response relationships, even at residential exposure levels, that RF/MW increases the risk of cancer over the whole body.

The author in reference to studies not cited by WHO nor ICNIRP, provides a brief summary of a number (27) of those studies[99] which are relevant to RF/MW

[99] Vignati, M & Giuliani, L 1997, 'Radio-frequency exposure near high-voltage lines', Environ. Health Perspectives, vol. 105, suppl. 6, pp. 1569-1573.

Djordevic, Z et al. 1979, 'A study of the health status of radar workers', Aviat space environ, vol. 50, pp. 396-398.

Friedman, HL 1981, [LETTER] 'Are chronic exposure to microwaves and polycythemia associated', New England J Med, vol. 304, no. 6, pp. 357-?.

Lin, RS et al 1985, 'Occupational exposure to electromagnetic fields and the occurrence of brain tumors', *J Occ Med*, vol. 27, pp. 413-419.

Maskarinec, G & Cooper, J 1993, 'Investigation of a childhood leukemia cluster near low-frequency radio towers in Hawaii, SER Meeting, Kestone, Colorado, June 16-18, *Am J Epidem*, vol. 138, p. 666.

Bini, MG et al. 1986, 'Exposure of workers to intense RF electric fields that leak from plastic sealers', *J Microwave Power*, vol. 21, pp. 33-44.

De Guire, L et al. 1987,'Increased incidence of malignant melanoma of the skin in workers in the telecommunications industry', *Brit J Indust Med*, vol. 45, pp. 824-828.

Thomas et al. 1987,

Preston-Martin, S et al. 1989, 'Risk factors for gliomas and meningiomas in males Los Angeles County', Cancer Research, vol. 49, p. 6137.

Johnson, CC & Spitz, CC 1989, 'Childhood nervous system tumors, an assessment of risk associated with parental operations involving use, repair or manufacture of electrical and electronic equipment', *Int J Epid*, vol. 18, p.756.

Nilsson, R et al. 1989, 'Microwave effects on the central nervous system – a study of radar mechanics', Health Physics, vol. 56, no. 5, pp. 777-779.

Hayes, RB et al. 1990, 'Occupational risk for testicular cancer: a case control study, *Int J Epid*, vol. 19, no. 4, pp. 825-831.

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. RMA ID 21150.

Savitz, DA & Chen, J 1990, 'Parental occupation and childhood cancer: Review of epidemiological studies, Environ Health Perspectives, vol. 88, pp. 325-337.

Tornqvist, S et al. 1991, 'Incidence of leukaemia and brain tumours in some 'electrical occupations'', Br J Industrial Medicine, vol. 48, pp. 597-603.

Quan, R et al. 1992,'Effects of microwave radiation on anti-infective factors in human milk', Paediatrics, vol. 89, no. 4, pp. 667-669.

Floderus, B Tornqvist, S and Stenlund, C 1994, 'Incidence of selected cancers in Swedish railway workers, 1961-79', *Cancer Causes and Control*, vol. 5, no. 2, pp. 189-194.

Loomis, DP Savitz, DA and Ananth, CV 1994, 'Breast cancer mortality among female electrical workers in the United States', *J Natl Cancer Institute*, vol. 86, no. 12, pp. 921-925.

Dosemeci, M & Blair, A 1994, 'Occupational cancer mortality among women employed in the telephone industry', *J Occup Med*, vol. 36, no. 11, pp. 1204-1209.

Savitz, DA and Loomis, DP 1995, 'Magnetic field exposure in relation to leukemia and brain cancer, mortality among electric utility workers', Am J Epidemiol, vol. 141, no. 2, pp. 123-34.

Cantor, KP et al. 1995, 'Occupational exposures and female breast cancer mortality in the United States', *J Occup Med*, vol. 37, no. 3, pp. 336-348.

Tynes, T et al. 1996, 'Incidence of breast cancer in Norwegian female radio and telegraph operators', Cancer Causes and Control, vol. 7, no. 2, pp. 197-204.

In respect of the Skrunda Radar:

Kolodynski, AA & Kolodynski, VV 1996, 'motor and psychological functions of school children living in the area of the Skrunda Radio location Station in Latvia', *Sci Tot Environ*, vol. 180, pp. 87-93.

Balode, Z 1996, 'Assessment of radio-frequency electromagnetic radiation by the micronucleus test in bovine peripheral erythrocytes', *Sci Total Environ*, vol. 180, no. 1, pp. 81-86.

Balodis, V et al. 1996, 'Does the Skrunda Radio Location Station diminish the radial growth of pine trees?", *Sci Total Environ*, vol. 180, pp.57-64.

Magone 1996, 'The effect of electromagnetic radiation from the Skrunda Radio location Station on Spirodela polyrhiza (L.) Schleiden cultures', *Sci Tot Environ*, vol 180, pp. 75-80.

Stenlund, C & Floderus, B 1997,(no further details are provided for this citation).

exposure. The Applicant Mr FR, in relation to his own occupational exposures highlighted the relevance of data from Garland et al. 1990:

> Navy Electrician's mate have an excess risk of leukaemia, RR = 2.4 (1.0-5.0)

The author concludes similarly to his paper at paragraph 51.1 [o], that:

> …the target for the recommended standard is 10 nW/cm^2.
>
> Since the mean Schumann Resonance intensity is close to 0.1 pW/ cm^{2,} and adverse health effects are observed for higher and lower intensity levels, then endogenous ELF and RF/MW modulated signals can interfere with hearts, brains and cells at extremely low intensities, approaching zero exposure. Hence minimization of exposure to protect workers and public health.

The Applicant Mr FR included in his submission the 464 citations of medical science studies referenced in this report.

vv. Hargreaves, P Ward, D and Cherry, N n.d.[100]

> We hope that the information presented here will encourage the Australian and new Zealand governments to thoroughly investigate health concerns, to abandon the ICNIRP out of date standard, and to adopt the new Salzburg Guidelines of 0.1 mW/cm. This is lower than that of Russia, China, Switzerland, Italy etc. who are already 1000 times lower ICNIRP.

This report discusses the views of many international medical scientists on the possible effects of exposure to electromagnetic radiation, provides human and animal case studies in relation to living near AM and or FM radio transmission towers, and discusses various health surveys and public hearings held in respect of possible effects of exposure to electromagnetic radiation, in New Zealand.

ww. Tornqvist, S et al. 1991[101]

> …study…to explore the association between occupations expected to be exposed to electromagnetic fields and the occurrence of leukaemia and brain tumours.
>
> For all selected "electrical occupations" the SMRs for total leukaemia and brain tumours were near unity.
>
> Increased risks were noted for all leukaemia among electrical/electronic engineers and technicians, (SMR 1.3; 95%CI 1.0-1.7) as well as in the sub-groups of telegraph/telephone (2.1; 1.1-3.6) and machine (2.6; 1.0-5.8) industries.
>
> Risk for chronic lymphoid leukaemia was increased in the same occupational category (1.7; 1.1-2.5) and in the sub-group of machine industry (4.8; 1.0-14.0), as well as for all. linesmen (2.0; 1.03.5) and power linesmen (2.8; 1.1-5.7).

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100 Hargreaves, P Ward, D and Cherry N n.d. *Hidden dangers of electromagnetic radiation from Communication towers, power lines and cell phones*, a report commissioned by Ross, R and Wilkinson, T of Christchurch Combined Residents Association, accessed by the Applicant n.d. via http://canterbury.cyberplace.org.nz/ouruhia/hidden.doc.

101 Tornqvist, S Knave, B Ahlbom, A and Persson T 1991, 'Incidence of leukaemia and brain tumours in some electrical occupations', *Br J Ind Med*, vol. 48, pp. 597-603. RMA ID 1693.

xx. Cherry, N 2000[102]

In 1995…Court…set a public exposure limit of 2m W/cm^2 for ..GSM cell site. …based on evidence of biological effects, including calcium ion efflux, enhanced ODC activity and EEG change down to 2.9m W/cm^2. There was also epidemiological evidence of childhood leukaemia at 2.4m W/cm^2. The decision also stated that this should be revised with new evidence. Subsequently two Australian studies were carried out to assure the public that both cell phones and cell sites were safe. Both of these studies, Hocking et al. (1996) and Repacholi et al. (1997), showed that leukaemia/lymphoma was more than doubled for people and mice.

Analogue cell phones use FM RF/MW signals and digital cell phones use pulsed microwaves that are very similar to radar signals.

The author / report refers to Lilienfeld et al 1978 and, Robinette, et al. 1980 discussed above, disputing that these studies show no adverse health effect.

Robinette et al. (1980)…Table 1: Mortality Incidence per 1000 and Risk Ratio (AT/ET) as an indication of the high exposure (AT) to low exposure (ET) difference.

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[102] Cherry, N 2000, 8 June *Health effects associated with mobile base stations in communities: the need for health studies*, pp. 1-36, accessed by Applicant n.d. via http://pages.britishlibrary.net/orange/cherryonexplevel.htm. RMA ID 1.18 F8 (68), FF ItemID 15916.

Hocking, B et al. 1996,

Repacholi, MN et al. 1997,

Lilienfeld, AM et al. 1978,

Robinette, CD et al. 1980,

Milham, S 1988,

Szmigielski, S 1996,

Selvin et al. 1992,

Dolk, H et al. 1997:

Altpeter, E.S et al. 1995, *Study of health effects of Shortwave Transmitter Station of Schwarzenburg, Berne, Switzerland*, University of Berne, Institute for Social and Preventative Medicine.

Abelin, T 1999, *Sleep disruption and melatonin reduction from exposure to a shortwave radio signal*, Seminar at Canterbury Regional Council, New Zealand. August.

Beall, C et al. 1996, Brain tumors among electronics industry workers', *Epidemiology*, vol. 7, no. 2, pp. 125-130.

Grayson, JK 1996, 'Radiation exposure, socioeconomic status, and brain tumour risk in the us air force, a nested case-control study'. *American J. of Epidemiology*, vol. 143, no. 5, pp. 480-486.

Thomas, TL et al. 1987, 'Brain tumor mortality risk among men with electrical and electronic jobs: A case-control study', *J. Nat. Canc. Inst*, vol. 79, no. 2, pp. 233-238.

Zaret, MM 1977, 'Potential hazards of hertzian radiation and tumors', *NY State J Med*, pp. 146-147.

Davis, RL and Mostofl, 1993, 'Cluster of testicular cancer in police officers exposed to hand-held radar', *Am. J. Indust. Med*, vol. 24, pp. 231-233.

Hayes, RB et al. 1990, 'Occupational risk for testicular cancer: a case control study, *Int J Epid*, vol. 19, no. 4, pp. 825-831.

Tynes, T et al. 1996, 'Incidence of breast cancer in Norwegian female radio and telegraph operators', *Cancer Causes Control*, vol. 7, no. 2, pp. 197-204.

Cantor, K.P et al. 1995, 'Occupational exposures and female breast cancer mortality in the United States', *Journal of Occupational Medicine*, vol. 37, no. 3, pp. 336-348.

Low High Risk Ratio 95%CI

Lymphatic/Hematopoietic 1.4 3.1 2.22 1.02-4.81

This shows a dose-response relationship for…death by leukaemia/Lymphoma.

Lilienfeld et al. 1978…Table 7: Cancer Mortality Rates: Adult Dependants

Obs. Exp. SMR (95%CI)

Leukaemia 10.3 3.4 (0.1-18.9)

This report submits that the above studies show elevated risk for leukaemia and that these findings are consistent with Milham, S 1988, and Szmigielski, S 1996, and then provides the same data as in Cherry, N 2000 above (cited at footnote 60) in which this author also provides the same data as his paper cited at footnote 50, in relation to the studies cited at footnote 51.

The author / report provides comment on the following epidemiological studies of residential RF/MW exposure:

– Selvin et al. 1992

…high powered outputs from the Tower [Sutra Tower, San Francisco]…over 980 kW of VHF TV and FM radio, and 18,270 kW of UHF TV…

Table 8: radial rings, with estimated population, Risk Ratios and Cumulative Risk Ratios, for white childhood brain tumor, Leukaemia, Leukaemia + Lymphoma, and All Cancer, in association with RF/MW exposure from the Sutra Tower, San Francisco.

Distance (km)	<0.99	1-1.99	2-2.49	2.5-2.99	3-3.49	3.5-3.99	4-4.49	4.5-4.99	5-5.99	6-8
Est. Population	1138	4334	3558	4489	5146	5566	4939	5386	8141	7988
Estimated personal mean dose in m W/cm2.	0.25	0.05	0.08	0.06	0.06	0.05	0.03	0.04	0.015	0.007
Leukaemia	1.26	1.32	2.02	1.92	1.67	1.80	2.03	1.33	0.53	1.26
Cumulative	1.26	1.31	1.59	1.70	1.69	1.72	1.77	1.70	1.48	1.44
Leuk + Lymph	2.47	1.08	2.63	2.08	2.54	1.85	2.27	1.56	0.57	1.05
Cumulative	2.47	1.37	1.86	1.94	2.10	2.05	2.08	2.00	1.73	1.62

The dose-response trend analysis uses a least squares fit… For leukaemia ($t = 3.31$, $p<0.01$), Leukaemia and Lymphoma combined ($t = 3.81$, $p<0.005$), Non-Hodgkin Lymphoma ($t = 1.94$, $p<0.05$) and Hodgkin Lymphoma ($t = 7.26$, $p<0.001$).

– Hocking, B et al. 1996

Table 9: Rate Ratios (RR) and 95% confidence intervals (CI) for cancer incidence and mortality in the population of the inner area compared to the outer area, adjusted for age, sex and calendar period.

Cancer Type RR (95% CI) Cases

Incidence
Total Leukaemia 1.24 (1.09-1.40) 1206
Lymphatic Leukaemia 1.32 (1.09-1.59) 536
Myeloid Leukaemia 1.09 (0.91-1.32) 563
Other Leukaemia 1.67 (1.12-2.49) 107
Mortality
Total Leukaemia 1.17 (0.96-1.43) 847
Lymphatic Leukaemia 1.39 (1.00-1.92) 267
Myeloid Leukaemia 1.01 (0.82-1.24) 493
Other Leukaemia 1.57 (1.01-2.46) 87

– Dolk, H et al. 1997

Radial adult leukaemia patterns for the 21 site UK study…

There are two types of radial transmission signals and two types of radial cancer patterns:

Type A: UHF signals that are low near the tower rise to a broad peak between 2 and 6 km and then decline with distance, Figure 6.

Type B: VHF signals have a peak within 1 km and decline with distance in an undulating fashion, Figure 2.

[Cancer rates near towers]…all follow a Type A pattern which is a dose response relationship of cancer rate as a function of mean exposure. This for all radial cancers outlined in the Tables they follow a dose response relationship appropriate to their radiation patterns.

…shows elevated childhood leukaemia and brain tumor, and a set of dose-response relationships which are likely to be highly significant…for cancer at a wide range of body sites including All Cancer, Leukaemia, Non-Hodgkin's Lymphoma, Brain Cancer, Bladder Cancer, Prostate Cancer, Skin Melanoma, Male and Female Breast Cancer and Colorectal Cancer. This is also consistent with Robinette et al. (1980), Szmigielski (1996) and Milham (1985, 1988).

– Alpeter et al. 1995 and Abelin 1999

This study investigated reductions in melatonin and sleep disturbance in people exposed to short wave radio stations and in relation to RF exposure.

The author / report summaries conclusions based on his interpretations of the studies reviewed, citing the studies and in some cases findings, by the various sources of exposure (Broadcast towers) and health effects. The author / report submits that these studies:

[form]…a coherent, consistent, integrated set of studies showing a causal relationship between sleep disturbance, chronic fatigue and cancer in association with extremely low mean RF exposure levels experiences in residential situations in the vicinity of radio and TV transmission towers.

Increase the incidence of many types of cancer, including leukaemia, brain tumor, testicular cancer, genitourinary and breast cancer, Robinette et al. (1980), Milham (1985, 1988), Szmigielski (1996), Hocking et al. (1996), Dolk et al. (1997 a, b), Beall et al. (1996), Grayson (1996), Thomas et al. (1987), Lilienfeld et al. (1978), Zaret (1989), Davis and Mostofl (1993), Hayes et al. (1990), Tynes et al. (1996), Cantor et al. (1995).

…over 40 studies have shown adverse biological or human health effects specifically from cell phone radiation. These research results to date clearly show that cell phones and cell phone radiation are a strong risk factor for all of the adverse health effects identified for EMR because they share the same biological mechanisms.

Because the biological mechanisms for cell phone radiation mimics that of EMR, and the dose response relationships have a threshold of ZERO, and this includes genetic damage, there is extremely strong evidence to conclude that cell sites are risk factors for: Cancer, especially brain tumour and leukaemia, but all other cancers also.

The Applicant Mr FR included in his submission the 176 citations of medical science studies referenced in this report.

yy. Gofman, J n.d.[103]

Many health professionals fear that such forms of energy have subtle effects on immune, nervous and endocrine systems. MRI (NMR) is not ionizing; it uses emf and rf waves to "excite" and "relax" hydrogen molecules. These excessive vibrations transfer their energy into heat. The real threat lies in their ability to produce "pulsating fields" of energy which cause atoms and molecules to become excited and vibrate, thus creating friction and heat. Heat can cause a chain of biological events; initially destruction of tissue, altered immune function, cell break-down and possible chromosomal damage.

Studies have found increased levels of leukemia among high power and telephone linemen, power station operators and shipyard electricians. Polish researchers discovered a 3-fold increase in the incidence of cancer in military personnel regularly exposed to microwaves and radio frequencies.

zz. Blask, D 2003[104]

Melatonin is involved in circadian rhythm regulation, sleep, hormonal expression of darkness, seasonal reproduction, retinal physiology, antioxidant free-radical scavenging, cardiovascular regulation, immune activity, cancer control, and lipid and glucose metabolism. It is also a new member of an expanding group of regulatory factors that control cell proliferation and loss and is the only known chronobiotic hormonal regulator of neoplastic cell growth… This is a potentially unifying model for melatonin's chronobiological inhibitory regulation of cancer growth in maintaining host-cancer balance.

aaa. McLean, L ed 2000[105]

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103 Gofman, J n.d. *Radiation sickness*, accessed by the Applicant n.d. via http://www.geocities.com/julanenova/files/research.doc.

104 Blask, D 2003, *Melatonin, chronobiology and cancer*, presented at Office of Cancer Complementary and Alternative Medicine, Invited Speaker Series February 2003 at Lipsett Auditorium National Institutes of Health.

Measurements of specific absorption rate (SAR) are an inappropriate method of ascertaining `safety' of RF technology.

The standard must take into account the athermal (below-heating) effects of EMR… The existing standard is based on the erroneous presumption that adverse health effects occur only if the body is heated by 1 degree C.

There is ample evidence that adverse effects occur at much lower, or athermal, levels that do not require heating of the body. According to renowned researcher,

Dr Ross Adey, "The laboratory evidence for athermal effects of both ELF and RF/Microwave fields now constitutes a major body of scientific literature in peer-reviewed journals. It is my personal view that to continue to ignore this work in the course of standard setting is irresponsible to the point of being a public scandal." (August 1995.)

AS2772.1 is outdated.

Exposure to electromagnetic radiation from power sources (P) or radiofrequency sources (RF) such as mobile phones and VDUs has been linked with a range of health problems as summarised below.

B3.2 Leukemia

Alfredsson (ELF), Cancer Causes Control 7(3), pp. 377, 1996

Wertheimer and Leeper (P), Am. J. Epidemiol. 109(3), pp. 273, 1979

Savitz (P), Am. J. Epidemiol. 128(1), pp. 21, 1998

Coleman (P), Br. J. Cancer 60(5), pp. 793, 1989

Juutilainen (P), Int. Arch. Occup. Environ. Health, 62(4), pp. 289, 1990

London (P), Am J. Epidemiol. 134(9), pp. 923, 1991

Feychting and Ahlbom (P), Am. J. Epidemiol. 138(7), pp. 467, 1993

Fajardo-Gutierrez (P), Bol. Med. Hosp. Infant Mex. 50(1), pp. 32, 1993

Matanoski (P), Am. J. Epidemiol. 137(6), pp. 609, 1993

London (P), Am. J. Ind. Med. 26(1), pp. 47, 1994

Theriault (P), Am. J. Epidemiol. 139(6), pp. 550, 1994

Wertheimer (P), Bioelectromagnetics 16(2), pp. 86, 1995

Miller (P), Am. J. Epidemiol. 144(2), pp. 150, 1996

Michaelis (P), Cancer Causes Control 8(2), pp. 167, 1997

Linet (P), New England J. Med. 337(1), pp. 1, 1997

Feychting (P), Epidemiology 8(4), pp. 384, 1997

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105 McLean, L ed. 2000, [LETTER & REPORT] to *Senate Inquiry into EMR and Executive Summary of report*, Electromagnetic Radiation Alliance of Australia.

Li (P), Epidemiology 8(1), pp. 25, 1997

Li (P), J. Occup. Environ. Med. 40(2), pp. 144, 1998

Wartenberg (P), Am. J. Public Health 88(12), pp. 1787, 1998

Green (P), Int. J. Cancer 82(2), pp. 161, 1999

Angelillo (P), Bulletin of WHO 77, pp. 906, 1999

Greenland (P), Microwave News Sept. Oct, 1999

Milham (RF), Environ. Health Perspect. 62, pp. 297, 1985

Milham (RF), Am. J. Epidemiol. 127(1), pp. 50, 1988

Floderus, Cancer Causes Control 4(5), pp. 465, 1993

Fear (P), Brit. J. Cancer 73(7), pp. 935, 1996

Dolk (RF), Am. J. Epidemiol. 145(1) pp. 1, 1997

The authors of the report comment (in the Applicant submission he also highlighted some sections in the report in support of his contentions on athermal effects of EMR) critically in respect of Standards Australia, Australian Communications Industry Forum Codes of Practice, Australian Radiation Protection and Nuclear Safety Agency's (ARPANSA) standards-setting process, National Health and Medical Research Council (NHMRC) guidelines for electric and magnetic fields and the views and standards developed by these organisations. Similarly, the policy response of Governments (Australian State and Federal) in respect of health effects of EMR are criticised as being inadequate.[106]

bbb. Woods, P 1997[107]

This is a copy of an address given by Councillor Peter Woods, President of the NSW Local Government Association, at the March 1997 Australian Institute of Environmental Health, Australian Institute of Building Surveyors, and Local Government & Shires Associations seminar 'Electromagnetic Radiation in the Environment: A Balanced view'.

ccc. Slesin, L 2003[108]

The author comments critically in respect of the National Institute of Environmental Health Sciences (NIEHS) and an NIEHS statement and refutes the statements medical science accuracy.

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106 Council notes that this aspect of Applicant Mr FR's submission contains no reference to any medical science that was available to (before) the RMA at the relevant times.

107 Woods, P 1997, *Opening address,* to Electromagnetic Radiation in the environment, a balanced view, Seminar of the Australian Institute of Environmental Health, the Australian Institute of Building Surveyors, NSW and Local Government & Shires Associations.

108 Slesin, L 2003, *US NIEHS advises - Its okay for children to live next to power lines*, Microwave News.

A statement posted on the institute's Web site advised: It doesn't matter whether or not a house is close to power lines. There is no valid association between nearby power lines and any cancer-including childhood leukemia.

ddd. Cherry, N 2000[109]

…the ICNIRP Guideline exposure level is set many orders of magnitude too high….It is based on the preconceived and long held view…that the only possible and only established biological effect of RF/MW exposure is tissue heating…This view has been intransigently maintained in the face of compelling laboratory and epidemiological evidence of adverse health effects that would have had a chemical declared carcinogenic…for humans many years ago.

The background RF/MW levels in Western cities are already in the range 1nW/cm^2 – 5nW/cm^2, except near cell sites and radio and TV towers. A practical option to avoid these demonstrated effects is to set the initial public exposure limit at 20 nW/cm^2 (0.02 μW/cm^2 aiming over 10 years, to reduce it to 10 nW/cm^2 (0.01 μW/cm^2)

ICNIRP's overall cancer assessment conclusion that: "Overall, the results of the small number of epidemiological studies published provide only limited information on cancer risk."

This conclusion is mistakenly based on flawed previous assessments, WHO (1993), inappropriate inclusion of (1) and (7), failure to review the data on effects (2, 3, and 4), incorrect claims of no significant effects when such effects are reported (5 and 6), failure to analyse the data in (8), inappropriate dismissal of significant well conducted studies (9 and 10) and inappropriate devaluing of residential studies (11, 12 and 13).

eee. Russell, P 1997[110]

The Australian Standard AS2772.1 - 1990 for radio frequency is one of the most stringent in

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109 Cherry, N 2000, *Criticism of the Health Assessment in the ICNIRP Guidelines for Radiofrequency and Microwave Radiation (100 kHz – 300 GHz). In* Cherry, N 2000, *Health effects of Electromagnetic radiation Evidence for the Australian Senate Committee*, 8 September, pp. 1-63. Attached to Eligible Person 2001-2003, Email to SMRC. RMA ID 30574.

(1) Barron, CI and Baraff, AA 1958, 'Medical considerations of exposure to microwaves (radar)', *Journal of the American Medical Association*, vol. 168, no. 9, pp. 1194-1199. RMA ID 25480.

(2) Robinette et al. 1980,

(3) Lilienfeld, AM et al. 1978,

(4) Selvin et al. 1992,

(5) Beall et al. 1996,

(6) Grayson1996,

(7) Rothman et al. 1996a,

(8) Rothman et al 1997b,

(9) Szmigielski et al. 1988,

(10) Szmigielski 1996

(11) Hocking et al. 1996

(12) Dolk et al. 1997a

(13) Dolk et al 1997b

110 Russell, P 1997, *Claims made on misinformation*, (no further citation provided by Applicant).

the Western world. It is set by Standards Australia, an independent non-government organisation, which includes representatives from the scientific community, Government agencies, the public, and industry.

The Australian and the international standard are based on solid scientific research and provide an ample margin of protection.

Meeting these stringent, science-based safety standards provide a sound basis for confidence that mobile phones and their base stations are safe.

fff. Unknown Author n.d.[111]

This is a critique of a study by Linet, M et al (1997) and its reported conclusion:

"Children exposed to electromagnetic fields by living near electrical power lines are not more susceptible to developing leukemia, a study released Wednesday shows."

The author states:

It is unfortunate that reporters and so-called experts who are now calling the NCI study as positive proof that a risk does not exist from long term exposure to powerline electromagnetic fields did not take the time to critically examine what the study had actually found, and also to examine the criteria which led to the NCI researcher's conclusions.

The NCI researchers actually acknowledge in no less than four places, a statistically significant increase in acute lymphoblastic leukaemia (ALL) in children exposed to powerline magnetic fields in excess of 3 milliGauss. This is a confirmation of many previous studies which have shown a similar level of association between childhood leukaemia and magnetic fields from electricity. The article in The Australian mentions that the researchers dismissed, as a "statistical fluke", a 24% increase in leukemia risk for children exposed to what is termed "especially high magnetic fields ".

The NCI researchers were able to dismiss this fact by arbitrarily setting a 2 mG level as a cut-off limit. The fact is that if they had used the 3 mG level as a cut off point in their calculations, the conclusions would have been exactly the opposite - that there is a significant risk.

ggg. Barron, CI & Baraff, AA 1958[112]

The applicant states that this is a discredited article:

The referencing of the six studies; Barron and Baraff 1958, Robinette et al 1980, Lilienfeld 1978, Selvin et al 1992, Beall et al 1996 and Grayson 1996 as finding no evidence of ill health effects cannot be regarded as justified in light of subsequent analysis of these studies by Dr. John Goldsmith, Ben Gurin University of the Negev, Israel, and Dr. Neil Cherry, Lincoln University, New Zealand. It was these errors in ICNIRP that constituted a significant part of the opposition to ICNIRP within the Standards Australia TE/7 Committee, mentioned above.

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111 Unknown Author n.d. *Putting a spin on science, the NCI Linet study*, (Linet, M et al. 1997, (study title not provided), New England J of Medicine).

112 Barron, CI & Baraff, AA 1958, 'Medical considerations of exposure to microwaves (radar)'. *JAMA*, vol,168, no. 9, pp. 1194-1199. RMA ID 25480.

hhh. Goldsmith, JR 1997[113]

[113] Goldsmith, JR 1997, 'Epidemiologic evidence relevant to radar (microwave) effects', *Environmental Health Perspectives*, vol. 105, suppl. 6, pp. 1579-1587. RMA ID 14259.

Ouellet-Hellstrom, R & Stewart, WF 1993, 'Miscarriages among female physiotherapists who report using radio and microwave frequency electromagnetic radiation', *Am J Epidemiol*, vol. 138, pp. 775-786.

Lilienfeld, AM et al. 1978, *Foreign service health status study – evaluation of health status of foreign service and other employees from selected eastern European posts*, a final report (contract number 6025-619073) to the U.S. Dept of State, July 31.

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and

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Vukelic M, Kontosic I, Jonjic A, Grubisic-Greblow H. Unpublished data.

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Garaj-Vhrovac, V Horvat, D and Koren, Z 1990, 'The effect of microwave radiation on the cell genome', *Mutat Res*, vol. 243, pp. 87-93.

Garaj-Vhrovac, V Horvat, D and Koren, Z 1991, 'The relation between colony-forming ability, chromosome aberrations, and incidence of micronuclei in V79 Chinese hamster cells exposed to microwave radiation', *Mutat Res*, vol. 263, pp.143-149.

Garaj-Vhrovac, V Fucic, A and Horvat, D 1992, 'The correlation between the frequency of micronuclei and specific chromosome aberrations in human lymphocytes exposed to microwaves', *Mutat Res*, vol. 281, pp. 181-186.

d'Ambrosio, G et al. 1995, 'Genotoxic effects of amplitude-modulated microwaves on human lymphocytes exposed *in vitro* under controlled conditions', *Electro Magnetobiol*, vol. 14, pp. 157-164.

Anderson, BS & Henderson, AK n.d. Unpublished data.

Maskarinec, G & Cooper, J 1993, [ABSTRACT] 'Investigation of a childhood leukemia cluster near low frequency radio towers in Hawaii', *Am J Epidemiol*, vol. 138, p. 666.

Hocking, B Gordon, IR Grain, HL and Hatfield, GE 1996, 'Cancer incidence and mortality and proximity to TV towers', *Medical Journal of Australia*, vol. 165, no. 2, pp. 601-605. RMA ID 14035.

This review updates previous reviews by the same author in 1995[114] assessing cancer risk from RF radiation.

> Ouellet-Hellstrom and Stewart (16) reported on a study of female physiotherapists who used either RF or short-wave apparatus… The frequency used by short-wave equipment was 27.12 MHz and by microwave equipment was 915 MHz and 2450 MHz.
>
> Of the case and control mothers, 11.9 and 9.5%, respectively, were using microwaves during pregnancy…the odds ratio (OR) for spontaneous abortion increased as the number of exposures increased from 5 or less to 20 or more per month. …For women exposed to short-wave radiation, 22.3% lost there baby…whereas the figure for unexposed women was 24.4%. Of the microwave-exposed women 47.7% has miscarriages…compared to 14.5% of non exposed women (at p.1580).
>
> Lilienfeld, AM et al. 1978 (17), [T]he exposures of U.S. embassy personnel in Moscow.
>
> (Table 1) Complications of pregnancy, childbirth, and puerperium (ICD-8 Codes 630-678) among women employees in the Foreign Service Health Status Study (FSHSS).

Ever [a,b]		**After index** [a,c]		***p***
Moscow	**Comparison**	**Moscow**	**Comparison**	0.04
19 (6%)	19 (3%)	11 (3.5%)	9 (1.3%)	
SMBR 1.7	SMBR 0.67			

> Abbreviations: SMBR, standardized morbidity ratio
> a Refers to the initial tours of duty during which exposures occurred.

Dolk, H et al. 1997, 'Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield Transmitter', *American Journal of Epidemiology*, vol. 145, no. 1, pp. 1-9. RMA ID 9620.

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Robinette, CD Silverman, C and Jablon, S 1980, 'Effects upon health of occupational exposure to microwave radiation (radar)', *Am J of Epidemiology*, vol. 112, no. 1, pp. 39-53.

Rothman, KJ et al. 1996, 'Assessment of cellular telephone and other radio frequency exposures for epidemiological research', Epidemiology, vol. 7, pp. 291-298.

Grayson, JK 1996, 'Radiation exposure, socioeconomic status, and brain tumour risk in the us air force, a nested case-control study'. *American J. of Epidemiology*, vol. 143, no. 5, pp. 480-486.

Szmigielski, S 1996, 'Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation', *The Science of the Total Environment*, vol. 180, pp. 9-17. RMA ID 10413.

Davis, RL & Mostoff, FK 1993, 'Cluster of testicular cancer in police officers exposed to hand-held radar', *Am J Ind Med*, vol. 24, pp. 231-233.

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. RMA ID 21150.

U.S. Environmental Protection Agency, n.d. Unpublished data. Cited in Sibbison, JB 1990, 'USA, Danger from electromagnetic fields', *Lancet*, vol. 336, no. 8707, p. 106.

[114] Goldsmith, JR 1995, 'Epidemiologic evidence of radiofrequency radiation (microwave) effects on health in military, broadcasting, and occupational studies', *International Journal of Occupational & Environmental Health*, vol. 1, no. 1, pp. 47-57. RMA ID 26401.

b Whether the condition occurred at any time; 'Whether the condition occurred after the initial tour of duty.

Daily (19) reported a statistically significant increase in immature red blood cells among workers exposed to radar.

Bach (23) found that rats exposed at 13 mW/cm^2 had changes in blood cell counts.

Goldini (24, 25) compared the haematological findings in 25 male air traffic control technicians exposed to radar… The radar was in the range of 1250 to 1350 MHz, with a strength varying from 10 to 20 μW/cm^2. …Radar-exposed workers had significantly lower levels of leukocytes and red blood cells than workers distant from the microwave source. In a follow up study of 49 radar-exposed technicians, thrombocyte and leukocyte counts decreased significantly but stayed within normal limits (25).

Tonascia and Tonascia (26)…hematological study of Moscow foreign service workers…found… Several statistically significant changes occurred over time in the Moscow group; specifically, mean hematocrit increased and a 3-fold increase in monocyte count occurred. Neutrophil percentages fell and then rose; the reverse pattern was observed for the lymphocytes

Vukelic et al. (27) studied the effects of RF radiation on physiotherapists and psychiatrists in Croatia. They found a significant positive correlation between length of service and white cell count, and an association of years of exposure with low red cell count.

Tornqvist et al. (28) studied 706 power station workers…found that the white blood cell counts were decreased slightly because of exposures to magnetic fields.

Garaj-Vrohac et al. (29) examined six men accidentally exposed while repairing microwave devices used for air traffic control…microwave field of 1250 to 1350 MHz with power density of 10 μg/W/cm^2 to 20 μg/W/cm^2… Both chromosomal and chromatid reactions occur.

Balode (30) …cattle in the field exposed *in vivo* near large military RF emitter in Skrunda…showed more positive micronuclei test results than unexposed cattle.

Scarfi (31)… Bovine lymphocytes *in vitro* respond to microwave exposure…

Garaj-Vhrovac (32)… Genotoxic changes are found in Chinese hamster cells in *vitro* (thymidine incorporation and chromosomal and chromotid changes) (32) and in human lymphocytes *in vitro* (33) using micronuclei tests.

Garaj-Vrohac et al. (29), (32), (33), (34) and d'Ambrosio (35)…have also demonstrated that radar exposures are mutagenic both *in vivo* and *in vitro*.

The authors state that:

…these findings are epidemiologically important because of the need for biological indicators of exposure and also because of the theory that somatic cell mutations lead to increased risk for cancer. The usefulness of such tests as a biological monitor seems clear from the data and the findings of excess numbers of mutation

Anderson (39)…to study the cancer incidence in the vicinity of radio broadcasting towers…

Table 4. Age-adjusted cancer and leukemia annual incidence rates for males and females in census tracts with broadcasting towers compared to those without such towers (Honolulu. Hawaii, 1979—1983) and compared to statewide rates per 100.000 (1978—1981).

	Males		**Females**	
Area	**Incidence**	**SIR[a]**	**Incidence**	**SIR[a]**
Leukaemia				

Tracts with towers	15.2 (15)	1.58	7.6 (8)	1.45
Tracts without towers	2.4 (1)	0.27	5.0 (2)	0.97
Statewide	9.4 (163)	-	5.3 (90)	-

a Standardized for age
b Number of cases in parentheses.
Statewide data are based on the Surveillance, Epidemiology, and End Results (SEER) Program Report 1973-1981.

Maskarinec (40)…a case-control study…a unusual number of children with leukemia in the small community… Based on the state's cancer registry, the number of cases to be expected was about one every 2 years or about seven in 14 years. Seven of the cases occurred during the 3 years 1982 to 1984. After 1985, case incidence returned to expected levels -one case every 2 years… Among the seven cases identified from 1982 to 1984, five were acute non lymphocytic leukemia (ANLL), whereas statewide, three of four cases were acute lymphocytic leukemia (ALL)…

In the case-control study of 14 cases and 56 matched controls of the same sex born within 6 months of the cases studied, no statistically significant risk factors were defined. There were, however, elevated OR for other cases of cancer in the family (OR=3.4 with 95% CI of 0.70-16.41) and for having ever resided within 2.2 miles of the Lualualei Naval Broadcast Facility and its two low frequency radio towers (OR 2.2; 95% CI of 0.65-7.56).

Hocking (11), for leukemia, however, the incidence rate ratio for adults was 1.24 (95% CI 1.09-1.40), whereas for children it was 1.58 (95% CI 1.07-2.34), with a mortality rate ratio of 2.32 (95% CI 1.35-4.01). The authors were unsuccessful in identifying confounders to explain these results.

For Dolk (12) the innermost area was within 2 km of the transmitter; adult leukemia relative risk (RR) was 1.83 (95% CI 1.22-2.74)

In a second Dolk et al. (13) study the same procedures were used to evaluate risks surrounding 20 other broadcast facilities in the UK for the same period; 3305 cases were identified, with an overall observed-to-expected (O/E) ratio of 1.03 (95% CI 1.0-1.07). Decline in risk with distance was significant for all sites combined… . In the band between 2 and 3 km from the transmitter the adult leukemia O/E ratio was 1.33.

Rothman et al. (9) tabulated studies that might relate leukemia to occupational or recreational exposures to RF radiation and studies that related such exposures to brain malignancies. The risk ratios for leukemia were >1.0 for 19 studies and 1 for 7.

Grayson (10) reported… The military-rank-adjusted OR is significantly elevated for RF: 1.38 (95% CI 1.01-1.90), but not for ELF: 1.28 (CI 0.95-1.74).

Szmigielski (41), who examined cancer by site among Polish military personnel during the period 1970 to 1989… The overall cancer morbidity for exposed personnel was 119.1/100,000 per year compared on an age-adjusted basis to 57.6 in the non exposed

group. The greatest O/E ratios were found for chronic myelocytic leukemia, 13.9; myeloblastic leukemia, 8.62; and non-Hodgkin's lymphoma, 5.82.

A cluster of six cases of testicular cancer among traffic policemen using microwave generators suggests that microwave exposures can cause cancer of the testicle (42).

Garland et al. (46) studied leukemia among occupational groups with potential electromagnetic field exposure in the U.S. Navy. …one occupational group, electrician's mate(s), showed consistent excess of risk for leukemia.

In the Robinette et al. (47) study, naval personnel were divided into occupational groups with low and high exposures by the occupational designator for the personnel.

Table 5. U.S. Naval personnel by occupational category during the Korean War and deaths by cause group. 1950 to 1974.

	Low Exposure				**High exposure**	
	RM	**RO**	**AE**	**ET**	**FT**	**AT**
No. of persons	9253	10,116	1412	13,078	3298	3733
Total deaths	296	308	61	441	144	198
From disease	161	165	22	199	81	77
From malignant disease	39	47	8	65	16	27
From malignancy of the lymphatic & hematopoietic systems	6	14	0	18	1	10

Table 6. U.S. Naval personnel by occupational category during the Korean War and crude death rates per 1000 by cause group, 1950 to 1974.

Total deaths rates	32.0	30.4	43.2	33.7	43.7	53.0
From disease	17.4	16.3	15.6	15.2	24.6*	20.62*
From malignant disease	4.21	4.65	5.66	4.97	4.85	7.23*
From malignancy of the lymphatic &	0.65	1.38	0.00	1.38	0.3	2.68*

hematopoietic systems

Death rates for the group with the highest exposure, aviation electronics technician(s) (AT), are significantly higher than those for the remaining men for all deaths, disease-related deaths, deaths from malignancy, and deaths from malignancy of the lymphatic and hematopoietic systems.

Table 7. Number of U.S. Naval personnel receiving Veterans Administration compensation in 1976, by diagnostic group, for two high-exposure groups (FT and AT) relative to the low-exposure groups exposed during the Korean War.

	FT & AT		All Others		FT & AT
Diagnostic group	**No.**	**Rate/1000**	**No.**	**Rate/1000**	**Expected No.**
Hemic, Lymphatic	3	0.4	10	0.30	2.08
Total diagnosis	477**	67.84	-	53.61	376.94
Total populations	7031		33,859		

* Significantly increased, $p < 0.05$, ** significant, $p < 0.01$

Abbreviations:

RM - radarman
RO – radioman
AE – aviation electrician's mate(s)
ET – electrician technician(s)
FT – fire control technician(s)
AT – aviation electronics technician(s)

Among the many tabulations from the Lilienfeld report (18), those for data about leukemia are shown in Table 8, based on data excerpted from the Lilienfeld report by Goldsmith (*8*). Although the numbers are small, there is significant excess for child dependents in both Moscow and other embassies, as well as an excess for employees and dependents in both locations. Estimated exposures at the Moscow embassy were from 5 to 18 $\mu W/cm^2$.

Table 8. Leukemia among U.S. embassy employees and child dependents in Moscow and other Eastern European embassies.

	Moscow embassy		Other embassies		Total
Population	**Obs**	**Exp**	**Obs**	**Exp**	**Observed / Expected**
Employees	2	0.8	3	1.7	5 / 2.5
Child dependents	2	0.5*	3	0.7*	5 / 1.2*
Total	4	1.3*	6	2.4*	10 / 3.7*

Significantly elevated O/E ration, $p < 0.05$. Based on table in Goldsmith (8).

The author concludes:

> Available data suggest that RF radiation be considered a carcinogenic risk, a position already taken in an internal U.S. EPA document (51) in 1990 when there was much less evidence of the potential harmfulness of RF radiation… The evidence may or may not justify more restrictive regulation of occupational exposure; for community exposures, however, the evidence justifies prudent avoidance

iii. Salford, LG et al. 2003[115]

> …serious neuronal damage in rat brains following exposure to microwave radiation from a cell phone…"…We cannot exclude that after some decades of (often) daily use, a whole generation of users may suffer negative effects, perhaps as early as middle age."

jjj. Moulder, J 1995[116]

> …radio frequency electromagnetic (RFEM) fields (3 kHz – 300 GHz)…These guidelines have been developed by panels of scientists and medical experts to protect human beings from known harmful levels of exposure to RFEM fields.
>
> The [American National Standards Institute/Institute of Electrical and Electronic Engineers] ANSI/IEEE (1992), [National Council on Radiation Protection and Measurements] NCRP (1996) and [International Radiation Protection Association] IRPA (1993) standards and recommendations are based on a [specific absorption rate] SAR of 4 W/kg threshold. Each incorporates safety factors to derive the recommendation that whole-body average exposure levels not exceed 0.4 W/kg in environments designated either occupational or "controlled", or 0.08 W/kg in environments designated either general-public or "uncontrolled".

115 Salford, LG et al. 2003, 'Nerve cell damage in mammalian brain after exposure to microwaves from GSM mobile phones', *Environmental Health Perspectives*, vol. 111, pp. 881-883, accessed by the Applicant n.d. via http://protectingourhealth.org/newscience/learning/2003/2003-0129salfordetal.htm.

116 Moulder, J 1995, 'Human exposure to microwaves and other radio frequency electromagnetic fields', *IEEE Engineering in Medicine and biology Magazine*, vol. 14, no. 3, pp. 336-337.

Mr FR submitted that he relied upon the data and findings[117] of Lilienfeld, Ady / Adey, Goldstein / Goldsmith, and others, which differed and were critical of the high level for exposures in these guidelines.

kkk. Richardson, DB et al. 2005[118]

> …current understanding of radiation-induced tumorigenesis and the etiology of lymphatic neoplasia provides a strong mechanistic basis for expecting that ionizing radiation exposure increases CLL risk.
>
> The epidemiologic evidence of radiation-CLL associations is weak; however, given the limitations of the reviewed studies, these findings do not offer a persuasive basis for concluding that CLL is an exception to general principles of radiation carcinogenesis. In addition, there is a problem of logical inconsistency if the government continues to assert that CLL is non radiationgenic whereas SLL is radiogenic. Contemporary classification schemes hold that B-cell CLL and SLL are analogous diseases and should be considered as a single disease entity.

The Applicant Mr FR included in his submission the 48 citations of medical science studies referenced in this article, which reviewed the molecular, clinical, and epidemiological evidence regarding the radiogenicity of CLL.

lll. Lai, H & Singh, NP 2004[119]

> [in rats brain cells]…exposure to a 60-Hz magnetic field at 0.01 mT for 24 hours caused a significant increase in DNA single- and double-strand breaks. Prolonging the exposure to 48 hr caused a larger increase.
>
> We hypothesize that exposure to 60-Hz magnetic field initiates an iron-mediated process (e.g., the Fenton reaction) that increases free radical formation in bran cells, leading to DNA strand breaks and cell death.

The Applicant Mr FR included in his submission the 69 citations of medical science studies referenced in this article.

mmm. Diem, E et al. 2005[120]

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117 Lilienfeld, AM et al. 1978, *Foreign service health status study – evaluation of health status of foreign service and other employees from selected eastern European posts*, a final report (contract number 6025-619073) to the U.S. Dept of State, July 31.

Ady (sic) Adey, WR – There are no publications in the available information, but Moulder, J 1995 cites.

Goldstein (sic) Goldsmith, JR – 1995 and 1997 publications are in the available information, but EE cites.

Hocking, B Gordon, IR Grain, HL and Hatfield, GE 1996, 'Cancer incidence and mortality and proximity to TV towers', *Medical Journal of Australia*, vol. 165, no. 2, pp. 601-605. RMA ID 14035.

118 Richardson, DB et al. 2005, 'Ionizing radiation and chronic lymphocytic leukemia', *Environmental Health Perspectives*, vol. 113, no. 1, accessed by the Applicant n.d. via http://ehp.niehs.nih.gov/members/2004/7433/7433.html

119 Lai, H & Singh, NP 2004, 'Magnetic-field-induced DNA strand breaks in brain cells of the rat', *Environmental Health Perspectives*, vol. 12, no. 6, pp. 687-694.

RF-EMF exposure (1800 MHz; SAR 1.2 or 2 W/kg; different modulations; during 4, 16 and 24 h; intermittent 5 min on/10 min off or continuous wave) induced DNA single- and double-strand breaks. Effects occurred after 16 h exposure in both cell types [cultured human diploid fibroblasts and cultured rat granulosa cells] and after different mobile-phone modulations. The intermittent exposure showed a stronger effect in the comet assay than continuous exposure.

nnn. Los Angeles Times 2003[121]

Federal and independent funding of medical research may not be sufficient to counterbalance the biases of research underwritten by private interests, but it is vastly greater in amount than independent funding available to examine health risks associated with chemical exposures. Here, research by chemical interests with an economic stake in the outcome dramatically outweighs independent investigations. **As a result, scientific literature on chemical exposures is littered with false assurances about safety**. (Applicant added emphasis).

ooo. Slessin, L 2005[122]

The Applicant Mr FR states:

[These] articles…discussing the more than apparent vested interests of big business such as the power/utility and telecommunications industries and their attempt to discredit, water down, or refute the findings of EMF/EMR researchers in regard to the harmful effects of exposures to these forms of radiation.

The authors' summaries of articles for 2005 and 2004 include criticisms of the approach by the WHO to electromagnetic fields (EMFs) on the basis that; important studies that highlight non-thermal effects were not included in the WHO health and biological effects literature reviews; Electric Utility representatives provided input into WHO documents and guidelines, whereas independent researchers in the main were not provided the same opportunities; the WHO claims cell phones are safe and children need take no special precautions, yet advocates in the media precautionary policies (promoting use of hands free headsets) to protect children; WHO discredited data and findings on children living near powerlines having a higher than expected rate of leukaemia; funding was required for research on TV antennas, home and work Wi-Fi hot spot transmitters, cell phones and brain cancer to follow-up on effects from 'regular use' at thousands of minutes per

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120 Diem, E et al. 2005, 'Non-thermal DNA breakage by mobile-phone radiation (1800 MHz) inhuman fibroblasts and in transformed GFSH-R17 rat granulosa cells in vitro', *Mut Res / Gen Tox and Environ Mutagenesis*, vol. 583, pp. 178-183.

121 Los Angeles Times, 24 January 2003, [EDITORIAL] *Funding can taint findings*, accessed by the Applicant n.d. via http://www.protectingourhealth .org/press/2003/2003-0124-LAT-taintedfindings.htm.

122 Slessin, L 2005, ed. – summaries of a Year of Article Reviews, Microwave News (no further citation provided by Applicant).

The Council noted the examples provided by Slessin, L 2005, but was unable to determine the relevant medical science data and or article findings that the Applicant wanted to highlight in support of his contentions concerning electromagnetic fields and chronic lymphoid leukaemia.

month (2005); and the inadequacy of EMF exposure guidelines, including those adopted in the UK, US.

51.2. Chemicals[123] - includes all aromatic hydrocarbons – Benzene, Toluene, Xylene, Carbon Tetrachloride and others as contended by Mr FR and he submits;

> **Polychlorinated Biphenols (PCBs):** …in 1964, I unknowingly handled PCB contaminated oil that regularly leaked from one of the HF transmitters. This oil was wiped from the floor twice weekly using bare hands and a rag, and from leaking capacitor cases.
>
> **Beryllia:** I frequently handled beryllium oxide insulators while repairing electronic equipment… Protective gloves, goggles or breathing masks that should be used when handling these items were not available… I had to contend with was Beryllium dust, created from the Jason pistols that were used to remove rust or paint from the ships hull, bulkheads or decks. Air ventilation came from outside air intakes that forced air below decks. If de-rusting took place near the air intakes, dust containing beryllium particles would be sucked in and distributed to living areas below decks.
>
> **Benzene:** I used a range of benzene-based solvents throughout my entire enlistment period. As was the norm during that period, no protective clothing or masks were used…

a. Gillig, R n.d.[124]

> The only known risk factor for other types of CLL [other than Adult T cell leukaemia] is exposure to benzene.

b. Sullivan, LM 1999[125]

> Service connection for chronic lymphocytic leukaemia is granted.

c. Savitz, DA & Andrews, KW 1997[126]

> Researchers at the University of North Carolina School of Public Health concluded that benzene was linked to many forms of leukemia including acute myelogenous leukemia AML as well as acute and chronic lymphocytic and myeloid leukemia.

d. Metzer Law Group n.d.[127]

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123 Mr FR provided an electronic copy of his oral submission, Oral hearing of 31 March 2011.

124 Gillig, R n.d. *General discussion on etiology of selected cancer types – leukemia*, Public Health Assessment, PSC Resources, Massachusetts.

125 Sullivan, LM 1999, *Appeal decision citation no. 9930795*, Board of Veterans' Appeals, Washington, DC. Cited in February 2006 Submission to the Council at pp. 19-20.

Council notes that this US DVA Appeal decision does not reflect the Council's two-step process in applying the reasonable hypothesis and balance of probabilities tests as set down by the full Federal Court (see [**Error! Reference source not found.**] - [110]).

126 Savitz, DA & Andrews, KW 1997, 'Review of epidemiologic evidence on benzene and lymphatic and hematopoietic cancers', *American Journal of Industrial Medicine*, vol. 31, no. 3, pp. 287-295. RMA ID 13051, Cited in February 2006 Submission to the Council at p. 21.

127 Metzger Law Group, *Benzene a human carcinogen*, accessed by the Applicant n.d. via http://www.toxictorts.com/tox _benzene.html. Cited in February 2006 Submission to the Council at pp. 22-23.

Among the disease that have been associated in the literature with benzene are…chronic lymphocytic leukemia (CLL)…

The greatest risk is to workers who use various petroleum solvents that contain benzene.

Epidemiologic studies have shown that workers who use solvents in different trades have increased risks of developing cancer and blood diseases from benzene.

e. PSC Resources n.d.[128]

Adult T cell leukemia is a type of CLL caused by a virus, Human T-Cell Leukemia/Lymphoma Virus-1 (HTLV-1)…The only known risk factor for other types of CLL is exposure to benzene.

f. US Environmental Protection Agency 1998[129]

Benzene is classified as a "known" human carcinogen (Category A) under the Risk Assessment guidelines of 1986.

Epidemiologic studies and case studies provide clear evidence of a casual association between exposure to benzene and acute non lymphocytic leukemia (ANLL) and also suggest evidence for chronic non lymphocytic leukemia (CNLL) and chronic lymphocytic leukemia (CLL).

g. Occupational Disease Panel 1997[130]

It came to our attention that in addition to the well established link between benzene exposure and leukemia, there is a growing body of evidence which supports a relationship between benzene exposure and lymphatic cancers.

Considerable evidence also exists to link benzene to…chronic lymphatic leukemia…

A probable connection between benzene and leukaemia and pre-leukemia cancer has been recognised in two other jurisdictions..

h. Linet, MS & Blattner, WA 1998[131]

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128 PSC Resources n.d. Public Health Assessment, , *Appendix B – General discussion on aetiology of selected cancer types – Leukaemia*, Massachusetts, accessed by the Applicant n.d. via http://www.atsdr.cdc.goc/HAC/PHA/psc/psc_p4.html.

129 US Environmental Protection Agency 1998, *Carcinogenic effects of benzene*, an update, accessed by the Applicant n.d. via http://www.epa.gov/iris/subst/0276.htm#evid, pp. 1-69.

130 Occupational Disease Panel, 1997, *Report to the Workers' Compensation Authority on ODP revisions to Schedule 3, phase 2, Chapter 5 Benzene and Leukaemia, Chapter 6 Summary of Panel's findings, and Chapter 7 The Panel's recommendations*, Toronto, Ontario, accessed by the Applicant n.d. via http://www.canoshweb.org/odp/html/rpt14a.htm. (includes 51 references)

The Applicant Mr FR included in his submission the 51 citations of medical science studies referenced in the Occupational Disease Panel, 1997 report.

Council notes that this US jurisdiction finding does not reflect the Council's two-step process in applying the reasonable hypothesis and balance of probabilities tests as set down by the full Federal Court (see [108] - [110]).

131 Linet, MS & Blattner, WA 1998, *The epidemiology of chronic lymphocytic leukemia*, in Polliack, A & Catovsky, D eds. *Chronic lymphocytic leukemia*, Harwood Academic publishers, New York, accessed by Applicant n.d. via http://www.aegis.com/pubs/nmap/509-lymphoma.html.

CLL has been linked to occupational exposure to benzene, xylene, coal-based solvents, carbon disulfide, and carbon tetrachloride.

Chemicals and solvents linked to leukemia: benzene, pesticides, arsenic, chromium, butadiene, dioxin, ethylene oxide, heptachlor, herbicides, hydrazine, mycotoxins, perchloroethylene, polychlorinated biphenyls (PCBs), toluene, tetrachloroethylene, trichloroethylene, trinitrotoluene

i. Remedy's Health.com 1999[132]

Agricultural chemicals, in particular, have been linked with an increased risk of chronic lymphocytic leukemia (CLL). In addition, some reports suggest that leukemia risk may be increased in workers exposed to dioxin, styrenes, butadienes, or ethylene oxides.

j. Glass, DC et al. 2003[133]

Matched analysis showed that the risk of leukemia…for the 13 case-sets with greater than 8ppm-years cumulative exposure, the odds ratio was 11.3 (95% confidence interval = 2.85-45.1).

The risks for acute non lymphocytic leukemia and chronic lymphocytic leukaemia were raised for the highest exposed workers.

k. Sapkota, A & Buckley, TJ n.d.[134]

Benzene has also been long established as a known carcinogen (references 9-11).

Exposure to benzene is associated with acute non lymphocytic leukemia and chronic lymphocytic leukaemia (references 12-19).

Known indoor sources of benzene and PAHs include cleaning products, paints, glues, and tobacco smoke for benzene (references 27-30)…

l. Penney, J 1995[135]

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132 Remedy's health.com communities 1999, *Leukemia Causes*, accessed by the Applicant n.d. via http://www.oncologychannel.com/leukemias/causes.shtml.

133 Glass, DC et al. 2003, 'Leukemia Risk Associated With Low-Level Benzene Exposure', *Epidemiology*, vol. 14, no. 5, pp 569-77. RMA ID 36950 & 30508.

134 Sapkota, A & Buckley, TJ n.d. The mobile source effect of curbside 1,3-Butadiene, benzene, and particle-bound polycyclic aromatic hydrocarbons assessed at a tollbooth, accessed by the Applicant n.d. via http://www.ecochem.biz/Library/sapkota.pdf

135 Penney, J 1995, *Report to the Occupational disease panel (Industrial Disease Standards Panel) on occupational exposure to benzene and leukaemia*, accessed by Applicant n.d. via http://www.canoshweb.org/odp/html/rp7.htm.

Kalf, 1987
Girard and Revolt 1970
Thorpe, 1974,
Aksoy and co-workers, 1974, 1976, 1977, 1985,
Infante et al and Rinsky et al. 1977, 1981, 1987,
Linos et al. 1980,
Rushton and Alderson 1981
Schottenfield et al (1981)
Decoufle et al 1983

This is a comprehensive report on “Occupational Exposure to Benzene and Leukaemia”, in general and includes comment on the medical science evidence in respect of, association, case reports, epidemiology, category of studies, and on the basis of various haematological disorders.

… approximately 1 % of benzene is absorbed from skin contact. However dermal absorption is enhanced and may approach 5% when skin is cracked, blistered or abraded…

Cigarette smoke, both mainstream and sidestream, is estimated to account for about 50% of non-occupational exposure.

Benzene also appears to have a cytotoxic effect on marrow cells of intermediate differentiation regardless of cell line, including promyelocytes, myelocytes, and basophilic erythroblasts. …Benzene metabolites hydroquinone, p-benzoquinone and phenol also decrease the ability of stromal cells to support formation of granulocytic/macrophage progenitors. (Kalf, 1987).

Girard and Revolt (1970) in a hospital-based case-control study…1966-1969. ...had evidence of previous exposure to benzene and toluene compared to five (4%) controls, for relative risks of…4.1 (1.4-12.0)…(It is assumed that haematologic effects are likely to be due to benzene present as a contaminant in toluene.)

Thorpe (1974), found 18 leukaemia cases among 38,000 active workers and pensioners from 8 European affiliates of a large U.S. oil company during the years 1962-1971. Workers who had quit before retirement were not included. Workers were classified as exposed (for five years minimum to products containing at least 1 percent benzene) or not (no or occasional exposure). The SMR for leukaemia in exposed workers was 121 (37-205) when compared to the general populations in the countries where the affiliates were located.

Aksoy and co-workers(1974, 1976, 1977, 1985)… 28,500 Turkish shoe workers exposed to benzene between 1967 and 1973. … Non-exposed cases had higher rates of acute lymphocytic, chronic myelocytic and chronic lymphocytic leukaemias. Aksoy updated this study in 1985, reporting a total of 51 cases of leukaemia.

Infante et al and Rinsky et al (1977, 1981, 1987), a series of studies and follow-ups have been carried out on workers employed in the manufacture of rubber hydrochloride (trade name Pliofilm) at three Ohio plants. …Fifteen deaths were observed from lymphatic and haematopoietic cancers versus 6.6 expected. Nine leukaemias were observed versus 2.7

Arp et al, Checkoway et al 1983, 1984
Wongsrichanalai 1989
Wong and Raabe (1995)
IARC, 1982;
OSHA, 1987;
Snyder and Kalf, 1994
Cox, 1991;
Eastmond, 1993
Aksoy, 1989;
Yin, 1989

expected for an SMR of 337 (154-641), in comparison to the general population of U.S. white men. … All leukaemias were myelocytic or monocytic.

Linos et al (1980), carried out a case-control study of 138 leukaemia cases and 276 controls. … A relative risk of 3.3 (0.6-28) was based on four exposed cases and three controls. Three of the exposed cases were identified as having chronic lymphocytic leukaemia.

Rushton and Alderson(1981), conducted a case-control mortality study of workers in 8 oil refineries in the United Kingdom… Exposures were classified, as low, medium or high based on work histories. The relative risk for medium or high exposures compared with low exposures was 2.0 (1.0-4.0). Leukaemia cases were identified as: 10 lymphatic, leukaemias (3 acute, 5 chronic, 2 unspecified); 15 myeloid leukaemias (6 acute, 4 chronic and 5 unspecified); and 5 others, including 4 acute monocytic and 1 "other" acute. Relative risks for different types of leukaemia were not presented.

Schottenfield et al (1981) in a preliminary study of the morbidity and mortality of U.S. petroleum workers…observed statistically significance increases in the incidence of acute and chronic lymphocytic leukaemias among refinery workers…Seven leukaemias were observed compared to 2.8 expected, for a standardized incidence ration (SIR) of 272.

Decoufle et al (1983) studied 259 males employed during 1947-1960 at a chemical plant where benzene was used in large quantities. Investigators found 4 deaths from lymphoreticular cancers, compared to 1.2 expected for an SMR of 364 (RR 3.7). Three deaths were due to leukaemia compared to 0.4 expected (RR 6.8), including one case of chronic lymphocytic…leukaemia.

Wong and colleagues(1980, 1983) conducted a mortality study of 4602 male chemical workers occupationally exposed to benzene at 7 plants for least 6 months between 1946 and 1975. … Wong found 7 deaths due to leukaemia in all exposed , workers compared to none in the non exposed group. Continuously exposed workers had an excess of lymphopoietic cancer. ...Wong found a significant dose-response relationship by cumulative exposure (not duration). Wong concluded that there was a significant association between occupational exposure to benzene and leukaemia, and all lymphopoietic cancers including non-Hodgkin's lymphoma.

Arp et al, Checkoway et al 1983, 1984, conducted one of several case-control studies, investigating the relationship between leukaemia and solvent exposures in the rubber industry… Relative risks for lymphocytic leukaemia were 4.5 for workers with primary exposure and 1.5 for workers with secondary exposure. Relative risks for workers exposed to other solvents were almost identical. Checkoway et al studied 11 of the lymphocytic cases (no distinction between primary and secondary exposure) and 1350 controls. Relative risk of lymphocytic leukaemia was 2.5 in benzene exposed workers, but was also elevated for workers exposed to other solvents. Most workers had exposures to several solvents.

Wongsrichanalai (1989) reported the mortality experience of 9484 white male petroleum refinery workers, during the period 1940 through 1984, comparing the number of deaths observed with those expected based on mortality rates among U.S. white men. The overall SMR was 77. A statistically significant increase in leukaemia deaths was observed (44 observed versus 29.6 expected) for an SMR of 149 (108-200). Six men with leukaemia mentioned on the death certificate, but not coded as the underlying code of death, were

not included in the analysis. An excess in all lymphatic and haematopoietic cancers was also seen, with an SMR of 126 (101-155). Evaluation of leukaemia mortality by cell type revealed excesses of lymphocytic, myelocytic and monocytic leukaemias.

Wong and Raabe (1995) carried out a meta-analysis of epidemiological cohort studies of petroleum workers in the U.S. and U.K. Data from 10 studies was incorporated, including the Rushton and Wongsrichanalai studies described above.

The meta-SMRs were calculated as follows:

Acute myelogenous 1.	0.93	(0.73 - 1.16)
Chronic myelogenous 1.	0.94	(0.65 - 1.31)
Acute lymphocytic 1.	1.32	(0.81 - 2.01)
Chronic lymphocytic 1.	0.87	(0.64 - 1.16)

Benzene exposures of petroleum workers in these studies were generally lower than levels in the Pliofilm plants or among Turkish shoemakers. The authors conclude that benzene can increase the risk of AML given a high enough exposure over an extended time, but that petroleum workers were not at risk because benzene exposure levels have been reduced below the threshold for leukaemogenesis, which they suggest occurs at 200 to 500 ppm-years of cumulative exposure.

The finding of significant increases in chromosomal aberrations in blood and bone marrow and in, lymphocytes from benzene-exposed symptomatic workers has been confirmed by several other investigations. (IARC, 1982; OSHA, 1987; Snyder and Kalf, 1994)

In the bone marrow, these metabolites are transformed to p-benzoquinone, o-benzoquinone and trans-trans-muconaldehyde (TTM). P-benzoquinone causes a variety of genotoxic, clastogenic, and cytotoxic effects, including inhibition of cell division, of DNA and RNA synthesis, growth of bone marrow stromal cells, induction of DNA breaks, micronuclei and sister chromatid exchanges, and binding to DNA and protein. (Cox, 1991; Eastmond, 1993)

Several cell populations of the blood-forming system are susceptible to the effects of benzene, metabolites, including pluripotential stem cells, haematopoietic stem cells, early progenitor cells at intermediate stages of differentiation in all cell lines (myeloid, lymphoid, and erythroid), and, cells of the bone marrow stromal microenvironment especially stromal macrophages. (Kalf, 1987; Snyder and Kalf, 1994). Erythroid cells appear to be particularly sensitive to benzene.

There is clear scientific agreement that benzene causes acute myelogenous types of leukaemia. Ironically, scientific unanimity on this issue may be functioning to impede resolution of a broader question, whether benzene causes a range of other types of leukaemia and lymphatic diseases with which it has been associated. For example, several researchers, looking to investigate the effects of low-level exposures to benzene in various industries, appear to have focused narrowly on the incidence of AML rather than incidence of all types of leukaemia, reinforcing the AML-benzene link, but making more difficult the assessment of linkage with other leukaemias and lymphatic disorders.

