

NSW PUBLIC HEALTH BULLETIN

Year in Review 2007

Year in review: communicable disease surveillance, NSW, 2007

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In this issue, we present our annual review of notifiable diseases among New South Wales (NSW) residents. Readers interested in the details of notifications for specific diseases are referred to in Tables 2–6 where diseases are reported by: year of onset; month of onset; Area Health Service (AHS); and age group and sex.

Trends

Among the 46706 notifications of medical conditions by doctors, hospital staff and laboratory staff in NSW residents in 2007, highlights included:

Conditions most frequently reported

- *Chlamydia trachomatis* infections: 12447 cases (181 per 100000 population) with the highest crude rates by geographical area in the South Eastern Sydney Illawarra (Randwick region), Sydney South West (Camperdown region), Hunter New England (Tamworth region) and Greater West (Broken Hill region) AHSs.
- Hepatitis C: 4259 cases (62 per 100000 population) with the highest crude rates in the Greater Western (Broken Hill region), North Coast (Lismore region) and Sydney South West (Camperdown region) AHSs.
- Hepatitis B: 2656 cases (39 per 100000 population) with the highest crude rates in the Sydney South West (Camperdown and Liverpool regions) and Sydney West (Parramatta region) AHSs.
- *Salmonella* infections: 2564 cases (37 per 100000 population) with the highest crude rates in the North Coast (Lismore region), Northern Sydney Central Coast (Gosford region) and Sydney South West (Camperdown region) AHSs.
- Pertussis: 2093 cases (30 per 100000 population) with the highest crude rates in Greater Western (Dubbo region), Sydney West (Parramatta region) and South Eastern Sydney Illawarra (Randwick region) AHSs.

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

- Measles: four cases in 2007, the lowest annual count to date and a striking decrease compared with 2348 cases notified in 1993. No local measles transmission occurred in 2007 with all four cases resulting from exposure overseas.
- Meningococcal serogroup C disease: 10 cases reported for 2007, the lowest number of notifications since laboratory reporting began in 1991, largely due to the introduction of meningococcal C vaccination in late 2003.
- Gonorrhoea: 1384 cases in 2007 compared with 1736 cases in 2006, a decrease of 20%.
- Hepatitis A: a record low number of 65 cases, decreased from 1119 in 1991, perhaps in part due to the introduction of a commercially based vaccination in the 1990s. Travel to endemic countries was the most commonly reported risk factor for disease acquisition in 2007.
- Psittacosis: 34 cases, a 64% decrease compared with 2006.
- Leptospirosis: eight cases, down from 66 in 2001.

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

- *Salmonella* infections: 2564 cases, the highest annual count to date.¹ This increase is mainly due to a large point-source outbreak affecting 319 people who ate Vietnamese-style pork or chicken rolls from a bakery.
- Infectious syphilis, primarily affecting homosexual men residing in metropolitan Sydney.
- Mumps: 323 cases, a steady increase from 28 cases in 2001. This increase occurred mainly in the second half of the year in young adults in South East Sydney Illawarra (Randwick region) AHS.

- Legionnaire disease: 73 reported *Legionella pneumophila* infections in part due to an outbreak in Sydney central business district in January.
- Influenza with high rates of disease reported in July and August. There were 25 reported influenza (influenza A) outbreaks in 2007: 21 in aged-care

Table 1. The five most commonly reported notifiable diseases by age group, NSW, 2007

Age group	Rate/100000
Children under 5 years	
1. <i>Salmonella</i> infection	146
2. Giardiasis	129
3. Influenza	97
4. Cryptosporidiosis	44
5. Pertussis	41
Children (5 to 15 years)	
1. <i>Salmonella</i> infection	37
2. Giardiasis	27
3. Chlamydia*#	24
4. Pertussis	21
5. Influenza	19
Young adults (16 to 24 years)	
1. Chlamydia*	818
2. Hepatitis C	53
3. Hepatitis B	48
4. Gonorrhoea	40
5. <i>Salmonella</i> infection	40
Adults (25 to 44 years)	
1. Chlamydia*	241
2. Hepatitis C	124
3. Hepatitis B	74
4. Gonorrhoea	41
5. Giardiasis	32
Adults (45 to 64 years)	
1. Hepatitis C	70
2. Pertussis	39
3. Hepatitis B	37
4. Arboviral infection	34
5. Chlamydia*	30
Older Adults (65 years)	
1. Influenza	36
2. Pertussis	33
3. <i>Salmonella</i> infection	24
4. Arboviral infection	21
5. Invasive pneumococcal disease	19
* refers to <i>Chlamydia trachomatis</i> infection.	
# two-thirds of the notifications reported in this age group were in 15 year olds. Where a case is reported in a child under 16 years old, 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.	

- facilities, three in military facilities and one in a boarding school.
- Verotoxigenic *Escherichia coli* (VTEC) infections: 23 cases reported, compared with 10 cases reported in 2006. All cases were investigated and no epidemiological links were identified.
- Giardiasis: 1940 cases reported compared with 1725 reported in 2006.

Conditions least frequently reported

There were no reported cases of anthrax, avian influenza, botulism, chancroid, diphtheria, granuloma inguinale, lyssavirus, plague, polio, rabies, severe acute respiratory syndrome (SARS), smallpox, tularaemia, typhus, viral haemorrhagic fever or yellow fever in NSW in 2007.

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 Fig. 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 the institutional gastrointestinal outbreaks as 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 highest rates observed in regional areas followed by rural and metropolitan areas.
- Rates of hepatitis C infection were comparable across rural, regional and metropolitan areas. Most of these cases will represent chronic infection rather than acute hepatitis C acquisition and as such may not accurately reflect the recent spread of the hepatitis C epidemic.
- Arboviral infections are more commonly reported in people residing in rural and regional areas than in metropolitan areas, relating to the distribution of infected mosquitoes.
- Higher rates of disease are reported for Justice Health compared with the rest of NSW, likely related to higher testing rates for bloodborne viruses and sexually transmitted infections on entry into correctional facilities. Within this population, hepatitis C was the most commonly reported infection, attributable to high rates of injecting drug use.

Age distribution of notifiable diseases

- Gastrointestinal and respiratory diseases are most commonly reported in children aged under 5 years. This is influenced by the higher testing rates in this age group.

Table 2. Disease notifications by year of onset of illness^a, NSW, 1991–2007

Condition	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	
Adverse event after immunisation	9	31	23	40	28	56	70	95	16	42	111	178	219	186	107	70	224	
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	
Arboviral infection	408	343	656	381	539	1227	1806	783	1220	980	1191	665	1024	1148	1088	1917	1498	
Barmah Forest virus ^b	6	6	25	39	271	172	185	134	249	197	401	396	451	403	448	644	573	
Ross River virus ^b	297	324	599	331	236	1031	1598	583	952	750	717	183	494	701	584	1221	841	
Other ^b	105	13	32	11	32	24	23	66	19	33	73	86	79	44	56	52	84	
Blood lead level ≥ 15ug/dL ^b	Not notifiable until December 1996																	
Botulism	0	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	0	
Brucellosis ^b	2	2	4	4	2	1	3	3	2	1	1	2	3	7	3	10	3	
Chancroid ^b	Not notifiable until December 1998																	
<i>Chlamydia trachomatis</i> infection										2469	3509	4500	5823	7788	10030	11285	12057	12447
Congenital chlamydia ^b	Not notifiable until August 1998																	
Chlamydia – other ^b	Not notifiable until August 1998																	
Cholera ^b	1	0	1	0	1	3	1	1	2	0	1	1	0	1	0	3	2	
Creutzfeldt–Jakob disease ^b	Not notifiable until April 2004																	
Cryptosporidiosis ^b	Not notifiable until December 1996																	
Foodborne illness (NOS) ^c	2765	253	106	213	270	211	255	201	151	147	56	41	1071	550	309	507	763	
Gastroenteritis (institutional)	158	406	443	296	1359	554	939	738	673	697	775	1752	3583	12784	1395	10641	10488	
Giardiasis ^b	Not notifiable until August 1998																	
Gonorrhoea ^b	392	491	382	357	428	522	636	1054	1291	1060	1364	1527	1328	1442	1579	1736	1384	
Haemolytic uraemic syndrome	Not notifiable until December 1996																	
<i>Haemophilus influenzae</i> serotype ^b	212	217	124	61	29	13	17	11	13	8	7	10	6	5	7	11	7	
Hib epiglottitis ^b	15	57	32	21	6	2	5	1	2	2	1	1	0	3	0	1	1	
Hib meningitis ^b	48	103	53	17	11	4	3	3	3	1	1	1	0	0	2	0	2	
Hib septicaemia ^b	11	26	24	12	8	3	1	4	6	4	2	3	1	2	4	6	2	
Hib infection NOS ^b	138	31	15	11	4	4	8	3	2	1	3	5	5	0	1	4	2	
Hepatitis A ^b	1119	901	579	585	614	958	1426	927	421	201	197	149	124	137	83	95	65	
Hepatitis B	1492	3169	3603	3983	4007	3504	3167	2957	3508	3972	4556	3546	2845	2812	2742	2518	2656	
Hepatitis B – acute viral ^b	409	112	95	74	61	43	53	58	77	100	94	88	74	53	56	53	56	
Hepatitis B – other ^b	1083	3057	3508	3909	3946	3461	3114	2899	3431	3872	4462	3458	2771	2759	2686	2465	2600	
Hepatitis C	851	3895	5896	7820	6878	7000	6926	7206	8598	8297	8654	6694	5249	4915	4364	4392	4259	
Hepatitis C – acute viral ^b	22	26	22	16	32	18	19	112	112	222	295	152	127	59	43	55	53	
Hepatitis C – other ^b	829	3869	5874	7804	6846	6982	6907	7094	8486	8075	8359	6542	5122	4856	4321	4337	4206	
Hepatitis D ^b	0	8	12	19	19	9	11	3	14	12	11	9	12	14	15	15	11	
Hepatitis E ^b	0	0	1	2	0	3	6	4	7	9	6	6	6	8	7	10	8	
HIV infection ^b	823	693	589	502	536	447	423	402	377	352	340	393	413	407	391	369	404	
Influenza	Not notifiable until December 2000																	
Influenza – Type A ^b	Not notifiable until December 2000																	
Influenza – Type B ^b	Not notifiable until December 2000																	
Influenza – Type A & B ^b	Not notifiable until December 2003																	
Influenza – Type NOS ^b	Not notifiable until December 2000																	
Legionellosis	37	104	66	60	75	74	33	46	41	41	68	44	60	80	89	78	105	
<i>Legionella longbeachae</i> ^b	0	14	13	8	16	30	9	19	12	12	29	21	37	27	24	22	29	
<i>L. pneumophila</i> ^b	16	80	34	30	35	34	18	22	22	26	38	22	23	51	64	55	73	
Legionnaire disease other	21	10	19	22	24	10	6	5	7	3	1	1	0	2	1	1	3	
Leprosy	1	7	5	3	3	2	0	0	1	2	4	0	2	5	1	1	4	
Leptospirosis ^b	28	21	16	14	6	33	33	50	56	54	66	39	39	40	35	18	8	
Listeriosis ^b	11	13	12	10	14	22	23	28	22	18	12	11	28	30	25	26	22	
Lymphogranuloma venereum (LGV) ^b	0	0	0	0	0	0	0	0	0	0	0	0	0	1	2	1	0	
Malaria ^b	171	110	174	184	96	204	173	158	174	232	157	105	120	101	206	140	98	
Measles	495	805	2348	1484	596	191	273	119	32	36	31	8	18	12	5	60	4	
Measles laboratory confirmed	19	76	460	302	138	35	98	19	13	22	18	6	14	11	4	48	4	
Measles – other	476	729	1888	1182	458	156	175	100	19	14	13	2	4	1	1	12	0	
Meningococcal disease	128	121	153	142	113	161	218	186	221	253	234	216	202	149	140	107	112	
Meningococcal – serogroup B ^b	0	3	7	7	23	36	53	55	95	93	90	105	100	81	73	54	76	
Meningococcal – serogroup C ^b	0	4	6	9	8	35	55	55	60	64	38	54	45	24	16	13	10	
Meningococcal – serogroup W135 ^b	0	0	0	0	1	0	2	4	4	4	2	2	2	5	8	5	2	
Meningococcal – serogroup Y ^b	0	0	1	1	0	1	0	7	1	7	2	2	5	3	3	1	5	
Meningococcal – other	128	114	139	125	81	89	108	65	61	85	102	53	50	36	40	34	19	
Mumps ^b	8	23	13	11	14	27	29	39	33	92	28	29	35	65	111	155	323	
Paratyphoid ^{b,d}	20	8	9	11	12	15	5	9	5	14	11	13	22	10	0	0	0	
Pertussis	49	217	1533	1405	1369	1156	4246	2309	1415	3691	4437	2012	2772	3569	5809	4918	2093	
Pneumococcal disease (invasive) ^b	Not notifiable until December 2000																	
Psittacosis ^b	Not notifiable until December 2000																	
Q fever ^b	167	213	403	267	201	287	258	236	164	132	144	310	288	223	143	175	215	
Rubella	60	324	1186	233	2376	636	153	78	46	191	58	35	24	18	10	37	9	
Congenital rubella ^b	1	0	2	4	1	5	0	0	1	0	0	0	1	1	0	0	1	
Rubella – other ^b	59	324	1184	229	2375	631	153	78	45	191	58	35	23	17	10	37	8	
<i>Salmonella</i> infection ^{b,d}	1115	819	1001	1125	1393	1250	1721	1826	1470	1426	1671	2112	1842	2145	2184	2071	2564	
Shigellosis ^b	Not notifiable until December 2000																	
Syphilis	580	873	730	963	835	662	510	611	584	580	547	646	842	1042	841	892	1115	
Congenital syphilis	1	1	0	2	6	3	3	0	3	2	1	1	3	1	5	4	4	
Infectious syphilis ^{b,c}	1	3	6	29	132	72	57	45	86	81	67	128	244	302	242	232	434	
Syphilis – other ^b	578	869	724	932	697	587	450	566	495	497	517	595	739	594	656	677		
Tetanus	5	2	5	4	0	1	3	3	1	3	0	0	1	1	1	2	2	
Tuberculosis ^b	429	394	389	394	443	410	422	382	483	448	416	447	386	431	452	463	452	
Typhoid ^b	11	3	7	1	0	3	5	1	0	3	5	14	13	35	25	31	26	
Verotoxin-producing <i>Escherichia coli</i> infections ^b	Not notifiable until December 1996																	

^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. ^dFrom 2005, all paratyphoid recorded as salmonellosis. ^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. Due to data delay AIDS notifications will be reported in a later edition.

