NSW PUBLIC HEALTH BULLETIN

Year In Review 2006

Year in review: communicable disease surveillance, New South Wales, 2006

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In this issue, we review the trends in reports of notifiable diseases among NSW residents received by the NSW Public Health Units (PHU) for 2006. Readers interested in the details of notifications for specific diseases are referred to Tables 1–5, where diseases are reported by: year of onset, month of onset, Area Health Service (AHS), age group and sex. Table 6 shows the number of people with selected notifiable conditions who were reported to have died at the time of follow-up by their local PHU.

Trends

Among the 47 608 NSW residents with medical conditions notified by doctors, hospital staff, and laboratory staff for 2006, highlights included:

Conditions most frequently reported
- Chlamydia: 11 864 cases (173 per 100 000 people), with the highest crude rates by geographical area in the South Eastern Sydney/Illawarra (Randwick region), Hunter/New England (Newcastle region) and Sydney South West (Camperdown region) AHSs
- Pertussis: 4918 cases (73 per 100 000 people), with the highest crude rates in the Greater Western (Broken Hill and Dubbo regions) and Greater Southern (Albury region) Health Areas. Note, a proportion of pertussis notifications may have been false-positive tests caused by the performance of a commonly used serology test kit (see http://www.health.nsw.gov.au/infect/pdf/false_pertussis_tests.pdf)
- Hepatitis C: 4470 cases (65 per 100 000 people), with the highest crude rates in the Sydney South West (Camperdown region), North Coast (Lismore region) and Greater Western (Bathurst region) AHSs
- Hepatitis B: 2543 cases (37 per 100 000 people), with the highest crude rates in the Sydney South West and Sydney West (Parramatta region) AHSs

- Salmonella infections: 2058 cases (26 per 100 000 population), with the highest crude rates in the North Coast (Lismore region), Greater West (Dubbo region) and Hunter New England (Tamworth region) AHSs.

Conditions with the most meaningful declines in the number of notifications compared with previous years
- Hepatitis B: 2543 cases, steadily declining from 4458 cases in 2001, with the most pronounced fall (43% since 2002) in the 35 to 44 year age group, possibly as a result of the ongoing impact of immunisation
- Pneumococcal disease (invasive): 564 cases, a continued decline from 906 in 2004 and most pronounced in the 0-to-2-year age group, possibly a result of the inclusion of the pneumococcal vaccination on the universal childhood vaccination schedule in early 2005
- Meningococcal disease: 107 cases, a continued decline from 253 in 2000, the lowest number of notifications since laboratory reporting began in 1991 and in part due to the introduction of meningococcal C vaccination in late 2003
- Leptospirosis: 17 cases, down from 66 in 2001, the cause of the decline is unclear.

Conditions with the most meaningful increases in the number of notifications compared with previous years
- Chlamydia: 11 864 cases, continuing its rise since it became notifiable in the late 1990s
- Infectious syphilis: re-emerging as a significant issue in the gay community in late 2006
- Barmah Forest virus infection: 644 cases, due to outbreaks mainly in coastal areas of rural NSW in early 2006
- Ross River virus infection: 1225 cases, up from 583 in 2005, due to outbreaks across rural NSW in early 2006

- Gastroenteritis in institutions: 10636 cases associated with 436 outbreaks, mostly due to norovirus infections
- Mumps: 154 cases, up from 111 in 2005, mainly in young adults and the highest number of laboratory notifications since laboratory reporting began in 1991
- Legionnaires' disease, due to infection with *Legionella pneumophila*: 54 cases, in part due to outbreaks in western and northern Sydney
- Measles: 48 cases, due to two outbreaks, one in March 2006 related to contact at a hospital emergency department and the second, larger outbreak, in people associated with a national spiritual tour.

Note that the apparent increase in giardiasis (1720 cases) and verotoxigenic *Escherichia coli* infections (10 cases) are probably related to changes in laboratory testing practices resulting in a higher proportion of samples tested for these organisms than in the past.

Conditions least frequently reported

There were no reported cases of avian influenza, botulism, chancroid, diphtheria, donovanosis, granuloma inguinale, plague, polio, rabies/lyssavirus, SARS, smallpox, tularaemia, typhus, viral haemorrhagic fevers or yellow fever in NSW in 2006.

Conditions associated with the largest numbers of reported deaths

Deaths reported via the surveillance mechanisms for notifiable conditions may not include all deaths associated with these conditions. PHUs routinely investigate all cases of some notifiable conditions (eg, tuberculosis, measles, meningococcal disease) in order to implement control measures; the investigations are likely to identify associated deaths. However, there are other notifiable conditions (eg, chlamydia and gonorrhoea) where PHUs do not conduct routine investigations and for which deaths will not be identified as part of the notification or public health management process. Conversely, some deaths in patients following notification may be due to conditions other than that notified. Deaths were most frequently reported for the following notifiable diseases:

- Invasive pneumococcal disease: 61 deaths
- HIV infection: 36 deaths, including 13 people who died from AIDS, and 23 people with HIV infection who died of causes other than AIDS
- Tuberculosis: 21 deaths.

Outbreaks and threats

Several notable disease outbreaks and threats were reported in 2006 in NSW. These included:

- Outbreaks of Legionnaires' disease in western Sydney in January 2006[1] and northern Sydney in March 2006[2] thought to be related to contaminated cooling towers.
- Two outbreaks of measles[2]
- An outbreak of keratoconjunctivitis in patients of an eye clinic in regional NSW in March 2006[2]
- Four cases of lymphogranuloma venereum identified in early 2006, the first cases in NSW[3]
- A cluster of cases of *Salmonella* Saint-Paul infection in October 2006 linked to contaminated rockmelons[4]
- Three cases of cholera in November 2006 linked to consumption of raw imported whitebait.[5]

Conclusions

Sexually transmitted infections, gastroenteritis (particularly norovirus) and arboviruses continue to be significant public health problems in NSW. Much progress has been made with other diseases, such as hepatitis B, invasive pneumococcal disease and meningococcal disease, in which declines were identified. Outbreaks of diseases, that have largely been brought under control, for example measles and mumps (where many young adults remain unvaccinated), continue to pose a threat.

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 PHUs for investigation and control.

References

1. NSW Department of Health Communicable Diseases Report, NSW, for January and February 2006. *N S W Public Health Bull* 2006; 17(3–4): 58–9.

2. NSW Department of Health Communicable Diseases Report, NSW, for March and April 2006. *N S W Public Health Bull* 2006; 17(5–6): 88–91.

3. NSW Department of Health Communicable Diseases Report, NSW, for July and August 2006. *N S W Public Health Bull* 2006; 17(9–10): 154–5.

4. NSW Department of Health Communicable Diseases Report, NSW, for September and October 2006. *N S W Public Health Bull* 2006; 17(11–12): 182–3.

5. NSW Department of Health Communicable Diseases Report, NSW, for November and December 2006. *N S W Public Health Bull* 2007; 18(1–2): 28–32.

Table 1. Disease notifications by year of onset of illness[a], NSW, 1991–2006

Condition	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Adverse events after immunisation	9	31	23	40	28	56	70	95	16	42	111	178	219	184	106	65
Arboviral infection	409	343	656	381	539	1227	1806	783	1220	980	1191	664	1024	1148	1087	1920
Barmah Forest virus[b]	6	6	25	39	271	172	185	134	249	197	401	396	451	403	448	644
Ross River virus[b]	297	324	599	331	236	1031	1598	583	952	750	717	182	494	701	583	1225
Other[b]	106	13	32	11	32	24	23	66	19	33	73	86	79	44	56	51
Blood lead level ≥15 µg/dL[b]	Not notifiable until December 1996						710	874	691	985	513	516	338	304	234	281
Botulism	0	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0
Brucellosis[b]	2	2	4	4	2	1	3	3	2	1	1	2	3	7	3	9
Chancroid[b]	Not notifiable until December 1998								1	0	0	0	0	0	0	0
Chlamydia trachomatis infection[b]	Not notifiable until August 1998								2469	3509	4500	5823	7788	10020	11285	11864
Cholera[b]	1	0	1	0	1	3	1	1	2	0	1	1	0	1	0	3
Creutzfeldt–Jakob disease[b]	Not notifiable until April 2004													6	8	8
Cryptosporidiosis[b]	Not notifiable until December 1996						157	1130	121	133	195	306	203	357	849	779
Food-borne illness (NOS)[e]	2765	253	106	213	270	211	255	201	151	147	56	41	1071	550	309	507
Gastroenteritis (institutional)	158	406	443	296	1359	554	939	738	673	697	775	1752	3583	12784	1395	10636
Giardiasis[b]	Not notifiable until August 1998								1091	978	967	864	1028	1235	1447	1720
Gonorrhoea[b]	392	491	382	357	428	522	636	1054	1291	1060	1364	1527	1328	1442	1579	1696
Haemolytic uraemic syndrome	Not notifiable until December 1996						3	6	11	9	2	7	5	9	11	11
H. influenzae type b	212	217	124	61	29	13	17	11	13	8	7	10	6	5	7	11
Hib epiglottitis[b]	15	57	32	21	6	2	5	1	2	2	1	1	0	3	0	1
Hib meningitis[b]	48	103	53	17	11	4	3	3	3	1	1	1	0	0	2	0
Hib septicaemia[b]	11	26	24	12	8	3	1	4	6	4	2	3	1	2	4	6
Hib infection (NOS) [b]	138	31	15	11	4	4	8	3	2	1	3	5	5	0	1	4
Hepatitis A[b]	1119	901	579	585	614	958	1426	927	421	201	197	149	124	137	83	95
Hepatitis B	1492	3169	3603	3984	4007	3505	3167	2957	3513	3973	4558	3547	2844	2812	2742	2543
Hepatitis B: acute viral[b]	409	112	95	74	61	43	53	58	77	100	94	88	74	53	56	54
Hepatitis B: other[b]	1083	3057	3508	3910	3946	3462	3114	2899	3436	3873	4464	3459	2770	2759	2686	2489
Hepatitis C	851	3895	5897	7820	6878	7001	6925	7206	8602	8293	8659	6694	5248	4916	4365	4470
Hepatitis C: acute viral[b]	22	26	22	16	32	18	19	112	112	222	295	151	127	59	43	40
Hepatitis C: other[b]	829	3869	5875	7804	6846	6983	6906	7094	8490	8071	8364	6543	5121	4857	4322	4430
Hepatitis D[b]	0	8	12	19	19	9	11	3	14	12	11	9	12	14	15	15
Hepatitis E[b]	0	0	1	2	0	3	6	4	7	9	6	6	6	8	7	10
HIV/AIDS																
HIV infection[b]	822	694	590	502	537	447	422	403	378	356	340	394	413	408	395	371
AIDS	441	433	483	555	483	376	214	181	133	134	102	117	148	100	109	84
Influenza											244	1012	861	1011	1414	616
Influenza – Type A[b]	Not notifiable until December 2000										216	770	767	797	1055	420
Influenza – Type B[b]	Not notifiable until December 2000											27	241	55	161	280	150
Influenza – Types A & B[b]	Not notifiable until December 2003													26	65	37
Influenza – Type (NOS) [b]	Not notifiable until December 2000										1	1	39	27	14	9
Legionellosis	37	104	66	60	75	74	33	46	41	41	68	44	60	80	89	77
L. longbeachae[b]	0	14	13	8	16	30	9	19	12	12	29	21	37	27	24	22
L. pneumophila[b]	16	80	34	30	35	34	18	22	22	26	38	22	23	51	64	54
Legionnaires' disease – other	21	10	19	22	24	10	6	5	7	3	1	1	0	2	1	1
Leprosy	1	7	5	3	3	2	0	0	1	2	4	0	2	5	1	1
Leptospirosis[b]	28	21	16	14	6	33	33	50	56	54	66	39	39	40	35	17
Listeriosis[b]	11	13	12	10	14	22	23	28	22	18	12	11	28	30	25	26
Lymphogranuloma venereum (LGV) [b]	0	0	0	0	0	0	0	0	0	0	0	0	0	1	2	1
Malaria[b]	171	110	174	184	96	204	173	158	174	232	157	105	120	101	206	140
Measles	495	805	2348	1484	596	191	273	119	32	36	31	8	18	12	5	60
Measles lab confirm[b]	19	76	460	302	138	35	98	19	13	22	18	6	14	11	4	48
Measles (other)	476	729	1888	1182	458	156	175	100	19	14	13	2	4	1	1	12
Meningococcal disease	128	121	153	142	113	161	218	186	221	253	234	216	202	149	140	107
Meningococcal – Type B[b]	0	3	7	7	23	36	53	55	95	93	90	105	100	81	73	54
Meningococcal – Type C[b]	0	4	6	9	8	35	55	55	60	64	38	54	45	24	16	13
Meningococcal – Type W135[b]	0	0	0	0	1	0	2	4	4	4	2	2	2	5	8	5
Meningococcal – Type Y[b]	0	0	1	1	0	1	0	7	1	7	2	2	5	3	3	1
Meningococcal disease – Other	128	114	139	125	81	89	108	65	61	85	102	53	50	36	40	34
Mumps[b]	8	23	13	11	14	27	29	39	33	92	28	29	35	65	111	154
Paratyphoid[b,d]	20	8	9	11	12	15	5	9	5	14	11	13	22	10	0	0
Pertussis	49	217	1533	1405	1369	1156	4246	2309	1415	3691	4437	2012	2772	3567	5809	4918
Pneumococcal disease (invasive) [b]	Not notifiable until December 2000										444	861	802	906	641	564
Psittacosis[b]	Not notifiable until December 2000										38	155	87	81	121	94
Q fever[b]	167	213	403	267	201	287	258	236	164	132	144	309	288	223	143	175
Rubella[b]	60	324	1186	233	2376	636	153	78	46	191	58	35	24	18	10	37
Rubella (congenital) [b]	1	0	2	4	1	5	0	0	1	0	0	0	1	1	0	0
Rubella[b]	59	324	1184	229	2375	631	153	78	45	191	58	35	23	17	10	37
Salmonella infection[b,d]	1170	802	980	1101	1366	1224	1698	1812	1438	1399	1644	2100	1839	2134	2176	2058
Shigellosis[b]	Not notifiable until December 2000										134	85	59	96	135	75
Syphilis	580	873	730	965	833	662	512	611	585	580	547	646	843	1042	840	881
Syphilis infectious[b,c]	1	3	6	29	132	72	57	45	86	80	67	128	245	302	242	210
Syphilis congenital	1	1	0	2	6	3	3	0	3	2	1	1	2	1	5	4
Syphilis other[b]	578	869	724	934	695	587	452	566	496	498	479	517	596	739	593	667
Tetanus	5	2	5	4	0	1	3	3	1	2	0	0	1	1	1	2
Tuberculosis[b]	429	394	389	394	443	410	422	382	483	448	416	447	386	430	449	461
Typhoid[b]	38	20	28	25	27	30	28	18	32	28	32	26	16	39	28	35
Verotoxigenic *Escherichia coli* infection[b]	Not notifiable until December 1996						2	0	1	1	6	3	5	16	10

[a]Year of onset: the earlier of patient-reported onset date, specimen date or date of notification.
[b]Laboratory-confirmed cases only.
[c]Includes Syphilis primary, Syphilis secondary, Syphilis <1 year duration and Syphilis newly acquired.
[d]From 2005, all paratyphoid recorded as salmonellosis.
[e]Foodborne 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: Diphtheria[b], Granuloma inguinale[b], Lymphogranuloma venereum[b], Plague[b], Poliomyelitis[b], Rabies, Typhus[b], Viral haemorrhagic fever, Yellow fever.

Table 2.   Disease notifications by month of onset of illness[a], NSW, 2006

Condition	Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.	Total
Anthrax	1	0	0	0	0	0	0	0	0	0	0	0	1
Adverse events after immunisation	7	9	13	10	8	5	0	4	4	2	2	1	65
Arboviral infection	255	291	412	267	159	90	89	62	60	85	90	60	1920
Barmah Forest virus[b]	50	68	110	88	79	48	51	34	23	40	36	17	644
Ross River virus[b]	198	219	299	174	79	37	28	25	34	40	50	42	1225
Other[b]	7	4	3	5	1	5	10	3	3	5	4	1	51
Blood lead level ≥15 µg/dL[b]	35	37	52	15	20	11	13	16	20	25	20	17	281
Brucellosis[b]	0	0	1	0	0	0	2	1	1	2	2	0	9
Chlamydia trachomatis infection[b]	1051	1020	1141	852	1084	965	916	988	930	958	1061	898	11864
Cholera[b]	0	0	0	0	0	0	0	0	0	0	3	0	3
Creutzfeldt–Jakob disease[b]	0	2	0	2	0	1	0	0	1	0	0	2	8
Cryptosporidiosis[b]	115	149	158	103	63	20	14	14	17	16	70	40	779
Gastroenteritis (institutional)	0	1	0	0	0	0	0	0	0	0	0	0	1
Giardiasis[b]	154	221	177	151	150	112	119	164	87	114	137	134	1720
Gonorrhoea[b]	176	167	158	138	154	142	127	127	119	138	128	122	1696
Haemolytic uraemic syndrome	3	1	0	0	0	0	0	0	0	0	4	3	11
H. influenzae type b	0	1	0	0	0	1	2	1	4	0	1	1	11
Hib epiglottitis[b]	0	0	0	0	0	0	0	0	0	0	0	1	1
Hib septicaemia[b]	0	1	0	0	0	0	1	1	2	0	1	0	6
Hib infection (NOS)[b]	0	0	0	0	0	1	1	0	2	0	0	0	4
Hepatitis A[b]	8	15	13	9	9	8	7	9	7	5	1	4	95
Hepatitis B	203	187	191	187	235	215	221	212	241	209	221	221	2543
Hepatitis B: acute viral[b]	6	7	3	7	2	6	3	0	3	3	10	4	54
Hepatitis B: other[b]	197	180	188	180	233	209	218	212	238	206	211	217	2489
Hepatitis C	376	402	416	277	359	358	364	375	413	354	396	380	4470
Hepatitis C: acute viral[b]	2	7	7	4	5	2	5	0	4	1	3	0	40
Hepatitis C: other[b]	374	395	409	273	354	356	359	375	409	353	393	380	4430
Hepatitis D[b]	1	1	1	2	3	1	2	2	0	1	1	0	15
Hepatitis E[b]	1	1	1	0	0	1	1	1	0	1	2	1	10
HIV/AIDS													
HIV infection[b]	26	40	38	24	27	19	29	31	26	36	47	28	371
AIDS	9	9	7	5	4	10	5	8	6	10	6	5	84
Influenza	11	9	13	13	19	25	114	205	116	45	29	17	616
Influenza – Type A[b]	8	7	7	8	10	11	71	153	82	32	25	6	420
Influenza – Type B[b]	2	0	2	3	6	13	37	45	28	6	1	7	150
Influenza – Types A & B[b]	1	0	4	2	3	1	6	7	3	6	1	3	37
Influenza – Type (NOS)[b]	0	2	0	0	0	0	0	0	3	1	2	1	9
Legionellosis	8	4	21	10	10	4	3	2	4	2	6	3	77
L. longbeachae[b]	1	2	5	4	2	2	1	1	2	0	1	1	22
L. pneumophila[b]	6	2	16	6	8	2	2	1	2	2	5	2	54
Legionnaires' disease – other	1	0	0	0	0	0	0	0	0	0	0	0	1
Leprosy	0	0	0	0	0	0	0	0	1	0	0	0	1
Leptospirosis[b]	3	2	3	2	3	0	0	0	1	1	1	1	17
Listeriosis[b]	2	1	6	3	1	1	0	2	5	1	2	2	26
Lymphogranuloma venereum (LGV)[b]	0	0	1	0	0	0	0	0	0	0	0	0	1
Malaria[b]	9	13	13	3	11	17	9	15	15	15	9	11	140
Measles	0	0	11	28	16	1	0	1	0	2	1	0	60
Measles lab confirm[b]	0	0	11	19	13	1	0	1	0	2	1	0	48
Measles (other)	0	0	0	9	3	0	0	0	0	0	0	0	12
Meningococcal disease	5	12	10	5	6	6	10	19	13	7	8	6	107
Meningococcal – Type B[b]	3	2	5	3	4	3	5	12	6	3	3	5	54
Meningococcal – Type C[b]	0	2	0	0	1	0	1	3	3	2	1	0	13
Meningococcal – Type W135[b]	0	1	0	0	0	0	1	0	1	1	1	0	5
Meningococcal – Type Y[b]	0	0	0	0	1	0	0	0	0	0	0	0	1
Meningococcal disease – Other	2	7	5	2	0	3	3	4	3	1	3	1	34
Mumps[b]	13	4	2	10	17	24	31	19	14	6	7	7	154
Pertussis	359	362	419	338	420	455	700	740	730	228	81	86	4918
Pneumococcal disease (invasive) [b]	28	22	23	50	58	65	77	83	50	44	30	34	564
Psittacosis[b]	12	10	10	7	6	7	10	5	5	13	6	3	94
Q fever[b]	22	10	9	7	13	16	18	15	16	17	15	17	175
Rubella[b]	2	0	2	1	4	7	5	4	5	3	0	4	37
Salmonella infection[b,d]	256	256	245	175	142	99	97	93	115	201	200	179	2058
Shigellosis[b]	11	11	5	5	9	3	2	5	5	6	9	4	75
Syphilis	70	70	76	49	59	66	65	63	78	92	117	76	881
Syphilis infectious[b,c]	21	16	21	13	14	12	8	10	12	15	46	22	210
Syphilis congenital	0	1	1	1	0	1	0	0	0	0	0	0	4
Syphilis other[b]	49	53	54	35	45	53	57	53	66	77	71	54	667
Tetanus	0	0	0	0	0	0	0	0	0	1	0	1	2
Tuberculosis[b]	55	40	40	33	37	41	40	47	40	36	33	19	461
Typhoid[b]	2	4	2	2	1	4	4	1	4	2	4	5	35
Verotoxigenic Escherichia coli infection[b]	1	2	1	3	0	0	0	0	0	0	1	2	10

[a]Month of onset: the earlier of patient-reported onset date, specimen date or date of notification.

