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ORIGINAL COMMUNICATION

NMOSD and MS prevalence in the Indigenous populations of Australia and New Zealand

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Abstract

Background We studied the prevalence of neuromyelitis optica spectrum disorder (NMOSD) and multiple sclerosis (MS) in Indigenous populations of Australia and New Zealand with the aim of assessing potential differences.

Methods Cases of possible NMOSD and MS were collected from Australia and New Zealand. Clinical details, MR imaging, and serologic results were used to apply 2015 IPND diagnostic criteria for NMOSD and 2010 McDonald criteria for MS. Frequencies of self-determined ethnic ancestry were calculated for confirmed NMOSD, suspected NMOSD, and MS. Prevalence rates for NMOSD and MS according to ancestry were compared.

Results There were 75 cases with NMOSD, 89 with suspected NMSOD, and 101 with MS. NMOSD cases were more likely to have Asian, Indigenous, or Other ancestry compared to suspected NMOSD or MS. There were no differences in the clinical phenotype of NMOSD seen in Indigenous compared to European ancestry populations. Per 100,000, the prevalence estimate for NMOSD in people with Māori ancestry was 1.50 (95% CI 0.52–2.49) which was similar to those with Asian ancestry 1.57 (95% CI 1.15–1.98). NMOSD prevalence in Australian Aboriginal and Torres Strait Islander populations was 0.38 (95% CI 0.00–0.80) per 100,000.

Conclusion The prevalence of NMOSD in the Māori population is similar to South East Asian countries, reflecting their historical origins. The prevalence of MS in this group is intermediate between those with South East Asian and European ancestry living in New Zealand. Both NMOSD and particularly MS appear to be uncommon in the Indigenous populations of Australia.

Keywords Neuromyelitis optica · Aquaporin · Māori · Aboriginal and Torres Strait Islander · Genetics

Introduction

Neuromyelitis optica spectrum disorder (NMOSD) [42] refers to a group of neurological presentations that are usually associated with antibodies to aquaporin 4 (AQP4) [21]. Antibodies to AQP4, a water channel found in high density on astrocytes, are thought to be pathogenic, initiating a cascade of immune cell activation predominantly against periependymal astrocytes via complement fixation [42]. The resulting astrocytopathy is destructive and leads to potentially debilitating deficits affecting the optic nerves, spinal cord, and periependymal regions of the brain that often show limited recovery [16]. The underlying aetiology of NMOSD is poorly understood, but there is evidence for a genetic contribution [11, 25].

Multiple sclerosis (MS) is the archetypal demyelinating disease of the central nervous system and presents with recurrent attacks of symptoms in most patients [10]. Lesions of the optic nerve and spinal cord are again common, but are generally less severe and have a different anatomical distribution. Lesions of the brainstem, cerebellum, and cerebral hemispheres are more commonly seen in MS than in NMOSD [18]. MS arises as a consequence of a mixture of environmental and genetic factors with exposure to Epstein–Barr virus, relative vitamin D deficiency, lack of

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sun exposure, smoking, obesity, diets high in saturated fat, HLA DR1501, and approximately 200 other genetic locations, contributing to susceptibility risk [28, 30].

The importance of genetic involvement in MS was suggested by studies of familial disease [27] and differential prevalence amongst diverse populations [9]. Aboriginal and Torres Strait Islander populations are the First Peoples (Indigenous populations) of Australia and Māori are the Indigenous population of New Zealand. Surveys from the 20th Century did not find any Aboriginal and Torres Strait Islander people with MS [14, 15, 26]. The prevalence of MS in people with Māori ancestry in New Zealand is approximately one-quarter of that seen in the population with European ancestry [31]. Understanding of the causes of NMOSD is less clear and studies of the relative prevalence of this condition compared to MS in different populations may be informative.

The First Peoples of Australia arrived via land bridges and island chains from South East Asia somewhere between 40,000 and 80,000 years ago at periods of lower sea levels associated with recurrent ice ages [41]. Anthropological and more recent genetic data indicate that the arrival of the First Peoples of Australia occurred as part of the earliest migrations out of Africa [19, 23]. There is some genetic evidence for contact with populations from modern-day India about 4000 years ago [34] and a cluster of cases of spinocerebellar ataxia type 3 (SCA3: Machado–Joseph disease) amongst the aboriginal people of Arnhem Land in the Northern Territories of Australia [7] supports other evidence that Portuguese exploration and trading in Australia pre-dated the arrival of Captain Cook and other European settlers by more than 250 years [39]. Following widespread settlement of Australia by Europeans, a combination of disease, conflict, and oppressive governmental policies has led to a decline in Aboriginal and Torres Strait Islander populations to their present level (approximately 3% of the total population in 2011).

