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

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').

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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

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(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:

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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 2012

both 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.

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[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 Substance[12,] 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:

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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- I beta (IL- Ibeta), 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 cycloxoygenase-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:

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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. Schiamberg, 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.

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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. 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', *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, 'Chloracen, 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 Toxins [22] 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

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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 lymphoctosis.

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.

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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 contration 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 guidelines[28] 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:

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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

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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:

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[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.

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35 Lee, D.H. Steffes, M. Jacobs, D.R. Jr. 2007, 'Positive associations of serum contration 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 2004[39] 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

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37 Guo, Y.L. Yu, M.L. Hsu, C.C. Rogan, W.J. 1999, 'Chloracen, 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 [41] 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 [44] 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]).

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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's[48] 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

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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 capacitators 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 CONTENDED 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 arthritis[52] 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

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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]

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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]

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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.

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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 ratios[69] 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]

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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.

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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

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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 phases[84]:

- 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 women[88] 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]

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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 arthritis[93]. 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.

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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 conduced 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, Pfeger 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 be 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). he 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.

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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.

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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 OA[108].

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 POP[109], and prevalence of all arthritis and further analyzed by the subtype of arthritis. [110]

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[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 osteoarthritis[111].

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.

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[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 evidence[114] 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**.

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[114] See footnote 2 above.

Articles cited in the Council's analysis

Appendices

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). 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 B	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.
Appendix C	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).

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).

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 & losuo, T.1997, 'Serum immunoglobulins and the risk of rheumatoid arthritis', *Ann Rheum Dis*, 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', *Int J Toxicol*, vol. 21 pp.333-40.	
14861	Brown, D.P. 1987, 'Mortality of Workers Exposed to Polychlorinated Biphenyls - An Update', *Arch Environ Health*, vol. 42, no. 6, pp. 333-9.	
45909	Carpenter, D.O. 2006, 'Polychlorinated biphenyls (PCBs): routes of exposure and effects on human health', *Reviews on Environmental Health*, 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', *International Immunopharmacology*, 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', *Ann Epidemiol,* vol. 15, no. 10, pp.762-70.	
46526	Dooley, M.A. & Hogan, S.L. 2003, 'Environmental epidemiology and risk factors for autoimmune disease', *Curr Opin Rheumatol,* vol.15, pp.99-103.	
46454	Edwards, C.J. & Cooper, C 2005, 'Early environmental factors and rheumatoid arthritis', *Clin Exp Immunok*, 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', *Am J Ind Med*, vol.13, pp.415-27.	
47835	Eskandari, F. Webster, J.I. & Sternberg, E.M. 2003, 'Neural immune pathways and their connection to inflammatory diseases', *Arthritis Res Ther*, vol. 5, no. 6, pp.251-65.	Applicant

48175	Franchini, M. Ria,l M. Buiatti, E. Bianchi, E. 2004, 'Health effects of exposure to wste incinerator emissions: a reqiew of epidemiological studies', *Ann 1st Super Sanita*, vol. 40, no. 1, pp.101-15.	
47397	Garabrant, D.H. & Dumas, C. 2000, 'Epidemiology of organic solvents and connective tissue disease', *Arthritis Research*, 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', *Arthritis & Rheumatism*, vol. 56, no. 10, pp.3189-201.	
18242	Guo, Y.L. Yu, M.L. Hsu, C.C. Rogan, W.J. 1999, 'Chloracen, Goiter, Arthritis, and Anemia after Polychlorinated Biphenyl Poisoning: 14-Year Follow-Up of the Taiwan Yucheng Cohort', *Environmental Health Perspectives,* 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)', *Am J Ind Med*, 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', *Chemosphere*, 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', *Environ Health*, vol. 6 p.25.	
47839	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.	Applicant & Commissions
30189	Khuder, S.A. Peshimam, A.Z. & Agraharam, S. 2002, 'Environmental risk factors for rheumatoid arthritis', *Reviews on Environmental Health*, vol.17, pp.307-315.	
47943	Kimbrough, R.D. Keouskas, C.A. 2003, 'Human exposure to polychlorinated biphenyls and health effects',*Toxicol Rev,* 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', *Journal of Epidemiology*, 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', *HSMHA Health Reports*, 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', *Chemosphere,* 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', *Biol Pharm Bull,* vol. 25, no.9, pp.1165-8.	Applicant
46247	Lee, D.H. Steffes, M. Jacobs, D.R. Jr. 2007, 'Positive associations of serum contration 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.	Applicant & Commissions
5681	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.	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', *J Occup Environ Med*, vol. 46, no.6, pp.565-76.	
46581	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.	
30227	National Academy of Sciences 2003, 'Gulf War and Health effects: Volume 2. Insecticides and solvents, *National Academy Press, Washington, DC*, 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', *J Toxicol Environ Health, A*, vol. 70, pp.1873-7.	
48955	Okumura, M. 1984, 'Past and current medical states of Yusho patients', *Am J Ind Med*, vol. 5, pp.13-8.	
46618	Oliver, J.E. Silman, A.J. 2006, 'Risk factors for the development of rheumatoid arthritis', *Scand J Rheumatol*, 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', *Occup Environ Med*, vol. 61, pp.233-8.	Commissions
27665	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.	
47827	Ross, G. 2004, 'The public health implications of polychlorinated biphenyls (PCBs) in the environment', *Ecotoxicology and Environmental Safety*, 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', *Environ Health*, 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', *American Journal of Epidemiology*, 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', *Environ Research*, 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', *Arthritis Research & Therapy,* 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', *Environ Health Perspect*, 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', *Environ Health Perspect*, 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', *Environ Health Perspect*, vol. 104, no. 1, pp.52-8.	Applicant
48814	Tryphonas, H. 1995, 'Immunotoxicity of PCBs (Aroclors) in relation to Great Lakes', *Environ Health Perspect*, 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', *Fukouka Igaku Zasshi*, vol. 90, no. 5, pp.147-9 (Abstract)	Applicant

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', *Environ Health Perspect*, vol. 109, no. 3, pp. 265-273.	
47841	World Health Organisation (WHO) 1992, 'Polychlorinated Biphenyls and Terphenyls' (2nd Edition), *Environmental Health Criteria 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', *Ann Occup Hyg*, vol. 50, no. 1, pp.65-73.	
47838	Yoshimura, T. 2003, 'Yusho in Japan', Ind Health, 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', *Environ Health Perspect,* 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. Semin Arthritis Rheum, Vol 27 pp 123-40.
46717	Adams A, Lehman TJA (2005). Update on the pathogenesis and treatment of systemic onset juvenile rheumatoid arthritis. Curr Opin Rheumatol, 17:612-6.
46534	Adams A, Lehman TJA (2005). Update on the pathogenesis and treatment of systemic onset juvenile rheumatoid arthritis. Curr Opin Rheumatol, 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. Placenta, 28:378-82.
27868	Adebajo AO, Hazleman BL (1996). Malaria and rheumatoid arthritis in West Africa. Clinical & Experimental Rheumatology, Vol 14 pp 346.
46527	Adlesic M, Verdrengh M, Bokarewa M, Dahlberg L, et al (2007). Histamine in rheumatoid arthritis. Scand J Immunol, 65:530-7.
30493	Advisory Committee on Immunization Practices (1996). Update: Vaccine side effects, adverse reactions, contraindications, and Precautions. Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR,45/No. RR-12 pp 1-35.
46466	Agrawal S, Misra R, Aggarwal A (2007). Autoantibodies in rheumatoid arthritis: association with severity of disease in established RA. Clin Rheumatol, 26:201-4.
27348	Aho K & Heliovaara M (1998). [Letters, matters arising] Stressful life events and rheumatoid arthritis. Ann Rheum Dis Vol 57:263.
5650	Aho K, Heliövaara M (1993). Alcohol, androgens and arthritis (Letter to the Editor). Ann Rheum Dis, 52(12) pp 897.
46536	Aho K, Heliovaara M (2004). Risk factors for rheumatoid arthritis. Ann Med, 36:242-51.
47840	Aho K, Heliovaara M, Knekt P, Reunanen A, et al (1997). Serum immunoglobulins and the risk of rheumatoid arthritis. Ann Rheum Dis, 56(6): 351-6.
46701	Ahuja SR, Oak J, Sawant S, Kulkarni MV (2003). Arthritis in HIV. Indian Journal of Pediatrics, 70(10):835-6.
27410	Al-Allaf AW, Sanders PA, Ogston SA & Marks JS (2001). A case-control study examining the role of physical trauma in the onset of rheumatoid arthritis. Rheumatology Vol 40:262-266.

46465	Alamanos Y, Drosos AA, Epidemiology of adult rheumatoid arthritis. Autoimmunity Reviews, 4:130-6.
47061	Alamanos Y, Voulgari PV, Drosos AA (2006). Incidence and prevalance of rheumatoid arthritis, based on 1987 American College of Rheumatology criteria: a systematic review. Semin Arthritis Rheum, 36:182-8.
46467	Alamanos Y, Voulgari PV, Drosos AA (2006). Incidence and prevalence of rheumatoid arthritis, based on the 1987 American College of Rheumatology criteria: a systematic review. Semin Arthritis Rheum, 36:182-8.
27357	Alarcon GS (1999). [Letter] The influence of sex on the frequency of erosive disease and extraarticular manifestations in rheumatoid arthritis: comment on the article by Weyand, et al Arthritis & Rheumatism Vol 42:587.
27641	Alarcon GS (1999). The influence of sex on the frequency of erosive disease and extraarticular manifestations in rheumatoid arthritis: comment on the article by Weyand, et al[comment]. [Comment. Letter] Arthritis & Rheumatism. 42(3):587; discussion 589-90.
30667	Al-Awadhi AM, Olushi SO, Al-Zaid NS, et al (1999). Prevalence of hypothyroidsim among Arabs with rheumatoid arthritis. Rheumatology, 38: 472-3.
27582	Albano SA, Santana-Sahagun E, Weisman MH (2001). Cigarette smoking and rheumatoid arthritis. Seminars in Arthritis and Rheumatism, 31(3) pp 146-59.
48957	Aletaha D, Smolen JS (2006). Remission of rheumatoid arthritis: should we care about definitions? Clin Exp Rheumatol, 24(Suppl 43): S45-51.
48768	Aletaha D, Smolen JS (2006). Remission of rheumatoid arthritis: should we care about definitions? Clin Exp Rheumatol, 24 (6 Suppl 43): 45-51.
30268	Al-Refaie FN, Wonke B & Hoffbrand AV (1994). Arthropathy in thalassaemia patients receiving deferiprone. Lancet, 344:262-3.
30578	Alsalameh S, et al (1998). [Letter] New onset of rheumatoid arthritis during interferon Beta-1B treatment in a patient with multiple sclerosis: comment on the case report by Jabaily and Thompson.. Arthritis & Rheumatism 41:754.
27353	Altschuler EL (1999). Parvovirus B19 and the pathogenesis of rheumatoid arthritis: a case for historical reasoning. Lancet Vol354:1026-27.
46436	Alvarez-Lafuente R, Fernandez-Gutierrez B, de MiguelS, Jover JA, et al (2005). Potential relationship between herpes viruses and rheumatoid arthritis: analysis with quantitative real time polymerase chain reaction. Ann Rheum Dis, 64:1357-9.
27613	American Thoracic Society (1997). Adverse effects of crystalline silica exposure. Am J Respir Crit Care Med, 155: 761-8.
30456	Andersson U & Erlandsson-Harris H (2004). HMGB1 is a potent trigger of arthritis. Journal of Internal Medicine, 255:344-350.
46959	Anonymous (2004). Systematic adverse effects of Hepatitis B vaccines are rare. Prescrire International, 13(74): 218-23.
30534	Anonymous (n.d.). Vaccine Adverse Events Reporting System (VAERS) data: Guide to interpreting case report information obtained from the vaccine adverse Event Reporting System. http://www.vaers.org/info.htm.

29080	Anthony DD & Haqqi T M (1999). Collagen-induced arthritis in mice: An animal model to study the pathogenesis of rheumatoid arthritis. Clin Exp Rheumatol Vol 17: 240-244.
29153	Anthony DD & Haqqi TM (1999). Collagen-induced arthritis in mice: an animal model to study the pathogenesis of rheumatoid arthritis. Clin Exp Rheumatol; 17:240-244.
5651	Arnett FC, Edworthy SM, Bloch DA, McShane DJ, et al (1988). The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis & Rheumatism, 31(3) pp 315-324.
48173	Askling J (2007). Malignancy and rheumatoid arthritis. Curr Rheumatol Rep, 9(5): 921-6. [Abstract]
46468	Atta AM, Carvalho EM, Jeronimo SMB, Sousa Atta MLB (2007). Serum markers of rheumatoid arthritis in visceral leishmaniasis: rheumatoid factor and anti-cyclic citrullinated peptide antibody. Journal of Autoimmunity, 28:55-8.
6483	Avila MH, Liang MH, Willett WC, Stampfer MJ, et al (1990). Reproductive factors, smoking, and the risk for rheumatoid arthritis. Epidemiology, Vol 1 pp 285-291.
46469	Balandraud N, Roudier J, Roudier C (2004). Epstein-Barr virus and rheumatoid arthritis. Autoimmunity Reviews, 3:362-7.
47399	Bannwarth B (1996). Drug-induced rheumatic disorders. Rev Rhum (Engl Ed), 63(10): 639-47.
45974	Bannwarth B (2007). Drug-induced musculoskeletal disorders. Drug Safety, 30(1): 27-46.
27362	Barrett JH, Brennan P, Fiddler M & Silman A (2000). Breast-feeding and postpartum relapse in women with rheumatoid and inflammatory arthritis. Arthritis & Rheumatism Vol 43:1010-5.
46533	Bartfai T, Waalen J, Buxbaum JN (2007). Adipose tissue as a modulator of clinical inflammation: does obesity reduce the prevalence of rheumatoid arthritis? J Rheumatol, 34:488-92.
30299	Bartram SA (1998). Experience with methotrexate-associated pneumonitis in northeastern England: comment on the article by Kremer, et al Arthritis & Rheumatism, 41:1327-8.
47301	Barzilai O, Sherer Y, Ram M, Izhaky D, Anaya JM, Shoenfeld Y (2007). Epstein-Barr virus and cytomegalovirus in autoimmune diseases. Are they truly notorious? A preliminary report. Ann NY Acad Sci, 1108:567-77.
27506	Bazzi A, Portnoff K, & Pischel KD (2003). [Letters] Is decaf a decoy? Comment on the article by Mikuls, et al Arthritis & Rheumatism, 48(3):862.
46613	Bazzi A, Portnoff K, Pischel KD (2003). [Comment] Is decaf a decoy? Comment on the article by Mikuls et al. Arthritis & Rhuematism, 48(3):862.
46470	Benedek TG (2006). The history of bacteriologic concepts of rheumatic fever and rheumatoid arthritis. Semin Arthritis Rheum, 36:109-23.
46435	Bengtsson C, Nordmark B, Klareskog L, Lundberg I, Alfredsson L (2005). Socioeconomic status and the risk of developing rheumatoid arthritis: results from the Swedish EIRA study. An Rheum Dis, 64:1588-94.

46573	Benito-Garcia E, Feskanich D, Hu FB, Mandl LA, Karlson EW (2007). Protein, iron, and meat consumption and risk for rheumatoid arthritis: a prospective cohort study. Arth Res Ther, 9:R16.
30269	Berkovitch M, Laxer RM, Inman R, et al (1994). Arthropathy in thalassaemia patients receiving deferiprone. Lancet, 343:1471-2.
47825	Bernard A, Broeckaert F, De Poorter G, De Cock A, et al (2002). The Belgian PCB/Dioxin incident: analysis of the food chain contamination and health risk evaluation. Environmental Research Section A, 88: 1-18.
47826	Bernard A, Fierens S (2002). The Belgian PCB/Dioxin incident: a critical review of health risks evaluations. Int J Toxicol, 21: 333-40.
47398	Bernatsky S, Clarke A, Suissa S (2008). Antimalarial drugs and malignancy: no evidence of a protective effect in rheumatoid arthritis. Ann Rheum Dis, 67(2): 277-8.
5652	Bernelot Moens H, Corstjens A, Boon C (1994). Rheumatoid arthritis is not associated with prior tonsillectomy or appendectomy. Clin Rheumatol, 13(3) pp 483-486.
29133	Bethelot J-M, Bataille R, maugars Y & Prost A (1999). Synovial membrane metaplasia to secondary lymphoid organs: role in the pathogenesis of auto-immune arthritis.Rev Rhum [Eng Ed]; 66:410-418.
46513	Bhatia SS, Majka DS, Kittelson JM, Parrish LA, et al (2007). Rheumatoid factor seropositivity is inversely associated with oral contraceptive use in women without rheumatoid arthritis. Ann Rheum Dis, 66:267-9.
48769	Bombardieri NM, Allessandri C, Labbadia G et al (2004). Role of anti-cyclic citrullinated peptide antibodies in discriminating patients with rheumatoid arthritis from patients with chronic hepatitis C infection-associated polyarticular involvement. Arthritis Res Ther, 6(2): R137-41.
27748	Bond C & Cleland LG (1996). Rheumatoid arthritis: are pets implicated in its etiology? Seminars in Arthritis and Rheumatism 25:308-317.
46434	Borchers AT, Selmi C, Cheema G, Keen CL, et al (2006). Juvenile idiopathic arthritis. Autoimmunity Reviews, 5:279-98.
30536	Bosma TJ, Etherington J, O'Shea S, Corbett K, Cottam F, Holt L, Banatvala JE & Best JM (1998). Rubella virus and chronic joint disease: is there an association? Journal of Clinical Microbiology, 36:3524-6.
46718	Bourikas LA, Sidiropoulos PI, Goulielmos GN, et al (2006). [Letter] Concomitant onset of Graves disease and rheumatoid arthritis after a serious life event. JCR, 12(6):326-7.
46537	Bovin LF, Brynskov J, Hegedus L, Jess T, et al (2007). Gene expression profiling in autoimmune diseases: chronic inflammation or disease specific patterns? Autoimmunity, 40(3):191-201.
27864	Brady TJ (1998). Integration of stress research in rheumatoid arthritis: from Alexander to Zautra and back again. Arthritis Care and Research, Vol 11 pp 77-79.
29079	Breebaart AC, Bijlsma JWJ & van Eden W (2002). 16-year remission of rheumatoid arthritis after unusually vigorous treatment of closed dental foci. Clinical and Experimental Rheumatology Vol20: 555-557.
46532	Brennan F, Beech J (2007). Update on cytokines in rheumatoid arthritis. Curr Opin Rheumatol, 19:296-301.
5653	Brennan P, Silman A (1994). Breast-feeding and the onset of rheumatoid arthritis. Arthritis and Rheumatism, 37(6) pp 808-813.
27593	Brennan P, Silman A (1996)[LETTER]. Role of androgens in the aetioloy of rheumatiod arthritis. Annals of Rheumatic Diseases, 55(6) p 404.

