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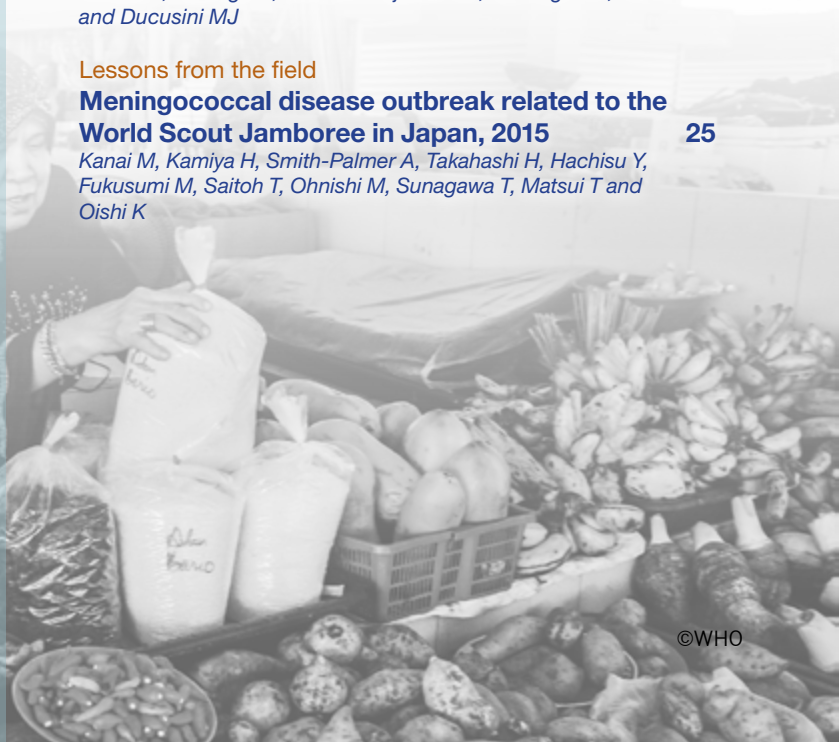
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A *Salmonella* Typhimurium outbreak linked to Vietnamese bread rolls in South Western Sydney, Australia, 2015

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Introduction: In September 2015, the South Western Sydney (SWS) Public Health Unit was notified of a cluster of *Salmonella* Typhimurium (STm) cases with a common multiple-locus variable-number tandem repeats analysis (MLVA) pattern. An investigation was conducted to identify a source and contain the outbreak.

Methods: The cluster was initially identified through routine geographic information system cluster scanning applied to the New South Wales Notifiable Conditions Management System. Additional cases were identified through a complaint to local council about a bakery. The bakery was inspected and 48 environmental and food swabs were collected for analysis.

Results: A total of 26 suspected cases were identified, of which 14 were interviewed. STm MLVA type 3-16-9-11-523 was identified in 19 of 26 case stool specimens. Most cases (12/14) consumed bread rolls containing pork or chicken with chicken liver pâté and raw egg mayonnaise filling. Five cases identified a common bakery exposure. Environmental and food samples from the bakery isolated STm with an identical MLVA pattern.

Discussion: An STm cluster in SWS was investigated and found to be linked to Vietnamese bread rolls containing pork or chicken with chicken liver pâté and raw egg mayonnaise filling. Confirmation of a distinct MLVA pattern among STm isolates from clinical, food and environmental samples provided evidence to establish an epidemiological link between the cases and the implicated premises and informed public health action to contain the outbreak.

Approximately 4.1 million cases of domestically acquired foodborne gastroenteritis occur in Australia annually. *Salmonella* is a frequently implicated organism and is responsible for the majority of hospitalizations and deaths attributable to foodborne infections. *Salmonella* Typhimurium (STm) is the most common serovar in Australia.¹

In New South Wales (NSW) salmonellosis is a notifiable condition under the Public Health Act 2010. Laboratories are required to report positive culture results of *Salmonella* species to NSW Health. In NSW, *Salmonella* isolates are referred to Pathology West – Institute for Clinical Pathology and Medical Research, the state reference laboratory, for further characterization, including serotyping and DNA sequence-based subtyping with multiple-locus variable-number tandem repeats analysis (MLVA).² The data are entered into the NSW Notifiable Conditions Information Management Systems (NCIMS) managed by NSW Health. NSW Health routinely

tracks *Salmonella* using SaTScan v9.4.2, a geographic information system (GIS) software programme, to identify spatiotemporal clusters of STm that have been notified through NCIMS. SaTScan can detect spatial patterns and disease clusters before obtaining MLVA typing results, which can take up to two weeks to be completed.

In September 2015, the Communicable Diseases Branch of NSW Health alerted the South Western Sydney Public Health Unit to a geographical cluster of seven cases of STm infection, in residents of South Western Sydney (SWS). The cases symptom onsets appeared to be clustered in time from 2 to 14 September 2015, lived in close proximity to each other and had South-East or East Asian surnames. Six of the seven cases initially tested had a common MLVA pattern (3-16-9-11-523), suggesting an epidemiological link. An investigation was then undertaken to confirm cases, characterize and identify a common source to control the outbreak and prevent future outbreaks.

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METHODS

Epidemiological investigation

To investigate this STm cluster, the following case definitions were developed:

1. A suspected case was defined as a resident of SWS with onset of symptoms (vomiting, diarrhoea, and/or abdominal pain) in the first two weeks of September 2015 with an epidemiological link (similar exposure, similar food product, relative or carer of case) to the cluster.
2. A confirmed case was a suspected case with laboratory-confirmed evidence of STm with MLVA pattern 3-16-9-11-523.

All suspected and confirmed cases were contacted via telephone and interviewed after consent was obtained. Each case was interviewed using a nationally validated standardized *Salmonella* hypothesis-generating questionnaire. Information about clinical presentation and onset date, hospital admission and treatment, and contact and environmental exposures was obtained. Specifically, information on home food purchases, eating outside the home, special diets and open-ended questions on food consumed was obtained for the seven days before symptom onset. A further section detailed a range of specific high-risk foods that may have been consumed during the priority period. Data were entered into Microsoft Excel 2010 for analysis.

Environmental investigation

In response to the complaint made about the bakery, the New South Wales Food Authority (NSWFA) was informed and an inspection of the premises carried out on 25 September 2015. Food and environmental swab samples ($n = 48$) were collected from around the bakery and sent for laboratory testing.

RESULTS

Epidemiological investigation

A total of 26 suspected STm cases were identified in SWS between 1 and 15 of September 2015; 24 had STm isolated from stool specimens and were entered into NCIMS. The remaining two were household contacts of

two cases that had STm isolates who made a complaint to the SWS local council. The household contacts had similar food exposures to the notified cases and developed symptoms during the same period; however, stool specimens were not obtained from them. MLVA patterns were available for the 24 cases from whom STm were isolated. Of these 24 cases, 19 had a common MLVA pattern 3-16-9-11-523, and the other five had other MLVA patterns (Fig. 1).

Twelve of the 19 cases with the MLVA pattern 3-16-9-11-523 were interviewed. In addition, one of the five cases with a different MLVA pattern was interviewed before the MLVA results were available, and one case linked to the cluster but whose specimen was not taken was also interviewed (Fig. 1).

Table 1 describes the demographic and exposure profile of the 14 interviewed cases. The cases ranged in age from 1 to 77 years old with 43% between 1 to 16 years old. There were more females (57%) than males (43%). Of the nine (64%) cases interviewed, seven presented to the hospital emergency department (seven required admission and two were treated in the emergency department). The majority of cases reported eating pork rolls (64%).

Of the 12 confirmed cases interviewed, 11 consumed a Vietnamese roll with mayonnaise and pâté (eight containing pork and three containing chicken) from a bakery, and the other case consumed a beef dish in a restaurant. Among the 12 interviewed, four purchased the roll from a specific bakery within 24 hours before symptom onset, while six cases purchased a Vietnamese roll in the same vicinity/postcode but could not recall the street or bakery name. The remaining two cases did not recall eating a Vietnamese bread roll in the area of interest. No other common food exposure was identified.

Environmental investigation

Findings from the inspection found the bakery sells up to 300 Vietnamese bread rolls containing raw egg mayonnaise/butter and pâté mix daily.

Samples from the chicken liver pâté mix taken from the storage fridge, raw meat (pork), pâté blender and blade and a shoe swab from the food preparation area had identical MLVA patterns to the cluster.

Figure 1. Recruitment of cases

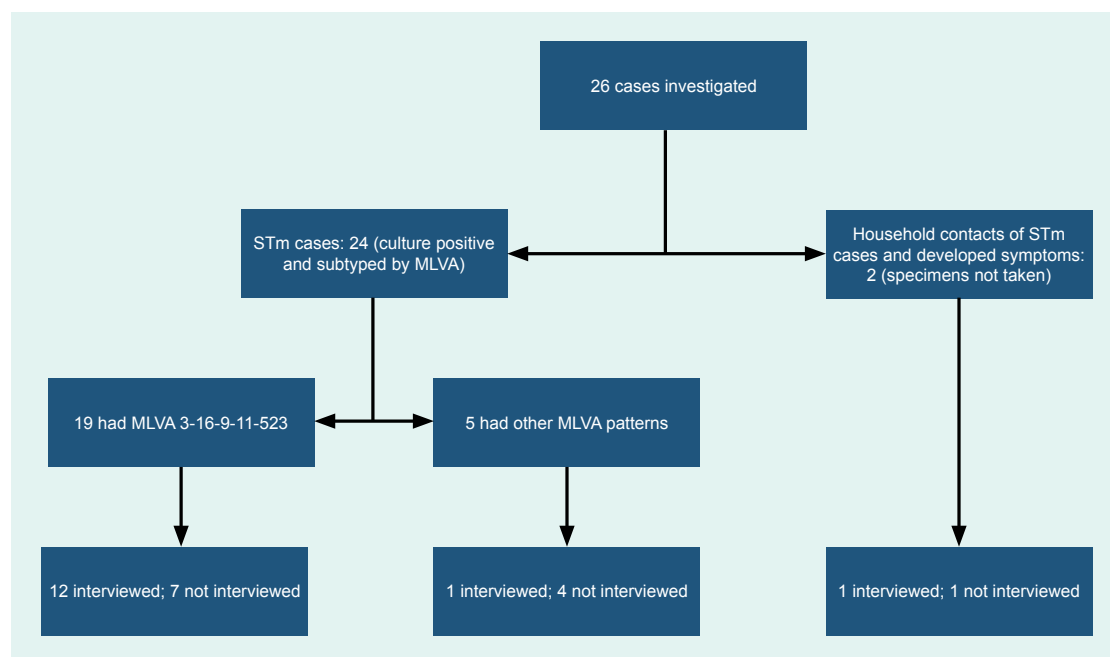


Table 1. Demographic and exposure characteristics for interviewed STm cases, South Western Sydney, Australia, 2015

| Characteristic | n | (%)* |
|------------------------------|---|------|
| Gender | | |
| Female | 8 | 57 |
| Male | 6 | 43 |
| Age group (years) | | |
| 1–16 | 6 | 43 |
| 17–30 | 2 | 14 |
| 31–40 | 3 | 21 |
| 40+ | 3 | 21 |
| Hospital presentation | | |
| Yes | 9 | 64 |
| No | 5 | 36 |
| Food exposure | | |
| Pork roll | 9 | 64 |
| Chicken roll | 3 | 21 |
| No roll | 2 | 14 |

* Percentages are rounded to nearest whole number and may not add up to 100%.

bakery has been banned from using raw egg mayonnaise or chicken liver pâté as they were not able to demonstrate sufficient expertise in the safe preparation and storage of these items.

DISCUSSION

The application of spatiotemporal cluster scanning and MLVA typing enabled the detection of an outbreak of STm in NSW that facilitated a multiagency intervention to prevent further spread of the infection. The outbreak was linked to the consumption of Vietnamese bread rolls (containing pork or chicken with chicken liver pâté and raw egg mayonnaise filling) with an epidemiological and microbiological link to a common source. Molecular typing identified the same MLVA pattern found in several cases in the pâté and pork sampled from the bakery, confirming these items as the likely sources of infection in cases that were linked to the bakery. In previous reports, eggs, pork, chicken and salad rolls have been implicated in large outbreaks.^{1–5}

Control measures

The NSWFA issued a prohibition order on the sale of all bread rolls from the implicated bakery. Follow-up environmental testing was all negative, and the prohibition order was removed once the NSWFA was satisfied that food safety knowledge and practices had improved. The

The detection of STm from foods and surfaces around the implicated bakery suggests substandard food handling and general hygiene practice. A survey conducted by Food Standards Australia New Zealand in 2007 found that food handling in bakeries was less compliant than in other types of businesses.⁶ Other factors contributing to *Salmonella* outbreaks include inadequate storage and

refrigeration, the use of expired eggs, mixing of old and new batches of food items and poor general cleaning practices.⁴

Foodborne disease costs Australia 1.2 billion Australian dollars (Aus\$) each year, largely due to hospital presentations, losses in productivity from days off work or caring for affected family members.^{1,3} In 2000, the Australian Government established OzFoodNet as a joint initiative with Australia's state and territory health authorities to improve national surveillance of foodborne outbreaks, identify ways to minimize foodborne illness and to further understand the causes of foodborne illnesses. In 2008, a study conducted by OzFoodNet found that the costs averted from successful outbreak investigations was between Aus\$ 85 000 and Aus\$ 1.3 million due to early identification and removal of contaminated food from the food supply chain.¹ Early identification and removal of these foods in this outbreak was critical in minimizing the costs associated with further cases.

This study has several limitations. Not all cases with a matching MLVA were interviewed; hence, it is unclear what the source of their infection was. Additionally, due to time constraints and resources, controls were not recruited for the study, which could have further strengthened the evidence against the implicated food items and bakery. However, the distinct MLVA pattern among *Salmonella* isolates from clinical, food and environmental samples provided strong evidence to establish an epidemiological link between the cases and the implicated premises. Lastly, sufficient data were unavailable to examine egg mayonnaise as a potential source.

The application of GIS to routine surveillance enabled the detection of geospatial clustering of STm cases with an identical MLVA pattern in NSW in September 2015. In-depth investigation established an epidemiological link between several cases and food and environmental samples taken from an implicated bakery. While it was not possible to link all cases with the same MLVA pattern to the bakery, the evidence enabled local health officials

to carry out enforcement actions that led to the business being banned from preparing their own chicken liver pâté and raw egg mayonnaise, restricting the spread of STm within the community.

Conflicts of interest

None declared.

Funding

None.

