

NSW PUBLIC HEALTH BULLETIN

Year in Review 2008

Year in review: communicable disease surveillance, NSW, 2008

*Communicable Diseases Branch,
NSW Department of Health*

In this issue, we present our annual review of notifiable diseases reported in New South Wales (NSW) residents. For greater depth of detail, refer to Tables 2–6, which show disease-specific data reported by: year of onset; month of onset; area health service (AHS); and age group and sex.

Trends

Among the 53 573 notifications of medical conditions by doctors, hospital staff and laboratory staff in NSW residents in 2008, highlights included:

Conditions most frequently reported

- Chlamydia: 14 043 cases (201 per 100 000 population), with the highest crude rates by geographical area in the Greater Western (Broken Hill region), South Eastern Sydney Illawarra (Randwick region), Sydney South West (Camperdown region) and Hunter New England (Tamworth region) AHSs.
- Pertussis: 8756 cases (126 per 100 000 population), with the highest crude rates in the North Coast (Lismore region), Sydney West (Penrith and Parramatta regions), South Eastern Sydney Illawarra (Wollongong region) and Greater Western (Dubbo region) AHSs.
- Hepatitis C: 3916 cases (56 per 100 000 population), with the highest crude rates in the Greater Western (Broken Hill and Dubbo regions), North Coast (Lismore region) and Sydney South West (Camperdown region) AHSs.
- Hepatitis B: 2638 cases (38 per 100 000 population) with the highest crude rates in the Sydney South West (Camperdown and Liverpool regions) and Sydney West (Parramatta region) AHSs.
- *Salmonella* infection: 2263 cases (32 per 100 000 population) with the highest crude rates in the

North Coast (Lismore region) and Northern Sydney Central Coast (Gosford and Hornsby regions) AHSs.

Conditions with the most meaningful declines in the number of notifications compared with previous years

- Hepatitis A: cases have more than halved in number since 2002 (69 cases in 2008 compared with 149 in 2002 and 421 in 1999). This may be due in part to the introduction of a commercial vaccine in the 1990s. Travel to countries where Hepatitis A is endemic was the most commonly reported risk factor for disease acquisition in 2008.
- Hepatitis C: cases have decreased by over 50% in number in the last 10 years (3916 cases in 2008 compared with 8598 cases in 1999). The cause of this decline is unclear. It may reflect a real decrease in transmission related to prevention programs, or it may reflect a decrease in hepatitis C testing.
- Meningococcal serogroup C disease: notifications continue to decline (nine cases reported for 2008), largely due to the introduction of meningococcal C vaccination in late 2003.
- Meningococcal serogroup B disease: notifications have decreased steadily over the past few years. In 2008, there were 49 cases reported, compared with 103 in 2002. The reason for this decrease is unclear.
- Rubella: notifications have decreased from 191 cases in 2000 to 17 cases in 2008. This may be due to higher rates of immunisation over the past decade.

Conditions with the most meaningful increases in the number of notifications compared with previous years

- Pertussis has shown the greatest increase in the number of infections, up from 2100 in 2007 to 8756 in 2008;

Table 1. The five most commonly reported notifiable diseases by age group, NSW, 2008

| Age group | Rate/100 000 |
|---|--------------|
| Children under 5 years | |
| 1. Pertussis | 264 |
| 2. <i>Salmonella</i> infection | 120 |
| 3. Giardiasis | 100 |
| 4. Influenza | 55 |
| 5. Cryptosporidiosis | 38 |
| Children and young adults (5–24 years) | |
| 1. Chlamydia** | 437 |
| 2. Pertussis | 187 |
| 3. <i>Salmonella</i> infection | 34 |
| 4. Hepatitis C | 24 |
| 5. Hepatitis B | 22 |
| Adults (25–44 years) | |
| 1. Chlamydia* | 265 |
| 2. Hepatitis C | 105 |
| 3. Pertussis | 92 |
| 4. Hepatitis B | 72 |
| 5. Gonorrhoea | 39 |
| Adults (45–64 years) | |
| 1. Pertussis | 91 |
| 2. Hepatitis C | 71 |
| 3. Hepatitis B | 38 |
| 4. Chlamydia* | 37 |
| 5. Influenza | 23 |
| Older adults (≥65 years) | |
| 1. Pertussis | 70 |
| 2. Influenza | 36 |
| 3. <i>Salmonella</i> infection | 25 |
| 4. Arboviral infection | 22 |
| 5. Pneumococcal disease | 21 |
| Totals | |
| 1. Chlamydia* | 201 |
| 2. Pertussis | 126 |
| 3. Hepatitis C | 56 |
| 4. Hepatitis B | 38 |
| 5. <i>Salmonella</i> infection | 32 |

*refers to *Chlamydia trachomatis* infections.

**where a case is reported in a child under 16 years of age, the relevant public health unit contacts the treating doctor outlining his/her obligation to notify the Department of Community Services.

Source: NSW Notifiable Diseases Database.

this reflects a large, statewide outbreak that continues in 2009, as well as improved diagnostic technology.

- Chlamydia has been reported at the highest rate since it became a notifiable disease in 1998 (14 043 cases in 2008), reflecting a long-standing trend of increases in notifications of this disease.
- The number of Ross River virus notifications increased from 844 in 2007 to 1155 in 2008. This is consistent

with past cyclical fluctuations in Ross River virus activity.

- The number of *Salmonella* infections showed a small decline compared with the previous year (2263 in 2008 compared with 2555 in 2007), but numbers remain high compared with the 10-year average.
- The number of verotoxigenic *Escherichia coli* infections remained higher than usual, with 17 cases reported in 2008, compared with an average of four cases per year for the 10-year period prior to 2007. All cases were investigated and no epidemiological links were identified.
- The number of cases of infectious syphilis remained at comparatively high levels in 2008, reflecting an outbreak among men who have sex with men residing in inner-Sydney.

Conditions least frequently reported

There were no reported cases of anthrax, avian influenza, botulism, chancroid, diphtheria, lyssavirus, plague, polio, severe acute respiratory syndrome (SARS), smallpox, typhus, viral haemorrhagic fever or yellow fever in NSW in 2008. One case of tetanus was reported.

Top five notifiable diseases

Rates for the most commonly reported notifiable diseases for each age group and geographical area of residence at the time of notification are presented in Figure 1 and Table 1. These lists indicate the relative importance of notifiable diseases only and should not be used to indicate the spread of all infectious diseases in NSW. It should also be noted that these rates are heavily influenced by testing practices and, in many instances, do not necessarily indicate the true or relative incidence in the community. Finally, these lists do not include institutional gastro-intestinal outbreaks because comprehensive demographic data are not collected for such outbreaks.

Geographical distribution of notifiable diseases

- *Chlamydia trachomatis* infection was the most commonly reported infection across NSW, with the highest rates observed in rural areas, followed by regional and metropolitan areas.
- The rate of pertussis infections was highest in rural areas, particularly in northern NSW, followed by metropolitan and regional areas.
- Rates of hepatitis C infection were comparable across rural, regional and metropolitan areas. Most of these cases represent chronic infection rather than acute hepatitis C acquisition and as such may not accurately reflect the recent spread of hepatitis C in the community.
- Arboviral infections were more commonly reported in people residing in rural and regional areas than in metropolitan areas.
- Tuberculosis was most frequently reported in metropolitan areas, and was rare in rural regions.

Table 2. Disease notifications by year of onset of illness^a, NSW, 1991–2008

| Condition | 1991 | 1992 | 1993 | 1994 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 | 2001 | 2002 | 2003 | 2004 | 2005 | 2006 | 2007 | 2008 | | |
|--|------------------------------------|------|------|------|------|------|------|------|------|------|------|------|------|-------|-------|-------|-------|-------|-------|-------|
| Adverse event after immunisation | 9 | 31 | 23 | 40 | 28 | 56 | 70 | 95 | 16 | 42 | 111 | 178 | 219 | 187 | 107 | 71 | 234 | 248 | | |
| Anthrax | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | | |
| Arboviral infection | 408 | 343 | 656 | 381 | 539 | 1227 | 1806 | 783 | 1220 | 980 | 1191 | 665 | 1023 | 1152 | 1090 | 1917 | 1500 | 1851 | | |
| Barmah Forest virus ^b | 6 | 6 | 25 | 39 | 271 | 172 | 185 | 134 | 249 | 197 | 401 | 396 | 451 | 405 | 450 | 644 | 573 | 533 | | |
| Ross River virus ^b | 297 | 324 | 599 | 331 | 236 | 1031 | 1598 | 583 | 952 | 750 | 717 | 183 | 493 | 703 | 584 | 1221 | 844 | 1155 | | |
| Other ^b | 105 | 13 | 32 | 11 | 32 | 24 | 23 | 66 | 19 | 33 | 73 | 86 | 79 | 44 | 56 | 52 | 83 | 163 | | |
| Blood lead level ≥ 15 µg/dL^b | Not notifiable until December 1996 | | | | | | 710 | 874 | 691 | 984 | 513 | 516 | 338 | 303 | 234 | 298 | 292 | 260 | | |
| Botulism | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | | |
| Brucellosis^b | 2 | 2 | 4 | 4 | 2 | 1 | 3 | 3 | 2 | 1 | 1 | 2 | 3 | 7 | 3 | 10 | 4 | 2 | | |
| Chancroid^b | Not notifiable until December 1998 | | | | | | | | | | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| Chlamydia trachomatis infection | | | | | | | | | 2469 | 3511 | 4500 | 5823 | 7788 | 10035 | 11288 | 12059 | 12461 | 14043 | | |
| Congenital chlamydia ^b | Not notifiable until August 1998 | | | | | | | | | | 14 | 18 | 16 | 15 | 23 | 28 | 46 | 39 | 31 | 39 |
| Chlamydia – other ^b | Not notifiable until August 1998 | | | | | | | | | | 2455 | 3493 | 4484 | 5808 | 7765 | 10007 | 11242 | 12020 | 12430 | 14004 |
| Cholera^b | 1 | 0 | 1 | 0 | 1 | 3 | 1 | 1 | 2 | 0 | 1 | 1 | 0 | 1 | 0 | 3 | 2 | 2 | | |
| Creutzfeldt-Jakob disease^b | Not notifiable until April 2004 | | | | | | | | | | | | | | 6 | 8 | 10 | 7 | 6 | |
| Cryptosporidiosis^b | Not notifiable until December 1996 | | | | | | 157 | 1130 | 121 | 134 | 195 | 306 | 203 | 357 | 849 | 779 | 545 | 484 | | |
| Foodborne illness (NOS)^e | 2765 | 253 | 106 | 213 | 270 | 211 | 255 | 201 | 151 | 147 | 56 | 41 | 1071 | 550 | 309 | 507 | 763 | 667 | | |
| Gastroenteritis (institutional) | 158 | 406 | 443 | 296 | 1359 | 554 | 939 | 738 | 673 | 697 | 775 | 1752 | 3583 | 12784 | 1395 | 10641 | 10488 | 10135 | | |
| Giardiasis^b | Not notifiable until August 1998 | | | | | | | | | | 1091 | 979 | 967 | 863 | 1028 | 1235 | 1449 | 1725 | 1946 | 1783 |
| Gonorrhoea^b | 392 | 491 | 382 | 357 | 428 | 522 | 636 | 1054 | 1291 | 1060 | 1364 | 1526 | 1329 | 1443 | 1580 | 1738 | 1383 | 1332 | | |
| Haemolytic uraemic syndrome | Not notifiable until December 1996 | | | | | | 3 | 6 | 11 | 9 | 2 | 7 | 5 | 9 | 11 | 11 | 13 | 17 | | |
| Haemophilus influenzae serotype b | 212 | 217 | 124 | 61 | 29 | 13 | 17 | 11 | 13 | 8 | 7 | 10 | 6 | 5 | 7 | 11 | 7 | 9 | | |
| Hib epiglottitis ^b | 15 | 57 | 32 | 21 | 6 | 2 | 5 | 1 | 2 | 2 | 1 | 1 | 0 | 3 | 0 | 1 | 1 | 1 | | |
| Hib meningitis ^b | 48 | 103 | 53 | 17 | 11 | 4 | 3 | 3 | 3 | 1 | 1 | 1 | 0 | 0 | 2 | 0 | 2 | 2 | | |
| Hib septicaemia ^b | 11 | 26 | 24 | 12 | 8 | 3 | 1 | 4 | 6 | 4 | 2 | 3 | 1 | 2 | 4 | 6 | 2 | 3 | | |
| Hib infection NOS ^b | 138 | 31 | 15 | 11 | 4 | 4 | 8 | 3 | 2 | 1 | 3 | 5 | 5 | 0 | 1 | 4 | 2 | 3 | | |
| Hepatitis A^b | 1119 | 901 | 579 | 585 | 614 | 958 | 1426 | 927 | 421 | 201 | 197 | 149 | 124 | 137 | 83 | 95 | 65 | 69 | | |
| Hepatitis B | 1492 | 3169 | 3603 | 3983 | 4007 | 3504 | 3167 | 2957 | 3508 | 3972 | 4555 | 3546 | 2845 | 2811 | 2744 | 2513 | 2637 | 2638 | | |
| Hepatitis B – acute viral ^b | 409 | 112 | 95 | 74 | 61 | 43 | 53 | 58 | 77 | 100 | 94 | 88 | 74 | 53 | 56 | 53 | 56 | 46 | | |
| Hepatitis B – other ^b | 1083 | 3057 | 3508 | 3909 | 3946 | 3461 | 3114 | 2899 | 3431 | 3872 | 4461 | 3458 | 2771 | 2758 | 2688 | 2460 | 2581 | 2592 | | |
| Hepatitis C | 850 | 3895 | 5896 | 7818 | 6878 | 6999 | 6926 | 7206 | 8598 | 8295 | 8650 | 6692 | 5246 | 4916 | 4365 | 4397 | 4210 | 3916 | | |
| Hepatitis C – acute viral ^b | 22 | 26 | 22 | 16 | 32 | 18 | 19 | 112 | 111 | 222 | 295 | 152 | 127 | 59 | 43 | 55 | 65 | 24 | | |
| Hepatitis C – other ^b | 828 | 3869 | 5874 | 7802 | 6846 | 6981 | 6907 | 7094 | 8487 | 8073 | 8355 | 6540 | 5119 | 4857 | 4322 | 4342 | 4145 | 3892 | | |
| Hepatitis D^b | 0 | 8 | 12 | 19 | 19 | 9 | 11 | 3 | 14 | 12 | 11 | 9 | 12 | 14 | 15 | 15 | 11 | 14 | | |
| Hepatitis E^b | 0 | 0 | 1 | 2 | 0 | 3 | 6 | 4 | 7 | 9 | 6 | 6 | 6 | 8 | 7 | 10 | 8 | 14 | | |
| HIV infection^b | 824 | 693 | 589 | 503 | 536 | 449 | 423 | 404 | 377 | 350 | 341 | 394 | 412 | 403 | 391 | 367 | 390 | 322 | | |
| Influenza | | | | | | | | | | | 244 | 1012 | 861 | 1011 | 1414 | 617 | 1918 | 1813 | | |
| Influenza – Type A ^b | Not notifiable until December 2000 | | | | | | | | | | 216 | 770 | 767 | 797 | 1055 | 421 | 1488 | 744 | | |
| Influenza – Type B ^b | Not notifiable until December 2000 | | | | | | | | | | 27 | 241 | 55 | 161 | 280 | 150 | 180 | 971 | | |
| Influenza – Type A & B ^b | Not available until December 2003 | | | | | | | | | | | | | 26 | 65 | 37 | 43 | 81 | | |
| Influenza – Type NOS ^b | Not notifiable until December 2000 | | | | | | | | | | | | | 1 | 39 | 27 | 14 | 9 | 207 | |
| Legionellosis | 37 | 104 | 66 | 60 | 75 | 74 | 33 | 46 | 41 | 41 | 68 | 44 | 60 | 80 | 89 | 78 | 105 | 89 | | |
| <i>L. longbeachae</i> ^b | 0 | 14 | 13 | 8 | 16 | 30 | 9 | 19 | 12 | 12 | 29 | 21 | 37 | 27 | 24 | 22 | 29 | 51 | | |
| <i>L. pneumophila</i> ^b | 16 | 80 | 34 | 30 | 35 | 34 | 18 | 22 | 22 | 26 | 38 | 22 | 23 | 51 | 64 | 55 | 74 | 37 | | |
| Legionnaires' disease – other | 21 | 10 | 19 | 22 | 24 | 10 | 6 | 5 | 7 | 3 | 1 | 1 | 0 | 2 | 1 | 1 | 2 | 1 | | |
| Leprosy | 1 | 7 | 5 | 3 | 3 | 2 | 0 | 0 | 1 | 2 | 4 | 0 | 2 | 5 | 1 | 1 | 4 | 4 | | |
| Leptospirosis^b | 28 | 21 | 16 | 14 | 6 | 33 | 33 | 50 | 56 | 54 | 66 | 39 | 39 | 40 | 35 | 17 | 9 | 17 | | |
| Listeriosis^b | 11 | 13 | 12 | 10 | 14 | 22 | 23 | 28 | 22 | 18 | 12 | 11 | 28 | 30 | 25 | 26 | 22 | 34 | | |
| Lymphogranuloma venereum (LGV)^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 1 | 0 | 1 | | |
| Malaria^b | 171 | 110 | 174 | 184 | 96 | 204 | 173 | 158 | 174 | 233 | 157 | 105 | 121 | 101 | 206 | 140 | 98 | 116 | | |
| Measles | 495 | 805 | 2348 | 1484 | 596 | 191 | 273 | 119 | 32 | 36 | 31 | 8 | 18 | 12 | 5 | 60 | 4 | 39 | | |
| Measles – laboratory confirmed | 19 | 76 | 460 | 302 | 138 | 35 | 98 | 19 | 13 | 22 | 18 | 6 | 14 | 11 | 4 | 48 | 4 | 34 | | |
| Measles – other | 476 | 729 | 1888 | 1182 | 458 | 156 | 175 | 100 | 19 | 14 | 13 | 2 | 4 | 1 | 1 | 12 | 0 | 5 | | |
| Meningococcal disease | 128 | 121 | 153 | 142 | 113 | 161 | 218 | 186 | 221 | 253 | 234 | 216 | 202 | 149 | 140 | 107 | 112 | 81 | | |
| Meningococcal – serogroup B ^b | 0 | 3 | 7 | 7 | 23 | 36 | 53 | 55 | 95 | 93 | 90 | 105 | 100 | 81 | 73 | 54 | 76 | 49 | | |
| Meningococcal – serogroup C ^b | 0 | 4 | 6 | 9 | 8 | 35 | 55 | 55 | 60 | 64 | 38 | 54 | 45 | 24 | 16 | 13 | 10 | 9 | | |
| Meningococcal – serogroup W135 ^b | 0 | 0 | 0 | 0 | 1 | 0 | 2 | 4 | 4 | 4 | 2 | 2 | 2 | 5 | 8 | 5 | 2 | 5 | | |
| Meningococcal – serogroup Y ^b | 0 | 0 | 1 | 1 | 0 | 1 | 0 | 7 | 1 | 7 | 2 | 2 | 5 | 3 | 3 | 1 | 5 | 4 | | |
| Meningococcal – other | 128 | 114 | 139 | 125 | 81 | 89 | 108 | 65 | 61 | 85 | 102 | 53 | 50 | 36 | 40 | 34 | 19 | 14 | | |
| Mumps^b | 8 | 23 | 13 | 11 | 14 | 27 | 29 | 39 | 33 | 92 | 28 | 29 | 35 | 65 | 111 | 155 | 323 | 77 | | |
| Paratyphoid^{b,d} | 20 | 8 | 9 | 11 | 12 | 15 | 5 | 9 | 5 | 14 | 11 | 13 | 22 | 10 | 0 | 0 | 0 | 0 | | |
| Pertussis | 49 | 217 | 1534 | 1405 | 1369 | 1156 | 4246 | 2309 | 1416 | 3692 | 4439 | 2011 | 2772 | 3568 | 5811 | 4921 | 2100 | 8756 | | |
| Pneumococcal disease (invasive)^b | Not notifiable until December 2000 | | | | | | | | | | 444 | 862 | 802 | 906 | 641 | 565 | 523 | 548 | | |
| Psittacosis^b | Not notifiable until December 2000 | | | | | | | | | | 38 | 155 | 87 | 81 | 121 | 94 | 35 | 41 | | |
| Q fever^b | 167 | 213 | 403 | 267 | 201 | 287 | 258 | 236 | 164 | 132 | 144 | 310 | 288 | 223 | 143 | 176 | 205 | 164 | | |
| Rubella | 60 | 324 | 1186 | 233 | 2376 | 636 | 153 | 78 | 46 | 191 | 58 | 35 | 24 | 18 | 10 | 37 | 9 | 17 | | |
| Congenital rubella ^b | 1 | 0 | 2 | 4 | 1 | 5 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | | |
| Rubella – other ^b | 59 | 324 | 1184 | 229 | 2375 | 631 | 153 | 78 | 45 | 191 | 58 | 35 | 23 | 17 | 10 | 37 | 8 | 17 | | |
| Salmonella infection^{b,d} | 1170 | 802 | 980 | 1101 | 1366 | 1224 | 1698 | 1812 | 1438 | 1401 | 1644 | 2100 | 1838 | 2137 | 2176 | 2060 | 2555 | 2263 | | |
| Shigellosis^b | Not notifiable until December 2000 | | | | | | | | | | 134 | 85 | 59 | 96 | 135 | 75 | 71 | 109 | | |
| Syphilis | 580 | 871 | 730 | 961 | 833 | 662 | 510 | 610 | 584 | 580 | 547 | 645 | 839 | 1041 | 840 | 891 | 1094 | 1034 | | |
| Congenital syphilis | 1 | 1 | 0 | 2 | 6 | 3 | 3 | 0 | 3 | 2 | 1 | 1 | 3 | 1 | 6 | 4 | 4 | 2 | | |
| Infectious syphilis ^{b,c} | 1 | 3 | 6 | 29 | 132 | 72 | 57 | 45 | 86 | 80 | 67 | 128 | 244 | 302 | 242 | 234 | 460 | 416 | | |
| Syphilis – other ^b | 578 | 867 | 724 | 930 | 695 | 587 | 450 | 565 | 495 | 498 | 479 | 516 | 592 | 738 | 592 | 653 | 630 | 616 | | |
| Tetanus | 5 | 2 | 5 | 4 | 0 | 1 | 3 | 3 | 1 | 3 | 0 | 0 | 1 | 1 | 1 | | | | | |