The Māori population of New Zealand is widely understood to have arrived by canoe from neighbouring Polynesian islands approximately 700 years ago [5]. European discovery of New Zealand is attributed to the Dutch explorer Abel Tasman in 1642 and widespread European settlement occurred from the mid-18th Century [5]. The Māori population represents about 15% of the total New Zealand population [36]. Whilst the introduction of diseases and conflict also impacted the population, the signing of the Treaty of Waitangi in 1840 is credited with engendering a greater degree of social and cultural cohesion between Māori and Europeans in New Zealand [20].

We recently performed a nationwide prevalence survey of NMOSD across Australia and New Zealand [6]. We have previously demonstrated a threefold higher prevalence of NMOSD in populations with Asian ancestry compared to the remaining population with predominantly European ancestry [6]. Data were collected regarding ethnic ancestry in cases of possible NMOSD and a cohort of age- and sex-matched patients with MS. With the aim of establishing any ethnic differences in the prevalence of NMOSD, we have compared the frequency of NMOSD and MS in the Indigenous populations of Australia and New Zealand.

Methods

Case ascertainment

Possible cases of NMOSD (ICD-10 G36) and age- and sex-matched MS (ICD-10 G35) were identified through a network of 23 public neurology clinics specialising in demyelinating diseases of the central nervous system across Australia and New Zealand. These centres match the population distribution of both countries. Possible cases of NMOSD were identified on the basis of clinical features suggestive of the diagnosis and clinical data were collected using a standard questionnaire as previously described [6]. All cases of possible NMOSD and MS were tested for AQP4 antibodies. Clinical and MR imaging data were collected and were used in conjunction with antibody testing for AQP4 to assign a diagnosis of NMOSD using the 2015 IPND criteria [42], suspected NMOSD (cases of possible NMOSD not meeting 2015 IPND criteria), or MS (2010 McDonald criteria) [33]. Cases were excluded if insufficient clinical data to make a diagnosis were supplied, if an alternate diagnosis became apparent or if AQP4 antibody status was unknown. Institutional human research ethics committee approval was obtained for all participating sites. All participants gave written informed consent. The period of data collection was from 1 January 2011 to 31 December 2013. The prevalence date was 1 July 2013.

Ethnic ancestry was self-determined and recorded for all participants according to the Australian Standard Classification of Cultural and Ethnic Groups 2011 [2] and Stats NZ [37] approved terms with some rationalisation of subgroups (see Table 1).

Study design

We have undertaken three primary analyses. First, we compared the relative frequency of ethnic ancestry in the confirmed cases of NMOSD, suspected NMOSD, and age- and sex-matched MS cases. Second, we have compared the basic demographic and clinical phenotype data for European and Indigenous groups in NMOSD. Third, we have estimated an adjusted prevalence of confirmed NMOSD (2015 IPND criteria) for each ethnic ancestry group using Lincoln–Peterson capture–recapture methodology

Table 1 Ethnic ancestry groups

Original Ancestry Group	Analysis Group
Australian	European
European	
New Zealand	
Aboriginal/Torres Strait Islander	Indigenous
Māori	
Polynesian	Pasifika
Oceanian	
Asian	Asian
African	Other
American	
Other	

with the second source of cases being laboratory identified cases with positive AQP4 antibodies as previously described [22]. Detailed ethnic ancestry beyond Asian/non-Asian (with the non-Asian population being largely European) was not available for the laboratory derived cases of NMOSD. Consequently, for the non-Asian subgroups, adjusted prevalence figures were estimated using a proportional method based on values for all non-Asians within each state/country. Age data were not available for the laboratory derived cases, so age-adjustment was not possible for NMOSD. Because the MS cases collected were only a small sample for case matching with NMOSD no prevalence estimates for MS could be based on these data. For comparison purposes, we have therefore used prior estimates of MS prevalence data for the same region from the past 20 years, three in Australia [4, 35, 43] and one in New Zealand [31]. The relevant McDonald criteria of the time were used for the diagnosis of MS and the same approved terms were used to define self-determined ethnic ancestry [2, 37]. The three Australian studies were combined proportional to their study size.

