



Epidemiology and control of the 1997 measles epidemic in Auckland

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*Presented at the 10th Colloquium of the Spatial Information Research Centre,
University of Otago, New Zealand, 16-19 November, 1998*

Abstract

There was an epidemic of measles in New Zealand in 1997. The majority (62%) of cases occurred in the Auckland region, where there were 1225 notified cases, 175 hospitalisations, but no deaths. Rates of disease were highest in children 6 months to 4 years old. The rates in Pacific Islands people (230 per 100 000) were over three times those in Maori (67 per 100 000) and four times those in Europeans (53 per 100 000). The epidemic commenced in April, became established in May, and peaked in July. A geographic information system was used to map cases and to target vaccination efforts. Early control measures included ring vaccination, lowering the age for the first dose of measles, mumps and rubella vaccine (MMR) to 12 months, and advising that all children should have had at least one dose of MMR. At the end of May, a mass immunisation campaign, to give all children 2-10 years old an early second dose of MMR, commenced. The age of the first dose was lowered again, to 6 months. The mass campaign achieved an estimated 56% coverage among 6-10 year old children. An estimated 57% of children 6-11 months were vaccinated. The epidemic began to decline in August. Areas where cases persisted were identified and targeted for further vaccination efforts. It is likely a large number of cases, but fewer hospitalisations, were prevented. The relatively low immunisation coverage achieved, at least for children aged 6-10 years, indicates ongoing vaccination of this cohort will be required.

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Introduction

A measles epidemic was predicted to occur in New Zealand in 1997 or 1998.^{1,2} The previous most recent epidemic occurred in 1991, and resulted in an estimated 30-60 000 cases, 629 hospitalisations, and 7 deaths. Based on mathematical modelling, as many as 70 000 cases were predicted to occur in an epidemic in 1997 or 1998.³ An immunisation coverage survey conducted in 1996 in the Auckland and Northland regions demonstrated coverage of 80.2% for the first dose of measles, mumps and rubella vaccine (MMR),⁴ which is insufficient to prevent measles epidemics. Low coverage rates reported for Maori and Pacific Islands children (63.7% and 74.6%, respectively) were particularly concerning.

Mass immunisation campaigns have successfully averted predicted epidemics in other countries.^{5,6,7} A mass immunisation campaign was planned to prevent the predicted New Zealand epidemic. The rationale was that a mass campaign would reduce the number of susceptible children by increasing seroconversion rates among children who had not responded to their first dose and by providing an opportunity to vaccinate children who had never received MMR. The early start of the epidemic in April 1997 necessitated a change in strategy to one of epidemic control. This paper describes the epidemic in the Auckland region and the range of control strategies employed to minimise its impact, with a particular focus on the use of a geographic information system (GIS).

Methods

Measles surveillance: Since June 1996, medical practitioners have been legally required to notify all measles cases to their local public health office. In preparation for the expected epidemic, Auckland Public Health (APH) took several steps to enhance surveillance. These steps included encouraging doctors to confirm cases serologically, prompting doctors to notify laboratory-confirmed cases that had not been notified, and requesting weekly updates of hospital discharges of measles cases. Notification data were entered into a regional database for analysis.

Measles cases were defined as confirmed or probable according to the current national case definitions.⁸ Probable cases had fever $\approx 38^{\circ}\text{C}$, generalised maculopapular rash for ≈ 3 days, and either cough, coryza, conjunctivitis or Koplik's spots. Confirmed cases had these clinical symptoms and either laboratory confirmation or epidemiological linkage to a laboratory-confirmed case. Laboratory confirmation is defined as either isolation of measles virus, demonstration of measles-specific IgM antibody, or a significant rise in measles antibody titre.

In practice, it was often not possible to confirm that the rash was present for ≈ 3 days, as doctors were encouraged to notify as early as possible. It was also not possible to collect clinical information on some laboratory-reported cases. Some uncertainty remains regarding the status of cases meeting clinical criteria but for whom serology was negative. Results of tests done within the first few days after the onset of symptoms are especially uncertain, because IgM may not be detectable at that stage.⁹ These cases were generally retained as probable cases unless >4 days had elapsed between the onset of rash and the collection of the blood specimen.

GISEPI: A regional GIS was established to link topographic and statistical data with disease notification data (GISEPI). Data incorporated into GISEPI included statistical boundaries and populations, roads, street address ranges, school and early childhood centre locations, and general practice (GP) locations. GISEPI was used to assign reported cases a street

location and to create maps of disease incidence.

Disease control measures: Communications and policy documents were reviewed to establish the relationship between control measures and epidemic phase.