Table 3. Disease notifications by month of onset of illness^a, NSW, 2007

Conditions	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
Adverse event after immunisation	6	3	11	11	59	39	20	23	21	13	13	5	224
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0
Arboviral infection	97	97	163	234	196	99	83	80	89	114	127	119	1498
Barmah Forest virus ^b	43	35	76	125	77	29	32	27	27	34	38	30	573
Ross River virus ^b	45	52	76	102	113	66	42	46	61	76	84	78	841
Other ^b	9	10	11	7	6	4	9	7	1	4	5	11	84
Blood lead level ≥ 15ug/dL ^b	7	7	26	9	24	15	38	47	36	23	20	11	263
Botulism	0	0	0	0	0	0	0	0	0	0	0	0	0
Brucellosis ^b	1	1	1	0	0	0	0	0	0	0	0	0	3
Chancroid ^b	0	0	0	0	0	0	0	0	0	0	0	0	0
<i>Chlamydia trachomatis</i> infection	1039	1196	1203	913	1080	958	1011	970	927	1138	1157	855	12447
Congenital chlamydia ^b	2	2	3	3	3	2	4	3	3	0	3	3	31
Chlamydia – other ^b	1037	1194	1200	910	1077	956	1007	967	924	1138	1154	852	12416
Cholera ^b	0	0	1	0	0	1	0	0	0	0	0	0	2
Creutzfeldt–Jakob disease ^b	1	0	0	3	0	2	1	0	0	0	0	0	7
Cryptosporidiosis ^b	37	42	25	32	33	16	18	15	22	35	157	112	544
Foodborne illness (NOS) ^e	77	67	395	21	38	21	30	15	34	38	27	0	763
Gastroenteritis (institutional)	154	221	423	438	562	873	1794	1471	1543	1679	935	395	10488
Giardiasis ^b	161	191	245	151	185	159	147	149	122	122	190	118	1940
Gonorrhoea ^b	144	119	133	118	132	116	87	91	96	125	98	125	1384
Haemolytic uraemic syndrome	2	1	1	1	0	0	1	0	1	1	2	3	13
<i>Haemophilus influenzae</i> serotype b	0	0	0	0	0	1	1	2	0	1	2	0	7
Hib epiglottitis ^b	0	0	0	0	0	0	0	0	0	0	1	0	1
Hib meningitis ^b	0	0	0	0	0	1	0	0	0	0	1	0	2
Hib septicaemia ^b	0	0	0	0	0	0	1	1	0	0	0	0	2
Hib infection NOS ^b	0	0	0	0	0	0	0	1	0	1	0	0	2
Hepatitis A ^b	9	11	3	3	4	5	8	6	2	3	5	6	65
Hepatitis B	242	204	268	224	224	219	232	215	205	210	240	173	2656
Hepatitis B – acute viral ^b	9	4	4	3	8	7	2	1	5	1	11	1	56
Hepatitis B – other ^b	233	200	264	221	216	212	230	214	200	209	229	172	2600
Hepatitis C	372	344	422	323	399	330	331	384	357	378	346	273	4259
Hepatitis C – acute viral ^b	4	9	3	4	7	3	8	7	4	2	2	0	53
Hepatitis C – other ^b	368	335	419	319	392	327	323	377	353	376	344	273	4206
Hepatitis D ^b	2	0	1	2	2	0	2	0	1	1	0	0	11
Hepatitis E ^b	0	0	4	0	0	0	1	1	0	1	0	1	8
HIV infection ^b	34	45	32	34	47	23	31	35	27	31	35	30	404
Influenza	37	33	37	51	26	90	583	754	179	66	39	23	1918
Influenza-Type A ^b	29	18	30	30	15	68	526	601	102	35	24	9	1487
Influenza-Type B ^b	3	12	5	8	5	10	27	38	29	22	13	8	180
Influenza-Type A & B ^b	3	0	2	10	3	6	4	6	5	3	1	0	43
Influenza-Type NOS ^b	2	3	0	3	3	6	26	109	43	6	1	6	208
Legionellosis	14	12	8	13	7	10	7	2	4	4	12	12	105
<i>Legionella longbeachae</i> ^b	3	5	3	6	1	2	3	0	1	1	2	2	29
<i>L.pneumophila</i> ^b	11	7	5	7	6	8	4	1	3	3	8	10	73
Legionnaire disease other	0	0	0	0	0	0	0	1	0	0	2	0	3
Leprosy	1	1	1	0	0	0	0	0	0	0	0	1	4
Leptospirosis ^b	1	1	3	0	0	0	0	1	1	0	1	0	8
Listeriosis ^b	3	2	1	3	1	1	2	2	1	0	4	2	22
Lymphogranuloma venereum (LGV) ^b	0	0	0	0	0	0	0	0	0	0	0	0	0
Malaria ^b	9	9	10	5	8	6	5	12	10	12	7	5	98
Measles	0	1	1	0	1	0	1	0	0	0	0	0	4
Measles laboratory confirmed	0	1	1	0	1	0	1	0	0	0	0	0	4
Measles – other	0	0	0	0	0	0	0	0	0	0	0	0	0
Meningococcal disease	7	0	10	5	7	9	13	21	9	12	8	11	112
Meningococcal – serogroup B ^b	6	0	4	2	2	5	8	17	8	7	7	10	76
Meningococcal – serogroup C ^b	1	0	3	2	2	0	0	0	1	0	1	0	10
Meningococcal – serogroup W135 ^b	0	0	1	0	0	1	0	0	0	0	0	0	2
Meningococcal – serogroup Y ^b	0	0	0	0	0	2	1	0	0	1	0	1	5
Meningococcal – other	0	0	2	1	3	1	4	4	0	4	0	0	19
Mumps ^b	15	4	5	11	29	17	15	27	38	56	59	47	323
Pertussis	122	136	100	112	167	174	211	180	177	268	255	191	2093
Pneumococcal disease (invasive) ^b	25	18	30	36	35	61	85	78	48	43	38	25	522
Psittacosis ^b	5	3	3	5	5	1	0	2	1	5	3	1	34
Q fever ^b	23	18	14	14	20	15	15	15	16	21	24	20	215
Rubella	0	2	1	0	1	4	0	0	0	0	1	0	9
Congenital rubella ^b	0	0	0	0	0	1	0	0	0	0	0	0	1
Rubella – other ^b	0	2	1	0	1	3	0	0	0	0	1	0	8
<i>Salmonella</i> infection ^{b,d}	233	314	510	317	186	126	108	114	115	155	202	184	2564
Shigellosis ^b	4	4	9	7	8	4	6	12	3	4	4	6	71
Syphilis	93	93	107	78	105	97	78	118	83	85	98	80	1115
Congenital syphilis	0	1	1	0	0	0	0	0	0	0	1	1	4
Infectious syphilis ^{b,c}	40	38	29	33	41	43	35	41	27	30	49	28	434
Syphilis – other ^b	53	54	77	45	64	54	43	77	56	55	48	51	677
Tetanus	0	0	0	0	0	0	1	0	0	0	0	1	2
Tuberculosis ^b	54	45	49	38	27	46	31	36	37	41	28	20	452
Typhoid ^b	2	6	3	6	2	1	3	2	0	0	1	0	26
Verotoxin - producing <i>Escherichia coli</i> infections ^b	1	1	1	1	1	0	0	0	0	5	8	5	23

^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. Due to data delay AIDS notifications will be reported in a later edition.

Table 4. Disease notifications by Area Health Service of residence (2005 AHS boundaries), crude rates per 100 000 population, NSW, 2007

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	7.12	8.14	2.22	4.83	5.79	2.57	1.68	1.39	2.46
Anthrax	0	0	0	0	0	0	0	0	0
Arboviral infection	22.11	64.13	66.71	84.07	15.62	57.46	38.05	76.72	74.85
Barmah Forest virus ^b	2.62	50.25	4.45	9.66	2.31	20.24	9.51	31.24	39
Ross River virus ^b	19.12	12.92	62.26	73.44	12.73	36.88	27.42	44.09	31.98
Other ^b	0.37	0.96	0	0.97	0.58	0.34	1.12	1.39	3.87
Blood lead level \geq 15ug/dL ^b	3	1.91	11.12	71.5	4.63	3.77	0.56	0.69	1.05
Botulism	0	0	0	0	0	0	0	0	0
Brucellosis ^b	0	0	0	0	0.58	0	0	0	0
Chancroid ^b	0	0	0	0	0	0	0	0	0
<i>Chlamydia trachomatis</i> infection	176.6	126.3	231.3	143	206.6	229	232.2	123.6	217.2
Congenital chlamydia ^b	0.37	0.96	2.22	0	0.58	0.34	0	0.35	0.7
Chlamydia – other ^b	176.2	125.4	229	143	206	228.7	232.2	123.2	216.5
Cholera ^b	0	0	0	0	0	0	0	0	0
Creutzfeldt–Jakob disease ^b	0	0	0	0	0	0.17	0	0	0
Cryptosporidiosis ^b	19.49	6.7	4.45	16.43	19.1	7.21	35.26	9.37	17.22
Giardiasis ^b	17.62	15.31	8.89	42.52	16.78	28.65	33.02	19.09	5.97
Gonorrhoea ^b	5.25	1.91	0	3.87	6.37	12.87	5.6	2.43	14.76
Haemolytic uraemic syndrome	0	0	0	0	0.58	0.86	0.56	0	0
<i>H.influenzae</i> serotype b	0.37	0	0	0	0	0.17	0	0.35	0
Hib epiglottitis ^b	0	0	0	0	0	0	0	0	0
Hib meningitis ^b	0.37	0	0	0	0	0	0	0.35	0
Hib septicaemia ^b	0	0	0	0	0	0.17	0	0	0
Hib infection NOS ^b	0	0	0	0	0	0	0	0	0
Hepatitis A ^b	0	0	0	0.97	0.58	0.17	0	0.35	1.76
Hepatitis B	13.49	11.01	22.24	9.67	1.16	8.4	10.63	5.9	12.65
Hepatitis B – acute viral ^b	0.75	1.44	0	0.97	0	1.37	0	0	0.7
Hepatitis B – other ^b	12.74	9.57	22.24	8.7	1.16	7.03	10.63	5.9	11.95
Hepatitis C	38.98	55.04	77.82	68.6	61.34	55.07	50.93	48.25	76.25
Hepatitis C – acute viral ^b	0.37	1.44	6.67	4.83	0.58	0.69	1.68	0	0
Hepatitis C – other ^b	38.61	53.6	71.15	63.77	60.76	54.38	49.25	48.25	76.25
Hepatitis D ^b	0	0	0	0	0	0	0	0	0
Hepatitis E ^b	0	0	0	0	0	0	0	0	0
HIV infection ^b	0.75	1.44	0	1.93	1.16	3.09	0.56	1.39	1.41
Influenza	14.98	35.9	22.24	19.33	37.04	37.22	45.33	15.27	58.32
Influenza-Type A ^b	13.87	33.5	22.24	16.43	34.72	32.42	40.85	14.23	21.43
Influenza-Type B ^b	0.37	1.44	0	2.9	1.74	4.8	3.36	0	1.05
Influenza-Type A & B ^b	0.37	0.96	0	0	0	0	0.56	0	1.05
Influenza-Type NOS ^b	0.37	0	0	0	0.58	0	0.56	1.04	34.79
Legionellosis	1.12	2.39	0	0	0	0.85	2.24	1.38	1.05
<i>L. longbeachae</i> ^b	0.37	0.48	0	0	0	0.34	1.12	0.69	0
<i>L. pneumophila</i> ^b	0	1.91	0	0	0	0.51	0.56	0.69	1.05
Legionnaire disease other	0.75	0	0	0	0	0	0.56	0	0
Leprosy	0	0	0	0	0	0	0	0	0
Leptospirosis ^b	0	0	0	0.97	0	0.17	0.56	0.69	1.05
Listeriosis ^b	0	0	2.22	0	0	0.86	0	0	0
Lymphogranuloma venereum (LGV) ^b	0	0	0	0	0	0	0	0	0
Malaria ^b	1.12	2.87	0	0	0.58	2.4	1.12	1.39	0.7
Measles	0	0	0	0	0	0	0	0	0
Measles laboratory confirmed	0	0	0	0	0	0	0	0	0
Measles – other	0	0	0	0	0	0	0	0	0
Meningococcal disease	1.49	2.87	0	2.9	1.16	1.54	1.68	0.69	2.1
Meningococcal – serogroup B ^b	0.75	2.39	0	2.9	1.16	1.03	1.12	0.69	1.05
Meningococcal – serogroup C ^b	0.37	0	0	0	0	0.17	0	0	0.7
Meningococcal – serogroup W135 ^b	0	0.48	0	0	0	0	0	0	0
Meningococcal – serogroup Y ^b	0	0	0	0	0	0	0	0	0
Meningococcal – other	0.37	0	0	0	0	0.34	0.56	0	0.35
Mumps ^b	0.75	0	0	0	1.16	0.86	0.56	0	0
Pertussis	23.99	26.32	13.34	55.08	10.42	34.31	35.81	16.66	32.33
Pneumococcal disease (invasive) ^b	7.87	6.7	15.57	14.49	6.94	10.98	9.51	9.03	7.03
Psittacosis ^b	1.5	0.48	2.22	1.93	1.74	0.86	0	0.35	0.7
Q fever ^b	1.5	6.22	6.67	44.45	4.63	3.6	31.9	5.55	9.49
Rubella	0	0	0	2.9	0	0	0.56	0	0
Congenital rubella ^b	0	0	0	0	0	0	0	0	0
Rubella – other ^b	0	0	0	2.9	0	0	0.56	0	0
<i>Salmonella</i> infection ^{b,d}	31.86	26.8	15.57	27.06	27.2	32.59	43.65	27.08	76.6
Shigellosis ^b	0	1.44	0	0.97	0	0.51	0.56	1.04	2.81
Syphilis	3.37	3.83	31.13	15.46	9.26	4.29	4.48	9.37	4.22
Congenital syphilis	0	0	0	0	0.58	0	0	0	0
Infectious syphilis ^{b,c}	0.37	0.96	2.22	0	1.74	2.06	1.12	0.69	1.76
Syphilis – other ^b	3	2.87	28.91	15.46	6.94	2.23	3.36	8.68	2.46
Tetanus	0	0	0	0	0	0	0	0	0.35
Tuberculosis ^b	1.12	2.87	0	0	0.58	2.74	0.56	1.39	1.41
Typhoid ^b	0.37	0	0	0	0	0	0	0	0
Verotoxin-producing <i>Escherichia coli</i> infections ^b	1.12	0.48	0	0	0	1.54	2.24	0	0.35

^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. ^eAHS further divided into the geographical region covered by their component Public Health Unit.

^fRate is based on a denominator of 8000 persons. ^hIncludes cases with unknown PHU. NOS: not otherwise specified. No case of the following diseases have been notified since 1991: Plague^e, Diphtheria^e, Granuloma inguinale^e, Lyssavirus^e, Poliomyelitis^e, Rabies, Smallpox, Typhus^e, Viral haemorrhagic fever, Yellow fever.

Due to data delay AIDS notifications will be reported in a later edition.