[b]Laboratory-confirmed cases only.

[c]Includes Syphilis primary, Syphilis secondary, Syphilis <1 year duration and Syphilis newly acquired.

[d]From 2005, all paratyphoid recorded as salmonellosis.

NOS: not otherwise specified.

Table 3. Disease notifications by Area Health Service of residence (2005 AHS boundaries), crude rates per 100000 population, NSW, 2006[a]

Condition	Greater Southern[e]		Greater Western[e]			Hunter/New England[e]		North Coast[e]		Northern Syd/ Central Coast[e]		South Eastern Syd/Illawarra[e]		Sydney South West[e]		Sydney West[e]		Justice Health[f]
	Albury	Goulburn	Broken Hill	Dubbo	Bathurst	Newcastle	Tamworth	Pt Macquarie	Lismore	Gosford	Hornsby	Wollongong	Randwick	Camperdown	Liverpool	Penrith	Parramatta	
Anthrax	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Adverse events after immunisation	3.0	4.9	0.0	0.0	1.7	0.9	1.1	0.7	0.7	1.3	0.5	0.3	0.9	0.0	0.1	0.7	0.7	0.0
Arboviral infection	68.5	21.5	68.5	84.6	26.5	61.5	56.2	111.0	143.7	32.2	5.4	17.5	6.4	3.7	2.6	10.2	2.1	0.0
Barmah Forest virus[b]	12.8	5.4	15.0	10.5	0.6	30.9	8.9	53.0	47.7	14.5	1.0	4.8	1.6	2.0	0.1	1.2	0.3	0.0
Ross River virus[b]	55.3	15.2	53.5	74.2	25.9	30.2	47.3	57.3	93.5	17.4	3.1	12.5	3.2	1.6	1.9	8.3	1.4	0.0
Other[b]	0.4	1.0	0.0	0.0	0.0	0.4	0.0	0.7	2.5	0.3	1.4	0.3	1.6	0.2	0.6	0.6	0.4	0.0
Blood lead level ≥15 µg/dL [b]	2.6	2.4	8.6	57.1	6.3	6.6	1.7	0.4	1.4	1.3	1.5	2.9	4.7	3.7	5.1	3.7	0.7	0.0
Brucellosis[b]	0.0	0.0	0.0	0.0	1.2	0.0	0.6	0.0	0.0	0.3	0.1	0.0	0.3	0.2	0.1	0.0	0.0	0.0
Chlamydia trachomatis infection[b]	174.2	134.5	167.1	191.1	184.4	257.8	206.5	122.6	200.9	158.7	134.4	154.3	266.6	243.5	86.8	104.3	118.3	937.5
Cholera[b]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.6	0.0	0.0	0.0	0.0
Creutzfeldt–Jakob disease[b]	0.0	0.5	0.0	0.0	0.0	0.2	0.6	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.2	0.6	0.0	0.0
Cryptosporidiosis[b]	15.1	13.7	12.9	22.8	14.4	13.1	18.9	8.1	21.3	4.7	17.6	8.8	10.7	11.2	6.3	9.8	5.7	0.0
Gastroenteritis (institutional)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0
Giardiasis[b]	20.3	18.6	2.1	31.4	8.1	27.4	28.4	10.5	5.0	23.7	44.1	19.4	34.2	26.3	13.9	27.1	26.3	0.0
Gonorrhoea[b]	1.9	2.0	4.3	8.6	5.2	10.8	5.0	5.6	13.2	8.9	16.8	9.6	72.5	78.2	14.8	9.8	21.1	75.0
Haemolytic uraemic syndrome	0.0	0.0	0.0	1.0	0.6	0.2	0.0	0.0	0.4	0.0	0.1	0.3	0.1	0.2	0.2	0.0	0.1	0.0
H. influenzae type b	0.4	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.3	0.1	0.3	0.0	0.2	0.1	0.9	0.1	0.0
Hib epiglottitis[b]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0
Hib septicaemia[b]	0.4	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.3	0.1	0.0	0.0	0.0	0.0	0.6	0.0	0.0
Hib infection (NOS) [b]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.2	0.0	0.3	0.1	0.0
Hepatitis A[b]	0.4	1.0	4.3	1.0	1.2	0.4	0.0	0.0	1.1	1.6	1.2	0.8	1.7	2.0	1.6	0.6	3.1	0.0
Hepatitis B	10.5	12.2	25.7	7.6	5.2	10.5	11.7	9.8	9.6	12.7	34.2	14.3	39.6	91.7	61.0	16.3	69.6	487.5
Hepatitis B: acute viral[b]	0.8	0.5	4.3	0.0	1.2	1.2	0.6	0.4	1.1	0.3	0.4	1.1	0.7	1.4	0.5	0.7	0.7	37.5
Hepatitis B: other[b]	9.8	11.7	21.4	7.6	4.0	9.3	11.1	9.5	8.5	12.3	33.8	13.3	38.9	90.4	60.5	15.7	69.0	450.0
Hepatitis C	43.3	52.3	53.5	69.4	70.9	60.5	50.7	50.9	79.3	58.5	26.6	54.2	60.5	88.2	69.2	49.5	49.1	6363.0
Hepatitis C: acute viral[b]	0.8	2.4	0.0	1.9	0.0	1.1	0.0	1.1	0.0	0.0	0.5	0.0	0.3	1.0	0.0	0.9	0.1	87.5
Hepatitis C: other[b]	42.5	49.9	53.5	67.5	70.9	59.4	50.7	49.9	79.3	58.5	26.1	54.2	60.3	87.2	69.2	48.6	48.9	6275.0
Hepatitis D[b]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.1	0.0	0.1	0.0	0.2	0.0	1.2	12.5
Hepatitis E[b]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0	0.1	0.0	0.0	0.0	0.4	0.0	0.5	0.0
HIV/AIDS																		
HIV infection[b]	1.1	0.0	0.0	0.0	0.0	2.5	1.7	1.8	0.0	0.0	4.2	1.3	0.0	16.5	18.3	3.4	3.8	0.0
AIDS	0.0	0.5	0.0	0.0	0.0	0.4	1.1	1.1	1.1	1.6	0.6	0.0	2.2	2.2	0.4	0.9	1.3	0.0
Influenza	8.7	6.8	0.0	2.9	7.5	6.5	31.2	6.3	22.1	1.9	4.2	5.1	14.5	4.7	4.5	4.0	15.9	12.5
Influenza – Type A[b]	7.2	3.4	0.0	1.0	5.2	4.2	26.7	4.2	18.5	1.3	3.5	3.2	7.4	3.3	2.9	2.2	10.7	12.5
Influenza – Type B[b]	1.5	2.4	0.0	1.9	1.7	2.3	2.8	0.7	3.2	0.3	0.4	1.6	4.5	0.8	1.7	0.6	5.2	0.0
Influenza – Types A & B[b]	0.0	0.0	0.0	0.0	0.6	0.0	0.0	1.4	0.0	0.3	0.3	0.3	2.6	0.2	0.0	1.2	0.0	0.0
Influenza – Type (NOS) [b]	0.0	1.0	0.0	0.0	0.0	1.7	1.7	0.0	0.4	0.0	0.1	0.0	0.0	0.4	0.0	0.0	0.0	0.0
Legionellosis	1.1	0.0	0.0	0.0	1.7	1.7	0.0	0.4	0.0	0.6	2.0	1.1	0.9	0.4	0.5	1.2	2.7	0.0
L. longbeachae[b]	0.8	0.0	0.0	0.0	1.2	0.9	0.0	0.4	0.0	0.6	0.4	0.0	0.5	0.2	0.1	0.3	0.3	0.0
L. pneumophila[b]	0.4	0.0	0.0	0.0	0.6	0.9	0.0	0.0	0.0	0.6	1.6	1.1	0.4	0.2	0.4	0.9	2.3	0.0
Legionnaires' disease – other	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0
Leprosy	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Leptospirosis[b]	0.0	0.5	0.0	1.0	0.6	0.7	3.9	0.7	0.7	0.0	0.6	0.3	0.0	0.0	0.0	0.0	0.0	0.0
Listeriosis[b]	0.4	0.0	0.0	0.0	0.6	1.1	0.6	0.4	0.0	0.0	0.6	0.0	0.3	0.4	0.4	0.6	0.3	0.0
Lymphogranuloma venereum (LGV) [b]	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria[b]	1.5	1.5	0.0	0.0	1.2	2.5	2.8	0.7	3.9	0.6	2.4	1.1	1.4	2.6	1.4	0.6	4.2	0.0
Measles	0.4	0.0	0.0	0.0	0.6	0.2	0.0	0.4	0.4	0.6	1.1	0.3	1.4	1.8	0.5	2.8	1.3	0.0
Measles lab confirm[b]	0.4	0.0	0.0	0.0	0.6	0.2	0.0	0.4	0.4	0.6	1.1	0.3	1.1	1.2	0.5	0.6	1.3	0.0
Measles (other)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.6	0.0	2.2	0.0	0.0
Meningococcal disease	1.5	0.0	0.0	3.8	0.0	1.7	1.1	1.1	1.1	1.6	1.4	2.1	1.8	2.2	1.8	0.9	1.6	12.5
Meningococcal – Type B[b]	0.8	0.0	0.0	2.9	0.0	0.5	0.6	0.7	0.7	1.0	0.6	1.3	0.4	1.6	1.0	0.3	1.0	0.0
Meningococcal – Type C[b]	0.4	0.0	0.0	1.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.3	0.4	0.2	0.2	0.0	0.1	0.0

Table 3. *continued*

Condition	Greater Southern[e]		Greater Western[e]			Hunter/New England[e]		North Coast[e]		Northern Syd/Central Coast[e]		South Eastern Syd/Illawarra[e]		Sydney South West[e]		Sydney West[e]		Justice Health[f]
	Albury	Goulburn	Broken Hill	Dubbo	Bathurst	Newcastle	Tamworth	Pt Macquarie	Lismore	Gosford	Hornsby	Wollongong	Randwick	Camperdown	Liverpool	Penrith	Parramatta	
Meningococcal – Type W135[b]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.1	0.3	0.0	0.0	0.1	0.0	0.1	0.0
Meningococcal – Type Y[b]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal disease – Other	0.4	0.0	0.0	0.0	0.0	0.7	0.6	0.4	0.4	0.3	0.5	0.3	1.1	0.4	0.5	0.6	0.3	12.5
Mumps[b]	0.8	0.0	0.0	0.0	1.2	0.4	0.6	0.4	1.8	0.0	3.5	0.8	3.8	5.3	2.1	2.5	3.3	0.0
Pertussis	111.3	70.4	141.4	125.5	40.9	66.8	86.3	21.8	38.4	45.2	69.3	47.0	93.4	73.9	62.5	72.3	93.4	0.0
Pneumococcal disease (invasive)[b]	8.3	11.3	8.6	9.5	15.0	13.6	4.5	7.4	8.2	8.9	6.3	6.4	6.9	7.1	7.4	8.0	8.5	0.0
Psittacosis[b]	2.3	1.5	2.1	0.0	5.2	4.7	0.0	1.1	2.9	0.6	0.3	1.6	0.4	0.4	0.8	2.5	0.8	12.5
Q fever[b]	3.0	6.9	2.1	21.9	8.1	3.2	22.8	3.9	10.3	0.0	0.1	2.1	0.4	0.0	0.2	0.3	0.1	0.0
Rubella[b]	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.4	0.4	0.3	0.4	0.0	1.5	1.6	0.7	0.0	0.5	0.0
Salmonella infection[b,d]	24.1	27.4	12.9	39.0	24.8	29.7	38.4	25.6	67.9	18.3	29.6	25.8	30.4	30.6	26.7	34.8	26.4	12.5
Shigellosis[b]	0.4	0.0	2.1	1.9	0.6	0.4	0.6	1.4	3.2	0.0	0.9	0.3	2.8	2.7	0.4	0.3	0.5	0.0
Syphilis	0.4	3.9	45.0	19.0	5.2	2.3	6.7	4.2	8.2	4.7	5.2	5.6	23.2	39.8	16.6	7.4	12.5	275.0
Syphilis infectious[b,c]	0.0	0.0	0.0	0.0	1.2	0.7	0.6	0.0	1.1	0.0	1.4	0.3	12.3	12.4	0.6	0.9	1.3	0.0
Syphilis congenital	0.0	0.0	0.0	1.0	0.0	0.0	0.6	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0
Syphilis other[b]	0.4	3.9	45.0	18.1	4.0	1.6	5.6	4.2	7.1	4.7	3.7	5.3	10.9	27.4	16.0	6.5	11.1	275.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.1	0.0	0.0	0.0	0.0	0.0
Tuberculosis[b]	1.9	1.0	2.1	0.0	0.0	2.3	1.1	1.4	1.1	3.8	5.9	1.3	8.4	16.5	11.2	2.8	13.8	0.0
Typhoid[b]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.3	0.6	0.0	0.9	0.4	0.7	0.3	1.4	0.0
Verotoxigenic *Escherichia coli* infection[b]	0.0	1.5	0.0	0.0	0.0	0.4	0.6	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.1	0.0	0.3	0.0

[a] Year of onset: the earlier of patient-reported onset date, specimen date or date of notification.

[b] Laboratory-confirmed cases only.

[c] Includes Syphilis primary, Syphilis secondary, Syphilis <1 year duration and Syphilis newly acquired.

[d] From 2005, all paratyphoid recorded as salmonellosis.

[e] AHS further divided into the geographical region covered by their component Public Health Unit.

[f] Rate is based on a denominator of 8000 persons

NOS: not otherwise specified.

Table 4. Disease notifications by Area Health Service of residence (2005 AHS boundaries), NSW, 2006[a]

Condition	Greater Southern[e]		Greater Western[e]			Hunter/New England[e]		North Coast[e]		Northern Syd/Central Coast[e]		South Eastern Syd/Illawarra[e]		Sydney South West[e]		Sydney West[e]		Justice Health[f]	Total[f]
	Albury	Goulburn	Broken Hill	Dubbo	Bathurst	Newcastle	Tamworth	Pt Macquarie	Lismore	Gosford	Hornsby	Wollongong	Randwick	Camperdown	Liverpool	Penrith	Parramatta		
Anthrax	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Adverse events after immunisation	8	10	0	0	3	5	2	2	2	4	4	1	7	0	1	11	5	0	65
Arboviral infection	182	44	32	89	46	352	101	316	404	102	44	66	52	19	22	33	16	0	1920
Barmah Forest virus[b]	34	11	7	11	1	177	16	151	134	46	8	18	13	10	1	4	2	0	644
Ross River virus[b]	147	31	25	78	45	173	85	163	263	55	25	47	26	8	16	27	11	0	1225
Other[b]	1	2	0	0	0	2	0	2	7	1	11	1	13	1	5	2	3	0	51
Blood lead level ≥15 µg/dL[b]	7	5	4	60	11	38	3	1	4	4	12	11	38	19	43	12	5	0	281
Brucellosis[b]	0	0	0	0	2	0	1	0	0	1	1	0	2	2	1	0	0	0	9
Chlamydia trachomatis infection[b]	463	275	78	201	320	1475	371	349	565	502	1086	581	2172	1242	729	339	909	75	11864
Cholera[b]	0	0	0	0	0	0	0	0	0	0	0	0	0	3	0	0	0	0	3
Creutzfeldt–Jakob disease[b]	0	1	0	0	0	1	1	0	0	1	0	0	0	0	2	2	0	0	8
Cryptosporidiosis[b]	40	28	6	24	25	75	34	23	60	15	142	33	87	57	53	32	44	0	779
Gastroenteritis (institutional)	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0
Giardiasis[b]	54	38	1	33	14	157	51	30	14	75	356	73	279	134	117	88	202	0	1720
Gonorrhoea[b]	5	4	2	9	9	62	9	16	37	28	136	36	591	399	124	32	162	6	1696
Haemolytic uraemic syndrome	1	0	0	0	0	1	0	0	0	0	1	1	0	1	1	3	1	0	11
H.influenzae type b	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
Hib epiglottitis[b]	1	0	0	0	0	1	0	0	0	1	1	0	0	0	0	2	0	0	6
Hib septicaemia[b]	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0	1	1	0	4
Hib infection (NOS)[b]	0	0	0	1	1	1	0	0	1	0	1	1	1	1	2	0	1	0	11
Hepatitis A[b]	1	2	2	1	2	2	0	0	3	5	10	3	14	10	13	2	24	2	95
Hepatitis B	28	25	12	8	9	60	21	28	27	40	276	54	323	468	512	53	535	39	2543
Hepatitis B: acute viral[b]	2	1	2	0	2	7	1	1	3	1	3	4	6	7	4	2	5	3	54
Hepatitis B: other[b]	26	24	10	8	7	53	20	27	24	39	273	50	317	461	508	51	530	36	2489
Hepatitis C	115	107	25	73	123	346	91	145	223	185	215	204	493	450	581	161	377	509	4470
Hepatitis C: acute viral[b]	2	5	0	2	0	6	0	3	0	0	4	0	2	5	0	3	1	7	40
Hepatitis C: other[b]	113	102	25	71	123	340	91	142	223	185	211	204	491	445	581	158	376	502	4430
Hepatitis D[b]	0	0	0	0	0	0	0	0	1	0	1	0	1	0	2	0	9	0	15
Hepatitis E[b]	0	0	0	0	0	0	0	1	1	0	1	0	0	0	3	0	4	0	10
HIV/AIDS																			
HIV infection[b]	3	0	0	0	0	14	3	5	0	0	34	5	0	84	154	11	29	0	371
AIDS	0	1	0	0	0	2	2	3	3	5	5	0	18	11	3	3	10	0	84
Influenza	23	14	0	3	13	37	56	18	62	6	34	19	118	24	38	13	122	1	616
Influenza – Type A[b]	19	7	0	1	9	24	48	12	52	4	28	12	60	17	24	7	82	1	420
Influenza – Type B[b]	4	5	0	2	3	13	5	2	9	1	3	6	37	4	14	2	40	0	150
Influenza – Types A & B[b]	0	0	0	0	1	0	0	4	0	1	2	1	21	1	0	4	0	0	37
Influenza – Type (NOS)[b]	0	2	0	0	0	0	3	0	1	0	1	0	0	2	0	0	0	0	9
Legionellosis	3	0	0	0	0	10	0	1	0	0	3	0	0	1	1	1	2	0	
L. longbeachae[b]	2	0	0	0	2	5	0	0	0	0	13	4	4	1	3	3	18	0	22
L. pneumophila[b]	1	0	0	0	1	5	0	0	0	2	0	0	3	1	0	0	1	0	54
Legionnaires' disease – other	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Leprosy	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Leptospirosis[b]	0	0	0	1	0	4	7	2	2	0	0	1	0	0	0	0	0	0	17
Listeriosis[b]	1	0	0	0	1	6	1	1	0	0	5	0	2	2	3	2	2	0	26
Lymphogranuloma venereum (LGV)[b]	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Malaria[b]	4	3	0	0	2	14	5	2	11	2	19	4	11	13	12	2	32	0	140
Measles	1	0	0	0	0	1	0	1	1	2	9	1	11	9	4	9	10	0	60
Measles lab confirm[b]	1	0	0	0	1	1	0	1	1	2	9	1	9	6	4	2	10	0	48
Measles (other)[b]	0	0	0	0	0	0	0	0	0	0	0	0	2	3	0	7	0	0	12
Meningococcal disease	4	0	0	4	0	10	2	3	3	5	11	8	15	11	15	3	12	1	107
Meningococcal – Type B[b]	2	0	0	3	0	3	1	2	2	3	5	5	3	8	8	1	8	0	54
Meningococcal – Type C[b]	1	0	0	1	0	3	0	0	0	0	0	1	3	1	2	0	1	0	13

