Specialist Medical Review Council

Reasons for Decision

Section 196W
Veterans' Entitlements Act 1986

Re: Statement of Principles No. 72 of 1999
In Respect of Myeloma
Matter No. 2002/1
Requests for Review Declaration No 7

DECLARATION

1. In relation to the Repatriation Medical Authority (the RMA) Statement of Principles No. 72 of 1999 in respect of myeloma and death from myeloma, made under subsection 196B(2) of the *Veterans' Entitlements Act 1986* (the VEA), the Specialist Medical Review Council (the Council) declares, under subsection 196W(5) of the Act, that it is of the view that the sound medical-scientific evidence available to the RMA at the time it determined, amended, or last amended that Statement of Principles was insufficient to justify:

(a) an amendment of the Statement of Principles to include as a factor or factors:

(i) parasitic diseases, including malaria;

(ii) antigenic stimulation; or

(iii) parasitic disease, including malaria, precipitating antigenic stimulation; and

(b) any other amendment of that Statement of Principles.

THE SPECIALIST MEDICAL REVIEW COUNCIL

2. 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. In the absence of the Convener, the Convener must appoint one of the Councillors selected for the purposes of the review as Presiding Councillor at all meetings of the Council for the purposes of that review.

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

4. Professor Alex Cohen AO, MD, FRACP was appointed by the Convener as the Presiding Councillor for this review. The other members of the Council were:

(i) Dr Tony Ryan a Consultant in Public Health and Adjunct Professor and Honorary Senior Fellow of the Department of Public Health at the University of Western Australia;

(ii) Dr David Joske the Head of the Department of Haematology and Director of Bone Marrow Transplantation at Sir Charles Gairdner Hospital and a Consultant Haematologist from the Western Australia Centre for Pathology and Medical Research “Pathcentre” and Clinical Senior Lecturer of the Department of Medicine at the University of Western Australia; and

(iii) Dr Noemi Horvath a Senior Haematologist in the Division of Haematology of the Institute of Medical and Veterinary Science of South Australia and Director of the Clinical Haematology and Bone Marrow Transplantation Unit of Royal Adelaide Hospital.

THE LEGISLATION

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

6. The functions and powers of the Council must be seen in light of the function and purpose of Statements of Principles in the scheme of the VEA. The significance of Statements of Principles to claims under the VEA for pensions in

relation to eligible service is apparent from sections 120A and 120B of the VEA. Section 120 is also of importance.

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

(a) the information:

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

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

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

8. 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. 72 of 1999 in respect of myeloma and death from myeloma, being a Statement of Principles determined by the RMA under section 196B(2) of the VEA ('the reasonable hypothesis standard').

9. In conducting its review, the Council must review all the information that was available to the RMA at the time it determined, amended, or last amended the Statement of Principles.

10. 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

11. On 28 October 1999, the RMA under subsection 196B(2) of the VEA determined Statement of Principles No. 72 of 1999 in respect of myeloma and death from myeloma.

12. On 10 November 1999 the making of the Statement of Principles was notified in the Gazette (No. 45, p. 3912).

13. On 22 November 1999, in accordance with section 196D of the VEA and sections 46A and 48 of the *Acts Interpretation Act 1901* the Statement of Principles was tabled in both the House of Representatives and in the Senate.

14. On 6 January 2000 the Council received an application for review of Statement of Principles No. 72 of 1999 (the application). Specifically the application was concerned with a decision of the RMA of 28 October 1999 not to add a factor in relation to malaria and other parasitic diseases to Statement of Principles No. 72 of 1999 in respect of myeloma and death from myeloma.

15. The Council held a meeting for the purposes of this review, and heard oral submissions on Friday 12 July 2002.

16. The applicant was unable due to personal circumstances to attend the meeting of the Council held on 12 July 2002, and chose not to have a representative appear before the Council to make an oral submission complementing the applicant's written submission.

17. The Repatriation Commission made a written submission. Dr Jon Kelley, representing the Repatriation Commission, made an oral submission to the Council complementing the written submission at the Council's meeting on 12 July 2002.

SUBMISSIONS

Applicant's submission

18. The application to the Council was supported by two letters from the late Professor W R Pitney who was Dean and Professor of Medicine in the University of New South Wales at the time of their writing. These letters were taken by the Council to comprise the basis upon which the applicant sought review of the Statement of Principles. The letters had previously been submitted by the applicant to the RMA, and were part of 'the information' available to the RMA, the subject of review by the Council.