Table 4. continued

Condition	Northern Sydney Central Coast ^f		South Eastern Sydney Illawara ^f		Sydney South West ^f		Sydney West ^f		Justice Health
	Gosford	Hornsby	Wollongong	Randwick	Camperdown	Liverpool	Penrith	Parramatta	
Adverse event after immunisation	4.84	1.73	5.08	3.07	1.14	1.93	5.98	3.63	0
Anthrax	0	0	0	0	0	0	0	0	0
Arboviral infection	21.63	4.96	26.74	5.28	3.42	1.68	4.41	3.12	12.5
Barmah Forest virus ^b	5.17	0.74	18.45	0.49	0.57	0.24	0.63	0.78	12.5
Ross River virus ^b	14.85	2.73	7.49	2.09	1.52	0.84	3.15	1.95	0
Other ^b	1.61	1.49	0.8	2.7	1.33	0.6	0.63	0.39	0
Blood lead level ≥ 15ug/dL ^b	0.97	1.24	5.61	1.59	2.66	2.77	2.52	3.89	0
Botulism	0	0	0	0	0	0	0	0	0
Brucellosis ^b	0	0	0	0.12	0	0.12	0	0	0
Chancroid ^b	0	0	0	0	0	0	0	0	0
<i>Chlamydia trachomatis</i> infection	186.9	133.3	154.6	280.1	253.8	99.9	128.8	135.1	1188
Congenital chlamydia ^b	0.32	0.37	0	0.12	0.19	0.36	0.63	1.17	0
Chlamydia – other ^b	186.6	132.9	154.6	279.9	253.6	99.54	128.2	134	1188
Cholera ^b	0	0.12	0	0	0.19	0	0	0	0
Creutzfeldt–Jakob disease ^b	0.32	0	0.53	0.12	0	0.12	0.31	0	0
Cryptosporidiosis ^b	7.1	5.33	4.01	4.66	4.18	5.79	6.61	4.41	0
Giardiasis ^b	26.15	38.77	22.19	40.6	38.21	14.36	26.76	34.62	25
Gonorrhoea ^b	9.36	15.48	8.56	57.53	56.46	11.58	11.97	12.45	100
Haemolytic uraemic syndrome	0	0	0.27	0.12	0.19	0.12	0	0.13	0
<i>H.influenzae</i> serotype b	0	0.12	0	0.12	0	0.12	0	0.13	0
Hib epiglottitis ^b	0	0.12	0	0	0	0	0	0	0
Hib meningitis ^b	0	0	0	0	0	0	0	0	0
Hib septicaemia ^b	0	0	0	0	0	0	0	0.13	0
Hib infection NOS ^b	0	0	0	0.12	0	0.12	0	0	0
Hepatitis A ^b	0	1.61	0.8	1.23	1.14	1.45	0.94	1.17	0
Hepatitis B	10.66	35.42	12.3	47.35	81.17	72.39	12.28	72.23	550
Hepatitis B – acute viral ^b	0.65	0.74	0	1.72	0.57	1.45	0.31	0.13	12.5
Hepatitis B – other ^b	10.01	34.68	12.3	45.63	80.6	70.94	11.97	72.1	537.5
Hepatitis C	60.05	25.15	49.47	48.46	75.85	59.72	50.06	49.15	7675
Hepatitis C – acute viral ^b	0	0	0	0.25	3.99	0.6	0	0.13	50
Hepatitis C – other ^b	60.05	25.15	49.47	48.21	71.86	59.12	50.06	49.02	7625
Hepatitis D ^b	0.32	0	0.53	0.37	0	0	0.31	0.52	0
Hepatitis E ^b	0.32	0.12	0	0.37	0.57	0	0	0	0
HIV infection ^b	2.58	3.84	2.41	15.7	18.44	2.9	0.94	3.89	0
Influenza	17.43	17.59	16.31	22.69	8.55	14.12	39.04	60.04	37.5
Influenza-Type A ^b	12.27	10.16	12.3	14.35	5.51	9.29	31.49	55.89	37.5
Influenza-Type B ^b	0.32	0.74	2.41	6.38	2.85	1.21	4.41	3.37	0
Influenza-Type A & B ^b	0	0.25	1.07	1.96	0	0	2.83	0.65	0
Influenza-Type NOS ^b	4.84	6.44	0.53	0	0.19	3.62	0.31	0.13	0
Legionellosis	1.3	0.99	1.6	1.6	1.52	1.57	3.15	2.47	0
<i>L. longbeachae</i> ^b	0.65	0.25	1.07	0.25	0	0.48	0.63	0.65	0
<i>L. pneumophila</i> ^b	0.65	0.74	0.53	1.35	1.52	1.09	2.52	1.82	0
Legionnaire disease other	0	0	0	0	0	0	0	0	0
Leprosy	0	0	0	0	0	0	0	0.52	0
Leptospirosis ^b	0	0	0	0	0	0	0	0	0
Listeriosis ^b	0	0.37	0.53	0.61	0.19	0.24	0.31	0.26	0
Lymphogranuloma venereum (LGV) ^b	0	0	0	0	0	0	0	0	0
Malaria ^b	0.65	1.24	1.6	0.98	1.71	0.6	1.89	2.07	0
Measles	0	0.12	0	0.12	0	0.12	0	0	0
Measles laboratory confirmed	0	0.12	0	0.12	0	0.12	0	0	0
Measles – other	0	0	0	0	0	0	0	0	0
Meningococcal disease	1.94	1.6	1.07	2.08	0.95	1.45	1.57	1.82	0
Meningococcal – serogroup B ^b	0.97	1.36	0.8	1.1	0.76	1.09	1.26	1.04	0
Meningococcal – serogroup C ^b	0.32	0	0.27	0.37	0.19	0	0	0	0
Meningococcal – serogroup W135 ^b	0	0	0	0	0	0.12	0	0	0
Meningococcal – serogroup Y ^b	0.65	0.12	0	0.12	0	0	0	0.13	0
Meningococcal – other	0	0.12	0	0.49	0	0.24	0.31	0.65	0
Mumps ^b	0.32	6.32	3.21	16.68	5.89	3.26	3.15	5.19	0
Pertussis	24.54	34.81	20.86	39.13	33.65	18.46	27.39	41.11	0
Pneumococcal disease (invasive) ^b	7.1	6.94	9.63	6.13	7.79	5.31	7.87	6.22	12.5
Psittacosis ^b	0	0	0.8	0.25	0	0.24	1.57	0.39	0
Q fever ^b	0.97	0.12	3.21	0.12	0	0	0.31	0.26	0
Rubella	0	0.12	0	0	0.19	0	0	0.39	0
Congenital rubella ^b	0	0	0	0	0	0	0	0.13	0
Rubella – other ^b	0	0.12	0	0	0.19	0	0	0.26	0
<i>Salmonella</i> infection ^{b,d}	45.84	41.99	22.99	36.19	45.43	30.28	32.12	39.68	12.5
Shigellosis ^b	0	1.61	0.27	1.84	2.28	0.72	0	0.52	0
Syphilis	6.78	6.69	8.29	40.85	47.72	16.65	8.5	12.32	237.5
Congenital syphilis	0	0.12	0	0	0	0	0	0.26	0
Infectious syphilis ^{b,c}	0.32	2.11	1.87	29.2	17.49	1.33	1.26	3.63	25
Syphilis – other ^b	6.46	4.46	6.42	11.65	30.23	15.32	7.24	8.43	212.5
Tetanus	0	0	0.27	0	0	0	0	0	0
Tuberculosis ^b	1.29	5.33	2.67	7.73	12.93	9.77	4.09	16.6	0
Typhoid ^b	0	0.25	0	0.25	0.57	0.48	0	1.69	0
Verotoxin-producing <i>Escherichia coli</i> infections ^b	0	0.12	0	0	0	0.12	0.63	0.13	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. ^fAHS further divided into the geographical region covered by their component Public Health Unit.

^eRate is based on a denominator of 8000 persons. ^bIncludes cases with unknown PHU. 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.

Due to data delay AIDS notifications will be reported in a later edition.

Table 5. Disease notifications by Area Health Service of residence (2005 AHS boundaries)

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	19	17	1	5	10	15	3	4	7
Anthrax	0	0	0	0	0	0	0	0	0
Arboviral infection	59	134	30	87	27	335	68	221	213
Barmah Forest virus ^b	7	105	2	10	4	118	17	90	111
Ross River virus ^b	51	27	28	76	22	215	49	127	91
Other ^b	1	2	0	1	1	2	2	4	11
Blood lead level ≥ 15ug/dL ^b	8	4	5	74	8	22	1	2	3
Botulism	0	0	0	0	0	0	0	0	0
Bruceellosis ^b	0	0	0	0	1	0	0	0	0
Chancroid ^b	0	0	0	0	0	0	0	0	0
<i>Chlamydia trachomatis</i> infection	471	264	104	148	357	1335	415	356	618
Congenital chlamydia ^b	1	2	1	0	1	2	0	1	2
Chlamydia – other ^b	470	262	103	148	356	1333	415	355	616
Cholera ^b	0	0	0	0	0	0	0	0	0
Creutzfeldt–Jakob disease ^b	0	0	0	0	0	1	0	0	0
Cryptosporidiosis ^b	52	14	2	17	33	42	63	27	49
Gastroenteritis (institutional)	120	583	247	38	60	1929	167	65	583
Giardiasis ^b	47	32	4	44	29	167	59	55	17
Gonorrhoea ^b	14	4	0	4	11	75	10	7	42
Haemolytic uraemic syndrome	0	0	0	0	1	5	1	0	0
<i>Haemophilus influenzae</i> serotype b	1	0	0	0	0	1	0	1	0
Hib epiglottitis ^b	0	0	0	0	0	0	0	0	0
Hib meningitis ^b	1	0	0	0	0	0	0	1	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	1	1	1	0	1	5
Hepatitis B	36	23	10	10	2	49	19	17	36
Hepatitis B – acute viral ^b	2	3	0	1	0	8	0	0	2
Hepatitis B – other ^b	34	20	10	9	2	41	19	17	34
Hepatitis C	104	115	35	71	106	321	91	139	217
Hepatitis C – acute viral ^b	1	3	3	5	1	4	3	0	0
Hepatitis C – other ^b	103	112	32	66	105	317	88	139	217
Hepatitis D ^b	0	0	0	0	0	0	0	0	0
Hepatitis E ^b	0	0	0	0	0	0	0	0	0
HIV infection ^b	2	3	0	2	2	18	1	4	4
Influenza	40	75	10	20	64	217	81	44	166
Influenza-Type A ^b	37	70	10	17	60	189	73	41	61
Influenza-Type B ^b	1	3	0	3	3	28	6	0	3
Influenza-Type A & B ^b	1	2	0	0	0	0	1	0	3
Influenza-Type NOS ^b	1	0	0	0	1	0	1	3	99
Legionellosis	3	5	0	0	0	5	4	4	3
<i>Legionella longbeachae</i> ^b	1	1	0	0	0	2	2	2	0
<i>L. pneumophila</i> ^b	0	4	0	0	0	3	1	2	3
Legionnaire disease other	2	0	0	0	0	0	1	0	0
Leprosy	0	0	0	0	0	0	0	0	0
Leptospirosis ^b	0	0	0	1	0	1	1	2	3
Listeriosis ^b	0	0	1	0	0	5	0	0	0
Lymphogranuloma venereum (LGV) ^b	0	0	0	0	0	0	0	0	0
Malaria ^b	3	6	0	0	1	14	2	4	2
Measles	0	0	0	0	0	0	0	0	0
Measles laboratory confirmed	0	0	0	0	0	0	0	0	0
Measles – other	0	0	0	0	0	0	0	0	0
Meningococcal disease	4	6	0	3	2	9	3	2	6
Meningococcal – serogroup B ^b	2	5	0	3	2	6	2	2	3
Meningococcal – serogroup C ^b	1	0	0	0	0	1	0	0	2
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	0	0	0	0	2	1	0	1
Mumps ^b	2	0	0	0	2	5	1	0	0
Pertussis	64	55	6	57	18	200	64	48	92
Pneumococcal disease (invasive) ^b	21	14	7	15	12	64	17	26	20
Psittacosis ^b	4	1	1	2	3	5	0	1	2
Q fever ^b	4	13	3	46	8	21	57	16	27
Rubella	0	0	0	3	0	0	1	0	0
Congenital rubella ^b	0	0	0	0	0	0	0	0	0
Rubella – other ^b	0	0	0	3	0	0	1	0	0
<i>Salmonella</i> infection ^{b,d}	85	56	7	28	47	190	78	78	218
Shigellosis ^b	0	3	0	1	0	3	1	3	8
Syphilis	9	8	14	16	16	25	8	27	12
Congenital syphilis	0	0	0	0	1	0	0	0	0
Infectious syphilis ^{b,c}	1	2	1	0	3	12	2	2	5
Syphilis – other ^b	8	6	13	16	12	13	6	25	7
Tetanus	0	0	0	0	0	0	0	0	1
Tuberculosis ^b	3	6	0	0	1	16	1	4	4
Typhoid ^b	1	0	0	0	0	0	0	0	0
Verotoxin-producing <i>Escherichia coli</i> infections ^b	3	1	0	0	0	9	4	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. ^eAHS further divided into the geographical region covered by their component public health unit.

^fRate is based on a denominator of 8000 persons. ^hIncludes cases with unknown PHU. NOS: not otherwise specified. No case of the following diseases have been notified since 1991: Plague^b, Diphtheria^a, Granuloma inguinale^b, Lyssavirus^b, Poliomyelitis^b, Rabies, Smallpox, Typhus^b, Viral haemorrhagic fever, Yellow fever.

Due to data delay AIDS notifications will be reported in a later edition.

Table 5. continued

Condition	Northern Sydney Central Coast ^f		South Eastern Sydney Illawara ^f		Sydney South West ^f		Sydney West ^f		Justice Health	Total
	Gosford	Hornsby	Wollongong	Randwick	Camperdown	Liverpool	Penrith	Parramatta		
Adverse event after immunisation	15	14	19	25	6	16	19	28	0	224
Anthrax	0	0	0	0	0	0	0	0	0	0
Arboviral infection	67	40	100	43	18	14	14	24	1	1498
Barmah Forest virus ^b	16	6	69	4	3	2	2	6	1	573
Ross River virus ^b	46	22	28	17	8	7	10	15	0	841
Other ^b	5	12	3	22	7	5	2	3	0	84
Blood lead level ≥ 15ug/dL ^b	3	10	21	13	14	23	8	30	0	263
Botulism	0	0	0	0	0	0	0	0	0	0
Brucellosis ^b	0	0	0	1	0	1	0	0	0	3
Chancroid ^b	0	0	0	0	0	0	0	0	0	0
<i>Chlamydia trachomatis</i> infection	579	1076	578	2283	1335	828	409	1042	95	12447
Congenital chlamydia ^b	1	3	0	1	1	3	2	9	0	31
Chlamydia – other ^b	578	1073	578	2282	1334	825	407	1033	95	12416
Cholera ^b	0	1	0	0	1	0	0	0	0	2
Creutzfeldt–Jakob disease ^b	1	0	2	1	0	1	1	0	0	7
Cryptosporidiosis ^b	22	43	15	38	22	48	21	34	0	544
Gastroenteritis (institutional)	431	1908	366	878	701	770	78	1552	12	10488
Giardiasis ^b	81	313	83	331	201	119	85	267	2	1940
Gonorrhoea ^b	29	125	32	469	297	96	38	96	8	1384
Haemolytic uraemic syndrome	0	0	1	1	1	1	0	1	0	13
<i>Haemophilus influenzae</i> serotype b	0	1	0	1	0	1	0	1	0	7
Hib epiglottitis ^b	0	1	0	0	0	0	0	0	0	1
Hib meningitis ^b	0	0	0	0	0	0	0	0	0	2
Hib septicaemia ^b	0	0	0	0	0	0	0	1	0	2
Hib infection NOS ^b	0	0	0	1	0	1	0	0	0	2
Hepatitis A ^b	0	13	3	10	6	12	3	9	0	65
Hepatitis B	33	286	46	386	427	600	39	557	44	2656
Hepatitis B – acute viral ^b	2	6	0	14	3	12	1	1	1	56
Hepatitis B – other ^b	31	280	46	372	424	588	38	556	43	2600
Hepatitis C	186	203	185	395	399	495	159	379	614	4259
Hepatitis C – acute viral ^b	0	0	0	2	21	5	0	1	4	53
Hepatitis C – other ^b	186	203	185	393	378	490	159	378	610	4206
Hepatitis D ^b	1	0	2	3	0	0	1	4	0	11
Hepatitis E ^b	1	1	0	3	3	0	0	0	0	8
HIV infection ^b	8	31	9	128	97	24	3	30	0	404
Influenza	54	142	61	185	45	117	124	463	3	1918
Influenza-Type A ^b	38	82	46	117	29	77	100	431	3	1487
Influenza-Type B ^b	1	6	9	52	15	10	14	26	0	180
Influenza-Type A & B ^b	0	2	4	16	0	0	9	5	0	43
Influenza-Type NOS ^b	15	52	2	0	1	30	1	1	0	208
Legionellosis	4	8	6	13	8	13	10	19	0	105
<i>Legionella longbeachae</i> ^b	2	2	4	2	0	4	2	5	0	29
<i>L. pneumophila</i> ^b	2	6	2	11	8	9	8	14	0	73
Legionnaire disease other	0	0	0	0	0	0	0	0	0	3
Leprosy	0	0	0	0	0	0	0	4	0	4
Leptospirosis ^b	0	0	0	0	0	0	0	0	0	8
Listeriosis ^b	0	3	2	5	1	2	1	2	0	22
Lymphogranuloma venereum (LGV) ^b	0	0	0	0	0	0	0	0	0	0
Malaria ^b	2	10	6	8	9	5	6	16	0	98
Measles	0	1	0	1	0	1	0	0	0	4
Measles laboratory confirmed	0	1	0	1	0	1	0	0	0	4
Measles – other	0	0	0	0	0	0	0	0	0	0
Meningococcal disease	6	13	4	17	5	12	5	14	0	112
Meningococcal – serogroup B ^b	3	11	3	9	4	9	4	8	0	76
Meningococcal – serogroup C ^b	1	0	1	3	1	0	0	0	0	10
Meningococcal – serogroup W135 ^b	0	0	0	0	0	1	0	0	0	2
Meningococcal – serogroup Y ^b	2	1	0	1	0	0	0	1	0	5
Meningococcal – other	0	1	0	4	0	2	1	5	0	19
Mumps ^b	1	51	12	136	31	27	10	40	0	323
Pertussis	76	281	78	319	177	153	87	317	0	2093
Pneumococcal disease (invasive) ^b	22	56	36	50	41	44	25	48	1	522
Psittacosis ^b	0	0	3	2	0	2	5	3	0	34
Q fever ^b	3	1	12	1	0	0	1	2	0	215
Rubella	0	1	0	0	1	0	0	3	0	9
Congenital rubella ^b	0	0	0	0	0	0	0	1	0	1
Rubella – other ^b	0	1	0	0	1	0	0	2	0	8
<i>Salmonella</i> infection ^{b,d}	142	339	86	295	239	251	102	306	1	2564
Shigellosis ^b	0	13	1	15	12	6	0	4	0	71
Syphilis	21	54	31	333	251	138	27	95	19	1115
Congenital syphilis	0	1	0	0	0	0	0	2	0	4
Infectious syphilis ^{b,c}	1	17	7	238	92	11	4	28	2	434
Syphilis – other ^b	20	36	24	95	159	127	23	65	17	677
Tetanus	0	0	1	0	0	0	0	0	0	2
Tuberculosis ^b	4	43	10	63	68	81	13	128	0	452
Typhoid ^b	0	2	0	2	3	4	0	13	0	26
Verotoxin-producing <i>Escherichia coli</i> infections ^b	0	1	0	0	0	1	2	1	0	23

^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. ^eAHS further divided into the geographical region covered by their component public health unit. ^fRate is based on a denominator of 8000 persons. ^gIncludes cases with unknown PHU. 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. Due to data delay AIDS notifications will be reported in a later edition.

- *Chlamydia trachomatis* infections account for the most notifications in adults with rates peaking at 818 per 100 000 in people aged between 16 and 24 years.
- Influenza is the most commonly reported notifiable disease in adults aged 65 years and older though this rate is markedly lower than that observed in children aged less than five years of age. Children and older adults are more likely to undergo testing for influenza.

