A protracted mumps outbreak in Western Australia despite high vaccine coverage: a population-based surveillance study

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Summary

Background In 2007–08, a genotype J mumps outbreak occurred among Aboriginal people in northern Western Australia, despite high vaccine coverage. In March, 2015, a second protracted mumps outbreak occurred in northern Western Australia and spread widely across rural areas of the state. This time the outbreak was caused by a genotype G virus and again primarily affected Aboriginal people. We aimed to describe the epidemiology of this outbreak.

Methods In this population-based surveillance study, we analysed statutory notifications and public health case follow-up data from the Western Australia Notifiable Infectious Diseases Database and vaccination information from the Australian Childhood Immunisation Register. An outbreak case of mumps was notified if the affected person was living in or visiting a community in Western Australia where there was active mumps transmission, and if mumps infection was confirmed by laboratory diagnosis or by an epidemiological link. We analysed case demographics, vaccination status, and age-standardised attack rates in Aboriginal and non-Aboriginal people by region of notification. Laboratory diagnoses were made by real-time RT-PCR, serology, or both, and carried out by the sole public pathology provider in Western Australia.

Findings Between March 1, 2015, and December 31, 2016, 893 outbreak cases were notified. 798 (89%) of 893 outbreak cases were reported in Aboriginal people. 40 (4%) of 893 people were admitted to hospital, and 33 (7%) of 462 men reported orchitis. Mumps attack rates increased sharply with age, peaking in the 15–19 age group. 371 (89%) of 419 people aged 1–19 years were fully vaccinated and 29 (7%) were partly vaccinated. Of the 240 people who tested positive by real-time RT-PCR and had also been tested for mumps-specific IgG and IgM, 165 (69%) were positive for IgG but negative for IgM, indicating the importance of RT-PCR testing for diagnosis in vaccinated populations. None of the cases from the 2007–08 genotype J outbreak were re-notified.

Interpretation The number of mumps outbreaks reported in recent years among highly vaccinated populations, including Indigenous populations, has been growing. More widespread and pre-emptive use of the third dose of measles, mumps, and rubella vaccine might be required to control and prevent future outbreaks in high-risk populations. Research should explore the benefit of increasing the intervals between vaccine doses to strengthen the durability of vaccine protection.

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Introduction

Before universal vaccination programmes were introduced, mumps was a common childhood infectious disease.[1] Although childhood vaccination has dramatically reduced mumps incidence, there has been a resurgence of mumps outbreaks in recent years among adolescents and young adults in many high-income countries, despite high vaccine coverage, raising questions about the duration of effectiveness of mumps-containing vaccines.[2]

The live attenuated Jeryl Lynn strain mumps vaccine was first recommended in 1981, as part of the Australian national vaccination schedule for children at 12 months of age.[3] In 1992, a two-dose measles, mumps, and rubella (MMR) schedule began with the second dose administered at age 12 years. In 1998, the second dose was moved to age 4 years with catch-up offered to children aged 4–16 years. Hence, all individuals born in Australia since 1981 should have received two doses of mumps-containing vaccine.[3]

Western Australia is Australia's largest state and is sparsely populated except for the southwestern corner. Of 2·5 million residents, 2 million live in the capital, Perth.[4] Besides Perth, the state is generally divided into seven regional areas for the provision of health services. Although only 4% of the Western Australia population identify as Aboriginal, the proportion is higher outside Perth, including 44% in the Kimberley region.[4] Since mumps became a notifiable disease in Western Australia in 1993, incidence has remained low. Between 1993 and 2007 there were an average of 23 cases notified annually (range 7–39; Communicable Disease Control Directorate [CDCD], unpublished data). These cases primarily reflected infections acquired overseas with limited local transmission.[5] In 2007–08, a mumps outbreak occurred in the Kimberley, a large, sparsely populated region in

Research in context

Evidence before this study

We searched PubMed without language restrictions using the keywords "mumps" and "outbreak", and "mumps vaccine", and Google Scholar without language restrictions using the keywords "mumps outbreak" for papers published through June, 2018. Although universal childhood vaccination has dramatically reduced mumps incidence, there has been a resurgence in recent years of mumps outbreaks in high-income countries, primarily among adolescents and young adults. This resurgence has occurred despite high vaccine coverage in most of these populations, raising questions about the duration of effectiveness of mumps vaccines and their ability to cross-protect against infections due to heterologous virus strains. Many reported outbreaks have been in student populations, particularly in residential settings, and there have been a few reports showing ethnic differences in susceptibility.

