



THE NEW ZEALAND MEDICAL JOURNAL

Journal of the New Zealand Medical Association, 01-
April-2005, Vol 118 No 1212

Table of contents

Current issue

Search journal

Archived issues

NZMJ Obituaries

1887-2006

Classifieds

Hotline (free ads)

How to subscribe

How to contribute

How to advertise

Contact Us

Copyright

Other journals

The epidemiology of multiple sclerosis in New Zealand

Multiple sclerosis (MS) is a chronic inflammatory demyelinating disease of the central nervous system resulting in severe neurological disability. An accurate estimate of the number of people in New Zealand with MS is currently unknown, partly because hospital outpatient and private neurologist records (the majority of diagnostic care) are not centrally collected and administered.

The epidemiology of MS in New Zealand is of particular interest for several reasons outlined below.

An **increasing disease prevalence** has been consistently observed with decreasing latitude in both New Zealand and Australia.^{1,2} Three explanations for this trend have been proposed: a concentration of genetically susceptible individuals, fewer sunshine hours in the south,³ and unknown environmental factors associated with colder climates.²

Increasing incidence and prevalence of MS has been observed **worldwide⁴ without explanation.** It has been postulated that improved diagnostic techniques such as magnetic resonance imaging (MRI) and an increase in the number of neurologists per population head make it easier to identify cases. **However,** the prevalence has continued to increase in areas even where there has been a reduction in the number of neurologists per population head, as was the situation in Novosibirsk, Russia in 2003, according to local neurologist Larisa Sperling (personal communication to Lou Gallagher, 2003).

Improved patient survival resulting from modern medical treatments has also been shown to be an insufficient explanation, as both incidence and prevalence have increased in areas where no treatment is available.

MS prevalence studies have been conducted in New Zealand since 1968. However, they are not directly comparable since there has been significant variation in case identification methods, clinical definitions of MS, demographic differences in the reference population studied, and inconsistent time periods during which the studies have been conducted. This is a common problem throughout the world, resulting in a situation where only the crudest of comparisons of MS prevalence by geographic region can be made.

According to previous studies, the prevalence of MS in New Zealand Maori seems to be substantially lower than in the European population.⁵⁻⁸ Explanations for this apparent ethnic disparity in MS prevalence include differences in socioeconomic factors (Maori are less likely to present with MS symptoms to medical practitioners) and differences in environmental factors (Maori are less likely to live in areas with exposure to environmental triggers of MS). However, another plausible explanation is that differential susceptibility to MS between Maori and European groups is partially conferred by variation in genetic inheritance,⁸ as has been observed among subpopulations overseas.⁴ If Maori genetic inheritance confers some degree of immunity to MS, how much Maori ancestry is enough?, and what genetic variants are specifically protective for individuals with Maori ancestry?

The optimal epidemiological design for studying the aetiology of complex diseases of relatively low prevalence such as MS is a case-control study. Given that MS aetiology has a significant genetic component it is prudent to design such studies to take into account the relative contribution of genetic and non-genetic factors. This means testing known and suspected MS susceptibility genes such as the vitamin D receptor gene.⁹

To begin addressing the hypothesis that Maori are genetically protected from MS it will also be important to obtain ancestral information either by patient self-report (based on ancestry of four grandparents) or inferred by ancestry-informative genetic markers. This information will allow ancestry-specific genetic risk of MS to be determined and also provide the data to control for ancestry as a potential confounder.

Of paramount importance for such epidemiological research is a comprehensive, clinically well-characterised and standardised MS patient registry. A national registry would also allow tracking of important changes in disease incidence and provide

researchers and clinicians with a resource of patients for new drug trials and for monitoring drug treatments on a national basis.

It is hoped that the first national prevalence survey being jointly funded by the Health Research Council and the MS Society will initiate meaningful epidemiological research on MS in New Zealand in the future. Of particular note will be studies illuminating the relative contribution of ancestry-specific genetic susceptibility and environmental exposures to MS prevalence.

Lou Gallagher (Environmental Epidemiologist), and
Rod Lea (Genetic Epidemiologist)
Population and Environmental Health Programme
Institute of Environmental Science and Research, Limited
Kenepuru Science Centre
Porirua

References:

1. McLeod JG, Hammond SR, Hallpike JF. Epidemiology of multiple sclerosis in Australia. With NSW and SA survey results. *Med J Aust.* 1994;160:117–22.
2. Miller DH, Hammond SR, McLeod JG, et al. Multiple sclerosis in Australia and New Zealand: are the determinants genetic or environmental? *J Neurol Neurosurg Psychiatry.* 1990;53:903–5.
3. van der Mei IA, Ponsonby AL, Dwyer T, et al. Past exposure to sun, skin phenotype and risk of multiple sclerosis: case-control study. *BMJ.* 2003;327:316.
4. Pugliatti M, Sotgiu S, Rosati G. The worldwide prevalence of multiple sclerosis. *Clin Neurol Neurosurg.* 2002;104:182–191.
5. Chancellor AM, Addidle M, Dawson K. Multiple sclerosis is more prevalent in northern New Zealand than previously reported. *Intern Med J.* 2003;33:79–83.
6. Fawcett J, Skegg DC. Geographic distribution of MS in New Zealand: evidence from hospital admissions and deaths. *Neurology.* 1988;38:416–8.
7. Hornabrook RW. The prevalence of multiple sclerosis in New Zealand. *Acta Neurol Scand.* 1971;47:426–38.
8. Miller DH, Hornabrook RW, Dagger J, Fong R. Ethnic and HLA patterns related to multiple sclerosis in Wellington, New Zealand. *J Neurol Neurosurg Psychiatry.* 1986;49:43–6.
9. Partridge JM, Weatherby SJ, Woolmore JA, et al.

Susceptibility and outcome in MS: associations with polymorphisms in pigmentation-related genes. *Neurology*. 2004;62:2323–5.

[Current issue](#) | [Search journal](#) | [Archived issues](#) | [Classifieds](#) | [Hotline \(free ads\)](#)
[Subscribe](#) | [Contribute](#) | [Advertise](#) | [Contact Us](#) | [Copyright](#) | [Other Journals](#)