Table 3. Disease notifications by month of onset of illness^a, NSW, 2008

| Condition | Jan. | Feb. | Mar. | Apr. | May | Jun. | Jul. | Aug. | Sep. | Oct. | Nov. | Dec. | Total |
|--|------|------|------|------|------|------|------|------|------|------|------|------|-------|
| Adverse event after immunisation | 12 | 38 | 65 | 29 | 28 | 12 | 11 | 10 | 23 | 10 | 8 | 2 | 248 |
| Anthrax | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Arboviral infection | 238 | 312 | 310 | 181 | 132 | 84 | 86 | 89 | 92 | 98 | 125 | 104 | 1851 |
| Barmah Forest virus ^b | 59 | 66 | 96 | 65 | 44 | 26 | 24 | 30 | 28 | 26 | 38 | 31 | 533 |
| Ross River virus ^b | 168 | 233 | 199 | 113 | 76 | 41 | 49 | 44 | 53 | 55 | 71 | 53 | 1155 |
| Other ^b | 11 | 13 | 15 | 3 | 12 | 17 | 13 | 15 | 11 | 17 | 16 | 20 | 163 |
| Blood lead level ≥15 µg/dL^b | 22 | 15 | 16 | 25 | 51 | 15 | 11 | 16 | 25 | 27 | 17 | 20 | 260 |
| Botulism | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Brucellosis^b | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 2 |
| Chancroid^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Chlamydia trachomatis infection | 1160 | 1309 | 1145 | 1239 | 1235 | 1095 | 1225 | 1161 | 1155 | 1160 | 1125 | 1034 | 14043 |
| Congenital chlamydia ^b | 1 | 3 | 3 | 6 | 2 | 2 | 4 | 3 | 3 | 3 | 3 | 6 | 39 |
| Chlamydia – other ^b | 1159 | 1306 | 1142 | 1233 | 1233 | 1093 | 1221 | 1158 | 1152 | 1157 | 1122 | 1028 | 14004 |
| Cholera^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 2 |
| Creutzfeldt-Jakob disease^b | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 1 | 0 | 1 | 2 | 6 |
| Cryptosporidiosis^b | 98 | 54 | 60 | 54 | 32 | 26 | 36 | 24 | 19 | 9 | 26 | 46 | 484 |
| Foodborne illness (NOS)^e | 44 | 79 | 86 | 74 | 46 | 100 | 17 | 43 | 17 | 11 | 75 | 75 | 667 |
| Gastroenteritis (institutional) | 436 | 273 | 338 | 493 | 886 | 937 | 1820 | 2639 | 1095 | 629 | 425 | 164 | 10135 |
| Giardiasis^b | 159 | 198 | 188 | 158 | 186 | 136 | 147 | 152 | 129 | 106 | 106 | 118 | 1783 |
| Gonorrhoea^b | 116 | 113 | 132 | 105 | 110 | 99 | 119 | 122 | 102 | 111 | 107 | 96 | 1332 |
| Haemolytic uraemic syndrome | 0 | 1 | 1 | 3 | 1 | 1 | 1 | 1 | 1 | 0 | 2 | 5 | 17 |
| Haemophilus influenzae serotype b | 0 | 1 | 0 | 1 | 4 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 9 |
| Hib epiglottitis ^b | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Hib meningitis ^b | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| Hib septicaemia ^b | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 3 |
| Hib infection NOS ^b | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 3 |
| Hepatitis A^b | 10 | 7 | 5 | 5 | 1 | 3 | 6 | 5 | 7 | 8 | 4 | 8 | 69 |
| Hepatitis B | 215 | 207 | 203 | 229 | 215 | 182 | 215 | 227 | 212 | 256 | 242 | 235 | 2638 |
| Hepatitis B – acute viral ^b | 0 | 2 | 3 | 6 | 4 | 3 | 3 | 5 | 6 | 5 | 2 | 7 | 46 |
| Hepatitis B – other ^b | 215 | 205 | 200 | 223 | 211 | 179 | 212 | 222 | 206 | 251 | 240 | 228 | 2592 |
| Hepatitis C | 297 | 311 | 287 | 259 | 295 | 339 | 331 | 301 | 374 | 381 | 376 | 365 | 3916 |
| Hepatitis C – acute viral ^b | 0 | 2 | 2 | 0 | 2 | 1 | 3 | 1 | 4 | 2 | 5 | 2 | 24 |
| Hepatitis C – other ^b | 297 | 309 | 285 | 259 | 293 | 338 | 328 | 300 | 370 | 379 | 371 | 363 | 3892 |
| Hepatitis D^b | 0 | 1 | 1 | 1 | 4 | 1 | 0 | 2 | 1 | 2 | 1 | 0 | 14 |
| Hepatitis E^b | 2 | 2 | 1 | 0 | 1 | 0 | 2 | 1 | 1 | 1 | 3 | 0 | 14 |
| HIV infection^b | 31 | 31 | 33 | 22 | 26 | 34 | 27 | 20 | 18 | 29 | 26 | 25 | 322 |
| Influenza | 14 | 26 | 50 | 38 | 74 | 71 | 232 | 423 | 442 | 225 | 128 | 90 | 1813 |
| Influenza – Type A ^b | 7 | 14 | 30 | 23 | 42 | 29 | 74 | 110 | 143 | 129 | 82 | 61 | 744 |
| Influenza – Type B ^b | 4 | 8 | 12 | 10 | 24 | 37 | 147 | 305 | 287 | 83 | 35 | 19 | 971 |
| Influenza – Type A & B ^b | 2 | 2 | 2 | 2 | 6 | 4 | 11 | 8 | 12 | 13 | 10 | 9 | 81 |
| Influenza – Type NOS ^b | 1 | 2 | 6 | 3 | 2 | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 17 |
| Legionellosis | 6 | 9 | 9 | 6 | 12 | 6 | 3 | 7 | 5 | 6 | 10 | 10 | 89 |
| <i>L. longbeachae</i> ^b | 4 | 4 | 4 | 2 | 9 | 3 | 3 | 5 | 4 | 2 | 6 | 5 | 51 |
| <i>L. pneumophila</i> ^b | 2 | 4 | 5 | 4 | 3 | 3 | 0 | 2 | 1 | 4 | 4 | 5 | 37 |
| Legionnaires' disease – other | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Leptospirosis | 1 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 4 |
| Leptospirosis^b | 2 | 0 | 2 | 3 | 1 | 2 | 3 | 1 | 0 | 1 | 1 | 1 | 17 |
| Listeriosis^b | 9 | 3 | 4 | 2 | 3 | 1 | 5 | 0 | 3 | 2 | 0 | 2 | 34 |
| Lymphogranuloma venereum (LGV)^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| Malaria^b | 7 | 10 | 10 | 12 | 10 | 10 | 17 | 9 | 5 | 6 | 9 | 11 | 116 |
| Measles | 4 | 7 | 4 | 6 | 14 | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 39 |
| Measles – laboratory confirmed | 4 | 6 | 4 | 4 | 12 | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 34 |
| Measles – other | 0 | 1 | 0 | 2 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 5 |
| Meningococcal disease | 3 | 2 | 3 | 3 | 4 | 11 | 15 | 11 | 15 | 4 | 5 | 5 | 81 |
| Meningococcal – serogroup B ^b | 3 | 1 | 0 | 1 | 2 | 8 | 12 | 7 | 10 | 1 | 3 | 1 | 49 |
| Meningococcal – serogroup C ^b | 0 | 1 | 0 | 0 | 1 | 1 | 1 | 1 | 2 | 0 | 1 | 1 | 9 |
| Meningococcal – serogroup W135 ^b | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 2 | 1 | 0 | 5 |
| Meningococcal – serogroup Y ^b | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 4 |
| Meningococcal – other | 0 | 0 | 2 | 1 | 1 | 1 | 2 | 2 | 2 | 1 | 0 | 2 | 14 |
| Mumps^b | 27 | 13 | 8 | 2 | 5 | 3 | 3 | 2 | 6 | 2 | 4 | 2 | 77 |
| Pertussis | 232 | 204 | 245 | 295 | 351 | 350 | 512 | 564 | 872 | 1340 | 1790 | 2001 | 8756 |
| Pneumococcal disease (invasive)^b | 16 | 18 | 26 | 38 | 52 | 70 | 66 | 80 | 72 | 34 | 42 | 34 | 548 |
| Psittacosis^b | 1 | 2 | 4 | 4 | 6 | 5 | 2 | 7 | 3 | 2 | 1 | 4 | 41 |
| Q fever^b | 12 | 15 | 17 | 10 | 10 | 5 | 14 | 22 | 10 | 14 | 18 | 17 | 164 |
| Rubella | 0 | 0 | 0 | 1 | 2 | 2 | 2 | 2 | 2 | 3 | 1 | 2 | 17 |
| Congenital rubella ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Rubella – other ^b | 0 | 0 | 0 | 1 | 2 | 2 | 2 | 2 | 2 | 3 | 1 | 2 | 17 |
| Salmonella infection^{b,d} | 226 | 239 | 285 | 248 | 192 | 104 | 145 | 120 | 110 | 136 | 215 | 243 | 2263 |
| Shigellosis^b | 6 | 9 | 7 | 7 | 5 | 6 | 6 | 10 | 16 | 10 | 15 | 12 | 109 |
| Syphilis | 70 | 98 | 92 | 91 | 88 | 79 | 83 | 79 | 81 | 105 | 81 | 87 | 1034 |
| Congenital syphilis | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 2 |
| Infectious syphilis ^{b,c} | 28 | 49 | 40 | 36 | 35 | 39 | 30 | 31 | 30 | 35 | 31 | 32 | 416 |
| Syphilis – other ^b | 42 | 49 | 52 | 55 | 52 | 40 | 53 | 47 | 51 | 70 | 50 | 55 | 616 |
| Tetanus | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Tuberculosis^b | 62 | 44 | 41 | 52 | 31 | 29 | 46 | 40 | 55 | 32 | 30 | 26 | 488 |
| Typhoid^b | 3 | 4 | 3 | 3 | 8 | 1 | 1 | 4 | 2 | 5 | 4 | 5 | 43 |
| Verotoxin-producing Escherichia coli infections^b | 3 | 2 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 0 | 2 | 6 | 19 |

^aYear of onset; the earlier of patient reported onset date, specimen date or date of notification. ^bLaboratory-confirmed cases only. ^cIncludes Syphilis primary, Syphilis secondary, Syphilis <1-year duration and Syphilis newly acquired. ^dIncludes all paratyphoid cases. ^eFoodborne illness cases are only those notified as part of an outbreak. NOS: not otherwise specified. No case of the following diseases have been notified since 1991: Plague^b, Diphtheria^b, Granuloma inguinale^b, Lyssavirus^b, Poliomyelitis^b, Rabies, Smallpox, Typhus^b, Viral haemorrhagic fever, Yellow fever.

Table 4. Disease notifications by area health service of residence (including breakdown by 2005 AHS boundaries), crude rates per 100 000 population, NSW, 2008

| Condition | Greater Southern ^f | | Greater Western ^f | | | Hunter New England ^f | | North Coast ^f | |
|---|-------------------------------|----------|------------------------------|-------|----------|---------------------------------|----------|--------------------------|---------|
| | Albury | Goulburn | Broken Hill | Dubbo | Bathurst | Newcastle | Tamworth | Port Macquarie | Lismore |
| Adverse event after immunisation | 5.6 | 6.2 | 4.4 | 3.9 | 10.4 | 2.4 | 2.8 | 0.7 | 3.5 |
| Anthrax | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Arboviral infection | 65.5 | 21.8 | 150.8 | 122.2 | 24.8 | 57.7 | 67.3 | 75.3 | 131.5 |
| Barmah Forest virus ^b | 3.4 | 6.7 | 22.2 | 8.7 | 0.6 | 19.8 | 11.8 | 41.4 | 66.4 |
| Ross River virus ^b | 61.8 | 13.3 | 128.6 | 112.6 | 23.1 | 36.7 | 52.7 | 32.5 | 61.9 |
| Other ^b | 0.4 | 1.9 | 0.0 | 1.0 | 1.2 | 1.2 | 2.8 | 1.4 | 3.1 |
| Blood lead level $\geq 5 \mu\text{g/dL}^b$ | 4.1 | 1.0 | 33.3 | 77.0 | 6.3 | 6.3 | 1.7 | 0.3 | 2.4 |
| Botulism | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Brucellosis ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.6 | 0.0 | 0.0 |
| Chancroid ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| <i>Chlamydia trachomatis</i> infection | 184.6 | 146.7 | 399.1 | 229.0 | 193.1 | 260.5 | 276.3 | 148.1 | 228.5 |
| Congenital chlamydia ^b | 1.5 | 0.0 | 0.0 | 0.0 | 0.6 | 1.4 | 1.1 | 0.0 | 0.0 |
| Chlamydia – other ^b | 183.1 | 146.7 | 399.1 | 229.0 | 192.5 | 259.1 | 275.2 | 148.1 | 228.5 |
| Cholera ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Creutzfeldt-Jakob disease ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.3 | 0.0 | 0.0 | 0.0 |
| Cryptosporidiosis ^b | 6.0 | 1.4 | 0.0 | 5.8 | 11.0 | 5.6 | 11.2 | 4.5 | 9.4 |
| Foodborne illness (NOS) ^e | 20.6 | 0.0 | 0.0 | 0.0 | 0.0 | 14.8 | 0.0 | 26.7 | 0.0 |
| Gastroenteritis (institutional) | 72.6 | 90.2 | 59.9 | 85.6 | 207.5 | 225.7 | 22.4 | 13.3 | 84.2 |
| Giardiasis ^b | 15.0 | 18.0 | 11.1 | 35.6 | 20.8 | 29.0 | 18.5 | 10.3 | 4.2 |
| Gonorrhoea ^b | 4.1 | 2.9 | 6.7 | 9.6 | 1.7 | 17.4 | 4.5 | 4.8 | 11.1 |
| Haemolytic uraemic syndrome | 0.0 | 0.5 | 0.0 | 1.0 | 0.0 | 0.3 | 0.0 | 0.7 | 0.0 |
| <i>Haemophilus influenzae</i> serotype b | 0.0 | 0.0 | 0.0 | 0.0 | 0.6 | 0.2 | 0.0 | 0.0 | 0.0 |
| Hib epiglottitis ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Hib meningitis ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.6 | 0.0 | 0.0 | 0.0 | 0.0 |
| Hib septicaemia ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.2 | 0.0 | 0.0 | 0.0 |
| Hib infection NOS ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Hepatitis A ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.2 | 0.0 | 0.0 | 0.4 |
| Hepatitis B | 8.6 | 13.8 | 46.6 | 9.6 | 2.9 | 9.9 | 7.9 | 5.5 | 6.3 |
| Hepatitis B – acute viral ^b | 0.4 | 1.4 | 6.7 | 2.9 | 0.6 | 0.9 | 0.0 | 0.0 | 0.7 |
| Hepatitis B – other ^b | 8.2 | 12.3 | 39.9 | 6.7 | 2.3 | 9.0 | 7.9 | 5.5 | 5.6 |
| Hepatitis C | 52.8 | 49.8 | 82.0 | 59.7 | 47.3 | 56.3 | 37.6 | 49.6 | 74.4 |
| Hepatitis C – acute viral ^b | 0.8 | 0.5 | 0.0 | 3.9 | 0.0 | 1.0 | 0.0 | 0.3 | 0.0 |
| Hepatitis C – other ^b | 52.1 | 49.4 | 82.0 | 55.8 | 47.3 | 55.3 | 37.6 | 49.3 | 74.4 |
| Hepatitis D ^b | 0.0 | 0.0 | 0.0 | 1.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Hepatitis E ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| HIV infection ^b | 1.1 | 1.4 | 0.0 | 1.0 | 1.2 | 1.7 | 1.7 | 2.7 | 1.4 |
| Influenza | 31.5 | 36.1 | 11.1 | 20.2 | 17.3 | 30.5 | 29.7 | 16.8 | 66.8 |
| Influenza – Type A ^b | 13.5 | 18.0 | 4.4 | 8.7 | 9.2 | 8.9 | 11.2 | 5.1 | 20.2 |
| Influenza – Type B ^b | 17.2 | 17.1 | 6.7 | 11.6 | 6.3 | 21.7 | 17.9 | 9.6 | 40.4 |
| Influenza – Type A & B ^b | 0.0 | 1.0 | 0.0 | 0.0 | 1.7 | 0.0 | 0.6 | 2.1 | 2.1 |
| Influenza – Type NOS ^b | 0.8 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 4.2 |
| Legionellosis | 1.1 | 1.4 | 0.0 | 0.0 | 2.3 | 1.5 | 1.7 | 1.0 | 1.1 |
| <i>L. longbeachae</i> ^b | 0.8 | 1.0 | 0.0 | 0.0 | 1.7 | 1.0 | 1.1 | 0.7 | 0.7 |
| <i>L. pneumophila</i> ^b | 0.4 | 0.5 | 0.0 | 0.0 | 0.6 | 0.5 | 0.6 | 0.3 | 0.4 |
| Legionnaires' disease – other | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Leprosy | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Leptospirosis ^b | 0.0 | 1.0 | 0.0 | 3.9 | 0.0 | 0.9 | 0.0 | 0.3 | 1.0 |
| Listeriosis ^b | 0.0 | 1.4 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.4 |
| Lymphogranuloma venereum (LGV) ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Malaria ^b | 1.1 | 1.9 | 0.0 | 1.0 | 0.0 | 0.9 | 1.1 | 1.7 | 0.4 |
| Measles | 0.0 | 0.5 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Measles – laboratory confirmed | 0.0 | 0.5 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Measles – other | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Meningococcal disease | 1.9 | 1.9 | 0.0 | 2.9 | 1.2 | 1.0 | 1.1 | 0.0 | 1.1 |
| Meningococcal – serogroup B ^b | 1.1 | 1.0 | 0.0 | 1.9 | 0.6 | 1.0 | 1.1 | 0.0 | 0.4 |
| Meningococcal – serogroup C ^b | 0.4 | 0.0 | 0.0 | 1.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.4 |
| Meningococcal – serogroup W135 ^b | 0.0 | 0.5 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Meningococcal – serogroup Y ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Meningococcal – other | 0.4 | 0.5 | 0.0 | 0.0 | 0.6 | 0.0 | 0.0 | 0.0 | 0.4 |
| Mumps ^b | 0.0 | 0.0 | 0.0 | 1.9 | 0.6 | 0.2 | 0.0 | 0.0 | 0.4 |
| Pertussis | 114.6 | 96.8 | 119.7 | 177.1 | 74.9 | 82.4 | 47.6 | 73.6 | 295.0 |
| Pneumococcal disease (invasive) ^b | 8.2 | 8.1 | 15.5 | 10.6 | 13.3 | 10.9 | 5.6 | 3.8 | 6.6 |
| Psittacosis ^b | 1.9 | 0.0 | 0.0 | 2.9 | 2.9 | 1.0 | 0.0 | 1.0 | 0.4 |
| Q fever ^b | 4.9 | 6.2 | 20.0 | 15.4 | 1.2 | 2.1 | 16.8 | 7.5 | 9.4 |
| Rubella | 0.4 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Congenital rubella ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Rubella – other ^b | 0.4 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| <i>Salmonella</i> infection ^{b,d} | 28.1 | 23.7 | 33.3 | 27.9 | 20.8 | 33.6 | 37.0 | 30.8 | 48.4 |
| Shigellosis ^b | 0.4 | 0.5 | 0.0 | 0.0 | 1.2 | 0.0 | 0.6 | 0.7 | 1.4 |
| Syphilis | 2.6 | 5.7 | 73.2 | 6.7 | 2.3 | 4.1 | 4.5 | 5.1 | 8.7 |
| Congenital syphilis | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Infectious syphilis ^{b,c} | 1.5 | 0.5 | 2.2 | 1.0 | 0.0 | 1.0 | 1.1 | 0.3 | 2.4 |
| Syphilis – other ^b | 1.1 | 5.2 | 71.0 | 5.8 | 2.3 | 3.1 | 3.4 | 4.8 | 6.3 |
| Tetanus | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.3 | 0.0 |
| Tuberculosis ^b | 3.4 | 1.0 | 0.0 | 0.0 | 0.0 | 2.2 | 0.6 | 2.7 | 1.4 |
| Typhoid ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.7 |
| Verotoxin-producing <i>Escherichia coli</i> infections ^b | 0.4 | 0.0 | 0.0 | 1.0 | 0.0 | 1.0 | 1.7 | 0.0 | 0.4 |

^aYear of onset: the earlier of patient reported onset date, specimen date or date of notification. ^bLaboratory-confirmed cases only. ^cIncludes Syphilis primary, Syphilis secondary, Syphilis <1-year duration and Syphilis newly acquired. ^dIncludes all paratyphoid cases. ^eFoodborne illness cases are only those notified as part of an outbreak. ^fAHS further divided into the geographical region covered by their component Public Health Unit. NOS: not otherwise specified. No case of the following diseases have been notified since 1991: Plague^b, Diphtheria^b, Granuloma inguinale^b, Lyssavirus^b, Poliomyelitis^b, Rabies, Smallpox, Typhus^b, Viral haemorrhagic fever, Yellow fever.