Continued next page

Table 4. *continued*

Condition	Greater Southern[e]		Greater Western[e]			Hunter/New England[e]		North Coast[e]		Northern Syd/Central Coast[e]		South Eastern Syd/Illawarra[e]		Sydney South West[e]		Sydney West[e]		JHS	Total[f]
	Albury	Goulburn	Broken Hill	Dubbo	Bathurst	Newcastle	Tamworth	Pt Macquarie	Lismore	Gosford	Hornsby	Wollongong	Randwick	Camperdown	Liverpool	Penrith	Parramatta		
Meningococcal – Type W135[b]	0	0	0	0	0	0	0	0	0	1	1	1	0	0	0	0	1	0	5
Meningococcal – Type Y[b]	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
Meningococcal disease – Other	1	0	0	0	0	4	1	1	1	1	4	1	9	2	4	2	2	1	34
Mumps[b]	2	0	0	0	2	2	1	1	5	0	28	3	31	27	18	8	25	0	154
Pertussis	296	144	66	132	71	382	155	62	108	143	560	177	761	377	525	235	718	0	4918
Pneumococcal disease (invasive)[b]	22	23	4	10	26	78	8	21	23	28	51	24	56	36	62	26	65	0	564
Psittacosis[b]	6	3	1	0	9	27	0	3	8	2	2	6	3	2	7	8	6	1	94
Q fever[b]	8	14	1	23	14	18	41	11	29	0	1	8	3	0	2	1	1	0	175
Rubella[b]	0	0	0	0	0	1	0	1	1	1	3	0	12	8	6	0	4	0	37
Salmonella infection[b,d]	64	56	6	41	43	170	69	73	191	58	239	97	248	156	224	113	203	1	2058
Shigellosis[b]	1	0	1	2	1	2	1	4	9	0	7	1	23	14	4	1	4	0	75
Syphilis	1	8	21	20	9	13	12	12	23	15	42	21	189	203	139	24	96	22	881
Syphilis infectious[b,c]	0	0	0	0	2	4	1	0	3	0	11	1	100	63	5	3	10	0	210
Syphilis congenital	0	0	0	1	0	0	1	0	0	0	1	0	0	0	0	0	1	0	4
Syphilis other[b]	1	8	21	19	7	9	10	12	20	15	30	20	89	140	134	21	85	22	667
Tetanus	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	2
Tuberculosis[b]	5	2	1	0	0	13	2	4	3	12	48	5	68	84	94	9	106	0	461
Typhoid[b]	0	0	0	0	0	0	0	0	1	1	5	0	7	2	6	1	11	0	35
Verotoxigenic *Escherichia coli* infection[b]	0	3	0	0	0	2	1	0	0	0	0	1	0	0	1	0	2	0	10

[a] Year of onset: the earlier of patient-reported onset date, specimen date or date of notification.

[b] Laboratory-confirmed cases only.

[c] Includes Syphilis primary, Syphilis secondary, Syphilis <1 year duration and Syphilis newly acquired.

[d] From 2005, all paratyphoid recorded as salmonellosis.

[e] AHS further divided into the geographical region covered by their component Public Health Unit.

[f] Includes cases with unknown PHU.

NOS: not otherwise specified.

Table 5.   Disease notifications by age group and sex of the case, NSW, 2006[a]

Condition	0–4 years		5–24 years		25–44 yrs		45–64 years		65+ years		Total		Total[e]
	F	M	F	M	F	M	F	M	F	M	F	M	
Anthrax	0	0	0	0	0	0	0	1	0	0	0	1	1
Adverse events after immunisation	12	17	8	5	5	0	10	5	3	0	38	27	65
Arboviral infection	2	5	111	101	371	361	374	391	97	105	955	963	1920
Barmah Forest virus[b]	1	2	54	32	141	110	131	112	30	31	357	287	644
Ross River virus[b]	1	3	54	61	223	243	231	270	65	72	574	649	1225
Other[b]	0	0	3	8	7	8	12	9	2	2	24	27	51
Blood lead level ≥15 µg/dL[b]	6	6	1	37	4	140	2	72	1	11	14	266	281
Brucellosis[b]	0	0	1	1	0	2	0	4	0	1	1	8	9
Chlamydia trachomatis infection[b]	31	22	4563	2077	2101	2552	113	366	5	14	6814	5031	11864
Cholera[b]	0	0	0	0	0	0	0	0	3	0	3	0	3
Creutzfeldt–Jakob disease[b]	0	0	0	0	0	0	2	0	2	4	4	4	8
Cryptosporidiosis[b]	144	190	98	102	139	57	20	11	12	2	413	362	779
Giardiasis[b]	193	263	138	179	298	282	148	109	63	44	840	877	1720
Gonorrhoea[b]	2	0	109	340	99	930	16	189	1	5	227	1464	1696
Haemolytic uraemic syndrome	3	2	1	3	1	0	1	0	0	0	6	5	11
H. influenzae type b	2	2	0	0	2	0	0	0	3	2	7	4	11
Hib epiglottitis[b]	0	0	0	0	0	0	0	0	0	1	0	1	1
Hib septicaemia[b]	0	2	0	0	1	0	0	0	2	1	3	3	6
Hib infection (NOS)[b]	2	0	0	0	1	0	0	0	1	0	4	0	4
Hepatitis A[b]	5	2	21	19	14	13	7	5	3	6	50	45	95
Hepatitis B	5	9	190	221	645	728	248	377	33	63	1121	1399	2543
Hepatitis B: acute viral[b]	0	3	7	7	11	17	4	4	0	0	22	31	54
Hepatitis B: other[b]	5	6	183	214	634	711	244	373	33	63	1099	1368	2489
Hepatitis C	7	10	254	313	925	1539	386	842	78	77	1650	2781	4470
Hepatitis C: acute viral[b]	0	1	5	6	8	13	2	4	0	1	15	25	40
Hepatitis C: other[b]	7	9	249	307	917	1526	384	838	78	76	1635	2756	4430
Hepatitis D[b]	0	0	1	2	3	5	1	3	0	0	5	10	15
Hepatitis E[b]	0	0	4	2	1	3	0	0	0	0	5	5	10
HIV/AIDS													
HIV infection[b]	1	1	9	23	30	214	7	80	0	4	47	322	371
AIDS	0	0	0	0	5	47	2	29	0	1	7	77	84
Influenza	67	63	64	73	65	50	67	49	61	57	324	292	616
Influenza – Type A[b]	46	46	36	46	44	35	47	43	44	33	217	203	420
Influenza – Type B[b]	18	15	23	26	13	10	11	5	12	17	77	73	150
Influenza – Types A & B[b]	1	1	5	1	6	5	7	1	5	5	24	13	37
Influenza – Type (NOS)[b]	2	1	0	0	2	0	2	0	0	2	6	3	9
Legionellosis	0	0	0	0	3	5	12	22	12	23	27	50	77
L. longbeachae[b]	0	0	0	0	1	1	6	3	3	8	10	12	22
L. pneumophila[b]	0	0	0	0	2	4	6	19	9	14	17	37	54
Legionnaires' disease – other	0	0	0	0	0	0	0	0	0	1	0	1	1
Leprosy	0	0	0	0	0	1	0	0	0	0	0	1	1
Leptospirosis[b]	0	0	1	2	2	7	0	4	0	1	3	14	17
Listeriosis[b]	0	1	0	0	3	0	2	0	7	13	12	14	26
Lymphogranuloma venereum (LGV)[b]	0	0	0	0	0	0	0	1	0	0	0	1	1
Malaria[b]	3	5	11	37	12	35	13	19	0	4	39	100	140
Measles	8	13	16	4	11	5	1	2	0	0	36	24	60
Measles lab confirm[b]	7	10	12	2	10	4	1	2	0	0	30	18	48
Measles (other)	1	3	4	2	1	1	0	0	0	0	6	6	12
Meningococcal disease	15	19	21	17	12	8	8	3	3	1	59	48	107
Meningococcal – Type B[b]	9	14	11	7	2	3	4	3	1	0	27	27	54
Meningococcal – Type C[b]	0	1	4	4	2	0	1	0	1	0	8	5	13
Meningococcal – Type W135[b]	1	0	1	0	0	0	2	0	1	0	5	0	5
Meningococcal – Type Y[b]	0	0	0	1	0	0	0	0	0	0	0	1	1
Meningococcal disease – Other	5	4	5	5	8	5	1	0	0	1	19	15	34
Mumps[b]	0	2	23	28	37	43	12	7	2	0	74	80	154
Pertussis	82	104	325	266	915	517	1246	678	431	341	3000	1906	4918
Pneumococcal disease (invasive)[b]	24	38	14	33	39	52	69	77	101	117	247	317	564
Psittacosis[b]	0	1	6	3	10	13	16	22	6	17	38	56	94
Q fever[b]	1	1	8	13	15	49	17	50	5	16	46	129	175
Rubella[b]	1	1	2	5	10	14	1	1	1	0	15	21	37
Salmonella infection[b,d]	272	278	271	308	195	203	163	151	132	78	1033	1019	2058
Shigellosis[b]	5	5	8	9	11	21	6	5	1	4	31	44	75
Syphilis	1	3	22	34	128	305	44	170	52	119	247	631	881
Syphilis infectious[b,c]	0	0	3	17	14	131	2	39	0	3	19	190	210
Syphilis congenital	1	3	0	0	0	0	0	0	0	0	1	3	4
Syphilis other[b]	0	0	19	17	114	174	42	131	52	116	227	438	667
Tetanus	0	0	0	0	0	0	0	0	1	1	1	1	2
Tuberculosis[b]	5	2	28	38	88	79	54	63	39	64	214	246	461
Typhoid[b]	4	1	4	6	6	11	1	1	0	1	15	20	35
Verotoxigenic *Escherichia coli* infection[b]	0	1	1	2	0	1	2	1	1	1	4	6	10

[a]Onset: the earlier of patient-reported onset date, specimen date or date of notification.

[b]Laboratory-confirmed cases only.

[c]Includes Syphilis primary, Syphilis secondary, Syphilis <1 year duration and Syphilis newly acquired.

[d]From 2005, all paratyphoid recorded as salmonellosis.

[e]Includes cases with unknown age and sex and people who identify as transgender.

NOS: not otherwise specified. F: female. M: male.

Table 6.   Disease notifications where the case had died at the time of public health follow-up, by year of onset of illness[a], NSW, 1991–2006

Condition	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006
Haemolytic uraemic syndrome	0	0	0	0	0	0	0	0	1	1	0	1	0	1	1	1
Hepatitis A[b]	2	1	0	0	0	0	1	2	0	0	0	1	0	0	0	0
H. influenzae type b	4	4	4	1	0	2	0	0	0	1	1	0	0	0	0	2
HIV/AIDS deaths[c]	350	337	393	432	365	280	134	76	74	87	62	67	64	58	38	36
Legionnaires' disease – *L. pneumophila*[b]	1	10	5	3	4	6	2	0	2	1	2	0	2	2	2	1
Measles	3	2	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Meningococcal disease – Type B[b]	0	0	1	1	3	0	4	2	7	6	2	8	6	4	4	4
Meningococcal disease – Type C[b]	0	0	1	1	0	2	2	10	4	4	5	10	6	1	2	1
Meningococcal disease – Type W135[b]	0	0	0	0	0	0	0	0	1	0	0	0	0	0	2	1
Meningococcal disease – Type Y[b]	0	0	0	0	0	0	0	1	0	1	0	0	0	0	1	0
Meningococcal disease – Other	3	8	9	13	4	5	1	4	2	3	0	1	2	1	0	0
Meningococcal (total)	3	8	11	15	7	7	7	17	14	14	7	19	14	6	9	6
Pneumococcal disease (invasive) [b]	0	0	0	0	0	0	0	0	0	0	6	96	75	89	61	61
Tetanus	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Tuberculosis[b]	10	26	31	25	23	16	21	25	29	40	33	39	22	28	22	27
Verotoxigenic *Escherichia coli* infections[b]	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0

[a]Year of onset: the earlier of patient reported onset date, specimen date or date of notification.

[b]Laboratory-confirmed cases only.

[c]Deaths in people with HIV may be reported as related to AIDS or where the cause is apparently unrelated to their HIV infection. HIV/AIDS deaths are reported by date of death rather than onset of illness.

Enhanced surveillance of hepatitis B infection in inner-western Sydney

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Abstract: Objectives: We developed an enhanced surveillance system for hepatitis B to improve the detection of newly acquired cases and to collect epidemiological data. **Methods**: The study was undertaken from February to June (inclusive) 2005 at the Sydney South West Area Health Service Eastern Zone Public Health Unit. A letter was sent to treating doctors on receipt of a notification, requesting additional information on cases. Cases identified by the treating doctors as newly acquired were followed up by telephone. **Results**: There were 295 notifications of hepatitis B in the period, of which three were newly acquired infections. Only one of these three cases was identified through enhanced surveillance. Information on ethnicity was obtained. **Conclusions**: This enhanced surveillance system is of limited value as an ongoing process. We recommend that it be undertaken periodically to monitor the epidemiology of the disease.

Hepatitis B is a viral infection that can cause chronic inflammation of the liver and may lead to cirrhosis and hepatocellular carcinoma.[1] In Australia in 2005, 6793 unspecified hepatitis B notifications were reported to the National Notifiable Disease Surveillance System, with 235 of these reported as being incident (or newly acquired) cases.[2] In NSW, there were 54 newly acquired cases reported.[2] Furthermore, it has been estimated that the prevalence of chronic hepatitis B infection in Australia ranges from 0.47–0.87%.[3] Hepatitis B is preventable by vaccination, and further spread can be avoided through administration of immunoglobulin prophylaxis within 14 days of exposure,[1] as well as through the provision of information about modes of transmission and infection control. In addition, new treatment modalities are proving to be effective.[4] It would therefore be beneficial for cases of newly acquired hepatitis B to be detected in a timely manner to allow effective public health action to be instituted, as well as to facilitate early referral for management.

Identifying newly acquired cases of hepatitis B can be problematic, however, as laboratories usually only test and report results for positive surface antigen (HBsAg), which indicates unspecified infection with hepatitis B, rather than IgM antibody to core antigen (HBcAb IgM), which indicates newly acquired infection. In addition, clinical details or related results such as liver function tests are very rarely provided to the Public Health Unit (PHU). Identification of newly acquired cases is therefore highly dependent on the treating doctor notifying the PHU. While medical practitioners are required under the NSW *Public Health Act 1991* to notify acute episodes of viral hepatitis, it is acknowledged that under-reporting is a significant issue.[5,6] In addition, laboratories provide limited demographic data (age, sex and postcode only), resulting in an incomplete picture of the epidemiology of hepatitis B.

A recent report of an enhanced surveillance system for hepatitis B in south-eastern Sydney stated that it was effective in increasing the detection of newly acquired cases by three-fold (from three to 10 cases) over a six-month period.[7] However, this system required a reasonably high staff workload and relied on access to supplementary investigation results and clinical details. Consequently, it was unsuitable for implementation at our centre. An alternative enhanced surveillance system for hepatitis B infection was therefore developed, with the aims of improving both the detection of newly acquired cases and the collection of epidemiological data to inform targeted public health action. This article presents the results of this enhanced surveillance and discusses the implications of its implementation.

Methods

In NSW, hepatitis B infection is notifiable by laboratories and acute viral hepatitis is notifiable by medical practitioners and hospitals under the *Public Health Act 1991*. The case definition for newly acquired hepatitis B is provided in Box 1.[8]

Enhanced surveillance was undertaken from February to June (inclusive) 2005 at the Sydney South West Area Health Service (SSWAHS) Eastern Zone PHU, which services a large, ethnically diverse population of approximately 500 000 in inner-western Sydney.[9] After receipt of

- Detection of HBsAg in a patient shown to be negative within the last 24 months, or
- Detection of HBsAg and IgM to hepatitis B core antigen, in the absence of prior evidence of HBV infection, or
- Detection of HBV by nucleic acid testing, and IgM to hepatitis B core antigen, in the absence of prior evidence of HBV infection.

Source: Notifiable Diseases Manual, NSW Department of Health 2004

a laboratory notification of HBsAg, a letter and two-page questionnaire were mailed to the medical practitioner who requested the test. Due to limited human resources, a reminder letter was not sent to treating doctors who did not respond. The letter outlined the reasons for undertaking this enhanced surveillance. The first page of the questionnaire requested demographic details of the case (gender, occupation, country of birth, language spoken at home, and Aboriginal and Torres Strait Islander status), and after providing the case definition of a newly acquired case of hepatitis B, it requested the practitioner's opinion as to whether this case of hepatitis B was newly acquired. In addition, information regarding household or sexual contacts and the use of hepatitis B vaccine for these contacts was requested, as well as asking if the medical practitioner required further information regarding hepatitis B, either for their patients or to assist with tertiary referrals.

If the case was considered newly acquired, the medical practitioner was asked to complete the second page of the questionnaire. This requested information regarding the presence and onset date of symptoms, and possible risk exposures. If further information was required for public health follow-up, it was obtained by telephoning the medical practitioner and the patient.

Data from returned questionnaires were entered into the NSW Health Notifiable Diseases Database and into a separate database created in Microsoft Excel 2000, and frequencies of selected characteristics were calculated.

In addition to the laboratory notifications of HBsAg, the PHU also received telephone notifications of acute hepatitis B directly from medical practitioners. The surveillance officers then obtained demographic and risk factor information regarding these cases from the medical practitioner and, if permission was given, directly from the patient. These cases were considered to be the 'baseline' number of notifications that would occur if the enhanced surveillance system was not in place.