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References

1. OzFoodNet Working Group. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet network, 2011. *Commun Dis Intell Q Rep.* 30 Jun 2015;39(2):E236–64. PMID:26234259
2. Sintchenko V, Wang Q, Howard P, Ha CW, Kardamanidis K, Musto J, et al. Improving resolution of public health surveillance for human *Salmonella enterica* serovar Typhimurium infection: 3 years of prospective multiple-locus variable-number tandem-repeat analysis (MLVA). *BMC Infect Dis.* 2012 03 31;12(1):78. PMID:22462487 doi:10.1186/1471-2334-12-78
3. Kirk MD, McKay I, Hall GV, Dalton CB, Stafford R, Unicomb L, et al. Food safety: foodborne disease in Australia: the OzFoodNet experience. *Clin Infect Dis.* 01 Aug 2008;47(3):392–400. PMID:18558879 doi:10.1086/589861
4. Norton S, Huhtinen E, Conaty S, Hope K, Campbell B, Tegel M, et al. A large point-source outbreak of *Salmonella* Typhimurium linked to chicken, pork and salad rolls from a Vietnamese bakery in Sydney. *West Pac Surveill Response.* 21 June 2012;3(2):16–23. PMID:23908908 doi:10.5365/wpsar.2012.3.1.001
5. Maguire HC, Codd AA, Mackay VE, Rowe B, Mitchell E. A large outbreak of human salmonellosis traced to a local pig farm. *Epidemiol Infect.* Apr 1993;110(2):239–46. PMID:8472766 doi:10.1017/S0950268800068151
6. 2007 National Food Handling Survey Final Report. Canberra: Food Standards Australia New Zealand; 2008 (<https://www.foodstandards.gov.au/publications/documents/2007%20National%20Food%20Handling%20Survey%20Main%20report%20FINAL.pdf>).

Annual vaccine-preventable disease report for New South Wales, Australia, 2014

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This report provides an epidemiological description of selected vaccine-preventable diseases in New South Wales (NSW), Australia, for 2014 to inform ongoing disease monitoring and control efforts. A trend of increasing pertussis notifications was observed, beginning midway through 2014 with the highest disease rates in the 5–9 year age group. Measles notifications increased to 67 cases in 2014 from 34 cases in 2013. Measles cases were associated with travel-related importations—predominantly from the Philippines—and secondary transmission increased compared to 2013 involving three main disease clusters. Notifications of invasive meningococcal disease continued to decline across the state with meningococcal B remaining the most common serogroup in NSW. Increasing rates of pertussis notifications from mid-2014 may indicate the beginning of an epidemic, ending the period of low transmission observed in 2013 and the first half of 2014. An increase in measles notifications in 2014, including secondary transmission, indicates the continued need for public health actions including robust follow-up and awareness campaigns.

Australia has a national immunization schedule funded by the Commonwealth Government and administered by the states and territories with recommended vaccines listed in the Australian Immunization Handbook.¹ States and territories are responsible for public health follow-up and maintaining notification databases on conditions with nationally defined case definitions.²

Monitoring vaccine-preventable diseases enables identification of high-priority events that require urgent attention and facilitates public health response. Ongoing monitoring also enables identification of high-risk groups, changes in affected groups over time, public health interventions and informing policy and programmes.

New South Wales (NSW) is divided into 15 local health districts (LHDs), each with 12 public health units (PHUs). PHUs have the responsibility to follow up on events of public health significance, including vaccine-preventable diseases. Medical practitioners, hospital general managers and diagnostic laboratories are required to notify certain conditions under the State's public health legislation.³ These notifications are reviewed by PHU surveillance officers and, if consistent with the case definition, are entered into the NSW Notifiable Conditions Information Management System (NCIMS).

This report describes the notifications for diphtheria, invasive *Haemophilus influenzae* type b disease, measles, mumps, invasive meningococcal disease (IMD), pertussis, invasive pneumococcal disease (IPD), rubella and tetanus in NSW for 2014.

METHODS

Cases were notified if they met the nationally agreed confirmed or probable case definition² and had a condition onset date in 2014. Information on each of these notifications was collected as part of standard PHU case follow-up as described in the NSW and national control guidelines⁴ and entered into NCIMS as per reporting legislative requirements.

Crude annual disease incidence rates were calculated by year of notification and by age group for 2014 using HealthStats NSW⁵ population estimates (modified from Australian Bureau of Statistics data).

Case counts and rates were analysed by age, sex, vaccination status (verified through the Australian Childhood Immunization Register, general practitioners or health care records where possible) and geographic residence where information was available.

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Ethics

This work involved the use of NSW disease notification data and was collected as part of standard public health action; as such, no ethics approval was required.

RESULTS

Table 1 shows the total number of notifications and rate per 100 000 each year since 1991, **Table 2** shows notifications and rates by age group, and **Table 3** shows notifications and rates by LHD.

Diphtheria

No cases of diphtheria were notified in NSW in 2014. However, one case of cutaneous diphtheria, which is not notifiable in NSW, was reported in a male in his 60s. Public health investigation found that the infection was acquired in Indonesia.

Invasive *Haemophilus influenzae* type b disease

Six cases of invasive *Haemophilus influenzae* type b disease were notified in 2014 (0.1 cases per 100 000 population). This rate has been consistent over the past 10 years, indicating a low level of infections.

Three of the six cases were less than 5 years of age; of these, one was fully vaccinated for age, and one was partially vaccinated for age. All other cases occurred in unvaccinated individuals. One of the six cases was female.

Measles

Measles notifications increased from 34 cases in 2013 to 67 cases in 2014. Secondary transmission occurred and was associated with three main disease clusters following the initial importation. Of the 67 cases, 28 were acquired outside Australia and two additional cases outside NSW. Most importations were from the Philippines (12 cases), Viet Nam (seven cases) and Indonesia (five cases). Females comprised 45% of cases in 2014.

Of the 67 cases, 49 occurred in individuals who were either not vaccinated or did not know their vaccination status. Of the other 18 cases, 16 were vaccinated (five were recorded as receiving one dose, five with two doses, six with unknown number of doses) and two had

no information collected on vaccination status. Thirty-four of the cases were genotyped: 21 were genotype B3, 10 genotype D8, two genotype D9 and one genotype G3.

Mumps

Mumps notifications slightly decreased to 79 cases in 2014 compared to 91 in 2013. The highest rates were reported in adolescents and young adults. Females comprised 54% of cases. Mumps cases are not routinely followed up by PHUs in NSW.

Invasive meningococcal disease

Thirty-six cases of IMD were notified in 2014, down from 46 cases in 2013. Two deaths occurred among these cases: one was in the 0–4 year age group (serogroup B) and one was in the over 85 year age group (serogroup Y). The highest rate of disease was in the 0–4 year age group (2.8 cases per 100 000 population; 14 cases) followed by the over 85 year age group (1.9 cases per 100 000 population; three cases). Females comprised 44% of cases in 2014.

All of the cases notified in 2014 had a serogroup identified. Serogroup B was the most common, with 22 cases accounting for 61% of notified IMD in NSW. Serogroup Y and serogroup W135 each accounted for 19.4% of cases (seven cases each).

Pertussis

There was an upward trend in the number of pertussis notifications from mid-2014 (**Fig. 1**), increasing from 171 notifications in January to 525 in December. The increase was primarily among school-age children, with the highest rates in the 5–9 year age group (134.6 cases per 100 000 population) and the 10–14 year age group (115.2 cases per 100 000 population). A total of 3131 cases were notified in 2014, up from 2342 in 2013. One death was notified in an unvaccinated infant. Females comprised 55% of cases in 2014.

A total of 400 cases occurred in children in the 0–4 year age group. A total of 79 cases occurred in infants aged less than 12 months, of which 18 were unvaccinated and one in which the parent could not recall. Of those who were vaccinated, 54 were fully vaccinated and six partially vaccinated, for age.

High crude notification rates were seen in most LHDs.

Table 1. Number and rate per 100 000 of case notifications by year of onset for selected vaccine-preventable diseases, New South Wales, Australia, 1991 to 2014

| Year | <i>Haemophilus influenzae</i> type b (invasive) | | Measles | | Meningococcal disease (invasive) | | Mumps | | Pertussis | | Pneumococcal disease (invasive)* | | Rubella | | Tetanus | |
|------|---|------|---------|------|----------------------------------|------|-------|------|-----------|-------|----------------------------------|------|---------|------|---------|------|
| | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate |
| 2014 | 6 | 0.1 | 67 | 0.9 | 36 | 0.5 | 79 | 1.1 | 3131 | 41.7 | 516 | 6.9 | 10 | 0.1 | 1 | 0.0 |
| 2013 | 9 | 0.1 | 34 | 0.5 | 46 | 0.6 | 91 | 1.2 | 2342 | 31.6 | 472 | 6.4 | 12 | 0.2 | 2 | 0.0 |
| 2012 | 2 | 0.0 | 172 | 2.4 | 65 | 0.9 | 105 | 1.4 | 5843 | 80.0 | 581 | 8.0 | 10 | 0.1 | 1 | 0.0 |
| 2011 | 4 | 0.1 | 90 | 1.3 | 71 | 1.0 | 68 | 0.9 | 13 197 | 182.8 | 530 | 7.3 | 17 | 0.2 | 1 | 0.0 |
| 2010 | 6 | 0.1 | 26 | 0.4 | 73 | 1.0 | 40 | 0.6 | 9344 | 130.8 | 497 | 7.0 | 13 | 0.2 | 1 | 0.0 |
| 2009 | 6 | 0.1 | 19 | 0.3 | 92 | 1.3 | 40 | 0.6 | 12 549 | 177.9 | 476 | 6.8 | 7 | 0.1 | 2 | 0.0 |
| 2008 | 8 | 0.1 | 39 | 0.6 | 80 | 1.2 | 77 | 1.1 | 8754 | 126.1 | 546 | 7.9 | 17 | 0.2 | 1 | 0.0 |
| 2007 | 7 | 0.1 | 4 | 0.1 | 109 | 1.6 | 323 | 4.7 | 2096 | 30.7 | 519 | 7.6 | 8 | 0.1 | 2 | 0.0 |
| 2006 | 11 | 0.2 | 60 | 0.9 | 101 | 1.5 | 155 | 2.3 | 4910 | 72.8 | 562 | 8.3 | 37 | 0.6 | 2 | 0.0 |
| 2005 | 7 | 0.1 | 5 | 0.1 | 137 | 2.1 | 111 | 1.7 | 5796 | 86.6 | 642 | 9.6 | 10 | 0.2 | 1 | 0.0 |
| 2004 | 5 | 0.1 | 12 | 0.2 | 146 | 2.2 | 65 | 1.0 | 3564 | 53.6 | 903 | 13.6 | 17 | 0.3 | 1 | 0.0 |
| 2003 | 6 | 0.1 | 18 | 0.3 | 197 | 3.0 | 36 | 0.5 | 2769 | 41.8 | 801 | 12.1 | 23 | 0.4 | 1 | 0.0 |
| 2002 | 10 | 0.2 | 8 | 0.1 | 212 | 3.2 | 29 | 0.4 | 2014 | 30.6 | 881 | 13.4 | 35 | 0.5 | 0 | 0.0 |
| 2001 | 7 | 0.1 | 31 | 0.5 | 232 | 3.6 | 28 | 0.4 | 4436 | 67.9 | 444 | 6.8 | 58 | 0.9 | 0 | 0.0 |
| 2000 | 8 | 0.1 | 35 | 0.5 | 248 | 3.8 | 91 | 1.4 | 3693 | 56.9 | ID | ID | 191 | 2.9 | 3 | 0.1 |
| 1999 | 13 | 0.2 | 34 | 0.5 | 217 | 3.4 | 33 | 0.5 | 1413 | 22.0 | NN | NN | 45 | 0.7 | 1 | 0.0 |
| 1998 | 11 | 0.2 | 119 | 1.9 | 185 | 2.9 | 38 | 0.6 | 2306 | 36.4 | NN | NN | 78 | 1.2 | 3 | 0.1 |
| 1997 | 17 | 0.3 | 272 | 4.3 | 218 | 3.5 | 30 | 0.5 | 4243 | 67.6 | NN | NN | 153 | 2.4 | 3 | 0.1 |
| 1996 | 13 | 0.2 | 191 | 3.1 | 161 | 2.6 | 27 | 0.4 | 1154 | 18.6 | NN | NN | 631 | 10.2 | 1 | 0.0 |
| 1995 | 29 | 0.5 | 596 | 9.7 | 113 | 1.8 | 14 | 0.2 | 1368 | 22.3 | NN | NN | 2374 | 38.8 | 0 | 0.0 |
| 1994 | 61 | 1.0 | 1483 | 24.5 | 142 | 2.3 | 11 | 0.2 | 1405 | 23.2 | NN | NN | 229 | 3.8 | 4 | 0.1 |
| 1993 | 124 | 2.1 | 2345 | 39.1 | 153 | 2.6 | 13 | 0.2 | 1533 | 25.5 | NN | NN | 1184 | 19.7 | 5 | 0.1 |
| 1992 | 217 | 3.6 | 804 | 13.5 | 121 | 2.0 | 23 | 0.4 | 218 | 3.7 | NN | NN | 323 | 5.4 | 2 | 0.0 |
| 1991 | 212 | 3.6 | 494 | 8.4 | 128 | 2.2 | 8 | 0.1 | 49 | 0.8 | NN | NN | 59 | 1.0 | 5 | 0.1 |

* Invasive pneumococcal disease was not notifiable (NN) until 2000; in 2000 there were incomplete data (ID).