MMR immunisation campaign coverage: Coverage for school-aged children was estimated from information recorded on the forms used to obtain parental consent for children to be vaccinated at school. Around 86% of the forms were returned. Children were classified as 'likely to be vaccinated' if their parents gave consent, or if the reason for non-consent was either that the child was already fully vaccinated (ie, two doses of MMR or measles vaccine after 12 months of age) or that the child would be vaccinated through a GP. Coverage estimates were calculated only for school children aged 6-10 years, as some 5 year old children would have been vaccinated by GPs prior to the start of the school-based programme. The 1996 census 5-9 year old population was used as a denominator for coverage estimates for these 6-10 year olds.

The number of vaccines delivered by GPs was collected using immunisation benefit claim data. These data do not generally distinguish first from second doses, and are only available aggregated for the Auckland and Northland regions. Therefore, a coverage estimate was only made for children 6-11 months old, as vaccinations in this age group would all have been for a first dose and this age group was not vaccinated in Northland. Consequently, coverage was not estimated for 1-4 year old children.

The number of vaccines delivered by targeted programme providers was derived from direct reports to the regional health authority.

Results

Descriptive epidemiology of the measles epidemic: Between July 1996 and March 1997 there were no confirmed cases of measles notified in the Auckland region. The first case was notified on 30 March 1997. By 31 December 1997, there were 1225 notified cases

(crude annual incidence rate of 113 per 100 000 population), 797 (65%) of which were laboratory confirmed. Confirmation rates increased with age, from 46% for cases under 6 months to 98% for cases 25-29 years.

Sixty-one percent (485) of the laboratory-confirmed cases were notified, without prompting by APH. The remaining 39% (312) were notified after prompting by APH. However, in the early phase of the epidemic, APH prompted doctors the same day that the laboratory result was reported. It is likely that a number of these cases would have been notified without this contact. Thus, the true notification rate of laboratory-confirmed cases was probably greater than 61%. It is difficult to estimate the degree of under-notification of cases that were not laboratory confirmed, but it was likely to be greater than 40%.

The epidemic peaked in the first week of July, with 89 cases notified that week (*Figure 1*). Cases had dramatically fallen by the last week of September, and reached a baseline just above the pre-epidemic level by early December.

The incidence of measles was highest for children aged 6-11 months and 1-4 years (597 and 621 per 100 000, respectively). A second peak in incidence occurred in the 20-24 year age group (103 per 100 000). Gender rates were similar (*Figure 2*). The highest rates of disease, in children under 10 years, occurred in Mangere, Otara, Point England, Glendene, and the Central city (*Figure 3*).

Age-standardised rates were considerably higher for Pacific Islands people than other ethnic groups. The rate for

Figure 1: Measles cases, Auckland, January - December 1997

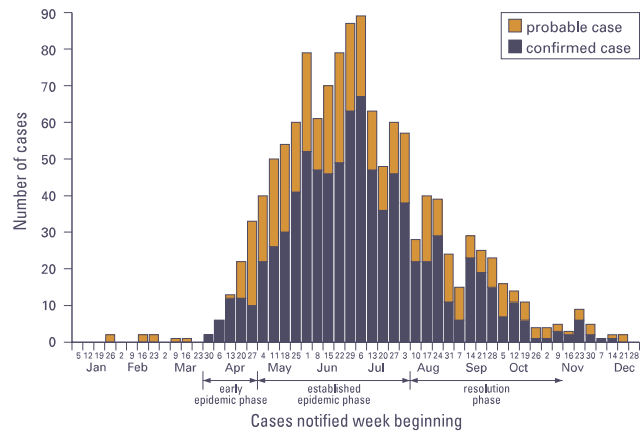


Figure 2: Measles incidence by age and sex, Auckland, 1997

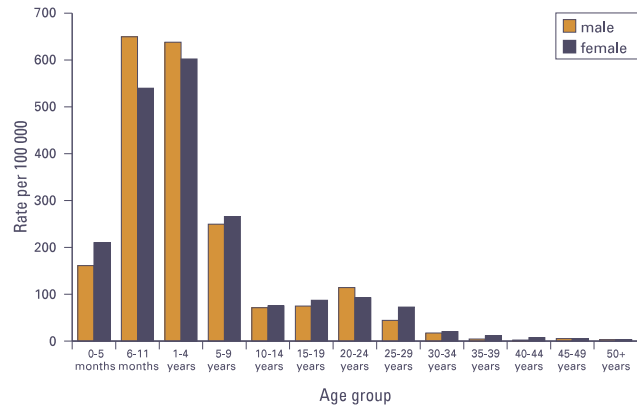


Figure 3. Measles rates for children under 10 years, by census area unit, Auckland 1997.



Pacific Islands people, directly standardised to the 1996 Auckland population, was 230 cases per 100 000. Maori had the next highest rate (67 per 100 000), followed by Europeans (53 per 100 000) and other ethnicities (23 per 100 000).

