

A prolonged mumps outbreak among highly vaccinated Aboriginal people in the Kimberley region of Western Australia

Revele D Bangor-Jones, Gary K Dowse, Carolien M Giele, Paul G van Buynder, Meredith M Hodge and Mary M Whitty

The mumps vaccine used in Australia is a live attenuated vaccine derived from the Jeryl Lynn strain (genotype A).¹ A single dose produces measurable antibodies in more than 97% of people,^{2,3} and vaccine effectiveness is reported as about 80% after one dose and about 88% after two doses.²

Theoretically, all residents of Western Australia born since 1981 should have received two doses of mumps vaccine, as a single dose was introduced at age 12 months in 1981, followed by a two-dose schedule in 1994 (with the second dose initially at age 12 years and later at age 4–5 years). However, vaccine coverage in the 1980s was not high, and uptake of the second dose varied.⁴ A catch-up program was conducted for children aged 4–16 years in 1998, but school-based delivery was limited to primary schools,⁴ and a booster measles–mumps–rubella (MMR) campaign for 18–30-year-olds in 2000–2005 had poor uptake in WA (unpublished data, Communicable Disease Control Directorate, WA Department of Health, Perth, WA).

Nevertheless, between 1993 and 2006, the number of notified cases of mumps in WA fluctuated between nine and 37 per year, with most cases in non-Aboriginal people. However, in 2007, notified cases rose to 109 per year, as a result of an outbreak among Aboriginal people living in the Kimberley region. This contrasted with a maximum of four cases per year notified from this region during the previous 14 years (unpublished data, Communicable Disease Control Directorate). In 2007, the Kimberley region had an estimated total population of 34 345, of whom 51% were Aboriginal.⁵ The region has six towns, with populations of 2000–15 000 each, and more than 200 discrete Aboriginal communities, with populations ranging from a few families to over 500 people. The mumps outbreak was epidemiologically linked to a cluster of cases in a boarding school in Darwin in the Northern Territory, which borders the Kimberley region.⁶

We investigated the epidemiology of the Kimberley mumps outbreak and the evidence for whether it arose from failure to

ABSTRACT

Objective: To describe a prolonged outbreak of mumps in the Kimberley region of Western Australia in 2007–2008.

Design: Descriptive analysis of all mumps cases notified to the WA Notifiable Infectious Diseases Database for the period 1 July 2007 to 30 June 2008.

Main outcome measures: Notified cases of mumps by patients' place of residence, age, Indigenous or non-Indigenous ethnicity, vaccination status and method of diagnosis.

Results: 84% (153/183) of mumps notifications in WA over the study period occurred in the Kimberley region or were directly linked to Kimberley cases. Median age of patients was 18 years (range, 2–63 years), and 54% of patients were aged less than 20 years. Almost all (92%) were Australian Aboriginal people; 67% (102/153) had received at least one dose of mumps vaccine, and 52% had received two doses. The highest notification rate (1816 cases per 100 000 population) was in the Aboriginal 15–19-years age group, and 92% of these patients had received at least one dose of mumps vaccine. Almost all outbreak cases (94%) were laboratory confirmed. Genotyping was performed on 20 mumps virus isolates: all were genotype J.

Conclusion: A prolonged outbreak of mumps occurred in a well defined, highly vaccinated, predominantly young Aboriginal population in the remote Kimberley region of WA. This outbreak raises questions about the effectiveness and scheduling of the current vaccine (which is genotype A-derived), especially for Aboriginal people. Surveillance of circulating mumps virus genotypes and neutralisation studies will help in evaluating the protection provided by the current vaccine against genotypically different strains.

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vaccinate, or primary or secondary vaccine failure.

METHODS

We extracted details of all mumps notifications from the WA Notifiable Infectious Diseases Database for the period 1 July 2007 to 30 June 2008. Notified cases were included in the outbreak investigation if they were in Kimberley residents or in people with a history of travel to the Kimberley region during the outbreak period. Cases were excluded if infection was acquired overseas, or no epidemiological link to Kimberley cases could be identified.

