



Specialist Medical Review Council

Reasons for Decisions

*Section 196W
Veterans' Entitlements Act 1986*

**Re: Statements of Principles Nos. 68 & 69 of 2008
In Respect of Rheumatoid Arthritis**

Request for Review Declaration No. 18

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SUMMATION

1. In relation to the Repatriation Medical Authority (the RMA) Statement of Principles No. 68 of 2008 in respect of rheumatoid arthritis and death from rheumatoid arthritis, 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 the sound medical-scientific evidence available to the RMA is insufficient to justify an amendment to Statement of Principles No. 68 of 2008 to include factors for exposure to mineral oils containing polychlorinated biphenyls (PCBs) or acute PCB exposure or to amend the definition of 'mineral oil' as contended.

2. In relation to the RMA Statement of Principles No. 69 of 2008 in respect of rheumatoid arthritis and death from rheumatoid arthritis, made under subsection 196B (3) of the VEA, the Council under subsection 196W of the VEA:

DECLARES that the sound medical-scientific evidence available to the RMA is insufficient to justify an amendment to Statement of Principles No. 69 of 2008 to include factors for exposure to mineral oils containing PCBs or acute PCB exposure.

THE SPECIALIST MEDICAL REVIEW COUNCIL

3. 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. 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.
4. When a review is undertaken the Council is constituted by three to five Councillors selected by the Convener. When appointing Councillors, the Minister is required to have regard to the branches of medical-science that would be necessary for deciding matters referred to the Council for review.
5. Professor John Funder was the Presiding Councillor for this review. Professor Funder is a Senior Fellow at Prince Henry's Institute of Medical Research at Monash Medical Centre, and holds honorary appointments at Monash, Melbourne University and the University of Queensland. He has been President of the Australian Society for Medical Research (1979) and the Endocrine Society of Australia (1984), and Chairman of the International Society for Endocrinology (1996-2000).

6. The other members of the Council were:

(i) Professor Rachelle Buchbinder

Professor Buchbinder is Director of the Monash Department of Clinical Epidemiology at Cabrini Hospital and Professor, Department of Epidemiology and Preventive Medicine, School of Public Health and Preventive Medicine, Monash University. She is a rheumatologist and clinical epidemiologist.

(ii) Associate Professor Geoff Littlejohn

Associate Professor Littlejohn is Associate Professor of Medicine and Director of Rheumatology at Monash Medical Centre, Melbourne, and Adjunct Professor at Edith Cowan University, Perth. He completed an MD thesis on Diffuse Idiopathic Skeletal Hyperostosis in Toronto and has remained an international expert in that field. He has also published widely in other rheumatic disorders including inflammatory joint disease, chronic pain syndromes and osteoarthritis.

(iii) Associate Professor Peter Nash

Associate Professor Nash is the Director of the Rheumatology Research Unit within the Department of Medicine at the University of Queensland. He is a member of the Professional Affairs and Scientific Advisory Committees of the Australian Rheumatology Association, and the Therapeutic Committee of the Australia and New Zealand Bone & Mineral Society. He is the former Chair of the Therapeutics Committee of the Australian Rheumatology Association, and former Deputy Chair of the National Health and Medical Research Council's Musculoskeletal panels.

THE LEGISLATION

7. 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.

8. 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

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

- 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
9. 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. 68 of 2008, in respect of rheumatoid arthritis and death from rheumatoid arthritis, being a Statement of Principles determined by the RMA under section 196B(2)³ of the VEA ('the **reasonable hypothesis test**') and
 - Statement of Principles No. 69 of 2008, in respect of rheumatoid arthritis and death from rheumatoid arthritis, being a Statement of Principles determined by the RMA under section 196B(3)⁴ of the VEA ('the **balance of probabilities test**').

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;

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

10. Specifically, the Applicant contended that there was sound medical-scientific evidence on which the RMA could have relied to include as a factor or factors in Statements of Principles Nos. 68 and 69 of 2008, exposure to mineral oils PCBs or acute PCB exposure.
11. 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 Statements of Principles (the relevant times) and is constrained to conduct its review by reference to that information only.⁵
12. 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

Application for review by the Council

13. On 22 October 2008, the RMA under subsections 196B(2) and (3) of the VEA determined Statements of Principles Nos. 68 and 69 of 2008, in respect of rheumatoid arthritis. The Statements of Principles took effect from 5 November 2008.
14. On 25 October 2008 the Statements of Principles were registered on the Federal Register of Legislative Instruments.
15. On 10 November 2008 in accordance with section 42 of the *Legislative Instruments Act 2003* the Statements of Principles were tabled in the House of Representatives and in the Senate.
16. An Application for Review of Statements of Principles Nos. 68 and 69 of 2008 was received by the Council on 19 January 2009.⁶ The Application contended that the Statements of Principles should include a factor or factors concerning exposure to PCBs and Mineral Oils.
17. 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 rheumatoid arthritis and invited eligible persons or organisations so authorised to make submissions to the Council.⁷ The Council gazetted two

(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.

⁶ Within the time prescribed by section 196Y(2) of the VEA.

⁷ Gazette Notice No. 6 of 18 February 2009, pages 438 and 439.

subsequent notices as to the dates by which written submissions must be received by the Council.⁸

The information sent by the RMA to the Council

18. By email dated 17 February 2009 the RMA, under section 196K of the VEA, sent to the Council the information the RMA advised was available to (before) it at the relevant times, as listed in Appendix B.
19. By agreement between the RMA and the Council, information the RMA advised was available to (before) it at the relevant times is posted on a secure website (referred to as FILEForce). It is made accessible by the Council to the Repatriation Commission and the Military Rehabilitation and Compensation Commission (the Commissions), the Applicant and other participants in the review via confidential password.

Notification of Preliminary Decisions on Proposed Scope of Review and Proposed Pool of Information

20. In separate letters, dated 22 March 2012, to each of the Applicant and the Commissions, the Council in summary:
 - advised of the Council's preliminary decisions on the proposed scope of the review and proposed pool of information;
 - invited the Applicant and Commissions to make any written comments as to the Council's preliminary decisions by close of business on 20 April 2012; and
 - advised that if any written comments were made, any complementary oral comments could be made at a hearing of oral submissions complementing the written submissions.
21. No comments were received.
22. The Council held a meeting on 18 July 2012 to consider all the written submissions and complementary oral submissions.

Proposed Scope of Review

23. The Council's preliminary decision on the scope of the review, as advised to the Applicant and Commissions on 22 March 2012, was as follows:

'Without limiting the scope of its 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 RMA could have relied to amend either or both of the Statements of Principles by:

⁸ Gazette Notices No. 2 of 20 January 2010 and No. SG231 of 30 December 2010.

- (i) the possible inclusion of a factor or factors, or possible amendment of factors 6(c), 6(g) and the definition of 'mineral oil' in paragraph 9 of Statement of Principles No. 68 of 2008, as contended, for exposure to mineral oils containing PCBs or acute PCB exposure ; and
- (ii) the possible inclusion of a factor or factors in Statement of Principles No. 69 of 2008 as contended, for exposure to mineral oils containing PCBs or acute PCB exposure.

Proposed Pool of Information

- 24. As mentioned above, 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) at the relevant times. That comprises all the information that was available to the RMA when it determined the original Statements of Principles in respect of rheumatoid arthritis 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 the information which was available in October 2008 when the RMA determined the 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 rheumatoid arthritis which was in the possession of the RMA at the time it (the RMA) made the decision that triggered the Council's review.
- 25. The chronology of the RMA sending the information to the Council is detailed in [18]. As mentioned above, all the information which was available to the RMA at the relevant times was made available to the Applicant and the Commissions for the purposes of the review.
- 26. In determining its preliminary view on the proposed pool of information the Council applied the methodology it had advised the Applicant and Commissions on 22 March 2012, i.e. that the pool of information should comprise the information:
 - that was available to (before) the RMA at the relevant times;
 - which was sent by the RMA to the Council under section 196K of the VEA;
 - which was considered by the Council to be sound medical-scientific evidence as defined in section 5AB(2) of the VEA being information which:
 - (1) epidemiologists would consider appropriate to take into account; and
 - (2) in the Council's view 'touches on' (is relevant to) exposure to mineral oils containing PCBs or acute PCB exposure; and
 - which has been evaluated by the Council according to epidemiological criteria, including the Bradford Hill criteria.⁹

⁹ 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, pp. 295 - 300.

27. Information which the RMA advised was not available to (not before) the RMA at the relevant times, was not taken into account by the Council for the purposes of the review, as it could only be considered as 'new information' (see [233] *et seq*).
28. A copy of the Council's preliminary list of the proposed pool of information was forwarded to the Applicant and the Commissions and is attached at Appendix A.

APPLICANT'S SUBMISSIONS

29. The Applicant made:
 - a written submission dated 25 February 2010 and
 - an oral submission complementing his written submissions on 18 July 2012both of which were taken into account by the Council.¹⁰
30. In his Application to the Council of 19 January 2009, the Applicant stated that his grounds for review were as follows:

That [factor 6 (c)] in Statement of Principles No. 68 of 2008:

...infers cutaneous contact with Mineral Oil for a cumulative period of 2500 hours is the same as inhaling respirable silica dust for the same period.

31. The Applicant contended that the Statements of Principles do not address the particular characteristics of oils containing PCBs or acute PCB exposure.

I believe the SoP has merit when applied to vapour and surface contact with Mineral Oils but does not address oils containing PCBs or an acute PCB exposure

32. He claimed that evidence that he had previously supplied to the RMA

...indicates that mineral oils containing PCBs are highly penetrative and rapidly absorbed through the skin with resultant allergic and immune responses.

...even the slightest exposure affects the immune system.

33. The Applicant also contended that the latency period for PCB exposure should be expanded, claiming that evidence he had previously submitted to the RMA has shown that:

...for PCB exposure where exposure has ceased, clinical onset is more likely around 15-25 years and not within 10 years as generalised in the SOP.

¹⁰ The information upon which the Applicant relied, being information which the RMA advised was available to (before) the RMA at the relevant times, is listed in Appendices A and C.

34. In his written submission dated 25 February 2010, which was received by the Council on or about 25 February 2010, the Applicant contended that the Statements of Principles should include:

...highly chlorinated mineral oils, specifically PCBs.

35. He claimed that that RMA's decision

...appears to be general in nature regarding mineral oils and does not address my request for inclusion of highly chlorinated mineral oils, specifically PCBs. The chlorine additive changes the characteristic of the mineral oil making it a highly penetrative substance and a possible carcinogenic . It has been shown to affect the immune system resulting in hypersensitivity and/or autoimmunity.

Further claiming that:

Studies of human exposure have shown rheumatoid arthritis has been linked to PCB exposure and animal studies have shown even the slightest exposure effects the immune system.

The information I have previously submitted appears to have been overlooked as the SOP amendment considers all mineral oils to have the same characteristics ...as silica gel for exposure purposes.

36. The Applicant claimed that PCBs were 'used extensively by the RAAF in high voltage transformers in Radar installations', and that as a member of the RAAF he was exposed to PCBs in 1981 and subsequently diagnosed with rheumatoid arthritis in 1983.
37. In his written submission to the RMA dated 20 July 2006 (RMA ID S1.12), to which he refers the Council, the Applicant describes an incident which occurred while he was a member of the RAAF, in which he experienced cutaneous exposure to transformer oil.

The coils inside the transformer overheated and burnt out. They were located on a wooden rod at the bottom of the transformer enclosure - approximately 60cm from top. I reached into the tank as we had no tools of that length for extraction nor any protective equipment - oil covered the whole of both arms and the upper portion of my overall sleeves. Spillage occurred onto my overall front and legs as the rod and coils were extracted. Excess oil was wiped off my skin while we replaced the coils, reinserted the rod, topped up the oil, reinstalled the transformer and tested. After approximately one hour, I washed my hands and forearm with soap leaving the rest of my body until I went home for a shower where I removed my soiled clothing approximately 4-6 hours later. The contaminated overalls and underwear were washed in the family washing machine by my wife and I continued wearing them long after discharge.

Transformer oils are normally highly chlorinated, especially French transformers used in a sealed environment which this one was - Thomson-CSF was the French manufacturer. Most research has been conducted on oral, atmospheric intake through lungs/skin and contact with contaminated surfaces.

He claims that the contact he experienced was

...an acute skin exposure via immersion where there is no available data so I have had to rely on existing exposures and outcomes as a guideline. Current research data has been obtained through experimentation on rats, monkeys, (reportedly closest to humans), human cadavers and monitoring results of recorded exposures.

38. The Applicant provided a copy of a RAAF internal memo which, he claims, confirm that the materials he was working with contained PCBs.¹¹
39. In his oral submission the Applicant claimed that PCBs are significantly more toxic than silica gels and questioned why the SoPs use the same exposure doses for silica and mineral oils.

...trying to compare ...something that's really highly toxic to touch, to ingest, to smell (with) silica gel which really is more dust, silicosis... ingestion through the breathing system.

40. The Applicant referred the Council to the ATSDR 2011 Priority List of Hazardous Substance¹², supplied by him, and claimed that in terms of toxicity, the ATSDR listed

Silica gel [at] around about 700 ...and PCBs was number 5 on the list.¹³

41. The Applicant made submissions to the Review Council on the basis of his interpretation of the following articles which he claimed support his request to include PCB exposure in the SOP as a cause of rheumatoid arthritis.¹⁴

42. The papers touched on:

42.1. Hormonal and neuronal mechanisms in immune system regulation:

¹¹ RAAF internal memo dated 28 October 1983, titled 'STI Radio General/26: Identification and Disposal of Transformers and Capacitors Containing Polychlorinated Biphenyl (PCB) compounds.' RMA ID S1.12

¹² Agency for Toxic Substances and Disease Registry (ATSDR) (USA) 2011, Division of Toxicology and Environmental Medicine. Detailed Data for 2011 Priority List of Hazardous Substances.

¹³ Regardless of the way in which articles were referenced by the participants in the review, the Council has provided the full citations of articles in footnotes in accordance with the 'Author-date' system described in the Commonwealth of Australia 2002, *Style manual*, 6th edn, John Wiley & Sons Australia Ltd, pp. 187-232.

¹⁴ Papers relied on by the Applicant as provided in his submission to RMA of 20 July 2006 (RMA ID S 1:12) have not been separately identified, and unless listed at Appendix A, were not included in the Preliminary Pool.

- **Eskandari et al, 2003**¹⁵ from which he noted the authors saying:

Perturbations of these regulatory systems could potentially lead to either over activation of immune responses and inflammatory disease, or over suppression of the immune system and increased susceptibility to infectious disease.