Table 2. Number and rate per 100 000 population of case notifications by age group for selected vaccine-preventable diseases in New South Wales, Australia, 2014

| Age group (years) | <i>Haemophilus influenzae</i> type b (invasive) | | Measles | | Meningococcal disease (invasive) | | Mumps | | Pertussis | | Pneumococcal disease (invasive) | | Rubella | | Tetanus | |
|-------------------|---|------|---------|------|----------------------------------|------|-------|------|-----------|-------|---------------------------------|------|---------|------|---------|------|
| | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate |
| 0–4 | 3 | 0.6 | 12 | 2.4 | 14 | 2.8 | 7 | 1.4 | 400 | 80.8 | 69 | 13.9 | 0 | 0 | 0 | 0 |
| 5–9 | 0 | 0.0 | 4 | 0.9 | 0 | 0 | 8 | 1.7 | 636 | 134.6 | 11 | 2.3 | 1 | 0.2 | 0 | 0 |
| 10–14 | 0 | 0.0 | 6 | 1.3 | 3 | 0.7 | 3 | 0.7 | 518 | 115.2 | 5 | 1.1 | 0 | 0 | 0 | 0 |
| 15–19 | 0 | 0.0 | 15 | 3.2 | 3 | 0.7 | 9 | 1.9 | 122 | 26.3 | 9 | 1.9 | 0 | 0 | 0 | 0 |
| 20–24 | 0 | 0.0 | 11 | 2.2 | 1 | 0.2 | 7 | 1.4 | 86 | 17.0 | 6 | 1.2 | 1 | 0.2 | 0 | 0 |
| 25–29 | 0 | 0.0 | 8 | 1.5 | 0 | 0.0 | 1 | 0.2 | 82 | 15.2 | 12 | 2.2 | 3 | 0.6 | 0 | 0 |
| 30–34 | 1 | 0.2 | 6 | 1.1 | 2 | 0.4 | 10 | 1.8 | 128 | 23.6 | 11 | 2.0 | 0 | 0 | 1 | 0.2 |
| 35–39 | 0 | 0.0 | 4 | 0.8 | 0 | 0.0 | 9 | 1.8 | 128 | 25.6 | 20 | 4.0 | 2 | 0.4 | 0 | 0 |
| 40–44 | 0 | 0.0 | 1 | 0.2 | 1 | 0.2 | 2 | 0.4 | 175 | 33.3 | 18 | 3.4 | 1 | 0.2 | 0 | 0 |
| 45–49 | 0 | 0.0 | 0 | 0 | 0 | 0 | 3 | 0.6 | 145 | 29.9 | 38 | 7.8 | 2 | 0.4 | 0 | 0 |
| 50–54 | 0 | 0.0 | 0 | 0 | 1 | 0.2 | 4 | 0.8 | 125 | 25.0 | 30 | 6.0 | 0 | 0 | 0 | 0 |
| 55–59 | 0 | 0.0 | 0 | 0 | 1 | 0.2 | 4 | 0.9 | 122 | 26.4 | 41 | 8.9 | 0 | 0 | 0 | 0 |
| 60–64 | 1 | 0.2 | 0 | 0 | 1 | 0.2 | 4 | 1.0 | 127 | 31.0 | 46 | 11.2 | 0 | 0 | 0 | 0 |
| 65–69 | 1 | 0.3 | 0 | 0 | 3 | 0.8 | 3 | 0.8 | 103 | 27.9 | 49 | 13.3 | 0 | 0 | 0 | 0 |
| 70–74 | 0 | 0.0 | 0 | 0 | 1 | 0.4 | 2 | 0.7 | 80 | 29.5 | 34 | 12.5 | 0 | 0 | 0 | 0 |
| 75–79 | 0 | 0.0 | 0 | 0 | 2 | 1.0 | 2 | 1.0 | 68 | 33.0 | 31 | 15.0 | 0 | 0 | 0 | 0 |
| 80–84 | 0 | 0.0 | 0 | 0 | 0 | 0 | 1 | 0.7 | 48 | 31.2 | 32 | 20.8 | 0 | 0 | 0 | 0 |
| 85+ | 0 | 0.0 | 0 | 0 | 3 | 1.9 | 0 | 0 | 37 | 23.1 | 54 | 33.8 | 0 | 0 | 0 | 0 |

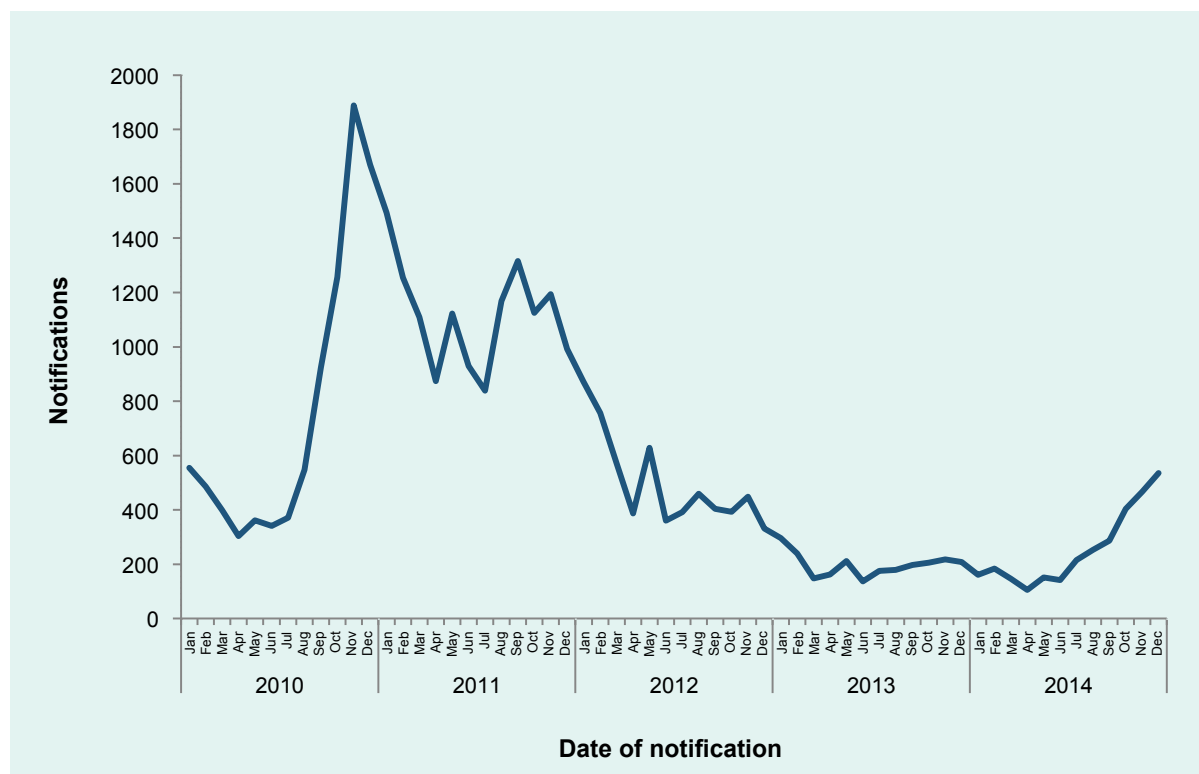
Table 3. Number and rate per 100 000 population of case notifications by local health district# for selected vaccine-preventable diseases in New South Wales, Australia, 2014

| Local Health District | <i>Haemophilus influenzae</i> type b | | Measles | | Meningococcal disease | | Mumps | | Pertussis | | Pneumococcal disease (invasive) | | Rubella | | Tetanus | |
|------------------------|--------------------------------------|------|---------|------|-----------------------|------|-------|------|-----------|------|---------------------------------|------|---------|------|---------|------|
| | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate |
| Central Coast* | 0 | 0 | 11 | 3.3 | 3 | 0.9 | 1 | 0.3 | 24 | 7.3 | 21 | 6.3 | 3 | 0.9 | 0 | 0 |
| Far West* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 23 | 74.6 | 6 | 19.5 | 0 | 0 | 0 | 0 |
| Hunter New England | 1 | 0.1 | 1 | 0.1 | 11 | 1.2 | 3 | 0.3 | 441 | 48.8 | 79 | 8.8 | 0 | 0 | 0 | 0 |
| Illawarra Shoalhaven* | 0 | 0 | 1 | 0.3 | 1 | 0.3 | 10 | 2.5 | 164 | 41.5 | 20 | 5.1 | 0 | 0 | 0 | 0 |
| Mid North Coast* | 1 | 0.5 | 0 | 0 | 0 | 0 | 1 | 0.5 | 27 | 12.7 | 18 | 8.5 | 0 | 0 | 0 | 0 |
| Murrumbidgee* | 0 | 0 | 0 | 0 | 2 | 0.8 | 3 | 1.3 | 233 | 97.5 | 20 | 8.4 | 0 | 0 | 0 | 0 |
| Nepean Blue Mountains* | 1 | 0.3 | 1 | 0.3 | 2 | 0.6 | 4 | 1.1 | 177 | 49.0 | 25 | 6.9 | 0 | 0 | 0 | 0 |
| Northern NSW* | 1 | 0.3 | 2 | 0.7 | 1 | 0.3 | 1 | 0.3 | 61 | 20.7 | 14 | 4.8 | 0 | 0 | 1 | 0.3 |
| Northern Sydney | 0 | 0 | 8 | 0.9 | 3 | 0.3 | 12 | 1.4 | 427 | 48.0 | 44 | 5.0 | 1 | 0.1 | 0 | 0 |
| South Eastern Sydney | 0 | 0 | 12 | 1.4 | 4 | 0.5 | 14 | 1.6 | 530 | 60.1 | 56 | 6.4 | 2 | 0.2 | 0 | 0 |
| South Western Sydney | 0 | 0 | 5 | 0.5 | 4 | 0.4 | 9 | 1.0 | 149 | 16.1 | 69 | 7.5 | 1 | 0.1 | 0 | 0 |
| Southern NSW* | 0 | 0 | 0 | 0 | 1 | 0.5 | 1 | 0.5 | 147 | 72.6 | 14 | 6.9 | 0 | 0 | 0 | 0 |
| Sydney | 0 | 0 | 12 | 2.0 | 0 | 0 | 8 | 1.3 | 144 | 23.5 | 32 | 5.2 | 1 | 0.2 | 0 | 0 |
| Western NSW* | 0 | 0 | 4 | 1.4 | 2 | 0.7 | 1 | 0.4 | 149 | 53.8 | 27 | 9.7 | 0 | 0 | 0 | 0 |
| Western Sydney | 2 | 0.2 | 10 | 1.1 | 1 | 0.1 | 11 | 1.2 | 427 | 47.1 | 66 | 7.3 | 2 | 0.2 | 0 | 0 |

Note: Some (*) LHDs have small populations with fewer than 500 000 people and a small number of cases. As such, the rates per 100 000 can be unstable.

Immunization coverage data by LHD are available in annual immunization coverage reports.⁶

Figure 1. Pertussis notifications by month of onset, NSW, January 2010 to December 2014



Invasive pneumococcal disease

A total of 517 cases of IPD were notified in 2014, up from 471 in 2013. Females comprised 50% of cases in 2014. A total of 53 deaths were identified, three of which were in infants: one aged 1 month (serotype 22F, non-vaccine type), one aged 2 months (serotype 23B, non-vaccine type) and one aged 1 year (serotype 23B, non-vaccine type). Of the remaining deaths, four were in the 33–49 year age group, 14 in the 50–64 year age group and 32 in people aged 65 years or older. Pneumonia was the leading cause of IPD in adults aged 50 years and over (65%) and bacteraemia in children under the age of 5 years (47%). Of the 390 cases that occurred in the age groups that are followed up by PHUs (0–4 year age group or 50 years or over), 16 (4%) were notified in Aboriginal people, among whom case notification rates were higher than in non-Aboriginal people (26.4 and 12.6 per 100 000, respectively, $p=0.010$).

The rate of IPD in children under 5 years of age was 13.9 cases per 100 000 population, up from 12.5 cases per 100 000 in 2013. The proportion of cases under 5 years of age with meningitis was 6% higher than in previous years, up from 10% to 16% ($p=0.03$). Serotype 19A was the leading cause of all IPD in children (26%) followed by 19F (11%), both of which are included in the current 13-valent vaccine. In children under 5 years of age, 51% of disease was caused by non-vaccine serotypes and this rate continues to increase. Vaccination data were available for 100% (69 cases) of notifications under the age of 5 years. A total of 50 cases (73%) were fully vaccinated and 17 cases (24%) were either partially vaccinated or too young to have received their first dose. There were two cases (3%) whose parents chose not to vaccinate. There were 16 cases (26%) of vaccine serotype disease in fully vaccinated children. Serotype 19A accounted for 59% of vaccine failures and serotypes 3 (24%), 19F (12%) and 14 (5%) were responsible for the remainder of cases. The number of vaccine failures in children under 5 years reported in 2014 was higher than previously reported.

Rubella

Ten cases of rubella were notified in 2014, seven of whom were female. Cases ranged from 7 to 48 years of age. No cases of congenital rubella were notified in 2014.

Tetanus

One case of tetanus was notified in 2014 in an adult who had not been vaccinated.

DISCUSSION

The majority of notifiable vaccine-preventable diseases remain well controlled in NSW with case counts and rates well below historical levels. However, both measles and pertussis remain a persistent public health challenge. High immunization rates and rapid public health response are required to maintain measles elimination and control outbreaks of pertussis.

The last notified case of respiratory diphtheria in NSW occurred in 1991. Cutaneous diphtheria does not meet the NSW or national case definition (clinical evidence of pharyngitis/laryngitis or toxic symptoms), but due to the risk of transmission to the respiratory tract, public health follow-up is warranted. The inclusion of cutaneous diphtheria as a notifiable disease in future national surveillance is under consideration.

Measles cases that were acquired outside Australia in 2014 were predominantly imported from the Philippines. The Philippines experienced a measles outbreak associated with increased measles circulation in the Western Pacific Region⁶ that was exacerbated by the disruption associated with Typhoon Haiyan. The most common genotype observed in NSW was B3, which is the predominant genotype in the Philippines.

Children receive their first dose of measles-containing vaccine at 12 months of age per the National Immunization Program schedule.¹ In 2014, there were five cases of measles in children under 12 months, of which three acquired disease outside of Australia in measles-endemic areas (the Philippines and Indonesia). Although these children were too young to be vaccinated under the national schedule, the Australian Immunization Handbook advises that measles–mumps–rubella vaccine can be given as early as 9 months,¹ which may be appropriate when infants travel to areas that are endemic or are experiencing an outbreak.

IMD cases continue to decrease following the implementation of the national meningococcal C

immunization programme in 2003.⁷ No serogroup C cases were notified in 2014. Serogroup B remains the most frequent cause of IMD in NSW; however, even in the absence of a publicly funded vaccine, notifications have been decreasing (22 cases notified in 2014, and 27 in 2013). Seven cases of IMD were caused by serogroup W135, which is a slight increase over the preceding years. Further study on this serogroup—particularly further genetic characterization⁸—may help elucidate a connection, if any, to the global spread of W135 following the 2000 Haj.⁹

The highest rate of IMD (1.2 notifications per 100 000) was observed in the Hunter New England LHD (11 cases), with an unusually high proportion of serogroup Y (four of the 11 cases; 36.4%). This represents more than half of the seven serogroup Y cases in NSW in 2014. No epidemiological link was identified between the cases.