19. The first of these letters dated 18 August 1983 was addressed to Mr T M Garrett, Pensions Officer for Sydney Legacy. It related to a decision by the Repatriation Commission, on appeal, to reject a relationship between multiple myeloma and other unknown factors, which were accepted for the 'allied conditions' of leukaemia and lymphoma. Professor Pitney expressed the view that,

since myeloma was a condition with a very long latency period, it could well be present for a number of years before the diagnosis was made.

20. The second letter from Professor Pitney dated 17 October 1983 was addressed to Mr F Hibbett, Assistant Pensions Officer Sydney Legacy. In this letter Professor Pitney pointed out once more the long latency period for the development of multiple myeloma making it possible that the genesis of the condition could have its source or 'trigger factor' during war service.

21. In this letter he stated that: 'antigenic stimulation from malaria or other parasitic diseases is associated with proliferation of plasma cells and antibody production.' He postulated that cell proliferation could give rise to the emergence of a mutant cell with neoplastic properties. He posed the question as to how long could such a malignant cell remain dormant, or only proliferate slowly, before the tumour mass reached a size sufficient to cause overt disease.

22. In his letter of 17 October 1983, Professor Pitney drew attention to the long latency period and slow development in numbers of abnormal plasma cells. He stated that there was good evidence for a long period of quiescence before the emergence of disease, or for it not to progress at all to clinically threatening dimensions.

Repatriation Commission's submission

23. The submissions (both written and oral) were made on behalf of the Repatriation Commission by Dr Jon Kelley. In introducing the oral submission Dr Kelley stated that, in the search for relevant material to present to the SMRC, all reports (among the information available to the RMA) that provided epidemiological information on the role of malaria and other parasitic diseases in relation to the development of myeloma had been identified. Additionally, those reports which had explored a relationship between antigenic stimulation and the development of myeloma were also studied in view of the basis of the application for review of the Statement of Principles.

24. In the material which had been so identified there had been a total of 13 reports. All of these provided information on, and consideration of, the relationship (if any) between antigenic stimulation and myeloma. Three reports provided information on the association with malaria, but none contained reference to other parasitic diseases.

25. Dr Kelley stated that the direct evidence on the relationship (if any) between malaria and myeloma comprises three case control studies from Europe and the United States. In the view of the Commission, the best of these was the 1987 study by Koepsell et al, which was a population based study of around 700 myeloma

cases set in the United States. In this study, within those 700 myeloma cases, there were only six that had a history of malaria. No association was found in that study between malaria and myeloma risk.

26. A second study quoted by Dr Kelley was that of Gramenzi et al in 1991, on a smaller group of seven myeloma cases with a history of malaria. A non-statistically significant increase of malaria was found between cases and with an odds ratio adjusted for age and gender of 1.8 and, after further adjustment for some other factors, of 1.6.

27. In both the Koepsell and Gramenzi studies the confidence limits included unity and the results were non-statistically significant.

28. The third study of malaria and myeloma by Cuzick and De Stavola in 1988 was set in hospitals in England and Wales and involved 400 myeloma cases. This study reported finding no association between primary malaria and myeloma risk, but did not provide any numerical data.

29. The Repatriation Commission submitted that the RMA had on two occasions specifically considered whether malaria could play a role in the aetiology of multiple myeloma. The first was in 1997, and was undertaken in response to a letter from Dr John R Sullivan dated 1 May 1996. Dr Sullivan had averred, with references, that malaria was capable of producing immune stimulation citing the Tropical Splenomegaly Syndrome as an instance where hypergammaglobulinaemia occurred in association with peripheral B-cell lymphocytosis or uninhibited B-cell production of IgM and cryoglobulins occurred.

30. The Repatriation Commission submitted, on the basis of the material available to the RMA, and in accordance with the definition of sound medical-scientific evidence in the VEA, that there was:

(i) no evidence to support the suggestion that parasitic diseases other than malaria; and

(ii) insufficient evidence to support the suggestion that malaria

are possible links or elements in a reasonable hypothesis connecting operational service with multiple myeloma.

