NSW Public Health Bulletin

YEAR IN REVIEW: COMMUNICABLE DISEASE SURVEILLANCE, 2005

In this issue, we review the trends in reports of notifiable diseases among NSW residents received by the NSW public health units in 2005. Tables 1–5 report specific diseases by: year of onset, month of onset, area health service, and age group and sex. To assist with characterising the disease epidemiology, figures for area health services have been further divided into the geographical region covered by their component public health unit offices. Table 6 shows the number of people with notifiable conditions who were reported to have died at the time of follow-up by their local public health unit.

TRENDS

Among the 49,338 NSW residents with medical conditions notified by doctors, hospital staff, and laboratories for 2005, highlights are described below.

Conditions most frequently reported

- 1. Chlamydia: 11,284 cases, or 166 per 100,000 population, with the highest crude rates in the South Eastern Sydney/ Illawarra (Randwick region), Sydney South West (Camperdown region) and Hunter/ New England (Newcastle region) health areas
- 2. Pertussis: 5,801 cases, or 85 per 100,000 population, with the highest crude rates in the Greater Western (Dubbo region), South Eastern Sydney/ Illawarra (Randwick region) and Sydney South West (Camperdown region) health areas
- 3. Hepatitis C: 4,452 cases, or 65 per 100,000 population, with the highest crude rates in the Sydney South West and North Coast (Lismore region) health areas
- 4. Hepatitis B: 2,763 cases, or 41 per 100,000 population, with the highest crude rates in the Sydney South West and Sydney West (Parramatta region) health areas
- 5. Salmonella infections: 2,179 cases, or 32 per 100,000 population, with the highest crude rates in the North Coast (Lismore region), Sydney West (Parramatta region), and Sydney South West (Camperdown region) health areas.

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

- 1. Hepatitis C (4,452 cases, steadily declining from 8,682 in 2001), possibly as a result of declining unsafe injecting practices
- 2. Hepatitis B (2,763 cases, steadily declining from 4,560 cases in 2004), possibly as a result of increased immunisation coverage
- 3. Gastroenteritis in institutional settings, most likely due to norovirus infection (1,395 cases, a sharp decline from 12,784 in 2004 when a large community-wide outbreak occurred)
- 4. Q fever (142 cases, down from 309 in 2002), in part due to immunisation of high risk occupational groups
- 5. Meningococcal disease (141 cases, a steady decline, in both serogroups B and C, from 253 in 2000), in part a result of immunisation against meningococcal serogroup C infection
- 6. Hepatitis A (83 cases, the lowest number recorded since 1991)
- 7. Rubella (10 cases, declining from 191 in 2000), possibly as a result of increased immunisation coverage
- 8. Measles (five cases, the lowest number recorded since 1991), possibly as a result of increased immunisation coverage.

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

- 1. Chlamydia (11,284 cases), continuing its rise since it became notifiable in the late 1990s, possibly due to both better detection and a real increase in infections from unprotected sex
- 2. Pertussis (5,801 cases, up from 3,566 in 2004), most likely due to a state-wide outbreak largely among adults
- 3. Giardiasis (1,446 cases), a steady annual rise since 2002 and probably related to new laboratory testing practices
- 4. Influenza (1,414 cases), due for the most part to changes in laboratory reporting in early 2005
- 5. Cryptosporidiosis (849 cases, the highest number notified since 1998) due to two outbreaks, along with new laboratory testing practices
- 6. Malaria (204 cases, up from 101 in 2004), due to largely asymptomatic disease identified through screening of refugees from Africa
- 7. Shigellosis (135 cases), a steady rise in cases since 2003
- 8. Mumps (111 cases, up from 65 in 2004), mainly in young adults
- 9. Legionnaires' disease, due to infection with *Legionella pneumophila* (64 cases, up from 51 in 2004), partly the result of an outbreak in Wollongong early in the year

10. Verotoxigenic Escherichia coli infections (16 cases, up from 5 in 2004), probably related to changes in testing practices at one laboratory.

Conditions least frequently reported

There were no reported cases of chancroid, diphtheria, donovanosis, granuloma inguinale, lymphogranuloma venereum (LGV), plague, polio, rabies, severe acute respiratory syndrome (SARS), typhus, viral haemorrhagic fever or yellow fever in 2005.

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. Public health units routinely investigate all cases of some notifiable conditions (for example tuberculosis, measles, and meningoccocal disease) in order to implement control measures, and are likely to identify associated deaths. However, there are other notifiable conditions (for example chlamydia and gonorrhoea) where no routine investigation takes place and for which deaths will not be identified. 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:

- 1. Invasive pneumococcal disease (61 deaths)
- 2. HIV infection (36 deaths), including 19 people who died from AIDS, and 17 people with HIV infection who died of causes other than AIDS
- 3. Tuberculosis (21 deaths).

OUTBREAKS AND THREATS

Several notable disease outbreaks and threats were reported in 2005 in NSW. These included:

- an outbreak of Legionnaires' disease in Wollongong in February, thought to be related to contaminated cooling towers¹
- a state-wide outbreak of pertussis²
- an outbreak of cryptosporidiosis in May, related to swimming, and a second, possibly related to contact with calves, in late 2005^{3,4}
- an outbreak of enteroviral meningitis in the South Eastern Sydney/ Illawarra Health Area in February⁵
- two clusters of meningococcal disease, one associated with students residing on the same floor of a university residential college and the other linked to travel on a school bus.⁶

CONCLUSIONS

Some communicable diseases, notably chlamydia, pertussis and cryptosporidiosis, remain significant public health problems. However, much progress has been made with other diseases such as hepatitis B, hepatitis C, hepatitis A, measles, Q fever and meningococcal disease. We thank all those general and specialist medical practices, laboratories, hospitals, schools, childcare centres, and others, who have notified diseases of public health significance to their local public health units for investigation and control.

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DISEASE NOTIFICATIONS BY YEAR OF ONSET OF ILLNESS,* NSW, 1991 TO 2005

Conditions	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002		2004	
AIDS	441 9	433 31	483 23	555 40	483 28	375 56	214 70	181 95	132 16	134 42	102 111	114 178	145 219	100 184	91 106
Adverse event after immunisation Arbovirus infection: total*	409	343	656	381	539	1227	1806	783	1220	980	1191	662	1024	1148	1093
Barmah Forest virus infection*	6	6	25	39	271	172	185	134	249	197	401	395	451	403	448
Ross River virus infection*	297	324	599	331	236	1031	1598	583	952	750	717	181	494	701	589
Arboviral other*	106	13	32	11	32	24	23	66	19	33	73	86	79	44	56
Blood lead level ≥ 15µg/dl*	not						710	874	691	986	513	516	338	303	232
Botulism	0	0	0	0	0	0	0	0	1	0	0	0	0	1	0
Brucellosis*	2	2	4	4	2	1	3	3	2	1	1	2	3	7	3
Chancroid*	not notif								1 2469	0 3507	0 4500	0 5824	0 7785	0 10022	0
Chlamydia*	not notif 1	iable ui 0	1 Augi	ust 199 0	o 1	3	1	1	2469	3507	4500	5624 1	0	10022	11284
Cholera* Creutzfeldt-Jakob Disease	not notif	-		-		5			2	0			0	6	8
Cryptosporidiosis*	not notif				1996		157	1130	121	133	195	306	203	357	849
Food-borne illness (NOS)	2765	253	106	213	270	211	255	201	151	147	56	41	1071	550	309
Gastroenteritis (institutional)	158	406	443	296	1359	554	939	738	673	697	775	1752	3583	12784	1395
Giardiasis*	not notif							405	1091	978	967	863	1028	1235	1446
Gonorrhoea*	392	491	382	357	428	522	636	1054	1291	1060	1364	1527	1330	1445	1578
H.influenzae type b: total*	212	217	124	61	29	13	17	11	13	8	7	10	6	5	7
H.influenzae type b epiglottitis*	15 48	57 103	32 53	21 17	6 11	2 4	5 3	1 3	2 3	2 1	1	1	0	3 0	0 2
<i>H.influenzae</i> type b meningitis*	40 11	26	24	12	8	4	1	4	6	4	2	3	1	2	4
<i>H.influenzae</i> type b septicaemia* <i>H.influenzae</i> type b infection (NOS)*	138	20 31	15	11	o 4	4	8	4	2	4	2	5	5	2	4
Hepatitis A*	1119	901	579	585	614	958	1426	927	421	201	197	149	124	137	83
Hepatitis B: total*	1492	3169	3603	3983	4006	3507	3169	2957	3514	3974	4560	3548	2845	2813	2763
Hepatitis B: newly acquired*	409	112	95	74	61	43	53	58	77	100	94	88	74	53	56
Hepatitis B: other*	1083	3057	3508	3909	3945	3464	3116	2899	3437	3874	4466	3460	2771	2760	2707
Hepatitis C: total*	852	3895	5897	7822	6882	7001	6928	7211	8605	8298	8682	6699	5252	4927	4452
Hepatitis C: newly acquired*	22	26	22	16	32	18	19	112	112	222	295	153	127	60	41
Hepatitis C: other*	830	3869	5875	7806	6850	6983	6909	7099	8493	8076	8387	6546	5125	4867	4411
Hepatitis D*	0 0	8 0	12 1	19 2	19 0	9 3	11 6	3 4	14 7	12 9	11 6	9 6	12 6	14 8	15 7
Hepatitis E*	822	693	589	∠ 503	537	3 447	421	403	377	353	339	394	414	8 407	388
HIV infection*	not notif					447	3	405	11	9	2	7	5		11
Haemolytic uraemic syndrome Influenza: total*	not notif						0	0		0	244	1012	861	1012	1414
Influenza: Type A*	not notif										216	770	767	797	1055
Influenza: Type B*	not notif	iable ur	ntil Dece	ember 2	2000						27	241	55	162	281
Influenza: Type AB*	not notif	iable ur	ntil Dece	ember 2	2003									26	64
Influenza; Type (NOS)*	not notif										1	1	39	27	14
Legionnaires' disease: total*	37	104	66	60	75	74	33	46	40	41	68	44	60	80	89
Legionnaires' disease: L. longbeachae*	0	14 80	13 34	8 30	16	30 34	9	19	11	12	29	21	37	27	24
Legionnaires' disease: <i>L. pneumophila</i> *	16 21	10	34 19	22	35 24	34 10	18 6	22 5	22 7	26 3	38 1	22 1	23 0	51 2	64 1
Legionnaires' disease: other* Leprosy	1	7	5	3	3	2	Ő	Ő	1	2	4	Ó	2	5	1
Leptospirosis*	28	21	16	14	6	33	33	50	56	54	66	39	39	40	35
Listeriosis*	11	13	12	10	14	22	23	28	22	18	12	11	28	30	25
Malaria*	171	110	174	184	96	203	173	158	174	232	157	105	120	101	204
Measles: total	496	805	2348	1484	596	191	273	119	32	36	31	8	18	12	5
Measles: laboratory confirmed*	20	76	460	302	138	35	98	19	13	22	18	6	14	11	4
Measles: other	476	729	1888	1182	458	156	175	100	19	14	13	2	4	1	1
Meningococcal disease (invasive): total	128 0	121 3	153 7	142 7	113 23	161 36	219 54	186 55	221 95	253 93	234 90	216 105	202 100	149 81	141 73
Meningococcal disease: type B* Meningococcal disease: type C*	0	4	6	9	23	35	55	55	60	93 64	38	54	45	24	15
Meningococcal disease: type C Meningococcal disease: type W135*	0	0	0	ő	1	0	2	4	4	4	2	2	-10	5	8
Meningococcal disease: type Y*	Õ	Ő	1	ĩ	Ö	1	0	7	1	7	2	2	5	3	3
Meningococcal disease: type 1 Meningococcal disease: other	128	114	139	125	81	89	108	65	61	85	102	53	50	36	42
Mumps*	8	23	13	11	14	27	29	39	33	92	28	29	35	65	111
Paratyphoid*##	20	8	9	11	12	15	5	9	5	14	11	13	22	10	##
Pertussis	49	217		1405	1369	1156	4246	2309	1415	3688	4438	2012	2771	3566	5801
Pneumococcal disease: invasive*	not notif										444	861	801	905	643
	not notif		ntil Dece 403	ember 2 267	2000 201	287	258	236	164	132	38 144	155 309	87 288	81 223	121 142
Psittacosis*		212		201		636	258 153	236 78	46	191	58	309	288 24	223 18	142
Psittacosis* Q fever*	167	213 324		233	23/6		100	10							10
Psittacosis* Q fever* Rubella: total*	167 60	324	1186	233 229	2376 2375		153	78	45	191	58	35	23	17	
Psittacosis* Q fever* Rubella: total* Rubella*	167			233 229 4	2376 2375 1	631 5	153 0	78 0	45 1	191 0	58 0	35 0	23 1	17 1	0
Psittacosis* Q fever* Rubella: total*	167 60 59	324 324	1186 1184	229	2375	631									
Psittacosis* Q fever* Rubella: total* Rubella: Rubella: congenital* Salmonellosis*##	167 60 59 1	324 324 0 802	1186 1184 2 980	229 4 1101	2375 1 1366	631 5	0	0	1	0	0	0	1	1	0
Psittacosis* Q fever* Rubella: total* Rubella* Rubella: congenital*	167 60 59 1 1170	324 324 0 802	1186 1184 2 980	229 4 1101	2375 1 1366	631 5	0	0	1	0	0 1643	0 2100	1 1838	1 2134	0 2179
Psittacosis* Q fever* Rubella: total* Rubella: Rubella: congenital* Salmonellosis* ^{##} Shigellosis*	167 60 59 1 1170 not notif 579 1	324 324 0 802 ïable ur 873 3	1186 1184 2 980 ntil Deco 730 6	229 4 1101 ember 2 965 29	2375 1 1366 2000 834 132	631 5 1224 662 72	0 1698 512 57	0 1812 612 45	1 1438 586 87	0 1397 581 81	0 1643 134 545 67	0 2100 85 647 128	1 1838 59 841 245	1 2134 96 1042 302	0 2179 135 845 242
Psittacosis* Q fever* Rubella: total* Rubella: congenital* Salmonellosis*## Shigellosis* Syphilis: total	167 60 59 1 1170 not notif 579 1 1	324 324 0 802 iable ur 873 3 1	1186 1184 2 980 ntil Dece 730 6 1	229 4 1101 ember 2 965 29 2 2	2375 1 1366 2000 834 132 7	631 5 1224 662 72 4	0 1698 512 57 3	0 1812 612 45 1	1 1438 586 87 3	0 1397 581 81 3	0 1643 134 545 67 3	0 2100 85 647 128 3	1 1838 59 841 245 7	1 2134 96 1042 302 0	0 2179 135 845 242 8
Psittacosis* Q fever* Rubella: total* Rubella: congenital* Salmonellosis*## Shigellosis* Syphilis: total Syphilis: infectious*+ Syphilis: congenital Syphilis: other*	167 60 59 1 1170 not notif 579 1 1 577	324 324 0 802 iable ur 873 3 1 869	1186 1184 2 980 ntil Dece 730 6 1 723	229 4 1101 965 29 2 934	2375 1 1366 2000 834 132 7 695	631 5 1224 662 72 4 586	0 1698 512 57 3 452	0 1812 612 45 1 566	1 1438 586 87 3 496	0 1397 581 81 3 497	0 1643 134 545 67 3 475	0 2100 85 647 128 3 516	1 1838 59 841 245 7 589	1 2134 96 1042 302 0 740	0 2179 135 845 242 8 595
Psittacosis* Q fever* Rubella: total* Rubella: congenital* Salmonellosis*## Shigellosis* Syphilis: total Syphilis: infectious*+ Syphilis: congenital Syphilis: other* Tetanus	167 60 59 1 1170 not notif 579 1 1 577 5	324 324 0 802 iable ur 873 3 1 869 2	1186 1184 2 980 ntil Deco 730 6 1 723 5	229 4 1101 965 29 2 934 4	2375 1 1366 2000 834 132 7 695 0	631 5 1224 662 72 4 586 1	0 1698 512 57 3 452 3	0 1812 612 45 1 566 3	1 1438 586 87 3 496 1	0 1397 581 81 3 497 2	0 1643 134 545 67 3 475 0	0 2100 85 647 128 3 516 0	1 1838 59 841 245 7 589 1	1 2134 96 1042 302 0 740 0	0 2179 135 845 242 8 595 1
Psittacosis* Q fever* Rubella: total* Rubella: congenital* Salmonellosis*## Shigellosis* Syphilis: total Syphilis: infectious*+ Syphilis: congenital Syphilis: other*	167 60 59 1 1170 not notif 579 1 1 577	324 324 0 802 iable ur 873 3 1 869	1186 1184 2 980 ntil Dece 730 6 1 723	229 4 1101 965 29 2 934	2375 1 1366 2000 834 132 7 695	631 5 1224 662 72 4 586	0 1698 512 57 3 452	0 1812 612 45 1 566	1 1438 586 87 3 496	0 1397 581 81 3 497	0 1643 134 545 67 3 475	0 2100 85 647 128 3 516	1 1838 59 841 245 7 589	1 2134 96 1042 302 0 740	0 2179 135 845 242 8 595