Outbreaks and threats

Several notable disease outbreaks and threats were reported in 2007 in NSW. These included:

- an outbreak of Legionnaire disease in South East Sydney Illawarra AHS related to a contaminated cooling tower in Circular Quay in Sydney (January 2007).²
- hepatitis C transmission linked to a general medical

practice in South East Sydney Illawarra Health Service that specialised in provision of vitamin and mineral injections (March 2007).³

- a sushi chef who was working while infectious with hepatitis A. Sydney South West Area Health Service provided immunoglobulin to over 400 people who had eaten potentially contaminated sushi. No subsequent hepatitis A cases were reported (March 2007).³
- a *Salmonella* infection outbreak associated with eating pork and chicken rolls from a bakery in Sydney South West Area Health Service (March 2007).³

Conclusions

Controlling the spread of sexually transmitted infections, in particular, remains a priority for NSW. This is evident in the re-emergence of infectious syphilis in the gay community and the high rates of *Chlamydia trachomatis* infections in young adults.

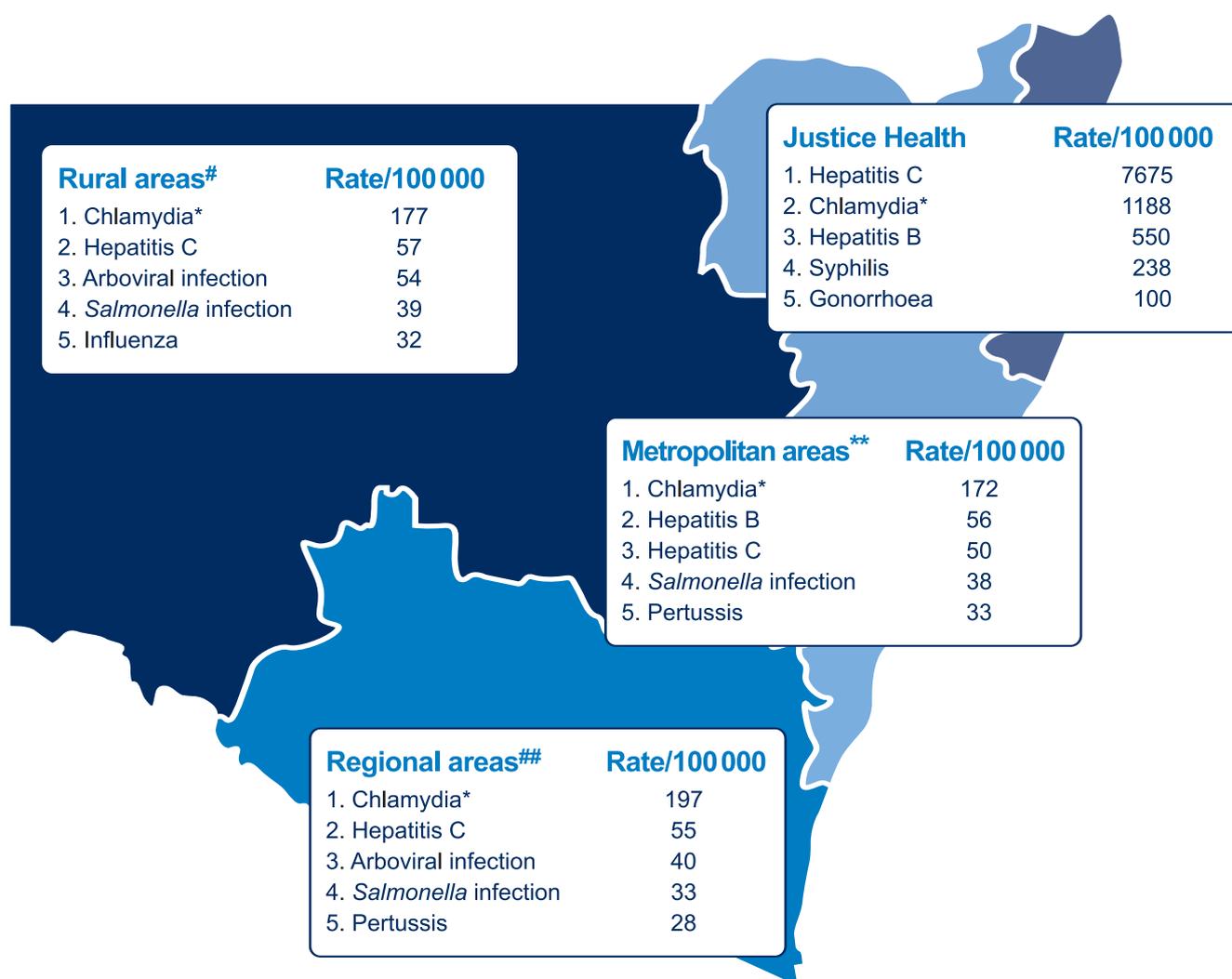


Figure 1. The five most commonly reported notifiable diseases by geographical area of residence at the time of notification in NSW, 2007. # Includes Greater Southern, Greater Western, Hunter New England (Tamworth region) and North Coast Area Health Services. ## Includes Northern Sydney Central Coast (Gosford region), South East Sydney Illawarra (Wollongong region) and Hunter New England (Newcastle region). *Refers to notifications of *Chlamydia trachomatis*. ** Includes Northern Sydney Central Coast (Hornsby region), South East 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 the case, NSW, 2006

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	15	18	133	5	21	2	16	2	6	6	191	33	224
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0
Arboviral infection	6	1	108	74	285	242	283	300	88	109	770	726	1498
Barmah Forest virus ^b	3	0	35	24	91	91	130	134	30	33	289	282	573
Ross River virus ^b	3	1	64	44	175	132	143	150	56	73	441	400	841
Other ^b	0	0	9	6	19	19	10	16	2	3	40	44	84
Blood lead level ≥ 15ug/dL ^b	0	9	3	46	7	109	3	71	1	14	14	249	263
Botulism													0
Brucellosis ^b	0	0	0	2	0	0	0	0	0	1	0	3	3
Chancroid ^b													0
<i>Chlamydia trachomatis</i> infection	22	18	4783	2299	2172	2571	111	400	9	24	7097	5313	12447
Congenital chlamydia ^b	14	13	3	0	0	0	0	0	0	0	18	13	31
Chlamydia – other ^b	8	5	4780	2299	2172	2571	111	400	9	24	7079	5300	12416
Cholera ^b	0	0	0	0	0	1	0	1	0	0	0	2	2
Creutzfeldt-Jakob disease ^b	0	0	0	0	0	0	3	1	2	1	5	2	7
Cryptosporidiosis ^b	80	108	78	122	61	50	19	17	4	4	242	301	544
Giardiasis ^b	227	329	159	202	375	259	137	121	74	51	973	963	1940
Gonorrhoea ^b	0	0	102	245	84	731	19	195	1	4	206	1175	1384
Haemolytic uraemic syndrome	2	3	1	2	2	0	1	1	1	0	7	6	13
<i>Haemophilus influenzae</i> serotype b	3	1	0	1	0	0	1	1	0	0	4	3	7
Hib epiglottitis ^b	0	0	0	0	0	0	0	1	0	0	0	1	1
Hib meningitis ^b	2	0	0	0	0	0	0	0	0	0	2	0	2
Hib septicaemia ^b	0	1	0	0	0	0	1	0	0	0	1	1	2
Hib infection NOS ^b	1	0	0	1	0	0	0	0	0	0	1	1	2
Hepatitis A ^b	4	3	7	12	11	15	4	5	2	2	28	37	65
Hepatitis B	6	7	232	215	694	748	242	384	45	64	1219	1418	2656
Hepatitis B – acute viral ^b	1	0	8	5	15	13	2	9	1	1	27	28	56
Hepatitis B – other ^b	5	7	224	210	679	735	240	375	44	63	1192	1390	2600
Hepatitis C	9	15	229	243	876	1543	368	820	60	71	1543	2692	4259
Hepatitis C – acute viral ^b	1	1	10	6	11	18	2	4	0	0	24	29	53
Hepatitis C – other ^b	8	14	219	237	865	1525	366	816	60	71	1519	2663	4206
Hepatitis D ^b	0	0	0	0	1	6	1	3	0	0	2	9	11
Hepatitis E ^b	0	0	1	4	0	3	0	0	0	0	1	7	8
HIV infection ^b	0	0	3	21	34	240	10	89	2	3	49	353	404
Influenza	174	244	180	200	199	197	203	180	167	170	923	991	1918
Influenza-Type A ^b	141	202	138	160	161	149	149	134	129	122	718	767	1487
Influenza-Type B ^b	10	18	17	14	13	27	21	19	18	22	79	100	180
Influenza-Type A & B ^b	0	0	5	6	3	3	6	7	4	9	18	25	43
Influenza-Type NOS ^b	23	24	20	20	22	18	27	20	16	17	108	99	208
Legionellosis	0	0	1	0	4	11	19	30	9	30	33	71	105
<i>Legionella longbeachae</i> ^b	0	0	1	0	1	3	8	5	2	9	12	17	29
<i>L. pneumophila</i> ^b	0	0	0	0	3	8	10	24	6	21	19	53	73
Legionnaire disease other	0	0	0	0	0	0	1	1	1	0	2	1	3
Leprosy	0	0	0	0	1	0	1	1	0	1	2	2	4
Leptospirosis ^b	0	0	1	1	0	3	1	1	0	1	2	6	8
Listeriosis ^b	1	0	0	0	2	1	0	2	8	8	11	11	22
Lymphogranuloma venereum (LGV) ^b													0
Malaria ^b	0	1	0	1	1	1	0	0	0	0	1	3	98
Measles	0	1	6	24	14	29	2	18	1	2	23	74	4
Measles laboratory confirmed	0	1	0	1	1	1	0	0	0	0	1	3	4
Measles – other													0
Meningococcal disease	18	25	21	14	4	10	8	4	4	4	55	57	112
Meningococcal – serogroup B ^b	15	18	12	10	2	9	4	3	3	0	36	40	76
Meningococcal – serogroup C ^b	0	1	4	0	1	1	2	1	0	0	7	3	10
Meningococcal – serogroup W135 ^b	0	1	0	0	0	0	1	0	0	0	1	1	2
Meningococcal – serogroup Y ^b	0	0	1	0	0	0	0	0	1	3	2	3	5
Meningococcal – other	3	5	4	4	1	0	1	0	0	1	9	10	19
Mumps ^b	2	3	26	64	83	116	13	14	1	0	125	197	323
Pertussis	100	74	217	165	346	210	400	259	176	134	1239	842	2093
Pneumococcal disease (invasive) ^b	40	43	16	24	38	54	55	72	82	98	231	291	522
Psittacosis ^b	0	0	1	0	2	1	6	15	1	7	10	23	34
Q fever ^b	1	0	12	21	25	49	23	64	9	11	70	145	215
Rubella	1	2	0	0	4	1	0	0	0	0	5	3	9
Congenital rubella ^b	0	0	0	0	0	0	0	0	0	0	0	0	1
Rubella – other ^b	1	2	0	0	4	1	0	0	0	0	5	3	8
<i>Salmonella</i> infection ^{b,d}	310	317	328	370	264	290	208	236	128	99	1238	1313	2564
Shigellosis ^b	1	2	9	4	11	21	9	9	3	2	33	38	71
Syphilis	1	4	14	42	131	455	52	266	49	97	247	864	1115
Congenital syphilis	1	2	0	0	0	0	0	0	0	0	1	2	4
Infectious syphilis ^{b,c}	0	0	3	21	17	274	5	106	1	7	26	408	434
Syphilis – other ^b	0	2	11	21	114	181	47	160	48	90	220	454	677
Tetanus	0	0	0	0	0	0	0	0	1	1	1	1	2
Tuberculosis ^b	4	4	37	45	76	95	54	60	33	41	204	245	452
Typhoid ^b	3	1	7	4	7	2	1	0	0	1	18	8	26
Verotoxin-producing <i>Escherichia coli</i> infections ^b	0	2	1	2	4	3	3	0	3	5	11	12	23

^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.

Due to data delay AIDS notifications will be reported in a later edition.

Institutional gastrointestinal outbreaks and foodborne illness are excluded from the table as complete demographic data is not routinely collected.

While transmission of some vaccine preventable diseases has been limited in NSW, the challenge still remains to increase vaccination rates among adolescents and young adults to reduce their susceptibility to diseases such as mumps, measles and pertussis.

The increase in *Salmonella* infections serves as a timely reminder to all to ensure thorough cooking and safe handling of high-risk foods such as raw chicken and other meats, and undercooked, cracked or soiled eggs, while the Legionnaire disease outbreak highlights the importance of cooling tower maintenance.

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.

References

1. OzFoodNet, 4th Quarterly and Annual Reports 2007. Accessed at <http://www.health.nsw.gov.au/publichealth/infectious>.
2. NSW Department of Health Communicable Diseases Report, NSW, for January and February 2007. *NSW Public Health Bull* 2007; 18(3–4): 66–8.
3. NSW Department of Health Communicable Diseases Report, NSW, for March and April 2007. *NSW Public Health Bull* 2007; 18(5–6): 100–3.

Erratum. The following correction should be made in Table 5 of the 2005 Annual Report (*NSW Public Health Bull* 2006; 17(5–6): 74): the headings 'Male' and 'Female' should be interchanged on each column.

Evaluation of a targeted immunisation program for Aboriginal and Torres Strait Islander infants in an urban setting

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Abstract: A conjugate pneumococcal vaccination program for Aboriginal and Torres Strait Islander children in an urban setting initially achieved poor uptake. A targeted intervention was developed to raise awareness among hospital staff, in general practice and in parents of eligible children. An evaluation of the intervention found moderate levels of increased awareness, use of promotional materials and an increase in vaccination. However, significant structural barriers remained.

Background

In February 2002, the 7-valent pneumococcal conjugate vaccine (7vPCV) (Prevenar; Wyeth, Sydney) was provided free in NSW for Aboriginal and Torres Strait Islander infants and others at high risk of invasive pneumococcal disease (IPD) at two, four and six months of age.¹ This was a response to higher rates of IPD in Aboriginal and Torres Strait Islander children compared with the total child population.² (For ease of reporting, henceforth Aboriginal and Torres Strait Islander will be referred to as Aboriginal.)

Based on Australian Bureau of Statistics population statistics, it was estimated that 1200 doses of the vaccination would be required for approximately 400 Aboriginal babies born each year within Western Sydney and Wentworth Area Health Services. However, twelve months after 7vPCV was introduced to the schedule, only 406 vaccine doses had been ordered.

Intervention to improve uptake of 7vPCV

A steering group was convened to identify ways to improve the uptake of 7vPCV among Aboriginal infants in

Western Sydney and Wentworth Area Health Services. The group consisted of representatives from the Western Sydney and Wentworth Public Health and Aboriginal Health Units, the Western Sydney Division of General Practice, the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, and the Daruk Aboriginal Medical Service (AMS).

Coverage rates for vaccines recommended for all children were higher than the state average for Aboriginal children (93% fully vaccinated in Sydney West v. 87% in NSW) and also for non-Aboriginal children (91% v. 90%, respectively).³ The steering group decided that the main reasons for low coverage of 7vPCV in Aboriginal children were likely to be a lack of knowledge about the targeted program among both parents and providers, and failure in identifying eligible infants. An intervention was designed to facilitate the identification of the Indigenous status of babies in maternity hospitals and general practice (GP), and to provide parents of Aboriginal infants with relevant educational materials. These aims were implemented through six actions:

- maximising the identification of Aboriginal infants by ward staff at the three local maternity hospitals
- training sessions for all maternity hospital staff, Aboriginal Liaison Officers (ALOs), community health centres and council vaccination staff in the two area health services
- posters and information sheets were mailed to all local immunisation providers
- personal contact between parents and ALOs
- provision of information to parents by ALOs
- placement of an 'eligible for free 7vPCV' sticker in the babies' Child Health Records (Blue Books) by maternity ward staff.

The sticker was intended to act as a prompt for providers when the child attended hospital for other vaccinations or reasons. An information session was also held at the local GP division meeting, but only a small proportion of GPs attended.

The intervention was fully operational by the last quarter of 2003 and continued until the 7vPCV vaccination was funded for all Australian infants in January 2005.

Evaluation methods

An evaluation was conducted 12 months after the commencement of the intervention to assess:

- its impact on identifying Aboriginal babies at maternity hospitals
- the success of sticker placement in Blue Books
- parents' and providers' knowledge about the vaccination program
- remaining barriers to the identification of Aboriginal babies
- the contribution of different immunisation service providers to 7vPCV vaccinations.

Interviews

Structured telephone or face-to-face interviews were carried out with all ward-based maternity staff members responsible for placing stickers, ALOs at the two largest hospitals in the area and AMS nursing staff. Structured telephone interviews were also sought from all GPs in the two area health service areas who had ordered 7vPCV during the intervention period.

Nursing staff at Daruk AMS took part in structured interviews. In addition, during a six-week data collection period during May and June 2004, the Child Health Records of all Aboriginal babies born after the commencement of the intervention were checked for the presence of information on 7vPCV and the sticker.