The cost to the PHU of implementing this enhanced surveillance system was calculated by recording the amount of staff time taken for data entry, questionnaire and letter preparation and mail out, and telephone follow-up with treating doctors.

Results

Enhanced surveillance

There were 295 notifications of hepatitis B in the period February to July 2005 (average monthly notification rate of 9.9 per 100 000 people). Median time taken to send questionnaires to treating doctors was 11 days from receipt of the laboratory notification (range 1–36 days). Three notifications were confirmed cases of newly acquired hepatitis B. Two of these were notified directly to the PHU by the treating medical practitioner, and the remaining case was detected as a result of the enhanced surveillance. There were eight additional cases that medical practitioners indicated on the questionnaire were newly acquired, but after follow-up by telephone it was determined that these cases did not meet the case definition for a newly acquired case. Out of the 293 laboratory notifications, 204 questionnaires were returned (response rate 69.6%).

There were 104 cases (51%) that had household contacts. Of these cases, it was indicated by medical practitioners that for 67 (64.4%), hepatitis B vaccination had been instituted for their contacts.

Less than a quarter of medical practitioners requested further information regarding hepatitis B, either for the information of their patients ($n = 48$, 23.1%) or to assist in tertiary referral ($n = 43$, 21.1%).

The additional staff time that this enhanced surveillance required was estimated to be four hours per week. If a public health nurse (5th year) were employed to perform these duties, the additional cost to the PHU would be approximately $85 per week, or $4500 per year.[10] If, however, an existing public health surveillance officer were used to carry out this surveillance, this would likely be at the expense of an alternative communicable disease control activity.

Demographics of cases

The demographic characteristics of these cases are shown in Table 1 and country of birth data are shown in Figure 1. Cases were predominantly male (56%) and aged 20–40 years. The majority of cases notified to the PHU spoke a language other than English at home (66%) with 31% speaking a Chinese dialect. Cases also tended to be born overseas, primarily in China (28%) and Vietnam (21%).

In all three cases of newly acquired hepatitis B, risk exposures were both unprotected sexual contact and intravenous drug use. These three people were all born in Australia, two were male and all were aged less than 30 years.

Table 1.   Demographic characteristics of people notified with hepatitis B, Sydney South West Area Health Service (Eastern Zone), NSW, February to July 2005 (*n* = 204)

Characteristic		*n*	%
Gender	Male	114	55.9
	Female	89	43.6
	Unknown	1	0.5
Age (years)	Median	35	
	Range	7–90	
Language other than English spoken at home		134	65.7

Demographics of catchment population

In the 2001 census, 50.6% of the PHU catchment population were male. The area is culturally diverse, with almost 40% of residents born overseas. The most common overseas countries of birth were China (4.2%), the UK (3.5%), Italy (2.9%), Greece (2.6%) and Lebanon (2.3%).[9]

Discussion

This enhanced surveillance system was able to increase the detection of cases of newly acquired hepatitis B over a six-month period by one case. It also provided additional information regarding ethnicity that would otherwise not be available. In addition, it was helpful in obtaining some (albeit limited) data regarding the needs of medical practitioners with respect to information regarding hepatitis B, and the extent to which medical practitioners undertake appropriate management of contacts.

Our results differ from those of a recently reported enhanced surveillance system.[7] This may be due to several reasons. We did not have access to the results of supple-
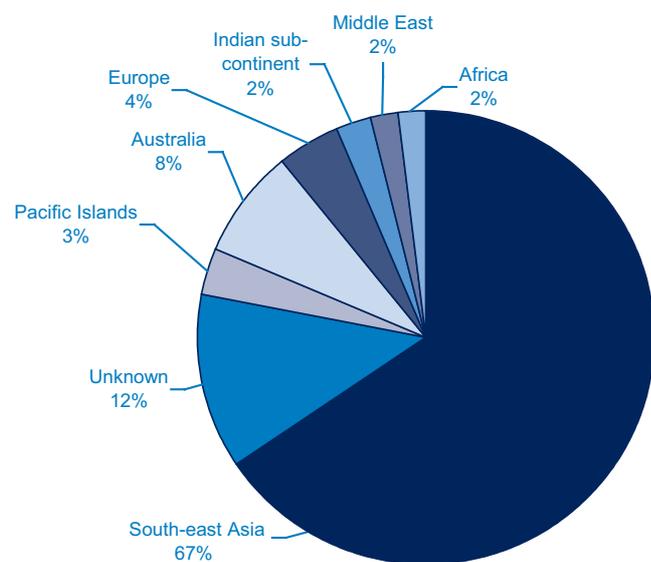
Figure 1.   Country of birth of people notified with hepatitis B infection, Sydney South West Area Health Service (Eastern Zone), NSW, February to July 2005 (*n* = 204)

mentary laboratory investigation or clinical details and were therefore unable to predict case classification before contacting treating doctors. This is unlikely to have made a difference to our results, demonstrated by the relatively large numbers of 'false positive' cases. It is possible that the treating doctors in our PHU catchment area do not under-report newly acquired cases of hepatitis B as is widely believed, and therefore enhanced surveillance for this reason is less important.

The inner-western region of Sydney has one of the highest notification rates of hepatitis B in NSW, which has been attributed to the large numbers of residents born in countries where hepatitis B is endemic.[11] This is reflected in these results, where, compared with the demographic profile of the catchment population, a disproportionately high number of people notified with hepatitis B were born overseas, particularly in South-east Asia where hepatitis B carriage rates have been estimated to be up to 20%.[12]

Vaccination of household contacts of people with hepatitis B infection is known to reduce the risk of spread.[1] Vaccination had been commenced in almost two-thirds of cases with household contacts, moderately lower than levels found in a previous study in northern Sydney (88%),[13] but greater than studies in the United Kingdom (38%)[14] and the United States (55%).[15] Had there been more time between receipt of laboratory notification and mailing out of enhanced surveillance questionnaires, this vaccination rate may have been even higher. There could be other reasons for non-vaccination, however, such as previous vaccination, contraindication to vaccine, the failure of the contact to attend for vaccination, or the attendance of the contact to another provider for vaccination. This information was not collected by this enhanced surveillance, but it would be possible to do so in the future to enable a better understanding of reasons for the non-vaccination of contacts.

Despite the good response rate (which is likely to have been higher if additional human resources were available to send reminder letters to non-responding treating doctors), only one additional case of newly acquired hepatitis B was detected using this enhanced surveillance system, which did enable public health follow-up that would otherwise not have occurred. This was at the cost, however, of additional staff time spent in following up eight other cases that turned out to be 'false positives'. In addition, demographic information including ethnicity and Aboriginality could be collected on all cases of hepatitis B, which assists in the understanding of the epidemiology of this disease, and could be used, in combination with other methods, to estimate the prevalence of hepatitis B.[3] In the current climate of competing health funds, these benefits need to be weighed against the costs of such a surveillance system (including the time required by the

treating doctors to complete the questionnaire). We would recommend that an option is to periodically undertake enhanced surveillance for a limited time, for example, six to 12 months every two to three years, to monitor the epidemiology of this disease. This would assist in filling the gap in data on the prevalence and incidence of this significant public health problem and contribute to informed public health practice.

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Prevalence of Mantoux positivity and annual risk of infection for tuberculosis in New South Wales prisoners, 1996 and 2001

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Abstract: Objectives: This study compares the prevalence of Mantoux positivity among prisoners in NSW in 1996 and 2001 and examines factors associated with *Mycobacterium tuberculosis* infection. **Design:** Cross-sectional random samples of prisoners, including a longitudinal cohort of prisoners screened in both 1996 and 2001. **Setting:** 29 correctional centres. **Participants:** 747 men and 167 women participated in the 2001 NSW Inmate Health Survey; a cohort of 104 prisoners from the 1996 and 2001 NSW Inmate Health Surveys. **Results:** The prevalence of Mantoux positivity remained stable between 1996 and 2001 (12% and 14%, $p = 0.2$), and increased among prisoners from Asian backgrounds (21% and 47%, $p = 0.02$). The annual risk of infection in the cohort among those detained continuously between 1996 and 2001 was 3.1%, and among recidivists it was 2.7% ($p = 0.6$). **Conclusion:** The risk of *M. tuberculosis* infection for Australian prisoners is assessed to be approximately four times higher than that for the community, however there is no attributable risk to the prison environment itself.

Tuberculosis is a chronic infection of *Mycobacterium tuberculosis* which typically occurs in two phases: (1) the initial infection, which often goes unnoticed, and (2) years later, progression to an active disease, which can be infec-tious. Infectious disease is of public health importance as transmission, via the air-borne route, is possible.

International experience has highlighted the role that prisons and prisoners play in community transmission of tuberculosis. Prisons may be important sentinel sites in which to monitor tuberculosis control at a community level. Several studies have highlighted the impact of tuber-culosis within correctional centres.[1,2] The high volume of prisoner movement enhances the potential for the spread of air-borne and droplet-borne illnesses within the correc-tional setting.[3,4] There are over 9000 full-time prisoners and periodic detainees in NSW, accounting for around 180 000 prisoner movements each year between correctional facili-ties, courthouses, community visits and hospitals.

Prisons are reported to be a risk for tuberculosis transmis-sion independent of the predisposing risk factors of the prison population. In New York prisons, overcrowding and poor ventilation were reported as independent risk factors.[5] However, these reports referred to a time and place of poor tuberculosis control in the community.

Given that prisons have many of the risk factors associated with tuberculosis transmission, eg, overcrowding, signifi-cant numbers of overseas-born individuals and frequent movement of prisoners, it is essential that this population be continually monitored. Ongoing surveillance will iden-tify changes in the epidemiology of tuberculosis in the NSW prison system and determine changes in the annual risk of infection for the state's prisoner population.

Australia has achieved a sustained high level of tubercu-losis control. The 2001 tuberculosis notification rate was 5.1 per 100 000 population (being 1.0 per 100 000 popula-tion for non-Aboriginal Australian born citizens). Australia had a mid-range incarceration rate of 148 pris-oners per 100 000 adult population in 2001. NSW houses around 35% of all prisoners in Australia.

The prison environment is implicated as a factor in the epi-demiology of tuberculosis, both within the prison and in the general community. There were specific concerns regarding the transmission of tuberculosis within the NSW prison system in 1996,[6] which prompted the inclusion of Mantoux screening in both the 1996 and the 2001 NSW Inmate Health Surveys.[7,8]

We examined the prevalence of Mantoux positivity in two cross-sectional surveys of prisoners conducted in NSW, and estimated the annual risk of infection from tuberculosis using a prisoner cohort.

Methods

A detailed methodology for the Inmate Health Surveys has been previously described.[9] The cross-sectional surveys involved screening prisoners from all 29 correctional centres in NSW. Participants were randomly selected according to sex, age and Aboriginal status. A random sample of around 10% full-time men and 34% full-time women prisoners across the state was taken.

Participants underwent an extensive face-to-face interview covering self-reported physical and mental health (chronic health complaints, disability, recent health complaints and symptoms and traumatic brain injury). In addition, biological samples were taken to screen for a range of infectious diseases (eg, HIV, hepatitis B and hepatitis C) and sexually transmitted infections (eg, chlamydia and gonorrhoea).

Mantoux positivity was measured as a proxy for infection with *M. tuberculosis.* A 0.1-mL solution of purified protein derivative (PPD) (CSL Vaccines, 10 IU) was administered intracutaneously and the reaction was assessed after 72 hours using the ballpoint pen method.[10] All prisoners in the samples were offered a Mantoux test.

According to the NSW Health Policy Directive,[10] Mantoux reactions were considered to be positive if the reaction was:
- ≥10 mm in persons with no previous vaccination history, nor evidence of BCG, or
- ≥15 mm in persons with a previous BCG vaccination history.

Nurses involved in Mantoux testing were recruited from community-based tuberculosis clinics, and were accredited specifically for the performance and interpretation of the Mantoux test. Inmates who were determined to have a positive Mantoux result were recommended for specialist assessment.

The prisoner cohort involved identifying all prisoners who had participated in the 1996 Inmate Health Survey and who were also in detention at the time of the survey in 2001. The Offender Management System[11] was used to locate these individuals within the NSW correctional system. The Justice Health Human Research and Ethics Committee and the Department of Corrective Services both granted ethics approval for the Inmate Health Surveys.

Assessing the rate of Mantoux positivity per annum and reporting annual risk of infection determined the incidence of Mantoux conversion. The International Union of Tuberculosis and Lung Disease Guidelines were used.[12] The chi-square test was used as appropriate. Forward stepwise logistic regression was used to determine risk factors independently associated with Mantoux positivity. Risk factors entered into the multivariate model were: sex, age, Aboriginality, region of birth, year of survey and history of BCG vaccination. All analyses were conducted using STATA 8.2.[13]

Results

Cross-sectional surveys (1996 and 2001)

A total of 789 prisoners (657 men and 132 women) were surveyed in 1996 and 914 (747 men and 167 women) in 2001. The response rate was 90% for the 1996 survey, and 85% for the 2001 survey. Mantoux tests were performed on 639 (81%) prisoners in 1996 and 803 (88%) in 2001. The median age of participants who consented to having the Mantoux test was 31 years in 1996 and 30 years in 2001 ($p = 0.04$). Aboriginal prisoners were younger than non-Aboriginal prisoners in each survey (28 years *v.* 33 years in 1996, $p < 0.001$; 27 years *v.* 32 years in 2001, $p < 0.001$).

The prevalence of Mantoux positivity remained stable between 1996 (12%) and 2001 (14%) ($p = 0.2$) (Table 1). However, there was an increase in the prevalence of Mantoux positivity among non-Aboriginal prisoners from 11% in 1996 to 16% in 2001 ($p = 0.04$) and among prisoners reporting Asian countries of birth (21% in 1996 to 46% in 2001, $p = 0.02$).

The Mantoux prevalence was similar between the two surveys in 1996 and 2001, after adjusting for sample differences (Table 2). Risk factors independently associated with a positive Mantoux test were Aboriginal status, increasing age and birth outside Australia. In 1996, four prisoners tested positive for antibodies to HIV and one of these prisoners was Mantoux positive. In 2001, one prisoner tested positive for HIV and was Mantoux positive.

No active case of tuberculosis was detected in either cross-sectional survey.

Cohort (1996–2001)

Of the 789 prisoners screened in 1996, 193 were identified as being in detention at the time of the 2001 Inmate Health Survey. Twenty-four percent (48/193) had been detained continuously between the two surveys. One hundred and eighty one (94%) prisoners agreed to be rescreened; of these, 104 (57%) received a Mantoux test regardless of their 1996 status (97 negative, seven positive in 1996). (Table 3) A total of 18 of 97 (19%) participants (17 men and one woman) were Mantoux negative in 1996 and positive when retested in 2001. The calculated annual risk of infection was 3.1%. None of the cohort was HIV antibody positive.

Table 1. Prevalence of Mantoux positivity in NSW prisoners by demographic characteristics and year of survey

| | 1996 | | 2001 | | |
	No. tested	No. Mantoux positive (%)	No. tested	No. Mantoux positive (%)	*p*-value
Total	639	76 (12)	803	115 (14)	0.2
Sex					
Male	536	68 (13)	654	94 (14)	0.4
Female	103	8 (8)	149	21 (14)	0.1
p-value		0.2		0.9	
Age					
<25 years	185	9 (5)	247	17 (7)	0.4
25–40 years	262	32 (12)	340	53 (16)	0.2
>40 years	191	35 (18)	216	45 (21)	0.5
p-value		<0.001		<0.001	
Aboriginal					
No	450	51 (11)	578	92 (16)	0.04
Yes	189	25 (13)	225	23 (10)	0.3
p-value		0.5		0.04	
Region of birth					
Australia	501	44 (9)	638	61 (10)	0.7
Oceania (other)	23	3 (13)	41	5 (12)	0.9
Asia	29	6 (21)	56	26 (46)	0.02
Europe	29	15 (52)	47	15 (32)	0.09
Other	18	4 (22)	17	7 (41)	0.2
Not reported	39	4 (10)	4	1 (25)	0.4
p-value		<0.001		<0.001	
Previous BCG vaccination					
No	589	65 (11)	736	101 (14)	0.1
Yes	50	11 (22)	67	14 (21)	0.9
p-value		0.02		0.1	

Source: NSW Inmate Health Surveys, 1996 and 2001

The median age among the Mantoux converters was 34 years (range 24 to 62 years). A similar rate of Mantoux conversion was found between Aboriginal and non-Aboriginal participants and among participants of different ages (Table 4). Of the 18 Mantoux converters, 15 (83%) reported being born in Australia, one in Europe, one in Asia and one had an unknown region of birth. The prevalence of Mantoux converters was similar among participants detained continuously (21%) and among recidivists (ie, those who had spent some time in the community between surveys) (16%, *p* = 0.6).

Discussion

This report is the first in a randomly selected prisoner cohort and, therefore, the first report of annual risk of infection for tuberculosis in a custodial environment. These data are comparable to those of a survey of police recruits (11% of cadets were reported to be Mantoux positive).[14] Importantly, the results are higher than for community-based surveys of children, but the difference in age group is important to note.[15,16]

We estimate that the annual risk of infection for tuberculosis is approximately four times that for the general community.[14,15] However, the risk relates to the population risks of incarcerated individuals, rather than the risk of infection within prison. The work of Dwyer et al. proposed this 10 years previously.[17] Our study supports this hypothesis.

No undetected cases of active tuberculosis were identified by either survey, indicating that the risk of transmission of tuberculosis infection in the NSW prison system is low. If similar conditions existed in NSW to those found in European prisons, we could reasonably have expected to detect some active cases of tuberculosis; however, this did not eventuate.

The risk factors for Mantoux positivity have remained the same between 1996 and 2001. Certain risk factors are predictors of Mantoux positivity and should be used to prioritise screening for tuberculosis infection in Australian prisons. Birth overseas was a significant risk factor. In 2001, 79% of new tuberculosis cases in Australia occurred in overseas-born persons (particularly, those from high

Table 2. Mantoux positivity in NSW prisoners according to year of survey, demographic characteristics, and adjusted odds ratios, 1996 and 2001

	No. tested	No. Mantoux positive (%)	p-value	Adjusted OR	95% CI	p-value
Total	1442	191 (13)				
Year of survey						
1996	639	76 (12)		1.0		
2001	803	115 (14)	0.2	1.2	0.9–1.7	0.3
Sex						
Male	1190	162 (14)		1.0		
Female	252	29 (12)	0.4	0.7	0.5–1.1	0.2
Age						
<25 years	432	26 (6)		1.0		
25–40 years	602	85 (14)		2.9	1.8–4.6	<0.001
>40 years	407	80 (20)	<0.001	3.8	2.3–6.2	<0.001
Aboriginal						
No	1028	143 (14)		1.0		
Yes	414	48 (12)	0.2	1.6	1.0–2.3	0.03
Region of birth						
Australia	1139	105 (9)		1.0		
Oceania (other)	64	8 (13)		2.0	0.9–4.5	0.09
Asia	85	32 (38)		8.3	4.9–14.1	<0.001
Europe	76	30 (39)		6.0	3.4–10.4	<0.001
Other	35	11 (31)		5.0	2.3–10.9	<0.001
Not reported	43	5 (12)	<0.001	–	–	–
Previous BCG vaccination						
No	1325	166 (13)		1.0		
Yes	117	25 (21)	0.007	1.0	0.6–1.8	0.9

Source: NSW Inmate Health Surveys, 1996 and 2001

prevalence countries such as Vietnam, China, the Philippines, India and Indonesia).[18] Among the Australian-born participants, 21% of new cases were reported as being Aboriginal,[18] a group who are significantly overrepresented in NSW prisons (1.4% of the general population but approximately 20% of the NSW prisoner population).[19]

This study experienced logistical problems associated with conducting Mantoux screening in prisons, with a number of prisoners unavailable for follow-up skin test readings and for chest x-rays. This inability to follow-up prisoners was primarily due to release from gaol or internal loss due

Table 3. Mantoux test results among NSW prisoners who participated in the 1996 and 2001 Inmate Health Surveys

| Mantoux test 1996 | Mantoux test 2001 | | |
	Negative	Positive	Total
Negative	79	18	97
Positive	2	5	7
Total	81	23	104

to transfers within the prison system. Some prisoners were unable to attend local chest x-ray clinics or hospitals for security reasons. These issues have been highlighted in a previous report.[3] Further research is required to assess the management of the Mantoux converters.