42.2. Immune and inflammatory responses to Polychlorinated biphenyl (PCB)

- **Kwon et al, 2002**¹⁶ about which the Applicant noted the authors saying:

Mast cells are critical for initiating innate immune and inflammatory responses by releasing a number of pro-inflammatory mediators. In this report, the effects of polychlorinated biphenyl (PCB) on the expression of cyclooxygenase-2 and pro-inflammatory cytokines such as interleukin-1 beta (IL-1beta), IL-6 and tumor necrosis factor (TNF)-alpha in human leukemic mast cell line were investigated.

Stimulating the cells with PCB activated NF-kappaB. However, pre-treating them with a NF-kappaB pathway inhibitor, pyrrolidine dithiocarbamate, suppressed PCB-induced NF-kappaB activation. This suggests that PCB induces cyclooxygenase-2 and pro-inflammatory cytokine expression, and that this induction occurs through NF-kappaB.

42.3. PCB-induced superoxide anion (O₂⁻) production

- **Tithof et al 1996**¹⁷ about which the Applicant noted the authors saying:

The release of arachidonic acid by inflammatory cells upon exposure to PCBs may represent an additional mechanism of PCB-induced toxicity. Arachidonic acid and its metabolites have been implicated in a variety of inflammatory disease states including septicemia (36, 37), rheumatoid arthritis (38), and systemic lupus erythematosus (39), as well as diseases characterized by cellular transformation such as hypertrophic transformation of the skin (40) and colon cancer (41).

42.4. Evaluation of the effects of PCBs on human health

- **Melius et al**¹⁸ about which the Applicant noted the authors saying:

¹⁵ Eskandari, F. Webster. JI. Sternberg, EM. 2003, 'Neural immune pathways and their connection to inflammatory diseases', *Arthritis Res Ther*, vol. 5, no. 6, pp.251-65. RMA ID 47835

¹⁶ Kwon, O. Lee, E. Moon, T.C. Jung, H. Lin, C.X. Nam, K.S. Baek, S.H. Min, H.K. & Chang, H.W. 2002, 'Expression of cyclooxygenase-2 and pro-inflammatory cytokines induced by 2,2',4',5,5'-hexachlorobiphenyl (PCB 153) in human mast cells requires NF-kB activation', *Biol Pharm Bull*, vol. 25, no. 9, pp.1165-8. RMA ID 47836

¹⁷ Tithof, P.K. Schiamburg, E. Peters-Golden, M. Ganey. P.E. 1996 'Phospholipase A2 is involved in the mechanism of activation of neutrophils by polychlorinated biphenyls', *Environ Health Perspect*. Vo. 104, No. 1, pp. 52–58. RMA ID 47837

¹⁸ Melius, JM . Steele, G. Sanderson, LM. Faroon, ON.1996, 'Proceedings of the Expert Panel Workshop to Evaluate the Public Health Implications for the Treatment and Disposal of Polychlorinated Biphenyls - Contaminated Waste', *Chapter 2 – Expert Panel Report*, Agency for Toxic Substances and Disease Registry (ATSDR)

Recent data show that the immune system of monkeys appears to be one of the most sensitive indicators of PCB exposure. Monkeys exposed to very low levels (5 µg/kg/day of Aroclor 1254) had a significant decrease in IgG and IgM immunoglobulin levels in primary response to challenge with sheep red blood cells (Tryphonas et al. 1989). Also, elevation of complement (CH50 level) in monkeys exposed to Aroclor 1254 has been observed. Elevated complement levels have been associated with rheumatoid arthritis and systemic lupus erythematosus in humans.

Neurologic effects of PCB exposure appear to be more important for children who are exposed as fetuses rather than as infants, or for adults who have been occupationally exposed.

and

To fully understand the impact of PCBs on the immune system, it may be appropriate to evaluate how much of a reduction in IgG and IgM constitutes an adverse health effect. In addition, it may be appropriate to evaluate indicators of auto-immune dysfunction (for example, rheumatoid arthritis and systemic lupus erythematosus) in humans.

42.5. Serum immunoglobulins and the risk of rheumatoid arthritis:

- **Aho et al, 1997**¹⁹ about which the Applicant noted the authors saying:

RA is associated with several autoantibodies specific enough to serve as diagnostic and prognostic markers of the disease. ..Of these, rheumatoid factor (RF) and the two closely related antibodies, antikeratin antibody (AKA) and antiperinuclear factor, have been shown to precede the onset of clinical RA.

42.6. Mass exposures to PCBs in Taiwan and Japan:

- **Tsuji et al 1999**²⁰ from which he noted:

Autoantibodies were present in some patients of Yusho; 45.6% for antinuclear antibody, 12.7% for rheumatoid factor and 11.1% for thyroglobulin antibody.

- **Guo et al 1999**²¹ from which he noted:

Although autoimmune-mediated arthritis cannot be ruled out, immunologic evaluation in the early years after exposure actually showed suppressed serum IgA and IgM and decreased helper-T cells, which had returned to normal levels 3 years later.

http://web.archive.org/web/20041025015407/http://www.atsdr.cdc.gov/HAC/PCB/b_pcb_c2.html
Date accessed not provided. (Included in the Applicant's submission to the RMA dated 20 July 2006, RMA ID S1.12)

¹⁹ Aho, K. Heliövaara, M. Knekt, P. Reunanen, A. Aromaa, A. Leino, A. Kurk, I.P. Heikkilä, R. Palosuo, T & Iosuo, T. 1997, 'Serum immunoglobulins and the risk of rheumatoid arthritis', *Ann Rheum Dis*, vol. 56, no. 6, pp.351-6. RMA ID 47840

²⁰ Tsuji, H. Hirahashi, T. Ogata, H. & Fujishima, M. 1999, 'Serum immunoglobulin concentrations and autoantibodies in patients with Yusho', *Fukouka Igaku Zasshi*, vol. 90, no. 5, pp.147-9 (Abstract) RMA ID 47834

²¹ Guo, Y.L. Yu, M.L. Hsu, C.C. Rogan, W.J. 1999, 'Chloracene, Goiter, Arthritis, and Anemia after Polychlorinated Biphenyl Poisoning: 14-Year Follow-Up of the Taiwan Yucheng Cohort', *Environmental Health Perspectives*, vol. 107, no. 9. RMA ID 18242

and

Immune-mediated inflammatory reaction and arthritis can be caused by chemical exposure.

In his oral submission, the Applicant commented that in the:

Yucheng and Yushi exposures ...there was 12.7 per cent .. rheumatoid factor [in] exposed people.

...when you look at those Yucheng, Yushi outcomes they just say arthritis, whether it be treated with medication or surgical or whatever. So they're not very specific but they do mention that the men have herniated discs, so it's there.

42.7. The effect of chemicals on the immune system

- **Environmental Research Foundation**, Statement on Immune Toxins²² from which the Applicant noted:

Experimental lab studies demonstrate that certain synthetic chemicals affect the immune system ... for example, many chlorine-containing compounds.

...from experiments on laboratory animals, it is known with certainty that many classes of common chemicals can change the immune system and can cause hypersensitivity and autoimmune diseases. In humans, hypersensitivity is often expressed as an allergic reaction. Autoimmune diseases include diabetes, multiple sclerosis, rheumatoid arthritis, lupus, and a dozen other diseases.

42.8. Vitamin A deficiency in autoimmune diseases:

- **Plapp, F, 2002**²³ from which he cites the author as saying:

PCB mixtures have been found to decrease levels of retinol (Vitamin A) in the liver of rats (Innami et al., 1976; Kato et al., 1978; Hudecova et al., 1979), rabbits (Villeneuve et al., 1971a), and in the plasma of pigs (Guoth et al., 1984).

- **Plapp, F, 2003**²⁴ from which he cites the author as saying:

Exposure to any of a wide range of environmental chemicals (ECs) causes environmental illness (EI) in both humans and wildlife. ECs include pesticides such as insecticides and herbicides and non-pesticides such as PCBs and dioxin. Vitamin A (retinol), and vitamin A hormone (retinoic acid), must be present in our bodies for normal functioning of the immune system and for the protein synthesis processes

²² Environmental Research Foundation, 'Statement on Immune Toxins', <http://wgbis.ces.iisc.ernet.in/envis/doc97html/miscrw51.html> (Included with the Applicant's submission to the RMA of 20 July 2006, RMA ID S1.12)

²³ Plapp, F. 2002, The role of vitamin A deficiency in autoimmune diseases including Gulf War Syndrome, chronic fatigue syndrome, multiple chemical sensitivity and fibromyalgia. 25 11 2002 [web page] http://www.prohealth.com/library/showarticle.cfm?id=4089&t=CFIDS_FM (Included with the Applicant's submission to the RMA of 20 July 2006, RMA ID S1.12)

²⁴ Plapp, F, 2003, Perilous pathways - environmental Chemicals and environmental illness: a major role for vitamin A. [Web page] <http://www.westonaprice.org/environmental-toxins/perilous-pathways>. (Included with the Applicant's submission to the RMA of 20 July 2006, RMA ID S1.12)

involved in reproduction. Lack of retinoic acid, the hormone form of vitamin A, characterizes most human autoimmune diseases.

The question is whether these diseases are caused by lack of vitamin A or whether lack of vitamin A is caused by autoimmune disease? It turns out that lack of vitamin A is a precondition for the development of many if not all autoimmune diseases. There are three reasons for a lack of vitamin A in humans. One reason is genetic, a second is dietary, and the third involves exposure to ECs. The third reason, exposure to many ECs, is also associated with development of autoimmune illness. When vitamin synthesis is low, there is less vitamin A hormone available to activate the immune system. The result is increased frequency of autoimmune diseases.

- **Diseases Data Base - Anisocytosis**²⁵ which the Applicant cites as recording that anisocytosis is a feature of Vitamin A deficiency.

In relation to anisocytosis, the applicant supplied a copy of his RAAF medical record for October 1981, about which he comments, that:

After 18 months of persistent flu like conditions, burning feeling throughout and feeling run down [a] blood sample was taken showing slight **anisocytosis** and lymphocytosis.

42.9. PCB exposure and rheumatoid arthritis:

- **Lee et al 2007**²⁶ from which he cited the authors saying:

...dioxin-like polychlorinated biphenyls (PCBs) or nondioxin-like PCBs were positively associated with arthritis in women.

and

For subtypes of arthritis, respectively, RA was more strongly associated with PCBs than was OA.

and the authors' conclusions that:

The possibility that background exposure to PCBs may be involved in pathogenesis of arthritis, especially RA, in women should be investigated in prospective studies.

- **The La Salle Electrical Utilities Company Morbidity Study II 2004**²⁷

in respect of which the Applicant commented:

Interestingly, Rheumatoid Arthritis percentages in this report appear to be around 8-14% and is consistent with the reported 12.7% Rheumatoid Factor in Yusho poisoning.

²⁵ Diseases Data Base – Anisocytosis (Included with the Applicant's submission to the RMA of 20 July 2006, RMA ID S1.12)

²⁶ Lee, D.H. Steffes, M. Jacobs, D.R. Jr. 2007, 'Positive associations of serum contraction of polychlorinated biphenyls or organochlorine pesticides with self-reported arthritis, especially rheumatoid type, in women', *Environmental Health Perspectives*, vol. 115, no.6, pp. 883-888. RMA ID 46247

²⁷ Illinois Department of Public Health and the University of Chicago 2004, *The La Salle Electrical Utilities Company Morbidity II Study : Final Report*. The Illinois Department Of Public Health and the University of Illinois at Chicago, School Of Public Health. RMA ID 47839

In his oral submission, the Applicant claimed that:

...about 12.7 for women and I think it was about 10.9 for men and if you have a look at the La Salle [study] how many people ... 20 out of 400 and a bit more for the females, and actually how it relates roughly to that, about one per cent less.

42.10. Penetration Capabilities of PCBs

The Applicant also provided with his submission South Australian Government Workplace Safety guidelines²⁸ which detail safety precautions recommended for workers such as avoiding skin contact with PCBs through wearing special protective gloves.

The Applicant claimed that

PCBs are able to penetrate ordinary plastic or rubber gloves within a few minutes

42.11. Also provided by the Applicant were references to various websites on the penetrative properties of oils containing PCBs and on the toxic properties of PCBs and these are listed at **Appendix C**.

The Applicant did not comment on the Proposed Scope of the Review and Proposed Pool of Information decisions

43. The Applicant made no comment on the Council's proposed scope of review and proposed pool of information decisions, other than to refer to the new information noted above.

THE COMMISSIONS' SUBMISSIONS

44. The Commissions made a written submission dated 7 February 2011. A Medical Officer with the Department of Veterans' Affairs, representing the Commissions, made an oral submission complementing the Commissions' written submission at the Council's meeting on 18 July 2012.²⁹
45. The Commissions submitted that the information that was available to the RMA at the relevant times on mineral oil and rheumatoid arthritis, was '...limited in both extent and quality'.
46. The Commissions identified four reports on original studies concerning mineral oils and rheumatoid arthritis, and an additional commentary on one of the studies.
47. Of the original studies **concerning mineral oils**, the Commissions cited:

²⁸ South Australian Government 2000, 'Safeguards: GS 28 Polychlorinated Biphenyls (PCBS)', Dept Admin & Information Services. http://www.safework.sa.gov.au/uploaded_files/gs28i.pdf (Included with the Applicant's submission to the RMA of 20 July 2006, RMA ID S1.12)

²⁹ The information upon which the Commissions relied, being information which the RMA advised was available to (before) the RMA at the relevant times, is listed in Appendix A. RMA ID 48814

47.1. **Sverdrup et al 2005**³⁰ in respect of which the Commissions submitted the authors:

...undertook a population-based case-control study in Sweden to examine the association between occupational mineral oil exposure and rheumatoid arthritis.

The Commissions submitted that the study was:

...the best of the available evidence...

which

...indicates a small increased risk from occupational exposure to mineral oils that just reaches statistical significance in seropositive men.

47.2. **Miller 2006**³¹, in respect of which the Commissions submitted the authors:

...provided an expert opinion and commentary on the Sverdrup et al (2005) study, reporting that there was low statistical power, possible recall bias due to the retrospective nature of questionnaire surveys and no dose-response relationship in the study.

47.3. **Reckner Olsson et al 2004**³², which the Commissions submitted was a case-control study that:

...examined the association between occupational exposures and rheumatoid arthritis. Exposures to asphalt, asbestos, organic dust, vibrations, mineral dust, crops/forage, mineral oil, man made mineral fibres, fertilisers, grain, farm animals and pesticides were examined.

The Commissions submitted that the authors:

...found a positive association but this did not reach statistical significance.