Added value of this study

Our analysis of statutory notification data for Western Australia found that during the large genotype G mumps outbreak, the risk for Aboriginal Australians to acquire mumps was 37 times higher than the risk for non-Aboriginal Australians living in the same areas, despite high vaccination coverage in both groups. Mumps attack rates peaked in people aged 15–19 years, and the proportion of mumps cases increased with time since the second measles, mumps, and rubella (MMR) vaccine dose, plateauing once 7–9 years had elapsed. An older age cohort with a longer interval between mumps vaccine doses appeared to be at lower risk than those in whom the interval between doses was shorter; longer intervals might confer better vaccine protection. None of the cases from the 2007–08 genotype J outbreak were re-notified, suggesting greater cross-protection might be conferred by natural infection than by the current Jeryl Lynn strain mumps vaccine. Many previously vaccinated individuals were IgG positive and IgM negative, indicating that serology in vaccinated individuals might be an ineffective method of mumps diagnosis. A staged mumps ring vaccination intervention in affected communities was insufficient to contain the spread of the outbreak. Large household sizes and high amounts of population mobility and social interaction are likely to have contributed to the disparity in mumps incidence between Aboriginal and non-Aboriginal people, on a background of waning immunity and immune escape.

Implications of all the available evidence

Available vaccines do not seem to offer sufficient protection to prevent mumps outbreaks in adolescents and young adults in settings and population groups with high levels of mobility and social interaction. Both waning immunity and immune escape could play a role in decreasing the effectiveness of the Jeryl Lynn strain mumps vaccine over time, but it is unclear whether more factors affect the immunogenicity of the vaccine in some population groups. To prevent similar outbreaks, the need for an optimal age for routine administration of a third dose of the MMR vaccine in at-risk populations should be considered. In populations with high levels of residential crowding, social interaction, and mobility, the interval between the first and second dose of MMR could be increased as a way to strengthen the durability of vaccine protection.

the state's north. In total, 183 cases of mumps were notified to the CDCD during this period. 153 (84%) of these people lived in the Kimberley or were epidemiologically linked to the outbreak, and 141 (92%) of 153 were Aboriginal Australians.[6] In the 12 years preceding this outbreak, less than one case per year was reported from this region (CDCD, unpublished data).

We describe here a second, much larger mumps outbreak that started in the Kimberley region in March, 2015, and extended to December, 2016, and consider the role of immunological and other factors in explaining the apparent increased susceptibility to mumps among Aboriginal Australians.

Methods

Case definition and data sources

An outbreak case of mumps was defined as a case notified between March 1, 2015, and Dec 31, 2016, that involved a person living in or visiting a community in Western Australia where there was active mumps transmission (ie, at least one other confirmed case of mumps was likely to have been infectious within the incubation period of this new case).

Cases were classified as either confirmed or probable. A confirmed case was either positive for mumps by PCR, had clinical evidence (acute parotitis or swelling of other salivary glands lasting 2 days or more) and detection of mumps IgM (if there was no recent mumps vaccination), or had clinical evidence and an epidemiological link to a confirmed case. An epidemiological link involved contact between two people at a time when one was likely to be infectious and the other contracted disease within 12–25 days after this contact, and at least one person in the chain of transmission had laboratory-confirmed mumps infection.[7] A probable case was a person who had clinical evidence of mumps illness with linkage to a group or community with known active mumps transmission. All probable and confirmed cases were combined for any analyses. Children younger than 1 year were excluded from analyses because they were not eligible for vaccination, but were counted as cases.

Case information including demographics, clinical details, laboratory details, Aboriginality, and vaccination status were recorded in the WA Notifiable Infectious Diseases Database, which is an electronic database containing complete statutory disease notification data

since 1990. Mumps has been notifiable in Western Australia since 1993. Disease spread was determined by characterising the cases in terms of time, place, and person. Hospital admission or presence of orchitis were ascertained from diagnosing clinicians during public health follow-ups or from laboratory request forms. Vaccination status was confirmed through the Australian Childhood Immunisation Register, a population register with vaccination records for all Australian children born since 1995,[8] and if possible also through a medical record management system used by the regional Population Health Units, most applicable to individuals born before 1995. If the vaccination status of individuals born before 1995 could not be ascertained locally, records were checked in other Population Health Units in the state.