(Aksoy, 1989; Yin, 1989) In some populations of benzene-exposed workers there is a disproportionately high incidence of AML compared to other leukaemias, and disproportionately low incidence of other types.) Interestingly, neither of these investigators argues that benzene causes only AML. Aksoy reviewed several studies of benzene-exposed workers in which AML predominates, together with two studies in which

chronic types of leukaemia occur more frequently and suggests factors to explain the differences, Including: higher benzene content (and exposures) appear to correspond more to acute leukaemias; mixed solvent exposures may be more closely related to chronic leukaemias. (Aksoy, 1989)

The author concludes:

Benzene and its metabolites are toxins to almost all cells of the bone marrow.

Considerable evidence also exists to link benzene to chronic myelogenous leukaemia, chronic lymphatic leukaemia, multiple myeloma and myelofibrosis and myeloid metaplasia. A number of studies also report an association with acute lymphoblastic leukaemia, non-Hodgkin's lymphoma, Hodgkin's disease and thrombocythemia.

…there is certainly enough evidence, to sustain a "probable connection" for several of these disorders

m. Smith, MT & Zhang, L 1998[136]

Numerous studies have associated exposure to benzene with increased levels of chromosome aberrations in circulating lymphocytes of exposed workers. Increased levels of chromosome aberrations have, in turn, been correlated with a heightened risk of cancer, especially for hematologic malignancy

The Authors state that the established causes of Leukaemia are:

Heredity, radiation, chemical exposures, and treatment with chemotherapeutic agents have been implicated in the development of leukemia. Viral infection by at least one known virus, human $_{T}$_ cell leukemia/lymphotropic virus type I (HTLV-1), is a well-understood cause of adult T-cell leukemia (17).

Hayes and Yin (34,35)…have established that benzene causes AML and MDS in humans and have also suggested that benzene exposure may be associated with non-Hodgkin's lymphoma, lymphocytic leukemia, lung cancer, and nasopharyngeal cancer.

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136 Smith, MT Zhang, L 1998, 'Biomarkers of leukemia risk: benzene as a model', *Environ Health Perspect*. Vol. 106, Suppl. 4, pp. 937-946.

Franchini, G 1995, 'Molecular mechanisms of human T-cell leukemia/lymphotropic virus type I infection', *Blood*, vol. 86, pp. 3619-3639.

Hayes, RB et al. 1996, 'Mortality among benzene-exposed workers in China', *Environ Health Perspect*, vol. 104, pp. 13491352.

Yin, SY et al. 1996, 'A cohort study of cancer among benzene-exposed workers in China: overall results', *Am J Ind Med*, vol. 29, pp. 227-235.

Wallace, L 1990, 'Major sources of exposure to benzene and other volatile organic chemicals', *Risk Anal*, vol. 10, pp. 59-64.

Knox, EG 1994. 'Leukaemia clusters in childhood: geographical analysis in Britain', *J Epidemiol Community Health,* vol. 48, pp.369-376.

van Steensel-Moll, HA Valkenburg, HA and van Zanen, GE 1985, 'Childhood leukemia and parental occupation, A register-based case-control study', *Am J Epidemiol*, vol. 121, pp. 216-224.

Shu, XO et al. 1988, 'A population-based case-control study of childhood leukemia in Shanghai, Cancer, vol. 62, pp. 635-644.

Buckley, JD et al. 1989, 'Occupational exposures of parents of children with acute non lymphocytic, leukemia, a report from the Children's Cancer Study Group', *Cancer Res*, vol. 49, pp. 4030-4037.

McKinney, PA et al. 1991, 'Parental occupations of children with leukaemia west Cumbria, north Humberside, and Gateshead, *Br Med J*, vol. 302, pp. 681-687.

Another major source of public exposure to benzene is cigarette smoking. A pack-a-day smoker inhales approximately 2 mg/day, and non-smokers who live travel, or work with smokers are exposed to benzene through side-stream or second-hand smoke (44).

Knox (123) reported that cases of childhood leukemia commonly occur closer to industrial installations. He concluded: "The common patterns of close association of clustered and non clustered cases imply a common etiological component arising from a common environmental hazard--namely the use of fossil fuels, especially petroleum" (123). This work implicates benzene and petroleum products in the development of childhood leukemia…

The findings of Knox and co-workers are consistent with earlier reports from. Holland (127), China (128), the United States (129), Britain (130), and Japan (131) of an association between parental exposure to solvents containing benzene and increased risk of childhood leukemia.

51.3. The Applicant Mr FR included in his submission the 138 citations of medical science studies referenced in this article.

a. Unknown Author n.d.[137]

The following diseases are associated with benzene exposure:
Acute Myelogenous Leukemia
Acute Lymphatic Leukemia
Chronic Myelogenous Leukemia
Chronic Lymphatic Leukemia (Applicant added emphasis)
Hodgkin's Disease
Hairy Cell Leukemia

b. Unknown Author 2003[138]

[There] are studies performed by the University of North Carolina School of Public Health that conclude that benzene is linked to many forms of leukemia, including AML, Chronic Lymphocytic and Myeloid Leukemia.
Cigarette smoke is a significant source of benzene for those who smoke or are breathing in second hand smoke, particularly in the home".

Benzene exposure and Risks for chronic lymphocytic leukemia (CLL) tended to show elevations in nested case-control studies, with possible dose response relationships in at least two of the three studies. However, cohort studies on CLL show no such risks.

c. Khalade, A et al. 2010[139]

…we synthesized the existing epidemiologic evidence on the relation between occupational exposure to benzene and the risk of leukaemia, including all types combined and the four main sub-groups acute myeloid leukaemia (AML), acute lymphocytic leukaemia (ALL), chronic lymphocytic leukaemia (CLL), chronic myeloid leukaemia (CML).

[Conclusions] Our study provides consistent evidence that exposure to benzene at work increases the risk of leukaemia with a dose-response pattern.

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137 Unknown Author n.d. *Benzene and leukemia*, accessed by Applicant n.d. via http://www.benzene-leukemia-center.com/at_risk.htm.

138 Unknown Author 2003, *Part 2 – Chemicals in the workplace, horrors of benzene exposure – how to protect your health*, accessed by Applicant n.d. via www.fuelline.com/login/newsdtl.cfm?section=around_industry&eventdate=10%2F21%2F02.

139 Khalade, A et al. 2010, Exposure to benzene at work and the risk of leukemia: a systematic review and meta-analysis', *Environmental Health,* vol. 9, p. 31, accessed by the Applicant n.d. via http://www.ehjournal.net/content/9/1/31.

d. Metzger Law Group n.d.[140]

Many epidemiologic studies have been conducted of benzene-exposed workforces. While the strongest causal association found in these studies is for benzene and Acute Myelogenous Leukemia (AML), a causal association has also been found for Chronic Lymphocytic Leukemia (CLL) based upon several studies. (Applicant added emphasis)

Chinese Epidemiologic Study Group of Leukemia 1992

Notably, the greatest risk (a greater than 3-fold increase in leukemia) was found for those cases exposed to benzene who had CLL. Importantly, this finding was statistically significant to a 95% confidence interval.

Wongsrichanalai, C et al. 1989

…a study investigating mortality from leukemia at a refinery in Illinois. They found significant excesses of lymphocytic leukemia which declined over time: 1950 through 1959 (SMR = 505, 95% CI = 164 to 1180); 1960 through 1969 (SMR = 282, 95% CI = 103 to 615)

Rushton, L et al. 1997

…a case-control study of leukemia in the British petroleum industry. They reported a statistically significant excess of CLL in workers with cumulative benzene exposure between 0.26 and 0.50 ppmy (OR = 10.57; 95% CI 1.27 to 87.92), and an even larger excess of CLL in workers with cumulative benzene exposure between 0.60 and 1.64 ppmy (OR = 11.21; 95% CI 1.20 to 104.4).

Huebner, W et al. 1997

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140 Metzger Law Group, *Benzene and Chronic Lymphocytic Leukemia*, accessed by Applicant, n.d. via http://www.toxictorts.com/index.php/toxic-chemicals/benzene/benzene-and-chronic-lymphocytic-leukemia.

Chinese Epidemiologic Study Group of Leukemia 1992, [CHINESE] 'Risk factors analysis of leukemia and aplastic amenia in China', *Acta Academiae Mediicine Sinicae*, vol. 14, no. 2, pp. 185-189.

Wongsrichanalai, C et al. 1989, 'Mortality from Leukemia and Other Diseases Among Workers at a Petroleum Refinery', *J Occup Med*, vol. 31, no. 2, pp. 106-111.

Rushton, L et al. 1997, 'A Case-Control Study to Investigate the Risk of Leukaemia Associated with Exposure to Benzene in Petroleum Marketing and Distribution Workers in the United Kingdom', *Occup Envir Med*, vol. 54, pp. 152-166.

Huebner, W et al. 1997, 'Mortality Experience of a Young Petrochemical Industry Cohort: 1979-1992 Follow-Up Study of US-Based Employees', *J Occup Environ Med*, vol. 39, no. 10, pp. 970-982.

Lewis, R J et al. 2000, 'Mortality Among Three Refinery/Petrochemical Plant Cohorts: I. 1970 to 1982 Active/ Terminated Workers', *J Occup Environ Med*, vol. 42, no. 7, pp. 721-729.

Monash University, 2001, *Lympho-haematopoietic Cancer and Exposure to Benzene in the Australian Petroleum Industry* (June). (no further citation provided)

Falconer, E 1933, 'An Instance of Lymphatic Leukemia Following Benzol Poisoning', *Am J Med Sci*, vol. 186, pp. 353-361.

Tareeff, E. M 1963, 'Benzene Leukemias', *Acta Unio Int Contra Cancrum*, vol. 19, pp. 751-755.

Browning, E 1965, *Toxicity and Metabolism of Industrial Solvents*. (no further citation provided)

Goguel, A 1967, 'Les Leucemies Benzeniques De La Region Parisienne Entre 1950 et 1965 (Etude De 50 Observations)', [Benzene Leukemia in the Paris Area Between 1950 and 1965 (Study of 50 Cases)], *Nouv Rev Fr d'hemat*, vol. 7, no. 5, pp. 465-480.

Aksoy, M 1987, 'Chronic lymphoid leukaemia and hairy cell leukaemia due to chronic exposure to benzene: report of three cases', Brit J Haematol, vol. 66, pp. 209-211.

…a mortality study of Exxon petrochemical employees. They calculated a Standardized Mortality Ratio (SMR) of 1.81 for chronic lymphocytic leukemia, which was not quite statistically significant (95% CI, 0.90 to 3.24).

Lewis, RJ et al. 2000

…a further update of the Exxon petrochemical worker cohort was published. In this study, the same excess of CLL was found (SMR 181), but the excess was statistically significant (95% CI = 168 to 645).

Monash University 2001

…a case-control study of petroleum industry workers with leukemia. They found 11 CLL cases among 33 leukemia cases (33%), which constituted a greater than 7-fold significantly increased risk of CLL in workers with the highest benzene exposure. A dose-response relationship was also observed.

Falconer, E 1933

…reported the first case of CLL due to benzene exposure. Falconer, E., "An Instance of Lymphatic Leukemia Following Benzol Poisoning

Tarreeff, EM 1963

…described 16 Russian workers with benzene leukemia, of which 3 (18.7%) had CLL.

Browning, E 1965

…6 cases from the literature, comprising 9.8% of the cases collected.

Goguel, A 1967

…reported a series of benzene-leukemias in France of which 8 cases (18.2%) were CLL.

Askoy, M 1987

…described 3 of 58 workers with benzene-induced leukemia, of which two had CLL and one had HCL.

The author(s) conclude:

The above-described epidemiologic studies and case series together comprise a sufficient body of data to conclude that benzene causes Chronic Lymphocytic Leukemia (CLL).

51.4. Mr FR submits in respect of:

Carbon tetrachloride: I frequently used it as a universal solvent for cleaning work bench-tops, cleaning and degreasing motors, circuit cards, flux removal and grease films from the back of equipment racks, cable connectors and equipment.

Perchloroethylene/Tetrachloroethylene: I used this chemical to strip metalwork bare, prior to priming and repainting equipment cases. Again, no protective clothing or gloves were used, and one simply put up with the fumes. I also slept in a small compartment for 3 months with 2 others, who in their spare time used this solvent in a drum to dry clean uniforms.

Ethyl benzene: I frequently used this chemical as did other technicians, for heavy duty cleaning. As was standard practice of the day, no protective clothing, goggles or mask were

used, as these items were just not available. The item was placed in a bath of ethyl benzene and scrubbed with a paint brush and bare hands.

Trichloroethane: I regularly used this common all purpose cleaner, degreaser and solvent with bare hands, paint brush and rag, on equipment repairs in the workshops, on aircraft electronic bays during regular maintenance checks, and throughout my enlistment period.

Diesel exhaust fumes: I worked in an environment that was frequently filled with diesel exhaust fumes that emanated from the hydraulic test rigs used to operate and test an aircraft's landing gear… On board ship, this operation was performed in the hangar where the air would be blue from exhaust fumes. I frequently worked in the hangar on aircraft under these conditions, and breathed exhaust or other fumes in the mess if work was performed adjacent to external air intakes.

Trichloroethylene: I regularly used this common all purpose cleaner and degreaser with bare hands, paint brush and rag, on module repairs, in aircraft electronic bays during regular maintenance, and throughout my enlistment period.

a. Blair, A et al. n.d.[141]

Chronic lymphocytic leukemia (CLL) and non-Hodgkin's lymphoma (NHL) are two lymphatic and hematopoietic cancers… Since, they are both largely, B-cell malignancies, considering them together may provide etiologic clues.

An ecologic study of leukemia in New Jersey (Fagliano et al. 1990) found a correlation between leukemia incidence rates among females and the level of volatile organic compounds (trichloroethylene and related solvents) in public water supplies.

A high incidence of leukaemia was observed among children exposed inutero to private wells contaminated with solvents including trichloroethylene and perchloroethylene (Lagakos et al., 1986) and exposure was also linked to immunologic abnormalities in family members of the affected children (Byers et al., 1988).

A population-based case-control study in Massachusetts found an elevated risk of leukemia with exposure to tetrachloroethylene-contaminated drinking water (Aschengrau et al., 1993).

The Applicant Mr FR included in his submission the 69 citations of medical science studies referenced in this article.

51.5. Aircraft fuel (AVGas) and associated chemicals (MEK, sealants)[142] – Mr FR submits;

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141 Blair, A et al. n.d. *Occupational and environmental risk for chronic lymphocytic leukemia and non-Hodgkin's lymphoma*, (no further citation provided by Applicant).

Fagliano, J et al. 1990, 'Drinking water contamination and the incidence of leukemia, An ecologic study', *Am J Public Health*, vol. 80, pp. 1209-1212.

Lagokos, SW Wessen, BJ and Zelen, M, 1986, 'An analysis of contaminated well water and health effects in Woburn, Massachusetts', J Am Stat Assoc, vol. 81, pp. 583-596.

Byers, VS et al. 1988, 'Association between clinical symptoms and lymphocyte abnormalities in a population with chronic domestic exposure to industrial solvent contaminated domestic water supply and a high incidence of leukemia', Cancer Immunol Immunother, vol. 27, pp. 77-81.

Aschengrau, A et al. 1993, 'Cancer risk and tetrachloroethylene-contaminated drinking water in Massachusetts', *Arch Env Health*, vol. 48, pp. 284-292.

142 Mr FR provided electronic copy of oral submission, Oral hearing of 31 March 2011.

Aviation Fuels: it was common to see the fuel truck coming alongside and upwind from the aircraft to refuel it…The fuel used on the Tracker was AVGas, a volatile fuel with an octane rating of 130…I was able to assist with the cleaning up of small fuel spills using a rag. Because rubbish bins are banned from the flight line, the fuel soaked rag would be stuffed in an overalls pocket for later disposal. Fuel fumes would soon surround one's workspace, and not being unpleasant, were soon forgotten.

In addition to these fumes was the strong smell of aircraft exhausts when aircraft were running up on the flight line before take-off. These exhaust fumes also contain toxic gasses. I was also exposed to jet and helicopter fuels that contained toxic additives…

51.6. Herbicides and Pesticides[143] Mr FR submits;

a. Institute of Medicine (IOM), Veterans and Agent Orange: Update 2002[144], National Academy of Sciences (NAS) The report concluded that:

> However, a recent re-examination of the evidence revealed sufficient evidence of an association between exposure to herbicides sprayed during the Vietnam War and the risk for development of a specific form of leukemia - chronic lymphocytic leukemia (CLL) - in veterans.
>
> The IOM has stated that there is now "sufficient evidence of an association" between herbicides used in Vietnam and chronic lymphocytic leukemia

This is the highest category of association that the IOM-NAS has used in its Agent Orange reports. Mr FR contends that the IOM-NAS medical scientific report is sufficient evidence of herbicides such as Agent Orange to be an accepted factor for CLL.

– Mr FR notes[145] the American Veteran's Affairs Secretary Principi extended benefits to veterans as:

> …compelling evidence has emerged within the scientific community that exposure to herbicides such as Agent Orange is associated with CLL'.

Mr FR highlighted that:

> More than 9,000 potentially relevant studies were identified in the searches, and more than 1,000 were reviewed.

b. Powell, UR 2002[146]

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143 Ibid.

144 Institute of Medicine 2003, *Veterans and Agent Orange: update 2002*, 'Committee to review the health effects in Vietnam veterans of exposure to herbicides (fourth biennial update)', The National Academic Press, Washington DC. RMA ID 28337 and 29493.

145 American Veteran's Affairs, n.d. website accessed by Applicant n.d. via http:///www1.va.gov/agentorange/docs/IOMIDENTIFIESLEAKWCHRONICLYMLEUKEMIA.docs.

146 Powell, UR 2002, *Appeal decision citation no. 0206819*, Board of Veterans' Appeals, New York.

Council notes that this US jurisdiction finding does not reflect the Council's two-step process in applying the reasonable hypothesis and balance of probabilities tests as set down by the full Federal Court (see [**Error! Reference source not found.**] - [**Error! Reference source not found.**]).

Entitlement to service connection for chronic lymphocytic leukemia / small lymphocytic lymphoma as a result of exposure to herbicides is granted.

c. Muller, J 2002[147]

This study clearly demonstrates that distillation of water with hydrophobic chemicals such as dioxin-like chemicals of organochlorine pesticides results in contamination of the distilled water. The extent of co-distillation largely depends on the physico-chemical properties of the compounds of interest. 2,3,7,8-tetrachlorodibenzodioxin, the primary contaminant in Agent Orange, the defoliant used in the Vietnam conflict, co-distils relatively rapidly and thus will be enriched in the distilled water.

51.7. Tobacco[148] (by which the Council understands the Applicant to mean tobacco smoking and passive smoking)

Environmental Tobacco Smoke: My instructors encouraged me to smoke in 1958 as I kept falling asleep in class, and I became a medium to heavy smoker, giving it up when diagnosed with CLL in 1986… the Melbourne had no air conditioning and relied on forced and recirculated air for its internal ventilation. As a result, the atmosphere in the mess was **thick** with cigarette smoke every night… I lived and slept in these smoky conditions for months at a time.

a. Unknown Author n.d.[149]

Cigarette smoking is a known lifestyle-related risk factor for leukemia. Potential leukemia-causing chemicals in tobacco smoke include benzene, polonium-210, and polycyclic aromatic hydrocarbons (PAHs).

When cigarette smoke is inhaled, polonium-210 and lead-2l0 affect not only the lungs but many other sites including various organs (bladder, cervix, pancreas, prostate) and, lymph nodes.

51.8. Asbestos[150];

[HMAS Melbourne] The ship was literally plastered with asbestos, used as lagging around pipes, wall and ceiling insulation, and to reduce condensation effects on steel surfaces in living areas….I was tasked with another person to dismantle the asbestos fire curtain….The asbestos and other dust that came from this curtain was unbelievable, and being in the tropics, stuck to our sweaty bodies so that we itched and scratched until able to shower and change clothes.

147 Muller, J 2002, *Examination of the Potential Exposure of Royal Australian Navy (RAN) Personnel to Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans via Drinking Water*, The National Research Centre for Environmental Toxicology (ENTOX)[NRCET], A Report to the Department of Veteran Affairs, Australia. RMA ID 27791

148 Ibid.

149 Unknown Author n.d. *Etiology and Cigarette smoking*, Accessed by Applicant n.d. via http://www.meddean.luc.edu/lumen/MedEd/elective/pulmonary/lungca.htm.

150 Ibid.

> In early 1962, I was tasked to assist others in removing asbestos from an area …HMAS Melbourne. This job took several days, and was very hot, dusty and messy as the ventilation was turned off to minimise spreading asbestos dust.
>
> Another source of asbestos exposure was 'anti-flash' protective clothing we wore during action stations or exercises. These garments were asbestos impregnated cotton gloves reaching one's elbow, and a hood covering head and shoulders, leaving only a small opening for the eyes.

a. Schwartz, DA et al.[151];

> Exposure to asbestos was assessed by classifying each job held by a subject into one of four categories, based on the estimated intensity of exposure to asbestos.
>
> Evidence was found for a modest increasing risk of CLL with increasing asbestos exposure…the risk for CLL was 1.1 (95% CI = 0.8-1.6) for low, 1.2 (CI = 0.8-1.8) for medium, and 1.4 (CI = 0.8-2.3) for high peak asbestos exposure (p value for trend = 0.13).

b. Becker, N Berger, J and Bolm-Audorff, U 2001[152]

> …we describe the results of the six cohort and 16 case-control studies…A causal relationship between asbestos exposure and the subsequent development of lymphomas can not be derived from the available results…quite a number of the studies and the combined analysis indicate a (weakly) increased risk…

c. Cancer Consultants n.d.[153]

> An increased incidence of CLL has been linked to chronic exposure to hair dye, asbestos and possibly brick mortar. The incidence of CLL appears to be increased in agricultural workers, which suggests that sustained exposure to agricultural chemicals or exposure to animal transmitted diseases may increase the incidence of CLL….Swedish Researchers have documented an increased incidence of CLL in railway workers, which suggests that exposure to low frequency magnetic fields could be a cause of CLL.

Applicant's comment on the Scope of Review and Pool of Information

52. The Applicant submitted[154] that:

> I support and applaud the Council's proposed preliminary scope of review and believe that my referenced articles will illustrate the necessity for these factors to be included in the SOP for CLL.

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151 Schwartz, DA et al. 1988, 'B cell neoplasms and occupational asbestos exposure', *American Journal of Industrial Medicine*, vol 14, pp. 661-71. RMA ID 21056.

152 Becker, N Berger, J and Bolm-Audorff, U 2001, 'Asbestos exposure and malignant lymphomas-a review of the epidemiological literature', *Int Arch Occup Environ Health,* vol. 74, no. 7, pp. 459-69. RMA ID 26350.

153 Cancer Consultants, *Leukemia Cancer treatment & prevention*, accessed by the Applicant n.d. via http://patient.concerconsultants.com/leukemia_cancer_treatment.aspx?id+779.

154 23 March 2011 Applicant letter and confirmation of references included in his submissions to the RMA and SMRC.

53. The Applicant further requested that the Council consider some materials (listed in **Appendix D**) that he provided to the Council, which were not available to (not before) the RMA[155] (which the Applicant contended were in existence at the relevant times, and so could have been accessed by the RMA). The Council noted the Applicant's submission about these materials, but was unable to take them into account for the purposes of the review because they were not available to (not before) the RMA at the relevant times.

a. Rossie – Blue Water Navy Vietnam Veterans Association [LETTER] –see footnote 18

b. IOM Update 2008, 2009 – see [45]

c. Third Australian Vietnam Veterans' Study, 2005 – see [45]

d. Hugo, V 2008, *Men become accustomed to poison by degrees,* unpublished report, Vietnam Veterans Federation, ACT Branch, Page, Australia. – see [45].

e. Pirogova, E Vojisavljevic, V Cosic, L 2008, *Non-thermal effects of 500MHz – 900Mhz microwave radiation on enzyme kinetics*, Engineering in Medicine and Biology Society, 30th Annual International Conference of the IEEE. See [45].

Mr AR

54. In his applications of May 2005 and April 2008, the Applicant stated that his grounds for review were as follows:

> I believe in determining the above SOP the RMA did not take into account the findings of the U.S. Department of Veterans Affairs into the association between Agent Orange and CLL. Their report "Veterans and Agent Orange: Update 2002" published 23 January 2003 concludes that "there is sufficient evidence of exposure to herbicides sprayed during the Vietnam War and the risk of development of CLL". This report is available on the web and also in hard copy. The NRCET report commissioned by the Commonwealth Government, clearly showed that the Naval personnel were exposed to high levels of dioxins through the distillation of seawater.

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155 This information was in addition to the provision of primary and secondary sourced citations, abstracts, and links to web (www) pages / information, provided by the Applicant Mr FR in his written submissions to the Council (and the RMA – in 2000 and 2004), and identified by the Council as new information in footnotes of these REASONS.

The applicant Mr AR provided copies of an abstract of the Veterans and Agent Orange: Update 2002 report[156] and the Executive Summary of the NRCET report[157].

55. The Applicant Mr AR made a written submission dated 5 December 2005, which was taken into account by the Council. As mentioned above, the Applicant also made a comprehensive oral submission complementing his written submissions[158].

56. Mr AR supported his contentions on the basis of interpretations of the following articles. [159]

a. NRCET report:

> …unequivocally indicates that RAN personnel were exposed to high levels of dioxins through the distillation of estuarine seawater off the coast of Vietnam.
>
> …this report was commissioned by the Federal Government following The Vietnam Veterans Mortality study that showed the highest level of mortality among the forces in Vietnam occurred in RAN personnel…

b. IOM 2002 VAO report:

> …states that "there is sufficient evidence of an association between herbicide exposure (2,4-dichloro-penoxyacetic acid, 2,4,5, trichlorophenoxyacetic acid and its contaminant TCDD: cacodylic acid and picloram) and CLL.
>
> As a result, the Secretary of the Department of Veterans Affairs (U.S.) stated that veterans with CLL would get a disability pension of about $2,300 per month. This report was reviewed by officials and scientists both within and outside the DVA.

c. WHO classification 98234/3

> …[WHO] now considers SLL and CLL to be the same disease.

57. Mr AR contended that the statements of Principles for other disease contain a factor for 'being in Vietnam' and ingesting potable water and that his contention was that his CLL was caused by TCDD's, which he was exposed to in RAN service in Vietnam.

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156 Institute of Medicine 2003, Veterans *and Agent Orange Update 2002*, Committee to review the health effects in Vietnam veterans of exposure to herbicides (fourth biennial update), National Academy Press, Washington, D.C.

157 Muller, J 2002, The National Research Centre for Environmental Toxicology ENTOX, NRCET 2002, *Examination of the Potential Exposure of Royal Australian Navy (RAN) Personnel to Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans via Drinking Water*, A Report to the Department of Veteran Affairs, Australia.

158 The discussion of the Applicants submissions set out in these Reasons is derived from the written submissions and the complementary oral submission here identified.

159 5 December 2005 submission at p. 1&2.

> …you look at the SOPs for Hodgkin's lymphoma, non-Hodgkin's lymphoma, myeloma…and they all contain a factor of being in Vietnam and being subject to ingesting potable water that was distilled from the waters there[160].

58. Mr AR contended[161] that:

> …if you look at the factors for Non-Hodgkin's Lymphoma, including the service in Vietnam, and you consider that CLL and SLL are the same, either we should take the factors from 28-2010 and put them into SOPs 9 and 10 or include CLL under the SOP 28-2010. I see it very simple as that.

59. In conclusion Mr AR concludes that:

> …it appears that the RMA has been intent on trying to prove little evidence of causation of CLL by TCDDs, rather than acknowledging the body of evidence that links them.[162]

Applicant's comment on the Scope of Review and Pool of Information

60. Mr AR contended[163] that the scope of the review should be extended to review Statements of Principles dealing with the same family of diseases.

> For instance these include:
>
> 28/2004 - Hodgkin's lymphoma,
>
> 28/2010 - Non-Hodgkin's lymphoma
>
> 55/2003 – Myeloma

Mr AR submitted that as the definition of Non-Hodgkin's lymphoma included Small Lymphocytic Leukaemia (SLL), that the factors for NHL (applying also to SLL) may apply also to CLL.

> CLL is closely related to small lymphocytic lymphoma (SLL): in fact many oncologists consider them the same disease.
>
> …I believe a comparison of the SOPs is essential…

Further, that the Council include in its review that[164]:

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160 Oral hearing of 31 March 2011, transcript at p.30.

161 Oral hearing of 31 March 2011, transcript at p.31.

162 5 December 2005 submission at p. 2.

163 March 2011 comment on the Scope of Review.

CLL is caused by exposure to TCDDs

The exposure occurred by ingestion of distilled water in Vietnam increasing the levels of dioxin…[which] has now been accepted in the following SOPs:

28/2004, 28/2010, 55/2003 28/2005 with the inclusion of the following factor

(r) being:

(i) on land in Vietnam, or
(ii) at sea in Vietnamese waters, or
(iii) on board a vessel and consuming potable water supplied on that vessel, when the water supply had been produced by evaporative distillation of estuarine Vietnamese waters,
for a cumulative period of at least 30 days, at least five years

before the clinical onset of non-Hodgkin's lymphoma; or

ELIGIBLE PERSONS SUBMISSIONS

Mrs F

61. An eligible person Mrs F made a written submission sent to the Council by an email dated 16 December 2005. The written submission included a copy of the Bowen report[165]. The Council advised Mrs F that she could send the Bowen report to the RMA asking the RMA to undertake an investigation on the basis of the Bowen report and any other new information that the RMA may identify. The Council also advised that as the report was not information which was available to (before) the RMA, it could only consider the Bowen report as new information.

62. Mrs F made a number of further written submissions, all of which were taken into account by the Council. These comprised a letter dated 4 July 2006, attaching information[166] provided by a DVA AAT Advocate, copies of her DVA compensation claim decision[167] dated 17 June 2009, written submission prepared on or about 10

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164 Presiding Councillor Professor Joske stated at the Hearing of oral submissions on 31 March 2011 that Council only had jurisdiction to make a review decision on the contents of Statements of Principles 9 and 10 of 2005, concerning chronic lymphoid leukaemia, transcript at p. 31.

165 The Bowen report - Brown, RH Minnett, HC and White, FWG 1992, 'Edward George Bowen 1911-1991', *Historical Records of Australian Science*, vol. 9, no. 2. In Biographical Memoirs of Deceased Fellows, Australian Academy of Science, accessed on 19/12/2005 at http://www.asap.unmelb.edu/bsparcs/aasmemoirs/bowen.htm, pp. 1-18.

This report was not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

166 Department of Veterans Affairs (DVA) AAT Advocate information – Respondents statement of facts and contentions, dated 29 June 2006.

These papers were not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

167 Department of Veterans Affairs (DVA) delegate of the Repatriation Commission decision dated 17 June 2009, with attached reasons for decision, a medical treatment document, a military service

November 2005 sent to the Council by email on 14 December 2005 and a written submission prepared on or about 23 February 2006, respectively. As mentioned above, the Applicant also made a comprehensive oral submission complementing his written submissions[168].

63. Mrs F contended that exposure to the early microwave aircraft radar as was with ground based radar the subject of research by E.D. Bowen and others commencing in 1935-1936. Mrs F supported her contentions on the basis of interpretations of the following articles.

a. Edward George Bowen 1911-1991 (see fn 165)

The research group that E.D. Bowen was part of introduced forward-looking Air to Surface Vessels (ASV) radar adopted by the RAF in 1938-39.

> …wavelength to 1.5m…the standard wavelength for metre-wave airborne radar. (at p. 4 of 18)
>
> …the power from the aircraft transmitter was radiated in a wide beam forward, and the returns from the target were received on two simple antennas mounted on either side of the aircraft to give overlapping beams in the forward direction. The receiver was connected in rapid sequence to these two antennas by a fast rotating switch, and the signals were displayed as deflections to the right and left of a vertical time base on a single cathode-ray tube. (at p. 5 of 18)
>
> The first installation of ASV Mk I was made in a Hudson aircraft in December 1939. (at p. 5 of 18)
>
> [The ASV Mk II]…in the UK alone, 6,000 sets were made, and many thousands were produced in the USA and Canada. It was fitted to patrol and reconnaissance aircraft all over the world and used in anti-submarine patrols with about 110 aircraft fitted [1941] with ASV Mk II… (at p. 6 of 18)
>
> AI (Air Inception)…the first AI would measure the relative direction of the target by four antennas mounted on the fighter; two 'azimuth antennas' would give overlapping lobes in the azimuth plane and two 'elevation antennas' would give overlapping lobes in elevation… (at p. 6 of 18)
>
> The final engineering of AI Mk III was undertaken at the RAE [Royal Aircraft Establishment] and introduced into service as AI Mk IV in Blenheim and Beaufighters in the autumn of 1940.
>
> …the US made an important decision that the development of metre-wavelength radar should be the responsibility of the Armed Services, and that the development at. centre metre wavelengths should be the responsibility of a special Microwave Committee. (at p. 8 of 18)

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testimonial and an annotated copy of the Bowen report submitted in support of her DVA compensation claim by Mrs F.

168 The discussion of the Applicants submissions set out in these Reasons is derived from the written submissions and the complementary oral submission here identified.

…the Microwave Committee set up a special laboratory, the Radiation Laboratory at MIT… So successful was the programme at the Radiation Laboratory that the first experimental airborne 10cm radar was tested in a Douglas B18 …on 27 March 1941.

b. Szmigielski, S 1996 [169] (at PubMed Abstract)

Cancer morbidity…[study]…military career personnel in Poland during a period of 15 years (1971-1985). …The population size varied…with a mean count of 128,000 persons each year; each year about 3,700 of them (2.98%) were considered as occupationally exposed to RF/MW.

The cancer morbidity rate for RF/MW-exposed personnel for all age groups (20-59 years) reached 119.1 per 100,000 annually (57.6 in non-exposed) with an OER of 2.07, significant at $P < 0.05$.

…malignancies of the haematopoietic system and lymphatic organs (OER = 6.31). Among malignancies of the haematopoietic / lymphatic systems, the largest differences in morbidity rates between exposed and non-exposed personnel was found for chronic myelocytic leukaemia (OER = 13.9), acute myeloblastic leukaemia (OER = 8.62) and non-Hodgkin lymphomas (OER = 5.82).

The high incidence of cancer of the hemato-lymphatic organs follows the break down given in the lower half of the diagram. [Figure 14 at p. 61] LGR: malignant lymphogranulamatosis; LS, LM, lymphosarcomas and lymphomas; CLL, chronic lymphatic leukemia; ALL, acute lymphoblastic leukemia; CML, chronic myelocytic leukemia; AML, acute myeloblastic leukemia and PL, plasmocytoma (plasma cell leukemia).

This data shows that the microwave exposed group…had increased malignancies in every category organ, significantly increased in…colo-rectum, skin cancer…highly significant in blood and lymph organs. (at p. 61)

This is the largest of any study carried out to this time that shows that RF/MW exposure increased the cancer in every organ in the body, but some organs are more reactive than others, especially the blood and lymph organs. This is consistent with melatonin reduction, a calcium ion efflux mechanism and suppression of the immune system.

The data is summarised in three tables, in parallel with the summary diagrams of Szmigielski, S et al 1988 above, morbidity of body organs, haematopoietic malignancies and age grouped relationships. (at p. 62)

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169 Szmigielski, S 1996, 'Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation', *The Science of the Total Environment,* vol 180, pp. 9-17.

Council notes that MrsF submitted pages with tables (reproduced above from Szmigielski, S 1996, but that the text of the submitted pages appeared to come from another unknown document, which as well as citing Szmigielski, S 1996, also cites Szmigielski, S et al 1998.

Szmigielski, S et al 1998, *Immunological and cancer-related aspects of exposure to low-level microwave and radiofrequency fields*, in Marino, AA ed. 1988, *Modern Bioelectricity*, Marcel Dekker, New York, pp. 861-925.

Szmigielski, S et al 1998 - This report was not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

Table 1 (at p. 12)
Incidence of neoplasms (per 100000 subjects annually) in military personnel exposed and non-exposed (control) to radiation frequency and microwave radiation

Localization of malignancies	Incidence per 100000 annually non-exposed (expected)	Incidence per 100000 annually exposed (observed)	Exposed/non-exposed ratio (observed/ expected)	95% confidence limits	Significance (P-value)
Oral cavity	2.65	1.82	0.71	0.42-1.32	N.S.
larynx	1.96	2.12	1.08	0.82-1.24	N.S.
Oesophageal and stomach	4.83	15.64	3.24	1.85-5.06	< 0.01
Colorectal	3.96	12.65	3.19	1.54-6.18	< 0.01
Liver, pancreas	2.43	3.58	1.47	0.76-3.02	N.S.
Laryngeal, lung	21.89	23.26	1.06	0.72-1.56	N.S.
Bones	1.53	1.03	0.67	0.36-1.42	N.S.
Skin, including melanomas	3.28	5.46	1.67	0.92-4.13	< 0.05
Kidney and prostatic	4.58	3.96	0.86	0.54-1.67	N.S.
Nervous system, including Tumours Thyroid	2.28	4.36	1.91	1.08-3.47	< 0.05
	1.38	2.12	1.54	0.82-2.59	N.S.
Haematopoietic system and lymphatic organs	6.83	43.12	6.31	3.12-14.32	< 0.001
All malignancies	57.60	119.12	2.07	1.12-3.58	< 0.05

Data are based on registration of all newly diagnosed cases of neoplasms during 1971-1985 in the whole population (aged 20-59 years) of military career personnel in Poland. Exposed, subjects exposed occupationally to radiofrequency and microwave radiation; Non-exposed, subjects non-exposed to the radiation, used as control group for calculation of expected morbidity for the exposed group.

In the 1988 data analysis, three sub-categories of leukaemia and lymphoma were significantly increased and 4 are very highly increased, Lymphoma, Chronic myelocytic leukaemia, Acute Myeloblastic leukaemia and Total leukaemia/Lymphoma. (At p. 62)

Table 2
Incidence of haematopoietic and lymphatic malignancies (per 100000 subjects annually) in military personnel exposed and non-exposed (control) to radiofrequency and microwave radiation

Type of haematopoietic/ lymphatic malignancy	Incidence per 100 000 annually non-exposed (expected)	Incidence per 100 000 annually exposed (observed)	Exposed/non-exposed ratio (observed/ expected)	95% confidence limits	Significance (P-value)
Malignant lymphogranulomatosis (Hodgkin's disease)	1.73	5.12	2.96	1.32-4.37	:0.05
Lymphoma (non-Hodgkin) and lymphosarcoma	1.82	10.65	5.82	2.11--9.74	< 0.001
Chronic lymphocytic leukaemia	1.37	5.04	3.68	1.45-5.18	<0.01
Acute lymphoblastic leukaemia	0.32	1.84	5.75	1.22-18.16	< 0.05
Chronic myelocytic leukaemia	0.88	12.23	13.90	6.72-22.12	<0.001
Acute myeloblastic leukaemia	0.71	6.12	8.62	3.54-13.67	< 0.001
Myeloma (plasmocytoma)	No cases (0.00)	2.12	ND	ND	
Total	6.83	43.12	6.31	3.12-14.32	< 0.001

Data are based on registration of all newly diagnosed cases of malignancies of the haematopoietic system and lymphatic organs during 1971-1985 in the whole population (aged 20-59 years) of military career personnel in Poland. Subjects exposed occupationally to radiofrequency and microwave radiation; Non-exposed, subjects non-exposed to the radiation, u d as control group for calculation of expected morbidity for the exposed group; ND, not determined.

The Haematopoietic/lymphatic cancers are all highly significantly increased in every age group.

Table 3
Incidence of neoplasms (per 100000 subjects annually) in age groups of military personnel exposed and non-exposed (control) to radiofrequency and microwave radiation

Age group (years)	All forms/localizations of neoplasms					Haematopoietic/lymphatic malignancies				
	Incidence per 100000 subjects annually non-exposed expected	Incidence per 100000 subjects annually exposed observed	Exposed/non-exposed ratio (observed/ expected)	95% confidence limits	Signi-ficance P-value	Incidence per 100000 Subjects Annually Non-Exposed expected	Incidence Per 1000000 Subjects Annually Exposed observed	Exposed/non-exposed ratio (observed/ expected)	95% confidence limits I	Significance (P-value)
20-29	11.62	27.11	2.33	1.23-3.12	< 0.05	2.12	17.30	8.16	3.11-22.64	<0.01
30-39	18.37	42.28	2.30	1.04-3.06	< 0.05	3.08	26.43	8.58	3.46-19,58	<0.01
40-49	84.29	161.62	1.92	0.98-2.84	< 0.05	8.32	73.25	8.80	4.13-15,27	<0.01
50-59	186.71	274.13	1.47	0.92-2.12	N.S.	24.31	108.62	4.47	2.56-6.81	<0.01
All age	57.60	119.12	2.07	1.12-3.58	< 0.05	6.83	43.12	6.31	3.12-14.31	<0.001

Data are based on registration of all newly diagnosed cases of neoplasms during 1971-1985 in the whole population (aged 20-59 years) of military career personnel in Poland. Exposed, subjects exposed occupationally to radio frequency and microwave radiation; Non-exposed, subjects non-exposed to the radiation, used as control group for calculation of expected morbidity for the exposed group.

c. Sage Associates [170]

Mrs F highlighted the following aspects of a larger Table:

Studies by Increasing Power Density

Power Density	Reported Biological Effects	References
1.3-5.7 μW/cm^2	Two-fold increase in childhood leukemia from AM-FM exposure	Dolk, 1997
10 μW/cm^2	Significant differences in visual reaction time and reduced memory function	Chiang, 1989
30 μW/cm^2 (0.015 W/Kg SAR)	Immune system effects – elevation of PFC count (antibody producing cells)	Veyret, 1991
100 μW/cm^2	Changes in immune system function	Elekes, 1996

[170] Sage, C of Sage Associates *Studies Matrix*, accessed by MrsF at 06/04/2006 via http://www.wave-guide.org/library/studies.html , pp. 1-4.

Studies by Increasing Specific Absorption Rate

SAR	Reported Biological Effects	References
0.0317 W/Kg	Decrease in eating and drinking	Ray & Behari, 1990
0.26 W/Kg	Harmful effects to the eyes/certain drugs can sensitize eyes to RFR	Kues, 1992
2-3 W/Kg	Cancer acceleration in skin and breast tumors at 50-75% of standard	Szmigielski, 1982

Listing of Full Citations Referenced above

Study	Description
Mann, K et al. 1996	'Effects of pulsed high-frequency electromagnetic fields on human sleep', *Neuropsychology*, 1996, vol. 33, pp. 44-47.
Szmigielski, S et al. 1982 (sic - 1996)	'Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation', *Sci Total Environ*, vol. 180, pp. 9-17.

d. Goldsmith, JR 1997 171

> Even if cancer risks cannot yet be unequivocally demonstrated, some measure of protection should be taken as early as possible because it may take some time to determine the definitive relationship between RF and cancer. (at p. 1579)

Mrs F highlighted the possible effects of radiofrequency as identified in evidence up to 1994 and reviewed in this study, which she submitted were the symptoms of CLL:

> …changes in blood counts…headache, ocular problems, fatigue, dizziness, memory impairment, and sleep difficulties… (at p. 1580)

Mrs F also highlighted Goldsmith's comments in respect of other studies[172] he reviewed:

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171 Goldsmith, JR 1997, 'Epidemiologic evidence relevant to radar (Microwave) effects,' Environ Health Perspectives, vol. 105, suppl. 6, pp. 1579-1587.

172 (19) Daily, LE 1943, 'A clinical study of the results of exposure of laboratory personnel to radar and high frequency radio', *U.S. Naval Medical Bulletin*, no. 41, pp. 1052-1056. Cited in Steneck, NH et al. 1980, 'Origins of U.S. safety standards for microwave radiation', *Science*, vol. 208, pp. 123-127.

(24) Goldoni, J 1990, 'Hematological changes in peripheral blood of workers occupationally exposed to microwave radiation', *Health Phys*, vol. 58, pp. 205-207.

(25) Goldoni, J 1990, Unpublished data.

(26) Tonascia, JA & Tonascia, S Unpublished data.

(18) Jacobson, G Unpublished data.

(29) Garaj-Vrohac, V Fucic, A and Pevalek-Kozlina, B 1993, 'The rate of elimination of chromosomal aberrations after accidental exposure to microwaves', *Bioelectrochem Bioenerg*, vol. 30, pp. 319-325.

(32) Garaj-Vhrovac, V Horvat, D and Koren, Z 1990, 'The effect of microwave radiation on the cell genome', *Mutat Res*, vol. 243, pp. 87-93.

When radar was first identified as a health risk, Daily (19) reported a statistically significant increase in immature red blood cells among workers exposed to radar. (at p. 1580)

Goldini (24,25) compared the haematological findings in 25 male air traffic control technicians exposed to radar… The radar was in the range of 1250 to 1350 MHz, with a strength varying from 10 to 20 μW. Radar-exposed workers had significantly lower levels of leukocytes and red blood cells than workers distant from the microwave source. (at p. 1580-1581)

The initial examination of Moscow embassy workers…was done to study the possible effects of radiation on chromosomes in blood samples (26)…in February 1966, 3 to 4 years after the microwave irradiation was first detected, samples were taken for chromosomal analysis. Twenty spreads were scored per sample; results were shown in Table 2 (18). (at p. 1581)

Garaj-Vrohac et al. (29) examined six men accidentally exposed while repairing microwave devices used for air traffic control in Zagreb. These subjects usually worked alternate days in a microwave field of 1250 to 1350 MHz with power density of 10 μg/W to 20 W/cm2. The accidental exposure was greater than these figures but how much is not known. The results of chromosome aberration analysis during 1984 to 1990 showed no increase in chromosomal abnormalities…(at p. 1581)

A series of studies from Croatia and Italy have also demonstrated that radar exposures are mutagenic both in vivo and in vitro (29,32-35). These findings are epidemiologically important because of the need for biological indicators of exposure and also because of the theory that somatic cell mutations lead to increased risk for cancer. The usefulness of such tests as a biological monitor seems clear from the data and the findings of excess numbers of mutations among chromosomes n blood of the group exposed at the Moscow embassy (17). (at p. 1581-1582)

Rothman et al. (9) tabulated studies that might relate leukemia to occupational or recreational exposures to RF radiation…The risk ratios for leukemia were >1.0 for 19 studies and ≤1 for 7. (at p. 1583)

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(33) Garaj-Vhrovac, V Horvat, D and Koren, Z 1991, 'The relation between colony-forming ability, chromosome aberrations, and incidence of micronuclei in V79 Chinese hamster cells exposed to microwave radiation', *Mutat Res*, vol. 263, pp.143-149.

(34) Garaj-Vhrovac, V Fucic, A and Horvat, D 1992, 'The correlation between the frequency of micronuclei and specific chromosome aberrations in human lymphocytes exposed to microwaves', *Mutat Res*, vol. 281, pp. 181-186

(35) d'Ambrosio, G et al. 1995, 'Genotoxic effects of amplitude-modulated microwaves on human lymphocytes exposed *in vitro* under controlled conditions', *Electro Magnetobiol*, vol. 14, pp. 157-164.

(17) Lilienfeld, AM et al. 1978, *Foreign service health status study, Evaluation of health status of Foreign Service and other employees from selected eastern European posts*, Final report contract 6025-619073, Department of State, Washington, US.

(9) Rothman, KJ et al. 1996, 'Assessment of cellular telephone and other radio frequency exposures for epidemiological research', Epidemiology, vol. 7, pp. 291-298. See text related to footnote 108.

(41) Szmigielski, S 1996, 'Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation', *The Science of the Total Environment*, vol. 180, pp. 9-17. RMA ID 10413. See text related to footnote 51, 90, 99, 211.

(47) Robinette, CD Silve, C and Jablon, S 1980, 'Effects upon health of occupational exposure to microwave radiation (Radar)', Am Journal of Epidemiology, vol. 112, no. 1, pp. 39-53.

…Szmigielski (41), who examined cancer by site among Polish military personnel during the period 1970 to 1989. He found a relationship between exposures to high frequency (RF radiation) and cancer morbidity. (at p. 1584)

…Robinette et al. (47)…rates of deaths attributable to…malignancy of the lymphatic and hematopoietic systems. Death rates for…Low exposure…for radarman – 6; radioman – 14; aviation electrician's mate – 0…and High exposure…for electronic technician(s) – 18; fire control technician(s) – 1; aviation electronics technician(s) – 10. Death rates for aviation electronics technician(s)…per 1000 by cause group…was 2.68, which was significantly increased, p <0.05 compared to less exposed groups. (at p. 1584)

Mrs F also highlighted Goldsmith's final comment on his interpretations from his review of literature:

There seems to be some evidence from the Moscow study and community studies in the vicinity of large FM and TV broadcasting facilities that exposures as low as 2 μW/cm2 may have long-term health effects.

A comprehensive and critical review of the epidemiological data available on health risks from RF exposure should be carried out and the reasonable measures for avoidance of the identified risks should be described and evaluated. (at p. 1586)

e. Richter, E et al. 2000 [173]

Mrs F submitted that this study was directly relevant to her personal circumstances and her contentions as her husband had been an Air Traffic Controller in the peace time RAAF for 30 years. She highlighted the authors' interpretations of other studies which she submits also support her contentions.

Accidental exposure to high levels of radar in air traffic controllers has been associated with chromosomal changes.6 Long term studies by Szmigielski et al.7 among Polish army personnel exposed to non-ionizing radiation have shown substantial increases in cancer compared with non-exposed personnel. Goldsmith's review6 suggests that there may be increased risks for cancer, reproductive effects, and other outcomes from exposures far below current regulatory levels. Goldsmith showed that in a cohort of U.S. diplomatic personnel exposed to RF/MW radiation, risks for cancer increased following exposures to levels as low as 5-18 μw.cm2.

In respect of the Richter, E et al. study findings Mrs F highlighted the following:

Following reports of the two clusters in an Israeli newspaper, we received from…over 100 individuals with similar occupational exposures to RF/MW radiation from radar. Ex-workers

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173 Richter, E et al. 2000, 'Cancer in radar technicians exposed to radiofrequency/microwave radiation', sentinel episodes, *International Journal of Occupational & Environmental Health*, vol. 6, no. 3, pp. 187-193.

[6]Goldsmith, JR 1995, 'Epidemiologic evidence of radiofrequency radiation (microwave) effects on health in military, broadcasting, and occupational studies,' *Int J Occ Environ Health*, vol. 1, pp. 47-57.

[7]Szmigielski, S 1996, 'Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation', *The Science of the Total Environment,* vol. 180, pp. 9-17.

reported leukaemia, testicular cancer, sarcoma of the foot, non-Hodgkin's lymphoma, and cancers of the thyroid, larynx, nasal gland, stomach, eye and brain. Median latency periods in this larger group were also extremely brief – less than five years – especially for leukemia, lymphoma, and testicular cancer…(at p. 191)

The findings that there was a plausible sequential distribution of latency periods for the different tumor types argues for a common exposure source for all of them. Data from the above-cited sources indicate that leukaemia, lymphoma, testicular cancer, and bran cancer are tumor types known to follow short induction periods from environmental exposures. (at p. 191)

f. Robinette, CD et al. 1980 [174]

Mrs F submitted that this study was also directly relevant to her personal circumstances and her contentions as her husband had been a Navigator during WWII. She highlighted the authors interpretations which she submits also support her contentions.

Since the development of radar during World War II, microwave-generation devices have been used in an increasing variety of military, industrial, scientific and general population appliances. Microwave sources are used almost universally in systems of telecommunications, surveillance and navigation…(at p. 39)

There is no way of integrating occupational exposure over time for men in high-exposure occupations. Their mean exposure might be very low, perhaps below 1 mW/cm2 for duty hours, but their exposure pattern is of particular interest because it infrequently includes exposures larger than 100 mW/cm2. Although this experience will not support analysis in terms of a dose-response curve, it does permit a test of whether a carefully defined class of men, known to be exposed to the highest levels in the Navy environment, did in fact show a disturbances in health associated with their occupation, i.e., with their exposure to microwave radiation. (at p. 42)

Mrs F highlighted the following results the authors provided by way of tables of which the highlighted aspects are:

Table 5 Mortality by cause of death (1950-1974): US enlisted Naval personnel exposed to microwave radiation during the Korean War period. (at p. 44)

Lymphatic and hematopoietic system, ICD code (8th Revision) 200-209:	
Low exposure:	
Radioman	6 deaths
Radarman	14 deaths
Aviation electrician's Mate	-
High exposure:	
Electronics Technician	18 deaths

[174] Robinette, CD Silve, C and Jablon, S 1980, 'Effects upon health of occupational exposure to microwave radiation (Radar)', Am Journal of Epidemiology, vol. 112, no. 1, pp. 39-53.

Fire Control Technician	1 deaths
Aviation Electronics Technician	10 deaths

Table 6 Number of deaths from disease and mortality ratios by exposure class (1950-1974): US enlisted Naval personnel exposed to microwave radiation during the Korean War period. (at p. 45)

Lymphatic and hematopoietic system, ICD code (8th Revision) 200-209:	
Low Exposure:	
Number of deaths	20
Mortality Ratio	0.83
High exposure:	
Total	
Number of deaths	26
Mortality Ratio	1.18
Electronics Technician	
Number of deaths	15
Mortality Ratio	1.06
Fire Control Technician and Aviation Electronics Technician	
Number of deaths	11
Mortality Ratio	1.40

Table 8 Mortality from malignant neoplasm of the lymphatic and hematopoietic system, high exposure group: US enlisted Naval personnel exposed to microwave radiation during the Korean War period. (at p. 46)

Lymphatic leukemia (No cases were designated as chronic lymphocytic leukemia)	
Low Exposure:	
Number of deaths	2
High exposure:	
Electronics Technician	
Number of deaths	-
Fire Control Technician and Aviation Electronics Technician	
Number of deaths	2
Total	
Number of deaths	2

Table 9 Number of deaths from disease and mortality ratios by Hazard Number: US enlisted Naval personnel exposed to microwave radiation during the Korean War period. (at p. 47)

Lymphatic and hematopoietic system, ICD code (8th Revision) 200-209:	
Low Exposure:	
Number of deaths	20
Mortality Ratio	0.83
High exposure:	
Total	
Number of deaths	26
Mortality Ratio	1.18
Hazard Number = 0	
Number of deaths	6
Mortality Ratio	1.09
Hazard Number = 1-5000	
Number of deaths	12
Mortality Ratio	1.04
Hazard Number = 5001+	
Number of deaths	8
Mortality Ratio	1.64

Table 12 Number of men receiving VA compensation, December 1976, and rates per 1000 men by diagnosis and exposure class: US enlisted Naval personnel exposed to microwave radiation during the Korean War period. (at p. 51)

Hemic, Lymphatic:	
Low Exposure:	
Number receiving compensation	10
Rate	0.5
High exposure:	
Total	
Number receiving compensation	3
Rate	0.1
Electronics Technician	
Number receiving compensation	-
Rate	-
Fire Control Technician and Aviation Electronics Technician	
Number receiving compensation	3
Rate	0.4

Mrs F highlighted the authors' final comments in respect of:

> It was not possible in this study to determine occupational exposure to microwaves after discharge from service… A subsample of living men with high and low exposure patterns during service, however, can be identified for intensive individual follow-up. This would permit assessment of the functional, nonspecific and behavioural signs and symptoms reported to affect health and work performance. (at p. 52)

64. Mrs F did not make an oral submission, notwithstanding she was afforded the opportunity to do so.

Mr G

65. An eligible person, Mr G made a written submission sent to the Council dated 20 August 2006. Mr G attached a number of medical reports specific to his health status and reports on his individual military service and claimed exposures.

66. Mr G contacted the Council in September 2010 in respect of the status of the SHOAMP report[175]. The Council advised Mr G that this report was not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

67. Mr G submitted that his exposure to aviation fuel, MEK, other chemicals used to clean and prepare the inside of aircraft fuel tanks prior to resealing, and the various sealants mixed and applied to seal the tanks were associated with chronic lymphoid leukaemia. He submitted that the F111 SHOAMP document supported his contentions.

68. Mr G did not make a written submission to the Council directly on the merits or otherwise of how the F111 SHOAMP document supported his contentions other than to state:

> …the F111 SHOAMP document clearly stated aircraft fuel was carcinogenic and was one of the causes of CLL and Depression. (at written submission 20 August 2007)

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175 SHOAMP report:

The University of Newcastle Research Associates Limited with assistance from Health Services Australia, the Australian Institute of Health and Welfare and Queensland Medical Laboratories 2003, *Study of Health Outcomes in Aircraft Maintenance Personnel (SHOAMP), Phase II Interim Mortality and Cancer Incidence Study*, administered by the Department of Veterans' Affairs, on behalf of the Department of Defence, vol. 2, pp. 1-112, accessed by SMRC on 12 September 2010 at htpp:\\www.defence.gov.au\health/research/shoamp/i-shoamp.htm.

The University of Newcastle Research Associates Limited with assistance from Health Services Australia, the Australian Institute of Health and Welfare and Queensland Medical Laboratories 2004, *Study of Health Outcomes in Aircraft Maintenance Personnel (SHOAMP), Second Mortality and Cancer Incidence Study,* administered by the Department of Veterans' Affairs, on behalf of the Department of Defence, vol. 4, pp. 1-177, accessed by SMRC on 12 September 2010 at htpp:\\www.defence.gov.au\health/research/shoamp/i-shoamp.htm.

This report was not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

69. Mr G did not make an oral submission, notwithstanding he was afforded the opportunity to do so.

Mr T and Mr M

70. Mr T and Mr M did not make a written submission to the Council and therefore could not make a complementing oral submission.

COMMISSIONS' SUBMISSIONS

71. The Repatriation Commission made written submissions dated January 2006, and February 2011 and an oral submission complementing its written submission on 31 March 2011[176].

72. A Medical Officer with the Department of Veterans' Affairs represented the Repatriation Commission at the Council's meeting on 31 March 2011 and was the principal author of the Repatriation Commission's written submission to the Council.

73. The Commission submitted that much of the information that had been available to (before) the RMA was of limited relevance in deciding the current review.

Small lymphocytic leukaemia (SLL)

74. The Commissions submitted that (at P-34):

> …it's [SLL] currently not in the CLL SOP and it is in the NHL SOP.
>
> …it [the Commissions] would like the NHL [Non-Hodgkin's Lymphoma] SOP broken up into more discrete entities as far as the epidemiology might allow and we do have a preference for grouping CLL and SLL in the one SOP. …but changing the name of the SOP to chronic lymphoid leukaemia and small lymphoid lymphoma.
>
> It's really a question of timing of that…in 2005, there really wasn't much epidemiology about SLL. …There now is some on aspects such as smoking and you do see studies that look at follicular NHL versus SLL versus other types…
>
> The other aspect…is that we wouldn't like to see it in two SOP at once.

75. In the Repatriation Commission's submission, the Commissions summarised their interpretations of the studies that had been available to the RMA reporting results for exposure to herbicides and risk of SLL. [177] (at p. 27 of 39)

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[176] The discussion of the Repatriation Commissions submissions set out in these Reasons is derived from the written submissions and complementary oral submission here identified.

No evidence could be identified that specifically examined SLL risk from herbicide exposure.

However, Tatham et al (1997), using data from a large population-based case-control study by the US Centres for Disease Control, did examine occupational exposures and risk of NHL sub-types, including a category of small cell diffuse lymphoma. This category, totalling 185 cases, comprised 124 cases of SLL and 61 cases of diffuse small cleaved lymphomas.

Results were reported for exposure to chlorophenoxy herbicides, two other general herbicide categories and exposure to pesticides. The odds ratios for these exposures and risk of small cell diffuse lymphoma were all less than 1.0 (0.45 to 0.73) and less than the corresponding odds ratio for all NHL combined (which also did not exceed 1.0).

This study also found a positive association between service in Vietnam and the small cell diffuse lymphoma sub-group (13 cases, OR 1.4, 95 % CI, 0.76 to 2.70).

Extremely low frequency electric and magnetic fields (ELF EMF)

76. The Commissions submissions focussed on electric and magnetic fields generated at 50 Hertz (Hz) or 60 Hz by electric power and also at 16 2/3 Hz by electrified railways in some countries.

77. In the Repatriation Commission's submission, the Commissions summarised their interpretations of the 21 studies reporting results for extremely low frequency electric and magnetic fields and risk of CLL that had been available to the RMA.

> …17 original studies reporting on occupational exposure…
>
> ...four on residential EMF exposure…
>
> …and one meta-analysis

78. The Commissions submitted:

> The most important epidemiological study for the purposes of this review is the 1993 study by Floderus *et al.*[2] This study reports the highest statistically significant level of risk and provides evidence in support of a dose-response effect. This is the strongest available evidence in support of a causal association between EMFs (particularly magnetic field exposure) and CLL.

a. Floderus, B et al. 1993 [178]

CLL Cases	Selected results

[177] Tatham, L et al. 1997, 'Occupational risk factors for subgroups of non-Hodgkin's lymphoma', *Epidemiology*, vol. 8 no. 5, pp. 551-558.

[178] Floderus, B et al. 1993, 'Occupational Exposure to Electromagnetic Fields in Relation to Leukemia and Brain Tumours: A Case-Control Study in Sweden', *Cancer Causes and Control*, vol. 4, pp. 465-476.

		Risk	(95% CI)
104	OR for estimated mean daily exposure by quartile		
	<- 0.15μT	1.0	
	0.16 - 0.19μT	1.1	(0.5-2.3)
	0.20 - 0.28μT	2.3	(1.1-4.3)
	>- 0.29μT	3.0	(1.6-5.8)

…and to a lesser extent the Feychting et al 1997 paper.

– Feychting, M et al. 1997 [179]

CLL Cases	Selected results		
		Risk	(95% CI)
97	OR for tertiles of mean workday exposure		
	<- 0.12μT	1.0	
	0.13 - 0.19μT	1.2	(0.7-1.9)
	>- 0.2μT	1.7	(1.0-2.9)

79. The Commissions also submitted that the other 15 epidemiological studies [180] generally are suggestive or weakly supportive of an association, but that the findings were inconsistent.

80. The Commissions considered other evidence (reviews that discuss the in vitro, animal and human laboratory evidence[181]) of importance related to whether it was biologically plausible that EMF exposure is leukaemogenic. (at p. 14 of 39)

> There is no clear evidence from cellular studies that exposure to EMF at levels that are likely to be encountered can affect biological processes. There have been some positive results but these have not been independently replicated. There is no convincing evidence that exposure to EMF is directly genotoxic nor that it can bring about the transformation of cells in culture.
>
> The carcinogenic and leukaemogenic potential of EMF exposure has been extensively studied in animal models, particularly rodents, with consistently negative results.
>
> The laboratory evidence indicates that EMF exposure is not carcinogenic in humans.

81. The Commissions submitted that:

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179 Feychting, M Forssen, U and Floderus, B 1997, 'Occupational and residential magnetic field exposure and leukemia and central nervous system tumors', *Epidemiology*, vol. 8, no. 4, pp. 384-389.

180 See **Appendix J** (Commissions Relied Upon information that was available to (before) the RMA at the relevant times)

181 See **Appendix J**.

> There is certainly, as has been noted in the evidence, that EMR can have biological effects, but whether it can have leukaemogenic effects…is where I didn't see evidence. (at P-49)
>
> Biological plausibility has not been established. (at p. 14 of 39)

82. The Commissions submitted in conclusion that: (at P-35 to 36)

> …given where the reasonable hypothesis test sits and given what a low hurdle it is, this is just about on the line… This could really go either way…we really leave it to the Council to decide as to whether we're just over the line or just short of the line for the reasonable hypothesis SOP…[exposure] does create an operational problem for the Department because however you express it, it's very difficult to assess whether the exposure has occurred because there is no active in the workplace measurement of exposure..
>
> …generally the evidence for CLL is not of a reasonable satisfaction or balance of probabilities standard.

Radiofrequency and microwave electromagnetic radiation (RF / MW)

83. The Repatriation Commission's submission refers to radiation with a frequency in the range of 3 Kilo Hz to 300 Giga Hz, including: (at p. 16 of 39)

> …that produced by microwaves, radar systems, mobile phones and other wireless communication devices and also radio and television transmitters (but excluding extremely low frequency radiation). This covers a wide spectrum of exposures and it would be desirable to be able to consider different parts of this spectrum separately. However, the nature of the available evidence does not readily allow such differentiation.

84. The Commissions identified two cohort studies in occupationally exposed subjects and three ecological studies of residential exposure that report results for risk of CLL.

85. The Commissions submitted that the most important and best evidence are the occupational exposure studies by Groves, FD et al. 2002 and Szmigielski, S 1996. The Commissions summarised their interpretations of these two studies, that had been available to the RMA and which reported results for radiofrequency and microwave electromagnetic radiation and risk of CLL.

b. Groves, FD et al. 2002 [182] (at p. 17 of 39)

> …40 year mortality study of US Korean war naval veterans. ... Exposure assessment was based on job title. … The finding from this study of most significance for the current review concerned relative risk for men with high radar exposure potential compared with men with low exposure potential.

[182] Groves, FD et al. 2002, 'Cancer in Korean War Navy technicians: mortality survey after 40 years', American Journal of Epidemiology, vol. 155, no. 9, pp. 810-18.

