Creutzfeldt-Jakob disease surveillance in Australia, update to December 2013

Genevieve M Klug, Alison Boyd, Shannon Sarros, Christiane Stehmann, Marion Simpson, Catriona McLean, Colin L Masters, Steven J Collins

Abstract

Nation-wide surveillance of transmissible spongiform encephalopathies including Creutzfeldt-Jakob disease, is performed by the Australian National Creutzfeldt-Jakob Disease Registry, based at the University of Melbourne. Surveillance has been undertaken since 1993. Over this dynamic period in transmissible spongiform encephalopathy research and understanding, the unit has evolved and adapted to changes in surveillance practices and requirements, the emergence of new disease subtypes, improvements in diagnostic capabilities and the overall heightened awareness and understanding of Creutzfeldt-Jakob disease and other transmissible spongiform encephalopathies in the health care setting. In 2013, routine surveillance continued and this brief report provides an update of the surveillance data collected by the Australian National Creutzfeldt-Jakob Disease Registry prospectively from 1993 to December 2013, and retrospectively to 1970. The report highlights the recent multi-national collaborative study published that has verified the correlation between surveillance intensity and reported disease incidence. *Commun Dis Intell* 2014;38(4):E348–E355.

Keywords: Creutzfeldt-Jakob disease, prion disease, transmissible spongiform encephalopathy, disease surveillance

Introduction

In 1993, the Allars inquiry[1] into the use of cadaver-derived pituitary hormones under the Australian Human Pituitary Hormone Program and the association with 4 medically acquired (iatrogenic) Creutzfeldt-Jakob disease (CJD) deaths recommended the formation of an Australian surveillance unit to monitor further cases of iatrogenic CJD in Australia. The Australian National Creutzfeldt-Jakob disease Registry (ANCJDR) was established in October 1993 within the department of pathology at the University of Melbourne. The monitoring of further Australian iatrogenic CJD cases related to pituitary hormone treatment for infertility or short stature and contaminated dura mater grafts remains one of the core objectives of the ANCJDR; however, the ANCJDR's activities have changed to encompass the surveillance of all types of CJD including sporadic, genetic and variant CJD and other transmissible spongiform encephalopathies (TSEs) such as Gerstmann Sträussler-Sheinker syndrome and fatal familial insomnia.

As described previously,[2] human prion disease can arise sporadically or from genetic or iatrogenic aetiologies. Detailed evaluation of each suspected case added to the register is undertaken to determine whether a case can be excluded from suspicion or classified as a definite, probable or possible TSE case according to World Health Organization (WHO) diagnostic criteria.[3] CJD was made a notifiable disease in all states and territories of Australia as of June 2006. Most initial notifications to the ANCJDR arise through diagnostic testing available through the Registry and this occurs prior to health department notification.

The global incidence of CJD is commonly reported to be 1 case per million per year; however, in most countries with long-standing surveillance systems in place such as France and Switzerland, annual incidence has been reported above this quoted figure.[4] Incidence rates as high as 1.2–2.4 cases per million per year have been reported.[4] Temporally, human prion disease incidence has increased in most countries including Australia, as surveillance mechanisms have evolved, diagnostic testing capabilities have improved and there is generally greater awareness of this rare disease in the health care setting. A recent multi-national collaborative study has verified that surveillance intensity positively correlates with reported disease incidence.[5]

In 2013, several changes have occurred that have or will influence the level of suspected case notifications and possibly future incidence rates of CJD in Australia. These include more discerning practices in relation to suspected case referrals, in part due to ever increasing demands through the diagnostic testing; the suspension of the Queensland autopsy service since January 2013; and an increased number of genetic prion diseases classified in 2013 returning the proportion of genetic cases closer to expected levels. In this report, updated surveillance figures to 31 December 2013 are provided for all retrospective (to 1970) and prospective cases ascertained from 1993 onwards and discussed in relation to these changes and how these may influence case notifications, classifications and overall incidence.