Table 4. (Continued)

| Condition | Northern Sydney Central Coast ^f | | South Eastern Sydney Illawarra ^f | | Sydney South West ^f | | Sydney West ^f | | Justice Health |
|--|---|---------|--|----------|--------------------------------|-----------|--------------------------|------------|-------------------|
| | Gosford | Hornsby | Wollongong | Randwick | Camperdown | Liverpool | Penrith | Parramatta | |
| Adverse event after immunisation | 4.8 | 2.3 | 4.2 | 3.0 | 2.3 | 2.9 | 5.6 | 4.5 | 0.0 |
| Anthrax | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Arboviral infection | 17.5 | 5.8 | 13.5 | 5.6 | 4.9 | 1.9 | 9.7 | 6.7 | 0.0 |
| Barmah Forest virus ^b | 4.8 | 0.6 | 2.7 | 0.2 | 0.8 | 0.0 | 0.6 | 0.3 | 0.0 |
| Ross River virus ^b | 11.7 | 2.1 | 9.3 | 1.3 | 2.1 | 1.1 | 6.9 | 2.7 | 0.0 |
| Other ^b | 1.0 | 3.1 | 1.6 | 4.0 | 2.1 | 0.8 | 2.2 | 3.7 | 0.0 |
| Blood lead level $\geq 15 \mu\text{g/dL}^b$ | 1.0 | 0.6 | 1.3 | 1.8 | 1.7 | 3.3 | 5.0 | 1.5 | 0.0 |
| Botulism | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Brucellosis ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 |
| Chancroid ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Chlamydia trachomatis infection | 198.6 | 143.4 | 174.3 | 300.8 | 279.9 | 127.9 | 148.3 | 140.7 | 1913.0 |
| Congenital chlamydia ^b | 0.3 | 0.3 | 0.5 | 0.2 | 0.8 | 0.6 | 0.0 | 1.0 | 0.0 |
| Chlamydia – other ^b | 198.3 | 143.2 | 173.8 | 300.6 | 279.2 | 127.3 | 148.3 | 139.6 | 1913.0 |
| Cholera ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.2 | 0.0 | 0.3 | 0.0 | 0.0 |
| Creutzfeldt-Jakob disease ^b | 0.3 | 0.1 | 0.3 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 |
| Cryptosporidiosis ^b | 7.6 | 9.0 | 3.7 | 12.3 | 3.0 | 3.5 | 10.3 | 7.3 | 0.0 |
| Foodborne illness (NOS) ^e | 21.6 | 0.0 | 20.1 | 0.0 | 15.4 | 0.0 | 64.5 | 0.0 | 175.0 |
| Gastroenteritis (institutional) | 211.9 | 199.1 | 103.2 | 115.7 | 305.3 | 49.2 | 192.9 | 172.7 | 0.0 |
| Giardiasis ^b | 25.7 | 40.2 | 23.6 | 41.2 | 23.9 | 14.3 | 31.2 | 24.6 | 25.0 |
| Gonorrhoea ^b | 8.3 | 15.7 | 8.5 | 55.4 | 47.0 | 10.1 | 10.6 | 13.3 | 62.5 |
| Haemolytic uraemic syndrome | 0.0 | 0.3 | 0.3 | 0.0 | 0.4 | 0.5 | 0.3 | 0.1 | 0.0 |
| Haemophilus influenzae serotype b | 0.6 | 0.0 | 0.0 | 0.1 | 0.2 | 0.1 | 0.6 | 0.0 | 0.0 |
| Hib epiglottitis ^b | 0.3 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Hib meningitis ^b | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Hib septicaemia ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 | 0.3 | 0.0 | 0.0 |
| Hib infection NOS ^b | 0.3 | 0.0 | 0.0 | 0.0 | 0.2 | 0.0 | 0.3 | 0.0 | 0.0 |
| Hepatitis A ^b | 0.6 | 2.0 | 1.6 | 0.7 | 1.5 | 1.2 | 0.3 | 2.3 | 0.0 |
| Hepatitis B | 9.2 | 36.0 | 14.6 | 48.6 | 83.1 | 66.0 | 12.2 | 69.5 | 587.5 |
| Hepatitis B – acute viral ^b | 0.0 | 0.5 | 0.5 | 0.7 | 1.3 | 0.5 | 0.3 | 0.3 | 12.5 |
| Hepatitis B – other ^b | 9.2 | 35.5 | 14.0 | 47.9 | 81.8 | 65.5 | 11.8 | 69.2 | 575.0 |
| Hepatitis C | 56.5 | 21.2 | 46.0 | 49.4 | 60.0 | 52.0 | 43.0 | 35.6 | 7250.0 |
| Hepatitis C – acute viral ^b | 0.0 | 0.0 | 0.3 | 0.1 | 0.0 | 0.1 | 0.0 | 0.3 | 62.5 |
| Hepatitis C – other ^b | 56.5 | 21.2 | 45.8 | 49.3 | 60.0 | 51.8 | 43.0 | 35.3 | 7188.0 |
| Hepatitis D ^b | 0.0 | 0.0 | 0.0 | 0.2 | 0.0 | 0.4 | 0.3 | 0.5 | 37.5 |
| Hepatitis E ^b | 0.0 | 0.1 | 0.3 | 0.1 | 0.4 | 0.4 | 0.3 | 0.6 | 0.0 |
| HIV infection ^b | 1.9 | 3.1 | 0.8 | 14.1 | 14.1 | 2.0 | 2.2 | 3.5 | 0.0 |
| Influenza | 9.8 | 12.3 | 23.5 | 19.4 | 16.0 | 16.8 | 42.7 | 47.0 | 37.5 |
| Influenza – Type A ^b | 3.5 | 5.1 | 11.6 | 6.7 | 9.8 | 6.4 | 22.1 | 20.4 | 25.0 |
| Influenza – Type B ^b | 6.0 | 6.8 | 10.6 | 11.2 | 6.0 | 10.3 | 12.5 | 24.6 | 12.5 |
| Influenza – Type A & B ^b | 0.0 | 0.4 | 1.3 | 1.6 | 0.2 | 0.0 | 8.1 | 1.9 | 0.0 |
| Influenza – Type NOS ^b | 0.3 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 |
| Legionellosis | 1.0 | 0.9 | 1.6 | 1.0 | 0.8 | 1.1 | 2.2 | 2.2 | 0.0 |
| <i>L. longbeachae</i> ^b | 0.6 | 0.6 | 1.1 | 0.5 | 0.6 | 0.2 | 1.6 | 0.9 | 0.0 |
| <i>L. pneumophila</i> ^b | 0.3 | 0.3 | 0.5 | 0.4 | 0.2 | 0.8 | 0.6 | 1.3 | 0.0 |
| Legionnaires' disease – other | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Leprosy | 0.0 | 0.0 | 0.0 | 0.0 | 0.2 | 0.1 | 0.0 | 0.3 | 0.0 |
| Leptospirosis ^b | 0.0 | 0.0 | 0.3 | 0.0 | 0.2 | 0.0 | 0.0 | 0.0 | 0.0 |
| Listeriosis ^b | 0.3 | 0.4 | 0.5 | 0.6 | 1.1 | 0.6 | 0.6 | 0.8 | 0.0 |
| Lymphogranuloma venereum (LGV) ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.2 | 0.0 | 0.0 | 0.0 | 0.0 |
| Malaria ^b | 0.6 | 2.1 | 2.7 | 1.2 | 3.0 | 0.6 | 1.6 | 2.9 | 0.0 |
| Measles | 0.0 | 0.4 | 0.3 | 0.5 | 0.8 | 2.1 | 0.9 | 0.6 | 0.0 |
| Measles – laboratory confirmed | 0.0 | 0.4 | 0.3 | 0.5 | 0.6 | 1.8 | 0.9 | 0.5 | 0.0 |
| Measles – other | 0.0 | 0.0 | 0.0 | 0.0 | 0.2 | 0.4 | 0.0 | 0.1 | 0.0 |
| Meningococcal disease | 1.0 | 1.1 | 1.9 | 1.2 | 0.4 | 1.3 | 2.5 | 0.8 | 0.0 |
| Meningococcal – serogroup B ^b | 0.6 | 0.5 | 1.1 | 0.6 | 0.4 | 0.8 | 1.6 | 0.4 | 0.0 |
| Meningococcal – serogroup C ^b | 0.0 | 0.3 | 0.3 | 0.1 | 0.0 | 0.1 | 0.0 | 0.1 | 0.0 |
| Meningococcal – serogroup W135 ^b | 0.0 | 0.0 | 0.0 | 0.2 | 0.0 | 0.1 | 0.3 | 0.0 | 0.0 |
| Meningococcal – serogroup Y ^b | 0.0 | 0.0 | 0.5 | 0.1 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 |
| Meningococcal – other | 0.3 | 0.4 | 0.0 | 0.1 | 0.0 | 0.1 | 0.6 | 0.3 | 0.0 |
| Mumps ^b | 0.6 | 1.0 | 1.1 | 3.5 | 1.9 | 0.8 | 0.3 | 1.3 | 0.0 |
| Pertussis | 125.6 | 123.6 | 187.0 | 121.9 | 83.7 | 68.0 | 195.0 | 191.0 | 25.0 |
| Pneumococcal disease (invasive) ^b | 9.8 | 6.7 | 7.1 | 6.3 | 7.5 | 6.9 | 8.1 | 9.7 | 0.0 |
| Psittacosis ^b | 0.3 | 0.1 | 0.0 | 0.2 | 0.2 | 0.6 | 2.2 | 0.1 | 0.0 |
| Q fever ^b | 1.3 | 0.0 | 2.9 | 0.2 | 0.0 | 0.0 | 0.0 | 0.3 | 12.5 |
| Rubella | 0.3 | 0.7 | 0.0 | 0.2 | 0.6 | 0.4 | 0.0 | 0.1 | 0.0 |
| Congenital rubella ^b | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Rubella – other ^b | 0.3 | 0.7 | 0.0 | 0.2 | 0.6 | 0.4 | 0.0 | 0.1 | 0.0 |
| Salmonella infection ^{b,d} | 44.1 | 42.3 | 25.4 | 32.2 | 31.2 | 25.9 | 29.6 | 28.8 | 0.0 |
| Shigellosis ^b | 0.3 | 1.5 | 1.3 | 5.2 | 3.2 | 1.2 | 0.0 | 1.0 | 0.0 |
| Syphilis | 7.9 | 7.5 | 7.9 | 35.4 | 38.7 | 15.1 | 9.4 | 13.3 | 187.5 |
| Congenital syphilis | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 |
| Infectious syphilis ^{b,c} | 1.3 | 3.0 | 1.6 | 24.3 | 19.7 | 2.1 | 2.2 | 3.2 | 12.5 |
| Syphilis – other ^b | 6.7 | 4.6 | 6.4 | 10.9 | 19.0 | 13.0 | 7.2 | 10.0 | 175.0 |
| Tetanus | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Tuberculosis ^b | 1.6 | 6.5 | 1.9 | 8.5 | 13.9 | 10.5 | 4.4 | 17.4 | 0.0 |
| Typhoid ^b | 0.0 | 0.3 | 0.0 | 0.6 | 1.5 | 1.2 | 0.0 | 2.1 | 0.0 |
| Verotoxin-producing <i>Escherichia coli</i> infections ^b | 0.3 | 0.1 | 0.0 | 0.1 | 0.4 | 0.1 | 0.3 | 0.0 | 0.0 |

^aYear of onset: the earlier of patient reported onset date, specimen date or date of notification. ^bLaboratory-confirmed cases only. ^cIncludes Syphilis primary, Syphilis secondary, Syphilis <1-year duration and Syphilis newly acquired. ^dIncludes all paratyphoid cases. ^eFoodborne illness cases are only those notified as part of an outbreak. ^fAHS further divided into the geographical region covered by their component Public Health Unit. NOS: not otherwise specified. No case of the following diseases have been notified since 1991: Plague^b, Diphtheria^b, Granuloma inguinale^b, Lyssavirus^b, Poliomyelitis^b, Rabies, Smallpox, Typhus^b, Viral haemorrhagic fever, Yellow fever.

Table 5. Disease notifications by area health service of residence (including breakdown by 2005 AHS boundaries) of case, NSW, 2008

| Condition | Greater Southern ^f | | Greater Western ^f | | | Hunter New England ^f | | North Coast ^f | |
|---|-------------------------------|----------|------------------------------|-------|----------|---------------------------------|----------|--------------------------|---------|
| | Albury | Goulburn | Broken Hill | Dubbo | Bathurst | Newcastle | Tamworth | Port Macquarie | Lismore |
| Adverse event after immunisation | 15 | 13 | 2 | 4 | 18 | 14 | 5 | 2 | 10 |
| Anthrax | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Arboviral infection | 175 | 46 | 68 | 127 | 43 | 338 | 120 | 220 | 378 |
| Barmah Forest virus ^b | 9 | 14 | 10 | 9 | 1 | 116 | 21 | 121 | 191 |
| Ross River virus ^b | 165 | 28 | 58 | 117 | 40 | 215 | 94 | 95 | 178 |
| Other ^b | 1 | 4 | 0 | 1 | 2 | 7 | 5 | 4 | 9 |
| Blood lead level $\geq 15 \mu\text{g/dL}^b$ | 11 | 2 | 15 | 80 | 11 | 37 | 3 | 1 | 7 |
| Botulism | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Brucellosis ^b | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| Chancroid ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| <i>Chlamydia trachomatis</i> infection | 493 | 309 | 180 | 238 | 335 | 1527 | 493 | 433 | 657 |
| Congenital chlamydia ^b | 4 | 0 | 0 | 0 | 1 | 8 | 2 | 0 | 0 |
| Chlamydia – other ^b | 489 | 309 | 180 | 238 | 334 | 1519 | 491 | 433 | 657 |
| Cholera ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Creutzfeldt-Jakob disease ^b | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 |
| Cryptosporidiosis ^b | 16 | 3 | 0 | 6 | 19 | 33 | 20 | 13 | 27 |
| Foodborne illness (NOS) ^e | 55 | 0 | 0 | 0 | 0 | 87 | 0 | 78 | 0 |
| Gastroenteritis (institutional) | 194 | 190 | 27 | 89 | 360 | 1323 | 40 | 39 | 242 |
| Giardiasis ^b | 40 | 38 | 5 | 37 | 36 | 170 | 33 | 30 | 12 |
| Gonorrhoea ^b | 11 | 6 | 3 | 10 | 3 | 102 | 8 | 14 | 32 |
| Haemolytic uraemic syndrome | 0 | 1 | 0 | 1 | 0 | 2 | 0 | 2 | 0 |
| <i>Haemophilus influenzae</i> serotype b | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 |
| Hib epiglottitis ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Hib meningitis ^b | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Hib septicaemia ^b | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Hib infection NOS ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Hepatitis A ^b | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| Hepatitis B | 23 | 29 | 21 | 10 | 5 | 58 | 14 | 16 | 18 |
| Hepatitis B – acute viral ^b | 1 | 3 | 3 | 3 | 1 | 5 | 0 | 0 | 2 |
| Hepatitis B – other ^b | 22 | 26 | 18 | 7 | 4 | 53 | 14 | 16 | 16 |
| Hepatitis C | 141 | 105 | 37 | 62 | 82 | 330 | 67 | 145 | 214 |
| Hepatitis C – acute viral ^b | 2 | 1 | 0 | 4 | 0 | 6 | 0 | 1 | 0 |
| Hepatitis C – other ^b | 139 | 104 | 37 | 58 | 82 | 324 | 67 | 144 | 214 |
| Hepatitis D ^b | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| Hepatitis E ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| HIV infection ^b | 3 | 3 | 0 | 1 | 2 | 10 | 3 | 8 | 4 |
| Influenza | 84 | 76 | 5 | 21 | 30 | 179 | 53 | 49 | 192 |
| Influenza – Type A ^b | 36 | 38 | 2 | 9 | 16 | 52 | 20 | 15 | 58 |
| Influenza – Type B ^b | 46 | 36 | 3 | 12 | 11 | 127 | 32 | 28 | 116 |
| Influenza – Type A & B ^b | 0 | 2 | 0 | 0 | 3 | 0 | 1 | 6 | 6 |
| Influenza – Type NOS ^b | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 12 |
| Legionellosis | 3 | 3 | 0 | 0 | 4 | 9 | 3 | 3 | 3 |
| <i>L. longbeachae</i> ^b | 2 | 2 | 0 | 0 | 3 | 6 | 2 | 2 | 2 |
| <i>L. pneumophila</i> ^b | 1 | 1 | 0 | 0 | 1 | 3 | 1 | 1 | 1 |
| Legionnaires' disease – other | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Leprosy | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Leptospirosis ^b | 0 | 2 | 0 | 4 | 0 | 5 | 0 | 1 | 3 |
| Listeriosis ^b | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Lymphogranuloma venereum (LGV) ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malaria ^b | 3 | 4 | 0 | 1 | 0 | 5 | 2 | 5 | 1 |
| Measles | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Measles – laboratory confirmed | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Measles – other | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Meningococcal disease | 5 | 4 | 0 | 3 | 2 | 6 | 2 | 0 | 3 |
| Meningococcal – serogroup B ^b | 3 | 2 | 0 | 2 | 1 | 6 | 2 | 0 | 1 |
| Meningococcal – serogroup C ^b | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| Meningococcal – serogroup W135 ^b | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Meningococcal – serogroup Y ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Meningococcal – other | 1 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| Mumps ^b | 0 | 0 | 0 | 2 | 1 | 1 | 0 | 0 | 1 |
| Pertussis | 306 | 204 | 54 | 184 | 130 | 483 | 85 | 215 | 848 |
| Pneumococcal disease (invasive) ^b | 22 | 17 | 7 | 11 | 23 | 64 | 10 | 11 | 19 |
| Psittacosis ^b | 5 | 0 | 0 | 3 | 5 | 6 | 0 | 3 | 1 |
| Q fever ^b | 13 | 13 | 9 | 16 | 2 | 12 | 30 | 22 | 27 |
| Rubella | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Congenital rubella ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Rubella – other ^b | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| <i>Salmonella</i> infection ^{b,d} | 75 | 50 | 15 | 29 | 36 | 197 | 66 | 90 | 139 |
| Shigellosis ^b | 1 | 1 | 0 | 0 | 2 | 0 | 1 | 2 | 4 |
| Syphilis | 7 | 12 | 33 | 7 | 4 | 24 | 8 | 15 | 25 |
| Congenital syphilis | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Infectious syphilis ^{b,c} | 4 | 1 | 1 | 1 | 0 | 6 | 2 | 1 | 7 |
| Syphilis – other ^b | 3 | 11 | 32 | 6 | 4 | 18 | 6 | 14 | 18 |
| Tetanus | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Tuberculosis ^b | 9 | 2 | 0 | 0 | 0 | 13 | 1 | 8 | 4 |
| Typhoid ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| Verotoxin-producing <i>Escherichia coli</i> infections ^b | 1 | 0 | 0 | 1 | 0 | 6 | 3 | 0 | 1 |

^aYear of onset: the earlier of patient reported onset date, specimen date or date of notification. ^bLaboratory-confirmed cases only. ^cIncludes Syphilis primary, Syphilis secondary, Syphilis <1-year duration and Syphilis newly acquired. ^dIncludes all paratyphoid cases. ^eFoodborne illness cases are only those notified as part of an outbreak. ^fAHS further divided into the geographical region covered by their component Public Health Unit. ^gRate is based on a denominator of 8000 persons. ^hIncludes cases with unknown Public Health Unit. NOS: not otherwise specified. No case of the following diseases have been notified since 1991: Plague^b, Diphtheria^b, Granuloma inguinale^b, Lyssavirus^b, Poliomyelitis^b, Rabies, Smallpox, Typhus^b, Viral haemorrhagic fever, Yellow fever.