Pertussis notifications increased markedly from mid-way through 2014, indicating the potential beginning of an epidemic. Despite Australia having a long-established vaccination programme for pertussis, periodic epidemics do occur.¹⁰ Epidemics of pertussis occurred in 2008–09 and 2010–11 and generally occur every three to four years. Previous pertussis epidemics have been shown to be associated with increased infant hospitalizations and increased morbidity and mortality.¹⁰ This is reflected in the 2014 data, with a high proportion of cases in those aged 14 years or less. Notifications will continue to be monitored.

The rate of IPD increased in 2014 across most age groups (except people aged 65–84 years). For the first time since 13-valent pneumococcal conjugate vaccine (PCV-13) was introduced in 2011, the rate of IPD in children under 5 years increased, to 13.9 per 100 000 (up from 12.6 per 100 000 in 2013), although this is still lower than the rate of IPD when PCV-13 was introduced in 2011 (19.0 per 100 000). The proportion of IPD due to non-vaccine serotypes has increased by 29% since PCV-13 introduction. The increase in IPD incidence in children under 5 years is concerning; in addition, 11 cases (16%) were diagnosed with meningitis—a significant increase in life threatening illness. This is the highest percentage of meningitis reported in children (average 6% per year) since pneumococcal surveillance began in 1990. Higher rates of IPD in Aboriginal populations were observed despite high vaccination coverage.

Data from NSW disease surveillance systems are subject to the limitations inherent in any disease surveillance programme. The number of notifications reflects health-seeking behaviour and testing practices in NSW. The effect of this limitation will vary by condition. For high-severity diseases such as IMD or measles, it is likely that all cases will be captured in surveillance, but for conditions such as pertussis, notifications will represent only a proportion of the actual cases. In these cases, numbers of notifications will represent trends rather than absolute numbers.

CONCLUSION

The majority of vaccine-preventable diseases remain well controlled in NSW. While the number of measles and IPD notifications increased, crude incidence rates remained low. The exception observed was pertussis, which had increasing numbers of notifications, a phenomenon expected every three to four years as immunity from either vaccination or infection is not long-lasting. Control of pertussis in NSW, as elsewhere, remains a challenge, with waning pertussis immunity following vaccination or infection leading to periodic outbreaks.

Conflicts of interest

None of the authors have conflicts of interest pertinent to this manuscript.

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References

1. The Australian Immunisation Handbook. 10th ed. Canberra: Australian Government Department of Health and Ageing; 2013 (<http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/Handbook10-home>).

2. Australian national notifiable diseases and case definitions. Canberra: Communicable Diseases Network Australia; 2017 (<http://www.health.gov.au/casedefinitions>).
3. Public Health Act 2010. Sydney: New South Wales Government; 2010 (<http://www.legislation.nsw.gov.au/#/view/act/2010/127>).
4. NSW Health Infectious Disease Control Guidelines. Sydney: New South Wales Government; 2016 (<http://www.health.nsw.gov.au/Infectious/controlguideline/Pages/default.aspx>).
5. HealthStats NSW [online database]. Sydney: New South Wales Government; 2015 (<http://www.healthstats.nsw.gov.au>).
6. Measles Case Distribution by Month and WHO Regions. 2008–2015. Geneva: World Health Organization; 2015 (http://www.who.int/immunization/monitoring_surveillance/burden/vpd/surveillance_type/active/big_measlesmonthlyregionaldistribution_PDF.pdf?ua=1, accessed 8 July 2015).
7. Booy R, Jelfs J, El Bashir H, Nissen MD. Impact of meningococcal C conjugate vaccine use in Australia. *Med J Aust.* 2007 Feb 05;186(3):108–9. pmid:17309394
8. Golparian D, Unemo M. Will genome analysis elucidate evolution, global transmission and virulence of *Neisseria meningitidis* lineages? *EBioMedicine.* 2015 02 09;2(3):186–7. pmid:26137558 doi:10.1016/j.ebiom.2015.02.001
9. Abad R, López EL, Debbag R, Vázquez JA. Serogroup W meningococcal disease: global spread and current affect on the Southern Cone in Latin America. *Epidemiol Infect.* 2014 Dec;142(12):2461–70. pmid:24831052 doi:10.1017/S0950268814001149
10. Pillsbury A, Quinn HE, McIntyre PB. Australian vaccine preventable disease epidemiological review series: pertussis, 2006–2012. *Commun Dis Intell Q Rep.* 2014 09 30;38(3):E179–94. pmid:25391404

An outbreak of foodborne norovirus gastroenteritis linked to a restaurant in Melbourne, Australia, 2014

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Introduction: In May 2014 an outbreak of norovirus occurred among patrons of a restaurant in Melbourne, Australia. Investigations were conducted to identify the infectious agent, mode of transmission and source of illness, and to implement controls to prevent further transmission.

Methods: A retrospective case-control study was conducted to test the hypothesis that food served at the restaurant between 9 and 15 May 2014 was the vehicle for infection. A structured questionnaire was used to collect demographic, illness and food exposure data from study participants. To ascertain whether any food handlers had experienced gastroenteritis symptoms and were a possible source of infection, investigators contacted and interviewed staff who had worked at the restaurant between 9 and 16 May 2014.

Results: Forty-six cases (including 16 laboratory-confirmed cases of norovirus) and 49 controls were interviewed and enrolled in the study. Results of the analysis revealed a statistically significant association with illness and consumption of grain salad (OR: 21.6, 95% CI: 1.8–252.7, $p = 0.015$) and beetroot dip (OR: 22.4, 95% CI: 1.9–267.0, $p = 0.014$). An interviewed staff member who reported an onset of acute gastrointestinal illness on 12 May 2014 had prepared salads on the day of onset and the previous two days.

Discussion: The outbreak was likely caused by person-to-food-to-person transmission. The outbreak emphasizes the importance of the exclusion of symptomatic food handlers and strict hand hygiene practices in the food service industry to prevent contamination of ready-to-eat foods and the kitchen environment.

Noroviruses are non-enveloped, single-stranded RNA viruses, recognized as a leading cause of acute gastroenteritis worldwide.¹ There are currently six recognized norovirus genogroups, three of which (GI, GII and GIV) cause human illness.² Norovirus is transmitted via the faecal–oral route primarily through close contact with an infected person, contact with contaminated fomites or consumption of contaminated food or water.³ The average incubation period is between 24 and 48 hours, with symptoms including acute-onset vomiting, diarrhoea, nausea, myalgia and low-grade fever.⁴ Infected individuals shed the virus while symptomatic; however, shedding has been documented before the onset of symptoms, after symptoms have resolved and by asymptomatic infected individuals.^{5–7} Contamination of food by both symptomatic and asymptomatic infected food handlers has been well documented.^{8–14}

Commencing 13 May 2014, the Victorian Department of Health and Human Services, Communicable Disease Prevention and Control Unit received reports of gastrointestinal illness in patrons following a banquet lunch at a Mediterranean-style restaurant on 11 May 2014. In response, an outbreak investigation was initiated with the local council health department to identify the infectious agent, the mode of transmission, the source of illness and to implement controls to prevent further transmission.

METHODS

Epidemiological investigation

A retrospective case-control study was conducted to test the hypothesis that food served at the restaurant was the vehicle for infection. Study participants were recruited

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from the restaurant's booking list and contacted for phone interviews. A structured questionnaire was used to collect demographic, illness and food exposure data from study participants.

A probable case was defined as a person who ate food at the restaurant between 9 and 15 May 2014 and had onset of vomiting and/or diarrhoea or two or more symptoms of fever, nausea, abdominal pain and headache between 24 and 48 hours after consumption. A confirmed case met the probable case definition and also had norovirus detected by polymerase chain reaction (PCR) in a faecal specimen. Controls were patrons identified during the interview process who did not meet the definition of a probable or confirmed case but had eaten at the restaurant between 9 and 15 May 2014.

To ascertain whether any food handlers had experienced gastroenteritis symptoms and were a possible source of infection, investigators contacted and interviewed staff who worked at the restaurant between 9 and 16 May 2014.

Data analysis was conducted using Stata 13 (StataCorp, College Station, TX). Univariate analysis was used to calculate *p*-values (2-sided Fisher exact), odds ratios (OR) and 95% confidence intervals (CI) for food exposure variables. Variables with a *p*-value <0.05 in univariate analysis were included in a multivariable logistic regression model. Backward elimination was used to refine the model with the variable with the highest *p*-value >0.05 removed at each elimination step. Variables found to be statistically significant on univariate and multivariable analyses were reported.

Environmental and laboratory investigation

Environmental health officers from the council health department conducted an environmental investigation at the restaurant. Food samples were obtained and tested for *Salmonella* spp., coagulase-positive staphylococci, *Bacillus cereus* and *Clostridium perfringens* at the Microbiological Diagnostic Unit Public Health Laboratory (MDU PHL). Food samples were not tested for norovirus as the molecular detection of norovirus in food remains prohibitively expensive, time consuming and often unsuccessful because of the heterogeneous distribution of low numbers of virus particles in foods.¹³

Stool specimens were obtained from cases where possible and tested for bacterial enteric pathogens at MDU PHL. The specimens were tested for norovirus by reverse transcription polymerase chain reaction (RT-PCR) at the Victorian Infectious Diseases Reference Laboratory and nucleotide sequencing of norovirus RNA was conducted where appropriate, as previously reported.¹⁴

Ethics and permissions

Ethics approval was not sought as the investigation was undertaken as part of a public health response to an outbreak.

RESULTS

Epidemiological investigation

Forty-six cases (including 16 confirmed cases) and 49 controls were identified and interviewed. The majority of cases dined at the restaurant on 11 May 2014 with most experiencing an onset of symptoms during the afternoon of 12 May 2014 or the morning of 13 May 2014. The last recorded case ate at the restaurant on 15 May 2014 and had an onset of symptoms on 17 May 2014. An incubation period was recorded for 29 cases, and a median of 28 hours (range 7 to 57 hours) was observed. Symptom duration was recorded for 27 cases, and a median duration of two days (range 0.5 to 5 days) was reported. Symptom characteristics of the cases are described in [Table 1](#).

Twelve staff members who worked between 9 and 16 May were interviewed during the investigation. Only one staff member reported experiencing acute gastroenteritis during this period with an onset of illness on 12 May 2014. The staff member had prepared salads on the day of onset and the previous two days. The staff member was unwilling to provide a faecal specimen for laboratory testing.

In univariate analysis, foods significantly associated with illness were the beetroot dip, grain salad (a cold salad containing freekeh wheat, lentils, parsley and nuts), coleslaw, calamari and lamb cutlets ([Table 2](#)). In the final multivariable logistic regression model, two foods remained significantly associated with illness: the grain salad (OR: 21.6, 95% CI: 1.8–252.7, *p* = 0.015) and the beetroot dip (OR: 22.4, 95% CI: 1.9–267.0, *p* = 0.014).

Table 1. Symptom characteristics of 46 cases identified among patrons of a restaurant in Melbourne, Australia, 2014

| Symptom | Number of cases reporting | Percentage of cases reporting |
|----------------|---------------------------|-------------------------------|
| Diarrhoea | 45 | 98% |
| Abdominal pain | 39 | 85% |
| Nausea | 33 | 72% |
| Vomiting | 29 | 63% |
| Headache | 27 | 59% |
| Fever | 16 | 35% |

Table 2. Food items associated with illness by univariate analysis

| Food Item | Cases (n = 46) (%) | Controls (n = 49) (%) | Odds ratio (95% confidence interval) | p-value |
|--------------|-----------------------|--------------------------|---|---------|
| Beetroot dip | 11 (24) | 1 (2) | 15.1 (2.0–662.4) | 0.001 |
| Grain salad | 45 (98) | 37 (76) | 14.6 (2.0–637.3) | 0.002 |
| Coleslaw | 18 (39) | 9 (18) | 2.9 (1.0–8.3) | 0.025 |
| Calamari | 9 (20) | 2 (4) | 5.7 (1.1–56.6) | 0.018 |
| Lamb cutlets | 9 (20) | 2 (4) | 5.7 (1.1–56.6) | 0.018 |

A review of food frequency data revealed 45 cases (98%) recalled eating the grain salad, and one case did not consume any other food from the restaurant except the grain salad. By comparison, only 11 of the 46 cases (24%) reported consumption of the beetroot dip.

Environmental and laboratory investigation

Sixteen of the 23 stool specimens were positive for GII norovirus. Additional studies indicated this was the epidemic strain GII.Pe/GII.4_Sydney_2012. All food and stool samples tested were negative for bacterial pathogens.

A senior staff member at the restaurant reported the grain salad was generally prepared in batches of approximately 12 kilograms to be used over a period of three to four days. It was mixed in large containers by hand; gloves were not always worn during the mixing process. An unusually large 19 kilogram batch of grain salad was made in preparation for the 11 May 2014 lunch. The salad did not undergo any processing steps (such as cooking) that would have inactivated norovirus potentially introduced by an infected food handler during preparation.

The environmental investigation revealed some of the handwashing facilities in food preparation areas were obstructed and not supplied with soap.

DISCUSSION

This point source outbreak of norovirus among restaurant patrons is likely to have been caused by person-to-food-to-person transmission. The results of the case-control study analysis, the higher frequency of consumption of the grain salad among cases, the likelihood an infectious food handler prepared this menu item and the ready-to-eat nature of the product all support the grain salad as the most likely vehicle of infection, though it is possible that the beetroot dip may have also been contaminated. The large batch size of the grain salad and the extended time over which it was served likely contributed to the protracted onsets of illness among patrons.

Although norovirus infection was not confirmed in any food handlers at the restaurant, it is suspected that ready-to-eat food was contaminated by a food handler during the pre-symptomatic or the early-symptomatic stage of illness. The lack of adequate handwashing facilities in food preparation areas further supports this

hypothesis, as does anecdotal evidence that salads were often mixed with bare hands. National food standards in Australia require food handlers to take all practical measures to prevent unnecessary contact with ready-to-eat food and detail requirements for the availability and use of handwashing facilities by food handlers.¹⁵

National food standards in Australia also require a food business to exclude employees having a foodborne disease from handling food until a medical practitioner advises the employee no longer has or is carrying the disease.¹⁵ While this requirement aims to minimize the risk of food contamination by ill food handlers, it may also discourage reporting of gastroenteritis among food handlers, many of whom are employed on a casual basis and receive no entitlements when absent from work for medical reasons.

Several limitations were identified in this study. The retrospective nature of the case-control study makes it impossible to rule out recall bias, which may have been exacerbated by the large number of food choices available on the banquet menu. The lack of a practical method for the molecular detection of norovirus in food samples meant that it was not possible to confirm the presence of norovirus in the implicated foods. Food handlers, including asymptomatic individuals, were not tested for norovirus infection during the investigation, so the presence of an infected food handler at the premises could not be laboratory-confirmed.