Methods

Patients with a suspected human prion disease are notified to the ANCJDR predominantly through referral for diagnostic cerebrospinal fluid (CSF) 14-3-3 protein detection. Other mechanisms include personal communication with clinicians, families, hospitals and CJD-related groups, and health record searches through hospitals or health departments. Once notified to the ANCJDR, referrals are assessed and if the suspicion of prion disease is supported, the case will be added to the register as a suspected case for continued investigation with the aim of exclusion or classification according to World Health Organization diagnostic criteria. Investigation of register cases can be prolonged as the ANCJDR requires the next-of-kin consent to access and compile the appropriate clinical information from various health information sources for comprehensive evaluation. Response times can vary as the information can be extensive or sources numerous. Medico-demographic questionnaires are offered and forwarded to families if they are willing to contribute, providing valuable information for analysis and evaluation.

The classification of a register case remains as 'incomplete' until the investigation is completed or a definitive result from neuropathological assessment is obtained. Cases may be excluded from the register on the basis of neuropathological examination or after thorough clinical history evaluation. A 'definite' classification requires brain tissue examination, including immunohistochemically and 'probable' and 'possible' cases are reliant on specific clinical profile and diagnostic test outcomes being met as previously described.[3] In this report, the total number of confirmed CJD cases include those that have been classified during 2013.

In conjunction with the ANCJDR's surveillance responsibilities, the Registry provides a diagnostic platform for ante- and post-mortem diagnostic testing for human prion diseases. The testing of CSF for the presence of a family of low molecular weight proteins called 14-3-3 is performed weekly by the ANCJDR. This test, first introduced in 1997, has been readily utilised by the health community and since its introduction referrals have increased substantially to around 400 referrals each year. As described previously, the test provides an increasingly larger proportion of initial notifications of suspected human prion disease to the ANCJDR each year. The ANCJDR also offers services for genetic testing, and Western blot analysis of tonsil and brain tissue from biopsies or autopsies to supplement immunohistochemical assessment. The ANCJDR actively promotes these diagnostic tests so that these options are available to clinicians and families to facilitate the most accurate diagnosis and classification of suspected cases should they wish to pursue these avenues of investigation.

Annual human prion disease incidence rates were calculated using direct age-standardisation, based on the Australian Bureau of Statistics 2000 estimated resident population for Australia and for each state and territory.[6] Population based rates of post-mortem examination in suspected human prion disease were calculated using the Australian Bureau of Statistics 1993–2013 Australia demographic statistics for specific states.[7–11] Health information is collected through a combination of public health and surveillance responsibilities, based on the national notification of communicable diseases. Surveillance activities for the period reported were conducted under ethical approval granted by The University of Melbourne Human Research Ethics Committee.

Statistical analysis (Log-Rank test) was performed using Stata (Intercooled Stata 7, Stata Corporation, College Station, TX).

Results

Fifty-two suspected human prion disease cases were added to the CJD surveillance register in 2013. Cases were notified via request for 14-3-3 CSF test (40 cases), personal communication from clinicians (7 cases), hospitals (2 cases), the CJD Support group network (1 case), and direct health department notifications (2 cases). The proportions of these initial notification sources are consistent with those in previous years and the overall trends for all register cases (Table 1).

While the suspected case notifications added to the register in 2013 were consistent with those observed in 2012 (n=53 cases), notifications were 37% lower compared with the long-term average for the years 1993 to 2012. By the same comparison, fewer notifications from Victoria, Western Australia and Tasmania were received during 2013 (Figure 1), continuing the trend observed during the previous year. Notifications were also lower in Queensland in 2013. Although lower notifications than the long-term average were observed in New South Wales in 2013, there was a modest increase from the previous year returning notifications closer to expected levels. The remaining states and territories remained unchanged from the previous year. Overall, it is estimated that the number of cases added to the register in 2013 was about 20 cases lower than the long-term average of case notifications for 1993 to 2013 (about 70 case notifications per year). While fluctuations are to be expected with annual CJD notifications, it should be noted that since 2009, notifications have been consist-

Table 1: Source of initial notification of suspected transmissible spongiform encephalopathies cases ascertained between 1993 and 2013