Table 5. (Continued)

| Condition | Northern Sydney Central Coast ^f | | South Eastern Sydney Illawarra ^f | | Sydney South West ^f | | Sydney West ^f | | Justice Health | Total |
|--|---|---------|--|----------|--------------------------------|-----------|--------------------------|------------|-------------------|-------|
| | Gosford | Hornsby | Wollongong | Randwick | Camperdown | Liverpool | Penrith | Parramatta | | |
| Adverse event after immunisation | 15 | 19 | 16 | 25 | 12 | 24 | 18 | 35 | 0 | 248 |
| Anthrax | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Arboviral infection | 55 | 47 | 51 | 46 | 26 | 16 | 31 | 52 | 0 | 1851 |
| Barmah Forest virus ^b | 15 | 5 | 10 | 2 | 4 | 0 | 2 | 2 | 0 | 533 |
| Ross River virus ^b | 37 | 17 | 35 | 11 | 11 | 9 | 22 | 21 | 0 | 1155 |
| Other ^b | 3 | 25 | 6 | 33 | 11 | 7 | 7 | 29 | 0 | 163 |
| Blood lead level $\geq 15 \mu\text{g/dL}$ ^b | 3 | 5 | 5 | 15 | 9 | 28 | 16 | 12 | 0 | 260 |
| Botulism | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Brucellosis ^b | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 2 |
| Chancroid ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| <i>Chlamydia trachomatis</i> infection | 626 | 1163 | 659 | 2476 | 1489 | 1076 | 476 | 1099 | 153 | 14043 |
| Congenital chlamydia ^b | 1 | 2 | 2 | 2 | 4 | 5 | 0 | 8 | 0 | 39 |
| Chlamydia – other ^b | 625 | 1161 | 657 | 2474 | 1485 | 1071 | 476 | 1091 | 153 | 14004 |
| Cholera ^b | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 2 |
| Creutzfeldt-Jakob disease ^b | 1 | 1 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| Cryptosporidiosis ^b | 24 | 73 | 14 | 101 | 16 | 29 | 33 | 57 | 0 | 484 |
| Foodborne illness (NOS) ^e | 68 | 0 | 76 | 0 | 82 | 0 | 207 | 0 | 14 | 667 |
| Gastroenteritis (institutional) | 668 | 1615 | 390 | 952 | 1624 | 414 | 619 | 1349 | 0 | 10135 |
| Giardiasis ^b | 81 | 326 | 89 | 339 | 127 | 120 | 100 | 192 | 2 | 1783 |
| Gonorrhoea ^b | 26 | 127 | 32 | 456 | 250 | 85 | 34 | 104 | 5 | 1332 |
| Haemolytic uraemic syndrome | 0 | 2 | 1 | 0 | 2 | 4 | 1 | 1 | 0 | 17 |
| <i>Haemophilus influenzae</i> serotype b | 2 | 0 | 0 | 1 | 1 | 1 | 2 | 0 | 0 | 9 |
| Hib epiglottitis ^b | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Hib meningitis ^b | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 2 |
| Hib septicaemia ^b | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 3 |
| Hib infection NOS ^b | 1 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 3 |
| Hepatitis A ^b | 2 | 16 | 6 | 6 | 8 | 10 | 1 | 18 | 0 | 69 |
| Hepatitis B | 29 | 292 | 55 | 400 | 442 | 555 | 39 | 543 | 47 | 2638 |
| Hepatitis B – acute viral ^b | 0 | 4 | 2 | 6 | 7 | 4 | 1 | 2 | 1 | 46 |
| Hepatitis B – other ^b | 29 | 288 | 53 | 394 | 435 | 551 | 38 | 541 | 46 | 2592 |
| Hepatitis C | 178 | 172 | 174 | 407 | 319 | 437 | 138 | 278 | 580 | 3916 |
| Hepatitis C – acute viral ^b | 0 | 0 | 1 | 1 | 0 | 1 | 0 | 2 | 5 | 24 |
| Hepatitis C – other ^b | 178 | 172 | 173 | 406 | 319 | 436 | 138 | 276 | 575 | 3892 |
| Hepatitis D ^b | 0 | 0 | 0 | 2 | 0 | 3 | 1 | 4 | 3 | 14 |
| Hepatitis E ^b | 0 | 1 | 1 | 1 | 2 | 3 | 1 | 5 | 0 | 14 |
| HIV infection ^b | 6 | 25 | 3 | 116 | 75 | 17 | 7 | 27 | 0 | 322 |
| Influenza | 31 | 100 | 89 | 160 | 85 | 141 | 137 | 367 | 3 | 1813 |
| Influenza – Type A ^b | 11 | 41 | 44 | 55 | 52 | 54 | 71 | 159 | 2 | 744 |
| Influenza – Type B ^b | 19 | 55 | 40 | 92 | 32 | 87 | 40 | 192 | 1 | 971 |
| Influenza – Type A & B ^b | 0 | 3 | 5 | 13 | 1 | 0 | 26 | 15 | 0 | 81 |
| Influenza – Type NOS ^b | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 17 |
| Legionellosis | 3 | 7 | 6 | 8 | 4 | 9 | 7 | 17 | 0 | 89 |
| <i>L. longbeachae</i> ^b | 2 | 5 | 4 | 4 | 3 | 2 | 5 | 7 | 0 | 51 |
| <i>L. pneumophila</i> ^b | 1 | 2 | 2 | 3 | 1 | 7 | 2 | 10 | 0 | 37 |
| Legionnaires' disease – other | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 |
| Leprosy | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 2 | 0 | 4 |
| Leptospirosis ^b | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 17 |
| Listeriosis ^b | 1 | 3 | 2 | 5 | 6 | 5 | 2 | 6 | 0 | 34 |
| Lymphogranuloma venereum (LGV) ^b | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| Malaria ^b | 2 | 17 | 10 | 10 | 16 | 5 | 5 | 23 | 0 | 116 |
| Measles | 0 | 3 | 1 | 4 | 4 | 18 | 3 | 5 | 0 | 39 |
| Measles – laboratory confirmed | 0 | 3 | 1 | 4 | 3 | 15 | 3 | 4 | 0 | 34 |
| Measles – other | 0 | 0 | 0 | 0 | 1 | 3 | 0 | 1 | 0 | 5 |
| Meningococcal disease | 3 | 9 | 7 | 10 | 2 | 11 | 8 | 6 | 0 | 81 |
| Meningococcal – serogroup B ^b | 2 | 4 | 4 | 5 | 2 | 7 | 5 | 3 | 0 | 49 |
| Meningococcal – serogroup C ^b | 0 | 2 | 1 | 1 | 0 | 1 | 0 | 1 | 0 | 9 |
| Meningococcal – serogroup W135 ^b | 0 | 0 | 0 | 2 | 0 | 1 | 1 | 0 | 0 | 5 |
| Meningococcal – serogroup Y ^b | 0 | 0 | 2 | 1 | 0 | 1 | 0 | 0 | 0 | 4 |
| Meningococcal – other | 1 | 3 | 0 | 1 | 0 | 1 | 2 | 2 | 0 | 14 |
| Mumps ^b | 2 | 8 | 4 | 29 | 10 | 7 | 1 | 10 | 0 | 77 |
| Pertussis | 396 | 1002 | 707 | 1003 | 445 | 572 | 626 | 1492 | 2 | 8756 |
| Pneumococcal disease (invasive) ^b | 31 | 54 | 27 | 52 | 40 | 58 | 26 | 76 | 0 | 548 |
| Psittacosis ^b | 1 | 1 | 0 | 2 | 1 | 5 | 7 | 1 | 0 | 41 |
| Q fever ^b | 4 | 0 | 11 | 2 | 0 | 0 | 0 | 2 | 1 | 164 |
| Rubella | 1 | 6 | 0 | 2 | 3 | 3 | 0 | 1 | 0 | 17 |
| Congenital rubella ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Rubella – other ^b | 1 | 6 | 0 | 2 | 3 | 3 | 0 | 1 | 0 | 17 |
| <i>Salmonella</i> infection ^{b,d} | 139 | 343 | 96 | 265 | 166 | 218 | 95 | 225 | 0 | 2263 |
| Shigellosis ^b | 1 | 12 | 5 | 43 | 17 | 10 | 0 | 8 | 0 | 109 |
| Syphilis | 25 | 61 | 30 | 291 | 206 | 127 | 30 | 104 | 15 | 1034 |
| Congenital syphilis | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 2 |
| Infectious syphilis ^{b,c} | 4 | 24 | 6 | 200 | 105 | 18 | 7 | 25 | 1 | 416 |
| Syphilis – other ^b | 21 | 37 | 24 | 90 | 101 | 109 | 23 | 78 | 14 | 616 |
| Tetanus | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Tuberculosis ^b | 5 | 53 | 7 | 70 | 74 | 88 | 14 | 136 | 0 | 488 |
| Typhoid ^b | 0 | 2 | 0 | 5 | 8 | 10 | 0 | 16 | 0 | 43 |
| Verotoxin-producing <i>Escherichia coli</i> infections ^b | 1 | 1 | 0 | 1 | 2 | 1 | 1 | 0 | 0 | 19 |

^aYear of onset: the earlier of patient reported onset date, specimen date or date of notification. ^bLaboratory-confirmed cases only. ^cIncludes Syphilis primary, Syphilis secondary, Syphilis <1-year duration and Syphilis newly acquired. ^dIncludes all paratyphoid cases. ^eFoodborne illness cases are only those notified as part of an outbreak. ^fAHS further divided into the geographical region covered by their component Public Health Unit. ^gRate is based on a denominator of 8000 persons. ^hIncludes cases with unknown Public Health Unit. NOS: not otherwise specified. No case of the following diseases have been notified since 1991: Plague^b, Diphtheria^b, Granuloma inguinale^b, Lyssavirus^b, Poliomyelitis^b, Rabies, Smallpox, Typhus^b, Viral haemorrhagic fever, Yellow fever.

- Higher rates of bloodborne diseases and sexually transmissible infections (e.g. chlamydia, syphilis and hepatitis B and C) were reported for Justice Health compared with the rest of NSW. This is likely to be related to testing for these diseases on entry into correctional facilities. Within the prison population, hepatitis C was the most commonly reported infection, likely related to risk factors among people who are incarcerated.

Age distribution of notifiable diseases

- Gastrointestinal and respiratory diseases were most commonly reported in children aged under 5 years. This may be partly due to high testing rates for these diseases in children.
- Pertussis notifications were highest in the group aged 5–24 years, affected both sexes equally, and were also high in females aged 25–44 years, perhaps reflecting increased testing and/or infection of women of child-bearing age.

- Pertussis was also the most commonly reported notifiable disease in adults aged 65 years and older.
- Chlamydia was most common in the group aged 5–24 years, with females accounting for twice as many notifications as males. This is likely to be partly due to higher screening rates for chlamydia in women.

Outbreaks and threats

Several notable disease outbreaks and threats were reported in 2008 in NSW. These included:

- An outbreak of pertussis which first appeared in northern NSW. The highest age-specific incidence was seen in children aged under 1 year.³
- There were five strains of influenza circulating in 2008, with an epidemic of influenza B. An earlier peak of influenza than seen in previous years may have been due to the influx of overseas travellers for World Youth Day in July.

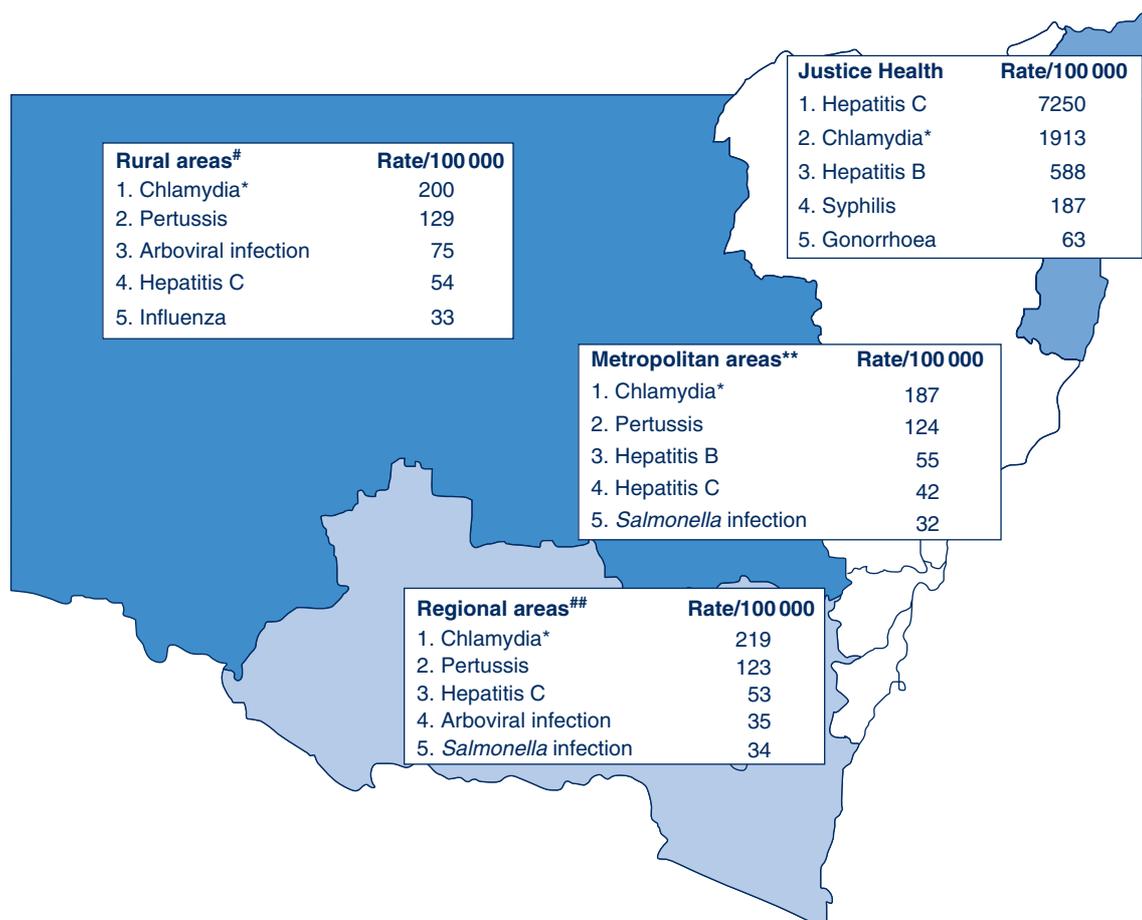


Figure 1. The five most commonly reported notifiable diseases by geographical area of residence at the time of notification in NSW, 2008. [#]Includes Greater Southern, Greater Western, Hunter New England (Tamworth region) and North Coast Area Health Services. ^{##}Includes Northern Sydney Central Coast (Gosford region), South Eastern Sydney Illawarra (Wollongong region) and Hunter New England (Newcastle region) Area Health Services. *Refers to notifications of *Chlamydia trachomatis*. **Includes Northern Sydney Central Coast (Hornsby region), South Eastern Sydney Illawarra (Randwick region), Sydney South West and Sydney West Area Health Services. Source: NSW Notifiable Diseases Database.