For CLL there were nine cases with low exposure potential and eleven with high exposure potential, giving a relative risk of 1.08 (95% CI, 0.44 to 2.66).

c. Szmigielski, S 1996 [183] (at p. 17 of 39)

> …a 15 year retrospective cohort study of cancer morbidity in Polish military personnel occupationally exposed to RF/MW radiation. … Cancer incidence (per 100,000 annually) was compared in the exposed and non-exposed groups, but not to expected rates in the general population. No actual case numbers were provided.
>
> For CLL the exposed/non-exposed ratio was 3.68 (95% CI, 1.45 to 5.18). This would appear to have been based on three cases in the exposed group (not stated in the study…)
>
> The results of this study are inconsistent with other studies… The methodology of this study has been extensively criticised by other authors.
>
> It's a positive finding but it kind of stands in isolation. (at P-37)

86. The Commissions submitted that there were three related residential studies available to the RMA and which reported results for radiofrequency and microwave electromagnetic radiation and risk of CLL. The Commissions summarised their interpretations of these studies.

a. Dolk, H et al. 1997a & b and Cooper, D et al. 2001 (an update of Dolk, H et al. 1997a) [184]

The Commissions submitted that:

> …positive results in the first [Dolk et al. 1997a] study were not replicated in the two subsequent studies. Results are summarised in the table below:

Table 3. Studies of CLL and residential exposure to RF/MW radiation

Distance of residence from tower	Dolk'97 [40] 1 tower 96 cases O/E (95% CI)	Dolk'97 (2) [41] 20 towers 969 cases O/E (95 % CI)	Cooper '01 [42] 1 tower 114 cases O/E (95 % CI)
0 - 2km	2.56 (1.11 - 5.05)	1.20 (0.83 - 1.74)	1.42 (0.59 - 2.92)

183 Szmigielski, S 1996, 'Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation', *The Science of the Total Environment,* vol. 180, pp. 9-17.

184 Dolk, H et al. 1997, 'Cancer incidence near radio and television transmitters in Great Britain. II. All High Power Transmitters', *American Journal of Epidemiology*, vol. 145, no. 1, pp. 10-17; and

Dolk, H et al. 1997, 'Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield Transmitter,' *American Journal of Epidemiology*, vol. 145, no. 1, pp. 1-9.

Cooper, D Hemmings, K Saunders, P 2001 [LETTER] 'Re "Cancer incidence near radio and television transmitters in Great Britain, (Dolk H et al. 1997) I Sutton Coldfield transmitter, (Dolk H et al. 1997) II All high power transmitters"'. *American Journal of Epidemiology*, vol. 153, no. 2, pp. 202-204.

2 - 4.9 km	2.31	1.13	
4.9 - 7.4 km	1.27	0.99	
7.4 - 10 km	1.12	0.97	
0 - 10 km	1.32 (1.08 - 1.62)	1.02 (0.95 - 1.08)	1.19 (0.97 - 1.40)

WE observed/expected
95 % CI 95% confidence interval

87. The Commissions submitted that 3 of the above studies[185] provide information in relation to exposure:

a. Dolk, H et al 1997

> …field strength measurements for the Sutton Coldfield transmitter. Transmissions were in the 30 MHz - 1GHz frequency range. The maximum field strengths at any point were 0.0013 milliwatts per square centimetre (mW/cm) for TV and 0.005 mW/cm2 for FM radio.

b. Szmigielski, S 1996

> …reported exposure was mostly in the 150 - 3500 MHz range with field strengths of <0.2 mW/cm2 at 80-85 % of posts and some incidental exposure at >0.6 mW/cm2.

c. Groves, FD et al 2002

> …reported usual exposure to microwave frequencies in the potentially highly exposed subjects of below 1 mW/cm^2, but potential for exposures exceeding 100 mW/cm^2.

88. The Commissions summarised their interpretations of two other studies [186]: (at p. 19-20 of 39)

a. Morgan, RW et al. 2000

> …epidemiological studies of RF/MW radiation with results for outcomes other than CLL. …the best and most noteworthy. This was a large, well conducted, cohort mortality study of Motorola employees with potential for RF/MW radiation exposure from a range of communication devices.
>
> Exposure assessment was based on a detailed job exposure matrix with over 9,000 job titles classified into four categories of exposure. The cohort comprised nearly 200,000 workers, with nearly 17,000 having moderate or high exposure. At the end of the study there were 6,300 deaths.

185 ibid

186 Morgan, RW et al. 2000, 'Radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems', *Epidemiology*, vol. 11, no. 2, pp. 118-127.

Krewski, D et al. 2001, 'Potential health risks of radiofrequency fields from wireless telecommunication devices', *Journal of Toxicology & Environmental Health*, part B, critical reviews, vol. 4, no. 1, pp. 1-143.

The study findings did not support an association between exposure and lymphatic or haematopoietic cancers. For leukaemia (87 cases), rate ratios were all around or below 1.0 for all the models tested.

b. Krewski, D et al 2001

…information concerning laboratory based studies and the biological effects of RF/MW radiation. This evidence has been well reviewed by a Royal Society of Canada expert panel. [32] In brief summary, although some biological effects of low (non-thermal) level RF/MW radiation have been observed, the available evidence to date does not suggest that such radiation is leukaemogenic.

89. The Commissions submitted in conclusion [187] that: (at p.-20 of 39)

Limited epidemiological evidence is available that directly examines the risk of CLL from RF/MW exposure. The extent and quality of this evidence is not sufficient to permit a satisfactory evaluation of risk. Inadequate exposure assessment in the available studies is the major limitation, but there are substantial other methodological shortcomings.

Positive associations between RF/MW exposure and CLL have been reported in a study of Polish military personnel and in the first of a series of studies of populations living close to radiofrequency transmission towers.

[Dolk, H et al 1997] The Commission considers that residential distance from a transmission tower is not a reliable or useful proxy for exposure and that the residential exposure studies do not provide meaningful support for an association between RF/MW exposure and CLL.

[Szmigielski, S 1996] The Commission does not consider that the Szmigielski study, with (evidently) only three CLL cases and serious methodological concerns, is sufficient to point to an association between RF/MW exposure and CLL.

The strength of any association between RF/MW exposure and CLL is really unknown due to a lack of adequate quality data, there is no consistent support across the available studies, a biological gradient has not been demonstrated and no biologically plausible mechanism has been established. In the Commission's view, the inclusion of a factor for RF/MW exposure in Instrument 9 of 2005 is not warranted.

Herbicides / Dioxin (TCDD)

90. In the Repatriation Commission’s submission, the Commissions provided some background to exposure to dioxins. (at p 21 of 39)

The main potential exposure to dioxin that is of interest concerns dioxin as a contaminant of Agent Orange and other herbicide formulations used in Vietnam.

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187 Council notes that this does not reflect the Council's two-step process in applying the reasonable hypothesis and balance of probabilities tests as set down by the full Federal Court (see [108**Error! Reference source not found.**] - [110]).

Agent Orange and several other herbicide formulations used in Vietnam contained 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). An inadvertent contaminant in the manufacture of 2,4,5-T was 2,3,7,8-tetrachlorodibenzopara-dioxin (TCDD). The other constituents used in the colour-coded herbicides in Vietnam were 2,4-dichlorophenoxyacetic acid (2,4-D), cacodylic acid and picloram. These were not contaminated by TCDD…used by the US military and sprayed extensively

The Australian experience…didn't directly spray or handle those herbicides [used by the US]…their exposure…to some other herbicides, particularly bromacil and diquat…there was also some paraquat used, some Tordon and something called polychlorate…(at P-43)

91. In the Repatriation Commission's submission, the Commissions provided some background to exposure to pesticides. (at P-43)

Insecticide exposures as well that might be of relevance: things like malathion and DDT, dieldrin, chlordane, diazinon, pyrethins, lindane.

92. The Commissions advised the Council that: (at P-42)

…the non-Hodgkin's lymphoma SOP and other SOPs that have factors covering this [exposure to herbicides (and dioxins / TCDD) and pesticides]…deal with it in a number of ways. There's factors for herbicides generally, there's factors for TCDD…there are factors that cover service in Vietnam generally, without any exposure being given, for service on land or at sea…and attached to that latter factor…another factor for potable water which really does not fit with the factor its attached to – not a factor for being on land or at sea in Vietnam…specifically a TCDD exposure or at least a potential TCDD exposure.

…the Commissions would encourage you [the Council] in looking at this issue, to look at it in similar terms of the factors for herbicides in general or any specific herbicides in particular or the dioxin-contaminated herbicides.

…there are no factors in the CLL SOP but…in 1995, the Commission used its powers under section 180A of the Act to effectively give coverage to veterans with the four leukaemia subtypes, including CLL, so that claims for CLL from Vietnam veterans who were on land or sea for 30 days do succeed despite the RMA SOP not having a factor[188].

Being exposed to herbicides is defined in the Commission determination as below.
"being exposed to herbicides in Vietnam" may be said to have occurred only if the person had:

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188 The Commissions submitted that the evidence the section 180A determination was based on was:

Institute of Medicine 1994, *Veterans and Agent Orange: Health Effects of Herbicides Used in Vietnam*, Committee to review the health effects in Vietnam Veterans of exposure to herbicides, The National Academic Press, Washington DC.

MacLennan, R & Smith, P 1994, *Veterans and Agent Orange – Health effects of herbicides used in Vietnam*, report to the Department of veteran's Affairs, Canberra.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

(a) rendered more than 30 days service on land in Vietnam; or

(b) regularly eaten fish, fish products, crustaceans, shellfish or meat from Vietnam; or

(c) regularly eaten food cooked with water from Vietnam discoloured by sediment, or regularly drunk water from Vietnam discoloured by sediment; or

(d) regularly inhaled dust in a defoliated area in Vietnam or regularly inhaled herbicide fog in Vietnam; or

(e) sprayed or decanted herbicides in Vietnam as an occupational requirement.

93. The Commissions submitted that:

> …in terms of trying to identify specific chemicals, I think you could make a case for CLL for having a factor for being a farmer or being a rural worker. If you look at studies like those ones cited in the Veterans and Agent Orange Report…

94. In the Repatriation Commission's submission, the Commissions summarised their interpretations of the studies that had been available to the RMA reporting results for exposure to pesticides, herbicides / dioxin and risk of CLL.

a. IOM Update 2002 [189] (at p 23-24 of 39)

> In the 2002 update to the NAS Veterans and Agent Orange report (VAO 2002), evidence concerning CLL was evaluated separately from the other leukaemias. In the initial 1994 report and the subsequent biennial updates up to 2000, leukaemia had been considered as a combined group. The conclusion in the initial and subsequent reports up to 2000 had been that there was inadequate or insufficient evidence to determine whether an association exists between leukaemia and exposure to herbicides (2,4-D; 2,4,5-T and its contaminant TCDD; cacodylic acid; and picloram). In the 2002 update it was concluded that for CLL there was sufficient evidence of an association. Following release of VAO 2002, the US Department of Veterans' Affairs extended benefits to Vietnam veterans with CLL.
>
> The NAS had previously classified three other malignancies as having sufficient evidence of an association (non-Hodgkin's lymphoma, Hodgkin's lymphoma and soft tissue sarcoma).
>
> The thing that had changed [between Update 2000 to Update 2002] was the specific directive, I think, from the US government to reinvestigate…to look more carefully at the Leukaemia sub-types…up until a point they [IOM, NAS] looked at the Leukaemia collectively. …and the WHO classification had come along and there were a number of things in combination that caused them [IOM, NAS] to change what they had said in 2000 [Update 2000]. There were no new studies between Update 2000 and 2002. (at P-49)
>
> CLL is thus the only disease where the views of the RMA and the NAS are not in accord.

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189 Institute of Medicine 2002, [PREPUBLICATION COPY] *Veterans and Agent Orange Update 2002*, Committee to review the health effects in Vietnam veterans of exposure to herbicides (fourth biennial update), Chapter 6, National Academy Press, Washington, D.C., pp. 175-299.

Council notes that this does not reflect the Council's two-step process in applying the reasonable hypothesis and balance of probabilities tests as set down by the full Federal Court (see [108] – [110]).

The original studies evaluated in VAO 2002, all of which were available to the RMA (seven studies) are summarized in the table below.

"**TABLE 6-50** Selected Epidemiologic Studies- Chronic Lymphocytic Leukemia" [190]

Reference	Study Population	Exposed Cases[a]	Estimated Relative Risk (95% *CI*)[a]
OCCUPATIONAL			
Studies Reviewed in *Update **1998***			
Waterhouse et al., 1996	Residents of Tecumseh, Michigan-CLL	10	SIR =1.8 (0.8-3.2)
Amadori et al., 1995	CLL in Italian workers		
	Farming or animal-breeding workers	15	2.3 (0.9-5.8)
	Farming	5	1.6 (0.5-5.2)
	Animal-breeding	10	3.1 (1.1-8.3)
Studies Reviewed in *VAO*			
Hansen et al., 1992	Danish gardeners		
	All gardeners-CLL	6	2.5 (0.9-5.5)
	Male gardeners-CLL	6	2.8 (1.0-6.0)
Brown et al., 1990	Residents of Iowa and Minnesota		
	CLL, ever farmed	156	1.4 (1.1-1.9)
	CLL, any herbicide use	74	1.4 (1.0-2.0)
Blair and White, 1985	Residents of Nebraska		
	All cases, all leukemia- farming	1,084	1.3
	CLL	248	1.7 (CI did not include 1.0)
Burmeister et al. 1982	Residents of Iowa		
	CLL in white male farmers using herbicides		1.9 (1.2-3.1)
ENVIRONMENTAL			
Studies Reviewed in *Update 2000*			
Bertazzi et al., 2001	Seveso residents-20-year follow-up-lymphatic leukemia		
	Zone A	0	-
	Zone B	2	1.1 (0.3-4.4)
	Total	2	1.0 (0.2-3.9)

[a]Given when available.
- When information was denoted by a dash in the original study. Abbreviations: USDA, United States Department of Agriculture.

> There's quite a bit of positive associations being found there [Morris Brown et al. 1990 in Iowa and Minnesota farmers; Blair and White 1985 in Nebraska farmers; Burmeister et al. 1982 in Iowa farmers; and Hanson et al. 1992 in Danish gardeners], a lot of associations being looked at. (at P-44)

b. Brown, LM et al. 1990 [191] (at p. 25 of 39)

[190] For Table 6-50, see p. 374 of:

Institute of Medicine 2002, *Veterans and Agent Orange Update 2002*, Committee to review the health effects in Vietnam veterans of exposure to herbicides (fourth biennial update), National Academy Press, Washington, D.C.

In the Commissions; view the population-based case-control study of pesticide exposure and leukaemia by Brown et al is the most important…

…significant positive associations were found for the following categories: ever farmed; use of any herbicide; use of any insecticide; use of any animal insecticide; use of carbonates; use of organophosphates; ever handled dichlovos; and ever handled nicotine.

CLL risk in those who: ever handled 2,4-D was 1.3 (45 cases, 95 % CI, 0.8-2.0); ever handled 2,4,5-T was 1.6 (10 cases, 95 % CI, 0.7-3.4); and first handled 2,4,5-T at least 20 years prior to interview was 3.3 (7 cases, 95 % CI, 1.2-8.9).

No consistent dose-response patterns (by days per year handled) were seen for any of the herbicides or insecticides.

c. Michalek, JE et al. 1990;

Dalager, NA & Kang, NK 1997

Ketchum, NS et al. 1999 [192] (at 25 of 39)

US Air Force and Army chemical corps personnel who were involved in spraying herbicides in Vietnam had high potential for herbicide / TCDD exposure and have been shown to have elevated serum levels of TCDD. A number of mortality studies on these subjects have been published. The cohorts have been too small and too young to adequately assess CLL risk. The studies do not indicate any excess risk of cancer, but have included only two cases of leukaemia.

d. Australian Institute of Health and Welfare (AIHW) 2000 [193] (at P-44)

Other studies have examined larger cohorts of Vietnam veterans in general. …but the studies do not attempt to quantify exposure or stratify subjects on the basis of exposure. Only the Australian morbidity study has any information on CLL, but this is not helpful in assessing CLL risk from herbicide exposure.

e. IOM Update 2004 and Update 2006 [194] (at p. 8 of 11 (2011))

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191 Morris Brown, L et al. 1990, 'Pesticide exposures and other agricultural risk factors for leukemia among men in Iowa and Minnesota', *Cancer Research*, vol. 50, no. 20, pp. 6585-6591.

192 Michalek, JE et al. 1990, 'Health status of air force veterans occupationally exposed to herbicides in Vietnam', *JAMA*, vol. 264, no. 14, pp. 1832-1836.

Dalager, NA & Kang, HK 1997, 'Mortality among army chemical corps Vietnam Veterans', *American Journal of Industrial Medicine*, vol. 31, pp. 719-726.

Ketchum, NS et al. 1999, 'Serum dioxin and cancer in veterans of operation ranch hand', *American Journal of Epidemiology*, vol. 149, no. 7, pp. 630-639.

193 Australian Institute of Health and Welfare (AIHW) 2000, *Morbidity of Vietnam Veterans. Adrenal gland cancer, leukaemia and non-Hodgkin's lymphoma: Supplementary Report no 2* (AIHW cat. No. PHE 28). Canberra: AIHW. ISBN 1 74024 083 9.

194 Institute of Medicine 2005, *Veterans and Agent Orange: update 2004*, Committee to review the health effects in Vietnam veterans of exposure to herbicides (fifth biennial update), The National Academic Press, Washington DC.

Update 2004, in the section on CLL, cited one new study by Akhtar *et al* (2004)[195], which showed no excess of lymphopoietic cancers in US Ranch Hand veterans, based on national incidence rates. Results were not provided separately for CLL.

In Update 2006, in the section on CLL., new studies identified were The Australian Vietnam Veterans health study [Wilson, EJ et al. 2005]…and a 1997 study by Hertzman *et al* that had not been previously considered.

Update 2004 and Update 2006 both affirmed the conclusion made in Update 2002 (discussed in the Commission's 2006 submission) that there was sufficient evidence of an association between exposure to the compounds of interest and CLL. These conclusions were again based largely on the evidence linking herbicide exposure to non-Hodgkin's leukaemia (NHL) and the plausibility of a common aetiology for CLL and NHL.

f. Wilson, EJ et al. 2005 [196] (at P-44)

…it found 58 cases of CLL for a standardised incident ratio of 1.55 and that was statistically significant at confident intervals starting at 1.15 [95% CI 1.15 – 1.95, see at Table 6-3, p. 84].

So there's an excess rate of CLL in Australian Vietnam veterans overall, and then by branch of service, that excess is there in army and in navy, but not for air force…

Table 1. CLL incidence in Australian male Vietnam veterans vs general population 1982-2000

Branch of Service	No of cases	SIR*	95% CI
All	58	1.55	1.15 - 1.95
Army	42	1.68	1.18 - 2.19
Navy	12	1.51	0.78 - 2.63
Air Force	4	0.87	0.24 - 2.23

*results are for "scenario 1" from the study, which excluded veterans whose status was unknown

Institute of Medicine 2007, [PREPUBLICATION COPY] *Veterans and Agent Orange: update 2006*, Committee to review the health effects in Vietnam veterans of exposure to herbicides (sixth biennial update), The National Academic Press, Washington DC.

195 Akhtar, FZ et al. 2004, 'Cancer in US Air Force veterans of the Vietnam war', *J Occup and Environ Med*, vol. 46, no. 2, pp. 123-136.

The Council notes that Akhtar, FZ et al. 2004 was a follow-up study of that by Ketchum, NS et al. 1999 and that the Akhtar, FZ et al. 2004 article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

196 Wilson, EJ et al. 2005, *Cancer Incidence in Australian Vietnam veterans study,* Department of Veterans' Affairs, Canberra.

Wilson, EJ et al. 2005, *The Third Australian Vietnam veterans mortality study,* Department of Veterans' Affairs, Canberra.

Wilson, EJ et al. 2005, *Australian National Service Vietnam veterans Mortality and Cancer Incidence study,* Department of Veterans' Affairs, Canberra.

The mortality study, using a similar methodology, reported a non-significantly elevated standardized mortality ratio (SMR) for lymphoid leukaemia in veterans. but did not provide results for CLL.

The national servicemen cancer incidence study, comparing Vietnam veterans with non-Vietnam veterans and with the general population, reported a relative risk for CLL in the veterans 0.90 (95% CI, 0.31 to 2.45) versus non-veterans. There were 8 observed cases, versus 9 expected from the general population data.

These reports provide no information or analysis concerning possible dioxin or dioxin-contaminated herbicide exposure in the veterans.

g. Hertzman, C et al. 1997 [197] (at p. 8 of 11 (2011))

…was a study of mortality and cancer incidence in British Columbia sawmill workers exposed to chlorophenate wood preservatives. The chlorophenates the workers, were exposed to were dioxin-contaminated, but the exposure was to hexa-, hepta- and octa-chlorinated isomers rather than TCDD.

An excess of incident CLL was found in exposed mill workers. There were 24 cases found versus 14.3 expected, giving an SIR of 1.67 (95% CI, 1.16 to 2.36). Results based on cumulative hours of exposure to chlorophenates were also provided for CLL. No trend with increasing exposure was seen.

95. In the Repatriation Commission's submission, the Commissions summarised their interpretations of the studies that had been available to the RMA reporting results for exposure to exposure to dioxin and risk of CLL.

The best available evidence concerning dioxin exposure and cancer risk comes from industrially-exposed cohorts and from the population exposed to TCDD following the industrial accident in Seveso. Studies of industrially-exposed cohorts include considerably higher exposure levels and better exposure assessments than studies of agricultural workers. The Seveso cohort had lower exposure levels than seen in industrial cohorts but good TCDD exposure assessment. (at p. 26 of 39)

a. IARC – Kogevinas, M et al. 1997 [198] (at p. 26 of 39)

…multicentre historical cohort mortality study of phenoxy herbicide production workers and sprayers. This study combined 36 cohorts from 12 countries and comprised 21,863 subjects and approximately ½ million person-years of follow-up. Exposure was reconstructed from individual job records, questionnaires and in some subjects, by measurement of TCDD levels in serum and adipose tissue.

In workers exposed to TCDD-contaminated phenoxy herbicides, the study reported mortality rates slightly higher than expected for cancer overall with an SMR of 1.12 (95 % CI, 1.04 to 1.21). For workers with 20 or more years of exposure the SMR for cancer overall was 0.92.

197 Hertzman, C et al. 1997, 'Mortality and cancer incidence among sawmill workers exposed to chlorophenate wood preservatives', *American Journal of Public Health*, vol. 87, no. 1, pp. 71-79.

198 IARC – Kogevinas, M et al. 1997, 'Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins', *American Journal of Epidemiology*, vol. 145, no. 12, pp. 1061-1075.

No results were presented for CLL, but for leukaemia combined (16 deaths) the SMR was 0.73 (95 % CI, 0.42 to 1.19).

b. Steenland, K et al. 1999 [199] (at p. 26 of 39)

The most recent available report on the largest of the individual cohorts in the IARC study… This report includes a TCDD exposure-response analysis, finding a statistically significant positive linear trend in SMRs with increasing exposure for all cancers combined and for lung cancer. Excess cancer was limited to the most highly exposed workers, with exposures that were likely to have been 100 to 1000 times higher than those experienced by the general population. There was no excess of haematopoietic cancers in the most highly exposed subjects (3 cases, 4.8 expected).

c. Bertazzi, PA et al. 2001 [200] (at p. 26 of 39)

For subjects in the highly exposed zones (A and B) the relative risk for all cancers after 20 years was 1.0. For lymphatic leukaemia two cases were observed, versus 2.1 expected.

d. IARC 1997 [201] (at p. 27 of 39)

IARC concluded that there was limited evidence of carcinogenicity in humans, but sufficient evidence of carcinogenicity in animals. Those findings, in combination with mechanistic considerations, led IARC to classify TCDD in Group 1 - carcinogenic to humans.

e. Popp, JA et al. 2006 [202] (at p. 8 and 9 of 11 (2011))

This report considered the toxicological. animal and human epidemiological evidence concerning the carcinogenicity of TCDD.

The report concluded that the available information supported a non-linear, threshold dose-response relationship for human cancer risk from TCDD exposure.

f. Read, D et al. 2007 [203] (at p. 7 of 11 (2011))

The study utilized cancer registry data to compare rates in the population within the New Plymouth Territory with those in the rest of the country. Within the region [in the suburb of Paritutu] there had been a 2,4,5-T manufacturing plant. operating from 1962 to 1987. Residents who lived in proximity to the plant had potential non-occupational exposure to TCDD from pollution from the plant. Workers at the plant had potential occupational exposure to TCDD.

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199 Steenland, K et al. 1999, 'Cancer, heart disease, and diabetes in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin', *J Natl Cancer Inst*, vol. 91, no. 9, pp. 779-786.

200 Bertazzi, PA et al. 2001, 'Health effects of dioxin exposure: a 20-year mortality study', *Am J Epidemiol*, vol. 153, no. 11, pp. 1031-1044.

201 IARC 1997, *Polychlorinated Dibenzo-para-Dioxins and Polychlorinated Dibenzofurans*, vol 69, International Agency for Research on Cancer Overall Evaluations of Carcinogenicity Risks to Humans, IARC, Lyon, France.

202 Popp, JA et al. 2006, 'A weight-of-evidence analysis of the cancer dose-response characteristics of 2,3,7,8-Tetrachlorodibenzodioxin (TCDD)', *Toxicol Sci*, vol. 89, no. 2, pp. 361-369.

203 Read, D et al. 2007, 'Cancer incidence and mortality in a New Zealand community potentially exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin from 2,4,5-trichlorophenoxyacetic acid manufacture', *Aust N Z J Public Health*, vol. 31, no. 1, pp. 13-18.

[The study]…provided SMRs and SIRs for CLL, for five year periods from 1970 to 2001, for New Plymouth area residents versus the general New Zealand population. Elevated SIRs were reported for the periods 1970-74 and 1980-84. but no consistent pattern was seen for CLL, for the other specific cancers examined (NHL, Hodgkin's disease and soft tissue sarcoma), or for cancer overall.

g. 't Mannetje, A et al. 2005 [204] (at p. 7 of 11 (2011))

… examined mortality in cohorts of New Zealand phenoxy herbicide production workers and sprayers over a 32 year period. There were 813 production workers and 699 sprayers classified as exposed to phenoxy herbicides, TCDD and higher chlorinated dioxins. Exposure was mostly to 2,4,5-T. The production workers were front the Paritutu, New Plymouth manufacturing plant mentioned above [Read, D et al. 2007]. There were no deaths from CL.L in the plant production workers, nor in the sprayers, during the study period.

96. The Commissions submitted in conclusion that: (at p. 28 of 39)

In the available information there are no epidemiological reports that specifically show an increased risk of CLL following exposure to TCDD. In the industrially-exposed cohorts, with the highest exposure to TCDD-contaminated 2,4,5-T, there are no data on CLL.

Studies of Vietnam veterans have not shown them to be at increased risk of CLL. Data from animal studies are also not supportive.

Only one study [Morris Brown, L et al 1990] is available that reports on risk of CLL from exposure specifically to 2,4-D or 2,4,5-T. That study found weakly positive associations between exposure to those agents and CLL, but these were not statistically significant except for exposure to 2,4,5-T at least 20 years before interview.

However, the study also found a large number of other statistically significant positive associations for exposure to an array of different types of other herbicides and insecticides. The range of different, concomitant exposures in the study subjects raises the possibility of positive associations being due to confounding.

[Wilson, EJ et al. 2005]…its quite hard to know what to make of that data, but there is an excess of CLL from having been in Vietnam, so on that basis the Commission…invite you [the Council] to look at whether that warrants having a factor in the SOP or not. (at P-45)

97. The Commissions noting the differing views of the RMA and of the NAS [205] also submitted in conclusion that: (at p. 28 of 39)

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[204] 't Mannetje, A et al. 2005, 'Mortality in New Zealand workers exposed to phenoxy herbicides and dioxins', *Occup Environ Med*, vol. 63, pp. 34-40.

[205] See in these Reasons [94].

> The Commission prefers not to offer a firm view at this time on whether a dioxin or herbicides factor should be included in the CLL RH SOP.

98. In the Repatriation Commission's submission, the Commissions summarised their interpretations of the studies that had been available to the RMA reporting results for exposure to exposure to dioxin via potable water and risk of CLL.

a. NRCET 2002[206]

> That study found that the evaporative distillation process used on Navy ships in the Vietnam War era to produce potable water did not remove, but rather would have enriched certain contaminants, including dioxins. That is, if dioxins, including TCDD, were present in the source water, their concentration would likely be increased in the potable water that was produced.
>
> The study did not test whether 2,4-D or 2,4,5-T could be similarly co-distilled.
>
> A number of RMA SOPs, such as those for non-Hodgkin's lymphoma and myeloma (but not the leukaemias) recognise being in Vietnam (during the Vietnam war) as a causal factor. SOPs that have that factor are now being progressively amended by the RMA to include "consuming potable water produced by evaporative distillation of estuarine Vietnamese waters".

99. In the Repatriation Commission's submission, the Commissions did not submit a position in respect of a contended factor for exposure to dioxin via potable water and risk of CLL. (at P-47)

> The Commission would like to leave it to you [the Council] and not put any position on whether that association warrants inclusion of a factor.

Solvents, including Benzene

100. In the Repatriation Commission's submission, the Commissions submitted: (at p. 29 of 39)

> Organic solvents that have been evaluated for their carcinogenic potential include the following: acetone; benzene; carbon tetrachloride; isopropanol; methyl chloride; methylene chloride; methyl ethyl ketone; styrene; tetrachloroethylene; trichloroethane; trichloroethylene; toluene; and xylene.
>
> Extensive evidence is available concerning benzene and leukaemia risk. For the other solvents the Commission could find no data on CLL risk from specific solvent exposures and no evidence of a statistically significant increased risk of leukaemia from exposure to any specific solvent.

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206 Muller, J 2002, *Examination of the Potential Exposure of Royal Australian Navy (RAN) Personnel to Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans via Drinking Water*, The National Research Centre for Environmental Toxicology (ENTOX)[NRCET], A Report to the Department of Veteran Affairs, Australia. RMA ID 27791

101. In the Repatriation Commission's submission, the Commissions summarised their interpretations of the studies that had been available to the RMA reporting results for benzene and risk of CLL. (at 29 of 39)

The RMA had reports available on three long-term cohort studies of workers heavily exposed to benzene. These reports were on Chinese factory workers the Pliofilm cohort in the USA, and Italian shoe factory workers.

a. Chinese factory workers [207] (at 29 of 39)

Hayes, RB et al. 1997; and

Hayes, RB et al. 2000

b. Pilofilm cohort in the USA [208] (at 29 of 39)

Rinsky, RA et al. 2002; and Risky, RA et al. 1987

Paxton, MB et al. 1994

Crump, KS 1993

Wong, O 1995

c. Italian Shoe factory workers [209] (at 29 of 39)

Seniori Costantini, A et al. 2003

All three studies reported strongly elevated risks of leukaemia from benzene exposure with clear dose-response effects.

The Chinese and US studies found significantly elevated risks particularly for acute myeloid leukaemia.

There were no cases of CLL in the Pilofilm cohort and there appear to have been no cases of CLL in the Chinese cohort (not specified - a non-significant excess of lymphocytic leukaemia was noted but attributed to five cases of acute lymphocytic leukaemia).

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207 Hayes, RB et al. 1997, 'Benzene and the dose-related incidence of hematologic neoplasms in China', Journal of the National Cancer Institute, vol. 89 no. 14, pp. 1065-1071.

Hayes, RB 2000, 'Benzene and lymphohematopoietic malignancies in China, J Toxicol Environ Health, vol. 61, no. 5-6, pp. 419-432.

208 Rinsky, RA et al. 2002, 'Benzene exposure and hematopoietic mortality: a long-term epidemiological risk assessment', *American Journal of Industrial Medicine*, vol. 42, no. 6, pp. 474-480.

Rinsky, RA et al. 1987, 'Benzene and Leukemia. An epidemiologic risk assessment', *NEJM*, vol. 316, no. 17, pp. 1044-1050.

Paxton, MB et al. 1994, 'Leukemia risk associated with benzene exposure in the pilofilm cohort: 1, Mortality update and exposure distribution, *Risk Analysis*, vol. 14, no. 2, pp. 147-154.

Crump, KS 1993, 'Risk of benzene- induced leukemia: A sensitivity analysis of the pilofilm cohort with additional follow- up and new exposure estimates', *Journal of Toxicology and Environmental Health*, vol. 42, pp. 219-242.

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

209 Seniori Costantini, A et al. 2003, 'Exposure to benzene and risk of leukemia among shoe factory workers', *Scand J Work Environ Health*, vol. 29, no. 1, pp. 51-59.

The Italian study provided no information on leukaemia sub-types.

102. In the Repatriation Commission's submission, the Commissions summarised their interpretations of the studies that had been available to the RMA reporting results for benzene (less heavily exposed workers) and risk of CLL. (at 29 of 39)

> …other studies of much less heavily exposed workers, particularly in the petrochemical industry, were available, with a number having results for CLL.
>
> Low exposure levels and low case numbers, particularly for leukaemia sub-types, limit the power of such studies to detect excess risks.

a. Rushton, l & Romaniuk, H 1997 [210] (at 29 of 39)

> …had 31 CLL cases and assessed risk by cumulative exposure and other variables. Significantly elevated risks were not seen (except when a reference category with only one case subject was used) and there was no evidence for an increasing risk with increased exposure.

b. Glass 2003 211 (at 29 of 39)

> Australian…study of low level benzene exposure in petroleum industry workers reported on 11 CLL cases. For leukaemia, risk was reported for six levels of cumulative lifetime exposure. Because of low case numbers the three lowest and two highest groups were combined in the CLL analysis, giving three exposure categories. Increased risks were then seen in the higher two categories but these were not statistically significant.

c. Wong, O & Raabe, GK 1995 [212] (at 30 of 39)

> A meta-analysis of leukaemia cell-type-specific mortality in 19 cohorts of petroleum workers… There were 83 CLL deaths in the total cohort. Analyses of the total cohort and various subsets found lower than expected mortality rates for CLL, with SMRs around 0.8 to 0.9. Data were not available for analysis by level of exposure.

103. The Commissions concluded: [213] (at 30 of 39)

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210 Rushton, L & Romaniuk, H 1997, 'A case-control study to investigate the risk of leukaemia associated with exposure to benzene in petroleum marketing and distribution workers in the United Kingdom', *Occupational and Environmental Medicine*, vol. 54, pp. 152-166.

211 Glass, DC et al. 2003, 'Leukemia Risk Associated With Low-Level Benzene Exposure', *Epidemiology*, vol. 14, no. 5, pp. 569-577.

212 Wong, O & 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.

213 Council notes that this does not reflect the Council's two-step process in applying the reasonable hypothesis and balance of probabilities tests as set down by the full Federal Court (see [108] – [110]).

In the Commission's view this evidence leaves open the possibility that benzene exposure is associated with CLL, but is insufficient to point to a causal association.

Tobacco

104. In the Repatriation Commission's submission, the Commissions summarised their interpretations of the 4 studies reporting results for smoking and risk of CLL that had been available to the RMA.

a. Adami et al. 1998 [214]

- Long-term prospective cohort study of smoking and risk of leukaemia, lymphoma and myeloma;
- CLL cases – 169;
- CLL not related to smoking status (data not provided).

b. Friedman, 1993 [215]

- Long-term prospective cohort study of smoking and risk of leukaemia, lymphoma and multiple myeloma;
- CLL cases – 117;

Men (n=71)

status	**OR**	**(95% CI)**
Current	0.8	(0.5 – 1.5)
Former	1.0	(0.5 – 1.8)

Women (n=46)

status	**OR**	**(95% CI)**
Current	0.6	(0.3 – 1.3)
Former	0.6	(0.2 – 1.7)

c. Stagnaro et al 2001[216]

- Population based case-control study of smoking and haematolymphopoietic malignancies;
- CLL cases – 123;

status	**OR**	**(95% CI)**

[214] Adami, J et al 1998, 'Smoking and the risk of leukaemia, lymphoma, and the multiple myeloma (Sweden)', *Cancer Causes & Control*, vol 9, no. 1, pp. 49-56.

[215] Friedman, GD 1993, 'Cigarette smoking, leukemia, and multiple myeloma', *Annals of Epidemiology*, vol 3, pp. 425-428.

[216] Stagnaro, E et al. 2001, 'Smoking and hematolymphopoietic malignancies', *Cancer Causes Control*, May, vol. 12, no. 4, pp. 325-334.

Ever	0.87	(0.61 – 1.2)
Current	0.81	(0.53 – 1.2)
Former	0.93	(0.62 – 1.4)

No trend with cigarettes per day or with smoking duration

d. Brown et al 1992[217]

- Population based case-control study of smoking and risk of leukaemia;
- CLL cases – 215;

status	**OR**	**(95% CI)**
Ever	1.6	(1.1 – 2.4)
Current	1.7	(1.0 – 2.7)
Former	1.5	(1.0 – 2.3)

Positive…no trend seen with cigarettes per day or duration of smoking

105. The Commissions concluded:

> Although one of these studies shows a positive result association between smoking and CLL, there are two long term prospective studies showing no association and there is no evidence of a dose-response trend.
>
> In the Commission's view, these data do not support a causal association [218] between smoking and CLL.

Repatriation Commission's comments on the Scope of Review and Pool of Information

106. The Commission stated that there were no other factors it wanted the Council to consider apart from the contended factors.

107. The Commission concurred with the Council's proposed pool of information.

REASONS FOR THE COUNCIL'S DECISION

The Council's Task

108. In conducting a review the Council follows a two-step process. It first identifies the pool of information, ie by identifying from all the information that was available to the RMA when it determined, amended, or last amended the Statements of Principles,

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217 Brown, LM et al. 1992, 'Smoking and risk of leukemia', *American Journal of Epidemiology*, vol. 135, no. 7, pp. 763-768.

218 The Council notes that the Commissions use of " a causal association" and "point to a casual association" methodology which does not reflect the Council's two step process in applying the reasonable hypothesis test as set down by the full Federal Court (see [108] – [110])

the sound medical-scientific evidence (as that term is defined in section 5AB(2) of the VEA (see [10] above)) which touches on (ie is relevant to) the issue of whether a particular kind of injury, disease or death can be related to service.

109. The second step requires the Council to determine whether:

a. there is sound medical-scientific evidence in the pool of information that points to (as opposed to merely leaves open)[219] the relevant possibility, that is whether the contended factors (if found to exist in a particular case) could provide a link or element in a reasonable hypothesis connecting Chronic Lymphoid Leukaemia or death from Chronic Lymphoid Leukaemia to relevant[220] service[221]). The Council had to find that the hypothesis contended for was reasonable, and not one which was 'obviously fanciful, impossible, incredible or not tenable or too remote or too tenuous'[222].

b. the sound medical-scientific evidence in the pool of information concerning the contended factors (if found to exist in a particular case) could provide a relevant connection between Chronic Lymphoid Leukaemia or death from Chronic Lymphoid Leukaemia and relevant[223] service according to a standard of satisfaction 'on the balance of probabilities', or as being 'more probable than not'.

110. In these Reasons the association for both the reasonable hypothesis test (paragraph [109]) and the balance of probabilities test (paragraph [109]) are respectively referred to as the 'relevant association'.

111. It was with these tests firmly at the forefront of its collective mind that the Council considered the sound medical-scientific evidence in the pool of information and the submissions made by the Applicant, the Commissions and the eligible persons referable to the contended factors.

112. In forming its judgement of whether the sound medical-scientific evidence pointed to the relevant association, the Council was conscious that the reasonable hypothesis test is 'a test of possibility'[224] and 'an unusually light burden'[225]. If the reasonable

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219 See full Federal Court decision at paragraph 49 per Branson J.

220 Relevant service here refers to operational, peacekeeping and hazardous service, and warlike and non-warlike service as those terms are defined in the VEA and the MRCA.

221 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.

222 See the full Federal Court decision in *Repatriation Commission v Bey* (1997) 79 FCR 364 which cited with approval these comments from Veterans' Review Board in *Stacey* (unreported 26 June 1985), all of which were in turn cited with approval in the Moore J decision at paragraph 33.

223 Relevant service here refers to eligible war service (other than operational service), defence service (other than hazardous service), and peacetime service as those terms are defined in the VEA and the MRCA.

224 See full Federal Court decision at paragraph 49 citing with approval Spigelman CJ in the New South Wales Court of Appeal decision at paragraph 111.

225 See full Federal Court decision at paragraph 55 per Branson J.

hypothesis test was found not to be satisfied, the balance of probabilities test necessarily could not be met.

SCOPE OF REVIEW

113. The Council decided to confine its attention to those matters identified in paragraph [33] above excluding consideration of that part of the electromagnetic spectrum covering ionising radiation. The Council considered the chemical exposure, dioxin, in its overall consideration of herbicides and pesticides.

114. When the Council initially considered Mr FR's written submissions it was not clear whether he contended for a factor for exposure to ionising radiation as well as for non-ionising radiation (as to the distinction, see further below at [131]). At the hearing of oral submissions, and in his written outline of submissions which he handed to the Council on that day, Mr FR made no contention in respect of a factor for ionising radiation, referring to ionising radiation only in support of his contentions in respect of EMR.[226]

115. Mr FR did not refer the Council to any sound medical scientific evidence in the information available to the RMA in respect of ionising radiation. The Commissions and other interested parties made no submissions on ionising radiation at all.

116. The Council therefore decided to exclude further consideration of ionising radiation from its Review, confirming the other factors and scope described at [33] above, that is:

> a) the possible inclusion rather than the existing exclusion in the kind of injury, disease or death set out in paragraph 2. (b) of both of the Statements of Principles of 'small lymphocytic lymphoma'; and
>
> of small lymphocytic lymphoma and B-cell chronic lymphoid leukaemia in the following ways:
>
> b) the possible inclusion of factors for which the Applicant and Eligible Persons who made submissions contend for:
>
> - Non-ionising Radiation;
>
> - Chemicals - includes all aromatic hydrocarbons – Benzene, Toluene, Xylene, Carbon Tetrachloride and others as contended; and
>
> - Aircraft fuel (AVGas) and associated chemicals (MEK, sealants) and exposure due to F111 fuel tank repairs;

[226] SMRC Oral Presentation Sydney 23/3/2011.

- Herbicides and Pesticides and Dioxin

- Asbestos; and

- Tobacco (by which the Council understands the Applicant to mean tobacco smoking)

POOL OF INFORMATION

117. As mentioned above, the first step for the Council was to determine the pool of information from the information that was available to (before) the RMA when it determined, amended, or last amended the Statements of Principles, as sent to the Council by the RMA under section 196K (in 2005 and subsequently in 2009).

118. As set out in paragraph [34] above, the Council's preliminary view was that the pool of information should comprise sound medical-scientific evidence as defined in section 5AB(2) of the VEA being information which:

- epidemiologists would consider appropriate to take into account; and
- in the Council's view, 'touches on' (is relevant to) 'contended factors' and has been evaluated by the Council according to epidemiological criteria, including the Bradford Hill criteria.

119. The Council's final view on the pool of information was that it should comprise the sound medical-scientific evidence the Council had identified on a preliminary basis (**Appendix A**). In reaching this decision the Council took into account the written submissions and complementary oral submissions and considered whether any of the information to which it was referred, should be in the pool. [227]

120. The Council noted the Applicant's and eligible persons' reference to articles and reports which were not available to (not before) the RMA (see **Appendix D, G, H**). As mentioned above, the Council is unable to (and so did not) consider information which was not available to (not before) the RMA at the relevant times.

[227] The Council's summaries of submissions includes footnotes for cited articles (both as available or new information) given emphasis (comment) by the person (or organisation) making the submission. The Council also includes in its Reasons for Decisions, Appendices that list the information relied upon by those making submissions, but limits those lists to articles commented upon (primary source citations) by those making submissions and does not include articles cited (secondary source citations) within articles.

COUNCIL'S INTRODUCTORY COMMENTS

Biology and Classification of Leukaemia

121. The Council noted that to understand the sound medical scientific evidence concerning chronic lymphoid leukaemias and particularly in considering the contentions of the Applicant, the Commissions and interested persons in respect of the inclusion of small lymphocytic lymphoma in the Statements of Principles concerning chronic lymphoid leukaemia, it is necessary to have some understanding of disease definition in the Statements of Principles, the biology, and the classification of the disease.

Chronic Lymphoid Leukaemia (as defined in the SoPs)

122. At paragraph 2 of Statement of Principles No. 5 of 2005 CLL is defined as:

> **Kind of injury disease or death**
>
> (b) **…"chronic lymphoid leukaemia"** means an indolent malignant neoplasm of B-lymphocytes or T-lymphocytes, characterised by proliferation and accumulation of morphologically mature-appearing but biologically immature lymphocytes in the blood, bone marrow and lymphoid tissue. This definition includes hairy cell leukaemia, adult T-cell leukaemia (ATLL) and chronic lymphocytic leukaemia, but excludes myeloma, Hodgkin's lymphoma, and all forms of non-Hodgkin's lymphoma including small lymphocytic lymphoma.
>
> (c) chronic lymphoid leukaemia attracts ICD-10-AM code C91.1, C91.3, C91.4 or C91.5.

123. The Council notes that the term CLL in common health professional usage refers to the common B-cell chronic lymphocytic leukaemia, rather than the other disease entities referred to above (at [122]) in the Statements of Principles definition (Hairy Cell Leukaemia, ATLL, and T-cell CLL). [228] In these reasons the Council uses B-cell CLL to refer to chronic lymphocytic leukaemia, and CLL when referring broadly to the various chronic lymphoid leukaemias.

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[228] Gilling, R n.d. *General discussion on etiology of selected cancer types – leukemia*, Public Health Assessment, PSC Resources, Massachusetts. Also see also at footnote 120.

> Adult T cell leukemia is a type of CLL caused by a virus, Human T-Cell Leukemia/Lymphoma Virus-I (HTLV-I) with a unique clinical and geographic profile and will not be discussed further in these reasons.

Traci, E et al. 2011, 'BRAF mutations in hairy-cell leukemia', *N Engle J Med*, vol. 364, no. 24, pp 2305–2315.

> Hairy Cell Leukaemia was first labelled as such in 1966 and it also has a unique clinical syndrome distinct from B-cell CLL and requiring different treatment. Very recent research identifies this disease with unique mutations of the BRAF gene, confirming its biological distinction from other chronic lymphoproliferative diseases. This too will not be discussed further in these reasons.

These two articles were not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

124. B-cell CLL is chiefly an adult disease; 90% of cases occur in people over 50 years old.[229].

125. Leukaemia is the term applied collectively to a group of cancers arising from specific stem cells, or the progeny of these, that normally give rise to the cells of the blood and lymph glands.

126. There are four major types into which most sub-types of leukaemia can be categorized (acute lymphoid leukaemia (ALL), chronic lymphoid leukaemia (CLL), chronic myeloid leukaemia (CML) and acute myeloid leukaemia (AML)). In some cases, within these categories, further discrete biological entities are recognised, eg acute promyelocytic leukaemia as a rare subtype of AML; or T-cell ALL compared with B-cell ALL. Epidemiologic and basic scientific research over the past twenty-five years has revealed patterns of incidence, risk factors and in many instances, causative or specific-disease associated chromosomal or DNA changes which vary for each biological subtype.

127. Chronic Lymphoid Leukaemias are clinically, aetiologically, epidemiologically, therapeutically and prognostically distinct from CML. In early epidemiological studies, these two entities were often aggregated as "chronic Leukaemia"; in the light of more modern understanding, such data is often poorly contributory.

128. The WHO Classification, introduced in 1994 as the REAL Classification, expanded, and serially revised since then, attempts to define discrete biological entities as closely as possible according to modern scientific understanding. Epidemiological and cancer registry leukaemia classification has lagged behind the WHO by some years.

The nature of Radiation

129. Radiofrequency (RF) electromagnetic radiation (EMR) is the transfer of energy by radio waves. RF EMR lies in the frequency range between 3 kilohertz (kHz) to 300 gigahertz (GHz). RF EMR is non-ionising radiation, meaning that it has insufficient energy to break chemical bonds or remove electrons (ionisation).

130. The Council notes that electromagnetic radiation is a broad term covering a wide spectrum of energies. The following Table may assist understanding: [230]

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[229] Accessed by the SMRC in May 2013 via http://www.cancer.org.au/about-cancer/types-of-cancer/leukaemia.html.

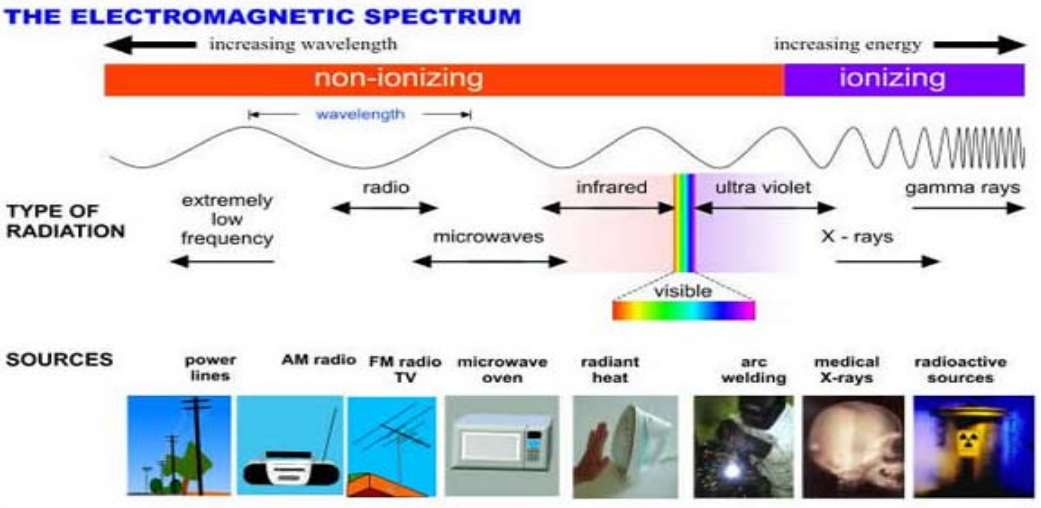
In 2009, 2576 people in Australia were diagnosed with leukaemia. Chronic lymphocytic leukaemia is the most common type of leukaemia in Australia; in 2009 1,062 people were diagnosed. There were 898 people diagnosed with acute myeloid leukaemia, 326 with acute lymphocytic leukaemia and 290 with chronic myeloid leukaemia. In 2007, there were 1,211 deaths due to these four cancer types. Acute myeloid leukaemia caused the most deaths, with 721 deaths in 2007. Chronic lymphocytic leukaemia caused 309 deaths, chronic myeloid leukaemia caused 92 deaths and acute lymphocytic leukaemia caused 89 deaths.

TABLE 1. Sources of electromagnetic radiation from low frequency power lines to radar units

Frequency	Designation	Examples
0–30 Hz	Sub-extremely low frequency	Direct current power lines
30–300 Hz	Extremely low frequency	Alternating current power lines, audio, submarine communications
0.3–3 kHz	Voice frequency	Voice and audio
3–30 kHz	Very low frequency	Long-range communications, navigation, audio
30–300 kHz	Low frequency	Radio navigation, marine communications, long-range communications
0.3–3 MHz	Medium frequency	AM* radio, radio navigation, amateur radio, communications, marine radiophone, industrial equipment
3–30 MHz	High frequency	CB* radio, amateur radio, international communications, medical diathermy, industrial equipment
30–300 MHz	Very high frequency	Television, FM* radio, amateur radio, air traffic control, industrial equipment, police/fire/emergency radio
300–3,000 MHz	Ultra high frequency	Radar, television, CB radio, amateur radio, radio navigation, microwave ovens, medical diathermy, cell phones, industrial equipment, police/fire/emergency radio
3,000–30,000 MHz	Super high frequency	Radar, satellite communication, amateur radio, police/fire radio, taxi dispatchers
30,000–300,000 MHz	Extremely high frequency	Radar, satellite communications, amateur radio, police/fire radio

* AM, amplitude modulation; CB, citizens' band; FM, frequency modulation.

131. The Council notes that radiation waves below a certain wavelength are non-ionising, ie lacking sufficient energy to cause an electron to leave an atom. This includes microwaves and radiowaves which may cause thermal injury to living tissues. Ionising radiation such as medical isotopes and radioactive decay sources may directly cause damage to cellular DNA. See table below: [231]



230 Groves, FD et al. 2002, 'Cancer in Korean War Navy technicians: mortality survey after 40 years', American Journal of Epidemiology, vol. 155, no. 9, pp. 810-818.

231 Source: www.arpansa.gov.au/radiationprotection/basics/ion_nonion.cfm, accessed by SMRC on 30 April 2013.

This information was not available to the RMA at the relevant times, and as new information was provided by Council only to illustrate the nature of the electromagnetic spectrum.

The nature of herbicides, pesticides, dioxin and potable water

132. The Council notes this data provided by the Commissions [232] to the Council by way of background in relation to military service and these exposures.

> Agent Orange and several other herbicide formulations used in Vietnam contained 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). An inadvertent contaminant in the manufacture of 2,4,5-T was 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD). The other constituents used in the colour-coded herbicides in Vietnam were 2,4-dichlorophenoxyacetic acid (2,4-D), cacodylic acid and picloram. These were not contaminated by TCDD.

133. In respect of Dioxin specifically the Commissions [233] submitted to the Council:

> A study commissioned by the Department of Veterans' Affairs (Australia) from the National Research Centre for Environmental Toxicology, published in 2002, examined the potential exposure of Royal Australian Navy personnel serving in Vietnam to dioxins and furans via drinking water. That study found that the evaporative distillation process used on Navy ships in the Vietnam war era to produce potable water did not remove, but rather would have enriched certain contaminants, including dioxins. That is, if dioxins, including TCDD, were present in the source water, their concentration would likely be increased in the potable water that was produced. The study did not test whether 2,4-D or 2,4,5-T could be similarly co-distilled. Unlike TCDD, these herbicides readily degrade in the environment.
>
> Australian Navy ships are known to have used estuarine water in Vietnam for evaporative distillation. Such water could have been polluted by TCDD consequent to herbicide spraying in Vietnam.

134. The Council notes that exposure to herbicides, pesticides and dioxin feature within the Statements of Principles regime for a number of diseases. The Commissions [234] also submitted:

> A number of RMA SOPs, such as those for non-Hodgkin's lymphoma and myeloma (but not the leukaemias) recognise being in Vietnam (during the Vietnam war) as a causal factor. SOPs that have that factor are now being progressively amended by the RMA to include "consuming potable water produced by evaporative distillation of estuarine Vietnamese waters".

Applicant MrAR also submitted[235] that existing factors in relation to ingesting potable water for Hodgkin's Lymphoma, non-Hodgkin's Lymphoma and myeloma, and in relation to existing factors, including that relating to 'service in Vietnam' for non-Hodgkin's Lymphoma should be included for CLL.

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232 Commissions 2006 January, Submission to the SMRC, at [72].

233 Commissions 2006 January, Submission to the SMRC, at [79] and [80].

234 Commissions 2006 January, Submission to the SMRC, at [81].

235 Oral hearing of 31 March 2011, transcript at p. 30.

…if you look at the factors for Non-Hodgkin's Lymphoma, including the service in Vietnam, and you consider that CLL and SLL are the same, either we should take factors from 28-2010 and put them into SOPs 9 and 10 or include CLL under SOP 28-2010.

135. The Council also notes, that the Non-Hodgkin's Statements of Principles, referred to by the Commission and contended by Applicant MrAR has in Statement of Principles 28 of 2010 existing factors for herbicides, pesticides and dioxin as follows: [236]

At p. 4 of 8 and [6] (q), (r) and (s);

(q) inhaling, ingesting or having cutaneous contact with a phenoxy acid herbicide from the specified list, for a cumulative period of at least 1000 hours, within a consecutive period of 10 years, before the clinical onset of non-Hodgkin's lymphoma, where the first exposure occurred at least five years before the clinical onset of non-Hodgkin's lymphoma, and where that exposure has ceased, the clinical onset of non-Hodgkin's lymphoma occurred

within 25 years after cessation; or

(r) being:
(i) on land in Vietnam, or
(ii) at sea in Vietnamese waters, or
(iii) on board a vessel and consuming potable water supplied on that vessel, when the water supply had been produced by evaporative distillation of estuarine Vietnamese waters,
for a cumulative period of at least 30 days, at least five years before the clinical onset of non-Hodgkin's lymphoma; or

(s) inhaling, ingesting or having cutaneous contact with a chemical agent contaminated by 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD), for a cumulative period of at least 1000 hours, within a consecutive period of 10 years, before the clinical onset of non-Hodgkin's lymphoma, where the first exposure occurred at least five years before the clinical onset of non-Hodgkin's lymphoma, and where that exposure has ceased, the clinical onset of non-Hodgkin's lymphoma occurred within 25 years after cessation;

the following chemicals are listed in respect of these existing factors for exposure to herbicides, pesticides and dioxin:

At pp. 5-7 of 8 and [9] Other definitions

"a phenoxy acid herbicide from the specified list" means:
(a) 2,4-dichlorophenoxyacetic acid (2,4-D);
(b) 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), or
(c) 2-methyl-4-chlorophenoxyacetic acid (MCPA);

"inhaling, ingesting or having cutaneous contact with a chemical agent contaminated by 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD)" means:

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236 RMA Statements of Principle 28 of 2010 for Non-Hodgkin's lymphoma, accessed by the SMRC from the RMA web site www.rma.gov.au Council notes that the contents of Statements of Principles for Hodgkin's Lymphoma and Myeloma have two or more factors in the same or similar terms to those of Non-Hodgkin's Lymphoma in relation to herbicides, pesticides, dioxin, being in Vietnam or Vietnamese waters or potable water.

(a) decanting or spraying;
(b) cleaning or maintaining equipment used to apply;
(c) being sprayed with;
(d) handling or sawing timber treated with;
(e) being in an environment shrouded in dust from timber treated with; or
(f) using cutting oils contaminated with one of the following chemicals:

(i) 2,4,5-trichlorophenoxyacetic acid;
(ii) 2,4,5-trichlorophenoxypropionic acid;
(iii) 2,4,5-trichlorophenol;
(iv) 2-(2,4,5-trichlorophenoxy)-ethyl 2,2-dichloropropionate;
(v) *o,o*-dimethyl-o-(2,4,5-trichlorophenyl)-phosphorothioate;
(vi) pentachlorophenol;
(vii) 2,3,4,6-tetrachlorophenol;
(viii) 2,4,6-trichlorophenol;
(ix) 1,3,4-trichloro-2-(4-nitrophenoxy)benzene;
(x) 2,4-dichloro-1-(4-nitrophenoxy)benzene; or
(xi) 2,4-dichloro-1-(3-methoxy-4-nitrophenoxy)-benzene;

THE COUNCIL'S ANALYSIS OF THE INFORMATION BEFORE THE RMA

136. The Council noted a number of issues with the information that was available to the RMA at the relevant times and how that information should be considered and weighted in both the first and second steps of the Council's task [see [108] and [109]).

137. The rarity of adult leukaemias is a problem for medical research. Generally, different leukaemia subtypes only account for a small percentage of cancer cases in epidemiological research. [237]

138. Other concerns faced by medical researchers are that:

e. B-cell CLL is frequently asymptomatic for many years before treatment, leading to under-diagnosis or late diagnosis of this disease in many subjects.

f. Some epidemiological studies assessing CLL incidence use only “death from CLL” (mortality), which will under-represent the incidence of the disease. Council noted that many will die with the disease, diagnosed or not, rather than from it.

g. Peripheral blood flow cytometry has become a diagnostic standard for B-cell CLL only relatively recently (since mid 1990’s[238]). previously the need for invasive bone marrow biopsy may have lead to under diagnosis.

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[237] Gillig, R n.d. *General discussion on etiology of selected cancer types – leukemia*, Public Health Assessment, PSC Resources, Massachusetts.

This article was not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

139. The diagnostic test for B-cell CLL has become a relatively simple blood test referred to as peripheral blood flow cytometry. This diagnostic test for B-cell CLL has replaced bone marrow biopsy and has either never been notifiable to population-based cancer registries in many countries, or not until recently in others.

140. These features of B-cell CLL – aggregation with other leukaemias, rarity, under-diagnosis, and under-reporting – greatly hamper epidemiological studies.

141. In addition, several methodological problems beset the scientific assessment of radiation exposure causing human cancers. These include:

- Poor or no individual exposure assessment. None of the radiation studies in medical-scientific evidence before Council used individual-subject dosimetry for radiation exposure which would be technically very challenging if not impossible. Early studies assumed exposure more or less intuitively by job title; later or better studies have measured exposure in certain jobs and assumed accumulative years of exposure from these measurements.

- Miss-classification bias due to the reasons outlined regarding leukaemia classification.

- Potential for publication bias - weighted either in favour of series with an increased incidence (ie positive findings more publishable) or in favour of no increase (especially when studies were sponsored by the industry under scrutiny).

- Many of the studies suffered a lack of statistical power with small numbers of subjects. This leads to wide confidence intervals around relative risks, with consequent difficulty in concluding the statistical significance of the result.

- Many case-control studies were reviewed. With respect to a number of exposures, these studies are prone to recall bias where cases (people with a disease, such as CLL) are more likely to recall exposures than control cases. This was not controlled for in any way in the studies examined.

- Electromagnetic studies have involved occupational, residential (proximity to known radiation sources such as high-powerlines of the Sutton Coldfield nuclear power station) and more recently mobile phone usage. Residential studies have generally not allowed for amount of time at home

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238 Erber, WN 2010, *Diagnostic techniques in hematological malignancies*, Cambridge Univercity press, New York.

This article was not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

versus away from home such as occupation, nor geographical mobility of the population and are regarded as frequently flawed.

– Exposures are more thoroughly sought in cases than in controls leading to a bias of evidence in cases versus controls.

– Ascertainment bias referring to the potential that in case controlled studies, the study cohort may have been more closely evaluated for the end point of interest i.e. in this case SLL/CLL (i.e. detailed questioning, reviews of hospital records etc) than the control group, leading to an apparent increase in incidence due to the increase in ascertainment rather than real differences in incidence.

Contended factors

142. Having settled the pool of information, the second question for the Council to consider was whether:

142.1. there is sound medical-scientific evidence in the pool of information that points to a potential 'contended factors' factor as a link or element in a reasonable hypothesis connecting chronic lymphocytic leukaemia to relevant service (see [109] a) and if so,

142.2. whether the relevant association exists on the balance of probabilities (See [109] b);

143. The Council considered the contended factors for the review in the following categories:

– small lymphocytic lymphoma and chronic lymphocytic leukaemia;

– Non-ionising radiation;
  – Radio frequencies RF and MW;
  – Extremely Low Frequency radiation ELF – EMR;

– Herbicides and Pesticides;
  – Dioxin;

– Potable Water;

– Solvents ;

– Benzene;

- Other Aromatic hydrocarbons;
- AV gas, MEK & Sealants assoc. with FIII fuel tank sealants;
- Asbestos; and
- Tobacco smoking

144. The Council considered all the evidence in the pool. However, given the large number of articles in the pool, the Council in these Reasons focuses upon its analysis of those articles that it considered most pertinent to the issues before it.

145. Ultimately, matters of weight are questions for the Council in the exercise of its expertise and scientific judgement, noting that the Councillors are appointed to a particular review because of their specialist expertise in the particular condition (in this case chronic lymphoid leukaemias).

COUNCIL'S ANALYSIS OF SALIENT ARTICLES / THE SOUND MEDICAL-SCIENTIFIC EVIDENCE IN THE POOL TOUCHING ON THE CONTENDED FACTORS

146. The Council took into account all the submissions made to it, both written and oral. [239] However, the Council's task is to determine

- whether there is sound medical-scientific evidence that was available to the RMA at the relevant times that 'points to' a relevant association, [240] and if so
- whether the sound medical-scientific evidence satisfies the balance of probabilities test. [241]

147. The Council having closely analysed all the information in the pool, placed particular weight on the articles discussed in detail below.

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239 From the Applicant Mr FR at [44] to [53].
From Applicant Mr AR at [54] to [59].
From Mrs F at [61] to [64].
From Mr G at [65] to [69].
From the Commissions at [71] to [105].

240 See [109].

241 See [109].

The possible inclusion rather than the existing exclusion in the kind of injury, disease or death set out in paragraph 2. (b) of both of the Statements of Principles of 'small lymphocytic lymphoma'

148. The Council has considered the contentions:

- of Mr FR that SLL is the same disease as B-cell CLL and should not be excluded from the Chronic Lymphoid Leukaemia Statements of Principles; and

- of Mr AR:

> …if you look at the factors for Non-Hodgkin's lymphoma, … and you consider that CLL and SLL are the same, either we should take the factors from 28-2010 [that is, concerning non-Hodgkin's lymphoma] and put them into SOPs 9 and 10 [of 2005, concerning CLL) or include CLL under the SOP 28-2010.

- of the Commissions' indicating a preference for grouping B-cell CLL and SLL in a single set of Statements of Principles, and inviting some guidance on whether the epidemiological evidence would allow for:

> …the NHL SOP broken up into more discrete entities as far as the epidemiology might allow. [242]

Small Lymphocytic Lymphoma (SLL) and B-cell CLL

149. The Council notes that the RMA has included small lymphocytic lymphoma in its definition of Non-Hodgkin's lymphoma. At paragraph 3 of Statement of Principles No. 28 of 2010:

> **Kind of injury disease or death**
>
> (b) For the purposes of this Statement of Principles, **"non-Hodgkin's lymphoma"** identifies a heterogeneous group of malignant lymphoproliferative diseases that originate from T and B lymphocytes, which lack Reed-Sternberg cells, and present as solid tumours of the immune system. This definition includes Burkitt's lymphoma, small lymphocytic lymphoma, mycosis fungoides, and non-Hodgkin's lymphoma arising within parenchymal organs, and excludes plasma cell malignancy, hairy cell leukaemia, Waldenström's macroglobulinaemia, and chronic lymphoid leukaemia.

150. Small lymphocytic Lymphoma (SLL) is a haematological malignancy in which biologically-identical cells to B-cell CLL are found in lymph node or bone marrow or other tissue biopsies, but unlike B-cell CLL, not the blood stream. Thus, CLL is now diagnosed with a blood test whereas SLL is generally diagnosed on tissue biopsy

[242] At p. 34 of Commissions oral submission complementing the written submission.