Procedures

The clinical specimens that were collected were predominantly buccal or throat swabs and urine samples for real-time RT-PCR and blood for serology. Most samples were tested at PathWest Laboratory Medicine (Nedlands, WA, Australia), the sole public pathology provider in WA. If a sample was tested at another laboratory, we requested that the sample be sent to PathWest for confirmatory testing and genotyping. Mumps-specific IgM and IgG antibodies were detected using enzyme immunoassay and immunofluorescence (DiaSorin Liaison XL, Saluggia, Italy). RT-PCR targeted the haemagglutinin-neuraminidase (*HN*) gene. Genotyping of all RT-PCR-positive cases was attempted until August, 2015, using the small hydrophobic (*SH*) gene of the mumps virus[9] as the target. After this date only a selection of cases were genotyped, including initial cases in previously unaffected communities or regions, and cases for which vaccine-associated symptoms were suspected. The prototype *SH* genotype G sequence for the 2015–16 outbreak was first identified in Perth in 2013, and was used as the comparator sequence throughout the outbreak.

Statistical analysis

We analysed case demographics, vaccination status, and age-standardised attack rates in Aboriginal and non-Aboriginal people by region of notification. We calculated rate ratios by comparing attack rates in Aboriginal and non-Aboriginal people. For age-standardised attack rates and rate ratios, 95% confidence intervals are presented. We sourced population data using the Rates Calculator (version 9.5.5), a software package developed by the Western Australia Department of Health (Perth, WA, Australia) and based on 2011 Australian census data. Rates were age-standardised using the direct method. Individuals were considered fully vaccinated if they had received two or more doses of the MMR vaccine.[10] Partly vaccinated individuals had received only one dose. To explore the possible effects of waning immunity on susceptibility to mumps infection, we measured the distribution of mumps outbreak cases by years since the second dose of MMR vaccine, excluding individuals who received more than two doses, those who received their most recent dose within 2 weeks of mumps onset, and those who received their first dose at less than 12 months of age.[10] We used Stata (version 14.2; College Station, TX, USA) for all analyses.

Role of the funding source

There was no funding source for this study. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

Between March 1, 2015, and Dec 31, 2016, 932 mumps cases were notified in Western Australia. 39 cases did not meet the outbreak case definition because the individuals had acquired the infection overseas or had no identifiable link to the outbreak. Of the 893 outbreak cases remaining, 865 were classified as confirmed and 28 as probable.

798 (89%) of 893 outbreak cases were in Aboriginal people, and 797 (89%) were in residents of the sparsely populated Kimberley, Pilbara, and Goldfields regions (table 1). The sex and ages of Aboriginal and non-Aboriginal patients were similar. More Aboriginal patients than non-Aboriginal patients were residents of remote areas. 40 (4%) people were admitted to hospital and 33 (7%) of 462 men reported orchitis, with similar proportions in Aboriginal and non-Aboriginal patients. None of the cases from the 2007–08 genotype J outbreak were re-notified (CDCD, unpublished data).

	Aboriginal people (n=798)	Non-Aboriginal people (n=95)	Total (n=893)
Sex			
Male	417 (52%)	45 (47%)	462 (52%)
Female	381 (48%)	50 (53%)	431 (48%)
Age (years)			
Mean	22·4 (1·7)	23·8 (13·0)	22·5 (11·9)
Median	20 (14–30)	22 (14–34)	20 (14–30)
Region of residence*			
Kimberley	402 (50%)	44 (46%)	446 (49·9%)
Pilbara	209 (26%)	19 (20%)	228 (25·5%)
Goldfields	115 (14%)	8 (8%)	123 (13·8%)
Midwest	40 (5%)	12 (13%)	52 (5·8%)
Wheatbelt	8 (1%)	1 (1%)	9 (1·0%)
Perth	24 (3%)	11 (12%)	35 (3·9%)
Men with orchitis	30/417 (7%)	3/45 (7%)	33/462 (7%)
Admitted to hospital	34 (4%)	6 (6%)	40 (4%)

Data are n (%), mean (SD) or median (IQR).*People with mumps onset in boarding schools were listed by their school address rather than by their permanent home address in a rural or remote region.

Table 1: **Demographic characteristics of individuals who acquired mumps in Western Australia between March, 2015, and December, 2016**

After the first cluster of cases in small residential communities in the eastern part of the Kimberley in March, 2015, the disease spread across the Kimberley to other remote communities and towns (figure 1). A case with recent epidemiological links to the Kimberley was identified in May, 2015, at a boarding school in the Goldfields region, and in June, 2015, at a boarding school in Perth. In July, 2015, cases were notified in the Pilbara after a football match involving teams from the Kimberley and Pilbara. The Pilbara region then contributed 150 cases between July 14 and Dec 2, 2015, overlapping with further transmission in the Goldfields region commencing in September, 2015, followed by cases in the Midwest starting in October, 2015.