Statistics

Results are presented as n/N (%) or median (range) as appropriate. Population estimates based on ethnic ancestry were based on census data from Australia [1] and New Zealand [36] for 2013. In the case of the New Zealand Māori population, the responses to the census question relating to ancestry rather than cultural identity was used. The Australian census only asks about cultural identity in relation to Aboriginal and Torres Strait Islander peoples. Comparisons were made using appropriate non-parametric statistics (Chi-square test or Mann–Whitney U test) with significance set at $p < 0.05$. No correction for multiple testing was performed on the basis that these analyses were purely exploratory. All statistical analyses were performed using SPSS® v24 (IBM®, US).

Data availability statement

Complete de-identified, individual level data are available to appropriately qualified researchers on request to the corresponding author, subject to approval of the proposed

project by the Griffith University, Human Research Ethics Committee.

Results

In total, 296 cases of suspected NMOSD and MS were referred and 31/296 (10%) were excluded. Final diagnostic classification was NMOSD in 75 cases, suspected NMOSD in 89 cases, and MS in 101 cases. AQP4 antibodies were positive in 68/75 (91%) of NMOSD cases.

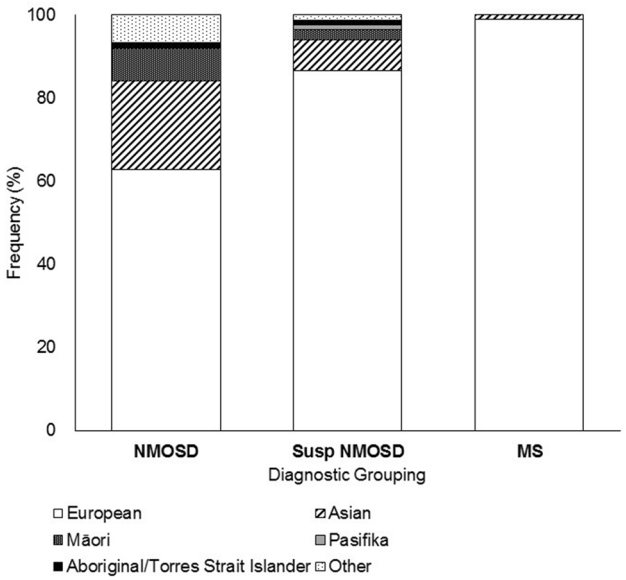

Fig. 1 Relative frequencies of ethnic ancestries in NMOSD, suspected NMOSD, and MS

Comparison of ethnic ancestry in NMOSD, suspected NMOSD and MS

The distribution of ethnic ancestry within each diagnostic group is shown in Fig. 1 and Supplementary Table 1. NMOSD was more common in Asian, Indigenous, and African ancestry populations when compared to MS, with suspected NMOSD cases showing an intermediate pattern. These differences were significant for the overall distribution ($X^2 = 43.693$; $p < 0.0001$), NMOSD compared to MS ($X^2 = 41.407$; $p < 0.0001$), and Indigenous compared to European ($X^2 = 13.58$; $p = 0.0002$). The MS cohort did not include any New Zealand cases, and consequently, there were no data relating to the prevalence of MS in Māori and Pasifika groups from the present study. However, these populations represent only 3% of the total population surveyed across Australia and New Zealand and only one case would have been expected based on the previously observed prevalence in these groups [31]. No cases of MS with Asian ancestry were expected, based on the previously reported prevalence in this population [43]. However, one such case was seen.

Clinical features of NMOSD in indigenous and European populations

The clinical features of cases with Indigenous and European ancestry are shown in Table 2. The only statistically significant difference was fewer cases being female in the Indigenous ancestry group. However, numbers for this group were small and the possibility of a type I error is high.

Table 2 Comparison of clinical features of NMOSD in Indigenous Peoples of Australia and New Zealand and European ancestry populations

Clinical feature	Indigenous	European	*p* value
N	7	47	
Age (years)—median (range)	42 (19 – 81)	50 (19 – 85)	ns
Sex (female)—n/N (%)	4/7 (57)	45/47 (96)	0.01
Age at onset (years)—median (range)	39 (16 – 78)	42 (15 – 85)	ns
Disease duration (years)—median (range)	2.9 (0.1 – 20.7)	3.9 (2.1 – 43.1)	ns
Clinical course—n/N (%)			ns
Monophasic	1/7 (14)	7/47 (15)	
Relapsing remitting	6/7 (86)	38/47 (81)	
Secondary progressive	0/7 (0)	2/47 (4)	
EDSS—median (range)	4.0 (0.0 - 6.5)	4.0 (0.0 - 8.0)	ns
Relapses—median (range)	2 (1 – 6)	4 (1 – 11)	ns
Annualised relapse rate—mean (SD)	1.0 (0.3 – 11.4)	0.8 (0.2 – 12.6)	ns
LESCL—n/N (%)	5/7 (71)	33/40 (83)	ns
Initial MR brain imaging normal—n/N (%)	7/7 (100)	29/43 (67)	ns