This outbreak emphasizes the importance of the exclusion of symptomatic food handlers and strict hand hygiene practices in the food service industry to prevent contamination of ready-to-eat foods and the kitchen environment. It is essential that food regulators continue to promote and enforce these requirements on food business operators to prevent future outbreaks of norovirus caused by infectious food handlers.

Conflicts of Interest

None.

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References

1. Ahmed SM, Hall AJ, Robinson AE, Verhoef L, Premkumar P, Parashar UD, et al. Global prevalence of norovirus in cases of gastroenteritis: a systematic review and meta-analysis. *Lancet Infect Dis*. 2014 Aug;14(8):725–30. pmid:24981041 doi:10.1016/S1473-3099(14)70767-4
2. Robilotti E, Deresinski S, Pinsky BA. Norovirus. *Clin Microbiol Rev*. 2015 Jan;28(1):134–64. pmid:25567225 doi:10.1128/CMR.00075-14
3. Patel MM, Hall AJ, Vinjé J, Parashar UD. Noroviruses: a comprehensive review. *J Clin Virol*. 2009 Jan;44(1):1–8. pmid:19084472 doi:10.1016/j.jcv.2008.10.009
4. Matthews JE, Dickey BW, Miller RD, Felzer JR, Dawson BP, Lee AS, et al. The epidemiology of published norovirus outbreaks: a review of risk factors associated with attack rate and genogroup. *Epidemiol Infect*. 2012 Jul;140(07):1161–72. pmid:22444943 doi:10.1017/S0950268812000234
5. Atmar RL, Opekun AR, Gilger MA, Estes MK, Crawford SE, Neill FH, et al. Norwalk virus shedding after experimental human infection. *Emerg Infect Dis*. 2008 Oct;14(10):1553–7. pmid:18826818 doi:10.3201/eid1410.080117
6. Rockx B, De Wit M, Vennema H, Vinjé J, De Bruin E, Van Duynhoven Y, et al. Natural history of human calicivirus infection: a prospective cohort study. *Clin Infect Dis*. 2002 Aug 01;35(3):246–53. pmid:12115089 doi:10.1086/341408
7. Teunis PFM, Sukhrie FHA, Vennema H, Bogerman J, Beersma MFC, Koopmans MPG. Shedding of norovirus in symptomatic and asymptomatic infections. *Epidemiol Infect*. 2015 Jun;143(08):1710–7. pmid:25336060 doi:10.1017/S095026881400274X
8. Ozawa K, Oka T, Takeda N, Hansman GS. Norovirus infections in symptomatic and asymptomatic food handlers in Japan. *J Clin Microbiol*. 2007 Dec;45(12):3996–4005. pmid:17928420 doi:10.1128/JCM.01516-07
9. Maritschnik S, Kanitz EE, Simons E, Höhne M, Neumann H, Allerberger F, et al. A Food Handler-Associated, Foodborne Norovirus GII.4 Sydney 2012-Outbreak Following a Wedding Dinner, Austria, October 2012. *Food Environ Virol*. 2013 Sep 12;5(220). pmid:24026524
10. Friedman DS, Heisey-Grove D, Argyros F, Berl E, Nsubuga J, Stiles

- T, et al. An outbreak of norovirus gastroenteritis associated with wedding cakes. *Epidemiol Infect.* 2005 Dec;133(06):1057–63. pmid:16274502 doi:10.1017/S0950268805004760
11. Mayet A, Andreo V, Bedubourg G, Victorion S, Plantec J, Soullie B, et al. Food-borne outbreak of norovirus infection in a French military parachuting unit, April 2011. *Euro Surveill.* 2011 July 28;16(30):19930. pmid:21813082
 12. Chen M-Y, Chen W-C, Chen P-C, Hsu S-W, Lo Y-C. An outbreak of norovirus gastroenteritis associated with asymptomatic food handlers in Kinmen, Taiwan. *BMC Public Health.* 2016 May 04;16(1):372. pmid:27143036 doi:10.1186/s12889-016-3046-5
 13. Stals A, Baert L, De Keuckelaere A, Van Coillie E, Uyttendaele M. Evaluation of a norovirus detection methodology for ready-to-eat foods. *Int J Food Microbiol.* 2011 Feb 28;145(2-3):420–5. pmid:21333370 doi:10.1016/j.ijfoodmicro.2011.01.013
 14. Bruggink LD, Dunbar NL, Catton MG, Marshall JA. Norovirus genotype diversity associated with gastroenteritis outbreaks in Victoria in 2013. *Commun Dis Intell Q Rep.* 2015 March 31;39(1):E34–41. pmid:26063096
 15. Australia New Zealand Food Standards Code. Canberra, A.C.T: Food Standards Australia New Zealand; 2016 (www.foodstandards.gov.au/code, accessed 28 April 2017).

The burden of congenital rubella syndrome in the Philippines: results from a retrospective assessment

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Introduction: In line with the regional aim of eliminating rubella and congenital rubella syndrome (CRS), phased introduction of rubella-containing vaccines (RCV) in the Philippines' routine immunization programme began in 2010. We estimated the burden of CRS in the country before widespread nationwide programmatic RCV use.

Methods: We performed a retrospective chart review in four tertiary hospitals. Children born between 1 January 2009 and 31 December 2014 and identified as possible CRS cases based on the presence of one or more potential manifestations of CRS documented in hospital or clinic charts were reviewed. Cases that met the clinical case definition of CRS were classified as either confirmed (with laboratory confirmation) or probable (without laboratory confirmation). Cases that did not fulfil the criteria for either confirmed or probable CRS were excluded from the analysis.

Results: We identified 18 confirmed and 201 probable cases in this review. Depending on the hospital, the estimated incidence of CRS ranged from 30 to 233 cases per 100 000 live births. The estimated national burden of CRS was 20 to 31 cases per 100 000 annually.

Discussion: This is the first attempt to assess the national CRS burden using in-country hospital data in the Philippines. Prospective surveillance for CRS and further strengthening of the ongoing measles-rubella surveillance are necessary to establish accurate estimates of the burden of CRS and the impact of programmatic RCV use in the future.

Rubella, also known as German measles, is an exanthematous disease that commonly causes mild fever and rash that begins on the face and gradually spreads to the neck, trunk and extremities. While most infections are mild, infection in a pregnant woman may cause devastating foetal malformations and may result in stillbirths, miscarriage or a pattern of birth defects known as congenital rubella syndrome (CRS).^{1–3}

The use of effective rubella-containing vaccines (RCV) has resulted in significant reductions in the incidence of rubella and CRS in countries that have included rubella vaccines in their national immunization programmes. In 2015, it was announced that the countries in the World

Health Organization (WHO) Region of the Americas had eliminated endemic transmission of rubella and CRS.⁴ Before routine rubella vaccination, the incidence of CRS worldwide ranged from 10 to 20 cases per 100 000 live births to 80 to 400 cases per 100 000 live births during intra-epidemic and epidemic periods, respectively.^{3,5–7} Globally, it is estimated that there were 105 391 cases of CRS in 2010, representing a decline of 11.6% from 1996.⁸ In the WHO Global Vaccine Action Plan 2011–2020, a goal to eliminate both measles and rubella in at least five regions of the WHO was established.⁹ In October 2014, the WHO Regional Committee for the Western Pacific Region included rubella elimination plus CRS prevention as one of eight regional immunization goals specified by the

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Regional Framework for Implementation of the Global Vaccine Action Plan in the Western Pacific.¹⁰ To support this goal, the Technical Advisory Group on Immunization and Vaccine Preventable Diseases in the Western Pacific Region recommended enhancing surveillance activities for rubella and CRS with case detection and thorough outbreak investigations as well as appropriate case management and vaccination of susceptible contacts.¹¹ In the Philippines, rubella surveillance is conducted as part of measles surveillance. No CRS surveillance currently exists anywhere in the Philippines.

In the Philippines, a pilot project introduced RCV in five of the 18 regions of the country in 2009. In 2010, RCV was incorporated into the national routine immunization programme targeting children aged 12–15 months with the combined measles-mumps-rubella (MMR) vaccine. Children up to the age of 95 months were additionally covered by a national measles and rubella supplemental immunization campaign in 2011.¹² Coverage for MMR gradually rose from 31% in 2011 to 38% in 2012–2013, and 64% in 2014; it was 62% in 2015. MMR coverage remained low due to vaccine stock-outs in 2013 and 2015 and delayed reporting from the 18 regions.¹³ To date, women of childbearing age have not been targeted systematically for rubella vaccination in the Philippines.

We aimed to estimate the burden of CRS in the country through a retrospective chart review to provide a baseline before widespread introduction of rubella vaccines. This information is important for evaluating the impact of the introduction of RCV into the immunization programme.

METHODS

We conducted a retrospective review of hospital records in four large hospitals in the country. These hospitals, which are public, tertiary training hospitals equipped with subspecialists capable of managing CRS, are known to have the highest annual CRS consultations. They were selected based on their large catchment area that encompasses the three main island groups of the Philippines as well as their ability to provide care to CRS cases. Two of the hospitals were in Metro Manila in the most populated island of Luzon (Philippine General Hospital, PGH, in the City of Manila, and Philippine Children's Medical Center, PCMC, in Quezon City), one in Cebu City in the Visayas (Vicente Sotto Memorial Medical

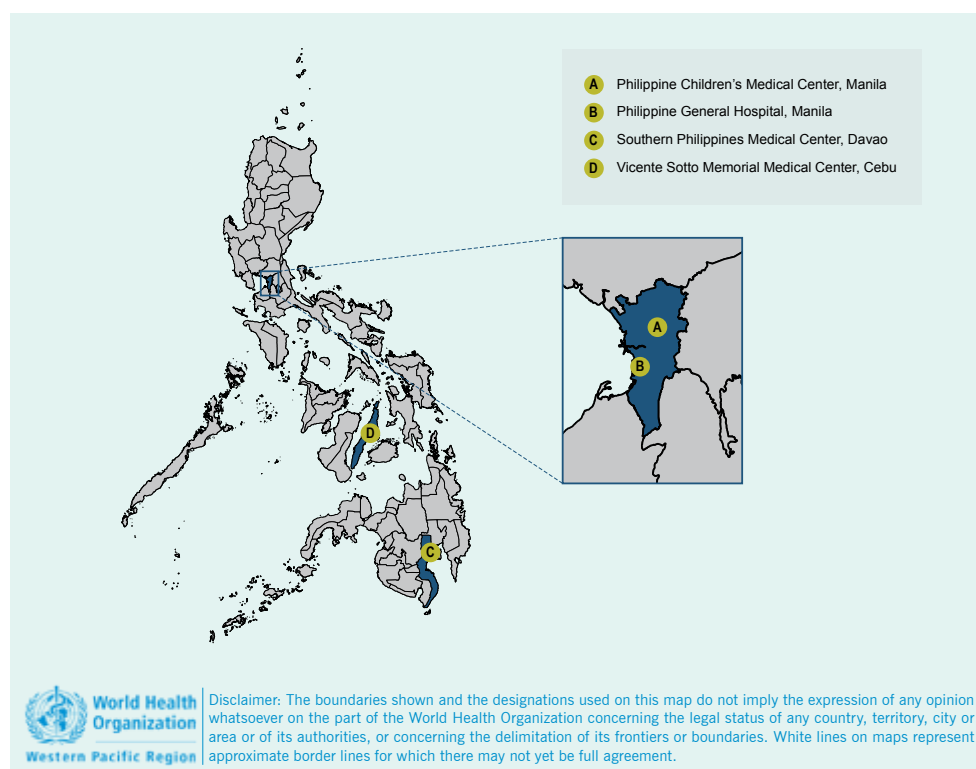
Center, VSMMC) and one in Davao City in Mindanao (Southern Philippines Medical Center, SPMC) (Fig. 1).

Records review and case classification

The following patients were included in the review: children born between 1 January 2009 and 31 December 2014 who were hospitalized or received outpatient care at one of the study sites from 1 January 2009 until 31 December 2014 with:

- documented positive rubella immunoglobulin M (IgM) laboratory test result; OR
- International Classification of Disease (ICD)-9¹⁴ or ICD-10¹⁵ discharge code consistent with one or more manifestation(s) of CRS; ICD-9/ICD-10 codes used in the chart review were:
 1. congenital rubella syndrome (771.0/P35);
 2. cataracts (743.3/Q12);
 3. congenital glaucoma (743.2/Q15-H40);
 4. deafness and hearing impairment (389.1/H90);
 5. congenital heart disease (745–747/Q20–Q26);
 6. dermal erythropoiesis (759.89/P83.8);
 7. microcephaly (742.1/Q02); OR
- written documentation in the medical record of one or more manifestation(s) of CRS using the following diagnostic keywords:
 1. cardiac—congenital heart disease (CHD);
 2. patent ductus arteriosus (PDA);
 3. peripheral pulmonary artery stenosis;
 4. congenital cardiopathy;
 5. ventricular septal defect;
 6. ophthalmologic: cataract, microphthalmia, glaucoma, pigmentary retinopathy;
 7. auditory: deafness, hearing loss/hearing impairment;
 8. dermatologic: purpura, “blueberry muffin rash”; and
 9. others: microcephaly, mental retardation, developmental delay, neonatal jaundice, hepatosplenomegaly, meningoencephalitis, radiolucent bone disease, “rule out ToRCH infection,” congenital rubella syndrome or congenital rubella infection (including “suspected CRS” or “rule out congenital rubella”).

Figure 1. Map of the Philippines with location of the study hospitals



We excluded the following in our review: infants <2500 g with isolated PDA or isolated microcephaly and no other signs of CRS, documented negative rubella-specific IgG test for the child, documented positive laboratory test for other potential etiology of CRS manifestation (e.g. positive cytomegalovirus or toxoplasmosis test) in the absence of a positive rubella laboratory test and not a resident of the Philippines.

Charts were retrieved from all eligible cases. Information collected from the charts included hospital location; patient's province and region of residence; location of birth, maternal and infant demographics; infant's clinical signs and symptoms; maternal history; and laboratory tests performed. Data were collected on standard forms and entered securely into an electronic database using Epi Info™ 7 (Centers for Disease Control and Prevention, Atlanta, Georgia, USA). Participants were coded using a unique surveillance identification number.