46445	Brenner J, Solitar BM, Golden BD (2000). Rheumatic manifestations of current pharmacopeia. Current Rheumatology Reports, 2:151-5.
41237	Brinton LA, Buckley LM, Dvorkina O, Lubin JH, et al (2004). Risk of connective tissue disorders among breast implant patients. Am J Epidemiol, 160(7): 619-627.
45978	Brogan BL, Olsen NJ (2003). Drug-induced rheumatic syndromes. Current Opinion in Rheumatolgy, 15: 76-80.
5654	Brousset P, Caulier M, Cantagrel A, Dromer C, et al (1993). Absence of Epstein-Barr virus carrying cells in synovial membranes and subcutaneous nodules of patients with rheumatoid arthritis. Ann Rheum Dis, Vol 52 pp 608-609.
14861	Brown DP (1987). Mortality of workers exposed to polychlorinated biphenyls-an update. Environ Health, Vol 42 pp 33-40.
30131	Brown LM, Gridley G, Olsen JH, Mellemkjaer L, Linet MS and Fraumeni JF Jr (1997). Cancer risk and mortality patterns among silicotic men in Sweden and Denmark. J Occup Environ Med, 39:633-638.
5655	Brown MA, Bertouch JV (1994). Rheumatic complications of influenza vaccination. Aust NZ J Med, Vol 24 pp 572-573.
46429	Bruyn GAW; Turesson C, Matteson EL (2004). [Letters] Extraarticular rheumatoid arthritis and drug-induced syndromes: understanding the role of tumor necrosis factor inhibitors. J Rheumatol, 31(7):1461-2.
47405	Buchs N, Chevrel G, Miossec P (1998). Bacillus calmette-guerin induced aseptic arthritis: an experimental model of reactive arthritis. J Rheumatol, 25(9): 1662-5.
46600	Buckley CD (2003). Why does chronic inflammatory joint disease persist? Clin Med, 3(4): 361-6.
46702	Bueso MB, Caballero R, Castro HJ, Barakat A, Bellanti JA (2006). Recurrent infections and joint pain. Allergy Asthma Proc, Vol 27 pp 164-171.
48733	Bugatti S, Cotullo V, Caporali R, Montecucco C (2007). B cells in rheumatoid arthritis. Autoimmunity Reviews, 7: 137-42.
46433	Calabrese LH, Kirchner E, Shrestha R (2005). Rheumatic complications of human immunodeficiency virus infection in the era of highly active antiretroviral therapy: emergence of a new syndrome of immune reconstitution and changing patterns of disease. Seminars in Arthritis and Rheumatism, 35:166-74.
46715	Calcagni E, Elenkov I (2006). Stress system activity, innate and T helper cytokines, and susceptibiolity to immune-related diseases. Ann NY Acad Sci, 1069:62-76.
46432	Caliskan R, Masatlioglu S, Aslan M, Altun S, et al (2005). The relationship between arthritis and human parvovirus B19 infection. Rheumatol Int, 26:7-11.
29961	Callahan LF (2003). Social epidemiology and rheumatic disease. Current Opinion in Rheumatology, 15:110-115.
31980	Calvert GM, Rice FL, Boiano JM, Sheehy JW & Sanderson WT (2003). Occupational and Environmental Medicine, 60:122-129.
46431	Cantaert T, De Rycke L, Bongartz T, Matteson EL, et al (2006). Citrullinated proteins in rheumatoid arthritis. Crucial. . .but not sufficient! Arthritis & Rhuematism,l 54(11):3381-9.

46260	Cantorna MT, Mahon BD (2004). Mounting evidence for vitamin D as an environmental factor affecting autoimmune disease prevalence. Exp Biol Med, 229:1136-42.
30329	Caplan A (1953). Certain unusual radiological appearances in the chest of coal-miners suffering from rheumatoid arthritis. Thorax, 8:29-37.
46430	Caramella C, Avouac J, Sogni P, Puechal X, et al (2007). Association between rheumatoid arthritis and primary biliary cirrhosis. Joint Bone Spine, 74:279-81.
27660	Carette S, Surtees PG, Wainwright NWJ, Khaw K-T, et al (2000). The role of life events and childhood experiences in the development of rheumatoid arthritis. Journal of Rheumatology, Vol 27 pp 2123-30.
46438	Carlens C, Brandt L, Klareskog L, Lampa J, Askling J (2007). The inflammatory reflex and risk for rheumatoid arthritis: a case-control study of human vagotomy. Ann Rheum Dis, 66:414-6.
45909	Carpenter DO (2006). Polychlorinated biphenyls (PCBs): routes of exposure and effects on human health. Reviews on Environmental Health, 21(1) pp 1-23.
46437	Carty SM, Snowden N, Silman AJ (2004). Should infections still be considered as the most likely triggering factor for rheumatoid arthritis? Ann Rheum Dis, 63(Suppl II):ii46-9.
30553	Centers for Disease Control and Prevention (2003). Surveillance Summaries, MMWR, 52 (No. SS-1) January 24.
27671	Cerhan JR, Saag KG, Criswell LA, Merlino LA, Mikuls TR (2002). Blood transusion, alchol use, and anthropometric risk factos for rheumatoid arthritis in older women. J Rheumatol ,Vol 29 pp 246-254.
27503	Cerhan JR, Saag KG, Merlino LA, Mikuls TR, Criswell LA (2003). Antioxidant micronutrients and risk of rheumatoid arthritis in a cohort of older women. American Journal of Epidemiology, Vol 157 pp 345-354.
46463	Cesur M, Ozbalkan Z, Temel MA, Karaarslan Y (2007). Ethnicity may be a reason for lipid changes and high Lp(a) levels in rheumatoid arthritis. Clin Rheumatol, 26:355-61.
46538	Chandrashekara S, Patil R, Vadiraja HS, Shobha A (2006). The incidence of proteus mirabilis infection increases in patients on treatment but does not trigger disease activity. Clin Rheumatol, 25:520-3.
30295	Chazerain P, Meyer O, & Kahn M-F (1992). Rheumatoid arthrtis-like disease after alpha-interferon therapy. Annals of Internal Medicine, 116:427.
30495	Chen RT, Pless R & DeStefano F (2001). Epidemiology of autoimmune reactions induced by vaccination. Journal of Autoimmunity, Vol 16 pp 309-318.
47406	Chevrel G, Zech C, Miossec P (1999). [Comment] Severe uveitis followed by reactive arthritis after bacillus calmette-guerin therapy. J Rheumatol, 26(4): 1011.
46456	Chinniah K, Mody GM, Bhimma R, Adhikari M (2005). Arthritis in association with human immunodeficiency virus infection in black African children: causal or coincidental? Rheumatology, 44:915-20.
46544	Choi HK (2004). Diet and rheumatoid arthritis: red meat and beyond. Arthritis & Rheumatism, 50(12):3745-7.
46714	Choi HK (2005). Dietary risk factors for rheumatic diseases. Curr Opin Rheumatol, 17:141-6.

46531	Choi HK (2005). Dietary risk factors for rheumatic diseases. Curr Opin Rheumatol, 17:141-6.
48172	Clavel G, Grados F, Cayrolle G, Bellony R, et al (1999). Polyarthritis following intravesical BCG immunotherapy (abstract). Rev Rhum, 66(2): 115-8.
27331	Cleland LG, James MJ (1997). Rheumatoid arthritis and the balance of dietary N-6 and N-3 essential fatty acids. British Journal of Rheumatology, 36: 513-4.
30744	Cohen MG, Kevat S, Prowse MV & Ahern MJ (1988). Two distinct quinidine-induced rheumatic syndromes. Ann Intern Med., 108:369-71 [ABSTRACT].
45977	Cohen MG, Prowse MV (1989). Drug-induced rheumatic syndromes. Diagnosis, clinical features and management. Med Toxicol Adverse Drug Exp, 4: 199-218.
46535	Colbert RA (2005). [Comment] Pediatric and heritable disorders. Curr Opin Rheumatol, 17:566-7.
30567	Committee to Assess the Safety and Efficacy of the Anthrax Vaccine (2002). The Anthrax Vaccine: Is It Safe? Does It Work? Institute of Medicine, National Academy Press, Washington DC.
30666	Cooper GS, Miller FW, Germolec DR (2002). Occupational exposures and autoimmune diseases. International Immunopharmacology, 2(2-3): 303-13.
47689	Cope AP, Schulze-Koops H, Aringer M (2007). The central role of T cells in rheumatoid arthritis. Clin Exp Rheumatol, 25(Suppl 46): S4-11.
46458	Costenbader KH, Feskanich D, Mandl LA, Karlson EW (2006). Smoking intensity, duration, and cessation, and the risk of rheumatoid arthritis in women. Am J Med, 119:503-11.
46459	Costenbader KH, Karlson EW (2006). Cigarette smoking and autoimmune disease: what can we learn from epidemiology? Lupus, 15:737-45.
46261	Costenbader KH, Karlson EW (2006). Epstein-Barr virus and rheumatoid arthritis: is there a link? Arthritis Research & Therapy, 8(1):204.
46611	Criswell LA (2004). Familial clustering of disease features: implications for the etiology and investigation of systemic autoimmune disease. Arthritis & Rheumatism, 50(6):1707-8.
46460	Criswell LA, Merlino LA, Cerhan JR, Mikuls TR, et al (2002). Cigarette smoking and the risk of rheumatoid arthritis among postmenopausal women: results from the Iowa Women's Health Study. Am J Med, 112:465-71.
27496	Criswell LA, Merlino LA, Cerhan JR, Mikuls TR, et al (2002). Cigarette smoking and the risk of rheumatoid arthritis among postmenopausal women: results from the Iowa Women's Health Study. Am J Med. 112:465-471.
46441	Criswell LA, Saag KG, Mikuls TR, Cerhan JR, et al (2006). Smoking interacts with genetic risk factors in the development of rheumatoid arthritis among older Caucasion women. Ann Rheum Dis, 65:1163-7.
48770	Cronstein B (2007). Interleukin-6: A Key Mediator of Systemic and local symptoms. Bulletin of the NYU Hospital for Joint Diseases, 65(suppl 1): 11-15

46964	Cruz AAV, Akaishi PMS, Vargas MA, de Paula SA (2007). Association between thyroid autoimmune dysfunction and non-thyroid autoimmune diseases. Ophthalmic Plastic and Reconstructive Surgery, 23(2): 104-108.
27678	Csepregi A, Nemesanszky E, Rojkovich B, Poor G (2001). Rheumatoid arthritis and hepatitis B virus: evaluating the pathogenic link. Journal of Rheumatology, Vol 28 pp 474-477.
30637	Csepregi A, Rojkovich B, Nemesanszky E, et al (2000). [Concise Communication] Chronic seropositive polyarthritis associated with hepatitis B virus-induced chronic liver disease: a sequel of virus persistence. Arthritis & Rheumatism 43:232-3.
48772	Cutolo M (2004). Estrogen metabolites: increasing evidence for their role in rheumatoid arthritis and systemic lupus erythematosus. J Rheumatol, 31(3): 419-421.
46530	Cutolo M (2007). Sex and rheumatoid arthritis: mouse model versus human disease. Arthritis & Rheumistism, 56(1):1-3.
48771	Cutolo M, Capellino S, Sulli A et al (2006). Estrogens and autoimmune diseases. Ann N Y Acad Sci, 1089: 538-547.
27651	Cutolo M, Foppiani L, Prete C, Ballarino P, Sulli A,et al (1999). Hypothalamic-pituitary-adrenocortical axis function in premenopausal women with rheumatoid arthritis not treated with glucocorticoids. J Rheumatol 26:282-8.
46716	Cutolo M, Lahita RG (2005). Estrogens and arthritis. Rheum Dis Clin N Am, 31:19-27.
46712	Cutolo M, Otsa K, Aarkre O, Sulli A (2005). Nocturnal hormones and clinical rhythms in rheumatoid arthritis. Ann NY Acad Sci, 1051:372-81.
47401	Cutolo M, Otsa K, Uprus M, Paolino S, Seriolo B (2007). Vitamin D in rheumatoid arthritis. Autoimmunity Reviews, 7: 59-64.
47393	Cutolo M, Straub RH (2006). Stress as a risk factor in the pathogenesis of rheumatoid arthritis. Neuroimmunomodulation, 13: 277-82.
30452	Cutolo M, Villaggio B, Craviotto C, Pizzorni C, Seriolo B & Sulli A (2002). Sex hormones and rheumatoid arthritis. Autoimmunity Reviews, 1:284-9.
46713	Cutulo M, Capellino S, Sulli A, Serioli B, et al (2006). Estrogens and autoimmune diseases. Ann NY Acad Sci, 1089:438-47.
46462	Cuzzocrea S (2006). Role of nitric oxide and reactive oxygen species in arthritis. Current Pharmaceutical Design, 12:3551-70.
5656	Da Silva JAP (1991). Heat shock proteins: the missing link between hormonal and reproductive factors and rheumatoid arthritis? Ann Rheum Dis, Vol 50 pp 735-739.
5657	Dabbagh AJ, Trenam CW, Morris CJ, Blake DR (1993). Iron in joint inflammation. Ann Rheum Dis, Vol 52 pp 67-73.
46590	Danner S, Sordet C, Terzic J, Donato L, et al (2006). Epidemiology of juvenile idiopathic arthritis in Alsace, France. J Rheumatol, 33:1377-81.
48734	De Roos AJ, Cooper GS, Alavanja MC, Sandler DP (2005). Rheumatoid arthritis among women in the agricultural health study: risk associated with farming activities and exposures. Ann Epidemiol, 15(10): 762-70.
27805	de Souza EP, Segunda PTE, Jose FF, Lemaire D, Santiago M (2001). Rheumatoid arthritis induced by alpha-interferon therapy. Clinical Rheumatology, Vol 20 pp 297-99.

5658	De Vries EGE, Willemse PHB, Biesma B, et al (1991). [Letter] Flare-up of rheumatoid arthritis during GM-CSF treatment after chemotherapy. The Lancet, Vol 338 pp 517-518.
30265	de Vries EGE, Willemse PHB, Biesma B., et al (1991). Flare-up of rheumatoid arthritis during GM-CSF treatment after chemotherapy. Lancet, 338:517-8.
27340	Deighton C (1997). Smoke gets in your joints? Annals of the Rheumatic Diseases Vol 56:453-4.
46705	Deighton C, Criswell LA (2006). Recent advances in the genetics of rheumatoid arthritis. Current Rheumatology Reports, 8:394-400.
46576	Deighton C, Criswell LA, Lum RF, Silman A (2007). Ages of onset suggestive of genetic anticipation in rheumatoid arthritis multicase sibships can be explained by observational bias. Rheumatology, 46:120-3.
31175	Deighton CM, Gray JW, Bint AJ & Walker DJ (1992). Anti-Proteus antibodies in rheumatoid arthritis same-sex sibships. British Journal of Rheumatology, 31:241-5.
5659	Deighton CM, Sykes H, Walker DJ (1993). Rheumatoid arthritis, HLA identity, and age at menarche. Ann Rheum Dis, Vol 52 pp 322-326.
30356	Dekkers JC, Geenen R, Evers AWM, Kraaimaat FW, Bijlsma JWJ & Godaert GIR (2001). Biopsychosocial mediators and moderators of stress-health relationships in patients with recently diagnosed rheumatoid arthritis. Arthritis Care & Research, 45:307-16.
48088	Del Porto F, Lagana B, Biselli R, Donatelli I, et al (2006). Influenza vaccine administration in patients with systemic lupus erythematosus and rheumatoid arthritis. Safety and immunogenicity. Vaccine, 24(16): 3217-23.
27494	del Rincon I, Williams K, Stern MP, Freeman GL & Escalante A (2001). High incidence of cardiovascular events in a rheumatoid arthritis cohort not explained by traditional cardiac risk factors. Arthritis & Rheumatism, 44: 2737-45.
30492	Department of Health, France (2000). Vaccination anti hepatite B mise a jour des donnees et des etudes de pharmacovigilance. [in French] Http://agmed.sante.gouv.fr/pdf/6/vhbrap.pdf
27661	Desein PH, Stanwix AE, Moomal Z; Symmons D, Harrison B (2001)[LETTERS]. Rheumatoid arthritis and cardiovascular disease may share similar risk factors. Rheumatology, Vol 40 pp 703-4.
46444	di Fazano CS, Bertin P, Vergne P, Bonnet C, Treves R (2001). The pharmacological management of drug-induced rheumatic disorders. Expert Opin Pharmacother, 2(10):1623-31.
30665	Ding M, Chen F, Shi X, Yucesoy B, Mossman B, Vallyathan V (2002). Diseases caused by silica: mechanisms of injury and disease development. International Immunopharmacology, Vol 2 pp 173-182.
30455	Distler JHW, Wenger RH, Gassman M, Kurowska M, Hirth A, Gay S & Distler O (2004). Physiologic responses to hypoxia and implications for hypoxia-inducible factors in the pathogenesis of rheumatoid arthritis. Arthritis & Rheumatism, 50:10-23.
46455	Dixon WG, Symmons DPM (2005). Does early rheumatoid arthritis exist? Best Practice & Research Clinical Rheumatology, 19(1):37-53.
29960	Dooley MA, & Hogan SL (2003). Environmental epidemiology and risk factors for autoimmune disease. Current Opinion in Rheumatology. 15:99-103.

46526	Dooley MA, Hogan SL (2003). Environmental epidemiology and risk factors for autoimmune disease. Curr Opin Rheumatol, 15:99-103.
46554	Doran M (2007). [Comment] Rheumatoid arthritis and diabetes mellitus: evidence for an association? J Rheumatol, 34(3):460-2.
46525	Doran M (2007). Rheumatoid arthritis and diabetes mellitus: evidence for an association? J Rheumatol, 34(3):460-2.
46524	Doran MF, Crowson CS, O'Fallon WM, Gabirel SE (2004). The effect of oral contraceptives and estrogen replacement therapy on the risk of rheumatoid arthritis: a population based study. J Rheumatol, 31:207-13.
27499	Doran MF, Pond GR, Crowson CS, O'Fallon WM & Gabriel SE (2002). Trends in incidence and mortality in rheumatoid arthritis in Rochester, Minnesota, over a forty-year period. Arthritis & Rheumatism, 46:625-631.
27664	Doube A (2000). [Letter] Vitamin D deficiency and rheumatoid arthritis. New Zealand Medical Journal, 113(1112) pp 259.
27616	Douglas WAC (1997). [Letter] Ross River virus disease and rheumatoid arthritis. MJA 167:229-30.
30291	Duell PB & Chestnut CH (1991). Exacerbation of rheumatid arthritis by sodium fluoride treatment of osteoporosis. Arch Intern Med, 151:783-4.
5660	Duell PB, Chesnut CH (1991). Exacerbation of rheumatoid arthritis by sodium fluoride treatment of osteoporosis. Arch Intern Med, Vol 151 pp 783-784.
46453	Eaton WW, Byrne M, Ewald H, Mores O, et al (2006). Association of schizophrenia and autoimmune diseases: linkage of Danish national registers. Am J Psychiatry, 163(3):521-8.
30581	Ebringer A, Corbett M, Macafee Y, Baron R, et al (1985) Antibodies to proteus in rheumatoid arthritis. Lancet, August 10:305-7.
31174	Ebringer A, Cox NL, Abuljadayel I, Ghuloom M, et al (1988). Klebsiella antibodies in ankylosing spondylitis and Proteus antibodies in rheumatoid arthritis. British Journal of Rheumatology, 27 (Suppl II): 72-85.
46580	Ebringer A, Rashid T (2006). Rheumatoid arthritis is an autoimmune disease triggered by proteus urinary tract infection. Clin Dev Immunol, 13(1):41-8.
31179	Ebringer A, Rashid T, Wilson C (2003). Rheumatoid arthritis: proposal for the use of anti-microbial therapy in early cases. Scand J Rheumatol, 32: 2-11.
46454	Edwards CJ, Cooper C (2005). Early environmental factors and rheumatoid arthritis. Clin Exp Immunok, 143:1-5.
46439	Edwards CJ, Goswami R, Goswami P, Syddall H, et al (2006). Growth and infectious exposure during infancy and the risk of rheumatoid factor in adult life. Ann Rheum Dis, 65:401-4.
5661	Einsele H, Steidle M, Müllur CA, Fritz P, et al (1992). Demonstration of cytomegalovirus (CMV) DNA and anti-CMV response in the synovial membrane and serum of patients with rheumatoid arthritis. J Rheumatol, Vol 19 pp 677-681.
48774	El Mahou S, Popa L, Constantin A et al (2006). Remitting seronegative symmetrical synovitis pitting oedema after BCG instillation. Clin Rheumatol, 25(4): 566-67.
46706	Elewaut D (2005). Natural killer T cells and rheumatoid arthritis: friend or foe? Arthritis Research &Therapy, 7(2):88-9.
46579	Elkayam O (2006). Safety and efficacy of vaccination against influenza in patients with rheumatoid arthritis. Clin Dev Immunol, 13(2-4):349-51.