Mantoux conversion occurs when a person with a previously negative Mantoux test result shows a positive result, indicating that the person has become exposed to *M. tuberculosis*. By definition, it is an incident case of infection, but not an incident case of disease (a diagnosis dependent on sputum examination, x-ray and clinical signs and symptoms). Importantly, recently infected individuals are rarely infectious themselves, ie, they do not excrete organisms into the environment. We identified 10 continuously detained Mantoux converters, suggesting that infection occurred while the custodial authority had a 'duty of care' for the individual. It does not indicate at which time or place the conversion occurred.

There have been numerous reports of prison-to-community spread of tuberculosis.[17,20] We were concerned about an undetected ongoing micro-epidemic in the prison

Table 4.   Demographic characteristics of Mantoux converters among NSW prisoners who participated in the 1996 and 2001 Inmate Health Surveys

	No. participants	No. Mantoux conversion (%)	*p*-value
Total	97	18 (19)	
Sex			
Male	86	17 (20)	
Female	11	1 (9)	0.4
Age (in 2001)			
<25 years	41	7 (17)	
25–40 years	33	7 (21)	
>40 years	23	4 (17)	0.9
Aboriginal			
No	75	14 (19)	
Yes	22	4 (18)	0.9
Region of birth			
Australia	81	15 (18)	
Oceania (other)	3	0 (0)	
Asia	3	1 (33)	
Europe	3	1 (33)	
Other	4	0 (0)	
Not reported	3	1 (33)	0.7
Continuous imprisonment			
Yes	48	10 (21)	
No	49	8 (16)	0.6

system, as evidenced by the Mantoux conversions among the 1996–2001 cohort. However, we were unable to identify a point-source of infection due to the nature of the study, a population-based cross-sectional survey.

It is notable that there was no difference in annual risk of infection between recidivists and those continuously incarcerated. This situation indicates that the risk of exposure to *M. tuberculosis* is related to the antecedents of incarceration and not to the prison environment itself. This hypothesis may only be applicable to low prevalence environments for both tuberculosis and HIV. The contrary position has been proposed in a high tuberculosis prevalence environment, where distinct risks for community and prison are reported.[21]

Importantly, the higher prevalence of tuberculosis infection among prisoners was similar in the two subgroups within the cohort – those continuously incarcerated and those who had spent some of the intervening period in the community. We were unable to attribute a specific risk of tuberculosis infection to incarceration, rather the risk may be common to that subsection of the general population who are prisoners. This situation is in distinct contrast to the situation with hepatitis C and Australian prisons.[22] In low-tuberculosis-incidence countries like Australia, prisons are not a reservoir of infection but a temporary repository of people with higher exposure to tuberculosis.

Despite improvements in the delivery of health services to prisoners and better surveillance capacity, the imprecision of the diagnosis of tuberculosis infection and the substantial risk of false-positive results in a low prevalence population precludes against recommending routine Mantoux screening for NSW prisoners. The hallmark of tuberculosis control will remain rapid identification of active disease, with prompt isolation of tuberculosis suspects. Sputum microscopy remains the 'gold standard' for diagnosis as the assessment of public health risk is critical in mounting a response to tuberculosis in a low-prevalence country.

Justice Health and the Department of Corrective Services now have an effective referral process to the negative pressure cells at the Long Bay Hospital, based on the principles of early referral, infection control and secure and safe transportation. The link between the governance structure of prisoner health and the potential benefits to public health, have been proposed in the context of tuberculosis.[23] NSW prisoners are provided with health care directly from community health funds through an independent health authority, Justice Health NSW. This position is consistent with that proposed by Baroness Stern:[23]

'Restructuring the prison medical service so that it is part of the national health system has been regarded as a prerequisite of the protection of prisoners' rights.'

While the risk of tuberculosis transmission in the NSW prison system is currently considered to be low, prisoners can be sentinels for community based tuberculosis control programs. With good control programs in the community and proper governance of prisoner health services, the public health is well served. In low-prevalence situations, prisoners can be protected, and prisons may not pose added health risk to the community. This has been the Australian experience with HIV.[24] So too with tuberculosis.

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Recent substance use and high levels of psychological distress among secondary school students in New South Wales

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Abstract: In 2002, the NSW Department of Health, in conjunction with The Cancer Council NSW, carried out the NSW School Students Health Behaviours Survey. The methodology and summary descriptive results have been previously described [NSW Department of Health. The health behaviours of secondary school students in New South Wales 2002. *N S W Public Health Bull* 2004; 15(S-2): 1–86]. In the survey, 18% of NSW secondary school students reported high levels of psychological distress, 30% reported recently using alcohol, 13% reported recently using tobacco, and 9% reported recently using cannabis. With regard to the other illicit drugs, 2.0% reported recently using amphetamines, 1.0% cocaine, 1.4% hallucinogens (LSD, heroin) and 1.9% ecstasy. The survey did not, however, examine associations between substance use and high levels of psychological distress. These associations are described in this article.

In 2002, the NSW Department of Health, in conjunction with The Cancer Council NSW, carried out the NSW School Students Health Behaviours Survey.[1] Previous interstate and overseas studies have identified an association between substance use and psychological distress in adolescents.[2–5] A cross-sectional survey of Victorian secondary school students found that students reporting high levels of anxiety and depression were almost twice as likely to report using tobacco after controlling for potential confounders including year level, sex, alcohol use and parental tobacco use.[6] A United Kingdom study of adolescents aged 13–15 years found that both regular tobacco users and regular alcohol users were four times more likely to have a psychiatric disorder. They also found that regular

tobacco users who also used cannabis or cannabis and alcohol were seven and 14 times more likely to have a psychiatric disorder, respectively.[7] In a recent prospective cohort study, cannabis use was also found to moderately increase the risk of psychotic symptoms in young people but with a much stronger effect in those with evidence of predisposition for psychosis.[8]

However, interpretation and comparison of these various studies are made difficult because of the varying definitions and methods used to assess substance use and mental health problems. The basis for this association, including its causation and strength, is unclear, as is the effect of the possible interaction between the various substances and of confounders such as age and sex.

A recent review of cannabis use concluded that although an association between cannabis use and depression and anxiety existed in adolescents it had not been established whether the relationship was causal.[9] Several alternative hypotheses have been cited in the literature to explain the associations between substance use and mental health disorders other than a causative one. These include:
(1) the self-medication theory, which states that drug abuse is driven by attempts to alleviate psychological distress;
(2) a reciprocal relationship between the two where unhappiness brings about substance use, which in turn results in unhappiness, which may then result in symptoms greater than the sum of their individual effects;
(3) substance use reflects a 'willingness to contravene the law'; and
(4) substance misuse is the result of other confounding features in their lives, which cause psychological distress and substance use.[9]

The school setting provides an opportunity to further examine the association between substance use and high psychological distress across a broad range of adolescents. No recent studies on this association in Australia have been published. This article explores whether there are associations between substance use, either alone or in combination, and high psychological distress in NSW secondary school students aged 12–17 years. It considers the strength of any such associations, and discusses the impli-

cations of any associations for ongoing monitoring, and policy and program development.

Methods

Measures

High psychological distress was based on the student reporting feelings of depression, anxiety, stress, or being in trouble about their behaviour in the last six months, using three questions from the Western Australian Child Health Survey (WACHS),[10] which were based on items adapted from the Achenbach Child Behaviour Checklists and the Mental Health Inventory.[11–13] Those who had experienced a problem were asked 'how bad' it was for them at the time. A student who responded 'almost more than I can take' to any of the three problems was considered to have experienced high psychological distress.

Recent substance use included alcohol in the last week, tobacco smoking in the last week, cannabis in the last four weeks, and 'other illicit drugs' (including amphetamines, cocaine, LSD, heroin, ecstasy only) in the last four weeks.

Socioeconomic status was determined by allocating students to quintiles based on postcode, ranked from least to most disadvantaged, using the Index of Relative Socio-Economic Disadvantage.[14]

Age was dichotomised into two groups (12–15 years and 16–17 years). These age groups correspond approximately to junior and senior secondary school years.

Source of data

The source of data was the NSW School Students Health Behaviours Survey 2002 (HOIST).[15] The survey instrument was a self-administered questionnaire, which had a core questionnaire and two supplementary questionnaires. The recent substance use questions were included in the core questionnaire and the psychological distress questions were included in one of the supplementary questionnaires. This gave a sample of 3506 students who had completed both the recent substance use and psychological distress questions.

Data analysis

The data were analysed using SAS version 8.02.[16] The SURVEYMEANS procedure was used to calculate the estimated prevalence and 95% confidence intervals for all students and for students by age and sex. Cox's proportional hazard regression model, with time fixed at 1, was used to calculate estimated prevalence rate ratios and their 95% confidence intervals separately for each subgroup using SUDAAN where there were sufficient clusters within the strata and to adjust for socioeconomic status.[17]

The analysis was weighted to adjust for differences between the survey sample and the target population of secondary school students caused by stratification and clustering in the survey design and non-response. The data were analysed, stratifying by school sector (government, Catholic and independent) and level (junior secondary, up to year 10, and senior secondary, years 11 and 12), and clustering by the individual school from which the student came.

Results

Recent substance use

Almost 36% (35.6%) of all students reported using substances recently. Twenty-three percent reported using only one substance (17.4% alcohol, 3.4% tobacco, 1.2% cannabis and 0.7% other illicit drugs) (Table 1).

A significantly higher proportion of students aged 16–17 years used any substances recently (49.1%) compared with 13–15 year olds (30.4%). This age difference was consistent for any tobacco use (24.7% versus 9.6%), drinking any alcohol (38.3% versus 25.3%), and using two or more substances (17.9% versus 7.1%).

There was no significant difference seen in the recent use of any substances according to sex except for a higher proportion of males (11.2%) using any cannabis compared with females (7.4%).

High psychological distress

High psychological distress was reported by 17.5% of secondary school students in the 2002 survey. A significantly higher proportion of females (21.5%; 95% CI: 19.0–24.0%) had high psychological distress compared with males (13.5%; 95% CI: 11.3–15.6%). This difference was present in both substance users and non-users. There was no difference in high psychological distress between the 12–15 years and 16–17 years age groups.

Recent substance use and high psychological distress

Of all students, 8.4% (95% CI: 7.1–9.7%) had both recently used substances and experienced high psychological distress, 27.1% (95% CI: 24.8–29.5%) of all students had recently used substances and not suffered from high psychological distress, while 8.9% (95% CI: 7.1–9.7%) of all students had high psychological distress but had not recently used substances. Just over half of students (55.4%; 95% CI: 52.7–58.1%) had neither recently used substances nor suffered from high psychological distress.

Almost one quarter (23.7%) of students who recently used substances had experienced psychological distress, compared with 13.8% among students who had not recently used substances. Of students with high psychological distress almost half (48.2%; 95% CI: 42.2–54.2%) had used substances recently compared with 33.0% (95% CI: 30.0–36.0%) of students without high psychological distress.

As the proportion of students with high psychological dis-

Table 1.   Recent substance use for all students and by age and sex, NSW, 2002

Substance use pattern	Males % (95% CI)	Females % (95% CI)	12–15 years % (95% CI)	16–17 years % (95% CI)	All students % (95% CI)
Any substance	34.9(31.0–38.8)	36.2(33.0–44.0)	30.4(27.5–33.2)	49.1(43.6–54.5)	35.6(32.7–38.4)
Alcohol only	18.5 (16.3–20.8)	16.3 (14.0–18.5)	17.0(15.0–18.1)	19.6(16.1–23.2)	17.4 (15.8–19.0)
Any alcohol	29.8 (27.1–32.5)	28.0 (25.2–30.8)	25.3(22.3–27.8)	38.3(32.3–43.8)	28.9 (26.9–30.8)
Tobacco only	2.2 (1.3–3.1)	4.7 (3.0–6.3)	2.4 (1.6–3.1)	6.3 (3.5–9.0)	3.4 (2.5–4.4)
Any tobacco	12.2 (10.1–14.3)	15.4 (12.9–17.8)	9.6(7.9–11.3)	24.7(19.5–29.8)	13.8 (12.2–15.4)
Cannabis only	1.6 (0.8–2.4)	0.9 (0.4–1.4)	1.0(0.6–1.5)	1.8(0.2–3.4)	1.2 (0.8–1.7)
Any cannabis	11.2 (9.2–13.2)	7.4 (5.8–9.0)	7.8(6.6–9.2)	13.2(8.5–17.7)	9.3 (8.0–10.6
Illicit drugs only	0.7 (0.3–1.1)	0.7 (0.2–1.2)	0.9(0.5–1.3)	0.3 (0–0.7)	0.7 (0.4–1.0)
Any other illicits	4.6 (3.4–5.9)	3.1 (1.9–4.2)	3.3(2.5–4.1)	5.3(3.1–7.4)	3.9 (3.0–4.7)
Two substances	4.7(3.6–5.8)	3.4(2.2–4.5)	3.9(2.9–4.8)	4.6(2.4–6.8)	4.1(3.1–5.0)
Three substances	5.1(3.6–6.6)	2.7(1.7–3.7)	2.7(2.0–3.5)	6.9(4.9–9.0)	3.9(3.1–4.7)
All drugs (alcohol, tobacco, illicit drugs and cannabis)	1.0 (0.5–1.5)	1.2 (0.4–2.0)	0.8(0.5–1.2)	1.9(0.1–3.6)	1.1 (0.6–1.6)
More than one substance	9.9(8.2–11.6)	10.3 (8.0–12.5)	7.1 (5.7–8.4)	17.9(13.5–22.3)	10.1(8.4–11.8)
No substances	63.1 (60.2–65.9)	64.4 (61.4–67.4)	68.8(66.0–72.0)	50.7(45.3–56.0)	63.7 (61.7–65.8)

Note: Recent substance use included alcohol in the last week, tobacco smoking in the last week, cannabis in the last four weeks, and 'other illicit drugs' (amphetamines, cocaine, LSD, heroin, ecstasy) in the last four weeks.
Data source: 2002 New South Wales School Students Health Behaviours Survey (HOIST, Health Outcomes Information and Statistics Toolkit).
Analysis: NSW Health Survey Program, Centre for Epidemiology and Research, NSW Department of Health.
CI, confidence interval.

tress who had only used one drug recently (alcohol only, 20%; tobacco only, 21.7%; cannabis only, 22.7%; other illicit drugs only, 32%) was not significantly different from students with any recent use of that substance whether alone or in combination with other substances (any alcohol, 24.1%; any tobacco, 28.2%; any cannabis,

27.5%; any other illicit drugs, 33.6%), comparisons of high psychological stress in users and non-users was completed using 'any' recent use of a substance (Table 2).

Overall, the prevalence of high psychological distress was higher in recent substance users (23.7%) compared with

Table 2.   High psychological distress by recent substance use for all students and by age and sex, NSW, 2002

Substance	Males % (95% CI)	Females % (95% CI)	12–15 years % (95% CI)	16–17 years % (95% CI)	All students % (95% CI)
Any substance	17.3 (13.2–21.4)	30.3 (26.4–34.2)	22.7 (19.2–26.1)	25.3 (19.2–31.2)	23.7 (20.6–26.7)
Alcohol only	15.3 (10.3–20.3)	25.5 (19.0–31.9)	19.7(15.3–24.1)	20.8(14.5–27.0)	20.0 (16.0–24.1)
Any alcohol	17.7 (13.7–21.6)	31.0 (26.8–35.2)	23.5 (19.7–27.3)	25.3 (20.8–29.8)	24.1 (21.2–27.1)
Tobacco only	19.9 (1.9–37.9	22.5 (8.2–36.8)	12.9(3.3–22.4)	30.3(7.0–53.6)	21.7 (10.4–32.9)
Any tobacco	17.7 (13.7–21.6)	31.0 (26.8–35.2)	27.4 (21.6–33.2)	29.0 (20.2–37.8)	28.2 (23.1–33.3)
Cannabis only	16.0 (0.0–41.3	35.0 (6.0–63.9)	18.4(0.8–36.0)	29.1 (0–84.4)	22.7 (3.1–42.4)
Any cannabis	17.8 (10.2–25.5)	42.1 (32.9–51.3)	28.2 (20.6–35.8)	26.4 (14.7–38.1)	27.5 (20.9–34.1)
Other illicits only	17.2 (0.0–42.2)	47.2 (10.4–83.9)	27.4(4.2–50.6)	32.4(0–74.0)	32.0 (9.9–54.1)
Any other illicit	29.2 (16.7–41.8)	40.3 (24.9–55.8)	34.1 (22.8–45.3)	32.9 (13.4–52.5)	33.6 (23.4–43.9)
Two substances	14.2(6.8–21.6)	37.1(22.7–51.4)	26.0(16.9–35.0)	18.7(4.3–33.2)	23.7(16.1–31.3)
Three substances	24.2(11.0–37.4)	33.2(18.9–47.4)	30.6(20.0–41.3)	24.0(8.2–40.0)	27.3(17.5–37.5)
All four substances	32.3(12.7–52.0)	65.3(46.8–83.8)	50.2(29.0–71.3)	50.7(17.0–84.4)	50.4(32.0–68.8)
More than one substance	23.8 (15.8–31.7)	38.1 (31.1–45.1)	33.3 (25.3–41.4)	28.7(21.5–35.9)	31.0 (26.0–36.2)
No substances	11.2(9.0–13.4)	16.4 (13.4–19.5)	13.2 (10.9–15.5)	13.8 (11.3–20.7)	13.8(12.0–15.7)

Note: Recent substance use included alcohol in the last week, tobacco smoking in the last week, cannabis in the last four weeks, and 'other illicit drugs' (amphetamines, cocaine, LSD, heroin, ecstasy) in the last four weeks.
Data source: 2002 New South Wales School Students Health Behaviours Survey (HOIST, Health Outcomes Information and Statistics Toolkit).
Analysis: NSW Health Survey Program, Centre for Epidemiology and Research, NSW Department of Health.
CI, confidence interval.