Data analysis

Data analysis was performed using Epi Info™ 7. We used the case definition from WHO surveillance standards^{16,17} to classify the identified cases (**Box 1**). Estimated annual

incidence rates were calculated using different methods. First, we computed hospital-specific incidence including only babies who were born at PGH, SPMC or VSMC in the analysis. Since few deliveries occurred in PCMC, incidence rate for this hospital was not calculated. The numerator was the respective number of probable or confirmed CRS cases in one of the three study sites and the denominator was the number of live births in the same hospitals from 1 January 2009 to 31 December 2014. To calculate the national incidence rates, we used the method previously used by Bloom, et al. using cataract detection in Morocco to calculate the national burden¹⁹ with the following formula:

$$I = (CRSp + CRSc) \times \frac{1}{\%C} \times \frac{1}{\%CRS \text{ cases with cataracts}}$$

Where I = incidence, CRSp = probable CRS cases, CRSc = confirmed CRS cases, %C = percentage of overall cataract care provided at three participating hospitals, and %CRS cases with cataract = CRS cases with cataracts based on previous literature.

Based on previous studies, 16–25% of CRS cases have cataracts.^{20,21} For the national incidence estimation, we obtained the proportion of cataract care provided by

Box 1. Case definition and classifications used in the study^{16,18}

Case definition of congenital rubella syndrome

An illness, usually manifesting in infancy, resulting from rubella infection *in utero* and characterized by signs and symptoms from the following:

- Category (A): cataracts/congenital glaucoma, congenital heart disease (most commonly patent ductus arteriosus or peripheral pulmonic stenosis), loss of hearing, pigmentary retinopathy.
- Category (B): purpura, hepatosplenomegaly, jaundice, microcephaly, developmental delay, meningoencephalitis, radiolucent bone disease.

Laboratory criteria for diagnosis

- Isolation of rubella virus, or
- Demonstration of rubella-specific immunoglobulin M (IgM) antibody, or infant rubella antibody IgG level that persists at a higher level and for a longer period than expected from passive transfer of maternal antibody (i.e. rubella titre that does not drop at the expected rate of a twofold dilution per month), or
- Polymerase chain reaction (PCR) positive for rubella virus.

Case classifications

Suspected: a case that has some compatible clinical findings but does not meet the criteria for a probable case.

Probable: a case that is not laboratory confirmed but has any two complications listed in category (A) of the clinical description or one complication from category (A) and one from category (B), and lacks evidence of any other etiology.

Confirmed: a case that has any one complication listed in category (A) of the clinical description, or one complication from category (A) and one from category (B), and meets the above criteria for laboratory diagnosis.

each participating hospital by using the insurance claims for ICD-10 code Q12 (congenital cataract and congenital diseases of the lens) from PhilHealth (the National Health Insurance Programme). Based on the claims from PhilHealth from 2009 to 2013, PGH, PCMC, SPMC and VSMC accounted for 7%, 0%, 2% and 1% of all cataract care in the country, respectively, or 10% cumulatively for all hospitals.²² This database included reports from both private and public hospitals in the country that managed cases of congenital cataracts.

Ethics

This study was reviewed and approved by the Ethics Review Committee of the WHO Regional Office for the Western Pacific (2015.8.PHL.2.EPI), and the ethical review boards of the University of the Philippines Manila (UPM-REB 2015-205-01), PCMC, VSMC and the SPMC.

RESULTS

Out of 4339 unique entries identified from medical records, we identified 18 laboratory-confirmed cases and 201 probable CRS cases from the four hospitals. The majority of suspected cases came from PGH (1849), followed by PCMC (1091), SPMC (939) and VSMC (459). Both SPMC and VSMC had no confirmed cases due to the absence

of laboratories capable of performing a rubella IgM test in either Davao City or Cebu City. Clinical manifestations of CRS were predominantly cardiac (83.3% and 86.1% among confirmed and probable cases, respectively), audiologic (50% and 33.3% among confirmed and probable cases, respectively) and ophthalmologic (27.8% and 25.4% among confirmed and probable cases, respectively). Among all confirmed and probable CRS cases, the mean age of diagnosis was 9.9 months (range: 3 days–72 months) with more cases among males (55.7%) and the mean age of mothers was 27.8 (± 5.2) years, with only 13.2% reporting rashes on prenatal history by recall (**Tables 1 and 2**). The most common cardiac presentation was patent ductus arteriosus.

We obtained the number of live births in PGH, VSMC and SPMC. Using each hospital's live births, the estimate for CRS incidence ranged from 30 to 233 cases per 100 000 live births (**Table 3**).

There were 52 cataract cases among the 219 confirmed and probable cases identified from 2009 to 2014. Based on PhilHealth claims for congenital cataracts from 2009 to 2013, PGH, PCMC, SPMC and VSMC together accounted for 10% of all cataract cases nationwide. Thus, there were an estimated 520 diagnosed cataract cases nationally from 2009 to 2014. Using the reported live births in the country during the

Table 1. Characteristics of confirmed and probable CRS cases

| Characteristics | Confirmed (n = 18) | Probable (n = 201) |
|---|--------------------|--------------------|
| Demographic profile | | |
| Age at presentation (in months, mean \pm std dev) | 3.5 \pm 4.0 | 10.5 \pm 14.6 |
| Male, n (%) | 12 (66.7%) | 110 (54.7%) |
| Year of birth, n (%) | | |
| 2009 | 1 (5.6%) | 42 (20.9%) |
| 2010 | 3 (16.7%) | 39 (19.4%) |
| 2011 | 7 (38.9%) | 41 (19.9%) |
| 2012 | - | 33 (15.9%) |
| 2013 | 4 (22.2%) | 30 (13.9%) |
| 2014 | 3 (16.7%) | 21 (10.0%) |
| Age of mother (in years, mean \pm std dev) | 25.5 \pm 5.7 | 28.0 \pm 5.3 |
| History of maternal rash (n,%) | 4 (22.2%) | 25 (12.4%) |
| Hospital | | |
| Philippine Children's Medical Center (PCMC) | 10 (55.6%) | 80 (39.8%) |
| Philippine General Hospital (PGH) | 8 (44.4%) | 68 (33.8%) |
| Southern Philippines Medical Center (SPMC) | - | 37 (18.4%) |
| Vicente Sotto Memorial Medical Center (VSMMC) | - | 16 (8.0%) |
| Clinical presentation* | | |
| Cardiac | | |
| Patent ductus arteriosus | 11 (61.1%) | 113 (56.2%) |
| Pulmonary stenosis | 2 (11.1%) | 33 (16.4%) |
| Ventricular septal defect | 2 (11.1%) | 27 (13.4%) |
| Ophthalmologic | | |
| Cataract (bilateral or unilateral) | 5 (27.8%) | 47 (23.4%) |
| Glaucoma | | |
| Pigmentary retinopathy | - | 2 (1.0%) |
| Audiologic | | |
| Sensorineural hearing loss | 6 (33.3%) | 63 (31.3%) |
| Deafness | 3 (16.7%) | 3 (1.5%) |
| Others | | |
| Neonatal jaundice | 1 (5.6%) | 79 (39.3%) |
| Mental retardation | 2 (33.3%) | 35 (14.4%) |
| Hepatosplenomegaly | 3 (33.3%) | 21 (10.4%) |
| Radiolucent bone disease | 1 (5.6%) | - |
| Purpura | 3 (33.3%) | - |
| Meningoencephalitis | 1 (5.6%) | - |

* Cases may have more than one cardiac and ophthalmologic manifestation. Figures should not be considered as part of a whole.

same period,²³ and adjusting by 4–6.25 times (the inverse of 16–25% of CRS cases have cataracts), then an estimated 2080 to 3250 CRS cases nationally from 2009 to 2014, or an annual incidence of 20 to 31 CRS cases per 100 000 live births.

Table 2. Clinical profile of confirmed and probable CRS cases

| Clinical manifestations | n |
|--|------------|
| Confirmed cases | |
| CHD, hearing loss | 7 |
| CHD* | 4 |
| Cataract* | 3 |
| CHD, cataract, hearing loss | 1 |
| CHD, cataract | 1 |
| Hearing loss* | 1 |
| Hepatosplenomegaly, meningoencephalitis | 1 |
| Total | 18 |
| Probable cases | |
| CHD, jaundice | 59 |
| CHD, hearing loss | 32 |
| CHD, cataract† | 24 |
| CHD, hepatosplenomegaly with or without jaundice | 23 |
| CHD, global developmental delay and/or mental retardation | 20 |
| Hearing loss, global developmental delay and/or mental retardation | 14 |
| Cataract, hearing loss | 8 |
| CHD, cataract, hearing loss | 8 |
| Cataract, mental retardation and/or global developmental delay | 4 |
| Hearing loss, jaundice | 2 |
| Cataract, jaundice | 2 |
| CHD, pigmentary retinopathy | 1 |
| Congenital glaucoma, hearing loss‡ | 1 |
| Hearing loss, Pigmentary retinopathy, | 1 |
| CHD, meningoencephalitis | 1 |
| CHD, microcephaly | 1 |
| Total | 201 |

CHD=congenital heart defect

* Had other minor manifestations

† Among the 24 patients, one had CHD, bilateral cataract and congenital glaucoma

‡ Had combined bilateral cataract and congenital glaucoma

DISCUSSION

We documented the occurrence of CRS in the Philippines; cardiac and ophthalmologic defects were the most common findings, similar to previous studies conducted in Sudan,²⁴ Viet Nam²⁵ and the Philippines.²⁶ Our estimates for CRS varied widely by hospital. WHO estimates that there were 150 cases of CRS per 100 000 live births in the Philippines in 2010, or about 2674 cases of CRS, much higher than estimates obtained in

Table 3. Clinical profile of confirmed and probable CRS cases

| Hospital* | Live Births (2009–2014) | Cases | Incidence (per 100 000 live births) |
|---|-------------------------|-------|-------------------------------------|
| Philippine General Hospital (PGH) | 32 681 | 76 | 233 |
| Southern Philippines Medical Center (SPMC) | 77 915 | 37 | 47 |
| Vicente Sotto Memorial Medical Center (VSMMC) | 54 217 | 16 | 30 |
| PGH, SPMC, VSMMC | 166 983 | 127 | 76 |

* Since few deliveries occurred in PCMC, incidence rate for this hospital was not calculated.

this review.²⁷ Previous estimates of CRS were based on modelling using rubella seroprevalence data together with the incidence of infection during gestation²⁸ or with immunization coverage in the different countries,⁸ while this study was a retrospective assessment of CRS using admission records.

The national estimate we obtained based on cataract care is conservative. First, our review covered only a small proportion of the country and is not representative of the entire population. We conducted chart reviews in four public hospitals that were the biggest tertiary public referral centres in the country's three major island groups and located in urbanized centres. As CRS diagnosis requires consultation with subspecialists that is typically unavailable at small hospitals, most cases should have been referred to one of these hospitals. A closer review of the data from PGH and PCMC showed that only 59% and 57%, respectively, of the patients came from Metro Manila; the rest came from other areas. But despite the four hospitals' large catchment areas, there are more than 1800 hospitals in the Philippines. In addition, since only 40% of Philippines' hospitals are government-owned, some patients may have sought care in the private sector. It is estimated that 30% of the population use private fee-for-service medical care.²⁹ Second, there are differences in the hospitals included in the study. The higher incidence seen in PGH compared to SPMC and VSMMC may be due to the nature of deliveries performed at PGH. PGH is the largest training and referral hospital in the Philippines and only high-risk pregnancies are admitted; hence normal deliveries are limited at the hospital. PGH is also considered to have the most complete subspecialty services; thus patients requiring

complicated case management are often transferred to this hospital. Conversely, the hospitals in Cebu and Davao did not have adequate laboratories to diagnose CRS. Subspecialty services (paediatric ophthalmology and audiology) were also inconsistently available during the inclusive dates under review. Thus, children seeking eye care and hearing tests may have sought care at private health facilities and therefore possibly missed. With the passage of a law in 2009 that requires mandatory hearing screening of all newborns, more public facilities are able to conduct hearing testing and identify cases. Third, as in any retrospective chart review, we encountered difficulties in retrieving patient records and abstracting information from clinical sources. A significant number of medical records were missing in the archiving facilities of respective hospitals. Retrieved medical records, likewise, had incomplete documentation. The incomplete records and inaccurate coding may also result in misclassification and reduce our estimates. Fourth, we found many cases in which care from hospitals was sought late. Many children with hearing and visual impairment were seen after 5 years of age and therefore were missed in this retrospective case finding. In PGH, only 30% of children with hearing loss were referred before 1 year of age,³⁰ and CRS was the most common (36%) etiology of hearing loss in 94 patients who underwent cochlear implantation.³¹ Fifth, the estimate on the national incidence is likely to be an underestimate due to the low utilization and coverage of PhilHealth for the lower economic strata from 2009 to 2014. Although 88% of the population were enrolled in 2015 in PhilHealth, from 2009 to 2014 PhilHealth utilization remained low.²² Lastly, the phased introduction of RCV may have affected our results since RCV was initially introduced in 2009 before inclusion into the national routine immunization programme targeting children aged 12–15 months with the MMR vaccine and as supplemental immunization campaigns in children up to the age of 95 months in 2011 resulting in low RCV coverage initially but increasing coverage as the study progressed. However, by 2014, the national childhood RCV coverage was <70% due to vaccine stock-outs and in Metro Manila, RCV coverage was <50%. At this vaccine coverage, it is unlikely that susceptible pregnant women would benefit from herd immunity.³²

Currently, women of childbearing age are not systematically targeted for rubella vaccination in the Philippines. In 2002, 15% of women in an urban antenatal clinic remained susceptible to rubella.²⁶ In the

absence of vaccination, a large cohort of this population remains at risk for being infected with rubella during pregnancy. From 1 January to 22 October 2016, there were 119 laboratory-confirmed cases of rubella out of 1732 suspected measles-rubella cases captured by the Philippine Department of Health surveillance. Of these, 23% of cases were among women aged 16 to 30 years.³³

To the best of our knowledge, this is the first attempt to obtain an estimate of the burden of CRS using hospital data in the Philippines. The estimates varied widely by hospital and the national estimate we obtained was substantially lower than those obtained from models. Prospective surveillance will be important to obtain the true burden of CRS in the Philippines. New CRS surveillance guidelines are now available and these will be used as the country strengthens its rubella surveillance and plans to embark on a prospective CRS surveillance. Care must be taken in choosing potential surveillance sites to obtain reliable data.

Conflicts of interest

None.