Table 6. Disease notifications by age group and sex of case, NSW, 2008

| Condition | 0-4 years | | 5-24 years | | 25-44 years | | 45-64 years | | ≥65 years | | Total | | Total ^a |
|---|-----------|-----|------------|------|-------------|------|-------------|-----|-----------|-----|-------|------|--------------------|
| | F | M | F | M | F | M | F | M | F | M | F | M | |
| Adverse event after immunisation | 23 | 27 | 142 | 4 | 15 | 1 | 21 | 5 | 8 | 0 | 209 | 37 | 248 |
| Anthrax | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Arboviral infection | 3 | 4 | 117 | 95 | 360 | 336 | 342 | 377 | 104 | 109 | 926 | 921 | 1851 |
| Barmah Forest virus ^b | 0 | 0 | 22 | 29 | 95 | 86 | 106 | 128 | 30 | 36 | 253 | 279 | 533 |
| Ross River virus ^b | 2 | 4 | 75 | 56 | 232 | 217 | 218 | 212 | 70 | 66 | 597 | 555 | 1155 |
| Other ^b | 1 | 0 | 20 | 10 | 33 | 33 | 18 | 37 | 4 | 7 | 76 | 87 | 163 |
| Blood lead level ≥15 µg/dL ^b | 9 | 16 | 3 | 35 | 4 | 112 | 4 | 62 | 1 | 12 | 21 | 237 | 260 |
| Botulism | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Brucellosis ^b | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 2 | 2 |
| Chancroid ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Chlamydia trachomatis infection | 32 | 26 | 5438 | 2612 | 2380 | 2850 | 204 | 433 | 7 | 24 | 8061 | 5945 | 14043 |
| Congenital chlamydia ^b | 17 | 17 | 1 | 1 | 1 | 2 | 0 | 0 | 0 | 0 | 19 | 20 | 39 |
| Chlamydia – other ^b | 15 | 9 | 5437 | 2611 | 2379 | 2848 | 204 | 433 | 7 | 24 | 8042 | 5925 | 14004 |
| Cholera ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 2 | 2 |
| Creutzfeldt-Jakob disease ^b | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 0 | 3 | 1 | 5 | 6 |
| Cryptosporidiosis ^b | 73 | 100 | 68 | 72 | 74 | 48 | 19 | 13 | 9 | 7 | 243 | 240 | 484 |
| Giardiasis ^b | 173 | 285 | 159 | 191 | 338 | 252 | 147 | 129 | 60 | 46 | 877 | 903 | 1783 |
| Gonorrhoea ^b | 0 | 1 | 105 | 257 | 109 | 651 | 23 | 169 | 3 | 12 | 240 | 1090 | 1332 |
| Haemolytic uraemic syndrome | 6 | 1 | 1 | 4 | 1 | 0 | 2 | 0 | 1 | 1 | 11 | 6 | 17 |
| Haemophilus influenzae serotype b | 1 | 0 | 0 | 3 | 0 | 1 | 0 | 0 | 2 | 2 | 3 | 6 | 9 |
| Hib epiglottitis ^b | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| Hib meningitis ^b | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 2 |
| Hib septicaemia ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 1 | 2 | 3 |
| Hib infection NOS ^b | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 1 | 0 | 1 | 2 | 3 |
| Hepatitis A ^b | 1 | 1 | 12 | 18 | 9 | 15 | 3 | 3 | 5 | 2 | 30 | 39 | 69 |
| Hepatitis B | 4 | 6 | 187 | 219 | 650 | 766 | 255 | 411 | 55 | 61 | 1151 | 1463 | 2638 |
| Hepatitis B – acute viral ^b | 0 | 0 | 8 | 3 | 1 | 22 | 2 | 5 | 1 | 4 | 12 | 34 | 46 |
| Hepatitis B – other ^b | 4 | 6 | 179 | 216 | 649 | 744 | 253 | 406 | 54 | 57 | 1139 | 1429 | 2592 |
| Hepatitis C | 11 | 10 | 221 | 215 | 738 | 1340 | 420 | 813 | 67 | 61 | 1457 | 2439 | 3916 |
| Hepatitis C – acute viral ^b | 1 | 0 | 3 | 2 | 10 | 6 | 2 | 0 | 0 | 0 | 16 | 8 | 24 |
| Hepatitis C – other ^b | 10 | 10 | 218 | 213 | 728 | 1334 | 418 | 813 | 67 | 61 | 1441 | 2431 | 3892 |
| Hepatitis D ^b | 0 | 0 | 0 | 1 | 0 | 7 | 2 | 4 | 0 | 0 | 2 | 12 | 14 |
| Hepatitis E ^b | 0 | 0 | 2 | 4 | 1 | 4 | 0 | 2 | 1 | 0 | 4 | 10 | 14 |
| HIV infection ^b | 0 | 0 | 8 | 29 | 20 | 192 | 3 | 64 | 1 | 5 | 32 | 290 | 322 |
| Influenza | 113 | 139 | 195 | 198 | 229 | 184 | 243 | 160 | 162 | 181 | 943 | 863 | 1813 |
| Influenza – Type A ^b | 27 | 39 | 69 | 66 | 102 | 76 | 121 | 84 | 75 | 80 | 395 | 345 | 744 |
| Influenza – Type B ^b | 84 | 100 | 122 | 123 | 114 | 100 | 102 | 61 | 74 | 88 | 496 | 473 | 971 |
| Influenza – Type A & B ^b | 2 | 0 | 4 | 7 | 11 | 8 | 15 | 9 | 13 | 12 | 45 | 36 | 81 |
| Influenza – Type NOS ^b | 0 | 0 | 0 | 2 | 2 | 0 | 5 | 6 | 0 | 1 | 7 | 9 | 17 |
| Legionellosis | 0 | 0 | 0 | 0 | 3 | 7 | 15 | 24 | 13 | 27 | 31 | 58 | 89 |
| <i>L. longbeachae</i> ^b | 0 | 0 | 0 | 0 | 2 | 3 | 8 | 14 | 7 | 17 | 17 | 34 | 51 |
| <i>L. pneumophila</i> ^b | 0 | 0 | 0 | 0 | 1 | 4 | 7 | 10 | 5 | 10 | 13 | 24 | 37 |
| Legionnaires' disease – other | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 1 |
| Leprosy | 0 | 0 | 1 | 0 | 0 | 2 | 0 | 0 | 0 | 1 | 1 | 3 | 4 |
| Leptospirosis ^b | 0 | 0 | 1 | 2 | 0 | 5 | 0 | 8 | 0 | 1 | 1 | 16 | 17 |
| Listeriosis ^b | 2 | 2 | 1 | 1 | 5 | 3 | 3 | 5 | 5 | 7 | 16 | 18 | 34 |
| Lymphogranuloma venereum (LGV) ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 |
| Malaria ^b | 1 | 2 | 12 | 24 | 11 | 33 | 6 | 22 | 1 | 3 | 31 | 84 | 116 |
| Measles | 2 | 7 | 7 | 9 | 6 | 8 | 0 | 0 | 0 | 0 | 15 | 24 | 39 |
| Measles – laboratory confirmed | 2 | 6 | 7 | 6 | 6 | 7 | 0 | 0 | 0 | 0 | 15 | 19 | 34 |
| Measles – other | 0 | 1 | 0 | 3 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 5 | 5 |
| Meningococcal disease | 12 | 14 | 15 | 15 | 4 | 6 | 8 | 2 | 3 | 2 | 42 | 39 | 81 |
| Meningococcal – serogroup B ^b | 11 | 8 | 10 | 8 | 3 | 4 | 4 | 1 | 0 | 0 | 28 | 21 | 49 |
| Meningococcal – serogroup C ^b | 0 | 1 | 2 | 2 | 0 | 0 | 1 | 1 | 2 | 0 | 5 | 4 | 9 |
| Meningococcal – serogroup W135 ^b | 1 | 2 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 2 | 3 | 5 |
| Meningococcal – serogroup Y ^b | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 2 | 4 |
| Meningococcal – other | 0 | 3 | 1 | 3 | 1 | 2 | 3 | 0 | 0 | 1 | 5 | 9 | 14 |
| Mumps ^b | 2 | 4 | 9 | 13 | 19 | 23 | 6 | 1 | 0 | 0 | 36 | 41 | 77 |
| Pertussis | 611 | 595 | 1770 | 1682 | 1132 | 685 | 938 | 657 | 399 | 270 | 4850 | 3889 | 8756 |
| Pneumococcal disease (invasive) ^b | 39 | 57 | 16 | 14 | 27 | 47 | 58 | 85 | 108 | 96 | 248 | 299 | 548 |
| Psittacosis ^b | 0 | 0 | 2 | 0 | 5 | 1 | 12 | 14 | 4 | 3 | 23 | 18 | 41 |
| Q fever ^b | 0 | 0 | 5 | 9 | 16 | 42 | 19 | 57 | 4 | 12 | 44 | 120 | 164 |
| Rubella | 1 | 1 | 2 | 1 | 4 | 6 | 0 | 2 | 0 | 0 | 7 | 10 | 17 |
| Congenital rubella ^b | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Rubella – other ^b | 1 | 1 | 2 | 1 | 4 | 6 | 0 | 2 | 0 | 0 | 7 | 10 | 17 |
| Salmonella infection ^{b,d} | 261 | 286 | 302 | 330 | 246 | 235 | 174 | 178 | 136 | 102 | 1119 | 1131 | 2263 |
| Shigellosis ^b | 3 | 7 | 9 | 6 | 10 | 47 | 7 | 18 | 1 | 1 | 30 | 79 | 109 |
| Syphilis | 3 | 1 | 21 | 46 | 113 | 395 | 58 | 240 | 52 | 104 | 247 | 786 | 1034 |
| Congenital syphilis | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 2 |
| Infectious syphilis ^{b,c} | 0 | 0 | 7 | 27 | 11 | 264 | 3 | 95 | 1 | 8 | 22 | 394 | 416 |
| Syphilis – other ^b | 2 | 0 | 14 | 19 | 102 | 131 | 55 | 145 | 51 | 96 | 224 | 391 | 616 |
| Tetanus | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 1 |
| Tuberculosis ^b | 1 | 1 | 49 | 69 | 90 | 100 | 46 | 58 | 29 | 45 | 215 | 273 | 488 |
| Typhoid ^b | 4 | 4 | 10 | 6 | 7 | 9 | 3 | 0 | 0 | 0 | 24 | 19 | 43 |
| Verotoxin-producing <i>Escherichia coli</i> infections ^b | 3 | 1 | 4 | 2 | 1 | 0 | 1 | 3 | 3 | 1 | 12 | 7 | 19 |

^aYear of onset: the earlier of patient reported onset date, specimen date or date of notification. ^bLaboratory-confirmed cases only. ^cIncludes Syphilis primary, Syphilis secondary, Syphilis <1-year duration and Syphilis newly acquired. ^dIncludes all paratyphoid cases. ^eIncludes cases with unknown age and sex and people who identify as transgender. NOS: not otherwise specified. F: female. M: male. Institutional gastrointestinal outbreaks and foodborne illness are excluded from the table as complete demographic data is not routinely collected.

- There were a number of discrete foodborne salmonella outbreaks, several of which were traced back to raw egg products in a range of foods.³
- There were several clusters of measles cases from January 2008, with 38 cases reported between January and June, compared with four cases reported in the same period in 2007.^{1,2} One was associated with an English language school, one was associated with an under-immunised population in the Blue Mountains, and one was associated with transmission in an emergency department.

Conclusions

Controlling the spread of communicable diseases remains a priority for NSW. Vaccine-preventable diseases and sexually transmissible infections are of particular concern. This is exemplified by the re-emergence of infectious syphilis amongst men who have sex with men and the high rates of chlamydia in young adults.

The transmission of vaccine-preventable diseases, including measles and pertussis, also increased in NSW in 2008

compared with previous years. This highlights the challenge of increasing vaccination rates among adolescents and young adults, as well as the importance of promoting and maintaining high vaccination rates in infants.

We thank all those general and specialist medical practices, laboratories, hospitals, schools, child-care centres, and others who have notified diseases of public health significance to their local public health units for investigation and control.

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Investigation of equine influenza transmission in NSW: walk, wind or wing?

**Paula J. Spokes^{A,C}, Andrew J. N. Marich^B,
Jennie A. Musto^B, Kate A. Ward^A,
Adam T. Craig^A and Jeremy M. McAnulty^B**

^ANSW Public Health Officer Training Program,
NSW Department of Health

^BCommunicable Diseases Branch, NSW Department of Health

^CCorresponding author. Email:
paula.spokes@doh.health.nsw.gov.au

Abstract: Objectives: An outbreak of equine influenza occurred in New South Wales in 2007. In addition to the local spread of the disease between bordering properties, windborne spread over several kilometres had been postulated as a possible method of transmission in this outbreak. This study aimed to describe potential modes of transmission for a property infected with equine influenza where no apparent epidemiological links to other infected properties were reported. **Methods:** A semi-structured questionnaire was administered to owners of affected properties. The questionnaire collected detailed transmission-risk information, including personnel movements, equipment sharing, and horse and other animal movements. **Results:** Interviews with property owners from one geographic area suggested the potential for birds and other animals – rather than wind – to facilitate transmission of equine influenza. **Conclusion:** This study described the potential for mechanical spread of equine influenza. Further research, including laboratory testing of bird plumage following contact with infected horses, may be useful to confirm the possibility of avian fomite transmission.

In August 2007, the New South Wales (NSW) Department of Primary Industries (DPI) identified an outbreak of equine influenza (EI) in the Sydney area. More than 5000 properties in NSW were eventually affected by the equine influenza A, H3N8 virus.

While the mode of transmission of EI is incompletely understood, the virus is thought to be transmitted via droplets from infected, coughing horses.¹ The virus can survive on skin, fabrics and surfaces of contaminated equipment, but survival in the air may be reduced in conditions of high relative humidity (Table 1).¹ Animals other than horses are not thought to be epidemiologically significant for the spread of EI.

Infected, coughing horses have been reported to spread the EI virus 35 m and possibly further under favourable air and wind-drift conditions.¹ In an outbreak of EI in 1965, horses segregated 27.4 m away from known infected horses reportedly became infected; however, virus transfer by people or equipment could not be excluded.² Virus spread by humans and fomites may have played a significant role in the spread of EI in the 2007 outbreak in NSW (Table 1).³ An enquiry into the outbreak found that the virus most likely left the Eastern Creek Quarantine Station on the contaminated clothing or equipment of a person who had been in contact with an infected horse.⁴

In addition to the local spread of EI between bordering properties, windborne spread of EI over several kilometres – dependent on atmospheric and climatic conditions – had been postulated as a possible method of transmission in the 2007 outbreak in NSW.⁵ Anecdotal reports of windborne spread over distances of up to 8 km have been suggested during outbreaks in South Africa in 1986 and Jamaica in 1989, although other modes of transmission such as contaminated personnel and equipment could not be excluded.^{6,7} Direct contact with infected horses and contaminated equipment and associated personnel were identified as the most important factors in the rapid spread of EI in the South African outbreak in 1986.⁸

Windborne spread of infection has not been reported for human influenza viruses and, as such, the 2007 EI outbreak presented a unique opportunity to understand the potential for the windborne spread of influenza that may be relevant to both horses and humans.

The NSW *Exotic Diseases of Animals Act 1991* requires anyone with contact with infected horses or horse products (including objects or vehicles) to comply with disinfection guidelines.⁹ Penalties, including fines or imprisonment, apply to people who do not comply. Information was

Table 1. Conditions required for the viability of equine influenza virus on some surfaces

| Surface | Conditions required | Length of virus viability |
|----------------------------|---|---------------------------|
| Fabric/clothing | Humidity of 35–40% Temperature of 28°C | 8–12 hours |
| Stainless steel or plastic | Humidity 35–40% Temperature of 28°C | 24–48 hours |
| Tap water (pH 7.0) | Temperature up to 37°C | 2 days |
| Soil | In dark storage Temperature of 18°C | 24 hours |
| Soil | In direct sunlight Temperature of 15°C | 8 hours |

Source: Animal Health Australia. Disease strategy: Equine influenza (Version 3.0). Australian Veterinary Emergency Plan (AUSVETPLAN), Edition 3. Canberra: Primary Industries Ministerial Council; 2007.

provided to property owners regarding disinfection practices and on-farm biosecurity measures. Property owners who suspected that their horses had been infected with EI were required, under the Act, to contact their local veterinarian or the DPI disease hotline.¹⁰

During the outbreak some infected properties were geographically isolated from known infected properties and restricted areas, and had no apparent epidemiological links to a source of infection. These properties presented an opportunity to explore factors that may have been associated with transmission, including the likelihood of wind-borne spread.

In this study we aimed to describe potential modes of transmission for a property infected with EI where there were no apparent epidemiological links to other infected properties.

Methods

The study area was located on the south-western outskirts of Sydney in NSW. The area was chosen because it contained several infected properties geographically separate from infected properties with known epidemiological links. The area was also located near the Local Disease Control Centre (LDCC) where the investigation team was based.

Epidemiologists from the LDCC and NSW Health reviewed the case-file information to collect onset dates and identify infected properties from the study area. Properties with no known epidemiological links to an infected property were determined through case-note review and discussion with field veterinarians involved in the initial investigation. Outbreak maps developed by DPI using the FrontGate Geographical Information System program were used to locate infected and neighbouring properties.¹¹ Daily weather observations from an airport,

approximately 6 km north-east of the study group, were used as a proxy measure for the area of interest.

A semi-structured questionnaire was developed to collect detailed transmission-risk information including personnel movements, equipment sharing, and horse and other animal movements. The questionnaire was administered to owners of infected properties to identify potential epidemiological links. The questionnaire was administered via a telephone interview or in person at property boundaries because of biosecurity measures and the risk of infection for non-infected properties. One property (property E) was studied in detail because of its apparent geographical separation from other infected properties. Owners of properties with no horses (as reported by neighbours) were not interviewed. Interviews were conducted in October 2007.

Results

Four properties within a 1 km radius in this area (properties A, B, C and D) reported EI infection to the DPI. The first case (property A) reported the onset of symptoms as 4 September 2007. This property was subsequently found to have epidemiological links to Centennial Park, a significant spread site prior to the statewide lockdown of the movement of horses. Other properties within the study area which shared common boundaries with property A subsequently reported infection (Table 2). Property E reported to the LDCC onset of EI symptoms on 10 September. This property was approximately 1 km from the initial case, with no shared borders or reported close contacts with neighbouring horses.

Property E was a stock horse stud property with 16 horses, including two horses kept on the property by another owner. The property owner reported that all 16 horses eventually developed clinical symptoms of EI. Property E was approximately 1 km away from the nearest known

Table 2. Equine influenza in selected study area properties, equine influenza outbreak, NSW, 2007

| Property | Horses | | Onset date |
|----------|--------|--------|------------|
| | n | % sick | |
| A | 75 | 100 | 04/09/2007 |
| B | 11 | 100 | 06/09/2007 |
| C | 6 | 100 | 08/09/2007 |
| D | 67 | 67 | 09/09/2007 |
| E | 16 | 100 | 10/09/2007 |
| F | 8 | 75 | 20/09/2007 |
| G | 1 | 0 | – |
| H | 0 | – | – |
| I | 0 | – | – |
| J | 0 | – | – |

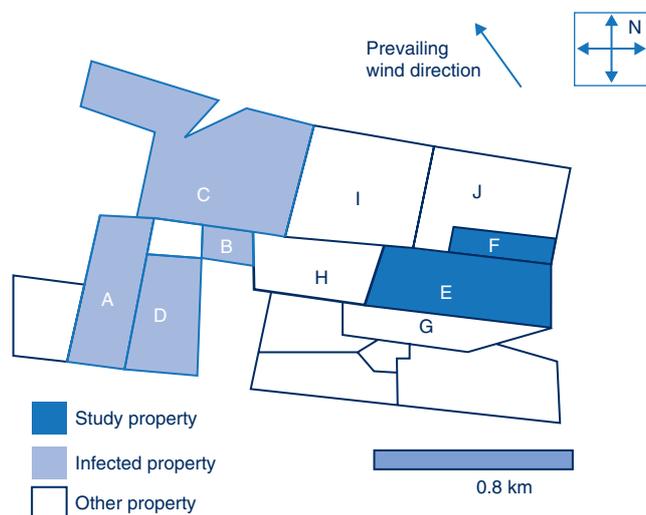


Figure 1. Selected study area properties, equine influenza outbreak, NSW, 2007. Source: NSW Department of Primary Industries. FrontGate Geographical Information System.

infected property. There are five properties surrounding property E. Of these, three (H, I and J) were reported by the owner of property E to have no resident horses (Figure 1).

The owner reported that none of the horses were moved off property E or shared equipment with other properties in the 10 days prior to the onset of symptoms. He reported that visitors to the property, including the owner of the two agisted horses on the property, had no contact with other horses. Other family members living on the property had minimal contact with the horses and reportedly had no contact with other horses outside property E. All horses on property E were fed grain pellets from a local supplier. The owner reported that there had been no deliveries to the

property in the 10 days prior to the onset of symptoms. There was one dog on property E that was reported to visit property F regularly.

The owner stated that he also owned horses on another property 1–2 km away but reported no contact with these horses since the onset of symptoms in horses on property E. The horses on the other property had not displayed any clinical symptoms.

The owner noted that when sick, horses coughed up undigested grain pellets, coughing sputum over the feed. A number of birds had been observed eating this feed and bathing in water troughs. Two dead birds were subsequently seen in the horse yards but were discarded and therefore not available for testing.

Property F first reported symptoms on 20 September (10 days after the onset of illness on property E). The owner reported that six of the eight horses on the property developed clinical symptoms of EI. Property F shared a border with property E, which was likely to have been the source of infection because of its close proximity.

Property G shared a border with property E and had one 30-year-old horse. The horse had not been broken in, no equipment was used (and therefore shared), and the horse had never left the property. The owner onsite had checked the horse daily for clinical symptoms and it was asymptomatic at the time of interview.

Property I reported horses had been kept on the property approximately 12 months before. The owners of properties H and J reported no horses as currently resident onsite.

Daily observations from a weather station approximately 6 km north-east of the study group were used as a proxy measure for the area of interest. Weather conditions during the incubation period for the initial case on property E (estimated from 4 to 10 September) were obtained from the Bureau of Meteorology.¹² The weather station reported rain and easterly and south-easterly winds during this period.

Discussion

This study of geographically separate properties infected with EI with no apparent epidemiological links found that transmission had occurred with a separation of approximately 1 km between known infected properties. Infection still occurred despite the owner on property E reporting implementation of biosecurity measures such as disinfection of equipment and personnel, minimising visitors and their contact with horses and moving horses away from boundary lines.

The owner of property E did not report possible transmission by nose-to-nose contact with infected horses, shared

equipment, or visitors to the property with other horse contacts. However, it is difficult to exclude fomite transmission as the source of infection because of reliance on accurate recall and the legal requirements and penalties that could result from disclosing such information.

Interviews with property owners in the study identified the possibility of mechanical transfer of infection by birds or other animals in the spread of EI. Five property owners (A, B, D, E and H) reported an increased number of birds around properties in recent months that were observed eating horse grain and bathing in water troughs. Birds that ate food in and around feed bins may have been exposed to respiratory secretions from infected horses to become a source of mechanical virus transfer. These birds were not available for testing. The owner of property E hypothesised that the birds travelled between stables looking for food, particularly during the current drought when the usual food supplies were limited.