47.4. **Lundberg et al 1994**³³ in which the Commissions submitted the authors:

...performed a retrospective cohort study in Sweden, examining the association between rheumatoid arthritis and occupational exposures in general. Exposures to

³⁰ Sverdrup, B. Kallberg, H. Bengtsson, C. Lundberg, I. Padyukov, L. Alfredsson, L. & Klareskog, L. 2005, 'Association between occupational exposure to mineral oil and rheumatoid arthritis: results from the Swedish EIRA case-control study', *Arthritis Research & Therapy*, vol. 7, pp. R1296-R1303. RMA ID 36816

³¹ Miller, F.W. 2006, 'Is occupational exposure to mineral oil a risk factor for rheumatoid arthritis?' *Nature Clinical Practice*, vol. 2, no. 3, pp.130-1. RMA ID 46581

³² Reckner Olsson, A. Skogh, T. Axelson. O. & Wingren, G. 2004, 'Occupations and exposures in the work environment as determinants for rheumatoid arthritis', *Occup Environ Med*, vol. 61, pp.233-8. RMA ID 30451

³³ Lundberg, I. Alfredsson, L. Plato, N. Sverdrup, B. Klareskog, L. & Lkeinau, S. 1994, 'Occupation, occupational exposure to chemicals and rheumatological disease', *Scand J Rheumatol*, vol. 23, pp. 305-310. RMA ID 5681

mineral oils, cutting oils, polychlorinated biphenyls, petrol, pesticides, aliphatic hydrocarbons, organic solvents, heavy metals, asbestos and engine exhaust were examined.

The Commissions submitted that the study found a:

relative risk of hospitalisation due to rheumatoid arthritis of 1.0 (95% confidence interval, 0.8 to 1.3) in men with exposure to mineral oils or cutting oils. In women the relative risk was 1.1 (95% confidence interval, 0.6 to 2.1).

47.5. **Sverdrup, Klareskog & Keleinau 1998**³⁴ in respect of which the Commissions submitted, the authors:

...performed an animal experimental study on the induction of acute arthritis in Dark Agouti rats after exposure to different chemicals.

The Commissions submitted that this:

...solitary animal study examined the association between mineral oil and transient acute arthritis and showed some positive findings but this was only for intradermal injections and not for oral exposure. Further, this finding was not accompanied by the presence of auto antibodies.

48. The Commissions concluded in relation to mineral oils that:

Mineral oil exposure was not well specified or quantified in the available studies. The studies lacked statistical power. The limited evidence available did not establish a dose-response effect. The studies, being retrospective, were subject to recall bias. Potential confounding by other exposures was not well addressed.

The Commissions considers that the available evidence is just sufficient to indicate that seropositive rheumatoid arthritis (only) may be caused by mineral oil exposure.

But that

the evidence falls well short of meeting the balance of probabilities standard of proof.

49. From the information that was available to the RMA at the relevant times, the Commissions identified three studies investigating the possible association between PCBs and rheumatoid arthritis and one further study with results for PCB exposure and arthritis (but not specifically rheumatoid arthritis).

50. Of the studies **concerning PCBs**, the Commissions cited:

50.1. **Lee et al 2007**³⁵, in respect of which the Commissions submitted the authors:

³⁴ Sverdrup, B. Klareskog, L. & Keleinau, S. 1998, 'Common commercial cosmetic products induce arthritis in the DA rat', *Environ Health Perspect*, vol.106, no.1, pp.27-32. RMA ID 48785

...used data from a National Health and Nutrition Examination Survey (NHANES 1999-2002) in the US to investigate cross-sectional associations between serum PCB concentrations and self-reported arthritis. The NHANES survey had included measurement of background levels of persistent organic pollutants (POPs), including PCBs.

The Commissions submitted that the study found:

...an association between increased background PCB level and self-reported rheumatoid arthritis, with odds ratios in females of 1.0, 7.6, 6.1 and 8.5 across quartiles for dioxin-like PCBs and 1.0, 2.2, 4.4 and 5.4 across quartiles for nondioxin-like PCBs.

and that

...the elevated risks were statistically significant [for women].

However

...for males the study did not find a significant association of PCBs with arthritis in general and did not provide results for the subcategory of rheumatoid arthritis.

The Commissions concluded that this study:

...reported a positive association between increasing background exposure levels of PCBs and rheumatoid arthritis in women. This cross-sectional study was not able to examine the temporal relationship between exposure and disease. A plausible biological mechanism is not apparent and it is difficult to reconcile the results of this study with negative results in much more highly exposed subjects..

50.2. **Lundberg et al 1994**³⁶, which the Commissions submitted included results for exposure to mineral oils (see above) also:

examined rheumatoid arthritis risk from exposure to PCBs.

...diagnosis was based on hospital discharge reports and exposure was derived from a job exposure matrix.

³⁵ Lee, D.H. Steffes, M. Jacobs, D.R. Jr. 2007, 'Positive associations of serum contraction of polychlorinated biphenyls or organochlorine pesticides with self-reported arthritis, especially rheumatoid type, in women', *Environmental Health Perspectives*, vol. 115, no.6, pp. 883-888. RMA ID 46247

³⁶ Lundberg, I. Alfredsson, L. Plato, N. Sverdrup, B. Klareskog, L. & Lkeinau, S. 1994, 'Occupation, occupational exposure to chemicals and rheumatological disease', *Scand J Rheumatol*, vol. 23, pp. 305-310. RMA ID 5681

...there were 25 men (and no women) with exposure to PCBs and rheumatoid arthritis. The relative risk in those men was 1.1 (95% confidence interval of 0.7 to 1.7).

50.3. **Guo et al 1999**³⁷, which the Commissions submitted was a study of the exposure of subjects of 'a mass poisoning event, involving a cohort of 2000 people who had ingested contaminated rice cooking oil in 1979'.

The Commissions submitted that the study found:

...an association between PCB exposure and unspecified arthritis, with an odds ratio of 4.1 in males (95% confidence interval, 1.8 to 11.2) and 1.3 in females (95% confidence interval, 0.8 to 2.3)³⁸

The Commissions submitted that, 'The presence of chloracne is a marker for significant PCB exposure' and that the study found:

The lifetime prevalence of arthritis in men who had had chloracne was 11.8% compared with 6.8% in men without chloracne (difference not statistically significant). For women the lifetime prevalence of arthritis was 8% in those with chloracne and also 8% in those without.

50.4. The La Salle Electrical Utilities Company Morbidity Study II 2004³⁹ which the Commissions submitted was a:

...retrospective general morbidity survey of the La Salle electrical utilities company in Illinois, with diagnosis based on client's self report and with estimated levels of PCB exposure.

and found

...a self reported history of rheumatoid arthritis in 46 out of 330 males and 20 out of 252 females.

The results included a Cox regression analysis relating years of exposure to time on onset of disease. This gave a hazard ratio that represented the proportional increased hazard for each additional five years worked at the site. In men the hazard ratio was 0.72 (95% confidence interval 0.38 to 1.35) and in women it was 1.08 (95% confidence interval of 0.89 to 1.35).

51. In his oral submission, the Commissions' representative submitted that given their wide use up until the 1970s, most people are exposed to PCBs to some extent, at

³⁷ Guo, Y.L. Yu, M.L. Hsu, C.C. Rogan, W.J. 1999, 'Chloracne, Goiter, Arthritis, and Anemia after Polychlorinated Biphenyl Poisoning: 14-Year Follow-Up of the Taiwan Yucheng Cohort', *Environmental Health Perspectives*, vol. 107, no. 9. RMA ID 18242

³⁸ Table 2. Prevalence (%) of reported diseases ever diagnosed by a physician in Yucheng and control groups in Taiwan, 1993, Guo et al 1999, p. 717

³⁹ Illinois Department of Public Health and the University of Chicago 2004, *The La Salle Electrical Utilities Company Morbidity II Study : Final Report*. The Illinois Department Of Public Health and the University of Illinois at Chicago, School Of Public Health. RMA ID 47839

low levels through exposure to environmental pollution working its way into the food chain.

[PCBs have] been used or been contaminants in products like inks and lubricants, waxes, adhesives, dyes, pesticides, insulating materials, paints and other surface coatings, and, in that group, obviously, is mineral oils as well, at least historically. They're not in mineral oils today.

52. The Commissions concluded in relation to **PCBs** that the available information:

...leaves open the possibility of an association between background levels of PCB exposure and rheumatoid arthritis in women, but the evidence is insufficient to indicate a causal association. The evidence does not indicate any association in men and does not establish on the balance of probabilities that rheumatoid arthritis can be caused or worsened by PCB exposure.

Commissions' comments on the Proposed Scope of the Review and Proposed Pool of Information decisions

53. The Commissions sought no amendment to the Council's proposed scope of review.

54. The Commissions did not propose any alteration to the Council's proposed decision on the pool of information.

REASONS FOR THE COUNCIL'S DECISION

The Council's Task

55. In conducting a review the Council follows a two-step process. The Council first identified the pool of information, i.e. it identified from all the information that was available to (before) the RMA at the relevant times the sound medical-scientific evidence (as that term is defined in section 5AB(2) of the VEA (see [8] above)) which in its view 'touches on' (i.e. is relevant to) the issue of whether rheumatoid arthritis can be related to service.

56. The second step required the Council to determine whether:

56.1. there is sound medical-scientific evidence in the pool that indicates ('points to' as opposed to merely 'leaves open'⁴⁰) the relevant possibility ie whether exposure to mineral oils containing PCBs or acute PCB exposure (if found to exist in a particular case) could provide a link or element in a reasonable hypothesis connecting

⁴⁰ See full Federal Court decision at [49] per Branson J.

rheumatoid arthritis or death from rheumatoid arthritis to relevant service.⁴² 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.'⁴³

- 56.2. on the sound medical-scientific evidence in the pool exposure to mineral oils containing PCBs or acute PCB exposure (if found to exist in a particular case) could provide a relevant connection between rheumatoid arthritis or death from rheumatoid arthritis and relevant service according to a standard of satisfaction 'on the balance of probabilities', or as being more probable than not.
57. In these Reasons the association for both the reasonable hypothesis test (at 56.1] and the balance of probabilities test at [56.2]) are respectively referred to as the 'relevant association'.
58. 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 and the Commissions referable to the matters within the scope of review.
59. In forming its judgement on whether there is sound medical-scientific evidence that indicates (ie 'points to' as opposed to merely 'leaving open') the relevant association, the Council was conscious that the reasonable hypothesis test is 'a test of possibility'⁴⁵ and 'an unusually light burden.'⁴⁶ If the reasonable hypothesis test was found not to be satisfied, the balance of probabilities test necessarily could not be met.

Scope of Review

60. The Council's final view on the scope of the review was that it should comprise the scope which the Council had identified on a preliminary basis in respect of exposure to mineral oils containing PCBs or acute PCB exposure (see [20]).

⁴¹ Relevant service here refers to operational, peacekeeping and hazardous service, British nuclear test defence service, and warlike or non-warlike service as those terms are defined in the VEA and the MRCA.

⁴² 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 [29].

⁴³ 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 [33].

⁴⁴ Relevant service here refers to eligible war service (other than operational service), defence service (other than hazardous service and British nuclear test defence service) and peacetime service as those terms are defined in the VEA and the MRCA.

⁴⁵ See full Federal Court decision at [49] citing with approval Spigelman CJ in the New South Wales Court of Appeal decision at [111].

⁴⁶ See full Federal Court decision at [55] per Branson J.

Pool of Information

61. The Council's final decision on the pool of information was that it should comprise the sound medical-scientific evidence it had identified on a preliminary basis as listed in Appendix A.
62. 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, could or should be in the pool.
63. As mentioned above, the Council noted the Applicant's references to and submissions concerning information which was not available to (not before) the RMA (see Appendix D). As mentioned above, the Council in its review was unable to (and so did not) consider information which was not available to (not before) the RMA at the relevant times.

THE COUNCIL'S ANALYSIS OF THE INFORMATION BEFORE THE RMA

Preliminary comment on rheumatoid arthritis

64. Set out below are some general and introductory comments on rheumatoid arthritis and the Council's analysis of the information in the pool.
65. Statements of Principles Nos. 68 and 69 of 2008 define rheumatoid arthritis as:
 - a multisystem disease persisting for a continuous period of at least six weeks, characterised by inflammatory synovitis, usually involving peripheral joints in a symmetrical distribution, sometimes including cartilage damage and bone erosions and changes in joint integrity, in addition to systemic manifestations. This definition excludes juvenile rheumatoid arthritis and non-rheumatoid inflammatory arthritis, including post-infectious and reactive arthropathies.
66. The current relevant factor in Statement of Principles No. 68 of 2008 (the reasonable hypothesis statement of principles) is:

for seropositive rheumatoid arthritis only, inhaling, ingesting or having cutaneous contact with mineral oil, for a cumulative period of at least 2500 hours within a continuous period of ten years before the clinical worsening of rheumatoid arthritis, and where exposure to mineral oil has ceased, the clinical onset/worsening has occurred within ten years of cessation

 - 'mineral oil' means complex mixtures of straight chain and polycyclic aromatic hydrocarbons distilled from crude oil, and includes cutting oil, motor and lubricating oils, form oil, hydraulic oil and molten bitumen;
 - 'seropositive rheumatoid arthritis' means rheumatoid arthritis accompanied by serological evidence of elevated levels of rheumatoid factor or the presence of anti-cyclic-citrullinated peptide antibodies;

67. There are no factors in Statement of Principles No 69 of 2008 (the balance of probabilities statement of principles) for exposure to mineral oils or PCBs. There is no factor in either Statement of Principles which provides for a single acute large dose of mineral oil or of PCBs.
68. The Council noted that PCBs are heavily chlorinated substances (i.e. there are several chlorine containing rings in their chemical structure). A number of reports discuss differentially chlorinated mixtures of PCBs and dioxins (Polychlorinated dibenzofurans (PCDFs) and polychlorinated dibenzodioxins (PCDDs). Commercial or trade names for PCB mixtures, such as Aroclor and Kanechlor, sometimes appear in the literature in the pool.
69. The Council also noted mineral oils themselves do not contain PCBs but in the past, were either intentionally combined with PCBs or sometimes were found to be unintentionally contaminated with PCBs, in particular transformer oils.⁴⁷
70. In the literature, these oils are variously referred to as, mineral oils, hydraulic oils or cutting oils, without specific reference to PCB content.
71. The Council noted that production of PCBs have been banned in most countries since the 1970's⁴⁸ and have been phased out gradually, although they still existed in industrial uses when the subjects of studies were exposed to the various types of oils.⁴⁹
72. The mineral oils described in the existing factor for mineral oil exposure (in the reasonable hypothesis Statement of Principles) would necessarily include mineral oils containing PCBs. The existing factor accommodates long term or chronic exposure of 2500 hours or more, but does not address a single or acute exposure to either mineral oils or PCBs. Neither the Applicant nor the Commissions contended that the exposures covered by the existing factor in the reasonable hypothesis statement of principles should not be accommodated or should be reduced; the Applicant contending that the factor should be expanded to include

⁴⁷ 'Transformer oil or insulating oil is usually a highly-refined mineral oil that is stable at high temperatures and has excellent electrical insulating properties. It is used in oil-filled transformers, some types of high voltage capacitors, fluorescent lamp ballasts, and some types of high voltage switches and circuit breakers. Its functions are to insulate, suppress corona and arcing, and to serve as a coolant.' From Wikipedia, the free encyclopedia, (updated on 24 November 2012) Transformer oil, Retrieved, 6 December 2012, http://en.wikipedia.org/wiki/Transformer_oil

⁴⁸ Production of PCBs in the United States was suspended in 1977: *See for example, Emmett EA, Maroni M, Schmith JM, Levin BK, Jefferys J 1988*, 'Studies of transformer repair workers exposed to PCBs: 1. Study design, PCB concentrations, questionnaire, and clinical examination results', *Am J Ind Med*, vol. 13, pp. 415. RMA ID 47832

⁴⁹ 'In Australia there is an ongoing management plan to safely dispose of PCBs from transformers and other equipment. Between 1996 and 2001, for example in Queensland, 8200 tonnes of PCBs were treated from oils in capacitors and other equipment, of which 1200 tonnes were in transformers'. **Environment Protection and Heritage Council 2002**, Report of the review of the ANZECC Polychlorinated Biphenyls Management Plan, p30. Retrieved 6 December 2012. http://www.ephc.gov.au/sites/default/files/CMgt_Rev_Report_of_the_Review_of_ANZECC_Polychlorinated_Biphenyls_Management%20Plan_200209.pdf

acute exposures to mineral oils and particularly acute exposures to PCBs; the Commissions contending for no amendment.