year of onset = the earlier of patient reported onset date, specimen date or date of notification; * laboratory-confirmed cases only;

NOS = not otherwise specified; + includes syphilis-primary, syphilis-secondary, syphilis < 1 yr duration and syphilis-newly acquired No case of the following diseases have been notified since 1991: diphtheria*, granuloma inguinale*, lymphogranuloma venereum*, plague*, poliomyelitis*, rabies, typhus*, viral haemorrhagic fever and yellow fever ## from 2005, all paratyphoid recorded as salmonellosis

DISEASE NOTIFICATIONS BY MONTH OF ONSET OF ILLNESS,# NSW, 2005

							with of (Jacob					
Conditions	JAN	FEB	MAR	APR	MAY	JUN	nth of C JUL	AUG	SEP	ост	NOV	DEC	TOTAL
AIDS	8	9	8	10	9	9	10	9	7	4	3	5	91
Adverse event after immunisation	3	4	17	17	10	6	9	9	11	6	10	4	106
Arbovirus infection: total*	69	69	150	152	125	56	62	57	48	47	117	141	1093
Barmah Forest virus infection*	33	38	55	60	55	30	28	29	22	25	44	29	448
Ross River virus infection*	34 2	23 8	91 4	92 0	63 7	23 3	29 5	26 2	24 2	17 5	65 8	102 10	589 56
Arboviral:other*	10	20	28	19	14	21	19	22	19	13	27	20	232
Blood lead level ≥ 15µg/dl* Botulism	0	20	20	0	0	0	0	0	0	0	0	20	232
Brucellosis*	Õ	Õ	Ő	Ő	Ő	1	Õ	Ő	1	õ	1	Õ	3
Chlamydia*	896	1031	1032	919	1025	901	916	939	885	940	995	805	11284
Cholera*	0	0	0	0	0	0	0	0	0	0	0	0	0
Creutzfeldt-Jakob disease*	2	0	0	0	0	1	1	2	0	0	2	0	8
Cryptosporidiosis*	84	42	62	118	97	55	44	38	13	51	143	102	849
Giardiasis*	119	156	159	119	122	142	103	105	99	92	121	109	1446
Gonorrhoea*	120	119	140	131	130	120	147	130	131	156	137	117	1578
H.influenzae type b: total*	0	1 0	1 0	0 0	0 0	1 0	0 0	0 0	1 0	1 0	1 0	1 0	7 0
<i>H.influenzae type b</i> epiglottitis*	0	0	1	0	0	0	0	0	0	1	0	0	2
<i>H.influenzae type b</i> meningitis* <i>H.influenzae type b</i> septicaemia*	Ő	1	Ó	ŏ	ŏ	1	ŏ	Ő	1	0	1	ŏ	4
H.influenzae type b (NOS)*	0	0	Ō	0	0	0	0	Ō	0	0	0	1	1
Hepatitis A*	9	7	5	5	6	9	4	13	2	5	12	6	83
Hepatitis B: total*	217	244	271	224	237	232	225	224	252	215	222	200	2763
Hepatitis B: acute viral*	5	10	3	4	3	6	4	5	4	3	3	6	56
Hepatitis B: other*	212	234	268	220	234	226	221	219	248	212	219	194	2707
Hepatitis C: total*	317	357	394	379	390	367	373	399	366	376	367	367	4452
Hepatitis C: acute viral*	1	1	5	3	5	4	2	5	4	6	3	2	41
Hepatitis C: other*	316 0	356	389	376	385 0	363 0	371	394 4	362	370	364	365	4411
Hepatitis D*	2	2 1	1 0	2 1	0	0	2 1	4	3 0	0 0	1	0 0	15 7
Hepatitis E* HIV infection*	29	44	37	40	43	35	19	29	29	28	33	22	388
Haemolytic uraemic syndrome	2	1	0	0	0	0	1	1	0	0	1	5	11
Influenza: total*	117	111	49	98	65	65	202	332	217	88	42	28	1414
Influenza: Type A*	98	86	36	74	36	39	168	249	161	56	33	19	1055
Influenza: Type B*	13	18	10	20	26	18	21	72	52	25	4	2	281
Influenza: Type AB*	6	5	3	2	2	3	11	10	4	7	5	6	64
Influenza: Type (NOS)*	0	2	0	2	1	5	2	1	0	0	0	1	14
Legionnaires' disease: total*	9	13	6	7	6	4	4	11	11	8	8	2	89
Legionnaires' disease: L. longbeachae*	3 6	0 13	2 3	1 6	3 3	2 2	3 1	0 11	5 6	2 6	3 5	0 2	24 64
Legionnaires' disease: <i>L. pneumophila</i> *	0	0	1	0	0	2	0	0	0	0	0	2	1
Legionnaires' disease: other*	1	0	Ó	0	0	0	0	0	0	0	0	0	1
Leprosy Leptospirosis*	4	6	5	2	ĩ	õ	2	Ő	5	4	4	2	35
Listeriosis*	2	4	2	2	5	õ	2	1	1	3	3	0	25
Malaria*	12	32	77	7	12	6	4	16	8	12	9	9	204
Measles: total	0	2	1	2	0	0	0	0	0	0	0	0	5
Measles: laboratory confirmed*	0	2	1	1	0	0	0	0	0	0	0	0	4
Measles: other	0	0	0	1	0	0	0	0	0	0	0	0	1
Meningococcal disease (invasive): total	12	14	7	13	13	9	13	17	11	10	5	17	141
Meningococcal disease: type B*	4 5	10 1	6 0	8 1	4 1	7 0	8 1	11 0	6 1	3 1	1	5 3	73
Meningococcal disease: type C*	5 0	0	0	0	2	0	0	2	1	3	0	0	15 8
Meningococcal disease: type W135*	1	0	0	1	1	0	0	0	ò	0	0	0	3
Meningococcal disease: type Y* Meningococcal disease: other	2	3	1	3	5	2	4	4	3	3	3	9	42
Mumps*	15	13	6	9	15	8	7	11	7	10	4	6	111
Pertussis	330	308	345	354	523	640	667	748	614	515	434	323	5801
Pneumococcal disease: invasive*	29	21	36	35	46	92	92	78	80	40	48	46	643
Psittacosis*	7	6	14	21	11	12	8	12	11	6	7	6	121
Q fever*	11	4	12	8	17	14	8	12	15	7	17	17	142
Rubella: total*	1	1	0	3	1	1	0	0	2	0	0	1	10
Rubella*	1	1	0	3	1	1	0	0	2	0	0	1	10
Rubella: congenital*	0	0	0	0	100	0	0	0	0	0 191	0	0	0
Salmonellosis*##	225 15	236 15	243 13	221 16	199 13	111 9	115 15	110 4	98 12	181 13	233 6	207 4	2179 135
Shigellosis*	15 57	15 87	88	69	89	9 76	15 56	4 77	66	62	б 72	4 46	845
Syphilis: total	18	32	26	12	22	22	17	24	23	17	17	40	843 242
Syphilis: infectious*+ Syphilis: congenital*	2	0	20	2	1	1	0	24	23	0	1	1	242
Syphilis: other*	37	55	62	55	66	53	39	53	43	45	54	33	595
Tetanus	0	0	0	0	0	1	0	0	0	0	0	0	1
Tuberculosis*	69	33	29	27	29	40	40	47	31	42	34	32	453
Typhoid*	7	7	3	1	1	1	2	2	1	1	2	0	28
Verotoxigenic Escherichia coli infections*	0	2	0	2	0	1	2	1	0	1	2	5	16

onset = the earlier of patient reported onset date, specimen date or date of notification
* laboratory-confirmed cases only NOS = not otherwise specified
+ includes syphilis-primary, syphilis-secondary, syphilis < 1 yr duration and syphilis-newly acquired
from 2005, all paratyphoid recorded as salmonellosis

DISEASE NOTIFICATIONS BY AREA HEALTH SERVICE OF RESIDENCE (2005 AHS BOUNDARIES), CRUDE RATES PER 100,000 POPULATION, NSW, 2005*