EDSS expanded disability status scale, *LESCL* longitudinally extensive spinal cord lesion

Table 3 Estimated prevalence of NMOSD and MS according to ethnic ancestry

Ancestry	NMOSD (ANZ)				MS (AUS)*	MS (NZ)†
	Cases	Adj Cases‡	Population	Adj Prevalence‡ (95% CI)	Prevalence (95% CI)	Prevalence (95% CI)
European	47	113	20,390,241	0.55 (0.45–0.66)	75.2 (71.0–79.3)	103.4 (99.5–107.3)
Asian	16	51	3,259,047	1.57 (1.15–1.98)	1.53 (0.19–2.88)	n/a
Māori	6	9	598,605	1.50 (0.52–2.49)	n/a	15.9 (12.6–19.2)
Pasifika	0	0	295,941	0.00 (0.00–0.24)	n/a	1.61 (0.03–3.21)
Aboriginal/Torres Strait Islander	1	3	798,101	0.38 (0.00–0.80)	0.00 (0.00–0.95)	n/a
African	3	7	380,000	1.84 (0.48–3.21)	n/a	n/a

All prevalence figures are per 100,000

ANZ Australia and New Zealand, *AUS* Australia, *NZ* New Zealand, *Adj* Adjusted, *n/a* not available

*Data derived from [4, 35, 43]

†Data from [31]

‡Adjusted for capture–recapture methodology, geographical distribution, age, and sex (where possible).

NMOSD and MS prevalence estimates in ethnic ancestry subgroups

Table 3 gives the numbers of cases and estimated adjusted prevalence of NMOSD for each ethnic ancestry group from the present study together with prevalence estimates for MS in the same groups from contemporary studies. The three prevalence studies from Australia [4, 35, 43] identified no Aboriginal and Torres Strait Islander people with MS (personal communication, Allan Kermode, Bruce Taylor, and Michael Barnett). NMOSD prevalence for African and Māori peoples were similar to that seen for those with Asian ancestry [1.57 (95% CI 1.15–1.98)] [6]. However, the confidence intervals for all these groups overlap the figure for European ancestry. The figures for NMOSD prevalence in Aboriginal and Torres Strait Islander peoples was similar to that seen in Europeans and no cases were seen in the Pasifika group. However, this was the smallest population studied. The prevalence estimates for the Asian population in NMOSD [1.57 (1.15–1.98)] and MS [1.53 (0.19–2.88)] were similar. This is in keeping with prior studies in Asia that have reported a prevalence ratio for NMOSD and MS that is close to 1:1 [29]. The highest prevalence figure for NMOSD was seen in those with African descent [1.84 (95% CI 0.48–3.21)].

Discussion

We have demonstrated differential prevalence of NMOSD in various ancestral groups when compared to MS. Suspected NMOSD cases have an ancestral distribution somewhere between NMOSD and MS, suggesting that these may be a mixed group. We have found differences in the prevalence of NMOSD in the Indigenous populations of Australia and New Zealand: with prevalence in those with Māori ancestry being similar to those with Asian ancestry, whilst the Aboriginal and Torres Strait Islander groups had a lower prevalence, which is closer to that seen in Europeans. Another striking difference is that MS is almost unheard of in Aboriginal and Torres Strait Islander peoples, whilst MS in those with Māori ancestry is approximately one-quarter of that seen in New Zealanders with European ancestry. Finally, we did not find any major differences in clinical features of NMOSD amongst those with Indigenous ancestry compared to those with European ancestry. However, the numbers were small, and given the differences in prevalence, it may not be appropriate to group Māori, and Aboriginal and Torres Strait Islander ancestries together.

One advantage of our comparison of ethnic ancestry between NMOSD, suspected NMOSD, and MS is that the process of case ascertainment and data collection was the same for all three groups. There was also good consistency between our clinically collected MS cohort and the previously collected ethnic ancestry specific prevalence data. The relative prevalence in those of Asian ancestry was 1% of the overall European MS prevalence. Our estimates of prevalence in Pasifika, Aboriginal, and Torres Strait Islander and African populations have wide confidence intervals reflecting the small numbers.

