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Year in review

Year in review: health protection in NSW, 2011

Centre for Health Protection
NSW Ministry of Health

Health protection involves the prevention and control of threats to health from communicable diseases and the environment. Health protection is achieved through a complex array of activities involving multiple agencies. Health protection activities include:

- immunisation
- the provision of safe environments including clean water, food and air
- disease surveillance, epidemiological investigations, risk assessments, capacity building, quality assurance, providing expert advice
- the development and application of legislation, regulations, policies and guidelines, distributing resources, and monitoring of program performance and outcomes.

In New South Wales (NSW) in 2011, these functions were carried out by a range of groups: at the Ministry of Health the Communicable Diseases Branch, the AIDS/Infectious Diseases Branch and the Environmental Health Branch; public health units within the local health districts; local government; other government agencies; and the community.

In this report we highlight the major health outcomes and achievements related to the health protection activities of NSW Health in 2011. The health outcomes described in this report are measured mainly through routine surveillance data that are derived from notifications of selected diseases provided by doctors, hospitals and laboratories to public health units under the former *NSW Public Health Act 1991*.*

The degree to which these notification data reflect the true incidence of disease varies between conditions, as many people with infectious disease will not be diagnosed with the disease or notified to public health units. However for some diseases, such as measles and tuberculosis, where a large proportion of people with the infection develop symptoms, present for health care and undergo confirmatory diagnostic testing soon after symptoms develop, notification data can come close to approximating the incidence of disease. For other conditions, such as hepatitis C, hepatitis B and chlamydia, where many people with the infection do not develop acute symptoms, and many acute infections are not diagnosed, notifications alone cannot be used to approximate the incidence of the disease.

Health outcomes related to environmental hazards (e.g. air pollution, poor water quality and poor housing) are harder to quantify. This is because a single environmental exposure may cause a number of different diseases and these diseases may also be caused by a number of other exposures. For example, particulate air pollution is known to cause heart disease, asthma and lung cancer. However, smoking, a high fat diet, allergies and infections may also cause these diseases. As a consequence, it is rarely possible to relate exposure to environmental hazards directly to health outcomes. Instead, hazards are monitored to identify and manage changes in population exposure. For example, the Environmental Health Branch maintains a comprehensive drinking water quality monitoring system and the NSW Office of Environment and Heritage maintains a network of air quality monitors.

Tables 1–6 summarise disease-specific data on notifiable conditions reported by: year of onset of illness; month of onset of illness; local health district; age group; and sex.

Vaccine-preventable diseases

Notification data

In 2011 there were:

- over 13 000 **pertussis** case notifications, a record number, mostly in 5–9-year old children (over 4000 notifications), followed by 0–4-year old children (over 2400) and 10–14-year olds (over 2300). There was considerable variation in pertussis notification rates between local health districts. The pertussis epidemic highlights the

10.1071/NB12073

Table 1. Disease notifications by year of onset of illness, NSW, 2002–2011

Condition	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	Total
Adverse event after immunisation	178	219	187	107	72	241	259	126	176	204	1769
Anthrax	0	0	0	0	1	0	0	0	1	0	2
Arboviral infection	659	1020	1140	1078	1918	1499	1848	1411	1556	1171	13 300
Barmah Forest virus infection[a]	395	451	400	449	643	574	530	358	255	457	4512
Ross River virus infection[a]	182	492	697	575	1221	842	1155	909	1067	571	7711
Other[a]	82	77	43	54	54	83	163	144	234	143	1077
Blood lead level ≥15 ug/dl[a]	496	331	298	226	294	274	261	178	229	257	2844
Botulism	0	0	1	0	0	0	0	0	0	2	3
Brucellosis[a]	2	3	7	3	9	4	1	4	3	6	42
Chancroid[a]	0	0	0	0	0	0	0	0	0	0	0
Chlamydia trachomatis infection	5812	7775	10 003	11 268	12 056	12 462	14 029	15 001	18 254	20 536	127 196
Congenital chlamydia[a]	15	23	28	46	39	31	44	52	37	31	346
Chlamydia – other[a]	5797	7752	9975	11 222	12 017	12 431	13 985	14 949	18 217	20 505	126 850
Cholera[a]	1	0	1	0	3	2	2	3	2	0	14
Creutzfeldt-Jakob disease[a]	*NN*	*NN*	6	8	11	9	8	11	8	9	70
Cryptosporidiosis[a]	305	203	353	849	778	544	486	1463	348	359	5688
Diphtheria	0	0	0	0	0	0	0	0	0	0	0
Foodborne illness (NOS)[b]	41	1071	550	309	507	763	667	902	927	797	6534
Gastroenteritis (institutional)	1752	3583	12 784	1395	10 641	10 488	10 135	11 876	7651	9071	79 376
Giardiasis[a]	862	1028	1233	1450	1723	1945	1783	2099	2300	2367	16 790
Gonorrhoea[a]	1520	1325	1430	1571	1736	1383	1330	1654	2301	2879	17 129
Haemolytic uraemic syndrome	7	5	9	11	11	13	17	4	3	4	84
H. influenzae type b[a]	10	6	5	7	11	7	9	6	6	4	71
Hepatitis A	149	123	137	83	95	65	69	98	83	57	959
Hepatitis B	3369	2730	2674	2705	2485	2602	2534	2639	2605	2540	26 883
Hepatitis B – acute viral[a]	88	74	53	56	52	56	45	37	35	31	527
Hepatitis B – other[a]	3281	2656	2621	2649	2433	2546	2489	2602	2570	2509	26 356
Hepatitis C	6223	4902	4596	4295	4318	4160	3740	3806	3816	3329	43 185
Hepatitis C – acute viral[a]	144	123	58	43	56	64	26	41	39	44	638
Hepatitis C – other[a]	6079	4779	4538	4252	4262	4096	3714	3765	3777	3285	42 547
Hepatitis D[a]	9	12	14	14	15	11	14	9	9	8	115
Hepatitis E[a]	6	6	8	7	10	8	14	17	14	20	110
HIV infection[a]	395	412	404	392	366	388	323	329	307	330	3646
Influenza	1010	862	940	1422	681	2061	1867	12856	1601	5625	28 925
Influenza – Type A[a]	768	769	793	1134	488	1704	841	12580	1415	3955	24 447
Influenza – Type B[a]	241	55	120	267	183	183	1006	162	143	1566	3926
Influenza – Type A&B[a]	*NN*	*NN*	0	10	2	2	3	12	36	29	94
Influenza – Type NOS[a]	1	38	27	11	8	172	17	102	7	75	458
Legionellosis	44	60	80	88	78	104	90	94	93	91	822
Legionella longbeachae[a]	21	37	26	23	22	29	52	64	47	31	352
L. pneumophila[a]	22	23	52	64	56	73	38	28	38	57	451
Legionnaires' disease – other	1	0	2	1	0	2	0	2	8	3	19
Leprosy	0	2	5	1	1	4	4	0	1	2	20
Leptospirosis[a]	39	39	40	35	17	9	17	18	22	39	275
Listeriosis[a]	11	28	30	25	26	22	34	27	26	21	250
Lymphogranuloma venereum[a]	0	0	1	2	1	0	3	4	56	39	106
Malaria[a]	105	120	100	205	140	97	115	91	122	76	1171
Measles	8	18	12	5	60	4	39	19	26	90	281
Meningococcal disease	213	198	146	137	101	109	80	92	74	71	1221
Meningococcal – serogroup B[a]	105	100	81	73	54	76	49	57	49	43	687
Meningococcal – serogroup C[a]	54	45	24	16	12	9	9	7	5	2	183
Meningococcal – serogroup W135[a]	2	2	5	8	5	2	5	5	4	4	42
Meningococcal – serogroup Y[a]	2	5	3	3	1	5	4	3	3	4	33
Meningococcal – other	50	46	33	37	29	17	13	20	13	18	276
Meningococcal – conjunctivitis	3	4	3	3	5	3	1	4	2	1	29
Mumps[a]	29	36	65	111	155	323	77	40	39	65	940
Paratyphoid[a,c]	13	22	10	0	0	0	0	0	0	0	45
Pertussis	2014	2771	3567	5800	4914	2099	8757	12 437	9326	13 053	64 738
Pneumococcal disease (invasive)[a]	881	801	902	641	561	519	548	474	500	524	6351
Psittacosis[a]	155	88	81	121	94	35	40	22	16	19	671
Q fever[a]	308	287	220	143	177	205	167	139	137	114	1897
Rotavirus[a]	*NN*	*NN*	*NN*	*NN*	*NN*	*NN*	*NN*	*NN*	1381	1054	2435
Rubella	35	24	18	10	37	9	17	7	13	17	187
Congenital rubella[a]	0	1	1	0	0	1	0	0	0	0	3
Rubella – other[a]	35	23	17	10	37	8	17	7	13	17	184
Salmonella infection[a,c]	2110	1853	2139	2157	2052	2531	2275	2740	3766	3486	25109
Shigellosis[a]	85	59	96	134	74	71	109	153	116	132	1029
Syphilis	460	785	755	550	617	823	841	936	811	775	7353
Congenital syphilis	2	3	1	10	4	5	3	0	0	3	31
Syphilis infection[a,d]	126	242	294	241	230	457	428	529	419	416	3382
Syphilis – other[a]	332	540	460	299	383	361	410	407	392	356	3940
Tetanus	0	1	1	1	2	2	1	2	1	1	12
Tuberculosis[a,e]	448	388	413	463	454	457	494	513	468	306	4404
Typhoid[a]	26	15	38	27	35	34	43	47	30	45	340
Verotoxin-producing *Escherichia coli* infections[a]	6	3	5	16	10	23	19	21	10	10	123

Onset of illness: the earlier of patient reported onset date, specimen date or date of notification.
[a]Laboratory-confirmed cases only.
[b]Foodborne illness cases are only those notified as part of an outbreak.
[c]From 2005, all paratyphoid recorded as salmonellosis.
[d]Includes syphilis primary, syphilis secondary, syphilis < 1 y duration and syphilis newly acquired.
[e]Tuberculosis data for 2011 was incomplete at the time of report and subject to change.
NOS: not otherwise specified.
NN: not notifiable for that year.
No case of the following diseases have been notified since 1991 : plague[a], diphtheria[a], granuloma inguinale[a], lyssavirus[a], poliomyelitis[a], rabies, smallpox, typhus[a], viral haemorrhagic fever, yellow fever.
2009 influenza data: cases reported to public health units; contain 50 laboratory notifications from either interstate residents or overseas travellers.
Source: Notifiable Conditions Information Management System, Centre for Health Protection, NSW Health.

Table 2. Incidence rate of disease notifications in NSW (per 100 000 population), 2002–2011

Condition	Onset year									
	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011
Adverse event after immunisation	2.7	3.3	2.8	1.6	1.1	3.5	3.7	1.8	2.5	2.8
Anthrax	0.0	0.0	0.0	0.0	1.0	0.0	0.0	0.0	1.0	0.0
Arboviral infection	9.9	15.4	17.0	15.9	28.1	21.7	26.4	19.8	21.8	16.2
Barmah Forest virus infections[a]	6.0	6.8	6.0	6.6	9.4	8.3	7.6	5.0	3.6	6.3
Ross River virus infections[a]	2.7	7.4	10.4	8.5	17.9	12.2	16.5	12.8	14.9	7.9
Other[a]	1.2	1.2	0.6	0.8	0.8	1.2	2.3	2.0	3.3	2.0
Blood lead level ≥15 ug/dl[a]	7.5	5.0	4.4	3.3	4.3	4.0	3.7	2.5	3.2	3.6
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis[a]	0.0	0.0	0.1	0.0	0.1	0.1	0.0	0.1	0.0	0.1
Chancroid[a]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chlamydia trachomatis infection	87.6	116.5	149.1	166.8	176.9	180.4	200.0	211.4	255.1	284.9
Congenital chlamydia[a]	0.2	0.3	0.4	0.7	0.6	0.4	0.6	0.7	0.5	0.4
Chlamydia – other[a]	87.4	116.2	148.7	166.1	176.3	180.0	199.4	210.7	254.6	284.5
Cholera[a]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Creutzfeldt-Jakob disease[a]	*NN*	*NN*	0.1	0.1	0.2	0.1	0.1	0.2	0.1	0.1
Cryptosporidiosis[a]	4.6	3.0	5.3	12.6	11.4	7.9	6.9	20.6	4.9	5.0
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Foodborne illness (NOS)[b]	0.6	16.1	8.2	4.6	7.4	11.1	9.5	12.7	13.0	11.1
Gastroenteritis (institutional)	26.4	53.7	190.6	20.6	156.1	151.9	144.5	167.4	106.9	125.9
Giardiasis[a]	13.0	15.4	18.4	21.5	25.3	28.2	25.4	29.6	32.2	32.8
Gonorrhoea[a]	22.9	19.9	21.3	23.3	25.5	20.0	19.0	23.3	32.2	39.9
Haemolytic uraemic syndrome	0.1	0.1	0.1	0.2	0.2	0.2	0.2	0.1	0.0	0.1
H. influenzae type b[a]	0.2	0.1	0.1	0.1	0.2	0.1	0.1	0.1	0.1	0.1
Hepatitis A	2.3	1.8	2.0	1.2	1.4	0.9	1.0	1.4	1.2	0.8
Hepatitis B	50.8	40.9	39.9	40.0	36.5	37.7	36.1	37.2	36.4	35.2
Hepatitis B – acute viral[a]	1.3	1.1	0.8	0.8	0.8	0.8	0.6	0.5	0.5	0.4
Hepatitis B – other[a]	49.5	39.8	39.1	39.2	35.7	36.9	35.5	36.7	35.9	34.8
Hepatitis C	93.9	73.4	68.6	63.5	63.3	60.2	53.3	53.7	53.3	46.2
Hepatitis C – acute viral[a]	2.2	1.8	0.9	0.6	0.8	0.9	0.4	0.6	0.5	0.6
Hepatitis C – other[a]	91.7	71.6	67.7	62.9	62.5	59.3	52.9	53.1	52.8	45.6
Hepatitis D[a]	0.1	0.2	0.2	0.2	0.2	0.2	0.2	0.1	0.1	0.1
Hepatitis E[a]	0.1	0.1	0.1	0.1	0.2	0.1	0.2	0.2	0.2	0.3
HIV infection[a]	6.0	6.2	6.0	5.8	5.4	5.6	4.6	4.6	4.3	4.6
Influenza	15.2	12.9	14.0	21.1	10.0	29.9	26.5	181.2	22.4	78.0
Influenza –Type A[a]	11.6	11.5	11.8	16.8	7.2	24.7	12.0	177.3	19.8	54.9
Influenza – Type B[a]	3.6	0.8	1.8	4.0	2.7	2.7	14.3	2.3	2.0	21.7
Influenza – Type A&B[a]	*NN*	0.0	0.0	0.1	0.0	0.0	0.0	0.2	0.5	0.4
Influenza – Type NOS[a]	0.0	0.6	0.4	0.2	0.1	2.5	0.2	1.4	0.1	1.0
Legionellosis	0.6	0.9	1.2	1.2	1.1	1.5	1.2	1.3	1.3	1.2
Legionella longbeachae[a]	0.3	0.6	0.4	0.3	0.3	0.4	0.7	0.9	0.7	0.4
L. pneumophila[a]	0.3	0.3	0.8	0.9	0.8	1.1	0.5	0.4	0.5	0.8
Legionnaires' disease – other	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0
Leprosy	0.0	0.0	0.1	0.0	0.0	0.1	0.1	0.0	0.0	0.0
Leptospirosis[a]	0.6	0.6	0.6	0.5	0.2	0.1	0.2	0.3	0.3	0.5
Listeriosis[a]	0.2	0.4	0.4	0.4	0.4	0.3	0.5	0.4	0.4	0.3
Lymphogranuloma venereum[a]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.8	0.5
Malaria[a]	1.6	1.8	1.5	3.0	2.1	1.4	1.6	1.3	1.7	1.1
Measles	0.1	0.3	0.2	0.1	0.9	0.1	0.6	0.3	0.4	1.2
Meningococcal disease	3.2	3.0	2.2	1.9	1.5	1.5	1.2	1.3	1.1	1.0
Meningococcal – serogroup B[a]	1.6	1.5	1.2	1.1	0.8	1.1	0.7	0.8	0.7	0.6
Meningococcal – serogroup C[a]	0.8	0.7	0.4	0.2	0.2	0.1	0.1	0.1	0.1	0.0
Meningococcal – serogroup W135[a]	0.0	0.0	0.1	0.1	0.1	0.0	0.1	0.1	0.1	0.1
Meningococcal – serogroup Y[a]	0.0	0.1	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.1
Meningococcal – other	0.8	0.7	0.5	0.5	0.4	0.2	0.2	0.3	0.2	0.2
Meningococcal – conjunctivitis	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0
Mumps[a]	0.4	0.5	1.0	1.6	2.3	4.7	1.1	0.6	0.5	0.9
Paratyphoid[a,c]	0.2	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Pertussis	30.4	41.5	53.2	85.8	72.1	30.4	124.8	175.3	130.4	181.1
Pneumococcal disease (invasive)[a]	13.3	12.0	13.4	9.5	8.2	7.5	7.8	6.7	7.0	7.3
Psittacosis[a]	2.3	1.3	1.2	1.8	1.4	0.5	0.6	0.3	0.2	0.3
Q fever[a]	4.6	4.3	3.3	2.1	2.6	3.0	2.4	2.0	1.9	1.6
Rotavirus[a]	*NN*	*NN*	*NN*	*NN*	*NN*	*NN*	*NN*	*NN*	19.3	14.6
Rubella	0.5	0.3	0.3	0.1	0.5	0.1	0.2	0.1	0.2	0.2
Congenital rubella[a]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella – other[a]	0.5	0.3	0.3	0.1	0.5	0.1	0.2	0.1	0.2	0.2
Salmonella infection[a,c]	31.8	27.8	31.9	31.9	30.1	36.7	32.4	38.6	52.6	48.4
Shigellosis[a]	1.3	0.9	1.4	2.0	1.1	1.0	1.6	2.2	1.6	1.8
Syphilis	6.9	11.7	11.3	8.1	9.1	11.9	11.9	13.2	11.4	10.7
Congenital syphilis	0.0	0.0	0.0	0.1	0.1	0.1	0.0	0.0	0.0	0.0
Syphilis infection[a,d]	1.9	3.6	4.4	3.6	3.4	6.6	6.1	7.5	5.9	5.8
Syphilis – other[a]	5.0	8.1	6.9	4.4	5.6	5.2	5.8	5.7	5.5	4.9
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tuberculosis[a,e]	6.8	5.8	6.2	6.9	6.7	6.6	7.0	7.2	6.5	4.2
Typhoid[a]	0.4	0.2	0.6	0.4	0.5	0.5	0.6	0.7	0.4	0.6
Verotoxin-producing *Escherichia coli* infections[a]	0.1	0.0	0.1	0.2	0.1	0.3	0.3	0.3	0.1	0.1

Onset of illness: the earlier of patient reported onset date, specimen date or date of notification.
[a]Laboratory-confirmed cases only.
[b]Foodborne illness cases are only those notified as part of an outbreak.
[c]From 2005, all paratyphoid recorded as salmonellosis.
[d]Includes syphilis primary, syphilis secondary, syphilis < 1 y duration and syphilis newly acquired.
[e]Tuberculosis data for 2011 was incomplete at the time of report and subject to change.
NOS: not otherwise specified.
NN: not notifiable for that year.
No case of the following diseases have been notified since 1991 : plague[a], diphtheria[a], granuloma inguinale[a], lyssavirus[a], poliomyelitis[a], rabies, smallpox, typhus[a], viral haemorrhagic fever, yellow fever.
2009 influenza data: cases reported to public health units; contain 50 laboratory notifications from either interstate residents or overseas travellers.
Source: Notifiable Conditions Information Management System, Centre for Health Protection, NSW Health.