Studies into foot-and-mouth disease transmission have reported that birds may act as potential fomites for mechanical transfer as respiratory secretions – and consequently virus – adhere to feathers.¹³ The foot-and-mouth disease virus is reportedly able to survive for short periods on the body of animals, including for up to 91 hours on the feathers of live birds.¹⁴ The EI virus has been reported to survive in water and soil for varying time periods dependent on temperature and pH;¹ however we were unable to find data on the survival of the virus on feathers or other animals.

While birds are one potential mode of mechanical transfer of EI, it is possible that dogs or other mammals may also facilitate the spread of the EI virus for a short time and distance in the vicinity of an EI outbreak.

The weather conditions reported during the incubation period for property E (rain and south-easterly winds) indicate that windborne transmission of EI from property A would be unlikely. Research into windborne spread of foot-and-mouth disease found that transmission was reliant on high humidity, low wind speeds, and the absence of heavy rain.¹⁵ Further experimental trials would be necessary to test the feasibility of windborne virus transmission for EI.

There were time delays of approximately 3 weeks between the notification of infection and the interview. This may have resulted in inaccurate recall of information by the property owners. In addition, owners would have been aware of legal requirements of reporting and compliance with biosecurity practices. Owners interviewed in the study were unable to be guaranteed confidentiality, and may have been less likely to report breaches in practice during their interview. Owners of infected properties in only one cluster were interviewed and so these findings may not be generalised.

Because of biosecurity measures implemented on infected properties and the risk of infection to reportedly non-infected properties, interviews were limited to either a telephone call or were conducted at property boundaries. Consequently, interviewers were not able to carry out a physical investigation of the properties. The study was, however, able to use the FrontGate Geographic Information System to ascertain property borders.

Conclusion

The hypothesis-generating questionnaires did not identify epidemiological links between these infected properties but described the potential for mechanical spread via birds or other animals. Further research, including laboratory testing of bird plumage following contact with infected horses, may be useful to confirm the possibility of avian fomite transmission. Additional study of clusters in other areas may be useful to better understand the epidemiological features of EI.

Acknowledgments

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Diagnostic and typing methods for investigating *Legionella* infection

**Christopher C. Blyth^A, D. Nicholas Adams^A
and Sharon C. A. Chen^{A,B}**

^ACentre for Infectious Diseases and Microbiology Laboratory Services, Sydney West Area Health Service

^BCorresponding author. Email: Sharon.chen@swahs.health.nsw.gov.au

Abstract: *Legionella* infection is an important cause of community-acquired pneumonia in Australia. Morbidity and mortality is significant. Diagnosis remains a challenge with infection often unrecognised, particularly early in the course of illness. An understanding of available diagnostic methods and their limitations is important to public health practitioners and clinicians alike.

Legionella infections are responsible for 2–15% of community-acquired pneumonia.^{1,2} Morbidity and mortality varies greatly depending on the underlying health of the patient, the promptness of specific therapy and whether the disease is sporadic, nosocomial or part of an outbreak.³ Outbreaks or case clusters occur in community-acquired and nosocomial settings with cooling towers, spas and contaminated hot and cold water plumbing commonly implicated.¹ *Legionella* infections are notifiable throughout Australia, with approximately 300–350 cases reported each year (data from 2001 to 2007).⁴

Numerous diagnostic methods and the typing of isolates are available to assist with epidemiological investigations. This paper will review these methods and how they can be used by public health practitioners to manage potential cases and suspected outbreaks.

Microbiology and clinical spectrum

Legionella spp. are ubiquitous environmental Gram-negative bacteria. They are able to survive in moist environments for long periods of time and grow well at temperatures ranging from 20 to 42°C.⁵ They have an

increased tolerance to chlorine and thus enter water-supply systems and proliferate in thermal habitats, including air-conditioning towers, hot water systems, shower heads, taps, spas and respiratory ventilators.⁶ There are currently more than 50 species described, including at least 16 serogroups of *L. pneumophila*.⁵

Infections range from a severe multisystem disease including pneumonia to an asymptomatic infection.^{1,5,7} Pneumonia due to *L. pneumophila* is termed Legionnaires' disease. Worldwide, *L. pneumophila* serogroup 1 is the most common cause of Legionnaires' disease. Pneumonia can be caused by other *Legionella* spp.; *L. longbeachae*, *L. bozemanii*, *L. dumoffii* and *L. micdadei* are the most frequently described.^{1,2,5,8,9} Pontiac fever, a self-limiting non-pneumonic febrile illness, is also described.

In the period 1991–2000 in Australia, *L. pneumophila* was responsible for 51% of cases of clinical disease, with *L. pneumophila* serogroup 1 the most frequently reported pathogen.¹⁰ *L. longbeachae* is another frequent pathogen in Australia, responsible for 42% of the total number of cases.¹⁰

Laboratory diagnosis from clinical specimens

It is not possible to distinguish patients with Legionnaires' disease from other forms of pneumonia by clinical or radiological means.^{11,12} As a result, laboratory confirmation is essential for diagnosis. Although diagnostic methods have improved, no currently available test is able to diagnose all *Legionella* infections in a timely fashion, with a high degree of sensitivity and specificity. The available methods are summarised in Table 1.

Definitive legionellosis is defined by the Public Health Laboratory Network as isolation of *Legionella* spp., detection of *Legionella* antigen in urine, seroconversion or significant increase in serum *Legionella* antibody levels.¹³ Suggestive legionellosis is defined as detection of *Legionella* antigen by direct fluorescent antigen (DFA), detection of *Legionella* DNA by polymerase chain reaction (PCR), or a single high antibody level to *L. pneumophila* or *L. longbeachae*.¹³ These laboratory

Table 1. Comparison of different microbiological methods to diagnose *Legionella* infection

| Test | Specimen | Sensitivity (%) | Specificity (%) | Laboratory turnaround time | Comments |
|-------------------|--|-----------------|-----------------|----------------------------|---|
| Culture | Respiratory samples including sputum and BAL | <10–80* | 100 | 3–7 days | Detects all species and serogroups. Species other than <i>L. pneumophila</i> may be detectable only after 10 days of incubation. ⁵ |
| DFA staining | Respiratory samples including sputum and BAL | 25–70* | >95 | <4 hours | Technically demanding. Sensitivity consistently less than for culture. |
| Antigen detection | Urine | 70–90 | >95 | <3 hours | Only reliable for detection of <i>L. pneumophila</i> serogroup 1. |
| PCR | Respiratory samples including sputum and BAL | 80–100 | >90 | <4 hours | Detects all species and serogroups. |
| Serology | Serum | 30–50 | >90 | 3–10 weeks | Must test both acute and convalescent samples. Interpretation of a single sample can be misleading. |
| | Urine | 46–86 | >90 | | |
| | Serum | 60–80 | >95 | | |

BAL: bronchoalveolar lavage.
DFA: direct fluorescent antibody.
PCR: polymerase chain reaction.
*Depends on the severity of disease.
Source: Murdoch DR. Diagnosis of *Legionella* infection. *Clin Infect Dis* 2003;36(1): 64–9.

definitions are used in combination with clinical parameters to identify, for public health purposes, confirmed and probable cases of *Legionella* infection.¹⁴

Culture

Isolation of *Legionella* spp. by culture is considered the ‘gold standard’ for diagnosis because of its superior specificity. *Legionella* spp. are most frequently isolated from respiratory tract specimens (e.g. sputa, bronchoalveolar lavage (BAL), lung). Lung biopsy specimens have the greatest yield but are rarely performed.⁵ Bronchoscopic samples have a greater diagnostic yield compared with expectorated sputum samples.¹⁵ In most laboratories, polyvalent or monoclonal antisera are used to identify presumptive *L. pneumophila* and *L. longbeachae*.¹³ These techniques are unreliable for other species, owing to a high degree of cross-reactivity between different species with molecular techniques preferred.

The major advantage of culture for diagnosis is that all *Legionella* spp. are able to be detected by this method. A culture isolate is also required for further epidemiological typing or for susceptibility testing.

There are, however, inherent problems with *Legionella* culture because the organism is fastidious and slow growing (often taking 5 days or more to grow).¹³ Specifically formulated media (most frequently buffered charcoal

yeast-extract media) are required to enhance the growth of *Legionella* spp. and suppress other respiratory bacteria. Patients with Legionnaires’ disease are often non-productive of sputum and therefore require invasive procedures to obtain respiratory samples (e.g. BAL fluid). The yield from culture depends on the severity of the illness: 15–25% of mild pneumonia cases are culture positive compared with 95% in cases of severe pneumonia causing respiratory failure.¹⁵ Delays in sputa processing and prior specific antimicrobial therapy decrease the yield.⁵

Fluorescent microscopy

Direct fluorescent-antibody (DFA) staining is a rapid method of directly detecting *Legionella* spp. in respiratory secretions and tissue samples. Although rapid, it is insensitive, requiring large organism numbers for visualisation (i.e. severe disease). Reported sensitivity of fluorescent microscopy varies but is consistently less than that of culture.¹⁵ Furthermore, it is technically demanding, requiring experienced laboratory personnel. False positive results may occur because of cross-reactions with other bacteria and yeasts.⁵ Problems with both sensitivity and specificity have limited the use of DFA staining in most laboratories.

Legionella urinary antigen tests

Soon after *L. pneumophila* was identified as the cause of Legionnaires’ disease, it was noted that *Legionella*

'antigen' could be found in patients' urine. The antigen detected is a component of the *Legionella* cell wall. Antigenuria can be detected as early as 1 day after the onset of symptoms and can persist for months despite therapy.¹ Popular formats include the enzyme immunoassay (EIA) and immunochromogenic test (ICT).

The two most frequently used tests have excellent sensitivity and specificity for *L. pneumophila* serogroup 1. The *Legionella* Urinary Antigen EIA (Binax, Inverness Medical: Scarborough, Maine) has a sensitivity of 70–90% and specificity approaching 100% for *L. pneumophila* serogroup 1.^{2,15–17} The ICT membrane assay (NOW *Legionella* Urinary Antigen Test: Binax, Inverness Medical: Scarborough, Maine) is simple to perform, rapid and its sensitivity and specificity are similar to those of EIA.¹⁸ Similar to culture and fluorescent microscopy, an association between clinical severity and test sensitivity occurs.¹⁷ Results can be obtained in 3 hours with the Binax EIA and in 15 minutes with the Binax NOW kits.

Attempts to create a *Legionella* urinary antigen test to detect species and serogroups other than *L. pneumophila* serogroup 1 have been problematic (sensitivity 29–31% for species other than *L. pneumophila* serogroup 1).¹⁹ In particular, no commercial assay is available to reliably detect *L. longbeachae* in urine.

Polymerase chain reaction

PCR-based detection of *Legionella* DNA in sputum, urine and blood has been described.^{1,6,15} PCR amplifies minute amounts of *Legionella* DNA, providing results within a short time and enabling detection of infection caused by all *Legionella* species and serogroups. Molecular methods can be formulated to incorporate real-time or multiplex formats. Despite the availability of commercial assays (e.g. Chlamyge kit, Argene Inc, NY), *Legionella* PCR is available only in a limited number of laboratories in Australia.

When testing clinical samples from the lower respiratory tract, PCR has been shown to have sensitivity equal to or greater than culture.^{20–22} False positive results have been reported using both in-house and commercial assays.⁶ *Legionella* DNA can also be detected from other samples, but with reduced sensitivity (30–86%).¹⁵

Serology

Serological testing for *Legionella* infection is a valuable epidemiological tool but is of less immediate benefit to physicians because of delayed seroconversion. Indirect immunofluorescent assays (IFA) and enzyme-linked immunosorbent assays (ELISA or EIA) are the most frequently performed tests.¹³ IFA remains the standard reference test and is validated for *L. pneumophila* and

L. longbeachae.¹⁵ ELISA assays are designed to provide a sensitive screen for legionellosis and detect IgM using *L. pneumophila* serogroup 1 or *L. longbeachae* sonicated whole cells as antigens.

Using IFA, a cut-off equal to or greater than 1:128 is recommended as evidence of recent or past infection. A single titre of 1:512 or higher for either *L. pneumophila* or *L. longbeachae* is a sensitive indicator of infection but may represent past infection or, on rare occasions, infection with another species.¹³ The demonstration of seroconversion or a four-fold rise in titre on a convalescent sample is required for diagnosis of definitive *Legionella* infection. In most cases, seroconversion is detected within 3–4 weeks; however, up to 10 weeks has been reported.²³ A proportion of people with a proven *Legionella* infection do not develop detectable *Legionella* antibodies.¹⁵ Cross-reactive antibodies are occasionally found in patients with other infections or non-infectious conditions. Clinicians should be encouraged to obtain convalescent samples after a minimum of 3 weeks. If there is no seroconversion after this time and clinical suspicion remains high, an additional convalescent sample should be obtained. IgM measured by ELISA can become positive earlier in the course of illness compared with IFA, although it may remain elevated for years and numerous cross-reactions can occur.¹³

Identification of *Legionella* spp. from environmental specimens

Attempts to culture *Legionella* spp. from environmental sources may be undertaken to investigate a clinical case cluster or as a part of the regular surveillance. An environmental investigation is generally not required following individual cases; however, the decision to investigate should be made by individual public health units, taking local factors into consideration.¹⁴ A number of tools, including electronic maps of registered cooling towers, may be utilised to identify potential point sources (Vicky Sheppard, pers. comm.).

A number of NATA-registered laboratories process environmental samples for *Legionella*. Culture methods are similar to those used in clinical laboratories. Following heat treatment to reduce growth of other bacteria, an aliquot of water is incubated on selective media. Following growth of suspicious colonies, antisera are used to identify presumptive *L. pneumophila*.

Typing of *Legionella* isolates

Approximately 4% of community-acquired and 37% of nosocomial *Legionella* infections constitute case clusters.¹ Standard serotyping of isolates is inadequate in epidemiological investigations because *L. pneumophila* serogroup 1 is the predominant organism in outbreaks. Further methods are required for subtyping or differentiation between potentially related strains.

Serological typing to identify 12 'type' strains within *L. pneumophila* serogroup 1 has been described.¹ Not all of the monoclonal antibodies from this panel are available in Australia;¹³ thus, molecular methods are usually preferred.

Various molecular methods are available for genotyping of clinical and environmental *Legionella* isolates in suspected case clusters. These include amplified fragment length polymorphism (AFLP) analysis, pulsed-field gel electrophoresis (PFGE), restriction fragment length polymorphism (RFLP) analysis and multi-locus sequence typing (MLST). The choice of method depends on the preference of the laboratory performing the test. Compared with DNA fragment-based methods (e.g. AFLP, PFGE or RFLP), DNA sequencing (e.g. MLST) is robust, offers greater reproducibility and allows results to be shared and compared between laboratories.^{5,24}

Subtyping of clinical and, if available, environmental isolates of *Legionella* is a powerful epidemiological tool to identify linked clinical cases and the possible common environmental source. Subtyping of *Legionella* spp. should be performed only if there is clear epidemiological evidence linking more than one case. Given the increasing use of non-culture-based methods, subtyping is limited by the infrequent isolation of *Legionella* spp. in culture. European data indicate that *Legionella* infections were diagnosed by culture in only 10% of cases.⁸

A rational approach to diagnosis

A rational approach to diagnosis is required because of the difficulty in distinguishing *Legionella* infection from other causes of community-acquired pneumonia. A diagnosis is necessary to enable identification and management of potential point sources. Testing algorithms may vary with different situations (e.g. a suspected outbreak compared with isolated cases). As each diagnostic method has limitations, a combination of tests is recommended.¹⁵

Based on the current evidence, it is our opinion that patients presenting with possible acute *Legionella* infection should have respiratory specimens cultured for *Legionella*, if available, combined with a *Legionella* urinary antigen test. Where available, a PCR-based assay to detect *Legionella*, together with a urinary antigen test, is a sensitive alternative; however, culture should still be attempted to obtain an isolate for identification and for genotyping if indicated. Reliance on urinary antigen tests will miss non-*L. pneumophila* serogroup 1 infections, including *L. longbeachae*. Fluorescent microscopy has little role, except in patients presenting with severe disease who have a negative *Legionella* urinary antigen. Serology remains the only method of documenting recent past infection. This may be of particular assistance where an alternative explanation for pneumonia has not been found or for epidemiological investigation of outbreaks where a point source is suspected.

When a culture is available, molecular typing of clinical and environmental isolates is a powerful tool for identifying linked clinical cases and any possible common environmental sources.

Conclusion

Well-established methods such as culture for *Legionella* and urinary *Legionella* antigen detection remain the mainstay of diagnosis of *Legionella* infections. Newer methods, including PCR-based assays, are likely to become more widely available in the future. Given the current limitations of laboratory diagnosis, patients presenting with pneumonia will continue to receive empiric therapy against *Legionella*.

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Mass gatherings and public health: preparing for World Youth Day 2008

**Jan Fizzell^{A,B}, Sophie E. Tyner^C
and Jeremy M. McAnulty^D**

^ANSW Public Health Officer Training Program,
NSW Department of Health

^BBiopreparedness Unit, NSW Department of Health

^CCounter-Disaster Unit, Ambulance Service of NSW

^DCommunicable Diseases Branch, NSW Department of Health

This Bug Breakfast seminar was held prior to the World Youth Day activities in Sydney, New South Wales (NSW) in July 2008.

Public health challenges and mass gatherings

Public health preparedness for mass gatherings is essential for the delivery of a safe and healthy event. The World Health Organization recently defined mass gatherings as 'events attended by a sufficient number of people to strain the planning and response resources of a community, state or nation'.¹

Public health challenges at mass gatherings include:

- the potential for communicable disease outbreaks
- mitigation of the risk of crowd crush
- the consideration of the likelihood of a terrorist event
- the likelihood of temperature-related illnesses (hypothermia and/or hyperthermia)
- sanitation
- events where active participation is encouraged (e.g. 'fun runs')
- access to safe food and water
- the possibility of crowd violence, noise issues, and the likely use and/or misuse of drugs and alcohol.

These challenges are compounded when events are of long duration, where little data relating to past experience with the event exists, and when protocols for managing the type of mass gathering are not readily available.

Some of the mitigation strategies that can be adopted include:

- rigorous pre-event planning
- regulation
- using appropriate health education strategies
- encouraging appropriate engineering to reduce the health risks to participants

- encouraging harm minimisation strategies
- encouraging pre-event vaccination
- pre-event surveillance and intelligence-gathering to better assess the risks of a particular event.

Planning for World Youth Day 2008

World Youth Day 2008 (WYD08) is a large-scale international gathering of Catholic youth which is taking place in July 2008. During 'Days in the Dioceses' from 10 to 14 July, participants can visit and undertake a program of activities at Diocese in locations across Australia and New Zealand. Participants will converge in Sydney for a program of major events, which is being held from 15 to 20 July. In Sydney, a large number of participants will stay in shared accommodation on the floors of school halls or commercial facilities.

Extensive public health planning has been undertaken for WYD08. This includes the formation of the WYD08 Public Health Working Party in September 2006, which has served as a conduit for early and regular engagement with key stakeholders including event organisers, laboratory services, local government and fellow government agencies. A public health project officer has been coordinating activities since November 2007, in order to assist all stakeholders in delivering a consistent response to WYD08.

There are significant communicable disease risks with an event of this type, especially considering the prolonged group contact and limited sanitation facilities available at temporary accommodation venues (such as school halls). To minimise the risks to participants, several strategies are being undertaken including encouraging pre-event vaccination and the provision of pre-event information encouraging hand washing and respiratory etiquette amongst pilgrims. Intra-event vaccination clinics may also be deployed (e.g. for Hepatitis A or varicella). A food preparation and inspection program has been developed by the NSW Food Authority, in consultation with local government. Advice has been provided to event organisers regarding the need to provide adequate hand washing facilities and isolation areas for sick participants.

The existing communicable disease surveillance program will be enhanced. Calls to a health advice line for participant accommodation team leaders and presentations to special event onsite medical units will be monitored for

conditions of public health interest. The Australian Government Department of Health and Ageing will assist by monitoring for international events that may have an impact on WYD08. Cooperation has been sought from other jurisdictions regarding pre-event health surveillance. The NSW Department of Health and all area health service public health units will remain on high alert and ready to respond to outbreaks of disease during WYD08, using agreed protocols.

Mass gatherings present both challenges and opportunities. Many of the strategies developed for managing

WYD08 will be applicable in other public health emergencies, including mass evacuations and large-scale communicable disease outbreaks.

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Methicillin-resistant *Staphylococcus aureus*

What is methicillin-resistant *Staphylococcus aureus*?