A second, larger epidemic wave of mumps began in late December, 2015. Between Jan 1, 2016, and March 31, 2016, there was a dramatic resurgence of 227 cases in the Kimberley alone (figure 2), this time including towns from the eastern part of the region that had been relatively unaffected in 2015. The number of notified cases was declining in the Pilbara, with 17 in January, 12 in February, 16 in March, and five in April, and in the Goldfields and Midwest the number of notified cases remained continuously low. By September, 2016, the overall number of notified cases had declined substantially to 13, although there was an extended tail with onset of the last attributed outbreak case on Dec 30, 2016 (figure 2).

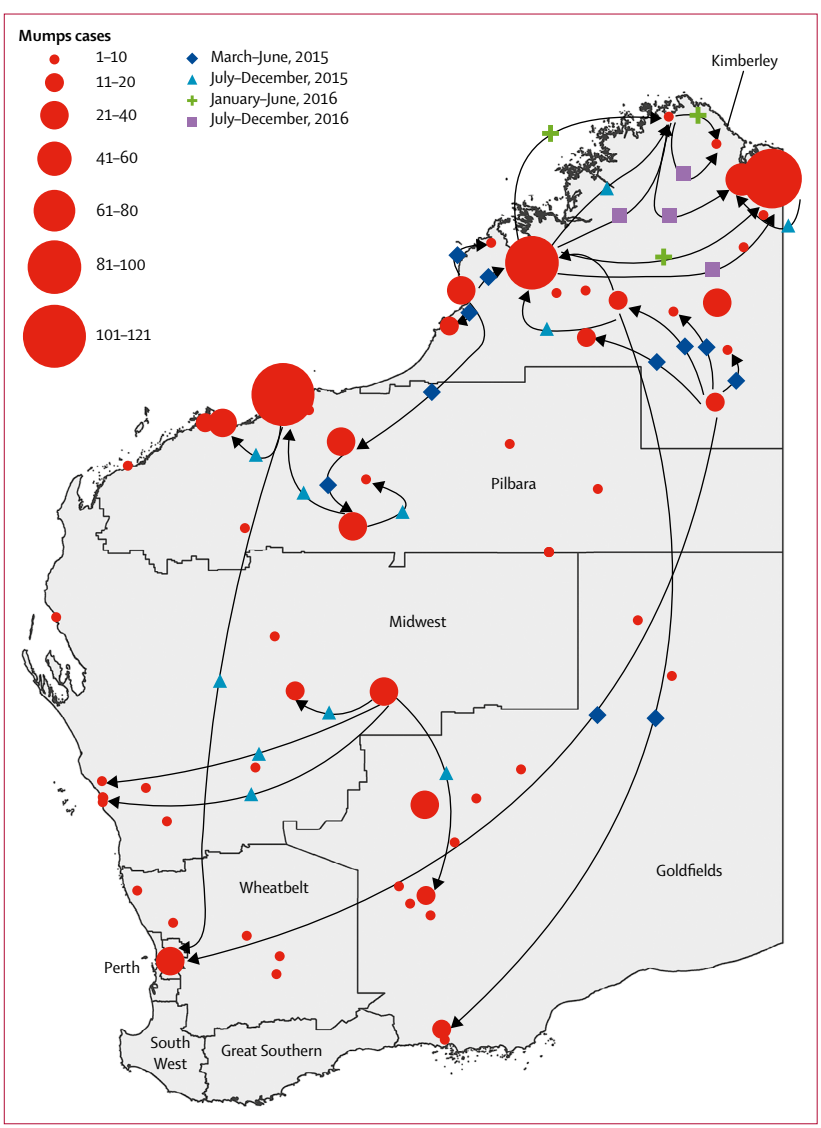

Figure 1: **Relative magnitude of mumps case clusters in Western Australia between March, 2015, and December, 2016, along with identified directions of disease spread**

In the four regions with substantial transmission, age-standardised attack rates in Aboriginal people younger than 40 years were 37 times those of non-Aboriginal people (table 2). Attack rates in Aboriginal people were highest in the Kimberley (27·9 [95% CI 25·2–30·8] cases per 1000), the Pilbara (25·9 [22·5–29·8]), and the Goldfields (18·9 [15·6–22·9]) regions. Mumps attack rates in Aboriginal people were highest in the 15–19 years age group, plateaued for those in the 20–34 years age group, and were lowest in people aged 50 years and older, with only 25 (3%) of 893 outbreak cases in this age group (figure 3).