Table 3. Disease notifications by month of onset of illness, NSW, 2011

Condition	Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.	Total[f]
Adverse event after immunisation	3	10	59	28	23	13	14	12	13	9	11	9	204
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0
Arboviral infection	188	173	198	113	106	62	48	57	42	56	67	61	1171
Barmah Forest virus infection[a]	95	65	70	26	37	31	24	26	15	28	25	15	457
Ross River virus infection[a]	70	95	117	83	60	21	15	23	20	20	26	21	571
Other[a]	23	13	11	4	9	10	9	8	7	8	16	25	143
Blood lead level ≥15 ug/dl[a]	19	41	16	39	16	15	18	24	21	21	22	5	257
Botulism	0	0	0	0	1	0	0	0	1	0	0	0	2
Brucellosis[a]	0	1	2	0	1	0	0	0	1	0	1	0	6
Chancroid[a]	0	0	0	0	0	0	0	0	0	0	0	0	0
Chlamydia trachomatis infection	1559	1777	1977	1487	1775	1839	1675	1773	1669	1717	1813	1475	20 536
Congenital chlamydia[a]	8	1	0	4	1	3	1	0	4	2	4	3	31
Chlamydia – other[a]	1551	1776	1977	1483	1774	1836	1674	1773	1665	1715	1809	1472	20 505
Cholera[a]	0	0	0	0	0	0	0	0	0	0	0	0	0
Creutzfeldt-Jakob disease[a]	0	2	1	0	0	2	0	1	1	1	1	0	9
Cryptosporidiosis[a]	35	28	46	39	47	19	31	19	12	26	28	29	359
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0
Foodborne illness (NOS)[b]	190	72	56	92	118	0	33	45	102	11	69	9	797
Gastroenteritis (institutional)	271	705	592	1077	1354	1239	1319	806	493	528	291	396	9071
Giardiasis[a]	177	292	370	202	205	176	170	158	148	144	169	156	2367
Gonorrhoea[a]	123	136	165	145	130	102	102	122	135	151	152	190	1653
Haemolytic uraemic syndrome	1	1	0	0	1	0	0	1	0	0	0	0	4
H. influenzae type b[a]	0	0	2	0	2	0	0	0	0	0	0	0	4
Hepatitis A	10	2	11	4	7	2	1	5	1	6	5	3	57
Hepatitis B	218	225	248	202	208	179	218	228	206	222	209	177	2540
Hepatitis B – acute viral[a]	6	3	1	3	2	2	4	3	3	2	2	0	31
Hepatitis B – other[a]	212	222	247	199	206	177	214	225	203	220	207	177	2509
Hepatitis C	281	261	326	251	276	283	292	317	269	257	291	225	3329
Hepatitis C – acute viral[a]	6	5	4	0	6	3	2	6	4	0	3	5	44
Hepatitis C – other[a]	275	256	322	251	270	280	290	311	265	257	288	220	3285
Hepatitis D[a]	2	1	0	1	1	2	1	0	0	0	1	0	9
Hepatitis E[a]	0	4	4	0	3	1	1	1	2	0	1	0	17
HIV infection[a]	23	30	38	31	31	22	30	24	34	24	17	26	330
Influenza	125	108	128	103	169	631	1595	1547	621	310	176	112	5625
Influenza – Type A[a]	107	91	101	81	123	507	1226	861	368	239	158	93	3955
Influenza – Type B[a]	13	13	17	19	40	119	357	665	236	56	15	16	1566
Influenza – Type A&B[a]	0	1	5	1	4	1	3	5	8	0	1	0	29
Influenza – Type NOS[a]	5	3	5	2	2	4	9	16	9	15	2	3	75
Legionellosis	2	8	13	20	7	5	10	2	3	5	5	11	91
Legionella longbeachae[a]	1	4	5	0	2	3	4	0	1	4	3	4	31
L. pneumophila[a]	1	4	7	20	5	2	5	2	2	1	2	6	57
Legionnaires' disease – other	0	0	1	0	0	0	1	0	0	0	0	1	3
Leprosy	0	0	0	0	1	0	0	0	0	0	1	0	2
Leptospirosis[a]	5	3	7	4	2	8	2	1	2	2	1	2	39
Listeriosis[a]	3	2	1	1	4	1	1	2	0	1	1	4	21
Lymphogranuloma venereum[a]	6	5	4	3	5	3	2	3	2	2	1	3	39
Malaria[a]	6	5	8	9	4	4	9	8	5	8	5	5	76
Measles	1	22	21	5	4	2	5	14	5	1	6	4	90
Meningococcal disease	7	5	9	4	2	7	7	6	8	7	6	3	71
Meningococcal – serogroup B[a]	4	1	6	3	1	4	4	4	5	4	4	3	43
Meningococcal – serogroup C[a]	0	0	0	0	0	0	0	0	0	1	1	0	2
Meningococcal – serogroup W135[a]	0	1	1	0	0	0	1	1	0	0	0	0	4
Meningococcal – serogroup Y[a]	0	0	0	1	0	1	1	0	0	1	0	0	4
Meningococcal – other	3	3	2	0	1	2	1	1	3	1	1	0	18
Meningococcal – conjunctivitis	0	1	0	0	0	0	0	0	0	0	0	0	1
Mumps[a]	2	4	8	6	4	6	4	8	2	4	6	11	65
Paratyphoid[a,c]	0	0	0	0	0	0	0	0	0	0	0	0	0
Pertussis	1447	1080	1112	924	1048	923	824	1181	1246	1164	1124	980	13 053
Pneumococcal disease (invasive)[a]	11	29	24	38	48	59	68	64	62	42	33	46	524
Psittacosis[a]	3	2	0	2	1	1	1	1	2	3	2	1	19
Q fever[a]	10	6	8	9	10	7	9	10	14	10	9	12	114
Rotavirus[a]	66	76	76	59	66	60	58	92	120	185	121	75	1054
Rubella	0	1	6	1	2	2	0	1	1	0	3	0	17
Congenital rubella[a]	0	0	0	0	0	0	0	0	0	0	0	0	0
Rubella – other[a]	0	1	6	1	2	2	0	1	1	0	3	0	17
Salmonella infection[a,c]	597	487	462	294	271	168	182	165	140	211	229	280	3486
Shigellosis[a]	18	13	13	9	9	9	8	5	6	13	15	14	132
Syphilis	66	56	84	52	57	60	61	82	62	79	76	40	775
Congenital syphilis	0	0	2	0	0	0	0	1	0	0	0	0	3
Syphilis infection[a,d]	35	32	43	25	33	38	29	42	38	34	43	24	416
Syphilis – other[a]	31	24	39	27	24	22	32	39	24	45	33	16	356
Tetanus	0	0	1	0	0	0	0	0	0	0	0	0	1
Tuberculosis[a,e]	35	39	29	26	24	26	28	21	25	27	19	7	306
Typhoid[a]	10	10	5	4	1	0	7	1	1	3	1	2	45
Verotoxin-producing *Escherichia coli* infections[b]	0	0	0	1	1	1	0	1	2	2	0	2	10

Onset of illness: the earlier of patient reported onset date, specimen date or date of notification.
[a]Laboratory-confirmed cases only.
[b]Foodborne illness cases are only those notified as part of an outbreak.
[c]Includes syphilis primary, syphilis secondary, syphilis < 1 y duration and syphilis newly acquired.
[d]Includes all paratyphoid cases.
[e]Tuberculosis data for 2011 was incomplete at the time of report and subject to change.
[f]Total cases including Justice Health services, other than local health district not allocated and overseas cases.
NOS: not otherwise specified.
No case of the following diseases have been notified since 1991 : plague[a], diphtheria[a], granuloma inguinale[a], lyssavirus[a], poliomyelitis[a], rabies, smallpox, typhus[a], viral haemorrhagic fever, yellow fever.
Source: Notifiable Conditions Information Management System, Centre for Health Protection, NSW Health.

Table 4. Disease notifications by local health district of residence, NSW, 2011 (based on onset of illness)

Condition	Sydney	Central Coast	Far West	Hunter New England	Illawarra Shoalhaven	Mid North Coast	Murrum-bidgee	Nepean Blue Mountains	North Sydney	Northern NSW	South Eastern Sydney	South Western Sydney	Southern NSW	Western Sydney	Western NSW	Justice Health	Other[f]	Overseas	Total[g]
Adverse event after immunisation	6	0	1	23	20	3	19	5	43	3	10	5	22	33	11	0	0	0	204
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Arboviral infection	12	19	56	223	47	131	182	11	32	244	26	22	33	15	113	0	3	2	1171
Barmah Forest virus infections[a]	0	7	16	76	20	73	51	3	5	152	3	1	18	2	29	0	1	0	457
Ross River virus infections[a]	3	6	40	129	4	58	129	6	6	86	2	3	11	3	82	0	2	1	571
Other[a]	9	6	0	18	23	0	2	2	21	6	21	18	4	10	2	0	0	1	143
Blood lead level ≥15 ug/dl[a]	11	7	42	11	5	0	62	10	4	0	13	21	4	19	48	0	0	0	257
Botulism	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	0	0	2
Brucellosis[a]	0	0	0	2	0	0	0	0	0	0	0	1	0	3	0	0	0	0	6
Chancroid[a]	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chlamydia trachomatis infection	2021	950	87	2841	1115	570	780	850	1580	943	3518	1785	437	1691	919	187	224	38	20536
Congenital chlamydia[a]	1	0	0	5	2	0	3	0	2	2	1	7	1	3	4	0	0	0	31
Chlamydia – other[a]	2020	950	87	2836	1113	570	777	850	1578	941	3517	1778	436	1688	915	187	224	38	20505
Cholera[a]	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Creutzfeldt-Jakob disease[a]	1	0	0	2	1	0	1	0	0	0	1	1	0	1	1	0	0	0	9
Cryptosporidiosis[a]	32	14	1	45	21	24	9	19	50	25	50	18	5	39	7	0	0	0	359
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Foodborne illness (NOS)[b]	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Gastroenteritis (institutional)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Giardiasis[a]	249	80	5	257	129	46	74	133	421	15	454	149	44	186	120	2	3	0	2367
Gonorrhoea[a]	592	47	2	209	74	23	23	92	215	60	896	247	19	269	36	11	51	13	2879
Haemolytic uraemic syndrome	0	0	0	0	0	0	0	0	0	0	1	1	2	0	0	0	0	0	4
H. influenzae type b[a]	0	0	0	2	0	0	0	0	0	2	0	0	0	0	0	0	0	0	4
Hepatitis A	5	1	0	2	1	0	3	1	3	0	10	10	0	18	1	1	1	0	57
Hepatitis B	389	25	7	68	47	20	32	64	282	20	367	510	19	578	44	35	29	4	2540
Hepatitis B – acute viral[a]	2	0	0	5	0	0	2	3	2	3	3	3	1	2	4	1	0	0	31
Hepatitis B – other[a]	387	25	7	63	47	20	30	61	280	17	364	507	18	576	40	34	29	4	2509
Hepatitis C	321	160	24	319	184	98	142	127	146	168	335	398	111	314	188	254	37	3	3329
Hepatitis C – acute viral[a]	0	0	2	15	1	3	0	0	0	3	3	2	1	1	10	2	1	0	44
Hepatitis C – other[a]	321	160	22	304	183	95	142	127	146	165	332	396	110	313	178	252	36	3	3285
Hepatitis D[a]	0	0	0	0	0	1	0	1	0	0	1	3	0	2	0	0	0	0	8
Hepatitis E[a]	1	0	0	1	0	1	0	0	2	0	4	3	0	8	0	0	0	0	20
Influenza	319	94	15	967	130	133	152	379	520	468	590	500	138	939	258	4	16	3	5625
Influenza – Type A[a]	240	67	10	630	103	101	90	280	385	300	396	346	100	662	211	3	12	3	3955
Influenza – Type B[a]	74	25	3	328	25	30	45	94	127	162	177	144	30	258	39	1	4	0	1566
Influenza – Type A&B[a]	0	0	0	2	1	0	0	4	0	0	17	0	2	2	1	0	0	0	29
Influenza – Type NOS[a]	5	2	2	7	1	2	1	1	8	6	0	10	6	17	7	0	0	0	75
Legionellosis	7	3	0	5	13	0	2	6	10	0	16	7	2	15	5	0	0	0	91
Legionella longbeachae[a]	1	3	0	2	8	0	1	2	3	0	3	2	1	4	1	0	0	0	31
L. pneumophila[a]	6	0	0	2	5	0	1	4	7	0	13	5	1	9	4	0	0	0	57
Legionnaires' disease – other	0	0	0	1	0	0	0	0	0	0	0	0	0	2	0	0	0	0	3
Leprosy	0	0	0	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0	2
Leptospirosis[a]	0	0	0	4	3	2	16	0	0	6	2	0	2	0	4	0	0	0	39
Listeriosis[a]	0	1	0	2	2	1	1	2	3	0	4	3	0	2	0	0	0	0	21
Lymphogranuloma venereum[a]	13	0	0	0	0	0	0	2	2	0	13	1	0	7	0	0	0	1	39
Malaria[a]	7	2	0	11	2	3	3	5	3	1	9	7	0	21	2	0	0	0	76
Measles	9	3	0	2	12	0	0	0	2	3	12	11	10	26	0	0	0	0	90
Meningococcal disease	6	2	0	15	6	0	3	4	9	1	5	6	1	7	6	0	0	0	71
Meningococcal – serogroup B[a]	3	1	0	11	3	0	2	3	4	1	3	5	1	4	2	0	0	0	43
Meningococcal – serogroup C[a]	0	0	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0	0	2
Meningococcal – serogroup W135[a]	0	1	0	0	0	0	1	0	1	0	0	0	0	0	1	0	0	0	4
Meningococcal – serogroup Y[a]	1	0	0	1	1	0	0	0	0	0	0	1	0	0	0	0	0	0	4
Meningococcal – other	2	0	0	2	2	0	0	1	4	0	2	0	0	2	3	0	0	0	18
Meningococcal – conjunctivitis	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1
Mumps[a]	10	0	0	0	1	1	1	3	9	4	16	5	2	12	0	0	1	0	65
Paratyphoid[a,c]	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Pertussis	585	347	53	776	1071	316	1286	1150	1554	698	1316	1231	435	1507	707	2	19	0	13053
Pneumococcal disease (invasive)[a]	38	25	2	70	35	9	18	36	51	19	71	51	16	40	41	0	0	2	524
Psittacosis[a]	0	0	0	3	1	0	0	5	3	2	0	1	0	3	1	0	0	0	19
Q fever[a]	1	1	1	26	17	16	6	1	2	19	0	2	10	0	12	0	0	0	114
Rotavirus[a]	89	28	2	133	17	18	18	69	223	60	118	55	11	125	85	0	1	2	1054
Rubella	3	1	0	1	0	0	0	0	3	0	4	2	0	3	0	0	0	0	17
Congenital rubella[a]	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rubella – other[a]	3	1	0	1	0	0	0	0	3	0	4	2	0	3	0	0	0	0	17
Salmonella infection[a,c]	307	130	15	382	122	129	203	111	464	180	426	474	79	353	89	3	15	4	3486
Shigellosis[a]	27	5	0	3	5	2	1	4	16	2	31	11	2	18	4	0	0	1	132
Syphilis	187	17	7	29	38	3	3	23	27	5	214	83	16	82	29	2	9	1	775
Congenital syphilis	0	0	0	0	0	0	0	0	0	0	0	0	0	1	2	0	0	0	3
Syphilis infection[a,d]	122	1	0	11	16	3	0	10	14	2	174	18	10	24	5	0	5	1	416
Syphilis – other[a]	65	16	7	18	22	0	3	13	13	3	40	65	6	57	22	2	4	0	356
Tetanus	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Tuberculosis[a,e]	34	4	1	9	15	4	12	9	40	2	64	13	4	84	5	3	1	2	306
Typhoid[a]	9	0	0	3	1	0	0	1	3	0	7	5	0	15	0	0	1	0	45
Verotoxin-producing *Escherichia coli* infections[a]	1	0	0	3	1	0	2	0	0	1	0	1	1	0	0	0	0	0	10

Onset of illness: the earlier of patient reported onset date, specimen date or date of notification.
[a]Laboratory-confirmed cases only.
[b]Foodborne illness cases are only those notified as part of an outbreak.
[c]Includes all paratyphoid cases.
[d]Includes syphilis primary, syphilis secondary, syphilis < 1 y duration and syphilis newly acquired.
[e]Tuberculosis data for 2011 was incomplete at the time of report and subject to change.
[f]Includes disease notifications with unknown local health district.
[g]Total cases including Justice Health services, other than local health district not allocated and overseas cases.
NOS: not otherwise specified.
No case of the following diseases have been notified since 1991 : plague[a], diphtheria[a], granuloma inguinale[a], lyssavirus[a], poliomyelitis[a], rabies, smallpox, typhus[a], viral haemorrhagic fever, yellow fever.
Source: Notifiable Conditions Information Management System, Centre for Health Protection, NSW Health.

Table 5. Incidence rate of disease notifications by local health district of residence, NSW (per 100 000 population), 2011 (based on onset of illness)