(lymph node or bone marrow). The Council noted that the numbers of SLL cases is small; SLL making up 6% [243] of NHL and CLL cases outnumbering SLL cases 9 to 1.

151. Further, that the diagnosis of SLL and CLL may be dependant on which tests (lymph node biopsy or a blood test/bone marrow biopsy) were performed in making the diagnosis. This has the potential consequence that either diagnosis does not necessarily preclude the other diagnosis, (depending on the results of the diagnostic blood tests). Any distinction between them for the purposes of determining whether the condition is connected with service is artificial.

Council's consideration of the available information on B-cell CLL/SLL

152. The Council acknowledged that the current Statements of Principles definition of Chronic lymphoid leukaemias (including Hairy Cell Leukaemia, ATLL, and T-cell CLL and excluding small lymphocytic lymphoma) and that for Non-Hodgkin's lymphoma (including small lymphocytic lymphoma) were consistent with previous classifications of the diseases. However, the Council is cognisant that the classifications of these diseases have changed, and the new, updated classifications appear in medical science studies.

153. In the 1994 original Revised European American Lymphoma (REAL) Classification[244], B-cell CLL and small lymphocytic lymphoma (also called small cell lymphoma) were first recognised as the same disease.

154. The subsequent revision of the REAL classification to become the WHO classification [245] confirmed the earlier recognition of B-cell CLL as the same disease as SLL.

155. In the 10th revision of the International Classification of Diseases, small cell B-cell lymphoma was classified as a Non-follicular lymphoma attracting Code C83.0. Lymphoid leukaemias were coded C91, with chronic lymphocytic leukaemia of B-cell type attracting code C91.1

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243 Armitage, JO & Weisenburger, DD 1998, 'New approach to classifying non-hogkin's lymphomas, clinical features of the major histological subtypes', *J of Clin Oncology*, vol. 16, no. 8, pp. 2780-2795.

This article was not available to the RMA at the relevant times, and so could only be considered by the Council as new information.

244 Harris, NL et al 1994, 'A revised European-American classification of lymphoid neoplasms: a proposal from the International Lymphoma Study Group', *Blood*, vol. 84, pp. 1361-1392.

245 Harris, NL et al. 1999 World Health Organization Classification of Neoplastic Diseases of the Hematopoietic and Lymphoid Tissues, Report of the Clinical Advisory Committee Meeting November 1997 - Airlie House, Virginia, *J Clin Oncol*, vol. 17, no. 12, pp. 3835 – 49.

Harris, NL et al. 2000, The World Health Organization classification of neoplasms of the hematopoietic and lymphoid tissues: report of the clinical advisory committee meeting - Airlie House, Virginia, November, 1997. The Hematology Journal, vol 1, pp. 53-66 and vol. 13, no. 2, pp. 193-207.

Harris, NL et al. 2001, *WHO classification of tumours of haematopoietic and lymphoid tissues*. In Jaffe, ES Harris, NL Stein, H and Vardiman, JW eds. 2001, *Pathology and genetics of tumours of haematopoietic and lymphoid tissue.* International Agency for Research on Cancer (IARC), IARC Press: Lyon, pp. 12-13, 109-117, 119-187, 189-203.

156. ICD-10 was endorsed by the Forty-third World Health Assembly in May 1990 and came into use in WHO Member States as from 1994. The 11th revision of the classification will continue until 2015. [246]

157. Thus, the World Health Organisation[247] classification of tumours of the haematopoietic and lymphoid tissues:

> …considers small lymphocytic lymphoma (SLL) to be consistent with chronic lymphoid leukaemia (CLL). SLL cases are restricted to cases with the tissue morphology and immunophenotype of CLL but are non-leukaemic (not present in the circulating, or peripheral, blood stream). (p. 127)

158. That position was considered in the 'Veterans and Agent Orange Update 2002' [248] which noted:

> In the proposed World Health Organization classification of non-Hodgkin's lymphoid neoplasms, CLL and its lymphomatous form, small lymphocytic lymphoma, are mature B-cell neoplasms (Jaffe et al., 2001). …
>
> Diffuse small-cell lymphocytic lymphoma is the term for the condition of patients with lymphomatous presentation of CLL. Patients seek medical attention for painless generalized lymphadenopathy that in many cases has lasted for several years. Unlike the situation in CLL, the peripheral blood may be normal or reveal only mild lymphocytosis. However, the bone marrow is positive in 75-95% of cases. Both small-cell lymphocytic lymphoma and CLL can transform into aggressive NHL known as Richter's syndrome … [249]

COUNCIL'S CONCLUSION ON SLL AND CLL

159. The Council concurs with the broad agreement in the scientific community that B-cell CLL) and SLL are the same disease with the same causes, natural history, response to treatment and prognosis.

160. In the Council's view after the change in the classifications system in 1994 and particularly in the last 10 years there has been a body of published medical science studies that provides data specific to B-cell CLL and SLL and on which the RMA

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246 http://www.who.int/classifications/icd/en/ accessed 25 June 2013.

247 Muller-Hermelink, HK et al. 2001: In , Jaffe ES, Vardiman JW, Stein H, et al. eds. 2001, *World Health Classification of Tumours: Pathology and genetics of tumours of haematopoietic and lymphoid tissues*, International Agency for Research on Cancer (IARC), IARC Press, Lyon

248 Institute of Medicine 2003, 'Veterans and Agent Orange Update 2002, The National Academic Press, Washington DC 2003. www.iom.edu/.../Veterans-and-Agent-Orange-Update-2002.aspx RMA ID 29493 FILEForce ID 19414

249 at page 283.

could have relied to amend the Statements of Principles for CLL to include SLL as the same disease, or to make new Statements of Principles for B-cell CLL and SLL as the same disease.

161. The Council was so satisfied on the basis of its collective medical-scientific expertise, confirmed by the information available to the RMA, particularly Harris, NL et al 1994, Harris, NL et al. 1999 Harris, NL et al. 2000 (see [153] and [154]). The Council found its conclusion was reinforced by new information. [250]

162. The Council's concurrence with the broad agreement in the scientific community that B-cell CLL and SLL are the same disease with the same causes, natural history, response to treatment and prognosis is relevant to its review of the information available to the RMA when it last decided not to amend the Statements of Principles concerning CLL.

163. As SLL was and remains classified in ICD-10am as a non-Hodgkin's lymphoma, the Council has paid careful attention to cases described as chronic lymphoid leukaemia or non-Hodgkin's lymphoma under ICD-10am or earlier ICD classifications, to determine whether the scientific evidence is relevant to the kind of injury, disease or death to which Statements of Principles 9 and 10 of 2005 apply, that is, to chronic lymphoid leukaemia excluding small lymphocytic lymphoma.

164. The Council noted that the heterogeneous nature of the thirty-plus different types of non-Hodgkin's lymphoma, has the consequence that any risk factor applicable to one non-Hodgkin's lymphoma subtype may not necessarily apply to another subtype. The Council has therefore not assumed that the medical scientific evidence applicable to any non-Hodgkin's lymphoma subtype necessarily applies to B-cell CLL / SLL.

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250 Swerdlow, SH et al. eds. 2008, WHO Classification of Tumors of Haematopoietic and Lymphoid Tissues, 4th Ed., International Agency for Research on Cancer, vol 2, no. 2, WHO Press, Geneva (not available to the RMA at the relevant times, and so could only be considered by the Council as new information).

This is the WHO classification which continues to recognise CLL and SLL as the same disease. SLL and B-cell CLL continue to be included in the same chapter of the current iteration.

Tsimberidou, AM et al. 2007 'Assessment of Chronic lymphocytic Leukaemia and Small Lymphocytic Lymphoma by absolute lymphocyte counts in 2,126 patients: 20 years of experience at the University of Texas M. D. Anderson cancer Center', Journal of Clinical Oncology, vol. 25, 4648-4656, accessed on 7 June 2009 for personal use by Constantine Tam at 60.242.134.127 (not available to the RMA at the relevant times, and so could only be considered by the Council as new information).

The Council noted this report, reviewing the experience of the M. D. Anderson Cancer Center in Houston, Texas, recognized internationally as a centre of excellence for research and treatment of B-cell CLL. This twenty year retrospective study analysed the clinical features of 2,126 consecutive subjects managed between 1985-2005, labelled as CLL compared to those labelled as SLL (by modern criteria of peripheral blood lymphocyte count less than 5 x 109/L). They found no differences in rates of response, survival (the overall median survival was approximately 10 years), and failure-free survival.

165. The Council was satisfied that there is sound medical-scientific evidence on which the RMA could have relied upon to amend the Statement of Principles to include small lymphocytic leukaemia as the same disease as chronic lymphocytic leukaemia (B-cell CLL).

166. The sound medical-scientific evidence on which the RMA would rely to determine the factors which would exist in a Statement of Principles that includes both B-cell CLL and SLL as the same disease, may be derived from information held by the RMA in relation to any of the haematopoietic cancers and any new information, hence the Council decided to direct the RMA to undertake an investigation.

167. The Council noted that the RMA has gazetted a full investigation (notice dated 27/10/2010) under section 196B(7) of the VEA) of Statements of Principles Nos. 9 and 10 of 2005 in respect of chronic lymphoid leukaemia and death from chronic lymphoid leukaemia and the RMA could amalgamate or incorporate the investigation directed by the Council, into that investigation.

Should There Be A Non-Ionising Radiation Factor?

Floderus, B 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*, vol. 4, pp 465 – 476. (RMA ID 1669, FILEForce ID 18822).

168. This retrospective case control study examined the occupation exposure to low frequency electromagnetic fields (EMF)[251] of 250 male leukaemia patients compared to 1,121 male controls from the mid region of Sweden diagnosed from 1983 – 1987.

169. Leukaemia cases were obtained from the cancer register and confirmed with hospital records and relatives where available. Information on occupational exposure from a self-completed work history questionnaire. Two controls per subjects were obtained from the census of 1980, matched on age.

170. Workplaces were identified from the questionnaires and electromagnetic field measurements obtained. The authors extrapolated EMF case exposure from these measurements and the job held the longest in the preceding decade before diagnosis.

171. EMF exposure was reported in quartiles of increasing level of exposure according to occupation with Q1 used as a reference value and 90th percentile exposure. 252

[251] EMF refers to electric and magnetic fields generated at 50 Hz or 60 Hz by electric power and also at 16 2/3 by electrified railways in some countries (including Sweden).

[252] See page 468.

172. The authors provided the following results for mean age adjusted relative risk of chronic lymphocytic leukaemia by EMF exposure for cases in job held longest during the 10 years before diagnosis.[253]

Q1	≤ 0.15 μT	OR = 1.0	
Q2	0.16 – 0.19 μT	OR = 1.1	95% CI 0.5 – 2.3
Q3	0.20 – 0.28 μT	OR = 2.2	95% CI 1.1 - 4.3
Q4	≥0.29 μT	OR = 3.0	95 % CI 1.6 – 5.8
P90	≥0.41 μT	OR = 3.7	95% CI 1.8 – 7.7
Time above 0.20 μT			
	≤ 16%	OR = 1.0	
	17 – 23 %	OR = 1.3	95% CI 0.7 – 2.5
	24 – 28%	OR = 1.2	95% CI 0.7 – 2.3
	≥ 29%	OR = 2.4	95 % CI 1.3 – 4.3
	≥39%	OR = 2.5	95% CI 1.3 – 5.0

173. The authors provided the following results for relative risk of chronic lymphocytic leukaemia by EMF exposure controlling for benzene, ionizing radiation, solvents and urban/rural residence. [254]

Benzene adjusted			
Q1	≤ 0.15 μT	OR = 1.0	
Q2	0.16 – 0.19 μT	OR = 1.2	95% CI 0.6 – 2.5
Q3	0.20 – 0.28 μT	OR = 2.5	95% CI 1.3 – 4.8
Q4	≥0.29 μT	OR = 3.3	95% CI 1.7 – 6.4
P90	≥0.41 μT	OR = 4.1	95% CI 2.0 – 8.5
Ionizing radiation adjusted			

[253] Table 3 page 470.

[254] Table 4 page 471.

Q1	≤ 0.15 μT	OR = 1.0	
Q2	0.16 – 0.19 μT	OR = 1.3	95% CI 0.6 – 2.7
Q3	0.20 – 0.28 μT	OR = 2.6	95% CI 1.3 – 5.1
Q4	≥0.29 μT	OR = 3.6	95% CI 1.8 – 6.1
P90	≥0.41 μT	OR = 4.7	95% CI 2.2 – 9.7
Solvents adjusted			
Q1	≤ 0.15 μT	OR = 1.0	
Q2	0.16 – 0.19 μT	OR = 1.1	95% CI 0.5 – 2.4
Q3	0.20 – 0.28 μT	OR = 2.5	95% CI 1.3 – 4.8
Q4	≥0.29 μT	OR = 3.0	95% CI 1.5 – 6.1
P90	≥0.41 μT	OR = 4.2	95% CI 1.9 – 9.4
No solvents			
Q1	& Q2	OR = 1.0	
Q3	0.20 – 0.28 μT	OR = 1.3	95% CI 0.5 – 3.5
Q4	≥0.29 μT	OR = 2.0	95% CI 0.8 – 4.9
P90	≥0.41 μT	OR = 2.6	95% CI 0.8 – 8.2
Possible solvents			
Q1	& Q2	OR = 0.8	95% CI 0.4 – 1.8
Q3	0.20 – 0.28 μT	OR = 2.4	95% CI 1.2 – 4.6
Q4	≥0.29 μT	OR = 2.8	95% CI 1.3 – 5.8
P90	≥0.41 μT	OR = 3.5	95% CI 1.5 – 7.9
Probable solvents			
Q1	& Q2	OR = 1.1	95% CI 0.3 – 3.9

Q3	0.20 – 0.28 µT	OR = 1.8	95% CI 0.2 – 15.5
Q4	≥0.29 µT	OR = 2.8	95% CI 1.4 – 5.7
P90	≥0.41 µT	OR = 4.3	95% CI 1.8 – 10.3
rural			
Q1	≤ 0.15 µT	OR = 1.1	
Q2	0.16 – 0.19 µT	OR = 1.5	95% CI 0.6 – 4.0
Q3	0.20 – 0.28 µT	OR = 2.6	95% CI 1.1 – 6.2
Q4	≥0.29 µT	OR = 3.4	95% CI 1.5 – 7.8
P90	≥0.41 µT	OR = 3.8	95% CI 1.5 – 9.4
urban			
Q1	≤ 0.15 µT	OR = 1.0	
Q2	0.16 – 0.19 µT	OR = 0.9	95% CI 0.3 – 2.8
Q3	0.20 – 0.28 µT	OR = 2.0	95% CI 0.7 – 5.3
Q4	≥0.29 µT	OR = 2.7	95% CI 1.1 – 7.1
P90	≥0.41 µT	OR = 5.5	95% CI 2.0 – 15.2

174. The authors provided the following results for age adjusted relative risk of chronic lymphocytic leukaemia by EMF exposure from census data [255]

Q1	≤ 0.15 µT		
	study participants	OR = 1.0	
	study and non respondents	OR = 1.0	
Q2	0.16 – 0.19 µT		
	study participants	OR = 1.0	95% CI 0.5 – 2.1
	study and non respondents	OR = 0.9	95% CI 0.5 – 1.6
Q3	0.20 – 0.28 µT		
	study participants	OR = 1.9	95% CI 1.0 – 3.6

[255] Table 8 page 474.

	study and non respondents	OR = 1.6	95% CI 0.9 – 2.7
Q4	≥0.29 µT		
	study participants	OR = 2.3	95% CI 1.2 - 4.3
	study and non respondents	OR = 1.6	95% CI 0.9 – 2.7
P90	≥0.41 µT		
	study participants	OR = 2.6	95% CI 1.3 – 5.4
	study and non respondents	OR = 1.7	95% CI 0.9 – 3.3

175. The authors concluded:[256]

> The findings of an association between EMF exposure and chronic lymphocytic leukaemia were based on the job held longest during the decade before diagnosis, which speaks in favor of a late effect.

Council's comments

176. The Council considered this was a persuasive study because exposure was related to chronic lymphocytic leukaemia specifically and a dose response effect was found.

177. In the Council's view this study provided some evidence of an association between EMF and chronic lymphocytic leukaemia, but there were some methodological weaknesses which limit the validity of this finding.

178. Problematically, there were a number of subsequent studies by the same group that failed to achieve statistical significance, thus not repeating that finding (Feychting, M et al. 1997, RMA 20987; Floderus, B et al. 1999, RMA 22475; Hakansson, N et al. 2002, RMA 26856). These will be discussed in detail below.

179. The Council considered that this study:

- points to a relevant association.

Feychting, M et al. 1997, 'Occupational and residential magnetic field exposure and Leukaemia and central nervous system tumors', *Epidemiology*, vol. 8, no. 4, pp. 384-389. RMA ID 20987 FILEForce ID 18904

180. This case control study examined 325 people with a diagnosis of leukaemia aged 16 years or more living within 300m of transmission lines in Sweden and occupational EMF exposure in a combined analysis during the period 1960 - 1985.

181. Exposure information was obtained from census data and previous measures of magnetic field exposure. Each case was matched with 2 controls

[256] Page 474

182. The authors provided data for

- relative risk of chronic lymphocytic leukaemia according to occupational exposure [257]

0.13 – 0.19 μT	RR = 1.4	95% CI 0.7 – 2.5
≥0.20 μT	RR = 1.9	95% CI 1.0 – 3.8

- relative risk of chronic lymphocytic leukaemia according to occupational exposure adjusted for age and sex [258]

0.13 – 0.19 μT	RR = 1.2	95% CI 0.7 – 1.9
≥0.20 μT	RR = 1.7	95% CI 1.0 – 2.9

- relative risk of chronic lymphocytic leukaemia according to residential exposure adjusted for age and sex, residents with occupational exposure ≥0.13 μT were excluded [259]

0.1 – 0.19 μT	RR = 0.9	95% CI 0.4 - 2.1
≥0.20 μT	RR = 0.8	95% CI 0.3 – 1.8

- relative risk of chronic lymphocytic leukaemia according to occupational and residential exposure adjusted for age and sex [260]

Only residential exposure (Occupational exposure <0.2 μT and residential exposure ≥0.20μT)	RR = 0.8	95% CI 0.3 - 2.3
Only occupational exposure (Occupational exposure ≥0.20 μT and residential exposure <0.2 μT)	RR = 1.5	95% CI 0.8 – 2.7
Both residential and occupational exposure (Occupational exposure ≥0.20 μT and residential exposure ≥0.20 μT)	RR = 2.1	95% CI 0.4 – 10.4

183. The authors concluded: [261]

> Our data provide some support for the hypothesis of an association between occupational magnetic field exposure and leukemia. Workers in occupations exposed to magnetic fields

[257] Table 1 page 386.

[258] Table 2 page 387.

[259] Table 3 page 387.

[260] Table 4 page 388.

[261] Page 387.

were at higher risk of AML and chronic lymphocytic leukaemia than workers in unexposed occupations.

184. The authors noted the small numbers of cases were a limitation of their study.

Council's comments

185. The Council considered this study provided mixed evidence about any association between chronic lymphocytic leukaemia and exposure to EMF. The relative risks for occupational exposures (see [182]) whilst including unity (1.0) do suggest a dose response effect.

186. There were some limitations to the study. As it was confined to persons residing close to high voltage power lines, the extrapolation to other settings may be limited. There were some weaknesses in measurement of EMF and no attempt was made to measure EMF exposure from sources inside the person's house. The imputed measurement of exposure from the power lines was not well justified, and the fact that it was based on exposure for a minimum of one year at any time during the period studied and prior to diagnosis means that the exposure is possibly not relevant to an association with chronic lymphocytic leukaemia.

187. In addition, the occupational classification of EMF exposure is known to be imprecise as so many possible sources of exposure are not measured leading to considerable misclassification. No data was published on whether the measurement of residential exposure was obtained blind to case control status.

188. The increased odds ratio of chronic lymphocytic leukaemia associated with higher EMF exposure was only of borderline statistical significance, and was present only for occupational exposure.

189. Nevertheless, overall and despite these methodological weaknesses, the Council considered that this study:

- points to a relevant association.

Floderus, B et al. 1999, 'Occupational magnetic field exposure and site-specific cancer incidence: a Swedish cohort study,' *Cancer Causes & Control*, vol. 10, pp. 323-332. RMA ID 22475 FILEForce ID 18551

190. This case control study of 1,596,959 men and 806,278 women examined the association of site specific cancer in relation to occupational magnetic field exposure from 1971 to 1984 in Sweden.

191. Exposure was assessed from census information non occupations that were linked to a job exposure matrix based on measurements.

192. The authors reported

– relative risk for chronic lymphocytic leukaemia in relation to occupational magnetic field exposure among men during 1971 – 1977 and 1971 – 1984 [262]

1971 - 1977		
Medium exposure 0.084 – 0.115 μT	RR = 0.8	95% CI 0.6 – 1.0
High exposure ≥0.116 μT	RR = 1.2	95% CI 0.9 – 1.5
1971 - 1984		
Medium exposure 0.084 – 0.115 μT	RR = 0.9	95% CI 0.7 – 1.0
High exposure ≥0.116 μT	RR = 1.1	95% CI 0.9 - 1.2

– relative risk for chronic lymphocytic leukaemia in relation to occupational magnetic field exposure among women during 1971 – 1977 and 1971 – 1984 [263]

1971 - 1977		
Medium exposure 0.084 – 0.115 μT	RR = 2.1	95% CI 1.2 – 3.7
High exposure ≥0.116 μT	RR = 1.8	95% CI 1.0 – 3.1
1971 - 1984		
Medium exposure 0.084 – 0.115 μT	RR = 1.6	95% CI 1.0 – 2.3
High exposure ≥0.116 μT	RR = 1.7	95% CI 1.2 – 2.4

193. The authors concluded: [264]

> Observed associations were weak and there were no evident exposure-response relationships. For women, associations were seen for cancer of the lung, breast, corpus uteri, malignant melanoma and chronic lymphocytic leukaemia.

Council's comments

194. The Council considered that this was not a study of chronic lymphocytic leukaemia but of many cancers and with many comparisons of the data collected. There was a weak statistically significant result for medium and high exposure for women (RR = 1.6 and 1.7 respectively above) which was not reflected in males, however the study used job title as a surrogate measure of exposure.

195. The Council considered this study:

– did not point to, but merely left open the possibility of a relevant association.

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[262] Table 2 page 327.

[263] Table 3 page 329.

[264] Page 323.

Hakansson, N et al. 2002, 'Cancer incidence and magnetic field exposure in industries using resistance welding in Sweden', *Occup Environ Med*, vol. 59, no. 7, pp. 481-486. RMA ID 26856 FILEForce ID 18620

196. This retrospective cohort study of 537,692 mean and 180, 529 in the engineering industry examined the association between exposure to high levels of extremely low frequency magnetic fields (ELF-MF) and cancer incidence from 1985 – 1994 in Sweden.

197. Occupation data was obtained from census information linked to a ELF-MF job exposure matrix.

198. The cohort was matched against the cancer registry for the years 1985 – 1994.

199. The authors provide the following results for relative risk of chronic lymphocytic leukaemia in relation to occupational ELF-MF exposure for men and women. 265

Men (n = 39 for medium; 5 for high; and 5 for very high)			
Medium exposure	0. 164– 0.250 μT	RR = 1.0	95% CI 0.6 – 1.8
High exposure	0.250 – 0.530 μT	RR = 0.6	95% CI 0.2 – 1.6
Very high exposure	> 0.530 μT	RR = 1.1	95% CI 0.4 – 1.1
Women (n = 3 for medium; 3 for very high)			
Medium exposure	0. 164– 0.250 μT	RR = 1.4	95% CI 0.6 – 3.3
High exposure	0.250 – 0.530 μT	RR = 1.9	95% CI 0.7 – 4.9
Very high exposure	> 0.530 μT	RR = 4.6	95% CI 1.3 – 15.9

200. The authors concluded: [266]

> We did not see an increased risk for leukaemia among men, while there were increased risks among exposed women, but based on small numbers [trend p = 0.120].

Council's comments

201. The Council considered a strength of this study was that it attempted to define a group who were truly likely to have very high levels of EMF exposure. The results were different for men (no association with high exposure) and women (elevated risk which was only statistically significant for very high exposure). There is likely to

265 Table 2 page 483.

266 Page 485.

remain substantial misclassification because of unmeasured inter-individual variation in exposure. The study population was relatively young with some 50% under the age of 50 years and results are based on very small numbers of cases.

202. In the Council's view this study provided some evidence of:

- a lack of association between chronic lymphocytic leukaemia and exposure to medium to very high levels of extremely low frequency magnetic fields.

Theirault, G et al. 1994, 'Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec, Canada, and France:1970-1989', *Am J Epidemiol*, vol. 139, no. 6, pp. 550-572. RMA ID 10435 FILEForce ID 18845

203. This case control study with 3 nested cohorts examined the association between cancer and electric utility workers from 1970 – 1989 in Ontario and Quebec Canada and France. The authors examined exposure to magnetic fields in the 50-60MHz range.

204. The study cohorts were:

a. Electricite de France – Gaz de France: 170, 000 men;

b. Ontario Hydro: 31, 543 men; and

c. Hydro Quebec: 21, 749 men.

205. Cases were identified from company medical files, cancer registries and death certificates. Cases were validated through consultation with physicians at the health centres. Controls were other employees form the same utility matched to the case on year of birth.

206. Exposure was assessed indirectly by combining information from the workers occupational histories and estimates for each job they held.

207. Assessment of magnetic fields was obtained by measuring current levels of exposure and estimating past levels.

208. The authors provided the following adjusted odds ratio for chronic lymphocytic leukaemia among electric workers from all three cohorts combined according to MF exposure. Median exposure 3.1 μT years and 90th percentile of exposure 15.7 μT years. [267]

> median (n = 24)	OR = 1.48	95% CI 0.50 – 4.40

[267] Table 3 page 560.

>90th percentile (n = 6)	OR = 1.71	95% CI 0.44 – 6.66

209. The authors provided the following adjusted odds ratio for chronic lymphocytic leukaemia among electric utility workers from each of the three cohorts according to cumulative MF exposure. Median exposure 3.1 μT years and 90th percentile of exposure 15.7 μT years. [268]

Quebec		
> median	OR = 0.25	95% CI 0.02 – 2.56
>90th percentile	OR = 0.27	95% CI 0.02 – 4.22
Ontario		
> median	OR = 2.14	95% CI 0.39 – 11.64
>90th percentile	OR = 3.67	95% CI 0.40 – 50.07
France		
> median	OR = 4.79	95% CI 0.45 – 70.57
>90th percentile	OR = 2.82	95% CI 0.04 – 67.95

210. The authors provided the following adjusted odds ratio for chronic lymphocytic leukaemia among electric utility workers from all three cohorts combined according to cumulative MF exposure and time of exposure in relation to diagnosis. Median exposure 3.1 μT years and 90th percentile of exposure 15.7 μT years. [269]

0-5 years		
> median	OR = 4.06	95% CI 0.74 – 22.38
>90th percentile	OR = 3.25	95% CI 0.37 – 28.6
0 – 20 years		
> median	OR = 4.07	95% CI 0.90 – 18.02
>90th percentile	OR = 4.03	95% CI 0.76 – 21.35
> 20 years		
> median	OR = 0.62	95% CI 0.09 – 4.45
>90th percentile	OR = 0.73	95% CI 0.06 – 8.33
ALL		
> median	OR = 1.36	95% CI 0.41 – 4.46
>90th percentile	OR = 1.74	95% CI 0.38 – 7.89

[268] Table 4 page 562.

[269] Table 5 page 563.

211. The authors provided the following adjusted odds ratio for chronic lymphocytic leukaemia among electric utility workers by cumulative MF exposure. [270]

0 - 3.09 μT years	OR = 1.0	
3.1 – 6.89 μT years	OR = 1.11	95% CI 0.31 – 3.92
6.9 – 15.69 μT years	OR = 2.18	95% CI 0.58 – 8.27
> 15.7 μT years	OR = 1.71	95% CI 0.44 – 6.66

212. The authors provided the following adjusted odds ratio for chronic lymphocytic leukaemia among electric utility workers by mean MF exposure. [271]

< 0.2 cf ≥ 0.2μT	OR = 1.40	95% CI 0.52 – 3.77
< 0.3 cf ≥ 0.3μT	OR = 0.89	95% CI 0.35 – 2.29

213. The authors reported finding no association with magnetic field exposure and chronic lymphocytic leukaemia.

Council's comments

214. This study found no evidence of a significant association between chronic lymphocytic leukaemia risk and cumulative exposure to EMF (eg OR in those with ≥ 90th percentile compared to those with ≤ median exposure was 1.71, 95% CI 0.44-6.66). This was a positive, but not significant association. There was also no association between chronic lymphocytic leukaemia risk and mean exposure (eg OR in those with ≥0.3μT compared to those with ≤ 0.2μT exposure was 0.89, 95% CI 0.35-2.29).

215. The Council considered that this study:

- does not point to but merely leaves open the possibility of a relevant association.

Harrington, JM et al. 2001, 'Leukaemia mortality in relation to magnetic field exposure: findings from a study of United Kingdom electricity generation and transmission workers, 1973-97', *Occup Environ Med*, 1 May, vol. 58, no. 5, pp. 307-314. RMA ID 25808 FILEForce ID 18484

216. This retrospective cohort study investigated the association between leukaemia mortality and magnetic field exposure in 83,997 employees (72 954 men and 11 043 women) of the former central electricity generating board of England and Wales from the period 1973 – 1997.

[270] Table 6 page 565.

[271] Table 7 page 566.

217. Cases employed for at least 6 months were selected according to work history and personnel records available on the computerized personnel information system.

218. Details of the estimation of exposure to magnetic fields according to location of employment and type of activity were provided.

219. The authors provide the relative risk of mortality from chronic lymphocytic leukaemia (n = 39 deaths) by levels of estimated cumulative MF exposure. Adjusted RR 1(RRadj1) for sex and attained age, adjusted RR 2(RRadj2) for sex, attained age, calendar period, year of starting employment. [272]

0 – 2.4 µT per year	RRadj1 = 1.0	
	RRadj2 = 1.0	
2.5 – 4.9 µT per year	RRadj1 = 0.64	95% CI 0.19 – 2.17
	RRadj2 = 0.69	95% CI 0.20 – 2.39
5.0 – 9.9 µT per year	RRadj1 = 0.59	95% CI 0.22 – 1.57
	RRadj2 = 0.67	95% CI 0.24 – 1.89
10.0 – 19.9 µT per year	RRadj1 = 0.92	95% CI 0.39 – 2.18
	RRadj2 = 1.07	95% CI 0.41 – 2.78
≥ 20µT per year	RRadj1 = 1.14	95% CI 0.34 – 3.86
	RRadj2 = 1.40	95% CI 0.39 – 4.98

220. The authors provide the relative risk of mortality from chronic lymphocytic leukaemia (n = 28 deaths) by levels of estimated MF exposure received in the preceding 5 years. Adjusted RR 1(RRadj1) for sex and attained age, adjusted RR 2(RRadj2) for sex, attained age, calendar period, year of starting employment. [273]

0 µT per year	RR = 1.0	
0.01 – 0.49 µT per year	RRadj1 = 1.47	95% CI 0.56 – 3.86
	RRadj2 = 1.43	95% CI 0.53 – 3.87
≥ 50µT per year	RRadj1 = 0.32	95% CI 0.04 – 2.42
	RRadj2 = 0.30	95% CI 0.04 – 2.32

[272] Table 6 page 311.

[273] Table 8 page 312.

221. The authors concluded: [274]

> No significant positive trends were found for the risks of various types of leukaemia (chronic lymphocytic leukaemia, AML, CML, all leukaemia) either with lifetime cumulative exposure to magnetic fields or with such exposures received in the most recent 5 years.

Council's comments

222. The Council considered this was an important result against an association, with no association found for chronic lymphocytic leukaemia for any dose level and because the methodology was based upon some measurement of occupational exposure and extrapolated to a larger group. Using death data only however, is a major methodological limitation leading to underestimation of chronic lymphocytic leukaemia cases.

223. The Council considered that this study provided some evidence:

- against a relevant association.

Savitz, DA & Loomis, DP 1995, 'Magnetic field exposure in relation to leukemia and brain cancer mortality among electric utility workers', *Am J Epidemiol*, vol. 141, no. 2, pp. 123-134. RMA ID 1706 FILEForce ID 18819

224. This historical cohort mortality study of 138,905 men employed at 5 large electric power companies in the US between 1950 and 1986 examined the relationship between electromagnetic field exposure and leukaemia and brain cancer mortality.

225. Cohort participants were identified from personnel records and mortality data was obtained from the national death index via social security numbers. Cause of death was obtained from death certificates.

226. Exposure data was estimated by linking individual work histories to data from workshift magnetic field measurements.

227. Mortality data was compared to the US population.

228. The authors reported the following standardized mortality ratios for leukemia and aleukaemia in the cohort of US utility workers. [275]

164 deaths	SMR = 0.76	95%CI 0.64 – 0.88

229. The authors reported the following adjusted mortality rate ratios for leukemia by duration of employment in selected occupations[276]

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[274] Page 307.

[275] Table 2 page 128.

[276] Table 3 page 129.

Duration of employment < 5 years (82 deaths)		
Exposed occupations	RR = 1.0	
Lineman	RR = 1.0	
Electrician	RR = 1.0	
Power plant operator	RR = 1.0	
Duration of employment 5 - < 20 years (54 deaths)		
Exposed occupations	RR = 1.31	95% CI 0.87 – 1.97
Lineman	RR = 0.53	95% CI 0.23 – 1.23
Electrician	RR = 1.35	95% CI 0.62 – 2.94
Power plant operator	RR = 1.10	95% CI 0.92 – 1.30
Duration of employment > 20 years (28 deaths)		
Exposed occupation	RR = 1.00	95% CI 0.60 – 1.65
Lineman	RR = 0.68	95% CI 0.25 – 1.86
Electrician	RR = 2.50	95% CI 1.08 – 5.76
Power plant operator	not provided	

230. The authors provide the risk ratio of chronic lymphocytic leukaemia by magnetic field exposure [277]

Total exposure (34 deaths)		
0-<0.6 μT-years	RR = 1.00	
0.6 - <1.2 μT-years	RR = 1.33	95% CI 0.49 – 3.63
1.2 - <2.0 μT-years	RR = 1.98	95% CI 0.77 – 5.09
2.0 - <4.3 μT-years	RR = 0.55	95% CI 0.17 – 1.82
Per μT-years	RR = 0.96	95% CI 0.78 – 1.09
Past 2-10 years (34 deaths)		
0-<0.6 μT-years	RR = 1.00	

[277] Table 5 page 131.

>0 - <0.2 μT-years	RR = 1.31	95% CI 0.54 – 3.18
0.2 - <0.4 μT-years	RR = 1.08	95% CI 0.38 – 3.07
Per μT-years	RR = 1.47	95% CI 0.52– 4.20
Past 10 – 20 years (34 deaths)		
0 μT-years	RR = 1.00	
>0.0 - <0.3 μT-years	RR = 1.35	95% CI 0.46 – 3.92
0.3 - <0.5 μT-years	RR = 1.82	95% CI 0.61 – 5.41
≥0.5 μT-years	RR = 1.02	95% CI 0.32 – 3.25
Per μT-years	RR = 0.67	95% CI 0.44 – 1.74
> 20 years (34 deaths)		
0μT-years	RR = 1.00	
0 - <0.4 μT-years	RR = 1.41	95% CI 0.44 – 4.52
0.4 - <1.1 μT-years	RR = 1.27	95% CI 0.38 – 4.24
≥1.1 μT-years	RR = 0.66	95% CI 0.18 – 2.45
Per μT-years	RR = 0.92	95% CI 0.75 – 1.12

231. The authors concluded: [278]

> Mortality from most causes, including leukemia and brain cancer, was reduced in this cohort of electric utility workers relative to the US population In comparisons among electric utility workers, leukemia mortality was somewhat increased among men who worked as electricians, but not among linemen, power plant operators or exposed occupations in general. Quantitative indices of magnetic field exposure gave little indication of an increased risk with increasing exposure for leukemias in the aggregate, nor was there clear evidence of an increased risk with increasing exposure for acute myeloid or chronic lymphocytic leukaemia.

Council comments

232. The Council notes no dose effect was seen.

233. This study provides moderate evidence against there being an association between chronic lymphocytic leukaemia risk and EMF exposure, but the number of cases of chronic lymphocytic leukaemia was relatively small (n = 34), based upon mortality only and this limited the value of the study results.

[278] Page 129.

234. The Council considered that this study provides moderate evidence:

– against a relevant association.

Groves, FD et al. 2002, 'Cancer in Korean War Navy technicians: mortality survey after 40 years', *American Journal of Epidemiology*, vol. 155, no. 9, pp. 810-18. RMA ID 25344 FILEForce ID 18500

235. This retrospective cohort study examined the all cause mortality of 40,890 Navy veterans of the Korean War from 1950 – 1954 with potential exposure to high-intensity radar.

236. The cohort death rates were compared with mortality rates for white US men using standardized mortality ratios and the death rates for men in occupations considered a priori to have high radar exposure were compared with the rates for men in low exposure occupations.

237. Mortality data were obtained from records of the American Department of Veterans Affairs.

238. The authors provided the standardized mortality ratios for lymphocytic leukaemia without distinction between acute or chronic according to radar exposure potential. [279]

Low radar exposure potential n = 17	SMR = 1.31	95% CI 0.81 – 2.11
High radar exposure potential n = 16	SMR = 1.12	95% CI 0.69 – 1.83
Total cohort n = 33	SMR = 1.21	95% CI 0.86 – 1.70

239. The authors provided relative risks of chronic lymphoid leukaemia for men (n = 9) with low exposure potential compared to men (n = 11) with high radar exposure potential. [280]

RR = 1.08	95% CI 0.44 – 2.66

240. The authors provided relative risks of chronic lymphoid leukaemia for men involved in repair of radar or electrical systems compared to radio and radar operators. [281]

Aviation electrician mates (low exposure) n = 1	RR = 1.06	95% CI 0.12 – 9.25

[279] Table 3 page 814.

[280] Table 4 page 815.

[281] Table 5 page 816.

Electronics technician (high exposure) n = 7	RR = 1.13	95% CI 0.41 – 3.16
Fire control technician (high exposure) n = 1	RR = 0.54	95% CI 0.06 – 4.55
Aviation electronic technician (high exposure) n = 3	RR = 1.41	95% CI 0.35 – 5.70

241. The authors concluded: [282]

> …for the occupations with high potential for radar exposure, no significant excesses were found for all malignant neoplasms combined lymphoid malignancies, brain cancer or testicular cancer.

242. The authors overall conclusion [283] was:

> Radar exposure had very little effect on mortality in this cohort of US Navy veterans.

Council's comments

243. The Council noted that this paper is in fact a further follow up of the Robinette, CD et al. 1980 [284] study of 20,000 US Navy personnel.

244. In the Council's view the critical comparison[285] is between the high radar and the low radar exposure groups. Men in the high radar exposure group had significant reductions in total mortality (All causes RR = 0.87), deaths from cancer (RR 0.80), and within cancers, deaths from lung and related cancers (RR 0.73). A lack of any dose response effect argues against an association.

245. For chronic lymphocytic leukaemia there were 9 cases with low exposure potential and 11 with high exposure potential (RR 1.08, CI 0.44 - 2.66). These low case numbers make any conclusions for B-cell CLL difficult.

246. The Council considered flaws in this study included that the actual exposure levels are unknown, and RF exposures may have been substantial in both groups. There is no information available on lifestyle confounding factors such as smoking or on the exposures or occupations of this group after their service experience. In addition, the study relied upon mortality.

247. The Council considered this an important study:

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282 Page 818.

283 See page 810.

284 Robinette, CD Silve, C and Jablon, S 1980, 'Effects upon health of occupational exposure to microwave radiation (Radar)', Am Journal of Epidemiology, vol. 112, no. 1, pp. 39-53.

285 See Table 4, p. 815.

– weakly negative for a relevant association.

Szmigielski, S 1996, 'Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation', The Science of the Total Environment, vol 180, pp. 9-17. RMA ID 10413 FILEForce ID 18848

248. The retrospective case control study compared cancer morbidity in the whole population of military career personnel in Poland during the 15 year period 1971-1985, comparing subjects exposed occupationally to radiofrequencies (RF) and microwaves (MW) (observed rates) to morbidity rates in the non-exposed groups (expected rates).

249. Morbidity rates were calculated annually for the whole population and divided into four age groups (20-29 , 30-39, 40 – 49, and 50-59).

250. Morbidity data was obtained from central and regional military hospital records and the central military medical board.

251. Exposure data was obtained from military data related to occupation and records of RF/MW field intensity measurements at and around service posts. Out of a mean of 128,000 subjects per year about 3% were considered as occupationally exposed to RF / MW [286].

252. RF monitoring was carried out routinely in areas where RF-emitting equipment was used. Only annual group data relating to the number of individuals by group, type of service and other exposures, were available on the cohort. These were used to calculate cancer rates. No individual monitoring was carried out.

253. The authors obtained the following incidence for cancer of 2.07, $p<0.05$ observed vs expected, and of haematopoietic system and lymphatic malignancies (per 100,000 subjects annually) and odds ratio observed / expected ratio. [287]

Incidence / 100,000	43.12	OR = 6.31	95% CI 3.12 – 14.32

254. The authors obtained the following incidence of chronic lymphocytic leukaemia (per 100,000 subjects annually) and odds ratio observed / expected ratio. [288]

Incidence / 100,000	5.04	OR = 3.68	95% CI 1.45 – 5.18

255. The authors obtained the following incidence of haematopoietic system and lymphatic malignancies (per 100, 000 subjects annually) and odds ratio observed / expected ratio per aged group. [289]

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[286] See Abstract at page 9.

[287] See Table 1 at page 12.

[288] See Table 2 at page 14.

AGE	INCIDENCE / 100,000	ODDS RATIO	95% CI
20 – 29	17.30	8.16	3.11 – 22.64
30 – 39	26.43	8.58	3.46 – 19.58
40 – 49	73.25	8.80	4.13 – 15.27
50 – 59	108.62	4.47	2.56 – 6.81

256. The authors concluded: [290]

> Cancer morbidity rates are high enough to prove that the incidence of all malignancies is doubled in RF/MW-exposed personal, compared to their unexposed colleagues of the same socio-economic status and living under similar environmental and working conditions. This difference is due to higher morbidity rates of malignancies of the haemopoietic system and lymphatic organs.
>
> The most frequent type of this form of malignancy in the RF/MW-exposed group appeared to be non-Hodgkin's lymphoma and lymphosarcoma and chronic lymphocytic leukaemia, both developing mainly in the 40-49 and 50-59 years.

Council's comments

257. In the Council's view 5 cases per 100,000 subjects is not much different from the general population expectation of chronic lymphocytic leukaemia, with the general population data for Australia / United States of America being in the order of (males and females) 4 in 100,000.

258. Council notes the study has been criticised in the literature as follows:

- having potential for systemic bias due to exposure only being recorded in cancer cases;
- lack of detail with individual age information on these records; and
- lack of adjustment for age with discrepant variations. These latter however, could perhaps explain the 10-year age groupings and may explain both the low expected rate and the differences seen between the exposed and the non-exposed groups (Krewski, D et al [291]). Alternatively, this could be partly attributed to a "healthy worker" effect (ie those in full-

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289 See Table 3 at page 14

290 Page 13-15

291 Krewski, D et al. 2001a – See [455.g]

time work are generally healthier than those who are not), but this is unlikely to explain the result entirely.

– finally, any cancers that developed after military service were not detected in this study.

259. The Council considered this a deeply flawed study with low cancer rates and few incident cases involved for the statistical analysis (n = 3 for chronic lymphocytic leukaemia). Further, health records of exposed personnel were collected, but the methodology used to calculate rates and RRs is unclear, as there are more cases than controls and no case numbers.

260. In the Council's view exposure was not well measured as exposure was based on data collected by military safety groups of RF / MW field intensities at and around service locations. Further, the authors state that individual exposure (daily or monthly exposure 'dose') was difficult to assess as exposure was variable and frequent short-lasting incidents of overexposure occurred frequently.

261. Council concluded that this study:

– did not point to, but merely left open the possibility of a relevant association.

Dolk, H et al. 1997a, 'Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield Transmitter', *American Journal of Epidemiology*, vol. 145, no. 1, pp. 1-9. RMA ID 9620 FILEForce ID 18858.

262. This retrospective cohort analysis examined the cancer incidence for residents age 15 years and older in a 10 km radius of the Sutton Coldfield transmitter at the northern edge of the city of Birmingham from 1974 – 1996.

263. The authors identified 17,409 incident adult cancer cases diagnosed between 1974 and 1986 in a population of approximately 408,000 living within 10km of the Sutton Coldfield transmitter, compared to 16,861 expected. This was a 3% excess, which was not significant after adjusting for socioeconomic status, showed no trend with increasing distance from the transmitter. The ratio of observed to expected cancer cases living within 2 km of the transmitter was 1.09.

264. The authors reported that the only specific cancer types to show an excess risk with proximity to the tower were the leukaemias (O/E ratio 1.83, 95% confidence interval 1.22 – 2.74, based on 23 observed vs. 12.59 expected within 2 km of the tower). A decline in risk with distance from the tower was also reported, with the observed numbers in the more distant areas falling below the numbers expected.

265. The authors provided the following risk ratio for chronic lymphocytic leukaemia (8 cases were observed versus 3.12 expected within 2km, the figures were 96 versus

72.56 respectively within 10km) according to distance of residence from the transmitter. [292]

Distance 0.2 Km	OR = 2.56	95% CI 1.11 – 5.05
Distance 0-10Km	OR = 1.32	95% CI 1.08 – 1.62

266. The authors provided the following risk ratio for chronic lymphocytic leukaemia according to distance of residence from the transmitter without confidence intervals. [293]

Distance 0-2 Km	OR = 2.56
Distance 2 – 4.9 Km	OR = 2.31
Distance 4.9 – 7.4 Km	OR = 1.27
Distance 7.4 – 10 Km	OR = 1.12

267. The authors concluded, without reference to chronic lymphocytic leukaemia. [294]

> The main finding was the confirmation of a reported excess of leukemias near the Sutton Coldfield radio and television transmitter, and a decline in risk with distance from the site.

Council's comments

268. Council's combined comments for Dolk, H et al 1997 (I and II) and Cooper, D et al. 2001 are at the end of the descriptions of the three studies.

Dolk, H et al. 1997b, 'Cancer incidence near radio and television transmitters in Great Britain. II. All High Power Transmitters', *American Journal of Epidemiology*, vol. 145, no. 1, pp .10-17. RMA ID 9621 FILEForce ID 18857.

269. This retrospective cohort analysis examines the cancer incidence from 1974 – 1986 near 20 high power television and frequency modulation radio transmitters in Great Britain to place in context he findings related to the Sutton Coldfield transmitter study.

270. The authors provided the following odds ratio for chronic lymphocytic leukaemia by distance of residence from 20 combined power radio and TV transmitters (excluding Sutton Coldfield) in persons aged 15 years or more. [295]

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[292] Table 1 page 4.

[293] Table 4 page 6.

[294] Page 6.

[295] Table 1 page 13.

Distance 0.2 Km	OR = 1.20	95% CI 0.83 – 1.74
Distance 0-10Km	OR = 1.02	95% CI 0.95 – 1.08

271. The authors provided the following risk ratio for chronic lymphocytic leukaemia according to distance of residence from the transmitter without confidence intervals.[296]

Distance 0-2 Km	OR = 1.20
Distance 2 – 4.9 Km	OR = 1.13
Distance 4.9 – 7.4 Km	OR = 0.99
Distance 7.4 – 10 Km	OR = 0.97

272. The authors concluded without reference to chronic lymphocytic leukaemia:[297]

> …when we combined results for all 20 transmitters together, there was a significant decline in risk of leukemia with distance from transmitters, although the excess was observed only beyond 2 km and was small in comparison with that observed within 2 km of Sutton Coldfield.

Council's comments

273. Council's combined comments for Dolk, H et al 1997a & b (I and II) and Cooper, D et al. 2001 are at the end of the descriptions of the three studies.

Cooper, D et al. 2001, [COMMENT] 'Re Cancer incidence near radio and television transmitters in Great Britain. 1. Sutton Coldfield transmitter; 11. All high power transmitters', *American Journal of Epidemiology*, 153(20): 202-204. Erratum: Am J Epidemiol, 2007 166:1107

274. This research letter reanalysed the data relating to cancer incidence according to the population in residence near the Sutton Coldfield television (TV) transmitter in England (Sutton Mast).

275. Cancer data from the years 1987 – 1994 were extracted from the west Midlands Cancer Intelligence Unit database.

276. The authors reported the following odds ratio for chronic lymphocytic leukaemia according to distance of residence from the transmitter. [298]

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296 Table 4 page 16.

297 Page 15.

298 Table 1 page 203.

Females		
0 – 2 Km distance	OR = 0.98	95% CI 0.12 – 3.53
0 – 10 Km distance	OR = 1.46	95% CI 1.08 – 1.84
Males		
0 – 2 Km distance	OR = 1.73	95% CI 0.56 – 4.03
0 – 10 Km distance	OR = 1.00	95% CI 0.74 – 1.26
All persons		
0 – 2 Km distance	OR = 1.42	95% CI 0.59 – 2.92
0 – 10 Km distance	OR = 1.19	95% CI 0.97 – 1.40

277. The authors did not provide a conclusion related to chronic lymphocytic leukaemia and there was no further discussion of chronic lymphocytic leukaemia specifically in the replies to the letter.

Council's comments

278. The Council commented on Dolk, H et al 1997a&b (I and II) and Cooper, D et al. 2001.

279. Council noted that the leukaemic subtypes were examined (Table 1, p. 4) and for "chronic lymphatic" an OR = 2.56, 95% CI 1.11-5.05 for 0.2 km and OR = 1.32, 95% CI 1.08-1.62 for 0-10 km. Thus, the first study gave a positive result for increased incidence of chronic lymphocytic leukaemia near the transmitter.

280. This result for chronic lymphocytic leukaemia or other cancers was not replicated in the wider study across Britain, nor in the follow-up study (Cooper et al, above). None of the other cancer types reported showed any relationship of risk to distance from the tower, although increasing trends in incidence with proximity to the tower were noted for skin melanoma and bladder cancer.

281. Council noted that in the larger study conducted in the whole of Great Britain assessing cancer risk and proximity to 20 tower transmitters, risk of leukaemia, skin melanoma, and bladder cancer was assessed using the same methodology, and numbers of observed cases were all approximately equal to expected numbers, with no trends in rates with distance from towers.

282. Council noted that the paper by Cooper et al 2001, updated and reanalysed the data from the first study by Dolk et al 1997a using different statistical methods and failed to find any consistent positive associations with leukaemias or other cancers. The OR within 2 km for chronic lymphocytic leukaemia was 0.98 (95% CI 0.12 – 3.53) and within 10km 1.46 (95% CI 1.08 – 1.84); the lack of apparent decline with distance argues against a causative association.

283. Two major limitations of these investigations are the use of proximity to a transmitting tower as a proxy for RF exposure, and the lack of individual exposure and confounder information due to the ecological design. Since the study period was so long, the effect of population movement also weakens the validity of the results.

284. The Council considered these studies collectively provide uncertain evidence of an association between radiofrequency (TV) transmissions and chronic lymphocytic leukaemia; and

- did not point to, but merely left open the possibility of the relevant association.

Hocking, B et al. 1996, 'Cancer incidence and mortality and proximity to TV towers', Medical Journal of Australia, vol. 165, no. 2, pp 601-605. RMA ID 21127 FILEForce ID 18995

285. This ecological retrospective cohort study examined the cancer incidence and mortality in 9 municipalities or Northern Sydney, 3 of which surround TV towers from the period 1972 – 1990.

286. Cancer incidence was obtained from the NSW cancer registry.

287. Data was compared between incidence of 3 inner municipalities immediately surrounding the TV towers, and hence exposed to more RF radiation and 6 outer municipalities. The calculated power density in the more exposed areas ranged from 8 to 0.2 μW/cm2 at a 4 km radius. At a distance of 12 km, power density was 0.02 μW/cm².

288. The authors reported the following risk ratio of cancer incidence and mortality data for lymphatic Leukaemia:

a. the incidence rate ratio for:

adults	RR = 1.24	95% CI 1.09 – 1.40
children	RR = 1.58	95% CI 1.07 – 2.34

b. with a mortality rate ratio of:

	MR = 2.32	95% CI 1.35 – 4.01

and the following risk ratio of cancer incidence and mortality data for lymphatic Leukaemia of the inner compared to outer area adjusted for age, sex and calendar period: [299]

[299] Table 3 page 604.

Incidence (536 cases)	RR = 1.32	95% CI 1.09 – 1.59
Mortality (267cases)	RR = 1.39	95% CI 1.00 – 1.92

289. The authors concluded: [300]

> We found an association between increased childhood leukaemia incidence and mortality and proximity to TV towers.
>
> We found no significant overall trends across time for brain cancer or leukaemia incidence for all ages combined…

Council's comments

290. The Council noted that the authors did not discuss findings in relation to a specific diagnosis of chronic lymphocytic leukaemia. The Council considered that all of the childhood cases were likely to be ALL and 90% of the adult cases were likely to be chronic lymphocytic leukaemia.

291. For leukaemia, the incidence rate ratio for adults was 1.24 (95% CI 1.09 – 1.40), whereas for children it was 1.58 (95% CI 1.07-2.34), with a mortality rate ratio of 2.32 (95% CI 1.35 – 4.01).

292. The authors were unsuccessful in identifying confounders to explain these results. The study was based on ecological exposures with no individual exposure levels, resulting in limitless potential confounders. The Council viewed this study as weakly supportive of an association between RF emissions and leukaemias generally, and weakly supportive of a relevant association with RF emissions for chronic lymphocytic leukaemia.

293. Overall, the Council considered this study.

- points to a relevant association between RF emissions and chronic lymphocytic leukaemia.

Kheifets, LI et al. 1997, 'Occupational electric and magnetic field exposure and leukemia. A meta-analysis', *Journal of Occupational Environmental Medicine*, vol. 39 (11), pp. 1074-1091. RMA ID 25834 and 21351

294. This meta analysis considered the evidence for the association between leukaemia and occupational exposure to electric and magnetic fields.

295. The authors provided a review of studies published up to 1996. A total of 70 papers were identified and 39 met the inclusion criteria. Most were in the US or Sweden.

[300] Pages 601 and 603 respectively.

Those including an assessment of chronic lymphocytic leukaemia are reported here.[301]

a. Floderus, B et al. 1993 [302]

chronic lymphocytic leukaemia	OR = 2.5	95% CI 1.6 – 3.9

b. Floderus, B et al. 1994 [303], Tornqvist, S et al. 1991 [304], Linet, MS et al. 1988 [305], Tornqvist, S et al. 1986 [306]

chronic lymphocytic leukaemia	SMR = 1.2	95% CI 0.8 – 1.8

c. London, S et al. 1994 [307] Wright, W et al. 1982 [308]

chronic lymphocytic leukaemia	OR = 1.3	95% CI 1.0 – 1.8

d. Theriault, G et al. 1994 [309]

France	OR = 4.8	95% CI 0.5 – 70.6
Canada Ontario	OR = 2.1	95% CI 0.4 – 11.6
Canada Quebec	OR = 0.3	95% CI 0.02 – 2.6

e. Tynes, T 1992 [310]

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301 Table 1 page 1075 - 1078.

302 Floderus, B et al. 1993, 'Occupational Exposure to Electromagnetic Fields in Relation to Leukemia and Brain Tumours: A Case-Control Study in Sweden', *Cancer Causes and Control*, vol. 4, pp. 465-476.

303 Floderus, B et al. 1994, 'Incidence of selected cancers in Swedish railway workers, 1961-79', *Cancer Causes and Control*, vol. 5, pp. 189-194.

304 Tornqvist, S et al. 1991, 'Incidence of Leukaemia and Brain Tumours in Some Electrical Occupations', *Br J Ind Med*, vol. 48, pp. 597-603.

305 Linet, MS et al. 1988,' Leukemias and occupation in Sweden: a registry-based analysis. *American Journal of Industrial Medicine*, vol. 14, pp. 319-330.

306 Tornqvist, S et al. 1986, 'Cancer in the electric power industry', Br J Ind Med, vol. 43, pp. 212-213.

Tornqvist, S et al. 1986, was not available to (before) the RMA at the relevant times, however the information above is cited in **Kheifets, LI, et al.** 1997 and as such was considered by the Council.

307 London, S et al. 1994, 'Exposure to magnetic fields among electrical workers in relation to leukemia risk in Los Angeles County', *American Journal of Industrial Medicine*, vol. 26, no. 1, pp. 47-60.

308 Wright, W et al. 1982, 'leukaemia in workers exposed to electrical and magnetic fields', Lancet, ii, pp. 1160-1161.

Wright, W et al. 1982, was not available to (before) the RMA at the relevant times, however the information above is cited in **Kheifets, LI, et al.** 1997 and as such was considered by the Council.

309 Theriault, G et al. 1994, 'Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec, Canada, and France:1970-1989', *Am J Epidemiol*, vol. 139, no. 6, pp. 550-572.

chronic lymphocytic leukaemia	SIR = 1.0	95% CI 0.6 – 1.4

f. Loomis, DP and Savitz, DA 1990 and 1991 [311]

chronic lymphocytic leukaemia	OR = 0.6	95% CI 0.3 – 1.1

g. Pearce, NE et al. 1989, 1986, 1985 [312]

chronic lymphocytic leukaemia	OR = 3.4	95% CI 1.38 – 8.9

h. Milham, S 1988, and Milham, S 1985 [313]

chronic lymphocytic leukaemia	SMR = 1.1	95% CI 0.4 – 2.4

i. Gillman, PA et al. 1985 [314]

chronic lymphocytic leukaemia	OR = 6.3	no confidence interval / reported as significant

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[310] Tynes, T et al. 1992, 'Incidence of cancer in Norwegian workers potentially exposed to electromagnetic fields', *Am J Epidemiol,* vol. 136, pp. 81-88. – 37,945 male Norwegian electrical workers exposed to extremely low frequencies and radio frequencies between 1961 – 1985. Exposure was based on job titles.

Tynes, T et al. 1992, was not available to (before) the RMA at the relevant times, however the information above is cited in **Kheifets, LI, et al.** 1997 and as such was considered by the Council.

[311] Loomis, DP & Savitz, DA 1990, 'Mortality from brain cancer and leukaemia among electrical workers', *British Journal of Industrial Medicine*, vol. 47, pp. 633-638.

Loomis, DP & Savitz, DA 1991, 'Occupation and leukemia mortality among med in 16 states : 1985 – 1987 Am J Ind Med, vol. 19, pp. 509-521. - US cases among 410,651 male deaths in 16 US states, 1985-1986.

Loomis, DP & Savitz, DA 1991, was not available to (before) the RMA at the relevant times, however the information above is cited in **Kheifets, LI, et al.** 1997 and as such was considered by the Council.

[312] Pearce, N et al. 1989, 'Case-control studies of cancer in New Zealand electrical workers', *Int J Epidemiolo,* vol. 18, pp. 55-59

Pearce, N et al. 1986, 'leukemia among New Zealand agricultural workers, a cancer registry-based study', *Am J Epidemiol*, vol. 124, pp. 402-409.

Pearce, N et al. 1985, 'Leukaemia in electrical workers in New Zealand', *Lancet*, pp. 811-812.

Pearce, N et al. 1989, 1986, 1985, were not available to (before) the RMA at the relevant times, however the information above is cited in **Kheifets, LI, et al.** 1997 and as such was considered by the Council.

[313] Milham, S 1988, 'Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies', *American Journal of Epidemiology*, vol. 127, no. 1, pp. 50-54. - 67,829 amateur radio operators between 1979 – 1984, no exposure details provided.

Milham, S 1985, 'Mortality in workers exposed to electromagnetic fields', *Environ Health Perspect*, vol. 62, pp. 297-300.

Milham, S 1988 and 1985*,* were not available to (before) the RMA at the relevant times, however the information above is cited in **Kheifets, LI, et al.** 1997 and as such was considered by the Council.

[314] Gillman, PA et al. 1985, 'Leukemia risk among U.S. white male coal miners: a case-control study', *J Occup Med*, vol. 27, pp. 669-671.

Gillman, PA et al. 1985, was not available to (before) the RMA at the relevant times, however the information above is cited in **Kheifets, LI, et al.** 1997 and as such was considered by the Council.

j. Coleman, M 1983 [315]

chronic lymphocytic leukaemia	PIR = 1.3,	no confidence interval provided

296. The authors provided methods for their assessment of study and calculation of pooled risk estimates according to a fixed effect model and a random effect model.

297. The authors provided the following pooled risk estimate for chronic lymphocytic leukaemia for both models from 12 studies, stating the random effect model as the more appropriate from statistical analysis.[316]

Fixed effect model	pooled RR = 1.44	95% CI 1.25 – 1.67
Random effect model	pooled RR = 1.55	95% CI 1.10 – 2.19

298. The authors provided an analysis of heterogeneity and sensitivity of the pooled data. The authors acknowledged the limitations of their review in assessing the contribution of publication bias.

299. The authors concluded, without specific reference to chronic lymphocytic leukaemia: [317]

> In conclusion, we found a small but significant elevation in risk of leukemia in relation to estimates of potential workplace magnetic field exposure. The risk was higher for some specific jobs and for more specific leukemia subtypes.

Council's comments

300. The authors pooled all studies up to 1996 and used sample measures for various occupations. Variations in exposure for different occupations were noted (ibid Fig. 3) which adds credence to their review. The pooled risk estimate for chronic lymphocytic leukaemia was 1.44 [CI 1.25-1.67] with some publication bias found by funnel plot (ie non-publication of small studies with negative findings).

301. There was substantial heterogeneity in results, and the Council notes that the authors also concluded:

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315 Coleman, M et al. 1983, 'Leukaemia incidence in electrical workers,' *Lancet*, 1983, I, pp. 982-983
Coleman, M et al. 1983, was not available to (before) the RMA at the relevant times, however the information above is cited in **Kheifets, LI, et al.** 1997 and as such was considered by the Council.

316 Table 4 page 1081.

317 Page 1089.

The apparent lack of a clear pattern of exposure and risk substantially detracts from the hypothesis that measured magnetic fields in the work environment are responsible for the observed excess risk of leukaemia.

302. The Council considered this study provides weak evidence of an association between EMF and chronic lymphocytic leukaemia; and

– points to a relevant association.

Kheifets, LI et al. 1999, 'Comparative analyses of the studies of magnetic fields and cancer in electric utility workers: studies from France, Canada, and the United States', *Occupational & Environmental Medicine*, vol;. 56, no. 8, pp. 567-574. RMA ID 23819 FILEForce ID 18543

303. This nested case-control study summarized the data available from five cohorts from three studies to consider the relationship between occupational exposure to magnetic fields and risk of brain cancer and leukaemia.

304. The five cohorts were electric utility workers analysed in the following studies;

a. Sahl et al 1993 [318]

Southern California Edison (SCE)	Cohort size 36 221

b. Theriault et al 1994 [319]

Electricite de France (EdF)	Cohort size 170 000
Ontario Hydro (OH)	Cohort size 31 543
Hydro Quebec (HQ)	Cohort size 21 749

c. Savitz and Loomis 1995 [320]

5 US Utilities (UNC)	Cohort size 138 905

305. The authors used the following published results for leukaemia risk ratio in the comparative analysis. [321]

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318 Sahl, JD et al. 1993, 'Cohort and nested case-control studies of hematopoietic cancers and brain cancer among electric utility workers', *Epidemiology*, vol. 4, no. 2, pp. 104-114.

Sahl et al 1993, was not available to (before) the RMA at the relevant times, however the information above is cited in **Kheifets, LI, et al.** 1999 and as such was considered by the Council.

319 Theriault, G et al. 1994a, 'Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec Canada and France: 1970-89', Am J Epidemiology, vol. 139, pp. 550-572. See above [295.d].

320 Savitz, DA and Loomis, DP 1995, 'Magnetic field exposure in relation to leukemia and brain cancer mortality among electric utility workers,' *Am J Epidemiol*, vol. 141, pp. 123-134.

SCE	RR 1.1	95% CI 0.8-1.5
EdF, OH & HQ	RR 1.8	95% CI 0.8-4.0
Edf	RR 1.9	95% CI 0.5-7.8
OH	RR 3.6	95% CI 0.9-14.3
HQ	RR 0.5	95% CI 0.04-3.8
UNC	RR 1.1	95% CI 0.6-2.1

306. The authors described the data for risk of leukaemia in response to cumulative magnetic field exposure expressed as microtesla-years (μT-years) according to four of the cohort groups. [322]

Edf		
>1yr	RR 1.04	95% CI 0.72-1.50
10 yr	RR 0.69	95% CI 0.14-3.4
>10yr	RR 1.11	95% CI 0.7-1.75
HQ		
>1yr	RR 1.81	95% CI 0.46-1.42
10 yr	RR 0.90	95% CI 0.22-3.6
>10yr	RR 0.72	95% CI 0.34-1.55
UNC		
>1yr	RR 1.02	95% CI 0.84-1.24
10 yr	RR 1.46	95% CI 0.68-3.10
>10yr	RR 1.00	95% CI 0.8-1.24
SCE		
>1yr	RR 1.12	95% CI 0.95-1.33

[321] Page 570 table 3.

[322] Page 571 table 4.