30489	Elkayam O, Paran D, Caspi D, Litinsky I, Yaron M, Charboneau D, and Rubins JB (2002). Clinical Infectious Diseases, 34:147-53.
27504	Elkayam O, Yaron M & Caspi D (2002). Safety and efficacy of vaccination against hepatitis B in patients with rheumatoid arthritis. Annals of the Rheumatic Diseases, 61:623-5.
48773	Elo O, Vuojolahti P, Janhunent H et al (1985). Recent PCB accidents in Finland. Environmental Health Perspectives, 60: 315-19
47832	Emmett EA, Maroni M, Schmith JM, Levin BK, Jefferys J (1988). Studies of transformer repair workers exposed to PCBs: I. study design, PCB concentrations, questionnaire, and clinical examination results. Am J Ind Med, 13: 415-27.
47835	Eskandari F, Webster JI, Sternberg EM (2003). Neural immune pathways and their connection to inflammatory diseases. Arthritis Res Ther, 5(6): 251-65.
46447	Etzel CJ, Chen WV, Shepard N, Jawaheer D, et al (2006). Genome-wide meta-analysis for rheumatoid arthritis. Hum Genet, 119:634-41.
5662	Euller-Ziegler L, Ziegler G, Ramaioli A, Maurin H (1967). Life events and disease activity in rheumatoid arthritis. Arthritis and Rheumatism, 34(S) pp 179.
30355	Evers AWM, Kraaimaat FW, Geenen R, Jacobs JWG & Bijlsma JWJ. (2003). Stress-vulnerability factors as long-term predictors of disease activity in early rheumatoid arthritis. Journal of Psychosomatic Research, 55:293-302.
32939	Fantini F, Gattinara M, Gerloni M, et al (1992). Severe anemia associated with active systemic-onset juvenile rheumatoid arthritis successfully treated with recombinant human erythropoietin: a pilot study. Arthritis Rheum 35:724-6.
48775	Feitsma AL, Worthington J, Van der Helm-van Mil HM, et al (2007). Protective effect of noninherited maternal HLA-DR antigens on rheumatoid arthritis development. PNAS, 104(50): 19966-70.
31978	Fields S (1997). Exposing ourselves to art. EHP 105(3):284.
30235	Firestein GS (2002). Rheumatoid arthritis. Scientific American, Rheumatology:II Rheumatoid Arthritis:1-16.
46523	Firestein GS (2005). Immunologic mechanisms in the pathogenesis of rheumatoid arthritis. J Clin Rheumatol, 11:S39-44.
46732	Firestein GS (2005). Immunologic mechanisms in the pathogenesis of rheumatoid arthritis. JCR, 11(3):S39-44.
47403	Fomin I, Caspi D, Levy V, Varsano N, Shalev Y, et al (2006). Vaccination against influenza in rheumatoid arthritis: the effect of disease in modifying drugs, including TNFa blockers. Ann Rheum Dis, 65: 191-4.
48776	Fonnum F, Mariussen E, Reistad T (2006). Molecular mechanisms involved in the toxic effects of polychlorinated biphenyls (PCBs) and brominated flame retardants (BFRs). J Toxicol Environ Health A, 69(1-2): 21-35.
47944	Fonnum F, Mariussen E, Reistad T (2006). Molecular mechansisms involved in the toxic effects of polychlorinated biphenyls (PCBs) and brominated flame retardants (BFRs). Journal of Toxicology and Environmental Health, Part A, 69: 21-35.
46711	Fontecchio G, Fioroni MA, Azzarone R, Battistoni C, et al (2007). Genetic predisposition to rheumatoid arthritis in a Tuscan (Italy) ancient human remain. Int J Immunopathol Pharmacol, 20(1):103-9.

31182	Ford DK (1997). Understanding rheumatoid arthritis. Journal of Rheumatology, 24:1464-6.
27866	Ford DK ,Tingle AJ (1996). Lymphocytes from site of disease indicate probable etiology of "infective-immune" diseases such as rheumatoid arthritis. Infectious Agents & Disease, Vol 5 pp 223-230.
46528	Forsblad d'Elia H, Larsen A, Mattsson L-A, Waltbrand E, et al (2003). Influence of hormone replacement therapy on disease progression and bone mineral dinsity in rheumatoid arthritis. J Rheumatol, 30:1456-63.
48175	Franchini M, Rial M, Buiatti E, Bianchi E (2004). Health effects of exposure to waste incinerator emissions: a review of epidemiological studies. Ann 1st Super Sanita, 40(1): 101-15.
47688	Franco-Paredes C, Diaz-Borjon A, Senger MA, et al (2006). The ever-expanding association between rheumatologic diseases and tuberculosis. Am J Med, 119: 470-7.
46448	Franssila R (2006). Viral causes of arthritis. Best Practice & Research Clinical Rheumatology, 20(6):1139-57.
46449	Fryzek JP, Holmich L, McLaughlin JK, Lipworth L, et al (2007). A nationwide study of connective tissue disease and other rheumatic conditions among Danish women with long-term cosmetic breast implantation. Ann Epidemiol, 17:374-9.
17306	Fukuda K, Nisenbaum R, Stewart G, Thompson WW, Robin L, Washko RM, et al (1998). Chronic multisymptom illness affecting air force veterans of the Gulf war. JAMA, 280(11) pp 981-988.
20350	Fulco CE, Liverman CT, Sox HC [Eds] (2000) Gulf War and Health: Volume 1 - Depleted Uranium, Pyridostigmine Bromide, Sarin, Vaccines. Institute of Medicine, National Academy Press, Washington DC.
27676	Gabriel SE (2001). The epidemiology of rheumatoid arthritis. Rheumatic Disease Clinics of North America 27:269-281.
20608	Gabriel SE, Crowson CS & O'Fallon WM (1999). Comorbidity in arthritis. Journal of Rheumatology Vol 26 pp 2475-9.
27355	Gabriel SE, Crowson CS & O'Fallon WM (1999). The epidemiology of rheumatoid arthritis in Rochester, Minnesota, 1955-1985. Arthritis & Rheumatism Vol 42:415-20.
44485	Garabrant DH, Dumas C (2000). Epidemiology of organic solvents and connective tissue disease. Arthritis Res, 2(1) pp 5-15.
47397	Garabrant DH, Dumas C (2000). Epidemiology of organic solvents and connective tissue disease. Arthritis Research, 2: 5-15.
46578	Garcia-Carrasco M, Escarcega RO (2006). Extrahepatic autoimmune manifestations of chronic hepatitis C virus infection. Annals of Hepatology. 5(3):161-3.
46697	Garcia-Patos V (2007). Rheumatoid nodule. Seminars in Cutaneous Medicine and Surgery, 26:100-7.
48777	Garcia-Poma A, Segami MI, Mora CS, et al (2007). Obesity is independently associated with impaired quality of life in patients with rheumatoid arthritis. Clin Rheumatol., 26(11): 1831-35.
31181	Gaston JSH (2001). Infection in the aetiology of spondyloarthropathies. Clin Med JRCPL, 1:104-7.
46730	Geenen R, van Middendorp H, Bijlsma JWJ (2006). The impact of stressors on health status and hypothalamic-pituitary-adrenal axis and autonomic nervous system responsiveness in rheumatoid arthritis. Ann NY Acad Sci, 1069:77-97.

30532	Geier DA & Geier MR (2002). A one year followup of chronic arthritis following rubella and hepatitis B vaccination based upon analysis of the Vaccine Adverse Events Reporting System (VAERS) database. Clinical and Experimental Rheumatology, 20:767-771.
46514	Geier DA, Geier MR (2005). A case-control study of serious autoimmune adverse events following hepatitis B immunization. Autoimmunity, 38(4):295-301.
48778	Geier MR, Geier DA (2004). A case-series of adverse events, positive re-challenge of symptoms, and events in identical twins following hepatitis B vaccination: analysis of the Vaccine Adverse Event Reporting System (VAERS) database and literature review.
48174	Geier MR, Geier DA, Zahalsky AC (2003). A review of hepatitis B vaccination. Expert Opin Drug Saf, 2(2): 113-22.
27335	Geirsson AJ & Onundarson PT (1996). [Letter - Author's reply] G-CSF and exacerbation of rheumatoid arthritis. American Journal of Medicine Vol100:666.
46450	Gergely P Jr, Perl A, Poor G (2006). Possible pathogenic nature of the recently discovered TT virus: does it play a role in autoimmune rheumatic diseases? Autoimmunity Reviews, 6:5-9.
46262	Giakoumi X, Tsironi M, Floudas C, Polymeropoylos E, et al (2005). Rheumatoid arthritis in Thalassemia intermedia: coincidence or association? IMAJ, 7:667-9.
27673	Gilroy CB, Keat A, & Taylor-Robinson D (2001). The prevalence of Mycoplasma fermentans in patients with inflammatory arthritides. Rheumatology 40:1355-1358.
32234	Giordano N, Borghui C, Sancasciani S, et al (1990). Efficacy and safety of an iron chelating agent, deferoxamine, in patients with rheumatoid arthritis and hyposideremic anemia. Current Therapeutic Research, 48:959-68.
27369	Glaser JH (2000). [Letter] Coffee consumption, RF, and the risk of RA. Ann Rheum Dis Vol 59:1000.
48735	Gold LS, Ward MH, Dosemeci M, De Roos AJ (2007). Systemic autoimmune disease mortality and occupational exposures. Arthritis & Rheumatism, 56(10): 3189-201.
46522	Goldacre MJ, Wotton CJ, Seagroatt V, Yeates D (2007). Immune-related disease before and after vasectomy: an epidemiological database study. Human Reproduction, 22(5):1273-8.
46720	Gorman JD (2006). Smoking and rheumatoid arthritis: another reason to just say no. Arthritis & Rheumatism, 54(1):10-3.
46461	Gorman JD (2006). Smoking and rheumatoid arthritis: another reason to just say no. Arthritis & Rheumatism, 54(1):10-13.
46451	Gorman JD, Lum RF, Chen JJ, Suarez-Almazor ME, et al (2004). Impact of shared epitope genotype and ethnicity on erosive disease. Arthritis & Rheumatism, 50(2):400-12.
46521	Gossec L, Baro-Riba J, Bozonnat M-C, Daures J-P, et al (2005). Influence of sex on disease severity in patients with rheumatoid arthritis. J Rheumatol, 32:1448-51.
27408	Grant WB (2000). The role of meat in the expression of rheumatoid arthritis. British Journal of Nutrition,Vol 84 pp 589-595.
27383	Grober SE (2001). [Comment] Coffee or decaff? Ann Rheum Dis, 60: 87.

5663	Gross K, Combe C, Krüger K, Schattenkirchner (1995). Arthritis after hepatitis B vaccination: report of three cases. Scand J Rheumatol, Vol 24 pp 50-52.
18242	Guo YL, Yu ML, Hsu CC, Rogan WJ (1999). Chloracne, goiter, arthritis, and anemia after polychlorinated biphenyl poisoning: 14-year follow-up of the Taiwan Yucheng cohort. Environmental Health Perspectives, 107(9): 715-719.
40028	Gustavsson P, Hogstedt C (1997). A cohort study of Swedish capacitor manufacturing workers exposed to polychlorinated biphenyls (PCB's). Am J Ind Med, 32(3): 234-9.
47828	Hagmar L, Wallin E, Vessby B, Jonsson BAG, Bergman A, Rylander L (2006). Intra-individual variations and time trends 1991-2001 in human serum levels of PCB, DDE and hexachlorobenzene. Chemosphere, 64: 1507-13.
32232	Hahtola PA, Jarvenpaa RE, Lounatmaa K, et al (2000). Hard metal alveolitis accompanied by rheumatoid arthritis. Respiration, 67:209-12.
5664	Hakala M, Ilonen J, Reijonen H, Knip M, et al (1992). No association between rheumatoid arthritis and insulin dependent diabetes mellitus: an epidemiologic and immunogenic study. J Rheumatol, Vol 19 pp 856-858.
30742	Hamilton JD, Thomson EA, Porter D, Hunter JA, Madhok R and Capell HA (2000). Lifestyle influences on outcome in rheumatoid arthritis. Sco Med J., 45:137-139.
46729	Harbuz MS, Richards LJ, Chover-Gonzalez AJ, et al (2006). Stress in autoimmune disease models. Ann NY Acad Sci, 1069:51-61.
48780	Harel-Meir M, Sherer Y, Shoenfeld Y (2007). Tobacco smoking and autoimmune rheumatic diseases. Nat Clin Pract Rheumatol, 3(12): 707-15.
27489	Harrison BJ (2002). Influence of cigarette smoking on disease outcome in rheumatoid arthritis. Curr Opin Rheumatol, 14:93-7.
27580	Harrison BJ, Silman AJ (2000)[EDITORIAL]. Does smoking influence disease outcome in patients with rheumatoid arthritis? Journal of Rheumatology, 27(3) pp 569-70.
30307	Harrison BJ, Silman AJ, Barrett EM, Scott DGI, & Symmons DPM (1997). Presence of psoriasis does not influence the presentation or short term outcome of patients with early inflammatory polyarthritis. Journal of Rheumatology, 24:1744-9.
27493	Harrison BJ, Silman AJ, Wiles NJ, Scott DGI, & Symmons DPM (2001). The association of cigarette smoking with disease outcome in patients with early inflammatory polyarthritis. Arthritis & Rheumatism, 44:323-330.
48781	Harrison BJ, Thomson W, Pepper L, et al (1997). Patients who develop inflammatory polyarthritis (IP) after immunization are clinically indistinguishable from other patients with IP. Br J Rheumatol, 36(3): 366-69.
49012	Harrison's Online (2007). Rheumatoid arthritis. Part 13, Section 2, Chapter 301.
45973	Hart FD (1984). Drug-induced arthritis and arthralgia. Drugs, 28: 347-354.
5665	Haugen MA, Kjeldsen-Kragh J, Førre Ø (1994). A pilot study of the effect of an elemental diet in the management of rheumatoid arthritis. Clinical and Experimental Rheumatology, Vol 12 pp 275-279.

5666	Hazenberg MP, Klasen IS, Kool J, Ruseler-van Embden JGH, Severijnen AJ (1992). Are intestinal bacteria involved in the etiology of rheumatoid arthritis? APMIS, Vol 100 pp 1-9.
5667	Hazes JMW, Dijkmans BAC, Vandenbroucke JP, de Vries RPP, Cats A (1990). Lifestyle and the risk of rheumatoid arthritis: cigarette smoking and alcohol consumption. Ann Rheum Dis, Vol 49 pp 980-982.
5668	Hazes JMW, van Zeben D (1991). Pregnancy and its effect on the risk of developing rheumatoid arthritis. Ann Rheum Dis, Vol 50 pp 71-74.
5669	Heath CW, Fortin PR (1992). Epidemiologic studies of rheumatoid arthritis: future directions. J Rheumatol, 19(S 32) pp 74-79.
27712	Heikkila R, Aho K, Heliovaara M, Knekt P, Reunanen A, Aromaa A, Leino A & Palosuo T (1998). Serum androgen-anabolic hormones and the risk of rheumatoid arthritis. Ann Rheum Dis 57:281-285.
48782	Heilmann C, Grandjean P, Weihe P, et al (2006). Reduced antibody responses to vaccinations in children exposed to polychlorinated biphenyls. PLoS Med., 3(8): 1352-9.
48956	Heilmann C, Grandjean P, Weihe P, et al (2006). Reduced antibody responses to vaccinations in children exposed to polychlorinated biphenyls. PLoS Medicine, 3(8): 1352-9.
5670	Heliövaara M, Aho K, Aromaa A, Knekt P, Reunanen A (1993). Smoking and risk of rheumatoid rrthritis. J Rheumatol, 20(11) pp 1830-1835.
27372	Heliovaara M, Aho K, Knekt P, Impivaara O, Reunanen A, Aromaa A (2000). Coffee consumption, rheumatoid factor, and the risk of rheumatoid arthritis. Ann Rheum Dis, 59: 631-5.
27370	Heliovaara M, Aho K, Knekt P, Reunanen A & Aromaa A (2000). [Comment] Coffee consumption, RF, and the risk of RA. Ann Rheum Dis, 59: 1000.
30306	Heliovaara M, Aho K, Knekt P, Reunanen A, Aromaa A (1996). Serum cholesterol and risk of rheumatiod arthritis in a cohort of 52 800 men and women. British Journal of Rheumatology, 35: 255-7.
27594	Heliovaara M, Aho K, Knekt P, Reunanen A, Aromaa A (1996). Serum cholesterol and the risk of rheumatoid arthritis in a cohort of 52,800 men and women. British Journal of Rheumatology, 35(3) pp 255-7.
27379	Heliovaara M, Aho K, Knekt P, Reunanen A, Aromaa A (2001). [Comment] Coffee consumption, RF, and the risk of RA. Ann Rheum Dis, 60: 541.
27384	Heliovaara M, Aho K, Knekt P, Reunanen A, et al (2001). [Comment] Coffee or decaff? Ann Rheum Dis, 60: 87
5671	Heliövaara M, Aho K, Reunanen A, Knekt P, Aromaa A (1995). Parity and risk of rheumatoid arthritis in Finnish women. Br J Rheumatology, 34(7) pp 625-628.
48456	Herrick RF, Meeker JD, Hauser R, Altshul L, Weymouth GA (2007). Serum PCB levels and congener profiles among US construction workers. Environ Health, 6:25.
47945	Hertz-Picciotto I, Park H-Y, Dostal M, Kocan A, et al (2008). Prenatal exposures to persistent and non-persistent organic compounds and effects on immune system development. Basic Clin Pharmacol Toxicol. 102(2):146-54.
46442	Hess EV (1998). [Comment] Minocycline and autoimmunity. Clin Exp Rheumatol, 16:519-21.

27648	Hirsch R, Lin J-P, Scott Jr WM, Ma LD, Pillemer SR, Kastner DL, Jacobsson LH, Bloch DA, Knowler WC, Bennett PH, & Bale SJ (1998). Rheumatoid arthritis in the Pima Indians. Arthritis & Rheumatism 41:1464-9.
48457	Hochberg MC, Johnston SS, John AK (2008). The incidence and prevalence of extra-articular and systemic manifestations in a cohort of newly-diagnosed patients with rheumatoid arthritis between 1999 and 2006. Curr Med Res Opin, 24(2): 469-80.
48958	Hochberg MC, Johnston SS, John AK (2008). The incidence and prevalence of extra-articular and systemic manifestations in a cohort of newly-diagnosed patients with rheumatoid arthritis between 1999 and 2006. Current Medicine Research and Opinion, 24(2): 469-80.
27597	Hoffman RW, O'Sullivan FX, Schafermeyer KR, Moore TL, et al (1997). Mycoplasma infection and rheumatoid arthritis. Analysis of their relationship using immunoblotting and an ultrasensitive polymerase chain reaction detection method. Arthritis & Rheumatism, 40(7) pp 1219-28.
46510	Holdke B, Karmaus W, Kruse H (1998). Body burden of polychlorinated biphenyl compounds in whole blood of 7-10-7ear-old children in the area of hazardous wast incineration facility. Gesundheitswesen, 60(8-9): 505-12. [Abstract]
48783	Holick M (2004). Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers and cardiovascular disease. Am J Clin Nutr, 80(suppl): 1678S-88S.
46699	Holmdahl R (2007). Nature's choice of genes controlling chronic inflammation. Ernst Schering Foundation Symposium Proceedings, 4:1-15.
48759	Holmdahl R, Lorentzen JC, Lu S, Olofsson P, et al (2001). Arthritis induced in rats with non-immunogenic adjuvants as models for rheumatoid arthritis. Immunological Reviews, 184: 184-202.
48786	Holoshitz J, Ling S (2007). Nitric oxide signaling triggered by the rheumatoid arthritis shared epitope: a new paradigm for MHC disease association? Ann N Y Acad Sci, 1110: 73-83.
18836	Hotopf M, David A, Hull L, Unwin C, Wessely S (2000). Role of vaccinations as risk factors for ill health in veterans of the Gulf war: cross sectional study. BMJ, Vol 320 (7246) pp 1363-7.
27345	Houssien D, Scott DL & Jonsson T (1998). [Letters, Matters arising] Smoking, rheumatoid factors, and rheumatoid arthritis. Ann Rheum Dis Vol57:175-6.
46719	Houtman PM, Jansen TL, Blanken R (2006). [Letter] Anaphylactic reaction in a patients with rheumatoid arthritis: a rare side effect of methotrexate with etanercept as a provoking factor. JCR, 12(6):321-2.
46520	Hsu FC, Starkebaum G, Boyko EJ, Dominitz JA (2003). Prevalence of rheumatoid arthritis and hepatitis C in those age 60 and older in US population based study. J Rheumatol, 30:455-8.
47404	Huber LC, Stanczyk J, Jungel A, Gay S (2007). Epigenetics in inflammatory rheumatoid diseases. Arthritis & Rheumatism, 56(11): 3523-31.
46708	Huggett MT, Armstrong R (2006). Adalimumab-associated pulmonary fibrosis. Rheumatology, 45:1312-3.