Method	Register cases* (%)	Cases removed from the register† (%)	Overall
CSF 14-3-3 protein test request (Since September 1997)	51.8	49.3	50.8
Personal communications			
Neurologists	13.5	12.4	13.0
Neurologists (mail-out reply cards)	2.8	1.8	2.4
Neuropathologists	8.4	8.5	8.4
Neuropathologists (mail-out reply cards)	0.6		0.4
Pituitary Hormones Task Force	1.8	3.2	2.3
Family	3.0	2.5	2.8
Molecular biologist	0.1		0.1
Hospital	0.4	1.3	0.8
Death certificates	9.9	5.6	8.2
Hospital and health department searches			
Hospital medical records	4.8	11.5	7.4
Health department search/state	3.4	7.9	5.1
Morbidity data	1.4	3.6	2.3
Direct health department notification	1.4	0.3	1.0
CJD counselling service	0.4	0.6	0.5
Combined CSF/genetic test request	0.4	0.9	0.6
Genetic test request	0.3	1.7	0.8
Victorian brain bank network		0.2	0.2
Coroner's post mortem request	0.1	0.2	0.1
CJD support group	0.1		0.1
Press	0.1		0.05
UK surveillance unit	0.1		0.05
Total	100.0	100.0	100.0

CSF Cerebrospinal fluid

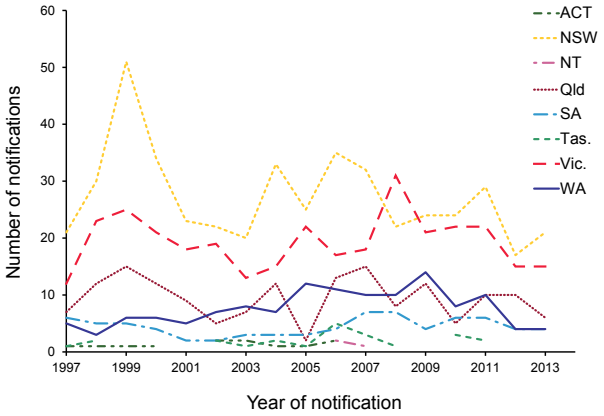
* Register cases; includes all cases currently on the register as classified cases or cases still under investigation.

† Cases removed from the register; includes all suspected cases excluded from the register after detailed investigation.

ently declining in Western Australia. During this period, CSF 14-3-3 protein testing referrals from Western Australia have remained stable; however, there have been fewer cases within this referral group where the suspicion for CJD warrants formal addition to the register and therefore lower case notifications.

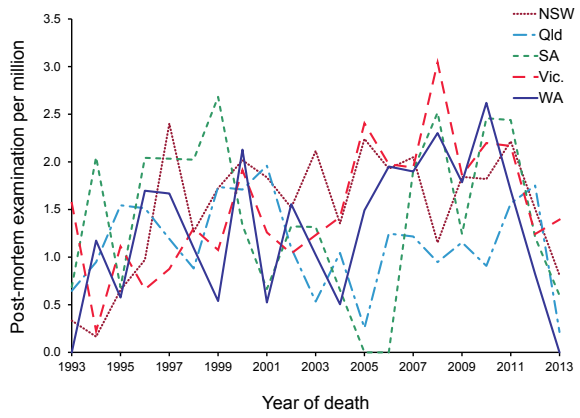
As of 31 December 2013, the majority of the 52 suspected cases added to the register in 2013 were classified as incomplete. Eleven cases were excluded after detailed clinical follow-up (2 cases) or neuropathological examination (9 cases); 9 cases were classified as definite and 1 as probable prion disease.

Figure 1: Prospective notifications of suspected transmissible spongiform encephalopathies cases notified to the ANCJDR, 1997 to 2013, by state or territory and year



Excluding the post-mortem rate in 2013 where figures are still provisional, the average post-mortem rate for all suspected cases on the register who died between 1993 and 2012 is 62%. Over this period, the rate of post-mortems being performed has steadily increased from around 50% during 1993 to 1995 to 70% since 2005. This high post-mortem rate underpins the strong and consistent number of confirmed Australian human prion disease cases recorded since 2005. A comparison of the post-mortem rates in the 5 most populous states in Australia indicates that the overall trend in post-mortem rates has been positive from 1993 to 2012 (Figure 2) with the exception of South Australia where rates have been variable over this period. Excluding the rates shown for 2013, where results are provisional as some post-mortem results are still pending, it is apparent that the post-mortem rate has been declining in Western Australia since 2010. While decreases in New South Wales, South Australia and Victoria are also seen in 2012, they are not as substantial as in Western Australia.