Condition	Sydney	Central Coast	Far West	Hunter New England	Illawarra Shoalhaven	Mid North Coast	Murrumbidgee	Nepean Blue Mountains	North Sydney	Northern NSW	South Eastern Sydney	South Western Sydney	Southern NSW	Western Sydney	Western NSW
Adverse event after immunisation	0.9	0.0	3.2	2.6	5.1	1.4	6.5	1.4	5.0	1.0	1.2	0.5	9.4	3.8	4.1
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Arboviral infection	1.5	6.0	173.3	24.9	12.0	60.8	60.3	3.2	3.8	79.9	3.1	2.3	16.3	1.8	40.5
Barmah Forest virus infection[a]	0.0	2.2	44.9	8.6	5.1	34.1	17.5	0.9	0.6	49.7	0.4	0.1	8.9	0.2	10.4
Ross River virus infection[a]	0.3	1.9	128.4	14.3	1.0	26.7	42.1	1.7	0.7	28.2	0.2	0.3	5.4	0.4	29.4
Other[a]	1.2	1.9	0.0	2.0	5.9	0.0	0.7	0.6	2.5	2.0	2.5	1.9	2.0	1.2	0.7
Blood lead level ≥15 ug/dl[a]	1.7	2.2	134.8	1.2	1.3	0.0	20.5	2.9	0.4	0.0	1.6	2.4	2.0	2.2	17.9
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.4
Brucellosis[a]	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.4	0.0
Chancroid[a]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chlamydia trachomatis infection	344.6	293.7	276.0	318.3	287.0	261.4	256.7	242.5	185.3	309.9	418.6	200.1	210.7	200.1	331.7
Congenital chlamydia[a]	0.2	0.0	0.0	0.6	0.5	0.0	1.0	0.0	0.2	0.7	0.1	0.8	0.5	0.4	1.5
Chlamydia – other[a]	344.4	293.7	276.0	317.7	286.5	261.4	255.7	242.5	185.1	309.2	418.5	199.3	210.2	199.7	330.2
Cholera[a]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Creutzfeldt-Jakob disease[a]	0.2	0.0	0.0	0.2	0.3	0.0	0.3	0.0	0.0	0.0	0.1	0.1	0.0	0.1	0.4
Cryptosporidiosis[a]	5.4	4.4	3.2	5.0	5.4	11.2	3.1	5.5	6.0	8.4	6.0	1.9	2.5	4.6	2.6
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Foodborne illness (NOS)[b]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gastroenteritis (institutional)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Giardiasis[a]	42.4	25.1	16.0	28.7	33.2	21.0	25.0	37.3	49.7	5.0	54.1	16.6	21.7	22.2	43.6
Gonorrhoea[a]	101.5	14.8	6.4	23.3	19.1	10.8	7.5	26.3	25.0	19.5	106.9	28.0	9.4	31.8	10.4
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.5	0.0	0.0
H. influenzae type b[a]	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0
Hepatitis A	0.9	0.3	0.0	0.2	0.3	0.0	1.0	0.3	0.4	0.0	1.2	1.1	0.0	2.2	0.4
Hepatitis B	66.5	7.9	22.5	7.6	12.1	9.4	11.0	18.5	33.2	6.4	43.6	57.3	9.4	69.0	16.4
Hepatitis B – acute viral[a]	0.3	0.0	0.0	0.6	0.0	0.0	0.7	0.9	0.2	1.0	0.4	0.3	0.5	0.2	1.5
Hepatitis B – other[a]	66.2	7.9	22.5	7.0	12.1	9.4	10.3	17.6	33.0	5.4	43.2	57.0	8.9	68.8	14.9
Hepatitis C	55.2	49.6	77.0	35.2	47.4	45.4	47.2	36.2	17.0	54.4	39.9	44.0	54.8	37.4	68.5
Hepatitis C – acute viral[a]	0.0	0.0	6.4	1.7	0.3	1.4	0.0	0.0	0.0	1.0	0.4	0.2	0.5	0.1	3.7
Hepatitis C – other[a]	55.2	49.6	70.6	33.5	47.1	44.0	47.2	36.2	17.0	53.4	39.5	43.8	54.3	37.3	64.8
Hepatitis D[a]	0.0	0.0	0.0	0.0	0.0	0.5	0.0	0.3	0.0	0.0	0.1	0.3	0.0	0.2	0.0
Hepatitis E[a]	0.2	0.0	0.0	0.1	0.0	0.5	0.0	0.0	0.2	0.0	0.5	0.3	0.0	1.0	0.0
Influenza	53.7	28.8	44.9	106.9	33.5	58.4	50.3	109.1	61.2	152.7	70.1	55.2	67.2	110.7	93.1
Influenza – Type A[a]	40.0	20.7	28.9	69.5	26.5	44.4	34.9	80.4	45.4	97.7	47.1	38.1	48.4	78.1	76.7
Influenza – Type B[a]	12.8	7.5	9.6	36.4	6.4	13.1	15.1	27.2	14.8	53.0	21.0	16.0	14.8	30.4	13.4
Influenza – Type A&B[a]	0.0	0.0	0.0	0.2	0.3	0.0	0.0	1.2	0.0	0.0	2.0	0.0	1.0	0.2	0.4
Influenza – Type NOS[a]	0.9	0.6	6.4	0.8	0.3	0.9	0.3	0.3	1.0	2.0	0.0	1.1	3.0	2.0	2.6
Legionellosis	1.2	0.9	0.0	0.5	3.4	0.0	0.3	1.5	1.2	0.0	2.0	0.8	1.0	1.8	1.9
L. longbeachae[a]	0.2	0.9	0.0	0.2	2.1	0.0	0.3	0.6	0.4	0.0	0.4	0.2	0.5	0.5	0.4
L. pneumophila[a]	1.0	0.0	0.0	0.2	1.3	0.0	0.0	0.9	0.8	0.0	1.6	0.6	0.5	1.1	1.5
Legionnaires' disease – other	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0
Leptospirosis[a]	0.0	0.0	0.0	0.3	0.8	0.9	5.5	0.0	0.0	2.0	0.2	0.0	1.0	0.0	1.5
Listeriosis[a]	0.0	0.3	0.0	0.2	0.5	0.5	0.3	0.6	0.2	0.0	0.5	0.3	0.0	0.2	0.0
Lymphogranuloma venereum[a]	2.2	0.0	0.0	0.0	0.0	0.0	0.0	0.6	0.2	0.0	1.6	0.1	0.0	0.7	0.0
Malaria[a]	1.2	0.6	0.0	1.2	0.5	1.4	1.0	1.2	0.4	0.3	1.1	0.7	0.0	2.5	0.7
Measles	1.6	0.6	0.0	0.2	3.1	0.0	0.0	0.0	0.2	1.0	1.4	1.3	3.0	3.0	0.0
Meningococcal disease	1.0	0.6	0.0	1.6	1.6	0.0	1.0	1.2	1.1	0.3	0.6	0.7	0.5	0.8	2.2
Meningococcal – serogroup B[a]	0.5	0.3	0.0	1.2	0.8	0.0	0.7	0.9	0.5	0.3	0.4	0.6	0.5	0.5	0.7
Meningococcal – serogroup C[a]	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0
Meningococcal – serogroup W135[a]	0.0	0.3	0.0	0.0	0.0	0.0	0.3	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.4
Meningococcal – serogroup Y[a]	0.2	0.0	0.0	0.1	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0
Meningococcal – other	0.3	0.0	0.0	0.2	0.5	0.0	0.0	0.3	0.5	0.0	0.2	0.0	0.0	0.2	1.1
Meningococcal – conjunctivitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0
Mumps[a]	1.7	0.0	0.0	0.0	0.3	0.5	0.3	0.9	1.1	1.3	1.9	0.6	1.0	1.4	0.0
Paratyphoid[a,c]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Pertussis	100.0	106.5	170.1	87.2	275.5	145.5	433.7	323.8	182.4	226.3	157.0	137.7	212.2	178.0	257.6
Pneumococcal disease (invasive)[a]	6.2	7.2	6.4	7.7	9.0	4.2	6.2	10.4	6.1	6.0	8.5	5.7	7.9	4.8	15.3
Psittacosis[a]	0.0	0.0	0.0	0.3	0.3	0.0	0.0	1.4	0.4	0.3	0.0	0.1	0.0	0.4	0.4
Q fever[a]	0.2	0.3	3.2	3.0	4.1	6.1	1.7	0.3	0.2	6.0	0.0	0.2	4.4	0.0	4.1
Rotavirus[a]	15.2	8.8	6.4	14.7	4.4	8.4	6.2	20.0	26.3	19.1	14.1	6.1	4.9	14.8	31.3
Rubella	0.3	0.3	0.0	0.1	0.0	0.0	0.0	0.0	0.4	0.0	0.5	0.2	0.0	0.4	0.0
Congenital rubella[a]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella – other[a]	0.3	0.3	0.0	0.1	0.0	0.0	0.0	0.0	0.4	0.0	0.5	0.2	0.0	0.4	0.0
Salmonella infection[a,c]	51.5	40.2	48.1	42.6	31.4	58.0	67.8	31.8	54.2	58.4	50.1	52.3	38.0	41.1	33.1
Shigellosis[a]	4.5	1.6	0.0	0.3	1.3	0.9	0.3	1.2	1.9	0.7	3.7	1.3	1.0	2.2	1.5
Syphilis	31.7	5.3	22.5	3.0	9.6	1.4	0.7	6.1	3.2	1.7	25.3	9.3	7.9	9.3	9.6
Congenital syphilis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.7
Syphilis infection[a,d]	20.6	0.3	0.0	1.1	3.9	1.4	0.0	2.6	1.7	0.7	20.6	2.0	4.9	2.5	1.5
Syphilis – other[a]	11.1	5.0	22.5	1.9	5.7	0.0	0.7	3.5	1.5	1.0	4.7	7.3	3.0	6.7	7.4
Tetanus	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tuberculosis[a,e]	5.9	1.3	3.2	1.0	3.6	1.9	4.1	2.6	4.5	0.7	7.6	1.5	2.0	8.8	1.1
Typhoid[a]	1.6	0.0	0.0	0.2	0.3	0.0	0.0	0.3	0.4	0.0	0.8	0.6	0.0	1.8	0.0
Verotoxin-producing *Escherichia coli* infections[a]	0.2	0.0	0.0	0.3	0.3	0.0	0.7	0.0	0.0	0.3	0.0	0.1	0.5	0.0	0.0

Onset of illness: the earlier of patient reported onset date, specimen date or date of notification.
[a]Laboratory-confirmed cases only.
[b]Foodborne illness cases are only those notified as part of an outbreak.
[c]From 2005, all paratyphoid recorded as salmonellosis.
[d]Includes syphilis primary, syphilis secondary, syphilis < 1 y duration and syphilis newly acquired.
[e]Tuberculosis data for 2011 was incomplete at the time of report and subject to change.
NOS: not otherwise specified.
No case of the following diseases have been notified since 1991: plague[a], diphtheria[a], granuloma inguinale[a], lyssavirus[a], poliomyelitis[a], rabies, smallpox, typhus[a], viral haemorrhagic fever, yellow fever.
Source: Notifiable Conditions Information Management System, Centre for Health Protection, NSW Health.

Table 6. Disease notifications by age group and sex of the case, NSW, 2011 (based on onset of illness)

Condition	0–4 yrs		5–24 yrs		25–44 yrs		45–64 yrs		65+ yrs		Total		Total[f]
	F	M	F	M	F	M	F	M	F	M	F	M	
Adverse event after immunisation	37	39	24	12	12	4	20	1	39	15	132	71	204
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0
Arboviral infection	4	1	77	69	203	196	213	228	66	112	563	606	1171
Barmah Forest virus infection[a]	1	0	24	29	73	65	88	100	27	49	213	243	457
Ross River virus infection[a]	3	1	32	21	108	98	103	109	36	59	282	288	571
Other[a]	0	0	21	19	22	33	22	19	3	4	68	75	143
Blood lead level ≥15 ug/dl[a]	12	14	5	43	4	111	3	57	1	5	25	230	257
Botulism	1	1	0	0	0	0	0	0	0	0	1	1	2
Brucellosis[a]	0	0	0	2	0	1	0	2	1	0	1	5	6
Chancroid[a]	0	0	0	0	0	0	0	0	0	0	0	0	0
Chlamydia trachomatis infection	36	19	8061	4187	3305	3924	193	706	6	49	11 601	8885	20 536
Congenital chlamydia[a]	19	11	0	0	0	0	1	0	0	0	20	11	31
Chlamydia – other[a]	17	8	8061	4187	3305	3924	192	706	6	49	11 581	8874	20 505
Cholera[a]	0	0	0	0	0	0	0	0	0	0	0	0	0
Creutzfeldt-Jakob disease[a]	0	0	1	0	0	0	0	2	2	4	3	6	9
Cryptosporidiosis[a]	55	87	46	50	43	46	11	14	5	2	160	199	359
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0
Foodborne illness (NOS)[b]	0	0	0	0	0	0	0	0	0	0	0	0	0
Gastroenteritis (institutional)	0	0	0	0	0	0	0	0	0	0	0	0	0
Giardiasis[a]	248	353	206	257	451	380	175	152	82	47	1162	1189	2367
Gonorrhoea[a]	1	0	244	576	262	1281	54	428	2	24	563	2309	2879
Haemolytic uraemic syndrome	0	1	0	0	0	1	0	0	1	1	1	3	4
H. influenzae type b[a]	1	3	0	0	0	0	0	0	0	0	1	3	4
Hepatitis A	1	4	9	16	7	8	5	4	2	1	24	33	57
Hepatitis B	2	2	159	176	616	690	266	426	68	68	1111	1362	2540
Hepatitis B – acute viral[a]	0	0	2	2	8	14	3	1	1	0	14	17	31
Hepatitis B – other[a]	2	2	157	174	608	676	263	425	67	68	1097	1345	2509
Hepatitis C	3	6	148	162	606	1105	386	792	49	53	1192	2118	3329
Hepatitis C – acute viral[a]	0	1	9	3	10	15	3	2	1	0	23	21	44
Hepatitis C – other[a]	3	5	139	159	596	1090	383	790	48	53	1169	2097	3285
Hepatitis D[a]	0	0	1	0	1	3	1	1	1	0	4	4	8
Hepatitis E[a]	0	0	2	3	3	4	3	5	0	0	8	12	20
HIV infection[a]	0	0	1	39	18	205	2	61	0	4	21	309	330
Influenza	427	506	829	795	863	603	534	460	298	298	2951	2662	5625
Influenza – Type A[a]	322	393	462	419	648	449	437	385	220	213	2089	1859	3955
Influenza – Type B[a]	105	110	355	367	202	141	83	63	66	70	811	751	1566
Influenza – Type A&B[a]	0	1	3	3	4	0	2	1	6	9	15	14	29
Influenza – Type NOS[a]	0	2	9	6	9	13	12	11	6	6	36	38	75
Legionellosis	0	0	0	0	7	12	12	21	11	27	30	60	91
L. longbeachae[a]	0	0	0	0	2	4	5	8	6	6	13	18	31
L. pneumophila[a]	0	0	0	0	4	8	7	11	5	21	16	40	57
Legionnaires' disease – other	0	0	0	0	1	0	0	2	0	0	1	2	3
Leprosy	0	0	0	0	0	2	0	0	0	0	0	2	2
Leptospirosis[a]	0	0	2	3	2	9	1	16	1	5	6	33	39
Listeriosis[a]	0	0	0	0	2	0	2	3	5	9	9	12	21
Lymphogranuloma venereum[a]	0	0	0	0	0	18	0	19	0	2	0	39	39
Malaria[a]	0	0	4	11	9	30	5	10	0	7	18	58	76
Measles	12	9	25	16	14	14	0	0	0	0	51	39	90
Meningococcal disease	9	15	13	13	5	3	3	3	5	2	35	36	71
Meningococcal – serogroup B[a]	4	9	11	8	1	1	2	2	3	2	21	22	43
Meningococcal – serogroup C[a]	0	0	0	0	1	0	0	0	1	0	2	0	2
Meningococcal – serogroup W135[a]	3	0	0	0	0	0	1	0	0	0	4	0	4
Meningococcal – serogroup Y[a]	0	0	1	2	0	0	0	0	1	0	2	2	4
Meningococcal – other	2	6	1	3	3	2	0	1	0	0	6	12	18
Meningococcal – conjunctivitis	1	0	0	0	0	0	0	0	0	0	1	0	1
Mumps[a]	0	1	11	10	14	16	5	7	0	1	30	35	65
Paratyphoid[a,c]	0	0	0	0	0	0	0	0	0	0	0	0	0
Pertussis	1228	1169	3544	3448	1213	583	764	508	329	228	7078	5936	13 053
Pneumococcal disease (invasive)[a]	32	38	19	22	33	34	72	86	94	93	250	273	524
Psittacosis[a]	0	1	0	2	2	2	4	6	0	2	6	13	19
Q fever[a]	0	1	3	9	6	29	13	43	3	7	25	89	114
Rotavirus[a]	231	326	111	116	45	28	46	32	71	44	504	546	1054
Rubella	0	0	4	1	6	5	0	1	0	0	10	7	17
Congenital rubella[a]	0	0	0	0	0	0	0	0	0	0	0	0	0
Rubella – other[a]	0	0	4	1	6	5	0	1	0	0	10	7	17
Salmonella infection[a,c]	370	454	493	481	428	337	278	253	214	169	1783	1694	3486
Shigellosis[a]	7	5	10	9	15	35	10	30	2	8	44	87	132
Syphilis	1	3	12	33	55	339	39	205	35	52	142	632	775
Congenital syphilis	1	2	0	0	0	0	0	0	0	0	1	2	3
Syphilis infection[a,d]	0	0	3	21	7	252	4	124	0	4	14	401	416
Syphilis – other[a]	0	1	9	12	48	87	35	81	35	48	127	229	356
Tetanus	0	0	0	1	0	0	0	0	0	0	0	1	1
Tuberculosis[a,e]	2	2	31	30	65	74	18	33	16	35	132	174	306
Typhoid[a]	2	2	8	9	6	16	1	1	0	0	17	28	45
Verotoxin-producing *Escherichia coli* infections[a]	0	2	1	0	1	0	3	1	0	2	5	5	10

Onset of illness: the earlier of patient reported onset date, specimen date or date of notification.
[a]Laboratory-confirmed cases only.
[b]Foodborne illness cases are only those notified as part of an outbreak.
[c]From 2005, all paratyphoid recorded as salmonellosis.
[d]Includes syphilis primary, syphilis secondary, syphilis < 1 y duration and syphilis newly acquired.
[e]Tuberculosis data for 2011 was incomplete at the time of report and subject to change.
[f]Includes cases with unknown age and sex and people who identify as transgender.
NOS: not otherwise specified. F: female. M: male.
Institutional gastrointestinal outbreaks and foodborne illness are excluded as complete demographic data are not routinely collected.
No case of the following diseases have been notified since 1991: plague[a], diphtheria[a], granuloma inguinale[a], lyssavirus[a], poliomyelitis[a], rabies, smallpox, typhus[a], viral haemorrhagic fever, yellow fever.
Source: Notifiable Conditions Information Management System, Centre for Health Protection, NSW Health.

difficulty in controlling this vaccine-preventable disease. While notification rates were the highest on record, it is unclear to what extent this high rate is due to waning immunity, enhanced identification through better laboratory diagnosis, greater clinician awareness or whether other factors are at play
- 90 **measles** case notifications, of which nine (10%) were imported from overseas and the remainder were either linked to these cases or locally acquired. Two of the larger clusters of cases of measles were identified in schools within the boundaries of the Western Sydney and Greater Southern Local Health Districts. One case of measles encephalitis was reported. The number of measles cases reported in 2011 was the highest on record since 1998, the year of the Australian Measles Control Campaign. The immunisation of international travellers and immigrant groups with low immunisation rates should remain a key feature of the vaccine-preventable disease control strategies
- 71 **meningococcal disease** case notifications, the lowest number in recent years. Of these, 43 were due to serogroup B (61%), four were due to serogroup W135 (6%) and another four serogroup Y (6%); only two were due to serogroup C (3%) and 18 were caused by an unknown serogroup (25%). The two cases of meningococcal C disease were both in adults. Meningococcal notifications have been declining for more than a decade, mostly for meningococcal C disease for which a vaccine was introduced in 2003
- 65 **mumps** case notifications, an increase from the 39 reported in 2010. The highest monthly notifications were reported in December ($n = 11$), the most reported in any month since February 2008
- 524 **invasive pneumococcal disease** case notifications compared with 504 notifications in 2010. Serotype 19A was identified as the cause of infection in 56% of cases in children under 5 years of age and in 24% of the other case-patients.

Prevention activities

Highlights for infant immunisation in 2011 included:
- immunisation rates for children and adolescents remained high, with NSW reaching coverage benchmarks of 90% for children at 1 and 2 years of age, with an increase in coverage at 5 years of age to just below 90%
- immunisation coverage for Aboriginal children improved in the 2- and 5-year age groups.

Further work is required to improve coverage rates for Aboriginal children in the 1-year age group (Table 7).

In 2011, the NSW School-Based Vaccination Program vaccinated:
- 77% of Year 7 and 66% of Year 10 students with a booster dose of diphtheria-tetanus-pertussis vaccine

Table 7. Comparison of the proportion of children fully immunised according to the Australian Childhood Immunisation Register for three age groups, for all children in NSW and for Aboriginal children, in 2010 and 2011

	All children		Aboriginal children	
	2010	2011	2010	2011
1 year of age	91.4%	91.2%	86.6%	85.9%
2 years of age	92.4%	92.4%	91.9%	92.1%
5 years of age	87.6%	89.4%	82.3%	84.9%

- 81% of Year 7 girls with at least one dose of human papillomavirus vaccine and 71% of Year 7 girls with three doses of the vaccine
- 68% of Year 7 children with at least one dose of hepatitis B vaccine and 63% of Year 7 children with two doses of the vaccine (hepatitis B vaccination is only offered to children who have not previously received a full course)
- 45% of Year 7 children with varicella vaccine (varicella vaccination is only offered to children without a history of infection or vaccination).

These data do not include children who received these free vaccines from general practitioners (GPs) or other immunisation providers.

Initiatives to improve vaccination coverage in 2011 included:
- the introduction into the routine immunisation schedule of a 13-valent conjugate pneumococcal vaccine and implementation of a supplementary dose program for children who had received three doses of the 7-valent vaccine
- the continued provision of free pertussis vaccine for new parents, grandparents and other adults who regularly care for infants under 12 months of age, in an effort to indirectly protect those babies in light of the ongoing pertussis epidemic
- the development of strategies and health-system capacity to follow-up Aboriginal children who are overdue for vaccination
- the provision of free measles-mumps-rubella vaccine to unvaccinated people born in or since 1966 and to contacts of people with measles, to prevent further transmission in the community
- working with Divisions of General Practice, local councils and community health centres to improve coverage in areas of low vaccination coverage.

Bloodborne viruses

Notification data

In 2011 there was:
- a stable number of total **hepatitis B** case notifications ($n = 2540$); 51% were in people aged 25–44 years (total notifications are mainly of people whose time of infection

is unknown). However the number of hepatitis B notifications thought to be newly acquired has steadily declined over the last 5 years, from 56 reported in 2007 to 31 in 2011
- a slight decrease in **hepatitis C** case notifications (3329 notifications, down from 3816 in 2010). Case-patients were most commonly men aged 25–44 years (33%), men aged 45–64 years (24%) and women aged 25–44 years (18%). The number of notifications of newly acquired hepatitis C infections remained stable ($n = 44$, compared with 39 in 2010; the annual average over the previous 5 years is 45)
- a slight increase in case notifications of **human immunodeficiency virus** (HIV) with 330 reported in 2011 (compared to 307 in 2010). The age groups most affected were those aged 30–39 years ($n = 123$; 37%) and 20–29 years ($n = 90$; 27%). In 2011, 277 notifications (84%) were reported to be homosexually acquired.

Improvements in methods for data cleaning have identified duplicate notifications for hepatitis B and C cases, resulting in a more accurate count of cases and a reduction in the overall number of notifications for previous years, particularly for before 2005.

Prevention activities

NSW Health has a range of policies and strategies in place to control the spread of HIV, hepatitis B and hepatitis C, including regular campaigns to promote safer sex, needle and syringe programs to provide sterile equipment to injecting drug users, and support of the management of patients with sexually transmissible infections and hepatitis C. Highlights in 2011 included:
- ACON (formerly the AIDS Council of NSW) developed an HIV prevention campaign for gay men called *The Big Picture* (www.hivthebigpicture.org.au), designed to inform gay men in NSW about the risks for HIV transmission. It also offered the gay community strategies for keeping infection rates low: maintain condom use, increase HIV testing, encourage the disclosure of HIV status and restrict sex without condoms to men of the same HIV status
- the NSW Expanded Medication Access Scheme for HIV Section 100 drugs (Highly Specialised Drugs Program) to provide people with HIV with improved access to treatment in NSW. This scheme was developed in light of both long-standing and recent evidence of the effectiveness of HIV antiretroviral therapy in preventing the sexual transmission of HIV.[1] Under the new Scheme, patients who are stable on their medications and meet certain criteria can elect to have their medication delivered to any address of their choosing rather than attend a hospital pharmacy. A number of select retail pharmacies also operate as pick up locations for patients
- NSW publicly funded HIV and sexual health clinics provided 79 046 occasions of services to 6536 clients related to HIV treatment, management and care (an increase of 2.2% of occasions of services and 1.0% of clients compared with 2010)
- the first phase of a hepatitis C prevention campaign, aiming to increase awareness of the transmission and prevention of hepatitis C for young people. Brochures and posters were distributed to 750 general practices in NSW. In 2010/11, the NSW Needle and Syringe Program comprised over 940 outlets (332 public sector outlets, 159 dispensing machines and 450 pharmacies)
- Approximately 9.98 million needles and syringes were dispensed and over 13 400 referrals were provided to drug treatment services, hepatitis services and other health and welfare agencies for people who inject drugs
- 3014 patients with hepatitis C were referred for hepatitis C assessment, 1062 patients initiated treatment and 989 patients completed treatment.