73. The Council considered that animal studies may sometimes support the biological plausibility of an association. However results from animal studies are not generalisable to humans and are at best used as initial research that may indicate a need for further studies on human subjects.
74. Laboratory based studies of human cells are used in medical research for exploring potential pathological mechanisms, such as examining inflammatory responses to toxins. Processes occurring at the cellular level can be misleading as many other processes contribute to human health effects. Whilst such studies may generate further research, only some would lead to further discoveries and they can often produce a range of conflicting results. Such studies can be material which epidemiologists would consider appropriate to take into account, but the weight to be attached to their results when considering causes of diseases varies, and is generally relatively low.

DOES THE SOUND MEDICAL-SCIENTIFIC EVIDENCE 'POINT TO' OR 'LEAVE OPEN' THE RELEVANT ASSOCIATION

75. As mentioned above, having settled the pool of information, the second question for the Council to consider was whether there is sound medical-scientific evidence in the pool that indicates ('points to') a contended factor in the scope of the review as a link or element in a reasonable hypothesis connecting rheumatoid arthritis to relevant service (see 56.1] and footnotes), and if so, whether the relevant association exists on the balance of probabilities (see 56.2] and footnotes).
76. The only basis upon which the Council can review the contents of a Statement of Principles is by reviewing all the information that was available to (before) the RMA at the relevant times, in order to ascertain whether there was sound medical-scientific evidence upon which the RMA could have relied to amend either or both of the Statements of Principles.
77. The Council considered all the articles in the pool. In these Reasons, the Council focused its discussion upon its analysis of those articles which it considered most pertinent to the scope of review.
78. 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 rheumatoid arthritis) and the matters within the scope of the review.

THE COUNCIL'S ANALYSIS OF THE INFORMATION IT CONSIDERED MOST IMPORTANT AS BEING POTENTIALLY REFERABLE TO THE CONTENTED FACTOR

Original Studies

Reckner Olsson, A. Skogh, T. & Wingren, G. 2000, 'Occupational determinants for rheumatoid arthritis'. *Scandinavian Journal of Work, Environment and Health*, vol. 26, no. 3, pp. 243-9. **RMA ID 27665**

79. This was a case control study of occupational exposures to evaluate possible occupational determinants of importance for rheumatoid arthritis based on information on lifetime occupational exposure history.
80. Cases of rheumatoid arthritis were identified retrospectively from 1980 to 1995 at the University Hospital in Linköping, Sweden. All cases met the American Rheumatism Association (ARA)⁵⁰ 1987 criteria. The study comprised 422 cases (among which 79% were rheumatoid factor (RF) seropositive) and 859 randomly selected referents.⁵¹ Exposure data were collected through a postal questionnaire. Latency of 20 years, based on previously published reports regarding latency between the first exposure and onset of rheumatoid arthritis⁵² was used in the analysis to enable comparison with earlier findings. To be regarded as exposed, subjects were required to have had a minimum duration of exposure of 6 months. All analyses had been adjusted for age, smoking and occupational factors identified.
81. The only overall significant finding was for asphalters, for whom there was found to be a significant occupational correlation (OR 14.0, 5% CI, 1.2-799.0 without the latency requirement). The authors noted that contact with asphalt involves multiple exposures, such as coal tar, crude oils, polycyclic aromatic hydrocarbons and bitumen fumes, and asphalters are also exposed, for example to ultraviolet radiation, mineral dust and engine exhaust.⁵³
82. When only seropositive rheumatoid arthritis was analysed, results were similar to those for combined rheumatoid arthritis.
83. Some limitations were acknowledged by the authors as to potential 'non-response bias'.⁵⁴

Council's Comments

84. The Council noted that while asphalt and hydraulic oil as mineral oils may be contaminated with PCBs this issue is not addressed in the paper. The Council

⁵⁰ The American Rheumatism Association (ARA), changed its name to the American College of Rheumatology (ACR) in 1988.

⁵¹ p.244

⁵² p.244

⁵³ p.248

⁵⁴ p.247

noted the finding that only asphalters had a significantly increased risk. Although bitumen is included in the definition of mineral oils in the current statement of principles, there were many other possible exposures within the asphalt which could have confounded this association. The confidence interval was very wide, the numbers exposed to asphalt in the study were very small and the number of cases exposed was very low (four asphalter cases, one exposed referent in the stratified analysis,⁵⁵ seven exposed cases, one referent exposed to asphalt in the occupational exposures analysis).⁵⁶

85. The Council considered that this study leaves open the relevant association with mineral oils contaminated with PCBs, and does not touch on a relevant association with PCB exposure.

Reckner Olsson, A. Skogh, T. Axelson, O. & Wingren, G. 2004, 'Occupations and exposures in the work environment as determinants for rheumatoid arthritis', *Occup Environ Med*, vol. 61, pp.233-8. **RMA ID 30451**.

86. This was a second case-referent study led by Reckner Olsson to further explore associations between rheumatoid arthritis and several occupational categories previously associated with rheumatoid arthritis. Researchers used postal questionnaires to gather lifelong occupational history for 293 incident diagnosed rheumatoid arthritis cases, recruited from ten rheumatology units, and 1346 population controls. Pooled analyses were also performed with data from the previous study, making a combined total of 713 cases and 2204 referents.⁵⁷
87. Data were stratified into three age categories from 16 to 75 years and further stratified into three smoking levels. All cases met the American Rheumatism Association (ARA) 1987 criteria.
88. Specific occupational exposures examined by multivariate analyses were asphalt, asbestos, organic dust, vibrations, mineral dust crops and or forage, mineral oils, fertilisers, grain, farm animals, pesticides. After adjustment for age and smoking, vibration was the only occupational factor found to be significantly associated with rheumatoid arthritis.⁵⁸
89. No statistically significant increased risk was found for mineral oil for the incident cases (OR 1.8, 95% CI 0.6-5.5)⁵⁹, nor for the pooled cases (OR 1.2, 95% CI 0.7-2.1).⁶⁰ This did not vary much with the duration of exposure.⁶¹ Notably, these calculations required a latency of 20 years between the exposure and onset.⁶²

⁵⁵ p.245

⁵⁶ Table 3, p.246

⁵⁷ p.234 The prevalent cases and referents are the participants reported in Reckner et al (2000)

⁵⁸ see Table 3 p. 236

⁵⁹ If the odds ratio crosses or includes the value of 1, no statistically increased risk is demonstrated.

⁶⁰ Table 3 p.236

90. Limitations noted by the authors were that fewer participants in the comparison respondents were of lower socioeconomic status, which the authors considered may indicate a loss of subjects with occupational history of more hazardous exposures.
91. They also speculated on the possibility that persons with symptoms of rheumatoid arthritis may have changed from occupations with more strenuous work tasks prior to being diagnosed (and therefore potentially reducing the number of exposed cases from the analysis).⁶³

Council's Comments

92. The Council noted that reported exposure to mineral oil was not specific for PCBs, i.e. there was no information about whether the mineral oils contained PCBs. The effect size for exposure to mineral oils was not statistically significant and there was no correlation between extent of exposure and effects.
93. The Council considered the study to be only marginally relevant to a possible association between rheumatoid arthritis and mineral oil, leaving open the relevant association. The study did not touch on the relevant association with exposure to PCBs.

Lundberg, I. Alfredsson, L. Plato, N. Sverdrup, B. Klareskog, L. & Lkeinau, S. 1994, 'Occupation, occupational exposure to chemicals and rheumatological disease', *Scand J Rheumatol*, vol. 23, pp. 305-310. **RMA ID 5681.**

94. This large, population-based cohort study, looked for potential associations between occupations or occupational exposures and rheumatoid arthritis. All Swedish adults born between 1905 and 1945 who had stated the same occupations in the censuses of 1960 and 1970 were eligible for inclusion. A total of 375,035 men and 140,139 women from 13 of 26 Swedish counties were included in the final analysis.
95. The study population was followed for evidence of incident rheumatoid arthritis through linkage data from the Swedish Hospital Discharge Register. During the three year observation period from 1980 to 1983, 896 males and 629 females were treated for rheumatoid arthritis.
96. Extensive questionnaires were used and a job-exposure matrix (JEM) was developed in order to estimate occupational exposures.
97. Relative risks were calculated against the background rate of the entire population.⁶⁴

⁶¹ Table 6 p.237

⁶² Table 3 p.236 (key).

⁶³ p.236

⁶⁴ p.307

98. Based on estimated exposures calculated from the job-exposure matrix, no increase in relative risk of hospitalisation was found among workers exposed to mineral or cutting oils and rheumatoid arthritis in men (OR 1.0, 95% CI. 0.8-1.3) or in women (OR 1.1, 95% CI 0.6- 2.1). Nor was there any association between PCBs per se and rheumatoid arthritis in men (RR 1.1, 95% CI 0.7-1.7); while no women reported exposure to PCBs.⁶⁵
99. There was no change in relative risk among males exposed to mineral oils in several occupations such as toolmakers, machine-tool setters and operators (RR 1.2, 95%CI 0.8-1.7) and machinery and engine repairmen.⁶⁶ Very few women were employed in these occupations.
100. In general there were rather small possible differences in the relative risk of rheumatoid arthritis in different exposure groups and different occupations. A moderately increased risk of rheumatoid arthritis was observed in some occupations exposed to substantial use of organic solvent, such as farmers, lacquerers, concrete workers, and male hairdressers (not female hairdressers)⁶⁷.
101. The authors noted that whilst the criteria for working in the same job at 10 year intervals may have ensured continuity of exposure, workers in 'light jobs' may have been able to remain in their jobs with rheumatoid arthritis, whereas workers in 'heavy jobs' may have left, which would underestimate the risk of rheumatoid arthritis in the heavy occupations.⁶⁸
102. As the cases were hospitalised, the study could not detect those cases which had not been hospitalised for rheumatoid arthritis (i.e. under-detection of cases could potentially bias toward the null).

Council's Comments

103. This is one of the few studies that identified PCBs as a possible adjuvant for rheumatoid arthritis. No overall significant association was found between mineral oils and risk of hospitalisation for rheumatoid arthritis in either men or women, nor was there any association between PCBs and risk of hospitalisation for rheumatoid arthritis in men. There were no data on women exposed to PCBs. The study indicated no overall significant association for men or women between rheumatoid arthritis and mineral oils or PCBs.
104. The Council considered that the data from this study does not support a relevant association between mineral oils and rheumatoid arthritis nor does it support a relevant association with PCBs.

⁶⁵ Table II p.307

⁶⁶ p.307

⁶⁷ Table III p.307

⁶⁸ p.308-309.

Lundberg, I. Alfredsson, L. Plato, N. Sverdrup, B. Klareskog, L. & Lkeinau, S. 1994, 'Occupation, occupational exposure to chemicals and rheumatological disease', *Scand J Rheumatol*, vol. 23, pp. 305-310. **RMA ID 36816.**

105. This was a population-based case control study of incident cases of rheumatoid arthritis among the Swedish population aged 18-70. A sample of 1419 Swedish adults with rheumatoid arthritis was compared to 1,674 randomly selected controls, matched in regard to a range of lifestyle factors and occupational exposures. Women made up the majority of participants, but as only men reported substantial occupational exposure to mineral oils; only men were retained in the analysis, leaving 407 cases and 486 controls. Of these, 135 cases and 132 controls reported exposure to mineral oils. The mean age was 50-53 years.
106. All cases met the American College of Rheumatology (ACR) 1987 criteria and were newly diagnosed within the seven-year time period of the study. They were categorised as RF positive if they met the cut-off value of 20 for rheumatoid factor. Antibodies to citrullinated peptides were analysed in cases and controls and a level above 25 µu/ml was regarded as positive.
107. Genotyping was carried out on 81 cases to identify shared epitope genes, which are known risk factors for the positive but not the negative types of RA.
108. Information about environmental exposures was gathered initially through a comprehensive postal questionnaire. Specific questions were asked about occupational exposures to cutting oil, motor oil, form oil, hydraulic oil and asphalt. Subjects who reported exposure to any of those mineral oils were classified as exposed to any mineral oil.
109. Odds ratios⁶⁹ of developing rheumatoid arthritis, for both positive and negative subtypes, were calculated for each type of oil, and adjusted for the potential confounding effects of age, residential area and smoking.
110. Only hydraulic oil reached borderline statistical significance. Combined, the oils increased the risk of rheumatoid arthritis by 30% (RR 1.3, 95% CI 1.0-1.7) which was of borderline significance. For the RF positive types of arthritis, each of the oils showed increases in the direction of the effect.⁷⁰ However, these effects were not individually statistically significant and when the combined oils were analysed, the effect was again of borderline significance (RR 1.4, 95% CI 1.0-2.0). No association was found between mineral oil exposure and RF-negative cases.⁷¹

⁶⁹ Odds ratios were calculated p.R1298.. '...interpreted as the relative risk (RR) because the study was population based' ; p.R1299

⁷⁰ Table 1, p.R1298.

⁷¹ Table 2, p.R1296.