Staphylococcus aureus (commonly known as ‘staph’) are common, usually harmless bacteria. Many healthy people carry these bacteria on their skin or in their nose. Sometimes, however, they can cause infection and serious illness. Some strains of staph are resistant to methicillin and other antibiotics. These are known as methicillin-resistant *Staphylococcus aureus* (MRSA). MRSA infection is commonly known as ‘golden staph’.

What is community-acquired MRSA?

MRSA infections occur frequently among people in hospitals and other health care facilities. Some MRSA strains, known as community-acquired MRSA (CaMRSA), spread readily between people in the community. CaMRSA strains are often quite different to MRSA strains associated with hospitals and may cause infections in otherwise healthy people.

What are the symptoms?

Like ordinary staph, CaMRSA can cause infections:

- of the skin surface (e.g. boils and impetigo (school sores))
- under the skin (e.g. abscesses and cellulitis)
- of the bone, blood, lungs and other parts of the body.

How is it spread?

CaMRSA can get into the body through broken skin or sores, resulting in redness, pimples, swelling, tenderness or boils. Infections can become serious, leading to infections of the blood or pneumonia. CaMRSA can be spread by:

- touching or squeezing an infected body area, such as a boil or open wound
- using towels, clothes or bed sheets that have been used by a person with an MRSA infection
- using grooming items that have been used by a person with an MRSA infection
- not washing your hands carefully.

Who is at risk?

CaMRSA skin infections can affect anyone. Crowding and frequent skin-to-skin contact can increase the risk of infection, so outbreaks tend to occur in schools, dormitories, military barracks, households, jails and child-care centres. Cuts or abrasions, contact with contaminated items and surfaces, and infrequent washing increase the risk of

infection. People who have health problems such as diabetes or a poor immune system, or who have broken skin due to wounds or dermatitis, are also more likely to get an infection.

How is it prevented?

- Hand washing is important to prevent the spread of CaMRSA. Hands should be thoroughly washed with soap and running water for 10–15 seconds before and after touching or dressing an infected area, before handling or eating food, after going to the toilet, after blowing your nose and after touching or handling unwashed clothing or linen.
- Cover boils or other skin infections with a waterproof dressing. People who handle food must make sure that they do not contaminate any food and must keep any sores or skin infections completely covered with a waterproof dressing.
- Do not share personal items (e.g. clothes, towels or bed sheets) or grooming items (e.g. nail scissors, tweezers, razors and toothbrushes). If you share a bed with someone, keep sores or wounds covered overnight.

In addition to general hygiene, specific measures exist to help prevent the spread of MRSA in child-care centres, schools and among sporting groups.

MRSA in child-care centres and schools

- Teachers, children and families should understand the importance of hand washing, covering mouths while coughing and staying home if sick.
- Hand washing products (soap dispensers, running water and paper towel) should be available and accessible.
- Activities should allow time for hand washing (before eating and after going to the toilet).
- If open skin wounds cannot be kept covered, temporary exclusion from child care or school may be considered until the wound is healed or drainage of pus from the wounds can be contained using a sealed bandage.
- Surfaces such as counters, desks and toys that come into contact with uncovered or poorly covered infections should be cleaned daily with detergent, and whenever visibly contaminated.

MRSA in sporting groups

- People who have skin infections or open wounds that cannot be kept covered should not participate in contact sports until the wound has healed or drainage can be contained.
- People who have skin infections or open wounds should be excluded from common spas or saunas.
- People who have uncovered skin wounds should not share towels or sports equipment that is in contact with the skin.

How is it diagnosed?

Staph infections are usually diagnosed on the basis of their appearance and the presence of any related symptoms (e.g. fever). To diagnose an infection of MRSA, a doctor will need to take a swab or sample from the boil, wound or other site of infection for laboratory testing.

How is it treated?

Your doctor will advise on the best treatment for your infection. Many CaMRSA skin infections can be treated by draining the abscess or boil. Letting the pus drain out safely is often the only treatment that is needed. Drainage of boils or abscesses should only be performed by a doctor, trained nurse or health worker under sterile conditions. It is important to keep the wound well protected with a waterproof bandage so the infection is not spread to others.

In some circumstances CaMRSA is treated with antibiotics. If you are given an antibiotic, take all doses as

instructed, even if the infection improves. It is possible for a CaMRSA skin infection to come back after it appears cured.

What is the public health response?

Public health units can advise on the control of outbreaks. CaMRSA is not a notifiable condition in NSW.

For more information please contact your doctor, local public health unit or community health centre.

This factsheet is available at: http://www.health.nsw.gov.au/factsheets/infectious/methicilresist_staph.html



Communicable Diseases Report, NSW, July and August 2009

**Communicable Diseases Branch,
NSW Department of Health**

For updated information, including data and facts on specific diseases, visit www.health.nsw.gov.au and click on Public Health then Infectious Diseases, or access the site directly at: <http://www.health.nsw.gov.au/publichealth/infectious/index.asp>.

Figure 4 and Tables 2 and 3 show reports of communicable diseases received through to the end of August 2009 in New South Wales (NSW).

Invasive meningococcal disease

Twenty-seven cases of invasive meningococcal disease were reported in July and August in NSW, bringing the total number of cases to 68 so far this year. Two adult deaths were reported during July and August 2009. In comparison to August in 2008, there were 51 cases reported and one death.

There has been a downward trend in meningococcal notifications across all area health services in NSW since 2000. The highest numbers of notifications are reported among children aged less than 5 years at onset, with a second peak in the 15–24 year age group.

A vaccine against meningococcal C was added to the National Immunisation Program Schedule in January 2003. Consequently, serogroup C meningococcal disease is now mainly seen in adults and in unimmunised children. Serogroup B is the most common form of meningococcal disease in NSW. Of the 27 cases notified during July and August, 14 cases were due to serogroup B and two cases were due to serogroup C.

A media alert was released in August, reminding the public to be alert for the symptoms of meningococcal disease during winter and spring, the peak seasons for infection. An alert was also sent to GPs throughout NSW, highlighting the importance of early diagnosis and treatment of meningococcal disease.

Pertussis (whooping cough)

Monthly notifications of pertussis continue to decline steadily from the peak of the outbreak in December 2008. There were 1207 cases notified with onset in July and August, compared with 2082 in the preceding 2 months. The decrease was noted across all area health services in NSW. Comparison of data over time must be undertaken with caution however because of: recent changes in the use of diagnostic technologies (including the increasing use of nucleic acid testing); and changes in case ascertainment over time (related to increased awareness of the disease among doctors and the broader community).

The highest number of cases continue to be reported for children aged less than 15 years at onset, specifically children aged 0–4 and 5–9 years. Because pertussis immunity wanes over time, many older children and adults are susceptible to infection and can be the source of new infections in infants. Timely immunisation of infants is important because unvaccinated infants are at the highest risk of infection and of associated complications.

Since March 2009 and for a limited time in NSW, free pertussis (dTpa) vaccine has been available for: all new parents; couples who are planning a pregnancy; grandparents; and any other adults who will regularly care for infants aged less than 12 months. From March to August this year, 122 326 letters were sent to new parents in NSW, highlighting the pertussis outbreak and informing them of the availability of the free vaccine.

Measles

One case of measles was notified in July in NSW, bringing the total for the year to 10. This case was reported in an infant (too young for the Measles-Mumps-Rubella (MMR) vaccine) on their return from overseas travel. Thirty-nine cases of measles were notified in 2008. The majority of measles cases notified so far this year have been in young people recently returned from overseas travel, or in their contacts.

Many people born between 1966 and 1980 remain susceptible to measles because most people in this age group have not been exposed to measles infection and those who were routinely immunised typically received only one dose. Two doses are required to provide a high level of protection. Anyone born after 1965 should ensure that they

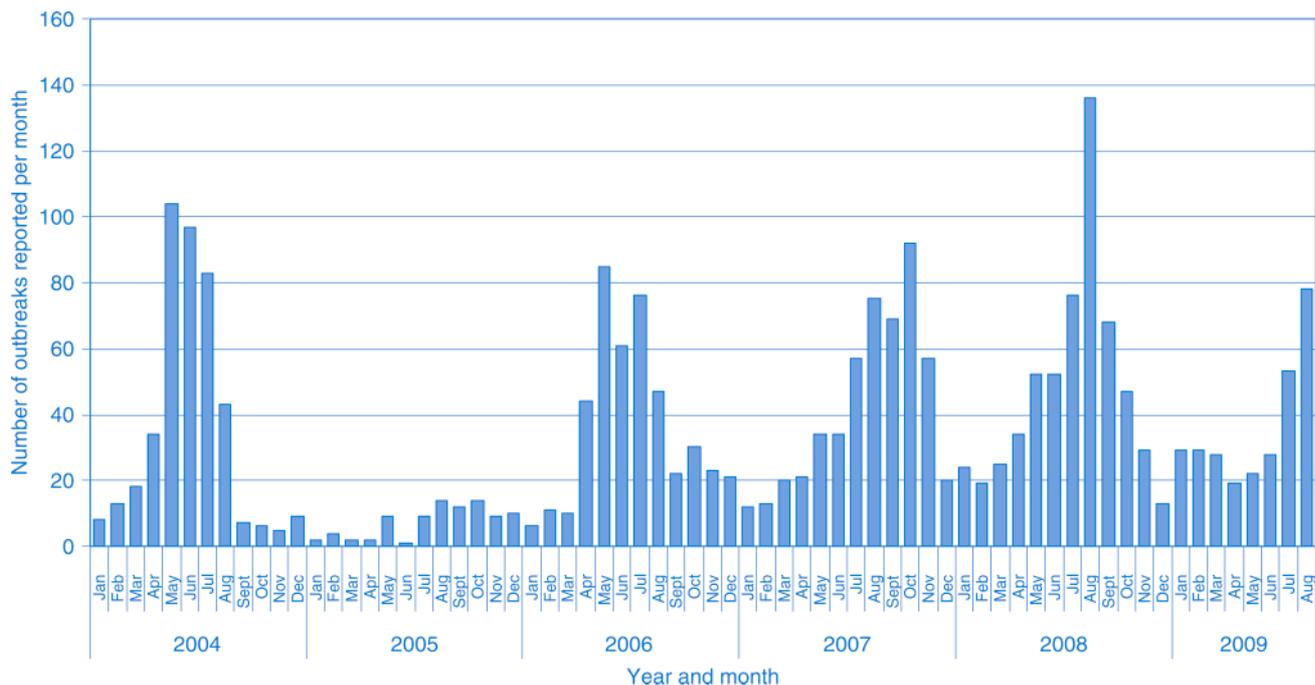


Figure 1. Number of outbreaks of gastrointestinal illness in institutions (e.g. aged-care facilities, child-care centres, schools, hospitals, etc.) reported to NSW Health for each month between 1 July 2004 and 31 August 2009.

have had two doses of MMR vaccine, unless they know they are immune.

Gastroenteritis in institutions

From 1 July 2004 to 31 August 2009, 747 outbreaks of gastrointestinal illness in institutions, affecting at least 16 256 people, were reported to NSW Health. Outbreaks of viral gastroenteritis are more commonly seen in winter months (Figure 1).

In 2009, between 1 July and 31 August, 131 outbreaks of gastrointestinal illness in institutions were reported, affecting 1804 people. This represents a small increase of 4% over the median number of outbreaks reported during the same time period from 2004 to 2008 ($n = 126$), and a significant decrease of 46% on the median number of people affected as a result of the outbreaks ($n = 3340$) (Table 1).

Twenty percent of the outbreaks were caused by norovirus, 4% by rotavirus, 1% by *Clostridium difficile* and *Campylobacter* respectively, and 75% were of unknown aetiology but were suspected to have been caused by person-to-person spread of a viral illness after investigating the epidemiological evidence and clinical symptoms of those affected. Fifty-five percent of the outbreaks occurred in aged-care facilities, 24% in hospitals, 20% in child-care centres, and 1% in a military facility.

Data collected on the number of presentations to emergency departments are consistent with the data shown in

Table 1. Number of outbreaks of gastrointestinal illness in institutions reported to NSW Health between 1 July 2009 and 31 August 2009 and number of people affected by these

| | Number of outbreaks | Number of people affected |
|------------------|---------------------|---------------------------|
| 2004 | 126 | 3341 |
| 2005 | 23 | 295 |
| 2006 | 123 | 3340 |
| 2007 | 132 | 3265 |
| 2008 | 212 | 4211 |
| Median 2004–2008 | 126 | 3340 |
| 2009 | 131 | 1804 |

this report, with no increase in presentations during the time period 1 July–31 August 2009 when compared with data collected from the previous 5 years.

In children aged 0–4 years, presentations to emergency departments for gastrointestinal illness during the period July–August 2009 decreased when compared with earlier years (Figure 2).

This decrease may be due to the introduction of the rotavirus vaccination into the National Immunisation Program in July 2007. The Eastern Sydney Laboratory Surveillance Program, based at the South Eastern Sydney Illawarra Public Health Unit, shows a reduction in the number of cases of rotavirus (Figure 3).

Category: age group
0-4 years

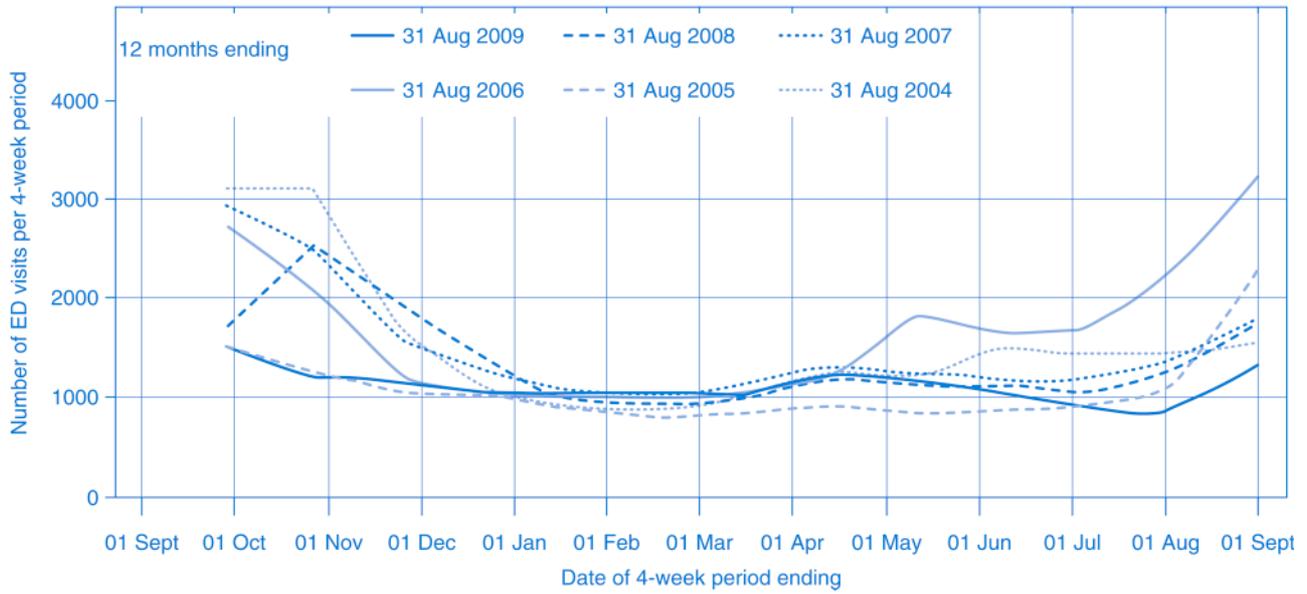


Figure 2. Emergency department visits for gastrointestinal illness in children aged 0-4 years by 4-week counts, ending 30 August 2009.

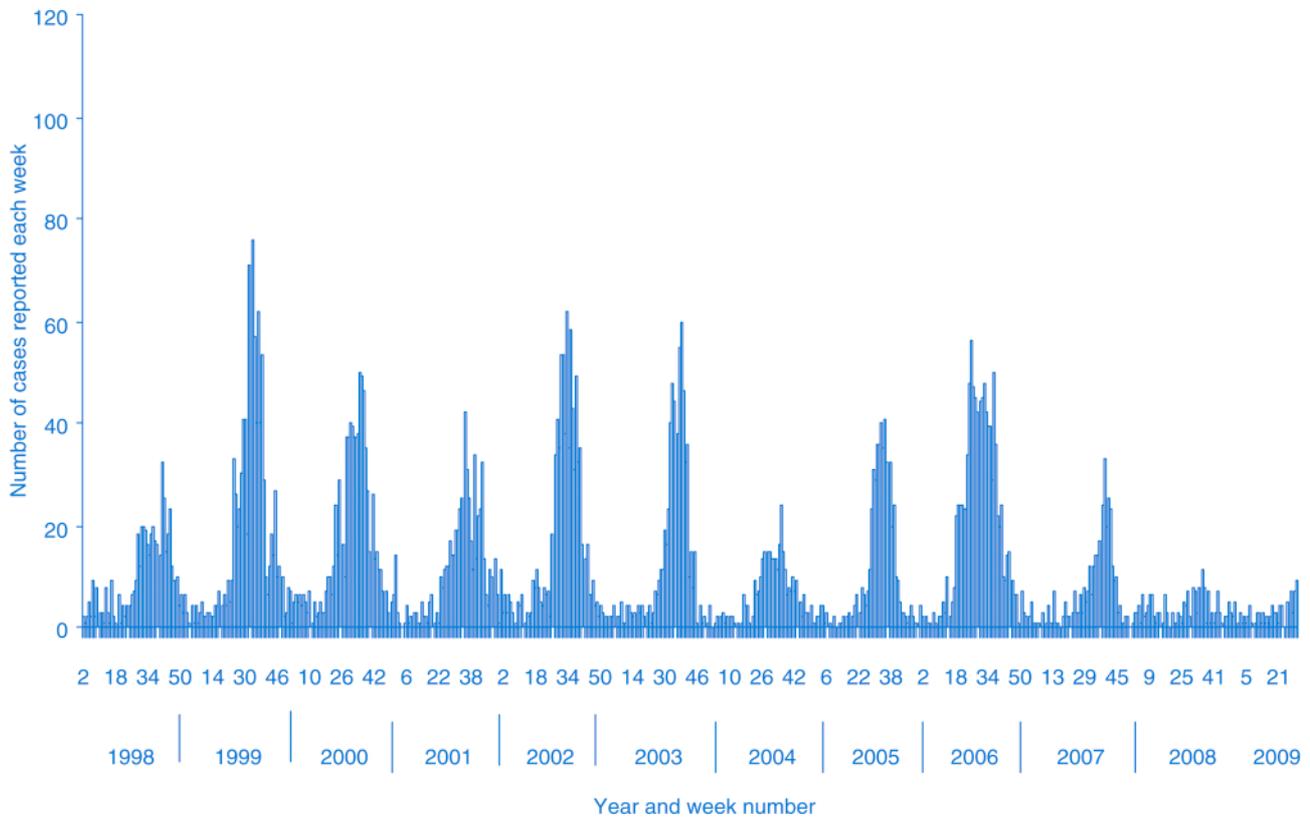


Figure 3. Number of cases of rotavirus reported each week by Eastern Sydney Laboratory Surveillance Program, 1998-2009.

Figure 4. Reports of selected communicable diseases, NSW, January 2004 to June 2009, by month of onset.

Preliminary data: case counts in recent months may increase because of reporting delays.

Laboratory-confirmed cases only, except for measles, meningococcal disease and pertussis.

BFV, Barmah Forest virus infection; RRV, Ross River virus infection; lab conf, laboratory confirmed;

Men Gp C and Gp B, meningococcal disease due to serogroup C and serogroup B infection; other/unk, other or unknown serogroups.

NB: Multiple series in graphs are stacked, except gastroenteritis outbreaks.