The vaccination status of Aboriginal and non-Aboriginal people who acquired mumps was similar enough that we only reported vaccination status of all outbreak cases (table 3). For those aged 1–19 years who were most likely to have a record in the Australian Childhood Immunisation Register, 371 (89%) of 419 were fully vaccinated and 29 (7%) were partly vaccinated. The proportion of people with unknown vaccination status increased with age (table 3).

485 (54%) of 891 individuals who acquired mumps had received two documented doses of MMR vaccine. 69 (8%) had received more than two documented doses of MMR vaccine.

To measure the distribution of all mumps outbreak cases by years since the second MMR dose, ten people who received an MMR dose within 2 weeks of mumps onset and 44 people who received their first MMR dose before 12 months of age were excluded from the analysis, leaving a total of 431 people. The proportion of mumps cases increased with time since the second MMR vaccine dose, peaking at 16–18 years, although once 7–9 years had elapsed the proportion of cases remained relatively steady (figure 4). At 19–21 years since the second dose there was a sharp decline in the proportion of mumps cases, with only 21 (5%) of 431 people having been fully vaccinated more than 18 years before acquiring the disease (figure 4).

744 (83%) of 893 mumps cases were laboratory confirmed. 668 (90%) of these 744 were laboratory confirmed by RT-PCR, 70 (9%) by IgG and IgM, and six (1%) by IgM alone. Of the 240 people who tested positive for RT-PCR and had also been tested for mumps-specific IgG and IgM, 165 (69%) were positive for IgG but

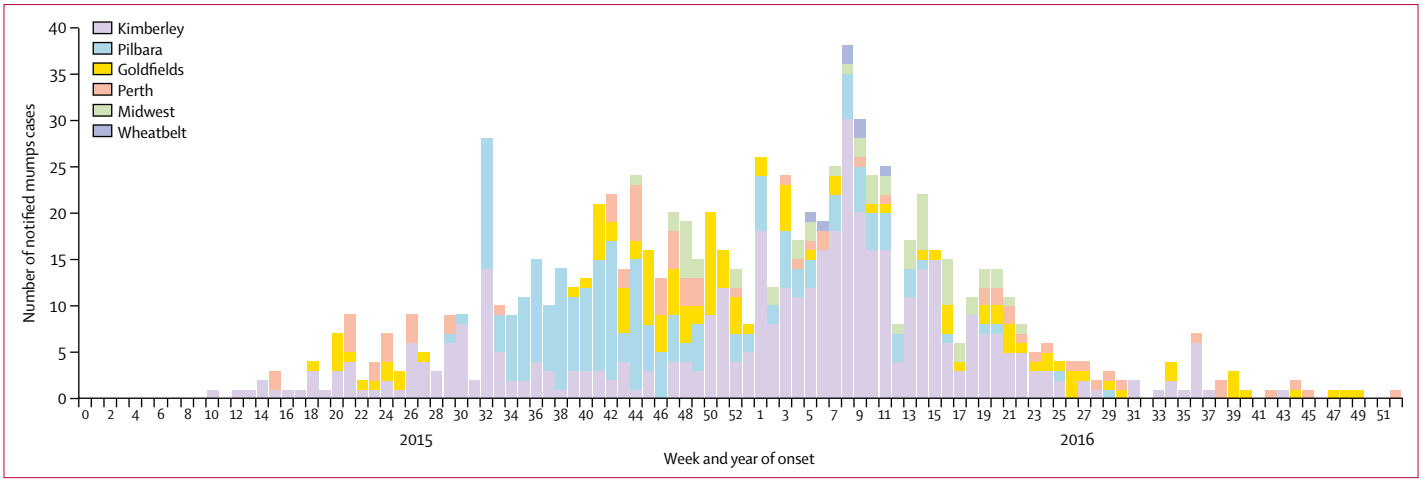

Figure 2: **Epidemic curve of 893 mumps outbreak cases in Western Australia by region affected, from March, 2015, to December, 2016**

negative for IgM, 72 (30%) were positive both for IgG and IgM, two (1%) were negative for IgG but positive for IgM, and one (<1%) was negative both for IgG and IgM.

Genotyping was attempted for 217 (32%) of 668 RT-PCR positive cases and was successful for 133 (61%) of those 217 cases. All successfully genotyped samples were genotype G. 11 (8·3%) of 133 genotyped samples showed sporadic nucleotide polymorphisms within the 400–500 base pair region sequenced. A lineage with a synonymous *SH* gene mutation *G261T* emerged in the west Kimberley town of Broome in June, 2015, and then spread through the west Kimberley and Pilbara regions but not to the east Kimberley, where the prototype outbreak genotype G sequence continued to circulate. *G261T* became the dominant lineage, accounting for 110 (83%) of the 133 outbreak viruses genotyped.