Council's Comments

111. The council considered this to be a critical paper.
112. Both anti CCP+⁷² and RF+⁷³ sub-types of rheumatoid arthritis showed associations with mineral oils, although the effect sizes were not large and the associations were of borderline statistical significance.
113. The Council noted a lack of information about PCBs in the study. It is unclear whether the Swedish oils were contaminated with PCBs. However the range of time covered by the study includes dates before PCBs were banned, which allows for the possibility that some of the mineral oils could have been contaminated.
114. There was also a lack of precision about what the researchers considered to be occupational exposures to mineral oils. There was no quantification of exposure doses.
115. The Council considered that the study
- points to an association between mineral oils in anti-CCP+ and RF+ sub-types of rheumatoid arthritis only. This is at the Reasonable Hypothesis level only and would not satisfy the Balance of Probabilities test.
 - does not provide sufficient information to assess the necessary duration or extent of exposure to mineral oils beyond an 'occupational exposure'; leaving open a relevant association for an acute exposure to mineral oils.

Guo, Y.L. Yu, M.L. Hsu, C.C. Rogan, W.J. 1999, 'Chloracne, Goiter, Arthritis, and Anemia after Polychlorinated Biphenyl Poisoning: 14-Year Follow-Up of the Taiwan Yucheng Cohort', *Environmental Health Perspectives*, vol. 107, no. 9. **RMA ID 18242**.

116. This was a 14 year follow-up study of morbidity in subjects involved in a mass exposure to ingested cooking oils contaminated by PCBs and polychlorinated dibenzofurans (PCDFs) that occurred in Taiwan. A range of symptoms originally reported by 1999 exposed persons was collectively described as 'Yucheng' (oil disease).⁷⁴
117. The exposed subjects, identified from a registry, were matched for age, sex and neighbourhood to a control group. Usable information was available on 705 exposed subjects (men aged 50 ±12.4; women 47.0±12.4) and 693 controls, with no major differences in sex, education level, occupation and smoking history between the groups. No pre-exposure clinical data were reported. Follow-up health outcomes were obtained by telephone interview.

⁷² Anti-cyclic-citrullinated peptide antibodies

⁷³ Rheumatoid factor

⁷⁴ p. 715

118. As the original exposure involved oil which had been heated and reheated, the PCBs had been partly oxidised into PCDFs and other polychlorinated multiple ring structures. Average duration of use of the contaminated oils was eight months; estimated consumption being 1g of PCBs and 3.8 mg of PCDFs. Following the exposure, median blood PCB concentrations in the exposed persons were found to be 10-20 times higher than the background population levels; PCDF levels were of an order of 100-1000 times higher than background levels.⁷⁵
119. Fourteen years after the exposure, the serum levels of PCB and PCDF measured in a subset of women from the cohort were still respectively, 7-fold and 50-80 times higher than in the controls.
120. Arthritis was reported 4.1 times more often in the exposed men than in their controls (OR 4.1, 95% CI 1.8-11.2) and medication was required 3.1 times more often.⁷⁶ In exposed women there was no difference compared with controls.
121. Although the authors did not rule out the possibility of autoimmune-mediated arthritis, evaluation in the early years after exposure had actually shown suppressed serum IgA and IgM and decreased helper-T cells.⁷⁷ Experimental studies on rats had previously demonstrated that TCDD could induce immunologic reactions but although such evidence had not been replicated in humans, the authors suggested that as these chemicals have been shown to have endocrine effects, the possibility of a chemically induced endocrine reaction causing spine and joint disease 'warrants further investigation'.⁷⁸
122. At 14 year follow-up, skin and oral problems were reported with a considerably elevated frequency in the exposed group: 17% of the exposed subjects described acne-like skin lesions compatible with chloracne, compared to 1.3% of control subjects. Other symptoms reported more frequently were hyperkeratosis, abnormal nails, gum swelling, pigmentation, broken teeth, skin allergy, headache and goitre.⁷⁹ In women, anaemia was doubled in those exposed. Various other common chronic conditions were increased but were not statistically different between the two groups.⁸⁰
123. The authors pointed out the similarity of their observations to those of the Japanese 'Yusho' cohort,⁸¹ with the additional finding of a doubled rate of skin allergy in the exposed Yucheng cohort at follow-up.

Council's Comments

⁷⁵ p.715 (as cited from an earlier report)

⁷⁶ p. 716 , nb Table 2 p.717 reads 'medication or surgery'

⁷⁷ p. 718

⁷⁸ p. 718

⁷⁹ p. 716

⁸⁰ p 718 and Table 2 p 717

⁸¹ Yoshimura et al (1985) & (2003), also discussed in these reasons.

124. The Council considered whether the association found in this study between PCB exposure and unspecified arthritis, with an odds ratio of 4.1 in males (95% confidence interval, 1.8 to 11.2) and 1.3 in females (95% confidence interval, 0.8 to 2.3) indicates a relevant association between rheumatoid arthritis and PCB exposure. The Council noted that the type of arthritis reported by the exposed men was not specified in the paper with the resultant possibility that osteoarthritis played a significant role in the association described. The Council considered it noteworthy that the immune responses recorded over time moved in the opposite direction (IgM levels initially suppressed) to what would be expected if the arthritis reported was rheumatoid arthritis. The records of immune responses are insufficient to determine whether the type or types of arthritis reported were solely or mainly osteoarthritis, but it does demonstrate that rheumatoid arthritis was not the sole type of arthritis reported.
125. The Council considered that this study was inconclusive and does not support a relevant association between rheumatoid arthritis and acute PBC exposure.

De Roos, A.J. Cooper, G.S. Alavanja, M.C. & Sandler, D.P. 2005, 'Rheumatoid arthritis among women in the agricultural health study: risk associated with farming activities and exposures', *Ann Epidemiol*, vol. 15, no. 10, pp.762-70. **RMA ID 48734**

126. This was a nested case-control study of occupational exposures among female farm workers with rheumatoid arthritis. Physician-confirmed cases (n = 135) were matched to five controls each (n = 675) by birth date. Logistic regression was used to adjust for birth date and state.
127. The authors did not identify any strong risk factors for rheumatoid arthritis. Risk of rheumatoid arthritis was not associated with mixing or applying pesticides overall, or with any pesticide class, nor did it vary by number of days or years of use. Some of the specific chemicals listed, including the PCB Arachlor (OR 0.5, 95% CI 0.1-1.6), had no significant associations with rheumatoid arthritis

Council's Comments

128. The Council considered that this study does not support a relevant association between PCBs and rheumatoid arthritis.

Illinois Department of Public Health and the University of Chicago 2004, *The La Salle Electrical Utilities Company Morbidity II Study : Final Report*. The Illinois Department Of Public Health and the University of Illinois at Chicago, School Of Public Health. **RMA ID 47839**.

129. This study was conducted by a government department to investigate the health effects on community and workers at an electrical utilities company. The company had manufactured electrical capacitors for almost 40 years, using PCBs and other volatile organic compounds in the manufacturing process. The PCBs were used as a dielectric material and trichloroethylene (TCE) as a degreasing/cleaning agent. Chlorinated naphthalene had been used prior to the use of PCBs and in some processes, after that. In the early years oils containing PCBs had also been

reportedly used for dust control. In the area with the highest use of PCBs and chlorinated naphthalene, lead exposures were also reportedly prominent.⁸²

130. As the company had been dismantled, information about processes and chemicals was obtained through interview. Interviews established that there was PCB contamination throughout the facility, and that all employees were exposed to varying degrees.
131. Of the chemicals, Aroclor 1242 was used initially, followed by Aroclor 1016. Later di-(2)-ethylhexyl phthalate (DEHP) was used instead of PCBs. Methyl ethyl ketone was used for 'limited cleaning purposes'. Onsite PCB contamination was measured (after closure of the plant) and was found in the soil at the site. Polychlorinated dioxins (PCDE) and furans were also found in soil samples at higher than toxicity equivalent quotient (TEQ) recommendations. Measurements also detected these substances in soils outside of the plant site.⁸³
132. Individual PCB exposures were estimated by combining the self-reported job histories of 262 former employees with research data. A pilot study found PCB serum concentrations in a sample of 60 former workers were significantly elevated compared to a sample of 32 residents (14.3ppb vs 3.6ppb). Levels correlated significantly with reported PCB exposure and length of employment and/or length of residence in the area.
133. The overall health study consisted of three phases⁸⁴:
 - a retrospective cohort mortality study of 3,305 former employees;
 - a cross-sectional morbidity study of 191 former employees and 26 community residents, which examined PCB levels in relation to a number of health outcomes including clinical and laboratory tests of participants;
 - 'Morbidity II': a cross-sectional telephone survey of 596 former employees, using a modified stratified sampling method.
134. Preliminary results from the mortality study suggested associations with thyroid and stomach cancer, liver/biliary cancer in women. In the Morbidity I study, some exposures were associated with diabetes. A number of clinical markers and possible effects on children born to female workers were identified.⁸⁵
135. The third part of the study, Morbidity II, (which was information available to the RMA) surveyed surviving persons aged over 65 years who had worked for more than 3 years for the company, plus a sample of those who had worked less than

⁸² p.2

⁸³ pp.3-4

⁸⁴ pp.6-7

⁸⁵ p.7

3 years. The 596 persons interviewed were one-fifth of the original cohort. Health outcomes for the employees and their children were analysed.

136. A Cox proportional hazards regression model was used to evaluate the effect of the amount of exposure to PCBs on workers, taking into the model the time each worker began working there and other relevant covariates.⁸⁶
137. Only those diseases for which there were at least five events were analysed separately.⁸⁷
138. For self-reported rheumatoid arthritis, there were 46 reported cases out of 330 women⁸⁸ and 20 reported cases out of 252 men included in that question. Controlling for age and BMI at age 40, the hazard ratio for women was not statistically significant (HR 1.08, 95% CI 0.89-1.35). For men the hazard ratio was low and not statistically significant (HR 0.72, 95% CI 0.38-1.37).⁸⁹
139. After controlling for potential confounders, the only significant finding was a positive association with breast cancer among female former employees; which the authors found interesting given the lower than expected rate for the County and in the mortality study. Despite potential for selection bias and a number of other potential confounders, the authors stated:

given the coherence with animal literature suggesting estrogenic aetiologies for breast cancer, implies that the finding perhaps should not be completely discounted,⁹⁰

140. A simultaneous study of the County's cancer mortality data from 1969 -1983 found that only pancreatic cancer in males was significantly elevated compared to other similar Counties. A State cancer registry also found that the study area had a lower overall incidence of cancers than other areas in Illinois. For prostate cancer, this lower incidence was statistically significant.⁹¹
141. Limitations to the study noted by the authors were the inherent survivor bias, recall bias, and a lack of sufficient endpoints. However, they considered that their results were consistent with literature reports. They also noted the lack of individual precision in exposure measurement contending that a high correlation between years of exposure and serum PCB levels supported the validity of their exposure measures.⁹²

⁸⁶ p.21

⁸⁷ p.23

⁸⁸ Approximately 14% of women and 8% of men.

⁸⁹ Tables 11a, p.56 and 11b, p.57

⁹⁰ p.24

⁹¹ p.5-6

⁹² pp. 24-25

Council's Comments

142. The Council considered that this was a useful negative study as it provided evidence of no association between PCB exposures and rheumatoid arthritis. In men, the study indicates the possibility that there may be less risk of rheumatoid arthritis in those who had a longer duration of exposure to PCBs.
143. As the rheumatoid arthritis was self-reported, the Council considered it quite likely that some of the participants would have actually had osteoarthritis, which has a very different aetiology to rheumatoid arthritis⁹³. The Council noted that the rate of self-reported rheumatoid arthritis in the participants, who were all aged over 65, was 14% in women and 8% in men, is consistent with the current consensus on background population prevalence among persons over 65 years of age when the reported rheumatoid arthritis is based on self-report (as for this study). The Council noted that estimates based on self-report show the prevalence of rheumatoid arthritis to be much higher than the prevalence of clinically confirmed rheumatoid arthritis for persons over 65 (approximately 3%).^{94 95}
144. The Council considered that this study does not support a relevant association between rheumatoid arthritis and PCBs.

Yoshimura, T. & Hayabuchi, H . 1985, 'Relationship between the amount of rice oil ingested by patients with yusho and their subjective symptoms', *Environ Health Perspect*, vol. 59, pp.47-51. **RMA ID 48819**

145. This study was conducted several years after accidental community exposure to cooking oil contaminated by PCBs in Japan. The authors aimed to clarify the relationship between the quantity of cooking oils consumed and the subjective symptoms reported by the persons exposed to the contaminated oils. (Criteria for objective symptoms of 'Yusho disease' had previously been established). The symptoms were also compared to symptoms cited in health checks of a clerical worker control group. Individual consumption of the oil was estimated from interviews and serum PCB levels taken. Yearly checkups were held at a factory and data compared to those from healthy workers. Analysis was carried out for data collected in 1970, 1971 & 1977.

⁹³ See for example, Helmick et al who reported that 21.6% of all US adults ≥65 years were found to have self-reported a doctor diagnosis of any type of arthritis. Helmick CG, Felson DT, Lawrence RC, Gabriel S, Hirsch R, Kwoh CK, Liang MH, Kremers HM, Mayes MD, Merkel PA, Pillemer SR, Reveille JD, Stone JH; National Arthritis Data Workgroup. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part I.. *Arthritis Rheum*. 2008 58(1):15-25. p 15.

⁹⁴ See for example, Symmons et al: 'In studies conducted on the general population, including groups of older women, approximately 12% to 22% of people correctly self-reported rheumatoid arthritis when compared to clinical diagnosis. Symmons D, Mathers C, Pfeffer C, The global burden of rheumatoid arthritis in the year 2000, http://www.who.int/healthinfo/statistics/bod_rheumatoidarthritis.pdf , accessed 7 Dec 2012, p 40

⁹⁵ See for example, p 824-5 of K Liao & E Karlson, 'Classification and epidemiology of rheumatoid arthritis', in *Rheumatology*, 5th Eds Hochberg M, Silman M, Smolen J, Weinblatt M, Weisman M (Eds), 2011, Philadelphia, Mosby Elsevier, pp 823-828.

146. Among 12 subjective symptoms studied (examined by clinician) numbness of the limbs, coughing, expectoration, and the sensation of 'elevated' teeth showed a dose-response relationship with the amount of contaminated oil consumed, suggesting a close relationship to Yusho. Consistent high rates of complaints of general fatigue and eye discharge were considered possibly to be connected with Yusho, although no dose-response relationships were determined.
147. Other subjective symptoms, such as fever, headache, dizziness, abdominal pain, swelling in the joints, changes in menstruation, and loss of hair failed to show consistent dose-response relationships. However, the authors still considered it 'impossible to deny a causal relationship' [with Yusho].
148. All of the symptoms, except fever, were found to be statistically increased compared to the control group of clerical workers.⁹⁶

Council's Comments

149. The Council noted that the study addressed only the subjective symptoms of Yusho and did not mention rheumatoid arthritis.
150. The Council considered that this study does not support the relevant relationship.