48787	Huizinga TW, Amos CI, van der Helm-van Mil, et al (2005). Refining the complex rheumatoid arthritis phenotype based on specificity of the HLA-DRBI shared epitope for antibodies to citrullinated proteins. Arthritis Rheum, 52(11): 3433-38.
29964	Huizinga TWJ (2003). Genetics in rheumatoid arthritis. Best Practice & Research Clinical Rheumatology, 17:703-716.
46880	Husman TM, Luosujarvi RA (2004). [Letter] Reply to letter by Lange. Clin Rheumatol, 23: 567.
27495	Hutchinson D (2001). [Comment] Are cases of rheumatoid arthritis in smokers and lifelong nonsmokers representative of different rheumatoid disease processes? Arthritis & Rheumatism, 44: 2942-3.
27573	Hutchinson D, Lynch MP, Moots RJ, et al (2001). [Comment] The influence of current cigarette smoking on the age of onset of rheumatoid arthritis (RA) in individuals with sporadic and familial RA. Rheumatology, 40: 1068-70.
27477	Hutchinson D, Moots R (2001). [Comment] Cigarette smoking and severity of rheumatoid arthritis. Rheumatology, 40: 1426-7.
27378	Hutchinson D, Moots R (2001). [Comment] Coffee consumption, RF, and the risk of RA. Ann Rheum Dis, 60: 540-1.
27381	Hutchinson D, Shepstone L, Moots R, Lear JT, Lynch MP (2001). Heavy cigarette smoking is strongly associated with rheumatoid arthritis (RA), particularly in patients without a family history of RA. Ann Rheum Dis, Vol 60 pp 223-7.
27865	Huyser B, Parker JC (1998). Stress and rheumatoid arthritis: an integrative review. Arthritis Care ad Research, Vol 11 pp 135-145.
46440	Hvatum M, Kanerud L, Hallgren R, Brandtzaeg P (2006). The gut-joint axis: cross reactive food antibodies in rheumatoid arthritis. Gut, 55:1240-7.
27646	Iannello S, Camuto M, Cantarella S, Cavaleri A, Ferriero P, Leanza A, Milazzo P & Belfiore F (2002). Rheumatoid syndrome associated with lung interstitial disorder in a dental technician exposed to ceramic silica dust. A case report and critical literature review. Clin Rheumatol 21:76-81.
46695	Iannone F, Lapadula G (2003) [Letter] Human immunodeficiency virus-1 and rheumatoid arthritis. J Rheumatol, 30(10):2294-5.
46567	Iannone F, Lapadula G; Wegrzyn, Miossec P (2003). [Letters] Human immunodeficiency virus-1 and rheumatoid arthritis: a puzzling association. J Rheumatol, 30(10):2294-5.
47839	Illinois Department of Public Health and the University of Chicago: Final Report. The La Salle electrical utilities company morbidity II study. Obtained from: http://hdl.handle.net/123456789/5549
46519	Inanc M (2007). Very early 'rheumatoid' arthritis cohorts: limited by selection. Rheumatology, 46:185-7.
30411	Independent Review Group (n. d.). The report of the Independent Review Group: Silicone Gel Breast Implants. What is Silicone? Http:??www.silicone-review.gov.uk/silicone/index.htm.
5672	Inman RD (1991). Infectious etiology of rheumatoid arthritis. Rheumatic Diseases Clinics of Nth America, 17(4) pp 859-870.
30271	Inoue K-I, Kawahito Y, Sano H (2000). Troglitazone as an anti-inflammatory agent. Arch Intern Med, 160: 3004.

27352	Inoue K-I, Kawahito Y, Sano H (2000). Troglitazone as an anti-inflammatory agent. Arch Intern Med, 160: 3004.
47578	Institute of Medicine (2007). Veterans and Agent Orange: update 2006. Committee to review the health effects in Vietnam veterans of exposure to herbicides (sixth biennial update), The National Academic Press, Washington DC. [PREPUBLICATION COPY: Uncorrected Proofs]
48788	Ionnou Y, Isenberg DA (2000). Current evidence for the induction of autoimmune rheumatic manifestations by cytokine therapy. Arthritis Rheum, 43(7): 1431-42.
30491	Iyngkaran P, Limaye V, Hill C, Henderson D, Pile KD, & Rischmueller M (2003). Rheumatoid vasculitis following influenza vaccination. Rheumatology, 42:907-9.
46452	Jaakkola JJK, Gissler M (2005). Maternal smoking in pregnancy as a determinant of rheumatoid arthritis and other inflammatory polyarthropathies during the first 7 years of life. Int J Epidemiol, 34:664-71.
30745	Jabaily JA & Thompson JS (1997). Effects of interferon beta-1B in rheumatoid arthritis: a case report. Arthritis Rheum, 40:1370 [ABSTRACT].
27347	James WH (1997). [Comment] Further evidence that low androgen values are a cause of rheumatoid arthritis: the response of rheumatoid arthritis to seriously stressful life events. Ann Rheum Dis, 56: 566.
27349	James WH (1998). [Letters, matters arising-Author's reply] Stressful life events and rheumatoid arthritis. Ann Rheum Dis, Vol 57 pp 263.
30560	Janowsky EC, Kupper LL & Hulka BS (2000). Meta-analyses of the relation between silicone breast implants and the risk of connective-tissue diseases. N Engl J Med, 342:781-90.
46518	Janssens ACJW, Steyerberg EW, Jiang Y, Habbema JDF, et al (2006). Value of the HLA-DRB1 shared epitope for predicting radiographic damage in rheumatoid arthritis depends on the individual patient risk profile. J Rheumatol, 33:2383-9.
46428	Jarrett SJ, Cunnane G, Conaghan PG, Bingham SJ, et al (2003). Anti-tumor necrosis factor-a therapy-induced vasculitis: case series. J Rheumatol, 30: 2287-91.
46577	Jarvis JN, Petty HR, Tang Y, Frank MB, et al (2006). Evidence for chronic, peripheral activation of neutrophils in polyarticular juvenile rheumatoid arthritis. Arthritis Research & Therapy, 8(5):R154.
27501	Jawaheer D & Gregersen PK (2002). The search for rheumatoid arthritis susceptibility genes: a call for global collaboration. Arthritis & Rheumatism, 46:582-4.
46517	Jawaheer D, Lum RF, Gregersen PK, Criswell LA (2006). Influence of male sex on disease phenotype in familial rheumatoid arthritis. Arthritis & Rheumatism, 54(10):3087-94.
27650	Jefferies W McK (1998). The etiology of rheumatoid arthritis. Medical Hypotheses 51:111-4.
46893	Jessop DS, Richards LJ, Harbuz MS (2004). Effects of stress on inflammatory autoimmune disease: destructive or protective? Stress, 7(4): 261-6.
46728	Johannesson M, Hultqvist M, Holmdahl R (2006). Genetics of autoimmune diseases: a multistep process. CTMI, 305:259-76.

30302	Johnson DM, Hayat SQ, & Burton GV (1999). Rheumatoid arthritis complicating adjuvant Interferon-alpha therapy for malignant melanoma. Journal of Rheumatology, 26:1009-10.
46575	Johnson SM, Bruckner F, Collins D (2007). Distribution of mycoplasma pneumoniae and mycoplasma salivarium in the synovial fluid of arthritis patients. J Clin Microbiol, 45(3):953-7.
46529	Jolly M (2004). [Comment] Article by Forsblad d'Elia et al. J Rheumatol, 31(7):1462-3.
46446	Jolly M, Curran JJ (2004). Chlamydial infection preceding the development of rheumatoid arthritis: a brief report. Clin Rheumatol, 23:453-5.
27576	Jonsson T, Thorsteinsson J, Valdimarsson H (1998). Does smoking stimulate rheumatoid factor production in non-rheumatism individuals? APMIS, 106(10) pp 970-4.
32689	Joshi VK (2003) Squamous cell carcinomas of the head and neck. BMJ Vol 326: 282-3.
47302	Joshi VR, Balakrishnan C (1994). Drug induced rheumatic disorders. JAPI, 42(11): 895-8.
30292	Joyce DA, Will RK, Hoffman DM, Laing B, & Blackbourn SJ (1991). Exacerbation of rheumatoid arthritis in patients treated with methotrexate after administration of folinic acid. Annals of the Rheumatic Diseases, 50:913-4.
5673	Julkunen H, Räsänen JA, Kataja J (1974). Severe trauma as an etiologic factor in rheumatoid arthritis. Scand J Rheumatol, Vol 3 pp 97-102.
46574	Kageyama A, Torikoe K, Iwamoto M, Masuyama J-I, et al (2004). Nocardia arthritidis sp.nov., a new pathogen isolated from a patient with rheumatoid arthritis in Japan. J Clin Microbiol, 2366-71.
27382	Kaipiainen-Seppanen O, Aho K & Nikkarinen M (2001). Regional differences in the incidence of rheumatoid arthritis in Finland in 1995. Ann Rheum Dis Vol 60:128-132.
46516	Kaipiainen-Seppanen O, Kautiainen H (2006). Declining trend in the incidence of rheumatoid factor-positive rheumatoid arthritis in Finland 1980-2000. J Rheumatol, 33:2132-8.
29967	Kalla AA & Tikly M (2003). Rheumatoid arthritis in the developing world. Best Practice & Research Clinical Rheumatology, 17:863-875.
46515	Kallberg H, Padyukov L, Plenge RM, Ronnelid J, et al (2007). Gene-gene and gene-environment interactions involving HLA-DRB1, PTPN22, and smoking in two subsets of rheumatoid arthritis. Am J Hum Genet, 80:867-75.
32940	Kaltwasser JP, Kessler U, Gottschalk R, et al (2001). Effect of recombinant human erythropoietin and intravenous iron on anemia and disease activity in rheumatoid arthritis. J Rheumatol 28:2430-6.
27354	Karlson EW, Lee I-M, Cook NR, Manson JE, Buring JE & Hennekens CH (1999). A retrospective cohort study of cigarette smoking and risk of rheumatoid arthritis in female health professionals. Arthritis & Rheumatism Vol42:910-7.
29969	Karlson EW, Mandl LA, Aweh GN, & Grodstein F (2003). Coffee consumption and risk of rheumatoid arthritis. Arthritis & Rheumatism, 48:3055-3060.

46589	Karlson EW, Mandl LA, Aweh GN, Grodstein F (2003). Coffee consumption and risk of rheumatoid arthritis. Arthritis & Rheumatism, 48(11):3055-60.
46593	Karlson EW, Mandl LA, Hankinson SE, Grodstein F (2004). Do breast-feeding and other reproductive factors influence future risk of rheumatoid arthritis? Arthritis & Rheumatism, 50(11):3458-67.
46592	Karouzakis E, Neidhart M, Gay RE, Gay S (2006). Molecular and cellular basis of rheumatoid joint destruction. Immunology Letters, 106:8-13.
32209	Kashiwazaki S, Mizushima Y, Nobunaga M, Kondo H & Hirashima K (1996). Recombinant human erythropoietin therapy for anaemia in rheumatoid arthritis. Japanese Journal of Rheumatology, 6:333-344.
46594	Kaufmann J, Kielstein V, Kilian S, Stein G, Hein G (2003). Relation between body mass index and radiological progression in patients with rheumatoid arthritis. J Rheumatol, 30:2350-5.
27344	Kavanaugh A (1998). [Letters, Matters arising] Lipid profiles in patients with rheumatoid arthritis. Ann Rheum Dis Vol57:175.
29962	Kawaguchi Y, Matsuno H, Kanamori M, Ishihara H, Ohmori K, & Kimura T (2003). Radiological findings of the lumbar spine in patients with rheumatoid arthritis, and a review of pathologic mechanisms. Journal of Spinal Disorders and Techniques, 16:38-43.
27409	Kempsell KE, Cox CJ, McColm AA, Bagshaw JA, Reece R, Veale DJ, Emery P, Isaacs JD, Gaston JSH & Crowe JS (2001). Detection of Mycobacterium tuberculosis Group Organisms in human and mouse joint tissue by reverse transcriptase PCR: Prevalence in Diseased synovial tissue suggests lack of specific association with rheumatoid arthritis. Infection and Immunity Vol 69:1821-1831.
31177	Kerr JR, Cartron JP, Curran MD, Moore JE, et al (1995). A study of the role of parvovirus B19 in rheumatoid arthritis. British Journal of Rheumatology, 34:809-813.
46721	Khazeni N, Homer RJ, Rubinowitz AN, Chupp GL (2006). Massive cavitary pulmonary rheumatoid nodules in a patients with HIV. Eur Respir J, 28:872-4.
30189	Khuder SA, Peshimam AZ & Agraharam S (2002). Environmental risk factors for rheumatoid arthritis. Reviews on Environmental Health, 17:307-315.
28264	Kim W, Min S, Cho M, Youn J, et al (2000). The role of IL-12 in inflammatory activity of patients with rheumatoid arthritis (RA). Clinical & Experimental Immunology. 119:175-81.
38757	Kimbrough RD, Doemland ML, Mandel JS (2003). A mortality update of male and female capacitor workers exposed to polychlorinated biphenyls. JOEM, 45(3) pp 271-282.
47943	Kimbrough RD, Krouskas CA (2003). Human exposure to polychlorinated biphenyls and health effects. Toxicol Rev 22(4): 217-33.
30660	Kingsley G (1997). Microbial DNA in the synovium - a role in aetiology or a mere bystander. Lancet, 349(9058):1038-9.
48043	Kitamura K, Kikuchi Y, Watanabe S, Waechter G, et al (2000). Health effects of chronic exposure to polychlorinated dibenzo-P-dioxins (PCDD), dibenzofurans (PCDF) and coplanar PCB (Co-PCB) of municipal waste incinerator workers. Journal of Epidemiology, 10(4): 262-70.

5674	Kjeldsen-Kragh J, Hvatum M, Haugen M, Førre Ø, Scott H (1995). Antibodies against dietary antigens in rheumatoid arthritis patients treated with fasting and a one-year vegetarian diet. Clinical and Experimental Rheumatology, Vol 13 pp 167-172.
45933	Kjoller K, Friis S, Mellemkjaer L, McLaughlin JK, et al (2001). Connective tissue disease and other thrumatic conditions following cosmetic breast implantation in Denmark. Arch Int Med, 161(7) pp 973-979.
47058	Kjoller K, Holmich LR, Fryzek JP, Jacobsen PH, et al (2004). Self-reported musculoskeletal symptoms among Danish women with cosmetic breast implants. Ann Plast Surg, 52:1-7.
46623	Klareskog L, Alfredsson L, Rantapaa-Dahlqvist S, Berglin E, et al (2004). What precedes development of rheumatoid arthritis? Ann Rheum Dis, 63(Suppl II):ii28-ii31.
30661	Klareskog L, Lorentzen J, Padyukov L & Alfredsson L (2002). Genes and environment in arthritis: can RA be prevented? Arthritis Res, 4 (Suppl 3):S31-S36.
47059	Klareskog L, Padyukov L, Alfredsson L (2007). Smoking as a trigger for inflammatory rheumatic diseases. Curr Opin Rheumatol, 19:49-54.
46572	Klareskog L, Padyukov L, Lorentzen J, Alfredsson L (2006). Mechanisms of disease: genetic susceptibility and environmental triggers in the development of rheumatoid arthritis. Nature Clinical Practice, 2(8):425-33.
46588	Klareskog L, Padyukov L, Ronnelid J, Alfredsson L (2006). Genes, environment and immunity in the development of rheumatoid arthritis. Curr Opin Immunol, 18:650-5.
46587	Klareskog L, Stolt P, Lundberg K, Kallberg H, et al (2006). A new model for an etiology of rheumatoid arthritis. Arthritis & Rheumatism, 54(1):38-46.
5675	Klockars M, Koskela R-S, Järvinen E, Kolari PJ, Rossi A (1987). Silica exposure and rheumatoid arthritis: a follow up study of granite workers 1940-81. BMJ, Vol 294 pp 997-1000.
27366	Knekt P, Heliovaara M, Aho K, Alfthan G, Marniemi J & Aromaa A (2000). Serum selenium, serum alpha-tocopherol, and the risk of rheumatoid arthritis. Epidemiology Vol11:402-5.
44075	Knights KM, Tsoutsikos P, Miners JO (2005). Novel mechanisms of nonsteroidal anti-inflammatory drug-induced renal toxicity. Expert Opin Drug Metab Toxicol, 1(3) pp 399-408.
41701	Knights SE, Leandro MJ, Khamashta MA, Hughes GRV (1998). Minocycline-induced arthritis. Clin Exp Rheumatol, Vol 16 pp 587-590.
5676	Koeger AC, Lang T, Alcaix D, Milleron B, et al (1995). Silica-associated connective tissue disease: a study of 24 cases. Medicine-Baltimore, 74(5) pp 221-237.
27596	Koeger A-C, Rozenberg S, Bourgeois P (1996)[LETTER]. Scuba diving: a new risk for lung fibrosis and connective tissue disease? Journal of Rheumatology, 23(11) p2010.
5677	Koepsell TD, Dugowson CE, Nelson JL, Voigt LF, Daling JR (1994). Non-contraceptive hormones and the risk of rheumatoid arthritis in menopausal women. Intern J Epidemiology, 23(6) pp 1248-1255.
27653	Koh ET, Yap A U-J, Koh CKH, Chee TSG, Chan SP, & Boudville IC (1999). Temporomandibular disorders in rheumatoid arthritis. Journal of Rheumatology 26:1918-22.

27666	Kollias G, Douni E, Kassiotis G, Kontoyiannis D (1999). On the role of tumor necrosis factor and receptors in models of multiorgan failure, rheumatoid arthritis, multiple sclerosis and inflammatory bowel disease. Immunological Reviews, Vol 169 pp 175-94.
30300	Kremer JM, Alarcon GS, Weinblatt ME., et al (1997). Clinical, laboratory, radiographic, and histopathologic features of methotrexate-associated lung injury in patients with rheumatoid arthritis: a multicenter study with literature review. Arthritis & Rheumatism, 40:1829-37.
46591	Krishnan E (2003). Smoking, gender and rheumatoid arthritis - epidemiological clues to etiology. Results from the behavioural risk factor surveillance system. Joint Bone Spine, 70:496-502.
32233	Krishnan E (2003). Smoking, gender and rheumatoid arthritis - epidemiological clues to etiology: Results from the behavioral risk factor surveillance system. Joint Bone Spine, 70:496-502.
29958	Krishnan E, Sokka T & Hannonen P (2003). Smoking-gender interaction and risk for rheumatoid arthritis. Arthritis Research & Therapy. 5(3):R158-R162.
27675	Kroot E-J JA, van Gestel AM, Swinkels HL, Albers MMC, van de Putte LBA & van Riel PLCM (2001). Chronic comorbidity in patients with early rheumatoid arthritis: a descriptive study. J Rheumatol 28:1511-7.
46598	Kuhn KA, Kulik L, Tomooka B, Braschler KJ, et al (2006). Antibodies against citrullinated proteins enhance tissue injury in experimental autoimmune arthritis. J Clin Invest, 116(4):961-73.
27674	Kuiper S, va Gestel AM, Swinkels HL, de Boo TM, da Silva JAP, & van Riel PLCM (2001). Influence of sex, age, and menopausal state on the course of early rheumatoid arthritis. J Rheumatol 28:1809-16.
49013	Kuratsune M, Yoshimura T, Matsuzaka J, Yamaguchi A (1971). Yusho, a poisoning caused by rice oil contaminated with polychlorinated biphenyls. HSMHA Health Reports, 86(12): 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. Chemosphere, 67: 1194-201.
46726	Kvien TK, Uhlig T, Odegard S, Heiberg MS (2006). Epidemiological aspects of rheumatoid arthritis. The sex ratio. Ann NY Acad Sci, 1069:212-22.
47836	Kwon O, Lee E, Moon TC, Jung H, et al (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, 25(9): 1165-8.
30431	La Civita L, Fadda P, Gaudino C, Bentivenga C & Olivieri I (2003). Can hepatitis C virus infection and interferon-a undo the HLA-DRB1*0302 protection against rheumatoid arthritis? Letter to the Editor. Rheumatology, 42:383-4.
49011	La Rocca C, Mantovani A (2006). From environment to food: the case of PCB. Ann 1st Super Sanita, 42(4): 410-6.
46457	Lange JH (2004). [Comments] Endotoxin as a factor for joint pain and rheumatoid arthritis. Clin Rheumatol, 23: 566-7.
30555	Lathrop SL, Ball R, Haber P, Mootrey GT, et al (2002). Adverse event reports following vaccination for Lyme disease: December 1998 - July 2000. Vaccine, 20:1603-1608.