	Greater	Southern [^]	Gre	eater Weste	ern^	Hunter Engl		North	Coast^
Conditions	Albury	Goulburn	Broken HIII	Dubbo	Bathurst	Newcastle	Tamworth	Port Macquarie	Lismore
AIDS	0.4	0.0	0.0	1.0	0.6	0.4	0.0	0.7	0.7
Adverse event after immunisation	6.4	0.5	4.2	0.0	2.3	3.2	2.2	1.8	1.1
Arbovirus infection: total*	23.4	10.9	59.3	32.4	26.0	40.8	32.9	96.4	77.3
Barmah Forest virus infection*	4.9	4.4	2.1	4.8	1.2	18.1	8.9	56.7	41.0
Ross River virus infection*	17.3	4.0	55.1	27.6	24.8	22.2	24.0	39.7	34.5
Arboviral:other*	1.1	2.5	2.1	0.0	0.0	0.5	0.0	0.0	1.8
Blood lead level ≥ 15µg/dl*	0.4	4.4	0.0	8.6	7.5	9.9	0.0	1.1	0.7
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chlamydia*	142.5	117.0	196.9	130.6	180.8	228.5	208.4	106.7	209.0
Cholera*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Creutzfeldt-Jakob disease*	0.4	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0
Cryptosporidiosis*	20.0	4.4	6.4	30.5	11.6	14.1	36.8	6.7	15.8
Giardiasis*	13.6	11.4	19.1	38.1	13.3	25.7	19.5	7.4	5.0
Gonorrhoea*	4.2	3.0	0.0	11.4	5.8	15.0	11.1	3.2	18.4
H.influenzae type b: total*	0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.0	0.4
H.influenzae type b epiglottitis*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
H.influenzae type b meningitis*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4
H.influenzae type b septicaemia*	0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.0	0.0
H.influenzae type b (NOS)*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis A*	0.8	0.0	0.0	1.9	1.2	0.4	2.2	0.0	0.7
Hepatitis B: total*	9.8	10.9	33.9	12.4	5.2	11.1	15.0	3.9	11.2
Hepatitis B: acute viral*	1.9	1.5	0.0	1.0	0.0	0.5	0.0	0.0	1.1
Hepatitis B: other*	7.9	9.4	33.9	11.4	5.2	10.6	15.0	3.9	10.1
Hepatitis C: total*	35.8	62.7	63.5	59.1	59.5	59.8	40.7	51.0	86.0
1 · · · · · · · · · · · · · · · · · · ·	1.1	4.0	0.0	1.9	0.0	0.7	0.0	0.0	0.0
Hepatitis C: acute viral*	34.7	58.8	63.5	57.2	59.5	59.1	40.7	51.0	86.0
Hepatitis C: other*	0.0	0.0	2.1	0.0	0.0	0.4	0.0	0.0	1.1
Hepatitis D*	0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.0	0.0
Hepatitis E*	0.4	0.0	0.0	1.9	1.2	1.9	0.6	1.8	3.2
HIV infection*	0.4								0.0
Haemolytic uraemic syndrome		0.5	0.0	0.0	0.6	0.2	0.6	0.4	
Influenza: total*	6.4	6.9	16.9	62.0	7.5	13.5	5.6	8.2	19.1
Influenza: Type A*	4.2	5.9	14.8	45.8	7.5	9.9	3.9	5.3	14.8
Influenza: Type B*	1.1	1.0	0.0	3.8	0.0	3.7	1.1	0.7	4.3
Influenza: Type AB*	0.0	0.0	0.0	12.4	0.0	0.0	0.0	2.1	0.0
Influenza: Type (NOS)*	1.1	0.0	2.1	0.0	0.0	0.0	0.6	0.0	0.0
Legionnaires' disease: total*	0.4	1.5	4.2	0.0	1.2	0.7	0.0	0.4	0.0
Legionnaires' disease: L.longbeachae*	0.0	1.0	0.0	0.0	0.6	0.7	0.0	0.0	0.0
Legionnaires' disease: L. pneumophila*	0.4	0.5	4.2	0.0	0.6	0.0	0.0	0.4	0.0
Legionnaires' disease: other*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Leptospirosis*	0.8	1.0	0.0	1.9	0.6	0.5	4.5	1.1	3.2
Listeriosis*	0.8	1.5	0.0	0.0	0.0	0.7	1.1	0.0	0.0
Malaria*	0.8	1.5	2.1	1.0	1.2	5.3	0.0	1.8	2.9
Measles: total	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Measles: laboratory confirmed*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Measles: other	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal disease (invasive): total	1.5	2.5	4.2	1.9	3.5	1.9	1.7	0.7	2.2
Meningococcal disease: type B*	1.1	0.0	2.1	1.9	2.3	0.9	1.1	0.4	1.1
Meningococcal disease: type C*	0.4	1.0	0.0	0.0	0.0	0.7	0.0	0.0	0.0
Meningococcal disease: type 0 Meningococcal disease: type W135*	0.0	0.0	0.0	0.0	1.2	0.2	0.0	0.0	0.0
Meningococcal disease: type Y*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
0 31	0.0	1.5	2.1	0.0	0.0	0.0	0.6	0.4	1.1
Meningococcal disease: other	0.0	0.5	0.0	3.8	0.0	0.2	0.0	0.4	2.9
Mumps*	87.5	69.1	42.4	293.6	29.5	84.9	43.5	37.9	68.7
Pertussis	7.9	7.9	12.7	13.3	16.2	15.0	43.5	9.2	7.9
Pneumococcal disease: invasive*	5.3	1.0	0.0	0.0	2.9	3.3	3.9	9.2 1.1	1.4
Psittacosis*									
Q fever*	1.5	3.5	10.6	19.1	4.0	4.2	15.0	4.6	7.6
Rubella: total*	0.0	0.0	0.0	0.0	0.0	0.4	0.6	0.4	0.4
Rubella*	0.0	0.0	0.0	0.0	0.0	0.4	0.6	0.4	0.4
Rubella: congenital*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Salmonellosis*##	21.5	21.2	16.9	24.8	13.3	28.7	34.5	25.5	77.0
Shigellosis*	1.5	0.5	42.4	11.4	0.6	1.1	1.1	1.4	3.6
Syphilis: total	1.1	2.0	52.9	9.5	4.6	3.3	11.1	5.7	7.9
Syphilis: infectious*+	0.0	0.0	2.1	0.0	1.2	1.6	0.0	0.0	2.9
Syphilis: congenital*	0.0	0.0	0.0	0.0	0.6	0.0	0.0	0.0	0.0
Syphilis: other*	1.1	2.0	50.8	9.5	2.9	1.8	11.1	5.7	5.0
Tetanus	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tuberculosis*	1.1	2.5	0.0	0.0	1.7	2.3	0.6	1.1	0.7
100010010010			0.0	0.0	0.0	0.0	0.0		0.0
Typhoid*	0.0	0.0	0.0					0.4	

year of onset = the earlier of patient reported onset date, specimen date or date of notification * laboratory-confirmed cases only NOS = not otherwise specified

+ includes syphilis-primary, syphilis-secondary, syphilis < 1 yr duration and syphilis-newly acquired ## from 2005, all paratyphoid recorded as salmonellosis

AHS further divided into the geographical region covered by their respective Public Health Unit offices
 ** Rate is based on a denominator of 8000 persons

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TABLE 3 continued

DISEASE NOTIFICATIONS BY AREA HEALTH SERVICE OF RESIDENCE (2005 AHS BOUNDARIES), CRUDE RATES PER 100,000 POPULATION, NSW, 2005*

		Sydney/	South I Sydney/	Eastern Illawarra [^]	Sydney So	outh West [^]	Sydne	ey West^	Justice Health *
Conditions	Gosford	Hornsby	Wollongong	Randwick	Camperdown	Liverpool	Penrith	Parramatta	nounn
AIDS	1.0	0.4	0.0	3.1	4.2	0.6	1.6	1.1	0.0
Adverse event after immunisation	2.9	0.9	0.0	2.1	0.6	0.2	2.2	0.8	12.5
Arbovirus infection: total*	7.4	2.6	3.0	2.9	3.4	1.1	2.5	1.3	0.0
Barmah Forest virus infection*	1.9	0.5	1.6	0.3	0.4	0.2	0.3	0.3	0.0
Ross River virus infection*	5.5	0.8	1.4	1.2	1.2	0.6	1.9	0.5	0.0
Arboviral:other*	0.0	1.4	0.0	1.4	1.8	0.2	0.3	0.5	0.0
Blood lead level ≥ 15µg/dl*	2.6	1.0	0.5	1.4	3.2	4.1	5.6	4.9	0.0
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis*	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.3	0.0
Chlamydia*	131.6	135.9	143.7	260.0	251.7	84.3	116.6	114.7	1000.0
Cholera*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Creutzfeldt-Jakob disease*	0.0	0.3	0.3	0.0	0.0	0.4	0.0	0.0	0.0
Cryptosporidiosis*	9.3	12.2	10.5	12.9	11.3	7.5	18.7	9.5	0.0
Giardiasis*	17.4	32.5	14.3	30.9	22.3	13.3	26.7 4.7	22.6	0.0 37.5
Gonorrhoea*	4.5	12.6	13.2	79.6	71.5	10.3		10.7	
H.influenzae type b: total*	0.0 0.0	0.0 0.0	0.3 0.0	0.1 0.0	0.0 0.0	0.0 0.0	0.6 0.0	0.0 0.0	0.0 0.0
H.influenzae type b epiglottitis*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
<i>H.influenzae type b</i> meningitis*									
H.influenzae type b septicaemia*	0.0 0.0	0.0 0.0	0.0 0.0	0.1 0.0	0.0 0.0	0.0 0.0	0.3 0.3	0.0 0.0	0.0 0.0
H.influenzae type b (NOS)*	0.0	1.1	0.0	1.1	3.2	1.6	1.2	1.7	0.0
Hepatitis A*	10.6	36.5	14.5	45.6	3.2 102.9	85.0	13.4	62.3	512.5
Hepatitis B: total*	0.0	36.5 0.4	0.3	45.6 1.5	1.8	1.0	0.3	02.3	512.5 62.5
Hepatitis B: acute viral*	10.6	36.1	14.3	44.1	101.2	84.0	13.1	62.1	450.0
Hepatitis B: other*	62.4	25.5	58.4	64.4	92.7	70.6	48.8	46.5	430.0 6238.0
Hepatitis C: total*	0.0	0.1	0.0	0.0	1.8	0.0	0.6	0.4	112.5
Hepatitis C: acute viral*	62.4	25.4	58.4	64.4	90.9	70.6	48.2	46.1	6125.0
Hepatitis C: other*	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.7	25.0
Hepatitis D* Hepatitis E*	0.0	0.1	0.0	0.0	0.2	0.1	0.0	0.4	0.0
HIV infection*	2.3	3.0	1.6	18.0	19.0	1.3	3.1	3.4	0.0
Haemolytic uraemic syndrome	0.0	0.1	0.3	0.3	0.0	0.0	0.3	0.0	0.0
Influenza: total*	7.1	19.8	10.0	53.5	13.0	19.4	12.7	27.7	50.0
Influenza: Type A*	4.5	14.6	7.5	39.7	9.5	15.3	10.6	20.0	37.5
Influenza: Type B*	1.0	3.1	1.4	12.0	3.2	3.6	1.6	6.9	12.5
Influenza: Type AB*	1.6	1.7	1.1	1.7	0.0	0.1	0.6	0.7	0.0
Influenza: Type (NOS)*	0.0	0.4	0.0	0.0	0.4	0.4	0.0	0.1	0.0
Legionnaires' disease: total*	1.9	2.4	4.0	0.6	1.0	0.2	1.9	2.3	0.0
Legionnaires' disease: L. longbeachae*	0.3	0.8	0.3	0.3	0.2	0.2	0.0	0.5	0.0
Legionnaires' disease: L. pneumophila*	1.6	1.6	3.8	0.4	0.6	0.0	1.9	1.7	0.0
Legionnaires' disease: other*	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0
Leprosy	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0
Leptospirosis*	0.3	0.0	0.3	0.0	0.0	0.1	0.3	0.0	0.0
Listeriosis*	0.0	0.4	0.3	0.5	0.4	0.1	0.0	0.4	0.0
Malaria*	2.3	2.2	2.4	2.5	1.2	2.8	1.6	7.3	0.0
Measles: total	0.0	0.2	0.0	0.3	0.0	0.0	0.0	0.1	0.0
Measles: laboratory confirmed*	0.0	0.1	0.0	0.3	0.0	0.0	0.0	0.1	0.0
Measles: other	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal disease (invasive): total	4.2	1.6	0.8	2.7	1.0	1.8	2.8	2.5	0.0
Meningococcal disease (invasive): total	2.3	1.0	0.8	1.2	0.8	0.6	1.9	1.2	0.0
Meningococcal disease: type D*	0.6	0.1	0.0	0.1	0.0	0.1	0.3	0.1	0.0
Meningococcal disease: type W135*	0.6	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.0
Meningococcal disease: type Y*	0.0	0.1	0.0	0.0	0.2	0.1	0.0	0.0	0.0
Meningococcal disease: other	0.6	0.4	0.0	1.0	0.0	1.0	0.6	1.2	0.0
Mumps*	0.6	3.6	0.5	2.6	2.8	1.1	0.9	1.3	0.0
Pertussis	52.8	84.0	63.0	129.3	103.9	60.3	92.7	99.4	12.5
Pneumococcal disease: invasive*	11.6	7.7	9.2	6.5	10.5	8.2	11.2	10.2	0.0
Psittacosis*	1.0	0.3	2.4	0.6	0.2	1.0	10.3	0.7	0.0
Q fever*	0.3	0.4	0.5	0.1	0.2	0.5	0.6	0.0	0.0
Rubella: total*	0.3	0.1	0.0	0.0	0.4	0.0	0.3	0.0	0.0
Rubella*	0.3	0.1	0.0	0.0	0.4	0.0	0.3	0.0	0.0
Rubella: congenital*	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Salmonellosis*##	23.2	30.5	19.4	35.1	34.8	31.4	29.9	39.2	0.0
Shigellosis*	1.3	2.1	0.3	2.4	2.8	0.7	0.9	1.2	0.0
Syphilis: total	3.2	5.2	4.9	22.6	30.8	22.2	5.0	13.1	75.0
Syphilis: infectious*+	0.3	2.4	0.8	15.2	11.1	0.4	0.9	1.7	12.5
Syphilis: congenital*	0.0	0.3	0.0	0.0	0.2	0.1	0.0	0.3	0.0
Syphilis: other*	2.9	2.6	4.0	7.5	19.6	21.7	4.0	11.1	62.5
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tuberculosis*	1.9	5.5	1.9	8.4	15.0	10.8	3.1	15.2	12.5
Typhoid*	0.6	0.4	0.3	0.5	1.6	0.5	0.3	0.4	0.0
	0.0		0.0	0.0	0.0	0.1	0.3	0.1	0.0

year of onset = the earlier of patient reported onset date, specimen date or date of notification * laboratory-confirmed cases only NOS = not otherwise specified