REASONS FOR THE COUNCIL'S DECISION

Pool of information and scope of the review

31. Having reviewed all of the information available to (before) the RMA at the time of creation of the Statement of Principles, the Council was of the view there was no article which was so methodologically flawed that for that or any other reason, it should be excluded from the pool of information. Accordingly, the Council had regard to all the information which was available to the RMA at the time it created the Statement of Principles.

32. After reviewing all the information, the Council decided to confine its attention to the factor/s contended for by the applicant. The Council's task was thus to ascertain whether there was sufficient sound medical-scientific evidence that:

(i) parasitic diseases, including malaria;

(ii) antigenic stimulation; or

(iii) parasitic disease, including malaria, precipitating antigenic stimulation;

(if found to exist in a particular case) could provide a link or element in a reasonable hypothesis connecting operational service to myeloma and death from myeloma.

The Council's analysis of 'the Information'

Consideration of the Applicant's Submission

33. The primary focus of the applicant's submission was that antigenic stimulation constituted a possible link or element connecting operational service with myeloma and death from myeloma. Consideration of whether antigenic stimulation might be responsible in some way for triggering changes that ultimately result in the development of myeloma, has engaged scientific attention for some time. The Council noted that most of the studies seeking to establish this link took place about twenty years ago, as reflected in the views of Professor Pitney, which formed the basis of the applicant's submission. The Council found that since 1994 there have been very few articles supporting this contention.

34. However, as recently as 1995 the authoritative text, the *Oxford Textbook of Oncology*, stated at page 1853:

> It is attractive to speculate that myeloma may, in some part, be either initiated or promoted by chronic antigenic stimulation. High levels of polyclonal immunoglobulins in black populations and anecdotal cases of specificity of the paraprotein for a known antigen provide some support for this view but it remains unproven.

35. It was evident from the information available to the RMA that the esteemed Australian scientist, Professor Don Metcalf, had been approached on a number of occasions for his opinion (in some based upon specific instances) as to the connection (if any) between past exposure to malaria and the subsequent development of lymphosarcoma. In a letter dated 12 April 1983 he commented:

> Although this case was argued on the basis of malarial infections, an equal argument could have been made on the basis of any number of severe and/or sustained infections suffered during war service. [After listing a number of possible examples Professor Metcalf continued]: A sensible set of guidelines here would be: Was the infection **systemic**? Was it **'severe'** eg malaria, dengue? Were there **repeated** episodes of infection? (emphasis added)
>
> Although the case happened to be argued on four cases of lymphosarcoma, it could equally have been argued on the basis of other cancers of lymphoid tissues or some types of leukaemia. These are all cancers of the sets of white cells whose proliferation is stimulated by foreign antigens.

36. Professor Metcalf then went on to list a number of ß-lymphocyte cancers, including multiple myeloma that he considered would come under this rubric. He then itemised a number of cancers for which there was no evidence of a connection between infections and subsequent cancer development, and concluded with three specific infections – hepatitis B virus, schistosomiasis and human T-cell virus infection – in respect of each of which he considered there was indisputable evidence for a causal relationship.

37. In a letter dated 1 November 1989 Professor Metcalf drew attention to his previous opinions in 1983 and 1986 in which he had considered:

> the possibility that severe, recurrent or generalised infection such as malaria might be a contributing factor to the subsequent development in a later life of a cancer of ß-lymphocytes such as multiple myeloma. The proposed link was at best tenuous.

38. Further, Professor Metcalf in his letter of 1 November 1989 drew attention to numerous experiments which his group had carried out over the years to substantiate the possibility that severe and sustained antigenic stimulation might produce malignant change in blood cells. He stated that:

> in no instance has leukaemia or cancer of blood cells resulted. **Our published conclusions from these studies are that, when normal blood cells are placed under intense proliferative stimulation, this situation does not result in the subsequent development of cancer in these cells. In the light of these subsequent studies I am now of the opinion that the original hypothesis proposing such a link is untenable** (emphasis added).

39. The final paragraph of Professor Metcalf's letter of 1 November 1989 states:

> I am therefore of the opinion that **there is no reasonable hypothesis linking the malaria likely to have been suffered by this veteran during his war service and the subsequent development by him of multiple myeloma** (emphasis added).