Sexually transmissible infections

Notification data

In 2011 there was:
- a similar number of **infectious syphilis** case notifications compared with recent years (416, compared with 419 in 2010, and a 5-year average of 412)
- a decrease in the number of **lymphogranuloma venereum** (LGV) case notifications (39, compared with 56 in 2010). All notifications were reported in males, and 37 (95%) case-patients were aged between 25 and 64 years. The number of notifications decreased in late 2010 following an outbreak earlier in that year. The outbreak in NSW occurred within the global context of increased European rates of LGV infection in men who have sex with men[2]
- a continued increase in the number of **chlamydia** case notifications (20 536, up 13% from 18 254 in 2010); 57% of cases notified were female and 59% of cases were aged between 15 and 24 years
- an increase in the number of **gonorrhoea** notifications (2879, up 26% from 2301 in 2010). Men made up 80% of cases and 54% of these case-patients were aged 25–44 years.

Overall, notifications of sexually transmissible infections (STIs) in NSW continue to rise with chlamydia continuing to be the most commonly notified STI in NSW. Much of the increase in chlamydia and at least some of the increase in gonorrhoea notifications may relate to increased screening and case detection.

Prevention activities

Highlights in 2011 included:
- the NSW STIs in Gay Men Action Group (STIGMA) (a partnership of state and local prevention agencies based in central and south eastern Sydney) developed

educational resources for updating GPs on recent research and epidemiology of STIs. STIGMA also distributed a campaign aimed at gay men promoting testing for STIs and contact tracing, based around the National Gay Men's Syphilis Action Plan: http://stigma.net.au/resources/National_Gay_Mens_Syphilis_Action_Plan.pdf
- NSW Health continued the second phase of the successful 2009 HIV and STI education campaign, *Get Tested, Play Safe*: http://www.gettested.com.au. The aim of the campaign was to reinforce STI awareness, increase testing and improve safer sex behaviour among young people. Television, radio, online and print advertising was used statewide and pilot partnerships with music festivals extended the reach of the campaign
- the NSW STI Programs Unit developed a *Sexually Transmissible Infections Contact Tracing Tool* for use in general practice. Copies were distributed to all GPs in NSW, and through the *NSW Public Health Bulletin* to selected local health districts.[3] The Tool is a quick reference guide to assist doctors to understand their contact tracing responsibilities, the steps involved in best practice contact tracing, and key points for the management of STI contacts. For more information see: http://www.stipu.nsw.gov.au/content/Image/May_2011_Contact_tracing_tool_final_version.pdf
- publicly funded sexual health clinics provided 25 851 occasions of service (an 8.2% increase compared to 2010) related to STI treatment, management and care, providing services to 12 930 clients (a 3.3% increase compared to 2010).

Enteric diseases (infectious, food and water)

Notification data

In 2011 there was:
- a 15% increase in enteric disease case notifications (6484) compared with the average annual count for the previous 5 years
- a 31% increase in salmonellosis case notifications (3486) compared with the annual average for the previous 5 years. The increase was in part explained by an ongoing increase in *Salmonella* Typhimurium 170 infections, a serovar previously associated with the consumption of contaminated eggs
- a decrease in the reports of outbreaks of probable foodborne disease (47 outbreaks affecting 797 people, compared with 59 outbreaks affecting 728 people in 2010)
- a stable number of reports of outbreaks of probable viral gastroenteritis in institutions (525 notifications affecting 9071 people, compared with 518 outbreaks affecting 9359 people in 2010)
- 10 point-source outbreaks of *Salmonella* Typhimurium, most likely associated with the consumption of sauces and desserts prepared with raw eggs.

Prevention activities

NSW Health works with OzFoodNet nationally and the NSW Food Authority locally to investigate and control foodborne outbreaks and food-contamination incidents, and to make prevention recommendations.

NSW Health is the public health regulator of major water utilities through operating licences and memoranda of understanding (Hunter Water Corporation, Sydney Water Corporation and Sydney Catchment Authority). In 2011 the Water Unit and public health units worked with these utilities to:
- ensure compliance with relevant guidelines including the *Australian Drinking Water Guidelines*[4] and the *Australian Guidelines for Water Recycling*[5]
- monitor compliance of utilities with the *NSW Fluoridation of Public Water Supplies Act 1957*.

The Water Unit and public health units also exercise public health oversight of more than 100 water utilities in regional NSW through the NSW Health *Drinking Water Monitoring Program*,[6] which provides guidance on drinking water monitoring and is supported by NSW Health laboratories. In 2011 regional sampling compliance was very good with:
- 97% of expected microbiological samples taken (compared with 96% in 2010)
- 100% of expected chemistry samples taken (the same as in 2010).

NSW Health has commenced a major upgrade of the web-based NSW Drinking Water Database that will help water utilities and public health units better manage drinking water quality. NSW Health is also supporting rural water utilities to develop risk-based drinking water management systems, which will be required under the *Public Health Act 2010*. In 2011, NSW Health helped four small water utilities develop management systems that will help ensure the safety of the drinking water supply. More water utilities will receive assistance in 2012.

NSW Health is responsible for reviewing licence applications from private recycled water or drinking water suppliers under the *Water Industry Competition Act 2006.* In 2011, the Water Unit and public health units:
- reviewed 14 licence applications for recycled water
- advised local councils and the NSW Office of Water on more than 20 new and ongoing recycled water schemes regulated under the *Local Government Act 1993*.

Respiratory disease (infectious and environmental)

Notification data

In 2011 there was:
- an increase in the number of **Legionnaires' disease** case notifications due to *Legionella pneumophila* (57 compared to 38 cases in 2010). Public health investigations did not identify a common source for these cases

- *L. pneumophila* cases peaked in April 2011 with 20 cases reported. Public health officers worked closely with local councils at this time to inform owners of registered cooling towers (one possible source of *L. pneumophila* bacteria) on the need to comply with regulations to minimise the risk of *Legionella* contamination. A statewide media release was also issued in May 2011 to reinforce this message
- an increase in the number of notifications of **influenza** (5625 compared to 1601 notifications in 2010). As only laboratory confirmed cases of influenza are notifiable and only a very small proportion of all people with influenza are tested, it is difficult to draw conclusions about the true level of influenza activity in the community based on these data. There were at least 61 admissions to intensive care units for treatment of influenza-associated illnesses
- a cluster of **influenza isolates resistant to influenza antiviral medications**. Routine resistance testing of a selection of NSW influenza A samples detected 31 influenza A pandemic (H1N1) 2009 virus isolates with the H275Y neuraminidase mutation associated with resistance to oseltamivir and peramivir. None of these showed resistance to zanamivir. Of these isolates, 29 were collected from patients in the Hunter region whose ages ranged from 4 months to 58 years, and included three pregnant women. Of these case-patients, six required hospitalisation. There were **no deaths.**
- **a decrease in the number of notifications of tuberculosis** (306 compared to 468 cases in 2010). At the time of this report the tuberculosis data for 2011 remained incomplete. Five cases of multidrug resistant tuberculosis (MDR TB) were identified, a similar number as in previous years (five in 2010 and 10 in 2009).

Prevention activities

Highlights in 2011 included:

- a campaign that focused on three respiratory disease prevention messages: *Cover your face when you cough or sneeze*; *Wash your hands*; and *Stay at home if you're sick so you don't infect others*. The campaign included radio advertising, advertisements on public transport, digital media and the distribution of *The Spread of Flu is Up to You* posters
- seasonal influenza vaccine provided free under the National Immunisation Program for people at high risk of severe influenza complications. The NSW Health Population Health Survey estimated that 32% of all respondents (95% confidence interval [CI]: 30–35) interviewed during August and September 2011 had received a seasonal influenza vaccine in the previous 12 months, a slight increase in vaccine uptake compared to the estimate for the same period in the previous year (29% [95% CI: 27–32]). For respondents aged 65 years and over (one of the identified high-risk groups), the estimated vaccination rate was 73% (95% CI: 69–77), which is similar to the rate for previous years.

Vectorborne diseases

Notification data

In 2011 there was:

- a decrease in Ross River virus infection notifications (571, a 46% decline compared with 1067 in 2010)
- an increase in the number of Barmah Forest virus infection notifications (457, 79% higher than the 255 notified in 2010)
- two confirmed cases of locally acquired Murray Valley encephalitis, from the Western NSW and Hunter New England Local Health Districts, the first reported cases since 2008
- one Kunjin virus infection case notification, probably acquired in the Illawarra Shoalhaven Local Health District, the first NSW case notified since 2001
- a 40% decrease in the number of dengue fever case notifications in 2011 (130 compared with 218 in 2010). All of the dengue fever cases in 2011 were linked to international travel, with travel to Indonesia the most commonly reported exposure site (48%), followed by Thailand (10%), India and the Philippines (both 8%). While there is no local transmission of dengue fever in NSW, it is the most common mosquitoborne viral disease in humans worldwide, and represents a major international public health concern
- a 38% decrease in the number of malaria case notifications (76, compared with 122 in 2010). Travel to Papua New Guinea was the most commonly reported exposure site (25%), followed by India (13%) and Ghana (11%).

The NSW arbovirus surveillance program includes: mosquito trapping and the monitoring of virus activity in mosquito populations; and, monitoring for antibodies to Murray Valley encephalitis and Kunjin virus infection in sentinel chicken flocks located in a number of strategic sites in rural NSW from mid-spring to mid-autumn when transmission of arbovirus infections is most common.

With increasing international travel, exotic mosquitoborne diseases such as dengue fever, malaria and chikungunya will pose an ongoing risk for people travelling to endemic areas. International trade also increases the risk of the importation of exotic mosquito species which are able to transmit these infections locally.

Prevention activities

- In 2011 statewide media releases warning about the increased risk of mosquitoborne infections and how to prevent them were issued in February, March, April and December. These were supplemented by a *Fight the Bite* public education campaign in high-risk districts incorporating radio advertising, the distribution of posters and brochures, and a range of local media messaging by Public Health officials. See http://www.health.nsw.gov.au/resources/publichealth/environment/hazards/pdf/ftb_hr_cl.

Zoonotic diseases

Notification data

In 2011 there was:

- a modest decrease in Q fever case notifications (114 compared with 137 in 2010). Q fever was the most commonly notified zoonotic disease in 2011 (114 case notifications)
- a slight increase in brucellosis infections (six, compared with three in 2010). Three cases were acquired overseas, two infections were in feral pig hunters in northern NSW and the source of one case was unknown
- no human case of Hendra virus infection in humans, despite 10 fatal cases in horses in northern NSW. NSW Health worked closely with the North Coast Public Health Unit and the Department of Primary Industries to investigate and control outbreaks and prevent infection in humans.

Environmental exposures and risk assessment

On 8 August 2011, an accident at the Orica ammonium nitrate plant on Kooragang Island, Newcastle, resulted in the deposition of a chemical called hexavalent chromium on an area of Stockton that lay directly downwind of the plant. Hunter New England Population Health and the Ministry of Health's Environmental Health Branch, assisted by an expert panel, assessed the health risks associated with this event, provided health information to the public and advice to other agencies on matters such as the cleanup of the chemical deposition. As a result of this incident, a number of changes have been made to strengthen the NSW Government response to pollution incidents. First, the Environment Protection Authority has been reconstituted as an independent entity. Second, several amendments have been made to the *Protection of the Environment Operations Act 2007*. An important change is that polluters are now required to notify NSW Health immediately of any incidents causing, or threatening to cause, material harm to the environment.

Aboriginal health

NSW Housing for Health

Housing for Health is an evidence-based housing repair and maintenance program that focuses on improving the safety and health of residents.[7] Since 1998, over 11 500 Aboriginal people living in nearly 2600 houses in 78 Aboriginal communities have benefited from the Housing for Health program. Nearly 75 000 items that relate to improved safety and health have been repaired through the program. An evaluation of the NSW Housing for Health program found that populations exposed to the program were 40% less likely to be hospitalised with infectious diseases compared with the rest of the rural NSW Aboriginal population.[8]

In 2011 the Housing for Health program:

- completed projects in Bourke, Enngonia, Wilcannia, La Perouse and Coffs Harbour. The Coffs Harbour project was a trial program with the Aboriginal Housing Office and Housing NSW to integrate Housing for Health with the broader Aboriginal Housing Office Backlog and Maintenance Program
- commenced new projects in Purfleet and Walhallow.

Aboriginal Communities Water and Sewerage Program

Clean water and functioning sewerage systems are a prerequisite for good health. Widespread availability of these essential services improves health by reducing communicable diseases such as skin infections and diarrhoeal illness. The Aboriginal Communities Water and Sewerage Program is a joint partnership between the NSW Government and the NSW Aboriginal Land Council.[9] The Program aims to ensure adequate operation, maintenance and monitoring of water supplies and sewerage systems in more than 60 Aboriginal communities in NSW. NSW Health is involved in the development and implementation of the Program.

In 2011:

- a further seven Aboriginal communities with a combined population of around 1000 people began receiving improved water and sewerage services, bringing the total to 38 communities of over 4000 people who have received improved water and sewerage services under the Program. Public health units are working with communities, the NSW Office of Water, local water utilities and service providers to implement Risk-Based Water and Sewerage Management Plans
- an Aboriginal traineeship has been approved by the Aboriginal Communities Water and Sewerage Program steering committee to ensure that Aboriginal people obtain the necessary skills for employment with local water utilities. Funding of $66 000 each year for the next 2 years will be spent for training eight Aboriginal people. This initiative is being managed by Aboriginal Affairs NSW.

The Aboriginal Environmental Health Officer Training Program

The Aboriginal Environmental Health Officer Training Program aims to increase opportunities for workforce participation by Aboriginal people and enhance the involvement of Aboriginal people in improving environmental health outcomes. The Program also contributes to addressing current workforce shortages in environmental health. Since 1998, 11 Aboriginal Environmental Health Officers have graduated from the Program.

In 2011:

- seven Aboriginal Environmental Health Officer Trainees were participating in the Program. The percentage of Aboriginal people employed within the NSW Health Environmental Health workforce increased from 0% ($n = 0$) in 1998 to over 20% ($n = 12$) in 2011.

The program is now being expanded in partnerships with Local Government to increase Aboriginality in the overall NSW environmental health workforce (much of which is based in Local Government)
- four new trainee positions were created under funding agreements between the Aboriginal Environmental Health Unit, regional public health units and Local Governments.

Negotiations are in place for further Local Government trainee positions which will start during 2012.

Acknowledgment

Protecting the health of the community is a collaborative effort, involving public health units, clinicians, laboratory scientists, affected communities, and other government and community-based organisations. We thank all those involved for the role they played in NSW in 2011.

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*The *Public Health Act 2010* (NSW) (http://www.health.nsw.gov.au/phact/)
The *Public Health Act 2010* (NSW) was passed by the NSW Parliament in December 2010 and commenced on 1 September 2012. The Public Health Regulation 2012 was approved in July 2012 and commenced, along with the *Public Health Act 2010* (NSW), on 1 September 2012. The objectives of the Regulation are to support the smooth operation of the Act. The Act carries over many of the provisions of the *Public Health Act 1991* (NSW) while also including a range of new provisions.

Sample size calculations for the design of health studies: a review of key concepts for non-statisticians

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Abstract: Sample size calculations before conducting a health study or clinical trial are important to provide evidence that the proposed study is capable of detecting real associations between study factors. This review aims to clarify statistical issues related to the calculation of sample sizes and is illustrated with an example of a recent study design to improve health outcomes related to water and sewage in NSW Aboriginal communities. The effect of power, significance level and effect size on sample size are discussed. Calculations of sample sizes for individual-based studies are modified for more complex trial designs by multiplying individual-based estimates by an inflationary factor.

Sample size calculations are an important consideration when designing a health study.[1,2] Investigators need to provide suitable calculations to ensure that a study is capable of detecting a real effect due to an intervention. While there are articles available to assist researchers who have some statistical background with sample size calculations,[2] there are few available for those with limited statistical knowledge. This review is based on a literature review of relevant articles that the authors have found useful. It provides a background understanding for the researcher to be able to more easily communicate with the statistician during the sample size calculation process. We introduce important concepts in a clear and non-technical account to a reader who is uneasy with basic statistics. Suitable references will be given to enable the interested reader to go beyond the scope of this review.

While studies may be conducted to examine differences between treatment groups or to estimate some population statistic,[1] here we focus on the former. We introduce the reader to the steps involved in calculating a sample size for an individual-based randomised control trial with treatment and control groups and a binary outcome (two categories). These principles apply to other types of outcomes. The review also discusses the calculation of sample sizes for more complex study designs.

Calculation of sample sizes for studies in which individuals are randomised

Three fundamental factors are involved in calculating sample sizes: significance level, power and effect size (defined in Table 1). We recommend Kirby et al. for a more detailed discussion.[2] When consulting a statistician for a sample size calculation, a researcher can help assist the process with a knowledge of these three parameters. Various sample size calculators are available online, which further explain the relationship of these three components to sample size (these tools should be used with appropriate statistical advice).[3]

The process for calculating a sample size is:[4]

1. Specify the null and alternative hypotheses, power, effect size and significance level.
2. Define the study population.
3. Estimate the required parameters (e.g. means, standard deviations) from the available data. These estimates are often derived from pilot studies and literature searches.
4. Calculate a range of sample sizes for a range of parameters (to provide different scenarios).
5. Choose the most appropriate sample size from these scenarios, given the study constraints.

Example

A proposed study to examine the intervention of improved water and sewage on health outcomes in discrete NSW Aboriginal communities (Aboriginal Communities Water and Sewage Program Health Outcomes Evaluation) provides an illustration of sample size calculations. The health outcome under consideration is the presence of intestinal infections. The measure for the study is expressed as a relative risk (RR), which is the ratio of the probability of intestinal infections in the Aboriginal communities before and after the intervention. Sample size formulae for binary outcomes (presence or absence of

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Table 1. The fundamental components of sample size estimation

Component	Definition	Example
Null hypothesis	A statement that the intervention has no effect (treatment groups are equivalent), defined in terms of an appropriate measure calculated for the treatment and control groups.	Examples include differences in means or probabilities, relative risks and hazard ratios.
Significance level	The significance level (α) is defined as the chance that the study will incorrectly report that the two treatment groups differ when they are equivalent (Type I error, false positive).	Typical values of α include 5% and 1%. If the study (at the 5% level) was rerun 20 times, we expect to incorrectly reject the null hypothesis once.
Power	Power is defined as the chance that the study will correctly report that the two treatment groups differ. The power is the chance that the study will **not** make a Type II error (a false negative).	Common values of power include 80% and 90%. In practice, power and significance level involve trade-offs with one another. Increasing power will come at the cost of a higher significance level.
Effect size	The **alternative hypothesis** is the hypothesis that the two treatment groups differ by at least some pre-specified amount. This amount is the effect size (δ), the detectable difference between the two treatment groups.	

Table 2. Total (treatment and control) sample sizes for various effect sizes for studies in which individuals are randomised, assuming the probability of intestinal infection before intervention to be 0.051 and equal numbers in the two groups, using the Housing for Health study*

	Effect size			
	Worst case	Housing for Health intervention*	Intermediate case	Best case
Effect size (reduction)	20%	43%	50%	60%
Relative risk	0.80	0.57	0.50	0.40
Power = 80%, α = 10%	10 798	2154	1550	1034
Power = 80%, α = 5%	13 604	**2686**	1928	1280
Power = 80%, α = 1%	20 052	3912	2796	1844
Power = 90%, α = 10%	14 806	2914	2090	1384
Power = 90%, α = 5%	18 078	3536	2530	1670
Power = 90%, α = 1%	25 440	4932	3518	2314

α: significance level.
Note: sample sizes are rounded up to be conservative.
*Closing the gap: 10 years of Housing for Health in NSW. NSW Health 2010.

intestinal infections) are given in Wittes and Campbell et al. (with formulae for other situations).[4,5]

Sample size calculations are based on a set of assumptions. For this example we assume that information from the previous Housing for Health in NSW study[6] holds true for our proposed study. From this study, we estimate the probability of intestinal infection before the intervention as 0.051. We assume that there are equal numbers of people in both the treatment and the control groups, significance level 5% and power 80%. We alter effect size (the difference between probabilities before and after intervention) to give us a range of sample sizes corresponding to different scenarios. In addition to the reduction in the prevalence of intestinal infections of 43% seen in the Housing for Health in NSW study, we also present a worst case of a 20% reduction, an intermediate reduction of 50% and a best reduction of 60%. The resulting sample sizes for the Aboriginal Communities Water and Sewage Program Health Outcomes Evaluation are calculated from formulae 7B and 7C in Wittes[4] (Table 2). From Table 2, we see that the smaller the detectable difference, the larger the sample size required (if all other parameters are held constant). The Housing for Health in NSW study reported a relative

reduction of 43%; the probability of intestinal infection after the intervention is $(1-0.43)\times 0.051=0.029$. The absolute effect size is $0.051-0.029=0.022$. The corresponding sample size is 2686 (Table 2).