Vaccination status was confirmed using the Australian Childhood Immunisation Register⁷ and the WA Health Care and Related Encounters (HCARE) databases⁸ and was classified according to the number of doses of mumps vaccine received (one, two or unknown). Notification rates for the Kimberley region were calculated from population estimates derived from the Australian Bureau of Statistics 2006 census using the method of Codde.⁵ Vaccine effi-

cacy (VE) was calculated using the screening method:

$$VE = 1 - [x/(1-x) \times (1-y)/y],$$

where x = proportion of cases vaccinated, and y = proportion of population vaccinated.⁹

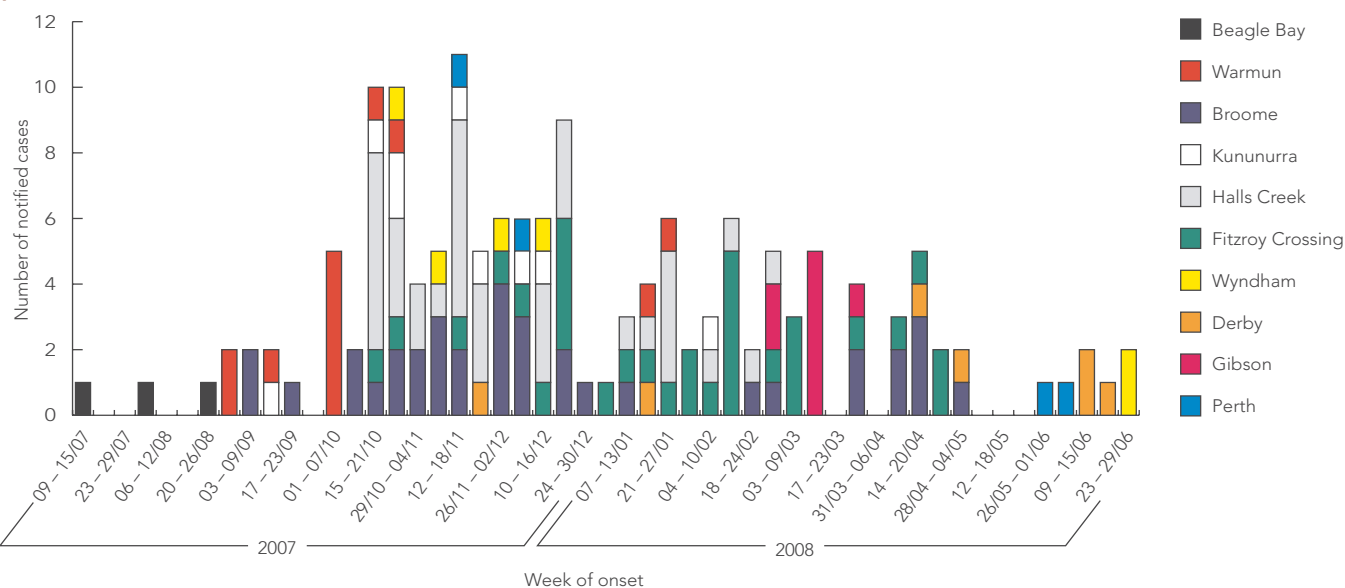
Laboratory results for notified cases were verified by cross-referencing with laboratory data. Ethics approval was not required for this analysis as it was part of routine disease surveillance.

During the outbreak, information sheets for health providers and fact sheets for patients were distributed across the Kimberley region. Providers were encouraged to perform diagnostic tests for all suspected cases and to offer mumps vaccination to contacts of patients if vaccination status was unknown or incomplete. Patients were advised on hand washing, isolation and other infection control practices.

RESULTS

Between 1 July 2007 and 30 June 2008, 183 confirmed mumps cases were notified in WA. The Kimberley outbreak accounted for 84%

1 Epidemic curve of notified mumps cases associated with the 2007–2008 Kimberley outbreak, by date of illness onset and place of residence



of these cases (153/183), and comprised mainly Kimberley residents (92%, 141/153). Twelve patients (8%) were living elsewhere but were epidemiologically linked to the Kimberley region: eight were teenagers from the Kimberley region attending boarding schools in Perth (two patients) or Gibson in the Goldfields region (six patients); two patients were epidemiologically linked to the Goldfields cases; and the remaining two were travellers who visited the Kimberley region during the outbreak period.