40. In a letter dated 18 February 1991, Professor Metcalf reiterated these views. Whilst not dealing specifically with multiple myeloma, he stated his own experience, and that of his group, as being unable to substantiate a role for chronic infection/antigenic stimulation in the development of haemopoietic disorders.

Consideration of the Repatriation Commission's written and oral submissions

41. The Council noted the paucity of studies that point to a link between malaria and myeloma.

42. The Council applied standard epidemiological criteria to the studies referred to in the Repatriation Commission's submissions. The Council agreed the few investigations which had been undertaken did not support a link, particularly in terms of strength of association, biological gradient and consistency.

43. There was considerable discussion concerning the relationship between the B-cell and immune suppression of T lymphocytes. The consensus of the Council was that this stimulation followed a different pathway from that associated with the proliferation of B-cells in myeloma, and that no true correlation in epidemiological terms could be established.

Council's Conclusions

Malaria

44. The Council accepted that malaria interacts with the immune system in a pronounced way, such as in the Tropical Splenomegaly Syndrome. However, it was of the view that this was an isolated response. The great majority of those persons suffering from malaria do not show this particular response. This response does not occur sufficiently often to satisfy epidemiological criteria of a link with myeloma. Further, although myeloma is known to be a chronic B-cell malignancy and there is known to be an increased incidence of B-cell malignancies in

conditions of T-cell immunosuppression, such immunosuppression is not known to be present as a consequence of malaria.

45. On the basis of these considerations, the Council was of the view that there was insufficient sound medical-scientific evidence that malaria could provide a link or element in a reasonable hypothesis connecting operational service to myeloma and death from myeloma.

Antigenic Stimulation

46. The Council considered that if there were a connection between antigenic stimulation and myeloma (either alone or in combination with malaria or other parasitic diseases) it would be expected that studies would have identified a much larger number of cases pointing to such a link. On the contrary, the Council noted only sparse and tenuous linkage had been identified.

47. The Council noted further that attempts had been made to link the idio-type of the antibody of the paraprotein to common antigens. As a result, it had been shown that a lot of paraproteins are directed towards a variety of particular external stimuli. This tended rather more to favour as a link to myeloma familial association, rather than antigenic association.

48. Although the suggested link had seemed interesting and attractive during that period in which Professor Pitney's letters had been written and Professor Metcalf had first considered the hypothesis further research and opinion had subsequently failed to support such a link.

49. The Council noted Professor Metcalf's conclusion that the hypothesis (to which he had originally subscribed) that placing normal blood cells under intense proliferative stimulation resulted in the subsequent development of cancer in those cells, was untenable. The Council considered Professor Metcalf's conclusion (that the appropriate epidemiological and clinical criteria were lacking) was supported by the studies of Koepsell et al (SMRC Folder 4, Article 36); Eriksson (SMRC Folder 3, Article 26); Lewis et al (SMRC Folder 3, Article 14); Linet et al (SMRC Folder 4, Article 37).

50. Whilst the requisite connectivity might have been considered a reasonable hypothesis at the time of its first formulation (in or about 1983) there was now a significant body of sound medical-scientific evidence which refuted and discredited it.

51. On the basis of these considerations, the Council was of the view that there was insufficient sound medical-scientific evidence that antigenic stimulation could provide a link or element in a reasonable hypothesis connecting operational

service to myeloma and death from myeloma. While this might have been considered a reasonable hypothesis at the time of its first formulation, the Council concluded there was now a significant body of sound medical-scientific evidence to refute it.

Parasitic disease, including malaria, precipitating antigenic stimulation

52. For the reasons set out above, the Council concluded that there was insufficient sound medical-scientific evidence that antigenic stimulation (however caused) could provide a link or element in a reasonable hypothesis connecting operational service to myeloma and death from myeloma. Consequently, the Council necessarily concluded that there was similarly insufficient sound medical-scientific evidence that antigenic stimulation, precipitated by parasitic disease, including malaria, could provide a link or element in a reasonable hypothesis connecting operational service to myeloma and death from myeloma.