A number of barriers impede access to healthcare for many Aboriginal and Torres Strait Islander people [13], and this means that our prevalence estimates for NMOSD in these groups may be an underestimate due to reduced reporting and testing. The age profile of Aboriginal and Torres Strait Islander peoples is different to the remainder of the Australian population with average life expectancy being 12 years shorter [32, 40].

A number of socioeconomic inequities remain, but geographically closer proximity to healthcare services means

that the estimates of prevalence in the Māori population may be more accurate.

The finding of prevalence rates of NMOSD in Aboriginal and Torres Strait Islander peoples (0.38 per 100,000) that are similar to and possibly lower than European populations is consistent with the First Peoples of Australia being part of an early wave of migration out of Africa [38]. Our finding of a prevalence rate of NMOSD in Māori populations of New Zealand (1.50 per 100,000) being similar to that seen in South East Asian populations and those of Asian ancestry in Australia and New Zealand is consistent with a relatively recent migration through Polynesian islands, which probably started in the Philippines from adjacent regions of Asia (Indonesia, Malaysia, Vietnam, and China) [17]. As in other studies [12], the highest prevalence rate we observed was in those with African Ancestry (1.84 per 100,000). However, this rate is considerably lower than other African populations that have been studied [12] and may be an underestimate owing to uncertainties regarding the ethnic ancestry of the migrant population to Australia and New Zealand from Africa, with the largest group being from South Africa with European ancestry [3]. It has been noted that genetic studies of all ethnic groups from around the world excepting those from Africa show evidence of a significant bottleneck in genetic diversity dating from 50–100,000 years ago [24]. Another notable difference between African and other populations is the absence of admixture of genes from other proto-humans (e.g. Neanderthals) [24].

In conclusion, we have demonstrated differential prevalence rates for NMOSD in European, Asian, African, Māori, and Aboriginal and Torres Strait Islander populations using a common methodology, and these data are consistent with the direction of difference observed for the populations previously studied. We have not identified any differences in NMSOD clinical phenotype in the Indigenous populations of Australia and New Zealand as compared with Europeans. Ongoing genetic studies in different populations in those with and without disease may help in elucidating the genetic factors associated with susceptibility to NMOSD.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00415-021-10665-9.

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Declarations

Conflicts of interest The authors declare no conflicts of interest of significance relevant to this manuscript. MHB has received honoraria for participation in advisory boards and travel sponsorship from Novartis, BioCSL, Genzyme, and Biogen Idec. MBo has received travel sponsorship and honoraria from Sanofi-Genzyme, Teva, Novartis, Biogen Idec, and Roche. BJB has received honoraria as a board member for GlaxoSmithKline, Biogen Idec, ViiV Healthcare, and Merck Serono, has received speaker honoraria from ViiV Healthcare, Boehringer Ingelheim, Abbott, Abbvie, and Biogen Idec; has received travel sponsorship from Abbott and Viiv Healthcare, and has received research support funding from EI Lilly, GlaxoSmithKline, ViiV Healthcare, and Merck Serono. SAB has received honoraria for attendance at advisory boards and travel sponsorship from Bayer-Scherring, Biogen-Idec, Merck-Serono, Novartis, and Sanofi-Genzyme, has received speakers honoraria from Biogen-Idec and Genzyme, is an investigator in clinical trials sponsored by Biogen Idec, Novartis, and Genzyme, and was the recipient of an unencumbered research grant from Biogen-Idec. HB has received honoraria for serving on scientific advisory boards for Biogen Idec, Novartis, and Sanofi-Genzyme, has received conference travel sponsorship from Novartis and Biogen Idec, has received honoraria for speaking and acting as Chair at educational events organised by Novartis, Biogen Idec, Medscape, and Merck Serono,