Figure 1 shows the effect that power, significance level and effect size have on sample size. Figure 1(a) shows the relationship of different effect sizes on sample size. A decreasing relative reduction means a smaller difference to be detected between treatment and control outcomes which requires a larger sample size. The effect of significance level is shown in Figure 1(b). Ideally, a study should mistakenly reject a true null hypothesis of no treatment effect as few times as possible. For this to occur, a smaller significance level and consequently a larger sample size are required. Figure 1(c) shows the effect of power on sample size. Increased power means a study is more likely to correctly reject a null hypothesis of no treatment effect and a larger sample size is required. A study with more precise estimates of treatment effects will have higher power and lower significance level; this situation comes at the cost of a larger sample size. We recommend Kirby et al. to describe the relationship of significance level, power and effect size on sample size.[2]

Calculation of sample sizes for studies in which clusters of individuals are randomised

The Aboriginal Communities Water and Sewage Program Health Outcomes Evaluation study is a more complicated design as the community (not the individual) receives the intervention. The intervention is an improved water and sewage program. Such an intervention cannot feasibly be delivered to individuals. The clusters are communities and the intervention is randomised to clusters. The sample size calculation for a cluster study involves calculating the corresponding sample size for an individual study and multiplying this by an inflationary factor to account for

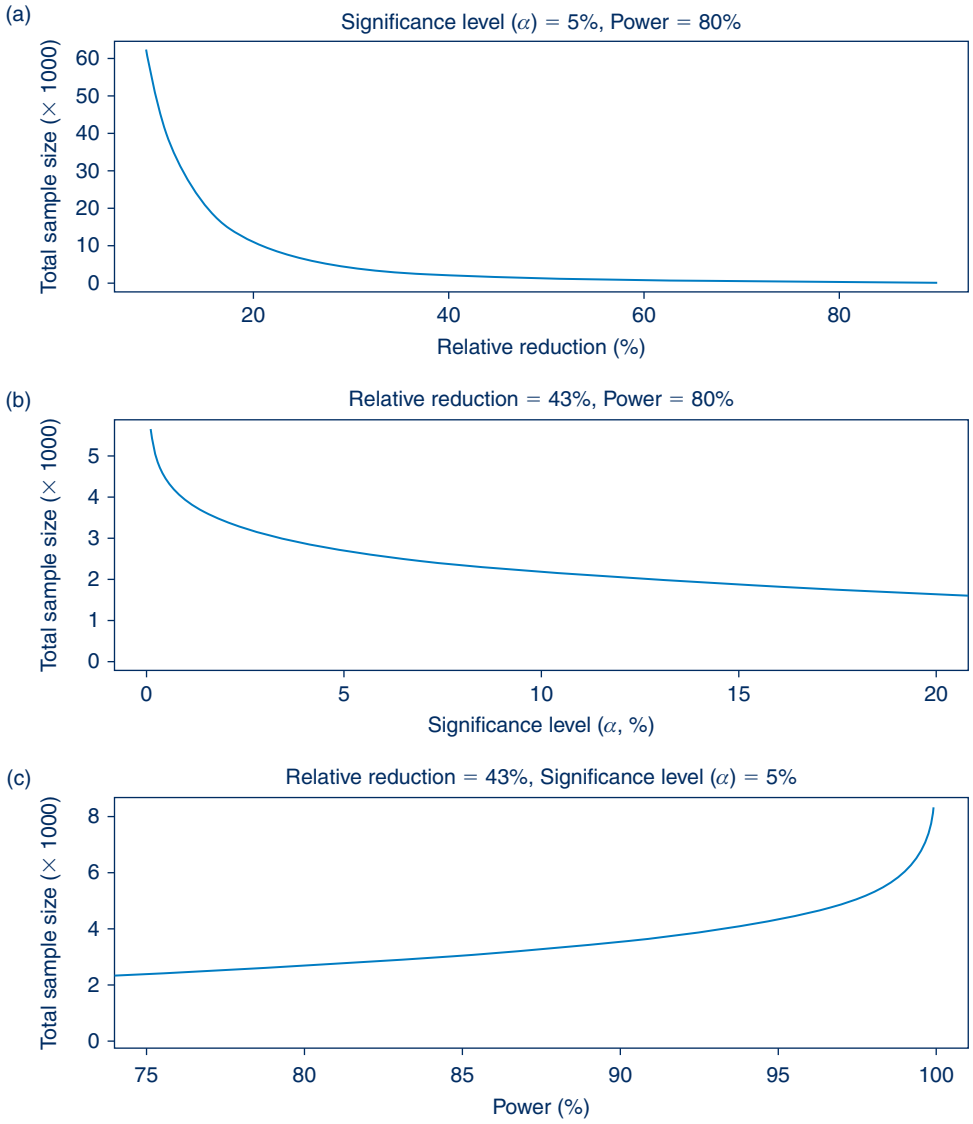

Figure 1. The effect of effect size (a), significance level (b) and power (c) on sample size. Calculations assume the probability of intestinal infection before the intervention to be 0.051 and equal numbers in the two groups.

the more complex trial design.[7–10] This inflationary factor is called the **design effect** (DE). Eldridge et al. provide formulae for design effects for various continuous (e.g. blood pressure, weight) and binary (e.g. whether the patient has the disease or not) outcomes.[7] The estimation of a design effect for cluster randomised control trials involves three factors: mean size of clusters, variation of cluster size and **intra-cluster correlation** (ICC).

The intra-cluster correlation can be regarded as a measure of the degree of similarity in outcomes between clusters.[11] There have been previous papers presenting intra-cluster correlations for different cluster units and populations.[12,13] Appropriate intra-cluster correlations for binary outcomes are discussed in Ridout et al.[14] These outcomes have an associated variance, which can be modelled as two components: variation in outcomes **between** clusters and variation in outcomes **within** each cluster. The intra-cluster correlation is the ratio of the between-cluster variation to total variation (the sum of the between and the within). The intra-cluster correlation is between 0 and 1. Small values of intra-cluster correlation imply that variation within clusters is much greater than variation between clusters and the clustering effect of individuals is less important. If the intra-cluster correlation is zero, outcomes can be regarded as being the same between clusters. The intra-cluster correlation is estimated from available data on cluster sizes and the number of outcomes (intestinal infections) within each cluster.

Example

Information about the size of clusters must be included in our study. We base this on the Housing for Health in NSW study.[6] The clusters are of different sizes and therefore we estimate a mean cluster size and cluster size variation (using standard deviation). From the Housing for Health in NSW study,[6] the mean cluster size = 150.7 and standard deviation = 103.5.

We estimated the intra-cluster correlation using the cluster information from the Housing for Health in NSW study (formula 7 in Ridout et al.[14]). The intra-cluster correlation is estimated as 0.007. We present estimated sample sizes in Table 3. In addition to the reduction in the prevalence of intestinal infections of 43% seen in the Housing for Health in NSW study, we also present a worst case of a 20% reduction, an intermediate of 50% and a best of 60% (assuming 80% power, 5% significance level and equal numbers in groups). We multiply the individual sample sizes presented in Table 2 by the design effect to obtain the estimates in Table 3. From Table 3, the corresponding sample size is 7074.

Figure 2 shows the relationships of intra-cluster correlation and cluster size on sample size. From Figure 2(a), it is apparent that the estimate of the intra-cluster correlation will have a large impact on sample size. As outcomes between clusters become more heterogeneous, the intra-cluster correlation increases. This decreases precision in the resulting outcome estimates from the clusters, and larger samples are thus needed. If the intra-cluster correlation is zero and there is no variation between clusters, the design effect (DE) = 1 and the resulting sample size is equivalent to an individual-level trial size.

Individual-level studies are more efficient than cluster-level studies[7] which is reflected by the larger sample size in response to increased (mean) cluster size shown in Figure 2(b). All other things being equal, an increasing cluster size standard deviation results in increased sample size (Figure 2(c)). Intuitively, increased standard deviation reflects increasing disparity between the size of the clusters. Due to less precise estimates, a larger sample size is required. Trials are more statistically efficient for similar sized clusters and need smaller sample sizes. Larger samples are required for increasing mean and standard deviation.

Table 3. Total (treatment and control) sample sizes for various scenarios for studies in which clusters of individuals are randomised. Corresponding design effects are shown in brackets. Sample sizes are derived from Table 2 (design effect multiplied by sample size with 80% power and 5% significance level, subject to rounding), using the Housing for Health study*

	Worst case	Housing for Health intervention*	Intermediate case	Best case
Effect size (reduction)	20%	43%	50%	60%
Relative risk	0.80	0.57	0.50	0.40
ICC = 0.001 (DE = 1.22)	16 620	3282	2356	1564
ICC = 0.005 (DE = 2.11)	28 680	5662	4064	2698
ICC = 0.007 (DE = 2.63)	35 830	**7074**	5078	3372
ICC = 0.01 (DE = 3.21)	43 754	8638	6202	4116
ICC = 0.05 (DE = 12.04)	164 356	32 450	23 294	15 464
ICC = 0.1 (DE = 23.16)	315 108	62 216	44 658	29 648

ICC: intra-cluster correlation.
DE: design effect.
*Closing the gap: 10 years of Housing for Health in NSW. NSW Health 2010.

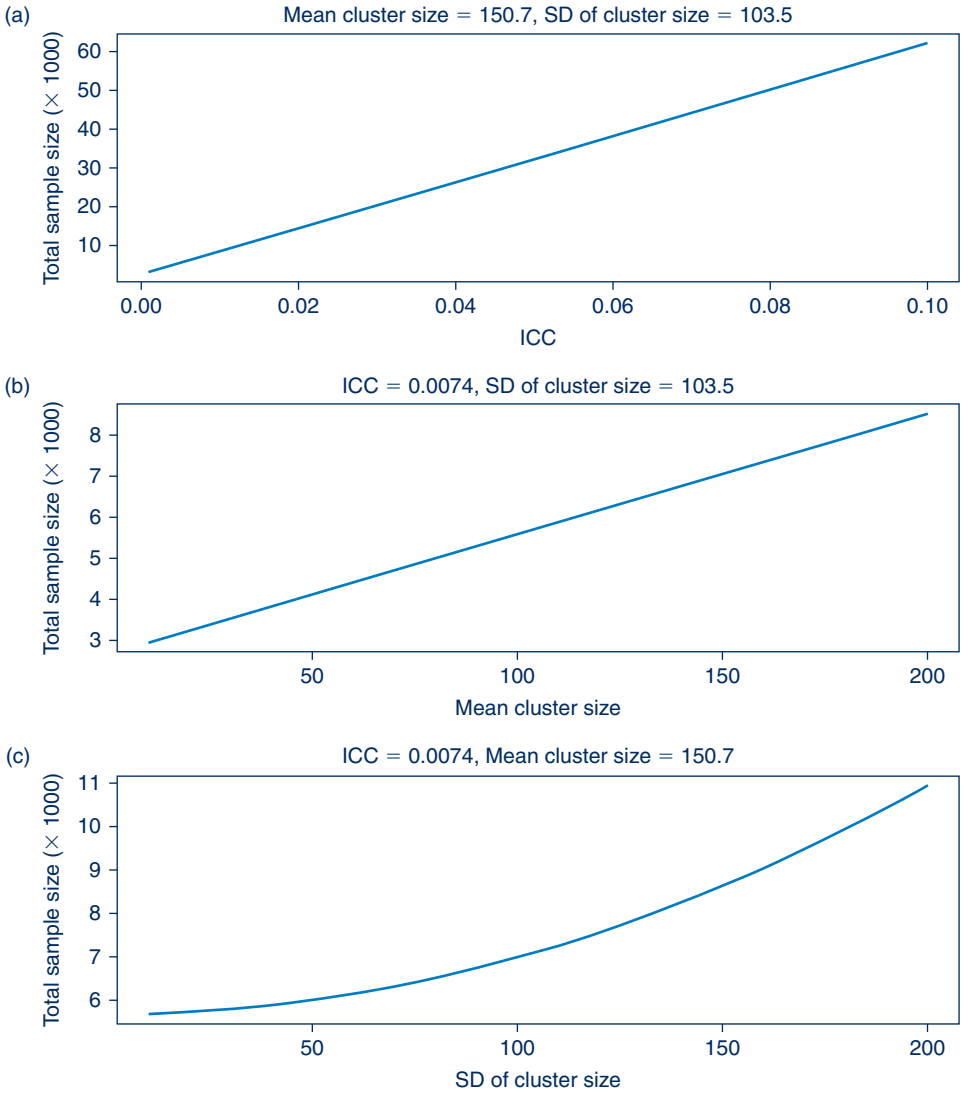

Figure 2. Effect of intra-cluster correlation (ICC) (a), mean cluster size (b) and standard deviation (SD) of cluster size (c) on sample size. Calculations assume the parameter estimates from the Housing for Health in NSW study are correct, reduction of 43%, power = 80%, significance level = 5% and equal numbers in the two groups.

Other factors affecting sample size calculations

There are other important factors that need to be accounted for in sample size calculations, including losses to follow-up, unequal treatment group sizes and the noncompliance of subjects to the intervention.[1,2,4] If the study investigator is able to provide an estimate of these factors to the statistician, the calculation of the required sample size will be improved.

Discussion

The calculation of sample sizes is based on several parameters; the researcher should at least be aware of power, significance level and effect size. Increased power, smaller significance level and smaller effect sizes translate into larger sample sizes. The researcher and statistician are faced with selecting the most appropriate sample size from an appropriate set of parameters (subject to financial and logistical constraints).

Sample size calculations for more complex study designs can be regarded as multiplying the estimated sample size from an equivalent individual-level study by a design effect. Additional considerations involved in the calculation of this design effect include estimating the intra-cluster correlation and the sizes of the clusters, losses to follow-up and noncompliance.

Sample size calculations are an important and complex part of study design and should be discussed by study investigators and statisticians as early as possible during the design of a study design.

Acknowledgment

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Typhoid and paratyphoid fever in Western Sydney Local Health District, NSW, January–June 2011

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Abstract: We undertook a study of enteric fever, caused by *Salmonella enterica enterica* subtypes Typhi and Paratyphi A, presenting in residents of the Western Sydney Local Health District for the period January–June 2011. Twelve cases of *S.* Typhi and eight of *S.* Paratyphi A were notified. Patients were predominantly young adults (median age 26 years, 70% female) who had been visiting friends and relatives in India, Samoa, Bangladesh or Sri Lanka. No cases were associated with travel for less than 3 weeks; 17 (85%) required hospitalisation. None received pre-travel vaccination; reasons cited for this included pregnancy, expense, being too busy, or considering the disease too mild to warrant vaccination. Three *S.* Typhi isolates acquired at large social gatherings in Samoa had the same serotype and susceptibility profiles; these results were communicated to Samoan public health personnel. There are opportunities to strengthen enteric fever prevention, including pre-travel health advice and *S.* Typhi vaccination for people visiting endemic areas for 3 or more weeks, especially those in the vulnerable 'visiting friends and relative' category.

Enteric fever is caused by *Salmonella enterica* subspecies *enterica* serovars Typhi (*S.* Typhi) and Paratyphi (*S.* Paratyphi), with infection transmitted by the faecal-oral route.[1,2] While uncommon in Australia due to public health measures, worldwide the disease accounts for significant morbidity and mortality in children and adults.[3] A 2004 study estimated that 21 650 974 illnesses and 216 510 deaths occur each year caused by typhoid globally; paratyphoid accounts for an estimated 5 412 744 illnesses.[2] Asia has the highest estimated typhoid incidence rates at 274 per 100 000 population, south-central Asia being the most heavily-burdened area within this region. The Pacific Islands are categorised as having moderate incidence.[2,3]

In Australia, enteric fevers are notifiable (i.e. hospitals and laboratories are required to report cases) and require extensive follow-up to prevent spread.[4] Appropriate clinical management comprises administration of an antibiotic to which the isolate is susceptible (usually azithromycin or ceftriaxone) and the use of contact precautions.[5] On notification to the public health unit, the source of the infection is established and risk examined, including whether the patient has an occupation that may allow transmission (e.g. food handling; carer for children, elderly people or those with impaired immunity). In such cases, the patient is required to abstain from work until follow-up stool specimens are culture negative. Household contacts are given hygiene advice and recommendations to provide stool specimens for screening. Contacts working in transmission-prone occupations are required to demonstrate two uninfected stool samples before resuming work.[4]

The Western Sydney Local Health District (LHD) of New South Wales (NSW) has a high proportion of migrants from typhoid-endemic areas. Residents born overseas comprise 31–59% of the populations in the five local government areas within the boundaries of the Western Sydney Local Health District, and 25–75% of the population of the LHD speak a language other than English at home.[6] Foreign-born Australians visiting friends and relatives in their country of origin are well known to bear a disproportionate burden of the imported cases of enteric fever and other travel-related infections. Despite previous awareness-raising efforts, this group remains at risk.[7]

During the 5-year period 2006–2010, between 12 and 24 cases of enteric fever were reported in the Western Sydney Local Health District each year (rates per 100 000 population of 1.5–3.1) (Figure 1). However, in the 6 months from January–June 2011, 20 cases were reported, providing an estimated annual rate of 4.9 per 100 000 population. We investigated this higher-than-usual number of typhoid and

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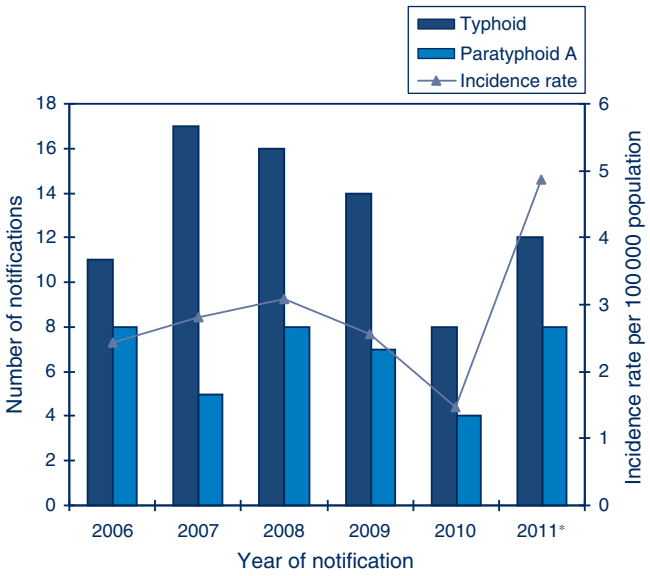

Figure 1. Numbers of annual notifications and incidence rates per 100 000 population for typhoid and paratyphoid A (enteric fever), Western Sydney Local Health District, NSW, for each year of the period January 2006–June 2011.
***2011 notifications to 30 June**
Source: Notifiable Conditions Information Management System, NSW Health.

paratyphoid cases in the Western Sydney Local Health District including describing the epidemiological, demographic and microbiological characteristics of these case-patients.

Methods

We undertook a retrospective, descriptive study of all confirmed cases of typhoid and paratyphoid A notified in the Western Sydney Local Health District between 1 January and 30 June 2011. Epidemiological, clinical and microbiological data were collated for each case-patient from public health unit, clinical and laboratory records. All age groups were included. A case of enteric fever was defined as the isolation of *S.* Typhi or *S.* Paratyphi from a sterile site or isolation from stool in a patient with the clinical features compatible with enteric fever. Serotyping was performed at the NSW Enteric Reference Laboratory, Institute for Clinical Pathology and Medical Research, according to the White-Kauffmann-Le Minor Scheme. Screening for reduced quinolone susceptibility was performed for isolates reported susceptible to ciprofloxacin, by testing nalidixic acid susceptibility using disc diffusion. This is an indicator of potential treatment failure if quinolone antibiotics are used for a patient.