	Aboriginal people		Non-Aboriginal people		Rate ratio
	Cases	Attack rates (per 1000 population)	Cases	Attack rates (per 1000 population)	
Kimberley	365	27·9 (25·2–30·8)	39	2·8 (2·0–3·8)	10·2 (7·3–14·6)
Pilbara	190	25·9 (22·5–29·8)	15	0·4 (0·3–0·7)	63·6 (37·6–116·0)
Goldfields	100	18·9 (15·6–22·9)	5	0·02 (0·07–0·4)	118·6 (49·2–373·4)
Midwest	38	6·4 (4·7–8·8)	11	0·4 (0·2–0·7)	15·7 (7·9–34·1)
Total	693	23·7 (22·1–25·6)	70	0·6 (0·5–0·8)	36·8 (28·8–47·8)

Data in parentheses are 95% CI.

Table 2: **Age-standardised mumps attack rates in the four regions with significant mumps transmission for people aged 1–39 years (n=763)**

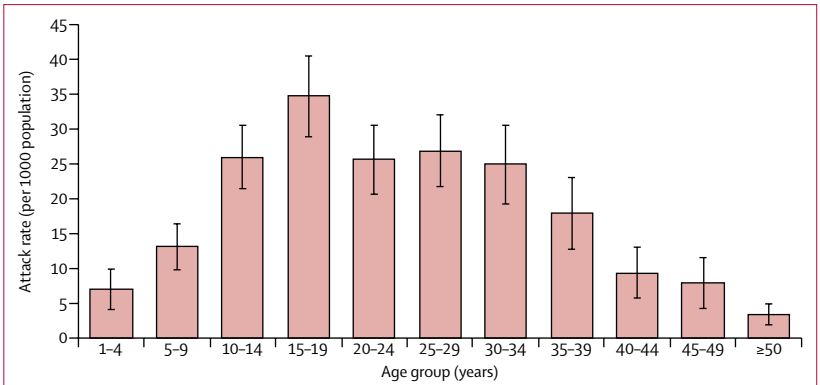

Figure 3: **Mumps attack rates by age group for Aboriginal people in the Kimberley, Pilbara, and Goldfields regions of Western Australia between March, 2015, and December, 2016**
Error bars indicate 95% confidence interval.

Discussion

This mumps outbreak was the largest one in Australia since national mumps notification began in 1995. Most outbreak cases were seen in highly vaccinated Aboriginal people younger than 35 years, corresponding with age cohorts born when childhood mumps vaccination began in Australia in 1981. The outbreak affected people in many remote communities, towns, and several boarding schools, and spread across more than 2 million km² of the sparsely populated northern regions of Western Australia. Many of the smaller affected communities in remote areas were only accessible by four-wheel drive vehicle or light aircraft, which made outbreak control activities challenging.

Mumps outbreaks among highly vaccinated populations have been reported with increasing frequency over the past decade in Europe,[11] the USA,[12–14] Canada,[15] and Western Australia.[6] The suggested causes for these outbreaks include social conditions favouring intense exposure,[12,16] and waning of vaccine-induced immunity.[2,13,17]

Immunity is more likely to wane when there is no natural exposure to disease.[1] Before universal childhood vaccination, mumps immunity was boosted by re-exposure to wild-type mumps infections.[18] Waning immunity is likely to be a major factor responsible for the sustained transmission observed in highly vaccinated populations. None of the 2007–08 genotype J outbreak cases in the Kimberley were also cases in this genotype G outbreak, suggesting greater cross-protection might be

	Fully vaccinated	One dose	Not vaccinated	Unknown
1–4 (n=21)	18 (86%)	3 (14%)	0	0
5–9 (n=76)	70 (92%)	3 (4%)	0	3 (4%)
10–14 (n=152)	139 (91%)	11 (7%)	1 (1%)	1 (1%)
15–19 (n=170)	144 (85%)	12 (7%)	3 (2%)	11 (6%)
20–24 (n=123)	81 (66%)	27 (22%)	2 (2%)	13 (11%)
25–29 (n=114)	53 (46%)	25 (22%)	1 (1%)	35 (31%)
30–34 (n=92)	42 (46%)	18 (20%)	1 (1%)	31 (34%)
35–39 (n=58)	4 (7%)	13 (22%)	2 (3%)	39 (67%)
≥40 (n=85)	3 (4%)	9 (11%)	10 (12%)	63 (74%)
Total (n=891)	554 (62%)	121 (14%)	20 (2%)	196 (22%)

The vaccine provided was the measles, mumps, and rubella vaccine. The fully vaccinated group includes 69 individuals who received more than two recorded doses of mumps vaccine. Two children younger than 1 year were excluded from these analyses because they were not eligible for vaccination. Data are n (%).