Yoshimura T 2003, 'Yusho in Japan', *Ind Health*, vol. 41, no. 3, pp.139-48. RMA ID 47838

151. This report describes the events and sequelae of the outbreak of a 'strange disease' which occurred in western Japan in 1968. A group of symptoms, characterised by acne-like eruptions, skin pigmentation and eye discharge, became known as 'Yusho' or oil disease. Diagnostic criteria were established.
152. After eliminating various possible aetiologies, the outbreak was eventually traced to ingestion of rice bran cooking oils from a particular company shipped on February 5 - 6, 1968.
153. Careful examination of a production flow chart for the rice oil identified suspect causal agents. Kanechlor 400, a PCB / tetra chlorinated biphenyl used for heat transfer, was found to have contaminated the rice oil. PCBs were found in the fatty tissues of patients. Later analyses questioned whether PCBs alone or combined with PCDFs and /or PCDDs may have been contained in the Kanechlor 400.
154. The authors note:

Further investigation showed that the most important causal agents for Yusho were PCDFs, but not PCBs...

⁹⁶ p.49 & Table 3

In 1990, coplanar PCBs, PCDFs and PCDDs had been classified into 'the group of dioxin ' by the Japanese Government. Therefore, terminology of chemical substances for dioxin should be carefully identified according to definition.⁹⁷

155. An epidemiologic research group conducted a case control and a cohort study as well as further analytical and descriptive reports, published elsewhere. The case control study involved 121 each of individual cases and controls, as well as 69 household cases with 207 household controls; the cohort study followed 266 possibly exposed individuals, and 130 control individuals who normally used the oil but had not used the suspect shipment. The attack rate [of Yusho] in the exposed group was 64%, whereas the unexposed group had no cases.
156. The amount of the suspect oil consumed was recorded at the time of the event, so the researchers were able to match quantities of consumed to cases. Dose-response relationships were found in terms of both case rate and severity.
157. Extensive annual health examinations were carried out for the Yusho patients, including serum PCB levels (and PCDFs from 2002). The incidence of liver diseases was found to be very high amongst the Yusho patients, with a standardised mortality rate (SMR) of liver cancer, 5.9 and for all cancers, 3.26.
158. By 1993 a range of subjective and objective symptoms were still prevalent. No analysis was reported of these symptoms in relation to controls.
159. Another serious sequel of the epidemic was the occurrence of stillbirths, births with abnormal dark skin colouring and subsequent developmental abnormalities in babies born to Yusho families. Later, it was confirmed that PCBs and PCDFs transferred via placenta and breast milk.⁹⁸
160. The study does not report on rheumatoid arthritis.

Council's Comments

161. The Council noted that this study does describe an acute exposure to PCBs, but the Yusho disease is a different disease to rheumatoid arthritis.
162. As no report of rheumatoid arthritis is mentioned, despite regular health checks, the Council considered that this study indicated negative findings for an association between rheumatoid arthritis and acute exposure to PCBs and in fact indicates against such an association.
163. The Council considered that this study touches on but **does not indicate the relevant association with PCBs**. The Council therefore considered this to be a useful negative study.

⁹⁷ p.145

⁹⁸ p.147

Tsuji, H. Hirahashi, T. Ogata, H. & Fujishima, M. 1999, 'Serum immunoglobulin concentrations and autoantibodies in patients with Yusho', *Fukouka Igaku Zasshi*, vol. 90, no. 5, pp.147-9 [abstract only] **RMA 47834**

164. This was an abstract of a follow-up study relating to the Yusho mass exposure reported by Yoshimura et al (1985) and later by Yoshimura (2003) & discussed elsewhere in these Reasons.
165. PCB levels were studied in 79 patients with Yusho in 1997. Autoantibodies were present in 10 cases (12.7%) for rheumatoid factor. The authors found no significant correlations between blood PCB concentrations and serum immunoglobulin concentrations, or presence of autoantibodies.

Council's Comments

166. The presence of rheumatoid factor in blood, on its own, is not a precise indicator of development of the disease rheumatoid arthritis and does not always indicate that a person has rheumatoid arthritis. The Council noted that the study did not examine rheumatoid arthritis and in that sense was not very useful.
167. The Council further noted that the prevalence of rheumatoid factor (RF) in this study population did not differ from background population for the age group.⁹⁹ Background levels of RF positivity in older populations are approximately 15%. The finding of a 12.7% prevalence of RF in the subjects who had Yusho disease was similar to the prevalence in the general population of people, which in the Council's view, tends to point against an increased occurrence of rheumatoid arthritis in the cohort.¹⁰⁰
168. The Council further noted that the levels of antibodies found in the study did not correlate with PCB concentrations measured in the subjects' blood.
169. The Council thus considered that this study was relevant to a possible association with PCBs but indicated against such an association.

Gold, L.S. Ward, M.H. Dosemeci, M & De Roos, A.J. 2007, 'Systemic autoimmune disease mortality and occupational exposures', *Arthritis & Rheumatism*, vol. 56, no. 10, pp.3189-201. **RMA ID 48735**

170. This was a study of mortality from systemic autoimmune diseases in relation to occupations and occupational exposures.

⁹⁹ As the contamination occurred approximately 30 year earlier, the Council considered that the study group would have been, on average, older adults.

¹⁰⁰ See for example, Dequeker, J. Van Noyen, R. Vandepitte, J. 1969, 'Age-related rheumatoid factors. Incidence and characteristics', *Annals of Rheumatic Disease*, vol. 28, no. 4, pp. 431-6

171. The authors examined death certificates from 26 US states for which a cause was listed as rheumatoid arthritis (n = 36,178), systemic lupus erythemous (SLE) systemic sclerosis, or other systemic autoimmune disease. Job exposures were calculated from the 'usual occupation', being the occupations the decedent had held for the longest period of time. Only decedents over the age of 25 were included to allow for the potential of at least five years' working history before death.
172. Additionally, authors estimated risks associated with occupational exposures, which were assigned using a job-exposure matrix.
173. Results suggested that death with some systemic autoimmune diseases may be associated with occupational exposures encountered in farming and industry. Farming as usual occupation was found to be associated with significantly increased risk of death with rheumatoid arthritis (OR 1.31, 95% CI 1.22-1.39). Mining machine operators also an increased risk (OR 1.27, 95% CI 1.10-1.47).
174. Exposure to animals was the only specific exposure with a statistically significant positive association to rheumatoid arthritis. Exposure to pesticides had very small increased relative odds of borderline statistical significance (OR 1.05, 95% CI 1.00 -1.09). There was no specific analysis in relation to PCBs or mineral oils.
175. The authors acknowledged the limitation of this type of study in that autoimmune diseases tend to be under-reported on death certificates.
176. They did point out however, the very large number of decedents included, which allowed the study to be representative of the US population.¹⁰¹

Council's Comments

177. The Council noted that although this was a very large study, the outcome examined was mortality, not morbidity or incidence, and therefore of limited usefulness to this review.
178. PCBs were not measured as an individual exposure. Although PCBs may have been included in some fertilisers, there were various other chemicals which might have been involved. Farming was also a very broadly categorised exposure, and could have been confounded by various other factors, such as exposure to animals.
179. The Council considered that this study touches on, but did not support the relevant association, as it was inconclusive and not specific.

¹⁰¹ p.3199

Kimbrough, R.D. Keouskas, C.A. 2003, 'Human exposure to polychlorinated biphenyls and health effects', *Toxicol Rev*, vol. 22, no. 4, pp. 217-33. **RMA ID 47943**

180. This was a review of literature on PCBs in relation to potential human health effects concluding that studies were inconclusive and did not provide clinical evidence that PCBs at levels encountered with human exposure produce adverse health effects.
181. The differences in PCB blood or tissue concentrations between controls and cases, or between the upper and lower end of various environmentally exposed groups of children or adults, were small.
182. Although some effects were found to be statistically significantly different, they did not, according to these authors, appear to be biologically significant. Many studies on the effects of PCBs are difficult to interpret because the range of normal values for clinical and neurobehavioural tests are not provided or appropriately considered and there was no, or inadequate, control for potential confounders. The authors reported that even the apparent association with some cancers appear to be chance observations resulting from multiple comparisons, since the increase of specific cancers was not consistent between studies and was no longer present in some cohorts when studies were repeated at a later date, with longer follow-up.
183. Overall, the authors considered the data failed to demonstrate conclusive adverse health effects of PCBs at concentrations encountered with environmental human exposures.

Council's Comments

184. Although this was a review, and therefore not primary evidence, the Council considered this to be a useful negative study because the review identified no evidence in the literature of rheumatoid arthritis being a health effect of PCBs.
185. The Council considered that this study does not support the relevant association.

Lee, D-H. Steffes, M. & Jacobs, D. R. Jr. 2007, 'Positive associations of serum concentration of polychlorinated biphenyls or organochlorin pesticides with self-reported arthritis, especially rheumatoid type, in women', *Environmental Health Perspectives*, vol.115, no. 6 pp. 883-888. **RMA ID 46247**

186. In this study, the investigators examined potential cross-sectional associations of serum concentrations of toxic organic chemical compounds with self-reported clinical diagnoses of arthritis in 1,721 adults ≥ 20 years of age in the National Health and Nutrition Examination Survey (NHANES) 1999–2002.¹⁰²

¹⁰² p.883

187. They postulated that background exposure to PCBs may be involved in triggering arthritis through a chronic inflammatory process ¹⁰³ In support of this contention, they cited previous findings that 'persistent organic pollutants ' (POPs) disrupt the endocrine system and markedly influence the immune system. ¹⁰⁴ They defined these as:

organic chemical compounds that are highly toxic, persist in the environment, bio-accumulate in fatty tissues of living organisms, travel long distances, and naturally flow toward colder climates ...Humans are usually exposed to POPs through their food supply...¹⁰⁵

188. When blood concentrations of a range of these compounds were measured in participants, dioxin-like or non-dioxin-like PCBs were found to be positively associated with the prevalence of arthritis in women. When analysed by subtype, the trends according to quartiles of exposure were suggestive of dose-response relationships.¹⁰⁶ The adjusted odds ratio for rheumatoid arthritis in women, by quartile were 1.0, 7.6 (95%CI 1.7-34.4), 6.1 (95%CI 1.2-29.7), and 8.5 (95% CI 1.6-44.5) respectively for dioxin-like PCBs (p for trend = 0.05), 1.0, 2.2 (95% CI 0.6-7.4), 4.4 (95% CI 1.3-15.2), and 5.4 (95%CI 1.4-20.3) for non-dioxin-like PCBs (p < 0.01).¹⁰⁷ Osteoarthritis showed no association with PCBs.

Adjusted ORs for unspecified arthritis subtype (n=168) were weaker than those of RA but stronger than for OA (Table 4), as we expected because these cases were considered likely to be a mixture of mostly RA and OA¹⁰⁸.

189. Results were all adjusted for age, race income, BMI and smoking.
190. No clear associations between PCDDs or PCDFs and arthritis were demonstrated.
191. Diagnosis of rheumatoid arthritis was based on the participants' recall of a clinical diagnosis. The authors argued that

...although the validity of report of all types of arthritis combined is high, validity of the subtype of arthritis based on questionnaire has been reported to be low (Star et al.1996). Given this fact, the predominance of RA and OA among all arthritis types, and our hypothesis that jointly involved RA and OA, we primarily focused our investigation on the association between serum concentrations of POP¹⁰⁹, and prevalence of all arthritis and further analyzed by the subtype of arthritis.¹¹⁰

103 p.883

104 p.883

105 p.883

106 p.886

107 p.886

108 p.886

109 'persistent organic pollutants'

110 p.883

192. However, as subjects were unaware of their serum PCB levels, the authors argued that any misclassification was not likely to be biased, and they considered it unlikely to explain why the association was much stronger for rheumatoid arthritis than it was for osteoarthritis¹¹¹.
193. Methodological advantages of the study included the population based sample selection, standardised survey method and use of blood samples to provide objective measure of exposure to the chemicals.

Council's Comments

194. The Council noted that a statistically significant association with a suggested dose-response relationship rheumatoid arthritis was reported for women only for non-dioxin like PCBs and a borderline association for dioxin-like PCBs.
195. However, the Council pointed out that the study weakness was that rheumatoid arthritis was self-reported, and it considered that self-report of rheumatoid arthritis is unreliable, although self-report of a clinical diagnosis is less unreliable. The number of cases of self-reported rheumatoid arthritis ($n = 93$) was low and the Council considered that the number of sub-analyses weakened the validity of the findings.
196. In addition, the cross-sectional design of the study leaves open, in the Council's opinion, the possibility that the PCB exposures occurred after the onset of RA.
197. The Council further noted that the results for men were not statistically significant and did not demonstrate a dose-response relationship.
198. The Council considered that this paper leaves open the relevant association between PCBs and rheumatoid arthritis in both women and men.

Other studies commented on by the Council.

Emmett, E.A. Maroni, M. Schmith, J.M. Levin, B.K. & Jefferys, J 1988, 'Studies of transformer repair workers exposed to PCBs: 1. Study design, PCB concentrations, questionnaire, and clinical examination results', *Am J Ind Med*, vol.13, pp.415-27. **RMA ID 47832**

199. This cross-sectional study compared 55 transformer repairmen, 38 currently, and 17 previously exposed to PCBs (predominantly Aroclors (1260) with 56 non-exposed subjects. PCBs exposures occurred from air and contaminated surfaces, predominantly from Aroclor 1260 with some exposure to Aroclor 1242. Each worker underwent: a questionnaire; standardized medical examination; delayed hypersensitivity testing; and determination of serum and adipose tissue lipid total PCB concentrations. The study objectives were to determine whether abnormalities caused by PCBs were occurring in a population exposed to Aroclor 1260, to compare PCBs in past exposed and current transformer workers, and to

¹¹¹ p.886

define PCBs in a population without industrial exposure. The study, published in 1988, was performed in 1980. The exposed groups were responsible for maintenance and repair of approximately 1,100 high voltage electrical transformers. The shop had been in existence for approx 9 years (some employees having previously worked at similar tasks). Occupational exposure to leaking transformers was quite common. The average work time in the shop was 3.75 years.

200. Adipose and serum [PCBs] were significantly higher in the currently exposed transformer workers but previously exposed workers did not differ significantly from comparison subjects.

No subjects presented with a classical syndrome (chloracne, jaundice, etc) clinically recognizable as compatible with PCB toxicity as described in the literature and summarized by Kimbrough [1980]¹¹².