Historical incidence data were extracted from the Notifiable Conditions Information Management System (NSW Health). Estimated resident populations in 2007 and 2010 were compiled by the Sydney West Epidemiology Unit from the census (Australian Bureau of Statistics).[6] Ethics approval was not required as data collection was routine public health practice.

Results

Twenty culture-positive cases were notified to the public health unit overseeing the Western Sydney Local Health District between January and June 2011: 12 *S.* Typhi, and eight *S.* Paratyphi A. Nineteen isolates were from blood (plus stool in one instance) and one was from stool only. Three isolates showing ciprofloxacin susceptibility had reduced susceptibility or resistance to nalidixic acid (Table 1), thus full quinolone susceptibility was identified in only six isolates (one *S.* Typhi isolate from India, all three *S.* Typhi from Samoa, and two *S.* Paratyphi from India). Serotyping results from the three isolates that were acquired by case-patients in Samoa were the same (Table 1), suggesting a potential outbreak; two affected case-patients had attended the same large gathering and the third had attended a conference in Samoa.

Demographics

The median age of case-patients was 26 years (range 1–45 years) with the highest incidence in the 20–29-year age group (Table 2). Fourteen (70%) were females of whom two were pregnant. All but two were resident in Australia; of the two non-residents, one was a recent immigrant and the other a traveller from overseas.

Infection source

All case-patients had recently visited settings where typhoid is endemic (Table 3). The majority ($n = 17$) were travellers visiting family or friends. The median duration of travel was 28 days (range 21–70 days); no infections were reported in people who travelled for less than 3 weeks. Onset of illness was usually within 1 week of arrival in Australia (in one instance the interval was 23 days) (Table 4).

Signs and symptoms

Of the 19 case-patients with a symptom history available, the most commonly reported symptom was diarrhoea ($n = 12$, 63%), followed by vomiting (52%), headache (31%), abdominal pain (26%) and lethargy (21%). None reported constipation. The most commonly reported clinical sign was fever. There were no serious complications and no deaths (Table 3).

Immunisation status

None of these 20 case-patients were immunised against typhoid. The reasons provided for lack of vaccination included, in one patient each, pregnancy, expense, being 'too busy' or thinking that the disease was too mild to warrant prevention.

Table 1. Typing and susceptibility results of the 12 isolates of *Salmonella* Typhi and 8 isolates of *Salmonella* Paratyphi A from 20 culture positive case-patients in Western Sydney Local Health District, January–June 2011

Isolate	Origin	Serotype	Phage type	Ciprofloxacin susceptibility	Nalidixic acid susceptibility
Salmonella Typhi					
1	India	*S.* Typhi 9,12,Vi:d:-	E1	RS	–
2	India	*S.* Typhi 9,12,Vi:d:-	E1	S	R
3	India	*S.* Typhi 9,12,Vi:d:-	E1	RS	–
4	India	*S.* Typhi 9,12,Vi:d:-	E1	RS	–
5	India	*S.* Typhi 9,12,Vi:d:-	E1	RS	–
6	India	*S.* Typhi 9,12,Vi:d:-	E1	RS	–
7	India	*S.* Typhi 9,12,Vi:d:-	E1	S	S
8	India	*S.* Typhi 9,12,Vi:d:-	E9	S	RS
9	India	*S.* Typhi 9,12,Vi:d:-	Untypable	S	R
10*	Samoa	*S.* Typhi 9,12,Vi:d:-	E1	S	S
11*	Samoa	*S.* Typhi 9,12,Vi:d:-	E1	S	S
12*	Samoa	*S.* Typhi 9,12,Vi:d:-	E1	S	S
Salmonella Paratyphi A					
1	India	*S.* Paratyphi A 2,12:a:-	1	S	S
2	India	*S.* Paratyphi A 2,12:a:-	1	RS	–
3	India	*S.* Paratyphi A 2,12:a:-	1	S	R
4	India	*S.* Paratyphi A 2,12:a:-	13	S	S
5	India	*S.* Paratyphi A 2,12:a:-	2	RS	–
6	India	*S.* Paratyphi A 2,12:a:-	4L	RS	–
7	Sri Lanka	*S.* Paratyphi A 2,12:a:-	1	RS	–
8	Bangladesh	*S.* Paratyphi A 2,12:a:-	Not typed	R	–

*Identical typing profile.
One isolate from each patient is shown.
S: susceptible; RS: reduced susceptibility; R: resistant.
Source: NSW Enteric Reference Laboratory, Institute for Clinical Pathology and Medical Research (ICPMR).

Transmission-prone occupations

One patient was a chef who had a close contact working in farming. One was a community worker for disabled children and another was a teacher. These three patients were advised to refrain from working until their infection had been successfully cleared.

Discussion

This descriptive study provides a snapshot of recent enteric fever cases in the Western Sydney Local Health District of NSW. None of these case-patients had pre-travel vaccination and all infections were contracted outside Australia. In keeping with previous reports, these findings illustrate that migrants or people from non-English speaking backgrounds are at particular risk when they stay in typhoid-endemic areas for 3 weeks or more. Three case-patients were employed in potentially transmission-prone occupations, illustrating the importance of appropriate clinical questioning and public health action to prevent autochthonous transmission within Australia. There were no known instances of secondary enteric fever infections related to these cases.

Reasons for year-to-year variation in enteric fever numbers may reflect outbreaks in the countries visited or changing travel patterns. The possibility of an enteric fever outbreak in Samoa was suspected on the basis of three case-patients having attended large gatherings there and they were all infected with the same serotype. NSW Health officials contacted Samoan health authorities, however no increase in instances of enteric fever had been recognised locally. In another Pacific nation, Fiji, a ban was placed by government officials on public gatherings in the province of Bua in May 2011 due to an outbreak of typhoid.[8]

'Visitors of friends and relatives' (17 of these case-patients) are a recognised high-risk group for travel-acquired infections. They are less likely to seek pre-travel advice, more likely to engage in behaviours which increase likelihood of exposure to pathogens (e.g. staying with family in rural areas, drinking local water supplies), and often stay for longer periods.[7,9] Improving pre-travel health advice to this at-risk group, in particular efforts to tailor pre-travel public health messages to Western Sydney Local Health District's migrant population, could be effective in increasing the uptake of pre-travel vaccination and assist with preventing these diseases.

Table 2. Characteristics of 20 case-patients confirmed with enteric fever in Western Sydney Local Health District, NSW, January–June 2011

Characteristic	*n*	%
Sex		
Female	14	70
Age (years)		
0–4	1	5
5–9	3	15
10–14	1	5
15–19	0	0
20–29	12	60
30–39	2	10
40–49	1	5
≥50	0	0
Country of birth		
Typhoid-endemic country[a]	13	65
Typhoid non-endemic country[b]	7	35
First language		
English	13	65
Not English	7	35
Country of acquisition		
India	15	75
Bangladesh	1	5
Sri Lanka	1	5
Samoa	3	15
Reason for travel		
Visiting relatives or friends	17	85
Holiday	1	5
Visiting Australia	1	5
Immigration to Australia	1	5
Duration of travel (weeks)		
1–2	0	0
3–4	9	45
≥5	9	45
Traveller to Australia	1	5
New immigrant	1	5

[a]India, Sri Lanka, Bangladesh, Samoa, Fiji.
[b]Australia, New Zealand, England.
Source: Parramatta Public Health Unit.

Table 3. Clinical signs and symptoms presented by 19 of the 20 case-patients with enteric fever in Western Sydney Local Health District, NSW, January–June 2011

	n	%
Documented clinical sign		
Fever (temperature >37°C)	19	100
Tachycardia	10	50
Relative bradycardia	1	5
Rose spots (erythematous macules usually on abdomen or chest)	1	5
Splenomegaly	0	0
Hepatomegaly	0	0
Symptoms		
Diarrhoea	12	63
Nausea and vomiting	10	52
Lethargy/malaise	4	21
Headache	6	31
Abdominal pain	5	26
Constipation	0	0

Table 4. Time of onset of symptoms relative to time of arrival in Australia of 20 case-patients with enteric fever in Western Sydney Local Health District, NSW, January–June 2011

Estimated incubation period (days)*	*n*	%
1–5 (before arrival)	4	20
1–7 (after arrival)	9	45
8–14 (after arrival)	4	20
15–21 (after arrival)	2	10
>22 (after arrival)	1	5

*Symptom onset time relative to arrival in Australia.
Source: Parramatta Public Health Unit and chart review.

The costs to the health system associated with the care of these case-patients was considerable, with most requiring hospitalisation. In addition the individuals and their families experienced costs and distress. In comparison, the recommended retail cost of vaccination, excluding general practitioner fees, of both oral and parenteral preparations is less than $50; a combined hepatitis A and typhoid parenteral vaccine is also available.[10,11] The relatively short duration of immunity (assumed as 3 years) from typhoid vaccine may be a deterrent to some people in vaccine uptake.

Young adults and school-aged children were the most affected, most likely reflecting the age of the travelling population. However, infrequent occurrence of confirmed cases in infants has also been demonstrated and attributed to the potential reluctance to obtain blood samples in infants,[12,13] or to typhoid being milder or atypical in preschool years due to undeveloped reticuloendotheial systems.[14] Infection is thought to confer at least partial protective immunity, which may account for lower rates in older travellers who were previously resident in endemic regions.[15]

Pregnant women were over-represented in this series. Pregnancy poses a particular risk for gastroenteric infections due to reduced gastrointestinal and biliary peristaltic activity and increased prevalence of biliary 'sludge' and concretions during pregnancy.[16] Vaccination (with parenteral vaccine, as live oral formulations are contraindicated in pregnancy)[4,5] is particularly recommended for pregnant women travelling to endemic areas because of their increased risk, and because of the relative contraindication to certain antimicrobial drugs during gestation.

Clinical features were mostly in keeping with previously published literature. Diarrhoea is a well-recognised symptom especially in the first week of illness, but the more classical picture is of constipation. Sixty-three percent of the patients in our series reported diarrhoea, exceeding rates of 6.5–25% reported elsewhere in immunocompetent people,[1,17,18] perhaps due to their early stage of illness or reporting bias. One person experienced no abdominal symptoms, highlighting the importance of a high index of suspicion for this infection in returned travellers, and appropriate specimen collection. Rose spots were uncommon; these occur in 5–50% of enteric fever patients,[1] however they are easily missed on people with dark skin and inexperienced clinicians may not recognise this sign. While no serious complications occurred in this group, relatively high complication rates of 10–15% are reported in endemic countries, possibly related to late presentation and under-reporting of milder cases.[1]

Most isolates showed reduced susceptibility or resistance to quinolone antibiotics, although all isolates from case-patients who acquired their infection in Samoa were fully sensitive. Knowledge of the geographical site of infection acquisition is helpful in determining empirical therapy. Current Australian guidelines recommend azithromycin as first-line therapy, or ceftriaxone if an intravenous agent is required, and still include the quinolone antibiotic, ciprofloxacin, as an alternative if infection was not acquired in the Indian subcontinent or South-East Asia.[5,15] Our findings confirm the appropriateness of these recommendations.

This study was limited by its retrospective design, with potential for recall bias. Multiple physicians at different centres managed the patients, and notes were designed for medical purposes not audit, therefore the data were incomplete. While the numbers are small, they highlight issues relevant to efforts to improve prevention strategies.

Conclusion

Migrants returning to their home country may underestimate the risk of infection, and may require targeted public health pre-travel advice, especially if they intend to stay in a typhoid-endemic area for long periods. While there is no vaccine against paratyphoid fever, typhoid vaccination could have prevented up to 60% of these infections. Consequently travel health advice should be accessible and culturally appropriate.

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Trends and risk factors for hepatitis A in NSW, 2000–2009: the trouble with travel

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Abstract: Aim: To analyse trends in hepatitis A notifications and information on exposure to risk factors, in particular international travel, collected through routine surveillance in NSW. **Methods:** Hepatitis A notification data for the period 2000–2009 were extracted from the Notifiable Diseases Database and analysed by age group, gender, area of residence and exposure risk factors, including travel, food eaten and contact with other possible infectious cases. **Results:** The notification rate for hepatitis A in NSW fell from 3.0 cases per 100 000 population in 2000 to 1.4 cases per 100 000 population in 2009. Notification rates were highest among people aged 20–24 years and residents of metropolitan Sydney. Travel to a country where hepatitis A is endemic was a risk exposure identified in 43% of cases. **Conclusion:** International travel to highly endemic countries continues to be the most common risk factor for hepatitis A infection notified in NSW despite recommendations that travellers be vaccinated prior to travel to these areas.

Hepatitis A is a vaccine-preventable viral illness[1] and has been estimated by the World Health Organization to result in 1.5 million clinical cases each year, with the majority of cases occurring in countries that have unsafe water and poor sanitation infrastructure.[2] The transmission of hepatitis A virus is primarily from person-to-person by the faecal-oral route, most frequently from close contacts such as household members. Less common routes of exposure include ingestion of contaminated water and food sources.[3] Other risk groups in which there were large outbreaks of hepatitis A in the 1990s include men who have sex with men and intravenous drug users.[4,5]

The onset of signs and symptoms of acute hepatitis A infection are usually abrupt and include fever, malaise, anorexia, nausea, abdominal discomfort, dark urine and jaundice.[6] In infants and children aged less than 5 years, symptoms are mild or absent in more than 80% of cases.[2] In adults, symptoms are more pronounced and potentially life-threatening if underlying liver disease is present.[6] The incubation period of hepatitis A ranges from 15 to 50 days with an average of 20–30 days, whilst the duration of signs and symptoms of infection vary between individuals but normally resolve within 2 months of onset.[3] The communicability of hepatitis A virus infection starts at up to 2 weeks before the onset of prodromal symptoms and ceases 1 week after onset of jaundice. Following infection with hepatitis A virus, lifetime immunity is usually conferred.[2,3]

Travel by individuals from low hepatitis A incidence countries, such as Australia, to countries with higher endemicity has emerged as a preventable risk factor.[7,8] A review of New South Wales (NSW) hepatitis A notification data conducted in 2006[9] found that travel to countries with moderate to high hepatitis A endemicity was a risk factor for more than 50% of notified cases. A highly effective vaccine has been available in Australia since 1994,[6] and is recommended prior to travel to endemic areas.[10]

The aim of this study was to examine trends in hepatitis A notifications in NSW for the period 2000–2009 and review data on exposure to hepatitis A risk factors identified through routine surveillance. A focus was whether international travel continued to be the most important risk factor.

Methods

Doctors, hospitals and laboratories in NSW are required to notify the local public health unit of cases of hepatitis A under the direction of the NSW *Public Health Act 1991*.* A confirmed diagnosis requires serological detection of

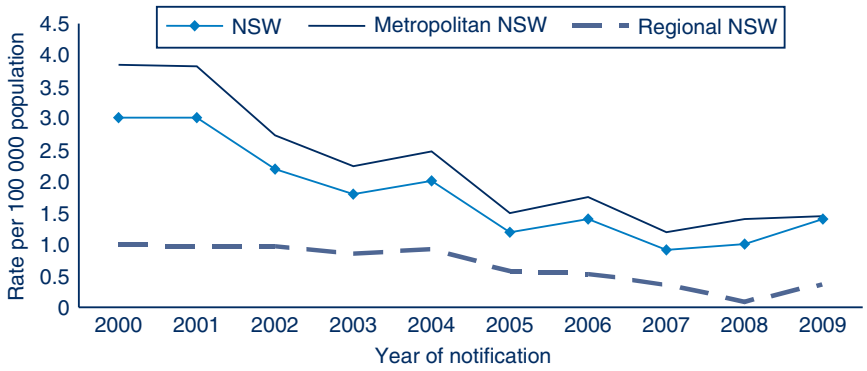

Figure 1. Comparison of annual hepatitis A notification rate per 100 000 population for regional and metropolitan residents of NSW, 2000–2009. Source: NSW Health Notifiable Diseases Database (now known as the Notifiable Conditions Information Management System).

anti-hepatitis A IgM, in the absence of recent vaccination, or detection by nucleic acid testing.[11] Public health unit staff are required to collect demographic and risk factor information for incident cases and enter this data into the Notifiable Diseases Database (NDD) (known since 2010 as the Notifiable Conditions Information Management System). This information includes age, gender, Aboriginality, area health service of residence and self-reported hospitalisation. Hepatitis A notifications were extracted from the NDD for the period 2000–2009 using the NSW Health data warehouse, HOIST (Health Outcomes Information Statistical Toolkit). Australian Bureau of Statistics mid-year population estimates were used to calculate crude annual notification rates.

The exposure information recorded in the NDD was travel history and country of travel, household contact of a hepatitis A case, eating raw shellfish, male-to-male sexual contact, exposure to raw sewage, drug use and attendance at child-care centres. We also performed subanalyses of exposure and hospitalisation information by age group.

Results

There were 1203 notifications of hepatitis A in NSW between 2000 and 2009. The highest number of notifications was reported in 2001 ($n = 200$) and the lowest in 2007 ($n = 65$). Since 2005, there have been fewer than 100 notifications each year. Annual notification rates of hepatitis A in NSW have remained stable at less than 3.0 per 100 000 population since 2001 (Figure 1). After falling to a low of 0.9 per 100 000 population in 2007, the rate increased to 1.4 per 100 000 population by 2009 (rates per 100 000 are yearly rates unless otherwise specified). Hepatitis A notifications rates were two- to three-fold higher in metropolitan populations compared with regional populations (Figure 1). Only 19 people were identified as Aboriginal from all notifications: a 10-year notification rate of 1.2 case-patients per 100 000 population.

Age and gender

The age range of individuals notified with hepatitis A in NSW was 11 months to 97 years with 50% of cases between the ages of 20 and 44 years. The 20–24-year age group had the highest notification rate for hepatitis A of 3.2 per 100 000 population (Figure 2), while the notification rate was below 1.0 per 100 000 population in the age groups over 54 years.

Overall, 62% of notifications were in males, however the gender imbalance decreased over the review period. During the first 5 years (2000–2004), 66% of notifications were in males, while 53% of notifications during the last 5 years (2005–2009) were in males.

Hospitalisation

Twenty-one percent ($n = 252$) of all notified cases between 2000 and 2009 for whom data were collected reported a period of hospitalisation during their infection. Of these, 57% ($n = 143$) were male. The highest hospitalisation rate was 0.6 per 100 000 population in the 20–24-year and 25–29-year age groups. All other age groups had hospitalisation rates of 0.5 per 100 000 population or lower.

Risk factors

Of the 1203 hepatitis A notifications, risk factor information was identified and recorded for 886 (74%). Travel to an endemic area or being a household contact of someone who had recently travelled to an endemic area were the most frequently identified risk factors. Together, these accounted for 69% of cases with documented risk factor information (Table 1). Where contact with a recently notified case of hepatitis A was reported, most cases identified a household member as the source case.

Identification of male-to-male sexual contact has declined as an exposure risk to hepatitis A, recorded in 53 (6%)

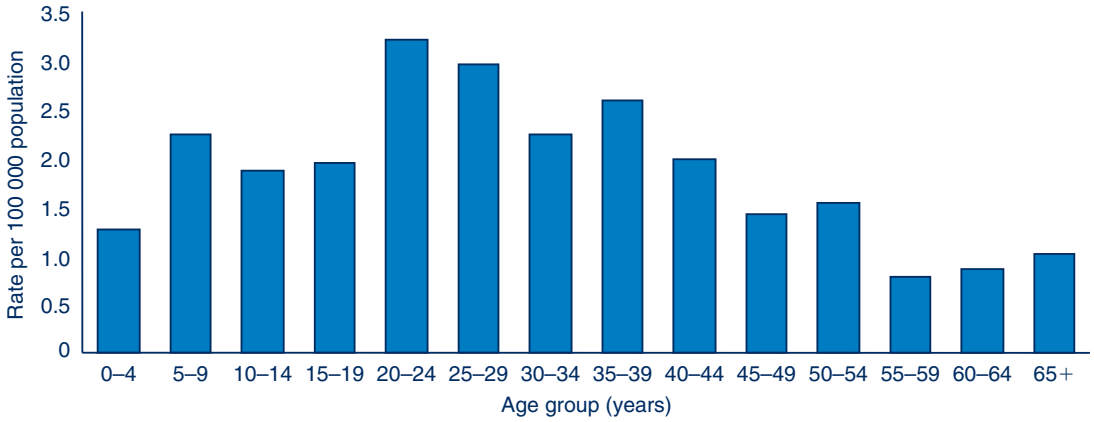

Figure 2. Hepatitis A notification rate per 100 000 population, NSW, 2000–2009, by age group. Source: NSW Health Notifiable Diseases Database (now known as the Notifiable Conditions Information Management System).