DECISION

53. For the reasons discussed above, the Council was unanimous in its decision that the sound medical-scientific evidence available to the RMA at the time it determined, amended, or last amended Statement of Principles No. 72 of 1999 was insufficient to support:

(i) parasitic diseases, including malaria;

(ii) antigenic stimulation; or

(iii) parasitic disease, including malaria, precipitating antigenic stimulation;

as a link or element in a reasonable hypothesis connecting operational service to myeloma and death from myeloma. Further the Council was of the view that the sound medical-scientific evidence available to the RMA at the relevant times was insufficient to justify any other amendment of Statement of Principles No. 72 of 1999. Accordingly, the Council made the Declaration set out in paragraph 1 above.

EVIDENCE BEFORE THE COUNCIL

Documents

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

Appendix A

SMRC Folder No.	Title No	Title
3	1	Koranda FC (1981). Antimalarials. J Am. Acad. Dermatol 4(6) pp 650-655.
3	2	Hoar Zahm S, Blair A and Weisenburger DD (1992). Sex differences in the risk of multiple myeloma associated with agriculture. British Journal of Industrial Med. Vol 49 pp 815-816.
3	3	Demers PA, Boffetta P, et.al (1995). Pooled reanalaysis of cancer mortality among five cohorts of workers in wood-related industries. Scand J. Work Environ Health Vol 21 pp 179-190.
3	4	Aksoy M (1989). Hematotoxicity and carcinogenicity of benzene (1989). Environmental Health Perspectives Vol 82 pp 193-197.
3	5	Wong O, Trent LS and Whorton MD (1994). An updated cohort mortality study of workers exposed to styrene in the reinforced plastics and composites industry. Occup. Environ. Med. Vol 51 pp 386-396.
3	6	Wong O (1995). Risk of acute myeloid leukaemia and multiple myeloma in workers exposed to benzene. Occup. Environ.Med. Vol 52 pp 380-384.
3	7	Ichimaru M and Mabuchi K (1991). Multiple myeloma among atomic bomb survivors - II. Biological effects. J. Radiat. Res. Supp. pp 168-171.
3	8	Morris Brown L, Burmeister LF, Everett and Blair A (1993). Pesticide exposures and multiple myeloma in Iowa men. Cancer Causes and Control Vol 4 pp 153-156.
3	9	Morris Brown L, Gibson R, Burmeister LF Schuman LM, Everett GD and Blair A (1992). Alcohol consumption and risk of leukemia, non-hodgkin's lymphoma, and multiple myeloma. Leukemia Research 16(10) pp 979-984.
3	10	Friedman GD and Herrinton LJ (1994). Obesity and multiple myeloma. Cancer Causes and Control Vol 5 pp 479-483.

3	11	Heineman EF, Hoar Zahm S, McLaughlin JK, Vaught JB and Hrubec Z (1992). A prospective study of tobacco use and multiple myeloma: evidence against an association. Cancer Causes and Control Vol 3 pp 31-36.
3	12	Heineman EF, Olsen JH, Pottern LM, Gomez M, Raffn E and Blair A (1992). Occupational risk factors for multiple myeloma among Danish men. Cancer Causes and Control Vol 3 pp 555-568.
3	13	Herrinton LJ, Koepsell TD and Weiss NS (1992). Comment - Smoking and multiple myeloma. Cancer Causes and Control Vol 3 pp 391-392.
3	14	Lewis DR, Pottern LM, Brown LM et.al (1994). Multiple myeloma among blacks and whites in United States: the role of chronic antigenic stimulation. Cancer Causes and Control Vol 5 pp 529-539.
3	15	Pottern LM, Heineman EF, Olsen JH, Raffn E and Blair A (1992). Multiple myeloma among Danish women: employment history and workplace exposures. Cancer Causes and Control Vol 3 pp 427-432.
3	16	Filipovich AH, Spector BD and Kersey J (1980). Immunodeficiency in humans as a risk factor in the development of malignancy. Preventive Medicine Vol 9 pp 252-259.
3	17	Friedman GD (1986). Multiple myeloma: Relation to propoxyphene and other drugs, radiation and occupation. International Journal of Epidemiology 15(3) pp 424-426.
3	18	Selected Cancers Cooperative Study Group (1990). The Association of selected cancers with service in the US military in Vietnam. 1. Non-Hodgkin's Lymphoma. Arch Intern Med. Vol 150 pp 2473-2483.
3	19	Cuzick J (1981). Special Article - Radiation-Induced Myelomatosis. N Engl. J Med. 304(4) pp 204-210.
3	20	Lloyd RD and Taylor GN (1991). NTS Fallout-induced multiple myeloma in Utah-(Correspondence). Health Physics 61(5) pp 671-674.