Table 3. Prevalence rate ratios for high psychological distress by 'any' recent substance use for all students and by age and sex, NSW, 2002

Substance	Males PRR (95% CI)	Females PRR (95% CI)	12–15 years PRR (95% CI)	16–17 years PRR 95% CI	All students PRR (95% CI)
Any alcohol	1.5 (1.2–2.0)	1.7(1.4–2.2)	1.7 (1.4–2.1)	1.4 (1.1–1.7)	1.6 (1.4–1.9)
Any other illicits	2.3 (1.5–3.6)	1.9(1.3–2.8)	2.2 (1.6–3.0)	1.6 (1.01–2.8)	2.0(1.5–2.7)
Any cannabis	1.4 (0.9–2.2)	2.1(1.7–2.7)	1.9(1.4–2.5)	1.3(0.9–2.1)	1.7(1.3–2.2)
Any tobacco	1.5 (1.2–2.0)	1.7(1.4–2.2)	1.8 (1.5–2.3)	1.6 (1.2–2.3)	1.8 (1.5–2.2)
Any substance	1.5 (1.2–2.0)	1.8(1.4–2.3)	1.7(1.4–2.1)	1.5(1.01–2.3)	1.7(1.4–2.1)
Any substance adjusted for SES	1.6(1.2–2.1)	1.8 (1.4–2.3)	1.7(1.4–2.1)	1.6 (1.1–2.3)	1.7 (1.4–2.1)
More than one substance	1.7 (1.2–2.4)	2.1(1.7–2.5)	2.1 (1.6–2.7)	1.5 (1.3–1.9)	1.9 (1.6–2.2)

Note: Recent substance use included alcohol in the last week, tobacco smoking in the last week, cannabis in the last four weeks, and 'other illicit drugs' (amphetamines, cocaine, LSD, heroin, ecstasy) in the last four weeks.
Data source: 2002 New South Wales School Students Health Behaviours Survey (HOIST, Health Outcomes Information and Statistics Toolkit).
Analysis: NSW Health Survey Program, Centre for Epidemiology and Research, NSW Department of Health.
PPR, prevalence rate ratio. CI, confidence interval. SES, socioeconomic status.

non-users (14.0%) (PRR 1.7; 95% CI: 1.4–2.1). This was consistent for alcohol (PRR 1.6; 95% CI: 1.4–1.9), tobacco (PRR 1.8; 95% CI: 1.5–2.2), cannabis (PRR 1.7; 95% CI: 1.3–2.2) other illicit drugs (PRR 2.0; 95% CI: 1.5–2.7) and two or more drugs (PRR 1.9; 95% CI: 1.6–2.2) (Table 3).

The association between high psychological distress and recent substance users of any of the four categories of substances was consistent for all students, younger (junior secondary), older (senior secondary), male and female students. The association between high psychological distress and recent use in all students, younger (junior secondary), older (senior secondary), male and female students was also consistent for use of any alcohol, use of any tobacco, use of any other illicit drugs and use of more than one substance. However, there was no association between high psychological distress and cannabis use in males or in students over 16 years. We were unable to examine this association by age and sex due to inadequate numbers of clusters within strata. The association with 'any' recent substance use was not affected when adjusted for socioeconomic status (Table 3).

Discussion

Recent substance use is associated with high psychological distress in NSW secondary school students for all substances. Just over 8% of NSW secondary school students were found to have both engaged in recent substance use and experienced high levels of psychological distress. This subgroup of students appears to constitute a potentially vulnerable minority of students in NSW schools that is at high risk of acute and long-term psychological and health problems.

Students who reported recent substance use were more likely to have high psychological distress than those who did not for all students, both sexes, and most age groups.

The results are consistent with a large number of studies.[2–9] The strength of the association with high psychological distress found in this study in a school setting was, however, less than that reported for recent substance use in a community sample of teenagers aged 13–15 years with a psychiatric diagnosis based on structured interview.[7] In contrast, the strengths of the associations between high psychological distress and recent substance use are similar to those found for adults and adolescents in Australia and the United States (odds ratio of about 1.5–2.0).[18–24]

Although the substances studied have varying pharmacological effects, the strength of association found between high psychological distress and recent substance use did not vary significantly between substances. This suggests a non-pharmacological association and that the effect of recent substance use in this association represents a general type of behaviour rather than one related to the specific properties of a particular drug. Although no dose response curve was able to be determined, taking more than one drug did not make high psychological distress more likely.

As the NSW School Students Health Behaviours Survey is repeated every three years, it provides the opportunity to monitor the effectiveness of school programs by using as indicators the prevalence of recent substance use, high psychological distress, and both high psychological distress and recent substance use among students.

Conclusion

Recent substance use is associated with high psychological distress in NSW secondary school students for all substances. A significant minority of students appear to be at high risk. The lack of variation in the association between substances suggests a non-pharmacological association.

Acknowledgements

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A discussion of the potential benefits to injury surveillance through inclusion of date of injury in hospitalisation data in New South Wales and Australia

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Abstract: Most hospitalised injury cases have sudden onset at a specific time and date, due to a well-defined external cause (eg, a road crash). Date of injury is not presently recorded in routine hospital separations data in NSW or Australia, though it is in New Zealand. Benefits of adding date of injury to the Inpatient Statistics Collection would include: more accurate estimation of the population incidence of serious injury; better assessment of the health system utilisation and costs attributable to injurious events; and better linkage of hospital data with other data relevant to injury measurement and control (eg, road crash data).

Injury has been identified as a health priority area in Australia, and is the subject of three national plans endorsed in July 2005 by Australian health ministers.[1–3] Injuries leading to hospitalisation include the most serious non-fatal injuries, and incur substantial costs both to the health system and society. In 2001–02, injuries accounted for about 7% of all hospitalisations in Australia.[4] High-quality information about the incidence of injury leading to hospitalisation, and trends in incidence, is needed, for example, to assess the burden of injury, determine priorities for injury prevention, guide and evaluate injury prevention and assist service planning.

However, in Australia, accurate estimates of the incidence of injuries leading to hospitalisation are not readily available from routinely collected data because of multiple counting of the same injuries in estimates of the incidence of injury.

In Australia, reports on injury indicators have indicated the potential value of including information on the date of injury in routinely collected hospitalisation data.[5,6] In this paper we discuss the potential benefits of adding such a variable to hospitalisation data to improve surveillance of serious injury in Australia.

Identification of incident cases of injuries leading to hospitalisation

The Inpatient Statistics Collection (ISC) covers all inpatient separations from public and private hospitals in NSW. In NSW, all hospitals are required to submit details to the NSW Department of Health about every inpatient episode of care which ends with the discharge, transfer or death of the patient, or by the patient becoming a different type of patient, requiring transfer within the same hospital. All states in Australia maintain similar collections, which suffer from the problems we describe below.

When estimating incidence of injury in a population, each new case of injury should be counted only once. However, the number of injury-related 'separations' from hospital is not equivalent to the number of incident injury cases that result in hospitalisation, because some cases of injury result in more than one episode of in-hospital care, each of which ends with a 'separation' event. Hence, patients who have been readmitted or transferred from another hospital for treatment of the same injury are recorded more than once. This is particularly an issue for patients with serious injuries who are likely to have a series of readmissions for ongoing treatment or rehabilitation, for example, patients with extensive burns who require a number of skin grafts.[7,8] In New Zealand, which to our knowledge is the only jurisdiction that captures date of injury information in hospitalisation data, it has been estimated that about 9% of all inpatient episodes that were assigned an external cause code were readmissions,[7] but accurate estimates are not available for Australia. Furthermore, the rate of re-admission in New Zealand is not uniform across mechanisms of injury.[7] In addition, patterns of coding of the ways in which episodes in hospital end (for example, as a discharge, transfer or type-change separation) may not be constant over time in Australian hospitalisation data, or between geographical areas.[6] This further complicates

assessment of trends and differences in population incidence of cases.

The ISC does not currently contain a unique patient identifier. However, probabilistic record linkage (in this case an 'internal' linkage of the ISC to itself) can be used to identify the same patient across different episodes of care. However, reliance on the use of a unique patient identifier (or an approximate identifier obtained through record linkage) to identify repeat admissions has some drawbacks.[8] For example, a presumed repeat admission with the same diagnosis and external cause codes as the first admission may in fact be an admission for a new case of injury, such as a hip fracture on the other side of the injured person's body resulting from a fall, or a second episode of intentional self-poisoning. Probabilistic data linkage is limited as it relies heavily on ICD 10-AM codes in the principal diagnosis field to distinguish between 'multiple admissions' for the same injury event and 'new admission(s)' of the same person for a different injury event.

In the absence of a unique personal identifier and date of injury, some studies[9,10] have assumed that each discharge refers to a single episode of injury, thereby resulting in an overestimation of incidence rates. Others have used one or other of two variables, 'mode of separation' and 'mode of admission', which are available in Australian hospitalisation data, to identify and omit groups of records likely to be for second or subsequent inpatient episodes concerning the same injury.[11] Excluding such records assumes that the subsequent separation will be recorded and also meet the study definition of injury (eg, requiring a principal diagnosis of injury). Also, these methods cannot account for readmissions following discharge home.

If used with a unique patient identifier or record linkage, a 'date of injury' field would simplify the identification of incident cases of injury-related hospitalisations.[12] Even when a unique patient identifier is not available, use of probabilistic record linkage and date of injury will enable the period between the date of injury occurrence and admission to be determined. This would provide a useful indication of whether a particular hospital admission is likely to have been the first episode in hospital due to that injury incident.

Identification of incident injury events occurring in hospitals

A 'date of injury' item would also allow a distinction to be made between injuries that occur in the community and lead to initial admission and injuries that occur during the course of an episode in hospital (for example, a fall from a hospital bed). These cases are of special importance to health care services. Accurate data on these cases are vital

to inform strategies designed to prevent injury in health care institutions, particularly hospitals.

Currently, the only indication of these cases is provided by the ICD-10-AM place of occurrence codes,[13] which are not specific enough to provide an accurate picture of injuries that occur after admission to hospital. Place of occurrence codes are not able to distinguish between injury sustained in a hospital or in an outpatient clinic, nor between injury sustained in hospital by an admitted patient or a visitor. Although a 'Diagnosis onset type' variable (item 000773), designed to identify postadmission conditions, is included in the National Health Data Dictionary,[14] it is not recorded in the ISC in NSW.

Better record linkage of hospital data to other injury-related datasets

A date of injury item would also be beneficial to any record linkage of hospital separations data to other sources that contain information on injury, such as road crash data collected by police and workers' compensation data. Such linkages of datasets are important for providing a comprehensive picture of the circumstances and causes of injury, and are useful for providing a better understanding of the outcomes of various injuries. For linkage of road crash data with hospital discharge data, for example, date of injury in the hospital discharge data would assist assessment of whether matched records are, in fact, true matches.

Latency between injury and first admission

In Australia, it is likely that admission to hospital for an injury takes places soon after the occurrence of the injury. However, this is not always the case, and those cases in which first admission is delayed may warrant special attention. This is because late admission might mark instances of injuries not usually requiring admission that have resulted in complications, or injuries occurring in circumstances (geographical or social) leading to late presentation to hospital. Inclusion of a 'date of injury' variable would allow direct calculation of the latency between injury and first admission.

Conclusion

Prevention needs to be guided by a clear understanding of the burden of, and causal factors associated with, injury. Information about the date of injury is critical for linking these to relevant exposures, most of which are acute in nature. The availability of a 'date of injury' item in the New Zealand hospital discharge data has allowed accurate identification of first admissions for injuries, injuries occurring in health care institutions and improved injury surveillance.[7,12] The introduction of such a variable to NSW and Australian hospitalisation data would result in similar outcomes and enhance the value of the data used to support injury prevention and control in NSW and Australia.

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A review of the efficacy of human Q fever vaccine registered in Australia

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Abstract: Background: Q fever remains an important occupational zoonosis in rural Australia. Although Q fever vaccine is recommended in high-risk occupational groups, its availability has been limited in recent years. **Method**: A literature review of the efficacy of the human Q fever vaccine registered in Australia was conducted. **Results**: Seven relevant vaccine efficacy studies were identified but no large double-blind, randomised, placebo-controlled studies have been conducted. Vaccine efficacy has ranged from 83–100% but limitations of study designs hamper a precise estimate of vaccine efficacy. **Conclusion**: Despite the shortcomings of efficacy studies, the Q fever vaccine available in Australia has considerable protective benefit in established high-risk environments, particularly of an occupational nature.

Background
Disease burden
Q fever is a zoonosis caused by *Coxiella burnetii*. It has been identified in a wide range of wild and domestic animal hosts including arthropods, birds, rodents, marsupials and livestock, but the commonest reservoirs are cattle, sheep and goats. *C. burnetii* can withstand harsh environmental conditions including desiccation, and are shed in excreta, milk and, particularly, birth products of infected animals.[1]

Humans become infected primarily by inhaling aerosols contaminated by *C. burnetii*. Established higher risk occupations include abattoir and farm workers, and veterinarians. Indirect exposures through a contaminated environment or ingestion of contaminated unpasteurised milk have also been recognised but their contribution is not well quantified.[1] A Victorian study estimated that the average annual risk of Q fever among abattoir workers to be 62.6/1000 over the initial 10 years of employment.[2] Unfortunately, there is currently only a limited understanding of the stratification of risk by nature of exposure, the possible variable pathogenicity of different *C. burnetii* strains, and the disease modulating effect of human immune responses.

Many infected people are asymptomatic or only experience a self-limiting febrile illness. Although Q fever infections generally respond to antibiotic treatment, they may result in significant morbidity, including pneumonia, hepatitis, chronic endocarditis and post-Q fever fatigue syndrome, and occasionally death. Recrudescence may occur, especially in pregnant and immunocompromised patients.

Australia reported between 465 and 757 cases annually between 2000 and 2004. This is likely to be an underestimate because of difficulties in diagnosing Q fever and the current passive disease surveillance system. Data from the Notifiable Diseases Database in NSW, which reported 47% of all Australian Q fever cases in 2004, indicated that many notified cases still occurred among known high-risk groups. In 2004 and 2005 (to 22 November), 41% of cases with an identified occupation were farm workers and 13% were abattoir workers, meat workers, livestock handlers or veterinarians.

Disease control
A national vaccination program against Q fever that initially targeted abattoir workers, and subsequently also shearers and people employed on sheep and cattle farms, was conducted in Australia between 2001 and 2004. The program was evaluated by the National Centre for Immunisation Research and Surveillance for the Australian Government Department of Health and Ageing, but the resulting report has not yet been published.

Although environmental containment measures, including environmental cleaning and use of personal protective equipment, are often recommended as adjuncts to vaccination for Q fever control, no specific studies of the effectiveness of these measures have been conducted.

Human Q fever vaccines
Three types of vaccine have been proposed for providing human protection against Q fever: the attenuated live vaccine (produced and trialled in Russia but subsequently

abandoned because of concern about its safety); chloroform–methanol residue extracted vaccine or other extracted vaccines (trialled in animals but not humans); and the whole-cell formalin-inactivated vaccine, which is considered acceptably safe for humans.[3] Australia is the only developed country that manufactures this latter Q fever vaccine on a commercial basis and it has been available in Australia since 1989. Ongoing Q fever vaccine availability in Australia became uncertain during late 2005.

Methods

A search of the Medline and Embase databases was conducted to identify all published English language articles on the efficacy of the formalin-inactivated Henzerling strain phase I vaccine in humans, using subject headings and keywords: Q fever, *Coxiella burnetii*, Henzerling, 'Q-vax', vaccine, vaccination and immunisation (and corresponding variants of these keywords). Original reports referenced in articles were also accessed.

Results

Seven vaccine studies on the efficacy of the formalin-inactivated Henzerling strain phase I vaccine in humans were identified in the literature (Table 1).

One article reported two challenge studies that have been conducted on human volunteers.[3] The study, performed in the late 1950s, involved 24 subjects and tested an earlier formulation with a three-dose schedule. The reported study conducted between 1966 and 1968 pooled results from two studies conducted in parallel and involved 38 subjects. Both found vaccine efficacy to be 92%, but with broad confidence limits due to the small number of subjects.

A limited, randomised, blinded, placebo-controlled trial of the Q fever vaccine (using influenza vaccine as a placebo-control) that utilised a sequential analysis design was conducted in the late 1980s in three Queensland abattoirs with different characteristics.[4] This study was terminated after 15 months when one-sided 95% statistical significance was reached between the two study arms, with seven Q fever cases reported in the placebo-control group with no cases in the trial group. The protective efficacy of this vaccine was not estimated in this study. The method of randomisation was not clearly described, nor the intention-to-treat principle applied.

A retrospective cohort analysis of a 1998 Q fever outbreak investigation in a southern NSW abattoir during the second phase of a local Q fever vaccination program provided some useful efficacy information.[5] Nineteen staff members were vaccinated during the first phase, at least six weeks before the onset of the first outbreak case. No exposure differences were apparent between these 19 people and the 68 susceptible people who were eligible for vaccination during the second phase. Since no Q fever cases occurred in the 19 vaccinated individuals while 37 cases occurred in the 68 unvaccinated individuals, vaccine efficacy was calculated at 100%, although the lower confidence limit cannot be accurately estimated because of absence of cases in the vaccinated individuals.

Three open trials (non-randomised studies without control groups) provided additional insight into the efficacy of the available Q fever vaccine.[6–8] Two Q fever cases were diagnosed among 2553 vaccinated employees from three different abattoirs in South Australia between 1985 and 1990, while 55 cases were reported among 1365 unvaccinated employees during this period, indicating a vaccine efficacy of 98% with a 95% lower confidence limit of 92%.[6] Results may have been affected by volunteer selection bias, uncertain risk exposure estimates and inadequate active Q fever case ascertainment.

An earlier open trial in three South Australian abattoirs reported four Q fever cases amongst 924 vaccinated workers and 34 cases amongst 1349 unvaccinated workers between June 1981 and January 1983 (Relative risk of vaccinated (RR) = 0.17, 95% CI 0.06–0.48, $p = 0.00027$).[7] This study also suffered limitations, as the unvaccinated group included employees who were not enrolled and enrolled volunteers who were not vaccinated, either due to the presence of immune markers or other unstated reasons. The vaccinated group also included a small number of volunteers who had detectable immune markers before vaccination. A greater proportion of unvaccinated workers were deployed in higher risk areas.

A follow-up study of Q fever cases between mid-1981 and 1988 among workers from five different abattoirs in South Australia[8] included subjects from the study mentioned above.[7] Some subjects in this study were also included in the study described earlier.[6] Vaccine efficacy was reported as 100%, with eight Q fever cases among 3532 vaccinated subjects all attributed to exposure before vaccine-induced immunity would have developed. Among the subgroup considered at high risk of exposure, three of 2715 vaccinated subjects developed disease, whereas 52 of 2012 unvaccinated subjects developed disease, a protective efficacy estimate of 96% (95% CI 86–99%, $p < 0.0001$). Analysis of a subgroup of workers from a single abattoir, without immune markers of the disease before contemplating vaccination, revealed two cases of Q fever among 690 vaccinated individuals and seven cases among 61 unvaccinated subjects, an efficacy estimate of 97% (95% CI 88–99%, $p < 0.0001$).