Table 1. Risk factors reported by notified hepatitis A cases, NSW, 2000–2009

Risk factor[a]	*n*	%
Travel to endemic areas	411	46
Household contact of a case that travelled to an endemic area	205	23
Ate raw shellfish	74	8
Contact with another possible case	69	8
Male-to-male sexual contact	60	7
Recreational drug use	29	3
Contact with raw sewage	25	3
Attends child-care centre	13	1
Total	**886**	–

[a]Categories are not mutually exclusive.
Source: NSW Health Notifiable Diseases Database (now known as the Notifiable Conditions Information Management System).

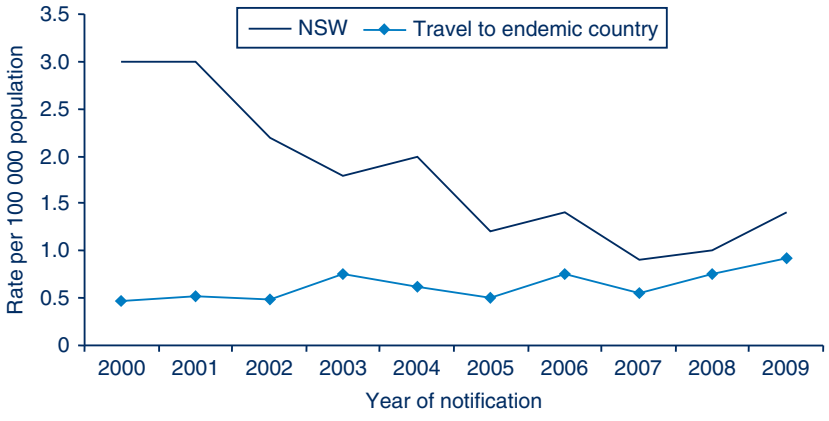

Figure 3. Annual hepatitis A notification rate per 100 000 population, NSW, 2000–2009, and the rate for cases with travel as an identified risk factor. Source: NSW Health Notifiable Diseases Database (now known as the Notifiable Conditions Information Management System).

notified cases during 2000–2004 and only in seven (1%) for 2005–2009.

Notification rates of cases with any travel history as a risk factor increased from 0.76 per 100 000 population in 2008 to 0.92 per 100 000 population in 2009 (Figure 3). The highest proportion of notified cases with travel recorded as a risk factor was 77% in 2008. This decreased to 65% in 2009, however, the overall notification rate increased from 1.0 per 100 000 to 1.5 per 100 000 in the same period.

Region of travel

International travel was identified as a primary risk factor for exposure to hepatitis A in 49% ($n = 431$) of all notifications; 411 (95%) of these had a history of recent travel to countries with moderate-to-high hepatitis A endemicity. This group had a median age at onset of infection of 22 years compared to a median of 32 years in cases where travel was not implicated. The regions of travel identified included South and South East Asia (44%), West and Central Asia (16%), Oceania (19%) and East Asia (6%). Travellers acquiring hepatitis A in West and Central Asian countries had the youngest median age at onset of 14 years, whereas the median age was 28 years among those travelling to other regions. The destinations most frequently recorded by cases included India, Lebanon, Pakistan, Indonesia, Fiji and Vanuatu.

Discussion

The notification rate of hepatitis A in NSW has remained stable at less than three cases per 100 000 population since 2001, with metropolitan populations experiencing much higher notification rates than regional areas of NSW. The notification rate was highest in the 20–29-year age group, and lowest in adults aged 55 years and older. Risk factors for exposure to hepatitis A such as male-to-male sexual contact and recreational drug use consistently declined in frequency over the period analysed, while travel to countries with moderate-to-high levels of endemicity has increased. Being a household contact and eating raw shellfish continued to be reported as risk factors. During the period of analysis there were no large scale foodborne outbreaks of hepatitis in NSW, although an increase in hepatitis A notifications in 2009 has been linked to semi-dried tomatoes in Victoria.[12]

This study used passive surveillance data which were limited by case reports with incomplete information on hepatitis A risk factors. Missing information may reduce the accuracy of the true prevalence of risk behaviours, as self-reporting can increase or decrease the incidence of behaviours such as male-to-male sexual contact and recreational drug use. Having experienced public health practitioners investigate case-patients improves the collection of accurate information while more complete case reports, including risk factor information, would enhance the usefulness of hepatitis A surveillance data.

The lowest hepatitis notification rate was in people aged 55 years and over. This age group has similar travel risks to younger people, with an Australian study from 2003 finding that travel to endemic countries was not more common in younger people than among older age groups.[13] Lower hepatitis A notification rates in people older than 55 years may be due to exposure to hepatitis A earlier in life, before improvements to sanitation infrastructure, and to the availability of hepatitis A vaccination.[2] Nationally, the annual hepatitis A notification rate has declined substantially from more than 50 cases per 100 000 population in 1970, to two per 100 000 population between 2003 and 2005.[1]

The low notification rates in young children may not reflect a low incidence of hepatitis A, as young children are more likely to have unrecognised or asymptomatic disease, which has been linked to sustained hepatitis A virus transmission in the community.[14] The hospitalisation rate was highest in young adults aged 20–29 years at 0.6 per 100 000 population, which reflects the increase of recognisable symptoms in adults with hepatitis A.[6] National notification data show higher hospitalisation rates in this age group, with the highest among males aged 34–59 years and females aged 60 years and over.[1] Aboriginal populations have high rates of hepatitis A immunity; this group have been notified and hospitalised with hepatitis A more often than non-Indigenous populations of Australia.[15] The targeted Indigenous hepatitis A vaccination program for the Top End of Australia excludes Indigenous children in NSW so we would not expect to see any impact of vaccination in NSW Aboriginal populations, in whom rates of hepatitis A in the absence of vaccination programs are high.[16]

Travel to countries of higher endemicity was the most important risk factor for acquiring hepatitis A among NSW cases, which confirms the results of a 2006 study that found that travel to these areas is a risk exposure for hepatitis A in NSW.[9] In 2002, travel was added as an exposure risk to be reported for each notified case of hepatitis A in NSW, which has resulted in improved recording of risk factor information over the period of analysis. By 2003, the average number of Australians travelling overseas on short holidays (less than 3 months) was 300 000 per month.[17] There has been a 72% increase to 517 000 travellers per month in 2009.[17] Studies in the United States of America and Canada have also identified that travel to countries with high endemicity is a common risk factor for acquisition of hepatitis A.[14,18]

Consultation for pre-travel health advice is an important opportunity for travellers to receive hepatitis A vaccination. A survey of pre-travel advice-seeking behaviour among Australian travellers identified low levels of advice seeking with only 33% of 503 surveyed travellers visiting a doctor or travel health clinic prior to their last international trip. For travellers to areas of high endemicity for hepatitis A or B, 41% had sought pre-travel advice with almost half of those obtaining pre-travel vaccination.[13] In comparison, a recent survey of Canadians found that only 24% of travellers to endemic countries had been vaccinated against hepatitis A.[19] Measures that increase the awareness and uptake of pre-travel vaccination of hepatitis A are likely to reduce the incidence of hepatitis A and its local transmission in the community. Any reduction in the

incidence of hepatitis A will have benefits for individuals as well as reduce the need for treatment and public health response.

Conclusion

The hepatitis A notification rate has remained stable in NSW and the most common risk factor for hepatitis A in NSW continued to be travel to countries of moderate-to-high endemicity or being a household contact of a person with hepatitis A infection who has travelled to one of these countries. Strategies that effectively inform the travelling public about the risks posed by hepatitis A and increase the uptake of pre-travel vaccination are likely to reduce the burden of hepatitis A in NSW.

Acknowledgment

This work was completed while EF was an employee of the NSW Public Health Officer Training Program, funded by the NSW Ministry of Health. The Program is offered in partnership with the University of New South Wales. He undertook this work while based at the Communicable Diseases Branch, NSW Ministry of Health.

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*The *Public Health Act 2010* (NSW) (http://www.health.nsw.gov.au/phact/)
The *Public Health Act 2010* (NSW) was passed by the NSW Parliament in December 2010 and commenced on 1 September 2012. The Public Health Regulation 2012 was approved in July 2012 and commenced, along with the *Public Health Act 2010* (NSW), on 1 September 2012. The objectives of the Regulation are to support the smooth operation of the Act. The Act carries over many of the provisions of the *Public Health Act 1991* (NSW) while also including a range of new provisions.

Overweight and obesity are common in rich and poor

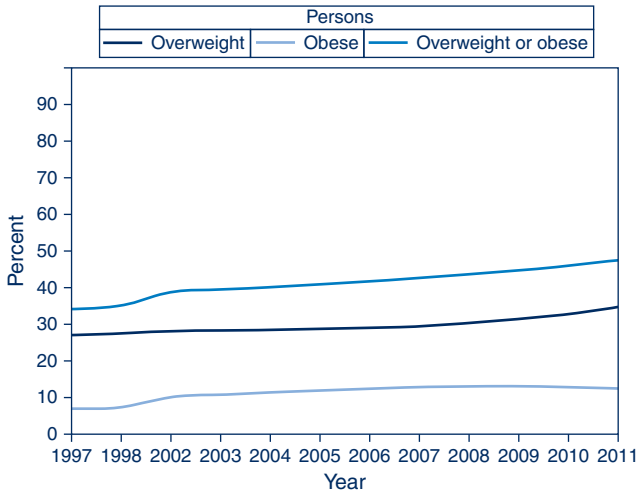

Figure 1. Overweight or obesity by sex, persons aged 16 years and over, NSW, least disadvantaged quintile, 1997 to 2011.

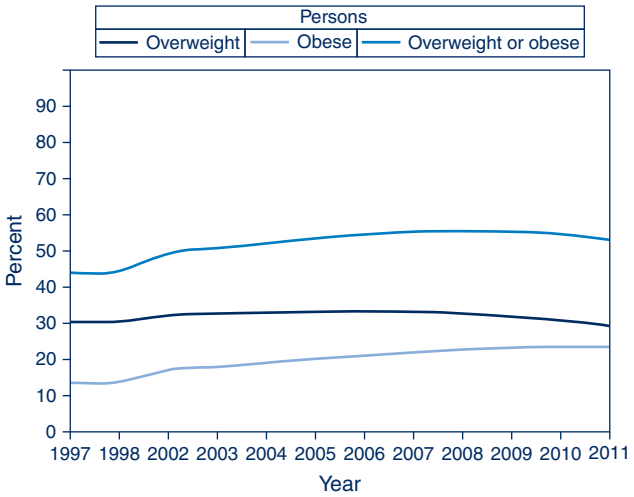

Figure 2. Overweight or obesity by sex, persons aged 16 years and over, NSW, most disadvantaged quintile, 1997 to 2011.

Source: NSW Adult Population Health Survey (SAPHaRI). Centre for Epidemiology and Evidence, NSW Ministry of Health.

A key performance indicator for the NSW health system is the prevalence of 'overweight or obesity' in the population. The amalgamation of the two categories of 'obese' and 'overweight' in this indicator masks differing trends in these individual categories over time. For the entire NSW population, the total category of 'overweight or obese' increased by 11.1% between 1997 and 2011. The increase for the 'obese' category alone was however 8.5% over this period and 'overweight' alone was 2.6%. The above figures compare these trends in the highest socioeconomic status (SES) (least disadvantaged) quintile (Figure 1) and the lowest SES (most disadvantaged) quintile (Figure 2). These figures show that, while the rate of increase of total 'overweight and obesity' between 1997 and 2011 is similar in the two groups, the rate of increase for the 'obese' category alone was higher in the low SES group compared to the high SES group over this period. The prevalence of 'obesity' alone increased from 7.2% to 12.7% between 1997 and 2011 for the high SES category (a 5.5% increase overall) and from 13.7% to 23.8% over the same period for the low SES group (10.1% overall).

In summary, while overweight and obesity are common across all socioeconomic groups in NSW, obesity is more common and is increasing more rapidly in lower socioeconomic groups.

The indicator covering *Overweight or Obesity* includes those who are overweight or obese: that is, with a Body Mass Index (BMI) of 25.0 or higher: overweight (BMI from 25.0 to 29.9) and obese (BMI of 30.0 and over).

Further information on these definitions and methods is available from the Health Statistics NSW website at: www.healthstats.nsw.gov.au/indicator/beh_bmi_age.

10.1071/NB12104

Rapid health assessments

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Rapid health assessments (RHAs) are used during an emergency response to gather information about the health status and needs of an affected population. They are a systematic way of collecting information in a complex emergency situation, with the information gathered assisting in planning, directing and implementing an appropriate response.[1] RHAs provide decision makers and partner agencies with a rapid insight into the health needs of an affected population.

The World Health Organization (WHO) recommends RHAs should be completed as soon as possible following an emergency and performed by a multidisciplinary team of qualified personnel, with an appropriate range of expertise.[2] The WHO outlines four steps in a RHA: planning, data collection, analysis and interpretation of findings, and presentation of results and conclusions.[2] RHAs can be used in a range of emergency settings and focus on different areas of concern; they can be completed in an international or local context and gather information on issues such as mental health, communicable disease and nutrition.

Rapid health assessments in an international context

RHAs are commonly used as part of an international public health emergency response; an example is the use of a RHA in a refugee or internally displaced persons camp. In this situation, RHAs involve the collection of information describing the demographics, mortality, morbidity, nutritional status, vital needs, shelter and security of the population. This information enables the prioritisation of interventions and identifies areas requiring further assessment. In a refugee or internally displaced persons camp, control and prevention of communicable disease and potential epidemics is a high priority; as a result, mass vaccination against measles is an important intervention which may occur following a RHA. Within large refugee camps and internally displaced persons camps continuous rapid assessments using networks of clinics, camp coordinators and camp visitors are used to monitor the evolving health profile within the camp.

Rapid health assessments in a local context

RHAs may be required as part of a public health emergency response following a natural disaster such as severe storm or flood. In a local response, a RHA would be completed in coordination with relevant emergency response organisations and may contribute to the overall initial impact assessment, completed by the Local Emergency Operations Controller during the response phase.

As a type of RHA, rapid cluster surveys can provide information to assist in preparing and planning for natural disasters. A local example of a rapid cluster survey was completed following a storm disaster in the Hunter region of NSW. The aim of the survey was to describe the impact of the storm, assess household disaster preparedness and identify information sources used before and during the disaster.[3] The survey found that: the storm had a major impact on households and essential services, many households lack basic disaster equipment including radios/batteries, local radio networks provide the most useful information, and awareness of the disaster role of local media networks could be strengthened.[3] There were a number of public health implications from the survey findings such as the need for: promoting the role of media networks during disasters, encouraging disaster preparation in households, and ensuring good communication occurs between emergency management organisations.[3]

For a RHA to be completed successfully the best available source of information should be determined, with the information collected addressing the needs of the users. Standardised methods and coordination should occur throughout the process to assist in distinguishing between emergency and long-term needs of the affected population.[4] RHAs are a practical and functional tool that can be used as part of a public health emergency response to identify health needs and assist with prioritisation of services.

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10.1071/NB12088

One Health and Hendra virus: a collaborative approach in action

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The One Health initiative (http://www.onehealthinitiative.com/) encompasses the health of humans, animals and the environment, recognising the indivisible interconnections that exist between these domains. As over 60% of emerging and re-emerging diseases are transmitted from animals to humans (zoonoses), the One Health initiative has significant potential to reduce the global health threat caused by infectious diseases.[1]

The concept of One Health is not new as the union of human and animal health has long been understood and accepted; however our view of zoonoses has traditionally been compartmentalised within public health and veterinary medicine, with little attention given to environmental health elements and cross-disciplinary collaborations. The One Health approach has gained increasing global attention, marked by the partnerships of major human health, animal health and environmental organisations. An integrated One Health approach requires commitment at global, national, state and local levels.[2]

The management of zoonoses in New South Wales (NSW) involves human, animal and environmental health sectors. For human health, the Centre for Health Protection, NSW Ministry of Health, is responsible for policy development and the coordination of statewide surveillance and response activities. NSW public health units detect and respond to local infectious disease issues and work collaboratively with members of the One Health team. For animal health, the Department of Primary Industries (DPI) oversees the health of livestock and companion animals. Regional veterinary officers work closely with local stakeholders, including human and environmental health experts. The NSW Office of Environment and Heritage monitors the health of free-ranging wildlife and zoo collection animals. Wildlife disease events are reported to the Australian Wildlife Health Network, who maintain the national wildlife disease surveillance database.[3]

Hendra virus infection: a One Health approach

Hendra virus infection was first described after an outbreak of severe respiratory disease in 18 horses and two humans in Brisbane in 1994. To date, there have been 24 cases in Queensland and nine in northern NSW. Horses appear to be incidental hosts following exposure to Flying-foxes or their excretions. Horse-to-horse transmission occurs infrequently and is more likely when horses are in very close contact. Infected horses usually experience rapid deterioration from brief but severe respiratory or neurological disease with a high case fatality rate. One dog became serologically positive probably after contact with an infected horse. In seven cases, Hendra virus infection has spread from horses to humans following close contact and has resulted in pneumonic or encephalitic illnesses. Four of these people have died. There is no evidence of human infection from other sources, such as direct contact with Flying-foxes. There is no known treatment and clinical management has been based on supportive measures.[4]

Hendra virus in horses is a notifiable disease in all Australian jurisdictions. If suspected, the NSW DPI works with NSW Health to complete a prompt epidemiological investigation to identify and monitor people at risk and provide advice to minimise the risk of infection to humans and other horses. The property where the horse cases are located is quarantined and animals that are infected are euthanased following guidelines in the *AUSVETPLAN Response Policy Briefs*. The human health response follows the *Hendra Virus National Guideline for Public Health Units* and includes the provision of information for all people at risk and testing and follow-up on a case-by-case basis. Open and transparent communication between all team members and to the general public is an important component of Hendra virus investigations.[4] The formation of the cross-border Intergovernmental Hendra Virus Taskforce further demonstrates support for the One Health approach in minimising the impacts of this potentially devastating disease on human and animal health.

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 10.1071/NB12079

Communicable Diseases Report, NSW, May and June 2012

Communicable Diseases Branch
NSW Department of Health

For updated information, including data and facts on specific diseases, visit www.health.nsw.gov.au and click on **Public Health** and then **Infectious Diseases**. The communicable diseases site is available at: http://www.health.nsw.gov.au/publichealth/infectious/index.asp.

Figure 1 and Tables 1 and 2 show notifications of communicable diseases received in May and June 2012 in New South Wales (NSW).

Enteric infections

Outbreaks of suspected foodborne disease

In March, NSW Health were notified of a complaint regarding a banquet at a Sydney restaurant (restaurant A). Eighteen people from a group of 24 who dined at the restaurant developed gastrointestinal symptoms following the meal. An epidemiological investigation was conducted after four of these people who became ill returned stool samples which tested positive for *Salmonella* (*Salmonella* Typhimurium MLVA 3-9-9-12-523: STm strain A). All those people who became ill had consumed the same dessert, a Bombe Alaska, containing raw egg meringue. NSW Food Authority officers inspected restaurant A and requested that it cease serving this dessert as it contained raw egg. The eggs used at restaurant A were purchased from an egg farm (egg farm A). This farm was inspected in April 2012. This egg farm also supplied another 10 businesses and sold eggs directly to the public.

Salmonella (STm strain A) was previously uncommon in NSW, but from January to March 2012 it was one of the most commonly reported types among cases with salmonellosis in south west Sydney. STm strain A was also identified in three case-patients who had complained of illness after a party at an unrelated restaurant in Sydney in January 2012. At the time, that investigation did not identify an association between illness and any particular food item. However, a profiterole cake consumed at the party had been supplied by a bakery in the area (bakery A). Bakery A was listed as one of the wholesale customers of egg farm A. The NSW Food Authority inspected bakery A in May 2012 and the same STm strain A was identified on re-usable piping bags, machine nozzles and from a bowl of freshly whipped cream.

NSW Health and OzFoodNet epidemiologists then interviewed additional confirmed cases of STm strain A (27 case-patients) and of another closely related strain (MLVA 3-9-8-12-523: STm strain B) (30 case-patients) that had been notified between January and April to determine whether they had consumed food prepared by either restaurant A or bakery A, from any of the other businesses that egg farm A supplied, or whether they had bought eggs directly from egg farm A. Despite the delay from the time of their illness to being interviewed, 46% (30/65) case-patients reported consuming eggs from either restaurant A or bakery A in their incubation period and a further case reported purchasing eggs directly from the farm. The NSW Food Authority are working with farm A to reduce the possibility of contamination of eggs occurring in the future.

Respiratory infections

Influenza

Influenza activity increased markedly in May and June 2012, consistent with an early start to the winter influenza season. Increased activity was measured by: increased notifications of laboratory-confirmed cases; an increased proportion of respiratory tests positive for influenza; outbreaks of influenza in aged-care facilities; and increased numbers of people presenting to 59 of the state's largest emergency departments with influenza-like illness and pneumonia, especially people aged over 65 years.