serves on steering committees for trials conducted by Biogen Idec and Novartis, is chair (honorary) of the MSBase Foundation, which has received research support from Merck Serono, Novartis, Biogen Idec, Genzyme Sanofi, and CSL Biopharma, and has received research support form Merck Serono. WMC has been the recipient of travel sponsorship from, and provided advice to, Bayer Schering Pharma, Biogen-Idec, Novartis, Genzyme, Sanofi-Aventis, BioCSL and Merck-Serono. RCD has received research funding from the National Health and Medical Research Council, MS Research Australia, Star Scientific Foundation, Pfizer Neuroscience, Tourette Syndrome Association, University of Sydney, and the Petre Foundation and has received honoraria from Biogen-Idec and Bristol-Myers Squibb as an invited speaker. MjF-P has received travel sponsorship from Biogen-Idec and Merck Serono. RH has received honoraria, educational support, and clinic funding from Novartis, Biogen Idec, Genzyme, and BioCSL. AGK has received scientific consulting fees and/or lecture honoraria from Bayer, BioCSL, Biogen-Idec, Genzyme, Merck, Novartis, Sanofi-Aventis, and Teva. TJK has received travel sponsorship from Novartis, BioCSL, Novartis, Merck Serono, and Biogen Idec, has received speaker honoraria from Biogen Idec, BioCSL, Merck Serono, Teva, Genzyme, and Novartis, has received research support from Biogen Idec, Genzyme, GlaxoSmithKline, Bayer-Schering, and Merck Serono, and has received scientific consulting fees from GlaxoSmith-Kline China, Biogen-Idec, and Novartis. JK has received remuneration for advisory board activities and presentations from Bayer Healthcare, Biogen Idec, BioCSL, Genzyme, and Novartis. CK has received travel support, honoraria, and advisory board payments from Biogen Idec, Bayer, Genzyme, Novartis, and Serono. JL-S has received unencumbered funding as well as honoraria for presentations and membership on advisory boards from Sanofi Aventis, Biogen Idec, Bayer Health Care, CSL, Genzyme, Merck Serono, Novartis Australia, and Teva. RALM has received honoraria for attendance at advisory boards and travel sponsorship from Bayer-Scherring, Biogen-Idec, CSL, Merck-Serono, Novartis, and Sanofi-Genzyme. MPMa has received travel sponsorship, honoraria, trial payments, research, and clinical support from Bayer Schering, Biogen Idec, BioCSL, Genzyme, Novartis, and Sanofi Aventis Genzyme. DFM has received honoraria for attendance at advisory boards from Biogen-Idec and Novartis, and travel sponsorship from Bayer-Scherring, Biogen-Idec, and Sanofi-Genzyme. PAMcC has received honoraria or travel sponsorship from Novatis, Sanofi-Avnetis, and Biogen Idec. JAP has received travel sponsorship, honoraria for presentations, and membership on advisory boards from Biogen Idec and Novartis and Sanofi Aventis. JDP has received honoraria for seminars or advisory boards from Teva, Biogen, Sanofi-Genzyme, Novartis, Merck, Bayer and research grants or fellowships from Merck, Novartis, Bayer, Biogen, Sanofi-Genzyme and Teva. SWR has received travel sponsorship, honoraria, trial payments, research and clinical support from Aspreva, Baxter, Bayer Schering, Biogen Idec, BioCSL, Genzyme, Novartis, Sanofi Aventis Genzyme, and Servier, and is a director of Medical Safety Systems Pty Ltd. CPS has received travel sponsorship from Biogen Idec, Novartis, and Bayer-Schering. IS has received remuneration for Advisory Board activities from Biogen, CSL, and Bayer Schering and educational activities with Biogen, CSL and travel sponsorship from Biogen, Novartis, and Bayer Schering. MS has received research support from Novartis, Biogen Idec, and BioCSL. JSp has received honoraria for lectures and participation in advisory boards, and travel sponsorship from Novartis, BioCSL, Genzyme, and Biogen Idec. BVT has received travel sponsorship from Novartis and Bayer Schering. AV and the University of Oxford hold patents and receive royalties for antibody testing. PW and the University of Oxford hold patents for antibody assays and have received royalties, and have received speaker honoraria from Biogen Idec and Euroimmun® AG, and travel grants from the Guthy-Jackson Charitable Foundation. EW has received honoraria for participation in advisory boards from Biogen-Idec and Novartis, travel sponsorship from Biogen-Idec, Bayer-Schering, and Teva, and is an investigator in clinical trials funded by Biogen-Idec and Teva. MPP has received consulting fees and research funding from Atara Biotherapeutics. DA, SBh, SBl, KB, WBr, WBu, CSB, CCM, LC, AC, CD, KD, DF, DG, SHa, APDH, SHe, SHo, SJ-S, SS, AJK, M-WL, CL, CO'G, MPM, CS, RS, JSt, AV, SV, MWa, RCW, MWo, and EMY report no disclosures.

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