The index case was one of 12 Aboriginal students from Beagle Bay, a remote community 120 km from Broome, who returned home for the holidays from a boarding school in Darwin where a cluster of mumps cases had been identified. Two further cases occurred in Beagle Bay, followed by cases in the Kimberley community of Warmun, and the towns of Broome, Kununurra, Halls Creek, Fitzroy Crossing, Wyndham and Derby. The epidemic peaked in October–November 2007 (Box 1).

Demographic characteristics

Of the outbreak cases, 92% (141/153) were in Aboriginal people, 49% of patients (75/153) were male, and the median age was 18 years (range, 2–63 years). Most patients (82%, 126/153) were aged 5–29 years, with 22% (34/153) aged 15–19 years (Box 2). Among the Kimberley Aboriginal population, notification rates were highest in the 15–19-years age group (1816 per 100 000 population), and the notification rate ratio of Abo-

iginal to non-Aboriginal residents of the Kimberley region was 11.5 (141/17 153 v 12/16 851).

Vaccination status and severity of illness

Among the outbreak patients, 67% (102/153) had received at least one dose of vaccine: 52% (80/153) had received two doses, and 14% (22/153) had received only one dose (Box 3). Most patients under 25 years of age (88%, 92/105) had a documented history of receiving at least one dose of vaccine, with 94% (32/34) of patients in the 15–19-years age group having this history (Box 3). Vaccine efficacy for Aboriginal people under 15 years of age, calculated by the screening method, was estimated to be 56% for full vaccination, based on an estimated 90% vaccine coverage.

Clinical data were not reliably collected; however, there were three documented cases of orchitis (2%) and eight documented hospitalisations (5%), one of which was for mumps meningitis.

Laboratory and genotyping results

Of the outbreak cases, 94% (144/153) were laboratory confirmed, and the remaining nine were diagnosed based on clinical criteria combined with an epidemiological link to a laboratory-confirmed case. Investigations performed were: serological testing, 48% of cases (73/153); polymerase chain reaction (PCR) testing of throat swabs, 93% (142/153); and both serological and PCR

2 Age distribution of patients and age-specific notification rates for Kimberley mumps outbreak

Age group (years)	All outbreak patients, no. (%)	Aboriginal patients	
		No. (%)	Rate per 100 000*
0–4	3 (2%)	3 (2%)	97
5–9	19 (12%)	19 (13%)	920
10–14	27 (18%)	24 (17%)	1256
15–19	34 (22%)	34 (24%)	1816
20–24	22 (14%)	19 (13%)	1191
25–29	24 (16%)	21 (15%)	1521
30–34	12 (8%)	11 (8%)	894
35–39	4 (3%)	2 (1%)	172
40+	8 (5%)	8 (6%)	203
Total	153 (100%)	141 (100%)	822

* Aboriginal population of Kimberley region used as denominator. ◆

testing, 44% (68/153) (Box 4). Serological tests gave discordant results (IgG-positive, IgM-negative) in 68% of cases tested (50/73), but all patients who were IgM-negative underwent PCR testing, with positive results for mumps virus in 94% (47/50). Genotype J virus was identified in 20 of the cultured mumps isolates.