Australian Childhood Immunisation Register data analysis

Analysis was undertaken to determine: the estimated proportion of infants recorded as Aboriginal who had received a first dose of 7vPCV from November 2001 to October 2004; and the distribution of doses administered by provider type (AMS, community health, local council, GP) during the first six months of 2004. Public health units provided vaccine for the intervention program. To obtain distribution data, records of 7vPCV requests to public health units for use in Aboriginal infants born between 1 November 2003 and 30 April 2004 were merged with Australian Childhood Immunisation Register (ACIR) data.

Evaluation results

Interviews

During the intervention, hospital admission departments, previously advised by maternity ward staff and social workers, provided ALOs with the names of Aboriginal babies.

Interviews of staff at maternity hospitals revealed several barriers to identifying babies as Aboriginal; these barriers continued after staff training had taken place. Key barriers were:

- the reliance on software that recorded the Aboriginal status of mothers, but not of fathers

- identifying babies discharged after hours, on weekends or after only a short stay; babies of non-resident mothers (such as babies admitted to neonatal intensive care)
- the difficulty of maintaining staff awareness in this urban setting where few Aboriginal babies were seen
- the difficulty of maintaining staff awareness among new or relief staff.

Daruk AMS staff reported that their clients were very aware of the 7vPCV vaccine and the need for their babies to receive it.

The extent to which the program was responsible for improving knowledge was uncertain; however, the nursing staff felt it played a substantial role. Nurses also reported that there was now a greater community awareness of the vaccine and that the hospital-based information was supporting this knowledge.

Thirteen babies born after November 2003 were seen during the six weeks that data were collected. Six babies (46%) had the sticker in their Child Health Record, and one had the information postcard in the sleeve of the Child Health Record. Nurses estimated that approximately 60% of all babies who were eligible to receive the 7vPCV vaccination had the sticker in their Child Health Record.

General practice

During the first eight months of the program, 27 of approximately 700 GPs in Western Sydney and Wentworth Area Health Services ordered 7vPCV for an Aboriginal infant born after the intervention commenced. Twenty-three GPs were interviewed; these GPs had vaccinated 29 Aboriginal babies. At GP consultations, Child Health Records were brought for 24 of the babies (83%), seven Child Health Records had a sticker (29%) and, in four consultations (14%), the sticker had contributed to the identification and vaccination of the baby.

Table 1. Number of Aboriginal and Torres Strait Islander babies born between 1 November 2003 and 30 April 2004 for whom dose 1 of the 7vPCV schedule was ordered from Western Sydney and Wentworth Area Health Services between 1 January and 30 June 2004, by provider type

Provider type	Vaccinations	
	<i>n</i>	%
Aboriginal Medical Service	25	33
Community Health Centre	13	17
Council	3	4
General practice	34	45
Total	75	100

Source: Australian Childhood Immunisation Register.

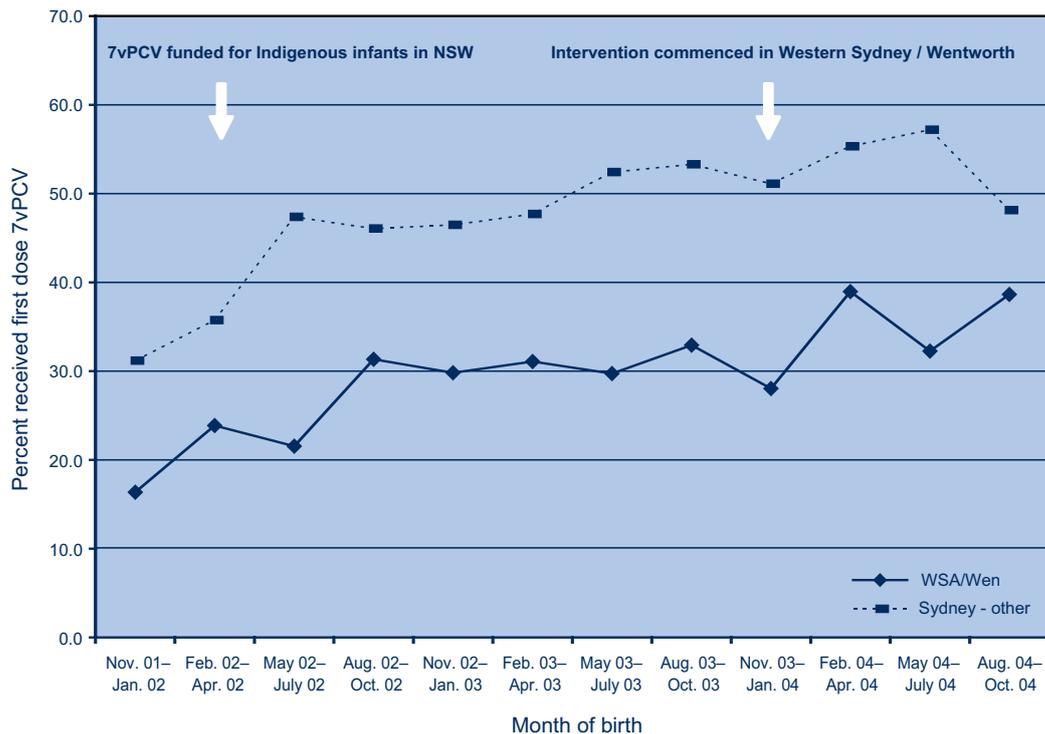


Figure 1. The proportion of Aboriginal and Torres Strait Islander infants that received the first dose of 7vPCV, Western Sydney and Wentworth Area Health Services compared with other areas of Sydney. WSA/Wen: Western Sydney and Wentworth Area Health Services. Source: Australian Childhood Immunisation Register.

Australian Childhood Immunisation Register data analysis

Table 1 presents the results of data on service provider types that reported 7vPCV vaccinations to the Australian Childhood Immunisation Register (ACIR) during the evaluation period. The largest proportion of babies was vaccinated by GPs (45%), followed by the AMS (33%).

Figure 1 presents data on the proportion of Aboriginal infants who received the first dose of 7vPCV during the intervention. The Western Sydney and Wentworth Area Health Services had consistently lower coverage compared with other parts of Sydney. This increased in Western Sydney from approximately 30% before the intervention to approximately 40% afterwards, but was still below the 50% vaccination coverage of Aboriginal infants in the rest of Sydney.

Discussion

This evaluation has found a moderate impact from an intervention designed to improve the uptake of 7vPCV vaccination of Aboriginal infants in an urban setting. Results suggested a high level of awareness among AMS clients, but only 4% of GPs in the area were known to have vaccinated an Aboriginal infant in the first eight months of the intervention. On follow up, the increased identification of Aboriginal babies and the placement of stickers in Child Health Records were successful for approximately half of Aboriginal infants who attended the AMS.

The methods used in both the intervention and the evaluation were limited by the resources available but were regarded as appropriate.⁴ Community consultation was limited to the inclusion of the local AMS; only health care workers were interviewed and the evaluation did not include a pre-intervention phase or, with the exception of ACIR coverage data, a comparison group in a non-intervention area. Nevertheless, the results are consistent with previous findings of low uptake of vaccination programs targeted at Aboriginal people, lower vaccination coverage in Aboriginal people in urban areas, low rates of identification of Aboriginal people in NSW hospitals and the considerable difficulties associated with overcoming these issues.^{5–7}

Identification of Indigenous status is fundamental to understanding and addressing equity of access to health care services for Aboriginal people. It is a key objective of both the NSW Aboriginal Health Strategic Plan and the guidelines to improve identification of Indigenous status in the public hospital system.^{8,9} However, to improve Aboriginal identification and maximise the effectiveness of targeted programs in influencing change, there are system-level barriers that need to be addressed. The implementation of any program targeting Aboriginal people will need to acknowledge and consider the barriers identified in this evaluation.

While the AMS is the largest single provider of immunisation services for Aboriginal babies, this evaluation has

shown that for these infants in an urban setting, most will be immunised by a non-Aboriginal provider. Any program in a similar urban setting that targets either Aboriginal immunisation service provision or the needs of immunisation providers should consider the significant role of non-Aboriginal providers. However, most GPs do not immunise Aboriginal babies, and those that do will only immunise small numbers in individual practices. These results have implications for the delivery and use of scarce resources for these immunisation programs in general practice. Future directed programs would need to consider whether (and to whom, and how) to direct resources preferentially. To address this problem it would be helpful to explore the role of divisions of general practice; possible registers of GPs with an interest in Aboriginal health; and the development of formal links between GPs, divisions of general practice and the AMS. More community-based research involving Aboriginal people may uncover other useful strategies.

Conclusion

An intervention developed at the local level has been partially successful in improving the impact of vaccination targeted at Aboriginal children in an urban setting. However, significant structural barriers need to be addressed before equity of access is achieved. These include complete recording of Indigenous status in hospitals and increased awareness in general practice.

References

1. National Health and Medical Research Council. Australian Immunisation Handbook, 8th edn. Canberra: Australian Government Department of Health and Ageing, 2003.
2. Roche P, Krause V, Andrews R, Carter L, Coleman D, Cook H, et al. Invasive pneumococcal disease in Australia, 2002. *Commun Dis Intell* 2003; 27: 466–77.
3. NSW Department of Health Quarterly report: Australian Childhood Immunisation Register. *NSW Public Health Bull* 2005; 16(11–12): 205–211.
4. Australian Bureau of Statistics, Australian Institute of Health and Welfare. The Health and Welfare of Australia's Aboriginal and Torres Strait Islander peoples, 2005. Report No. 4704.0. Canberra: 2005.
5. Hull BP, McIntyre PB. What do we know about 7vPCV coverage in Aboriginal and Torres Strait Islander children? *Commun Dis Intell* 2004; 28: 238–43.
6. Menzies R, McIntyre P, Beard F. Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia, 1999 to 2002. *Commun Dis Intell* 2004; 28: . S1
7. Australian Institute of Health and Welfare. Improving the quality of Indigenous identification in hospital separations data. AIHW Cat no. HSE 101. Canberra: AIHW: 2005.
8. NSW Health Department 1999. Aboriginal Health Strategic Plan. State Health Publication No (AH) 990151.
9. NSW Health Department 2000. Better Practice Guidelines to improve the Level of Aboriginal and Torres Strait Islander Identification in the NSW Public Health System. State Health Publication No (CSP) 980163.

Aboriginal and Torres Strait Islander peoples at higher risk of invasive meningococcal disease in NSW

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Abstract: *Objective:* To assess the completeness of data describing Aboriginal and Torres Strait Islander status in NSW invasive meningococcal disease notifications and determine the relative risk for invasive meningococcal disease among Aboriginal and Torres Strait Islander peoples in NSW.

Methods: Surveillance data from the NSW Notifiable Diseases Database was reviewed for 5-year periods between 1991 and 2005.

Results: Invalid and missing data on Aboriginal and Torres Strait Islander status decreased from 42% to 8% during the study period. Higher rates of disease were found in young children and significantly higher rates in Aboriginal and Torres Strait Islander children aged 0–4 years compared with their non-Aboriginal counterparts.

Conclusion: Aboriginal and Torres Strait Islander children in NSW experience higher rates of notified invasive meningococcal disease than non-Aboriginal children.

Background

Invasive Meningococcal Disease (IMD) is a serious but uncommon bacterial infection. The disease usually presents as meningitis or septicaemia, or a combination of the two presentations, with a case fatality rate of approximately 10% despite appropriate antibiotic therapy.¹ Pneumonia, arthritis and conjunctivitis may also occur. Higher rates of disease occur in children aged less than one year, children aged 1–4 years and adolescents 15–19 years of age.¹ Reported risk factors for IMD include household crowding, chronic underlying illness, active and passive smoking, some immunosuppressive illnesses and anatomical or functional asplenia.²

Disease rates are higher among some population groups, such as African-Americans.³ These higher disease rates have been attributed to other risk factors such as poverty and overcrowding, while higher mortality rates have been linked to limited access to health care services.^{3,4} Living conditions, such as overcrowding, can result in a higher exposure to potential carriers of *Neisseria meningitidis*.⁴

There are little published data describing the risk of IMD among Aboriginal and Torres Strait Islander peoples. A north Queensland study found a 3-fold greater risk for Aboriginal and Torres Strait Islander peoples for the period 1995 to 1999.⁵ The incidence of IMD in Aboriginal and Torres Strait Islander peoples in Western Australia was six times greater than that of the non-Aboriginal population for the period 1990–1995.⁶ The Australian Institute of Health and Welfare reported notification rates between 7.4 and 11.3 per 100 000 in the years 2000, 2001, 2003 and 2004 in Aboriginal and Torres Strait Islander peoples but no comparisons with non-Aboriginal Australians were provided.^{7,8} To date, the Australian Institute of Health and Welfare summary of health performance indicators has not included IMD notifications from NSW as the data has not demonstrated adequate completeness for Aboriginal and Torres Strait Islander status. In 2001, the NSW Public Health Network commenced a data quality improvement project for recording Aboriginal and/or Torres Strait Islander status for selected diseases, including IMD.

The aims of the study were to assess the completeness of data describing Aboriginal and/or Torres Strait Islander status in NSW invasive meningococcal disease data contained within the NSW Notifiable Diseases Database; and to describe the relative risk for Aboriginal and Torres Strait Islander peoples being notified with IMD in NSW compared with the non-Aboriginal population.

Methods

Data on meningococcal disease is collected in NSW under the requirements of the *Public Health Act (1991)*, with all cases of meningococcal disease meeting the case definitions of the National Notifiable Diseases Surveillance System being notifiable by pathology laboratories, hospitals and doctors to public health units.⁹ Case information is entered into the NSW Notifiable Diseases Database.

Table 1. Trends in notification of invasive meningococcal disease in Aboriginal and Torres Strait Islander people and non-Aboriginal people, and the completeness of the recording of Aboriginal and Torres Strait Islander status, NSW 1991–2005

Years	N	Non-Aboriginal	Aboriginal and Torres Strait Islander	Aboriginal and/or Torres Strait Islander status not recorded or invalid data	
		n	n	n	%
1991–1995	657	346	34	277	42
1996–2000	1036	720	50	266	26
2001–2005	935	806	55	74	8
Total	2628	1872	139	617	76

Source: NSW Notifiable Diseases Database.

NSW meningococcal disease notification data since the promulgation of the *Public Health Act* in 1991 were sourced from HOIST (Health Outcomes Information and Statistical Toolkit, NSW Health). Analysis was performed using Microsoft Excel 2003. Five-year study periods were defined (1991–1995, 1996–2000 and 2001–2005) with mid-term estimate population figures from the Australian Bureau of Statistics 1991, 1996 and 2001 censuses used as denominators.

The recording of Aboriginal and/or Torres Strait Islander status was assessed as complete if a valid response was recorded in the Aboriginal and/or Torres Strait Islander field in the Notifiable Diseases Database. A valid response was defined as 'yes' or 'no'.

Five-year mean notification rates were calculated for comparison purposes. The risk of being notified with meningococcal disease in the Aboriginal and Torres Strait Islander population was calculated and then compared with the risk for the non-Aboriginal population (relative risk). Age standardisation was performed using the direct method to control for the higher proportion of younger people in the Aboriginal and Torres Strait Islander population. The non-Aboriginal population in NSW was used as the standard. For ease of reference in reporting, 'Aboriginal' will be used to refer to both groups combined.

Controlling for socioeconomic status was not feasible with

the notification data available. There is no routine collection of a notified individual's socioeconomic status, and the small numbers of notifications would not support an ecological analysis.

Results

During the period under study, there were 2628 notifications of invasive meningococcal disease in NSW residents. Of these notifications 139 were recorded as Aboriginal people (Table 1). In the period 1991–1995, 277/657 (42%) of notifications of IMD in NSW did not record Aboriginal status, or the data was invalid. In the most recent period, 2001–2005, 74/935 (8%) of notifications in NSW did not include valid data on Aboriginal status (Table 2).

IMD notification rates in non-Aboriginal people over the three study periods ranged from 2.11–3.17 per 100 000 population, while for Aboriginal people the rates ranged from 6.02–7.90 per 100 000 population. There was a statistically significant two- to three-fold increased risk of IMD across the three study periods for Aboriginal people in NSW (Table 2).

The highest notification rates for IMD in NSW during the period under review were seen in young children. In the period 2001–2005, non-Aboriginal children aged 0–4 years experienced an IMD rate of 12.37 per 100 000 population, while the rate was 40.99 per 100 000 population among Aboriginal children in this age group. After direct

Table 2. Notification rates and relative risk of invasive meningococcal disease for Aboriginal and Torres Strait Islander peoples compared with non-Aboriginal people in New South Wales, 1991–2005

Years	Notification rates/100 000 population		Relative risk	95% confidence intervals
	Non-Aboriginal	Aboriginal and Torres Strait Islander		
1991–1995	2.11	6.02	2.85	2.02 to 4.02
1996–2000	3.17	7.88	2.48	1.87 to 3.30
2001–2005	2.69	7.90	2.94	2.24 to 3.86

Source: NSW Notifiable Diseases Database.