27350	Lauerma H, Lehtinen V, Jourkamaa M, Jarvelin M-R, Helenius H, & Isohanni M (1998). Schizophrenia among patients treated for rheumatoid arthritis and appendicitis. Schizophrenia Research Vol 29:255-261.
46247	Lee D-H, Steffes M, Jacobs DR Jr (2007). Positive associations of serum concentration of polychlorinated biphenyls or organochlorine pesticides with self-reported arthritis, especially rheumatoid type, in women. Environmental Health Perspectives, 115(6) pp 883-888.
46597	Lee H-S, Irigoyen P, Kern M, Lee A, et al (2007). Interaction between smoking, the shared epitope, and anti-cyclic citrullinated peptide. A mixed picture in three large North American rheumatoid arthritis cohorts. Arthritis & Rheumatism, 56(6):1745-53.
46596	Lee W, Weisman MH (2006). The predictive power of anti-cyclic citrullinated peptide antibodies: window into understanding gene/environment/immunity interactions. J Rheumatol, 33(7):1216-8.
30743	Levesque MC, Ward FE, Jeffery DR, and Weinberg JB (1999). Interferon-beta1A-induced polyarthritis in a polyarthritis in a patient with the HLA-DRB1*0404 allele. Arthritis Rheum, 42:569-73 [ABSTRACT].
46617	Li J, Schiottz-Christensen B, Olsen J (2005). Psychological stress and rheumatoid arthritis in parents after death of a child: a national follow-up study. Scand J Rheumatol, 34:448-50.
46259	Li W, Wang M, Irigoyen P, Gergersen PK (2006). Inferring causal relationships among intermediate phenotypes and biomarkers: a case study of rheumatoid arthritis. Bioinformatics, 22(12):1503-7.
27871	Liang MH, Karlson EW (1996). Female hormone therapy and the risk of developing or exacerbating systemic lupus erythematosus or rheumatoid arthritis. Proceedings of the Association of American Physicians, 108(1) pp 25-28.
48732	Ling S, Pi X, Holoshitz J (2007). The rheumatoid arthritis shared epitope triggers innate immune signaling via cell surface calreticulin. The Journal of Immunology, 179: 6359-67.
46622	Linn-Rasker SP, van der Helm-van Mil AHM, van Gaalen FA, et al (2006). Smoking is a risk factor for anti-CCP antibodies only in rheumatoid arthritis patients who carry HLA-DRB1 shared epitope alleles. Ann Rheum Dis, 65:366-71.
5678	Linos A, Kaklamanis E, Kontomerkos A, Koumantaki Y, et al (1991). The effect of olive oil and fish consumption on rheumatoid arthritis - a case control study. Scand J Rheumatol, Vol 20 pp 419-426.
5679	Linos A, Worthington JW, Palumbo PJ, O'Fallon WM, Kurland LT (1980). Occurrence of Hasimoto's thyroiditis and diabetes mellitus in patients with rheumatoid arthritis. J Chron Dis, Vol 33 pp 73-77.
5680	Lipsky PE (1994). Rheumatoid arthritis. Harrison's Principles of Internal Medicine13th Ed. Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, & Kasper DL (Editors) McGraw-Hill New York. Chapter 285 pp 1648-1655.
27515	Lipsky PE (2001). Rheumatoid Arthritis. In Harrison's Principles of Internal Medicine, 15th Edition, McGraw-Hill, New York, Vol 2 pp 1928-1937.
46255	Lipsky PE (2007). Why does rheumatoid arthritis involve the joints? NEJM, 356(23):2419-20.
41882	Lipworth L, Tarone RE, McLaughlin JK (2004). Silicone breast implants and connective tissue disease. An updated review of the epidemiologic evidence. Annals of Plastic Surgery, 52(6) pp 598-601.

46599	Lockshin MD (2006). Sex differences in autoimmune disease. Orthop Clin N Am, 37:629-33.
46607	Lorenz W, Sigrist G, Shakibaei M, Mobasheri A, Trautmann C (2006). A hypothesis for the origin and pathogenesis of rheumatoid arthritis. Rheumatol Int, 26:641-54.
46606	Lormeau C, Falgarone G, Roulot D, Boissier M-C (2006). Rheumatologic manifestations of chronic hepatitis C infection. Joint Bone Spine, 73:633-8.
46700	Low JM, Moore TL (2005). A role for the complement system in rheumatoid arthritis. Current Pharmaceutical Design, 11:655-70.
5681	Lundberg I, Alfredsson L, Plato N, Sverdrup B, et al (1994). Occupation, occupational exposure to chemicals and rheumatological disease. Scand J Rheumatol, Vol 23 pp 305-310.
46571	Lundy SK, Sarkar S, Tesmer LA, Fox DA (2007). Cells of the synovium in rheumatoid arthritis. T lumphocytes. Arth Res Ther, 9:202.
46605	MacGregor AJ, Steer SE (2006). Translating genetic information into clinical disease risk in rheumatoid arthritis. J Rheumatol, 33(12):2376-8.
46256	Machold KP, Stamm TA, Nell VPK, Pflugbeil S, et al (2007). Very recent onset rheumatoid arthritis: clinical and serological patient characteristics associated with radiographic progression over the first years of disease. Rheumatology, 46:342-9.
30490	Maillefert JF, Sibilia J, Toussirot E, Vignon E, Eschard JP, et al (1999). Rheumatic disorders developed after hepatitis B vaccination. Rheumatology, 38:978-83.
30568	Maillefert JF, Tonolli-Serabian, Cherasse, Demoux, Tavernier, & Piroth (2000). Arthritis following combined vaccine against diphtheria, poliomyelitis, and tetanus toxoid. Clinica and Experimental Rheumatology, 18:255-6.
46257	Majka DS, Holers VM (2006). Cigarette smoking and the risk of systemic lupus erythematosus and rheumatoid arthritis. Ann Rheum Dis, 65(5) pp 561-563.
46703	Makinen H, Hannonen P, Sokka T (2006). Definitions of remission for rheumatoid arthritis and review of selected clinical cohorts and randomised clinical trials for the rate of remission. Clin Exp Rheumatol, 24(Suppl 43):S22-8.
47833	Mallin K, McCann K, D'Aloisio A, Freels S, et al (2004). Cohort mortality study of capacitor manufacturing workers, 1944-2000. JOEM, 46(6): 565-76.
46258	Manfredsdottir VF, Vikingsdottir T, Jonsson T, Geirsson AJ, et al (2006). The effects of tobacco smoking and rheumatoid factor seropositivity on disease activity and joint damage in early rheumatoid arthritis. Rheumatology, 45:734-40.
29078	Manzi S & Wasko MCM. (2001). Getting to the heart of the matter in systemic lupus and rheumatoid arthritis. Bulletin on the Rheumatic Diseases Vol 50: 1-4.
46883	Marasini B,et al; Del Puente A, et al (2004). [Letters] Rheumatoid arthritis and thyroid disease; Reply. Clin Exp Rheumatol, 22(2): 265-6.
29959	Markovits D, Schapira D, Wiener A & Nahir AM. (2003). Silica-related rheumatoid arthritis wihout lung involvement. Clin Rheumatol. 22:53-55.
46731	Marks DJB, Mitchison NA, Segal AW, Sieper J (2006). Can unresolved infections precipitate autoimmune disease? CTMI, 305:105-25.

46604	Maruotti N, Crivellato E, Cantatore FP, Vacca A, Ribatti D (2007). Mast cells in rheumatoid arthritis. Clin Rheumatol, 26:1-4.
27374	Masdottir B, Jonsson T, Manfredsdottir V, Vikingsson A, Brekkan A & Valdimarsson H. (2000). Smoking, rheumatoid factor isotypes and severity of rheumatoid arthritis. Rheumatology Vol 39:1202-5.
27356	Masi AT & Aldag JC. (1999). [Letter] Significantly increased frequency of baseline IgA anti-type II collagen positivity in the placebo group in a study of oral type II collagen treatment of rheumatoid arthritis: comment on the article by Barnett, et al Arthritis & Rheumatism Vol 42:585-587.
27414	Masi AT, Aldag JC & Malamet RL. (2001). [CORRECTION] Heavy cigarette smoking and RA. Annals of the Rheumatic Diseases Vol61:189.
27380	Masi AT, Aldag JC & Malamet RL. (2001). Heavy cigarette smoking and RA. Annals of the Rheumatic Diseases Vol60:1154-5.
46879	Masi AT, Aldag JC, Chatterton RT (2006). Sex hormones and risks of rheumatoid arthritis and developmental or environmental influences. Ann N Y Acad Sci, 1069: 223-35.
27652	Masi AT, Chrousos GP, & Bornstein SR. (1999). [Editorial] Enigmas of adrenal androgen and glucocorticoid dissociation in premenopausal onset rheumatoid arthritis. Journal of Rheumatology 26:247-250.
27578	Masi AT, Fecht T, Aldag JC, Malamet RL, et al (1998)[LETTER]. Smoking and rheumatoid arthritis: comment on the letter by McDonagh and Walker. Arthritis & Rheumatism, 41(1) pp 184-5.
5682	Mason JH, Anderson JJ, Meenan RF, Weaver AL, Haralson KM (1990). Do life stressors influence disease activity in rheumatoid arthritis? Arthritis and Rheumatism, 33(S) pp 138.
5683	Massarotti EM, Liu NY, Mier J, Atkins MB (1992). Chronic inflammatory arthritis after treatment with high dose interleukin-2 for malignancy. Am J Med, Vol 92 pp 693-697.
48087	Mathieu S, Maillet B, Ristori JM, Soubrier M (2007). [Letter] Rheumatoid arthritis induced by labial injection of hyaluronic acid: Just a coincidence? Joint Bone Spine, 74: 512-3.
27575	Mattey DL, Dawes PT, Clarke S, Fisher J, et al (2002). Relationship among the HLA-DRB1 shared epitope, smoking, and rheumatoid factor production in rheumatoid arthritis. Arthritis & Rheumatism 47(4) pp 403-7.
27577	Mattey DL, Dawes PT, Fisher J, Brownfield A, et al (2002). Nodular disease in rheumatoid arthritis: association with cigarette smoking and HLA-DRB1/TNF gene interaction.. Journal of Rheumatology, 29(11) pp 2313-8.
27500	Mattey DL, Hutchinson D, Dawes PT, Nixon NB, Clarke S, Fisher J, Brownfield A, Alldersea J, Fryer AA & Strange RC. (2002). Smoking and disease severity in rheumatoid arthritis: association with polymorphism at the glutathione S-transferase M1 locus. Arthritis & Rheumatism, 46:640-6.
30584	McCallum RM, Patel DD, Moore JO & Haynes BF. (1997). Arthritis syndromes associated with Human T Cell Lymphotropic Virus Type 1 infection. Medical Clinics of North America 81(1):261-276.
27571	McDonagh JE, Walker DJ (1997)[LETTER]. Smoking and rheumatoid arthritis - observations from a multicase family study: comment on the article by Silman, et al Arthritis & Rheumatism, 40(3) p 594.
27598	McDonagh JE, Walker DJ (1997)[LETTER]. Smoking and rheumatoid arthritis - observations from a multicase family study: comment on the article by Silman, et al Arthritis & Rheumatism, 40(3) p 594.

46601	McGrath H Jr; Mikuls TR, Cerhan JR et al (2002). [Letters] Intolerance to caffeine may increase susceptibility to rheumatoid arthritis: comment on the article by Mukuls et al. Arthritis & Rheumatism, 46(11):3104-5.
30305	McHugh NJ, Whyte J, Harvey G, & Haustein UF. (1994). Anti-topoisemerase I antibodies in silica-associated systemic sclerosis: a model for autoimmunity. Arthritis & Rheumatism, 37:1198-1205.
46722	Medina F, Perez-Saleme L, Moreno J (2006). Rheumatoic manifestations of human immunodeficiency virus infection. Infect Dis Clin N Am, 20:891-912.
27617	Meier LG, Barthel HR & Seidl C. (1997). Development of polyarthritis after insertion of silicone breast implants followed by remission after implant removal in 2 HLA-identical sisters bearing rheumatoid arthritis susceptible genes. J Rheumatol 24:1838-41.
27361	Mercado F, Marshall RI, Klestov AC, & Bartold PM. (2000). Is there a relationship between rheumatoid arthritis and periodontal disease? J Clin Periodontol Vol 27:267-72.
29968	Mercado FB, Marshall RI, & Bartold PM. (2003). Inter-relationships between rheumatoid arthritis and perodontal disease: A review. Journal of Clinical Periodontology, 30:761-772.
30453	Merlino LA, Cerhan JR, Criswell LA, Mikuls TR & Saag KG. (2003). Estrogen and other female reproductive risk factors are not strongly associated with the development of rheumatoid arthritis in elderly women. Semin Arthritis Rheum, 33:72-82.
46603	Merlino La, Cerhan JR, Criswell LA, Mikuls TR, Saag KG (2003). Estrogen and other female reproductive risk factors are not strongly associated with the development of rheumatoid arthritis in elderly women. Semin Arthritis Rheum, 33:72-82.
30454	Merlino LA, Curtis J, Mikuls TR, Cerhan JR, Criswell LA & Saag KG. (2004). Vitamin D intake is inversely associated with rheumatoid arthritis: results from the Iowa Women's Health Study. Arthritis & Rheumatism, 50:72-77.
46602	Merlino LA, Curtis J, Mikuls TR, Cerhan JR, Criswell LA, Saag KG (2004). Vitamin D intake is inversely associated with rheumatoid arthritis. Arthritis & Rheumatism, 50(1):72-7.
30328	Miall WE, Caplan A, Cochrane AL, et al (1953). An epidemiological study of rheumatoid arthritis associated with characteristic chest x-ray appearances in coal-workers. British Medical Journal, Vol II:1231-6.
46263	Michalsen A, Riegert M, Ludtke R, Backer M, et al (2005). Mediterranean diet or extended fasting's influence on changing the intestinal microflora, immunoglobulin A secretion and clinical outcome in patients with rheumatoid arthritis and fibromyalgia: an observational study. BMC Complementary and Alternative Medicine, 5:22.
46582	Michou L, Croiseau P, Petit-Teixeira E, et al (2006). Validation of the reshaped shared epitope HLA-DRB1 classification in rheumatoid arthritis. Arthritis Research & Therapy, 8:R79.
46264	Michou L, Lasbleiz S, Rat A-C, Migliorini P, et al (2007). Linkage proof for PTPN22, a rheumatoid arthritis susceptibility gene and a human autoimmunity gene. PNAS, 104(5):1649-54.
5685	Micromedex Inc. (1995) Micromedex International Healthcare Series (CD-ROM - Monograph; Vol 82) DRUGDEX(R) - Drug Evaluation Monographs. Accessed 1996 June 13.

27491	Mikuls TR, Cerhan JR, Criswell LA, et al (2002). Coffee, tea, and caffeine consumption and risk of rheumatoid arthritis: Results from the Iowa Women's Health Study. Arthritis & Rheumatism, 46:83-91.
46614	Mikuls TR, Cerhan JR, Criswell LA, Merlino L, et al (2002). Coffee, tea, and caffeine consumption and risk of rheumatoid arthritis. Results from the Iowa women's health study. Arthritis & Rheumatism, 46(1):83-91.
27677	Mikuls TR, Saag KG (2001). Comorbidity in rheumatoid arthritis. Rheumatic Disease Clinics of North America, Vol 27 pp 283-303.
29966	Mikuls TR. (2003). Comorbidity in rheumatoid arthritis. Best Practice & Research Clinical Rheumatology, 17:729-752.
46892	Milanino R, Buchner V (2006). Copper: role of the 'Endogenous' and 'Exogenous' Metal on the Development and Control of Inflammatory Processes. Reviews on Environmental Health, 21(3): 153-215.
27669	Mili F, Helmick CG, & Zack MM. (2002). Prevalence of arthritis: analysis of data from the US Behavioural Risk Factor Surveillance System, 1996-99. J Rheumatol 29:1981-8.
46581	Miller FW (2006). Is occupational exposure to mineral oil a risk factor for rheumatoid arthritis? Nature Clinical Practice, 2(3):130-1.
46960	Mincione GP, Taddei GL, Wolovsky M, Calzolari A, Mincione F (1992). Detection of human papillomavirus (HPV) DNA Type 6/11 in a conjunctival papilloma by in situ hybridization with biotinylated probes. Pathologica, 84: 483-88.
48736	Mobley JL (2004). Is rheumatoid arthritis a consequence of natural selection for enhanced tuberculosis resistance? Medical Hypothesis, 62: 839-43.
46612	Molberg O, Sollid LM (2006). A gut feeling for joint inflammation - using coeliac disease to understand rheumatoid arthritis. Trends in Immunology, 27(4):188-94. Erratum: Trends in Immunology, 27(6):254.
36872	Molina V, Shoenfeld Y (2005). Infection, vaccines and other environmental triggers of autoimmunity. Autoimmunity, 38(3): 235-245.
46610	Moroldo MB, Chaudhari M, Shear E, Thompson SD, et al (2004). Juvenile rheumatoid arthritis affected sibpairs. Arthritis & Rheumatism, 50(6):1928-34.
27516	Mosquera-Martinez JA. (2001). Rheumatoid arthritis associated with ulcerative colitis. Annals of the Rheumatic Diseases. 60:1155.
47963	Motivala SJ, Khanna D, FitzGerald J, Irwin MR (2008). Stress activation of cellular markers of inflammation in rheumatoid arthritis. Arthritis & Rheumatism, 58(2): 376-83.
48737	Mouly S, Berenbaum F, Kaplan G (2001). Remitting seronegative symmetrical synovitis with pitting edema following intravesical bacillus calmette-guerin instillation. J Rheumatol, 28: 1699-701.
31977	Murphy EA, Bell AL, Wojtulerski J, et al (1994). Study of erythropoietin in treatment of anaemia in patients with rheumatoid arthritis. BMJ, 309:1337-8.
30428	Nadir F, Fagiuioli S, Wright HI, Nadir A, Hopp E, Gavaler J & Van Thiel DH. (1994). Rheumatoid arthritis: a complication of interferon therapy. J Okla State Med Assoc, 87:228-230.
27872	Nagao Y, Sata M, Noguchi S, Tajiri N, et al (1999). Various extrahepatic manifestations caused by hepatitis C virus infection. International Journal of Molecular Medicine, 4(6) pp 621-5.