10 yr	RR 1.03	95% CI 0.75-1.42
>10y	RR 1.14	95% CI 0.94-1.39

307. The authors described the data for risk of leukaemia in response to magnetic field exposure intensity expressed as microtesla-years (μT-years) according to four of the cohort groups: [323]

Edf	>1yr	RR 1.16	95% CI 0.52-2.80
HQ	>1yr	RR 0.94	95% CI 0.05-1.98
UNC	>1yr	RR 1.49	95% CI 0.86-2.60
SCE	>1yr	RR 1.39	95% CI 0.84-2.30

308. The authors described the data for combined cohort data for the risk of leukaemia in response to magnetic field exposure intensity expressed as microtesla-years (μT-years) for a range of exposure variables excluding cohort OH: [324]

Continuous data	RR 1.09	95% CI 0.97-1.23
0-4 μT-years	RR 1.0	n.a.
4- 8 μT-years	RR 1.07	95% CI 0.57-2.01
8-16 μT-years	RR 1.0	95% CI 0.65-1.55
>16 μT-years	RR 1.35	95% CI 0.85-2.14

309. The authors described the data for combined cohort data for the risk of leukaemia in response to magnetic field exposure intensity expressed as microtesla-years (μT-years) for a range of exposure variables including cohort OH(1): [325]

Continuous data	RR 1.09	95% CI 0.98-1.21
0-4 μT-years	RR 1.0	n.a.
4 – 8 μT-years	RR 1.26	95% CI 0.70-2.26
8-16 μT-years	RR 1.44	95% CI 0.64-3.24

[323] Page 571 table 4.

[324] Page 572 table 6.

[325] Page 572 table 6.

>16 µT-years	RR 1.48	95% CI 0.96-2.30

310. The authors described the data for combined cohort data for the risk of leukaemia in response to magnetic field exposure intensity expressed as microtesla-years (µT-years) for a range of exposure variables including cohort OH (1): [326]

Continuous data	RR 1.10	95% CI 0.98-1.23
0-4 µT-years	RR 1.01	n.a
4- 8 µT-years	RR 1.18	95% CI 0.73.1.90

311. The authors concluded: [327]

> …overall the studies showed little or not relation between continuous magnetic fields and leukaemia.
>
> …separate analyses of acute myeloid leukaemia (AML) and chronic lymphocytic leukaemia (chronic lymphocytic leukaemia) were conducted. These analyses were not very informative because of small numbers; however, they provided no indication that results for these subtypes differed from results for all leukaemia.
>
> …we found no indication of an association with chronic lymphocytic leukaemia either in individual studies or in comparison with overall leukaemia or acute myeloid leukaemia.

Council's comments

312. The Council considered this article presents a comparison of three conflicting studies, [328] showing a small increase of brain tumours and leukaemia.

313. The Council noted the review included all studies with suitable data and took steps to make data sets comprehensible using complex statistics. the authors found a RR of brain cancer/ leukaemia of 1.09, with numbers insufficient to comment further regarding chronic lymphocytic leukaemia.

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326 Page 572 table 6.

327 Page 571, 572 & 573.

328 Sahl, JD et al. 1993, 'Cohort and nested case-control studies of hematopoietic cancers and brain cancer among electric utility workers', Epidemiology, vol. 4, pp. 104-114.

Sahl et al 1993, was not available to (before) the RMA at the relevant times, however the information above is cited in **Kheifets, LI, et al.** 1999 and as such was considered by the Council.

Theriault, G et al. 1994, 'Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec, Canada and France, 1970-1980', Am J Epidemiol, vol. 139, pp. 550-572.

Savitz, DA & Loomis, DP 1995, 'Magnetic field exposure in relation to leukemia and brain cancer mortality in electric utility workers', *Am J Epidemiol*, vol. 141, pp. 123-134.

314. The Council noted that in pooling these data, although the EdF, HQ and UNC studies provide little evidence of a positive trend for Leukaemia, the addition of the positive SCE resulted in a weak association, with a best estimate of a relative risk of 1.09 / 10 T-years of exposure (95% CI 0.98 to 1.21) [329].

315. Council found that this paper:

– does not point to, but merely left open the possibility of a relevant association.

Habash, RW et al. 2003, 'Health risks of electromagnetic fields. Part I: Evaluation and assessment of electric and magnetic fields', *Crit Rev Biomed Eng*, vol. 31, no. 3, pp. 141-195. RMA ID 30575 FILEForce ID 19125

316. This review article evaluates the potential health risks associated with residential and occupational exposure to EMF from laboratory, epidemiology and clinical studies.

317. The authors provided a discussion and review of literature resulting in the consensus of maximal permissible exposure (MPE) values for EMF. Figures were provided as a summary of several organisations documented exposure standards. [330]

318. The authors provided a comprehensive review of the identification of sources of EMF exposure and the data from measurement surveys. [331]

319. The authors provided a review of epidemiological studies investigating the link between EMF and disease, considering studies related to childhood cancer separately to their review of studies of adult cancers.

320. In respect of adult cancers associated with occupational exposure, studies including the haematopoietic system are noted here.

a. Theriault, G et al. 1994 [332]

b. Savitz, DA and Loomis, DP 1995 [333]

c. Floderus, B et al. 1993 [334]

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329 See at p. 573.

330 Table 1 page 4.

331 Table 2 page 10.

332 Theriault G, et al. 1994, 'Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec, Canada, and France:1970-1989', *Am J Epidemiol*, vol. 139(6) pp 550-572.

333 Savitz DA, Loomis DP (1995). Magnetic field exposure in relation to leukemia and brain cancer mortality among electric utility workers. *Am J Epidemiol*, 141(2) pp 123-134.

d. Miller, AB et al. 1996 [335]

e. Villeneuve, PJ et al. 2000 [336]

321. The authors provided a review of the literature considering non cancer health effects of exposure to EMF and the literature from cellular and animal studies. Relevant studies involving the haematopoietic system are:

a. Khalil, AM and Qassem, W 1991 [337]

b. House, RV et al. 1996 [338]

c. Aldinucci, C et al. 2003 [339]

322. The authors summarized the reviews of the literature on health effects of EMF. [340]

323. The authors concluded: [341] and [342]

> … current evidence from laboratory and epidemiological studies on the association between EMF exposure and cancer or other harmful health outcomes is inconsistent and inconclusive. Whereas early studies focusing on residents living near high-voltage transmission lines provided some evidence of a link between the risk of (childhood)

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334 Floderus B, Persson T, Stenlund C, Wennberg A, (1993). Occupational Exposure to Electromagnetic Fields in Relation to Leukemia and Brain Tumours: A Case-Control Study in Sweden*. Cancer Causes and Control*, vol. 4 pp 465-476.

335 Miller AB, To T, Agnew DA, Wall C, Green LM (1996). Leukemia following occupational exposure to 60-Hz electric and magnetic fields among Ontario electric utility workers. *American Journal of Epidemiology*, vol. 144 pp 150-160.

336 Villeneuve, PJ et al 2000 Leukemia in electric utility workers: the evaluation of alternative indices of exposure to 60 Hz electric and magnetic fields. *Am J Indust Med* 2000; 37: 607-617 - elevated risk of leukemia seen in senior workers who spent the most time in electric fields above certain thresholds, in the range of 10-40 V/m

337 Khalil and Qassem 1991 Cytogenetic effects of pulsing electromagnetic field on human lymphocytes in vitro: chromosomes aberrations, sister chromatid exchanges and cell kinetics. *Mutat Res* 1991; 247: 141-146. - choromosomal aberrations by exposing human lymphocyte cultures to a pulsing EM field.

338 House, RV et al. 1996, 'Immune function and host defence in rodents exposed to 60Hz magnetic fields', *Fundam Appl Toxicol*, vol. 34, pp. 228-239. - exposed mice and rats to 2,200 and 1000 µT (60 Hz) continuously. No significant changes in the distribution of lymphocyte subsets in the spleens of exposed mice were observed compared to controls.

339 Aldinucci et al 2003 The effect of exposure to high flux density static and pulsed magnetic fields on lymphocyte function*. Bioelectromagnetics,* vol. 24, pp. 373-379 – human white blood cells no effect of a 4275-mT field on the inflammatory response of normal or leukemic cells no effect of a 4275-mT field on the inflammatory response of normal or leukemic cells.

These three articles at fn 336 to 338 were not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

340 Table 4 page 36.

341 Page 34.

342 Page 40.

leukemia and EMF as characterized by Wertheimer and Leeper 1979 [343] most of the subsequent studies using actual field measurements failed to confirm the initial findings.

Since 1979, there has been a flurry of scientific activity to evaluate the possibility that exposure to EMF from power lines and other sources may cause cancer. Overall, the currently available epidemiological and toxicological data do not provide clear evidence that EMF is associated with an increased risk of cancer, although there is some epidemiological evidence of linkages between EMF and childhood leukaemia. There is also no convincing evidence from cellular and animal studies that EMF can directly damage DNA or promote tumor growth.

Council's comments

324. The Council noted a number of epidemiological studies are discussed, and the general issues are reviewed, including the Floderus et al 1993 and Miller et al papers. Animal studies are discussed with no direct evidence of causation but some question mark about promotion of cancer. Clinical studies are discussed.

325. Also, the authors state that the overall evidence is inconsistent / inconclusive and they cite the conclusions of many previous reviews.

326. The Council noted the authors concluded that there is no clear evidence of EMF association with cancer, but there is some epidemiological link with childhood leukaemia and further study is needed.

327. The Council considered this review was more focused upon a possible link with childhood leukaemia, than adult chronic lymphocytic leukaemia and as such:

- did not point to but merely leaves open the possibility of a relevant association.

Preece, AW et al. 2000, 'Power frequency electromagnetic fields and health, Where's the evidence?', *Phys Med Biol*, vol. 45, no. 9, pp. R139-R154. RMA ID 28541 FILEForce ID 18653

328. This review article provides a summary of the debate regarding the proposition that extremely low frequency (ELF) electromagnetic fields (EMF) may be associated with an increased risk of cancer or other health hazards.

329. In the background to this paper there is discussion on the conflicting evidence and various views required within a multidisciplinary understanding of the risks of electromagnetic fields.

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[343] Wertheimer, N and Leeper, E 1979, 'Electric wire configurations and childhood cancer', *Am J Epidemiol*, vol. 109, pp. 273-284.
This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

330. It is stated that against the sceptical view are good epidemiological studies from Feychting 1993, Olsen 1993, Verkasalo 1993 and Ahlbon 1993. See below at [339]

331. The authors state that the rigorous criteria of Bradford Hill 1965 have prevented acceptance of such causes of agents of smoking and asbestos early on in the history.

332. There was a discussion of epidemiology and it is stated that "despite the absence of a plausible mechanism", "the impression persists of a possible association between certain cancers and EMF exposure", the authors go on to review a number of studies with contradictory results and "meta analysis by Wattenberg 1998 [344] and Angelillo & Villari 1999 [345,] both of which analysing 12-14 studies conclude that there is an increase relative risk of leukaemia of approximately 30% with electromagnetic field exposure.

333. This article then goes on to "statements from official agencies and professional bodies" and in particular a 1997 NRC report [346] which states "based on a comprehensive evaluation of published studies relating to the effects of power frequency, electric and magnetic fields… the current body of evidence does not show that exposure to these fields presents a human health hazard"

334. A 1998 NIEHS [347] working group report is quoted "there is limited evidence that residential exposure to ELF magnetic fields is carcinogenic to children…there is limited evidence that occupational exposure to ELF magnetic field is carcinogenic to humans on the basis of results of studies of chronic lymphocytic leukaemia" the authors of the current paper make the statement that the IARC International Agency for research on cancer defines limited evidence as a positive association for which causal interpretation is credible but chance, bias or confounding factors cannot be ruled out with reasonable confidence.

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344 Wartenberg, N & Leeper, E 1979, 'Electrical wiring configurations and childhood cancer, *Am J Epidemiol*, vol. 109, pp. 273-284.

Leukaemia OR = 1.4 95% CI 1.10 – 1.87

345 Angelillo, IF & Villari, P 1999, 'Residential exposure to electromagnetic fields and childhood leukaemia, a meta-analysis, *Bull World Health Org*, vol. 77, pp. 906-915.

Childhood Leukaemia

Pooled RR = 1.46 – wiring configuration codes 95% CI 1.05 – 2.04

Pooled RR = 1.59 – TWA (24h) magnetic field 95% CI 1.14 – 2.22

346 NRC – National Research Council 1997, *Possible health effects of exposure to residential electric and magnetic fields*, NAP, Washington, DC.

These five articles/reports were not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

347 NIEHS – Portier, CJ & Wolfe, MS eds. 1998, *Assessment of the health effects from exposure to power-line frequency electric and magnetic fields, a working report,* NIH Publication 98-3981; and

Wolfe, MS 1999, *Health effects from exposure to power-line frequency electric and magnetic fields*, NIH Publication 99-4493.

335. The NIEHS 1999 report states the scientific evidence suggesting ELF-EMF exposure pose any health risk is weak, the strongest evidence…in human populations with two forms of cancer, childhood leukaemia and chronic lymphocytic leukaemia in occupationally exposed adults". They go on to say "the epidemiological studies demonstrate for some methods of measuring exposure a fairly consistent pattern of a small increased risk with increasing exposure", with no consistent alternative explanation found. The NIEHS report is further quoted "the lack of connection between human data and experimental data (animal and mechanistic) severely complicates the interpretation of these results…the human data…shows some consistency that is difficult to ignore".

336. The authors also quote a report from the IEEE Engineering in Medicine Biology and Society EMBS Committee [348] who reviewed additional data from 1996 to 1999. They considered that large recent studies for comprehensive exposure assessment do not provide compelling evidence for causal link between residential exposure to magnetic field and cancer but are sufficient to categorise power frequency magnetic fields as a possible carcinogen within the IARC definition.

337. This definition is a possible carcinogen is used for exposure, circumstances for which there is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals.

338. The overall report from the EMBS was that data are not sufficient to support a causative link between weak power frequency magnetic fields and cancer.

339. The authors list the following studies from the Scandinavian studies of residence close to power lines as key epidemiological data raising a possible risk of cancer from electromagnetic fields particularly in children.

    a. Feychting and Ahlbom 1993 [349]

    b. Olsen et al 1993 [350]

    c. Verkasalo et al 1993 [351]

    d. Ahlbom et al 1993 [352]

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348 Committee on Man and Radiation (COMAR) 2000, 'Possible health hazards from exposure to power-frequency electric and magnetic fields, a COMAR Technical Information Statement', *IEEE Eng Med Biol*, vol. 19, pp. 131-137.

349 Feychting, M & Ahlbom, A 1993, 'Magnetic fields and cancer in children residing near Swedish high-voltage power lines,' *Am J Epidemiol*, vol. 138, pp. 467-481.

350 Olsen, JH et al. 1993, 'Residence near high voltage facilities and risk of cancer in children,' *Br Med J* 307 891-895.

351 Verkasalo, PK et al. 1993, 'Risk of cancer in Finnish children living close to power lines,' *Br Med J,* vol. 307 895-899.

352 Ahlbom, A et al. 1993, 'Electromagnetic fields and childhood cancer *Lancet* 342 1295-6.

340. The authors summarized the experimental evidence for ELF EMF as a carcinogen without specific reference to the impact on the haematopoietic system.

341. The authors summarized evidence for ELF EMG exposure from household appliances and the assessment of exposure from environmental and occupational exposures such as power lines

342. The authors made the following conclusion without additional discussion regarding chronic lymphocytic leukaemia. [353]

> …the strongest evidence for possible health effects comes from associations observed in human population with two forms of cancer: childhood leukaemia and chronic lymphocytic leukaemia in occupationally exposed adults. However, overall the scientific evidence suggesting the ELF EMF exposures pose any health risk is weak.

343. The authors also conclude.

- There is in some epidemiological studies a consistent pattern of increased risk with increasing exposure.
- No mechanism for carcinogenic effects of magnetic fields has been established.
- Almost all recent statement by official agencies and bodies from 1997 to 2000 conclude that data are insufficient to show that power frequency, electric and magnetic fields are a human health hazard.
- Further research seems to be required in the area of mechanism.

Council's comments

344. The Council considered this was a balanced review because the epidemiological data was reviewed critically and was found to be weakly supportive of an association for ELF EMR in those occupationally exposed.

345. The Council viewed this paper as:

- does not point to but merely leaves open the possibility of a relevant association.

Havas, M 2000, 'Biological effects of non-ionizing energy: a critical review of the reports by the US Institute of Environmental Health Sciences as National Research Council and the US National they relate to the broad realm of EMF bioeffects',

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These five articles were not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

353 Page R151.

National Research Council Canada; NRC Research Press, *Environmental Reviews*, vol 8, no. 3, pp. 173-253. RMA ID 24131, FILEForce ID 18501

346. This review critically evaluates the US National Research Council (NRC) [354] and the US National Institute of Environmental Health Science (NIEHS) [355] reports on health concerns related to residing near powerlines, cell phone antennas or television and radio broadcast towers.

347. The background includes a discussion of different categories of fields including geofields, technofields and biofields. It is noted that frequencies lower than microwave were previously assumed to be safe and then a number of publications raising concerns about the safety of such fields, are cited, namely:

    a. Korobkova, VP et al. 1972 [356] - 500kV (500 & 750 kV lines) substation switchyard workers in Soviet Union – stated that "a current of 80-120 μA flowing through a man for a long time affects him unfavourably."

    b. Nordstrom, S Birke, E and Gustavsson, L 1983 [357] (and 1984 – no citation), - effects of high voltage power lines on substation workers and their families.

    c. Wertheimer, N and Leeper, E 1979 [358] - residential exposure to power line frequency lines in Denver, Colorado – reported an increased incidence of childhood Leukaemia, lymphoma, and nervous system tumours…

348. They then reviewed a large number of studies looking at adult residential and occupational exposures. The problem with ascertaining exposure due to wire codes and difficulties of measuring exposure in vivo and in vitro are acknowledged.

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354 NRC 1997, *Possible Health Effects of exposure to residential electric and magnetic fields*, National Research Council (US) committee on the possible effects of electromagnetic fields on biologic systems 1997, National Academy Press, Washington D.C. See [333]

355 Portier CJ and Wolfe MS eds. 1998, *Assessment of health effects from exposure to power line frequency electric and magnetic fields*, National Institute of environmental health sciences working group report of the national institutes of health. NIH publication No. 98-3981, Research Tricangle Park N.C.

356 Korobkova, VP et al. 1972, 'Influence of the electric field in 500 and 750 kV switchyards on maintenance staff and means for its protection'. In International Conference in August 1972 on Large High Voltage Electric Systems, Paris, CIGRE, Paris.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

357 Nordstrom, S Birke, E and Gustavsson, L 1983, 'Reproductive hazards among workers at high voltage substations', *Bioelectromagnetics*, vol. 4, pp. 91-101.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

358 Wertheimer, N and Leeper, E 1979, 'Electric wiring configuration and childhood cancer', *Am J Epidemiol*, vol. 109, no. 3, pp. 273-284.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

349. The author noted that both committees (NRC and NIEHS) focused on low frequency electric and magnetic fields (those associated with power distribution at 50 and 60 Hz. Consequences of exposure to ionizing radiation, UV, visible infrared, MW and RF were not included in the reports.

350. The author stated: [359]

> A narrow focus on power line frequencies in both reports (with insufficient assessment of higher frequencies associated with cell phones, for example) and an absence of occupational exposure and electromagnetic sensitivities in the NR mandate are the key weakness.

351. The author summarized the NRC document:

> NRC a review of 520 references published from 1953 – 1996.
>
> Executive summary followed by 7 chapters (1) introduction, (2) exposure and physical interactions, (3) cellular and molecular effects, (4) animal and tissue effects, (5) epidemiology, (6) risk assessment and (7) research needs.

352. The author summarized the NIEHS document:

> NIEHS 830 references published form 1941 – 1998.
>
> Five chapters (1) introduction, (2) Occurrence and measurement of extremely low frequency electromagnetic fields, (3) internal dosimetry, (4) biological data relating to the toxicity of ELF and (5) final summary and evaluation.

353. The author reported the NIEHS evaluation and votes on Cancer in adults [360]

> …there is inadequate evidence that residential exposure to extremely low frequency electromagnetic fields is carcinogenic to adults.

354. The author reported the NIEHS evaluation and votes on chronic lymphocytic leukaemia in adults: [361]

> There is limited evidence that occupational exposure to ELF EF is carcinogenic to adults. This evaluation is based on the results of studies of chronic lymphocytic leukemia.

355. The author reported the NIEHS evaluation and votes on Cancer in experimental animal models: [362]

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359 Page 180.

360 Table 4, page 189.

361 Table 5, page 190.

362 Table 6, page 191-192.

> There is inadequate evidence in experimental animals for carcinogenicity from exposure to ELF EF.

356. The author reported the NIEHS evaluation and votes on the immune system: [363]

> There is no evidence in experimental animals for effects of ELF EMF on the immune system.

357. The author reported the NIEHS evaluation and votes on mechanism of EMF on cellular function: [364]

> A limited number of well-performed studies provide moderate evidence for mechanically plausible effects of EMF greater than 0.1 MT (100μT, 1000 MG) in vitro at endpoints generally regarded as reflecting the action of toxic agents.
>
> There is weak evidence for an effect of fields lower than approximately 0.1 MT (100μT, 1000 MG).

358. The author reported the NIEHS overall evaluation related to carcinogenicity and chronic lymphocytic leukaemia: [365]

> The overall evaluation of the majority of the working group is that extremely low frequency EMF can be classified as "possibly carcinogenic" (group 2B) and that this "is a conservative, public-health decision based on limited evidence of an increased risk for childhood leukemias with residential exposure and an increased occurrence of chronic lymphocytic leukaemia associated with occupational exposure. For these particular cancers, the results of vivo, in vitro, and mechanistic studies do not confirm or refute the findings of the epidemiological studies." (NIEHS 1998, p. 402)

359. The author provided a comprehensive review of the calculation of domestic exposure both near and far from power lines incorporating data from household appliances.

360. The author provided a comprehensive review of the calculation of occupational exposure both in considered high and low risk activities.

361. The author provided a summary of the epidemiological evidence of studies of chronic lymphocytic leukaemia and MF exposure. [366]

    a. London et al 1994 [367] - 4 cases of chronic lymphocytic leukaemia

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363 Table 6, page 191-192.

364 Table 6, page 191-192.

365 Page 193.

366 Table 18, page 222.

Exposure > 0.8 μT	RR = 0.8	95% CI 0.4 – 1.5

b. Floderus et al 1993 [368] - 17, 33, 41, 22 chronic lymphocytic leukaemia cases respectively

Exposure 0.16 - 0.19 μT	OR = 1.1	95% CI 0.5 – 2.3
Exposure 0.2 - 0.28 μT	OR = 2.2	95% CI 1.1 – 4.3
Exposure ≥ 0 29 μT	OR = 3.0	95% CI 1.6 – 5.8
Exposure ≥ 0.41 μT	OR = 3.7	95% CI 1.8 – 7.7

c. Theriault et al 1994 [369] - 24 and 6 chronic lymphocytic leukaemia cases respectively

France & Canada

Exposure ≥ 3 1 μT-year	OR = 1.5	95% CI 0.5 – 4.4
Exposure ≥ 16 μT-year	OR = 1.7	95% CI 0.44 – 6.7

d. Savitz and Loomis 1995 [370] 5 chronic lymphocytic leukaemia cases respectively

Exposure ≥ 2 0 μT-year	OR = 0.55	95% CI 0.17 – 1.8

e. Feytching et al 1997 [371] - see footnote 208 below

37 chronic lymphocytic leukaemia cases Occupational TWA < 0.20	OR = 1.2	95% CI 0.7 - 1.9
28 chronic lymphocytic leukaemia cases Occupational TWA ≥ 0.20	OR = 1.7	95% CI 1.0 - 2.9

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367 London, S et al. 1994 Exposure to magnetic fields among electrical workers in relation to leukemia risk in Los Angeles County Am J Ind Med 26: 47- 60. (data also used for the cohort from Kheifets LI, et al. 1997**)**

368 Floderus B, et al. 1993, Occupational Exposure to Electromagnetic Fields in Relation to Leukemia and Brain Tumours: A Case-Control Study in Sweden. Cancer Causes and Control, Vol 4 pp 465-476. (data also used for the cohort from Kheifets LI, et al. 1997)

369 Theriault G, et al. 1994, Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec, Canada, and France:1970-1989. *Am J Epidemiol*, 139(6) pp 550-572. (data also used for the cohort from Kheifets LI, et al. 1997 and in 1999)

370 Savitz, DA & Loomis, DP 1995, Magnetic field exposure in relation to leukemia and brain cancer mortality among electric utility workers*. Am J Epidemiol*, 141(2) pp 123-134. (data also used for the cohort from Kheifets LI, et al. 1999**)**

371 Feychting, M 1997, Occupational exposure to electromagnetic fields and adult leukaemia: a review of the epidemiological evidence. *Radiation and Environmental Biophysics*, 35(4) pp 237-242.

This article was not available to (before) the RMA at the relevant times, however the information above is cited in **Havas, M** 2000 and as such was considered by the Council.

26 chronic lymphocytic leukaemia cases:		
Occ exp TWA > 2.0 μT Resid exp low	OR = 1.5	95% CI 0.8 - 2.7
2 chronic lymphocytic leukaemia cases:		
Occ exp TWA > 2.0 μT Resid exp high	OR = 2.1	95% CI 0.4 - 10

362. The author argues that in the NRC and NIEH reviews, the interpretation was conservative, leaning to the side of "no effect". He asserts that the evidence is considerably stronger than appears in their evaluation, if a broader literature is examined.

363. The author also states that he does not agree that epidemiological evidence indicates a causal relationship between EMF exposure and childhood and adult cancer. What has been documented is an association, and he suggests this maybe one of promotion rather than initiation.

364. The author describes plausible mechanisms for toxic effects of electromagnetically fields which have been demonstrated. He reviews a number of mechanisms including the aforementioned melatonin production, effects on mitosis and DNA synthesis and ions fluxes. He argues that, as with other environmental pollutants such as DDT or asbestos, the effects are often observed long before the mechanism is understood.

365. The author's conclusion based on the NRC and NIEHS documents are as follows:

- Electric and magnetic fields can affect living organisms which can be harmful and can occur at low intensities found in residential settings.

- In adults, the link between EMF exposure and leukaemia, brain tumours and cancer is convincing. Two forms of leukaemia predominate- AML and chronic lymphocytic leukaemia with some evidence of a dose response relationship

Council's comments

366. The Council considered this was a detailed review and notes that this report as a whole and particularly for acute myeloid leukaemia (AML) and chronic lymphocytic leukaemia , included a dose response relationship.

367. The Council viewed this review as weakly supportive of an association between EMF/ RF and chronic lymphocytic leukaemia and

- points to a relevant association.

Krewski, D et al. 2001b, 'Recent advances in research on radiofrequency fields and health', *Journal of Toxicology & Environmental Health*, Part B, Critical Reviews, vol. 4, no.1, pp 145-159. RMA ID 25130 FILEForce ID 18478

368. This review article is an update on publications since the Royal Society of Canada report of 1999 and considered the available evidence for radiofrequency effects on health, namely referring to laboratory.

369. The authors reviewed the evidence regarding the thermal effects of radiofrequency, noting experimental evidence supports both increased and no change to core heating. Local temperature changes in response to radiofrequency have been reliably demonstrated. [372]

370. The authors reviewed the evidence for the relationship between EMF exposure and ornithine decarboxylase (ODC) activity in both cells and tissues. [373]

    – Experimental evidence suggest some relationship between ODC activity and EMF exposure in mammary and dermal tissue.

    – The authors referred to evidence for support that constitutive ODC over expression induced by oncogenes can lead to an altered phenotype which maybe related to malignant transformation.

371. The authors reviewed the evidence for the relationship between EMF exposure and melatonin:[374]

    – enhanced calcium efflux and ornithine decarboxylase activity in the brain and increased permeability of the blood brain barrier;

    – increased circulating melatonin;

    – increased cell proliferation.

372. The authors reviewed the evidence for carcinogenicity but not in the haematopoietic system.

373. The authors referenced Morgan et al 2000 [375] with regard to their findings of cause specific mortality (SMR = 0.54 95% CI 0.33 – 0.83) due to all lymphomas and leukaemias.

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372 Page 146.

373 Page 146-148.

374 Page 148-149.

375 Morgan, RW et al. 2000, Radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems. Comment on: Epidemiology, 11(2) pp 118-27. RMA ID 28377 FILEForce ID 18645

374. The authors concluded that the data are "inadequate for a comprehensive evaluation of risk" and the conclusion of the British study (Stewart report 2000 [376]) was: "balance of evidence… does not suggest…adverse health effects." It is noted that further studies are needed and that a precautionary approach is recommended.

375. The authors did not specifically discuss results related to chronic lymphocytic leukaemia.

Council's comments

376. The Council considered this study touched on the contended factor regarding non-ionizing radiation.

377. The Council considered this study:

- does not point to but merely leaves open the possibility of a relevant association.

Moulder, JE et al. 1999, 'Cell phones and cancer: what is the evidence for a connection?', *Radiation Research*, vol. 151, no. 5, pp. 513-531. RMA 23853

378. This review article considered the causal relationship between RF radiation from cell phones and cancer.

379. The authors provided a review of the physics and technology of cell phones.

380. The authors summarized a number of studies of workers with exposure to RF radiation and risk of leukaemia. [377]

a. Hill, DA 1998 378,

Leukaemia	RR = 0.8	95% CI 0.3 – 1.9

b. Lilienfeld, AM et al. 1978 [379],

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376 Independent Expert Group on Mobile Phones2000, *Mobile Phones and Health*, National Radiological protection Board, Chilton, United Kingdom.

377 Table 2 page 518

378 Hill, DA 1998, *Longitudinal study of a cohort with past exposure to radar: the MIT radiation laboratory follow up study*, University of Michigan dissertation Service, Ann Arbot 1988.

A case controlled cohort study of 1456 MIT laboratory workers in the development of radar applications between 1940 & 1946 with at least 30 year follow-up. The conclusion was of no increased risk of overall cancer and no evidence for an exposure – response trend in those cases with a cancer diagnosis

This article was not available to (before) the RMA at the relevant times, however the information above is cited in **Moulder, JE et al.** 1999 and as such was considered by the Council.

379 Lilienfeld, AM et al. 1978. Foreign service health status study – evaluation of health status of foreign service and other employees from selected eastern European posts: Final report. Johns Hopkins University , Baltimore, MD.

Leukaemia	SMR = 2.5	95% CI 0.3 – 9.0

c. Robinette, CD et al. 1980 [380]

Lymphatic and hematopoietic system	SMR = 1.6	not statistically significant

d. Milham, S 1988 [381]

Lymphatic and hematopoietic system	SMR = 1.2	95% CI 1.0 - 1.5
Leukaemia	SMR = 1.2	95% CI 0.9 - 1.7

e. Tynes,T 1992 [382]

Leukaemia	SIR = 2.8	95% CI 1.3 - 5.4

f. Szmigielski, S 1996 [383]

Lymphatic and hematopoietic system	IR = 6.3	95% CI 3.1 - 14

g. Muhm, J 1992 [384]

A case controlled health experience survey of 1800 foreign service employees at the American embassy in Moscow, including measurements of several different exposed areas of the Moscow embassy. They found no evidence that individuals in the Moscow group experienced higher mortality for any cause including cancer.

This article was not available to (before) the RMA at the relevant times, however the information above is cited in **Moulder, JE et al.** 1999 and as such was considered by the Council.

380 Robinette, CD et al. 1980, Effects upon health of occupational exposure to microwave radiation (radar). *American Journal of Epidemiology,* vol. 112 (1): 39-53.

381 Milham, S 1988, Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies', *Am J Epidemiol,* vol. 127, pp. 50-54.

67,829 amateur radio operators between 1979 – 1984, no exposure details provided. 98 cases Lymphatic and hematopoietic system, 36 cases of Leukaemia, 6 cases of chronic lymphocytic leukaemia.

See Table 2 at page 53 – Analysis of leukaemia deaths

chronic lymphocytic leukaemia

ICD 8 (6 cases) SMR = 1.09 95% CI 0.40 - 2.38

382 Tynes, T et al. 1992. Incidence of cancer in Norwegian workers potentially exposed to electromagnetic fields. *Am J Epidemiol* 136: pp. 81-88.

945 male Norwegian electrical workers exposed to extremely low frequencies and radio frequencies between 1961 – 1985. No exposure details provided.

This article was not available to (before) the RMA at the relevant times, however the information above is cited in **Moulder, JE et al.** 1999 and as such was considered by the Council.

383 Szmigielski, S 1996, 'Cancer morbidity in subjects occupationally exposed to high-frequency (radiofrequency and microwave) electromagnetic radiation,' *Sci Total Environ*, vol. 180, pp. 9-17.

384 Muhm, J 1992, Mortality investigation of workers in an electromagnetic pulse test program. *J Occup Med,* vol. 34, pp. 287-292.

Lymphatic and hematopoietic system	SMR = 3.3	95% CI 0.40 - 12
Leukaemia	SMR = 4.4	95% CI 0.1 – 24

381. The authors provided a summary of the evidence for the genotoxic potential of RF radiation and concluded: [385]

> Tests of the potential genotoxic activity of RF radiation have been extensive. The majority of the studies have indicated no significant genotoxicity.

382. The authors provided a summary of the evidence for the long term animal exposure studies with RF radiation:

a. Repacholi et al 1997 [386] examined the possibility that long term exposure to pulse modulated RF radiation would enhance the incidence of lymphoblastic lymphomas in transgenic mice moderately predisposed to develop lymphoblastic lymphomas.

383. Results of the authors study [387] showed the incidence of lymphoma was higher in the mice exposed to RF radiation than in the sham-exposed group. These results were not interpreted with regard to their impact on humans or more broadly to all non-Hodgkin's lymphoma such as chronic lymphocytic leukaemia.

384. With regard to the evidence for the long term animal exposure studies the authors concluded: [388]

> Taken together, these studies present no compelling evidence that long-term exposure to RF radiation has a negative impact on the general health of animals.

385. The authors provided a table summary for the weight of evidence criteria for RF radiation and cancer. [389] There was no clear conclusion and no specific outcome related to chronic lymphocytic leukaemia.

Council's comments

386. Although this review was focused upon exposure from cell (mobile) phones, Council felt the genotoxicity review confirmed others in this area.

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304 male workers in an electromagnetic pulse test program from 1970-1986. Exposure to nuclear exposition related electromagnetic pulses with frequency range 10 kHz – 100 MHz with potential exposure of 30 days to 6 months.

385 Page 523-524.

386 Repacholi et al 1997. Lymphomas in Eμ -PIM1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields. *Radiat. Res* 147, 631-640.

387 Table 6 page 528.

388 Page 528.

389 Table 7 page 529.

387. The Council considered this study:

– did not point to, but merely left open the possibility of a relevant association.

California Department of Health Services (DHS) Anon 2001, An evaluation of the possible risks from electric and magnetic fields (EMFs) from power line, internal wiring, electrical occupations and appliances: Executive summary, Draft 3 for public Comment, California Public Utilities Commission: 1-19, 72-109. RMA ID 22112 FILEForce ID 18538

388. This is a report from the California Public Utilities Commission in which three scientists are asked to review studies about the possible health problems from electric and magnetic fields (EMF) from power lines, wiring in buildings, certain jobs and appliances.

389. They examined epidemiological findings in order to develop a degree of confidence using the IARC criteria, [390] which bases the assessment on the quality of evidence, against the California guidelines which base their estimate on the degree of confidence, and considers emerging evidence.

390. The evaluation also included extensive consultation with the Department of Health Services epidemiologists and toxicologists, evaluating the training, the a priori views of the reviewers, the process of risk estimation and the comparison of this evaluation to the evaluation of other health organisations so that this report could be considered well calibrated.

391. In the opening general public statement, the authors did not discuss the risk of chronic lymphocytic leukaemia.

392. The three authors summarized their opinion on the role of EMF as a cause of adult leukaemia with one review considering EMF to be causal and the other 2 considering EMF to be a possible cause for leukaemia. [391]

393. The authors based their conclusions regarding the role of EMF in adult leukaemia on Kheifets 1997 [392] and provided a graph of the pattern of epidemiological evidence. [393]

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390 The Council considered that the California Department of Health Services (DHS) Unnamed Scientists 2001 decisions and the therein cited IARC carcinogen protocol did not reflect the Council's two-step process as set down by the full Federal Court (see [108] to [110])

391 Page 72.

392 Kheifets LI, London SJ, Peters JM (1997). Leukemia risk and occupational electric field exposure in Los Angeles County, California. *American Journal of Epidemiology*, 146(1) pp 87-90. RMA ID 21004 FILEForce ID 18910

393 Figure 8.1 page 73 and the Summary Table 8.1.4 pp. 80-86 – based primarily on the data from Kheifets, LI et al. 1997.

394. The authors summarized the epidemiological studies including an analysis of chronic lymphocytic leukaemia.

a. Floderus, B et al. 1993 and 1992 [394] See [168]

– Study population: males in 1980 employed and living in Sweden from 1983 – 1987. 250 cases of leukaemia

All leukaemia	OR = 1.5	95% CI 1.1 – 2.0
chronic lymphocytic leukaemia	OR = 2.5	95% CI 1.6 – 3.9

b. Floderus, B et al. 1994; Tornquist, 1991; Linet et al. 1988 (7); Tornquist 1986 [395]

– Study population: 1,906,660 men employed in 1960 (133,687 in selected electrical occupations) in Sweden from 1961 – 1979 – 334 cases of leukaemia.

All leukaemia	SMR = 1.1	95% CI 0.9 – 1.4
chronic lymphocytic leukaemia	SMR = 1.2	95% CI 0.8 – 1.8

c. London et al. 1994[396] and Wright 1982 [397]

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394 Floderus, B et al. 1993 and 1992 – Table 8.1.4 – No further details of the citation for this study was provided in the **California DHS,** 2001 available information sent by the RMA to the SMRC. The Council notes the following study and data:

Floderus B, Persson T, Stenlund C, Wennberg A, 1993, Occupational Exposure to Electromagnetic Fields in Relation to Leukemia and Brain Tumours: A Case-Control Study in Sweden*. Cancer Causes and Control*, Vol 4 pp 465-476 RMA ID 1669 FILEForce ID 18822 See Table 3, p. 470:

0.16 – 0.19 μT	OR = 1.1	95% CI 0.5 – 2.3
0.20 – 0.28 μT	OR = 2.2	95% CI 1.1 – 4.3
≥ 0.29 μT	OR = 3.0	95% CI 1.8 – 7.7

395 Floderus, B et al. 1994; Tornquist, 1991; Linet et al. 1988 (7); Tornquist 1986 Table 8.1.4 – No further details of the citation for this study was provided in the **California DHS,** 2001 available information sent by the RMA to the SMRC. The Council notes the following study and data:

Floderus, B Tornqvist, S Stenlund, C 1994, 'Incidence of selected cancers in Swedish railway workers, 1961-79', *Cancer Causes and Control*, vol. 5, pp. 189-194. See Table 2, p. 192:

1961-69 – n = 39	RR range 1.3 – 2.7	95% CI's not statistically significant
1970-79 – n = 51	RR range 0.5 – 1.1	95% CI's not statistically significant

396 Table 8.1.4 – No further details of the London et al. 1994 and Wright et al. 1982, citations for these studies was provided in the **California DHS,** 2001 available information sent by the RMA to the SMRC. The Council notes the following study and data:

London, S et al. 1994, Exposure to magnetic fields among electrical workers in relation to leukemia risk in Los Angeles County Am J Ind Med 26: 47- 60. Average magnetic field exposure data at Table VII, p. 56.

18 chronic lymphocytic leukaemia cases leukaemia	Exposure 0.18 – 0.8 μT RR = 1.6	chronic lymphocytic 95% CI 1.2 – 2.3
4 chronic lymphocytic leukaemia cases	Exposure > 0.8 μT	
chronic lymphocytic leukaemia	RR = 0.8	95% CI 0.4 – 1.5

397 Wright et al. 1982,' Leukaemia in workers exposed to electrical and magnetic fields', *Lancet* 2, pp. 1160-61.

– Study Population: Cases among males with known occupation in Los Angeles county cancer registry 1972 – 1990 – 2,355 cases of leukaemia.

All leukaemia	OR = 1.3	95% CI 1.1 – 1.6
chronic lymphocytic leukaemia	OR = 1.3	95% CI 1.0 – 1.8

d. Theriault et al 1994 [398] - See from [203]

– Study population: 170, 000 male utility workers in France from 1978 – 1989 – 71 cases of leukaemia

All leukaemia	OR = 1.4	95% CI 0.6 – 3.1
chronic lymphocytic leukaemia	OR = 4.8	95% CI 0.5 – 70.6

e. Theriault et al 1994 [399] - See from [203]

– Study population: 31, 543 male utility workers in Ontario Canada 1973 – 1988 – 45 cases of leukaemia.

All leukaemia	OR = 3.1	95% CI 1.1 – 9.7
chronic lymphocytic leukaemia	OR = 2.1	95% CI 0.4 – 11.6

f. Theriault et al 1994 [400] - See from [203]

– Study population: 21, 749 male utility workers in Quebec Canada 1973 – 1988 – 24 cases of leukaemia.

All leukaemia	OR = 0.3	95% CI 0.04 – 1.8
chronic lymphocytic leukaemia	OR = 0.3	95% CI 0.02 – 2.6

g. Tynes, 1992 [401]

This article was not available to (before) the RMA at the relevant times, however the information above is cited in **California DHS,** 2001 and as such was considered by the Council.

398 Table 8.1.4 – No further details of the Theriault, G et al. 1994, citation for this study (footnotes 397-399) was provided in the **California DHS,** 2001 available information sent by the RMA to the SMRC. The Council notes the following study and data (See from [203]):

Theriault G, Goldberg M, Miller AB, et al (1994). Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec, Canada, and France:1970-1989. *Am J Epidemiol*, 139(6) pp 550-572. RMA ID 10435 FILEForce ID 18845.

399 Theriault G, Goldberg M, Miller AB, et al (1994). Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec, Canada, and France:1970-1989. *Am J Epidemiol*, 139(6) pp 550-572. RMA ID 10435 FILEForce ID 18845.

400 Theriault G, Goldberg M, Miller AB, et al (1994). Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec, Canada, and France:1970-1989. *Am J Epidemiol*, 139(6) pp 550-572. RMA ID 10435 FILEForce ID 18845.

401 Table 8.1.4 – No further details of the Tynes et al. 1992, citation for this study (footnotes 398 - 400) was provided in the **California DHS,** 2001 available information sent by the RMA to the SMRC. The Council notes the following study and data cited and reported in **Kheifets, LI et al.** 1997. The Tynes, T et al, 1992 article was not available to (before) the RMA at the relevant times, however the information above is cited in **California DHS,** 2001 and in **Kheifets, LI et al.** 1997, and as such was considered by the Council.

Tynes, T et al. 1992. Incidence of cancer in Norwegian workers potentially exposed to electromagnetic fields. *Am J Epidemiol* 136: 81-8837.

– Study population: 37, 945 male Norwegian electrical workers, 1961 – 1985 – 107 cases of leukaemia

All leukaemia	SIR = 1.1	95% CI 0.9 – 1.3
chronic lymphocytic leukaemia	SIR = 1.0	95% CI 0.6 – 1.4

h. Loomis 1991 and 1990 [402]

– Study population: 410, 651 male deaths in 16 US states, 1985 – 1986 – 3,400 cases of leukaemia.

All leukaemia	OR = 1.0	95% CI 0.8 – 1.2
chronic lymphocytic leukaemia	OR = 0.6	95% CI 0.3 – 1.1

i. Pearce 1989, (Pearce et al.) 1986 and 1985 [403]

– Study population: New Zealand cases among males from NZ cancer registry 1979 – 1983 – 546 cases of leukaemia.

All leukaemia	OR = 1.6	95% CI 1.0 – 2.5
chronic lymphocytic leukaemia	OR = 3.4	95% CI 1.38 – 8.9

j. Milham 1988 and 1985 [404]

– Study population: US deaths among 67,829 male licensed amateur radio operators 1979 – 1984 – 36 cases of leukaemia.

All leukaemia	SMR = 1.2	95% CI 0.9 – 1.7

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945 male Norwegian electrical workers exposed to extremely low frequencies and radio frequencies between 1961 – 1985. No exposure details provided.

402 Loomis DP, Savitz DA 1991, Occupation and leukemia mortality among med in 16 states : 1985 – 1987 Am J Ind Med 19 509-521 US: cases among 410,651 male deaths in 16 US states, 1985-1986

403 Pearce, NE et al. 1989, Case-control studies of cancer in New Zealand electrical workers *Int J Epidemiolo* 18: 55-59.

Pearce, NE et al. leukaemia among New Zealand agricultural workers: a cancer registry-based study,' *Am J Epidemiol*, vol. 124, pp. 402 – 409.

Pearce, NE et al. Leukaemia in electrical workers in New Zealand, *Lancet*, pp. 811-812.

These 3 articles were not available to (before) the RMA at the relevant times, however the information above is cited in **California DHS,** 2001 and reported in **Kheifets, LI et al.** 1997and as such was considered by the Council.

404 Milham, S 1988, Mortality by license class in amateur radio operators. *Am J Epidemiol,* 128: 1175-1176

67,829 amateur radio operators between 1979 – 1984, no exposure details provided. 98 cases Lymphatic and hematopoietic system, 36 cases of Leukaemia, 6 cases of chronic lymphocytic leukaemia.
See Table 2 at page 53 – Analysis of leukaemia deaths – ICD 8:

Leukaemia	SMR = 1.24	95% CI 0.87-1.72
chronic lymphocytic leukaemia	SMR = 1.09	95% CI 0.4 - 2.38

Milham, S 1985,' Mortality in workers exposed to electromagnetic fields,' *Environ Health Perspect*, vol. 62, pp. 297-300.

The 1985 article was not available to (before) the RMA at the relevant times, however the information above is cited in **California DHS,** 2001 and reported in **Kheifets, LI et al.** 1997 and as such was considered by the Council.

chronic lymphocytic leukaemia	SMR = 1.1	95% CI 0.4 – 2.4

k. Gilman et al 1985 [405]

– Study population: 19,000 male coal miners with 6,066 death certificates reviewed prior to 1985, increased leukaemia risk – 40 cases of leukaemia

All leukaemia	OR = 2.5	95% CI 1.1 – 5.9
chronic lymphocytic leukaemia	OR = 6.3	$P < 0.05$

l. Coleman 1983 [406]

– Study population: 6.5 million English patients identified through the South Thames Cancer registry 1961 – 1979 – 113 cases of leukaemia and 33 cases of chronic lymphocytic leukaemia.

All leukaemia	PIR = 1.2	95% CI 1.0 – 1.4
chronic lymphocytic leukaemia	PIR = 1.3	no CI provided

Council's comments

395. The Council notes the findings presented in Table 8.1.4 with 11 of the 44 studies including a finding (RR or OR) for chronic lymphocytic leukaemia, nine studies greater or equal to unity and two studies less than or equal to unity. Three (3) studies exclude unity namely Floderus, B et al, 1993, OR 2.5 (1.6-3.9); Pearce, NE et al. 1986, OR 3.4 (1.38-8.9) and Gillman et al. 1985, OR 6.3 ($P < 0.05$).

396. In the executive summary it is stated [407] that there is a greater than 50% chance that there is an increase lifetime risk of childhood leukaemia, adult brain cancer, motor neurone disease and miscarriage from exposure to electromagnetic fields at home or at work.

397. All three reviewers say there is "at least" a 10 – 50% likelihood of a small life time risk of adult leukaemia and one reviewer said that the risk may be higher than that estimate.

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This article was not available to (before) the RMA at the relevant times, however the information above is cited in **California DHS,** 2001 and reported in **Kheifets, LI et al.** 1997and as such was considered by the Council.

406 Coleman, M et al. 1983, 'Leukaemia incidence in electrical workers', *Lancet*, pp. 982-983. – 33 chronic lymphocytic leukaemia cases.

This article was not available to (before) the RMA at the relevant times, however the information above is cited in **California DHS,** 2001 and reported in **Kheifets, LI et al.** 1997and as such was considered by the Council.

407 The Council considered that the California Department of Health Services (DHS) Unnamed Scientists 2001 decisions and the therein cited IARC carcinogen protocol did not reflect the Council's two-step process as set down by the full Federal Court (see [108] to [109]).

398. The Council notes that the California DHS 2001 report may not be peer reviewed, but that the studies reviewed were. How the studies were selected is not clear, which is problematic as the selection of studies may bias the findings of the report.

399. The Council considered that this study to be an important paper due to rigorous review by three reviewers being in agreement regarding a 10-50% risk of adult Leukaemia from EMF, possibly higher according to 1 of 3 reviewers.

400. The Council considered this study weakly supported an association between chronic lymphocytic leukaemia and radiation exposure and;

- points to a relevant association.

Breckenkamp, J Berg, G and Blettner, M 2003, 'Biological effects on human health due to radiofrequency/microwave exposure: a synopsis of cohort studies', *Radiation & Environment Biophysics*, vol. 42, no. 3, pp. 141-154. RMA ID 30499 FILEForce ID 19541

401. This cohort study meta-analysis reviewed nine cohort studies published between 1980 and 2002 examining the biological effects on human health from exposure to radiofrequencies / microwaves.

402. The authors provided a summary of each studies reported mortality ratio for haematopoietic malignancies. [408]

a. Muhm, 1992 [409]

- 304 male workers in an electromagnetic pulse test program from 1970-1986. Exposure to nuclear exposition related electromagnetic pulses with frequency range 10 kHz – 100 MHz with potential exposure of 30 days to 6 months.

Hematopoietic system (2 cases)	SMR = 3.31	95% CI 0.40 - 11.96
Lymphoma (1 case)	SMR = 10.87	95% CI 0.28 - 60.56
Leukaemia (1 case)	SMR = 4.37	95% CI 0.11 – 24.33

b. Robinette, et al. 1980 [410]

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408 Table 3 page 147 – 148.

409 Muhm, J 1992, 'Mortality investigation of workers in an electromagnetic pulse test program', *J Occup Med,* vol. 34, pp. 287-292.

410 Robinette, CD et al. 1980, 'Effects upon health of occupational exposure to microwave radiation (radar)', American Journal of Epidemiology, vol. 112, no. 1, pp. 39-53.

See Table 6 at page 45 – ICD 8th Revision – Number of deaths and mortality ratios by exposure class:

Low exposure - 20 cases	SMR = 0.83

– 40,890 US Navy men who served during the Korean War 1950-1974. Exposure to microwave radiation (radar) for electronic equipment repair (maximum opportunity for exposure = 26 deaths) and equipment operation (minimum potential for exposure = 20 deaths).

Lymphatic and hematopoietic system	MR = 1.18	no CI provided

c. Milham, S 1988 [411]

– 67,829 amateur radio operators between 1979 – 1984, no exposure details provided. Radio frequency radiation exposure.

Lymphatic and hematopoietic system	SMR = 1.23	95% CI 0.99-1.52;
Leukaemia	SMR = 1.24	95% CI 0.87-1.72

d. Morgan, RW et al. 2000 [412] - (See Non- Contributory list)

– 195,775 employees of Motorola 1976-1996. Exposure Radio frequencies (wireless communication technologies).

Lymphatic and hematopoietic system	SMR = 0.54	95% CI 0.33 – 0.83
Leukaemia	SMR = 0.77	95% CI 0.38 - 1.38

e. Szmigielski, S 1996 [413] - See from [248]

High exposure – 26 cases SMR – 1.18

See Table 8 at page 46 – Mortality from malignant neoplasms of the lymphatic and hematopoietic system

Lymphatic leukaemia (no cases designated chronic lymphocytic leukaemia)

low exposure = 2 high exposure = 2

411 Milham, S 1988, Mortality by license class in amateur radio operators. *Am J Epidemiol* 128: 1175-1176

67,829 amateur radio operators between 1979 – 1984, no exposure details provided. 98 cases Lymphatic and hematopoietic system, 36 cases of Leukaemia, 6 cases of chronic lymphocytic leukaemia.

See Table 2 at page 53 – Analysis of leukaemia deaths – ICD 8:

chronic lymphocytic leukaemia	SMR = 1.09	95% CI 0.4 - 2.38

412 Morgan, RW et al. 2000, 'Radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems', *Comment on: Epidemiology*, 11(2) pp. 118-27. RMA ID 28377 FILEForce ID 18645

203 cases for Lymphatic and hematopoietic system (91 were Non-Hodgkin's Lymphoma) and 87 cases for leukaemia.

413 Szmigielski, S 1996, Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. *Science of The Total Environment* Volume 180, Issue 1, 2 February 1996, Pages 9-17. RMA ID 10413 FILEForce ID 18848

– 128,000 military career personnel 1971-1085. Exposure to Radio frequencies /microwaves (pulse-modulated high frequency electromagnetic fields) range 150 MHz to 3.5 GHz.

Lymphatic and hematopoietic system	OER = 6.31	95% CI 3.12 – 14.32

f. Groves, FD et al. 2002 [414] - See from [235]

– 40,890 US Navy personnel who served during the Korean War 1950-1997. Exposure to Microwaves (radar equipment)

Lymphoma and multiple myeloma	SMR = 0.89	95% CI 0.72 – 1.09
Leukaemia	SMR = 1.14	95% CI 0.90 - 1.44

g. Tynes, et al. 1992 [415]

– 37,945 male Norwegian electrical workers exposed to extremely low frequencies and radio frequencies between 1961 – 1985. No exposure details provided.

non-Hodgkin's lymphoma	SIR = 0.77	95% CI 0.60-0.98;
Leukaemia	SIR = 1.08	95% CI 0.89-1.31

h. Finkelstein, MM 1998 [416]

– 22,197 Ontario Police offers exposed to microwaves (traffic radar units) between 1970 – 1995. Frequency range 10.525 GHz (early devices), 24.15 GHz (since 1975), 35.0 GHz (mid 1990).

Leukaemia	SIR = 0.60	95% CI 0.31-1.05

403. The authors discussed the limitations of the studies with regard to outcome reporting, no specific comment on the outcomes for chronic lymphocytic leukaemia were discussed.

Council's comments

404. The Council noted that there are some biases in these studies and there is not a consistent result.

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414 Groves, FD et al. 2002, 'Cancer in Korean War Navy technicians: mortality survey after 40 years,' *American Journal of Epidemiology*, vol. 155(9), pp. 810-818. RMA ID 25344 FILEForce ID 18500

415 Tynes et al 1992. Incidence of cancer in Norwegian workers potentially exposed to electromagnetic fields. *Am J Epidemiol* 136: 81-88 37.

This article was not available to (before) the RMA at the relevant times; however the information above is cited/reported in **Moulder, JE et al.** 1999 and cited/reported in **Breckenkamp, J et al.** 2003 and as such was considered by the Council.

416 Finkelstein MM 1998 Cancer incidence among Ontario police officers. *Am J Ind Med* 34: 157-162.

405. Also, the literature search is summarised in table 3 in this paper showing an increase in haematological malignancies with confidence intervals excluding unity in the Smigelski and Milham studies. The Szmigielski was the only positive study.

406. The Council considered this study:

– does not point to but merely left open the possibility of a relevant association.

Elwood, JM 1999, A critical review of epidemiologic studies of radiofrequency exposure and human cancers. *Environ Health Perspect*, Feb, vol. 107, Suppl. 1, pp. 155-168. RMA ID 26009

407. This is a very extensive systematic literature review of the epidemiological studies on RF in particular, published during 1988-98.

408. It is noted that there are a number of background concerns amongst the public and that the ICNIRP (International Committee On Non-Ionising Radiation Protection, discussed above) bases its guidelines for RF exposures on avoiding thermal effects of electromagnetic radiation restricting whole body to less than 0.4 watt/kg occupational, and 0.08 watt/kg in the general population. This is based on data showing a threshold for the most sensitive tissues of 4 watts/kg and the question is whether there are any effects caused by electromagnetic radiation below these thermal thresholds.

409. The ICNIRP concluded that low level exposure is “unlikely” to initiate cancer that there have been many negative studies in vitro evaluating DNA damage and mutations, and that there are some positive studies of strand breaks with questionable methodology, and that some studies have shown malignancies in rats from microwaves or promotion of previously initiated tumours.

410. Of note, one study [417] showed an increase incidence of lymphomas in genetically predisposed mice at likely sub thermal levels. A number of biochemical effects on calcium and brain electrical activity and t-cytotoxic activity and others have been noted.

411. Overall the comment is that the results of these studies have been mixed and therefore it is difficult to argue that the carcinogenic mechanism can be ruled out.

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[417] Repacholi, MH et al. 1997, ‘Lymphomas in Eμ-Pim 1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields,’ Radiat Res, vol. 147, pp. 631-640.

Lymphoma RR = 2.4 95% CI 1.3 – 4.5

This article was not available to (before) the RMA at the relevant times, however the information above is cited/reported in **Elwood, JM** 1999 and as such was considered by the Council.

412. The current review was prompted by a lack of good epidemiological reviews by the ICNIRP and recent data that was not included in that report, particularly by Bergqvist 1997 [418] .

413. A literature search from 1988 to1998 was undertaken for epidemiological studies focused on radiofrequency rather than extremely low frequency radiation, in particular in humans.

414. A number of categories of study were outlined as follows:

Cluster studies:

415. Three studies looking at leukaemia, testicular tumours and leukaemia and lymphoma were hypothesis generating only (i.e. looking at the data to determine future research possibilities).

a. The Sutton Coldfield study [419] showed an OR of 2.56 (95% CI 1.11 – 5.05) with a P value of 0.007, for chronic lymphocytic leukaemia. Also, see from [262]

General population with exposure to TV and radio studies:

416. The studies in the general population with exposures included five studies looking at T.V. and radio signals and these included a further analysis of the Sutton Coldfield study, a study a 21 UK transmitters.

a. Dolk, H et al. 1997b [420] - a chronic lymphocytic leukaemia OR of 1.2 (95% CI 0.83 - 1.46), and other leukaemia OR were not elevated. Also see from [269]

b. The Sydney study [421] - a trend of leukaemia in children, with further analysis showing a borderline significant increase in childhood leukaemia. Also see [285]

c. A San Francisco study [422] that showed statistical non-significant data for Lymphoma and leukaemia RR = 1.03.

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418 Bergqvist, U 1997, *Review of epidemiological studies*, pp. 147-170. In Kuster, N et al eds. 1997, *Mobile Communications Safety*, Chapman & Hall, London.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

419 Table 1 at p. 156 - Dolk, H, et al. 1997a, **'**Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield Transmitter', *American Journal of Epidemiology*, 145(1) pp 1-9. RMA ID 9620 FILEForce ID 18858

420 Table 2 at p. 158 - Dolk, H et al 1997, 'Cancer incidence near radio and television transmitters in Great Britain. II. All High Power Transmitters', *American Journal of Epidemiology*, vol. 145, no. 1, pp. 10-17.

421 Table 2 at p. 158 - Hocking, B et al. 1996, 'Cancer incidence and mortality and proximity to TV towers', *Medical Journal of Australia*, 165(2) pp 601-605.

422 Table 2 at p. 158 - Selvin, S et al. 1992, 'Distance and risk measures for the analysis of spatial data: a study of childhood cancers', *Soc Sci Med*, vol. 34, pp. 769 – 777.

d. A Honolulu study [423] that showed an increased in adult leukaemia but methodological problems are reported.

Studies of Occupational groups:

417. There were 5 cohort studies reviewed: [424]

a. The Polish military study [425] showed a OER = 3.68 (95% CI 1.45 – 5.18) increase in chronic lymphocytic leukaemia. See from [248]

– but this study was possibly suffering from bias due to more sources of information on radiofrequency exposure, i.e. hospital records, in cases than in controls.

– An increase in all cancer types and an increase in relative risk of leukaemia and lymphoma of OER = 6.31 (95% CI 3.12 – 14.32) was noted and similar increase in cancers that have otherwise rarely been implicated in non ionising radiation.

b. The US navy study [426] was a negative study comparing very high and low dose exposures that have no unexposed control.

> Confidence limits and a test of trend over categories of exposure have been applied to the Robinette et al published data because the original did not present such measures….
>
> Lymphatic and hematopoietic OER = 1.64 95% CI 0.70 – 3.25

c. US amateur radio operators study [427] suffered from a lack of information on other sources of exposure, of note there was an increase of AML SMR = 1.76 (95% CI 1.03 – 2.85) and "other lymphatic tissue" cancer SMR = 1.62 (95% CI 1.17 – 2.18), all others were lower.

This article was not available to (before) the RMA at the relevant times, however the information above is cited/reported in **Elwood, JM** 1999 and as such was considered by the Council.

423 Environmental Epidemiology Program 1986, Cancer incidence in census tracts with broadcasting towers in Honolulu, Hawaii, Report submitted to the Honolulu City Council, Hawaii. In Dolk, H et al. 1997a at p. 17.

Leukaemia RR = 1.56 99% CI 0.86 – 2.63

This article was not available to (before) the RMA at the relevant times, however the information above is cited in **Elwood, JM** 1999 and is cited/reported in Dolk, H et al. 1997a, and as such was considered by the Council.

424 See Table 3, page 161.

425 Szmigielski, S 1996, Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. *Science of The Total Environment* Volume 180, Issue 1, 2 February 1996, Pages 9-17. RMA ID 10413 FILEForce ID 18848

426 Robinette, CD et al. 1980, 'Effects upon health of occupational exposure to microwave radiation (radar)', *American Journal of Epidemiology,* vol. 112, no. 1, pp. 39-53.

427 Milham, S 1988, Mortality by license class in amateur radio operators. *Am J Epidemiol* 128: 1175-1176.

Lymphatic and hematopoietic	SMR = 1.23	95% CI 0.99 – 1.52
Leukaemia	SMR = 1.24	95% CI 0.87 – 1.72
chronic lymphocytic leukaemia	SMR = 0.86	95% CI 0.17 – 2.50

d. Also, a Norwegian radio operators study [428] looking at breast cancer risk found no significant increase in leukaemia or lymphoma and this was considered to be an interesting observation. The data for leukaemia was:

Leukaemia	RR = 1.1	95% CI 0.1 – 4.1

e. The Electric utility workers in Canada study [429] showed no increase in leukaemia or lymphoma, brain tumours or melanoma. For chronic lymphocytic leukaemia an RR = 2.98 (95% CI 0.21 – 2.38). See from [203]. There was a significant association with lung cancer with a RR = 3.11 (95% CI 1.60 – 6.04) which was unexpected and this was considered to be methodologically the strongest of the studies reviewed but applied to pulsed EMF (5 to 20 MHz,) and was negative for leukaemia (which, however, was increased in the Polish study (See from [248]); and was positive for lung cancer which was negative in the Polish study.

Case control studies:

418. The author reviewed 6 case-control studies, [430] but none of these reported data in respect of leukaemia or lymphatic cancers.

419. Other human studies, Australian telecom, [431] no increase in chromosomal aberrations, in vitro blood exposed to high dose microwave, increased chromosomal aberrations and micro nuclei.

Council's comments

420. This was a systematic search of published data looking at radiofrequency exposure which did not include electromagnetic fields in general or unpublished or government

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428 Tynes et al 1996. 'Incidence of breast cancer in Norwegian female radio and telegraph operators', *Cancer Causes Control,* vol. 7, pp. 197-204.

This article was not available to (before) the RMA at the relevant times, however its abstract was available to (before) the RMA, and the information above is cited/reported in **Elwood, JM** 1999, and as such was considered by the Council.

429 Theriault, G et al. 1994, Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec, Canada, and France: 1970-1989. *Am J Epidemiol*, 139(6) pp 550-572. RMA ID 10435 FILEForce ID 18845.

430 See Table 4, page 163.

431 Garson, OM et al. 1991, 'A chromosomal study of workers with long term exposure to radiofrequency radiation,' *Med J Aust*, vol. 155, pp. 289-292.

reports. There was a discussion of the Lilienfeld study [432] of US embassy workers in Russia, showing an increase in leukaemia and lymphoma, but this study suffered multiple methodological problems. The commentary by Bergqvist [433] suggested that there was no overall increase in cancer and a non-significant increase in leukaemia and breast cancer (two cases). Goldsmith's [434] commentary suggested that no valid conclusions could be drawn from the Lilienfeld study data.

421. Limitations on studies of radiofrequency exposure- these are either case control studies with poor estimates of exposure or cohort studies and that the population studies are subject to the ecological fallacy.

422. It is noted that many studies suffered from bias, confounding and chance variation factors.

423. The assessment of the available data suggests that exposure data from residential exposure is poor and that occupational exposure assessments are better but are indirect. Time relationships are poorly documented and the strength of association varies. The strongest come from the Polish data (See Szmigielski above and from [248]) but the problem is that this showed an increase in all cancers possibly due to bias. The association was strong in utility workers but was for lung cancer not seen in other studies and in the Sydney study (See Hocking above and from [285]) there was childhood leukaemia but the UK study (See Sutton Coldfield above and from [262] Dolk, H et al. 1997a) was equivocal and dose response finding have been inconsistent. Overall there was a lack of consistent and specific effect across studies and figure 1 in this manuscript shows a spread of outcomes in adult and childhood leukaemia. There is also concern about biologic plausibility with conflicting data.

424. The conclusions of this study are that in relation to radiofrequency exposure there are inconsistent results, with no type of cancer consistently associated. However, it is also noted that this is a similar situation to early phase studies of tobacco and asbestos. Currently the data falls short on strength and consistency.

425. The review stressed that there are inconsistent results and came to no clear conclusions.

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[432] Lilienfeld, AM et al. 1978, Foreign service health status study – evaluation of health status of foreign service and other employees from selected eastern European posts. Final Report (Contract number 6025-619073) to the U.S Dept of State.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

[433] Bergqvist, U 1997, *Review of epidemiological studies*, pp. 147-170. In Kuster, N et al eds. 1997, *Mobile Communications Safety*, Chapman & Hall, London.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

[434] Goldsmith, JR 1995, 'Epidemiologic evidence of radiofrequency radiation (microwave) effects on health in military, broadcasting, and occupational studies,' *International journal of occupational & environmental health*, vol. 1(1) pp. 47-57.

426. The Council considered that this was an important, if now dated, review that fails to provide much support for causation of chronic lymphocytic leukaemia by radiation.