Table 3. Age standardised invasive meningococcal disease notification rates for non-Aboriginal people and Aboriginal and Torres Strait Islander peoples in NSW, and the relative risk of notification in Aboriginal and Torres Strait Islander peoples, NSW, 2001–2005

Age group years	Notification rate/100 000 population		Relative risk	95% confidence intervals
	Non-Aboriginal	Aboriginal and Torres Strait Islander		
0–4	12.37	40.99	3.31	2.35 to 4.68
5–19	4.07	3.54	0.87	0.45 to 1.69
20+	1.49	2.56	1.72	0.89 to 3.33
Total	2.69	7.90	2.94	2.24 to 3.86

Source: NSW Notifiable Diseases Database.

age-standardisation for the period 2001–2005, the relative risk remained significantly higher for Aboriginal children aged 0–4 years of age (Table 3).

Discussion

The recording of Aboriginal status in NSW has improved since 1990, with invalid data decreasing from 42% to 8%. This improvement in recording of status justifies the comparison of risk among Aboriginal and non-Aboriginal people in NSW.

The risk of IMD is not homogenous across the population of NSW. Our analysis confirms that young children are at increased risk, but importantly indicates that Aboriginal status is also associated with higher rates of disease. Other countries also have demonstrated heterogenous risk among different portions of their population. In the United Kingdom, IMD incidence and mortality are socially patterned, with IMD incidence in the most deprived quintile being twice that of the most affluent quintile.¹⁰ In New Zealand, significantly higher rates of IMD have been reported in Maori (relative risk = 2.2) and Pacific Islander people (relative risk = 3.8) when compared with the European population.¹¹ Aboriginal people are the most disadvantaged group in Australia.¹² Two important risk factors associated with increased risk of IMD are more common among Aboriginal people, namely having a smoker among close contacts, including maternal smoking, and sharing a bedroom.^{13–15} It is not possible to explore the causal interaction of these factors from notifiable disease data. Further research into these factors could lead to the development of more informed prevention strategies.

The early recognition and diagnosis of meningococcal infection can lead to reduced risk of complications.¹⁶ In addition to clinicians being aware of a higher risk of IMD in young children, this analysis indicates an even higher risk in young Aboriginal children.

Conclusions

The completeness of the data on Aboriginal and/or Torres Strait Islander status in notifications of invasive meningococcal

disease in NSW has improved sufficiently to warrant inclusion in the Australian Institute of Health and Welfare's Performance Indicators report. This will further the understanding of meningococcal disease across Australia.

In NSW, Aboriginal children 0–4 years of age have a significantly higher risk of invasive meningococcal disease when compared with non-Aboriginal children.

References

- Hogan D, McAnulty J. *EpiReview: Meningococcal disease in New South Wales, 1991–2002* *N S W Public Health Bull* 2004; 15: 39–43. doi:10.1071/NB04011
- Baltimore RS. Recent trends in meningococcal epidemiology and current vaccine recommendations. *Curr Opin Pediatr* 2006; 18: 58–63. doi:10.1097/01.mop.0000193265.78506.7f
- Sharip A, Sorvillo F, Redelings MD, Mascola L, Wise M, Nguyen DM. Population-Based Analysis of Meningococcal Disease Mortality in the United States 1990–2002. *Pediatr Infect Dis J* 2006; 25: 191–4. doi:10.1097/01.inf.0000202065.03366.0c
- Gardner P. Prevention of Meningococcal Disease. *N Engl J Med* 2006; 355: 1466–73. doi:10.1056/NEJMcp063561
- Harley D, Hanna JN, Hills SL, Bates JR, Smith HV. Epidemiology of invasive meningococcal disease in north Queensland, 1995 to 1999. *Commun Dis Intell* 2002; 26: 44–50.
- Olesch CA, Knight GJ. Invasive meningococcal infection in Western Australia. *J Paediatr Child Health* 1999; 35: 42–8. doi:10.1046/j.1440-1754.1999.t01-1-00337.x
- Standing Committee on Aboriginal and Torres Strait Islander Health and Statistical Information Management Committee. National summary of the 2001 and 2002 jurisdictional reports against the Aboriginal and Torres Strait Islander health performance indicators. Canberra: Australian Institute of Health and Welfare; 2004. AIHW cat. No. IHW12.
- Standing Committee on Aboriginal and Torres Strait Islander Health and Statistical Information Management Committee. National summary of the 2003 and 2004 jurisdictional reports against the Aboriginal and Torres Strait Islander health performance indicators. Canberra: Australian Institute of Health and Welfare, 2006. AIHW cat. No. IHW16. Available from: <http://www.aihw.gov.au/publications/index.cfm/title/10234>

9. Commonwealth of Australia. Guidelines for the early clinical and public health management of meningococcal disease in Australia. Canberra: Commonwealth Department of Health and Aged Care; 2007. Available from: <http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-pubs-other-mening-2007.htm>
10. Heyderman RS, Ben-Shlomo Y, Brennan CA, Somerset M. The incidence and mortality for meningococcal disease associated with are deprivation: an ecological study of hospital episode statistics. *Arch Dis Child* 2004; 89: 1064–8. doi:10.1136/adc.2003.036004
11. Martin D, McDowell R. The epidemiology of meningococcal diseases in New Zealand in 2003. Wellington: Ministry of Health, New Zealand; 2004.
12. NSW Department of Aboriginal Affairs. Introducing Indigenous Australia. Background briefing. Sydney: NSW Department of Aboriginal Affairs; 2004. Available at www.daa.nsw.gov.au
13. Population Health Division. The health of the people of New South Wales: Report of the Chief Health Officer. Sydney: NSW Department of Health; 2008. Available at: <http://www.health.nsw.gov.au/public-health/chorep/toc/choindex.htm>. Accessed 28 April 2008.
14. Sorensen HT, Labouriau R, Jensen ES, Mortensen PB, Schonheyder HC. Fetal growth, maternal prenatal smoking, and risk of invasive meningococcal disease: a nationwide case-control study. *Int J Epidemiol* 2004; 33: 816–20. doi:10.1093/ije/dyh169
15. Robinson P, Taylor K, Nolan T. Risk factors for meningococcal disease in Victoria, Australia in 1997. *Epidemiol Infect* 2001; 127: 261–8.
16. Ninis N, Phillips C, Bailey L, Pollock JI, Nadel S, Britto J et al. The role of healthcare delivery in the outcome of meningococcal disease in children: case-control study of fatal and non-fatal cases. *BMJ* 2005; 330: 1475–80. doi:10.1136/bmj.330.7506.1475

Australian Bat Lyssavirus: examination of post-exposure treatment in NSW

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Abstract: Ten years after the recognition of Australian Bat Lyssavirus, it is timely to review the occurrence of the virus in native microbat and flying fox species in Australia, and the effectiveness of post-exposure treatment in humans. Differences between post-exposure treatment protocols adopted by state and territory health departments were examined. In Queensland and the United States of America, post-exposure treatment is withheld in people who are bitten by bats that subsequently test negative for ABLV and rabies, respectively. The good outcomes from these protocols support the revised NSW policy, which delays post-exposure treatment for up to 48 hours for minor exposures while awaiting bat test results. Post-exposure treatment can be withheld or ceased if the bat test result is negative.

Two deaths have been caused by Australian Bat Lyssavirus (ABLV) infection in Australia. The clinical disease was a progressive encephalitis indistinguishable from rabies. ABLV is phylogenetically closely related to rabies virus, lyssavirus 1. Since ABLV was recognised, all state and territory health departments in Australia have adopted protocols to administer post-exposure treatment (PET) to people bitten or scratched by bats, but there are jurisdictional differences. As it is a decade since the first human case was diagnosed, it is timely to review the NSW protocol against current practice in other jurisdictions, contemporary epidemiological understanding of ABLV, and the evidence for effectiveness of post-exposure treatment.

ABLV in Australia

Australian Bat Lyssavirus, genotype 7 of the lyssavirus genus, is a member of the family Rhabdoviridae that shares many serotypic, antigenic and molecular sequence

features with classical rabies virus.¹ It was first reported in July 1996 in a black flying fox (*Pteropus alecto*) from Ballina, NSW, and has subsequently been confirmed in five species of Australian bat, four species of flying fox (suborder Megachiroptera, genus *Pteropus*) and one species of insectivorous bat (suborder Microchiroptera, *Saccolaimus flaviventris*).² Two Queensland women are known to have succumbed to disease caused by ABLV: one woman from Rockhampton died in November 1996, within 5 weeks of a scratch from a microbat, probably a yellow-bellied sheath-tailed bat (*S. flaviventris*); and the second woman, from Mackay, died in December 1998, more than two years after a bite from a flying fox. Their ante-mortem clinical presentation was indistinguishable from classic rabies infection.^{3,4}

Australia has five flying foxes in the genus *Pteropus*. They eat fruit and live in large colonies, often with multiple species roosting together. Australia also has a diversity of small insectivorous microbats, with approximately 80 species recorded. In NSW, the grey headed flying fox (*P. poliocephalus*) inhabits predominantly coastal and mountainous regions; the black flying fox (*P. alecto*) is found mainly in the northern coastal area; and the little red flying fox (*P. scapulatus*) has a range that extends further west, encompassing most of the state.⁵ The microbat, *S. flaviventris*, is distributed throughout Australia, except South Australia and the southern parts of Western Australia.

Prevalence in Queensland and NSW

To allow for an assessment of the risk, it is necessary to establish the prevalence of ABLV among flying foxes and microbats. Prevalence data from bats submitted through an arrangement of Queensland public health units with Queensland Scientific Services during July 1998 to February 2006, and from bats submitted through an arrangement of NSW public health units with Australian Animal Health Laboratories between 1995 and 2005 are presented in Table 1.

It is important to note that the four microbats from NSW that tested positive for ABLV were all detected in the first quarter of 1999, but were not identified to the species level. These are similar findings to a large screening program conducted in partnership with Department of Primary Industries in Queensland from June 1996 to March 2002.⁶ Among submitted ill or injured animals, 69/974 (7.1%) of flying foxes and 5/158 (3.2%) of microbats were ABLV positive using direct fluorescent antibody testing (DFAT).

All five positive microbats were *S. flaviventris*, and these were five of the seven *S. flaviventris* specimens tested. In another large survey of caught wild bats, none of 475 wild-caught flying foxes were DFAT positive.⁷ Eight of 266 (3%) wild-caught flying foxes were antibody positive. Of 318 wild-caught microbats, none were DFAT positive while 9 (2.8%) were antibody positive.

The only microbat found to be DFAT positive, the yellow bellied sheath tail (*S. flaviventris*), is secretive and rarely seen by humans, and appears to have a high prevalence of infection. It has its own sub-strain of ABLV virus, which was responsible for a death in a bat handler. Four other species of small bats have been found to have antibodies to ABLV. There is uncertainty about whether these species can transmit infection. The antibodies may represent seroconversion without infectivity or response to an insect rhabdovirus.

Exposures in Queensland

In a series of bat exposures reported in Brisbane, those exposed to bat bites were largely members of the public who approached a bat (55%) and professional or volunteer bat handlers (20%).⁸ In 15% of cases, the bat initiated contact. Over the last 10 years, the Brisbane Southside Public Health Unit has administered over 300 courses of PET. Comparable figures for NSW are not collated.

Laboratory testing of bats

Queensland Health uses the Queensland Scientific Services laboratory in Brisbane for testing the brains of submitted bats for ABLV. All other states use the Australian Animal Health Laboratory in Geelong. Both laboratories use a DFAT on brain impression smears and also perform confirmatory polymerase chain reaction tests on all specimens. Complete concordance has been documented between these tests.⁹ International experts state that either test alone is considered adequate for confirmation of rabies infection.^{10,11} The Australian Animal Health Laboratory also performs a DFAT on salivary gland tissue. Extensive experience with lyssavirus 1 has indicated that PET is unnecessary if the test results in the bat are negative (C Rupprecht, pers comm).

Effectiveness of post-exposure treatment

Of the 53 positive bats submitted by public health units (Table 1), it is not known how many bit more than one person. However, it would be safe to estimate that the number of people exposed to known positive bats is between 50 and 100. Experience with rabies in countries where vaccine is not available shows that not everyone bitten by a rabid dog develops disease, so infection is unlikely to be universal in humans following a bite by a positive bat.¹²

Cross protection offered by rabies vaccine against ABLV is supported by two published papers. Tests conducted at the Centers for Disease Control and Prevention, Atlanta, Georgia, in mice vaccinated with four different rabies vaccines, found that all five mice challenged with ABLV survived. These tests did not include the vaccine currently used in Australia; however, they provided the basis for the current PET recommendation in Australia.¹ A second study published in 2005, examined cross protection against both ABLV and European Bat Lyssavirus. It demonstrated protection in only 15 of 19 mice challenged with ABLV.¹³ Cross neutralisation with antibodies from 52 volunteers immunised with the human diploid cell vaccine (HDCV, as used in Australia) against various lyssaviruses, including ABLV, showed similar levels of neutralisation against all viruses, especially in low and mid level responders. As the vaccine was not fully protective in mice, and there are considerable interspecies differences in the virulence of the various lyssavirus genotypes, this is not convincing evidence of the vaccine's efficacy against ABLV in humans. Nevertheless, there has not been a failure of properly administered PET for rabies in the United States of America since 1979.¹⁰

It is reassuring that there is no evidence of development of encephalitis documented among individuals administered PET. However, this does not guarantee 100% efficacy in preventing ABLV given the occasional prolonged incubation period of lyssavirus infection and the current incomplete documentation of administration and follow up of individuals initiated on PET.

In locations where treatment was withheld due to bats

Table 1. Australian Bat Lyssavirus prevalence in Queensland bats submitted to Queensland Scientific Services, July 1998 to February 2006, and NSW bats submitted to Australian Animal Health Laboratories, January 1995 to December 2005

	Queensland			NSW			Total		
	Tested <i>n</i>	Positive <i>n</i>	%	Tested <i>n</i>	Positive <i>n</i>	%	Tested <i>n</i>	Positive <i>n</i>	%
Flying foxes	485	30	6.2	249	18	7.2	734	48	6.5
Microbats	115	1	0.9	68	4	5.9	183	5	2.7
Total	600	31	5.2	317	22	6.9	917	53	5.8

Source: Queensland Scientific Services and Australian Animal Health Laboratories.

testing negative, there have so far been no negative outcomes. Brisbane Southside Public Health Unit reported that of 246 exposures managed from 1996 to 2003, PET was either withheld or ceased for 65 individuals after a negative bat result.⁸ Updating this data in 2005 provided an additional 59 people exposed to bat bites or scratches, of whom 26 had the bat tested (all were negative) and 24 were not administered PET (Jarvinin, pers comm.).

Australian post-exposure treatment protocols

In Australia, a variety of protocols are available to guide PET following a bat exposure. These include the National Guidelines, the 8th edition of the *Immunisation Handbook* and various state and territory notifiable disease guidelines.^{14,15} Only South Australia and Tasmania do not have their own guidelines and defer exclusively to the National Guidelines for PET. Protocols share many features, for example: similar approaches to all bat exposures whether flying foxes or microbats; emphasis on the importance of immediate rigorous wound cleaning; using the same doses and schedules for immunoglobulin (HRIG) and rabies HDCV; and omitting HRIG where more than 7 days has elapsed since the first vaccine dose.

All protocols emphasise the necessity of testing the bat for ABLV. However, protocols differ notably in two aspects: the influence of bat brain testing for ABLV on PET and the influence of exposure type on PET. The Queensland, Australian Capital Territory and Northern Territory protocols explicitly state that PET should be withheld or ceased if the implicated bat's brain tests negative for ABLV. The Victorian protocol and NHMRC *Immunisation Handbook* also indirectly advocate this approach. The National Guideline defers this decision to local public health units. However, the former NSW protocol explicitly and uniquely stated that PET is not affected by the results of tests on the bat.

Protocols in NSW, Australian Capital Territory and Victoria distinguish between severe and mild exposures. Severe bites are: bites on the face or neck; bites by a sick or abnormally behaving bat; or unprovoked or multiple bites. These states advocate immediate initiation of PET for a severe bite, but a delay of up to 48 h for other exposures. Another inconsistency is HRIG administration. Queensland and Victoria recommend excluding HRIG if the bite occurred more than 12 months previously, while the NHMRC *Immunisation Handbook* suggests administration in this circumstance. Other protocols are silent on this issue.