46609	Naithani R, Mahapatra M, Kumar R, Kumar A, Agrawal N (2007). Arsenic trioxide induced acute flare-up of rheumatoid arthritis in a patient with APL. Ann Hematol, 86:151-2.
47394	Nandakumar KS, Holmdahl R (2007). Collagen antibody induced arthritis. Methods Mol Med, 136: 215-23.
28388	National Academy of Sciences (2003). Gulf War and Health. Volume 2. Insecticides and Solvents. National Academy Press, Washington, DC. pp 111-112, 144-147, 327-328, 335-338, 347-351.
30227	National Academy of Sciences (2003). Gulf War and Health. Volume 2. Insecticides and Solvents. National Academy Press, Washington, DC. pp 518-520.
47391	Naz SM, Symmons DPM (2007). Mortality in established rheumatoid arthritis. Best Pract Res Clin Rheum, 21(5): 871-83.
5686	Nelson JL, Koepsell TD, Dugowson CE, Voigt LF, et al (1993). Fecundity before disease onset in women with rheumatoid arthritis. Arthritis and Rheumatism, 36(1) pp 7-14.
29077	Nesher G & Mates M. (2000). Palindromic rheumatism: effect of dietary manipulation. Clinical and Experimental Rheumatology Vol 18 pp 375-378.
47407	Nesher G (1999). [Comment] Bacillus calmette-guerin induced reactive arthritis. J Rheumatol, 26(7): 1638.
48738	Neuberger GB, Aaronson LS, Gajewski B, Embretson SE, et al (2007). Predictors of exercise and effects of exercise on symptoms, function, aerobic fitness, and disease outcomes of rheumatoid arthritis. Arthritis & Rheumatism (Arthritis Care & Research), 57(
46733	Newkirk MM (2006). Antibacterial antibodies and arthritis. Is there a link? JCR, 12(1):1-2.
47831	Nichols BR, Hentz KL, Aylward L, Hays SM, Lamb JC (2007). Age-specific reference ranges for polychlorinated biphenyls (PCB) based on the NHANES 2001-2002 survey. J Toxicol Environ Health Part A, 70: 1873-7.
46608	Nielen MMJ, van Schaardenburg D, Lems WF, van de Stadt RJ, et al (2006). [Letter] Vitamin D deficiency does not increase the risk of rheumatoid arthritis: comment on the article by Merlino et al. Arthritis & Rheumatism, 54(11):3719-20.
46615	Niewold TB, Harrison MJ, Paget SA (2007). Anti-CCP antibody testing as a diagnostic and prognostic tool in rheumatoid arthritis. Q J Med, 100:193-201.
46962	Nigrovic PA, Lee DM (2005). [Discussion] Mast cells in autoantibody responses and arthritis. Novartis Found Symp, 271: 210-214.
46961	Nigrovic PA, Lee DM (2005). Mast cells in autoantibody responses and arthritis. Novartis Found Symp, 271: 200-209.
46569	Nigrovic PA, Lee DM (2007). Synovial mast cells: role in acute and chronic arthritis. Immunological Reviews, 217:19-37.
31183	Nikkara S, Roivainen A, Hannonen P, Mottonen T, Luukkainen R, Yli-Jama T & Toivanen P. (1995). Persistence of parvovirus B19 in synovial fluid and bone marrow. Annals of the Rheumatic Diseases, 54:597-600.
48789	Noonan CW, Pfau JC, Larson TC, Spence MR (2006). Nested case-control study of autoimmune disease in an asbestos-exposed population. Environ Health Perspect, 114(8): 1243-47.

46723	Nowak UM, Newkirk MM (2005). Rheumatoid factors: good or bad for you? Int Arch Allergy Immunol, 138:180-8.
46265	Nyhall-Wahlin B-M, Jacobsson LTH, Petersson IF, Turesson C (2006). Smoking is a strong risk factor for rheumatoid nodules in early rheumatoid arthritis. Ann Rheum Dis, 65(5) pp 601-606.
27332	Odeh M. (1997). New insights into the pathogenesis and treatment of rheumatoid arthritis. Clinical Immunology and Immunopathology Vol 83:103-116.
48955	Okumura M (1984). Past and current medical states of Yusho patients. Am J Ind Med, 5: 13-8.
46704	Olech E, Merrill JT (2006). The prevalence and clinical significance of antiphospholipid antibodies in rheumatoid arthritis. Current Rheumatology Reports, Vol 8 pp 100-108.
46618	Oliver JE, Silman AJ (2006). Risk factors for the development of rheumatoid arthritis. Scand J Rheumatol, 35:169-74.
46890	Oliver JE, Worthington J, Silman AJ (2006). Genetic epidemiology of rheumatic arthritis. Curr Opin Rheumatol, 18: 141-146.
30451	Olsson AR, Skogh T, Axelson O, Wingren G (2004). Occupations and exposures in the work environment as determinants for rheumatoid arthritis. Occup Environ Med, 61: 233-8.
27665	Olsson AR, Skogh T, Wingren G (2000). Occupational determinants for rheumatoid arthritis. Scandinavian Journal of Work, Environment and Health, 26(3) pp 243-9.
46692	Olsson AR, Skogh T, Wingren G (2004). Aetiological factors of importance for the development of rheumatoid arthritis. Scand J Rheumatol, 33:300-6.
46550	Orozco G, Rueda B, Martin J (2006). Genetic basis of rheumatoid arthritis. Biomedicine & Pharmacotherapy, 60:656-62.
30234	OSHA, NIOSH (?). Silicosis - OSHA & NIOSH recommended limits of silica dust exposure. Http://www.silicosis.com/niosh/
46889	Ostensen M, Villiger PM (2007). The remission of rheumatoid arthritis during pregnancy. Semin Immunopathol, 29(2) pp 185-191.
46583	Otsuki T, Maeda M, Murakami S, Hayashi H, et al (2007). Immunological effects of silica and asbestos. Cellular & Molecular Immunology, 4(4):261-8.
46549	Padyukov L, Silva C, Stolt P, Alfredsson L, et al (2004). A gene-environment interaction between smoking and shared epitope genes in HLA-DR provides a high risk of seropositive rheumatoid arthritis. Arthritis & Rheumatism, 50(10):3085-92.
46548	Panayi GS (2005). B cells: a fundamental role in the pathogenesis of rheumatoid arthritis? Rheumatology, 44(Suppl 2):ii3-ii7.
30457	Papadopoulos IA, Katsimbri P, Alamanos Y, Voulgari PV & Drosos AA. (2003). Early rheumatoid arthrtis patients: relationship of age. Rheumatol Int, 23:70-4.
47272	Papadopoulos NG, Alamanos Y, Voulgari PV, et al (2005). Does cigarette smoking influence disease expression, activity and severity in early rheumatoid arthritis patients? Clin Exp Rheumatol, 23: 861-6.
27867	Parks CG, Conrad K, Cooper GS (1999). Occupational exposure to crystalline silica and autoimmune disease. Environmental Health Perspectives, 107(S 5) pp 793-802.

46547	Patberg WR, Rasker JJ (2004). Weather effects in rheumatoid arthritis: from controversy to consensus: a review. J Rheumatol, 31:1327-34.
46546	Pattison DJ, Harrison RA, Symmons DPM (2004). The role of diet in susceptibility to rheumatoid arthritis: a systematic review. J Rheumatol, 31:1310-9.
48790	Pattison DJ, Silman AJ, Goodson NJ, et al (2004). Vitamin C and the risk of developing inflammatory polyarthritis: prospective nested case-control study. Ann Rheum Dis, 63(7):843-47.
46545	Pattison DJ, Symmons DPM, Lunt M, Welch A, et al (2004). Dietary risk factors for the development of inflammatory polyarthritis. Arthritis & Rheumatism, 50(12):3804-12.
46543	Pedersen M, Jacobsen S, Garred P, Madsen HO, et al (2007). Strong combined gene-environment effects in anti-cyclic citrullinated peptide-positive rheumatoid arthritis. Arthritis & Rheumatism, 56(5):1446-53.
46542	Pedersen M, Jacobsen S, Klarlund M, Frisch M (2006). Socioeconomic status and risk of rheumatoid arthritis: a Danish case-control study. J Rheumatol, 33:1069-74.
46281	Pedersen M, Jacobsen S, Klarlund M, Pedersen BV, et al (2006). Environmental risk factors differ between rheumatoid arthritis with and without auto-antibodies against cyclic citrullinated peptides. Arthritis Research & Therapy, 8:R133.
46882	Pedersen M, Stripp C, Klarlund M, Olsen SF, et al (2005). Diet and risk of rheumatoid arthritis in a prospective cohort. J Rheumatol, 32(7): 1249-52.
32942	Peeters HRM, Jongegn-Lavrencic M, Vreugdenhil G & Swaak AJG. (1996). Effect of recombinant human erythropoietin on anaemia and disease activity in patients with rheuamtoid arthritis and anaemia of chronic disease: a randomised placebo controlled bouble bl
31976	Peeters HRM, Jongen-Lavrencic M, Bakker CH, et al (1999). Recombinant human erythropoietin improves health-related quality of life in patients with rheumatoid arthritis and anaemia of chronic disease; utility measures correlate strongly with disease activity measures. Rheumatol Int, 18:201-6.
27351	Peeva E, Fishman AD, Goddard G, Wadler S and Barland P. (2000). Rheumatoid arthritis exacerbation caused by exogenous interleukin-12. Arthritis & Rheumatism Vol34:461-3.
30531	Penglis PS, Bond C, Humphreys I, McCluskey J, Cleland LG. (2000). Genetic susceptibility and the link between cat exposure and rheumatoid arthritis. Seminars in Arthritis and Rheumatism, 30 pp 111-120.
46566	Persijn JP; Straub RH, Bijlsma JWJ, Cutolo M (2006). [Letters] How psychological stress via hormones and nerve fibers may exacerbate rheumatoid arthritis: comment on the review by Straub et al. Arthritis & Rheumatism, 54(2):682-4.
5687	Pinals RS (1989). Traumatic arthritis and related conditions Arthritis and Allied Conditions. A Textbook of Rheumatology Eleventh Edition. McCarty DJ (Ed). Lea and Febiger Philadelphia. Chapter 87 p 1375.
32941	Pincus T, Olsen NJ, Russell IJ, et al (1990). Multicenter study of recombinant human erythropoietin in correction of anemia in rheumatoid arthritis. American Journal of Medicine 89:161-8.
30327	Pittau E, Bogliolo A, Tinti A, Mela Q, Ibba G, Salis G & Perpignano G. (1997). Development of arthritis and hypothyroidism during alpha-interferon therapy for chronic hepatitis C. Clinical & Experimental Rheumatology, 15:415-9.

27336	Pladevall-Vila M, Delclos GL, Varas C, Guyer H, Brugues-Tarradellas J, and Anglada-Arisa A. (1996). Controversy of oral contraception and risk of rheumatoid arthritis: meta-analysis of conflicting studies and review of conflicting meta-analyses with speci
48791	Plenge RM, Seielstad M, Padyukov L , et al (2007). TRAF1-C5 as a risk locus for rheumatoid arthritis - a genomewide study. N Engl J Med, 357(12): 1199-1209.
46619	Podas T, Nightingale JMD, Oldham R, Roy S, et al (2007). Is rheumatoid arthritis a disease that starts in the intestine? A pilot study comparing an elemental diet with oral prednisolone. Postgrad Med J, 83:128-31.
30303	Pope JE, Joneja M, Hong P (2002). Anti-androgen treatment of prostatic carcinoma may be a risk factor for development of rheumatoid arthritis. J Rheumatol, 29 pp 2459-62.
30301	Pope JE, Stevens A, Howson W, Bell DA (1998). The development of rheumatoid arthritis after recombinant Hepatitis B vaccination. J Rheumatol, 25 pp 1687-93.
46541	Prahalad S, Fraser AM, O'Brien E, Kerber RA, et al (2003). [Comment] Lack of association between birth order and juvenile idiopathic arthritis. Arthritis & Rheumatism, 48(10): 2989-90.
41895	Prince MM, Hein MJ, Ruder AM, Waters MA, et al (2006). Update: cohort mortality study of workers highly exposed to polychlorinated biphenyls (PCB's) during the manufacture of electrical capacitors, 1940-1998. Environmental Health, 5(13).
44866	Prince MM, Ruder AM, Hein MJ, Waters MA, Whelan EA, et al (2006). Mortality and exposure response among 14,458 electrical capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). Environ Health Perspect, 114(10) pp 1508-1514.
5688	Pritchard MH (1992). An examination of the role of female hormones and pregnancy as risk factors for rheumatoid arthritis, using a male population as control group. Br J Rheumatol, Vol 31 pp 395-399.
27490	Provenzano G, Donato G, Brai G, Rinaldi F (2002). [Comment] Prevalence of allergic respiratory diseases in patients with RA. Annals of the Rheumatic Diseases, 61: 281.
42092	Pugliese A, Beltramo T, Torre D, Roccatello D (2006). Parvovirus B19 and immune disorders. Cell Biochem Funct [Epub ahead of print].
45979	Quiceno GA, Cush JJ (2007). Iatrogenic rheumatic syndromes in the elderly. Rheum Dis Clin N Am, 33: 123-34.
27411	Radstake TRDJ, Barrera P, Albers JMC, Swinkles HL, van de Putte LBA & van Riel PLCM. (2000). Familial vs sporadic rheumatoid arthritis (RA). A prospective study in an early RA inception cohort. Rheumatology Vol 39:267-273.
46266	Ramirez AS, Rosas A, Hernandez-Beriain JA, Orengo JC, et al (2005). Relationship between rheumatoid arthritis and mycoplasma pneumoniae: a case-control study. Rheumatology, 44:912-4.
46540	Rashid T, Ebringer A (2007). Rheumatoid arthritis is linked to proteus - the evidence. Clin Rheumatol, 26:1036-43.
46884	Rashid T, Jayakumar KS, Binder A, Ellis S, et al (2007). Rheumatoid arthritis patients have elevated antibodies to cross-reactive and non cross-reactive antigens from Proteus microbes. Clin Exp Rheumatol, 25: 259-67.

27870	Rashid T, Tiwana H, Wilson C, Ebringer A (2001). Rheumatoid arthritis as an autoimmune disease caused by Proteus urinary tract infections: a proposal for a therapeutic protocol. Israel Medical Association Journal, Vol 3 pp 675-80.
46539	Rass P, Pakozdi A, Lakatos P, Zilahi E, et al (2006). Vitamin D receptor gene polymorphism in rheumatoid arthritis and associated osteoporosis. Rheumatol Int, 26:964-71.
47396	Ravikumar R, Anolik J, Looney RJ (2007). Vaccine responses in patients with rheumatoid arthritis. Current Rheumatology Reports, 9: 407-15.
30535	Ray P, Black S, Shinefield H, Dillon A, et al (1997). Risk of chronic arthropathy among women after rubella vaccination. JAMA, 278:551-6.
27413	Reckner Olsson A, Skogh T & Wingren G. (2001). [CORRECTION] Comorbidity and lifestyle, reproductive factors, and environmental exposures associated with rheumatoid arthritis. Annals of the Rheumatic Diseases Vol 60: 1161.
27412	Reckner Olsson A, Skogh T & Wingren G. (2001). Comorbidity and lifestyle, reproductive factors, and environmental exposures associated with rheumatoid arthritis. Annals of the Rheumatic Diseases Vol 60: 934-939.
30669	Reid DM, Brown T, Reid TMS, Rennie JAN & Eastmond CJ. (1985). Human parvovirus associated-arthritis: a clinical and laboratory description. Lancet, February 23:422-425.
46267	Remmers EF, Plenge RM, Lee AT, Graham RR, et al (2007). STAT4 and the risk of rheumatoid arthritis and systemic lupus erythematosus. NEJM, 357(10):977-86.
46616	Reveille JD, Williams FM (2006). Rheumatologic complications of HIV infection. Best Practice & Research Clinical Rheumatology, 20(6):1159-79.
30304	Romieu I, Hernandez-Avila M, & Liang MH. (1989). Oral contraceptives and the risk of rheumatoid arthritis: a meta-analysis of a conflicting literature. British Journal of Rheumatology, 28(suppl 1):13-7.
5689	Rook GA, Lydyard PM, Stanford JL (1993). A reappraisal of the evidence that rheumatoid arthritis and several other idiopathic diseases are slow bacterial infections. Ann Rheum Dis, Vol 52 pp S30-S38.
46891	Rose CD (2006). Epidemiology of juvenile rheumatoid arthritis in the Americas. An update. JCR, 12(3): 129-30.
27546	Rosenman KD, Moore-Fuller M & Reilly MJ. (1999). Connective tissue disease and silicosis. American Journal of Industrial Medicine 35(4): 375-381.
46551	Rosenstein ED, Greenwald RA, Kushner LJ, Weissmann G (2004). Hypothesis: the humoral immune response to oral bacteria provides a stimulus for the development of rheumatoid arthritis. Inflammation, 28(6):311-8.
48086	Rosner I, Rozenbaum M, Toubi E, Kessel A, Naschitz JE, Zuckerman E (2004). The case for hepatitis C arthritis. Semin Arthritis Rheum, 33: 375-87.
47827	Ross G (2004). The public health implications of polychlorinated biphenyls (PCBs) in the environment. Ecotoxicology and Environmental Safety, 59: 275-91.
46559	Rothfield NF, Lim AA (2006). Systemic lupus erythematosis evolving into rheumatoid arthritis. J Rheumatol, 33(1):188-90.

46556	Roux CH, Saraux A, Le Bihan E, Fardellone P, et al (2007). Rheumatoid arthritis and spondyloarthropathies: geographical variations in prevalence in France. J Rheumatol, 34:117-22.
48792	Rudel RA, Seryak LM, Brody JG (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. Environ Health, 7:2
41902	Ruder AM, Hein MJ, Nilsen N, Waters MA, et al (2006). Mortality among workers exposed to polychlorinated biphenyls (PCB's) in an electrical capacitor manufacturing plant in Indiana: an update. Environmental Health Perspectives, 114(1): 18-23.
5690	Rudge SR, Baron FM, Drury P (1982). Lack of association between rheumatoid arthritis and Type 1 (insulin-dependent) diabetes. J Rheumatol, 9(2) pp 343-344.
32231	Ruediger HW (2000). Hard metal particles and lung disease: coincidence or causality? Respiration 67:137-8.
27338	Saag KG, Cerhan JR, Kolluri S, Ohashi K, Hunninghake GW & Schwartz DA. (1997). Cigareete smoking and rheumatoid arthritis severity. Annals of the Rheumatic Diseases Vol 56:463-9.
30270	Sakurai A & Hashizume K. (2000). Deterioration of rheumatoid arthritis with troglitazone: a rare and unexpected adverse effect. Archives of Internal Medicine, 160:118-9.
27806	Salvador G, Sanmarti R, Ros I, Halperin I (1999). Idiopathic hypoparathyroidism associated with adult rheumatoid arthritis. Clinical Rheumatology, Vol 18 pp 334-6.
47962	Sanchez-Pernaute O, Ospelt C, Neidhart M, Gay S (2008). Epigenetic clues to rheumatoid arthritis. J Autoimmun, 30(1-2): 12-20.
27407	Sangha O. (2000). Epidemiology of rheumatic diseases. Rheumatology Vol 39(suppl 2):3-12.
27869	Sany J, Dropsy R, Daures JP (1998). Cross-sectional epidemiological survey of rheumatoid arthritis patients seen in private practice in France. Descriptive results (1629 cases). Revue du Rhumatisme (English Edition). 65(7-9):462-70.
27358	Saraux A, Guedes C, Belghomari H, et al (1999). [Letter] Sex-associated factors and the presentation of rheumatoid arthritis: comment on the article by Weyand, et al Arthritis & Rheumatism Vol 42:588-9.
27643	Saraux A, Guedes C, Belghomari H, Youinou P, & Le Goff P. (1999). Sex-associated factors and the presentation of rheumatoid arthritis: comment on the article by Weyand, et al[comment]. [Comment. Letter] Arthritis & Rheumatism. 42(3):588-9; discussion 589-90.
30314	Saslaw S & Klainer AS. (1969). Rheumatoid syndrome during isoniazid therapy. American Review of Respiratory Disease, 100:221-223.
46558	Saurenmann RK, Rose JB, Tyrrell P, Feldman BM, et al (2007). Epidemiology of juvenile idiopathic arthritis in a multiethnic cohort. Arthritis & Rheumatism, 56(6):1974-84.
46881	Sawada S, Takei M (2005). Epstein-Barr virus etiology in rheumatoid synovitis. Autoimmunity Reviews, 4: 106-10.
46555	Sawade S, Takei M (2005). Epstein-Barr virus etiology in rheumatoid synovitis. Autoimmunity Reviews, 4:106-10.