+ includes syphilis-primary, syphilis-secondary, syphilis < 1 yr duration and syphilis-newly acquired

from 2005, all paratyphoid recorded as salmonellosis

^ AHS further divided into the geographical region covered by their respective Public Health Unit offices

** Rate is based on a denominator of 8000 persons

NUMBER OF DISEASE NOTIFICATIONS BY AREA HEALTH SERVICE OF RESIDENCE (2005 AHS BOUNDARIES), NSW, 2005#

	Greater	Southern [^]	Gre	eater Weste	ern^	Hunter Engl		North	Coast^
Conditions	Albury	Goulburn	Broken HIII	Dubbo	Bathurst	Newcastle	Tamworth	Port Macquarie	Lismore
AIDS	1	0	0	1	1	2	0	2	2
Adverse event after immunisation	17	1	2	0	4	18	4	5	3
Arbovirus infection: total*	62	22	28	34	45	232	59	272	215
Barmah Forest virus infection*	13	9	1	5	2	103	16	160	114
Ross River virus infection*	46	8	26	29	43	126	43	112	96
Arboviral:other*	3	5	1	0	0	3	0	0	5
Blood lead level $\geq 15\mu g/dl^*$	1	9	0	9	13	56	0	3	2
Botulism	0	0	0	0	0	0	0	0	0
Brucellosis*	0	0	0	0	0	0	0	0	0
Chlamydia*	378	237	93	137	313	1299	374	301	581
Cholera*	0	0	0	0	0	0	0	0	0
Creutzfeldt-Jakob disease*	1	0	0	0	0	1	0	0	0
Cryptosporidiosis*	53	9	3	32	20	80	66	19	44
Giardiasis*	36	23	9	40	23	146	35	21	14
Gonorrhoea*	11	6	0	12	10	85	20	9	51
H.influenzae type b: total*	0	0	0	0	0	2	0	0	1
H.influenzae type b epiglottitis*	0	0	0	0	0	0	0	0	0
<i>H.influenzae type b</i> meningitis*	0	0	0	0	0	0	0	0	1
<i>H.influenzae type b</i> septicaemia*	0	0	0	0	0	2	0	0	0
H.influenzae type b (NOS)*	0	0	0	0	Ō	0	Ō	0	0
Hepatitis A*	2	Õ	Õ	2	2	2	4	õ	2
Hepatitis B: total*	26	22	16	13	9	63	27	11	31
Hepatitis B: acute viral*	5	3	0	1	Ő	3	0	0	3
Hepatitis B: other*	21	19	16	12	9	60	27	11	28
Hepatitis C: total*	95	127	30	62	103	340	73	144	239
Hepatitis C: total* Hepatitis C: acute viral*	33	8	0	2	0	4	0	0	200
	92	119	30	60	103	336	73	144	239
Hepatitis C: other*	0	0	1	0	0	2	0	0	200
Hepatitis D*	1	0	0	0	0	0	0	0	0
Hepatitis E*	1	0	0	2	2	11	1	5	9
HIV infection*	1	1	0	2	1		1	1	9
Haemolytic uraemic syndrome						1			
Influenza: total*	17	14	8	65	13	77	10	23	53
Influenza: Type A*	11	12	7	48	13	56	7	15	41
Influenza: Type B*	3	2	0	4	0	21	2	2	12
Influenza: Type AB*	0	0	0	13	0	0	0	6	0
Influenza: Type (NOS)*	3	0	1	0	0	0	1	0	0
Legionnaires' disease: total*	1	3	2	0	2	4	0	1	0
Legionnaires' disease: L. longbeachae*	0	2	0	0	1	4	0	0	0
Legionnaires' disease: L. pneumophila*	1	1	2	0	1	0	0	1	0
Legionnaires' disease: other*	0	0	0	0	0	0	0	0	0
Leprosy	0	0	0	0	0	0	0	0	0
Leptospirosis*	2	2	0	2	1	3	8	3	9
Listeriosis*	2	3	0	0	0	4	2	0	0
Malaria*	2	3	1	1	2	30	0	5	8
Measles: total	0	0	0	0	0	0	0	0	0
Measles: laboratory confirmed*	0	0	0	0	0	0	0	0	0
Measles: other	0	0	0	0	0	0	0	0	0
Meningococcal disease (invasive): total	4	5	2	2	6	11	3	2	6
Meningococcal disease (invasive). total Meningococcal disease: type B*	3	0	1	2	4	5	2	1	3
Meningococcal disease: type D	1	2	0	0	0 0	4	0	Ö	Ő
Meningococcal disease: type C Meningococcal disease: type W135*	O	0	õ	õ	2	1	0	Ő	Ő
	0	0	0	0	0	0	0	0	0
Meningococcal disease: type Y*	0	3	1	0	0	1	1	1	3
Meningococcal disease: other		3		4	0	4	0		
Mumps*	1		0					1	8
Pertussis	232	140	20	308	51	483	78	107	191
Pneumococcal disease: invasive*	21	16	6	14	28	85	3	26	22
Psittacosis*	14	2	0	0	5	19	7	3	4
Q fever*	4	7	5	20	7	24	27	13	21
Rubella: total*	0	0	0	0	0	2	1	1	1
Rubella*	0	0	0	0	0	2	1	1	1
Rubella: congenital*	0	0	0	0	0	0	0	0	0
Salmonellosis*##	57	43	8	26	23	163	62	72	214
Shigellosis*	4	1	20	12	1	6	2	4	10
Syphilis: total	3	4	25	10	8	19	20	16	22
Syphilis: infectious*+	0	0	1	0	2	9	0	0	8
Syphilis: congenital*	0	0	0	0	1	0	0	0	0
Syphilis: other*	3	4	24	10	5	10	20	16	14
21	0	1	0	0	0	0	0	0	0
Tetanus Tuberaulacia*	3	5	0	0	3	13	1	3	2
Tuberculosis*	0	5 0	0	0	0	0	0	1	2
Typhoid*	0	1	0	0	0	10	0	0	0
Verotoxigenic Escherichia coli infections*									

year of onset = the earlier of patient reported onset date, specimen date or date of notification * laboratory-confirmed cases only NOS = not otherwise specified

(a) = includes cases with unknown PHU + includes synhilic primary (11)

+ includes syphilis-primary, syphilis-secondary, syphilis < 1 yr duration and syphilis-newly acquired

from 2005, all paratyphoid recorded as salmonellosis

^ AHS further divided into the geographical region covered by their respective Public Health Unit offices

TABLE 4 continued

NUMBER OF DISEASE NOTIFICATIONS BY AREA HEALTH SERVICE OF RESIDENCE (2005 AHS BOUNDARIES), NSW, 2005#

Conditions Goard Hornary Walkings Partons Parton Parton Parton AUDS 3 0 17 2 2 7 8 1 0 11 Adverse event after immunisation 3 7 0 17 2 2 7 6 1 0 128 Brass fibre virus infection: 17 6 5 10 2 1 4 0 558 Blood lend lend - 15gigdt 8 8 2 11 16 34 18 7 0 2 0 <		Northerr Centra	Sydney/		Eastern Illawarra [^]	Sydney We		Sydne	ey West^	Justice Health	TOTAL (a)
Arborns P T 0 17 3 2 7 6 1 106 Barmal Forest virus infection* 6 4 6 2 2 2 1 1 23 17 9 6 10 0 1083 Barmal Forest virus infection* 17 0 1 0 10<	Conditions							Penrith	Parramatta		
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Borules 0 </td <td></td>											
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Chalamy 409 102 534 2044 1274 698 377 867 860 10 Credutfield-Jakko disease* 0		0	0	0	0	0	1	0	2	0	3
Circuitability decodes 0 2 1 0 0 3 0 0 8 Giardiasi' 29 98 39 104 67 62 60 72 0 849 Giardiasi' 64 261 53 249 113 110 86 171 0 144 Hinduraze type b: total' 0 0 1 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 <		409	1092	534	2094	1274	698	375	867	80	11284
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Tuberculosis* 6 44 7 68 76 89 10 115 1 453 Typhoid* 2 3 1 4 8 4 1 3 0 28											
Typhoid* 2 3 1 4 8 4 1 3 0 28											
	Verotoxigenic <i>Escherichia coli</i> infections*	0	1	0	0	0	1	1	1	0	16

year of onset =the earlier of patient reported onset date, specimen date or date of notification * laboratory-confirmed cases only NOS = not otherwise specified

(a) = includes cases with unknown PHU

+ includes syphilis-primary, syphilis-secondary, syphilis < 1 yr duration and syphilis-newly acquired

from 2005, all paratyphoid recorded as salmonellosis

^ AHS further divided into the geographical region covered by their respective Public Health Unit offices

DISEASE NOTIFICATIONS BY AGE GROUP AND SEX OF THE CASE, NSW, 2005#

	0-	-4 yrs	5-	24 yrs	25-	44 yrs	45-	64 yrs	65	+ yrs		Total	
Conditions	М	F	М	F	М	F	М	F	М	F	М	F	Total ^(a)
AIDS	0	0	0	1	7	44	5	32	0	0	12	77	91
Adverse event after immunisation	33	19	4	9	6	1	16	3	11	4	70	36	106
Arbovirus infection: total*	6 2	2 0	55 24	50 21	225 85	181 77	205 86	236 102	49 17	79 33	540 214	548 233	1093 448
Barmah Forest virus infection*	4	2	24 25	21	129	91	112	125	30	43	300	285	589
Ross River virus infection* Arboviral:other*	0	0	6	5	11	13	7	9	2	3	26	30	56
Blood lead level $\geq 15\mu g/dl^*$	2	5	1	31	4	100	3	76	2	8	12	220	232
Botulism	0	0	0	0	0	0	0	0	0	0	0	0	0
Brucellosis*	0	1	0	2	0	0	0	0	0	0	0	3	3
Chlamydia*	16	23	4305	2093	1955	2416	97	339	3	14	6376	4885	11284 ^(b)
Cholera*	0 0	0 0	0	0	0	0 0	0 2	0 2	0 1	0 3	0 3	0 5	0 8
Creutzfeldt-Jakob disease*	148	199	136	166	98	63	12	14	6	4	400	446	849
Cryptosporidiosis* Giardiasis*	161	196	130	179	259	229	89	111	60	29	699	744	1446
Gonorrhoea*	1	0	93	312	80	925	12	145	1	6	187	1388	1578 ^(b)
H.influenzae type b: total*	1	3	0	0	0	0	0	0	2	1	3	4	7
H.influenzae type b epiglottitis*	0	0	0	0	0	0	0	0	0	0	0	0	0
H.influenzae type b meningitis*	0	2	0	0	0	0	0	0	0	0	0	2	2
H.influenzae type b septicaemia*	1	1	0	0	0	0	0	0	1	1	2	2	4
H.influenzae type b (NOS)*	0 4	0 1	0 15	0 20	0 8	16	0 5	0 6	1 5	0 2	1 37	0 45	1 83
Hepatitis A*	4	10	228	237	669	827	255	404	41	72	1197	1551	2763
Hepatitis B: total* Hepatitis B: acute viral*	0	1	7	9	7	18	4	5	1	3	19	36	56
Hepatitis B: other*	4	9	221	228	662	809	251	399	40	69	1178	1515	2707
Hepatitis C: total*	6	11	289	308	936	1620	370	750	73	73	1675	2762	4452 ^(b)
Hepatitis C: acute viral*	0	0	3	9	13	15	1	0	0	0	17	24	41
Hepatitis C: other*	6	11	286	299	923	1605	369	750	73	73	1658	2738	4411 ^(b)
Hepatitis D*	1	0 0	0	4 3	1 2	4 0	1 0	4 2	0 0	0	3 2	12 5	15
Hepatitis E*	0	0	9	28	17	243	5	82	0	4	31	357	7 388
HIV infection* Haemolytic uraemic syndrome	1	3	1	1	0	243	2	1	1	1	5	6	11
Influenza: total*	122	143	172	144	219	118	132	120	130	114	775	639	1414
Influenza: Type A*	100	114	118	104	153	85	101	94	101	85	573	482	1055
Influenza: Type B*	21	26	43	32	49	26	26	18	19	21	158	123	281
Influenza: Type AB*	1	1	11	4	14	5	5	8	8	7	39	25	64
Influenza: Type (NOS)*	0	2 0	0	4 3	3 7	2 12	0	0 24	2 9	1 23	5 27	9 62	14 89
Legionnaires' disease: total*	0 0	0	0	0	1	4	11 5	24 5	9 3	23 6	27	62 15	24
Legionnaires' disease: L. longbeachae*	0	0	0	3	6	7	6	19	6	17	18	46	64
Legionnaires' disease: <i>L. pneumophila</i> * Legionnaires' disease: other*	Ő	Ő	ŏ	Ő	õ	1	Ő	0	õ	0	0	1	1
Leprosy	Ō	Ō	1	Ō	Ō	0	Ō	Ō	Ō	Ō	1	0	1
Leptospirosis*	0	1	2	7	4	7	3	10	1	0	10	25	35
Listeriosis*	0	1	0	0	0	1	7	3	9	4	16	9	25
Malaria*	8	13	44	46	20	26	8	34	2	2	82	121	204
Measles: total	0 0	0 0	1	1 0	1	2 2	0	0 0	0	0	2 2	3 2	5 4
Measles: laboratory confirmed* Measles: other	0	0	0	1	0	0	0	0	0	0	0	1	1
Meningococcal disease (invasive): total	15	28	19	32	15	9	4	8	10	1	63	78	141
Meningococcal disease: type B*	10	16	13	18	5	3	2	1	4	1	34	39	73
Meningococcal disease: type C*	1	1	0	4	4	3	0	2	0	0	5	10	15
Meningococcal disease: type W135*	0	0	0	1	1	0	0	1	5	0	6	2	8
Meningococcal disease: type Y*	1	0	0	0	0	0	1	0	1	0	3	0	3
Meningococcal disease: other	3 0	11 3	6 12	9 19	5 19	3 37	1 6	4 7	0 6	0 2	15 43	27 68	42 111
Mumps* Pertussis	133	135	461	326	1210	687	1275	802	417	347	3496	2297	5801
Pneumococcal disease: invasive*	73	66	17	21	35	80	55	81	105	107	285	355	643
Psittacosis*	0	1	7	2	19	12	20	27	9	24	55	66	121
Q fever*	0	0	6	12	13	42	11	47	5	6	35	107	142
Rubella: total*	0	2	0	0	4	1	1	0	1	0	6	3	10
Rubella*	0	2	0	0	4	1	1	0	1	0	6	3	10
Rubella: congenital*	0	0	0	0	0	0	0	0	0	0	0	0	0
Salmonellosis*##	248 17	318 23	267 14	354 12	258 27	225 14	163 9	144 13	104 0	90 4	1040 67	1131 66	2179 135
Shigellosis* Syphilis: total	4	23	30	45	113	278	9 54	182	46	84	247	592	845
Syphilis: infectious*+	0	0	4	22	7	156	5	43	40	4	16	225	242
Syphilis: congenital*	2	2	0 0	3	0	0	Ő	0	Õ	0	2	5	8
Syphilis: other*	2	1	26	20	106	122	49	139	46	80	229	362	595
Tetanus	0	0	0	0	0	0	0	0	0	1	0	1	1
Tuberculosis*	9	3	30	37	98	84	54	53	30	54	221	231	453
Typhoid*	0 0	0 0	6 5	14 4	3 0	2 1	2 3	1	0	0	11 9	17 7	28
Verotoxigenic Escherichia coli infections*	U	0	5	4	0	1	3	1	1	1	9	/	16