DISCUSSION

This prolonged outbreak of mumps in the Kimberley region of WA affected predomi-

3 Vaccination status of Kimberley mumps outbreak patients, by age group

Age group (years)	No. of doses of mumps vaccine (no. of patients [% of age group])			
	Unknown	One dose	Two doses	At least one dose
0–4 (n = 3)	0	2 (67%)	1 (33%)	3 (100%)
5–9 (n = 19)	0	2 (11%)	17 (89%)	19 (100%)
10–14 (n = 27)	3 (11%)	3 (11%)	21 (78%)	24 (89%)
15–19 (n = 34)	2 (6%)	6 (18%)	26 (76%)	32 (94%)
20–24 (n = 22)	8 (36%)	2 (9%)	12 (55%)	14 (64%)
25–29 (n = 24)	15 (63%)	6 (25%)	3 (13%)	9 (37%)
30–34 (n = 12)	12 (100%)	0	0	0
35–39 (n = 4)	3 (75%)	1 (25%)	0	1 (25%)
40+ (n = 8)	8 (100%)	0	0	0
All ages (n = 153)	51 (33%)	22 (14%)	80 (52%)	102 (67%)

nantly young Aboriginal people. The peak incidence was 1816 per 100 000 population in those aged 15–19 years, and the sexes were affected equally. Two-thirds of the patients had received at least one dose of mumps vaccine, and more than half had received two doses. Almost all cases (94%) were laboratory confirmed, with the remainder diagnosed based on clinical signs and epidemiological links to proven cases. Genotyping was performed on cultured isolates from 20 cases, all of which were genotype J.

Mumps outbreaks have been reported recently in other highly vaccinated populations, with similar patterns of age distribution.^{10–14} An outbreak in the United States in 2006 (genotype G) predominantly affected 18–24-year-old students, 51% of whom had received two doses of vaccine.¹⁰ In Canada, a mumps outbreak in 2007 (genotype G) predominantly affected the 20–29-years age group (58% of cases); most patients had received at least one dose of vaccine.¹¹ Similar outbreaks have been reported recently in Moldova,¹² the Czech Republic¹³ and South Korea.¹⁴ An outbreak in the United Kingdom in 2005 (genotype G) affected 56 000 people, but differed in that only a minority of patients had received two vaccine doses.¹⁵ Recent outbreaks in Ireland¹⁶ and in the Netherlands (genotype D)¹⁷ also affected populations with relatively low mumps-vaccine coverage.

There are several possible causes of the limited effectiveness of the mumps vaccine in outbreak settings.¹⁸ The estimated herd-immunity threshold for mumps is 88%–92% in non-outbreak settings, and may be higher in high-risk exposure settings.¹⁹ Vaccine effectiveness is about 80% after one dose^{2,3,20} and about 88% after two.^{20,21}

Even if there were 95% coverage with 95% vaccine effectiveness, population immunity would be 90%, barely reaching the level needed for herd immunity. Coverage data for the Kimberley region are not available for the 1990s, but for 2002–2008, completion of two doses of MMR vaccine by the age of 6 years varied in the order of 80%–95%.⁷ High vaccination coverage in the outbreak area is supported by the finding that all 73 patients who underwent serological testing were positive for mumps IgG.

The Kimberley region is remote, geographically large and very hot for most of the year. Delivering effective vaccines to this area is a logistical challenge, and there may have been primary vaccine failures as a result of a breach of the cold chain. However, vaccine storage temperatures have been logged for the past 15 years as part of quality assurance, and more than 90% of immunisations are provided by well trained Department of Health nurses. Moreover, the outbreak occurred across a wide range of age groups and communities in the region, making cold-chain breach an unlikely cause.

It is unclear why this outbreak was confined to Aboriginal residents of the Kimberley region. The persistence of the epidemic in Broome accords with the movement of Aboriginal people from remote communities and smaller towns to this major commercial, social and medical hub of the Kimberley region. As among college students involved in outbreaks in other countries,^{11,14,22} the social dynamics within Aboriginal communities include overcrowded living arrangements, extensive travel between communities and failure to adhere to isolation requests. This may have facilitated disease transmission, including transmission from

4 Results of mumps laboratory tests (n = 153)

Test	No. tested	Result	
		Positive	Negative
Culture	37 (24%)	23 (62%)	14 (38%)
PCR	142 (93%)	131 (92%)	11 (8%)
Serology			
IgG	73 (48%)	73 (100%)	0
IgM	73 (48%)	23 (32%)	50 (68%)