Figure 2: Rate of post-mortem examination in transmissible spongiform encephalopathies suspected case deaths per million population in New South Wales, Queensland, South Australia, Victoria, and Western Australia



Queensland post-mortem rates in contrast, continued to increase from 2005 to 2012 until the CJD autopsy service in that state was suspended. In the other states and territories, post-mortem rates are variable over the surveillance period and show no clear trends due to low population and low numbers of post-mortems being performed.

As of 31 December 2013, there were 989 cases on the register with 757 of these being classified as probable or definite CJD cases. An additional definite iatrogenic case who was treated in Australia, and died in the United Kingdom is included in Table 2; however this case is not classified as an Australian case due to the location at death and is thereby excluded from the overall statistical analysis of Australian CJD cases. Since the start of surveillance, 663 suspected prion disease cases have been excluded from the register after detailed follow-up, with 25 of these being excluded in 2013 (19 after neuropathological examination).

In 2013, 20 cases were re-classified from incomplete to definite prion disease, 4 cases to probable and a single case who died in 2002 was re-classified as possible sporadic CJD, bringing the total number of possible cases to 15. Fourteen of these cases were sporadic and one was iatrogenic CJD (Table 2). Of the 216 incomplete cases, 136 are presently alive. In 2013, the number of incomplete cases under evaluation by the ANCJDR has remained consistent with the number of incomplete cases in 2012. In contrast, there has been a 50% reduction in the number of cases excluded from the register and a 35% reduction in the number of cases classified from incomplete to definite, probable or possible in 2013 compared with 2012.

Age-standardised mortality rates show that the rate of human prion disease mortality in Australia during the period of 1970 to 2013 is increasing, except in 2013, where case evaluation is pending for the majority of deaths (Figure 3) and incidence

Table 2: Classification of Australian National Creutzfeldt-Jakob Disease Register cases, Australia, 1970 to 2013

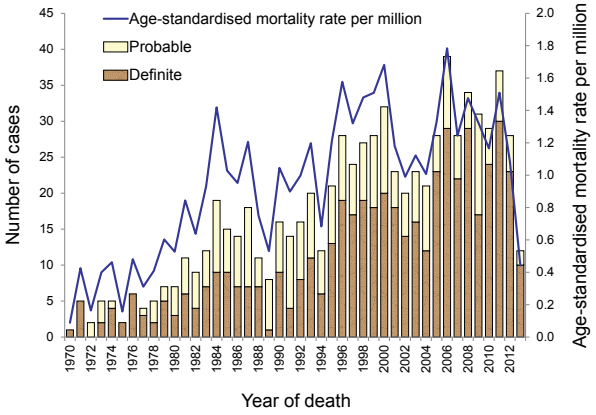
Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	448	49	5*	0	0	502
Probable	241	11	4	0	0	256
Possible	14	0	1	0	0	15
Incomplete					216†	216
Total	703	60	10	0	216	989

* Includes 1 definite iatrogenic case who received pituitary hormone treatment in Australia but disease onset and death occurred while a resident of the United Kingdom. This case is not included in statistical analysis since morbidity and mortality did not occur within Australia.

† includes 136 living cases.

is therefore provisional. In 2013, the age-adjusted mortality rate was 0.44 deaths per million per year and this would be expected to increase after further investigation and classification of incomplete cases. The mean annual age-adjusted mortality rate during the period from 1970 to 2012 was 1.0 death per million (range, 0.1–1.8). For the prospective surveillance period of 1993 to 2012, the mean annual rate is 1.3 deaths per million (range, 0.7–1.8). By state and territory, the majority of regions in Australia have a mean age-adjusted mortality rate above 1 case per million per year between 1993 and 2012 (range, 1.0–1.5). The exceptions are Tasmania and the Northern Territory with 0.7 and 0.9 deaths per million respectively. Restriction of the surveillance data to the period between 2003 and 2012 allows comparisons between states and territories during a timeframe of relatively consistent surveillance practices, diagnostic capabilities and utility with the exception of MRI diagnostics. (Table 3). During this period, Tasmania, the Northern Territory and Queensland have lower than expected mean mortality rates, while Western Australia and Victoria have the highest TSE mortality in Australia.