3	21	Uchiumi H, Murakami H, Matsushima T, Tamura J, Sawamura M, Naruse T and Tsuchiya J (19??). Does sarcoidosis induce multiple myeloma? Letters and Correspondence page 220.
3	22	Morrison HI, Wilkins K, Semenciw R, Mao Y and Wigle D (1992). Review - Herbicides and Cancer. Journal of the Natl. Cancer Inst. 84(24) pp 1866-1874.
3	23	McIntyre OR (19??). Myeloma - Chapter 28 - Part Two Specific Neoplasms pp 433-435, 452-457.
3	24	Herrinton LJ, Weiss NS, Koepsell TD et.al (1994). Exposure to hair-coloring products and the risk of multiple myeloma. Am.Journal of Public Health 84(7) pp 1142-1144.
3	25	Joseph G, Barker RL, Yuan B, Martin A, Medeiros J and Peiper SC (1994). Posttransplantation plasma cell dyscrasias. Cancer 74(7) pp 1959-1964.
3	26	Eriksson M (1993). Rheumatoid arthritis as a risk factor for multiple myeloma: A case-control study. Eur. J Cancer 29A(2) pp 259-263.
3	27	Brownson RC (1991). Cigarette smoking and risk of multiple myeloma. Journal of the Natl. Cancer Inst. 83(14) pp 1036-1037.
3	28	Morris Brown L, Everett GD, Burmeister LF and Blair A (1992). Hair dye use and multiple myeloma in white men. Am. J. Public Health 82(12) pp 1673-1674.
3	29	Boice Jr. JD, Morin MM, Glass AG et.. al (1991). Diagnostic X-ray procedures and risk of leukemia, lymphoma and multiple myeloma. JAMA 265(10) pp 1290-1294.
3	30	Hansen ES (1993). A follow-up study on the mortality of truck drivers. Am. Journal of Industrial Medicine Vol 23 pp 811-821.
3	31	Eriksson M and Karlsson M (1992). Occupational and other environmental factors and multiple myeloma: A population based case-control study. British Journal of Industrial Medicine Vol 49 pp 95-103.
3	32	Demers PA, Vaughan TH et.al (1993). A case-control study of multiple myeloma and occupation. American Journal of Industrial Medicine Vol 23 pp 629-639.

3	33	Council on Scientific Affairs (1988). Cancer risk of pesticides in agricultural workers - Council Report. JAMA 260(7) pp 959-966.
3	34	Bourguet CC and Logue EE (1993). Antigenic stimulation and multiple myeloma. Cancer 72(7) pp 2148-2154.
3	35	Lindquist R, Nilsson B, Ellund G and Gahrton G (1987). Increased risk of developing acute leukemia after employment as a painter. Cancer Vol 60 pp 1378-1384.
3	36	Morris P D, Koepsell T D, Daling J R, Taylor J W, Lyon J L, Swanson G M, Child M and Weiss N S (1986) Toxic substance exposure and multiple myeloma: A case-control study. JNCI Vol 76 No 6 pp 987-994.
3	37	Schnatter AR, Katz AM, Nicolich MJ & Theriault G (1993) A Retrospective Mortality Study among Canadian Petroleum Marketing and Distribution Workers Environmental Health Perspectives Supplements 101 (Suppl 6) pp 85 - 99.
3	38	Christie D, Robinson K, Gordon I and Bisby J (1991). A prospective study in the Australian petroleum industry. II. Incidence of cancer. British Journal of Industrial Med. Vol 48 pp 511-514.
3	39	Inskip PD, Kleinerman RA, Stovall M, Cookfair DL et. al (1993). Leukemia, lymphoma and multiple myeloma after pelvic radiotherapy for benign disease. Radiation Research Vol 135 pp 108-124.
3	40	Ott MG, Teta MJ and Greenberg HL (1989). Lymphatic and hematopoietic tissue cancer in a chemical manufacturing environment. Am. Journal of Industrial Med. Vol 16 pp 631-643.
3	41	Bertazzi PA, Pesatori AC, Consonni D, et al (1993). Cancer incidence in a population accidentally exposed to 2,3,7,8-Tetrachlorodibenzo-para-dioxin. Epidemiology 4(5) pp 398-406.
3	42	Preston DL, Kusumi S, Tomonaga M, Izumi S, Ron E et. al. (1994). Cancer incidence in atomic bomb survivors. Part III: Leukemia, lymphoma and multiple myeloma, 1950-1987, Radiation Research Vol 137 pp S68-S97.