onset = the earlier of patient reported onset date, specimen date or date of notification

onset = the earlier of patient reported onset date, specimen date of date of notification
 * laboratory-confirmed cases only NOS = not otherwise specified
 + includes syphilis-primary, syphilis-secondary, syphilis < 1 yr duration and syphilis-newly acquired
 (a) = includes cases with unknown age and sex
 (b) = includes people who identify as transgender
 ## from 2005, all paratyphoid recorded as salmonellosis
 M = male F = female

DISEASE NOTIFICATIONS WHERE THE CASE HAD DIED AT THE TIME OF PUBLIC HEALTH FOLLOW-UP, BY YEAR OF ONSET OF ILLNESS,* NSW, 1991–2005

Conditions	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005
Creutzfeldt-Jakob disease*		tifiable u			1995	1990	1997	1990	1999	2000	2001	2002	2003	2004	6
	not not		inui Apr	1 2004	~	~	~	~	~			~	~	3	0
H.influenzae type b: total*	4	4	4	1	0	2	0	0	0	1	1	0	0	0	0
H.influenzae type b epiglottitis*	0	1	0	0	0	0	0	0	0	1	0	0	0	0	0
H.influenzae type b meningitis*	2	3	3	0	0	0	0	0	0	0	0	0	0	0	0
H.influenzae type b septicaemia*	0	0	0	1	0	2	0	0	0	0	0	0	0	0	0
H.influenzae type b infection (NOS)*	2	0	1	0	0	0	0	0	0	0	1	0	0	0	0
HIV*/AIDS infection (total) [£]	350	337	393	432	365	280	134	76	74	87	62	67	64	58	36
Cause AIDS	341	320	374	399	277	205	124	71	65	73	46	37	40	23	19
Cause Other	9	17	19	33	88	75	10	5	9	14	16	30	24	35	17
Haemolytic uraemic syndrome	not not	tifiable u	intil Dec	cember	1996		0	0	1	1	0	1	0	1	1
Listeriosis*	0	0	2	2	2	9	1	5	4	4	3	1	6	3	3
Measles (total)	1	2	0	0	0	0	0	0	0	0	0	0	0	0	0
Measles laboratory confirmed*	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Measles: other	1	2	0	0	0	0	0	0	0	0	0	0	0	0	0
Meningococcal disease (invasive): total	3	8	11	15	7	7	7	17	14	14	7	19	14	6	9
Meningococcal disease-Type B*	0	0	1	1	3	0	4	2	7	6	2	8	6	4	4
Meningococcal disease—Type C*	0	0	1	1	0	2	2	10	4	4	5	10	6	1	2
Meningococcal disease—Type W135*	0	0	0	0	0	0	0	0	1	0	0	0	0	0	2
Meningococcal disease—Type Y*	0	0	0	0	0	0	0	1	0	1	0	0	0	0	1
Meningococcal disease-other	3	8	9	13	4	5	1	4	2	3	0	1	2	1	0
Pertussis	0	0	0	0	2	2	3	1	1	2	0	0	1	1	1
Pneumococcal disease: invasive*	not not	ifiable ι	intil Dec	cember	2000						6	95	71	88	61
Tetanus	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
Tuberculosis*	10	26	31	25	23	16	21	25	29	40	33	39	22	28	21
Verotoxigenic Escherichia coli infections*	not not	tifiable u	intil Dec	cember	1996		0	0	0	0	0	0	0	0	1

year of onset = the earlier of patient reported onset date, specimen date or date of notification
 * laboratory-confirmed cases only NOS = not otherwise specified
 £ 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

THE INVESTIGATION OF AN OUTBREAK OF CRYPTOSPORIDIOSIS IN NEW SOUTH WALES IN 2005

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In April 2005 the Communicable Diseases Branch at the NSW Department of Health noticed an increase in the number of cases of cryptosporidiosis reported across NSW. This article describes how this outbreak of cryptosporidiosis was investigated and analysed.

Cryptosporidiosis is caused by the protozoan parasite *Cryptosporidium* and the symptoms include watery diarrhoea, abdominal cramping, loss of appetite, fever, vomiting and nausea.¹ The incubation period for cryptosporidiosis is one to 12 days. Treatment is supportive only; however, most healthy people tend to recover within two to 26 days.² An infected person will excrete infectious oocysts for several weeks after their symptoms resolve.²

Cryptosporidiosis is spread by the faecal-oral route. Transmission can be person-to-person, animal-to-person, waterborne and, in rare cases, foodborne¹. *Cryptosporidium* oocysts are small (4–6 μ m) and can survive under adverse environmental conditions, including chlorination.^{1,2} Outbreaks of cryptosporidiosis are usually associated with contaminated drinking water supplies and swimming pools.¹ To control *Cryptosporidium* in public swimming pools it is recommended that pools are 'superchlorinated' or 'shock-dosed' at least fortnightly.³

In 1996 cryptosporidiosis became notifiable by laboratories in NSW under the *NSW Public Health Act 1991*. Laboratories are required to notify public health units of positive cases of cryptosporidiosis; the public health units, in turn then enter information about the cases into the NSW Notifiable Diseases Database within three days of receiving this notification. The Communicable Diseases Branch uses the Notifiable Diseases Database to monitor the number of cases of cryptosporidiosis in NSW.

We investigated this outbreak using epidemiological, environmental and laboratory methods to determine the likely cause of the outbreak and develop public health response strategies.

METHODS

A case was defined as a person with laboratory-confirmed cryptosporidiosis notified between 1 May 2005 and 12 August 2005, with an onset date of illness after 31 March 2005. The Notifiable Diseases Database for NSW contained basic demographic information for all the cases of cryptosporidiosis notified during this period.

To investigate these cases further we designed a data collection form that included questions seeking additional demographic information about possible exposures during the 12 days before the onset of symptoms. Public health units were asked to contact the cases and interview them using this form, and to return the completed forms to the Communicable Diseases Branch. The data was entered into a central database, and the demographic characteristics and risk factors for disease were analysed.

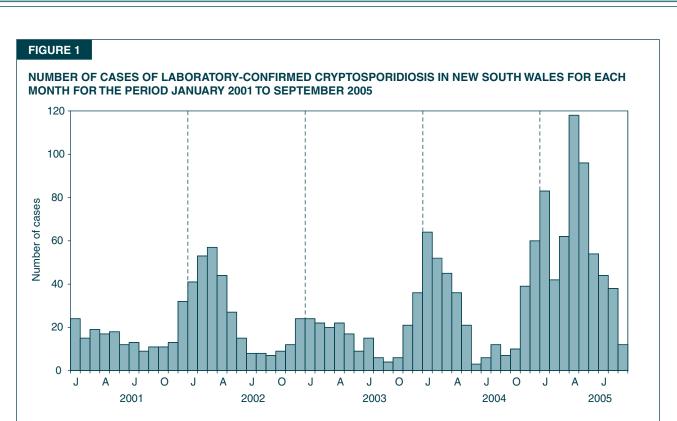
In June 2005 an environmental investigation was undertaken that involved testing swimming pool water for Cryptosporidium oocysts. Pools selected for testing were those where more than two cases had reporting swimming during their exposure period and where at least one of these had reported onset of illness within 10 days of swimming in the pool in the month of June 2005. Environmental health officers collected approximately 100 litres of water from each pool. The water was collected in 10 litre bladders from different locations around the perimeter of the pool and this sample was then sent to Sydney Water to identify and enumerate the number of Cryptosporidium oocysts. Environmental health officers also asked about superchlorination practices at the associated pools and reviewed pool disinfectant records for documentation of superchlorination practices.

We also reviewed laboratory stool testing procedures because of anecdotal reports that laboratories were increasing the number of stools being tested for *Cryptosporidium*. The laboratory investigation involved contacting a convenient sample of laboratories about changes in their stool testing procedures for *Cryptosporidium*. A private metropolitan laboratory, a public metropolitan laboratory and a public regional laboratory were contacted. Medical Benefits Scheme changes relevant to the laboratory diagnosis of cryptosporidiosis were also reviewed, and we also analysed Medicare data for item number 69336 (which pertains to stool tests for ova, cysts and parasites), by accessing the Medical Benefits Scheme health statistics on the Medicare Australia website.⁴

RESULTS

Epidemiological investigation

There were 254 cases of cryptosporidiosis notified from 1 May to 12 August 2005 with an onset date after 31 March 2005. Figure 1 displays the number of cases of cryptosporidiosis reported in NSW from January 2001 to September 2005. A rise in notifications can be seen each summer. In March 2005 the number of cases continued to increase beyond the summer months, and peaked in April 2005. Notification rates were more than three times higher than expected for that time of the year.





CHARACTERISTICS AND REPORTED EXPOSURES OF THE INTERVIEWED CASES OF CRYPTOSPORIDIOSIS NOTIFIED IN NEW SOUTH WALES BETWEEN 1 MAY AND 12 AUGUST 2005, WITH ONSET DATES AFTER 31 MARCH 2005 (N=178)

Characteristic or exposure	Number of cases with characteristic / Total number of responses*	Percentage		
Demographic characteristics:				
Female	90/178	51		
Resided in Sydney metropolitan area	151/178	85		
Reported exposures:				
Contact with person with diarrhoea	56/165	34		
Attended childcare centre	50/175	29		
Swam anywhere	95/169	56		
Swam in public pool	89/169	53		
Drank town water supply	142/154	92		
Contact with animals	79/174	45		

*The total number of responses varies due to incomplete surveys

TABLE 2

CRYPTOSPORIDIUM OOCYST LEVELS IN FIVE POOLS THAT HAD MORE THAN TWO REPORTED CASES OF CRYPTOSPORIDIOSIS, SYDNEY, JUNE 2005

Pool	Locality within Sydney	Date tested	Number of cases in 6 weeks before testing	Cryptosporidium oocyst count / Litres of water tested
А	South-West	10 June	3	1/80
В	Western	17 June	>5	636/107
С	South-West	20 June	>3	907/100
D	North	23 June	>5	Large pool: 1/100 Small pool: 0/100

Public health unit staff were able to contact 178 of the 254 cases to interview them (or, in the case of children, their families) about possible sources of infection during the exposure period, a follow-up rate of 70 per cent. The median age of the cases was 5.1 years, with a range from 1 month to 57 years. Demographic characteristics and risk factor information are provided for these cases in Table 1.

Eighty-nine cases (53 per cent) reported swimming in a public pool in the 12 days before the onset of symptoms. These cases reported swimming at a total of 35 pools. Of these pools, 22 were named by one case, six were named by two cases, three were named by between three and five cases, and four were named by more than five cases.

Environmental investigation

Throughout June we identified five swimming pools (at four swimming centres) with more than two associated cases of cryptosporidiosis, and where at least one case had swum in that pool in June within 10 days of onset of illness. Water samples were collected from these pools and tested for the presence of *Cryptosporidium* oocysts (Table 2).