NB: Outbreaks are more likely to be reported by nursing homes and hospitals than by other institutions.

| NSW Population | |
|----------------|-----|
| Male | 50% |
| <5 y | 7% |
| 5-24 y | 27% |
| 25-64 y | 53% |
| 65+ y | 13% |
| Rural | 46% |

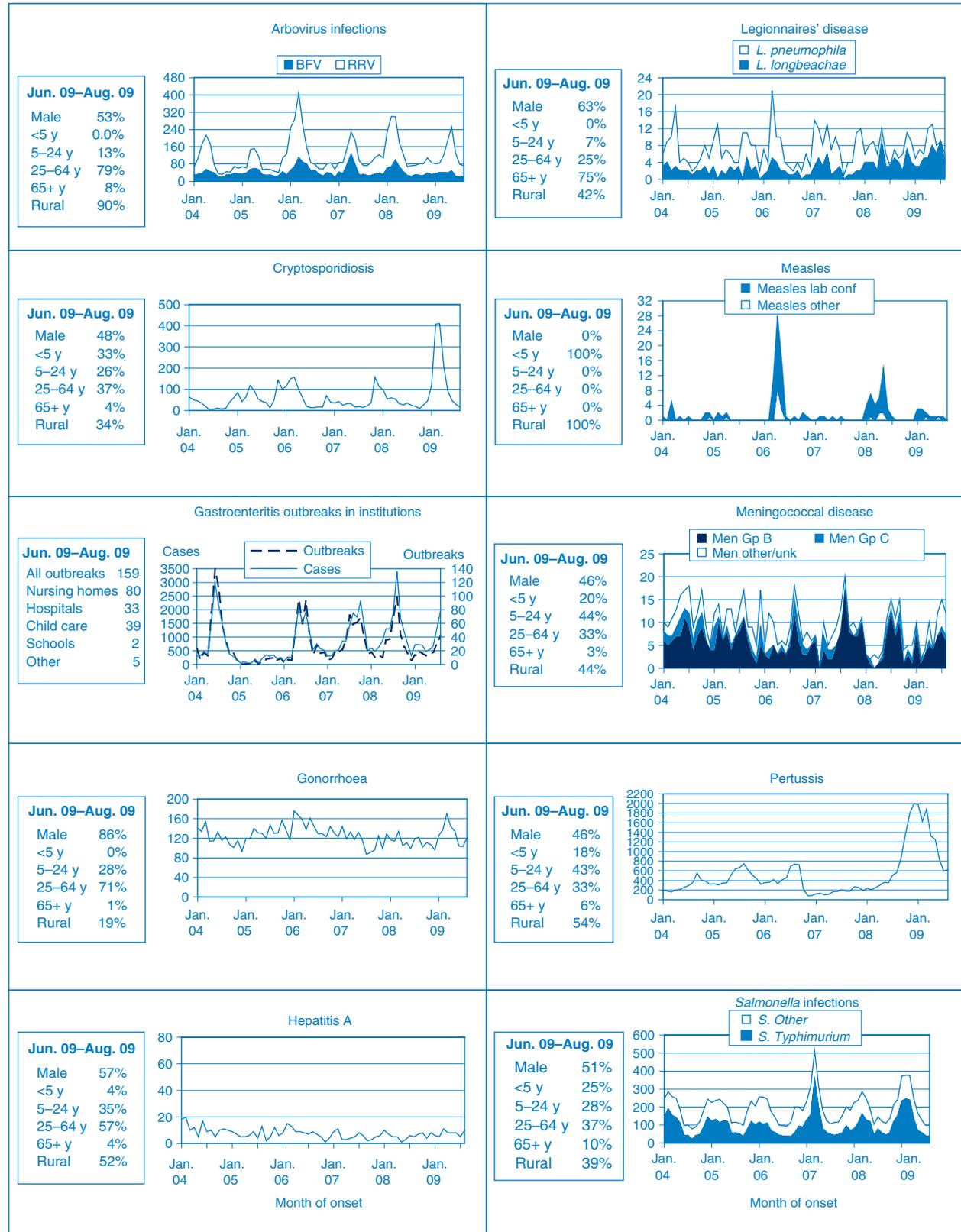


Table 2. Reports of notifiable conditions received in July 2009 by area health services

| Condition | Area Health Service (2009) | | | | | | | | | | | | Total Year to date ^b | | | | | | | |
|--|----------------------------|----|-----|-----------------|-----|-----|--------------------|-----|-------------|-----|-----|-------------------------------|---------------------------------|-----|--------------------------------|-----|-------------|-----------------------|-------------------|----|
| | Greater Southern | | | Greater Western | | | Hunter New England | | North Coast | | | Northern Sydney Central Coast | | | South Eastern Sydney Illawarra | | Sydney West | | Sydney South West | |
| | GMA | SA | FWA | MAC | MWA | HUN | NEA | MNC | NRA | CCA | NSA | ILL | SES | CSA | WEN | WSA | JHS | For July ^b | Total | |
| Bloodborne and sexually transmitted | | | | | | | | | | | | | | | | | | | | |
| Chancroid ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Chlamydia (genital) ^a | 68 | 24 | 13 | 24 | 34 | 158 | 25 | 28 | 50 | 61 | 67 | 64 | 246 | 125 | 55 | 114 | 22 | 1275 | 8794 | |
| Gonorrhoea ^a | 1 | - | 3 | - | 2 | 5 | - | 1 | - | 1 | 9 | 4 | 35 | 21 | - | 5 | - | 102 | 913 | |
| Hepatitis B – acute viral ^a | - | - | - | - | - | - | - | - | - | - | - | 1 | - | - | - | - | - | - | 2 | 23 |
| Hepatitis B – other ^a | 2 | 3 | 1 | 1 | 1 | 8 | 2 | 3 | - | 6 | 41 | 3 | 33 | 46 | 4 | 43 | 7 | 255 | 2220 | |
| Hepatitis C – acute viral ^a | - | - | - | - | - | 2 | - | - | - | - | - | - | - | - | - | - | - | 2 | 19 | |
| Hepatitis C – other ^a | 14 | 20 | 6 | 8 | 8 | 34 | 13 | 24 | 17 | 35 | 30 | 23 | 49 | 48 | 23 | 36 | 43 | 483 | 3791 | |
| Hepatitis D – unspecified ^a | - | - | - | - | - | - | - | - | - | 1 | - | - | - | - | - | - | - | 1 | 5 | |
| Lymphogranuloma venereum | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 2 | |
| Syphilis | 1 | 2 | 1 | 1 | 1 | 3 | 1 | - | - | 4 | 9 | 2 | 37 | 20 | 2 | 9 | - | 104 | 724 | |
| Vectorborne | | | | | | | | | | | | | | | | | | | | |
| Barmah Forest virus ^a | - | - | 2 | - | - | 5 | - | 7 | 6 | 1 | - | - | - | - | - | - | - | 21 | 254 | |
| Ross River virus ^a | 4 | 1 | 3 | - | 1 | 13 | 3 | 10 | 23 | 2 | - | - | - | - | - | - | - | 61 | 700 | |
| Arboviral infection (other) ^a | - | - | - | - | - | - | - | 1 | - | 1 | 6 | 2 | 2 | - | - | 3 | - | 16 | 118 | |
| Malaria ^a | 2 | 1 | - | - | - | - | - | - | - | - | 1 | - | 2 | - | - | 2 | - | 12 | 60 | |
| Zoonoses | | | | | | | | | | | | | | | | | | | | |
| Anthrax ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | |
| Brucellosis ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 2 | |
| Leptospirosis ^a | - | - | - | - | - | 1 | - | - | - | - | - | - | - | - | - | - | - | 1 | 14 | |
| Lyssavirus ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | |
| Psittacosis ^a | 1 | - | - | - | 2 | - | - | - | - | - | 1 | - | - | - | 1 | - | - | 5 | 19 | |
| Q fever ^a | 1 | 1 | 1 | 2 | 2 | 1 | 2 | - | 1 | - | 1 | 1 | - | - | - | - | - | 12 | 101 | |
| Respiratory and other | | | | | | | | | | | | | | | | | | | | |
| Blood lead level ^a | - | - | 2 | 2 | 2 | 4 | - | - | 1 | 1 | 1 | - | - | - | 3 | 1 | - | 17 | 151 | |
| Invasive pneumococcal infection ^a | - | 2 | - | 2 | 2 | 3 | 2 | - | 2 | 2 | 5 | 5 | 12 | 6 | 10 | 7 | - | 62 | 265 | |
| <i>Legionella longbeachae</i> infection ^a | - | - | - | - | - | - | - | 1 | 1 | 1 | - | 4 | - | 1 | - | 1 | - | 9 | 35 | |
| <i>Legionella pneumophila</i> infection ^a | - | - | - | - | - | - | - | - | - | - | - | - | 1 | - | - | - | - | 1 | 23 | |
| Legionnaires' disease (other) ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 1 | 2 | |
| Leprosy | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | |
| Meningococcal infection (invasive) ^a | - | - | - | 1 | - | 2 | 1 | - | 1 | - | 2 | 1 | 2 | 1 | - | 3 | - | 14 | 54 | |
| Tuberculosis | - | 1 | - | - | - | 1 | - | - | 3 | 2 | 2 | 1 | 7 | 3 | 7 | 14 | - | 42 | 248 | |
| Vaccine-preventable | | | | | | | | | | | | | | | | | | | | |
| Adverse event after immunisation | 3 | - | - | - | - | 2 | - | - | - | - | - | - | - | - | 1 | 2 | - | 12 | 91 | |
| <i>H. influenzae b</i> infection (invasive) ^a | - | - | - | - | - | 1 | - | - | - | - | - | - | - | - | 1 | - | - | 1 | 6 | |
| Measles | - | - | - | - | - | - | - | - | - | - | - | - | 1 | - | - | - | - | 3 | 10 | |
| Mumps ^a | - | - | - | - | - | 1 | - | - | - | - | - | - | - | - | - | - | - | 3 | 25 | |
| Pertussis | 12 | 39 | 4 | 19 | 32 | 69 | 12 | 45 | 50 | 27 | 51 | 45 | 38 | 25 | 47 | 64 | - | 645 | 10034 | |
| Rubella ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 6 | |
| Tetanus | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 2 | |
| Enteric | | | | | | | | | | | | | | | | | | | | |
| Botulism | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | |
| Cholera ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 3 | |
| Cryptosporidiosis ^a | 9 | 10 | 1 | 5 | 2 | 15 | 3 | 1 | 2 | 8 | 17 | 4 | 26 | 8 | 7 | 23 | 1 | 23 | 1333 | |
| Giardiasis ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 160 | 1298 | |
| Haemolytic uraemic syndrome | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 3 | |
| Hepatitis A ^a | - | - | - | - | - | - | - | 1 | - | - | - | 2 | 1 | - | - | 1 | - | 7 | 56 | |
| Hepatitis E ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 1 | 12 | |
| Hepatitis B ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 3 | 18 | |
| Listeriosis ^a | 3 | 3 | - | 1 | 1 | 6 | 4 | 4 | 1 | 7 | 15 | 5 | 14 | 8 | 5 | 15 | - | 100 | 1816 | |
| Salmonellosis ^a | 1 | - | - | - | - | - | - | - | - | - | 1 | 6 | 1 | 1 | 1 | 1 | - | 11 | 118 | |
| Shigellosis ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 3 | 30 | |
| Typhoid ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 17 | |
| Verotoxin producing <i>E. coli</i> ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 2 | |
| Miscellaneous | | | | | | | | | | | | | | | | | | | | |
| Creutzfeldt-Jakob disease | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 9 | |
| Meningococcal conjunctivitis | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 2 | |

^a laboratory-confirmed cases only. ^b includes cases with unknown postcode. NB: Data are current and accurate as at the preparation date. The number of cases reported is, however, subject to change, as cases may be entered at a later date or retracted upon further investigation. Historical Area Health Service configurations are included for continuity/ comparison purposes and to highlight regional differences. NB: Influenza data has not been provided here since May 2009. See www.health.nsw.gov.au/PublicHealth/Infectious/z.asp#fl for up-to-date information. NB: From 1 January 2005, Hunter New England AHS also comprises Great Lakes, Gloucester and Greater Icare LGAs (LGA, Local Government Area). Sydney West also comprises Greater Lithgow LGA. NB: HIV and AIDS data are reported separately in the Public Health Bulletin quarterly. GMA, Greater Murray Area; MAC, Macquarie Area; FWA, Far West Area; HUN, Hunter Area; HUN, Hunter Area; SA, Southern Area; ILL, Illawarra Area; MNC, North Coast Area; MWA, Mid Western Area; NSA, Northern Sydney Area; CSA, Central Sydney Area; WSA, Western Sydney Area; WEN, New England Area; WEN, North Coast Area; WSA, South Western Sydney Area; JHS, Justice Health Service.

Table 3. Reports of notifiable conditions received in August 2009 by area health services

| Condition | Area Health Service (2009) | | | | | | | | | | Total For August ^b | Total Year to date ^b | | | | | | | |
|---|----------------------------|----|-----|---------------------|-----|-----|-----------------|-----|-----------------|-------------------|-------------------------------|---------------------------------|---------------------|----------------------|-------------------|-----|-----------------------|-----|-----------------|
| | Greater Southern GMA | SA | FWA | Greater Western MAC | MWA | HUN | New England NEA | MNC | North Coast NRA | Central Coast CCA | | | Northern Sydney NSA | Sydney Illawarra ILL | South Eastern SES | CSA | Sydney South West SWS | WEN | Sydney West WSA |
| Bloodborne and sexually transmitted | | | | | | | | | | | | | | | | | | | |
| Chancroid ^a | - | 23 | 6 | 20 | 30 | 131 | 34 | 36 | 60 | 50 | 94 | 63 | 204 | 116 | 94 | 39 | 112 | 6 | - |
| Chlamydia (genital) ^a | 50 | - | 1 | - | - | 4 | - | 4 | 2 | 3 | 11 | 4 | 35 | 27 | 14 | 2 | 11 | - | 1177 |
| Gonorrhoea ^a | 2 | - | - | - | - | - | - | - | - | - | - | - | - | - | 1 | - | - | - | 120 |
| Hepatitis B – acute viral ^b | 3 | 1 | - | 2 | - | 5 | 2 | - | - | 3 | 27 | 7 | 35 | 43 | 46 | 4 | 59 | 2 | 1 |
| Hepatitis B – other ^c | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 242 |
| Hepatitis C – acute viral ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 19 |
| Hepatitis C – other ^a | 9 | 8 | 6 | 11 | 16 | 42 | 7 | 23 | 18 | 25 | 26 | 23 | 52 | 40 | 55 | 24 | 31 | 27 | 449 |
| Hepatitis D – unspecified ^b | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 1 |
| Lymphogranuloma venereum | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 2 |
| Syphilis | - | 3 | 8 | 1 | 2 | 3 | - | 1 | 4 | 3 | 6 | 1 | 30 | 22 | 10 | 2 | 7 | - | 104 |
| Vectorborne | | | | | | | | | | | | | | | | | | | |
| Barmah Forest virus ^a | - | - | - | - | - | 5 | - | 9 | 6 | 2 | - | - | - | - | - | - | - | - | 22 |
| Ross River virus ^a | 5 | 1 | 2 | 2 | 2 | 10 | 2 | 7 | 11 | 1 | 1 | - | 1 | - | - | 2 | 1 | - | 49 |
| Arboviral infection (other) ^a | - | - | - | - | - | 3 | - | 1 | 1 | - | 3 | 1 | - | 2 | - | - | 1 | - | 749 |
| Malaria ^a | 1 | 1 | - | 1 | - | 1 | - | - | - | - | 1 | - | 1 | 1 | 2 | - | 1 | - | 12 |
| Zoonoses | | | | | | | | | | | | | | | | | | | |
| Anthrax ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Brucellosis ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 2 |
| Leptospirosis ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 14 |
| Lyssavirus ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Psittacosis ^a | 1 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Q fever ^a | 1 | 1 | - | 2 | 2 | - | - | - | 2 | - | - | - | - | - | - | - | - | - | 1 |
| Respiratory and other | | | | | | | | | | | | | | | | | | | |
| Blood lead level ^a | - | - | - | 3 | - | 5 | - | - | - | - | - | - | - | - | 1 | - | - | - | 9 |
| Invasive pneumococcal infection ^a | 1 | 4 | - | - | 1 | 8 | 1 | 1 | 2 | 6 | 5 | - | 7 | 9 | 8 | 2 | 5 | - | 59 |
| Legionella longbeachae infection ^a | - | - | - | - | 1 | - | - | - | - | 1 | - | - | 1 | 2 | - | - | 2 | - | 7 |
| Legionella pneumophila infection ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 42 |
| Legionnaires' disease (other) ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 1 |
| Leptosy | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 2 |
| Meningococcal infection (invasive) ^a | 1 | 1 | - | - | 1 | 2 | - | 1 | 1 | 1 | 2 | 1 | 2 | 3 | 1 | - | - | - | 13 |
| Tuberculosis | 1 | 2 | - | - | - | 1 | - | 1 | 1 | 1 | 2 | 1 | 8 | 2 | 2 | - | 4 | - | 25 |
| Vaccine-preventable | | | | | | | | | | | | | | | | | | | |
| Adverse event after immunisation | 3 | - | - | - | - | - | - | - | - | - | 1 | - | - | - | 1 | 1 | 1 | - | 7 |
| H. influenzae b infection (invasive) ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 6 |
| Measles | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 10 |
| Mumps ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 27 |
| Pertussis | 27 | 38 | 3 | 10 | 35 | 93 | 21 | 20 | 41 | 24 | 45 | 32 | 63 | 35 | 30 | 49 | 54 | 620 | 10654 |
| Rubella ^a | - | 1 | - | - | - | - | - | - | - | - | 1 | - | - | - | - | - | - | - | 2 |
| Tetanus | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 8 |
| Enteric | | | | | | | | | | | | | | | | | | | |
| Botulism | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Cholera ^a | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 3 |
| Cryptosporidiosis ^a | - | - | - | 1 | - | - | - | 3 | - | - | 11 | 1 | 9 | 3 | 1 | 1 | - | - | 30 |
| Giardiasis ^a | 5 | 8 | 1 | - | 3 | 16 | 7 | 3 | - | 2 | 34 | 9 | 33 | 16 | 8 | 8 | 11 | - | 164 |
| Haemolytic uraemic syndrome | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 3 |
| Hepatitis A ^a | 3 | - | - | - | - | 1 | - | - | - | - | 2 | - | 1 | - | 3 | - | - | - | 10 |
| Hepatitis E ^a | - | - | - | - | - | - | - | - | - | - | - | - | 1 | - | - | - | 1 | - | 2 |
| Listeriosis ^a | - | - | - | - | - | - | - | - | - | - | - | - | 1 | 1 | - | - | - | - | 14 |
| Salmonellosis ^a | 5 | 1 | - | - | - | 12 | 6 | 2 | 3 | 4 | 11 | 2 | 16 | 10 | 15 | 9 | 16 | - | 20 |
| Shigellosis ^a | - | - | - | - | - | - | - | - | - | - | 1 | - | 2 | 3 | - | - | - | - | 113 |
| Typhoid ^b | - | - | - | - | - | - | - | - | - | - | 1 | - | 1 | 3 | 1 | - | 1 | - | 8 |
| Verotoxin producing E. coli ^a | - | - | - | - | - | 1 | - | - | - | - | - | - | 1 | - | - | - | - | - | 4 |
| Miscellaneous | | | | | | | | | | | | | | | | | | | |
| Creutzfeldt-Jakob disease | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 9 |
| Meningococcal conjunctivitis | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 2 |

^aLaboratory-confirmed cases only. ^bIncludes cases with unknown postcode. ^cData is incomplete. NB: Data are current and accurate as at the preparation date. The number of cases reported is, however, subject to change, as cases may be entered at a later date or retracted upon further investigation. Historical Area Health Service configurations are included for continuity/ comparison purposes and to highlight regional differences. NB: Influenza data has not been provided here since May 2009. See www.health.nsw.gov.au/PublicHealth/Infectious/a-z.asp# for up-to-date information. NB: From 1 January 2005, Hunter, New England AHS also comprises Great Lakes, Gloucester and Greater Taree LGAs (LGA, Local Government Area), Sydney West also comprises Greater Lithgow LGA. NB: HIV and AIDS data are reported separately in the Public Health Bulletin quarterly. GMA, Greater Murray Area; MAC, Macquarie Area; NEA, New England Area; FWA, Far West Area; WSA, Western Sydney Area; CSA, Central Sydney Area; MWA, Mid Western Area; MNC, North Coast Area; NSA, Northern Sydney Area; SA, Southern Area; ILL, Illawarra Area; SWS, South Western Sydney Area; JHS, Justice Health Service.

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Editors

Dawn Simpson
Dr Lynne Madden
BSc(Med)Hons1, MBBS, MPH, MSc, FFPH, FAFPHM

Editorial Manager

Kristy Mannix

Editorial correspondence

Please address all correspondence and submissions to:
The Editor, *NSW Public Health Bulletin*
Locked Mail Bag 961
North Sydney NSW 2059 Australia
Email: phbulletin@doh.health.nsw.gov.au
Telephone: +61 2 9424 5876
Fax: +61 2 9391 9232

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