Table 3: **Vaccination status by age group (years) of individuals who acquired mumps in Western Australia between March, 2015, and December, 2016**

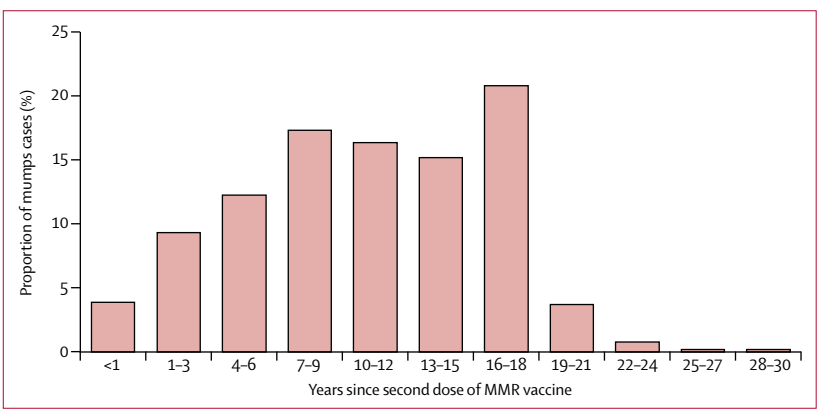

Figure 4: **Distribution of 431 mumps outbreak cases by years since second dose of the MMR vaccine**
Ten people who received an MMR dose within 2 weeks of mumps onset and 44 people who received their first MMR dose before 12 months of age were excluded from the analysis, leaving a total of 431 people. MMR=measles, mumps, and rubella.

conferred by natural infection with a heterologous strain than by the current Jeryl Lynn strain mumps vaccine.

The increase in mumps cases with increasing time since completion of the second dose of the mumps-containing vaccine supports the importance of waning immunity. There might have been fewer people who received their second vaccine dose more than 21 years ago because people born around the time of the mumps vaccine introduction in 1981 were more likely to have been exposed to endemic mumps virus during childhood. The proportion could have also been lower because the timing of the second MMR vaccine dose in Australia was moved from 12 years of age to 4 years of age in 1998. People born from 1994 onwards will have received the second dose of MMR vaccine at age 4–5 years (3–4 years after the first dose), but those born before this would have progressively longer gaps of 5–11 years between doses. Those individuals who in 2015–16 had received their second dose 19 or more years ago belong to the age cohort that received the second dose 11 years after the first. Increasing time between the first and second doses has previously been hypothesised to provide better protection against mumps.[19,20] Richard and colleagues[21] reported increased vaccine effectiveness against mumps during an outbreak if the second dose was given 4–8 years after the first dose. Australia has now moved to a schedule in which the MMR vaccine is delivered at 12 and 18 months after birth, which, considering our findings, could increase the population at risk during a mumps outbreak.

Other mumps outbreaks that disproportionately affected certain ethnic subgroups have also been reported. In 2009–10, 97% of people who acquired mumps in an outbreak in New York City (NY, USA) were Orthodox Jews.[12] Barskey and colleagues suggested that intense exposure associated with Orthodox Jewish study practices, and not differential vaccination status, might have contributed to transmission.[12] Similarly, during a 2009–10 mumps outbreak in Guam, attack rates were substantially higher in Chuukese and Pohnpeian residents than in other Guam residents;[22] these two subpopulations had the highest household crowding indices and this explained the difference in attack rates, rather than differential vaccination coverage.[22] A large mumps outbreak in northwest Arkansas, USA, in 2016 predominantly affected Marshallese residents (57%);[23] dense living environments of up to 20 people in a three-bedroom home probably contributed to the sustained spread in this community. Aboriginal Australians also commonly reside in crowded living environments in which hygiene may be poor because of inadequate and poorly maintained health hardware. On average, two to eight people from Aboriginal communities will share one bedroom,[24,25] which probably played a role in the sustained transmission in the 2015–16 and 2007–08 mumps outbreaks.[6]

Many Aboriginal Australians are also highly mobile and social, consistent with the impact of colonisation and their historically nomadic culture.[26] Aboriginal Australians often move between remote communities and town centres to access education and essential services, for continuity of land practices, to preserve important familial relationships, and for sport and cultural or ceremonial practices.[27] Mobility within and between communities is likely to have contributed substantially to mumps transmission during the 2015–16 outbreak, which spread rapidly over large distances to discrete communities and towns, despite very low overall population density.