427. The Council considers this review:

- does not point to but merely leaves open the possibility of a relevant association.

Elwood, JM 2003, Epidemiological Studies of Radio-Frequency Exposures and Human Cancer. *Bioelectromagnetics Supplement*, vol 6, pp. S63-S73. RMA ID 30503 FILEForce ID 19503

428. This review article considered whether the available epidemiological studies of radio frequency exposures is associated with and increased risk of human cancer. The review discusses the Morgan, RW et al. 2000, Motorola study.

429. Studies reviewed that included an assessment of cancers of the adult haematopoietic system included:

a. Dolk, H et al. 1997a & b [435] (for 1997a see Elwood, JM 1999 [415.a], and from [262] and for 1997b see [416.a] and from [269]. For Cooper et al. 2001 [436] – see from [274].

b. Hocking, B et al. 1996 [437] See Elwood, JM 1999 [416.b] and from [285].

c. Szmiegielski, S 1996 – see Elwood, JM 1999 [417.a] and from [248].

d. Robinette et al. 1980 [438] - see Elwood, JM 1999 [417.b].

e. Milham, S 1988 [439] - see Elwood, JM 1999 [417.c].

f. Michelozzie et al. 2002 [440]

- Studied the mortality from adult leukaemia and the incidence of childhood leukaemia in relationship to the Vatican radio station. No data is provided.

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435 Dolk, H et al. 1997a, 'Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield Transmitter', *American Journal of Epidemiology*, 145(1) pp 1-9.. RMA ID 9620 FILEForce ID 18858

436 Cooper, D et al 2001, [COMMENT] 'Re Cancer incidence near radio and television transmitters in Great Britain. 1. Sutton Coldfield transmitter; II All high power transmitters', Am J of Epidemiology, vol. 153, pp. 202-204, Erratum Am J Epidemiol, 2007, pp. 166:1107.

437 Hocking, B et al. 1996, 'Cancer incidence and mortality and proximity to TV towers', *Medical Journal of Australia*, vol. 165, no. 2, pp 601-605. RMA ID 21127 FILEForce ID 18995

438 Robinette, CD et al. 1980 Effects upon health of occupational exposure to microwave radiation (radar). *American Journal of Epidemiology,* 112, pp. 39-53.

439 Milham, S 1988 Mortality by license class in amateur radio operators. *Am J Epidemiol* 128: 1175-1176.

440 Michelozzie, P et al. 2002, 'Adult and childhood leukemia near a high-power radio station in Rome Italy', *Am J Epidemiol,* vol. 155, pp. 1096-1103.

There was a statistically significant decline in leukemia mortality with increasing distance from the transmitters for men, but no association for women and a nonsignificant decrease in risk for both sexes combined. [441]

g. Morgan, RW et al. 2000 [442] – (See Non- Contributory list)

– The authors report that the study is limited by the use of a qualitative job exposure matrix rather than actual exposure measurements and the young age of the cohort.

All employees:		
Lymphatic and hematopoietic	SMR = 0.77	95% CI 0.67 – 0.89
Lymphomas and leukaemias	SMR = 0.54	95% CI 0.33 – 0.83
High RF exposure category employees:		
Lymphatic and hematopoietic	RR = 0.70	95% CI 0.27 – 1.47
Leukaemias	RR = 0.99	95% CI 0.39 – 2.09
non-Hodgkin's lymphoma	RR = 0.58	95% CI 0.12 – 1.74

h. Groves, FD et al. 2002 [443] (a follow-up of Robinette et al. 1980) – Also see from [235]

i. Johansen et al. 2001 [444]

– 420, 095 Danish cell phone subscribers, with 58% using a digital system and the remainder an analogue system. No Leukaemia or chronic lymphocytic leukaemia data is provided.

There was no increase in salivary gland tumors or leukaemia. The pattern of incidence ratios is consistent with a distribution of mobile phone use characterized by higher socioeconomic status, and as a correlate, a lower rate of smoking. The study was not able to assess intensity of use and the average period of follow-up was only 3.1 years. [445]

430. The author concluded: [446]

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441 See page S64.

442 Morgan, RW et al. 2000, 'Radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems. Comment on: Epidemiology, vol. 11(2), pp. 118-27. RMA ID 28377 FILEForce ID 18645

443 Groves, FD et al. 2002, 'Cancer in Korean War Navy technicians: mortality survey after 40 years. American *Journal of Epidemiology*, vol. 155(9), pp. 810-18. RMA ID 25344 FILEForce ID 18500

444 Johansen et al 2001 Cellular telephones and cancer – a nationwide cohort study in Denmark,' *J Natl Cancer Inst*, vol. 93, pp. 203-207.

445 See page S66.

446 Page S69.

> The epidemiological evidence does not give clear or consistent results which indicate a causal role of RF exposures in human cancer.

431. The authors of this review conclude that:

> The epidemiological results fall short of the strength and consistency of evidence that is required to come to a conclusion that RF emissions are a cause of human cancer.

Council's comments

432. The Council considered this review, as for the previous Elwood, JM et al. 1999 review:

- does not point to but merely leaves open the possibility of an association.

Lai, H & Singh, N 1995, 'Acute low-intensity microwave exposure increased DNA single-strand breaks in rat brain cells', *Bioelectromagnetics*, vol. 16, pp. 207-210. RMA ID 5220 FILEForce ID 18757

433. This experimental study reports the results of levels of DNA single strand breakage in brain cell assays following acute exposure to low intensity microwaves in male Sprague Dawley rats.

434. The authors report the following results:

a. In rats exposed to pulsed microwaves for 2 hours, increases in brain cell DNA single-strand breaks were not observed immediately but were observed in a dose rate dependent manner at 4 hours post exposure; and

b. in rats exposed to 2 hours of continuous wave 2450 MHZ microwaves increases in brain cell DNA single-strand breaks were observed both immediately as well as at 4 hour post exposure.

435. The authors did not discuss the implication for humans apart from human brain cells.

Council's comments

436. The Council considered the key finding was that there was an increase in single strand breaks immediately after exposure. The authors comment that the mechanism for this is uncertain. The authors performed a similar experiment (Lai, H & Singh, N [447]) to find an increase in the number of doubles-stranded DNA breaks as well.

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[447] Lai, H & Singh, P 1996, 'Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation', *Int J Radiat Biol*, vol. 69, pp. 513-521.
Lai, H & Singh, P 1997, 'Acute exposure to 60 Hz magnetic field increases DNA strand breaks in rat brain cells', *Bioelectromagnetics*, vol. 18, no. 2, pp. 5-15.

437. Council was of the view this study:

- supports biological plausibility of an association between microwaves and cancers.

Utteridge, TD et al. 2002, 'Long-term exposure of Emicro-Pim1 transgenic mice to 898.4 MHz microwaves does not increase lymphoma incidence', Radiation Research, vol 158, no. 3, pp. 357-364. RMA ID 26556, FileForce 18482

438. In this study 120 Eμ-Pim1 heterozygous mice and 120 wild type mice were exposed for 1h our/day 5 days per week for up to 104 weeks.

439. No differences were found.

Council's comments

440. Council notes that contrary to the findings of Repacholi et al (below), no significant differences were found in incidences of lymphomas between RF EMF exposed and sham exposed groups at any of the exposure levels, thus eliminating any dose rate–response effect.

441. In view of the far more precise dosimetry the Council considers the findings of this experimental study are highly credible.

442. Council considers this paper:

- negative for the biological plausibility of an association between microwave exposure and lymphoma (haematopoietic).

Repacholi MH, et al. 1997,[448] **'**Lymphomas in E-Pim1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields', *Radiat Res*, 147:631–640.

443. Cages holding five mice each were spaced around a vertical monopole centered at a ground plane in a small shielded room. The incident levels ranged from 2.6 to 13 W/m2, and the normalized SARs were from 0.003 to 0.32 W/kg per W/m2.

444. The results were tabulated as incidences of lymphoblastic or nonlymphoblastic lymphoma, renal disease only, or with both lymphoma and renal disease. The increase of lymphoblastic lymphoma was not significant, but higher incidences of all lymphomas and nonlymphoblastic lymphoma were significant.

Lai, H & Singh, P 2004, 'Magnetic-field-induced DNA breaks in brain cells of the rat', *Environ Health Perspect*, vol. 112, no. 6, pp. 687-694.

These 3 articles were not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

448 The Council provides this description and analysis of **Repacholi MH, et al. 1997, which** is new information, only in so far as it provides context to the analysis of Utteridge, TD et al. 2002 above.

Council's comments

445. The Council's concerns about this study were regarding the exposures, such as doing them with five mice each in small cages, the general inadequacy of the dosimetry, and particularly that the interactions among the mice in each cage likely caused large specific absorption rate (SAR) variations.

446. Council considered this paper:

- supported biological plausibility of an association between EMF and haematopoietic cancers;
- however, dosimetry was variable and there is no evidence regarding chronic lymphocytic leukaemia specifically.

Krewski, D et al. 2001a, 'Potential health risks of radiofrequency fields from wireless telecommunication devices', *Journal of Toxicology & Environmental Health*, part B, critical reviews, vol. 4, no. 1, pp. 1-143. RMA ID 23906 FILEForce ID 18541

447. This review considered about 470 original studies, reviews and reports - peer review publications and 30 invited submissions in relation to the safety of exposure to radiofrequency (RF) fields according to 3 terms of reference:

a. Do the provision of the Guidelines for safe exposure to RF contained in Health Canada's Safety code 6, protect both RF workers and the general population from the thermal effects associated with exposure to radiofrequency fields?

b. What are the biological and/or potential adverse health effects associated with exposure to RF fields?

c. What are the non thermal biological effects and /or potential adverse health effects associated with exposure to RF fields emitted from wireless telecommunication devices such as wireless phone and base station transmitters?

- Is there evidence that such non thermal effects, If any could be greater for children or other population subgroups?
- What are the implications for safety code 6 of the panel's scientific review of the currently available data on biological effects and the potential adverse health effects of exposure to RF? In particular, should the phenomenon of non thermal effects be considered in safety code 6?
- What research is needed to better understand the potential health consequences for non thermal effects?

448. The large number[449] of studies included studies on tumour genesis, promotion, altered cell proliferation, and DNA damage.

449. With regard to RF field exposure the authors concluded: [450]

> …RF field exposure from wireless communication sources depend upon a number of variables. Environmental RF field exposure from wireless systems base station antennas are very weak. However, local partial body exposures resulting from the use of cellular phones themselves are stronger and at times approach the recommended limits of Safety Code 6. Furthermore, occupational exposure of workers who must work near the base station antennas may be strong enough to require control measures to limit exposure, particularly when many antennas are collocated on a particular site.

450. With regard to effects on in vitro systems the authors concluded: [451]

> In general, the effects on in vitro systems – whether they are intact cells in culture, subcellular components, or tissue cultures – are difficult to relate to potential adverse health effects on intact organisms. In addition, many of the in vitro experiments have been conducted with high specific absorption rates (SAR), such that some of the changes reported would not occur with exposure less than those allowed by SC6.

451. With regard to radiofrequency effects on Ca2+ the authors concluded: [452]

> …this body of data (reviewed) suggest that ELF-modulated RF radiation may effect Ca2+ efflux from brain tissue.

452. With regard to ornithine decarboxylase (ODC) and polyamines following exposure to electromagnetic fields (EMF) and potential relationship to cancer the authors:

a. Discussed evidence for cancers other than those of the haematopoietic system;

b. Discussed evidence for the enhancement of several known oncogenes including c-myc, Ki-ra and Ha-ras;

c. Discussed evidence for transient increases in ODC activity in both cultured mammalian cells and animals exposed to amplitude modulation RF or MW fields and 50 – 60 Hz electric / magnetic fields. [453]

d. Concluded: [454]

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449 See Table 7 at pp. 61-71.

450 Page 25.

451 Page 26.

452 Page 31.

453 Table 4 page 39.

454 Page 43.

> …it is possible that this small change in ODC activity brought about by ELF is unrelated to human cancer risk. However, both the apparent persuasiveness of the data and the potential for interaction suggest that further research in this area is needed.

453. The authors provided a summary of the evidence for genotoxic effects of radio frequency fields including the following reference to studies related to the haematopoietic system. [455]

a. Prausnitz, S and Susskind, C 1962 [456]. Initiation study in mice exposed to 92.7 GHz, increased incidence of leucosis and leukaemia;

b. Repacholi, MH et al. 1997 [457]. Pin1 mice (transgenic animals prone to leukemia) 0.90 GHz for 1 hour / day, 19 months (2.6 – 13 W/cm2) similar to that used in mobile telecommunications, twofold increase in the risk of developing lymphoma;

c. Meltz, ML et al. 1989, 1990 [458] used a mouse lymphoma assay with 2450 MHz; 48.8 W/m2 and 30W/kg exposure showing no effect on mutation frequency;

d. Maes, A et al. 1995 [459] performed in vivo studies on human lymphocytes with 954 MHz exposure resulting in increased chromosomal aberrations. Additionally studied human lymphocytes of antenna maintenance workers exposed to various frequencies for at least a year for at least 1 hour per day and found no chromosomal aberrations;

e. Khalil, AM et al. 1993 [460] performed in vivo studies on human lymphocytes with 167 MHz exposure resulting in increased chromosomal aberrations;

f. Huang, AT et al. 1997 [461] performed in vivo studies on Chinese hamster blood lymphocytes with 2450 Mhz CW, up to 21 W/Kg 15 min / d for 5 consecutive days and found no evidence of chromosomal aberrations;

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455 Table 7 at pages 61 – 71.

These following 14 articles were not available to (before) the RMA at the relevant times, however the information above is cited in Table 7 by **Krewski, D et al. 2001a** and as such was considered by the Council.

456 Prauznitz, S and Susskind, C 1962, 'Effects of chronic microwave irradiation of mice', *IRE Trans. Biomed. Electron* 9: 104-108.

457 Repacholi, MH et al. 1997, 'Lymphomas in Eμ-Pim1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields', *Radiat. Res,* 147: 631 – 640.

458 Meltz, ML et al. 1989, 'Absence of mutagenic interaction between microwaves and mitomycin C in mammalian cells', *Environ. Mol. Mutagen* 13: 294-303 ; Meltz et al 1990 Proflavin and microwave radiation: Absence of a mutagenic interaction *Bioelectromgagnetics* 1: 149-157.

459 Maes, A et al. 1995, 'Cytogentic effects of microwaves from mobile communication frequencies (954 MHz)', *Electromagnetobiology,* 14: 91-98.

460 Khalil, AM et al. 1993, 'A preliminary study on the radiofrequency field-induced cytogentic effects in cultured human lymphocytes', *Dirasat* 20: 121 – 130.

461 Huang, AT et al 1977, 'The effect of microwave radiation (2450 MHz) on the morphology and chromosomes of lymphocytes', *Radio. Sci* 12: 173 – 177.

g. Garson, OM et al. 1991 [462] examined human lymphocytes of radiolinemen with occupational exposure of 0.4 to 20,000Mhz and found no chromosomal aberrations;

h. Lloyd, DC et al. 1984 and 1986 [463] examined human lymphocytes from subjects exposed to 2450 MHz CW, up to 200 W/kg for 20 min and found no chromosomal aberrations;

i. Garaj-Vrhovac, V et al. 1992 [464] examined human lymphocytes from subjects exposed to 7700 Mhz 0.5 (30min), 30mW/cm2 (10, 30 or 60 mins) and showed increased chromosomal aberrations;

j. Maes, A et al. 1993 [465] examined human lymphocytes from subjects exposed to 2450 MHz for 30 and 120 min and showed increased chromosomal aberrations and increased micro-nucleus formation, but no effect on sister chromatid exchange (SCE);

k. Several studies of RF exposure showed no effect on SCE in human lymphocytes (Wolff, S et al. 1985 [466]) and bone marrow (McRee, DI et al. 1981, McRee, DI and MacNicols 1981 [467], Brown, RF and Marshall, SV 1982 [468]). However Khalil, AM et al. 1993 showed an increased in SCE in human lymphocytes exposed to 167 MHz.

454. The authors concluded: [469]

> …overall, a number of different assays for studying genotoxicty have failed to produce consistent positive findings regarding RF fields.

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462 Maes, A et al. 1995, See fn 90.

463 Lloyd, DC et al. 1984, 'No clastogenic effect from in vitro microwave irradiation of G_0 human lympohcytes *Int J Radiat Biol* 46: 135-141;

Lloyd, DD et al. 1986, 'Absence of chromosomal damage in human lympohcytes exposed to microwave radiation with hyperthermia', *Bioelectromagnetics* 7: 235-237.

464 Garaj-Vrhovac, V et al. 1992, 'The correlation between the frequency of micronuclei and specific chromosome aberrations in human lymphocytes exposed to microwave radiation in vitro', *Mutat Res* 281: 181-186.

465 Maes, A et al. 1993, In vitro cytogenetic effects of 2450 MHz waves on human peripheral blood lymphocytes. *Bioelectromagnetics* 14: 495 – 501.

466 Wolff, S et al. 1985, 'Magnetic resonance imaging: Absence of invitro cytogenic damage', J Nuclear Radiol, vol. 155, pp. 163-165.

467 McRee, DI et al. 1981, 'Incidence of sister chromatid exchange in bone marrow cells of the mouse following microwave exposure', *Radiat Res*, vol. 85, pp. 340-348.

468 Brown, RF and Marshall, SV 1982, 'Sister chromatid exchange in marrow cells of mice exposed to RFR (400, 800 and 1200 MHzCW) (metting abstract). In 4th Biolectomagnetics Society annual meeting – abstracts, Lost Angeles, June 28 – July 2 page 51 abstr no G31.

469 Page 74.

455. The authors provided a summary of the epidemiological studies of RF field exposure and health effects. Those including the adult haematopoietic system are listed here: [470]

a. Dolk, H et al. 1997a; [471] - see from [262]

   – chronic lymphocytic leukaemia OR = 2.56 (1.11 – 5.05) for 0.2 KM and OR = 1.32 (1.08 – 1.62) for 0-10 km

b. Hocking, B et al 1996; [472] - see from [285]

– Leukaemia incidence for adults	RR = 1.24	1.09 – 1.40

c. Milham, S 1985; [473]

d. Milham, S 1988; [474] - not analysed in these Reasons

– Lymphatic and hematopoietic system	SMR = 1.23	0.99 - 1.52
– Leukaemia	SMR = 1.24	0.87 - 1.72
– Chronic Lymphatic Leukaemia	SMR = 1.09	0.40 - 2.38

e. Muhm, JM 1992; [475] - not analysed in these Reasons

– Hematopoietic system	SMR = 3.31	0.40 - 11.96
– lymphoma	SMR = 10.87	0.28 - 60.56
– Leukaemia	SMR = 4.37	0.11 - 24.33

f. Robinette, CD et al. 1980; [476] - not analysed in these Reasons

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[470] Table 8 page 75 – 81.

[471] Dolk H, et al. 1997, 'Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield Transmitter', *American Journal of Epidemiology*, 145(1) pp 1-9. RMA ID 9620 FILEForce ID 18858

[472] Hocking, B et al. 1996, 'Cancer incidence and mortality and proximity to TV towers', *Medical Journal of Australia*, vol. 165, no. 2, pp 601-605**.** RMA ID 21127 FILEForce ID 18995 See [285]

[473] Milham 1985, 'Mortality in workers exposed to electromagnetic fields', *Environ Health Perspect*, vol. 62, pp. 297-300. - mortality data of amateur radio operators no cases of lympoblastic leukaemia.

This article was not available to (before) the RMA at the relevant times, however the information above is cited in Table 8 by **Krewski, D et al. 2001a** and as such was considered by the Council.

[474] Milham S 1988, 'Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies', *Am J Epidemiol,* vol. 127, no. 127, no. 1, pp. 50-54. - 67,829 amateur radio operators between 1979 – 1984, no exposure details, other than by occupation provided.

[475] Muhm J 1992, Mortality investigation of workers in an electromagnetic pulse test program. *J Occup Med* 34: 287-292.

- 304 male workers in an electromagnetic pulse test program from 1970-1986. Exposure to nuclear exposition related electromagnetic pulses with frequency range 10 kHz – 100 MHz with potential exposure of 30 days to 6 months.

– Lymphatic and hematopoietic system

SMR = 1.18 no CI provided

g. Szmigielski, S 1996; [477] - See from [248]

– chronic lymphocytic leukaemia incidence

5.04 per 100,000 OR = 3.68 1.45 – 5.18

456. The authors concluded: [478]

> The epidemiological studies published to date are of fair overall quality, with exposure assessment being the greatest limitation to interpretation. No high excess risks were observed in any studies of adequate design, and risk estimates were generally less than that at the limit of what is detectable in most epidemiological investigations.

Council's comments

457. The Council noted the authors' review of laboratory evidence. They reviewed experimental evidence of genotoxicity in biological organisms exposed to RF and list many negative results, (In Table 7 about 71 study results show no chromosomal damage effect and about 40 study results show chromosomal damage effect). For example one report of increased chromosomal aberrations in rat kangaroo marrow cells exposed to RF of 2450 MHz 0.2, 1.0 or 5.0 W/cm2 for up to 30 minutes [479]. Also, quoted were effects seen in BALB mice, mice sperm cells, Chinese hamster cells, and human lymphocytes [480]

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476 Robinette et al 1980 Effects upon health of occupational exposure to microwave radiation (radar). *American Journal of Epidemiology* 112 (1): 39-53.

477 Szmigielski, S 1996, Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. *Science of The Total Environment* Volume 180, Issue 1, 2 February 1996, Pages 9-17.RMA ID 10413 FILEForce ID 18848

478 Page 98.

479 See Table 7 at page 63 and Yao, KTS & Jiles, MM 1970, *Effects of 2450 MHz microwave radiation on cultivated rat kangaroo cells*, pp. 123-133. In Cleary, SF ed. 1969, *Biological effects and health implications of microwave radiation*, Medical College of Virginia Symposium – Proceedings, Department of Health, Education, and Welfare, Washington DC.

This article was not available to (before) the RMA at the relevant times, however the information above is cited in Table 7 by **Krewski, D et al. 2001a** and as such was considered by the Council.

480 See Table 7 at pages 65-67 and Manikowska et al. 1979; Manikowska-Czerska et al. 1985; Lloyd et al. 1984, 1986; Kerbacher et al. 1990; Alam et al. 1978; Meltz et al. 1990b; Wolff et al. 1985; Garaj-Vrhovac et al. 1990a, 1990b, 1991, and 1992; Maes et al. 1993, 1997; Yoa 1976, 1982; Chen et al. 1974; Yao 1978; Antipenko and Koveshnikova 1987; Balade 1996, Vijayalaxmi-Frei et al. 1997; Haider et al. 1994; Ciaravino et al. 1987, 1991; Khalil et al. 1993, McRee et al. 1981; McRee and MacNicols 1981.

These articles were not available to (before) the RMA at the relevant times, however the information above is cited in Table 7 by **Krewski, D et al. 2001a** and as such was considered by the Council.

458. Overall the authors concluded that sub-thermal exposures are not known to produce adverse health effects despite much discussion about cell-proliferation cell membrane and other effects, as well as toxicology including the variable effect of DNA damage for which further research is needed.

459. The Council also noted the discussion of tumour induction in animals, for which most studies have been negative, and effects on tumour progression, again most studies showing there are no increases in tumour progression from radiofrequency exposure. Some studies quoted include Prausnitz, S and Suskin, C 1962 [481] who showed increased leukaemia genesis; and Repacholi, M 1997 [482] demonstrating increased lymphomas.

460. The Council noted that the Prausnitz, S and Suskin, C 1962 study was the first to suggest tumour genesis and has been criticized, and the Repacholi, M 1997 study showing increased lymphomas in mice has not been replicated.

461. The Council provides conclusions at the end of Heynick, LN et al. 2003, below.

Heynick, LN et al. 2003, 'Radio frequency electromagnetic fields: cancer, mutagenesis, and genotoxicity', *Bioelectromagnetics Supplement*, vol. 6, pp. S74-S100. RMA ID 38753 FILEForce ID 19089

462. This critical review considered the epidemiological studies (not reviewed in these Reasons) and experimental investigations (25 different, peer-reviewed and published research studies involving various in vivo murine cancer models) on cancer and related effects from exposure to non ionizing electromagnetic fields in the nominal frequency range of 3kHz to 300 GHz.

463. The authors discussed the following studies with regard to evidence for occupation or environmental exposures to RFEMF and an association with cancer. Those listing results from haematopoietic system are included here.

a. Milham, S 1985 [483]

b. Milham, S 1988 [484] not analysed in these Reasons

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[481] Prausnitz, SH & Susskind, C 1962, 'Effects of chronic microwave irradiation on mice' *IRE Trans Biomed Electron*, vol. 9, pp. 104-108.

This article was not available to (before) the RMA at the relevant times, however the information above is cited in Table 7 by **Krewski, D et al. 2001a** and as such was considered by the Council.

[482] Repacholi, MH et al. 1997, 'Lymphomas in Eμ-Pim1 transgenic mice exposed to pulsed 900MHz electromagnetic fields', *Radiat Res*, vol. 147, pp. 631-640.

This article was not available to (before) the RMA at the relevant times, however the information above is cited in Table 7 by **Krewski, D et al. 2001a** and as such was considered by the Council.

[483] Milham, S 1985 Silent Keys: Leukaemia mortality in amateur radio operators Lancet 6: 812. - mortality data of amateur radio operators no cases of lympoblastic leukaemia.

This article was not available to (before) the RMA at the relevant times, however the information above is cited in **Heynick, LN, et al. 2003** and as such was considered by the Council.

- Lymphatic and hematopoietic system SMR = 1.23
- Leukaemia SMR = 1.24

c. Pearce, NE et al. 1989 [485]

d. Garland, FC et al. 1988 [486]

e. Tynes, T 1992 [487]

f. Hocking, B et al. 1996 [488] - see from [285]

- Leukaemia incidence for adults RR = 1.24 1.09 – 1.40

g. Szmigielski, S 1996 [489] - See from [248]

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[484] Milham S 1988 Mortality by license class in amateur radio operators. *Am J Epidemiol* 128: 1175-1176. - 67,829 amateur radio operators between 1979 – 1984, no exposure details provided.

Lymphatic and hematopoietic system	SMR = 1.23	95% CI 0.99 - 1.52
Leukaemia	SMR = 1.24	95% CI 0.87 - 1.72

This article was not available to (before) the RMA at the relevant times, however the information above is cited in **Heynick, LN, et al. 2003** and as such was considered by the Council. Further, Milham S Jr (1988). [Comment] Mortality by license class in amateur radio operators. American Journal of Epidemiology, l 127: 1175-1176 was available information and confirms the SMR results data above, but without the confidence intervals.

[485] Pearce, N et al. 1989, 'Case-control studies of cancer in New Zealand electrical workers', *Int J Epidemiolo* 18: 55-59.

- 19 904 cases during 1982- 1984; only significant excess -= CML – three main findings emerged. First, there is an elevated leukaemia risk in New Zealand electrical workers (odds ratio (OR) = 1.62, 95% confidence interval (CI) 1.04–2.52), but little evidence of increased risks for other cancer sites. Second, contrary to other published studies, the increased risk was primarily for chronic leukaemia (OR = 2.12) rather than acute leukaemia (OR = 1.25), and for lymphatic leukaemia (OR = 1.73) rather than myeloid leukaemia (OR = 1.22). Third, the increased risk was strongest for certain categories of electrical work including radio and television repairers (OR = 7.86, 95% CI 2.20–28.09), electricians (OR = 1.68, 95% CI = 0.75– 3.79), linemen (OR = 2.35, 95% CI 0.97–5.70) and power station operators (OR = 3.89, 95% CI 1.00–15.22).

[486] Garland FC et al. 1988, non-Hodgkin's lymphoma in U.S. Navy personnel. Arch Environ Health 43: 425 – 429 - active duty naval personnel during 1974 – 1979 – no excess of non-Hodgkin's lymphoma

Council notes a study in the same series by: Garland, FC et al. 1990, 'Incidence of leukemia in occupations with potential electromagnetic field exposure in United States Navy personnel', *Am J of Epidemiol*, vol. 132, no. 2, pp. 293-303. - active duty naval personnel during 1974 – 1979 – incident rate for leukemia of 6.0 (Navy) versus 6.5 (US population) per 100,000 – a non-significant statistical difference.

[487] Tynes, T et al. 1992, Incidence of cancer in Norwegian workers potentially exposed to electromagnetic fields. *Am J Epidemiol* 136: 81-88.

- 37, 945 male Norwegian electrical workers exposed to extremely low frequencies and radio frequencies between 1961 – 1985. No exposure details provided

non-Hodgkin's lymphoma	SIR = 0.77	95% CI 0.60 - 0.98
Leukaemia	SIR = 1.08	95% CI 0.89 - 1.31

The above 3 articles by Pearce, NE et al. 1989; Garland, FC et al. 1988; and Tynes, T et al. 1992 were not available to (before) the RMA at the relevant times, however the information above is cited in **Heynick, LN, et al. 2003** and as such was considered by the Council.

[488] Hocking, B et al. 1996, 'Cancer incidence and mortality and proximity to TV towers', *Medical Journal of Australia*, vol. 165, no. 2, pp 601-605**.** RMA ID 21127 FILEForce ID 18995

– chronic lymphocytic leukaemia incidence

5.04 per 100,000 OR = 3.68 1.45 – 5.18

h. Dolk, D et al. 1997a [490] - see from [262]

– chronic lymphocytic leukaemia OR = 2.56 (1.11 – 5.05) for 0.2 km and OR = 1.32 (1.08 – 1.62) for 0-10 km

i. Cooper, D et al. 2001 [491] - see from [274]

– chronic lymphocytic leukaemia OR = 0.98 (0.12 – 3.53) for 0.2 km and OR = 1.46 (1.08 – 1.84) for 0-10 km.

464. The authors discussed the following studies with regard to evidence for mobile phones and cancer. The majority of studies discussed the evidence for an association between mobile phones and cancer of the brain. One study discussing cancer of the haematopoietic system was discussed.

a. Morgan, RW et al. 2000 [492] - (See Non- Contributory list)

– Lymphatic/hematopoietic cancers SMR and CI were below 1.0

465. The authors concluded: [493]

> Collectively, none of the epidemiologic papers discussed in occupational or environmental exposures and cancer provide credible, statistically significant findings that chronic exposure to RFEMF emitted by various sources can initiate or promote any form of cancer in humans.

466. The authors discussed evidence for cancer association with animal studies exposed invivo. Those including evidence of cancer of the haematopoietic system are included here.

a. Skidmore, WD and Baum, SJ 1974. [494] Exposed Sprague Dowley rats and Leukaemia prone mice to continuous electromagnetic pules (EMP). Leukaemia in

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489 Szmigielski, S 1996, Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. *Science of The Total Environment,* vol. 180, Issue 1, 2 February, pp. 9-17**.** RMA ID 10413 FILEForce ID 18848

490 Dolk, H et al. 1997, Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield Transmitter*. American Journal of Epidemiology*, 145(1) pp 1-9. RMA ID 9620 FILEForce ID 18858

491 Cooper, D et al. 2001, [Comment] 'Re Cancer incidence near radio and television transmitters in Great Britain. 1. Sutton Coldfield transmitter; 11. All high power transmitters.', *American Journal of Epidemiology*, 153(2): 202-204. Erratum: *Am J Epidemiol*, (2007) 166:1107. RMA ID 21127 FILEForce ID 18995

492 Morgan, RW et al. 2000, 'Radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems', Epidemiology, vol. 11, no. 2, pp.118-127.

493 Page S82

494 Skidmore, WD and Baum, SJ 1974, 'Biological effects in rodents exposed to 108 pulses of electromagnetic radiation', *Health Phys,* vol. 26, pp. 391-398.

the AKR/J mice did not occur earlier in exposed mice nor was there percentage higher than in sham exposed mice.

b. Repacholi, MH et al. 1997. [495] Exposure of Eμ-Pim1 transgenic – increased in lymphoma incidence.

c. Utteridge, TD et al 2002. [496] Exposed 120 Eμ-Pim1 heterozygous mice and 120 wild type mice for 1h our/day 5 days per week for up to 104 weeks. No differences were found contrary to the results of Repacholi, MH et al. 1997.

d. Vijayalaxmi, FMR et al. 1997. [497] Genotoxicity in peripheral blood cells of mice exposed to EMF.

467. The authors discussed evidence for cancer association with experiments involving mammalian live tissues and cells exposed in vitro. Those including evidence of cancer of the haematopoietic system are included here.

a. Stodolnik-Baranska, W and Ostrowski, K 1974. [498] Investigated whether exposure of human lymphocyte cultures stimulated with the mitogen phytohemagglutinin (PHA) to pulsed RFEMF would produce chromosomal aberrations. A rise in chromosomal breaks was reported without statistical analysis.

b. Chen, K-M et al. 1974, [499] Investigated whether exposure of Chinese hamster cells and human amnion cells to 2.45 GHz CW RFEMF would produce chromosomal aberrations. No significant differences were found.

c. Garaj-Vrhovac, V et al. 1992. [500] Chromosomal aberrations in lymphocytes from samples of exposed human whole blood.

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495 Repacholi et al. 1997, 'Lymphomas in Eμ -PIM1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields. *Radiat. Res* 147, 631-640.

The Skidmore, WD and Baum, SJ 1974; and Repacholi et al. 1997, articles were not available to (before) the RMA at the relevant times, however the information above is cited in **Heynick, LN, et al. 2003** and as such was considered by the Council.

496 Utteridge, TD et al 2002, 'Long term exposure of Eμ-Pim1 transgenic mice to 898.4 MHz microwaves does not increase lymphoma incidence', *Radiat Res*, vol. 158, pp. 357-364. RMA ID 26556, FIILEForce 18482

497 Vijayalaxmi, FMR et al. 1997, 'Frequency of micronuclei in the peripheral blood and bone marrow of cancer prone mice chronically exposed to 2450 MHz radiofrequency radiation', *Radiation Research*, 147: 495 – 500.

498 Stodolnik-Baranska, W and Ostrowski, K 1974, *The effects of microwaves on human lymphocyte cultures,* In Czerksi, P et al. eds. *Biologic effects and health hazards of microwave radiation*, Warsaw: Polish Medical Publishers, pp. 189-195.

499 Chen, K-M et al 1974, 'Chromosomal aberrations of linivn cells induced by microwave radiation', *Envrion Lett*, vol. 6, pp. 37-46.

500 Garaj-Vrhovac, V et al. 1992, 'The correlation between the frequency of micronuclei and specific chromosome aberrations in human lymphocytes exposed to microwave radiation in vitro', *Mutat Res* 281: 181-186.

d. Meltz, ML et al. 1989 and 1990. [501] Results from exposure of mammalian cells and mouse leukemic cell lines.

e. Maes, A et al. 1995. [502] Exposure of human whole blood.

f. Vijayalaxmi, LBZ et al. 2000. [503] Exposure of cultured human blood lymphocytes from exposed subjects.

g. MacNamee, JP et al. 2002, [504] Exposure of human leukocytes impact on DNA. No evidence of DNA damage was found.

468. The authors found, in summary that of the 25 studies reviewed three were inconclusive, three were positive, and the other 19 were negative for biological plausibility.

469. The authors concluded: [505]

> Overall, pending any positive findings of new studies under way or planned, the findings of this review indicate that there is no reproducible scientifically valid experimental basis for the claims about a linkage between EMF exposures and the initiation, promotion or co promotion of cancer.

Council's comments for Krewski, D et al. 2001a and Heynick, LN et al. 2003

470. The Council considered that in respect of biological plausibility for non-ionising radiation and carcinogenicity there was:

– more evidence of a negative association than a positive one.

471. Overall, Council is of the view that:

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501 Meltz, ML et al. 1989, 'Absence of mutagenic interaction between microwaves and mitomycin C in mammalian cells', *Environ. Mol. Mutagen* 13: 294-303.

Meltz, ML et al. 1990, 'Proflavin and microwave radiation: Absence of a mutagenic interaction', *Bioelectromgagnetics* 1: 149-157.

502 Maes et al 1995 cytogentic effects of microwaves from mobile communication frequencies (954 MHz). *Electromagnetobiology* 14: 91-98.

503 Vijayalaxmi, LBZ et al. 2000, 'Primary DNA damage in human blood lymphocytes exposed in vitro to 2450 MHz radio-frequency radiation', *Radiat Res*, 153: 479-486.

504 MacNamee, JP et al. 2002, 'DNA damage and micronucleus induction in human leukocytes after acute in vitro exposure to a 1.9 GHz continuous-wave radiofrequency field', *Radiat Res,* vol. 158, pp. 523-533.

The above 9 articles: Vijayalaxmi, FMR et al. 1997 at fn 126 to MacNamee, JP et al. 2002 at fn 133 were not available to (before) the RMA at the relevant times, however the information above is cited in **Heynick, LN, et al. 2003** and as such was considered by the Council.

505 Page S96.

– the positive laboratory data, although limited and less than negative results, does support a reasonable hypothesis that Non-ionising radiation in the form EMF, RF or microwaves can be carcinogenic.

472. Such evidence specific to chronic lymphocytic leukaemia is however lacking and the Council therefore concludes with respect to the biological plausibility of an association between chronic lymphocytic leukaemia and non-ionising radiation, that the evidence:

– does not point to but merely left open the possibility of a relevant association.

THE COUNCIL'S CONCLUSIONS

473. The Council noted the Applicant (MrFR) submitted that he had been exposed to electro-magnetic radiation and other agents over a period of 12 years of military service. He submitted that his general exposure to EMR totalled an estimated 6,590 hours and that this included two significant 200 kilowatt active radar exposure events.

474. In the Council's view the sound medical-scientific evidence was in relation to exposures that were not of the magnitude contended by the Applicant.

475. The Council also noted that a person making a submission to the Council (Mrs F) contended that exposure to the earliest aircraft radar, possibly like the Air to Surface (ASV) radar adopted in 1938-39 in Britain, with wavelengths to 1.5 metres and other exposures as a Military Air Craft Controller were related to chronic lymphocytic leukaemia.

476. The Council noted that the Applicant, a person making a submission to the Council and the Commissions each made submissions concerning the data in some of the following articles that the Council also considered important:

Szmigielski, S 1996;

Robinette, CD et al. 1980 and the updated data in Groves, FD et al. 2002;

Floderus, B et al. 1993;

Dolk, H et al. 1997 a & b and the updated data in Cooper, D et al. 2001;

Morgan, RW et al 2000;

Krewski, D 2001a;

477. The Commissions submitted that Floderus, B et al. 1993 was the strongest available evidence, with the 'highest statistically significant level of risk' in relation to EMFs and chronic lymphocytic leukaemia, with other epidemiological studies being 'suggestive or weakly supportive of an association, concluding that the data may 'go either way' in respect to meeting the reasonable hypothesis test.

478. The Commissions concluded that the evidence in relation to RF/MW exposure and chronic lymphocytic leukaemia provides no consistent support, with the data in Dolk, H et al 1997; Szmigielski, S 1996 while positive to an association does not provide meaningful support for an association (due to proxy exposure – distance from transmission towers and very low (3) case numbers respectively).

479. In the Council's view, the sound medical-scientific evidence did not allow for the conclusions to be drawn for separate sub-entities of non – ionising radiation. In particular the evidence on exposure often did not take into account many sources of radiation and the measurement of exposure that was made was by surrogate measures such as job title or by ecological data (at the population level and not individual level exposures) such as distance from powerlines or TV transmitters was inherently unreliable.

480. The Council found that the each of the studies by:

Floderus, B et al. 1993

Feychting, M et al. 1997

Kheifets, LI et al. 1997

Havas, M 2000

California Department of Health Services (DHS) Anon 2001

pointed to (as opposed to merely left open) a relevant association.

481. The Council also found that the studies by:

Lai, H & Singh, N 1995

Krewski, D et al. 2001a

Heynick, LN et al. 2003

support the biological plausibility of cancers generally, however data specifically for chronic lymphocytic leukaemia (B-cell CLL) is lacking.

482. The Council considered that the studies that, in the Council's view, left open the possibility of the relevant association also provide some support for the studies that pointed to a relevant association as the data in some of those studies shows non statistically significant trends for an elevation of risk for:

– Haematopoietic cancers; or

– Leukaemia and lymphoma; or

– Non-Hodgkin's lymphoma

483. The Council considered that the studies by:

Floderus, B et al. 1999;

Theirault, G et al. 1994;

Groves, FD et al. 2002;

Szmigielski, S 1996;

Dolk, H et al. 1997a; Dolk, H et al. 1997b; and Cooper, D et al. 2001;

Hocking, B et al. 1996;

Kheifets, LI et al. 1999;

Habash, RW et al. 2003;

Preece, AW et al. 2000;

Krewski, D et al. 2001 b;

Moulder, JE et al. 1999;

Breckenkamp, J Berg, G and Blettner, M 2003;

Elwood, JM 1999;

Elwood, JM 2003;

did not point to but merely left open the possibility of the relevant association.

484. The Council considered that the studies by:

Hakansson, N et al. 2002

Harrington, JM et al. 2001

Savitz, DA & Loomis, DP 1995

were against the possibility of the relevant association.

485. The Council considered that the effect of the study by:

Utteridge, TD et al. 2002

was against biological plausibility.

486. The Council also considered other articles that in its view were sound medical-scientific evidence that touched on the potential non-ionising radiation factor, that were in Council's final analysis non contributory[506]:

Feychting, M & Ahlbom, A 1994;

Tynes, T & Haldorsen, T 2003;

Kheifets, LI et al. 1997;

Miller, AB et al. 1996;

d'Amore et al 2001;

Coleman, MP et al. 1989;

Verkasalo, PK 1997;

Morgan, RW et al. 2000, '

Li, C-Y et al. 1997;

Loomis, DP & Savitz, DA 1990;

Pachocki, KA & Gajewski, AK 1991;

Floderus, B et al. 1994;

Alfredsson, L et al. 1996;

Tornquist, S et al. 1991;

Fear, NT et al. 1996;

Feychting, M 1996;

International Commission on Non-Ionizing Radiation Protection 1998;

Cherry N 2000;

[506] Articles that did not, in Council's final analysis, contribute to either advance or weaken the state of evidence of the possibility of a relevant association (ie were non contributory).

487. The Council did not focus in these Reasons on those articles that in its view were non contributory, as those studies:

- did not provide data specific to chronic lymphocytic leukaemia;
- provided data on haematopoietic cancers, leukaemia and lymphoma, and or Non-Hodgkin's lymphoma that the Council could not draw conclusions from concerning chronic lymphocytic leukaemia;
- were in respect of such small number of cases of chronic lymphocytic leukaemia, haematopoietic cancers, leukaemia and or lymphoma that the Council could not draw conclusions from concerning chronic lymphocytic leukaemia.

and therefore the Council has focussed in these Reasons on the relevant studies described and commented upon above.

488. The Council having considered all the information both individually and collectively, and conscious that the reasonable hypothesis test is one of possibility, and an unusually light burden, was convinced that the reasonable hypothesis test had been met.

489. Being satisfied that the reasonable hypothesis test was met, the Council considered the balance of probabilities and was not so satisfied. Neither individually nor collectively does the sound medical-scientific evidence, in the Council's opinion, indicate the relevant association to the balance of probabilities level of satisfaction.

490. The Council therefore recommends that the RMA, in undertaking the investigation recommended by the Council (See at [1] - [4] above) considers whether there is a new body of sound medical-scientific evidence available that, together with the sound medical-scientific evidence already considered by the RMA, including information considered by the RMA concerning Chronic Lymphoid Leukaemia and Non Hodgkin's Lymphoma or any haematopoietic cancers, so far as being of potential relevance to Chronic Lymphocytic Leukaemia / Small Lymphocytic Lymphoma, justifies exposure to non-ionizing radiation as a factor.

Should There Be A Herbicide, and or Pesticide and or Dioxin Factor(s)?

491. The Council has been requested to review the information touching on (relevant to):

a. exposure to herbicides; and / or

b. exposure to pesticides, and / or

c. exposure to dioxin; and / or

d. consuming potable water when the water supply had been produced by evaporative distillation.

492. As a threshold proposition, the Council noted that it was very difficult to ascertain from the studies which touched on herbicide, pesticide and dioxin exposure the precise nature of the exposure, i.e., whether it was to herbicides, pesticides, dioxins, or a combination.

493. The Council considered all the studies in the pool and identified from the pool those studies it considered most methodologically sound and persuasive. The Council's analysis of the articles it considered most important regarding exposure to herbicides and/or pesticides and/or dioxins is summarised below.

Kogevinas, M et al 1997, 'Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins, American', *Journal of Epidemiology*, vol. 145, no. 12, pp. 1061-1075. RMA ID 18850

494. This study describes an International Agency for Research on Cancer (IARC) cohort of 21,863 herbicide workers in 36 cohorts in 12 countries. The subjects were followed from 1939 to 1992, with exposure being reconstructed using job records, company exposure questionnaires, and serum and adipose tissue dioxin levels[507].

495. The cohort included workers employed in production and in the spraying of herbicides and pesticides. The IARC cohort includes cohorts reported on separately by other authors and commented upon in these Reasons[508].

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507 At Abstract, p. 1061

508 Bertazzi, PA Consonni, D Bachetti, S Rubagotti, M Baccarelli, A Zocchetti, C Pesatori, AC 2001, 'Health effects of dioxin exposure: a 20-year mortality study', *American Journal of Epidemiology*, vol. 153, no. 11, pp. 1031-1044.

Steenland, K Piacitelli, L Deddens, J Fingerhut, M and Chang, LI 1999, 'Cancer, heart disease, and diabetes in workers exposed to 2,3,7,8-Tetrachlorodibezo-dioxin', Journal of the National Cancer Institute, vol. 91, no. 9, pp. 779-786.

Ott, MG & Zober, A 1996, 'Cause specific mortality and cancer incidence among employees exposed to 2,3,7,8-TCDD after a 1953 reactor accident', Occupational and Environmental Medicine, vol. 53, pp. 606-612.

Hooiveld, M Heederik, DJ Kogevinas, M Boffetta, P Needham, LL Patterson, DG Jr and Bueno-de-Mesquita, HB 1998, 'Second Follow-up of a Dutch Cohort Occupationally Exposed to Phenoxy Herbicides, Chlorophenols, and Contaminants', American Journal of Epidemiology, vol. 147, no. 9, pp. 891-901.

496. Workers exposed to TCDD or higher chlorinated dioxins [509] did not have an increased rate of death (SMR = 1.00, 95% CI 0.97 – 1.04).. The rate of death from non-Hodgkin's lymphoma (ICD-9 codes 200, 202) in workers exposed to TCDD or higher chlorinated dioxins was [not significantly] elevated (24 deaths; SMR = 1.39, 95% CI 0.89 – 2.06).

497. The authors report findings [not statistically significant] for leukaemia (ICD-9 codes 204-208) against three categories of workers exposed to TCDD or higher chlorinated dioxins (16 deaths; SMR = 0.73, 95% CI 0.42 – 1.19), workers not exposed (17 deaths; SMR = 1.44, 95% CI 0.84 – 2.30) and all workers exposed to any phenoxy herbicide or chlorophenol (34 deaths; SMR = 1.00, 95% CI 0.69 – 1.39).

498. The authors conclude that:

> These findings indicate that exposure to herbicides contaminated with TCDD and higher chlorinated dioxins may be associated with a small increase in overall cancer risk..... [510]

Council's comments

499. The Council considered this was an important study because this is the most comprehensive study of occupational exposure to herbicides and dioxins. However the study is silent on chronic lymphocytic leukaemia and reports leukaemia as a single entity (CLL is included but the number of cases is not known).

500. The Council considered that this study:

- did not point to but merely left open the possibility of a relevant association.

Steenland, K et al. 1999, 'Cancer, heart disease, and diabetes in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin', *J Natl Cancer Inst*, vol. 91, no. 9, pp. 779-786.

501. This study extended the follow up period by 6 years of the analysis of 5,132 chemical workers in 12 US plants [511], which produced TCDD-contaminated chemicals (including Agent Orange) exposing the workers to high levels of TCDD [512].

502. Blood was drawn from 253 workers and a estimated mean serum level of 2000 parts per trillion in lipids of TCDD at the time of last exposure was found. This compared with 6-8 parts per trillion for the general population[513].

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509 See Table 4 at p. 1068.

510 At Abstract p. 1061.

511 Steenland, K et al 1999 subjects are included in the IARC study reported by Kogevinas, M et al 1997. See from [494]

512 At Abstract and p. 779 & 780.

503. A job–exposure matrix was also developed. The matrix assigned each worker a quantitative exposure score for each day worked, based on the concentration of TCDD in processed materials, the fraction of the day worked on those processes, the estimated amount of TCDD contamination reaching exposed skin or the potential for inhaling TCDD-contaminated dust [514].

504. The mortality results for the entire cohort of 5,132 and for 608 men who had chloracne, as noted in the plant medical records are provided [515]. The group of 608 men who had chloracne had an excess of risk of 25% for all cancers and for lymphatic and hematopoietic tissue cancers had a slightly elevated excess risk. There was an significant positive trend for increasing exposure to TCDD for all cancers risk (P = 0.02), but the data for hematopoietic cancers while showing an elevation was based only on 3 observed and 4.8 expected deaths. [516]

505. The results show that:

> Workers with the highest estimated levels of TCDD exposure had a higher rate of all cancers combined, due to a generalised increase rather than an excess at one or two specific sites. At p. 784
>
> Mortality from non-Hodgkin's lymphoma … was unremarkable. At p. 785

Council's comments

506. In the Council's view this is a good study of some of the subjects included in the Kogevinas, M et al 1997 IARC study. There was a small (13%) increase risk of all cancers combined and a dose-response relationship, with convincing evidence of excess risk confined to very high (100-1000 times higher than the general population) exposed workers. There was also a slightly increased risk of lymphatic and hematopoietic cancers in the chloracne subcohort.

507. However the study is silent in respect of chronic lymphocytic Leukaemia. The data is of mortality only and there is not enough data on leukaemia generally and lymphatic

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513 At p. 799.

514 At p. 780.

515 At p. 782, Table 1. **Cohort mortality results: selective causes**

Death category (ICD-9 code)	No. of deaths	SMR (95% CI)
All cancers (140-208)	377	1.13 (1.02 - 1.25)
Non-Hodgkin's Lymphoma (200, 202)	12	1.10 (0.56 – 1.91
Leukaemia and aleukaemia (204-208)	10	0.81 (0.38 – 1.48)
Mortality results for chloracne subcohort (n=608); selective causes		
All cancers (140-208)	73	1.25 (0.98 – 1.57)
Lymphatic and hematopoietic (200-208)	6	1.13 (0.41 – 2.46)

516 See Table 2 at page 783.

and hematopoietic tissue cancers to determine risk for sub entities, including for B-cell CLL.

508. The Council considered that this study:

- did not point to, but merely left open the possibility of a relevant association.

Waterhouse, D et al. 1996, 'Cancer incidence in the rural community of Tecumseh, Michigan. A pattern of increased lymphopoietic neoplasms'*, Cancer*, vol. 77, no.4, pp.763-770. RMA ID 20974 FILEForce ID 18879

509. This prospective study considered site-specific cancer numbers in 7,016 males and females in a rural farming community in Michigan in 1959-1987 to investigate the possible association between environmental exposure and the development of cancer

510. The authors implemented 3 study designs to test their hypothesis.

a. A survey of total and site specific cancer incidence among 7,016 males and females;

b. An analysis for each county in Michigan the comparative annual number of acres and percentage of acreage treated with agricultural chemicals; and

c. A nested case control study.

511. The survey implement involved:

a. Questionnaires were developed to assess the following risk factors: cigarette smoking and alcohol use; history of similar neoplasm among first-degree relatives; occupation exposures to benzene, petrochemicals, or agricultural chemicals; household exposure to pesticide/herbicide sprays; and exposure to radiation.

b. Cancer diagnosis were obtained from individual questionnaires and verified from medical records, physician and treatment summaries, discharge summaries, pathology reports and death certificates where possible. Cancer diagnoses were classified as definite, suspect, unconfirmed and misclassified

c. The observed incidence of cancer was compared to the expected number, based on age, sex, race, calendar period and site specific cancer incidence rates reported by the Connecticut tumour registry to calculate a standardized incidence ratio for each cancer (SIR)

512. From the survey results:

– The SIR for leukemias, lymphomas and myeloma (ICD-9 codes 201 - 208.9) for females was 1.24 (95% CI 0.85-1.75) and for males was 1.39 (95% CI 1.00-1.88) [517]

– A significantly increased SIR for males and females combined for NHL, HL and CLL was 1.40 (95% CI 1.03-1.86; P = 0.03) and for CLL alone SIR was 1.75 (95% CI 0.84 – 3.22) [518]

513. Assessment of environmental exposures to agricultural chemicals and the subsequent impact on cancer rates included:

a. Assessment of the levels of environmental exposures to agricultural chemicals, namely pesticides and herbicides, by geographic coding of the number and proportion of chemical sprayed or treated acreage in 1978, and for the period 1982-1987 for all counties of Michigan;

b. Comparison of cancer incidence trends to Lenawee County and the Wayne-Oakland-Macob tricounty area in Michigan encompassed since 1973 by the National Cancer Institute (NCI) Surveillance, Epidemiology and End Results (SEER) program.

514. From the environmental exposure to agricultural chemicals study, the authors observed:

> Tecumseh (Lenawee county) experienced the highest average annual levels of spraying or treatment with agricultural chemicals [519]
>
> …when compared with the SEER tricounty registry referent population, the SIR for NHL in females living in Tecumseh was 1.92 (95% CI 1.07-3.11; P=0.02). The trend in Tecumseh males for increased risks of lymphomas and leukemias persisted when compared with the SEER Caucasian male population. There were significantly more leukemias (International Classification of Disease: Oncology [ICDO] = M9800-9949) observed in Tecumseh males (SIR, 1.920; 95% CL, 1.07-3.14; P = 0.01) [520]

[517] Page 767 table 2.

[518] Page 767 table 3.

Site	Observed cases	SIR	95% CI
Non-Hodgkin's lymphoma	25	1.14	0.74-1.69
Hodgkin's disease	13	2.00	1.06-3.42
Chronic lymphocytic leukemia	10	1.75	0.84-3.22
Total	48	1.40	1.03-1.86

[519] Page 768 figures 1&2.

[520] Page 767.

515. A Case control study of 42 male and 32 female patients with lymphomas and leukaemias were studied, with four controls matched individually to each patient by sex and year of birth.

516. From results of the case control study the authors observed

> …a family history of lymphoma, leukaemia, or multiple myeloma (p=0.01) was significantly associated in the patients. [521]

517. In the case control study, the odds ratios (males and females combined): [522]

- for having worked on a farm was 1.45 (95% CI 0.45-4.66);
- household regular use of pesticide sprays was 1.33 (95% CI 0.33 – 5.48);
- first degree relative with lymphoma, leukaemia or myeloma was 3.81 (95% CI 1.49-9.79).

518. There was insufficient power to evaluate the significance of an interaction of family history and exposure to pesticides. The previous TCHS database on the use of pesticides did not distinguish herbicides from insecticides and fungicides, or allow for estimation of intensity and duration of exposures whether protective equipment used. [523]

519. The authors reviewed additional studies describing a positive association of NHL or CLL with pesticides supporting their findings.[524] They noted that:

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[521] Page 767

[522] Page 769 table 4

[523] Page 768

[524] Hoar SK et al. Agricultural herbicide use and risk of lymphoma and soft-tissue sarcoma. *JAMA* 1986; 256: 1141-7.

> A population based case-control study NHL = 172 and 3 controls for each patient. Farm herbicide use was found to be associated with NHL OR 1.6 (95% CI 0.9,2.6) – no distinction was made between the NHL pathology.

Zahm, SH et al.1990, A case-control study of non-Hodgkin Lymphoma and the herbicide 2,4-dichloropheoxyacetic acid (2,4-D) in eastern Nebraska. Epidemiology; 1:349-56.

> Telephone interviews were conducted with 201 white men diagnosed with NHL (including CLL) and 725 controls. OR = 1.5 (95% CI 0.9, 2.5) for NHL among men who mixed or applied 2,4-D.

Wigle DT et al 1990, Mortality study of Canadian male farm operators: non-Hodgkin's lymphoma mortality and agricultural practices in Saskatchewan. J Natl Cancer Inst: 82: 575-82

> A cohort study of mortality experience (1971-1985) of almost 70,000 male farmers found a significant dose response relationship between risk of NHL and acres sprayed with herbicides as well as with dollars spent on fuel and oil for farming purposes. Paper in FILEForce but not in CLL series

Corrao, G et al 1989, Cancer risk in a cohort of licensed pesticide users. Scand J Work Environ Health; 15: 203-9

> During the period 1947-1998, the incidence of NHL in the United States has increased about 3% per year among white women and men. Concurrently, the overall incidence of Hodgkin's disease has not changed significantly in white women and men, except for a decreasing trend in the subgroup 65 years and over, in part due to decreasing misclassification of 10-20% of 'Hodgkin's disease' patients who are now being correctly classified as having NHL. [footnotes omitted]

Council's comments

520. The Council considered this was a persuasive study suggesting increased lymphoma, but not B-cell CLL specifically, with pesticide exposure. Also, the study had reasonably rigorous exposure assessment, incidence and mortality endpoints, three overlapping methodologies, and looked for confounders.

521. The Council considered that this study was supportive of a statistically non-significant trend of association between B-cell CLL and questionnaire determined pesticides, therefore the study:

- did not point to but merely left open the possibility of a relevant association.

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. RMA ID 20971 FILEForce ID 18882

522. This population based case control study was conducted in an agricultural area of Italy and included occupational histories and interview data on 164 men and women diagnosed with NHL (based on the ICD-9 codes 200 and 202) and 23 with CLL diagnosed in 1988-90 and 977 controls. The controls were matched for location of residence age and sex.

523. Occupational questionnaires included information on residential history, community life, dietary habits, smoking, alcohol consumption, family history of cancer, medical history, use of drugs, exposure to radiation and work history. Work history included relative time periods for each work experience.

> A cancer risk assessment in a cohort of 25,945 male farmers and licensed pesticide users. SIR for malignant lymphatic tissue was 1.4 (95% CI 1.0-1.9) and haematopoietic tissue 1.1 (95% CI 0.8-1.5)

Cantor et al Pesticides and other agricultural risk factors for non-Hodgkin lymphoma among men in Iowa and Minnesota. Cancer Res 1992; 52: 2447-55.

> Interview data was analysed from 622 white men with newly diagnosed NHL (CLL 13.7%) and 1245 population-based controls in Iowa and Minnesota. Men who ever farmed were at slightly elevated risk of NHL, OR = 1.2 (95% CI 1.0-1.5). Exposure to multiple chemicals posed a problem of associating risk to individual chemicals.

These 5 articles were not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

524. Combined incidence of NHL and CLL were analysed for association with: [525]

- Altitude living OR 0.55 (95% CI 0.35-0.86)
- First degree relative with haemolymphopoietic cancer OR 1.92 (95% CI 0.87 - 4.25)
- A positive medical history for herpes zoster OR 2.09 (95% CI 1.34 – 3.27)

525. The authors noted the following associations of occupations with a diagnosis of CLL: [526]

- Agriculture or animal-breeding or fishing OR 2.31 (95% CI 0.92-5.78)
- Farmers OR 1.62 (95% CI 0.50 – 5.21)
- Farmer breeders OR 3.05 (95% CI 1.12 – 8.32), when farmer-breeders were divided into two groups on the basis of time spent in the industry, the following results were obtained:

< 23 years	OR 1.89	95% CI 1.18 – 3.00
≥ 23 years	OR 1.68	95% CI 1.01 – 2.77

526. The authors assessed the impact of latency for combined NHL/CLL cases according to the following groups:

- Farmer-breeders started and finished working before 1955 had an OR of 3.41 (95% CI 1.73 – 6.70)
- Farmer-breeders who worked before and after 1955 had an OR of 1.72 (95% CI 1.07 - 2.76)
- Farmer-breeders whose work experience began after 1955 had an OR 1.19 (95% CI 0.61 – 2.33)

527. The authors note:

> …the effect of time period could be more related to the way of use of chemicals than to their amount [527]

528. The authors concluded

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525 Page 376 table 2

526 Page 376 table 4 & page 377 table 5.

527 Page 378.

> …the results from this preliminary analysis point to the possible role of chemicals and of their use in the risk of NHL or CLL [528]

Council's comments

529. The Council considered this is a weak study using essentially retrospective memory-based data without good exposure assessment (broad job titles were used as a surrogate for exposure). It works backwards from the diagnosis, rather than prospectively from a period of exposure. Apparent absence of time-dosage effect in their results argues against real effect.

530. There was no dose effect as farmer-breeders who worked for longer than the median work duration of 23 years had a similar OR to those who worked for less than 23 years (OR 1.68 and 1.89, respectively). Farmer-breeders who started worked after 1955 (the year of mass introduction of pesticides) did not have increased risk compared with those who completed work before 1955, even when a latency effect was taken into account. Comparing farmer-breeders and farmers, farmer-breeders had longer working periods (median 23 vs 14 years), and were more likely to personally use chemicals (50% vs 23%).

531. The Council considered this study provides weak supportive evidence for the relevant association.

532. The Council considered this study:

- did not point to but merely left open the possibility of a relevant association.

Hansen, ES 1992, 'A cohort study on cancer incidence among Danish gardeners', *Am J Ind Med*, vol.21, no.5, pp. 651-660. RMA ID 29603 FILEForce ID 19379

533. This historical cohort study considered 4,015 employed Danish gardeners (859 females and 3,156 males who were members of the Danish Union of General Workers) from May 1975 to end 1984. The cohort cancer risk was compared to the expected cancer incidence by sex, age and period specific for the total Danish population with data obtained from the Danish Cancer Registry.

534. The cohort included only person's aged 30 years and older, to account for latency time assuming gardeners would start their trade at a younger age. Data from the age group 80-84 yrs was disregarded. ICD, 7th revision , nos. 200, 202, and 205) and 23 with CLL (ICD, 7th revision, nos. 204.0)

535. Standardized morbidity ratios (SmbR) were obtained for 11 cancer sites (ICD 7th revision).[529]

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[528] Page 378.

For lymphatic and hematopoietic tissue (200 - 205):

Males	SmbR = 144	95% CI 80-237
Females	SmbR = 137	95% CI 17-495
Males and females combined	SmbR = 143	95% CI 83-229

536. Standardized morbidity ratios were then calculated for the 6 diagnosed male CLL (ICD 7th revision no 204.0) cases: [530]

Males	SmbR = 275	95% CI 101-599
Females	SmbR = 0	95% CI 0-1,677
Males and females combined	SmbR = 251	95% CI 92-546

and 8 NHL (ICD 7th revision no 200, 205) cases,

Males	SmbR = 173	95% CI 63-376
Females	SmbR = 364	95% CI 44-1,314
Males and females combined	SmbR = 200	95% CI 86-393

537. The duration of exposure prior to diagnosis was calculated for the 6 gardeners diagnosed with CLL. This ranged from 32 – 44 years with data available for only 3 of the 6 gardeners.[531] For the 8 gardeners diagnosed with NHL, this ranged from 5 to 45 years for the 7 cases for which duration of exposure data was available.

538. The authors did not identify any overt bias in their cohort study.

539. The authors stated their observed increase in observed cases of CLL and NHL is in agreement with several other studies investigating the association between chlorinated pesticides such as phenoxy acetic compounds and DDT with lymphopoietic neoplasms.

540. The authors concluded:

> …our finding of an increased incidence of soft tissue sarcoma and lymphatic neoplasms in the pesticide exposed cohort agreed with our expectations based on the scientific literature relating to this topic. [532]

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529 Page 655 table II.

530 Page 656 table IV.

531 Page 656 table V.

532 Page 657.

Council's comments

541. The Council notes that although only 6 cases of CLL were observed, this is morbidity, not mortality data and so Council considers that it is moderately stronger than would otherwise appear.

542. Council considered this study provides weak supportive evidence for an association for pesticides and B-cell CLL. Exposure was determined by job titles, a surrogate measure and therefore specific exposure data was not available. While most elevations of risk were non-statistically significant the SmbR for males and CLL (275, 95% CI 101 – 599) was elevated and statistically significant, but based on only 6 cases.

543. The Council considered this study:

- did not point to but merely left open the possibility of a relevant association.

Morris-Brown, L et al 1990, 'Pesticide exposures and other agricultural risk factors for leukemia among men in Iowa and Minnesota. *Cancer Research*, vol. 50, no. 20, pp. 6585-6591. RMA ID 29618 FILEForce ID 19513

544. This population based case control interview study between 1981-1984, considered the association between leukaemia and farming in 578 white men with leukaemia and 1245 controls living in Iowa and Minnesota.

545. Cases were identified retrospectively in the first year and prospectively in the two years after the study commenced. Cases were obtained from the Iowa Tumour Registry and in Minnesota, from a specially created surveillance network including hospitals and pathology laboratories.

546. Questionnaires were standardized and detailed information was obtained concerning residential history, drinking water sources, non-farm occupational history, smoking and alcohol use, use of unpasteurized dairy products, medical conditions, family history of cancer and farm activities.

547. Information regarding the use of 24 animal insecticides, 34 crop insecticides, 38 herbicides and 16 fungicides used on farms was obtained. Positive interview results for pesticide use underwent a supplemental interview which included information concerning the number of days per year that each pesticide was personally handled. This included a total of 90 cases and 203 controls.

548. The most common leukemia cell type was CLL (n= 244, 42.2%). [533]

[533] Page 6585.