Discussion

ABLV has never been found in a bat caught during wildlife surveys, but has been present in the brains of approximately 6% of bats with human contact. Although wildlife surveys suggest that the risk from small bats is

less than from large bats, there is sufficient uncertainty that the recommendation to regard all bats as infective should remain.

Laboratory tests on the brain of the bat, when available, can reliably detect the presence of Lyssavirus as supported by the concordance of different tests. Evidence from Queensland and the USA suggests that where a bat brain tests negative, it is not necessary to administer PET. There was no disease in the 89 people not given PET following the Queensland protocol, and similar positive experience with the same testing and management protocol following animal bites for thousands of people each year in the USA.¹⁰

Evidence to support rabies protocols distinguishing severe and mild bites comes from reports of PET failures after dog bites in Thailand, in which a common feature was bites to the head or neck.^{16,17} PET failure is more likely after multiple dog bites, and the incubation time is shorter for bites closer to the brain, supporting the suggestion for PET to be initiated more promptly after such bites. There is a relative paucity of evidence from animal studies on the effectiveness of rabies vaccine against ABLV as the research was conducted only in small numbers of mice; however, further work in this area is currently underway.

Unfortunately there is currently no aggregation of data on the number or completion of PET courses in NSW, although vaccine supply data suggests up to 130 per year.

The current NSW guideline requests testing of all bats. Although this could lead to under-reporting of bites by carers who do not want the animal destroyed, the use of bat testing to guide PET decisions will result in some savings through reduced PET administration.

Conclusion

The lack of adverse outcomes in Queensland and the USA supports withholding PET following bat exposures in non-severe bites if the bat tests negative. There is no direct evidence of the acceptable length of delay while waiting for a laboratory result, however 48 hours seems reasonable. Bats should be submitted without delay for ABLV testing directly to AAHL or QSS. For any bite PET may be suspended on the basis of a negative laboratory bat test.

It would be sensible to collate bat exposure and PET experience across NSW and Australia, including matching the bat results to the public health unit patient record. Public education on avoiding bat contact, and what to do if it occurs, remains the mainstay of preventing human ABLV infection. For a map of the distribution of big bats in Australia, see <http://www.newscientist.com/article/mg16021635.200-bats-out-of-hell.htm>.¹⁸

Acknowledgements

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References

- Hooper P, Lunt R, Gould A, Samaratinga H, Hyatt A, Gleeson L et al. A new Lyssavirus: the first endemic rabies related virus recognised in Australia. *Bull Inst Pasteur* 1997; 95: 209–18. doi:10.1016/S0020-2452(97)83529-5
- Gould AR, Hyatt A, Lunt R, Kattenhelt J, Hengstberger S. Characterisation of a novel lyssavirus isolated from Pteropid bats in Australia. *Virus Res* 1998; 54: 165–87. doi:10.1016/S0168-1702(98)00025-2
- Fraser G, Hooper P, Lunt R, Gould A, Gleeson L, Hyatt A. Encephalitis caused by a lyssavirus in fruit bats in Australia. *Emerg Infect Dis* 1996; 2: 327–31.
- Hanna J, Carney I, Smith G, Tannenberg A, Deverill J, Botha J et al. Australian bat lyssavirus infection: a second human case, with long incubation period. *Med J Aust* 2000; 172: 597–9.
- Hall L, Richards G. Flying foxes fruit and blossom bats of Australia. Sydney: UNSW Press, 2000.
- Barrett J. Australian Bat Lyssaviruses. Thesis, University of Queensland, 2005.
- Field H. The ecology of Hendra virus and Australian Bat Lyssavirus. Thesis, University of Queensland, 2005.
- Young M, McCall B. Trends in potential exposure to Australian bat Lyssavirus in south east Queensland, 1996 to 2003. *Commun Dis Intell* 2004; 28: 258–60.
- Warrilow D, Harrower B, Smith I, Field H, Taylor R, Walker C et al. Public health surveillance for ABLV in Queensland, Australia 2000, 2001. *Emerg Infect Dis* 2003; 9: 262–4.
- Rupprecht C, Gibbons R. Prophylaxis against rabies. *N Engl J Med* 2004; 351: 2626–35. doi:10.1056/NEJMcp042140
- Jackson A, Warrell M, Rupprecht C. Management of rabies in humans. *Clin Infect Dis* 2003; 36: 60–3. doi:10.1086/344905
- Cleaveland S, Fevre EM, Kaare M, Coleman PG. Estimating human rabies mortality in the United Republic of Tanzania from dog bite injuries. *Bull World Health Organ* 2002; 80: 304–10.
- Brookes S, Parsons G, Johnson N, McElhinney L, Fooks A. Rabies human diploid cell vaccine elicits cross-neutralising and cross-protecting immune response against European and Australian bat lyssaviruses. *Vaccine* 2005; 23: 4101–9. doi:10.1016/j.vaccine.2005.03.037
- Commonwealth Department of Health and Ageing. Australian Bat Lyssavirus Information for medical practitioners. Canberra: Commonwealth Department of Health and Ageing, 2001.
- National Health and Medical Research Council. Australian Immunisation Handbook, 8th edn. Canberra: National Health and Medical Research Council, 2003.
- Wilde H, Choomkasien P. Failure of rabies postexposure treatment in Thailand. *Vaccine* 1989; 7: 49–52. doi:10.1016/0264-410X(89)90010-8
- Wilde H, Sirikawin S. Failure of postexposure treatment of rabies in children. *Clin Infect Dis* 1996; 22: 228–32.
- Anderson I. Bats out of hell. *New Scientist* 1998; 2163: 40. Available from <http://www.newscientist.com/article/mg16021635.200-bats-out-of-hell.htm>

Recreational water: surfing the bugs

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There are many risks associated with the use of recreational water, including exposure to infectious diseases. Infectious diseases are transmitted either through contact with skin or mucous membranes, inhalation or ingestion. The infectious organisms include bacteria, viruses and parasitic protozoa, which occur naturally in recreational water or water that has been contaminated. Sources for contamination include: sewage effluent, livestock, domestic animals, wildlife, population using the water (bather shedding), industrial processes and farming activities.

Recreational water and infectious diseases

A wide variety of infectious diseases can be transmitted through the use of recreational water including gastrointestinal disease, respiratory disease, ear infections, skin disease, liver or renal disease, central nervous system infections and keratitis. An association between contaminated water and gastrointestinal disease as well as respiratory disease has been shown in randomised controlled trials conducted in the United Kingdom.^{1,2}

A prospective cohort study conducted in Sydney from 1989 to 1990 found that swimmers at Sydney ocean beaches were more likely to report respiratory, ear and eye symptoms than beach-goers who did not swim.³ Since then, the water quality in Sydney has improved due to the commissioning of the deep-water ocean outfalls in the early 1990s. A multi-centre study is now being planned to assess the current water quality and help verify whether current guidelines are applicable to the Australian environment.

Beachwatch programs

'Beachwatch' began in Sydney in 1989 and was expanded to harbour swimming sites in 1994, the Hunter and Illawarra regions in 1996 and regional councils along the NSW coast in 2004 under the 'Beachwatch Partnership Program'.

Water quality samples are collected from swimming locations every six days and analysed for both thermotolerant coliforms and enterococci. These bacteria indicate the presence of sewage and results are usually available within 24–48 hours of sample collection.

The Beachwatch program guidelines are currently based on the *Australian guidelines for recreational use of water*.⁴ A swimming site passes the guidelines if, for five samples collected in one month, the median does not exceed 150 cfu/100 mL thermotolerant coliforms and 35 cfu/100 mL enterococci, and the second highest value is below 600 cfu/100 mL thermotolerant coliforms and 100 cfu/100 mL enterococci.

Providing the community with regular and reliable information on beach water quality is a priority. Beachwatch issues a daily advisory warning, weekly star ratings, monthly media releases and annual State of the Beaches reports, and provides data on the *SoEdirect* website (<http://www.soedirect.nsw.gov.au>).

Information collected by Beachwatch is also used by Sydney Water to prioritise short- and long-term sewerage system maintenance works.

Blue-green algae and its potential health effects

Blue-green algae (BGA) are photosynthetic bacteria that are a natural part of the aquatic environment. They occur in low numbers in even the most pristine quality waters. When there is an excess of nutrients in the water, BGA can form a prolific dense growth or bloom. These blooms mainly occur in freshwater and can pose a public health risk.

Some BGA can produce highly potent toxins. However, not all BGA produce toxins and the same species can be toxic as well as non-toxic depending on the environment, physiology and genetics. The main toxins produced by BGA are hepatotoxins, which damage the liver and other internal organs, or neurotoxins, which can cause paralysis and respiratory arrest. Possible long-term effects include hepatocellular carcinoma.

BGA also produce endotoxins, which are contact irritants. They are generally only a nuisance and can affect around 15% of healthy people coming into contact with them. Symptoms include dermatitis, conjunctivitis, stomach cramps, nausea, fever, headaches and flu-like symptoms.

In Australia, several species can be hepatotoxic, including *Microcystis aeruginosa*, *Microcystis flos-aquae*, *Cylindrospermopsis racaborski* and *Nodularia spumigena*. *Anabaena circinalis* is the main neurotoxin producer in Australia.

A few epidemiological studies conducted in Australia have shown contact irritation following exposure to BGA.^{5–8}

Management of BGA occurs on several levels and varies from reducing nutrients entering the water body, placement of warning signs and BGA cell removal in water filtration plants.

References

1. Kay D, Fleisher JM, Salmon RL, Jones F, Wyer MD, Godfree AF et al. Predicting likelihood of gastroenteritis from sea bathing: results from randomised exposure. *Lancet* 1994; 344: 905–9. doi:10.1016/S0140-6736(94)92267-5
2. Fleisher JM, Kay D, Salmon RL, Jones F, Wyer MD, Godfree AF. Marine Waters Contaminated with Domestic Sewage: Nonenteric Illnesses Associated with Bather Exposure in the United Kingdom. *Am J Public Health* 1996; 86: 1228–34.
3. Corbett SJ, Rubin GL, Curry GK, Kleinbaum DG. The Health Effects of Swimming at Sydney Beaches. *Am J Public Health* 1993; 83: 1701–6.
4. NHMRC. Australian guidelines for recreational use of water. Australian Government, 1st edition, 1990.
5. Pilotto LS, Douglas RM, Burch MD, Cameron S, Beers M, Rouch GJ et al. Health effects of exposure to cyanobacteria (blue-green algae) during recreational water-related activities. *Aust N Z J Public Health* 1997; 21: 562–6.
6. Pilotto L, Hobson P, Burch MD, Ranmuthugala G, Attenwell R et al. Acute skin irritant effects of cyanobacteria (blue-green algae) in healthy volunteers. *Aust NZ J Pub Health* 2004; 28: 220–4.
7. Stewart I, Webb PM, Schluter PJ, Fleming LE, Burns JW, Gantar M et al. Epidemiology of recreational exposure to freshwater cyanobacteria – an international prospective cohort study. *BMC Public Health* 2006; 6: 93. doi:10.1186/1471-2458-6-93
8. Stewart I, Robertson IM, Webb PM, Schluter PJ, Shaw GR. Cutaneous hypersensitivity to freshwater cyanobacteria – human volunteer studies. *BMC Dermatol* 2006; 6: 6. doi:10.1186/1471-5945-6-6

Anthrax

What is anthrax?

Anthrax is a bacterial disease caused by infection with *Bacillus anthracis*. The same bacteria can lead to three forms of the disease:

- cutaneous anthrax
- intestinal anthrax
- inhalational (or pulmonary) anthrax.

Anthrax occurs among grazing animals in many parts of the world, including livestock in parts of western NSW. Anthrax is a very rare disease in humans.

What are the symptoms?

- People who contract cutaneous anthrax develop dark coloured, painless lesions within 3 to 10 days (usually between 5 and 7 days) of exposure. These lesions can be associated with swelling of the surrounding tissue. Even without treatment, four out of five people with cutaneous anthrax survive. With treatment, patients generally make a full recovery.
- People who contract intestinal anthrax develop abdominal pain and fever between 3 and 7 days after exposure, and typically death follows soon after.
- People who contract anthrax by inhalation may first have flu-like symptoms. Over several days, the disease can progress with severe breathing difficulties and shock. Inhalational anthrax has a 60–90% fatality rate. The incubation period for inhalational anthrax is most frequently between 1 and 5 days but may be as long as 60 days.

How is it spread?

- In approximately 95% of cases of anthrax, the bacteria gain entrance through broken skin or wounds (which can cause cutaneous anthrax) from a source such as the carcass of an infected animal.
- Anthrax bacteria can also be ingested in poorly prepared meat from infected animals (which can cause intestinal anthrax) or breathed in (which can cause inhalational or pulmonary anthrax). Intestinal and inhalational anthrax in humans have not been recorded in Australia.
- In late 2001, several people in the USA contracted anthrax from spores that were maliciously distributed through the mail. Both cutaneous and inhalational anthrax were reported.
- Anthrax bacteria may remain in the soil for many years in the form of spores that survive being dried out. These spores are usually the cause of infections in grazing animals. However, human infection from

the source of spores is unlikely, as a large concentration of spores is needed for infection to occur.

- Anthrax is not known to be transmitted from person to person.

Who is at risk?

Each year several cases of anthrax in livestock are reported. The handling of infected animals and their carcasses represents a risk to people.

How is it prevented?

- Anyone who handles material potentially contaminated with anthrax should wear gloves, overalls and rubber boots, and ensure that skin breaks are protected with sealed waterproof dressings.
- All contaminated items and clothing should be stored in labelled double plastic bags until exposure to anthrax is excluded. If anthrax is confirmed, all contaminated items need to be either incinerated or sterilized at 121°C for 30 minutes.
- Thorough hand washing and showering with soap are also a very important protection against infection.
- In some cases where a person has had significant exposure to anthrax spores, antibiotics may help prevent infection.
- A vaccine is available to people who have an ongoing risk of exposure, such as workers handling infected animals or animal products. However, immunisation is not recommended for the general population due to the extremely low risk of infection.

How is it diagnosed?

- Confirmation requires isolation of anthrax bacteria from the blood, skin lesions or respiratory secretions of patients.
- Cutaneous anthrax can be suspected based on the appearance of the ulcer.

How is it treated?

Several antibiotics, including penicillin, doxycycline and ciprofloxacin, can be used to treat anthrax infections.

What is the public health response?

- Laboratories must notify the local public health unit of any suspected or confirmed anthrax cases.
- Public health unit staff will investigate all cases to find out how the infection occurred, identify other people at risk of infection, implement control measures and provide other advice.

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

Communicable Diseases Report, NSW, March and April 2008

**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 **Infectious Diseases**. The communicable diseases site now uses browser-friendly html formats to improve accessibility and, as a result, has a new address <http://www.health.nsw.gov.au/publichealth/infectious/index.asp>.

Tables 1 and 2 and Figure 1 show reports of communicable diseases received through to the end of April 2008 in NSW.

Measles continues to circulate in NSW

Three confirmed cases of measles were notified during March in the Sydney area. One case of measles was confirmed in a partially immunised female aged in her twenties who had recently returned from England. An unimmunised contact who received Normal Human Immunoglobulin (NHIG) on day 7 post-exposure subsequently developed symptoms of sore throat, cough, coryza, fever and rash, and was confirmed with measles. A male aged in his forties, who recently returned from travel to Japan, was also confirmed with measles in March.

During April, a further seven cases of measles were confirmed in young adults ranging in age from 16 to 28 years, bringing the total number of cases to 20 in NSW this year. One of these cases was an international student from an English language college in Sydney. Three cases were subsequently linked to this case: a household contact; a staff member from the college; and a student from the college, all aged in their 20s.

In response to these cases, public health units across Sydney have conducted clinics to promote immunisation for susceptible contacts. Children and young adults born during or since 1966 who have never had measles and people who travel overseas should make sure they have had two doses of MMR vaccine. For more information, see: <http://www.health.nsw.gov.au/PublicHealth/Infectious/a-z.asp>.

Enterics disease

In March, NSW public health units investigated 14 outbreaks of gastroenteritis including 10 suspected to be caused by person-to-person spread and four suspected to be foodborne.

Of the suspected person-to-person outbreaks, six were reported from aged care facilities where 81 people were affected. Four outbreaks were reported from childcare centres where 27 people were affected. All were suspected to be caused by viral infections, although norovirus was confirmed in only one outbreak that was in an aged care facility.

The four outbreaks that were suspected to be foodborne, affected 101 people (ranging from seven to 50 people per outbreak). In the largest outbreak, 50 of approximately 100 people residing in an institutional setting were ill with symptoms, including vomiting and diarrhoea. *Clostridium perfringens* toxin was identified in stool specimens from three ill patients. Epidemiological evidence suggested that a curry meal was the likely vehicle for infection. In another outbreak, *Salmonella* bacteria were identified in some of the stools of 14 people who were ill after eating a common meal. The epidemiological investigation suggested that a dessert that had included raw eggs was the likely vehicle. The cause of the remaining two outbreaks remains unclear.