There were 175 hospitalisations with measles coded as the first diagnosis in Auckland during 1997 (overall hospitalisation rate of 16 per 100 000). Approximately 14% of the notified cases were hospitalised. These hospitalised cases included one possible post-measles encephalitis and 65 cases of post-measles pneumonia. There were 93 admissions for uncomplicated measles and 16 admissions for measles with other complications, such as otitis media. The highest hospitalisation rate was among children under 1 year (334 per 100 000). The second highest rate was for children 1-4 years (82 per 100 000). Rates were also relatively high for 20-24 years olds (43 per 100 000). No deaths were recorded as being due to measles.

Control measures: The epidemic fell into three phases: early epidemic, established epidemic, and resolution (*Figure 1*). The control measures used during each phase of the epidemic are described.

Early epidemic: The period from 30 March to the end of April was classified as early epidemic. This phase was characterised by clustering of cases in Waitakere City and spread into areas on the western boundary of Auckland City. The first cases in Manukau City were notified at the end of April.

It was not possible to proceed immediately with a mass immunisation campaign, due to both insufficient vaccine and the need for time to plan a comprehensive campaign. Ring vaccination was used to limit the spread of the measles virus when cases occurred. Vaccination within 72 hours of exposure may protect contacts from disease. Cases in early childhood centres were followed actively to ensure that all susceptible contacts within the centre were either

vaccinated or excluded (for 14 days). Schools where cases had occurred were unable to identify susceptible children, as immunisation registers had not yet been introduced. Nevertheless, parents of children attending affected schools were advised to vaccinate susceptible children or keep them away for 14 days.

During this early phase of the epidemic, the age for administration of the first dose of MMR was brought forward to 12 months, and advice was given that all children 1-10 years of age should have had a least one dose of MMR.

Established epidemic: By the beginning of May, the epidemic was established, with cases in all seven territorial authority areas of the Auckland region. Initial notification data showed a high rate of disease in children aged 6-11 months. GPs were therefore advised to vaccinate children in this age group, even though a low seroconversion rate at this age would necessitate two further doses after 12 months of age.

During this phase of the epidemic, the mass immunisation campaign was implemented. All children 2-10 years of age were offered an early second dose of MMR (the dose is routinely given at 11 years of age). Children 5-10 years of age were offered vaccination at school while GPs vaccinated children under 5 years of age. Additional, targeted vaccination programmes, using community-based vaccinators, were provided for groups known to have low MMR coverage. The school-based programme commenced late in May and GP vaccination in the first week of June. Disease maps were used to prioritise vaccination in areas where the highest rates of disease were occurring.

Resolution phase: The school-based programme was completed at the end of the school term in mid-July, and the GP-based programme tapered off more gradually over August and September. The completion of the campaign was accompanied by a dramatic fall-off in notifications during August, which marked the beginning of the resolution phase of the epidemic. This phase, like the early phase, was characterised by clustering of cases in particular areas, with notification rates remaining high in areas



of Manukau City, such as Mangere and Otara. GISEPI was used to generate street level maps of recent cases in these areas. These maps were used by mobile vaccinators to more directly target streets with ongoing disease.

Vaccination coverage: An estimated 56% of 6-10 year olds were likely to have been vaccinated (including those who were previously fully vaccinated). There was considerable geographic and ethnic variation in coverage (*Figure 4*). Pacific Islands children had the lowest coverage (40%), with Maori only slightly higher (43%). European children were the most likely to be vaccinated (60%), followed by other ethnicities (53%). However, actual coverage was likely to be higher as some children not returning consent forms would have been vaccinated by their GPs.

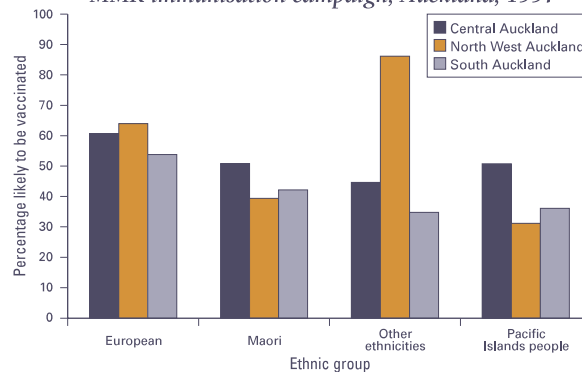
The number of children vaccinated in the targeted programmes in the Auckland and Northland regions was small (3085). These programmes were targeted mainly at pre-school children, so the number of 6-10 year olds included would have been small and unlikely to significantly alter the coverage rates reported above for this age group.