27618	Schaeverbeke T, Bebear C, Lequen L, Dehais J, & Bebear C. (1998). [Letter] Mycoplasmas in the joints of patients with rheumatoid arthritis and other inflammatory rheumatic disorders: comment on the article by Hoffman, et al Arthritis & Rheumatism 41:754-7
46963	Schett G (2007). Cells of the synovium in rheumatoid arthritis. Osteoclasts. Arthritis Research & Therapy, 9: 203.
46965	Scrivo R, Di Franco M, Spadaro A, Valesini G (2007). The immunology of rheumatoid arthritis. Ann N Y Acad Sci, 1108: 312-22.
30309	Seaman JM, Goble M, Madsen L, Steigerwald JC (1985). Fasciitis and polyarthritis during antituberculous therapy. Arthritis & Rheumatism, 28: 1179-84.
32798	Segerstrom SC, Miller GE (2004). Psychological stress and the human immune system: a meta-analytic study of 30 years of inquiry. Psychological Bulletin, 130(4) pp 601-630.
27615	Selden SM & Cameron AS. (1996). Changing epidemiology of Ross River virus disease in South Australia. MJA 165:313-7.
47824	Selgrade MK (2007). Immunotoxicity - the risk is real. Toxicological Sci, 100(2): 328-32.
30308	Shabtai R, Shapiro MS, Orenstein D, Taragan R & Shenkman L. (1984). The antithyroid arthritis syndrome reviewed. Arthritis & Rheumatism, 27:227-9.
30554	Shadomy SV, Ball R, Woo J, Miller FW & Braun MM. (2001). Arthritis after Lyme vaccine: selected epidemiologic features based on follow-up evaluations of Vaccine Adverse Event Reporting System (VAERS) reports. [ABSTRACT] Arthritis Rheum 2001; 44 (suppl):S230.
30267	Shapiro S. (1982). Rheumatoid arthritis, the pill, and self-selection. Lancet, December 4: 1282.
48793	Shields PG (2006). Understanding population and individual risk assessment: the case of polychlorinated biphenyls. Cancer Epidemiol Biomarkers Prev, 15(5); 830-39.
27505	Sibilia J & Maillefert JF. (2002). Vaccination and rheumatoid arthritis: induction of rheumatoid arthritis by vaccination against hepatitis B: myth or reality? Annals of the Rheumatic Diseases, 61:575-6.
27346	Silman A, Bankhead C, Rowlinson B, Brennan P, Symmons D & Gatrell A. (1997). Do new cases of rheumatoid arthritis cluster in time or in space? International Journal of Epidemiology Vol26:628-634.
5691	Silman A, Kay A, Brennan P (1992). Timing of pregnancy in relation to the onset of rheumatoid arthritis. Arthritis and Rheumatism, 35(2) pp 152-155.
30664	Silman AJ & Pearson JE. (2002). Epidemiology and genetics of rheumatoid arthritis. Arthritis Res, 4(suppl 3):S265-272.
5692	Silman AJ (1989). Rheumatoid arthritis and infection: a population approach. Ann Rheum Dis, Vol 48 pp 707-710.
5693	Silman AJ (1991). Is rheumatoid arthritis an infectious disease? An epidemic would answer the question. BMJ, Vol 303 pp 200-201.
5694	Silman AJ (1992). Parity status and the development of rheumatoid arthritis. American Journal of Reproductive Immunology, Vol 28 pp 228-230.
5695	Silman AJ (1993). [Comment] Smoking and the risk of rheumatoid arthritis: editorial. J Rheumatol, 20(11) pp 1815-1816.

27498	Silman AJ (2001). [Comment] Do genes or environment influence development of rheumatoid arthritis? BMJ, 323: 4-5.
30671	Silman AJ (2002). Do genes or environment influence development of rheumatoid arthritis? BMJ, 324:266-267.
27570	Silman AJ, Newman J, MacGregor AJ (1996). Cigarette smoking increases the risk of rheumatoid arthritis. Results from a nationwide study of disease-discordant twins. Arthritis & Rheumatism, 39(5) pp 732-5.
27502	Silman AJ. (2002). The changing face of rheumatoid arthritis: Why the decline in incidence? Arthritis & Rheumatism, 46:579-581.
28339	Sim M, Abramson M, Forbes A, Ikin J, et al (2003). Australian Gulf War Veterans' Health Study (Vol 2), Commonwealth of Australia.
46553	Simard JF, Mittleman MA (2007). Prevalent rheumatoid arthritis and diabetes among NHANES III participants aged 60 and older. J Rheumatol, 34:469-73.
14100	Sinks T, Steele G, Smith AB, Watkins K and Shults RA (1992). Mortality among workers exposed to polychlorinated biphenyls. American Journal of Epidemiology, 136(4) pp 389-398.
46268	Sircar D, Ghosh B, Ghosh A, Haldar S (2006). Juvenile idiopathic arthritis. Indian Pediatrics, 43:429-33.
27614	Sluis-Cremer GK, Hessel PA, Hnizdo E & Churchill AR. (1986). Relationship between silicosis and rheumatoid arthritis. Thorax 41:596-601.
31180	Smith JB & Haynes MK. (2002). Rheumatoid arthritis - a molecular understanding. Ann Intern Med, 136:908-922.
27579	Smith MD (1998)[LETTER]. Adhesion molecules at the synovial level in rheumatoid arthritis: comment on the review article by Mojcik and Shevach. Arthritis & Rheumatism, 41(1) pp 185-6.
47408	Smith MD (2000). [Comment] Bacillus calmette-guerin induced reactive arthritis. J Rheumatol, 27(6): 1570.
30638	Soderlund M, von Essen R, Haapasaari J, et al (1997). Persistence of parvovirus B19 DNA in synovial membranes of young patients with and without chronic arthropathy. Lancet 349(9058):1063-1065.
46693	Sood A, Midha V, Sood N (2004). Rheumatoid arthritis probably induced by pegylated interferon in a patient with chronic hepatitis C. Ind J Gastroenterol, 23(1):28-9.
46557	Soy M, Guildiken S, Arikan E, Altun BU, Tugrul A (2007). Frequency of rheumatoid diseases in patients with autoimmune thyroid disease. Rheumatol Int, 27:575-7.
45976	Spears CJ, Batchelor JR (1987). Drug-induced autoimmune disease. Adv Nephrol, 16: 219-29.
30285	Spector TD & Silman AJ. (1987). Oral contraceptives and rheumatoid arthritis: bias in observational studies of cause-effect relationships. J Chron Dis 40:1063-7.
5696	Spector TD, Hochberg MC (1990). The protective effect of the oral contraceptive pill on rheumatoid arthritis: an overview of the analytic epidemiological studies using meta-analysis. J Clin Epidemiol, 43(11) pp 1221-1230.
29963	Staples JA, Ponsonby A-L, Lim L L-Y, & McMichael AJ. (2003). Ecologic analysis of some immune-related disorders, including type 1 diabetes, in Australia: latitude, regional ultraviolet radiation, and disease prevalence. Environmental Health Perspectives, 111:518-523.

47829	Stark AD, Costas K, Chang HG, Vallet HL (1986). Health effects of low-level exposure to polychlorinated biphenyls. Environ Research, 41: 174-83.
46724	Stavropoulos-Kalinoglou A, Metsios GS, Koutedakis Y, et al (2007). Redefining overweight and obesity in rheumatoid arthritis patients. Ann Rheum Dis, 66:1316-21.
29135	Steenland K, Brown D (1995). Mortality study of gold miners exposed to silica and nonasbestiform amphibole minerals: an update with 14 more years of follow-up. American Journal of Industrial Medicine, Vol 27 pp 217-229.
27600	Steenland K, Nowlin S, Ryan B, Adams S (1992). Use of multiple-cause mortality data in epidemiologic analyses: US rate and proportion files developed by the National Institute for Occupational Safety and Health and the National Cancer Institute. American Journal of Epidemiology, Vol 136 pp 855-862.
46584	Stojanovich L (2006). Influenza vaccination of patients with systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA). Clin Dev Immunol, 13(2-4):373-5.
47961	Stojanovich L, Marisavljevich D (2008). Stress as a trigger of autoimmune disease. Autoimmun Rev, 7(3): 209-13.
46620	Stolt P, Bengtsson C, Nordmark B, Lindblad S, et al (2003). Quantification of the influence of cigarette smoking on rheumatoid arthritis: results from a population based case-control study, using incident cases. Ann Rheum Dis, 62:835-41.
46725	Stolt P, Kallberg H, Lundberg I, Sjogren B, et al (2005). Silica exposure is associated with increased risk of developing rheumatoid arthritis: results from the Swedish EIRA Study. Ann Rheum Dis, 64:582-6.
29970	Stolt P, Nordmark B, Lindblad S, et al (2003). Quantification of the influence of cigarette smoking on rheumatoid arthritis: results from a population based case-control study, using incident cases. Annals of the Rheumatic Diseases, 62:835-41.
46885	Straub RH, Cutolo M (2006). Does stress influence the course of rheumatic diseases. Clin Exp Rheumatol, 24: 225-8.
46562	Straub RH, Cutolo M (2007). Circadian rhythms in rheumatoid arthritis. Implications for pathophysiology and therapeutic management. Arthritis & Rheumatism, 56(2):399-408.
46565	Straub RH, Dhabhar FS, Bijlsma JWJ, Cutolo M (2005). How psychological stress via hormones and nerve fibers may exacerbate rheumatoid arthritis. Arthritis & Rheumatism, 52(1):16-26.
27334	Stricker RB & Goldberg B. (1996). [Letter] G-CSF and exacerbation of rheumatoid arthritis. American Journal of Medicine Vol100:665.
39977	Study of Health Outcomes in Aircraft Maintenance Personnel - SHOAMP, Vol 3, Qualitative Interviews. Final Report July 2003.
37770	Study of Health Outcomes in Aircraft Maintenance Personnel (SHOAMP) (2003). Volume 2: Mortality and cancer incidence study: interim report, July 2003. Commonwealth of Australia.
35415	Study of Health Outcomes in Aircraft Maintenance Personnel (SHOAMP)(2003). Volume 1: Literature Review: Final Report July 2003. Commonwealth of Australia.
32791	Study of Health Outcomes in Aircraft Maintenance Personnel (SHOAMP)(2004). Volume 5: Report on the general health and medical study. Commonwealth of Australia.

30496	Sturkenboom MC, Fautrel B, Rozenberg S, Begaud B, Roullet E & Heinzlef O. (2000). Vaccinations against hepatitis B and rheumatoid arthritis. Pharmacoepidemiology and Drug Safety, 9, Suppl 1:S71-2.
31172	Subair H, Tiwana H, Fielder M, Binder A, et al (1995). Elevation in anti-Proteus antibodies in patients with rheumatoid arthritis from Bermuda and England. J Rheumatol, 22:1825-8.
27507	Susman E. (2002). Smoking inflames joints. Environmental Health Perspectives. 110:A741.
30670	Svendsen AJ, Holm NV, Kyvik K, et al (2002). Relative importance of genetic effects in rheumatoid arthritis: historical cohort study of Danish nationwide twin population. BMJ, 324:264-266.
27497	Svendwen AJ, Holm NV, Kyvik K, et al (2001). Relative importance of genetic effects in rheumatoid arthritis: historical cohort study of Danish nationwide twin population. BMJ, 323:1-4.
46280	Sverdrup B, Kallberg H, Bengtsson C, Lundberg I, et al (2005). Association between occupational exposure to mineral oil and rheumatoid arthritis: results from the Swedish EIRA case-control study. Arthritis Research & Therapy, 7:R1296-303.
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. Arthritis Research & Therapy Vol7 pp R1296-R1303.
48785	Sverdrup B, Klareskog L, Keleinau S (1998). Common commercial cosmetic products induce arthritis in the DA rat. Environ Health Perspect, 106(1): 27-32.
46552	Symmons D (2005). [Comment] Commentary: juvenile idiopathic arthritis - issues of definition and causation. Int J Epidemiol, 34:671-2.
27364	Symmons D, Harrison B (2000). Early inflammatory polyarthritis: results for the Norfolk Arthritis Register with a review of the literature. I. Risk factors for the development of inflammatory polyarthritis and rheumatoid arthritis. Rheumatology, 39: 835-43.
27644	Symmons DP, Bankhead CR, Harrison BJ, Brennan P, Barrett EM, Scott DG, & Silman AJ. (1997). Blood transfusion, smoking, and obesity as risk factors for the development of rheumatoid arthritis. Arthritis & Rheumatism 40:1955-1961.
46269	Symmons DPM (2007). Classification criteria for rheumatoid arthritis - time to abandon rheumatoid factor? Rheumatology, 46:725-6.
27572	Symmons DPM, Bankhead CR, Harison BJ, Brennan P, et al (1997). Blood transfuson, smoking, and obesity as risk factors for the development of rheumatoid arthritis. Results from a primary care-based incident case-control study in Norfolk, England. Arthritis
5697	Symmons DPM, Chakravarty K (1993). Can immunisation trigger rheumatoid arthritis? Ann Rheum Dis, Vol 52 pp 843-844.
46279	Symmons DPM, Silman AJ (2006). What determines the evolution of early undifferentiated arthritis and rheumatoid arthritis? An update from the Norfolk Arthritis Register. Arthritis Research & Therapy, 8(4): 214.
27508	Symmons DPM. (2002). Epidemiology of rheumatoid arthritis: determinants of onset, persistence and outcome. Best Practice & Research Clinical Rheumatology. 16:707-722.

29965	Symmons DPM. (2003). Environmental factors and the outcome of rheumatoid arthritis. Best Practice & Research Clinical Rheumatology, 17:717-727.
46564	Szekanecz Z, Koch AE (2007). Macrophages and their products in rheumatoid arthritis. Curr Opin Rheumatol, 19:289-95.
30639	Takahashi Y, Murai C, Shibata S, Munakata Y, et al (1998). Human parvovirus B19 as a causative agent for rheumatoid arthritis. Proc Natl Acad Sci USA 95:8227-32.
31975	Tanaka N, Ito K, Ishii S, Yamazaki I (1999). Autologous blood transfusion with recombinant erythropoietin treatment in anaemic patients with rheumatoid arthritis. Clin Rheumatol, 18:293-8.
46888	Tandon V, Sharma S, Mahajan A, Khajuria V, Kumar A (2006). Pregnancy and rheumatoid arthritis. Indian Journal of Medical Sciences, 60(8): 334-43.
48811	Tee PG, Sweeney AM, Symanski E, et al (2003). A longitudinal examination of factors related to changes in serum polychlorinated biphenyl levels. Environ Health Perspect, 111(5): 702-07.
31979	Telfer JF & Brock JH. (2002). Expression of ferritin, transferrin receptor, and non-specific resistance associated macrophage proteins 1 and 2 (Nramp1 and Nramp2) in the human rheumatoid synovium. Ann Rheum Dis, 61:741-4.
5698	Thomas DJB, Young A, Gorsuch AN, Bottazzo GF, Cudworth AG (1983). Evidence for an association between rheumatoid arthritis and autoimmune endocrine disease. Ann Rheum Dis, Vol 42 pp 297-300.
48810	Tinazzi E, Ficarra V, Simeoni S, et al (2005). Reactive arthritis following BCG immunotherapy for bladder carcinoma. Clin Rheumatol, 24(4): 425-427.
46561	Tirosh A, Mandel D, Mimouni FB, Zimlichman E, et al (2006). Autoimmune diseases in asthma. Ann Intern Med, 144(12):877-83.
47837	Tithof PK, Schiamberg E, Peters-Golden M, Ganey PE (1996). Phospholipase A2 is involved in the mechanism of activation of neutrophils by polychlorinated biphenyls. Environ Health Perspect, 104(1): 52-8.
30746	Tiwana H, Wilson C, Cunningham P, Binder A, & Ebringer A. (1996). Antibodies to four gram-negative bacteria in rheumatoid arthritis which share sequences with the rheumatoid arthritis susceptibility motif. Br J Rheumatol, 35:592-4 [ABSTRACT].
31173	Tiwani H, Wilson C, Cunningham P, Binder A & Ebringer A. (1996). Antibodies to four gram-negative bacteria in rheumatoid arthritis which share sequences with the rheumatoid arthritis susceptibility motif. British Journal of Rheumatology, 35:592-4.
46710	Toh ML, Miossec P (2007). The role of T cells in rheumatoid arthritis: new subsets and new targets. Curr Opin rheumatol, 19:284-8.
27492	Torrey EF & Yolken RH. (2001). The schizophrenia-rheumatoid arthritis connection: infectious, immune, or both? Brain, Behaviour, and Immunity, 15:401-410.
46694	Toussirot E, Pertuiset E, Martin A, Melac-Ducamp E, et al (2006). Association of rheumatoid arthritis with multiple sclerosis: report of 14 cases and discussion of its significance. J Rheumatol, 33(5):1027-8.
48813	Toussirot E, Roudier J (2007). Pathophysiological links between rheumatoid arthritis and the Epstein-Barr virus: an update. Joint Bone Spine, 74: 418-426.

47392	Treharne GJ, Lyons AC, Booth DA, Kitas GD (2007). Psychological well-being across 1 year with rheumatoid arthritis: coping resources as buffers of perceived stress. British Journal of Health Psychology, 12: 323-45.
47395	Treharne GJ, Lyons AC, Booth DA, Kitas GD (2007). Psychological well-being across 1 year with rheumatoid arthritis: coping resources as buffers of perceived stress. British Journal of Health Psychology, 12: 323-45.
48814	Tryphonas H (1995). Immunotoxicity of PCBs (Aroclors) in relation to Great Lakes. Environ Health Perspect, 103 Suppl 9: 35-46.
46585	Tsai WS, Yang YH, Wang LC, Chiang BL (2006). Abrupt temperature change triggers arthralgia in patients with juvenile rheumatoid arthritis. J Microbiol Immunol Infect, 39:465-70.
47834	Tsuji H, Hirahashi T, Ogata H, Fujishima M (1999). Serum immunoglobulin concentrations and autoantibodies in patients with Yusho. Fukouka Igaku Zasshi, 90(5): 147-9. [Abstract]
47389	Tsukadaira A, Okubo Y, Takashi S, Kobayashi H, Kubo K (2002). Repeated arthralgia associated with granulocyte colony stimulating factor administration. Ann Rheum Dis, 61: 849-50.
46698	Tsurumoto T, Matsumoto T, Yonekura A, Shindo H (2006). Nanobacteria-like particles in human arthritis synovial fluids. Journal of Proteome Research, 5:1276-8.
46443	Tubach R, Kaplan G, Berenbaum F (1999). [Comment] Highly positive dsDNA antibodies in minocycline-induced lupus. Clin Exp Rheumatol, 17(1):124-5.
30662	Tuomi T, Heliovaara M, Palosuo M & Aho K. (1990). Smoking, lung function, and rheumatoid factors. Ann Rheum Dis, 49:753-6 [ABSTRACT].
27367	Turner S & Cherry N. (2000). Rheumatoid arthritis in workers exposed to silica in the pottery industry. Occup Environ Med Vol57:443-447.
30298	Ueno Y & Sohma T. (1992). Alpha-Interferon-induced nodular rheumatoid arthritis in renal cell carcinoma. Annals of Internal Medicine, 117:266-7.
5699	Ueno Y, Sohma T (1992). [Comment] Alpha-interferon-induced nodular Rheumatoid Arthritis in renal cell carcinoma. Ann Intern Med, 117(3) pp 266-267.
27569	Uhlig T, Hagen KB, Kvien TK (1999). Current tobacco smoking, formal education, and the risk of rheumatoid arthritis. Journal of Rheumatology, 26(1) pp 47-54.
17336	Unwin C, Blatchley N, Coker W, Ferry S, Hotopf M, Hull L, Ismail K, Palmer I, David A, Wessely S. (1999). Health of UK servicemen who served in Persian Gulf War. The Lancet, Vol 353 pp 169-178.
5700	van de Laar MAFJ, van der Korst JK (1992). Food intolerance in rheumatoid arthritis. I. A double blind, controlled trial of the clinical effects of elimination of milk allergens and azo dyes. Ann Rheum Dis, Vol 51 pp 298-302.
46278	van der Helm-van Mil A, Breedveld FC, Huizinga TWJ (2006). Definition of disease states in early arthritis: remission versus minimal disease activity. Arthritis Research & Therapy, 8:216.
46277	van der Helm-van Mil A, Verpoort KN, Breedveld FC, et al (2005). Antibodies to citrullinated proteins and differences in clinical progression of rheumatoid arthritis. 7:R949-58.