The only economic study undertaken in Australia on Q fever vaccine predated the completion of the national

Table 1. Summary of efficacy studies of the formalin-inactivated Henzerling strain phase I vaccine for human protection against Q fever

Study reference	Time/location	Study type	Sample and design	Results	Vaccine efficacy (95% CI)	Some limitations
Ormsbee et al.[3]	Late 1950s, USA	Human experimental challenge study – controlled clinical trial	a) 16 volunteers vaccinated with three doses of the precursor 'Smadel' vaccine formulation; subsequently challenged with C. burnetii b) 64 volunteers vaccinated (30 µg), then challenged with C. burnetii 5–10 months later	a) Vaccinated Yes: Q fever disease Yes 1, No 15; No: Yes 6, No 2 b) Vaccinated Yes: Yes 2, No 30; No: Yes 5, No 1	92% (42–99%) 92% (70–98%)	This earlier study was performed with the 3-dose schedule of the Smadel vaccine with 120 µg per dose – different from the current formulation. Number of subjects studied was small. Methods were not reported in detail. Data pooled from two separate studies for study B reported
Shapiro et al.[4]	Late 1980s, three abattoirs of different characteristics in Queensland	Randomised, single-blind, placebo-controlled study	Sequential analysis – terminated after 15 months when one-sided 95% statistical significance was reached	Vaccine more efficacious than placebo in protecting subjects against clinical Q fever	Not estimated	Randomisation method was not clearly reported. Intention-to-treat principle was not applied
Gilroy et al.[5]	1998, an abattoir in Southern NSW	Retrospective cohort study	Part of a Q fever outbreak investigation. Compared attack rates between unvaccinated and vaccinated staff	Vaccinated Yes: Q fever disease Yes 0, No 19; No: Yes 37, No 31	100%	Exposure risks of subjects were not able to be quantified or compared between the vaccinated and unvaccinated
Ackland et al.[6]	1985–90, three abattoirs in South Australia	Retrospective, non-randomised, uncontrolled cohort study	2553 vaccinated employees during the study period, compared with 1365 unvaccinated employees within the study period as control	Vaccinated Yes: Q fever disease Yes 2, No 2551; No: Yes 55, No 1310	98% (92–100%)	Potential biases include selection bias, subjects volunteering for vaccination, uncertain and variable risk exposure by time and space, and inadequate case ascertainment
Marmion et al.[7]	1981–83, three abattoir sites of varying sizes in South Australia	Retrospective, non-randomised, uncontrolled cohort study	924 vaccinated employees including a small number who had immune markers before vaccination; 1349 unvaccinated employees as control, some not enrolled in study; some enrolled but not vaccinated	Vaccinated Yes: Q fever disease Yes 4, No 920; No: Yes 34, No 1315	83% (52–94%)	Both the vaccinated and unvaccinated groups consisted of subjects with variable Q fever immunity before vaccination. Potential confounders not quantified, not matched
Marmion et al.[8]	1981–88, five abattoir sites of varying sizes in South Australia, and a heterogeneous group of abattoir visitors vaccinated	Retrospective, non-randomised, uncontrolled cohort study	3532 vaccinated subjects in total, mostly abattoir employees and 212 visitors; unvaccinated employees or clinic attendees with records served as control. Some data on more homogeneous subgroups were available for analysis	Subgroup: subjects with high risk of exposure: Vaccinated Yes: Q fever disease Yes 3, No 2712; No: Yes 52, No 1960 Subgroup: subjects without immune markers, at a single site: Vaccinated Yes: Yes 2, No 688; No: Yes 7, No 54	96% (86–99%) 97% (88–99%)	Some subjects in other reported studies were also included in this study. Complete availability of records was not ascertained. Participating abattoirs were recruited at different times for varying duration during the study period

vaccination program and assumed a vaccine efficacy of 98%. It concluded on the basis of direct and indirect costs resulting from Workers' Compensation claims, that application of Q fever vaccine in high risk occupational settings would result in cost savings from a societal perspective.[9] Extrapolating 1993–94 data from NSW, an Australian review of Q fever conservatively estimated that the disease costs Australia approximately A$1 million and more than 1700 weeks of work time lost annually.[10]

Discussion

Although no large-scale, double-blind, randomised, placebo-controlled studies have evaluated the Australian registered Q fever vaccine, and despite the obvious limitations in the design of published studies, which mean that no precise measure of vaccine efficacy is available, study findings consistently suggest that vaccine efficacy is high compared with other vaccines currently used in Australian public health programs (eg, greater than 80% for pertussis vaccine, and ~30–80% for influenza vaccine for older people, depending on the selected outcome indicator). Although uncertainty with respect to the lower efficacy confidence limit may constrain decisions regarding the vaccine's application in lower risk situations, in established high-risk environments, particularly of an occupational nature, it has considerable protective value.

It is important that ongoing availability of an effective Q fever vaccine be ensured for protecting high-risk groups.

Acknowledgements

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Bug Breakfast in the *Bulletin*: Q fever

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Q fever is a zoonotic disease caused by the obligate intracellular Gram-negative bacterium *Coxiella burnetii*.[1] This Bug Breakfast discussed the disease, natural history and issues affecting population control.

Background

Q fever has been described in most countries of the world; however, it is a notifiable disease in only a few.[1] It has a low infectious dose and survives harsh environmental conditions.[2] As a result, it has been classified as a group B bioterrorist agent by the US Centers for Disease Control and Prevention (CDC).

Cattle, sheep and goats have been considered the primary human reservoir for *C. burnetii*; however, many other animals including Australian native species are known to be infected. Although most infected animals exhibit very few clinical signs, they shed *C. burnetii* in their milk, urine, faeces and particularly in amniotic fluid and placenta.[2]

Human transmission occurs through inhalation of aerosols of infected body fluids, the ingestion of unpasteurised milk or dairy products, or by inhalation of dried infectious dusts. Person-to-person transmission is rare. Those most at risk of Q fever are abattoir workers, livestock workers and veterinarians, farmers, shearers and laboratory workers. The Australian annual notification rate peaked at 4.9 per 100 000 persons in 1993 and decreased to 1.7 per 100 000 persons in 2005.[3] The highest incidence of Q fever occurs in south and central-western Queensland and northern NSW areas with men aged 40–44 years having the highest age-specific rate.[4,5]

Natural history, diagnosis and treatment

The incubation period for Q fever is usually 19–21 days (range one to six weeks). The illness presents with rapid onset of fevers, chills, profuse sweating, headaches, and muscle and joint pain, although the subclinical to clinical ratio is approximately 3 : 1. Occasionally hepatitis, pneumonia or neurological manifestations may occur in the acute illness. If *C. burnetii* persists, it leads to chronic,

localised Q fever infection. Endocarditis (particularly in those with underlying valvular heart disease) is the most common manifestation of chronic, localised infection. In addition, a postinfective fatigue syndrome (in the absence of ongoing infection), termed post-Q fever fatigue syndrome, is commonly reported.[6]

C. burnetii exists in two antigenic phases in laboratory culture systems, Phase I and Phase II. In nature, the organism exists in the Phase I state and is virulent and highly infectious.[1] Antibodies to both appear in a characteristic order in acute Q fever: IgM Phase II, IgM Phase I, IgG Phase II and then IgG Phase I. The appearance of IgG antibodies may be delayed up to six to eight weeks following the onset of symptoms.[7]

Diagnosis of acute Q fever requires a high index of clinical suspicion and relies on acute and convalescent serology. Although IgM antibody detection from a single serum sample is convenient, this assay is commonly associated with false positive test results.

Acute Q fever is treated with doxycycline, although rifampicin and ciprofloxacin are also effective. Chronic Q fever is characterised by ongoing illness, localised tissue injury and high titres of Phase I antibodies (IgG and IgA), and requires prolonged treatment with doxycycline, rifampicin and hydrochloroquine.

Population control of Q fever

Australia has access to the only licensed vaccine against Q fever available worldwide (QVax, CSL Ltd). Immunisation of high-risk occupational groups has been the major population control method; however, significant gaps remain in our knowledge of the optimal immunisation strategy. The vaccine is a purified suspension of formalin-inactivated *C. burnetii*. This vaccine is highly effective,[8] but its uptake is constrained by the fact that serological and skin testing are required prior to immunisation to prevent hypersensitivity reactions in those who may have been previously exposed. The vaccine produces protection via generation of cellular immunity (rather than antibodies) and is likely to provide lifelong protection.

A Federally-sponsored National Q fever Management Program to support screening and immunisation commenced in 2001 and concluded in NSW in June 2004.[9] The program resulted in most abattoir workers being immunised; however, many other at-risk rural populations remain unimmunised and account for the majority of notifications in many regions of Australia. Although respira-

tory precautions and personal protective equipment are likely to provide some protection in high-risk settings, their efficacy in rural working environments has not been evaluated and their practicality is often questionable.

At the time of presenting the Q fever Bug Breakfast in March 2006, the manufacturer had planned to discontinue the production of QVax. This prompted concern among rural communities and public health professionals. However, in May 2007 the Australian Government and the manufacturer committed to funding the construction of the BP Marmion Q Fever Vaccine Building in Melbourne[10] to produce and test Q fever vaccine. It is anticipated to be operational by July 2009. The manufacturer has also stated that vaccine supply will continue until this facility is operational.

Q fever remains a significant public health concern, particularly for rural communities. Despite the commitment to ongoing vaccine supply and testing, further understanding of the risk factors associated with acquiring Q fever in rural communities is required.

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Diphtheria

What is diphtheria?

Diphtheria is a contagious and potentially life-threatening bacterial infection that usually affects the nose and throat. Diphtheria was a common cause of death in children up until the 1940s but has now almost disappeared in Australia due to immunisation. It is re-emerging as a problem in some areas of the world with poor immunisation rates.

The bacteria that cause diphtheria are called *Corynebacterium diphtheriae* and *Corynebacterium ulcerans*. Some of these bacteria also produce a toxin.

What are the symptoms?

- Symptoms begin two to five days after exposure to the diphtheria bacteria. The first symptoms are usually a sore throat and a mild fever. A membrane forms over the throat and tonsils that can make it hard to swallow and breathe. The infection can also cause the lymph glands and tissues on both sides of the neck to swell.
- A toxin formed by some diphtheria bacteria can cause inflammation of heart muscle and nerves which can be fatal.
- Sometimes diphtheria can cause small skin sores that form larger ulcers, commonly on the legs.
- People usually take between two and five days to show symptoms after infection.

How is it spread?

- Diphtheria bacteria can live in the mouth, nose, throat or skin of infected individuals.
- People can get diphtheria by breathing in the diphtheria bacteria after an infected person has coughed or sneezed. People can also get diphtheria from close contact with discharges from an infected person's mouth, nose, throat or skin.
- People with diphtheria are infectious for up to four weeks from the onset of symptoms. Some people may become carriers of the bacteria and are infectious for longer.
- *C. ulcerans* is occasionally associated with consumption of unpasteurised milk or contact with animals.

Who is at risk?

Anyone who comes in contact with diphtheria during its infectious phase who has not had diphtheria in the past or has not been fully immunised is at risk.

How is it prevented?

- Diphtheria vaccination protects against the disease. It is part of the standard vaccination schedule and is given as DTP vaccine, which protects against diphtheria, tetanus and pertussis (whooping cough) in combination.
- DTP vaccine should be given at two, four and six months of age, followed by booster doses at four years and at 15 years of age.
- A high vaccination rate must be maintained to protect the population from resurgence of this disease.

How is it diagnosed?

- A doctor can diagnose diphtheria based on a clinical examination when the membrane is seen in the throat, and by testing throat swabs in a laboratory.
- Special laboratory tests are needed to detect the toxin.

How is it treated?

Diphtheria infection is treated with antibiotics. Sometimes an antitoxin is also needed.

What is the public health response?

Laboratories, school principals and directors of childcare centres and hospitals are required to notify cases of diphtheria to the local Public Health Unit under the *Public Health Act*, 1991. Public Health Units investigate cases and their contacts to review possible sources of infection and prevent further spread. Cases are isolated until they are not infectious. People who have had close or prolonged contact with a case may also be at risk of infection. Some contacts of cases are given antibiotics to stop the infection from developing.

NSW HEALTH

Communicable Diseases Report, New South Wales, May and June 2007

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**.

Figure 3 and Tables 1 and 2 show reports of communicable diseases received through to the end of May and June 2007 in NSW.

Meningococcal disease

Eight cases of meningococcal disease were notified in NSW in each of May and June. The number of case reports of meningococcal disease has fallen steadily in NSW from 253 in 2000 to 104 in 2006. As the peak season for this disease is approaching, NSW Health recently faxed general practitioners the following key messages:
- Early diagnosis and treatment by GPs can be life-saving: benzylpenicillin is the treatment of choice for suspected cases, preferably intravenously
- Patients should be reassessed when the diagnosis is uncertain
- Meningococcal C conjugate vaccine is still free for people up to 25 years of age who were not vaccinated during the school vaccination program.

A biweekly review of meningococcal disease, as well as treatment and prevention guidelines, appears at the above website.

Influenza

Influenza activity increased markedly in the second half of June to moderate levels, as measured by an upturn in the rate of patients attending emergency departments and the number of respiratory specimens from patients that have tested positive for influenza A.

Influenza symptoms include: fever, headache, myalgia, lethargy, coryza, sore throat and cough. Children may also have nausea, vomiting and diarrhoea.

Treatment is largely supportive although neuraminidase inhibitors can decrease the severity and duration of the illness if treatment is commenced within the first 48 hours of the onset of illness.

To help reduce its spread, people with influenza are encouraged to:
- Use tissues and cover the mouth and nose when coughing or sneezing
- Wash hands frequently with soap and running water for 10 seconds, especially after coughing or blowing the nose
- Stay away from school, work and crowded places while unwell.

Influenza vaccine is recommended for:
- All people aged 65 years and older
- Aboriginal people 50 years and older, and younger Aboriginal adults with medical risk factors
- Children (over six months of age) and adults with chronic illnesses
- Residents of nursing homes and other long-term care facilities
- Contacts of high-risk patients.

Influenza vaccination is not recommended by the National Health and Medical Research Council (NHMRC) as a universal population-based vaccination program for children; however, it can be given to people over six months of age who wish to reduce their likelihood of becoming ill with influenza. Full protection is usually achieved within 10 to 14 days of vaccination.

For details, see the weekly review of influenza activity at the above website.

Legionnaires' disease

Seven cases of legionnaires' disease due to infection with *Legionella pneumophila* were reported in May and four in June, including two people who died. None of the cases were linked. The number of cases is in line with seasonal expectations.

Enteric diseases

May

Eight outbreaks of food-borne disease were investigated by NSW Public Health Units in May 2007. These outbreaks affected between two and 17 people each, and were linked to four restaurants, three cafés and one aged-care facility. In one outbreak, reported by Sydney West Area Health Service (Penrith office), *Salmonella* infection was

identified as the cause of the illness affecting nine people whose common exposure was eating deep-fried ice cream at a restaurant. Further investigation of the source of ingredients used in the deep-fried ice cream is underway.

There were 27 outbreaks of gastrointestinal disease in institutions reported in NSW in May. These outbreaks included 17 aged-care facilities, five child-care centres, two hospitals and one school. Viruses were confirmed as the cause in seven outbreaks (rotavirus in one and norovirus in six), and the remaining 20 had clinical and epidemiological features consistent with viral gastroenteritis. These features can include nausea, vomiting, diarrhoea, fever, abdominal pain, headache and muscle aches and a high secondary attack rate.

Norovirus is the most commonly reported cause of diarrhoeal outbreaks in aged-care facilities in NSW, and rotavirus is a common cause of viral gastroenteritis outbreaks in child-care centres. Control depends on early recognition that an outbreak is occurring; exclusion (or isolation) of infectious cases; careful hand washing; care with food preparation and environmental cleaning.

For more information about norovirus, see: http://www.health.nsw.gov.au/infect/pdf/viral_gastro.pdf.

For information about rotavirus see: http://www.ncirs.usyd.edu.au/facts/rotavirus_vaccine_for_children_sep_2006.pdf

For guidelines on managing outbreaks in institutional settings see: http://www.health.nsw.gov.au/pubs/2004/gastroctrl_fs.html.

June

NSW Public Health Units investigated nine suspected food-borne disease outbreaks in June. These outbreaks were reported to affect between two and 10 people and occurred after people had eaten food from a café, restaurant or takeaway outlet. Scombroid poisoning was suspected to be the cause of illness in two people who ate grilled tuna. Their symptoms resolved following treatment with antihistamines. In most of the other outbreaks, there was insufficient evidence to identify specific pathogens or food items causing illness, and viral infection remains a likely cause for many of the illnesses.

Twenty outbreaks of gastroenteritis in institutions affecting more than 350 people were notified in June. The settings for these outbreaks were: aged-care facilities (11), child-care facilities (7), a hospital and a residential institution. Norovirus was confirmed to be the cause of four of the outbreaks, all in aged-care facilities. Although viral gastrointestinal disease in institutions tends to increase during winter, the rates of illness appears to be lower this year compared to 2006.

Statewide outbreak of cryptosporidiosis in NSW, November 2005–May 2006

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Cryptosporidiosis is a diarrhoeal disease caused by the intestinal parasite, *Cryptosporidium*.[1] There are several species of cryptosporidium, but two species, *Cryptosporidium parvum* and *Cryptosporidium hominis,* are thought to be responsible for the majority of infections in humans.[2] Humans are thought to be the major reservoir for *C. hominis,* while *C. parvum* infects humans and a range of animals including livestock.[3]

The major symptom of cryptosporidiosis is diarrhoea, which may be profuse and watery and is often associated with cramping abdominal pain. The incubation period is one to 12 days and a person with cryptosporidiosis may excrete oocysts for several weeks after symptoms resolve.[1] The mode of transmission is the faecal–oral route and includes animal-to-person, person-to-person, water-borne and rarely, food-borne transmission.[1] Outbreaks have been associated with swimming pools, drinking water supplies and, occasionally, consumption of contaminated beverages.[1]

Cryptosporidium has been notifiable to the NSW Health Department since 1996, and several outbreaks linked to swimming in contaminated pools have been identified.[4–7] Several hundred cases of cryptosporidiosis are reported in NSW each year and the number of cases tends to increase in the warmer months.

In November 2005, analysis of routinely collected notification data by NSW Health identified a sharp increase in the number of people with cryptosporidiosis: 114 cases were reported in November, up from 28 in October. Initial interviews with cases indicated that an unusually high number (28%) had reported contact with animals or farms. We received anecdotal reports from a NSW District veterinarian of concurrent cryptosporidiosis in calves with considerable numbers of oocysts detected in the faeces of sick calves. NSW Health subsequently issued a media release warning of the risk of cryptosporidiosis following contact with farm animals, urging people to take care with hygiene after animal contact, and (to avoid contamination of swimming pools) advising people not to swim if they had had diarrhoea in the previous week. To identify the reasons for the increase in notifications, we initiated a descriptive study of cases in NSW notified between 1 November 2005 and 29 May 2006.

Methods

A case of cryptosporidiosis was defined as a resident of NSW who had a laboratory confirmed case of cryptosporidiosis, and who was notified between 1 November

2005 and 29 May 2006. Demographic characteristics of notified cases were routinely entered into NSW Health's Notifiable Diseases Database (NDD). We asked NSW Public Health Units to interview all cryptosporidiosis cases notified between 1 November 2005 and 29 May 2006 with a standard questionnaire in order to identify potential risk factors including attendance at preschool, contact with another case, contact with pets and farm animals and farm visits. These data were entered into *Net Epi Case Manager,* a web-based database developed by the Centre for Epidemiology and Research (NSW Health).[8] NDD and *Net Epi* data were then analysed using SAS.[9] We analysed cases by exposure, date of onset, report date, area of residence, gender and age group. We ranked exposures in the following order: contact with farm animals (including farm visits), swimming, contact with another case and other exposure (such as preschool attendance, contact with pets and drinking untreated water).

Results

In December 2006, 844 cases of cryptosporidiosis were reported between 1 November 2005 and 29 May 2006. From November, cases of cryptosporidiosis occurred at higher than expected rates: 119 cases were reported in December, 104 in January, 130 in February, 179 in March, 109 in April and 89 to 29 May 2006. The epidemic curve shows a peak of cases in the weeks commencing 27 November and 12 March (Figure 1).

Demographic characteristics

Of all 844 cases, 397 (47%) resided in metropolitan Sydney and 53% in the remainder of NSW. The majority of the cases reported between 1 November 2005 and 31 December 2005 resided in areas outside of metro-

politan Sydney (233; 96%); this number fell to 267 (44%) among cases reported from 1 January to 29 May 2006. Overall, higher numbers were reported in the Northern Sydney, Hunter, South Eastern Sydney and Northern Rivers Areas (pre-2005 AHS boundaries).

Of all cases, 49% were male and 51% female. By age group, 41% of cases were aged 0–4 years, 18% were aged 5–9 years, 8% of cases were aged 10–19 years, 20% were aged 20–34 years and 13% were aged 35 or over.

Reported exposures

Of the 844 cases, PHU staff interviewed 464 (55%) about possible exposures. The most frequent exposures reported by cases in the two weeks before onset of signs and symptoms were: pet contact (59%), swimming (55%), preschool attendance (26%), drinking untreated water (20%) (of whom 67% drank tank water), other animal contact (21%) and visiting a farm (18%). Almost one third (30%) of cases reported contact with either another possible or notified case. The usual drinking water source at home was reported as town water for the majority of cases (75%), but 11% also reported drinking tank water at home; 5% of cases also reported drinking tank water at work or another place.