Immune escape is another plausible contributing factor for disease spread, because of the mismatch between the Jeryl Lynn strain vaccine and the genotype G wild-type strain. Rubin and colleagues[28] reported a reduced ability of immunisation to effectively neutralise an outbreak G strain compared with the vaccine strain. This neutralising ability decreased with increasing time since vaccination. Previous serological studies have shown that the Jeryl Lynn vaccine offers some cross-protection against heterologous infection;[17] however, the

strength of this cross-protection, the presence of a threshold effect, and the role of other factors such as waning immunity and increased infection risk is unknown. Although long-term protection of the Jeryl Lynn vaccine against heterologous infection might decrease with time, there is evidence of short-term protection for outbreak control.[2,29]

Prevention and control strategies for this outbreak were implemented to reduce the number of people susceptible to mumps because of waning immunity or under-vaccination. Booster vaccinations—usually with a third dose of MMR vaccine—were offered to all members of households and other close contacts (eg, classmates or sports team-mates), and if feasible a ring vaccination intervention provided booster vaccinations to all age-eligible people in boarding schools and other defined community settings where one or more cases had occurred. The third dose of MMR vaccine prevented further mumps transmission in defined communities when implemented with high coverage over a short time (unpublished), but this progressive strategy was not sufficient to control spread across regions and to larger population centres.

At the time of the 2015–16 mumps outbreak, children and teenagers younger than 20 years were most likely to have complete vaccination records. Some people were not able to provide any evidence of earlier vaccination and attempts to locate these records elsewhere were unsuccessful. These issues most likely resulted in an under-ascertainment of the proportion of people aged 20–34 years who were, in fact, partly or fully vaccinated.

We may have overestimated our population at risk when calculating attack rates at the regional level, given that cases were not reported in some communities. However, based on otherwise widespread transmission in the Kimberley, Pilbara, and Goldfields Aboriginal populations, we made the assumption that exposure risk was relatively uniform across the high incidence regions. Many mumps infections were also likely to be subclinical or undiagnosed, such that real attack rates were probably higher. Before widespread vaccination as many as one third of cases were subclinical;[1] however, in vaccinated populations the proportion of undiagnosed mumps infections is difficult to determine.[30]

It is unclear if other factors (eg, genetic or nutritional) that affect immunity may also have contributed to the increased susceptibility to mumps of Aboriginal people in the 2007–08 and 2015–16 Western Australia outbreaks. Future immunological research should focus on testing the avidity of antibodies to mumps virus, including in Aboriginal and non-Aboriginal Australians.[17] Specifically, research should explore why mumps outbreaks appear to affect some populations disproportionately, including whether this observation simply reflects exposure intensity on a background of waning immunity, or whether this is the result of other factors affecting the immunogenicity of the vaccine in these groups. Although neutralising antibody titres are a correlate of protection, the surrogate immunological marker for mumps has not yet been established.[1,31]

In conclusion, a number of factors might have contributed to the size and duration of this large genotype G mumps outbreak that primarily affected highly vaccinated Aboriginal Australians, including waning immunity, immune escape, large household size with suboptimal hygiene, and high levels of mobility and social interaction. Interventions to prevent such outbreaks need to consider the practicalities of public health messaging, including the difficulties of case isolation in crowded home environments. Progressive MMR booster ring vaccination in affected communities was unable to contain the spread of this outbreak. More widespread, region-wide, and pre-emptive use of a third dose of MMR might be required to control similar mumps outbreaks, so the optimal age for routine administration of a third dose of MMR vaccine should be considered.[13] Future research should assess the benefit of increasing the interval between the first and second dose of MMR as a way to strengthen the durability of vaccine protection.

Contributors

DWW, AE, AL, JD, CH, MG, HL, and GKD were involved in the outbreak investigation and response. DWW, AE, AL, JD, and GKD were involved in the collection and cleaning of the data. AL oversaw the laboratory analyses. All authors contributed to the conception and design of the study. DWW did the statistical analyses and wrote the first draft of the manuscript. DWW, AL, and GKD analysed the data and revised the manuscript. All authors reviewed, provided constructive feedback, and approved the final version of the manuscript.

Declaration of interests

We declare no competing interests.

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