Public health unit staff contacted these swimming centres to discuss superchlorination practices and review pool disinfectant records. The operators of pools A, B and D reported that the pools were superchlorinated at least fortnightly. The operator of pool C, which had the highest *Cryptosporidium* oocyst count, reported the pool had not been superchlorinated for more than six months. There were incomplete superchlorination records at pool A, and there were no records at pools B, C and D.

Laboratory stool testing investigation

Investigation of changes to Medical Benefits Scheme item numbers relevant to *Cryptosporidium* stool testing revealed several alterations to the wording for item number 69336 in recent years, with a significant change occurring in May 2003. As a result of this change laboratories could charge against this item using commercial rapid antigen *Cryptosporidium* detection tests where a faecal concentration was also performed. Previously, private laboratories reported that stools were not necessarily tested for *Cryptosporidium* unless specifically requested by the doctor because of the laborious nature of detection techniques.

Three NSW diagnostic laboratories were contacted about changes in their stool testing for cryptosporidiosis. The two public laboratories reported no change in the proportion of stools being tested for the presence of *Cryptosporidium*. The third laboratory, a private laboratory, reported an increased rate of testing for *Cryptosporidium* as of November 2004, from approximately 10 per cent to 100 per cent of stools. This laboratory notified 56 per cent of the interviewed cases in the period between May and August 2005.

Data obtained from the Medicare Australia website showed that the rates of diagnostic services (per 100,000

population) for the Medical Benefits Scheme item number 69336 in NSW were 760 in 2001–02, 753 in 2002–03, 1004 in 2003–04 and 1013 in 2004–05.

Public health intervention

In response to the outbreak, NSW Health released a media statement on 10 June 2005 highlighting the increased numbers of cryptosporidiosis cases in the community, and advising swimmers with a diarrhoeal illness to stay out of pools for at least a week after their symptoms resolved. The media release also advised parents on how to prevent *Cryptosporidium* in swimming pools (for example, by putting waterproof tight fitting pants over young children's swimmers and changing nappies in bathrooms rather than at the poolside).

Public health unit staff contacted swimming centres associated with two or more cases to advise on infection control practices (particularly in toileting areas and near change tables) and disinfectant practices, including superchlorination and shock-dosing. Swimming centres were provided with, and asked to use, 'Keep the cryptobug out of our pool' posters, signage and fact-sheets. Two swimming centres introduced the practice of routinely asking parents if their children had recent or current diarrhoea so that they could be excluded from swimming classes. One public health unit reported contacting local general practitioners to request that they advise the parents of children with diarrhoea that they should not swim in a pool until at least seven days after their symptoms had resolved.

DISCUSSION

Data from the interviewed cases as well as the pool testing results supports the hypothesis that this outbreak was, at least in part, associated with swimming in contaminated pools.

In NSW, cryptosporidiosis rates tend to rise during the summer months. This outbreak occurred during autumn and winter (April to August 2005), with the majority of cases resident in the Sydney metropolitan area. Of the interviewed cases, 53 per cent reported swimming in a public pool in the 12-day period before the onset of illness. To determine whether this exposure was likely to be associated with illness, we compared our findings with the results of a case-control study by Puech et al (2001)⁵, which investigated a cryptosporidiosis outbreak in NSW in the summer of 1997–98. In the study by Puech et al, 59 per cent of cases reported swimming in a public pool in the exposure period compared with a much lower rate of swimming for controls (38 per cent).

While there are inherent limitations in comparing findings from our investigation with those of Puech et al's study, the results of Puech et al's study provides some evidence that the rate of swimming in a public pool reported by cases at the time of our investigation would have been higher than might be expected in a control group (especially as this outbreak occurred during autumn and winter). In Puech et al's study, the reported rates of having attended a childcare centre among controls were similar to the rates of cases in our study (29 per cent in both); however, the cases in our study reported higher rates of having contact with a person with a diarrhoeal illness compared to the controls in Puech et al's study (34 per cent vs 25 per cent respectively). This suggests that person-to-person spread may also have played a role in this outbreak.

Laboratory policy and practice can potentially influence notification rates for communicable diseases. There was some evidence to suggest that changes in private laboratory practices may have contributed to the increased number of notifications. While this might explain an overall increase in the rates of cryptosporidiosis in NSW compared with previous years, it does not account for the unusual peak of cases in April to August. Alternatively, increased awareness of cryptosporidiosis within the general population, or increased testing by doctors, may have potentially increased notification rates of cryptosporidiosis. However there was no reason to believe that these factors were contributing to this outbreak.

There are several limitations to this study. Firstly, it is descriptive and therefore cannot provide robust evidence as to causation. However, comparisons of the reported exposures in our cases and those of controls interviewed for a similar outbreak, combined with the findings from the environmental investigation, provides a useful insight into the risk of illness associated with pool swimming in this outbreak.

Secondly, the response rate among cases was 70 per cent. There is no evidence to suggest, however, that the cases we interviewed were significantly different from those who were not. For example, the median age of cases for whom interview information was available was 5.06 years compared with 5.04 years for the entire sample. Eighty-five per cent of interviewed cases were from a metropolitan area compared with 81 per cent for the entire sample. Thirdly, measurement error is likely to have affected the results of pool testing for *Cryptosporidium* oocysts due to the time-lag between reported exposures and pool sampling.

Finally, we were able to contact only three laboratories about changes in their stool testing procedures for cryptosporidiosis. While the private laboratory that accounted for more than 50 per cent of notified cases for this investigation reported a recent increase in their rate of stool testing for cryptosporidiosis, we were not able to obtain data to determine the number of stools tested for cryptosporidiosis from this laboratory in 2005 and previous years, or the number of cryptosporidiosis cases notified from this laboratory over previous years.

Health professionals should routinely provide information to all persons with cryptosporidiosis about the prevention of its spread. The NSW Health fact sheet for cryptosporidiosis specifically advises against swimming while symptomatic and for at least one week afterwards.⁶

To prevent pools being reinfected with *Cryptosporidium* it is important that pool operators comply with the NSW Health *Protocol for minimising the risk of Cryptosporidium contamination in public swimming pools and spa pools.*³ Pools which have large 'bather loads', especially of young children, should consider increasing the frequency of superchlorination. Other strategies to minimise reinfection in pools includes the use of cryptosporidiosis signage at pools, and asking patrons about recent symptoms of diarrhoea when entering the swimming complex.

ACKNOWLEDGEMENTS

We would like to acknowledge the assistance of the public health unit staff who undertook interviews with cases, the environmental health officers who investigated pool premises, the NSW Health Water Unit staff who provided timely advice and resources, and Jan Lanser and others who provided information for the laboratory investigation.

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MANAGING A CLUSTER OF CRYPTOSPORIDIOSIS ASSOCIATED WITH A PUBLIC SWIMMING POOL

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Between 10 May and 20 June 2005, Sydney West Centre for Population Health (SWCPH) was notified of 29 cases of cryptosporidiosis. SWCPH staff interviewed these cases about their potential sources of exposure to infection and found that 11 (38 per cent) swam at the same indoor heated public pool. The onset of illness in these 11 cases ranged from late April to early June. Nine of these cases were children, six of whom were aged three years and under. Six of the eleven cases also reported other potential sources of infection, including contact with relatives who had diarrhoeal illness, contact with pets, and attendance at a childcare centre or play group.

The pool was used almost exclusively for learn-to-swim classes, which included non-toilet-trained infants. These infants are required to wear swimmers with waterproof tight fitting pants over them when they enter the pool. The pool operator reported using liquid stabilised chlorine dioxide in combination with super-chlorination on a regular basis in accordance with current NSW guidelines (see: http:// internal.health.nsw.gov.au/public-health/ehb/general/pools/ cryptopools.pdf).

Council staff checked the pool's testing logbook fortnightly, and had found no obvious problems in the management of

the pool in the weeks before this cluster of cryptosporidiosis was identified.

In response to the outbreak, SWCPH staff contacted the pool manager and recommended that the pool be superchlorinated with a 'shock' dose of liquid stabilised chlorine dioxide on the evening of 24 May and with liquid sodium hypochlorite on the following evening. Liquid stabilised chlorine dioxide, which has recently become available, can 'clean' a pool in only two to four hours, whereas sodium hypochlorite, the traditional treatment, takes around 14 hours. Council staff re-tested the pool on 25 and 26 May and found that it met the requirements for an indoor heated swimming pool prescribed by the NSW guidelines.

However, following the identification of further cases, SWCPH staff arranged for the pool's water to be tested for *Cryptosporidium* oocysts on 17 June. The sample tested positive (636 oocysts in 107 litres). In response the pool was super-chlorinated on 17 June and again on 20 June. SWCPH staff also recommended that the pool management implement additional strategies to prevent contamination with *Cryptosporidium*. These included improved procedures for asking parents about diarrhoeal illness in themselves and their children and increasing the period of exclusion for individuals with diarrhoea from one week to two. No further cases of cryptosporidiosis have been associated with this pool.

GENOTYPING OF *MYCOBACTERIUM TUBERCULOSIS* IN NEW SOUTH WALES: RESULTS FROM 18 MONTHS OF A STATEWIDE TRIAL

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Several molecular typing methods are available to assist public health practitioners in identifying clusters of recently acquired tuberculosis cases.^{1,2} Molecular typing or fingerprinting investigates variations in microbial populations, defines specific clones and identifies outbreaks by matching molecular fingerprints of epidemiologically linked isolates. The combination of two or more methods, with different preselected genomic loci in the *Mycobacterium tuberculosis* genome, have been used to identify and track outbreaks, define high-risk groups and target prevention strategies.^{2,3,4} Table 1 compares three current typing methods.

In contrast to epidemiological methods, the use of genotypic methods to define clusters is controversial. Genotypedefined clusters are used to calculate the transmission index or average number of secondary cases from a single source case. These clusters appear to result from recently transmitted infection with rapid progression to clinical disease.⁵ Routine genotyping has shown that transmission of tuberculosis occurs more readily than previously thought,⁶ with substantial proportions (28–72 per cent) of urban cases occurring in clusters.^{78,9} By contrast, conventional contact tracing may identify only 10 per cent of clustered cases.⁷ DNA fingerprinting has demonstrated the existence and worldwide transmission of families of genetically related strains and local dissemination of successful clones.

The Centre for Infectious Diseases and Microbiology (CIDM) at Westmead has been genotyping all *M. tuberculosis* complex (including *M. tuberculosis, M. africanum, M. bovis, M. bovis* bacillus Calmette-Guerin (BCG), the rarely isolated species *M. microti, M. canettii,* and the newly described seal pathogen, *M. pinnipedii)* isolates from NSW since 2003. This report is a review of the results for the period December 2003 to May 2005.

METHODS

Isolates

All 420 *M. tuberculosis* complex isolates referred to the NSW Mycobacterium Reference Laboratory, CIDM, between December 2003 and May 2005, are included.

Molecular typing methods

All isolates were tested by mycobacterial interspersed repetitive units (MIRU) typing and spoligotyping, according to published methods.^{10,11} Clinical isolates with matching MIRU and spoligotype numerical codes were then subjected to IS6110 restriction fragment length polymorphism (RFLP) analysis.¹² Quality control strains of *M.tuberculosis* and *M.bovis* BCG were used to monitor the performance of the genotyping techniques.

Cluster analysis

Comparison of IS6110 RFLP gel profiles was performed using the Bionumerics Edition 3.0 package (Applied Maths, Koutrai, Belgium) using standard methods. A cluster was

TABLE 1

Name of method	Genomic target	Method	Result format	Turnaround- time	Discriminatory power	Cost
Spoligotyping	Direct repeat region	Single PCR; dot-blot hybridisation to detect presence/ absence of 43 spacer sequences	15 digit code	Days; can be done directly on specimens	High sensitivity; low specificity	Relatively low
Mycobacterial interspersed repetitive units (MIRU) typing	12 loci (can be more or less)	Multiple PCR; amplicons size indicates number of repeat sequences at each locus	12 digit code	Days; can be done directly on specimens	Depends on number of loci targeted; 12 loci high sensitivity; moderate specificity	Medium; depends on number of loc targeted
IS6110 restriction fragment length polymorphism (RFLP) analysis	IS6110 (0-20 copies)	DNA cut with restriction enzyme; fragments separated on gel; probed for presence of IS6110	Image – number/size of fragments containing IS	Weeks (requires lots of high quality DNA)	Gold standard – high specificity. Not suitable for strains with <5 copies of IS6110	High

defined as a group of isolates that were indistinguishable by all three methods. Laboratory cross-contamination, as a possible source of clustering, was investigated by checking the time of the processing in the laboratory and the clinical history of patients. The rate of recent transmission (RRT)¹³ was calculated as:

RRT (per cent) = (No. of isolates clustered – No. of clusters)/Total isolates typed x100

RESULTS

Spoligotyping identified seven of 420 (1.7 per cent) isolates as members of *M. tuberculosis* complex other than *M. tuberculosis*, namely *Mycobacterium bovis* (2 isolates), *M.bovis* BCG (3 isolates), *M. canettii* (1 isolate), and *M.caprae* (1 isolate).