3	43	Bethwaite PB, Pearce N and Fraser J (1990) Cancer risk in painters: study based on the New Zealand Cancer Registry. British J of Industrial Medicine Vol 47 pp 742-6.
3	44	Hoar Zahm S, Weisenburger DD, Babbitt PA, Saal RC, Vaught JB and Blair A (1992). Use of hair coloring products and the risk of lymphoma, multiple myeloma, and chronic lymphocytic leukemia. Am J Pub Health 82(7) pp 990-998.
3	45	Linet MS, McLaughlin JK, Malker HSR, Chow W, Weiner JA, Stone BJ, Ericsson JLE and Fraumeni JF (1994). Occupation and hematopoietic and lymphoproliferative malignancies among women: a linked registry study. JOM 36(11) pp 1187-1198.
3	46	Viel JF and Richardson ST (1993). Lymphoma, multiple myeloma and leukaemia among French farmers in relation to pesticide exposure. Soc Sci Med. 37(6) pp 771-777.
3	47	Blair A, Hoar Zahm S, Pearce N E, Heineman EF and Fraumeni JF (1992). Clues to cancer etiology from studies of farmers. Scand J Work Environ Health, 1992, Vol 18 pp 209-215.
4	1	Pukkala E and Notkola V (1997). Cancer incidence among Finnish farmers, 1979-93. Cancer Causes and Control, Vol 8 pp 25-33.
4	2	Anttila A, Pukkala E, Sallmen M, Hernberg S and Hemminki K (1995). Cancer incidence among Finnish workers exposed to halogenated hydrocarbons. JOEM, 37(7) pp 797-806.
4	3	Bertazzi PA, Zocchetti C, Guercilena S, Consonni D, Tironi A, Landi MT & Pesatori AC. (1997). Dioxin exposure and cancer risk: a 15-year mortality study after the "Seveso Accident". Epidemiology, Vol 8 (6) pp 646-52.
4	4	Sans S, Elliott P, Kleinschmidt I, Shaddick G, Pattenden S, Walls P, Grundy C and Dolk H (1995). Cancer incidence and mortality near the Baglan Bay petrochemical works, South Wales. Occupational and Environmental Medicine, Vol 53 pp 217-224.
4	5	La Vecchia C, Negri E, D'Avanzo B and Franceschi S (1989). Occupation and lymphoid neoplasms. British Journal of Cancer, Vol 60 pp 385-388.

4	6	Christie D, Robinson K, Gordon I and Bisby J (1991). A prospective study in the Australian petroleum industry. II Incidence of cancer. British Journal of Industrial Medicine, Vol 48 pp 511-514.
4	7	Christie D, Robinson K, Gordon I and Bisby J (1991). A prospective study in the Australian petroleum industry. I Mortality. British Journal of Industrial Medicine, Vol 48 pp 507-510.
4	8	Yardley-Jones A, Anderson D and Parke DV (1991). The toxicity of benzene and its metabolism and molecular pathology in human risk assessment. British Journal of Industrial Medicine, Vol 48 pp 437-444.
4	9	Khuder SA & Mutgi AB. (1997). Meta-analyses of multiple myeloma and farming. American Journal of Industrial Medicine, Vol 32 (5) pp 510-6.
4	10	Bezabeh S, Engel A, Morris CB & Lamm SH. (1996). Does benzene cause multiple myeloma? An analysis of the published case-control literature. Environmental Health Perspectives, Vol 104 Suppl 6 pp 1393-8.
4	11	Wong O & Raabe GK. (1997). Multiple myeloma and benzene exposure in a multinational cohort of more than 250,000 petroleum workers. Regulatory, Toxicology & Pharmacology, Vol 26 pp 188-199.
4	12	Fritschi L & Siemiatycki J. (1996). Lymphoma, myeloma and occupation: results of a case-control study. International Journal of Cancer, Vol 67 (4) pp 498-503.
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