In April, NSW public health units investigated 20 outbreaks of gastroenteritis including 16 where person-to-person spread was implicated, two suspected to be foodborne and two suspected to be related to an environmental exposure. The NSW Food Authority inspected commercial premises associated with these outbreaks.

The 16 outbreaks where person-to-person spread was suspected affected a total of 178 people. Eight occurred in child care centres and affected 82 people, seven occurred in aged care facilities and affected 90 people, and one occurred in a hospital and affected six people. Clinical specimens were submitted for testing from six of 13 suspected person-to-person gastroenteritis outbreaks. Rotavirus was confirmed in stool samples from one aged care facility outbreak, and in another both rotavirus and Norovirus were identified in stool samples. The causative agent was not confirmed for the remaining outbreaks.

Of the two suspected foodborne gastroenteritis outbreaks, one was a small cluster of three cases of *Salmonella*

Typhimurium infection. All cases reported eating take-away salad that contained mayonnaise dressing made from raw egg (a known risk factor for salmonellosis).

One of the suspected environmental exposure outbreaks was due to Shiga toxin-producing *Escherichia coli* (STEC) O26 among a group of 250 Japanese students who were visiting Sydney during part of the incubation period for their illness. In total, 75 students (including 39 asymptomatic students) tested positive for STEC O26 after they returned home to Japan. STEC is carried by animals, such as cattle. People are infected when they come into contact with the faeces of an infected animal or person, either directly or indirectly. STEC is spread through consuming contaminated food (e.g. undercooked burgers, unwashed salad vegetables and unpasteurised milk or milk products), drinking or swimming in contaminated water, person-to-person contact (e.g. contact with faeces of an infected person) and contact with animals on farms or petting zoos. The students had visited a wildlife park and eaten at several restaurants. Despite an investigation, the source of infection, whether in Australia or Japan, remains unclear.

An outbreak of *Salmonella* Bioser Java that is clustered around the Northern Beaches area, and suspected to be due to an environmental exposure, is currently under investigation.

An outbreak of *Salmonella* Typhimurium (MLVA type 3-12-9-10-550) that was reported in February has continued throughout March and April. A total of 65 cases have now been reported with most infections occurring in March. Of the 65 cases, 37 (57%) were male and the median age of cases was 19 years (range 1–84 years). Cases mainly lived in metropolitan Sydney and an exploratory investigation commenced in mid April. Hypothesis-generating interviews have been conducted and, although the source of the outbreak remains unclear, 13 of 18 cases reported eating eggs during the incubation period. Of these 13, seven reported eating raw eggs, including two young males who drank raw egg milkshakes. The NSW Food Authority is assisting with the ongoing investigation.

Murray Valley Encephalitis

In February 2008, Murray Valley Encephalitis (MVE) was detected in *Culex annulirostris* mosquitoes that were trapped near Griffith. In March 2008, MVE was detected

in sentinel chicken flocks at Macquarie Marshes in western NSW and Leeton in the Riverina area of southern NSW. Seroconversions of sentinel chickens were also subsequently reported in three Victorian locations along the Murray River.

The majority of people infected with MVE will have no symptoms. Of those who do, symptoms include:

- high fever
- severe headache
- seizures or fits (especially in young children)
- tremors
- neck stiffness
- lethargy, irritability, drowsiness
- vomiting
- nausea
- diarrhoea
- dizziness
- confusion
- coma in severe cases.

Previous seroconversions occurred in flocks of sentinel chickens in NSW in 2001 and 2003 without associated human illness.

As part of enhanced surveillance for possible human cases of MVE, public health units from the Greater West, Hunter New England and Greater Southern Area Health Services have worked with selected local general practitioners to promote serological testing of patients presenting to general practitioners and local hospitals with consistent symptoms. No evidence of recent seroconversion to MVE has been found in those who were tested and there have been no reports of clinical cases of MVE in these areas to the end of April. As mosquito activity falls with the low temperatures in autumn and winter, the risk of human transmission is expected to decrease.

Public health units issued alerts to their local communities about avoiding mosquito bites. The advice included that people who live in or who visit these areas should avoid being outside in the late afternoon and at dusk, wear light-coloured, long-sleeved, loose-fitting clothing and use an effective insect repellent. Residents should also remove any containers that may hold water from around their homes and fit fly screens to their windows and doors.

Figure 1. Reports of selected communicable diseases, NSW, January 2004 to April 2008, 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 infections; RRV, Ross River virus infections; 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%

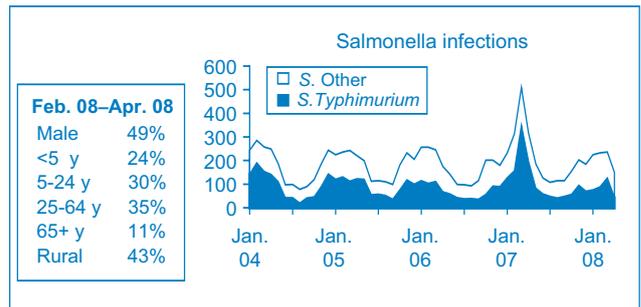
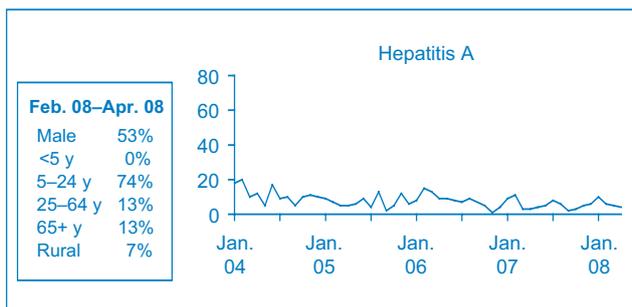
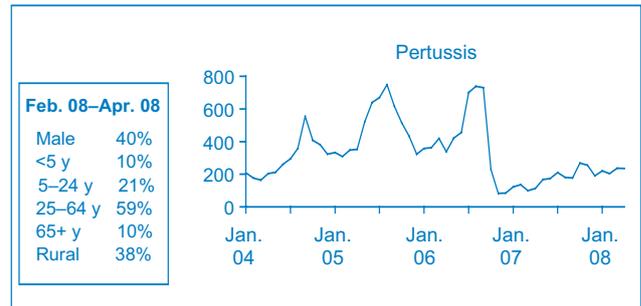
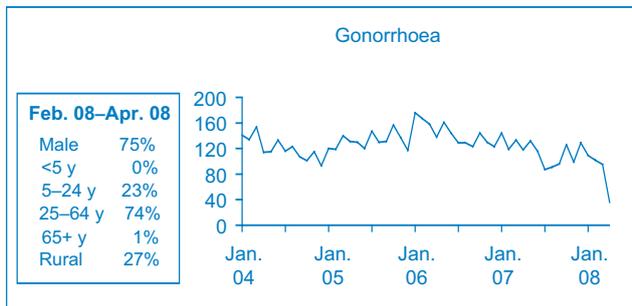
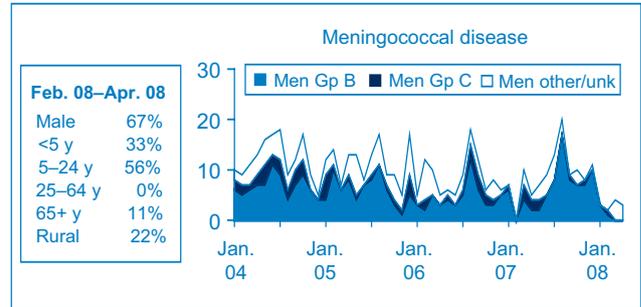
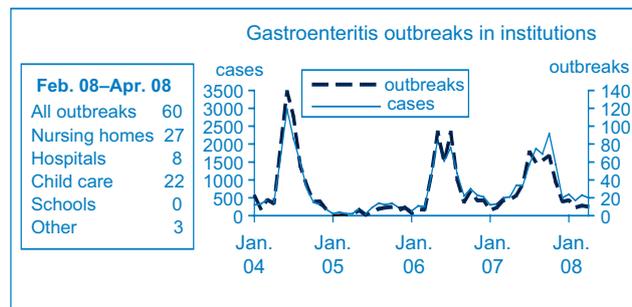
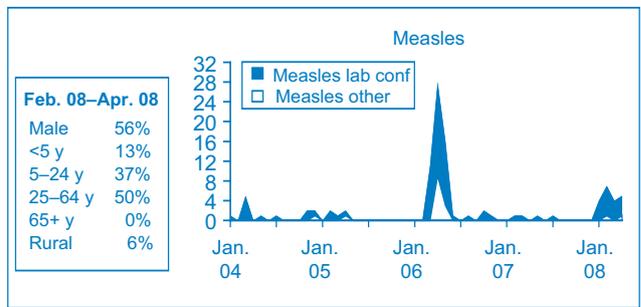
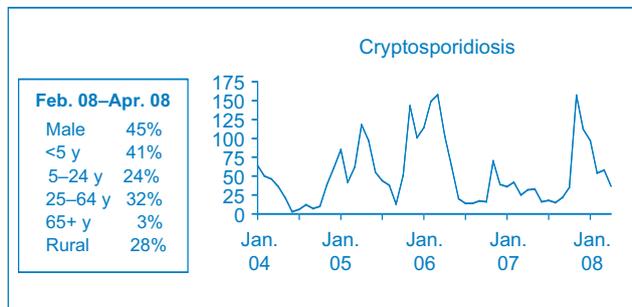
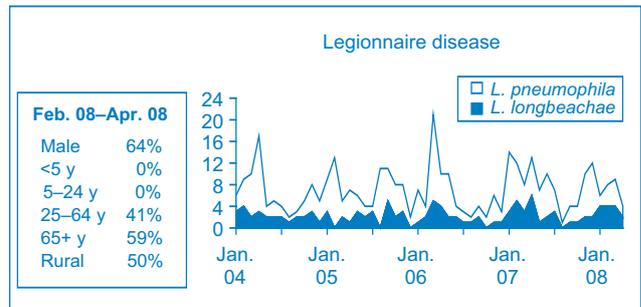
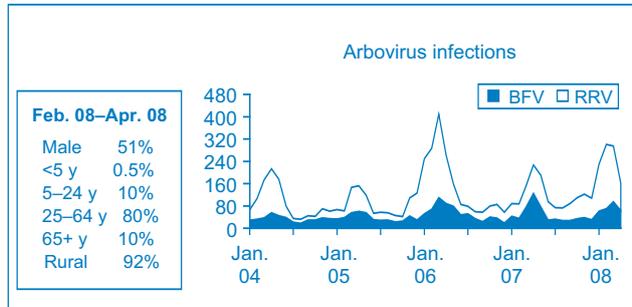


Table 1. Reports of notifiable conditions received in March 2008 by Area Health Services

Condition	Area Health Service (2008)												Total For March ^c	To date ^c Total							
	Greater Southern			Greater Western			Hunter New England		North Coast			Northern Sydney Central Coast			Sydney South West		Sydney West		JHS		
	GMA	SA	FWA	MAC	MWA	HUN	NEA	MNC	NRA	CCA	NSA	ILL	SES	CSA	SWS	WEN	WSA	JHS			
Bloodborne and sexually transmitted																					
Chancroid ^d	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlamydia (genital) ^a	32	24	3	27	23	132	60	48	56	57	76	42	169	-	-	29	93	2	885	3032	
Gonorrhoea ^a	2	1	-	1	-	10	1	-	5	2	5	-	34	2	-	1	8	1	73	282	
Hepatitis B – acute viral ^a	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	1	3	3
Hepatitis B – other ^a	1	4	-	-	-	8	2	4	2	5	27	5	16	-	1	10	57	3	147	551	
Hepatitis C – acute viral ^b	1	1	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	2	3	3
Hepatitis C – other ^a	18	13	1	8	9	36	15	19	27	17	10	16	-	-	2	21	-	226	1044	1044	
Hepatitis D – unspecified ^b	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Lymphogranuloma venereum	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Syphilis	1	1	-	-	-	4	1	-	3	4	4	3	28	2	3	1	10	-	67	224	224
Vectorborne																					
Barmah Forest virus ^a	-	4	3	1	-	12	2	20	43	2	-	-	-	-	-	-	-	-	67	211	211
Ross River virus ^a	20	2	7	24	6	39	8	11	51	8	-	8	3	2	-	6	4	-	200	585	585
Arboviral infection (Other) ^a	-	1	-	-	-	-	-	-	1	2	-	2	1	1	-	1	2	-	12	38	38
Malaria ^a	-	-	-	-	-	-	-	-	-	-	1	2	1	-	2	-	1	-	9	25	25
Zoonoses																					
Anthrax ^a	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Brucellosis ^a	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Leptospirosis ^a	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	4	4
Lyssavirus ^a	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Psittacosis ^a	-	-	-	-	1	1	-	-	-	-	-	-	-	-	-	-	-	-	2	6	6
Q fever ^a	-	1	-	5	-	1	1	4	1	-	-	2	1	-	-	-	1	-	16	52	52
Respiratory and other																					
Blood lead level ^b	-	-	-	-	-	1	1	-	-	1	1	-	-	-	-	-	-	-	11	51	51
Influenza ^a	1	2	-	7	1	1	2	-	5	1	1	5	2	-	-	2	157 ^d	-	177	213	213
Invasive pneumococcal infection ^a	-	-	-	1	-	4	-	-	1	2	-	2	5	-	4	-	3	-	22	53	53
Legionella longbeachae infection ^a	-	-	-	-	-	1	-	1	1	-	-	1	-	-	-	-	-	-	4	8	8
Legionella pneumophila infection ^a	-	-	-	-	-	1	-	-	-	1	-	1	-	-	1	1	-	-	5	13	13
Legionnaire disease (other) ^b	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Leptosy	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Meningococcal infection (invasive) ^a	1	-	-	-	-	2	-	-	-	-	-	3	1	2	-	2	1	-	2	7	7
Tuberculosis	-	-	-	-	-	-	-	-	-	-	3	1	3	2	-	2	-	-	14	83	83
Vaccine-preventable																					
Adverse event after immunisation	2	4	-	2	5	2	-	-	1	1	1	2	2	3	1	4	7	-	38	80	80
H. influenzae b infection (invasive) ^b	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Measles	-	-	-	-	-	-	-	-	-	1	1	1	1	-	1	-	-	-	3	13	13
Mumps ^a	-	-	-	-	-	-	-	1	1	-	-	-	3	-	-	-	1	-	6	54	54
Pertussis	4	6	-	4	2	13	3	7	41	1	23	14	28	18	21	8	34	-	227	663	663
Rubella ^a	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Enteric																					
Botulism	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cholera ^a	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cryptosporidiosis ^a	2	1	-	2	3	15	4	2	3	6	14	1	17	1	1	1	5	-	53	218	218
Giardiasis ^a	2	7	-	3	6	15	4	1	3	6	26	8	17	-	-	10	16	-	129	457	457
Haemolytic uraemic syndrome	-	-	-	-	-	-	-	1	-	-	-	-	1	-	2	-	1	-	4	2	2
Hepatitis A ^a	-	-	-	-	-	-	-	-	-	-	-	-	1	1	1	-	-	-	4	22	22
Hepatitis E ^a	-	-	-	-	-	-	-	-	-	-	-	-	1	1	1	-	-	-	2	4	4
Listeriosis ^a	-	-	-	-	-	23	6	6	18	21	27	14	32	3	1	6	34	-	215	654	654
Salmonellosis ^a	19	3	-	1	-	-	-	-	-	-	-	1	1	1	1	1	-	-	3	20	20
Shigellosis ^a	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	2	9	9
Typhoid ^d	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	2	6	6
Verotoxin producing E. coli ^{2a}	1	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-
Miscellaneous																					
Creutzfeldt-Jakob disease	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Meningococcal conjunctivitis	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

^aLaboratory-confirmed cases only. ^bHIV and AIDS data are reported separately in the Public Health Bulletin quarterly. ^cIncludes cases with unknown postcode. ^dArtefact due to results of a clinical trial being batch reported. NB: From 1 January 2005, Hunter/New England AHS also comprises Great Lakes, Gloucester and Greater Taree LGAs, Sydney West also comprises Greater Lithgow LGA. NB: Data is 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. GMA, Greater Murray Area; MAC, Macquarie Area; MWA, Mid Western Area; NEA, New England Area; NRA, Northern Rivers Area; SA, Southern Area; SES, South Eastern Sydney Area; SWS, South Western Sydney Area; WEN, Wentworth Area; WSA, Western Sydney Area; CSA, Central Sydney Area; FWA, Far West Area; HUN, Hunter Area; MNC, North Coast Area; NSA, Northern Sydney Area; WSA, Western Sydney Area; MWA, Mid Western Area; SWS, South Western Sydney Area; JHS, Justice Health Service.

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