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References

- McLean H, Redd SB, Abernathy E, Icenogle JP, Wallace G. Rubella. In: Roush SW, Baldy LM, editors. Manual for the surveillance of vaccine-preventable diseases. Atlanta: Centers for Disease Control and Prevention; 2012 (<https://www.cdc.gov/vaccines/pubs/surv-manual/chpt14-rubella.html>).
- McLean H, Redd SB, Abernathy E, Icenogle JP, Wallace G. Congenital rubella syndrome. In: Roush SW, editor. Manual for the surveillance of vaccine-preventable diseases. Atlanta: Centers for Disease Control and Prevention; 2012 (<https://www.cdc.gov/vaccines/pubs/surv-manual/chpt15-crs.html>).
- Robertson SE, Featherstone DA, Gacic-Dobo M, Hersh BS. Rubella and congenital rubella syndrome: global update. Rev Panam Salud Publica. 2003 Nov;14(5):306–15. pmid:14870758 doi:10.1590/S1020-49892003001000005
- Americas region is declared the world's first to eliminate rubella. Washington, DC: World Health Organization Regional Office for the Americas; 2015 (http://www2.paho.org/hq/index.php?option=com_content&view=article&id=10798%3Aamericas-free-of-rubella&Itemid=1926&lang=en, accessed 4 March 2017)
- Lawn JE, Reef S, Baffoe-Bonnie B, Adadevoh S, Caul EO, Griffin GE. Unseen blindness, unheard deafness, and unrecorded death and disability: congenital rubella in Kumasi, Ghana. Am J Public Health. 2000 Oct;90(10):1555–61. pmid:11029988 doi:10.2105/AJPH.90.10.1555
- Thant KZ, Oo WM, Myint TT, Shwe TN, Han AM, Aye KM, et al. Active surveillance for congenital rubella syndrome in Yangon, Myanmar. Bull World Health Organ. 2006 Jan;84(1):12–20. pmid:16501710 doi:10.2471/BLT.05.022814
- World Health Organization. Rubella vaccines: WHO position paper. Wkly Epidemiol Rec. 2011 Jul 15;86(29):301–16. pmid:21766537
- Vynnycky E, Adams EJ, Cutts FT, Reef SE, Navar AM, Simons E, et al. Using seroprevalence and immunisation coverage data to estimate the global burden of congenital rubella syndrome, 1996-2010: A Systematic Review. PLoS One. 2016 03 10;11(3):e0149160. pmid:26962867 doi:10.1371/journal.pone.0149160
- World Health Organization. Global Vaccine Action Plan. Decade of vaccine collaboration. Vaccine. 2013 Apr 18;31 Suppl 2:B5–31. pmid:23734366 doi:10.1016/j.vaccine.2013.02.015
- Regional Committee for Western Pacific. Regional Framework for Implementation of the Global Vaccine Action Plan in the Western Pacific. Manila: World Health Organization; 2014 October 16. Report No.: WPR/RC65.R5. Available from: http://www.wpro.who.int/about/regional_committee/65/documents/wpr_rc065_08_epi_en.pdf.
- 24th Meeting of the Technical Advisory Group on Immunization and Vaccine Preventable Diseases in the Western Pacific Region. Manila: WHO Regional Office for the Western Pacific; 2015 (http://iris.wpro.who.int/bitstream/handle/10665.1/12687/RS_2015_GE_16_PHL_eng.pdf?ua=1).
- Family Health Office - Expanded Program for Immunization. The Philippine Immunization Program Strategic Plan for 2015–2019. Manila: Department of Health, Philippines; 2015.
- Family Health Office - Expanded Program for Immunization. The Philippine Immunization Program Strategic Plan for 2016–2022. Manila: Department of Health, Philippines; 2016.
- ICD-9. International Statistical Classification of Diseases and Related Health Problems 9th Revision. Geneva: World Health Organization; 1978 (<http://www.who.int/iris/handle/10665/39473>).
- ICD-10. International Statistical Classification of Diseases and Related Health Problems 10th Revision. Geneva: World Health Organization; 2016 (<http://apps.who.int/classifications/icd10/browse/2016/en>).
- Vaccines and biologicals: WHO-recommended standards for surveillance of selected vaccine-preventable diseases. Geneva: World Health Organization; 2008 (http://apps.who.int/iris/bitstream/10665/68334/1/WHO_V-B_03.01_eng.pdf).
- Introducing rubella vaccine into national immunization programmes: a step by step guide. Geneva: World Health Organization; 2015 (http://apps.who.int/iris/bitstream/10665/184174/1/9789241549370_eng.pdf).

18. Centers for Disease Control and Prevention (Hamborsky J, Kroger A, Wolfe S, editors). *Epidemiology and prevention of vaccine preventable diseases*. Washington, DC: Public Health Foundation; 2015 (<https://www.cdc.gov/vaccines/pubs/pinkbook/index.html>).
19. Bloom S, Rguig A, Berraho A, Zniber L, Bouazzaoui N, Zaghloul Z, et al. Congenital rubella syndrome burden in Morocco: a rapid retrospective assessment. *Lancet*. 2005 Jan 8-14;365(9454):135–41. pmid:15639295 doi:10.1016/S0140-6736(05)17703-4
20. Reef SE, Plotkin S, Cordero JF, Katz M, Cooper L, Schwartz B, et al. Preparing for elimination of congenital rubella syndrome (CRS): summary of a workshop on CRS elimination in the United States. *Clin Infect Dis*. 2000 Jul;31(1):85–95. pmid:10913402 doi:10.1086/313928
21. Wolff SM. The ocular manifestation of congenital rubella. *Trans Am Ophthalmol Soc*. 1972;70:577–614. pmcid: PMC1310470
22. Stats & Charts 1st Semester 2015. Manila: Philippine Health Insurance Corporation; 2015 (http://www.philhealth.gov.ph/about_us/statsncharts/snc2015_1st.pdf, accessed 15 May 2016).
23. Live Births Philippines 2014. Manila: Philippine Statistics Authority; 2015 (<https://psa.gov.ph/content/live-births-philippines-2014>, cited May 5 2015).
24. Adam O, Ali AK, Hübschen JM, Muller CP. Identification of congenital rubella syndrome in Sudan. *BMC Infect Dis*. 2014 06 04;14(1):305. pmid:24898017 doi:10.1186/1471-2334-14-305
25. Toda K, Reef S, Tsuruoka M, Iijima M, Dang TH, Duong TH, et al. Congenital rubella syndrome (CRS) in Vietnam 2011–2012–CRS epidemic after rubella epidemic in 2010–2011. *Vaccine*. 2015 Jul 17;33(31):3673–7. pmid:26087296 doi:10.1016/j.vaccine.2015.06.035
26. Lopez AL, Raguindin PFN, Silvestre MA, Fabay XCJ, Vinarao AB, Manalastas R. Rubella and congenital rubella syndrome in the Philippines: a systematic review. *Int J Pediatr*. 2016;2016(8):8158712. pmid:28115948
27. Vynnycky E, Adams EJ, Cutts FT, Reef SE, Navar AM, Simons E, et al. Using seroprevalence and immunisation coverage data to estimate the global burden of congenital rubella syndrome, 1996–2010: a systematic review. *PLoS One*. 2016 03 10;11(3):e0149160. pmid:26962867 doi:10.1371/journal.pone.0149160
28. Cutts FT, Vynnycky E. Modelling the incidence of congenital rubella syndrome in developing countries. *Int J Epidemiol*. 1999 Dec;28(6):1176–84. pmid:10661666 doi:10.1093/ije/28.6.1176
29. Health service delivery profile: Philippines. Manila: WHO Representative Office in the Philippines; 2012 (http://www.wpro.who.int/health_services/health_service_delivery_profiles/en/, accessed 29 December 2016).
30. Chiong C, Ostrea E Jr, Reyes A, Llanes EG, Uy ME, Chan A. Correlation of hearing screening with developmental outcomes in infants over a 2-year period. *Acta Otolaryngol*. 2007 Apr;127(4):384–8. pmid:17453458 doi:10.1080/00016480601075431
31. Chiong CM, Villanueva EM. Cochlear implantation in chronic otitis media. *Acta Med Philipp*. 2012;46(3):21–6.
32. Anderson RM, May RM. Vaccination against rubella and measles: quantitative investigations of different policies. *J Hyg (Lond)*. 1983 Apr;90(02):259–325. pmid:6833747 doi:10.1017/S002217240002893X
33. Measles-Rubella Cases. Morbidity Week 42. Manila: Epidemiology Bureau, Department of Health, Philippines; 2016 (http://www.doh.gov.ph/sites/default/files/statistics/MEASLESRUBELLA42_compressed.pdf, accessed 29 December 2016).

Meningococcal disease outbreak related to the World Scout Jamboree in Japan, 2015

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Problem: Six invasive meningococcal disease cases occurred among Scottish and Swedish nationals associated with the World Scout Jamboree (WSJ), an international mass gathering, held in Japan. The index case developed symptoms while returning home. The strains from all six cases were identical and seldom seen in Japan.

Context: Over 33 000 participants from 155 countries attended WSJ. At the Jamboree site, participants of the North of Scotland's and Sweden's units camped within the same subcamp and kept the same schedule of events. No information was available about the Swedish and Scottish cases' close personal contact history.

Action: Health Protection Scotland investigated Scottish cases, conducted active case finding, provided chemoprophylaxis, vaccinated close contacts and advised Scottish WSJ participants and contacts to seek medical care if they developed symptoms. The Public Health Agency of Sweden recommended chemoprophylaxis to all participants in Sweden. In Japan, the Ministry of Health, Labour and Welfare (MHLW) requested the Scout Association of Japan advise all participants to seek medical attention if they developed symptoms. MHLW shared information about the event with local authorities, medical associations, and the Ministry of Education, Culture, Sports, Science and Technology.

Outcome: No additional case related to WSJ has been reported. This outbreak highlighted the risk for international spread of invasive meningococcal disease at international mass gatherings.

Discussion: Assessing risk, educating participants, enhancing surveillance and sharing timely information among related countries are significant for prevention and response against invasive meningococcal disease outbreaks at mass gatherings.

PROBLEM

The 23rd World Scout Jamboree (WSJ), held in Yamaguchi prefecture, Japan, from 28 July to 8 August 2015, was a mass gathering in which over 33 000 participants attended from 155 countries. Throughout the event, participants slept in shared tents and participated in socializing activities.¹ These types of close interactions can increase the risk of infectious diseases.

Six cases of invasive meningococcal disease (IMD) related to WSJ, including three scouts and one parent from Scotland and two scouts from Sweden, were reported by public health agencies in the United Kingdom and Sweden after WSJ had ended. The index case developed symptoms while returning to Scotland. The strains from

all six cases were identical and belonged to serogroup W,² a serogroup that has seldom been documented in Japan. The Ministry of Health, Labour and Welfare (MHLW) of Japan was notified and began an investigation. This paper summarizes the experience and lessons learnt from the IMD outbreak in the mass-gathering setting in Japan.

CONTEXT

WSJ is an official event of the World Organization of the Scout Movement that is designed for scouts aged 14 to 17 to live together, experience different cultures and take part in activities.¹ After Japan, the United Kingdom and Sweden were represented by the largest number of participants at the 2015 WSJ.

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At the Jamboree site, there were three hubs (Northern, Eastern and Western), each with four subcamps. The North of Scotland and Sweden units camped at Ishizuchi subcamp in the Western hub with 50 other units from different parts of the world. Each unit comprised 40 participants (36 scouts and four leaders) with two scouts per tent. Shared kitchen, shower and bathroom facilities were at the centre of each hub (Fig. 1). Scouts located in the same subcamp shared one schedule of events during WSJ. There were discotheques and campfire events at night that all participants were expected to attend.

During WSJ, every participant was required to report his or her health condition to WSJ headquarters every morning. Collected information was reported to the local jurisdiction of WSJ every day for syndromic surveillance. No participants developed meningitis symptoms during WSJ.

Between 8 and 19 August 2015, four meningococcal cases were confirmed by Health Protection Scotland (HPS), including three members of the North of Scotland WSJ unit and one family member of a participant. The onset date for the first case was 8 August (during return travel to Scotland), for the second case 11 August, the third 12 August and the fourth 16 August. The fourth case was the secondary case, a household contact (parent) of a scout from the North of Scotland unit. All four cases in Scotland received proper treatment and recovered without complications.^{3,4} A fourth scout, who was not a case, had a sore throat with onset 8 August, was prescribed amoxicillin on 10 August and was diagnosed with Group G streptococcus by throat swab microbiological analysis.

Two confirmed cases from the Swedish WSJ unit were reported by Public Health Agency of Sweden. One case, a scout who returned from Japan on 9 August, developed symptoms on 14 August and recovered after intensive treatment.^{3,4} This case attended a cultural day at the campsite hosted by the North of Scotland unit. The second case, also a scout, developed symptoms on 12 August and was later confirmed by serology.⁴ No information was available about close personal contact among the cases.

All six cases were confirmed as *Neisseria meningitidis* strain W: P1.5, 2, 36–2: F1–1: ST-11 (cc11) (see Table 1), which was indistinguishable from the strain

that has been increasing in England since 2009 and a recently increasing IMD capsular group W in Scotland.^{2,4} Based on data available (March 2013 to July 2016), this strain has not been reported recently in Japan.⁵ No IMD case (a nationally notifiable disease) associated with this outbreak has been reported in Japan as of 5 March 2016.

In this outbreak associated with WSJ, the attack rate (AR) among United Kingdom participants (scouts and leaders) was 102.2 cases per 100 000 participants (three cases in 2934 participants). For Swedish participants (scouts and leaders), the AR was 136.4 cases per 100 000 participants (two cases in 1466 participants). Among all participants in Ishizuchi subcamp, the AR was 240.4 per 100 000 (five cases in 2080), and for all WSJ participants (scouts and leaders), the AR was 19.5 per 100 000 (five in 25 649).