201. A number of neurobehavioral and irritant symptoms were significantly more prevalent in the exposed group, but 'were probably not related to PCBs'. Comedones were more frequent in the exposed group, 'classical chloracne' was not found. Cutaneous delayed hypersensitivity responses to mumps and to trichophyton antigens did not differ between the groups.

202. Both serum and adipose PCBs were considerably higher in the currently exposure workers. Serum but not adipose concentrations were associated with length of time spent in PCB related activities.

Although both adipose and serum [PCBs] were considerably higher in the occupationally exposed group, this difference was due to the levels in currently exposed workers and neither serum nor adipose [PCBs] were significantly different between past exposed workers and those in the comparison group.

203. This was interpreted as reflecting substantial elimination of PCBs.

204. However, the authors noted that 'a significant positive relationship could still be found in retired transformer workers with the highest PCB exposure'.

Council's Comments

205. The Council noted that this study was particularly relevant to the Applicant who experienced cutaneous contact with transformer oil. It dealt with chronic exposures to PCBs but recorded no findings of rheumatoid arthritis.

206. The Council found this study therefore did not support the relevant association between rheumatoid arthritis and PCB exposure and tended to indicate against such an association.

¹¹² p.421

Stark, A.D. Costas, K. Chang, H.G. & Vallet, H.L. 1986, 'Health effects of low-level exposure to polychlorinated biphenyls', *Environ Research*, vol.41, pp.174-83. **RMA ID 47829**

207. This study was conducted following an accidental mass exposure to PCBs. An oil spill from a transformer in Syracuse, New York, with no subsequent fire, provided an opportunity for examining the effects of low-level PCB exposure without the confounding presence of furans and dioxins.
208. Analysis of the oils after the explosion showed 64% PCB (Arochlor 1254) and the remainder trichlorobenzene.
209. There were 52 individuals exposed to PCB among building personnel, police, firemen, and public utility employees. Sixty-eight non-exposed were matched to the exposed group by sex, age, employer, and job description. Data were collected on the exposed relative to their activities at the spill site, their location, possible routes of exposure, duration of exposure, and subsequent health effects. Exposed and non-exposed were interviewed for past medical history and relevant symptoms. Blood chemistry was studied for a range of clinical indicators, including fasting blood PCB level measurement. Six weeks after the spill, all tests were repeated, except for the CBC and PCB levels.
210. Laboratory results were unremarkable. Some transient skin irritation, believed to be associated with PCBs, was noted in the exposed subjects. Significant trends in PCB blood level were shown in relation to occupation, age, duration of exposure, and level of alcohol consumption, and triglycerides. Results were controlled for age and alcohol consumption.
211. The authors noted that only length of exposure was significantly associated with PCBs, and the person with the highest PCB concentration had 34 years experience with a utility company. The utility company employees who were exposed to the spill were normally involved in servicing transformers, with the opportunity for occupational exposure to PCB oil. Utility company employees were found to have the highest mean blood PCB level of all occupations in the study. They concluded that this finding was reasonable because the repairmen were occupationally exposed, and 'PCB contact with skin and clothes was a commonplace event'.¹¹³

Council's comments

212. The Council noted that the study touches on the relevant association to the extent that the subjects had both acute and long-term exposure to PCBs, from working with transformers. The Council further noted that the PCB blood levels of the acutely exposed subjects were not significantly elevated compared to those who did not have acute exposure.

¹¹³ p.180

213. The Council noted that after six weeks from the exposure there was no rheumatoid arthritis reported.
214. The Council considered that although the study did not directly address rheumatoid arthritis, the absence of any findings of the disease in either acutely or chronically exposed subjects points against an association.

Sinks, T. Steele, G. Smith. A.B. Watkins, K. & Shults, R.A. 1992, 'Mortality among Workers Exposed to Polychlorinated Biphenyls', *American Journal of Epidemiology*, vol.136, no. 4. RMA ID 14100

215. The authors conducted a retrospective cohort analysis (1957-1986) comparing the mortality of 3,588 electrical capacitor manufacturing workers (male and female) with known exposure to PCBs with age-, sex-, and calendar time-specific mortality rates for all whites in the United States.
216. Proportional hazards modelling were also performed to examine any possible association between cumulative PCB exposure and site-specific cancer mortality.
217. No statistically significant associations were found. All-cause mortality and total cancer mortality was less than expected. More deaths were found than expected from malignant melanoma and cancer of the brain and nervous system, but the results were inconclusive.

Council comments

218. The Council noted that whilst the study did examine workers with known exposure to PCBs, the outcome of the study was death from cancer, which is not relevant to rheumatoid arthritis.
219. The Council considered that the study does not touch on the relevant association.

Edwards, C.J. & Cooper, C 2005, 'Early environmental factors and rheumatoid arthritis', *Clin Exp Immunok*, vol.143, pp.1-5. RMA ID 46454

220. This review examined whether early environmental effects on growth and infectious exposure may influence the likelihood of developing autoimmune diseases such as rheumatoid arthritis.
221. The authors hypothesised that the lack of success defining the environmental factors important in developing rheumatoid arthritis may be due to previous studies concentrating only on the time around disease onset.

There is evidence of production of the auto antibodies rheumatoid factor (RF) and anti-cyclic citrullinated peptides (anti-CCP) and increased levels of C-reactive protein (CRP) years before RA becomes clinically apparent.

222. In addition, they considered that early life events, including intrauterine growth retardation, and early infections, may have long lasting effects on immune function, potentially contributing to rheumatoid arthritis.

Council's comments

223. The Council noted that this paper focuses on early development. It made no reference to PCBs or mineral oils and was therefore not directly relevant to the association. The authors suggest that a developing immune system exposed to improved standards of hygiene is more likely to produce RF and perhaps begin the pathological process that leads to RA, but their findings are not conclusive.
224. The Council considered this paper does not support a relevant association.

THE COUNCIL'S CONCLUSIONS ON WHETHER THERE SHOULD BE A FACTOR(S) FOR EXPOSURE TO MINERAL OILS CONTAINING PCBs OR ACUTE PCB EXPOSURE

225. The Council, having closely analysed all the information in the pool, placed particular weight on the articles discussed in detail above. The first critical question in the second step of the Council's review was whether there is sound medical-scientific evidence that indicates ('points to', as opposed to merely 'leaves open') the possibility that exposure to mineral oils containing PCBs or acute PCB exposure (if found to exist in a particular case) could provide a link or element in a reasonable hypothesis connecting rheumatoid arthritis or death from rheumatoid arthritis to relevant service. Only if the Council was satisfied of a reasonable hypothesis, could the Council go on to consider the balance of probabilities.
226. The Applicant contended that the reasonable hypothesis factor for mineral oils should be expanded to include exposure to mineral oils containing PCBs or acute PCB exposure.
227. The Council looked for evidence concerning rheumatoid arthritis and mineral oils and PCBs and noted that a number of gaps existed in the available information. Most studies did not record objective measures of either mineral oil or PCB exposures, whereas some studies dealing with proven exposure, did not report objective clinical diagnoses of rheumatoid arthritis.
228. The only epidemiological study which points to a relevant association between mineral oils and rheumatoid arthritis, and thus supports the existing factor in Statement of Principles No. 68 of 2008 is Sverdrup et al (2005), a population based case control study, which found small positive associations between occupational exposures to mineral oils and positive sub-types of RA only and made no mention or findings in respect of PCBs.
229. None of the studies specifically considered acute exposures to mineral oils (which may have contained PCBs). Reckner Olsson et al (2000 & 2004), Lundberg et al

(1994), Sverdrup et al (2005) Gold et al (2007) and Kimborough et al (2003) all studied occupational exposures.

230. Acute exposures to PCBs were considered by Guo et al (1999), Yoshimura et al (1985 and 2003), Tsuji (1999) and Stark et al (1986) but none found a statistically significant association with rheumatoid arthritis. The positive findings by Guo et al of increased reports of arthritis after acute PCB exposure (from ingested contaminated cooking oils) were insufficiently specific to the type of arthritis to permit the Council to find that research indicated a relevant association between rheumatoid arthritis and acute PCB exposure.
231. 230. The Council was not satisfied that the study by Lee et al (2007) indicates a relevant association for women or men, for the reasons expressed at [194] – [198]; in addition to the design weaknesses mentioned, the statistically significant association reported for women was based on self-reported rheumatoid arthritis which the Council, in their collective expertise, considered is not reflective of clinical diagnoses.
232. The Council was therefore satisfied that the sound medical-scientific evidence available to the RMA is insufficient to justify an amendment to the existing factor for mineral oils in Statement of Principles 68 of 2008 to include exposure to mineral oils containing PCBs or acute PCB exposure. As the Council found the sound medical-scientific evidence insufficient to justify amendment to the reasonable hypothesis Statement of Principles, it did not go on to consider the balance of probabilities Statement of Principles.

NEW INFORMATION

233. The Council considered the 'new information' in **Appendix C** (ie information that was not available to (not before) the RMA at the relevant times) to which it was referred with respect to the contended factors.
234. The new information was not taken into account for the purposes of the review, but rather was considered to determine whether, in the Council's view, it warranted the Council making any directions or recommendations to the RMA.
235. In the Council's view, any such direction or recommendation should only be made if it were to form the view that the new information:
- comprised sound medical-scientific evidence as defined in section 5AB(2) of the VEA, being information which epidemiologists would consider appropriate to take into account; which
 - in the Council's view 'touches on' (is relevant to) the contended factors; and
 - has been evaluated by the Council according to epidemiological criteria, including the Bradford Hill criteria.

236. The Council considered that the new toxicity studies and hazard guidelines provided by the applicant being based on evidence from studies on effects in response to certain toxins, were not relevant to the review, particularly where they did not refer specifically to the condition of rheumatoid arthritis.
237. The Council further considered that a number of website articles referred by the applicant were not sound medical-scientific evidence¹¹⁴ in that they were opinion based, were not subject to peer review or, so far as concerning causes of rheumatoid arthritis, did not meet the applicable criteria for assessing causation currently applied in the field of epidemiology .
238. The Council was not sufficiently persuaded of the matters in [235] to make any recommendation or direction to the RMA concerning the undertaking of a fresh investigation specifically on this basis.

DECISION

239. The Council made the declarations summarised in **paragraphs 1 and 2** above.

EVIDENCE BEFORE THE COUNCIL

240. Preliminary list of the proposed pool of information, as advised to the Applicant and Commissions by letters dated 22 March 2012 (see [26] of the Reasons) is listed in **Appendix A**
241. The information considered by the Council (being the information that the RMA advised was available to (before) the RMA at the relevant times and which the RMA sent to the Council in accordance with section 196K of the VEA) is listed in **Appendix B**.
242. As mentioned above, the information upon which the Council understands the Applicant and the Commissions relied (being information which the RMA advised was available to (before) the RMA at the relevant times and which the RMA sent to the Council in accordance with section 196K of the VEA) is also listed in **Appendix A**.
243. The information to which the Applicant 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 Council in reaching its review decision) is listed in **Appendix C**.

¹¹⁴ See footnote 2 above.

Articles cited in the Council's analysis

Appendices

Appendix A	<p>Preliminary list of the proposed pool of information, as advised to the Applicant and Commissions by letters dated 22 March 2012 (see 26] of the Reasons).</p> <p>This list also identifies the information upon which the Applicant and the Commissions relied (being information which the RMA advised was available to (before) the RMA at the relevant times and which the RMA sent to the Council in accordance with section 196K of the VEA).</p>
Appendix B	<p>Information forwarded to the Council under section 196K of the VEA referable to the Council's review of Statements of Principles Nos. 68 and 69 of 2008.</p>
Appendix C	<p>Material that the RMA advised was not available to (not before) the RMA (which the Applicant contended was in existence at the relevant times, and so could have been accessed by the RMA).</p>

APPENDIX A

Preliminary list of the proposed pool of information, as advised to the Applicant and Commissions by letters dated 22 March 2012 (see [26] of the Reasons), including information upon which the Applicant and the Commissions relied (being information which the RMA advised was available to (before) the RMA at the relevant times and which the RMA sent to the Council in accordance with section 196K of the VEA).

This list also identifies the information upon which the Applicant and the Commissions relied (being information which the RMA advised was available to (before) the RMA at the relevant times and which the RMA sent to the Council in accordance with section 196K of the VEA).

APPENDIX A

RMA ID	Details	Relied on by
47840	Aho, K. Heliovaara, M. Knekt, P. Reunanen, A. Aromaa, A. Leino, A. Kurk, I.P. Heikkilä, R. Palosuo, T & Iosuo, T. 1997, 'Serum immunoglobulins and the risk of rheumatoid arthritis', <i>Ann Rheum Dis</i> , vol. 56, no. 6, pp.351-6.	Applicant
47826	Bernard, A. & Fierens, S. 2002, 'The Belgian PCB/Dioxin incident: a critical review of health risks evaluations', <i>Int J Toxicol</i> , vol. 21 pp.333-40.	
14861	Brown, D.P. 1987, 'Mortality of Workers Exposed to Polychlorinated Biphenyls - An Update', <i>Arch Environ Health</i> , vol. 42, no. 6, pp. 333-9.	
45909	Carpenter, D.O. 2006, 'Polychlorinated biphenyls (PCBs): routes of exposure and effects on human health', <i>Reviews on Environmental Health</i> , vol. 21, no. 1, pp. 1-23.	
35415	Commonwealth of Australia 2003, 'Study of Health Outcomes in Aircraft Maintenance Personnel (SHOAMP) Volume 1: Literature Review. Final Report July 2003.	
30666	Cooper, G.S, Miller, F.W. & Germolec D.R. 2002, 'Occupational exposures and autoimmune diseases', <i>International Immunopharmacology</i> , vol. 2, pp. 303-313.	
48734	De Roos, A.J. Cooper, G.S. Alavanja, M.C. & Sandler, D.P. 2005, 'Rheumatoid arthritis among women in the agricultural health study: risk associated with farming activities and exposures', <i>Ann Epidemiol</i> , vol. 15, no. 10, pp.762-70.	
46526	Dooley, M.A. & Hogan, S.L. 2003, 'Environmental epidemiology and risk factors for autoimmune disease', <i>Curr Opin Rheumatol</i> , vol.15, pp.99-103.	
46454	Edwards, C.J. & Cooper, C 2005, 'Early environmental factors and rheumatoid arthritis', <i>Clin Exp Immunok</i> , vol.143, pp.1-5.	
47832	Emmett, E.A. Maroni, M. Schmith, J.M. Levin, B.K. & Jefferys, J 1988, 'Studies of transformer repair workers exposed to PCBs: 1. Study design, PCB concentrations, questionnaire, and clinical examination results', <i>Am J Ind Med</i> , vol.13, pp.415-27.	
47835	Eskandari, F. Webster, J.I. & Sternberg, E.M. 2003, 'Neural immune pathways and their connection to inflammatory diseases', <i>Arthritis Res Ther</i> , vol. 5, no. 6, pp.251-65.	Applicant