Figure 3: Number of definite and probable transmissible spongiform encephalopathies cases and age-standardised mortality rate,* Australia, 1970 to 2013, by classification and year



* Age-standardised mortality rates were calculated using the Australian Bureau of Statistics 2000 estimated resident population for Australia.

The proportions of human prion disease aetiologies represented on the register have remained similar to previous years, with the exception of genetic cases. Previously we have reported that the annual number of genetic cases had declined in recent years.[2] In 2013, 6 cases were classified as genetic definite or probable prion disease. Two of these were new cases confirmed as definite genetic cases in 2013, whereas the remaining four were re-classified from definite or probable sporadic cases to genetic cases after further investigation or information was provided to the ANCJDR. The classification of these cases has returned the proportion of genetic TSE closer to the levels expected in the Australian population (Figure 4). Overall, the vast majority of human prion disease cases are sporadic (91.0%) while genetic and iatrogenic cases represent 7.9 and 1.1% respectively of all definite and probable cases.

Based on 757 definite and probable human prion disease cases, 53% per cent were female. Similar proportions for gender exist for all human prion disease aetiologies. Median ages at death for the overall case group or by specific aetiology are largely unchanged from the previous reporting

Table 3: Transmissible spongiform encephalopathies deaths and age-adjusted mortality rates, 2003 to 2012, by year and state or territory

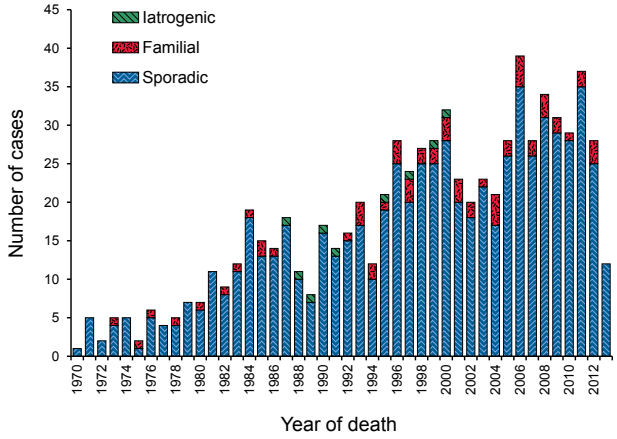
Year	03	04	05	06	07	08	09	10	11	12	13*	Total	Mean age-adjusted mortality rate† (deaths/million/year)
ACT		1		1		2		1				5	1.35
NSW	7	11	10	12	10	6	11	5	14	6	5	97	1.24
NT				2	1							3	0.97
Qld	3			7	2	4	4	2	5	6	2	35	0.68
SA	1	2	1	1	3	5	2	4	4	2	1	26	1.46
Tas.			1	2						1		4	0.71
Vic.	9	5	11	10	6	13	9	13	9	10	4	99	1.71
WA	3	2	5	4	6	4	5	4	5	3		41	1.68
Aus.	23	21	28	39	28	34	31	29	37	28	12	310	1.30

* Provisional figures.

† Age-standardised mortality rates (2003–2012) were calculated using the Australian Bureau of Statistics 2000 estimated resident population for Australian states and territories.

period. Sixty-six years is the median age at death for all cases overall with only a single year difference between males (66 years) and females (67 years). For sporadic cases, 67 years is the median age at death overall and for both males and females. For genetic prion disease, there is a 4 year age difference between males (58 years) and females (62 years) and overall the median age of death from genetic prion disease is 60 years. As there have been no further iatrogenic cases identified since the last reporting period at 31 December 2012, there has been no change to the previously reported median age at death for iatrogenic cases.[2]

Figure 4: Definite and probable human prion disease cases, 1970 to 2013, by aetiology and year



Duration of illness is typically short for human prion disease, especially sporadic CJD, with the median length of illness duration for all cases combined being 3.9 months. By aetiology, median duration was found to be 3.5 months for sporadic cases (range, 0.9–60 months), 6.25 months for iatrogenic cases (range, 2–25 months) and 5.8 months for genetic cases (range, 1.25–192 months). Within 6 months of disease onset, 70% of all TSE cases were deceased. By aetiology, 72% of sporadic, 53% of genetic and 56% of iatrogenic human prion disease were deceased 6 months after the onset of symptoms. Survival is significantly shorter in sporadic CJD than the genetic form ($P < 0.0001$ by Log Rank Test).