Molecular diversity

Of 413 sequential isolates of *M.tuberculosis*, 273 (66 per cent) and 176 isolates (43 per cent) were individually grouped by spoligotyping and MIRU typing, respectively,

and 105 isolates (25 per cent) were clustered by both methods. Of 273 isolates grouped by spoligotyping, 71 (26 per cent) belonged to the Beijing family of *M. tuberculosis* strains. The numbers of isolates belonging to other recognised groups are shown in Figure 1.

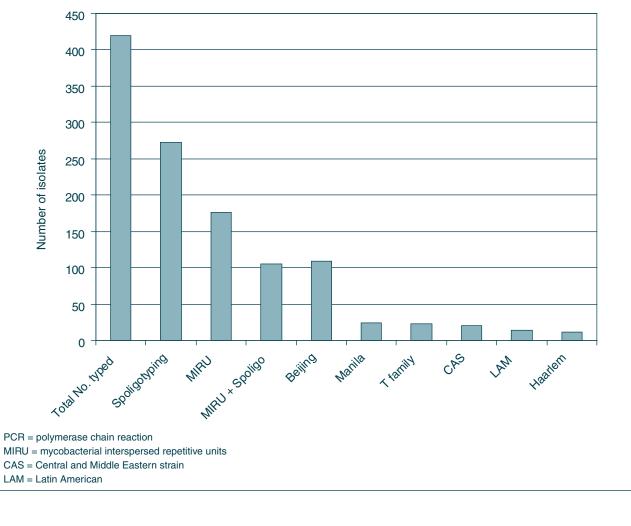
Clusters

Eight clusters, involving a total of 20 isolates (4.8 per cent), were identified, based on all three typing methods. Five clusters contained only two isolates, two contained three isolates and the other contained four isolates. Three stored isolates were later identified as belonging to cluster 1.

Cluster 1 comprised six isolates: three collected during the study period and a further three isolates collected outside the study period. They were linked, but not initially recognised as being epidemiologically related, by geographic proximity and risk factors. The index case was diagnosed and treated in 2000. A sixth case was identified by routine genotyping later in the study period. The RFLP pattern for this cluster consisted of 12 bands.

FIGURE 1

CLUSTERED ISOLATES BASED ON TWO PCR-BASED TYPING METHODS AND MAJOR *M. TUBERCULOSIS* FAMILIES IDENTIFIED BY SPOLIGOTYPING



Clusters two, three and four consisted of isolates from patients who had recently migrated from the Philippines (four cases), the Sudan (two cases) and the Indian subcontinent (two cases), respectively. There were no identifiable links between patients within clusters; the patients' infections were probably independently acquired in their countries of origin.

Cluster five consisted of two isolates from patients who resided in different Australian states and had no obvious epidemiological links (but warrant further investigation).

Clusters six, seven and eight represented probable crosscontamination. In all three clusters there was one isolate from a patient with typical smear-positive tuberculosis; the others were from patients in whom the diagnosis of tuberculosis was considered unlikely. Clusters six and seven comprised two and three isolates respectively, referred for confirmatory identification. Isolates in both clusters were recovered from specimens from different patients, processed in the same laboratories at the same time. The two isolates in cluster eight were recovered from patients who had attended the same clinic for bronchoscopy two weeks apart; the same bronchoscope was used for both procedures.

Only patients from clusters one and five were included in the calculation of the rate of recent transmission (RRT), which was calculated as 1.4 per cent (Table 2).

DISCUSSION

Our results highlight the diversity of *M. tuberculosis* strains involved in tuberculosis infections in this country, most of these infections being acquired elsewhere. The most prominent strains identified during the study by spoligotyping belong to the W-Beijing family (more than one quarter of all isolates examined), which was first described in China and neighbouring countries in 1995¹⁴ and has since spread to many parts of the world, especially Asia and Russia.^{2,14,15,16} They are highly transmissible

and often found predominantly in younger patients and they have an increased tendency to develop multidrugresistance.^{15,16} There is some evidence that BCG vaccination is less effective against Beijing genotype strains than others.¹⁷ The high proportion of Beijing genotype strains reflects the migration patterns into NSW.

The low level of clustering of *M.tuberculosis* isolates in this study confirms that recent transmission of tuberculosis in NSW is uncommon. Several clusters may reflect reactivation of latent tuberculosis infections in migrants from high incidence countries where M. tuberculosis strains are more homogenous.^{3,18} However, the possibility of recent transmission from direct contact, for example in a refugee camp or detention centre before arrival in Australia, cannot always be excluded. The rate of recent transmission (1.4 per cent) in this study is lower than that reported from other low-incidence countries (Table 2). However, these findings should be interpreted with caution. Studies of short duration (i.e. less than two years) may significantly underestimate the level of clustering because of the long incubation period of tuberculosis.^{2,20,21} Cluster 1, in this study, was identified because of genotyping of more recent isolates several years after the first three cases had presented. Cluster size can be significantly underestimated unless a high proportion of the total isolates from a population over a significant period (usually at least 3 years) are genotyped.²²

There is a growing body of evidence to support the role of *M. tuberculosis* genotyping in the detection and tracking of outbreaks of infection.^{4,6,18} Increased migration from high-prevalence areas increases the risk of spread of multidrug-resistant *M. tuberculosis* and the need for earlier detection of outbreaks.²³ Clustering reflects the efficiency of therapy, the interval between disease onset and the start of treatment and the regional dominance of more successful strains of *M. tuberculosis*.²² A better knowledge of expanding clones, such as the Beijing strain, is urgently needed in order to define better control measures.^{4,23,24}

TABLE 2

COMPARISON OF FINDINGS FROM NSW WITH THREE RECENT INTERNATIONAL STUDIES THAT HAVE USED MOLECULAR TYPING OF *M.TUBERCULOSIS* TO DESCRIBE THE EPIDEMIOLOGY OF THE DISEASE

	London, UK ¹³	Denmark ¹⁸	Italy ¹⁹	NSW (this study)
Number of isolates genotyped	57	1549	248	420
Methods used	IS6110 RFLP, spoligotyping	IS6110 RFLP	IS6110 RFLP, spoligotyping	IS6110 RFLP, spoligotyping, MIRUs
Duration of study	3 years	5 years	1 year	1.5 years
Rate of clustering %	15.8	49	33	4.9
Recent transmission rate %	8.8	57*	15	1.4
Percentage of tuberculosis due to Beijing strain	Not reported	Not reported	2.8	25.9

RFLP = restriction fragment length polymorphism; MIRU = mycobacterial interspersed repetitive units.

*Active transmission among native Danes reported only (two strains were responsible for 40% of all clustered cases among native Danes; the sample included two large clusters among HIV positive drug users).

No single typing method is ideal. PCR-based methods are rapid and relatively inexpensive; when combined, they can quickly exclude clustering in three quarters of cases, significantly reducing the need for IS6110 RFLP typing. Patients who need additional follow-up can be identified more rapidly, secondary cases treated more quickly and new cases prevented. Although IS6110 is regarded as the 'gold standard' it often requires several weeks' culture of M. tuberculosis to obtain adequate DNA and inter-laboratory comparison of results can be difficult. Spoligotyping alone is relatively non-discriminatory but provides valuable data about the prevalence of various M. tuberculosis families and can rapidly differentiate sub-species within the M. tuberculosis complex (for example M.bovis, M.bovis BCG, and *M.canetii*), which can otherwise only be identified by time-consuming biochemical tests. As far as we know, this is the first time that M. canetti and M. caprae have been identified in Australia.²⁵ The combination of three methods, as used in NSW, is probably the most cost-effective approach in the long term if clustered cases are rapidly identified and investigated, but more detailed analysis of data, over a longer period, is required.

These data will be used in future as a baseline for real-time monitoring of transmission dynamics of tuberculosis cases in NSW. They will contribute to a national genotyping project (based on MIRU typing only, initially), which may identify links between patients travelling interstate (such as those in cluster five). A project is currently in progress in NSW to link the genotyping database with tuberculosis case notification data. A comprehensive national tuberculosis genotyping network linked to the National Notifiable Diseases Surveillance System would provide continuous monitoring of transmission trends and allow identification of widespread outbreaks.

ACKNOWLEDGEMENTS

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BUG BREAKFAST* IN THE BULLETIN

CHLAMYDIA, GONORRHOEA AND SYPHILIS

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Chlamydia, gonorrhoea and syphilis are the three most commonly reported sexually transmissible bacterial infections in NSW.¹ These sexually transmissible infections (STIs) are important, not only because of their burden of disease and long-term complications, but also because they increase the risk of HIV transmission.² This Bug Breakfast provided an overview of these infections and outlined the NSW Sexually Transmissible Infections Strategy for 2006–2009.

CHLAMYDIA

Chlamydia is a common bacterial sexually transmissible infection worldwide, causing more than 43 million new chlamydial infections each year.³ Genital chlamydia is caused by *Chlamydia trachomatis* serovars D-K. Chlamydiae are obligate intracellular parasites, and one of the smallest of all bacteria. Transmission occurs most commonly during sexual contact, with vertical transmission occurring during childbirth. The incubation period is usually seven to 14 days, and infection is commonly asymptomatic. Recurrent infections are common.⁴

Genital chlamydia is one of the most frequently reported notifiable conditions in Australia, with 35,189 diagnoses in 2004.⁵ The population rate of diagnosis has more than doubled, from 91.4 cases per 100,000 in 2000 to 186.1 cases per 100,000 in 2004.⁵ This rise is mostly in young heterosexuals, and is thought to be due to a combination of: an increase in the rate of testing of both symptomatic and asymptomatic people; an increase in the sensitivity of testing since the introduction of nucleic acid amplification tests; and a real rise in the rate of infection.^{6, 7, 8} The long-term complications of infection are particularly severe in women, and include pelvic inflammatory disease, infertility, ectopic pregnancy, and chronic pelvic pain.

GONORRHOEA

Gonorrhoea is caused by the gram-negative bacterium *Neisseria gonorrhoeae*. The incubation period of gonorrhoea is usually two to seven days. The incidence of gonorrhoea in Australia increased from 31.4 cases per 100,000 in 2000 to 37.0 cases per 100,000 in 2004.⁵ The rates of gonorrhoeal infection are higher among Aboriginal people compared with non-Aboriginal people, and are also higher in the Northern Territory.⁵

The symptoms of gonorrhoea overlap with those of genital chlamydia.⁴ Apart from urethritis, infection is usually asymptomatic, and re-infection with gonorrhoea is common. Complications include pelvic inflammatory disease and infertility and, rarely, disseminated gonococcal infection.

SYPHILIS

Syphilis is caused by the spirochaete bacterium, *Treponema pallidum* subs. *Pallidum*. Transmission of syphilis occurs through direct contact with exudative lesions, and through vertical transmission from mother to child in utero. The incubation period is usually between 10 and 90 days, with an average of three weeks. Syphilis infection usually produces a primary lesion (a chancre); however, infection may be asymptomatic.⁹ Primary infection is followed by secondary and then latent or tertiary syphilis. Syphilis infection can have severe long-term complications if left untreated.

In NSW, the rate of diagnosis of syphilis has almost doubled between 2000 and 2004, with the increased rate almost completely confined to homosexual men, particularly those from South-East Sydney and Central Sydney who were HIV positive, highly sexually active, or recreational drug users.^{5,9}

Management of infection with chlamydia, gonorrhoea or syphilis includes patient education, the provision of appropriate antibiotic therapy, and contact tracing.

NSW SEXUALLY TRANSMITTED INFECTIONS STRATEGY 2006–2009:

NSW Health has recently released its first Sexually Transmissible Infections Strategy.¹⁰This strategy emphasises STIs that have: significant morbidity; are associated with poorer long-term health outcomes such as infertility; facilitate the transmission of other infections such as HIV; are disproportionately prevalent within vulnerable populations; and are likely to be amenable to prevention and control efforts. Therefore the strategy gives particular priority to chlamydia, gonorrhoea and syphilis.

^{*}Bug Breakfast is the name given to a monthly series of hour-long breakfast seminars on communicable diseases delivered by the NSW Department of Health's Division of Population Health.

The strategy identifies priority population groups including:

- Aboriginal people
- gay and other homosexually active men
- heterosexuals with recent partner change
- people who inject drugs
- people with HIV/AIDS
- sex workers
- young people.

The Strategy aims to increase condom use and reduce STI transmission rates among all priority populations. Two specific targets have also been set in relation to syphilis for 2009, on the advice of organisations working with key communities. These are to:

- eliminate syphilis transmission within Aboriginal communities and
- reduce rates of syphilis among gay and other homosexually active men by 50 per cent.

CONCLUSION

NSW is experiencing rising rates of STIs, with increased incidence of chlamydia primarily in young heterosexuals, and gonorrhoea and infectious syphilis primarily in homosexual men. The NSW Sexually Transmissible Infections Strategy 2006–2009 aims to reduce transmission and associated morbidities of these sexually transmissible infections.

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COMMUNICABLE DISEASES REPORT, NEW SOUTH WALES, FOR MARCH AND APRIL 2006

For updated information, including data and facts on specific diseases, visit www.health.nsw.gov.au and click on **Infectious Diseases.**

TRENDS

Tables 2 and 3 and Figure 2 show reports of communicable diseases received through to the end of March and April 2006 for each area health service in NSW.