549. The following results for CLL associations were obtained. [534]

never farmed	OR = 1.0	95% CI N/A
ever farmed	OR = 1.4	95% CI 1.1 - 1.9
farmed (yr)		
1-9	OR = 1.6	95% CI 1.0-2.4
10-29	OR = 1.2	95% CI 0.8-1.9
30-44	OR = 2.0	95% CI 1.3 - 3.0
45+	OR = 1.1	95% CI 0.7 - 1.8
used any fungicide	OR = 1.4	95% CI 0.7 - 2.7
used any herbicide	OR = 1.4	95% CI 1.0 - 2.0
used any insecticide	OR = 1.3	95% CI 1.0 - 1.8
animal farming	OR = 1.3	95% CI 1.0 – 1.8
crop farming	OR = 1.2	95% CI 0.8 - 1.8
animal and crop farming	OR = 1.2	95% CI 0.8 - 1.8

550. The following results for CLL associations with specific chemical use were obtained: [535]

carbamates used on crops 21 cases	OR 2.0	95% CI 1.1 - 3.5;
carbamates used on animals 5 cases	OR 3.1	95% CI 1.0 - 9.3;
total organophospates insecticides used on animals 25 cases	OR 1.7	95% CI 1.0 - 2.8;
including:		
halogenated aliphatics	OR 2.9	95% CI 1.5 - 5.6;

534 Page 6586 table 1.

535 Page 6587 text and page 6588 text – note tables 2,3 4,5 &6 report only combined leukaemia risk data.

16 cases		
halogenated aromatics 9 cases	OR 2.7	95% CI 1.2 - 6.3;
non halogenated aromatics 10 cases	OR 2.5	95% CI 1.0 - 6.4;
captan fungicide 5 cases	OR 2.5	95% CI 0.8 - 7.2;
ever handled 2,4-D 45 cases	OR 1.3	95% CI 0.8 - 2.0;
ever handled DDT on crops 16 cases	OR 1.36	95% CI 0.9 - 3.0;
ever handled insecticide dichlorvos 11 cases	OR 2.2	95% CI 1.0 - 4.6;
ever handled insecticide nicotine 15 cases	OR 1.8	95% CI 1.0 - 3.5;
farmers who had handled nicotine at least 20 years prior to interview: 15 cases, 36 controls	OR 2.4	95% CI 1.2 - 4.6;
use of DDT on animals 36 cases	OR 1.5	95% CI 0.9 - 2.3;

551. Overall, the authors noted:

> … no significant associations with leukemia for exposure to specific fungicides, herbicides (including 2,4-D and 2,4,5-T), or crop insecticides. However, significantly elevated risks for leukemia of ≥ 2.0 were seen for exposure to specific animal insecticides…, and the natural product pyrethrins (OR 3.7) and the chlorinated hydrocarbon methoxychlor (OR 2.2).[abstract]

552. The authors acknowledged the results need to be considered in the context of bias associated with case-control methods and the inaccuracies associated with interview data.

553. The authors considered the strengths of their study:

- Relatively large population based study;
- Pathology slides were reviewed to verify diagnosis; and
- In person interviews were conducted.

Council's comments

554. The Council noted that the Commissions regard this as the best study because it is the only one that has attempted to ascertain individual exposure to specific agents and only one with any useful dose-response data. Consequently [they say] a very large number of associations were examined with many statistical associations being found.

555. In the Council's view the population size not very large. Nevertheless this is an important study. In support of an association is the finding for ever-farmed (OR = 1.4 95% CI 1.12 – 1.9) and the elevated risks for 'used any herbicide', 'used any insecticide', and for 'animal farming', providing moderate support for an association between pesticide use and B-cell CLL specifically. No clear patterns of dose-response were shown although statistically significant results were shown for carbonates on crops and on animals, organophosphates insecticides used on animals including halogenated aliphatics, halogenated aromatics, non-halogenated aromatics, and possibly dichlorvos and nicotine. The Council noted that strong evidence was not shown for 2,4-D.

556. The Council considered this study:

– pointed to a relevant association .

Blair, AD & White, A 1985, 'Leukemia cell types and agricultural practices in Nebraska', *Arch environ* Health, vol. 40 no.4, pp.211-214. RMA 29620 FILEForce ID 19498

557. This retrospective case control survey considered the association of leukaemia with agricultural practices in Nebraska. Death certificates for 1,084 leukaemia deaths from 1957-1974 were compared to 2,168 controls (2 non-leukaemia deaths per case) matched for sex, race, county of usual residence, age at death (+/- 2 yrs) and calendar year of death. Usual occupation as recorded on the death certificate was used to assess occupational association.

> Since the ICDA 7th Revision combines acute lymphatic and acute myeloid leukemias, causes of death among the cases from the original Nebraska study were recoded according to ICDA 8th Revision rules to further evaluate the relationship between farm practices and leukemia cell types. (p. 211)

558. The authors found the following odds ratios for CLL: (Note CI were not supplied)[536]

all cases (n = 248) OR = 1.67
CI not supplied however note provided CI did not include 1.00

age at death ≤ 65 (n = 54) OR = 1.73

[536] Page 213 table 1.

age at death ≥ 66 (n = 194) OR = 1.65
CI not supplied however note provided CI did not include 1.00

year of birth ≤ 1889 (n = 101) OR = 1.72

year of birth 1890-1900 (n = 81) OR = 2.05
CI not supplied however note provided CI did not include 1.00

year of birth ≥ 1901 (n = 66) OR = 1.17

559. The authors stratified agricultural practices according to location as follows:

- Eastern third of state: farming: corn, hog and chicken production including heavy use of insecticides, herbicides and fertilizers;
- Southern region: wheat producing; and
- Northern distribution: cattle and dairy production.

560. The authors found the following Odds ratios for CLL in the three stratified locations. Each agricultural region was further sub classified into high and low representing a greater and lesser concentration of the farming practices and exposures respectively & year of birth: [Note – no CIs provided] [537]

Eastern High;

year of birth ≤ 1889 OR = 2.18
CI not supplied however note provided CI did not include 1.00

year of birth 1890-1900 OR = 1.73

year of birth ≥ 1901 OR = 1.00

Eastern Low;

year of birth ≤ 1889 OR = 0.93

year of birth 1890-1900 OR = 7.00
CI not supplied however note provided CI did not include 1.00

year of birth ≥ 1901 OR = 1.00

Southern High;

year of birth ≤ 1889 OR =1.38

[537] Page 213 table 2.

year of birth 1890-1900 OR = 1.82

year of birth ≥ 1901 OR = 0.74

Southern Low;

year of birth ≤ 1889 OR = 2.03
CI not supplied however note provided CI did not include 1.00

year of birth 1890-1900 OR = 2.24

year of birth ≥ 1901 OR = 1.34

Northern High;

year of birth ≤ 1889 OR = 3.30

year of birth 1890-1900 OR = 2.90
CI not supplied however note provided CI did not include 1.00

year of birth ≥ 1901 OR = 1.0

Northern low;

year of birth ≤ 1889 OR =1.00

year of birth 1890-1900 OR = 1.51

year of birth ≥ 1901 OR = 1.00

561. The authors made the following observation.

> It is interesting that higher relative risks for chronic lymphatic leukemia occurred among farmers residing in counties associated with cattle and dairy products, a pattern distinctly different from the other cell types. This resembles findings from epidemiologic studies of well-known leukemogens such as radiation and benzene that indicate the etiology of chronic lymphatic leukaemia differs from other cell types'[538]

562. The authors observed the following limitations of their study:

> These findings must be interpreted cautiously since they depend upon data from death certificates and indirect measuring of agricultural exposure. Inaccurate recording or classification of cause of death or usual occupation on the death certificate would tend to dilute the strength of theses associations. Use of death certificates afforded no opportunity to compare cases and controls for potential confounding factors such as tobacco use. [539]

[538] Page 213-214.

[539] Page 214.

563. For some cell types, the number of cases was quite small.

564. Many comparisons were made, therefore, several statistically significant ORs would be expected to occur due to chance alone.

Council's comments

565. The Council noted that overall the CI for CLL was 1.7 (and CI “did not include 1”) for farming occupational groups vs non-farmers. A mortality not incidence study and death certificates may not mention underlying CLL (as the authors concede). The study was poorly designed and presented, but nevertheless showed statistically significant elevated data (OR’s).

566. However, the absence of the methodology for the determining the above data shown in Tables 1 and 2 by the authors made it difficult for the Council to give weight to the results.

567. The Council considered this study provides some evidence in favour of an association for pesticide use and B-cell CLL, assuming cattle farmers use herbicides and pesticides.

568. The Council considered this study:

- did not point to but merely left open the possibility of a relevant association.

Burmeister, LF et al. 1982, ‘Leukemia and farm practices in Iowa’, *Am J Epidemiol*, vol. 115, no. 5, pp. 720-728. RMA ID 29619 FILEForce ID 19496

569. This retrospective case control survey considered the association of leukaemia with agricultural practices in Iowa. Death certificates for 1,675 white male Iowans over aged 30 who died from leukaemia from 1964 -1978 were compared to controls (2 non-leukaemia deaths per case) matched for sex, race, county of usual residence, age at death (+/- 2 yrs) and calendar year of death. Usual occupation as recorded on the death certificate was used to assess occupational association.

570. Research methods from Blair, AD & White, A 1985 (See from [557]) were replicated for this study.

571. Iowa counties were ranked according to:

- 1949 crop and livestock production provided by the Iowa Crop and Livestock Reporting Service; and

- Pesticide usage provided by the 1964 agricultural census; and

– Geographical distributions for the highest producing / usage 33 Iowa counties were established for milk cows; born; soybeans; herbicides and egg laying chickens. [540]

572. The authors observed the following Odds ratios for white male Iowa farmers according to their year of birth for CLL (ICD 8th Revision code 204.1): [541]

year of birth before 1890	OR = 1.00	95% CI 0.54 - 1.85
year of birth 1890-1900	OR = 2.03	95% CI 1.24 - 3.10
year of birth after 1900	OR = 1.80	95% CI 1.09 - 2.97

573. The authors observed the following Odds ratios for white male Iowa farmers according to selected county crop and livestock production 1968-1978 for CLL: [542]

Soybeans /acre		
Highest 33 counties	OR = 2.21	95% CI 1.33 - 3.66
Other counties	OR = 1.39	95% CI 0.97 - 2.00
Corn/acre		
Highest 33 counties	OR = 1.34	95% CI 0.87 - 2.06
Other counties	OR = 1.94	95% CI 1.29 - 2.92
Milk cows		
Highest 33 counties	OR = 1.19	95% CI 0.76 - 1.85
Other counties	OR = 2.10	95% CI 1.41 - 3.14
Egg laying chickens		
Highest 33 counties	OR = 1.36	95% CI 0.82 - 2.25
Other counties	OR = 1.80	95% CI 1.25 - 2.59
Herbicides		
Highest 33 counties	OR = 1.89	95% CI 1.16 - 3.08
Other counties	OR = 1.50	95% CI 1.03 - 2.18

[540] Page 722 figure 1.

[541] Page 726 table 5.

[542] Page 726 table 6.

574. The authors consider the results as a group without specific discussion of CLL.

Council's comments

575. Council considered this is a very old study, predating the Morris Brown et al. study of Iowa and Minnesota farmers discussed from [544] above, using now dated methodology. It used occupation on death certificate as record of occupation, which Council considers an inadequate methodology. The use of exposure by farmer and non-farmer was a very indirect measure. Council noted that it is not cited in recent reviews.

576. Council noted that the overall estimated RR for CLL (248 exposed cases) was 1.9 (1.2 - 3.1).

577. Overall Council considered this study provided:

- weak supportive evidence of an association; and
- did not point to but merely left open the possibility of a relevant association.

Lee, WJ et al. 2004, 'Cancer incidence among pesticide applicators exposed to alachlor in the agricultural health study', *Am J epidemiol*, vol. 159, no. 4, pp. 373-380. RMA ID 40648 FILEForce ID 19087

578. This prospective cohort study (The Agricultural Health Study) considered the association of cancer in 26,510 pesticide applicators with exposure to alachlor (2-chloro-2',6'-diethyl-N-(methoxymethyl) acetamilide a herbicide used mainly in production of corn, soybeans and peanuts) who were recruited between 1993 – 1997 in Iowa and North Carolina.

579. Cohort members were matched to cancer registry files in Iowa and North Caroline and to state death registries.

580. Date was obtained via self-administered enrolment questionnaire and included exposure data on 22 pesticides, ever/never use information for 28 additional pesticides and data on use of personal protective equipment, pesticide application methods, pesticide mixing, equipment repair, smoking, alcohol consumption, cancer history of first-degree relatives and basic demographic data.

581. Two lifetime alachlor exposure variables were constructed:

- Lifetime exposure days based on the number of years applied and frequency of application; and
- Intensity weighted exposure days.

582. The authors found the following adjusted rate ratios (RR) for applicators exposure compared to applicators not exposed to alachlor. [543]

All lymphohematopoietic cancers (ICD-9 200 - 208) – (70 vs 65 observed cases)	RR = 0.83	95% CI 0.54 - 1.26
Non Hodgkin lymphoma (ICD-9 200, 202) - (29 vs 32 observed cases	RR = 0.50	95% CI 0.26 - 0.94
Leukemia (ICD-9 204-208) – (26 vs 19 observed cases)	RR = 1.53	95% CI 0.72 - 3.25

583. The authors found the following adjusted rate ratios (RR) for lifetime alachlor exposure days and intensity weighted alachlor exposure days for:[544]

All lymphohematopoietic cancers

Lifetime alachlor exposure days (*p* value = 0.02)

0.1-19.9 (14 cases)	RR = 1.0	95% CI not supplied
20.0-56(12 cases)	RR = 0.67	95% CI 0.27 - 1.66
56.1-116 (16 cases)	RR = 1.59	95% CI 0.70 - 3.63
≥116.1 (26 cases)	RR = 2.04	95% CI 0.89 - 4.65

Intensity weighted alachlor exposure days (p value = 0.03)

0.1-101.9 (15 cases)	RR = 1.0	95% CI not supplied
102.0-253.1 (12 cases)	RR = 0.99	95% CI 0.40 - 2.44
253.2-710.4 (18 cases)	RR = 2.14	95% CI 0.95 - 4.83
≥710.5 (23 cases)	RR = 1.00	95% CI 1.00 - 5.89

Leukemia

Lifetime alachlor exposure days (p value = 0.05)

0.1-19.9 (6 cases)	RR = 1.0	95% CI not supplied
20.0-56 (6 cases)	RR = 0.76	95% CI 0.19 - 3.07
56.1-116 (4 cases)	RR = 1.16	95% CI 0.28 - 4.82
≥116.1 (10 cases)	RR = 3.01	95% CI 0.82 - 11.0

Intensity weighted alachlor exposure days (p value = 0.17)

0.1-101.9 (7 cases)	RR = 1.0	95% CI not supplied
102.0-253.1 (7 cases)	RR = 0.99	95% CI 0.27 - 3.58
253.2-710.4 (3 cases)	RR = 0.85	95% CI 0.19 - 3.73
≥710.5 (9 cases)	RR = 2.83	95% CI 0.74 - 10.9

543 Page 376 table 2.

544 Page 377 table 3.

NHL

Lifetime alachlor exposure days (p value = 0.53)		
0.1-19.9 (5 cases)	RR = 1.0	95% CI not supplied
20.0-56(4 cases)	RR = 0.55	95% CI0.12 - 2.48
56.1-116 (8 cases)	RR = 1.50	95% CI 0.42 - 5.40
≥116.1 (10 cases)	RR = 1.14	95% CI 0.30 - 4.36
Intensity weighted alachlor exposure days (p value = 0.42)		
0.1-19.9 (5 cases)	RR = 1.0	95% CI not supplied
20.0-56(3 cases)	RR =0.62	95% CI 0.11 - 3.44
56.1-116 (10 cases)	RR = 2.40	95% CI 0.65 - 8.82
>116.1 (9 cases)	RR = 1.40	95% CI 0.32 - 6.11

584. The authors concluded: [545]

> …a significant exposure-response trend was observed for all lymphohematopoietic cancers combined using either the lifetime exposure-days or intensity-weighted exposure–days, rising to an over twofold increased risk in the highest category.

585. The authors noted the following limitations of their study:

- the difficulty in assessing exposure to a single agent in pesticide applicators;
- Changes in the formulation of alachlor over the years studied; and
- Low case numbers for NHL (27) and leukemia (26).

Council's comments

586. The Council considered this study showed a significant dose exposure effect for leukaemia (at [582] [583] above) and therefore was weak support for the relevant association, noting there was no B-cell CLL specific data.

587. The Council considered this study:

- did not point to, but merely left open the possibility of a relevant association.

Mills, PK et al. 2005, 'Lymphohematopoietic cancers in the United Farm Workers of America (UFW), 1988-2001', *Cancer Causes & Control*, vol. 16, pp. 823-830. RMA ID 38743 FILEForce ID 19081

[545] Page 377.

588. This nested case-control study examined associations between 131 Hispanic farm workers diagnosed with lymphohematopoietic cancers in California between 1988 and 2001.

589. Cases were obtained from the California Cancer Registry and linked with Hispanic farm workers who were 'ever' members of the United Farm Workers of America (UFW). Exposure data was obtained via duration of union membership. Data from the UFW also provided data on growers including nature of crops and commodities most commonly produced an as well as geographic location. Cases were assessed for work histories listing when and where they worked. For each case, five members of the UFW who had not been diagnosed with any cancer were matched on gender, Hispanic ethnicity and +/- one year of birth.

590. Information on pesticide use was obtained from the Pesticide databank, a database of historical pesticide use records collected by the California Department of Food and Agriculture and maintained at the University of California, Davis.

591. Types of lymphohematopoietic cancers were evaluated separately.

Cancer	Males	Females	Total
All Non-Hodgkin's Lymphoma	45	15	60
Nodal NHL	28	10	38
Extra nodal NHL	17	5	22
All Leukemia	35	16	51
Lymphocytic Leukemia	16	7	23
Granulocytic Leukemia	12	8	20
Other Leukemia	7	1	8
Multiple Myeloma	14	6	20
Total	94	37	131

592. The authors provided the following age and sex adjusted odds ratios for lymphohematopoietic cancers by work in usual crop/commodity. [546]

Citrus	OR = 0.63	95%CI 0.29 - 1.33
Vegetables	OR = 1.67	95%CI 1.12 - 2.48
Grapes	OR = 0.85	95%CI 0.59 - 1.23
Horticulture	OR = 0.48	95%CI 0.19 - 1.24
Mushrooms	OR = 0.30	95%CI 0.04 - 2.34
Strawberries	OR = 1.34	95%CI 0.54 - 3.32

and the results for work with vegetables by sex for lymphohematopoietic cancers for:

Males	OR = 1.76	95% CI not given
Females	OR = 1.40	95% CI not given

and the results for works in vegetables, female, diagnosed with leukaemia:

[546] Page 825 table 2.

	OR = 4.01	95%CI 1.11 - 14.56

593. The authors provided data on a comparison of low and high areas of use of a number of chemicals and the adjusted odds ratio for 23 cases of lymphocytic leukaemia, [547] and the adjusted odds ratio for 60 cases of NHL (combined 38 cases of NHL – nodal and 22 cases of NHL extranodal). [548]

Methyl bromide		
Lymphocytic Leukaemia	OR = 2.07	95%CI 0.64 - 6.73
NHL	OR = 1.48	95%CI 0.81 - 2.71
Diazinon		
Lymphocytic Leukaemia	OR = 1.42	95%CI 0.46 - 4.43
NHL	OR = 1.39	95%CI 0.76 - 2.53
Malathion		
Lymphocytic Leukaemia	OR = 2.88	95%CI 0.94 - 8.80
NHL	OR = 1.77	95%CI 0.99 - 3.17
Dichloro-propane		
Lymphocytic Leukaemia	OR = 1.13	95%CI 0.37 - 3.44
NHL	OR = 0.89	95%CI 0.48 - 1.66
Captan		
Lymphocytic Leukaemia	OR = 1.17	95%CI 0.37 - 3.65
NHL	OR = 0.85	95%CI 0.46 - 1.57
Simazine		
Lymphocytic Leukaemia	OR = 1.53	95%CI 0.48 - 4.90
NHL	OR = 1.65	95%CI 0.89 - 3.04
Chlorothalonil		
Lymphocytic Leukaemia	OR = 2.06	95%CI 0.65 - 6.60
NHL	OR = 1.15	95%CI 0.60 - 2.18
Mancozeb		
Lymphocytic Leukaemia	OR = 2.32	95%CI 0.71 - 7.58
NHL	OR = 0.92	95%CI 0.45 – 1.88

[547] Page 826 table 3.

[548] Page 826 table 3.

Methyl parathion		
Lymphocytic Leukaemia	OR = 2.54	95%CI 0.83 - 7.77
NHL	OR = 0.62	95%CI 0.31 - 1.24
Nitrofen		
Lymphocytic Leukaemia	OR = 2.22	95%CI 0.63 - 7.79
NHL	OR = 1.24	95%CI 0.62 - 2.47
Propyzamide		
Lymphocytic Leukaemia	OR = 2.19	95%CI 0.60 - 7.96
NHL	OR = 0.68	95%CI 0.34 - 1.36
Toxaphene		
Lymphocytic Leukaemia	OR = 2.72	95%CI 0.80 - 9.22
NHL	OR = 0.94	95%CI 0.48 - 1.86
Trifluralin		
Lymphocytic Leukaemia	OR = 1.90	95%CI 0.60 - 6.06
NHL	OR = 0.89	95%CI 0.44 - 1.78
2,4-D		
Lymphocytic Leukaemia	OR = 1.47	95%CI 0.33 - 6.59
NHL	OR = 3.80	95%CI 1.85 - 7.81
Maneb		
Lymphocytic Leukaemia	OR = 2.67	95%CI 0.84 - 8.52
NHL	OR = 1.10	95%CI 0.59 - 2.07

594. The authors provided the following adjusted odds ratios by sex for leukaemia (male n = 35; female n= 16) and for NHL (male n = 45; female n = 15) and specific chemicals [549]:

Methyl bromide			
Leukaemia	Male	OR = 0.90	95%CI 0.37 - 2.19
	Female	OR = 2.70	95%CI 0.80 - 9.13
NHL	Male	OR = 1.23	95%CI 0.59 - 2.57
	Female	OR = 3.78	95%CI 1.11 - 12.82
Diazinon			
Leukaemia	Male	OR = 0.90	95%CI 0.37 - 2.19
	Female	OR = 2.70	95%CI 0.80 - 9.13
NHL	Male	OR = 1.97	95%CI 0.97 - 4.0
	Female	OR = 0.80	95%CI 0.23 - 2.81

[549] Page 929 table 4.

Malathion			
leukaemia	Male	OR = 1.19	95%CI 0.51 - 2.76
	Female	OR = 4.91	95%CI 1.21 - 19.89
NHL	Male	OR = 2.01	95%CI 0.99 - 4.1
	Female	OR = 1.92	95%CI 0.60 - 6.18
Dichloro-propane			
Leukaemia	Male	OR = 1.06	95%CI 0.45 - 2.52
	Female	OR = 2.28	95%CI 0.67 - 7.82
NHL	Male	OR = 0.97	95%CI 0.47 - 2.02
	Female	OR = 0.97	95%CI 0.28 - 3.39
Captan			
Leukaemia	Male	OR = 1.06	95%CI 0.44 - 2.54
	Female	OR = 1.55	95%CI 0.43 - 5.62
NHL	Male	OR = 1.07	95%CI 0.52 - 2.20
	Female	OR = 0.57	95%CI 0.15 - 2.89
Simazine			
Leukaemia	Male	OR = 0.76	95%CI 0.29 - 2.01
	Female	OR = 0.95	95%CI 0.26 - 3.49
NHL	Male	OR = 1.64	95%CI 0.78 - 3.44
	Female	OR = 2.65	95%CI 0.83 - 8.50
Chlorothalonil			
Leukaemia	Male	OR = 1.28	95%CI 0.53 - 3.09
	Female	OR = 4.78	95%CI 1.11 - 20.44
NHL	Male	OR = 1.43	95%CI 0.68 - 2.98
	Female	OR = 0.71	95%CI 0.15 - 3.28
Mancozeb			
Leukaemia	Male	OR = 1.83	95%CI 0.74 - 4.51
	Female	OR = 4.78	95%CI 1.11 - 20.44
NHL	Male	OR = 1.34	95%CI 0.60 - 2.96
	Female	OR = 0.22	95%CI 0.02 - 2.07
Methyl parathion			
Leukaemia	Male	OR = 1.50	95%CI 0.63 - 3.59
	Female	OR = 1.87	95%CI 0.49- 7.0
NHL	Male	OR = 0.70	95%CI 0.31 - 1.58
	Female	OR = 0.55	95%CI 0.12 - 2.46
Nitrofen			
Leukaemia	Male	OR = 1.76	95%CI 0.71 - 4.34

	Female	OR = 3.08	95%CI 0.71 - 13.3
NHL	Male	OR = 1.53	95%CI 0.69 - 3.38
	Female	OR = 0.88	95%CI 0.17 - 4.46
Propyzamide			
Leukaemia	Male	OR = 1.45	95%CI 0.56 - 3.72
	Female	OR = 2.93	95%CI 0.74 - 11.5
NHL	Male	OR = 0.80	95%CI 0.35 - 1.82
	Female	OR = 0.56	95%CI 0.12 - 2.50
Toxaphene			
Leukaemia	Male	OR = 1.88	95%CI 0.75 - 4.75
	Female	OR = 3.78	95%CI 0.93 - 15.35
NHL	Male	OR = 1.12	95%CI 0.51 - 2.45
	Female	OR = 0.74	95%CI 0.17 - 3.26
Trifluralin			
Leukaemia	Male	OR = 1.33	95%CI 0.55 - 3.25
	Female	OR = 4.51	95%CI 1.24 - 16.38
NHL	Male	OR = 1.60	95%CI 0.44 - 2.24
	Female	OR = 0.87	95%CI 0.20 - 3.73
2,4-D			
Leukaemia	Male	OR = 0.55	95%CI 0.15 - 2.06
	Female	OR = 3.73	95%CI 0.77 - 18.00
NHL	Male	OR = 3.79	95%CI 1.58 - 9.11
	Female	OR = 5.23	95%CI 1.30 - 20.90
Maneb			
Leukaemia	Male	OR = 1.49	95%CI 0.62 - 3.56
	Female	OR = 2.94	95%CI 0.74 - 11.60
NHL	Male	OR = 1.47	95%CI 0.70 - 3.08
	Female	OR = 0.62	95%CI 0.15 - 2.57

595. The authors made note of the associations between leukaemia and malathion, mancozeb, chorothalonil and trifluralin and in both males and females the association between 2,4 D and NHL.

596. The authors made reference to other studies showing an association between chronic lymphocytic leukaemia and pesticidal exposures.[550]

[550] References:

Blair A, White DW 1985, Leukemia cell types and agricultural practices in Nebraska Arch Environ Health 40: 211-214.

597. The authors noted the following limitations of their study;

- Relatively small sample size;
- Wide range of pesticide exposure data obtained; and
- Case controls were not interviewed and only work histories were available which allowed only a small amount of variables to be controlled for.

Council's comments

598. The Council considered that the authors made a good attempt to define exposure, however exposure was inferred from job and crop histories rather than an individual's actual exposure. Further, the study is underpowered with small case number and the majority of results are not statistically significant.

599. The Council considered important the trend in results for 23 cases of lymphocytic leukaemia (see [593]) with elevated OR's (greater than 1) and in the same direction, but not statistically significant for all the chemicals listed.

600. The Council considered this study:

- was weakly supportive of an association; and
- did not point to but merely left open the possibility of a relevant association.

Read, D et al. 2007, 'Cancer incidence and mortality in a New Zealand community potentially exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin from 2,4,5-trichlorophenoxyacetic acid manufacture', *Aust N Z J Public Health*, vol. 31, no. 1, pp. 13-18. RMA ID 45664 FILEForce ID 19103

601. This retrospective cohort analysis considered the association between dioxin exposure and cancer rates in New Plymouth, the site of a former 2,4,5-T manufacturing plant and the rest of New Zealand.

602. Cancer data was obtained from the New Zealand cancer registry from 1970-2001 for 8,013 cancer registrations and 4,235 cancer deaths in New Plymouth and for 367,570 cancer registrations and 192,379 cancer deaths in the rest of New Zealand.[551] Mortality and cancer registrations for the 2000 and 2001 years were

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Brown LM et al. 1990, Pesticide exposures and other agricultural risk factors for leukemia among med in Iowa and Minnesota Cancer Res 50: 6585-6591.

Sperati et al. 1999, mortality among male licensed pesticide users and their wives. Am J Indust Med 36: 142-146.

Study reviewed mortality data on 2978 male farmers and 2586 farmers wives from 1971-1973. Lymphoid leukaemia males SMR 1.68 (.35-4.91) and female SMR 1.69 (0.04-9.41).

This article by Sperati et al. 1999, was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

551 Page 14 table 1.

coded under the ICD 10th Revision and the data for earlier years were coded under a combination of ICD-8 and ICD-9:

Description	ICD-8	ICD-9	ICD-10
All cancers	140-209	140-208	C00-C97
Non-Hodgkin's lymphoma	200.0-200.8, 202.0-202.2, 202.8-202.9	200.0-200.8, 202.0-202.2, 202.8-202.9	C830, C833, C837, C822, C840, C841, C845, C859, C967
Chronic lymphocytic leukaemia	204.1	204.1	C911

603. The authors found the following standardized mortality rates (SMR) for CLL according to 5 year periods:[552]

Period 1970-74	(7 deaths)	SMR = 169	95%CI 68 - 349
Period 1975-79	(7 deaths)	SMR = 175	95%CI 70 - 361
Period 1980-84	(6 deaths)	SMR = 137	95%CI 50 - 298
Period 1985-89	(4 deaths)	SMR = 84	95%CI 23 - 217
Period 1990-94	(6 deaths)	SMR = 113	95%CI 42 - 250
Period 1995-99	(8 deaths)	SMR = 133	95%CI 57 - 264
Period 2000-01	(2 deaths)	SMR = 78	95%CI 9 - 284

604. The authors found the following standardized incidence rates (SIR) for CLL according to 5 year periods: [553]

Period 1970-74	(16 cases)	SIR = 251	95%CI 144-408
Period 1975-79	(7 cases)	SIR = 87	95%CI 35-180
Period 1980-84	(21 cases)	SIR = 255	95%CI 158-391
Period 1985-89	(16 cases)	SIR = 143	95%CI 82-234
Period 1990-94	(13 cases)	SIR = 90	95%CI 48-155
Period 1995-99	(19 cases)	SIR = 88	95%CI 53-139
Period 2000-01	(12 cases)	SIR = 107	95%CI 55-187

605. The authors note the following challenge when interpreting the data from the period 1970-74: [554]

> The period 1970-74 is the only one that shows an elevated cancer risk for all cancers and at least one of the four specific cancers. Assuming a 10-year minimum latency period and the cause was TCDD, the period of exposure would have been 1960-64, which is partially outside of the 2,4,5-T manufacturing period and before TCP was manufactured on site

606. The authors noted: [555]

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552 Page 15 table 3.

553 Page 16 table 4.

554 Page 16.

555 Page 17.

> Although increases were seen for CLL and NHL in two time periods, the large confidence intervals resulting from a small number of cases make it difficult to draw conclusions from the data.

Council's comments

607. The Council noted that 5 of 7 time periods for mortality rates show an elevation in risk, but not to significance. For incidence 2 out of 7 time periods show a statistically significant increase in risk. However, the Council considered the elevation and the author's results for incidence generally do not support a true biological effect from dioxin contaminants-exposure due to the exposure and latency period issues highlighted by the authors. Further, the Council considered important that there was no stronger relationship over time, which while this was not against any relationship it was not in favour either.

608. The Council considered this study:

- did not point to but leaves open the possibility of a relevant association.

Institute of Medicine (IOM) 1994, update 2002, update 2004, update 2006

609. The NAS IOM reports' [556] findings [557] are summarised on the basis of an association (if any exists) between specific health problems and exposure to herbicides:

> …sufficient evidence of an association, limited/suggestive evidence, inadequate /insufficient evidence to determine whether an association exists and limited/suggestive evidence of no association.
>
> …methodologically the findings were determined on whether a statistical association exists, the increased risk of each disease for those exposed during Vietnam service (United States

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556 On 6 February 1991 the Congress of the United States of America directed the Secretary of Veterans Affairs to request the National Academy of Sciences (NAS) to conduct a comprehensive review and evaluation of the available scientific and medical information regarding the health effects of exposure to Agent Orange and other herbicides used during the Vietnam conflict.

The NAS is a nongovernmental organisation dedicated to the furtherance of science and technology and to their use for the promotion of general public welfare. The 16 members of the NAS Institute of Medicine (IOM) Committee which undertook the review and evaluation (as with all IOM committees), voluntarily serve without compensation and are drawn from Universities and the private sector, and are elected by their peers on the basis of exemplary professional achievement. This Committee's expertise included experts from occupational and environmental medicine, toxicology, epidemiology, pathology, clinical oncology, psychology, neurology and biostatistics. As with all reports from the IOM, the committee's work was reviewed by an independent panel of distinguished experts.

557 NAS IOM 1994; Summary of findings set out in Executive Summary Table 1-1 at p. 6.

The Council notes that the methodology of the NAS is to determine whether a statistical association exists; the increased risk of each disease for those individuals exposed during Vietnam service; and whether a plausible biologic mechanism or other evidence of a causal relationship exists (see page 5 of the 1994 – Executive Summary).

This methodology does not reflect the Council's two-step process in applying the reasonable hypothesis test as set down by the full Federal Court (see [108] - [109]).

of America forces) and whether a plausible biologic mechanism or other evidence of a causal relationship exists. [558]

610. The 1994 authors concluded with regard to strength of evidence for leukaemia without clarifying sub type: [559]

> …there is inadequate or insufficient evidence to determine wether an associations exists between exposure to herbicides (2,4-D; 2,4,5-T and its contaminant TCDD; cacodylic acid and picloram) and leukaemia.

611. The update 1996 [560] and 1998 [561] and 2000[562] conclusion was unchanged from that stated in 1994.

612. The 2002 update was the first to consider CLL separately from other forms of leukaemia. The Committee noted:

> In the proposed World Health Organization classification of non-Hodgkin's lymphoid neoplasms, CLL and its lymphomatous form, small lymphocytic lymphoma, are mature B-cell neoplasms (Jaffe et al., 2001). …
>
> The requirements for diagnosis of CLL include an absolute peripheral-blood lymphocyte count of more than 10 x 109 per liter, a predominant population of mature-looking lymphocytes, and a hypercellular or normal cellular bone marrow …
>
> Diffuse small-cell lymphocytic lymphoma is the term for the condition of patients with lymphomatous presentation of CLL. Patients seek medical attention for painless generalized lymphadenopathy that in many cases has lasted for several years. Unlike the situation in CLL, the peripheral blood may be normal or reveal only mild lymphocytosis. However, the bone marrow is positive in 75-95% of cases. Both small-cell lymphocytic lymphoma and CLL can transform into aggressive NHL known as Richter's syndrome …
>
> In response to a request … and because CLL shares more traits (immunohistochemical characteristics, B-cell origin, and progression to an acute aggressive form of NHL) with NHL than with other types of leukemia, the committee reassessed the available epidemiologic data to determine whether CLL merited reclassification …[563]

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558 NAS IOM 1994, at p. 221.

559 Page 571.

560 NAS IOM update 1996, at p. 247.

561 NAS IOM update 1998, at p. 390.

562 NAS IOM update 2000, at p. 380.

563 Page 283.

613. The Committee concluded that for CLL there was sufficient evidence of an association and the US Department of Veterans Affairs extended benefits to Vietnam Veterans with CLL. [564]

> ...there is sufficient evidence of an association between exposure to at least one of the chemicals of interest (2,4-D; 2,4,5-T and its contaminant TCDD; cacodylic acid and picloram) and CLL.

614. The 2004 update review of literature concluded, similar to the update 2002 that there was sufficient evidence of an association between exposure to at least one of the chemicals of interest and CLL. [565]

615. The 2006 update review of literature concluded:

> …that there is sufficient evidence of an association between exposure to the compounds of interest and CLL. [566]

616. The Council's overall consideration of the IOM reviews and the studies therein, can be found after Council's description and comments on Update 2002 and 2006 below.

Institute of Medicine 2003, 'Veterans and Agent Orange Update 2002, The National Academic Press, Washington DC 2003. www.iom.edu/.../Veterans-and-Agent-Orange-Update-2002.aspx RMA ID 29493 FILEForce ID 19414

617. This update is the 5th in the series from the first 1994 publication of the National Academy of Sciences (NAS), Institute of Medicine's committee detailing review of the scientific evidence of the possible health effects to Vietnam Veterans following exposure to Agent Orange and other herbicides in Vietnam. It is the first of the IOM report series to include a separate subsection on CLL.

618. The authors summarized studies reviewed in the 1998 publication with ERR values for CLL: 567

a. Waterhouse et al. 1996; [568] see from [509]

   – Residents of Tecumseh Michigan

   Cases = 10 SIR = 0.8 95% CI 0.8 - 3.2

564 NAS IOM update 2002, at p. 376.

565 NAS IOM update 2004, at p 337

566 NAS IOM update 2006, at p. 409.

567 Page 374 table 6.50.

568 Waterhouse, D et al. 1996, 'Cancer incidence in the rural community of Tecumseh, Michigan. A pattern of increased lymphopoietic neoplasms', *Cancer*, vol. 77, no.4, pp.763-770.

b. Amadori et al. 1995, [569] - see from [522]

– CLL in Italian workers:

Farming or animal-breeding workers;
Cases = 15 ERR = 2.3 95% CI 0.9 - 5.8

Farming;
Cases = 5 ERR = 1.6 95% CI 0.5 - 5.2

Animal – breeding;
Cases = 10 ERR = 3.1 95% CI 1.1 - 8.3

619. The authors summarized studies reviewed in previous reviews in the series Veterans and Agent Orange: Health effects of Herbicides Used in Vietnam – with ERR values for CLL: [570]

a. Hansen et al. 1992, [571] - see from [533]

– Danish gardeners

all gardeners CLL	Cases = 6	ERR = 2.5	95% CI 0.9 - 5.5
male gardeners CLL	Cases = 6	ERR = 2.8	95% CI 1.0 - 6.0

b. Morris-Brown et al. 1990, [572] - see from [544]

– Residents of Iowa and Minnesota

CLL ever farmed	Cases = 156	ERR = 1.4	95% CI 1.1 - 1.9
CLL any herbicide use	Cases = 74	ERR = 1.4	95% CI 1.0 - 2.0

c. Blair and White 1985, [573] - see from [557]

– Residents of Nebraska;

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569 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.

570 Page 374 table 6.50.

571 Hansen, ES 1992, 'A cohort study on cancer incidence among Danish gardeners', *Am J Ind Med*, vol.21, no.5, pp. 651-660.

572 Morris Brown L et al. 1990, 'Pesticide exposures and other agricultural risk factors for leukemia among med in Iowa and Minnesota', Cancer Res 50: 6585-6591.

573 Blair, A & White, DW 1985, 'Leukemia cell types and agricultural practices in Nebraska, *Arch Environ Health,* 40: 211-214.

All cases, all leukemia – farming;

Cases = 1084		ERR = 1.3	
CLL	Cases = 248	ERR = 1.7	CI did not include 1

d. Burmeister et al. 1982, [574] - see from [569]

CLL in white male farmers using herbicides	ERR = 1.9	95% CI 1.2 - 3.1

620. The authors summarized studies reviewed in update 2000 with ERR values for CLL [575]

a. Bertazzi et al. 2001[576] Follow-up of the population exposed to dioxin after the 1976 accident in Seveso, Italy extended to 1996. Three containment zones (A,B and R) and total population of nearly 300,000. (See list of non contributory articles)

Lymphatic leukaemia

Zone A		ERR = 0	
Zone b	cases = 2	ERR = 1.1	95% CI 0.3 - 4.4
Total	cases = 2	ERR = 1.0	95% CI 0.2 - 3.9

621. The authors noted that no relevant Vietnam-veteran studies which specifically investigate CLL had been published since Update 2000.

622. The authors concluded: [577]

> The reanalysis of the epidemiologic studies indicates that farming occupation, especially where there is exposure to the herbicides 2,4-D and 2,4,5-T, is associated with significant risk of CLL mortality. Many more studies support the hypothesis that herbicide exposure can contribute to NHL risk. Most cases of CLL and NHL reflect malignant transformation of B-lymphocyte progenitor cells, so these diseases could have a common aetiology.
>
> Based on its evaluation, the committee concludes that there is sufficient evidence of an association between exposure to at least one of the chemicals of interest (2,4-D, 2,4,5-T or its contaminant TCDD, picloram or cacodylic acid) and CLL.

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574 Burmeister, LF et al. 1982, 'Leukemia and farm practices in Iowa', *Am J Epidemiol*, vol. 115, no. 5, pp. 720-728.

575 Page 374 table 6.50.

576 Bertazzi et al 2001 'Health effects of dioxin exposure: a 20-year mortality study. American Journal of Epidemiology 153 (11): 1031-1041.

577 Page 375 & 376.

> The limited data available on Vietnam veterans do not suggest that they are at increased risk for CLL.

Institute of Medicine 2007, 'Veterans and Agent Orange Update 2006, Committee to review the health effects in Vietnam veterans of exposure to herbicides (sixth biennial update) The National Academic Press, Washington DC 2003 RMA ID 47578 FILEForce ID 19450

623. The authors of this 7th publication in the series considered two new studies since the review in 2004. [578]

a. Hertzman et al. 1997, [579] see from [631]

– British Columbia sawmill workers;

CLL	exposed cases = 24	ERR = 1.7	95% CI 1.2 - 2.4

b. ADVA 2005 / Wilson et al. 2005a, [580] see from [641]

– Australian Vietnam veterans vs Australian population incidence of CLL

all branches	cases = 58	ERR = 1.55	95% CI 1.15 - 1.95
Navy	cases = 12	ERR = 1.51	95% CI 0.78 - 2.63
Army	cases = 42	ERR = 1.68	95% CI 1.18 - 2.19
Air Force	cases = 4	ERR = 0.87	95% CI 0.24 - 2.23

624. The authors concluded: [581]

> On the basis of its evaluation of the evidence reviewed here and in previous VAO reports, the committee concludes that there is sufficient evidence of an association between exposure to the compounds of interest and CLL.

Council's comments on IOM review reports Update 2002 and Update 2006

625. The Council was not as convinced as the IOM by the data reviewed in update 2002. The Council's view on considering 7 of the articles which were also reviewed by the IOM, was that one study (Morris Brown, L et al. 1990) points to the possibility of a

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578 Page 406-407 at Table 6.49.

579 Hertzman C et al. 1997, 'Mortality and cancer incidence among sawmill workers exposed to chlorophenate wood preservatives', *American Journal of Public Health*, vol. 87, no. 1, pp. 71-79.

580 Wilson, EJ et al. 2005a, *Cancer Incidence in Australian Vietnam Veterans study*, Department of Veterans' Affairs, Canberra.

581 Page 408.

relevant association and that 5 other studies merely left open the possibility of such an association.

626. The Council's consideration of the 7th study by Bertazzi, PA et al. 2001 was that it was non contributory as it is not specific to B-cell CLL. While it reports 2 cases of lymphatic leukaemia and 28 cases of lymphohemopoietic cancers, the Council was of the view that conclusions on that data could not be made for B-cell CLL.

627. The Council considered the studies (reviewed in IOM update 2006) by Hertzman, C et al. 1997 and Wilson, EJ et al. 2005a were in support of an association for dioxin and B-cell CLL. Hertzman et al. 1997 is positive for the relevant association based on dioxin contaminated chlorophenates.

628. The Update 2002, 2004 and 2006 support an association but without data on individuals / cases, herbicide / pesticide exposure. The Council noted and considered important that the US IOM/NAS took the data, particularly including the Wilson, EJ et al. 2005a, Cancer Incidence in Australian Vietnam Veterans paper, as supportive to reach the conclusion that there was sufficient evidence for the purposes of its own review, of an association between herbicides/dioxin exposure and CLL.

629. The Council, cognizant of its task in this Review, was not as convinced as the IOM by Wilson, EJ et al. 2005a, which could not specify what exposure was related to the elevation of B-cell CLL risk reported in that study. To the extent that the exposure could be related to herbicides, pesticides and or dioxin in the Council's view the data was weak, but supportive of the possibility of a relevant association.

630. The Council considered in relation to the IOM reviews:

- Update 2002 did not point to but merely left open the possibility of a relevant association; and
- Update 2006 points to a relevant association; and
- considered together Updates 2002 and 2006 point to a relevant association

Hertzman, C et al. 1997, 'Mortality and cancer incidence among sawmill workers exposed to chlorophenate wood preservatives', *American Journal of Public Health*, vol. 87, no. 1, pp. 71-9. RMA ID 18955 FILEForce ID 20065

631. This retrospective cohort study considered the association between mortality and cancer in 23,829 sawmill workers exposed to chlorophenate wood preservatives, in British Columbia, Canada. The authors state that: [582]

[582] Page 71.

> Like the chlorophenoxy herbicides (2,4-D and 2,4,5,-T), chlorophenates belong to a class of dioxin contaminated substances.
>
> The dioxin isomers readily detected in them are hexa-, hepta-, and octa-chlorinated isomers rather than 2,3,7,8-tetrachlorodibenzo-p-dioxin…
>
> Workplace exposure to penta- and tetrachlorophenates occurs through direct skin contact… Breathing chlorophenate vapour, aerosols, and contaminated sawdust has also been a route of exposure…

632. Workers employed for at least 1 year between 1950 and 1985 in 11 chlorophenate-using (23,829 workers) and 3 non-using sawmills (2658 workers used as controls) were recruited.

633. Data was obtained from personal work records, the British Columbia Death File and the British Columbia Cancer Incidence File. Summary exposure scores were calculated for each job title over selected time periods.

634. The authors reported the following standardised mortality ratios (SMRs) for chlorophenate using and non-using saw mill workers for selected cancers. [583]

Exposed mill workers (n = 23 829)		
All NHL (36 cases)	SMR = 1.08	95% CI 0.80 - 1.43
Lymphosarcoma [584] (23 cases)	SMR = 1.46	95% CI 1.00 - 2.07
Other NHL (13 cases)	SMR = 0.74	95% CI 0.44 – 1.19
Leukemia (39 cases)	SMR = 0.96	95% CI 0.72 – 1.26
Unexposed mill workers (n = 2 658)		
All NHL (1 case)	SMR = 0.62	95% CI 0.20 - 2.94
Lymphosarcoma (0 cases)	SMR = 0.54	95% CI 0.00

635. The authors reported the following SMRs by cumulative hours of exposure to chlorophenates for selected cancers [585]

All NHL (37 cases)		
< 120 hours (3)	SMR = 0.90	95% CI 0.24 - 2.32

583 Page 73 table 2.

584 Council note that this is an outdated term, which no longer has any meaning. The data is included only to accurately describe the results of the study.

585 Page 75 table 3.

120 – 1,999 hrs (7)	SMR = 0.78	95% CI 0.36 - 1.46
2,000 – 3,999 hrs (3)	SMR = 0.47	95% CI 0.13 - 1.20
4,000 – 9,999 hrs (13)	SMR = 1.31	95% CI 0.77 - 2.08
≥ 10,000 hrs (11)	SMR = 0.88	95% CI 0.49 - 1.45)
Other NHL (16 cases)		
< 120 hours (2)	SMR = 1.04	95% CI 0.18 – 3.30
120 – 1,999 hrs (2)	SMR = 0.41	95% CI 0.07 - 1.28
2,000 – 3,999 hrs (2)	SMR = 0.58	95% CI 0.10 - 1.83
4,000 – 9,999 hrs (5)	SMR = 0.99	95% CI 0.39 – 2.09
≥ 10,000 hrs (3)	SMR = 0.43	95% CI 0.11 – 1.10

636. The authors reported the following standardized incidence ratios (SIR) for exposed and unexposed workers for selected diseases [586]

Exposed workers (n = 23 829)		
NHL (63 cases)	SIR = 1.20	95% CI 0.96 - 1.48
CLL (24 cases)	SIR = 1.67	95% CI 1.26 – 2.36
Non exposed workers (n = 2 658)		
NHL (2 cases)	SIR = 0.57	95% CI 0.10-1.80

637. The authors reported the following SIRs by cumulative hours of exposure to chlorophenates for NHL and CLL [587]

NHL (65 cases)		
< 120 hours (4 cases)	SMR = 0.68	95% CI 0.23 - 1.56 P = 0.04
120 – 1,999 hrs (9 cases)	SMR = 0.59	95% CI 0.31 - 1.03
2,000 – 3,999 hrs (11 cases)	SMR = 1.04	95% CI 0.59 – 1.74
4,000 – 9,999 hrs (15 cases)	SMR = 1.02	95% CI 0.63 - 1.57
≥ 10,000 hrs (26 cases)	SMR = 1.30	95% CI 0.91 - 1.80
CLL (25 cases)		

[586] Page 76 table 4.

[587] Page 77 table 5.

< 120 hours (2 cases)	SMR = 1.43	95% CI 0.24 - 4.50 *P* = 0.76
120 – 1,999 hrs (3 cases)	SMR = 0.84	95% CI 0.23 - 2.19
2,000 – 3,999 hrs (5 cases)	SMR = 1.97	95% CI 0.77 – 4.14
4,000 – 9,999 hrs (6 cases)	SMR = 1.40	95% CI 0.60 - 2.76
≥ 10,000 hrs (9 cases)	SMR = 1.39	95% CI 0.72 - 2.43

Council's comments

638. The Council considered this study relevant to the contentions regarding Herbicides / Dioxins.

639. The Council considered that the study results weakly support the possibility of a relevant association between chlorophenates and CLL, as the data for CLL shows an elevated statistically significant risk (SIR = 1.67), but no dose response was found and exposure was determined by the reconstruction of timelines for chemicals used at sawmills and by job titles, without specific individual dose measurements.

640. The Council considered this study:

- points to a relevant association.

Wilson, EJ et al. 2005a, *Cancer Incidence in Australian Vietnam Veterans study*, Department of Veterans' Affairs, Canberra. RMA ID 43077 FILEForce ID 19108

641. This retrospective cohort study of 57,864 living male Australian personal who served in Vietnam between 23 May 1962 and 1 July 1973, examined the incidence of cancer diagnosed during the period 1982 to 2000. The study compares the cancer incidence rates to the number expected based on cancer incidence in the Australian community.

642. Cancer diagnoses were obtained from the national cancer statistics clearing house. Cancer diagnoses were classified according to ICD-10 codes:

Leukaemia C91-C95

Non-Hodgkin's lymphoma C82-C85, C96

643. The authors reported the following standardised incidence ratios (SIR) for: [588]

Leukaemia (130 cases)	SIR = 1.18	95% CI 0.98 - 1.38

[588] Page 82 table 6.2.

NHL (126 cases) SIR = 0.67 95% CI 0.55 - 0.79

and for: [589]

CLL (58 cases) SIR = 1.55 95% CI 1.15 - 1.95

CLL among 12,935 Navy Vietnam veterans: [590]

CLL – Navy (12 cases) SIR = 1.51 95% CI 0.78 - 2.63

CLL among 39, 517 Army Vietnam veterans: [591]

CLL – Army (42 cases) SIR = 1.68 95% CI 1.18 - 2.19

CLL among 4, 388 Air Force Vietnam veterans: [592]

CLL – Air Force (4 cases) SIR = 0.87 95% CI 0.24 - 2.23

644. The authors discussed the following strengths of their study;

- Large size;
- Good quality data;
- 97.5% vital status of the veteran cohort ascertained;
- Relatively homogeneous population.

645. The authors discussed the following weaknesses of their study:

- Potential mismatches between individuals enrolment details in the armed services and civilian life.
- Allocation of a cancer diagnosis by matching name and other details from separate databases

646. The authors discussed:

> Recently, the National Academy of Science published a review reclassifying chronic lymphoid leukaemia from the category of inadequate or insufficient evidence of association to the category to sufficient evidence of association with dioxin exposure. [593]

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589 Page 84 table 6.3.

590 Page 88 table 6.6.

591 Page 91 table 6.8.

592 Page 95 table 6.11.

593 Page 115, IOM 2003, 'Veterans and Agent Orange, Update 2002', Washington, CD: The National Academies Press. Paper reviewed in these Reasons –See from [617].

> The present study finds that the overall rate of lymphoid leukaemia was elevated but this was a consequence of the elevation in the incidence of chronic lymphoid leukaemia. [594]

Council's comments

647. The Council considered this study important for it's incident data, noting the authors acknowledge it was done by matching name and other details. However the study lacks herbicide, pesticide or dioxin exposure data.

648. The Council considered important the Navy (SIR = 1.51) and Army (SIR = 1.68) data, which showed for Navy 12 cases of CLL out of 1,073 cancers diagnosed among 12,935 Navy Vietnam veterans; and for Army 42 cases of CLL out of 3,013 cancers diagnosed among 39,517 Army Vietnam veterans. In the Council's view this data is supportive of the relevant association noting that without accurate exposure or dose data, an association is plausible despite some uncertainty that the elevations of risk for B-cell CLL can be attributed to Herbicides / Pesticides / Dioxins exposure.

649. Nevertheless, the Council was persuaded that the demonstrated increased incidence of B-cell CLL in servicemen who went to Vietnam indicates a relevant association.

650. The Council considered that this study:

- points to a relevant association.

Wilson, EJ et al. 2005b, *Australian National Service Vietnam Veterans Mortality and Cancer Incidence Study*, Department of Veterans' Affairs, Canberra. RMA ID 41295 FILEForce ID 19113

651. This retrospective cohort study of Australian National Service personnel who served in Vietnam between 1966 and July 1973, examined all deaths during the period from completion of Vietnam service to 31 December 2001 and all cancers diagnosed from 1982 to 31 December 2000.

652. The cohort comprised 43,969 national servicemen; 19,240 Vietnam veterans and 24,729 non-veterans.

653. Relative mortality and cancer incidence rates were calculated for National Service men who served in Vietnam compared to those National Service personnel who served only in Australia during the Vietnam era. Standardised ratios comparing the National Service cohort to the Australian population were also calculated.

654. The authors provided the following:

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[594] Page 115.

relative risk ratio for death between National Service veterans and non veterans for neoplasms: [595]

Death (neoplasms) (290)	RR = 1.16	95% CI 0.98 - 1.36

relative risk ratio for cancer incidence (diagnosis) (1982-2000) between National Service veterans (8 cases) and non-veterans (11 cases) for CLL: [596]

CLL	RR = 0.90	95% CI 0.31 - 2.45

relative risk ratio for death for lymphoid leukaemia death (1996-2001) between National Service veterans (2 cases) and non-veterans (6 cases): [597]

Lymphoid Leukaemia	RR = 0.42	95% CI 0.04 - 2.35

and SIR for CLL for national servicemen examined 1982-2000: [598]

CLL (19 cases)	SIR = 1.24	95% CI 0.75 - 1.94

SIR for CLL for national servicemen, veterans: [599]

CLL (8 cases)	SIR = 1.18	95% CI 0.52 - 2.33

SIR for CLL for national servicemen, non-veterans examined 1982-2000: [600]

CLL (11 cases)	SIR = 1.30	95% CI 0.65 - 2.32

SMR for lymphoid leukaemia for national servicemen: [601]

Lymphoid Leukaemia (8 cases)	SIR = 0.82	95% CI 0.36 - 1.60

SMR for lymphoid leukaemia for national servicemen, veterans: [602]

Lymphoid Leukaemia (2 cases)	SIR = 0.47	95% CI 0.06 - 1.66

SMR for lymphoid leukaemia for national servicemen, non-veterans: [603]

Lymphoid Leukaemia (6 cases)	SIR = 1.09	95% CI 0.40 - 2.37

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595 Page 33 Table 3-2.

596 Page 37 Table 3-3.

597 Page 39 Table 3-4.

598 Page 141 Table C.4.

599 Page 142 Table C.5.

600 Page 143 Table C.6.

601 Page 144 Table C.7.

602 Page 145 Table C.8.

603 Page 146 Table C.9.

655. The authors concluded: [604]

> This study detected 19 cases of chronic lymphoid leukaemia diagnosed amongst National Servicemen between 1982 and 2000. Compared to the general population, the incidence was elevated for both the veteran and non-veteran groups, but the confidence intervals were wide and the results were not statistically significant. Comparing the two National Service groups showed no difference in incidence.

656. The authors refer to two other DVA reports showing an:

> Elevated SIR for CLL amongst all veterans and the Army veterans and a higher trend for Navy veterans; [605]
>
> A trend towards higher mortality from lymphoid leukaemia. [606]

657. The authors discussed the following limitations of the study:

- No information regarding exposure or dose to herbicides or pesticides or life habits such as cigarette smoking and alcohol consumption is available.
- The cohort were relatively young at the completion of the study.
- Inaccuracy due to loss to follow up.

Council's comments

658. The Council considered this study important because of the use of such a good control population of similar health and age characteristics [ie servicemen who didn't go to Vietnam], and the use of incidence data. The Council notes the authors found a non-significant trend towards increased CLL (19) cases.

659. As with Wilson, EJ et al. 2005a, in the Council's view this data may be supportive of a relevant association. It is plausible, but without accurate exposure or dose data, and there is uncertainty that the elevations of risk for B-cell CLL can be attributed to Herbicides / Pesticides / Dioxins exposure.

660. The Council considered that this study:

- did not point to but merely left open the possibility of a relevant association.

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604 Page 55.

605 Wilson, EJ et al. 2005, *Cancer Incidence in Australian Vietnam Veterans study*, Department of Veterans' Affairs, Canberra. Paper reviewed in these Reasons – See from [641]

606 Wilson, EJ et al. 2005, *The Third Australian Vietnam Veterans Mortality Study*, Department of Veterans' Affairs, Canberra. Study considered non-contributory in these Reasons – (See list of non contributory articles)

IARC 1997, International Agency for Research of Cancer, 1997, Polychlorinated dibenzo-*para*-dioxins and polychlorinated dibenzofurans. IARC monographs on the evaluation of carcinogenic risks to humans. Vol. 69. Lyon, France RMA ID 30598 FILEForce ID 19121

661. This monograph details the evidence used by the IARC in assigning the status of Group 1 carcinogen to 2,3,7,8-tetrachloridibenzo-p-dioxin (TCDD).

662. The authors reviewed the available evidence for carcinogenicity of TCDD according to the following:

- Exposure data;
- Human carcinogenicity data;
- Animal carcinogenicity data; and
- Other relevant data.

663. The authors summarized the following information on exposure data: [607]

> Mean background levels of 2,3,4,8-TCDD in human tissues today are in the range of 2-3 ng/kg fat. Available data suggest that these levels have decreased by a factor of 3-5 since the late 1970s… Human exposures related to occupation or accidents have led to tissue levels of 2,3,4,5-TCDD up to several orders of magnitude higher than background levels.

664. The authors referred to studies with direct 2,3,4,8-TCDD measurements and to studies involving heavy exposure to herbicides as the most compelling information with regard to human carcinogenicity data in particular:

a. Fingerhut et al 1991 [608]

Total lymphatic & hematopoietic tissue (ICD-9 200 - 208) – 24 deaths observed	SMR = 109	95% CI 70 - 162
NHL (ICD-9 200, 202) – 10 deaths observed	SMR = 137	95% CI 66 - 254
Lymphosarcoma & reticulosarcoma (ICD-9 200) – 5 deaths observed	SMR = 142	95% CI 46 - 332
Other lymphatic (ICD-9 202) – 5 deaths observed	SMR = 133	95% CI 43 - 313
Leukemia and aleukemia	SMR = 67	95% CI 24 – 146

607 Page 3.

608 Fingerhut, MA et al 1991, 'Cancer Mortality in Workers Exposed to 2,3,4,8-Tetrachlordibenzo-p-Dioxin', *NEJM*, vol. 324, no. 4, pp 212-218. (See list of non contributory articles)

(ICD-9 204-208) – 6 deaths observed

b. Hooiveld et al 1996 [609]

leukaemia (ICD-9 codes 204-208) there was one death

	SMR = 1.0	95% CI 0.0 - 5.7

non-Hodgkin's lymphoma (ICD-9 codes 200, 202) from 3 deaths

	SMR = 3.8	95% CI 0.8 – 11.0

c. Ott and Zober 1996 [610]

1953 to 1992

2 lymphatic / haematopoietic deaths	SMR = 1.2	95% CI 0.2 – 4.5

One of deaths occurred in the lowest TCDD dose group and one in the highest TCDD dose group

d. Morris Brown, L et al. 1990[611]

Leukaemia (340 cases living / 238 deceased)

Use of one or more herbicides	RR = 1.2	95% CI 0.9 – 1.6
Use of Phenoxy herbicides	RR = 1.2	95% CI 0.9 – 1.6
Exposure to 2,4,5-T (20 yrs prior)	RR = 1.8	95% CI 0.8 – 4.0

Mixed, handled or applied 2,4,5-T

Acute non-lymphocytic leukaemia	RR = 2.1	95% CI 0.9 – 4.9
CLL	RR = 1.6	95% CI 0.7 – 3.4

Mixed, handled or applied 2,4,5-T (20 yrs prior)

CLL	3.3	95% CI 1.2 – 8.9

e. Flesch-janys et al 1995 [612]

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609 Hooiveld, M et al. 1996, 'Preliminary results of the second follow-up of a Dutch cohort of workers occupationally exposed to phenoxy herbicides, chlophenols and contaminants.' *Organohalogen Compounds* 30: 185-189. (See list of non contributory articles)

610 Ott MF, Zober A. 1996 'Cause specific mortality and cancer incidence among employees exposed to 2,3,7,8-TCDD after a 1953 reactor accident.' *Occup Environm* Med 53: 606-612. (See list of non contributory articles)

611 IARC 1997, at Table 37 p. 198.

612 Fleshc-Janys et al 1995 'Exposure to polychlorinated dioxins and furans and mortality in a cohort of workers form a herbicide-producing plat in Hamburg, Federal Republic of Germany. *Am J Epidemiol* 142: 1165-1176.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

665. The authors reviewed the available animal experimental data and concluded: [613]

> In summary, 2,3,7,8-TCDD administered at low doses by different routes to rats and mice causes tumors at multiple sites. It also causes tumours in hamsters.

666. The authors reviewed that available information with regard to the following other relevant data:

- Kinetics;
- Toxic effects;
- Effects on reproduction;
- Genetic effects;
- Mechanistic considerations;
- Ah receptor;
- Gene expression; and
- Comparison of tissue concentrations in humans and animals.

667. The authors concluded: [614]

> Overall, the strongest evidence for the carcinogenicity of 2,33,7,8-TCDD is for all cancers combined, rather than for any specific site… This lack of precedent for a multi-site carcinogen without particular sites predominating means that the epidemiological findings must be treated with caution; on the other hand, the lack of precedent cannot preclude the possibility that in fact 2,3,7,8-TCDD, at high doses does act as a multi-site carcinogen. It should be borne in mind that the general population is exposed to levels far lower than those experienced by the industrial populations.

668. The authors summarized the overall evaluation: [615]

> '2,3,7,8-Tetrachlorodibenzo-para-dioxin is carcinogenic to humans (Group 1);

However the same population was reviewed in Becher, H et al. 1996, 'Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins', *Cancer causes and control*, vol. 7, pp. 312-321;

All Haematopoietic (13 cases)	RR = 1.7	95% CI 0.9 – 2.9
NHL (6 cases)	RR = 3.3	95% CI 1.2 – 7.1

(See list of non contributory articles)

613 Page 5.

614 Page 4.

615 Page 8 – 9.

In making the overall evaluation, the working group took into consideration the following supporting evidence:

> 2,3,7,8-TCDD is a multi-site carcinogen in experimental animals that has been shown by several lines of evidence to act through a mechanism involving the Ah receptor;
>
> …this receptor is highly conserved in an evolutional sense and functions the same way in humans as in experimental animals; and
>
> …tissue concentrations are similar both in heavily exposed human populations in which an increased overall cancer risk was observed and in rats exposed to carcinogenic dosage regimens in bioassays.

669. The authors final evaluation: [616]

> There is *limited evidence* in humans for the carcinogenicity of 2,3,7,8-tetrachloro-dibenzo-*para*-dioxin.
>
> There is *sufficient evidence* in experimental animals for the carcinogenicity of 2,3,7,8-tetrachloro-dibenzo-*para*-dioxin.
>
> There is *evidence suggesting lack of carcinogenicity* in experimental animals for dibenzo-*para*-dioxin.

Council's comments

670. The Council considered this study an authoritative review that reached the conclusion that dioxin is associated with cancer, with some biological plausibility mechanisms reviewed. Council does not find the IARC conclusions controversial, but notes that they have been questioned (see Cole, P et al. 2003, below).

671. The Council considered that this study:

- did not point to but merely left open the possibility of a relevant association.

Cole, P et al. 2003, 'Dioxin and cancer: a critical review', Regulatory Toxicology Pharmacology, vol. 38, no. 3, pp. 378-388. RMA ID 29796, FILEForce ID 19330

672. This review article argues against the decision to designate 2,3,7,8-tetrachloridibenzo-p-dioxin (TCDD) as a Group 1 carcinogen by the International Agency for Research on Cancer (IARC) in its 1997 Monograph 69. [617]

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[616] Page 342.

This methodology does not reflect the Council's two-step process in applying the reasonable hypothesis test as set down by the full Federal Court (see [108] - [109]).

673. The authors review the evidence relied on by IARC to reach its decision regarding the status of TCDD as a Group 1 carcinogen to opine the decision unsupportable because it depends on an analogy of mechanisms of carcinogenicity between animals and humans and on evaluation of the epidemiologic evidence the authors consider to be inappropriate. Part II of the article reviews subsequent epidemiologic reports and Part III addresses the US Environmental Protection Agency's ongoing risk assessment.

674. The authors challenged the conclusions regarding the Ah receptor with regard to animal studies and the classification of other polychlorinated dibenzo-para-dioxins as group 3, despite the assumption that all these compounds act in a similar way. They concluded:

> In the presence of these uncertainties about mechanisms, the evolutional conservation of Ah cannot be seen as decisive.