Between 1 January and 31 December 2013, no variant CJD or further iatrogenic CJD cases were identified in Australia.

Discussion

The reduced numbers of formal prion disease notifications to the ANCJDR observed in 2012 and 2013 has prompted a review of internal and external factors that may be contributing to the lower number of notifications. One likely factor is a more clinically discerning approach by the ANCJDR before formally adding cases to the register for investigation. This has been necessary due to improved clinician awareness of diagnostic capacity with consequent increased number of suspected case notifications based on suggestive diagnostic investigation results. These include notifications received by the ANCJDR through CSF 14-3-3 protein detection but especially MRI, straining human resources. It is also clear that there are fewer notifications from some states and territories, specifically Victoria, Western Australia and Tasmania in both 2012 and 2013. It is unclear why the numbers from Victoria have decreased and this will be closely monitored in 2014.

In Western Australia, a greater reliance on local health services to manage case investigation and in particular those cases that proceed to post-mortem examination, may contribute to the reduced number of notifications. This may limit the involvement and ability of the ANCJDR to ascertain the true level of suspicion for these cases. The ANCJDR will assess options for improving ascertainment in Western Australia during 2014.

Another important and possibly contributing factor to declining notifications is the suspension of the Queensland CJD autopsy service, which ceased in January 2013 and still remains suspended as of 31 December 2013. It is unclear when the service may be resumed. During 2013, the ANCJDR was aware of several suspected CJD cases in Queensland where autopsies were requested by clinicians and/or families. The effect of this suspension will be marked, particularly on incidence rates in Queensland but also on the overall Australian incidence rates in 2013. Furthermore, notification rates may be lowered as a consequence. As there is only a single year of data with lower notifications in Queensland, notification rates will need to be reviewed during 2014 to determine whether the 2013 data was an isolated fluctuation.

Monitoring annual suspected human prion disease notifications is of importance due to the relationship of this parameter with overall incidence. The ANCJDR led a multi-national collaborative study and demonstrated that greater surveillance intensity incorporating suspected case notifications, referrals for diagnostic testing and post-mortem examination correlates with increased reported disease incidence.[5] In 2013, the ANCJDR published an analysis of datasets from 10 countries with similar surveillance systems and determined a predictive relationship between surveillance intensity and disease incidence. It is therefore of concern that there has

been a sustained change to notification levels and these need to be monitored and assessed carefully in the context of the ANCJDR and local state services activities and processes.

The number of cases classified as definite and probable prion diseases in 2013 (24 cases) is smaller than the number classified in 2012 (39 cases). Definite case classification declined marginally (16% decrease) in 2013 and probable case classifications were 70% lower. An explanation for the lower levels of classifications and exclusion of register cases is in part due to the inflation of the number of cases classified or excluded in 2012 due to concerted efforts by the ANCJDR to classify outstanding cases. After an exceptional year of case classification in 2012, classifications were expected to be lower in 2013 in comparison and return to pre-2012 levels. The ANCJDR aims to maintain a consistent level of case classification with attention focused on probable case classification in 2014.

Despite the decrease in suspected case notifications in 2012, the incidence rate in 2012 has been maintained at expected levels (1.1 cases per million per year). This provides some reassurance that while case notifications have been lower in 2012, the ANCJDR has maintained the ability to detect the expected annual number of prion disease cases in the Australian population despite changes to ANCJDR approaches. No firm conclusions can be made regarding whether these trends will continue in 2013 as the incidence rates are provisional at the time of reporting; however the number of definite cases are predicted to be lower than expected in 2013 due to the suspension of the Queensland autopsy service.

The proportion of annual TSE cases due to genetic prion disease has returned to expected levels during 2013. This is pleasing given the concerns in 2012 that genetic prion disease was under-ascertained between 2009 and 2012, possibly due to the de-centralisation of genetic services to external laboratories and a disconnect with the ANCJDR regarding genetic testing outcomes. Processes have been established in order to redress this issue by genetic services (in conjunction with the CJD support group network), and this has in part contributed to an increased number of genetic prion disease cases classified in 2013. While these processes will prove valuable for case classification in future, the majority of the genetic prion disease identified in 2013 was classified after case investigation, underscoring the utility and importance of comprehensive case evaluation by the ANCJDR.

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