46560	van der Helm-van Mil AHM, Huizinga TWJ, de Vries RRP, Toes REM (2007). Emerging patterns of risk factor make-up enable subclassification of rheumatoid arthritis. Arthritis & Rheumatism, 56(6):1728-35.
46563	van der Helm-van Mil AHM, Kern M, Gregersen PK, Huizinga TWJ (2006). Variation in radiologic joint destruction in rheumatoid arthritis differs between monozygotic and dizygotic twins and pairs of unrelated patients. Arthritis & Rheumatism, 54(6):2028-30.
46621	van Halm VP, Nielen MMJ, Nurmohamed MT, van Schaardenburg D, et al (2007). Lipids and inflammation: serial measurements of the lipid profile of blood donors who later developed rheumatoid arthritis. Ann Rheum Dis, 66:184-8.
48817	Van Larebeke N, Hens L, Schepens P, et al (2001). The Belgian PCB and dioxin incident of January-June 1999: exposure data and potential impact on health. Environ Health Perspect, 109(3): 265-273.
5701	Vandenbroucke JP, Witteman JCM, Valkenburg HA, Boersma JW, et al (1986). Noncontraceptive hormones and rheumatoid arthritis in perimenopausal and postmenopausal women. JAMA, 255(10) pp 1299-1303.
30266	Vanderbroucke JP. (1983). Oral contraceptives and rheumatoid arthritis. Lancet, July 23:228-9.
46727	Vassilopoulos D, Mantzoukis D (2006). Dialogue between the brain and the immune system in inflammatory arthritis. Ann NY Acad Sci, 1088:132-8.
46570	Vasudev M, Zacharisen MC (2006). New-onset rheumatoid arthritis after anthrax vaccination. Annals of Allergy, Asthma & Immunology, 97:110-2.
5702	Vautier G, Carty JE (1994). [Comment] Acute sero-positive rheumatoid arthritis occurring after hepatitis vaccination. Br J Rheumatol, Vol 33 pp 991.
46707	Verges J, Montell E, Tomas E, Cumelles G, et al (2004). Weather conditions can influence rheumatic diseases. Proc West Pharmacol Soc, 47:134-6.
21007	Vergne P, Bertin P, Bonnet C, Scotto C, Treves R. (2000). Drug-induced rheumatic disorders. Incidence, prevention and management. Drug Safety, 23(4): 279-293.
27873	Vernhes JP, Schaeverbeke T, Fach J, et al (1997). Chronic immunity-driven polyarthritis in hairy cell leukaemia. Report of a case and review of the literature. Revue du Rhumatisme (English Edition), 64(10) pp 578-81.
30284	Vessey MP, Villard-Mackintosh L, & Yeates D. (1987). Oral contraceptives, cigarette smoking and other factors in relation to arthritis. Contraception, 35:457-464.
5703	Vessey MP, Villard-Mackintosh L, Yeates D (1987). Oral contraceptives, cigarette smoking and other factors in relation to arthritis. Contraception, 35(5) pp 457-464.
27333	Vidarsson B, Geirsson AJ, & Onundarson PT. (1995). Reactivation of rheumatoid arthritis and development of leukocytoclastic vasculitis in a patient receiving granulocyte colony-stimulating factor for Felty's syndrome. American Journal of Medicine Vol 98 pp
5704	Vidarsson B, Geirsson ÁJ, Önundarson PT (1995). Reactivation of rheumatoid arthritis and development of leukocytoclastic vasculitis in a patient receiving granulocyte colony-stimulating factor for Felty's syndrome. Am J Med, Vol 98 pp 589-591.

46276	Vierboom MPM, Jonker M, Bontrop RE, & Hart B (2005). Modeling human arthritic diseases in nonhuman primates. Arthritis Research & Therapy, 7:145-54.
5705	Voigt LF, Koepsell TD, Nelson JL, Dugowson CE, Daling JR (1994). Smoking, obesity, alcohol consumption, and the risk of rheumatoid arthritis. Epidemiology, 5(5) pp 525-532.
27360	Walker JG, Littlejohn GO, McMurray NE & Cutolo M. (1999). Stress system response and rheumatoid arthritis: a multilevel approach. Rheumatology Vol 38:1050-7.
5706	Wallace DJ (1987). The role of stress and trauma in rheumatoid arthritis and systemic lupus erythematosus. Seminars in Arthritis and Rheumatism, 16(3) pp 153-157.
5707	Wallace DJ (1994). Does stress or trauma cause or aggravate rheumatic disease? Baillière's Clinical Rheumatology, 8(1) pp 149-159.
5708	Wallace DJ, Meztger AL (1994) Can an earthquake cause flares of rheumatoid arthritis or lupus nephritis. Arthritis and Rheumatism, 37(12) pp 1826-1828.
30289	Webb J, Shilton J, Wicks J (1988). Quinidine-induced lupus erythematosus and arthritis. Medical Journal of Australia, Vol 149 pp 53-54.
46568	Wegrzyn J, Livrozet J-M, Touraine J-L, Miossec P (2002). Rheumatoid arthritis after 9 years of human immunodeficiency virus infection: possible contribution of tritherapy. J Rheumatol, 29:2232-4.
46511	Weidler C, Harle P, Schedel J, Schmidt M, et al (2004). Patients with rheumatoid arthritis and systemic lupus erythematosus have increased renal excretion of mitogenic estrogens in relation to endogenous antiestrogens. J Rheumatol, 31: 489-94.
46709	Weissmann G (2006). The pathogenesis of rheumatoid arthritis. Bulletin of the NYU Hospital for Joint Diseases, 64(1-2):12-5.
46586	Weissmann G (2006). The pathogenesis of rheumatoid arthritis. Bulletin of the NYU Hospital for Joint Diseases, 64(1-2):12-5.
46886	Wells G, Boers M, Tugwell P for the MDA Working Group. Low disease activity state in rheumatoid arthritis: concepts and derivation of minimal disease activity. Clin Exp Rheumatol, 24(Suppl 43): s52-s59.
47402	Westhoff G, Rau R, Zink A (2007). Radiographic joint damage in early rheumatoid arthritis is highly dependent on body mass index. Arthritis & Rheumatism, 56(11): 3575-82.
27359	Weyand CM & Goronzy JJ. (1999). [Letter - Author's reply] Sex-associated factors and the presentation of rheumatoid arthritis: comment on the article by Weyand, et al Arthritis & Rheumatism Vol 42:589-90.
27599	Weyand CM, Goronzy JJ (1997). Pathogenesis of rheumatoid arthritis. Medical Clinics of North America, 81(1) pp 29-55.
46595	Weyand CM, Goronzy JJ (2006). Pathomechanisms in rheumatoid arthritis - time for a string theory? J Clin Invest, 116(4):869-71.
46270	Weyand CM, Goronzy JJ (2006). Pathomechanisms in rheumatoid arthritis - time for a string theory. J Clin Invest, 116(4):869-71.
27640	Weyand CM, Schmidt D, Wagner U & Goronzy JJ. (1998). The influence of sex on the phenotype of rheumatoid arthritis. Arthritis & Rheumatism 41:817-822.

27663	Weyand CM, Schmidt D, Wagner U, Goronzy JJ (1998). The influence of sex on the phenotype of rheumatoid arthritis. Arthritis & Rheumatism, 41(5) pp 817-22.
27376	Weyand CM. (2000). New insights into the pathogenesis of rheumatoid arthritis. Rheumatology Vol39(suppl1):3-8.
45980	Weyland CM, Goronzy JJ (2006). Pathomechanisms in rheumatoid arthritis - time for a string theory? J Clin Invest; 116(4): 869-71
46271	White D, Fayez S, Doube A (2006). Atherogenic lipid profiles in rheumatoid arthritis. NZMJ, 119:1240.
30668	White DG, Mortimer PP, Blake DR, et al (1985). Human parvovirus arthropathy. Lancet, February 23:419-421.
47841	WHO (1992). Polychlorinated Biphenyls and Terphenyls. Environmental health criteria (2nd edition). 140.. Obtained from: http://www.inchem.org/documents/ehc/ehc/ehc140.htm
47409	WHO (2008). Global advisory committee on vaccine safety, 12-13 December 2007. Weekly epidemiological record, 83(4): 37-44.
47060	Wiik AS (2007). The immune response to citrullinated proteins in patients with rheumatoid arthritis. Clin Rev Allergy Immunol, 32:13-21.
27595	Wilder RL (1996). Adrenal and gonadal steroid hormone deficiency in the etiopathogenesis of rheumatoid arthritis. Journal of Rheumatology, 23(suppl 44) pp 10-2.
46696	Williams RO (2006). Pathogenesis and therapy of rheumatoid arthritis. Ernst Schering Found Symp Proc, 1:107-30.
31974	Wilson A, Yu H-T, Goodnough LT & Nissenson AR. (2004). Prevalence and outcomes of anemia in rheumatoid arthritis: a systematic review of the literature. Am J Med 116(7A):50S-57S).
27581	Wilson K, Goldsmith CH (1999)[EDITORIAL]. Does smoking cause rheumatoid arthritis? Journal of Rheumatology, 26(1) pp 1-3.
45975	Wilson LE, Widman D, Dikman SH, Gorevic PD (2002). Autoimmune disease complicating antiviral therapy for Hepatitis C virus infection. Semin Arthritis Rheum, 32: 163-73.
46275	Winchester R (2006). Reshaping Cinderella's slipper: the shared epitope hypothesis. Arthritis Research & Therapy, 8:109.
48818	Wingfors H, Selden A|I, Nilsson C, Haglund P (2006). Identification of markers for PCB exposure in plasma from Swedish construction workers removing old elastic sealants. Ann Occup Hyg, 50(1): 65-73.
31178	Wolfe F & Zwillich SH. (1998). The long-term outcomes of rheumatoid arthritis: a 23-year prospective, longitudinal study of total joint replacement and its predictors in 1,600 patients with rheumatoid arthritis. Arthritis & Rhematism, 41:1072-1082.
30663	Wolfe F & Zwillich SH. (1998). The long-term outcomes of rheumatoid arthritis: a 23-year prospective, longitudinal study of total joint replacement and its predictors in 1,600 patients with rheumatoid arthritis. Arthritis Rheum, 41:1072-82 [ABSTRACT].
27574	Wolfe F (2000). The effect of smoking on clinical, laboratory, and radiographic status in rheumatoid arthritis. Journal of Rheumatology, 27(3) pp 630-7.
27662	Wolfe F, Anderson J (1999). Silicone filled breast implants and the risk of fibromyalgia and rheumatoid arthritis. Journal of Rheumatology, Vol 26 pp 2025-8.

30407	Woodhouse NJY, Mohamedally SM, & Saednejad F. (1976). Letters to the Editor: Acute rheumatoid arthritis developing in a patient treated with salmon calcitonin: evidence against a major anti-inflammatory action of this hormone. J Pharm Pharmac, 28:792-3.
31176	Woolf AD & Cohen BJ. (1995). Parvovirus B19 and chronic arthritis - causal or casual association? Ann Rheum Dis, 54:535-6.
27752	Wordsworth P. (2000). Arthritis and inflammatory bowel disease. Current Rheumatology Reports 2:87-8.
30233	Workplace Health and Safety, Queensland Government. (2001). Silica dust in building and construction. http://www.whs.qld.gov.au/brochures/bro011v1.pdf
46272	Wraith DC, Goldman M, Lambert P-H (2003). Vaccination and autoimmune disease: what is the evidence? Lancet, 362:1659-66.
47400	Yamada R, Yamamoto K (2007). Mechanisms of disease: genetics of rheumatoid arthritis - ethnic differences in disease-associated genes. Nature Clinical Practice, 3(11): 644-50.
46273	Yamamoto K, Yamada R (2007). Lessons from a genomewide association study of rheumatoid arthritis. NEJM, 357(12):1250-1.
47838	Yoshimura T (2003). Yusho in Japan. Ind Health, 41(3): 139-48.
48819	Yoshimura T, Hayabuchi H (1985). Relationship between the amount of rice oil ingested by patients with yusho and their subjective symptoms. Environ Health Perspect, 59:47-51.
5709	Youinou P, Buisson M, Berthelot J-M, Jamin C, et al (1992). Anti-Epstein-Barr virus-nuclear antigen-1, -2A and -2B antibodies in rheumatoid arthritis patients and their relatives. Autoimmunity, Vol 13 pp 225-231.
46887	Youinou P, Jamin C, Saraux A (2007). B-cell: a logical target for treatment of rheumatoid arthritis. Clin Exp Rheumatol, 25: 318-28.
47390	Young A, Koduri G (2007). Extra-articular manifestations and complications of rheumatoid arthritis. Best Pract Res Clin Rheum, 21(5): 907-27.
30494	Zanetti AR. (2001). Update on hepatitis B vaccination in Italy 10 years after its implementation. Vaccine, 19:2380-2383.
5710	Zautra AJ, Okun MA, Robinson SE, Lee D, et al (1989). Life stress and lymphocyte alterations among patients with rheumatoid arthritis. Health Psychology, 8(1) pp 1-14.
48739	Zautra AJ, Parrish BP, Van Puymbroeck CM, Tennen H, et al (2007). Depression history, stress, and pain in rheumatoid arthritis patients. J Behav Med, 30: 187-97.
30354	Zautra AJ. (2003). Comment on "Stress-vulnerability factors as long-term predictors of disease activity in early rheumatoid arthritis". Journal of Psychosomatic Research, 55:303-4.
46471	Ziegler A, Uchanska-Ziegler B (2006). [Letter] Rheumatoid arthritis etiology and HLA-linked odorant receptor gene polymorphisms: comments on the article by Klareskog et al. Arthritis Rheum, 54(8):2705-6.
27753	Zucker-Franklin D. (2000). Non-HIV retroviral associations with rheumatic disease. Current Rheumatology Reports 2:156-162.
5711	Zvaifler NJ (1989). Etiology and pathogenesis of rheumatoid arthritis. Arthritis and Allied Conditions. A Textbook of Rheumatology Eleventh Edition. McCarty DJ (Ed). Lea and Febiger Philadelphia. Section IV Chapter 40 pp 659-660.

46274	Zvaifler NJ (2006). Relevance of the stroma and epithelial-mesenchymal transition (EMT) for the rheumatic diseases. Arthritis Research & Therapy, 8:210.

RMA ID Number	Information received in relation to investigation 264-263 concerning Rheumatoid Arthritis as at 7 October 2008.

S1.1	Repartiation 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 theRepatriation 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
S1.8	Repatriation Medical Authority (RMA) 2004 , 'Addendum to Rheumatoid Arthritis Submission', dated 1 September 2004.
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

S1.14	Name Provided (and removed under s1961 of the VEA) 2007, Request to theRepatriation Medical Authority for Review of Statements of Principles for Rheumatoid Arthritis, dated 21 November 2007
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.

Agency for Toxic Substances and Disease Registry (ATSDR), (updated 25 Oct. 2011), 'Detailed Data Table for the 2011 Priority List of Hazardous Substances that will be the subject of Toxicological Profiles. Retrieved 19 November 2011 http://www.atsdr.cdc.gov/SPL/index.html
Arthritiscures US, 2008-2011 'Polychorinated biphenyls and rheumatoid arthritis'. Retrieved 19 November 2011 http://www.arthritiscures.us/polychlorinated-rheumatoid-arthritis/polychlorinated-rheumatoid-arthritis.htm
Coalition for post-tubal women, 2001-2004 'Can tubal ligation cause menopause? What do doctors say', Retrieved 19 November 2011 http://www.tubal.org/CanTLcauseMeno.htm
Fischbein, A & Wolf, M. 1987, 'Conjugal exposure to polychlorinated biphenyls', *Br J Ind Med,* vol.44, pp. 284-286.
Garner, C.E. & Matthews, H.B. 1998, 'The effect of chlorine substitution on the dermal absorption of polychlorinated biphenyls', *Toxicol Appl Pharmacol*. Vol.149, no.2, pp.150-8.
Milbrath. M.O. Wenger, Y. Chang, C.W. Emond, C. Garabrant, D. Gillespie, B.W. & Jolliet. O. 2008, 'Apparent Half-Lives of Dioxins, Furans, and Polychlorinated Biphenyls as a Function of Age, Body Fat, Smoking Status, and Breast-Feeding', *Environ Health Perspect*, vol. 117, no.3, pp.417-25.
Chemical Industry Archives, (updated: 27 March 2009) 'Monsanto Knew about PCB Toxicity For Decades'. Retrieved 19 November 2011 http://www.chemicalindustryarchives.org/dirtysecrets/annistonindepth/toxicity.asp
Moysich, K.B. Ambrosone C.B. Vena, J.E. Shields, P.G. Mendola, P. Kostyniak, P. Greizerstein, H. Graham, S. Marshall, J. R. Schisterman, E.F. & Freudenheim, J. L. 1998, 'Environmental Organochiorine Exposure and Postmenopausal Breast Cancer Risk', *Cancer Epidemiology, Biomarkers & Prevention*, vol. 7, pp.181-188.
National Center for Environmental Health, 2005, Third National Report on Human Exposure to Environmental Chemicals, *NCEH Pub*. No. 05-0570 http://www.clu-in.org/download/contaminantfocus/pcb/third-report.pdf
United States Environmental Protection Agency (EPA), 'Polychlorinated biphenyls (PCBs) And you. . .' Retrieved 19 November 2011 http://yosemite.epa.gov/R10/TRIBAL.NSF/af6d4571f3e2b1698825650f0071180a/1e4f27736563fc3a882571db00661b15/$FILE/910-F-99-001PCBS.pdf

Skerfving, S. 1995, 'Exposure to pollutants through consumption of contaminated fish - Scand J Work Environ Health, vol. 21, pp.81 -3.
United States Environmental Protection Agency, 1994 'An EPA TSCA assistance document designed to provide the regulated community with Agency interpretations to frequently posed questions', EPA 910-F-99-001, Retrieved 19 November 2011 http://www.epa.gov/osw/hazard/tsd/pcbs/pubs/manual.pdf
US Occupational Health and safety Administration (OSHA), 'Occupational Safety and Health Guideline for Chlorodiphenyl (42% Chlorine) (personal protective equipment) ', Retrieved 19 November 2011 http://osha.gov/SLTC/healthguidelines/chlorodiphenylchlorine/recognition.html
Wester, R.C. Bucks, D.A. Maibach, H.I. & Anderson, J. 1983, 'Polychlorinated biphenyls (PCBs): dermal absorption, systemic elimination, and dermal wash efficiency', *J Toxicol Environ Health*, vol. 12, nos.4-6, pp. 511-9.
Wester, R.C. Maibach, H.I. Bucks, D.A. McMaster, J. Mobayen, M. Sarason, R. & Moore, A. 1990, 'Percutaneous absorption and skin decontamination of PCBs: in vitro studies with human skin and in vivo studies in the rhesus monkey', *J Toxicol Environ Health*, vol.31, no. 4, pp.235-46.