MEASLES RETURNS

Between mid-March and the end of April 2006, 38 confirmed cases of measles were reported in NSW. In comparison there were no cases reported between April 2005 and March 2006. Prior to 1966, nearly all children in NSW were at some point infected with wild measles, but the introduction of immunisation has dramatically reduced the incidence of this disease. The last large-scale outbreak of measles in NSW occurred in 1993, when 2348 cases were reported.1 Since then the incidence of measles in NSW has fallen substantially. This decline has been assisted by the National Measles Control Campaign of 1998, which included mass vaccination of children in primary schools.² In September 1999 local transmission was probably interrupted for the first time.³ Since 2002, between five and 18 cases have been reported annually in NSW. Here we report the characteristics of the cases that have occurred up to the end of April 2006. These cases appear to be associated with two distinct outbreaks (Figure 1).

Characteristics of the measles cases

The ages of the 38 measles patients ranged from 10 months to 57 years, with over three quarters aged under 15 years. Thirty-four were NSW residents and four were visitors who either contracted measles or were diagnosed with measles while in NSW. At least two thirds of the patients had not been immunised (Table 1). Only four of the 15 children aged between 12 months and four years were appropriately immunised for their age (i.e. had received one dose of the measles, mumps and rubella vaccine (MMR)).

Outbreak 1

Three cases, and a further eight secondary cases, were linked to a common exposure at the Emergency Department of a hospital on March 1. Despite a search of medical records, however, a definite source case has not been identified. Because no measles case had been reported in the preceding 10 months in NSW, it is hypothesised that the source of this outbreak was an unidentified sick traveller.

Outbreak 2

Twenty of the cases with an onset of illness from mid-April were associated with a national tour of a spiritual leader. A further 18 cases had been reported in other states to the end of April. It is likely that some members of the tour group were infected with measles prior to their arrival in Australia. Low immunisation rates in the families of some of these cases, and extensive travel within NSW by some cases whilst infectious prior to their diagnosis, suggests that secondary and tertiary cases are likely to present in coming weeks.

Unlinked cases

A further seven of the 38 NSW cases have not been linked to either of these two outbreaks, suggesting that measles was spreading through parts of the community in April.

Interventions

In response to these outbreaks, NSW Health issued several media releases and communicated directly with general practitioners. This may have resulted in an increased clinical awareness about measles and testing for measles by health professionals, leading to improved identification of cases.

Measles is a highly preventable disease. Clinicians or health care managers should:

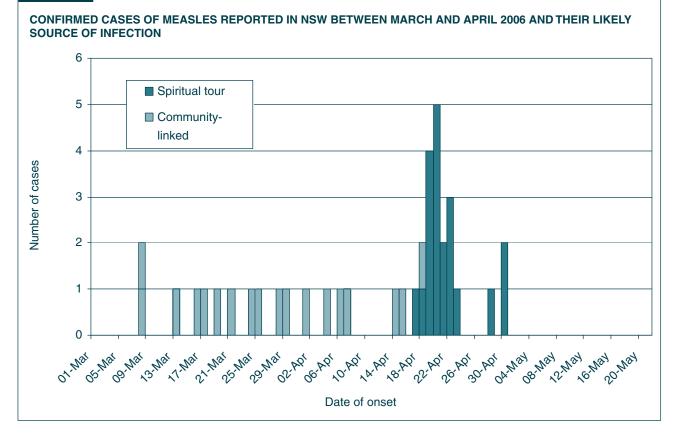
• ensure that all staff are immune to measles. Two doses of MMR vaccine are recommended for staff born during

TABLE 1

THE AGE, IMMUNISATION STATUS AND PLACE OF RESIDENCE OF MEASLES CASES REPORTED IN NSW BETWEEN MARCH AND APRIL 2006 (N=38).

Characteristic	Cas	ses
	n	%
Age group (years)		
<1	3	8
1-4	15	39
5-14	11	29
15-40	7	18
>40	2	5
Immunisation status against measles		
(MMR dose received)		
None	25	66
One	4	11
Two	0	0
Probably immunised	2	5
Unknown	7	18
Place of residence		
NSW		
Northern Sydney / Central Coast AHS*	8	21
South Eastern Sydney / Illawarra AHS*	4	11
Sydney South West AHS*	8	21
Sydney West AHS*	14	37
Other states		
Tasmania	1	3
Queensland	1	3
Overseas		
California	2	5
*AHS =Area Health Service		

FIGURE 1



or after 1966 unless they have documented evidence of immunity

- maintain a high index of suspicion for cases
- prevent transmission by ensuring that suspected cases (people presenting with fever, cough, coryza, conjunctivitis or rash) are shown immediately to a separate room, and do not wait in the general waiting room
- for suspected cases, notify the local public health unit, and collect diagnostic specimens, including a nose/ throat swab or aspirate on a viral transport swab and a first pass urine sample for measles immunofluorescence (and, if negative, a polymerase chain reaction test) and culture, and serum for measles IgM
- ensure that cases remain in isolation until four days after the onset of their rash.

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KERATOCONJUNCTIVITIS IN THE GREATER SOUTHERN AREA HEALTH SERVICE

In March 2006 the Greater Southern Public Health Unit (GSPHU) received a report of a number of cases of keratoconjunctivitis in a regional centre. A subsequent investigation by GSPHU and the Communicable Diseases Branch of the NSW Department of Health identified 66 patients with this condition, 47 of whom were diagnosed with viral conjunctivitis at a local eye clinic. Adenovirus was identified in three of four eye swabs taken from patients. Secondary cases among household members have been reported by 11 cases. Some cases described a severe, painful illness that incapacitated them for up to three weeks.

Keratoconjunctivitis is an acute viral disease of the eye. Symptoms include a sore and itchy red eye, with swollen lids, photophobia, a clear or yellow discharge that can make the lids stick together (especially on waking), blurred vision, and sometimes fever, headache and tiredness. It is typically caused by an adenovirus, and is transmitted by direct contact. It is commonly spread between household members as it is highly contagious. The incubation period is usually between five days and two weeks, and patients are infectious from a day or two before until about two weeks after the onset of their symptoms.

There is no specific treatment for viral conjunctivitis, so prevention is key. Patients should:

- stay out of school until symptoms have resolved or until they are cleared by a doctor
- avoid touching their eyes
- if they touch their eyes, wash their hands thoroughly with soap and running water
- avoid touching other people unless their hands are freshly washed
- throw away or carefully wash (in hot water and detergent) items that touch their eyes
- not share eye makeup or other items used on the eyes (ie, towels, tissues, eye drops, eye medications)
- use a separate towel and face cloth for each member of the household
- cover their mouth and nose when coughing or sneezing
- use disposable tissues to blow their nose, sneeze or cough
- if visiting a doctor or clinic, explain that they have viral conjunctivitis, so the clinic can implement measures to prevent spread of infection.

To identify the extent and factors contributing to the outbreak, the GSPHU and the Communicable Diseases Branch are conducting a case-control study.

A fact sheet on viral conjunctivitis can be found at: www. health.nsw.gov.au/infect/pdf/viral_conjunctivitis_cdfs. pdf.

LEGIONNAIRES' DISEASE IN NORTHERN SYDNEY

The Northern Sydney/Central Coast Public Health Unit (NSCCPHU) has reported an outbreak of Legionnaires' disease in six patients whose common exposure was visiting the Chatswood area. All patients were diagnosed with infection due to *Legionella pneumophila* serogroup 1 based on positive urinary antigen tests. This infection has previously been associated with exposure to contaminated aerosolized water. Possible sources include aerosols emitted from air-conditioning cooling towers that are often located on top of large buildings.

NSCCPHU staff interviewed the cases about possible exposures during the incubation periods of their illness (ie, two-ten days before onset). Five cases reported the onset of their illness in early March, and all but one reported visiting the Chatswood central business district (CBD) in either late February or the first few days of March. The remaining case reported a slightly later onset date (10 March), and was living in a respite care facility about 1 km from the Chatswood CBD but had not visited the Chatswood CBD. After interviewing the first two cases, NSCCPHU staff initiated a series of actions that included:

- a review of the cooling towers in the areas visited by the cases
- active surveillance to identify other possible cases. This included alerting Emergency Departments, laboratories and general practitioners in the area, and asking them to report other possible cases. Public Health Units in other areas were advised about the outbreak.
- asking the local council to contact cooling tower operators to remind them about the need to properly maintain all cooling towers according to the requirements of the NSW Public Health Act (1991)
- issuing media releases to inform the public about the outbreak.

Over a hundred cooling towers in the Chatswood area were inspected and the maintenance practices reviewed. The main objective was to ensure that there was no ongoing risk to the community. The cooling towers were tested for the presence of *Legionella* bacteria as a quality measure; however, a likely source of infection was not identified. NSCCPHU staff also reviewed possible sources of infection in the respite care facility. Because cooling towers are cleaned and maintained on a routine basis, it is possible that the source of the outbreak had been cleaned as part of the tower's regular maintenance, and therefore was not able to be identified as a potential source at the time of the investigation.

Legionnaires' disease outbreaks can be prevented through careful maintenance procedures that minimise the risk of contamination and these measures are mandated by the *NSW Public Health Act 1991*. For more information on Legionnaires' disease, see: www.health.nsw.gov.au/public-health/ehb/general/microbial/microbial.html.

ENTERIC DISEASES

In March, NSW Health received an increased number of reports of outbreaks of enteric diseases in childcare and aged care facilities.

In April, NSW Health's Public Health Real-Time Emergency Department Surveillance System (PHREDSS) detected an increase in people presenting to Emergency Departments (EDs) with vomiting and diarrhoea compared with previous weeks. After accounting for variations in the dates of the Easter break, this increase mirrored similar increases in each of the previous five years. As PHREDSS data do not include information on specific pathogens, the cause of the increase was unclear. On further analysis, it was found that the increase occurred mainly in children aged 0–4 years, and to a lesser extent in other people aged up to 34 years. By the end of April, however, the increase in presentations due to vomiting and diarrhoea was seen across all age groups.

Public health units were asked to contact those Emergency Departments with increases in presentations for vomiting and diarrhoea and request that clinical staff collect stool specimens from patients presenting with diarrhoea for analysis for common bacterial, viral and parasitic pathogens. Reports from public health units suggest that norovirus was the predominant pathogen identified in these patients.

Norovirus is one of the commonest causes of gastroenteritis in the community, particularly in the winter months. Symptoms include nausea, vomiting, diarrhoea, fever, abdominal pain, headache and muscle aches. The illness is self-limiting. However, it is highly contagious, and is mainly transmitted via the hands of people with the illness, either via direct contact with other people or indirectly via food. People with diarrhoea or vomiting should: wash their hands thoroughly with soap and running water for at least 10 seconds after using the toilet and before touching objects; and not handle food for other people until at least two days after their complete recovery. For more information on viral gastroenteritis see www.health.nsw. gov.au/infect/pdf/viral_gastro.pdf. Guidelines have been developed for the management of outbreaks in institutional settings (see www.health.nsw.gov.au/pubs/2004/gastroctrl_fs.html).

FIGURE 2

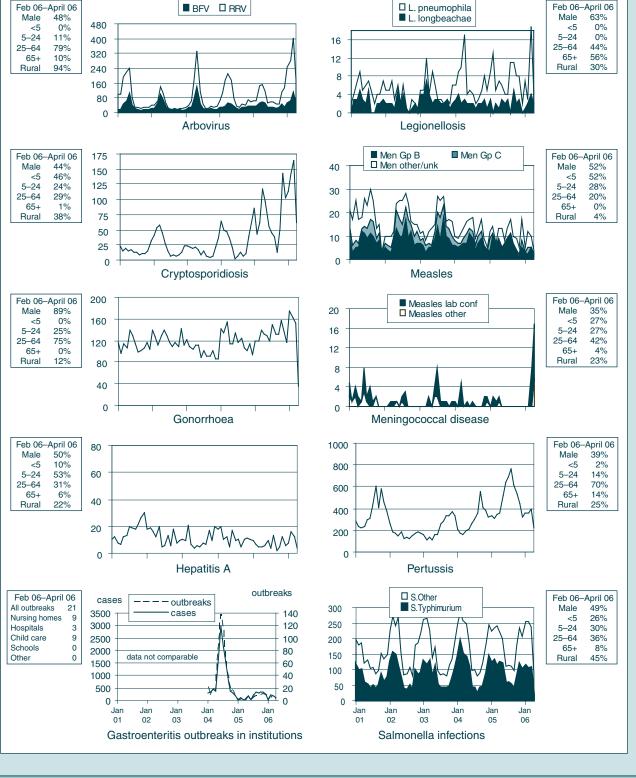
REPORTS OF SELECTED COMMUNICABLE DISEASES, NSW, JAN 2001 TO APR 2006, BY MONTH OF ONSET

Preliminary data: case counts in recent months may increase because of reporting delays. Laboratory-confirmed cases only, except for measles, meningococcal disease and pertussis BFV = Barmah Forest virus infections, RRV = Ross River virus infections Lab conf = laboratory confirmed Men Gp C and Gp B = meningococcal disease due to serogroup C and serogroup B infection, other/unk = other or unknown serogroups. NB: multiple series in graphs are stacked, except

gastroenteritis outbreaks. NB: Outbreaks are more likely to be reported

by nursing homes and hospitals than by other institutions

NSW pop	ulation
Male	50%
<5 yrs	7%
5–24 yrs	27%
25-64 yrs	53%
65+ yrs	13%
Rural	46%



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