675. The authors challenged the conclusion drawn from the animal studies providing an alternate interpretation of the data which did not support the statement regarding similar tissue concentrations between animals and highly exposed human populations.

676. The authors provided a critical evaluation of the epidemiological data used by the IARC the concluded: [618]

> The epidemiologic evidence concerning TCDD based on the pre-1988 literature and presented in the IARC Monograph is most compatible with inadequate evidence of carcinogenicity in humans and not with limited evidence of carcinogenicity. This is so because the category, limited, requires evidence of positive associations, and these are not seen with any degree of consistency for TCDD. Importantly, there is little evidence of any positive associations in the most heavily exposed industrial or accident cohorts.

677. The authors provided a review of epidemiologic studies after 1997 including:

a. Fingerhut et al 1991 [619] - extended review of cohorts by 6 years in Steenland et al. 1999;

NHL (ICD-9 200, 202) – 10 deaths observed	SMR = 137	95% CI 66 - 254
Leukemia and aleukemia (ICD-9 204-208) – 6 deaths observed	SMR = 67	95% CI 24 – 146

617 International Agency for Research of Cancer, 1997, Polychlorinated dibenzo-*para*-dioxins and polychlorinated dibenzofurans. IARC monographs on the evaluation of carcinogenic risks to humans. vol. 69. Lyon, France. – See from [661]

618 Page 383.

619 Fingerhut, MA et al 1991, 'Cancer Mortality in Workers Exposed to 2,3,4,8-Tetrachlordibenzo-p-Dioxin', NEJM, vol. 324, no. 4, pp 212-218. – (See list of non contributory articles)

b. Steenland et al 1999; [620]

NHL (12 deaths)	SMR = 1.10	95% CI 0.56 - 7.91,
Leukemia and aleukemia (10 deaths)	SMR = 0.81	95% CI 0.38 - 1.48

Cole, P et al. 2003 state that Steenland et al 1999, provide only aggregated results for the whole follow-up (Fingerhut and Steenland data) which:

> …retain significance solely on the basis of the earlier data. The important negative findings of the extended follow-up are not mentioned… [621]

c. Dalager and Kang 1997; [622]

All lymphopoietic cancers:		
Vietnam (4 cases)	SMR = 0.83	95% CI 0.22 - 2.12
Non Vietnam (1 case)	SMR = 0.28	95% CI 0.00 - 1.55
Leukemia:		
Vietnam (2 cases)	SMR = 1.04	95% CI 0.12 - 3.76
Non Vietnam (1 case)	SMR = 0.68	95% CI 0.01 - 3.77

d. Mahan et al 1997; [623]

Cole, P et al. 2003 make no comment in relation to Lymphopoietic cancers or leukaemia specifically, but relate elevations in mortality risk for all causes of diseases of the digestive system and cancers to excess for liver cirrhosis and associated conditions possibly explained by hepatitis-B and C infections.

e. Hooiveld et al 1998; [624]

All workers		
Cancer (20 deaths)	SMR = 1.7	95%CI 1.1 – 2.7
NHL (1 death)	SMR = 3.9	95% CI 0.1 – 21.8
Workers exposed in the accident		

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620 Steenland et al 1999 'Cancer, heart disease and diabetes in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *J Natl. Cancer Inst*. 91 (9) 779-786. See from [501]

621 Cole, P et al. 2003 at Page 384.

622 Dalager, NA & Kang, HK 1997, 'Mortality among army chemical corps Vietnam Veterans', American Journal of Industrial Medicine, vol. 31, pp719-726. This 1997 article was considered by the Council as non-contributory.

623 Mahan, CM et al. 1997, A case control study of lung cancer among Vietnam veterans *J Occup. Environ*. Med 39 (8), 740-747.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

624 Hooiveld et al 1998. Second follow-up of a Dutch cohort occupationally exposed to phenoxy herbicides, chlorphenosl and contaminants. Am J Epidemiol. 147 (9), 891-901. (See list of non contributory articles)

leukaemia (ICD-9 codes 204-208) - 1 death

SMR = 1.0	95% CI of 0.0 - 5.7

non-Hodgkin's lymphoma (ICD-9 codes 200, 202) - 3 deaths

SMR = 3.8	95% CI 0.8 – 11.0

Cole, P et al. 2003 state that while this is a positive study for exposure to TCDD its inclusion in an expanded and updated cohort investigation in Kogevinas et al. 1997 means that the Hooiveld data no longer provides independent information.

f. Kogevinas et al 1997; [625] - See the data in footnote 625 below.

g. Hoppin et al 1998; [626]

Cole, P et al. 2003 state that this study provides data on chlorophenols, rather than TCDD or TCDD contaminated phenoxyherbicides.

h. Pesatori et al 1998; [627]

Cole, P et al. 2003 state that: [628]

> …the overall relative mortality from causes of death other than cancer in the Seveso population was no more than 1.0 in both genders in all three contaminated zones.
>
> This provides evidence against the carcinogenity…of even relatively high exposures of TCDD.

i. Ketchum et al 1999. [629]

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625 Kogevinas et al 1997 'Cancer mortality in workers exposed to phenoxy herbicides, chlorophenos and dioxins. An expanded and updated international cohort study. *Am J Epidemiol* 145 (12), 1061-1075

IARC Retrospective cohort study of 21,863 male and female workers in 36 cohort and 12 countries exposed to phenoxy herbicides, chlorophenols and dioxins.

Results for any exposure:

NHL	SMR = 1.27	95%CI 0.88 - 1.78,
Leukaemia	SMR = 1.00	95%CI 0.69 - 1.39

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

626 Hoppin et al 1998 Occupational chlorophenol exposure and soft tissue sarcoma risk among med aged 30-60 years. *Am J Epidemiol* 148 (7), 693-703.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

627 Pesatori et al 1998 Dioxin exposure and non-malignant health effects: a mortality study. Occup Environ Med 55(2), 126-131.

This article was not available to (before) the RMA at the relevant times, and so could only be considered by the Council as new information.

628 Cole, P et al. 2003 Page 385.

629 Ketchum et al 1999. Serum dioxin and cancer in veterans of Operation Ranch Hand Am J Epidemiol. 149 (7), 630-639. (See list of non contributory articles)

Cole, P et al. 2003 states [630]:

> Found fewer cancer deaths than expected (OR = 0.7 95% CI = 0.3 – 1.4) among those in the high exposure category.

678. From the review of post 1997 epidemiologic studies the authors concluded: [631]

> The epidemiologic studies published in 1997 or later further weaken the poor evidence concerning the alleged human carcinogenicity of TCDD, and when combined with the pre-1997 literature, reinforce the view that the evidence for human carcinogenicity of TCDD is inadequate at most.

679. The Authors provided the final conclusion: [632]

> It is clear from this review that the evidence does not support the IARC's classification of TCDD as a Group I carcinogen. In fact, the evidence indicates that TCDD is not carcinogenic to human beings at low levels and that it may not be carcinogenic to them even at high levels.

680. The authors did not review any studies considering the association between Dioxins (TCDD) and CLL.

Council's comments

681. The Council considered the authors' critical review provides a case for an IARC classification for TCDD of inadequate, as opposed to limited, evidence for carcinogenity. In the Council's view, however, the studies by Kogevinas, M et al 1997, Hooiveld, M et al. 1996, and to a lesser extent Steenland, K et al. 1999 and Fingerhut, MA et al. 1991, used by the authors in support of that case, do provide some support for carcinogenity, at least for TCDD and possibly for herbicides. The Council agreed with Cole et al that Ketchum, NS et al. 1999 is a very negative study for any relevant associations.

682. In the Council's view therefore, the strength of the conclusions that can be drawn from Cole's conclusion on carcinogenity and the data reviewed in this study, which was in relation to lymphopoietic cancers, leukaemia, and NHL and not specifically for B-cell CLL, is diminished.

683. The Council considered that this study:

- did not point to but merely left open the possibility of a relevant association.

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630 Cole, P et al. 2003 Page 385.

631 Cole, P et al. 2003 Page 385.

632 Cole, P et al. 2003 Page 386.

COUNCIL CONCLUSIONS REGARDING HERBICIDES/PESTICIDES & DIOXIN

684. Council noted the Commissions' submission that in Australia, Vietnam veterans are covered for CLL by a Commissions' determination under section 180A of the VEA, which would be reviewed after the Council decided whether herbicides, and or pesticides and or dioxins were supported by the information in this review. CLL is the only leukaemia for which the RMA Statements of Principle differ from the DVA in the US by not including a factor for herbicides, and or pesticides and or dioxins.

685. As a threshold proposition, for exposure to herbicides, pesticides and dioxin, the Council noted that it was very difficult to ascertain from the studies which touched on such exposures the precise nature of the exposure, ie., whether it was dioxins, herbicides, or pesticides, or a combination. The Commissions submitted that of the relevant studies, only Morris-Brown et al 1990 study showed a weakly positive association and the Wilson et al 2005a (Cancer Incidence in Australian Vietnam Veterans study) showed an excess of CLL from being in Vietnam. The Commissions preferred not to offer a firm view on whether a dioxin or herbicide factor should be included.[633]

686. The Council considered the studies provide mixed results with regard to association between B-cell CLL and herbicides, pesticides & dioxins. Most studies reviewed did not provide data for B-cell CLL alone with the consequence that the Council applied their collective expertise to interpret data on leukaemia and NHL.

687. The Council noted that most of the data support an association between pesticide exposure and NHL, but the studies use superseded NHL classifications.

688. The Council considered that there was a trend to association in the Australian Vietnam Veteran studies, ie a non-significant increased number of CLL cases over expected; but the Australian Veteran papers provide no specific data about herbicide, pesticide and or dioxin exposures.

689. The Council considered that the combined effect of the studies by:

Morris Brown, L et al. 1990;

IOM Update 2006;

Wilson, EJ et al. 2005 a (Cancer Incidence in Australian Vietnam Veterans); and

Hertzman, C et al. 1997; and

633 Written Submission page 28 of 39.

that pointed to (as opposed to merely left open) a relevant association, and in particular:

Waterhouse, D et al. 1996;

Amadori, D et al. 1995;

Lee, WJ et al. 2004

Mills, PK et al. 2005

that did not point to but merely left open the possibility of a relevant association, and provides evidence in support of a positive trend for the relevant association;

taken collectively is a body of sound medical-scientific evidence that was available to the RMA that justifies amending the reasonable hypothesis Statement of Principle to include herbicides, pesticides and dioxin as a factor.

690. Further, the Council considered that the combined effect of the studies by:

Kogevinas, M et al. 1997;

Steenland, K et al. 1999;

Hansen, ES et al. 1992;

Blair, AD & White, A 1985;

Burmeister, LF et al. 1982;

Read, D et al. 2007;

IOM 2003 Update 2002;

Wilson et al. 2005b (Australian National Service Vietnam Veterans Mortality and Cancer Incidence);

IARC 1997;

Cole, P et al. 2003;

did not point to but merely left open the possibility of a relevant association. These studies were in addition to those studies that similarly did not point to but merely left open the possibility of a relevant association, but they showed a positive trend for the relevant association listed at [689].

691. The Council considered other articles that in its view were sound medical scientific evidence that touched on the potential herbicides, pesticides, dioxin factor(s), that were in Council's final analysis non contributory[634]:

Ott, MF & Zober, A 1996;

Visitainer, PF et al. 1995;

Becher, H et al. 1996;

Tatham, L et al. 1997;

Dalager, NA & Kang, HK 1997;

Alavanja, MCR et al. 2005;

McLean, D et al. 2006;

Torchio, P et al. 1994;

Young, AL 2004

Wilson 2005 (The Third Australian Vietnam Veterans Mortality)

Australian Institute of Health and Welfare (AIHW) 2000;

Hooiveld, M et al. 1998

Bertazzi, PA et al. 2001;

Fingerhut, MA et al. 1991;

Blair, A et al. 2004;

Blair, A et al. 2005;

t'Mannetje, A et al. 2005;

IOM 1994 (1st in series);

IOM 2005, Update 2004;

Ketchum, NS et al. 1999;

Crane, PJ et al. 1997

692. The Council did not focus in these Reasons on those articles as they:

634 Articles that did not, in Council's final analysis, contribute to either advance or weaken the state of evidence of the possibility of a relevant association (i.e, were non contributory).

- did not provide data specific to CLL;
- provided data on haematopoietic cancers, leukaemia and lymphoma, and or Non-Hodgkin's lymphoma that the Council could not draw conclusions from concerning B-cell CLL; and/or
- were in respect of such small number of cases of CLL, haematopoietic cancers, leukaemia and or lymphoma that the Council could not draw conclusions from concerning B-cell BCLL.

693. The Council having taken into account all the information, none of which indicated against the relevant association, but much of which, as described above, indicated or lent some support to a relevant association, was convinced that the reasonable hypothesis test had been met.

694. The Council's view was that the sound medical-scientific evidence did not support that it was more probable than not that herbicides, pesticides and dioxin, separately or in combination a associated with B-cell CLL. That is, the Council was not satisfied by the sound medical-scientific evidence of a relevant association on the balance of probabilities.

Should There Be a Potable Water Factor?

Muller, J et al. 2002, *Examination of the Potential Exposure of Royal Australian Navy (RAN) Personnel to Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans via Drinking Water*, The National Research Centre for Environmental Toxicology (ENTOX)[NRCET], A Report to the Department of Veteran Affairs, Australia.

695. This study investigated the potential for exposure of sailors on Australian Navy ships and Army small ships to contaminants via potable [635] water. Laboratory experiments were carried out to assess whether PCDD/Fs [636] and DMA [637] can co-distil in significant quantities in the distillation units of ships. The study also evaluated the potential exposure level and pathways.

696. The study resulted from observations in studies undertaken by the Crane et al. 1997 (See the list of non contributory articles at p. XXX) for the Australian Government that found the highest elevation in mortality was among veterans of the Royal Australian Navy, rather than the land forces and air forces. Uncertainty remains as to

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635 Potable water is defined in SoPs 28 of 2005 as "means water used for drinking water, food preparation and beverage production;" At p. 4 of 5 of instrument No. 28 of 2005.

636 Agent Orange herbicide (n-butyl ester of 2,4-D and issocytl ester of 2,4,5,-T)…was contaminated with up to ~ 45 ppm of 2,3,7,8-TCDD and traces of 1,2,3,7,8-PeCDD (Young et al 1978 quoted in IOM, 1999). At p. 11. A list of the chemicals and blank used in the Muller et al 2002 experiments is provided in Table 1 of that study (at p. 15).

637 Agent Blue herbicide was the third most commonly used herbicide in Vietnam and consisted of a aqueous solution of Dimethylarsenic acid (DMA). At p. 9.

whether this increase in mortality is related to the use of “Agent Orange” contaminated with polychlorinated dibenzodioxins and dibenzofurans (PCDD/Fs).

697. They noted the water consumed by the crew of supply ships and other vessels which regularly visited the conflict areas in Vietnam has an unusual history. It was often collected from near-shore marine waters that received run-off from areas which had been sprayed with Agent Orange and Agent Blue. To make this water suitable for drinking and other purposes aboard ship the water was distilled aboard.

698. In general, sea water was fed into an evaporator where the water was boiled by a combination of heating and reduced pressure (vacuum) and the vapour was condensed in the condenser from which it was pumped into feed tanks (Figure 1).

699. The phase One findings of the study found these results clearly demonstrated that organochlorins and dioxins had the potential to co-distill relatively rapidly

700. Interesting in this first experiment 2,3,7,8-tetrachlorodibenzodioxin, the most toxic of all PCDD/Fs and the main contaminant in Agent Orange, was found at about 85% of the quantity observed in the non distilled samples and thus co-distilled the most.

701. Co-distillation of dioxins and organochlorines from water collected from the Brisbane River (water was added to known amount of chemicals of interest) demonstrated that the process is reproducible using estuarine water.

702. To evaluate the exposure of personnel on board ships the authors considered consumption of contaminated water from distilled water and other potential pathways. Due to the lack of information on water concentrations of PSDD/Fs in Vietnamese estuarine waters they attempted to calculate water concentrations from concentrations in fish from Vietnam waters taken in the 1970’s:

> …5 L of this water per day. The direct consumption of this water would lead to a daily body burden of about 0.4 – 7 ng/day. At p. 33.

703. For a 70 kg person the body burden would be in the range of 12 – 200 pg / kg bw per day for a 14 day period in addition to the background exposure through TCDD and other dioxin-like chemicals in the food and in the environment.

704. The phase two exposure level findings of the study found that dimethylarsenic acid does not co-distill at significant levels…and thus the drinking water was unlikely to be contaminated with dimethylarsenic acid.

705. No de-novo synthesis of TCDD or any other dioxins from the other compounds of Agent Orange was detected under the experimental conditions. However, the copper element on board ships was probably significantly hotter than in the simulation experiments…and thus these results should not be used as absolute evidence that such a formation did not occur in the distillation units of the RAN ships.

> …it is likely that solely the consumption of drinking water resulted in exposure levels that exceeded the recommended Total Monthly Intake (TMI) values for TCDD of 70 pg / kg bw / month significantly. At p. 7.

706. The authors concluded that Subsequent ingestion [potable water] by sailors on board ships (as well as soldiers and airmen, who were passengers) is thus a vector for exposure to these chemicals.

707. The effect of salinity on co-distillation could not be determined, but the studies indicated that suspended particles or a sorption compartment in the water reduced the tendency for co-distillation. However, enrichment of TCDD in the distilled water was still high at 1.4 g TSS L-1.

708. The authors noted the acceptable intake guidelines of dioxin-like chemicals – dioxins, furans, dioxin like PCBs as agreed by the World Health Organisation (WHO), European Commission Scientific Committee on Food (EC-SCF), and the joint Food and Agriculture Organisation of the United Nations FAO / WHO Expert Committee on Food Additives, compared to the study findings on daily body burdens above:

– Table 4 (At p. 37):

Standard	pg / kg bw / day	pg / kg bw / week	pg / kg bw / month
WHO consultation 1998	1-4	(7-28)	(30-1200)
EC-SCF 2001	(2)	14	(60)
FAO / WHO 2001	(2.3)	(16)	70

Exposure values in brackets were calculated for the other periods for easier comparison.

Council's comments

709. The Council considered this was a persuasive study because it provides a plausible mechanism for possibly clinically significant exposure to 2,4-D and related chemicals, via the ingestion of potable water.

710. Council notes the authors have experimentally demonstrated co-distillation of dioxins and organochlorins in the potable water distilled on ships.

711. It could explain the increased rates of increased rates of mortality seen in naval personnel compared to army or air force, if it were assumed that exposure to 2,4-D and other contaminants were the cause of that increased mortality.

712. Council moreover considers that this study negates the conclusion of a related study by Young et al. 2004.

713. The Council considers that this study alone is not sufficient for the Council to determine exposure to herbicides / pesticides / dioxin for B-cell CLL. That can only be considered as such if Dioxins and related chemicals are supported by the relevant information, subject to review, as a factor.

714. The Council considers this study:

- supports potable water as a mechanism for exposure to herbicides / pesticides / dioxin.

SUBMISSIONS REGARDING DIOXIN

715. The Council was conscious that the submissions made to it concerning the contended potable water factor included submissions concerning exposure to dioxins and that the Muller, J et al. 2002 paper sought to ascertain whether water from Vietnamese waters was likely to be contaminated, including by dioxin.

716. The Council was also conscious that the submissions made to it in relation to dioxin included submissions concerning the nature of any dioxin factor in the CLL Statements of Principles, referring to existing factors in other Statements of Principles for Hodgkin's lymphoma, non-Hodgkin's lymphoma and Myeloma, and contending that a similar factor be inserted into the CLL Statements of Principles.

717. Further, the contentions were specifically that the dioxin factor(s) in the non-Hodgkin's Statement of Principles No. 28 of 2010, including factors for being in Vietnam, and for ingesting potable water that was distilled from Vietnamese estuarine waters should be inserted into the Statements of Principles for CLL, on the basis that B-cell CLL and SLL are the same disease.

COUNCIL'S CONCLUSIONS – POTABLE WATER

718. The Council concluded that the sound medical-scientific evidence available to the RMA at the relevant times touching on the contended potable water factor and the sound medical-scientific evidence relevant to dioxin, herbicides and pesticides is sound medical-scientific evidence on which the RMA could have relied to amend the CLL Statements of Principles to include a potable water factor.

719. The Council considered that study by

Muller, J et al. 2002;

that supports potable water as a mechanism for exposure to dioxin; together with the combined effect of the studies by:

Morris Brown, L et al. 1990;

IOM Update 2006;

Wilson, EJ et al. 2005 a (Cancer incidence); and

Hertzman, C et al. 1997; and

that pointed to (as opposed to merely left open) a relevant association in respect of herbicides, pesticides and dioxin, and in particular:

Waterhouse, D et al. 1996;

Amadori, D et al. 1995;

Lee, WJ et al. 2004

Mills, PK et al. 2005

that did not point to but merely left open the possibility of a relevant association in respect of herbicides, pesticides and dioxin, but provide evidence in support of a positive trend for such an association;

taken collectively, is sound medical-scientific evidence that justifies amending the reasonable hypothesis Statement of Principle, to include a potable water factor, but does not meet the standard of being more probable than not.

720. The Council considered another article that in its view was sound medical scientific evidence that touched on the potential potable water factor, that was in Council's final analysis non contributory:[638]

Young, AL et al. 2004

721. The Council did not focus in these Reasons on that articles that was, in its view non contributory, as it did not provide data specific to CLL or other malignant diseases.

722. The Council therefore considered that the RMA, in characterising a factor for herbicides, pesticides and dioxin should include exposures as a result of potable water.

[638] Articles that did not, in Council's final analysis, contribute to either advance or weaken the state of evidence of the possibility of a relevant association (i.e, were non contributory).

EXPOSURE TO SOLVENTS

723. The Council note that there are a large number of possible solvents. The information that was available to (before) the RMA and sent by the RMA to the SMRC was related to:

a. specific agents such as benzene; or

b. defined epidemiological groups across different countries, such as farmers.

724. The Council therefore focussed on those agents for which there was information relating to specific exposures.

Should There Be An Exposure to Benzene Factor?

Australian Institute of Petroleum (AIP) 2001, *Lympho-haematopoietic Cancer and Exposure to Benzene in the Australian Petroleum Industry Technical Report and Appendices*, Canberra, (accessed at http://www.aip.com.au/pef/case_study.pdf) RMA ID 28266, FILEForce 18687

Glass, DC et al. 2003, 'Leukemia risk associated with low-level benzene exposure', Epidemiology, vol. 14, no. 5, pp. 569-577.

725. This report on the Australian Petroleum Industry and lympho-haematopoietic (LH) cancer, was commissioned due to an apparent excess of LH cancer detected by Health Watch (prospective study of all causes of death and cancer incidence in Australian petroleum industry, started 1980). Total cohort size was 16000 male workers and retirees.

726. This full report was published in 2001 as was an associated article[639] and had 221 pages. It was later published in a shorter format in 2003 [640].

727. An apparent excess number of deaths from leukaemia (SMR 2.0, 95% CI 1.3-2.9) and myeloma (SMR 1.9, 95% CI 1.0-3.3) triggered the current nested case control study of LH cancers. The cancer cases were collected from self-report, and not only from death certificates.

728. There was a total of 79 cases of LH cancers (33 leukaemia, 31 NHL, 15 Myelomas). Each patient was matched to five controls of same age, chosen randomly from the cohort. Individual benzene exposure was estimated from company records and

639 Glass, DC et al. 2001 'Validation of exposure estimation for benzene in the Australian petroleum industry', *Toxicology and Industrial Health* 2001 17(4) pp 113.

640 Glass, DC et al. 2003, 'Leukemia Risk Associated With Low-Level Benzene Exposure', *Epidemiology*, vol. 14, no. 5, pp. 569-77. An abstract of this article was available to (before) the RMA and therefore the Council; RMA ID 19489 FILEForce 030508.

interviews. Exposure levels[641] were low in comparison to older studies (mean 0.2ppm, range 0.001 - 2.07ppm, cumulative mean 4.9ppm-years, range 0.005 - 57.3).

729. Leukaemia risk was increased at relatively low exposure levels (2 ppm-years cumulative, or highest exposure >0.8 ppm). The highest risk category (>8 ppm-years) had the highest odds ratio (11.3, 95% CI 2.9 – 45.1) [642].

730. With respect to CLL, 11 cases were present in the cohort. When exposure was divided into quintiles of cumulative exposures, there was a positive association between CLL and the highest quintile (a total of 5 cases, OR 5.78, 95% CI 1.26 – 23.02) [643]. Logistic regression analysis of cumulative exposure demonstrated odds ratios of 1.0 (reference category) for < 4ppm-years, 2.76 for 4 - 8 ppm-years, and 4.52 for >8 ppm-years. However, the 95% CI for all the categories in the logistic regression crossed unity.

Table 42: Leukaemia Type Odds Ratios by Quintile (Quintiles 1-3 amalgamated)

	Controls	Cases	Case: Control	OR[#]	OR*[26]	95% Conf. Interval	
CLL							
1-3	37	4	0.11	1.00	1.00		
4	10	2	0.20	1.85	1.98	0.37	10.76
5	8	5	0.62	**5.78**	**5.39**	**1.26**	**23.02**

[#] Odds ratios are relative to Quintiles 1-3

Council's comments

731. The Council considered this was important study providing evidence of a possible association, based however on 11 cases only (See [730]) The data for the 5th quintile based on 5 cases was statistically significant and the data overall showed a trend with a dose response.

732. The Council considered that this study,:

- points to a relevant association.

Wong, O & 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. – RMA ID 7345, FILEForce 18750

641 The Council noted that the National Institute for Occupational Safety and Health (NIOSH) exposure limit for benzene is 0.1 PPM and the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit is 0.5 PPM. The OSHA (US legal) limit is 1 PPM.

642 See pp. 74-75.

643 See Table 43 at pp. 75.

733. This meta-analysis was based on combined databases of US and UK petroleum workers, in order to determine risks of leukaemia by specific subtypes. Cohort size was 208,741 workers over a 63 year period (1937 – 1989). This population is nearly 200 times larger than the original pliofilm cohort [644], and more than 10 times the size of the Australian "Health Watch" cohort [645].

734. There were 498 deaths attributable to leukaemia, 327 classifiable by subtype (148 AML, 34 ALL, 62 CML, 83 CLL). Compared with population data, there was no increased risk of death from any leukaemia subtype. This absence of association (even for AML) was attributed to the low level of benzene exposure in petroleum workers.

735. The authors highlighted that in the reports from individual cohorts used in this meta-analysis, there were conflicting information about the relationship between CLL and benzene, but the overall (meta-analysis) relationship was not significant:

736. In the US-UK combined cohort, 83 CLL deaths were observed, whereas 98.89 were expected; and the CLL meta-SMR was 0.84 (95% CI, 0.67-1.04). [646]

Council's comments

737. The Council considered this was a neutral study because of the low levels of exposure as evidenced by the lack of an association with AML, which was contrary to what the Council had expected the authors to find. The authors made a simple comparison of petroleum workers versus the general population using SMR, with no attempt to quantify the exposed dose.

738. The Council considered that this study:

- did not point to but merely left open the possibility of a relevant association.

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644 Pliofilm cohort. Council notes that there were no CLL deaths in the pliofilm cohort:

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.

Paxton, MB et al. 1994, 'Leukemia risk associated with benzene exposure in the pliofilm cohort: 1. Mortality update and exposure distribution', *Risk Analysis*, vol. 14, pp. 147-154.

Rinsky, RA et al. 1987, 'Benzene and Leukemia. An epidemiologic risk assessment', *NEJM*, vol. 316, no. 17, pp. 1044-1050.

645 Health Watch cohort – Australian Institute of Petroleum **(AIP) 2001**, *Lympho-haematopoietic Cancer and Exposure to Benzene in the Australian Petroleum Industry Technical Report and Appendices*, Canberra, (accessed at http://www.aip.com.au/pef/case_study.pdf)

646 See at p. 313 and Table 6 and Fig. 4, at p. 314.

COUNCIL'S CONCLUSIONS – BENZENE

739. The Council considered that the there was one positive relevant study as reported by:

Australian Institute of Petroleum (AIP) 2001

Glass, DC et al. 2003

that pointed to the relevant association.

740. The Council considered that the data from this study, while pointing to a relevant association, was based on a low number (11) of cases and on death certificates (SMR analysis) which are known not to be sensitive for identifying cases of CLL. Thus the Council was not satisfied by this study on the balance of probabilities.

741. The Council considered the much larger meta analysis of petroleum workers by Wong, O and Raabe, GK 1995 to be relevant but as no effect of benzene was shown as against 83 cases of CLL, the Council was not satisfied that it indicated a relevant association.

742. The Council also considered other articles that in its view were sound medical-scientific evidence that touched on the potential benzene factor, that were in the Council's final analysis non contributory:[647]

Hayes, RB et al. 1997 and 2000

Costantini, A et al. 2003

Glass, DC et al. 2000 and 2001 (methods papers – See Glass, DC et al 2003)

Rinsky, RA et al. 1987 and 2002

Crump, KS 1993

Paxton, MB et al. 1994

Ireland, B et al. 1997

743. The Council did not focus on those articles in these Reasons as they:

[647] Articles that did not, in Council's final analysis, contribute to either advance or weaken the state of evidence of the possibility of a relevant association (i.e, were non contributory).

- did not provide data specific to CLL;

- provided data on haematopoietic cancers, leukaemia and lymphoma, and or Non-Hodgkin's lymphoma from which the Council could draw no conclusions concerning B-cell CLL; and/or

- were in respect of such small numbers of cases of CLL, haematopoietic cancers, leukaemia and or lymphoma that the Council could draw no conclusions concerning B-cell CLL.

744. The Council having considered all the information both individually and collectively, and conscious that the reasonable hypothesis test is one of possibility, and an unusually light burden, was convinced that the reasonable hypothesis test had been met.

745. Being satisfied that the reasonable hypothesis test was met, the Council considered the balance of probabilities and was not so satisfied. Neither individually nor collectively does the sound medical-scientific evidence, in the Council's opinion, indicate the relevant association to the balance of probabilities level of satisfaction.

746. The Council therefore recommends that the RMA, in undertaking the investigation recommended by the Council (See at [1] - [4] above) considers whether there is a new body of sound medical-scientific evidence available that, together with the sound medical-scientific evidence already considered by the RMA, including information considered by the RMA concerning Chronic Lymphoid Leukaemia and Non Hodgkin's Lymphoma or any haematopoietic cancers, so far as being of potential relevance to Chronic Lymphocytic Leukaemia / Small Lymphocytic Lymphoma, justifies exposure to benzene as a factor.

New Information

747. The Council was aware of information that was not available to (before) the RMA at the relevant times, and therefore could not and did not take it into account in making its decision above.

748. The Council considered the following new information sound medical-scientific evidence, that the meta-analyses by Khalade, A et al. 2010 and Vlaanderen, J et al. 2011 in particular support a relationship between B-cell CLL and benzene exposure. The Council provides the citations below for the information of the RMA:

- Cocco, P et al. 2010, 'Occupational exposure to solvents and risk of lymphoma subtypes, results from the Epilymph case-control study', *Occup Environ Med*, vol. 67, no. 5, pp. 341–347.

- Boberg, E et al. 2011, 'The role of residence near hazardous waste sites containing benzene in the development of hematologic cancers in upstate New York', *Int J Occup Med Environ Health*, vol. 24, no. 4, pp. 327-38.

– Vlaanderen, J et al. 2011, 'Occupational benzene exposure and the risk of lymphoma subtypes: a meta-analysis of cohort studies incorporating three study quality dimensions', *Environ Health Perspect*, vol. 119, no. 2, pp. 159-67

– Khalade, A et al. 2010, 'Exposure to benzene at work and the risk of leukemia: a systematic review and meta-analysis', *Environ Health*, vol. 9, no. 31.

– Blair, A et al. 2007, 'Chemical exposures and risk of chronic lymphocytic leukaemia', *Br J Haematol*, vol. 139, no. 5, pp. 753-61.

Should There Be An Exposure To Other Aromatic Hydrocarbons – Toluene, Xylene, Carbon Tetrachloride and Others Factors?

COUNCIL'S CONCLUSIONS – OTHER AROMATIC HYDROCARBONS

749. The Council carefully considered the articles that in its view were sound medical-scientific evidence that touched on a relevant association with other hydrocarbons, and in particular:

National Occupational Health and Safety Commission 2004, *Exposure Standards: Xylene*

National Occupational Health and Safety Commission 2004, *Exposure Standards: Carbon Tetrachloride*

Integrated Risk Info System (EPA) (IRIS), *CARBON TETRACHLORIDE*

Gérin, M et al. n.d.

Linet, MS et al. 1987

Amadori, D et al. 1995

Svensson, B et al. 1990

750. The Council decided that these articles non-contributory. Each article either:

– did not provide data specific to CLL;

– provided data on haematopoietic cancers, leukaemia and lymphoma, and or Non-Hodgkin's lymphoma that the Council could not draw conclusions from concerning B-cell CLL;

– were in respect of such small number of cases of CLL, haematopoietic cancers, leukaemia and or lymphoma that the Council could not draw conclusions from concerning B-cell CLL;

and the Council therefore did not focus on them in these Reasons.

751. The Council decided that, overall, the sound medical-scientific evidence was insufficient to justify any amendment to the Statements of Principles to include other aromatic hydrocarbons as a factor, there being no information in respect of other aromatic hydrocarbons that specifically addressed CLL or from which the Council could draw relevant conclusions.

752. The Council therefore recommends that the RMA, in undertaking the investigation recommended by the Council (See at [1] - [4] above) considers whether there is a new body of sound medical-scientific evidence available that, together with the sound medical-scientific evidence already considered by the RMA, including information considered by the RMA concerning Chronic Lymphoid Leukaemia and Non Hodgkin's Lymphoma or any haematopoietic cancers, so far as being of potential relevance to Chronic Lymphocytic Leukaemia and Small Lymphocytic Lymphoma, justifies exposure to other hydrocarbons as a factor.

Should There Be An Exposure To Aircraft Fuel (AVGas) and associated chemicals including MEK & Sealants Involved In FIII Fuel Tank Repairs Factor?

COUNCIL'S CONCLUSIONS – AV GAS, MEK & SEALANTS (FIII)

753. The Council considered the articles that in its view were sound medical-scientific evidence that touched on the relevant association with aircraft fuel and associated chemicals and in particular,

> Hunter Medical Research Institute and University of Newcastle Research Associates 2003 (SHOAMP)

754. The Council did not focus on that article in these Reasons as, in the Council's view, the study provided almost no information specific to CLL (the Council noted only 1 leukaemia case occurred in the exposed group and the subtype of that leukaemia case was not stated, compared with 6 cases in each of the Amberley and Richmond)

755. The Council considered the sound medical-scientific evidence available to the RMA was insufficient to justify any amendment to the Statements of Principles in respect of AV Gas and associated chemicals in relation to aircraft fuel tank de-seal / reseal activities as there was no information that provided data sufficiently specific to B-cell CLL or from which the Council could draw any relevant conclusion.

756. The Council therefore recommends that the RMA, in undertaking the investigation recommended by the Council (See at [1] - [4] above) considers whether there is a new body of sound medical-scientific evidence available that, together with the sound medical-scientific evidence already considered by the RMA, including information considered by the RMA concerning Chronic Lymphoid Leukaemia and Non Hodgkin's Lymphoma or any haematopoietic cancers, so far as being of potential relevance to Chronic Lymphocytic Leukaemia / Small Lymphocytic Lymphoma, justifies exposure to AV Gas and associated chemicals in relation to aircraft fuel tank de-seal / reseal activities as a factor.

Should There Be An Exposure To Asbestos Factor?

Schwartz, DA et al. 1988, 'B cell neoplasms and occupational asbestos exposure', *American Journal of Industrial Medicine*, vol. 14, no. 6, pp. 661-671. RMA ID 16958, FILEForce ID 19001

757. This retrospective case controlled study developed a measure of occupational exposure to asbestos and evaluated the etiologic role of asbestos exposure in B cell neoplasms.

758. Data was obtained in a secondary retrospective analysis of a multicenter, population-based, case controlled study in which the primary role was the role of antigenic stimulation in the occurrence of CLL and myeloma.

759. Cases (CLL N = 429; myeloma N= 698) were identified from population based cancer registries in four geographic areas. Control (N = 1,683) populations were obtained from each of the four areas. [648]

760. The authors developed a classification system to assign asbestos exposure from direct in-person interviews using information from cases and controls work history. [649] From this index they calculated two measures of asbestos exposure:

- The peak asbestos exposure was the highest level an individual had experienced during his or her work career; and
- A summary measure of asbestos exposure weighted by intensity and years of exposure. [650]

761. The authors calculated relative risks adjusted for age, gender, race and study site and compared CLL cases and controls.

762. The authors findings were:

a. Among all study subjects, the adjusted risk of CLL showed a modest (but not statistically significant) tendency to increase with increasing peak asbestos exposure (p Value 0.13): [651]

low peak asbestos exposure	RR = 1.1	95% CI 0.8 - 1.6
medium asbestos exposure	RR = 1.2	95% CI 0.8 - 1.8
high peak asbestos exposure	RR = 1.4	95% CI 0.8 - 2.3

b. A similar trend was observed in the adjusted risk for CLL with an increasing level of weighted asbestos exposure (p Value 0.09): [652]

low peak asbestos exposure	RR = 1.0	95% CI 0.7 - 1.5
medium asbestos exposure	RR = 1.4	95% CI 0.9 - 1 22
high peak asbestos exposure	RR = 1.3	95% CI 0.9 - 1.9

although the adjusted RR for those with high exposure was slightly less than those with medium asbestos exposures. [653]

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[648] See Table II at p. 665 for a summary comparison of study and control study subjects.

[649] See Table I at p. 664 for a summary of the asbestos exposure index.

[650] See at p. 664.

[651] See Table III at p. 666.

[652] See Table IV at p. 666.

c. Among white subjects, the adjusted risk of CLL with an increasing level of peak or weighted asbestos exposure was stronger than for persons of other races:

Peak asbestos exposure (p Value 0.05): [654]

low peak asbestos exposure	RR = 1.3	95% CI 0.9 - 1.9
medium asbestos exposure	RR = 1.2	95% CI 0.8 - 1 9
high peak asbestos exposure	RR = 1.7	95% CI 1.0 - 3.0

weighted asbestos exposure (p Value 0.03): [655]

low peak asbestos exposure	RR = 1.1	95% CI 0.7 - 1.6
medium asbestos exposure	RR = 1.7	95% CI 1.1 – 2.8
high peak asbestos exposure	RR = 1.4	95% CI 0.9 – 2.1

763. In the entire study population and also among white study subjects, the highest relative risk for either measure of asbestos exposure was observed in those individuals who were 10-19 years from first exposure. [656]

764. The authors considered the strengths of their study were:

a. the range of geographical regions studied;

b. the requirement for newly diagnosed cases of CLL; and

c. consideration for both the size and length of time of exposure to asbestos.

765. The authors considered the limitation of their study was the reliance on the subjects' occupational history as a measure of exposure to asbestos.

766. In summary the authors stated:

> Our findings are at least suggestive that an association may exist between asbestos exposure and developing CLL. [657]

Council's comments

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653 See Table IV at p. 666.

654 See Table III at p. 666.

655 See Table IV at p. 666.

656 See Table V at p. 667.

657 See at p. 669.

767. The Council noted the authors' methodological comments on the consideration of exposure size and length of time of exposure, with work history as a surrogate for exposure as above. The Council considered this study shows a weak positive trend for peak and weighted asbestos exposure, which was significant in white males.

768. The Council considered this study:

– points to a relevant association.

Becker, N et al. 2001, 'Asbestos exposure and malignant lymphomas - a review of the epidemiological literature', *Int Arch Occup Environ Health*, vol. 74, no. 7, pp. 459-469 RMA ID 26350 FILEForce ID 18523

769. This literature review described 6 cohort and 16 case controlled studies published between 1966 and 1999 containing information on asbestos exposure and risk of lymphoma.

770. Relative risks or odds ratios with confidence intervals were reported where available. In addition the authors used this data to compute a combined evaluation, however with regard to this calculation they note:

> The studies turned out to be much too heterogeneous in terms of the design to be used as a tool for confirmation. [658]

771. For CLL no cohort studies and 3 case control studies were available. Case control studies were obtained from hospital records or cancer registries and established asbestos exposure via occupational history on interview.[659]

772. Results for asbestosis from the case control studies [660] were:

a. Linet, MS et al. 1987a - 342 diagnosed cases of CLL at hospitals in Baltimore, together with an equal number of controls.

Job-exposure matrix A	OR = 1.1	95% CI 0.65-1.92
Job-exposure matrix B	OR = 0.65	95% CI 0.4-1.1[661]

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658 See at p. 460.

659 See at p. 464 and Table 3 Summary of case control studies.

660 Council notes that the papers Linet, MS et al. 1987 and Schwarz, DA et al. 1988 reviewed in Becker, N et al. 2001, were not included in the Goodman, M et al. 1999 meta analysis. It is assumed that these studies did not meet the inclusion criteria of results reported as standardised incidence rates.

Linet, MS et al. 1987, 'Comparison of methods for determining occupational exposure in a case-control interview study of chronic lymphocytic leukemia', *J of Occupational Med*, vol. 29, no. 2, pp. 136-141.

661 Table 2 at p. 138 supra.

– Job-exposure matrix A - Hoare et al and B - National Occupational Hazard Survey, NOHS 1978.

b. Schwartz, DA et al. 1988 (see from [757] above) - 429 cases of CLL, from cancer registers of various hospitals and 1,683 population controls.

exposure: low	OR = 1.3	95% CI 0.9-1.9
medium	OR = 1.2	95% CI 0.8-1.9
high	OR = 1.7	95% CI 1.0-3.0

c. Pasqualetti, P et al.1991[662]; 65 cases of CLL and 1,240 age and gender matched hospital controls, with an odds ratio for asbestos exposure of:

OR = 4.7	95% CI 1.2 – 18.1

773. The authors note however that the Pasqualetti, P et al.1991 study:

> … contains no indication of how the asbestos exposure or level of exposure were categorized. It also fails to state how many exposed persons were included in the analysis. Finally, it is not clear whether any adjustment was made for the different sources of information used in the study. [663]

774. The authors reported their combined assessment of the 3 case control studies with two different models for CLL as: [664]

fixed effect model	OR = 1.3	95% CI 1.07 - 1.7
random effect model	OR = 1.5	95% CI 0.8 - 2.8

775. The authors note however:

> Among the three studies on CLL the work of Linet et al. [1987] gives a negative answer to the question of increased risk; that of Schwartz et al. [1988] positive results. The study by Pasqualetti et al.[1991] provided significantly positive results, but is so poorly documented that judgment is difficult.

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662 Pasqualetti, P et al. 1991, 'Occupational risk for hematological malignancies, Am J Hematol, vol. 38, pp. 147-149.

This article was not available to the RMA at the relevant times, and so could only be considered by the Council as new information, however the information for Pasqualetti, P et al. 1991 above as cited in **Becker, N et al. 2001** was considered by the Council as information available to the RMA at the relevant times.

663 See at p. 464.

664 See at p. 467 and Table 5.

776. The authors concluded:

> The combined ORs indicate, for all sub-entities of lymphomas under consideration (NHL, CLL, plasmacytoma), an increased asbestos related risk.[665]

777. The authors discussed the following considerations:

- The challenge of classification inconsistence among pathologists with specific reference to CLLs traditionally classified as leukaemia's but more recently as lymphomas and therefore the potential of underreporting CLL cases in studies.
- The challenge of accurately determining exposure assessment.
- Overestimation of risk from positive result publication bias.
- Lack of information to accurately address the issue of biological plausibility.

778. A number of recommendations for future studies were suggested based on these considerations.

Council's comment

779. The Council considered this meta-analysis found a weak positive association based on 3 studies, but the result was shown in the fixed effect model, which in the Council's view is more likely to provide a positive effect than in the random effect model which would be more convincing evidence of an association.

780. The Council considered that this study:

- did not point to, but merely left open the possibility of a relevant association.

COUNCIL'S CONCLUSIONS – ASBESTOS

781. The Council considered that the combined effect of the studies by:

Schwartz, DA et al. 1988

that pointed to (as opposed to merely left open) a relevant association, and

Becker, N et al. 2001

that left open the possibility of a relevant association,

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665 See at p. 467.

provide sound medical-scientific evidence to justify amending the reasonable hypothesis Statement of Principle, but did not did not satisfy the balance of probabilities test.

782. The Council also carefully considered other articles that in its view were sound medical-scientific evidence that touched on the relevant potential asbestos factor that were in Council's final analysis non-contributory:[666]

> Goodman, M et al. 1999
>
> Kagan, E & Jacobson, RJ 1983

783. The Council did not focus on those articles in these Reasons as it could not draw any conclusions from them as they:

- did not provide data specific to CLL; and
- in the case of Kagan, E & Jacobson, RJ 1983, there were no control cases and no confounders were evaluated.

784. The Council having considered all the information both individually and collectively, and conscious that the reasonable hypothesis test is one of possibility, and an unusually light burden, was convinced that the reasonable hypothesis test had been met.

785. Being satisfied that the reasonable hypothesis test was met, the Council went on to consider whether, on the sound medical-scientific evidence available, it is more probable than not that B-cell CLL can be related to exposure to asbestos. The Council decided that the sound medical-scientific evidence does not indicate the relevant association to the level of satisfaction of the balance of probabilities.

Should There Be A Exposure To Tobacco Smoking Factor?

Stagnaro, E et al. 2001, 'Smoking and hematolymphopoietic malignancies', *Cancer Causes Control*, vol. 12, no. 4, pp. 325-334. RMA ID 26238, FILEForce 18512

786. This Italian case-control study of 2,734 patient with blood cancers (1,450 non-Hodgkin's lymphoma (NHL), 365 Hodgkin's Disease, 270 Multiple Myeloma, 649 Leukemia), compared 1,779 population controls (gender and 5 year age matched). Subjects were interviewed (face-to-face) about smoking history.

> Because the classification of CLL differs somewhat in published studies, we considered results for CLL combined with small cell NHL, as well as separately, to allow comparison with previous analyses.[667]

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666 Articles that did not, in Council's final analysis, contribute to either advance or weaken the state of evidence of the possibility of a relevant association (i.e, were non contributory).

787. An association was found with NHL (OR 1.2, 95% CI 1.0 – 1.4) described by the authors as slight, but significant, with the association being specific for follicular lymphoma in women (ever smoker vs never smoked, OR 2.3, 95% CI 1.4 – 3.8) [668]. No other significant associations were found.

788. There were 345 patients with SLL: smoking was not associated with increased risk, whether analysed by "never-" vs "ever-smoker" (OR 1.0, 95 % CI 0.77-1.3), number of cigarettes per day (OR's of 0.7-1.3, with the highest exposure group having the lowest risk), and duration (OR's of 0.97-1.0) [669].

789. There were 214 cases of CLL, and the association was negative whether considered by "never vs ever" smoker (OR 0.87, 95% CI 0.61-1.2), cigarettes per day (OR's of 0.58-1.3, with the highest exposure group having the lowest risk), and duration of smoking (OR's of 0.76-0.91) [670].

Council's comments

790. The Council considered this was a strong study because it was a large study with a substantial number of cases.

791. The Council found the complete lack of association, and the reverse association between number of cigarettes per day and SLL/CLL, to be evidence that smoking is not associated with CLL.

792. The Council considered that this study indicated:

- against the possibility of a relevant association.

Friedman, GD 1993, 'Cigarette smoking, leukemia, and multiple myeloma', *Annals of Epidemiology*, vol. 3, pp. 425-428. – RMA ID 20968, FILEForce 18885

793. This paper discussed the findings of a US study of 142,991 persons who received health check-ups through a health maintenance program (Kaiser Permanente). Subjects had their health check-up between 1964 and 1972, and cancer data was available up to 1988 (through Kaiser hospital records and SEER data).

794. There were 117 cases of CLL. There were no associations between CLL and smoking status, whether considered former or current. Relative risk (RR) for the various categories ranged between 0.6 – 1.0 [671].

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667 See page 327.

668 See Table 2 at p. 329 and at p. 328.

669 See Table 3 at p. 330.

670 See Table 4 at p. 331.

671 See at p. 426.

Council's comments

795. The Council considered this was a strong study providing evidence that smoking is not associated with CLL.

796. The Council considered that this study indicated:

- against the possibility of a relevant association.

Morris Brown, L et al. 1992, 'Smoking and risk of leukemia', *American Journal of Epidemiology*, vol. 135, no. 7, pp. 763-768. – RMA ID 21505, FILEForce 18890

797. This paper discussed the findings of an US (Iowa and Minnesota) case-control study of 578 white men with leukaemia, and 820 controls. Cases were identified between 1981 – 1984 in Iowa via the Iowa Health Registry (a SEER member), and from a "network of hospitals and pathology laboratories" in Minnesota. Subjects were interviewed for history of cigarette and other exposures. The authors excluded 425 control patients because of their deceased status.

798. The headline result was that tobacco users have increased risk of total leukaemia (RR 1.4, 95% CI 1.1-1.9). There were 40 CLL cases in non-tobacco users, compared with 203 in tobacco users (RR 1.6, 95% CI 1.1-2.3) [672]. When analysed by number of cigarettes per day or number of years smoked, there was no dose-response effect in CLL.

Council's comments

799. Although a positive association between all leukaemia (including CLL) and smoking was reported, the Council had concerns about the robustness of the study methodology. Specifically:

a. the manner in which cases were identified from Minnesota was not stated clearly, and it was not possible to gauge whether selection bias may have occurred in this population; and

b. one-third of control subjects were excluded due to their deceased status, and therefore the control population may have been healthier than normal controls. Indeed, if the analysis was to be preformed with the deceased control subjects included, the RR for leukaemia becomes statistically non-significant (RR 1.3, 95% CI 1.0 – 1.7).

c. the lack of dose-response effect between smoking and CLL points away from an association.

800. The Council considered that this study:

[672] See Table 1 at p. 765 and p. 764.

– did not point to, but merely left open the possibility of a relevant association.

Garfinkel, L & Boffetta, P 1990, 'Association between smoking and leukemia in two American cancer society prospective studies', *Cancer*, vol. 65, pp. 2356-2360. – RMA ID 21504, FILEForce 18891

801. This paper discussed an analysis based on two prospective studies [673] conducted by the American Cancer Society: Cancer Prevention Study I (CPS I) and II (CPS II). CPS I collected exposure information via questionnaire from >1,000,000 subjects in 1959, and followed yearly until 1965. Subjects who died had their diagnoses obtained from death certificates, and those who died of leukemia had the information verified with their hospitals / doctors. CPS II was similar and enrolled >1,200,000 subjects in 1982, with mortality followed up to 1986.

802. The SMR for lymphocytic leukemia was not elevated in either study (SMR 1.02 and 1.24 for CPS I and II [no CI provided, but P was not < 0.05]), with no apparent dosage effect seen (patients are subdivided into 1-19 cigarettes daily vs >20 cigarettes daily).

Council's comments

803. The Council considered this was a persuasive study because it used prospective information and had a huge number of subjects. However, mortality is not a sensitive way of capturing CLL occurrence.

804. The Council considered that the CPS I and II studies provide strong evidence:

– against the possibility of a relevant association

International Agency for Research on Cancer (IARC) 2004, Overall Evaluations of Carcinogenicity Risks to Humans: *Tobacco Smoke and Involuntary Smoking*, IARC Monograph, vol. 83. IARC, Lyon, France. – RMA ID 32051, FILEForce 19133

805. This IARC publication [674] is on tobacco smoke and involuntary smoking. In the section on leukemia, the IARC stated that myeloid leukaemia was observed to be

673 Hammond, EC 1996, 'Smoking in relation to death of one million men and women'. In Haenszel, W ed. 1966, *Epidemiological approached to the study of cancer and other diseases*, Bethesda MD, National Cancer Institute, pp. 127-204.

Kinlen, LJ & Rogot, E 1988, 'Leukemia and smoking habits among United States veterans, Br Med J, vol. 297, pp. 657-659.

These articles were not available to the RMA at the relevant times, and so could only be considered by the Council as new information. However, the data at [775864] from the two studies is data cited in **Garfinkel, L & Boffetta, P 1990** and as such was considered by the Council.

674 IARC publication of Monographs relating to carcinogenic risk of chemicals to humans commenced in 1969. With regard to biological and epidemiological data, only reports that have been published or accepted for publication in the openly available scientific literature are reviewed by the [IARC] working groups. The evidence related to carcinogenicity from studies in humans is classified into

causally related to smoking, with an adequate number of studies showing a dose-effect relationship [675].

806. The major contributing chemical was presumed to be benzene, which is estimated to account for approximately half of the excess AML risk in smokers.

807. With regards to lymphoid leukaemia (including CLL) [676],

> Only two of eight studies provide any evidence of an increased risk associated with smoking (Paffenbarger et al. 1978; Linet et al. 1991) and in neither case was the excess risk statistically significant. …some data are also available from case-control studies…Brownson et al. 1993 undertook an overview of the published studies of cigarette smoking and adult leukaemia, they found that a pooled analysis of eight case-control series…gave relative risks of 1.1 (95% CI 1.0-1.2) for all leukaemias, 0.9 (95% CI, 0.8-1.1) for chronic lymphoid leukaemia…

it stated that no clear evidence of any risk was seen in studies [677] to date.

Council's comments

808. The Council considered this was a authoritative review from an influential and important body in the cancer science community that failed to find evidence to support a link.

809. The Council considered that this study:

- not point to, but merely left open the possibility of a relevant association

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categories: Sufficient evidence, limited evidence, Inadequate evidence of carcinogenicity; Evidence suggesting lack of carcinogenicity (IARC Monographs vol. 83, 2004, Preamble at p. 9-31).

The Council notes that the methodology of the IARC relating to determining sufficient, limited and inadequate evidence does not reflect the Council's two step process in applying the reasonable hypothesis test as set down by the full Federal Court (see [108] to [109])

675 See at p. 822.

676 See at p. 822-823.

677 See at p. 822-823

Paffenbarger, RS et al 1978, 'Characteristics in youth predictive of adult onset malignant lymphomas, melanomas, and leukaemia: Brief communication', *J natl Cancer Inst*, vol. 60, pp. 89-92.

Linet, MS et al 1991, 'Cigarette smoking and Leukaemia, results from the Lutheran Brotherhood Study', *Cancer Causes Control*, vol. 2, pp. 413-417.

Brownson, RC et al. 1993, 'Cigarette smoking and adult Leukaemia, Arch intern Med, vol. 153, pp. 469-427.

These 3 articles were not available to the RMA at the relevant times, and so could only be considered by the Council as new information. However, the data at [8488870] from the three studies is cited in **IARC 2004** and as such was considered by the Council.

Herrington, LJ & Friedman, GD 1998, 'Cigarette smoking and risk of non-Hodgkin's lymphoma subtypes', *Cancer Epidemiology, Biomarkers & Prevention*, vol. 7, no. 1, pp. 25-28. – RMA ID 13003, FILEForce 18976

810. This paper discussed the same subjects as the Kaiser Permanente cohort [678]. This study included more patients (252,836, smoking history between 1964 – 1991), and studied Non-Hodgkin's Lymphoma (NHL).

811. Using the SEER[679] and Kaiser Permanente cancer databases, 674 incidence NHL cases were identified up to 1993.

812. At Table 2 [680], RR was significant only for follicular lymphoma, without a dose-effect. Fifty-six cases of SLL were present, with a relative risk (RR) of 1.0 (0.5-1.8) for former smokers and 0.5 (0.3-1.1) for current smokers.

Council's comments

813. The Council considered this was a negative and underpowered (not enough cases) study. The confidence intervals were very wide.

814. The Council considered that this study provides weak evidence:

- against the possibility of a relevant association

Zahm, SH et al. 1997, 'Tobacco and non-Hodgkin's lymphoma: combined analysis of three case-control studies United States, *Cancer Causes & Control*, vol. 8, no. 2, pp. 159-166. – RMA ID 13045, FILEForce 18975

815. This paper discussed the combined analysis of 3 US-based population case-control studies, after the authors had identified 2 then recent studies which were supportive of an association between smoking and NHL [681]. There were 1177 cases of NHL, and 3625 controls. There was no association between NHL and tobacco use (OR 1.0, 95% CI 0.8 - 1.1), with a suggestion to perhaps an increased risk in females (OR 1.3, 95% CI 0.9 - 1.9). There was no dose-response gradient.

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678 Friedman, GD 1993, 'Cigarette smoking, leukemia, and multiple myeloma', *Annals of Epidemiology*, vol. 3, pp. 425-428. See from [793]

679 Surveillance, Epidemiology, and End results cancer registry (SEER).

680 Table 2 at pp. 27.

681 Morris Brown, L et al. 1992, 'Smoking and risk of non-Hodgkin's lymphoma and multiple myeloma', *Cancer Causes Control*, vol. 3, pp. 49-55.

Linet, MS et al. 1992, 'Is cigarette smoking a risk factor for non-Hodgkin's lymphoma or multiple myeloma? Results form the Lutheran Brotherhood Cohort Study*, Leukemia Res*, vol. 16, pp. 621-624.

These 2 articles were not available to the RMA at the relevant times, and so could only be considered by the Council as new information. However, the data from the two studies was cited in **Zahm, SH et al.1997** and as such was considered by Council.

816. The OR for small lymphocytic lymphoma (n=121) was 0.7 (95% CI 0.5-1.1).

Council's comments

817. The Council considered this was a negative study due to the findings at [815], which indicate a reduced risk for SLL in smokers.

818. The Council considered that this study provides weak evidence:

– against the possibility of a relevant association

COUNCIL'S CONCLUSIONS –SMOKING

819. The Council found no study that pointed to the possibility of a relevant association. The Council considered that the combined effect of the studies by:

Stagnaro, E et al. 2001

Zahm, SH et al. 1997

Garfinkel, L & Boffetta, P 1990

International Agency for Research on Cancer (IARC) 2004

Friedman, GD 1993

Herrington, LJ & Friedman, GD 1998

that indicated against the relevant association; and the studies by:

Morris Brown, L et al. 1992

that did not point to but merely left open the possibility of a relevant association between smoking and CLL was insufficient to justify any amendment to the Statements of Principles in respect of tobacco smoking.

820. The Council also carefully considered other articles that in its view were sound medical-scientific evidence that touched on the potential tobacco smoking factor that were in the Council's final analysis non-contributory:[682]

National Health and Medical Research Council (NHMRC) 1997

[682] Articles that did not, in Council's final analysis, contribute to either advance or weaken the state of evidence of the possibility of a relevant association (i.e, were non contributory).

Nordstrom, M et al. 1998

Adami, J et al 1998

Freedman, DS et al. 1998

821. The Council did not focus on those articles in these Reasons as those studies:

- did not provide data specific to CLL;
- provided data on haematopoietic cancers, leukaemia and lymphoma, and or Non-Hodgkin's lymphoma that the Council could not draw conclusions from concerning CLL or B-cell CLL;
- were in respect of such small number of cases of CLL, haematopoietic cancers, leukaemia and or lymphoma that the Council could not draw conclusions from concerning CLL or B-cell CLL.

THE COUNCIL'S CONCLUSIONS – ALL FACTORS

822. The Council closely analysed all the information in the pool when considering each of the contended factors within the scope of the review, placing particular weight on the articles discussed in detail above.

823. The Council considered that the relevant association must be analysed on the basis of the whole body of information in the pool. The Council closely analysed all the information in the pool which touched on the contended factors. However, it placed particular weight on the articles discussed in detail above.

824. The critical question for the Council was whether the sound medical-science 'points to, as opposed to merely leaves open, the possibility of a relevant association.' [683] It is only if the Council answered that question in the affirmative, that it needed to consider whether the relevant association was established on the balance of probabilities.

825. Always cognisant that the reasonable hypothesis standard is a 'test of possibility' and 'an unusually light burden', the Council considered the combined effect of the information as follows.

Non-ionising Radiation

826. The Council considered that the studies by:

Floderus, B et al. 1993

[683] See full Federal Court decision at [49] per Branson J and [61] above.

Feychting, M et al. 1997

Kheifets, LI et al. 1997

Havas, M 2000

California Department of Health Services (DHS) Anon 2001

point to (as opposed to merely leaves open) a relevant association (non-ionising radiation with B-cell CLL), but do not satisfy the balance of probabilities test;

827. The Council considered that the studies by:

Lai, H & Singh, N 1995

Krewski, D et al. 2001a

support the biological plausibility of the relevant association, that is, for non-ionising radiation with B-cell CLL.

828. The Council considered that the studies that, in the Council's view left open the possibility of a relevant association, also provide some support to the Council's decision.

Herbicides, Pesticides, Dioxin and Potable Water

829. The Council considered that the study by

Muller, J et al. 2002;

that supports potable water as a mechanism for exposure to dioxin; together with the studies by:

Morris Brown, L et al. 1990;

IOM Update 2006;

Wilson, EJ et al. 2005 a (Cancer incidence); and

Hertzman, C et al. 1997; and

that pointed to (as opposed to merely left open) a relevant association, and in particular:

Waterhouse, D et al. 1996;

Amadori, D et al. 1995;

Lee, WJ et al. 2004

Mills, PK et al. 2005

that did not point to but merely left open the possibility of a relevant association, but provides evidence in support of a positive trend;

is a body of sound medical-scientific evidence that was available to the RMA that justifies amending the reasonable hypothesis Statement of Principle, but does not satisfy the balance of probabilities test.

Benzene

830. The Council considered that :

Australian Institute of Petroleum (AIP) 2001

Glass, DC et al. 2003

study pointed to the relevant association and having considered the whole of the information, the Council was satisfied that the sound medical-scientific evidence that was available to the RMA justifies amending the reasonable hypothesis Statement of Principles but not the balance of probabilities Statement of Principles.

Other Aromatics / Hydrocarbons - toluene, xylene and styrene and carbon tetrachloride

831. The Council decided that, overall, the sound medical-scientific evidence was insufficient to justify any amendment to the Statements of Principles to include other aromatic hydrocarbons as a factor, there being no information in respect of other aromatic hydrocarbons that specifically addressed B-cell CLL or from which the Council could draw relevant conclusions..

AV Gas, MEK & Sealants (FIII)

832. The Council considered the sound medical-scientific evidence available to the RMA was insufficient to justify any amendment to the Statements of Principles in respect of AV Gas and associated chemicals in relation to aircraft fuel tank de-seal / reseal activities as there was no information that provided data sufficiently specific to B-cell CLL or from which the Council could draw any relevant conclusion.

Asbestos

833. The Council considered that the study by:

Schwartz, DA et al. 1988

that pointed to (as opposed to merely left open) the relevant association, and the study by:

Becker, N et al. 2001

that did not point to but merely left open the possibility of a relevant association, provide sound medical-scientific evidence sufficient to justify amending the reasonable hypothesis Statement of Principle, but not the balance of probabilities Statement of Principles.

Tobacco smoking

834. The Council considered that the combined effect of the studies by:

Stagnaro, E et al. 2001

Zahm, SH et al. 1997

Garfinkel, L & Boffetta, P 1990

International Agency for Research on Cancer (IARC) 2004

Friedman, GD 1993

that were against the relevant association; and the studies by:

Herrington, LJ & Friedman, GD 1998

Morris Brown, L et al. 1992

that did not point to but merely left open the possibility of a relevant association, did not provide sound medical-scientific evidence was sufficient to amend the Statements of Principles.

DECISION

835. The Council made the declarations, directions and recommendations in paragraphs 1 to 4 above.

EVIDENCE BEFORE THE COUNCIL

Documents

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

837. As mentioned above, the information upon which the Applicants, other persons and the Commissions relied (being information which was available to (before) the RMA

and sent to the Council by the RMA in accordance with section 196K of the VEA is listed in **Appendices C** (Mr FR) and **E** (Mr AR) and **F** (Mrs F) and **I** (Commissions).

838. The information to which the Applicant, other persons and the Commissions referred (being information which was not available to (not before) the RMA, and so was not considered by the Council in reaching its decision) is listed in **Appendices D** (Mr FR) and **G** (Mrs F) and **H** (Mr G) and **J** (Commissions).

ARTICLES CITED BY THE COUNCIL

Exposure to non ionising radiation

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Appendices

Appendix A	Preliminary list of the proposed pool of information, as advised to the Applicants (Mr FR & Mr AR), Mrs F, Mr G, Mr T, Mr M and the Commissions by letters dated 11 March 2011 (see [32] – [33] and the final pool of information see [119].
Appendix B	Information available to (before) the RMA and sent to the Review Council by the RMA under section 196K via FileForce in April 2009.
Appendix C	Information upon which the Applicant Mr FR relied (being information which was available to (before) the RMA and sent to the Review Council by the RMA in accordance with section 196K of the VEA).
Appendix D	The information to which the Applicant Mr FR referred (being information which the RMA advised was 'new information', that is, information which was not available to (not before) the RMA at the relevant times, and so was not considered by the Review Council in reaching its review decision). The Council has listed only the primary source cited articles and not secondary source citations from within those primary articles.
Appendix E	Information upon which the Applicant Mr AR relied (being information which was available to (before) the RMA and sent to the Review Council by the RMA in accordance with section 196K of the VEA).
Appendix F	Information upon which Mrs F relied (being information which was available to (before) the RMA and sent to the Review Council by the RMA in accordance with section 196K of the VEA).
Appendix G	The information to which Mrs F referred (being information which the RMA advised was 'new information', that is, information which was not available to (not before) the RMA at the relevant times, and so was not considered by the Review Council in reaching its review decision).
Appendix H	The information to which Mr G referred (being information which the RMA advised was 'new information', that is, information which was not available to (not before) the RMA at the relevant times, and so was not considered by the Review Council in reaching its review decision).

Appendix I	Information upon which the Commissions relied (being information which was available to (before) the RMA and sent to the Review Council by the RMA in accordance with section 196K of the VEA).

Note: Appendices A to I are part of the Reasons for Decisions and for printing purposes are provided as a separate document.