Over time, the number of cases reporting specific exposures changed (Figure 2). Swimming remained an important exposure throughout the period. The proportion of cases that reported swimming as an exposure peaked in January (58%) and decreased sharply in April (10%). Farm visits and contact with farm animals were more prominent exposures in the earlier stages of the outbreak (in November 2005, 21% reported contact with farm animals or visiting a farm), although some cases also reported these exposures in
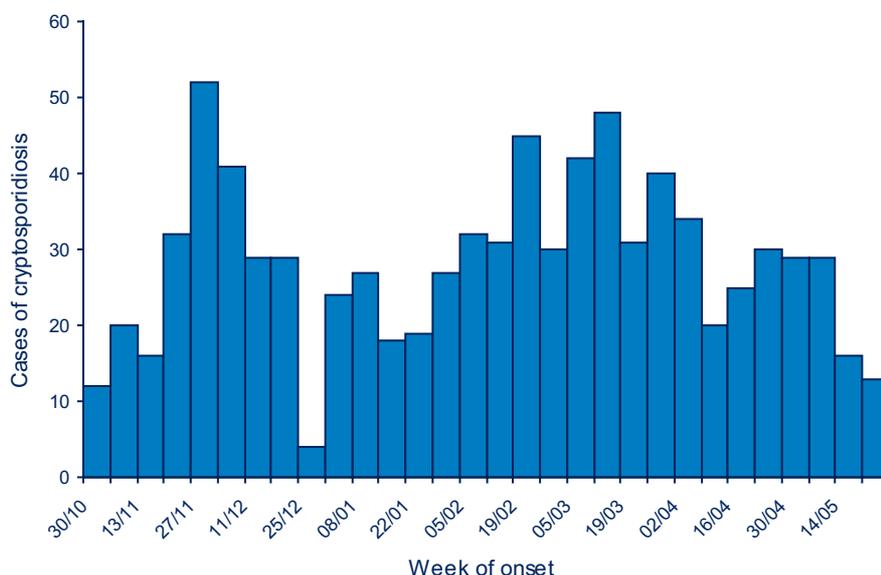

Figure 1. Cases of cryptosporidiosis notified in NSW, 30 October 2005 to 27 May 2006 (by onset date). Source: NSW Health Notifiable Diseases Database
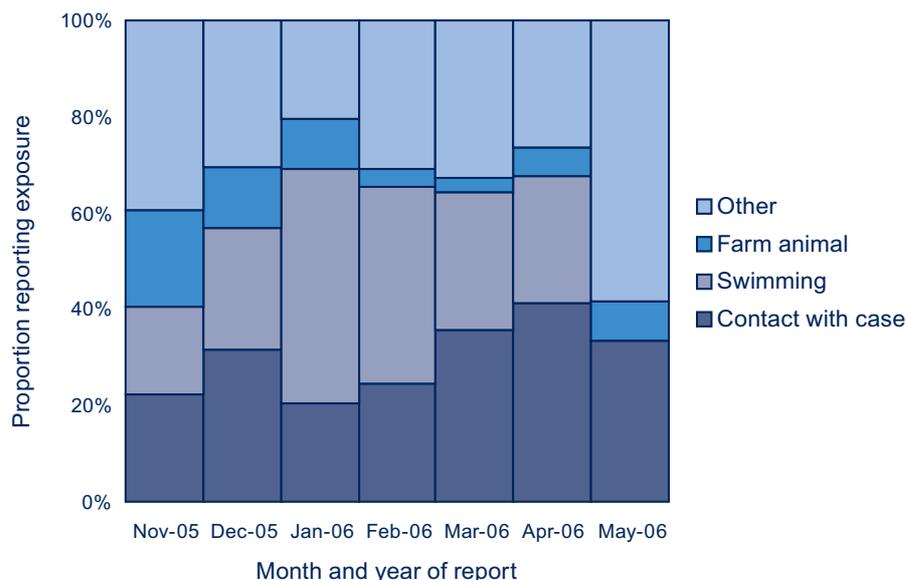
Figure 2. **Exposures of cryptosporidiosis cases (as proportion of total) in NSW by month of report, 1 November 2005 to 29 May 2006 (only one exposure reported for each case)**

December (8%) and January (7%). In November, a small number of cases (six) reported direct contact with scouring calves on their properties. The proportion of cases that reported contact with a possible or notified case peaked in March (42%), and varied from 20% in January to 42% in March. Preschool attendance was also reported consistently throughout the period (15% on average), with a lower proportion of cases reported in December and January as could be expected in holiday periods.

Prior to December 2005, the majority of cases were outside of metropolitan Sydney and in 2006, the proportion fell to under half. Also, prior to December 2005, a greater proportion of cases reported a farm visit or contact with farm animals than in early 2006 and swimming as an exposure was reported by a greater proportion of people in December and January.

Comment
In this outbreak a large proportion of cases reported swimming, pet contact, preschool attendance, farm visits, animal contact or contact with another case in their incubation period. The number of cases reporting specific exposures changed over time and may reflect changing risk in the course of the outbreak. Patterns of changing geographical distribution and changing exposures in this outbreak support the hypothesis that much of the outbreak began in rural areas of NSW where contact with farms and animals were risk factors, then over time spread via contamination of swimming pools and person-to-person contact.

A limitation in this analysis is a lack of a comparison group, which makes it difficult to draw causal links between exposures and illness. In addition, in recent years, two species of *Cryptosporidium: C. hominis* and *C. parvum* have been recognised to infect people. The laboratory data do not allow us to differentiate between the contributions each of them made in this outbreak. It may be that a high proportion of the cases identified earlier in this outbreak were *C. parvum* (associated with farm animals) than later (when person-to-person and swimming pool spread predominated in the outbreak).

Cryptosporidiosis transmission can be prevented by careful hand washing, avoidance of drinking contaminated drinking and pool water, and avoidance of swimming in pools while infectious. In light of an outbreak in 2007, the Centre for Health Protection conducted a poster campaign designed to educate swimmers about hygiene and pools.

In future outbreaks it would be useful to gather information on cryptosporidiosis species on cases from differing geographical areas and with different exposures in order to ascertain the relative contribution that the species make in outbreaks, and determine the geographical distribution of the two species.

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Erratum. In the Communicable Diseases Report for January and February 2007 (*N S W Public Health Bull* 2007; 18(3–4): 69) the following correction should be made.

Gastroenteritis outbreaks in institutions

Dec 06–Feb 07	
All outbreaks	33
Nursing homes	17
Hospitals	3
Child care	10
Schools	0
Other	3
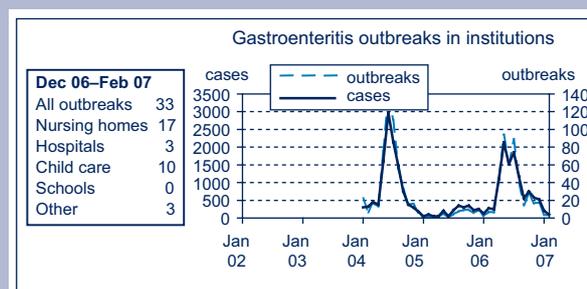
Figure 3.   Reports of selected communicable diseases, NSW, Jan 2002 to June 2007, by month of onset.
Preliminary data: case counts in recent months may increase because of reporting delays.
Laboratory-confirmed cases only, except for measles, meningococcal disease and pertussis.
BFV, Barmah Forest virus 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.
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%

Arbovirus infections

Apr 07–Jun 07	
Male	49%
<5 y	0.5%
5–24 y	11%
25–64 y	75%
65+ y	13%
Rural	92%

Legionnaires' disease

Apr 07–Jun 07	
Male	62%
<5 y	0%
5–24 y	0%
25–64 y	62%
65+ y	39%
Rural	46%

Cryptosporidiosis

Apr 07–Jun 07	
Male	54%
<5 y	28%
5–24 y	25%
25–64 y	44%
65+ y	3%
Rural	46%

Measles

Apr 07–Jun 07	
Male	100%
<5 y	0%
5–24 y	0%
25–64 y	100%
65+ y	0%
Rural	0%

Gastroenteritis outbreaks in institutions

Apr 07–Jun 07	
All outbreaks	20
Nursing homes	11
Hospitals	1
Child care	7
Schools	0
Other	1

Meningococcal disease

Apr 07–Jun 07	
Male	55%
<5 y	46%
5–24 y	23%
25–64 y	18%
65+ y	9%
Rural	36%

Gonorrhoea

Apr 07–Jun 07	
Male	84%
<5 y	0%
5–24 y	25%
25–64 y	75%
65+ y	0%
Rural	9%

Pertussis

Apr 07–Jun 07	
Male	38%
<5 y	10%
5–24 y	15%
25–64 y	59%
65+ y	16%
Rural	36%

Hepatitis A

Apr 07–Jun 07	
Male	55%
<5 y	36%
5–24 y	36%
25–64 y	18%
65+ y	9%
Rural	9%

Month of onset

Salmonella infections

Apr 07–Jun 07	
Male	49%
<5 y	21%
5–24 y	27%
25–64 y	42%
65+ y	10%
Rural	43%

Month of onset

Table 1. Reports of notifiable conditions received in May 2007 by Area Health Services

Condition	Greater Southern		Greater Western			Hunter/New England		North Coast		Northern Syd/Central Coast		South Eastern Syd/Illawarra		Sydney South West		Sydney West		Justice Health	Total	
Area Health Service (2007)	GMA	SA	FWA	MAC	MWA	HUN	NEA	MNC	NRA	CCA	NSA	ILL	SES	CSA	SWS	WEN	WSA	Health	For May	To date
Blood-borne and sexually transmitted																				
Chancroid[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Chlamydia (genital)[a]	35	23	15	14	48	154	32	26	39	50	96	33	261	100	93	33	86	–	1160	5506
Gonorrhoea[a]	1	–	–	1	–	3	2	1	6	2	13	2	66	30	12	3	13	4	157	690
Hepatitis B – acute viral[a]	–	1	–	1	–	–	–	–	2	1	–	–	–	1	1	–	–	–	7	25
Hepatitis B – other[a]	2	1	5	2	2	8	2	2	4	2	49	4	48	51	87	6	34	2	311	1464
Hepatitis C – acute viral[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1	16
Hepatitis C – other[a]	14	10	7	13	38	48	20	21	22	30	34	25	59	65	92	25	35	35	601	2725
Hepatitis D – unspecified[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	1	1	–	3	6
Lymphogranuloma venereum	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Syphilis[a]	–	–	5	2	4	2	–	3	3	3	2	6	47	30	15	1	5	1	130	521
Vector-borne																				
Barmah Forest virus[a]	–	24	1	–	1	10	2	9	17	3	2	26	2	–	1	1	3	–	102	340
Ross River virus[a]	–	5	4	10	7	49	5	13	14	4	4	5	1	1	3	1	1	–	127	385
Arboviral infection (Other)[a]	–	–	–	–	–	–	–	–	1	–	–	2	2	–	–	–	–	–	5	41
Malaria[a]	1	–	–	–	–	–	–	–	–	–	1	–	2	–	–	–	1	–	5	43
Zoonoses																				
Anthrax[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Brucellosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	4
Leptospirosis[a]	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	1	7
Lyssavirus[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Psittacosis[a]	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	1	1	–	3	19
Q fever[a]	1	1	–	5	3	1	3	2	4	1	–	–	–	–	–	–	–	–	21	96
Respiratory and other																				
Blood lead level[a]	–	–	–	2	–	2	–	–	–	–	–	1	1	3	3	–	–	–	12	58
Influenza[a]	1	1	–	1	–	1	–	–	3	1	4	2	4	3	1	5	9	–	36	178
Invasive pneumococcal infection[a]	2	–	1	1	2	1	1	–	3	–	5	2	4	3	1	–	–	–	26	135
Legionella longbeachae infection[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	1	1	1	–	–	4	17
Legionella pneumophila infection[a]	–	–	–	–	–	–	–	1	–	–	1	1	1	–	2	1	–	–	7	36
Legionnaires' disease (other)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Meningococcal infection (invasive)[a]	1	–	1	–	–	–	–	1	–	1	–	1	2	–	–	1	–	–	8	30
Tuberculosis	–	–	–	–	–	3	–	–	–	–	9	–	4	–	5	–	5	–	26	145
Vaccine-preventable																				
Adverse event after immunisation[b]	3	–	–	1	–	2	–	–	3	–	–	1	7	–	–	2	2	–	21	43
H.influenzae b infection (invasive)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Measles	–	–	–	–	–	–	–	–	–	–	–	–	–	1	1	–	–	–	2	4
Mumps[a]	–	3	–	–	2	–	–	–	–	2	–	–	6	2	1	1	4	–	21	29
Pertussis	8	–	–	3	–	13	2	7	4	9	20	–	32	11	8	10	24	–	156	613
Rubella[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	6
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Enteric																				
Botulism	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cholera[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Cryptosporidiosis[a]	–	–	1	2	–	6	1	3	4	3	4	2	9	1	8	1	1	–	46	182
Giardiasis[a]	2	3	1	13	4	23	4	3	1	6	35	9	42	14	6	13	33	–	212	973
Haemolytic uraemic syndrome	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	1	6
Hepatitis A[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	1	2	–	4	28
Hepatitis E[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	5
Listeriosis[a]	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	1	11
Salmonellosis[a]	9	4	4	1	10	14	7	13	11	18	32	6	38	13	25	9	14	–	229	1567
Shigellosis[a]	–	–	–	–	–	–	–	1	1	–	–	–	1	1	–	–	2	–	6	28
Typhoid[a]	–	–	–	–	–	–	–	–	–	–	1	–	–	1	–	–	2	–	4	25
Verotoxin-producing E.coli[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	5
Miscellaneous																				
Creutzfeldt–Jakob disease	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Meningococcal conjunctivitis	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

[a] Laboratory-confirmed cases only.
[b] HIV and AIDS data are reported separately in the Public Health Bulletin quarterly.
NB: From 1 Jan 2005, Hunter/New England AHS also comprises Great Lakes, Gloucester & Greater Taree LGAs, Sydney West also comprises Greater Lithgow LGA.

Table 2. Reports of notifiable conditions received in June 2007 by Area Health Services

Area Health Service (2007). Column groupings: Greater Southern (GMA, SA, FWA); Greater Western (MAC, MWA); Hunter/New England (HUN, NEA); North Coast (MNC, NRA); Northern Syd/Central Coast (CCA, NSA); South Eastern Syd/Illawarra (ILL, SES); Sydney South West (CSA, SWS); Sydney West (WEN, WSA); Justice Health; Total (For June, To date).

Condition	GMA	SA	FWA	MAC	MWA	HUN	NEA	MNC	NRA	CCA	NSA	ILL	SES	CSA	SWS	WEN	WSA	Justice Health	For June	To date
Blood-borne and sexually transmitted																				
Chancroid[b]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Chlamydia (genital)[a]	40	24	4	9	23	100	33	30	53	29	113	46	183	105	46	37	82	5	969	6609
Gonorrhoea[a]	1	–	–	–	–	3	–	–	2	–	14	–	38	29	10	4	9	1	111	819
Hepatitis B – acute viral[a]	–	–	–	–	–	1	–	–	1	–	–	–	1	–	–	–	–	–	3	30
Hepatitis B – other[a]	5	2	–	1	–	5	2	–	1	2	27	2	30	44	48	6	76	4	256	1760
Hepatitis C – acute viral[a]	–	–	–	–	–	1	–	–	–	–	–	–	1	1	–	–	–	–	3	19
Hepatitis C – other[a]	12	11	7	10	11	29	10	13	22	22	19	27	47	51	57	20	54	49	476	3273
Hepatitis D – unspecified[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1	7
Lymphogranuloma venereum	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Syphilis	1	–	1	1	1	5	–	–	2	–	16	4	37	24	13	–	9	–	114	618
Vector-borne																				
Barmah Forest virus[a]	–	8	–	2	–	14	–	10	7	–	–	4	–	–	–	–	1	–	46	392
Ross River virus[a]	2	4	–	4	–	24	6	14	7	7	1	6	2	–	–	1	1	–	79	472
Arboviral infection (Other)[a]	1	–	–	–	–	1	–	1	2	–	–	1	1	–	–	1	–	–	7	50
Malaria[a]	1	–	–	–	–	1	–	–	1	–	2	1	1	–	–	1	1	–	9	52
Zoonoses																				
Anthrax[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Brucellosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	4
Leptospirosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	7
Lyssavirus[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Psittacosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	–	–	2	22
Q fever[a]	–	1	–	4	–	2	6	–	4	–	–	–	–	–	–	–	–	–	17	114
Respiratory and other																				
Blood lead level[a]	4	1	–	4	1	1	–	1	1	–	–	1	–	–	–	–	–	–	14	86
Influenza[a]	2	1	–	–	–	11	–	1	1	–	6	1	9	4	4	4	13	–	51	232
Invasive pneumococcal infection[a]	3	1	–	–	–	5	2	5	–	–	7	3	4	5	4	5	4	–	48	185
Legionella longbeachae infection[a]	–	–	–	–	–	1	1	–	–	–	–	–	–	–	–	1	1	–	4	21
Legionella pneumophila infection[a]	–	–	–	–	–	1	–	–	–	–	–	–	1	–	–	1	1	–	4	41
Legionnaires' disease (other)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2
Meningococcal infection (invasive)[a]	–	1	–	1	–	–	–	–	1	–	1	2	2	1	1	1	–	–	8	38
Tuberculosis	–	–	–	–	–	–	–	–	1	–	7	–	3	–	5	–	5	–	15	168
Vaccine-preventable																				
Adverse event after immunisation[b]	1	–	–	–	1	4	–	3	1	–	2	2	3	1	–	2	5	–	25	76
H.influenzae b infection (invasive)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Measles	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Mumps[a]	–	–	–	–	–	–	–	–	–	–	5	–	10	3	1	–	–	–	19	72
Pertussis	5	7	–	4	2	16	5	1	8	10	25	3	32	6	8	11	23	–	167	782
Rubella[a]	–	–	–	–	–	–	–	–	–	–	2	–	–	1	–	–	2	–	5	11
Tetanus	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	3	3
Enteric																				
Botulism[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cholera[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1	2
Cryptosporidiosis[a]	–	1	–	1	–	2	1	–	2	1	1	1	1	–	3	–	2	–	16	199
Giardiasis[a]	3	2	–	2	1	12	2	3	1	4	23	10	30	17	8	7	18	–	142	1116
Haemolytic uraemic syndrome	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	6
Hepatitis A[a]	–	–	–	–	–	–	–	–	–	–	3	–	–	1	1	1	2	–	7	35
Hepatitis E[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	5
Listeriosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	1	–	–	13
Salmonellosis[a]	10	1	–	1	1	5	4	6	13	2	21	9	22	12	16	13	14	–	149	1722
Shigellosis[a]	–	–	–	–	–	–	1	–	–	–	2	–	–	–	–	–	–	–	3	34
Typhoid[a]	–	–	–	–	–	1	–	–	–	–	–	1	–	–	–	–	1	–	3	28
Verotoxin-producing E.coli[a]	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	1	6
Miscellaneous																				
Creutzfeldt–Jakob disease	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	1	1
Meningococcal conjunctivitis	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

[a] Laboratory-confirmed cases only.

[b] HIV and AIDS data are reported separately in the Public Health Bulletin quarterly.

NB: From 1 Jan 2005, Hunter/New England AHS also comprises Great Lakes, Gloucester & Greater Taree LGAs, Sydney West also comprises Greater Lithgow LGA.

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