48175	Franchini, M. Ria, I M. Buiatti, E. Bianchi, E. 2004, 'Health effects of exposure to waste incinerator emissions: a review of epidemiological studies', <i>Ann 1st Super Sanita</i> , vol. 40, no. 1, pp.101-15.	
47397	Garabrant, D.H. & Dumas, C. 2000, 'Epidemiology of organic solvents and connective tissue disease', <i>Arthritis Research</i> , vol. 2, pp.5-15.	
48735	Gold, L.S. Ward, M.H. Dosemeci, M & De Roos, A.J. 2007, 'Systemic autoimmune disease mortality and occupational exposures', <i>Arthritis & Rheumatism</i> , vol. 56, no. 10, pp.3189-201.	
18242	Guo, Y.L. Yu, M.L. Hsu, C.C. Rogan, W.J. 1999, 'Chloracne, Goiter, Arthritis, and Anemia after Polychlorinated Biphenyl Poisoning: 14-Year Follow-Up of the Taiwan Yucheng Cohort', <i>Environmental Health Perspectives</i> , vol. 107, no. 9.	Applicant & Commissions
40028	Gustavsson, P. & Hogstedt, C. 1997, 'A cohort study of Swedish capacitor manufacturing workers exposed to polychlorinated biphenyls (PCB's)', <i>Am J Ind Med</i> , vol. 32, no. 3, pp. 234-9.	
47828	Hagmar, L. Wallin, E. Vessby, B. Jonsson, B.A.G. Bergman, A & Rylander, L. 2006, 'Intra-individual variations and time trends 1991-2001 in human serum levels of PCB, DDE and hexachlorobenzene', <i>Chemosphere</i> , vol. 64, pp.7-13.	
48456	Herrick, R.F, Meeker, J.D. Hauser, R. Altshul, L. Weymouth. G. A. 2007, 'Serum PCB levels and congener profiles among US construction workers', <i>Environ Health</i> , vol. 6 p.25.	
47839	Illinois Department of Public Health and the University of Chicago 2004, <i>The La Salle Electrical Utilities Company Morbidity II Study : Final Report</i> . The Illinois Department Of Public Health and the University of Illinois at Chicago, School Of Public Health.	Applicant & Commissions
30189	Khuder, S.A. Peshimam, A.Z. & Agraharam, S. 2002, 'Environmental risk factors for rheumatoid arthritis', <i>Reviews on Environmental Health</i> , vol.17, pp.307-315.	
47943	Kimbrough, R.D. Keouskas, C.A. 2003, 'Human exposure to polychlorinated biphenyls and health effects', <i>Toxicol Rev</i> , vol. 22, no. 4, pp. 217-33.	
48043	Kitamura, K. Kikuchi, Y. Watanabe, S. Waechter, G. Sakurai, H. & Takada, T. 2000, 'Health effects of chronic exposure to polychlorinated dibenzo-P-dioxins (PCDD), dibenzofurans (PCDF) and coplanar PCB (Co-PCB) of municipal waste incinerator workers', <i>Journal of Epidemiology</i> , vol. 10, no. 4, pp.262-70.	

49013	Kuratsune M, Yoshimura T, Matsuzaka J, Yamaguchi A 1971, 'Yusho, a poisoning caused by rice oil contaminated with polychlorinated biphenyls', <i>HSMHA Health Reports</i> , vol. 86, no. 12, pp.1083-91.	
47830	Kuusisto, S. Lindroos, O. Rantio, T. Priha, E. & Tuhkanen, T. 2007, 'PCB contaminated dust on indoor surfaces - health risks and acceptable surface concentrations in residential and occupational settings', <i>Chemosphere</i> , vol. 67, pp.1194-201.	
47836	Kwon, O. Lee, E. Moon, T.C. Jung, H. Lin, C.X. Nam, K.S. Baek, S.H. Min, H.K. & Chang, H.W. 2002, 'Expression of cyclooxygenase-2 and pro-inflammatory cytokines induced by 2,2',4',5,5'-hexachlorobiphenyl (PCB 153) in human mast cells requires NF-kB activation', <i>Biol Pharm Bull</i> , vol. 25, no.9, pp.1165-8.	Applicant
46247	Lee, D.H. Steffes, M. Jacobs, D.R. Jr. 2007, 'Positive associations of serum contraction of polychlorinated biphenyls or organochlorine pesticides with self-reported arthritis, especially rheumatoid type, in women', <i>Environmental Health Perspectives</i> , vol. 115, no.6, pp. 883-888.	Applicant & Commissions
5681	Lundberg, I. Alfredsson, L. Plato, N. Sverdrup, B. Klareskog, L. & Lkeinau, S. 1994, 'Occupation, occupational exposure to chemicals and rheumatological disease', <i>Scand J Rheumatol</i> , vol. 23, pp. 305-310.	Commissions
47833	Mallin, K. McCann, K. D'Alosio, A. Freels. S, Piorkowski, J. Dimos, J. & Persky, V. 2004, 'Cohort mortality study of capacitor manufacturing workers, 1944-2000', <i>J Occup Environ Med</i> , vol. 46, no.6, pp.565-76.	
46581	Miller, F.W. 2006, 'Is occupational exposure to mineral oil a risk factor for rheumatoid arthritis?' <i>Nature Clinical Practice</i> , vol. 2, no. 3, pp.130-1.	
30227	National Academy of Sciences 2003, 'Gulf War and Health effects: Volume 2. Insecticides and solvents', <i>National Academy Press, Washington, DC</i> , pp. 518-520.	
47831	Nichols, B.R. Hentz, K.L. Aylward, L. Hays, S.M. Lamb, J.C 2007, 'Age-specific reference ranges for polychlorinated biphenyls (PCB) based on the NHANES 2001-2002 survey', <i>J Toxicol Environ Health, A</i> , vol. 70, pp.1873-7.	
48955	Okumura, M. 1984, 'Past and current medical states of Yusho patients', <i>Am J Ind Med</i> , vol. 5, pp.13-8.	
46618	Oliver, J.E. Silman, A.J. 2006, 'Risk factors for the development of rheumatoid arthritis', <i>Scand J Rheumatol</i> , vol. 35, pp.169-74.	

30451	Reckner Olsson, A. Skogh, T. Axelson. O. & Wingren, G. 2004, 'Occupations and exposures in the work environment as determinants for rheumatoid arthritis', <i>Occup Environ Med</i> , vol. 61, pp.233-8.	Commissions
27665	Reckner Olsson, A. Skogh, T. & Wingren, G. 2000, 'Occupational determinants for rheumatoid arthritis', <i>Scandinavian Journal of Work, Environment and Health</i> , vol. 26, no. 3, pp.243-9.	
47827	Ross, G. 2004, 'The public health implications of polychlorinated biphenyls (PCBs) in the environment', <i>Ecotoxicology and Environmental Safety</i> , vol.59, pp.275-91.	
48792	Rudel, R.A. Seryak, L.M. & Brody, J.G. 2008, 'PCB-containing wood floor finish is a likely source of elevated PCBs in residents' blood, household air and dust: a case study of exposure', <i>Environ Health</i> , vol.7, p.2	
14100	Sinks, T. Steele, G. Smith. A.B. Watkins, K. & Shults, R.A. 1992, 'Mortality among Workers Exposed to Polychlorinated Biphenyls', <i>American Journal of Epidemiology</i> , vol.136, no. 4.	
47829	Stark, A.D. Costas, K. Chang, H.G. & Vallet, H.L. 1986, 'Health effects of low-level exposure to polychlorinated biphenyls', <i>Environ Research</i> , vol.41, pp.174-83.	
36816	Sverdrup, B. Kallberg, H. Bengtsson, C. Lundberg, I. Padyukov, L. Alfredsson, L. & Klareskog, L. 2005, 'Association between occupational exposure to mineral oil and rheumatoid arthritis: results from the Swedish EIRA case-control study', <i>Arthritis Research & Therapy</i> , vol. 7, pp. R1296-R1303.	Applicant & Commissions
48785	Sverdrup, B. Klareskog, L. & Keleinau, S. 1998, 'Common commercial cosmetic products induce arthritis in the DA rat', <i>Environ Health Perspect</i> , vol.106, no.1, pp.27-32.	Commissions
48811	Tee, P.G. Sweeney, A.M. Symanski, E. Gardiner, J.C. Gasior, D.M. & Schantz, S.L. 2003, 'A longitudinal examination of factors related to changes in serum polychlorinated biphenyl levels', <i>Environ Health Perspect</i> , vol. 111, no. 5, pp.702-07.	
47837	Tithof, P.K. Schiamberg, E. Peters-Golden, M. Ganey. P.E. 1996, 'Phospholipase A2 is involved in the mechanism of activation of neutrophils by polychlorinated biphenyls', <i>Environ Health Perspect</i> , vol. 104, no. 1, pp.52-8.	Applicant
48814	Tryphonas, H. 1995, 'Immunotoxicity of PCBs (Aroclors) in relation to Great Lakes', <i>Environ Health Perspect</i> , vol.103 suppl 9, pp.35-46.	Applicant
47834	Tsuji, H. Hirahashi, T. Ogata, H. & Fujishima, M. 1999, 'Serum immunoglobulin concentrations and autoantibodies in patients with Yusho', <i>Fukouka Igaku Zasshi</i> , vol. 90, no. 5, pp.147-9 (Abstract)	Applicant

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48817	Van Larebeke, N. Hens, L. Schepens, P. Covaci, A. Baeyens, J. Everaert, K. Bernheim, J.L. Vlietinck, R. & De Poorter, G. 2001, 'The Belgian PCB and dioxin incident of January-June 1999: exposure data and potential impact on health', <i>Environ Health Perspect</i> , vol. 109, no. 3, pp. 265-273.	
47841	World Health Organisation (WHO) 1992, 'Polychlorinated Biphenyls and Terphenyls' (2nd Edition), <i>Environmental Health Criteria</i> 140.	Applicant
48818	Wingfors, H. Selden, A.I. Nilsson, C. & Haglund, P. 2006, 'Identification of markers for PCB exposure in plasma from Swedish construction workers removing ole elastic sealants', <i>Ann Occup Hyg</i> , vol. 50, no. 1, pp.65-73.	
47838	Yoshimura, T. 2003, 'Yusho in Japan', <i>Ind Health</i> , vol. 41, no.3 , pp.139-48.	Applicant
48819	Yoshimura, T. & Hayabuchi, H . 1985, 'Relationship between the amount of rice oil ingested by patients with yusho and their subjective symptoms', <i>Environ Health Perspect</i> , vol. 59, pp.47-51.	

APPENDIX B

Information forwarded to the Council by the RMA under section 196K of the VEA referable to the Council's review of Statements of Principles Nos. 68 & 69 of 2008.



Australian Government
Repatriation Medical Authority

Rheumatoid Arthritis

RMA ID Number	Reference List for investigation #264-3 as at 7 October 2008
27749	Abdel-Nasser AM, Rasker JJ, Valkenburg HA (1997). Epidemiological and clinical aspects relating to the variability of rheumatoid arthritis. <i>Semin Arthritis Rheum</i> , Vol 27 pp 123-40.
46717	Adams A, Lehman TJA (2005). Update on the pathogenesis and treatment of systemic onset juvenile rheumatoid arthritis. <i>Curr Opin Rheumatol</i> , 17:612-6.
46534	Adams A, Lehman TJA (2005). Update on the pathogenesis and treatment of systemic onset juvenile rheumatoid arthritis. <i>Curr Opin Rheumatol</i> , 17:612-6.
46464	Adams KM, Yan Z, Stevens AM, Nelson JL (2007). The changing maternal "self" hypothesis: a mechanism for maternal tolerance of the fetus. <i>Placenta</i> , 28:378-82.
27868	Adebajo AO, Hazleman BL (1996). Malaria and rheumatoid arthritis in West Africa. <i>Clinical & Experimental Rheumatology</i> , Vol 14 pp 346.
46527	Adlesic M, Verdrengh M, Bokarewa M, Dahlberg L, et al (2007). Histamine in rheumatoid arthritis. <i>Scand J Immunol</i> , 65:530-7.
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S1.1	Repatriation Commission 1996, 'Statement of Principles concerning Rheumatoid Arthritis', dated 7 August 1996
S1.2	Repatriation Medical Authority (RMA) 1996, 'Statement of Principals for Rheumatoid Arthritis', dated 2 September 1996
S1.3	Name Provided (and removed under s1961 of the VEA) 2002, 'Request to the Repatriation Medical Authority for Review of Statements of Principles for Rheumatoid Arthritis', dated 26 September 2002.
S1.4	Name Provided (and removed under s1961 of the VEA) 2004, 'Submission to the Repatriation Medical Authority', dated 1 September 2004.
S1.5	Repatriation Commission 2004, 'Rheumatoid arthritis and smoking', Submission by the Repatriation Commission to the Authority for consideration in the investigation which resulted in Statements of Principles Nos 32 and 33 of 2004.
S1.6	Repatriation Medical Authority (RMA) 2004, 'Rheumatoid Arthritis', dated 26 May 2004.
S1.7	Repatriation Medical Authority (RMA) 2004, 'Does smoking cause RA?', dated 1 June 2004
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S1.9	Name Provided (and removed under s1961 of the VEA), 2005, 'Request to the Repatriation Medical Authority for Review of Statements of Principles for Rheumatoid Arthritis', dated 14 February 2005
S1.10	Repatriation Medical Authority (RMA) 2005, 'Rheumatoid arthritis and PCBs', dated 15 February 2005
S1.11	Name Provided (and removed under s1961 of the VEA) 2006, 'Request to the Repatriation Medical Authority for Review of Statements of Principles 68 and 69 for Rheumatoid Arthritis', dated 14 June 2006
S1.12	Name Provided (and removed under s1961 of the VEA), 2006, Submission to the Repatriation Medical Authority for Review of Statements of Principles for Rheumatoid Arthritis, dated 20 July 2006.
S1.13	Repatriation Medical Authority (RMA) 2006, 'Polychlorinated biphenyls and rheumatoid arthritis', dated 24 July 2006

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S3.1	RMA Medical Researcher 2008, 'Investigation into Rheumatoid Arthritis', dated October 2008
S3.2	RMA Medical Researcher 2008, 'Summary of Issues in relation to investigation into Rheumatoid Arthritis', dated October 2008.
S3.3	Repatriation Medical Authority (RMA) 2008, 'Summary of studies table in relation to investigation into Rheumatoid Arthritis', dated October 2008

APPENDIX C

List of the information upon which the Applicant relies, which the RMA has advised the Council is new information because it was not available to (not before) the RMA.

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