Overall, activity was well above that seen in the past 2 years for this time of year but remained below the peak levels seen during the influenza pandemic in 2009 and the severe influenza season in 2007.

Laboratory testing data for influenza showed that the most common influenza strain circulating was the new A/Victoria/361/2011-like strain, although influenza B viruses continued to circulate at low levels. No influenza antiviral resistance was reported.

The WHO Collaborating Centre on Influenza has advised that current influenza vaccines are likely to induce

significant protection against the new H3N2 strain, and that the current B strain is well matched to the vaccine strain.

For a more detailed report on respiratory activity in NSW see: http://www.health.nsw.gov.au/PublicHealth/Infectious/influenza_reports.asp

Vaccine-preventable diseases

Meningococcal disease

Twelve cases of meningococcal disease were notified in NSW in May and June 2012 (six in May and six in June), an increase from nine for the same period in 2011. The age of the case-patients ranged from 2 months to 65 years and included five children aged under 5 years. There were no deaths notified in this period. Seven cases were due to serogroup B (for which there is no vaccine), one was due to serogroup Y, two were unable to be typed, and for two there was insufficient specimen collected.

Of the nine cases notified during the same period in 2011, five were due to serogroup B, one to serogroup Y and the remaining three were of an undetermined serogroup.

It is recommended that a single dose of the vaccine for meningococcal C disease be given to all children at the age of 12 months as well as to people at high risk of the disease.[1]

Measles

Fifteen cases of measles were notified in NSW in May and June 2012. This was an increase from six cases reported for the same period in 2011.

One case of measles notified during this period was epidemiologically linked to a previously reported cluster that commenced in April and was associated with a young adult returning home from Thailand while infectious (total case-patients, $n = 4$).

A cluster of six cases notified during May and June were linked to the emergency department of the Children's Hospital at Westmead and subsequently at a high school formal. The average age of case-patients was 11 years (range: 7 months to 32 years). Of the six case-patients, three were too young to be vaccinated, two were unsure of their vaccination status and one reported receiving only one measles-containing vaccine. Cases in this cluster were notified across metropolitan Sydney.

A second cluster of six cases notified during May and June were reported primarily in under-immunised teenage students at a high school in the Sydney South West Local Health District. The case-patients were aged between 13 and 19 years. Of the six case-patients, three were not vaccinated, two were unsure of their vaccination status and one reported receiving only one measles-containing vaccine.

A further two sporadic measles cases were also notified during this period. The first case was an unvaccinated 1-year old infant who acquired their infection in China. The second case was a 7-month old infant (who was too young to be vaccinated) whose source of infection remains unknown (measles virus genotype B3). While the measles virus genotype B3 has been the predominant genotype identified in African countries in recent years, and to a lesser degree in other continents,[2] it has not been identified during the past decade in NSW. Given the high sensitivity of measles surveillance in NSW and the lack of previously identified measles virus genotype B3, this suggests the infant's infection was transmitted from an unknown person who acquired their infection overseas. No further transmission was identified from either of these sporadic cases.

While a public health investigation did not identify a direct epidemiological link between the Thailand cluster or the two clusters reported during May and June, the clusters were found to have the same measles virus genotype and sequence of D8. In the absence of an epidemiological linkage between the clusters, the genotype information indicates that it is likely that there have been further undiagnosed (and therefore un-notified) cases of measles in the community.

Under-immunised high school students and infants too young to be vaccinated have been at the greatest risk of disease in these measles clusters. Two doses of measles-mumps-rubella (MMR) vaccine at 12 months and at 4 years of age are recommended for all children.

Pertussis (whooping cough)

During May and June 2012, 962 cases of pertussis were notified in NSW. This is a decrease from the 2036 cases notified for the same period in 2011.

NSW Health recently announced a refocused strategy to help prevent severe disease and death from pertussis by offering free vaccination to new mothers in maternity units or at their GP within 2 weeks of giving birth.[3] The program will continue until more definitive evidence becomes available about the effectiveness of vaccinating adults to protect new babies. NSW Health is collaborating with the National Centre for Immunisation Research and Surveillance in a study to assess the effectiveness of this strategy.

Direct protection for young infants remains available through free vaccination that is administered at 2, 4 and 6 months of age. The first dose can be provided as early as 6 weeks of age. There is also a booster dose at 3½ to 4 years of age.

Sexually transmissible infections/bloodborne viruses

HIV infections

In 2011, 330 people were newly diagnosed with HIV infection in NSW, an increased number than in 2010 ($n = 307$), but similar to the previous 3-year average of 320. Of those newly diagnosed in 2011, 309 (94%) were male and the median age at diagnosis was 35 years. The probable risk exposure category of most of the case-patients was reported to be homosexual contact ($n = 277$, 84%), with the exposure category of the others reported to be: heterosexual contact ($n = 42$, 13%), injecting drug use ($n = 8$, 2.4%) and other/unknown source ($n = 3$, 0.9%).

The median age of homosexually-acquired HIV case-patients was 34 years and more than half of these people ($n = 163$, 59%) were Australian-born. Three-quarters (73%) of homosexually-acquired infections were thought to be acquired in Australia, while 14% were reported to have been acquired overseas. There was an increase in both the number and proportion of homosexually-acquired HIV infections notified in 2011 ($n = 277$, 84%) compared to 2010 ($n = 235$, 77%).

Of the 42 heterosexually-acquired cases, 26 (67%) were male and 16 (43%) were female. The median age was 40 years. Fifteen case-patients (36%) were born in Australia, 12 (29%) were born in high HIV prevalence countries,[4] 14 (33%) were born in countries not classified as having a high prevalence of HIV infection and there was one (2.4%) case-patient for whom the country of birth was not stated.

Of the eight case-patients whose likely risk exposure was injecting drug use, four were men and four were women. The median age of this group was 41 years.

Promoting safe sex practices, particularly among men with homosexual exposures, remains important in the prevention of HIV infection.

A summary of 2011 HIV surveillance data has been published on the NSW Health website at http://www.health.nsw.gov.au/resources/publichealth/infectious/diseases/hiv_aids/new_diagnoses_hiv_infection_2011.pdf and http://www.health.nsw.gov.au/resources/publichealth/infectious/diseases/hiv_aids/hiv_surveillance_data_2011.pdf.

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Figure 1. Reports of selected communicable diseases, NSW, Jan 2004 to June 2012, by month of onset

Preliminary data: case counts in recent months may increase because of reporting delays.
Laboratory-confirmed cases only, except for measles, meningococcal disease and pertussis.
BFV = Barmah Forest virus infections, RRV = Ross River virus infections,
lab conf = laboratory confirmed,
Men Gp C and Gp B = meningococcal disease due to serogroup C and serogroup B infection, other/unk = other or unknown serogroups.
NB: Multiple series in graphs are stacked, except gastroenteritis outbreaks.
NB: Outbreaks are more likely to be reported by nursing homes and hospitals than by other institutions.

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

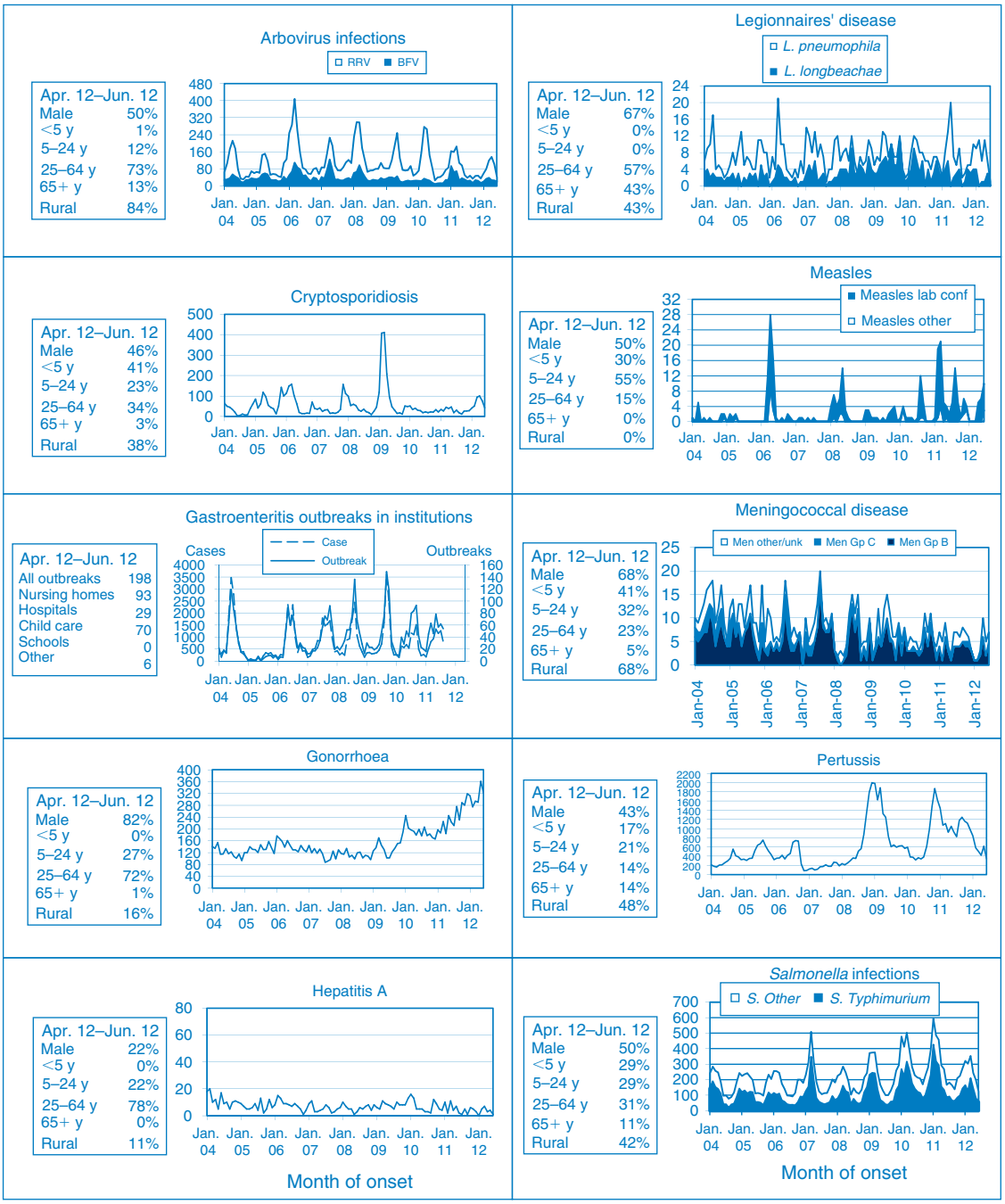

Table 1. Reports of scheduled medical conditions received in May 2012 by Local Health District, NSW

Condition	Local Health District																Total	
	Murrumbidgee	Southern NSW	Western NSW	Far West	Hunter New England	Northern NSW	Mid North Coast	Central Coast	Northern Sydney	South Eastern Sydney	Illawarra Shoalhaven	Sydney	South Western Sydney	Western Sydney	Nepean Blue Mountains	Justice Health	For May[b]	Year to date[b]
Bloodborne and sexually transmitted																		
Chancroid[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Chlamydia (genital)[a]	54	27	74	12	348	98	57	95	142	364	124	207	212	178	94	22	2109	9425
Gonorrhoea[a]	1	2	1	3	32	5	4	4	26	113	11	93	35	42	9	2	383	1673
Hepatitis B – acute viral[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	10
Hepatitis B – other[a]	4	3	5	3	6	1	2	4	15	27	7	48	33	54	2	5	219	1003
Hepatitis C – acute viral[a]	–	–	1	–	2	–	–	–	–	1	–	–	–	–	–	–	4	18
Hepatitis C – other[a]	8	8	15	8	25	15	4	24	8	29	11	37	29	34	9	12	276	1427
Hepatitis D – unspecified[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Lymphogranuloma venereum	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	6
Syphilis	2	2	2	–	3	–	–	1	5	20	2	10	9	–	1	–	57	283
Vectorborne																		
Barmah Forest virus[a]	1	1	–	–	12	11	4	1	1	–	4	–	–	–	–	–	35	160
Ross River virus[a]	27	2	12	1	28	18	6	4	–	–	5	3	2	–	5	1	114	389
Arboviral infection (other)[a]	–	2	1	–	2	4	1	2	9	3	–	7	4	–	3	–	38	149
Malaria[a]	1	–	–	–	–	–	–	–	1	1	1	–	1	3	–	–	8	25
Zoonoses																		
Anthrax[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Brucellosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	1	2
Leptospirosis[a]	1	1	–	–	–	2	–	–	–	–	1	–	–	–	–	–	5	–
Lyssavirus[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Psittacosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	–	2	8
Q fever[a]	–	–	–	1	1	6	1	–	–	–	1	–	–	–	–	–	10	59
Respiratory and other																		
Blood lead level[a]	7	1	10	12	14	2	–	–	4	1	1	3	3	2	1	–	61	182
Influenza[a]	2	1	3	–	8	19	–	1	36	36	1	18	32	44	8	–	209	536
Invasive pneumococcal infection[a]	2	–	2	–	10	2	1	4	7	7	8	5	1	7	5	–	61	158
Legionella longbeachae infection[a]	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	1	12
Legionella pneumophila infection[a]	–	–	–	–	1	–	–	–	–	–	–	1	–	4	1	–	7	39
Legionnaires' disease (other)[a]	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	1	5
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Meningococcal infection (invasive)[a]	1	–	1	–	–	–	–	–	1	2	1	–	–	–	–	–	6	22
Tuberculosis	–	–	–	–	–	–	1	–	2	9	–	4	1	3	–	–	20	120
Vaccine-preventable																		
Adverse event after immunisation	8	4	–	–	4	3	2	–	1	–	–	1	2	1	–	–	26	80
H. *influenzae b* infection (invasive)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Measles	–	–	–	–	–	–	–	–	1	–	–	–	–	5	1	–	7	12
Mumps[a]	–	–	–	–	–	1	–	–	8	5	–	3	2	3	–	–	22	52
Pertussis	25	27	37	–	63	30	22	15	58	37	53	19	51	88	92	–	617	3175
Rubella[a]	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	1	6
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Enteric																		
Botulism	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cholera[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	1	1
Cryptosporidiosis[a]	3	4	1	–	13	10	4	4	16	15	4	9	5	9	3	–	100	398
Giardiasis[a]	3	5	11	–	19	2	4	9	36	51	16	18	20	14	18	1	227	1067
Haemolytic uraemic syndrome	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2
Hepatitis A[a]	–	–	–	–	–	1	–	–	–	3	–	1	–	2	–	–	7	19
Hepatitis E[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Listeriosis[a]	–	–	–	–	–	–	–	–	–	1	–	–	1	–	–	–	2	16
Rotavirus[a]	5	–	4	–	13	2	1	–	1	9	–	1	2	12	5	–	55	284
Salmonellosis[a]	14	6	8	2	23	20	9	8	32	26	9	23	31	22	4	5	242	1517
Shigellosis[a]	–	–	1	–	–	–	1	–	1	1	–	–	3	1	1	–	9	62
Typhoid[a]	1	–	–	–	1	–	–	–	–	1	–	–	1	1	1	–	6	25
Verotoxin-producing *E. coli*[a]	–	1	–	–	–	2	–	–	–	–	–	–	–	–	–	–	3	10
Miscellaneous																		
Creutzfeldt–Jakob disease	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Meningococcal conjunctivitis	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

[a]Laboratory-confirmed cases only. [b]Includes cases with unknown postcode.
NB: Data are current and accurate as at the preparation date. The number of cases reported is, however, subject to change, as cases may be entered at a later date or retracted upon further investigation.
Data is reported as of Local Health District.
Source: Notifiable Conditions Information Management System, Centre for Health Protection, NSW Ministry of Health.

Table 2. Reports of scheduled medical conditions received in June 2012 by Local Health District, NSW

Condition	Local Health District																Total	
	Murrumbidgee	Southern NSW	Western NSW	Far West	Hunter New England	Northern NSW	Mid North Coast	Central Coast	Northern Sydney	South Eastern Sydney	Illawarra Shoalhaven	Sydney	South Western Sydney	Western Sydney	Nepean Blue Mountains	Justice Health	For June[b]	Year to date[b]
Bloodborne and sexually transmitted																		
Chancroid[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Chlamydia (genital)[a]	56	28	59	17	208	71	45	75	158	261	81	150	121	141	50	9	1533	10 958
Gonorrhoea[a]	6	1	3	9	18	5	3	5	19	118	7	70	30	27	9	–	331	2004
Hepatitis B – acute viral[a]	–	–	–	–	–	–	–	–	–	1	–	–	1	–	–	–	2	12
Hepatitis B – other[a]	1	–	2	1	3	1	6	6	24	24	11	29	36	33	2	3	182	1185
Hepatitis C – acute viral[a]	–	–	–	–	2	1	1	–	–	–	–	–	–	–	–	–	4	22
Hepatitis C – other[a]	6	8	8	7	17	18	10	16	14	15	12	26	35	25	15	19	251	1678
Hepatitis D – unspecified[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Lymphogranuloma venereum	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	1	7
Syphilis	1	1	2	1	–	–	1	3	1	20	2	–	–	–	–	–	32	315
Vectorborne																		
Barmah Forest virus[a]	–	–	–	–	2	10	5	2	1	–	2	–	1	–	–	–	23	183
Ross River virus[a]	7	–	–	1	9	7	1	1	1	–	1	1	3	–	–	–	32	421
Arboviral infection (other)[a]	–	1	–	–	1	2	–	–	3	4	1	–	3	1	–	–	16	165
Malaria[a]	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	1	26
Zoonoses																		
Anthrax[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Brucellosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2
Leptospirosis[a]	1	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	2	15
Lyssavirus[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Psittacosis[a]	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	1	9
Q fever[a]	1	–	–	–	3	1	1	–	–	–	1	–	–	–	–	–	7	66
Respiratory and other																		
Blood lead level[a]	23	1	13	3	7	–	–	1	–	4	2	–	1	3	–	–	58	240
Influenza[a]	3	12	12	1	87	121	20	20	222	189	14	97	200	274	54	–	1326	1862
Invasive pneumococcal infection[a]	4	4	4	1	6	1	1	2	9	7	5	2	7	6	5	–	64	222
Legionella longbeachae infection[a]	–	–	–	–	–	–	–	–	–	–	2	–	–	–	–	–	2	14
Legionella pneumophila infection[a]	1	–	–	–	–	–	–	–	–	1	–	1	–	–	–	–	3	42
Legionnaires' disease (other)[a]	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	1	6
Leprosy	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	1
Meningococcal infection (invasive)[a]	–	–	–	–	1	–	1	–	–	–	3	–	–	1	–	–	6	28
Tuberculosis	–	–	–	–	–	–	–	–	4	4	1	3	2	2	–	–	16	136
Vaccine-preventable																		
Adverse event after immunisation	1	–	1	–	2	–	–	–	3	–	4	–	–	6	–	–	17	97
H. *influenzae b* infection (invasive)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Measles	–	–	–	–	–	–	–	–	1	–	–	–	6	1	–	–	8	20
Mumps[a]	–	–	–	–	–	–	–	1	3	7	–	–	1	1	–	–	13	65
Pertussis	19	37	26	–	22	20	15	18	27	24	41	9	40	21	26	–	345	3520
Rubella[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	1	7
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Enteric																		
Botulism	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cholera[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Cryptosporidiosis[a]	1	1	1	–	3	–	1	5	11	11	–	3	2	3	–	–	42	440
Giardiasis[a]	2	1	6	–	23	2	2	9	28	18	8	16	8	9	9	–	141	12 108
Haemolytic uraemic syndrome	–	–	1	–	–	–	–	–	2	–	–	–	–	1	–	–	4	6
Hepatitis A[a]	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	1	20
Hepatitis E[a]	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	1	4
Listeriosis[a]	–	–	–	–	–	–	–	–	–	1	1	–	–	–	1	–	3	19
Rotavirus[a]	1	–	1	–	14	5	1	2	4	11	–	7	1	4	6	–	57	341
Salmonellosis[a]	8	1	6	2	18	8	3	2	20	13	8	17	17	11	5	–	139	1656
Shigellosis[a]	1	–	–	–	–	1	–	–	–	2	–	1	–	–	–	–	5	67
Typhoid[a]	–	–	–	–	–	–	–	–	–	–	–	–	2	–	–	–	2	27
Verotoxin-producing *E. coli*[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	10
Miscellaneous																		
Creutzfeldt–Jakob disease	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Meningococcal conjunctivitis	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

[a]Laboratory-confirmed cases only. [b]Includes cases with unknown postcode.
NB: Data are current and accurate as at the preparation date. The number of cases reported is, however, subject to change, as cases may be entered at a later date or retracted upon further investigation.
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