ACTION

According to HPS, active investigation was conducted in Scotland and chemoprophylaxis and vaccination were appropriately provided for close contacts.⁴ In addition, HPS emailed a letter to all scouts and leaders in the United Kingdom who attended WSJ to alert them to the incident and the signs and symptoms of meningitis and to advise them to seek medical care if they became symptomatic.^{3,4}

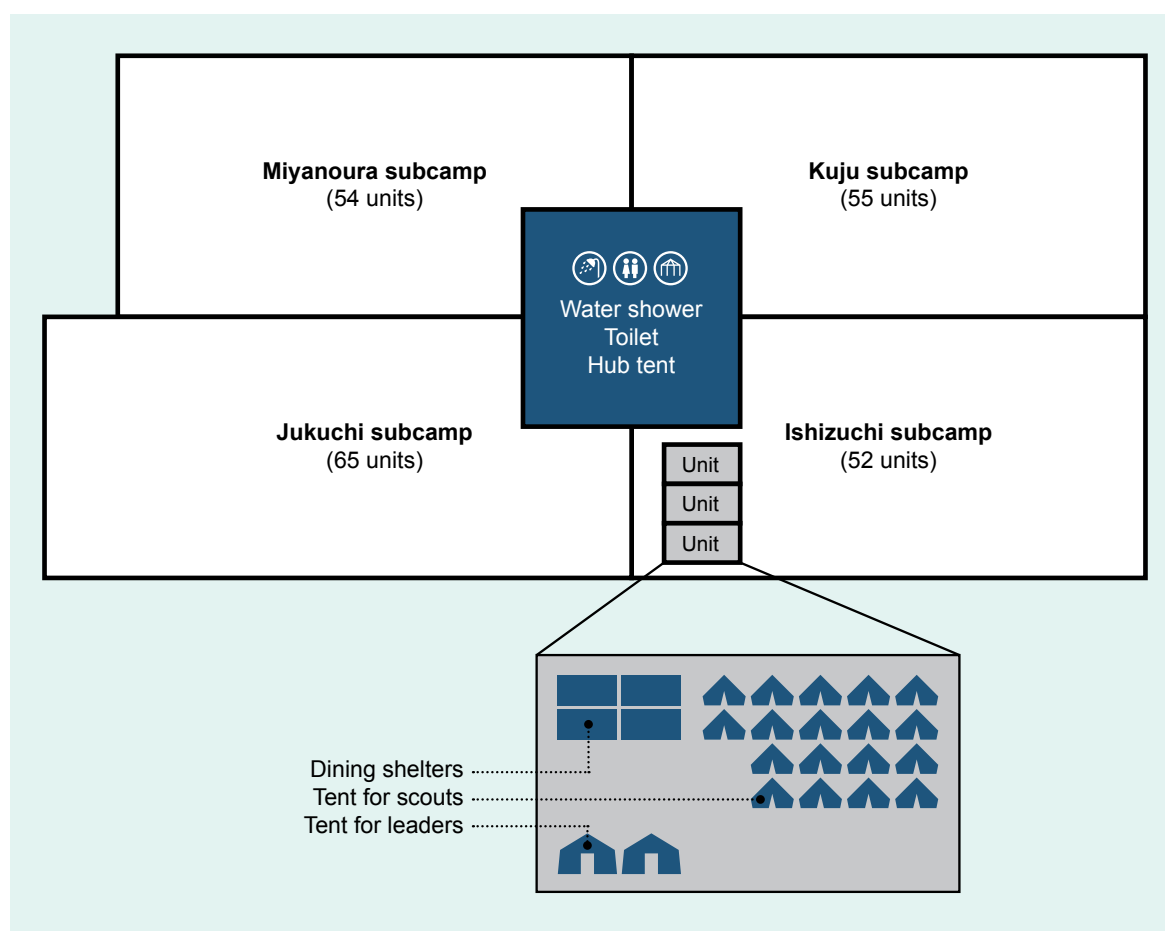
The public health actions in Sweden included a recommendation for all participants to seek health care to receive chemoprophylaxis and have nasopharyngeal and throat swabs taken.⁴

In Japan, MHLW held a teleconference with Scottish authorities to collect information on the cases. On 14 August, MHLW requested the Scout Association of Japan to advise WSJ participants to visit hospitals as soon as possible if they developed symptoms of meningococcal disease. On 19 August, MHLW advised all local health authorities to inform all medical institutions in their areas of the notice sent from the Scout Association of Japan (Fig. 2).

OUTCOME

No additional IMD case related to WSJ has been reported. An IMD outbreak occurred across multiple countries and was associated with a mass gathering. The event, WSJ,

Figure 1. Site map (Western Hub) of World Scout Jamboree 2015

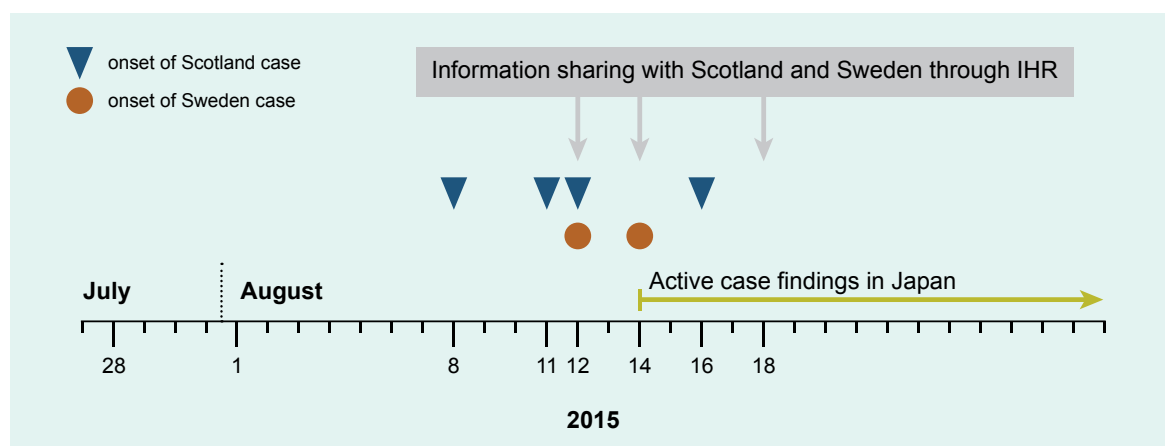


Western Hub had the following subcamps and units: 52 units in Ishizuchi subcamp, 65 units in Jakuchi subcamp, 55 units in Kuju subcamp, and 54 units in Miyanoura subcamp. Each unit consisted of 36 scouts and four leaders. Two scouts shared a tent.

Table 1. Linelist of confirmed cases of meningococcal outbreak associated with World Scout Jamboree (n = 6)

| Case No. | Unit | Onset date | Symptoms | Serogroup | Outcome |
|----------|-------------------------------------|------------|---|-----------|-----------|
| 1 | North of Scotland Scout | 8 Aug | Conjunctivitis, Fever, Headache, Nausea | W (ST11) | Remission |
| 2 | North of Scotland Scout | 11 Aug | Cough, Headache, Neck stiffness | W (ST11) | Remission |
| 3 | North of Scotland Scout | 12 Aug | Sour throat, Fever, Headache, Photophobia | W (ST11) | Remission |
| 4 | Parent of a North of Scotland scout | 16 Aug | Vomit, Myalgia, Headache, Photophobia | W (ST11) | Remission |
| 5 | Sweden Scout | 14 Aug | Signs of meningitis and septicemia | W (ST11) | Remission |
| 6 | Sweden Scout | 12 Aug | No info | W (ST11) | Remission |

Figure 2. Timeline of the onset of meningococcal cases related with World Scout Jamboree



brought a large number of people together from all over the world, including countries with high incidences of meningococcal disease. This meningococcal outbreak highlighted the potential risk of IMD outbreaks in mass gatherings even in low incidence countries.

DISCUSSION

Meningococcal disease is listed in the International Health Regulations as a disease with potential serious public health impact and rapid international spread.⁶ In 2000 and 2001, meningococcal outbreaks caused by serogroup W were reported in England and France, respectively, both related to travellers to the Hajj.⁷

The AR reported in the 23rd WSJ (240.4 cases per 100 000 in Ishizuchi subcamp and 19.5 per 100 000 for all WSJ) far exceeded the annual incidence rate in Japan in 2014 (0.03 per 100 000 population),⁵ which was lower than that for the United States of America (0.3 per 100 000 population in 2009), Europe (0.9 per 100 000 population in 2009) and Australia (1.2 per 100 000 population in 2009).⁸ One IMD outbreak has been reported in Japan in recent decades.⁹ The carriage rate of *N. meningitidis* in the nasopharynx has been reported as 0.4% in Japan,¹⁰ which is lower than that for other countries.

The risk of transmission of meningococcal disease can increase with close and prolonged contact, such as among household members, or with kissing or sharing food or drinking utensils with patients and carriers.³

Although there was no information available about close personal contact among participants, the close living environment and events of WSJ, such as discotheques and campfires, may have increased the risk of spreading meningococcal disease. Even when the incidence of IMD is low in the host country, a mass gathering produces special circumstances that can lead to IMD outbreaks among participants.

In the United Kingdom, serogroup B has been responsible for the majority of IMD cases, as well as for most European countries over the past decades;¹¹ however, serogroup W has increased rapidly since 2009 and accounted for 25% of all IMD cases in England in 2014 and 2015.¹¹ The strain associated with this outbreak was indistinguishable from the strain that has been circulating in England and Scotland recently.

A total of 77 IMD cases were reported to the Japanese National Epidemiological Surveillance of Infectious Disease system, between 25 March 2013 and 26 July 2015. Among reported cases, four were serogroup W.^{5,12} Genetic analysis of these strains, however, revealed that they were not identical to the WSJ-associated strain. Based on these findings, we speculate that the index case was carrying *N. meningitidis* before attending WSJ or acquired meningococcal from someone who was a carrier at the event.

Until Scottish authorities notified Japan about the case, Japanese authorities were not aware of this outbreak because there were no domestic IMD cases

related to WSJ. Once Scottish authorities notified Japan, it was able to begin a risk assessment for Japanese participants and the general public. This situation highlights the importance of international information sharing. Information on epidemiological investigation and gene analysis from other countries is essential to understanding the outbreak and response in a correct and timely manner.¹³

In an outbreak, it is recommended that prophylaxis be given to all close contacts of a case and that people identified as high risk be vaccinated.^{3,14,15} Tetravalent meningococcal conjugate vaccines against groups A, C, Y and W (MCV4) have been available in Japan since May 2015 and could provide an effective measure to prevent IMD outbreaks at mass gatherings in Japan. Currently, however, MCV4 is not included in the routine vaccination schedule due to low incidence of IMD. MCV4 vaccination of high-risk groups merits serious consideration for protecting against this potentially fatal disease with documented international transmission.

A mass gathering produces special circumstances that can lead to IMD outbreaks among participants even in low incidence countries. Keys to an early and effective response include identifying potential risks, raising awareness among all participants, enhancing surveillance and strengthening communication among participant countries. Prophylaxis is recommended for all close contacts, and vaccination is an available prevention and control measure.

Conflicts of Interest

None declared.

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References

1. The 23rd World Scout Jamboree. Tokyo: Scout Association of Japan; 2014 (<http://www.23wsj.jp/about-23wsj.html>, accessed 30 June 2016).
2. Lucidarme J, Scott KJ, Ure R, Smith A, Lindsay D, Stenmark B, et al. An international invasive meningococcal disease outbreak due to a novel and rapidly expanding serogroup W strain, Scotland and Sweden, July to August 2015. *Euro Surveill.* 2016 Nov 10;21(45):30395. PMID:27918265 doi:10.2807/1560-7917.ES.2016.21.45.30395
3. Outbreak of invasive meningococcal disease in the EU associated with a mass gathering, the 23rd World Scout Jamboree, in Japan. Stockholm: European Centre for Disease Prevention and Control; 2015 (<http://ecdc.europa.eu/en/publications/Publications/Meningococcal-disease-scouts-EU-August-2015.pdf>, accessed 30 June 2016).
4. Smith-Palmer A, Oates K, Webster D, Taylor S, Scott KJ, Smith G, et al. IMT and investigation team in Sweden. Outbreak of *Neisseria meningitidis* capsular group W among scouts returning from the World Scout Jamboree, Japan, 2015. *Euro Surveill.* 2016 Nov 10;21(45):30392. PMID:27918267 doi:10.2807/1560-7917.ES.2016.21.45.30392
5. Trends in invasive meningococcal disease, week 13, 2013 to week 52, 2014, Japan. Tokyo: National Institute of Infectious Diseases; 2015 (<http://www.nih.go.jp/niid/ja/bac-meningitis-m/bac-meningitis-iasrs/5864-pr4271.html>, accessed 30 June 2016).
6. International Health Regulations (2005) Second Edition. Geneva: World Health Organization; 2008 (http://whqlibdoc.who.int/publications/2008/9789241580410_eng.pdf, accessed 30 June 2016).
7. Shafi S, Booy R, Haworth E, Rashid H, Memish ZA. Hajj: health lessons for mass gatherings. *J Infect Public Health.* 2008;1(1):27–32. PMID:20701842 doi:10.1016/j.jiph.2008.08.008
8. Halperin SA, Bettinger JA, Greenwood B, Harrison LH, Jelfs J, Ladhani SN, et al. The changing and dynamic epidemiology of meningococcal disease. *Vaccine.* 2012 May 30;30 Suppl 2:B26–36. PMID:22178525 doi:10.1016/j.vaccine.2011.12.032
9. Fukusumi M, Kamiya H, Takahashi H, Kanai M, Hachisu Y, Saitoh T, et al. National surveillance for meningococcal disease in Japan, 1999–2014. *Vaccine.* 2016 Jul 25;34(34):4068–71. PMID:27291085 doi: 10.1016/j.vaccine.2016.06.018.
10. Tanaka H, Kuroki T, Watanabe Y, Asai Y, Ootani K, Sugama K, et al. [Isolation of *Neisseria meningitidis* from healthy persons in Japan]. *Kansenshogaku Zasshi.* 2005 Aug;79(8):527–33. PMID:16167783 doi:10.11150/kansenshogakuzasshi1970.79.527
11. Campbell H, Saliba V, Borrow R, Ramsay M, Ladhani SN. Targeted vaccination of teenagers following continued rapid endemic expansion of a single meningococcal group W clone (sequence type 11 clonal complex), United Kingdom 2015. *Euro Surveill.* 2015 07 16;20(28):21188. PMID:26212140 doi:10.2807/1560-7917.ES2015.20.28.21188
12. Meningococcal disease cases in Scotland and Sweden, following attendance at the World Scout Jamboree, Yamaguchi, Japan, July 28–August 8, 2015. Tokyo: National Institute of Infectious Diseases; 2015 (<http://www.nih.go.jp/niid/en/id/997-disease-based/sa/bac-meningitis/idsc/iasr-in/5879-pr4272e.html>, accessed 30 June 2016).

13. Public Health for Mass Gatherings. Key Considerations. Geneva: World Health Organization; 2015 (http://www.who.int/ihr/publications/WHO_HSE_GCR_2015.5/en/, accessed 25 January 2017).
14. Cohn AC, MacNeil JR, Clark TA, Ortega-Sanchez IR, Briere EZ, Meissner HC, et al.; Centers for Disease Control and Prevention (CDC). Prevention and control of meningococcal disease: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep*. 2013 Mar 22;62 RR-2:1–28. PMID:23515099
15. Immunisation against infectious disease and Children's health. Meningococcal: the green book, chapter 22. London: Public Health England; 2013 (<https://www.gov.uk/government/publications/meningococcal-the-green-book-chapter-22>, accessed 5 April 2017).



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