Between June and September, there were 6629 immunisation benefit claims for MMR vaccination of children under 1 year old. The 1996 census under 1 year population was 17 346. Assuming half the population under 1 year were 6-11 months old in June and another third had reached 6 months by the end of September, approximately 11 564 children 6-11 months old would have been eligible for vaccine. Coverage for children 6-11 months was therefore about 57%.

Discussion

Throughout New Zealand, 1964 cases of measles were notified in 1997. The majority (1225, 62%) of these cases occurred in the Auckland region. As measles has only recently become a notifiable disease, this epidemic is the first for which extensive

Figure 4: Coverage among 6-10 year old children during the MMR immunisation campaign, Auckland, 1997^a



^a includes children already fully immunised

data are available for non-hospitalised cases. The relatively high notification rate (61%) for laboratory-confirmed cases in Auckland suggests medical practitioners responded well to the inclusion of measles on the schedule of notifiable disease. A high laboratory confirmation rate suggests medical practitioners also responded well to requests that they confirm the diagnosis, although it could also reflect a higher notification rate for confirmed disease.

It is difficult to determine how effective the control measures were overall. It is possible that the early phase measures delayed the establishment of the epidemic by several weeks. It was considered that any time bought by such measures would be vital in terms of allowing time to implement a mass immunisation campaign.

The effectiveness of the mass immunisation campaign can be measured by comparing the final number of cases notified with the predicted size of the epidemic. Given that about 21 000 cases (30% of 70 000)³ were expected in the Auckland region, it seems likely that a large number of cases (over 90%) were prevented, even allowing for considerable under-notification. However, it is likely that the proportion of hospitalisations prevented was lower. Assuming that the 1997 epidemic would have been comparable in size and case-hospitalisation rate to the 1991 epidemic, between 180 and 270 (30% of 600 to 900)¹ hospitalisations might have been expected in the Auckland region. In fact, 175 hospitalisations



occurred, despite a much lower number of cases overall.

A high apparent hospitalisation rate has been observed previously.¹⁰ Differential under-reporting of non-hospitalised cases may be part of the explanation. The relatively high number of hospitalisations may also be related to the considerably higher rate of disease among Pacific Islands people. This elevated risk cannot be explained by pre-existing lower MMR coverage rates alone, given that Maori had lower coverage than Pacific Islands children in the 1996 survey.⁴ Lower coverage for Pacific Islands children during the campaign may have contributed to the higher rate of disease in these children, but other factors contributing to rapid transmission within this population may also have been important.

Regardless of the ethnic variation in coverage, the relatively low coverage in school-aged children in Auckland indicates there will be an ongoing need to provide an opportunity for a second dose for the current 5 to 10 year old cohort. Measles vaccination of infants aged 6-11 months is considered an effective intervention measure in an outbreak.¹¹ There were high notification and hospitalisation rates for children under 1 year during this epidemic. Coverage of about 57% for children aged 6-11 months was clearly insufficient to prevent a large number of cases. Even if coverage in this age group had been complete, lower seroconversion rates at this age would still have allowed cases to occur. Nevertheless, the aim of vaccination for under 1 year olds was to protect the recipients who did convert, given the higher risk of complications from measles at this age. To some extent this measure may have succeeded considering the relatively low proportion of hospital admissions with complications.

The second peak in age-specific rates in the 20-24 year age group may have also contributed to the high rate of measles in children under 1 year old. If the high rate in the 20-24 age group reflects low levels of protective antibody, women in this age group would be less likely to pass on protective levels of antibody to their infants. The prevalence of protective antibod-

ies in this 20-24 year old cohort is unknown, but, if low, could reflect vaccination policy between 1969 and 1975, when children were routinely vaccinated at 10 months. This cohort would not have received MMR in Form One. However, they would have been more likely to be exposed to wild measles.

GIS contributed to the public health response to this epidemic in two ways. First, it facilitated targeting of intensive vaccination, particularly in the resolution phase of the epidemic which may have accelerated the decline of disease in areas where cases were persisting despite the immunisation campaign. Second, it provided a means of correlating disease incidence data with other factors, such as socio-economic status. The areas identified as having a high incidence of measles also had social and economic conditions, such as a high household occupancy rates, that increase the risk of disease transmission. The demonstration of such relationships highlights that the potential for children to benefit from immunisation is probably greatest in such areas and that these areas should therefore be allocated high priority.

Acknowledgements

The authors would like to acknowledge the contributions of Ruth Pirie, Taina Tupou, Bronwyn Thompson, Helen Mills, and the communicable disease team of Auckland Public Health; Colleen Fakalogotoa, Plunket; Dr Kitty Croxson, Paul Austin, and all the serology team at Auckland Virology Laboratory. GISEPI was built by Uniservices based on a desktop GIS application (ArcView 3.0).¹²

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