PCR = polymerase chain reaction. ♦

individuals with subclinical and mild vaccine-modified disease.^{10,23}

There could be differences in the immunogenicity of the mumps vaccine in Aboriginal compared with non-Aboriginal people, because of either genetic or general health differences. Aboriginal people have significantly poorer living conditions and health indicators, which may affect their ability to mount an adequate immune response. However, there is no general indication of inability to respond to other routine childhood vaccines. Rates of most vaccine-preventable diseases targeted by the childhood immunisation program differ little between Aboriginal and non-Aboriginal children; the differences that do exist may reflect failure to vaccinate, rather than vaccine failure.^{24,25}

Secondary vaccine failure is another possible explanation for this outbreak. The duration of post-vaccination immunity is unknown, and there are no data correlating specific antibody titre with susceptibility to mumps.²⁶ Immunity may wane in the absence of continued natural exposure.¹⁰ In the early vaccine era, the vaccine effect was boosted by asymptomatic reinfection with circulating wild-type virus. As the amount of wild-type virus is reduced by increasing vaccination coverage, the opportunity for it to boost immunity decreases.³ In Australia, those born after 1990 experienced higher levels of vaccination coverage and less exposure to wild-type virus.^{4,27} Notification data indicate virtually no mumps transmission in the years preceding this outbreak.

Mumps vaccine-induced immunity may be less effective against heterologous strains, especially with waning levels of neutralising antibody.^{10,28,29} The current mumps vaccine strain is of genotype A lineage, while the virus identified in this outbreak was genotype J. Previous neutralisation tests have shown that vaccination with a mumps vac-

cine containing only one virus genotype may not protect against infection with different genotypes.²⁹ It remains unclear whether the genotype J mumps virus responsible for this outbreak is a recent incursion to Australia. Genotype J was identified in Japan in 1994^{30,31} and in the United Kingdom in 1997³² as a sporadic virus. Prospective monitoring of the genotypes of circulating mumps virus in Australia and elsewhere will be important in investigations of both endemic and epidemic mumps.

This study has limitations. Many of the affected communities are remote. A significant number of cases might have been missed, because they were asymptomatic or mild, or because of limited access to medical care and testing. Data on disease severity were not consistently collected, and it was difficult to locate vaccination records for individuals aged over 25 years. There were also difficulties interpreting results of serological tests, as mumps IgM may be transient or undetectable in infected individuals who have received mumps vaccine.³ After the outbreak was recognised, clinicians were encouraged to take swabs for PCR testing, and ultimately a remarkable 94% of cases were laboratory confirmed.

The prolonged mumps outbreak among Aboriginal residents of the Kimberley region of WA occurred in a highly vaccinated group, with a peak incidence in the 15–19-years age group. The cause is likely to be multifactorial, including social factors leading to increased opportunities for virus transmission, waning immunity, and the presence of mumps virus genotype J strain against which the vaccine strain may confer inadequate immunity. It remains unclear whether the current vaccine strain (genotype A-derived) provides adequate protection against other genotypes, and whether the current two-dose Australian schedule should be modified to prolong protection against disease, especially during epidemic activity. Surveillance of circulating mumps virus genotypes, and neutralisation studies may help determine the effectiveness of the current mumps vaccine.

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COMPETING INTERESTS

None identified.

AUTHOR DETAILS

Reve D Bangor-Jones, MB BCH, MRCP, MPH, Public Health Medical Registrar¹

Gary K Dowse, BMedSc(Hons), MSc, FAFPHM, Medical Epidemiologist¹

Carolien M Giele, BSc(Hons), MPH, GradDipClinEpi, Senior Epidemiologist¹

Paul G van Buynder, MB BS, MPH, FAFPHM, Director¹

Meredith M Hodge, MB BS, MPHTM, FRACP, Senior Microbiology Registrar²

Mary M Whitty, RN, RM, ChildHlthCert, Senior Public Health Nurse³

1 Communicable Disease Control Directorate, WA Department of Health, Perth, WA.

2 PathWest Laboratory Medicine WA, Perth, WA.

3 Kimberley Public Health Unit, Broome, WA.

Correspondence:

reve.d.bangor-jones@health.wa.gov.au

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